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Do changes in cardiovascular risk factors explain changes in mortality from stroke in Finland?

Erkki Vartiainen, Cinzia Sarti, Jaakko Tuomilehto, Kari Kuulasmaa

Abstract

***Objectives*—To estimate the extent to which the changes in the main cardiovascular risk factors (blood pressure, smoking, and serum cholesterol concentration) can explain the observed changes in mortality from stroke in Finland during the past 20 years.**

***Design*—Predicted changes in mortality from cerebrovascular disease mortality were calculated by a proportional hazards model from data obtained in cross sectional population surveys in 1972, 1977, 1982, 1987, and 1992. Predicted changes were compared with the observed changes in mortality statistics.**

***Setting*—North Karelia and Kuopio provinces, Finland.**

***Subjects*—16 741 men and 16 389 women aged 30-59 randomly selected from the national population register, of whom 14 054 men and 14 546 women participated.**

***Main outcome measures*—Levels of risk factors and predicted and observed changes in mortality from cerebrovascular disease.**

***Results*—The observed changes in diastolic blood pressure, total serum cholesterol concentration, and smoking in the population from 1972 to 1992 predicted a 44% fall in mortality from stroke in men and changes in diastolic blood pressure and smoking predicted a 34% fall in women. The observed fall in mortality from stroke was 66% in men and 60% in women.**

***Conclusions*—Two thirds of the fall in mortality from stroke in men and half in women can be explained by changes in the three main cardiovascular risk factors.**

Department of Epidemiology and Health Promotion, National Public Health Institute Mannerheimintie 166, FIN-00300 Helsinki, Finland
Erkki Vartiainen, *head of laboratory*
Cinzia Sarti, *senior researcher*
Jaakko Tuomilehto, *professor*
Kari Kuulasmaa, *senior statistician*

Correspondence to:
Dr Vartiainen.

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Introduction

Mortality from stroke has been falling in most industrialised countries in the past 20 to 30 years.[1] Although there are many studies on risk factors for stroke, little is known about the extent to which changes in the main cardiovascular risk factors (blood pressure, serum cholesterol concentration, and smoking) explain this fall.

Prospective studies on the risk factors for stroke have shown that high systolic or diastolic blood pressure is the most important risk factor in men and women.[2-6] A review of 14 randomised trials on hypertensive treatment showed that a fall in mean diastolic blood pressure of 5-6 mm Hg is associated with a 35-40% fall in mortality from stroke.[7-8] A meta-analysis on cigarette smoking and stroke showed an excess risk of stroke among male and female smokers, increasing with the number of cigarettes smoked.[9] Low serum cholesterol concentration is a risk factor for cerebral haemorrhage[2 3] but not subarachnoid haemorrhage.[10 11] High serum cholesterol concentration predicts cerebral infarction.[11-13] This divergent effect on the different subtypes of stroke may explain why total serum cholesterol concentration does not seem to be a significant predictor of all stroke.[5 6]

We studied the extent to which changes in blood pressure, smoking, and total serum cholesterol concentration can explain the fall in mortality from stroke and evaluated the relative importance of each of these risk factors. Similar analyses on ischaemic heart disease have been published.[14]

Subjects and methods

The levels of coronary risk factors in the provinces of North Karelia and Kuopio were assessed in five cross sectional population surveys (in 1972, 1977, 1982, 1987, and 1992). For each survey an independent random sample was drawn from the national population register. In the 1972 and the 1977 surveys a random sample of 6·6% of the population born during 1913-47 was drawn in both areas. In 1982, 1987, and 1992 the sample included people aged 25-64 years; the samples were stratified so that at least 250 subjects of each sex and 10 year age group were chosen in each area. The common age range in all the five surveys was 30-59 years, which is the age range used in this analysis. Because different people took part in each survey we could not measure changes within subjects.

The survey methods followed the World Health Organisation protocol for the monitoring trends and determinants in cardiovascular disease (MONICA) project in 1982, 1987, and 1992, and these methods were comparable with those used in 1972 and 1977. Each survey followed the same methods as closely as possible, and both areas were treated in the same way. Blood pressure was measured in the right arm of sitting subjects after five minutes' rest. The fifth phase of the Korotkoff sounds was recorded as the diastolic pressure. The bladder cuff was shorter (23 cm) in 1972 and 1977 than in 1982, 1987, and 1992 (42 cm).

Serum cholesterol concentration was measured from frozen samples by the Liebermann-Burchard method in 1972 and 1977,[15] whereas in 1982, 1987, and 1992 it was measured in fresh sera by an enzymatic method (CHOD-PAP, Boehringer Mannheim). The enzymatic assay gave 2·4% lower values than the Liebermann-Burchard method. We therefore corrected cholesterol values from 1972 and 1977 for this bias. All cholesterol measurements were made in the same central laboratory standardised against national and international reference laboratories.

Smoking was assessed by a standard self adminis-

TABLE I—*Rate of participation by year and area in men and women aged 30-59 years who participated in five surveys of cardiovascular risk factors in Finland. Values are percentages (numbers of subjects)*

	North Karelia		Kuopio	
Year	Men	Women	Men	Women
1972	94 (1834/1959)	96 (1973/2056)	91 (2665/2918)	94 (2769/2949)
1977	87 (1785/2063)	91 (1845/2020)	89 (2616/2933)	92 (2756/2996)
1982	77 (1229/1599)	84 (1276/1511)	83 (1207/1459)	88 (1011/1143)
1987	79 (1194/1521)	87 (1270/1485)	82 (624/762)	87 (649/744)
1992	69 (521/759)	81 (611/750)	76 (582/768)	85 (622/735)

TABLE II—*Levels of cardiovascular risk factors by year and sex. Values are means (SD) unless stated otherwise*

	1972	1977	1982	1987	1992
Men:					
Cholesterol (mmol/l)	6·78 (1·27)	6·55 (1·23)	6·28 (1·20)	6·23 (1·21)	5·90 (1·09)
Diastolic blood pressure (mm Hg)	92·8 (12·0)	91·0 (11·7)	87·8 (13·0)	88·4 (11·6)	84·2 (12·1)
No (%) of smokers	2322 (52)	2020 (47)	982 (42)	700 (39)	410 (37)
Women:					
Cholesterol (mmol/l)	6·72 (1·33)	6·36 (1·31)	6·07 (1·30)	5·94 (1·22)	5·54 (1·06)
Diastolic blood pressure (mm Hg)	91·8 (12·7)	87·6 (11·5)	84·6 (11·9)	83·5 (11·4)	79·6 (11·6)
No (%) of smokers	522 (11)	543 (12)	356 (16)	320 (16)	249 (20)

TABLE III—*Proportional hazards regression model for risk of stroke*

	Estimate of coefficient	Hazard ratio for unit increase of risk factor (95% confidence interval)
Men:		
Age (years)	0·10	1·11 (1·09 to 1·13)
Diastolic blood pressure (mm Hg)	0·05	1·05 (1·04 to 1·06)
Smoking (yes, no)	0·61	1·84 (1·38 to 2·43)
Cholesterol (mmol/l)	0·12	1·12 (1·01 to 1·26)
Women:		
Age (years)	0·12	1·13 (1·10 to 1·15)
Diastolic blood pressure (mm Hg)	0·04	1·04 (1·03 to 1·11)
Smoking (yes, no)	0·94	2·56 (1·65 to 3·99)
Cholesterol (mmol/l)	−0·06	0·95 (0·84 to 1·07)

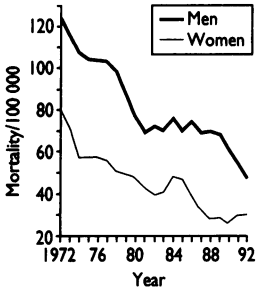

FIG 1—*Mortality from stroke standardised for age in men and women aged 35-64 years. Three year moving means*

TABLE IV—*Observed and predicted percentage fall in mortality from stroke compared with 1972 (95% confidence interval)*

	Observed	Predicted
Men:		
1972	0	0
1977	17 (2 to 32)	14 (11 to 18)
1982	42 (29 to 53)	29 (23 to 36)
1987	45 (33 to 55)	29 (22 to 35)
1992	62 (52 to 69)	44 (35 to 52)
Women:		
1972	0	0
1977	31 (14 to 45)	15 (10 to 19)
1982	51 (37 to 61)	21 (14 to 28)
1987	60 (48 to 70)	25 (17 to 32)
1992	63 (51 to 72)	34 (22 to 42)

tered questionnaire. We classified respondents into smokers (those who had smoked cigarettes, cigars, or a pipe regularly for at least one year and had smoked more than once a day, on average, during the preceding six months) and non-smokers (those who had never smoked regularly and those who had smoked regularly but had stopped smoking no later than six months before the survey).

The subjects examined in 1972, 1977, and 1982 were followed up until 1992 for death from stroke (ICD 430-438 as the underlying cause of death) by linking them with the national mortality register. Cox's proportional hazards regression models were fitted to the follow up data. Analyses were made separately for men and women. Age and baseline values of serum total cholesterol concentration and diastolic blood pressure were included as continuous variables and smoking status as a dichotomised variable. Cohort study year (1972, 1977, and 1982), interactions between risk factors, and second order terms in risk factors were left out from the final model because they were not significant. Data on these variables were available for 14 053 men, of whom 198 had died of stroke, and for 14 512 women, of whom 163 had died of stroke.

The predicted fall in mortality from stroke was calculated by the fitted Cox's model. This was done separately for each survey year by entering the mean risk factor values from each survey into the model. The use of the mean risk factor levels has been shown to approximate well to the sum of the individual hazards.[16] The relative importance of each risk factor was estimated by changing the value of only one risk factor in the Cox's model and keeping the other risk factors at the 1972 level.

The approximate confidence intervals of the predicted changes in mortality were calculated from the standard errors of the coefficients in the Cox's model and the standard errors of the changes in the mean values of the risk factors assuming that these estimates were statistically independent.

The actual annual mortality from stroke was obtained from the national mortality register for the two provinces studied. Mortality from stroke was standardised for age in 10 year age groups, with the 1972 population of Finland as standard. The percentage fall in mortality from stroke was calculated by using three year moving means from 1972 to 1992. The 95% confidence interval for the observed fall between two years was calculated from a log-linear model, with age group and year as covariates and assuming Poisson distribution for the number of deaths.

Results

Table I shows the sample sizes and participation rates in the five surveys. The participation rate was over 90% in the first survey in 1972 but fell over time.

From 1972 to 1992 diastolic blood pressure decreased by 8·6 mm Hg in men and 12·2 mm Hg in women. The standard deviation of diastolic blood pressure was about the same in 1972 as in 1992. The entire blood pressure distribution was shifted to the left. Total serum cholesterol decreased by 0·88 mmol/l in men and 1·18 mmol/l in women (table II). Among men, the prevalence of smoking decreased from 53% to 37% but in women it rose from 11% to 20%.

Age standardised mortality from stroke in men fell from 124/100 000 in 1972 to 50 in 1992. In women the corresponding decrease was from 80 to 35/100 000 (fig 1).

Table III shows the Cox's model calculated from the data on the cohorts. In men diastolic blood pressure, smoking, and serum cholesterol concentration all contributed to the risk of mortality from stroke. In women only diastolic blood pressure and smoking were statistically significant in predicting mortality from stroke.

Table IV and figure 2 show the observed fall in mortality from stroke from the mortality statistics, the predicted fall based on the proportional hazards model, and the observed changes in risk factors in men from 1972 to 1992. The predicted fall was 44% when all three risk factors were taken into account. Mortality from stroke actually fell by 66%. Mortality fell faster in the 1970s than in the early 1980s.

We estimated the relative importance of each risk

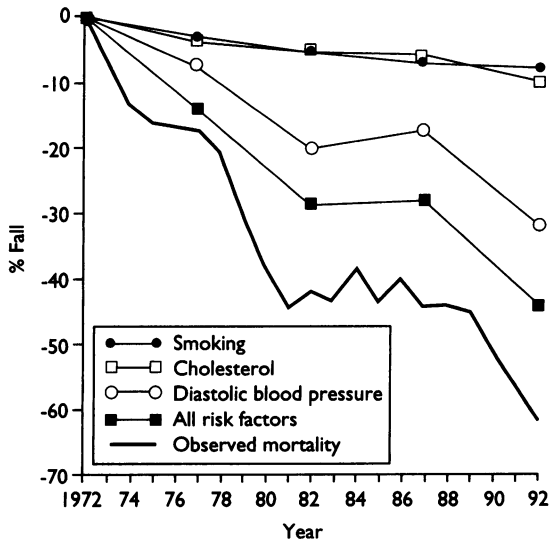

FIG 2—*Observed and predicted fall in mortality from stroke in men aged 35-64 years. Predicted fall is shown for each risk factor separately and together*

factor separately by changing one risk factor at a time in the model and keeping other risk factors at the 1972 level. In men the observed 8·6 mm Hg decrease in blood pressure predicted a 32% fall in mortality from stroke, the 0·88 mmol/l decrease in serum cholesterol predicted a 10% fall in mortality from stroke, and the observed decrease in smoking from 53% to 37% predicted a 8% fall in mortality from stroke.

In women the observed mortality from stroke fell by 63% (fig 3 and table IV). The 12·2 mm Hg observed decrease in diastolic blood pressure predicted a 38% fall in mortality from stroke and the increase in smoking observed from 11% to 20% predicted a 9% increase in mortality. When both of these changes in risk factors were included in the proportional hazard model, the fall in mortality predicted by the model was 34%. Serum cholesterol was not significantly associated with mortality from stroke in women and was not included in the model.

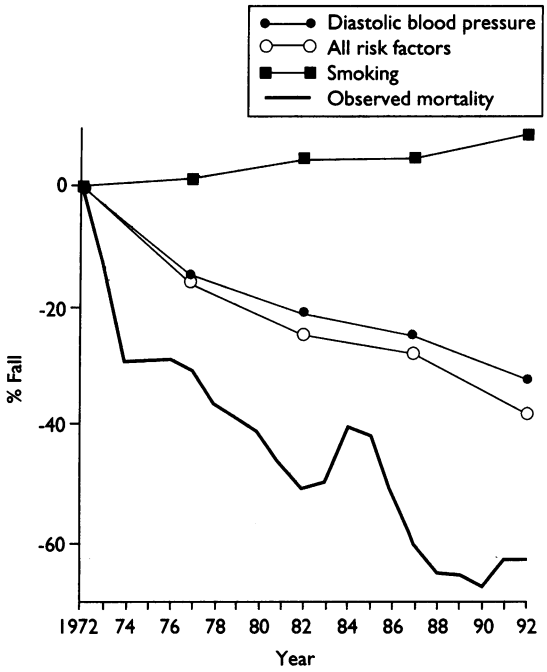

FIG 3—*Observed and predicted fall in mortality from stroke in women aged 35-64 years. Predicted fall is shown separately for each risk factor*

Discussion

We were able to predict 71% of the observed fall in mortality from stroke in men and 54% in women by changes observed in risk factors. About half of the fall was associated with the fall in diastolic blood pressure. Our estimates are based on one blood pressure measurement. If the regression-dilution bias in diastolic blood pressure in the Cox's model was corrected for by 60%, as proposed by MacMahon *et al*,[8] 86% of the observed fall in mortality from stroke in men and 84% in women was predicted. Most of the increase in smoking occurred in the youngest age groups in women, and this may not yet be affecting mortality from stroke. If smoking was not changed in the model the proportion of mortality from stroke in women that could be explained by the blood pressure change alone, taking into account the regression dilution bias, increased to 90%.

ACCURACY OF PREDICTIONS

It is important that changes in risk factors were measured accurately from 1972 to 1992. The comparability between our surveys was good for smoking as prevalence was assessed with the same questions in all surveys. It is unlikely that the reliability of self reporting of smoking has changed substantially during the study. The use of a longer cuff to measure blood pressure from 1982 onwards may have led to overestimation of the decrease in blood pressure in obese subjects. Shorter cuff size is known to overestimate blood pressure values, but this error is restricted to obese subjects with an arm circumference of more than 33 cm,[17,18] which represents only 5-10% of the population. Although the method of measuring serum cholesterol also changed in 1982, the difference between the two methods was quantified and corrected for.

Taking the proportional hazard model from the 1972, 1977, and 1982 cohorts separately, changing the follow up time, or pooling the cohorts together did not have any significant effect on predicted fall in mortality from stroke. We therefore decided to include all three cohorts to obtain as many stroke cases as possible for the prospective analyses.

The predicted mortality was calculated from the measured risk factor levels without assuming any lag time. Results from blood pressure trials suggest that the benefits of treatment to lower blood pressure on mortality from stroke occur quickly and that those of stopping smoking are seen in two years.[7,19] To estimate a lag time at the population level the risk factors should be measured while the levels are increasing in a population and also when they start to fall. Such data are not available.

Blood pressure, cholesterol concentration, and smoking accounted for 52%, 16%, and 13% respectively of the observed fall in mortality from stroke in men. The sum of these three proportions is 81% and not 71%, the fall which these risk factors explain together. This is because some of the deaths prevented by, say, the fall in smoking would have been prevented by the fall in blood pressure or cholesterol even if the prevalence of smoking had not changed.

CHANGES IN RISK FACTORS

Although serum cholesterol was not a powerful predictor of stroke in the Cox's model, it was the second most important risk factor predicting the fall in mortality from stroke in men because the change in serum cholesterol in Finland has been quite large. Most strokes occurring in middle aged men are cerebral infarctions.[20] In women cholesterol did not predict mortality from stroke and was not included in the analyses.

Antihypertensive drug treatment on a large scale was started in Finland in the 1970s. Drug treatment can explain only part of the decrease in the mean value of blood pressure since the whole blood pressure distribution has been shifted towards lower values. Salt intake has been high in Finland[21] but has fallen since the late 1970s by about 15%, which can partly explain the change in blood pressure. We have shown previously that blood pressure in this population can be reduced by increasing polyunsaturated fats in diet.[22] Use of saturated fats has decreased and the use of monounsaturated and polyunsaturated fats greatly increased in Finland, which may also partly explain the change in blood pressure.[23]

The steep fall in mortality from stroke observed in the 1970s levelled off during the early 1980s. During that period treatment of hypertension was less effective[24] and changes in levels of blood pressure, smoking, and serum cholesterol were small. From 1987 to 1992 the levels of these risk factors decreased more rapidly, except for smoking among women. The levelling off in mortality from stroke fitted well with the trends in risk factors.

The fall in mortality from stroke in the whole of Finland was about the same as in the two provinces studied here. Since Finland is a small country with a homogeneous population these two areas are probably

Key messages

- The reason for the fall in death rates from stroke in industrialised countries is unclear
- In this study blood pressure and smoking were independent risk factors for stroke in both sexes and serum cholesterol concentration was also a risk factor in men
- Mortality fell by 62% in men and 63% in women over 20 years
- Changes in risk factors explained 71% of the fall in men and 54% in women
- Continued emphasis on promoting healthier lifestyles and effective treatment for hypertension are essential to maintain the fall in deaths

representative of development in the entire country. It should not be taken for granted that the decreasing trend will automatically continue. Our data show that the fall will continue only if preventive measures targeted on the primary risk factors, particularly on blood pressure, are effective.

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Differences in mortality after fracture of hip: the East Anglian audit

C J Todd, C J Freeman, C Camilleri-Ferrante, C R Palmer, A Hyder, C E Laxton, M J Parker, B V Payne, N Rushton

Abstract

***Objective*—To investigate differences between hospitals in clinical management of patients admitted with fractured hip and to relate these to mortality at 90 days.**

***Design*—A prospective audit of process and outcome of care based on interviews with patients, abstraction from records with standard proforma, and follow up at three months. Data were analysed with χ^2 test and forward stepwise regression modelling of mortality.**

***Setting*—All eight hospitals in East Anglia with trauma orthopaedic departments.**

***Patients*—580 consecutive patients admitted for fracture of neck of femur.**

***Main outcome measure*—Mortality at 90 days.**

***Results*—Patients admitted to each hospital were similar with respect to age, sex, pre-existing illnesses, and activities of daily living before fracture. In all, 560 (97%) were treated surgically, by a range of grades of surgeon. Two hundred and sixty one patients (45%; range between hospitals 10-91%) received pharmaceutical thromboembolic prophylaxis, 502 (93%; 81-99%) perioperative antibiotic prophylaxis. The incidence of fatal pulmonary emboli differed between patients who received and those who did not receive prophylaxis against deep vein thrombosis (P=0·001). Mortality at 90 days was 18%, differing significantly between hospitals (5-24%). One hospital had significantly better survival than the others (odds ratio 0·14; 95% confidence interval 0·04-0·48; P=0·0016).**

***Conclusions*—No single factor or aspect of practice accounted for this protective effect. Lower mortality may be associated with the cumulative effects of several aspects of the organisation of treatment and the management of fracture of the hip, including thromboembolic pharmaceutical prophylaxis, antibiotic prophylaxis, and early mobilisation.**

Introduction

In the financial year 1990-1, 55748 people were admitted to hospital with a fracture of the neck of femur in England.[1] The incidence of such fractures has been increasing for several years and is predicted to continue to increase.[2] Fractures of the hip affect primarily women aged 65 years and over. Preferred treatment is normally surgical correction.[3] Patients with a hip fracture occupy about 20% of orthopaedic beds in England and Wales, and the average length of stay is 30 days.[2] Mortality at one year after fracture is 12-20% above that expected for the age-sex group, and most of this excess occurs within the first four months.

Correspondence to:
Dr Todd.

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