Optimal risk factors in the population: prognosis, prevalence, and secular trends

Data from Göteborg population studies

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Aims To assess the prognosis and prevalence of optimal risk factors in the population.

Methods and Results Data from several Göteborg population studies were used. Optimal risk factors were defined as serum cholesterol <5 mmol·l⁻¹, blood pressure <140/90 without treatment and being a non-smoker. In a 20-year follow-up of 7130 men aged 47 to 55 at baseline a group of 117 men who were optimal with respect to cholesterol, blood pressure and smoking were identified. In this group there was only one death from coronary disease, corresponding to 0·4 deaths per 1000 years, whereas the overall risk of coronary death in the study was 4·8 per 1000 years. Among men and women aged 25 to 34 in the Göteborg MONICA study 1995, less than half were optimal on all three scores, and in men and women aged 55 to 64, only 7% and 6%, respectively, were optimal. If body mass index below 25 was included only 34% and 37%, respectively, of men and women aged 25 to 34 were optimal, and 11% and 22% among men and women aged 35 to 44. In an analysis of secular trends over 30 years in four successive cohorts of men aged 50 the prevalence of optimal risk factors with respect to cholesterol, blood pressure and smoking increased from 1963 to 1993 but was still only 11% in 1993.

Conclusions As expected, optimal risk factors with respect to serum cholesterol, blood pressure and smoking confers a very low risk of coronary death. However, the prevalence of optimal risk factor status in the Swedish population is still low.

Key Words: Coronary disease, risk factors, smoking, cholesterol, blood pressure, population study.

See page 105 for the Editorial comment on this paper

Introduction

Coronary heart disease is largely preventable. The huge variations in this disease between populations as well as over time bear ample witness to this. As population characteristics change, so do coronary heart incidence and mortality. The geographical variation is well known[12]. A recent development is the increasing incidence in the developing countries[3]. Ischaemic heart disease is estimated to be the leading cause of lost life-years until at least 2020[4].

In the established market economies the last decades have seen important decreases in the incidence of myocardial infarction and coronary mortality. Swedish figures for coronary heart disease are in the middle range of the European countries[2], and the average annual decrease in coronary heart disease incidence between 1987 and 1996 in men and women aged 30 to 89 has been 2.2% and 1.8%, respectively[5]. Similar changes have been noted in men and women in Göteborg since the beginning of the 1980s[6]. Changes in the cardiovascular risk factor pattern seem to account for this[7–10]. The decrease in incidence in men below 65 years of age corresponds well to the decreasing levels of the three main risk factors[10]. However, rates of coronary disease in Sweden are still disturbingly high.

Comprehensive action for the prevention of coronary heart disease has to include a population strategy in addition to individual secondary prevention and primary prevention in high risk men and women. Risk factor goals for prevention in subjects with known coronary disease and in healthy high risk subjects are based on solid scientific evidence derived from a multitude of experimental and observational studies, which have been summarized in the second joint task force of European countries[11].
European and other societies on coronary prevention\[11\].

In this document, the ideal goals for prevention of coronary diseases and other atherosclerotic diseases have been defined as a diet compatible with ideal weight, and a plasma total cholesterol level of less than 5 mmol.l\(^{-1}\), regular exercise, the avoidance of all forms of tobacco, and a blood pressure of less than 140/90 mmHg. Levels of risk factors in various populations are known from a large number of studies\[8,9,12-19\], usually in terms of mean levels and prevalence of pre-defined high levels. However, the prevalence of optimal risk factors is rarely described. In Göteborg, prospective and cross-sectional studies of representative population samples have been carried out since 1963. In the present investigation, we used data from several of these samples to identify low risk subjects, and to describe prognosis, prevalence, and secular trends with respect to optimal risk factor status.

Methods

For the prospective part of the study, data were taken from the multifactor primary prevention study which started in Göteborg in 1970 and included all men in the city who had been born between 1915 and 1925, except those born in 1923\[20\]. The intervention group of 10 000 men comprised a random third of the men in the trial, with two control groups of 10 000 men each. At follow-up, after 11-8 years, there were no significant differences in risk factor levels or in outcome with respect to cardiovascular, cancer or all-cause mortality between the intervention or any of the control groups\[20\], and we consider the study group to be reasonably representative of the background population in the city. The present study deals only with the intervention group in the intervention group with complete data and no prior infarction or diabetes (n=7130 out of a total of 7495 participant men), aged 47 to 55 (mean 51) years at baseline. A first screening examination took place between January 1970 and March 1973. Data on smoking habits, diabetes, and history of myocardial infarction were collected by a postal questionnaire which was sent to all subjects along with an invitation to the study. Screening examinations were performed in the afternoon. Weight and height were measured. Blood pressure was measured to the nearest 2 mmHg after 5 min rest with the subject seated. Because blood pressure was generally high in the afternoon a systolic reading of 140 or of diastolic of 90 was included in the normotensive group. Serum cholesterol concentration (from a sample taken after fasting for at least 2 h) was determined according to standard laboratory procedures.

All subjects in the multifactor primary prevention study were followed until 31 December 1993 (mean 19-7 years). The Swedish national register on deaths due to specific causes from the years 1970 to 1993 was matched against a computer file of the men in the study. In 1987, there was a change from the 8th to the 9th revision of the International Classification of Diseases, but for the broad groupings used in the present study this will have made no difference. Coronary death was defined as ICD codes 410-414. During the first 11-8 years of the follow-up there were 329 first non-fatal myocardial infarctions registered by the Göteborg Myocardial Infarction Register in the study population\[20\]. As this register only records events in persons younger than 65 years of age there were no data concerning non-fatal events during the latter part of the follow-up.

The MONICA study population and survey methods have been described\[8\]. Random population samples of men and women aged 25 to 64 years were sent a postal questionnaire, and on returning the questionnaire they were invited to a screening examination in 1995. Participation rates were 64% and 63% for men and women, respectively. All examinations were done in the morning. Blood pressure was measured in the sitting position, after 5 min rest, with a random zero mercury sphygmomanometer. Weight and height as well as waist circumference were measured with the subject in light indoor clothing, without shoes. All blood samples were taken after an overnight fast. Serum cholesterol and triglycerides were determined according to WHO MONICA criteria. LDL cholesterol was calculated according to Friedewald’s formula. Systolic and diastolic (Korotkoff phase V) pressure was always measured before venipuncture according to the WHO MONICA standard after at least 5 min rest with the subject in the sitting position. Before the examination, all participants had completed a postal questionnaire dealing with smoking habits.

For the study of secular trends, data from the study of men born in 1913, 1923, 1933 and 1943 were used. Starting in 1963 and with ten year intervals, four population samples of men aged 50 and living in the city of Göteborg, Sweden, have been examined with respect to cardiovascular risk factors\[6\]. In 1963 all men born in 1913 on dates divisible by three were invited. Of the 973 invited men, 855 took part in the study (88%). For the 1973 study all men born in 1923 on the 3rd, 15th and 27th day of each month were invited, providing a sample of 292, of which 226 (77%) participated. For the 1983 and 1993 studies, a random sample of half of all men in the city born in 1933 and 1943 were invited; 776 (76%) and 798 (55%), respectively, took part\[9\]. All examinations were done in the morning after an overnight fast. Blood pressure measurements were done in the sitting position by a physician (except in 1983 when measurements were done by a nurse) using a standard cuff and a mercury manometer. Only Korotkoff phase 4 was available in 1963. In 1993 both phase 4 and phase 5 were registered. The mean difference (2 mm) was deducted from the 1963 diastolic readings when creating categorical blood pressure variables. Data on smoking habits were collected by a postal questionnaire. Fasting serum cholesterol and triglyceride measurements were determined according to standard laboratory procedures.
CHD = coronary heart disease; SBP/DBP = systolic/diastolic blood pressure.

or treatment, and smoker.

esterol >6

sure >160/95 or treatment) (4) High risk: total cholesterol >6 mmol . l

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5 mmol . l

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50 mmol . l

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49 mmol . l

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1 701 (10) 13 729 53 3·9 212 15·4

1 3231 (45) 63 166 384 6·1 1045 16·5

Blood pressure

<140/90, no treatment 2168 (30) 43 846 119 2·7 507 11·6

140–160/90–95, no treatment 1407 (20) 28 113 104 3·7 379 13·5

Treatment, or SBP >160, or DBP <95 3555 (50) 68 799 454 6·6 1265 18·4

Smoking habits at baseline

Non-smoker 3610 (51) 73 784 242 3·3 793 10·7

Smoker 3520 (49) 66 794 435 6·5 1358 20·3

Risk factor status at baseline

Optimal 117 (2) 2414 1 0·4 19 7·9

Low risk 894 (13) 18 720 30 1·6 124 6·6

Moderate risk 5310 (74) 104 958 497 4·7 1631 15·5

High risk 809 (11) 14 666 149 10·2 377 25·7

Relative risk by risk factor status at baseline

Age-adjusted

RR of CHD

95%

confidence interval

Age-adjusted

RR of death

95%

confidence interval

Optimal 0·09 (0·01–0·64) 0·51 (0·33–0·80)

Low risk 0·34 (0·24–0·49) 0·42 (0·35–0·51)

Moderate risk 1·00 — 1·00 —

High risk 2·22 (1·85–2·66) 1·72 (1·54–1·92)

All relationships between events/deaths and risk factors *P<0·001.

1Optimal: total cholesterol <5 mmol . l

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1 and blood pressure <140/90 without treatment, and non-smoker; Low risk: non-smoker and

(total cholesterol 5–6·5 mmol . l

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1 and/or blood pressure 141–160/91–95 without treatment); Moderate risk: smoker or (total cholesterol

≥6·5 mmol . l

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–

·

1 and/or blood pressure >160/95 or treatment); High risk: total cholesterol >6·5 mmol . l

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1 and blood pressure >160/95 or treatment, and smoker.

CHD = coronary heart disease; SBP/DBP = systolic/diastolic blood pressure.

Statistical methods

We used the SAS statistical package (version 6·12). In the prospective study, proportional hazards analyses were used to adjust for age. Four categories of risk were created: (1) optimal: total cholesterol <5 mmol . l

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1 and blood pressure <140/90 without treatment, and non-smoker; (2) Low risk: non-smoker and (total cholesterol 5–6·5 mmol . l

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1 and/or blood pressure 141–160/91–95 without treatment); (3) Moderate risk: smoker or (total cholesterol ≥6·5 mmol . l

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–

·

1 and/or blood pressure >160/95 or treatment) (4) High risk: total cholesterol >6·5 mmol . l

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1 and blood pressure >160/95 or treatment, and smoker. Three dummy variables were created, with moderate risk as a reference, as three quarters of the population in the primary prevention study were in that category. In the MONICA study simple correlation tests were used to assess relationships between age and continuous or graded variables. For the analysis of secular trends, the respective cohorts were assigned numbers from one to four and correlation tests were used to assess relationships between the cohort number and continuous variables. Mantel–Haenszel tests were used to assess the statistical significance of differences between the cohorts with respect to graded variables.

Results

In a first step we analysed the impact on coronary and total mortality of total serum cholesterol, blood pressure and smoking in 7130 men aged 47 to 55 (mean 51) years and followed for 20 years (Table 1). All relationships were in the expected direction and magnitude. A small group of 117 men (2% of the population) were characterized with the majority of the population, who were either smokers and/or had total cholesterol of ≥6·5 mmol . l

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1 and/or blood pressure 141–160/95 or treatment). In this group one coronary death occurred over 2414 observation years, corresponding to 0·4 deaths per 1000 years. The overall risk of coronary death over the 20-year follow-up was 4·8 deaths per 1000 observation years. In comparison with the majority of the population, who were either smokers and/or had total cholesterol of ≥6·5 mmol . l

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1 or above, and/or blood pressure >160/95 but not all three, the age-adjusted relative risk was 0·09, albeit with wide confidence intervals (0·01–0·64). The group of men with optimal risk factors also had low all-cause mortality.

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Table 2  Outcome with respect to fatal and non-fatal events according to serum cholesterol, smoking, and hypertension and combined risk factors in men examined in 1970–72 and followed until 1983

<table>
<thead>
<tr>
<th>Risk factor status at baseline</th>
<th>Number of men (% of total)</th>
<th>Observation years</th>
<th>CHD events (fatal and non-fatal) (n)</th>
<th>Per 1000 observation years</th>
<th>All deaths (n)</th>
<th>Per 1000 observation years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>117 (2)</td>
<td>1283</td>
<td>1</td>
<td>0.8</td>
<td>6</td>
<td>4.6</td>
</tr>
<tr>
<td>Low risk</td>
<td>894 (13)</td>
<td>9853</td>
<td>19</td>
<td>1.9</td>
<td>39</td>
<td>3.9</td>
</tr>
<tr>
<td>Moderate risk</td>
<td>5310 (74)</td>
<td>57243</td>
<td>316</td>
<td>5.5</td>
<td>480</td>
<td>8.2</td>
</tr>
<tr>
<td>High risk</td>
<td>809 (11)</td>
<td>8101</td>
<td>145</td>
<td>17.9</td>
<td>139</td>
<td>16.1</td>
</tr>
</tbody>
</table>

Relative risk by risk factor status at baseline

<table>
<thead>
<tr>
<th>Relative risk status at baseline1</th>
<th>Age-adjusted RR of CHD</th>
<th>95 per cent confidence interval</th>
<th>Age-adjusted RR of death</th>
<th>95 per cent confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>0.14</td>
<td>(0.02-1.02)</td>
<td>0.55</td>
<td>(0.25-1.23)</td>
</tr>
<tr>
<td>Low risk</td>
<td>0.37</td>
<td>(0.23-0.59)</td>
<td>0.50</td>
<td>(0.36-0.70)</td>
</tr>
<tr>
<td>Moderate risk</td>
<td>1.00</td>
<td></td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>High risk</td>
<td>3.08</td>
<td>(2.56-3.75)</td>
<td>1.94</td>
<td>(1.60-2.34)</td>
</tr>
</tbody>
</table>

1See legend to Table 1.

We also calculated the ten-year risk of a coronary heart disease event over the first 12 years of the study, where we had data on non-fatal events (Table 2). The ten-year risk of a coronary heart disease event in the low-risk group was 0.8%. In the group with the most adverse risk factor profile (smoker, blood pressure above 160/95 or treatment, serum cholesterol above 6.5 mmol l⁻¹) the ten-year risk was 17.9%, and in the total study group the ten-year risk was 6.1%.

Data from the 1995 MONICA survey show comparatively favourable risk factor levels in the youngest group, men and women aged 25 to 34 (Table 3), but as expected, overall risk factor profiles deteriorated with age in both men and women. At ages 35 to 44, less than half had serum cholesterol below 5 mmol l⁻¹. In the highest age group 18% of the men and 16% of the women had serum cholesterol below 5 mmol l⁻¹, and 39% and 46%, respectively, were normotensive. The majority at all ages were non-smokers.

If very strict criteria were used to define optimal risk factors (total cholesterol <5 mmol l⁻¹, LDL <3 mmol l⁻¹, HDL >1 mmol l⁻¹, triglyceride <2 mmol l⁻¹, waist <94 cm (women <80 cm), blood pressure <140/90 mmHg without treatment, non-smoker, normal weight (body mass index <25) few, even among the youngest age group, were optimal, 27% among the men and 35% among the women (Table 4). Among men and women aged 55 to 64 this combination was almost non-existent. Applying less rigid criteria, based on total cholesterol, blood pressure, smoking and body mass index only (total cholesterol <5 mmol l⁻¹, blood pressure <140/90 without treatment, non-smoker, normal weight (body mass index <25)) did not, however, yield very different results. With these less rigid criteria, 34% and 37%, respectively, of the youngest men and women were optimal, and 3% and 4% of men and women aged 55 to 64.

In a further step, we defined a set of optimal risk factors, based on serum cholesterol, systolic blood pressure and smoking only, without the weight criterion, in accordance with the risk charts[11]. Of the men and women aged 25–34, 46% and 47%, respectively, had total cholesterol <5 mmol l⁻¹, blood pressure <140/90 without treatment, and were non-smokers. Corresponding figures for men and women aged 55 to 64 were 7% and 6%.

In a final analysis (Table 5) we investigated the distribution of optimal risk factors over a period of 30 years in four cohorts of men who were 50 years when they were examined in 1963, 1973, 1983 and 1993. The proportion of men with serum cholesterol below 5 mmol l⁻¹ increased from 9%–22%, and the proportion of men who were normotensive (below 140/90 without treatment) increased from 45%–61%. In 1963 less than half of the men, or 44%, were non-smokers, whereas in 1993, 69% did not smoke. Very few men, or 2%, in 1963 had optimal levels of all three risk factors. In 1993, the proportion had increased, but was still only 11%. Rates of overweight and obesity increased over the period[9]. If normal body weight was added to the criteria only 6%, or 45 men overall, were optimal.

Discussion

In the prospective part of the study, we found essentially what we expected. An optimal risk factor profile carries a very low risk of coronary death and myocardial infarction. If this pattern were to prevail, this would mean pronounced changes in the panorama of disease. We also found, however, that even though the risk factor profile today is generally better than 30 years ago, optimal levels are still rare, particularly in the middle-aged and older population. Also, obesity is increasing[9], with associated metabolic disturbances and diabetes.
Table 3  Risk factors by age and sex in men and women aged 25 to 64 in the Göteborg MONICA study

<table>
<thead>
<tr>
<th>Age group</th>
<th>Men (n)</th>
<th>Women (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>25–34</td>
<td>(151)</td>
<td>(181)</td>
</tr>
<tr>
<td>35–44</td>
<td>(184)</td>
<td>(220)</td>
</tr>
<tr>
<td>45–54</td>
<td>(189)</td>
<td>(242)</td>
</tr>
<tr>
<td>55–64</td>
<td>(217)</td>
<td>(223)</td>
</tr>
</tbody>
</table>

### Men (n)

#### Total cholesterol, mmol. l⁻¹, mean (SD)
- <5.0 mmol. l⁻¹: 4.86 (0.89)
- 5.0–6.49 mmol. l⁻¹: 5.36 (1.08)
- ≥6.50 mmol. l⁻¹: 5.78 (1.11)

#### HDL cholesterol, mmol. l⁻¹, mean (SD)
- <2.0 mmol. l⁻¹: 3.01 (0.83)
- 2.0–4.99 mmol. l⁻¹: 3.38 (0.98)
- ≥5.0 mmol. l⁻¹: 3.73 (1.03)

### Women (n)

#### Total cholesterol, mmol. l⁻¹, mean (SD)
- <5.0 mmol. l⁻¹: 4.70 (0.89)
- 5.0–6.49 mmol. l⁻¹: 5.09 (0.91)
- ≥6.50 mmol. l⁻¹: 5.61 (1.08)

#### HDL cholesterol, mmol. l⁻¹, mean (SD)
- <2.0 mmol. l⁻¹: 1.23 (0.27)
- 2.0–4.99 mmol. l⁻¹: 1.23 (0.31)
- ≥5.0 mmol. l⁻¹: 1.27 (0.37)

### Systolic Blood Pressure (mmHg, mean (SD))
- <140/90, no treatment: 78 (9)
- 140-160/90-95, no treatment: 88 (11)
- Treatment, or SBP >160, or DBP >95: 142 (21)

### Diastolic Blood Pressure (mmHg, mean (SD))
- <90, no treatment: 72 (130)
- 90–119, no treatment: 11 (28)
- Treatment, or SBP >160, or DBP >95: 142 (21)

All relationships between age and risk factors P<0.001, except smoking in women (P=0.22) and HDL cholesterol (P=0.02 in men and P=0.03 in women).
Compared to the risk charts used in the European recommendations[11] the absolute risk in the men of the primary prevention study appeared to be lower. The risk charts were developed using Framingham data that may not be wholly applicable to Swedish populations. However, a 12-year follow-up study of middle-aged men and women from eastern Finland examined in the 1970s demonstrated risks that were not dramatically different from those found in the primary prevention study[21]. A recent study compared the Framingham risk model for coronary heart disease mortality rates with two national samples from the U.S. population and found a fairly accurate rank ordering while the prediction of absolute risk was less precise[22]. From a clinical point of view, prediction of absolute risk is essential, as treatment decisions will be based on the individual clinician’s conception of risk in an individual patient[23].

Limitations of the present study are that there were only single measurements of the relevant risk factors, and that risk factor status may have changed during the follow-up period.

### Table 4 Optimal risk factors by age and sex in men and women aged 25 to 64 in the Göteborg MONICA study. Figures are percentages (n)

<table>
<thead>
<tr>
<th>Age group</th>
<th>25–34</th>
<th>35–44</th>
<th>45–54</th>
<th>55–64</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% (n)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>All risk factors optimal</strong></td>
<td>27 (41)</td>
<td>10 (18)</td>
<td>3 (5)</td>
<td>2 (4)</td>
</tr>
<tr>
<td><strong>Optimal including body mass index &lt;25</strong></td>
<td>34 (51)</td>
<td>11 (21)</td>
<td>5 (9)</td>
<td>3 (7)</td>
</tr>
<tr>
<td><strong>Optimal</strong></td>
<td>46 (69)</td>
<td>23 (43)</td>
<td>10 (19)</td>
<td>7 (15)</td>
</tr>
<tr>
<td><strong>Low risk</strong></td>
<td>30 (45)</td>
<td>34 (62)</td>
<td>29 (54)</td>
<td>24 (52)</td>
</tr>
<tr>
<td><strong>Moderate risk</strong></td>
<td>25 (37)</td>
<td>42 (78)</td>
<td>60 (114)</td>
<td>67 (145)</td>
</tr>
<tr>
<td><strong>High risk</strong></td>
<td>0 (1)</td>
<td>1 (2)</td>
<td>1 (2)</td>
<td>2 (5)</td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% (n)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>All risk factors optimal</strong></td>
<td>35 (63)</td>
<td>19 (42)</td>
<td>12 (28)</td>
<td>3 (6)</td>
</tr>
<tr>
<td><strong>Optimal including body mass index &lt;25</strong></td>
<td>37 (67)</td>
<td>22 (49)</td>
<td>14 (34)</td>
<td>4 (9)</td>
</tr>
<tr>
<td><strong>Optimal</strong></td>
<td>47 (85)</td>
<td>30 (67)</td>
<td>21 (50)</td>
<td>6 (14)</td>
</tr>
<tr>
<td><strong>Low risk</strong></td>
<td>25 (46)</td>
<td>27 (60)</td>
<td>31 (75)</td>
<td>36 (80)</td>
</tr>
<tr>
<td><strong>Moderate risk</strong></td>
<td>28 (50)</td>
<td>42 (92)</td>
<td>48 (115)</td>
<td>55 (123)</td>
</tr>
<tr>
<td><strong>High risk</strong></td>
<td>0 (1)</td>
<td>1 (2)</td>
<td>1 (2)</td>
<td>3 (6)</td>
</tr>
</tbody>
</table>

1 All risk factors optimal: total cholesterol <5 mmol l⁻¹, LDL <3 mmol l⁻¹, HDL >1, triglyceride <2 mmol l⁻¹, waist 94 cm (80 cm in women), pressure <140/90 without treatment, non-smoker, normal weight (body mass index <25), no diabetes.

### Table 5 Distribution of risk factors 1963 to 1993 in 50-year old men in Göteborg

<table>
<thead>
<tr>
<th>Age group</th>
<th>25–34</th>
<th>35–44</th>
<th>45–54</th>
<th>55–64</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% (n)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total cholesterol, mmol l⁻¹, mean (SD)</strong></td>
<td>6·42 (1·12)</td>
<td>6·46 (1·34)</td>
<td>6·12 (1·12)</td>
<td>5·88 (1·04)</td>
</tr>
<tr>
<td>&lt;5·0 mmol l⁻¹</td>
<td>9 (81)</td>
<td>12 (26)</td>
<td>12 (130)</td>
<td>22 (177)</td>
</tr>
<tr>
<td>5·0–6·49 mmol l⁻¹</td>
<td>45 (382)</td>
<td>46 (103)</td>
<td>48 (367)</td>
<td>49 (386)</td>
</tr>
<tr>
<td>≥6·50 mmol l⁻¹</td>
<td>46 (392)</td>
<td>43 (96)</td>
<td>35 (268)</td>
<td>29 (231)</td>
</tr>
<tr>
<td><strong>Blood pressure, mmHg, mean (SD)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>138·2 (20·9)</td>
<td>137·4 (22·1)</td>
<td>134·0 (16·9)</td>
<td>128·7 (17·1)</td>
</tr>
<tr>
<td>Diastolic</td>
<td>89·6 (12·5)</td>
<td>89·9 (14·1)</td>
<td>86·2 (10·6)</td>
<td>84·4 (10·6)</td>
</tr>
<tr>
<td>% (n)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;140/90, no treatment</td>
<td>45 (386)</td>
<td>40 (90)</td>
<td>51 (388)</td>
<td>61 (482)</td>
</tr>
<tr>
<td>140-160/90-95, no treatment</td>
<td>26 (223)</td>
<td>22 (50)</td>
<td>22 (168)</td>
<td>19 (150)</td>
</tr>
<tr>
<td>Treatment, or SBP &gt;160, or DBP &gt;95</td>
<td>29 (246)</td>
<td>38 (85)</td>
<td>27 (209)</td>
<td>20 (162)</td>
</tr>
<tr>
<td>% (n) current non-smokers</td>
<td>44 (375)</td>
<td>47 (103)</td>
<td>63 (483)</td>
<td>69 (548)</td>
</tr>
<tr>
<td><strong>Risk factor status</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Optimal</strong></td>
<td>2 (17)</td>
<td>3 (6)</td>
<td>7 (51)</td>
<td>11 (84)</td>
</tr>
<tr>
<td><strong>Low risk</strong></td>
<td>30 (257)</td>
<td>28 (63)</td>
<td>28 (211)</td>
<td>33 (261)</td>
</tr>
<tr>
<td><strong>Moderate risk</strong></td>
<td>65 (552)</td>
<td>67 (151)</td>
<td>62 (471)</td>
<td>54 (432)</td>
</tr>
<tr>
<td><strong>High risk</strong></td>
<td>3 (29)</td>
<td>2 (5)</td>
<td>4 (32)</td>
<td>2 (17)</td>
</tr>
</tbody>
</table>

1Korotkooff phase IV.
2See legend to Table 1.

All trends P<0.001.

**Risk prediction in the Primary Prevention study**

Compared to the risk charts used in the European recommendations[11] the absolute risk in the men of the primary prevention study appeared to be lower. The risk charts were developed using Framingham data that may not be wholly applicable to Swedish populations. However, a 12-year follow-up study of middle-aged men and women from eastern Finland examined in the 1970s demonstrated risks that were not dramatically different from those found in the primary prevention study[21]. A recent study compared the Framingham risk model for coronary heart disease mortality rates with two national samples from the U.S. population and found a fairly accurate rank ordering while the prediction of absolute risk was less precise[22]. From a clinical point of view, prediction of absolute risk is essential, as treatment decisions will be based on the individual clinician’s conception of risk in an individual patient[23].

Limitations of the present study are that there were only single measurements of the relevant risk factors, and that risk factor status may have changed during...
follow-up, which could have attenuated associations. In particular, there were changes in smoking status\(^2\), but the approximately twofold increase in risk is similar to the relative risk of 1.7 found in the most recent coronary prediction model from Framingham\(^3\). Risk factor levels in the primary prevention study were generally high. Blood pressure levels were high, probably due to the fact that the screening sessions were conducted during the afternoon. As a consequence, the group with optimal risk factor levels identified was very small, 2% of the entire group. Nevertheless, in the cohorts born in 1913 and 1923, where the men were examined in the morning, the proportion of men with optimal risk factors was very similar, at about 2% to 3%. Measurements of several other relevant factors were not available in this study, which was designed in the late 1960s, such as serum triglycerides, HDL cholesterol, waist to hip ratio, plasma fibrinogen, insulin or blood glucose.

**Risk factors in the population**

In general, risk factor levels in the general population in Sweden are fairly high, from a global perspective. Among the MONICA populations, Göteborg ranked among the middle with respect to serum cholesterol, and smoking in men, but among the highest with respect to smoking in women\(^4\). Means of body mass index for both men and women were among the lowest among the MONICA populations. With respect to blood pressure, Göteborg ranked comparatively low, chiefly due to low pressure, among the youngest study participants\(^5\).

With the exception of the MONICA study, comparability with other studies is difficult, not only because methodology varies from study to study, but also because of different ages, and presentation of age adjusted means or proportions. Both serum cholesterol and blood pressure increased substantially with age. In the MONICA study, Göteborg had among the highest increases with age in men\(^6\). But increasing blood pressure with age may not be a natural phenomenon. In a study of a Tanzanian, a Brazilian and an Italian population, blood pressure was correlated with age only in the latter two\(^7\). In the African population, who consumed a low salt ‘fish and vegetable’ diet, blood pressure did not increase with age.

We have been able to identify few studies that describe the prevalence of optimal risk factors in the population, but this, however, is an area that is difficult to review fully because of the wealth of information available. One analysis, very similar in design to our study was done in the population of the Chicago Heart Association Detection Project in Industry\(^8\). Using cut-off levels which were only slightly different from those used in our study, 5% of the men and 7% of the women were found to have no risk factors, and a very low risk of coronary death over 22 years of follow-up, 1.3 and 0.7 per 1000, respectively, in men and women. Another sectional analysis of optimal risk factor status was carried out in a southern Korean population in 1990 to 1992\(^9\). Korea is a low risk country with respect to coronary disease but age-adjusted ischaemic heart disease mortality has increased dramatically since 1981 according to national statistics. About 60% of the men and women in the sample had serum cholesterol below 200 mg . dl\(^{-1}\) (5.2 mmol . l\(^{-1}\)). The prevalence of hypercholesterolaemia, defined as 240 mg . dl\(^{-1}\) (6.2 mmol . l\(^{-1}\)) or more, increased sharply with age in women but not in men. With a cut-off of 240 mg . dl\(^{-1}\) for serum cholesterol but otherwise the same criteria for normotension and smoking, more than four out of five Korean women aged 35–44, and 41% of those aged 55–59, had optimal risk factor status with respect to blood pressure, serum cholesterol and smoking. The estimate in Korean men was much lower, due to their very high rates of smoking.

Disturbingly, but concomitant with the high rates of coronary heart disease in Sweden, the oldest cohort in Göteborg, those 55–64 years old, comprised very few subjects with optimal risk factor status, even if only serum cholesterol, blood pressure and smoking habit were used to define optimal risk factors. If ideal levels of body mass index were included to define optimal risk factor status, this group dwindled to almost nothing, or 3%–4%. In the youngest group aged 25 to 44, only about one third had optimal risk factor status. In the next age stratum, men and women aged 35–44, only 11% of the men and 22% of the women had ideal risk factor status.

**Secular trends**

With respect to serum cholesterol, smoking and hypertension decreasing trends were seen for all three during the period 1963–1993 in men\(^10\). Several studies have reported declines in population levels of serum cholesterol\(^11\–\(^13\)) usually against a background of decreasing incidence of coronary disease. In eastern Finland the rate of death from coronary disease has decreased to less than half of its previous level\(^14\) and about half of the observed decrease in mortality rate could be explained by a decrease in cholesterol level in the population. As the atherosclerotic process starts at a fairly young age, the contribution of a decrease in serum cholesterol levels to the decline in coronary mortality rate may be even greater than that estimated from measurements of cholesterol in middle age\(^15\). The most likely explanation for the decrease in serum cholesterol levels in Sweden, as well as in other countries, is dietary changes, such as the introduction of low-fat dairy products and oil-based soft margarine. Decreasing levels of hypertension have also been reported from a large number of MONICA populations\(^16\–\(^18\)).

In Göteborg, coronary heart disease incidence in middle-aged men has decreased 30%–40% since 1975\(^19\), concomitant with similar decreases in risk factor status\(^20\), and similar decreases have been reported with respect to mortality on a national level\(^21\). However, official statistics show that the decrease in incidence of
smoking must be a health policy priority. Lipids and blood pressure as well as to a reversal in the intervention aimed at shifts in the distribution of blood factors is useful and distinguishes between people at moderate risk this will in heart disease should be targeted for intervention by the healthy individuals at high risk of developing coronary disease in the population only to a comparably small extent. In order to achieve important reductions of coronary disease in the population only to a comparatively small extent. In order to achieve important reductions in coronary heart disease incidence and mortality, intervention aimed at shifts in the distribution of blood lipids and blood pressure as well as to a reversal in the trend in increasing obesity in the population and less smoking must be a health policy priority.

From a clinical point of view, the concept of risk factors is useful and distinguishes between people at high and at low risk so that the most intensive intervention may be used in those in greatest need of reduction in risk. Patients with established coronary disease and healthy individuals at high risk of developing coronary heart disease should be targeted for intervention by the medical profession. However, as the majority of events occur in persons at moderate risk this will influence rates of coronary disease in the population only to a comparatively small extent. In order to achieve important reductions in coronary heart disease incidence and mortality, intervention aimed at shifts in the distribution of blood lipids and blood pressure as well as to a reversal in the trend in increasing obesity in the population and less smoking must be a health policy priority.

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References


