Patients, families and populations at high risk for coronary heart disease

M. Higgins

Departments of Epidemiology and Internal Medicine, University of Michigan, Michigan, USA

Introduction

High risk for coronary heart disease may be defined and assessed in terms of absolute, relative or attributable risk. The ranges of absolute and relative risks for individuals extend from very low to very high, depending on the absence or presence of single or multiple risk factors, pre-clinical or manifest disease. Risk also varies among families and populations. A general goal is to match the intensity of intervention to the level of absolute risk\(^\text{[1]}\). But the number of people at low or moderate risk exceeds the number at high risk and also influences attributable risk for populations. Consideration of benefits and costs of intervening is important, in relation to efficacy of treatment, risks of adverse side effects and financial costs. However, even when short-term risk is low, for example among children and young adults, it is important to make the most of opportunities to reduce risk of coronary heart disease later in life, especially for those whose relative risks are high. Approaches to reducing risk, which emphasize healthy lifestyles, are appropriate for populations and families as well as for patients and high risk individuals. Implementation of these approaches would reduce risk attributable to unhealthy behaviours.

High risk patients

Patients who have had a heart attack or in whom coronary heart disease, cardiovascular disease, diabetes, hyperlipidaemia, or hypertension have been diagnosed are well known to be at very high risk and are candidates for secondary or primary prevention of coronary heart disease. Since they are under medical care, they should be well placed to receive appropriate, efficacious medical or surgical treatments and guidance about heart-healthy behaviours and lifestyles. However, not all such patients receive appropriate medical care, and many patients with these conditions are not diagnosed\(^\text{[2]}\). Similarly, although major risk factors for coronary heart disease are well known, many apparently healthy people are unaware of their high risk status or are not benefiting from established, effective interventions.

Several risk appraisal instruments are in use; they provide quantitative or qualitative estimates of the probability of an event in the next few years. Most of them apply to people who do not have diagnosed coronary heart disease and many of them are based on experience in the Framingham Heart Study. A recent publication from the Framingham Heart Study provided information about risks for coronary heart disease in patients who have survived a coronary heart disease event or stroke as well as for individuals who are free of cardiovascular disease (Table 1)\(^\text{[3]}\). The associations of age, sex, dyslipidaemia, hypertension, diabetes, smoking and other risk factors with incidence of coronary heart disease in apparently healthy people are well known, but similar associations in patients with prevalent cardiovascular disease have received less attention. The latest Framingham risk models also assign points for some additional risk factors, including menopausal status, triglycerides and, inversely, for alcohol use. To estimate risk, measurements of cholesterol, other lipids, blood pressure, glucose tolerance, body weight and height, may be needed as well as information on prevalent cardiovascular disease, diabetes, smoking habits, and family history. Some instruments give credit for protective factors such as physical activity, use of alcohol, hormone replacement therapy, and aspirin.

In practice, some tables such as the Sheffield tables and the European or British Societies risk prediction tables are simple to use\(^\text{[4–7]}\). They are guides for
screening, risk assessment and treatment. Recommendations for cholesterol measurement are based on preliminary assessment of absolute risk of coronary death considering age, sex, and the presence of other risk factors. In the U.S. the National Cholesterol Education Program (NCEP) follows the recommendations of the Second Adult Treatment Panel[8]. Measurement of cholesterol is advocated for all adults and indications for making additional lipid measurements are given. Treatment guidelines are based on the extent and type of lipid abnormalities and the presence or absence of other risk factors. Whether cholesterol should be measured in all adults, or only in those whose global level of risk is potentially above some cut point, e.g. 15% or 30% over the next 10 years, is controversial. A recent publication using results of the Air Force/Texas Coronary Atherosclerosis Prevention Study (AFCAPS/TexCAPS) showed that first acute major coronary events were reduced by lovastatin in individuals who did not meet either European or NCEP guidelines for treatment, as well as in those who did[54]. Given the low cost of cholesterol measurement, public awareness and interest in cholesterol, and the availability of effective therapy, its measurement in all adults is justified, in the author’s opinion[8].

There is general agreement that blood pressure should be measured and elevated levels treated. On the other hand, electrocardiograms to detect left ventricular hypertrophy, and non-invasive imaging of carotid arteries or electron beam computed tomography to detect calcification of coronary arteries should be used only when there are medical indications, or research objectives. Guidelines for detecting and treating risk factors should be reevaluated and, if necessary, revised as the science on which they are based advances. Medical priorities, economic and political influences also bear on recommendations for reducing coronary heart disease risk and are discussed below.

Many new risk or protective factors have been suggested and are being investigated. They include genetic polymorphisms, lipids, homocysteine, dietary constituents and supplements, markers of inflammation, psychosocial factors, evidence of endothelial dysfunction and pre-clinical atherosclerosis. Recently, C reactive protein and homocysteine levels have been reported to predict coronary heart disease as well as or better than cholesterol[9–12].

The efficacy of lipid lowering therapies to reduce morbidity and mortality from coronary heart disease has been established in randomized controlled clinical trials, some of which are shown in Table 2 (Gotto AM, personal communication)[6–13]. There is convincing evidence that statins are useful for secondary and primary prevention of coronary heart disease in men, women, the elderly, and diabetics and in people with elevated and so-called ‘normal’ levels of total or LDL cholesterol, or low levels of HDL cholesterol. Clinical trials have also demonstrated that antihypertensive drugs reduce coronary heart disease and stroke events[14,15]. In addition to drug treatments, healthy lifestyles, including nutritious diets and physical activity, avoidance of smoking and overweight, have been shown to result in lower rates

### Table 1 Coronary risk appraisal: Framingham study.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Men First</th>
<th>Men Subsequent</th>
<th>Women First</th>
<th>Women Subsequent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Total C/HDL-C</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>BP Rx × BP</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Diabetes</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Cigarette Smoking</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Menopause</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age × Menopause</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triglycerides</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol (neg)</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


### Table 2 Recent prevention trials

<table>
<thead>
<tr>
<th>Trial</th>
<th>Intervention</th>
<th>Type</th>
<th>Reduction in LDL-C (%)</th>
<th>Increase in HDL-C (%)</th>
<th>Reduction in cardiovascular events (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AFCAPS/TexCAPS</td>
<td>Lovastatin</td>
<td>Primary</td>
<td>25</td>
<td>6</td>
<td>37</td>
</tr>
<tr>
<td>WOSCOPS</td>
<td>Pravastatin</td>
<td>Primary</td>
<td>26</td>
<td>5</td>
<td>31</td>
</tr>
<tr>
<td>CARE</td>
<td>Pravastatin</td>
<td>Secondary</td>
<td>32</td>
<td>5</td>
<td>24</td>
</tr>
<tr>
<td>VA-HIT</td>
<td>Gemfibrozil</td>
<td>Secondary</td>
<td>25</td>
<td>8</td>
<td>22</td>
</tr>
<tr>
<td>LIPID</td>
<td>Pravastatin</td>
<td>Secondary</td>
<td>25</td>
<td>5</td>
<td>24</td>
</tr>
<tr>
<td>4S</td>
<td>Simvastatin</td>
<td>Secondary</td>
<td>35</td>
<td>8</td>
<td>34</td>
</tr>
</tbody>
</table>

AFCAPS/TexCAPS=Airforce/Texas Coronary Atherosclerosis Prevention Study; WOSCOPS=West of Scotland Coronary Prevention Study; CARE=Cholesterol And Recurrent Events trial; VA-HIT=Veterans Administration—High Density Lipoprotein Intervention Trial; LIPID=Long-term Intervention with Pravastatin in Ischemic Disease; 4S=The Scandinavian Simvastatin Survival Study. Reproduced with permission of Gotto AM.
of coronary heart disease and regression of lesions in patients and high risk individuals\[16,17\]. The Lyon Diet Heart Study found that event free survival was improved in survivors of a first myocardial infarction who ate a Mediterranean-type diet compared with controls who were advised to follow a prudent diet\[19\]. Clinical trials that are now underway should provide evidence for or against the use of hormone replacement therapy, antioxidants, dietary supplements and other pharmacological and non-pharmacological treatments.

Advances in pharmaco-genetics are expected to allow targeting of therapies to responsive patients. Sodium restriction and weight loss were associated with greater blood pressure lowering in subjects with angiotensinogen genotype AA\[19\]. Response to statins has been reported to vary with apolipoprotein E and CETP genotypes\[20,21\]. Clinical trials have also identified interventions which are not beneficial or which may even be harmful to some people. An example of an unsuccessful treatment is a randomized controlled trial of stress management, which failed to lower blood pressure in the Trial of Hypertension Prevention\[22\]. Recent reports have indicated that Vitamin E did not reduce events in patients with cardiovascular disease or in high risk individuals without coronary heart disease\[23\]. Adverse effects of hormone replacement therapy have occurred in recent trials of women, calling into question the reduction in coronary heart disease attributed to oestrogen use in observational studies\[24\].

Even though a great deal is known about detecting and reducing risk, there are problems in making full use of this knowledge. There is also some controversy about implementing what we know for individuals at moderate levels of risk. Costs and benefits of following NCEP guidelines for treatment with lipid lowering drugs have been assessed\[25\]. Secondary prevention is much more cost effective than primary prevention as fewer patients will have to be treated to prevent many more events. Primary prevention is more cost effective if it targets high risk rather than medium risk individuals. Nevertheless opportunities to reduce risk will be missed if safe and effective treatments are applied too narrowly.

Some challenges to taking full advantage of existing opportunities to reduce coronary heart disease risk, morbidity and mortality are related to problems in medical care, such as failure to detect high risk patients with coronary heart disease, associated diseases or risk factors, and failure to manage patients and high risk individuals optimally. Appropriate treatments may not be prescribed, and patients do not always accept treatments or follow recommendations for changes in behaviour. In the U.S., access to medical care is not universal, and quality of care is variable. A recent report has drawn attention to medical mistakes, which are said to outnumber motor vehicle accidents, breast cancer or AIDS deaths in the U.S. (44 000-98 000 deaths per year)\[26\]. This estimate has been challenged\[27\] and defended\[28\].

Financial and medical resources vary at individual and community levels and competing needs may not support prevention of coronary heart disease as the highest priority. However, targeting treatments to susceptible individuals should yield improvements in the balance of both health and economic benefits and costs.

The emphasis that is being given currently to identifying, diagnosing, and improving treatments for patients and high risk individuals can be expected to result in further reductions in coronary heart disease mortality and morbidity. Primary prevention of risk factors through adoption of healthy lifestyles is also focusing on individuals. Many people have responded to information about diet, smoking, exercise and weight control and have adopted healthy lifestyles\[29\]. Thus there can be no doubt that attention to estimating and reducing risk in individuals has contributed and will continue to contribute to the declines in coronary heart disease morbidity and mortality. But I believe progress could be enhanced if more attention were given to high-risk families and populations.

**High risk families**

We have known for a long time that coronary heart disease and many risk factors cluster in families. One of the major hypotheses of the Tecumseh Community Health Study, initiated over 40 years ago by Thomas Francis and directed for many years by Fred Epstein, was that new onsets of disease will occur with increased frequency in families of affected individuals whether disease is due to genetic, or environmental factors, or the interaction of the two\[30\]. A family is defined in The Dictionary of Epidemiology as ‘two or more people united by blood, marital, or adoptive ties’\[31\]. Members of families may or may not live in the same household, and of course, the definition, structure and function of a family change over time. This makes the family a unique resource for investigating the relationships of nature and nurture to health and disease; the family also provides unique opportunities for promoting health and preventing disease.

A positive family history is recognized as an independent risk factor for coronary heart disease, but categorizing a family history as positive or negative is not as informative as a quantitative family risk score which is based on information on the size, age and sex composition of a family as well as age at onset of coronary heart disease in affected members. The score compares observed or reported experience of coronary heart disease with the experience expected for first-degree relatives in a family. The expected experience may be determined from coronary heart disease incidence rates by age and sex in the population to which the family belongs, or from some comparable population for which such data are available\[32,33\]. Family risk scores were calculated in the NHLBI Family Heart Study and in other studies, and used to rank families according to level of risk\[34\]. This risk score applies to the whole family, but it can be modified to represent risk for each individual member by excluding his or her own coronary heart disease history.
Even if a quantitative score is not calculated, it is useful for medical practitioners to know that risk scores increase as the number of affected first degree relatives increases, and the score is higher the earlier the age at onset of coronary heart disease. Families with several surviving affected members are rare in the general population but families with elevated risks are more common.

The contribution of genetics to risk factor resemblance among family members can be inferred and heritability estimates can be calculated from correlations of variables among different types of relatives. Correlations of biomedical variables were higher among first-degree blood relatives than between spouses in the Family Heart Study. Their magnitude varied from about 0.2 for blood pressure, triglycerides, insulin, PAI-1, and fibrinogen, to 0.4 for lipoprotein A and 0.5 for height; they were intermediate for body mass index, total and LDL- and HDL-cholesterol. Maximum heritability estimates, equal to twice the correlations among first-degree relatives, express the extent to which phenotypic variation in a population is the result of genetic variation; they estimate the degree to which a trait is genetically or culturally determined within the family. However, assortative mating between spouses, such as occurs for height and education, influences these correlations. Familial correlations of behavioural risk factors showed a different pattern. For smoking, alcohol use, exercise, and dietary fat intake they were higher between spouses than among first-degree blood relatives.

Numerous studies have shown a strong resemblance in smoking habits between parents and children, and spouses and among siblings. In the Family Heart Study population consisting of adults aged 25 and over, only spouses were likely to be living, eating and exercising together. Familial clustering of some behaviours is greater when children are younger.

Because extensive data indicate that coronary heart disease and its risk factors aggregate in families, some families qualify for intensive intervention. Dyslipidaemias are very common in blood relatives of patients with early onset coronary heart disease, the most frequent were high lipoprotein (a), dyslipidaemia or familial combined hypercholesterolaemia. However, there was no identifiable familial lipid abnormality in nearly half of the relatives, and dyslipidaemias were rare among spouses.

The MED-PED programme (MEDical intervention to prevent early death in PEDiges) acts on the known genetic aetiology of familial hypercholesterolaemia and other inherited forms of high cholesterol and high blood pressure, by registering patients and recruiting their blood relatives for screening, and treatment, if necessary. In the U.S., this programme has identified about four cases of previously unrecognized hypercholesterolaemia for each known case. The programme is an international cooperative programme in about 30 countries, coordinated by the World Health Organization.

Relatives of patients with coronary heart disease and with familial dyslipidaemias have been responsive to invitations for screening, case finding, and treatment, if indicated. There is some evidence that intervention in families can be successful. The Tromso Family Study is one such example. In this study, high-risk men without coronary heart disease and their families were randomly assigned to a control group or to a group given advice on diet, smoking, and exercise. Six years later, men in the intervention group had significantly greater reductions in serum cholesterol and triglyceride levels, a composite risk score, and the percentage with more than one risk factor, and they gained less weight. Some risk factors were also lower in the wives of intervention group men, and dietary habits were better in these men, their wives and children. Unfortunately, smoking rates and physical activity were not different in the two sets of families. It should be noted that the intervention focused on the men, while less attention was given to other members of the family. The authors concluded that ‘a family approach to intervention is well accepted and feasible for use in primary care’.

Middle-aged couples were the focus of The British Family Heart Study, which included a 1 year randomized screening and lifestyle intervention trial in primary care centres in 13 towns. Changes in risk factors were greater in participants in intervention than control practices. Blood pressures, cigarette smoking, weight, cholesterol and the Dundee coronary risk score all improved further and the improvements were greatest in those with the highest levels initially. Risk factor levels and changes were correlated between partners. The authors concluded, ‘it is likely that lifestyle interventions targeted at . . . couples may result in a greater reduction in cardiovascular risk factors, possibly through mutual reinforcement of lifestyle changes’.

Because atherosclerosis begins at an early age, and we believe that healthy behaviours are best acquired in youth, the family should be a target for primary prevention and prevention efforts should be targeted to families with young children as well as to high-risk families. Nevertheless attempts to estimate risk and prevent coronary heart disease in families are rare, and there are problems in dealing with families. These include difficulties in recruiting complete families, and ethical, legal and social issues, especially if genetic studies are involved. Socioeconomic barriers to participation and compliance are magnified when several individuals are involved. Some problems are inherent in the clinical characteristics of coronary heart disease. Case fatality rates are high, therefore affected relatives may be dead. Family histories may be inaccurate or incomplete, diagnoses and risk factor status of relatives may not be known. Changes over time in diagnosis, treatment, prevalence of risk factors, and mortality confound comparisons across generations.

Nevertheless families are an appropriate target for risk assessment, case finding and prevention. Population strategies consisting of advice about healthy diets, physical activity, weight control and smoking avoidance are appropriate for all families and all their members. Pharmacological and other medical treatments are appropriate for those with coronary heart disease or at high risk for developing it. Benefits and costs of familial
approaches should be measured and compared with similar approaches directed to individuals. It is likely that the burden of coronary heart disease, including the development of risk factors in the first place, could be reduced if more attention were given to characterizing good and bad family environments, and to reducing risks in families.

**Populations at high risk of coronary heart disease**

A difficulty in making prognozes for patients and families is that estimates of risk apply to groups of people, not to individuals. For example, if the probability of having a heart attack in the next ten years is 30%, then 70% of people with similar risk estimates will not have had a heart attack at the end of ten years. Sherlock Holmes expressed the situation as follows ‘... while the individual man is an insoluble puzzle, in the aggregate he becomes a mathematical certainty. You can, for example never foretell what any one man will do, but you can say with precision what an average number will be up to’\(^{[42]}\).

Although this statement is not an accurate reflection of our ability to estimate probabilities of future coronary heart disease, it is relevant to the contrast between estimates of probability for individuals versus populations. Vital and health statistics show that mortality, morbidity and risk factor levels vary within narrow limits from year to year within a population. However, they differ substantially among populations and within subgroups of populations. Moreover trends over time have been beneficial in some populations and in some subgroups, whereas others are experiencing higher coronary heart disease rates. Gaps between high risk and low risk subgroups have widened\(^{[43]}\).

National mortality statistics show that coronary heart disease death rates are very high in countries formerly in the USSR, and in Eastern Europe; they are intermediate in northern Europe and America and low in Southern Europe Japan and China (Fig. 1). Recent publications from the WHO Monica Project have shown that mortality, incidence rates, and risk factor prevalence rates differ substantially among the 38 participating countries, which used standardized definitions and methods\(^{[44]}\).

The principal goals of this study were to investigate relationships between trends in risk factors and trends in mortality and coronary heart disease events, and to relate trends in medical care to trends in mortality and case fatality. Coronary heart disease event rates declined in most, but not all countries and trends in most risk factors were in the right direction. However, body mass index generally increased in both sexes and smoking habits improved less in women than in men. Associations between trends in risk factors and trends in coronary heart disease events were statistically significant, but weak; they were stronger in men than women, and were better when allowance was made for a four-year lag period\(^{[45]}\).

Trends in medical care were more strongly related to trends in coronary heart disease mortality, case fatality, and event rates. Trends in a treatment score explained...
52% of the variance in event rates among men, and 30% among women[46]. Two-thirds of the decline in coronary heart disease mortality was attributed to the decline in event rates and one third to the decline in case fatality. Changes in coronary care and secondary prevention were strongly linked to the decline in coronary heart disease end-points. Possible explanations for these patterns, other than that advances in medical care have had a greater impact than improvements in lifestyle-related risk factors, include the possibility that lag times are greater for primary prevention and that measurement of behavioural risk factors is less precise than measurement of biomedical variables. Analyses of the contributions of lifestyle changes and medical interventions to the decline in coronary heart disease mortality in the U.S. indicate that the two sets of factors contributed about equally to the decline between 1968 and 1976, but that medical care has been responsible for a larger proportion of the decline between 1980 and 1990[47]. It is clear that personal behaviours and medical care influence coronary heart disease mortality, morbidity, and risk factors, but their relative importance may vary over time, from place to place and among subgroups within populations.

Death rates and trends in death rates over time differ among subgroups defined by sex, age, socio-economic circumstances and racial or ethnic group in the U.S. Risk factor distributions and trends also differ substantially. Smoking, hypertension, and cholesterol levels have generally improved, whereas overweight has increased. High blood pressure affects the black population disproportionately and overweight and obesity are more prevalent in black and Hispanic women[48]. High blood pressure, obesity, cigarette smoking and physical inactivity are more prevalent among people with less formal education in the U.S. (Fig. 2). Patterns are similar if income is used to subdivide the population, and differences between the black and white populations are partially accounted for by differences in education and income among the racial groups. Changes over time have accentuated many of these differences[48]. Availability, quality and use of medical care as well as individual health and behavioural risk characteristics vary among and within populations and are reflected in different morbidity, and mortality rates.

Individual and familial coronary heart disease-related characteristics aggregate in communities, but in addition certain characteristics of communities themselves have been reported to influence coronary heart disease risk. Ecological characteristics include characteristics of the biological, social and physical environment and aspects of health care (Table 3). Some of them, such as gene frequencies, mean income or mean level of education are derived from data for individuals in a community but some ecological factors may only be measurable at the level of the community. Examples include unemployment or population mobility, climate, temperature variability, recreational facilities, and number of cardiologists or hospital beds per unit of population. One population genetic characteristic which has been related

![Figure 2 Prevalence of risk factors by years of education.](image_url)

**Table 3 Ecological risk factors**

<table>
<thead>
<tr>
<th>Social environment</th>
<th>Physical environment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unemployment rate</td>
<td>Climate</td>
</tr>
<tr>
<td>Income/income disparity</td>
<td>Altitude</td>
</tr>
<tr>
<td>Education</td>
<td>Water hardness</td>
</tr>
<tr>
<td>Population mobility</td>
<td>Recreational facilities</td>
</tr>
<tr>
<td>Social support</td>
<td>Occupation</td>
</tr>
<tr>
<td></td>
<td>Pollution</td>
</tr>
<tr>
<td></td>
<td>Housing</td>
</tr>
<tr>
<td>Biological environment</td>
<td>Health care</td>
</tr>
<tr>
<td>Gene/allele frequencies</td>
<td>Physicians, other professionals</td>
</tr>
<tr>
<td>Food supply</td>
<td>Medical facilities</td>
</tr>
<tr>
<td>Infectious agents</td>
<td>Quality of care</td>
</tr>
<tr>
<td></td>
<td>Attitudes and practices</td>
</tr>
</tbody>
</table>
to higher coronary heart disease mortality is the frequency of the apolipoprotein E epsilon 4 allele\(^{(49)}\). In a population of elderly Finnish men there was also an interaction between traditional risk factors in individuals and the distribution of apolipoprotein E genotypes in communities\(^{(49)}\). Another ecological characteristic reported to be associated with higher cardiovascular disease mortality is income inequality; this association is in addition to that with black–white segregation, which is itself a marker for differences in education, income and other social factors (Lynch J, personal communication).

Interpretation of ecological associations is always difficult. Ecological risk factors may have an impact that reflects or varies with individuals’ characteristics, or they may have an independent effect. For example, individuals’ smoking habits as well as community attitudes and regulations on smoking influence exposure. Individuals’ health practices as well as availability and quality of medical resources in the community influence preventive and therapeutic care. Lower coronary heart disease risks enjoyed by well-educated and more affluent people are attributable to many behaviours, exposures and lifestyle factors as well as to availability, quality and use of medical care.

It has been suggested that the background level of risk in the community might influence treatment of individuals\(^{(51)}\). Mortality from coronary heart disease has been shown to vary at any given level of blood pressure in different countries. It is three times higher in Northern Europe and the U.S. than in Japan and Southern Europe\(^{(51)}\). Nevertheless, evidence that morbidity and mortality are reduced by treating high blood pressure with safe and inexpensive drugs is overwhelming. The suggestion that the blood pressure level at which treatment is to be initiated may vary according to the background risk for hypertensive events, must be weighed against this evidence and take account of the prevalence of other risk factors as well as risks for other cardiovascular diseases, including stroke, prevailing in the community in which the patient lives. Medical conditions in individual patients as well as doctor–patient preferences must also be considered.

Decisions about national, community and individual interventions are likely to be influenced by educational, economic and political factors as well as by scientific evidence, medical and other priorities. Community interventions are rare and their impact in the U.S. has been disappointing\(^{(52)}\). On the other hand, the success of the North Karelia Project, some school-based programmes and national education programmes has provided support for such approaches to reducing risk of coronary heart disease. Community interventions face many more difficulties in design, implementation and evaluation than is the case for studies in individuals or even in families. A model known as the REAIM model has been proposed for evaluating them\(^{(53)}\). The dimensions to be scored are: reach, efficacy, adoption, implementation and maintenance. Of two hypothetical programmes, one may reach a high percentage of individuals and be adopted, implemented effectively and

<table>
<thead>
<tr>
<th>Condition</th>
<th>Baseline</th>
<th>Target</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD deaths per 100 000</td>
<td>208</td>
<td>166</td>
</tr>
<tr>
<td>High blood pressure %</td>
<td>28</td>
<td>16</td>
</tr>
<tr>
<td>Hypertensives controlled %</td>
<td>18</td>
<td>50</td>
</tr>
<tr>
<td>High total cholesterol %</td>
<td>21</td>
<td>17</td>
</tr>
<tr>
<td>Obesity %</td>
<td>23</td>
<td>15</td>
</tr>
</tbody>
</table>

Table 4 Healthy People 2010 Objectives

Age-adjusted to year 2000 standard population. CHD=coronary heart disease.

1998

maintained by a fairly large proportion of the schools, work sites or communities for which it is appropriate, but it may score lower on efficacy because biological, behavioural or quality of life effects of the intervention on individuals are not as good as those with an alternative high cost–high efficacy intervention. However, the latter intervention may not reach as high a percentage of participants and it may not be adopted, implemented, or maintained as well. The weights to be given to each component of the REAIM score may not be equal and they will vary from intervention to intervention and among populations, but the goal of considering several dimensions and developing objective evidence for benefits and costs of alternative population-based interventions is commendable.

National goals and objectives for improving cardiovascular health and quality of life through the prevention, detection and treatment of risk factors, early diagnosis and treatment of heart attacks and strokes, and prevention of recurrent cardiovascular events are described in Healthy People 2010\(^{(29)}\). The two broad goals are to increase the years and quality of healthy life and to eliminate health disparities. Some of the Healthy People 2010 targets for heart disease and related conditions are shown in Table 4, along with baseline levels. Targets are also specified for health indicators including nutrition, physical activity, smoking, and healthy weight. Progress towards meeting the goals, which vary for subgroups of the population, will be monitored, and strategies to reach them as well as the goals themselves may be modified. Progress towards meeting the goals for 2000 was mixed; some were met but for some, such as obesity, change was in the wrong direction\(^{(29)}\).

In summary

While the decline in coronary heart disease mortality has been impressive in recent decades, coronary heart disease is still a major health problem; disparities in some heart disease-related conditions have increased. Treatment of heart attacks, coronary heart disease, dyslipidemia and hypertension has improved dramatically, and smoking cessation and prevention efforts are working. National educational programmes, media attention to

Eur Heart J, Vol. 22, issue 18, September 2001
science and health and the adoption of healthy lifestyles by millions of people have also contributed to the reduction in coronary heart disease rates and risks, but much remains to be done. Coronary heart disease is still the leading cause of death in the developed world and it is increasing in importance in the developing world. If fuller use were made of what we know now, the burden of coronary heart disease would be much less than it is. We can also be sure that advances in molecular and epidemiological genetics, and medical care will lead to better approaches to treatment, primary and secondary prevention in patients and high risk individuals. Further progress would also follow if more attention were given to families and disadvantaged populations.

References


Eur Heart J, Vol. 22, issue 18, September 2001


