Prevention of coronary heart disease

Report of a WHO Expert Committee

World Health Organization
Technical Report Series
678

World Health Organization, Geneva 1982
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WHO EXPERT COMMITTEE ON PREVENTION OF CORONARY HEART DISEASE
Geneva, 30 November–8 December 1981

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PREVENTION OF CORONARY HEART DISEASE

Report of a WHO Expert Committee

INTRODUCTION

A WHO Expert Committee on Prevention of Coronary Heart Disease met in Geneva from 30 November to 8 December 1981. Dr T. Lambo, Deputy Director-General, opened the meeting on behalf of the Director-General.

Cardiovascular diseases are a leading cause of mortality and morbidity in industrial countries, and they are also emerging as a prominent public health problem in developing countries. Being aware of this situation, the Twenty-ninth World Health Assembly in 1976 adopted a resolution (WHA29.49) inviting the Director-General to prepare a long-term programme in the field of cardiovascular diseases with special emphasis on promoting research on prevention, etiology, early diagnosis, treatment and rehabilitation, and on coordinating international cooperative activities. It also urged Member States to implement programmes for the control and prevention of cardiovascular diseases wherever necessary and feasible.

Coronary heart disease (CHD) has become the most important cardiovascular cause of premature disability and mortality in spite of the substantial knowledge concerning its prevention and control that has accumulated over the past three decades. In many populations (Table 1) CHD death rates are stationary or rising, in contradistinction to declining rates for other diseases. It was therefore considered essential to prepare an authoritative statement on the prevention of this disease which would facilitate the implementation of resolution WHA29.49 by Member States as well as by the World Health Organization. The need to organize the

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1 "Impairment of heart function due to inadequate blood flow to the heart compared to its needs, caused by obstructive changes in the coronary circulation to the heart". Synonym: "Ischaemic heart disease" (17).
Table 1. Mortality trends in 1968-1977: slope of first-order regression line (% per year), average of 5-year age groups between 40 and 69 years

<table>
<thead>
<tr>
<th>Countries</th>
<th>All causes</th>
<th>CVD</th>
<th>CVD − Cer D</th>
<th>Cer D</th>
<th>IHD</th>
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<tr>
<td>Canada</td>
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<td>−0.9</td>
<td>−1.5</td>
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</tr>
<tr>
<td>UK: Scotland</td>
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<td>-0.5</td>
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</table>

* Source: reference 32.

Key: Data based on the eighth (1965) revision of the International Classification of Diseases, Injuries, and Causes of Death, Vol. 1 (Geneva, World Health Organization, 1967), as follows:

- CVD = Diseases of the circulatory system (items 390–458).
- CVD = Cer D = Diseases of the circulatory system, excluding cerebrovascular disease (items 390–429 and 440–458).
- Cer D = Cerebrovascular disease (items 430–438).
- IH = Ischaemic heart disease (items 410–414).
- M = males; F = females.
present Expert Committee was also lent urgency by the fact that the Organization had not yet convened such a group to deal specifically with the issue of prevention of coronary heart disease.

1. BACKGROUND

1.1 Nature of the problem

Populations in which coronary heart disease is common are characterized by widespread and severe involvement with coronary atherosclerosis—a fibrous fatty change in the arteries serving the heart muscle, often associated with thrombosis. The disease may, without warning, result in sudden death; or it may manifest itself as an acute and often fatal attack of myocardial infarction, or as angina pectoris, congestive heart failure, or arrhythmias. It causes death or disability in many who are still in the active years of life and whose children are still young. Its personal and social costs are profound, both for the individuals and families involved and for the countries in which it is common.

1.2 The potential for prevention

The first argument for a preventive approach is the sheer size of the problem. The next is the early onset and insidious development of atherosclerosis, whose clinical manifestations appear when underlying arterial disease is far advanced and when injury to heart muscle is common. Many patients die suddenly, moreover, and thus do not reach medical care. Despite advances in treatment, the mortality among survivors of an acute heart attack is still high—around 10% in the first year and 5% yearly thereafter (27, 56).

Population death rates for CHD are not stationary but may rise or fall substantially over relatively short periods (see Table 1). Migrant populations tend to acquire the disease levels of their adopted cultures (36, 42). These observations indicate the operation of powerful environmental factors and thus the potential for prevention. This is further illustrated by the existence within high-incidence populations of some groups with substantially lower rates, and by the low incidence in Japan, despite a high level of economic development. CHD is not, therefore, the inevitable consequence of either aging or affluence.
The decline in CHD death rates which has recently been so striking in several countries has not tended to be accompanied by any rise in the rates for deaths from other causes at the same ages (32). CHD prevention can thus confer an actual extension of life and health and bring overall benefit to individuals and societies. The realization of this potential for prevention constitutes a leading challenge to all concerned with public health.

1.3 Preventive strategies

A comprehensive plan for prevention has three components: (1) a population strategy—for altering the life-style and environmental characteristics, and their social and economic determinants, that are the underlying causes of mass CHD; (2) a high-risk strategy—for bringing preventive care to individuals at special risk; and (3) secondary prevention—for averting recurrences and the progression of disease in those already afflicted.

In many areas of the world, including a few developed countries, life-styles have not as yet acquired the pattern associated with a high CHD incidence, and the average level of critical risk factors is still favourable. Economic advance and changing life-styles threaten to undermine this situation, and in such circumstances the need becomes urgent to consider primordial prevention—that is, preventing the emergence of predisposing conditions in countries in which they have not yet appeared.

These strategies are considered in the different sections of the report.

Prevention among individuals (particularly those at high risk) and secondary prevention have been scrutinized in a number of excellent reports by national and international bodies1, of which the conclusions are substantially in agreement. Thus in the present report it has been possible to deal with these aspects in a summary fashion only, and to concentrate attention on strategies for prevention in the population as a whole, in countries with both a high and a low incidence of CHD, focusing mainly on the control of underlying causes.

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1 A list of these publications is available, on request, from the Office of Library and Health Literature Services, World Health Organization, 1211 Geneva 27, Switzerland.
1.3.1 *The population strategy*

When serum-cholesterol levels are compared in different populations, it can be seen (Fig. 1) that the whole distribution is positioned at higher levels in a population in which CHD is common than in a low-incidence population. Such upwardly displaced distribution of risk characteristics in a whole population are thought to be the reason for mass atherosclerosis and a high overall incidence of CHD. They largely reflect environmental differences and thus are amenable to change.

These high average levels of blood lipids and blood pressure are probably recent in historical and evolutionary terms, and atherosclerosis and hypertension can be seen as a failure of adaptation in the face of modern eating patterns, smoking habits, and physical inactivity. The incidence of CHD depends on the predomi-
nance of these characteristics in the whole population, which, in turn, reflects the behaviour of a majority of the population.

From Fig. 1 it can be seen that the amount of variability in risk factors is proportionately much the same in each population. Theoretically attractive though it might be to have everyone centred around some "ideal" value, in practice it seems that the only way to lower the average to an acceptable level is to lower the whole distribution. Individual variation is inevitable, reflecting both variability of habits and also a large genetic component. Such individual differences determine who within a population will develop disease and who will remain healthy.

Fig. 2 shows how the individual risk increases at higher levels of serum cholesterol; but most of the CHD cases attributable to the cholesterol-associated risk occur, not from the few at high risk, but

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*Fig. 2. Prevalence distribution (histogram) of serum-cholesterol concentrations related to coronary heart disease mortality (interrupted line) in men aged 55-64 years. The number above each column represents an estimate of attributable deaths per 1000 population per 10-year period.*

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from the large numbers exposed to a lesser risk. To mitigate this situation a general lowering of the risk distribution is called for—that is, a population approach to prevention.

The need to shift the average level of the underlying factors in the direction of “biological normality” is the logical basis of primary prevention. It necessarily implies an approach at the level of whole communities; and it involves all ages—including children—not only because atherosclerosis starts early but also because behaviour patterns, including eating and smoking habits, are commonly established in childhood.

1.3.2 The “high-risk strategy”

Etiology and prevention are concerned with the causes of the total burden of disease in the population and how it can be reduced; they are also involved in the identification and correction of individual susceptibility.

By means of a simple test, such as blood-pressure and serum-cholesterol measurement, it is possible to identify individuals at special risk. Prediction can be considerably strengthened by considering simultaneously the results for several risk factors. Thus in one typical study (33), it was found that 43% of CHD cases occurred among the 20% of the study population with the highest estimated risk. Such individuals are in special need of preventive care (see section 3), and their recognition and treatment could make an important contribution to prevention.

Unfortunately, it is also true that in the study cited more than half of the CHD cases occurred in those who were not recognizably at special risk, and this is one limitation of the high-risk strategy. The reason for this can be seen in Fig. 2.

1.4 Aims of the report

In view of the fact that CHD is primarily a mass disease, reflecting the general characteristics of the life-style adopted by the population, the present report aims to provide a scientific basis and rationale, as well as specific recommendations, for public health policy and community action programmes.

The prevention of CHD involves action by all sectors of the community—government, education (including the mass media), industry, the medical services, and the people themselves. The role
of personal physicians and health personnel, traditionally important in the care of individual patients, is also vital in the population approach to prevention. Not only do they influence governmental policy-making, but through communication with patients, as well as by means of personal example and leadership, they play a major role in increasing public awareness and in modifying attitudes and behaviour that affect health.

The population approach to preventing CHD urgently requires the development and implementation of new forward-looking policies and techniques. Though it needs to be integrated into existing health services and prevention schemes, this approach demands specific efforts commensurate with the size and special features of the CHD problem. It also calls for better information systems to identify problems and monitor changes.

2. PREVENTION IN WHOLE POPULATIONS

2.1 The scientific basis

A rational policy of prevention first requires the identification of the primary causes of atherosclerosis and CHD, as well as (where possible) their intermediary mechanisms. The Committee judged that the major determinants of population rates of CHD had now been identified: an inappropriate national diet aggravated by physical inactivity and overweight (reflected in the mass raising of blood lipids and blood pressure), and widespread cigarette-smoking. Considerable progress has also been made in elucidating mechanisms, including the recent substantial work on thrombotic factors (47).

Community prevention programmes have demonstrated the feasibility of risk-factor reduction by hygienic means—that is to say, the adoption of a healthier life-style—in whole communities (12, 37). Large-scale controlled trials have further indicated the successful and safe lowering of blood-pressure and blood-lipid levels among high-risk persons (47). Other trials have examined the effect of risk-factor reduction on the incidence of CHD (54, 55). Such trials have been undertaken among middle-aged people, mostly among those at special risk, in order to test the reversibility of risk in people in whom arterial changes are often already advanced. There seems little likelihood that scientific experiments will be able to
demonstrate the effectiveness of primary prevention in the population as a whole, and thus decisions on preventive policy must be reached without prior experimental test.

"Natural experiments" are clearly occurring, involving significant modifications in life-style, social and economic conditions and health care, associated with large changes in CHD mortality (Table 1). Interpretation is complex, although sometimes associations can be seen with risk-factor changes. More could be learned about the feasibility, safety and effectiveness of preventive strategies by closer surveillance of these trends.

Clearly, further research is needed. On some important issues the present evidence is insufficient to justify recommendations. In a number of key areas, however, the balance of evidence indicates a sufficient assurance of safety, and a sufficient probability of major benefits, to warrant action now. The evidence for this judgement is similar in nature and strength to that which led in the past to policy decisions on such matters as the control of air pollution, sanitary improvements, and the formulation of standards for national dietary requirements. Public health policy has to be based on the best-informed judgement. The Expert Committee attempted to arrive at such a judgement with respect to the major components of life-styles in developed and developing countries.

2.2 Strategy and objectives

2.2.1 Life-styles and coronary heart disease

(1) Diet and blood cholesterol

(a) Evidence and rationale

The rationale for a community-wide strategy for the primary prevention of atherosclerosis and CHD involving dietary change is based on a well-established triangular relationship between habitual diet, blood-cholesterol levels and CHD.

With respect, first, to the blood cholesterol-CHD relationship, comparisons of populations show a strong association between average population total cholesterol (TC) levels and CHD incidence

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1 Plasma cholesterol is carried in the lipoproteins—the protein molecules which transmit fats and cholesterol in a soluble state and which are important in the metabolic processes leading to atherosclerosis.
rates (20, 21). Within populations TC predicts risk up to late middle age, the relationship being continuous over a wide range of TC levels (20).

The TC–CHD relationship is considered to be causal for the following reasons: the manifestation of its strength, graded character, consistency, and independence; the demonstration that the trait precedes the disease; the coherence with clinical and experimental data; and the fact that logical mechanisms have been delineated for the effect.

Blood TC levels in adults reflect well the levels of low-density lipoprotein (LDL), identified as the major source of cholesterol in atherosclerotic plaques. LDL is also mainly responsible for population differences in TC, for TC trends in populations, and for the relation of TC levels to individual risk. It accounts for between 60% and 80% of the TC.

High-density lipoprotein (HDL) cholesterol makes up about 20–30% of TC. It is strongly and inversely related to individual risk of CHD, but its presence explains little of the differences in rates between populations.

The new knowledge of lipoprotein roles in transport and metabolism helps to elucidate the mechanisms of genetic–environmental interaction in the causation of atherosclerosis. It extends but does not weaken evidence of mass behavioural effects on CHD risk factors or population evidence based largely on the simple measurements of TC.

With respect to diet–blood cholesterol relationships, there is a generally strong correlation between the saturated fat and cholesterol composition of habitual diets of populations and their mean TC or LDL levels. Similarly, changes in national diets are accompanied primarily by changes in average population TC (1). These findings are consistent with those in controlled diet experiments, so that average changes in TC can be predicted quantitatively from known changes in dietary composition (22). Considering the evidence as a whole, the Expert Committee judged the diet–blood lipoprotein relationship to be causal.

However, the diet–blood cholesterol evidence is limited in one important respect—namely, the difficulty encountered in demonstrating an association within populations between the current diet and the current TC levels of individuals. Genetic differences between individuals, as well as technical problems in characterizing the diet of individuals and their average TC levels,
must contribute to this problem. Persons having similar eating patterns may have different TC levels. How is this compatible with the important dietary influence demonstrated in comparisons between populations and in the clear results of experiments on diet and blood-lipid levels?

Table 2 may help to explain this paradox. An individual with very “favourable” intrinsic (possibly genetic) mechanisms for lipid handling, when receiving a diet shown experimentally to raise TC on average by 2.59 mmol/l (100 mg/dl) might have a relatively low TC level of 4.53 mmol/l (175 mg/dl), whereas another individual on the same diet might have intrinsic factors contributing to a higher TC level. Similarly, someone with a clear genetic defect in lipoprotein metabolism may have a high TC level (7.76 mmol/l; 300 mg/dl) while on a diet that only minimally raises TC. This illustrates the important interplay between dietary and intrinsic determinants of individual blood-cholesterol levels.

This diet–TC interrelation is further illustrated for populations in Table 3, in which it is assumed that large populations will have a relatively similar frequency of the several genes that influence blood-lipoprotein metabolism. Under this assumption, diets which elevate blood cholesterol substantially (e.g., the Finnish-type diet) might result in a mean TC of 6.48 mmol/l (250 mg/dl) for the adult population, while with a diet which raises TC only minimally (e.g., in Japan), the mean population TC value would approach the lowest “intrinsic” population response, at 3.89 mmol/l (150 mg/dl).
### Table 3. A model of population diet-serum cholesterol relationships, with population examples*

<table>
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<th></th>
<th>Japan (0)</th>
<th>Greece (+25)</th>
<th>Italy (+50)</th>
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<th>Finland (+100)</th>
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</table>

*NOTE: It is assumed that the polygenic determinants of blood-cholesterol levels are randomly and equally distributed among large heterogeneous populations, such that a mean population serum-cholesterol value of 150 mg/dl (3.89 mmol/l) would prevail (6.S.D. ± 37.5 mg/dl [0.97 mmol/l]) in the presence of an habitual average diet having neutral properties in respect of cholesterol. On this mean and population distribution of intrinsic responsiveness is superposed the average habitual diet effect for a population, which is either neutral or cholesterol-raising according to the country's measured diet composition and properties determined in controlled Minnesota diet experiments.*


Intermediate mean values of TC levels are portrayed with other national diets. Thus it is concluded that population mean TC levels and distributions are predominantly determined by habitual population diet.

The Expert Committee knew of no population in whom CHD is common that does not also have a relatively high mean level of TC (i.e., greater than 5.17 mmol/l (200 mg/dl) in adults). Mass elevation of blood cholesterol is therefore considered a factor necessary for the occurrence of severe coronary atherosclerosis on a mass scale, and thus mass CHD. With such a raised population mean TC level and the usual distribution around that mean, there will also be a relatively high prevalence of individuals with distinctly high levels.

For any given mean level and distribution of TC values there is a considerable range of CHD incidence, so that other factors, both known and possibly unknown, also contribute to CHD frequency.

Diets in populations having high average TC levels and mass CHD are characterized by relatively high saturated-fat and cholesterol consumption, a relatively high energy intake in relation to energy expenditure (with resultant high relative weight and prevalence of obesity), and relatively low complex carbohydrate consumption. The Committee concluded that the elevated cholesterol levels usually observed in many affluent populations would necessarily imply this kind of diet pattern.

The population findings are congruent with extensive clinical and animal observations. In animals, increased dietary cholesterol and
fat and attendant changes in serum cholesterol-lipoprotein are necessary for the experimental induction of atherosclerotic lesions, and correction of these altered levels is the prerequisite for regression of the lesions (8). Both "natural experiments" in vegetarian groups and large preventive trials demonstrate the feasibility, safety and effectiveness of dietary change in lowering TC and LDL levels in high-incidence populations.

The Oslo Heart Study (15), a randomized controlled trial of primary CHD prevention among middle-aged men at high CHD risk, has reported favourable and significant results in regard to CHD incidence and mortality, based on reduced intake of saturated fat and cholesterol (without recommendation of a high polyunsaturated-fat intake) and on efforts to achieve the cessation of smoking. Earlier single-factor diet trials of blood-cholesterol reduction in primary prevention also reported positive results, but they suffered from certain shortcomings in design, size and analysis, and the findings were not conclusive.

No single-factor diet trials in the general population are currently in progress or planned. The impediments are obvious: the large sample sizes required (estimated at 50,000–250,000) and the consequently huge costs; the need to begin prevention in middle age; and the difficulty in interpreting results because of other self-initiated changes in health behaviour. It is therefore concluded that the best basis for a medical care and public health policy is the extensive evidence already in hand. The Expert Committee assessed the current evidence as warranting a policy for CHD prevention that would include long-term community-wide efforts to encourage safe and palatable dietary patterns to reduce TC distributions in countries with a high CHD incidence, and to prevent the emergence of such levels in countries not yet experiencing a large burden of CHD.

With respect to the safety of a diet aimed at lowering population TC levels in high-incidence countries, it should be noted that countries in which such dietary patterns are found, and in which mean TC and CHD rates are usually lower, may also have excellent life expectancy (58). In fact, in 1978 the life expectancies of Greek and Japanese men were among the highest of any population group in the world. These data indicate that economically developing countries now having low CHD rates may continue to work for improved nutrition while avoiding the negative aspects of dietary patterns in affluent societies with high CHD rates.
Comparisons between developed countries indicate that those with higher saturated-fat and cholesterol intake generally have higher rates of colon and breast cancer. Moreover, recent diet-related falls in population mean TC levels (I) and concomitant declines in CHD mortality rates in the USA have not been associated with unfavourable disease trends. Rather, a substantial decline in death rates for all causes has been registered (see Table 1 and reference 32).

Some recent reports have suggested that within certain countries there may, on the contrary, be an association between low TC and higher rates of malignancy; but others have been negative in this respect, and yet others have noted that the association is manifest particularly in the first year of follow-up, suggesting that the low TC is a consequence of unrecognized pre-existent cancers (26). Taken as a whole the data are inconsistent, and it is concluded that they do not establish a causal relationship. At most, any cancer risk attributable to low TC is extremely small compared with the cardiovascular risk due to high levels. The evidence relating to this matter does not affect the propriety of the nutritional recommendations put forward here for lowering TC in populations in whom the mean is relatively high.

All these findings indicate that a lowering of the population TC distribution, through progressive changes in the composition of the habitual diet of the population, is a promising primary preventive strategy in high-incidence countries. The long-term strategy for all countries, including those with a low incidence, is to prevent the mass risk-factor elevations in the first place. This implies that low-incidence countries should seek to preserve the features of their eating patterns associated with low levels of CHD risk factors.

The rationale for specific objectives in promoting changes in the diet of populations is based on these observations among population groups as well as on the congruence with extensive clinical and experimental evidence. However, it must also be based on feasibility and cost, on consideration of local schedules for reaching particular goals, and on careful estimates of where each population stands with respect to its blood TC distribution and its mass CHD problem.

(b) Optimum population blood-cholesterol levels: guidelines

On the basis of the foregoing observations an attempt is made to suggest optimum blood-cholesterol levels for populations. Fig. 3 (5), in the third curve to the right, shows the distribution of adult
Fig. 3. "Ideal, feasible and existing" total serum-cholesterol levels

A: In adults

IDEAL MEAN 160 mg/dl (4.14 mmol/l)
S. D. = 25 mg/dl (0.65 mmol/l)
FEASIBLE MEAN 190 mg/dl (4.92 mmol/l)
S. D. = 30 mg/dl (0.78 mmol/l)
PRESENT MEAN 210 mg/dl (5.44 mmol/l)
S. D. = 35 mg/dl (0.91 mmol/l)

Total serum cholesterol

B: In youth (ages 5-18 years).

IDEAL MEAN 110 mg/dl (2.85 mmol/l),
S. D. = 20 mg/dl (0.52 mmol/l)
FEASIBLE MEAN 140 mg/dl (3.62 mmol/l),
S. D. = 25 mg/dl (0.65 mmol/l)
PRESENT MEAN 160 mg/dl (4.14 mmol/l),
S. D. = 25 mg/dl (0.65 mmol/l)

Total serum cholesterol

NOTE: These idealized, smoothed curves (on the right, interrupted lines) portray the present distribution believed feasible to obtain by a continuation of current changes in eating patterns in the USA over the next 10 years. The left-hand curves (solid lines) are those thought ideal with respect to freedom from a large population burden of atheroclerotic diseases. These curves also display the phenomena that skewness and the relative excess of individuals having high values tend to diminish as the population mean is lowered.

TC values in middle-aged men in the USA as at 1975–76. This mean value of 5.44 mmol/l (210 mg/dl) compares with a mean of about 6.09 mmol/l (235 mg/dl) that prevailed in the 1950s and early 1960s, indicating the possibility of achieving a mass change. The new mean and distribution in the USA—still relatively high—are similar to or exceed by those in many affluent cultures in various parts of the world. Countries with such TC levels and distributions generally have a significant burden of CHD. In contrast, populations in many areas whose mean cholesterol values are around 4.14 mmol/l (160 mg/dl) or lower, with a corresponding distribution (curve (solid line) on the left), manifest little or no clinical CHD or severe atherosclerosis. Moreover, populations with mean TC values ranging between 4.14 mmol/l (160 mg/dl) and 5.17 mmol/l (200 mg/dl) are to be found over wide areas (the Mediterranean basin and the Orient); these people have intermediate or low CHD rates, are well nourished, show no excess of noncardiovascular disease mortality and have good life expectancy at all ages.

The above-stated means and distributions are not proposed as specific goals for all populations but are intended as examples of what might be considered physiologically optimum, or feasible, for cultures having a large burden of CHD. They are congruent with recommendations made in the light of an independent examination of clinical and experimental pathological evidence for such optimum levels (2).

According to present knowledge, some people following dietary guidelines will have TC values above the population mean and may be at a level of risk requiring them to seek special advice (see section 3). Others will have values below the mean. For them it is relevant to note that there is no clearly identifiable TC threshold at which an effect on individual CHD risk is discernible. Thus the potential benefit is uncertain and may be related primarily to these individuals’ particular configuration of risk factors.

(c) Dietary guidelines

Nutrient and eating pattern guidelines are needed to achieve an optimum mean population TC level for adults, which is considered to be under 5.17 mmol/l (200 mg/dl). These dietary principles should not, however, prejudice normal growth and development of children or the quality of nutrition for adults; nor should they detract from the pleasure of eating.
The Expert Committee considered that the following dietary changes would be appropriate for high-incidence populations:
— a reduction in saturated fat and dietary cholesterol, which together are the primary factors that raise blood-cholesterol levels; this can be assisted by replacing some of the saturated fat by mono-unsaturated and polyunsaturated fat;
— an increase in complex carbohydrate consumption;
— avoidance or correction of overweight; and
— a reduction of cholesterol intake to below 100 mg per 4.18 MJ (1000 kcal) per day, or below an average of 300 mg for the adult population.

Current research findings suggest that the intake of polyunsaturated fats may have favourable effects other than the lowering of TC, including inhibition of thrombus formation. However, present evidence regarding effectiveness and safety does not justify the giving of general advice to increase the intake of any particular fatty acid. Observational and experimental studies indicate, for any population, an appropriate upper limit of 10% of energy intake in the form of polyunsaturated fats—i.e., avoidance of a high intake.

As to total fat, population data indicate that it is possible to have low rates of atherosclerosis and little CHD, along with good nutrition, with as little as 10% of energy intake deriving from fats (as in Japan), or as much as 40% (as in Crete), provided that saturated-fat intake is less than 10% and that polyunsaturated fat accounts for 3% or more of daily energy intake and that dietary cholesterol is not high. Nevertheless, a lowering of total fat intake is appropriate for populations in whom the level of physical activity is low and obesity is common. For such populations, to achieve a good intake of all essential nutrients, without a higher total energy intake, foods of low-energy and high-nutrient density require to be emphasized. Since all fats are high-energy foods, moderation is advisable (e.g., 20-30% of total energy intake).

An increased carbohydrate intake is desirable to emulate the diets characteristic of populations with little CHD. These diets are predominantly complex carbohydrates deriving from vegetables, fruit, whole grains and legumes, high in fibre.

The prevention or correction of obesity is of great importance, along with a diet of the composition just mentioned, in order to achieve optimum TC levels. This calls for moderation in total energy intake and, for persons engaged in work with little or no physical activity, increased leisure-time exercise. The Multiple Risk Factor
Intervention Trial in the USA (34) found that with a weight loss through exercise of as little as 3–4 kg by obese men, a substantially greater reduction in TC is achieved, and maintained, than that deriving from a change in dietary composition. All components of the blood lipid-lipoprotein spectrum are favourably affected by weight loss; LDL-cholesterol reduction is augmented, the level of triglycerides falls and that of HDL-cholesterol rises. These changes occur even in persons on antihypertensive medication. Weight control also deserves emphasis because it is useful for preventing and correcting high blood pressure and diabetes.

The following recommended dietary guidelines1 are based on many attractive traditional eating patterns:

**Emphasize:**
- appropriately combined foods of plant origin: beans, cereal grains, vegetables (cooked and raw), and fruit (offering good-quality protein, low fat, low saturated fat, low cholesterol, low sodium, low refined sugar, high complex carbohydrates, high minerals, vitamins and fibre, and lower energy intake);
- fish, poultry and lean meats, used in small portions and eaten less often as the main dish (offering good-quality protein, low fat, low saturated fat, low cholesterol, and lower energy intake);
- low-fat dairy products for adults (offering good-quality protein and minerals, low saturated fat, low cholesterol, and lower energy intake);
- less oils and fats in food preparation and in spreads; preference to be given to liquid vegetable oils.

**De-emphasize:**
- high-fat meats from domestic breeds as principal protein source (also high in saturated fat and cholesterol; and providing high energy intake);
- high-fat dairy products: whole milk, cream, cheeses (also high in saturated fats and cholesterol; and providing high energy intake);
- whole eggs, unless a major source of protein (egg yolks are high in dietary cholesterol);
- commercially baked products (high in saturated fat and providing high energy intake);

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1 All dietary recommendations must take account of local circumstances, including the presence of malnutrition and the availability of food. Recommendations for sodium intake are to be found in the section on blood pressure, pages 24–26.
— alcoholic beverages (providing high energy intake; low in nutrients).

(2) Blood pressure

(a) Evidence and rationale

The control and prevention of high blood pressure in populations are central to the prevention of stroke. The rationale for their inclusion in a programme for preventing CHD is based on the extensive and congruent evidence from epidemiological, clinical, pathological and experimental observations that high blood pressure is a major factor in the development of CHD.

The means and distribution of blood pressure, and the prevalence of high blood pressure, appear to differ widely between populations (though estimates of these are difficult because measurements are not standardized). These differences may contribute to population differences in CHD rates (13, 14). They suggest that powerful environmental factors interact with widespread human susceptibility to raise blood pressure. Strong international correlations are observed with salt intake: average population diastolic pressure appears to increase by 0.8 mmHg (0.1 kPa) per g of habitual salt intake (14a).

Within populations, blood pressure usually rises with age, so that by middle age the mean population diastolic pressure is high, as is the prevalence of elevated blood pressure (50). Systolic pressure, independent of weight change, continues to rise with age. It has been demonstrated consistently that both systolic and diastolic values of blood pressure are strong, graded and independent predictors of CHD risk. In some high-incidence populations the upper 20% of the distribution of blood pressure has a 4 times greater relative CHD risk than the lower 20%, with a continuous relationship between risk and the blood-pressure level.

These findings, reinforced by data from clinical and experimental studies, lead to the conclusion that high blood pressure contributes significantly to mass CHD.

Factors associated with high blood pressure include family history, obesity and weight gain, alcohol intake, and (in some cultures) low educational and socioeconomic status. The effect of individual salt intake on individual blood pressure is difficult to study, owing, in part, to problems of variability in measurement. A significant relationship may be found when great care is taken to
examine salt consumption and pressure over a wide range of intake and to reduce the effect of variability by multiple measurements.

Experimental evidence is generally consistent with clinical and population observations. Some data suggest that weight reduction and reduced sodium intake each make an independent contribution to the lowering of the blood pressure and enhance the effect of drug treatment of high blood pressure (38, 45).

The ultimate potential for prevention of high blood pressure in populations is illustrated by its virtually total absence in a few traditional, isolated, subsistence economies. The people are generally physically active, obesity is rare, and the sense of community is strong. Habitual salt intake is usually under 3 g daily (35).

The population data also imply that with a reduction of 5 g in the average daily salt intake of the population, average diastolic pressure can be lowered by 4 mmHg (0.5 kPa). Data on body-weight in high-incidence populations suggest that the new occurrence of high blood pressure in adult populations might be reduced by as much as one-quarter in some black populations, and by as much as one half in some white, through the control and prevention of overweight in the population (7).

Relatively small shifts in the distribution of blood pressure might be expected to produce relatively large potential effects in the reduction of complications due to high blood pressure, including CHD. The total effect of shifting the mean diastolic pressure of a population by a few mmHg might add significantly to the current effects of antihypertensive drug control in that population. Finally, the rationale for primary CHD prevention involves the possibility of primary prevention of elevated blood pressure in the first place, by salt reduction and weight control, though this possibility has not yet been well tested. Such a lowering of the whole distribution is the only means of benefiting those in the middle of the distribution, where much of the added burden of CHD arises.

(b) Strategy and objectives

The physiological objectives of a population approach to CHD prevention include the reduction of mean population blood pressure and of the frequency of uncontrolled high blood pressure. It is important that the local burden should be assessed by systematic and regular surveillance of the blood pressure and its related risk characteristics. A desirable average population value for diastolic
pressure is probably below 80 mmHg (10.7 kPa) for adults aged 30 or more.

The behavioural objectives of the control of high blood pressure are largely described in this report in the sections on diet (pages 14–24), physical activity (pages 28–30) and body-weight (pages 30–31). These recommendations are conducive to the control and prevention of obesity in the population, to the lowering of sodium intake and the relative increase of potassium intake, and to a reduction in alcohol consumption. The recommended level of salt consumption in the population is also a local decision, based on food preservation practices and on the schedule set for reaching the behavioural objectives. This, in turn, is related to socioeconomic issues and to the severity of the high blood pressure burden in a community.

The estimated effects of the control of obesity in the population, and the relative infrequency of adult high blood pressure when the average daily salt intake is below 5 g, suggests that the consumption of salt should approach 5 g daily or less, and that the measures previously outlined to control weight in the community should be introduced.

(3) Smoking

(a) Evidence and rationale

The health hazards of smoking for adults and young people are well established (57). The specific relationship of cigarette-smoking to CHD has been documented in population studies, though the detailed mechanisms remain uncertain.

Within populations, individual CHD risk is strongly related to the frequency and type of self-reported smoking. The relationship in high-incidence populations is strong, often dose-related, consistent, independent of other risks, and predictive. Therefore, despite the paucity of experimental data or clearly identified mechanisms, the association is considered to be causal.

Smoking interacts with other potent CHD precursors, so that even light smoking may carry a significant risk. Within low-incidence populations, with low levels of the other CHD risk factors, associations have been found only after long follow-up.

CHD mortality rates of ex-smokers are substantially lower than those of current smokers, as are recurrence rates of myocardial infarction (57). While the relative increase in CHD risk associated with smoking diminishes with age, the attributable (absolute excess) risk is large at all ages for both men and women.
The mechanisms by which tobacco-smoking influences CHD are not yet clear, although there seems to be a link with inhalation. Thus pipe-smoking generally carries less risk of CHD than does cigarette-smoking. However, former cigarette-smokers who change to pipes or cigars and continue to inhale probably remain at risk. Nicotine increases sympathetic discharge and is related experimentally to thrombosis, endothelial damage and cardiac arrhythmias. Carbon monoxide may cause direct vascular damage or act through relative hypoxia. It is not known whether one or both of these substances, or some of the other many toxic compounds in cigarette smoke, may be responsible for the effect on CHD. Some smokers who change to a low-tar/low-nicotine brand of cigarette appear to inhale more, thereby maintaining their nicotine intake while actually increasing the retention of gas-phase products. Present evidence does not support the promotion of a so-called “safer cigarette” so far as the effects on the heart are concerned. In high-incidence countries, CHD is the main cause of smokers’ excess mortality, hence the effect on total mortality of further reductions in tar/nicotine yield is problematical.

Smoking, especially of cigarettes, is a central issue in primary prevention. A population approach is crucial to reducing its frequency and to its eventual elimination because of the extent of the habit, governed as it is by social pressures.

(b) Strategies and objectives

The main aim of smoking-control programmes, including those for CHD prevention, is to create a situation in which nonsmoking is regarded as normal behaviour. Cigarette-smokers in the population should be systematically and repeatedly advised, supported and encouraged to give up the habit entirely. A switch in consumption to low-tar/low-nicotine cigarettes, or to pipe- or cigar-smoking, cannot be advocated, because there is no adequate evidence that the risk of CHD is thereby reduced. Nor can support be given to the aim of smoking fewer cigarettes daily, since this is so rarely sustained. Specific population goals include the following:
— a community attitude conducive to cessation and prevention;
— increased social unacceptability of tobacco-smoking;
— fewer or no young people taking up smoking; and
— an increase in the rate of smoking cessation among young people and adults.
The action programmes to achieve these behavioural goals require sustained and adequate funding to permit multiple approaches across the entire community. The present Committee, endorsing the programme proposed by the WHO Expert Committee on Smoking Control (52, p. 72) affirmed the following proposals relevant to CHD prevention and achievement of the above goals:

(i) Nonsmoking should be regarded as the normal social behaviour and all action which can promote the development of this attitude should be taken.
(ii) There should be a total prohibition of all forms of tobacco promotion.
(iii) Promotion of the export of tobacco and tobacco products should be discouraged. Tobacco-growing and manufacturing industries should be progressively reduced in size, while every effort should be made to protect the livelihood of those involved.

(4) Physical activity

(a) Evidence and rationale

Consideration of the role of habitual physical activity is part of the population approach to CHD prevention. A sedentary life-style for the majority of people is a fairly recent phenomenon. In developed countries this tendency towards inactivity has become considerably more marked on account of the increased availability of transport, more sedentary occupations, and the adoption of leisure-time pursuits involving no physical exercise. This mass change in life-style involves a great diminution in average population energy expenditure and in the frequency of peak or vigorous activities. In this century alone the average energy output of populations has decreased in developed countries as a result of sedentary living. The result is mass obesity and diminished work capacity. The latter may affect survival from a heart attack. Obesity, in turn, is an important determinant of the major precursors of CHD—high blood pressure, diabetes mellitus and elevated blood-cholesterol levels.

No direct relationship between habitual activity levels and the risk of heart attack has been established, either in individuals or in populations. The explanation for this omission is not only that habitual activity is poorly measured and present status and population trends are not monitored, but also that a primary prevention trial on exercise effects in CHD is not considered feasible. Thus, public health decisions about physical activity in CHD prevention
programmes are based primarily on the experimental evidence that regular physical activity will, through the reduction of body-weight and the lowering of blood-lipid levels, blood pressure and insulin activity, favourably modify atherosclerosis (25). Exercise helps to control body-weight in sedentary populations, while allowing a reasonable intake of nutrients, and it very probably helps to prevent these risk factors from becoming elevated in the first place.

Because of these considerations the Expert Committee gave a higher priority to this factor than might be indicated by the strength of the association of physical activity with CHD. The issue is important to affluent and developing nations alike. Great innovation is required in community education in order to attain healthy levels of physical activity in urban work and the general pattern of living.

(b) Strategy and objectives

Because of the strong cultural determinants of sedentary living-patterns, the strategy of control and prevention of elevated CHD risk factors in many modern societies involves the reintroduction of regular activity into daily life. A programme should seek a social climate for the adoption of physical activity as part of a healthy lifestyle, through community networks of promotional and educational services, and through the provision of greater opportunities and facilities for such activity.

Objectives for community programmes promoting enhanced physical activity include:
— increased average energy output in the population;
— a larger proportion of children and young people engaged in vigorous activities;
— increased frequency, duration and intensity of currently enjoyed leisure-time activities;
— increased acceptance of physical activity as a normal part of everyday life;
— enhancement of skills in sports; and
— increased community facilities and opportunities for exercise and for individual and family recreational activities.

Strategies to achieve these objectives are concerned partly with creating the necessary environment and partly with instituting educational measures aimed at:
— instructing the general public, through classes for young people and adults, mass media and community organizations;
— training and motivating leaders to expand their personal and family activities;
— educating the health professions to promote physical activity in their patients, as well as to set an example as leaders of public opinion;
— devising school programmes to increase the effectiveness of physical education by new curricula and teacher training in the development of lifetime personal activity habits and skills in youth;
— promoting increased physical activity and sports facilities at the work-place; and
— introducing campaigns to increase opportunities and facilities in the community for individual and group physical activity.

(5) Body-weight
(a) Background and rationale

The rationale for including the prevention and control of obesity in the strategy of CHD prevention in populations is based not on any large, independent association with CHD risk, but on the strong evidence that weight reduction helps to lower elevated levels of blood cholesterol and blood pressure and to decrease the prevalence of diabetes, at least in high-incidence cultures (24).

For example, on the basis of relationships found between weight gain and the recent occurrence of raised blood pressure in populations, it has been estimated in studies carried out in the USA that the control of weight in the whole community might reduce the incidence of high blood pressure by 25% in black populations and up to 50% in whites (7). In addition, weight loss improves insulin sensitivity and glucose tolerance (25). A significant reduction of diabetes in the population might be expected, as well as a reduction in the other disabilities and risks associated with gross overweight.

(b) Strategy and guidelines

The population strategy for controlling obesity in CHD prevention programmes includes reducing the existing frequency of obesity and preventing the development of obesity in populations not yet obese, including the people of some developing countries.

The strategy must consider not only the mass nature of obesity, but the profound sedentariness of many modern cultures whose members can ill afford, for good nutritional reasons, to reduce their
energy intake. The strategy must involve foods of low-energy and high-nutrient density: the aim should be to create new eating patterns—not to promote "dieting".

The long-term control and prevention of obesity requires the enhancement of physical activity in the population and the encouragement of appropriate personal skills and social supports, along with changes in community opportunities for greater physical activity.

The feasibility and safety of weight reduction has been demonstrated in some of the recent mass prevention trials, but not yet in populations as a whole. Careful planning and innovation are called for, using multiple educational strategies. The programme objective in overweight cultures should be that the population progressively approach the mean values and distributions of populations where leanness is the norm.

The specific objectives are identical to those outlined in the sections on diet and physical activity (pages 14–24 and 28–30 respectively).

(6) Diabetes mellitus

(a) Evidence and rationale

It has long been known that clinically recognized adult diabetes mellitus is associated with excess CHD risk. In some populations it makes an important overall contribution to CHD rates. The frequency of CHD among diabetics in developing countries is less than in developed countries that have high CHD rates, although it may still be greater than among nondiabetics. This phenomenon is thought to be due to lower values for blood cholesterol because of the different diets in those countries.

Simple glucose intolerance does not appear to be a powerful risk factor, even in high-incidence populations. Evidence is lacking about the effect of satisfactory control of glucose and insulin levels on the large-vessel complications of diabetes, including CHD.

Prevention of the onset in adults of non-insulin-dependent diabetes (52) is relevant to CHD prevention because of its association with CHD risk, its metabolic interrelations with obesity, blood lipids and blood pressure, and, possibly, its effect on heart-muscle function. Primary prevention of non-insulin-dependent diabetes is particularly relevant to prevention of CHD in areas of rapid economic development, because of the evidence that persons migrating from low- to high-incidence areas tend eventually to
become obese and develop diabetes and vascular complications (36).

The proportion of non-insulin-dependent diabetes preventable by weight control, although not known, is thought to be substantial.

(b) Recommendations and strategies

The importance of preventing non-insulin-dependent diabetes as part of the community-wide strategy for primary CHD prevention is recognized. This requires the control and prevention of obesity, which have been dealt with above in the sections on diet and blood lipids, physical activity, and body-weight.

(7) Psychological and social factors

Several behaviour patterns and psychological and social variables have been related to CHD risk in individuals and in the population at large. With respect to “stress”, or “response to stress”, the lack of definition and quantitative measurement is severely limiting.

The best-studied behavioural factor is “type A behaviour pattern”, associated with competitive drive, hostility, and a sense of urgency, or impatience. It contains many behavioural and physiological components which are not yet measurable, and cross-cultural comparisons are difficult to make. Nevertheless, within high-incidence populations, type A behaviour is significantly and independently associated with future CHD risk (39). The association is moderately strong but its consistency has not been sufficiently tested and no direct causal link has been established. Further, it is not clear that this behaviour pattern can be substantially changed, either in individuals or in populations; nor is it known how any such change might affect CHD risk. Nevertheless, the behaviour pattern remains important for the population problem: possibly its occupational and cultural determinants may contribute to the burden of CHD in the population.

Other characteristics of social organization and personal status have been thought to aggravate CHD, though none is considered a primary determinant. It is possible that some of these factors might be favourably modified with respect to occupation, living conditions, working hours, education, and socioeconomic status.

The Expert Committee noted the danger that public and professional misconceptions about “stress”, whereby it is assigned a primary role in the genesis of CHD, may divert attention from demonstrated needs in prevention.

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(8) Alcohol

Long-standing ideas about the possibly protective effect of alcohol consumption on CHD risk have recently been tested in population studies, though all these studies have been hampered by differences in drinking patterns and the beverages consumed, as well as by the absence of a valid quantitative method of assessment.

Within populations, recent studies generally suggest a higher CHD incidence in nondrinkers and in heavy drinkers, with more favourable CHD rates among light and moderate drinkers. However, CHD rates rise significantly—as do other disease rates—when the daily intake of alcohol exceeds 75 g (about 5 drinks). The contribution of alcohol is independent of other major risk factors (10).

Nevertheless, alcohol is strongly and consistently correlated with several CHD risk factors. The prevalence and incidence of hypertension, and the average level of blood pressure, are significantly related to a high intake, as is evidenced by a decrease in blood pressure levels among institutionalized alcoholics on withdrawal of alcohol.

Plausible mechanisms can be advanced for the causal role of alcohol in obesity and hypertension, on the one hand, and for certain blood-lipoprotein findings, on the other. Many studies now confirm that alcohol raises blood levels of high-density lipoprotein and very-low-density lipoprotein; the level of low-density lipoprotein decreases and there is little or no effect on the total cholesterol level (16).

Despite the evidence (unsubstantiated) of a possibly favourable effect of moderate alcohol consumption on CHD risk, the Expert Committee did not recommend the use of alcohol either as an individual or as a public health preventive measure. High alcohol intake is associated with excess risk of CHD, hypertension and many other physical, mental and social problems. These profound metabolic and social consequences of excess alcohol consumption, as well as the increasing intake in many countries, accentuate the public health nature of the alcohol problem, which has been dealt with in other WHO publications (see, for example, reference 53).

(9) Drinking-water

Between communities there is a consistent association of hard water (mineral content) with low CHD rates, although studies fail to find an association with individual risk (9, 29, 57). In the United
Kingdom, CHD mortality has tended to increase more in towns that have introduced water-softening (30). The Expert Committee acknowledged the water–CHD association as real, without knowing if it is causal.

No action was recommended because more information is needed. However, if community water-softening is under consideration, the association with cardiovascular mortality should be taken into account before any measures are adopted. As in the case of trace elements, the evidence is insufficient to recommend at this time any alteration of the content of water supplies for reasons connected with the prevention of CHD.

(10) Oral contraceptives

Within high-risk populations, oral contraceptive use is associated with increased risk of CHD, and with high blood-pressure and total serum-cholesterol levels (23, 28, 41). The risk from oral contraceptive use is compounded by cigarette-smoking. In the absence of information on the significance of oral contraceptive use on population rates of CHD, the risk might be reduced by a more selective use of these substances (see section 3.2.6), based on medical supervision and prescription. In developing countries these recommendations may not be appropriate, owing to the lower risk of CHD, limited resources and different priorities.

2.2.2 Prevention in youth

Although manifest CHD occurs mostly in middle age or later, the underlying atherosclerotic process may start early in life. In low-incidence populations the onset of atherosclerosis may possibly take place in adolescence or young adulthood, as shown in Fig. 4 (18, 43).

The first argument for starting prevention early in life is thus substantiated by the early onset of the disease. The second is that the major CHD risk factors, and the behavioural patterns conducive to them, also begin in youth. In many countries the first cigarette-smoking experience, potentially leading to the establishment of the habit, starts in children aged 6–11 years. Very rapidly, the habit is fully established: in some countries, smoking prevalence rates of up to 40% are recorded in girls and boys aged 15 years, and up to 50% by age 19—rates as high as in middle age.

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Fig. 4. Age-related dynamics of "incidence" of atherosclerosis plaques in selected developed and developing populations, derived from autopsy studies; prevalence rates at age 20 (X).
Nutritional habits, potentially leading to elevated blood-cholesterol levels, are acquired early in life. Serum cholesterol remains consistently in the same part of the distribution 4 years after having been measured in children aged 1–14 years (3), a phenomenon called “tracking”. Skinfold thickness in children—a measure of obesity—and blood pressure have also been shown to “track” years later in youth (6). In a study of college alumni, it has been shown that during young adulthood blood-pressure level, relative body-weight and habitual physical activity are substantially related to blood pressure, the frequency of high blood pressure, and to CHD incidence 30 years later (31).

The rationale for prevention in youth as part of the community-wide strategy of CHD prevention is thus based on evidence of the early onset of disease and of the adoption of habits leading to elevated risk characteristics (by interaction between individual susceptibility and cultural behaviour), and, finally, on the observation that bad habits are probably easier to avoid than to abandon.

Although risk-factor studies and preventive programmes in youth involve a potential risk of attaching harmful labels, which imply high risk or early disease, this should be avoidable within the context of education for healthy living habits. The question is how best to carry out CHD prevention in youth effectively and efficiently. A number of studies are taking place on the methodology of such approaches in children, based on school curricula adapted to this purpose, peer education strategies, youth-directed media programmes, and family education. Many preventive programmes among youth are now actually under way.

The principal objective of prevention in youth is to avert the development of elevated risk factors in the first place. The strategy is so integrated that a consistent health message is conveyed, during the passage of time, to the entire community—to the young as well as to adults. The methods involve schools, family groups, community institutions and governments. A strategic hypothesis to be tested is that children may help as agents for change in the total community picture.

2.2.3 National strategy

Effective population prevention of CHD can only come about through national policy, planning, development and commitment.
Specific efforts against heart disease, as against cancer and other major diseases, are considered justified because of the nature and size of the problem, requiring a special effort to achieve significant progress. Preventive efforts against CHD should be seen as part of a more general programme against noncommunicable diseases. Control of CHD risk factors could lead at the same time to the reduction of respiratory disease, some cancers, diabetes, etc. Policymakers should see this as an argument reinforcing the case for efforts against CHD, and the different components of prevention should effectively complement and strengthen each other.

A national effort is most effective when well planned, persistent and adequately funded. A national effort will make local efforts easier. Early examples of national initiatives include the several governmental statements over the last two decades in the USA on suggested eating patterns, and the National High Blood Pressure Education Programme (46), which has existed in that country since 1973. This has had a distinct influence on the control of hypertension in the United States population. Such efforts are also well represented by the Swedish Campaign for a Nonsmoking Generation (44); community-wide in orientation, it focuses on women in pre- and postnatal clinics and will continue through the early childhood years and schooldays of their offspring.

Data available from many sources demonstrating the propensity of communities to change, of individual health behaviour to change, and of customs to change in relatively short periods provide a basis for some optimism, once the fundamental national commitment is made to a long-term strategy of primary prevention, and at least modest resources are made available to implement that strategy. This is illustrated by the changes in smoking habits among physicians in the United Kingdom and among educated adults generally in the USA, and by the changes in eating patterns in the latter country over the last 15 years. The increase in leisure-time physical activity in Europe and North America also demonstrates that people can and do change their behaviour. This potential simply needs improved planning and focus, enhanced facilities, coordination and leadership.

Action programmes are required to achieve substantial preventive efforts in populations, through helping communities to adopt healthier behaviour patterns and to lower their CHD risk-factor distributions. These undertakings require skilled and detailed community analyses, careful preparation of the community and its
involvement with planning. Because of the several risk behaviours involved, and the several audiences to be reached, community prevention programmes should include multiple behavioural strategies, reaching the whole community as well as targeted subgroups. This emphasizes the role of education and the effective organization of community leaders and resources. Legislation should be employed, where appropriate. Action programmes contain the following components:

(1) **Professional education**

This approach seeks more efficient and systematic counselling by professional personnel on health behaviour. Training involves the medical and allied health professions in combination and emphasizes experience of change in personal and family health behaviour by the professional health workers themselves, as well as their orientation to prevention and guided practice in risk assessment and counselling. Improved professional understanding is thereby achieved for the behavioural change process, as well as increased credibility.

(2) **Community leader education**

This approach seeks the efficient introduction into the community of preventive concepts and improved health behaviour through the enhanced understanding and the direct involvement of leaders of public opinion. Their behaviour and understanding of community health goals, their serving as role models and activists, and their management skills help to implement preventive strategies.

(3) **Public education**

This approach seeks to increase the awareness and knowledge of alternative health behaviour by education, given direct or through the media, for all age groups in a continuing, consistent programme. Direct public education may include the following:

— systematic education through risk-factor measurements utilizing either the usual primary health care facilities or special services at work or elsewhere, combined with health information and counselling;

— adult education classes on risk factors and prevention;

— school health programmes emphasizing teaching by peers of effective social behaviour; and

— health education for senior citizens dealing with the specific health behaviour of older people.
(4) Mass media education

This approach seeks to increase community awareness of the major health issues, to guide individuals to available facilities and to provide an atmosphere throughout the community which supports individual change towards more healthy behaviour, as exemplified by antismoking and antihypertension media campaigns. These alert the public to the need for control and prevention, to issues and controversies surrounding prevention, and to community prevention resources. More detailed guidelines to alternative health behaviour are provided through the written media—newspapers and pamphlets—as a first step towards increasing community skills in achieving behavioural change.

(5) Community organization

This approach seeks to enhance existing resources for health promotion, to achieve direct modification of the environment, to support wider choices and opportunities for change, to train community leaders, and to implement programmes in relation to smoking, eating and exercise.

(6) Environmental change

Encouragement of more healthy community behaviour may be achieved by modifying the environment. Examples of this complementary strategy are actions on the part of governmental and voluntary bodies to provide recreational facilities which lead to increased physical activity, to introduce targeted marketing practices promoting alternative foods and eating patterns, and to prohibit smoking in public places.

2.2.4 Prevention in developing countries

(1) Background and rationale

The Twenty-ninth World Health Assembly, in its resolution WHA29.49, recognized the emergence of a trend towards an increase in heart and blood-vessel diseases that accompanies socioeconomic change in developing countries. It emphasized that, with adequate research and prevention programmes, the untoward health consequences experienced by industrialized societies could be avoided by developing countries. A logical approach to this challenge is called "primordial prevention", aiming at preventing the emergence and entrenchment of social, economic and cultural
patterns of living that are known to contribute to elevated risk-factor distributions and CHD incidence in developed countries.

As a result of a continuing decline in mortality from infectious and parasitic diseases, life expectancy at birth in the developing countries as a group improved from 42 years in 1950 to 55 years in 1975. The corresponding figure projected for the year 2000 is between 65 and 70 years, and it is estimated that by that time 60% of the world’s elderly (i.e., persons over 65 years of age) will be living in the developing countries (49, pp. 233, 239). At this rate of demographic transition, noncommunicable diseases, notably heart and blood-vessel diseases (especially CHD), are likely to assume public health dimensions even before the infectious and parasitic diseases have been brought fully under control.

In some developing countries—e.g., Malaysia, Mauritius, Singapore, and Sri Lanka—CHD is already an important cause of death among adults. In others—e.g., Ghana and the Ivory Coast—CHD is present to be found in urban middle- and upper-income groups, among those who have acquired behaviour which contributes to elevated risk characteristics. As socioeconomic development progresses, these risk factors may become widespread phenomena leading to mass CHD, and increasing urbanization may accelerate this trend. Furthermore, it is estimated that by the turn of the century developing countries will contribute 41% of the world’s total urban population, as compared with the 1950 figure of 27% (49, p. 234).

Recent evidence that CHD mortality is declining in certain affluent and industrial societies confirms that mass premature CHD is not an unavoidable accompaniment of socioeconomic development. It provides justification for timely efforts aimed at primordial prevention of CHD in the developing countries.

(2) Strategy and objectives

The objective of primordial prevention of CHD is to prevent or inhibit the development of elevated CHD risk-factor distributions in the population as a whole. The principles and strategies discussed in the preceding parts of this section for preventive approaches in high-incidence populations are also largely applicable in developing countries. Their scope of action includes:

— national policy and programme on nutrition and eating patterns (providing the agricultural sector, the food industry and those responsible for food imports with guidelines to ensure a healthy diet for the different age groups of the population);
— policy on smoking control and related national strategies in respect of agriculture, tobacco imports, and education;
— programme for the prevention and control of hypertension; and
— programme for the promotion of physical activity and the avoidance of obesity.

The details of implementing strategy for primordial prevention will vary with local circumstances and local health priorities. In all developing countries the approach should be aimed at the whole population, with special attention given to children and adolescents to inculcate healthy habits and modes of living from an early age. At the same time efforts should also be made to reduce or modify risk factors (i.e., primary prevention) in groups that have already acquired the habits that promote them (see section 3).

(3) Primordial prevention and primary health care

The main approach to the primordial prevention of CHD is through education: "education concerning prevailing health problems and methods of preventing and controlling them" (48, p. 53) is one of the basic elements of the primary health care approach, which countries have accepted as the key to attaining the social goal of Health for All by the Year 2000.

Primordial prevention in general, and more particularly as it applies to CHD, is therefore most appropriately developed in relation to strategies for primary health care—e.g., the detection and control of high blood pressure and health education concerning smoking, diet and obesity can be integrated into primary health care. The methodology for such an approach needs to be formulated and tested in the local situation and then adapted as necessary to suit other circumstances.

The high levels of government commitment and community involvement required for primary health care are also necessary for the implementation of the political and social action required for effective primordial prevention. Securing public and government support for a population-wide preventive approach is crucial to any strategy of primordial prevention. Such motivation is likely to be more successful and persuasive if it is based on concrete and locally relevant data. This implies the establishment of data-collection systems and supporting research to define the CHD burden, to monitor population disease trends and to determine risk-factor levels and related health behaviour.
3. PREVENTION IN INDIVIDUALS AT SPECIAL RISK

Many CHD cases occur in individuals who can readily be identified in the medical setting as being at special risk. This implies an important preventive task for physicians and other professional health workers. The Bethesda Conference on the prevention of CHD (34) identified four objectives in this regard: (1) to promote the concept that prevention is an integral and beneficial part of medical practice; (2) to identify areas in which the physician can make significant contributions; (3) to provide guidance to physicians in various settings; and (4) to motivate those not now actively engaged.

To implement this preventive approach, each physician in a high-incidence country needs a plan for identifying individuals at risk, objectives for their management, and a scheme for counselling and follow-up (11). This plan should involve all members of the medical team.

3.1 Identifying risk

For each adult patient under care the smoking history should be noted, along with any strong family history of CHD and any personal history of diabetes or CHD; the blood pressure should be recorded and a note made of weight and obesity. Where these suggest special risk, a serum-cholesterol measurement is desirable (provided that reliable laboratory facilities exist). Taken together, the information on combined multiple risk factors permits an overall assessment of the broad level of CHD risk; individually, the risk-factor levels identify needs for specific advice.

3.2 Specific advice

3.2.1 Smoking

Complete cessation of cigarette-smoking should be the advice given to all, and particularly to those with a special risk of CHD. The relative risk associated with smoking is about the same for all individuals of a given age; but the absolute excess (attributable risk) is much greater if elevated blood pressure, elevated serum cholesterol or diabetes is also present.

Advice needs to be unequivocal and accompanied by explanation and with offers of continued interest and help for those wishing to
stop the habit. The physician’s own example is influential. If these efforts are a routine part of practice they can have a major community impact: in a recent survey in the USA the commonest reason given for having stopped smoking was “advice from my doctor”. Much new knowledge has been developed on how to help people to give up smoking. However, the means for conveying the necessary behaviour-modification techniques to primary health care teams are urgently needed.

3.2.2. Blood pressure

The detection and control of hypertension are central to the prevention of stroke. It is likely that control may also reduce the risk of fatal CHD (50), although the trials that have been carried out have not completely answered this question. It is additionally important in CHD prevention to identify lesser degrees of elevated blood pressure, as this indicates some increase in CHD risk for the individual. For the population as a whole, the total of attributable cases of CHD rises at least as much among the many cases of less severe blood-pressure elevation as among the smaller number under treatment for more severe hypertension. Individuals in this so-called mild or borderline hypertensive group require preventive advice on all risk factors and related health behaviour.

3.2.3 Serum-cholesterol level

Where this is known, individuals may best be considered in 3 risk categories and given appropriate advice, as follows:

1. Less than 5.69 mmol/l (220 mg/dl). General advice on a healthy eating pattern to avoid the usual rise with age and to set a good family example.

2. 5.69–6.73 mmol/l (220–260 mg/dl). Specific dietary advice (see pages 14–24).

3. Over 6.73 mmol/l (260 mg/dl). Fuller investigation and treatment are indicated.

3.2.4 Physical inactivity and overweight

The principles of management have already been described in sections 2.2.1 (4) and 2.2.1 (5).
3.2.5 Diabetes

Diabetics have an increased CHD risk (see section 2.2.1 (6)). Correction of overweight by energy-intake restriction and regular exercise; advice, where appropriate, about stopping smoking; correction of elevated serum-cholesterol level; and hygienic measures or drug treatment, as appropriate, for elevated blood pressure—all these are considered to be important in the prevention of CHD among diabetics.

3.2.6 The use of oral contraceptives

The risk of fatal myocardial infarction appears to be 2 or 3 times greater when oral contraceptives are used. In low-incidence populations and low-risk individuals this produces only a slight absolute increase in risk. However, the additional risk becomes important in women over the age of 30 who also smoke or who have either elevated blood pressure or elevated blood cholesterol, and in all women past the age of 40.

In high-incidence countries oral contraceptives should be used only under medical supervision, which would include regular measurement of blood pressure. Prescribing policy should be selective, the use of these preparations being discouraged in older women, and especially in those with increased levels of CHD risk factors. Smoking greatly increases the risk of CHD in women using oral contraceptives and users should therefore be specially counselled about the dangers of smoking.

4. PREVENTING THE RECURRENCE AND PROGRESSION OF CORONARY HEART DISEASE

A substantial proportion of CHD deaths occur in people already known to have the disease; measures to influence the course of already-recognized CHD might help significantly to reduce the total attributable mortality.

Following myocardial infarction (an acute heart attack) the prognosis is dominated by factors associated with the severity of heart damage. During this period the outlook may be substantially
improved by appropriate drug therapy—and medical advances in this field are rapid. The subject is beyond the scope of this report, as are the topics of coronary artery (by-pass) surgery, resuscitation after cardiac arrest, and the use of pacemakers. Here the concern is rather with measures intended to slow the progression of the underlying disease process. The long-term prognosis after heart attack is influenced by many of the same risk factors that provoked the first attack, suggesting that atherosclerosis continues to progress, and hence that preventive measures are still relevant. Despite the lack of evidence so far from controlled preventive trials, the Expert Committee recommended that, for every CHD patient, planned preventive measures should be part of the usual clinical care (40). The expectation of long-term benefit is unlikely to be less, and could well be more, than in primary prevention.

The principles governing preventive measures after the onset of clinical disease are the same as those already set out in the previous sections of this report. These measures should normally begin as soon as possible after the diagnosis is made; but the personal characteristics of each patient must be considered, and the timing and nature of advice modified accordingly.

4.1 Smoking

After myocardial infarction the prognosis for smokers is much worse than for nonsmokers, but this excess risk is not seen in those who stop smoking. Patients should therefore be advised to cease smoking immediately, and this advice should be reinforced by frequent repetition; moreover, they should be helped in their efforts to eschew the habit.

4.2 Physical activity

Controlled trials have indicated a tendency for physical activity to have a positive effect on outcome in CHD patients; but, owing to the insufficient number of subjects, this finding does not attain statistical significance. Regular dynamic exercise appropriate to individual capacity is recommended as a rehabilitative measure in view of its effects on symptoms and work capacity and the help it gives in controlling risk-factor levels.
4.3 Diet and eating patterns

Elevated blood cholesterol probably affects long-term prognosis. Controlled intervention trials have been too restricted in the number of subjects and too short in duration; their findings have been inconclusive. The Expert Committee recommended that in general the same dietary principles should be followed as in primary prevention (see pages 21–24). This also applies to the management of overweight and the need to moderate the intake of alcohol.

4.4 Diabetes and hypertension

These should be managed according to the usual clinical criteria.

5. CONCLUSIONS AND RECOMMENDATIONS

(1) Despite medical advances, coronary heart disease (CHD) remains a major worldwide public health problem affecting both sexes.

(2) Rates differ widely from country to country; they are rising in some countries and falling in others. These differences and changes demonstrate the important potential role of prevention.

(3) Individual differences in CHD risk within high-incidence populations and the ability to modify the risk-factor levels also demonstrate the preventive potential.

(4) With regard to several key preventive measures, the balance of evidence indicates sufficient assurance of safety and a sufficient probability of major benefits to warrant action at the population level. The evidence is similar in nature and strength to that governing past policy decisions on air pollution control, sanitary improvements, and the formulation of dietary requirements.

(5) Preventive strategies include: (a) altering the mass characteristics of life-style and environment which are the underlying causes of mass disease (prevention in whole populations); (b) in low-incidence countries, preventing the development of these precursors (primordial prevention in whole populations); (c) within a population, identifying and helping individuals at special risk; and (d) preventing recurrences and progression of disease (secondary prevention).
(6) In high-incidence countries the levels of major risk factors are too high in the majority of people. Most cases of CHD occur among the large number in whom risk factors are moderately elevated, not among the small number with high values. Only a mass (population) approach can help this larger group.

(7) CHD risk is significantly influenced by a number of personal and population characteristics and their combination. These, in turn, are largely determined by sociocultural factors and are therefore modifiable. Such characteristics include elevated blood pressure and blood cholesterol and the associated eating and activity patterns, and smoking.

(8) The relationships between habitual diet, blood cholesterol-lipoprotein levels and CHD are well established and are judged to be causal. In high-incidence countries a lowering of the population distribution of blood-cholesterol levels is recommended through progressive changes in eating patterns. As a guideline, a population average value of under 5.17 mmol/l (200 mg/dl) is likely to be associated with no more than a moderate frequency of CHD. To achieve such a level, the consumption of saturated fats must be limited to less than 10% of energy intake, that of dietary cholesterol to an average of under 300 mg per day and obesity must be avoided. These limitations are consistent with attractive and widely found eating patterns. Some of the reduction in saturated fat may be made up by mono- and polyunsaturated fats. Present evidence regarding effectiveness and safety does not justify the giving of general advice to increase the intake of any particular fatty acid.

(9) Even a small reduction in the average blood pressure of the population could bring about a large reduction in CHD. On the basis of the evidence as a whole, it is recommended that populations should be encouraged to reduce the consumption of salt in the direction of 5 g daily or less. The importance of controlling obesity and excess alcohol consumption in the population is stressed.

(10) Smoking, especially of cigarettes, contributes significantly to the occurrence of CHD. The first objective is that nonsmoking should come to be regarded as normal behaviour, and strategies for achieving this are available. As far as CHD is concerned, present evidence does not support promotion of the so-called “safer cigarette”.

(11) Lack of physical activity relates to increased population levels of the major CHD risk factors, primarily through the high prevalence of obesity. Population levels of obesity are greatly
influenced by average energy expenditure. Regular exercise may help to reduce high blood pressure and blood cholesterol. A higher priority is therefore given to physical activity in CHD prevention than is justifiable simply by its association with CHD. Regular physical activity should be a part of normal daily life.

(12) Diabetes mellitus contributes to the occurrence of CHD. Cases of onset in adults (non-insulin-dependent diabetes) might be averted to an important extent by preventing obesity.

(13) High alcohol intake is associated with an increased risk of CHD and high blood pressure, as well as with other profound physical and social harm. Increased alcohol intake is not recommended as a preventive measure in CHD, either in populations or in individuals. Moderate intake may be associated with a somewhat lower risk of CHD, but causality has not been established.

(14) Community water supplies should not be softened without first considering the association with cardiovascular mortality.

(15) In countries with a high CHD incidence, oral contraceptives should, where possible, be prescribed under medical supervision so as to discourage their use by women at a higher risk of CHD. In particular, oral contraceptive users should not smoke.

(16) The underlying atherosclerotic process leading to mass CHD begins in youth, along with the appearance of its major risk characteristics—elevated blood pressure and blood cholesterol—and associated risky behaviour, including smoking. Preventive measures should be introduced in childhood, in order to avert the development of these CHD precursors.

National programme planning should be initiated for youth, as part of a comprehensive disease prevention and health promotion effort. Effective educational programmes, including those aimed at the prevention of smoking, are now available; they should be adapted for local needs, implemented, and then evaluated. Well-informed young people may act as agents promoting improved health behaviour in the community.

(17) The heavy social burden of CHD in high-incidence countries, as well as the threat of a future burden in developing countries, influenced as it is by sociocultural factors, requires commitment to long-term national strategies for effective prevention.

National governments should challenge their health leaders and their legislators to prepare action plans, with time-based goals, for analyses of the local situation, and to initiate and evaluate such plans. They should prepare estimates of the requisite community
resources and funding. This calls for specific programmes for the control and prevention of high blood pressure and for improving national patterns of eating, physical activity and smoking. These must be coordinated in a comprehensive national disease surveillance, prevention and health promotion strategy involving the medical profession, the health services, schools, community organizations, the agricultural sector, the tobacco and food industries, and the mass media.

(18) In some developing countries CHD is already a serious problem, which threatens to become more widespread as socioeconomic development progresses. Policies on eating patterns and nutrition, the control of smoking and avoidance of obesity and sedentary living are essential to prevent the development of the risk-factor patterns, familiar in developed countries, that are associated with high rates of CHD. This involves the maintenance of a healthy life-style and active surveillance in countries in which the incidence is rising.

(19) Physicians and other health personnel have an important role to play in identifying and helping individuals at special risk of CHD, as well as in spreading an understanding of the causes and prevention of heart disease and in forming public opinion and policies. Their personal example is also important. Educational efforts should be mounted for the effective orientation of physicians and allied personnel to the systematic practice of prevention and health promotion.

(20) Many CHD events occur in persons already known to have the disease. This gives an opportunity for preventing recurrences and progression (secondary prevention). Preventive efforts should follow the same principles as in primary prevention, taking account of patients’ individual circumstances.

(21) An international working group should design approaches to curricula for training responsible medical officers in the strategies and methods of community prevention of CHD, within the context of comprehensive preventive efforts. These medical officers should initiate national courses for appropriate health personnel.

(22) Special attention should be paid to the establishment of appropriate information systems, including those dealing with mortality and incidence, case-fatality and treatment trends, population risk-factor levels and their trends, as well as sociocultural influences on population characteristics relevant to prevention. The new WHO project on Monitoring Trends and Determinants in
Cardiovascular Diseases (MONICA project) is a promising initiative which deserves the strong and full support of governments.

(23) The promotion of CHD prevention activities would be facilitated by the regular collection of information on their progress in different countries and by the giving of wide publicity to such reports. The WHO International Clearinghouse on Smoking and Health is an example of a suitable approach.

(24) The implementation of projects on the primordial prevention of CHD should be promoted in developing countries in collaboration with WHO. The newly established pilot areas for primary health care in several countries might be invited to cooperate in such an effort.

(25) For optimum implementation of national CHD prevention programmes, more knowledge is needed on the health attitudes and behaviour both of individuals and of entire populations, on the influences and constraints governing changes of behaviour, and on the degree of compliance with preventive recommendations.

(26) There is an urgent need for research into the primary prevention of arterial hypertension along several promising lines, including eating and activity patterns in populations.

(27) Innovative epidemiological investigations are needed on simultaneous trends, and lags in trends, of CHD incidence, treatment, risk-factor distributions and behaviour. The WHO MONICA project (see paragraph 22) and several pilot investigations sponsored by the United States National Institutes of Health are examples.

(28) Research and demonstrations in specific areas that are not yet well developed—particularly in respect of eating patterns and physical activity programmes—should be promoted and the findings tested.

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