CORONARY HEART DISEASE
THE NEED FOR ACTION

CORONARY HEART DISEASE
THE NEED FOR ACTION

Office of Health Economics
12 Whitehall London SW1A 2DY

© February 1990. Office of Health Economics.

ISSN 0473 8837

OFFICE OF HEALTH ECONOMICS

The Office of Health Economics was founded in 1962 by the Association of the British Pharmaceutical Industry. Its terms of reference are:

To undertake research on the economic aspects of medical care.

To investigate other health and social problems.

To collect data from other countries.

To publish results, data and conclusions relevant to the above.

The Office of Health Economics welcomes financial support and discussions on research problems with any persons or bodies interested in its work.

INTRODUCTION

At the start of the 1980s, coronary heart disease (CHD) appeared destined to become the United Kingdom's 'disease of the decade'. Comparisons between nations had revealed that the countries of the United Kingdom were becoming increasingly isolated at the top of the international league table for CHD mortality. As a result there were calls from many quarters for urgent action to bring the death rates down. The measures proposed by the experts – primarily, appropriate responses to the established risk factors for the disease – became a topic of growing interest for the general media. Coverage was increasingly given to the changes required in smoking, dietary and other habits and to the ways in which the desired objectives might be achieved. Yet just as public awareness of the problem was reaching new heights, reports began to emerge about the discovery of a new and lethal virus affecting mankind. Subsequently, the reporting of medical issues in the lay press and by the broadcasting media has been dominated by the human immunodeficiency virus and its fatal consequence, acquired immune deficiency syndrome.

This development does not, however, alter the fact that CHD remains the nation's most substantial contemporary healthcare problem. In England and Wales, CHD was responsible for 153,084 deaths during

Table 1 **R and D expenditure on cardiovascular diseases.**

	British Heart Foundation	*Medical Research Council*[1]	*British Pharmaceutical Industry*[2,3]
	£ millions	£ millions	£ millions
1980/81	4.0	2.8 (6.3)	50.2
1981/82	5.1	2.7 (6.3)	59.2
1982/83	7.7	3.3 (8.7)	72.0
1983/84	6.2	4.1 (10.3)	86.2
1984/85	8.6	5.0 (11.1)	96.6
1985/86	9.2	4.7 (11.2)	110.0
1986/87	11.5	4.3 (11.9)	122.4
1987/88	11.8	4.7 (11.3)	133.6
	64.1	31.6	730.2

1 The figures in brackets show expenditures not directly allocated to the cardiovascular system heading that do nevertheless have relevance to this particular system.

2 Expenditures are calculated from estimates that about 20 per cent of total industry R and D spend goes to cardiovascular systems projects (Centre for Medicines Research – private communication).

3 Figures are for calendar years starting in 1980.

Sources Annual reports of the Association of Medical Research Charities, the Medical Research Council and the Association of the British Pharmaceutical Industry.

1988 – equivalent to nearly 27 per cent of total mortality. More specifically, it is a major cause of premature death, especially for males. The 1988 CHD death toll among persons aged 15–64 years generated a loss of potential working life estimated at nearly 240,000 years and males accounted for 82 per cent of the total. Alternatively, CHD caused the death of one person under 65 years of age every 19 minutes throughout 1988.

The magnitude of the problem posed by CHD is reflected in the size of the research effort aimed at clarifying the mechanisms underlying pathogenesis and at discovering more effective treatments. Table 1 shows that the pharmaceutical industry, the British Heart Foundation (the largest charity supporting heart research) and the government via the Medical Research Council, spent an estimated £150 million on cardiovascular system research in 1987 – or a cumulative total for the 1980s so far of £730 million. It is, however, preventive action to cut the incidence of CHD that provides the fundamental long-term key to reducing the substantial National Health Service and other economic and social costs generated by the disease. The objective of this paper, which updates parts of an earlier OHE publication on the topic (Wells, 1982), is to quantify these resource implications. In so doing it underscores the pressing need for the public health education campaign – 'Look after your heart' – launched in Spring 1987 by the Department of Health and the new Health Education Authority.

HOW MANY CASES?

Comprehensive data showing the total number of people suffering from the various manifestations of CHD (see Box) do not exist. The fact that some individuals remain asymptomatic even though marked coronary artery disease is present implies that traditional measures of health service usage provide only a limited insight into the extent of the problem. However, a number of community surveys concerned with specific age groups have been undertaken and these show that the prevalence of CHD is high. For example, the British Regional Heart Study found that one-quarter of middle-aged men showed evidence (based on questionnaire and electrocardiogram investigation) of CHD (Shaper *et al*, 1984).

The survey, which involved 7,735 men aged 40–59 years drawn at random from general practices in 24 British towns, also made clear that prevalence increases rapidly with age. The figures contained in Table 2 indicate that the overall prevalence of CHD, using electrocardiogram *or* questionnaire evidence, rises from 17.6 per cent among individuals aged 40–44 years to 31.2 per cent in the 55–59 years age group. Severe CHD, as indicated by positive findings from both questionnaire *and* electrocardiogram, is seen to increase to an even greater extent over the age spectrum: prevalence rises fourfold between 40–44 years and 55–59 years, with almost one man in ten in the latter age bracket affected in this way.

The nature of coronary heart disease

The heart circulates blood throughout the body by means of electrically-stimulated contractions of its muscle, the myocardium. The latter is the principal constituent of the wall of the heart and is dependent on a continuous flow of blood if it is to function efficiently and survive. The heart muscle receives its blood supply before any other part of the body via arteries situated at the base of the aorta. Blood is initially channelled through three major arteries and is then distributed throughout the myocardium by a complex network of progressively smaller vessels.

In coronary heart disease, interruptions to the blood flow result from a number of processes including those which lead to a thickening of the intima layer of the artery walls thereby narrowing the bore (lumen) of the vessels at the affected sites. These lesions are patches of atheroma and consist of deposits of lipids (fatty materials, including cholesterol) and scar tissue. Uncertainty persists about the precise relationship between most of the CHD risk factors and the cellular developments in atherosclerosis (Ross, 1986). Nevertheless, at advanced stages, the latter can lead to severe occlusion of the coronary arteries, giving rise to or laying the foundation for, the three principal manifestations of CHD – angina pectoris, acute myocardial infarction and sudden death.

Angina pectoris is the gripping pain experienced in the chest and often extending to the neck and left arm when an additional workload is placed upon the heart during, for example, physical exertion or profound emotional stress. The precise mechanisms are ill-defined, but the pain is the result of an inadequate supply of oxygen reaching the heart's muscle at such times of extra demand, usually because of the presence of atherosclerotic obstructions in the coronary arteries, which set a limit to possible increases in myocardial blood flow. When the activity precipitating the episode ceases, the demand for, and supply of, oxygen return once again to equilibrium and the pain recedes.

Anginal symptoms which develop in persons at rest and are not therefore prompted by a raised heart rate, as well as episodes of pain increasing in their frequency or severity of presentation, are together referred to as 'unstable' angina. Until recently the transient reduction of blood flow in unstable angina was thought to result principally from spasm in the coronary arteries but new research has pointed to the potential importance of recurrent thrombosis at the site of a ruptured atherosclerotic plaque (Fuster and Chesebro, 1986).

Acute myocardial infarction. Sustained loss of blood supply to any part of the myocardium rapidly leads to a cessation of normal contraction and is followed by death of the heart muscle in the affected area. The extent of this myocardial destruction is an important determinant of the prospects for survival and further morbidity. Substantial atherosclerotic narrowing is found in at least one of the major coronary arteries in most cases of acute myocardial infarction and eventual occlusion may be the result of an intimal tear at such points of narrowing. However, the role of thrombosis in arterial occlusion in this way, as well as in the formation of atheromatous plaques, is a source of debate (Stehbens, 1985).

Sudden cardiac death is the term applied to cases where the patient, often in apparently good health, collapses entirely without warning or with only fleeting premonitory symptoms. Post-mortem examinations indicate that the majority of patients placed in this category show no evidence of occlusive thrombosis but have instead severe coronary atheroma, generally with over 85 per cent stenosis of the major vessels (Davies, 1982). Death is usually the result of spontaneous ventricular fibrillation and may be defined as 'sudden and unheralded' (Mitchell, 1978) in order to differentiate such occurrences from fatalities which occur suddenly after infarction, due, for example, to extensive myocardial necrosis leading to pump failure. United States data suggest that about one quarter of heart attacks fall into the sudden death category. About 80 per cent of the patients involved have a known history of CHD but for the remainder sudden cardiac death is the first indication of coronary heart disease (Eisenberg *et al*, 1986).

Table 2 **Prevalence of various diagnostic categories of ischaemic heart disease by age group, percentages.**

Diagnostic category	*Age Group*			
	40–44	*45–49*	*50–54*	*55–59*
Questionnaire (Q):				
Possible MI and angina	1.1	2.2	3.5	4.2
Possible MI only	4.5	5.7	7.7	7.2
Definite angina only	1.4	2.2	3.1	5.1
Possible angina only	1.5	1.6	2.6	2.5
Electrocardiogram (ECG):				
Myocardial infarction	1.7	3.3	5.2	6.6
Myocardial ischaemia	8.8	8.1	11.2	12.9
Combined:				
Q or ECG	17.6	21.1	28.0	31.2
Q and ECG	2.3	3.4	7.0	9.3

Note MI, myocardial infarction.

Source Shaper *et al.* 1984.

Application of the findings of the British Regional Heart Study to contemporary population data suggest that 1.38 million men aged between 40 and 59 years in England and Wales have evidence of CHD. Within this total, approximately one person in every five (that is 303,000 men) may be regarded as severely affected by the disease. Most cases, nearly 60 per cent, involve men who are in their fifties and at this age one in four of those affected may be categorised as severe.

As with prevalence, comprehensive incidence data are in short supply. With some notable exceptions, such as the Framingham Study in the United States, few studies reflecting first manifestations of all types of coronary event, which are also representative of the population as a whole, have been undertaken. However, the Royal College of General Practitioners has recently published its third national survey of morbidity in general practice and this report provides information on CHD incidence as seen from the primary care perspective. The figures contained in Table 3 show that for both sexes the incidence of the various clinical manifestations of CHD increases sharply with age. However, important differences between the sexes are clearly apparent. For example, with the single exception of angina in the 25–44 age grouping, male incidence rates for all CHD forms exceed those for females throughout the age spectrum. In addition, within the age band 45–74 years, myocardial infarction and angina are of approximately equal significance for males, whilst among females the latter is the more frequently experienced condition.

Combined with 1987 population data, the rates shown in Table 3 yield a total of 404,000 new cases of CHD in general practice each year in England and Wales. Within this total, as many as 26 per cent of new events involve males aged 45–64 years, even though these individuals account for only 11 per cent of the population as a whole. It is, however, inevitable, that data from this source will understate the true burden of new coronary disease in the community.

Focusing on middle-aged males, the incidence of myocardial infarction in particular has been singled out for more detailed analysis in several studies. The condition itself accounts for between 40 and 50 per cent of new CHD manifestations in men aged 45–64 years and it is, of course, among this group that premature mortality from heart disease is at its most substantial (see later). Against this background, several special registers have been established to monitor the annual occurrence of heart attacks. In Edinburgh, for example, Armstrong and coworkers (1972) reported a coronary attack rate among men aged 40–69 years of 15.5 per 1,000. The Tower Hamlets study in East London found a definite acute myocardial infarction rate of 7 per 1,000 among males in the age range 45–64 years (Tunstall Pedoe *et al*, 1975). More recently the British Regional Heart Study has indicated a combined acute myocardial infarction or sudden coronary death rate of 6.2 per 1,000 men aged 40–59 years per annum (Shaper *et al*, 1985).

The latter figure disguises a range from 2.1 per 1,000 among those aged 40–44 to 9.6 for the 55–59 years age group. In total, the study suggested that over the course of a year there might now be as many as 36,000 heart attacks among males aged 40–59 years in England and Wales and that nearly two out of every five of these episodes would occur in the much narrower age range of 55–59 years.

Table 3 **Incidence of coronary heart disease, males and females by age, England and Wales 1981/82, rates per 1,000 population.**

	Males			*Females*		
Age	*Acute myocardial infarction*	*Angina of effort*	*Other coronary heart disease*	*Acute myocardial infarction*	*Angina of effort*	*Other coronary heart disease*
15–24	0.0	—	0.1	—	—	—
25–44	0.8	0.3	0.2	0.2	0.4	0.1
45–64	7.4	6.8	3.6	2.5	4.4	1.6
65–74	12.8	14.2	6.9	6.8	9.0	5.5
75+	13.9	14.4	8.4	9.2	9.5	6.8
All ages	3.4	3.3	1.8	1.9	2.6	1.4

Source Royal College of General Practitioners, 1986.

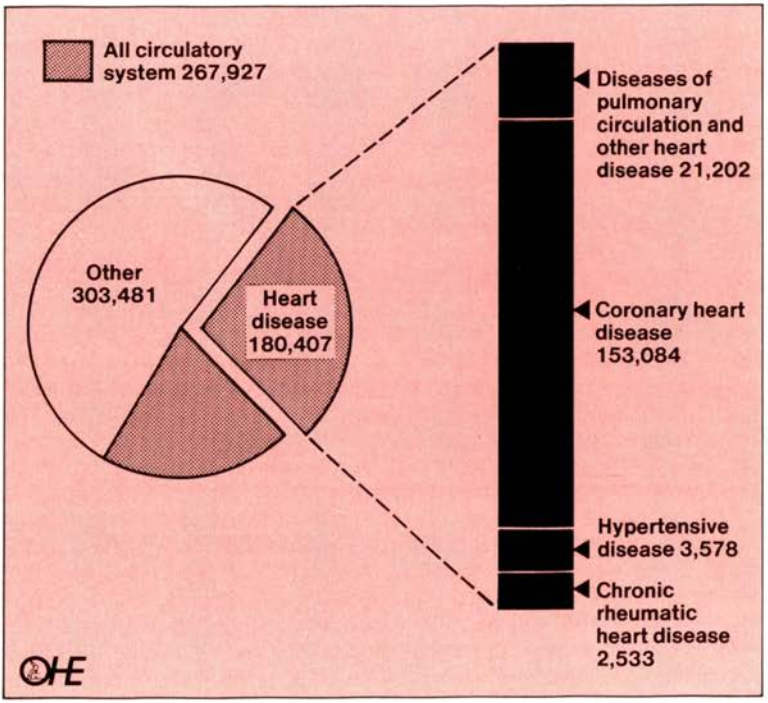
CHD MORTALITY PATTERNS

Diseases of the circulatory system were responsible for 47 per cent of the total number of deaths recorded in England and Wales in 1988 (Figure 1). Conditions specifically relating to the heart accounted for 67 per cent of the deaths within this broad group (a large proportion of the remaining deaths were caused by strokes). Consequently, one death in every three in England and Wales is due to heart disease.

However, Figure 1 also makes clear that heart disease is a broad term embracing a wide range of specific disorders. Of these, coronary heart disease is clearly the most significant. In 1988, it caused 153,084 deaths, that is 27 per cent of the total for England and Wales in that year.

Table 4 shows the age distribution of mortality from CHD. Four-fifths of deaths from this cause occur in persons aged 65 years or more. Nevertheless, the disease remains a significant cause of premature mortality. In 1988, 27,553 people under 65 years died from CHD. Most

Figure 1 **Mortality in England and Wales in 1988.**



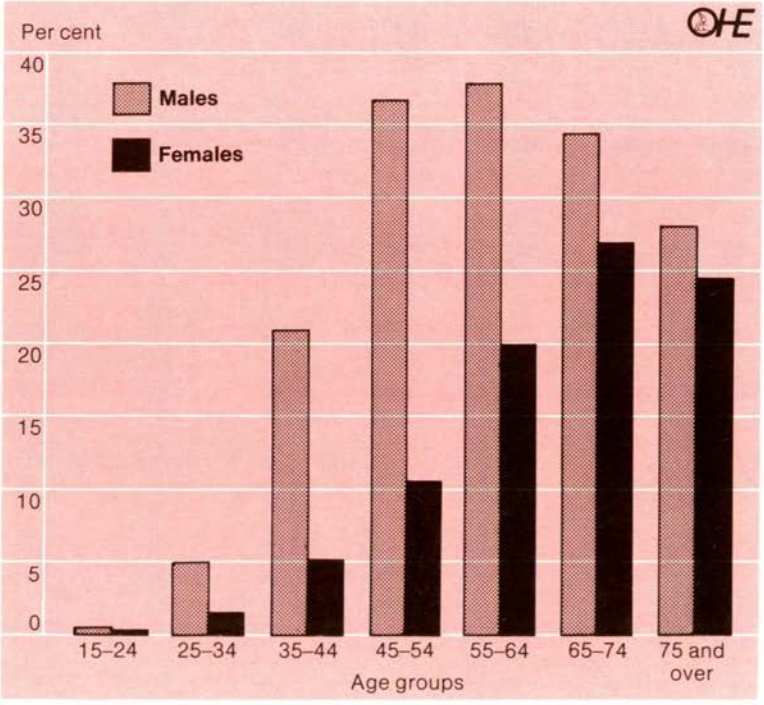
Source OPCS Monitor DH2 89/2.

Table 4 **Age Distribution of CHD Mortality in England and Wales, 1988.**

Age group	*Males*	*Females*	*All Persons*
Under 15	1	2	3
15–24	14	4	18
25–34	167	27	194
35–44	1,231	209	1,440
45–54	5,038	897	5,935
55–64	15,120	4,843	19,963
65–74	27,945	15,265	43,210
75 and over	35,364	46,957	82,321
All ages	84,880	68,204	153,084

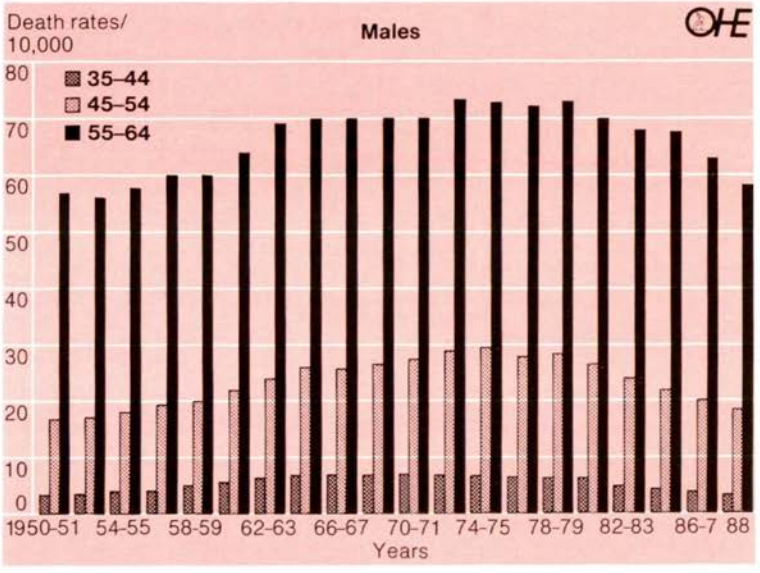
Source OPCS Monitor DH2 89/2.

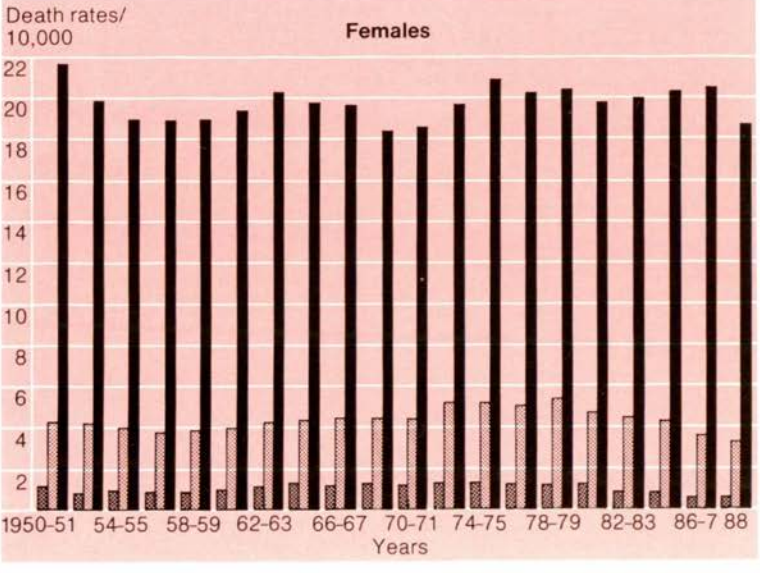
Figure 2 **Deaths from CHD as a percentage of all deaths at selected ages, England and Wales, 1988.**



Source OPCS Monitor DH2 89/2.

Figures 3a and 3b **Deaths from coronary heart disease, at ages 35–44, 45–54, 55–64, England and Wales, rates per 10,000, 1950–1988.**





Source OPCS Mortality Statistics.

of these fatalities involved males. Below the age of 65 years male CHD deaths outnumbered those for females by a ratio of 3.61 to 1.00 (above this age the ratio is 1.02 to 1.00). The significance of CHD for males of working age is further illustrated in Figure 2. Overall, the condition accounts for 30 per cent of male deaths under 65 years, but this proportion reaches about two out of every five between 45 and 64 years.

It would be inappropriate, however, to underestimate the significance of CHD as a cause of premature death among females (Figure 2). In 1988, the disease accounted for 14 per cent of female mortality below 65 years, a proportion that reached 20 per cent for those aged between 55 and 65 years.

Available data suggest that there has been little or no change in the proportional significance of CHD as a cause of death for either sex since OHE last published on the subject seven years ago. However, a more optimistic picture is revealed by an examination of mortality rates. Figures 3a and 3b plot CHD rates for men and women over the 35-year period since 1950. Although caution has to be exercised in interpreting these data, because several revisions of the International Classification of Diseases have been implemented during the period, the broad pattern to emerge is one of increasing mortality rates for both sexes until the second half of the 1970s. Since then, however, improvements have been achieved. Table 5 shows that between 1978 and 1988 mortality rates declined in all age groups below 65 years for both sexes. In total, the reductions over the period meant that there were about 8,727 fewer CHD deaths among persons aged 35–64 years in 1988 than would have been the case without these improvements. Some 84 per cent of this 'saving' occurred in male deaths and was especially concentrated in the 45–64 years age range.

International comparisons

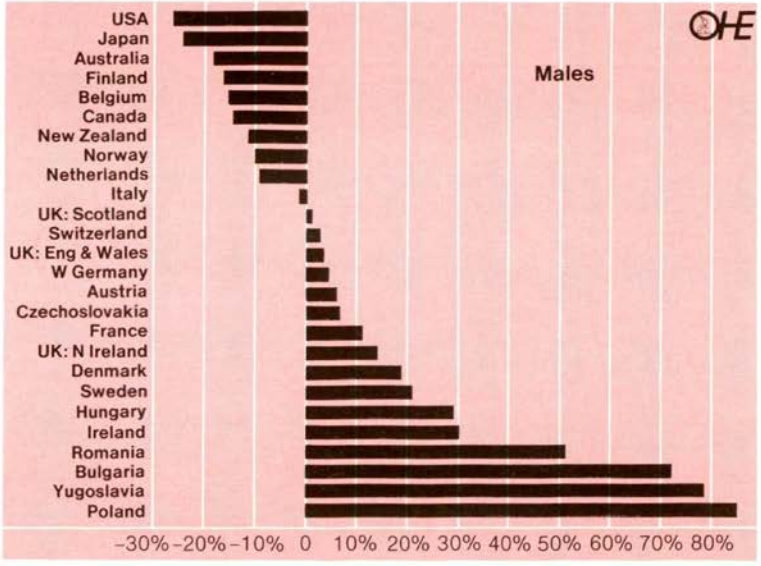
Welcome as these reductions clearly are, they might have been substantially greater had mortality rates in England and Wales declined to the same extent as in certain other nations of the developed world. Figure 4 shows for males and females the changes that occurred in

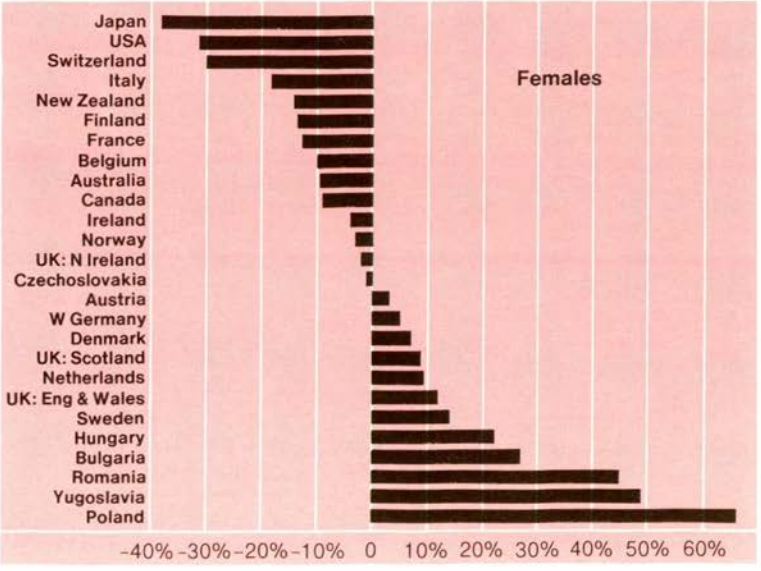
Table 5 **Percentage reductions in coronary heart disease mortality rates, England and Wales 1978–88 and consequent number of deaths avoided in 1988 (in brackets).**

Age group	*Males*	*Females*
35–44	–14.7 (915)	–1.1 (148)
45–54	–14.2 (2,589)	–19.0 (518)
55–64	–12.9 (3,850)	–12.0 (707)
Total	(7,354)	(1,373)

Source OPCS, Mortality Statistics 1986.

Figures 4a and 4b **Average percentage change in mortality from ischaemic heart disease at ages 40 to 69 years over 1968–1979 (based on the slopes on linear regressions fitted to mortality trends in 6 quinquennial age groups).**





Source Pisa and Uemura 1982.
(Reproduced by kind permission of the British Cardiac Society.)

Table 6 **Male CHD mortality rates per 100,000 in England and Wales and the United States.**

	1968		*1978*		*1986*	
Age group	*E+W*	*US*	*E+W*	*US*	*E+W*	*US*
35–44	63.4	92.8	61.6	59.6	40.6	37.2
45–54	245.1	355.0	279.2	260.4	209.3	154.9
55–64	698.1	960.9	732.5	704.4	639.7	446.0

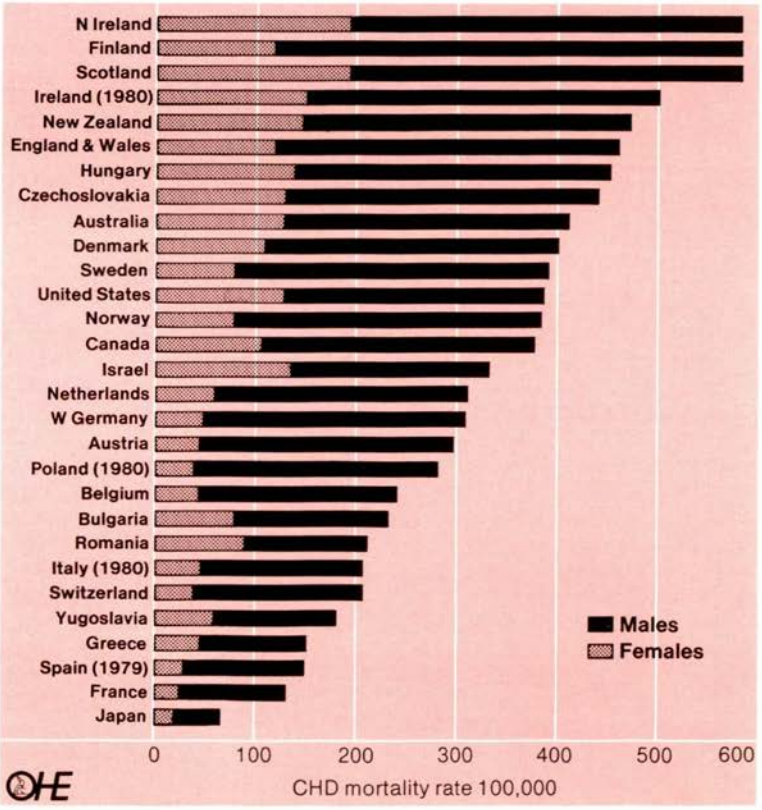
Sources OPCS and WHO World Health Statistics Annual.

CHD mortality for the 40–69 years age group in 26 nations between 1968 and 1979. Focusing on males (who account for three-quarters of CHD deaths in this age range in England and Wales) the diagram indicates that the most dramatic reduction occurred in the United States. In contrast, the rates for each of the nations of the United Kingdom marginally increased over the same period.

A more detailed examination of the data for 1968–78 (this timeframe avoids problems of inconsistency arising from a revision of the International Classification of Diseases in 1979) highlights the substantial differences in the CHD experiences of the US and England and Wales (Table 6). In the US in 1968, male CHD death rates at ages 35–44, 45–54 and 55–64 were 46, 45 and 38 per cent higher than those prevailing in England and Wales. During the next 10 years, the US rates for these age groups fell 36, 27 and 27 per cent respectively (for England and Wales the corresponding changes were a reduction of 3 per cent, an increase of 14 per cent and another increase of 5 per cent). As a result, all three US rates were slightly below the corresponding values in England and Wales in 1978.

The international picture at the start of the 1980s is shown in Figure 5. The countries of the United Kingdom occupy three of the six places at the top of the league with England and Wales in sixth position. Subsequently, as noted earlier, the CHD mortality rates for England and Wales started to decline. Yet the improvement has not matched that of the US. As Table 6 indicates, the gap between the rates for the two nations widened in all age groups shown between 1978 and 1986. Indeed, if it can be assumed that the US rates have continued to fall between 1986 and 1988 at the same pace as between 1978–86, then it may be estimated that a commensurate reduction in England and Wales rates over the whole period would have meant 17,078 deaths in 1988 among males aged 35–64, instead of the recorded total of 21,389 – a potential saving foregone of over 4,300 lives.

Figure 5 **Age-standardised mortality from coronary heart disease in 1981. (Rates per 100,000 population aged 40–69 years.)**



Source Tunstall-Pedoe, *et al.*

Table 7 **Patients consulting for CHD – rates per 1,000 at risk.**

	1981/82		
	Males	*Females*	*Persons*
Acute myocardial infarction, sub-acute ischaemic heart disease	5.5	2.9	4.1
Angina of effort	8.1	5.8	6.9
Other chronic ischaemic heart disease	6.2	4.5	5.3
	19.8	13.2	16.3

Source RCGP, 1986.

THE COST OF CHD TO THE NHS

The burden imposed by CHD on the health services is substantial. In the primary healthcare sector, the recently published third national survey of morbidity in general practice indicates that 1.6 per cent of the population – that is, 814,000 people in England and Wales – consult their family doctor during the course of a year because of CHD (Table 7). Analysis by age reveals that the patients consulting rates increase steadily with age for both sexes, peaking in the 75 years and over grouping (Table 8). Nevertheless, the rates are still significant below retirement age: four per cent of men and almost 2 per cent of women aged between 45 and 64 years consult their general practitioner each year for CHD. These proportions imply that at these ages patients consulting general practitioners outnumber persons dying from CHD by a ratio of 10 to 1.

The extent of the workload imposed by CHD on general practice may be gauged from the data on consultations collected in the third national morbidity survey. Table 9 shows that despite the predictably large degree of variation by sex and age grouping, overall 1.5 per cent of consultations are attributable to CHD. Application of this proportion to

Table 8 **Patients consulting for CHD, rates per 1,000 at risk, by age and sex.**

Age group	*Males*	*Females*	*Persons*
25–44	3.3	1.3	2.2
45–64	39.9	17.4	28.4
65–74	82.3	48.7	63.5
75+	87.9	61.6	70.5
All ages	19.8	13.2	16.3

Source RCGP, 1986.

Table 9 **Consultations for coronary heart disease: rates per 1,000 persons at risk, by age and sex.**

	All ages		*25–44*		*45–64*		*65–74*		*75+*	
	Rate	*as % of total*	*Rate*	*as % of total*	*Rate*	*as % of total*	*Rate*	*as % of total*	*Rate*	*as % of total*
Persons	49.9	1.5	7.1	0.2	95.0	2.7	174.5	4.0	211.7	3.9
Males	62.5	2.3	10.7	0.5	142.8	4.7	228.0	5.6	253.0	4.8
Females	38.4	1.0	3.5	0.1	49.9	1.3	132.4	2.9	191.0	3.5

Source RCGP, 1986.

Table 10 **Estimated NHS cost of CHD in 1987 in England and Wales.**

	£ million	
	1985	*1987*
Hospital care:		
In-patients	199	229
Out-patients	13	15
Primary care:		
General Medical Services	15	18
Medicines	139	184
Dispensing costs	28	34
	394	481

the total expenditure on the general medical services in England and Wales in 1987 yields a CHD cost of £18 million. It has, of course, to be recognised, that this approach to costing is subject to the limitations inherent in average data. It may be the case, for example, that patients suffering from CHD require longer consultation times than many other patient groups, in which case the estimate presented above may understate the true cost.

Estimates of the costs arising from the treatment of CHD in the primary-care setting must also take account of the expenditure on medicines prescribed by general practitioners. The estimated figure of £184 million shown in Table 10 is the total net ingredient cost generated by 26.6 million prescriptions for preparations acting on the heart in England and Wales in 1987. A further cost arises from the dispensing of these medicines and this sum is estimated at about £34 million in 1987.

It is once again unclear how accurately these figures represent expenditure on pharmaceuticals for CHD patients. Overstatement arises in that some of the medicines contained in the preparations acting on the heart category may not be prescribed specifically for the treatment of CHD. However, understatement is also inevitable, since vasodilators, antihypertensives, diuretics and tranquillisers have been excluded from the expenditure calculations because of the problems involved in allocating their costs between different therapeutic uses. Alone, preparations acting on the heart account for about 12 per cent of total pharmaceutical costs. This figure rises to 25 per cent when the four other groups noted above are included as well. The magnitude of this range is clearly substantial, although it is probable that the true level of medicines expenditure generated by CHD approaches the lower rather than the upper of these limits.

CHD also has very significant resource consequences for the hospital sector of the NHS. Data from the Hospital In-patient Enquiry indicate that there were 187,000 hospital admissions (strictly, discharges from

Table 11 **Hospital in-patient cases of CHD, England, 1985.**

	Discharges and deaths		*Mean duration of stay (days)*		*CHD discharges and deaths as % of total*	
Age	*Males*	*Females*	*Males*	*Females*	*Males*	*Females*
0– 4	0	40	—	5.5	0.00	0.02
5–14	0	0	—	—	0.00	0.00
15–44	8,440	1,640	7.2	6.9	1.45	0.18
45–64	61,770	18,660	8.0	8.4	11.18	3.49
65–74	31,150	19,650	9.4	10.4	8.08	5.65
75–84	16,360	19,790	12.2	14.6	5.53	5.07
85 and over	2,740	6,780	15.1	31.9	4.17	4.06
All Ages	120,460	66,560	9.0	13.2	5.08	2.47

Source Hospital In-patient Enquiry, 1985.

or deaths in hospital) for CHD in England in 1985 (Table 11). This figure represented 3.7 per cent of admissions for all causes but the proportion rises significantly in certain age groups. For example, CHD accounts for 11 per cent of all male admissions in the age range 45–64 years. Indeed, males of this age account for one-third of total CHD admissions to hospital and one-quarter of CHD hospital-bed days.

On average, hospitalised CHD cases spend 10.5 days as in-patients, yielding a total of 2.13 million bed days for the disease in England in 1985. In 1985/86, the average cost per day in large acute hospitals was estimated at approximately £95, so that CHD in-patient care may be calculated to have cost £187 million. Uprating on a pro-rata basis to include Wales increases this sum to £199 million. It is estimated that this total has risen to £229 million in 1987.

Finally, account has to be taken of hospital out-patient treatment for CHD. Figures for England show that there were 372,815 attendances at cardiology and cardiac surgery out-patient clinics in 1987. For Wales, a corresponding figure of 21,103 may be estimated from the data available. Given an average cost per attendance of approximately £36, this volume of out-patient treatment yields a total cost of £15 million. The sum is likely to understate the true level of cost since approaching one-fifth of the attendances were first visits and these are generally more expensive than subsequent attendances. However, shortfall from this source will be counterbalanced (to an unknown extent) by the fact that CHD is not the only diagnosis presenting at cardiology out-patient departments.

Table 10 shows that CHD cost the NHS almost £500 million in England and Wales in 1987. To put a contemporary perspective on the figure, resources of this magnitude would be required to treat 33,000

AIDS patients between diagnosis and death (assuming the cost of care per case to be the mean of the frequently quoted range of £10,000–£22,000) – about 15 times the number of diagnosed cases to date. Furthermore, it is possible that the CHD costs estimated here understate the true resource burden. For example, in the hospital sector both medical and surgical costs for CHD patients may be higher than the average expenditure figures employed to obtain the estimate shown in Table 10. It should also be recognised that a more comprehensive analysis than is possible in the present paper would need to include the costs of community nursing and social services for CHD patients, reductions in the quality of life for affected individuals and their families and the broader economic losses to the nation resulting from the premature exit of some CHD sufferers from the labour force.

Table 12 **Risk factors for coronary heart disease.**

	Characteristic	*Effects on the risk of coronary heart disease*
Principal risk factors	Smoking (cigarettes)	The greater the amount smoked currently, the greater the risk.
	Blood pressure	The higher the pressure the greater the risk.
	Blood cholesterol	The greater the concentration the greater the risk.
	Diabetes	People with diabetes have a higher risk.
	Family history	The longer parents live, the less the risk to their children.
	Obesity	Being overweight may increase the risk (unproven).
	Stress	Stress may increase the risk (unproven).
	Personality	Some types may be more prone than others (unproven).
	Physical activity	The less exercise customarily taken, the greater may be the risk (unproven).
	Hardness of tap water	The softer the tap water the greater may be the risk (unproven).

Source DHSS, 1981.

REDUCING THE BURDEN

Epidemiological and related evidence suggests that a reduction in the substantial resource burden outlined above is a perfectly feasible objective. A number of risk factors are associated with the occurrence of CHD (Table 12) and there is a broad consensus that appropriate lifestyle and other changes offer substantial potential for reducing the incidence of the disease. In this regard, a long-term study of the 'CHD experience' of 75,000 to 94,000 males employed by a large company in the United States between 1957 and 1983 reported a 28 per cent reduction in major coronary events (Pell and Fayerweather, 1985). This decline was concluded to have derived principally from a downward trend in CHD incidence and this in turn reflected a growing trend towards 'doing the right things' in the context of CHD prevention (Stamler, 1985).

International comparisons of CHD mortality patterns and lifestyles also underscore the significance of the risk factor concept. Marmot (1985), in a review of international CHD trends, has emphasised that shifts in mortality rates may result from alterations in case-fatality (attributable perhaps to treatment advances or changes in natural history) as well as from movements in incidence. In addition, the review highlighted a number of puzzling findings – for example, the apparently contradictory trends in lifestyle and CHD mortality in Sweden – and observed that 'the difficulty with multifactorial explanations is that they can be stretched and twisted, *post hoc*, to cover almost any set of data'. Nevertheless, Marmot concluded that 'it would be a remarkable accident if the discussed changes in diet, smoking, blood pressure and physical activity were unrelated to the improved CHD picture in many countries'.

In the specific context of Britain, the authors of the British Regional Heart Study have concluded from their work that 'overall, the levels of the major risk factors commonly encountered in British men have a marked effect on the risk of ischaemic heart disease' (Shaper *et al*, 1985). In recent years, favourable changes have been recorded in some of these factors but it is clear that further improvements on a considerable scale are still needed.

Cigarette smoking

Focusing first on smoking, which has been estimated to account for about one-quarter of coronary fatalities in men and women under 65 years of age, available evidence suggests that the overall risk for smokers of death from CHD is about twice that for non-smokers. Against this background, data from the General Household Survey provide a degree of encouragement. Table 13 shows that the proportion of cigarette smokers in the population fell between 1972 and 1986 from 52 to 35 per cent for males and from 41 to 31 per cent for females. Reductions have been observed for all age groups although among

Table 13 **Prevalence of cigarette smoking by sex and age, 1972 to 1986, Britain.**

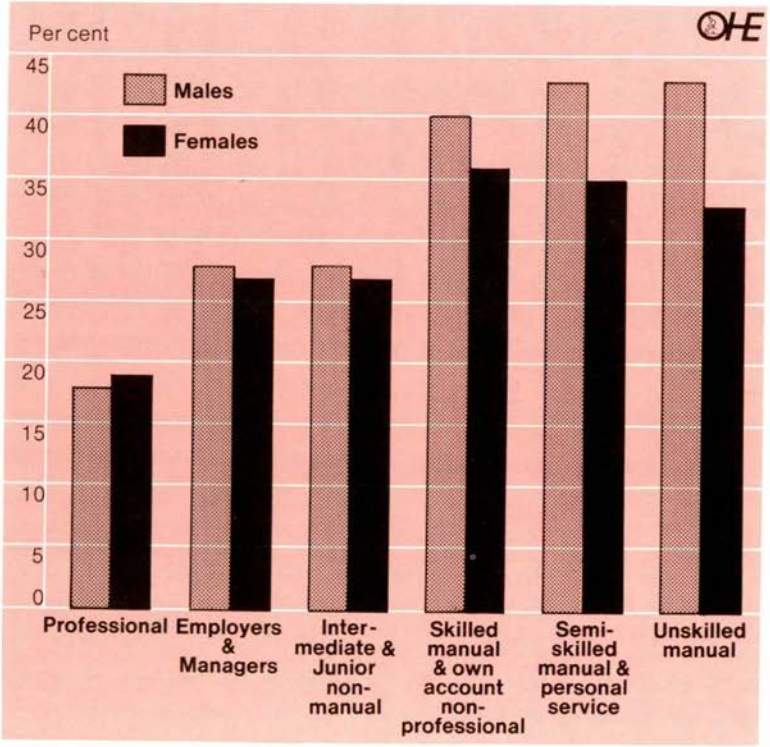
	Men							*Women*						
	Age							*Age*						
Year	*16–19*	*20–24*	*25–34*	*35–49*	*50–59*	*60 & over*	*All 16 & over*	*16–19*	*20–24*	*25–34*	*35–49*	*50–59*	*60 & over*	*All 16 & over*
	Percentage smoking cigarettes							Percentage smoking cigarettes						
1972	43	55	56	55	54	47	52	39	48	49	48	47	25	41
1974	42	52	56	55	53	44	51	38	44	46	49	48	26	41
1976	39	47	49	50	49	40	46	34	45	43	45	46	24	38
1978	35	45	48	48	48	38	45	33	43	42	43	42	24	37
1980	32	44	47	45	47	36	42	32	40	44	43	44	24	37
1982	31	41	40	40	42	33	38	30	40	37	38	40	23	33
1984	29	40	40	39	39	30	36	32	36	36	36	39	23	32
1986	30	41	37	37	35	29	35	30	38	35	34	35	22	31

Source General Household Survey, 1986.

females the declines have neither been as steady nor as marked as among males.

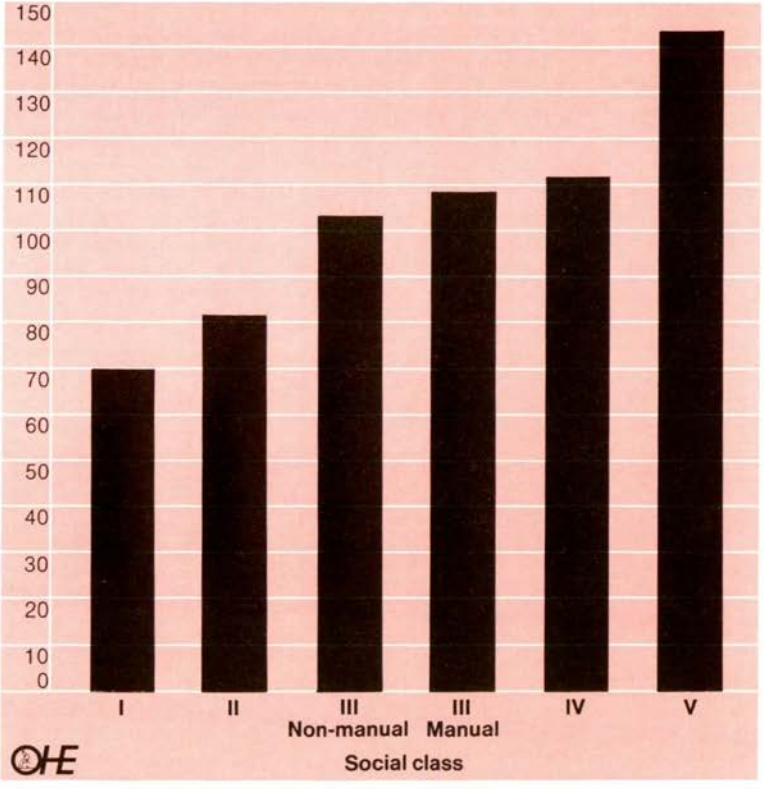
Major areas of concern do nevertheless persist. The fact remains that one person in every three in the population continues to smoke and that about 40 per cent of these individuals may be classified as 'heavy' smokers with a daily consumption of 20 cigarettes or more. In addition, the General Household Survey data reveal substantial disparities in smoking prevalence by social class. Figure 6 shows that only 18 per cent of professional males smoke cigarettes compared with 43 per cent among the unskilled manual grouping and this differential is mirrored in CHD mortality by social class (Figure 7). It is a source of some encouragement that smoking prevalence in the manual group did, in fact, drop by 18 per cent between 1980 and 1986, although counter-balancing this hopeful sign, the 1986 survey found that 55 per cent of unemployed males smoke cigarettes (43 per cent for females) despite

Figure 6 **Cigarette smoking by sex and socio-economic group, Britain, 1986, percentages.**



Source General Household Survey, 1986.

Figure 7 **Standard mortality ratios for CHD, males aged 20–64 years, England and Wales, 1979–83.**



Source OPCS, 1986a.

the fact that this proportion represents a slight drop since the start of the decade.

Yet, arguably the most worrying of recent trends concerns cigarette-smoking habits among children of school age (Table 16). A survey carried out in 1986 showed prevalence to increase with age, so that by their last compulsory year at school 19 per cent of boys and 30 per cent of girls were regular smokers. Overall, 10 per cent of children were categorised in this way. Consumption averaged 48 cigarettes per week but for one in four it increased to 70 per week. From these figures it was estimated that pupils in first through fifth forms in England and Wales in 1986 spent between £0.9 and £1.2 million each week on between 13 and 18 million cigarettes. Against these observations, however, it must be stressed that the prevalence figure for regular smokers in 1986 was one percentage point lower than that recorded by a similar survey

Table 14 **Smoking behaviour among secondary school children in England and Wales, by sex and school year, 1986.**

	Boys						*Girls*					
	1st year	*2nd year*	*3rd year*	*4th year*	*5th year*	*All years*	*1st year*	*2nd year*	*3rd year*	*4th year*	*5th year*	*All years*
Percentage who:												
Have never smoked	86	66	50	45	34	55	82	71	54	37	29	53
Tried smoking once	8	22	28	28	24	23	14	21	23	20	17	19
Used to smoke	3	5	11	12	17	10	1	4	10	18	17	10
Smoke occasionally	3	4	6	7	6	5	2	3	7	7	7	5
Smoke regularly	0	2	5	8	19	7	0	2	6	18	30	12

Source OPCS, Smoking among secondary school children in 1986, HMSO (1987).

undertaken in 1982, although it is too soon to be certain that this indicates the start of a downward trend in cigarette smoking among school children.

Cholesterol

Considerable attention has been directed at the link between diet, cholesterol and coronary heart disease. Diets rich in saturated fats are a cause of elevated serum cholesterol levels; these in turn give rise to the development of atheroma which predisposes to a risk of CHD. Biochemical investigation has demonstrated that cholesterol lipids are an important component of the atheromatous plaques found in diseased coronary arteries, but the key findings have stemmed from epidemiological research.

International population studies (for example, Keys 1970) have revealed a strong positive association between average total cholesterol levels and CHD: communities with high mean plasma cholesterol levels have raised CHD mortality rates. The available data show a high degree of consistency and there appears to be no population in which CHD is common without an accompanying high mean level of total cholesterol (WHO, 1982).

Within populations, a similar relationship is observed. Males suffering inherited hypercholesterolaemia experience an 8- to 10-fold excess risk of CHD (Stone *et al*, 1974). More broadly-based studies also show a clear relationship between CHD risk and cholesterol measures. For example, the Multiple Risk Factor Intervention Trial found among males aged 35 to 57 years at entry that the age-adjusted risks of CHD death in cholesterol quintiles 2 through 5 relative to the lowest quintile were 1.29, 1.73, 2.21 and 3.42 (Stamler *et al*, 1986). A continuous positive relationship between the two variables has also been reported from the British Regional Heart Study. Men in the top fifth of the cholesterol distribution had over three times the risk of those in the bottom fifth (Shaper *et al*, 1986). Furthermore, men in the middle interval for total cholesterol were found to have an estimated twofold risk compared with men at lower levels.

A generally strong positive correlation also exists between the saturated fat content of habitual diets of populations and mean total cholesterol levels. Furthermore, dietary experiments have generated results consistent with this finding (WHO, 1982). However, within populations, support for the relationship is limited. This observation has been regarded as significant negative evidence by critics of the dietary fat hypothesis, although it is more likely to be due to the imprecision of characterising the diet of individuals and the consequent difficulty in detecting small differences in the diet of people within a country (Wald,1987).

The evidence outlined above gives rise to concern about current serum cholesterol levels in Britain. The UK Risk Factor Prevalence Study (Guyer *et al*, 1985) found a mean serum cholesterol value of 5.7 mmol/l in a sample of 4,000 healthy men and women aged 25–59

Table 15 **Recommended average daily intakes of total fat and of saturated* and polyunsaturated fatty acids.**

Category	*Recommended average*
Total fat:	
g per day	77–87[1]
per cent energy	31–35[1]
Saturated fatty acids*:	
g per day	37
per cent energy	15
Polyunsaturated fatty acids:	
g per day	8.6–16.7[1]
per cent energy	3.5–6.8[1]
P/S ratio	0.23–0.45

*Inclusive of trans fatty acids.

1 Depends upon the P/S ratio; the upper limit corresponds to the recommended ratio of approximately 0.45.

Source DHSS, 1984.

years. In men aged 25–39 years, the mean was 5.6 mmol/l, rising to 6.0 mmol/l for those aged 40–59 years. The latter age group was found to have a yet higher average value of between 6.0 and 6.4 mmol/l in the British Regional Heart Study (Shaper *et al*, 1985). In contrast, populations located around the Mediterranean basin and the Orient have mean cholesterol levels ranging from 4.14 mmol/l to 5.17 mmol/l and experience low or intermediate CHD rates (WHO, 1982).

Against this background, a special panel of the Committee on Medical Aspects of Food Policy (COMA) was appointed in 1981 to examine the relationship between nutrition and cardiovascular disease. The report (DHSS, 1984) was published in 1984 and contained the recommendations that the consumption of total fat in the UK should be decreased so that it accounts for 35 per cent of food energy and saturated fats for 15 per cent (Table 15). Employing the cholesterol values found by the UK Risk Factor Prevalence Study as the baseline, Lewis and his colleagues (1986) have calculated that full compliance with the COMA report's recommendations would result in a 12 per cent decrease in the average cholesterol level to 5.0 mmol/l. This figure clearly compares favourably with the target of 5.2 mmol/l or below considered desirable by a number of authorities (WHO, 1982).

Averaged data have, of course, to be interpreted with caution. Even full compliance with the COMA guidelines would still leave 38 per cent of the population with 'excessive' (that is greater than 5.2 mmol/l) cholesterol levels (Lewis *et al*, 1986). Nevertheless, recent consumption trends would appear to be moving in the right direction. For example, the COMA report calculated from 1981 data that milk/cream and butter

accounted for more than one-third of saturated fat in the diet. Between 1981 and 1987 average weekly per capita consumption of these two items have fallen 8 per cent and 42 per cent respectively (CSO, 1989). Furthermore, the annual survey of Household Food Consumption and Expenditure indicates that saturated fat consumption has sustained a long-term reduction, falling to around 41 g/person/day in 1986 (MAFF, 1986). Yet this figure still exceeds the recommended average shown in Table 15. This is also the case with total fat intake (97 g/person/day in 1984) which, despite a sustained absolute decline, has consistently accounted for more than 40 per cent of energy intake since the mid-1960s. In 1986, the proportion was 42 per cent, which was 21 per cent above the level recommended by the COMA Report.

Raised blood pressure

Raised blood pressure is the third major independent risk factor for CHD. In some populations with a high incidence of CHD, the upper 20 per cent of the blood-pressure distribution has a four times greater relative risk of CHD than the lower 20 per cent (WHO, 1982). In the British Regional Heart Study of males aged 40–59 years, 40 per cent of the sample had systolic blood pressures equal to or greater than 148 mm Hg and, as a consequence, experienced twice the risk of major CHD as those at lower levels (Shaper *et al*, 1985). Analysis by diastolic pressure revealed that the top fifth of the distribution (individuals with pressures of 93 mm Hg or more) had a threefold CHD risk relative to those in the bottom quintile. Individuals with pressures between these limits (that is, a diastolic range from 72–92 mm Hg) were at intermediate risk. In other words, the relationship between blood pressure and CHD is continuous.

Surveys in the mid-1970s suggested that 3 per cent of middle-aged males had severely raised blood pressure with systolic and/or diastolic readings greater than 200 mm Hg and 115 mm Hg respectively (Reid *et al*, 1974) and that 26 per cent might be classified as mildly or moderately hypertensive or worse, that is diastolic pressures above 95 mm Hg (Hawthorne *et al*, 1974). In the absence of national monitoring systems, comprehensive data on the number of individuals with differing degrees of hypertension are not available. Nevertheless, extrapolation from the findings of the British Regional Heart Study suggests that over one million British males aged between 40 and 59 years may have diastolic pressures of 93 mm Hg or more. Furthermore, average blood-pressure levels in Britain are high compared to those found in nations with low CHD risk and, given the continuous positive relationship between the two variables, a shift in the population's blood-pressure distribution curve to the left would clearly be beneficial.

The three risk factors described above – cigarette smoking, elevated serum cholesterol levels and high blood pressure – are the major independent determinants of CHD risk. Each one exerts a powerful influence on the probability of the development of the disease irrespective of the presence or otherwise of other factors. In combination the effect is yet more substantial: raised cholesterol levels in conjunction with high

blood pressure and cigarette smoking increase the risk of CHD to more than eight times that for individuals without these factors. Consequently, reduction of these risk factors from the levels currently prevailing in Britain might be expected to yield benefit on a substantial scale.

It would be inappropriate, however, to disregard the potential gains from action on some of the 'secondary' factors identified in Table 12. Although uncertainty surrounds their impact in isolation, interaction with the major risk factors may be significant in influencing the development of CHD. Regular physical activity, for example, appears to be protective against CHD through a number of possible mechanisms. Yet participation rates for rigorous exercise taken during leisure time – the principal opportunity for physical exertion as an ever-increasing proportion of the labour force is engaged in relatively 'sedentary' occupations – are low (Table 16) and have shown relatively little increase over time (OPCS, 1986). Excess body weight constitutes another area for concern. The British Regional Heart Study (Shaper *et al*, 1985) reported a doubling of CHD risk in the top fifth of body mass index relative to the lowest fifth. The significance of excess weight for CHD may lie in its effect in raising blood pressure and serum-cholesterol levels as the study did not find it had independent risk factor status. Yet, irrespective of the precise pathways involved, estimates for 1981 suggest that over 40 per cent of middle-aged men and women in Britain are overweight (DHSS, 1984) and thereby highlight the need for action in this area.

Gaps in risk-factor knowledge

The risk-factor concept would therefore appear to provide a firm basis for intervention to reduce the contemporary levels of morbidity and mortality generated by CHD. It would be misleading, however, to suggest that these factors explain in full, or provide the complete solution to, the problem of heart disease. For example, the mechanisms whereby some of the major risk factors such as cigarette smoking and hypertension predispose to CHD are a source of uncertainty. More significantly, various aspects of the evidence concerning the individual risk factors demonstrate some degree of inconsistency. In the context of cigarette smoking, for example, risk of CHD is reduced when the habit is abandoned and some studies have indicated that there is very little delay before this benefit starts to become apparent. Indeed, the recently published report from the British Cardiac Society (1987) states that the risk declines to almost non-smoking levels after about three years. However, the British Regional Heart Study found a raised CHD risk in men who had given up smoking more than 20 years ago and concluded that 'in relation to CHD, the benefit of giving up smoking is more gradual and less than is widely accepted' (Cook *et al*, 1986). Also in contrast to other investigations, the study failed to observe a dose-relation between the number of cigarettes smoked and the risk of major CHD (Shaper *et al*, 1985).

In the context of dietary factors and CHD aetiology, perhaps one of

Table 16 **Sports, games and physical activities: participation rates in the 4 weeks before interview by age for males and for females, Britain, 1986.**

*Active sports, games and physical activities**	*Age*							
	16–19	*20–24*	*25–29*	*30–44*	*45–59*	*60–69*	*70 or over*	*Total*
Males – Outdoor	Percentage participating in the 4 weeks before interview							
Walking – 2 miles or more (incl rambling/hiking)	11	15	22	23	24	23	16	21
Football	23	17	11	4	1	nil	nil	6
Golf	3	5	5	5	6	4	2	5
Athletics – track and field (incl jogging)	8	7	8	6	2	Ø	nil	4
Fishing	6	3	5	4	3	2	1	3
Swimming (excl public pools)	3	3	3	3	3	2	1	3
Cycling	7	3	3	3	2	1	1	2
Tennis	4	3	2	2	1	1	nil	2
Cricket	2	3	2	2	1	Ø	Ø	1
Bowls	Ø	Ø	1	1	2	2	2	1
At least one activity†								
– excl walking	46	40	38	32	22	13	7	27
– incl walking	51	49	50	45	37	31	20	40
Indoor								
Snooker/billiards/pool	38	35	24	17	11	6	4	17
Swimming	14	14	13	13	5	4	1	9
Darts	22	17	13	10	7	3	1	9
Squash	6	6	9	5	2	Ø	nil	4
Gymnastics/athletics	9	8	5	3	1	nil	Ø	3
Badminton	5	4	3	3	1	Ø	nil	2
Bowls/tenpin	1	3	1	2	3	3	2	2
Table tennis	7	2	2	2	1	Ø	Ø	2
At least one activity†	66	59	51	42	25	14	6	35
Females – Outdoor								
Walking – 2 miles or more (incl rambling/hiking)	16	19	20	21	20	20	7	18
Swimming (excl public pools)	4	4	3	3	2	1	Ø	2
Cycling	3	2	2	2	1	Ø	Ø	1
Athletics – track and field (incl jogging)	4	2	3	2	Ø	nil	Ø	1
Tennis	4	1	1	2	1	Ø	Ø	1
At least one activity†								
– excl walking	20	17	12	13	0	4	2	10
– incl walking	30	32	27	29	24	22	9	24

*Active sports, games and physical activities**	*Age*							
	16–19	*20–24*	*25–29*	*30–44*	*45–59*	*60–69*	*70 or over*	*Total*
Indoor								
Swimming	19	18	19	13	6	4	1	10
Keep fit/yoga	6	10	9	8	4	2	1	5
Darts	6	5	4	4	2	1	1	3
Snooker/billiards/pool	11	8	3	3	1	Ø	Ø	3
Badminton	4	2	3	3	1	Ø	Ø	2
Squash	4	3	3	1	Ø	nil	nil	1
Bowls/tenpin	2	2	1	1	1	1	1	1
Gymnastics/athletics	4	2	2	1	Ø	Ø	nil	1

*Activities are listed in descending order of participation rates for all males/females in 1986. Includes only activities in which at least 1 per cent of males/females participated in the 4 weeks before interview in 1986.

†Total includes those activities not separately listed.

Source General Household Survey 1986.

the most significant areas of debate concerns the role of polyunsaturated (including the essential) fatty acids. Evidence exists for an inverse relationship between consumption of the latter and CHD incidence. Marmot (1985), for example, has shown for the United States that the falling CHD mortality rates have been accompanied by increased consumption of linoleic acid whilst saturated fat intake has changed little. This led to the suggestion that 'these data are far more consistent with a protective effect of essential fatty acids (linoleic acid) or of a high ratio of essential to saturated fatty acids than a harmful effect of saturated fats'. Additional support derives from the recently reported finding of a significant inverse and progressive relationship between adipose-tissue linoleic acid and the estimated risk of angina pectoris and acute myocardial infarction (Wood *et al*, 1987). Furthermore, in a 20-year population study in part of the Netherlands, Kromhout and co-workers (1985) reported that consumption of fish (which is rich in these 'protective' fatty acids) correlated inversely with death due to heart disease.

Polyunsaturated fatty acids reduce serum cholesterol and may have a beneficial influence on platelet function and aggregation, thrombogenesis and the structure and function of the endothelial wall. The low average dietary intake of these acids in the UK may therefore be a factor in the nation's high CHD mortality rates (Kitchin and Turner, 1986). Yet some concern has been expressed that diets rich in polyunsaturated fatty acids may have harmful effects. Against this background, the COMA report committee (DHSS, 1984) made no specific recommendation for increasing the consumption of polyunsaturated

fatty acids, though by reducing the intake of saturated fatty acids, the 'P/S' ratio would be raised and this would tend to decrease serum cholesterol levels.

In addition to the 'difficulties' arising with individual risk factors, it is clear that the concept as a whole does not, in its present form, fully predict CHD occurrence. Of the 20 per cent of healthy adult males aged 40–55 years with the highest CHD risk as a result of raised cholesterol concentrations and blood pressure, two-thirds will remain well over the subsequent 25 years (British Cardiac Society, 1987). Similarly, the UK Heart Disease Prevention Project found that of the subjects in the top 15 per cent of risk, only 7 per cent of those initially free of CHD, and 22 per cent of those with CHD at the start, suffered a myocardial infarction over the next five years (Heller *et al*, 1984). Nevertheless, the majority of individuals suffering CHD do have one or more of the major risk factors – in similar vein, most people who smoke cigarettes do not get lung cancer but the habit accounts for 80–90 per cent of lung cancer, cases (Wald, 1987).

The apparently limited power of the risk factors to predict the development of CHD reflects a combination of factors. Methodological considerations – especially difficulties relating to the accurate measurement of initial risk factor status and in taking account of subsequent changes – may be relevant. It is perhaps unrealistic to expect a few static measurements to explain a risk which depends upon a long, dynamic and variable natural history and thus uncritically to equate statistical with biological explanations for disease.

It is also possible that the so-called secondary risk factors and their interaction with the primary factors ought to be accorded a greater degree of importance. In addition, other potential determinants may need to be assimilated into the risk-factor equation: for example, alcohol consumption (Brenn, 1986), although new data have recently questioned its importance (Shaper *et al*, 1987), and poor nutrition in early life which may raise the susceptibility to the effects of an affluent diet (Barker and Osmond, 1986).

In the context of 'new' risk factors, particular attention has focused on the links between haemostatic function and CHD. Some individuals at apparently high risk for CHD and with moderate obstructive coronary atherosclerosis do not develop symptoms of CHD. The explanation may lie in the absence of acute processes (such as plaque rupture, thrombosis and coronary spasm) which convert silent or occult coronary atheroma into clinical manifestations (British Cardiac Society, 1987). Thrombotic processes may be especially important and the findings of the Northwick Park Heart Study indicate that high levels of Factor VII coagulant activity and of plasma fibrinogen may be key determinants of CHD risk (Meade *et al*, 1986). Indeed, the authors concluded that 'the biochemical disturbance leading to CHD may lie at least as much in the coagulation system as in the metabolism of cholesterol'. The task now is to clarify the determinants of thrombotic risk. Smoking, dietary fat and psychological or social factors such as job dissatisfaction (Markowe *et al*, 1985) may be important in this regard.

Strategic use of risk factors

At the same time as consideration has been given to the means of refining the risk factor concept, attention has also focused on the ways in which current understanding might most effectively be employed to reduce the incidence of CHD. In this context, two broad approaches have been proposed – the high risk and population strategies – and both have accompanying advantages and disadvantages.

The former seeks to identify individuals at high risk for CHD and to reduce their chances of progressing to a clinical event by intervening with suitable preventive measures. Proponents of this approach point to the disappointing results from mass health education trials and to the greater likelihood of individuals responding to advice when they realise they face a heightened risk of disease and are counselled on a one-to-one basis. In addition to encouraging subject (and physician) motivation in this way, the high-risk strategy is argued to represent a more cost-effective deployment of resources as it targets individuals in whom need and potential benefit are at a maximum. Furthermore, this approach may carry a more favourable cost : benefit ratio, since any drawbacks accompanying intervention may be set against gains that are potentially more substantial for high-risk individuals than for those at lower risk. Against this background, Oliver (1983) has argued that 'our strategy towards the prevention of coronary heart disease needs to be reconsidered and should be directed at those identified as being at particularly high risk'.

The high-risk strategy does, however, pose a series of difficult challenges. First, it is, of course, necessary to define high risk and it soon becomes apparent that this is not a 'fixed target'. Focusing on serum cholesterol, for example, the National Institutes of Health in the United States define high risk among subjects aged 40 years and over as a value of 6.72 mmol/l or greater (Anonymous, 1985). This level would identify 10 per cent of Americans in this age range who, on the basis of NIH recommendations, would require intensive treatment by diet and, in the event of an inadequate response, pharmaceutical therapy. Application of this cut-off point in Britain would imply that 31 per cent of males aged 40–59 years warrant intervention in this way (Shaper and Pocock, 1985). It has therefore been proposed that the best policy would be to aim to identify 'those above the 80th percentile of the distribution of serum cholesterol concentrations or blood pressure for that population for which it is planned to give advice' (Oliver, 1987).

The next problem to be addressed concerns the means of identifying persons at high CHD risk. One option would be universal screening but, apart from many other considerations, this could be extremely expensive. For example, Lloyd (1987) has calculated that the cost of a basic analysis to determine total cholesterol level, including an allowance for recall testing of the top 20 per cent of the population, is £5.40. This figure rises to £16.80 for more comprehensive analyses. Consequently, screening the male adult population aged between 15 and 59 years could cost between £82 million and £254 million. Furthermore, these figures exclude any allowance for the costs of counselling and treating

those found to be hyperlipidaemic and the expense of follow-up monitoring in subsequent years.

Universal screening of this type would also generate other difficult issues including, for example, what action to take for the large number of people found to be at intermediate risk, that is between, say, the 50th and 80th percentiles of the risk-factor distributions. In view of such problems, it has been argued that case-finding represents a more viable option than universal screening. The former might be approached in a number of ways. One possibility would be to employ family history data and screen all the first-degree relatives of patients who had developed clinical CHD under the age of 50 years (Oliver, 1987). An alternative strategy might be to offer additional 'tests' during patient-initiated visits to healthcare professionals (Holland and Breeze, 1987). In this regard, Shaper and his colleagues (1988) have devised a simple package of measures for use on an opportunistic basis in general practice which can help to identify individuals vulnerable to CHD (Table 17). From the British Regional Heart Study findings, it is calculated that a risk score based on the factors shown in the table will identify individuals in the top fifth of the risk distribution who collectively will contribute 54 per cent of the major cases of CHD over the subsequent five years.

A final issue that obviously has to be examined in evaluating the worth and practicalities of high-risk screening concerns the benefits of intervention. Assessment in this area is extremely complex, not least because the majority of individuals in the top quintile of risk will in fact remain free of clinical disease. Further confusion stems from the fact that intervention trials in high-risk subjects identified by risk factors have produced variable results (Tunstall Pedoe, 1987). For example, the American Multiple Risk Factor Intervention Trial (MRFITRG, 1982), involving high risk male volunteers, generated inconclusive results: the opposite views that risk-factor control is totally useless or fully effective were both capable of being supported by the findings (Rose, 1985).

Table 17 **Simple formula for identifying individuals at high CHD risk.**

7.5 × number of years spent smoking ('smoking years')
plus 4.5 × systolic blood pressure (average of two readings)
plus 265 if a man recalls a doctor diagnosis of CHD
plus 150 if current angina (chest pains on exertion)
plus 80 if parent died of 'heart trouble'
plus 150 if he is diabetic

Those scoring more than 1,000 are in the top 20 per cent of the distribution of the risk score.

Source Shaper, 1988.

Focusing specifically on pharmaceutical treatment, a recent review of trials (Mitchell, 1987) suggests a comparable absence of unequivocal guidelines in the specific context of CHD prevention. Inevitably, professional views differ on the thresholds for intervention and on the relevant benefits and costs that should be taken into account in assessing whether or not to institute therapy. Nevertheless, the British Cardiac Society (1987) considers that medicines may be required to treat very high levels of serum cholesterol and blood pressure and that individuals who might benefit from such therapy will be found among the top 10 per cent and top 20 per cent of the distributions for blood pressure and cholesterol respectively.

The population approach

In contrast to the high-risk strategy, the population approach seeks to reduce the incidence of CHD by lowering the mean level of risk factors in the population as a whole. For each of the main risk factors most of the attributable cases of CHD occur among that large section of the community with only average values (Rose, 1981). Thus the UK Heart Disease Prevention Project found that the top 15 per cent of the risk distribution predicted only 32 per cent of the subsequent cases of myocardial infarction (Heller *et al*, 1984). Similarly, the British Regional Heart Study reported that the high fifth segment of the distribution for any major risk factor did not yield more than one-third of the myocardial infarctions and cases of sudden death which occurred over the subsequent four to five years. These findings reflect the observation that 'a large number of people at a small risk may give rise to more cases of disease than the small number who are at high risk' (Rose, 1985a). The latter is illustrated in Table 18 by data from the British Regional Heart Study relating to blood pressure.

Within a population the distribution of a given risk factor may create

Table 18 **CHD cases associated with diastolic blood pressure values.**

Distribution Quintile	*Blood pressure mm Hg*	*Annual cases per 1,000 males aged 40–59*	*Male population aged 40–59 in E + W divided into 5 equal groups, 000's*	*Number of Cases*	
1	<72	3.39	1,117.24	3,787	
2	72–	5.69	1,117.24	6,357	67%
3	78–	5.23	1,117.24	5,843	
4	85–	6.31	1,117.24	7,050	
5	93–	10.47	1,117.24	11,698	33%
				34,735	

Source Calculated from results of the British Regional Heart Study (Shaper *et al*, 1985).

the impression that most people have 'normal' values. However, normal is not necessarily synonymous with acceptable or safe. International comparisons may show that in reality the apparently normal value represents a high level of absolute risk. For example, serum cholesterol values regarded as low in Finland would be considered high in Japan (Rose and Shipley, 1985). In such circumstances, a preventive strategy that succeeds in reducing the average population value for the risk factor may therefore be expected to yield substantial reductions in morbidity and mortality. Rose (1985) has calculated from the Framingham data, for example, that a 10 mm Hg lowering of the blood pressure distribution as a whole would correspond to a 30 per cent reduction in total attributable mortality. And in a new overview analysis of epidemiological data, Peto (1986) has estimated that a 10 per cent reduction of serum cholesterol throughout adult life would reduce CHD by about one third.

In addition to the magnitude of the savings in morbidity and mortality, the mass approach to prevention offers a number of other advantages over the high risk strategy. The objective is to remove the underlying causes of ill-health by changing society's norms to behaviour and may be illustrated in the context of cigarette smoking. Until the mid-1970s more than half of the British male population smoked cigarettes – smoking was the norm – but since then the proportion has fallen steadily and progress is being made towards a society in which non-smoking is considered normal. Success in this area and elsewhere should eventually diminish the need for sustained health education initiatives as more appropriate lifestyles become the accepted pattern. In contrast, because it fails to address the problem at source, the high risk strategy will always be needed to protect CHD vulnerable individuals inevitably present in each succeeding generation (Rose, 1985a).

At the same time, however, it is clear that a number of problems confront the mass approach to CHD prevention. In some instances, these difficulties are in essence the obverse of the advantages claimed for the high-risk strategy – for example, in the areas of cost-effectiveness and intervention cost-benefit ratios. But perhaps the main drawback stems from the fact that the mass approach yields only a small benefit to each individual. Although persons at only moderate CHD risk as a group generate more cases than those classified at high risk, their more favourable risk status means that individually most of them would be expected to remain well, at least for many years, irrespective of whether or not they choose to participate in programmes designed to reduce the overall toll of CHD. This observation has been characterised by Rose (1981) as the Prevention Paradox: 'A measure that brings large benefits to the community offers little to each participating individual'. The latter implies that risk portrayal is less likely to succeed in bringing about reductions in CHD incidence than initiatives highlighting and making possible the 'social rewards of enhanced self-esteem and social approval' (Rose, 1985a) which flow from the pursuit of lifestyles appropriate to tackling the 'causes' of CHD at root.

CONCLUSION

The high-risk and population strategies have sometimes been presented as alternative means to preventing CHD. It is clear, however, that the two approaches are not mutually exclusive. 'Each strategy has its advantages and disadvantages. The benefits of successful mass control are likely to be considerable but they will be hard to achieve. The return on selective control is likely to be limited but less difficult to realise, even though it implies requirements for screening. One is an ambitious, long-term policy whose main beneficiaries are likely to be the young and the unborn. The other has more limited objectives for here and now' (*Lancet*, 1983). Consequently, the recent report from the British Cardiac Society specifies both 'altering the mass characteristics of lifestyle which are the underlying causes of mass disease' and 'identifying and helping individuals at special risk within the population' as essential elements of a prevention strategy.

Within the framework of a twin approach to CHD prevention, a number of desirable developments may be readily identified. Health education campaigns at both national and local levels are necessary to create awareness of, and promote solutions to, mass health problems such as CHD. The Heartbeat Wales prevention programme, for example, is currently demonstrating that widely divergent groups within the community can be persuaded to collaborate in the interests of promoting better health (Sherman, 1986). The programme also aims to encourage CHD prevention activities and policies within the local industrial sector. In the United States, worksite health promotion initiatives have achieved widespread acceptance in recent years and have been shown to have significant potential for reducing hospital in-patient treatment costs (Bly *et al*, 1986).

In a long-term context, health education in schools clearly provides a major opportunity for future reductions in the incidence of CHD. The need for effective action in this area is underlined by the generally disappointing results of risk-factor intervention trials aimed at middle aged males. Flexibility exists with regard to how initiatives might best be integrated into existing curricula but it is essential to ensure that health education is 'given status and importance in the eyes of other teachers and the pupils by making use of senior teachers' (DES, 1986) if it is to avoid drifting into the ranks of a peripheral-interest activity. The DES consultative document also emphasised that 'schools should avoid practices which appear to run counter to the principles of good health education'. School meals, for example, should encourage healthy eating habits. In a similar but much broader context, other commentators have pointed to the need for the relevant policies of all government departments, notably the Ministry of Agriculture, to maintain consistency with the stated objectives of the Department of Health in the field of health promotion (Canterbury Report, 1983; *Lancet*, 1986).

General practice offers unique opportunities for both short and long-term prevention programmes. At the recent launch of a new initiative

aimed at enhancing the potential contribution of primary healthcare professionals, it was estimated that up to 40 per cent of premature CHD deaths in England and Wales could be avoided by appropriate action in this setting (Prentice 1987). Approximately 75 per cent of the population consult a general practitioner at least once each year and this proportion approaches 100 per cent over a five-year period. Each weekday, almost one million people in the UK visit their doctor. In addition to this high frequency of contact, research indicates that the public expects general practitioners to be actively interested in prevention (Wallace and Haines, 1984) and attaches a high degree of credibility to the advice provided by this source (McCron and Budd, 1979). Furthermore, evidence exists for the effectiveness and economy of some preventive interventions at the primary care level. With regard to smoking, for example, Jamrozik and colleagues (1984) have demonstrated the efficacy of the general practitioner's counsel in helping people to stop the habit and Williams' (1987) calculations suggest that advice in this particular area represents an extremely cost-effective approach to CHD prevention (Table 19).

At the present time, however, it is clear that the primary healthcare team's potential contribution to reducing the incidence of CHD is not being fully realised. For example, Fowler (1986) has reported that fewer than half of general practice records contain a recent blood-pressure measurement In contrast, Friedewald (1987) has estimated that 72 per cent of Americans have had their blood pressure checked within the last six months (on the basis of 1982 data). In addition, fewer than a

Table 19 **Relative costs per Quality Adjusted Life Year.**

Procedure	*Cost per QALY £*
Advice by GPs to stop smoking	180
Pacemaker implantation for atrioventricular heart block	700
Valve replacement for aortic stenosis	900
CABG for severe angina with LMD	1,040
CABG for severe angina with 3VD	1,270
CABG for moderate angina with LMD	1,330
Action by GPs to control hypertension	1,700
Action by GPs to lower serum cholesterol	1,700
CABG for severe angina with 2VD	2,280
CABG for moderate angina with 3VD	2,400
PTCA for severe angina with 1VD	2,400
CABG for mild angina with LMD	2,520
Heart transplantation	8,000

CABG = coronary artery bypass grafting. 1VD = 1 vessel disease. 2VD = 2 vessel disease. 3VD = 3 vessel disease. LMD = left main disease. PTCA = Percutaneous transluminal coronary angioplasty.

Source Williams, 1987.

quarter of general practice records in the UK contain information on smoking habits, and a yet smaller proportion mention diet, weight or alcohol consumption (Fowler, 1986). The explanations for these shortcomings include too little emphasis in medical education on preventive medicine and the time constraints encountered in general practice (Fowler, 1986a). Solutions to these problems are available – in the latter instance, for example, by extending the role of practice nurses – and must be acted upon if general practice is to realise its potential in CHD prevention: about 75 per cent of persons aged 35–64 years on a general practitioner's list have at least one major risk factor for CHD, 25 per cent have two and 10 per cent are at risk from all three (Fowler, 1986).

At the same time as health promotion/disease prevention strategies are being pursued, there is also a continuing need for investigation into the causes and treatment of CHD. With regard to the former, the British Cardiac Society (1987) has highlighted the importance of more research into areas such as thrombogenic risk and its determinants, the genetic influences on the susceptibility to and protection from atherosclerosis and CHD and ventricular fibrillation and sudden cardiac death. Better understanding in all of these areas may be expected to fill in some of the gaps that exist in the risk-factor concept at present and lead to improvements in its powers of prediction.

Turning to treatment, it is possible that therapeutic interventions have played some role in the recent modest declines in CHD mortality rates in England and Wales. Increasing recognition of the hazards of high blood pressure may be relevant – GP consultation rates for hypertension among males aged 40–64 increased 47 per cent between 1978 and 1988 (IMS, 1987) – and new opportunities have arisen for reducing post-myocardial-infarction fatality rates. The combined results of 24 randomised trials suggest that the use of beta-blocker medicines after infarction may reduce subsequent mortality by about 20 per cent (Peto, 1985). In addition, the number of coronary artery bypass grafts performed each year increased from 3,200 in 1978 to 11,466 in 1986 (DHSS, 1989). Whilst the impact of these and other developments on mortality rates remains uncertain, it is nevertheless clear that further research is necessary not only to refine existing and develop new techniques (such as balloon and laser angioplasty for compressing or dissolving atherosclerotic occlusions) but also to identify which patients will most benefit from such interventions in order to ensure a cost-effective allocation of scarce health care resources.

The objective of this paper has been to demonstrate the need for new initiatives to tackle coronary heart disease in this country. In England and Wales the disease is currently costing the nation four premature deaths and the National Health Service more than £55,000 each hour of every day throughout the year. The recent experience of other nations suggests that resource waste on this scale can be avoided and concerted action along the pathways outlined in this paper is now needed if England and Wales and the other countries of the UK are not to be further isolated at the top of the international CHD mortality league table.

REFERENCES

Anonymous (1985). *JAMA*, 253, 2080–2086.

Armstrong A, Duncan B, Oliver M F, Julian D G, Donald K W, Fulton M, Lutz W and Morrison S L (1972). *Br Heart J*, 34, 67.

Barker D J P and Osmond C (1986). *Lancet*, 1, 1077–1081.

Bly J L, Jones R C and Richardson J E (1986). *JAMA*, 256, 3235–3240.

Brenn T (1986). *J Epid Comm Hlth*, 40, 249–256.

British Cardiac Society (1987). Report of a Working Group on Coronary Disease Prevention.

Canterbury Report (1983). Coronary Heart Disease – Plans for Action. Pitman Publishing Ltd.

Central Statistical Office (1987). Social Trends, 17. London: HMSO.

Cook D G, Shaper A G, Pocock S J and Kussick S J (1986). *Lancet*, 2, 1376–1380.

Davies M J (1982). *Hospital Update*, March issue, 317–322.

Department of Education and Science (1986). Health Education from 5 to 16. Curriculum Matters 6. London: HMSO.

Department of Health and Social Security (1981). Prevention and Health: Avoiding heart attacks. HMSO.

Department of Health and Social Security (1984). Diet and Cardiovascular Disease. Report on Health and Social Subjects 28. London: HMSO.

Department of Health and Social Security (1987). Press release, 14 January, (87/21).

Dobbs J and Marsh A (1984). Smoking among secondary school children in 1984. OPCS.

Eisenberg M S, Bergner L, Hallstrom A P and Cummins R O (1986). *Scientific American*, 254, 25–31.

Fowler G (1986). *The Practitioner*, 230, 859–864.

Fowler G (1986a). *J Roy Soc Med*, 79, 633–636.

Friedewald W T (1987). Implementation in the USA. In: Screening for risk of coronary heart disease. Eds. Oliver M, Ashley-Miller M and Wood D. Chichester: Wiley.

Fuster V and Chesebro J H (1986). *New England Journal of Medicine*, 315, 1023–1025.

Guyer B, Lewis B, Mann J I, Rose L, Shepherd J and Wynder A (1985). UK National Risk Factor Prevalence Study interim results. Atherosclerosis VII symposium, Melbourne, Australia.

Hawthorne V M, Greaves D A and Beevers D G (1974). *Brit Med J*, 3, 600.

Heller R F, Chinn S, Tunstall Pedoe H D and Rose G (1984). *Brit Med J*, 1, 1409–1411.

Holland W W and Breeze E (1987). Implementation in the UK. In: Screening for risk of coronary heart disease. Eds Oliver M, Ashley-Miller M and Wood D. Chichester: Wiley.

Intercontinental Medical Stastistics (1987). Private Communication.

Jamrozik K, Vessey M, Fowler G, Wald N, Parker G and Van Vunakis H (1984). *Brit Med J*, 1, 1499–1503.

Kitchin A H and Turner R W D (1986). *Lancet*, 2, 344.
Kromhout D, Bosschieter E B and de Lezenne Coulander C (1985). *N Eng J Med*, 312, 1205–1209.
Lancet (1983). Diet and ischaemic heart disease, 2, 317–319.
Lancet (1986). Britain needs a food and health policy: the government must face its duty, 2, 434–436.
Lewis B, Mann J I and Mancini M (1986). *Lancet*, 1, 956–959.
Lloyd J K (1987). Testing for risk factors: methods, invasiveness and cost, (i) Lipids. In: Screening for risk of coronary heart disease. Eds Oliver M, Ashley-Miller M and Wood D. Chichester: Wiley.
Markowe H L J, Marmot M G, Shipley M J, Bulpitt C J, Meade T W, Stirling Y, Vickers M V and Semmence A (1985). *Brit Med J*, 291, 1312–1314.
Marmot M G (1985). *Acta Med Scand* (Suppl), 701, 58–65.
McCron R and Budd J (1979). Communication and health education. Leicester University Centre for Mass Communication Research.
Meade T W, Mellows S, Brozovic M, Miller G J, Chakrabarti R R, North W R S, Haines A P, Stirling Y, Imeson J D and Thompson S G (1986). *Lancet*, 2, 533–537.
Medical Research Council (1985). *Br Med J*, 291, 96–104.
Ministry of Agriculture, Fisheries and Food (1986). Household Food Consumption and Expenditure 1984. London: HMSO.
Mitchell J R A (1978). *Br Med Bull*, 34, 103–106.
Mitchell J R A (1987). What effect will screening have on CHD incidence? In: Screening for risk of coronary heart disease. Eds Oliver M, Ashley-Miller M and Wood D. Chichester: Wiley.
Multiple Risk Factor Intervention Trial Research Group (1982). *JAMA*, 248, 1465–1477.
Office of Population Censuses and Surveys (1985). General Household Survey 1983. London: HMSO.
Office of Population Censuses and Surveys (1986). Mortality Statistics, DH1, No 16. HMSO.
Office of Population Censuses and Surveys (1986a). Registrar General's Decennial Supplement on Occupational Mortality 1979–83. London: HMSO.
Oliver M F (1983). *Lancet*, 2, 37–38.
Oliver M F (1987). Problems and Limitations. In: Screening for risk of coronary heart disease. Eds Oliver M, Ashley-Miller M and Wood D. Chichester: Wiley.
Pell S and Fayerweather W E (1985). *N Eng J Med*, 312, 1005–1011.
Peto R (1985). Discussion on page 192 of The Value of Preventive Medicine. Eds Evered D and Whelan J. Ciba Foundation Symposium 110. London: Pitman.
Peto R (1986). Blood cholesterol, CHD and non-CHD mortality: evidence from all available randomised trials, reviewed in their epidemiologic context. Paper presented to a meeting on lipoproteins and atherosclerosis held in Helsinki in October.
Pisa Z and Uemura K (1982). World Health Statistics Quarterly, 35, 11–47.

Prentice T (1987). 'New heart disease project could save 60,000 lives a year'. *The Times*, 5 March.
Reid D D, Brett G Z, Hamilton P J S, Jarrett R J, Keen H and Rose G (1974). *Lancet*, 1, 469–473.
Rose G (1981). *Brit Med J*, 1, 1847–1851.
Rose G (1985). Role of controlled trials in evaluating preventive medicine procedures. In: The value of preventive medicine. Ed Evered D and Whelan J. Ciba Foundation Symposium 110. London: Pitman.
Rose G (1985a). *Int J Epid*, 14, 32–38.
Rose G and Shipley M (1986). *Brit Med J*, 2, 306–307.
Ross R (1986). *New England Journal of Medicine*, 314, 488–500.
Royal College of General Practitioners (1986). Morbidity Statistics from general practice 1981–82, third national survey. London: HMSO.
Shaper A G (1986). *Modern Medicine*, December issue, p9.
Shaper A G and Pocock S J (1985). *Br Med J*, 2, 480–481.
Shaper A G, Cook D G, Walker M and Macfarlane P W (1984). *Br Heart J*, 51, 595–605.
Shaper A G, Pocock S J, Walker M, Phillips A N, Whitehead T P and Macfarlane P W (1985). *J Epid Comm Hlth*, 39, 197–209.
Shaper A G, Pocock S J, Phillips A N and Walker M (1986). *Br Med J*, 2, 474–479.
Shaper A G, Phillips A N, Pocock S J and Walker M (1987). *Br Med J*, 1, 733–37.
Sherman J (1986). *The Health Service Journal*, 15 May issue, p 667.
Stamler J (1985). *N Eng J Med*, 312, 1053–1055.
Stamler J, Wentworth D and Neaton J D (1986). *JAMA*, 256, 2823–2828.
Stehbens W E (1985). *Lancet*, 2, 639–642.
Stone N J, Levy R I, Fredrickson D S and Verter J (1974). Circulation, 49, 476, 488.
Tunstall-Pedoe H D (1987). Yield versus effort. In: Screening for risk of coronary heart disease. Eds Oliver M, Ashley-Miller M and Wood D. Chichester: Wiley.
Tunstall-Pedoe H, Clayton D, Morris J N, Brigden W and McDonald L (1975). *Lancet*, 833–838.
Uemura K and Pisa Z (1985). *World Health Statistics Quarterly*, 38, 142–162.
Wald N (1987). Private Communication.
Wallace P G and Haines A P (1984). *Brit Med J*, 2, 534–536.
Wells N E J (1982). Coronary Heart Disease, the scope for prevention. London: Office of Health Economics.
Williams A (1987). Screening for risk of CHD: is it a wise use of resources? In: Screening for risk of coronary heart disease. Eds Oliver M, Ashley-Miller M and Wood D. Chichester: Wiley.
Wood D A, Riemersma R A, Butler S, Thomson M, Macintyre C, Elton R A and Oliver M F (1987). *Lancet*, 1, 177–183.
World Health Organisation (1982). Prevention of Coronary Heart Disease. Technical Report Series 678. Geneva: WHO.