*Cyril Fernando Memorial Oration*

# Major Risk Factors of Coronary Heart Disease in Sri Lankans

SHANTHI MENDIS*

*Journal of the Ceylon College of Physicians,* 1991, **24**, 17-34

Dr. Cyril Fernando, one of Sri Lanka's greatest physicians was born in Colombo on the 30th April 1900. He was educated at St. Benedict's College and University College Hospital, London. He graduated MBBS in 1926 and was elected a member of the Royal College of Physicians the same year; no mean achievement. Three years later he passed the London MD examination securing the gold medal for being the most outstanding candidate. He returned to Colombo in 1929 and served at the General Hospital Colombo for quarter of a century where he finally died in November 1955.

He was an eminent physician, a popular teacher of medicine and an active member of the Ceylon Branch of the British Medical Association, being successively its honorary secretary, treasurer, vice-president, and in 1940, president. He was also a frequent contributor to its journal. In 1939 "two cases of coronary disease in early life" was reported in the 36th volume of the Journal of the Ceylon Branch of the British Medical Association[1]. This case report was the first documented reference to coronary heart disease (CHD) in Sri Lanka and was presented by none other than Dr. Cyril Fernando, to whose memory we pay homage in this address. Dr. Cyril Fernando was one of the first in South East Asia to draw attention to the problem of CHD in the tropics. A disease which has become a leading cause of death and disability in some South East Asian countries including our own (fig. 1).

* *Senior Lecturer in Medicine, University of Peradeniya.*

## Coronary risk factors

To the man in the street and his family, a heart attack appears to occur on the whole, as an unexpected event. However CHD rarely comes out of the blue, appearing in a community or an individual without warning. The disease does not occur randomly and it is clear from overwhelming evidence accumulated from prospective studies such as the Framingham Heart Study[2] that there are characteristics or risk factors in communities and in individuals which are strongly associated with CHD.

In 1949 few people outside Massachusetts had ever heard of Framingham. Today, 40 years on, the town has become an international byword in cardiovascular epidemiology. The first recruits of this world famous prospective epidemiological study on CHD, were 5200 men and women who were all free from CHD. As a result of the 40 year followup of this cohort a number of "risk factors"

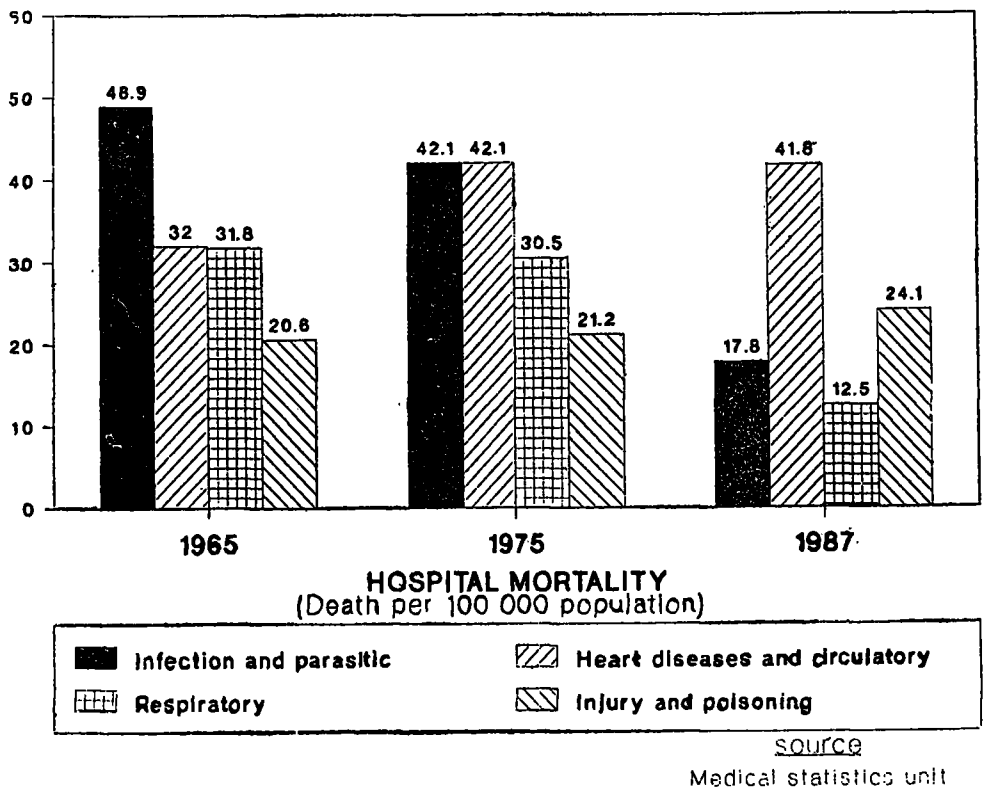


**Fig. 1**

were identified that increased the risk of developing CHD. Although more than half of the original sample are dead, the 6000 children of the initial cohort are currently being followed up. Data from the original cohort and their families, contributed in no small measure to the identification of major risk factors of CHD; hypertension, smoking and blood cholesterol.

However CHD remains a disease for which, to date meaningful mass screening for the overall population has not yet proven feasible. Moreover, often no detectable symptoms are present until a myocardial infarction and/or sudden coronary death occurs. Consequently the medical community must look toward primary and secondary prevention of CHD. Epidemiological research is the life blood of all such preventive strategies. The identification of groups at special risk, and medical and social risk factors such as blood cholesterol levels, blood pressure levels and smoking habits, have been and will continue

to be the mainstay of reducing CHD in the population at large.

What are the levels of coronary risk factors prevalent in Sri Lanka? Unfortunately although it is nearly half a century since the first case of CHD from Sri Lanka was reported[1], we still do not have adequate hard data on coronary risk factors in the general population of our country. Most of the information available on coronary risk factors is derived from hospital data. This information is not applicable to the population at large.

Over the last 13 years I have carried out several investigations to obtain the much needed information on this aspect of CHD. This address entitled 'Major risk factors of coronary heart disease in Sri Lankans" is based on these studies.

## Blood pressure as a major risk factor of CHD

Blood pressure is confirmed as an independent major risk factor for CHD. It has been demonstrated consistently that both systolic and diastolic values of blood pressure are strong, graded and independent predictors of coronary risk[3]. 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. Heart attack risk, seems to be at a minimum only at blood pressure levels below 120/80 mm Hg. These findings lead to the conclusion that high blood pressure contributes significantly to mass CHD.

## Prevalence of hypertension in Sri Lankans

There is very little up to date information on blood pressure levels for the Sri Lankan population and there is no serial data at all. From 1984 to 1986 we screened 10,005 Sri Lankan adults at the Teaching Hospital Peradeniya to obtain data on blood pressure and prevalence of hypertension[4]. They were between 20 and 84 years of age and were visitors to inpatients of the Teaching Hospital, Peradeniya. The majority were from areas in the Central Province and belonged to the middle and lower social classes. 60.6% of the subjects were males and 39.4% were females. The age and sex distribution of the patients screened are shown in table I. Standard blood pressure survey methods as recommended by the WHO were used to measure the blood pressure[5]. The prevalence of elevated blood pressure by age and sex is shown in Table II. The prevalence of hypertension among Sri Lankans was 43.5%. 29.94% had diastolic blood pressure 90-94 mm Hg, 10.56% had diastolic blood pressure levels 95-109

**Table I**

**Age and Sex Distribution of the Study Population Screened for Hypertension**

| *Age in years* | *Male* | *Female* |
|---|---|---|
| less than 21 | 458 (7.5%) | 317 ( 8%) |
| 21 — 30 | 1948 (32%) | 827 (21%) |
| 31 — 40 | 1532 (25%) | 903 (23%) |
| 41 — 50 | 955 (16%) | 820 (21%) |
| 51 — 60 | 700 (12%) | 659 (17%) |
| 61 — 70 | 368 ( 6%) | 315 ( 8%) |
| 71 + | 108 ( 2%) | 95 ( 2%) |
| Total | 6069 (100%) | 3936(100%) |

**Table II**

**Prevalence of Hypertension by Age and Sex (Elevated Blood Pressure per 1000)**

| *Age (years)* | *Male* | *Female* | *Total* |
|---|---|---|---|
| Diastolic ≥ 90 mmHg | | | |
| 20 | 119.9 | 86.1 | 102.1 |
| 21-30 | 194.9 | 108.9 | 169.3 |
| 31-40 | 291.7 | 195.8 | 256.8 |
| 41-50 | 481.2 | 388.6 | 438.9 |
| 51-60 | 498.3 | 486.5 | 492.5 |
| 61-70 | 643.5 | 492.9 | 568.7 |
| 71 + | 544.3 | 568.1 | 552.8 |
| Total | 312.5 | 278.3 | 299.4 |
| Diastolic ≥ 95 mmHg | | | |
| 20 | 0 | 3.7 | 1.4 |
| 21-30 | 43.7 | 32.2 | 37.4 |
| 31-40 | 86.7 | 57.2 | 75.9 |
| 41-50 | 192.4 | 129.5 | 163.7 |
| 51-60 | 246.6 | 208.0 | 227.4 |
| 61-70 | 319.4 | 239.4 | 279.7 |
| 71 + | 227.8 | 318.2 | 260.2 |
| Total | 109.3 | 99.7 | 105.6 |
| Diastolic ≥ 110 mmHg | | | |
| 20 | 0 | 0 | 0 |
| 21-30 | 5.2 | 1.2 | 4.0 |
| 31-40 | 20.4 | 13.7 | 17.9 |
| 41-50 | 49.3 | 41.8 | 45.9 |
| 51-60 | 86.7 | 68.8 | 77.8 |
| 61-70 | 125.0 | 112.7 | 118.9 |
| 71 + | 75.9 | 136.4 | 97.6 |
| Total | 29.8 | 32.3 | 30.8 |

mm Hg. 3.08% had more severe hypertension (DBP 110 mm Hg or above), (fig. 2).

Fig. 3 compares these results with the Community Hypertension Evaluation Clinic (CHEC) Programme conducted by Stamler and others[6] adopting similar methodology. The striking feature is that mild hypertension, which contributes substantially to the incidence of CHD is higher in Sri Lankans when compared to Americans.

**Prevention and control of hypertension**

The prevalence of hypertension appears to differ widely between populations. Strong international correlations are observed with salt intake; average population diastolic pressure appears to increase by 0.8 mm Hg per g of habitual salt intake[7]. The average daily salt intake in Sri Lankans has been reported to be as high as 8.7 g[8]. The high salt intake may be a possible causative factor for the high prevalence of hypertension among Sri Lankans.

Effective antihypertensive therapy had lowered the prevalence of deaths from stroke but worldwide studies demonstrated that the impact of effective antihypertensive drugs on CHD has been minimal[9]. Three hypothetical causes can be suggested for the failure of antihypertensive therapy to achieve its potential benefits in reducing CHD. Firstly it could be the result of commonly used antihypertensive drugs having adverse metabolic effects: adversely affecting lipid metabolism and lipid profiles. Secondly it may be because blood pressure is being inadequately controlled? Lastly, irreversible changes may be occurring in the coronary arteries due to hypertension and therefore it may be necessary to begin treatment earlier in order to bring about a reduction in CHD.

Since heart attack risk seems to be at a minimum only at a blood pressure of 120/80 mm Hg, the large number of Sri Lankans who are borderline or mild hypertensives are at a

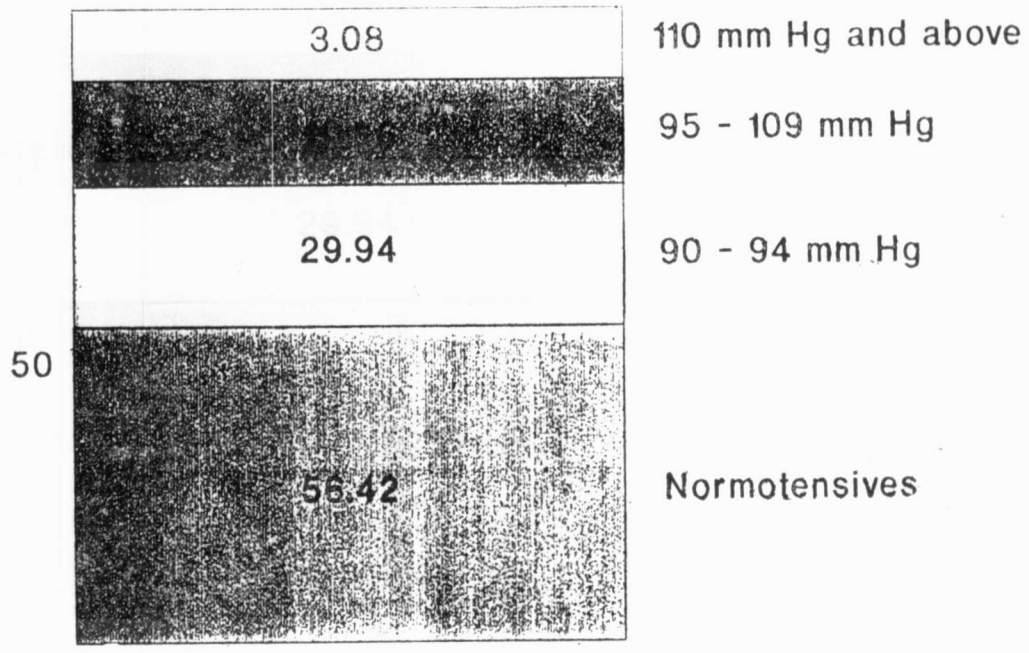


**Fig. 2**
Prevalence of Hypertension in the general population in Sri Lanka

higher risk of developing CHD. As treatment of hypertension does little for coronary risk[9] there is an urgent need to prevent the rise of blood pressure in middle age by measures such as low dietary salt intake and to control other risk factors such as smoking and elevated blood cholesterol levels in hypertensives.

## Tobacco smoking as a risk factor of CHD

Many prospective and retrospective epidemiological studies have shown that cigarette smoking is probably the major modifiable risk factor[2,3]. There is evidence that the influence of smoking is independent of, but also synergistic with other risk factors such as hypertension and high blood cholesterol[2]. This means that the effects are more than additive. Because the incidence of other risk factors for CHD varies in different countries, the relative importance of cigarette smoking also varies. Thus in Japan and Yugoslavia where the key cholesterol factor is at a low level, smoking causes little risk to the heart. Conversely in countries like Sri Lanka the ill effects of smoking are likely to be strongly concentrated in those with hypertension or elevated blood cholesterol levels.

Our studies show a high prevalence and a rising trend of smoking in Sri Lanka. In 1969 Uragoda conducted the first study on tobacco smoking in Sri Lanka[10]. 548 males and 579 females were interviewed in Kandy town in a house to house survey. The prevalence of tobacco smoking reported was 48.2% in males and 1.6% in females.

In 1986 we interviewed 2344 adult males and 1063 adult females from

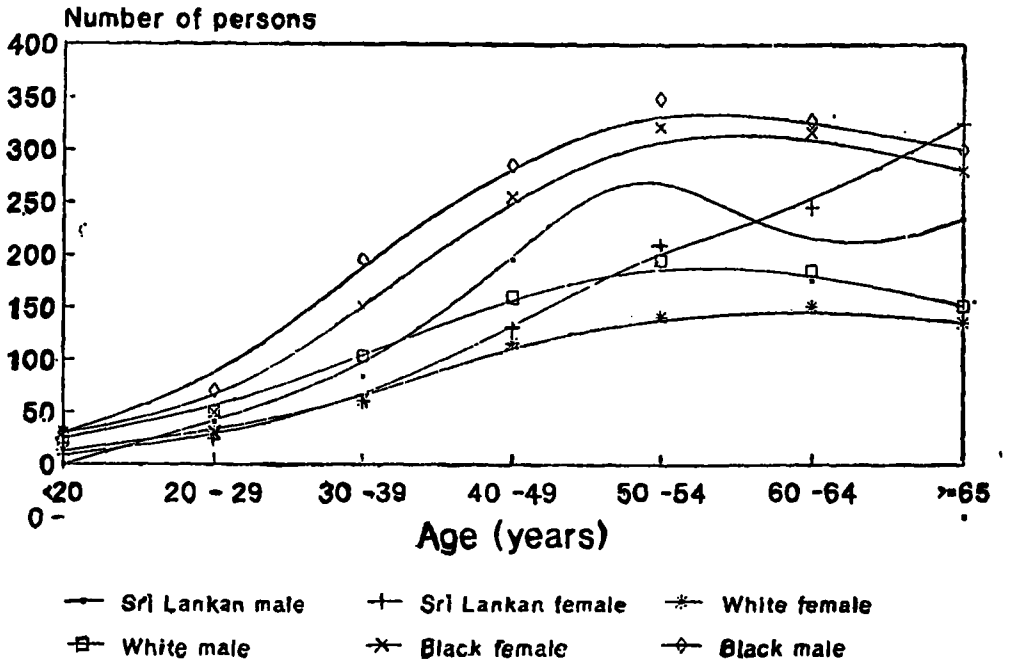


**Fig. 3**

Comparison of prevalence of elevated blood pressure at screening (diastolic≥95 mm Hg). Result of present study compared with community hypertension development program (CHEC)

the same locality in a house to house survey. The comprehensive version of the WHO standardised questionniare was used[11]. It consists of 25 items grouped under (a) social demography-7, (b) smoking behaviour-8, (c) attitudes and beliefs-10. Table III shows the sociodemographic distribution of the subjects interviewed.

**Smoking prevalence and behaviour**

Table IV shows the association between prevalence of smoking and sociodemographic factors. The prevalence of smoking was 71.2% in males and 2.2% in females, indicating a rapidly rising trend of smoking from 1969 to 1986. The average daily consumption by each smoker has also increased from an an average of 7 cigarettes, 8 beedies or 2 cheroots a day in 1969, to 8.3 cigarettes, 8.4 beedies or 1.2 cheroots in 1986. During 1985/1987 we also conducted a house to house smoking prevalence survey on a sample of rural adults (1186 males and 1247 females) from 2 villages close to Kandy[12]. The prevalence of smoking was 57.3% in males and 1% in females: a lower prevalence than in urban adults, in the 1980's but higher than the prevalence of smoking in urban adults in 1969.

**Table III**

**Sociodemographic distribution of the sample**

| | Age in years | | | | | |
|---|---|---|---|---|---|---|
| *Sex* | *19-29* | *30-39* | *40-59* | *60-69* | *Above 69* | *Total* |
| Male | 913(38.9%) | 575(24.5%) | 619(26.5%) | 148(6.3%) | 89(3.8%) | 2344 |
| Female | 436(41.0%) | 243(22.8%) | 271(26.2%) | 73(6.8%) | 33(3.1%) | 1063 |

**Ethnicity**

| Sinhalese | Tamil | Moors |
|---|---|---|
| 76.8% | 7.1% | 12.4% |

**Religion**

| Buddhist | Hindus | Muslim | Christian |
|---|---|---|---|
| 75.6% | 5.6% | 14.0 | 4.2% |

**Table IV**

**Association between prevalence of smoking and sociodemograplic variables**

| *Sociodemographic variable* | *Association* | *Degree of association* | *P* |
|---|---|---|---|
| Ethnicity | Prevalence highest among Sinhalese (74%) | 0.13 | <0.001 |
| Religion | Prevalence highest among Buddhists (74.9%) | 0.17 | <0.001 |
| Education | Prevalence highest among uneducacted (77.3%) | 0.16 | <0.001 |
| Occupation | Prevalence highest among unskilled workers (80%) | 0.27 | <0.001 |
| Marital status | Prevalence highest among widowed men (83.3%) | 0.17 | <0.001 |

In the case of children two studies conducted in school children in the Central Province (Table V) revealed a smoking prevalence of 7.2% in male school children from urban schools[13], and 4.3% in male school children in rural schools[14]: a lower prevalence compared to children from other developing countries[15].

The results of these studies also showed that only a minority (18.6%) of urban smokers had ever attempted to quit smoking and that 86.4% of those who attempted to quit were unsuccessful.

Among male smokers 42.5% and 20% smoked 6 to 10 and 11 to 20 cigarettes or beedies or cheroots a day. In the British Regional Heart Study[3] current smokers had a threefold increase in risk or major ischaemic heart disease when compared with men who had

**Table V**

**Prevalence of Smoking by Age and Locality in Boys (percentage prevalence)**

| *Age* | *Rural (n=931)* | *Urban (n=1122* |
|---|---|---|
| 11 | 1.9 | 1.6 |
| 12 | 0.1 | 1.3 |
| 13 | 1.9 | 1.2 |
| 14 | 4.2 | 3.5 |
| 15 | 3.6 | 6.5 |
| 16 | 4.0 | 9.2 |
| 17 | 10.8 | 13.1 |
| 18 | 11.3 | 12.0 |
| Total | 4.3 | 7.2 |

Chi square 10.65, df, = 1, $p < 0.01$

never smoked. Similar findings have been reported in many other prospective studies. The results of the British Regional Heart Study did not show a significant dose response relationship[3]. This means that light smokers (less than one pack a day) still carry a considerable excess risk.

**Attitudes and beliefs**

The prevalence of tobacco smoking is influenced by many sociocultural factors. Social and cultural factors govern the direction of community norms, attitudes and beliefs towards smoking. These in turn play an important role in the development of individual attitudes and beliefs which are reflected in behaviour patterns. In this study an attempt was made to study community and individual attitudes and beliefs towards tobacco smoking, within the constraints of a structured questionniare.

The responses to the statement "Smoking is harmful to health" were classified into three categories — namely agree, indifferent and disagree (table VI). The majority of both sexes endorsed this statement.

**Concern about harmful effects on their own and other's health:**

For the purpose of analyses, the responses were classified into two categories — concerned and not concerned (table VI). More than half the subjects were not concerned about the effects of smoking on their health or on the health of others.

**Influence of social norms**

The responses regarding social norms against smoking were classified into three categories — discourage, neutral and encourage — for the purpose of analyses (table VI). A significantly higher proportion of males than females (chi square 304.6, df=1, $p<0.001$), and male smokers than non smokers (chi square 151, df=1, $p<0.001$) feel that social norms encourage smoking.

**Control and prevention of smoking**

Since more than half the adult male population in our country are current smokers the impact of smoking on risk of CHD should continue to deserve considerable emphasis. The results of these studies show that although 93.5% of urban adults are aware of health hazards of smoking, the prevalence of smoking is high and that a significant number are not concerned about the ill effects of smoking on self and others. Therefore simple dissemination of information regarding health hazards of smoking per se is unlikely to reduce the prevalence of smoking in Sri Lankans. More insight into psychosocial and cultural factors that are instrumental in starting and maintaining tobacco

**Table VI**

**Attitudes and beliefs of the respondents social norms in relation to tobacco smoking, Sri Lanka**

| Sex | Smoking is harmful to health | | | |
|---|---|---|---|---|
| | *Agree* | *Indifferent* | *Disagree* | *Total* |
| M | 2219(94.6%) | 93(4.0%) | 32(1.4%) | 2344 |
| F | 968(91.1%) | 88(8.3%) | 7(0.6%) | 1063 |

| | Concern about own health | | |
|---|---|---|---|
| | *Concerned* | *Not concerned* | *Total* |
| M | 524(31.4%) | 1145(68.5%) | 1669 |
| F | 5 | 18 | 23 |

| | Concern about others health | | |
|---|---|---|---|
| | *Concerned* | *Not concerned* | *Total* |
| M | 987(59.1%) | 682(40.8%) | 1669 |
| F | 6 | 17 | 23 |

| | Social norms | | | |
|---|---|---|---|---|
| | *Discourage* | *Neutral* | *Encourage* | *Total* |
| M | 739(31.5%) | 908(38.7%) | 697(29.8%) | 2344 |
| F | 406(38.2%) | 637(59.9%) | 20(1.9%) | 1063 |

behaviour in Sri Lankans have to be obtained to devise effective anti-smoking strategies.

### Blood Cholesterol as a risk factor of CHD

The causal relationship between blood cholesterol, CHD and atherosclerosis is firmly established by many lines of evidence[16]. Firstly cholesterol accumulation in the artery wall is characteristic of atherosclerosis and atherosclerosis and CHD can be produced in experimental animals simply by raising their plasma cholesterol levels without introducing any other risk factors. Secondly as shown in the Framingham study[2] and many others[3] there is a clear correlation between risk of CHD and high serum cholesterol levels. Thirdly, Japanese and other populations with low plasma cholesterol levels have a much lower incidence of CHD and their rates tend to rise with migration to USA, indicating that the low incidence is not primarily genetic. Moreover the fact that an elevated low density lipoprotein levels can in itself be a sufficient cause of CHD has been shown by the Noble Prize winning research of Brown and Goldstein[17]. Finally and most important, direct intervention trials such as the Lipid Research Clinic Coronary

Primary Prevention Trial[16] have shown that reducing cholesterol levels concomitantly reduces the risk of CHD.

Interest in serum cholesterol as a risk factor of CHD has been intensified by reports from the US National Heart Lung and Blood Institute[18] and the Study Group of the European Atherosclerosis Society[19]. The medical profession in general remains unsure of the meaning of serum cholesterol findings and what strategy to adopt to deal with them.

In many affluent populations trends in national serum cholesterol levels have been studied[2 3 18 19]. But in Sri Lanka due to lack of financial resources such ambitious and costly studies have not been possible Available data have been collected from hospital patients and volunteer subjects. Three years ago we launched a prospective population study in the Central Province to study the prevalence of CHD and trends in risk factors of the general population in the Central Province. Preliminary data are available from the pilot phase of this study. Serum lipid profile is available on 188 randomly selected males from a village where a prevalence study on CHD was carried out in 1987/1988[20]. They were between 25 and 65 years of age. Plasma was separated from venous blood drawn from subjects after a 14 hour fast and frozen, till lipid analyses was carried out. Total cholesterol was measured using an enzyme kit (Cat No. 816302, Boehringer Mannheim GmbH). The same procedure was used to measure a HDL cholesterol after precipitation of plasma LDL and VLDL by hepairn and 92mM manganese chloride as reported earlier[21]. Triglycerides (TG) were measured using an enzymic test kit (Cat No. 816370, Boehringer Manheim GmbH). All analyses were carried out at the Institute of Pathological Biochemistry. Glasgow, UK with laboratory quality control. The results are shown in table VII. Since this is a relatively small sample it would be premature to arrive at any definite conclusions regarding the contribution of national serum cholesterol level based on these results. However, it is useful to compare the distribution of serum cholesterol levels in this sample with those of the International Project Monitoring Trends and determinants of cardiovascular disease (Monica Project)[22]. Since Scotland has the highest rate of CHD in the world[3] data from the Scottish Monica Project has been used for comparison. As shown in table VIII in the Scottish population aged 25 to 64 years, 75% of men are over the theshhold of 5.2 mmol/1 (200mg/dl), 34% of men are over the threshhold of 6.5 mmol/1 (250mg/dl) and 9% are over the threshhold of 7.8 (300mg/dl). These cut-points are those used by the Study Group of the European Atherosclerosis Society[19]. If the United States cut-point of 6.2 mmol/l (240 mg/dl) is used[18]. 44% of Scottish men are above the level. By comparison with the results reported from the UK our levels appear to be lower. However a little more than half the subjects were above the optimal threshhold of 5.2mmol/l recommended by the European Atherosclerosis Society[19]. About one fifth of the sample had serum cholesterol levels above the U.K. cut-point of 6.5mmol/l and about one third of the sample had serum cholesterol levels above the United States cut-points of 6.2mmol/l.

**Table VII**

**Lipid Profile of 188 Randomly Selected Males 25-65 years of Age**

| | *Mean* | *SD* |
|---|---|---|
| Serum Cholesterol (mmol/l) | 5.19 | 1.20 |
| Serum Triglycerides (mmol/l) | 1.56 | 1.14 |
| Low Density Lipoproteins (mmol/l) | 3.57 | 1.07 |
| High Density Lipoproteins (mmol/l) | 1.07 | 0.29 |

**Table VIII**

**Comparison of Distribution of Serum Cholesterol in Men Aged 25-65 years**

| *Serum Cholesterol* | *Present Study (Sri Lanka 1988/89)* | *Monica Study (Scotland 1986)* |
|---|---|---|
| Over 5.2 mmol/l | 52% | 75% |
| Over 6.5 mmol/l | 22% | 34% |
| Over 7.8 mmol/l | 4% | 9% |
| Over 6.2 mmol/l | 36% | 44% |

Therefore a substantial proportion of the population are at or above cut-points at which American and European cholesterol management algorithms recommend intensive lipid investigations and individual management. Application of imported cholesterol management algorithms based on global cut off points would lead to an overwhelming caseload of patients needing intensive lipid investigation and treatment incurring great costs. This the country cannot afford to do at the present time. More comprehensive random population surveys are needed in order to formulate cholesterol management alogorithms appropriate to our population. The primary intervention for almost everybody—nutritional advice and health education — can be given without knowledge of cholesterol readings. Unless and until more information on national blood cholesterol levels are available and individual advice is available at the the point at which cholesterol measurement might be made, cholesterol screening of the general population cannot be recommended.

**Diet and CHD**

Dietary modification constitutes the basis of all lipid lowering strategies aimed at reducing the risk of CHD. However it is not easy to make lasting modifications of lifestyle whether by means of changing diet giving up smoking or increasing the degree of exercise taken. In order to be successful it is important that the modifications are consistent with the existing cultural and social framework.

They should not include unnecessary restriction or limitations and new dietary risks should certainly not be introduced to the programme.

**Dietary trials with coconut fat**

For many years there has been controversy as to whether coconut fat which is a major component of of the dietary fat in our diet contribute to the high prevalence of CHD in Sri Lankans. Although many opinions had been expressed, when we started our studies on coronary risk factors in 1978 there were no data on the effect of dietary coconut on serum lipid and lipoprotein levels. Since then we have carried out two experimental trials to evaluate the effects of coconut fat on serum lipids and lipoproteins and platelet factors[23 24]. Methodology for analysis of lipids and lipoproteins have been outlined before[21]. In the first study blood lipid values and selected platelet related factors were measured in a group of 16 free living young adults ages 16 to 21, before and 8 weeks after they had been shifted from their usual rice and curry diet to a similar one in which the coconut oil was replaced by whole milk powder and corn oil. The substitution of whole milk powder and corn oil for coconut fat resulted in an increase of daily dietary cholesterol of about 100mg/day (fig. 4). The results of this investigation show that replacing coconut oil with corn oil plus milk fat, in the typical Sri Lankan diet results in a reduction of plasma total cholesterol, free cholesterol, cholesterol ester, plasma phospholipids, low density lipoprotein, and platelet factor four. An inconsistent increase was seen only in serum triglycerides (table IX). These results suggest that the special atherogenic effects of coconut fat that have been demonstrated in so many animal models may be similarly active in humans.

The high density lipoprotein levels have usually been found to decrease when corn oil is substituted for coconut fat[25]. The same effect was observed in the present study. The mean high density lipoproteins cholesterol levels in Sri Lankans appear to be lower than those of Western populations[21]. This may be a genetic[27] or may be related to the low fat and high complex carbohydrate contents of the Sri Lankan diet. Since high density lipoproteins are a protective factor for CHD further lowering of levels may offset the benefits of lowering cholesterol levels. Therefore we proceeded to carry out a more controlled dietary experiment to confirm these results, this time substituting coconut fat with soya milk and soya fat. Twenty five healthy young adults, 20 to 26 years of age participated in the study. None of the study subjects deviated more than 3% from their baseline (day 1) weight throughout the study. Physical activity was constant and none of the subjects received any medications during the study period. The study was divided into two feeding periods of 8 weeks each, a period sufficient to establish the effect of each diet on plasma lipids and lipoproteins. In between the two periods there was a washout period of three weeks to allow the effects of the preceding test diet to dissipate and plasma lipids and lipoproteins to return to baseline levels. The composition of the diets are shown in table X. All men showed a reduction of plasma total cholesterol

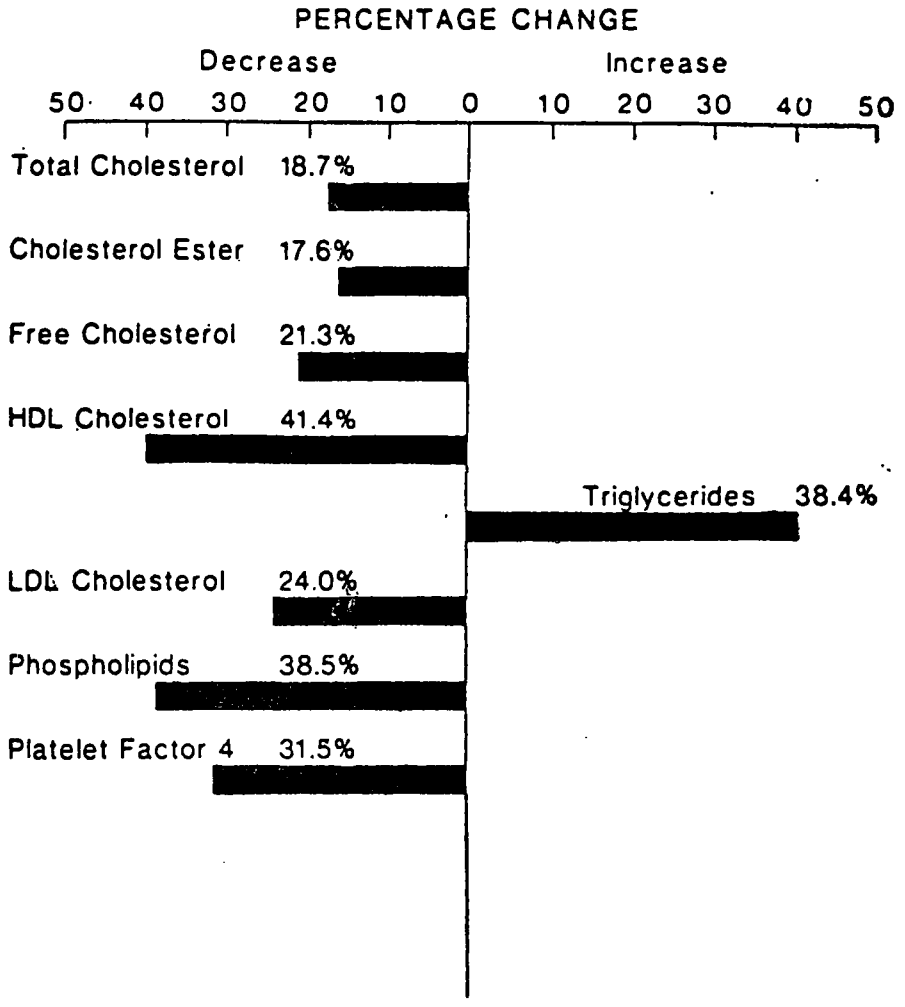


**Fig. 4** Acute effects of replacing coconut oil with cow's milk powder and corn oil in the average Sri Lankan diet (pooled data).

on the soya bean fat diet (table XI). The mean reduction was 21% (range 10 to 35%). All subjects showed a decrease in plasma triglycerides on the soya fat diet (mean decrease 25%, range 13 to 37%). Low density lipoprotein cholesterol fell during the peiod on the soya fat diet by an average of 23% (range 5 to 45%) The soya bean fat diet also lowered the high density lipoprotein cholesterol. The mean reduction was 15% (range 6 to 35%). Reciprocal changes were seen when coconut fat was reintroduced. The results of this investigation are in agreement with several metabolic studies that presented evidence that saturated fat per se increase the concentration of plasma cholesterol and polyunsaturated fat lower plasma total cholesterol in man[25]. Under specific experimental conditions the cholesterol elevating effect of saturated fats has been chiefly attributed their myristic acid (14:0) content[25]. The high myristic acid content of coconut fat (table XII) is likely to be an important factor in the setting of blood

**Table IX**

**Blood, Lipid and Platelet Factor 4 Levels Before and After Replacement of Coconut oil in Sri Lankan Diet**

| | *Phase 1* | *Phase 2* | *t test* |
|---|---|---|---|
| Total Cholesterol mg/dl Mean ± SE | 179.6 ± 9.1 | 146.0 ± 13.4 | $P < 0.05$ |
| Cholesteryl Easter mg/dl Mean ± SE | 125.9 ± 8.1 | 103.6 ± 8.7 | $P < 0.05$ |
| Free Cholesterol | 53.8 ± 1.9 | 42.4 ± 6.5 | $P < 0.1$ |
| Phospholipid mg/dl Mean ± SE | 168.8 ± 11.1 | 103.8 ± 22.0 | $P < 0.025$ |
| Trigylcerides mg/dl Mean ± SE | 38.8 ± 9.4 | 53.6 ± 6.8 | $P < 0.2$ |
| PF4 ng/dl Mean ± SE | 29.8 ± 6.5 | 20.4 ± 2.1 | $P < 0.1$ |
| HDL-Cholesterol mg/dl Mean ± SE | 43.43 ± 5.01 | 25.43 ± 3.95 | $P < 0.025$ |
| LDL-Cholesterol mg/dl Mean ±SE | 131.6 ± 8.9 | 100.3 ± 8.8 | $P < 0.05$ |

**Table X**

**Mean Composition of the Diets (Mean Daily Intake)**

| | *Soya bean fat* | *Coconut fat* |
|---|---|---|
| Energy (kJ) | 10020 | 10150 |
| (kcal) | 2395 | 2425 |
| Protein (g) | 85 | 84 |
| Fat (g) | 78 | 81 |
| Carbohydrate (g) | 350 | 359 |
| Cholesterol | 250 | 250 |
| P: S ratio | 4.0 | 0.25 |

**Table XI**

**Concentration of Plasma Lipids and Lipoproteins During Daily Consumption of Soya Bean Fat or Coconut Fat by Healthy Young Men**

| *Experimental* | *Base Line* | | *Soya Bean Fat* | | *Coconut Fat* | |
|---|---|---|---|---|---|---|
| *Period* | *Mean* | *SD* | *Mean* | *SD* | *Mean* | *SD* |
| Cholesterol (mmol/l) | 4.64 | 0.37 | 3.68*** | 0.42 | 4.6+++ | 0.39 |
| Triacylglycerol (mmol/l) | 1.42 | 0.44 | 1.06* | 0.42 | 1.45 | 0.41 |
| HDL – C (mmol/l) | 1.10 | 0.25 | 0.94* | 0.26 | 1.14 | 0.27 |
| LDL – C (mmol/l) | 2.95 | 0.43 | 2.27*** | 0.36 | 2.84 | 0.37 |

+++ $p < 0.001$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

cholesterol values in the coconut fat phase of the study.

The inter-relationships between diet, blood cholesterol, lipoprotein levels and coronary heart disease are well established. Numerous expert bodies have called for reductions in total fat, saturated fat and dietary salt[27]. In recent years more than 20 independent working parties from many countries have been appointed to review on the relationship of coronary heart disease to diet and to make recommendations. In 1982 the WHO made recommendations to the populations nutrient intakes including limiting saturated fats to less than 10% of calories, total fat to 30% of calories (P/S ratio 1.0), dietary cholesterol to under 300mg per day and dietary salt to 5g per day or less[28]. A few other expert committees feel that the achievement of a P/S ratio of 0.5–0.6 in the community which could be achieved without undue difficulty would be an adequate goal[27].

**Coconut fat in the Sri Lankan diet**

Since the total calories in a diet of a normal healthy adult is about 2250cals, 30% of this (675cals) can be provided by fat. This is equivalent to about 74g of fat. If an average size coconut is used for preparing meals in a five member household each person consumes about 25g of coconut fat. In a diet with a poly-unsaturated to saturated fat ratio of 0.5 about 50g could be from saturated fat. If coconut fat provides half of this quota only another 25g is left for other food rich in saturated fat such as meat and diary products. Therefore if coconut fat is used to prepare a rice and curry meal, inclusion of food items fried in coconut oil or 'pol sambol' or other foods rich in saturated fats would lower the P/S ratio to unacceptable levels. This

would be of particular importance in those with borderline or high blood cholesterol levels and other risk factors. Since available evidence definitely indicates that reducing coconut fat in the diet is an effective way of lowering blood cholesterol, reducing coconut fat in the diet would contribute to the effect of lipid lowering diets and drugs in hypercholesterolaemic individuals.

**Conclusions**

In this paper I have presented information on major risk factors of CHD based on studies conducted over a 13 year period. I do not claim that these studies by themselves are complete. Further research is needed to provide data to fill the gaps in knowledge with regard to national blood lipid levels and trends, smoking trends and other risk factors of CHD.

CHD today plays an enormous burden on the nation both in human and economic terms. Coronary heart disease is significantly influenced by a number of personal and population characteristics and their combinations. These in turn are largely determined by sociocultural factors and are largely modifiable. Such characteristics include elevated blood pressure and blood cholesterol and the associated eating and activity patterns and smoking. There appears to be considerable scope for reducing the incidence of the disease if the risk factors levels are reduced.

To prevent CHD, risk factors have to be controlled in individuals and in the population at large. Control measures applied on a population and those aimed at case finding and treating high risk individuals are complementary and interdependent. Case finding is best achieved by including risk factor assessment in the full clinical examination when individuals seek medical treatment. Individual treatment of risk factors should be guided by their severity and physician's judgement of the patient's total coronary risk, taking age into account. The major components of the populations strategy for prevention of coronary heart disease are improving nutrition, elimination of smoking, control of hypertension and promotion of a healthy lifestyle.

Smoking remains the major avoidable cause of CHD in Sri Lankans. More than half the adult population in Sri Lanka smoke and available evidence indicates that there is a rising trend in cigarette smoking. Clinicians have a major responsibility because they see many of the high risk individuals in whom the cardiovascular effects of smoking are concentrated. The most important treatment for mild hypertension and mild hypercholesterolaemia is to stop smoking and it ought to be regarded by clinicians at least as seriously as pharmacological treatment. A concerted effort to inform and educate the public regarding smoking, and to promote non-smoking as normal behaviour in children should be made. Of utmost importance for prevention and control of smoking is more energetic support from the central government along the lines recommended by the Royal College of Physicians i.e. in the fields of taxation, availability and sales promotion of tobacco products[15].

Even a small reduction in the average blood pressure of the population

could bring about a large reduction in CHD. Since according to present data there is a large burden of mild and borderline hypertensives in the general population, public should be encouraged to reduce the consumption of salt and to avoid other risk factors such as smoking and obesity.

Preliminary data from our studies have shown that blood cholesterol levels of 52% of the population 25 to 65 years old is above the optimal level. Since mass screening for blood cholesterol levels cannot be recommended for several reasons, lowering of the population distribution of blood cholesterol levels is recommended through progressive change in eating patterns. Since the cholesterol content and the total fat content of the average Sri Lankan diet is not unduely high the main adjustment necessary is to reduce the saturated fat content of the diet to below 10% of the energy intake.

Although some cases of CHD occur among people with high risk factor levels, most cases of CHD occur among the large numbers in whom risk factors are moderately elevated. Only a mass (population) approach can help this larger group. But unfortunately this is also the most complex (and expensive) approach to undertake, since it necessitates not only an extensive medical programme but also requires sociological and political change.

**Acknowledgements**

I wish to thank Professor James Shepherd of the Glasgow Royal Infirmary for giving permission to carry out lipid analysis in the Lipid Laboratory of the Glasgow Royal Infirmary, Glasgow, U.K. and Miss Ann Bell and Miss Christine Gourley for performing the analyses. The field workers of the tobacco smoking surveys and the Principals of the schools where children's smoking surveys were carried out are thanked for their cooperation and help. I am grateful to the final year medical students who helped in the epidemiological study of hypertension. The volunteers of the dietary trials from the Minor Seminary Ampitiya and the Open Prison Camp, Pallekale as well as the staff of these institutions are thanked for their participation and cooperation. Mr. Harsha Gamage is thanked for helping to type the script. IFS, Hantana, Kandy and the World Health Organization are thanked for financial and other assistance in these projects.

**REFERENCES**

1. Fernando C. Two cases of coronary disease in early life. *The Journal of the Ceylon Branch of the British Medical Association* 1989; **36**: 251-252.
2. Dawber T R. The Framingham Study. The epidemiology of atherosclerotic disease. Cambridge, Massachusetts: Harvard University Press, 1980.
3. Shaper A G and Pocock S J. Risk factors for ischaemic heart disease in British men. *British Heart Journal* 1987; **57**: 11-16.
4. Mendis Shanthi, Ranasinghe P, Dharmasena B D. Prevalence of hypertension in Sri Lanka. A large population study in the Central Province. *Public Health* 1988; **102**: 455-462.
5. WHO technical report series No 628, Arterial hypertension: report of WHO Expert Committee Geneva: 1978.
6. Stamler J, Stamler R, Reidlinger W F, Alegra G and Roberts R H. Community Hypertension Evaluation Clinic Programme. *J A M A* 1976; **35**: 2299.

7. Gleibermann L. Blood pressure and dietary salt in human populations. *Ecology of Food and Nutrition* 1973; **2**: 143–156.

8. Pallemulle L, Mendis Shanthi, Perera P L A P. Dietary intake of salt in Sri Lankans. *Proceedings of the Kandy Society of Medicine* 1983; **6**: 10.

9. Medical Research Council Working Party. Medical Research Council trial of treatment of mild hypertension: preliminary results. *B M J* 1985; **291**: 97–104.

10. Uragoda C G and Seneviratne B. Tobacco smoking in Ceylon *The Journal of Tropical Medicine and Hygiene*. 1971; iii: 145–147.

11. Mendis Shanthi. Tobacco smoking in an urban population. Proceedings of the 101st Annual Sessions of the Sri Lanka Medical Association 1988; Paper 42.

12. Mendis Shanthi. Tobacco use in a rural community in Sri Lanka. *Ceylon Medical Journal* 1988; **33**: 23-26.

13. Mendis Shanthi. Smoking habits of urban school children. *Proceedings of the Sri Lanka Association for the Advancement of Science* 1989; **12**: 7–8.

14. Mendis Shanthi. Tobacco use in a cohort of children in Sri Lanka. *British Journal of Drug Addiction* 1990; **85**: 397-399.

15. WHO. Smoking and health. Report of a regional seminar Kathmandu, Nepal, August 1984.

16. Lipid Research Clinics Program. The Lipid Research Clinic Coronary Prevention Trial Results. *J A M A* 1984; **251**: 351–364.

17. Brown MS and Goldstein J L. Expression of the familial hypercholesterolaemic gene in heterozygotes. Mechanism for a dominant disorder in man. *Science* 1979; **185**: 61-63.

18. US Cholesterol Education Campaign Expert Panel. Report of the national cholesterol education programme expert panel on detection, evaluation, and treatment of high blood cholesterol in adults. *Archives of Internal Medicine* 1988; **148**: 36-69.

19. Study Group of the European Atherosclerosis Society. The recognition and management of hypercholesterolemia in adults a policy statement of the European Atherosclerosis Society. *European Heart Journal* 1988; **9**: 571–600.

20. Mendis Shanthi. Ischaemic heart disease in 30-59 year males in a total community. *Proceedings of the Sri Lanka Association for the Advancement of Science* 1989; **12**: 7–8.

21. Mendis Shanthi, Read A E. Comparison of serum high density lipoprotein concentration in ischaemic heart disease patients and healthy adult males. *Proceedings of the Sri Lanka Association for the Advancement of Science* 1984; **40**: (1), **5**.

22. WHO Monica project. Geographical variation in the major risk factors of coronary heart disease in men and women aged 35–64, *World Health Statistics Quarterly* 1988; **41**: 115–37.

23. Mendis Shanthi, Weissler R W, Bridenstein R T, Podbielski F J. The effects of replacing coconut oil with corn oil on human serum lipid profiles and platelet derived factors active in atherogenesis. *Nutritional Reports International* 1989; **40**: 4: 773–782.

24. Mendis Shanthi, Kumarasunderam R. The effect of daily consumption of coconut fat and soya bean fat on plasma lipids and lipoproteins of young normolipidaemic men, *British Journal of Nutrition* 1990; **63**: 1–6.

25. Hegsted D M, McGrandy R B, Myers M L. Quantitative effects of dietary fat on serum cholesterol in man. *American Journal of Clinical Nutrition* 1965; **27**: 281-295.

26. Mendis Shanthi, Shepherd J, Packard C, Gaffney D. Genetic variations in the CETP gene and apo AI gene and its relations to coronary heart disease in the Sri Lankan population. *Journal of Atherosclerosis* 1990 (in press).

27. The Coronary Prevention Group. Catering for a healthy diet: London, The Coronary Prevention Group 1985.

28. WHO technical report series No 678. Prevention of coronary heart disease: Report of a WHO expert committee Geneva: 1982.