

**MODIFIABLE RISK FACTORS OF CARDIO VASCULAR
DISEASES
IN ADULTS AT SOOLAMANGALAM VILLAGE,
THANJAVUR
- A CROSS SECTIONAL DESCRIPTIVE STUDY**

Dissertation submitted for

**M.D. Branch XV
Community Medicine**



**THE TAMIL NADU
Dr.M.G.R.MEDICAL UNIVERSITY
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CERTIFICATE

This is to certify that this dissertation titled **MODIFIABLE RISK FACTORS OF CARDIO VASCULAR DISEASES IN ADULTS AT SOOLAMANGALAM VILLAGE, THANJAVUR - A CROSS SECTIONAL DESCRIPTIVE STUDY** is a bonafide work carried out by **Dr.V.V.ANANTHARAMAN** in the Institute of Community Medicine, Madras Medical College, Chennai - 600 003, under my guidance and is being submitted to the Tamil Nadu Dr.M.G.R.Medical University in partial fulfillment of the requirements for the degree of M.D. Branch XV - Community Medicine.

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CONTENTS

Chapter	Title	Page No.
1.	INTRODUCTION	1
2.	OBJECTIVES (AIM)	6
3.	JUSTIFICATION	7
4.	REVIEW OF LITERATURE	10
5.	MATERIALS AND METHODS	24
6.	OBSERVATIONS AND RESULTS	36
7.	DISCUSSION	53
8.	RECOMMENDATIONS	56
9.	SUMMARY AND CONCLUSION	63
10.	LIMITATIONS	65
	ABBREVIATIONS	
	BIBLIOGRAPHY	
	ANNEXURES	
	PROFORMA - ENGLISH	
	PROFORMA - TAMIL	
	MAPS	

STRUCTURED INTERVIEW SCHEDULE

Vanakkam,

I am doing M.D. Community Medicine in the Institute of Community Medicine, Madras Medical College, Chennai - 3. I am conducting a study on modifiable risk factors of cardio vascular diseases in adults at soolamangalam village, Thanjavur A cross sectional descriptive study. I would like your consent and cooperation in this regard.

Thank you,

Yours truly,

INTRODUCTION

Chronic Non - Communicable Diseases (NCDs) are assuming increasing importance among the adult population in both developed and developing countries. The prevalence of chronic diseases is showing an upward trend in most countries for the following reasons.

1. Increasing Life Expectancy in most countries and a greater number of people are living to older ages and are at greater risk to Chronic diseases of various kinds.
2. The Rapidly changing life styles and behavioural patterns of the people (ex : Smoking, Alcoholism etc.) Combined with predicted decrease in the morbidity and mortality from Infectious Diseases, the future burden of Non - Communicable Diseases is likely to be a major emerging Health Challenge for Developing Countries.

Cardio Vascular Diseases (CVD) is one of the major NCDs comprise of a group of Diseases of the Heart and the vascular system. The major conditions are Ischaemic Heart Disease (IHD), Hypertension, cerebro vascular disease (Stroke), Rheumatic Heart Disease (RHD) and congenital Heart Disease.

In India, an estimated 2.27 million people died due to CVD during 1990 and according to projections the number of deaths due to IHD was to increase from 1.17 million in 1990 to 1.59 million in 2000 and 2.03 million by 2010.

There were over 5 million persons suffering from CVD during 1999. A peculiar cause of concern is the relative early Age of CVD Deaths in India. India is at the mid - point of the emerging Epidemic and will face its full impact in the coming years. India can be benefited from the Strategy of primary prevention.

The prevalence of CVD is reported to be 2-3 times higher in the urban population as compared to the Rural population. In one study, the prevalence of IHD among adults (Based on Clinical and ECG Criteria) was estimated at 96.7 per 1000 population in the urban and 27.1 per 1000 in Rural Areas¹. The Incidence of CVD is also greater in urban Areas than in Rural Areas reflecting the acquisition of several risk factors, such as Tobacco consumption, lack of physical activity, unhealthy diet and obesity.

The present morbidity and mortality rates are the consequence of previous exposure to behavioural risk factors such as inappropriate Nutrition, Insufficient physical activity and increased Tobacco consumption. It is called the "lag-time" effect of risk factors for CVD. Over weight, central obesity, High Blood pressure, Dyslipidaemia, Diabetes and low cardio - Respiratory fitness are among the Biological factors contributing principally to increased risk.

It is now well established fact that a persistently high cholesterol level can almost certainly precipitate a cardiac event such as IHD. Still most people

do not have an idea of Nutritional requirements and a balanced diet. Unhealthy dietary practices include a high consumption of saturated fats, salts, and refined carbohydrates, as well as low consumption of vegetables and fruits and these tend to cluster together.

Coronary Heart Disease (CHD, Syn : Ischaemic Heart Disease) has been defined as impairment of Heart function due to inadequate blood flow to the heart compared to its needs caused by obstructive changes in the coronary circulation to the Heart². The WHO has drawn attention to the fact that CHD is our Modern "Epidemic", i.e., a disease that affects populations, not an unavoidable attribute of Ageing. CHD may manifest itself in many presentations.

- A. Angina Pectoris of Effort
- B. Myocardial Infarction
- C. Irregularities of the Heart
- D. Cardiac Failure
- E. Sudden Death

ROSE calculates that the "Incubation period" of CHD may be 10 years or more³. The pattern of CHD in India has been reported to be as follows :

- A. CHD appears a decade earlier compared with the Age Incidence in developed countries. The peak period is attained between 51 - 60 years.

- B. Males are affected more than Females.
- C. Hypertension and Diabetes account for about 40 percent of all cases.
- D. Heavy Smoking is responsible Aetiologically in a good number of cases⁴.

RISK FACTORS

The Aetiology of CVD is multifactorial. Studies have identified several important "Risk" factors (i.e., Factors that make the occurrence of the disease more probable). Some of the Risk factors are modifiable. Others are immutable. Presence of any one of the Risk factors places an individual in a High - risk category for developing CVD. The greater the number of Risk factors present, the more likely, one is to develop CVD.

RISK FACTORS FOR CVD

Not Modifiable	Modifiable
Age	High Blood Pressure
Sex	Obesity
Family History	Cigarette Smoking
Genetic Factors	Sedentary Habits
Personality	Dietary Habits
	Alcohol
	Stress

Since 1951, one of the best known large prospective studies, the **Framingham study**, has played a major role in establishing the nature of CVD Risk factors and their relative importance^{5,6}.

Various Epidemiological studies have confirmed the role of these Risk factors and reduction of morbidity and mortality due to CVD by controlling them. The benefit is more, if control is achieved in younger Age itself.

Control of Risk factors may be primary or Secondary. **Primary Prevention** implies control of Risk factors before the onset of CVD. **Secondary Prevention** denotes control of Risk factors after the clinical setting of CVD. Primary prevention is very important because 25% of patients may die even before they reach the doctor with the onset of CVD.

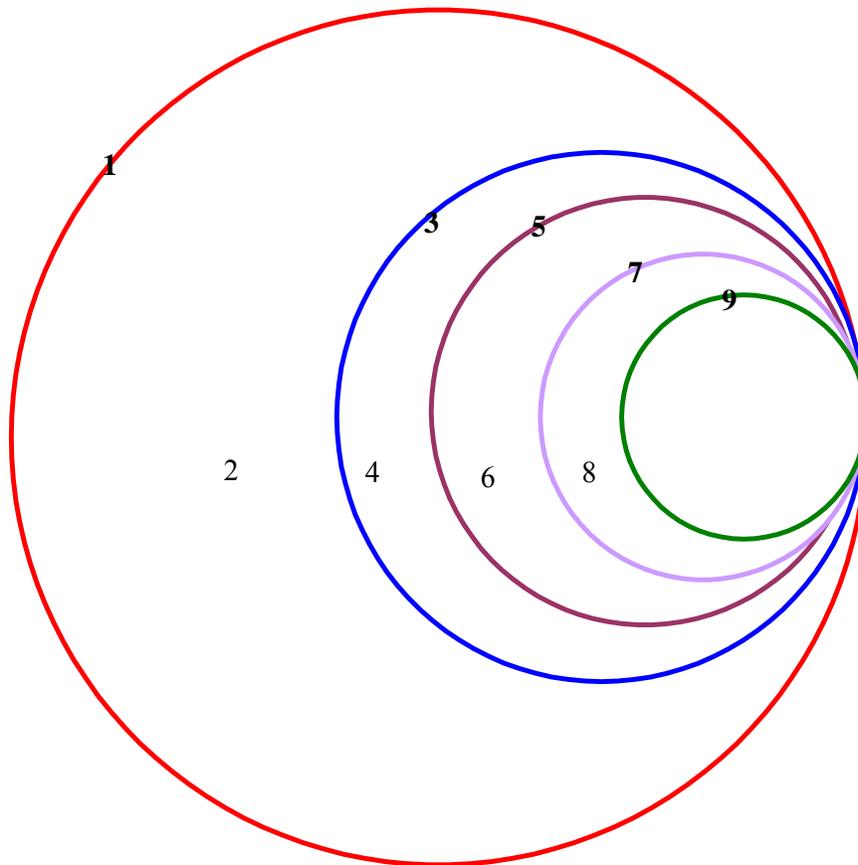
OBJECTIVES (AIM)

1. To estimate the prevalence of selected modifiable risk factors of cardiovascular Diseases (Hypertension, Obesity, Smoking, Physical Inactivity, Diet, Alcohol) in Adults aged 18 years and above at Soolamangalam Village, Thanjavur District.
2. To find out the Association of Risk factors among the various demographic variables.
3. To identify the adults with High Risk factors of CVD and provide necessary health care.

JUSTIFICATION

1. A large body of Data exists on the occurrence of CVD in Hospital patients. However there are only few studies on its prevalence in the general population. On Screening of persons over the age of 30 years by a 12 - lead ECG, in Chandigarh (Urban Population) the prevalence was found to be 65.4 and 47.8 per 1000 Males and Females respectively⁷. In a village in Haryana the prevalence was 22.8 and 17.3 per 1000 males and females respectively⁸.
2. The prevalence data in India are derived from two well - planned studies which screened all persons aged 20 - 60 years and followed WHO suggested criteria for diagnosis⁹ of Hypertension. The one in Rohtak is taken to represent the urban population¹⁰ and the other in a village in Haryana to represent Rural population in India¹¹. The prevalence of Hypertension was 59.9 and 69.9 per 1000 in males and females respectively in the urban population, and 35.5 and 35.9 per 1000 in males and females respectively in the Rural population.
3. The Risk factor Hypertension is an "Iceberg" disease. It became evident in the early 1970s, that only about half of the Hypertensive subjects in the general population of most developed countries were aware of the condition, only about half of those aware of the problem were being treated and only about half of those treated were considered adequately treated. If this was the situation in countries with highly developed medical services, in the developing countries like India, the proportion treated would be far too less (Rule of Halves).

HYPERTENSION IN THE COMMUNITY



AREAS OF THE CIRCLES = ACTUAL PROPORTIONS OBSERVED

- | | | | |
|----|------------------------|----|--------------------------|
| 1. | The whole community | 2. | Normotensive subjects |
| 3. | Hypertensive subjects | 4. | Undiagnosed hypertension |
| 5. | Diagnosed hypertension | 6. | Diagnosed but untreated |
| 7. | Diagnosed and treated | 8. | Inadequately treated |
| 9. | Adequately treated | | |

4. It has been estimated that obesity affect 20 - 40% of the Adults and 10 - 20% of children and Adolescents in developed countries.
5. Most of the Studies on Risk factors of CVD done only in developed countries or in Northern India, that too few decades back.
6. Scanty Information is available from South India regarding the prevalence of Risk factors of CVD. Most of the available information is either urban based or Hospital based.
7. Most of the Risk factors of CVD are easily modifiable on simple IEC activities (Information, Education and Communication).
8. Results of this study can be used for prevention and control of NCDs (CVDs) at Rural Community level.

In view of the above Reasons, A cross Sectional, Descriptive Study of Modifiable Risk factors of cardio vascular diseases in adults at Soolamangalam Village, Thanjavur is conducted to estimate the prevalence of risk factors in a Rural area of Central Tamil nadu.

REVIEW OF LITERATURE

A. HYPERTENSION

Hypertension is a chronic condition of concern due to its role in the causation of coronary Heart Disease, Stroke and other vascular complications. It is the commonest cardiovascular disorder, posing a major public health challenge to population in socioeconomic and epidemiological transition. It is one of the major risk factors for cardiovascular mortality which accounts for 20 - 50 percent of all deaths.

Definition of Hypertension is difficult and, by necessity arbitrary. **Sir George Peckering** first formulated a concept that Blood pressure in a population is distributed continuously as a Bell - shaped curve with no real separation between normotension and Hypertension¹³. There is also a direct relation between cardiovascular risk and blood pressure. The higher the blood pressure, the higher the risk of both stroke and coronary events. As a consequence, the dividing line between Normal and High blood pressure can be defined only in an operational way.

As Intervention trials included only adults aged 18 years or older, definition and classification of Hypertension refer to Adults not taking Anti - Hypertensive drugs and not actually ill, and based on the average of two or more readings on two or more occasions after initial screening.

CLASSIFICATION OF BLOOD PRESSURE MEASUREMENTS

Category	Systolic blood pressure (mm of Hg)	Diastolic blood pressure (mm of Hg)
Normal	< 130	< 85
High normal	130-139	85-90
Hypertension		
Stage 1 (mild)	140-159	90-99
Stage 2 (moderate)	160-179	100-109
Stage 3 (severe)	> 180	>110

When systolic and diastolic blood pressure fall into different categories, the higher category should be selected to classify the individual's blood pressure. "Isolated Systolic Hypertension" is defined as a systolic blood pressure of 140 mm of Hg or more and a diastolic blood pressure of less than 90 mm Hg.

Accurate Blood Pressure measurements are essential under Standardized conditions for valid comparison between persons or groups over time. Three sources of errors have been identified in the recording of blood pressure :

- A. **Observer Errors** : eg : Hearing Acuity, Interpretation of Korotkow sounds.

- B. Instrumental Errors :** eg : leaking valve, cuffs that do not encircle the arm. If the cuff is too small and fails to encircle the arm properly then too high a reading will be obtained.
- C. Subject Errors :** eg : the circumstances of examination. These include the physical environment, the position of the subject, external stimuli such as fear, anxiety, and so on¹⁴.

A few salient points need be mentioned about measuring blood pressure. A WHO study group¹⁵ recommended the sitting position than the supine position for recording blood pressure. In any study a uniform policy should be adopted, using either the Right or left arm consistently. The pressure at which the sounds are first heard (Phase I) is taken to indicate the systolic pressure. Near the diastolic pressure the sounds first become muffled (Phase IV) and then disappear (Phase V). Most of the studies have used phase V to measure diastolic blood pressure. The systolic and diastolic pressures should be measured at least three times over a period of at least 3 minutes and the lowest reading recorded. For reasons of comparability, the data should be recorded every where in a uniform way.

The blood pressure is the single most useful test for identifying individuals at a high risk of developing CHD. Hypertension accelerates the Atherosclerotic process, especially if Hyperlipidaemia is also present and contributes importantly to CHD. In the past, emphasis was placed on the

importance of Diastolic blood pressure. Many investigators feel that Systolic blood pressure is a better predictor of CHD than is the diastolic. However, both components are significant Risk factors. The risk role of "mild" hypertension is generally accepted¹⁶.

Hypertension is divided into **Primary** (Essential) and **Secondary**. Hypertension is classified as "Essential" when the causes are generally unknown. **Primary (Essential)** Hypertension is the most prevalent form of Hypertension accounting for 90% of all cases of Hypertension. Hypertension is classified as "**Secondary**" when some other disease process or abnormality is involved in its causation. Prominent among these are diseases of kidney (chronic glomerulo - nephritis and chronic pyelonephritis), tumours of the Adrenal glands, congenital narrowing of the Aorta and Toxemias of pregnancy. Altogether, these are estimated to account for about 10% or less of the cases of Hypertension.

High Blood pressure is a major risk factor for Stroke, CHD, Heart or kidney failure. The higher the pressure, The greater the risk and lower the expectation of life. Mortality rates from Hypertension are also misleading, as Hypertension is a grossly underreported factor in cardiovascular mortality. The bulk of mortality associated with Hypertension is due to cardiovascular disease.

B. OBESITY

Obesity may be defined as an abnormal growth of the Adipose tissue due to an enlargement of fat cell size (Hypertrophic obesity) or an increase in fat cell number (Hyperplastic obesity) or a combination of both¹⁷. Obesity is often expressed in terms of Body Mass Index (BMI). Overweight is usually due to obesity but can arise from other causes such as Abnormal muscle development or fluid retention¹⁸.

However, obese individuals differ not only in the amount of excess fat that they store, but also in the Regional distribution of the fat within the body. The distribution of fat induced by the weight gain affects the risk associated with obesity, and the kind of Disease that results. It is useful therefore, to be able to distinguish between those at Increased Risk as a result of "Abdominal fat distribution" or Android obesity from those with the less serious "Gynoid" fat distribution, in which fat is more evenly and peripherally distributed around the body.

Eating Habits (e.g : eating in between meals, preference to Sweets, Refined foods and fats) are established very early in life. The composition of the Diet, the periodicity with which it is eaten and the amount of Energy derived from it are all relevant to the aetiology of obesity. The basic cause of obesity is over Nutrition and Hyper - Energy food intake. A diet containing more energy than needed may lead to prolonged post - prandial Hyperlipidaemia and to deposition of Triglycerides in the Adipose tissue resulting in obesity. The accumulation of one kilo of fat corresponds to 7,700 k.cal of energy.

The body is composed of as under :

- a. The active mass (muscle, liver, heart etc.)
- b. The fatty mass (fat)
- c. The extracellular fluid (blood, lymph etc)
- d. The connective tissue (Skin, bones, connective tissue)

Structurally speaking, the state of obesity is characterised by an increase in the fatty mass at the expense of the other parts of the body. The water content of the body is never increased in case of obesity.

Body Mass Index, (BMI, Quetelet's Index) is a simple index of weight for Height that is commonly used to classify underweight, overweight and obesity in Adults. It is defined as the weight in kilograms divided by the square of the height in metres (kg/m^2). For example, an Adult who weighs 70 kg and whose Height is 1.75 m will have a BMI of 22.9.

$$\text{BMI} = 70(\text{kg}) / 1.75^2 (\text{m}^2) = 22.9$$

CLASSIFICATION OF ADULTS ACCORDING TO BMI

Classification	BMI	Risk of Co morbidities
Underweight	<18.50	Low (but risk of other clinical problems increased)
Normal range	18.50 - 24.99	Average
Overweight	≥ 25.00	
Pre obese	25.00 - 29.99	Increased
Obese class I	30.00 - 34.99	Moderate
Obese class II	35.00 - 39.99	Severe
Obese class III	≥ 40.00	Very severe

Obesity is classified as a BMI \geq 30.0. The classification shown is in agreement with that recommended by WHO¹⁸, but includes an additional subdivision at BMI 35.0 - 39.9 in recognition of the fact that management options for dealing with obesity differ above a BMI of 35. The WHO classification is based primarily on the association between BMI and mortality.

These BMI values are Age - Independent and the same for both sexes. The table shows a simplistic relationship between BMI and the risk of comorbidity, which can be affected by a range of factors, including the nature of the diet, Ethnic group and Activity level. The risks associated with increasing BMI are continuous and graded and begin at a BMI above 25.

C. SMOKING

Some people commit suicide by Drowning but many by smoking. A uniquely human habit, smoking has been identified as a major CVD risk factor^{20,21} with several possible mechanisms - carbon monoxide induced atherogenesis, Nicotine stimulation of Adrenergic drive raising both blood pressure and myocardial oxygen demand; Lipid metabolism with fall in "protective" high - density lipoproteins, etc.

Nicotine leads to damage of the intimal layer and endothelial wall lining of the coronary vessels. The vessel damage results in platelet adhesion clot formation, haemorrhage and the formation of Atherosclerotic plaque. The

cadmium in cigarette smoke is believed to be related to elevations in blood pressure.

It has been calculated that in countries where smoking has been a widespread habit, it is responsible for 25 percent of CHD deaths under 65 years of age in men. Cigarettes seem to be particularly important in causing sudden death from CHD especially in men under 50 years of Age²².

The degree of Risk of Developing CHD is directly related to the number of Cigarettes smoked per day²³ Filter cigarettes are probably not protective²⁴. There is evidence that the influence of smoking is not only independent of, but also synergistic with other risk factors such as Hypertension and elevated serum cholesterol. This means that the effects are more than additive.

The risk of death from CHD decreases on cessation of smoking. The Risk declines quite substantially within one year of stopping smoking and more gradually thereafter until, after 10 - 20 years, it is the same as that of Non - Smokers. For those who have had a Myocardial infarction, the risk of a fatal recurrence may be reduced by 50 percent after giving up smoking.

In most populations, smokers weigh some what less than ex smokers. Individuals who have never smoked fall somewhat between the two.

D. PHYSICAL INACTIVITY

Sedentary life - style is associated with a greater risk of the development of early CHD. There is evidence that Regular Physical exercise increases the concentration of HDL²⁵ and decreases both body weight and blood pressure which are beneficial to cardio vascular health. Physical activity by reducing body weight may have an indirect effect on blood pressure.

There is convincing evidence that regular physical activity is protective against unhealthy weight gain. Where as sedentary life style particularly sedentary occupation and inactive recreation such as watching television promote it. Physical activity and physical fitness are important modifiers of mortality and morbidity related to overweight and obesity²⁶. In some individuals a major reduction in activity without the compensatory decrease in habitual energy intake may be the major cause of increased obesity ex : in Athletes when they retire and in young people who sustain injuries etc. Physical inactivity may cause obesity which in turn restricts activity. This is a vicious circle. It is the reduced energy output that is probably more important in the aetiology of obesity than used to be thought²⁷. Increased physical activity is an important part of weight reducing programme. Regular physical exercise is the key to an increased energy expenditure.

The term physical activity refers to the regular participation in either structured or unstructured activity at least 3 to 5 times per week for at least 30

minutes. The terms physical activity and exercise are used interchangeably. In the context of the present study physical inactivity was conceptually defined as the lack of participation in regular physical activities at least once per week. Excessive physical activity refers to strenuous activity that an individual engages in for extended periods everyday during the week which results in deleterious physical and psychological effects.

E. DIET

The risk of CHD appears to increase as the plasma cholesterol concentration rises²⁸. There is a well established Triangular relationship between habitual diet, blood cholesterol levels and CHD. However, the optimal level of plasma total cholesterol remains a debated issue. Leading investigators have recommended that the upper most acceptable level should be 240 mg/dl. The optimum level has been suggested to be less than 200 mg/dl²⁹.

i. CHOLESTEROL

Cholesterol occurs in all foods of animal origin. Part of it is synthesized in the body. The plasma cholesterol is determined by :

- a. The amount absorbed from food
- b. The amount synthesized in the body
- c. The rate of catabolism and excretion in the bile
- d. Intestinal reabsorption of bile acids, and
- e. The equilibrium between plasma and tissues.

The extent to which cholesterol intake influences total cholesterol levels is highly variable.

ii. LIPOPROTEINS

Cholesterol is carried in plasma lipoproteins. Lipoproteins are divided into four major classes - chylomicrons, very low density lipoproteins (VLDLs), low density lipoproteins (LDLs) and high density lipoproteins (HDLs). The total serum cholesterol is the sum of the cholesterol in 3 lipoprotein fractions, viz., VLDL, LDL and HDL Cholesterol. Most of the serum cholesterol (close to 50%) is in LDL. Whereas LDL is dominated by its cholesterol content, VLDL is dominated by its triglyceride content.

LDL has been shown to function in the delivery of cholesterol to body cells. Excessive level of LDL thus leads to the accumulation of cholesterol in tissue cells particularly the smooth muscle cells of the vascular system. It is thus involved in the arteriosclerotic process. In contrast, HDL functions in the removal of cholesterol from cells. This mechanism underlines its protective effect in CHD. From long - term observations it became quite clear that HDL levels, the higher they are, the more protective they seem to be against CHD.

iii. FATTY ACIDS

High intake of saturated fatty acids over several weeks or months causes an increase in plasma cholesterol. Dietary unsaturated fatty acids with two or more double bonds have been shown to lower plasma cholesterol.

Polyunsaturated fatty acids (ex : Linoleic and Arachidonic acids) have an additional role, that is, to inhibit platelet aggregation and thus prevent Thrombus formation. Recent Research indicates that Arachidonic acid metabolises in the vascular endothelium to form two important metabolites, namely prostacyclin and Thromboxane. These two compounds have opposing effects on the cardiovascular system. Whereas thromboxane induces platelet aggregation, prostacyclin inhibits the same and prevents intravascular thrombus formation. Prostacyclin was found also to relax coronary blood vessels, thus opposing the action of Thromboxane. It has been suggested that generation of prostacyclin is the biochemical mechanism underlying the well-known ability of blood vessels to resist platelet aggregation. Linoleic acid which is the main precursor of Arachidonic Acid is therefore regarded as the body's best bulkwark against CHD.

It has been shown that cholesterol in blood can be reduced by controlling the amount and type of fat in the diet. Evidence is emerging that ischaemic myocardium may not metabolise all fatty acids equally, and that the accumulation of some of these in the myocardium may be more closely associated with sudden cardiac death³⁰.

iv. TRIGLYCERIDES

In a very large proportion of individuals with a raised cholesterol level, the blood concentration of triglycerides (TG) is also markedly increased.

Someworkers have indeed claimed that correlation between TG and CHD is as good as that between total serum cholesterol and CHD. Recently, it has been shown that TG levels act as a significant independent risk factor for CHD. Both cholesterol and TG are associated with specific proteins in the plasma to form lipoproteins.

The most important determinant of TG level is the activity of the enzyme, lipoprotein lipase in the endothelial lining of the capillaries and in a variety of tissues. This enzyme removes TG particularly from the very low density lipoproteins (VLDL) and converts these to the lipoproteins of higher density.

v. CARBOHYDRATES

Coronary heart disease rates are lowest in populations eating high carbohydrate diets. Neither high carbohydrate nor High sucrose feeding has induced Atherosclerosis in Animals. Several studies indicate that the risk of CHD and hypertension is inversely related to the consumption of dietary fibre. Most fibres reduce plasma Total and LDL cholesterol³¹.

vi. SALT

There is an increasing body of Evidence to the effect that a High Salt intake (i.e., 7 - 8 grams / day) increases blood pressure proportionately. Low sodium intake has been found to lower the blood pressure.

F. ALCOHOL

High Alcohol intake, defined as 75 grams or more per day is an independent risk factor for CHD, Hypertension and all cardio vascular diseases³². The evidence that moderate alcohol intake leads to a reduction in the risk of CHD is unsubstantiated³³. It appears that alcohol consumption raises systolic pressure more than the diastolic. But the finding that blood pressure returns to normal with abstinence suggests that Alcohol induced elevations may not be fixed, and do not necessarily lead to sustained blood pressure elevation³⁴. The relationship between alcohol consumption and adiposity was generally positive for men and negative for women³⁵.

MATERIALS AND METHODS

BACKGROUND INFORMATION

The village Soolamangalam, situated in Papanasam Taluk and Block in Thanjavur District of Central Tamilnadu. This has been selected for this community based study on modifiable risk factors of cardio vascular diseases in adults. This rural village is situated about 25 kms, away from Thanjavur Town. A sub centre is situated at Soolamangalam Village attached with Pandaravadai. Additional PHC which again comes under the main PHC Kabisthalam in Thanjavur Health Unit District. One Village Health Nurse and one Health Inspector are posted in this sub centre. The total population of Soolamangalam is 1974 with a Households of 428.

STUDY DESIGN

This study was done as a cross sectional descriptive study on the modifiable risk factors of cardio vascular diseases in adults at Soolamangalam Village, Thanjavur.

STUDY AREA AND POPULATION

Adults population aged 18 years and above of both sexes, normally living in Soolamangalam a Rural Village in Thanjavur District was considered eligible to be included in the study.

STUDY PERIOD

The study was conducted during the period between January 2006 and April 2006.

Sample Size

Formula

$$\eta = Z\alpha^2 \frac{PQ}{d^2}$$

where $Z\alpha^2 = 1.96$ for 95% confidence intervals.

Different sample sizes can be calculated based upon different prevalence level of different risk factors. Here the sample size was arrived with the prevalence of major CVD Risk factor hypertension 20%, as reported in a study conducted by ICMR and WHO in 2003 in India. (Surveillance of Risk factors for non communicable diseases. The WHO STEP wise approach)^{36,37}.

$$p = 20\%$$

$$q = 80\%$$

$$d = \text{Allowable error}$$

$$n = \text{number of samples is to be studied}$$

Allowed Error (limit of accuracy) = 20% of prevalence is allowed to calculate the value of 'd'.

$$d = 20\% \text{ of } 20\% = 4\%$$

So

$$\eta = Z\alpha^2 \frac{PQ}{d^2}$$

$$= (1.96)^2 \times \frac{20 \times 80}{4 \times 4} = 384$$

$$\text{Sample Size} = \mathbf{384}$$

STUDY POPULATION

The 2006 Assembly Election Voter's list of about 1974 population of Soolamangalam Village, Thanjavur was taken up. All the voters above 18 years old are picked up and serially numbered and used as sampling frame. It came around 1100 voters. By using simple random sampling technique (SRS), about 500 individuals were picked up, to attain sample size.

STUDY METHOD

The sampled 500 individuals, name and address were prepared from the sampling frame. They were interviewed on visiting their houses by using Ready made Structured Questionnaires prepared both in Tamil and English. In case of Absence or Refusal, the next individual is interviewed till 400 individuals are covered. Blood Pressure, Height, Weight, Measurements were carried out. All the particulars were recorded then and there in the proforma.

SELECTION CRITERIA

Inclusion Criteria :

- A. Men and Women aged above 18 years.
- B. Adults who can understand Tamil and reside at Soolamangalam Village, Thanjavur.
- C. Adults who are present at the time of study.
- D. Adults who are willing to participate in the study.

Exclusion Criteria :

- a. Adults who were previously diagnosed as Hypertensive and on treatment or not.
- b. Adults who are ill and are on treatment for other Ailments.
- c. Antenatal and postnatal mothers.
- d. Visitors or Guests who are not normally Residing in Soolamangalam Area.

DEVELOPMENT OF QUESTIONNAIRE

Tool selected for this study was the Structured Interview Schedule. After an extensive Review of Literature, Discussion with Experts and the Investigator's personal experience, a Structured Interview Schedule consisting of objective type questions was developed for the collection of Data. This comprehensive structured questionnaire was modified again by **pilot testing** and on expert advice. It was found that the tools were reliable and feasible.

The questionnaire was initially prepared in English and then it was translated in to Tamil so that the Questions were in the local language and easily understood by the Respondents. The translated questionnaire was retranslated in to English to Check if the translated Questionnaire conveyed the same meaning. The Questionnaire was then **pre-tested** in Sarabojirajapuram an area that was not included in the sampling frame.

Maximum care was taken in preparing the questionnaire to avoid wrong interpretation.

The pre - tested Structural Questionnaire consists of 2 parts. The first part sought information on the Socio - demographic aspects like Sex, Age, Education, Occupation, Income, Marital Status, Type of Family, Religion and History of CVD or Risk factors in the family.

The Second part includes information on, base line data and the life style:

1. Blood Pressure
2. Obesity
3. Smoking
4. Physical activity
5. Diet
6. Alcohol

DATA COLLECTION

Formal permission was obtained from the Director I/C. Institute of Community Medicine, Madras Medical College, Chennai - 3, and submitted to the Panchayat President of Soolamangalam Village.

The investigator visited the selected homes and introduced himself, explained the purpose of the interview, and obtained verbal consents.

Reassurance was given regarding the confidentiality of their responses. The structured interview schedule was introduced after inviting the adult to sit comfortably in a convenient place in the House. The Height and weight were also checked on the same day. During the course of Interview, the investigator obtained two measurements of blood pressure on each study participant with mercury column sphygmomanometer using a standardized technique. In case of Absence of Study Subject or Refusal, the next participant is interviewed till 400 individuals are studied.

A. MEASUREMENT OF BLOOD PRESSURE

Accurate measurement of blood pressure requires reliable, well maintained equipment, properly trained observer and knowledge of factors known to affect the blood pressure. Two readings were made a five minutes interval as per the WHO recommendation. When high blood pressure greater than or equal to 140/90 mm Hg was noted a third reading was made after thirty minutes. Lowest of the three was recorded.

The patient was allowed to sit quietly with the back supported for five minutes and arm supported at the level of the heart. It is seen that the following conditions are fulfilled before taking the measurement.

1. No caffeine during the hour preceding the reading.
2. No smoking during the fifteen minutes preceding the reading.
3. No exertion of any kind preceding the measurements.

EQUIPMENT

The gold standard for routine blood pressure measurement is a standard mercury column sphygmomanometer with cuff size 12 x 13 cms x 35 cms. The bladder should encircle and cover 2/3 rd of the length of the arm. The lower edge of the cuff should be about 2.5 cms above the anterior cubital fossa. The blood pressure was recorded with the help of the same sphygmomano meter and by the same observer. Manometer should be placed on horizontal surface at the level of the Heart. Every time before taking the reading 'O' reading was ensured.

Blood Pressure measures were obtained on the Right Arm. The cuff pressure was inflated 30 mm Hg above the level at which the Radial pulse disappeared, then deflated slowly at the rate of 2 mm / second and the reading recorded to the nearest 2 mm. The first and fifth korotkoff sounds were taken as indicative of the systolic and the diastolic blood pressure respectively.

CLASSIFICATION OF BLOOD PRESSURE MEASUREMENTS

Category	Systolic blood pressure (mm of Hg)	Diastolic blood pressure (mm of Hg)
Normal	< 130	< 85
High normal	130-139	85-90
Hypertension		
Stage 1 (mild)	140-159	90-99
Stage 2 (moderate)	160-179	100-109
Stage 3 (severe)	> 180	>110

B. BODY MASS INDEX

Body weight was recorded with the help of an adult portable weighing machine (Bath Room Scale). The same machine was used throughout the study.

The scale was adjusted to zero reading before weighing. Accuracy of the scale was checked every week with standard weight. Weighing was done with minimum dressing and without foot wears. Height was measured with steel centimeters tape. The body mass index was calculated using quetelet's index.

$$\text{B.M.I} = \frac{\text{Weight in Kg}}{\text{Height in m}^2}$$

CLASSIFICATION OF ADULTS ACCORDING TO BMI

Classification	BMI	Risk of co morbidities
Underweight	<18.50	Low (but risk of other clinical problems increased)
Normal range	18.50 - 24.99	Average
Overweight	≥ 25.00	
Pre obese	25.00 - 29.99	Increased
Obese class I	30.00 - 34.99	Moderate
Obese class II	35.00 - 39.99	Severe
Obese class III	≥ 40.00	Very severe

- C. Smoking :** Section C contains 3 questions with maximum score of 15 marks.
- D. Physical Activity :** Section D contains 5 questions and each question has 5 options, with a total score of 25 marks.
- E. Diet :** This Section E consists of 9 questions each question has 5 options and highest risk carries 5 marks and the lowest risk carries 1 mark. These 9 questions carry a total possible score of 45 marks.
- F. Alcohol :** This Section F has 3 questions with a maximum score of 15 marks.

SCORING

Each question has 5 options. First option carries 5 marks. Second option 4 marks. Third option 3 marks. Fourth option 2 marks and fifth option carries 1 mark.

The Classification of Scoring is as follows :

Level of Risk	Range (Score)
High Risk	> 75%
Moderate Risk	51 - 74%
Low Risk	< 50%

CONTENT VALIDITY

Various Experts from the fields of community medicine and cardiology validated the content validity of the Tool. The validation was performed also by Senior Physicians, Nutritionist and Statistician.

RELIABILITY OF THE TOOL

A Reliability test was done on the tool by Split-Half method which revealed a result of $r^1 = 0.8$.

After the interview was completed, the participant was thanked for his / her co-operation.

PLAN FOR DATA ANALYSIS

Both Descriptive and Inferential Statistics were used. Percentage distribution utilized bars and graphs; standard deviation was used to assess the prevalence of selected modifiable risk factors of CVD; and inferential statistics "Chi" square and ANOVA was used to study the strength of the association of data with demographic variables and to identify high risk for CVD among adults.

DEFINITIONS

Adult : For this study an adult person was defined as one aged 18 years and above.

Hypertension : Defined as either systolic blood pressure greater than or equal to 140 mm. Hg and / or diastolic blood pressure greater than or equal to 90 mm Hg and / or treatment with Anti Hypertensive medication. This definition excludes Hypertensives who have their blood pressure to a normotensive range by Non - pharmacological means.

Treatment of Hypertension : Defined as current use of a prescriptive medication for lowering elevated blood pressure among Hypertensive subject in our sample. The only treatment considered was pharmacological. In order to avoid misclassification of Normotensive individual on cardiovascular medication for indication other than Hypertension (such as for congestive Heart

failure or Angina) a diagnosis of treated Hypertensive was made only if the subject had a prior diagnosis of Hypertension and was on Anti - Hypertensive agents.

Control of Hypertension : Defined as pharmacological treatment associated with SBP and DBP less than 140 and 90 mm of Hg respectively.

OBSERVATIONS AND RESULTS

ORGANIZATION OF DATA

SECTION - A

Demographic variables of adults at Soolamangalam Village.

SECTION - B

Assessment of physiological baseline data.

SECTION - C

Assessment of the level of modifiable risk factors.

SECTION - D

Association between level of risk factors and demographic variables.

SECTION - E

Association between level of selected physiological baseline data and selected demographic variables.

SECTION A: Demographic variables of Adults at Soolamangalam Village.

TABLE - 1: FREQUENCY AND PERCENTAGE DISTRIBUTION OF DEMOGRAPHIC VARIABLES

n = 400

Demographic Variables		Frequency	Percentage %
Sex :	Male	180	45
	Female	220	55
Age in years :	18 - 30	68	17
	31 - 36	72	18
	37 - 42	88	22
	43 - 48	84	21
	49 - 54	32	8
	55 - 60	56	14
Education :	Illiterate	88	22
	Primary	60	15
	Middle	76	19
	High School	136	34
	Hr. Sec.	24	6
	Graduate	16	4
Marital Status :	Unmarried	8	2
	Married	372	93
	Widow / Widower	20	5
Type of Family :	Joint	216	54
	Nuclear	184	46
Religion :	Hindu	280	70
	Christian	120	30
Occupation :	Unskilled Labour	148	37
	Skilled Labour	56	14
	Office Worker	32	8
	Business	56	14
	Retired	8	2
	No Occupation	100	25
Income :	< Rs.500	100	25
	Rs.501-Rs.1000	88	22
	Rs.1001-Rs.1500	88	22
	Rs.1501-Rs.2000	44	11
	>Rs.2000	80	20
History of CVD :	No	244	61
	Paternal	28	7
	Maternal	36	9
	Both	8	2
	Don't know	84	21

Table 1 shows the analysis of demographic variables among adults in Soolamangalam Village. The data reveals that 55% of the sample were females and 45% were males (Fig.1). 22% of the population were between the age group of 37 years, to 42 years and only 8% were between 49 years to 54 years (Fig.2).

Analysis of educational status shows that 34% had high school education, 22% of the population were found to be illiterate, 19% had middle school education, 15% had primary education, 6% had higher secondary education and 4% had graduation. The majority of the study population 93% were married. Regarding religion, 70% were Hindus and 30% were Christians. The analysis of the occupational status shows that 37% were unskilled labourers and 2% were retired. 25% with income less than Rs.500 and 11% having income between Rs.1501 - Rs.2000 (Fig.3).

Analyzing the history of CVD in the family, 61% had no family history and only 2% had a history of both parents having CVD.

TABLE - 2
CLASSIFICATION OF BLOOD PRESSURE LEVELS AMONG
ADULTS

n = 400

Sl. No.	Category	Systolic B.P (mmHg)	Frequency	Percentage %	Diastolic B.P (mmHg)	Frequency	Percentage %
1.	Optimal B.P.	< 120	320	80	< 80	340	85
2.	Normal B.P.	< 130	44	11	<85	24	6
3.	High-normal B.P.	130-139	8	2	85-89	4	1
4.	Grade I hypertension (mild)	140-159	24	6	90-99	32	8
5.	Grade 2 hypertension (moderate)	160-179	4	1	100-109	-	-

Table 2 depicts the classification of blood pressure levels. 80% of the population fell in optimal systolic (<120 mmHg) blood pressure and 85% in optimal diastolic (< 80mmHg) blood pressure, 6% of the population fell in Grade I systolic (140 - 159 mmHg) hypertension and 8% fell in Grade I diastolic (90-99 mmHg) hypertension. Only 1% of population fell in Grade 2 systolic (160 - 179 mmHg) hypertension (Fig.4).

TABLE - 3
FREQUENCY AND PERCENTAGE DISTRIBUTION OF BODY MASS
INDEX

n = 400

Sl.No.	Classification of BMI in kg/m²	Frequency	Percentage %
1.	Under weight < 18.50	136	34
2.	Normal weight 18.51 - 24.99	232	58
3.	Over weight \geq 25.00	28	7
4.	Pre obese 25.01 - 29.99	4	1

Table 3 depicts the classification of adults according to body mass index. 58% of the population were in normal range, 7% were overweight and only 1% fell in the pre obese category (Fig.5).

SECTION B : Assessment of physiological baseline data for adults.

TABLE - 4
MEAN, STANDARD DEVIATION AND RANGE FOR
PHYSIOLOGICAL BASE LINE DATA

n = 400

Physiological Data	Mean		Standard deviation		Range			
	Male	Female	Male	Female	Minimum		Maximum	
					Male	Female	Male	Female
Height in cms	167.04	154.93	7.0	6.4	155	142	181	170
Weight in kgs	57.95	47.20	9.19	11.14	37	27	77	78
BMI (kg/m²)	20.10		3.56		12.50		33.77	
Blood Pressure								
Systolic mmHg	118.99		10.78		90		163	
Diastolic mmHg	77.30		7.67		50		96	

Table 4 reveals overall mean and standard deviations of physiological base line data. The mean score of 167.04 for males and 154.93 for females was found for height with a standard deviation of 7.0 and 6.4 respectively. Weight measurements revealed 57.95 as a mean score for males, and 47.20 for females with standard deviations of 9.19 for males and 11.14 for females.

The mean score of BMI was 20.10 with a standard deviation of 3.56. Systolic blood pressure reading showed a mean score of 118.99 with a standard deviation of 10.78. Diastolic blood pressure results showed a mean score of 77.30 with a standard deviation of 7.67.

SECTION C : Assessment of the level of modifiable risk factors among adults:**TABLE - 5****PERCENTAGE DISTRIBUTION OF MODIFIABLE RISK FACTORS ASSOCIATED WITH SMOKING**

n = 400

Sl.No.	Risk factors	Frequency	Percentage %
1.	Habit of smoking :		
	Daily	56	14
	Two or more times in a week	4	1
	Once in a month	-	-
	Rarely	36	9
	Never do	304	76
2.	Type or form of tobacco use		
	Cigar (Suruttu)	-	-
	Beedi	60	62.5
	Pipe	-	-
	Filter less Cigarette	12	12.5
	Filter Cigarette	24	25
3.	No. of cigarettes used per day		
	More than 20	16	16.67
	16-20	20	20.83
	11-15	24	25
	6-10	-	-
	1-5	36	37.5

Table 5 shows percentage distribution of the modifiable risk factors associated with smoking. 76% of the study population were non-smokers and only 14% population smoke daily, 62.5% of the smokers use Beedi and 25% use filter cigarettes. 16.67% smoke more than 20 cigarettes per day.

TABLE - 6
PERCENTAGE DISTRIBUTION OF MODIFIABLE RISK FACTORS
ASSOCIATED WITH PHYSICAL ACTIVITY

n = 400

Sl.No.	Risk Factors	Frequency	Percentage %
1.	Hours of household activities : Never do Less than 2 hours 2-5 hours 6-9 hours More than 10 hours	100 188 112 - -	25 47 28 - -
2.	Hours of external activities : Never do Less than 2 hours 2-5 hours 6-9 hours More than 10 hours	60 32 104 196 8	15 8 26 49 2
3.	Hours of rest including nap : More than 15 hours 12 - 14 hours 9 - 11 hours 6 -8 hours Less than 5 hours	- 16 108 272 4	- 4 27 68 1
4.	Exercises apart from daily activities : Never do Fast walking Fast cycling Swimming Gymnasium	84 168 128 20 -	21 42 32 5 -
5.	Hours of exercises : Less than 30 minutes 31 mts - 1 hour 1.01 hour - 1.30 hour 1.31 hour - 2 hours More than 2 hours	116 128 56 20 -	29 32 14 5 -

Table 6 shows the percentage distribution of prevalence of the modifiable risk factors associated with physical activity. Considering household activities, 47% perform less than 2 hours daily and 25% are non engaged. 15% of the population never do external activities, while 2% perform more than 10 hours. 42% of the population walks fast as exercise and 21% never exercise apart from their daily activities.

TABLE - 7 : PERCENTAGE DISTRIBUTION OF PREVALENCE OF MODIFIABLE RISK FACTORS ASSOCIATED WITH DIET

n = 400

Sl.No.	Risk Factors	Frequency	Percentage %
1.	Food Pattern :		
	Non - Vegetarian	348	87
	Ova - Vegetarian	24	6
	Lacto - Vegetarian	28	7
2.	Frequency of Non - Veg & Ova - Veg. diet:		
	Daily	4	1
	Two or more days in a week	76	19
	Once in a week	136	34
	Once in a month	132	33
	Rarely	24	6
3.	Type of cooking oil :		
	Coconut oil	20	5
	Groundnut oil	40	10
	Gingily oil	164	41
	Palm oil	144	36
	Refined oil	32	8
4.	Frequency of fried food usage :		
	Daily	4	1
	Two or more days in a week	64	16
	Once in a week	184	46
	Once in a month	104	26
	Rarely	44	11
5.	Frequency of coffee intake :		
	More than two times a day	92	23
	Twice in a day	192	48
	Only once in a day	84	21
	Rarely	20	5
6.	Frequency of pickle intake :		
	Daily	72	18
	Two or more days in a week	52	13
	Weekly once	112	28
	Rarely	140	35
	Never take	24	6
7.	Frequency of papad intake :		
	Daily	-	-
	Two or more days in a week	76	19
	Weekly once	148	37
	Rarely	176	44
	Never take	-	-
8.	Frequency of dried fish intake :		
	Daily	-	-
	Two or more days in a week	64	16
	Weekly once	188	47
	Rarely	80	20
	Never take	68	17
9.	Frequency of snack intake :		
	Daily	16	4
	Two or more days in a week	56	14
	Weekly once	100	25
	Rarely	220	55
	Never take	4	1

Table 7 depicts the percentage distribution of prevalence of modifiable risk factors associated with diet. 87% of the population were non - vegetarians, among them 34% consume once per week and 1% take daily. Among 400 samples, 23% consume coffee more than twice daily and 3% were non - coffee drinkers. 18% of the population consume pickles daily and 6% do not eat pickles. 44% of the population rarely eat papad, 19% consume papad two or more days per week but none of the subjects use papad daily. 47% of the sample eat fried fish once per week; and 16% eat two or more days per week; but none of the subjects eat dried fish daily.

TABLE - 8

**PERCENTAGE DISTRIBUTION OF MODIFIABLE RISK FACTORS
ASSOCIATED WITH ALCOHOL**

n = 400

Sl. No.	Risk factors	Frequency	Percentage %
1.	Alcohol consumption		
	Daily	4	1
	Two or more days in a week	4	1
	Weekly Once	16	4
	Rarely	64	16
	Never take	312	78
2.	Type of alcohol use		
	Arrack (country liquor)	20	22.73
	Gin/Brandy/Rum/Whisky	68	77.27
	Fortified wine	-	-
	Beer	-	-
	Toddy	-	-
3.	Amount of alcohol consumption per drink		
	More than 390ml (13 pegs and above)	-	-
	300-360ml (10-12 pegs)	8	9.09
	210-270ml (7-9 pegs)	20	22.73
	120-180ml (4-6 pegs)	44	50
	30-90ml (1-3 pegs)	16	18.18

Table 8 shows percentage distribution of the modifiable risk factors associated with alcohol consumption. 78% of the population are non-alcoholics and 22% of the population are alcoholics. Among the alcoholics 77.27% consume brandy or whisky. 50% of alcoholics drink 4-6 pegs per drink.

TABLE - 9
MEAN, STANDARD DEVIATION AND RANGE OF SCORES FOR
VARIOUS MODIFIABLE RISK FACTORS

n = 400

Sl. No.	Modifiable Risk factors	Mean	Standard deviation	Range	
				Minimum	Maximum
1.	Smoking	29.93	21.47	20	93.33
2.	Exercise	67.36	8.83	40	85
3.	Diet	57.50	7.43	30	75.56
4.	Alcohol	28.60	16.91	20	80
	Overall	57.16	5.94	42.86	77.78

Table 9 depicts the mean and standard deviation values for the various modifiable risk factors of CVD. The highest mean score of 67.36 was associated with exercise while the lowest 28.60 was related to alcohol consumption. The overall mean for modifiable risk factors among adults was 57.16 and the overall risk factors ranges from 42.86 to 77.78.

TABLE - 10**FREQUENCY DISTRIBUTION OF THE LEVELS OF RISK FOR
VARIOUS MODIFIABLE RISK FACTORS AMONG ADULTS**

n = 400

Sl. No.	Modifiable Risk factors	Low risk <50 %		Moderate risk 51-74%		High risk >75 %	
		No	%	No	%	No	%
1.	Smoking	340	85	24	6	36	9
2.	Exercise	8	2	296	74	96	24
3.	Diet	44	11	344	86	12	3
4.	Alcohol	328	82	68	17	4	1
	Overall	9	9	90	90	1	1

Table 10 elicits the score obtained by the clients for the modifiable risk factors. In view of diet, 11% were under low risk, and 3% were at high risk. Regarding exercise, 2% had low risk and 24% were high risk. It was found that that in smoking 85% were at low risk, and 9% were under high risk. Studying the alcohol intake pattern, 82% were under low risk and 1% were found to fall under high risk category. The overall findings revealed that 9% were found under low risk and only 1% were at high risk (Fig.6).

SECTION D : Association between level of risk factors and demographic variables

TABLE - 11
ASSOCIATION BETWEEN LEVEL OF RISK FACTORS AND
DEMOGRAPHIC VARIABLES

n = 400

Demographic variables	Number	Mean	S.D	F value (P value)
Sex				
Male	180	57.71	6.71	0.69 P = .41 (NS)
Female	220	56.71	5.25	
Age in year				
18-30	68	57.09	4.96	0.25 P = 0.94 (NS)
31-36	72	56.19	4.33	
37-42	88	58.06	6.47	
43-48	84	57.51	6.68	
49-54	32	56.17	5.89	
55-60	56	57.11	7.47	
Education				
Illiterate	88	55.20	5.07	1.30 P = 0.27 (NS)
Primary	60	57.93	4.22	
Middle	76	58.93	4.82	
High School	136	57.81	7.51	
Hr.Sec.	24	54.63	4.99	
Graduate	16	54.94	5.14	
Marital Status				
Unmarried	8	54.75	10.25	0.27 P = 0.76 (NS)
Married	372	57.27	5.93	
Widow	20	56.00	5.94	
Types of family				
Joint Family	216	56.77	6.48	0.51 P = 0.48 (NS)
Nuclear Family	184	57.62	5.28	
Religion				
Hindu	280	57.66	6.34	1.70 P = 0.19 (NS)
Christian	120	55.98	4.77	

Occupation				
Unskilled	148	56.49	4.13	1.61 P = 0.16 (NS)
Skilled	56	57.25	7.45	
Office Workers	32	55.52	4.89	
Business	56	56.45	7.42	
Retired	8	67.25	6.72	
No Occupation	100	58.21	6.32	
Income				
<Rs.500	100	58.21	6.32	1.30 P = 0.27 (NS)
Rs.501-Rs.1000	88	56.04	4.71	
Rs.1001-Rs.1500	88	56.96	3.58	
Rs.1501-Rs.2000	44	54.53	7.23	
>Rs.2000	80	58.74	7.61	
History of CVD				
No	244	57.54	6.03	0.94 P = 0.44 (NS)
Paternal	28	59.71	9.49	
Maternal	36	56.75	3.53	
Both	8	52.67	2.83	
Don't know	84	55.82	5.17	

Table 11 elicits the association between level of risk factors and demographic variables. In regard to sex, males showed a mean score of 57.71, with a standard deviation of 6.71, females were with a mean score of 56.71 and with a standard deviation of 5.25. The variance value $F=0.69$ was not found to be statistically significant. History of paternal CVD had a mean score of 59.71, with a standard deviation of 9.49. There is no statistical significance between the level of risk factors and demographic variables.

SECTION E : Association between level of physiological base line data and selected demographic variables

TABLE - 12

ASSOCIATION BETWEEN THE GRADE OF BMI AND SELECTED DEMOGRAPHIC VARIABLES

n = 400

Selected Demographic variables	Under BMI<18.50		Normal range 18.50-24.99		Over weight ≥ 25.00		Chi square
	No.	%	No.	%	No.	%	
Sex							7.95
Male	40	22.2	132	73.3	8	4.4	d.f=2
Female	96	43.6	100	45.3	24	10.9	p<0.01 (Sig)
Age in years							9.03
18-30	24	35.3	40	58.8	4	5.9	d.f = 10
31-36	28	38.9	40	55.6	4	5.6	p=0.53 (N.S)
37-42	24	27.3	56	63.6	8	9.1	
43-48	20	23.8	48	57.1	16	19.0	
49-54	20	62.5	12	37.5	-	-	
55-60	20	35.7	36	64.3	-	-	
Occupation							12.13
Unskilled	64	43.2	80	54.1	4	2.7	d.f = 10
Skilled	20	35.7	32	57.1	4	7.1	p=0.28 (N.S)
Office Workers	-	-	28	87.5	4	12.5	
Business	12	21.4	40	71.4	4	7.1	
Retired	-	-	8	100.0	-	-	
No occupation	40	40.0	44	44.0	16	16.0	
Income							17.09
<Rs.500	40	40.0	44	44.0	16	16.0	d.f = 8
Rs.501 - Rs.1000	28	31.8	66	68.2	-	-	p<0.05 (Sig)
Rs.1001 - Rs.1500	44	50.0	40	45.5	14	4.5	
Rs.1501 - Rs.2000	20	45.5	24	54.5	-	-	
>Rs.2000	4	5.0	64	80.0	12	15.0	
History of CVD							7.09
No	80	32.8	148	60.7	16	6.6	d.f = 8
Paternal	4	14.3	24	85.7	-	-	p=0.53 (NS)
Maternal	8	22.2	24	66.7	4	11.1	
Both	4	50.0	4	50.0	-	-	
Don't know	40	47.6	32	38.1	12	14.3	

Table 12 depicts the association between the level of BMI and selected demographic variables. Analysis shows that 73.3% of the male population had normal BMI (18.50-24.99kg/m²) and 4.4% of men were overweight (BMI ≥ 25.00 kg/m²). Statistically there is a significant association between the level of BMI with sex at the level p<0.01. Also there is a statistically significant association between the level of BMI and income (Fig.7).

DISCUSSION

The first objective was to estimate the prevalence of selected modifiable risk factors of cardio vascular diseases (Hypertension, obesity, smoking, physical inactivity, diet and alcohol) in adults aged 18 years and above at Soolamangalam Village, Thanjavur District.

HYPERTENSION

Analysis of the collected Data in table 2 depicts 80% of the population fell in optimal systolic (< 120 mm Hg) blood pressure and 85% in optimal diastolic (< 80 mm Hg) blood pressure, 6% of the population fell in Grade I systolic (140 - 159 mm Hg) hypertension and 8% fell in grade I diastolic (90 - 99 mm Hg) hypertension. Only 1% of population fell in grade II systolic (160 - 179 mm Hg) hypertension.

OBESITY

The frequency and percentage distribution of body mass index (BMI) in Table 3 reveals that 7% were over weight (≥ 25.00 kg/m²) and 1% fell in the category of pre obese (25.01 - 29.99 kg / m²). Table 12 reveals that there is a significant association between BMI and sex of the population at $P < 0.01$ level.

SMOKING

Analysing the data on smoking habits Table 10 reveals that 9% of the smokers are under high risk and 6% are under moderate risk.

PHYSICAL INACTIVITY

Regarding Exercise, as evident from Table - 10, 74% of the population are under moderate risk and 24% are under High risk. The same fact is appreciated by Paffenbarger et al., who found that men who did not exercise regularly were at 35% greater risk of being diagnosed as Hypertensive than those who exercised. WHO (1996) reports that exercise will lower systolic and diastolic BP by 5 - 10 mm Hg and sedentary individuals have 20 - 50% higher risk of developing CVD.

DIET

In relation to the Diet, 86% of the population were under moderate risk and 3% under high risk. Table 7 reveals that 23% of the population take coffee more than 2 times a day. It is evident from the Table - 7 that 18% of the study population are at risk for developing hypertension and CVD, as they consume pickles daily, 19% eat pappad two or more days in a week, and 16% consume dried fish two or more days in a week.

ALCOHOL

Regarding Alcohol consumption that, 17% are under moderate Risk and only 1% are under high risk.

The Second objective was to associate the data with various demographic variables. Chi Square and Analysis of variance (ANOVA) were

used to test the association of demographic variables with level of Risk factors. There was no Association between the level of Risk factors with demographic variables. However an association between the level of BMI with selected Demographic Variables in Table 12 found that BMI is significantly associated with Sex (at $P = < 0.01$ level) and BMI is also significantly associated with Income at $p = 0.05$ level.

The third objective was to identify the Adults with high risk factors of CVD and provide necessary health care. Adults with high risk modifiable risk factors of CVD were identified in Soolamangalam Village. They were given preventive and curative Health Care appropriately.

RECOMMENDATIONS

CVD is preventable, and the following strategies are recommended :

- a. Population strategy
 - i. Primary prevention
 - ii. Primordial prevention
- b. High risk strategy
- c. Secondary prevention

a. i. Primary Prevention

CVD is primarily a mass disease. The strategy should therefore be based on mass approach focusing mainly on the control of underlying causes (risk factors) in whole populations, not merely in individuals. This approach is based on the principle that small changes in risk factor levels in total populations can achieve the biggest reduction in mortality³⁸. That is, the aim should be to shift the whole risk - factor distribution in the direction of "biological normality"³⁹. This cannot obviously be done by medical means alone; it requires the mobilization and involvement of the whole community to alter its life - style practices that are associated with CVD.

Specific interventions

The population strategy centres round the following key areas :

1. Dietary changes : Dietary modification is the principal preventive strategy in the prevention of CVD. The WHO Expert Committee³⁹ considered the following dietary changes to be appropriate for high incidence populations :

- reduction of fat intake to 20-30 per cent of total energy intake.
- consumption of saturated fats must be limited to less than 10 per cent of total energy intake; some of the reduction in saturated fat may be made up by mono - and poly - unsaturated fats.
- a reduction of dietary cholesterol to below 100 mg per 1000 kcal per day.
- an increase in complex carbohydrate consumption (i.e., vegetables, fruits, whole grains and legumes)
- avoidance of alcohol consumption; reduction of salt intake to 5 g daily or less.

2. Smoking : The goal should be to achieve a smoke - free society, and several countries are progressing towards this goal.

To achieve the goal of a smoke - free society, a comprehensive health programme would be required which includes effective information and

education activities, legislative restrictions, fiscal measures and smoking cessation programmes.

3. Blood pressure : It has been estimated that even a small reduction in the average blood pressure of the whole population by a mere 2 or 3 mm Hg would produce a large reduction in the incidence of cardiovascular complications^{40,41}. The goal of the population approach to high blood pressure would thus be to reduce mean population blood pressure levels. This involves a multifactorial approach based on a "prudent diet" (reduced salt intake and avoidance of a high alcohol intake), regular physical activity and weight control. The potential benefits and the safety and low cost of this advice would justify its implementation.

4. Physical activity : Regular physical activity should be a part of normal daily life. It is particularly important to encourage children to take up physical activities that they can continue throughout their lives³⁹.

a. ii. Primordial prevention

A novel approach to primary prevention of CVD is primordial prevention. It involves preventing the emergence and spread of CVD risk factors and life styles that have not yet appeared or become endemic. This applies to developing countries in particular. These countries should seek to preserve their traditional eating patterns and life - styles associated with low levels of CVD risk factors.

Since the aetiology of CVD is multifactorial the approach to prevention should be multifactorial aimed at controlling or modifying as many risk factors as possible. The aim should be to change the community as a whole, not the individual subjects living in it⁴².

b. High risk strategy

1. Identifying risk : High risk intervention can only start once those at high risk have been identified. By means of simple tests such as blood pressure and serum cholesterol measurement it is possible to identify individuals at special risk³⁹. Individuals at special risk also include those who smoke, those with a strong family history of CVD, diabetes and obesity and young women using oral contraceptives.

2. Specific advice : Having identified those at high risk, the next step will be to bring them under preventive care and motivate them to take positive action against all the identified risk factors, e.g., an elevated blood pressure should be treated, the patient should be helped to break the smoking habit permanently - nicotine chewing gum can be tried to wean patients from smoking⁴³; serum cholesterol concentration should be reduced in those in whom it is raised, etc.

From a methodological point of view, however, high - risk approach suffers from the disadvantage that the intervention (e.g., treatment) may be effective in reducing the disease in a high - risk group, but it may not reduce

the disease to the same extent in the general population which consists of symptomatic, asymptomatic, high - risk, low - risk and healthy people⁴⁴. Further, unfortunately, more than half of the CVD cases occur in those who are not apparently at special risk, and this is one limitation of the high - risk strategy. Nevertheless, recognition and treatment of high - risk cases do make an important contribution to prevention.

c. Secondary prevention

Secondary prevention must be seen as a continuation of primary prevention. It forms an important part of an overall strategy. The aim of secondary prevention is to prevent the recurrence and progression of CVD. Secondary prevention is a rapidly expanding field with much research in progress (e.g., drug trials, coronary surgery, use of pace makers).

The principles governing secondary prevention are the same as those already set out in the above sections, e.g., cessation of smoking, control of hypertension and diabetes, healthy nutrition, exercise promotion, etc. The most promising results to date have come from beta - blockers which appear to reduce the risk of CHD mortality in patients who have already suffered at least one infarct in the order of 25 per cent. None of the preventive measures discussed earlier lose their importance even after the first attack. For example, cessation of smoking is the most effective single means of intervention currently available in the management of patients after a heart attack. The risk

of fatal infarction or sudden death is reduced by 20 - 50 per cent. If the patient does not stop smoking, nothing else is worth doing³⁸.

Despite advances in treatment, the mortality of an acute heart attack is still high : among survivors, around 10 per cent in the first year, and 5 per cent yearly thereafter. Delay in reaching hospital is still considerable even in big cities in the West and may be as much as 3.5 hours. About 30 per cent of all deaths occur within 30 minutes of onset. This is one of the reasons why coronary care units have failed to make impact on the total coronary mortality in the community^{45,46}.

Each strategy - population strategy, high - risk strategy, secondary prevention - has its advantages and disadvantages, but the population strategy has the greatest potential.

Revascularization procedures for patients with angina pectoris

The indications for coronary artery revascularization i.e. coronary artery bypass grafting (CABG) and percutaneous transluminal coronary angioplasty (PTCA) in patients with angina pectoris are often debated⁴⁷. There is general agreement that otherwise healthy patients in the following groups should undergo revascularization. (a) patients with unacceptable symptoms despite medical therapy to its tolerable limits; (b) patients with left main coronary artery stenosis greater than 50 per cent with or without symptoms; (c) patients with three vessel disease with left ventricular dysfunction (ejection fraction <

50 per cent or previous transmural infarction). (d) patients with unstable angina who after symptom control by medical therapy continue to exhibit ischaemia on exercise testing or monitoring; and (e) post - myocardial infarction patients with continuing angina or severe ischaemia on noninvasive testing.

CABG can be accomplished with a very low mortality rate (1-3 per cent) in otherwise healthy patients with preserved cardiac function. However, the mortality rate of this procedure rises to 4-8 per cent in older individuals and in patients who have had a prior CABG. Increasingly, younger individuals with focal lesions of one or more vessels are undergoing coronary angioplasty as the initial revascularization procedure, where coronary artery stenosis can be effectively dilated by inflation of a balloon under high pressure. PTCA is also possible but less successful in bypass graft stenosis.

The incidents of restenosis appears to be reduced with intracoronary stent placement and may be as low as 15-20 per cent. The number of PTCA and stent procedure now exceeds that of CABG operations. Several studies have shown PTCA to be superior to medical therapy for symptom relief but not in preventing infarction or death. In patients with no or only mild symptoms, aggressive lipid - lowering and antianginal therapy may be preferable to PTCA.

SUMMARY AND CONCLUSIONS

A cross sectional descriptive study of modifiable risk factors of cardiovascular diseases in adults at Soolamangalam Village, Thanjavur was conducted in a Rural Area of Central Tamil Nadu.

The study revealed the following findings :

1. 6% are having Grade I systolic hypertension (mild) and 1% are having Grade II systolic hypertension (moderate) while 8% of the population are having Grade I Diastolic Hypertension.
2. 7% are obese and 1% fall in the category of pre obese.
3. 9% of the smokers are under high risk and 6% are under moderate risk.
4. 74% of the sample population are under moderate risk, while 24% are under high risk as far as physical inactivity is concerned.
5. As per diet, 86% of the adults are under moderate risk and 3% under high risk. 87% of the population are non-vegetarians, 23% takes coffee more than two times in a day, 18% consumes pickle daily, 19% takes pappads two or more days and 16% take dried fish two or more days in a week.

6. In Alcohol consumption, 17% are under moderate risk and only 1% are under high risk.

The study concludes that the majority of the study population 90% have a moderate risk of developing CVD. Therefore, proactive preventive steps through healthy life style practices can prevent a major catastrophe in future.

LIMITATIONS

1. The study is carried out only in one village in a Rural Area and limited to selected modifiable risk factors.
2. Blood pressure measurements obtained on a single occasion only.
3. Since the study elicits the smoking, physical inactivity, diet, alcohol in the past 3 months, the possibility of recall bias cannot be ruled out.

**MODIFIABLE RISK FACTORS OF CARDIO VASCULAR DISEASES
IN ADULTS AT SOOLAMANGALAM VILLAGE, THANJAVUR
- A CROSS SECTIONAL DESCRIPTIVE STUDY**

PROFORMA

PART - I

SOCIO DEMOGRAPHIC DATA

- | | | | | |
|----|----------------|---|----------------------|-----|
| 1. | Code Number | : | | |
| 2. | Sex | : | Male | [] |
| | | | Female | [] |
| 3. | Age | : | 18 years to 24 years | [] |
| | | : | 25 years to 30 years | [] |
| | | : | 31 years to 36 years | [] |
| | | : | 37 years to 42 years | [] |
| | | : | 43 years to 48 years | [] |
| | | : | 49 years to 54 years | [] |
| | | : | 55 years to 60 years | [] |
| 4. | Education | : | Illiterate | [] |
| | | : | Primary | [] |
| | | : | Middle School | [] |
| | | : | High School | [] |
| | | : | Hr. Secondary | [] |
| | | : | Graduate | [] |
| | | : | Post graduate | [] |
| 5. | Marital Status | : | Unmarried | [] |
| | | : | Married | [] |
| | | : | Separated | [] |
| | | : | Divorced | [] |
| | | : | Widow / Widower | [] |
| 6. | Type of Family | : | Joint Family | [] |
| | | : | Nuclear Family | [] |
| 7. | Religion | : | Hindu | [] |
| | | : | Christian | [] |
| | | : | Muslim | [] |

II. WHAT FORM OF TOBACCO YOU USE?

- 1. Cigar (Churuttu) []
- 2. Beedi []
- 3. Pipe []
- 4. Filter Less cigarette []
- 5. Filter cigarette []

III. HOW MANY CIGARETTE DO YOU SMOKE A DAY?

- 1. More than 20 []
- 2. 16 - 20 []
- 3. 11 - 15 []
- 4. 6 - 10 []
- 5. 1 - 5 []

D. PHYSICAL ACTIVITY (SCORE 5 - 1) TOTAL 5 x 5 = 25

1. HOW MANY HOURS OF HOUSE HOLD ACTIVITIES YOU DO IN A DAY?

- 1. Never do []
- 2. Less than 2 hours []
- 3. 2 - 5 hours []
- 4. 6 - 9 hours []
- 5. More than 10 hours []

2. HOW MANY HOURS OF EXTERNAL ACTIVITIES YOU DO IN A DAY?

- 1. Never do []
- 2. Less than 2 hours []
- 3. 2 - 5 hours []
- 4. 6 - 9 hours []
- 5. More than 10 hours []

3. HOW MANY HOURS OF REST DO YOU TAKE INCLUDING NAP?

- 1. More than 15 hours []
- 2. 12 - 14 hours []
- 3. 9 - 11 hours []
- 4. 6 - 8 hours []
- 5. Less than 5 hours []

4. DO YOU GO FOR ANY EXERCISES APART FROM DAILY ACTIVITIES?

- 1. Never do []
- 2. Fast walking []
- 3. Fast cycling []
- 4. Swimming []
- 5. Gymnasium []

5. IF YOU DO EXERCISE HOW MANY HOURS YOU DO?

- 1. Less than 30 mts []
- 2. 31 mts - 1 hour []
- 3. 1.01 hr - 1.30 hrs []
- 4. 1.31 hr - 2 hours []
- 5. More than 2 hours []

E. DIET (SCORE 5 - 1) TOTAL 9 X 5 = 45

1. WHAT IS YOUR FOOD PATTERN ?

- 1. Lacto + Ova vegetarian + Mutton + Chicken + Fish + Beef + Pork + Organ Meat []
- 2. Lacto + Ova vegetarian + Mutton + Chicken + Fish + Beef []
- 3. Lacto + Ova vegetarian + Mutton + Chicken + Fish []
- 4. Lacto + Ova Vegetarian []
- 5. Vegetarian []

2. IF NON - VEGETARIAN HOW OFTEN DO YOU TAKE

- 1. Daily []
- 2. Two or more days in a week []
- 3. Once in a week []
- 4. Once in a month []
- 5. Rarely []

3. WHAT TYPE OF OIL DO YOU USE FOR COOKING

- 1. Coconut Oil []
- 2. Ground nut Oil []
- 3. Gingily Oil []
- 4. Palm Oil []
- 5. Refined Oil []

4. HOW OFTEN DO YOU USE FRIED FOODS?

- 1. Daily []
- 2. Two or more days in a week []
- 3. Once in a week []
- 4. Once in a month []
- 5. Rarely []

5. DO YOU TAKE COFFEE?

- 1. More than two times a day []
- 2. Twice a day []
- 3. Only once in a day []
- 4. Rarely []
- 5. Never Take []

6. DO YOU TAKE PICKLES?

- 1. Daily []
- 2. Two or more days in a week []
- 3. Once in a week []
- 4. Rarely []
- 5. Never Take []

7. DO YOU TAKE PAPADS?

- 1. Daily []
- 2. Two or more days in a week []
- 3. Once in a week []
- 4. Rarely []
- 5. Never Take []

8. DO YOU TAKE DRIED FISH ?

- 1. Daily []
- 2. Two or more days in a week []
- 3. Once in a week []
- 4. Rarely []
- 5. Never Take []

9. DO YOU HAVE THE HABIT OF EATING SNACKS?

- 1. Daily []
- 2. Two or more days in a week []
- 3. Weekly Once []
- 4. Rarely []
- 5. Never Take []

F. ALOCHOL (SCORE 5 - 1) TOTAL 3 x 5 = 15

1. DO YOU CONSUME ALCOHOL?

- 1. Daily []
- 2. Two or more days in a week []
- 3. Weekly Once []
- 4. Rarely []
- 5. Never do []

2. WHAT TYPE OF ALOCHOL YOU USE?

- 1. Arrack (Country liquor) []
- 2. Gin / Brandy / Rum / Whisky []
- 3. Fortified wine []
- 4. Beer []
- 5. Toddy []

3. NORMALLY HOW MUCH DO YOU CONSUME INA DAY

- 1. More than 390 ml (13 pegs and above) []
- 2. 300 ml - 360 ml (10 - 12 pegs) []
- 3. 210 ml - 270 ml (7 - 9 pegs) []
- 4. 120 ml - 180 ml (4 - 6 pegs) []
- 5. 30 ml - 90 ml (1 - 3 pegs) []

BIBLIOGRAPHY

1. WHO (2002). Health Situation in the South - East Asia Region 1998 - 2000, New Delhi.
2. WHO (1982) Techn. Rep. Ser. No.678.
3. Rose .G (1982) European Heart J, 3, Suppl. B.18.
4. Sinha B.C., (1970), J. Ind. Med. Assoc. 55 : 171.
5. Dawber, T.R. (1980), The framingham study, cambridge, M.A., Harvard University Press.
6. Kanel, W.B. et al., (1976), Am. J. Cardiol., 38 : 46.
7. Sarvothan S.G. and Berry J.N. (1968), Circulation, 37 : 339.
8. Dewan B.D., et al., (1974). Indian Heart J., 26 : 68.
9. Verma, I.C ed., (1986). Genetic Research in India, Sager Printers and Publishers, New Delhi.
10. Gupta S.P. et al., (1978), Ind. Heart J., 30 : 315.
11. Gupta S.P. et al., (1977), Ind. Heart J. 29 : 53.
12. WHO (1995), Tech. Rep. Ser. No.854.
13. WHO (1996), Techn. Rep. Ser. No.862.
14. Hart J.T. (1980) Hypertension, Library of General Practitioner, Series, Churchill Livingstone.
15. WHO (1983), Bull WHO, 61(1) 53.
16. WHO (1985), Primary Prevention of CHD, EURO, Rep and Studies 98, Copenhagen.
17. Hager, A (1981). Br. Med. Bull., 37 (3) 287.
18. Aykroyd, W.R and J. Mayer (1968), Food and Nutrition, Terminology, In: WHO Doc. Nu. 68.6, Geneva.
19. WHO (2003), Tech. Rep. Ser. No.916.

20. Slone, D. et al., (1978), N. Eng. J. Med. 298 : 1273.
21. Shaper A.G. et al., (1981), Brit Med. J. 283 : 179.
22. WHO (1979), Tech. Rep. Ser. No.636.
23. Bain, C, et al., (1978), Lancet, 1 : 1087.
24. Wald, N.J. (1976), Lancet, 1 : 136.
25. Miller, N.E. et al., (1979), Lancet, 1 : 111.
26. WHO (2003), Tech. Rep. Ser. No.916.
27. Falkner, F. ed., (1980) Prevention in Childhood of Health Problems in Adult Life, WHO, Geneva.
28. The pooling project Research Group (1978), J. Chr. Dis., 31 : 201.
29. Superko, H.R. et al., (1985), Am. J. Med., 78; 826 - 838.
30. Oliver, M.F. (1981) Br.Med. Bull, 37(1), 49 - 58.
31. Morris, J.N. et al., (1977), Brit Med. J. 2 : 1307 - 1314.
32. WHO (1985) primary prevention of CHD EURO Repand Studies 98, Copenhagen.
33. WHO (1986), Techn. Rep. Ser, No.732.
34. Hart, J.T. (1980). Hypertension, Library of General Practitioner Series, Churchill Livingstone.
35. WHO (1995). Tech. Rep. Ser. No.854.
36. Surveillance of Risk factors for Non communicable Diseases. The WHO STEP wise approach. Non communicable Diseases. and Mental Health, WHO, Geneva, 2003.
37. Gupta R. Trends in Hypertension Epidemiology in India J. Hum. Hypertens, 2004 : 18; 73 - 78.
38. Bradely .N (1984). In Medical Annual. D.J.P. Gray (ed). John Wright and Sons.

39. WHO (1982) Techn. Rep. Ser. No. 678.
40. WHO (1986) Techn. Rep. Ser. No.732.
41. Rose.G (1981). Brit. Med. J. 282 : 1847.
42. Puska. P et al., (1979). Brit. Med. J. 2 : 1173.
43. Lancet (1985) 1: 320.
44. Glesunov I, et al., (1973). Int. J. Epi2 (2), 137.
45. Rose. G. (1975). Brit. J. PSM, 29 : 147.
46. G.Lamm. (1979). In : Measurement of levels of Health, WHO Reg. Publ. EURO. Ser. No.7.
47. Lawrence M. Tierney Jr. Stephen J. Mcphee Maxine A. Papadakis, Current Medical Diagnosis and Treatments 41 sted. 2002. Lange Publication.
48. Park .K. Park's Text Book of Preventive and Social Medicine 18th Edition, 2005.
49. Strasser, T. (1972). WHO Chronicle, 26 : 451.
50. WHO (1974). WHO, Chronicle, 28(3) 11.
51. WHO (1978). Techn. Rep. Ser. No.628.
52. WHO (1962). Techn. Rep. Ser. No.231.
53. Miller, N.E. et al., (1979) Lancet 1 : 111.
54. Beaton G.H. (1976). In Nutrition in Preventive Medicine Annex 2, P.482, Beaton, G.H. and J.M. Bengoa (eds), WHO. Geneva Monograph Ser. No.62.
55. WHO (1996). Techn. Rep. Ser. No.862, Hypertension Control.
56. Bhirmanandam C.V. et al., (1988). Antiseptic Dec. 1988 : S1.
57. Dawber T.R. (1980). The Framingham Study Cambridge, M.A., Harvard University Press.

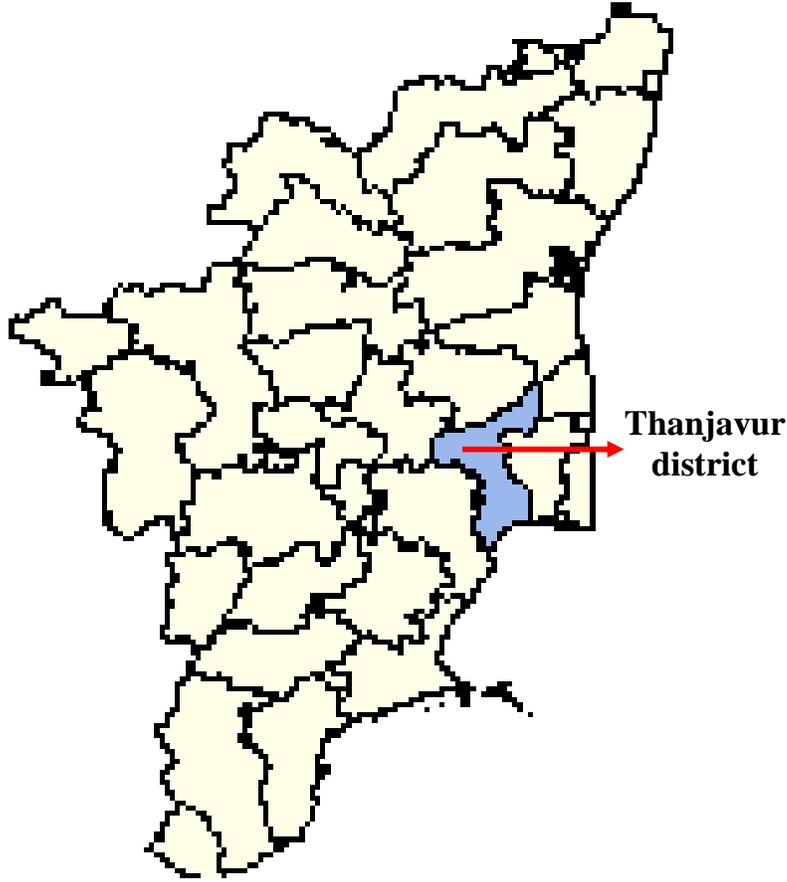
58. Kannel, W.B. et al., (1976). Am. J. Cardiol, 38 : 46.
59. WHO (1996). Techn. Rep. Ser. No.862.
60. Puska P. et al., (1983). Lancet, 11 : 1 - 5.
61. Beard, T.C. et al., (1982), Lancet, 2 : 455.
62. WHO (1997) EMRO : Techn. Pub. 22, Prevention and Control of Cardio Vascular Diseases.
63. WHO (2005). Diet, Nutrition and The Prevention of Chronic Diseases Tech. Rep. Ser. No.916.
64. Cardiovascular Disease - Diet, Nutrition and Emerging Risk factors - Keith Frayn, Sara Stanner First edition 2005. Blackwell Publishing.
65. South Asian Journal of Preventive Cardiology Vol.8, Number 3. 2004.
66. Heart Diseases A Text Book of Cardio Vascular Medicine Zipes, Libby, Bonow, Braunwald Elsevier Saunders 7th Edition 2005.
67. Cardiovascular Medicine James Willerson, Jay Cohn, Churchill Livingstone Second Edition, 2000 Edition.
68. Text Book of Cardiovascular Medicine Eric Topol Lippincott William & Wilkins Second Edition, 2002.
69. Coronary Artery Disease Essentials of Prevention and Rehabilitation Programs Peter Brubaker, Leonard Kaminsky, Mitchell Whaley, Human Kinetics, 2002.
70. Evidence based cardiology Salim Yusuf. BMJ Books, Second Edition, 2003.
71. Cardiology Michael Crawford John Dimarco, Walter Paulus Mosby Second Edition 2004.
72. Coronary Artery Disease in South Asians Epidemiology, Risk factors and Prevention Editor, G.H.R. Rao, 2001, Jaypee Brochers.
73. Coronary Artery Disease : Risk Promoters, Pathophysiology and Prevention Editor, G.H.R. Rao, 2005, Joypee Brothers.

74. Ligy Thomas K. Risk factors of Coronary Heart Disease. The Antiseptic Vol.91, No.5
75. Arun Bordia, Satish Arora - A comparative study on predisposing factors of Coronary Artery Disease in Rural and Urban Population. Indian J. Med. Res. 62, 4, April 1974.
76. J.Kaur, K.Bains. A study of the Risk factor profile of cardiovascular in Diseases Rural Punjabi Male Patients IJPH April - June 2006.
77. P.Mohanan, Risk Factors of Coronary Heart Disease in a Selected Community IJCM. Oct. Dec. 2005.
78. Natarajan V.S., Ischaemic Heart Diseases in Rural Elderly. The Antiseptic Vol.92, No.10.
79. V.K.Agrawal. Coronary Risk Factors in a Rural Community, IJPH Jan. - March. 2006.
80. Rajeev Gupta, Prevalence and Determinants of Coronary Heart Disease in a Rural Population of India. J. Clin. Epidemiology, Vol.50, No.2, pp.203 - 209, 1997.
81. S.L.Chada, Urban - Rural Differences in the prevalence of Coronary Heart Disease and its Risk factors in Delhi. WHO Bulletin OMS. Vol.75, 1997.
82. Enas A Enas. Coronary Artery Disease Epidemic in Indians : A cause for Alarm and Call. for Action. JIMA, Vol.98, No.11, Nov.2000.
83. Rajeev Gupta, Prevention of Coronary Heart Disease among Indians : Focus on primary prevention JIMA, Vol.98, No.11, Nov.2000.
84. Rajeen Gupta. Prevention of Coronary Heart Disease in India. An Epidemiological Perspective IJCM. Oct. - Dec. 2002.
85. S.P.Singh P. Sen. Coronary Heart Disease : The changing Scenario IJPSM Jan - June 2003.
86. K.Srinath Reddy, Responding to the threat of Chronic Diseases in India. The Lancet Vol.366, Nov.12, 2005.
87. Maxcy - Rosenau Last. Public Health and Preventive Medicine, 14th Edition, 1998. Mcgraw Hill.

88. Detels, McEwen, Beaglehole, Tanaka, Oxford Text Book of Public Health 4th Edition, Oxford, 2004.
89. WHO (2005), Cardiovascular Survey Methods Third Edition Russell V. Luepker.
90. Sunder Rao. An introduction to Biostatistics prentice Hall of India 4th edition, 2005.
91. Swaminathan M. Food & Nutrition. The Bangalore Printing and Publishing Co. Second Edition, Bangalore.

ABBREVIATIONS

NCD	-	Non communicable disease
CVD	-	Cardio vascular disease
RHD	-	Rheumatic heart disease
CHD	-	Coronary heart disease
IHD	-	Ischaemic heart disease
DBP	-	Diastolic blood pressure
SBP	-	Systolic blood pressure
BMI	-	Body mass index
WHO	-	World health organization
PHC	-	Primary health centre
ECG	-	Electro cardio gram
VLDL	-	Very Low density lipoproteins
LDL	-	Low density lipoproteins
HDL	-	High density lipoproteins
HTN	-	Hypertension
IEC	-	Information, Education & Communication
AMI	-	Acute myocardial infarction
TG	-	Triglycerides
BP	-	Blood pressure
NV	-	Non - vegetarian
NS	-	Not Significant



Thanjavur District
Blocks



(Map Not to Scale)
Digital Map Source : TWAD Board, Chennai
Web Design : NIC, TNSC

Thanjavur : Papanasam Block
Panchayat Villages



(Map Not to Scale)
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- 1. 20 US® ÷©Ø£mh []
- 2. 16 & 20 []
- 3. 11 & 15 []
- 4. 6 & 10 []
- []
- 5. 1 & 5 []

D. EhØ ö\ ØvÓß (©v`ö£s 5&1) ö©õzu® : 5 x 5 = 25

1. J, {õøÍUS GzuøÚ ©o ÷{µ® }]]PÒ Ámk ÷Áø»PÒ ö\`ÃµPÒ?

- 1. ö\`ÁvÀø» []
- 2. 2 ©o ÷{µzvØS® SøÓÁõÚ []
- 3. 2 & 5 ©o ÷{µ® []
- 4. 6 & 9 ©o ÷{µ® []
- 5. 10 ©o ÷{µzvØS® AvPCõP []

2.]]PÒ J, {õøÍUS öÁÍ ÷Áø»PÒ GzuøÚ ©o ÷{µ® ö\`Ã°PÒ?

- 1. ö\`ÁvÀø» []
- 2. 2 ©o ÷{µzvØS® SøÓÁõÚ []
- 3. 2 & 5 ©o ÷{µ® []
- 4. 6 & 9 ©o ÷{µ® []
- 5. 10 ©o ÷{µzvØS® AvPCõP []

3.]]PÒ GzuøÚ ©o ÷{µ® K`Ä Gk`¥°PÒ? CµÄ ÷{µ yUP-® ÷\°zx?

- 1. 15 ©o ÷{µzvØS® ÷©À []
- 2. 12 & 14 ©o ÷{µ® []
- 3. 9 & 11 ©o ÷{µ® []
- 4. 6 & 8 ©o ÷{µ® []
- 5. 5 ©o ÷{µzvØS® SøÓÁõÚ []

4.]]PÒ vÚ ÷Áø»PøÍ uÂµ HuõÁx EhØ£°Ø] ÷©ØöPõÒÃ°PÍõ?

- 1. ö\`ÁvÀø» []
- 2. ÂøµÄ {øh []
- 3. ÂøµÄõP av Ási KmkuÀ []
- 4. }a\À []
- 5. ÷uP`£°Ø] []

5.]]PÒ EhØ£°Ø] ÷©ØöPõÒÁuõÚõÀ GzuøÚ ©o ÷{µ® ö\`Ã°PÒ?

1. 30 |^ah_vØS® SøÓÁðÚ []
2. 31 |^ah® ñuÀ 1 ©o÷{μ® []
3. 1.01 ©o÷{μ® ñuÀ 1.30 ©o÷{μ® []
4. 1.31 ©o÷{μ® ñuÀ 2 ©o÷{μ® []
5. 2 ©o÷{μz_vØS® AvP©ðP []

E. EnÄ (©v¨ ö£s 5&1) ö©özu® 9 x 5 = 45

1. E[PÒ EnÄ¨ £ÇUP® GßÚ?

1. £ðÀ + ñmøh ø\Á® + Bmk CøÓa] + ÷PðÈ CøÓa] + «ß CøÓa] + ©ðmk CøÓa] + £ßÔ CøÓa] + EÖ¨i CøÓa] []
2. £ðÀ + ñmøh ø\Á® + Bmk CøÓa] + ÷PðÈ CøÓa] + «ß CøÓa] + ©ðmk CøÓa] []
3. £ðÀ + ñmøh ø\Á® + Bmk CøÓa] + ÷PðÈ CøÓa] + «ß CøÓa] []
4. £ðÀ + ñmøh ø\Á® []
5. ø\Á® []

2. }[PÒ Aø\Á® GÙÀ, G¨ö£ðÈx GÀ»ð® }[PÒ Es¥ºPÒ?

1. vÚ¬® []
2. J, Áðμz_vØS Çμsk (A) AuØS ÷©Ø£mh []
3. Áðμz_vØS J,¬øÓ []
4. ©ðuz_vØS J,¬øÓ []
5. A§ºÁ©ðP []

3. \ø©^{-3/4}US GßÚ ÁøP¬ öÚ Gsön´ £¬ßkzxQÖºPÒ?

1. ÷u[Pð´ Gsön´ []
2. Phø» Gsön´ []
3. {Àö»sön´ []
4. £ð©ðºÁ []
5. _zvP¶UP¨£mh Gsön´ []

4. }[PÒ GsönºÀ ö£ðÔzu EnÄ ö£ð,mPøí G¨ö£ðÈöuÀ»ð® E£÷¬øQ¨¥ºPÒ?

1. vÚ¬® []
2. J, Áðμz_vØS Çμsk (A) AuØS® ÷©Ø£mh {ðmPÒ []
3. Áðμz_vØS J,¬øÓ []
4. ©ðuz_vØS J,¬øÓ []
5. A§ºÁ©ðP []

5. }[PÒ Pð¨¤ Sî¨¥ºPíð?

1. vÚ-® CμskuhøÁUS÷©À []
2. vÚ-® CμskuhøÁ ©mk® []
3. vÚ-® J,uhøÁ ©mk® []
4. Aξ°Á©õP []
5. Es£vÀø» []

6. }[PÒ FÖPö´ Es¥°Píõ?

1. vÚ-® []
2. J, ÁõμzvØS Cμsk (A) AuØS® ÷©Ø£mh {õmPÒ []
3. ÁõμzvØS J,¬øÓ []
4. Aξ°Á©õP []
5. Es£vÀø» []

7. }[PÒ A´£í® Es¥°Píõ?

1. vÚ-® []
2. J, ÁõμzvØS Cμsk (A) AuØS® ÷©Ø£mh {õmPÒ []
3. ÁõμzvØS J,¬øÓ []
4. Aξ°Á©õP []
5. Es£vÀø» []

8. }[PÒ P,Áõk Es¥°Píõ?

1. vÚ-® []
2. J, ÁõμzvØS Cμsk (A) AuØS® ÷©Ø£mh {õmPÒ []
3. ÁõμzvØS J,¬øÓ []
4. Aξ°Á©õP []
5. Es£vÀø» []

9. E[PDUS ö{öÜSzwÛ Esq® £ÇUP® Eshõ?

1. vÛ-® []
2. J, ÁõµzvØS Cµsk (A) AuØS® ÷©Ø£mh {õmPÒ []
3. ÁõµzvØS J, -øÓ []
4. AŞ°Á©õP []
5. Es£vÀø» []

F. ©x£õÚ® (©v¨ö£s 5&1) ö©õzu® 3 x 5 =15

1. }[PÒ ©x£õÚ® A, çxÃ°Píõ?

1. vÛ-® []
2. J, ÁõµzvØS Cµsk (A) AuØS® ÷©Ø£mh {õmPÒ []
3. ÁõµzvØS J, -øÓ []
4. AŞ°Á©õP []
5. A, çxÁvÀø» []

2. }[PÒ GBÚ ÁøP¯ õÚ ©x£õÚ® A, çxÃ°PÒ?

1. \õµõ¯ ® []
2. âß, µµõçv, µ®, ÂìQ []
3. £õxPõUP¯£mh J°ß []
4. ¥° []
5. PÒ []

3. }[PÒ J, {õóíUS GÆÁíÄ ©x£õÚ® A, çxÃ°PÒ?

1. 390 ^{a1/4}US AvP©õP (13 ö£U) []
2. 300 ^{a1/4} -uÀ 360 ^{a1/4} (10&12 ö£U) []
3. 210 ^{a1/4} -uÀ 270 ^{a1/4} (7&9 ö£U) []
4. 120 ^{a1/4} -uÀ 180 ^{a1/4} (4&6 ö£U) []
5. 30 ^{a1/4} -uÀ 90 ^{a1/4} (1&3 ö£U) []

COPY OF TOOL FOR DATA COLLECTION (TAMIL)

ÁnUP®!

{õß ö\ßøÚ ©,zxÁUPÀ;¶ \-P©,zxÁ E⁻°|ø»z xøÓ°À -x|ø» £mh⁻£i⁻!
\-P©,zxÁ® -sÓõ® Bsk £izx Á,Q÷Óß. {õß £,Á-ø÷Óõ°US, Cu⁻ {õí÷{õ´Pøí
HØ£kzx®]» ©õøÓUTi⁻ PõµoPøí ÷u⁰øöukzx AøÁ £µÂ°,zuø»⁻ £øÔ B´Ä
{hzxQ÷Óß. CxSÔzx E[PÍß \®©uzøu²®, JzxøÇ⁻ø£²® Gv⁰£õ°UQ÷Óß.

{ßÔ,

u[PÒ Esø©²Òí