HABITUAL DIETARY INTAKES AND LEVELS OF RISK FACTORS ASSOCIATED WITH CORONARY HEART DISEASE AMONG INDIAN MATRICULANTS IN LENASIA

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DEDICATION

This thesis is dedicated to:

My husband Sayed Hoosen Mia,

my daughter Razia Banoo Mia, and

my sons, Sayed Mohamed Ridwaan Mia,

Zaakir Hoosein Mia and Mohamed Reza Mia.

My parents Mr and Mrs Abdulla and Ayesha Khan.

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SUMMARY

The incidence of coronary heart disease (CHD) among South African Indians is high. The aim of this study was to examine the presence and levels of risk factors for this disease among Indian adolescents, and to determine relationships between dietary intakes and levels of risk factors for CHD in this population group. Early detection of the presence of risk factors may lead to possible prevention of the development of CHD in individuals at risk.

A randomised homogeneous sample of 321 matriculants, selected from all matriculant volunteers who gave informed consent, from all six secondary schools in Lenasia, Transvaal, participated in the study. The study design used was that of a descriptive cross-sectional epidemiological survey.

Habitual dietary intakes were recorded using a validated food frequency questionnaire during individual home visits. Nutrient intakes were calculated with a computer programme based on the South African food tables. Special care was taken in the coding and analysis of the ingredients and cooking fats and oils used in the preparation of typical traditional Indian food dishes.

The main biochemical risk factors for CHD were determined. Serum and citrated plasma were prepared from venous blood samples taken between 08:00 and 10:00 from subjects who had fasted overnight, for the analysis

of serum lipid and lipoprotein concentrations (enzymatic colorimetic methods), plasma fibrinogen levels and factor VII coagulant activity (coagulation methods). Other variables such as body mass index, mid-upper-arm circumference and blood pressure were also measured with standardized methodology. Medical history of CHD and diabetes mellitus, smoking habits, physical activity levels and socio-economic background were assessed by means of a questionnaire. An analysis of variance, the Newman-Keuls Test, as well as Tukey's Test were calculated to obtain statistically significant differences of the measured variables between groups (males, females, vegetarians, non-vegetarians), using the SAS(R)-package. Pearson correlation coefficients and a step-wise regression analysis were calculated with the same package, in order to determine relationships between the various variables.

The results showed that the subjects had a high mean fat intake. Total fat provided more than 40% of total energy intake in both vegetarian and non-vegetarian subjects. The polyunsaturated:saturated fat ratio (0.8-1.0), however, approached the recommended dietary goal of 1.0, indicating liberal use of plant oils which are used for cooking. Dietary fibre intake was low (2.2-2.4 g/MJ).

Although the mean daily essential amino acid intake of all subjects was adequate for normal growth and development, the intake of vegetarians was significantly lower ($p < 0.05$) than that of non-vegetarians, probably due to the non-consumption of meat and meat products by vegetarians. The iron intake of all subjects was, however, adequate.

Mean calcium intakes of the non-vegetarian subjects were lower (but not significantly lower) than that of the vegetarians. All groups had a lower mean intake than the recommended dietary allowance of 1200 mg/day, especially the non-vegetarian females, who had a mean intake of 765 $\pm$ 371 mg/day. The magnesium, phosphorus, potassium, selenium, sodium, zinc and copper intakes of vegetarians were lower than that of non-vegetarians, significant for zinc and copper. The nicotinic acid (niacin), vitamin B_6, vitamin B_{12} and pantothenic acid intake of all vegetarians was significantly lower than that of non-vegetarians and also lower than the recommended dietary allowances for niacin, vitamin B_6 and B_{12}. The vitamin D, thiamin and riboflavin intake of vegetarians was lower (although not significantly) than that of non-vegetarians.

Using the South African age-specific cutpoints for serum cholesterol, it was found that a high percentage (51.7%) of the subjects had total cholesterol levels which placed them at moderate risk of CHD and a small percentage (5%) had levels at high risk for CHD. Almost half the subjects (49.9%) had fibrinogen levels greater than 250 mg/dl, an arbitrary cut-off point for an increased risk for CHD. Mean factor VII activity was in the normal range. More than one-fifth (21.2%) of the subjects were smokers. Physical activity levels were low.

From the results it was clear that strategies for lowering risk of CHD in this population group, should include early intervention in schools at primary and secondary levels. Anti-smoking campaigns should be initiated immediately. This should result in lower plasma fibrinogen levels.

Dietary advice could be given to reduce fat intake to the 'prudent' diet level of less than 30% of total energy intake, concentrating not only on food choices, but also on low fat and fat-free cooking methods. Advice on increasing dietary fibre intake, by eating more whole grain cereals, fresh fruit and vegetables should also be given. A lower fat and higher fibre intake should contribute to a lowering of total serum cholesterol levels. The large number of subjects with high total cholesterol and fibrinogen levels need to be advised individually on methods of lowering these values.

It was concluded that it would be advantageous to screen adolescents and to provide a follow-up service for those with high levels of CHD risk factors. The results of this study suggested that the dietary pattern of the Indian population group in South Africa possibly contributes to the high incidence of CHD in adult life.

OPSOMMING

Die voorkoms van koronêre hartvatsiekte (KHS) onder Suid-Afrikaanse Indiërs is hoog. Die doel van hierdie studie was om die teenwoordigheid en vlakke van die risikofaktore vir dié siekte onder Indiëradolessente te ondersoek, en om die verwantskap tussen dieetinnames en die vlakke van die risikofaktore vir KHS te bepaal. Vroeë opsporing van die teenwoordigheid van risikofaktore mag lei tot die moontlike voorkoming van die ontwikkeling van KHS in hoë-risiko individue.

'n Ewekansige, homogene steekproef van 321 matrikulante, geselekteer van vrywilligers wat ingeligde toestemming gegee het, van al ses sekondêre skole in Lenasia, Transvaal, het aan die studie deelgeneem. Die studie-ontwerp was dié van 'n beskrywende, deursnit-epidemiologiese opname.

Gebruiklike dieetinnames is met 'n getoetsde voedsel-frekwensievraelys tydens individuele tuisonderhoude gemeet. Nutriëntinnames is met 'n rekenaarprogram wat op die Suid-Afrikaanse voedseltabelle gebasseer is, ontleed. Spesiale voorsorg is getref om die bestandele, asook vette en olies gebruik in die voorbereiding van tradisionele Indiese disse, te kodeer en te analiseer.

Die belangrikste biochemiese risikofaktore vir KHS is bepaal. Serum en sitraatplasma is berei van veneuse bloedmonsters wat tussen 08:00 en 10:00 van die proefpersone wat oornag gevas het, verkry is. Hierdie monsters is gebruik vir die bepaling van serumlipiede en -lipoproteïene

(ensiematies-kolorimetriese metodes), asook plasmafibrinogeen en faktor VII - koagulantaktiwiteit (koagulasiemetodes). Ander veranderlikes soos liggaamsmassa-indeks, middelbo-armomtrek en bloeddruk is ook met gestandaardiseerde metodes gemeet. 'n Mediese geskiedenis van KHS en diabetes mellitus, rookgewoontes, fisiese aktiwiteitsvlakke en sosio-ekonomiese agtergrond is met behulp van 'n vraelys bepaal. 'n Variansie-analise, Newman-Keulstoets en Tukey se toets is bereken om statisties-betekenisvolle verskille van die gemete veranderlikes tussen groepe (seuns, dogters, vegetariërs, nie-vegetariërs) te bepaal, deur van die SAS(R)-pakket gebruik te maak. Pearson korrelasiekoëffisiënte en 'n stapsgewyse regressie-analise is met dieselfde pakket bereken, om die verwantskappe tussen die verskillende veranderlikes te bepaal.

Die resultate het getoon dat die proefpersone 'n hoë gemiddelde vetinname gehad het. Totale vet het meer as 40% van die totale energie in die dieet van beide vegetariese en nie-vegetariese proefpersone voorsien. Die poli-onversadigde: versadigde vetverhouding van 0.8-1.0 was egter naby die aanbevole verhouding van 1.0, wat die vrye gebruik van plantolies in die voorbereiding van voedsel weerspieël. Die inname van dieetvesel was laag (2.2-2.4 g/MJ). Die gemiddelde daaglikse inname van die essensiële aminosure van al die proefpersone was voldoende vir normale groei en ontwikkeling. Die inname van die vegetariërs was betekenisvol laer ($p < 0.05$) as dié van die nie-vegetariërs, waarskynlik as gevolg van die afwesigheid van vleis en vleisprodukte in hulle dieet. Die ysterinname van al die proefpersone was egter voldoende. Die gemiddelde kalsiuminname van die nie-vegetariese proefpersone was laer

(maar nie betekenisvol laer nie) as dié van die vegetariërs. Al die groepe het egter 'n laer gemiddelde inname as die aanbevole dieettoelae van 1200 mg/dag gehad, veral die nie-vegetariese dogters wat 'n gemiddelde inname van 765 $\pm$ 371 mg/dag gehad het. Die innames van nikotiensuur (niasien), vitamien B_6, B_{12} en pantoteensuur was betekenisvol laer by die vegetariërs as by die nie-vegetariërs, en ook laer as die aanbevole dieettoelae vir niasien, vitamien B_6 en B_{12}. Alhoewel vitamien D-,tiamien- en riboflavieninnames van die vegetariërs laer as dié van die nie-vegetariërs was, was hierdie verskille nie betekenisvol nie.

'n Vergelyking van die resultate met die Suid-Afrikaanse ouderdomspesifieke afsnypunte vir serumcholesterol, het getoon dat 'n hoë persentasie (51.7%) van die proefpersone totale cholesterolvlakke gehad het wat in die matige risikogrense vir KHS geval het, terwyl 'n klein persentasie (5%) se vlakke in die hoë risikogrens was. Ongeveer die helfte van die proefpersone (49.9%) se plasmafibrinogeenvlakke was groter as 250 mg/dl, 'n arbitrêre afsnypunt vir verhoogde risiko vir KHS. Gemiddelde waardes vir faktor VII-aktiwiteit was in die normale grense. Meer as 'n vyfde (21.2%) van die proefpersone het gerook. Fisiese aktiwiteitsvlakte was laag.

Uit die resultate het dit duidelik geblyk dat strategieë om die risiko vir KHS in hierdie bevolkingsgroep te verlaag, vroeë intervensie in primêre en sekondêre skole moet insluit. Teen-rookveldtogte behoort dadelik geimplementeer te word. Dit sal waarskynlik lei tot laer plasma-

fibrinogeenvlakke. Dieetadvies oor die verlaging van vetinname na die "omsigtige" vlak van minder as 30% van totale energieinname moet voorkeur geniet. Hierdie advies moet veral konsentreer op lae-vet voedselkeuses, maar ook op lae-vet en vetvrye gaarmaakmetodes. Advies oor 'n verhoogde dieetveselinname, deur meer volgrane en vars groente en vrugte te eet, behoort ook gegee te word. Dit mag bydra tot 'n verlaging van totale serumcholesterolvlakke. Die groot aantal proefpersone met verhoogde totale cholesterol- en fibrinogeen-vlakke behoort individuele raad te ontvang oor metodes om hierdie vlakke te verlaag.

Die gevolgtrekking is gemaak dat dit waarskynlik voordelig sal wees om adolessente te sif en 'n opvolgdiens te verskaf vir diegene wat verhoogde teenwoordigheid en vlakke van risikofaktore vir KHS het. Die resultate van die studie het gesuggereer dat die dieetpatroon van Suid-Afrikaanse Indiëradolessente moontlik mag bydra tot die hoë voorkoms van KHS in die volwasse lewe.

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CHAPTER 1

INTRODUCTION

Coronary heart disease (CHD), the leading cause of mortality and morbidity in industrial countries, has been found to be emerging as a public health problem in developing countries (WHO, 1982a). This multifactorial disease (Truswell, 1985) has been the subject therefore of many epidemiological surveys (Sarvotham and Berry, 1968; Miller et al., 1982; Lowry et al., 1983; Rossouw et al., 1983; Balleisen et al., 1985a, 1985b; Anon, 1986; Meade et al., 1986a; Derry et al., 1987; Steenkamp et al., 1988; Seedat et al., 1990). The disease has also been examined in many clinical studies (Lyon et al., 1956; Morrison, 1960; Hansen et al., 1962; Koranyi, 1963; Hood et al., 1965; Rose et al., 1965; Buzzard et al., 1982; Oh and Miller, 1985; Pyörälä, 1987). Experimental studies (Connor et al., 1961; Erickson et al., 1964; Hegsted et al., 1965; Keys et al., 1965a, 1965b; Dayton et al., 1969; Turpeinen et al., 1979) and several reviews (Levy, 1981; Oliver, 1982; Tunstall-Pedoe, 1982; Rossouw, 1983; Goldman and Cook, 1984; Stamler, 1985; Goldbourt and Neufeld, 1986; Gotto, 1989) have also contributed to our knowledge of CHD.

The term CHD is synonymous with ischaemic heart disease (IHD), defined by the WHO as "the cardiac disability, acute or chronic, arising from reduction or arrest of blood supply to the myocardium in association with disease processes in the coronary arterial system". The disease is

recognised through its common syndromes as neither the blood supply to the myocardium, nor the coronary arteries can be inspected in life without sophisticated investigations. Also, CHD usually implies occlusive atherosclerosis (atheroma plus thrombosis) as non-atheromatous coronary disease is comparatively rare.

The greatest challenge facing cardiology in westernized populations is the prevention of CHD. Generally, cardiologists have been reluctant to get involved in attempts to prevent CHD (Julian, 1986). In spite of substantial knowledge concerning prevention and control of CHD, it is still the major cause of premature disability and mortality (WHO, 1982b). Populations in which CHD is common are characterized by widespread and severe involvement with coronary atherosclerosis, often associated with thrombosis, resulting in sudden death. It may also manifest itself as an acute and often fatal attack of myocardial infarction (MI), as angina pectoris, congestive heart failure, or arrhythmias.

The primary risk factors of CHD are genetic as well as behavioural characteristics, eg. smoking and diet. The metabolic and cellular consequences of these primary risk factors directly influence the rate of progression of the disease, eg. abnormal lipoprotein and coagulation factor metabolism (Miller, 1983). It is appropriate to distinguish between modifiable (eg. diet) and immutable (eg. gender) risk factors in the context of preventive medicine. It must be recognized, however, that the term 'risk factors' implies only that these are factors associated with an increase in the risk of occurrence of the disease in a population

and that they have not been established as causative agents of the disease (Harper, 1983).

Diet is probably the fundamental environmental risk factor for CHD and atherosclerosis. Atherosclerosis takes years to develop. Coronary atherosclerosis probably develops from early childhood (WHO, 1982b). When symptoms occur in early adult life a familial predisposition may be suspected. The pathologic process is reversible if intervention is carried out before the formation of mature, advanced, atheromatous plaques. One of the characteristic materials that accumulates in atherosclerosis is cholesterol (Truswell, 1985).

Of the CHD predictors the link between diet, plasma lipids, and CHD is strong, although proof of a causal relationship may not be possible. Although the association between total plasma cholesterol and diet has been extensively studied, less is known about the dietary determinants of high density lipoprotein cholesterol (HDL-C), fibrinogen or factor VII levels, which have recently been shown to be predictors of CHD (Meade et al., 1986, Miller et al., 1986a). The suggestion that the ratio of HDL-C to total cholesterol may be a more useful predictor of CHD than total cholesterol is not universally accepted. Also, much controversy surrounds the relative contribution of two of the aetiologic components of CHD., i.e. the dietary fat/blood lipid and a thrombotic component (WHO, 1982a).

In the last three decades, atherosclerosis research has focused on the

cellular biology of atheroma, the metabolism of cholesterol and lipoproteins, the details of the lipid transport system and the apoproteins that are involved (reviewed by Schonfeld, 1983 and Gotto, 1989). The end-point in most epidemiological studies and clinical intervention trials is acute MI or death. It has been demonstrated in recent detailed investigations that the end-point is a thrombotic episode but no haemostatic factors were investigated in most of the major epidemiological or intervention trials, although at least two of the targets of intervention, smoking and plasma lipids, are known to influence haemostatic function (Smith, 1986).

Also, factors such as hyperlipidaemia, hypertension, smoking and diabetes mellitus are known to increase the risk of developing CHD at an early age, but it is not clear whether an aggregation of these risk factors alone may account for the early manifestations of CHD in certain families (Ibsen et al., 1982). People with familial increases in low-density lipoprotein cholesterol (LDL-C) (type II hyperlipidaemia) tend to have premature CHD, which is accelerated in homozygotes (Truswell, 1985).

Factors which have been associated with accelerated atherogenesis and which also occur with increased frequency in diabetes mellitus (DM) include hypertension, obesity and hyperlipoproteinaemia (Jialal et al., 1985). High prevalence of DM in migrant Indian population has been recorded in many studies (Omar et al., 1985). Statistics showing increased mortality from CHD and stroke associated with DM in England and Wales support the findings of large cohort studies (Fuller et al., 1983).

In adults the relationship between obesity and diabetes mellitus is well known and overt DM with adult onset as reflected by elevated blood glucose levels has been recognized as an independent risk factor for cardiovascular death (Berenson et al., 1982). In the Bogalusa Heart Study (Berenson et al., 1982), which is one of the major studies of cardiovascular risk factors in children, a close positive correlation was found between body fatness and insulin levels after glucose ingestion, which was independent of age. This relationship was strongest in the presence of high fasting levels of serum triglycerides or very low-density lipoprotein (VLDL). These observations may indicate early peripheral insulin resistance in obese children similar to that observed in obese adults with maturity-onset DM.

Coronary heart disease risk factors in children

Extensive studies conducted over the past two decades in large populations of children and young adults provide clues to the early origin of CHD (Berenson et al., 1980; Lauer and Shekelle, 1980; Berenson, 1986; Akerblom et al., 1991). Also, a special focus on tracking (which has been defined as the persistence of risk factor levels within a particular rank or percentile with regard to other children of the same age, race and gender) and determinants of blood pressure, lipids and lipoproteins provide insights for identification of many individuals who could develop CHD later in life (Berenson et al., 1992).

Several recent reports maintain that healthy children with elevated plasma cholesterol and LDL-C levels will prove to be adults with high levels and, consequently, at high risk of CHD (Dennison et al., 1989; Garcia and Moodie, 1989; Griffin et al., 1989; Garcia and Moodie, 1990). This prediction provides the merit for risk factor screening in children and young adults (Berenson et al., 1992). However, on the other hand, in a review of published studies by Newman et al. (1990) to estimate the potential risks and benefits of such screening it has been pointed out that childhood cholesterol levels are a poor predictor of high cholesterol levels in young adulthood and will be an even poorer predictor of CHD later in life.

Two studies have tracked cholesterol levels from childhood to adulthood (Orchard et al., 1983; Lauer et al., 1988) and other investigators have tracked lipid levels from childhood into the teenage years (Laskarzewski et al., 1979; Freedman et al., 1985). All these reported similar findings, namely, that more than half of the children who have cholesterol levels in the high range are unlikely to have cholesterol levels in the same range when they reach adulthood screening (Holtzman, 1991). Also there is no evidence that blood cholesterol levels can be lowered more easily in children than in adults, and it seems unlikely that cholesterol reduction in childhood will be more effective at preventing CHD than cholesterol reduction begun in middle age. It is stated that screening and interventions to lower blood cholesterol levels for millions of children would be expensive, could lead to labeling

and family conflicts, and may cause malnutrition and increased non-cardiovascular mortality (Newman et al., 1990).

Diet is considered a major environmental determinant of cardiovascular risk factors, but whether it has a greater impact on serum lipid levels in children than in adults, awaits further study (Berenson et al., 1982).

In the Bogalusa Heart Study the high caloric intake that occurred during the first year of life was an indication of the alimentary origin of obesity that may begin in infancy. High weight for height values were found in those with high blood pressure (BP) levels. It was also observed that adolescents or young adults with high BP ranking tended to become hypertensive later in life.

The life-style and behavioural correlates of change in the young adult offspring of the Framingham Heart Study cohort also offer prospective evidence which suggests that habits formed during young adulthood could have a substantial effect on lipid and lipoprotein profiles in men and women (Hubert et al., 1987).

According to recent and ongoing investigations into the role of clotting factors in CHD, it has been recommended that fibrinogen should be rated among the major risk factors for CHD, comparable in importance to smoking, high BP and high cholesterol levels (Meade et al., 1986b; Sgammato, 1987). Also, factor VII clotting activity, like blood cholesterol levels, increases with dietary fat intake (Miller et al., 1985; Miller

et al., 1986a) and studies of the fluid phase of coagulation have suggested the existence of a hypercoagulable state in hyperglycaemic subjects (Jones and Peterson, 1981).

Although prospective studies have been inconsistent in finding an association between obesity and CHD; android (central) obesity (in westernized populations) (McKeigue, 1990), is consistently associated with hyperinsulinaemia (Jarrett, 1987). It has also been supposed that lipid abnormalities and high blood pressure increase the tendency toward atherosclerosis through injury to the vessel wall and lipid deposition, but less is known about the possible interplay between these risk factors and the thrombotic process, which in turn may play a role in the development of the atherosclerotic plaque (Wilhelmsen et al., 1984).

The Problem

Racial differences in both incidence of CHD and its risk factors are known to occur (Lowry et al., 1983; Holtzman, 1991). In South Africa, CHD is the chief killer in the white and Indian, and to a lesser extent, in the coloured (persons of mixed race) populations (Walker, 1980). The age-standardized mortality rates from CHD (mean annual rate, 1978-1982) was 688 per 100 000 for whites, 780 per 100 000 for Indians, and 419 per 100 000 for coloureds, (deaths below 25 years of age excluded) (Derry et al., 1987). According to Miller et al., (1982), McKeigue et al., (1985) and Walker (1987), the rate of CHD among the Indian populations that have migrated from the Indian subcontinent to other parts of the world

(Central, East and South Africa, Singapore, Fiji, West Indies, and the United Kingdom), often exceeds that in the host countries. South African Indian males appear to have risk factors for CHD which could be in a more "severe" form than other population groups (Seedat et al., 1978; Seedat et al., 1990). It is unlikely that there is a single explanation for the high CHD mortality of all overseas Indian populations (Anon, 1986). However, many factors predispose to CHD and the contributions to risk in Indians and their compatriots may differ from country to country. Also, more important than calculation of relative risk is an understanding of the pathological processes which predispose Indians to CHD, and only then can effective preventive measures be introduced that might reverse the present unacceptable situation (Hughes et al., 1989).

It has been suggested that the diet of migrant Indians differs from the average diet in India and that this altered dietary habit contributes to their increased risk from CHD (Raheja, 1986). The average diet in India is essentially a low-fat, lactovegetarian diet. Milk and yoghurt mainly provide animal proteins, and the main source of fat is usually refined or hydrogenated vegetable oils. This diet is low in total and saturated fat, cholesterol and sucrose, but rich in potassium, magnesium, complex carbohydrates and dietary fibre (Raheja, 1986).

South African Indians cook their food in the traditional way, but "ghee" (clarified butter) is used to prepare most curries, which could increase fat intake and expose Indians to intakes of cholesterol oxides (Jacobson,

1987). Vegetarians here are also exposed to a high fat intake and cholesterol oxides. The non-vegetarians consume meat, eggs and dairy products, i.e. increased animal proteins and fats and decreased pulses, legumes and vegetables. The low-risk Indian diet could therefore become a high-risk atherosclerotic diet.

Little is known about the nutrient intakes of South African Indians. Mia (1983) reported nutrient intakes of adults aged 20-50 years. In that study, in order to measure fat intake correctly, special care was taken in the coding of traditional dishes as Indian housewives fry many foods in fats and oils.

Purpose of the study

The purpose of this study was to carry out a survey of the dietary patterns and nutrient intakes of Indian adolescents in Lenasia, Transvaal, in order to evaluate to what extent or how these could possibly contribute to CHD risk.

Specific objectives of the study were therefore to measure CHD risk factors and to examine the associations between these risk factors (serum total cholesterol, HDL-C, LDL-C, plasma fibrinogen and factor VII levels, BP and body mass index (BMI)) and habitual nutrient intakes in a randomized sample of Indian matriculants. In addition to measurements of nutrient intakes, serum lipids and plasma coagulation factors, anthropometric measurements were carried out, blood pressure measurements were

made by standard methods and a coronory heart disease risk factor questionnaire was completed for each subject. The correlation of this data could provide information about the impact of dietary intakes on CHD risk factor levels of adolescents. This information may be used in dietary recommendations to South African Indians.

Structure of the thesis

The relevant literature is reviewed in Chapter 2. Special attention is given to the history of CHD, the underlying atherosclerotic mechanisms and the various risk factors associated with this multifactorial disease. Emphasis is placed throughout on the situation in the Indian population - locally and abroad. To assist in the understanding of the Indian situation, a detailed review of the origin of South African Indian sub-groups is given in Appendix A, and the traditional Indian cuisine in Appendix B. This chapter is concluded with a review on the possible prevention of CHD.

The design, subjects, materials and methods used in this study are given in Chapter 3, (with appropriate Appendices C-H), the results in Chapter 4 and the discussion of these results in Chapter 5.

Conclusions drawn, and recommendations based on the salient observations, are made in Chapter 6.

CHAPTER 2

LITERATURE SURVEY

2.1 The History of Coronary Heart Disease

As has been reviewed by Walker (1987) CHD has been described as a disease of antiquity and has been verified by autopsies on Egyptian mummies from the XXI Egyptian Dynasty (about 1 000 B.C.) (Lie, 1978; Levy, 1981). Although the natural history of CHD was delineated only relatively recently, anginal pain and sudden death were known millenia ago (Levy, 1981). During the post-Vedic period (about 800 B.C.) in India, the ancient physicians, Charaka and Sushruta, wrote treatises about the circulation of blood. The Vedas described the heart as "a lotus with nine gates" (Mukerjee, 1975). The role of stress and strain and indiscretions of diet (today known as risk factors) in the pathogenesis of heart disease were explained by Sushruta at that stage already (Sen Gupta and Sengupta, 1859), and anginal pain was described as a "sense of constriction in the precordium, stitching, pain and sensations of churning or bursting or rubbing". Up until the 13th century A.D., various additions were made to these original observations, when Madhaba, Baba Mishra and Chakrapani and others wrote revised treatises on Ayurvedic medicine (Mukerjee, 1975).

Modern cardiology was conceived only after post-mortem examination was allowed by the Christian church early in the 16th century when important

discoveries in relation to the physiology and pathology of the cardiovascular system were made.

The Hippocratic School in Greece recognized angina pectoris and apoplexy (relating symptoms to advancing age), and possibly cerebal arteriosclerosis (Brothwell and Sandison, 1967), while the Roman philosopher, Seneca (4 B.C. to A.D. 65), recorded the anginal syndrome in recounting his own illness. Walker (1987) mentions the following in his review: "William Harvey (1719-1801) first recognized the existence of the third circulation, the coronary circulation (Lie, 1978). William Heberden (1719-1801) became known for his lecture on angina pectoris, while in 1707, the brilliant Italian physician, Lancisi, and in 1761, Morgagni, the anatomical pathologist, described cases of patients dying from CHD. Before the end of the 18th century, angina pectoris was related to occlusive disease of the coronary arteries both by Jenner (of smallpox fame) and Parry who was the first to recognize and describe exophthalmic goitre. In 1880, Weigert recognized the role of coronary atherosclerosis in myocardial infarction and the different effects of myocardial ischaemia, while in 1887 Ziegler published a clear description of the gross and histologic features of myocardial infarction from the onset to healing".

However, it was only in 1912 that the classic article of Herrick on coronary artery obstruction and myocardial infarction, finally attracted the full attention of the medical profession. Up until 1920, as stated in Osler's "Principles and Practice of Medicine" (McCrae, 1912), angina

pectoris was rated as a rare disease in hospitals, with an average of one case per year. Since the 1920s and for almost half a century (Tunstall-Pedoe, 1982), there has been a marked rise in the occurrence of CHD and in many Western populations the incidence rose to cause a third or more of all deaths.

In 1979, CHD ranked first among all diseases in terms of Social Security disability benefits in the United States and the cost was conservatively estimated to be over 27 billion dollars each year for direct medical care (National Heart, Lung and Blood Institute, 1981). It also became apparent that CHD mortality rates were high in the more prosperous countries and relatively low in the less technically advanced countries. However, CHD is commoner in poorer people in affluent countries (Walker, 1987).

2.2 *The Decline in Coronary Heart Disease Mortality Rates*

Marked decreases in CHD mortality rates occurred in all age and sex groups in both blacks and whites in the United States between 1968 and 1976 and between 1976 and 1980 (Levy, 1981; Goldman and Cook, 1984). Comparsion of mortality rates in various countries shows that during the 1970s, the United States experienced the most marked decline in CHD mortality among the industrialized countries, even in comparison to other countries with CHD mortality rates similar to those of the United States in 1968, such as Australia and Finland (World Health Organization,

1972-1979) as well as New Zealand (Uemera and Pisa, 1988). In Britain rates began falling after 1973-74, especially in the 35-44 year age group, and occurred in both men and women in each of the regions of England, Wales and Scotland (Heller et al., 1983). In Japan mortality from CHD began to decline in 1970 although their rate was already far lower than that of the United States and the United Kingdom (Ueshima et al., 1987).

The reasons for the decline in mortality rates have been ascribed by many to the effects of a reduction in the prevalence and severity of risk factors (Smith, 1982; Notes and News, 1982; Turner, 1982; Ball, 1983; Heller et al., 1983), which include changes in lifestyle such as decrease in cigarette smoking, blood pressure control, dietary modification, and an increase in physical activity (Levy, 1981), and to medical intervention in the form of coronary care units, improved emergency medical services, cardiopulmonary resuscitation and advances in surgical and medical treatment (Levy, 1981; Walker, 1987). However, in spite of the steady decline in CHD mortality rates in some countries, it is not known what has occurred with CHD morbidity, although it is known that fewer heart attacks occur. Also, it is difficult to determine whether the major credit for the decline should be attributed to improved medical and surgical treatment of existing disease or to the prevention of new disease.

2.3 Coronary Heart Disease in South Africa with Special Reference to the Indian Population

2.3.1 Comparison of South African population groups

In 1970 the age-adjusted CHD mortality rates in South Africa were 247 per 100 000 for white males and 73 per 100 000 for white females, 274 per 100 000 for Asian (mainly Indian) males and 119 per 100 000 for females, 125 per 100 000 for coloured males and 64 per 100 000 for females, and 13 per 100 000 for black males and 12 per 100 000 for females (Wyndham, 1982a; Walker, 1987). The age-standardized mortality rates from CHD (mean annual rate, 1978-1982) was 688 per 100 000 for white, 780 per 100 000 for Indians, and 419 per 100 000 for coloureds, (deaths below 25 years of age excluded) (Derry et al., 1987).

As with other Western populations, up to the 1930s, CHD among South African whites was rare and subsequently became exceedingly common (Walker, 1987). Rates have been reported to be higher for Afrikaans-speaking than for English-speaking societies in Johannesburg (Jenkins et al., 1980) while the rate for the Jewish segment (based on death certification) was reported to be the highest of all (Walker, 1980; Walker et al., 1980a).

For coloureds, CHD mortality rates were low, but increased to reach about half the rates of whites (Wyndham, 1979; Walker, 1980; Wyndham, 1982b; Steyn et al., 1985). Some reports stated that the rate appeared to be

higher in Cape Town than in Durban (Annual Report, 1979; Annual Report, 1980).

Among blacks CHD remains very uncommon and is extremely rare in rural areas. Reports from 1954 to 1980 indicated that coronary artery occlusion was rare among blacks (Higginson and Pepler, 1954; Reef and Isaacson, 1962). In Durban the rarity of myocardial infarction among blacks has been confirmed (Seedat et al., 1976; Chesler et al., 1978; Thandroyen et al., 1980).

In other parts of Africa, Nigeria, Ivory Coast, Ghana, Zaire, Zambia, Botswana and other African countries, CHD is extremely rare, if not entirely absent. In the United States of America (USA), CHD was the same for blacks and whites (Weisse et al., 1971). On the other hand, it is not understood why, in the USA, obese black women with hypertension, high cholesterol levels, and diabetes have such low incidences of myocardial infarction and sudden death from heart disease, as well as having minimal amounts of coronary artherosclerosis on post-mortem examination (Kuller et al., 1975).

The disparity between the frequency of CHD in blacks and whites was found to be even wider in Birmingham, England (Cruickshank et al., 1980), while in the East End of London in 1973, Caribbean immigrant blacks had "one-tenth of the average attack rate" (Tunstall-Pedoe et al., 1975).

2.3.2 Coronary heart disease among South African Indians

Several reviews (Wyndham, 1979; Walker, 1980; Wyndham, 1982b) pointed out that there has been a steady increase in the CHD mortality rate for South African Indians. These authors mention that at the King Edward VIII Hospital in Durban, coronary thrombosis was relatively common in the 1950's, although it formed only 0,5% of adult admissions (Cosnett, 1957). A generation later, CHD accounted for 11% of patients between 1970-1980 at the R.K. Khan Hospital in Durban. Of these patients, 22% were aged under 45 years and 68% were aged 46 to 70 years. The mortality rate of Indians from CHD was found to be higher than that of whites (Wyndham, 1979; Walker, 1980; Wyndham, 1982b). It has also been noted that the peak mortality from CHD occurs a decade earlier than in whites (Walker, 1975).

Although reports of the Durban City Health Department showed that mortality rates have been stable in the last few years, a decline of 27.5% (from 583 to 422 per 100,000) for Indians was observed between 1970 and 1985 (Steinberg et al., 1988). Rates declined among both males and females as well as in all the age groups studied.

2.3.3 Coronary heart disease in India

CHD is common in India, especially in the merchant and professional classes as well as among total vegetarian, low-fat eating, non-smoking, non-hypertensive, non-obese labourers (Murphy & Gemson, 1980). Even in

rural India, where about 90% of the population lives, CHD is far from rare (Walker, 1987).

A survey in Punjab, India, of adults from several states in northern India found major Q waves in 4% of men in all age groups between 40 and 70 years (Sarvotham and Berry, 1968). This frequency was less than half that among Indian men of comparable age in the Trinidad study (Miller et al., 1982).

2.3.4 Coronary heart disease in Indians overseas (abroad)

In 1979 it was estimated that more than 11 million people of Indian descent were dispersed throughout the world (Bahadur-Singh, 1979). Their migration dates essentially from the introduction of the colonial indentured-labour system which founded a large number of expatriate communities after 1830 (Appendix A), and thousands more emigrated as entrepeneurs and professionals. More than 7 000 men and women settled in Mauritius between 1834-1837, and between 1844 and 1917 many thousands arrived in the West Indies, mainly in Trinidad and Guyana (Miller et al., 1982). Whether they have settled in Africa (Cosnett, 1957; Shaper and Jones, 1959; Walker, 1963), Europe, (Tunstall-Pedoe et al., 1975), the Americas (Wattley, 1959; Ramdial and Poon-King, 1973), or other parts of Asia (Danaraj et al., 1959), the rate of CHD is much higher in Indians than in their compatriots of other ethnic descent. This susceptibility has been thought to be due possibly to the high prevalence of diabetes mellitus (Campbell, 1963; Poon-King et al., 1969), or to high serum cholesterol concentrations (Shaper and Jones, 1959).

In Britain, however, the Indian population is largely of more recent origin. Approximately 60% are first generation immigrants who arrived after 1960 (Anonymous, 1986), and who number about 1.3 million, of whom 40% are aged 30 years or more (Office of Population Censuses and Surveys, 1984). The early indications of their propensity to CHD came from countries with long-established populations, ie. from Singapore, Fiji, Natal, Uganda and Trinidad (Anonymous, 1986). This has been emphasized in a report prepared in 1986 by the Coronary Prevention Group for the Confederation of Indian Organization in Britain. The standardized mortality ratio per 100 000 for CHD at age 20-69 years was 119 in men of Indian descent in England and Wales and 128 in women during 1970-1972 (Marmot et al., 1984).

2.3.5 Coronary heart disease risk factors of Indians in other parts of the world

A detailed discussion of the nature of risk factors is given in section 2.5. It has been stated that there are differences in risk factors in Indians compared with whites (Beevers and Cruickshank, 1981; Eisenberg et al., 1981). In Birmingham, England, blind assessment of coronary arteriograms demonstrated significantly more severe disease in Asians with coronary artery disease (CAD) compared to whites with CAD, but the distribution of the disease was similar in both groups (Lowry et al., 1983). An assessment of risk factors showed that the Asian group had significantly more non-smokers, lower cholesterol level and weighed less than whites. This Asian group originated mainly from Pakistan, Punjab,

and a few from Bangladesh. The South African Indians originate from other parts of India (Appendix A).

Also, in the United Kingdom, the high CHD rates among Indians could not be explained by the dietary and other risk factors measured in a study by Mc Keigue et al., 1985. However, non-insulin dependent diabetes mellitus was not mentioned in this study.

A similar study was carried out in Port of Spain, Trinidad (Miller et al., 1982). Prevalence of CHD and fasting serum lipoprotein concentrations in ethnic groups were compared. The Indians, who originated from North India (Jha, 1974) had lower HDL-C concentrations and significantly higher LDL-C concentrations than other groups. After allowance for age and ethnic group, men with major Q waves or a history of positive myocardial infarction had a significantly greater ratio of LDL-C/HDL-C than men without either.

2.3.6 Coronary heart disease risk factors in the South African Indian population

In South Africa, the "epidemic" of three cardiovascular diseases, ie. CHD, cerebrovascular disease (stroke) and hypertensive heart disease, is of more serious proportions in Indians than in whites (Wyndham, 1979). Also, the life expectancy of South African Indians, beyond the age of 50 years, was less than that of Indians in India, in spite of enjoying better economic standards (Walker, 1973).

Most studies on myocardial infarction at young age have shown a high incidence of risk factors (Walker and Gregoratus, 1967; Glover et al., 1982; Kennelly, 1982). The presence of multiple risk factors has been demonstrated in young Indian men with myocardial infarction (mean age 36 years; range 21-40 years) (Sewdarsen et al., 1987). In this study cigarette smoking was the most important risk factor, and a notable difference between this and other studies was the higher incidence of diabetes and hypertension in young Indian men with myocardial infarction; 50% of the patients had hypercholesterolaemia and 53% hypertriglyceridaemia; mean HDL cholesterol levels were below 0,83 mmol/l; 47% had a family history of CHD.

In a recent study carried out among Durban Indians aged 15-69 years to determine the prevalence of risk factors of CHD (Seedat et al., 1990), the important risk factors in men were hypercholesterolaemia, hypertriglyceridaemia, diabetes and smoking, and in women, diabetes, hypercholesterolaemia, and hypertriglyceridaemia. Minor risk factors were hyperuricaemia, sedentary occupation, obesity in women and a positive family history of CHD. This study revealed that 52% (sex- and age-adjusted 45,5%) had at least one major risk factor at the higher (level A) and 68% (sex- and age-adjusted 61,9%) at the lower (level B) risk levels. Diabetes was strongly associated with a positive history of CHD.

However, the results of the risk factor prevalence study carried out in Durban (Seedat et al., 1990) probably cannot be extended to the

population of Lenasia as there are differences in socio-economic status and religious composition due to their region of origin in India and their reason for emigrating to South Africa, as is discussed in Appendix A. It is possible that these factors could for example influence eating patterns and nutrient intakes.

In Delhi, India, in a study carried out among industrial and rural male workers aged 30-39 years, the mean serum cholesterol concentration was 4.5 mmol/l compared with 5.8 mmol/l for Indian men aged 35-44 years in Port of Spain, Trinidad. Serum cholesterol concentrations and CHD prevalence were also higher in Indians than in the indigenous Africans or in Indians in north India (Shaper and Jones, 1959).

However, these studies were carried out more than thirty years ago. The prevalence of CHD should be assessed in present times as well as the levels and the interaction of CHD risk factors as there seems to be no specific risk factor or risk factor level that would predict the cause of the excessive CHD in overseas Indian immigrants. The reason for the trends in risk factor levels and occurrence of the disease at a younger age compared with other ethnic groups, should be examined.

2.4 Atherosclerosis

2.4.1 Introduction

CHD is the most important clinical expression of atherosclerosis

(Bronte-Stewart, 1958). Atherosclerotic disease is characterized by a focal intimal thickening of medium and large-sized arteries (Nilsson, 1986). The mechanisms leading to the initiation of atherosclerosis are very complex and have not been fully clarified (Copley, 1979). This disease begins early in life but rarely produces symptoms until middle age and often goes undetected until the time of the first heart attack, which is often fatal (Consensus Conference - Blood Cholesterol, 1985). Although atherosclerosis occurs universally in man, there are differences in the extent and severity of lesions between different members of the same community and marked differences in prevalence of significant lesions between different communities (McGill, 1968).

2.4.2 Pathogenesis of atherosclerosis

There are two major hypotheses for the pathogenesis of atherosclerosis. The "incrustation" theory of Rokitansky (Rokitansky, 1852), which was modified by Duguid and Robertson (Duguid and Robertson, 1957), suggests that intimal thickening results from fibrin deposition with subsequent organization by fibroblasts and secondary accumulation of lipids. More recently, the "response to injury" hypothesis (Ross and Glomset, 1973) suggests that endothelial cell injury with associated deposition of platelets can also initiate the intimal proliferation.

The second hypothesis, the "imbibition" hypothesis (Virchow, 1858 translated in 1971) was modified to become the lipid or filtration hypothesis which depends on the theory that lipids in the arterial wall represent a

transudation of blood lipids, which subsequently form complexes with acid mucopolysaccharides and that the arterial accumulation occurs because the mechanism for removing lipids could be overwhelmed (Ross and Harker, 1976; Small, 1977). The role and positive association of raised serum (plasma) cholesterol, and especially the fraction transported by the low-density liporoteins (LDL) in atherogenesis is generally accepted and no longer seriously debated. Raised circulating LDL-C level does not, however, explain all facets of this multifactorial disease, as the risk associated with a given lipid value is modulated by the level or presence of other risk factors (Tunstall-Pedoe and Smith, 1990).

At present, a single, more comprehensive hypothesis integrating these two hypotheses states that the pathogenesis of atherosclerosis appears to depend on a precise sequence of critical events based on the interaction of blood elements and lipids with the arterial wall, and, in turn, each of these may be modified by different risk factors (Fuster, 1982). A third theoretical approach, first proposed by Rindfleisch in 1872, concerns haemodynamical or/and haemorheological aspects which were further developed by Fry (1969), Caro et al. (1969, 1971), Caro (1973), Stehbens (1974) and others. A new theory of the pathogenesis of atherosclerosis was proposed by Copley (1979) which consisted mainly of three stages, ie. an initial or early stage, a mid-stage, and a late stage. According to Copley (1979) fibrin and fibrinogen have dominant roles in this theory.

Mechanisms of action

It has been stated that no single theory of atherogenesis can explain in detail all of the known risk factors which predispose to atherosclerotic vascular disease (Kadish, 1979). One view is that atherogenesis requires reduced fibrinolytic activity as a prerequisite for plaque formation. In patients with atherosclerosis, a number of alterations have been observed in haemostatic parameters (Ulutin, 1986). An increase in coagulation factors and their activities, a decrease in the natural inhibitors of coagulation, alterations in the biochemistry and function of platelets, a decrease in endothelial cell functions, changes in the monocyte-macrophage system, hypertrophy, migration of smooth muscle cells in the intima, and alterations in lipid metabolism have been described. Similar results were found in experimental animal models with atherosclerosis (reviewed by Ulutin, 1986).

It has been hypothesized that deposition of fibrin on the endothelium occurs as an integral part of the atherogenic process (Pilgeram, 1960). Fibrin has been shown to possess high affinity for lipids (O'Neal et al., 1959), thus providing a biochemical basis for deposition of lipids in the fibrin plaque. Also, increased conversion of clotting factor VIIc (factor VII concentration) to factor VIIa (activated factor VII) in plasma samples of healthy adults has recently been related to high plasma lipid concentrations (Miller et al., 1985).

2.5 Coronary Heart Disease Risk Factors

2.5.1 Introduction

The Framingham Heart Study (Kannel et al., 1967) demonstrated that certain variables (clinical findings) now called risk factors, are powerful predictors of future likelihood of developing CHD (Podell, 1983). Among the most important are cigarette smoking, hyperlipoproteinaemia, hyperlipidaemia, hypertension, diabetes mellitus, genetic factors (or family history of atherosclerotic disease) (Fuster, 1982; Mitchell, 1985). Social class is also an extremely powerful risk-marker Black et al., 1982; Pocock, et al., 1987). The influence of social class is complex and varies in different populations. It has been stated that CHD is a class-based disease rather than one based on race (Dreyer, 1984); in South Africa the white section of the population generally occupies a higher class position than the black population and is therefore more exposed to the causative factors of CHD.

The prevalence of several risk factors is influenced by socio-economic status. Regarding the situation in South Africa with its different ethnic populations with varying socio-economic status, it has been mentioned by James (1984) that for a given population the direction of the association between CHD and socio-economic status is time-dependent.

Early in the process of urbanization and industralization, persons of higher socio-economic status are at increased risk, whereas towards the

end of the process, lower socio-economic status persons are at increased risk (James, 1984). This may possibly explain the decline in CHD in some population groups in the Republic of South Africa and the rise in others as reported by Derry et al. (1987).

However, the predictability of CHD from risk factors poses a major limitation (Heller et al., 1984; Walker and Walker, 1985) as inaccuracies could occur through missed or misdiagnosed cases, through candidates waylaid or eliminated by other diseases, and through slow starters. It is also possible that not all risk factors are known, or measured. A staggering number of 246 factors which could play a role in the development of CHD has been identified (Hopkins and Williams, 1981).

Over 30 years of extensive epidemiological findings, consistent with clinical, pathological and animal experimental data, have identified at least four major or primary risk factors which occur with a high degree of frequency in individuals who have CHD (Winston, 1982). These are high plasma cholesterol, particularly elevated LDL-C, hypertension, cigarette smoking and diabetes. Based on recent prospective epidemiological studies (Wilhelmsen et al., 1984; Stone and Thorp, 1985; Meade et al., 1986a, 1986b; Kannel et al., 1987a, 1987b), hyperfibrinogenaemia and raised levels and activity of clotting factor VII should be added to this list. Secondary risk factors are high blood triglycerides (Assmann, 1990; Gianturco and Bradley, 1990; Shepherd, 1990), low plasma HDL (Covas and Prats, 1987; Galton, 1990; Ginsberg et al., 1990), obesity (Krotkiewski et al., 1983; Larsson et al., 1984; Waaler, 1984; Covas

and Prats (1987), stress, personality type and lack of exercise. Male sex, age, and family history of CHD are also implicated (Kannel et al., 1976). Epidemiological research (Keys et al., 1956; Morris et al., 1963; Kahn et al., 1969; Gordon, 1970) also points to a relationship between nutrition and cardiovascular diseases.

2.5.2 Coronary heart disease risk factors in children and adolescents

Studies of CHD risk factors in children began in the 1970's (Williams and Wynder, 1992). Independent studies in the United States (Kannel and Dawber, 1972; Drash, 1972; Haas, 1973; Williams and Wynder, 1976) and other countries began to provide a database for CHD risk factor levels in children by age, gender and nationality. The results of these studies suggested that CHD risk factor levels in children may parallel those in adults as well as reflect CHD mortality trends in their respective nations.

In a review (Martin, 1992) of data published during the last 15 years in Europe, regional and intra-country differences were found in the levels of total serum cholesterol, blood pressure, smoking habits, body weight, physical activity in leisure time and insulin dependent diabetes mellitus (IDDM) in children and adolescents.

However, it has been stated that the observed differences between independent studies might, at least partly, be caused by differences in methodology (Martin, 1992; Williams and Wynder, 1992). Thus, the most

appropriate way to collect comparable data about CHD risk factor levels among children and adolescents, even in Europe, would be a multicentre study where more attention could be paid to factors such as the use of a common protocol, the standardization of methods and survey procedure, validation of questionnaires, the quality control issue, including training and evaluation of observers, and common data analysis. Also, for adolescents, an index of physiological development, more precise than chronological age, should be included in comparative epidemiological studies, since biological risk factors such as cholesterol and blood pressure are closely related to growth and sexual maturation (Berenson et al., 1992). It has been stated that such a project would be a desirable complement to studies such as the WHO Monica Project which is monitoring CHD risk factors of 38 adult populations around the world (Tunstall-Pedoe, 1985; WHO Monica Project Principal Investigators, 1988). This would provide more conclusive data enabling researchers and health authorities to determine the actual needs for preventive activities and to assess the efficiency and effectiveness of ongoing prevention programmes.

The results of a study carried out in Hungary (Mihai and Jansco, 1991) suggest a significant association between parents' and children's fibrinogen levels. This study suggests that fibrinogen level is a definitive risk factor for identifying children at high risk for CHD.

Geographical studies (Forsdahl, 1977; Barker and Osmond, 1986; Barker et al., 1989a) from Norway, England and Wales, provide evidence for the hypothesis that the long-term effects of an adverse environment in foetal

life and infancy may be determinants of CHD in humans (Barker, 1992). There is also evidence that haemostatic variables, i.e. fibrinogen and factor VII (Barker et al., 1991), glucose tolerance (Hale et al., 1991), blood pressure (Folkow, 1982; Barker et al., 1989b; Thornberg, 1991) and lipid metabolism (Coates et al., 1983; Mott et al., 1991) are all susceptible to the programming effects of the environment in early life (Barker, 1990; Barker et al., 1990; Barker, 1991; Hale et al., 1991).

2.5.3 Hypercholesterolaemia as a risk factor for coronary heart disease

Introduction

Definition of hypercholesterolaemia

Two conflicting views have been put forward for the definition of an elevated plasma cholesterol level (Grundy, 1986). According to one view, hypercholesterolaemia should be defined as a cholesterol level above the 95th percentile for a given population while the alternate approach is to define it as a cholesterol concentration associated with significantly increased risk of CHD. This latter approach relates the plasma cholesterol level to clinical risk rather than merely to a population distribution. However, the graded relation between plasma cholesterol level and CHD results in the problem of defining what is a significant increase in risk. Despite this problem the latter approach was adopted at the consensus conference on cholesterol (Anonymous, 1985).

Causes of hypercholesterolaemia

The most dramatic form of primary hypercholesterolaemia occurs because of abnormalities in the gene encoding for LDL receptors (reviewed by Grundy, 1986). This condtition is responsible for less than 2% of all cases of plasma cholesterol levels over 240 mg/dl (6.21 mmol/l). The causes of hypercholesterolaemia in the remaining 98% of cases can be either genetic or dietary or a combination of both. Several genetic factors, besides those involving the primary genes encoding for receptors, could reduce the LDL receptors, or, alternatively, transport mechanisms for LDL receptors within cells might be defective, so that fewer receptors would reach the cell surface.

One study (Grundy and Vega, 1985) has shown that many patients with primary hypercholesterolaemia, other than those with familial hypercholesterolaemia (FH) have a decrease in fractional catabolic rates of LDL, which is consistent with reduced activity of LDL receptors. Thus, a decrease in the number of functioning LDL receptors is almost certainly not limited to FH, and is probably a major cause of other forms of hypercholesterolaemia. Secondary hypercholesterolaemia may be due to other conditions, e.g. hypothyroidism.

Individuals affected with FH can be heterozygous or homozygous. Heterozygosity is associated with reduced cellular LDL receptors which leads to premature manifestations of atherosclerosis early in middle life, while homozygotes have either no, or genetically defect, LDL receptors.

Affected individuals get severe atherosclerosis in adolescence. Thus, severe CHD can result from high blood cholesterol levels in the absence of any other contributory risk factors (Consensus Conference, 1985).

Diet is probably a significant factor for a large number of cases of primary hypercholesterolaemia. It is stated that some people may be overly sensitive to excess dietary cholesterol, saturated fatty acids, or total calories and may respond with an unusually marked increase in their LDL levels (Grundy and Vega, 1988).

2.5.3.1 Total blood cholesterol

Early in this century Ignatowsky fortuitoùsly induced atherosclerosis in rabbits by feeding a diet of milk, meat and eggs (Ignatowsky, 1909). Anitschow subsequently argued that the cholesterol in the animals' rations was the main culprit (Hermus, 1979). The fascination with the cholesterol molecule has resulted in 13 Nobel Prizes being awarded to scientists who researched this molecule.

The evidence that hypercholesterolaemia is a major risk factor for CHD, comes from more than two score prospective epidemiologic studies in many countries throughout the world (McGill, 1968; Pooling Project Research Group, 1978; Keys, 1980), from clinical investigations such as angiographic studies (Miller et al., 1981) and from post-mortem investigations (Garcia-Palmieri et al., 1977). In addition, powerful concordant evidence is available from animal experimental research in several species,

including non-human primates (reviewed by Lewis, 1980). The relationship between diet, especially dietary fat intake and blood cholesterol levels, is supported by many studies. A curvilinear relationship between TC or LDL-C and subsequent risk of CHD was confirmed in the report on over 350 000 men screened for the Multiple Risk Factor Intervention Trial Study (Stamler et al., 1986).

Cholesterol has been described as an essential yet potentially dangerous molecule (Rifkind and Lenfant, 1986). The very property of cholesterol that makes it essential in cell membranes, namely, its absolute insolubility in water, also makes it potentially lethal (Brown and Goldstein, 1986). When it accumulates in the arterial wall it cannot be readily mobilized, and its presence eventually leads to the development of an atherosclerotic plaque and its unfortunate sequelae.

For cholesterol to be transported safely in the blood, its concentration must be kept sufficiently low and its tendency to be deposited in the arterial wall must be controlled. Much information has accumulated in recent years of the complex relationship that exists between the major lipoprotein classes and mechanisms which control their concentration in plasma (Nye and Sutherland, 1984).

It seems that diet is a major determinant of blood cholesterol.

The proportion of energy intake derived from saturated fat plays an important role in differences in blood cholesterol among populations

(Keys, 1980). The concentrations of total cholesterol and cholesterol in the various lipoprotein fractions are also influenced by diet. Total and LDL-C concentrations are higher in meat eaters than in vegans, with vegetarians and fish-eaters having intermediate and similar values (Thorogood et al., 1987). These authors also showed that HDL-C concentration was highest in fish eaters but did not differ among vegetarians and non-vegetarians.

Studies of the LDL receptor in humans and other mammals and of LDL levels in newborns and in humans raised on low-fat diets suggest that total plasma cholesterol levels of approximately 110 to 150 mg/dl (2.8 to 3.8 mmol/l) may be physiological for human beings, and that the levels of plasma cholesterol in Western industrialized societies are inappropriately high (reviewed by Vorster et al., 1988a). Therefore, ingestion of animal fat in the diet results in an increase in plasma cholesterol level. However, the level does not increase equally for every person and thus both genetic and dietary factors determine individual plasma levels. The association between diet and CHD is discussed under section 2.6.

2.5.3.2 Low density lipoprotein cholesterol (LDL-C)

LDL is defined as that lipoprotein fraction with a density of between 1.019-1.063 g/ml; it carries about two-thirds of the cholesterol in normal plasma (Steinberg, 1985). Hypercholesterolaemia has been associated with an increased risk of developing CHD with the risk being positively correlated with LDL-C levels (Kannel and Castelli, 1979).

The mechanisms through which abnormal LDL-metabolism contributes to high TC levels and atherogenesis, are now better understood. Studies in patients with FH have shown that a specific LDL receptor exists on fibroblasts and liver cells that provides a major point of entry for the internalization of LDL into the cell (Brown et al., 1975). These authors showed that receptors for the apoB100 apoprotein of LDL occur on cells other than fibroblasts and that other sites are specific for apoE apoprotein which occurs on some lipoproteins. After binding to the receptor the LDL is internalized and hydrolyzed by intracellular lysosomal enzymes. The lipoprotein cholesterol thus released: (i) inhibits de novo cellular cholesterol synthesis (by inhibition of the enzyme HMG CoA reductase), (ii) activates esterification of excess cellular cholesterol by acyl:cholesterol acyltransferase (ACAT), and (iii) reduces the synthesis of LDL-receptors (Brown and Goldstein, 1981).

Thus cells have a system for metabolizing LDL to supply cholesterol for processes such as cell membrane synthesis (Brown and Goldstein, 1981). Normally the uptake of LDL-C by this route is tightly regulated to prevent accumulation of cholesterol in the cells. The efficiency of LDL metabolism by the LDL-receptor pathway in turn modulates plasma levels of LDL. However, it is not yet clear whether production or clearance of LDL is the main factor controlling LDL levels in patients with the common polygenic form of hypercholesterolaemia.

The precise mechanism whereby high levels of LDL (and hence LDL-C) lead to atherosclerotic lesions is imperfectly understood. Another hypothesis

has been put forward to explain how macrophages in the developing fatty streak may become loaded with cholesterol in response to high levels of LDL. Chemically modified LDL can be recognized by unique receptors on macrophages, referred to as the acetyl LDL receptor or the receptor for chemically modified LDL (Brown and Goldstein, 1983; Mahley and Innerarity, 1983; Steinberg, 1983; Mahley, 1985).

It has recently been stated that remnants derived from chylomicrons and large VLDL are taken up into the liver via the LDL receptor and, possibly the LDL-related protein (Shepherd and Packard, 1991). This process is mediated by apolipoprotein B (apoB). The liver has the capacity to secrete apolipoprotein B into a spectrum of VLDL particles (heterogeneous VLDL) varying in size and TG content (Shepherd, 1990). Normal circumstances favour the production of larger, TG-loaded particles which are not metabolized to LDL but instead are cleared directly from the circulation. Dietary cholesterol loading diminishes the size of the average VLDL particle and at the same time raises plasma LDL (Reviewed by Assmann 1990; Shepherd, 1990).

There is some evidence that raised triglycerides (TG), indicative of increased levels of very low density lipoprotein (VLDL), may be an independent risk factor for CHD (Assmann, 1990; Criqui, 1990; Gianturco et al., 1990; Ginsberg et al., 1990; Hamsten, 1990; Shepherd, 1990).

2.5.3.3 High-density lipoprotein cholesterol (HDL-C)

HDL is defined as that lipoprotein fraction with a density of between 1.063 g/ml and 1.21 g/ml. It contains a number of apolipoproteins, including apo A-1, apo A-2, the apo-C peptides, apo-D and apo-E, of which apo A-1 is quantitatively and functionally the most important (Berger, 1984). Freshly formed or secreted HDL exists briefly in the form of bilayered discs containing a high proportion of protein (mainly apo A-1) and phospholipid relative to cholesterol (Eisenberg, 1983). It is possible that these nascent discs (or similar forms) are the primary acceptors of excess cholesterol from extrasplanchnic cells before being converted or incorporated into the mature spherical particles which constitute the overwhelming proportion of the total HDL fraction of plasma under normal circumstances (Berger, 1984).

Total plasma HDL level is a composite of a number of subfractions. The most convenient clinical subdivision of HDL is into HDL_2 and HDL_3. HDL_2 is larger and less dense than HDL_3, and is enriched in cholesterol ester.

HDL also differs markedly from LDL in apoprotein and lipid composition. Hepatocytes have receptors that recognize HDL apoprotein E (Quarfordt et al., 1980). The uptake of HDL, internalization and degradation of HDL cholesterol, and possibly regeneration of cholesterol ester for HDL_3, can therefore be visualized. The liver appears to fill a prominent part in cholesterol handling. Since HDL_3 binds to arterial subintimal cells and promotes cholesterol efflux (Biesbroeck et al., 1983), HDL could fill a

role as the physiological counterpoise to LDL. Also, HDL is only one of a number of entities participating in reverse cholesterol transport. Other components include at least one enzyme, Lecithin : cholesterol acyltransferase (LCAT), one or more cholesterol ester transfer and exchange proteins (ECTEP), various receptors, LDL, VLDL and certain specific apolipoproteins present in the HDL fraction (Berger, 1984).

The negative association between the concentration of HDL-C in serum and the incidence of atherosclerosis (Berger, 1984) was reported by Barr et al. as early as 1951 and has been substantiated in many studies on Western populations (Reviewed by Berger, 1978 and by Eder and Gidez, 1983). However, despite the firmly established epidemiological evidence, the statistical link between HDL-C levels and a reduced incidence of atherosclerosis does not equally convincingly extend to special cases or to all population groups (Knuiman et al., 1980; Nestel et al., 1983; Gwynne, 1991). For instance, cholesterol feeding, which is unquestionably atherogenic in animal models can be associated with an elevated plasma HDL-C level as well as with high total cholesterol concentrations (Tan et al., 1980).

Under current NCEP-AHA (National Cholesterol Education Program - American Heart Association) guidelines, a low HDL-C level (below 35 mg/dl or 0.9 mmol/l) is considered a risk factor (Steinberg et al., 1989).

The reverse cholesterol transport hypothesis (Glomset, 1968; Biesbroeck et al., 1983; Berger, 1984; Gwynne, 1991) is currently the most

attractive hypothesis concerning the antiatherogenic action of HDL. It has been supported by a great deal of *in vitro* evidence, but, direct *in vivo* demonstration and quantification of reverse cholesterol transport in either experimental animals or humans is still lacking. In a recent update by Gwynne (1991), it has been stated that an understanding of the mechanism of reverse cholesterol transport is essential to develop the strategies for preventing CHD by modifying HDL function.

2.5.4 Hypercoagulability as a risk factor for coronary heart disease

2.5.4.1 Introduction

Hypercoagulability, which is an "increased thrombotic tendency" (Meade, 1984) represents a phase of consumption coagulopathy (synonyms: disseminated or generalized vascular coagulation) and is characterized as the activation of intravascular coagulation (Müller-Berghaus, 1977).

The alterations that have been observed in the haemostatic parameters of patients with atherosclerosis, are an increase in coagulation factors and their activities, a decrease in the natural caogulation inhibitors (Ogston, 1985), a decrease in the activity of the fibrinolytic system (Ogston, 1985), as well as alterations in the biochemistry and function of platelets (Packham and Mustard, 1980; Vermylen *et al.*, 1983; Packham and Mustard, 1986; Ulutin, 1986). The suggestion that the presence of a prethrombotic state can be evaluated by various parameters has resulted in the possibility of an early diagnosis of atherosclerotic alterations

and its potential prevention before a thrombus is formed. From the literature it is not always clear whether these associated alterations in haemoatatic variables are a cause or a consequence of atherosclerosis.

Hypercoagulability is frequently associated with activation of the fibrinolytic system (secondary fibrinolysis), as coagulation and fibrinolysis have several activators and inhibitors in common. Thrombosis has been considered to result from "haemostasis in the wrong place" (MacFarlane, 1977), or an unregulated and inappropriate form of haemostasis (Schafer, 1985). A pivotal event in the pathogenesis of myocardial infarction is thrombosis, with a 70% to 90% incidence of acute thrombotic occlusion of the artery supplying the infarcted zone in patients with transmural infarctions (Francis et al., 1987).

There is little doubt that a hypercoagulable state, combined with local stasis of blood, is the main cause of venous thrombogenesis (Thomas, 1985; Vorster et al., 1988b). However, the involvement of hypercoagulability in atherosclerosis and arterial thrombosis, has only recently been investigated in more detail.

2.5.4.2 Haemostatic function and coronary heart disease

° The role of fibrinogen

Fibrinogen is an acute-phase protein, which is synthesized in the liver. Its concentration in plasma is raised after infection or injury

(Humphries et al., 1987). In both men and women the fibrinogen concentration increases with age and obesity (Meade et al., 1979; Balleisen et al., 1985a). Plasma levels are higher in diabetics than in non-diabetics (Fuller et al., 1979). In women values are raised by the use of oral contraceptives and are high after the menopause (Meade and North, 1977; Meade et al., 1979).

Several prospective epidemiological studies have shown a direct association between the plasma fibrinogen concentration and the subsequent incidence of CHD (Meade et al., 1980; O'Connor et al., 1984; Wilhelmsen et al., 1984; Stone and Thorp, 1985; Meade et al., 1986b; Kannel et al., 1987). Also, high levels of coagulation factors are found in population groups known to be at high risk of CHD, and low levels in those at low risk (Meade, 1984). This association has also been demonstrated in clinical trials (Lowe et al., 1980b; Sugrue et al., 1985). The experimental evidence of the relation between thrombosis and atherosclerosis has been reviewed by Smith (1986). The contribution of smoking to abnormal haemorheology and the pathogenesis of CHD has been reviewed by Simpson (1984). Recent epidemiological studies (Stone and Thorp, 1985; Meade, et al., 1986a; Kannel et al., 1987a) have indicated that one of the mechanisms of the association between smoking and CHD is probably mediated through effects of agents in cigarette smoke on fibrinogen levels.

The mechanisms of activation of the coagulation system, and consequent formation of fibrin from fibrinogen, are illustrated in Figure 2.1.

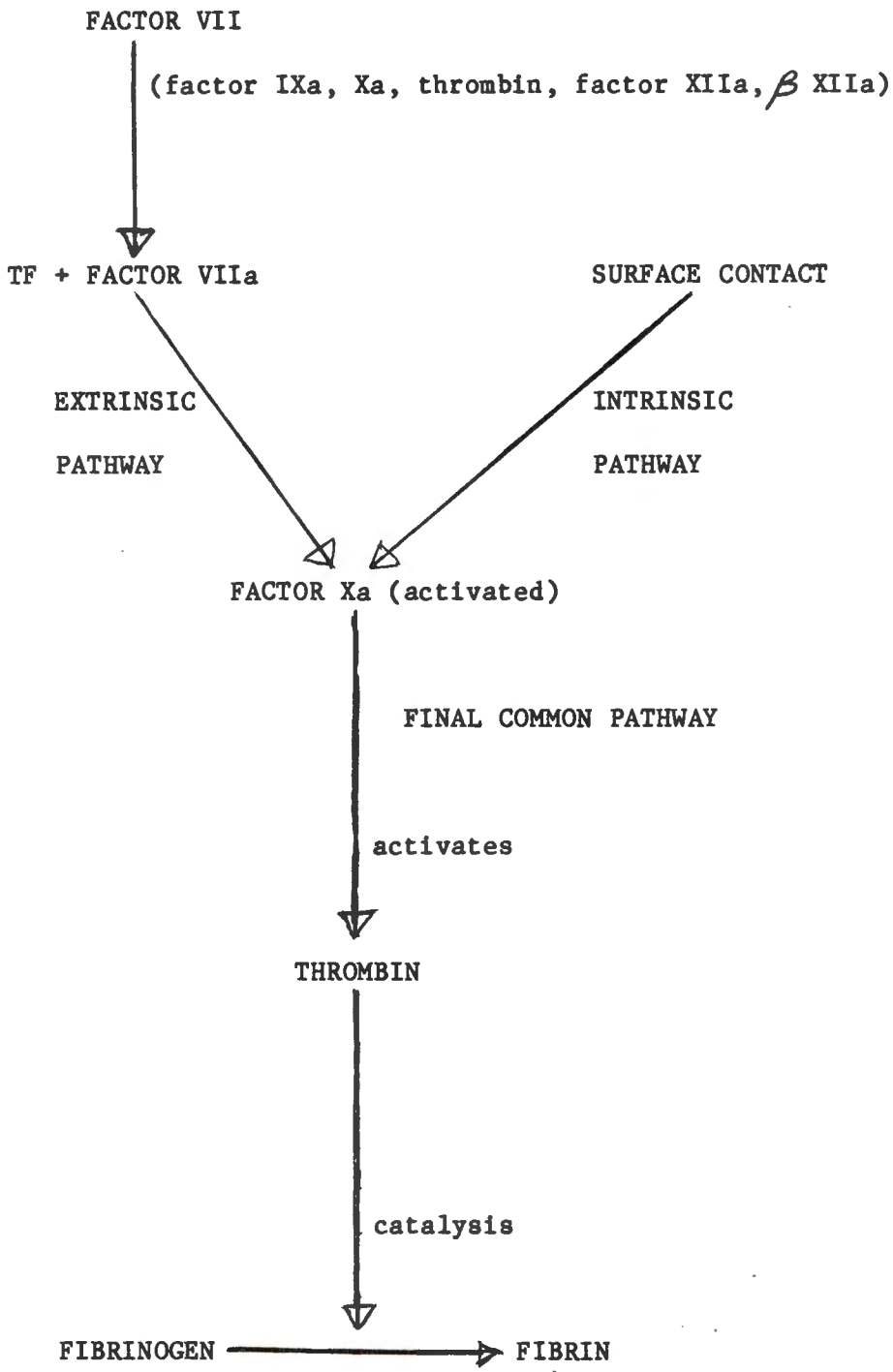


Figure 2.1. The coagulation system. TF = Tissue factor, (VIIa = activated factor VII) (adapted from Meade, 1981).

As indicated in figure 1, factor X may be activated by the extrinsic or intrinsic pathways. The pathway activated by tissue factor (TF) is "extrinsic" to the circulation itself and historically, early work has concentrated mainly on this system. TF which becomes available after trauma, results in physiological activation of the extrinsic system; TF from atheroma might be responsible for pathological activation.

The intrinsic system is the pathway activated as a result of contact between the blood and a surface (e.g. glass) without the requirement of an external factor. The extrinsic system, however, may have a greater pathophysiological significance. Activation of the extrinsic pathway results in much more rapid coagulation than activation of the intrinsic system (Jesty and Nemerson, 1977). Human factor VII may also have the unusual feature of bovine factor VII of circulating in an active or semi-active form. Thus, as soon as tissue factor is available, rapid *in vivo* coagulation will be initiated.

Another feature which suggests that activation of the extrinsic system may not be difficult to initiate is that factor VII does not seem to be inactivated by antithrombin III (Broze and Majerus, 1980). It has been shown how small amounts of an activated factor early in the coagulation sequence are likely to have very marked effects at later stages, thus increasing the likelihood of thrombosis (MacFarlane, 1964; Esnouf and MacFarlane, 1968). Thus, the extrinsic pathway seems to be delicately balanced between a high state of readiness for activation, on the one

hand, and the actual promotion of rapid, florid coagulation on the other. The physiological importance of a potentially swift response to injury, which this balance ensures, is clear. Also, it is not difficult to imagine the consequences which could follow pathological disturbance of the balance by advanced atheroma. It must be noted that factor VII activates factor X by two routes (Zur and Nemerson, 1980).

The final common pathway of the coagulation system consists of the activation of factor X, the conversion of prothrombin to thrombin and the production of insoluble fibrin from soluble fibrinogen.

The conversion of the soluble plasma protein fibrinogen to fibrin, which finally forms the visible clot, represents the terminal reaction in the coagulation cascade (Rötker et al., 1986). The fibrinogen/fibrin transition is initiated by the selective removal of one pair of fibrinopeptides A and B each by thrombin. The resulting fibrin monomers, designated as AABB-fibrin, polymerize to protofibrils and subsequently associate laterally forming a network of fibrin fibres (Ferry, 1952). Thrombin-activated factor XIII may stablize a fibrin clot by producing an intermolecular γ-glutamyl-ε-lysine side-chain bridge (Lorand and Konishi, 1964).

Several mechanisms have been proposed through which fibrinogen may influence atherogenesis (Kadish, 1979; Niewiarowsky and Rao, 1983; Meade et al., 1986b; Smith, 1986). Briefly stated (Vorster et al., 1988b) these mechanisms comprise, firstly a direct effect of fibrinogen

or fibrin by stimulating cell proliferation, by providing a scaffold along which cells may migrate, and by being involved in the tight binding of LDL and accumulation of lipids in the advanced plaque (Smith, 1986). Fibrin degradation products may also stimulate mitogenesis and collagen synthesis and may alter endothelial permeability. Secondly, fibrinogen may have an indirect effect by changing platelet function (Marguerie et al., 1986), blood viscosity (Turitto, 1982) and eventually blood pressure (Letcher et al., 1981), which may all contribute to the atherosclerotic process. It appears as if raised fibrinogen levels may be a promotive or a causative factor in the development of atherosclerosis, and not merely the result of the atherosclerotic process. The observation that raised fibrinogen levels and a hypercoagulable state prevail in young diabetic subjects before cardiovascular complications can be detected (Corbella et al., 1979) support this possibility.

2.5.4.3 Factor VII

Characterization of factor VII has an interesting history. Schofield (1924) investigated the haemorrhagic disease of cattle which results from the inclusion of spoiled sweet clover (which contains dicoumarol) in the fodder (Ackroyd, 1956). The injection of normal serum into the animal improved the condition. Subsequently an investigation of the action of dicoumarol on the clotting mechanism of dogs showed that the prolonged one-stage prothrombin time of the plasma of these animals could be corrected by the in vitro addition of a small amount of normal serum (Owen and Bollman, 1948). The factor in the serum was called prothrombin

conversion factor. After being given various different names, it was finally called factor VII (Koller et al., 1951).

The Northwick Park Heart Study (Meade et al., 1986b) indicated that high factor VII coagulant activity (VIIc) was associated with risk of CHD. It has been suggested that a high factor VIIc level may be characteristic of a hypercoagulable state (probably due to high fat intake (Miller et al., 1986a)) in men with CHD, in which activation of factor VIIc and thrombin generation precede the terminal event (Meade et al., 1984). Also, the presence of an abnormal factor VII complex in the plasma of men at risk for CHD has been demonstrated (Dalaker et al., 1985).

° Factors that influence factor VII level or activity

A rise in the steady state of concentration of several activated clotting factors will increase the coagulant activity of factor VII (Meade, et al., 1986b). This increase results from the cleavage of the native single-chain form of the protein to a two-chain form, αVIIa, which has a specific activity between 50 and 150 times that of the native protein. Since factor VII activity is inhibited only slowly in plasma (Broze and Majerus, 1980), its assay is a useful index of the rate of activation of those components of the coagulation system with which it interacts (Miller et al., 1985). In the presence of tissue factor, as would occur with the rupture of an atheromatous plaque, the rate of factor X activation by factor α VIIa is increased some 16 000-fold (Nemerson et al., 1980), thus increasing the potential rate of thrombin production in line

with the amplifying nature of the coagulation system (Macfarlane, 1964; Esnouf, 1984).

General epidemiological studies suggest that high levels of activity of factor VII may be of causal significance (Meade, 1984), particularly the oestrogen-dose-dependent effect of oral contraceptives of factor VII (Meade et al., 1976, 1977), which runs parallel to the dose-dependent effect that these preparations have on the risk of CHD and stroke (Inman et al., 1970; Royal College of Practitioners, 1974; Meade et al., 1980).

2.5.4.4 Evaluation of the role of coagulation in atherosclerosis and occlusive thrombosis

The hypothesis underlying the results of the Northwick Park Heart Study is that high clotting factor levels are of direct pathogenetic significance in CHD (Meade, 1984). Association, however, does not prove causation. Thus, high levels of factors VII and of fibrinogen may be simply "markers" of some other process or set of variables which are the real causes of CHD. In this context, the process most often suggested, is the development and progression of chronic arterial wall disease. Raised values of factor VII or fibrinogen occurring in response to inflammation and other stimuli may result in an increased risk of thrombosis. This is true after surgery, during and after pregnancy and in association with some malignant tumours. Increased risk of thrombosis in these circumstances is manifest in terms of venous rather than arterial disease. Nonetheless, high levels of factor VII and fibrinogen

may be contributing to "hypercoagulability" which has arisen for a number of different reasons. Thus, although high levels of factor VII and, particularly fibrinogen, associated with an increased risk of cardiovascular death, are secondary to chronic vessel wall disease, they may still be of causative significance in CHD.

It can be stated that randomized controlled trials help in the interpretation of observational data and to distinguish between association and causation. The secondary prevention trials of anticoagulants do suggest that the recurrence of CHD can be reduced through the lowering of levels of the vitamin-K-dependent clotting factors - factors II, VII, IX and X (reviewed by Meade, 1984).

2.5.5 Hypertension as a risk factor for coronary heart disease

2.5.5.1 Introduction

Hypertension is a heterogeneous entity with different genetic mechanisms. It has been stated that hypertension is an ecogenetic trait in that primitive populations do not have hypertension (Motulsky, 1987). It is necessary to discover the determinants of blood pressure because hypertension is a principal risk factor for CHD (Lichtenstein et al., 1986). Even those determinants having a small effect are important since a reduction in a population's mean blood pressure of 2-3 mmHg could potentially save as many lives as current antihypertensive therapy (Rose, 1981).

Age, body surface area, overweight, race and family history of hypertension have been found to be possible determinants of blood pressure from infancy to adulthood (Hofman and Valkenberg, 1983; Ibsen, 1985; Ounsted et al., 1985; Svensson and Hansson, 1985; Iaina et al., 1987).

Several studies have identified differences in blood pressure in teenagers by ethnicity (Goldbourt and Kark, 1982). Geographic and social class differences are also important factors (Huttunen et al., 1985). There might be a possibility that diabetes-prone South African Indians might have an 'early insulin resistance' state and thus the possible relationship between insulin resistance and hypertension is discussed in paragraph 2.5.9.2.

Data from a German epidemiological study suggest that coagulation factors (factor VIIc and fibrinogen) are associated with blood pressure (Balleisen et al., 1985b). In a study on the role of fibrinogen and fibrinogen concentration it has been shown that regardless of the haematocrit value, fibrinogen levels were elevated in hypertensive patients ($p < 0.006$) and, in association with the increased globulin concentration, fibrinogen was largely responsible for the increased plasma viscosity in hypertensive patients (Letcher et al., 1981). The same study indicated that elevated fibrinogen level also affected whole blood viscosity.

To explain hypertension as a risk factor for CHD, a haemodynamic theory of vessel wall injury has attracted a number of investigators (Packham

and Mustard, 1986). Although in normal vessels haemodynamic forces are unlikely to be strong enough to cause injury, the sheer stresses at stenoses may be great enough to remove the endothelium (Fry 1976; Gertz, et al., 1981; Joris et al., 1982), and this may also occur in vessels narrowed by vasospasm (Joris and Majno, 1981). However, early atherosclerotic plaques tend to form in regions of low velocity of blood flow and flow separation, rather than in regions of high velocity and high sheer stress (Zarins et al., 1983).

2.5.5.2 Prevalence of hypertension in Indians in South Africa, India and in Britain.

The age-adjusted prevalence of hypertension in three racial groups in Durban was found to be 25% for Zulus, 17,2% for whites and 14,19% for Indians (Seedat and Seedat, 1982). However, in an earlier random house-to-house study of 1 000 Indians in Durban, the prevalence of primary hypertension according to World Health Organization criteria was found to be 19% (females 22% and males 15%) (Seedat et al., 1978). The prevalence of hypertension, thus appeared to be higher than that of urban population groups in India (Padmavati and Gupta, 1959; Mathur et al., 1963; Malhotra, 1971; Dalal et al., 1977; Pai, 1977) as shown in Table 2.1. In a recent Durban study (Seedat et al., 1990) the prevalence of hypertension among Indian males was found to be 17.4% and among females it was 20%.

TABLE 2.1

Prevalence of hypertension in South Africa and India according to WHO criteria - as reported in some studies

Population Group	% Hypertensive		Reference
Blacks (Zulus) } South African	25%		
Whites } South African	17.2%		(Seedat & Seedat, 1982)
Indians } South African	14.19%		
	Male	Female	
South African Indians	15%	22%	(Seedat et al., 1978)
	17.4%	20.0%	(Seedat et al., 1990)
Indians in:			
Bombay	10%		(Dalal et al., 1977)
Madras	15.2%		
New Delhi	6.2%		(Malhotra, 1971)
New Delhi:			
(lower socio-economic group)	0.17%		(Padmavati and Gupta, 1959)
(higher socio-economic group)	2.5%		
Agra	4.3%		(Mathur, et al., 1963)
Bombay (slum dwellers in Malawani)	12,5%		(Pai, 1977)

In a population-based study of females living in Punjab, India and those who emigrated to Southall, London, age-adjusted prevalence of hypertension (blood pressure $\geq$ 160 mmHg systolic and/or > 95 mmHg diastolic) was found to be 23% in the migrants of Southall and 1,4% in the Punjab natives (Keil et al., 1980). A difference in the dietary habits of South and North Indians has been implicated in the explanation of the disparity in the incidence of hypertension between the two groups of 15,2% in the South compared with 6,2% in the North (Malhotra, 1971).

2.5.5.3 Tracking of blood pressure

Adult blood pressure patterns can be predicted from childhood patterns (Miall and Lowell, 1967; Zinner et al., 1967a). "Tracking" is reportedly much better in adults than in children (Clarke et al., 1976; Feinleib et al., 1980). Also, poor tracking over time would be a serious obstacle to any screening programme designed to identify and treat essential hypertension early in life. The findings of two earlier studies seem to show that children are "getting on track" as early as age 4 (Zinner et al., 1967b; De Swiet et al., 1980) and suggest that the interplay of gene-environment becomes manifest early.

Tracking of blood pressure in children was examined in a recent study (Berenson et al., 1992). Correlation coefficients for systolic blood pressure reading 12 years apart were highly significant beginning at age 5-6 years at baseline (17-18 years at follow-up).

2.5.6 Smoking as a risk factor for coronary heart disease

A substantial body of evidence exists incriminating the cigarette habit in CHD (reviewed by Kannel, 1981). This evidence is provided by prospective epidemiologic observations, by clinical and pathological correlations and laboratory and experimental findings. The chief components of cigarette smoke which have been identified as noxious to the cardiovascular system are nicotine and carbon monoxide (Kannel et al., 1968; Walld et al., 1973), which have been shown to evoke multiple adverse effects through cardiodynamic influences, atherogenesis, haemostatic changes, and vasculotoxic and inflammatory influences.

The pharmocologic effects of nicotine include stimulation of sympathetic nervous activity and release of catecholamines (Cryer et al., 1976), increases in myocardial irritability and heart rate, induction of vascular constriction and a transient rise in blood pressure, while platelets become more adherent (Doyle et al., 1976; The Health Consequences of Smoking, 1976; Friedman et al., 1979). Catecholamines mediate lipolysis causing an increase in plasma concentrations of free fatty acids and VLDL (Schoenenberger, 1982; Mjos, 1988).

The mechanism by which cigarette smoke lowers HDL-C is unknown (Muscat et al., 1991). The carbon monoxide build-up reduces the oxygen available to the myocardium (Kannel, 1981). The combination of these effects could precipitate sudden deaths and myocardial infarctions in cigarette smokers with a compromised coronary circulation (Karlsberg et al., 1981).

Of the three leading predictors of CHD, i.e. hypercholesterolaemia, hypertension and cigarette smoking, both hypertension and cigarette smoking are associated with altered haemorheology (Simpson, 1984). It has been suggested that the relationship between smoking and myocardial infarction occurs through the atherogenic effects of smoking, as well as the high levels of vascular occlusion (Hartz et al., 1981). Smoking also increases blood viscosity (Dintenfass, 1975; Lowe et al., 1980(a)) and in CHD, increased blood viscosity is asociated with the stenosis of coronary arteries (Lowe et al., 1980(b)).

Thus, it seems likely that in CHD where there is occlusion of more than one coronary artery, smoking-increased blood viscosity may act as a "trigger" for myocardial infarction (Simpson, 1984). Smoking could increase the blood viscosity by: (a) causing "smokers polycythaemia" (Smith and Landaw, 1978) which may be a relative polycythaemia because of a decrease in plasma volume, or an increase in haematocrit due to a carboxyhaemoglobin-mediated erythrocytosis (McAloon et al., 1980), and (b) smoking reduces the deformability of erythrocytes (Norton and Rand, 1981). The reduction in plasma volume could be due to increased transudation caused by the effects of poorly deformable erythrocytes in the microcirculation.

Some studies (Goldbourt and Medalie, 1977; Schoenberger, 1982; Elkeles, et al., 1983; Willett et al., 1983) have reported elevated blood cholesterol levels among persons who regularly smoke cigarettes, and lowered cholesterol levels among persons quitting smoking. Other

studies (Criqui et al., 1980; Philips et al., 1981; Elkeles et al., 1983; Willett et al., 1983; Wilson et al., 1983; Stamford et al., 1984; Lupien et al., 1988; Moffatt, 1988) have shown that smoking lowers HDL levels, resulting in an increased risk of CHD. Also, results obtained from several community based cholesterol screenings (Harris et al., 1989; Muscat et al., 1989; Wynder et al., 1989) have shown that levels of plasma cholesterol rise in association with the number of cigarettes smoked per day (Muscat et al., 1991).

Results of the Northwick Park Heart Study suggest that fibrinogen levels are higher in smokers than in non-smokers with intermediate values for ex-smokers (Meade et al., 1987). It has been suggested that a substantial proportion of the association between smoking and CHD is mediated through the plasma fibrinogen concentration (Meade et al., 1986b).

It has been reported that smoking is more common among unskilled manual workers who tend to be heavier (Khosla and Lowe, 1972), shorter (Rose and Marmot, 1981; Bingham et al., 1981), and have higher body mass index (Rose and Marmot, 1981) than non-manual workers. A study of the effects of smoking habit on nutrient intakes, controlling for the effects of social class, revealed that smoking habit had a greater effect than social class on intakes of dietary fibre, vitamins and minerals (smokers having lower intakes than non-smokers), but less effect than social class on intakes of energy and carbohydrate (Fehily et al., 1984).

2.5.7 Diabetes mellitus as a risk factor for coronary heart disease

2.5.7.1 Introduction

Diabetes mellitus (DM), which was known to Egyptian and Indian physicians 2 000 years ago (Sathe, 1973) and not mentioned by Hippocrates, has become a disease of considerable and increasing public health importance (Tokuhata, et al., 1975; Walker, 1977).

Two main types of diabetes are recognized: primary and secondary (Davidson et al., 1981). Most cases belong to the former group which is divided into insulin-dependent (IDDM) and non-insulin dependent (NIDDM). Although several contributing factors are known, the precise aetiology of DM is still uncertain.

A familial tendency to both IDDM and NIDDM undoubtedly exists. However, the specific biochemical defect and its mode of inheritance has yet to be identified. It has been stated that genetic factors are probably more important in those who develop diabetes after the age of 40, but in both young and old, environmental and other factors also operate and may determine which of those with a genetic predisposition develop the clinical syndrome and also when this occurs (Davidson et al., 1981).

Many of the metabolic abnormalities which probably contribute to the long-term complications of DM (Banga and Sixma, 1986), are possibly reversible

when hyperglycaemia is corrected (Jones and Peterson, 1981). Most cases of NIDDM can be corrected by following a diabetic diet (Cooppan, 1987).

2.5.7.2 Mortality from coronary heart disease in diabetic patients and in relation to degree of glycaemia

Increased mortality from CHD and stroke associated with DM has been supported by mortality statistics from England and Wales (Fuller et al., 1983), from other countries (Marks and Krall, 1971; Sasaki et al., 1978) and from the findings of several large cohort studies (Hayward and Lucena, 1965; Marks and Krall, 1971; Garcia et al., 1974; Shenfield et al., 1979). Several other studies (Pyörälä et al., 1979; Ducimetiére et al., 1982; Yano et al., 1982) as well as the Whitehall study (Fuller et al., 1980) have also shown an increased mortality from CHD in subjects with raised blood glucose concentrations indicative of glucose intolerance, although below those diagnostic of DM (WHO, 1980).

2.5.7.3 Prevalence of diabetes mellitus in Indians in South Africa, in Indian immigrants in other countries and in India

South African Indians, mainly in Natal, have been shown to have a much higher prevalence of DM than other racial groups (Campbell, 1963; Jackson and Huskisson, 1965; Marine et al., 1969). In the Transvaal, small surveys of special limited groups of Indians, confirmed the presence of a high frequency of diabetes in these groups (Walker et al., 1963; Walker, 1966). At present, the prevalence of DM among them is still high (Omar et al., 1985).

In Trinidad, the differences in risk of cardiovascular death between Indian and European men seemed to be accounted for by the high prevalence (19%) of NIDDM in Indians (Beckles et al., 1986). In Saigon, during the 1920s, the prevalence (30%) of diabetes in men of Indian descent was much higher than in other ethnic groups in the city (Montel, 1924). Reports of the same phenomenon have also come from Singapore (Cheah et al., 1979), Fiji (Zimmet et al., 1983), Guyana (Ashcroft et al., 1970), and England (Cruikshank et al., 1980; Mather and Keen, 1985). However, the extent to which their susceptibility to CHD is accounted for by their high prevalence of DM has not been ascertained.

Among people of the Indian subcontinent, NIDDM is common, especially in the affluent, obese, and middle-aged (Beckles et al., 1986). More than 70 years ago, studies in Calcutta showed that the average blood sugar of men in Bengal was higher than that reported for Europeans, and glucose tolerance was strongly associated with adiposity in Indians (McCay et al., 1916). The results of a recent survey amongst the urban population in the South of India, showed a much higher prevalence of diabetes than did a multicentre study done in 1975 by the Indian Council of Medical Research. The latter found an overall prevalence of 1.8% in the population aged over 15 years and a higher rate in urban than in rural areas (2.1% vs. 1.5%) (Ramchandran *et* al., 1988).

2.5.7.4 Vascular and haematologic alterations in diabetes mellitus

The long-term complications of DM may be either large vessel

(macro-vascular) disease or small vessel (micro-vascular) disease. Large-vessel disease is associated with thromboembolic events like myocardial infarction, peripheral occlusive vascular disease and stroke. Small-vessel disease, which is unique to diabetes, is characterized by thickening of the basement membrane, microthrombi and vascular proliferations. The consequences of small-vessel disease are diabetic retinopathy, kidney disease and only partly, polyneuropathy. It is suspected that these complications may relate to alterations in the haemostatic system (Ostermann and Van de Loo, 1986).

Pathological changes have been found in the endothelial cells, in the platelets (increased reactivity) and the plasma coagulation system; i.e. increased clotting factors and decreased fibrinolysis in patients with DM. Studies of the fluid phase of coagulation have suggested the existence of a hypercoagulable state in hyperglycaemic subjects (reviewed by Jones and Peterson, 1981). The clinical significance of these findings remain to be defined.

The findings of the Northwick Park Heart Study suggest a potentially important association between a thrombotic tendency and vascular disease in diabetes (Fuller et al., 1979). Mean values for factors VII and X, fibrinogen, and platelet adhesiveness were higher in the diabetics, but mean fibrinolytic activity and whole blood platelet counts were lower.

Antithrombin III values were also higher in the diabetics, which may have constituted a protective response to other changes favouring the onset of

vascular disease. Also, diabetics with retinopathy had higher factor VII and antithrombin III values, and those with proteinuria had higher values for factor VII, fibrinogen, and platelet adhesiveness than those without these complications. However, prospective data are needed to clarify whether the haemostatic abnormalities precede the onset of clinically manifest vascular complications or whether they are a consequence of them.

2.5.7.5 Interaction of coronary heart disease risk factors in diabetic subjects

In addition to the contribution of the hypercoagulable state in diabetic patients to atherogenesis and CHD, interaction of other risk factors may be of importance (Krut, 1979).

Evidence derived from autopsy and clinical data (Warren et al., 1966; Robertson and Strong, 1968; Bradley, 1971; West, 1978) shows that atherosclerosis tends to occur at an earlier age and with greater severity in the diabetic population than in the non-diabetic population (reviewed by Ganda, 1980). In known diabetics, macrovascular disease accounts for about 75% of all deaths, mainly from CHD (Marble, 1976; West, 1978). Despite evidence of striking heterogeneity in the aetiology of diabetes (Ganda and Soeldner, 1977; Pyke, 1979) the increased predilection for macrovascular complications is not restricted to any one type of diabetes.

In a review by Ganda (1980), it has been stated that the diabetic patient in addition to being susceptible to any, or a combination, of the pathogenetic factors operative in the non-diabetic, is at a particular disadvantage, because of at least three additional factors unique to the diabetic state: (i) associated presence of microangiopathy, (ii) hyperglycaemia and its consequences, direct or indirect, and (iii) hormonal aberrations underlying the altered metabolic/biochemical state either as a cause or an effect of diabetes. Also, often these subjects are overweight and present with mild glucose intolerance, slightly elevated blood pressure, hyperlipidaemia (Stamler, 1979; Fontbonne et al., 1988; Kaplan, 1989) and hypercoagulability (Fontbonne, et al., 1988; Fontbonne and Eschwege, 1991). These abnormalities may not separately determine a significant increase in cardiovascular risk (Fontbonne and Eschwege, 1991), but their clustering may interact to precipitate cardiovascular complications as is supported by the epidemiological findings of the Paris Prospective Study (Fontbonne et al., 1991).

The clustering of risk factors as a common clinical finding has given rise to the hypothesis that NIDDM is not constituted at random but is the multifaceted symptom of a basic abnormality, probably rooted in overweight and insulin resistance (Modan et al., 1985; Reavan, 1988). According to Fontbonne and Eschwege (1991) this hypothesis is not new. Decades ago, central obesity, i.e. preferentially abdominal fat deposits, was identified as a frequent concomitant of NIDDM and CHD (Vague, 1956; Feldman et al., 1969). This hypothesis has received new input from the results of prospective studies which identified central obesity (see

2.5.8; Kissebah et al., 1982; Krotkiewski et al., 1983; Larsson et al., 1984; Stern and Haffner, 1986; Ducimetiére et al., 1986; Donahue et al., 1987) or hyperinsulinaemia (Pyörälä, 1979; Welborn and Wearne, 1979; Ducimetiére et al., 1980), a direct marker of insulin resistance, as independent significant predictors of CHD incidence and mortality.

Furthermore, as pointed out by Bradley (1971), clinically significant atherosclerotic lesions are frequently unrelated to the duration or severity of overt diabetes. It may, in fact, be the presenting feature in individuals who may have had relatively mild diabetes, in the form of impaired glucose tolerance (chemical diabetes), for an uncertain duration. Also, diabetic persons have an increased prevalence of hypertension (50%), and glucose intolerance is more common in hypertensives (Kannel et al., 1991). Both share a strong relationship to excess weight, but the excess of hypertension in diabetic persons occurs in both lean and obese subjects. The risk of CHD, stroke and peripheral arterial disease increases with increasing blood pressure to the same degree in diabetic persons as in non-diabetic persons, but at any level of blood pressure, diabetics have a doubled risk of these diseases (Kannel et al., 1991).

Thus, greater efforts at primary prevention of both hypertension and diabetes are needed, including efforts at weight control (see 2.5.8.4), exercise (see 2.5.11), limitation of salt intake (see 2.6.6) and control of blood lipid levels.

2.5.8 Obesity as a risk factor for coronary heart disease

2.5.8.1 Obesity and its causes

Obesity is the accumulation of excess body fat, usually over a long period of time (Danforth, 1985). In general terms, men are considered obese when greater than 20% of their body weight is fat and women when greater than 25% of their body weight is fat. However, it is not easy to determine the amount of fat in the body.

In a mature organism obesity occurs when a positive balance develops between the energy intake and the energy expended, whereas, in a growing organism, obesity develops when the positive energy balance is larger than that required for normal growth and development. Gluttony and sloth are regarded as major causes of obesity in man (Forbes, 1984). However, it can be questioned whether all human obesity is the result of overeating or under-exercising, a combination of the two, or whether as in animal models, obesity in man results from a genetic or acquired defect in metabolism.

Danforth (1985) asks the question whether altered dietary composition alters the gain in fat and thus a tendency toward obesity. Are diet-induced adaptations in energy expenditure defective in some subjects, and thus predisposing them to obesity or perpetuating obesity once it is established? Would increasing the ratio of carbohydrate to fat in the diet make a difference in the prevalence of obesity? Is fat

more efficiently accumulated in the body from the fat than from the carbohydrate in the diet?

It is therefore still debated what effect, if any, an isocaloric diet with varying macronutrient composition may have on body composition (George et al., 1990). Results from two studies (Dreon et al., 1988; Romieu et al., 1988) suggest that fat intake may play a role in obesity regardless of total energy intake (EI). Of interest is a study in which a diet with a high carbohydrate to fat ratio resulted in a significant weight loss in females (Hammer et al., 1989).

2.5.8.2 Types of obesity

Evans et al. (1983) suggested that measurement of waist-to-hip girth ratio is a simple practical means of assessing body fat distribution. It compares well with more complex indices such as those based on skinfold thicknesses or the brachio-femoral adipo-muscular ratio. It may provide a useful marker of predisposition to metabolic aberrations and cardio-vascular disease.

Four types of human obesities are defined by topography of fat deposition (Bouchard, 1991). Type I is defined as excess weight-for-height or excess body fat without a particular concentration of fat in a given area of the body. The other three types are concerned with excessive accumulation of fat in some areas of the body, i.e. they are based on the anatomical distribution of body fat. Type II is defined as excess

subcutaneous fat on the trunk, particularly in the abdominal area, and is equivalent to the so-called android or male type of deposited fat. Type III is characterized by an excessive amount of fat in the abdominal visceral area. Type IV is defined as gluteo-femoral obesity and is observed primarily in women (gynoid obesity).

2.5.8.3 Relationship between type of obesity and coronary heart disease risk factors

Covas and Prats (1987) confirmed the role of obesity as a risk factor in the development of CHD among 1 388 individuals (1196 males and 192 females). These authors studied the incidence of obesity and overweight (expressed as body mass index (BMI)) and the relationship between obesity and other CHD factors. A relationship between obesity and CHD was also observed in a Norwegian longitudinal study of 1 700 000 subjects (Waaler, 1984). In these studies, interest has been focused mainly on quantity of adipose tissue and less on its distribution.

Several other studies examined the relationship between type of obesity and CHD risk. A cross-sectional study of men and women showed that cardiovascular risk factors such as hypertension, hypertriglyceridaemia, hyperinsulinaemia (see 2.5.9), and glucose intolerance were more pronounced in subjects with an android or masculine type of adipose tissue distribution - that is, in subjects with a high ratio of waist-to-hip circumference (Krotkiewski et al., 1983). Similar results were obtained in cross-sectional studies (Kissebah et al., 1982; Hartz et

al., 1983; Szathmary and Holt, 1983; Lapidus et al., 1984). Also, a high ratio of waist-to-hip circumference was associated with an increased risk of CHD in subjects in the Gothenburg longitudinal population study (Larsson et al., 1984).

Furthermore, other findings suggest an important link between central obesity [or Paunches (Tunstall-Pedoe, 1984) or Potbellies (Anderson, 1985)] and lipoprotein levels, and also perhaps, CHD risk.

Waist-hip circumference ratio (WHR) was used as a measure of central obesity in a study among South Asians and Europeans in Britain (Mattock et al., 1990). Triglyceride levels correlated significantly with serum insulin and WHR in both groups and the differences in triglyceride and HDL-C were explained statistically by a striking ethnic difference in WHR (0.97 in South Asians versus 0.93 in Europeans, $p < 0.001$). In addition, abdominal and subscapular fat deposits have been found to be more closely related with serum lipids than other fat deposits, particularly in males (Després et al., 1985).

Data from one study (Williams et al., 1992) support the concept of body fatness standards in white and black children and adolescents (aged 5 to 18 years) as significant predictors of CHD risk factors, i.e. elevated blood pressure, serum total cholesterol and serum lipoprotein ratios.

2.5.8.4 Prevention of obesity

The issue of obesity prevention and intervention in adolescents raises

many difficult questions (Johnston, 1985): what are normal adolescent growth and development patterns, the relationship between health status and the variability in fatness at specific ages, the criteria or cut-off points for adolescent obesity and what obesity prevention and intervention techniques are effective for adolescents? One of the year-2000 nutrition-objectives in the USA is the reduction of obesity in several American minority populations (Public Health Service, 1991). Because the risk of overweight in adults and the fact that many weight-related behaviours acquired during childhood and adolescence are likely to be maintained in adulthood, teenagers are a key group to target for obesity-prevention efforts (Jackson et al., 1991).

2.5.9 Insulin resistance as risk factor for coronary heart disease

2.5.9.1 Introduction

Insulin resistance may be defined as a sub-optimal biological response to insulin. It is often associated with increased circulating levels of insulin (Ferrannini et al., 1987). The results of epidemiological and clinical studies (Christlieb et al., 1985; Modan et al., 1985; Landsberg, 1986; Fontbonne and Eschwege, 1991; Fontbonne et al., 1991; Fruchart, 1991) have been consistent with the hypothesis that obesity and increased insulin resistance are associated with increased insulin secretion and in turn with high serum triglyceride levels and consequently low levels of serum HDL-C (Garcia-Webb et al., 1983). Thus, it has been suggested that hyperinsulinaemia might represent an early

metabolic alteration associated with the development of myocardial infarction and peripheral vascular disease. The proposed mechanisms for the relationships between insulin resistance and CHD are a possible effect of insulin resistance on lipoprotein abnormalities (Fruchart, 1991), or an effect on hypertension (see below).

2.5.9.2 Insulin resistance in essential hypertension

It has been stated (Ferrannini et al., 1987) that high blood pressure is prevalent in obesity and in diabetes; both conditions with insulin resistance. Also, multiple lines of evidence have linked elevated blood pressure with obesity (Havlik et al., 1983), while weight loss, in addition to improving insulin sensitivity (Golay et al., 1985) may lead to blood pressure reduction (Reisen et al., 1978) even more effectively than does antihypertensive medication with β-adrenergic antagonists (MacMahon et al., 1985). A recent epidemiologic study has suggested that high blood pressure is associated with glucose intolerance or NIDDM independently of obesity, and that the feature common to these three conditions, ie. obesity, NIDDM, and hypertension, is hyperinsulinaemia or insulin resistance, or both (Modan et al., 1985). However, direct proof of an aetiological role of insulin resistance, although ample for obesity (Olefsky et al., 1982) and NIDDM (De Fronzo et al., 1982), is lacking for hypertension. Also, it is not known which of the many in vivo actions of insulin (eg. inhibition of hepatic glucose release and lipolysis, stimulation of glucose and amino acid transport, oxidation and non-oxidative disposal of glucose and promotion of cellular potassium uptake

(Ferrannini et al., 1987; Shen et al., 1988), would be impaired in the hypertensive state, and what relation this might have to elevated blood-pressure levels.

In essential hypertension, reduced sodium-potassium-ATP-ase activity is suspected to be an inherent cellular malfunction (Hilton, 1986), and unsaturated free fatty acids have been implicated as inhibitors of the sodium pump (Ahmed and Thomas, 1971; Bidard et al., 1984; Tamura et al., 1985; Ng and Hockaday, 1986). On the other hand, insulin lowers plasma levels of free fatty acids and directly stimulates sodium-potassium-ATP-ase (Clausen, 1986). It is therefore conceivable that one link between insulin resistance and hypertension may be at the level of cellular cation transport.

2.5.9.3 Insulin resistance and android obesity

It has been suggested that the increased susceptibility of subjects with high waist-to-hip girth ratio to metabolic aberrations is largely mediated by insulin resistance (Evans and Kissebah, 1984). Also, women with high waist-to-hip girth ratio are characterized by enlargement of subcutaneous abdominal (but not femoral) fat cells and by an increase in adrogenic activity; the latter being correlated with both abdominal and adipocyte volume and with the degree of insulin sensitivity in vivo (Kissebah et al., 1982; Evans et al., 1983).

An increase in androgenic activity may result in enlargement of abdominal

fat cells and thus a high waist-to- hip girth ratio, leading to the development of insulin resistance both in adipose-tissue and in other metabolically active tissues, e.g. skeletal muscle (Kissebah et al., 1982). This in turn results in the development of metabolic aberrations and hence in an increased susceptibility to CHD (Larsson et al., 1984; Stern and Haffner, 1986; Donahue et al., 1987).

2.5.10 Family history as risk factor for coronary heart disease

It has been established that cases of premature CHD tend to aggregate in families (Berg, 1983). This author mentions that genetic studies may uncover evidence of an effect of genes on known risk factors, or show that the disorder aggregates in families in a way suggestive of either simple inheritance or genetic predisposition. This gives rise to a complex problem of interaction between environmental, nutritional or lifestyle factors on the one hand and the individual's genotype on the other. The familial component as risk for CHD has been demonstrated in several studies.

In Finland, a very high risk ratio for CHD by age 55 in probands of brothers was shown if the proband had CHD by age 46. In some families premature CHD occurrence was compatible with it being an autosomal dominant trait (Berg, 1989).

A United States study demonstrated that myocardial infarction in a first degree relative before age 55 is a highly significant risk factor (Nora

et al., 1980), stronger than cigarette smoking, blood pressure, or total serum cholesterol at conventional cut- off levels.

The results of a study of an Israeli population sample also provide evidence for familial aggregation of plasma cholesterol, triglyceride and HDL-C. This study indicated that much of the variation in lipid variables can be explained by genetic factors (Friedlander et al., 1982).

The heritability of lipid and lipoprotein indices were examined in one study of 98 monozygotic (MZ) and 100 dizygotic (DZ) twin pairs (Berg, 1989), as well as another 150 MZ pairs. Within pair correlation was used as a heritability estimate. The heritability estimate was extremely similar for levels of aoplipoprotein A-I (Apo A-I), apolipoprotein A-II. (Apo A-II), and apolipoprotein B (apo B) in the two series. The heritability level of atherogenic lipoprotein(a) (Lp(a)) was unity, confirming the inherited nature of this lipoprotein variation.

Blood pressure is under the influence of genes (Berg, 1989) and when within-pair correlation coefficient in MZ pairs was used to measure heritability, unexpectedly high estimates were obtained. However, with the exception of Lp(a) level, genetic factors explained only part of the quantitative-variations, providing space for risk factor modification by manipulation of life-style or diet. Also, the genes contributing to risk factor levels are mostly unknown. However, the availability of numerous restriction fragment length polymorphisms (RFLPs) at apolipoprotein loci has greatly increased the scope for application of the candidate gene

approach (Berg, 1989). Such polymorphisms exist also at the LDL receptor locus and at other loci-controlling structures or functions that could play a role in atherogenesis, thrombus formation, or protective processes.

It has been suggested that genetic factors play an important role in the high death rate from CHD among white South Africans with Afrikaans as their home language (Afrikaners). Seftel's work (Seftel et al., 1980) has indicated strongly that Afrikaners from the Witwatersrand area have a high prevalence of familial hypercholesterolaemia (FH). It has been stated that in districts where more than 80% of the white population are Afrikaners, rates of death (Pretorius, 1983) from CHD in males under the age of 50 years and in females under 55 years are higher than those for the rest of the country and that the reasons for this excess of CHD must a priori lie in an environmental or genetic predisposition of the disease.

However, the results of a study in India suggest that environmental factors are to a large extent responsible for the familial correlations of CHD that have been observed in healthy persons in previous studies (Mowar et al., 1977). According to Mowar this may be explained by the fact that the family members, close or distant, tend to share multiple characteristics of family in common, many of which are capable of mimicking genetic transmission factors. It is stated that the so-called family eating pattern, common dietary habits which are transmitted from

one generation to another seem to be most powerful with respect to serum cholesterol and other lipid parameters.

Further investigation of the siblings and the parents of Johannesburg Indian matriculants (this study) who had serum cholesterol levels greater than 5,5 m mol/l yielded seven index cases who qualified for further investigation for familial hypercholesterolaemia (Asvat et al., 1989, unpublished work). The prevalence of FH was found to be between 1:45 to 1:65 in this group of subjects studied and it was concluded that these findings are similar to the high prevalence rates in the other two high-risk South African groups, viz. the Afrikaner community and the Jewish community (Seftel et al., 1989).

2.5.11 Physical inactivity as a risk factor for coronary heart disease

Any bodily movement produced by skeletal muscles that results in energy expenditure can be defined as physical activity (Powell et al., 1989) which varies by day, season, and stage in life. It has been stated that lack of regular physical activity leads to a broad spectrum of chronic conditions such as CHD, hypertension, stroke, colon cancer, breast cancer, lung cancer, osteoporosis, non-insulin-dependent diabetes, low back pain, obesity, depression and anxiety (Powell et al., 1989). Epidemiological and clinical studies have suggested that physical activity delays the onset or slows the progression of coronary atherosclerosis (Brown and Milry, 1977). Considerable attention has been paid to the interrelationship between lipoprotein cholesterol levels,

exercise, and CHD (Smith et al., 1982). It was found that strenuous exercise was associated with higher HDL-C in men and women in an experiment where age, body mass index, alcohol use, and smoking were controlled. Diet however, was not (Haskell et al., 1980). There is also a predominance of reports linking increased exercise levels with diminished triglyceride (TG) levels as well as with lower levels of LDL-C and VLDL-C (Wood and Haskell, 1979).

There are several possible explanations for the inverse correlation between physical exercise and plasma TG levels (Slater et al., 1982). It is suggested that the increase in physical exercise results in an enhanced uptake of albumin-bound free fatty acids by skeletal muscle, which results in decreased entry of free fatty acids into the liver and this would cause a reduction in VLDL production. The release of newly-formed TG into the circulation would therefore diminish (Nikkilä et al., 1978). It has also been suggested that the initial step in a sequence of biochemical events leading to increased HDL-C levels in physically active persons might be an increased lipolytic rate of TG-rich lipoproteins resulting from the enhanced lipoprotein lipase (LPL) activity in their adipose tissues and skeletal muscles (Nikkilä et al., 1978).

Another explanation which complements the first is that the augmented blood flow through exercising skeletal muscle causes rapid lipolysis of VLDL-TG due to increased substrate-enzyme interaction (Slater et al., 1982). LPL (the key enzyme responsible for the lipolysis of VLDL-TG on the luminal surface of the capillary endothelium) exerts its lipolytic

function at maximal efficiency in the exercising muscle, as compared with the sedentary, reactive, noncontracting muscle, through which blood flow (and, therefore, enzyme-substrate contact) are minimal (Slater et al., 1982). It has been reported that the activity of skeletal muscle LPL increased more than twofold at the end of a 20km run in men who customarily engaged in vigorous exercise (Taskinen et al., 1980).

It has thus been suggested that physical exercise protects against CHD and concomitant premature death, although direct experimental evidence for this effect from intervention trials is lacking (Slater et al., 1982).

2.5.12 Stress as a risk factor for coronary heart disease

It has been known for many centuries that psychosocial and behavioural factors influence disease (Reviewed by Sinatra, 1984). In the 12th century, Maimonides (1135-1204) in his essays on health and youth, concluded that "emotional disturbances cause marked changes in the body" (Maimonides, 1958), and in 400 BC Hippocrates stated that "meditation heals evil thoughts, sadness and woes."

The concept of "conorary personality" was first propounded in the 1930s and 1940s both by Frans Alexander and Helen Dunbar (as quoted by Davies, 1981). They considered each psychosocial disease as representing a particular style of behaviour in which unventilated affect takes its toll of the appropriate physiological system. More recently, increasing

concern and interest has been focused on the influence of personality factors on coronary artery disease (Case et al., 1985). William Harvey's (1628) allusion to an intimate association between neural factors and the heart was made more than 350 years ago and yet it was not subjected to systematic direct scientific inquiry until the 1960s (Graboys, 1984). Over the past 30 years the approach to identifying persons at risk for cardiac death on the basis of personality factors has been greatly refined.

During the 1950s, two cardiologists from California, Meyer Friedman and Ray Rosenman, produced the concept of type-A coronary-prone behaviour (as quoted by Steptoe, 1985). From their clincial impressions of coronary patients, they identified a constellation of behavioural characteristics that included sustained aggression, ambition, competitiveness, and a chronic sense of time urgency and labelled them as type-A, while people who did not display these characteristics, but acted in a more relaxed and deliberate fashion, were labelled as type-B. It was hypothesized by Friedman and Rosenman that coronary risk would vary with the intensity of the behaviour pattern (Steptoe, 1985). Type-A behaviour can be assessed reliably by using Rosenman's structured interview (Rosenman, 1978), which has been developed into a video-taped clinical interview (Friedman et al., 1986). Also available for the assessment of type-A behaviour is the 14-item Bortner questionnaire which is used extensively in Great Britain and Western Europe (Bortner, 1969). Johnston et al., (1987) found that the Bortner questionnaire has adequate reliability and relates well to the Jenkins activity survey questionnaire which is more widely used in North America (Johnston and Shaper, 1983).

The predictive power of type-A behaviour pattern in CHD was examined in three large prospective American studies of people initially free of CHD (Sensky, 1987). The incidence of CHD was significantly greater in type-A than in type-B subjects, even after controlling for other risk factors such as cigarette smoking, blood pressure and serum cholesterol concentration in the Western Collaborative Group and Framingham Studies (Rosenman et al., 1975; Hayes et al., 1980), while the MRFIT study failed to confirm these results. However, some of the findings of this study are difficult to explain (Shekelle et al., 1985). Also, the incidence of type-A behaviour has been found to vary between populations, suggesting that not all environments and cultures elicit the same response pattern (Steptoe, 1985). It seems to be more prevalent in people with higher education and those with administrative responsibilities. High type-A ratings are found among hospital physicians and business-school graduates, and relatively low levels in labourers and other manual workers, or in people in non-competitive jobs (Zyzanski, 1978).

Many mechanisms have been proposed for the link between stress and CHD, but scientific proof of such a link will be difficult (Sobel, 1962). It has been proposed that stress acts mainly by increasing the secretion of noradrenaline, thereby producing vascular spasm, elevation of blood pressure, and endothelial damage (Rosenman, 1982). It is believed by some that stress may operate indirectly, by causing people to smoke and overeat, and hence to increase their risk of developing heart disease, as well as provoking excessive arousal of the autonomic nervous system

(Jenkins, 1982). It can be argued that the greatest problem that plagues research on this subject is the difficulty in defining stress as one man's stress is another's pleasurable challenge (Freeman, 1983).

A wealth of evidence has linked central and peripheral sympathetic mechanisms to changes in cardiac electrophysiology (Graboys, 1984). However, an independent relation between psychosocial stress and cardiac mortality has yet to be defined, due to the difficulty inherent in the qualification of higher neural activity. A causal link between acute psychologic stress and a cardiac event may be easier to define as sudden changes in sympathetic adrenergic tone and "in levels of catecholamine and neurotransmitters that may acutely alter cellular automaticity and excitability" (Graboys, 1984).

While the ongoing polemic dealing with diet and CHD and the relative significance of various risk factors continues to confuse correlation with causation (Anon, 1983b; Oliver, 1983; Finn, 1983), emotions and personality characteristics have been shown to have a prompt and profound effect on serum cholesterol, CHD and sudden death (Van Doornen and Orlebeke, 1982).

Considerable research effort has been expended in the study of the psychophysiological correlates of the Type A behaviour pattern (Harrison and Gorelczenko, 1990). It has been stated that raised cholesterol values, hypertension, and cigarette smoking may also be manifestations of type-A behaviour and responses to stress. Their association with CHD may

to a large extent be a function of this relationship (Reviews in Glass and Contradd, 1982; Dembroski et al., 1983; Rosch, 1983; Harbin, 1989).

It has been pondered whether modifications of type-A behaviour would be feasible or ethically acceptable as a method of reducing coronary risk in healthy people (Steptoe, 1985), since the logic of prevention implies that fit type-A individuals should be encouraged to alter their behavioural style (including their ambitions, gratifications and goals) for the sake of a potential threat to well-being in the future. However, beneficial effects of relaxation and stress reduction classes are so great that the financial and health care implications are potentially enormous (Marmot, 1985).

2.6 Diet and Coronary Heart Disease

2.6.1 Introduction

The hypothesis that diet plays an important role in the development of CHD was initially derived from the observation that the incidence and prevalence of the disease differed amongst population groups with different dietary patterns. Subsequent experimental work confirmed influences of several nutrients on the risk factors of the disease.

The diet-lipid theory of atherosclerosis was formulated in 1958 (Katz et al., 1958). It stated that "....atherosclerosis is a metabolic disease, in which altered cholesterol-lipid-lipoprotein metabolism plays a critical and decisive (but not exclusive) role". Thus, this early diet-heart hypothesis depended primarily on the role of lipids in the development of atherosclerosis (Kushi et al., 1985). This hypothesis has been supported by geographical (Armstrong et al., 1975) and metabolic-ward studies (Hegsted et al., 1965; Keys et al., 1965a, 1965b). Also, intervention trials of the effect of dietary change on CHD have suggested that altering the fatty acid composition of the diet may lower the risk that CHD will subsequently develop (Miettinen, 1975).

The effects of different nutrients and diet composition on the risk factors as well as incidence of CHD will now be discussed. Dietary factors which are thought to be related to hypertension, will be discussed separately in 2.6.6.

2.6.2 Total fat

It is estimated that the "natural" diet of our paleolithic ancestors consisted of approximately 21% of energy as fat, of which a large part was polyunsaturated (Eaton and Konnor, 1985). They consumed large amounts of meat, which provided 35% of their daily energy, but the meat of "natural" wild animals is much lower in fat, especially in saturated fatty acids, than that of modern farm animals (Katan, 1985).

The overwhelming evidence that total fat intake is associated with serum lipid and lipoprotein levels, has led to a recommendation in most developed countries that total fat should be limited to not more than 30% of energy intake (reviewed by Trusswell, 1987).

However, there is little evidence that fat intake is falling in any of the affluent populations (Katan, 1985). There are interesting trends, however, in the proportion of the main polyunsaturated fatty acid (PUFAT), linoleic acid (C18:2n-6) which is an essential fatty acid (EFA), in subcutaneous body fat of Americans which has been rising steadily over the past 20 years. This change agrees with their dietary changes, namely, a reduction in animal and dairy fat consumption, and an increased intake of vegetable oils. This is similar to the changes in the dietary fatty acid pattern in the Netherlands (Katan, 1985).

These dietary trends i.e. a high fat intake combined with a high intake of monounsaturated fatty acids (MUFATs) and polyunsaturated fatty acids (PUFATs) and a low intake of saturated fatty acids (SATFATs) can be regarded as a new dietetic phase. The contribution of these changes to reported falls in CHD incidence (see section 2.2) remains to be determined (Beynen and Katan, 1985).

2.6.3 Dietary fatty acids

2.6.3.1 Introduction

Diets usually comprise three categories of fatty acids: SATFAT, MUFAT

and PUFAT (Grundy et al., 1989). The general concept has arisen that SATFATs raise serum cholesterol levels and PUFATs lower cholesterol concentration (Grundy and Denke, 1990). This has resulted in the view that SATFATs should be reduced and PUFATs increased in the diet.

Studies carried out on the effects of various diets on blood cholesterol levels culminated in the publication of two equations, one derived by Hegsted et al., (1965), and the other by Keys et al., (1957), which summarized the effects of changes in saturated and polyunsaturated fat intake on circulating cholesterol (Horrobin and Manku, 1983). These effects, however, were disappointingly small. A rise in PUFAT intake equal to 1% of total calorie intake would produce, according to the Keys formula a fall in plasma cholesterol of only 1.31 mg/100 ml (0.04 mmol/l) or 1.65 mg/100 ml (0.05 mmol/l) according to the Hegsted formula, (mean 1.48 mg/100 ml) (0.04 mmol/l) and a fall in SATFAT equal to 1% of total calorie intake would produce a fall in cholesterol of only 2.74 mg/100 ml (0.08mmol/l) (Keys) or 2.16 mg/100 ml (0.06 mmol/l) (Hegsted). It can thus be seen that in order to produce changes in plasma cholesterol substantial enough to be worthwhile, large changes in dietary fatty acid patterns must be achieved.

2.6.3.2 Saturated fatty acids

Recent reports (Mattson and Grundy, 1985; Bonanome and Grundy, 1988; Grundy and Vega, 1988) confirm earlier observations that LDL-C levels, as well as TC concentrations, are raised by SATFATs in the diet. The mechanisms whereby SATFATs raise LDL levels are not completely understood.

It is probable that they interfere with LDL receptor-mediated clearance of LDL (Grundy and Denke, 1990). The serum-cholesterol raising action of SATFATs is manifest largely in the LDL-fraction; these fatty acids generally do not raise TG concentration, as might be expected if they were to stimulate the synthesis of VLDL, the precursor of LDL. As indicated by isotope-kinetic studies of LDL apo-lipoprotein B-100 (apo B) in humans (Shepherd et al., 1980) the LDL-raising action of SATFATs is due mainly to impaired removal of LDL from the circulation. SATFATs theoretically could act by a second mechanism, namely, by enhancing synthesis of apo-B containing lipoproteins (Mahley, 1985; Mahley et al., 1985).

Of interest are recent investigations based on older research results that have shown that SATFATs differ in their effects on lipoprotein metabolism. One example is that the SATFAT stearic acid (C18:0), does not raise the total or LDL cholesterol level (Bonanome and Grundy, 1988). Results from previous studies in humans imply that myristic aid (C14:0) is more cholesterolaemic than either lauric acid (C12:0) or palmitic acid (C16:0) (Hegsted et al., 1965; Keys et al., 1965). Results from a recent study in non-human primates suggest that the cholesterol-elevating potential of C14:0 is four times that of C16:0 (Hayes et al., 1991). This result is similar to that obtained by using the Hegsted equation, based on natural fats and oils fed to humans (Grande et al., 1970).

2.6.3.3 Monounsaturated fatty acids

MUFATs consisting mainly of oleic acid (n-9, cis 18:1)), were considered

until recently to be neutral in their effect on lipoprotein metabolism (Grundy et al., 1989), and have thus been thought to have no place in cholesterol-lowering diets.

However, many studies (Connor et al., 1964; Hegsted et al., 1965; Keys et al., 1965; Grundy, 1986; Baggio et al., 1988) indicate that oleic acid "lowers" plasma cholesterol when substituted for palmitic acid. If LDL-receptor activity is the prime variant, oleic acid presumably allows for the natural expression of receptor activity, whereas palmitic acid actively reduces the activity (Grundy and Denke, 1990). Also, in contrast to linoleic acid, oleic acid does not lower HDL-C and it apparently does not cause immune system suppression or predisposition to carcinogenesis in laboratory animals (Grundy et al., 1989).

Large quantities of oleic acid in the form of olive oil are consumed by people in the Mediterranean region. Their CHD rates are relatively low (reviewed by Grundy et al., 1989).

2.6.3.4 Polyunsaturated fatty acids

PUFATs have been generally looked on favourably because of their cholesterol-lowering properties. However, there are reports that PUFATs reduce HDL cholesterol levels (Grundy et al., 1989). Almost all studies have been done on a single PUFAT, cis-linoleic acid (cLA, C18:2), which has three major metabolic fates, which could relate to its hypocholesterolaemic effects.

Firstly, cLA itself, in an unmetabolized form, may be incorporated into lipoproteins, changing their characteristics. Most investigators seem to assume that this is the mechanism responsible for the cholesterol lowering effect (Horrobin and Manku, 1983). Secondly, cLA may be oxidized to provide energy. It is highly improbable that this route of metabolism accounts for the cholesterol-lowering effect. Thirdly, cLA may be metabolized to γ-linolenic acid (GLA) (C18:3n-3) and then on to other essential fatty acids and their eicosanoid metabolites (Mead and Fulco, 1976).

This third route of metabolism explains most of the essential fatty acid (EFA) effects of cLA. If the metabolism of cLA to GLA is blocked, as in cats in which the δ-6-desaturase enzyme is genetically absent, cLA has few if any of its expected effects as an EFA (Frankel and Rivers, 1978). It has long been argued that it is the EFA properties of cLA which are crucial for any action in reducing the risk of CHD (Sinclair, 1980). Also, there is some experimental evidence for this since Kingsbury et al., (1961) found that arachidonic acid, an essential metabolite of cLA, has a much greater cholesterol-lowering action than cLA itself. It was demonstrated that GLA in the form of evening primrose oil had a cholesterol-lowering effect far greater than that of linoleic acid as predicted by the Hegsted-Keys equations (Horrobin and Manku, 1983).

It should be remembered that replacing SATFATs in the diet with PUFATs, will reduce cholesterol levels because of a direct effect of the PUFAT, and also because of a replacement effect.

2.6.3.5 Omega-3 polyunsaturated fatty acids

In 1913 Stefansson described the high-fat diet (fish and seal meat) of Eskimos and their very low proneness to CHD and cancer (Walker, 1985). Fish oils are rich in omega-3 fatty acids, mainly eicosapentanoic (C20:5) and docosahexaenoic (C22:6) fatty acids (Ballard-Barbash and Callaway, 1987).

Numerous scientific publications, magazine and newpaper articles, conferences and workshops elaborated on the role of these fatty acids in health and disease (reviewed by Simopoulus, 1986).

Marine n-3 fatty acid supplementation has been shown to have beneficial anti-atherogenic actions related to atherosclerosis, thrombosis, lipid metabolism and mainly a reduction in TG levels (Nestel et al., 1984; Leaf and Weber, 1988). The effect of fish oil on the various lipoprotein fractions is variable and depends on the dose as well as on the existing plasma lipid pattern. HDL-C levels generally increase in subjects with normal lipid levels (Sanders and Roshanai, 1983; Thorngren et al., 1986). It has been observed that fish oil supplementation is associated with marked reductions in fibrinogen levels (Hostmark et al., 1988; Radack et al., 1989).

In addition to its effect on the risk factors of CHD as mentioned above, the evidence for a protective effect of dietary eicosapentaenoic acid against CHD is considerable (Fischer and Weber, 1984; Rogers et al.,

1987). The low death rate from coronary heart disease among Greenland Eskimos can be explained by their high fish consumption (Dyerberg et al., 1978; Bang, 1987). Also, in Japan, the lowest death rate from CHD is found on the island of Okinawa, where fish consumption is about twice as high as it is in mainland Japan (Kagawa et al., 1982).

Eicosapentaenoic acid (EPA) is the desaturation product of the actions of delta-6-desaturase (δ 6D) and delta-5-desaturase (δ5D) on alpha--linolenic acid (ALA) (Booyens, 1984). It seems that EPA is the direct metabolic precursor of eicosanoids of the 3-series, including the prostaglandin prostacyclin I_3 (PGI_3). EPA interferes with the balance between proaggregatory and vasoconstrictor thromboxane-A_2 and anti-aggregatory and vasodilator prostacyclin (PGI_3) and this could lead to less reactive platelets and lowered pressure reactivity of the vascular system. From the above it is clear that the protective effect of the omega-3 fatty acids against CHD, is probably mediated through many mechanisms.

It has been suggested that the consumption of as little as one or two fish dishes per week may be of preventive value in relation to CHD (Kromhout, et al.,1985). Before specific recommendations can be made about general use of fish oil supplements, further studies of their long-term efficacy and toxicity must be conducted (Ballard-Barbash and Callaway, 1987).

2.6.3.6 Trans fatty acids

This category of fatty acids is produced by the hydrogenation of vegetable oils; these oils are present in shortenings and margarine (Grundy et al., 1989). Few reports are available on the metabolic effects or fates of these fatty acids.

Trans fatty acids in the presence of cholesterol were reported to increase serum cholesterol levels of man only slightly less than a mixture of the saturated fatty acids, lauric acid and myristic acid (Vergroesen, 1972). It is argued that the adverse effects of trans fatty acids could be nullified provided sufficient linoleic acid is present in the diet (Houtsmuller, 1976). It was proposed that the levels of linoleic acid and trans fatty acids should be equal so that appropriate pairs of fatty acids in phospholipids could ensure proper fluidity of cellular membranes (Gottenbos, 1981; Kinsella, 1986).

2.6.4 Dietary cholesterol

2.6.4.1 Effect of dietary cholesterol on serum cholesterol levels

There is abundant evidence from studies in laboratory animals that dietary cholesterol raises serum cholesterol concentration in many species (Grundy and Denke, 1990). In primates the major effect of dietary cholesterol is an increase in the number of circulating LDL

particles (Rudel et al., 1985); the LDL particles also become larger in size and are enriched in cholesteryl esters.

However, in humans, the effects of dietary cholesterol on raising serum levels of TC and LDL-C are much less pronounced. Several studies in humans suggest that individual variability in the processing of dietary cholesterol may be a factor determining the serum cholesterol response (Quintao et al., 1971; Grundy, 1983; Grundy et al., 1989).

It has been suggested that the total component of the diet, the P/S ratio, amount of total fat and the presence of hypocholesterolaemic dietary fibre components, could modify or influence the effects of dietary cholesterol (Vorster et al., 1988b), because the effects of these components could be greater than the effect of dietary cholesterol. The scoring systems developed to predict serum cholesterol from dietary changes (Anderson et al., 1979; Keys, 1984; Hegsted, 1986) are based on intakes of total energy, saturated and polyunsaturated fat and cholesterol. These formulas do not incorporate a possible effect of hypocholesterolaemic dietary agents such as MUFAS (Grundy, 1987) and gel-forming dietary fibres (Vorster et al., 1988b). McNamara (1985) points out that the Keys (1984) model fails to address the relationship between dietary cholesterol intake, the fractional absorption of dietary and biliary cholesterol and any resultant change in serum cholesterol levels. The very large individual variation in absorption of cholesterol, between 20 and 85% of dietary cholesterol (McNamara, 1985), would render the scoring systems useless in predicting individual responses to diet.

However, many investigators have demonstrated that plasma cholesterol levels increase with increasing dietary cholesterol, at least up to a threshold of 300 to 600 mg/day (McGill, Jr., 1979). Increasing the cholesterol intake above this range appears to have little effect on plasma cholesterol levels, although individual responses vary considerably. It has been estimated that adding 250 mg/day of cholesterol to the usual American diet would produce an increase of 5 to 6 mg/dl (0.13-0.16 mmol/l)in plasma cholesterol concentration (Keys et al., 1974).

Although no association between dietary cholesterol intake and risk of CHD was found in some studies (Gordon et al., 1981; Dawber et al., 1982), the Honolulu Heart Program (McGee et al., 1984) and the Western Electric Study (Shekelle et al., 1981) demonstrated significant positive risk. Participants were followed for 19 years in the latter study in which, as mentioned by both Blum and Levy (1987) and Pyörälä (1987), the association between cholesterol intake and CHD risk was independent of other risk factors, even plasma cholesterol, suggesting that dietary cholesterol may affect risk through mechanisms other than effect on circulating cholesterol (reviewed by Gotto et al., 1990).

2.6.4.2 Oxidized cholesterol

Many oxidation derivatives of cholesterol have been found to be more potent than cholesterol itself in the inhibition of cholesterol biosynthesis by depressing activity of 3-hydroxy-3-methylglutaryl (HMG) CoA reductase, a rate limiting enzyme for cholesterol biosynthesis

(reviewed by Peng et al., 1979). A mixture of these oxidation compounds prepared from an aged cholesterol extract has proved to be cytotoxic in aortic smooth muscle cells (Peng et al., 1978) and angiotoxic as well as arteriosclerotic in rabbits (Imai et al., 1976). In contrast, purified cholesterol showed no toxic efects either in vitro or in vivo (Imai, et al., 1976; Peng et al., 1978). It has thus been hypothesized by Peng et al. (1979) that some auto-oxidation derivatives of cholesterol are probably the prime "cause" of atherosclerotic lesions and that the deposition of cholesterol and its esters is merely a secondary process. However, Smith and Van Lier (1970) showed that in atheromata, of man only, traces of 12 oxidation derivatives of cholesterol were found. Therefore, many more studies are needed to clarify the contribution of oxidized cholesterol to atherosclerosis.

The oxidation derivatives of cholesterol in conventional foodstuffs have not been comprehensively studied, but certain oxidation products have been found in dried egg yolks (Chicoye et al., 1968) and foods containing powdered whole milk (Acker and Greve, 1963).

For the preparation of typical Indian food dishes "ghee" (clarified butter) is recommended as cooking fat (McKeigue et al., 1985). It has been hypothesized that Indian "ghee" contains cholesterol oxides and could be an important source of dietary exposure to cholesterol oxides and an explanation for the high frequency of atherosclerosis in Indian migrant populations (Jacobson, 1987). This author showed that a substantial proportion of the cholesterol present in butter is oxidized

during preparation of "ghee" (12.3% of sterols). However, this work should be followed up with further research.

2.6.5 Dietary fibre

The term dietary fibre was first used by Trowell (1972) to describe a large number of plant cell wall constituents, naturally present in a complex botanical structure. Physiologically, dietary fibre has been defined as plant polysaccharides and lignin which are resistant to hydrolysis by the digestive enzymes of man (Trowell, et al., 1976; reviewed by Bingham, 1987). Chemically, dietary fibre can be classified into three major fractions (Southgate, 1986):-

Structural non-starch polysaccharides

- associated with the plant cell wall
- include non-cellulose polysaccharides (hemicellulose and some pectins) and cellulose

Structural non-polysaccharides

- predominantly lignin

Nonstructural non-starch polysaccharides

- include gums and mucilages secreted by plant cells
- include polysaccharides from algae and seaweed.

For simplicity and convenience, fibres can be grouped into two broad categories: water soluble and water insoluble. Soluble fibres include pectin, gums, certain hemicelluloses and storage polysaccharides. Insoluble fibres include cellulose, lignin and many hemicelluloses (Anderson, et al., 1987; Vinik and Jenkins, 1988).

Interest in the possible hypocholesterolaemic effect of dietary fibre started with the suggestion that high fibre diets may protect against CHD (Burkitt, 1969). It was later realized that soluble dietary fibre (non-starch polysaccharides) was responsible for the cholesterol-lowering effect of fibre (Tarpila et al., 1978; Kirby et al., 1981; reviewed by Miettinen, 1987; Grundy et al., 1989; Schweizer and Würsch, 1991).

The hypocholesterolaemic effect of high-fibre diets can be ascribed to a replacement effect (high-fibre foods replacing foods rich in energy, saturated fat or refined carbohydrates) or to a metabolic effect per se (Miettinen, 1987). The latter may be because of a direct effect on lipid metabolism by fibre interfering with lipid and bile acid absorption or because of an indirect effect as a result of effects on glucose absorption, insulin and glucagon responses and possibly also lipoprotein lipase activity and fatty acid metabolism (Miettinen, 1987).

The evidence of the hypocholesterolaemic effect of fibre comes from a large number of epidemiological studies involving populations with particular life-styles, experimental studies with high fibre diets, and studies in which diets were supplemented with fibre isolates (reviewed by Schweizer and Würsch, 1991).

From the literature it seems that a reasonable hypocholesterolaemic effect can be achieved by increasing fibre intake, ranging from 6-15% for high fibre foods such as oats and 10-30% for soluble fibre isolates (Topping, 1992).

Although many aspects of the original fibre hypothesis (Burkitt, 1969) remain controversial, the dietary fibre concept has gained enough credibility for health authorities to recommend an increase in fibre intake to a level substantially above the present consumption (Schweizer and Würsch, 1991).

2.6.6 Diet and blood coagulation

In addition to dietary effects on the conventional risk factors of CHD, the possibility that a prudent low-fat, high-fibre diet could protect against CHD through effects on the thrombotic system, should be considered.

Dietary oils and fats have long been thought to influence arterial thrombosis. The rather few experiments concerning the effects of dietary fats on arterial thrombosis suggest that saturated fats enhance thrombus formation, whereas unsaturated fats do not, or are even anti-thrombotic (Hornstra, 1974). The differences in thrombogenicity between sunflower oil (SO), hydrogenated coconut oil (HCO) and butterfat (BF) suggest that SO contains some antithrombotic factor which is missing in HCO and BF. The main constituents of SO are palmitic, stearic, oleic and linoleic

acids. The former three fatty acids occur in comparable amounts in both HCO and BF. Thus, it is tempting to speculate that linoleic acid is the antithrombotic fraction in SO. This is supported by the significant, positive relationship that exists between dietary linoleic acid content and thrombogenicity, no matter whether SO is the only dietary fat or whether it is mixed with HCO or BF (Hornstra, 1974).

Dietary fat intake has an acute influence on factor VII activity (Miller et al., 1986a) and on fibrinogen level (Pickart and Thaler, 1980). Dietary fat intake may therefore influence both thrombogenesis and atherogenesis, since factor VII has been shown to be one of the risk factors for CHD (Meade et al., 1986b). Furthermore, diets rich in protein and fat might increase VIIc, partly through the hepatic production of factor VII and partly through an effect of fat upon its activity state. The combined effect can be substantial (Miller et al., 1985; Miller, et al., 1986a; Scarabin, et al., 1986).

Thus, the possibility exists that dietary fat may predispose to CHD through its effects on the coagulation system, in addition to its well recognized effects on the plasma lipoproteins. It is suggested that this would help to account for the coexistence of fibrin and lipid in the evolving atherosclerotic plaque (Smith and Staples, 1981) and the interrelations between VIIc, plasma lipid concentrations, and risk of cardiovascular death (Meade et al., 1980; Miller et al., 1985).

A possible connection between dietary fibre intake and coagulation has

been indicated in the very limited literature on the subject (Vorster et al., 1988b). An epidemiological study in Wales demonstrated an independent inverse relationship between cereal fibre intake and fibrinogen concentrations (Fehily et al., 1982). Koepp and Hegewisch (1981) reported that a daily supplement of 0.45 g guar gum/kg body weight reduced mean plasma fibrinogen levels from 313 mg/dl to 263 mg/dl in diabetic children.

Experiments on various animal models indicated that the soluble dietary fibre component, konjac glucomannan, lowered plasma fibrinogen (Venter et al., 1990). However, more research is needed on the possible relationship between diet and coagulation.

2.6.7 Dietary factors and hypertension

2.6.7.1 Introduction

Epidemiological and clinical studies have produced ample evidence that dietary factors play an important role in the pathogenesis of hypertension in man (Huttunen and Tuomilehto, 1984; Huttunen et al., 1985). It has also been suggested that lacto-ovovegetarian diets may have a blood pressure lowering effect (Rouse et al., 1983; Margetts et al., 1986).

Specific dietary components that may influence the regulation of blood pressure, both in man and in experimental animals, include excessive

energy intake, the quantity and quality of dietary fat, dietary sodium (Na), potassium (K), calcium (Ca), magnesium (Mg) and alcohol (Düsing et al., 1984; Huttunen and Tuomilehto, 1984; Iacono et al., 1984). The effect of each dietary factor on hypertension is discussed below.

2.6.7.2 Polyunsaturated fatty acids

Dietary polyunsaturated fatty acids (PUFAT), i.e. eicosapentaenoic acid and docosahexaenoic acid, have been shown to lower blood pressure (Iacono et al., 1975; Oster et al., 1980; Vergroesen et al., 1980; Iacono et al., 1981a; Iacono et al., 1981b; Judd et al., 1981; Iacono et al., 1982a; Iacono et al., 1982b; Iacono et al., 1983; Singer et al., 1985). It has also been reported that diets containing low fat and a high polyunsaturated to saturated fat ratio (P/S) reduced both systolic and diastolic blood pressure as was demonstrated in normotensive males and females when free-living families in rural North Karelia, Finland, adopted a low-fat, high P/S diet (Iacono et al., 1983). The mechanisms by which blood pressure may be affected by changes in dietary fat intake remain unclear.

2.6.7.3 Energy

It has been stated that excessive energy intake may be the single most important nutritional factor in the pathogenesis of hypertension (Huttunen et al., 1985). An association between weight and hypertension has been observed in most populations in both primitive and acculturated

societies. Therapeutic weight reduction in obese hypertensive subjects lowers blood pressure, and the effect is evident in both sexes, in younger as well as in older patients, at all levels of blood pressure and in moderately overweight as well as in the very obese (Dahl et al., 1958; Reisen et al., 1978; Stamler et al., 1980; Tuck et al., 1981; Fagerberg et al., 1984). A variety of mechanisms have been offered to explain the pathogenesis and pathophysiology of hypertension in obesity (Frohlich et al., 1984). These include: (a) expanded blood volume, (b) elevated cardiac output associated with the volume expansion, (c) associated increased dietary sodium intake and increased vascular responsiveness to sodium, and (d) increased adrenergic participation in obesity-related hypertension (Frohlich et al., 1986).

2.6.7.4 Sodium

The epidemiological, clinical and experimental evidence which links excessive salt or sodium intake to hypertension has been the subject of many reviews (Freis, 1976; Bruce, 1984; Hegsted, 1991). Studies in which 24-hour urine excretions were used to quantify sodium intake demonstrated positive and highly significant correlations between sodium intake and both systolic and diastolic blood pressure in men and women (reviewed by Elliot, 1991). This association is stronger in instances of low calcium and high alcohol intake (Hamet et al., 1991).

Although there are those who argue that the beneficial effects of a lowered salt intake on blood pressure in a population will be small

(Heagerty, 1991), most dietary guidelines in the western world advise that salt intake should be reduced (Truswell, 1987). Hegsted (1991) mentions that there is no evidence that salt intake is beneficial, and that there is little likelihood that identification of the so-called "salt-sensitives" in a population will be successful. He advises that because it would be difficult for individuals to modify their diets, food manufacturers should be persuaded to reduce salt in the food supply - especially in tinned, dried and frozen "fast-food" products.

In many Western countries the actual level of salt intake is 150-200 mmol/d. It has been suggested that a gradual lowering of salt intake to a desirable level of about 100 mmol/d (6 g NaCl) would be beneficial to reduce hypertension, as has been observed in Belgium (Isaksson and Brubacher, 1986).

2.6.7.5 Potassium

It was first suggested by Addison (1928) that a diet high in potassium could exert an antihypertensive effect. This resulted in potassium being linked to the development of arterial hypertension (Luft and Weinberger, 1987).

Epidemiological data from two neighbouring northern Japanese villages (where salt intake is very high and there is a correspondingly high prevalence of hypertension) in which individuals ingested similar amounts of sodium, a lower blood pressure was identified in the village with the higher potassium intake (Sasaki, 1962).

Whereas correlations between blood pressure and sodium excretion have been very difficult to demonstrate within populations in the Western world, several investigators in the United States and Sweden have found a significant correlation between urinary sodium: potassium (Na:K) ratio and blood pressure (Watson et al., 1980; Ljungman et al., 1981).

A low-sodium, high-potassium diet has been associated with a decrease in resting blood pressure as well as an increase in baroreceptor activity (Mickelson et al., 1977). The blood-pressure-lowering effects of potassium have been variously attributed to a diuretic action, altered activity of the renin-angiotensin system, a direct alteration of peripheral resistance, the antagonism of the effects of a natriuretic hormone, and central or peripheral nervous system effects (Luft and Weinberger, 1987).

2.6.7.6 Calcium

A link between dietary calcium and hypertension was initially suggested on the basis of reports of an inverse relation between the level of water hardness and the prevalence of cardiovascular disease or hypertension in some regions of the United States and Great Britain (Stitt et al., 1973). Analysis of the Health and Nutrition Examination Survey showed that those individuals with blood pressure more than 160/95mm Hg consumed significantly less calcium than subjects with lower blood pressure (McCarron et al., 1984). Of the 17 nutrients examined in this study, differences in calcium intake most consistently separated normotensive and hypertensive subjects (Huttunen et al., 1985).

The mechanisms through which calcium intake could influence blood pressure are not clear. Blaustein (1977) hypothesises that calcium ions, through myofilament binding, are the immediate trigger for vascular muscle contraction, and that sodium ions play a critical role in the maintenance of calcium balance in vascular smooth muscle. It is possible that changes in serum calcium concentration and urinary calcium excretion observed in hypertensive subjects are secondary to abnormalities in renal tubular function, in the release of parathyroid hormone or in differing membrane ion pumps responsible for steady-state disposition of calcium across cell membranes. Intracellular free calcium levels have been reported to be increased in platelets from patients with hypertension (Erne et al., 1984).

It has been suggested that supplementation of 1g/day of elemental calcium may lower blood pressure in normotensive young adults (Belizan et al., 1983).

2.6.7.7 Magnesium

It has been indicated in studies from Australia (Parsons et al., 1961) and Canada (Neri et al., 1975) that the overall pattern of death from CHD seemed to be a function of magnesium intake. In various parts of the world, the annual death rate from CHD has been shown to be a direct linear function of the dietary calcium/magnesium ratio (Karppanen et al., 1978; Karppanen, 1986). The evidence that waterborne magnesium could serve as a protective factor against CHD is developed in Marier's review (Marier, 1982).

The association between magnesium intake and hypertension could possibly be explained by the fact that magnesium may be an important physiological regulator of vascular tone and reactivity (Ryan and Brady, 1984). The extracellular concentration of magnesium is important in the control of coronary arterial tone via regulation of vascular membrane magnesium-calcium ion exchange sites (Altura and Altura, 1982).

Nuts, cereals and sea foods are rich in magnesium (Mg), while sugar and fat are very poor sources (Wester, 1987). On a calorie basis vegetables are good sources of magnesium, especially green leafy vegetables which are rich in the Mg-containing chlorophyll. Among beverages, coffee, tea and cocoa are rich in Mg, while hard liquors are very poor. The amount of Mg in drinking water may vary widely from almost 0 to > 15 ug/ml (Schroeder, 1971). Refining and cooking of foods may diminish the Mg content substantially, e.g. refining whole wheat to patent flour, polishing rice, refining sugar and boiling vegetables. Thus, the magnesium intake of humans has possibly declined during the past few decades.

2.6.7.8 Alcohol

The association between high alcohol consumption and raised blood pressure was first described in 1915 (Lian, 1915). Over the last few decades confirmatory evidence has been obtained from population and clinical studies (Dawber et al., 1967; D'Alonzo and Pell, 1968; Beevers, 1977). Genetic factors may be important in the pathogenesis of hypertension related to alcohol consumption (Potter and Beevers, 1984).

However, there is much less consensus as to the question of the level at which alcohol consumption has a hypertensive effect (Veenstra, 1991). Some studies have found a continuous increase from no alcohol use to heavy use (Arkwright et al., 1981; Cooke et al., 1983; Cairns et al., 1984; Ueshima et al., 1989). In other studies (Gyntelberg and Meyer, 1974; Harburg et al., 1980; Harlan et al., 1984) a J-shaped relationship between alcohol use and blood pressure was found, in particular for women.

An investigation of the interrelationship of psychological assessment and alcohol consumption on blood pressure showed that alcohol had an independent pressor effect on systolic blood pressure and that this effect was enhanced in introverted subjects (Arkwright et al., 1982; Arkwright et al., 1983).

Ethanol causes a diuresis during acute intoxication but this is often followed by a period of antidiuresis. Water and sodium retention have therefore been postulated as two mechanisms by which alcohol may raise blood pressure (Potter and Beevers, 1984). It is however not clear whether this mechanism explains the total effect of alcohol intake on blood pressure (Linkolä et al., 1978).

2.7 Prevention of Coronary Heart Disease

2.7.1 Introduction

The possibility that CHD may be prevented by reducing the incidence and

levels of the risk factors associated with the disease, has attracted much attention in the literature.

It has been recommended that the detection of those at high risk of CHD could be accomplished by screening all individuals with a family history of hypercholesterolaemia or CHD in a parent before the age of 60 years (Glueck, 1986). The risk factors that are alterable and treatable in childhood include plasma cholesterol level (LDL-C), smoking, sedentary life-style, diet, obesity and diabetes mellitus (Editorial, 1986).

To establish the effectiveness of primary prevention through modification of risk factors, a series of important experimental or clinical trials are being conducted in the United States and other countries (Levy et al., 1989). Previously reported secondary prevention trials attempted to modify risk factors after a heart attack or stroke in order to reduce the risk of a second heart attack or death after the initial attack (Coronary Drug Project Research Group, 1973). Primary prevention trials select subjects who are free of clinical heart disease or stroke at entry to the study. Some of these trials will be discussed briefly.

2.7.2. Risk factor intervention trials

Primary prevention trials may address a single risk factor of CHD (unifactorial trials) or could try to reduce as many risk factors as possible (multifactorial trials).

Most of the primary single risk factor intervention trials are double-blinded and include a pharmacologic agent to lower either cholesterol or blood pressure compared with a placebo. Many of these studies are further limited to very high risk subjects with plasma cholesterol levels in the highest 10-15% of the population that are difficult or impossible to modify by dietary intervention (Davis and Havlik, 1977). However, the benefits of cholesterol lowering appeared to be equal regardless of whether drug or diet was used and whether fatal or non-fatal coronary events were considered (Yusuf et al., 1988). Stamler (1985) reviewed some of these unifactorial primary prevention trials, i.e.:

° Trials of fat modified diets :- (a) Los Angeles Veterans Administration Domiciliary Facility Study; (b) Finnish Mental Hospital Trial; (c) New York Anti-Coronary Club; (d) Minnesota Mental Hospitals Trial; (e) National Diet-Heart Study.

° Trials of Antihypercholesterolaemic Drugs:- (a) Clofibrate; (b) Colestipol; (c) Cholestyramine - the Lipid Research Clinics Coronary Primary Prevention Trial; (d) Other recent drug studies, eg. combinations of drugs that are highly effective in markedly lowering and often normalizing severe hypercholesterolaemia in persons with heterozygous familial hyperbetalipoproteinaemia. This has been demonstrated with nicotinic acid and colestipol (Kane et al., 1981; Illingworth et al., 1981) and with the cholesterol synthesis blocker compactin plus resin (Mabuchi et al., 1983). However, no data are available on the long-term benefit to risk ratio of these drug combinations for such high risk persons.

- Unifactor Trials of Exercise.
- Unifactor Trials of Smoking Cessation - Whitehall Trial.
- Unifactor Trials on Reducing Psychosocial Stresses and Incongruent Behaviour Patterns.

Multifactorial primary intervention trials include the following (Stamler, 1985):

- Chicago Coronary Prevention Evaluation Program,
- Oslo Primary Prevention Trial,
- Multiple Risk Factor Intervention Trial,
- European Factory-based Multifactorial Primary Prevention Trial,
- University of Southern California Clinical Research Study,
- WHO Multifactorial Trial in Factories.

The primary hypothesis in these studies is that the reduction of the risk factors will reduce the incidence of, and mortality from heart attack (Kuller, 1980). It is stated that the successful test of this hypothesis requires that subjects or groups should be successfully recruited and categorized at entry to the study, and that a very high percentage be successfully followed for the duration of the study.

The most basic problem of a controlled intervention trial is one of feasibility: in particular the difficulty of keeping two groups comparable for years in all other aspects of life-style, except the one being tested (Stamler, 1985).

Thus, the thrust of medical care and public health strategy for CHD primary prevention for the 1990s must be based on judgement assessment of the data currently on hand, from the trials already done, and from the epidemiological, (clinical, pathologic and animal experimental) studies that have produced the vast data base (Stamler, 1985).

It has been stated that the purpose of intervention studies is to yield estimates of the degree to which CHD is preventable (Epstein and Pyörälä, 1987). Unifactor trials aimed at primary prevention through serum cholesterol lowering have uniformly shown a benefit in favour of the treated group, and those in which only dietary intervention was used there was a lowering of serum lipids. In all of the seven major primary prevention trials concerned with anti-hypertensive treatment by using drugs, treatment in mild hypertensives reduced cardiovascular complications by between 20 and around 50%. However, the emphasis of intervention has shifted from high-risk groups to projects in whole communities and the entire population. Thus, for example, in the Multiple Risk Factor Intervention Trial (MRFIT) which is the prime example of a randomized study on high-risk volunteers (MRFIT Research Group, 1982), the results did not contribute to answering the question of whether preventive measures are effective in reducing the risk of CHD. The same conclusion applied to the Göteburg Study (Wilhelmsen et al., 1986), carried out among high-risk persons representative of the community. This was because risk factors were lowered to a similar degree in the treatment and comparison groups in both these studies.

However, as a whole, multifactorial trials lend support to the view that intervention aimed at lowering the major CHD risk factors will lead to a reduction in the risk of the disease itself.

2.7.3 The role of diet in prevention of coronary heart disease

Until World War II the scientific community had largely neglected the idea that CHD, which might, at least partly, be a nutritional disease and hence, could be prevented by dietary means (Turpeinen, 1979).

Since then, most efforts to demonstrate a possible role for diet to prevent CHD, concentrated on effects of dietary fat intake on serum lipids and lipoproteins.

A number of therapeutic trials in healthy and hypercholesterolaemic subjects have demonstrated that reductions in dietary cholesterol, total fat, or both, or an increase in polyunsaturates, could produce a significant decrease in serum cholesterol (McGandy et al., 1972; Miettinen et al., 1972; Stein et al., 1975; Levy et al., 1989). The efficacy of a reduction in dietary cholesterol alone to reduce serum cholesterol levels in free-living conditions has been questioned recently (Slater et al., 1976; Kummerow et al., 1977; Porter et al., 1977; Whyte et al., 1977; Flynn et al., 1979; Vorster et al., 1987; Vorster, et al., 1992).

Also, of importance is the observation that both plasma HDL-C and/or plasma apolipoprotein A, can be increased by dietary cholesterol and saturated fat feeding (Tan et al., 1980) and decreased by diets containing large amounts of polyunsaturated fat (Hjermann et al., 1979; Brussaard, 1980; Ernst et al., 1980; Tan et al., 1980; Vessby et al., 1980; Grundy, 1992). Thus, the response of HDL and subfractions to dietary cholesterol and to the amount and type of fat is still uncertain. However, studies that have reported decreases in HDL have been of short duration (Shepherd etal., 1978; Ernst et al., 1980; Vessby et al., 1980) involving small numbers of subjects (Shepherd et al., 1978; Vessby, et al., 1980), and have often entailed extremely high dietary polyunsaturated to saturated (P/S) fat ratio (Shepherd et al., 1978).

However, in spite of the controversy surrounding dietary modification of cholesterol and fat intake for the general population, and especially for children, there is general agreement that dietary modification remains the first step in treatment of both adult and paediatric subjects with primary or familial elevations of LDL-C (Stein et al., 1982). Difficulty in compliance with the dietary total and saturated fat modifications has resulted in limited general acceptability of the recommendations. Nevertheless, there has been a steady decrease in the consumption of dietary cholesterol, especially eggs (Statistical abstract of the United States, 1979).

An increased intake of dietary fibre, especially soluble fibre, has also been shown to reduce total and LDL cholesterol levels (see 2.6.5). Among research workers there is broad agreement that the type of diet that is least likely to cause diseases such as CHD, hypertension and type 2 diabetes, is one that provides a high proportion of calories in whole grain cereals, vegetables and fruit; limited amounts of animal protein, mainly from fish and poultry; limits the intake of fats and, if oils are to be used, gives preference to liquid vegetable oils; includes less dairy products, eggs and refined sugar; and is sufficiently restricted in amount so as not to cause obesity (Doll, 1982).

2.8 Regression of Atherosclerosis

From the above it is clear that primary prevention of CHD is possible by reducing the levels of known risk factors by medication, diet, or other life-style changes. An important question is whether reversal of established atherosclerotic lesions by these interventions is possible.

The view that human atherosclerosis might be reversible has been strongly supported by a number of observations on experimental atherosclerosis of animals and on cultured arterial cells (Nikkilä, 1978). In the most detailed studies of regression of coronary atherosclerosis in rhesus monkeys, it was noted that after 40 months on regression diets, the stenosis of coronary arteries decreased, as did the content of cholesterol and cholesteryl esters in atherosclerotic plaques (Armstrong

et al., 1970; Armstrong and Meagan, 1972). One study showed that severity of atherosclerosis and hypercholesterolaemia in cholesterol-fed rabbits could be decreased by ileal bypass surgery (Clarkson et al., 1973). Similar results were obtained in rabbits (Okuboye et al., 1968), in dogs (Scott et al., 1966), and the rhesus monkeys (Scott et al., 1967).

Over twenty clinical trials are presently being carried out to determine whether the progression of coronary atherosclerosis can be modified; in all of these, cholesterol-lowering drugs, plasmapheresis, or partial ileal bypass surgery are the primary interventions (Arnetzius, 1989).

The 1987 Cholesterol Lowering Atherosclerosis Study data (Blankenhorn et al., 1987) has shown that drug and dietary intervention to lower blood cholesterol level not only slowed the progression but also produced regression of coronary atherosclerosis in men with coronary bypass grafts.

The Life-style Heart Trial, which is the first randomised, controlled clinical trial to determine whether patients outside hospital can be motivated to make and sustain comprehensive life-style changes and, if so, whether regression of coronary atherosclerosis can occur as a result of life-style changes alone, has shown that this can be achieved (Ornish et al., 1990).

2.9 Recommendations for prevention of coronary heart disease

2.9.1 Introduction

The modification of risk factors is the major clinical and public health approach to the prevention of CHD (Ernst and Cleeman, 1988). In the USA, in order to coordinate national efforts to reduce the three major modifiable CHD risk factors, ie. high blood pressure, cigarette smoking, and high blood cholesterol, the National Heart, Lung and Blood Institute (NHBLI) established three educational programmes in 1972, namely, the NHBLI Smoking Education Program, the National High Blood Pressure Education Programme, and the National Cholesterol Education Program.

Their approach was to create awareness, increase knowledge and provide guidelines on who should be treated, when treatment should be offered, and what treatments are successful (Ernst and Cleeman, 1988). Subsequent recommendations have reflected the publication of major clinical trial results and the introduction of new anti-hypertensive agents, as well as evidence concerning effectiveness of nonpharmacologic treatment and further analysis of the database relating blood pressure levels to the risk of premature morbidity and mortality (Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure, 1977; The 1980 Report; 1984 Report).

Thus, from the early 1970s to 1980 the proportion of hypertensive individuals who have their blood pressure under control has more than

doubled (The 1980 Report). Also, the recognition of the high prevalence of high blood pressure and the cost and side effects of drugs has promoted increased interest and research into the role of diet in the management of high blood pressure - particularly mild hypertension in which diastolic blood pressures are in the range of 90 to 104 mmHg, and this has been reviewed by the Sub-committee on Nonpharmacologic Therapy of the 1984 Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (1986).

In a recent WHO Expert Committee Report, Prevention in Childhood and Youth of Adult Cardiovascular Diseases : Time for Action (1990), it was recommended that intervention against adverse levels of blood lipids and blood pressure and the use of tobacco must begin in youth if these risk factors are to be controlled to maximum benefit in adults on a population basis. Another recommendation was that countries develop and pursue a comprehensive population strategy for the primary prevention of CHD as part of their long-term national health development plan (Labarthe and Rockwell, 1992).

2.9.2 Modification of blood cholesterol levels to reduce coronary heart disease risk

2.9.2.1 Population strategy

It has been demonstrated that a mean population cholesterol level above 160 mg/dl (4.14 mmol/l) is a prerequisite for epidemic cardiovascular

disease (Breslow et al., 1989), and, although cholesterol elevation appears necessary for mass coronary disease, risk is potentiated by other factors such as hypertension, cigarette smoking, obesity, and family history.

In spite of a continuous controversy regarding the relationship between serum cholesterol levels, diet and CHD (Reiser, 1978; Steinberg et al., 1989), many investigators believe that the average cholesterol level for the whole population should be as low as possible (Grundy, 1986). The question of what constitutes an ideal plasma cholesterol level was addressed by a group of epidemiologists, clinical investigators, and experimental pathologists, who proposed that ideal cholesterol levels for adults should range from 130 to 190 mg/dl (3.36 to 4.91 mmol/l) (mean, 160 mg/dl [4.14 mmol/l]) (Stamler, 1979). The MRFIT data support this view because the range is below the zone of markedly increasing risk (Grundy, 1986).

Thus, for the sake of simplicity, the cholesterol concentration should ideally be below 200 mg/dl (5.17 mmol/l), although it should be emphasized that even below this value cholesterol levels and coronary risk are positively correlated (Grundy, 1986). It is stated that a large fraction of the population with hypercholesterolaemia has this disorder as a result of excessive intake of saturated fatty acids, cholesterol, and total calories, and that the use of diet modification as a first line of therapy for hypercholesterolaemia is resonable (Grundy et al., 1989).

Some investigators, however, have suggested that the response to dietary therapy may be inadequate for most patients with hypercholesterolaemia, and physicians will, therefore, have to resort to drug therapy in the majority of the patients to achieve the goals for LDL cholesterol lowering.

From the literature it is clear that a reduction in fat intake remains the major dietary recommendation to reduce cholesterol levels. In several countries where low-fat, high-carbohydrate diets are consumed, CHD rates are relatively low, and thus the concept has evolved that the prevalence of CHD among Americans would be reduced if the intake of total fat could be decreased (Grundy et al., 1989). The American Heart Association (AHA) has recommended that the American public decrease its fat intake from the current 37-40% of calories to 30% of calories or less. It is also advocated that the recommended 30% of total daily calories from fat be divided equally between PUFAT, MUFAT and SATFATs, ie. 10% of each. It is argued that this recommendation would help promote weight reduction in obese American citizens by reducing the intake of fat-rich, calorie-dense foods, and it would facilitate a decreased intake of saturated fatty acids and cholesterol, both of which are frequently associated with high-fat intakes.

In a report of the British Cardiac Society (BCS) (1987) Working Group on Coronary Heart Disease prevention, it was agreed that both population and high risk approaches are complementary and should be implemented simultaneously (Hart, 1987). The recommendations from the Panel on Diet

in Relation to Cardiovascular Disease (COMA) (1984) were endorsed; the unacceptability of high CHD rates should be reduced by action directed at reducing smoking, reducing dietary fat, and increasing exercise in the population. Dietary advice should be given to those with blood cholesterol concentrations above 6.5 mmol/l (250 mg/dl) and drug therapy may be required for levels above 7.8 mmol/l (300 mg/dl), while the aim would be to lower total cholesterol towards 5.2 mmol/l (200 mg/dl) (Anon, 1987).

However, it was suggested that a policy of reducing fat intake to 30% or less of dietary energy should apply only to populations such as those of Northern Europe and the United States where most of the fat in the diet comes from meat and dairy products (Keys, 1987). In Mediterranean areas many diets provide more than 30% of energy from fats and CHD is much less common than it is in Northern Europe and the United States of America. The peculiarity of the diets in Greece and other Mediterranean areas (Italy, French Provence, Spain and Portugal) is that they are low in saturated fats but not in total fat as olive oil is predominantly used in place of fats of animal origin (Keys, 1987).

A successful nutrition strategy to lower serum cholesterol levels will require implementation through clinical practice, government agencies, and food industries. It should include education, suitable food labelling, availability of nutritionally desirable food options in restaurants and institutions, and policy measures by national and supranational organizations (Lewis, 1987). The implementation of these

recommendations will be enhanced if they are endorsed by physicians, and particularly by cardiologists.

2.9.2.2 Lowering cholesterol levels of children

The BCS report (1987) also raises the important issue of dietary recommendations for children. It is stated that the need to avoid heart disease must be clearly balanced against the risk of inadequate nutrition (Anon, 1987).

The diet composition for the infant, child, and adolescent, the distribution of fats in the diet, the diagnosis and treatment of the child with a family history of early atherosclerosis, as well as the recommendations of the AHA pose several problems for the paediatrician (Barness, 1986; Kwiterovich, 1986). It is stated that since membrane formation and particularly nerve lipids require cholesterol, limiting this substance may not be without risk in infants (Barness, 1986). Similarly, hormone formation uses cholesterol as a precursor, and this may be important in the adolescent. Thus, until more direct ill effects of dietary cholesterol are noted, avoiding those foods that are high in cholesterol but that provide other benefits seems unreasonable. It seems appropriate to follow the total percentage of fat found in human milk, ie. 40% to 50% of calories as fat and about 150 mg/dl (3.87 mmol/l) of cholesterol, in the first year. The recommendation that fat be restricted to 30% of the calories seems to carry no risk after the first, or the second year.

In the first two years growth rate is maximum and thus care should be taken to ensure a normal growth rate without producing undernutrition or obesity. It has been found that a higher monounsaturate concentration in the diet gave a higher HDL level in adults (Grundy, 1986). Thus, it seems that a more appropriate distribution of dietary fatty acids should be saturates: monounsaturates: and polyunsaturates, in a ratio of 2:3:1 (Barness, 1986). Human milk contains appreciable amounts of n-3 acids and their long-chain products such as eicosapentaenoic acid and docosa-hexaenoic acid (Lands, 1986). These products may inhibit arachidonic acid, leukotriene, and prostaglandin formation (Dyerberg, 1986) and inhibition of these products may be associated with decreased blood clotting.

2.9.3 Modification of other risk factors

2.9.3.1 Obesity

For the maintenance of ideal body weight (i.e. an isoenergetic diet), the consumption of simple sugars (glucose, lactose, frutose, sugar) alcohol and saturated fats should be limited as much as possible (Vergroesen et al., 1978). Starch-containing products such as potatoes, rice and beans, vegetables and fruits are useful sources of minerals and vitamins, while most vegetable oils (except coconut, palm kernel, palm oil) such as soybean, sunflower, corn, safflower, ricebran oil are good sources of linoleic acid and vitamin E.

2.9.3.2 Oxidation of low density lipoprotein cholesterol

In recent years there has been much interest in the potential role of the antioxidants vitamin E, vitamin C, and beta-carotene in the prevention of atherosclerosis (Gey et al., 1991; Wong, 1991) by protecting cells from oxidative destruction. Rapid oxidation of LDL occurs only when the LDL molecule is depleted of its vitamin E and carotenoid content. Oxidatively modified LDL molecules contribute to atherogenesis in a number of ways: they attract monocytes into the subendothelial space; they are recognized by scavenger receptors; they inhibit the motility of the macrophages, trapping them at the site of the lesion; and they have cytotoxic properties which may contribute to endothelial injury.

2.9.3.3 Fibrinolytic activity and coagulation

Scattered reports suggest that individual foods may promote fibrinolysis, for example, onions, but a systematic re-examination of this suggestion is required (Truswell, 1985). Also, because of the high correlation of fibrinogen value with major cardiovascular disease risk factors, measures recommended to control CHD may also reduce fibrinogen level (Kannel et al., 1987). Already, there is evidence that quitting smoking reduces fibrinogen levels, but more direct experimental evidence is needed to determine whether weight reduction, cigarette smoking abatement, bloodletting, diabetes control, or treatment of hypertension will reduce fibrinogen level (Kannel et al., 1987b).

2.9.3.4 Physical activity

Individual choice of healthy life-styles is a key factor in maintaining and promoting health and emphasis should be placed on the positive actions that individuals and communities can take to protect and promote health. Promotion of physical exercise for health by WHO is facilitated by the current fashion for self-care and fitness, jogging, physical training, etc., which shows that new sportive lifestyles for everyone are spreading across geographical and cultural frontiers (Stroot, 1989).

2.9.4 Coronary heart disease prevention in South Africa

In South Africa, mean total fat intakes (24-hour recall method) of a subsample of the Coronary Risk Factor Study (CORIS) Afrikaans-speaking rural population varied from 35% to 37% of daily energy intake for different age groups (Wolmarans et al., 1988). Dietary polyunsaturated/ saturated fatty acid ratios varied from 0.48 to 0.59, while cholesterol intakes ranged from 243 mg/day to 500 mg/day and when expressed per 4.2 MJ (1 000 kcal) were similar for males and females. Although the results of this intrapopulation cross-sectional study showed no significant relationship between dietary variables and total serum cholesterol and HDL-C, the low percentage of respondents who complied with the prudent dietary guidelines add dietary risk factors to the already high prevalence of other major CHD risk factors in this population.

Thus the Heart Foundation has recognized the fact that the food industry is a powerful nutrition educator for the public (de Ruiter, 1987). It is stated that the ultimate purpose of disclosing nutrition information in the market-place is to help consumers assess nutrition value and to guide them towards health-promoting food purchases (Muller, 1984). The Heart Foundation decided to work with the food industry and to find and spread incentives to promote the production of food products with recognized health value, such as those with low fat, low kilojoule, low salt, high fibre and high natural vitamin and mineral content. This is considered essentially important at the initial stages of a national CHD prevention programme (de Ruiter, 1987).

However, it is stated that to be successful a dietary strategy must also be realistic (Rossouw, 1987). Red meat will always be a component of the South African diet, and there are sound nutritional reasons why this would in fact be desirable, as the hazard of the red meat lies in the overconsumption of fatty red meat, not in moderate consumption of lean meat. It is quite possible to stay within the prudent diet guidelines and eat a 90 g (cooked weight) portion of red meat four times a week at main meals, and a further four 30 g portions at light meals (Rossouw and Wolmarans, 1986).

The public health challenge is to provide positive health that will reduce the number of deaths from CHD, which has become the major scourge of develped countries. Adequate nutrition is a pre- requisite for this new approach that can add life to years and not merely years to life (Bannister, 1989). The dietary recommendations of the South African Diet

Consensus Panel (Dietary recommendations for the prevention of CHD) (1989) could be carried out to achieve this aim.

2.10 Conclusions

The focus of the WHO (1990) report on youth (age range birth to 24 years) was on existing knowledge of the vascular pathology of atherosclerosis beginning in advance of adulthood. The proneness of South African Indians to this multifactorial disease has been identified in some studies. CHD risk factor levels have been assessed in randomized family studies.

However, no intervention programmes have been initiated amongst South African Indians. There is an opportunity for primary prevention, or the avoidance of the social and environmental conditions which contribute to the development of their high CHD risk, i.e. if dietary modifications are introduced timeously.

Very little information is available re: nutrient intakes and risk factor levels in Indian adolescents among whom intervention against adverse levels of CHD risk factors should be initiated.

Therefore, the present study was undertaken to determine the habitual nutrient intakes of Indian adolescents as well as levels of CHD risk factors and to investigate possible correlations between dietary intakes and risk factor levels.

CHAPTER 3

STUDY DESIGN, SUBJECTS, MATERIALS AND METHODS

3.1 Introduction

General methodology and details of experimental methods are discussed in this chapter. The study design used was basically that of a descriptive cross-sectional epidemiological survey in which the levels of and association between various risk factors for CHD and nutrient intakes were measured in a homogeneous group of randomized subjects:

(1) Nutrient intakes were assessed

(2) Certain CHD risk factor levels were assessed

(3) Correlations were made between (1) and (2).

The researcher was part of a team, consisting of doctors, nurses, physiologists and nutritionists from the Carbohydrate and Lipid Research Group of the University of the Witwatersrand, and the Nutrition Research Group of the Potchefstroomse Universiteit vir Christelike Hoër Onderwys (PUCHO). The researcher organized the visits to the schools. All interviews were conducted by the researcher (habitual dietary intake questionnaire and the CHD risk factor survey questionnaire) as well as anthropometric and blood pressure measurements.

The measurement of the plasma coagulation factors were carried out by the

researcher in the Physiology Department of the PUCHO in Potchefstroom. The determinations of the serum lipids were made by the routine analyst at the Department of Chemical Pathology of the University of the Witwatersrand. However, the researcher analysed the lipids in 20 duplicate serum samples at the PUCHO. Correlation coefficients of results of the two laboratories on these samples were 0.99 ($p = 0.0001$) for total cholesterol, 0.98 ($p = 0.0001$) for triglycerides, 0.94 ($p = 0.0001$) for low density lipoprotein cholesterol and 0.85 ($p = 0.0001$) for high density lipoprotein cholesterol (see Table 3.1).

Table 3.1: Correlations between serum lipid results obtained in the two laboratories (n = 20)

Variable	University of the Witwatersrand		University of Potchefstroom		Pearson Correlation Coefficients*
	Mean	± SD	Mean	± SD	r
Total cholesterol (mmol/l)	4.20	0.55	4.60	0.66	0.99
Triglycerides (mmol/l)	0.80	0.24	0.94	0.24	0.98
LDL cholesterol (mmol/l)	2.58	0.50	2.93	0.62	0.95
HDL cholesterol (mmol/l)	1.25	0.23	1.26	0.22	0.85

* Calculated with the SAS(R) (1985) package. All p-values were ≤ 0.0001

3.2 Subjects

3.2.1 The population

Lenasia is the largest Indian group area in the Transvaal with a population of about 110 000. The area is 50 square kilometers and is situated 25 kilometres to the south-west of the Johannesburg central business district area. (Demographic data on Lenasia and its inhabitants is given in Appendix G.) The population from which the experimental sample was randomly selected, comprised all volunteers in their matric year in Lenasia. During 1988 the number of pupils in the matriculation class was 693. This was ascertained by questionnaire (Table 1, Appendix G) from each of the six high schools in the area by the researcher during late 1987 and early 1988.

After explaining the study design and procedures, each matriculant was asked to obtain parental/guardian consent to participate in the three-phased study programme.

The three phases were:

(i) Obtaining blood samples.

(ii) Anthropometric measurements and completing CHD risk factor survey questionnaire.

(iii) Dietary intakes and blood pressure measurements.

The researcher visited each of the six schools for the collection of the informed consent forms (Appendix D).

3.2.2 Ethical considerations

Permission to carry out this study was obtained from the Ethics Committees of the University of the Witwatersrand, the PUCHO, and from the Department of Education and Culture of the House of Delegates.

3.2.3 Randomization procedure - selection of sample

The religion, gender and dietary regimen (vegetarian/non-vegetarian) of each pupil was determined (Appendix C). Of the 693 pupils, 544 (79%) consented to participate. Of the 149 non-participants, 94 (63%) did not return the forms while 55 (37%) refused consent.

Reasons given for non-participation were:

(i) Some pupils were unwilling to make time available for the three phases of this study during the latter half of the matriculation year;

(ii) among adolescents, the indication of a reluctance to be examined may reflect general adolescent behaviour and developing independence (Berenson *et al.*, 1982);

(iii) some subjects feared venepuncture;

(iv) fear of learning the results and their implications in regard to careers, economics and insurance covers.

The data obtained from volunteers was processed by the Department of Biostatistics of the Medical Research Council at the University of the Witwatersrand. From this data, 300 subjects were randomly selected by a computer programme. The number of males and females selected were about equal. A number of reserve subjects was also selected to replace absentees on the days of venepuncture. However, all subjects as well as the reserves were available, resulting in 321 participating subjects.

Of the 321 randomly selected subjects, 148 (46%) were Muslims, 142 (44%) Hindu and 31 (10%) Christians. Of the 142 Hindu subjects, 31 (22%) were vegetarian. The religious distribution of this adolescent population is different from that of the general Indian population (Appendix A) in South Africa as reported by Arkin et al. (1989), based on the 1980 census, i.e. 62.38% are Hindu, 18.79% are Muslim, and 12.49% are Christians and 6.34% follow other religions. The dietary regimens (vegetarian/non-vegetarian) followed by this population, can probably not be extrapolated to the general population, as many more Hindu adults may be following the various vegetarian regimens.

3.2.4 Organization of the study

All subjects were addressed by the researcher during the school assembly one week prior to their blood sampling day. They were motivated to fast

overnight and present themselves at school without eating breakfast. Sampling was done on five consecutive days during July and August, 1988 (19th, 21st, 26th, 28th July and 2nd August). Blood sampling at the two smaller schools was done on the same morning. All samples were taken between 8:00 a.m. and 10:00 a.m. The subjects were given fruit juice (apple and grape) to break their fast. Subject details, anthropometric measurements, blood pressures, the CHD risk factor questionnaire and the dietary intakes, were done later during subsequent school and home visits by the researcher.

3.3 Biochemical Methods

3.3.1 Blood sampling

Antecubital venous blood samples were obtained from the subjects, who had fasted overnight, by doctors and registered nurses. The vacutainer method was used. Blood was collected into two glass 10 ml test-tubes from each subject. One test-tube contained 1 ml sterile citrate buffer solution (commercial citrate tube, pH = 4.5-4.8). The volumes of blood and citrate mixture was carefully noted to calculate a dilution factor from an experimentally obtained dilution curve for the eventual calculation of fibrinogen and factor VII concentrations. These tubes were immediately centrifuged and the plasma was divided into three aliquots. Two tubes were kept on ice while the third was kept (for factor VII) at room temperature to avoid cold activation. All these

tubes were transported to Potchefstroom where they were quickly frozen and kept at -30°C, for the later determination of the fibrinogen concentration and factor VII coagulation activity.

The second tube of blood was allowed to clot and centrifuged for the preparation of three aliquots of serum samples for the determination of serum lipids. Figure 3.1 illustrates the allocation of blood samples.

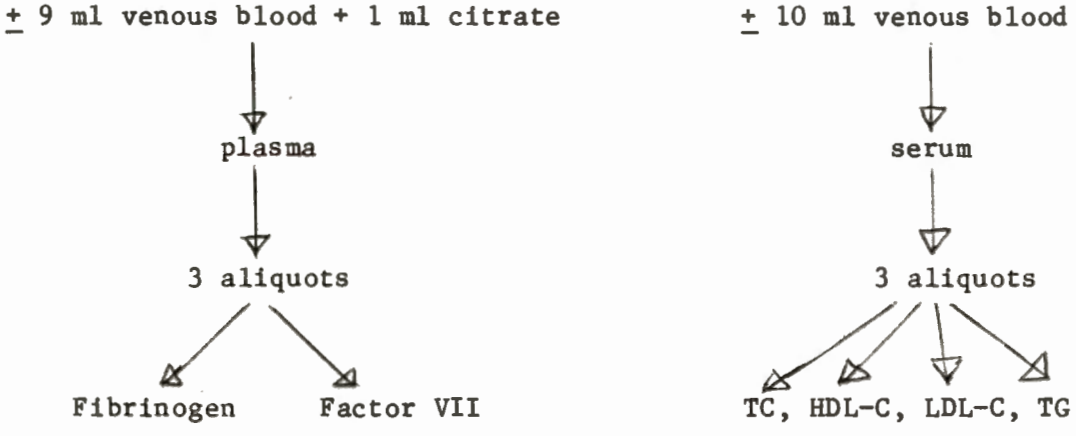


Figure 3.1: Allocation of blood samples

3.3.2 Haemostatic variables

Fibrinogen and factor VII

Plasma samples were thawed at 37°C within 2 minutes and fibrinogen and factor VIIc determinations were carried out in duplicate with a Behring

Fibrintimer(R) (Behring Institute, Marburg, Germany), using reagents, buffers, control and normal human plasmas from the Behring Institute.

Fibrinogen was measured with a revised method of Clauss (1957) in which clotting time of a sample is determined after addition of an excess amount of thrombin.

The method of Clauss on the fibrintimer was previously compared to a kinetic method from Boehringer Mannheim (Becker et al., 1984) by Vorster (1987a). Correlation between the two methods was: $r = 0.87$ ($p < 0.001$). The Behring Fibrintimer detects clot formation by a turbo-densitometric method and measures coagulation time automatically as the time required for clot formation after a specific treatment of a sample. Fibrinogen is expressed in mg/dl. The coefficient of variation (CV) of the Clauss method for the present study was between 4.74% and 7.7%.

Factor VII activity was determined on the principle of adding equal volumes of factor VII deficient plasma and diluted plasma from each subject. The coagulation reaction is triggered by adding a thrombokinase (Thromborel S(R).) and coagulation time is used to calculate factor VII activity (see Table 3.2).

Factor VIIc is expressed as a percentage of normal, in comparison with values obtained with normal control or standard human plasma (B1 : Code no. ORKE 07). The factor VII activity of this normal plasma was 78% (BI). The CV for factor VII determinations was 3.49%.

Table 3.2 outlines the details of the coagulation tests with the Fibrintimer(R).

Table 3.2: DETAILS OF COAGULATION TESTS FOR THE FIBRINTIMER(R)

TEST	PREPARATION STEP (37°C)	INCUBATION TIME	START REAGENT
Fibrinogen	0.2 ml plasma dilution (1+9 buffer solution**)	60s	0.1ml "multifibren"*** dissolved in Kaolin suspension
Factor VII	0.1 ml f-VII-deficient plasma 0.1 ml plasma dilution (1+19 buffer solution*)	60s	0.2 ml Thromborel S(R) (37°C)

* Diethylbarbiturate Acetate Buffer Solution pH 7.6

** Barbital Buffer Solution

*** Multifibren(R) fibrinogen reagent.

3.3.3 Serum lipids

Total serum cholesterol (TC), high-density lipoprotein cholestrol (HDL-C), and triglycerides (TG) were determined in duplicate with enzymatic- colorimetric kits from Boehringer Mannheim (Mannheim, West Germany) with internal and external standards and control sera which were treated and analysed like the samples.

Total cholesterol

The concentration of cholesterol in serum was measured using the CHOD-PAP method from Boehringer Mannhein (BM. Cat. no. 237 574). Cholesterol esterase hydrolyses the cholesterol esters in serum to form free cholesterol and fatty acids. The free cholesterol is then oxidized by cholesterol oxidase to produce hydrogen peroxide and 4-cholestenone. Hydrogen peroxide reacts, in the presence of peroxidase, with 4-amino-phenazone and phenol to give a coloured complex, 4-(para-benzoquinone-mono-imino)-phenazone (a quinoneimine dye) and water. Since the reaction takes place quantitatively, the concentration of the colour reagent (i.e. the intensity of the colour developed), is directly proportional to the cholesterol content in the serum sample which is measured colorimetrically with a spectrophotometer at 500 nm. A calibration curve was constructed for each series of determinations using Preciset(R) (internal cholesterol standard) cholesterol (BM : Cat. no. 125 512) in concentrations from 1.29 to 10.35 m mol/L. Precilip(R) (external serum standard) (BM : Cat. no. 125 059) and Precinorm(R) (external serum standard) (BM :

Cat. no. 781 827) was used as external standards. The CV of the method for TC for intra-day was 0.7% and for inter-day was 0.8%.

High-density lipoprotein cholesterol

Chylomicrons, VLDL and LDL are precipitated by adding phosphotungstic acid and magnesium ions to the sample (BM : Cat. no. 543 004). Centrifugation leaves HDL in the supernatant and the cholesterol fraction is determined enzymatically with the method described above for TC. A special control serum for HDL-C (BM : Cat. no. 651 281) was used as external standard. The CV of the measurements was between 1.17% and 1.7%.

Low-density lipoprotein cholesterol

Low density lipoprotein cholesterol (LDL-C) was calculated from the formula of Friedewald et al. (1972) as follows:

$$TC = HDL\text{-}C + LDL\text{-}C + VLDL\text{-}C$$

$$VLDL\text{-}C = TG/2.18$$

$$\text{Therefore} \quad LDL\text{-}C = TC - (HDL\text{-}C + TG/2.18) \text{ in mmol/l}$$

The values obtained are reliable, provided that

- no chylomicrons are present in the sample
- the triglyceride concentration does not exceed 400 mg/100ml (4.58 mmol/l)

- the sample does not show signs of type III hyperlipoproteinaemia (Friedewald et al., 1972).

Triglycerides

Serum triglycerides (TG) were determined with the Peridochrom(R) Triglycerides GPO-PAP assay method (BM : Cat. no. 701 904).

During the first enzymatic reaction mediated by lipase, TG in the serum is hydrolysed to glycerol and free fatty acids. In the presence of ATP, glycerol is catalysed to glycerol-3-phosphate by glycerol kinase and in the presence of oxygen glycerol-3-phosphate is catalysed by glycerol-3-phosphate oxidase to dihydroxyacetone phosphate and hydrogen peroxide. The hydrogen peroxide then reacts with 4-aminophenazone and 4-chlorophenol in the presence of peroxidase to form water, hydrochloric acid and 4-(p-benzoquinone-monoimino) phenazone. The latter, which is proportional to the hydrogen peroxide and therefore to the quantity of the glycerol, is measured colorimetrically with a spectrophotometer at 500 nm. The glycerol is proportional to the TG content in the serum sample. Precimat(R) Glycerol (internal TG standard) (BM : Cat. no. 166 588) was used as an internal standard and Precilip(R) (external serum standard) and Precinorm(R)L (external serum standard) for control of accuracy. The CV of the method was between 0% and 0.86%.

3.4 Other Measurements

3.4.1 Subject details

The CHD risk factor questionnaire (Appendix C) was used to record subject details, including gender, age and religion.

3.4.2 Anthropometric measurements

The subjects wore school uniforms without jerseys and wore socks and no shoes during these measurements.

3.4.2.1 Height

Height was measured to the nearest 0.5 mm using a plastic anthropometer. The sturdy vertical measuring rod was fitted with a movable plastic arm or head-piece set at right angles to the upright measuring rod. The subjects stood upright with feet together and back and buttocks touching the anthropometer. Their heads were erect and the direction of vision was straight ahead. Their arms were at their sides. The cross-bar or head-piece of the anthropometer was gently lowered onto the head making firm contact with the scalp without exerting undue pressure.

3.4.2.2 Mass (Weight)

Weight was measured to the nearest 0.5 kg using a portable calibrated

bathroom scale (EKS - Germany; capacity 120 kg). The Quetelet index or body mass index (BMI), a measure of ponderosity, weight/height^2 (kg/m^2) was then calculated for each subject.

3.4.2.3 Mid-upper-arm circumference

Mid-upper-arm circumference was measured to the nearest mm using a measuring tape 20mm in width. This measurement was made on the unclothed left upper arm, which was hanging freely, at a level midway between the tip of the acromial process of the scapula and the tip of the olecranon process of the ulna (WHO, 1966).

3.4.3 Blood pressure measurement

Blood pressure measurements were made after completing the habitual dietary record (60-90 minutes) by which time the subjects were at ease with the researcher and were comfortable and relaxed in the familiar environment of their homes. It was ensured that there were no smokers present during the interviews and that the noise level was low. All measurements were made at room temperature.

Apparatus

A previously calibrated mercury sphygmomanometer (Baumanometer) was used. Two cuff sizes were used. The 9 cm cuff was used for subjects with a mid-upper-arm circumference of 18-22 cm, the 12 cm cuff for those with a

mid-upper-arm circumference of 22-36 cm. The 15 cm cuff was not used as there were no subjects with a mid-upper-arm circumference of over 36 cm. The criteria used for hypertension were according to WHO standards (Simpson et al., 1965; Kirkendall et al., 1967); i.e. for subjects under the age of 20 years, a systolic blood pressure equal to or greater than the mean plus 2 standard deviations and/or a diastolic blood pressure equal to or greater than 90 mmHg. All blood pressure measurements were made by the researcher.

The subject was comfortably seated with the bared arm slightly flexed and relaxed. The cuff was applied to the left upper arm of the subject so that the bottom edge of the cuff was 4 cm above the antecubital space. A stethoscope receiver was placed snugly over the brachial artery, free from contact with the cuff. The cuff was slowly inflated (2-3 mmHg per heart beat) until the pressure in it was well above the systolic pressure in the brachial artery (20-30 mm above systolic pressure). The pressure in the cuff was decreased slowly (2-3 mm Hg per heart beat) (Van Zyl, 1983). Korotkoff phase I was recorded as systolic pressure and Korotkoff phase 5 was used as an index of diastolic pressure.

Three determinations were made for each subject over about 15 minutes and the lowest reading was recorded.

3.4.4 Coronary heart disease risk factor survey questionnaire (Appendix C)

3.4.4.1 Introduction

This questionnaire was completed (one month after blood samples were taken) by each subject under the personal supervision of the researcher on a class group basis at each of the six secondary schools.

Each question was read out and explained to the subjects. They were given enough time to complete each answer. Subjects were individually assisted with the difficulties they encountered. Each class required approximately 2 hours to complete the questionnaire (28 classes in total).

3.4.4.2 Demographic data

Personal details and day of interview were recorded. The gender, religion, age, as well as plans upon leaving school were recorded. The subjects who intended moving away from the Lenasia area were interviewed before their departure.

3.4.4.3 Medical history of subject and family

Medical history of hypertension, high blood cholesterol, obesity, diabetes mellitus and heart disease was assessed for each subject and his first degree relatives, and if death had occurred due to one of these

causes. The age of death of first degree relatives was recorded. The pupils were asked to state whether they were being treated by medication or diet for any of the above diseases.

3.4.4.4 Smoking habit

Smokers were asked to state the age when they began smoking, the form of smoking (cigarettes with or without filter), the number of cigarettes smoked per day and whether they inhaled the smoke.

3.4.4.5 Drinking habit

Consumers of alcoholic beverages such as beer, wine or spirits (Appendix C) were asked to state the age when they began, type, quantity and frequency of drinking habits.

3.4.4.6 Physical activity and exercise

Physical activity pattern (Appendix C) was categorized into time spent walking, manual labour and on sports.

3.4.4.7 Personality type

The Bortner Short Rating (1969) scale for coronary-prone behaviour (Appendix C) was used to assess behaviour pattern.

All the questions included in this scale were not relevant to the school-going subjects and thus a few questions which could give some idea of their stress levels were chosen from the list.

3.4.4.8 Age at menarche and use of contraceptives

Upon completing the Bortner scale, male subjects were allowed to leave the class. Female subjects were then asked to state age at menarche. They were also asked to state whether they used oral contraceptives as in younger women this usage results in substantially higher fibrinogen and factor VII values (Meade et al., 1976). However, not one female admitted to oral contraceptive usage. This was confirmed by visiting all pharmacies in the area as well as the family planning clinic. It seemed that the family planning clinic supplied oral contraceptives only to married women or those who had one or more children.

3.4.5 Dietary methods (assessing dietary intakes)

Subjects were asked to state whether they were pure vegetarians, lacto-vegetarian (consumer of plant foods with dairy products), lacto-ovo-vegetarian (consumer of plant foods with dairy products and eggs), non-vegetarian (consumer of plant and animal foods), or any other dietary regimen.

The habitual dietary intake was recorded in 286 of the 321 subjects using a food frequency questionnaire. The response rate was 286/321 (89%).

The questionnaire (Appendix D) used was developed for use in South African inter-ethnic populations. It had been previously validated against seven-day weighed records in all four South African population groups, including Indians. The reproducibility of the questionnaire was also tested (Vorster et al., 1985). The interview with each subject was conducted on a home-visit basis during the afternoons, evenings and weekends. The subjects were asked to mention any foods eaten which were not listed on the questionnaire. For some foods, recipes had to be noted.

Plastic food models and household utensils were used to assist subjects in determining portion sizes.

Both the validation procedures and the reproducibility tests showed that the questionnaire measures mean nutrient intakes of groups satisfactorily provided that portion sizes are illustrated clearly to subjects. Percentage differences in nutrient intakes measured by a 7-day weighed record and the questionnaire were $\leq$ 5% for 15 nutrients, between 5 and 10% for 12 nutrients and more than 10% but less than 20% only for ascorbic acid and calcium. The latter could have reflected real differences in intakes (Vorster, 1987b). The questionnaire was proved to be reliable and reproducible.

The researcher worked over nine months to complete the dietary questionnaires as appointments had to be made with the subjects for this time-consuming exercise. Some subjects presented great difficulties in

keeping appointments as they had started to work or to study. Thirty five subjects were "lost" to the study as some of these subjects refused to co-operate and thought it was unnecessary to be questioned about their dietary habits. Two of the subjects had emigrated to Canada without informing the researcher, while the rest just had no time available as they travelled long distances between their homes and places of employment and had other commitments at weekends. However, as all subjects had been well-informed during school assemblies about the aims of the entire study project, those who co-operated were enthusiastic and patient during the interview.

Each food item and quantity was coded by the researcher onto computer code forms (Appendix E) using the NRIND Food Quantities Manual (Langenhoven et al., 1986). Food intakes were converted to nutrient intakes (macro-, micro-nutrients, essential amino acids, fatty acids, vitamins and minerals) by computer using a programme based on local food tables (Gouws and Langenhoven, 1986a; Gouws and Langenhoven, 1986b). These tables, however, did not have codes for some of the "typical" Indian vegetables, spices and pulses and the food item codes that approximated these items were used. These are listed in Table 3.3.

In addition, for some typical and traditional Indian dishes such as sweet-meats and special soups, not presented in the food tables, each ingredient of the recipes was coded. These recipes are given in Appendix E.

Table 3.3: List of codes used as substitutes for food items not mentioned in the NRIND Food Quantities Manual (Langenhoven et. al., 1986)

CODE USED	NRIND FOOD ITEM	FOOD ITEM (THIS STUDY)	SIZE/ DESCRIPTION	WEIGHT OF FOOD ITEM IN GRAMS
4197	Roti, made with butter	Roti made with "ghee"	small/medium/ large	50/100/ 150
4046	Rice, puffed, plain (rice crispies, added minerals and vitamins)	Strawberry pops	125 ml	20
4241	Samoosa, with chick pea filling	Samoosas with lentils/soya beans filling	small medium large	42 85 150
4196	Samoosa with mutton filling	Samoosas with chicken/beef filling	small medium large	42 85 150

CODE USED	NRIND FOOD ITEM	FOOD ITEM (THIS STUDY)	SIZE/ DESCRIPTION	WEIGHT OF FOOD ITEM IN GRAMS
4242	Samoosa with potato filling	Samoosas with peas/mixed vegetable filling	small medium large	42 85 150
0024	Malted milk, dry powder (eg. Milo and Horlicks)	Nesquik/hot chocolate powder	125 ml	60
6516	Ice cream, commercial, sorbet (8% fat, coconut oil)	Diet ice cream	scoop 58 mm diameter	40
1002	Fried egg, in butter	Fried egg in "ghee"	Large egg 50 g + "ghee"	52
1026	Scrambled egg, whole milk, fried in poly-unsaturated margarine	Scrambled egg fried in oil	Medium/large X-large	45/50 55
1544	Sausage, beef, dry	Sausage, dry, mutton	150 x 30 x 15 mm	60

CODE USED	NRIND FOOD ITEM	FOOD ITEM (THIS STUDY)	SIZE/ DESCRIPTION	WEIGHT OF FOOD ITEM IN GRAMS
3513	Beans, white kidney, dried, cooked, canned (solids and liquid)	White dry beans (waal)/split dry beans (waal dhal)/ dry beans (chori)	125 ml	135
3509	Lentils, cooked	Pink lentils-split (masoor dhal)/ black gram lentil (masoor)	125 ml	90
3531	Chick peas, dried, cooked	Oil dhal (toovar) pea dhal/chana dhal	125 ml	85
3506	Peas, dried, cooked	Green chick peas (moong)/green chick pea dhal or mug dhal/moong (dhal)	125 ml	85
3529	TVP, chunks, plain	Soya frikkadelle	100 mm diam. x 12 mm	100

CODE USED	NRIND FOOD ITEM	FOOD ITEM (THIS STUDY)	SIZE/ DESCRIPTION	WEIGHT OF FOOD ITEM IN GRAMS
8027	Peas, fresh/frozen, cooked/canned with sugar	Fresh toovar peas shelled (toovar hing)	125 ml	85
8100	Beans, green, cooked (sunflower oil)	String beans (chori)/flat beans (papri) and guar phalli	125 ml shredded	75
8095	Imfino, raw ("marogo")	Amadumbe leaves, cooked (patta or patterbeli or patra or patterwella)	125 ml	90
4220	Wheat and malted barley, flakes/ granules (grape nuts/-flakes)	Laapsi (crushed wheat)	125 ml	45
4030	Pancake/crumpets, batter only (whole milk and sunflower oil)	Puris (flaky, flat, fried in sunflower oil)	70 mm diameter	25

CODE USED	NRIND FOOD ITEM	FOOD ITEM (THIS STUDY)	SIZE/ DESCRIPTION	WEIGHT OF FOOD ITEM IN GRAMS
9524	Chutney, tomato, peach "achaar"	Sweet mango "achaar"	125 ml	140
7026	Mango, raw	Mango achaar (pickles) with code 6536	125 ml	140
8195	Pumpkin and squash, summer type, cooked (sunflower oil)	Small/baby cucumber Indian type (gilorha), baby marrow Indian type, cooked in sunflower oil	125 ml	85
7034	Pawpaw, raw	Papino	cubes 125 ml	70
7131	Fruil rolls, dried (eg. peach, apricot, guava)	Bor (sour cherries) china fruit	1 unit	5

CODE USED	NRIND FOOD ITEM	FOOD ITEM (THIS STUDY)	SIZE/ DESCRIPTION	WEIGHT OF FOOD ITEM IN GRAMS
8009	Brussels sprouts, cooked	Drumsticks (Hekta ni phalli) and bitter gourd (karela)	125 ml	80
1633	Pork, trotters, cooked	sheep, trotters, cooked	125 ml	125
4089	Pudding, blancmange (whole milk)	Faluda (Agar-Agar, jelly with milk or china grass)	125 ml	90

3.5 Statistical methods

Statistical analysis was done with the help of the Biostatistical Consultation Service of the PUCHO.

All results were encoded and computerised. Means and standard deviations (SD) for all variables were calculated using the SAS(R)-package (SAS(R) User's Guides (1985).

Significant differences between groups (non-vegetarian, vegetarian, male and female) were calculated with analyses of variance and Student-Newman-Keul's (SNK) test and Tukey's Studentised tests. Level of $p < 0.05$ were regarded as statistically significant.

Pearson correlation coefficients as well as stepwise regression analysis were calculated using the SAS(R)-package. For the latter, a forward selection procedure was used, and the 0.50 statistically significant level was used for entry or stay in the model.

CHAPTER 4

RESULTS

In this section the results of this study are reported in Tables 4.1-4.14. Comparison of the results with those found in other studies and interpretation of the results will be made in the discussion section (Chapter 5).

The personal characteristics of the 321 randomly-selected subjects are presented in Table 4.1. Mean (± SD) values for age, height, weight, Quetelet or body mass index (BMI), mid-upper-arm circumference, systolic and diastolic pressure are given for non-vegetarian and vegetarian male and female subjects, as well as age at menarche of female subjects.

The number of vegetarians in this study was small (± 10%) and thus the results cannot be extrapolated to the general vegetarian population. However, as they were part of the randomly selected group, it was felt that on scientific grounds, dietary intake and blood data of vegetarians and non-vegetarians should be treated separately. All vegetarians in this study were lacto-ovo-vegetarians, thus their diet contained milk and eggs but no meat or fish.

Mean height of both male and female non-vegetarian subjects was similar to that of vegetarian subjects. Mean weight of vegetarian males was significantly lower ($p < 0.05$) than that of non-vegetarian males.

TABLE 4.1: Personal characteristics of subjects

Variables	MALE		FEMALE	
	Non-vegetarian	Vegetarian	Non-vegetarian	Vegetarian
	(n = 148) Mean ± SD	(n = 11) Mean ± SD	(n = 142) Mean ± SD	(n = 20) Mean ± SD
Age (years)	17.6 ± 1.1	17.4 ± 0.5	17.5 ± 0.8	17.4 ± 0.6
Height (cm)	168.8 ± 6.6	168.0 ± 4.1	155.3 ± 5.8	155.4 ± 6.5
Weight (kg)	[a]57.0 ± 8.1	[a]51.9 ± 5.0	50.1 ± 11.0	47.2 ± 6.2
Quetelet or Body mass index (kg/m^2)	20.0 ± 6.4	18.4 ± 4.6	20.8 ± 6.8	19.5 ± 6.2
Mid-upper-arm circumference(cm)	[b]24.0 ± 2.3	[b]22.3 ± 1.8	22.8 ± 2.8	22.0 ± 1.6
Systolic blood pressure (mmHg)	116.3 ± 10.3	117.8 ± 8.5	111.0 ± 8.8	111.0 ± 8.8
Diastolic blood pressure (mmHg)	72.6 ± 8.5	72.4 ± 7.3	68.9 ± 7.7	67.8 ± 6.4
Age at menarche (years)	-	-	12.7 ± 1.2	12.7 ± 1.2

Means with the same symbols differ significantly ($p < 0.05$).

Although vegetarian females had a lower mean value for weight than non-vegetarian females, this was not significant at the 5% level. The result was a slightly (but not significantly) lower BMI for both male and female vegetarians. Mean value for mid-upper-arm circumference of vegetarian males was significantly lower ($p < 0.05$) than that of non-vegetarian males, while that of vegetarian females was only slightly lower than that of non-vegetarian females and this was not significant at the 5% level.

Blood pressure levels (systolic and diastolic) of males and females were similar in both vegetarian and non-vegetarian groups. However, although the mean values were in the normal range for blood pressure in adolescents, 4 non-vegetarian males were found to have blood pressure readings in the hypertensive range (i.e. systolic/diastolic > 132/82 mmHg) (Sharma et al., 1991), or, as in the present study, mean $\pm$ 2 SD, i.e. 137/90 mmHg. Their systolic pressures ranged from 140 to 146 mmHg and diastolic pressures from 80 to 88 mmHg. These subjects were advised to consult their family doctors.

The mean value for age at menarche of vegetarian and non-vegetarian female subjects was the same.

Results of the questionnaire (as answered by the subjects) on levels of risk factors associated with coronary heart disease are given in Table 4.2. Of the total sample of subjects, 10.4% were Christians, 42.7% were Hindu, 45.3% were Muslim, and 1.6% were followers of other religions.

None of the participants were aware of or confessed to a medical history of hypertension, hypercholesterolaemia, diabetes mellitus or CHD. A small percentage of these subjects (4.2%) stated that they were obese. Of the total sample of subjects 3.3% indicated that they were on a special diet for acne problems, 0.3% were on cholesterol-lowering diets, while 1.6% were on weight reducing diets. It is interesting to note that one subject admitted to being on a cholesterol-lowering diet as none had stated that they had high cholesterol levels or admitted to having a family history of hypercholesterolaemia. Some subjects (2.3%) were on antibiotics for common colds and influenza. These subjects were not included in the study as we were able to substitute them with our randomly-selected reserve subjects.

One-third of the subjects indicated that their family income (based on occupation of parents) classed them socio-economically in the highest category, about one-third in the intermediate category, and one-third in the relatively poor category.

Of the total group of subjects 21.2% (68/321) were smokers and 2.9% had first smoked at the age of 10 years. Of these smokers 97.1% smoked cigarettes with filters and 2.9% smoked cigarettes without filters.

TABLE 4.2 Results of questionnaire on levels of risk factors associated with coronary heart disease among adolescents expressed as percentage of total (as answered by subjects)

WHOLE GROUP

Religion (Percentage)	Christian	Hindu	Muslim	Other
	10.4	42.7	45.3	1.6

Medical history of hypertension (Percentage)	Yes	No	Do not know
	0	84.7	15.3

Medical history of high blood cholesterol (Percentage)	Yes	No	Do not know
	0	77.8	22.2

Medical history of diabetes mellitus (Percentage)	Yes	No	Do not know
	0	91.5	8.5

Medical history of CHD (Percentage)	Yes	No	Do not know
	0	91.5	8.5

Obese (Percentage)	Yes	No	Do not know
	4.2	89.5	6.2

Medication at time of blood sampling (Percentage)	Antibiotics	Other medication	None
	2.3	1.3	96.4

Socio-economic status of parents (Percentage)	Business Executive	General Dealer/ Professional	Salesman/Manual Worker
	33.0	32.3	34.7

Smoking habits (Percentage of total)	Yes	No
	(68/321) 21.2	78.8

Age when first smoked (yr) (Percentage of smokers)	10	11	12	13	14	15	16	17	18	19
	2.9	1.5	0	5.9	7.4	25.0	29.4	23.5	2.9	1.5

Smoking - type	Cigarettes with filter	Cigarettes without filter
	97.1	2.9

Number of cigarettes smoked during weekends (Percentage of smokers)	1-5	6-10	11-15	16-20	21-25
	36.1	42.7	14.7	4.9	1.6

Number of cigarettes smoked during weekdays (Percentage of smokers)	1-5	6-10	11-15	16-20	21-25
	46.3	45.4	3.2	3.1	0

Consume alcoholic beverages (Percentage)	Yes	No
	20.1	79.9

About one-fifth of the subjects (20.1%) consumed alcoholic beverages, usually on social occasions, i.e. beer, spirits or wine; 34.6% (55/159) of this group were males and 5.6% (9/162) were females.

The distribution of male and female smokers, alcohol consumers and the level of physical activity is given in Table 4.3. Of the 159 males, 59 (37.1%) were smokers and of the 162 females, 9 (5.6%) were smokers. The mean age at which males first smoked was 15.6 $\pm$ 1.7 years and that of females was 15.0 $\pm$ 1.4 years.

TABLE 4.3 Distribution of male and female smokers, alcohol consumers and level of physical activity

Variables	Males (n = 159)	Females (n = 162)
Number of smokers	59	9
Percentage of smokers	37.1	5.6
Mean age when first smoked (years)	15.6 ± 1.7	15.0 ± 1.4
Number of subjects consuming alcoholic beverages	55	9
Percentage alcohol consumers	34.6	5.6
Number of subjects consuming beer	17	0
Number of subjects consuming wine	22	7
Number of subjects consuming spirits	16	2
Mean of number of hours of walking per week	14.7 ± 7.8	15.0 ± 8.8
Mean of number of hours spent on physical labour, e.g. gardening	3.2 ± 2.8	3.6 ± 3.5
Mean of number of hours spent on sporting activities, e.g. tennis, soccer	[a]3.7 ± 3.0	[a]1.7 ± 1.1

Means with the same symbols differ significantly ($p < 0.05$).

Of the 55 (34.6%) males consuming alcoholic beverages, 17 usually drank beer, 22 drank wine, while 16 usually had spirits. Of the 9 (5.6%) females consuming alcoholic beverages, 7 usually drank wine and 2 usually drank spirits.

There was no significant difference ($p < 0.05$) in the number of hours spent walking per week between males and females. This included walking to-and-from school, going shopping and around their homes. There was no significant difference between males and females in the number of hours spent doing physically laborious chores such as gardening, house-cleaning, washing and ironing. The sporting activity level of females was significantly lower ($p < 0.05$) than that of males who were more actively participating in soccer, tennis, swimming and physical exercises at school.

As it was difficult to assess the behaviour pattern of these subjects using the Bortner Short Rating (1969) Scale for coronary-prone behaviour, the results have not been reported here.

The mean daily habitual nutrient intakes of the subjects are given in Tables 4.4 - 4.9.

The mean energy intake of females, both vegetarian and non-vegetarian (Table 4.4) was lower than that of males. The value of the standard deviation is large in each category. The P/S ratio of all female subjects approximated 1.0 while that of vegetarian males was lower than that of non-vegetarian males, although this was not significant at the 5% level.

TABLE 4.4 Mean daily macro-nutrient intakes of non-vegetarian and vegetarian subjects

NUTRIENT	NUTRIENT INTAKES							
	Non-vegetarian				Vegetarian			
	Male (n = 126)		Female (n = 128)		Male (n = 11)		Female (n = 20)	
	Mean	(± SD)	Mean	(± SD)	Mean	(± SD)	Mean	(± SD)
Energy (kJ)	13439	(3902)	10296	(3099)	12965	(2256)	10003	(1907)
Total protein (g)	[a]108.2	(36.0)	[c] 83.2	(34.0)	[a] 72.7	(12.5)	[c] 52.8	(12.5)
Animal protein (g)	[b] 67.1	(30.0)	[d] 53.8	(29.8)	[b] 28.0	(9.8)	[d] 19.8	(8.0)
Plant protein (g)	41.0	(11.7)	[h] 29.3	(10.0)	44.6	(9.4)	[h] 32.8	(6.8)
Total fat (g)	147.6	(51.6)	[e]114.6	(39.3)	146.8	(28.2)	[e]126.4	(29.1)
Saturated fatty acids (g)	49.6	(21.0)	[j] 37.3	(15.2)	51.5	(14.4)	[j] 43.2	(11.9)
Monounsaturated fatty acids (g)	43.2	(16.3)	32.5	(12.4)	42.6	(11.2)	31.8	(8.3)
Polyunsaturated fatty acids (g)	40.9	(16.7)	35.3	(14.3)	38.2	(11.2)	39.5	(19.2)
* P/S ratio	0.9	(0.4)	1.0	(0.4)	0.8	(0.3)	1.0	(0.6)
Dietary Cholesterol: (mg)	[i]507.7	(286.4)	[f]363.5	(190.1)	[i]232.3	(97.6)	[f]187.7	(105.4)
(mg/MJ)	36.7	(11.8)	34.8	(12.6)	18.6	(9.5)	18.5	(9.7)
Total carbohydrate (g)	353.5	(98.0)	268.9	(85.8)	367.8	(88.1)	260.0	(50.9)
Sugar (g)	75.9	(48.2)	[g] 57.8	(33.7)	77.8	(48.6)	[g] 38.6	(21.1)
Dietary Fibre:(g)	29.1	(10.4)	22.0	(8.8)	30.4	(6.2)	24.1	(6.0)
(g/MJ)	2.2	(0.6)	2.2	(0.6)	2.4	(0.4)	2.4	(9.7)

Means with the same symbols differ significantly ($p < 0.05$).

* P/S ratio = polyunsaturated fatty acid/saturated fatty acid ratio.

The mean dietary cholesterol intake of vegetarian males and females was significantly lower ($p < 0.05$) than that of non-vegetarian males and females. This indicated that the vegetarians ate fewer eggs. However, total fat (MU-, PU-, and SAT-fatty acids plus cholesterol plus glycerol) intake of all vegetarians was high. The main source of their fat is animal fat from milk, cheese, butter and "ghee". However, the high P/S ratio indicates liberal use of plant oils.

Mean total carbohydrate intake of the females in both categories was lower than that of males, although the difference was not significant at the 5% level. Mean sugar intake of males was similar, while that of vegetarian females was significantly lower ($p < 0.05$) than that of non-vegetarian females. The mean dietary fibre intake of non-vegetarian and vegetarian males was similar, while that of non-vegetarian females was lower than that of vegetarian females, although this did not differ significantly ($p > 0.05$). Dietary fibre intake of males and females in both groups was low.

Mean values of percentage energy from protein, fat and carbohydrate of non-vegetarian and vegetarian males and females are given in Table 4.5.

TABLE 4.5 Percentage energy contribution of the macro-nutrients of non-vegetarian and vegetarian subjects

PERCENTAGE ENERGY FROM:	Non-vegetarian		Vegetarian	
	Males (n = 126)	Females (n = 128)	Males (n = 11)	Females (n = 20)
Energy (kJ)	13439 ± 3902	10296 ± 3099	12965 ± 2256	10003 ± 1907
Total protein	[a]13.7 ± 2.3	[b]13.7 ± 3.0	[a]9.6 ± 1.7	[b]9.0 ± 1.9
Animal protein	8.4 ± 2.4	8.8 ± 3.4	3.7 ± 1.5	3.3 ± 1.2
Total fat	41.5 ± 4.9	[c]42.1 ± 5.5	43.1 ± 5.4	[c]47.7 ± 5.3
Saturated fatty acids	14.1 ± 5.9	13.8 ± 5.6	15.4 ± 4.2	16.5 ± 4.5
Monounsaturated fatty acids	12.2 ± 4.6	12.0 ± 4.6	12.5 ± 3.3	12.5 ± 3.2
Polyunsaturated fatty acids	11.5 ± 4.7	13.0 ± 5.3	11.1 ± 3.3	15.3 ± 7.3
Added sugar	9.3 ± 4.7	[d]9.4 ± 4.6	9.8 ± 4.5	[d]6.8 ± 3.7
Carbohydrate	44.9 ± 4.6	44.6 ± 6.1	48.0 ± 5.2	44.4 ± 3.9

Means with the same symbols differ significantly ($p < 0.05$)

Table 4.5 indicates that the vegetarian males and females had a similar mean percentage energy value from protein which was, as can be expected, almost one-third (significantly) lower ($p < 0.05$) than that of non-vegetarians. Unexpected, however, was the percentage energy from total fat which was the lowest for non-vegetarian males and the highest for vegetarian females, which indicated that vegetarians fried most of their food in "ghee", butter, margarine and sunflower seed oil, and also had a high intake of cheese and milk. Mean percentage energy from carbohydrate was similar for non-vegetarian males and females and vegetarian females; the value for vegetarian males was slightly higher, although this was not significant at the 5% level.

The mean dietary intake of fatty acids of non-vegetarian and vegetarian males and females is given in Table 4.6.

The high levels of intake of oleic acid (C18-1) and linoleic acid (C18-2), both constituents of plant oil, indicate the use of oil as a cooking medium.

The mean intakes of myristic acid (C14:0), palmitic acid (C16:0), palmitoleic acid (C16:1) and linoleic acid (C18:2) of non-vegetarian females were significantly lower ($p < 0.05$) than those of vegetarian females. There were no significant differences in the intake of these fatty acids between the two groups of males. Although there was a significant difference between the males of the two groups as well as between the females of the two groups in the intake of arachidonic acid (C20:4), eicosapentaenoic acid (C20:5) and decosahexaenoic acid (C22:6), these intakes were probably too small to be meaningful.

TABLE 4.6 Daily dietary fatty acid intakes of non-vegetarian and vegetarian subjects

Fatty acid (g)		Non-vegetarian		Vegetarian	
		Males (n = 126)	Females (n = 128)	Males (n = 11)	Females (n = 20)
		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Myristic Acid	(C14-0)	5.0 ± 3.0	[a]3.6 ± 2.2	6.2 ± 2.6	[a]5.4 ± 2.5
Palmitic Acid	(C16-0)	22.4 ± 10.1	[b]16.3 ± 7.2	23.5 ± 6.6	[b]19.4 ± 6.2
Stearic Acid	(C18-0)	12.3 ± 5.0	9.3 ± 3.9	11.6 ± 3.0	10.2 ± 2.9
Arachidonic Acid	(C20-0)	0.3 ± 0.2	0.2 ± 0.1	0.3 ± 0.1	0.2 ± 0.1
Behenic Acid	(C22-0)	0.5 ± 0.3	0.4 ± 0.2	0.5 ± 0.2	0.4 ± 0.2
Lignoceric Acid	(C24-0)	0.2 ± 0.1	0.1 ± 0.1	0.1 ± 0.1	0.1 ± 0.1
Palmitoleic Acid	(C16-1)	1.3 ± 0.9	[c]1.0 ± 0.7	1.0 ± 0.7	[c]0.6 ± 0.2
Oleic Acid	(C18-1)	36.9 ± 14.6	27.2 ± 10.8	38.0 ± 11.2	28.8 ± 8.2
Linoleic Acid	(C18-2)	33.2 ± 15.0	[d]27.7 ± 13.0	31.8 ± 9.1	[d]33.8 ± 18.1
Linolenic Acid	(C18-3)	1.0 ± 0.4	0.7 ± 0.4	0.8 ± 0.2	0.7 ± 0.2
Arachiodonic Acid	(C20-4)	[f]0.14 ± 0.12	[e]0.01 ± 0.1	[f]0.01 ± 0.02	[e]0.01 ± 0.01
Eicosapentaenoic Acid	(C20-5)	[h]0.03 ± 0.05	[g]0.02 ± 0.03	[h]0.00 ± 0.00	[g]0.002 ± 0.006
Docosahexaenoic Acid	(C22-6)	[i]0.07 ± 0.08	[j]0.05 ± 0.06	[i]0.004 ± 0.002	[j]0.003 ± 0.010

Means with the same symbols differ significantly ($p < 0.05$).

Mean daily essential amino acid intake of non-vegetarian and vegetarian males and females is given in Table 4.7, illustrating adequate intakes for normal growth and development (Smit and Meyer, 1988). The mean daily intake of all ten essential amino acids of both vegetarian males and females were significantly lower ($p < 0.05$) than that of non-vegetarian males and females, probably because of the absence of meat in the vegetarian diet.

Mean daily dietary mineral intake of non-vegetarian and vegetarian males and females is given in Table 4.8.

The calcium intake of non-vegetarian females was lower although not significantly lower than that of vegetarian females, while vegetarian males had a higher intake than non-vegetarian males. The iron intake of all subjects was adequate. The magnesium, phosphorus, potassium, selenium and sodium intake of vegetarian males and females was lower (although not significantly, than that of non-vegetarian males and females. The zinc intake of vegetarian males and females was significantly lower ($p < 0.05$) than that of non-vegetarians. The copper intake of vegetarian males was lower, although not significantly, than that of non-vegetarians, while that of vegetarian females was significantly lower ($p < 0.05$) than that of non-vegetarian females.

TABLE 4.7 Daily dietary essential amino acid intake of non-vegetarian and vegetarian subjects

Essential Amino Acid (g) (daily intake)	Non-vegetarian		Vegetarian	
	Male (n = 126)	Female (n = 12)	Male (n = 11)	Female (n = 20)
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Isoleucine	$^{a}4.3 \pm 1.7$	$^{f}3.4 \pm 1.6$	$^{a}2.7 \pm 0.6$	$^{f}2.0 \pm 0.6$
Leucine	$^{b}7.0 \pm 2.8$	$^{g}5.4 \pm 2.6$	$^{b}4.6 \pm 1.2$	$^{g}3.4 \pm 1.0$
Lysine	$^{c}6.7 \pm 2.7$	$^{h}5.3 \pm 2.7$	$^{c}3.6 \pm 1.0$	$^{h}2.6 \pm 0.8$
Methionine	$^{d}2.1 \pm 0.8$	$^{i}1.6 \pm 0.8$	$^{d}1.1 \pm 0.3$	$^{i}0.8 \pm 0.2$
Phenylalanine	$^{e}4.0 \pm 1.6$	$^{j}3.1 \pm 1.4$	$^{e}2.8 \pm 0.7$	$^{j}2.0 \pm 0.5$
Threonine	$^{k}3.7 \pm 1.5$	$^{l}2.9 \pm 1.4$	$^{k}2.1 \pm 0.5$	$^{l}1.6 \pm 0.4$
Tryptophan	$^{m}1.1 \pm 0.4$	$^{n}0.8 \pm 0.4$	$^{m}0.6 \pm 0.2$	$^{n}0.4 \pm 0.1$
Valine	$^{o}4.8 \pm 1.9$	$^{p}3.7 \pm 1.7$	$^{o}3.2 \pm 0.8$	$^{p}2.4 \pm 0.7$
Arginine	$^{q}5.2 \pm 2.1$	$^{r}4.0 \pm 1.9$	$^{q}2.8 \pm 0.9$	$^{r}2.0 \pm 0.5$
Histidine	$^{s}2.6 \pm 1.0$	$^{t}2.0 \pm 1.0$	$^{s}1.5 \pm 0.4$	$^{t}1.1 \pm 0.3$
Total amino acids	41.4 ± 16.6	32.3 ± 15.5	25.2 ± 6.3	18.4 ± 5.1
% of total proteins	37.8 ± 3.9	38.1 ± 4.0	34.3 ± 4.6	34.6 ± 3.3

Means with the same symbols differ significantly ($p < 0.05$).

TABLE 4.8 Daily dietary mineral intake of non-vegetarian and vegetarian subjects

Minerals (mg)	Non-vegetarian		Vegetarian	
	Males (n = 126)	Females (n = 128)	Males (n = 11)	Females (n = 20)
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Calcium	991.5 ± 498.3	764.6 ± 371.9	1125.8 ± 342.3	875.2 ± 291.2
Iron	17.3 ± 6.0	13.0 ± 4.9	14.8 ± 4.2	11.2 ± 3.5
Magnesium	386.4 ± 129.1	286.8 ± 93.5	367.7 ± 103.2	262.4 ± 63.9
Phosphorus	1661.9 ± 601.7	1234.6 ± 450.0	1466.2 ± 316.6	1064.8 ± 276.6
Potassium	3536.6 ± 1244.4	2826.5 ± 860.4	3214.4 ± 576.5	2532.4 ± 620.0
Sodium	2779.5 ± 808.3	2026.9 ± 667.5	2664.0 ± 561.4	1750.6 ± 456.5
Zinc	^{d}16.6 ± 6.2	^{b}12.8 ± 6.4	^{d}9.2 ± 2.0	^{b}6.6 ± 1.8
Copper	2.2 ± 0.7	^{c}1.6 ± 0.6	1.8 ± 0.3	^{c}1.3 ± 0.3
Selenium	68.9 ± 33.6	51.4 ± 26.5	67.8 ± 60.2	43.0 ± 14.0

Means with the same symbols differ significantly ($p < 0.05$).

TABLE 4.9 Daily dietary vitamin intake of non-vegetarian and vegetarian subjects

MICRONUTRIENTS	Non-vegetarian		Vegetarian	
Vitamin	Males (n = 126)	Females (n = 128)	Males (n = 11)	Females (n = 20)
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Fat-soluble:				
Vitamin A (IU)	9282.2 ± 5332.7	8586.7 ± 5466.1	10635.6 ± 3026.4	9165.9 ± 4106.5
Vitamin A (RE)	1670.6 ± 991.0	1498.3 ± 1096.3	1597.5 ± 267.1	1278.5 ± 496.4
Vitamin D (mg)	1.8 ± 1.2	1.3 ± 0.9	1.7 ± 0.8	1.0 ± 0.6
Vitamin E (mg)	34.6 ± 17.4	[a]28.7 ± 14.4	36.4 ± 11.9	[a]35.9 ± 19.7
Water-soluble:				
Thiamin (mg)	1.6 ± 0.5	1.2 ± 0.4	1.5 ± 0.3	1.1 ± 0.3
Riboflavin (mg)	2.1 ± 1.0	1.6 ± 0.7	1.7 ± 0.4	1.3 ± 0.5
Nicotinic acid (mg)	[b]22.1 ± 7.4	[c]16.4 ± 6.7	[b]13.4 ± 3.8	[c]9.9 ± 3.2
Vitamin B_6 (mg)	[e]1.9 ± 0.7	[d]1.5 ± 0.6	[e]1.4 ± 0.4	[d]1.1 ± 0.4
Folic acid (ug)	[f]362.7 ± 163.2	260.3 ± 112.8	[f]246.2 ± 63.8	205.4 ± 62.0
Vitamin B_{12}(ug)	[g]9.4 ± 5.8	[h]8.0 ± 5.7	[g]4.5 ± 1.8	[h]2.8 ± 1.5
Ascorbic acid (mg)	119.0 ± 84.2	93.2 ± 63.4	101.2 ± 43.9	108.2 ± 49.3
Pantothenic acid (mg)	[i]6.4 ± 2.6	[j]4.7 ± 1.7	[i]4.2 ± 0.7	[j]3.3 ± 1.2
Biotin (ug)	23.7 ± 12.4	[k]17.3 ± 7.8	18.2 ± 6.3	[k]13.2 ± 6.6

Means with the same symbols differ significantly ($p < 0.05$).

Mean daily dietary vitamin intake of non-vegetarian and vegetarian males and females is given in Table 4.9.

Vitamin A (IU) intake of non-vegetarian males and females was lower, although not significantly than that of vegetarians, whereas the vitamin A (RE) of vegetarian males and females was lower, also not significantly, than that of non-vegetarians. The vitamin D, thiamin and riboflavin intake of vegetarian males and females was lower (although not significantly) than that of non-vegetarians. The vitamin E intake of non-vegetarian females was significantly lower than that of vegetarian females. There was no significant difference in the vitamin E intake of males. The nicotinic acid, vitamin B_6, vitamin B_{12} and pantothenic acid intake of all vegetarians was significantly lower ($p < 0.05$) than that of non-vegetarians. The folic acid intake of vegetarian males was significantly lower ($p < 0.05$) than that of non-vegetarian males but the difference in intake between the females was not significantly different. The ascorbic acid intake of vegetarian males was lower than that of non-vegetarian males, although not significantly, while that of vegetarian females was greater, although not significantly, than non-vegetarian females. The biotin intake of vegetarian females was significantly lower ($p < 0.05$) than that of non-vegetarian females, while the difference among males was not significant.

In Table 4.10, the values of protein, vitamin and mineral intakes are compared with the recommended dietary allowances (RDAs) (Food and Nutrition Board, National Academy of Sciences, 1989) for the specific age group, weight and height (Hamill et al., 1979) of males and females.

TABLE 4.10 Nutrient intakes of Indian non-vegetarian and vegetarian subjects compared with the Recommended Dietary Allowances (RDAs)* - Revised 1989

Age (years) and sex group	Fat-soluble vitamins						Water-soluble vitamins							Minerals				
	Weight≠ (kg)	Height≠ (cm)	Protein (g)	Vitamin A (RE)	Vitamin D (mg)	Vitamin E (mg)	Vitamin C (mg)	Thiamin (mg)	Riboflavin (mg)	Niacin (mg)	Vitamin B_6 (mg)	Folate (ug)	Vitamin B_{12} (ug)	Calcium (mg)	Phosphorus (mg)	Magnesium (mg)	Iron (mg)	Zinc (mg)	Selenium (mg)
Males 15-18 RDAs	66	176	59	1000	10	10	60	1.5	1.8	20	2.0	200	2.0	1200	1200	400	12	15	50
Vegetarian males 17.4 ± 0.5	52±5	168±4	73±12	1598 ±267	1.7 ±0.8	36±12	101 ±44	1.5 ±0.3	1.7 ±0.4	13,4 ±3.8	1.4 ±0.4	246 ±64	4.5 ±1.8	1126 ±342	1466 ±317	368 ±103	15±4	9±2	68 ±60
Non-vegetarian males 17.6 ± 1.1	57±8	169±7	107±31	1671 ±991	1.8 ±1.2	35±17	119 ±84	1.6 ±0.5	2.1 ±1.0	22.1 ±7.4	1.9 ±0.7	363 ±163	9.4 ±5.8	992 ±498	1662 ±602	386 ±129	17±6	17±6	69 ±34
Females 15-18 RDAs	55	163	44	800	10	8	60	1.1	1.3	15	1.5	180	2.0	1200	1200	300	15	12	50
Vegetarian females 17.4 ± 0.6	47±6	155±6	54±12	1278 ±496	1.0 ±0.6	36±20	108 ±49	1.1 ±0.3	1.3 ±0.5	9.9 ±3.2	1.1 ±0.4	205 ±62	2.8 ±1.5	875 ±291	1065 ±277	262 ±64	11±4	7±2	43 ±14
Non-Vegetarian females 17.5 ± 0.8	50±11	155±6	82±32	1498 ±1096	1.3 ±0.9	29±14	93 ±63	1.2 ±0.4	1.6 ±0.7	16.4 ±6.7	1.5 ±0.6	260 ±113	8.0 ±5.7	765 ±372	1235 ±450	287 ±94	13±5	13±6	51 ±26

* The allowances, expressed as average daily intakes over time, are intended to provide for individual variations among most normal persons as they live in the United States under usual environmental stresses.

≠ The median weights and heights of those under 19 years of age were taken from Hamill et al., 1979.

Both non-vegetarian and vegetarian males and females in this study were shorter and weighed less than males and females in the United States (Hamill et al., 1979). The intake of protein as well as vitamins A, D, E and C of all these subjects were higher than the RDAs, while thiamin and riboflavin intakes were similar to the RDAs. Niacin and vitamin B_6 intake of non-vegetarian males and females were almost equal to the RDA, while niacin intake of vegetarian males and females was almost one-third less than the RDA. Folic acid (or folate) intake was greater than the RDA in both males and females in both non-vegetarian and vegetarian categories. Vitamin B_{12} intake of vegetarian females was slightly higher than the RDA, while that of the non-vegetarian females, vegetarian and non-vegetarian males was four times greater than the RDA.

The calcium intake of these subjects was lower than the RDA; the intake of non-vegetarian females was almost one-third lower. The phosphorus intake of non-vegetarian males and females and that of vegetarian males was higher than the RDA while that of vegetarian females was lower. Magnesium intake of all the subjects was lower than the RDA. Iron intake of males was higher than the RDA, while the intake of non-vegetarian females was lower and that of vegetarian females was the lowest. The zinc intake of non-vegetarian males and females was higher than the RDA, while that of vegetarian males and females was lower than the RDA. The selenium intake of all these subjects was close to that of the RDA.

TABLE 4.11 Levels of biochemical risk factors of coronary heart disease of non-vegetarian and vegetarian subjects

	Non-vegetarian		Vegetarian	
	Males (n = 148)	Females (n = 142)	Males (n = 11)	Females (n = 20
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Total cholesterol (mmol/l)	[a]4.32 ± 0.71	4.39 ± 0.60	[a]3.74 ± 0.34	4.16 ± 0.71
HDL-C (mmol/l)	1.15 ± 0.24	1.28 ± 0.27	1.18 ± 0.16	1.19 ± 0.23
LDL-C (mmol/l)	[b]2.81 ± 0.64	2.73 ± 0.60	[b]2.20 ± 0.31	2.56 ± 0.68
% HDL of total cholesterol	[c]26.6 ± 8.4	29.2 ± 8.6	[c]31.6 ± 7.6	28.6 ± 9.1
Triglycerides (mmol/l)	0.79 ± 0.35	0.82 ± 0.30	0.78 ± 0.33	0.92 ± 0.40
Fibrinogen (mg/dl)	246.3 ± 60.7	273.5 ± 56.5	228.4 ± 34.7	293.1 ± 42.1
Factor VII (% of N)	77.4 ± 8.7	79.23 ± 13.1	80.0 ± 5.1	83.6 ± 5.8

Means with the same symbols differ significantly ($p < 0.05$)

Levels of biochemical risk factors of CHD of non-vegetarian and vegetarian subjects are given in Table 4.11.

The mean serum total cholesterol (TC) level of vegetarian females was lower than that of non-vegetarian females, although this was not significant at the 5% level, while that of vegetarian males was significantly lower ($p < 0.05$) than that of non-vegetarian males. The mean serum HDL-C level of males and females of both groups was not significantly different. The mean serum LDL-C level of vegetarian females was slightly lower, although not significantly, than that of non-vegetarian females, while that of vegetarian males was significantly lower ($p < 0.05$) than that of non-vegetarian males. The mean serum TG level of males and females of both groups did not differ significantly. The mean plasma fibrinogen and factor VII (% of normal) levels of males and females in both groups did not differ significantly.

The mean TC value of vegetarian females was within the normal range, i.e. below 4.1 mmol/l (Rossouw et al., 1988). Mean TC values of vegetarian males and non-vegetarian males and females, however, were greater than 4.1 mmol/l placing these subjects in the moderate-risk group for CHD (Rossouw et al., 1988).

The mean HDL-C value of these subjects was greater than 1.0 mmol/l. It has been recommended that the value of 1.0 mmol/l (> 35 mg/dl) be used to define an action limit for HDL-C below which the risk of CHD increases (Rossouw et al., 1988). Mean triglyceride (TG) values of these subjects

were well below the recommended value of 2.3 mmol/l (200 mg/dl) above which values should be regarded as abnormal.

Using the age-specific cut-off points (Rossouw et al., 1988) for TC (i.e. values below 4.1 mmol/l for 16- to 20-year olds being regarded as normal, between 4.1 mmol/l and 5.4-5.5 mmol/l as moderate-risk and above 5.5 mmol/l as high-risk), the distribution of subjects at low-, moderate- or high-risk for CHD is given in Table 4.12.

Of the female subjects, 2 (10%) vegetarian and 7 (5%) non-vegetarian, had TC values in the high- risk for CHD category, and of the male subjects, 7 (4.8%) non-vegetarians fell in this category. The number and percentages of subjects with fibrinogen levels in the normal and greater than 250 mg/dl range are also included.

Fibrinogen levels (> 250 mg/dl) were found in 48.9% (157/321) of these subjects. As shown in Table 4.2, 21.2% of these subjects were cigarette smokers. Cigarette smoking has been associated with high levels of fibrinogen in many studies (Meade et al., 1987). This could partly explain the higher fibrinogen levels found amongst subjects in this study particularly among the male subjects.

TABLE 4.12 Distribution of subjects at low-, moderate- and high-risk for coronary heart disease based on total cholesterol and fibrinogen levels

Variables		Non-vegetarian		Vegetarian	
		Males (n = 148)	Females (n = 142)	Males (n = 11)	Females (n = 20)
Total cholesterol:					
Low-risk	(n)	64	53	9	13
(< 4.1 mmol/l)*	(%)	43.2	37.3	81.8	65.0
Moderate-risk	(n)	77	82	2	5
(4.1 mmol/l-5.5 mmol/l)*	(%)	52.0	57.7	18.2	25.0
High-risk	(n)	7	7	0	2
(> 5.4 mmol/l)*	(%)	4.8	5.0	0	10.0
Fibrinogen:					
Normal range	(n)	94	56	9	5
(≤ 250 mg/dl)**	(%)	63.5	39.4	81.8	25.0
Elevated levels	(n)	54	86	2	15
(> 250 mg/dl)**	(%)	36.5	60.6	18.2	75.0

* (Rossouw et. al., 1988).

** Based on levels reported in prospective epidemiological studies (Wilhelmsen et. al., 1984; Stone and Thorp, 1985; Meade et. al., 1986a; Kannel et. al., 1987b).

It should be noted that of the total of 16 subjects whose TC values were in the high-risk category, 9 subjects (56.3%) also had fibrinogen levels greater than 250 mg/dl, while 89 of a total of 166 subjects (53.6%) with TC values in the moderate-risk category also had fibrinogen levels greater than 250 mg/dl. Thus, a very high percentage (30.5% or 98 subjects) had high levels of both TC and fibrinogen.

In the present study, one male non-vegetarian subject who was on a body-building diet swallowed four raw eggs every day. His energy intake is almost 3 times (30 659 kJ) that of the mean energy intake of non-vegetarian males. Macronutrient and micronutrient intake followed the same pattern.

This subject's serum cholesterol level was in the moderate to high risk range, i.e. 4.99 mmol/l; HDL-C was 0.88 mmol/l, LDL-C was 3.4 mmol/l, percentage HDL of TC was 21%, and TG was 1.47 mg/dl. His height was 171.0 cm, weight 59.5 kg, and mid-upper-arm circumference 26.6 cm. Fibrinogen level was high (> 250 mg/dl) 268 mg/dl and factor VII (% of N) was 85.8% of N.

In Table 4.13, significant Pearson correlation coefficients between measured variables are given. It should be kept in mind that these correlations only demonstrate associations between variables, and do not explain or indicate all determinants of a particular variable.

The expected high positive correlations of total serum cholesterol with LDL-C were observed. The positive correlation of TC with TG indicates that those subjects with high TC levels also tend to have higher TG levels. The positive significant correlations of TC and also of LDL-C with C20:4, vitamin B_{12}, vitamin A, MUFAT, dietary cholesterol and animal protein, illustrates the detrimental effect of nutrients found in foods from animal products on TC. Of particular interest is the relationship between dietary cholesterol and serum cholesterol, which has not been found in several other studies (Gordon et. al., 1981; Dawber et. al., 1982). The negative correlation of TC with plant protein may be an indication of a beneficial effect of plant foods, containing dietary fibre, on TC. However, no correlation was found between TC and dietary fibre.

The negative correlation of TG with HDL-C and percentage HDL-C of TC illustrates that raised TG levels are often accompanied by decreased HDL levels (Wolmarans et al., 1989). TG showed a weak but significant positive correlation with plasma fibrinogen. As can be expected, TG correlated positively with weight and mid-upper-arm circumference, showing the influence of obesity on TG, even in a group with a normal mean BMI. The significant positive correlation between weight, blood pressure and mid-upper-arm circumference confirms existing knowledge of the effect of overweight on blood pressure.

The significant positive correlation between fibrinogen and mid-upper-arm circumference, weight and TG illustrates a possible effect of overweight on these variables. It therefore seems that the factors that increase TG, may also increase fibrinogen levels.

A negative correlation between fibrinogen and fibre and plant protein was observed. This compares well with the findings of a Welsh study (Fehily et al., 1986).

The correlation between the different nutrients illustrates the clustering of specific nutrients in a specific source, i.e. fibre in foods rich in plant protein and carbohydrate and fats in foods with high energy density.

TABLE 4.13 Interrelationships among clinical and dietary variables (Pearson correlation coefficients)

VARIABLES	ALL SUBJECTS (n = 321)		Non-Vegetarian MALE (n = 148)		Non-Vegetarian FEMALE (n = 142)		Vegetarian MALE (n = 11)		Vegetarian FEMALE (n = 20)	
	r	p	r	p	r	p	r	p	r	p
TOTAL CHOLESTEROL										
x LDL-Cholesterol	0.9	0.0001	0.9	0.0001	0.1	0.0001	0.9	0.0001	0.9	0.0001
x % HDL-C of TC	-0.4	0.0001	-0.4	0.0001	-0.3	0.0001	-0.5	N.S 0.152	-0.6	0.0075
x HDL-Cholesterol	0.3	0.0001	0.3	0.0007	0.4	0.0001				
x TG	0.2	0.0001	0.2	0.003	0.2	0.018			0.4	0.0547
ARACHIDONIC ACID										
x (C20-4)	0.2	0.007	0.2	0.078			0.6	0.060		
x Vit B_{12}	0.2	0.010	0.1	0.104	0.1	N.S. 0.340				
x Animal Protein	0.1	0.017	0.1	0.100						
x Vita (RE)	0.1	0.028	0.1	0.156	0.1	0.102			0.4	N.S. 0.0802
x MUFAT (C16-1)	0.1	0.037	0.2	0.065						
x Plant Protein	-0.1	0.040	-0.1	0.137						
x Mid-upper-arm circumference	0.1	0.030			0.1	0.111	-0.5	N.S. 0.1113		
x Dietary Cholesterol	0.121	0.042					0.5	0.0997	0.5	0.0437
TRIGLYCERIDE										
x % HDL-C of TC	-0.5	0.0001	-0.4	0.0001	-0.5	0.0001	-0.7	0.0185	-0.5	0.0279
x HDL	-0.3	0.0001	-0.3	0.0001	-0.4	0.0001	-0.7	0.0262		
x TC	0.2	0.0001	0.2	0.003	0.2	0.018			0.4	0.0547
x LDL	0.2	0.0003	0.1	0.094	0.2	0.037				
x WT	0.2	0.001	0.1	0.131	0.4	0.0001			0.3	0.1988
x Mid-upper-arm circumference	0.2	0.006	0.1	0.174	0.3	0.0004			0.4	0.1008
x BP Systolic	0.2	0.012	0.2	0.009	0.1	N.S. 0.299				
x BP Diastolic	0.2	0.030	0.2	0.054	0.2	0.045	0.5	N.S. 0.131		
x Fibrinogen	0.1						0.7	0.0203	0.3	N.S. 0.1859
HDL										
x % HDL-C of TC	0.7	0.0001	0.7	0.0001	0.7	0.0001	0.8	0.0023	0.8	0.0001
x TG	-0.3	0.0001	-0.3	0.0001	-0.4	0.0001	-0.7	0.0262	-0.2	N.S. 0.3075
x TC	0.3	0.0001	0.3	0.0007	0.4	0.0001				
x Dietary Cholesterol	-0.2	0.004	-0.2	0.072			0.6	0.0753		
x Total Fat	-0.2	0.001								
x KJoul	-0.2	0.0004	-0.2	0.046						
x Fibre	-0.2	0.0008	-0.2	0.049						
x Weight	-0.2	0.0004			-0.2	0.031				
x Mid-upper-arm circumference	0.2	0.0001			-0.2	0.006			-0.5	0.0320
x Protein	-0.2	0.0005	-0.2	0.031	-0.1	0.140	0.3	N.S. 0.3753		
x Thiamine	0.2	0.0001	-0.2	0.021						
x Phosphorus	0.2	0.0003	-0.2	0.054			0.568	0.0684		
x Magnesium	-0.2	0.0004	-0.2	0.030						
x Sodium	-0.2	0.0005	-0.2	0.041						
x Zinc	-0.2	0.0005	-0.2	0.014			0.498	0.1185		
x C18-3	-0.2	0.0004	-0.2	0.066					0.487	0.0343
x Plant Protein	-0.2	0.0006	-0.2	0.041						
LDL										
x TC	0.9	0.0001	0.9	0.0001	0.9	0.0001	0.9	0.0001	0.9	0.0001
x % HDL-C of TC	-0.6	0.0001	-0.6	0.0001	-0.6	0.0001	-0.6	0.0635	-0.7	0.0002
x Animal Protein	0.2	0.0002	0.2	0.029	0.1	N.S. 0.218				
x Tryptophan	0.20	0.0005	0.2	0.053					0.4	N.S. 0.0757
x Dietary Cholesterol	0.2	0.0006							0.5	0.0153
x B_{12}	0.2	0.0007	0.2	0.032						
x Zinc	0.2	0.0007	0.2	0.049						
x C20-4	0.2	0.001	0.2	0.038						
x TG	0.2	0.007								
x Total Fat	0.2	0.032	0.2	0.037						

x Weight	0.2	0.007			0.2	0.048	-0.8	0.0031		
x MUFAT	0.1	0.018	0.2	0.017						
% HDL-C of TC										
x HDL	0.7	0.0001	0.7	0.0001	0.7	0.0001	0.8	0.0023	0.8	0.0001
x LDL	-0.6	0.0001	-0.6	0.0001	-0.6	0.0001	-0.6	N.S. 0.0635	-0.7	0.0002
x TG	-0.5	0.0001	-0.4	0.0001	-0.5	0.0001			-0.5	0.0279
x TC	-0.4	0.0001	-0.4	0.0001	-0.3	0.0002	-0.5	N.S. 0.1520	-0.6	0.0075
x Protein	-0.3	0.0001	-0.2	0.009			0.3	N.S. 0.3095		
x Animal Protein	-0.2	0.0001	-0.2	0.008	-0.2	0.038				
x Weight	-0.3	0.0001			-0.3	0.001	0.03	N.S. 0.2537		
x Mid-upper-arm circumference	-0.3	0.0001	-0.2	0.009	-0.3	0.0001			-0.5	0.0257
HEIGHT										
x Weight	0.5	0.0001	0.5	0.0001	0.5	0.0001			0.3	N.S. 0.155
x KJoul	0.4	0.0001								
x Total Cholesterol	0.4	0.0001								
x MUFAT	0.3	0.0001	0.2	0.009					-0.3	N.S. 0.2912
x SATFAT	0.3	0.0001	0.3	0.004						
x Total Fat	0.3	0.0001	0.2	0.014						
WEIGHT										
x Mid-upper-arm circumference	0.7	0.0001	0.8	0.0001	0.7	0.0001	0.8	0.0046	0.8	0.0001
x Height	0.5	0.0001	0.5	0.0001	0.4	0.0001			0.3	N.S. 0.1550
x BP Diastolic	0.3	0.0001	0.2	0.015	0.2	N.S. 0.096				
x BP Systolic	0.3	0.0001	0.2	0.007	0.2	0.012			0.4	N.S. 0.0749
x KJoul	0.2	0.0001					0.7	0.0093		
x Calcium	0.2	0.0001					0.7	0.0091		
x MUFAT	0.2	0.0003	0.2	0.015					0.2	N.S. 0.3905
x Total Cholesterol	0.2	0.0003								
x Age at Menarche					-0.1	N.S. 0.299				
MID-UPPER-ARM CIRCUMFERENCE										
x Weight	0.8	0.0001	0.8	0.0001	0.9	0.0001	0.8	0.0046	0.8	0.0001
x % HDL-C of TC	-0.3	0.0001	-0.2	0.009	-0.3	0.0002			-0.5	0.0257
x Height	0.3	0.0001			0.3	0.0002				
x BP Systolic	0.3	0.0001			0.3	0.002			0.4	N.S. 0.1246
x BP Diastolic	0.3	0.0001			0.3	0.001	0.6	N.S. 0.0725		
x LDL	0.2	0.0009			0.2	0.0198	-0.6	0.0404		
x TG					0.3	0.0004			0.4	N.S. 0.1008
FIBRINOGEN										
x Mid-upper-arm circumference	0.2	0.003	0.1	N.S. 0.312	0.4	0.0001			0.3	N.S. 0.1932
x Plant Protein	-0.2	0.006								
x Factor VII	0.2	0.005	0.1	N.S. 0.283	0.2	0.049			0.3	N.S. 0.1731
x Total Cholesterol	-0.2	0.012								
x Fibre	-0.1	0.020					-0.4	N.S. 0.2508		
x Weight	0.1	0.020	0.1	0.189	0.4	0.0001				
x TG	0.1	0.030			0.2	0.077			0.3	N.S. 0.1859
FACTOR VII										
x Fibrinogen	0.2	0.0047	0.1	0.283	0.2	0.049			0.3	0.1731
x C18-4	-0.2	0.019			0.2	0.030				
K JOUL										
x TG	0.2	0.0003								
x HDL	-0.2	0.0004								
x Total Fat					0.9	0.0001				
x Total Cholesterol					0.9	0.0001	0.9	0.0001	0.9	0.0001

BP SYSTOLIC

x BP Diastolic	0.7	0.0001	0.6	0.0001	0.7	0.0001			0.4	N.S. 0.0621
x Weight	0.3	0.0001	0.2	0.007	0.3	0.0006			0.4	N.S. 0.0749
x Mid-upper-arm circumference	0.3	0.0001	0.1	0.105	0.3	0.002			0.4	N.S.
x TTrans	0.2	0.0001	0.2	0.062	0.2	0.025			0.2	N.S. 0.3895
x SATFAT	0.2	0.0001								
x KJoul	0.2	0.0008								

BP DIASTOLIC

x BP Systolic	0.7	0.0001	0.6	0.0001	0.7	0.0001				
x WT	0.3	0.0001	0.2	0.015	0.3	0.0001				
x Mid-upper-arm circumference	0.3	0.0001	0.1	0.164	0.3	0.0001	0.6	N.S. 0.0725		
x TTrans	0.2	0.002	0.2	0.091						
x SATFAT	0.2	0.004	0.1	0.193			0.7	0.0222	0.3	0.2067
x KJoul	0.1	0.028								

PLANT PROTEIN

x Fibre	0.8	0.0001	0.8	0.0001	0.8	0.0001	0.9	0.0002	0.9	0.0001
x Total Cholesterol	0.7	0.0001	0.6	0.0001	0.8	0.0001			0.6	0.0101
x KJoul	0.7	0.0001	0.6	0.0001	0.7	0.0001			0.6	0.0140
x Total Fat	0.6	0.0001	0.4	0.0001	0.6	0.0001				
x MUFAT	0.5	0.0001			0.5	0.0001				
x PUFAT	0.4	0.0001	0.4	0.0001						

ANIMAL PROTEIN

x MUFAT	0.7	0.0001	0.7	0.0001	0.7	0.0001				
x KJoul	0.6	0.0001	0.7	0.0001						
x SATFAT	0.6	0.0001			0.7	0.0001				
x Total Fat	0.6	0.0001								

TOTAL FAT

x MUFAT	0.9	0.0001	0.9	0.0001	0.9	0.0001	0.8	0.0020	0.8	0.0001
x KJoul	0.9	0.0001	0.9	0.0001			0.7	0.0098	0.9	0.0001
x SATFAT	0.9	0.0001	0.9	0.0001	0.9	0.0001	0.7	0.0109	0.6	0.0125
x Sodium	0.7	0.0001			0.7	0.0001				
x T Trans	0.6	0.0001			0.7	0.0001				
x Mid-upper-arm circumference							0.7	0.0197		
x PUFAT									0.7	0.0024

SATFAT

x C18-0	1.0	0.0001	1.0	0.0001	0.9	0.0001	0.9	0.0001	0.9	0.0001
x Total Fat	0.9	0.0001	0.9	0.0001	0.9	0.0001	0.7	0.0109		
x MUFAT	0.8	0.0001	0.8	0.0001	0.9	0.0001	0.4	N.S. 0.1845	0.6	0.0085
x KJoul	0.8	0.0001	0.8	0.0001	0.8	0.0001	0.5	N.S. 0.1586		

MUFAT

x C18-1	1.0	0.0001	1.0	0.0001	1.0	0.0001	1.0	0.0001	1.0	0.0001
x Total Fat	0.9	0.0001	0.9	0.0001	0.9	0.0001	0.8	0.0020	0.8	0.0001
x KJoul	0.9	0.0001	0.9	0.0001	0.9	0.0001	0.7	0.0249	0.8	0.0001
x SATFAT	0.8	0.0001	0.8	0.0001	0.9	0.0001	0.4	0.1845	0.6	0.0085
x Dietary cholesterol	0.7	0.0001	0.7	0.0001						

PUFAT

X C18-2	1.0	0.0001	1.0	0.0001	0.9	0.0001	0.9	0.0003	1.0	0.0001
x C22-0	0.8	0.0001	0.8	0.0001	0.8	0.0001	0.7	0.0205	1.0	0.0001
x C20-0	0.7	0.0001	0.7	0.0001	0.7	0.0001	0.7	0.0129	0.8	0.0001
x C24-0	0.7	0.0001	0.7	0.0001	0.6	0.0001	0.5	0.080 N.S.	1.0	0.0001
x Total Fat	0.7	0.0001	0.7	0.0001	0.7	0.0001	0.4	0.1934 N.S.	0.7	0.0024 N.S.
x KJoul	0.6	0.0001	0.6	0.0001	0.6	0.0001	0.4	0.1878	0.4	0.0834
x MUFAT	0.5	0.0001			0.5	0.0001				
x C20-1	0.5	0.0001	0.5	0.0001	0.3	0.0005	0.6	0.0359	0.9	0.0001
x Total Cholesterol	0.4	0.0001	0.5	0.0001	0.4	0.0001				
x Plant Protein	0.4	0.0001	0.4	0.0001	0.4	0.0001				
x Fibre	0.3	0.0001			0.3	0.0015				

DIETARY CHOLESTEROL

x KJoul	0.7	0.0001	0.7	0.0001						
x SATFAT	0.7	0.0001	0.8	0.0001	0.7	0.0001			0.8	0.0001
x MUFAT	0.7	0.0001			0.7	0.0001				
x Total Fat	0.7	0.0001	0.7	0.0001						

TOTAL CHOLESTEROL (diet.)

x KJoul	0.9	0.0001	0.9	0.0001	0.9	0.0001	0.9	0.0001	0.9	0.0001
x Fibre	0.7	0.0001	0.6	0.0001	0.7	0.0001	0.4	N.S. 0.2116		
x Total Fat	0.7	0.0001	0.7	0.0001	0.6	0.0001	0.4	N.S. 0.2059		
x SATFAT	0.6	0.0001			0.6	0.0001			0.6	0.0028
x MUFAT	0.7	0.0001	0.6	0.0001	0.6	0.0001			0.7	0.0005

DIETARY FIBRE

x Plant Protein	0.8	0.0001	0.8	0.0001	0.8	0.0001	0.9	0.0002	0.9	0.0001
x Total Cholesterol (diet.)	0.7	0.0001	0.6	0.0001	0.7	0.0001			0.6	0.0028
x KJoul	0.6	0.0001	0.6	0.0001	0.6	0.0001				
x Total Fat	0.5	0.0001			0.4	0.0001				
x MUFAT	0.4	0.0001			0.4	0.0001				
x SATFAT	0.4	0.0001								

Sodium

x KJoul			0.8	0.0001	0.9	0.0001	0.6	0.0495		

TABLE 4.14 Results of the stepwise regression analysis

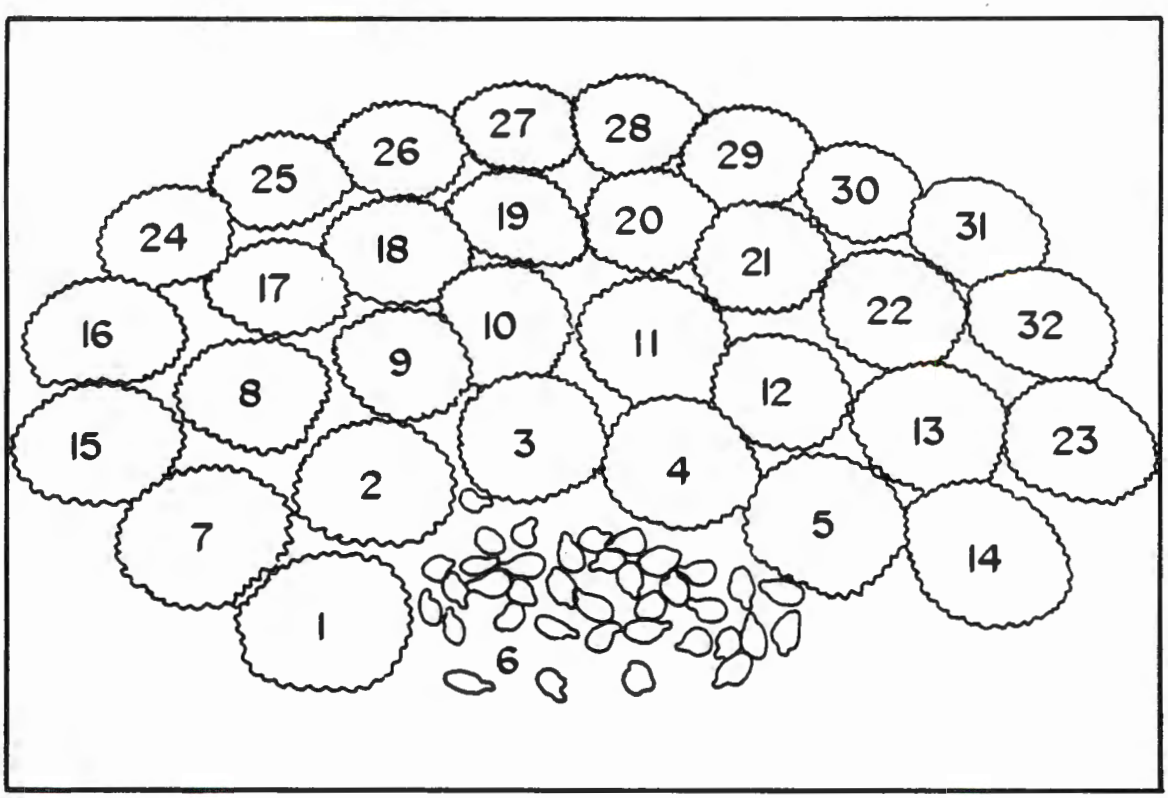
DEPENDENT VARIABLE INDEPENDENT VARIABLE	PARTIAL R^{**2}	MODEL R^{**2}	C(P)	F	PROB > F
TOTAL CHOLESTEROL					
LDL-C	0.8550	0.86	18142.5	1574.6	0.0001
HDL-C	0.1004	0.96	5396.4	599.2	0.0001
TG	0.0425	1.00	8.3	5303.5	0.0001
FIBRINOGEN					
Factor VII	0.0433	0.04	-5.6308	12.1	0.0006
% HDL of TC	0.0227	0.10	-15.7157	6.6	0.0105
Animal Protein	0.0092	0.10	-16.1980	2.7	0.1016
TRIGLYCERIDE					
LDL	0.0453	0.25	4856.01	16.1	0.0001
TC	0.5784	0.83	838.29	903.9	0.0001
HDL	0.1281	0.96	9.48	816.9	0.0001
C20-4	0.0008	0.96	5.51	4.9	0.0277
C8-0	0.0005	0.96	4.30	3.2	0.0733
LDL					
TC	0.8550	0.86	14994.7	1574.7	0.0001
HDL	0.0961	0.95	4880.6	523.2	0.0001
TG	0.0463	1.00	9.4	4775.4	0.0001
HDL					
% HDL of TC	0.5618	0.56	9675.30	342.3	0.0001
TC	0.3849	0.95	944.69	1923.4	0.0001
LDL	0.0064	0.95	800.91	36.4	0.0001
TG	0.0354	0.99	0.36	816.9	0.0001
C8-0	0.0002	0.99	-4.75	5.6	0.0189
C14-1	0.0001	0.99	-5.39	2.8	0.0967
FACTOR VII					
Fibrinogen	0.0433	0.04	-20.2926	12.1	0.0006
C18-4	0.0197	0.06	-23.3204	5.6	0.0189
C20-3	0.0132	0.08	-24-7000	3.8	0.0526

The step-wise regression analyses (Table 4.14) must be seen as a scientific tool which may be used to examine the determinants of the variation of a particular variable. Thus, from the table it is clear that 85% of the variation in TC of this group of subjects, could be explained by variations in LDL-C. HDL-C contributed 10% and TG 4%. Only 10.4% of the variation in fibrinogen could be explained by the variables measured in this study.

In Tables 4.15-4.20 some typical Indian food ingredients and cooked dishes have been described as shown in the respective photographs.

TABLE 4.15 Key to photograph of a selection of condiments, spices, legumes and dhals used in Indian cooking.

1. Whole mustard seeds (raai).
2. Coriander seeds (whole "dhunia" seeds).
3. Turmeric powder ("haldi", "arad").
4. Red chillie powder ("Lal mirchi").
5. Cummin ("jeera", "jeero") seeds.
6. Cardamom or "elachi" (whole cardamom pods).
7. Chana dhal (split gram dhal).
8. Chana flour (gram or Besan flour).
9. Cinnamon sticks ("tuj").
10. Black pepper-corns ("Kali mirchi").
11. Cloves ("lawang").
12. Another variety of coriander (whole "dhunia") seeds.
13. Fenugreek ("methi") seeds.
14. Fennel ("saumnf") seeds.
15. Oil ("toovar") dhal.
16. Sugar beans.
17. Whole roasted chana seeds.
18. Whole chana seeds.
19. Whole wheat.
20. Chana dhal (split gram dhal).
21. Roasted whole pea dhal.
22. Pea dhal (split peas or "mattar" dhal).
23. Betel nuts.
24. Red/black beans.
25. Butter beans.
26. Red/brown beans ("baked beans").
27. Cape sugar beans ("vaal" dhal).
28. Masoor dhal (split masoor or "pink" dhal).
29. Whole "masoor" seeds.
30. Moong dhal (split).
31. Moong (green Chinese pea).
32. Moong dhal (green Chinese pea-split).

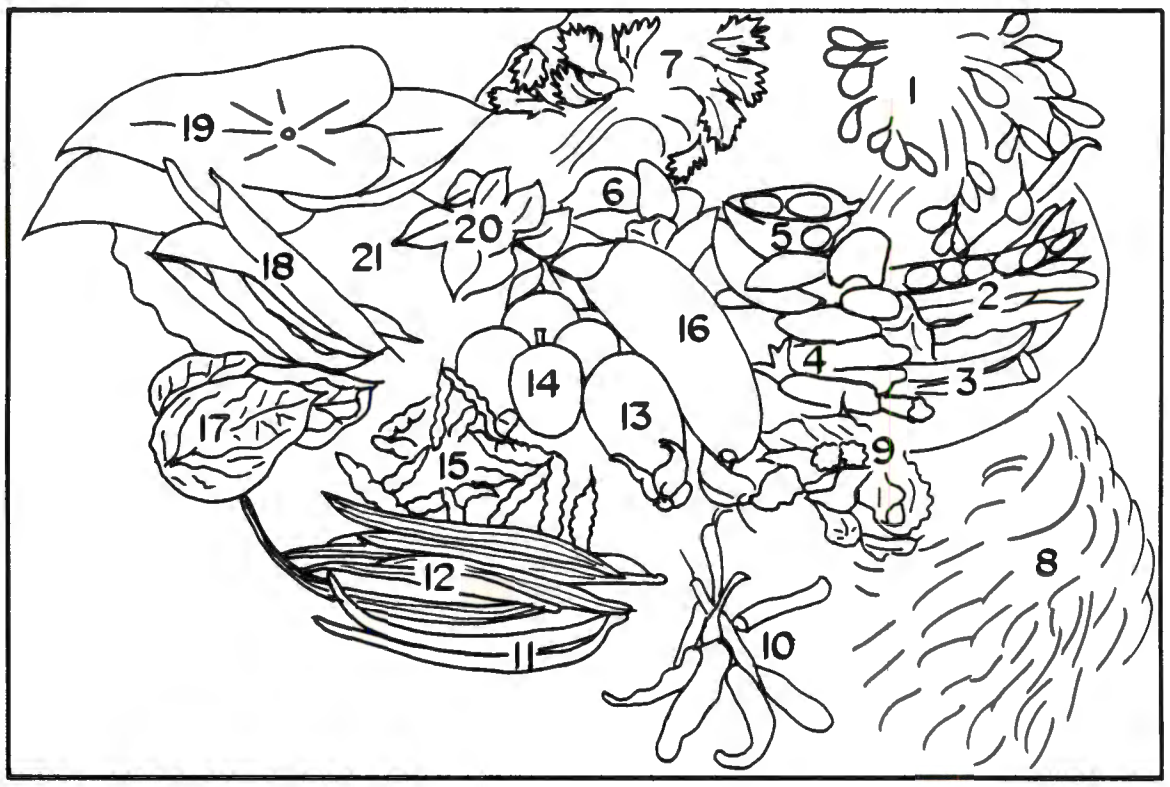


A SELECTION OF CONDIMENTS, SPICES, LEGUMES, DHALS AND SEEDS

USED IN INDIAN COOKING

Table 4.16 Key to photograph of a selection of Indian vegetables.

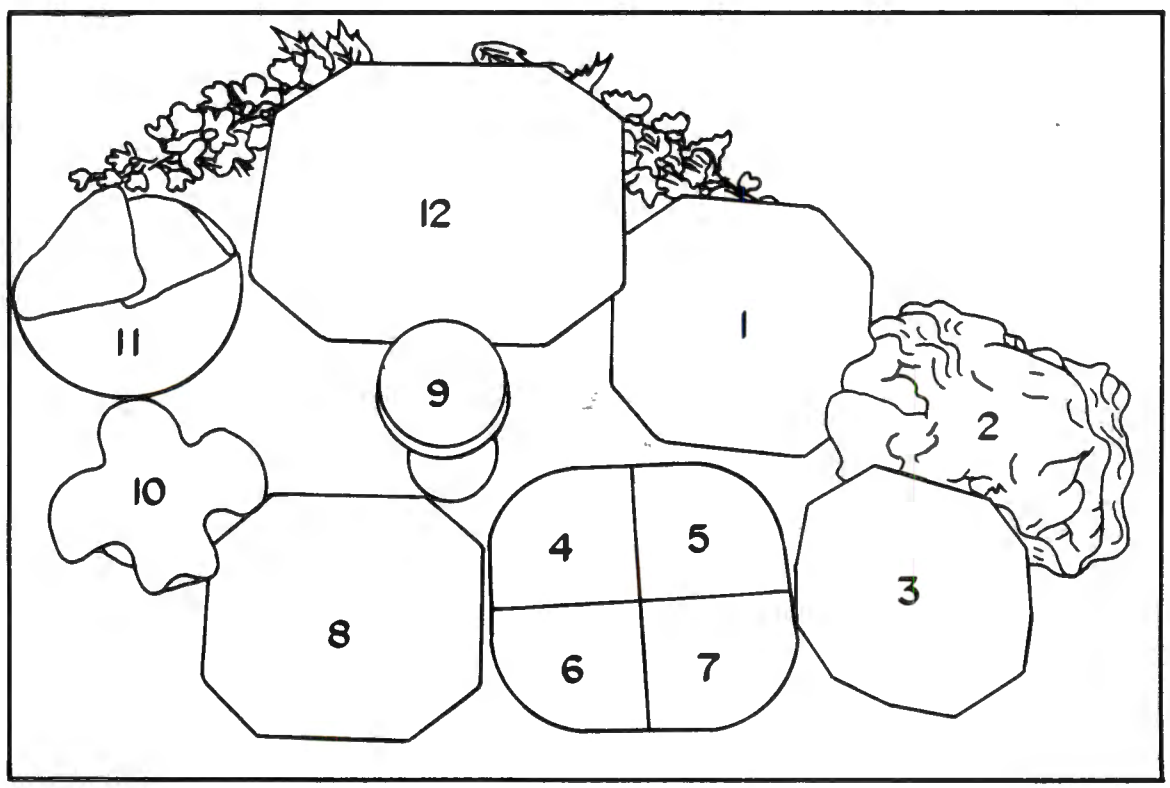
1. "Methi bhaji" (fenugreek leaves).
2. "Ghadra" beans (flat Indian beans).
3. Pickled "bhinda" (okra).
4. Baby brinjals.
5. "Dubla" beans (green Indian beans).
6. "Papri" (very flat Indian beans; often cooked with the pod).
7. "Dhunia" (coriander leaves).
8. "Hoowa" or "soowa bhaji" (dil "bhaji").
9. "Karela" (bitter gourd).
10. Red and green chilies.
11. "Chori" beans (green, small Indian beans).
12. Drumsticks ("hekta ni phalli").
13. Brinjal (egg-plant).
14. Green mangoes (used to make mango achaar).
15. "Toovar" (Indian type peas).
16. "Dodhi" ("doodhi") (summer type squash).
17. Betel-nut leaves.
18. Green butter beans.
19. Madumbi leaves.
20. "Karhi" leaves.
21. Madumbi roots.



A SELECTION OF INDIAN VEGETABLES

Table 4.17 Key to photograph of typical non-vegetarian food dishes

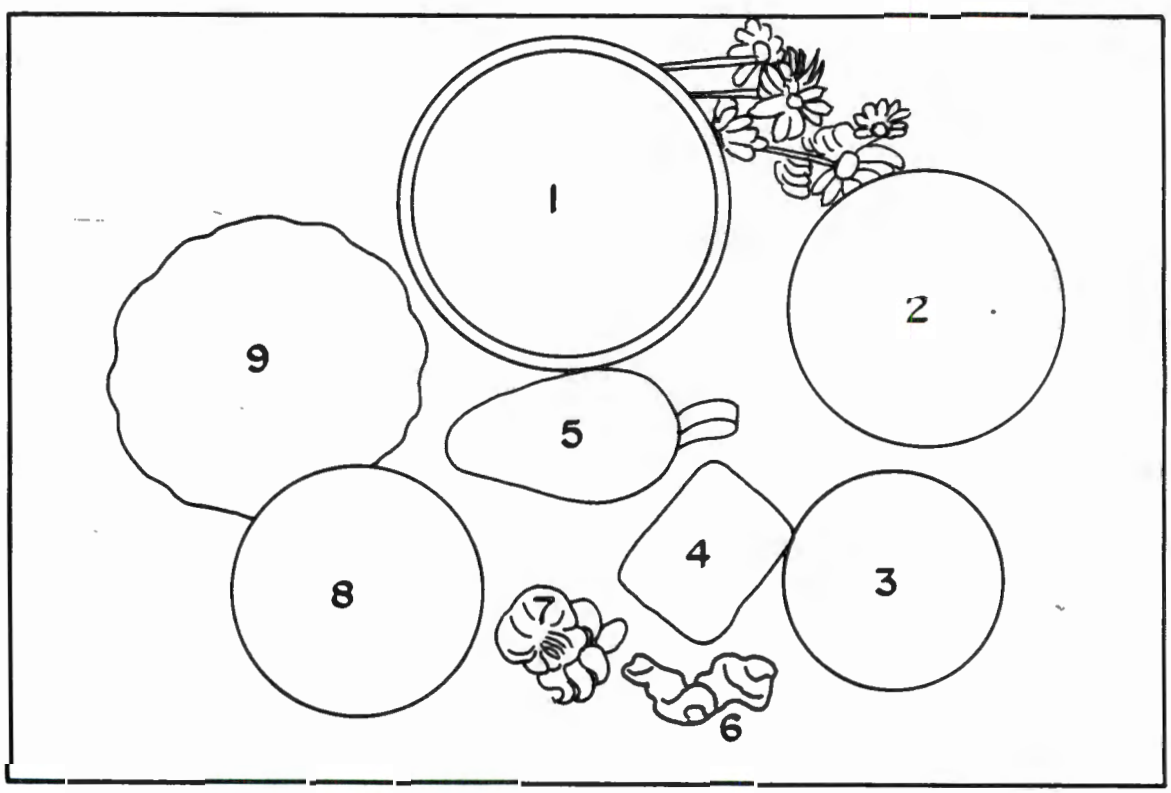
1. Curried mutton Chops.
2. Papadums ("papads").
3. Chicken and Indian and Western vegetable stew.
4. "Kachoomar" (cubed Western vegetable salad with dressing.
5. Mango and "methi achaar" (or pickles) in sun oil.
6. Mixed vegetable "achaar".
7. Lemon and red chillie achaar.
8. Chicken and potato curry.
9. "Dahi" (spiced sour milk or yoghurt).
10. Western vegetable salad - sliced.
11. "Roti" (brown and white flour).
12. Chicken "briyani" (rice, yoghurt, eggs, potatoes, black masoor and spices).



TYPICAL NON-VEGETARIAN FOOD DISHES

Table 4.18 Key to photograph of typical South-Indian vegetarian food dishes

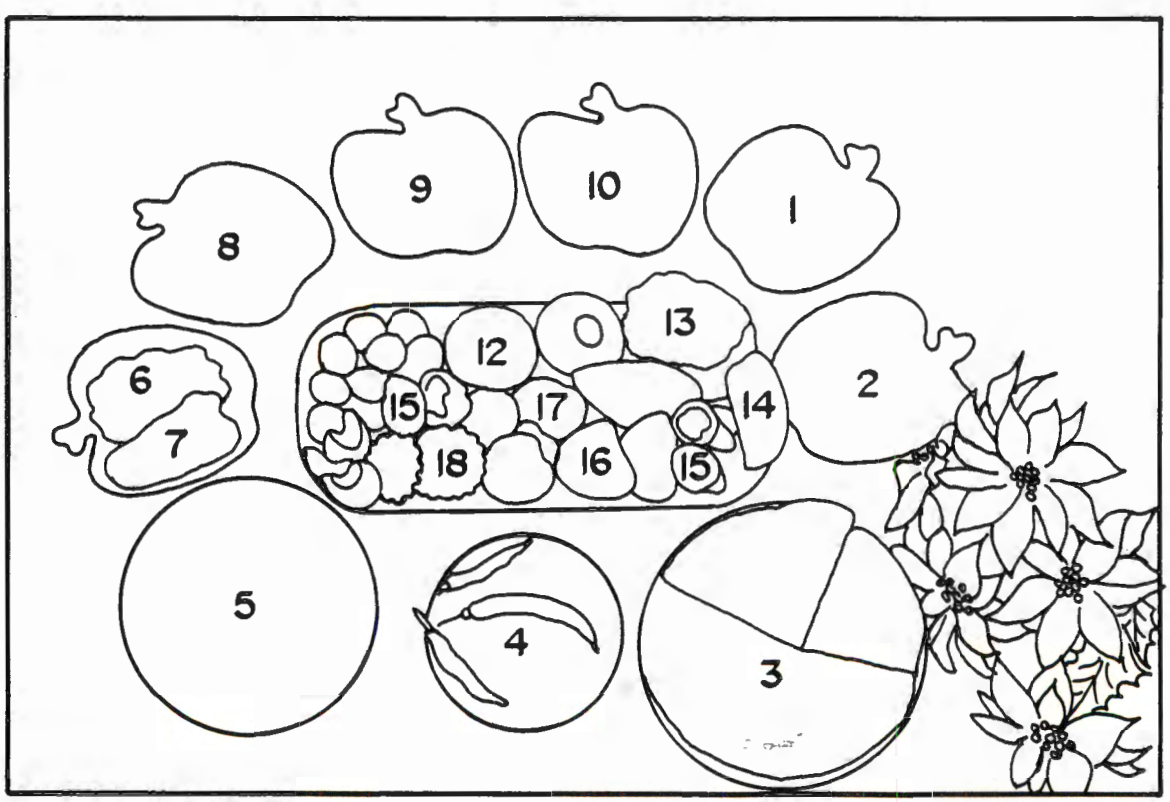
1. "Dokra" (dumplings made with spiced cake flour) curry with "drumsticks" (fibrous Indian vegetable).
2. Western vegetable salad (cubed and shredded) with cheese.
3. "Bajri roti" ("roti" made with wheat flour).
4. A block of "tamarind" (sour tasting) (also known as "aamli").
5. Tamarind soup (Mulgathunee mulga = chillie, thunee = water) or King's soup also known as the Englishman's soup.
6. Whole root ginger.
7. Cloves of garlic.
8. Potato and tomato curry.
9. Cooked rice.



TYPICAL SOUTH-INDIAN VEGETARIAN FOOD DISHES

Table 4.19 Key to photograph of typical North-Indian vegetarian food dishes and savouries

1. "Khuri" (sour milk or yoghurt soup).
2. Butter and sugar-bean with potato curry.
3. "Rotis".
4. Spinach curry with whole chillies.
5. "Chevra" (a mixture of "sev" spiced "chana" or Besan or gram flour vermicelli), "ganthia" (a thicker variety of spiced flour macaroni), peanuts and corn flakes.
6. "Chana dhal" curry.
7. Potato and oil dhal curry.
8. "Bhinda" curry (okra).
9. Moong dhal curry (green Chinese pea-split).
10. Rice with oil dhal and turmeric ("Khitchri").
11. "Bhajias" (spiced chana flour balls fried in deep oil).
12. Sweet Indian doughnuts.
13. "Khus khus puri" (poppy seed).
14. Banana "puri" (banana shaped pastry dipped in syrup).
15. "Moong dhal puri".
16. "Khopra puri" ("puris" filled with sweet coconut filling).
17. "Vadde" ("moong dhal" and spiced "chana" flour patties fried in deep oil.
18. "Laapsi puri" ("puris" with sweetened "laapsi" filling)



TYPICAL NORTH-INDIAN FOOD DISHES AND SAVOURIES

Table 4.20 Key to photograph of typical non-vegetarian savouries and sweetmeats (mithais).

SAVOURIES

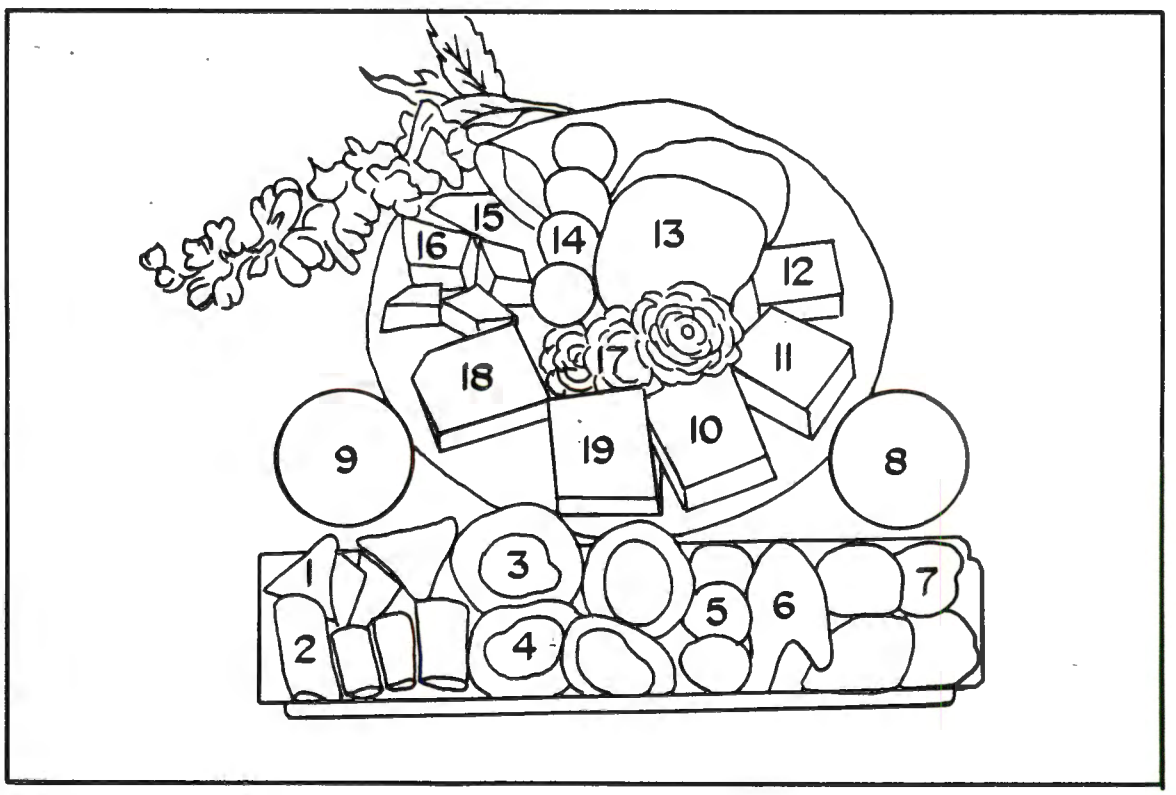
1. "Samoosas" (pyramid or triangle-shaped single-layered pastry filled with mutton or chicken mince).
2. Mince pies (butter pastry).
3. "Puri" (flat roti fried in deep oil).
4. "Patta" (madumbi leaf pinwheels with chana flour and spiced filling).
5. "Kebaabs" or "koftas" (spiced mince or meatballs).
6. Fish (spiced and fried).
7. Frikkadelles (spiced mince and bread patties) (or malay meatballs/ patties).
8. Tomato chutney (spiced and curry leaves used for garnishing).
9. "Dhunia" chutney (coriander leaves and chillies with spices).

SWEETMEATS

10. & 16. "Burfee" of two variations (klim milk and cream fudge).
11. & 12. Green and red "badaam halwa" (almond-Turkish delight).
13. "Sutherfeni" (or granny's hair as it is white and fragile and rolled into large pinwheels fried and dipped in syrup).
14. "Larwa" or "luddoo" ("chana" flour dough, minced, fried and rolled into golf balls).
15. "Goolab jamboo" or "jamun" (cake flour and condensed milk dough - deep fried.
17. "Jalebi" (pretzel-shaped cake and rice flour sweetmeat - deep fried and dipped in syrup).

18. "Khopra paak" (coconut fudge).

19. "Chana magaj" ("chana" flour fudge).



TYPICAL NON-VEGETARIAN SAVOURIES AS WELL AS SWEETMEATS ("MITHAIS")

CHAPTER 5

DISCUSSION

5.1 Introduction

In this section the results reported in Chapter 4 are discussed in more detail and comparisons are made with published results from other studies on similar groups of subjects. Although the number of subjects in some of the studies are quite small, these are the only data available for comparative purposes.

5.2 Anthropometry

Height

Although the number of vegetarians in the present study was small, it was observed that vegetarianism did not influence the height of either male or female subjects compared to non-vegetarians (omnivores) (Table 4.1). Mean age of these subjects was 17.5 years.

The height of these subjects can be compared with data from earlier studies (Rao and Sastry, 1977; Trivedi, 1977) carried out in India in regions where the South African Indians originate from (Appendix B) as well as with data from an earlier study (Walker and Walker, 1977) carried

out among Natal Indians and in the Transvaal among blacks (rural and urban) and whites. Rao and Sastry (1977) reported heights of 17 year- and 18 year-old well-to-do males only, while Trivedi (1977) reported heights of 17 year- and 18 year-old males and females from Gujerat University (GU) and from the Indian Council of Medical Research (ICMR), while Walker and Walker (1977) reported the heights of 18 year-old South African males and females.

Males

In the present study the mean height (± SD) of non-vegetarian males (n = 148) was 168.8 ± 6.6 cm and that of vegetarian males (n = 11) was 168.0 ± 4.1 cm. Mean height of 17 year-old GU males (n = 984) was 164.6 ± 6.8 cm, of ICMR males (n = 3 235), 161.4 ± 10.4 cm and of well-to-do males (n = 77), 173.0 ± 0.6 cm. Mean height of 18 year-old GU males (n = 818) was 166.0 ± 6.2 cm, of ICMR males (n = 3 006), 163.1 ± 9.6 cm and of well-to-do males (n = 169), 172.1 ± 0.5 cm. It must be noted that well- to-do 17 year-old males have been reported to be taller than 18 year-olds.

In South Africa, mean height (± SD) of Natal Indian males from the highest socio-economic group (n = 55) was 170.1 ± 4.6 cm, from the intermediate socio-economic group (n = 50), 168,6 ± 5.0 cm and from the relatively poor group (n = 50), 168.7 ± 4.3 cm. Mean (± SD) heights of Transvaal blacks from rural Ia area (n = 49) was 167.0 ± 5.5 cm, from

rural Ib area (n = 34), 166.7 ± 6.5 cm, rural II area (n = 63), 169.0 ± 7.1 cm, rural III area (n = 51), 168.8 ± 6.9 cm, from the urban area (n = 68), 168,5 ± 7.4 cm. Mean (± SD) height of Transvaal white males (n = 65) was 177.5 ± 4.3 cm (Walker and Walker, 1977). Mean height (± SD) of a small number of Western Transvaal 17 year-old white males (Van Zyl, 1983) (n = 15) was 170.3 ± 11.8 cm.

The mean height of males in the present study is similar to that of Indian males from Natal and the black adolescents (both rural and urban), and although they are slightly shorter than well-to-do Indian adolescents from India, they are taller than those from GU and the ICMR. They are shorter than white adolescents.

Females

In the present study, mean height (± SD) of non-vegetarian females (n = 142) was 155.3 ± 5.8 cm and that of vegetarian females (n = 20) was 155.4 ± 6.5 cm. Mean height (± SD) of females in the 17 year age-group from GU (n = 882) was 154.3 ± 5.9 cm, from the ICMR (n = 2 115), 151.5 ± 7.5 cm, in the 18 year age-group from GU (n = 374), 153.9 ± 5.7 cm, from the ICMR (n = 1 712), 151.7 ± 5.9 cm. It must be noted that the GU 17 year-olds were taller than the 18 year-olds. Mean height of 18 year-old Natal Indian females from the highest socio-economic group (n = 55) was 157.0 ± 4.7 cm, from the intermediate socio-economic group (n = 69), 158.5 ± 5.1 cm, from the relatively poor group (n = 75), 157.2 ± 5.3 cm. Subjects from the intermediate group were the tallest. Mean height of 18 year-old

white females (n = 71) from Johannesburg was 164.5 ± 7.6 cm, of black females from rural Ia area (n = 50) was 157.6 ± 5.6 cm, from rural Ib area (n = 39), 157.3 ± 5.9 cm, from rural II area (n = 64), 159.0 ± 5.5 cm, from rural III area (n = 58), 158.7 ± 5.8 cm, from the urban area (n = 60), 159.0 ± 6.0 cm. Mean height of 17 year-old Western Transvaal white females (n = 23) was 161.9 ± 6.1 cm.

The females in the present study were taller than the Indian females from India, but shorter than the Indian females from Natal. They were also shorter than the black females from the urban and different rural areas, and much shorter than white females from Johannesburg and the Western Transvaal.

However, although both males and females in the present study were slightly taller than the Indians of the same age-group from India who were from an environment close to the optimum because of their socio-economic circumstances (Rao and Sastry, 1977), it seems that they were shorter than Natal Indians, blacks from the urban and different rural areas and whites from Johannesburg and the Western Transvaal. This difference can thus probably be attributed to ethnicity and is probably not due to dietary or other environmental inadequacies (Walker and Walker, 1977). However, it should be noted that only limited published data was available on this age-group for South Africans and thus more research in this field is required.

Weight (Mass)

Males

In the present study, mean weight (± SD) (51.9 ± 5.0 kg) of vegetarian males (n = 11) was significantly lower ($p < 0.05$) than that (57.0 ± 8.1 kg) of non-vegetarian males (n = 148). Mean weight (± SD) of 17 year-old males from GU (n = 984) was 47.0 ± 6.6 kg, ICMR (n = 3 235), 45.7 ± 9.1 kg, the well-to-do group (n = 77), 57.9 ± 0.8 kg, Western Transvaal whites (n = 15), 65.0 ± 7.3 kg. Mean weight (± SD) of 18 year-old males from GU (n = 818) was 48.8 ± 6.7 kg, ICMR (n = 3 006), 47.4 ± 7.2 kg, well-to-do group (n = 169), 58.4 ± 0.5 kg, Natal Indians from the highest socio-economic group (n = 55), 53.9 ± 5.8 kg, from the intermediate socio-economic group (n = 50), 54.1 ± 5.6 kg, from the relatively poor group (n = 50), 53.8 ± 5.0 kg. Mean weight (± SD) of blacks from rural Ia area (n = 50) was 49.0 ± 6.3 kg, rural Ib area (n = 34), 47.5 ± 6.8 kg, rural II area (n = 63), 57.0 ± 7.2 kg, rural III area (n = 51), 56.4 ± 6.9 kg, urban blacks (n = 68), 57.0 ± 6.6 kg and Johannesburg whites (n = 65), 67.5 ± 7.2 kg. It is noteworthy that Natal adolescents from the intermediate socio-economic group were the heaviest.

In the present study, mean weight of Indian males was less than that of white males, but was more than that of the three groups of Natal Indians as well as that of blacks from two rural areas (Ia and Ib). Their weight was similar to that of black males from the latter two rural areas (II and III) and the urban area.

Females

In the present study, mean weight (± SD) of non-vegetarian females (n = 142) was 50.1 ± 11.0 kg and that of vegetarian females (n = 20) was 47.2 ± 6.2 kg. Mean weight (± SD) of 17 year-old females from GU (n = 882) was 42.4 ± 5.9 kg, ICMR group (n = 2 115), 42.4 ± 6.8 kg and the Western Transvaal group (n = 23), 60.8 ± 8.7 kg. Mean weight (± SD) of 18 year-old females from GU (n = 374) was 42.7 ± 6.0 kg, ICMR (n = 1 712), 42.4 ± 7.2 kg, Natal Indians from the highest socio-economic group (n = 55), 47.9 ± 4.1 kg, from the intermediate socio-economic group (n = 69), 47.0 ± 4.8 kg, from the relatively poor group (n = 75), 48.4 ± 5.1 kg, Johannesburg whites (n = 71), 57.1 ± 6.6 kg, blacks from rural Ia area (n = 50), 52.3 ± 7.7 kg, from rural Ib area (n = 39), 50.8 ± 7.9 kg, rural II area (n = 64), 56.6 ± 9.5 kg, rural III area (n = 58), 55.4 ± 9.2 kg, and the urban area (n = 60), 55.1 ± 9.9 kg.

Non-vegetarian females were heavier than vegetarians in the present study. Mean weight (± SD) of this latter group was similar to that of Natal Indian females. However, they weighed less than black females from the urban as well as rural areas and white females from Johannesburg and the Western Transvaal. Also, both groups from the present study were heavier than all three groups in India.

Quetelet or body mass index

Body mass index (BMI) (mean ± SD) of non-vegetarian males in the present study was 20.0 ± 6.4 kg/m^2, vegetarian males, 18.4 ± 4.6 kg/m^2, non-vegetarian females, 20.8 ± 6.8 kg/m^2 and vegetarian females 19.5 ± 6.2 kg/m^2. The weight of vegetarians was less than that of non-vegetarians. This resulted in the lower BMI of vegetarians.

In South Africa, values for BMI, stratified for age and gender, were recorded in the CORIS study among 3 white rural communities of the South-Western Cape (Rossouw et al., 1983) and for the coloured population of the Cape Peninsula (Steyn et al., 1985). The values given here are for the 15-24 year age-group. Mean BMI for males (n = 635) in the CORIS study was 22.9 ± 3.3 kg/m^2, for females (n = 658), 22.8 ± 3.6 kg/m^2, that for coloured males (n = 94), 20.9 ± 3.0 kg/m^2, that for coloured females (n = 103), 22.6 ± 4.3 kg/m^2. In the CORIS study BMI of males and females was similar, while that of coloured males was lower than that of coloured females. BMI of the subjects in the present study was lower than that recorded in the CORIS study as well as that of the coloured population.

Mid-upper-arm circumference

In the present study mean mid-upper-arm circumference (± SD) of non-vegetarian males (n = 148); was 24.0 ± 2.3 cm, which was significantly greater ($p < 0.05$) than that of vegetarian males (n = 11), 22.3 ± 1.8 cm. Mean Values (± SD) for mid-upper-arm circumference

of well-to-do males were reported in only one Indian study (Rao and Sastry, 1977); in the 17-year age-group (n = 77) it was 24.8 ± 0.2 cm and in the 18-year age group (n = 169) it was 25.4 ± 0.2 cm. Mean value (± SD) for mid-upper-arm circumference of Western Transvaal white males (n = 15) was 26.2 ± 1.9 cm.

Mean value (± SD) for mid-upper-arm circumference of non-vegetarian females (n = 142) in the present study was 22.8 ± 2.8 cm and that of vegetarian females (n = 20) was 22.0 ± 1.6 cm. Mean value (± SD) for mid-upper-arm circumference of Western Transvaal white females (n = 23) was 24.8 ± 2.3 cm.

It means that the mean mid-upper-arm circumference values of Western Transvaal white males and females were greater than those recorded in the present study.

5.3 Blood Pressure

Mean BP levels of the subjects in the present study were slightly lower compared with published data in this age-group of other ethnic groups in South Africa and Israel. Table 4.1 shows mean systolic BP of 111.0 ± 8.8 mmHg for both groups of females, 116.3 ± 10.3 mmHg for non-vegetarian males and 117.8 ± 8.5 mmHg for vegetarian males. Corresponding diastolic BP were 68.9 ± 7.7 mmHg, 67.8 ± 6.4 mmHg, 72.6 ± 8.5 mmHg and 72.4 ± 7.3 mmHg.

Mean systolic BP (± SD) of white males (n = 635) aged 15-24 years in the CORIS baseline study was 124.7 ± 12.4 mmHg and mean diastolic BP (± SD) was 75.3 ± 34.3 mmHg and mean systolic BP of females (n = 658) was 120.1 ± 11.1 mmHg and mean diastolic BP was 76.1 ± 9.1 mmHg (Rossouw et al., 1983).

In a cross-sectional study of risk factors for CHD in a random sample of 976 coloured people of the Cape Peninsula (Steyn et al., 1985), mean systolic BP (± SD) of males (n = 94) in the 15-24 year age-group was 120.4 ± 15.1 mmHg and the mean diastolic BP (± SD) was 76.3 ± 10.0 mmHg. The mean systolic BP (± SD) of females (n = 103) in the 15-24 year age-group was 114.9 ± 10.9 mmHg and the mean diastolic BP (± SD) was 73.2 ± 9.1 mmHg. Mean systolic BP (± SD) in a group of 17 year-old randomly selected white high school males and hostel students (n = 15) in the Western Transvaal (Van Zyl, 1983) was 118.5 ± 11.7 mmHg and that of females (n = 23) was 123.8 ± 10.3 mmHg and the mean diastolic BP (± SD) of males was 64.2 ± 14.4 mmHg and that of females was 70.9 ± 11.4 mmHg.

In a study carried out by Walker et al. (1980b), the mean systolic BP (± SD) of black males (n = 148) in the 17-year age-group from a rural area was 118.7 ± 10.6 mmHg, of rural females (n = 140), 117.8 ± 10.9 mmHg of males (n = 151) from the urban area, 119.0 ± 9.9 mmHg, of urban females (n = 152), 120.2 ± 10.9 mmHg. The corresponding mean diastolic BP (± SD) was: rural males, 68.8 ± 8.2 mmHg, rural females, 69.9 ± 8.9 mmHg, urban males, 72.5 ± 9.4 mmHg, urban females, 73.4 ± 6.9 mmHg.

Several studies have identified differences in BP in teenagers by ethnicity (Goldbourt and Kark, 1982). In a national sample of Israeli males aged 17 years, those whose parents were of Yemenite and Indian origin had lower BP levels than did those whose parents of European and American origin (Kark, 1977). In the Jerusalem Lipid Research Council (LRC) Study, 17 year-old Jewish residents of North African origin also showed lower BP levels (Lewis et al., 1982). However, in these studies the actual BP levels of the subjects have not been given.

It seems that the mean BP levels of the Indian adolescents in the present study were lower than published values of other groups of South African adolescents.

It seems therefore that the factors which contribute to the high prevalence of hypertension in Indian adults (Seedat et al., 1978; Seedat et al., 1990) are not yet operative in these adolescents and that an "early" elevation of BP is thus not responsible for the excessive CHD risk of this community.

5.4 Dietary Patterns and Nutrient Intakes

The main aim of this study was to examine the possibility that dietary patterns and nutrient intakes contribute to an increased risk of CHD in this population. Special attention was therefore given to obtaining dietary data. A validated food frequency questionnaire was used during

the individual interview of the subjects. The coding of the data for nutrient analysis, in which preparation and cooking methods, as well as ingredients of typical Indian food dishes were carefully accounted for, ensured that nutrient intake data was as reliable as possible. The significant correlations found between nutrient intakes and blood variables, such as the positive correlation of dietary cholesterol and saturated fat intake with TC, and the negative correlation of dietary plant protein with TC (Table 4.13) tend to support the assumption that the dietary intake data is reliable.

The most important observations regarding nutrient intakes were that the subjects had a nutritionally adequate diet re: intakes of energy [(non-vegetarian males (n = 126), 13.2 $\pm$ 3.1 MJ, non-vegetarian females (n = 128), 10.2 $\pm$ 2.8 MJ, vegetarian males (n = 11), 13.0 $\pm$ 2.3 MJ, vegetarian females (n = 20), 10.0 $\pm$ 1.9 MJ)], essential amino acids, vitamins and minerals with the exception of calcium intakes which were less than the RDA. However, the percentage of energy contribution by fat of approximately 42-48% (Table 4.5), was more than the 30% recommended in most guidelines for "protection" against CHD (Truswell, 1987).

The large standard deviation value of the energy intake mean was due to the large range between high and low values. Also, the greater prevalence of high energy and thus nutrient intakes among the highest socio-economic group of subjects was due to the high intake of all food categories, especially the dairy, meat and cereal (bread/roti) categories. This finding was similar to that found in Tasmanian

adolescents (Woodward, 1985). However, compared with the 15-19 year-old CORIS group (Wolmarans et al., 1988) where dietary data was collected with the use of the 24-hour recall method, mean total fat intake, and therefore fatty acid intake, as well as the P/S ratio of the subjects was higher. Mean total energy intake (± SD) of CORIS males (n = 96) was 12.7 ± 4.9 MJ, percentage energy from carbohydrate was 51.5 ± 7.6, from total protein, 13.8 ± 3.0, from animal protein, 9.2 ± 3.6, from total fat, 34.6 ± 5.8, P/S ratio, 0.6 ± 0.3, dietary fibre intake, 22.8 ± 12.4 g and mean total energy of CORIS females (n = 91) was 7.2 ± 2.5 MJ, from carbohydrates, 49.9 ± 8.0, percentage energy from total protein, 14.4 ± 3.6, from animal protein, 10.0 ± 4.0, from total fat, 35.1 ± 7.1, P/S ratio, 0.6 ± 0.3, dietary fibre intake, 14.3 ± 7.4 g.

It is important to note that the mean total energy intake as well as the mean percentage energy intake of the adolescents in the present study was similar to that recorded for adults (Mia, 1983), in the 20-25 year age-group. For males (n = 43) mean total energy intake was 13.2 MJ, percentage energy from carbohydrate, 45%, from fat, 40%, from protein, 15%, and for females (n = 37) total energy intake was 11.3 MJ, percentage energy from carbohydrate, 48%, from fat, 36%, from protein, 16%.

Very little data is available on dietary intakes of Indian adolescents as well as of other age groups. In a British Indian study (McKeigue et al., 1985) of 184 households the mean percentage energy intakes from carbohydrate was 48.6 ± 0.6, from fat, 38.8 ± 0.6, and from protein,

12.3 ± 0.2. Thus, the mean percentage energy intake from fat reported for British Indian households was slightly less than that of the adolescents in the present study, as well as that of the South African Indian adults.

The dietary pattern of the subjects in the present study is similar to that of the "high risk" diet in India (urban areas), as well as that of immigrant Indians among whom overnutrition is common (Raheja, 1988). Compared with the rural, traditional "low risk" diet in India, the "high risk" group consume more animal foods (lamb, chicken and beef) and dairy products and less milk and yoghurt, 35-40% fats (percentage of total energy) and refined cereals in favour of whole grain cereals, less pulses and legumes, vegetables (including onions and garlic), less fresh fruit and more processed fruit juice, more sucrose and less condiments and spices (Raheja, 1988).

It was surprising that mean total fat intakes (as percentage of total energy) of the vegetarian subjects in the present study were even higher than those of the non-vegetarian subjects. The dietary cholesterol intakes of vegetarian subjects, however, were substantially and significantly lower than those of the non-vegetarians (Table 4.4.). This indicates that the main sources of fat in the vegetarians' diet were of plant origin (as they ate no meat) such as oils and margarines. The saturated fatty acid intakes of the vegetarians were mainly contributed by milk and hydrogenated plant fats.

The study indicated that the high fat intake could partly be ascribed to the traditional Indian cooking methods where many foods are fried in fats and oils.

The question could be asked whether oxidized cholesterol found in "ghee" contributes to CHD in this Indian population. In India "ghee" has been the most desired traditional cooking fat but because of its high cost at present it is virtually replaced by vegetable oils (Raheja, 1988). This is the trend in the lower-income households in the present study (personal observation), but in the higher-income category, "ghee" is still used. Its use is recommended in most recipes in Indian cookery books, particularly in the popular local "Indian Delights" (Mayet, 1987). However, advice limiting the use of "ghee" is quite obscure and may not be read by the majority of housewives who use this book for cooking quite regularly.

It has been hypothesized that Indian "ghee" contains cholesterol oxides and that this dietary exposure to cholesterol oxides could provide an explanation for the high frequency of atherosclerosis in these Indian populations (Jacobson, 1987). "Ghee" is clarified butter and butter is the fat most frequently used to produce atherosclerosis in other species, including non-human primates (Turner, 1983). Butter is also the most hypercholesterolaemic of all fats tested. It increases the susceptibility to thrombosis, inhibits fibrinolysis, and alters the composition of important cells and tissues such as cholesterol esters and the phospholipids in platelet membranes in a manner likely to stimulate atherogenesis.

Raheja (1988) mentions that the present day Indian diet has a liberal quota of fats, fried, processed foods with a fair portion of these derived from animal sources and refined bakery products. Consequently this diet provides an excess of calories, fats (both saturated and poly-unsaturated) from refined or hydrogenated vegetable oils, dietary cholesterol, sucrose and sodium. There is a deficiency of plant fibre, anti-oxidants, many vitamins and essential minerals. The increased consumption of vegetable oils increases the requirements of essential fatty acids of the n-3 series and anti-oxidants, and, unless this diet is supplemented with a fair intake of fish, the "high risk" diet is deficient in this essential fatty acid. It has been stated that this may not be the case when the "low risk" diet is consumed in which there is a lower intake of fats (Raheja, 1988).

The high energy and fat intake of the adolescents in the present study poses a serious problem since it is possible that at this age they have already formed a fixed eating habit. Habitual over-nutrition could lead to obesity (Raheja, 1988) which is a risk factor for NIDDM, high clotting factor levels, hypertension, atherosclerosis and CHD.

From the above it is clear that in order to reduce fat intake of this group of subjects it is necessary to substitute skim milk for whole milk, low-fat cheese for high-fat cheese, low-fat yoghurt for high-fat yoghurt, and vegetable shortening for "ghee". The mean daily dietary intakes of calcium of these subjects is less than the RDA and these recommended measures would ensure an increased intake of calcium. Cooking methods

also require modification, e.g. poaching, boiling or grilling some food dishes instead of frying in fats and oils.

5.5 Serum Lipids

Total cholesterol

In the present study, relatively high mean TC values (Table 4.11) were found in non-vegetarian males and females and in vegetarian females, i.e. 4.32 ± 0.71 mmol/l, 4.39 ± 0.60 mmol/l and 4.16 ± 0.71 mmol/l, respectively. Mean TC value (3.74 ± 0.34 mmol/l) of vegetarian males was significantly lower than those of non-vegetarian males. This mean value was in the low risk category, i.e. < 4.1 mmol/l, according to the age-specific cut-off points for South Africans (Rossouw et al., 1988), while the mean values of the first 3 groups fell in the moderate-risk range, i.e. 4.1 mmol/l to 5.4-5.5 mmol/l.

The results obtained in the present study have been compared with those of adolescents in the CORIS white population (Rossouw et al., 1983) and in the coloured population of the Cape Peninsula (Steyn et al., 1985). The results in the latter two studies were grouped into stratified age-groups. The values reported here are those of the 15-24 year age-group. Both these studies reported a high prevalence of CHD risk factors in their study populations. The results in the present study are also compared with those reported for 17 year-old Jerusalem offspring of Jews

from 19 countries of birth (Halfon et al., 1982a) who attended an army medical examination a decade ago. In Israel immigrants from European and American countries have the highest rate of CHD, whereas those from North African countries have the lowest (Medalie et al., 1973); and in consistence with this, 17 year-old males and females of North African origin showed the lowest means for TC, TG and HDL-C, and the children of parents born in Europe and the Americas showed the highest levels.

The subjects were grouped into 4 areas. Mean TC values of males was (Israel; n = 947) 3.55 ± 0.67 mmol/l; (Asia; n = 1 005) 3.44 ± 0.66 mmol/l; (North Africa; n = 789) 3.29 ± 0.57 mmol/l; (Europe; n = 902) 3.56 ± 0.60 mmol/l, for females; (Israel; n = 874) 3.94 ± 0.73 mmol/l; (Asia; n = 820) 3.88 ± 0.74 mmol/l; (North Africa; n = 589) 3.75 ± 0.72 mmol/l; (Europe; n = 715) 4.01 ± 0.72 mmol/l. All mean values were below 4.1 mmol/l placing these subjects in the low-risk category for CHD.

In South Africa, mean serum TC values in the 15-24 year age-group of the CORIS population (Rossouw et al., 1983) was: males (n = 635), 4.56 ± 0.99 mmol/l, females (n = 658), 4.96 ± 0.97 mmol/l, and in the coloured population of the Cape Peninsula in the same age-group for males (n = 94) was 4.66 ± 0.73 mmol/l and for females (n = 103), 4.81 ± 0.73 mmol/l (Steyn et al., 1985). All four mean values were higher than those recorded in the present study and fell in the moderate-risk range for CHD.

It has been stated that at higher concentrations of cholesterol, CHD mortality was compounded (Grundy, 1986). If a risk ratio of 1.0 is assigned for a cholesterol level of 200 mg/dl (5.17 mmol/l), then at 150 mg/dl (3.88 mmol/l) the ratio is definitely lower, about 0.7, and at 250 mg/dl (6.47 mmol/l), the risk is doubled, and at 300 mg/dl (7.76 mmol/l) it is doubled again. In the present study, TC values of 16 subjects were in the double-risk range, i.e. > 6.47 mmol/l (Table 4.12).

The high fat intakes observed in the present study are probably partially responsible for the relatively high serum TC values, particularly in vegetarians. This finding is consistent with the hypothesis that it is not the cholesterol intake which is mainly responsible for raised serum TC level, but rather a high fat intake (Grundy, 1992), although a positive significant correlation of TC with dietary cholesterol was demonstrated in the present study.

Although a dose and effect relationship of the beneficial dietary effects of garlic, ginger, Bengal gram, lentils (legumes) and pulses, which are known to be hypocholesterolaemic because of their soluble fibre content and which are basic ingredients of Indian food dishes, has not been evaluated, it seems that any benefit derived from the consumption of these items is minimised by the high fat intake of the adolescents in the present study. This is demonstrated by the high (moderate- and high-risk) (51.7% and 5% respectively) serum TC values found in 56.7% of these (whole group) adolescents (Table 4.12).

High density lipoprotein cholesterol

Although mean HDL-C (mmol/l) (Table 4.11) values of all these subjects were greater than 1.0 mmol/l, i.e. 1.15 ± 0.24 (non-vegetarian males; n = 148), 1.28 ± 0.27 (vegetarian males; n = 11), 1.18 ± 0.16 (non-vegetarian females; n = 142), 1.19 ± 0.23 (vegetarian females; n = 20) (below which, values should be regarded as a positive risk factor for CHD) (Berger, 1984), this value is below 1.8 mmol/l which is regarded as a protective value. However, HDL-C concentration must be interpreted in regard to the lipid profile as a whole, with particular emphasis on the total (more especially LDL) cholesterol level. It has been stated that a low plasma TC concentration reduces the risk associated with a low HDL value while a high TC value potentiates the risk (Berger, 1984).

Mean fasting plasma HDL (mmol/l) of 17 year-old Israeli male inductees in the Jerusalem study (Halfon et al., 1982a) was: Israel (n = 935), 1.11 ± 0.27; Asia (n = 996), 1.08 ± 0.24; North Africa (n = 785), 1.06 ± 0.23; Europe (n = 890), 1.15 ± 0.26; and for females: Israel (n = 861), 1.30 ± 0.29; Asia (n = 804), 1.24 ± 0.30; North Africa (n = 584), 1.23 ± 0.28; Europe (n = 703), 1.31 ± 0.29. The females had higher mean HDL-C than did males. However, these values were higher than those recorded for all four groups in the present study.

Mean HDL-C (mmol/l) of the 15-24 year age-group CORIS males (n = 635) was 1.14 ± 0.24 and that of females (n = 658), 1.35 ± 0.27, while that of the coloured population of the Cape Peninsula in the same age-group of males

(n = 94) was 1.42 ± 0.32 and that of females (n = 103) was 1.60 ± 0.38. The females had a higher mean value than that of males, although both values were also higher than those recorded in the present study.

Triglyceride levels

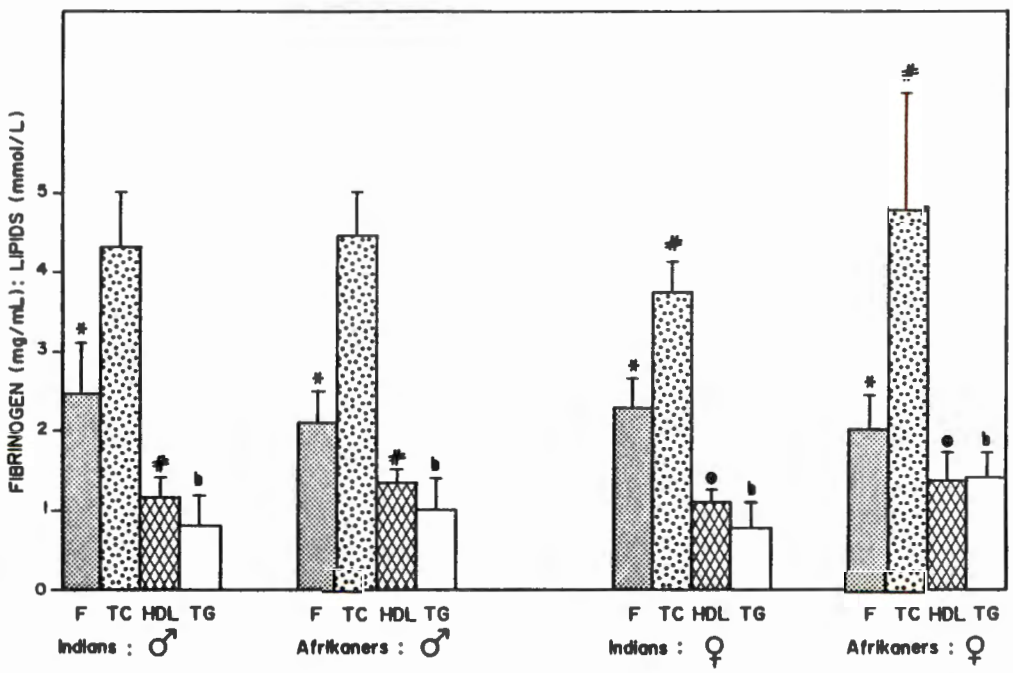
Mean TG (mmol/l) values of all four groups of subjects in the present study were well below [(non-vegetarian males (n = 148), 0.79 ± 0.35; vegetarian males (n = 11), 0.82 ± 0.30, non-vegetarian females (n = 142), 0.78 ± 0.33, vegetarian females (n = 20), 0.92 ± 0.40)] the recommended value of 2.28 mmol/l (200 mg/dl) above which values should be regarded as abnormal (Rossouw et al., 1988). Mean TG (mmol/l) values (Jerusalem study) of the males were: Israel (n = 936), 0.88 ± 0.40; Asia (n = 996), 0.89 ± 0.41; North Africa (n = 776), 0.82 ± 0.34; Europe (n = 890), 0.86 ± 0.38, and that of females was: Israel (n = 865), 0.87 ± 0.45; Asia (n = 817), 0.87 ± 0.36; North Africa (n = 583), 0.83 ± 0.35; Europe (n = 704), 0.85 ± 0.36. These values were all in the normal range and were similar to those observed in the present study. The CORIS study did not report TG levels, while the coloured population study reported serum TG (mmol/l) levels of non-fasting subjects, i.e. males (n = 94), 1.27 ± 0.82; females (n = 103), 1.13 ± 0.54. Although these values were within the normal range, they were higher than those of the present study as well as those reported in the Jerusalem study. Also, the study was carried out on non-fasting subjects and the higher TG levels may be due to exogenous TGs carried on chylomicrons (Halfon et al., 1982a).

Blood plasma TGs are carried in two major groups of lipoproteins, chylomicrons and VLDL (Brown and Karmally, 1985). Chylomicrons are secreted from the intestinal epithelial cells into the lymph during dietary fat absorption. They are cleared rapidly after entering the blood stream of normal people, virtually disappearing from the plasma within six hours of consuming a fat-containing meal. VLDLs are produced continuously by the liver as a means of ridding that organ of excess calories in the form of TGs.

Changing the dietary carbohydrate from 40% to 80% of consumed calories is known to markedly increase the VLDL-TG secretion in the plasma (Olefsky et al., 1974; Ginsberg et al., 1981). When diets containing 60-65% of complex carbohydrates from natural sources (Gotto et al., 1984) and 15 to 25% fat are used, the induced rise in plasma is markedly blunted (Antonis and Bersohn, 1961; Ahrens, 1974). When the source of carbohydrate is primarily from cereals (wheat, rice, corn), fruits and vegetables, the change in plasma TGs is less evident (Anderson, 1980). During periods of high carbohydrate consumption the TGs are highest early in the morning, at the time of taking the fasting blood sample (Kuo and Carson, 1959; Fredrickson, 1969; Schlierf et al., 1971). On diets containing higher fat content, the TG level rises throughout the day with a low point occurring prior to breakfast. Thus the average TG level over the entire day may not be higher while consuming a diet high in carbohydrates. Also, TG levels in free-living populations who habitually consume approximately two-thirds of their calories as carbohydrates (Japanese living in Japan) are consistently lower than those found in Western man (Kagan et al., 1974).

5.6 Plasma Fibrinogen

Almost half (48.9%) of the subjects in this study had plasma fibrinogen levels above 250 mg/dl (arbitrary cut-off point). The mean fibrinogen levels of both non-vegetarian and vegetarian males and females were higher than the mean levels found in 18-20 year-old white males (n = 71), 210 ± 42 mg/dl, and females (n = 69), 209 ± 55 mg/dl students (Figure 5.1) who followed a "Western" diet and who were also at high risk for CHD (Venter et al., 1991 and 1992).



F = fibrinogen
TC = total cholesterol
TG = triglycerides
HDL = high density lipoprotein cholesterol
♂ = males
♀ = females

Figure 5.1: Comparison of mean fibrinogen and lipid levels of Indian and Afrikaner males and females

The high fibrinogen levels found among males in the present study could possibly be explained by the high percentage of smokers (21.6%) (Meade et al., 1986a) and possibly by the high fat and low fibre intakes (Vorster, 1987b). However, as there were no female smokers, their high fibrinogen levels could possibly be attributed to the high fat intake (SATFAT, MUFAT and PUFAT) and low fibre intake (see Table 4.3), when compared to the recommended fibre intake of 5.4 to 6.0 g/1 000 KJ.

The risk of CHD in those with high fibrinogen levels were greater in younger than in older men in the NPHS (Meade et al., 1986b).

The high fibrinogen values found in the adolescents in this study might contribute towards a possible explanation for the as yet unexplained high prevalence of CHD and the early age of onset among immigrant Indians wherever they have settled. Therefore, it seems important that fibrinogen levels in adult Indians should be measured as part of the CHD risk profile, and that subjects with elevated levels should be treated.

A possible reason for the high fibrinogen levels observed in these subjects (high fat, low fibre intakes) has been mentioned already. However, other possibilities must also be examined. A raised level of circulating FFA (free fatty acids) is a known stimulant for fibrinogen synthesis (Pickart and Thaler, 1980). Thus any situation that raises the level of FFA will conceivably also raise the fibrinogen level, e.g. insulin resistant states, obesity (increased energy intakes) (Ferrannini et al., 1987) and smoking (Simpson, 1984).

In the present study TG showed a weak but significantly positive correlation with plasma fibrinogen (Table 4.13). Therefore, is it possible that the Indian population has a genetic predisposition to insulin resistance? This could also explain the high incidence of NIDDM and NIDDM(y) amongst the Indian population (Seedat et al., 1990), as well as the high fibrinogen levels observed in the present study.

The high level of the other risk factors of CHD found in these adolescent subjects could also partly provide an explanation for the 22% patients, under the age of 45 years, being admitted to the coronary care unit of a major South African Indian hospital (Mayet, 1982) and for the "epidemic" proportions of CHD (Seedat et al., 1990) among this population.

5.7 Plasma Factor VII

Factor VII levels were normal in this study. There is a strong possibility that dietary fat is an important determinant of factor VII activity (Miller et al., 1985) which may contribute to "hypercoagulability". It has been stated that a high fat intake may lead not only to coronary atheroma but also to fibrin deposition and thrombus formation through direct activation of the coagulation system (Miller et al., 1986a). There are indications (Miller et al., 1986a) that it is mainly postprandial lipaemia that activates factor VII. It should be kept in mind that in the present study, factor VII was determined in plasma samples of fasting subjects which may explain the normal values obtained despite the

high fat intake. Most of the current guidelines for prevention of CHD, recommended that not more than 30% of dietary energy should be provided by fat (Truswell, 1987).

5.8 Smoking Habit

A very high percentage of the subjects (Table 4.2) (whole group - 21.6%) were smokers while 2.9% had first smoked at the early age of 10 years. Most of these smokers were males (59/159 or 37.1%) and only a small number were females (9/162 or 5.6%). Mean age (± SD) (years) when males first smoked was 15.6 ± 1.7 and that of females was 15.0 ± 1.4. Percentage smokers in 15-24 year age-group in the CORIS study was: males (n = 635), 34.3% and females (n = 658), 11.1%, while that of the coloured population in the same age-group was: males (n = 94), 39.4%; females (n = 103), 34.0%.

It is noteworthy that the percentage was the highest among coloured males, followed by the Indian males in the present study. The CORIS males had the lowest percentage of smokers. Among females, the coloured group had the highest percentage smokers followed by the CORIS group, while the Indians had the lowest percentage smokers.

The percentage smokers in the Jerusalem LRC study (Halfon et al., 1982b) (in which a 20% random sample of all participants in the initial examination (Visit 1) were invited to attend a more extensive examination

(Visit 2) with 75.3% responding) was: males (grouped according to the father's place of birth) from Israel (n = 177), 31.1%, from Asia (n = 208), 30.8%, from North Africa (n = 128), 47.7%, and Europe (n = 156), 18.6%, and that of females was: Israel (n = 160), 19.4%, from Asia (n = 157), 14.0%, from North Africa (n = 131), 29.8%, and Europe (n = 123), 13.0%. Of these subjects 669 (31%) males and 571 (19%) females were current smokers. Prevalence of smoking was highest in male and female adolescents of North African origin and lowest in those of European origin.

The highest prevalence of Indian male smokers (present study) could be due to an acceptability of male smokers among adults as well as socially, while the low percentage smokers among females could be due to social intolerance of female smokers and lack of peer group pressure.

5.9 Socio-economic Status

Socio-economically (Table 4.2), one-third of the subjects were from the upper-income group, one-third from the intermediate group and one-third from the lower-income group (based on parent's occupations and as answered by the subjects).

5.10 Summary

The salient observations of the present study are:

* The anthropometric data indicated that South African Indian adolescents may be shorter than white South African adolescents, although, only limited comparative literature was available.

* Dietary intakes were adequate. It is therefore not clear whether the stature of these youths could be ascribed to previous malnutrition or to genetics.

* A very high percentage (21.6%) were smokers. This could have contributed to the higher fibrinogen levels observed among the males.

* The very high fat intake (> 40% of total energy) was similar to that of the "Western" diet. This was due to the high intake of high fat foods as well as traditional Indian cooking methods.

* TC values were high. This could have been the result of a high consumption of fatty foodstuffs of animal origin and a decreased consumption of unrefined plant foods.

* The factors that increased TC levels possibly also increased fibrinogen levels. Plasma fibrinogen levels were higher than reported values of white adolescents.

* Blood pressures were normal and lower than values reported for white and coloured South Africans of the same age-group. It therefore seems that the factors that contribute to the high prevalence of

hypertension in adult South African Indians are not yet operative in the Indian adolescents.

* The high incidence of hyperlipoproteinaemia could be due to dietary factors. The possibility that this could be due to genetic, environmental or socio-economic factors requires much more research. The high incidence of hyperfibrinogenaemia could be related to an early insulin resistant state. At this stage, however, this is only speculative and therefore more research in this field is needed.

* Positive correlations of TC with LDL-C and of TC with TG were observed. The positive significant correlations of TC and of LDL-C with arachidonic acid, MUFAT, dietary cholesterol and animal protein, illustrate the detrimental effect of nutrients found in foods from animal products on TC and LDL-C. The negative correlation of TG with HDL-C and on percentage HDL-C of TC illustrates that raised TG levels are often accompanied by decreased HDL levels. TG correlated positively with weight and mid-upper-arm circumference. There was a significant positive correlation between weight, BP and mid-upper-arm circumference confirming existing knowledge of the effect of overweight on BP. TG showed a weak but significant positive correlation with plasma fibrinogen. A negative correlation between fibrinogen and fibre and plant protein was observed. The significant positive correlation between fibrinogen and mid-upper-arm circumference, weight and TG illustrates the possible effect of overweight on these variables. It therefore seems that the factors that increase TG may also increase fibrinogen levels.

CHAPTER 6

CONCLUSIONS AND RECOMMENDATIONS

From the results of the present study in can be proposed that primary prevention efforts for CHD should be initiated in childhood, since risk factor levels tend to track over time and high-risk children can be identified early. Also, risk-related dietary, smoking and physical activity behaviours develop early in life and it is difficult to alter these well-ingrained health behaviours in adults.

Recommendations

For this population group dietary changes are necessary in order to comply with the prudent diet recommendations. Risk factor level screening for at least TC, fibrinogen, DM, BP, BMI and W/H ratio, is necessary for the reduction of potential risk. The smoking habit should be eradicated. Physical activity and exercise programmes should be initiated on a large scale for children, adolescents and adults.

Dietary recommendations

The high fat intake of the adolescents in the present study demonstrates the need to inculcate good nutrition habits in childhood. Although the basic Indian diet comprises many food items to provide nutritionally

well-balanced meals, the busy Indian housewife has little time or training to use the correct combinations of these items.

To achieve a nutritionally-balanced meal which is also anti-atherogenic, it is necessary to decrease total fat to less than 30% of energy and saturated fat intake from animal, dairy and bakery products to about 10% of total energy (Truswell, 1987). Approximately 1.0 to 2.0% of dietary energy consumed as essential fatty acids prevents clinical signs of deficiency (Raheja, 1988). Thus, diets containing 5 g of essential fatty acids (linoleic acid and alpha-linolenic acid) will meet these requirements.

It must be taken into account especially that in the Indian diet many foods are fried and that when cooking oils and margarines are heated to above 200°C the cis EFAs (OA and LA) are transformed to their trans isomers. Thus, fried foods, curries and bakery products contain substantial amounts of trans fatty acids (Sinclair, 1982) which are major factors in delta 6-desaturase inhibition and hence their metabolism to their eicosanoid and longer-chain derivatives. Trans fatty acids tend to resemble saturated fatty acids in biological systems (Beare-Rogers et. al., 1979). Thus, when margarine is recommended as a substitute for butter the soft variety should be selected and the quantity should be decreased. Also, "ghee" (clarified butter) which is still regularly used in many homes for cooking and frying food and which contains oxy-cholesterols, could be replaced by vegetable "ghee" or oils.

Increased intake of marine (cold water) fish, especially fatty fish (herrings, sardines, mackeral, pilchards, salmon) should also be recommended as their high contributions of PUFATs, particularly EPA, could inhibit the development of atherosclerosis. Also, marine n-3 fatty acid supplementation has been shown to have beneficial anti-atherogenic actions related to atherosclerosis, thrombosis and lipid metabolism (Leaf and Weber, 1988). Fish oil supplementation is associated with marked reductions in fibrinogen levels (Hostmark et. al., 1988; Radack et. al., 1989). Kromhout, et. al. (1985) found an average of 30 g fish eaten daily to be associated with a far lower mortality from CHD. Thus, in order to achieve a meaningful measure of protection consuming this quantity of fish daily for non-vegetarians in the present study would not be difficult to implement.

Total fat intake could be lowered by removing all visible fat from meat, and by substituting skim milk for whole milk and low fat cheese for whole milk cheese.

The fibre intake of the subjects in the present study was low. Thus fibre intake could be increased by eating cereals, pulses, legumes (dahls), vegetables and fruit. Many essential nutrients and anti-oxidants are found in complex carbohydrate foods such as whole grain cereals, pulses, vegetables and fruits. Also, fibre from fruits, vegetables, legumes and pulses have been shown to lower blood glucose (Jenkins, 1982; Raheja, 1988) and is also associated with reduced levels of plasma cholesterol (Kay, 1982; Vahouny, 1982, 1986). There is thus a

necessity for larger studies on the dietary effects of clotting factor values and of the extent to which any effects of diet on the incidence of CHD are mediated through haemostatic functions (particularly factor VII and fibrinogen).

Also an increase in the consumption of suitable combinations of cereals, pulses and legumes could provide an intake of complete proteins (as all are relatively deficient in one or more of the essential amino acids) (Raheja, 1988), similar to animal proteins which are complete. This could also result in a decreased intake of fat.

Spices and condiments are essential ingredients in Indian cooking (see Appendix B.2). They are used in small quantities as they add taste and flavour and provide many essential nutrients (Gopalan et. al., 1977). Spices and condiments commonly used (cloves, black pepper, green/red chillies, lemon, tamarind, turmeric, coriander, cumin seeds, etc.) are potent anti-oxidants or have aldose reductase inhibiting activity (Gopalan et. al., 1977; Chaudhry, et. al., 1983; Bland, 1984; Passwater, 1984). Thus, the use of traditional recipes will ensure that these dietary benefits can be of value.

In this study vegetarianism did not demonstrate any advantages over non-vegetarianism in the coronary context probably because all food is essentially prepared by frying in cooking fats, "ghee" and oils. Thus it is necesary to advise this population to prepare food by boiling, steaming, grilling and baking food articles without these additives.

In South Africa, the Heart Foundation, which has recognized the food industry as the most powerful nutrition educator of the public (de Ruiter, 1987), published "The New Generation Cookbook" in 1987 (Rossouw, 1987). For South African Indians who enjoy food cooked in the traditional way the publication of a cookbook adapting original recipes with the above recommendations, is essential.

Smoking

In the present study, a large percentage of adolescents had already acquired the smoking habit. The mechanisms through which smoking may contribute to CHD, have been delineated in Chapter 2.5.5. It has been stated that stress may operate indirectly by causing people to smoke and overeat, and hence increase their risk of developing CHD (Freeman, 1983). Anti-smoking lectures should be initiated at schools emphasizing the need to stop smoking immediately and for always. Non-smokers should be advised not to smoke at all.

Physical activity and exercise

It is necessary to advise these adolescents to devise regular physical activity and exercise programmes which could have beneficial effects on blood lipid and lipoprotein profiles, reduce stress levels, BP, and obesity.

Conclusion

From the above it is clear that to reduce the risk of CHD amongst this South African Indian population, attention should be given to the feasibility of screening for risk factors of CHD, dissemination of information on the "prudent diet", smoking and physical activity. Dietarily, the initiation of an increased intake of cereals, pulses and legumes (which are basic ingredients of Indian foods) as well as modifications in cooking methods could result in a decrease in fat intake and an increase in fibre intake.

APPENDIX A

ARRIVAL OF INDIANS IN SOUTH AFRICA

A.1 INTRODUCTION

The story of the Indian South African in fact began on Christmas Day, 1496, when Vasco da Gama, en route to India, first sighted a coastal land-mass of particular beauty and named it Natal (Phillips, 1975). A year later Melinde, a famous navigator picked up an Indian pilot and under his guidance "cast anchor two leagues from the city of Calicut". Having successfully opened the sea trading route between India and what were to become her European markets, he unintentionally sealed the fate of an Eastern civilization too old to remember its birth.

The first Indians arrived in South Africa either singly or in families as indentured labourers in Natal aboard two ships, the Truro from Madras (Meer, 1969) and the Balvedere from Calcutta (Bagwandeen, 1989). Labour demands in Natal during the 19th century for sugar, tea and wattle plantations, the railways and the mines, led to the introduction of Indians in South Africa between 1860 and 1911 (Albertyn et al., 1973; Meer, 1980).

In 1843 Natal became a British Colony and black labour proved to be unreliable. The majority of Indians in South Africa are descendants of the indentured workers brought to Natal between 1860 and 1911 (Albertyn et al., 1973), to develop the country's sugar belt. Their work included

the whole range of harvesting, transporting, milling and building dams, factories and houses (Meer, 1980).

Indian South Africans are thus just over a hundred and thirty years in this country, but they trace their roots in India to one of the world's oldest known civilizations, that of the Indus Valley, whose ruins indicate the existence of a technologically complex and literate culture as far back as 3 500 B.C. (Albertyn et al., 1973).

A.2 HISTORICAL BACKGROUND OF INDIANS IN INDIA

As reviewed by Meer (1980) four influences predominated in the thousands of years of intellectual and physical fusions that have resulted in the Indian people: the Dravidian, Aryan, Islamic and British.

India, which is the subcontinent of Asia, was invaded by the Aryans from somewhere in Southern Russia in about 2 000 B.C.. They dominated the country for almost 3 000 years. Though they were outwardly contemptuous of the indigeneous people and were responsible for the caste system, they recognized the superior Dravidian culture which they integrated with their own and laid down the foundations of Hindu civilization. The oral tradition of their scriptures, the Rig Veda was complemented on the Indian Peninsula by three other inspired works: the Yajur-Veda, the Sama-Veda and the Atharva-Veda. This in turn stimulated the epics of the Ramayana and Mahabharata and gave rise to the six systems of Hindu philosophy, to Jainism and Buddhism.

Up to the 10th century the Hindu kings retained supremacy in North and South India. Then came the Muslims. Compelled by their newly-founded Islamic zeal, and just as they had overpowered Southern Europe and set up the Moorish Empire, they now overpowered the Indians who had grown weak through internal dissension at that point in their history.

The first Muslim invaders were the Turks who ruled from temporary military camps and showed little interest in the indigeneous people and their customs. They were followed by the Moghuls and Afghans. The Moghuls inter-married and became recognized as Indians. Their rulers were the sons of Rajput princesses and their vast civil service was manned mainly by Rajput officials. Indian life became substantially influenced by Muslim monotheism and military discipline. The Moghuls were of Turkish extraction, spoke a Turkish language and were great admirers of the Persian way of life. Hence, the Islamic influence in India of dress, architecture, painting, dancing, music and literature is essentially Persian.

While the Moghuls were busy consolidating their empire in Northern India and the Deccan, the Europeans were looking for a sea-route to India. For years the European courts had savoured Indian spices and treasured Indian silks and other objects of luxury imported by land through Constantinople. Constantinople fell to the Turks in 1453 and the European trade route to the East was closed. In 1498, Vasco da Gama was conducted to Calcutta by Arab sailors. A new stream of foreign invasion began from the South of India and small portions were occupied by the French, Portuguese, Dutch and English (Phillips, 1975).

In the 18th century, the Moghul Empire began to crumble and the English began expanding their power. In the mid-nineteenth century the English became rulers of virtually the whole of India. The English invasion rode on the crest of the Industrial Revolution. Britain had harnessed mechanical energy and the emergent capitalists, in pursuit of markets and profits, swept aside men and their skills to make way for the industry of machines. Within half a century India lost her ancient, two-thousand year-old position of dominance in world trade based on her skilled crafts and processed goods. She now became reduced to a plantation of cheaply exploitable raw materials and a dumping ground for machine-made British goods. From being a manufacturer of cotton goods, she became an importer. Local manufacture of textiles was deliberately suppressed and skills that had passed through families for generations were rendered useless (Meer, 1980).

Whereas on the eve of British occupation, the import trade of India consisted of gold bullion, it now exported as much and more in revenue to Britain. The British evaded inland dues on trade and extracted exhorbitant taxes from land owners and local rulers, leaving the country destitute and its people helpless. In rural areas the peasants' land was auctioned to those who promised the highest tax return and eventually transferred in perpetuity to the tax collecting official (Zamindar) (Meer, 1980).

By 1832 there was no silver left in Bengal to meet British demands. The land-holding peasant became a rootless labourer who could be manipulated

in the factory or on the plantation, or induced to indenture himself on colonial plantations throughout the world. In this state of high vulnerability the Indians began contracting their labour in 1834 to the plantations of the world. Indentured labour was exported to Jamaica, British Guiana and Trinidad from 1844, to Natal from 1860 (Albertyn et al., 1973), to St. Lucia from 1866, Granada from 1858, the French colony of Reunion, Martinique, Guadeloupe, the French Guiana from 1860, the Danish colony of St. Croise and the Dutch colony of Surinam from 1870. Ill-treatment of indentured immigrants was a common complaint (Meer, 1980).

A.3 LABOUR FOR NATAL

From Southern India, 75% of the South African Indian immigrants came from from Madras, 18% from Andhra, 5% from Banglore and Mysore and 2% from Travencore (Kerala). Of the North Indians, 61% came from Uttar Pradesh, 31% from Bihar, 6% from Bengal, 2% from Central India - Nepal, Punjab and Orissa. Between 1860 and 1875, 85% of the immigrants to Natal from Madras were Hindu, and from the Calcutta Indians, 15% were Muslim, 13% Madrasi and 3% Christians.

In 1872 the Natal Indian population was about 5 700 adults who were deemed to be "free" Indians as they had completed their 10-year indentureship. By 1875, free immigrants, mainly from Gujerat (Bombay area) had also arrived in Natal. They were almost all "passenger" Indians, sufficiently well-to-do to pay their own fares. They were traders and professional men and they set up small trading stores both in the towns and in the country areas (Albertyn et al., 1973).

The aim of the passenger Indians or Muslim traders was to satisfy the local demand for Eastern food by the existing Indian population by importing such foods.

Between 1860 and 1911 a total of 107 529 indentured immigrants arrived in Natal. They moved from the sugar-cane fields into the rest of the country. They enjoyed the same citizenship rights as the whites until this was changed. They constituted an economic threat to the whites, especially in the field of commerce, and thus stringent legal measures were taken by the white legislative existing in South Africa prior to the Union in 1910, and subsequently by the South African government, restricting the residential and social mobility and occupation opportunities of the Indians.

Restrictions were also placed on Indians in the Transvaal in terms of Law 3 of 1885 and its amendment in 1887 (Arkin et al., 1989). The restrictions were further entrenched by the Asiatic Law Amendment Act, Act 2 of 1907, the Act of 1919, the Areas Reservation Bill of 1926, the Transvaal Asiatic Land Tenure Amendment Act, 30 of 1936, the Trading and Occupation of Land (Transvaal and Natal) Restriction Bill of 1943, which was dubbed the "Pegging Act", the Asiatic land Tenure and Indian Representation Act, 28 of 1946, which was commonly called the "Ghetto Act" and was the forerunner to the Group Areas Act Law 41 of 1950 (Arkin et al., 1989). This Act abrogated most of the previous laws and regulations.

Although indenturing came to an end in 1911, the indentured labour as such continued to be a fact in Natal up to at least 1934. The indenture scheme was designed in such a way that those who had completed their five-year period could claim a return passage to India or commute it for a grant out of Crown land and were free to live and work in Natal.

By 1911 the South African Indian population was 152 094 (960 72 males and 56 022 females) (Bureau of Statistics, 1970). Between 1911 and 1946 many laws, such as those mentioned ealier, were passed curtailing immigration of Indians and many schemes encouraging repatriation to India were made. Only 17 000 people left South Africa under these schemes. Up until 1961, the South African Indians were regarded as aliens by the government. It was only in 1962 that they were accepted as South African citizens. The Department of Indian Affairs was created on 03.08.1961 (Albertyn et al., 1973). Only then was attention focused on the social, educational and economic problems of this group of people.

A.4 RACIAL GROUPS IN INDIA

In 1970 the population was about 440 million people (Shreenivas et al., 1970). The different races include: (i) Dravidians in the South, (ii) Aboriginals like Bhils, Santhals and Mundas, scattered in the North, (iii) Aryans or Indo-Europeans in the plains of Northern India, (iv) Mongolians in the Northern hilly areas, and a small proportion of the population that consists of descendants from the Moghuls, Pathans (Afghanistanis) and their admixture with the local population. There are

also Brahmins in the South and North who have not eaten meat or eggs for many generations.

A.5 CULTURAL AND RELIGOUS ASPECTS OF THE SOUTH AFRICAN INDIANS

With a few exceptions, Indian South Africans are South African citizens by birth (Meer, 1969). They are composed of two so-called races, Dravidian and Aryan, having distinct cultural and anthropological characteristics (Seedat, 1982). The Dravidian (Tamil-Telegu) groups have many main language groups in India, but in Natal, for practical purposes, they are represented by Tamils and Telegus. Tamilian people are the largest of the language groups in Natal (Campbell, 1963). These languages are similar, except that Telegu contains more Sanskrit words. However, both languages share a remarkable complexity of grammar and pronunciation.

The Tamil- and Telegu-speaking people immigrated to South Africa from the South Indian states of Madras and Andra Pradesh. Indians from the North of India (Uttar Pradesh, Orissa and Bihar) were of the Hindu faith speaking Hindustani; vigorous Islamic proselytization took place among them in Natal and this has resulted in the South African Urdu-speaking Muslim people. However, there is a small but significant group of Urdu-speaking "passenger" Muslims who came from villages and towns in the State of Gujerat, i.e. from Rander - a district of Surat, Ranavav, Port Bander and Nausar (personal communication). The Urdu-speaking Muslim group can trace their roots in India to the Mongolian and Afghanistan

Muslims. They were originally converted to Islam by Arab Muslim desciples whose military exploits into Northern India and their intermingling with the local population dates as far back as the 7th and 10th centuries A.D.. There are also Pathans, Memans and Koknis who speak Pushtu, Meman and Kokni (or Marathi). The Kokni group originates from the Maharastran State, while the Pathans and Memons originate from the State of Gujerat.

The Hindustani-speaking people are the second largest group in Natal. Indians from the Bombay area comprise the Gujerati-speakers, Muslim and Hindu, and were almost all "passenger" Indians. The Muslims are the numerically commoner of the Gujerati speakers and comprise some of the wealthier citizens of South Africa. The Hindu Gujeratis (so-called Bunyas, or shrewd businessmen) are the smallest significant language group in Natal. They are strict vegetarians and their diet includes large amounts of purified carbohydrates. A diabetic diet is therefore very difficult to institute without making it very dull.

Home Language

The South African Indian community has shown a remarkable change in home languages.

Language	1980*	
	(1)	(2)
English	73,31	11,30
Afrikaans	1,22	-
English and Afrikaans	-	-
Tamil	1,59	0,99
Hindi	1,80	0,89
Telegu	0,23	0,14
Gujerati	2,16	0,86
Urdu	0,94	0,52
Other	4,05	-
	85,30	14,70

* Column (1) indicates the use of only one home language and column (2) indicates the more frequently used language where English and an Asian language are the home languages.

Adapted from: Statistical Year Book, 1964, South African Statistics, 1978, South African Statistics, 1986 (Arkin et al., 1989).

English has emerged as the main home language of most South African Indians. The switch from the use of the vernacular language to English is the result of the use of English as the medium of instruction in schools and the rapid rate of Westernisation.

Religion

The Indian population were the followers of the following religions according to the 1980 census:

Christianity	12,49 percent
Islam	18,79 percent
Hinduism	62,38 percent
Buddhist	0.07 percent
Confucian	0.08 percent
Other	6,19 percent

Hinduism is the religion with the largest following, with Islam and Christianity the other popular religions (Arkin et al., 1989).

A.6 PRESENT STATUS OF INDIANS IN SOUTH AFRICA

In official South African statistical publications, Indians are classified as Asians (98% of Asians are Indians). According to the 1970 census, there were 620 417 Asians in South Africa, of which 514 803 resided in Natal (Albertyn et al., 1973). The 1985 census indicated that the Indian population numbered 866 000 (Brijlal, 1989). According to a Press statement from the Central Statistical Service (CSS), the Indian population had grown to 951 000 by the end of March 1991.

Politically, Indians were given a semblance of representation in the consonance with the ideology of apartheid through advisory bodies in 1961, notably the Local Affairs Committees, and the establishment of the South African Indian Council (Bagwandeen, 1989). The former was concerned with recommendations to municipalities and local authorities and the latter was inaugurated as a statutory body comprising nominated members in 1968. In 1974 its composition changed somewhat when an electoral college comprising Local Affairs Committees elected members of the South African Indian Council. In 1982 this Council was almost entirely elected. However, these two bodies were critisized severely for promoting ethnicity since participation with them entrenched apartheid and enhanced an instrument of their own oppression.

The Nationalist government following on the United Party government remained intransigent in establishing the fundamental principles of democracy and power-sharing until the change of the constitution of the Republic of South Africa in 1983. A tricameral system gave a modicum of power to the Indians and Coloureds. Paradoxically the larger majority of the fabric of the South African population, the blacks, had been excluded. The anomalies of political dispensation and discrimination remained. However, on February 2, 1990, important steps were taken towards ending apartheid laws. The African National Congress and the South African Communist Party were unbanned and all political prisoners were released over a period of time. In 1991 all laws regarding racial classification and housing were removed from statute books.

The Indian population of South Africa is concentrated in the major industrial centres of the country, especially the Pretoria-Witwatersrand-Vereeniging (PWV) region and the Durban-Inanda-Pinetown (DIP) region. The DIP region accounts for 75% of the Indians in Natal and 62% of the Indians in South Africa. The majority of the population live in the sprawling townships of Chatsworth and Phoenix, which were created under the Group Areas Act (Law 41, 1950).

A.7 COMPOSITION OF THE SOUTH AFRICAN INDIAN POPULATION

The composition of the Indian population has been analysed according to sex, age, home language, religion and marital status (Brijlal, 1989). Over 75% of the economically active Indian population were employed in three major industrial groups: manufacturing (37,7%), commerce (25,7%), and services (13,2%). About 75% of the economically active population were males and the activity rate was 51.6%. This population made up about 3% of the total South African economically active population, with whites making up 22%, coloureds, 11% and blacks, 64%. About 65% of the economically active population were following three occupational groups: clerical and sales workers (32,7%), mining, production and related workers (22,7%), and professionals, technical and related workers (10,7%) (Central Statistical Services, 1987). According to a Press statement from the Central Statistical Service, only 335 000 people from a total population of 951 000 were economically active by the end of March 1991. Of the economically active population, 320 000 were workers and 15 000

were unemployed. (The CSS defines the unemployed as anyone 15 years of age and older who are not employed.) (Sunday Times Extra, July 28, 1991).

A.8 DISTRIBUTION OF THE INDIAN POPULATION ACCORDING TO GENDER

Of the 107 529 indentured Indians who arrived in Natal between 1860 and 1911, 61% were men, 25% women and 14% children. Thus in 1904 the masculinity rate was 207,5%. Over the years the number of females increased at a faster rate and by 1960 the masculinity rate was 103% and by 1985 the masculinity rate was 98%.

A.9 DISTRIBUTION ACCORDING TO AGE

In 1951 the 0-4 year group was 18,2% but fell to 12,5% in 1980, and the same applies to the 5 to 14 year group. There was a substantial increase in the 25 to 44 year group by 1980 and a slight increase in the 45 to 64 year group.

A.10 LEVEL OF EDUCATION

Since 1960 the level of education has improved considerably. Whereas in 1960 about 38% of the community had no education and 15% of them were below school-going age, in 1980 about 26% had no education and of this about 12,5% were below school-going age. The education of females has also improved tremendously as in 1960, 23% were uneducated and in 1980 only 15% were uneducated. The proportion of the population with Standard 10 and better increased from 1,4% in 1960 to 3,42% in 1970 and to 8,62% in 1980.

APPENDIX B

INDIAN CUISINE

B.1 THE HISTORY AND DEVELOPMENT OF INDIAN COOKING

In a vast country such as India, food varies from region to region and traditional dishes are as strongly influenced by religion and custom as they are by geography (Hale, 1989). The "halaal" and "haraam" injunctions of the Muslims, the "Kosher" of the Jewish people, the vegetarianism of Hindus and Buddhists, the gourmandism of Chinese dishes, are all due to restrictions (or lack thereof) imposed by religion. Travel, emigration and transportability of products have been contributory factors in the attainment of international status of some dishes, e.g. rice and curry/ tarkari/goulash (Mayet, 1987).

South African Indians are well-versed with traditional Indian food dishes that have been handed down through the generations in families, usually from mother to daughter. Variations in combination of spices and condiments as well as preparation methods of the different basic food articles occur within families as well as among different ethnic groups but the cooked meal is usually a perfect blend of the added spices, with no one spice so strong as to dominate the dish, unless this is particularly desired by the cook.

Central Asian Muslim cuisine, which is based on meat, was introduced to

India by the invasion of the Moghuls during the sixteenth century. The Muslim influence was strongest in North and Central India where meat cooking is at its best. The cooking in the South of India is mainly vegetarian, except in those coastal pockets where Christian, Zoroastrian, Jewish and Muslim influence has predominated (Hale, 1989). Regional cooking is also influenced by the staple food of the area. In the North of India where wheat grows, the food is drier and the sauces thicker than in the South, where rice is the staple diet. Indians eat food with their fingers and the chapati (roti) or flat, unleavened bread, is used to wrap and pick up dry food. In the South, the more liquid curries are better eaten with rice, which is more absorbent. Here, climate also plays an impoprtant role. The heavy rainfall contributes to the growth of an abundance of vegetables that make the vegetarian cuisine varied and exciting.

Most of India's large population are Hindus who never eat beef because they regard the cow as sacred. The majority are vegetarian, especially people from the higher castes. There are exceptions, such as the Brahmans of Kashmir, who being influenced by long years of Muslim rule, eat mutton. The Brahmans of Bengal and the Saraswat Brahmans of Mangalore eat fish because it is abundantly available, cheap and delicious. There is a large Muslim minority as well as smaller minority communities, which include the Catholics of Goa, the Syrian Catholics of Kerala, the Parsees and Jews of the West Coast and the reformed Hindu community of Jains, each having its own distinctive cuisine.

India is the land of home cooking as restaurants are unable to compete with private homes in the quality of ingredients used and the care taken in the preparation of each dish.

"Ghee" and oils have been described as "big items" in Indian cookery and have been recommended in many recipes. However, it is noted that "minimum" quantities must be used as "excess fat is wasteful and detrimental to health", in the "Indian Delight" (Mayet, 1987) recipe book, but generally housewives, especially the older generation, do not use ghee sparingly. "Ghee" (clarified butter) is usually made at home in India as well as in South Africa. However, in India, because "ghee" is so expensive, when necessary, is bought at a dairy. Thus, most households use the cheaper vanaspati, a hydrogenated vegetable fat. In the present study no mention was made of vanaspati, and its use could be recommended.

B.2 THE ROLE OF SPICES

Mayet (1987) mentions that traditional Indian cooking has its origin in the effort to preserve foodstuffs during the season of abundance in order to provide for leaner days to come. Early in the history of mankind, it was discovered that products which were slowly dried in the sun, kept for a longer period and rapid deterioration could be prevented by sprinkling certain powdered roots over meat. Thus, spices were originally used to combat deterioration and for their medical properties. The fact that the palate soon acquired a taste for them was secondary. Highly spiced

dishes have originated from countries with tropical and equatorial climates as nature has a way of compensating for its own drawbacks. Thus, in colder climates where food does not deteriorate so rapidly, spices do not grow in abundance as they do in those places where their need is essential. For the masses who relied on the products of their environment, this easy access to spices soon laid the foundation for "typical dishes" and national appetites.

Spices should be used sparingly as some are used for flavouring, some for colouring, some for tenderising, some for aiding digestion and some for decreasing flatulence (Mayet, 1987).

In Indian cooking, masala denotes the combination of spices and herbs which gives each dish its individuality (Hale, 1989).

Masalas may be either wet or dry. Wet masalas, which must be used immediately are ground with vinegar, water or coconut milk and form the base of all the spicy dishes cooked in the coastal areas of South India, while dry masalas, which do not have to be freshly ground each day, are more commonly used in the North. Over a hundred spices are known to Indian cooking. Spices are imported from India and East Africa by Indian South African spice merchants and thus most are available here.

The basic spices used for Indian cooking have been described in Chapter 3.

B.3 TYPICAL COOKING METHODS OF SOUTH AFRICAN INDIANS

Chillies are an essential ingredient to Indian cooking which provide the Oriental piquancy of these dishes (Hale, 1989). Fresh chillies are green or red and can be used fresh or dried. Dried chillies can be ground and stored. Different varieties and sizes are available which could be mild or fiery hot and thus must be used with discretion. The seeds are the most pungent.

Ginger, garlic and onions are most commonly used as flavourings and which give body to a dish. To make chutneys and sauces, fresh herbs such as dhanya (coriander leaves), curry leaves (kari patha), phudina (mint), tulsi (sweet basil), and tomatoes together with chillies, garlic and spices are used.

Imli (tamarind), vinegar and lemon juice are used as souring agents.

All of these spices and condiments are used to prepare both vegetarian and non-vegetarian meals. Vegetable curries are made from one or a number of vegetables. Pulses or lentils (dhals), of one kind or another, are a tasty and inexpensive source of protein. Although they form the most important part of vegetarian diets, they also form a major part of non-vegetarian diets and may be cooked with meat, fish and poultry. In India, there are nearly sixty varieties of dahls, of which the most common ones, such as moong (chinese chick peas), green and yellow masoor (Egyptian lentils), and channa (split peas), toovar (pigeon-pea), lombia

(black-eyed peas) and ramjah (red kidney beans) are all available in the West.

Modern methods of egg farming in India have made them acceptable by Indian vegetarians. In South Africa, there are ovo-vegetarians, lacto-ovo-vegetarians, acto-vegetarians and pure vegetarians.

In India, very little beef is eaten, while pork is eaten mainly by Christian communities on the west coast. Thus, generally meat-eaters eat mutton or chicken. In South Africa, the Muslim non-vegetarians eat mutton, beef and chicken, but not pork, while Hindu non-vegetarians do not eat beef. These are religious restrictions.

There are many different methods of cooking meat. Besides the popular curries, there are kormas (braised meat) that are cooked in yoghurt or cream or both. There are various kinds of kebaabs and sautéed and baked meat (Hale, 1989). Spicy meatballs (koftas) which may be plain or stuffed are served either dry or in a curry sauce, while roasted meats include those cooked on a split in a tandoor (clay oven). Duck, goose and partridge are eaten more often in India, but rarely in South Africa.

India boasts a coastline of over two and half thousand miles, providing thousands of varieties of fish and a large repertoire of seafood dishes (Hale, 1989), which are curried and spiced.

In India, in a middle-class Indian home, the main meal of the day usually

consists of two or three vegetable dishes, one of which would be dahl, a meat or a fish dish, if the household is not vegetarian, together with yoghurt, pickles and chutneys. Theses are served with chapatis (rotis), or rice, or both. A sweet dish is often included and eaten first or with the other dishes, rather than afterwards as is the custom in the West (Hale, 1989). The eating trend among South African Indians is much the same as in India. All the food is served at once, rather than in courses, and individuals can combine the different dishes to suit personal taste.

Some "typical" Indian food recipes are given in this section.

Curry Preparation

Curry (or tarkari) is the dish eaten with roti or rice. Finely sliced onions fried golden brown in ghee/oil form the basis of all good curries. To this are added meat/chicken/fish/vegetables which had been marinated in ginger/garlick/chillies and spices. Tomatoes which are finely cut/grated/minced/pulverised in a liquidiser are added to curry. The curry is cooked on low heat until the ingredients are tender.

Basic Curry with Gravy (eaten with roti, bread, or rice)

500 g meat or chicken
2-3 onions
1/4 cup ghee/oil
1 tsp. ginger - freshly pounded
½ tsp. garlick - freshly pounded
1 tsp. salt

1 teaspoon turmeric

½ tsp. ground coriander seeds

2 cardamons

½ tsp. ground cummin seeds

2-3 cloves and peppercorns

1 piece cinnamon (stick)

1 tsp. red chillies (freshly pounded or powder)

2 medium tomatoes

5 small potatoes - peeled and quartered

The cooked curry may be garnished with coriander leaves.

Vegetarian curries are cooked in a similar way using vegetables, lentils (dhals) and pulses. A variety of spices are added to the curry to produce different or desired flavours and aromas. Each ethnic group among the South African Indians has a special way of preparing specific dishes and cooking methods may vary from family to family as daughters are taught to cook in their early teens by their mothers, aunts and grannies.

Roti Preparation

Rotis are unleavened Indian type bread which are also known as chapatis, while rotlas are unleavened Indian type bread made from sorghum flour or mealie-meal. Rotis and rotlas are flat discs usually rolled out to a diameter of about 15 cm or more weighing about 50 g, but they could vary in size and weight as the housewife desires them. These discs are fried on hot griddles in ghee or oil.

Basic recipe for roti is given in this section, although it can vary from housewife to housewife with satisfactory results.

White/Brown Flour Roti

2 cups brown or white flour

1 tablespoon ghee/oil

2 tablespoon milk

1 cup boiling water

pinch of salt

Mix flour with ghee/oil and salt. Add milk and water and knead to a soft pliable dough. Divide dough into 4 sections. Roll out on a floured board into a flat disc. Brush with melted ghee/oil and sprinkle a little flour. Fold over in half. Brush top of fold and sprinkle with flour and fold over again. Fold into a circle and roll out to a diameter of about 15 cm. Place on hot griddle and turn over several times till lightly freckled. Brush over with melted ghee/oil and brown well on both sides.

"Haleem" (Broth-type Soup)

500 g leg mutton/chicken/trotters

200 g wheat

100 g barley

25 g "chana" (gram) dhal - boiled in water till tender and liquidized

10 g fresh ground garlic

5 ml salt

1 medium onion - sliced thin

2½ ml turmeric power ("haldi")

2½ ml dry red chillie powder

2½ ml whole cummin seeds ("jeera")

5 ml "garam masala" (mixed ground spices)

2 whole cardamon ("alachi"), 4 peppercorns, 4 cloves, 4 pieces cinnamon

2½ ml green chillies - chopped fine

2½ ml ground coriander ("dhana") seeds

2 whole green chillies sliced in half lengthwise

50 ml oil

30 ml "ghee"

15-20 ml chopped shallot

15-20 ml chopped coriander leaves, 2 sprigs fresh green mint

Method:

Add all spices, ginger, garlic, chillies to washed pieces of mutton/chicken/trotters and marinade for 1 hour. Braise sliced onions, jeera, cloves, peppercorns and cinnamon in oil until onions turn to a pale gold colour. Add mutton/chicken/trotters marinade. Mix well and cook slowly. Add water to fill pot. Add gram dhal, wheat and barley and cook for 3-4 hours until the haleem resembles a broth.

Braise a few onion rings and 1 ml jeera in the ghee until onions are a pale gold colour. Pour over haleem. Add "garam masala". Garnish with

chopped coriander leaves, shallot and a few sprigs of fresh mint just before serving.

Burfee

500 g Klim (powdered milk)
2 ml ground cardamon seeds
250 ml Ultra Mel fresh cream
1 ml rose essence
400 g icing sugar
50 g butter
1 cup water

Mix Klim powder and cream together until mixture resembles fine bread crumbs. Mix icing sugar, water and butter in a pot. Boil mixture. Reduce heat. Add Klim mixture and stir well. Cook slowly for 5-10 minutes. Add rose essence. Cool and set in a flat dish about 4 cm thick. Decorate with slivered almonds and pistachios. Cut into squares and serve.

Chana Magaj

100 g Klim (powdered milk)
500 g "chana" (gram) flour
20 ml "ghee"
400 g icing sugar

125 ml milk

500 g (2 cups) "ghee"

50 g almonds or pecan nuts - chopped fine and lightly toasted in oven

Add 20 ml "ghee" to gram flour and rub mixture till it resembles fine bread crumbs. Heat 500 g "ghee" in a pan. Add the crumbed mixture. Cook slowly and continue stirring to prevent scorching, until mixture turns a pale beige colour. Add sifted icing sugar slowly while preventing lumps from forming. Add almonds/pecan nuts. Cool and set in a flat dish about 4 cm thick. Decorate with slivered almonds and cut into squares.

B.4 GLOSSARY OF INDIAN FOODS

(vegetarian and non-vegetarian)

Aamli sauce - tamarind sauce

Aloo piaz saag - potato curry

Aloo saag - potatoes and spinach

Anda kari - egg curry

Baingan Bharta - aubergene (brinjal) pureé

Bhajias - chilli bites

Bhinda curry - okra curry

Bhuré Parathas - stuffed wholemeal fried bread

Biryani - exotic rice dish of India

Boti kebabs - spiced lamb kebaabs

Boti kebabs - spiced lamb meatballs

Chaat - special moong sprout

Chana chatpati - chick pea savoury

Chana dhal curry - chick pea curry

Chapli kebab - fried spicy meat patties

Coconut milk - infusion made from fresh grated coconut and hot water

Dahin aaloo - potatoes cooked with plain yoghurt

Dhal gosht - mutton/chicken cooked with lentils

Dhunia/mint dahi - coriander/mint chutney in plain yoghurt (to serve with biryani)

Dhunia chutney - coriander chutney

Dokras - savoury dumplings

Doodhi moothias - marrow dumplings

Doodhi curry - Indian calabash curry

Falooda - agar-agar and milk jelly

Fish masala curry - spiced fish curry

Ghawla - Indian pancake

Goolgoolas - sweet fritters

Goovar phalli curry - curry of narrow Indian beans

Haleem - Indian mixed soup or broth with herbs, meat, spices and dhals

Halwa, plain soji - taystee wheat halwa

Haranga - sprouted moong curry

Jardo - sweetened rice dish

Korma - curried lamb

Kofta kari - meatball curry

Khitchri - rice with lentils

Khari - sour milk gravy or soup

Kheera ka raita - yoghurt salad

Kalya-E-Khaas - chicken/mutton curry cooked in yoghurt

Kachoomer - grated vegetable salad

Khaman - pea flour savoury

Khitchro - mixed cereal broth

Khima puri - mince stuffed deep-fried Indian bread

Karela curry - bitter ground curry

Laai - milk and almond drink

Lagan - sour milk drink

Murghi pilaau - chicken pilaff

Moghlai korma - lamb cooked with yoghurt

Masala bangra - stuffed herrings

Moong ki dhal - green Chinese pea curry

Methi mango achar - fenugreek seed and mango pickle

Methi bhaji with khima - fenugreek leaves cooked with minced meat

Mitha samoosa - samoosa pastry with sweet filling

Mithi roti - roti with sweetened oil dhal filling

Mong dhal bhajias - Chinese pea chilli bites

Meen Molee - fish coconut curry

Naan - round leavened bread

Naan khataay - Indian shortbread with ghee

Ojri curry - tripe curry

Puri - deep fried bread/roti

Parathas - shallow fried bread

Papads/papadums - paper thin discs made from lentil/gram (chana)/rice flour

Pattas/pattarbelli - madumbi leaf rolls or pinwheels

Patta ni gant - madumbi (Arvi) or yams

Plain yakhni pilaau - mixed rice, mutton/chicken, dhal pilaff

Phulkas - wholewheat, small, thin pancake-like unleavened bread (toasted with or without oil/ghee

Rawayya - stuffed brinjal (eggplant)

Roti - flat unleavened bread discs

Samoosa - mince filled triangular or pyramid shaped pastry savouries

Seekh kebaba - meat-balls

Seviyan - vermicelli dessert

Soji Halwa - semolina dessert

Sutherfeni - sweetened flakey hair-thin strands twirled into pinwheels (granny's hair)

Soowa bhaji with moong dhal - dill herbs with green Chinese peas (split)

Sekta Sing curry - drumstick curry

Toovar or chana dhal dhokras - oil or gram lentil dumplings

Torai and sev curry - ridge gourds and semolina curry

Tikka - chicken or mutton cubes skewered and cooked over an open fire

Tandori murghi - spiced chicken roasted in clay oven

Urad dhal broth - split black bean broth

Vadde - South Indian chilli bite with mung dhal, soaked split peas or white long dhal (vhal dhal)

Vathoo kuri - duck curry

Vegetable cutlets - a mixture of vegetables made into cutlets.

APPENDIX C

No. ☐☐☐☐ 4

QUESTIONNAIRE 2: SUBJECT DETAILS

CORONARY HEART DISEASE RISK FACTOR

SURVEY QUESTIONNAIRE

DEMOGRAPHIC DATA :

NAME: _____ CLASS: _____

ADDRESS: _____

_____

TELEPHONE: _____ DATE OF BIRTH: _____

DATE OF INTERVIEW _____

DAY OF INTERVIEW:

MONDAY	= 1	TUESDAY	= 2	WEDNESDAY	= 3	☐ 5
THURSDAY	= 4	FRIDAY	= 5	SATURDAY	= 6	
SUNDAY	= 7					

PLEASE ANSWER THE FOLLOWING QUESTIONS:

1. SEX: MALE = 1
FEMALE = 2 ☐ 6

2. RELIGION: HINDU = 1 CHRISTIAN = 2 ☐ 7
MUSLIM = 3 OTHER = 4

3. YOUR AGE LAST BIRTHDAY : ☐☐ 8 9

4. WHAT ARE YOUR PLANS UPON LEAVING SCHOOL?

Study = 1
Work = 2 ☐ 10
Get Married/Housewife = 3

5. IF YOU PLAN TO STUDY :

Will you study at a local institution, = 1
Or elsewhere? = 2 ☐ 11

MEDICAL HISTORY OF SUBJECT AND FAMILY:

6. Were you informed/or do you know whether you have/or have you had any of the following?

YES = 1 NO = 2 DO NOT KNOW = 3

Hypertension (High Blood Pressure)	= 12	☐
High Blood Cholesterol or Blood Fats	= 13	☐
Obesity (Overweight)	= 14	☐
Constipation	= 15	☐
Diabetes	= 16	☐
Heart Disease	= 17	☐

7. Were you informed/or do you know whether any of your first degree relatives have/or have had any of the following?

Mother = 1, father = 2, sister = 3 (state number of sisters), brother = 4 (state number of brothers)

Hypertension	= 18-25	☐☐☐ 18
High Blood Cholesterol or Fats	= 22-25	☐☐☐ 22
Obesity	= 26-29	☐☐☐ 26
Constipation	= 30-33	☐☐☐ 30
Diabetes	= 34-37	☐☐☐ 34
Heart Disease	= 38-41	☐☐☐ 38
Stroke	= 42-45	☐☐☐ 42

8. Has any one of the above died? If yes, state at what age they died.

Mother = 46, 47 ☐☐ 46

Father = 48, 49 [ ][ ] 48 4

Sister(s) = 50-53
(If more than 1 sister has died, state the age of the eldest and the youngest) [ ][ ][ ][ ] 50 51 52 5

Brother(s) = 54-57
(If more than 1 brother has died, state the age of the eldest and the youngest) [ ][ ][ ][ ] 54 55 56 5

9. Are you on any special diet for :

YES = 1 NO = 2 DO NOT KNOW = 3

Hypertension	= 58	[ ] 58
Diabetes	= 59	[ ] 59
High Blood Cholesterol or Blood Fats	= 60	[ ] 60
Obesity	= 61	[ ] 61
Constipation	= 62	[ ] 62
Heart Disease	= 63	[ ] 63
Any other diet (eg. for acne)	= 64	[ ] 64

GENERAL DATA:

10. Do you use any medication at the moment?

YES = 1 NO = 2

Antibiotics	= 65	[ ][ ] 65 66
Drugs	= 66	

SMOKING HABITS:

11. Do you smoke?

YES = 1 NO = 2 [ ] 67

12. If you do smoke:

i) How old were you when you began to smoke? [ ][ ] 68 69

ii) Do you smoke: Cigarettes with filter = 1
Cigarettes without filter = 2
Cigars = 3
Pipe Tobacco = 4

iii) Do you inhale?

YES = 1 NO = 2

13. If you smoke cigarettes, how many do you usually smoke:

During weekends per day?

During the week per day?

NUTRITION:

14. Are you a:

Pure vegetarian (consumer of plant foods only) = 1

Lacto-Vegetarian (consumer of plant foods with dairy products) = 2

Lacto-ovovegetarian (consumer of plant foods with dairy products and eggs) = 3

Non-vegetarian (consumer of plant and animal foods) = 4

Other (specify) = 5

SOCIO-ECONOMIC STATUS (Socio-Economic Data of Nutritional Relevance)

15. Occupation of main wage earner/s.

Professional = 1
General Dealer = 2
Executive (Business) = 3
Manual Worker = 4
Salesman = 5

16. How many persons usually eat at home?

Adults (including yourself) = 78

5 - 16 years = 79

2 - 4 years = 80

Under 2 years = 81

DRINKING HABITS:

17. Do you consume alcoholic beverages?

YES = 1 NO = 2 [ ] 82

18. If so, what do you drink?

Beer	=	1	[ ] 83
Wine	=	2	[ ] 84
Spirits	=	3	[ ] 85
Other	=	4	[ ] 86

19. How often to you drink?

/day _____	=	1
/week _____	=	2
Occasionally _____	=	3

[ ] 87

PHYSICAL ACTIVITY AND EXERCISE:

20. How many hours per week do you spend:-

(a) walking to and from school, shops, sport, and helping with housework? [ ][ ][ ] 88 89 90

(b) on hard physical labour (sweat work) (eg. strenuous gardening) [ ][ ][ ] 91 92 93

(c) on exercises, tennis (competitive), skipping, squash, netball, soccer, swimming (heart rate increased) [ ][ ][ ] 94 95 96

Bortner Scale

Please answer the following questions about your personality. Two extreme characteristics are mentioned. Please indicate where you fit in on a scale between 1 and 7. There is no correct or incorrect answer. Please answer as you see yourself.

Example:

Do you get angry quickly or does it take a lot to make you lose your temper?

If "get angry quickly" = 1 to 3 and
"seldom gets angry" = 4 and
"take a lot to get angry = 5 to 7,
where do you fit in?

		1 2 3 4 5 6 7		
	Type A		Type B	
(1)	Are you never late - always on time?	1 2 3 4 5 6 7	Are you casual about appointments. Do not watch the clock?	1
(2)	Do you go all out to be successful in your work and relaxation?	1 2 3 4 5 6 7	Are you casual about tasks. Not bothered?	2
(3)	Are you a hurried person when you walk or eat. Tasks must be completed fast?	1 2 3 4 5 6 7	Are you not a hurried person? Do tasks peacefully. Can allow a task to take its own time.	3
(4)	Do you want to be the best at everything in life/always want to come out on top?	1 2 3 4 5 6 7	Are you content not always being the best in life/not always coming out on top?	4
(5)	Do you feel impatient when waiting?	1 2 3 4 5 6 7	Can you wait patiently?	5
(6)	Are you hard-driving? Do you work without resting?	1 2 3 4 5 6 7	Are you easy-going?	6

RESULT SHEET

WEIGHT _____ kg.

HEIGHT _____ cm.

MID-UPPER-ARM CIRCUMFERENCE _____ cm.

BODY MASS INDEX _____ kg/m^2

BLOOD PRESSURE : SYSTOLIC _____ mmHg

DIASTOLIC _____ mmHg

TOTAL SERUM CHOLESTEROL _____ m mol/l

HDL - CHOLESTEROL _____ m mol/l

LDL _ CHOLESTEROL _____ m mol/l

HDL - RATIO _____ % of TC

TOTAL TRIGLYCERIDE (Fasting) _____ (m mol/l)

FIBRINOGEN _____ (mg/dl)

FACTOR VII _____ (ratio)

APPENDIX D

CONSENT FORM

University of the Witwatersrand
Department of Medicine
Medical School
York Road
Parktown 2193

Telephone: 488-4911 Ext. 3627/3626/3818

IMPORTANT MEDICAL APPEAL TO ALL MATRICULANTS

Dear Parent/Guardian

During the years in practice in Lenasia it was clear that heart attacks are all too frequent in our community. We have the unenviable reputation of having the highest deathrate for heart attacks in the world. The risk factors for heart attacks are believed to be high blood pressure, sugar diabetes, lack of physical activity, stress and a high blood fat (i.e. Cholestrol). In order to reduce the frequency of heart attacks we need to determine the important risk factors in our community. We believe that dietary factors are important in the causation of heart attacks. School children form a unique part of our population in that they are free of high blood pressure, diabetes and are active physically.

The aim of our investigation is to determine the blood fats in matriculants and to determine the eating habits of these pupils. By performing a simple blood test we are able to identify those persons who are at risk long before the development of heart disease. By doing so we are then in a position to observe these individuals closely at a time when effective treatment is available.

It is in the interest of your child's health that I appeal to you to allow us to obtain a small sample of blood from him/her after an overnight fast (i.e. from midnight to 8.30 am, with any amount of water allowed). The procedure is harmless and relatively painless.

Please complete the attached form of consent and return to the school principal.

Yours faithfully,

M.S. ASVAT
Specialist Physician

CONSENT FORM

I .....
(Parent or Guardian's name)
agree to have my son/daughter
.....
(Pupil's name)
tested for the investigation.

Date: ....
(Parent/Guardian's signature)

APPENDIX E

COMPUTER CODE FORM : USED TO CODE FOOD INTAKE

	Subject Number				Computer Code				
1									8
9									16
17									24
25									32
33									40
41									48
49									56
57									64
65									72
73									80

	Subject Number				Computer Code				
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9									16
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49									56
57									64
65									72
73									80

APPENDIX F

QUESTIONNAIRE 3 : FREQUENCY QUESTIONNAIRE ON HABITUAL FOOD INTAKE

SUBJECT NO: .....

POTCHEFSTROOMSE UNIVERSITEIT VIR CHRISTELIKE HOËR ONDERWYS

NAME: ..... SEX: ..... CLASS: .....

ADDRESS: .....

TELEPHONE: ..... DATE OF BIRTH: .....

PLEASE ANSWER THE FOLLOWING QUESTIONS:

1. Are you on any special diet? (specify type and reason) .....

2. Do you supplement your diet with any of the following?

   (specify types and amounts) Vitamins .....
   Tonics .....
   Health foods .....
   Body building preparations .....
   Dietary fibre .....

3. Do you use extra salt at the table? How much? .....

4. How often do you eat curried food? .....

5. Do you eat curry mild or strong? .....

6. How often do you eat garlick .....
   ginger .....
   chillies .....
   herbs ..... (specify types)

7. Do you trim the fat from meat before it is prepared or cooked? .....

8. How many times a day do you eat? .....

9. Do you usually have breakfast? .....

10. What and when do you usually eat between main meals? .....
    .....

THE QUESTIONNAIRE IS ABOUT THE FOOD YOU USUALLY EAT.

Please indicate the amount* and the number of times that you eat this food. If there are many options per question underline and indicate the amount for each of those (use, # or any sign to indicate which food is realted to which amount).

* Amounts (use the symbol between brackets on the questionnaire). grammes (g); cups (c); teaspoons (t); tablespoons (T); millimeters (m); slices (s); portions (p).

State the thickness of your bread slices; regard 1 cm and less as (thin), between 1 and 2 cm as (medium) and thicker than 2 cm (thick).

FOOD ITEM AND DECRIPTION	Amount when eaten (C,T, etc)	Times eaten per day	per week	per month	never/ seldom
VEGETABLES: (state/fresh/frozen/canned)					
HOW MANY PORTIONS OF VEGETABLES DO YOU HAVE PER DAY?					
Raw salad vegetables e.g. tomatoes, peppers, lettuce, onions, cumcumbers, etc.					
Cook salad vegetables, e.g. beetroot, asparagus					
Grean leafy vegetables, e.g. spinach, marocho etc.					
Onions/tomatoes (cooked)					
Yellow vegetables e.g. pumpkin, squashes, carrots					
White vegetables e.g. turnips, parsnips, cauli= flower					
Sweet corn/baked beans					
Mixed vegetables					
Mushrooms, brinjal or others					
Potatoes (boiled, baked, mash, potato salad					
Chips (fried, oven/homemade)					
White/cheese sauce					
FRUIT: (state fresh, canned, dried, sugarglazed					
HOW MANY PORTIONS OF FRUIT DO YOU HAVE PER DAY?					
apples/pears (peeled/unpeeled)					
Stone fruits (peaches, plums etc.)					
Citrus					
Bananas					
Guavas					
Other (specify)					
Avocados/olives					
Berries (state)					
Stewed or canned fruit (with sugar/without)					
Dried fruit (state)					
Fruit rolls/glazed fruit					
Fruit salad (fresh/canned)					
FATS AND OILS: On bread, in salads, on vegeta= bles					
Butter					
Ghee (clarified butter)					
Margarine (hard or soft)					
Dripping, lard, hard fats, greaves					
Mayonnaise, salad dressing, french dressing, oil					
DRINKS: Tea/coffee					
Sugar used per cup					
Fruit juices (fresh, liquifruit, etc.)					
Fruit squashes (Oros, etc.)					
Fizzy drinks (lemonade, Coco Cola, etc.)					
Maghau					

)OD ITEM AND DESCRIPTION	Amount when eaten (C,T, etc)	Times eaten per day	per week	per month	never/ seldom
:CHOLIC DRINKS:					
antu Beer (Tlokwe, homemade)					
:er, stout, cider					
ierry, vermouth, dessert wine (state sweet/ :dium/dry)					
ible wine (medium/sweet/dry)					
)irits (brandy, whisky, gin, etc.)					
:quers					
:xed (state)					
/EET MEATS (specify)					
ny other food not mentioned (specify)					

APPENDIX G

DEMOGRAPHIC DATA OF LENASIA AND ITS POPULATION

HISTORICAL OVERVIEW

In 1954, a military camp which was situated about 32 kilometres south west of the Johannesburg Central Business District (CBD) area under the command of General Lenz, accommodated the first group of low income Indians in camp housing (Ally, 1989). Subsequently the eastern part of the camp which was separated from the main camp by a railway line, was named Lenasia and a few homes built there. A school was built with prefabricated building material to serve as a primary and high school which were then divided into the Lenasia Indian High School and Alpha Primary School. Pupils were transported by trains and buses from various areas in Johannesburg.

People who were unable to obtain homes in Johannesburg bought land and built homes in Lenasia, mainly teachers and some professionals. A private company built homes from 1954 onwards for people who moved to Lenasia from the Johannesburg suburbs of Fordsburg, Vrededorp, Newclare, Martindale, Newlands, Denver and Sophiatown. This was in accordance with Government policy of separate areas from different racial groups.

In 1965, with the passing of the Group Areas Legislation and the Community Development Act, the township was expropriated by the Group Areas Development Board from the private developers. This board undertook to build sub-economic and economic houses which were difficult to obtain and there has always been a housing shortage.

ADMINISTRATION OF LENASIA

From 1966, Lenasia was administered by the Transvaal Board for the Development of Peri-Urban Areas. In 1970 its administration was incorporated into that of the Johannesburg City Council which provided services such as electricity, refuse removal and sanitation.

Lenasia is divided into 11 extensions and appears like a blend of the orient and any new South African suburb. At present the different extensions are controlled by three authorities; (i) Johannesburg City Council and the Lenasia Management Committee, ie. the City Council's Indian representative body; (ii) the Transvaal Peri-Urban Board which controls Lenasia East/South (Extensions 8, 9, 10 and part of 11) and the Peri-Urban Board's appointed Indian representative body - the Lenasia East/South Consultative Committee; (iii) the Department of Local Government Housing and Agriculture which controls housing matters.

SERVICES AND AMENITIES IN LENASIA

Lenasia has a Civic Centre where residents pay rentals, water and electricity bills and rates and taxes. The Lenasia Clinic is part of this Centre. Innoculations against notifiable diseases are administered to childrten from birth onwards at this centre. It also houses a family planning clinic. There is also a well-equipped library at the Centre.

Other facilities include a post office, tele-communication service, fire brigade, bus service to Johannesburg, train service, a central business area and smaller shopping complexes and petrol filling stations.

There are many mosques, Hindu temples, churches and a cemetery for the different religious denominations.

There is an old-age home catering for a very limited number of people.

There are two recreational centres and private gym facilities at three centres.

One hospital has been built in the Lenasia South area. There is one private clinic run by a consortium of doctors. There is one out-patient clinic with certain week days set aside for the 'hypertension' clinic and 'diabetic' clinic.

Religious instruction is given to Muslim children on a daily basis, usually in the afternoons, after school. These institutions known as 'madressas' are run by private organizations and receive financial aid from parents and private donors.

Hindu children may attend 'Gujerati' classes in the afternoons at institutions which are also privately funded and managed.

Lenasia has many social welfare organizations which are privately managed and funded by donations and charities from the community. The organisations are the Johannesburg Institute of Social Services, Mental Health Society, Transvaal Indian Blind Association, Lenasia Heart Society, South African Tuberculosis Association, Cancer Society, Cardiac Rehabilitation Society, United Creative Enterprises which provides jobs for the mentally handicapped, M.C. Kharbhai School for the Deaf. All of these organizations provide facilities for the poor and needy and are managed by volunteer workers.

DEMOGRAPHIC DATA - SIZE OF COMMUNITY

The population of Lenasia constitutes the second largest concentration of Indians in South Africa; Durban and its surrounding areas in Natal, constituting the largest number. This population grew from 28 893 (1960 census) to a total of 110 000 in 1989 (City Council, President Association and Welfare organization estimates).

At present different extensions in Lenasia provide council housing for the lower-income group of people as well as owner-built homes for those from the upper-income groups.

When the present study was undertaken, there were six high schools in the area. Lenasia South has one more high school at present. There are over thirteen primary schools and three pre-primary schools for the 4-6 year olds.

The six high schools (see Table 1) from which the subjects for the present study were randomly selected are the following:-

1. Lenasia Secondary School. These pupils at this school are mainly from the upper socio-economic group.

2. Nirvana Secondary School. These pupils are a blend of upper, middle and lower socio-economic group.

3. Trinity Secondary School. Pupils at this school are from the middle to lower income group.

4. M.H. Joosub Technical College. Most pupils are from the lower income group and a few from the middle income group.

5. Azara Secondary School. Pupils at this school are from the middle to lower income group.

6. Topaz Secondary School. Pupils at this school are from the upper and middle and lower income group.

The socio economic composition at these schools is representative of the housing and income facilities of the areas surrounding each respective school.

Schools 1 - 4 have large school populations:- ± 1 000 pupils.

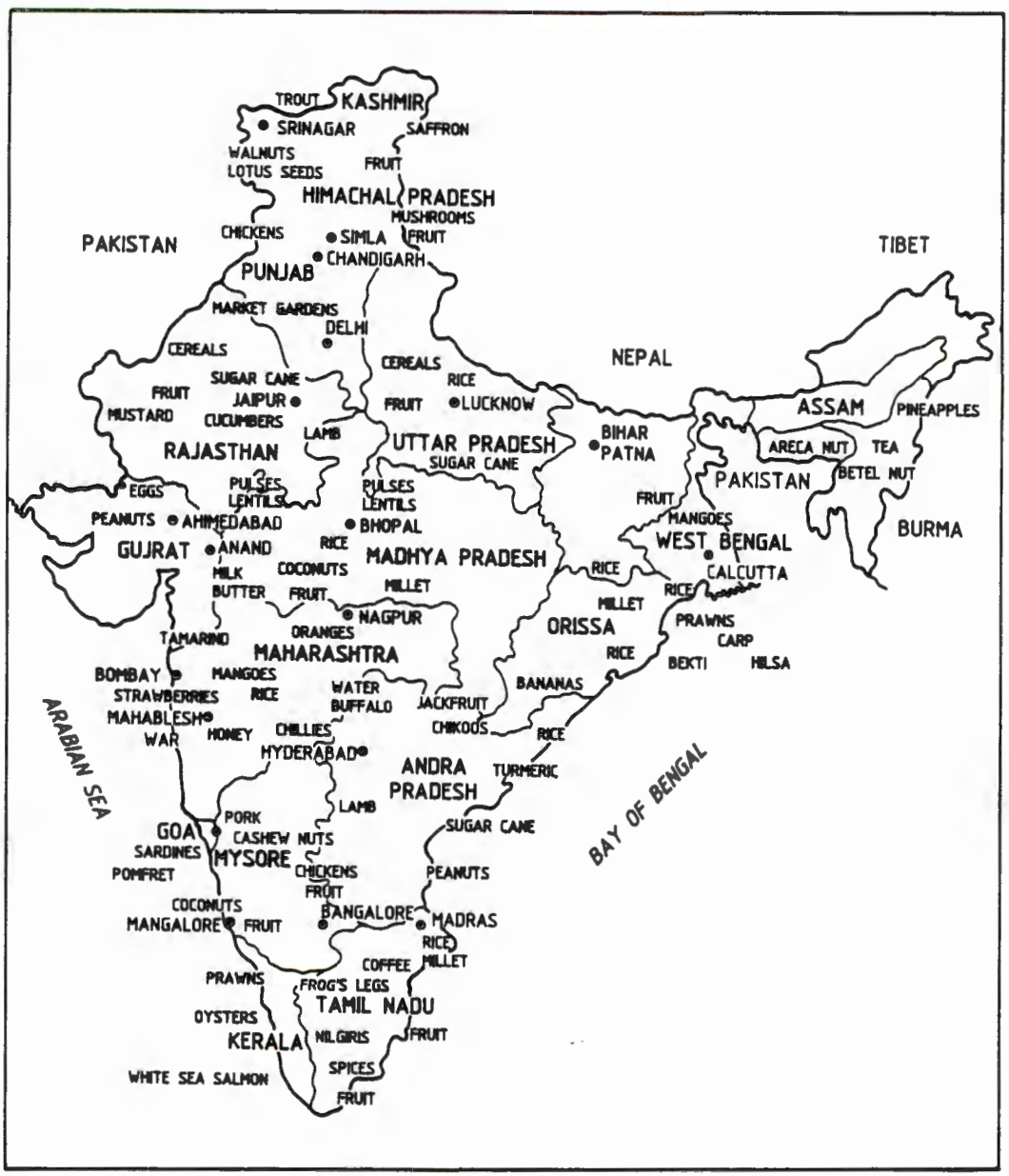
Schools 5 & 6 have about half the number of pupils compared with the larger schools.

TABLE 1. DISTRIBUTION OF MATRICULANTS IN LENASIA DURING 1988

SCHOOL	CONSENTED	DISCENTED	NO.OF FORMS NOT RETURNED	NO.MATRICULANTS
LENASIA SECONDARY	125	1	30	156
NIRVANA SECONDARY	132	41	14	187
TOPAZ SECONDARY	116	5	22	143
M.H. JOOSUB TECHNICAL COLLEGE	87	3	14	104
AZARA SECONDARY	39	5	5	49
TRINITY SECONDARY	45	0	9	54
TOTAL	544	55	94	693

APPENDIX H

MAP OF INDIA DEMARCATING THE VARIOUS REGIONS AND THE STAPLE FOOD OF THE AREA



LIST OF ABBREVATIONS

2,3-DPG	-	2,3-disphosphoglycerate
ACAT	-	acyl cholesterol acyl transferase
AHA	-	American Heart Association
ALA	-	alpha linolenic acid
Apo A-I	-	apolipoprotein A-1
Apo A-2	-	apolipoprotein A-2
Apo B	-	apolipoprotein B
Apo C	-	apolipoprotein C
Apo D	-	apolipoprotein D
Apo E	-	apolipoprotein E
ATP-ase	-	adenosine triphosphatase
BCS	-	British Cardiac Society
BF	-	butter fat
BMI	-	body mass index
BP	-	blood pressure
Ca	-	calcium
CAD	-	coronary artery disease
CHD	-	coronary heart disease
CLA	-	cis linoleic acid
CORIS	-	Coronary Risk Factor Intervention Study
DM	-	diabetes mellitus
DZ	-	dizygotic
ECTEP	-	cholesterol ester transfer and exchange proteins
EFA	-	essential fatty acid

EI - energy intake

EPA - eicosapentaenoic acid

Factor VIIa - activated factor VII

FH - familial hypercholesterolaemia

GLA - gamma-linolenic acid

GU - Gujerat University (India)

HCO - hydrogenated coconut oil

HDL - high density lipoprotein

Factor VIIc - factor VII concentration

IDDM - insulin dependent diabetes mellitus

HDL-C - high density lipoprotein cholesterol

HMG.CoA reductase - 3-hydroxy-3-methyl glutaryl CoA reductase

IHD - ischaemic heart disease

K - potassium

KHS - koronêre hartvatsiekte

LCAT - lecithin : cholesterol acyltransferase

LDL - low density lipoprotein

LDL-C - low density lipoprotein cholesterol

Lp(a) - lipoprotein (a)

LPL - lipoprotein lipase

Mg - magnesium

MI - myocardial infarction

MRFIT - Multifactorial Risk Factor Intervention Trial

MUFAT - monounsaturated fatty acid

MZ - monozygotic

Na - sodium

NaCl	-	sodium chloride
NHBLI	-	National Heart, Lung and Blood Institute
NIDDM	-	non-insulin dependent diabetes mellitus
PGI_3	-	prostacyclin I_3
P/S	-	polyunsaturated fatty acid/monounsaturated fatty acid ratio
PUFAT	-	polyunsaturated fatty acid
RFLPs	-	restriction fragment length polymorphisms
SATFAT	-	saturated fatty acid
SO	-	sunflower oil
TC	-	total cholesterol
TF	-	tissue factor
TG	-	triglyceride
USA	-	United States of America
VLDL	-	very low-density lipoprotein cholesterol
WHR	-	waist-hip circumference ratio
WHO	-	World Health Organization

CONGRESSES AT WHICH WORK WAS PRESENTED

1. Inaugural meeting of LASSA (Lipid and Atherosclerosis Society of Southern Africa). September 3-5, 1989, Johannesburg. M.S. ASVAT, F.B. MIA, B.I. JOFFE, S. BAKER, G. PILCHER, V. PANZ, H.H. VORSTER, D. MENDELSOHN and H.C. SEFTEL. The distribution of serum lipids, fibrinogen and factor VIIc in Indian matriculants of Johannesburg.

2. 13th Biennial Congress of the Nutrition Society of Southern Africa. 19-23 March, 1990, Cape Town. F.B. MIA, H.H. VORSTER, H.C. SEFTEL, M.S. ASVAT and A. KRUGER. Nutrient intakes and coronary heart disease risk factors of Indian matriculants.

3. Second National Congress of the Lipid and Atherosclerosis Society of Southern Africa. 1-3 March, 1992. F.B. MIA, H.H. VORSTER, H.C. SEFTEL and A. KRUGER. Nutrient intakes and coronary heart disease risk factor levels of Indian matriculants.

Parts of this work has also been presented at other congresses around the country by co-workers.

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