The prevalence of selected risk markers for noncommunicable diseases and associations with lifestyle behaviours in an Indian community in KwaZulu-Natal

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Thesis submitted for the degree Doctor of Philosophy at the Potchefstroom Campus of the North-West University

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November 2009

ABSTRACT

Globally, noncommunicable diseases have increased rapidly. They represent the major health burden in industrialised countries and are fast increasing in developing countries. In South Africa, a culturally diverse developing country, the scenario is no different: there is an increased prevalence of noncommunicable diseases amongst South Africans, and South African Indians in particular. Indian migrants throughout the world have a higher prevalence of diabetes, coronary heart disease and dyslipidaemia. Very few statistics exist on the health and nutrition profile of South African Indians.

The aim of this cross-sectional, epidemiological descriptive study was to conduct a survey on the prevalence of risk markers for noncommunicable diseases and on the associations of dietary and lifestyle exposures with the risk of noncommunicable diseases among apparently healthy adult Indians in KwaZulu- Natal, in order to make realistic recommendations for a sustainable intervention strategy.

Two-hundred-and-fifty apparently healthy Indians, aged 35-55 years, living in KwaDukuza, were randomly selected for this survey. Before the actual study, a quantitative food frequency questionnaire (QFFQ) was adapted for Indian diets and tested for comparative validity against three 24-hour food recalls as the reference method, and for reproducibility by a second administration in a subsample of 50 respondents. The QFFQ was found to be relatively reproducible but showed moderate-to-poor validity. When testing for reproducibility, the Spearman rank correlation coefficient was strongest for cholesterol (0.76) whereas total carbohydrate (0.153) accounted as the weakest non-significant correlation coefficient. With regards to the differences between the reported intakes of the two adrninistrations of the QFFQ, 13 of the 19 nutrients tested fell within a 10% difference thus showing good agreement. When the Bland-Altman procedure was used, there was significant proportional bias for all the nutrients except for saturated fatty acids. Furthermore, the proportion of individuals classified in the same or adjacent quartiles for energy and macronutrients intake ranged from 64% (total energy) to 92% (polyunsaturated fatty acids), showing relatively good agreement. In contrast, the results of the Spearman rank correlation and Pearson coefficients, paired t-tests, Bland-Altman technique and quartile distribution showed moderate to weak correlation for comparative validity between the two instruments. A

relatively low range for the Spearman rank correlation (-0.09 to 0.42) and the Pearson correlation coefficient (0.008 to 0.0556) was reported. Significant proportional bias was present for eight nutrients using the Bland-Altman technique and the proportion of individuals classified in the same or adjacent quartiles was relatively high.

Thereafter, the main study followed, examining the socio-demographic and anthropometric profile, diet, physical activity, blood pressure, fasting blood glucose, triglycerides and total cholesterol of respondents.

In order to determine the prevalence of risk markers, Asian cut-points were used. About 92% of respondents recorded diastolic blood pressure levels >85 mmHG, 88.8% of respondents recorded triglyceride levels >1.69 mmol/L, and 38.8% of respondents recorded fasting blood glucose levels >5.55 mmol/L. When using the cut-off points for central obesity as defined by waist circumference for men at ≥ 90 cm and women 80 cm, 100% of women and 87.4% of men were classified as centrally obese. In terms of physical activity, 62.5% of respondents were classified as inactive (<600 METS min). However, when using algorithms such as the European SCORE and a modified doubled score, respondents showed minimal risk for cardiovascular diseases.

The reported intake of fruit and vegetables were well below the recommended World Health Organization population nutrient goals (≥400g) were men reported a mean daily consumption of 221.9 g and women 236.9 g. In addition, 94.4% of respondents consumed below the estimated average requirement for fibre

Diet indices used to assess diet quality showed that the South African Indian diet reflects good diet quality. Two principal components were identified, based on the percentage of fat contributed to each food group: Factor 1 (Legumes, cereals and cereal products, vegetables), which accounted for added fat, and Factor 2 (Fats and oils, sugars and sweets and the milk group), which accounted for visible fat. According

to regression analyses, only fasting blood glucose was significantly inversely correlated with both factor scores for the whole sample.

The following associations of nutrient intake with clinical risk markers were noted: there were a number of significant correlations of risk score (the aggregate total of all risk markers defined by Asian standards) and risk score 1 (the aggregate of all risk markers with fasting blood glucose being doubled as compared to the other risk markers) with nutrient intake, where the percentage energy from fat showed the strongest correlation. Furthermore, waist circumference showed significant associations with most clinical risk markers. It was correlated with systolic blood pressure, fasting blood glucose, cholesterol, triglyceride and body mass index were body mass index showed the strongest association amongst all the clinical risk markers.

Collectively, the prevalence of risk markers and the noted associations of risk markers with diet and clinical parameters resonates into the call for dietary modification through increased consumption of fruit and vegetables, weight reduction, decreased intake of fat consumption and increased physical activity for a sustainable intervention strategy to reduce and control the burden of noncommunicable diseases in the Indian population of South Africa.

OPSOMMING

Nie-aansteeklike siektetoestande het wêreldwyd vinnig vermeerder. Dit verteenwoordig die belangrikste gesondheidslas in ontwikkelde lande en vermeerder vinnig in ontwikkelende lande. In Suid-Afrika, 'n kultureeldiverse en ontwikkelende land, is die scenario geensins anders nie: daar is 'n verhoogde voorkoms van nie-aansteeklike siektetoestande onder Suid-Afrikaners, en onder Suid-Afrikaanse Indiërs in die besonder. Indiese migrante dwarsoor die wêreld het 'n hoër voorkoms van diabetes, koronêre hartsiekte en dislipidemie. Baie min statistiek is beskikbaar oor die gesondheid- en voedingsprofiel van Suid-Afrikaanse Indiërs.

Die doel van hierdie dwarssnit, epidemiologies-beskrywende studie was om 'n opname te maak van die voorkoms van risikomerkers vir nie-aansteeklike siektetoestande en die verbande tussen dieet- en lewenstylfaktore en die risiko vir nie-aansteeklike siektetoestande onder oënskynlik gesonde volwasse Indiërs in KwaZulu-Natal, ten einde realistiese aanbevelings vir 'n volhoubare intervensiestrategie te kan maak.

Tweehonderd-en-vyftig oënskynlik gesonde Indiërs, 35-55 jaar oud, woonagtig in KwaDukuza, is ewekansig gekies vir die opname. Voor die werklike studie is 'n kwantitiewe voedselfrekwensievraelys (KVVV) aangepas vir Indiese diëte en vir vergelykende geldigheid getoets teen drie 24-uur-voedselherroepvraelyste as die verwysingsmetode, en vir herhaalbaarheid getoets deur 'n tweede administrasie in 'n subgroep van 50 respondente. Die KVVV is relatief herhaalbaar maar matig-tot-swak geldig gevind. Wanneer herhaalbaarheid getoets is, was die Spearman rangorde korrelasiekoëffisiënt die sterkste vir cholesterol (0.76) terwyl totale koolhidraat (0.153) die swakste nie-betekenisvolle korrelasiekoëffisiënt verteenwoordig het. Met betrekking tot die verskille tussen die gerapporteerde innames van die twee voedselfrekwensievraelyste, het 13 van die 19 nutriënte getoets binne 'n 10% verskil geval wat dus goeie ooreenstemming wys. Wanneer die Bland-Altman prosedure gebruik was, was daar betekenisvolle proporsionele sydigheid vir al die nutriënte behalwe vir versadigde vetsure. Verder het die proporsie individue wat in dieselfde of aangrensende kwartiele geklassifiseer het, gevarieer van 64% (totale energie) tot 92% (polionversadigde vetsure), wat relatief goeie ooreenstemming gewys het. In teenstelling hiermee, het die resultate van die Spearman rangorde korrelasiekoëffisiënt

en Pearson korrelasiekoëffisiënte, gepaarde t-toetse, Bland-Altman tegniek en kwartielverspreiding matig tot swak korrelasie vertoon vir vergelykende geldigheid tussen die twee instrumente. 'n Relatief lae reikwydte vir die Spearman rangorde korrelasie (-0.09 tot 0.42) en die Pearson korrelasiekoëffisiënt (0.008 tot 0.0556) is gerapporteer. Betekenisvolle proporsionele sydigheid was teenwoordig vir agt nutriënte met die gebruik van die Bland-Altman tegniek en die proporsie van individue wat in dieselfde of aangrensende kwartiele geklassifiseer is, was relatief hoog.

Daarna het die hoofstudie gevolg waarin die sosio-demografiese en antropometriese profiel, dieet, fisiese aktiwiteit, bloeddruk, vastende bloedglukose, trigliseriede en totale cholesterol van die respondente ondersoek is.

Ten einde die voorkoms van risikomerkers te bepaal, is Asiatiese afsnypunte gebruik. Ongeveer 92% van die respondente het diastoliese bloeddrukvlakke bo 85 mmHG vertoon, 88.8% het trigliseriedvlakke bo 1.69 mmol/L gehad, en 38.8% het vastende bloedglukosevlakke bo 5.55 mmol/L geregistreer. Wanneer die afsnypunte gebruik is vir sentrale vetsug soos gedefineer deur middelomtrek vir mans by 90 cm of meer en vroue by 80 cm of meer, is 100% van die vroue en 87.4% van die mans as sentraalvetsugtig geklassifiseer. In terme van fisiese aktiwiteit is 62.5% respondente as onaktief (<600 METS minute) geklassifiseer. Wanneer algoritmes soos die Europese *SCORE* en 'n gewysigde verdubbelde telling egter gebruik is, het respondente minimale risiko vir kardiovaskulêre siektes getoon.

Die gerapporteerde innames van vrugte en groente was ver onder die aanbevole WGO populasie nutriëntdoelwitte (gelyk aan of meer as 400 g) met mans wat 'n gemiddelde daaglikse inname van 221.9 g gerapporteer het en vroue 236.9 g. Verder het 94% van die respondente minder as die geskatte gemiddelde vereiste vir vesel ingeneem.

Dieetindekse, gebruik om dieetkwaliteit vas te stel, het goeie dieetkwaliteit getoon. Twee hoofkomponente is ge-identifiseer, gebaseer op die persentasie vet deur elke voedselgroep bygedra: Faktor 1 (Peulgroente, grane en graanprodukte, groente) wat bygevoegde vet verteenwoordig het en Faktor 2 (Vette en olies, suiker en soetigheid en die melkgroep) wat sigbare vet verteenwoordig het. Volgens regressie-analises was

slegs bloedglukose betekenisvol omgekeerd verwant aan beide faktortellings in die hele studiegroep.

Die volgende verbande tussen nutriëntinname en kliniese risikomerkers is opgemerk: daar was 'n aantal betekenisvolle korrelasies van risikotelling (die somtotaal van alle risikomerkers gedefinieer deur Asiatiese standaarde) en risikotelling 1 (die somtotaal van alle risikomerkers met vastende bloedglukose verdubbel in vergelyking met die ander risikomerkers) met nutriëntinname, met die sterkste korrelasie met persentasie energie vanaf vet. Verder het middelomtrek betekenisvolle assosiasies met die meeste kliniese risikomerkers vertoon. Dit het met sistoliese bloeddruk, vastende bloedglukose, cholesterol, trigliseriede en liggaamsmassa-indeks gekorreleer met liggaamsmassa-indeks die sterkste van al die kliniese risikomerkers. Risikotelling het die sterkste statisties betekenisvolle korrelasies met kliniese merkers vir die hele studiegroep getoon.

Die gesamentlike voorkoms van risikomerkers en die waargenome assosiasies van risikomerkers met dieet en kliniese veranderlikes noodsaak dieetwysiging deur verhoogde inname van groente en vrugte, gewigsverlies en verhoogde fisiese aktiwiteit vir 'n volhoubare intervensiestrategie om die las van nie-aansteeklike siektetoestande in die Indiërbevolking van Suid-Afrika te verminder en beheer.

ACKNOWLEDGEMENTS

The successful completion of this research thesis would not have been possible without the contribution and support of various people. I wish to express my sincere gratitude to a number of special people:

1. My promoter, Prof. C. S. Venter, for her expertise, guidance, wisdom and constant encouragement throughout the project. It was indeed a privilege and an honour to be mentored by an icon in South African nutrition;

2. Prof. Una MacIntyre, my co-promoter, for all her guidance, support and valuable input through her dietary expertise and her dynamic foresight, constantly *en*couraging me to try innovative nutrition research practices;

3. Dr Suria Ellis for her assistance and support with the statistical analysis;

4. Ms Mary Hoffman for the language editing of the thesis;

5. Dr Grieta Hanekom for her expertise in technical editing of the thesis;

6. My family: my husband, Daya for his support and, in particular, my children, Grishka and Kaiueran, for their unending patience and understanding during trying times;

7. Finally, I want to dedicate this research to my late dad. Your unstinting support, encouragement, wisdom and your silent fight against NCDs wove a nostalgic and personal thread through all my endeavours.

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AA	arachidonic acid
ACSM	American College of Sport Medicine
ADA	American Dietetic Association
AHA	American Heart Association
AI	Adequate intake
ALA	α-linolenic acid
AMDR	acceptable macronutrient distribution range
BMI	body mass index
BP	blood pressure
BTT	Birth-to-Ten
CAD	coronary artery disease
CHAD	Community Syndrome of Hypertension, Atherosclerosis and Diabetes
CHD	coronary heart disease
CHETNA	Children's Health Education Through Nutrition and Health Awareness programme
CHO	Carbohydrates
COPD	chronic obstructive pulmonary disease
CRA	comparative risk assessment
CRP	C-reactive protein
CV	Coefficient of variance
CVD	cardiovascular disease
DAEK	Dietary Assessment Education Kit
DALYs	disability adjusted life years
DBP	diastolic blood pressure
DCPP	Disease Control Priorities in Developing Countries
DHA	docosahexaenoic acid
E	Energy
EAR	Estimated average requirement
EER	Estimated energy requirements
EI	Energy intake
EPA	eicosapentaenoic acid
ESC	European Hypertension Society
FAO	Food and Agriculture Organisation
FDA	Food and Drug Association
FFA	free fatty acids
FFQs	Food frequency questionnaires
FNB	Food and Nutrition Board
GPAQ	Global Physical Activity Questionnaire
HDL	high-density lipoprotein
HIV/AIDS	human immunodeficiency virus/acquired immunodeficiency syndrome
HMR	Home Meal Replacements
IDF	International Diabetes Federation
IGT	impaired glucose tolerance
IHD	ischaemic heart disease
IPAQ	International Physical Activity Questionnaire
kJ	Kilojoule
KZN	KwaZulu-Natal
LA	linoleic acid
LDL	low-density lipoprotein

MARG	Medical Education for Children/ Adolescents for Realistic Prevention of Obesity and Diabetes and for Healthy Aging
MetS	metabolic syndrome
METs	Metabolic equivalents
MI	Myocardial infarction
MRC	Medical Research Council
MUFAs	monounsaturated fatty acids
n-3	omega-3
n-6	omega-6
NCD	Noncommunicable disease
NCEP/ATP	National Cholesterol Education Program Adult Treatment Panel
P:S	ratio of dietary polyunsaturated to saturated fatty acids
PAI-1	plasminogen activator inhibitor-1
PAL	physical activity level
PHC	Primary health clinic
PUFAs	polyunsaturated fatty acids
PURE	Prospective Urban and Rural Epidemiological
QFFQ	Quantitative food frequency questionnaire
RDA	Recommended Dietary Allowance
SACRA	South Africa Comparative Risk Factor Assessment
SADHS	South African Demographic and Health Survey
SAH	South African Hypertension Guidelines
SANBD	South African National Burden of Disease study
SASSI	Sustainable Seafood Initiatives
SBP	systolic blood pressure
SCORE	Systemic Coronary Risk Evaluation
SFA	saturated fatty acid
SSA	sub-Saharan Africa
STDs	sexually transmitted diseases
THUSA	Trans-disciplinary Health during Urbanisation in South Africa
TTM	Trans Theoretical Model
UK	United Kingdom
WC	waist circumference
WHO	World Health Organization
WHOCRA	World Health Organization Comparative Risk Factor Assessment
WHR	waist-hip ratio
WSR	waist-to-stature ratio

CHAPTER 1

INTRODUCTION

1.1 BACKGROUND TO THE PROBLEM: GLOBAL PERSPECTIVE

Introduction

Noncommunicable diseases (NCDs), also known as chronic diseases of lifestyle, are a group of diseases that share similar risk factors as a result of exposure over many decades to unhealthy diets, smoking, lack of regular exercise and, possibly, stress (Medical Research Council, MRC, 2006). The major risk factors are high blood pressure, high blood cholesterol, tobacco addiction, diabetes and obesity. These result in various long-term disease processes, culminating in high mortality rates attributable to strokes, heart attacks, tobacco and nutrition-induced cancers, chronic bronchitis, emphysema, renal failure and many others (MRC, 2006).

Global picture

Globally, NCDs have rapidly increased. They represent the major health burden in industrialised countries and are rapidly increasing in developing countries. In 1999, these diseases contributed to approximately 60% of deaths in the world and 47% of the global burden of disease (World Health Organization, 2002b). It is predicted that, globally, death from NCDs will increase by 77% between 1990 and 2020, and most of these deaths will occur in the developing regions of the world (Murray & Lopez, 1996). Progressively, in 2002, 28.2 million global deaths (58.6%) were attributable to NCDs, with the predicted mortality rate for 2020 being 49.6 million (72.6% of all deaths) (Vorster & Gibney, 2009).

In addition, the Global Burden of Diseases Study of the World Health Organization (WHO), the World Bank and the Harvard University showed that in the calculations of disability-adjusted life years (DALYs), the global pattern of NCDs was 40.9% for all diseases in 1990 and is projected to be 59.7% by 2020 (Murray & Lopez, 1996). Developing countries often experience a double burden with a combination of poverty-related diseases and NCDs. At the fifty-fifth World Health Assembly, the World Health

Director-General stated that NCDs are well known to wealthy societies, dominating in the middle- and upper-income countries. However, what is notable is that they are becoming more prevalent in developing countries where, together with infectious diseases, they create a double burden (WHO, 2002a). Furthermore, there are striking ethnic differences in cardiovascular disease (CVD) risk. Migrants of South Asian descent (encompassing people from India, Sri Lanka, Bangladesh, Nepal and Pakistan) worldwide have elevated risks of morbid and mortal events because of ischaemic heart disease (IHD) (McKeigue *et al.*, 1989; Lip *et al.*, 2007; Isharwal *et al.*, 2009). Adoption of a westernised lifestyle has different effects on metabolic and vascular dysfunction across populations and, according to Forouhi and Sattar (2006), ethnic groups show differences in levels of visceral adiposity, insulin resistance and novel cardiovascular risk markers such as C-reactive protein (CRP), adiponectin, haemostatic factors and plasma homocysteine. They are also of the opinion that marked differences in disease risk across racial and ethnic groups are probably due in part to genetic factors, host susceptibility and environmental factors, and can provide valuable aetiological clues in patterns of disease presentation, therapeutic needs and response to treatment. Therefore, Forouhi and Sattar (2006) recommend ongoing studies to increase understanding of ethnicity as a potential independent risk factor, thus enabling better identification of treatment targets and selection of therapy in specific populations.

Morbidity and mortality

It is generally accepted that the increase in morbidity and mortality from chronic disease is a result of changes in lifestyle due to industrialisation, urbanisation, economic development and increased food market globalisation, which leads to increased levels of smoking and high fat, high animal protein and low fibre intakes. Unhealthy diets and physical inactivity are among the leading causes of the major NCDs, including CVDs, type 2 diabetes and certain types of cancer (WHO, 2003b). The rapidly increasing prevalence of insulin resistance, the metabolic syndrome (MetS), dyslipidaemia and type 2 diabetes in South Asians has largely been linked to rapid changes in lifestyle and dietary patterns (Misra *et al.*, 2007). These conditions not only cause human suffering, they also threaten the economies of many countries as they impact on the older and experienced members of the workforce [in South Asians, particularly young men (Forouhi & Sattar, 2006)]. Furthermore, the complex disease profile of NCDs lays a huge burden on governments as the management of NCDs is highly costly (Uusitalo *et*

al., 2002*).* Owing to this evolution, the WHO in 2004 adopted a resolution on the prevention and control of NCDs. The key strategy of prevention is targeting the major risk factors, i.e. unhealthy diets and physical inactivity, in an integrated manner. Consequently, the WHO concluded that a unique opportunity exists to formulate and implement an effective strategy for substantially reducing death and disease worldwide by improving diet and promoting physical activity (WHO, 2004a).

MetS

People with MetS are at increased risk for CVD and mortality from CVD (Trevisan *et al.,* 1998). The MetS – defined as a combination of three or more risk factors for CVD, including central obesity, hypertriglyceridaemia, high blood pressure (BP), high fasting glucose and decreased high-density lipoprotein (HDL)-cholesterol – is common in Asian Indians (Das, 2003; Ramachandran *et al.,* 2003; Dwivedi *et al.,* 2004). The prevalence of MetS has been estimated to be between 28.8% and 41.1% in Asian Indian adults when using the National Cholesterol Education Program Adult Treatment Panel (NCEP/ATPIII) criteria with modified waist circumference (WC) criteria appropriate for Asian Indians [≥80 cm in women and ≥90 cm in men (Tan *et al.*, 2004)]. Important reasons for the apparent trend in MetS could be their excess body fat and adverse body fat patterning, including abdorninal adiposity, even when the body mass index (BMI) is within the currently defined normal limits (Zhu *et al.*, 2002; Kanjilal *et al.*, 2008). Underlying genetic tendency and lifestyle factors or early-life adverse events may contribute to this phenotype, but lifestyle factors alone or modulated by inherited factors appear to play an important role because obesity and dyslipidaemia become worse with urbanisation and migration (Misra & Vikram, 2004).

Behavioural intervention

The benefits of behavioural intervention in reducing NCDs in populations have been well proven in countries such as Finland, Japan, Singapore (WHO, 2002a) and South Korea (Lee *et al.,* 2002). Moreover, the experiences and results of the North Karelia project carried out in Eastern Finland support the idea that a well planned, community-based programme can have a major impact on lifestyle and risk factors (Puskha, 2002). With regard to South Korea, the government went to great lengths to advertise and teach the public that the traditional Korean diet is healthy and that the adoption of Western eating habits may have unfavourable effects (Lee *et al.,* 2002). Programmes

recently initiated in India to create awareness and prevent childhood obesity include CHETNA (Hindi for "The Awareness") (Children's Health Education Through Nutrition and Health Awareness programme) and MARG (Hindi for "The Patho") (Medical Education for Children/ Adolescents for Realistic Prevention of Obesity and Diabetes and for Healthy Aging) (Bhardwaj *et al.*, 2008). Several dietary changes are advised through lectures and printed leaflets, and through involvement of students in debates, skits and cookery contests. This large-scale programme aims, for the first time in South Asia, to reach hundreds of thousands of children in many cities of North India.

1.2 BACKGROUND TO THE PROBLEM: THE SOUTH AFRICAN SITUATION

South Africa, like many other developing countries, is undergoing an epidemiological transition. According to the first South African National Burden of Disease (SANBD) study, South Africa is experiencing a quadruple burden of disease, comprising pre-transitional diseases, the emerging chronic diseases, injuries and human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS) (Bradshaw *et al.,* 2003). Estimates for South Africa show that, despite the high burden of infectious diseases, NCDs accounted for 37% of deaths in the year 2000, CVD and diabetes together accounted for 19% of total deaths, and cancers accounted for a further 7.5% (Steyn *et al.,* 2006). Many of the deaths occurred in the middle-aged members of the workforce (MRC, 1999). Statistics on the prevalence of NCDs in South Africa in 1999 revealed that 6 million people suffered from hypertension, 4 million had diabetes, 7 million had suffered stroke and 4 million had hyperlipidaemia (MRC, 1999). In addition to this, about 56% of the population had at least one of these risk factors and about 20% was at a high risk of developing NCDs (MRC, 1999).

The level of mortality is one of the key indicators of the well-being and health status of a population. Estimates of age-standardised mortality rates for South Africans in 2000 show that all-cause mortality rates differ substantially between population groups (Steyn *et al.*, 2006). Death rates from CVD and diabetes were extremely high for Indians (607 and 111 per 100 000 respectively, compared with 361 and 49 per 100 000 for the total South African population). The death rates from NCDs accounted for 28.5% of the reported deaths of South Africans in the 35-64 year age group in 1998; however, with

HIV/AIDS as a competing cause, statistics on the proportion of reported deaths showed that 20% of deaths in 2000 in the 35-64 year age group were as a result of chronic diseases of lifestyle (Stats SA, 2005). The results regarding the leading causes of death based on age in 2000, showed that in young adults aged 15-45 years, HIV/AIDS, tuberculosis, homicide and road traffic accidents were the most common causes. Comparatively, in the age group over 45 years, CVD and lifestyle-related diseases featured amongst the leading causes of death (Bradshaw *et al.*, 2003). A statistical release in 2006 on the mortality and causes of deaths based on all death notifications showed that in terms of age differentials, the percentage of deaths in 2006 was 9.8% in the 30-34 year age group, 9.1% in the 35-39 year age group, 8.2% in the 40-44 year age group, 7.5% in the 45-49 year age group and 6.3% in the 50-54 year age group (Stats SA, 2006). This release also showed that diseases of the circulatory system accounted for 13.7% of all deaths and ranked as the third leading cause of death in South Africa in 2006 (Stats SA, 2006). The highest death rates from blood vessel disease in South Africa were found in Indian people, followed by coloureds, while whites and blacks had the lowest rates (Norman *et al.,* 2006). In a study conducted by Ranjith *et al.* (2005), significant differences in risk factor status were found between gender and age groups in South African Indians. The findings of the study showed that young South African Indians frequently had premature atherosclerosis with diffuse and aggressive disease (Ranjith *et al.,* 2005). Swart *et al.* (2005) support the findings of Ranjith *et al.* (2005) and are of the view that it is vital that these differentials be factored in for effective interventions.

1.3 BACKGROUND TO THE PROBLEM: THE SOUTH AFRICAN INDIAN SITUATION

The Indians in South Africa are an ethnic minority population group, originating from the Indian sub-continent as migrant labourers. With social transformation and equity, their lifestyles have changed dramatically. At the time of the 2001 census, the total population of Indians was 1 115 467, of whom the majority, totalling 798 275, resided in KwaZulu-Natal (KZN) (Stats SA, 2001). From the literature review hitherto, it is evident that few statistics exist on their health and nutrition profile and that the available published data on their dietary practices are limited. Indian migrants throughout the world have been reported to have a higher prevalence of diabetes, coronary heart

disease (CHD, defined as coronary artery disease with myocardial involvement) and dyslipidaemia, in comparison with their native counterparts (McKeique *et al.* 1989; Lip *et al.* 2007), and similarly, in South Africa these conditions have reached epidemic proportions (Seedat *et al.,* 1990; Seedat, 2009). Seedat *et al.* (1990) identified the most important risk factors among Indians in the Durban area, aged 15 to 69 years. These were: hypercholesterolaemia, hypertriglyceridaemia, diabetes and smoking among men, and diabetes, hypercholesterolaemia and hypertriglyceridaemia in women. Ferris *et al.* (2005) found that Asian-Indian South Africans were more insulin-resistant than BMI-matched whites. These results correspond to other cross-sectional data which also suggest that insulin resistance could account for much of the enhanced IHD risk in Asian Indians (Chaturvedi, 2003; Isharwal *et al.*, 2009).

As mentioned above, unhealthy diets and physical inactivity are among the leading causes of the major NCDs. The key strategy in the prevention of these diseases is the targeting of the major risk factors, i.e. unhealthy diets and physical inactivity, in an integrated manner (WHO, 2004a). These risk factors in South Asians (of Indian, Pakistani and Bangladeshi descent) in the United Kingdom have been studied by various researchers (McKeigue *et al.*, 1989; Sevak *et al.*, 1994; Farooqi *et al.*, 2000; Forouhi *et al.*, 2001; Sevak *et al.*, 2004; Forouhi & Sattar, 2006; Lovegrove, 2009; Gilbert & Khokhar, 2008; Bohpal & Rafnsoon, 2009). However, limited data are available on the activity and dietary patterns of South African Indians. The 2003 South African Demographic and Health Survey (SADHS) provided nationally representative data on physical activity of adults, which revealed that KwaZulu-Natal has the highest levels of inactivity for both men and women (66% and 81% respectively) (Department of Health, Medical Research Council & OrcMacro, 2007). With reference to the Indian population, it was found that 43.1% of men and 62.6% of women were inactive.

Dietary intakes of Indians were studied by Wolmarans *et al.* (1999) and Mia and Vorster (2000). Wolmarans *et al.* (1999) concluded that the ratio of dietary polyunsaturated to saturated fatty acids (P:S) in the diets of Indians in Durban was high (a finding which has been confirmed in Indian adolescents in Lenasia, Gauteng by Mia and Vorster in 2000) and that such a high P:S ratio might affect the oxidation of low-density lipoprotein, and therefore be a risk factor for CHD. A significantly higher intake of polyunsaturated fatty acids (PUFAs) – mainly as omega-6 (n-6) PUFAs from vegetable oils – was also

reported in Asian Indians in the UK, with a significantly lower intake of the cardioprotective omega-3 (n-3) long-chain PUFAs, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), than in whites (McKeigue *et al.*, 1985; Lesauvage *et al.*, 2001). Consistent with the dietary findings, studies reported a higher proportion of n-6 PUFAs linoleic acid (LA) and arachidonic acid (AA) and a lower proportion of n-3 long-chain PUFAs in plasma and membrane phospholipids in Asian Indians than in Caucasians (Das *et al.*, 1994; Lovegrove *et al.*, 2004). A high n-6 PUFA to n-3 PUFA ratio of the skeletal muscle membrane has been shown to affect insulin sensitivity adversely (Storlien *et al.*, 1996), which supports the hypothesis that a high n-6:n-3 dietary PUFA may contribute to the high prevalence of insulin resistance in this ethnic group (Brady *et al.*, 2004). Although CHD and type 2 diabetes are common amongst South African Indians, the n-3 fatty acid content of their diet has been investigated only in adolescents in Lenasia (Mia & Vorster, 2000). Therefore, there is a need for an in-depth investigation into the prevalence of NCDs and the diet and activity patterns of adult Indians in South Africa.

1.4 AIM OF THE STUDY

The aim of this cross-sectional, epidemiological, descriptive study was to conduct a survey of the prevalence of risk markers for NCDs, and of the associations of dietary and lifestyle exposures with the risk of NCDs, among apparently healthy adult Indians in KZN, and to make realistic recommendations for a sustainable intervention strategy.

1.5 OBJECTIVES

- To determine the reproducibility and comparative validity of a culturally modified quantitative food frequency questionnaire.

- To determine the prevalence of risk markers for NCDs (hypertension, increased fasting blood glucose, cholesterol and triglyceride levels, central obesity and physical inactivity).

- To establish whether an association exists between current trends in nutrient intakes and food patterns, with special reference to the n-6:n-3 fatty acid ratio and the prevalence of risk markers for NCDs.

- To establish whether an association exists between the current trends in physical activity and the prevalence of risk markers for NCDs.

- To suggest strategies for an integrated programme of prevention of NCDs in the target group.

1.6 DELIMITATIONS

The nutrient analyses of the diets of the respondents in this study were based on the available South African food composition tables (Langenhoven *et al.*, 1991). With regard to certain indigenous foods that were missing from the South African food composition tables, analyses were exported from the Indian food composition tables (Gopalan *et al.,* 2004*).* Further to this, suitable substitutes were used for foods that had no analysis in either the South African or the Indian food composition tables, e.g. variants of green leafy vegetables which grow wild in the veld and are prepared as a sautéed herb dish. Furthermore, owing to lack of infrastructure and human resources (the researcher and a nursing sister were responsible for all measurements), only finger-prick measurements of fasting glucose and total serum cholesterol and triglycerides were taken, apart from anthropometry, blood pressure measurements and questionnaires on dietary intakes, activity, demographics and medical history.

1.7 DEFINITION OF TERMS

The following definitions apply to terms used in the context of this thesis:

1.7.1 Noncommunicable diseases (chronic diseases of lifestyle)

NCDs develop over time in genetically susceptible individuals because of exposure to interrelated societal, behavioural and biological risk factors, where the risk factors

accumulate through the life course and manifest after decades of exposure (Vorster & Gibney, 2009). They are also referred to as chronic diseases; however, infectious diseases such as HIV/AIDS and tuberculosis are also chronic. These diseases are also called diseases of affluence, but this is a misnomer because they are also common in lower socio-economic groups. Chronic diseases of lifestyle include primarily CVD, neoplasms, diabetes and respiratory diseases (MRC, 2006). However, this thesis will focus on CVD and diabetes because of the extremely high death rate from these diseases among Indians and the established association between these diseases and lifestyle factors in various populations.

1.7.2 Epidemiological transition

Epidemiological transition refers to the complex changes in patterns of health, disease and mortality that result from demographic and associated economic and societal changes in a world population that is getting older (Drewnowski & Popkin, 1997; Shetty, 1997).

1.7.3 Nutrition transition

Nutrition transition is described as the gradual change over centuries from a high-fibre diet of low energy density to an energy-dense, low-micronutrient dietary pattern (Vorster & Gibney, 2009).

1.7.4 Risk factors and risk markers

Yusuf *et al.* (2001b) illustrated the link between risk markers and CVD, where they are classified into two categories, i.e. those that have been proved to be causal (risk factors) and those that show associations with CVD but for which a cause and effect association has yet to be proved (risk markers). These risk markers can be classified as predisposing (e.g. obesity, which raises BP, glucose and lipids) or direct (e.g. smoking). According to Stamler (2008), an established major risk factor is a trait common in the population, amenable to prevention and control, and with a significant, graded, strong, independent relation to one or more other established major risk factors and/or to CHD/CVD incidence and mortality. Based on critical assessment by independent expert

groups, an established major risk factor is a risk factor judged to be causatively related to CHD/CVD. Six established major risk factors have been identified: adverse diet pattern, a diet related to adverse levels of serum total cholesterol (and its fractions), high BP, high plasma glucose (diabetes), unhealthy BMI and smoking. Because adverse diet patterns play a role in unfavourably influencing four other established major risk factors (the so-called metabolic traits), and, in addition, directly and independently relates to CHD/CVD risk, it is the pivotal cause of epidemic CHD/CVD (Stamler, 2008). Most of the variables tested in this study meet the criteria for risk factors as defined by Stamler (2008) and Mahan and Escott-Stump (2008). However, to be technically consistent, the term *risk markers* will be used throughout the thesis.

1.7.5 South Asians

In this thesis, the term *South Asians* refers to individuals originating from India, Pakistan, Bangladesh, Nepal and Sri Lanka in the Indian subcontinent (Misra *et al.*, 2008). Other terms used, such as *Asian Indians*, *Indo-Asians*, *Indian Asians* and *Indo-Sikhs*, are reproduced as in the original studies and broadly refer to Indians from India and neighbouring countries.

1.8 IMPORTANCE OF THE STUDY

Indian migrants throughout the world have been reported to have a higher prevalence of diabetes, CHD and dyslipidaemia compared with their native populations (Ranjith *et al.*, 2005) and the situation is no different in South Africa, where these diseases have reached epidemic proportions (Seedat, 1996). However, little is known about the relationship between diet and lifestyle exposures and health outcomes amongst Indians in South Africa. It has been suggested that the altered dietary habits of the migrant Indians, compared with the average diet in India, could possibly contribute to the migrants' increased risk of CHD (Misra & Vikram, 2004). The focus of this research is a long-known existing problem, namely CHD. The associations and interactions between blood parameters and dietary, activity and anthropometric data in men and women were determined and, based on these results, intervention strategies were proposed to contribute to relieving the problem amongst Indians in KZN. The main aim of the study

was to gather enough relevant information to suggest appropriate intervention actions and programmes to reverse the underlying causes of NCDs amongst Indians. The results of the study may help to elucidate the role of diet in the development of obesity, hypertension and CHD among Indian men and women in KZN. Consequently, the findings of this study can be used to generate practical dietary recommendations and health intervention strategies to improve the quality of life of the Indians in South Africa whilst simultaneously reducing the health care cost generated by chronic diseases.

1.9 ORGANISATION OF THE REPORT

Chapter 1 reviews the background of the study in terms of the global perspectives of NCDs. It then reviews the South African situation and, lastly, focuses on the South African Indian situation in terms of the prevalence of NCDs. The purpose and the importance of this study are also justified in this chapter.

- Following the introductory chapter, the relevant literature is reviewed in Chapter 2 in terms of the risk factors, risk behaviours and disease states of NCDs. Existing recommendations for interventions are also reviewed in Chapter 2.

- The research method, data management and analysis design are described in Chapter 3. The habitual diet of the respondents was measured by administration of a quantitative food frequency questionnaire, developed and validated by MacIntyre *et al.* (2001a, b, c) in another study and modified and validated for this study to accommodate Indian food habits.

- The results of the validation and reproducibility study are presented in Chapter 4 and those of the prevalence and associations of risk markers in Chapter 5.

- The results are interpreted and discussed in Chapter 6.

- Conclusions drawn and recommendations based on observations made are presented in Chapter 7.

CHAPTER 2

LITERATURE REVIEW

2.1 INTRODUCTION

The health status and disease profile of human societies have been historically linked to the level of economic development and social organisation. With industrialisation and urbanisation, the major causes of death and disability have shifted from a predominance of nutritional deficiencies and infectious diseases to degenerative diseases. These paradoxical disease patterns have been termed the *epidemiological transition*, where emerging NCDs feature significantly and coexist with undernutrition (Omran, 1971).

The rising trend of NCDs is a consequence of the demographic and dietary transition and the globalisation of the economic process. Scientific evidence shows that unhealthy diets, physical inactivity and tobacco use are key global determinants of NCDs. Throughout human history, populations have experienced changes in their ecological relationship that have modified their diet and ultimately altered their disease patterns (WHO, 2002a). Figure 2.1 illustrates the consequences of the nutrition transition and the different health patterns associated with the various stages of transition (Vorster *et al.,* 1999). It is important to realise that the nutrition transition is characterised by a stepwise progression that depends on the balance of contributing factors. For example, whereas urbanisation in more industrialised countries has been associated with economic growth, in many developing countries it results in urban poverty.

Over the years, a huge body of literature has been amassed on chronic disease and the interrelationship between risk behaviours, risk markers and disease state. Figure 2.2 clearly depicts this interrelationship between these variables that coexist with each other (MRC, 2008). It is evident from this interrelationship that any intervention has to be based on a holistic overview of the risk behaviours, risk markers and disease state.

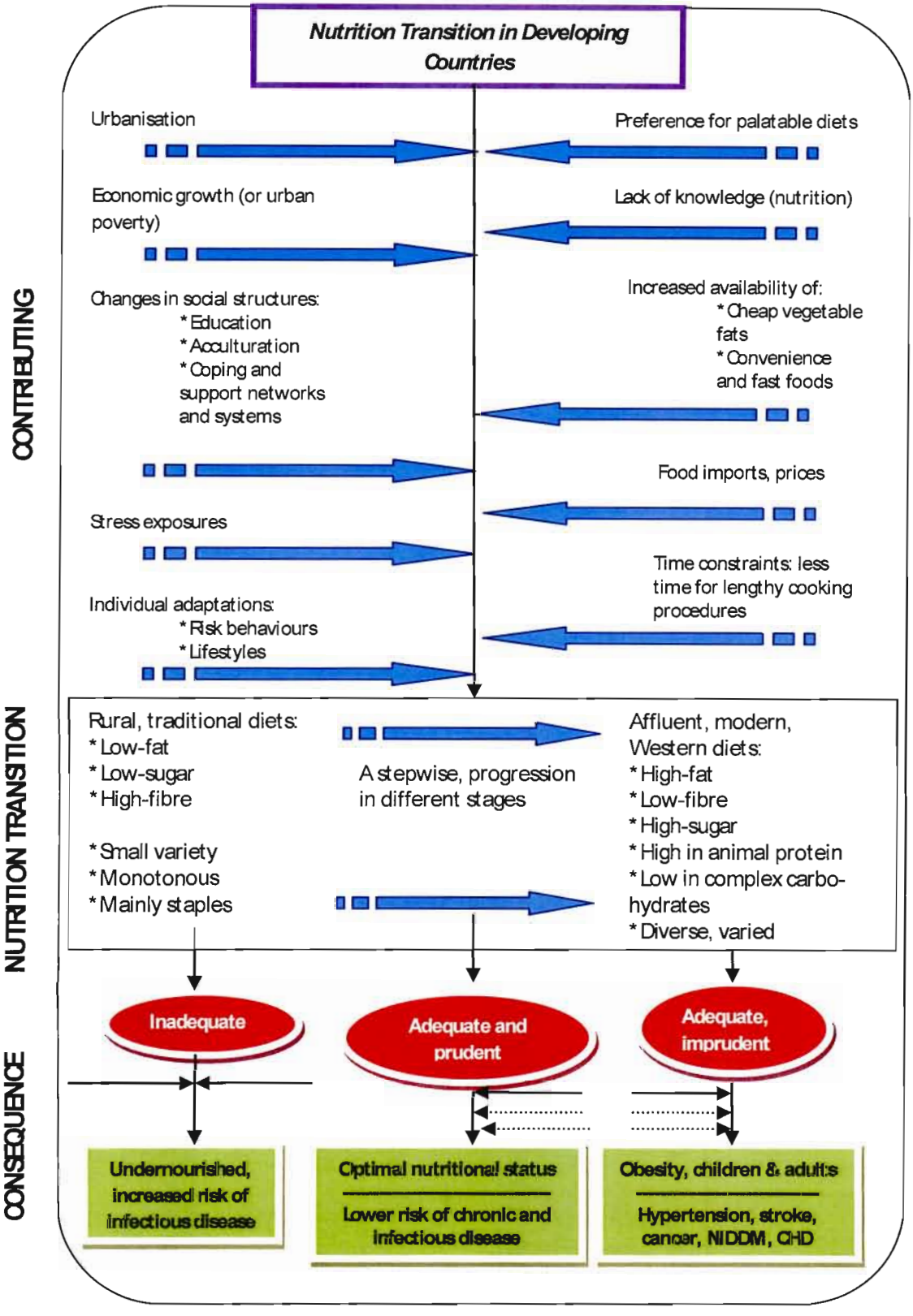

Figure 2.1: Schematic diagram of the contributing factors to and consequences of the nutrition transition (Adapted from Vorster *et al.*, 1999)

NIDDM = Type 2 diabetes, CHD = coronary heart disease

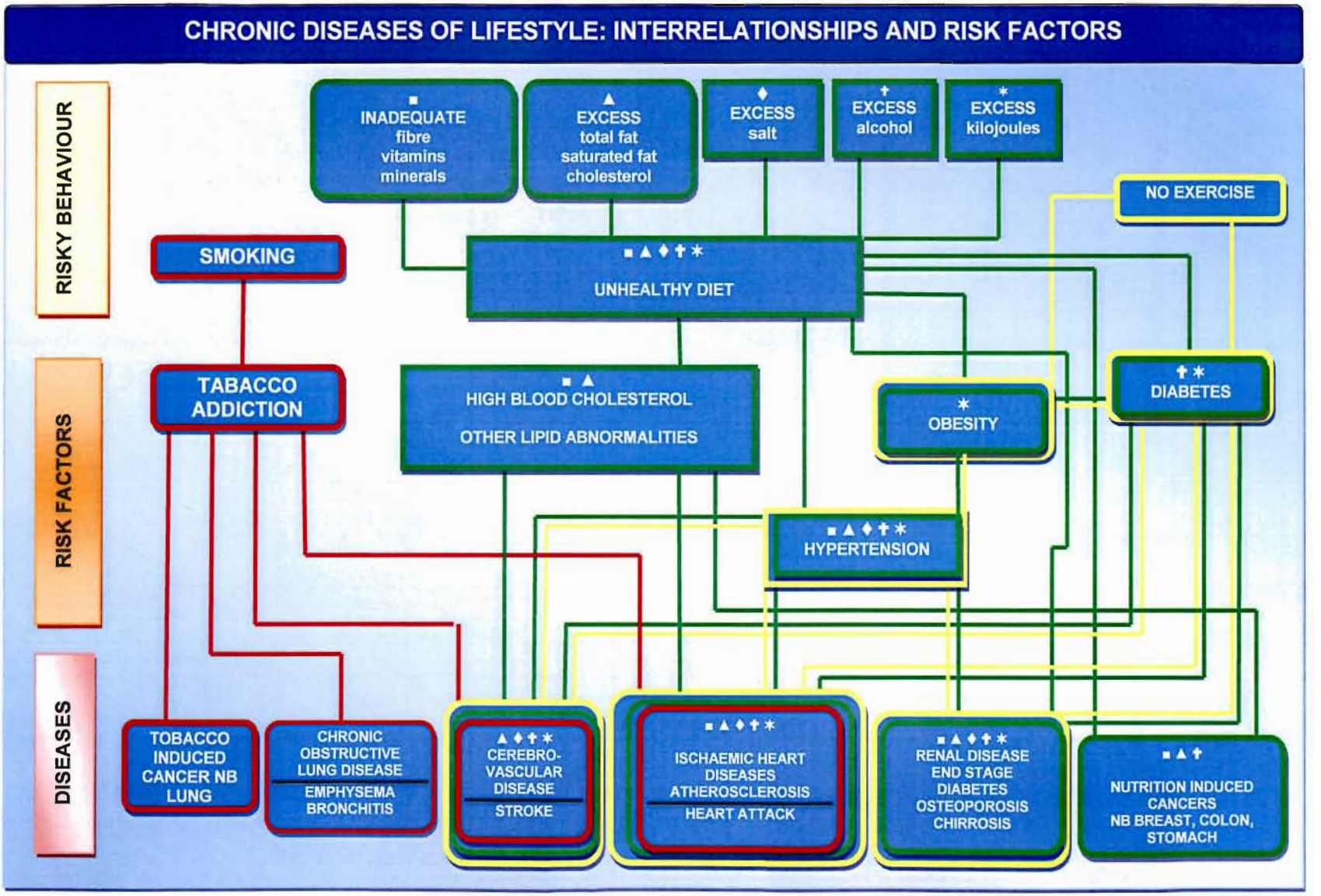

Figure 2.2: Chronic diseases of lifestyle: interrelationships and risk factors (MRC Diseases of Lifestyle Unit, 2008)

Vorster and Gibney (2009) go further to illustrate the interrelationship of risk markers with the prevalence of NCDs by showing how a chain of events is formed, starting with societal factors such as socio-economic status and environmental factors that influence behaviours, leading to biological risk factors which in turn lead to the development of the NCDs. It is important to note the clustering of risk markers, which has become a well described phenomenon. Obesity, for example is associated with insulin resistance, hyperlipidaemia and hypertension, all of which contribute to the development of CHD and diabetes (Vorster & Gibney, 2009). This phenomenon of clustering represents an opportunity to address more than one risk at a time (WHO, 2003b). Table 2.1 succinctly illustrates the interrelationship of factors that underpin NCDs.

Table 2.1: Risk factors for noncommunicable diseases (NCDs) (Vorster & Gibney, 2009).

Societal	Behavioural	Biological	NCDs
Socio-economic status	Smoking	Tobacco addiction	Lung disease
Cultural habits	Alcohol abuse	Alcohol addiction	Cardiovascular disease
Environmental factors	Lack of physical activity	Dyslipidaemia	Atherosclerosis
	Inappropriate diets:	Hyperlipidaemia	Cerebrovascular
	Inadequate	Insulin resistance	disease
	• fibre	Hypertension	Stroke
	• micronutrients	Obesity (body	Ischaemic heart disease
	Excess	composition)	Myocardial infarction
	• total fat		Diabetes
	• saturated fat		Dental caries
	• trans fat		Osteoporosis
	• cholesterol		Cirrhosis
	• salt (NaCl)		Diet-induced cancers
	• energy		

Epidemiological studies have clearly demonstrated a link between risk markers and CVD. Yusuf *et al.* (2001a) illustrated the link between risk markers and CVD, when they are classified into two categories, i.e. those that have been proved to be causal (risk factors) and those that show associations with CVD but for which a cause and effect association has yet to be proved (risk markers). These risk markers could be classified as predisposing to, e.g. obesity, which raises BP, glucose and lipids. The proven and

putative risk markers for CVD are recognised as follows: risk markers that are causally linked, i.e. tobacco consumption, elevated low-density lipoprotein (LDL) cholesterol, HDL cholesterol, hypertension, elevated glucose, physical inactivity, obesity and diet. Moreover, the following risk markers that show associations were identified by Yusuf *et al.* (2001a): low socio-economic status, elevated prothrombotic factors, fibrinogen, plasminogen activator inhibitor-1 (PAI-1), markers of infection and inflammation, elevated homocysteine, elevated lipoprotein (a) and psychological factors.

The burden of NCDs has cost implications, yet it is an area where most positive changes can be instituted successfully. The direct costs of NCDs come in the form of health care provision and the indirect costs involve impaired quality of life and premature death and disability (WHO/FAO, 2003b). The main goal of public health policy is to give people an opportunity to enjoy many years of healthy and active life. Sadly, however, agendas on NCDs are often left aside in the policy planning of less industrialised countries, as undernutrition and communicable diseases demand a great deal of attention (WHO, 2002a).

2.2 NONCOMMUNICABLE DISEASES IN SOUTH AFRICA

South Africa has a heterogeneous population of approximately 49 million inhabitants, comprised of 79.6% Africans, 9.1% whites, 9.0% coloureds and 2.5% Indians (Statistics South Africa, 2009). Because of the heterogeneity of the South African population, it is important to differentiate between the terms *racial group* and *ethnic group*. A racial group implies having a common gene; however, an ethnic group implies sharing a cultural identity, i.e. biological similarity plus shared social and lifestyle values (Seedat, 2007). As South Africa undergoes transformation, stark contrast in living conditions ranging from wealthy to underdeveloped rural areas is a characteristic phenomenon. The changing social, political and economic climate has resulted in urbanisation, which in turn has impacted on dietary and lifestyle patterns (Steyn *et al.*, 2006).

In order to determine the prevalence of NCDs in South Africa, it is essential that the disease profile of South Africans in terms of morbidity and mortality be reviewed. The South African Health Review (Health Systems Trust, 2006) found a steady decline in the health of the South African population in recent years. HIV/AIDS accounted for 39%

of all deaths in 2000 and diseases of lifestyle accounted for 38% of all deaths (Health Systems Trust, 2006). In the year 2000, the South African National Burden of Disease Study (SANBD) identified causes of premature mortality and morbidity experienced in South Africa. The findings of this study showed that NCDs accounted for 37% of deaths, of which CVD and diabetes were responsible for 19% of deaths (Bradshaw *et al.,* 2003). Stemming from the SANBD, the South Africa Comparative Risk Factor Assessment (SACRA) followed, with the primary aim of estimating DALYs and identifying risk factors based on the World Health Organization Comparative Risk Factor Assessment (WHOCRA) (Norman *et al.*, 2007a). Table 2.2 clearly illustrates that Indians had the highest age-standardised death rates per 100 000 from CVD and diabetes.

Table 2.2: Age-standardised death rates per 100 000 from chronic diseases of lifestyle (CDL): 2000 (Comparative risk factor assessment, 2006) (Norman *et al.*, 2007a)

	African	White	Coloured	Indian
All causes	1613	937	1304	1172
Cardiovascular diseases	375	384	406	607
Neoplasms	126	199	212	121
Diabetes	59	23	64	111
Respiratory diseases	93	70	103	64
Other CDL	116	91	82	96
Total CDL	769	767	867	1000

SACRA identified 17 risk factors (Norman *et al.*, 2007a). These risk factors were evaluated, the proportion of DALYs attributable to the risk factor established, and recommendations made for intervention. Table 2.3 illustrates deaths attributable to selected risk factors compared with deaths attributable to disease, injury or condition. Table 2.4 illustrates DALYs attributed to selected risk factors compared with DALYs attributed to disease, injury or condition. Caution must be used in interpreting these tables. Tables 2.3 and 2.4 have no intention of showing the relationship between the

risk factor and the disease, injury or condition, but rather a representation of the ranking of risk factors with the ranking of disease, injury or condition. For example, in Table 2.3 unsafe sex/sexually transmitted diseases (STDs) are the highest ranking cause of death attributed to a risk factor and HIV/AIDS is the highest ranking cause of death attributed to disease, injury or condition. It is evident from this example that the top-ranking risk factor is related to the top-ranking disease, injury or condition in terms of death. This supports the interrelationship of NCDs as discussed under 2.1. However, further down the table, BMI is ranked fifth for deaths attributed to risk factor whereas interpersonal violence and injury is ranked as fifth for deaths attributed to disease, injury or condition. It is evident from this example that no relationship can be drawn from this ranking. Therefore, the essence of Tables 2.3 and 2.4 is to be viewed in terms of rankings. However, the interrelationship of NCDs is evident in some rankings. Bradshaw *et al.* (2007a) recommended that any intervention has to take into account the social sphere, the health sphere and the development sphere. Likewise, the rankings of the risk factors and disease states on these tables are reflective of the current epidemiological transition, development stage and social transformation in South Africa.

Table 2.3: Deaths attributed to risk factor compared with the underlying causes of death (Norman *et al.*, 2007a).

Deaths attributed to risk factor			Deaths attributed to disease, injury or condition		
Rank	**Risk factor**	**% total deaths**	**Rank**	**Disease, injury or condition**	**% total deaths**
1	Unsafe sex/STDs	26.3	1	HIV/AIDS	25.5
2	High blood pressure	9.0	2	Ischaemic heart disease	6.6
3	Tobacco smoking	8.5	3	Stroke	6.5
4	Alcohol harm	7.1	4	Tuberculosis	5.5
5	High body mass index	7.0	5	Interpersonal violence (injury)	5.3
6	Interpersonal violence	6.7	6	Lower respiratory infections	4.4
7	High cholesterol	4.6	7	Hypertensive disease	3.2
8	Diabetes (risk factor)	4.3	8	Diarrhoeal disease	3.1
9	Physical inactivity	3.3	9	Road traffic injury	3.1

By 2000, the rates for the Community Syndrome of Hypertension, Atherosclerosis and Diabetes (CHAD) showed distinct health profiles for different population groups in South

Africa (MRC, 2006). Indians had the highest prevalence of IHD and diabetes. Blacks experienced high mortality rates from stroke and hypertensive heart disease. Whites and coloureds experienced high mortality rates from IHD (Norman *et al.,* 2006). These differences suggest that the risk factor profiles differ by population groups, which are at different stages of the health transition. In addition, in terms of dietary profile, different intakes among different ethnic groups were noted, with the white, coloured and Indian populations having mean carbohydrate intakes of less than 55% of energy (%E), mean fat intake of >30%E and added sugar intakes >10%E (MRC, 2006). In support of this dietary profile, Vorster *et al.* (1997) reported that 35% of total energy intake was contributed by fat in these population groups.

Table 2.4: DALYs (disability-adjusted life years) attributed to selected risk factors compared with the underlying causes of DALYs (Norman *et al.*, 2007a).

DALYs attributed to selected risk factors			DALYs attributed to disease, injury or condition		
Rank	Risk factor	% total DALYs	Rank	Disease, injury or condition	% total DALYs
1	Unsafe sex/STIs	31.5	1	HIV/AIDS	30.9
2	Interpersonal violence (risk factor)	8.4	2	Interpersonal violence injury	6.5
3	Alcohol harm	7.0	3	Tuberculosis	3.7
4	Tobacco smoking	4.0	4	Road traffic injury	3.0
5	High BMI	2.9	5	Diarrhoeal diseases	2.9
6	Childhood and maternal underweight	2.7	6	Lower respiratory infections	2.8
7	Unsafe water sanitation & hygiene	2.6	7	Low birth weight	2.6
8	High blood pressure	2.4	8	Asthma	2.2
9	Diabetes (risk factor)	1.6	9	Stroke	2.2
10	High cholesterol	1.4	10	Unipolar depressive disorders	2.0
11	Low fruit and vegetable intake	1.1	11	Ischaemic heart disease	1.8
12	Physical inactivity	1.1	12	Protein-energy malnutrition	1.3

2.3 INDIANS IN SOUTH AFRICA

In South Africa, CHD has reached epidemic proportions amongst the white and Indian populations (Seedat & Mayet, 1996) but was relatively uncommon in blacks until

recently (Sliwa *et al.*, 2008). Nevertheless, only limited data are available on the disease patterns amongst South African Indians despite the high incidence of CHD in this ethnic group (Ranjith *et al.,* 2005). A significant difference was found with respect to gender and age in the major cardiovascular risk factor profiles in Indians admitted to the Coronary Care Unit of a large hospital in Durban (Ranjith *et al.*, 2005). Cigarette smoking was the most important risk factor (81%) in young patients (≤ 45 years) compared with 61% for middle-aged patients (>45 - ≤65 years). Diabetes mellitus (21%) and hypertension (18%) were seen less frequently in young patients compared with 39% and 41% respectively in middle-aged patients, with the highest prevalence in women (54% diabetes and 60% hypertension). Sixty-eight percent of middle-aged patients had raised total serum cholesterol values (>5 mmol/L), 57% had raised LDL-cholesterol levels (>3 mmol/L), 38% raised triglyceride levels (>2 mmol/L), 57% low HDL-cholesterol (<1 mmol/L) and 39% were overweight (BMI >25 to ≤ 30 kg/m^2). Although CHD and type 2 diabetes are common occurrences amongst Indian South Africans, limited documented dietary data are available.

2.4 HEALTH STATUS OF MIGRANT INDIANS VERSUS INDIANS IN INDIA

It has been established that the relative prevalence of CHD varies across regions and from country to country (Tunstall *et al.,* 1991). Variation has also been observed between ethnic groups, with the incidence of CHD being particularly high among the Indian population that have emigrated from the Indian sub-continent (McKeigue *et al.,* 1985; Lovegrove *et al.*, 2004; Lip *et al.*, 2007; Bhopal & Rafnsson, 2009). South Asian migrants to the UK, South Africa, Singapore and North America experience CHD mortality that is 1.5 to 4 times higher than that of their indigenous group (Enas *et al.,* 1992). By age 55, South Asians (people originating from India, Bangladesh, Sri Lanka, Nepal and Pakistan) in the UK and Canada suffer prevalences of impaired glucose tolerance (IGT), central obesity, elevated triglycerides and low HDL (McKeigue *et al.,* 1991; Anand *et al.,* 2000) and type 2 diabetes at rates four to five times higher than those of Europeans, 19% versus 4% (McKeigue *et al.,* 1991; McKeigue *et al.,* 1993). According to the British Heart Foundation (2002), the mortality from coronary artery disease (CAD) in the United Kingdom (UK) is 55% and 41% higher in Indo-Asian men and women respectively than in matched whites. Asian Indians are known to have a greater susceptibility to type 2 diabetes and premature CAD compared with Europeans

(Enas *et al.,* 1992; Reddy & Yusuf, 1998). High rates of diabetes have been reported in South Asians in the UK (10% to 19%), Trinidad (21%), Fiji (25%), South Africa (22%), Mauritius (20%) and Canada (10%) (McKeigue *et al.,* 1989; Anand *et al.,* 2000). Seedat *et al.* (1982; 1996) concluded that emigrant Indians have a higher incidence of CVD than Indians in India owing to the combination of hypertension and diabetes mellitus. Furthermore, it has been found that South Asians in the UK have higher risk factor levels compared with their siblings living in India, with a BMI of 27 *vs.* 23 kg/m², lower HDL of 1.14 *vs.* 1.27 mmol/L and higher fasting glucose of 5.4 *vs.* 4.6 mmol/L, all due to the adaptation to urban lifestyle (Bhatnagar *et al.,* 1995). More recently, Mukhopadhyay *et al.* (2006) reported that South Asians in the UK develop type 2 diabetes 11 years earlier than Europeans (46 years versus 57 years) and at a BMI lower than their European counterparts (28.7 kg/m^2 versus 29.9 kg/m^2), suggesting that there are additional underlying early risk factors that contribute to the early onset of CHD and CVD in South Asians.

Elevated risk for CHD in South Asians compared with Europeans was previously largely explained by features of the insulin resistance syndrome, as revealed in cross-sectional data (McKeigue *et al.,* 1993). However, Forouhi *et al.* (2004) conducted a 15-year prospective CHD mortality study of the Southall cohort and reported that South Asian men had a 90% increase in the risk of CHD mortality compared with Europeans but that the classical factors of smoking, hypertension and hypercholesterolaemia did not account for the high levels of CHD, nor did adjustment for features of the insulin resistance syndrome or composite definitions of the metabolic syndrome. They concluded that the search for the explanation for elevated risk in South Asians continues. Novel risk markers and alternative explanations which have been investigated and reviewed by Forouhi *et al.* (2004) include haemostatic factors, inflammation, homocysteine, lipoprotein (a) and adiponectin. Furthermore, Yajnik *et al.* (2002) tested the "thrifty phenotype" hypothesis which posits that undernutrition in foetal and infant life followed by overnutrition later in life, predisposes individuals to diabetes and other chronic diseases in South Asians in India and demonstrated that the hyperinsulinaemic, insulin-resistant phenotype of Indians is present at birth. Bhopal and Rafnsson (2009) proposed that mitochondrial efficiency might explain the susceptibility to adiposity, metabolic syndrome, diabetes and cardiovascular diseases in South Asian populations, as illustrated in Figure 2.3. In the mitochondrial efficiency hypothesis,

Bhopal and Rafnsson (2009) proposed that ancestral changes in mitochondrial coupling efficiency enhanced the successful adaptation of South Asians to environmental stressors by maximizing the conversion of energy to adenosine triphosphate rather than heat, but that this adaptation may be disadvantageous where South Asians are physically inactive and consume high-energy diets.

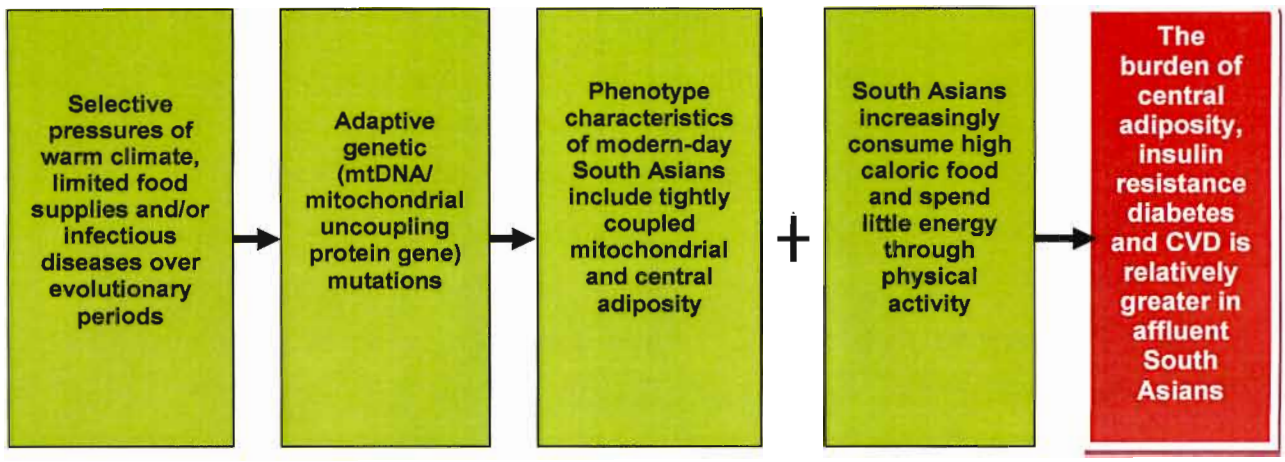

Figure 2.3: Outline of the mitochondrial energy efficiency hypothesis and its influence on health outcomes (Adapted from Bhopal & Rafnsson, 2009)

The predisposition of Indian Asians in the UK to insulin resistance, associated lipid abnormalities and CAD may, according to Lovegrove *et al.* (2004), be determined in part by genetic factors, although diet may also play a significant role. The National Food Survey in 1985 (McKeigue *et al.,* 1985) revealed lower total fat intake, higher total PUFA intake and a higher ratio of PUFAs to saturated fatty acid (SFA) in Indian Asians compared with Europeans. Sevak *et al.* (1994) also compared the dietary intake of South Asian men with Europeans in London and reported lower mean intake of energy and total fat and higher intake of starch, PUFAs and fibre in South Asians. It has been suggested that an imbalance in dietary n-6 and n-3 PUFAs may be a contributory factor to the insulin resistance and related blood lipid abnormalities of the metabolic syndrome in Indian Asians (Ghafoorunissa, 1998), although McKeigue's group in the UK reported in the eighties and early nineties that coronary risk was not related to dietary composition in South Asians (McKeigue *et al.*, 1985; Sevak *et al.*, 1994). Recently, Misra *et al.* (2009b) reviewed South Indian diets and reported low intake of MUFA, n-3 PUFA and fibre and high intake of fats, saturated fats, carbohydrates and trans-fatty

acids. These authors concluded that, in the generation of insulin resistance, the metabolic syndrome and type 2 diabetes, dietary factors are likely to have greater influence in Indian Asians than genetic factors, often overriding them.

2.5 RISK MARKERS

Unhealthy diets, physical inactivity and tobacco use have been well recognised as the major risk markers for NCDs (WHO, 2003b). Behavioural factors such as diet, smoking and physical inactivity, biological factors including hypertension, lipid abnormalities and obesity, and disease conditions such as diabetes and the metabolic syndrome as well as the clustering of risk markers will be reviewed in a separate section (2.5.3).

2.5.1 Behavioural factors

2.5.1.1 Dietary factors

2.5.1.1.1 Introduction

Diet and nutrition are significant factors in the endorsement and maintenance of health throughout life. Their role as determinants of chronic NCDs is well recognised and they therefore occupy a prominent position in prevention activities (WHO, 2003b).

2.5.1.1.2 Nutrition transition

Global diet is going through a remarkable transition: staple foods are becoming more refined and processed, fat and meat intake is increasing, more processed dairy products and other processed foods are consumed than before, and larger numbers of meals are eaten outside the home, making households more reliant on the food industry, food vendors and markets. Food consumption trends and dietary habits have largely been influenced by urbanisation (Drewnowski & Popkin, 1997). One of the effects of urbanisation is the increased participation of women in the labour force, which has indirectly affected the diet of the family (Ruel *et al.,* 2001). It is becoming more common to eat meals away from home or to eat Home Meal Replacements (HMR), with

convenience being a key factor. Sadly, however, this dietary transition is associated with the escalating trends of NCDs.

Global and regional food consumption patterns and trends have largely been pre-determined by economic development, industrialisation, market globalisation and urbanisation. Economic development has modified dietary preferences and has led to major changes in the composition of the diet, where the move from a traditional to a Western-type diet is a characteristic feature. This nutrition transition is characterised by excessive intakes of energy, fat and sugars (Drewnowski & Popkin, 1997) as mankind has an inherent preference for palatable, sugary, salty, fatty and smooth (finely textured) food (Vorster & Gibney, 2009). Adverse dietary change includes a shift in the structure of the diet towards one of higher energy density, with a greater role for fat and added sugars in foods, greater SFA intake (mostly from animal sources), reduced intakes of complex carbohydrates and dietary fibre and reduced fruit and vegetable intake (Drewnowski & Popkin, 1997). This adverse dietary change is partly due to the repercussions of the industrial revolution, which introduced radical changes in the method of food production, processing and storage, with food being developed into products that were highly processed and micronutrient-poor, all of which impact on current food consumption trends (WHO, 1990).

2.5.1.1.3 Global food supply and consumption

Data on the national availability of the main food commodities provide valuable insight into current diets and their evolution over time (WHO, 2002a). Food production trends have a direct bearing on dietary trends. Globally, the Food and Agricultural Organization (FAO) and the United Nations have published information on country-specific annual food supply including data on production, import, stock changes, export, domestic utilisation and waste. These figures give a rough but plausible estimate of the food supply situation in various parts of the world (FAO, 2000). Although the data do not take into consideration, for example, the differences in food availability among various population groups or in household food waste, they give an overall picture of the trends in food consumption on a national level. According to the data on food supply from food balance sheets, cereal availability has increased in developing countries during the last 30 years. However, the supply of traditional cereals like millet, sorghum and maize has

decreased and given way to more widely used global grains: wheat and rice. Various legumes provide a considerable amount of protein in the developing world (Bourne *et al.,* 1993; Liu, 1999). However, their availability and consumption are decreasing in many countries, giving way to animal products. There has been a considerable rise in vegetable oil supply worldwide. It was also found that the energy percentage of fat in the diet had a rising trend in all the countries with data on macronutrient intake (Chile, China, Cuba, Iran, Malaysia, Mexico, South Africa and Thailand) (Uusitalo *et al.,* 2002).

Steyn *et al.* (2006) compiled a report providing data from published research with respect to diet, dietary trends, nutritional status and diet-related chronic diseases, which were assessed in the context of trends in the chronic disease burden in South Africa over the past few decades. Food balance sheets used in this report showed that from 1962 to 2001 there were significant changes in intakes of total dietary energy, carbohydrate, protein and fat. The available energy supplies increased from 10 890 kJ per capita in 1962 to 12 221 kJ in 2001, the available protein increased from 68.4 g to 75.1 g, the available fat increased from 61.2 g to 79 g and the available carbohydrate supplies increased from 445 g to 478 g. The pattern reflected is indicative of the fact that from 1962 to 2001 more food was available to consumers (Steyn *et al.,* 2006).

According to the FAO (2000), food consumption data for South Africa showed that cereal availability increased from 169.3 kg per capita per annum in 1962 to 187.8 kg in 2001; vegetable oils increased from 5.7 to 14.5 kg, fruits increased from 24.1 to 36.0 kg, alcohol increased from 43.8 to 56.8 L, meat increased from 31.6 to 37.5 kg, eggs increased from 2.5 to 6.1 kg, and fish increased from 5.5 to 7.9 kg. It is evident from these results that the availability of staple cereals gradually increased, as did the other items mentioned above, which accounted for the overall increase in energy intake. Vegetable oil and meat per capita also increased significantly, which accounted for the large increase in fat and saturated fat intake.

To understand the dynamics of dietary changes, the main food groups consumed by South African adults and children in urban and rural areas were examined. The data were summarised from combined databases, using secondary data analyses where no national data were available, to show the dietary intake of adults (Steyn *et al.,* 2001; Nel & Steyn, 2002) and children (1-5 years) (Labadarios *et al.,* 2000). Although rural

dwellers had a higher cereal and vegetable intake, urban adults and children had a far higher consumption of most other food groups such as sugar, meat, vegetable oil, dairy, fruit, roots, tubers and alcohol.

2.5.1.1.4 Diet in the aetiology of NCDs

The role of diet in the aetiology of most NCDs is extremely important. A body of literature attests to the fact that diets and specific nutrient deficiencies and excesses influence the development of NCDs (Vorster & Gibney, 2009). A plethora of scientific publications has revealed – through ecological and epidemiological studies, interventions with specific nutrients and foods in placebo-controlled trials and through molecular and genetic research – that there is a consistent relationship between unhealthy diets and the emergence of NCDs (Vorster & Gibney, 2009). This body of knowledge amassed over the years has led to several sets of international guidelines to reduce the burden of nutrition-related NCDs (Vorster & Gibney, 2009). Nutrition is therefore seen as a major modifiable determinant of chronic diseases with compounding scientific evidence increasingly supporting the view that alterations in the diet have strong effects. As mentioned in Chapter 1, several countries have developed innovative interventions for promoting a healthy diet, e.g. South Korea has gone back to promoting the traditional diet (Lee *et al.,* 2002) and the Northern Karelia project in Finland adopted a well planned community-based approach (Pushka, 2002). The WHO endorsed the Global Strategy on Diet, Physical Activity and Health in 2004 and recently published a report on tried and tested interventions in diet and physical activity, presenting the evidence on effective interventions in various categories (WHO, 2009).

2.5.1.1.5 Population nutrient intake goals

The WHO published population nutrient intake goals (indicated in Table 2.5) for the prevention of death and disability from NCDs, where nutrient intake goals represent the population average intake that is judged to be consistent with the maintenance of health in a population (WHO, 2003b). The strength of evidence upon which these goals are based is discussed in section 2.6.

Table 2.5: The WHO population nutrient intake goals for prevention of death and disability from NCDs (WHO, 2003b)

Dietary factor (food or nutrient)	Goal (% of total energy, unless otherwise stated)
Total fat	15-30%
Saturated fatty acids	<10%
Polyunsaturated fatty acids	6-10%
n-6 fatty acids	5-8%
n-3 fatty acids	1-2%
Trans-fatty acids	<1%
Monounsaturated fatty acids	By difference[a]
Total carbohydrate	55-75%[b]
Free sugars	<10%[c]
Protein	10-15%
Cholesterol	<300 mg per day
Sodium chloride (sodium)	<5 g per day (<2 g per day)
Fruits and vegetables	≥400 g per day
Total dietary fibre	>25 g per day

[a] Monounsaturated fatty acids are calculated as total fat minus saturated plus polyunsaturated plus trans-fatty acids
[b] Energy from carbohydrates is the percentage energy available after taking into account that consumed as fat and protein
[c] Free sugars refer to all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer plus sugar naturally present in honey, syrups and fruit juices. It does not include sugars present in milk, fruit and vegetables

Fat

According to the WHO guidelines for population nutrient goals, the recommendations for total fat are formulated to include countries where the usual fat intake is typically above 30% as well as those where the usual intake may be very low, for example, less than 15%. Total fat energy of at least 20% is recognised as consistent with good health (WHO, 2003). Fatty acids have different roles and functions which must be understood in order to make effective recommendations for nutrition interventions. In studies of the effects of fatty acids on serum lipids, fat quality, fat quantity, cholesterol and numerous dietary substances have been investigated to see how they affect serum lipids and lipoprotein. Two points of comparison need to be borne in mind. Firstly, how do fatty acids compare with carbohydrate substitutes, and secondly, how do they compare when they replace SFAs? Generally, SFAs tend to elevate blood cholesterol in all lipoprotein fractions (both LDL- and HDL-cholesterol) when substituted for carbohydrates or other fatty acids. The most hypercholesterolaemic or atherogenic SFAs are lauric, myristic

and palmitic acids. Palmitic and myristic acids have the greatest effect and are abundant in diets rich in dairy products and meat (Mahan & Escott-Stump, 2008). SFAs raise LDL-cholesterol by decreasing LDL-receptor synthesis and activity (Mahan & Escott-Stump, 2008). The most effective replacements for SFAs in terms of CHD outcome are PUFAs (WHO, 2003). Replacement of SFAs with monounsaturated fatty acids (MUFAs) (as would happen when substituting olive oil for butter) lowers total serum cholesterol levels, LDL-cholesterol levels and triglyceride levels to about the same extent as PUFAs. If carbohydrates are replaced by LA, the most important PUFA, LDL cholesterol is lowered and HDL is raised (Mensink *et al.*, 2003). N-3 fatty acids do not affect total cholesterol but they do increase LDL-cholesterol, decrease triglycerides (Harris, 1997; Mahan & Escott-Stump, 2008) and may improve insulin sensitivity in subjects with impaired glucose tolerance (Popp-Snijders *et al.*, 1987). The only nutritionally important MUFA is oleic acid, which is abundant in olive and canola oils and also in nuts. With regard to trans-fatty acids, the trans-isomer of oleic acid raises LDL-cholesterol and decreases HDL-cholesterol levels; these acids should therefore be regarded as SFAs and should be restricted (Mahan & Escott-Stump, 2008). Trans-fatty acid intake has been associated with dyslipidaemia and an increased risk of type 2 diabetes and CVD (Ascherio *et al.*, 1999; Salmeron *et al.*, 2001). Trans-fatty acids occur naturally in food (butter and beef fat), as they are formed as a result of the bacterial fermentation in the rumen of cows and sheep. However, most trans-fatty acid intakes come from partially hydrogenated vegetable oils. The intake of trans-fatty acids should be no more than 1%E or about 1-3 g per day (Mahan & Escott-Stump, 2008). Ways to improve the lipid profile are discussed under 2.5.2.2.

Carbohydrates

With regard to carbohydrates, the WHO population nutrient guidelines advocate an intake providing 55-75% of total energy, with the recommended intake of free sugars being <10% of total energy (WHO, 2003). It is recognised that the higher intakes of free sugars threaten the nutrient quality of diets by providing significant energy without specific nutrients (WHO, 2003). Furthermore, Sevak *et al.* (1994) showed that the total carbohydrate and sucrose contents of the diets of South Indians residing in the UK were positively correlated with postprandial hyperinsulinaemia.

Fruit and Vegetables

The WHO population nutrient guidelines (WHO, 2003b) as well as the South African food-based dietary guidelines (Love & Sayed, 2001) prescribe an intake of ≥ 400 g of fruits and vegetables per day. Participants of the First Indo-US Healthcare Summit recommended at least 500 g of fruit and vegetables per day to prevent CAD among Indians (Enas *et al.*, 2008). In a study conducted by Ganesan *et al.* (2008), it was concluded that increased intake of fruit and vegetables could play a protective role against CVD in Asian Indians. The findings of the study showed that a higher intake of fruit and vegetables accounted for 48% of the protective effect against CVD risk factors. The beneficial effects of diets rich in fruit and vegetables have been well recognised for the prevention of chronic diseases, especially CVD, as they contain large amounts of nutrients such as folate, antioxidants and dietary fibre (Khaw & Barret-Connor, 1987; Rimm *et al.,* 1996).

Fibre

The WHO population nutrient intake goal prescribes >25 g fibre per day (WHO, 2003b). Soluble fibres lower total serum cholesterol and LDL-cholesterol. The proposed mechanisms for the hypocholesterolaemic effect of soluble fibre are that the fibre binds bile acids, which lowers serum cholesterol to replete the bile acid pool, and bacteria in the colon ferment the fibre to produce acetate, propionate and butyrate, which may inhibit cholesterol synthesis (Mahan & Escott-Stump, 2008). Insoluble fibres such as cellulose and lignin have no effect on serum cholesterol levels (Mahan & Escott-Stump, 2008). Asian Indians are known to have a lower BMI than Europeans (Raji *et al.,* 2001). However, for any given BMI, Asian Indians have a greater waist-to-hip ratio (Ramchandran *et al.*, 1997; Raji *et al.,* 2001) and more abdominal fat than Europeans. Some studies suggest that a diet rich in dietary fibre is protective against obesity and obesity is less common in a population that consumes a high-fibre diet (Van Itallie, 1978). The mechanisms for the underlying effect are possibly due to high intake of fibre, which further reduces energy intake, improves insulin sensitivity and reduces insulinaemia (Ludwig *et al.,* 1999). Viswanathan and Mohan (1991) showed a significant reduction in serum cholesterol among diabetic patients with a high-carbohydrate, high-fibre diet, and these effects were sustained for a long period of time (Viswanathan *et al.*, 1981).

Other factors

Other dietary factors such as alcohol, anti-oxidants and soy protein intake also need to be considered. Alcohol raises both triglyceride and HDL-cholesterol levels (Miettinen *et al.*, 1995). The effect of alcohol on triglycerides is dose-dependent and is greater in persons with triglyceride levels exceeding 1.69 mmol/L (Mahan & Escott-Stump, 2008). In terms of anti-oxidants, two dietary components that affect the oxidation potential of LDL-cholesterol are the level of LA in the particle and the availability of antioxidants. Soy protein lowers total cholesterol, LDL and triglycerides and has no effect on HDL-cholesterol. Plant stanols and sterols lower serum cholesterol (Miettinen *et al.*, 1995). Elevated serum homocysteine levels, independent of other cardiac risk factors, are associated with increased risk for CHD. Compared with Europeans, Asian Indian men in the UK have higher concentrations of plasma homocysteine (Chambers *et al.*, 2000). Data consistently show that dietary supplementation of folic acid, with or without other B vitamins, lowers homocysteine levels; however, this is not accompanied by lower heart disease risk (Van Horn *et al.*, 2008).

2.5.1.1.6 Characteristic features of the Indian diet

Apart from reviewing food consumption trends and existing interventions, it is also important to review the characteristic features of the Indian diet. The Indian sub-continent is characterised by cultural heterogeneity with a huge difference in food consumption patterns amongst the different communities across the Indian Diaspora (Ghosh *et al.,* 2003; Ghosh, 2004). This diversity in food consumption is a potential risk factor for many chronic conditions such as dyslipidaemia in Asian Indians. In general, vegetarianism tends to predominate, but variation in carbohydrate, fat and fibre intake depends largely on geographical region and caste of individual (Misra *et al.*, 2001).

Increased risk for diabetes and CHD in Asian Indians is confined to urban and emigrant populations (Raheja *et al.*, 1993). These populations have deviated from their traditional low-fat lactovegetarian diet and instead consume a high-fat, refined cereal diet, partially deficient in the n-3 fatty acids which are required for normal insulin action (n-6:n-3 ratio of 19-50 or more *vs.* a ratio of 5.5 in the traditional diet). Indians in the UK also consume almost twice the quantity of n-6 compared with the local population (24-26 g *vs.* 12-14 g,

i.e. 8-10% versus 5.1% energy), with lower n-3 intake (0.4-0.6% *vs.* 0.7% energy) (Raheja *et al.*, 1993).

In terms of Indian dietary intake in South Africa, little is known. In a study conducted by Wolmarans *et al.* (1999), one of the salient features of the Indian diet was the high dietary P:S ratio. This study also revealed that the Indian diet had elements of a typical Western-type diet in terms of energy distribution for total fat intake and dietary fibre intake (Wolmarans *et al.*, 1999).

2.5.1.1.7 Nutrients versus dietary patterns

Interpreting results from studies that examine single dietary factors such as nutrients is difficult because of the interrelationship of nutrients and of foods consumed. Therefore, examining dietary exposure in its totality is more attractive. Kant (2004) reviewed the literature on dietary patterns in relation to nutrient adequacy, lifestyle, demographic variables and health outcomes. Most published reports on the subject have used one or two methods: either the diet indexes/scores that assess compliance with prevailing dietary guidance as dietary patterns, or the data-driven method that uses factor/clusters analysis to derive dietary patterns. In this review, it was found that age, income and education have been reported to be among positive predictors of the so-called healthy dietary patterns (Kant, 2004). In examining diet as a multidimensional experience, food combinations consumed reflect individual food preferences modulated by a mix of genetic, cultural, social, health, environmental, lifestyle and economic determinants (Krondl & Coleman, 1986; Quandt, 1999; Van der Bree *et al.*, 1999). Kant concluded from her systematic review that the reported dietary indexes/scores as dietary patterns can be grouped into three major categories: dietary variety-based scores, scores derived from assessment of dietary guidelines and scores based on assessment of the Mediterranean diet (Kant, 2004).

In summary, diet and nutrition are prominent factors in the promotion and maintenance of good health throughout the life course. It is seen as the most modifiable factor of NCDs; therefore, all variables related to dietary factors need to be carefully assessed for successful intervention. Since the prevalence of metabolic abnormalities in Indian Asians compared with Caucasians may be attributable in part to differences in intakes

of n-6 and n-3 PUFAs (Ghafoorunissa, 1998), these long-chain fatty acids will be discussed in more detail.

2.5.1.2 Omega-6 and omega-3 fatty acids

2.5.1.2.1 Introduction

PUFAs of the n-6 and n-3 series are essential nutrients that exert an important influence on plasma lipids and serve cardiac and endothelial functions to impact the prevention and treatment of CHD (Stanley *et al.*, 2007). Both n-6 and n-3 PUFAs have distinct biological effects contributing to their cardioprotective action. While it is accepted that PUFAs of both series are dietary essentials, both the absolute intakes (g per day) and the n-6:n-3 ratio required to achieve optimal CHD health benefits are somewhat controversial, in part due to the failure to consider their intake in the context of total daily fat and total daily PUFA (i.e. as a percent of energy) consumed by the population under study (Stanley *et al.*, 2007). The role of n-6 and n-3 PUFAs in cardiovascular risk with reference to the "ideal" balance between the two series of fatty acids will be reviewed in this section with the focus on the day-to-day consumption of a healthy amount of essential fatty acids, not on their potential as corrective therapy for individuals with CHD.

PUFAs are natural constituents of animal and vegetable lipids, with the unique feature of containing more than one double bond (Burdge & Calder, 2005). Two PUFAs, LA (n-6 fatty acid, C18:2) and α-linolenic acid (ALA, n-3 fatty acid, C18:3) are termed essential because they cannot be synthesised by the body; therefore, these fatty acids have to be provided exogenously in the form of food. A complex series of desaturation and elongation reactions acting in concert transform LA and ALA to their higher unsaturated derivates: AA from LA and EPA and DHA from ALA. Both series compete for the activity of a rate-limiting Δ-6-desaturase and, although this enzyme shows greater substrate specificity for ALA, the overabundance of dietary LA gives it a quantitative advantage that limits the conversion of ALA to EPA *in vivo* (Burdge & Calder, 2005). Only small (2% to 5%) amounts of ALA are converted to EPA in humans (Davis & Kris-Etherton, 2003). Even less is converted to DHA (<1%) (Hussein *et al.*, 2005). EPA (20:5) and DHA (22:6) are found in cold water fish such as mackerel, salmon, herring, trout,

sardines and tuna. ALA is found in various plant sources, including flaxseed, walnuts, canola oil and soybeans (van Horn *et al.*, 2008). In the absence of preformed EPA and DHA from fish oil or supplements, the dietary ratio of n-6 to n-3 PUFAs in a western diet is determined, for the most part, by the relative proportion of LA (~95%) to ALA (~90%) (Stanley *et al.*, 2007). Because of the competition between LA and ALA for metabolism by Δ-6-desaturase, the n-6:n-3 fatty acid ratio is potentially useful. It may say something about the relative rates of metabolism of the two fatty acids *in vivo* (Stanley *et al.*, 2007).

2.5.1.2.2 Linoleic acid

Characteristics of linoleic acid

LA is the primary (in terms of mass consumed) essential fatty acid and represents the basis of the n-6 family. It is the most common PUFA incorporated into dynamic phospholipids needed for membrane structure and lipoproteins [particularly phospholipid-rich HDL involved in lipid transport (Wijendran & Hayes, 2004)]. Epidemiological evidence supports a role for dietary LA in reducing the risk of CHD. A cross-sectional study in healthy men from four European populations found that higher adipose tissue LA, a marker of dietary LA intake, was associated with lower CHD mortality (Riemersma *et al.*, 1986). In the National Heart, Lung, and Blood Institute Family Heart Study (Djousse *et al.*, 2001), the highest tertile of LA intake (11.7 g per day, or about 5% of energy at 8400 kJ per day energy intake) was associated with a lower prevalence odds ratio for coronary artery disease compared with the lowest tertile of LA intake (3.9 g per day or about 1.7% of energy), when adjusted for ALA intake and other relevant risk factors. Randomised controlled intervention studies have shown that diets high in LA reduce the incidence of CHD compared with diets higher in SFAs [National Cholesterol Education Program/ Third Adult Treatment Panel (NCEP/ATP111), 2002; Van Dam *et al.,* 2002] and LA intake has been inversely associated with the risk of type 2 diabetes (Van Dam *et al.,* 2002). On the other hand, some individuals and groups have recommended substantial reductions in n-6 fatty acids (Simopoulos *et al.,* 1999; Hamazaki & Okuyama, 2003; Sears, 2003; Simopoulos, 2008). Arguments for reduced LA intakes are based on the assumption that because CHD has an inflammatory component (Libby, 2006) and because the n-6 fatty acid AA is the substrate for the synthesis of a variety of pro-inflammatory molecules, reducing

LA intakes should reduce tissue AA content, which should reduce the inflammatory potential and therefore lower the risk for CHD.

Health benefits of linoleic acid

The cholesterol-lowering effect of LA is well established in human trials. In a meta-analysis of 60 feeding studies, the substitution of PUFAs (mainly n-6) for carbohydrate had favourable effects on the ratio of total cholesterol to HDL-C (Mensink *et al.,* 2003). Higher plasma PUFA levels are inversely associated with the ratio of total cholesterol to HDL-C (Siguel, 1996). These findings confirm an LDL-lowering effect of n-6 fatty acids beyond that produced by the removal of SFAs. Additionally, higher intakes of LA may improve insulin resistance (Salmeron *et al.,* 2001) and reduce the incidence of diabetes mellitus (Grimsgaard *et al.,* 1999), and higher serum LA levels are associated with lower BP (Mozaffarian *et al.,* 2004). LA is the most potent dietary fatty acid for reducing serum total cholesterol and LDL-C (Hegsted *et al.*, 1965; Mensink *et al.*, 2003).

A scientific advisory from the American Heart Association (AHA) recently reviewed evidence on the relationship between n-6 PUFAs and the risk of CHD and CVD (Harris *et al.*, 2009) and concluded that the combined results of observational and randomised controlled studies provide evidence that replacing SFA or refined carbohydrate with n-6 PUFAs (10% to 21% of energy) reduces CHD risk compared with lower intakes of n-6 PUFAS, with no clinical evidence of adverse events. The advisory undertaken by Harris *et al.* (2009) summarised the current evidence on the consumption of n-6 fatty acids. Aggregate data from randomised trials, case-control and cohort studies indicate that the consumption of at least 5% to 10% of energy from n-6 fatty acids reduces the risk of CHD relative to lower intakes. The data also suggest that higher levels appear to be safe and may be even more beneficial (as part of a low-SFA, low-cholesterol diet). In summary, the AHA supports n-6 PUFA intake of 5% to 10% of total daily energy intake in the context of other lifestyle and dietary recommendations (Harris *et al.,* 2009) and expressed concern that the percentage of individuals who meet the adequate intake (AI) (Food and Nutrition Board (FNB) of the Institute of Medicine, IOM, 2000) for this fatty acid would decrease with increased replacement of currently used vegetable oils (rich in n-6) with canola oil (rich in n-3 fatty acids). These observations suggest that a mixture of vegetable oils that contain ALA and LA could be used to optimise compliance with the AI for these fatty acids. The FNB issued an acceptable macronutrient distribution range

(AMDR) of 5% to 10% of energy as n-6 PUFAs and 0.6% to 1.2% of energy as ALA (FNB/IOM, 2000). The group also established AI values for ALA of 1.1 g/day for adult women and 1.6 g/day for adult men and for LA, 11-12 g/day for adult women and 14 to 17g /day for adult men (FNB/IOM, 2000).

2.5.1.2.3 Alpha-linolenic acid

ALA is the second primary essential fatty acid that can be elongated and desaturated to form highly specialised 22-carbon fatty acids within the n-3 family. Human studies suggest that dietary ALA (and its subsequent metabolic end products) may protect against CHD (Hu *et al.*, 1999b; Djousse *et al.*, 2001). The highest quintile of ALA intake (1.36 g per day or 0.6% of energy) was associated with 45% fewer cardiac deaths compared with the lowest quintile (0.71 g per day or 0.3% of energy) in women in the Nurses' Health Study (Hu *et al.*, 1999b). In the National Heart, Lung, and Blood Institute Family Heart Study (Djousse *et al.*, 2001), the highest quintile of ALA intake (1.1 g per day or 0.5% of energy intake) was associated with 40% lower mortality from coronary artery disease compared with the lowest quintile of ALA intake (0.5 g per day or 0.2% of energy), after adjusting for LA intake and other relevant factors. However, in a recent systematic review, the authors (Wang *et al.*, 2006) found no high-quality evidence of reduction in rates of all-cause mortality, cardiac and sudden death and possibly stroke by ALA. Two of the randomised controlled ALA dietary trials included in the review reported significant reductions or trends toward lower rates of all-cause mortality, cardiac or sudden death or non-fatal myocardial infarction (MI) (Singh *et al.*, 2002; de Lorgeril *et al.*, 1999), whereas the third trial (Bemelmans *et al.*, 2002) reported a decrease in the estimated IHD risk similar in extent to that found with increased LA intakes. The way in which dietary ALA may reduce CHD risk is not clear, but may have more to do with cardiac function (antiarrhythmic effect) than with plasma lipids (Hu *et al.*, 1999b).

2.5.1.2.4 Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)

EPA and DHA are potent triglyceride-lowering and effective antiarrhythmic agents (reviewed by Wijendran & Hayes, 2004). Considerable data suggest that DHA and EPA,

found primarily in oily fish, decrease the risk of CHD (Kris-Etherton *et al.,* 2002). In the Physicians' Health Study, consumption of one or more servings of fish per week was associated with a 52% lower risk of sudden cardiac death compared with less than one fish meal per month (Albert *et al.*, 1998), and in the Nurses' Health Study consumption of five or more servings of fish per week was associated with 45% fewer cardiac deaths compared with consumption of less than one fish meal per month (Hu *et al.*, 2002). Randomised clinical trials have also shown a protective effect of EPA and DHA on CHD mortality (reviewed by Wijendran & Hayes, 2004). However, a large randomised controlled trial to study the effect of dietary advice for men with angina to increase the intake of oily fish (two portions per week) and/or three oily fish capsules per day for 3-9 years resulted in a higher risk of cardiac death (Burr *et al.*, 2003). Inclusion of this study in a meta-analysis of the effect of long-chain n-3 fatty acids (EPA, DHA and docosapentaenoic acid) and shorter-chain ALA on total mortality, cardiovascular events and cancer, led the authors (Hooper *et al.*, 2006) to conclude that neither the long-chain nor the shorter-chain fats have a clear effect on these outcomes. Furthermore, according to the authors, the source (dietary or supplemental) and dose of n-3 fats did not seem to influence the effectiveness of long-chain n-3 fats. Their results differ from those of a systematic review by Bucher *et al.* (2002), which reviewed trials assessing the effects of long-chain n-3 fatty acids over at least six months in patients with CHD (not including the Burr *et al.* trial) and found significant protection from mortality and sudden death. Although there is no clear evidence to show that n-3 fatty acids alter total mortality or CVD events in those with CVD, for the general population the advice is to eat two portions of fish each week, one of which should be oily (Food Standards Agency, Committee of Toxicity, 2004). Table 2.6 summarises the biological effects of n-6 and n-3 polyunsaturated fatty acids on cardiovascular risk factors.

Lower circulating and tissue levels of long-chain n-3 PUFAs and a higher proportion of total fatty acids like n-6 PUFAs LA and AA have been reported in some Indo-Asian groups (McKeigue *et al.,* 1985; Conquer & Holub, 1998), which could be ascribed to a lack of the n-3 fatty acids in the diet, to a dietary excess of n-6 PUFAs or to a metabolic defect in the incorporation of long-chain n-3 PUFAs (Lovegrove *et al.*, 2004). Interestingly, intervention studies in South Asians with n-3 PUFA improved serum lipid profiles but have not yielded encouraging results with regard to insulin sensitivity (Brady *et al.*, 2004; Lovegrove *et al.*, 2004; Minihane *et al.*, 2005).

Table 2.6: Biological effects of n-6 and n-3 polyunsaturated fatty acids on cardiovascular risk factors (adapted from Wijendran & Hayes, 2004)

Fatty Acid	Parameters	Effect
Linoleic acid	Plasma lipids	
	TC	↓
	LDL-C	↓
	HDL-C	↑ or ↔ or ↓
	LDL-C:HDL-C	↓
	Hepatic LDL-C clearance	↑
	Hepatic LDL-C production	↓
Linolenic acid	Plasma lipids	
	LDL-C	↓ or ↔
	HDL-C	↓ or ↔
	LDL-C:HDL-C	↑ or ↔
	Arrhythmia	↓
	Thrombosis	↔ or ↓
EPA + DHA	Plasma lipids	
	TG	↓
	LDL-C	↑
	LDL-C:HDL-C	↑
	Hepatic TG and apo B secretion	↓
	Arrhythmia	↓
	Vascular endothelial function	Improved
	Blood pressure	↓
	Pro-inflammatory factors	↓ or ↔

HDL, high-density lipoprotein; TC, total cholesterol; TG, triglyceride, LDL-C, low-density lipoprotein cholesterol; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid

2.5.1.2.5 Ratio of dietary LA to ALA

The optimum ratio of dietary LA to ALA is a matter of intense scientific scrutiny (Kris-Etherton *et al.,* 2000). The FNB macronutrient report (FNB/IOM, 2000) cited a FAO/WHO recommendation (FAO, 1992) that concluded that a ratio of 5:1 to 10:1 is reasonable for adults. Some researchers have suggested that increasing the intake of n-3 fatty acids and decreasing that of n-6 fatty acids may be beneficial (Dolecek, 1992; Wijendran & Hayes, 2004). A decrease in the n-6 to n-3 ratio might have favourable effects on inflammation due to competition between LA and ALA for the Δ-6-desaturase enzyme that converts both of these fatty acids to inflammatory mediators (e.g. prostaglandins, leukotrienes and related metabolites). However, Pischon and colleagues (2003) found that an increased intake of n-6 fatty acids did not inhibit anti-

inflammatory effects of n-3 fatty acids among 859 healthy adults. In the study conducted by Mozzarrarian *et al.* (2005), ALA intake was inversely associated with the risk of CHD, whether or not n-6 fatty acid intake was above or below median intakes (11.2 g/day). These authors concluded that attention to relative intakes of n-3 and n-6 fatty acids may be less important than simply increasing the intakes of n-3 PUFAs. This position is supported by Goyens and colleagues who found that conversion of ALA into longer-chain n-3 fatty acids is not determined by the n-3 to n-6 ratio but by the absolute amounts of ALA or LA in the diet (Goyens *et al.,* 2006). Despite uncertainty in this area, the AHA Nutrition Committee concluded that total intakes of approximately 1.5 to 3 g/day ALA seem to be beneficial (Kris-Etherton *et al.*, 2002) and recommend that sources of vegetable oils and other foods that are high in ALA are regularly consumed.

The question of whether the ratio of n-6:n-3 PUFAs or the total amount of dietary PUFAs is of more importance to cardiovascular health has been addressed recently in a randomly controlled trial (OPTILIP) by Griffin *et al.* (2008), using a six-month, food-based intervention in older men and women. There were no effects of the n-6:n-3 ratio or EPA/DHA on insulin sensitivity and only minor effects on serum lipids. Additional EPA/DHA (~1 g per day) showed significant decreases in fasting and postprandial serum triglycerides and small, dense LDL. The conclusion reached was that the dietary n-6:n-3 PUFA ratio is of no predictive value in modifying CVD risk factors but reaffirmed the cardioprotective benefits of dietary EPA/DHA at levels of intake that are achievable through diet. The OPTILIP study also tested the hypothesis that lowering the dietary n-6:n-3 ratio might improve haemostatic factors (fibrinogen, activated factor XII and factor VII coagulant activity) (Sanders *et al.*, 2006). However, lowering the ratio by the addition of EPA/DHA did not influence these haemostatic factors. The stable isotope tracer study of Goyens *et al.* (2006) provided the most definite evidence so far that the absolute amounts of LA and ALA and not the relative proportions influence the conversion of ALA to long-chain n-3 PUFAs. The study tested the effects of a low LA and high ALA diet against a control group. It showed that lowering LA is more effective in promoting conversion of ALA to EPA, whilst increasing ALA facilitates conversion of EPA to DHA. The UK Food Standards Agency Workshop (Stanley *et al.,* 2007) reviewed experimental and theoretical evidence on the effects of dietary n-6:n-3 fatty acid ratio on cardiovascular health and recommended that "the use of the ratio to estimate CVD risk should be abandoned and that further research in this area is unlikely to be fruitful."

Advice to reduce n-6 fatty acids intakes is typically framed as a call to lower the ratio of dietary n-6 to n-3 fatty acids (Simopoulos *et al.,* 1999; Hamazaki & Okuyama, 2003; Sears, 2003, Simopoulos, 2008). Although increasing n-3 fatty acids does reduce the risk of CHD (Kris-Etherton et *al.,* 2002; Mozaffarian & Rimm 2006), it does not follow that decreasing n-6 levels will do the same. Furthermore, high intakes of n-3 fatty acids can cause excessive bleeding in some individuals. Thus, patients taking 3 g/day long-chain n-3 fatty acids should do so only under a physician's care (Kris-Etherton *et al.,* 2002). Higher n-6 fatty acid intakes can inhibit the conversion of ALA to EPA (Liou *et al.,* 2007) but such conversion is already quite low (Brenna, 2009) and whether additional small changes would have net effects on CHD risk after all the benefits of LA consumption are taken into account, is not clear. The focus on ratio rather than levels of intake of each type of PUFA has many conceptual and biological limitations (Harris *et al.,* 2009).

2.5.1.3 Smoking

Cigarette smoking accounts for a large burden of preventable disease in South Africa. Smoking causes many diseases such as lung cancer, chronic obstructive pulmonary disease (COPD) and CVD. According to the MRC (MRC, 2006), the smoking pattern displayed in South Africa illustrates the effect of an aggressive anti-smoking policy from 1990 to 2004, with a resultant decline in the prevalence of smoking.

Age, gender, 'race', cultural and economic characteristics all affect smoking prevalence rates. According to the SADHS in 1998, smoking prevalence was highest among coloured and Indian males, followed by black males. White males and coloured females had the same prevalence rate. In comparison with figures from 1998, data from the 2003 SADHS revealed a decrease across all race groups for men who smoke daily or occasionally, except for Indian men, who showed an increase and also have the highest prevalence at 56% compared with the average of 31.7% for all population groups. Furthermore, the rates of smoking daily or occasionally for women increased slightly across all race groups, particularly among Indian women, where the rate was 13%, compared with the mean of 9% for all population groups (Department of Health, Medical Research Council & OrcMacro, 2007).

In terms of mortality rates in South Africa which are attributable to tobacco, there is also a distinct variation between population groups and gender. In the year 2000, for men 30 years and older, the highest rate of tobacco-related deaths was found in coloureds (417/100 000), followed by blacks (279/100 000) and Indians (276/100 000), with the lowest rate in whites (215/100 000). The highest rate of tobacco-related deaths amongst women was prevalent amongst coloureds (200/100 000), followed by whites (104/100 000), then blacks (36/100 000) and finally, Indians, with a prevalence of 34/100 000 (Groenewald *et al.*, 2007).

2.5.1.4 Physical inactivity

Physical activity has been well recognised as a key health behaviour associated with reduced all-cause mortality and morbidity. With sedentary lifestyles there is a decline in energy expenditure, leaving individuals more susceptible to chronic diseases. The benefits of physical activity have been extensively reviewed. It appears to be protective for chronic diseases, decreasing serum triglyceride concentrations, improving tissue sensitivity to insulin, increasing fibrinolytic activity, decreasing clotting activity, increasing HDL-cholesterol concentrations and lowering BP (Morris *et al.*, 1966; Bouchard & Rankinen, 2001).

The results of the South African National Burden of Disease Study (SANBD) showed that in adults in the year 2000, 30% of IHD, 27% of colon cancer, 22% of ischaemic stroke, 20% of type 2 diabetes and 17% of breast cancer were attributed to physical inactivity (Joubert *et al.,* 2007b). Moreover, the findings showed that, compared with other regions and the global average, South African adults have a high prevalence of physical inactivity, with 49% of adult women and 43% of adult men being inactive (Joubert *et al.,* 2007b). The 2003 SADHS provided nationally representative data on physical activity of adults, which revealed that KwaZulu-Natal has the highest levels of inactivity for both men and women (66% and 81% respectively) (Department of Health, Medical Research Council & OrcMacro, 2007). With reference to the Indian population, it was found that 43.1% of men and 62.6% of women were inactive.

A systematic review of 17 studies in the UK, Fischbacher *et al.* (2004), reported substantially and consistently lower levels of physical activity and fitness in South Asians compared with Europeans. Although there are many determinants of levels of physical activity, cultural and religious issues may be particularly important in some groups such as South Asian Muslim women (Fischbacher *et al.*, 2004).

It is well established that associated health benefits of physical activity accrue in a dose-dependent manner with increasing frequency, duration and intensity (Bouchard *et al.,* 2001). The current public health recommendation is 30 minutes of accumulated moderate to vigorous activity on most days (WHO, 2003b). To lose weight, 60 minutes of physical activity is recommended per day. The recommendations of the American College of Sport Medicine (ACSM) and AHA for healthy adults under age 65 years are as follows: moderately intense cardio for 30 minutes a day, five days a week *or* vigorously intense cardio for 20 minutes a day, three days a week *and* eight to ten strength-training exercises, with eight to 12 repetitions of each exercise twice a week. Moderate-intensity physical activity means working hard enough to raise heart rate and break a sweat, yet still being able to carry on a conversation. It should be noted that to lose weight or maintain weight loss, 60 to 90 minutes of physical activity on most days may be necessary. The 30-minute recommendation is for the average healthy adult to maintain health and reduce the risk of chronic disease (ACSM and AHA, 2007).

2.5.2 BIOLOGICAL FACTORS

2.5.2.1 High blood pressure

High BP has been recognised as being responsible for the increasing proportion of the burden of disease both globally and regionally. Globally, analysis indicates about two-thirds of stroke and almost one-half of IHD cases are attributable to raised BP with the systolic BP ≥115 mm Hg (Norman *et al.*, 2006).

According to Seedat (2004), the prevalence of CVD and hypertension is rapidly increasing in sub-Saharan Africa (SSA). The control of BP was reported to be less than 1% in Tanzania (Edwards *et al.,* 2000) and 15% amongst blacks in South Africa (Steyn *et al.,* 2001). From this study, it was concluded that countries in SSA should develop

country-specific recommendations on the prevention and management of NCDs, as already recommended by the World Health Assembly and WHO (Seedat, 2004).

High BP is common in South Africa and people are usually unaware of the silent epidemic (Seedat, 1999). High BP is a risk factor for heart attack, stroke, left ventricular hypertrophy, renal diseases and blindness. It frequently co-exists with other risk factors for chronic diseases of lifestyle such as diabetes and obesity. The determinants of hypertension are high body weight, pulse rate and urinary sodium/potassium ratios. According to Seedat (1999), CHD is a major outcome related to hypertension in white and Indian populations in South Africa. In contrast, black patients are more prone to cerebral haemorrhage, malignant hypertension and kidney disease, whereas CHD was uncommon until recently (Sliwa *et al.,* 2008).

The 2003 SADHS provided nationally representative data on the prevalence of hypertension (Department of Health, Medical Research Council & OrcMacro, 2007). The mean systolic BP for those over the age of 15 years was 123 mmHg for men and 121 mmHg for women. The prevalence of hypertension using the 1999 World Health Organization cut-offs, suggesting that a person be considered as hypertensive with a BP ≥140/90 mm Hg and/or taking anti-hypertensive medication, was 40% and 51% for men and women respectively. The mean systolic BP of Indian men was 128 mmHg and that of Indian women 126 mmHg (Department of Health, Medical Research Council & OrcMacro, 2007).

In the study of the burden of disease attributed to high BP in adults aged 30 years and older, it was estimated that, in the year 2000, almost 47 000 deaths in South Africa were attributed to high BP, accounting for 9% of deaths and 2.4% of total DALYs, with high BP being the second leading risk factor in terms of death (Norman *et al.,* 2006). The results of this study endorsed the fact that high BP contributes significantly to the burden of disease in South Africa.

The study conducted by Morar *et al.* (1998) at the University of KwaZulu-Natal in Durban compared the BP profile of Indian and black students. It was found that the risk factors leading to CHD were more common in Indians than in black participants. Those with borderline hypertension (BP ≥130/85 and ≤140/90 mmHg) had statistically

significantly higher serum triglycerides and left ventricular mass than normotensives. The study concluded that metabolic risk factors for CHD in Indian people are apparent at an early age and thus the need to prevent risk factors leading to CHD at an early age was imperative (Morar *et al.,* 1998).

Gaziano (2006) argued that the 2006 South African Hypertension (SAH) Guidelines are too imprecise to assess cardiovascular risk, and that patients at high risk may be denied treatment and patients at low risk may be treated inappropriately. Rayner *et al.* (2007) responded to these criticisms by stating that the SAH risk charts are only guidelines which must be used in conjunction with good clinical judgement, assessment of target organ damage and recognition of associated conditions. Lifestyle interventions are critically important and are the first step in the treatment algorithm, regardless of CVD risk (Rayner *et al.,* 2007).

According to recent projections, by the year 2020, 6.4 million deaths due to CVD could occur in developing countries in the age group of 30-69 years (Reddy & Katan, 2004). This disability in middle age has major social and economic consequences owing to the severity of its complications and the frequency of non- or under-diagnosis. It is imperative that the fight against arterial hypertension should feature prominently in policy development (Seedat & Rosenthal, 2006).

Of the many risk factors related to high BP, the dietary exposure that has been most investigated is daily sodium intake (Law *et al.,* 1991; Gibbs *et al.*, 2000). Limitation of dietary sodium intake to meet these goals should be achieved by restricting daily salt intake to less than 5g per day (WHO, 2003b). Numerous clinical intervention trials, conducted to evaluate the effects of dietary salt reduction on BP levels, have been reviewed thoroughly (Midgley *et al.,* 1996; Cutler *et al.,* 1997). Based on an overview of 32 methodologically adequate trials, Cutler *et al.* (1997) concluded that a daily reduction of sodium intake by 70-80 mmol was associated with a lowering of BP both in hypertensive and normotensive individuals, with systolic and diastolic BP reductions of 4.8/1.9 mmHg in the former and 2.5/1.1 mmHg in the latter. The current guideline for South Africans is to reduce sodium intake to no more than 100 mmol per day (2.4 g sodium or 6 g sodium chloride/salt) (Charlton & Jooste, 2001).

As high BP contributes to a substantial burden of CVD in South Africa, the results discussed above indicate that there is considerable potential for health gain from implementing BP-lowering interventions that are known to be highly cost effective.

2.5.2.2 High blood cholesterol and other lipid abnormalities

2.5.2.2.1 Introduction

In a statistical release in 2006, diseases of the circulatory system accounted for 13.7% of all deaths and were ranked as the third leading cause of death in South Africa in 2006 (Stats SA, 2006). The study on the burden of disease attributable to high cholesterol in adults over 30 years showed that about 59% of IHD and 29% of the ischaemic stroke burden in adult males and females were attributed to high LDL (≥ 3.8 mmol/L), with marked variations by population group (Norman *et al.,* 2006). This study concluded that high cholesterol is an important cardiovascular risk factor in all population groups in South Africa. Furthermore, in terms of mortality rates attributable to elevated serum cholesterol concentrations, the highest prevalence was found amongst the Indian population, where it accounted for 22.2% of all deaths, followed by the white population, where it accounted for 20.5% of all deaths in 2000 (Norman *et al.,* 2006).

2.5.2.2.2 Desirable lipoprotein profile

A desirable lipoprotein profile is a total serum cholesterol concentration of less than 5.18 mmol/L, LDL-cholesterol less than 3.37 mmol/L, HDL-cholesterol greater than 1.0 mmol/L and triglyceride level less than 3.89 mmol/L (NCEP, 2002). LDL concentrations are classified as follows: optimal <2.59 mmol/L, near or above optimal 2.59 to 3.34 mmol/L, borderline high 3.36 to 4.11 mmol/L, high 4.14 to 4.89 mmol/L and very high >4.91 mmol/L (NCEP/ATPIII, 2001). Although drug therapy is often prescribed for those at moderate to high risk, dietary changes are recommended across the board. The strongest dietary determinants of elevated LDL-cholesterol concentration are dietary SFA and trans-fatty acid intakes. Trans-fatty acids increase LDL-cholesterol concentration slightly less than do SFAs but SFAs increase HDL-cholesterol concentration (Lichtenstein *et al.,* 1999). Elevated triglyceride concentrations are now

recognised as an independent risk factor for CHD (Mahan & Escott-Stump, 2008). Hypertriglyceridaemia is most common in MetS. Because of their roles in metabolism, triglyceride and HDL-cholesterol concentrations are inversely related. Because of large biological variability (<20%) in triglyceride measurements, a single sample analysed for blood triglyceride may not reflect true concentrations. Factors that increase triglyceride concentrations include diet (excessively low-fat, high in refined carbohydrate), oestrogens, alcohol, obesity, untreated diabetes, untreated hypothyroidism, chronic renal disease and liver disease (Mahan & Escott-Stump, 2008).

2.5.2.2.3 Dietary fats and CVD

The relationship between dietary fats and CVD has been extensively investigated, with consistent associations emerging from a wide body of evidence accrued from animal experiments, observational studies, clinical trials and metabolic studies in diverse human populations (Kris-Etherton *et al.,* 2001). Convincing associations for reduced risk for CVD include the consumption of fruits and vegetables, fish and fish oils (EPA and DHA), foods high in LA and potassium, increased physical activity and low to moderate alcohol intake. Furthermore, there is convincing evidence that myristic and palmitic acid, trans-fatty acids, high sodium intake and overweight contribute to an increase in risk (Joint WHO/FAO Expert Consultation, 2003b).

As mentioned previously, SFAs raise total and LDL-cholesterol, but individual fatty acids within this group have different effects, such as myristic and palmitic acids which account for the greatest effect (Mensink & Katan, 1992; Grundy & Vega, 1998). The most effective substitutions for SFAs in terms of CHD outcome are PUFAs, especially LA. This finding is supported by the results of several large randomised clinical trials, in which substitution of saturated and trans-fatty acids by polyunsaturated vegetable oils lowered CHD risk (Hu *et al.*, 1997). The traditional target is to restrict the intake of SFAs to less than 10% of daily energy intake and less than 7% for high-risk groups.

When MUFAs and n-6 PUFAs are substituted for SFAs in metabolic studies, they both lower plasma total and LDL-cholesterol concentrations (Kris-Etherton, 1999). As mentioned before, the only nutritionally important MUFA is oleic acid, which is abundant in olive and canola oil and in nuts, while the most important PUFA is LA, which is

abundant especially in soybean and sunflower oils. The most important n-3 PUFAs are EPA and DHA, found in fatty fish, and ALA found in plant foods. The biological effects of n-3 PUFAs are wide-ranging, involving lipids and lipoproteins, BP, cardiac function, arterial compliance, endothelial function, vascular reactivity and cardiac electrophysiology, as well as potent antiplatelet and anti-inflammatory effects (Mori, 2001). The long-chain n-3 PUFAs (EPA and DHA) powerfully lower serum triglycerides but they raise serum LDL-cholesterol (as indicated in Table 2.6). Therefore, their effect on CHD is probably mediated through pathways other than serum cholesterol (WHO, 2003). Diets should provide an adequate intake of PUFAs, i.e. in the range of 6-10% of daily energy intake. The suggestion of an optimal balance between intake of n-6 PUFAs and n-3 PUFAs, i.e. 5:1 to 10:1 (WHO, 2003b) is currently debated and has been discussed in more detail in section 2.5.1.2. Intake of oleic acid should make up the rest of the daily energy intake from fats, to give a daily total fat intake ranging from 15% up to 30% of daily energy intake (WHO, 2003b).

Similarly, another strategy to achieve this above-mentioned recommendation is to replace foods that are high in SFAs with those that are higher in unsaturated fatty acids. Canola oil has the potential to help consumers in this way because it has the lowest concentration of SFAs (US Department of Agriculture, 2005). Canola oil is composed predominantly of MUFAs and is the richest source of the n-3 fatty acid, ALA (Johnson *et al.*, 2007). Johnson *et al.* (2007) examined the effect on fatty acid intakes among US adults of substituting canola oil for selected vegetable oils and canola oil-based margarine for other spreads. Their theoretical model demonstrated increased compliance with dietary recommendations for SFAs, MUFAs and ALA. However, Nettleton (2007) pointed out that "to achieve dietary recommendations (for SFA as well as cardio protective foods such as whole grains, nuts, fish, fruits and vegetables), focus needs to extend beyond replacement of one oil by another." Emphasis on multiple aspects of dietary intake and dietary patterns is needed to deliver tangible public health messages, improve quality of life and reduce CHD morbidity and mortality.

Trans-fatty acids are geometrical isomers of cis-unsaturated fatty acids that adopt a SFA-like configuration. Metabolic studies have demonstrated that trans-fatty acids render the plasma lipid profile even more atherogenic than SFAs, by not only elevating LDL-cholesterol to similar levels but also decreasing HDL-cholesterol (Katan, 2000). To

promote cardiovascular health, diets should provide a very low intake of trans-fatty acids (hydrogenated oils and fats). In practice, this implies an intake of less than 1% of daily energy intake. This recommendation is especially relevant in developing countries where low-cost hydrogenated fat is frequently consumed (Katan, 2000).

Cholesterol in the blood and tissues originates from two sources, namely diet and endogenous synthesis with dairy fat and meat, the major dietary sources. Even though dietary cholesterol raises plasma cholesterol levels (Hopkins, 1992), observational evidence for an association of dietary cholesterol intake with CVD is contradictory (Hu *et al.*, 1999a). There is no requirement for dietary cholesterol and it is advisable to keep the intake as low as possible (Kris-Etherton *et al.,* 2001), at not more than 300 mg per day (WHO, 2003b).

Dietary plant sterols, especially sitostanol (2 to 3 g per day), reduce serum total cholesterol and LDL levels by as much as 14% by inhibiting cholesterol absorption (Miettinen *et al.,* 1995; Law, 2000). Commercial products made of these compounds are widely available, but their long-term effects and safety remain to be seen (Van Horn *et al.,* 2008).

2.5.2.2.4 Dietary fibre, fruit, vegetables, soy, alcohol, nuts and coffee and CVD

Most dietary fibres decrease plasma total and LDL-cholesterol, as reported by several trials (Anderson & Hanna, 1999). Numerous large cohort studies conducted in different countries have reported that a high-fibre diet (>25 g per day), as well as a diet high in wholegrain cereals, lowers the risk of CHD (Pietinen *et al.,* 1996a; Rimm *et al.,* 1996; Liu *et al.,* 1999; Truswell, 2002). Soluble fibre appears to have greater LDL-lowering potential than insoluble fibre but high total fibre remains inversely related to CHD (Jenkins *et al.*, 2002).

Although the consumption of fruits and vegetables has been widely believed to promote good health, evidence related to their protective effect against CVD has been presented only in recent years (Ness & Powles, 1997). Several ecological and prospective studies have reported a significant protective association for CHD and stroke with consumption of fruits and vegetables (Gilman *et al.,* 1995; Ness & Powles, 1997; Joshipura *et al.,*

1999; Liu *et al.*, 2000). Daily intake of fresh fruit and vegetables (including berries, green leafy and cruciferous vegetables and legumes) in an adequate quantity (400-500 g per day), is recommended to reduce the risk of CHD, stroke and high BP (WHO, 2003b; Love & Sayed, 2001). It has been estimated that low fruit and vegetable intake accounted for 3.2% of total deaths in South Africa in the year 2000 and 1.1% of the 16.2 million attributable DALYs. For both men and women, the largest proportion of total years of healthy life lost attributed to low fruit and vegetable intake (mean intake 235 g/d for men and 226 g/d for women) was for IHD (60.6% and 52%, respectively) (Schneider *et al.*, 2007).

Numerous trials point out that soy has a beneficial effect on plasma lipids (Crouse *et al.,* 1999). A composite analysis of 38 clinical trials found that an average consumption of 47 g of soy protein a day led to a 9% decline in total cholesterol and a 13% decline in LDL-cholesterol in subjects free of CHD (Anderson *et al.,* 1999). In 1999, the Food and Drug Administration (FDA) issued a health claim stating that 25 g soy protein per day was associated with reduced risk of CHD. The current research, however, does not support the earlier findings that were the basis for the health claim (van Horn *et al.*, 2008). Soy foods can be used as a plant protein substitute for animal protein foods high in SFA but the expected lowering of LDL-cholesterol may be modest and most likely in patients with hypercholesterolaemia (van Horn *et al.*, 2008). Soy is rich in isoflavones, compounds that are structurally and functionally similar to oestrogen. Several animal experiments suggest that the intake of these isoflavones may provide protection against CHD, but human data on efficacy and safety are still awaited (WHO, 2003b).

There is convincing evidence that low to moderate alcohol consumption (one to two alcoholic beverages per day) lowers the risk of CHD. In a systematic review of ecological, case-control and cohort studies in which specific associations were available between risk of CHD and consumption of beer, wine and spirits, it was found that all alcoholic drinks are linked with lower risk (Rimm *et al.,* 1999). It seems as if the effect of moderate alcohol consumption on protection against CHD is mediated through increased HDL-cholesterol and inhibition of oxidation of LDL-cholesterol (Agarwal, 2002). However, other health risks associated with alcohol (including hypertension and raised serum triglycerides) do not favour the consumption of alcohol. For those who elect to consume alcohol, moderation is recommended (van Horn *et al.*, 2008). This

recommendation is congruent with the food-based dietary guidelines for South Africans (Van Heerden & Parry, 2001).

Observational studies reported that nut consumption is associated with a reduced CHD risk (Hu *et al.*, 1999b). The unique nutrient profile of nuts may be responsible for this association. Nuts are high in unsaturated fatty acids and low in SFA. Some nuts, like walnuts, are high in ALA. Nuts are also a source of vegetable protein and plant sterols. Because of their nutrient profile, nuts may affect lipoprotein levels favourably. Furthermore, nuts may displace foods high in SFA and cholesterol (Van Horn *et al.*, 2008).

Boiled, unfiltered coffee raises total and LDL-cholesterol because coffee beans contain a terpenoid lipid called cafestol. Intake of large amounts of unfiltered coffee raises serum cholesterol and has been associated with CHD in Norway (Tverdal *et al.,* 1990). Subsequently, a shift from unfiltered, boiled coffee to filtered coffee has contributed significantly to the decline in serum cholesterol in Finland (Pietinen *et al.,* 1996).

2.5.2.3 Obesity

2.5.2.3.1 Introduction

Historically, obesity was associated only with more affluent countries. However, with the epidemiological transition, the prevalence of obesity is now competing with traditional public health concerns in the developing countries as a significant contributor to ill-health (WHO, 1998). Global trends in diet with increased consumption of refined foods, high intake of sugar and fat, coupled with physical inactivity are primarily responsible for obesity and its co-morbidities. It is widely acknowledged that excess body weight is associated with the increased risk of many diseases, such as hypertension, type 2 diabetes and CHD, which lead to serious health consequences (National Institutes of Health, 1998).

2.5.2.3.2 Prevalence of obesity

Obesity is a growing phenomenon in developing countries. In South Africa, 58% of women were overweight or obese in the 2003 SADHS (Department of Health, Medical Research Council & OrcMacro, 2007). In a sample of 4481 South African women aged 15 years and above, black women had the highest prevalence of overweight and obesity (60.9%), followed by Indian women (59.2%), women of mixed ancestry (51.2%) and then white women (48%). In a sample of 3275 South African men aged 15 years and older, the prevalence of overweight and obesity was highest in white men (48.1%), followed by Indian (44.3%) and men of mixed ancestry (36.2%), with the lowest prevalence in African men (27.2%) (Department of Health, Medical Research Council & OrcMacro, 2007).

2.5.2.3.3 Health implications

A study on the overall burden of disease attributable to body weight was conducted in South Africa in 2000 (Joubert *et al.,* 2007a). It was found that 87% of type 2 diabetes, 68% of hypertensive disease, 61% of endometrial cancer, 45% of ischaemic stroke, 38% of IHD, 31% of kidney cancer, 24% of osteoarthritis, 17% of colon cancer, and 13% of postmenopausal breast cancer was attributable to a BMI ≥ 21 kg/m². Excess body weight was estimated to have caused 36 504 deaths or 7% of all deaths in 2000, and 462 338 DALYs or 2.9% of all DALYs. The burden in women was approximately double that in men. The study confirmed the importance of recognising excess body weight as a major risk to health, particularly among women, highlighting the need to develop, implement and evaluate comprehensive interventions to achieve lasting change in the determinants and impact of excess body weight (Joubert *et al.,* 2007a).

A large number of clinical problems are associated with being obese. These can be categorised into those associated with excess adipose tissue, such as osteoarthritis, and those associated with the metabolic effects of the increased adiposity (Bray, 2004), such as CHD (Jooste *et al.,* 1988; Manson *et al.,* 1995), hypertension (Steyn *et al.*, 1990), type 2 diabetes mellitus (Lew, 1985; Pi-Sunyer, 1991; Residori *et al.*, 2003) and

certain types of cancer (Garti, 1985; Despres, 2001; NCEP, 2004). The metabolic effects of excess adipose tissue are exerted via increased release of free fatty acids (FFA) and the production of adipose-derived factors, known as adipokines, which have specific functions, all of which are not yet fully understood (Lovejoy *et al.,* 2001). Adiponectin has insulin-sensitising and antiatherogenic properties and may mediate some of its cardioprotective effects through its anti-inflammatory effects (Berg & Scherer, 2005). Havel (2004) hypothesises that adiponectin is regulated by adipocyte size and that a substantial reduction in adipocyte size is required to increase adiponectin production and circulating levels.

The distribution of the adipose tissue influences its metabolism and thereby disease risk, independently of the effects of the size of the adipose tissue stores. Accumulation of fat in the abdominal area, particularly in the visceral fat compartment, is associated with increased risk of insulin resistance, diabetes, hypertension, dyslipidaemias and atherosclerosis (Lovejoy *et al.,* 2001). Visceral adiposity is therefore the cornerstone of the MetS (Conway, 1995). Obesity and type 2 diabetes are closely associated in men and women of all ethnic groups (Chan *et al.,* 1994; Colditz *et al.,* 1995). The risk of type 2 diabetes increases with the extent and duration of overweight and the degree of central adiposity (Bray, 2004). Obesity is associated with an increased risk of CHD with a relative risk of approximately 2.8 and 3.4 for men and women respectively (Willett *et al.,* 1999). Obesity is an independent risk factor for CVD (Rashid *et al.,* 2003). Excess body weight adversely affects CVD risk factors, e.g. increased LDL-cholesterol levels, triglyceride levels, BP and blood glucose levels and reduced HDL-cholesterol levels, and increased risk of developing heart failure, CHD and stroke. The causes of obesity are multifactorial with large portion sizes, high intake of energy-dense foods and sedentary lifestyles being the main determinants (Rashid *et al.*, 2003). In a study investigating the effects of socio-economic and behavioural characteristics in explaining waist-hip ratio (WHR), it was revealed that occupation, housing, marital status, smoking condition, physical exercise, drinking habits and diet patterns cumulatively explain the total variation of the WHR in the study population (Ghosh, 2006b).

This apparent disease risk associated with the different adipose tissue deposits has important implications for South Africa, a country of diverse ethnic origin. Differences in regional fat distribution have previously been reported in American women, with African-

American women presenting with less visceral fat than do Caucasian women (Conway *et al.,* 1995; Perry *et al.,* 2000; Lovejoy *et al.,* 2001). It is well known that Asian Indians have a smaller build and excess body fat with predominant adiposity as compared with Caucasians. They tend to accumulate intra-abdominal or visceral fat without developing generalised obesity (WHO/IASO/IOTF, 2000; Ghosh *et al.,* 2004). As it has been established that body fat and BMI are different among different ethnic groups, BMI cut-offs should, therefore, be population-specific (Smalberger, 2008).

2.5.2.3.4 Criteria for diagnosis of obesity

When prevalence data and other statistics related to obesity are presented, the criteria used to obtain such information must be defined (Hubbard, 2000), as the definitions of overweight and obesity in adults have varied over time and from one study to another (Kuczmarski & Flegal, 2000). BMI (in kg/m²) is widely recognised as a weight-for-height index that has a high correlation with adiposity, but it does not quantify total body adiposity or convey information concerning regional fat distribution. However, as reviewed by Kuczmarski and Flegal (2000), it is an easily obtained measure that has been recommended for all age groups. Hubbard (2000) argues that although it may be useful to use similar criteria when comparing data from different studies and countries, a given BMI may vary when a specific population is observed and BMI cut-off points should be considered as a guide when assessing different populations. This has also been supported by a report co-sponsored by the WHO Western Pacific Region where increases in health-related risk factors were associated with a lower BMI in Asian populations (WHO/IASO/IOTF, 2000). Lower cut-off points for Asians were identified for overweight (BMI> 23.0) and obesity (BMI >25.0) (WHO/IASO/IOTF, 2000). Hubbard states that an extension of the rationale used in developing this regional WHO report could be applied to subpopulations within other countries (Hubbard, 2000).

Based on the premise that most studies relied predominantly on BMI to assess the association of adiposity with the risk of death and few studies probed whether the distribution of body fat contributes to the prediction of death, Pischon *et al.* (2008) set out to research the latter. Their data suggested that both general adiposity and abdominal adiposity are associated with risk of death, and support the use of WC or WHR in addition to BMI in assessing the risk of death, particularly in people with low

BMI. Abdominal obesity is more closely associated with the risk of several chronic diseases than is gluteofemoral obesity and large studies have suggested that WC or WHR as indicators of abdominal obesity may be a better predictor of the risk of disease than BMI (Expert Panel on the Identification, Evaluation and Treatment of Overweight and Obesity in Adults, 1998; Haslam & James, 2005; Wang *et al.,* 2005; Yusuf *et al.,* 2005). Adipose tissue, particularly from visceral fat deposits, secretes potential mediators in the development of chronic diseases (Haslam & James, 2005). This process may explain why abdominal fat distribution is related to the risk of death independently of BMI. Body mass is more closely related to the amount of visceral fat in men than in women (Krotkiewski *et al.,* 1983).

Kaur *et al.* (2008) assessed the association of four obesity-related indices, BMI, WC, WHR and waist-to-stature ratio (WSR), with hypertension and type 2 diabetes. In their study sample, WHR was the best predictor for type 2 diabetes. BMI and WC were good predictors for hypertension. The principal recommendation emerging from their findings was that WHR should be used routinely in a clinical setting together with BMI to detect persons with high risk (Kaur *et al.,* 2008). According to Graham *et al.,* 2007), there is no solid evidence for the superiority of BMI, WC or WHR in the prediction of cardiovascular risk. WC has the advantage of simplicity, may be a slightly better estimator of risk than BMI, but is probably more prone to measurement error.

2.5.2.3.5 Determinants of obesity

In order to understand the serious ramifications of obesity it is important to unpack the underlying mechanisms and determinants of the condition. Firstly, there is a massive body of evidence linking an adverse intra-uterine environment to the development of chronic disease later in life (Barker & Fall, 1993). There is now indisputable evidence, based on large numbers of epidemiological studies conducted in both developing and developed countries, that small size at birth in full-term pregnancies is linked with the subsequent "programming" of the major features of the MetS, i.e. glucose intolerance, increased BP, dyslipidaemia and increased mortality from CVD (Bouchard, 1990; Barker *et al.,* 1993; Bouchard, 1994; Rankinen *et al.,* 2002). Subsequent to this, Bouchard (1993) reported that approximately 75% of the variation in body fat

percentage and total fat mass is determined by culture and lifestyle, whereas 25% can be attributed to genetic factors.

Even though excessive energy intake is responsible for the development of obesity, high-fat diets promote fat accumulation significantly more than high-carbohydrate diets do because of the high energy density, metabolic efficiency, palatability, poor regulation and weak satiating effect of fat (Prentice, 1998). This scenario is especially relevant in South Africa where increased urbanisation is associated with the shift from a traditional to a more westernised diet. Future research should focus on intervention strategies aimed at reducing obesity and its morbid sequelae. These strategies should be culturally specific, with a particular focus on children and women, and should include dietary and behavioural aspects, as well as interactions with pharmacotherapy (MRC, 2004).

2.5.3 Disease conditions

2.5.3.1 Diabetes Mellitus

2.5.3.1.1 Introduction and prevalence

Diabetes is a common condition and its frequency is rising dramatically all over the world. The WHO estimated that in 1998 there were 135 million people with diabetes (King *et al.,* 1998). The estimate rose to 171 million in 2000 and has been projected to increase to 366 million in 2030 (Wild *et al.,* 2004). Diabetes is a common cause of non-traumatic amputations, the leading cause of blindness and accounts for a significant proportion of end-stage renal disease requiring dialysis and transplantation (Bradshaw *et al.,* 2007a). No national prevalence statistics for diabetes are available for South Africa. However, a number of epidemiological studies were conducted in selected communities in the 1980s/1990s (Levitt *et al.,* 1993; Omar *et al.,* 1994; Motala *et al.,* 2003). These reveal a clear rural-urban gradient with higher prevalence in urban settings and a varied gradient amongst different population groups. Studies have reported that the highest prevalence is in the Indian population followed by the coloured and black populations (Bradshaw *et al.*, 2007a). The study from the Burden of Disease Research Unit of the MRC used the comparative risk assessment (CRA) methodology

and the revised WHO criterion of a fasting venous plasma glucose concentration of >7 and/or ≥11.1 mmol/L taken two hours after a 75 g oral glucose challenge to define the presence of diabetes (WHO, 1985). The findings of this study showed that the prevalence of diabetes varied by age, sex and population group, with Indians having the highest prevalence of diabetes. In the respective age groups of 30 - 44 and 45 - 59 years, 9.7% and 21.7% of Indians had diabetes, with a greater prevalence in men (Bradshaw *et al.,* 2007a).

2.5.3.1.2 Management of diabetes

Mann (2006) has suggested that it is often necessary to base recommendations for the management of diabetes on surrogate end points such as glycaemia, body composition, lipoprotein profile, BP, insulin sensitivity and renal function. There is ample evidence for recommendations on reduction of energy intake and the increase of energy expenditure in terms of diabetes control (Mann, 2006). Insulin sensitivity is reduced and most of the metabolic abnormalities associated with diabetes are exaggerated in those that are overweight (Astrup *et al.,* 2002). According to Goldstein (1992), modest weight loss improves insulin sensitivity and glucose tolerance and reduces lipid levels and BP. Effects of fatty acids on insulin resistance and increased insulin sensitivity are noted when SFAs are replaced by cis-unsaturated fatty acids (Perez-Jimenez *et al.*, 2001; Vessby *et al.,* 2001; Summers *et al.,* 2002). With respect to carbohydrates, it is now apparent that the quality rather than the quantity of carbohydrate really matters, with vegetables, legumes, fruits and whole grain cereals being the most suitable sources (especially those with a low glycaemic index) (Mann, 2001). Soluble dietary fibre increases glycaemic control, reduces levels of total and LDL-cholesterol (Mann, 2001) and increases levels of HDL-cholesterol (Toeller *et al.,* 1999). Studies have shown that an increased risk of developing diabetes is prevalent amongst those that have a high proportion of SFAs in their plasma lipid esters, compatible with a high dietary intake of saturated fat (Vessby *et al.,* 1994). Likewise, people who exercise regularly and are not overweight (Manson *et al.*, 1991) and who consume a high proportion of LA (Laaksonen, 2002) have a reduced risk of developing diabetes. Increasing the consumption of n-3 fatty acids improves several cardiovascular risk factors in persons with diabetes and may reduce the risk of conversion from impaired glucose tolerance to type 2 diabetes (Kesavulu *et al.,* 2002; Kris-Etherton *et al.,* 2002). Several studies have

shown that lifestyle intervention involving dietary modification and increased physical activity reduces the risk of progression of impaired glucose tolerance to type 2 diabetes (Knowler *et al.,* 2002; McAuley *et al.*, 2002).

The study conducted by Ghosh (2006a) investigated anthropometric, metabolic and dietary fatty acid profiles in lean and obese Indian diabetic patients. The findings of the study showed that central obesity measures were significantly associated with dietary fatty acids and their ratio. Furthermore, the ratios of unsaturated fatty acids to SFAs were significantly negatively associated with lipids, lipoproteins (except HDL) and fasting glucose. He concluded that dietary management including dietary guidelines would be useful to retard the growing incidence of diabetes in the Indian population. In addition, Ghosh (2007) continued in another study to compare obesity measures, metabolic profiles and dietary fatty acids in lean and obese dyslipidaemic Asian Indian men and concluded that it was rational to argue that while dealing with dyslipidaemic Asian Indians, clinicians should consider metabolic profiles and dietary fatty acids simultaneously to better comprehend the condition.

With the overbearing prevalence of diabetes which deeply impacts on the quality of life, lifestyle modification can be seen as the cornerstone of both treatment and attempts to prevent type 2 diabetes (Mann, 2000).

2.5.3.2 Metabolic syndrome (MetS)

2.5.3.2.1 Introduction

The metabolic syndrome (MetS) and its components has become a serious problem worldwide and is a gateway to a host of chronic diseases. The MetS describes a cluster of risk factors related to CVD in which abdominal obesity, high BP, high blood glucose and abnormal lipid profile (low HDL-cholesterol, high triglycerides) cluster together in a person. The NCEP/ATP111, (NCEP/ATP111, 2001) recommends that three out of five clinical and or biochemical abnormalities should be present to justify the labelling.

2.5.3.2.2 Prevalence of MetS in Indians

There have been a number of studies on the prevalence of MetS in Indians living in India (Ramachandra *et al.,* 1998; Misra *et al.,* 2005) and abroad (Hughes *et al.,* 1997; Tan *et al.,* 2004; Shah *et al.,* 2005) as they have been considered to be a high-risk group for the MetS. In addition, several studies have been conducted worldwide on the prevalence of MetS across different ethnic groups (Misra *et al.,* 2002; Al-Lawati *et al.,* 2003; Yong-Woo, 2003; Kuninori *et al.,* 2005).

MetS is common in Asian Indians (Das, 2003; Ramchandran *et al.,* 2003; Dwivedi *et al.,* 2004). Asian Indians have long been considered to be population at high risk for both MetS and CVD. The prevalence of MetS has been estimated to be between 28.8% and 41.1% in Asian Indian adults when using the NCEP/ATPIII criteria with modified WC criteria appropriate for Asian Indians [≥ 80 cm in women and ≥ 90 cm in men (Tan *et al.,* 2004); ≥ 85 cm in women and ≥ 90 cm in men (Ramachandran *et al.,* 2003)].

2.5.3.2.3 Definitions of MetS

The NCEP/ATP111 (2001) and WHO (2004b) and more recently the International Diabetes Federation (IDF, 2005) have laid down guidelines to define MetS, and these have been applied to a number of studies. Insulin resistance is the pivotal feature in the WHO criteria. WC rather than BMI has been the differentiating aspect of the MetS definition by the ATP111 panel and IDF.

It has long been argued that the standard definitions of MetS under-represent the prevalence of MetS in the Asian Indian community, thereby delaying the commencement of definite preventive and therapeutic efforts in many individuals. The results of various studies have acknowledged ethnic variation in clinical measures and disease outcomes in different populations (Anand *et al.,* 2000; Tan *et al.,* 2004). In spite of the various criteria devised for MetS diagnosis, most studies agree that the prevalence of MetS in Asian Indians is under-represented when the ATP111 or WHO criteria are used (Misra *et al.,* 2005).

Misra *et al.* (2008) investigated the applicability of the NCEP/ATP111 definition of MetS in ethnic groups other than white Caucasians. The first problem in terms of the definition was the WC cut-off points. The NCEP/ATP111 definition of WC is ≥102 cm in men (94 cm according to the International Diabetes Federation, IDF, 2005) and ≥ 88 cm in women (80 cm according to the IDF). However, the cut-off point for men is not appropriate to Asians because of their inherent abdominal obesity (McNeely *et al.,* 2001). WC morbidity correlation studies in Chinese, Taiwanese, and Asian Indians show that WC cut-off points in these ethnic groups are lower than those defined by NCEP/ATP111 (Bei-Fan, 2002; Lin *et al.*, 2002). Insulin resistance, non-esterified fatty acids and dyslipidaemia are reported to be higher in Asian Indians at a lower WC owing to higher values of body fat, intra-abdominal fat and truncal fat (Banjeri *et al.,* 1997; Raji, 2001).

Wasir *et al.* (2008) set out to derive an optimum definition of MetS for Asian Indians. They compared the NCEP/ATP111 and the IDF definitions of MetS with two proposed candidate definitions in adult Asians. Candidate definitions of MetS were proposed by modifying the NCEP/ATP111 and IDF definitions by including the following modified variables into two combinations (MetS-ATP1 and MetS-IDF1): WC cut-off point as ≥90 cm in men and ≥80 cm in women, BMI cut-off point >23 kg/m^2 and impaired fasting blood glucose as >5.55 mmol/L, with WC as an obligatory criterion. Maximum overall and gender-specific prevalence of MetS was observed using the definition which included modified cut-off points of WC (non-obligatory), BMI and impaired fasting blood glucose in addition to other defining parameters. Even in subjects without abdominal obesity, a high prevalence of other abnormal defining parameters of the MetS was seen [hypertension (SBP ≥130 or DBP ≥85 mmHG), BMI >23 kg/m^2, hypertrigyceridemia >1.69 mmol/L and low levels of HDL-cholesterol]. Wasir *et al.* (2008) concluded that making abdominal obesity a mandatory criterion would lead to missing some cases of MetS. By including BMI and making WC a non-obligatory criterion, more cases of MetS are detected. For Asian Indians, making WC a mandatory variable in the definition of MetS would result in the non-inclusion of nearly 11% of cases which would otherwise be diagnosed as MetS according to the modified NCEP/ATP111 definition.

According to Zhu (2002), WC is strongly linked to obesity-associated risks. However, currently proposed WC risk thresholds are not based on association with obesity-related

risk factors but rather with BMI. He concluded that WC is more closely linked than BMI to cardiovascular risk factors. BMI is widely used for classification of overweight and obesity (Garrow & Webber, 1985; WHO, 1997). However, BMI does not account for the wide variation in body fat distribution, the nature of obesity across different individuals and populations, and the joint relation of body composition and body size to health outcomes (WHO, 1997; Michels *et al.,* 1998). Many studies have reported that body fat distribution is a more powerful predictor than BMI for risk factors, disease and mortality (Albrink & Meigs, 1964; Kissebah *et al.,* 1982; Larsson *et al.,* 1982; Blair *et al.,* 1984; Hartz *et al.,* 1984; Stevens *et al.,* 1992; Folsom *et al.,* 1993; Pi-Sunyer, 2000). Increased visceral or abdominal adipose tissue has been shown to be more strongly associated with MetS and CVD risk and a variety of chronic diseases (Larson *et al.* 1982; Vague *et al.,* 1988; Despres *et al.,* 1990; Steven *et al.,* 1992; Bjorntorp, 1993; Folsom *et al.,* 1993; Reeder *et al.,* 1997); therefore, measurements that are more sensitive to individual differences in abdominal fat might be more useful than BMI for identification of obesity-associated adipose tissue (Ross *et al.,* 1992; Pouliot *et al.,* 1994; Han *et al.,* 1995). WC is unrelated to height (Han *et al.,* 1997). WC correlates closely with BMI (Lean *et al.,* 1995; Onat, 1999) and total body fat (Lean *et al.,* 1996), and in effect is associated with CVD risk factors independent of BMI (United States Department of Health and Human Services, 2000). WC may be an effective clinical tool in assessing the risk of CVD (Lean *et al.,* 1998; Okosun *et al.,* 2000). Because populations may differ in the level of risk associated with a particular WC, it is not advisable to identify universally applicable risk thresholds (WHO, 1997). Further to this, women have a greater relative risk of CVD at a lower WC than men (WHO, 1997). Thus, the development of cut-off points specific to gender and ethnicity is warranted (Zhu *et al.,* 2002).

In this regard, the WHO has recognised the need for a population-specific modification of anthropometric measures. It recommends lower BMI definitions for overweight in Indians (23 kg/m^2, WHO, 2004), modified WC measures (≥90 cm in men and ≥ 80cm in women, IDF, 2005), and a WHR of 0.89 for men and women (Snehalatha, 2003). These cut-off points have been applied by several investigators when studying Asian Indians (Misra *et al.,* 2005; Tan *et al.,* 2005; Tillin *et al.,* 2005; Heng *et al.,* 2006).

The study conducted by Kanjilal *et al.* (2008) aimed to estimate the prevalence of MetS in Asians using the ATP111, WHO criteria and the modified definition which included lower cut-off points for WC (≥ 90 cm for men and ≥ 80 cm for women), BMI (>23 kg/m^2) and impaired fasting glucose. They concluded that the revision of the definition criteria for MetS with lowered cut-off points for WC and BMI is critical for the accurate assessment of MetS among Asian Indians (Kanjilal *et al.,* 2008).

2.5.3.2.4 Prevention and management of MetS

There is agreement in the literature that full expression of the MetS depends on a complex interaction between genetic determinants (still largely unknown) and acquired factors related mainly to lifestyle habits. Both intensive lifestyle interventions and some drugs may reduce the prevalence of the syndrome (Giugliano *et al.*, 2008). Prevention of weight gain and obesity may be the single most effective means of preventing MetS. In theory, the ideal diet should target many, if not all, of the dietary components thought to influence cardiometabolic risk, including all types of fat (saturated, polyunsaturated, monounsaturated and trans-fats), fibre, fish, carbohydrates and proteins. Although there is no such "all-inclusive" diet yet, it seems plausible that a Mediterranean-style diet has most of the desired attributes, including a lower content of refined carbohydrates, high content of fibre, moderate content of fat (mostly unsaturated) and a moderate to high content of vegetable proteins (Giugliano *et al.*, 2008).

According to Minich and Bland (2008), tailored dietary approaches beyond macronutrient ratio modification may be necessary to address metabolic measures effectively. These authors discuss various dietary approaches for MetS with a focus on dietary phytochemicals as they are known to affect insulin signalling. They concluded that a low-fat diet may serve as an adequate foundation upon which other dietary additions could be made. Further to this, a number of studies indicate that dietary patterns high in whole, unprocessed plant foods and abundant in phytochemicals may have benefits for MetS (Sonnenberg *et al.,* 2005; Baxter *et al.*, 2006; Esmaillzadeh *et al.*, 2007; Panagiotakos *et al.*, 2007). A survey of emerging literature indicates that the dietary approach may ultimately involve a diet high in phytochemicals that favourably target kinases involved in cellular signalling. The epidemiological evidence suggests that a lower prevalence of MetS is associated with dietary patterns rich in fruit,

vegetables, whole grains, dairy products and unsaturated fats (Baxter *et al.*, 2006). It was concluded from this review that no individual dietary component seems wholly responsible for the association of diet with the MetS, but rather that it is the quality of the diet that confers the most protection against the MetS (Minich & Bland, 2008). In terms of intervention, in addition to diet, physical activity has proved to be a useful lifestyle modification for MetS.

2.5.4 Clustering of risk markers

The clustering of health-related behaviours is a well described phenomenon that is clearly evident in early childhood. Impaired glucose tolerance and adverse lipid profile seen in childhood are clustered together with high BP and this relates strongly to obesity (Berenson *et al.*, 1991; Raitakari *et al.,* 1996; Twisk *et al.,* 1999; Tan *et al.,* 2000). Intergenerational effects, in terms of maternal birth size (Ramakrishnan *et al.,* 1999), poor growth (UNICEF, 1998) and obesity (WHO, 2004a) are also key determinants in the interaction between early and later factors throughout the life course. In terms of gene-nutrient interactions and genetic susceptibility, there is good evidence that nutrients and physical activity influence gene expression and have shaped the genome over several million years of human evolution (WHO, 2003b). Genes define opportunities for health and susceptibility to disease, while environmental factors will determine which susceptible individuals will develop diseases (WHO, 2003b). There is a vast volume of scientific evidence highlighting the importance of applying the life course approach to the prevention and control of chronic disease, subscribing to the intergenerational approach of prevention throughout the lifespan.

The overall Birth-to-Ten (BTT) project in South Africa (Steyn *et al.*, 2000) collected antenatal, birth and early development information on children, as well as information that could help identify factors related to the emergence of CVD (Yach *et al.,* 1990). In this BTT project, the LDL-cholesterol level of Indian children was significantly higher than that of blacks. The study suggests that the promotion of a healthy lifestyle should start in childhood and should target the risk factors found in each group (Steyn *et al.,* 2000). Similarly, the Barker hypothesis suggests that disturbed intra-uterine growth has a negative influence on the development of cardiovascular systems and favours the occurrence of hypertension, diabetes mellitus, insulin resistance, hypercholesterolaemia

and hyperuricaemia in adult life (Barker & Fall, 1993). Much attention has been given to interactions between early and later factors throughout the life course, among which low birth weight followed by subsequent adult obesity has been shown to impart a high risk of CHD (Frankel *et al.,* 1996; Yajnik, 2002) and diabetes (Lithell *et al.,* 1996).

2.6 SUMMARY OF THE ASIAN ENIGMA: PREDISPOSITION FOR CARDIOVASCULAR DISEASE AND DIABETES

There is an inherent difference between Asians and Caucasians with regard to risk factors for cardiovascular diseases and diabetes, with Asians having increased risk owing to increased incidence of insulin resistance and a cluster of cardiovascular risk factors such as central obesity, hyperglycaemia, dyslipidaemia and microalbuminuria. Cut-points used in this study were based on cut-points modified for Asian Indians in view of their morphological structure, as discussed under 2.5.3.2. Cut-points were based on the recommendations by the WHO for Asian populations (WHO/IASO/IOTF, 2000). Overweight and obesity were defined as >23 kg/m^2 and >25 kg/m^2 respectively. Increased WC was defined as ≥90 cm for men and ≥80 cm for women. Impaired fasting blood glucose was defined as >5.55 mmol/L. Hypertension was defined as SBP ≥130 or DBP ≥85 mmHG and hypertriglyceridaemia was defined as >1.69 mmol/L. More appropriately, Figure 2.4 succinctly illustrates the complex interactions of genetic, perinatal, nutritional and other acquired factors in the development of insulin resistance, type 2 diabetes and CHD in South Asians, which have to be taken into account for recommendations of intervention.

2.7 RECOMMENDATIONS FOR INTERVENTIONS

The huge body of knowledge amassed over the years has led to several sets of international dietary recommendations and guidelines to reduce the burden of NCDs. Selecting interventions should be based on a robust and transparent process of scientific evaluation of their effectiveness as well as assessment of their cost effectiveness, local applicability and appropriateness and likely effects on health inequalities (Lavis *et al.,* 2004; Steyn *et al.*, 2009; WHO, 2009). In order to make recommendations for interventions to reduce the risk of NCDs, it is imperative that one looks at the current global and national efforts in terms of intervention strategies.

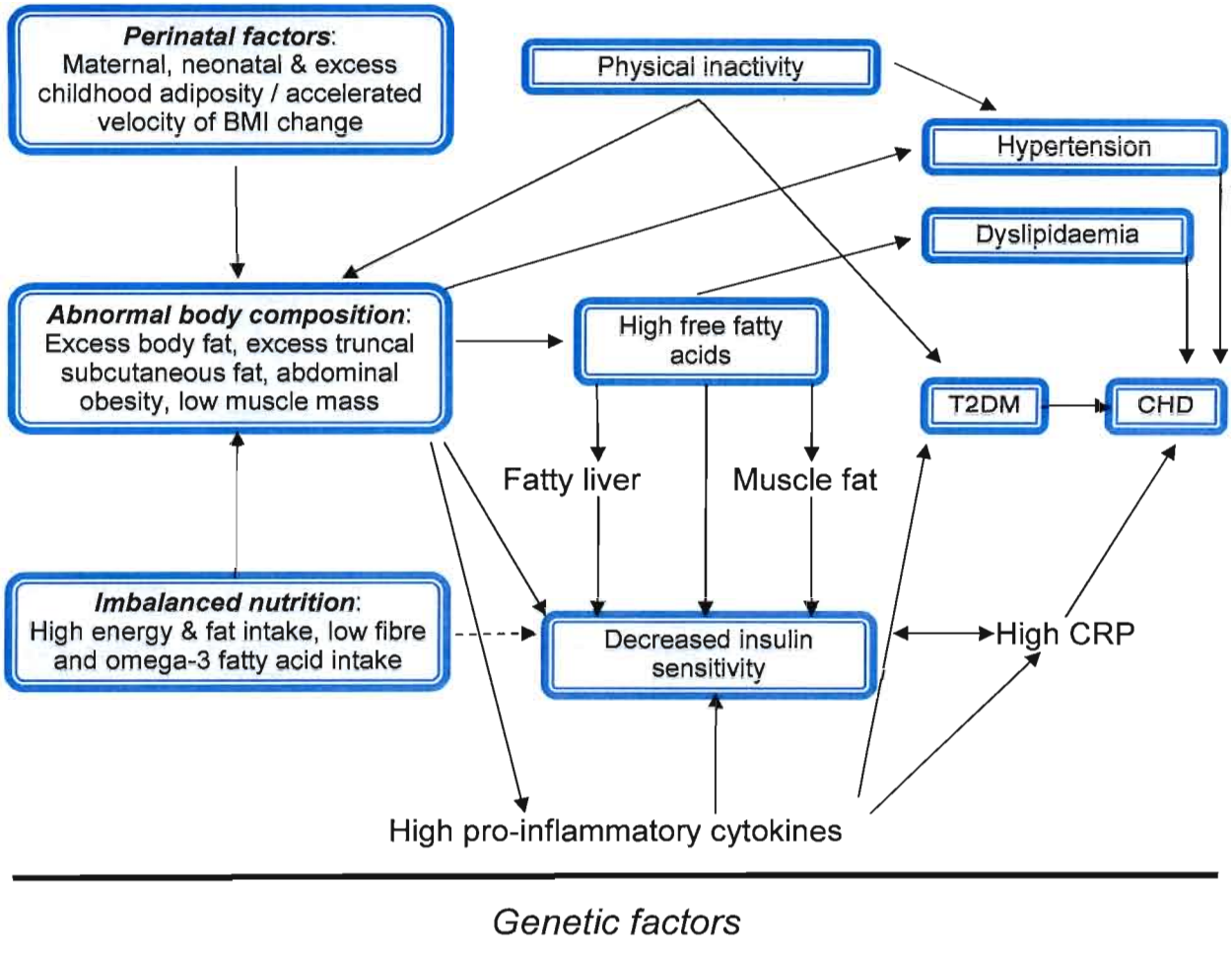

Figure 2.4: Complex interactions of genetic, perinatal, nutritional and other acquired factors in the development of insulin resistance, type 2 diabetes and coronary heart disease (CHD) in South Asians. CPR: C-reactive protein, T2DM: type 2 diabetes (Adapted from Misra *et al.*, 2007)

As noted in section 2.5.1.1, the WHO has developed population nutrient intake goals for preventing diet-related chronic diseases. These represent population average intake that is judged to be consistent with the maintenance of health in a population (refer to Table 2.4.) (WHO, 2003b). Ideally, these generic recommendations could be used as the basis for country-specific strategies and food-based dietary guidelines (WHO 2004a). Preferably, the definition of an increased risk or a decreased risk should be based on a relationship established by multiple randomised control trials of intervention that are representative of the target population. However, this type of evidence is not always available. Therefore, criteria used to describe the strength of evidence were

established. *Convincing evidence* is based on epidemiological studies showing consistent associations between exposure and disease (WHO, 2004a). *Probable evidence* is based on epidemiological studies showing fairly consistent associations between exposures and disease but with perceived shortcomings in the available evidence. *Possible evidence* is based mainly on findings from insufficient case-control and cross-sectional studies. *Insufficient evidence* is based on findings of a few studies which are suggestive but are insufficient to establish an association between exposures and disease (See Appendix A on strength of evidence).

In 2000, SACRA identified 17 risk factors based on the burden of disease experienced in South Africa (refer to section 2.2). These risk factors were expressed firstly as the burden attributable to risk factors in terms of death and secondly in terms of DALYs (Norman *et al.,* 2007a, presented in Tables 2.2 and 2.3). Recommendations for interventions were made. The recommendations were informed by international evidence but also included interventions that were considered to have a high potential or addressed a particular need of a country. Interventions were graded according whether the Cochrane review (The Cochrane Collaboration, 2007) or the Disease Control Priorities in Developing Countries (DCPP) considered them to be effective (Jamison *et al.,* 2006). The potential interventions and recommendations of SACRA are based on government, community and individual intervention methods (Bradshaw *et al.,* 2007) (see Appendix B).

The Fourth Joint Task Force of the European Society of Cardiology and other societies on cardiovascular disease prevention in clinical practice (Graham *et al.*, 2007) was commissioned to develop guidelines on CHD prevention. As a starting point, the Task Force took into account that more detailed guidance was sought by organisations. Furthermore, the approach to grading by the current European Atherosclerosis Society and the European Hypertension Society was examined in detail, as concern was expressed that the present system gave more priority to drug treatment. The group also considered recalibrating systemic coronary risk evaluation (SCORE) charts to allow for time trends in both mortality and risk factor distribution. In the present guidelines, this approach has been replaced with a simple relative risk chart to be used in conjunction with the SCORE absolute risk chart. Risk charts such as SCORE are intended to facilitate risk estimation in ostensibly healthy Europeans.

According to the AHA, improved diet and lifestyle are critical components in preventing CVD (Lichtenstein *et al.,* 2006). After much research and consultation, the AHA re-published diet and lifestyle recommendations for adults and children over two years, which are intended to reduce CVD in the American population (Lichtenstein *et al.,* 2006). Most of these guidelines correspond to the guidelines for Europeans (Graham *et al.*, 2007).

The goal of these guidelines is to assist those at low risk of CVD to maintain this state throughout life, and those at increased total CVD risk to reduce it by application of the following recommendations:

- Consume an overall healthy diet. There is an emphasis on the whole diet, rather than a specific nutrient, in order to ensure nutrient adequacy and energy balance (United States Department of Health and Human Services, 2005).
- Aim for a healthy body weight.
- Aim for a desirable lipid profile (as indicated in section 2.5.2.2).
- Aim for normal BP. A normal BP is a systolic BP <120 mmHg and a diastolic BP <80 mmHg. Elevated BP results from environmental factors (diet, physical inactivity and psychosocial factors) and genetic factors. Dietary modifications include lowering salt intake, weight loss, moderation of intake of alcohol, increasing potassium intake and an overall healthy diet.
- Aim for normal blood glucose level. A normal fasting blood glucose level is <5.55 mmol/L. However, diabetes is defined by a fasting blood glucose level of >6.99 mmol/L. It is recommended that energy intake should be reduced and physical activity increased to achieve a modest weight loss which can decrease insulin resistance and improve glucose control and metabolic abnormalities.
- Be physically active. An adult should accumulate >30 minutes of physical activity most days of the week. For individuals attempting to lose weight, 60 minutes of physical activity is recommended on most days of the week.
- Avoid use of and exposure to tobacco products.

Stemming from the AHA and the European Society of Cardiology diet and lifestyle goals for CVD reduction, the following diet and lifestyle recommendations were made by the American Dietetic Association (ADA) based on a lifestyle prescription philosophy (ADA, 2007b):

- Balance energy intake and physical activity to achieve or maintain a healthy body weight. To control kilojoule (kJ) intake, individuals should increase awareness of kJ content of foods and beverages and control portion sizes (Klein *et al.*, 2004). It is the position of the ADA that the overall pattern of food eaten is the most important focus of a healthy eating style. All foods can fit into this pattern if eaten in moderation with appropriate portion sizes, combined with regular physical activities (ADA, 2007b).
- Consume a diet rich in vegetables and fruits, whole-grain high-fibre foods and eat fish, especially oily fish, at least twice a week.
- Limit the intake of saturated fatty acids and trans-fatty acids and cholesterol.
- Minimise the intake of beverages and foods with added sugars.
- Choose and prepare foods with little or no salt.
- If alcohol is consumed, it should be done in moderation

Van Horn *et al.* (2008) provided a comprehensive and systematic review of the evidence associated with key dietary factors and the risk of cardiovascular diseases and concluded that lifestyle interventions are essential for the prevention of CVD, advocating a diet:

- low in SFA (<7%E), TFA (<1%E) and dietary cholesterol (<200 mg);
- rich in n-3 fatty acids, EPA, and DHA (500 mg/day for primary prevention; 1 g/day for secondary prevention and 2 to 4 g/day for TG-lowering; consumption of fish at least twice a week;
- ample in total dietary fibre (30 g/day) with emphasis on soluble fibre;
- that includes unsalted nuts (30 g/day) as tolerated and limited by energy needs; consider other vegetable protein sources such as soy and legumes;
- that includes skim/low-fat dairy foods and/or other calcium/vitamin D-rich sources;
- rich in vitamins, minerals, phytochemicals and antioxidants from multiple servings of fruits and vegetables and low in sodium (<2 300 mg/day);

- rich in B vitamins and fibre from food sources such as whole grains and vegetables;
- that may include plant sterols and stanols in high-risk individuals; and
- that achieves a healthy body weight and energy balance by increasing physical activity and maintaining an adequate energy intake.

With specific reference to South Africa, useful lessons can be drawn from the tobacco control policy (MRC, 2006), where legislation on advertising and distribution, restrictions on smoking in public places, increased taxation and health warnings have been employed in the interventions. Lessons from these experiences need to be used to develop strategies to influence eating habits, increase physical activity and reduce alcohol consumption. The development of the South African Food-Based Dietary Guidelines can be viewed as an important preventative strategy for NCDs in the population. This was a joint initiative between the Nutrition Society, Association for Dietetics in South Africa, Medical Research Council, industry and the Department of Health.

Some of the guidelines are particularly relevant to the prevention of chronic diseases such as CVD and type 2 diabetes, and are as follows (Vorster *et al.*, 2001):

- *Eat plenty of fruit and vegetables every day* – for their fibre, micronutrient, antioxidant and other essential properties;
- *Eat dry beans, peas, lentils and soy regularly* – for increased fibre, protein and flavonoid intake, stabilising blood glucose levels and decreasing serum cholesterol;
- *Eat fats sparingly* – to prevent a high intake of fat and saturated fats;
- *Use salt sparingly* – because of the detrimental effect of a high sodium intake on blood pressure;
- *Use food and drinks containing sugar sparingly and not between meals* – these foods have a low nutrient density;
- *If you drink alcohol, drink sensibly* – excessive alcohol consumption is a risk factor for CHD, hypertension and diabetes;
- *Be active* – an important preventative/management measure for diabetes, obesity and improved hypertension.

Communication of nutrition messages

Although there has been a large number of interventions and recommendations to reduce the risk factors for NCDs, behavioural change is problematic. The impact of nutrition information on the promotion of a healthy lifestyle depends on how effectively the nutrition messages are communicated to consumers (ADA, 2007a). Communication is more effective when the appropriate theories and models related to human behaviour are used (ADA, 2006). The following models are used in communicating nutrition messages:

- The Knowledge Attitude Belief model, based on exposure to new information. The individual attends to it and gains new knowledge by changing attitude and altering dietary patterns. This is effective only if the individual is highly motivated and the new information is easy to follow (Contento *et al.,* 1995).
- The Health Belief model is most widely used (Janz *et al.,* 2002). This model explains human behaviour and readiness to act *via* five main constructs i.e. perceived susceptibility, severity, benefits, barriers and self-efficacy.
- The Trans Theoretical model (TTM) includes a series of behaviour stages and processes of change. The TTM is currently the most popular stage model in health psychology (Horwath, 1999) and it has proved successful with a wide variety of simple and complex health behaviours, including smoking cessation, weight control, reduction of dietary fat and exercise acquisition (Prochaska *et al.,* 1994). Based on more than 15 years of research, the TTM has found that individuals move through a series of five stages (pre-contemplation, contemplation, preparation, action and maintenance) in the adoption of healthy behaviours or cessation of unhealthy ones. Pre-contemplation is the stage in which an individual has no intent to change behaviour in the near future. In the Contemplation stage, individuals openly state their intent to change within the next six months. Preparation is the stage in which individuals intend to take steps to change, usually within the next month (Prochaska *et al.,* 1992). The Action stage is one in which an individual initiates the behaviour change and the Maintenance stage involves working to prevent relapse and the consolidation of gains secured (Prochaska *et al.,* 1992).

- The Social Cognitive theory supports an educational intervention addressing behavioural capability, expectations, self-efficacy, observational learning and reinforcement, proposed by Miller and Dollard in 1941. The Social Cognitive theory is a learning theory based on the idea that people learn by watching what others do and that human thought processes are central to understanding personality (Miller & Dollard, 1941).
- The Precede-Proceed model arose out of the failure of the Health Belief model to account for environmental influences on behaviour. It is based on three sets of influences on people's behaviour, namely predisposing factors, enabling factors and reinforcing factors. Predisposing factors comprise an individual's attitudes, beliefs and values. Enabling factors include the developing of skills and reinforcing factors include support from the family, peers and community. This model proposes that health programme planning needs to occur through a series of levels such as administrative, educational, behavioural, epidemiological and social diagnosis (Green & Kreuter, 2005).

Social marketing has been used successfully to bring about voluntary behaviour change (Cairns & Stead, 2009). The authors explain that marketing uses client-focused research techniques such as segmentation of target groups. Segmentation concentrates on identifying which groups of interpersonal and external factors are most influential (positively or negatively) in behavioural change. For example, these factors may include aspirational values, real-life behaviours and real or perceived barriers to change. These findings may have little obvious link to fundamental health objectives such as reducing the fat content of the diet, but do provide the mechanism to facilitate desired change by linking to life-style choices of broad groupings of individuals. Marketing methods then use this knowledge to develop and refine genuine exchange of benefit 'offers' to the target groups. The principle of exchange is critical to the success of marketing and is based on the recognition that any voluntary change incurs costs (such as inconvenience, uncertainty of outcome), is optional and must therefore offer valued benefits (e.g. immediate outcomes, not long-term risk reduction). According to Cairns and Stead (2009), social marketing is best understood as a pragmatic framework for understanding how and why individuals make lifestyle choices and for devising and marketing desirable alternatives.

Promotion of healthy diets and physical activity

The WHO recommends the following policy principles for the promotion of healthy diets and physical activity (WHO, 2003b):

- Strategies should be comprehensive and address all major dietary and physical activity risks for chronic diseases together with other risks from a multisectoral perspective.
- Each country should select what will constitute the optimal mix of actions that are in accord with national capabilities, laws and economic realities.
- Governments have a central steering role in developing strategies, ensuring that actions are implemented, and monitoring their impact over the long term.
- Ministries of health have a crucial convening role, bringing together other ministries needed for effective policy design and implementation.
- Governments need to work together with the private sector, health professional bodies, consumer groups, academics, the research community and other nongovernmental bodies if sustained progress is to occur.
- A life-course perspective on chronic disease prevention and control is critical.
- Strategies should explicitly address equality and diminish disparities. They should focus on the needs of the poorest communities and population groups. Furthermore, since women generally make decisions about household nutrition, strategies should be gender-sensitive.
- There are limits to what individual countries can do alone to promote optimal diets and healthy living. Strategies need to draw substantially on existing international standards that provide a reference in international trade.

Based on experience in the implementation of local and national strategies, the WHO expert consultation group concluded that there are a number of prerequisites for successful intervention. These include leadership, effective communication, functioning alliances and an enabling environment. Furthermore, an effective communication strategy should focus on high priority, public health need, and adopt a proactive, positive, practical approach based on theories that promote behavioural change. Two complementary strategies are usually advocated, i.e. the population approach, where there is community-wide intervention, and the high-risk approach, where a few individuals are identified as being at high risk. They are then targeted with behavioural

and pharmacological intervention. To address the limitations of both approaches, it is suggested that the intergenerational approach of prevention should be used, incorporating both approaches throughout the lifespan, catalysing long-term changes (WHO, 2003b). Furthermore, The WHO recently published a report on tried and tested interventions with regard to diet and physical activity, presenting the evidence on effective interventions in various categories (WHO, 2009). These successful strategies have also been reviewed by Steyn *et al.* (2009).

2.8 SUMMARY AND CONCLUSION

In this chapter, the literature review was presented in the context of the risk markers, risk behaviours and disease states of NCDs, with special reference to the Indian population. The merits and demerits of n-6 and n-3 fatty acid consumption were reviewed in the context of disease states. Existing recommendations for interventions were also reviewed in this chapter with a view to adapting some recommendations in Chapter 6. In Chapter 3, the research methodology, the study design and statistical methods will be presented.

CHAPTER 3

METHODS

3.1 STUDY DESIGN

This was a cross-sectional, epidemiological study among apparently healthy Indian residents (35-55 yrs) from the KwaDukuza municipality, KZN. In the preliminary studies (n=25 respondents), the socio-demographic questionnaire was tested and recipe standardisation was conducted. The quantitative food frequency questionnaire (QFFQ) was tested for comparative validity against three 24-hour food recalls as the reference method, and for reproducibility by a second administration in a subsample of 50 respondents.

3.2 STUDY POPULATION

The Indian community of KwaDukuza municipality was selected for this study. KwaDukuza (Stanger) is approximately 100 km north of Durban and is a mainly farming community with a few light industries. The Indians located in this area originally arrived in the 1800s as indentured sugar cane labourers who were placed throughout KwaDukuza. The main reason for selection of this area for the research project was that it was representative of Indians from different categories of the Living Standards Measure (LSM). This characteristic demographic profile reflected in KwaDukuza makes it representative of all Indians living in KZN, unlike other Indian areas where most Indians in the upper LSM have relocated to more affluent areas. According to the 2001 census, there were 29 032 Indians living in the KwaDukuza municipality (Stats SA, 2001). There were 64 sub-areas, with Indians residing in 39 of those sub-areas (Stats SA, 2001). Sub-areas around Stanger comprised the main central business area (CBD) and had the largest number of Indian residents (Stats SA, 2001). For the purpose of this study, six sub-areas around Stanger were selected for the sample. The main reasons for selecting sub-areas in and around Stanger were that, firstly, they included the largest number of Indian residents in the KwaDukuza municipality (Stats SA, 2001)

and secondly, they were representative of people with different standards of living, as farming communities were located on the outskirts of the CBD. A map of KZN with special reference to Stanger is illustrated in Figure 3.1.

Figure 3.1: Map of KwaZulu-Natal
(http://www.bookinafrica.com.images/map/main/Kwazulu Natal gif)

3.3 SAMPLE

Based on power calculations (80% power to estimate the BMI of men and women accurately within two units on a 5% significance level, using BMI of Indians reported in the literature) the required sample size was 250 apparently healthy Indians. The latest records of ratepayers and electricity consumers from the valuation roll of the KwaDukuza Municipality were used for systematic random selection of household addresses. The municipality's roll provides a list of ratepayers with their addresses and contact details. The addresses were chosen at random and the occupants telephoned to identify whether anyone was eligible for participation in the study. Once eligibility was established, only one respondent at each household address was invited for participation. In the event that there was more than one household member who met the inclusion criteria, only one member was chosen from a draw by the researcher. Respondents were telephoned and informed about the study. If they were willing to participate in the study, a date was set up for the interview. However, if respondents from the chosen dwelling were unwilling to participate in the study, the adjacent house was chosen. The first house was randomly decided; thereafter, every third house was selected.

Individuals diagnosed previously with hypercholesterolaemia and/or diabetes mellitus were excluded because of possible dietary modifications. The other exclusion criteria were pregnancy, lactation, any acute or chronic illness, use of chronic medication and inability to communicate freely. The participants were, therefore, "apparently healthy" Indians.

The age selection range of 35-55 years was based on recommendations from the literature. In New Zealand, the recommendation regarding ages for incorporating cardiovascular risk assessment includes a degree of ethnic specificity, where South Asians are at risk from 35 years for males and 45 years for females (Lip *et al.,* 2007). Furthermore, CHD is diagnosed between five and ten years earlier in South Asians compared to other populations (Goyal & Yusuf, 2006). Hence the age selection range of 35-55 years was based on these findings, with the intention of targeting the most vulnerable age cohort in terms of the prevalence of NCDs in the Indian community of KZN. Annexure J illustrates the conceptual of methodology used in this study.

3.4 DATA COLLECTION

3.4.1 Dietary data

Habitual dietary intakes were measured by a validated QFFQ (Appendix D, MacIntyre *et al.,* 2001b) modified to Indian food habits and tested for comparative validity and reproducibility in a subsample of 50 subjects. In a study on the development of food frequency questionnaires and a nutrient database for the Prospective Urban and Rural Epidemiological (PURE) pilot study in South India, Bharathi *et al.* (2008) concluded that standard methods can be used to develop food frequency questionnaires (FFQs) for an Indian population with diverse dietary habits, as these are able to capture dietary intakes adequately. The previous day's food intake was determined by a 24-hour food recall.

3.4.1.1 The Quantitative Food Frequency Questionnaire (QFFQ)

The QFFQ has become the method of choice in many large-scale epidemiological studies, with the major advantage of establishing long-term habitual dietary intake (Willett, 1990). This retrospective method comprises a comprehensive list of foods and the respondent is required to recall the frequency of consumption per food item. The aim of using such a method is to capture the habitual dietary intake of the respondent, particularly when there are seasonal changes that may not be captured by the 24-hour food recall. However, this method is dependent on memory and therefore the recall of consumption could be imprecise. In order to determine portion sizes, the QFFQ is administered in conjunction with different measurements and aids such as portion books, food models and household measures. Common errors arising from the administration of the QFFQ are in the estimation of portion sizes and the usual frequency of the consumption of foods.

3.4.1.2. Development of the QFFQ

The QFFQ was adapted from MacIntyre *et al.* (2001a) in a preliminary study. For the preliminary study, a list of foods commonly consumed by Indian (South African) was compiled from interviews conducted in 25 households. One sub-area was selected

randomly from the six sub-areas chosen for this study. Thereafter, households were selected randomly, using the ratepayers' roll for the selected sub-area. Interviews focused on typical dishes consumed and combination dishes such as braised mutton and cabbage or calabash. It is known that the concentration of ingredients in certain preparations varies according to differences in religious and economic status (Herbert, 1985; Messer, 1997); therefore, recipes were collected for all the combination dishes as well as dishes not coded in the MRC Food Finder 3® (2006). Final construction of the recipes was determined by averaging three recipes per dish. Annexure G lists the standardised recipes and includes pictures of the indigenous food items.

A prototype QFFQ was developed from the food list. The biological name and the common Hindi, Tamil and Telegu names were added to assist respondents in identifying foods. Foods were grouped together, starting with the most frequently consumed staple foods and ending with the less common foods. The modified QFFQ included a cover page explaining important points on the administration of the QFFQ to the respondent. The questionnaire was arranged in columns, with a list of foods/drinks commonly consumed, amounts consumed and the frequency of consumption, which was subdivided into the number of times per day, per week or per month, and seldom (see Appendix D).

The prototype QFFQ was piloted amongst ten family units representative of the target population, selected from the preliminary study. Sample selection was based on a random sample from only one sub-area using the ratepayers' roll. The main aim of this exercise was to determine the completeness and appropriateness of the food list and the comprehensibility of the questionnaire.

3.4.1.3 The 24-hour food recall

The 24-hour food recall is a retrospective method whereby respondents have to recollect what they have had to eat and drink during the previous 24 hours. There is no literacy requirement and the respondent burden is relatively small. The administration time is short. A limitation of the 24-hour food recall is that the previous day's dietary intake may not be representative of a person's customary diet, as it yields no information on day-to-day variation of the food and nutrient intake. It is prone to

respondent bias, as there is the possibility that some people are embarrassed about what they eat.

The 24-hour food recall followed a chronological order from the first food/drink consumed for the day to the last. In order to help the respondents to remember what was eaten, they were asked to relate all the events during the day that preceded the interview, from the time they woke up till the time they went to bed. The first 50 respondents had three 24-hour food recalls (as part of the comparative validation study); thereafter, the remaining 200 respondents had one 24-hour food recall. A random selection of days was used, depending on the availability of the respondent. Weekends and religious and social events were avoided. Furthermore, days were also avoided if they were unusual in terms of normal dietary intake, e.g. dry fasting, saltless fast and whole-food fast, which are typical of a Hindu's religious prescription. However, fasts that exclude animal products in the diet for a day were not avoided, as these are observed by most Hindus for three days of the week, on average, while the range of the days depends largely on individuals' beliefs within Hinduism. Excluding these days would make administration of the questionnaire difficult and would not reflect true intake as this vegetarian fast forms part of the normal diet of Hindus, who represent the main religious denomination among Indians in South Africa.

3.4.1.4 Comparative reproducibility and validation

The QFFQ and the 24-hour food recalls were tested for comparative validity and reproducibility in a subsample of the first 50 respondents. The statistical methods and the results of the comparative validity and reproducibility study will be presented and discussed in Chapter 4.

3.4.1.5 Administration of the dietary questionnaires

Four fieldworkers (Indian women from the community, discussed in 3.10.1) were trained to administer the questionnaires used in the study, which included the socio-demographic and health questionnaire (see Appendix C), the 24-hour food recall and the QFFQ (see Appendix D). Administering the questionnaires took approximately two hours per respondent. This excluded clinical tests. Because of the length of the

questionnaires, they could not always be completed on the set date within the time limit. The fieldworker then arranged for a suitable alternative time to complete the questionnaires. On completion of the questionnaires, the respondent was given details and instructions about the clinical tests that were to follow.

3.5 TOOL KITS

In order to determine portion sizes, the MRC Dietary Assessment Education Kit (DAEK) was used in conjunction with utensils such as standard cup measures, plates, large serving spoons and ladles (Steyn & Senekal, 2002). Each fieldworker was supplied with these tools in a bag that was easy to carry. The DAEK consisted of generic life-size sketches of food portions and food photograph cards. On the front of each food photograph card was a specific food/drink item. The reverse of each food photograph card had information on the food codes from the Food Finder3® (2002) software program, guidelines to estimating portion sizes in the form of specific life-size sketches, and food quantities in grams for different portion sizes, derived from the MRC Food Quantities Manual (Langenhoven *et al.,* 1991), as well as weights obtained for packaging and products, specifically for fast foods.

The DAEK is presented in a file and all the photographs are colour-coded to simplify the finding of specific food items and their portion sizes. Once the respondent had indicated that he/she consumed a particular item, the fieldworker demonstrated the DAEK, together with the standardised utensils, to determine the size of the item consumed. The portion size was then recorded on the dietary questionnaire.

3.6 PHYSICAL MEASUREMENTS

Anthropometric measurements (weight, height, waist and hip circumference and percentage body fat) were taken by the researcher by means of standardised techniques and instruments. All instruments were checked for reliability at the start of each session. Initially, sessions were held at the Primary Health Clinic (PHC) in Stanger. However, subjects complained about travelling to the clinic and the response rate was poor. To combat this problem, respondents were visited at their homes. Telephone calls were made in advance to ensure that subjects were aware of the test

dates. Sessions were held over weekends and subjects that lived close to each other were grouped together. On average, 30 minutes were spent per respondent and clinical tests could be done on only four respondents per day, as respondents had to be fasting. Respondents were given clear instructions regarding the date and time, as well as the necessity of having nil per mouth from 22h00 the previous night.

For height measurement, the Panamedic Stature Meter 2M® was used (supplied by Sandler & Co., Pharmacy & Medical Sundries). Respondents were requested to be barefoot, and to stand up straight against the wall; measurements were recorded to the nearest cm. The Tanita body composition scale (bioelectrical impedance) model TBF 300®, Tanita Corporation, Tokyo, Japan was used to determine both body weight and body fat percentage. The bioelectrical impedance scale first indicated weight to the nearest gram and then the body fat percentage was calculated. All respondents had to be barefoot and wore lightweight clothes during these measurements. The BMI was calculated by using weight (kg)/height (m^2). WC was measured midway between the iliac crest and the lowest level of the last rib, using a non-flexible steel tape (Lufkin W606PM, Cooper Tools, USA). The NCEP/ATPIII criteria for WC and the modified criteria for Asian Indians were compared in the analysis of the data. Hip circumference was measured horizontally at the level of the larger lateral extension of the hips. All physical findings and anthropometry were completed before biochemical measurements were started.

3.7 BIOCHEMICAL MEASUREMENTS

Respondents were given instructions to fast for 12 hours. Initially, they were told to report to the assigned PHC facility. However, for logistical reasons, respondents preferred home visits. All biochemical measurements were recorded by a registered nurse. BP was recorded using a standard mercury sphygmomanometer. It was recorded in duplicate, with the subject in a sitting position, after five minutes of rest, according to standard guidelines. Finger-prick fasting blood glucose, triglycerides and total cholesterol were recorded using the Accutrend meter (Accutrend GCT kit, Accu-Check Softclix Pro Lancets) and strips (Accutrend Cholesterol, Glucose and Triglyceride Strips) supplied by Roche Products Pty Ltd, Randburg.

3.8 SOCIO-DEMOGRAPHIC AND MEDICAL HEALTH HISTORY DATA

Socio-demographic and medical health history data (including smoking and alcohol consumption) were collected using the existing adult questionnaire from the 2003 SADHS. These questionnaires were modified by excluding the dental health and occupational health sections from the original questionnaire. The questionnaire was then tested in a pilot study and reviewed for ease of comprehension by subjects. Minimal changes were effected. Firstly, *community health care clinic* (Question 1A) was changed to *primary health care clinic*, as the latter term is used in the area. Secondly, in order to ensure uniformity in the determination of the size of standard drinks for alcohol consumption, the DAEK was used (see Appendix D).

3.9 PHYSICAL ACTIVITY

Physical activity was measured by means of a questionnaire which has been validated in sites located in numerous countries (Global Physical Activity Questionnaire 1, GPAQ1, WHO, (http://www.who.int/chp/steps/GPAQ/en/index.html) (See Appendix C). The GPAQ1, introduced as part of the WHO STEP-wise approach to surveillance, provides for an overall measure of physical activity on the basis of intensity and duration, as well as physical activity levels within specific domains, including occupation, transport and leisure time.

Metabolic Equivalents (METs) are commonly used to express the intensity of physical activities, and are also used for the analysis of GPAQ data. METs are the ratio of a person's working metabolic rate relative to the resting metabolic rate. One MET is defined as the energy cost of sitting quietly, and is equivalent to a caloric consumption of 1 kcal/kg/hour. Therefore, as an example, when calculating a person's overall energy expenditure using the GPAQ data, 4 METs are assigned to the time spent in moderate activities and 8 METs to the time spent in vigorous activities. For this study, the GPAQ 1 was used, which was the first validated questionnaire. Thereafter, the GPAQ 2 was developed through refining the GPAQ1 by changing a total of three questions.

Consequently, the data were cleaned and recoded to the GPAQ2 questionnaire and analysed on the EpiInfo Analysis Program. Appendix F lists examples of moderate-

intensity activities [approximately 4 metabolic equivalents (Mets)] and vigorous-intensity activities (>8 Mets) that were used by the fieldworkers for differentiating between activities during administration of the questionnaire. In order to verify the results of the GPAQ1, pedometers (Product number SK01, Blue Chip Marketing, UK, supplied by Kellogg's, Private Bag X16, Gallo Manor) were used for step counting. Respondents used the pedometer for a normal day and recorded the steps in a dairy.

3.10 QUALITY CONTROL

3.10.1 Training and standardisation of fieldworkers

In order to recruit fieldworkers, an advertisement was placed in the KwaDukuza community paper with the following requirements: fieldworkers were to be matriculated Indian females (unemployed, aged +30years) with knowledge of Indian cuisine. Four fieldworkers were selected from the sample population and trained to administer the questionnaires in an interview set-up. Before the actual fieldworker training, a training manual was developed for use during the training and for cross-referencing during the administration of questionnaires (see Appendix E). The fieldworker training was run by the researcher A.Naicker, Prof C.S. Venter (supervisor) and Prof U.E. MacIntyre (co-supervisior). The training ran for two days and its aim was to equip the fieldworker adequately with the necessary skills to administer the questionnaires. At the start of the administration of the questionnaires, there were four fieldworkers; however, two found permanent employment and dropped out. To combat the manpower problem, the researcher opted to conduct fieldwork.

3.11 DATA MANAGEMENT AND ANALYSIS

3.11.1 Dietary data

The Food Finder 3 ® (MRC, 2002) dietary analysis software program based on the South African MRC food composition tables was used to capture all the dietary data. However, before any dietary data could be captured by this program, Indian ingredients and recipes not coded in the MRC food composition list had to be added (see Appendix G). In the case of typical Indian dishes where individual ingredients were not coded, the

nutritive value of Indian foods was used as a guide (Gopalan *et al.,* 2004). This handbook, "Nutritive value of Indian foods", provided detailed information on the nutrient composition of a wide range of common Indian foods available in different parts of India. The foods were listed with confirmed scientific names and in several Indian languages, with nutrient composition for 100 g serving sizes. After the nutritive composition of a new food item was coded, recipes not listed in the program had to be coded. In the preliminary study, combination dishes and recipes were collected and averaged (see Appendix G). This information was then added and appeared with its own code in the program. In some instances where an item could not be found in either the MRC list or the Indian food composition table, a suitable substitute was selected, e.g. a variety of green leafy vegetables. Further to this, common dishes were also cross-referenced with the recipe book used in the coding of the dishes in the Food Finder 3® dietary analysis software program to determine similarity before a dish could be selected (Gouws & Langehoven, 1986).

Once all the outstanding food items and recipes were coded, the researcher calculated all totals for the amount eaten per food in the QFFQ. Thereafter, all dietary data were captured by the researcher. The task was time-consuming because of the wide variety of foods and combination dishes eaten by Indians. Checks were conducted for key pad error and if the information was correct, it was saved. However, in doing so, a common error was compounded. In running a normal check per individual for correct values, sometimes the cursor was left in the first or last value of the food list, and the food list was then saved. It was discovered only later, after running the analysis and assessing the diets, that whenever the cursor was left on the first or on the last food item, the total amount was recorded as zero. Once this problem was identified, it was rectified.

From the literature review in Chapter 2, it can be concluded that diet is a major risk factor for chronic diseases, restricting the quality of life and longevity. In an attempt to identify groups of individuals with either a high or a low consumption of certain nutrients, certain defining nutritional characteristics can be identified, leading to a more focussed intervention. People who have freedom of choice do not consume single foods but a combination of several foods and, given the complexity of the human diet, conclusions about the effect of the consumption of a single nutrient, food, or dietary constituent on a specific health outcome may be misleading (Mertz, 1984; Kant, 1996). Therefore, a

number of epidemiologists propose the dietary pattern approach, where food in relation to disease is investigated (Kennedy *et al.,* 1995; Schwerin *et al.*, 1982). Two methods will be used to determine dietary patterns, namely Index construction and factor analysis.

There is a wide variety of diet quality indices available from the nutrition literature. Kant (1996) provides an overview of the published indices of overall diet quality and identifies three major approaches to the construction of indices of diet quality as reported in the literature, namely indices derived from nutrients only, those based on foods and food groups and those based on a combination of nutrients and foods. According to Thiele (2004), the purpose of the indices is to combine a large amount of information about eating habits into a single indicator, the advantage of which is that dietary behaviour can be analysed as a single factor evaluating many compounds of the human diet.

For the purposes of this study, the indices were constructed from nutrients only. In order to analyse the diet quality, 30 nutrients were included. Two dietary quality indices, a deficiency index (Index 1) and an excess index (Index 2), were calculated, following the procedure of Thiele *et al.* (2004). The deficiency index was calculated as the percentage of the reported nutrient intake of the Recommended Dietary Allowance (RDA) or the AI (for nutrients without an RDA) (Food and Nutrition Board, 2000). The deficiency index was calculated by subtracting the percentage difference between the reported index and recommended intake from 100. The excess index was calculated as follows:

*Index 2 = 100 – [(reported intake / recommended intake*100)-100]* (Thiele *et al.,* 2004).

The deficiency index was derived from 13 vitamins, 12 minerals and trace elements, protein, carbohydrate, omega-6 and n-3 fatty acids and dietary fibre (30 nutrients), with a maximum score of 3 000. The excess index comprised fat, cholesterol, the P:S ratio, sugar, alcohol and sodium, with a maximum score of 600. For indices, the higher the index, the better the diet quality. The components of the indices and examples of the calculation of the indices are given in Appendix H.

In order to determine a food pattern, factor analysis was carried out, where foods were separated into groups based on correlations between foods. For the purposes of this study, dietary intake was assessed by the use of the QFFQ. The Food Finder 3 program® (MRC, 2002) automatically aggregated foods listed on the QFFQ (243), on the basis of similarity of the type of food, into 12 groups, namely:

1. Cereals and cereal products
2. Vegetables
3. Fruit
4. Legumes and legume products
5. Nuts and seeds

6a. Full fat milk

6b. Low fat milk

7. Eggs
8. Meat and meat products
9. Fish and seafood
10. Fats and oils
11. Sugars, syrups and sweets

3.11.2 Risk score model

In order to determine the burden of risk factors on the prevalence of NCDs, the European SCORE model (Graham *et al.*, 2007) was used. The SCORE project was initiated to develop a risk-scoring system for use in the clinical management of cardiovascular risk in European clinical practice (Conroy *et al.,* 2003). The European SCORE model that was chosen to estimate the risk of CVD had the following categories: age, sex, smoking, total cholesterol and systolic blood pressure. In terms of the selection of the prediction tool, the cholesterol score was used as depicted below in Figure 3.2, as HDL values were not available. Lip *et al.* (2007) discussed the applicability of the European SCORE model and pointed out that it is limited by the lack of inclusion of HDL and diabetes as a prediction tool. However, Graham *et al.* (2007) also developed a risk-estimation system based on cholesterol/HDL ratio and reported no advantage of this score over that which included only cholesterol. In addition to the use of the European score, it has been recommended that in order to determine the CAD risk more appropriately among Asian Indians, one must multiply the 10-year risk factor by a factor of 2 when using the European risk algorithm (Enas *et al.,* 2008). This

recommendation is based on studies where Asian Indians have shown ≥2-fold risk pattern of dying from CAD from any combination of risk factors as compared with Caucasians (Forouhi *et al.*, 2006). For comparative purposes, two scores were developed for this study, namely "Risk score", which was an aggregate total of all risk markers defined by Asian standards, as presented in Chapter 2, section 2.6, and "Risk score 1" which was the aggregate of all risk markers, with fasting glucose counting twice as much as the other risk markers because of many and strong correlations with nutrients.

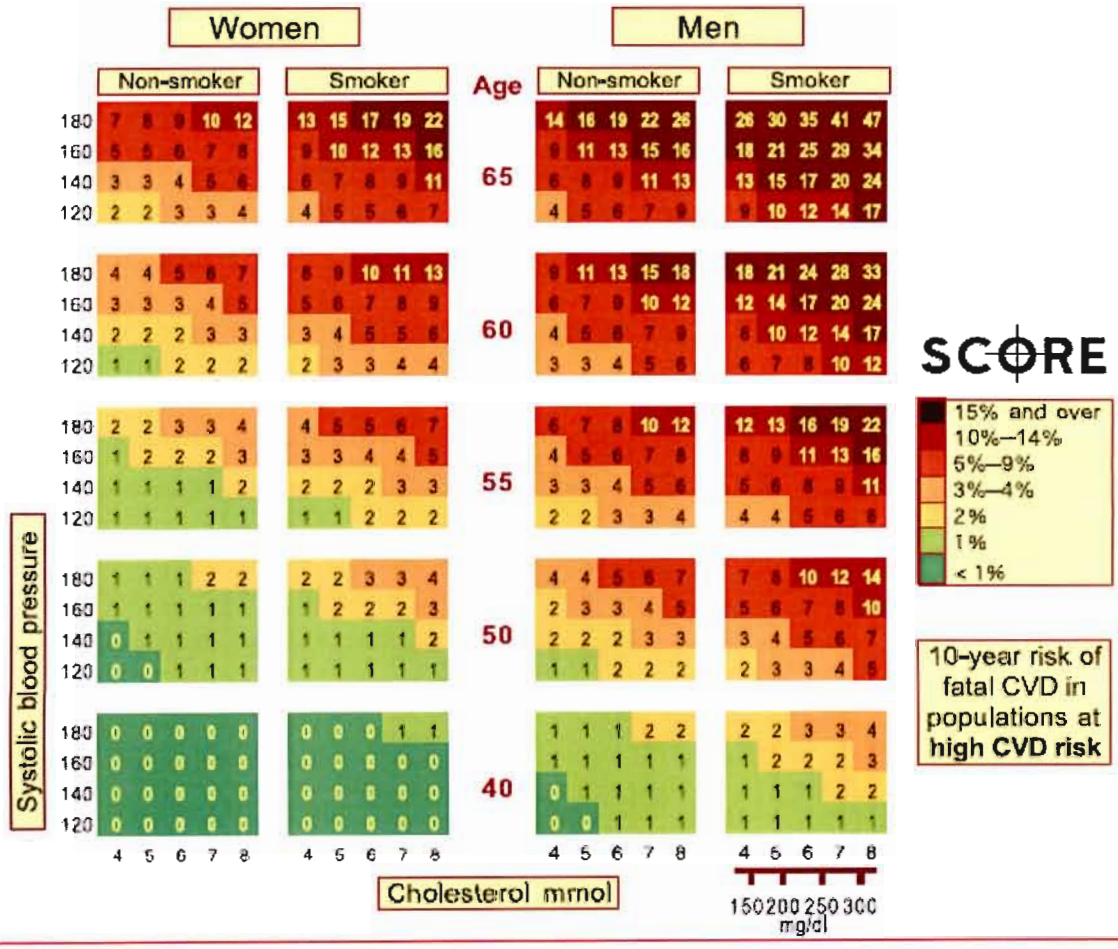

Figure 3.2: Ten-year risk of fatal cardiovascular disease in populations at high cardiovascular disease risk (Chart based on total cholesterol) (Conroy *et al.*, 2003). Systolic BP in mmHg, cholesterol in mmol/L

3.11.3 Statistical analysis

Once the dietary data were coded and checked, the nutrient analyses and the food lists were exported to Excel (Microsoft Excel, 2007). The statistical methods of the reproducibility and the comparative validity study are discussed in Chapter 4.

The physical activity questionnaire was captured and cleaned according to the GPAQ Analysis Guide, using EpiInfo (http://www.who.int/chp/steps/GPAQ/en/index.html). Descriptive statistics such as frequency tables, mean, median, mode and standard deviation were run for the socio-demographic questionnaire, anthropometric profile and clinical tests. BoxCox transformations were performed on all continuous variables to ensure normality of data, after which Pearson's correlations were used to detect associations between nutrient intake and clinical risk markers, nutrient intake and associated risks, and between physical activity and clinical risk markers.

Principal component factor analyses with Varimax rotation were performed on the percentages of total energy, carbohydrate, fat and saturated, polyunsaturated, monounsaturated, n-3 and n-6 fatty acid intakes derived from each of the food groups. Since the main interest of the study was fat intake, the first two principal components derived from the percentage total fat intake were used for further analyses. Factor scores were calculated, divided into quintiles, and sample means of the risk variables were calculated. Univariate chi-square tests were used to determine the relative importance of factor score quintiles on the risk variables. The Jonckheere-Terpstra test in SPSS was used to test for a trend in the median of risk variables for the different quintile groups of factor scores. Separate regression analyses were performed for each factor to test whether food patterns predicted changes in risk measures. The statistical SAS package was used to analyse data in consultation with Dr S. Ellis from the Statistical Consultation Service at the NWU.

3.12 ETHICS

Meetings were held with key persons (ward councillors) and groups (religious groups) in the community to obtain voluntary consent. The subjects were carefully briefed on the aim and procedures of the study before they signed the INFORMED consent form (see

Appendix I). Confidentiality was strictly observed. Permission was granted by the Ethics Committee of NWU (05M15).

3.13 PROBLEMS EXPERIENCED

A wide array of problems was experienced during the administration of the questionnaire and the collection of the clinical data. In terms of the administration of the questionnaire, subjects were alerted to the approximate length of time of the interview during the recruitment process; however, during the actual administration, respondents became tired or had unforeseen commitments and hence the fieldworkers and the researcher had to reschedule another interview to complete the process. With regard to fieldworkers, four were initially trained and worked in the pilot (preliminary) study. However, two found permanent employment during the course of the administration of the survey. To combat the manpower problem, the researcher also administered questionnaires. At the start of the survey, it was decided that once the respondents had completed their questionnaires, they would go to the PHC on set dates for their clinical tests. However, respondents did not arrive at the clinic and attendance was poor. Respondents complained about transport costs and unforeseen commitments. It was therefore decided that a registered nurse would visit households on set dates. This was much more successful, because households were grouped for appointments within a two-hour range, as respondents had nil per mouth. During the clinical tests, one Accutrend® machine was used; however, a need arose for another Accutrend® machine as readings took a long time. In order to avoid pricking each respondent's finger twice, another machine was purchased.

With regard to the capturing of dietary data on Food Finder 3®, it was very difficult to get trained personnel to assist with this task. The researcher was responsible for capturing all the data, and the long food lists of the respondents meant that capturing became a time-consuming task. Upon checking for keypad error for values listed, a critical error arose which was rectified only after the validation results were reviewed. While the operator was scrolling through all the food values, in most cases the cursor ended on either the first food item or the last food item, which was therefore erroneously highlighted. Once the "save" button was pressed, it appeared that all the values had been saved. However, wherever the cursor had been left the value became zero. This

was a critical error, as the first item was bread and the last item was atchar. The problem was rectified by previewing all recorded food lists, recapturing values and ensuring that the cursor was placed on a blank line during saving.

3.14 CONCLUSION

In this chapter the research methodology, data management and analysis were described. Shortcomings and problems experienced during the fieldwork were also discussed. The methods used to test comparative validity and reproducibility and the results of these tests are presented in Chapter 4.

CHAPTER 4

REPRODUCIBILITY AND COMPARATIVE VALIDITY OF THE QUANTITATIVE FOOD FREQUENCY QUESTIONNAIRE

4.1 INTRODUCTION

The QFFQ has become a well established method of dietary assessment in most large-scale epidemiological studies. Despite its limitations, it is sustained by its major advantage of establishing long-term habitual diet (Willett *et al.,* 1985). It is well recognised that there is no gold standard for directly assessing the validity of QFFQs; however, testing for comparative validity and reproducibility will allow the results of the study to be interpreted with greater confidence, as validity has been quantified.

The reproducibility of a questionnaire is measured by its ability to achieve similar results when administered at different times to the same subjects. Comparative validity refers to the ability of a questionnaire to estimate the dietary intake during the appropriate time period with relative accuracy compared with a reference method. The QFFQ and the 24-hour recalls were tested for reproducibility and comparative validity in a subsample of the first 50 respondents. Reproducibility was tested by comparing the nutrient intakes derived from the administration of the QFFQ on two occasions for the first 50 respondents. Comparative validity was tested by comparison of the average intakes of the three 24-hour recalls with the average intakes derived from the QFFQ. This chapter describes the methods and the respondents used for the purposes of determining reproducibility and comparative validity of the selected dietary assessment method. The results of the reproducibility and comparative validity study are presented and subsequently discussed.

4.2 METHODOLOGY

4.2.1 Sample

4.2.1.1 Sample size

The desired sample size was estimated at 20% (50) of the total sample of 250.

4.2.1.2 Selection of respondents

Respondents were randomly selected to make up the subsample of 50 from all six areas in the KwaDukuza Municipality, in accordance with the inclusion and exclusion criteria discussed in Chapter 3. Respondents were given an explanation of the purpose of the reproducibility and validity studies and were asked if they were willing to undergo the two QFFQs and three 24-hour recalls over a period of about five weeks. The length of time per administration of the dietary questionnaires was also declared to respondents so that they could make an informed choice in terms of participation. Upon consent from the individual, the reproducibility and the validity studies continued.

4.2.2 Data collection

For the administration of the 24-hour recall there was a two-week gap between administrations of the questionnaire. In total, the collection of data for the 24-hour recalls took five weeks per respondent. The days of the week selected for the administration of the 24-hour recalls ranged from Tuesdays to Fridays, avoiding days immediately following religious and social festivals. With regard to the QFFQ, there was a two-week break between the first and the second administration, with no limitations in terms of the day of the week each was administered. In terms of the timing of the administration of the QFFQ and the 24-hour recall, for every 24-hour recall recorded, a QFFQ followed until all administrations were complete. All repeat administrations of the questionnaires were conducted by the same fieldworker.

4.2.3 Statistical analyses

Dietary data were captured and cleaned as described in Chapter 3. Owing to the lack of agreement on the best ways of analysing results of reproducibility and validity data, it is necessary to use more than one statistical method in order to give greater credibility to the results. Therefore, a number of methods are presented simultaneously in order to illustrate the merits and limitations of each method. Reproducibility and comparative validity were tested for intakes of energy, macronutrients and micronutrients to be assessed in the main study. For purposes of reproducibility, the intakes of the QFFQ1 were compared with those of the QFFQ2 and for comparative validity, the average intakes of the three 24-hour recalls were used as a reference standard for comparison with the intakes from the QFFQ1. In epidemiological studies, the odds ratio, or relative risk of disease in relation to nutrient intake, is the most common measure of the association present. Therefore the QFFQ must be able to rank individuals along the distribution of intakes so that individuals with low intakes can be separated from individuals with high intakes (Masson *et al.,* 2002). Relative risk estimates will be accurate if the QFFQ can rank individuals (Sempos, 1992). Therefore, Spearman and Pearson correlation coefficients were calculated to assess the agreement of ranking of individuals in the QFFQs for reproducibility. In addition, they were calculated to assess agreement of ranking of individuals between the QFFQ and the average intakes from the three 24-hour recalls for comparative validity. All non-normally distributed data were $\log_e$ transformed prior to statistical analysis. The Pearson correlation coefficients between the average intakes from the three 24-hour recalls and the intakes from the QFFQ1 (r_{RQ}) were adjusted for within- and between-respondent variation in the three 24-hour recalls by dividing r_{RQ} by the attenuation factor (r_{RT}) where $r_{RT} = [d/(d+s_w^2/s_b^2)]^{0.05}$ where d is the number of 24-hour recalls, s_w^2 is the within-respondent variance and s_b^2 is the between-respondent variance (Nelson,1997). For the purpose of having a more representative presentation of the results, reproducibility and comparative validity were also tested by comparison of mean intakes (paired t-test), Bland-Altman plots and quartile distributions.

Under-reporting of energy intake is a well recognised phenomenon. It has been observed in a number of studies (Prentice *et al.,* 1986; Macdiarmid & Blundell, 1998). According to McCrory *et al.* (2002), that inaccurate energy-intake reports can result from

under-recording (defined as the failure to record all items and/or the amounts consumed), under-eating (defined as eating less than usual or than is required to maintain body weight) or a combination of both under-recording and under-eating. Most methods used in population-based studies generally assume a sedentary lifestyle and, in doing so, are restricted in identifying individuals with high energy expenditure. Therefore, for the purpose of determining under-reporting, a method was chosen that uses individualised estimates of energy requirements. Possible under-reporting and over-reporting of energy intake from both the 24-hour recalls and the QFFQ were identified, using the method of Rennie *et al.* (2007). The Estimated Energy Requirements (EER) were calculated using the formulae of the 2000 Food and Nutrition Board (FNB, 2000). The equations are sex- and age-specific and also take into account the physical activity level, weight and height of the respondent (Rennie *et al.,* 2007). Physical activity levels were derived from the analysis of the GPAQ questionnaires (WHO, http://www.who.int/chp/steps/GPAQ/en/index.html) as described in Chapter 3 section 3.9, where each subject was assigned to an activity level based on his or her GPAQ score.

The equations are as follows:

Men: Older than 19 years

EER (kCal) = 662 - (9.53 x age(y)) + PA x (15.91 x weight (kg) + 539.6 x height (m)

Women: Older than 19 years

EER (kCal) = 354 - (6.91 x age(y)) + PA x (9.36 x weight (kg) + 726 x height (m)

The results of the calculation of the EER were converted to kJ by multiplication by 4.2. PA is the physical activity coefficient which equates the physical activity level (PAL) with an estimated coefficient. Table 4.1 shows the PA coefficients used in the prediction equations for total energy expenditure in relation to the PAL as defined by the FNB (2000) and the equivalent PAL as derived from the GPAQ questionnaire (http://www.who.int/chp/steps/GPAQ/en/index.html).

Table 4.1: Physical activity level of respondents as defined by the FNB† and ††GPAQ with the physical activity coefficient

Physical activity level		†Physical activity coefficient	
†FNB	††GPAQ	Men	Women
Sedentary	Low	1	1
Low active	Moderate	1.11	1.12
Active	High active	1.25	1.27
High	-	1.48	1.45

†Food and Nutrition Board, 2000; ††GPAQ (http://www.who.int/chp/steps/GPAQ/en/index.html).

The calculation of the percentage of under-reporting and over-reporting of the reported energy intake is expressed as a percentage of EER= [100 x (EER – Reported EI)/EER]. Since normal day-to-day variation in energy intake (EI) and energy expenditure exists, exact agreement between EI and expenditure over fewer than seven days in an individual is unlikely. It has been estimated that dietary intake measurements are required for between seven and 32 days to classify an individual's energy intake according to the true intake with 90% confidence (Marr *et al.,* 1986: Nelson *et al.,* 1989). In a dietary collection period over a shorter term, it is possible that the measurements may represent the individual's actual intake during the recording period, but unless they are recorded for seven days, they would not represent habitual diet. Therefore, confidence limits of agreement between EI and EER, and thus the limitations of EER for identifying under-reporters at the individual level, need to be calculated using the method described by Black and Cole (2000). Cut-off points to identify possible over- and under-reporters were calculated using the following formula (Rennie *et al.,* 2007):

$CV_t = \sqrt{(CV^2_{EE} + CV^2_{EI}/d)}$ where CV_t is the coefficient of variation (CV) for both energy expenditure (EE) and reported energy intake (EI), d is the number of days of dietary reporting, and CV_{EE} and CV_{EI} are the CV for EE and EI respectively. Since the EER equations are based on doubly labelled water studies of seven days' duration, the CV_{EE} is taken as 8.2% (Rennie *et al.,* 2007).

Possible under-reporters and over-reporters were identified as respondents whose reported energy intake, expressed as a percentage of the EER, fell below (100-2SD) or

above (100+2SD) the EER, where CV_t was considered equivalent to the SD (Rennie *et al.*, 2007).

4.3 RESULTS

4.3.1 Reproducibility

4.3.1.1 Comparison of the results of the reported nutrient intakes between administrations of the QFFQ

4.3.1.1.1 Spearman rank and Pearson correlation coefficients

The Spearman rank and Pearson correlation coefficients were calculated to determine the level of agreement between the first and the second administrations of the QFFQ. Table 4.2 shows the correlation coefficients for the entire sample.

Table 4.2: Spearman rank and Pearson correlation coefficients (r) between the first and second administrations of the QFFQ (N=50)

Nutrient	Spearman r	P-value	†Pearson r†	P-value
Energy	0.156	0.278	0.178	0.215
Total protein	0.355	0.02*	0.494	0.0003*
Total fat	0.473	0.0005*	0.494	0.0003*
SAT FAT	0.427	0.002*	0.542	0.000*
MUFAs	0.442	0.001*	0.556	0.000*
PUFAs	0.591	0.000*	0.518	0.0001*
n-6	0.621	0.000*	0.547	0.000*
n-3	0.287	0.043*	0.286	0.044*
Cholesterol	0.757	0.0000*	0.801	0.000*
Total carbohydrate	0.153	0.287	0.246	0.085
Calcium	0.454	0.001*	0.451	0.001*
Iron	0.472	0.002*	0.426	0.002*
Zinc	0.395	0.004*	0.476	0.001*
Vitamin A	0.452	0.001*	0.536	0.000*
Thiamin	0.606	0.0000*	0.525	0.000
Riboflavin	0.534	0.00003*	0.549	0.000*
Niacin	0.559	0.0000*	0.630	0.000*
Vitamin C	0.669	0.0000*	0.508	0.000*

†log *e* transformed; Level of significance: *P<0.05; SAT FAT= saturated fatty acids; MUFAs=monounsaturated fatty acids; PUFAs=polyunsaturated fatty acids

Table 4.2 shows the Spearman rank correlation coefficients and the Pearson correlation coefficients for loge transformed data to improve normality for all the nutrients tested. The strongest Spearman rank correlation coefficient was for cholesterol (0.76, statistically significant) and the weakest non-significant correlation was for total carbohydrate (0.153), followed closely by energy, with a correlation of 0.156. The remaining coefficients varied between 0.2 and 0.6. The strongest statistically significant Pearson correlation was for cholesterol (0.80) and the weakest correlation was for energy (0.178, not significant), with the remaining correlation coefficients varying from 0.2 to 0.6. Of the total of the 19 nutrients that were assessed, the Spearman correlation coefficient totalled nine higher recordings than the Pearson correlation coefficient whilst the Pearson correlation coefficient totalled ten higher recordings than the Spearman correlation coefficient. Overall, the Pearson correlation coefficient tended to be higher than Spearman correlation coefficient with the Pearson correlation coefficients above 0.4 for 15 nutrients, whereas the Spearman correlation coefficients were above 0.4 for 13 nutrients. Both Spearman and Pearson correlation coefficients were above 0.4 for total fat, saturated fatty acids, monounsaturated fatty acids, PUFAs, n-6 fatty acids, cholesterol, calcium, iron, vitamin A, thiamin, riboflavin, niacin and vitamin C.

4.3.1.1.2 Paired t-tests for reported nutrient intakes between first and second administrations of the QFFQ

In order to test reproducibility of the QFFQ, the mean reported intakes of the first and the second administrations of the QFFQ (Table 4.3) were compared.

For the entire sample, the mean reported intakes of the second administration of the QFFQ were higher than for the first, with the exception of PUFAs, n-6 fatty acids and vitamin C. The difference, expressed as a percentage of the mean of the two administrations of the QFFQ, gives a clearer indication of the agreement between the results. A difference of less than 10% is considered a good agreement (Wheeler *et al.,* 1994).

Table 4.3: Median (25th; 75th percentile), mean (95% CI), standard deviation and difference between means of the first and second administrations of the QFFQ (N=50)

Nutrient	QFFQ 1			QFFQ 2					
	Median (25th;75th)	Mean (95% CI)	¶SD	Median (25th;75th)	Mean (95% CI)	¶SD	⁋ Mean Difference ⁋(%)	SD difference	P-value (paired t-test)
Energy (kJ)	7333 (5987;8140)	7210 (6769;7649)	1548	7889 (7151;8575)	7811 (7517;8195)	1033	601 (8.0)	1686	0.015*
Total protein (g)	54.4 (44.0;67.1)	56.7 (52.1;61.4)	16.2	60.2 (55.6;66.6)	60.2 (55.7;63.0)	9.8	3.5 (5.9)	14.5	0.100
Total fat (g)	67.1 (55.3;78.0)	68.1 (63.4;72.8)	16.6	71.3 (67.0;76.5)	71.1 (68.1;74.0)	10.4	3.0 (4.3)	15.1	0.161
SAT FAT (g)	19.0 (15.0;21.2)	19.0 (17.5;20.6)	5.4	20.9 (19;23)	20.6 (19.5;21.7)	3.97	1.6 (8.0)	4.9	0.025*
MUFAs (g)	18.5 (14.8;22.0)	18.9 (17.4;20.3)	16.4	20.6 (18.8;22.2)	20.1 (19.1;21.2)	3.7	1.2 (6.1)	4.73	0.067
PUFAs (g)	22.5 (19.1;26.3)	23.2 (21.5;24.9)	6.0	22.4 (20.8;25.5)	23.0 (22.0;23.9)	3.3	-0.2 (-0.8)	5.24	0.831
n-6 (g)	21.0 (18.0;24.9)	21.9 (20.2;23.5)	5.8	21.2 (19.5;24.3)	21.6 (20.6;22.5)	3.2	-0.3 (-1.3)	4.99	0.650
n-3 (g)	0.43 (0.36;0.53)	0.5 (0.41;0.49)	0.1	0.5 (0.46;0.57)	0.51 (0.49;0.54)	0.1	0.01 (12.5)	0.13	0.003*
Cholesterol (mg)	195.7 (137;255)	207.1 (180.4;233.7)	93.7	203.7 (191.5;242)	211.5 (194;229)	62.1	4.4 (2.1)	61.7	0.610
Carbohydrate (g)	205.1 (161.6;236.5)	201.1 (186.8;215.5)	50.4	230.4 (199;250)	225.3 (214.3;236.3)	38.7	24.3 (11.35)	54.8	0.003*
Calcium (mg)	415.0 (334.3;516.8)	432.0 (388.1;475.2)	153.3	482.5 (438;527)	480.4 (450.4;510.4)	105.5	48.6 (11.3)	127.7	0.0009*
Iron (mg)	9.3 (7.8;11.1)	9.7 (9.6;10.4)	2.9	10.0 (9.0;10.9)	10.1 (9.6;10.5)	1.5	0.4 (4.04)	2.53	0.174
Zinc (mg)	6.8 (5.8;8.7)	7.2 (6.7;7.8)	2.1	7.7 (7.2;8.7)	7.8 (7.4;8.1)	1.3	0.6 (8.0)	1.86	0.055
Vitamin A (mcg)	507.1 (383;712)	598.2 (506;691)	326.0	640 (529;678)	632.9 (576.5;689.3)	198.6	34.7 (5.6)	235.5	0.302
Thiamin (mg)	0.81 (0.63;0.93)	0.80 (0.74;0.87)	0.23	0.81 (0.75;0.90)	0.82 (0.78;0.85)	0.13	0.02 (2.4)	0.19	0.649
Riboflavin (mg)	1.0 (0.74;1.21)	1.0 (0.92;1.15)	0.40	1.1 (1.0;1.2)	1.1 (1.0;1.2)	0.24	0.1 (9.5)	0.31	0.105
Niacin (mg)	14.3 (12.7;16.5)	14.6 (13.4;15.8)	4.2	14.9 (14.0;16.6)	14.9 (14.1;15.7)	2.74	0.3 (2.06)	3.53	0.575
Vitamin C (mg)	71.0 (45.8;118.0)	94.3 (73.1;115.5)	74.5	61.2 (47.0;98)	78.4 (61.3;95.6)	60.2	15.9 (18.4)	49.4	0.027*

QFFQ 1; QFFQ 2: First and second administrations of the quantitative food frequency questionnaire; SD= standard deviation; ⁋Difference= mean of second-mean of first administration of the QFFQ; ⁋ Mean difference: = (QFFQ2- QFFQ1); ⁋% Mean difference = (QFFQ2- QFFQ1)/(QFFQ1+ QFFQ2)/2) *100; Level of significance:*P<0.05; SAT FAT= saturated fatty acids; MUFAs=monounsaturated fatty acids; PUFAs=polyunsaturated fatty acids

The mean percentage difference (Table 4.3) of all the nutrients tested was 7.3%, with energy, total protein, total fat, saturated fatty acids, monounsaturated fatty acids, PUFAs, n-6 fatty acids, cholesterol, iron, zinc, vitamin A, thiamin, riboflavin and niacin showing differences within 10%.

4.3.1.1.3 Agreement between the first and second administrations of the QFFQ (Bland-Altman method)

The Bland-Altman method plots the difference in intakes between the two administrations (QFFQ1 - QFFQ2) against the average of the intakes of the two administrations (QFFQ1 + QFFQ2/2). It is used to identify whether proportional bias is present. The desired situation is one of no proportional bias, i.e. the size of the difference between the two methods should be constant across all levels of average intake (Bland & Altman, 1986). Proportional bias, indicated by a significant Spearman rank correlation coefficient indicates that the difference increases/decreases in proportion to the average intake. Wide limits of agreement (mean difference ±2 SD) indicate poor agreement (Table 4.4).

As can be seen from Table 4.4, significant proportional bias was present for all nutrients except saturated fatty acids.

A better way of displaying the data is to plot the difference between the measurements by the two methods for each respondent against their average. Differences that lie between the mean difference ± 2SD are in agreement (Bland & Altman, 1986) and thus the number of outliers will define the strength of agreement between the two measurements.

Table 4.4: Summary of agreement between the first and the second administration of the QFFQ (N=50)

		Limits of agreement		
Nutrient	Spearman r	Lower limit	Upper Limit	P-value for Spearman r
Energy	0.340	-3974.8	2770.6	0.016*
Total protein	0.441	-32.38	25.52	0.001*
Total fat	0.444	-33.13	27.07	0.001*
SAT FAT	0.226	-11.32	8.13	0.114
MUFAs	0.310	-10.73	8.22	0.028*
PUFAs	0.519	-10.32	10.64	0.0001*
n-6	0.529	-9.68	10.3	0.0001*
n-3	0.381	-0.33	0.21	0.006*
Cholesterol	0.757	-129.5	120.4	0.000*
Total carbohydrate	0.397	-133.2	84.84	0.005*
Calcium	0.337	-304.1	206.7	0.016*
Iron	0.515	-5.49	4.51	0.0002*
Zinc	0.414	-4.2	0.32	0.002*
Vitamin A	0.509	-508.7	433.3	0.0001*
Thiamin	0.419	-0.40	0.38	0.002*
Riboflavin	0.342	-0.68	0.54	0.014*
Niacin	0.343	-7.34	6.78	0.015*
Vitamin C	0.398	-82.9	114.7	0.004*

Level of significance: *P<0.05; SAT FAT = saturated fatty acids; MUFAs = monounsaturated fatty acids; PUFAs = polyunsaturated fatty acids

Figures 4.1 to 4.4 display Bland-Altman scatter plots for saturated fatty acids, monounsaturated fatty acids, and n-6 and n-3 fatty acids. Figure 4.1 shows one respondent falling above the upper limit of agreement (8.13 g) and no one falling below the lower limit of agreement (-11.32 g) for saturated fatty acids. A Spearman rank correlation coefficient of r=0.226 (P>0.05) indicates that there was no significant proportional bias.

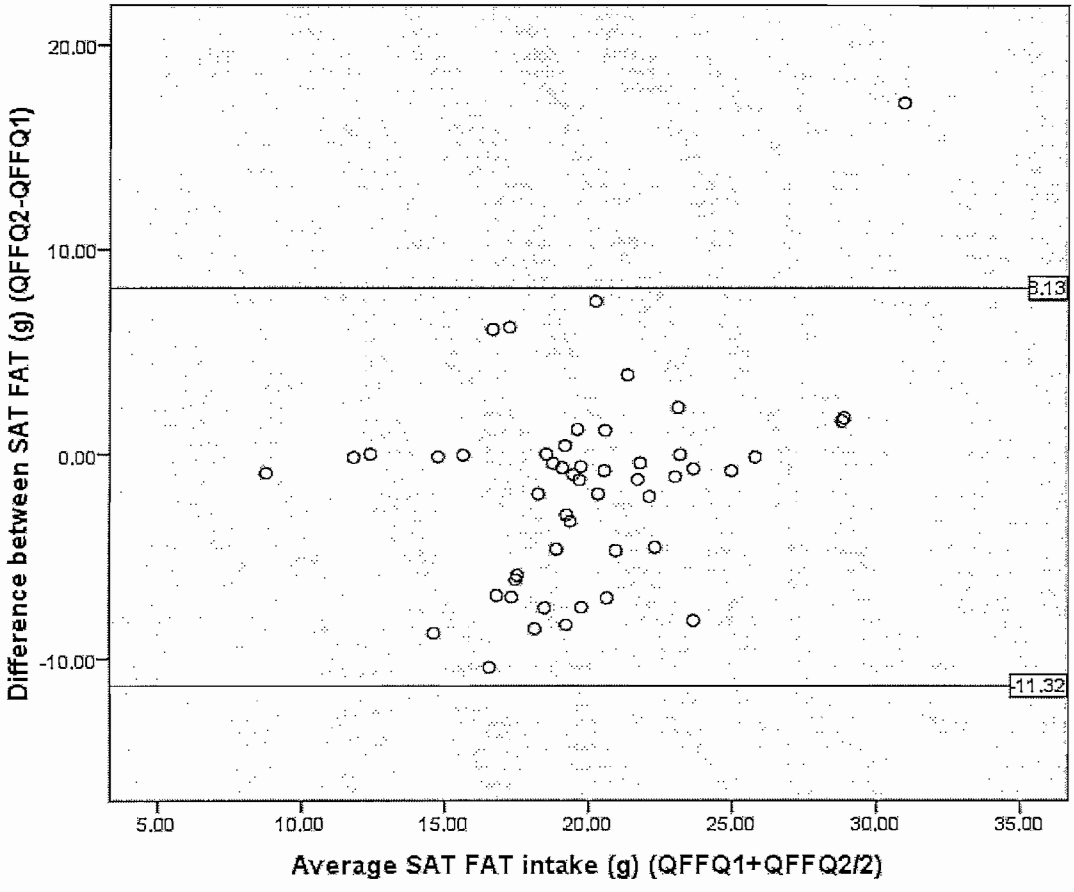

Figure 4.1: Bland-Altman plot for saturated fatty acids (N=50)

Figure 4.2 shows two respondents falling above the upper limit of agreement (8.22 g) for monounsaturated fatty acids and no respondents falling below the lower limit of agreement (-10.73). A Spearman rank correlation coefficient of r=0.310 (P<0.05) indicates that there was significant proportional bias.

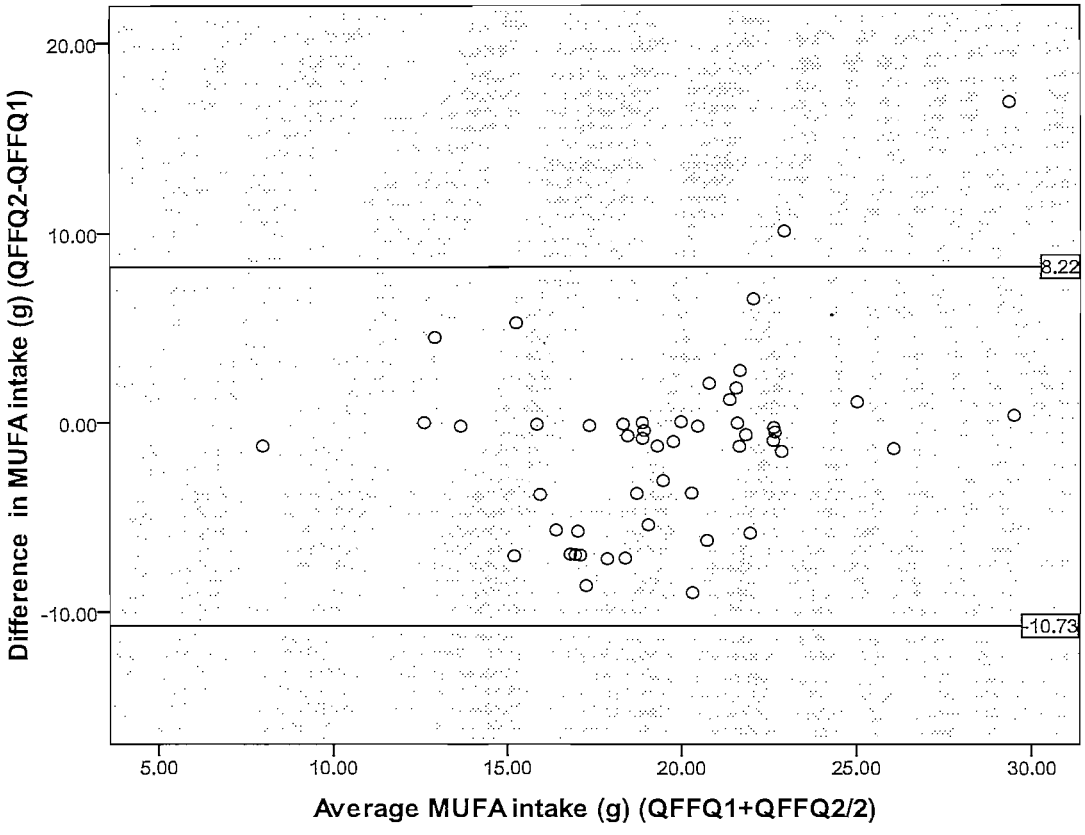

Figure 4.2: Bland-Altman plot for monounsaturated fatty acid intake (N=50)

Figure 4.3 shows three respondents falling above the upper limit of agreement (10.3 g) and no respondents falling below the lower limit of agreement (-9.68g). A Spearman rank correlation coefficient of r=0.529 (P<0.05) indicates that there was significant proportional bias.

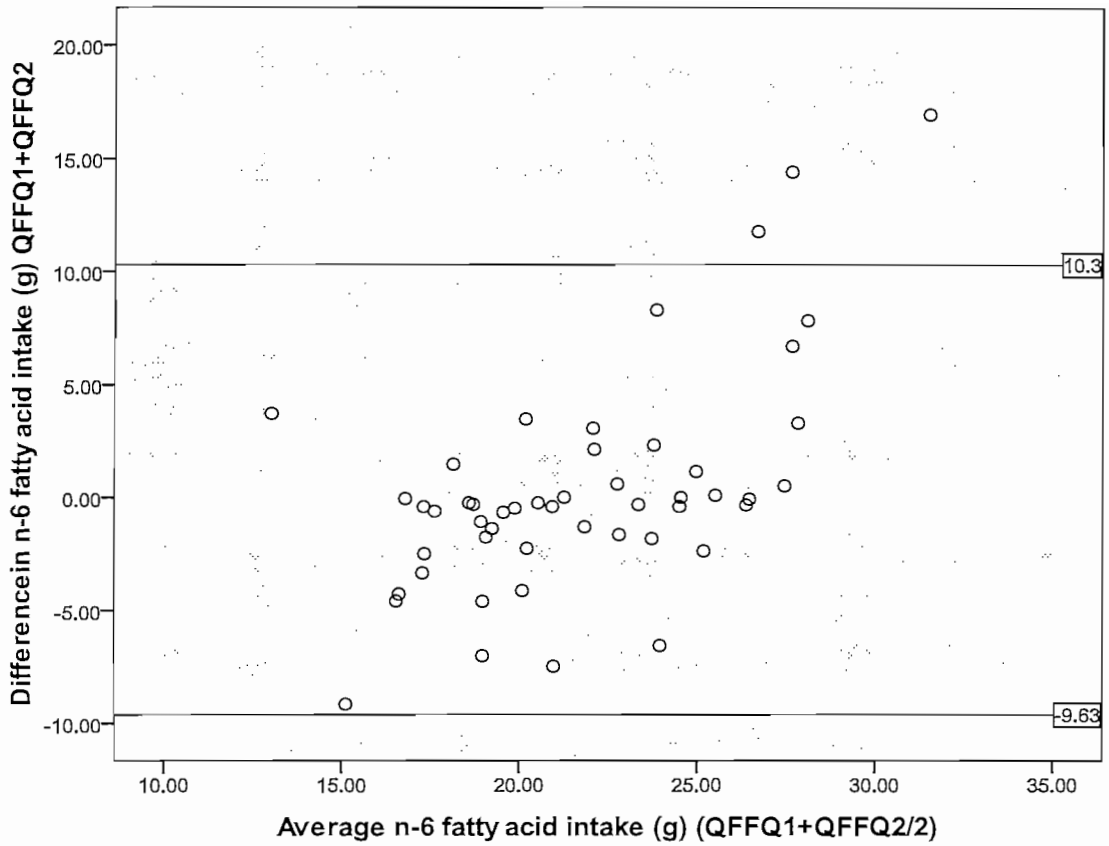

Figure 4.3: Bland-Altman plot for n-6 fatty acid intake (N=50)

Figure 4.4 shows one respondent falling above the upper limit of agreement (0.21g) and no respondents falling below the lower limit of agreement (-0.33g) for n-3 fatty acids. A Spearman rank correlation coefficient of $r=0.38$ ($P<0.05$) indicates that there was significant proportional bias.

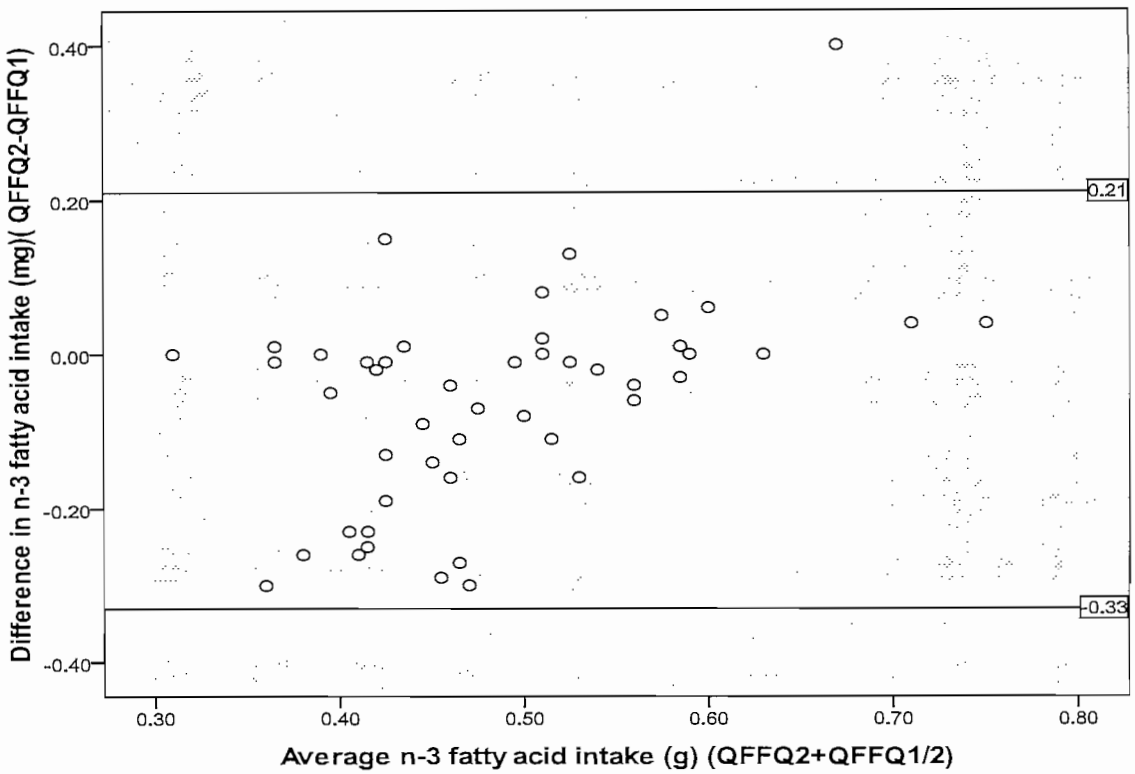

Figure 4.4: Bland-Altman plot for n-3 fatty acid intake (N=50)

4.3.1.1.4 Comparisons of distributions in quartiles between administrations of the QFFQ

In this statistical analysis, reproducibility is determined by comparing the positions of subjects within the distribution of the two administrations of the QFFQ. The distributions of each nutrient for each method are divided into quarters. The proportion of respondents in the same or adjacent quartiles of the distribution on both measures indicates the similarity in ranking of the methods (Table 4.5).

Table 4.5: Classification of respondents into the same and adjacent quartiles of the distribution for the two administrations for the QFFQ (N=50)

Quartile classification				
	Same or adjacent quartiles		Extreme opposite quartiles	
Nutrient	N	%	N	%
Energy	32	64	18	10
Total protein	37	74	13	8
Total fat	41	82	9	4
SAT FAT	40	80	10	2
MUFAs	41	82	9	4
PUFAs	46	92	4	2
n-6	42	84	8	2
n-3	37	74	13	6
Cholesterol	45	90	5	4
Total carbohydrate	33	66	17	14
Calcium	43	86	7	6
Iron	37	76	13	2
Zinc	47	94	3	4
Vitamin A	43	86	7	4
Thiamin	41	82	9	2
Riboflavin	41	82	9	4
Niacin	45	90	5	4
Vitamin C	45	90	5	4

SAT FAT= saturated fatty acids; MUFAs=monounsaturated fatty acids;
PUFAs=polyunsaturated fatty acids

The proportion of individuals classified by both administrations of the QFFQ in the same or adjacent quartiles for energy and macronutrient intake ranged from 64% (energy) to 92% (PUFAs). Slightly higher levels of agreement were observed for micronutrients, ranging from 76% (iron) to 94% (zinc). Misclassification into extreme opposite quartiles ranged from 2% to 10%, except for total carbohydrate (14%). The results in Table 4.5 show that more than 50% of respondents were classified in the same or adjacent quartiles on both administrations for intakes of all nutrients and, with the exceptions of energy and total carbohydrate, less than 10% of respondents were classified into extreme quartiles.

4.3.1.1.5 Misreporting of energy intake

Table 4.6 presents the results of the mean and SD of the percentage by which the sample under- and over-reported energy intakes in comparison with the EER. The calculation of the percentage under- and over-reporting is the reported energy intake expressed as a percentage of EER, where (100 x (EER-reported EI/EER).

Table 4.6: Mean and SD of the percentage of under- and over-reporting of energy intakes derived from QFFQ1 and QFFQ2 (N=50)

		*N (%)	Mean (SD) %	Men N	Mean (SD) %	Women N	Mean (SD) %
Under-reporting	QFFQ1	44 (88)	25.4 (14.5)	21	30.8 (13.8)	23	20.6 (13.6)
	QFFQ2	39 (78)	20.5 (10.9)	21	23.9(10.9)	18	16.6 (9.8)
Over-reporting	QFFQ1	6 (12)	17.9 (13.9)	1	21(0)	5	17.3(13.9)
	QFFQ2	11 (22)	11.8 (9.6)	1	49(0)	10	12.5(9.9)

*N = respondents for whom reported energy intake was less/more than Estimated Energy Requirement

Table 4.6 shows that the reported energy intake of the majority of respondents was lower than their EER in both the QFFQ administrations. For the total sample, the percentage by which energy intake was under-reported was slightly higher in the first QFFQ administration than in the second QFFQ, in that 88% of respondents under-reported in the QFFQ1 by a mean of 25.4% as compared with 78% of respondents who under-reported by a mean of 20.5% in the QFFQ2. A similar pattern was presented by the women. Although the number of men who reported lower energy intakes than the EER was the same for both administrations, the percentage by which men under-reported energy intake was lower on the second administration. There were no significant differences between the percentages of under-reporting between administrations or between genders ($P>0.05$). There was a small percentage of respondents who reported energy intakes higher than the EER, with most coming from the QFFQ2, mainly women.

Table 4.7 presents the number and percentages of respondents classified as under-reporters, where individuals were categorised as under-reporters using calculated cut-offs as discussed under section 4.2.3, where the EER calculated is age- and sex-

specific and takes into account physical activity level. The CVs for reported EI were 21.5% and 12% for the men for QFFQ1 and QFFQ2 respectively and 21.3% and 13.7% for the women. The cut-offs for identification of under-reporters were 70% and 78% of the EER for both men and women for the first and second administrations of the QFFQ respectively.

Table 4.7: Percentage of men and women classified as under-reporters by the QFFQ (N=50)

	Men Cut-off % EER n=22	N	%	Women Cut-off % EER n=23	N	%
QFFQ 1	70%	11	50	70%	7	30.4
QFFQ 2	78%	12	54.5	78%	5	21.9

EER= Estimated Energy Requirements

Table 4.7 shows that there was a larger percentage of men than women classified as under-reporters for both the administrations of the QFFQ, with the difference being statistically significant for the second administration (P=0.028).

4.3.2 Comparative validity

Comparative validity of the QFFQ was tested against the reference method, multiple 24-hour recalls, using a number of statistical methods.

4.3.2.1 Spearman rank and Pearson correlation coefficients

The usefulness of correlation coefficients in the determination of comparative validity has received much deliberation (Bland & Altman, 1986). However, the method has been used extensively in the past and is thus presented here for the purposes of comparison with other statistical methods. All non-normally distributed data were $\log_e$ transformed before the statistical analyses of the Pearson correlation coefficients and were adjusted for within- and between-respondent variations in the three 24-hour recalls, as discussed in section 4.2.3. Table 4.8 presents the Spearman rank and the Pearson correlation and log *e* transformed correlation coefficients between the average reported intakes of the three 24-hour recalls and the reported intake from the QFFQ for the whole sample.

Table 4.8: Spearman rank, Pearson and adjusted Pearson correlation coefficients between the average intakes of three 24-hour recalls and intakes derived from the QFFQ (N=50)

Nutrient	Spearman r	P-value	Pearson r†	P-value	†Adjusted Pearson r
Energy	0.293	0.039*	0.265	0.063	0.284
Total protein	-0.054	0.711	0.016	0.912	0.016
Total fat	0.123	0.390	0.142	0.326	0.153
SAT FAT	0.374	0.007*	0.333	0.018*	0.430
MUFAs	0.328	0.020*	0.288	0.071	0.312
PUFAs	0.032	0.824	0.029	0.841	0.036
n-6	0.054	0.712	0.053	0.264	0.048
n-3	0.123	0.393	0.110	0.457	0.113
Cholesterol	0.245	0.085	0.500	0.000*	0.556
Total carbohydrate	0.414	0.003*	0.393	0.005*	0.405
Calcium	0.421	0.002*	0.371	0.008*	0.404
Iron	0.022	0.879	-0.008	0.956	-0.008
Zinc	0.043	0.767	0.080	0.597	0.087
Vitamin A	0.077	0.593	0.041	0.776	0.047
Thiamin	0.041	0.779	0.067	0.645	0.074
Riboflavin	0.047	0.748	-0.005	0.970	-0.006
Niacin	0.097	0.499	0.157	0.276	0.169
Vitamin C	0.426	0.002*	0.467	0.001*	0.491
Vitamin B6	-0.094	0.516	0.031	0.883	0.041

Level of significance: *P<0.05, r>0.4;†log *e* transformed; SAT FAT= saturated fatty acids; MUFAs=monounsaturated fatty acids; PUFAs=polyunsaturated fatty acids

There appeared to be a weak-to-moderate correlation between the two instruments for all nutrients, as presented in Table 4.8. The Spearman rank correlation coefficients were significant (P<0.05) for energy, saturated fatty acids, monounsaturated fatty acids, total carbohydrates, calcium and vitamin C, whereas the Pearson rank correlation coefficients were significant for saturated fatty acids, cholesterol, total carbohydrate, calcium and vitamin C. The strongest correlations were for vitamin C, calcium and total carbohydrate, while vitamin B6 showed the weakest correlation for the Spearman rank correlation coefficients. The strongest correlation for the Pearson rank correlation coefficients was for cholesterol, while iron showed the weakest correlation. A correlation of r > 0.4 is taken as acceptable (Masson *et al.,* 2002). Adjustment for within- and between-respondent variation resulted in a slight, but non-significant

(P>0.05) strengthening of the Pearson correlation coefficients for 16 of the nutrients tested. However, the correlation coefficient for protein remained the same and n-6 fatty acids decreased.

4.3.2.2 Paired t-test

In order to test the comparative validity of the QFFQ, a comparison was made between the mean reported intakes of the average of the three 24-hour recalls and the mean of the QFFQs (Table 4.9).

For the entire sample, the average reported nutrient intakes of the three 24-hour recalls were higher than those of the QFFQ, with the exceptions of n-6 fatty acids, iron, thiamin and vitamin B_6. The mean percentage difference for the entire sample was 9.7%, with total protein, saturated fatty acids, n-3 fatty acids, cholesterol, total carbohydrate, calcium, iron, vitamin C, thiamin, riboflavin and vitamin B_6 showing differences within 10%. Only energy and PUFAs showed statistical significance.

Table 4.9: **Median (25^{th}; 75^{th} percentile), mean (95% confidence interval), standard deviation, mean difference and percentage difference between the average of three 24-hour recalls and the QFFQ (N=50)**

	Average of the three 24-hour recalls			QFFQ					
Nutrient	**Median (25^{th};75^{th})**	**Mean (95% CI)**	**SD**	**Median (25^{th};75^{th})**	**Mean (95% CI)**	**SD**	**†Mean Difference ††(%)**	**SD Difference**	**P-value (paired t-test)**
Energy (kJ)	7637 (6651;9269)	7955 (7410;8500)	1920	7333 (5987;8140)	7210 (6769;7649)	1548	746 (12.8)	2122	0.027*
Total protein (g)	59.6 (47.3;73.3)	60.2 (55.6;64.8)	16.1	54.4 (44.0;67.1)	56.7 (52.1;61.4)	16.2	3.5 (5.9)	23.3	0.296
Total fat (g)	74.9 (59.5;96.3)	79.0 (70.7;87.4)	29.4	67.1 (55.3;78.0)	68.1 (63.4;72.8)	16.6	11.0 (14.8)	32.0	0.114
SAT FAT (g)	18.4 (13.8;24.7)	19.7 (17.7;21.7)	6.9	19.0 (15.0;21.2)	19.0 (17.5;20.6)	5.4	0.7 (3.6)	7.23	0.959
MUFAs (g)	20.5 (15.7;25.6)	21.0 (18.9;23.0)	7.1	18.5 (14.8;22.0)	18.9 (17.4;20.3)	16.4	2.1 (10.5)	7.6	0.187
PUFAs (g)	27.6 (18.0;42.8)	31.0 (26.3;35.6)	16.4	22.5 (19.1;26.3)	23.2 (21.5;24.9)	6.0	7.8 (28.7)	17.0	0.036*
n-6 (g)	25.6 (16.2;40.5)	29.4 (24.8;34.1)	16.4	21.0 (18.0;24.9)	21.9 (20.2;23.5)	5.8	7.5 (29.2)	16.9	0.055
n-3 (g)	0.38 (0.31;0.48)	0.41 (0.36;0.46)	0.18	0.43 (0.36;0.53)	0.45 (0.41;0.49)	0.14	-0.04 (-9.3)	0.21	0.167
Cholesterol (mg)	192.8 (183.3;285.5)	220.9 (183.3;258.5)	132.3	195.7 (137.4;255.1)	207.1 (180.4;233.7)	98.7	13.8 (6.4)	143	0.726
Total carbo-hydrate (g)	212.1 (181.8;245.2)	214.7 (200.3;229.1)	50.7	205.1 (161.6;236.5)	201.1 (186.8;215.5)	50.4	13.6 (6.5)	54.0	0.080

	Average of the three 24-hour recalls			QFFQ					
Nutrient	**Median (25^{th};75^{th})**	**Mean (95% CI)**	**SD**	**Median (25^{th};75^{th})**	**Mean (95% CI)**	**SD**	**†Mean Difference ††(%)**	**SD Difference**	**P-value (paired t-test)**
Calcium (mg)	421.5 (317.4;534.2)	441.0 (395.3;486.7)	160.8	415.0 (334.3;516.8)	432.0 (388.1;475.2)	153.3	9.3 (2.0)	178.2	0.847
Iron (mg)	9.5 (7.0;11.0)	9.3 (8.6;10.1)	2.6	9.3 (7.8;11.1)	9.7 (9.6;10.4)	2.9	-0.23 (-4.2)	3.9	0.732
Zinc (mg)	7.6 (6.3;9.5)	8.0 (7.3;8.6)	2.3	6.8 (5.8;8.7)	7.2 (6.7;7.8)	2.1	0.82 (10.7)	3.02	0.089
Vitamin A (mcg)	378.8 (286.4;623.0)	760.9 (396.3;1125.5)	1282.8	507.1 (383;712)	598.2 (506;691)	326.0	162.7 (23.9)	1341	0.374
Thiamin (mg)	0.71 (0.57;0.88)	0.73 (0.66;0.78)	0.21	0.81 (0.63;0.93)	0.80 (0.74;0.87)	0.23	-0.07 (-9.12)	0.3	0.067
Riboflavin (mg)	0.92 (0.67;1.17)	1.04 (0.83;1.25)	0.74	1.0 (0.74;1.21)	1.03 (0.92;1.15)	0.40	0.01 (0.9)	0.83	0.472
Niacin (mg)	17.6 (11.9;20.3)	16.5 (14.8;18.2)	5.9	14.3 (12.7;16.5)	14.6 (13.4;15.8)	4.2	1.9 (12.2)	6.95	0.181
Vitamin C (mg)	65.8 (39.1;125.6)	95.4 (70.5;120.2)	87.4	71.0 (45.8;118.0)	94.3 (73.1;115.5)	74.5	1.1 (1.1)	80.7	0.5275
Vitamin B_6 (mg)	1.07 (0.83;1.34)	1.07 (1.00;1.19)	0.34	1.09 (0.87;1.33)	1.13 (1.02;1.22)	0.41	-0.06 (-5.4)	0.52	0.741

†

¶SD= standard deviation;†Difference= average intake of the three 24-hour food recalls – intake of QFFQ;††% difference = (average intake the three 24-hour food recalls – intake of QFFQ) / (average of the three 24-hour recalls + QFFQ1)/2)*100; $p<0.05$; SAT FAT= saturated fatty acids; MUFAs=monounsaturated fatty acids; PUFAs=polyunsaturated fatty acids

4.3.2.3 *Agreement between the average of three 24-hour recalls and the QFFQ (Bland-Altman method)*

Table 4.10 presents a summary of agreement between the average of the three 24-hour food recalls and the QFFQ.

Table 4.10: Summary of agreement between the average of the three 24-hour recalls and the QFFQ (N=50)

		Limits of agreement		
Nutrient	**Spearman r**	**Lower limit**	**Upper limit**	**P-value for Spearman r**
Energy	0.245	-3498	6366	0.086
Total protein	0.57	-43.1	69.9	0.000*
Total fat	0.667	-53	96	0.000*
SAT FAT	0.382	-13.8	21.7	0.006*
MUFAs	0.381	-13.1	22.8	0.000*
PUFAs	0.733	-26.2	51	0.000*
n-6	0.738	-26.3	50.7	0.000*
n-3	0.232	-0.46	0.63	0.104
Cholesterol	0.633	-272.2	429	0.000*
Total carbohydrate	-0.003	-94.4	162	0.98
Calcium	0.054	-347.1	534.6	0.707
Iron	-0.01	-8.03	11.7	0.95
Zinc	0.179	-5.31	9.06	0.213
Vitamin A	0.144	-2519.3	4023	0.319
Thiamin	-0.01	-0.68	0.9	0.948
Riboflavin	0.07	-1.65	2.49	0.615
Niacin	0.44	-12	20.85	0.001*
Vitamin C	0.22	-160.3	242.1	0.122
Vitamin B_6	-0.03	-1.073	1.56	0.848

Level of significance: *P<0.05; SAT FAT= saturated fatty acids; MUFAs=monounsaturated fatty acids; PUFAs=polyunsaturated fatty acids

As can be seen from Table 4.10, significant proportional bias was present for eight of the 19 nutrients, namely total protein, total fat, saturated fatty acids, MUFAs, PUFAs, n-6 fatty acids, cholesterol and niacin.

Bland-Altman scatter-plots provide a method of assessing whether the difference between the QFFQ and the 24-hour recall is the same across the range of intakes and whether the extent of agreement differs for low intakes when compared with high intakes, by plotting the difference between the QFFQ and the 24-hour recall against the average of the two methods. Figures 4.5 to 4.8 illustrate the Bland-Altman plots for energy, cholesterol, total fat and protein.

Figure 4.5 illustrates that for energy there was a fairly even spread of intake from 4 000 kJ to 11 000 kJ, with most points being between 6 000 and 8 000 kJ. There was also a tendency for the differences to get bigger as the mean intake increased, which is consistent as the difference for the highest intake was just below 4 000 kJ; however, the Spearman rank correlation coefficient between the mean and the difference of intakes was not significant (P>0.05). There was one outlier below -3 498 kJ and none above 6 366 kJ.

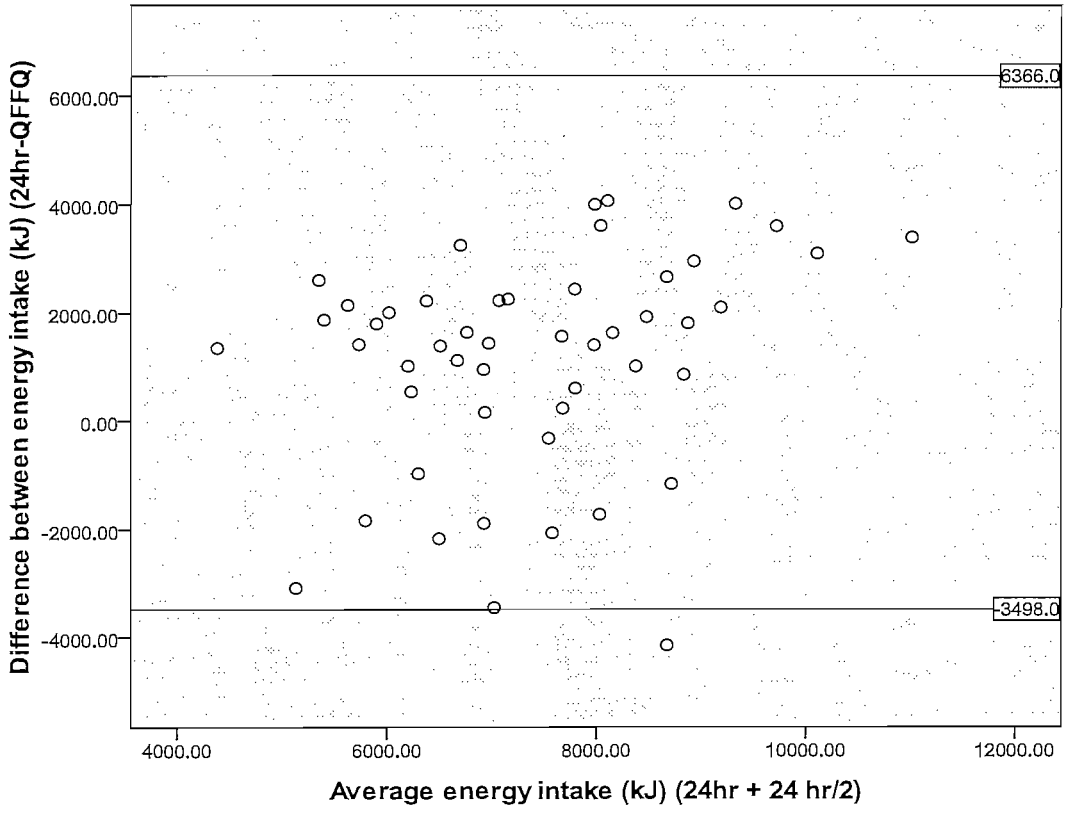

Figure 4.5: Bland-Altman plot for energy

In figure 4.6 the largest spread of cholesterol intake lay between 100 and 400 mg. There was also a tendency for the differences to get bigger as the mean intake increased, which is consistent as the difference for the highest intake was just below 300 *mg* and the Spearman rank correlation coefficient between the mean and the difference of intakes was statistically significant. There were no outliers below -272.2 mg and above 429 mg.

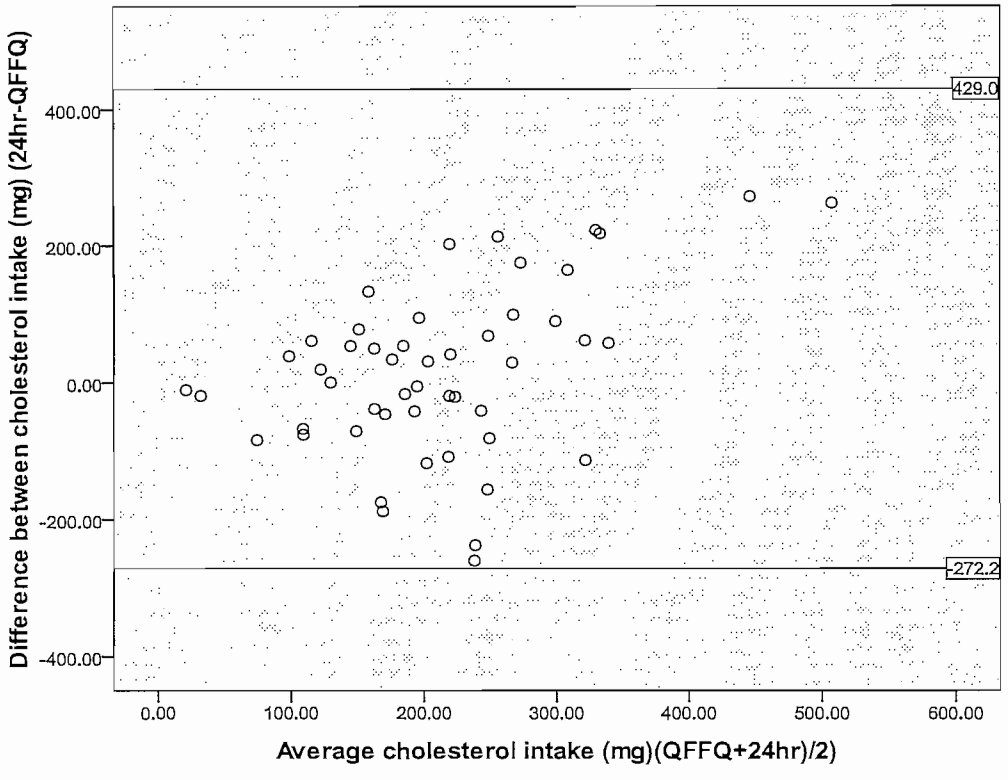

Figure 4.6: Bland-Altman plot for cholesterol

In Figure 4.7 the largest spread of fat intake lay between 60 and 80 g. There was also a tendency for the differences to get bigger as the mean intake increased, which is consistent as the difference for the highest intake was just below 100 g and the Spearman rank correlation coefficient between the mean and the difference of intakes was significant. There were no outliers below the lower limit of -53 g or above the upper limit of 96 g.

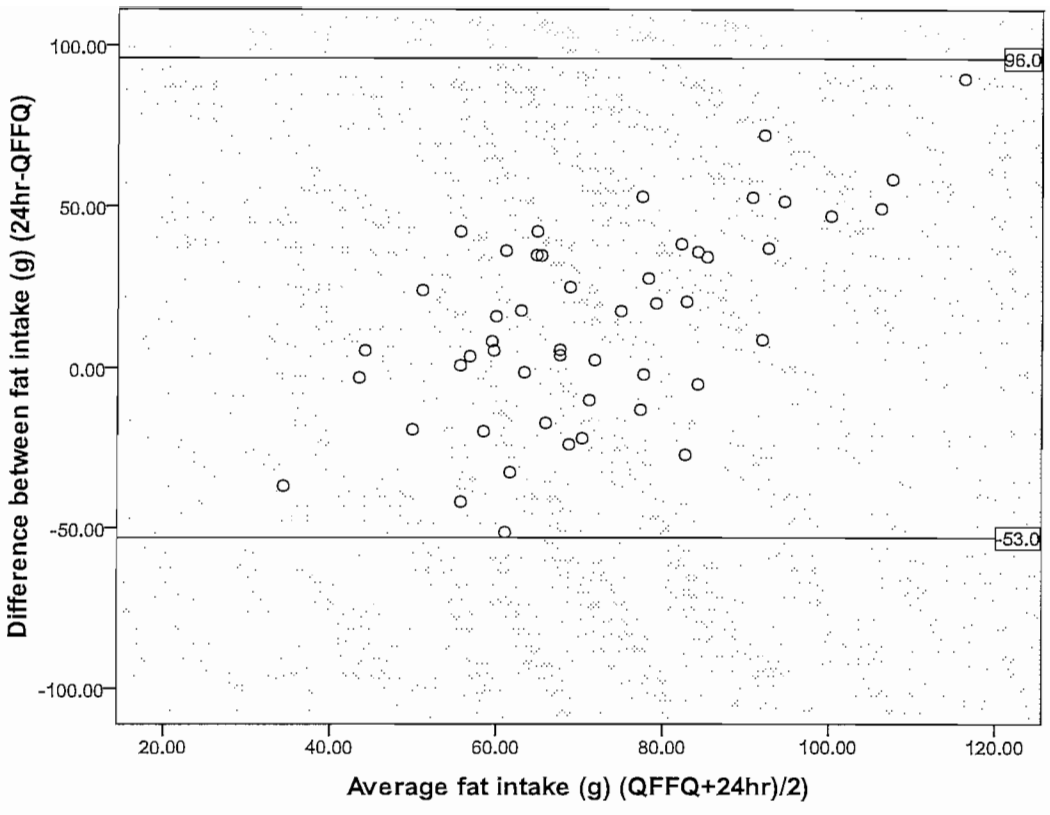

Figure 4.7: Bland-Altman plot for fat intake

Figure 4.8 illustrates that for protein intake, most points were between 50 and 70 g. The Spearman rank correlation coefficient between the mean and the difference of intakes was significant ($P<0.05$). There were two outliers below the lower limit of -43.1 g and none above the upper limit of 69.9 g.

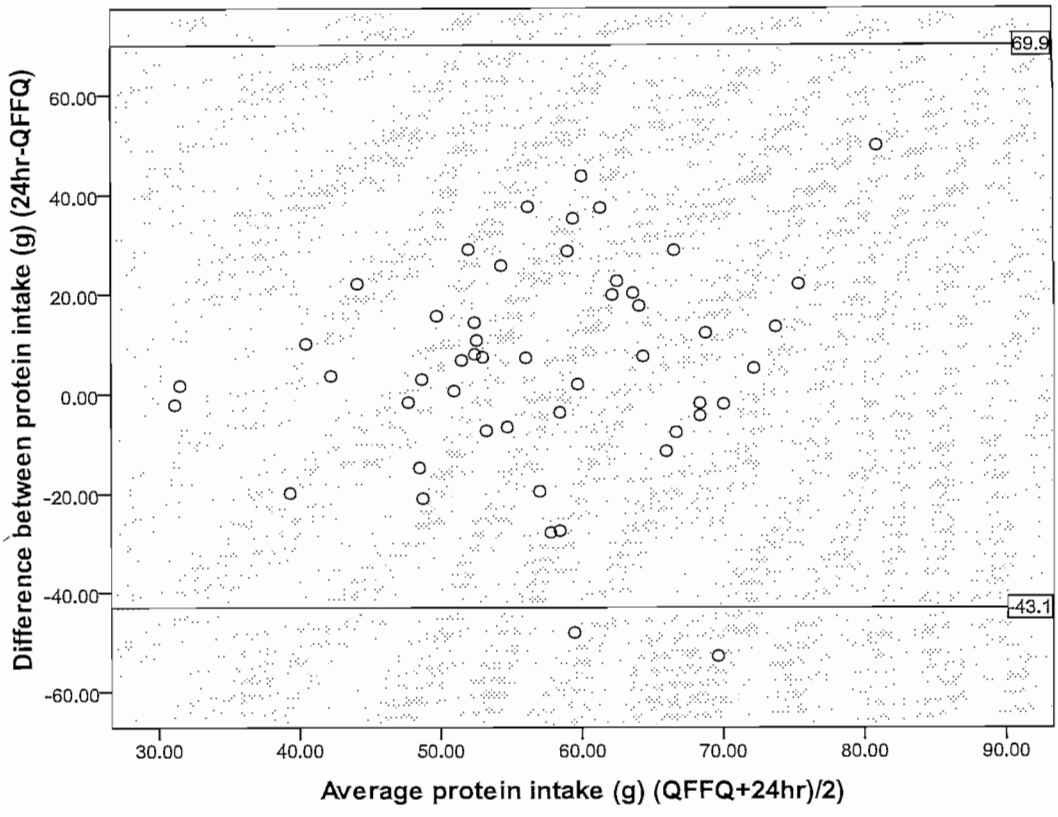

Figure 4.8: Bland-Altman plot for protein

4.3.2.4 Comparison of quartile distributions between the average of three 24-hour recalls and the QFFQ

The distributions of each nutrient for each method were divided into quartiles (Table 4.11).

According to Table 4.11, more than 50% of respondents were classified in the same or adjacent quartiles for all nutrients. The proportion of individuals who were classified by the QFFQ and the average of three 24-hour recalls in the same or adjacent quartiles for energy and macronutrient intake ranged from 64% (total protein) to 80% (saturated fat). Slightly lower levels of agreement were observed for micronutrients, ranging from 62% (vitamin B_6) to 74% (vitamin C). The proportion classified into the extreme opposite quartiles ranged from 2% to 18%, where total protein had the highest percentage of misclassification.

Table 4.11: Classification of respondents into the same and adjacent quartiles of the distribution for the average of three 24-hour recalls and QFFQ (N=50)

	Quartile classification			
Nutrient	**Same or adjacent quartiles**		**Extreme opposite quartiles**	
	N	%	N	%
Energy	38	76	4	8
Total protein	32	64	9	18
Total fat	32	64	3	6
SAT FAT	40	80	4	8
MUFAs	36	72	4	8
PUFAs	32	64	7	14
n-6	34	68	6	12
n-3	35	70	6	12
Cholesterol	37	74	6	12
Total carbohydrate	38	76	1	2
Calcium	31	62	3	6
Iron	32	64	8	16
Zinc	27	54	7	14
Vitamin A	35	70	6	12
Thiamin	32	64	7	14
Riboflavin	34	68	5	10
Niacin	32	64	5	10
Vitamin C	37	74	1	2
Vitamin B_6	31	62	7	14

SAT FAT= saturated fatty acids; MUFAs=monounsaturated fatty acids; PUFAs=polyunsaturated fatty acids

4.3.2.5 Misreporting of energy intake

Table 4.12 presents the results of the mean and SD of the percentage by which the reported EI < than EER for the average of three 24-hour recalls for the total group, and for men and women separately. The calculation of the percentage of under- and over-reporting is the reported energy intake expressed as a percentage of EER [100 x (EER-reported EI/EER)].

Table 4.12: Mean and SD of the percentage of under-reporting of energy intakes derived from the average of the three 24-hour recalls (N=50)

	Total *N	Mean (SD) %	Men N	Mean (SD) %	Women N	Mean (SD) %
Ave of 3 x 24-hrs	37	22.8 (14.1)	18	22.6 (14.5)	19	23.0 (14.0)

Ave = average; *N= respondents for whom average reported energy intake was less than the Estimated Energy Requirements

Table 4.12 shows that, using the average of the three 24-hour recalls, slightly fewer respondents (37) reported energy intakes lower than the EER, in comparison with the QFFQ (44) (Table 4.6) (P>0.05). The mean percentage of under-reporting (22.8%) was not significantly different from that for the QFFQ (25.4%). Likewise, there were no statistically significant differences between the percentages of under-reporting of the men and the women.

Table 4.13 presents the percentage of respondents that were classified as under-reporters for the 24-hour recall, where individuals were categorised as under-reporters using calculated cut-offs of 70% of the EER for the men and 68% for the women, as discussed in section 4.2.3. The CVs for reported energy intakes were 21% and 24% for the men and women respectively. Possible under-reporters were identified as respondents whose reported energy intake, expressed as a percentage of the EER, fell below (100-2SD) the EER (Rennie *et al.,* 2007).

Table 4.13: Percentage of men and women classified as under-reporters by the average of three 24-hour recalls (N=50)

	Men Cut-off % EER	N	%	Women Cut-off % EER	N	%
Ave of 3 x 24-hrs	70%	5	22.7	68%	4	14.3

Ave = average, EER = Estimated Energy Requirements

Table 4.13 shows that a larger, but not statistically significant, percentage of men than of women could be classified as under-reporters, using the average of the three 24-hour recalls. In comparison with the QFFQ (Table 4.7), the percentage of men classified as under-reporters by the QFFQ was significantly more (50%) than that of the average of the three 24-hour recalls (P=0.006). Although the percentage of women classified as under-reporters on the QFFQ (30.4%) was higher than on the 24-hour recalls, the difference was not statistically significant (P>0.05).

4.4 DISCUSSION

Comparison of reproducibility and comparative validity results is often difficult because of the many methodological and analytical differences between studies. Similarly, this

is the case for the present study, as there is no absolutely comparable population in the literature review. Reasonable comparisons could be made with studies from the Indian sub-continent, studies involving migrant Indian populations, studies in South Africa and the general results of reproducibility and validity studies. However, it must be borne in mind that the South African Indian population has a very complex and unique dietary intake pattern which has been transformed through adaptation to South Africa in terms of diverse cultural exposure, food availability and economic conditions, as discussed in Chapter 2. It is therefore imperative that this is taken into account during the discussion and interpretation of the results of all the statistical techniques.

4.4.1 Reproducibility of reported nutrient intakes

In terms of reproducibility of nutrients, the results of this study will be compared with those of other similarly structured studies. This study tested the reproducibility of the QFFQ over a very short period of time (three to five weeks) and will be compared with studies with a similar interval between administrations.

According to Willett (1994), correlation coefficients comparing nutrients have been in the range of 0.4 to 0.7 for a wide variety of populations and with the use of food frequency questionnaires that have varied in length and detail. Similarly, in a review carried out by Cade *et al.* (2001) on the development, validation and utilisation of FFQs from 1980 to 1999, correlation coefficients of 0.5 to 0.7 between two administrations were common. In this study the strongest Spearman rank correlation coefficient was for cholesterol (0.76) and the weakest non-significant correlation was for total carbohydrate (0.153), with the remaining coefficients varying between 0.2 and 0.6. The strongest Pearson correlation coefficient was for cholesterol (0.80) and the weakest correlation was for energy (0.178), with the remaining correlation coefficients varying from 0.2 to 0.6. Collectively, the range of correlation coefficients (0.17 to 0.8) was lower than those of similar studies; however, it was closest to those obtained in the study on dietary transition of Africans (THUSA) (0.14 to 0.75) (MacIntyre *et al.,* 2001a). A possible reason as to why cholesterol had the highest correlation coefficient is that it is derived from a small number of foods. Munger *et al.* (1992) stated that dietary intakes derived from only a few foods are easier to recall. Although the correlation coefficients obtained in the present study were lower than reported by most studies, they show similar trends (Kelemen *et al.,* 2003; MacIntyre *et al.,* 2001a). In the study conducted by Kelemen *et*

al. (2003) on the development and evaluation of a cultural FFQ for South Asians, Chinese and Europeans in North America, the energy-adjusted de-attenuated correlation coefficients ranged from 0.32 to 0.73 (South Asians), 0.17 to 0.84 (Chinese) and 0.30 to 0.83 (Europeans). With regard to South African studies, the THUSA study on Africans in the North-West Province, with a time period of eight to twelve weeks between administrations, showed correlation coefficients of 0.31 (energy), 0.31 (protein), 0.22 (vitamin A) and 0.20 (iron) (MacIntyre *et al.,* 2001a).

One must be guarded in the interpretation of the results of the correlation coefficient in terms of its limitations. According to Delcourt *et al.* (1994), a weak Pearson correlation coefficient could reflect a smaller range of intakes rather than weaker agreement when compared to other studies. On the other hand, an extreme value does not affect the Spearman rank correlation coefficient.

With regard to the differences between the reported intakes of the two administrations of the QFFQ, 13 of the 19 nutrients tested fell within a 10% difference, thus showing good agreement (Wheeler *et al.,* 1994). The mean reported intake of the second administration was higher than the first, with the exception of PUFAs, n-6 fatty acids and vitamin C. A similar trend was reported by MacIntyre *et al.* (2001a) and Mannisto *et al.* (1996), but differs from other studies where the first administration was higher than the second (Willett *et al.,* 1985; Lindroos *et al.,* 1993). With regard to energy intake, the percentage difference between measurements in the present study (8.0%) was slightly larger than that reported by MacIntyre *et al.* (2001a) over a short interval but slightly smaller than the differences obtained by Wheeler *et al.* (1995) (11.2 to 13.6%).

When the results of the Spearman rank and Pearson correlation coefficients are compared with the percentage difference, the Spearman rank correlation coefficients are weak yet significant, implying an agreement between the two measurements. In contrast, the mean reported intake did not differ significantly, implying that in the comparison between the two administrations, the means were fairly consistent. However, it must be borne in mind that the Spearman correlation coefficient measures linear relationships between rankings of the variables whilst the comparison of means compares group performance of the measurement. This is highlighted by the conflicting results for cholesterol, which has the highest correlation coefficient but does not fall within the 10% difference when means are compared. A possible reason for this is that

cholesterol is found in animal products, and the fact that Indians (Hindus) normally abstain from meat for at least three days per week for religious purposes could have contributed towards this difference in results.

In an attempt to further clarify the agreement between the two administrations of the QFFQ, the Bland-Altman procedure (Bland & Altman, 1986) was used, where it was found that there was significant proportional bias for all nutrients except saturated fatty acids.

In the attempt to determine whether the QFFQ was able to place a respondent in the same quartile of intake on both administrations, the proportion of individuals classified in the same or adjacent quartiles for energy and macronutrient intake ranged from 64% (total energy) to 92% (PUFAs) in total, showing relatively good agreement. The highest level of agreement was for PUFAs, followed closely by cholesterol, where both also had relatively good correlation coefficients and fell within the 10% difference when means were compared.

4.4.2 Misreporting of energy intake

According to Macdiarmid and Blundell (1998), under-reporting of food intake is one of the fundamental obstacles preventing the collection of accurate habitual dietary intake data. In terms of reliability of the QFFQ, using 70% of the EER as the cut-off for the QFFQ1, 50% of men and 25% of women under-reported, and when using the 78% cut-off for the QFFQ2, 54.5% of men and 17.9% of women under-reported. A possible reason why more men under-reported could be that men may find it difficult to determine portion sizes effectively, as Indian men are generally not involved in food preparation. Similarly, in a review on dietary intake methodology on immigrant European population groups by Ngo *et al.* (2009), quantification of specific portion sizes of traditional foods was problematic. However, body weight status was observed in several Asian migrant studies as a trend of under-reporting amongst overweight individuals (Pomerleau *et al.,* 1999; Jonnalagadda & Diwani, 2002).

4.5 IMPLICATIONS OF THE REPRODUCIBILITY RESULTS

When the reproducibility of a dietary instrument is assessed, it is important that it performs consistently from one administration to another. The Spearman rank correlation coefficients showed that the reproducibility was moderate-to-weak. However, satisfactory reproducibility was reflected by the small differences between the means of each measurement and a high percentage of subjects being classified within the same or adjacent quartiles for both administrations. Further to this, understanding the factors that could have affected reproducibility positively or negatively is equally important. In terms of methodology, a positive effect on reproducibility can be inferred, since the same fieldworker, complying with the same conditions, was responsible for both administrations. It is also important to note that dietary patterns would not have changed during the relatively short period of time between administrations. However, several authors suggested that a number of frequently consumed foods have better reproducibility than other foods (Ajani *et al.,* 1994; Feunekes *et al.,* 1995). Nonetheless, it must re-emphasised that the South African Indian diet is very complex and varied (using foods from the Indian sub-continent and indigenous South African foods) and very similar to the Indian diet from the Indian sub-continent in its characteristic plating and serving of a main dish and a number of side items on a plate (*tari*). From this, it can be extrapolated that the complexity of the diet may affect reproducibility.

A satisfactory reproducibility equates to having small differences between means of each measurement, no proportional bias and a high percentage of individuals classified in the same quartile. Saturated fatty acids performed well on all measures, with other nutrients being consistent on at least two analyses. Considering the factors that could have affected the reproducibility of the instrument and the results that have been presented, it can be concluded that QFFQ was a relatively reproducible dietary instrument, showing consistency.

The results of the Spearman rank correlation and Pearson coefficients, paired t-tests, Bland-Altman technique and quartile distribution are discussed with reference to other published studies for comparative purposes in order to assess the comparative validity of the QFFQ.

4.6.1 Agreement between nutrient intakes derived from the 24-hour recalls and the QFFQ

Energy

There appears to be a moderate-to-weak correlation between the two instruments for all nutrients. The ranges of the Spearman rank correlation coefficients (-0.09 to 0.42) and the Pearson correlation coefficients (0.008 to 0.556) obtained in the present study were similar to ranges reported by MacIntyre *et al.* (2001b) (0.14 to 0.6). The correlation coefficients obtained for energy were 0.293 and 0.284 for the Spearman rank and Pearson respectively, slightly higher than the following reported studies: Larkin *et al.* (1989) for black men (0.23) and Mannisto *et al.* (1996) for women (0.23). However, they are lower than those reported by Thompson and Margetts (1993) (0.38 and 0.36 for men and women respectively), and MacIntyre *et al.* (2001b) (0.31).

Protein

The correlation coefficient obtained for protein in this study was -0.054, which is much lower than that reported for the Cambridge questionnaire (0.13) by Bingham *et al.* (1994). Although the QFFQ, in comparison with the 24-hour food recall, is more representative of the habitual diet, the low correlation coefficient for protein could be due to the fact that only special occasions and days of festivities were excluded during the 24-hour recall administration. The 24-hour recall did not take into account normal abstinence from meat for religious purposes, as these days varied according to deities worshipped in compliance with the Hindu faith and hence averaged three working days in a week. For total carbohydrate, the Spearman correlation coefficient obtained in the present study was 0.414 and the Pearson correlation was 0.405, larger than the value reported by MacIntyre *et al.* (2000b) (0.3). However, it was similar to what was reported by Rimm *et al.* (1992) (0.4) and Lindroos *et al.* (1993) (0.43). The correlation coefficient obtained for fat in this study was 0.123, which was lower than in studies reported by MacIntyre *et al.* (2001b) (0.25) and Larkin *et al.* (1989) (0.23).

Micronutrients

In terms of micronutrients, the strongest correlation was for vitamin C (0.426), which was slightly lower than that reported by MacIntyre *et al.* (2001b) (0.6). The calcium correlation coefficient was 0.42, similar to the range reported in many studies (0.4 to 0.7, Rimm *et al.,* 1992; Thompson & Margetts, 1993). A possible reason for the strong vitamin C correlation is that it is present in a number of foods that form part of the Indian habitual diet.

With regard to studies on South Asians where 24-hour recalls were compared with FFQs, Wang *et al.* (2008) reported correlation coefficients from 0.35 (total fibre) to 0.84 (energy) in men and 0.24 (thiamin) to 0.62 (riboflavin) in women. Sevak *et al.* (2004) conducted another migrant South Asian study using similar instruments, where validity was very reasonable. In that study, the energy-adjusted Pearson correlation coefficients ranged from 0.24 (vitamin A) to 0.73 (protein) and energy-adjusted Spearman correlation coefficients ranged from 0.17 (vitamin A) to 0.76 (carbohydrate). In the study by Norimah and Margetts (1997) on the South Asian community in the UK, where the reference method differed from this study in that the FFQ was compared to the weighed diet record (WR), the correlation coefficients ranged from 0.26 to 0.38 (energy unadjusted) with the following correlation coefficients: 0.38 (carbohydrate), 0.26 (fat), 0.29 (energy) and 0.27 (protein). In relation to the findings of this study, the correlation coefficients for energy were equivalent and the correlation coefficients for carbohydrates were slightly higher. However, the correlation coefficients for fat and protein were much lower than those in the study by Norimah and Margetts (1997). Although, unlike the WR, the QFFQ and 24-hour recall could be influenced by memory recall, the similarities and differences presented in this study, as compared with the study by Norimah and Margetts (1997), could be due largely to ease of quantifying portion sizes. Fat intake would be harder to quantify because of the varying amounts of unsaturated fats used in food preparation, whereas carbohydrate intake would be easier to quantify as portion sizes are standardised and commonly used.

In South Africa, the only validation study so far was in the THUSA study. The following Spearman rank correlations were reported: 0.31 (energy), 0.31 (carbohydrate), 0.30 (protein), 0.22 (vitamin A) and iron (0.2), where the QFFQ was compared with seven-day weighed food records (MacIntyre *et al.,* 2001b).

With regard to the comparison of the results of the correlation coefficients and the mean difference between the two methods, the results are positively comparable, in that saturated fat, MUFAs, carbohydrate, calcium and vitamin C showed significant Spearman and Pearson correlation coefficients and were also within the 10% mean difference between methods.

Level of agreement and proportional bias

In an attempt to further assess the level of agreement and the presence of proportional bias between the reported nutrient intake of the QFFQ and the average of the three 24-hour recalls, the Bland-Altman procedure (Bland & Altman, 1986) was used. In this study, significant proportional bias was present for eight nutrients, namely total protein, total fat, saturated fat, MUFAs, PUFAs, n-6 fatty acids, cholesterol and niacin. Bland-Altman plots for energy, cholesterol and fat intake showed that, as the mean intake increased, the difference between mean of the two QFFQs and the average of the three 24-hour recalls grew larger. However, this pattern was not as pronounced for protein intake.

Misclassification patterns

The proportion of individuals classified in the same or adjacent quartile was relatively high. However, in a comparison with the Bland-Altman plots, a similar pattern of agreement was displayed for the quartile classifications, where protein intake had the highest percentage of misclassification. With reference to Indian migrant studies, the study by Sevak *et al.* (2004) among South Asians in the UK, where the FFQ was compared with the 24-hour recall, the proportion of individuals classified in the same or adjacent quartiles was high, ranging from 65% (vitamin A) to 96% (protein). Misclassification in opposite quartiles was very low (0% to 5%), except for vitamin A (10%). Wang et *al.* (2008) reported that the percentage of the men classified in the same and adjacent quartiles of a 24-food recall and FFQ ranged from 68% (fat) to 90% (vitamin A), with an average of 79%.

4.6.2 Misreporting of energy intake

In terms of under-reporting, when using the 70% cut-off for the QFFQ1, 50% of men and 30% of women respectively under-reported energy intakes, whereas when using the 70% cut-off for the 24-hour recall for men and 68% cut-off for women, 25% of men

and 14.3% of women under-reported on energy intake. The pattern of more men under-reporting again suggests that men are not skilled in determining portion sizes, the density of plated food and the type of fat used in the dish, as they are not primarily involved in food preparation in Indian households.

In some studies, under-reporting has been associated with a number of different subject characteristics such as gender, BMI, age, ethnicity, cultural factors, physical activity and dietary restraint (Lafay *et al.,* 1997; Pomerleau *et al.,* 1999; Mennen *et al.,* 2000). According to Macdiarmid and Blundell (1998), invalid dietary intakes can arise from different forms of behaviour including:

- food being eaten but deliberately NOT reported (intentional under-reporting);
- food consumption being reduced, or certain foods being avoided, during the period of study and
- food being eaten but genuinely forgotten (unintentional / unknowing under-reporting).

Moreover, with under-reporting being related to a number of factors, it is important to note that these factors are unlikely to be mutually exclusive, with each having an effect on under-reporting in varying degrees. Furthermore, in two studies on under-reporting in Sweden and Ireland, it was found that under-reporting was associated with a lower percentage of energy from total fat, saturated fat and carbohydrate, which could be an influential factor in under-reporting (Beekes *et al.,* 1999).

The main aim of the comparative validation was to determine whether the information obtained from the QFFQ was a true reflection of the diet of the Indian community studied. It is important to interpret the results together in order to arrive at a conclusion on the relative validity of the QFFQ. Although this study shows moderate-to-weak validity, it must be borne in mind that the complexity of the Indian habitual diet makes it difficult to determine true intake adequately. Similarly, Wang *et al.* (2008) reported that the assessment of dietary intake in China is difficult because subjects tend to mix multiple elements in one food or dish, which makes accurate estimation difficult. Although recipes were standardised in the present study through focus groups before the administration of questionnaires, it did not reflect true intake in terms of unsaturated fat usage per recipe per household.

The standardised amount of sunflower oil used in food preparation by each respondent does not reflect actual intake, as the amount of sunflower oil used to sauté a dish (which is the primary method of cooking amongst Indians) varies greatly between households. With sunflower oil being the main oil used in most food preparation methods, the household consumption of sunflower oil is approximately five litres per month, depending on the size of the household. To add to the increased consumption of sunflower oil, a variety of vegetable dishes are commonly served as side items to the main dish. These vegetables are predominantly sautéed in sunflower oil and chilli, in contrast to meat, which is self-basting, depending on the cut, and requires slightly less sunflower oil. These preparation methods are fairly common in most Indian households, but the amount of unsaturated fat used differs extensively, making true dietary estimation very difficult even with standardisation, and thus affecting the validity of the study.

Observational studies are the ideal but this type of study also comes with high cost and the possibility of error being introduced in measuring dietary intake, as the environment is generally controlled during the observation and hence individuals who usually have freedom of choice would be restricted in their responses.

4.7 IMPLICATIONS OF THE RESULTS FOR THE MAIN STUDY

Both reproducibility and relative validity are affected by a number of factors, including the nutrients and foods being assessed, the estimation of the amounts consumed, the complexity of the diet and the size and the characteristics of the study sample. In addition, the overall research design, methods and reporting will affect accuracy. The QFFQ is relatively reproducible; however, the study showed moderate-to-poor validity. The reliability and comparative validity study has the following implications for the main study:

- Since there is a possibility of inherent error in portion size estimation owing to the density of the food and the actual type of fat used in the dish, especially in the case of respondents that do not prepare the food themselves, extra care must be taken by the fieldworker to extract this information and care must be taken in the interpretation of the results. It is recommended that, during the administration of the dietary questionnaire, the person involved in the preparation and serving of

food must be present so that accurate estimates of food consumption are obtained in collaboration with the respondent.

- In the study conducted by Iqbal *et al.* (2008), lower correlations were observed which were attributed to participant fatigue, as the 24-hour recalls and the FFQ were administered simultaneously. In this study, the three 24-hour recalls and the two QFFQs were administered within a period of five weeks, so the poor correlations could also be attributed to participant fatigue. Refining the QFFQ in terms of the length will not do justice to the varied dietary patterns of Indians in KZN. It would be more appropriate to spread the administration over a longer period so as to limit respondent fatigue.
- Care must be taken in the design and administration of QFFQs to avoid double counting of foods. For example, error can be introduced in reporting rice intake and rice again in breyani (rice dish) or bread intake and bread again in a 'bunny' (bread and curry dish). To reduce the possibility of error, it is suggested that the fieldworker remind the respondent of foods where doubling can occur.

Although care was taken in the adaptation of the QFFQ to make it culturally sensitive to Indians, dietary assessment methods more sensitive to culture should be further explored.

4.8 CONCLUSION

This chapter described the methods used in the study of the comparative validity and reproducibility of the dietary questionnaire, and presented the results, which were discussed and interpreted in relation to other studies. In Chapter 5, the results of the main study will be presented.

CHAPTER 5

RESULTS OF THE MAIN STUDY

5.1 INTRODUCTION

In Chapter 4, the results of the study of the comparative validation and reproducibility of the QFFQ in the sub-sample were presented and discussed. In this chapter, the results of the main study will be presented. The main study was conducted to determine the prevalence of selected risk markers for NCDs and the association with lifestyle behaviours in apparently healthy Indians (35 – 55 yrs) living in KwaDukuza. The prevalence of risk markers for NCDs (unhealthy diets, smoking, physical inactivity, hypertension, central obesity, increased fasting blood glucose, cholesterol and triglycerides) and the possible associations with current trends in nutrient intakes and dietary patterns, with special reference to the n-6:n-3 fatty acid ratio, were investigated in order to propose strategies for an integrated programme of prevention of NCDs in the target group.

5.2 RESULTS

5.2.1 Sample description

A total of 250 respondents, comprising 111 males and 139 females from the six sub-areas of the KwaDukuza Municipality, was interviewed for the main study. Table 5.1 presents an overview of the socio-demographic characteristics and medical history of the study group. All the respondents completed the study. The mean age of the study group was 45.5 and 43.1 yrs respectively for men and women. Most of the respondents were Hindus and professionals, using the services of private doctors and pharmacies, and covered by medical aid. For the purpose of differentiating between a vegetarian and a non-vegetarian, the definitions of the ADA (2009) were used. According to the ADA (2009), a vegetarian is a person who does not eat meat (including fowl) or seafood, but the eating plans of vegetarians may vary considerably depending whether they are lacto-ovo-vegetarian, pollo-vegetarian or pesco-vegetarian. In the attempt to determine the number of respondents that were vegetarian, all eating plans of

vegetarianism were acknowledged as vegetarian. Using the above definitions, only 6.4% of respondents were vegetarians. There was minimal use of primary health clinics and government hospitals, probably due to the long waits and stigmatised poor service delivery in such institutions of health. A large percentage reported a family history of hypertension (63.2%), myocardial infarction (41.2%) and stroke (31.6%). Only 12.4% of the total group smoked. With regard to alcohol consumption, 30.4% of the total sample had consumed alcohol in the past year. Of this number, 76.3% were men and 23.6% were women. Beer consumption was highest, with 28% of the respondents consuming two beers per week, followed by 17% of the respondents who consumed two shots of brandy and the same percentage consuming two ciders per week.

Table 5.1: Socio-demographic characteristics of the respondents

Variables	N	%
Gender		
Men	111	44.4
Women	139	55.6
Age (yrs)		
35-45	131	52.4
46-55	119	47.6
Mean age	Men: 45.5 (SD 5.4)	Women: 43.1 (SD 5.8)
Religion		
Hindu	215	86
Muslim	30	12
Christian	5	2
Occupation		
Housewives	53	21.2
Blue-collar	79	31.6
Professional	116	46.4
Unemployed	2	0.8
Dietary preference		
Vegetarian	16	6.4
Non-vegetarian	234	93.6
Health service utilisation		
Primary health care clinic	16	6.4
Government hospitals	21	8.4
Private hospitals	11	4.4
Private doctors	55	22.0
Pharmacies	63	25.2
Medical aid cover	144	57.6
Medical history		
Family history of hypertension	158	63.2
	Men: 76	30.4
	Women: 82	32.8
Family history of heart attack	103	41.2
	Men:48	19.2
	Women: 55	22
Tobacco use		
Current smokers	31	12.4
Cigarettes per day	(N=31)	
10 or less	21	67.7
11 to 15	4	12.9
16-20	6	19.3
Alcohol use		
Ever consumed alcohol	77	30.8
Alcohol consumption within the past year	76	30.4
	Men: 58	76.3
	Women: 18	23.6
2 beers per week	Men: 22	28.9
(average 5% alcohol content)	Women: 1	1.3
2 ciders per week	Men: 5	6.5
(average 5% alcohol content)	Women: 8	10.5
2 (25ml x 2) shots brandy	Men 13	17.1
(average 30-42% alcohol content)		

SD = Standard deviation

5.2.2 Biological risk factors

5.2.2.1 Anthropometric profile

Table 5.2 presents the results of the anthropometric profile for the whole study group. The mean weight for the whole study group was 70.2 kg, with men having a mean weight of 75.8 kg and women 65.8 kg. The mean height for the whole study group was 1.6 m; 1.7 m for the men and 1.5 m for the women. The mean WC for the whole study group was 89.6 cm, with men recording a mean WC of 94.0 cm and women 86.1 cm. The mean hip circumference for the whole group was 100.3 cm, with men having a mean hip circumference of 103 cm and women 98.2 cm, while the mean waist: hip ratio was 0.89 for the entire sample, 0.91 for men and 0.87 for women. With regard to percentage of body fat, a close similarity of means for the whole group, men and women was noted. The mean for the whole group was 32.2%, 31.7% for men and 32.6% for women. A similar pattern was noted for the mean BMI. The mean BMI for the whole group was 26.3 kg/m^2, 26.1 kg/m^2 for men and 26.8 kg/m^2 for women.

Table 5.2: Anthropometry for the total group, men and women

	Total group N=250 Mean(SD)	Men N=111 Mean (SD)	Women N=139 Mean (SD)
Weight (kg)	70.2 (11.1)	75.8 (9.3)	65.8 (10.3)
Height (m)	1.6 (0.08)	1.7 (0.07)	1.5 (0.05)
Waist circumference (WC) (cm)	89.6 (6.4)	94.0 (4.7)	86.1 (5.4)
Body mass index (BMI) (kg/m^2)	26.3 (3.8)	26.1 (2.9)	26.6 (4.4)
Hip circumference (cm)	100.3 (5.3)	103.0 (4.3)	98.2 (5.1)
Waist: hip ratio (WHR)	0.88 (0.27)	0.91 (0.19)	0.87 (0.02)
Percentage body fat	32.2 (5.9)	31.7 (0.5)	32.6 (6.3)

SD= standard deviation

Table 5.3 presents the frequency distribution of the anthropometric profile with reference to standard classification systems. Cut-off points for BMI are represented according to the BMI criteria for Asians by the Regional Office for the Western Pacific Region of the WHO (Anuurad *et al.*, 2003). Waist circumference cut-off points were based on the recommendations of the WHO for Asian populations (NCEP/ATP111, as discussed in section 2.5.3.2), defined as ≥90 cm for men and ≥80 cm for women for central obesity. For men, the highest frequencies were recorded in the following ranges: height 1.70 to 1.79 m, weight 70.0 to 79.9 kg, WC 90 to 99 cm and hip circumference 100 to 109 cm. For women, the ranges with the highest frequencies were as follows: height 1.50 to 1.59 m, weight 60.0 to 69.9 kg, WC 80 to 89 cm, and hip circumference 90 to 99 cm. Using the Asian cut-off points to define central obesity, 100% of women had WC ≥80 cm and 87.4% of men had WC ≥90 cm. In terms of BMI and percentage of body fat, both men and women had the highest frequency for obesity class 1 and fell in the 30% to 39% body fat category.

Table 5.3: Frequency distributions of anthropometry profile with reference to standard Asian* classification systems

	Total group N=250	%	Men N=111	%	Women N=139	%
Weight (kg)						
48-49.9	2	0.8	0	0	2	1.4
50-59.9	46	18.4	5	4.5	40	28.7
60-69.9	80	32	24	21.6	56	40.2
70-79.9	73	29.2	49	44.1	24	17.2
80-89.9	38	15.2	26	23.4	12	8.6
90-99.9	11	4.4	6	5.4	2	1.4
Height (m)						
1.45-1.49	2	0.8	0	0	2	1.4
1.5-1.59	99	39.6	7	6.3	72	51.7
1.6-1.69	84	33.6	37	33.3	37	26.6
1.7-1.79	68	27.2	58	52.2	8	5.7
1.8-189	5	2	5	4.5	0	0
1.9-1.99	4	1.6	7	6.3	0	0
WC (cm)						
75-79	8	3.2	0	0	8	5.7
80-89	119	47.6	16	14.4	103	74.1
90-99	109	43.6	85	76.5	24	17.2
100-110	13	5.6	10	9	4	2.8
111-120	1	0.4	0	0	0	0
BMI (kg/m²)						
Underweight						
<18.4	1	0.4	1	0.9	0	0
Normal						
18.5-22.9	29	11.6	6	5.4	23	16.5
Overweight						
23.0-24.9	72	28.8	40	36	32	23
Obesity class I						
25.0-29.9	112	44.8	49	44.1	60	43.1
Obesity class II						
30.0 and >	39	15.6	15	13.5	24	17.2
Hip circumference (cm)						
80-89.9	8	3.2	1	0.9	7	5
90-99.9	105	42	19	17.1	86	77.4
100-109.9	129	51.6	89	80.1	40	36
111-119.9	8	3.2	2	1.8	6	5.4
Waist circumference (central obesity)						
≥90cm,	135	54	97	87.4	121	87.1
80-89 cm	115	46	14	12.6	18	12.9
Percentage body fat						
16-19	3	1.2	0	0	2	1.4
20-29	86	34.4	41	36.9	46	33
30-39	130	52	62	55.8	68	48.9
40-49	31	12.4	8	7.2	22	15.8

*Anuurad *et al.*, 2003; Wasir *et al.*, 2008
WC = Waist circumference; BMI = Body mass index

Figure 5.1 depicts the results of the frequency distribution of WC in a bar graph, where it is clearly shown that 80 to 89 cm was the highest frequency amongst women and 90 to 99 cm was the highest frequency amongst men.

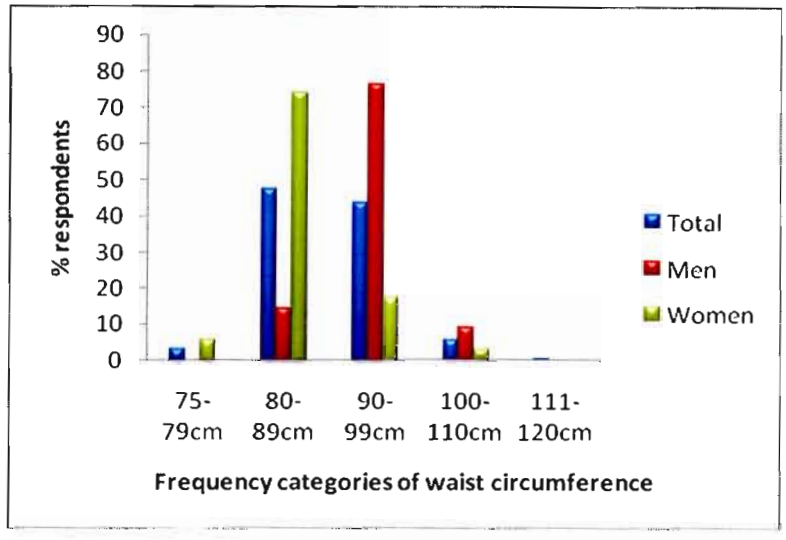

Figure 5.1: Frequency distribution of waist circumference

Figure 5.2 presents the results of the frequency distribution of the percentage body fat of the respondents in the current study in a bar graph, showing that the category of 30 - 39% body fat was the highest frequency in the total group, including both men and women.

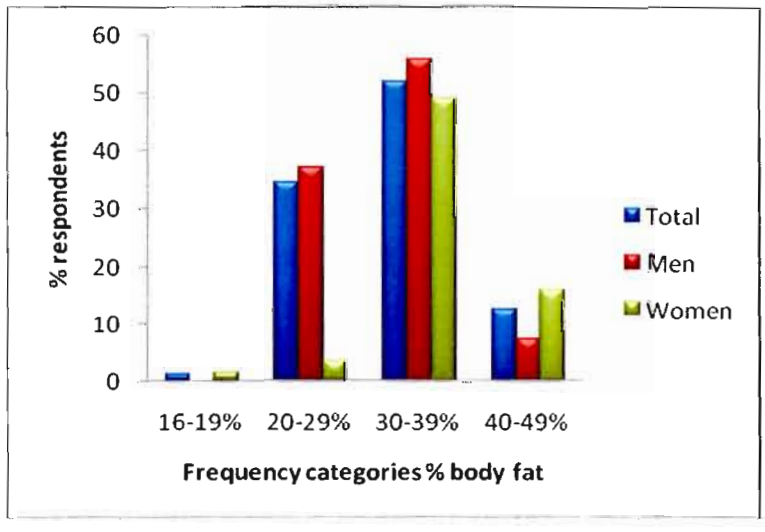

Figure 5.2: Frequency distribution of percentage of body fat

5.2.2.2 *Clinical profile*

In Table 5.4, the results of the clinical parameters are reported for the whole study group, and for men and women separately. The mean recorded SBP for the entire group was 123.4 mmHg. Men recorded a mean of 122.8 mmHg and women 124.0 mmHg, with the means very close to each other. The mean DBP for the whole group was 89.7 mmHg, with a slight variation in men with a mean of 89.3 mmHg, and in women a mean of 90.4 mmHg. Similarly, the means recorded for pulse units, total serum cholesterol and fasting glucose were almost the same for all the groupings, as reflected in Table 5.4. In contrast, the mean triglyceride levels varied between groups with a mean of 2.33 mmol/L for the entire group, 2.24 mmol/L for the men and 2.4 mmol/L for the women.

Table 5.4: Clinical parameters of the total group, men and women

	Total group N=250 Mean(SD)	Men N=111 Mean (SD)	Women N=139 Mean (SD)
SBP (mmHg)	123.4 (8.2)	122.8 (7.0)	124.0 (9.1)
DBP (mmHg)	89.7 (5.7)	89.3 (4.4)	90.4 (6.5)
Pulse units (BPM)	77.6 (5.1)	77.2 (4.2)	77.9 (5.7)
Fasting glucose (mmol/L)	5.5 (0.79)	5.41 (0.63)	5.5 (0.88)
Total serum cholesterol (mmol/L)	5.03(0.78)	5.07 (0.58)	5.01 (0.58)
Triglycerides (mmol/L)	2.3 (0.79)	2.2 (0.61)	2.4 (0.90)

SBP = systolic blood pressure; DBP = diastolic blood pressure; BPM = beats per minute; SD = standard deviation

Cut-off points for clinical parameters used in this study were defined in section 2.6. Impaired fasting blood glucose was defined as >5.55 mmol/L. Hypertension was defined as SBP ≥130 or DBP ≥85 mmHg and hypertriglyceridaemia was defined as >1.69 mmol/L. Table 5.5 presents the frequency distributions of clinical parameters. A SBP >130 mmHg was recorded by 6.3% of men and 6.4% of women. More respondents had DBP measurements above the cut-off value (93.8% of men, 92% of women). The highest recorded frequency for pulse for all subjects ranged from 70 to 79 beats per minute. Thirty-six percent of men and 40.2% women recorded a fasting blood glucose level of >5.55 mmol/L. With regard to cholesterol, 63% of men and 21.5% of women recorded levels >5.2 mmol/L. Both men (91.8%) and women (86.3%) recorded raised triglyceride levels (>1.69 mmol/L).

Table 5.5: Frequency distributions of clinical parameters with reference to cut-off points

Variable	Total group N=250	%	Men N=111	%	Women N=139	%
SBP						
<130 mmHg	234	93.6	104	93.6	130	93.5
>130 mmHg	16	6.4	7	6.3	9	6.4
DBP						
<85 mmHg	18	7.2	7	6.3	11	7.9
>85 mmHg	232	92.8	104	93.6	128	92
Pulse units (BPM)						
60-69	5	2	2	1.8	3	2
70-79	138	15.2	67	60.3	71	51
80-89	100	40	42	36	58	41.7
90-100	7	2.8	0	0	7	5
Fasting glucose						
<5.55 mmol/L	153	61.2	70	63	83	59.7
>5.55 mmol/L	97	38.8	41	36.9	56	40.2
Total serum cholesterol						
<5.2 mmol/L	150	60	41	36.9	109	78.4
>5.2 mmol/L	100	40	70	63	30	21.5
Triglycerides						
<1.69 mmol/L	28	11.2	9	8.1	19	13.6
>1.69 mmol/L	222	88.8	102	91.8	120	86.3

SBP = systolic blood pressure; DBP = diastolic blood pressure; BPM = Beats per minute

In order to determine the burden of risk factors on the prevalence of NCDs, the European SCORE model was used to estimate the risk of fatal CVD (section 3.11.2, Conroy *et al.,* 2003; Graham *et al.*, 2007), although concern has been expressed regarding the application of this and other existing score models for the Indian population (Enas *et al.*, 2008; Kanjilal *et al.*, 2008). The model had the following categories: age, gender, smoking, total cholesterol and systolic blood pressure. Based on these risk factors, two score charts are available, one for calculation of the 10-year risk of fatal CVD in populations at low risk and one for populations at high risk. The latter was used in the present study. Table 5.6 presents the results, where most respondents had a <1% risk score for fatal CVD.

Table 5.6: Frequency of respondents in score categories according to the European risk SCORE for fatal CVD*

Risk	SCORE	Total group N=250		Men N= 111		Women N= 139	
		Frequency N	%	Frequency N	%	Frequency N	%
Smoking	Yes	31	12.4	25	22.5	6	4.3
European	<1	178	71.2	58	52.3	120	86.3
SCORE (%)	1	51	20.4	34	30.6	17	12.2
	2	18	7.2	17	15.3	1	0.7
	4	3	1.2	2	1.8	1	0.7

European SCORE for risk of fatal CVD <1%, 1%, 2%, 4% (Conroy *et al.,* 2003; Graham *et al.*, 2007)

With reference to the European risk score presented in Table 5.6, Enas *et al.* (2008) indicated that in order to determine the CAD risk more appropriately among Asian Indians, one must multiply the 10-year risk factor by a factor of 2 when using the European risk algorithm. This recommendation is based on studies where Asian Indians have shown ≥2-fold risk pattern of dying from CVD from any combination of risk factors as compared with Caucasians (Forouhi *et al.,* 2006). Table 5.7 shows the modified risk score being doubled, where, although the frequencies remain the same as in Table 5.6, Asian Indians have twice the risk compared with Caucasians.

Table 5.7: Frequency of respondents where the European score algorithm is multiplied by a factor of 2

Risk	Score	Total group N=250		Men N= 111		Women N= 139	
		Frequency N	%	Frequency N	%	Frequency N	%
Smoking	Yes	31	12.4	25	22.5	6	4.3
European	1	178	71.2	58	52.3	120	86.3
SCORE (%)	2	51	20.4	34	30.6	17	12.2
	4	18	7.2	17	15.3	1	0.7
	8	3	1.2	2	1.8	1	0.7

5.2.3 Behavioural risk factors

5.2.3.1 Energy and nutrient intakes

Table 5.8 shows the mean and SD for the percentage by which energy intakes were misreported (under-reported and over-reported in comparison with the EER), where a large percentage of men (94%) and women (73%) reported energy intakes lower than their EERs in the QFFQ, with men having a higher mean percentage of under-reporting than women. In terms of over-reporting in the QFFQ, more women (27%) over-reported than men (6%). In the 24-hour recall, significantly fewer participants under-reported their energy intakes in comparison with their EERs (p=0.0000). The mean percentage by which energy intake was under-reported was, however, similar for the total sample and the men (p>0.05) and significantly higher than the QFFQ for the women (p=0.004). Significantly more respondents of both genders reported energy intakes higher than the EERs in the 24-hour recalls than in the QFFQ (P=0.0000) and the mean percentage by which energy intakes were over-reported was also significantly higher than the QFFQ for the whole sample (p=0.001), but not for the men and women separately (p>0.05).

Table 5.8: Mean and SD percentage by which energy intakes were over- and under-reported in the QFFQ and 24-hour recall in comparison with the Estimated Energy Requirement

		Total *N	Mean % (SD)	Men *N	Mean % (SD)	Women *N	Mean % (SD)
QFFQ	Over-reporting	44	11.5 (9.2)	7	11.7 (7.8)	37	11.4 (9.5)
	Under-reporting	206	22.0 (12.5)	104	27.9 (10.8)	102	15.9 (11.3)
24-hour recall	Over-reporting	111	26.8 (24.1)	36	22.4 (19.1)	75	28.9 (26.0)
	Under-reporting	139	23.2 (14.3)	75	22.9 (13.3)	64	23.5 (15.6)

* Number of respondents whose reported energy intake was higher or lower than the Estimated Energy requirement
SD =standard deviation; QFFQ = quantitative food frequency questionnaire

Table 5.9 presents the percentage of respondents that were classified as under-reporters. Cut-off points to identify possible over- and under-reporters were calculated using the formula by Rennie *et al.* (2007), as discussed under section 4.2.3. Possible under-reporters and over-reporters were identified as respondents whose reported

energy intake, expressed as a percentage of the EER, fell below (100-2SD) or above (100+2SD) the EER (Rennie *et al.,* 2007). A larger percentage of men than of women were classified as under-reporters. The CV for the reported EI for the 24-hour recall was 32% for women and 28% for men, compared with the CV of 16.8% (women) and 19% (men) for the QFFQ, indicating more variation in the 24-hour recall than in the QFFQ. Furthermore, this is also seen by the very low cut-off points used for under-reporting for the 24-hour recall: 42% for men and 34% for women. Consequently, because of the large amount of variation and the usage of lower cut-off points in the 24-hour recall, the results of the QFFQ will be reported in the next part of this study.

Table 5.9: Percentage of men and women categorised as over-reporters and under-reporters

		Men Cut-off % EER	N	%	Women Cut-off % EER	N	%
QFFQ	Over-reporters	128	0	0	126	2	1
	Under-reporters	72	47	42.3	74	19	13.7
24-hour recall	Over-reporters	158	2	1.8	166	5	3.6
	Under-reporters	42	6	5.4	34	0	0

QFFQ = Quantitative food frequency questionnaire

Table 5.10 presents the results of the mean daily nutrient and energy intakes derived from the QFFQ for the whole study group and for men and women, separately. The men reported slightly higher means than the women for all the macronutrients except for n-3 fatty acids, where the means were similar. For micronutrients, the men also reported slightly higher means except for vitamin K, where the women reported a higher mean intake than the men. Both men and women had similar means for vitamin D and thiamin intakes.

Table 5.10: Mean daily nutrient and energy intakes from the QFFQ

Nutrient	Total			Men			Women		
	Mean (SD)	Median	25th,75th percentile	Mean (SD)	Median	25th,75th percentile	Mean (SD)	Median	25th,75th percentile
Energy (kJ)	7481 (1383.1)	7420	6501.4, 8176.1	7815 (1514.1)	7638.6	6710.6,8529.8	7214.7 (1209.5)	7638.6	6325.4,8048.1
Total protein (g)	56.9 (14.9)	54.9	46.7,65.4	60.3 (16.5)	55.2	47.4,66.3	54.2(11.5)	53.4	44.8,60.5
Total fat (g)	69.7 (15.9)	68.4	58.5,78.0	69.2 (12.1)	68.7	59.2,78	73.8 (16.6)	65.1	54.1,74.0
SAT FAT (g)	19.8 (5.0)	19.4	16.1,22.1	19.5 (4.4)	19.2	17.1,22.1	19.9 (4.9)	18.8	15.1,21.0
MUFAs (g)	19.0 (5.0)	18.5	15.2,21.5	19.0 (3.8)	18.9	15.8,21.7	17.7 (4.5)	17.8	14.0,20.7
PUFAs (g)	23.2 (5.6)	22.3	19.2,26.0	24.5 (5.93)	22.1	19.3,26	22.2 (4.7)	20.9	18.6,24.4
P:S ratio	1.2 (0.2)	1.1	1.0,1.3	1.17 (0.2)	1.1	1.04,1.29	1.2 (0.3)	1.2	1.0,1.3
n-6 (g)	22.0 (5.3)	21.1	18.3-24.6	23.2 (4.7)	22.4	22.1,24.3	20.4 (4.7)	19.8	17.5,22.9
n-3 (g)	0.4 (0.1)	0.4	0.3,0.5	0.5 (0.1)	0.4	0.4,0.5	0.4 (0.1)	0.46	0.3,0.5
n-6:n-3 ratio	46.7 (13.1)	44.0	39.2,50.3	47.4 (14.4)	44.0	39.2,51.2	45.2 (12.0)	44.0	39.3,49.3
Cholesterol (mg)	199.7 (85.7)	193.8	150.8,239.1	204.7 (70.0)	202.8	162.5,252.4	184.3 (78.8)	184.2	137.3,215.8
Carbohydrate (g)	212.7 (38.0)	216.3	189.5,234.4	219.6 (207.2)	210.8	184.1,222.7	207.2 (33.9)	203.8	175.7, 225.4
Dietary fibre (g)	18.4 (4.2)	18.1	15.8, 21.1	18.8 (4.09)	17.7	15.4,20.5	18.1 (3.77)	17.2	15.0,20.4

Nutrient	Total			Men			Women		
	Mean (SD)	Median	25th,75th percentile	Mean (SD)	Median	25th,75th percentile	Mean (SD)	Median	25th,75th percentile
Calcium (mg)	461 (121.0)	445.7	394.7, 516.3	460.2 (119.1)	446.5	437.8,482.7	462.5 (123.0)	443.6	441.9,483.1
Iron (mg)	10.3 (2.4)	10.2	8.6,11.5	10.5 (2.7)	10.3	10.0,11.0	10.1 (2.3)	10.2	9.8,10.5
Sodium(mg)	1889 (472.6)	1881	1605,2135	2007 (552.1)	1980.7	1903.5,2111.2	1794 (374.0)	1819	1732,1857
Chloride (mg)	954 (449.2)	900	620,1244	1006.6 (439.1)	943.8	915.4,1097.8	912.3 (405.7)	890	842, 982
Zinc (mg)	7.3 (1.9)	7.07	6.1,8.3	7.8 (2.1)	7.4	7.4,8.2	6.9 (1.5)	6.9	6.7,7.2
Copper (mg)	1.2 (0.3)	1.1	0.9,1.3	1.2 (0.3)	1.1	1.1,1.3	1.1 (0.3)	1.1	1.1,1.2
Iodine (ug)	23.5 (8.3)	22.8	17.9,27.0	25.3 (9.1)	23.6	23.6,27.0	22.2 (7.4)	21.9	20.9,23.4
Manganese (ug)	2456.3 (607.3)	2426.2	2019.5, 2841.4	2530.6 (674.0)	2454.8	2403.8,2657.4	2397.0 (544.2)	2386.4	2305.8,2488.3
Magnesium (mg)	226.4 (48.07)	223.9	1946,254.1	232.9 (53.7)	226.5	222.8,243.0	221.2 (42.4)	217.2	214.1,228.3
Vitamin A (RE) (ug)	580.3 (250.2)	531.3	405.5, 686.0	606.3 (243.4)	610.6	560.5,652.1	559.5 (254.5)	510.5	516.8,602.2
Thiamin (mg)	0.8 (0.2)	0.8	0.7,0.9	0.8 (0.2)	0.8	0.8,0.9	0.8 (0.1)	0.8	0.8,0.8
Riboflavin (mg)	1.0 (0.3)	1.0	0.9,1.2	1.1 (0.3)	1.1	1.0,1.1	1.0 (0.3)	1.0	1.0,1.1
Niacin (mg)	14.9 (4.0)	14.5	12.6,17.3	15.7 (4.4)	15.1	14.9,16.6	14.2 (3.5)	14.1	13.7,14.8
Vitamin B6 (mg)	1.2 (0.3)	1.1	0.9,1.4	1.2 (0.3)	1.2	1.1,1.3	1.1 (0.3)	1.1	1.1,1.2
Folate (ug)	224 (54.8)	218	189,251	225.5 (62.4)	217.3	213.7,237.2	222.9 (48.2)	219.2	214.8,231.0

Nutrient	Total			Men			Women		
	Mean (SD)	Median	25th,75th percentile	Mean (SD)	Median	25th,75th percentile	Mean (SD)	Median	25th,75th percentile
Vitamin B12 (ug)	5.2 (2.9)	4.8	3.5,6.6	5.5 (2.9)	5.2	4.9,6.0	5.0 (2.9)	4.4	4.5,5.5
Pantothenate (mg)	4.1 (1.1)	1.0	3.4,4.6	4.3 (1.2)	4.0	4.1,4.5	4.0 (1.0)	4.0	3.8,4.1
Biotin (ug)	25.4 (7.2)	24.5	20.2,29.1	26.2 (7.6)	25.0	24.7,27.6	24.8 (6.9)	24.2	23.6,26.0
Vitamin C (mg)	89.1 (45.7)	82.0	56.8,112.1	90.7 (44.0)	84.6	82.4,98.9	87.9 (47.2)	80.5	80.0,95.8
Vitamin D (ug)	3.2 (3.0)	2.9	2.3,3.7	3.2 (1.2)	3.2	3.0,3.5	3.2 (3.8)	2.7	2.5,3.8
Vitamin E (mg)	18.3 (4.8)	17.9	15.0,20.9	19.4 (5.1)	18.7	18.4,20.4	17.5 (4.4)	17.5	16.7,18.2
Vitamin K (ug)	86.1 (34.2)	86.1	66.1,105.8	80.1 (29.2)	81.6	74.6,85.6	91.0 (37.2)	88.7	84.7,97.2

SD = standard deviation; SAT FAT = saturated fatty acids; MUFAs = monounsaturated fatty acids; PUFAs = polyunsaturated fatty acids

Table 5.11 presents the results of the percentage of energy derived from the macronutrients for the total group, where the largest amount of energy was derived from carbohydrates, followed by total fat.

Table 5.11: Percentage of energy from macronutrients for the total group from the QFFQ

Macronutrients	Mean % Energy (SD)	Median % energy	25^{th}, 75^{th} percentile
Total fat	35.4 (3.2)	35.1	33.2, 37.2
n-6 fatty acids	11.1 (1.8)	10.9	10.9, 11.9
n-3 fatty acids	0.2 (0.0)	0.2	0.21, 0.27
PUFAs	11.7 (1.8)	11.5	10.6, 12.6
MUFAs	9.6 (1.4)	9.6	8.8, 10.4
SAT FAT	10.0 (1.3)	10.0	9.2, 10.66
Carbohydrate	48.3 (4.1)	48.6	46.6, 50.8
Protein	12.9 (1.8)	12.8	12.0, 14.1

PUFAs=polyunsaturated fatty acids; MUFAs=monounsaturated fatty acids; SAT FAT= saturated fatty acids

Table 5.12 presents the results of the frequency distribution of energy according to the WHO population nutrient intake goals for the prevention of death and disability (WHO, 2003b). Total fat, PUFAs, n-6 fatty acids and free sugars recorded percentages higher than the goal recommended by the WHO. In contrast, n-3 fatty acids, dietary fibre and vegetable intakes were much lower than the recommended WHO goal. The low sodium intake could be due to the fact that it was very difficult to determine the exact sodium intake using the QFFQ.

Table 5.12: Comparison of mean intakes of men and women with the WHO population nutrient intake goals for prevention of death and disability from NCDs (WHO, 2003b)

		Mean reported intakes	
Dietary factor (food or nutrient)	**Goal**	**Men N=111**	**Women N=139**
Total fat %E	15-30	35.1	37.1
SAT FAT %E	<10	10.0	9.4
PUFAs %E	6-10	12.4	11.2
n-6 PUFAs %E	5-8	11.7	10.3
n-3 PUFAs %E	1-2	0.25	0.23
Trans fatty acids %E	<1	0.96	0.86
MUFAs %E	By difference	9.6	8.9
Total carbohydrate %E	55-75	49.9	47.0
Free sugars %E	<10	12.5	12.5
Protein %E	10-15	12.8	12.0
Cholesterol mg/day	<300	204.7	184.3
Mean sodium mg/day	<2000	1899	1739
Fruit and vegetables g/day	≥400	221.9	236.9
Dietary fibre g/day	>25	18.8	18.1

SAT FAT= saturated fatty acids; PUFAs=polyunsaturated fatty acids; MUFAs=monounsaturated fatty acids

To determine the adequacy of the dietary intakes of the respondents, the mean intakes as measured by the QFFQ were compared with the Estimated Average Requirement (EAR) (Food and Nutrition Board, 2000) of nutrients for men and women. Table 5.13 presents the frequency of respondents who consumed less than the EAR. Respondents who consumed less than the EAR varied with regard to both macronutrients and micronutrients. In terms of macronutrients, there was a high percentage of respondents who consumed less than the estimated EAR for fibre and protein, which was an unexpected finding in the case of protein, as only 6.4% of the respondents were vegetarians. Moreover, no respondents met the EAR for n-3 fatty acids, but practically all met the requirements for n-6 fatty acids. With regard to

micronutrients, the highest percentages of respondents who consumed less than the EAR were those for calcium (99.6%), folate (95.2%), vitamin D (94%) and pantothenic acid (82.8%).

Table 5.13: Respondents who consumed less than the Estimated Average Requirements (EAR)

Nutrient	Total N=250	%	Men N=111	%	Women N=139	%
Protein	91	36.4	54	48.6	37	26.6
n-6 PUFAS	15	6	13	11.7	2	1.4
n-3 PUFAs	250	100	111	100	139	100
Fibre	236	94.4	103	92.7	133	95.6
Calcium	249	99.6	111	100	138	99.2
Iron	27	10.8	3	2.7	24	17.2
Zinc	158	63.2	93	83.7	65	46.7
Iodine	250	100	111	100	139	100
Vitamin A	123	49.2	62	55.8	61	43.8
Thiamin	167	66.8	84	75.6	83	59.7
Riboflavin	89	35.6	55	49.5	34	24.4
Niacin	44	17.6	22	19.8	22	15.8
Vitamin B6	99	39.6	38	34.2	61	43.8
Folate	238	95.2	102	91.8	136	97.8
Vitamin B12	24	9.6	10	9.0	14	10.07
Panthothenic acid	207	82.8	88	79.2	119	85.6
Biotin	199	79.6	84	75.6	115	82.7
Vitamin C	81	32.4	47	42.3	34	24.4
Vitamin D	235	94	105	94.5	130	93.5
Vitamin E	18	7.2	7	6.3	11	7.9
Vitamin K	174	69.6	103	92.7	71	51.0

EAR = Estimated Average Requirements (Food and Nutrition Board, Institute of Medicine. Dietary Reference Intakes. Washington, DC. National Academy Press, 2000); PUFAs = polyunsaturated fatty acids

Figure 5.3 presents, in the form of a bar graph, the results of the percentage of respondents with intakes <EAR for vitamins, with vitamin D, folate and panthothenic acid having the highest percentage of respondents with intakes <EAR (Food and Nutrition Board, 2000).

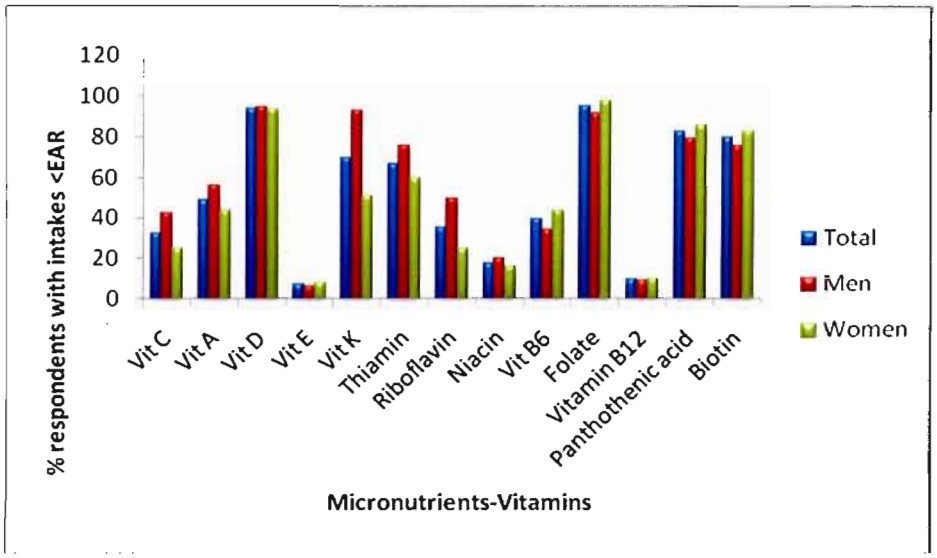

Figure 5.3: Percentage of respondents with intakes <EAR for vitamins (Food & Nutrition Board, 2000)

Figure 5.4 presents the results of the percentage of respondents with intakes <EAR for minerals in the form of a bar graph, with calcium and iodine having the highest percentage of subjects with intakes <EAR for minerals (Food and Nutrition Board, 2000).

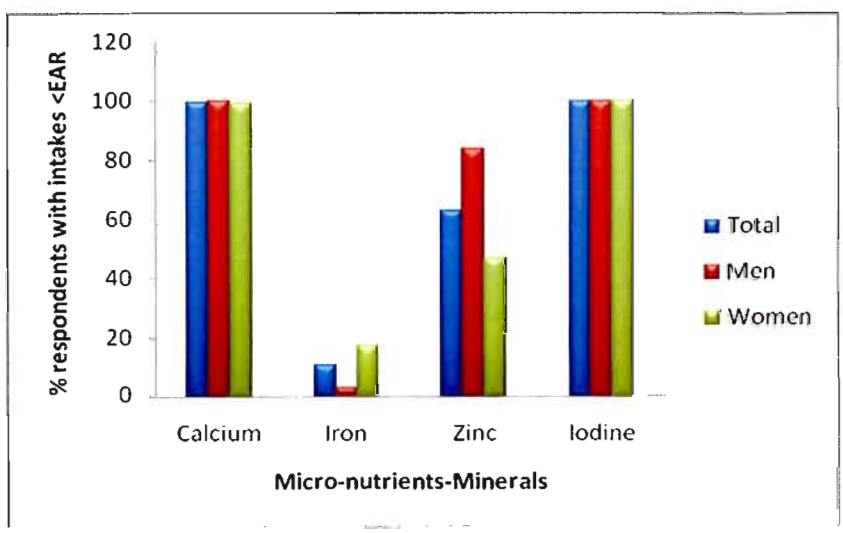

Figure 5.4: Percentage of respondents with intakes <EAR for minerals (Food & Nutrition Board, 2000)

Overall diet quality combines many compounds of the diet into a single indicator. Two dietary quality indices, a deficiency index (Index 1) and an excess index (Index 2), were calculated in this study, following the procedure of Thiele *et al.* (2004). The methodology of deriving the dietary quality indices has been explained in Chapter 3, Section 3.11.1. The deficiency index was derived from 13 vitamins, 12 minerals and trace elements, protein, carbohydrate, n-6 and n-3 fatty acids and dietary fibre (30 nutrients), with a maximum score of 3000. The excess index comprised fat, cholesterol, the P:S ratio, sugar, alcohol and sodium, with a maximum score of 600. For these indices, the higher the index, the better the diet quality. Table 5.14 presents the results of the dietary quality indices. Index 1 had a mean of 2259.8 and Index 2 had a mean of 518, both of which reflect good diet quality.

Table 5.14: Mean dietary quality indices

Indices	Total group N=250			Men N=111			Women N=139		
	Mean (SD)	Median	25th, 75th Percentile	Mean (SD)	Median	25th, 75th Percentile	Mean (SD)	Median	25th, 75th Percentile
*Index 1	2259.8 (198.4)	2260	2143, 2410	2255 (204.7)	2247	212.9 241.8	2265 (187.2)	2274	2156.2 410
#Index 2	518 (35.4)	522	502,5	515.1 (34.1)	516.9	502.9, 534.3	522.1 (35.5)	526	501.9, 548.5

* Deficiency index, max score 3000;
# Excess index, max score 600;
SD = standard deviation

Table 5.15 presents the results of the frequency distribution of the dietary quality indices, showing that, for Index 1, the largest percentage of respondents came from the 2000 to 2500 category, and for Index 2, the largest percentage of respondents came from the 500 to 559 category, reflecting good diet quality.

Table 5.15: Frequency of dietary quality indices

Indices	Total group		Men		Women	
	N=250	%	N=111	%	N=139	
***Index 1**						
1500-2000	26	10.4	15	13.5	10	7.1
2000-2500	201	80.4	88	79.2	118	84.8
2500-3000	23	9.2	8	7.2	11	7.9
#Index 2						
<400	1	0.4	1	0.9	0	0
400-499	50	20	22	19.8	34	24.4
500-559	198	79.2	88	79.2	104	74.8
600	1	0.4			1	0.7

* Deficiency index, max score 3000
\# Excess index, max score 600

5.2.3.2 Food group intakes

Table 5.16 presents the results of the percentage of energy and macronutrients supplied by each food group from the QFFQ, with cereals providing the highest percentage of energy, carbohydrate, fat and PUFAs. The meat group provided the highest percentage of protein, MUFAs and n-6 fatty acids, while the highest percentage of saturated fatty acids was shared by the cereal and meat groups. The cereal group included dishes such samoosas, roti, cakes, biscuits, macaroni cheese, and starch-based puddings, which could explain the high saturated fat intake. With reference to n-3 fatty acids, only milk, eggs, meat, fish and fats and oils contributed towards the percentage of energy and macronutrients.

Table 5.16: Percentage of energy and macronutrients supplied by each food group from the QFFQ

Food group	% energy Mean (SD) Median	% CHO Mean (SD) Median	% Fat Mean (SD) Median	% Protein Mean (SD) Median	% SAT FAT Mean (SD) Median	% PUFAs Mean (SD) Median	% MUFAs Mean (SD) Median	% n-6 Mean (SD) Median	% n-3 Mean (SD) Median
Cereals	38.7 (5.5) 38.6	47.0 (6.8) 46.2	30.7 (7.7) 30.3	28.1 (7.0) 27.3	29.5 (8.9) 29.1	35.2 (9.3) 35.0	25.8 (8.3) 24.7	22.3 (7.7) 21.1	0
Vegetables	6.7 (3.3) 6.2	5.1 (2.7) 4.8	9.1 (4.8) 8.4	4.2 (2.1) 3.8	4.9 (2.9) 4.4	15.9 (7.7) 14.5	6.9 (4.2) 6.1	3.6 (3.2) 2.9	0
Fruit	4.1 (1.7) 4.0	6.8 (2.8) 6.5	0.1 (0.1) 0.1	1.2 (0.6) 1.1	0.2 (0.1) 0.2	0.1 (0.1) 0.1	0.1 (0.0) 0.1	2.2 (2.3) 2.2	0
Legumes	5.5 (2.5) 5.3	4.5 (2.0) 4.3	3.3 (2.3) 3.1	10.9 (6.7) 9.8	2.6 (1.7) 2.4	4.6 (3.3) 4.2	2.9 (2.3) 2.6	22.9 (15.2) 22.6	0
Nuts & seeds	0.8 (1.4) 0.4	0.5 (1.4) 0.0	1.3 (1.9) 0.8	0.6 (1.6) 0.3	1.1 (1.5) 0.4	0.6 (1.7) 0.0	1.2 (2.7) 0.1	0.04 (0.3) 0.0	0
Milk	4.7 (1.7) 4.8	3.0 (1.6) 2.8	6.7 (2.5) 6.7	7.8 (3.3) 7.6	13.7 (4.9) 13.6	0.6 (0.2) 0.6	6.5 (2.5) 6.4	5.6 (2.9) 5.6	9.5 (7.6) 7.8
Eggs	1.2 (0.8) 1.1	0.1 (0.0) 0.1	2.4 (1.7) 2.2	2.5 (1.8) 2.2	2.0 (1.5) 1.85	2.1 (1.6) 2.0	2.9 (2.0) 2.6	0.1 (0.6) 0.0	21.5 (12.6) 20.9
Meat	14.3 (5.9)	3.3 (1.6) 3.4	24.0 (9.2) 25.3	31.8 (11.4) 34.4	29.5 (11.3) 31.3	12.8 (5.2) 13.6	34.0 (12.4) 36.3	36.7 (15.5) 38.8	39.5 (15.3) 41.8
Fish	3.2 (1.7) 3.3	0.7 (0.5) 0.8	4.4 (2.1) 4.4	10.2 (5.0) 10.3	3.1 (1.5) 3.2	6.3 (3.5) 6.1	3.8 (1.9) 3.9	2.4 (2.4) 2.3	11.2 (5.6) 11.0
Fats & oil	4.9 (1.9) 4.8	0.2 (0.1) 0.2	13.8 (5.0) 13.6	0.6 (0.7) 0.4	8.7 (3.7) 8.3	20.7 (7.7) 21.2	13.6 (5.5) 12.7	1.8 (1.7) 2.1	0.8 (1.9) 0.5
Sauces	2.4 (1.4) 2.3	21.2 (6.9) 22.6	2.0 (1.4) 1.9	0.2 (0.2) 0.2	3.4 (2.7) 3.2	0.2 (0.3) 0.2	1.3 (1.2) 1.1	1.3 (1.7) 0.0	0
Sugar + sweets	11.0 (3.7) 11.5	3.2 (2.0) 3.2	0	0	0.5 (0.7) 0.2	0.3 (0.6) 0.2	0.4 (0.5) 0.3	0.08 (0.4) 0.0	0
Beverage	0.8 (1.3) 0.0	1.3 (2.3) 0.0	0.2 (1.4) 0.0	0.6 (1.5) 0.0	0.2 (1.4) 0.0	0.01 (0.1) 0.0	0.11 (0.8) 0.0	0.0 (0.0) 0.0	0

CHO = carbohydrate;
SAT FAT = saturated fatty acids;
PUFAs = polyunsaturated fatty acids;
MUFAs =monounsaturated fatty acids;
SD = standard deviation

Table 5.17 presents the results of the percentage of micronutrients supplied by each food group from the QFFQ. The highest percentage of calcium came from the milk group. The highest percentage of vitamin A, vitamin B12, zinc and niacin came from the meat group. The cereal group also featured prominently amongst the micronutrients with a high percentage of iron, vitamin B6, riboflavin, thiamin and folate. Cereal-rich foods, e.g. bread and maize meal, are fortified in South Africa; however, the Food Finder 3® program did not include the fortification values. Therefore, it is most likely that the values reported are slightly lower than the actual percentage supplied by each food group.

Table 5.17: Selected micronutrients supplied by each food group from the QFFQ

Food Group	% Calcium Mean (SD) Median	% Vit A Mean (SD) Median	% Iron Mean (SD) Median	% Zinc Mean (SD) Median	% Vit C Mean (SD) Median	% Vit B_{12} Mean (SD) Median	% Vit B_6 Mean (SD) Median	% Riboflavin Mean (SD) Median	% Thiamin Mean (SD) Median	% Niacin Mean (SD) Median	% Folate Mean (SD) Median
Cereals	23.4 (6.8) 22.1	11.6 (11.5) 7.2	36.4 (11.4) 34.9	30.5 (7.7) 30.5	4.4 (5.2) 2.6	4.8 (8.8) 0.0	36.3 (13.9) 34.5	28.8 (15.0) 26.7	46.7 (12.1) 46.4	32.6 (13.7) 31.1	38.3 (11.5) 37.4
Vegetables	8.8 (3.9) 8.2	25.4 (16.6) 20.9	7.7 (4.4) 6.3	5.3 (3.5) 5.3	28.8 (15.9) 27.9	0.1 (0.6) 0.0	14.6 (7.3) 14.1	4.8 (3.6) 4.0	10.1 (5.6) 9.1	8.0 (6.6) 6.2	10.7 (4.3) 10.3
Fruit	1.9 (1.7) 1.3	3.7 (2.5) 3.3	3.3 (2.0) 3.1	1.8 (1.3) 1.8	28.5 (17.4) 25.0	0.01 (0.1) 0.0	7.7 (3.7) 7.1	3.0 (1.9) 2.8	5.6 (6.0) 4.9	2.8 (1.7) 2.6	8.0 (4.6) 7.5
Legumes	6.2 (3.3) 5.9	0.9 (0.6) 0.9	15.4 (7.6) 14.7	8.6 (5.2) 8.6	0.1 (0.4) 0.0	0.0 (0.0) 0.0	8.3 (4.0) 8.1	6.2 (4.8) 5.4	11.6 (5.4) 11.2	3.2 (1.9) 2.9	21.5 (7.8) 20.8
Nuts & seeds	2.3 (5.4) 0.2	0.1 (0.5) 0.0	1.4 (3.0) 0.0	0.0 (1.9) 0.0	0.0 (0.0) 0.0	0.0 (0.0) 0.0	0.3 (0.4) 0.0	1.2 (3.4) 0.0	0.8 (1.4) 0.0	0.8 (0.2) 0.0	1.0 (6.3) 0.0
Milk	32.7 (9.6) 33.3	11.1 (5.4) 10.7	0.7 (0.8) 0.8	7.4 (3.5) 7.4	0.8 (1.3) 0.0	12.3 (16.9) 7.7	3.1 (1.6) 3.1	16.2 (6.2) 16.0	2.2 (1.5) 2.2	0.4 (0.6) 0.0	2.7 (1.3) 2.6
Eggs	1.4 (1.1) 1.3	1.7 (1.7) 1.3	2.3 (1.7) 2.1	1.5 (1.3) 1.5	0.1 (0.6) 0.0	3.9 (3.1) 3.4	0.2 (0.5) 0.0	4.5 (3.4) 3.9	1.2 (1.3) 1.2	0.0 (0.0) 0.0	1.8 (1.4) 1.6
Meat	7.3 (3.7) 7.3	27.8 (21.7) 25.8	20.5 (9.7) 20.9	35.6 (12.3) 35.6	7.8 (6.1) 6.4	40.8 (19.9) 39.6	18.0 (9.2) 17.7	23.2 (13.7) 23.7	13.5 (8.0) 12.8	36.5 (13.9) 38.0	12.5 (8.1) 12.6
Fish	8.2 (5.0) 8.5	1.6 (1.1) 1.4	4.8 (2.9) 4.7	4.7 (3.2) 4.7	0.7 (1.3) 0.0	35.8 (35.6) 16.6	4.7 (2.8) 4.8	5.5 (3.1) 5.6	3.8 (2.4) 3.7	12.1 (6.0) 12.3	1.0 (0.8) 0.9
Fats & oil	0.2 (0.2) 0.2	11.9 (6.8) 11.0	0.2 (0.4) 0.0	0.5 (0.6) 0.5	0	0.0 (0.0) 0.0	0.4 (0.6) 0.0	0.0 (0.2) 0.0	0.1 (0.4) 0.0	1.4 (1.7) 0.7	0.6 (0.6) 0.5

SD = standard deviation

The importance of deriving food patterns for the purposes of dietary intervention has been discussed in Chapter 3, section 3.11.1. Table 5.18 presents the factor loadings for the first two principal components identified from the percentage of fat contributed by each food group, where in Factor 1, the higher loadings were for the legumes, cereals and vegetable groups (named the legumes, cereals and vegetables factor) and in Factor 2, the higher loadings were for the sweets and sugars, fats and oil, and the full-fat and low-fat milk groups (named the sugars, fats and oils and milk factor).

Table 5.18: Factor loadings for the first two principal components identified from the fat content in each food group.

	Factor loadings	
	Factor 1: legumes, cereals and vegetables	**Factor 2:** sugars, fats and oils and milk
Cereals, cereal products	0.61	-0.04
Vegetables	0.55	-0.29
Fruit	0.08	0.30
Legumes	0.75	-0.01
Nuts and seed	0.06	-0.70
Full-fat milk & milk products	0.24	0.37
Low-fat milk & milk products	0.13	0.34
Eggs	-0.54	0.03
Meat and meat products	-0.88	0.06
Fish and seafood	-0.64	-0.40
Fats and oils	-0.01	0.51
Sugar & sweets	0.17	0.55

5.2.3.3 *Physical activity*

Physical activity was measured by the GPAQ1, where information was collected on three activity level domains: occupation, travel and recreation activities. The method used was discussed in section 3.9. For the purposes of this study, the following definitions, as taken from the International Physical Activity Questionnaire (IPAQ) Committee Scoring Protocol (2005) (http://www.ipaq.ki.se/scoring.htm), are used for the overall reporting of the physical activity categories. They are divided into the following categories: inactive (low) 0-<600 METS-min, minimally active (moderate) 600-<3000 METS-min and sufficiently active (high) >3000 METS-min. Table 5.19 shows that, although most respondents were classified as inactive, they were predominantly women.

Table 5.19: Physical activity level

	GPAQ						Pedometer-step counter					
Categories	Total N=250	%	Men N=111	%	Women N=139	%	Total N=250	%	Men N=111	%	Women N=139	%
Low	163	65.2	68	61.3	105	75.5	173	69.2	68	61	105	75.5
Moderate	73	29.2	20	18	23	16.5	43	17.2	20	18	23	16.5
High	41	16.4	23	20.7	11	7.9	34	13.6	23	21	11	7.9

Low= 0-<600 METS-mins
Moderate = 600-<3000 METS-mins
High = >3000 METS-mins

5.3 *ASSOCIATIONS AMONGST VARIABLES*

Nutrient intakes and risk markers were not normally distributed (Shapiro-Wilk, $p< 0.05$), therefore all data were BoxCox transformed in order to allow for valid parametric

correlations and associations. All the following results are, therefore, on BoxCox transformed data and not interpreted in absolute terms.

5.3.1 Associations between dietary intakes and clinical risk markers

Table 5.20 presents the results for the Pearson partial correlations (BoxCox transformed, controlled for age and smoking) for dietary intake and associated risks. "SCORE" represents the European ten-year risk algorithm for CAD (Graham *et al.*, 2007). "Risk score" is the aggregate total of all risk markers defined by Asian standards, as presented in Chapter 2, section 2.6, and "Risk score 1" is the aggregate of all risk markers, with fasting glucose being doubled as compared to the other risk markers because many and strong correlations with nutrients were found and because Forouhi *et al.* (2007) suggested that South Asians are markedly sensitive to the impact of diabetes on CHD risk. There was a statistically significant correlation between Risk score and Risk score 1 for both men and women, and Index 1, whereas for Index 2, only men showed a negative significant correlation. There was a statistically significant but weak correlation between SCORE (European risk for fatal CAD), Risk score and Risk score 1, and the percentage of energy from n-6 fatty acids, total fat, MUFAs, protein and carbohydrate for men. In addition to the statistically significant correlation for Risk score and Risk score1 with Index 1 for women, there was also a statistically significant but weak correlation for Risk score and Risk score 1 with the percentage of energy from protein.

Table 5.20: Pearson partial correlations* between clinical risk scores and nutrients (BoxCox transformed, controlled for age and smoking)

	Total N=250			Men N= 111			Women N=139		
Variables	SCORE	Risk score	Risk score 1	SCORE	Risk score	Risk score 1	SCORE	Risk score	Risk score 1
P S	0.04	0.03	0.0	0.11	0.06	0.0	0.01	-0.01	-0.0
Dietary fibre	-0.06	0.14	0.1	-0.13	0.11	0.1	-0.09	0.17	0.1
**INDEX 1	-0.02	0.21	0.2	-0.07	0.23*	0.2*	-0.03	0.23*	0.2*
#INDEX 2	-0.09	-0.10	-0.1	-0.03	-0.30*	-0.2*	-0.12	0.04	0.0
n6 n3	0.12	-0.01	-0.0	0.14	0.02	0.0	0.14	-0.05	-0.0
n6 %E	0.13	0.05	0.0	0.20*	0.21*	0.2*	0.05	-0.06	-0.0
n3 %E	-0.04	0.06	0.0	-0.01	0.15	0.1	-0.15*	0.00	-0.0
Fat %E	0.23*	0.14	0.1	0.26*	0.37*	0.3*	0.13	0.00	0.0
PUFAs %E	0.13	0.08	0.0	0.21*	0.24*	0.2*	0.04	-0.04	-0.0
MUFAs %E	0.21	0.12	0.1	0.24*	0.25*	0.2*	0.10	0.05	0.0
SAT FAT %E	0.09	0.05	0.0	0.08	0.20*	0.2*	0.03	-0.03	0.0
Protein %E	0.09	0.20	0.2	0.20*	0.22*	0.2*	-0.08	0.21*	0.2*
Carbohydrate %E	-0.19*	-0.19	-0.2	-0.26*	-0.34*	-0.3*	-0.05	-0.11	-0.1

*Significant at p<0.05; **Deficiency index, # Excess index
PUFAs = polyunsaturated fatty acids; MUFAs = monounsaturated fatty acids; SAT FAT = saturated fatty acids; SCORE = represents the European ten-year risk algorithm for CAD; Risk score = is the aggregate total of all risk markers defined by Asian standards; Risk score 1 = is the aggregate of all risk markers, but fasting glucose was counted twice

Table 5.21 presents Pearson partial correlation analysis (BoxCox transformed) between clinical risk markers and the modified doubled European SCORE for the whole group, and men and women. There were a number of significant correlations between clinical variables for the total sample. BMI and triglycerides correlated with all the other variables except DBP in men. Interestingly, DBP had the fewest significant correlations with other clinical risk factors, although 93% of the respondents had raised DBP (Table 5.5).

Table 5.21: Pearson partial correlations* for clinical risk markers and modified doubled European SCORE (BoxCox transformed, controlled for age and smoking)

Variable		WC	SBP	DBP	Glucose	Chol	Trig	BMI	SCORE 2X
WC	Total	1.00	0.30*	-0.00	0.24*	0.29*	0.27*	0.54*	0.07
	Men	1.00	0.19*	-0.15	0.29*	0.33*	0.24*	0.60*	0.11
	Women	1.00	0.44*	0.13	0.44*	0.36*	0.47*	0.69*	-0.08
SBP	Total	0.3*	1.00	0.32*	0.16*	0.41*	0.32*	0.31*	0.07
	Men	0.19*	1.00	0.28*	-0.05	0.43*	0.29*	0.27*	0.17
	Women	0.44*	1.00	0.35*	0.29*	0.40*	0.34*	0.35*	-0.02
DBP	Total	-0.006	0.32*	1.00	0.02	0.03	0.14*	0.13*	-0.08
	Men	-0.15	0.28*	1.00	-0.08	-0.06	-0.02	0.00	-1.44
	Women	0.12	0.34*	1.00	0.09	0.10	0.23*	0.21*	-0.01
Glucose	Total	0.24*	0.16*	0.02	1.00	0.31*	0.47*	0.35*	0.00
	Men	0.29*	-0.05	-0.08	1.00	0.26*	0.44*	0.44*	0.10
	Women	0.49*	0.29*	0.09	1.00	0.36*	0.49*	0.30*	-0.02
Chol	Total	0.29*	0.41*	0.03	0.31*	1.00	0.49*	0.32*	0.11
	Men	0.33*	0.43*	-0.00	0.26*	1.00	0.55*	0.30*	0.13
	Women	0.36*	0.40*	0.102	0.363*	1.00	0.46*	0.34*	0.12
Trig	Total	0.27*	0.32*	0.14*	0.47*	0.49*	1.00	0.39*	0.10
	Men	0.24*	0.29*	-0.02	0.44*	0.55*	1.00	0.40*	0.25*
	Women	0.47*	0.34*	0.24*	0.49*	0.46*	1.00	0.38*	0.08
BMI	Total	0.54*	0.31*	0.13*	0.35*	0.32*	0.39*	1.00	-0.08
	Men	0.60*	0.27*	0.007	0.44*	0.30*	0.40*	1.00	-0.00
	Women	0.68*	351*	0.21*	0.30*	0.34*	0.38*	1.00	-0.03
SCORE2 X	Total	0.07	0.07	-0.00	0.00	0.11	0.10	-0.08	1.00
	Men	0.11	0.17	-0.14	0.10	0.13	0.25*	-0.00	1.00
	Woman	1.19	0.71	-0.08	-0.02	0.12	0.08	-0.03	1.00

*Significant at p<0.05
WC = waist circumference; SBP = systolic blood pressure, DBP = diastolic blood pressure; Chol = cholesterol; Trig = triglyceride; BMI = body mass index; SCORE 2X: modified doubled European risk score

Tables 5.22 to 5.24 present the results of the associations of nutrients, diet quality indices and risk score with clinical risk markers. In Table 5.22, WC showed the most significant associations with most variables for the total sample. Risk score showed the most statistically significant associations with all clinical risk markers for the whole study group.

Table 5.22: **Pearson partial correlations* between reported energy, nutrient intakes, indices and risk scores, and clinical risk markers for the whole sample (N=250)**

	Total N=250						
Variable	**WC**	**SBP**	**DBP**	**Glucose**	**Cholesterol**	**Triglyceride**	**BMI**
Energy (kJ)	0.30*	-0.05	-0.05	0.18*	0.11	-0.02	0.09
Total protein (g)	0.30*	0.02	-0.07	0.29*	0.16*	0.05	0.16*
Total fat (g)	0.33*	-0.03	-0.09	0.21*	0.12	0.02	0.13*
SAT FAT (g)	0.29*	-0.08	-0.12	0.22*	0.07	0.02	0.16*
MUFAs (g)	0.32*	-0.02	-0.10	0.25	0.11	0.03	0.12
PUFAs (g)	0.28*	0.03	-0.02	0.17*	0.10	0.01	0.07
P:S	-0.03	0.13*	0.12*	-0.07	0.03	-0.02	-0.10
Cholesterol (mg)	0.29*	0.07	-0.06	0.30*	0.12	0.09	0.13*
Carbohydrate (g)	0.22*	0.06	0.01	0.09	0.06	-0.06	0.08
Dietary fibre (g)	0.18*	0.07	0.07	0.07	0.10	-0.06	0.08
n-6 (g)	0.27*	0.07	0.07	0.15*	0.10	-0.06	0.04
n-3 (g)	0.21*	-0.04	-0.05	0.18*	0.09	0.01	0.12
n-6:n-3	0.04	0.15*	0.11*	-0.02	-0.05	0.02	-0.04
INDEX 1	0.12	0.01	0.01	0.24*	0.11	-0.02	0.06
INDEX 2	-0.13*	0.01	0.01	-0.06	-0.01	0.04	-0.00
Score 1	0.27*	0.11	-0.09	0.08	0.16*	0.16*	-0.01
Risk score	0.45*	0.41*	0.15*	0.63*	0.64*	0.64*	0.64*
(n6+n3) %E	0.03	0.07	0.05	-0.05	-0.01	-0.01	-0.08
n6 %E	0.06	0.12	0.05	0.02	0.01	0.01	0.01
n3 %E	0.01	-0.01	-0.03	0.08	0.02	0.02	0.08
Fat %E	0.19*	0.03	-0.12	0.16*	0.06	0.06	0.12
PUFAs %E	0.08	0.11	0.02	0.05	0.03	0.03	0.00
MUFAs %E	0.20*	0.01	-0.13*	0.21*	0.05	0.05	0.10
SAT FAT %E	0.14*	-0.09	-0.18	0.18*	-0.02	-0.02	0.16*
Protein %E	0.15*	0.10	-0.07	0.24*	0.12	0.12	0.15*
Carbohydrate %E	-0.19*	-0.10	0.08	-0.23*	-0.11	-0.11	-0.17

*Significant at $p<0.05$
SAT FAT= saturated fatty acids;
MUFAs=monounsaturated fatty acids;
PUFAs=polyunsaturated fatty acids;
P:S=polyunsaturated fatty acids:saturated fatty acids;
WC=waist circumference;
SBP=systolic blood pressure,
DBP=diastolic blood pressure;
BMI=body mass index;
Risk score = is the aggregate total of all risk markers defined by Asian standards; Risk score 1 = is the aggregate of all risk markers, but fasting glucose was counted twice.
Index 1=Deficiency index, max score 3000;Index 2= excess index, max score 600

In Table 5.23, WC and glucose showed the most significant associations with most variables for men. Risk score showed the most statistically significant associations with all clinical risk markers for men, except DBP.

Table 5.23: Pearson partial correlations* between reported energy, nutrient intakes, indices and risk scores, and clinical risk markers for men (N=111)

Variable	Men N=111						
	WC	SBP	DBP	Glucose	Cholesterol	Triglyceride	BMI
Energy (kJ)	0.32*	-0.06	-0.10	0.28*	-0.01	0.04	0.18
Total protein (g)	0.23*	-0.02	-0.07	0.35*	0.09	0.18	0.21*
Total fat (g)	0.37*	-0.00	-0.11	0.37*	0.10	0.18	0.27*
SAT FAT (g)	0.28*	-0.07	-0.14	0.36*	0.05	0.17	0.28*
MUFAs (g)	0.29*	0.01	-0.11	0.37*	0.08	0.19*	0.25*
PUFAs (g)	0.39*	0.08	-0.08	0.32*	0.14	0.15	0.20*
P:S	0.14	0.18	0.07	-0.05	0.12	-0.02	-0.10
Cholesterol (mg)	0.20*	0.07	-0.01	0.33*	0.10	0.18	0.22*
Carbohydrate (g)	0.23*	-0.10	-0.07	0.14	-0.13	-0.10	0.10
Dietary fibre (g)	0.21*	-0.03	-0.05	0.06	-0.07	-0.14	0.04
n-6 (g)	0.38*	0.08	-0.08	0.30*	0.14	0.14	0.19
n-3 (g)	0.21*	-0.12	-0.04	0.32*	0.04	0.12	0.17
n-6:n-3	0.14	0.25*	0.00	-0.05	0.10	0.06	0.00
INDEX 1	0.21*	-0.06	-0.03	0.26*	-0.05	-0.02	0.10
INDEX 2	-0.28*	-0.06	-0.02	-0.22*	-0.12	-0.14	-0.17
Score 1	0.08	0.19*	-0.11	0.18	0.18	0.24*	-0.08
Risk score	0.60*	0.31*	0.01	0.59*	0.58*	0.58*	0.70*
(n6+n3) % E	0.12	0.22*	-0.05	-0.08	0.09	-0.00	-0.02
n6 %E	0.22*	0.19*	-0.02	0.14	0.23*	0.18	0.06
n3 %E	0.02	-0.13	0.04	0.22*	0.08	0.316*	0.07
Fat %E	0.24*	0.11	-0.07	0.33*	0.27*	0.36*	0.27*
PUFAs %E	0.24*	0.19*	-0.02	0.18	0.25*	0.20	0.09
MUFAs %E	0.11	0.08	-0.08	0.29*	0.15	0.23*	0.20*
SAT FAT %E	0.08	-0.05	-0.13	0.30*	0.12	0.27*	0.27*
Protein %E	0.04	0.06	-0.02	0.23*	0.16	0.16	0.16
Carbohydrate %E	-0.17	0.11	0.02	0.33*	026*	0.27*	0.23*

*Significant at $p<0.05$
SAT FAT= saturated fatty acids;
MUFAs=monounsaturated fatty acids;
PUFAs=polyunsaturated fatty acids;
P:S=polyunsaturated fatty acids:saturated fatty acids;
WC=waist circumference;
SBP=systolic blood pressure,
DBP=diastolic blood pressure;
BMI=body mass index

In Table 5.24, glucose showed the most significant associations with most variables for women. Risk score showed the most statistically significant associations with all clinical risk markers for women.

Table 5.24: **Pearson partial correlations* between reported energy, nutrient intakes, indices and risk scores, and clinical risk markers for women (N=139)**

	Women N=139						
Variable	**WC**	**SPB**	**DBP**	**Glucose**	**Cholesterol**	**Triglycerides**	**BMI**
Energy (kJ)	0.17	-0.06	0.01	0.15	0.20*	-0.04	0.04
Total Protein (g)	0.26*	0.05	-0.05	0.30*	0.2*	0.02	0.13
Total fat (g)	0.17*	-0.06	-0.06	0.15	0.11	-0.06	0.05
SAT FAT (g)	0.19*	-0.10	-0.10	0.17*	0.07	-0.05	0.09
MUFAS (g)	0.21*	-0.06	-0.08	0.21*	0.11	-0.05	0.06
PUFAs (g)	0.10	-0.01	0.05	0.10	0.04	-0.08	-0.00
P:S	-0.11	0.11	0.16	-0.09	-0.02	-0.02	-0.10
Cholesterol (mg)	0.24*	0.06	-0.08	0.34*	0.13	0.05	0.08
Carbohydrate (g)	0.11	-0.04	0.08	0.07	0.21*	-0.03	0.01
Dietary fibre (g)	0.13	0.13	0.18*	0.08	0.23*	-0.01	0.11
n-6 (g)	0.08	0.01	0.09	0.07	0.04	-0.07	-0.01
n-3 (g)	0.16	0.02	-0.05	0.08	0.13	-0.07	0.10
n-6:n-3	-0.07	0.09	0.14*	0.00	0.01	0.00	-0.08
INDEX 1	0.14	0.06	0.05	0.22*	0.24*	-0.01	0.04
INDEX 2	0.05	0.06	0.02	0.03	0.07	0.13	0.08
Score 1	0.08	0.02	-0.05	0.06	0.13	0.17*	0.07
Risk score	0.58*	0.49*	0.25*	0.66*	0.70*	0.61*	0.62*
(n6+n3) % E	-0.09	-0.02	0.12	-0.01	-0.09	-0.00	-0.11
n6 %E	-0.06	0.07	0.10	-0.06	-0.14	-0.05	-0.05
n3 %E	0.04	0.08	-0.08	-0.03	-0.02	-0.06	0.09
Fat %E	0.03	-0.03	-0.14	0.06	-0.11	-0.07	0.03
PUFAs %E	-0.04	0.05	0.05	-0.02	-0.14	-0.06	-0.05
MUFAs %E	0.16	-0.05	-0.16	0.19*	-0.04	-0.04	0.04
SAT FAT %E	0.14	-0.12	0.20*	0.13	-0.13	-0.04	0.11
Protein %E	0.21*	0.13	-0.11	0.27*	0.08	0.07	0.16
Carbohydrate %E	-0.16	-0.08	0.11	-0.17	0.03	0.01	-0.13

*Significant at $p<0.05$
SAT FAT= saturated fatty acids;
MUFAs=monounsaturated fatty acids;
PUFAs=polyunsaturated fatty acids;
P:S=polyunsaturated fatty acids:saturated fatty acids;
WC=waist circumference;
SBP=systolic blood pressure,
DBP=diastolic blood pressure;
BMI=body mass index;

As indicated in Table 5.18, factors were derived based on the percentage of fat from each food group, namely: Factor 1: legumes, cereals and vegetables and Factor 2: sugars, fats & oils and the milk group. Regression analyses were performed between factor scores of the two factors and the various risk markers. According to Table 5.25, only blood glucose was significantly correlated with Factor 1 for men and Factor 2 scores for the whole sample, and with Factor 2 for women, where factors are independent continuous predictors together with risk factors.

Table 5.25: Regression analysis ß coefficients (adjusted for age, smoking, sex and energy intake) between factor scores and risk markers

Risk marker	Factor 1: Legumes, cereals, and vegetables			Factor 2: Sugars, fats & oils and milk		
	Total	*Men*	*Woman*	*Total*	*Men*	*Women*
WC	-0.01	0.00	0.01	-0.03	-0.07	0.04
SBP	-0.00	-0.06	0.01	0.01	0.07	-0.01
DBP	0.01	-0.00	0.01	-0.10	-0.06	-0.14
Glucose	-0.16	-0.17*	-0.10	-0.17*	-0.08	-0.17*
Cholesterol	-0.09	-0.09	-0.09	-0.05	0.05	-0.11
Trig	-0.04	-0.14	0.02	-0.08	-0.09	-0.09
BMI	-0.02	-0.10	0.02	-0.09	-0.09	-0.06

WC = Waist circumference;
SBP=systolic blood pressure,
DBP=diastolic blood pressure;
BMI = Body mass index;
Trig = Triglyceride

Factor scores were divided into quintiles, and the median of the various risk markers calculated for the upper, the lower and the middle three quintiles. Using Jonckheere-Terpstra tests to determine the trend in the median of risk variables for the different quintile groups (upper, lower or middle three), Table 5.26 shows that Factor 1 (legumes, cereals and vegetables) when divided into quintiles, shows no difference between medians for DBP. Factor 2 (sugar, fats & oils and milk) shows no difference between the medians of Score 2X and DBP. There was consistent difference between the medians of SBP, BMI, cholesterol and triglyceride across quintiles for Factor 2. Respondents in the lowest quintile for Factor 1 had the highest median SBP whereas respondents in the lowest quintile for factor 2 had the highest median SBP, glucose,

BMI, cholesterol, triglycerides, and kilojoules. Medians for respondents in the highest quintile for Factor 1 and 2 were similar for Score 2X, SBP, DBP, glucose and triglyceride. Factor 1 was significantly associated with Score 2X, WC, glucose and cholesterol, whereas factor 2 was significantly associated with glucose and BMI.

Table 5.26: Median values of risk markers according to quintiles for Factor 1 and Factor 2 scores

	Factor 1: Cereals, vegetables and legumes				Factor 2: Fats & oils, sweets & sugars and milk group			
	Q1	*Q2-Q4*	*Q5*	*P for trend*	*Q1*	*Q2-Q4*	*Q5*	*P for trend*
Score 2X	1.0	2.0	1.0	<0.01*	1.0	1.0	1.0	0.704
SBP	122.0	121.0	120.0	0.535	125.0	122.0	120.0	0.296
DBP	90.0	90.0	90.0	0.954	90.0	90.0	90.0	0.273
WC	88.5	92.0	87.0	0.013*	86.2	90.0	88.0	0.959
Glucose	5.3	5.5	5.1	0.016*	5.5	5.3	5.1	0.000*
BMI	25.4	25.2	25.4	0.423	26.1	25.4	24.7	0.028*
Cholesterol	4.9	5.0	4.7	0.019*	5.1	4.9	4.8	0.093
Triglyceride	2.1	2.1	1.9	0.130	2.2	2.1	1.9	0.054
Physical activity (categories)	0.0	0.0	0.0	0.053	3.9	0.0	0.0	0.260
Pedometer step counts	294.4	287.2	264.8	0.069	320	288	277.6	0.735

SCORE 2X = modified SCORE for Indians;
BP = blood pressure;
WC = waist circumference;
BMI = body mass index;
*Significant at p<0.05

5.3.2 Associations between physical activity and clinical risk markers

Table 5.27 presents the results of the Pearson correlation analysis between physical activity and clinical parameters for the whole group and for men and women. Correlations were very weak and not statistically significant.

Table 5.27: Pearson correlations between physical activity and clinical parameters for BoxCox transformed data

	WC	SBP	DBP	Glucose	Cholesterol	Trig	BMI
Total METS Total group N=250	0.12	0.12	0.00	0.10	0.07	0.01	0.00
Total METS Men N=111	-0.02	0.13	0.03	0.13	-0.01	0.09	0.02
Total METS Women N=139	0.08	0.11	-0.01	0.10	0.12	-0.03	-0.01

Significant at p<0.05
WC=waist circumference;
SBP=systolic blood pressure,
DBP=diastolic blood pressure;
Trig=triglyceride;
BMI=body mass index;
Total METS= metabolic equivalents

Table 5.28 presents the results of the observed frequencies between activity categories and the prevalence of risk markers, where it can be seen that low physical activity has the highest percentage frequencies of the prevalence of the following risk markers: hypertension (SBP >130 mmHg), WC (>90 cm men, >80 cm women) and triglycerides (>1.69 mmol/L).

Table 5.28: Observed frequencies between physical activity categories (low, moderate and high) and prevalence of risk markers

	BP		WC		Glucose		BMI		Cholesterol		Triglyceride	
Physical activity category	**No risk N=**	**Risk N=**	**No risk N=**	**Risk N=**	**No Risk**	**Risk N=**	**No risk N=**	**Risk N=**	**No Risk**	**Risk N=**	**No risk N=**	**Risk trig N=**
Inactive												
N=	8	165	17	156	119	54	77	96	113	60	19	154
%	3.2	66	6.8	62.4	47.6	21.6	30.8	38.4	45.2	24	7.6	61.6
Minimally active												
N=	1	42	3	40	16	27	14	29	21	22	5	38
%	0.4	16.8	1.2	16	6.4	10.8	5.6	11.6	8.4	8.8	2	15.2
Sufficiently active												
N=	5	29	4	30	19	15	18	23	18	16	4	30
%	2	11.6	1.6	12	7.6	6	7.2	9.2	7.2	6.4	1.6	12

Risk SBP >130 mmHg, Risk WC >90 cm men, WC >80 cm women, Risk glucose >5.55 mmol/L, Risk BMI>25, Risk cholesterol (chol) >5.2 mmol/L, Risk triglyceride (trig) >1.69mmol/L;
Inactive= 0-<600 MET-mins ; Minimally active = 600-<3000 MET-mins ; Sufficiently active = >3000 MET-mins

Table 5.29 presents the results of the observed frequencies of activity categories and the European SCORE and the Risk score. The European SCORE represents a ten-year risk algorithm for fatal CAD, whereas Risk score is the aggregate total of all risk markers defined by Asian standards, as presented in Chapter 2, section 2.6. The observed frequencies show that low physical activity has a very minimal effect on the development of CAD as predicted by the European SCORE. However for the Risk score, low physical activity accounts for a higher frequency for risk score 3, 4, 5 and 6.

Table 5.29: Observed frequencies between physical activity categories (low, moderate and high) and the European SCORE and Risk score

Physical activity category	European SCORE				Risk score					
	<1	1	2	4	1	2	3	4	5	6
Low	133	29	10	1	4	23	41	43	32	30
Row %	76.8	16.7	5.7	0.5	2.3	13.2	23.7	24.8	18.5	17.3
Moderate	29	10	4	0	0	2	5	14	9	13
Row %	67.4	23.2	9.3	0.0	0.0	4.6	11.6	32.5	20.9	30.2
High	16	12	4	3	0	3	7	9	10	5
Row %	47.0	35.2	11.7	4.8	0.0	8.8	20.5	26.4	29.4	14.7

Low= 0-<600 METS-mins
Moderate= 600-<3000 METS-mins
High = >3000 METS-mins

5.4 SUMMARY

Table 5.30 provides, in sub-tables, a summary of the objectives of the study in relationship with significant correlations.

Table 5.30: Summary of objectives in relationship with significant correlations

Objective 1: To determine the prevalence of risk markers for NCDs (hypertension, increased fasting blood glucose, cholesterol and triglyceride levels, central obesity and physical inactivity).

Prevalence of risk markers			
Variables	**Total**	**Men**	**Women**
Hypertension			
Mean SBP mmHg	123.5	123	123.4
>130 mmHg	6.4%	6.3%	6.4%
Mean DBP mmHG	89.7	89.3	90.4
>85 mmHG	92.8%	93.6%	92%
Fasting blood glucose			
Mean mmol/L	5.5	5.41	5.5
>5.55 mmol/L	97 (38.8%)	41 (36.9%)	56 (40.2%)
Cholesterol			
Mean mmol/L	5.03	5.07	5.01
>5.2 mmol/L	40%	63%	21.5%
Triglycerides			
Mean mmol/L	2.3	2.2	2.6
>1.69 mmol/L	88.8%	1.8%	86.3%
Central obesity			
Mean WHR	0.88	0.91	0.87
Obesity Class I	44.8%	44.1%	43.1%
Obesity Class 11	15.6%	13.5%	17.2%
Mean WC cm	89.6	94.0 WC ≥90 cm: 87.4%	86.1 WC ≥80 cm:100%
Physical inactivity			
Inactive = 0-<600 METS-mins	65.2%	61.3%	75.5%

Objective 2: To establish whether an association exists between current trends in nutrient intakes and food patterns, with special reference to n-6:n-3 fatty acid ratio and the prevalence of risk markers for NCDs. As can be seen from the table, there were strong associations between clinical risk scores and nutrients in men and there were strong associations between WC (risk markers) and nutrients for the whole group.

Associations of risk markers			
Variable	**Total**	**Men**	**Women**
Clinical risk scores with nutrients (statistically significant) (Table 5.20)	Positive correlation with fat %E and negative with carbohydrate %E	Positive with Index1, n6 %E, fat %E, PUFA%E, MUFA %E, SAT FAT %E, protein %E and negative with carbohydrate %E	Positive with Index1, protein %E and negative with n-3 fatty acids %E
Nutrient, diet quality indices, Risk score with clinical risk markers (Table 5.22, 5.23 & 5.24) (Strongest statistically significant)	WC with energy, protein, total fat, MUFAs, Risk score. Glucose with diet cholesterol and Risk score. WC and glucose showed the most significant associations with most variables. Risk score showed the most statistically significant associations with all clinical risk markers.	WC with energy, total fat, PUFAs, n-6 fatty acids, Risk score. SBP with n-6:n-3, Risk score Glucose with protein, total fat, saturated fat, MUFAs, PUFAs, diet cholesterol, n-6, n-3, Risk score, fat %E, SAT FAT %E, carbohydrate %E. BMI with total fat, saturated fat and MUFAs. Risk Score with WC, SBP, glucose, cholesterol and triglycerides.	WC with total protein, diet cholesterol, Risk score. Glucose with total protein, diet cholesterol, Risk score. Risk score with WC, SBP, glucose, cholesterol, BMI, triglycerides
Regression analyses between factor scores and risk markers	Blood glucose with Factor 1 & 2	None	Blood glucose with Factor 2
Association between position in quintile for Factor 1 & 2, and risk markers	SCORE 2X with Factor 1 WC with Factor 1 Glucose with Factor 1 & 2 BMI with Factor 2 Cholesterol with Factor 1 & 2	None	None

Objective 3: To establish whether an association exists between the current trends in physical activity and the prevalence of risk markers for NCDs.

No statistically significant association was found between current trends in physical activity and the prevalence of risk markers for NCDs.

5.5 SUMMARY OF RESULTS

According to the MRC (2008, Figure 2.2), NCDs are related to the following risky behaviours: smoking, unhealthy diet and lack of exercise, and to the following risk factors: tobacco addiction, raised total serum cholesterol and other lipid abnormalities, obesity, diabetes and hypertension. The term *unhealthy diet* includes inadequate fibre, vitamins and minerals, excess salt, alcohol and kilojoules (MRC, 2008). The diet and lifestyle exposure of the study population in KwaDukuza will be summarised in terms of these risky behaviours and risk factors.

5.5.1 Risky behaviours

5.5.1.1 Smoking

Only 31% of the respondents smoked. These were mainly men (81%), with the highest observed frequency of <10 cigarettes per day. Both the European SCORE and its modification by 2x to determine the risk of CAD for Asian Indians more appropriately, took into account smoking to determine the risk for CAD. Low risks for CAD were shown using these algorithms, and therefore smoking was not clearly identified as a risky behaviour in this study.

5.5.1.2 Unhealthy diets

In terms of the results of INDEX1 and INDEX 2, the overall diet quality was good. However, in terms of classifying a diet as healthy or unhealthy according to intake of fibre, vitamins and minerals (Figure 2.2), a large percentage of respondents consumed less than the EAR for calcium, folate and vitamin D. In addition, there was no vitamin or mineral that was consumed in amounts greater than the EAR. In terms of fat intake, no respondent met the EAR for n-3 fatty acids whilst most of them met the EAR for n-6

fatty acids. Furthermore, when the results of this study were aligned to the WHO population goals for prevention of death and disability from NCDs (WHO, 2003b), the percentage of energy from n-3 fatty acids, the daily fibre intake and fruit and vegetable intake in g/day were below the recommended goals, whereas the percentage of energy derived from total fat, PUFAS, n-6 fatty acids and free sugars were above the recommended goals. Mean sodium intake g/day, cholesterol intake mg/day, %E saturated fatty acids and %E protein were within the acceptable range of intake, but %E from carbohydrates was slightly lower than the recommended WHO goals. Although 30.4% of respondents, mainly men, consumed alcohol, the frequency and quantity were small with only two beers, two ciders or two shots of brandy per week.

5.5.1.3 Lack of exercise

The results of this study showed that a high percentage of respondents (61.3% of men and 75.5% of women) were physically inactive, hence intensifying risky behaviour.

5.5.2 Risk factors

5.3.2.1 Raised total serum cholesterol and other lipid abnormalities

The results of this study showed that a large percentage of men (63%) had elevated serum cholesterol levels (>5.2 mmol/L), whilst triglyceride levels were high for the whole group (88% >1.69 mmol/L), making the lipid profile a risk factor.

5.5.2.2 Obesity

A large percentage of respondents were classified with obesity class I (44.8%) followed by 15.6% classified as having obesity class II. In addition, 100% women had a WC ≥80 cm, showing slightly greater risk than men.

5.5.2.3 *Hypertension*

The study was conducted on apparently healthy Indians. Only 6.4% of respondents were hypertensive, with a mean SBP of 123.5 mmHg. However, 93.6% of men and 92% of women had raised DBP.

5.5.2.4 *Elevated fasting blood glucose*

The mean fasting blood glucose was 5.5 mmol/L, with 38.8% respondents having levels >5.5 mmol/L.

5.4 *CONCLUSION*

In this chapter the results of the study were presented in line with the objectives. The results of the study will be discussed in Chapter 6, followed by recommendations and conclusions in Chapter 7.

CHAPTER 6

DISCUSSION

6.1 INTRODUCTION

In the previous chapter the results of the main study were presented. In this chapter, the results of the main study are discussed in relationship to the objectives and comparisons made with published results of Indians in South Africa, India and Asian Indian migrants elsewhere in the world, primarily in the UK where many studies compared characteristics of migrant South Asians with those of Caucasians. Although the South African Indian population represents one of the largest outside the Indian subcontinent (Ranjith *et al.*, 2005), relatively little is known about their diet and risk profile for NCDs.

6.2 DISCUSSION OF RESULTS

The results of this study will be discussed in relationship with the objectives but as a starting point the main characteristics of the respondents will be summarised which may be useful in making practical recommendations for interventions.

The results in Table 5.1 showed that a substantial proportion of respondents were professionals (46.4%) while 21.2% respondents were housewives. Most respondents used pharmacies and private doctors probably due to the convenience of these facilities as from personal experience most public health facilities are often overcrowded. In addition to convenience, local pharmacies provide in-house clinical facilities such as blood pressure, blood sugar and cholesterol checking at a reduced cost. This information is very valuable in identifying suitable channels namely: the workplace and private medical facilities that can easily capture the target audience for effective intervention.

6.2.1 Prevalence of risk markers

The first objective formulated for this study was: "to determine the prevalence of risk markers for NCDs, namely hypertension, increased fasting blood glucose, cholesterol and triglyceride levels, central obesity and physical inactivity".

The following results will be assessed in order to determine the prevalence of risk markers for NCDs: anthropometric and clinical results, the European SCORE and the modified algorithm and risk score associations with clinical factors. Although HDL-C levels were not known, it is clear from the results of other clinical and anthropometric measurements that the respondents had characteristics typical of the metabolic syndrome, which supports previous reports of Indians in South Africa (Ranjith *et al.*, 2007) and India (Kanjilal *et al.,* 2008).

6.2.1.1 Hypertension

This study was carried out on apparently healthy Indians and only 6.4% respondents recorded SBP levels >135 mmHg with a mean of 123.5 mmHg whereas 92.8% respondents recorded DBP levels >85 mmHg with a mean of 89.7 mmHg (Table 5.4 and 5.5). In view of the high DBP levels, Khan & Khan (2008) indicate that individuals with MetS and a normal systolic function most often show abnormalities in left ventricular diastolic function. These findings are also evident in individuals having one or two MetS criteria. By default many respondents in this study showed characteristics of pre-metabolic syndrome through anthropometric and clinical measurements which can be related to the high levels of DBP amongst respondents. Furthermore, SBP rises steadily with age whereas DBP increases through the 4th and 5th decade, plateaus and then declines in the 6th and 7th decade (Norman *et al.,* 2007) which could also explain the high DBP levels as the age cohort for this study fell within the 4th and 5th decades. Evidence on the prevalence of hypertension and its contribution towards the burden of CHD in South Africa was discussed in section 2.5.2.1. The nationally representative data on the prevalence of hypertension in South Africa (SADHS, 2003) showed that 13.3% of Indians reported hypertension, with the mean SBP of Indian men 128 mmHg and the mean of Indian women 126 mmHg (Department of Health, Medical Research Council & OrcMacro, 2007). The results of the current study were more favourable for

SBP, perhaps because individuals with any acute or chronic illness, using chronic medication, were excluded from the investigation. However, a large percentage (63.2%) of the respondents had a family history of hypertension (Table 5.1). Similar results were recorded for Indians in South Africa in a hospital environment where 60% of patients with a diagnosis of MI had a family history of hypertension (Ranjith *et al.,* 2004). In a random house-to-house study of 1 000 Indians in Durban, the prevalence of essential hypertension according to the WHO was 19% (men 15%, women 22%) (Seedat, 2009). Seedat and co-workers (1990) found that the following factors were significantly associated with hypertension in Indians: hypertriglyceridaemia, obesity, hyperuricaemia, hypercholesterolaemia, excess alcohol intake and diabetes. The current study on apparently healthy Indians excluded diabetics and included only 30% who reported consuming moderate quantities of alcohol. SBP correlated significantly with all the other clinical and anthropometrical parameters except fasting glucose in men whereas DBP only correlated with SBP for men. Amongst women, DBP correlated with SBP, triglycerides and BMI (Table 5.23).

The results of studies amongst native Indians varied compared with the current study. Balagopal *et al.* (2008) reported a similar mean SBP of 125.2 mmHg for men and 119.5 mmHg for women and a mean DBP of 89 mmHg for men and 83 mmHg for women in a rural village of India. However, Radhika *et al.* (2007) reported lower levels of blood pressure (mean SBP of 118.7 mmHg for men and 115.8 mmHg for women; mean DBP of 74.5 mmHg for men and 71.8 mmHg for women) in an urban study in South India. Results similar to the current study were presented by migrant Asian Indian studies. Ajjan *et al.* (2007) reported a mean SBP of 124 mmHg for men and 129 mmHg for women and Lovegrove *et al.* (2004) reported a mean SBP of 122 mmHg for Indo-Asians in the UK.

It is evident from the results of the prevalence of hypertension, particularly with increased DBP, that recommendations to control and maintain blood pressure need to be incorporated into the strategies for an integrated programme of prevention.

6.2.1.2 Cholesterol and triglyceride levels

Evidence on the prevalence of elevated blood cholesterol and other lipid abnormalities as a risk factor for CHD in South Africa was described in section 2.5.2.2. In terms of cholesterol attributable mortality rates in South Africa, the highest prevalence was found amongst the Indian population, where it accounted for 22.2% of all deaths (Norman *et al.,* 2006). In the current study, a large percentage of men (91.8%) and women (86.3%) recorded a triglyceride level >1.69 mmol with a mean of 2.2 mmol/L for men and 2.4 mmol/L for women whereas 63% of men and 21.5% of women had raised cholesterol levels with a mean of 5.03 mmol/L for the whole sample. Elevated serum triglyceride levels (>2 mmol/L) were also reported by Ranjith *et al.* (2005) in 49% of young (<45 years) Indian patients with acute coronary syndrome in a large hospital in Durban. The biochemistry on admission of the patients <45 years recorded a mean triglyceride level of 2.6 mmol/L. In middle-aged patients the prevalence was somewhat lower where patients >45 years revealed a mean of 2.2 mmol/L. According to Thomas *et al.* (1986) low HDL-C and raised triglycerides are common characteristics of South Asian populations and is more pronounced in migrant Indian populations (Bhatnagar *et al.,* 1995). In an Asian migrant study by Ajjan *et al.* (2007) respondents were categorised according to the prevalence of MetS where a mean triglyceride level of 1.5 mmol/L was recorded for respondents without MetS and 2.0 mmol/L for those with MetS. In the current study, respondents could not be stratified according to prevalence of MetS because HDL-C values were not available. Another possible reason for the high triglyceride levels in an Asian migrant population was reported in the study by Yagalla *et al.* (1996) were a high carbohydrate intake and uneven diurnal distribution (no breakfast and large evening meals) were associated with high triglyceride levels and low HDL-C levels. The association between dietary factors and clinical factors are discussed later on in this chapter and it would be interesting to see if a pattern exists between carbohydrate intake and the increased triglyceride levels as reported by Yagalla *et al.* (1996).

There has been agreement that insulin resistance and WC are significantly correlated with triglyceride levels amongst South Asian men and women (Banjeri *et al.,* 1999; Panaliappan *et al.*, 2007). The high triglyceride levels in the current study may be explained by the high prevalence of central obesity amongst respondents (87% for men

and 100% for women). Furthermore, in a study by Singh *et al.* (1999), BMI, cholesterol and triglyceride levels showed an increasing trend associated with increased body fat, where respondents with >25% body fat reported higher triglyceride levels (1.9 mmol/L), as compared with respondents with 20-25% body fat (1.66 mmol/L). In the KwaDukuza study population the mean percentage body fat was 32.2%.

Studies have shown that the lipoprotein profile is more favourable in native Indians compared with migrant Indians. Misra *et al.* (2001) reported lower mean cholesterol levels of 5.03 mmol/L for men and 4.7 mmol/L for women. In the same study, the mean triglyceride levels reported were 1.3 mmol/L for men and 1.46 mmol/L for women. In comparison to Asian migrant studies, Yagalla *et al.* (1996) reported a mean cholesterol level of 5.4 mmol/L and a mean triglyceride level of 1.54 mmol/L. The mean cholesterol level reported in the current study is comparable to migrant Asian Indian studies in the UK but higher than in native Indians, however, the recorded mean triglyceride level for this study was way above the comparative range, which raises concern. A possible explanation for the difference in triglyceride values in native compared with migrant Asians could be the lower intake of long-chain n-3 PUFAs in migrant Indians compared with age- and weight-matched Europeans as was found by Lovegrove *et al.* (2004). In the current study the mean n-3 intake (0.24% of Energy) was even lower than in the migrant Indians in the Lovegrove *et al.* study (0.7%E). Other explanations may be the availability of the western diets (especially refined grains) and higher disposable incomes.

The findings of this study, in particular with regard to raised triglyceride levels, call for recommendations to maintain and control a desirable lipid profile.

6.2.1.3 Fasting blood glucose

Raised fasting blood glucose levels were seen in 39% of respondents. The mean fasting blood glucose level of men was 5.41 mmol/L and that of women 5.5 mmol/L. Evidence on the prevalence of increased fasting blood glucose as a risk marker for diabetes in South Africa was presented in section 2.5.3.1. The study by Misra *et al.* (2001) on the nutrition profile of urban slum dwellers in India reported a mean fasting blood glucose of 4.7 mmol/L for men and 4.8 mmol/L for women. Similar results were

presented by a migrant Asian Indian study by Ajjan *et al.* (2007), where respondents were classified according to the prevalence of MetS according to the IDF definitions. The mean fasting blood glucose level was 4.8 mmol/L for respondents without MetS, which was lower than the level reported in this study. However, the mean fasting blood glucose level was 5.6 mmol/L for respondents classified with MetS (Ajjan *et al.,* 2007). According to Seedat (2005), South African Indians have a 16.2% prevalence rate of diabetes and a 7.3% impaired glucose tolerance rate

6.2.1.4 Central obesity

In order to determine the prevalence of central obesity as a risk marker for NCDs, the anthropometric means and frequencies presented in Tables 5.2 and 5.3, using Asian cut-off points, will be used. The results clearly show that a large percentage of men and women fell in the obesity class I BMI category with a mean BMI of 26.1 kg/m² for men and 26.6 kg/m² for women. In addition, 100% women had a WC ≥80 cm and 87.4% men had a WC ≥90 cm, indicating central obesity. In terms of WHR, men recorded a mean of 0.91 and women 0.87. Furthermore, 62% men and 68% women fell in the 30 to 39% category for percentage body fat where men had a mean of 31.7% and women had a mean of 32.6%. Evidence on the prevalence of obesity in South Africa as a risk marker for hypertension, increased risk to insulin resistance and CVD has been discussed in section 2.5.2.3. In the 2003 SADHS (Department of Health, Medical Research Council & OrcMacro, 2007) 44.3% of Indian men and 59.2% of Indian women were overweight or obese with mean BMIs of 24.8 and 26.6 respectively for men and women. Compared with these figures, it appears that the BMI of Indian men increased in recent years but stabilised for women

It is a well established fact that Asian Indians have a smaller build and excess body fat with predominant abdominal adiposity as compared with Caucasians. They tend to accumulate intra-abdominal or visceral fat without developing generalised obesity (Ghosh *et al.,* 2004). As it has been established that body fat and BMI are different among different ethnic groups, Smalberger (2008) suggested that BMI cut-off points should be population specific. The relationship between BMI and body fat percentage was found to be different in Indians compared to Caucasians in Singapore

(Deurenberg-Yap *et al.*, 2000) and these authors noted the paradox of low BMI and high body fat percentage among Indians.

For comparison purposes to other studies, most studies did not incorporate ethnicity specific cut-off points to define obesity. Therefore, only studies using similar cut-off points as the present study will be compared. In a community-based diabetes prevention programme conducted in India by Balagopal *et al.* (2008), 32% men and 68% women were classified as obese (BMI>25.0 kg/m). In another study by Parimalavalli *et al.* (2009) on the anthropometric profile and nutrient intake of women in Tamil Nadu, 31% were classified as obesity class 1 and 16% were classified as obesity class 11. Balagopal *et al.* (2008) reported a mean WHR ratio of 0.9 for men and 0.8 for women with 66% men recording a WHR >0.89 and 53.9% women recording a WHR >0.81. Similar results were reported in several Asian Indian migrant studies. In the study by Yagalla *et al.* (1996) on Indian male physicians in the USA, the mean WHR was 0.91. Lovegrove *et al.* (2004) reported a mean WC of 88.6 cm amongst British Indo-Asians whereas Ajjan *et al.* (2007) reported a mean WC of 86 cm in South Asians without MetS and 95 cm in those with MetS. The gender of participants in the last two studies was not mentioned.

With reference to percentage body fat, in a study amongst Indians in the urban slum areas in Northern India, Misra *et al.* (2001) found that women had a high mean percentage body fat (26.7%), where the percentage body fat was defined as an indicator of obesity if it was greater than 25% in men and 30% in women (Hortobagyi *et al.,* 1994). Using the same definitions of percentage body fat as an indicator of obesity, the results in the current study showed that both men and women had a high mean percentage body fat (31.7% for men and 32.6% for women). The findings of this study in relation to central obesity are comparable to the results presented by Asian Indian migrant studies and studies in the Indian sub-continent, clearly indicating that Indians in KZN have a preponderance towards central obesity. Recommendations to reduce WC are merited by the prevalence of central obesity and the fact that visceral fat is a reliable marker of insulin resistance and hyperinsulinaemia, which are risk markers for the development of hypertension, dyslipidaemia, diabetes and CHD (Das, 2003). There is a large body of evidence on the association of the accumulation of abdominal fat and increased risk of insulin resistance, diabetes, hypertension, dyslipidaemia and

atherosclerosis (Lovejoy *et al.*, 2001). In this study WC correlated positively with SBP, glucose, total serum cholesterol, triglycerides and BMI (Table 5.21), displaying the characteristics of MetS in the respondents. Weight reduction may improve most of these metabolic abnormalities. For instance, a blood pressure reduction of 4.4/3.3 mm Hg can be expected when 5 kg is lost (Neter *et al.*, 2003).

6.2.1.5 Combination of risk markers

Algorithms, namely the European SCORE and the modified doubled SCORE, were used to determine the ten-year risk of a first fatal atherosclerotic event for respondents, as presented in Tables 5.6 and 5.7. The European SCORE incorporated the following risk markers in relation to objective one of the study: SBP and cholesterol as well as age, gender and smoking. Most respondents showed a <1% chance of risk of fatal CVD in ten years. However, this algorithm was doubled, as suggested by Enas *et al.* (2008), as Asian Indians have twice the risk of NCDs. This modification showed that most respondents had a 1% chance of developing fatal CVD in the next ten years with 1.2% having an 8% risk level. The overall risk markers used in this algorithm, namely cholesterol, SBP, gender, age and smoking, showed a minimal risk for CVD. Hence the prevalence of raised cholesterol and hypertension was watered down when used with a combination of other variables in this algorithm, namely age and smoking.

6.2.1.6 Physical activity

Physical inactivity is a well documented risk marker for NCDs and is discussed in detail in section 2.5.1.4. According to Table 5.19, a large percentage (65.2%) of respondents were classified as inactive (<600 METS min), mainly women. The pedometer step counting results was comparable to the GPAQ where 69.2% respondents were classified as inactive, mainly women. Similarly, the 2003 SADHS (Department of Health, Medical Research Council & OrcMacro, 2007) using the same classification categories as this study, revealed that 43.1% Indian men and 62.1% Indian women were classified as inactive (<600 METS min). According to the 2003 SADHS, few dedicated walking and cycling routes as well as safety and security issues are likely to compound the problem of low physical activity. In terms of studies amongst Indians in India with respect to physical activity, the study conducted by Balagopal *et al.* (2008) in

a rural village of India showed that 40.9% men and 65% women were physically inactive. With regard to physical activity levels amongst migrant Asian Indians, Fishbacher *et al.* (2004) conducted a review on the physical activity level amongst South Asians in the UK. The review showed that South Asians had a substantially and consistently lower level of physical activity compared with Europeans (Fishbacher *et al.,* 2004). These comparative studies support the findings of this study consistently showing an inherent trend of physical inactivity mainly amongst women. Table 5.1 showed that 21.2% of the total respondents were housewives. Although they might have been gainfully occupied doing the cooking and cleaning throughout the day, these activities are often equated to lower METS. This problem is further intensified by the daily preparation of a number of side dishes in addition to a main dish as more time is spent in the kitchen than engaging in physical activity to lower and maintain body weight. Furthermore, Fischbacher *et al.* (2004) state that another possible reason why physical activity levels are low is due to religious and cultural issues as reported by Asian Muslim women. With the observed low physical activity levels amongst Indians, it is imperative that appropriate interventions have to be considered in line with strategic targeting of a captured audience.

There is ample evidence that diet and exercise work better together than either component alone in the treatment of obesity and the metabolic syndrome components. Generally speaking, diet has been shown to be more effective for overall *weight loss*, but exercise has more of an effect on *weight maintenance* and *changes in body composition* over time (Sullivan, 2006).

6.2.2 Association of nutrient intakes with prevalence of risk markers

The second objective formulated for this study was: "to establish whether an association exists between current trends in nutrient intakes and food patterns, with special reference to n-6:n-3 fatty acid ratio and the prevalence of risk markers for NCDs".

In order to assess this objective, the current trends in nutrient intakes and food patterns will first be discussed. Thereafter, the observed associations between the nutrient intakes and the prevalence of risk markers will be discussed.

6.2.2.1 *Current trends in energy and nutrient intakes*

In terms of the current trends in nutrient intakes, Table 5.12 compared the mean intakes with the population nutrient goals for the prevention of death and disability from NCDs by WHO (2003b). The mean percentage energy from total fat, free sugars and n-6 fatty acids were above the recommended goal. The percentage energy from fat was 35.1% for men and 33% for women. In the study conducted by Wolmarans *et al.* (1999) on the dietary intakes of Indians living in the metropolitan area of Durban, similar results were presented for the percentage energy from fat where the results of the study were stratified according to different age cohorts. For the purposes of comparison to this study, the 35 to 44 year and 45 to 55 year age cohort will be used. Under the 35 to 44 year age cohort, men reported 33% and women 36% energy from total fat. For the 45 to 54 year cohort, men reported 35.9% and women 32.8% energy from total fat.

Similar results were presented by Asian Indian migrant studies. In a study on Asian Indian male physicians living in the United States, 32% of energy was derived from total fat (Yagalla *et al.,* 1996). The Indo-Asian study by Lovegrove *et al.* (2004) reported 37% energy from fat. In comparison with migrant Asian Indian studies, a native Indian study on the nutrition profile of urban slum dwellers in India by Misra *et al.* (2001) reported lower percentage energy from fat within acceptable range of the WHO recommended goals (24.7% for men and 28.7% for women).

Fatty acids

In the current study, the mean percentage energy from n-3 fatty acids and from carbohydrate, as well as the mean daily intake of fruit and vegetables and fibre, were below the recommended WHO goals. The mean fibre intake in this study was 18.8 g/day for men and 18.1 g/day for women, which is much higher than in the study by Wolmarans *et al.* (1999), where men had a mean intake of 8.3 g/4.2 MJ/day (less than 17 g/day) and women had a mean intake of 9.1 g/MJ/day (less than 14 g/day). In a migrant Asian study on Indo-Asians, Lovegrove *et al.* (2004) reported a mean fibre intake of 16.2 g/day. All these studies, however, reported fibre intake below the recommended WHO goals. The mean fruit and vegetable intake of men in the current study was 221.9 g/day and of women, 236.9 g/day, which was below the recommended

WHO intake of ≥400 g/day (WHO, 2003b). Nearly all the men (95%) and women (93.5%) consumed less than 400 g/day. The study by Radhika *et al.* (2008) on the association of fruit and vegetable intake with cardiovascular risk factors in urban South Indians reported a mean intake of 259.6 g/day for men and 270.4 g/day for women which is slighter higher than the findings in this study, but still lower than the recommended WHO goals. The highest quartile of fruit and vegetable intake in their study (more than 327 g/day) showed a significant inverse association with SBP, BMI, waist circumference, total cholesterol and LDL-cholesterol. These findings highlight the shortfall of the fruit and vegetable and the fibre intake in the KZN Indian diet and it underscores the importance of increasing fruit and vegetable and fibre intake for the purposes of intervention. An increment of one serving per day of fruit or vegetables is associated with a 6% lower risk of ischaemic stroke (Joshipura *et al.*, 1999). Although Indians in South Africa use a large variety of fruit and vegetables in their diet, a number of factors may be associated with the low intake of fruit and vegetables. Firstly, the use of the term vegetables is often used inconsistently, sometimes incorporating all vegetables plus legumes and pulses as in the case of the 5-a-Day campaign in South Africa (TRUST: 5-a-Day for Better Health, 2009). However, in the current study as well as in the WHO goal for fruit and vegetable intake (WHO, 2003b) legumes were not included in the fruit and vegetable group. Legumes provided about 6% of the total energy in the study. Secondly, as a method of extending the main dish for increasing the portion yield and adding variety to the dish, vegetables are often added to a meat dish e.g. cabbage to mutton, which were classified under the meat group rather than under the vegetable group. In doing so, the actual intake of vegetables was masked. Hence, it is important to factor these possible reasons for the low intake of fruit and vegetables into the recommendations for intervention. Furthermore, in totality the increase of fruit and vegetables would be only effective if healthy methods of cooking are used as the predominant method of cooking vegetables is sautéing in sunflower oil.

Wolmarans *et al.* (1999) reported a high intake of PUFAs, more than 10% energy in Indians with a mean dietary P:S ratio of 1.38-1.96 in men and 1.55-1.95 in women. Likewise, in this study, PUFAs provided more than 10% energy but the P:S ratio was lower than the study by Wolmarans *et al.* (1999) (1.1 for men and 1.2 for women). Wolmarans *et al.* (1999) justified the high P:S ratio on the basis that sunflower oil is the oil of choice in Indian cuisine. As a personal observation, currently, on average an

Indian household of four members will use up to five litres of sunflower oil for food preparation per month which accounts for the high P:S ratio. A possible reason why the P:S ratio is slightly lower than the study by Wolmarans *et al.* (1999) could have developed from the standardisation of indigenous recipes which did not appear in the Food Finder 3® analysis programme. Although the standardisation of recipes was based on the average of three recipes with the same recipe yield, the amount of sunflower oil used in these recipes differed greatly between respondents and therefore possibly did not reflect true fat intake of the entire sample. Furthermore, although sunflower oil is still used predominantly in food preparation in Indian households, other oils such as olive and canola oil have become more available in supermarkets giving consumers more choice. In this study, the mean percentage energy derived from n-6 fatty acids was 11.7% for men and 10.3% for women which exceeded the recommended WHO goal of 5 to 8%. In contrast, the percentage energy derived from n-3 fatty acids was 0.25% for men and 0.23% for women which was much lower than the WHO recommendation of 1 to 2%.

The intake of PUFAs expressed in terms of mass is a more optimal approach to n-6 and n-3 fatty acid balance than a ratio (Wijendran and Hayes, 2004) and the UK Food Standards Agency Workshop (Stanley *et al.*, 2007) recommended that the use of the ratio to estimate CVD risk should be abandoned. However, the ratio of n-6:n-3 fatty acids was originally one of the objectives of this study and very high mean ratios for n-6:n-3 fatty acids were reported for both men and women. Sevak *et al.* (1997) showed that South Asians consumed significantly less n-3 fatty acids than Caucasians in the UK (0.08% E *vs.* 0.13% E) but more n-6 fatty acids (5.4%E *vs.* 5.0%E). In another migrant Asian Indian study, Lovegrove *et al.* (2004) reported that the percentage energy derived from n-6 and n-3 fatty acids was 7% and 0.7% respectively. Respondents' diets in the Lovegrove study were then supplemented with either fish oil (4.0 g/day) or olive oil (control, 4.0 g/day) for twelve weeks. Fish-oil supplementation reversed low n-3 fatty acid levels and lipid abnormalities and was considered as an important, yet simple, dietary manipulation to reduce CAD risk in Indo-Asians with an atherogenic lipoprotein profile (Lovegrove *et al.,* 2004). In the study by Misra *et al.* (2001) on the nutrition profile of urban slum dwellers in India, the observed percentage energy from n-3 fatty acids was 1.3% for men and 1.7% for women compared to 3.1% and 3.8% for men and women respectively for n-6 fatty acids. It appears from the results of the current study

and the trend displayed by other published studies that the n-3 fatty acid intake of migrant Indians is below the recommended WHO goal while n-6 fatty acid intake is in excess of the recommended goal (Lovegrove *et al.,* 2004).

Possibly the n-3 fatty acid intake was low in the current study because most sources rich in n-3 fatty acids do not feature in the South African Indian diet for several reasons. First, oily fish intake is largely restricted due to availability, high cost and restrictions imposed by the Sustainable Seafood Initiatives (SASSI) based on sustainable choices from the South African fish populations. Sardines which are very popular in the Indian diet are seasonally available for only one month in a year. Although it can be purchased frozen, unfortunately there is a perception that it is only used as bait for the purposes of fishing. Furthermore, canned pilchards (in tomato sauce) and sardines are eaten only in small quantities as fresh fish is preferred. Secondly the use of flaxseed/linseed and its derivatives is very limited due knowledge barriers and the high cost. Thirdly sunflower oil is the preferred cooking oil as compared to the n-3 fatty acid rich canola oil mainly due the fact that Indians use large quantities of sunflower oil which is cheaper than canola oil. However, it must be borne in mind that canola oil would be a more suitable substitute for sunflower oil compared with olive oil as it does not mask the flavour of the traditional herbs and spices used in the Indian cuisine. Caution must be applied with the suggestion of using canola oil as an appropriate substitute for sunflower oil. Johnson *et al.* (2007) showed that the substitution of canola oil for other vegetable oils increases compliance with dietary recommendations for SFA and MUFAs and α-linolenic acid but not for linoleic acid. The n-6 fatty acid percentage energy in this study was 10.3% and 11.7% for men and women respectively and although the WHO goals recommend 5-8%E from n-6 fatty acids, the FNB and the AHA recommend at least 5-10%E for n-6 fatty acids. In fact, Harris *et al.* (2009) state that randomised trails in humans have shown reduced CHD risk with n-6 fatty intake of 11 to 21%E for up to 11 years with no harm and they are of the opinion that reduction of n-6 fatty acid intakes from their current levels would be more likely to increase than to decrease the risk of CHD. Therefore, in line of the argument by Harris *et al.* (2009), it would be more appropriate to recommend a mixture of two oils such as sunflower oil and canola oil which in terms of taste, cost and health reasons would be a more viable and acceptable option in Indian communities.

Many studies have made a dietary recommendation to increase the n-3 fatty acid levels in the diet. In a South African study, Van der Walt *et al.* (2008) reported that wild-growing dark green leafy vegetables (morogo) are rich in folate, with n-3 fatty acids in excess of n-6 fatty acids. Indians in South Africa consume these dark green leafy vegetables as a side item in small quantities as an accompaniment to the main dish. Ways to increase n-3 fatty acids in the Asian Indian habitual diet have also included increasing green leafy vegetables, rajmah (kidney beans), bajra (Sorghum *vulgare*) and chana (black gram), lobia (cow pea) and methi (fenugreek) (Misra *et al.,* 2001; Misra *et al.*, 2009b).

Carbohydrates

The percentage energy from carbohydrates (47.2% for men and 45.4% for women) was below the 55-75% range as recommended by the WHO goals (WHO, 2003b). Native Indians consume relatively more carbohydrates in the range of 59-65% energy (60.1% for men and 59.1% for women) (Misra *et al.,* 2001) as compared with migrant Asian Indian studies such as those of Lovegrove *et al.* (2004) (44% for men and 47% for women) and Sevak *et al.* (1994) (46%) in the UK. Furthermore, the reported range for percentage energy from carbohydrates by Wolmarans *et al.* (1999) on the dietary intake of Indians living in Durban was similar to this study for men and slightly higher for women (men: 49.3% and 45.5% respectively for the 35-44 year and the 45-54 year age cohorts) (women: 49.1% and 53% respectively for the 35-44 year and the 45-54 year age cohorts).

Energy intake

In contrast to the lower percentage energy from carbohydrates, the sugar intake in the current study was higher than the WHO goals. The mean percentage energy derived from free sugars was 12.5% for both men and women, which exceeded the WHO goals (<10%E). However, this range was comparable to the findings by Wolmarans *et al.* (1999) where the percentage energy from sugars was 13.5% for men and 11.2% for women. Furthermore, the mean energy intake was 7815 kJ for men as compared to a slightly higher range reported by Wolmarans *et al.* (1999) where the mean energy intake was 8500 kJ for men in the 35-44 year and 8100 kJ for the 45-54 year age cohorts. However, the mean energy intake for women (7214 kJ) in this study was much higher than in the study of Wolmarans *et al.* (1999) (5600 kJ and 5400 kJ respectively

for the 35-44 year and the 45-54 year age cohorts). Table 5.9 showed the percentage of men and women who under-reported (42.3% of men using the 72% of EER cut-off point and 13.7% of women using the 74% cut-off point) presenting higher under-reporting by men as reflected in the results of the reliability and comparative validation sample in Chapter 4. It was reported in Chapter 4 that a possible reason for the high level of under-reporting amongst in Indian men could be attributed to their difficulty in determining the portion sizes as they are not involved in food preparation. Similar results of under-reporting was presented in the study by Wolmarans *et al.* (1999) where the ratio of energy intake to basal metabolic rate (EI: BMR) was low for all age groups, reflecting under-reporting.

Protein

The percentage energy from protein (12.8% for men and 12.0% for women) fell within the range of WHO goals. A possible reason explaining the prudent intake could be due to the fact that the indulgence of animal protein is limited to certain days of the week due to religious worship which comes in the form of abstinence from meat. In days of abstinence from meat, the protein intake is balanced by the consumption of legumes. About 11%E from protein was supplied by legumes in this study. Furthermore, in the study by Wolmarans *et al.* (1999) a high percentage of energy came from plant protein.

Nutrient intakes /EAR

To assess the adequacy of the diet, the nutrient intakes of the QFFQ were compared with the EAR (Food and Nutrition Board, 2000). All respondents consumed less than the EAR for n-3 fatty acids (Table 5.13). In addition, a large percentage of respondents consumed less than the EAR for fibre, calcium, folate, thiamin and vitamin D, highlighting the low intake of fruit and vegetables. These results can be compared with only one other South African study on Indians, namely that of Mia and Vorster (2000) on Indian adolescents in Lenasia, since Wolmarans *et al.* (1999) measured only macro-nutrients in Indian diets. The diet of the adolescents was low in carbohydrate, high in fat, relatively high in plant proteins and fibre, more than adequate in ascorbic acid but relatively low in calcium. Therefore, it seems as if the diet of the adolescents was of somewhat better quality than that of the KwaDukuza adults. However, according to the indices of overall diet quality, the dietary pattern of the KwaDukuza respondents was good.

6.2.2.2 *Trends in food patterns*

To measure overall diet quality, most published studies as reported in a review by Kant (2004) use one of two methods namely: diet indices and data-driven methods which use factor analyses to derive food patterns and possibly explain nutrient-disease relationships. In the present study both methods were used. To report on diet quality, indices were calculated (Table 5.14), where the higher the index, the better the diet quality. Index 1 reported a mean of 2259.8 and Index 2 reported a mean of 518 of which Index 1 observed the highest frequency in the 2000-2500 category and Index 2 observed the highest frequency in the 500-559 category (Table 5.15) all of which reflect good diet quality. However, Index 2 had an inverse correlation with Risk score and Risk score 1 (Table 5.22). The higher the Risk Score and Risk score 1, the greater the risk towards CAD whereas a high index indicates a better quality of diet (i.e. the closer the diet is to the WHO recommendations). Therefore, the significant inverse correlation implies that the closer the diet was to the WHO recommendations, the lower the risk for CAD. Index 2 was also inversely correlated with WC for the whole sample and for men (Table 5.22 and 5.23), which could be expected: the better the quality of the diet the lower the waist circumference.

Two principal components were identified based on the percentage fat contributed by the food groups (Table 5.18). The reasons why the percentage of fat contributed by each food group was used to derive food patterns was firstly based on one of the primary objectives of this study namely: to determine whether an association existed between nutrient intakes and food patterns with special reference to n6:n-3 fatty acid ratio on the prevalence of risk markers for NCDs. Secondly, the use of factor analysis to derive dietary patterns is relatively new and although most studies use percentage energy to derive factors (Newby *et al.,* 2004a; Newby *et al.,* 2004b; Mikkilä *et al.,* 2007) the high level of underreporting of energy intake in this study implied that if percentage energy intake was used to derive Factors, it might not reflect true intake. Therefore, using the percentage fat contributed by each food group, Factor 1 was positively loaded for legumes, cereal and cereal products and vegetables and Factor 2 reflected a diet pattern where the highest factor loadings were from the fats and oils, sugar and sweets and the milk groups. The cooking practices and ingredient usage in South African

Indian cuisine are in agreement with Factor 1. The fat content in cereals and cereal products is based on added fat e.g. rotis are brushed with butter or ghee; onions and spices are sometimes tempered in butter or ghee and tossed into boiled rice; savoury pastries such as samoosas and puris are fried in sunflower oil; and butter or ghee is used in sweet puddings made from semolina, vermicelli (pasta) or white rice. The high addition of fat in cereal dishes could be justified on the basis of taste as most cereal dishes are bland. The high fat content in most legume dishes comes from the tempering of butter or ghee with spices for the purposes of finishing the dish e.g. split pea dhal. Vegetables are mainly prepared using sunflower oil, hence the high fat factor loading for vegetables. Factor 2 (fats and oils, sugars and sweets and milk group) had lower loadings than factor 1. The loading on fats and oils accounts for visible fat which can be controlled by choice e.g. many dishes use sunflower as a base for sautéing and taste is determined by spices unlike legume and cereal dishes in Factor 1. Sugars and sweets had slightly lower loading than fats and oils. Although a high fat content is used in the preparation of traditional Indian sweet meats (fudges and pastries), dishes of this nature are only prepared during festivities. According to Kant, (2004) the majority of studies examining the dietary patterns frequently choose to name the factor with the lower fat loading, higher fruit and vegetable and whole grain loading, the healthy pattern. The two dietary patterns derived from the factor analyses based on fat intake shows that Factor 1 with more positive loadings would be the unhealthy dietary pattern for Indians in KZN. Therefore, with the factor analysis being based on fat intake, it alerts the need to modify this dietary pattern by the reduction of added fat in these food groups. Factor 2 showing weaker loadings may be the healthier pattern as it comes from more visible sources of fat and are therefore easier to control.

According to regression analyses (Table 5.25), blood glucose showed the strongest ß-coefficients with Factor 1 and Factor 2 scores. While the ß-coefficient between Factor 1 and glucose (-0.16) for the whole sample was not significant, Factor 1 scores for the men and with Factor 2 scores for the whole sample and the women were significantly inversely correlated with blood glucose levels. Therefore, the higher the fat loading, the lower the blood glucose, which may be explained by fact that fat lowers the glycaemic index of foods (Collier & O'Dea, 1983) and therefore the glycaemic load. High glycaemic index diets have been associated with high postprandial blood glucose concentrations (Ludwig, 2002). This is confirmed by the medians of quintiles (Table

5.26) where although the median of the mid quintiles are higher than quintile 1, the median for quintile 5 is the lowest. The ß-coefficients were also negative for triglycerides which can be justified by the fact that the triglyceride levels are increased by refined carbohydrates. Although, BMI and cholesterol had negative ß-coefficients, they were very small, indicating a negligible correlation. In studies using percentage energy to calculate factors, Newby *et al.* (2004a) reported that a pattern high in reduced fat dairy products, cereal, fruit, fibre and other healthy foods was inversely associated with plasma triglycerides and in the study by Mikkilä *et al.* (2007), a dietary pattern reflecting "health-conscious" food choices was inversely related to CAD, but less strongly than the positive association of the traditional food pattern with CAD.

Furthermore, in order to determine the association between the two derived food patterns and risk markers, factor scores were divided into quintiles and the medians of the various risk markers for the lowest, middle and highest quintiles compared using the Jonckheere-Terpstra test (Table 5.26). Table 5.26 shows that Factor 1 was significantly associated with SCORE 2X (SCORE adjusted for the Indian population), WC, glucose and cholesterol. Factor 2 was significantly associated with glucose and BMI. However, it appears that in Factor 1, the medians for the mid quintile are higher for SCORE 2X, WC, glucose and cholesterol and although P for trend is significant there is only a small difference between the highest and the lowest quintiles.

In addition, the following trend was evident for the food group intake: for macronutrients, the cereal group provided the highest percentage of energy, carbohydrates, fat, saturated fat and PUFAs. Similarly, for micronutrients, the cereal group also provided the highest percentage of iron, vitamin B6, riboflavin, thiamin and folate (Tables 5.16 and 5.17).

It is evident from the factor analysis and the food group intake in terms of the macronutrient and micronutrient percentage energy intake, that cereal and cereal products, vegetables and legumes reflect the current pattern of intake amongst Indians in South Africa. Similarly, Wolmarans *et al.* (1999) also showed in their study that Indians in Durban reflected a cereal-based, vegetable dietary pattern. This information is valuable in making appropriate recommendations around the dietary patterns so as to

create a level of acceptance in the Indian community, as drastic changes are often difficult to accept and sustain.

6.2.2.3 Associations of nutrient intake with clinical risk markers

In order to determine the associations of nutrient intake, percentage energy from nutrients, diet quality indices and risk scores, Pearson partial correlations were performed (Table 5.20). There was a significant correlation of Risk Score (the aggregate total of all risk markers defined by Asian standards) and Risk Score 1 (aggregate total with fasting blood glucose counted twice as much as other risk markers) with Index 1 (positively) and Index 2 (negatively) and the percentage energy from n-6 fatty acids, total fat, PUFAs, MUFAs and protein (positively) and carbohydrate (negatively) for men. The strongest correlation was between percentage energy from fat with Risk Score and Risk Score 1, emphasising the unhealthy relationship between fat and risk markers for NCDs. There was a weak negative correlation between percentage energy from n-3 fatty acids for women and SCORE which indicates that a high percentage energy from n-3 fatty acids was related to low risk for CAD in this study group. However, there was a positive correlation between percentage energy from n-6 fatty acids and SCORE, Risk Score and Risk Score 1, which indicates that a high percentage energy from n-6 fatty acids was related to an increased risk of CAD.

Tables 5.22 to 5.24 presented the results of the associations of nutrients, percentage energy from nutrients, diet quality indices, Risk score and Risk score 1 with clinical risk markers. WC showed significant associations with most variables. Risk score showed the strongest statistically significant association with clinical risk markers for the whole group. The correlation (albeit weak) between waist circumference and percentage carbohydrate was an inverse one, indicating a possible favourable effect of higher carbohydrate intake on waist circumference in this study group. For men, WC and glucose showed the most significant associations with most variables. Risk score showed the strongest statistically significant association with clinical risk markers, which were positive for all, but negative for Index 2. For women, WC and glucose showed the most significant associations with most variables and Risk score showed the strongest statistical significance with clinical risk markers. Furthermore, in women, n-3 and n-3%E did not correlate with any risk markers, but in men n-3%E correlated positively

with glucose and triglycerides. This was a rather unexpected finding since ALA, EPA and DHA have been shown to decrease triglycerides (Egert *et al.*, 2009). Furthermore, neither n-3 nor n-3 %E correlated significantly with SBP or DBP. However, the n-6:n-3 ratio correlated significantly with SBP and DBP in the whole group, and the ratio as well as n-6%E correlated with SBP in men. The ratio correlated inversely with DBP in women, which is easier to explain since it is known that the long-chain n-3 fatty acids lower blood pressure, but controlled, double-blind studies do not support an independent effect of linoleic acid on blood pressure (Pietinen, 1994).

PUFAs, n-6 and n-3 fatty acids showed significant associations with blood glucose. Popp-Snijders *et al.* (1987) indicated that n-3 fatty acids improve insulin sensitivity in individuals with impaired glucose tolerance. However, intervention studies did not find encouraging results regarding the effect of n-3 fatty acids on insulin sensitivity (Brady *et al.,* 2004) and the OPTILIP study by Griffin *et al.* (2008) found no effect of n-6/n-3 fatty acid on insulin sensitivity. Mann (2006) suggested that insulin sensitivity is also improved when saturated fatty acids are replaced with PUFAs.

Collectively the prevalent associations in this study call for weight reduction which would improve the risk profile of the respondents. An awareness of the phenomenon of central obesity needs to be created as currently the ramifications of increased WC is not understood by the community and is often loosely termed the "tyre syndrome". Moreover, there is a general belief that it starts in women after giving birth as most adolescents and young adults do not show evidence of central obesity (Personal observation). Recommendations in terms of the use of appropriate cut-off points, physical activity and emphasis on dietary modification might be able to harness the clustering effect of insulin resistance.

6.2.3 Association of physical activity with prevalence of risk markers

The third objective formulated for this study was: "to establish whether an association exists between the current trends in physical activity and the prevalence of risk markers for NCDs".

From objective one, it was highlighted that there was a high prevalence of physical inactivity especially amongst women. In the attempt to determine the association of physical activity and the prevalence of risk markers, correlations were weak and not statistically significant (Table 5.30). However, the observed frequencies for the low physical activity category in Table 5.31 showed high frequencies for risk of BP, WC and triglycerides. On the other hand, the observed frequencies between the low physical activity category and the European SCORE model indicated the most frequencies in <1% risk for CAD. In comparison, the Risk score for the low physical activity category indicated higher frequencies for Risk 3, 4 and 5. It has to be noted, however, that physical activity is not a determinant in any of the risk score algorithms calculated in the study.

6.2.4 Strategies for an integrated programme of prevention

The fourth objective formulated for this study was: "to suggest strategies for an integrated programme of prevention of NCDs in the target group".

Strategies for an integrated programme of prevention will be based in Chapter 7 on the findings of objectives one to three and recommendations for interventions as discussed in section 2.3 in order to suggest lifestyle interventions to suit the target group. However, attitudes, knowledge and barriers to lifestyle changes first need to be identified before robust and sustainable interventions can be recommended. Strategies for an integrated programme of prevention will be primarily based on behavioural change (diet modification and physical activity) using the most appropriate mechanisms and channels to disseminate information and mobilise intervention.

6.3 CONCLUSION

In this chapter, the results of the study were discussed in relation to the objectives, starting with the prevalence of risk markers and then determining associations between risk markers and lifestyle behaviours in order to develop appropriate strategies for an integrated programme of prevention. Chapter 7 follows with the recommendations and conclusions on the salient observations of this study.

CHAPTER 7

RECOMMENDATIONS AND CONCLUSIONS

7.1 INTRODUCTION

The results of the study were discussed in the previous chapter in relationship to the objectives of this study. KwaZulu-Natal was the base for Indian migrants into South Africa and with time and the change of laws in South Africa, migrant Indians ventured into other regions of South Africa. Despite the regional influences in terms of the availability of ingredients and acculturation, the habitual diet of Indians in South Africa remained fairly homogenous. The reason for selecting KwaDukuza for this study was based on the fact that it embodies the characteristics of a typical Indian community and hence is representative of Indians in KZN. With the habitual diet of Indians in South Africa remaining homogenous, appropriate and practical recommendations proposed in this study in order to improve the quality of life amongst Indians in KwaDukuza can be used for all Indians in South Africa.

In order to propose strategies for an integrated programme of prevention of NCDs, it is important to view current national and international recommendations as discussed in section 2.7. Moreover, in order to ensure that the strategies are based on ethnic specificity, interventions and recommendations from the South Asian Diaspora and from the India sub-continent are valuable. Finally, in translating the findings of this study into appropriate recommendations, effectiveness of the recommendations for intervention would be measured by the communication model adopted as discussed in section 2.7.

7.2 INTERNATIONAL AND NATIONAL RECOMMENDATIONS FOR INTERVENTION

Asian Indians around the world have the highest rates of premature coronary artery disease. There have been a number of national and international recommendations for interventions through risk factor modification to prevent and decrease clinical manifestation of NCDs, as discussed in section 2.7. The main commonalities in these

recommendations are based on the premise of diet and lifestyle modification. Recently, Mayosi *et al.* (2009) provided a panel overview of South Africa's response to the burden of NCDs and recommended the identification of priorities for the next 15 years so as to relieve the pressure of the growing prevalence of NCDs on the health care system. Priority interventions at population level recommended by these authors to the national Department of Health in South Africa include maintaining and extending tobacco-control activities; monitoring, assessing and enforcing anti-alcohol legislation; food control legislation with public education for reducing the salt content of food and for substituting 2% of trans-fat with PUFAs; promotion of physical activity in schools and workplaces; strengthening of the district-based primary health system with integration of the care of NCDs and their risk factors and the development of a national surveillance system for all chronic diseases.

However, national guidelines are usually based on the general population of a country and ethnic populations are often under-represented in the formulation of these guidelines. Consequently, individuals from ethnic groups may be inadequately targeted for risk-reduction strategies, including screening for and treatment of NCDs. Lip *et al.* (2007) recognised a similar phenomenon in the UK with regard to the South Asians and compiled a set of specific guidelines for primary and secondary prevention of CVD.

Prevention is usually classified as **primordial, primary** and **secondary prevention** (Enas *et al.,* 2008). **Primordial** prevention aims to halt the growth of risk factors through healthy lifestyle changes. The application of primordial prevention on the whole population becomes a population-based strategy which creates a new generation in which low risk is the rule and high risk is the exception hence creating a new generation with low risk factor levels. **Primary** prevention aims to identify individuals with high risk factors who have not yet suffered a coronary event and are targeted for maximum lifestyle changes. **Secondary** prevention is aimed at reducing the mortality and morbidity among those diagnosed with CAD.

There are a number of successful interventions in India such as those reported by Balagopal *et al.* (2008); Ramachandran *et al.* (2006); Somannavar *et al.* (2008). Misra *et al.* (2009) recently reviewed South Asian diets and suggested changes in diet and lifestyle to prevent cardiovascular risk factors and the metabolic syndrome. They also

stressed the urgency of nationwide community intervention programmes aimed at creating awareness about the consequences of unhealthy food choices in India, other countries in South Asia and in migrant South Asians. Bearing in mind the results of the current study in KwaDukuza, it is therefore recommended that an Indian healthcare summit, similar to the INDO-US Healthcare Summit (Enas *et al.,* 2008) be initiated by the South African Department of Health as a population-based strategy. Preventive measures rest mainly on public education, media, legislation and government policy. This will have to involve effective partnerships among government, non-governmental organizations and civil society. Compliance with preventive measures is achievable only if the message is given repeatedly, consistently and unambiguously.

7.3 PRIMARY PREVENTION IN INDIANS IN KZN

Primary prevention aims to identify individuals with markedly elevated risk factors (hypertension, high cholesterol, fasting glucose and triglycerides and abdominal obesity) who have not yet suffered a coronary event and are targeted for maximum lifestyle changes. From the study on "healthy" Indians in KwaDukuza, it is clear that primary prevention is urgently needed; starting with a screening programme in places such as community halls, religious gathering-places of worship, workplaces, schools, popular shopping malls, pharmacies and general practitioner's consulting rooms. Most respondents in the study did not attend primary health clinics but private medical facilities where the responsibility of primary care is undertaken. There should be stricter treatment goals and lower thresholds for intervention than what is recommended for western populations (Enas *et al.,* 2008). Current risk prediction models are neither sensitive nor specific to recommend their widespread application in the South African Indian population. Bang *et al.* (2009) recently developed and validated an easy-to-implement diabetes screening score, which may be useful for self-assessment by Indians in KZN.

The risk markers that predict NCDs have now been identified. Specific details of an intervention can only be made after attitudes, knowledge and barriers to changes in diet and activity has been determined. Farooqi *et al.* (2000) conducted a study to this effect where the knowledge of and attitudes to lifestyle risk factors for CHD amongst South Asians in the UK was gauged. The results of the study showed that respondents

expressed a range of attitudes to and different levels of knowledge of lifestyle risk factors for CHD. Barriers to improving lifestyle with respect to diet and exercise included lack of information and cultural barriers. This study highlights the need for health promotion advice to be tailored, in particular the need for cultural sensitive advice.

Recommendations from the main findings of this study, in line with current national and international strategies and recommendations for an integrated programme of control and prevention of NCDs amongst Indians in South Africa, will now be summarised.

7.4 APPROPRIATE WEIGHT MANAGEMENT

The findings of this study showed that respondents had a high prevalence of central obesity, associated with all the others risk markers that were measured. However, the continuous rise in obesity rates in many countries suggests that success rates of current programs to combat obesity are questionable. According to Hill (2009), the poor long-term success rate in treating established obesity through lifestyle changes could be due to the large permanent change in diet and physical activity to keep weight off. Therefore, he suggests that the *small changes approach* is ideal to reduce obesity levels as it is more feasible to achieve and to maintain than larger changes. Small energy losses of up to 420 kJ per day in the form of a prudent diet and increased physical activity may benefit the Indian community in decreasing the prevalence of central obesity. According to Goldstein (1992) weight reduction has been shown to improve glycaemic control in obese patients with diabetes, reduce blood pressure in obese patients with hypertension and improved lipid levels in obese patients with dyslipidaemia. Energy restriction and/or weight reduction may result in a significant reduction in all the risk markers measured in this study including fasting blood glucose (Wing *et al.*, 1994), abdominal visceral fat area (waist circumference), total cholesterol and triglycerides (Ohkawara *et al.*, 2010) as well as blood pressure (Neter *et al.*, 2003).

Increasing knowledge with regard to the phenomenon of increased waist circumference is important as currently many Indians are unaware as to the actual cause and consequences of increased waist circumference and in the case of women, they often relate the development of increased waist circumference after giving birth (personal observation).

Standard definitions underrepresent the prevalence of NCDs in Asian Indian communities thereby delaying the commencement of definite preventive and therapeutic treatment. The dissemination of ethnic-specific cut-points by the Department of Health to the general public and the health profession would assist in identifying individuals at risk of NCDs.

7.5 PROMOTION OF REGULAR PHYSICAL ACTIVITY

7.5.1 Introduction

Physical activity is the key determinant of energy expenditure and thus is fundamental to energy balance. According to the WHO (2003b) at least 30 minutes of moderate-intensity physical activity five days a week will reduce the risk of several NCDs in adults such as cardiovascular disease, stroke, type 2 diabetes, colon cancer and breast cancer. There is a large amount of evidence amassed on the benefits of increasing physical activity on health such as the lowering of BP, control and maintenance of body weight, decreasing serum triglyceride concentrations, improving tissue sensitivity to insulin and control over risky behaviours (WHO, 2003b; Morris *et al.*, 1966; Bouchard *et al.*, 2001).

7.5.2 Physical activity in the workplace

The results of the study are amplified by the sedentary habits displayed by respondents where a high level of physical inactivity was noted. A large percentage of respondents (78%) were formally employed, hence, the workplace would be an ideal channel to improve physical activity as a large section of an adult population can be captured for intervention. Sorensen *et al.* (1996) cited a number of reasons in support of workplace interventions. Firstly, a large percentage of adults are employed in the formal sector and therefore, are a captured target group. Secondly, interventions at the workplace can be offered repeatedly which increases the chances of changing behaviour. Thirdly, the workplace is in a position to support individual behaviour change attempts by modifying the social and physical environment and finally the workplace provides

access to a large number of adults who may be difficult to reach through other intervention channels.

Kolbe-Alexander *et al.* (2008) conducted a study to determine the health and risk profile of South African employees and to measure their readiness to change and improve their behaviour through a wellness day. The findings of this study showed that employees' health and lifestyle habits were placing them at increased risk for NCDs. This study underscores the importance of determining the health and risk status of employees which could assist in identifying the appropriate interventions to reduce the risk of NCDs amongst employees. In a recent review on best practice workplace interventions by Steyn *et al.* (2009), it was highlighted that nutrition and physical activity intervention is the best way to improve employee health and behaviour. The review also highlights the key success factors for successful changes as well as the barriers to changes. Most importantly, the review provides an overview of results of studies which had best practice outcomes in the following categories: clinical outcomes such as serum lipids; behavioural changes such as dietary changes and knowledge and attitude changes. Two studies of particular interest were the study by Cook *et al.* (2001) and the study by Sorensen *et al.* (1999), which will be briefly discussed.

The Cook *et al.* (2001) study with an intervention period of six months was conducted at two manufacturing plants in New Zealand to reduce NCDs. After the intervention, fat intake and SBP were reduced, vegetable intake and physical activity were increased and nutrition knowledge improved compared with the control site. However, no difference was found in WC between the intervention and control group. Hence, it was suggested that the intervention should last longer. The Treatwell 5-a-Day study by Sorensen *et al.* (1999) is an excellent example of a worksite study which focussed on family participation with the primary goal aimed at increasing fruit and vegetable intake and decreasing fat intake. It was conducted at community health centres and was largely managed and supported by staff and had three components namely: the control group, workplace only and workplace plus family. Total fruit and vegetable intake increased significantly in the intervention compared with the control group. The authors concluded that the Treatwell 5-a-Day intervention model has the potential to enhance worksite-based intervention through incorporation of its family focus, especially given

the association of household support with individual eating habits. However, they also identified the barriers to involving families in worksite interventions.

Steyn et al. (2009) concluded in their review that numerous workplace interventions have shown significant improvement in employee's health and behaviours. However interventions have to be planned on existing evidence of best practice. Therefore, it is recommended that places of employment are an ideal channel to improve dietary habits as well as physical activity.

7.5.3 Physical activity for housewives

Twenty-one percent of the respondents in the current study were housewives. In order to improve the physical activity level of housewives it is recommended that walking routes and health clubs are established, using community halls. This effort can be successful if safe walking routes with road markings are identified and these walking routes published in the community paper.

Furthermore, the culture of physical activity is very limited in the current national education system. Therefore, in order to bridge this gap of physical inactivity amongst the young, schools and the community could engage in a memorandum of agreement where learners are allowed to use school sporting facilities after hours under the mentorship of community members. Reasons for lower levels of physical activity amongst South Asians in the UK have been explored in the Health Education Authority Second Health and Lifestyle Survey of black and minority ethnic groups (BMEGs) and it was found that in the UK, 60% of ethnic minority women reported ethnically specific reasons for non-participation in recreational physical activity, such as unwillingness to attend same-gender facilities (Johnson *et al.,* 2000). Therefore, in order avoid the similar situation, separate health clubs for men and women should be promoted to make women feel more comfortable to engage in physical activity. In many Indian communities in South Africa *Bhangra,* which is a form of Indian dance, has become very popular during festivities such as prayers and weddings. This should be promoted as a form of dancercise to encourage physical activity for the whole