Obesity, undernutrition and the double burden of disease in the Free State

Reinette Tydeman-Edwards

Dissertation submitted in fulfilment of the requirements for the degree

Magister Scientiae Dietetics

In the Faculty Health Sciences, Department of Nutrition and Dietetics, University of the Free State

Supervisor: Prof. CM Walsh

Bloemfontein **July 2012**

Declaration of Independent work

I certify that the dissertation hereby submitted by me for the M. Sc. Dietetics degree at the University of the Free State is my independent effort and had not previously been submitted for a degree at another university/faculty. I further more waive copyright of the dissertation in favour of the University of the Free State.

Reinette Tydeman-Edwards July 2012

Table of contents

Acknowledgements

Thank you to the following for making this study possible:

- Professor Corinna Walsh, Department of Nutrition and Dietetics, University of the Free State – for her support, guidance, patience and countless late nights.
- Mr Cornel van Rooyen and colleagues, Department of Biostatistics, University of the Free State – for their valuable input regarding the statistical analysis of the data.
- The National Research Foundation – for their financial support to execute this study.
- Dietetics students from the Department of Nutrition and Dietetics, University of the Free State – for their assistance in gathering the data.
- The study respondents – for taking part in the study.
- My husband, Gavin - for his love, moral support and encouragement.
- My son, Wayne – for just being there.
- My family and friends.
- The Lord, my strength and my salvation.

~ Dedicated to Gavin and Wayne ~

Index of Tables *Page*

Chapter 2

Chapter 3

Index of Tables (continued) *Page*

Chapter 4

Index of Tables (continued) *Page*

Anthropometry

Children

Index of Tables (continued) *Page*

Index of Figures ***Page***

Chapter 1

Chapter 2

List of Acronyms and Abbreviations

ADA	American Dietetics Association
AHA FS	Assuring Health for All in the Free State
AICR	American Institute for Cancer Research
AIDS	Acquired Immune Deficiency Syndrome
BFHI	Breastfeeding Hospital Initiative
BMI	Body mass index
BRISK	Black Risk Factor study
CDLs	Chronic diseases of lifestyle
CNPP	Center for Nutrition Policy and Promotion
CHD	Coronary heart disease
CHO	Carbohydrates
CI	Confidence Interval
COPD	Chronic obstructive pulmonary disease
CORIS	Coronary Risk Factor Study
CRC	Colorectal cancer
CRISIC	Coronary Risk Factor Study
CVD	Cardiovascular disease
DALYs	Disability adjusted life years
DHA	Docosahexanoic acid
DM	Diabetes mellitus
DNA	Deoxyribonucleic-acid
DoH	Department of Health
DRIs	Daily recommended intakes
EPA	Eicosapentanoic acid
FAO	Food and Agriculture Organization
FFQ	Food frequency questionnaire
FSRDPP	Free State Rural Development Partnership Programme
FYFS	First Year Female Students Project
GDP	Gross domestic product
GI	Glycemic index

List of Acronyms and Abbreviations (continued)

GL	Glycemic load
HDL	High-density lipoprotein
HIV	Human immunodeficiency virus
IHD	Ischemic heart disease
KH	Knee height
LBW	Low birth weight
LDL	Low-density lipoprotein
MAC	Mid-arm circumference
MI	Myocardial infarction
MRC Technical Report	Medical Research Council Technical Report
MTHFR	Methylenetetrahydrofolate reductase
MUCPP	Mangaung University Community Partnership Programme
MUFAs	Mono-unsaturated fatty acids
MGRS	Multicentre Growth Reference Study
NFCS	National Food Consumption Survey
PUFAs	Poly-unsaturated fatty acids
QoL	Quality of life
RUTF	Ready-to-use therapeutic food
S	Stature
SADHS	South African Demographic and Healthy Survey
SAFBDG	South African Food-based Dietary Guidelines
SASOM	South African Society of Obesity and Metabolism
SAVACG	The South African Vitamin A Consultative Group
SCF	Save the Children Fund
SCEC	Squamous cell esophageal carcinoma
SD	Standard deviations
SEMDSA	The Society for Endocrinology, Metabolism and Diabetes of South Africa
SFA	Saturated fatty acid
SNP	Single nucleotide polymorphism
TB	Tuberculosis

List of Acronyms and Abbreviations

THUSA	Transition, Health and Urbanisation in South Africa
THUSA BANA	Transition and Health during Urbanisation of South African children
UK	United Kingdom
UNICEF	United Nations Children's Fund
USA	United States of America
USDA	United States Department of Agriculture
USDHHS	Unites States Department of Health and Human Services
USSR	Union of Soviet Socialist Republics
VIGHOR	Vanderbijl Park Information Project on Health, Obesity and Risk Factor
W	Weight
WCRF	World Cancer Research Fund
WDF	World Diabetes Foundation
WHO	World Health Organization
WHR	Waist-to-hip ratio
WRFS	Weight and Risk Factor Study
YLD	Years lived with a disability
YLL	Years of life lost

Index of Appendices *Page*

Chapter 1 - Problem Statement

1.1. Motivation for the study

In this chapter an overview will be given of socio-economic and health challenges that may affect or be affected by the nutrition transition. The resultant double burden of disease, which includes both undernutrition and chronic diseases of lifestyle (CDLs), will be highlighted. In addition, the main aim and objectives for the current study will be defined and a brief outline of the dissertation given.

1.1.1. Socio-economic challenges

South Africa is a middle income country characterized by a variety of living conditions, from wealthy and middle income suburbs to deprived peri-urban areas, rural farms and under-developed rural areas (Steyn et al., 2006:6; Department of Health (DoH) et al., 2002:2). Increasing urbanization and changes in diet and health behaviours are occurring as a result of changing social, political and economic factors. Limited resources contribute to a high level of poverty in South Africa, with increasing numbers of informal settlements around cities and towns (Bourne et al., 2002:157; DoH et al., 2002:15). The Medical Research Council Technical Report on dietary changes and the health transition in South Africa (hereinafter referred to as the "MRC Technical Report") states that forty to fifty percent of South Africans are categorized as poor and 25% of these are ultra-poor (Steyn et al., 2006:11). Poverty is reported to be the highest in rural areas (Labadarios et al., 2005:534).

Almost a third of South African households live in informal and traditional settlements (Steyn et al., 2006:11). According to the South African Demographic and Health Survey (SADHS) conducted in 1998, 51% of all homes had their main walls plastered. Amongst shack settlements in urban areas, 16% had plastic, cardboard or corrugated iron walls. In rural areas, most homes had mud and plaster walls (DoH et al., 2002:15).

In 1998, about 39% of South Africans had piped water and 46% had their own flush or chemical toilet inside their homes. In urban areas, the main fuel used for cooking food was electricity, whereas in rural areas it was either wood or paraffin. Only 37% of rural households had an electricity supply as opposed to 84% of urban households (Steyn et al., 2006:11-12; DoH et al., 2002:16).

1.1.2. Health challenges

A quadruple burden of disease exists in South Africa, which consists of: a combination of poverty-related infectious diseases; life-style related non-communicable diseases (i.e. CDLs); human immunodeficiency virus (HIV) and/or Acquired Immune Deficiency Syndrome (AIDS); and injuries due to violence-related trauma (Steyn et al., 2006:6; Bradshaw et al., 2003:v; Bourne et al., 2002:157). After HIV/AIDS (29,8%), cardiovascular disease (CVD) (16,6%) and cancer (7,5%) were some of the leading causes of death for South Africans in 2000 (Bradshaw et al., 2003:v). In 2001, about sixty percent of deaths world-wide were attributed to CDLs and they contributed to 47% of the total burden of disease (Steyn et al., 2006:6).

In 1998, the United Nations Children's Fund (UNICEF) stated that for children younger than five years, half of the world's deaths occurred in Africa. In their opinion, Africa remains the most difficult place in the

world for a child younger than five to survive (UNICEF, 2008:22). Child mortality rates are higher in households where there are poor living conditions (i.e. no piped water, no flush toilet and/or no electricity) (DoH et al., 2002:105). According to the 1998 SADHS, mortality is significantly higher among children in rural areas in South Africa, as well as in Africa as a whole. Mortality rates were also found to be four times higher for black children younger than five years as opposed to their white counterparts (DoH et al., 2002:101). The mortality rate for black infants was 8,6% of live births in urban areas and 9,4% in rural areas. The mortality rate for urban children younger than five years of age was 12,5% and for rural children 13,9% (Labadarios et al., 2005:534). A decline in infant mortality rates was seen between 1970 and 1983 with a decline of 33% for white children, 64% for coloured children and 53% for black children (Yach et al., 1991:214). Bradshaw et al. (2003:11, Table 2.1) report estimated mortality rates for the year 2000 (using ASSA2000 model of the Actuarial Society of South Africa) at 98 per 1000 live births for boys under five years old and 91 per 1000 live births for girls under five years old.

Poor living conditions contribute to a high prevalence of infectious diseases, such as measles and tuberculosis (TB) (Steyn et al., 2006:34). TB remains the most commonly reported notifiable disease in South Africa. It was estimated that there were 127 798 cases of TB among persons older than 15 years in 1998 (DoH et al., 2002:176). Diarrhea can also be linked to poor living conditions. In the 1998 SADHS, there was a very high prevalence of diarrhea among children six to 23 months of age (23%). This finding was consistent with age-specific diarrhea morbidity patterns in other developing countries. The lower prevalence rate among children younger than six months (11%) may reflect the protective effect of breastfeeding. The total diarrhea prevalence rate was highest in black children (14%) (DoH et al., 2002:124). In addition to these, a high proportion of child deaths in South Africa are due to HIV/AIDS (35,1%) (Nannan et al., 2007:737).

In addition to the high burden of infectious diseases in South Africa, CDLs such as: obesity; diabetes; and CVD (including hypertension and stroke); as well as lung-, esophageal-, breast- and colorectal cancers, are also increasing. CDLs were previously limited to higher income groups, but this is no longer the case (Steyn et al., 2006:6). In the last three decades, CDLs have become prominent causes of morbidity and mortality, particularly in the black communities. In 2000, CDLs accounted for forty percent of deaths in females, and 36% of deaths in males in South Africa (37% cause of death for both sexes combined) (Bradshaw et al., 2003:iii, Table 2; Steyn et al., 2006:5, 12), with stroke being the most common fatal CDL for women and ischemic heart disease (IHD) for men (Steyn et al., 2006:5, 12). According to the 1998 SADHS, hypertension, IHD, diabetes mellitus (DM) and cancer were all reported more in urban than rural areas of South Africa (DoH et al., 2002:168). It was found that rural blacks had a significantly lower risk for hypertension than urban blacks (Steyn et al., 2008:378). Norman et al. (2007a:692) also state that urbanization among black South Africans predisposes them to hypertension. In 2000, it was estimated that hypertension caused nine percent of all deaths in South Africa, and contributed to 2,4% of all disability adjusted life years (DALYs). Murray and Lopez (1997a:1436) define the DALY as “the sum of life years lost due to premature mortality and years lived with disability adjusted for severity.” Hypertension contributes to fifty percent of stroke cases, 42% of IHD, 72% of hypertensive disease and 22% of other CVD burden in both adult male and female South Africans older than thirty years (Norman et al., 2007a:692; 695, Table III).

The only published data representative of CDL in a Free State community dates from 1995 (Mollentze et al., 1995). Mollentze et al. (1995:90) showed that 29% of the rural (QwaQwa) black population and 30,3% of the urban (Mangaung) black population were hypertensive. Diabetes was present in 4,8% of the rural and six percent of the urban sample. Hypercholesterolemia (high-risk) was present in 12,5% of rural men and six percent of urban men between 25 and 34 years of age. For moderate risk hypercholesterolemia, the figures were 34% and 44,8% respectively. The mean body-mass-index (BMI) for both rural and urban women exceeded 25 kg/m^2.

Because South Africa is a developing country with limited resources, it is of the utmost importance to limit the burden of disease. The direct costs of CDLs to South Africa is estimated to be as high as 6,8% of health care costs. Indirect costs are also involved, which include work days lost, doctors' visits, impaired quality of life (QoL) and premature mortality (Steyn et al., 2006:21). These are discussed in more detail later in this dissertation.

In 2000, HIV/AIDS accounted for 39% of all deaths in South Africa, while CDLs accounted for 38% of deaths (Seedat, 2007:318). When looking at actuarial models of projection of AIDS- and CDL mortality for 2010, the contribution of CDLs to the burden of disease in South Africa cannot be ignored, despite increasing rates of HIV and AIDS. It is projected that in 2025, one in ten South Africans will be sixty years or older. This may also increase the burden disease attributable to CDL (Steyn, 2005b:249).

1.1.3. The double burden of disease

Two types of malnutrition can be distinguished, namely undernutrition (resulting in underweight, wasting or stunting) and overnutrition (resulting in either overweight or obesity).

It is estimated that about 32% of children in Africa are undernourished (De Villiers & Senekal, 2002:1231). The South African Vitamin A Consultative Group (SAVACG) nationwide survey undertaken in 1994 found that undernutrition was a serious health problem for children younger than six years. The Free State province presented with the second highest percentage of stunted children in the country, together with the Eastern Cape province (De Villiers & Senekal, 2002:1231-1232).

The SAVACG and the NFCS surveys reported that 6,9% to 10,7% of children were underweight (weight-for-age below minus two standard deviations (<2SD) from the reference median), 16,1% to 27% were stunted (height-for-age <2SD), and 1,8% to 3,7% were wasted (weight-for-height <2SD) (Steyn et al., 2006:19). The prevalence of undernutrition was usually higher in rural areas in comparison with urban areas (Steyn et al., 2006:20). By using data from four nationwide surveys, including the Living Standards Measurements Survey conducted in 1994 in South Africa, Popkin et al. (1996:3012) concluded that 30,6% of South African black- and coloured children aged between 36 and 91 months were stunted. The NFCS found that nearly twenty percent of children aged between one and nine years old were stunted, and 17% were overweight (Labadarios et al., 2005:536).

Stunted children have a higher risk of being overweight or obese, either in childhood and/or adulthood (Popkin et al., 1996:3012), which raises the risk of developing CDLs in later life (Steyn et al., 2006:20; Mendez et al., 2005:720).

The 1998 the SADHS reported that 29% of men and 56% of women in South Africa were overweight (BMI between ≥25 kg/m² and <30 kg/m^2) (Steyn, 2005a:43, 45; DoH et al., 2002:244). Almost a tenth of South African men and a third of South African women were severely overweight or obese (BMI ≥ 30 kg/m²) (Steyn, 2005a:45; DoH et al., 2002:244). According to the MRC Technical Report, overweight/obesity was more prevalent in urban areas, which may be indicative of the nutrition transition (Steyn et al., 2006:20). In 1998, seven percent of all men included in the SADHS and 32% of all women had a waist-to-hip ratio (WHR) above the reference cut-off point for increased risk for chronic disease, with a predominantly higher percentage of women with a high WHR living in urban areas (DoH et al., 2002:245).

Abdominal obesity (WHR ≥ 1,0 in men; ≥ 0,85 in women) (Gibson, 2005:281) is associated with increased risk of insulin resistance, diabetes, hypertension, dyslipidemia and atherosclerosis (Goedecke et al., 2005:68). The 1998 SADHS showed that about a third (35,2%) of black South African women and 6,9% of black South African men had a high waist circumference that placed them at risk (Bourne et al., 2002:160). Among all populations groups in the 1998 SADHS, abdominal obesity was present in 42,2% of women and 9,2% of men; and was most common in black urban women and white urban men (Goedecke et al., 2005:65-66). Data from the 1998 SADHS showed that obesity appeared to start in women at a younger age, since ten percent of South African women were obese at age 15 to 24 years old (Goedecke et al., 2005:66). Steyn et al. (2006:5) stated that the presence of obesity (and sedentary lifestyle) contributed significantly to the increased prevalence of CDLs, as did the high prevalence of tobacco- and alcohol use.

A high prevalence of overweight and obesity amongst caregivers was also found in the same household as underweight or stunted children (Goedecke et al., 2005:71; Sawaya et al., 2003:170; Faber et al., 2001:410). In the rural Limpopo province, 31% of underweight children were found to have an overweight mother or caregiver, and in the rural North West Province, nearly fifty percent of mothers and/or caregivers of stunted and underweight children were found to be overweight. This occurrence is also found in other developing countries, such as Brazil, China, and Russia (Steyn et al., 2005:10). In their 1958 British cohort study, Li et al. (2004:185) also found that stunting in early life was associated with short adult stature. Childhood nutritional stunting is associated with long-term impairment of fat oxidation, a factor which strongly predicts obesity (Sawaya et al., 2003:172). Stunted Brazilian girls had significantly lower total energy expenditure compared to boys, which may help explain the particular high risk of obesity in stunted adolescent girls and women (Sawaya et al., 2003:172-173). Stunting is thus associated with risk of obesity and abdominal fatness in women (Sawaya et al., 2003:171).

A review conducted by Sawaya et al. (2003:171), comparing data of studies conducted in Brazil, Russia, China and South Africa, states that epidemiologic evidence supports the association between childhood undernutrition (also called “nutritional stunting” in the review) and adult obesity, and therefore related CDLs (Sawaya et al., 2003:171; Popkin et al., 1996). They postulate that during catch-up growth in infants and children recovering from undernutrition, there is a disproportionately greater replenishment of body fat stores as opposed to body protein stores. Catch-up growth can be defined as an increase in growth velocity in height and/or weight when some constraints on normal growth have been removed (Cameron, 2003:39). The combination of low birth weight (LBW) and small size during infancy, followed by accelerated weight gain from age three to 11 years, is predictive of hypertension, coronary heart disease (CHD) and type 2 DM (Bihl, 2003:757). Sawaya et al. (2003:171) reports an association between

short stature and increased risk for hypertension. The authors reported that stunted individuals had higher triglycerides, low-density lipoprotein (LDL) cholesterol and higher total cholesterol than non-stunted adults (Sawaya et al., 2003:171).

According to the UNICEF conceptual framework, one of the immediate causes of malnutrition (seen here as undernutrition) is inadequate dietary intake (Nannan et al., 2007:733; Schrimpton & Kachondham, 2003:5). In 1998, about 87% of South African babies were breastfed for at least some time. Only seven percent of babies younger than six months were, however, exclusively breastfed in 1998 (DoH et al., 2002:132). As expected, the prevalence of exclusive breastfeeding decreased as the child got older. About 16% of babies younger than two months were exclusively breastfed, whereas only 0,3% were breastfed by age six to seven months. Seventy percent of babies younger than six months received complementary feeds and 17% were not breastfed at all (DoH et al., 2002:134). About one-third of breastfed babies younger than two months also received infant formula, and just over half received other fluids. About 28% of all infants younger than five months received other foods. Nine percent of these children received meat, fish and eggs (DoH et al., 2002:138). When median duration of breastfeeding was investigated, rural children were breastfed longer than their urban counterparts, with black mothers breastfeeding their children the longest (DoH et al., 2002:135).

Inadequate nutritional intake, which leads to undernutrition, has short- and long term consequences for both adults and children (De Villiers & Senekal, 2002:1232). UNICEF expects that the rise in global food prices, especially in basic foods like vegetables, oils, grains, dairy products and rice, may increase the vulnerability of millions to hunger and undernutrition (UNICEF, 2008:24). Nutritional deficiencies related to undernutrition account for 1,2% of deaths in South Africa.

1.1.4. Implications of undernutrition

Undernutrition influences child development significantly, even before birth. Undernutrition also influences motor development, cognitive function and school performance, which can play a role in work capacity and reproductive health in adulthood (Victora et al., 2008:343, 345; Nannan et al., 2007:733). Decreased work capacity in adults can lead to reduced earning capacity and to a cycle of poverty and hunger (De Villiers & Senekal, 2002:1232), as also illustrated in Figure 1.1 The poor physical and mental development of an undernourished child, together with the loss of individual achievement and poor quality of life, play a significant role in social and economic development at the national level (Witten et al., 2002:online [unpublished]).

One of the most important implications of undernutrition in childhood is an increased risk of overweight and obesity in adulthood, as discussed in detail in the previous section on double burden of disease. When Popkin et al. (1996:3012) assessed the effects of previous stunting on present overweight status by examining risk ratio, they found a risk ratio of 2,6 for all races of South African children. There is an odds ratio of 1,8 when looking at the increased risk for overweight if also stunted (Goedecke et al., 2005:71; Labadarios et al., 2005:536; Steyn, 2005a:44). In many developing countries, increasing prevalence of adult obesity has been found to coincide with high prevalence of childhood undernutrition.

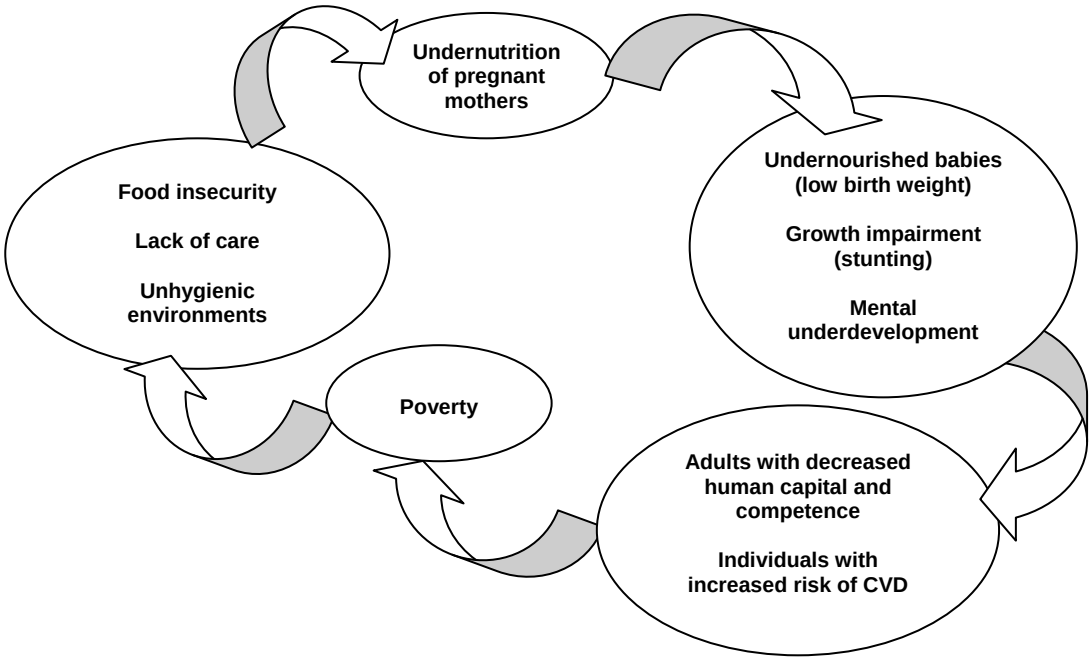


Figure 1.1. The vicious, inter-generational cycle of undernutrition and poverty (Vorster & Kruger, 2007:322, Figure 1).

Diabetes contributes significantly to the burden of disease in South Africa (Steyn, 2005a:43). According to Sawaya et al. (2003:171) catch-up growth is a risk factor for insulin resistance. Children born from women with high BMIs during pregnancy had more rapid growth during childhood, with an increased incidence of type 2 DM as adults (Forsen et al., 2000:176, 180).

Undernutrition in utero, low birth weight and poor maternal nutrition has also been associated with increased risk for osteoporosis, lung disease, immune dysfunction and mental disease (Victora et al., 2008:340).

1.1.5. The nutrition transition

The nutrition transition is identified as the progression from a traditional diet (low fat, high fibre diet) to a Western diet (high fat, low fibre, high energy diet, with habitual intake of fast foods) (Cameron, 2003:37; Bourne et al., 2002:157; MacIntyre et al., 2002:253). This transition is also usually accompanied by a behavioural transition towards a less active/more sedentary lifestyle (Cameron, 2003:73). Globally, traditional diets (largely plant-based) have been replaced by high-fat, energy-dense diets with a substantial content of animal-based foods (World Health Organization and Food and Agriculture Organization (WHO/FAO), 2002:6). Traditional diets consist of >60% to 65% of total energy from carbohydrates, and <25% of total energy from fat, whereas the Western diet consists of <50 to 55% of total energy from carbohydrates and >30% to 35% of total energy from fat. Fibre intake is also lower with a Western diet and free sugar intake is high (>10% of total energy) (Joubert et al., 2007:684; Steyn et al., 2006:13,14; Steyn, 2005a:36; Bourne et al., 2002:157,159; WHO/FAO, 2002:19). The nutrition transition plays a key role in increasing the risk for CDLs, and its role in South Africa cannot be ignored.

As part of the Transition, Health and Urbanisation in South Africa (THUSA) study undertaken in the North West Province, MacIntyre et al. (2002:239) reported that the dietary intakes between rural and urban communities showed a shift from the traditional diet to the Western diet associated with CDLs. The MRC

Technical Report states that intake of carbohydrates among black people in transition decreased with increased time of living in the city, while fat intake increased (Steyn et al., 2006:13). The report also states that the black population was the only ethnic group still undergoing transition from a traditional to a Western diet. The other groups (white, Coloured and Indian) already followed the Westernized dietary patterns (Joubert et al., 2007:684; Steyn et al., 2006:5). Steyn et al. (2006:15, Table 2.2.1) and Steyn (2005a:35, Table 4.2a) confirmed that urban blacks consumed more fat and less carbohydrates than their rural counterparts by comparing results from the 1998 SADHS, the Dikgale study (Steyn et al., 2001) and the BRISK study (Steyn et al., 1991). Also, by comparing the results of the Dikgale study (black rural) to the BRISK study (black urban), it was seen that vegetable and legume intake was greater in the Dikgale study, whereas meat intake was nearly double in the BRISK study. Fat intake was also six times greater in the BRISK study (Steyn et al., 2006:15; Steyn, 2005a:39). The THUSA study confirmed a nutrition transition, when comparing the dietary intake of rural and urban adults. In urban areas (as compared to rural areas) fat intake increased from 22,9% to 30,6%, carbohydrate decreased from 67,4% to 57,3%, and protein increased from 11,6% to 13,2% (Steyn, 2005a:43; MacIntyre et al., 2002:243, Table 1). In the North West Province, low intake of fruit, vegetables and milk was reported in all groups studied except for the upper middle class in the urban community. Alcohol consumption was higher in the rural community (MacIntyre et al., 2002:253). Steyn (2005a:36) mentions that the Coronary Risk Factor Study (CORIS study), which was conducted in rural Western Cape, found that fat intake was very high and did not conform to prudent dietary guidelines. Intake of meat, dairy and eggs was very high, which accounted for the high percentage total fat and saturated fat intake. There was also a high sugar intake, but fruit and vegetable intake was also high.

The MRC Technical Report reported that white males had the highest intake of fat (>30% of total energy), protein and added sugar (>10%) and the lowest intake of carbohydrates (<55%) (Steyn et al., 2006:34). In contrast, rural blacks had the highest intake of carbohydrates (>60% of total energy) and the lowest intake of protein, fat (<20%) and added sugar (<10%) (Steyn et al., 2006:35). Black urban males' intake seemed to be between these two extremes. At that time, Steyn et al. (2006:14) suggested that the transition in blacks from a traditional rural diet to an urban diet is approaching the completely Westernized diet of the white and Indian population.

In the past fifty years the intake of carbohydrates has decreased from 69,3% to 61,7% of total energy, with a relative decrease of 10,9%, and the fat intake amongst urban blacks increased from 16,4% to 26,2% of total energy, with a relative increase of 59,7% (Goedecke et al., 2005:71; Bourne et al., 2002:157). Shifts to the Western diet were also found amongst rural black communities. Steyn et al. (2006:13) found the same trends in their urban communities. Carbohydrate intake decreased from 61,4% to 52,8% and fat intake increased from 23,8% to 31,8%. Protein intake remained more or less the same over time, although the contribution from animal protein increased and the amount from plant protein decreased. These changes are all consistent with a population that is undergoing a nutrition transition. It is well known that an increased prevalence of CDLs results in populations following the so-called "Western diet" (Bourne et al., 2002:157). Other risk factors for CDLs, other than physical inactivity and obesity, are low intake of fruits and vegetables and high intake of alcohol (Steyn et al., 2006:6).

Alcohol consumption in South Africa increased from 1962 to 2001. Nearly thirty percent of adult males reported excessive alcohol use, compared with ten percent of females. High alcohol consumption is a risk

factor for CDLs and needs to be addressed in the prevention of CDLs (Steyn et al., 2006:18). The 1998 SADHS (DoH et al., 2002:238) found that 45% of adult men and 17% of women consumed alcohol. For the total population, the consumption rate was 28%. The consumption was slightly higher in urban areas for both sexes. For males, the highest drinking levels were reported in the Free State and Gauteng provinces. For females, the highest drinking levels were reported in the Free State, Western Cape and Northern Cape (DoH et al., 2002:238).

The health of a nation can be monitored by gathering information on the burden of disease. The information should be comprehensive, timely and precise so that practical and implementable health policies can be formulated, which will in turn meet the demand for appropriate health services and interventions. It is imperative to set priorities in a health sector where resources are scarce, to prevent wastage (Bradshaw et al., 2003:1). Investigation of changes in diet and the health transition in South Africa can contribute to the development of national strategy with a strong dietary policy component, which will be effective in the long term (Steyn et al., 2006:6). According to Steyn (2005a:45) active and progressive action is needed by policy makers, to prevent the burden of CDLs in South Africa from increasing in the next few decades. Steyn (2005a:45) also states that policy makers should be reminded of the very important role that diet plays as a determinant of most CDLs. It is impossible to prevent or manage CDLs without managing the dietary aspects. Thus, Steyn (2005a:45) implies that the population has to be educated regarding a healthy diet, being physically active and abstaining from excessive alcohol intake and tobacco use.

1.1.6. Current study

The current study formed part of the baseline of the larger Assuring Health for All in the Free State (AHA FS) Study, which aimed to provide the Free State with a direct estimate of the health/disease burden attributable to established and emerging risk factors for obesity, diabetes, and CVD., as well as infectious disease such as HIV/AIDS, TB and undernutrition, in both rural and urbanized communities. The results related to risk for CDL and the metabolic syndrome in the rural and urban communities included in the study have recently been published (Van Zyl et al., 2012:online). Factors that may have contributed to these health challenges (such as the role of diet), however, need to be determined in order to plan and implement relevant interventions to address the identified health challenges.

Since only a limited amount of studies have ever been conducted in the Free State, it would be prudent to gather information specific to the Free State region to obtain a clearer view of the current situation in the Free States as compared to the national- and African trends. By comparing data obtained from the rural AHA FS study with data obtained in the urban AHA FS study, the extent of the nutrition transition in the Free State can be determined. Findings from the AHA FS study can be used to develop appropriate educational and health promoting programs. The results can also be used to facilitate effective public health policies, which may alleviate the burden of disease for the Free State.

1.2. Aim and objectives

1.2.1. Main aim

The main aim of this study was to determine the diet and anthropometric status of adults (between 25 and 64 years old) and pre-school children (zero to seven years old) in rural and urban areas. Rural areas included Trompsburg, Philippolis and Springfontein. Urban areas included communities located around the Mangaung University Community Partnership Program (MUCPP) Clinic. In addition, this study investigated associations between anthropometric status of children and adults in rural and urban areas in order to determine whether a double burden of disease existed.

1.2.2. Objectives

In order to achieve the main aim, the following were determined in both urban and rural children and adults:

- Dietary food and drink intake; and
- Anthropometry.

1.3. Outline of the dissertation

Chapter 1	Problem statement
Chapter 2	Literature review
Chapter 3	Methodology
Chapter 4	Results
Chapter 5	Discussion of results
Chapter 6	Conclusion and recommendations

Chapter 2 – Literature review

2.1. Introduction

Many developing countries, including South Africa, face both the burden of overnutrition and undernutrition, known as the double burden of disease.

Only one study by Mollentze et al. (1995) has previously reported on the extent of CDLs in the Free State. More recently, the AHA-FS study was undertaken to provide more recent data from both rural and urban areas of the Free State to ascertain the current burden of disease in this province and to determine which factors may have contributed to it.

The costs of CDLs are tremendous, not only in monetary terms, but also in mortality and quality of life; even more so in developing countries such as South Africa (Steyn et al., 2006:21; Goedecke et al., 2005:65; Kruger et al., 2005:492; WHO/FAO, 2002:4).

In this chapter the focus will be on a review of the literature related to the nutrition transition and how it is related to undernutrition on the one hand and to various chronic diseases of lifestyle on the other. The influence of changes in diet will be highlighted.

2.2. The Barker theory

A number of studies have reported on maternal nutritional status and how it is associated with child undernutrition (Victora et al., 2008:340; Schrimpton & Kachondham, 2003:3). Countries with emerging economies; in particular India, China, the Caribbean, and South Africa, have contributed to epidemiological data related to the association of LBW with adult onset CDLs, such as: CVD (including CHD); DM; hypertension; dyslipidemia; stroke; and cancer. In developing countries, where the vast majority of LBW babies are born, a steep rise has been observed in the prevalence of obesity, diabetes and CVD (Levitt et al., 2005:58). There is increasing evidence that increased risk for CDLs begin in fetal life and continues into old age (WHO/FAO, 2002:31).

The Barker hypothesis (also known as the fetal origins theory) states that events occurring before birth may program a person to certain physiological responses, which can then lead to cardiovascular- and metabolic disorders in later life (Levitt et al., 2005:59). Early work by Barker and colleagues linked an adverse intra-uterine environment to the development of CDLs, and specifically found an association between LBW and CVD mortality (Goedecke et al., 2005:70; WHO/FAO, 2002:35). The Barker hypothesis suggests that disturbed intra-uterine growth has a negative influence on the development of the cardiovascular system and favours the occurrence of CDLs and morbidities (Seedat, 2007:318; Vorster & Kruger, 2007:322; Goedecke et al., 2005:70; Levitt et al., 2005:59; WHO/FAO, 2002:8). Small babies are believed to have suffered intra-uterine growth retardation, which affected their overall size and proportion; reducing the size and altering the function of various organs (e.g. the kidneys pancreas and liver) in order to compensate for normal brain growth (Cameron, 2003:39). Barker and his colleagues found that in certain areas in Britain where there were high death rates from CVD, there was also a high prevalence of infant mortality (Barker, 1990:1111). Small size at birth in full-term pregnancies is linked with a subsequent “programming” for the

metabolic syndrome; including: glucose intolerance; increased blood pressure; dyslipidemia; and increased mortality from CVD (Goedecke et al., 2005:71). The degree of elevated blood pressure, glucose intolerance and insulin resistance was greater when LBW was linked with adult obesity. This suggests an interaction between intra-uterine events and later environmental influences (Victora et al., 2008:340; Levitt et al., 2005:59).

LBW, due to slow fetal growth, increases the risk of type 2 diabetes and the metabolic syndrome. Insulin plays an important role during fetal growth and insulin- and glucose metabolism are altered in the fetus during undernutrition. Patients with type 2 DM may present with both insulin resistance and insulin deficiency, which may have been caused by adaptations during the fetal period (Forsen et al., 2000: 176).

2.2.1. Early life influences

Seedat (2007:318) suggests that many CDLs manifesting later in life may be related to two factors in early life, which may seem contradictory, namely: (1) poverty, where malnourished mothers give birth to malnourished LBW infants; and (2) prosperity, where a LBW child is exposed to a high-energy diet, which may lead to obesity. Cameron (2003:39) states that intra-uterine growth retardation can cause a child to adversely respond to a diet high in energy, fat and sodium, resulting in obesity later in life. Popkin et al. (1996:3010) propose two explanations for the link between LBW and subsequent obesity in later life: (1) the effect of undernutrition during pregnancy and infancy; and (2) gestational diabetes and poor diet. It was already suggested in 1976 that metabolic tissues such as the hypothalamus are reprogrammed as a result of early malnutrition during gestation. The setting of the hypothalamus to inappropriately alter appetite control, could possibly lead to obesity (Popkin et al., 1996:3014).

Levitt et al. (2005:58) state that the expression of the intra-uterine programming for obesity depends on early life experiences; maternal nutrition; post-natal nutrition; and timing of catch-up growth. It should, however, be kept in mind that many factors can have an impact on birth weight, including: birth order; gestational age; maternal age; maternal size; weight gain in pregnancy; maternal diabetes; maternal hypertension; maternal smoking; alcohol- and drug use; stress and infection. Care should thus be taken when assuming that LBW is the only factor involved in intra-uterine and fetal undernutrition (Levitt et al., 2005:61).

According to the WHO/FAO (2002:8), approximately 23,8% of thirty million new-born babies per year are affected by intra-uterine growth retardation. A child's growth pattern plays an important role in underlying disease pathways (i.e. restricted fetal growth followed by very rapid postnatal catch-up growth). Intra-uterine programming, together with subsequent early life influences that interact with genetic factors, influences an "adult chronic disease phenotype" (Goedecke et al., 2005:71).

2.2.2. Obesity

In 1990, over 4000 LBW infants born in Soweto and Johannesburg were enrolled in the Birth-to-Ten cohort study, who were then followed up for ten years. A higher risk of obesity, higher body fat and centralized fat patterns were present by five years of age in LBW children who demonstrated catch-up growth. Such risk factors have even been illustrated in South African urban children as young as one year of age (Cameron, 2003:39). Childhood obesity plays a larger role in the development of the metabolic

syndrome, than does adult obesity (Forsen et al., 2000:176). Associations between early life exposures with obesity and the "chronic disease phenotype" have been demonstrated in South African studies with large and representative samples of children, specifically the South African Birth-to-Twenty study that was initiated in 1990 in the metropolitan area of Soweto and Johannesburg. The "chronic disease phenotype" was prominent in LBW children who were above the median for BMI, or with central adiposity when they were older. The association between LBW and increased body fat content, were linked to reduced lean tissue mass.(Levitt et al., 2005:58-59).

The Birth-to-Twenty study found that twenty percent of black- and coloured children presented with catch-up growth, while they were also significantly taller, heavier and fatter throughout their childhood and more likely to be overweight or obese by nine years of age (Cameron, 2003:39; Levitt et al., 2005:59). Barker and his colleagues have shown that adults with LBWs or underweight at one year of age had a greater tendency to store fat abdominally (Popkin et al., 1996:3009).

Apart from the Birth-to-Twenty study, South African data concerning early life "programming" and subsequent obesity is limited. One study conducted by Mamabolo et al. (2005:online) involving 162 rural children followed from birth, from central Limpopo province, showed a high prevalence of stunting (48%), overweight (22%) and obesity (24%) at three years of age. Nineteen percent of the children were both stunted and overweight (Mamabolo et al., 2005:online). Rapid weight gain within the first year of life in children who were underweight at birth increased the risk six-fold for being overweight at three years of age. In the Birth-to-Twenty cohort, weight gain or growth velocity was associated with increased adiposity, measured by skinfold thickness (Goedecke et al., 2005:71; Levitt et al., 2005:59).

The explanation by Hales and Barker (1992:599) for the fetal origins of adult disease was the "thrifty *phenotype* hypothesis", initially associated specifically with type 2 DM (Levitt et al., 2005:61), which states that in developing countries, the progression to obesity and morbidity associated with LBW appears to depend on the interaction between birth weight and subsequent growth during critical developmental periods (Levitt et al., 2005:58). An adverse intra-uterine environment (related to poor fetal nutrition) programmes the fetus' metabolism to use subsequent nutrition sparingly (termed "nutritional thrift"). If this nutritional hardship persists, this physiological adaptation remains appropriate; but should the individual be exposed to improved nutrition, disease would occur due to "physiological maladaptation," e.g. glucose intolerance which may lead to diabetes (Levitt et al., 2005:61).

Alternatively, the theory of the "thrifty *genotype"* also exists, in an attempt to explain the overwhelming rise in diabetes prevalence among the Pima Indians and Nauruans, who have also experienced a nutrition transition. This theory proposes that certain genes persist in a population, since they would ensure survival during stages of famine. However, in stages of abundance these genes are disadvantageous (Levitt et al., 2005:61).

The time at which an insult occurs *in utero* may also influence the relationship between birth weight and subsequent adiposity. Persons who were exposed to the Dutch famine during the Second World War, while *in utero* during the first and second trimesters, were nearly three times more likely to become obese (Goedecke et al., 2005:71; Levitt et al., 2005:58; Popkin et al., 1996:3009).

Sawaya and Roberts (2003:online) raise the question whether the increase in the prevalence of obesity is greater among poor people of transitional societies, since these populations (especially women), have a higher susceptibility to the effects of a Western lifestyle (high intake of animal and processed food, low physical activity, etc.). They debate that one strong candidate for this higher susceptibility seems to be early undernutrition, as it could permanently programme the individual to increase and/or preserve fat stores (Sawaya & Roberts, 2003:online).

On the flip side, when looking at higher birth weight, numerous studies have shown a direct association between higher birth weight and higher BMI (Levitt et al, 2005:58). Large size at birth (macrosomia) is also associated with an increased risk of diabetes and CVD (WHO/FAO, 2002:31). The WHO/FAO (2002:32) state that higher birth weight has also been related to an increased risk of breast and other cancers. However, Victora et al. (2008:350) found no convincing evidence that child's higher body weight predicts cancer in later life, and some trials have even suggested that the opposite is true.

2.2.3. Hypertension

In a Jamaican study, blood pressure levels were found to be highest in 11 to 12 year old children who had retarded fetal growth and greater weight gain between ages seven and 11 years. Similar results were found amongst LBW Indian babies, where poor muscle-, but high fat preservation, was found (the "thin-fat" babies). An increased central adiposity was found in these children, with raised blood pressure (WHO/FAO, 2002:34). If adjustment for current body size (through BMI) is made, the association between LBW and high blood pressure is especially strong (WHO/FAO, 2002:34). The Birth-to-Twenty study found that systolic blood pressure was inversely associated with birth weight, without regard to current weight or height. For every one kilogram increase in birth weight, systolic blood pressure was 3,4 mmHg lower at five years of age. However, for children who fell into the lowest quartile for birth weight (<2 800 g) as well as in the highest quartile for current weight at five years of age, the blood pressure was the highest, which suggested that birth weight's effect on blood pressure may be intensified by events in early childhood and subsequent growth (Levitt et al., 2005:60).

2.2.4. Cardiovascular disease and diabetes mellitus

Van der Merwe and Pepper (2006:4) point out that if a previously stunted individual remains lean and maintains a "non-obesigenic lifestyle," that person can remain "metabolically healthy." The risk for insulin resistance in adulthood increases if there is weight gain in a previously undernourished individual due to food becoming abundant (Van der Merwe & Pepper. 2006:4). Persons with a LBW who became obese adults, have a higher risk for type 2 DM (Forsen et al., 2000:176).

Barker's study (1990:1111) found that men who suffered fetal growth retardation had higher mortality from CVD (related to dyslipidemia), as well as a higher prevalence of hypertension and DM. The WHO/FAO (2002:40) also states that LBW, followed by adult obesity, contributes to a particularly high risk for CHD and DM (WHO/FAO, 2002:40). The risk for impaired glucose tolerance is the highest in obese adults who had a LBW. When intra-uterine growth retardation is followed by rapid catch-up growth in weight and height, there is an increased risk of adult disease (WHO/FAO, 2002:40). A link between short stature and a higher risk of CHD, stroke and adult-onset DM is also probable (WHO/FAO, 2002:40).

Glucose intolerance is influenced by birth weight, weight at one year old, and a high BMI during childhood (Cameron, 2003:40). A study conducted by Forsen et al. (2000;176), in Helsinki, Finland, using data collected between 1964 and 1997, found that type 2 DM is programmed *in utero* in association with low rates of fetal growth. The increased risk for type 2 DM associated with small birth size, further increased when there were higher growth rates after seven years of age (Forsen et al., 2000:176).

The Birth-to-Twenty study, reported that LBW children who were above the median weight-for-age, had increased blood pressure at age five years, and a greater insulin response to oral glucose tolerance testing at age seven (Goedecke et al., 2005:71; Levitt et al., 2005:59; Steyn, 2005b:249). Post-natal and childhood growth patterns modulate the development of insulin resistance (Levitt et al., 2005:60). This confirms the relationship between poor fetal growth (as measured by birth weight), and both glucose intolerance and insulin resistance (Levitt et al., 2005:60). The study found that lower birth weight was linked to greater insulin production and higher glucose concentrations when children fasted for ten to twelve hours and then underwent an oral glucose tolerance test. The results suggested that the ability to clear glucose from the circulation through insulin secretion was reduced in LBW children. Shorter children at one and seven years old also presented with higher glucose concentrations. It has been postulated that poor fetal growth followed by higher post-natal growth leads to low ß-cell counts and higher insulin resistance (Cameron, 2003:40). Small-for-gestational age neonates with the greatest post-natal gain in weight, had the greatest risk of developing insulin resistance. These abnormalities were already evident in the first few week of life (Levitt et al., 2005:61).

Children born to mothers with high BMIs during pregnancy had more rapid growth during childhood, with an increased incidence of type 2 DM (Forsen et al., 2000:176, 180). These children had faster growth rates in height, weight, and BMI between the ages of seven and 15 years (Forsen et al., 2000:178). The offspring of heavier mothers also have higher rates of type 2 DM as adults (Forsen et al., 2000:180) and infants born to women with diabetes are often fatter and larger. Even if they are born with normal birth weights, they still have an increased risk of childhood obesity. High glucose during fetal development increases the risk of insulin resistance, onset of diabetes before the child-bearing years, and continuation into the next generation (Victora et al., 2008:348).

2.2.5 Correlation between undernutrition and overweight

A relationship is suggested between stunting and obesity in developing countries undergoing nutrition transition (Mukuddem-Petersen & Kruger, 2004:online). Nutritional stunting causes an increased risk of obesity (Sawaya & Roberts, 2003:online). There is the possibility that child undernutrition is linked to adult overweight, as the fetal origins hypothesis suggests. Persistent child undernutrition may contribute to the burden of overweight in women (Mendez et al., 2005:720). The risk of stunting with concurrent obesity is primarily related to low socio-economic status and maternal overweight. A child with an obese mother had more than double the risk of being overweight or obese (Fernald & Neufeld, 2007:628-629).

The association between childhood stunting and adult obesity has been investigated in several studies, amongst others by Popkin et al. (1996) and Sawaya et al. (1998). Mukuddem-Petersen and Kruger (2004:online) speculate that the reduced body mass of malnourished children results in decreased basal metabolic rate. Linear growth may then be limited when protein and other nutrient intakes are not

adequate, but potential fat deposition will continue (Mukuddem-Petersen & Kruger, 2004:online). Mild stunting might be associated with a greater susceptibility to the effects of a high-fat diet (Steyn et al., 2005:12). Increased body fat deposits in stunted children may be due to a lower fasting fat oxidation rate (Kruger et al., 2005, 492). A greater central fat distribution has been found in children stunted early in childhood (Steyn et al., 2005:12).

As previously mentioned, stunting in early childhood, is associated with excess weight gain later in life, as well as with the development of type 2 DM and hypertension (Van der Merwe & Pepper, 2006:4; Steyn et al., 2005:5). There is more rapid weight gain in childhood and adolescence, especially as sub-cutaneous fat, in stunted children as opposed to non-stunted children (Mukuddem-Petersen & Kruger, 2004:online). Independent of LBW, adult obesity is linked to cortisol axis activation with adult metabolic- and cardiovascular disease and elevated blood pressure in young adults (Van der Merwe & Pepper, 2006:4). Stunting in childhood also predisposes the child to overweight or obesity when sufficient food becomes available; which increases the risk of developing CDL (especially with a Westernized diet in adulthood) should the child develop into an obese adult (Vorster & Kruger, 2007:323; Steyn et al., 2006:20).

A study conducted in a Brazil shantytown in 1995, found a high prevalence of undernutrition in children (30%) with a shift toward overweight and obesity among adolescents (21% in girls and 8,8% in boys) and adults (14,6%) (Sawaya & Roberts, 2003:online). Sawaya and Roberts (2003:online) also found a high prevalence of obesity associated with stunting, indicating that obesity could occur when growth faltering occurred. Other studies that were conducted later, have replicated these findings, showing an association between stunting and overweight in other countries such as Russia, China and South Africa. All of these countries are undergoing nutrition transition (Fernald & Neufeld, 2007:629; Jinabhai et al., 2003:online; Sawaya & Roberts, 2003:online; Popkin et al., 1996:3013). A greater BMI was considered a reflection of a greater reduction in height, rather than greater increase in weight (Kruger et al., 2005, 492; Popkin et al., 1996:3014).

In Mexico, the prevalence of concurrent obesity or overweight with stunting was double among a rural group of children, than urban children. The prevalence in three year old urban children was 5,9%, and 12,1% for rural children (Fernald & Neufeld, 2007: 626). Popkin et al. (1996:3012) showed that children in China, Russia and South Africa who were stunted were also at risk of being overweight. However, Brazilian data did not show this effect. The relative risk of being overweight if stunted was 2,6 amongst South African children and 7,7 amongst Russian children (Jinabhai et al., 2003:online; Faber et al., 2001:401; Popkin et al., 1996:3012). “Small” and “thin” babies in India had poor muscle mass as well as visceral mass, but higher adiposity for a certain weight when compared with Caucasian babies (Van der Merwe and Pepper, 2006:4).

The high prevalence of undernutrition in South African children places them at a higher risk for CDLs in later life (Vorster & Kruger, 2007:323). The NFCS found that stunting increased the risk of being overweight (Steyn et al., 2006:34; Steyn, 2005a:44; Steyn et al., 2005:4). Joubert et al. (2007:688) mentions the coexistence of over- and undernutrition in the same household. Cameron (2003:38) states that recent research links stunting in infancy and childhood to obesity. Stunting itself confers an increased risk for overweight in children (Levitt et al., 2005:59). It is interesting to note that underweight children are often found in the same households as overweight caregivers. This is the case in both South Africa and

other developing countries, such as China, Russia and Brazil (Vorster & Kruger, 2007:323; Goedecke et al., 2005:71; Steyn et al., 2005:10; WHO/FAO, 2002:61; Faber et al., 2001:410). Where there is a high prevalence of obesity among black women, a high prevalence of stunting in children is found, particularly in rural areas (Steyn et al., 2005:5).

In the 1994 Living Standards Measurement Survey, 30,6% of South African children between three and six years old were found to be stunted and 7,5% were either overweight or obese. Thirteen percent of the children were both stunted and obese, as opposed to five percent who were neither stunted nor obese (Cameron, 2003:38).

In a rural area of Limpopo Province, 19% of children were both stunted and overweight. Having a greater length/height at one year of age was protective against stunting at the age of three, while gaining more weight within the first year of life increased the risk of being overweight at three years by 2,39 times. During the follow-up at three years of age, 48% of the children were stunted, nine percent were underweight, one percent were wasted, 18% were overweight and 24% were obese (Mamabolo et al., 2005:online). The NFCS in South African children between one and nine years of age reported that the prevalence of combined overweight and obesity (17,1%) at the national level was as high as that for stunting (19,3%) (Mamabolo et al., 2005:online; Steyn, 2005b:249).

In a study of nearly 500 school girls aged between ten and 15 years from the North West Province (THUSA-BANA study), stunting was associated with increased deposition of subcutaneous fat and centralization of body fat stores (Mukuddem-Petersen & Kruger, 2004:online).

The relative risk for overweight if stunted, was found to be 2,6 among South African children (Steyn et al., 2005:12; Jinabhai et al., 2003:online; Faber et al., 2001:410; Popkin et al., 1996:3012). Because the focus of the health services budget may still primarily be on the prevention and treatment of undernutrition, rather than overweight or obesity, it is important to determine whether undernutrition in young children may in the long term lead to overweight or obesity in older children and adults, since the negative health and economic consequences of obesity are prevalent in South Africa (Steyn et al., 2005:12).

2.3. The nutrition transition

According to the WHO/FAO, “rapid changes in diets and lifestyles have occurred with industrialization, urbanization, economic development and market globalization, which have a significant impact on the health and nutritional status of populations, particularly those in developing countries and in countries in transition” (WHO/FAO, 2002:1). Mukuddem-Petersen and Kruger (2004:online) classify the nutrition transition as “a sequence of characteristic changes in dietary patterns and nutrient intakes associated with social, cultural and economic changes during the demographic transition.” The WHO/FAO (2002:13) state that it entails changes in diet as well as reduced physical activity. The dietary changes include a shift towards a higher energy-density diet with increased intake of fat, especially saturated fat (from animal origin), refined carbohydrates and added sugars, as well as a reduced intake of vegetables and fruit (Steyn, 2005b:249; WHO/FAO, 2002:2,13,15). As mentioned previously; traditional diets in South Africa consisted of >60% to 65% of total energy from carbohydrates, and <25% of total energy from fat, whereas the Western diet consists of <50 to 55% of total energy from carbohydrates and >30% to 35% of

total energy from fat; and is lower in fibre intake and higher in free sugar intake (>10% of total energy) (Joubert et al., 2007:684; Steyn et al., 2006:13,14; Steyn, 2005a:36; Bourne et al., 2002:157,159; WHO/FAO, 2002:19).

The World Cancer Research Fund (WCRF) and American Institute for Cancer Research (AICR) state that food supplies usually become more secure with industrialization and urbanization, which consequently makes food more available for consumption. Diets usually become denser in energy, with fewer starchy foods and more fats; oils; sugars; and additives, as well as alcoholic drinks being ingested. At the same time, physical activity decreases, with the need for energy decreasing and the rate of overweight and obesity increasing (WRCF/AICR, 2007:4). Traditional diets and lifestyles are rapidly changing in developing countries even though food insecurity and undernutrition persist where CDLs are becoming epidemic (WHO/FAO, 2002:8). Kruger et al. (2005:493) state that globalization increases the CDL risk in urban populations, because an environment that promotes intake of fatty and sugary foods is created.

The traditional diet is associated with a low prevalence of degenerative diseases, whereas the Western diet is associated with increased prevalence of CDLs (Bourne et al., 2002:157). Modern dietary patterns are seen by the WHO/FAO (2002:5) as “risk behaviours that travel across countries and are transferable from one population to another *almost like an infectious disease*, affecting disease patterns globally” [emphasis added]. The nutrition transition contributes to a higher risk for developing CDLs, even in the poorest countries. The apparent accelerating pace of the transition is especially evident in low– and middle-income countries (WHO/FAO, 2002:13). The nutrition transition has important implications for preventive strategies to control CDLs (Jinabhai et al., 2003:online). The urbanized poor in developing countries are “at the greatest risk for developing health problems associated with dietary changes (Yach et al., 1991:212).

2.3.1. In the world

Rapid urbanization is occurring globally. In 1994, the rate was 44,5%; and it is projected to be 61% by the year 2025. Among developing countries, the rate was 12,6% in 1970; 21,9% in 1994; and is projected to be 43,5% by 2025 (Seedat, 2007:317).

Diets in developing countries are changing as income rises. The intake of staple foods, such as cereals, roots and tubers, has declined, while consumption of meat, dairy and oil has increased. The *per capita* meat consumption in developing countries rose by 150% between 1964 and 1966; as well as between 1997 and 1999. By 2030, the per capita consumption of livestock products could rise by a further 44%. Poultry consumption is predicted to grow the fastest (WHO/FAO, 2002:26). In developing countries, demand is predicted to grow faster than the production of meat products (WHO/FAO, 2002:27).

In many countries, like the United States of America (USA), eating patterns have changed over the past thirty years, with increased consumption of fast foods, pre-prepared meals and carbonated drinks (WHO/FAO, 2002:38). In Europe, North America, Oceania and Japan, life expectancy has continued to increase throughout the past century, with CDLs now being the main cause of death. It is estimated that approximately one-third of all cancers and one-quarter of CVD are attributable to an “affluent” diet. Many countries in Asia are in transition, both epidemiologically and demographically. In China, fat levels

in the diet have risen from 26% of total energy in 1981, to thirty percent in 1988, as the diets have become more Westernized (Darnton-Hill & Coyne, 1998:25).

An important feature of the nutrition transition is the increase in the quantity and quality of dietary fats. On average, Africa has the lowest fat consumption, while parts of Europe and North America have the highest consumption (WHO/FAO, 2002:17). The 2010 dietary guidelines for Americans recommend that total fat intake should provide twenty percent to 35% of the total energy in the diet, while saturated fat should provide less than ten percent of total energy (United States Department of Agriculture (USDA) and United States Department of Health and Human Services (USDHHS), 2010:24).

According to a study conducted by the FAO between 1988 and 1990, nineteen countries (mostly Sub-Saharan African and some South Asian), had a total fat intake of less than 15%; and 24 countries' total fat intake was above the maximum recommendation of 35%; the majority of these countries being in North America and Western Europe (WHO/FAO, 2002:18). The WCRF/AICR (2007:11) state that in recent years, rapid changes in diet have been seen in the middle- and low-income countries of Asia, Africa, the Middle East and Latin America, with a shift in energy consumption towards more fats, oils and added sugars. Various national dietary survey data show that the proportion of animal fat in total fat intake was lower than ten percent in the Democratic Republic of the Congo, Mozambique, Nigeria, Sao Tome and Principe, and Sierra Leone, while it was above 75% in Denmark, Finland, Hungary, Mongolia, Poland and Uruguay (WHO/FAO, 2002:19). In Japan, total fat intake has increased from 15% of dietary energy in 1965, to 25% in 1985 (Darnton-Hill & Coyne, 1998:25).

2.3.2. In Africa and South Africa

According to O'Keefe et al. (1983:679), only 12,6% of the black population of South Africa was urbanized in 1911. That figure has risen to 18% by 1960, and it was predicted to reach 75% by the year 2000. Analysis of the 1985 census data showed that South African blacks moved into urban areas at a rate of 3,5% per year. Yach et al. (1991:212) reported that the majority of the white population in South Africa was already urbanized in the early 1990s. It was predicted that the urban Black population would increase from 6,5 million in 1985 to more than twenty million by 2000 (Cameron, 2003:32; Yach et al., 1991:212). Urban areas were predicted to double in size by 2010. The expected increases in the urban population did not happen, however. The United Nations Commission for Human Settlement reported in 2001 that the urbanization rate was only 1,2% during the late 1990s (Cameron, 2003:32).

Currently, the black population represents approximately 76% to 79% of the total South African population (Steyn et al., 2006:6; Labadarios et al., 2005:534; Statistics South Africa, 1999:1). The percentage of the black population living in urban areas increased from 35,8% in 1993, to 43,3% in 1996 (Vorster, 2002:239). It should however be kept in mind that the 1996 census was the first comprehensive census conducted in South Africa (Bradshaw et al., 2003:1) and figures before that date should be interpreted with caution. Since the percentages of coloureds, Indians and whites in urban areas remained relatively constant for the period 1993 to 1996, the increase in urbanization from 48,3% to 53,7% was because of the rapid migration of blacks (Vorster, 2002:239). Kruger et al. (2005:492) state that the nutrition transition is occurring because the black population now (i.e. post-apartheid) has more freedom of movement and are more exposed to the global market economy.

Already in 1977, Seftel (1977:121) reported that the diseases in the urban black population in South Africa, were those of a population in transition. Two groups of new disorders were identified, namely (1) alcohol-related disorders; and (2) the diseases encountered in Western populations (i.e. CDLs). At that time, Seftel (1977:121) had already indicated that obesity and hypertension had attained epidemic proportions among urban Blacks. He even went so far as to state that their prevalence may actually have exceeded that of the white population. Other conditions, such as CHD, gout, gallstones and colonic cancer, which emerged later, were still uncommon amongst the black population at that time (Seftel, 1977:121). In 1983, O'Keefe et al. (1983:679) already noted a change in nutritional status in females; characterized by a shift from chronic malnutrition to obesity, and the appearance of more Westernized disease patterns.

In 1997, Darnton-Hill and Coyne (1998:27), reported that Sub-Saharan Africans were eating less coarse grains and roots and tubers and more wheat and rice. Africa was the only continent where intake of dietary fats had not increased (WHO/FAO, 2002:17). However, this is not the case in South Africa, as will be shown.

If rural Africans follow traditional lifestyles (including a traditional diet) they usually have a good nutritional status and very low risk factors for CDLs (Vorster & Kruger, 2007:323). With urbanization, however, the diet consists of more fats and oils (more total and trans-fatty acids), more animal-derived foods (more saturated fatty acids), less staples, fruits and vegetables (less unrefined carbohydrate) and less dietary fibre and micronutrients (Vorster & Kruger, 2007:323; Seedat, 2007:318). This diet is associated with increased risk for all CDLs, including CVD. Vorster and Kruger (2007:323), refer to "another double burden," where individuals who were programmed during fetal life and infancy through undernutrition are more vulnerable to CVD, develop an even greater risk for CVD in later life when they are exposed to modern/Westernized diets.

According to the MRC Technical Report on dietary changes and the health transition in South Africa (Steyn et al., 2006:6), the unhealthy lifestyles associated with CDLs are the same globally. Diets associated with CDLs consist of a high consumption of energy-dense foods with low levels of micronutrients or fibre, and are high in total fat, saturated fat, trans fatty acids, free sugars and salt (Steyn et al., 2006:6; Darnton-Hill & Coyne, 1998:25).

South Africa has become the most important African market for "Coca-Cola", and one of their largest markets in the world (Kruger et al., 2005:493). Rapid and unplanned urbanization have taken place which accelerated changes in traditional diets (Steyn et al., 2005:4; Kruger et al., 2005: 493), and easy access to high-fat foods and tobacco, which are contributing risk factors to CDLs (Kruger et al., 2005: 493).

Over a period of fifty years (1940 to 1990), fat intake amongst urban blacks increased from 16,4% to 26,2% of total energy, with a relative increase of 59,7%; and intake of carbohydrates decreased from 69,3% to 61,7% of total energy, with a relative decrease of 10,9% (Goedecke et al., 2005:71; Bourne et al., 2002:157). Shifts to the Western diet were also found amongst rural black communities. There was an increased prevalence of CDLs with a Western diet (Bourne et al., 2002:157). Bourne et al. (2002:158) found that as urban exposure increased, so did the "atherogenicity" of the diet. According to Goedecke et al. (2005:71), the changes that were reported over the fifty year period, were more

dramatic than those observed in Western countries undergoing rapid industrialization over longer periods.

As part of THUSA study undertaken from 1996 to 1998, MacIntyre et al. (2002:253) reported that the dietary intakes of urban communities in the North West Province indicated that a nutrition transition was present, with an increase in total fat and animal protein intake, and less carbohydrates being consumed (Vorster, 2002:241).. A high fat intake among urban subjects has been reported to contribute to increasing rates of obesity among South Africans (Goedecke et al., 2005:71; Kruger et al., 2005:493). The THUSA study found that the lowest fat intake was in rural participants (46 g per day) and the highest fat intake in urban middle-class populations (56,3 g per day) (Kruger et al., 2002:426).

In 2006, the intake of carbohydrates and fibre among black people in transition decreased with increased time of living in a city, while fat intake increased. Protein intake remained more or less the same, but the source changed from mostly plant-based to mostly animal-based protein (Steyn et al., 2006:13).

The 1998 SADHS (DoH et al., 2002), Dikgale study (Steyn et al., 2001) and BRISK study (Steyn et al., 1991) all confirm that urban blacks consume more fat and less carbohydrates than their rural counterparts. Again, protein intake stayed more or less the same in all above-mentioned studies. In the North West Province, low intake of fruit, vegetable and milk was reported in all groups studied, except for the upper middle class in urban communities. Alcohol consumption was higher in rural communities (MacIntyre et al., 2002:253). Men consumed alcohol far more frequently than women (Steyn, 2005b:249; MacIntyre et al., 2002:253). Alcohol consumption seemed to have increased in the South African population from 1962 to 2001, with thirty percent of males reported to use alcohol excessively as compared to ten percent of women (Steyn et al., 2006:18). In addition, high intakes of sodium and low intakes of foods containing potassium, calcium and magnesium contributed to the increased prevalence of hypertension in black communities (Steyn, 2005b:249).

The MRC Technical Report also noted that although rural populations had higher intakes of cereals and vegetables, urban populations consumed more sugar, meat, vegetable oil, dairy, fruit, roots, tubers and alcohol (Steyn et al., 2006:17). Vorster et al. (2005:online) state that the decreased intake of the South African staple food (maize porridge) and the increased intake of added fats and oils, as well as foods of animal origin during urbanization represents similar dietary patterns as observed in other developing countries. Their findings of decreased intake of total carbohydrate and increases in animal protein and total fat intake correlate with earlier studies comparing nutrient intakes between rural and urban South Africans (Vorster et al., 2005:online).

As far as dietary intake of children is concerned, the NFCS found that one in three children of all age groups in the Northern Cape, Mpumalanga, Northern Province and the Free State received less than half of their daily energy needs (Labadarios, et al., 2005:537). Intake of sugar, as a percentage of total energy, was highest in the Western Cape (15%) and Northern Cape (13%) and lowest in the Free State, Northern Province and Mpumalanga. The ratio of poly-unsaturated to saturated fats was 0,9 in Gauteng and Western Cape and 1,4 in the Eastern Cape and Northern Province. Girls received more energy

from fat than boys. Rural children received a greater percentage of energy from plant-based protein, similar to the national pattern (Labadarios et al., 2005:539).

Half of the South African population does not meet the local dietary guidelines of four portions of fruits and vegetables daily, while a quarter of the population eats none at all. Shifts towards the Western diet have been suggested in both rural and urban areas with a decrease in starchy foods and fibre consumption and an increase in fat intake (WCRF/AICR, 2007:9).

2.4. The burden of disease

As previously mentioned, many developing countries face a double burden of both overnutrition and undernutrition (Mendez et al., 2005:714).

2.4.1. In the world

Many developing countries have increased rates of child mortality, while obesity and CDLs prevalence are also increasing (Darnton-Hill & Coyne, 1998:23). In 1990, CDLs accounted for 28,1 million deaths worldwide (Murray & Lopez, 1997a:1436; Murray & Lopez, 1997b:1498). This amount is predicted to increase to 49,7 million by 2020 (Murray & Lopez, 1997b:1498). According to the World Health Organization's Global Status of Health Report, the Americas have ten percent of the global burden of disease and utilize 38% of the world's healthcare workers for fifty percent of the world's diseases. In contrast, in Africa 11% of the world's population has 24% of the world's diseases, but only three percent of the healthcare workers and one percent of the global financial resources (Seedat, 2007:317). There are 57 countries that cannot meet a widely accepted basic standard of healthcare, of which 36 countries are in Sub-Saharan Africa (Seedat, 2007:317). In high-income countries, only one percent of deaths occurred in children younger than 15 years, and 84% occurred in people older than sixty years (WHO, 2008:8). According to Murray and Lopez (1997c:1269), the developing world presents with 98% of all deaths worldwide among children younger than 15 years old; 83% of all deaths worldwide among 15- to 59-year-olds, and 59% of deaths worldwide among seventy-year-olds. The probability of death between birth and 15 years is 22% in Sub-Saharan Africa, compared to 1,1% in countries with established market economies (Murray & Lopez, 1997c:1269). The probability of adult death due to CDLs is also higher in Sub-Saharan Africa and other developing countries than in first world countries (Murray & Lopez, 1997c:1269). In 1990, CDLs already accounted for a higher burden of disease in countries like China, Latin Ame9rica and the Caribbean than Asia and the Middle East (Murray & Lopez, 1997a:1441).

An initial global assessment of the burden of disease was conducted from 1993 to 1996 by the Harvard School of Public Health, the World Bank and the WHO (Murray & Lopez, 1997a, 1997b and 1997c). With the application of various mathematical formulae and projections, consistent and coherent estimates were derived from the data. The study estimated disease and injury rates by age and sex for 1990 in eight world geographical regions and these were categorized according to DALYs (Bradshaw et al., 2003:1).

The primary indicator used to summarize the burden of premature mortality and disability (including temporary disability) was the DALY. Murray and Lopez (1997a:1436) define the DALY as "the sum of life years lost due to premature mortality and years lived with disability adjusted for severity." The DALY

is incidence-based, rather than prevalence-based and can be used as a summary measure of health gaps in a population. . The DALY consists of Years of Life Lost (YLL) due to premature mortality, plus Years lived with a disability (YLD), which are weighted according to the severity of the disability. Thus DALY = YLL + YLD (Bradshaw et al., 2003:3).

The DALY includes any health outcome that represents a loss of welfare. Only age and sex are included as individual characteristics in the set of variables. Health outcomes are also treated as alike, irrespective of where or to whom they occur. No preference is given to socio-economic setting (Bradshaw et al., 2003:4).

In 1997, developed countries accounted for 11,6% of the world's burden from all causes of death and disability and accounted for 90,2% of health expenditure worldwide. CDLs accounted for 40,9% of worldwide DALYs, malignant cancers for 5,1% and cardiovascular conditions for 9,7% (Murray & Lopez, 1997a:1436).

A change is predicted in the rank orders of disease burden for 15 leading causes of disability and death in the world, as shown by DALYs. IHD will be the most common cause of DALYs worldwide in 2020 (Seedat, 2007:318). The changes in ranking as predicted by Murray and Lopez (1997b:1499) for certain selected CDLs and other disease, are set out in Table 2.1. The summarized percentage distribution of current DALYs (for 1990) and projected DALYs (for 2020) for certain diseases are set out in Table 2.2.

Table 2.1. Global changes in ranking for selected causes of death from 1990 to 2020 (Murray & Lopez, 1997b:1499, Table 1 - amended)

Disorder	**Ranking**		**Change in ranking**
Within top 15	**1990**	**2020**	
Ischemic heart disease	1	1	0
Cerebrovascular disease	2	2	0
Chronic obstructive pulmonary disease	6	3	↑3
Trachea, bronchus and lung cancers	10	5	↑5
Liver cirrhosis	13	12	↑1
Stomach cancer	14	8	↑6
Diabetes mellitus	15	19	↓4
Outside top 15			
Liver cancer	21	13	↑8
HIV	30	9	↑21

In 1997, IHD and cerebrovascular disease (i.e. stroke) were already the leading CDL-specific causes of global DALYs (Murray & Lopez, 1997a:1436,1441). As can be seen from Table 2.1, HIV has made a big leap up the ranks and stomach cancer, liver cirrhosis, respiratory cancers and chronic obstructive pulmonary disease (COPD) have all moved up the ranks. Only DM has moved down the rankings.

Both CVD and malignant neoplasms are predicted to increase in both developed and developing countries by 2020 (Table 2.2). Interestingly, the projections for DM show that figures will remain relatively fixed. Murray and Lopez (1997b:1502) project that by 2020, almost sixty percent (59,7%) of DALYs worldwide will come from CDLs, as opposed to 40,9% in 1990. The projected increase in deaths from CDLs is expected to be larger for males (91%) than for females (61%) (Murray & Lopez, 1997b:1501). A selection of leading causes of DALYs in 2020 according to baseline projections is set out in Table 2.3.

Table 2.2. Percentage distribution of current (1990) and projected DALYs (2020 - in bold italics) for diabetes mellitus (DM), cardiovascular disease (CVD), malignant neoplasms (MN) and other neoplasms (ON) for certain countries in the world (Murray & Lopez, 1997a:1439, Table 2; Murray & Lopez, 1997b:1501, Table 2 - both amended)

	DM	CVD	MN	ON
Established market economies	2,4 / ***2,1***	18,6 / ***19,4***	15,0 / ***17,3***	0,9 / ***0,6***
Former socialist economies of Europe	1,1 / ***0,7***	23,2 / ***26,1***	11,7 / ***16,1***	0,6 / ***0,3***
India	0,8 / ***0,8***	8,2 / ***18,4***	2,5 / ***7,1***	0,1 / ***0,1***
China	0,5 / ***0,4***	11,0 / ***16,3***	8,7 / ***18,7***	0,4 / ***0,3***
Other Asia and islands	0,7 / ***0,9***	10,1 / ***15,6***	5,1 / ***11,6***	0,3 / ***0,2***
Sub-Saharan Africa	0,2 / ***0,2***	3,9 / ***6,0***	2,1 / ***4,5***	0,2 / ***0,2***
Latin America and the Caribbean	1,5 / ***1,6***	8,0 / ***13,2***	4,5 / ***8,5***	0,5 / ***0,4***
Middle Eastern Crescent	1,0 / ***1,0***	11,1 / ***17,7***	2,4 / ***5,3***	0,2 / ***0,2***
Developed	1,9 / ***1,5***	20,4 / ***22,0***	13,7 / ***16,8***	0,8 / ***0,5***
Developing	0,7 / ***0,7***	8,3 / ***13,8***	4,0 / ***9,0***	0,2 / ***0,2***
World	0,8 / ***0,8***	9,7 / ***14,7***	5,1 / ***9,9***	0,3 / ***0,2***

According to Table 2.3, major increases in DALYs are expected for some leading CDLs. Deaths and DALYs due to CDLs were projected to increase by 77% and 47% respectively (Murray & Lopez, 1997b:1502).

Table 2.3. Selection of projected leading causes of DALYs in 2020 according to baseline projections (Murray & Lopez, 1997b:1502, Table 3 – amended)

	World			***Developed***			***Developing***		
Disease	**Rank**	**DALYs (x10^6)**	**Cum %**	**Rank**	**DALYs (x10^6)**	**Cum %**	**Rank**	**DALYs (x10^6)**	**Cum %**
IHD	**1**	82,3	5,9	**1**	18,0	11,2	**3**	64,3	16,1
Stroke	**4**	61,4	21,1	**2**	9,9	17,4	**5**	51,5	24,6
COPD	**5**	57,6	25,3	**9**	4,9	46,0	**4**	52,7	20,4
TB	**7**	42,5	31,4				**6**	42,4	28,0
HIV	**10**	36,3	39,7				**10**	34,0	40,4
Lung Ca				**4**	7,3	28,0			
Alcohol use				**6**	6,1	36,1			

The WHO uses years of life lost (YLLs) to measure premature mortality that takes into account both the frequency of deaths and the age at which death occurs, and is used for the calculation of the DALYs. It should be kept in mind that a disease can have a low incidence, but can cause death or disability, and therefore will result in a high burden of disease or many YLLs (WHO, 2008:28-29). Based on the distribution of the world's deaths in 2004 (58,8 million), the South East Asia Region had the highest proportion of deaths (26%). High-income countries only contributed to 14% of the deaths, whereas the Western Pacific Region contributed to 18% and the African Region to 19% of deaths. However, based on the YLLs, the African Region accounted for 32% of all YLLs, followed by South East Asia (30%). This shows that people form Africa and South East Asia die at a relatively young age. The main causes of YLLs were peri-natal conditions; lower respiratory infections; diarrheal diseases; and HIV/AIDS. IHD (12,2% of all deaths) and cerebrovascular conditions/stroke (9,7% of all deaths) are responsible for only 5,8% and 4,2% of YLLs respectively (WHO, 2008:21-22).

Changes in the age structure of a population may result in substantial changes in the number of deaths, because death rates are strongly age-dependent for most causes (WHO, 2008:25). Aging populations in low- and middle-income countries will contribute to increased total deaths due to CDLs over the next 25

years. It is projected that global cancer deaths will increase from 7,4 million in 2004, to 11,8 million in 2030; and cardiovascular deaths from 17,1 million in 2004, to 23,4 million in 2030. CDLs are projected to account for over 75% of all deaths in 2030 (WHO, 2008:22). The four projected leading causes of death globally in 2030 will be IHD; cerebrovascular disease/stroke; COPD; and lower respiratory infections (WHO, 2008:23).

The majority of the world's poor still face hunger and undernutrition, which continue to dominate the health of the world's poorest nations. Nearly thirty percent of the world's population is currently suffering from one or more forms of undernutrition (WHO/FAO, 2002:8). People in lower socio-economic groups have the largest burden of disease and the least amount of resources for adequate treatment (Sawaya & Roberts, 2003:online). Therefore, the mortality is higher in poorer communities in developing countries; especially among children younger than five years of age and adults with CDLs (Sawaya & Roberts, 2003:online). Undernutrition affects about 200 million children younger than five years in developing countries (Nannan et al., 2007:733). Almost 16% of all DALYs worldwide in 1997 were attributed to childhood undernutrition (Murray & Lopez, 1997a:1436,1441). The rapidly increasing burden of CDLs is a key determinant of global public health. Already 79% of deaths attributable to CDLs are occurring in developing countries, predominantly in middle-aged men (WHO/FAO, 2002:31).

It is estimated that more than one billion adults in the world are overweight, of which at least 300 million are obese. Obesity, as a risk factor for CDLs, is a global public health concern (Kruger et al., 2005:491). Increases in obesity over the past thirty years have been paralleled by a dramatic rise in the prevalence of DM (WHO/FAO, 2002:61). Countries in transition, such as China, Brazil and South Africa, have an increased rate of obesity across all economic levels and age groups (Kruger et al., 2005:491).

Populations of many countries suffer the worst of both worlds, i.e. the effect of undernutrition, impacting mostly on infants, children and women of child-bearing age; and overnutrition, creating a variety of chronic health conditions in the middle-aged and older populations (Darnton-Hill & Coyne, 1998:25). The co-existence of undernutrition and obesity among the poor places a burden on social-, economic- and health care systems (Sawaya & Roberts, 2003:online).

Mendez et al. (2005:714) found that the prevalence of overweight amongst women exceeded underweight in over half of the countries studied, including the urban areas of mostly South American and African countries. The median ratio of overweight to underweight was 5,8:1 in urban and 2,1:1 in rural areas. In many of the developed countries, the ratio was about 20,0 (Mendez et al., 2005:719). Countries with high levels of urbanization had a high absolute prevalence of overweight, with little difference between urban and rural areas, and a high ratio of overweight to underweight. Even in poor countries, where underweight usually persisted, there was a fairly high prevalence of overweight in rural areas (Mendez et al., 2005:714).

2.4.2. In Africa

In 1995, the African population was estimated to be 580 million (Seedat, 2007:317). Between 1990 and 1995, two million children died annually before their first birthday. About 291 million African residents had an average income below US$1 (R8) a day in 1998; and 124 million of those younger than 39 years

were at risk of dying before forty years. During 1990 to 1995, about 205 million Africans had no access to health services and 249 million were without clean drinking water (Seedat, 2007:317).

In Africa, elevated blood pressure and cholesterol are among the top ten risk factors for the overall disease burden (WHO, 2008a:8). Poor households undergoing industrialization in urban areas are particularly affected by underweight coexisting with obesity (Kruger et al., 2005:491). Eleven percent of the world's population (in Africa) has 24% of the world's diseases, but only three percent of the world's healthcare workers and only one percent of global financial resources. Fifty-seven countries cannot meet a widely accepted basic standard of healthcare, of which 36 are in Sub-Saharan Africa (Seedat, 2007:317).

Deaths in children younger than five, showed a burden distribution which leaned heavily towards Africa (WHO, 2008:16). In 2004, almost half (46%) of all deaths in Africa occurred in children under 15 years. Only twenty percent of deaths occurred in people older than sixty years (WHO, 2008:8).

The main focus of public health programs in Sub-Saharan Africa over the past decade has been the eradication of undernutrition and infectious diseases (Kruger et al., 2005:491). In 1994, about a third (32%) of children in Africa were undernourished (De Villiers & Senekal, 2002:1231). In 1998, 43 million of the African population were stunted due to undernutrition (Seedat, 2007:317). In 2004, 13,7 million children younger than four years were wasted and 51,9 million were stunted (WHO, 2008:32).

Thirty-five percent of children younger than five are believed to be stunted in Africa as a whole, and 42% in Sub-Saharan Africa (Mamabolo et al., 2005:online). A higher prevalence of stunting in male children has occasionally been reported in Sub-Saharan Africa. In ten countries (including Cameroon, Ghana, Kenya, Malawi, Namibia, Nigeria, Tanzania, Uganda, Zambia and Zimbabwe) boys younger than five years were more likely to become stunted than girls. This sex difference was more pronounced in lower socio-economic groups, and might suggest that boys are more vulnerable to illness (Wamani et al., 2007:online).

2.4.3. In South Africa

It was already predicted in 1972 that increasing urbanization and a rise in socio-economic status in developing populations would increase their proneness to obesity, DM, hypertension and stroke. This has largely come true for the black population in South Africa (Bourne et al., 2002:159). It was predicted in 1988 that by the turn of the century about 75% of South Africa's population would be urbanized (Brink AJ. 1988, as referred to by Mollentze et al., 1995:90).

South Africa suffers from a quadruple burden of disease, which can be classified as a combination of: poverty-related infectious disease and underdevelopment; CDLs; violence-related trauma and injuries; and the HIV/AIDS epidemic (Steyn et al., 2006:6; Bradshaw et al., 2003:iii; Bourne et al., 2002:157). The double burden of disease consisting of undernutrition-related infections and overnutrition-related CDLs within families, communities and population groups, is also experienced in South Africa (Vorster & Kruger, 2007:323).

Compared with other regions of the world, South Africa has a higher overall burden of disease due to HIV/AIDS, injuries and respiratory diseases. The burden due to DM in South Africa is higher than any of the other regions, except for the American region (Norman et al., 2010 :online). CDLs, which were thought to be a problem of affluent countries, are now increasingly prevalent among all population groups in South Africa, while undernutrition remains a problem among children, particularly in rural areas (Kruger et al., 2005:491).

Globally, 4,1% of deaths are directly attributable to undernutrition, while in South Africa the amount of deaths is 38,3% (Nannan et al., 2007:737). Almost four percent (3,5%) of the burden from LBW is attributable to maternal underweight. In 2000, the burden attributable to underweight in both women and children accounted for 12,3% of deaths and 2,7% of all DALYs in South Africa (Nannan et al., 2007:733, 736). Almost 11% of the total DALYs were in children younger than five years old (Nannan et al., 2007:736). The role of undernutrition on: the progression of HIV and AIDS; the risk for CDLs; and the susceptibility of disease is still unknown. The burden of HIV and AIDS related to underweight cannot be quantified, due to insufficient data (Nannan et al., 2007:737). Among leading causes of death in South African children younger than five, LBW ranked second (12,1%) (Table 2.4) after HIV/AIDS (35,1%). Protein-energy malnutrition was ranked fifth (4,7%).

Table 2.4. Top twenty specific causes of death in children under five years, South Africa 2000 (Norman et al., 2010b)

Rank	Cause of death	Deaths	%
1	HIV/AIDS	33 735	35.1
2	Low birth weight	11 597	12.1
3	Diarrheal diseases	10 622	11
4	Lower respiratory infections	6 019	6.3
5	Protein-energy malnutrition	4 528	4.7
6	Neonatal infections	2 851	3
7	Birth asphyxia and trauma	2 523	2.6
8	Road traffic accidents	1 269	1.3
9	Congenital heart disease	1 213	1.3
10	Fires	1 145	1.2
11	Bacterial meningitis	1 124	1.2
12	Neural tube defects	999	1.0
13	Septicemia	965	1.0
14	Tuberculosis	741	0.8
15	Interpersonal violence	680	0.7
16	Drowning	556	0.6
17	Cot death	480	0.5
18	Down syndrome and other chromosomal	470	0.5
19	Congenital disorders of GIT	371	0.4
20	Sexually transmitted disease (excluding HIV)	252	0.3
	All causes	**96 158**	

The first South African Burden of Disease study conducted by Bradshaw et al. (2003) divided causes of death into three broad groups, i.e.: Group I (communicable diseases, maternal causes, perinatal conditions, and nutritional deficiencies); Group II (the CDLs); and Group III (the injuries) (Norman et al., 2006:3). In 2000, CDLs accounted for 21% of years of life lost (YLL), compared to HIV/AIDS, which accounted for 25% (Bradshaw et al., 2003:v). When looking at genders, CDLs contributed 21% of YLL in females and twenty percent in males, whereas HIV/AIDS contributed to 47% YLL in females, but only 33% in males. Injuries were proportionately higher in males (22% YLL) than in females (8% YLL) (Bradshaw et al., 2003:v).

In the NFCS conducted in 1999, nearly twenty percent of children were stunted, while 17% were considered overweight (Labadarios et al., 2005:536; Steyn, 2005b:249; Levitt et al., 2005:59; Goedecke

et al., 2005:71). Kruger et al. (2002:427) showed that overweight and obesity in black women of the North West Province in South Africa was not only related to diet, but also to inactivity, which illustrates that other lifestyle factors are also involved in increasing risk for CDLs. For the period 2002–2003, 44% of South African men and 49% of women aged between 18 and 69 years old were sedentary (WCRF/AICR, 2007:9).

WCRF/AICR (2007:8) states that in 2001, the population of South Africa was almost 47,5 million with a gross domestic product (GDP) of US$8 506 (about R68 048) per person. However, Bradshaw et al. (2003:ii), (as part of a study for the Initial Burden of Disease Estimates for South Africa) estimated the South African population at just over 45 million in 2000. In 2000, Bradshaw et al. (2003:ii) estimated the life expectancy at birth of South African males at 52,4 years and females 58,5 years of age, whereas the WCRF/AICR (2007:8) estimated the life expectancy at birth in 2001 at 47 years for men and 46 years for women. The WCRF/AICR (2007:8) also states that CDLs accounted for 53,9% of deaths, while infections, maternal-, perinatal- and nutritional conditions, contributed to 40,2% of deaths. Injuries caused only 5,9% of deaths in 2000. Bradshaw et al.'s data again contradicts this by showing that in 2000, CDLs were estimated to account for 37% of deaths, while infections, maternal, perinatal and nutritional conditions contributed to 21% of deaths and injuries 12%. WCRF/AICR used the 2006 South African census data, whereas Bradshaw et al. (2003:ii), used various resources, including the 1996 cause of death census data; the Department of Home Affairs data and the UNISA/MRC national injury mortality surveillance system to make their estimates.

2.5 Undernutrition

2.5.1 In the world

Undernutrition in young children captures society's extent of development and can be used as a marker for overall population well-being (Subramanyam et al., 2010:1). Undernutrition manifests as underweight (weight-for-age <-2SD from the reference median), wasting (weight-for-height <-2SD) and stunting (height-for-age <-2SD) (WHO, 2008:14; Nannan et al., 2007:733; Steyn et al., 2006:19). Subramanyam et al. (2010:1) states that "the nutritional status of young children is an important indicator of health and development—it is not only a reflection of past health insults but an important indicator of future health trajectories." Children younger than three years old are the most vulnerable to undernutrition, because their growth rate is the greatest. Therefore, their risk for growth retardation is also increased (Subramanyam et al., 2010:1).

The UNICEF conceptual framework (Figure 2.1) for the development of undernutrition explains that the causes of growth failure are complex, which promote a multi-factorial approach in intervention planning. The causes consist of immediate, underlying and basic. Immediate causes include: inadequate dietary intake; poor psychosocial care; and infectious disease. Underlying causes refer to: insufficient household food security; inadequate maternal- and child care; poor access to clean water; insufficient health services in an unhealthy environment; and inadequate female education and information. The basic causes include: available potential resources; economic structures in place; and political-, social- and cultural factors (Nannan et al., 2007:733; De Villiers & Senekal, 2002:1232; The World Bank &

UNICEF:2002:2-3; Darnton-Hill & Coyne, 1998:24). Victora et al. (2008:345) also state that poverty is both a cause and an outcome of poor human development

As can be seen from Figure 2.1., there is a relationship between poverty, undernutrition and underdevelopment (Vorster & Kruger, 2007:321). Undernourished children's diets are often lacking in energy, protein, vitamin A, iron, zinc and calcium. Children who are underweight are at increased risk of infections with an increased mortality risk. Undernutrition was found to contribute to between 35% and 56% of childhood deaths globally (WHO, 2008:14; Nannan et al., 2007:733), with 56% of deaths in children younger than five years in developing countries being attributable to undernutrition (Darnton-Hill & Coyne, 1998:24).

Figure 2.1. **The UNICEF Conceptual Framework** (The World Bank & UNICEF, 2002:18, Figure 1, amended)

Undernutrition can have long-lasting consequences on children's health, delay motor development, as well as impair cognitive function and school performance. In adults, it reduces work capacity and influences reproductive health (Victora et al., 2008:343,345; Nannan et al., 2007:733; Vorster & Kruger, 2007:322).

One of the WHO's millennium development goals is to halve (from 1990 levels) the proportion of children younger than five who are stunted, wasted or underweight by 2015 (Rehman et al., 2009:online). The first Millennium Development Goal aims to reduce poverty and hunger between 1990 and 2015 (Nannan et al., 2007:733).

An improvement in weight-for-age of only five percent can reduce child mortality by thirty percent (and for children under five years, by 13%) (Nannan et al., 2007:733). In 2005, the WHO estimated, by using the new child growth standards, that 32% of all children under five years old in developing countries were stunted and ten percent were wasted (Rehman et al., 2009:online). High levels of undernutrition are still found in South Asia, the Indo-Chinese countries and much of Africa (Darnton-Hill & Coyne, 1998:26). In 2004, 56,2 million of the world's children, aged between zero and four years, were wasted, of which 13,7 million (24,4%) were from Africa and another 27 million (48%) were from South East Asia. Another 182,7 million children younger than four years were stunted, where 51,9 million (28,4%) were from Africa and 76,5 million (41,9%) were from South East Asia (WHO, 2008:32, Table 7). Undernutrition in Indian children is mostly higher among the urban poor compared to rural areas (Kanjilal et al., 2010:online).

Subramanyam et al. (2010:3-4) state that in India greater household wealth was associated with lower probability of undernutrition. Children whose mothers had a greater education had a lower likelihood of undernutrition. The absolute rates of undernutrition in India remain higher than the majority of developing countries.

2.5.1.1 Stunting

Stunting is an indicator of chronic undernutrition (Waterlow, 1994:S1). Linear growth faltering causes stunting, which is an important public health problem for children living in developing countries where poverty, poor nutrition and a high prevalence of infectious diseases are common (Mamabolo et al., 2005:online). Short stature and short leg length are sensitive indicators of early socio-economic deprivation (WHO/FAO, 2002:35). Sawaya and Roberts (2003:online) postulate that in Brazil, stunting has been related to poverty levels more than to genetic background. Schrimpton and Kachondham (2003:3) state that stunted children are more likely to fall ill and die, compared to underweight or wasted children. Although the nutritional insult that caused stunting has happened in the past, the resulting consequences are ongoing (Schrimpton & Kachondham, 2003:4).

According to the criteria established by the WHO, the prevalence rate of 42% for stunting in 2002 in the Democratic People's Republic of Korea was still very high (Schrimpton & Kachondham, 2003:4). One-third of the world's young children in low-income countries were stunted due to undernutrition around 1997 (Darnton-Hill and Coyne; 1997:23). In 2004, an estimated 32% of children under five years of age were stunted (WHO, 2008:112). A non-representative survey of young children in government kindergartens in Korea, conducted in 1997 by the World Food Programme, found that 83% of children were stunted (Schrimpton & Kachondham, 2003:4). In Mexico, almost twenty percent of children younger than five years of age were stunted, with a higher incidence in rural areas (Fernald & Neufeld, 2007:624). The national average for stunting in Mexican children is 17%. The prevalence of stunting was 21,3% in non-indigenous (urban) children and 42,7% in indigenous (rural) children (Fernald & Neufeld, 2007:626).

In Tibet, the proportion of stunted children was greater in rural areas than in urban areas (Harris et al., 2001:345).

In 1992, 52,4% of Indian children younger than three years were stunted. The number decreased to 50,7% in 1998, and further to 44,7% in 2005 (Subramanyam et al., 2010:3). In the Indian National

Family Health Survey-3, a nationwide survey conducted between 2005 and 2006, it was found 38% of Indian children younger than three years were stunted (Kanjilal et al., 2010:online; Rehman et al., 2009:online). At age three, two-thirds (66%) of South Indian urban boys and 56% of girls were stunted. Factors associated with stunting in this study were: birth weight less than 2,5 kg; manual production of cigarettes for a daily wage in the household; maternal height less than 150 cm; being stunted, wasted or underweight at six months of age; and having at least one older sibling (Rehman et al., 2009:online).

Van der Merwe and Pepper (2006:4) note the interesting relationship between stunting and obesity in India, where the clinical measurement of BMI underestimates adiposity. When compared to white Caucasian babies, small and thin Indian new-borns had poor muscle and visceral mass but higher adiposity.

2.5.1.2 Underweight and wasting

While a large proportion of children are extensively underweight in many parts of the developing world, far fewer countries face the burden of acute undernutrition in young women. Underweight, however, still remains a concern, but predominates only among women living in rural areas of the least developed countries (Mendez et al., 2005:719-720). Maternal underweight leads to an increased risk of intra-uterine growth retardation and increases the risk of LBW (Victora et al., 2008:346; Nannan et al., 2007:734). This is also associated with increased mortality risk in early childhood (Nannan et al., 2007:734).

In 1997, more than thirty percent of the world's children under five years of age were underweight (Darnton-Hill & Coyne, 1998:24). In 2002, sixty percent of deaths among children under five years in developing countries were associated with malnutrition (WHO/FAO, 2002:8).

Sixty percent of Korean children were found to be underweight in 1998, "the worst rate of any nation in the world at the time" (Schrimpton & Kachondham, 2003:4). In 2002, an international survey found that rates have been reduced to 21%. A Korean government funded survey in 2003, found rates of 28%. According to Schrimpton and Kachondham (2003:4), this indicates that food aid has indeed been well used in Korea.

The prevalence of underweight among Indian children younger than three years old was 49,1% in 1992, 43,8% in 1998, and 40,3% in 2005 (Subramanyam et al., 2010:3). In the Indian National Family Health Survey-3, 19% of children were wasted and 46% were underweight at three years of age in 2005/6 (Kanjilal et al., 2010:online; Rehman et al., 2009:online). About eight percent of three year-old South Indian urban boys resident in slum areas and seven percent of girls were wasted. Another 43% of boys and 39% of girls were underweight in 2005/6 (Rehman et al., 2009:online). Between 15% to 94% of children were underweight in different slum populations in Northern India around 2004 (Rehman et al., 2009:online). India has one of the highest prevalences of underweight among children younger than five (43% in 2006), surpassed only by Bangladesh, Yemen and Timor-Leste (Subramanyam et al., 2010:1). India contributes to about one-third of the global burden of underweight for children under five years old (Subramanyam et al., 2010:1). Subramanyam et al. (2010:1) state that during the "prosperous" 1990s there was a decline in the prevalence of underweight by about 0,9%

per annum in India, whereas in China, with its rapidly growing economy, the decline was about five percent per year.

2.5.2 In Africa and South Africa

2.5.2.1 In adults

In 1998, less than six percent of South African females and 13% of males were underweight (Steyn et al., 2006:34; DoH et al., 2002:244). At that time, men and women aged between 15 and 24 years were the most likely to be underweight, as well as those living in the province of the Northern Cape and of Asian/Indian descent. There was a higher prevalence of underweight among rural black men than in the other groups of men (DoH et al., 2002:244).

About 10,5% of deaths worldwide due to LBW can be attributed to maternal underweight. This amount is about three times higher than South African estimates, because of the lower prevalence of underweight among South African women (Nannan et al., 2007:737). The prevalence of underweight in adult males (12,9%) and females (5,6%) is however, much lower than the prevalence of overweight and obesity (Steyn et al., 2006:21). The WCRF/AICR (2007:9) state that the NFCS undertaken in 1999 found that in rural areas, adults from lower-income households were shorter and had a lower BMI.

2.5.2.2 In children

When looking at the UNICEF Conceptual Framework depicted in Figure 2.1, the immediate determinants of undernutrition (poor diet and infections) appear in South African children who lack sufficient intake of dietary energy, protein, and micronutrients (vitamin A, iron, zinc and calcium). Underweight children are at risk for infectious diseases, e.g. diarrhea and pneumonia, which increase their mortality risk. Undernutrition was found to contribute to fifty percent of deaths in children, with a direct relationship between the degree of underweight and mortality. The concomitant presence of both infection and underweight obviously increases the risk of mortality, with the duration of infection in the undernourished child playing an important role (Nannan et al., 2007:733). The Birth-to-Ten study found in 1991 that the children in peri-urban areas were at higher risk for undernutrition, especially if they were born in rural areas and migrated afterwards (Yach et al., 1991:218).

Mamabolo et al. (2005:online) state that, compared to younger Sub-Saharan African children, a greater proportion of younger South African children are stunted (21% to 48%) rather than underweight (8% to 15%).

Steyn et al. (2006:19) state that the results from the SAVACG study conducted in 1994 and the NFCS conducted in 1999 were similar for pre-school children, with underweight ranging from 6,9% to 10,7%, stunting from 16,1% to 27%, and wasting from 1,8% to 3,7%. Undernutrition was higher in the rural areas (Nannan et al., 2007:734; Steyn et al., 2006:19), with the highest prevalence of stunting and underweight found among the children aged between one and three years old (Steyn et al., 2005:11). The NFCS showed that among children aged between one and nine years old, about one in ten children were underweight, about one in five children were stunted and about three percent were wasted (Vorster & Kruger, 2007:323; Steyn et al., 2006:34; Steyn et al., 2005:8; Labadarios & Nel,

2000:180, 183). Thirty-five percent of children were suffering from some form of undernutrition (Vorster & Kruger, 2007:323; Steyn et al., 2006:34).

The NFCS further found that the prevalence of all undernourished children (stunted, underweight and wasted) was higher in all age groups in rural areas (Labadarios & Nel, 2000:176) and that children between one and three years old were more severely affected. They had about double the prevalence of stunting and underweight when compared with the other age groups (Labadarios & Nel, 2002:180).

(i) Stunting

According to the NFCS, the national prevalence of stunting among South African children one to nine years old was 19,3% in 1999, with the highest prevalence (24,4%) amongst the one to three year olds (Steyn et al., 2005:4, 8). Van der Merwe and Pepper (2006:4) state that stunting is more prevalent in rural areas in South Africa, and that it remains a significant problem in school-aged children. The Birth-to-Ten cohort study found that the prevalence of stunting was 19,5% by two years of age (Cameron, 2003:38). The 1999 NFCS found that stunting affected nearly one in five children. Urban children (17%) were less affected (WCRF/AICR, 2007:9; Labadarios et al., 2005:533). Nearly 26% of children on commercial farms (Steyn et al., 2005:4, 8) and almost 24% of rural children were affected by stunting (Labadarios et al., 2005:535. At the national level, stunting was prevalent in 19,3% of children (Steyn et al., 2005:8).

The Eastern Cape and Free State provinces were found to have the second highest percentage of stunted children in South Africa during the SAVACG study (De Villiers & Senekal, 2002:1232). In the NFCS, the Free State province had the second largest prevalence of stunting (30%). The prevalence of severe stunting was also second largest in the Free State (10%) (Labadarios & Nel, 2000:174).

In the secondary anthropometric data analysis of the NFCS, it was found that stunting decreased with age and the highest prevalence was among children aged one to three years (24,4%) (Steyn et al., 2005:8; Labadarios & Nel, 2000: 176). The prevalence of stunting was 25.5% in children aged one to three years old; 21% in those aged four to six years; and 13% in those aged seven to nine years (Labadarios et al., 2005:536; Labadarios & Nel, 2000:168). The Northern Cape had the highest prevalence of stunting (31%), followed by the Free State (30%) and Mpumalanga (26%) (DoH, 2001:1).

In 1995, the South African National Nutrition Survey Study (SANNSS) group conducted a systematic review of dietary surveys since 1979. Their results showed that black rural children between two and six years old had the lowest energy and macronutrient intake, probably due to low fat intake, which could explain the high prevalence of stunting in this group (Vorster et al., 1995, as referred to by Witten et al., 2002:online [unpublished]).

In the THUSA BANA study conducted on ten to 15 year old children in the North West province, stunting was most prevalent in rural areas (girls 23,7% and boys 26,7%). The prevalence in informal settlements was 26,4% for boys and 13,7% for girls. The lowest prevalence was in urban girls (11,6%) and boys (17,1%). About one-third of the children (35,8%) lived in rural areas, 17,8%

lived in informal settlements and 46,4% lived in urban areas. About half (51%) of the stunted girls and 43% of the stunted boys came from the rural areas (Mukuddem-Petersen & Kruger, 2004:online).

New legislation that was introduced in the early 1990s to improve the QoL of the "previously disadvantaged" (i.e. non-White South Africans), included free health care for children under five years of age. A positive trend has been seen when comparing data from a 1978 study (Richardson, 1978:246-249) with the Birth-To-Ten study data in that height for non-White girls has been improving since the 1970s. An increase of between five to ten centimeters has occurred during this period, which translates to about four to five centimeters per decade. However, there has been no change in height patterns in the White population for the same period (Cameron, 2003:37).

When comparing data from the NFCS with the SAVACG, the national average for prevalence of stunting has not changed remarkably, but a slight increase was noted in urban children (Labadarios & Nel, 2000:178).

(ii) Underweight

Poverty, hunger and poor household food security are among the main factors contributing to high rates of childhood underweight, especially in rural areas (Nannan et al., 2007:738).

Nannan et al. (2007:733) state that 11,8% of children under five years old were underweight, which contributed to 12,3% of deaths. This amount is smaller than the global maternal and child underweight risk factor assessment finding of 34,7%. The prevalence of underweight in South Africa is lower than in many other developing countries and a high proportion of child deaths in South Africa are due to HIV/AIDS (35,1%) (Nannan et al., 2007:737). Protein-energy malnutrition attributed to 44,7% of the total burden of disease (Nannan et al., 2007:733, 736).

According to the NFCS, the prevalence of underweight in children was one in ten at the national level (Labadarios et al., 2005:533; Steyn et al., 2005:8). The highest prevalence was in one to three year olds (11,4%), in rural areas (10,9%) and particularly among children on commercial farms (13,7%) (Steyn et al., 2005:8). The WCRF/AICR (2007:9) state that the 1999 NFCS that ten percent of children between one and three years old consumed less than half of their recommended daily needs and another 26% consumed less than two-thirds. The prevalence of underweight decreased with age (Labadarios & Nel, 2000:176) from 13% in the children aged one to three years to eight percent in the children aged seven to nine years (Labadarios & Nel, 2000:168). Less than 1,5% of children were severely underweight (<-3SD), except on commercial farms where the prevalence was five percent (Labadarios et al., 2005:536). The Free State province had the fourth highest prevalence of underweight (14%) (Labadarios & Nel, 2000:174).

When comparing data from the NFCS with the SAVACG, the prevalence of underweight increased in rural children (especially in the Northern- and Eastern Cape) between 1994 and 1999 (Labadarios & Nel, 2000:178)

(iii) Wasting

The NFCS found that one in twenty children living in rural areas were wasted (Labadarios et al., 2005:536). At the national level 3,3% of children were wasted (Steyn et al., 2005:8) and one percent were severely wasted (<-3SD) (Labadarios et al., 2005:536). The prevalence of wasting remained constant in all age groups (Labadarios & Nel, 2000:169).

When comparing data from the NFCS with the SAVACG, the prevalence of wasting decreased in the Eastern Cape and the Free State (Labadarios & Nel, 2000:178)

2.6 Chronic diseases of lifestyle

CDLs are largely preventable diseases (WHO/FAO, 2002:5). The growing epidemic of CDLs is mostly related to dietary and lifestyle changes (WHO/FAO, 2002:1). The transition to CDLs is due to a greater intake of saturated fat and energy and a higher prevalence of other risk factors (Darnton-Hill & Coyne, 1998:25), such as: high blood pressure; dyslipidemia (including hypercholesterolemia); overweight and obesity; physical inactivity; tobacco use; and inadequate fruit and vegetable intake (Steyn et al., 2006:6). Vorster (2002:242) states that the genetic expression of CDLs is influenced by a combination of certain environmental factors, of which nutrition forms a part. CDLs are becoming an ever-increasing significant cause of disability and premature mortality, both in developing- and newly developed countries. As the prevalence of CDLs increases, additional burdens are placed on already overtaxed national health care budgets (WHO/FAO, 2002:2).

From the second half of the twentieth century, major shifts in disease patterns occurred in developed countries due to increased life expectancy and changes in diets and lifestyles, which contributed to an epidemic of CDLs. This occurrence is now also becoming apparent in developing countries (WHO/FAO, 2002:81).

Compared to other industrialized countries, the Republic of Korea has lower rates of CDLs and lower than expected levels of fat intake and obesity prevalence. According to WHO/FAO (2002:7), this can be explained by the continued intake of a traditional high-vegetable diet despite major social and economic changes.

In this section, the prevalence and epidemiology of CDLs, both globally and South Africa, will be discussed, followed by a discussion of specific CDLs.

2.6.1 Global prevalence and epidemiology

In developed countries, low socio-economic status is associated with higher risk of CVD and DM. However, in industrialized countries like China, an increased prevalence of CVD among the higher socio-economic groups has become apparent (WHO/FAO, 2002:39). The WHO/FAO (2002:39) anticipates that CVD will progressively shift to the more disadvantaged sectors of society. In fact, there is some evidence that this is already happening, especially in women of low-income groups in Brazil and South Africa (WHO/FAO, 2002:39).

Because type 2 DM and cancer peaks in older persons, the main burden of CDLs is observed in the aging or older population (WHO/FAO, 2002:40).

In 2002, infectious diseases caused forty percent of deaths worldwide, while CDLs accounted for sixty percent of all deaths. Also in 2002, international donors allocated US$2,9 billion (R23,2 billion) to infectious diseases, while only US$0,1 billion (R0,8 billion) were allocated to CDLs (World Diabetes Foundation (WDF), 2009:online). Age-adjusted mortality rates are 1,5 to 2,5 times higher among people with DM than in the general population. Much of the excess mortality in the white population can be attributed to CVD, especially CHD. Renal disease is a major contributor among Asians and American-Indians. However, infections are also an important contributor to mortality in some developing nations (WHO/FAO, 2002:73).

The leading causes of death by income group in 2004, as found by the WHO, are set out in Table 2.5 (WHO, 2008:12). Note that the amount of CDLs listed under the top ten for each type of country increased as the income of the countries increased. Also note the contribution of prematurity and LBW to deaths, especially in low-income countries, and the contribution of cancer in high-income countries.

The World Health Report of 2002 has identified ten risk factors that account for more than a third of all the deaths worldwide. They are: unsafe sex; alcohol consumption; tobacco consumption; obesity; hypertension; undernutrition; unsafe water; sanitation and hygiene; iron deficiency; indoor smoke from solid fuels; and hypercholesterolemia (Bradshaw et al., 2003:58).

Table 2.5. Leading causes of death by income group in 2004 (WHO, 2008:12 – emphasis added).

Disease or injury	% of total deaths	Disease or injury	% of total deaths
WORLD		**LOW-INCOME COUNTRIES**	
1. Ischemic heart disease	***12,2***	1. Lower respiratory infections	11,2
2. Cerebrovascular disease/stroke	***9,7***	***2. Ischemic heart disease***	***9,4***
3. Lower respiratory infections	7,1	3. Diarrheal diseases	6,9
4. COPD	5,1	4. HIV/AIDS	5,7
5. Diarrheal diseases	3,7	***5. Cerebrovascular disease/stroke***	***5,6***
6. HIV/AIDS	3,5	6. COPD	3,6
7. Tuberculosis	2,5	7. Tuberculosis	3,5
8. Trachea, bronchus, lung cancers	***2,3***	8. Neonatal infections	3,4
9. Road traffic accidents	2,2	9. Malaria	3,3
10. Prematurity and low birth weight	2,0	10. Prematurity and low birth weight	3,2
MIDDLE-INCOME COUNTRIES		**HIGH-INCOME COUNTRIES**	
1. Cerebrovascular disease/stroke	***14,2***	***1. Ischemic heart disease***	***16,3***
2. Ischemic heart disease	***13,9***	***2. Cerebrovascular disease/stroke***	***9,3***
3. COPD	7,4	***3. Trachea, bronchus, lung cancers***	***5,9***
4. Lower respiratory infections	3,8	4. Lower respiratory infections	3,8
5. Trachea, bronchus, lung cancers	***2,9***	5. COPD	3,5
6. Road traffic accidents	2,8	6. Alzheimer and other dementias	3,4
7. Hypertensive heart disease	***2,5***	***7. Colon and rectal cancers***	***3,3***
8. Stomach cancer	***2,2***	***8. Diabetes mellitus***	***2,8***
9. Tuberculosis	2,2	***9. Breast cancer***	***2,0***
10. Diabetes mellitus	***2,1***	***10. Stomach cancer***	***1,8***

Many CDLs play significant roles in both morbidity- and mortality; particularly IHD, hypertension, stroke, DM, lung-, esophageal-, breast- and colorectal cancers (Steyn et al., 2006:5). The mortality rate due to CDLs is highest in Europe (WHO, 2008:17). Mortality rates due to CDLs are second highest in Africa, followed by the Eastern Mediterranean and South-East Asia (WHO, 2008:17). In 2001, CDLs accounted for sixty percent of the estimated 56 million deaths globally and 47% of the global burden of disease

(Steyn et al., 2006:6). CDLs dominate the disease burden in high-income countries, which represents the older population, because CDL risk increases with age (WHO, 2008:48). Because most countries have an increase in life expectancy, larger proportions of their populations move into the age range where CDLs become the major cause of ill health and death (Darnton-Hill & Coyne, 1998:25). In many developing countries, population ageing and changes in the distribution of risk factors have accelerated the CDLs' share of disease burden (WHO, 2008:47). IHD and stroke are the largest sources of the burden of disease in low- and middle-income countries, especially in Europe, where CVD account for more than 25% of the total burden of disease (WHO, 2008:40).

Globally, the estimated prevalence of DM increased from 191 million in 2002, to 220,5 million in 2004, and there were 30,7 million stroke survivors in 2004 (WHO, 2008:112; WHO, 2008:32, Table 7). The estimated global prevalence of *angina pectoris* increased from 25 million in 2002, to 54 million in 2004 (WHO, 2008:114). In China, the prevalence rate of DM has risen by 0,1% every year since 1980 (when it was 0,67%) (Darnton-Hill & Coyne, 1998:25).

Hyperinsulinemia, hypertriglyceridemia, hypertension and central adiposity (all part of the metabolic syndrome) are becoming more common in developed countries (Darnton-Hill & Coyne, 1998:26).

Van der Merwe and Pepper (2006:4) state that the effect of ethnicity on obesity prevalence and CDLs has been studied in many parts of the world. In the USA, African-Americans, and particularly women, have the highest incidence of obesity, hypertension, IHD, and stroke. Compared to other ethnic groups in the USA, obese African-Americans have a "less atherogenic" lipid profile; as well as a lower risk for DM when compared to native American-Indians, but a higher risk than Caucasians (Van der Merwe & Pepper, 2006:4).

If compared to Caucasian women with a similar BMI and WHR, African-American women have lower insulin sensitivity. Black women also have less visceral fat (similar to South African women), but subcutaneous abdominal fat is significantly correlated with reduced insulin sensitivity and fasting insulin in African-American women, which may explain the high rate of DM seen in the African-American population (Van der Merwe & Pepper, 2006:4; Goedecke et al., 2005:68). White American women had higher total cholesterol, LDL and triglycerides, consistent with higher levels of visceral fat (Goedecke et al., 2005:68).

Nearly two-thirds of all deaths for ages 15 to 59 years in low- and middle-income countries are associated with CVD, cancer and other CDLs (WHO, 2008:17). In most countries, CVD is the leading cause of morbidity and premature death. This is followed by cancer in developed countries. The increase in DM also represents a global epidemic. In over three-quarters of countries, at least three to five leading causes of death are now CDLs (Darnton-Hill & Coyne, 1998:25). In industrialized countries, CVD incidence has declined, but less so in lower socio-economic countries (Darnton-Hill & Coyne, 1998:23).

Native American males and females have BMIs consistently and considerably greater than in the general American population, at each year of age from five to 18 years. Socio-economic status and ethnicity may have an impact on the prevalence of CVD and some cancers, as seen in the Native Americans and African-Americans of the USA, who have far higher levels of obesity and DM and/or hypertension. African-American females below the poverty level have a hypertension rate of forty percent, compared to African-American females above the poverty level (30%) and white American females above the poverty

level (22%). This pattern is also seen in the Maori and Aboriginal populations of New Zealand and Australia (Darnton-Hill & Coyne, 1998:25). Cardiovascular diseases are more numerous in China and India than in all economically developed countries of the world combined (WHO/FAO, 2002:5).

Recent evidence shows that CDLs are not limited to higher socio-economic groups in low and middle-income countries, but that unhealthy behaviours associated with these diseases, and the CDLs themselves, are also now becoming increasingly prevalent in poor communities in developing countries (Steyn et al., 2006:6). The gap between the rich and poor is widening, even within both developed and developing countries. Australia, Sweden, the United Kingdom (UK) and the USA all have seen the range between rich and poor widen over the last two decades (Darnton-Hill & Coyne, 1998:23). This growing gap leads to increased poor nutrition and disease among less affluent sectors in all countries. In wealthier nations, the less advantaged are more likely to suffer from obesity and CDLs and increased adult mortality. In less affluent nations, the poor have more food insecurity and suffer more from undernutrition and premature death (Darnton-Hill & Coyne, 1998:24). Many countries with an epidemiological picture similar to industrialized countries (many in the Pacific, South-East and East Asia and Eastern Europe), have an increasing prevalence of CDLs, with a higher frequency in urban areas and decline in CVD is not seen. In many of the countries of the former Union of Soviet Socialist Republics (USSR), an increase in CDL-related deaths has been seen (Darnton-Hill & Coyne, 1998:25).

One-fifth of all Group I deaths in adults aged 15 to 59 years, resulting from infectious and parasitic diseases and maternal and nutritional conditions, occur in South-East Asia (29%) and Africa (62%). This also includes 35% of adult deaths due to HIV/AIDS (WHO, 2008:17). Group I conditions largely occur in poorer populations, and decline as the population ages. At older ages, there is a greater occurrence of Group II (CDL) mortalities (WHO, 2008:8). About six in ten deaths globally in both men and women are due to CDLs (WHO, 2008:8, 40, 47). Lung cancer is globally the most common cancer, followed by breast cancer, colorectal cancer and stomach cancer. Lung cancer is also the leading cancer in the Western Pacific Region, but is less common than the other cancers in most other regions. Cervix cancer has the highest incidence in Africa and South-East Asia (WHO, 2008:29).

Table 2.6. Percentage of CDL-related deaths per country (WCRF/AICR, 2007:6, 10, 14, 16, 18, 20, 22, 24, 26-28).

	CVD	Cancer	Diabetes	Respiratory disease	Other
Australia	39	35	3	7	17
Brazil	48	20	6	11	15
China	44	22	2	22	10
Egypt	58	9	2	7	24
India	57	15	3	12	13
Japan	37	44	2	6	14
Mexico	31	18	16	8	28
Poland	55	30	2	3	11
South Africa	51	20	7	10	13
Spain	35	33	3	10	19
UK	42	33	1	8	16
USA	41	29	4	8	18
Average	***44,8***	***25,7***	***4,3***	***9,3***	***16.5***

Table 2.6 summarizes the data from the WHO, which reflects the percentages of deaths caused by certain CDLs. As can be seen, the five countries with the highest percentage of CVD-related deaths were Egypt (58%), India (57%), Poland (55%), South Africa (51%) and Brazil (48%). The five countries with the highest percentage of cancer-related deaths were Japan (44%), Australia (35%), Spain (33%), UK (33%)

and Poland (30%). The four countries with the highest percentage of DM-related deaths were Mexico (16%), South Africa (7%), Brazil (6%), and USA (4%). The five countries with the highest deaths rates due to respiratory disease were China (22%), India (12%), Brazil (11%), South Africa (10%) and Spain (10%).

Table 2.7 summarizes the estimated DALYs per 100 000 for WHO world regions, including South Africa. It should be interpreted with caution, since the separate amounts cannot be interpreted as a percentage of the total for all causes. When looking at non-communicable diseases (i.e. CDLs), South East Asia leads the pack with the highest amount of DALYs, followed by Europe, America, and Eastern Mediterranean. They are followed by Africa, South Africa and, lastly, Western Pacific. Comparing this with malignant neoplasms, South East Asia is again in the lead, followed by Western Pacific, America and South Africa. Africa, Europe and the Eastern Mediterranean have lower DALYs related to cancer. For DM interestingly, America is in the lead, with South Africa following close behind in second place, then South East Asia, Eastern Mediterranean and Europe. With CVD the picture looks different again, with South East Asia leading once again, followed by Europe, Eastern Mediterranean and then South Africa.

Table 2.7 Estimated DALYs per 100 000 for WHO world regions, including South Africa, 2000
(Norman et al., 2010:online, Table 1)

Cause	Africa	America	Eastern Mediterranean	South East Asia	Europe	Western Pacific	South Africa
All causes	55 233	17 206	27 688	17 527	27 676	15 722	34 309
Communicable, maternal perinatal and nutritional deficiencies	40 412	3 258	13 105	1 897	11 971	3 638	19 044
Infectious and parasitic	28 539	1 493	6 527	781	5 625	1 223	14 547
HIV/AIDS	11 358	286	371	87	747	80	11 241
Respiratory infections	4 852	496	2 367	445	2 114	875	1 036
Maternal conditions	2 013	234	871	179	724	165	225
Perinatal conditions	3 476	671	2 334	329	2 455	723	2 341
Nutritional deficiencies	1 532	364	1 006	163	1 053	651	895
Non-communicable	10 202	11 660	11 587	13 031	12 173	9 553	9 740
Malignant neoplasms	1 045	1 278	747	2 020	1 013	1 449	1 155
Other neoplasms	12	29	20	32	32	13	31
Diabetes mellitus	113	421	276	288	264	168	390
Endocrine disorders	253	276	226	110	57	73	166
Neuropsychiatric conditions	2 417	4 065	2 971	3 558	3 055	2 392	1 611
Sense organ diseases	895	536	763	475	861	383	554
Cardiovascular diseases	1 797	1 941	2 728	3 821	3 023	1 806	2 328
Respiratory diseases	1 199	980	983	734	1 088	1 488	1 618
Digestive diseases	979	741	949	811	978	581	846
Genitourinary diseases	430	226	378	212	267	207	332
Skin diseases	101	26	35	22	30	11	32
Musculoskeletal diseases	290	531	389	607	521	504	101
Congenital anomalies	592	454	903	216	832	375	493
Oral conditions	79	157	218	124	151	104	47
Injuries	4 619	2 288	2 995	2 599	3 532	2 531	5 524
Unintentional injuries	3 084	1 444	2 347	1 786	2 910	1 969	2 838
Intentional injuries	1 535	844	648	813	622	562	2 686

2.6.2 Prevalence and epidemiology in South Africa

As far back as 1977, it was found that obesity in South Africa was the most common and earliest to appear of all CDLs, and it was particularly prevalent and severe among women (Seftel, 1977:122). Around that time it was found that the “fattest women in the world” were to be found in Soweto and the backyards of Houghton. It was speculated that obesity was partly related to decreased physical activity,

but also to the perennial food supply in the city as well as status connected to being overweight. Already at that time it was noted that DM emerged frequently after two or three decades of being overweight. It was also noted that DM was relatively uncommon among undernourished rural dwellers. Around 1977, myocardial infarction (MI) only just started to appear among black urban dwellers. It was uncommon, but increasing (Seftel, 1977:122). By 1983, MI accounted for 12% of cardiac-related diseases in urban blacks (O'Keefe, et al., 1983:679)

It was also noted in 1977 that the black urban population with MI were considerably Westernized, indicated by their diet, occupation, degree of physical activity and the high prevalence of obesity, DM, hypertension and hypercholesterolemia. At that time, hypertension was, after violence, the next important cause of deaths in urban blacks in Johannesburg. In contrast, severe hypertension was rare in rural blacks. Even then, hypertension in black rural South Africans were compared to their counterparts in America, where hypertension has long been a major public health problem and more common than in white Americans. Even in 1977, a state of transition among urban black South Africans could already be seen (Seftel, 1977:122-123).

The MRC Technical Report states that the presence of obesity, a sedentary lifestyle, and the use of tobacco and alcohol all explain the increased prevalence of CVD, DM, hypertension, dyslipidemia and certain cancers (Steyn et al., 2006:5). It is now known that the black population in South Africa is vulnerable to hypertension, stroke and DM (Vorster et al., 2005:online). Urbanization was also found to be an independent predictor of hypertension or DM (Steyn, 2005b:250).

Obesity, excessive alcohol use, and a Western diet also relate to higher than expected rates of DM, especially in the black population; and abnormal dyslipidemia, especially in the white and Indian population (Steyn, 2005b:250). Elevated free fatty acids in the black population predispose the obese to type 2 DM (Kruger et al., 2005:491). In 2000, HIV/AIDS accounted for 39% of deaths, while CDLs accounted for 38% of deaths (Seedat, 2007:318). The revised burden of disease estimates showed a marked difference in proportion of deaths between genders for CDLs (37,1% for males; 44,9% for females) (Norman et al., 2006:9). When looking at actuarial models of projection of AIDS- and CDL mortality for 2010, the contribution of CDLs to the burden of disease in South Africa cannot be ignored despite increasing rates of AIDS. It is projected that in 2025, one in ten South Africans will be sixty years or older. This will also lead to an increased burden of CDLs (Steyn, 2005b:249).

The control of hypertension, DM, obesity and other risk factors for CVD in Sub-Saharan Africa is impaired by social, economic and cultural factors (Seedat, 2007:316). In 2004, the prevalence of DM in Africa was 9,7 million and there were 1,6 million stroke survivors (WHO, 2008:32).

Although the proportion of deaths from CDLs in people aged 35 to 64 had decreased from 28,5% in 1988 to twenty percent in 2002 (because of the high number of AIDS- related deaths) , the rate of CVD deaths in all populations groups remains high. Stroke and IHD accounted for 30,6% of deaths in men and 34,9% of deaths in women older than 65 years. In the group 15 to 44 years old, 2,4% of deaths in men are attributed to stroke and IHD, while 1,4% in women are stroke-related (Vorster & Kruger, 2007:323). According to the MRC Technical Report, CDLs accounted for 37% of all deaths in 2000, whereof CVD and DM accounted together for 19% and cancers for 7,5% (Steyn et al., 2006:6). CDLs accounted for

forty percent of female deaths and 36% of male deaths. Stroke was the most common CDL-related cause of death in women, and IHD in men. Hypertensive heart disease, DM and COPD were among the leading causes of fatal CDLs in 2000 (Steyn et al., 2006:12). DM mortality increased in all ethnic groups, mostly in blacks (Steyn et al., 2006:26). IHD is particularly high for the white and Indian population (Steyn et al., 2006:28). Stroke is a major public health problem amongst black South Africans (Kruger et al., 2005:494). In the 1998 SADHS, 13% of men and 16% of women reported a family history of IHD. Frequency was also higher in urban areas (DoH et al., 2002:168).

In 2000, type 2 DM accounted for the highest number of deaths and DALYs in women, followed by hypertension. IHD accounted for the most male deaths and DALYs, followed by type 2 DM and hypertension. Cancer contributed for 4,4% of the total burden in females and 1,1% in males (Joubert et al., 2007:686).

As Table 2.7 shows (estimated DALYs per 100 000 for WHO world regions), South Africa is quite low on the ranks for total DALYs from CDLs, but rankings are higher for cancer and CVD (both ranked fourth). South Africa has the highest DALYs for DM, second only to America Norman et al., 2010:online, Table 1).

Table 2.8. Deaths attributable to 17 selected risk factors compared with underlying causes of death (Norman et al., 2007c:638, Table I).

Rank	Risk factor	% total deaths	Rank	Disease, injury or condition	% total deaths
1	Unsafe sex/STIs	26.3	1	HIV/AIDS	25.5
2	High blood pressure	9.0	2	Ischemic heart disease	6.6
3	Tobacco smoking	8,5	3	Stroke	6.5
4	Alcohol harm	7.1	4	Tuberculosis	5.5
5	High BMI (excess body weight)	7.0	5	Interpersonal violence injury	5.3
6	Interpersonal violence (risk factor)	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	Diarrheal diseases	3.1
9	Physical inactivity	3.3	9	Road traffic injury	3.1
10	Low fruit and vegetable intake	3.2	10	Diabetes mellitus	2.6
11	Unsafe water, sanitation and hygiene	2.6	11	Chronic obstructive pulmonary disease	2.5
12	Childhood and maternal underweight	2.3	12	Low birth weight	2.2
13	Urban air pollution	0.9	13	Asthma	1.3
14	Vitamin A efficiency	0.6	14	Trachea/bronchi/lung cancer	1.3
15	Indoor air pollution	0.5	15	Nephritis/nephrosis	1.3
16	Iron deficiency anemia	0.4	16	Septicemia	1.2
17	Lead exposure	0.3	17	Esophageal cancer	1.1

The aim of the MRC's Comparative Risk Assessment study was to estimate the contributions of 17 selected risk factors to the burden of disease in South Africa in 2000. The study followed the WHO's standardized comparative risk assessment methodology (Norman et al., 2006:2). The 17 selected risk factors were identified based on the burden of disease experienced in South Africa, as well as on the input from various stakeholders, including the Department of Health (both provincially and nationally). The risk factors were chosen based on the following criteria: (i) likely to be among the leading causes of burden of disease and injury; (ii) evidence of causality; (iii) being potentially modifiable; and (iv) availability of data (Norman et al., 2007c:637). The results are set out in the Tables below as: deaths attributable to 17 selected risk factors compared with underlying causes of death (Table 2.8); and DALYs of the 17 risk factors compared to the DALYs of underlying causes of death (Table 2.9).

When looking again at Table 2.7., South Africa has similar DALY rates for HIV/AIDs when compared with the rest of Africa, but lower rates for other communicable, maternal, perinatal and nutritional conditions.

Its burdens due to CVD, respiratory diseases and DM are higher than the rest of Africa (Norman et al., 2010:online).

Table 2.9. DALYs attributed to 17 selected risk factors compared with the underlying causes of DALYs (Norman et al., 2007c:638, Table II).

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 (excess body weight)	2.9	5	Diarrheal diseases	2.9
6	Childhood and maternal underweight	2.7	6	Lower respiratory infections	2.8
7	Unsafe water sanitation and 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	Ischemic heart disease	1.8
12	Physical inactivity	1.1	12	Protein-energy malnutrition	1.3
13	Iron deficiency anemia	1.1	13	Birth asphyxia and birth trauma	1.2
14	Vitamin A deficiency	0.7	14	Diabetes mellitus	1.1
15	Indoor air pollution	0.4	15	Alcohol dependence	1.0
16	Lead exposure	0.4	16	Hearing loss, adult onset	1.0
17	Urban air pollution	0.3	17	Cataracts	0.9

Bradshaw et al. (2003:v) ranked the top twenty specific causes of premature mortality burden in years of life lost (YLL) under South Africans in 2000, as set out by Table 2.10. LBW was ranked fifth among females (3,3%) and seventh among males (3,3%). IHD ranked eighth in males (2,7%) and stroke ninth (2,3%). In females, stroke ranked sixth (3,1%) and IHD ninth (2,0%). DM ranked tenth among females (1,6%) and 15th among males (0,9%). PEM ranked 11th for males (1,4%) and 12th for females (1,4%). It appears that CDLs are only contributing to a small burden, whereas if all the percentages are added up for stroke, IHD, DM, hypertensive heart disease, and COPD, 8,3% of YLLs were attributed to CDLs.

Table 2.10 Top twenty specific causes of premature mortality burden (YLLs) by sex (Bradshaw et al., 2003:vii, Table 3 and Bradshaw et al., 2003:35, Table 3.2).

Males Rank	Cause of death	YLL	%	Females Rank	Cause of death	YLL	%	Persons Rank	Cause of death	YLL	%
1	HIV/AIDS	2 148 080	32.9	1	HIV/AIDS	2 517 330	46.3	1	HIV/AIDS	4 665 410	39.0
2	Homicide/violence	756 483	11.6	2	Diarrheal diseases	216 488	4.0	2	Homicide/violence	902 592	7.5
3	Tuberculosis	380 789	5.8	3	Tuberculosis	214 488	3.9	3	Tuberculosis	585 277	5.0
4	Road traffic accidents	344 868	5.3	4	Lower respiratory infections	209 240	3.8	4	Road traffic accidents	489 979	4.1
5	Lower respiratory infections	239 770	3.7	5	Low birth weight	180 274	3.3	5	Diarrheal disease	452 827	3.8
6	Diarrheal diseases	236 339	3.6	6	Stroke	140 097	3.1	6	Lower respiratory infections	449 010	3.8
7	Low birth weight	213 489	3.3	7	Homicide/violence	146 109	25.7	7	Low birth weight	393 763	3.3
8	Ischemic heart disease	175 906	2.7	8	Road traffic accidents	145 111	2.7	8	Stroke	318 083	2.7
9	Stoke	147 986	2.3	9	Ischemic heart disease	108 531	2.0	9	Ischemic heart disease	284 438	2.4
10	Suicide	123 822	1.9	10	Diabetes mellitus	86 154	1.6	10	Protein-energy malnutrition	171 433	1.4
11	Protein-energy malnutrition	93 556	1.4	11	Hypertensive heart disease	79 112	1.5	11	Suicide	163 544	1.4
12	COPD	74 459	1.1	12	Protein-energy malnutrition	77 877	1.4	12	Diabetes mellitus	145 421	1.2
13	Fires	70 535	1.1	13	Septicemia	55 808	1.0	13	Hypertensive heart disease	127 066	1.1
14	Septicemia	59 439	0.9	14	Fires	52 866	1.0	14	Fires	123 400	1.0
15	Diabetes mellitus	59 267	0.9	15	Cervix cancer	50027	0.9	15	Septicemia	115 247	1.0
16	Cirrhosis of liver	57 408	0.9	16	Neonatal infections	43 937	0.8	16	COPD	113 499	0.9
17	Trachea/bronchi/lung cancer	54 934	0.8	17	Asthma	43 037	0.8	17	Neonatal infections	06 819	0.8
18	Bacterial meningitis	54 876	0.8	18	Nephritis/nephrosis	43 025	0.8	18	Asthma	94 069	0.8
19	Neonatal infections	52 882	0.8	19	Suicide	39 721	0.7	19	Nephritis/nephrosis	93 973	0.8
20	Asthma	21 032	0.8	20	COPD	39 041	0.7	20	Bacterial meningitis	90 964	0.8
	All causes	**6 529 811**			**Al causes**	**5 438 011**			**All causes**	**11 967 822**	

When comparing the proportion of deaths due to HIV/AIDs and CDLs in South Africa with the YLLs, as set out by Table 2.11, it can be seen that the number of deaths attributable to CDLs is larger than the HIV/AIDS-related deaths, but when looking at YLLs, the years of life lost for HIV/AIDS are almost double that of CDLs in all groups, but more so in the female group. This occurrence can be explained by age distribution, where a large proportion of HIV/AIDS deaths occur in young adults and children younger than five. The decrease in the amount of YLLs related to CDLs therefore shows that these diseases occur in older age groups, where life expectancy diminishes (Bradshaw, et al., 2003:36)

Table 2.11 Comparison of the proportions of deaths and YLLs due to CDLs and HIV/AIDS
(Bradshaw et al., 2003:36, Table 3.3 - amended)

Group	Deaths	%	YLLs	%
Persons	CDLs	37	CDLs	21
	HIV/AIDS	30	HIV/AIDS	38
Male	CDLs	36	CDLs	20
	HIV/AIDS	26	HIV/AIDS	33
Female	CDLs	40	CDLs	21
	HIV/AIDS	34	HIV/AIDS	47

As can be seen from Table 2.12, the Revised Burden of Disease Estimates show that CDLs were actually responsible for 41% of all deaths in 2000 in South Africa (Norman et al., 2006:11) and not 37% as initially found by Bradshaw et al., 2003:36, Table 3.3).

Table 2.12 Initial and revised estimated number of deaths in each group and HIV/AIDS
(Norman et al., 2006:11, Table 4).

Group	Initial		Revised	
	No. of deaths	%	No. of deaths	%
Group I (HIV/AIDS)	165 859	29.8	132 990	25.5
Group I (Other)*	115 565	20.8	115 562	22.2
Group II (CDLs)	206 231	37.0	212 595	40.8
Group III (Injuries)	68 930	12.4	59 935	11.5
TOTALS	**556 686**	**100.0**	**521 082**	**100.0**

*Group I = communicable diseases, maternal causes, perinatal conditions, and nutritional deficiencies

As can be seen from Table 2.13, when comparing the top twenty leading causes of deaths found in the South African Burden of Disease (SABD) study with the WHO's estimates, the WHO's estimate for HIV/AIDS is almost double that of the South African study (46,9% vs. 25,5%). The top five causes were similar, with only slight differences in ranking for IHD, cerebrovascular disease and interpersonal violence. Stroke is ranked second by the WHO estimates, but third by the SABD, while IHD is ranked fifth by the WHO and second by the SABD. Road traffic injuries were ranked sixth by the WHO and ninth by the SABD (Norman et al., 2006:22).

Table 2.13 Cause of death estimates of South African Burden of Disease study vs. WHO
Norman et al., 2006:22, Table 9)

	SA NBD estimates 2000		*WHO estimates 2000*	
Rank	**Persons**	**% total deaths**	**Persons**	**% total deaths**
1	HIV/AIDS	25.5	HIV/AIDS	46.9
2	Ischemic heart disease	6.6	Stroke	3.5
3	Stroke	6.5	Tuberculosis	3.4
4	Tuberculosis	5.5	Interpersonal violence	3.1
5	Interpersonal violence	5.3	Ischemic heart disease	3.0
6	Lower respiratory infections	4.4	Road traffic accidents	2.4
7	Hypertensive disease	3.2	Diarrheal diseases	2.2
8	Diarrheal diseases	3.1	Lower respiratory infections	2.1
9	Road traffic accidents	3.1	Diabetes mellitus	2.1
10	Diabetes mellitus	2.6	COPD	1.3
11	COPD	2.5	Perinatal conditions	1.3
12	Low birth weight	2.2	Trachea, bronchus, lung cancers	0.9
13	Asthma	1.3	Nephritis and nephrosis	0.8
14	Trachea/bronchi/lung cancer	1.3	Esophageal cancer	0.7
15	Nephritis/nephrosis	1.3	Asthma	0.7
16	Septicemia	1.2	Hypertensive disease	0.6
17	Esophageal cancer	1.1	Self0inflicted injuries	0.6
18	Protein-energy malnutrition	1.1	Protein-energy malnutrition	0.5
19	Suicide	1.0	Cervix uteri cancer	0.4
20	Cirrhosis of liver	1.0	Congenital anomalies	0.4

When comparing the DALYs estimates by the SABD and WHO, as set out in Table 2.14, it can be seen that the first six conditions rank the same. Stroke, however, ranks 9th in the SABD but 10th in the WHO. Again, the WHO estimated HIV/AIDS much higher at 46,5% of DALYs compared to the SABD's

estimate if 30,9% (Norman et al., 2006:23). This may be due to the WHO using the UNAIDS HIV death estimates rather than the ASSA2002 estimates (Norman et al., 2006:26).

When looking at Table 2.15 it can be seen that CVD is the leading cause of CDL-related deaths in all South African population groups, but is highest amongst South African Asians, followed by the Coloured population. Malignant neoplasms are the second leading cause of CDL-related deaths, and are most common among the Coloured population, followed by the White population. Respiratory disease is ranked third, being more common under the Coloured and Black population groups. DM, ranked fourth, is far more common amongst Asians, followed much lower by Coloured and African population groups (Norman et al., 2006:25)..

Table 2.14 Estimated DALYs – SABD vs. WHO (Norman et al., 2006:23, Table 10).

	SA NBD estimates 2000		*WHO estimates 2000*	
Rank	**Persons**	**% total DALYs**	**Persons**	**% total DALYs**
1	HIV/AIDS	30.9	HIV/AIDS	46.5
2	Interpersonal violence	6.5	Interpersonal violence	4.0
3	Tuberculosis	3.7	Tuberculosis	2.8
4	Road traffic accidents	3.0	Road traffic accidents	2.6
5	Diarrheal diseases	2.9	Diarrheal diseases	2.5
6	Lower respiratory infections	2.8	Lower respiratory infections	1.8
7	Low birth weight	2.6	Unipolar depressive disorders	1.6
8	Asthma	2.2	Perinatal conditions	1.6
9	Stroke	2.2	Asthma	1.1
10	Unipolar depressive disorders	2.0	Stroke	1.0
11	Ischemic heart disease	1.8	Hearing loss, adult onset	0.8
12	Protein-energy malnutrition	1.3	Ischemic heart disease	0.8
13	Birth asphyxia and birth trauma	1.2	Congenital anomalies	0.8
14	Diabetes mellitus	1.1	Protein-energy malnutrition	0.8
15	Alcohol dependence	1.0	Diabetes mellitus	0.7
16	Hearing loss, adult onset	1.0	Cataracts	0.7
17	Cataracts	0.9	Lymphatic giardiasis	0.7
18	Hypertensive heart disease	0.9	Trachoma	0.6
19	Fires	0.9	Bipolar disorder	0.6
20	Falls	0.9	Schizophrenia	0.6

Norman et al. (2006:26) state that the revised estimates of the burden of disease in South Africa confirm the observed quadruple burden of disease and the need to respond to the diseases related to (1) poverty and under-development; (2) CDLs, (3) injuries; and (4) HIV/AIDS.

Table 2.15 Age standardized mortality rates for CDLs in South Africa, by population group (Norman et al., 2006:25, Table 12)

	African (black)	**White**	**Asian**	**Coloured**	**South Africa**
Malignant neoplasms	126.0	198.9	121.4	212.5	148.6
Benign neoplasms	2.2	3.0	1.6	1.4	2.3
Diabetes mellitus	58.5	23.0	111.4	64.0	49.4
Endocrine and metabolic	8.1	2.6	5.8	4.1	6.5
Neuropsychiatric conditions	21.9	25.4	21.6	18.0	23.7
Sense organs	0.0	0.0	0.0	0.0	0.0
Cardiovascular disease	375.3	384.4	606.9	406.2	360.5
Respiratory disease	92.8	69.9	64.2	103.1	83.3
Diseases of digestive system	46.1	29.5	30.7	31.2	40.9
Genito-urinary diseases	29.0	24.8	29.4	22.1	26.5
Skin diseases	0.2	0.0	0.0	0.4	0.2
Musculo-skeletal diseases	0.9	0.2	2.0	0.2	0.8
Congenital abnormalities	6.7	5.1	4.9	4.1	6.5
Oral conditions	0.0	0.0	0.0	0.0	0.0
Cot death	1.0	0.0	0.0	0.0	0.8

Urban black women have a lower mortality from IHD than white women. The black population also has a less atherogenic fasting lipid profile. For almost three decades, it was assumed obesity in the black South African population was without consequence, because there was no apparent association between obesity and IHD in them. However, it has now become evident that insulin resistance and

type 2 DM are more prevalent in black women. Urban black women who follow a Westernized diet and lifestyle, present with more hypertension (30%) and type 2 DM (7%) than do white women (15% and 3,6% respectively). The prevalence of obesity in black South African with DM, was 15% to 16% of men and 35% to 47% of women. Central obesity in black women were more strongly associated with components of the metabolic syndrome than BMI (Kruger et al., 2005:494). In the 1998 SADHS, 12% of men and 13% of women reported a family history of DM. Rates were much higher in the urban areas. Indian participants reported the highest rates. For cancer, five percent of men and six percent of women reported a family history, more often in urban participants (DoH et al., 2002:168).

In the 2003 SADHS, chronic conditions among males older than 15 years was as follows: hypertension (8,8%), heart attack/angina (2,7%), stroke (0,9%), high blood cholesterol (2%), DM (2,6%), and cancer (0,6%). In women, it was hypertension (18,8%), heart attack/angina (3,9%), stroke (1%), high blood cholesterol (2,1%), DM (3,9%), and cancer (0,8%) (DoH, 2004:23-24).

Increased serum triglycerides, LDL and plasma fibrinogen are all accepted risk factors for CVD. In the THUSA study, these levels were relatively low in most groups, although urban groups already had levels that needed intervention to protect against future CVD. Both triglycerides and LDL cholesterol are related to obesity and low activity, especially in women. The THUSA study also showed that blood pressure correlated positively and significantly with age, level of urbanization, WHR and smoking. Diastolic blood pressure correlated positively and significantly with BMI in women, as well as serum lipids and serum γ-glutamyl transferase (a marker of alcohol intake) (Vorster et al., 2005:online). In the 1998 SADHS, only four percent of respondents reported a family history of dyslipidemia, more frequently in whites (DoH et al., 2002:168).

The WCRF/AICR (2007:8) reports that in 2001, the most common cancers among South African men were found in the prostate; lungs; esophagus; colon; rectum; and bladder. Kaposi's sarcoma, attributable to the HIV and AIDS epidemic, has become more common in both men and women. In women, the most common cancers were in the cervix; breast; colon; rectum; lungs and esophagus (WCRF/AICR, 2007:9).

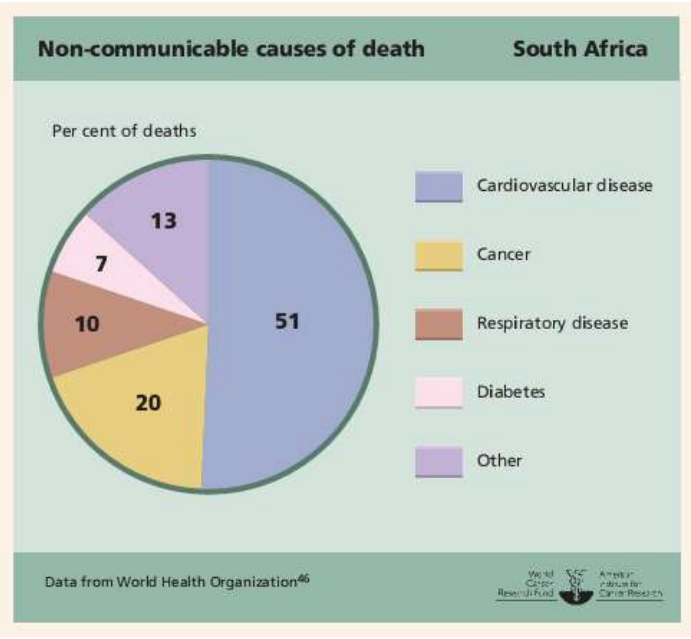


Figure 2.2: Breakdown of deaths in South Africa caused by CDLs (WCRF/AICR, 2007:8)

As can be seen from Figure 2.2, more than half of CDL-related deaths in South Africa were caused by CVD, and a further twenty percent were related to cancer (WCRF/AICR, 2007:8). The biggest contributor to CDL-related deaths is CVD.

The CVD-specific causes of death in South Africa in 2000 as shown in Table 2.16 were derived from the revised estimates of Burden of Disease for the Comparative Risk Factor Assessment for South Africa (Norman et al., 2010a:online). The top five causes were found as follows: IHD accounted for 35%; stroke for another 34,5%; hypertensive heart disease for 16,9%; inflammatory heart disease for 5,5%; and pulmonary embolism for 2,1%.

Table 2.16 Percentage of cardiovascular diseases by cause, South Africa, 2000
(Norman et al., 2010a:online)

Persons			Males			Females		
Rank	**Cause of death**	**%**	**Rank**	**Cause of death**	**%**	**Rank**	**Cause of death**	**%**
1	Ischemic heart disease	35	1	Ischemic heart disease	42	1	Ischemic heart disease	37
2	Stroke	35	2	Stroke	32	2	Stroke	30
3	Hypertensive heart disease	17	3	Hypertensive heart disease	12	3	Hypertensive heart disease	21
4	Inflammatory heart disease	5.5	4	Inflammatory heart disease	6.1	4	Inflammatory heart disease	4.9
5	Pulmonary embolism	2.1	5	Pulmonary embolism	2.1	5	Pulmonary embolism	2.1
6	Rheumatic heart disease	1.7	6	Non-rheumatic valvular disease	1.3	6	Rheumatic heart disease	1.9
7	Non-rheumatic valvular disease	1.7	7	Rheumatic heart disease	1.6	7	Non-rheumatic valvular disease	1.7
8	Peripheral vascular disorders	1	8	Aortic aneurism	1.1	8	Peripheral vascular disorders	0.9
9	Aortic aneurism	1	9	Peripheral vascular disorders	1.3	9	Aortic aneurism	0.7
10	Other cardiovascular	0.5	10	Other cardiovascular	0.6	10	Other cardiovascular	0.4
	All cardiovascular	100		All cardiovascular	100		All cardiovascular	100

2.6.3 Specific CDLs

Specific CDLs will be discussed in this section, with emphasis on: overweight and obesity (including the metabolic syndrome); DM; CVD; hypertension; stroke; CHD; and cancer.

2.6.3.1 Overweight and obesity

Obesity is seen as a global epidemic with an estimated 1,3 billion people overweight or obese. The WHO/FAO (2002:5) states that overweight and obesity are increasing at a substantial rate annually in developing countries. According to SASOM (2006b:online), obesity has been acknowledged as a very complex endocrine abnormality with a strong genetic predisposition, and not simply a lifestyle disease. The WHO has officially classified it as a chronic disease of “epidemic proportions;” in both the developed- and developing world (WHO/FAO 2002:3). Obesity is especially increasing among the poor population groups of developed- and developing countries, (Sawaya & Roberts, 2003:online). Almost all countries are experiencing an obesity epidemic (WHO/FAO, 2002:61).

Increased morbidity and mortality, with significantly increased healthcare costs, are just some of the health consequences of obesity (Kruger et al., 2005:492). Relative weight in adulthood as well as weight gain, are associated with an increased risk of breast-, colon-, rectum- and prostate cancer (WHO/FAO, 2002:34). There is clear evidence of a relationship between onset of obesity and cancer risk (WHO/FAO, 2002:35). The WHO lists being overweight as one of the ten leading risk factors for high mortality, thus contributing to the burden of disease in developing- and developed countries. Thus, the “epidemic of obesity,” together with its accompanying co-morbidities (e.g. heart disease, hypertension, stroke and DM), is not a health problem only found in industrialized countries (WHO/FAO, 2002:8). According to the WHO/FAO, (2002:61-62), mortality rates increase with an increase in BMI. As the BMI increases, so does the proportion of people with one or more co-morbid

conditions. More than half (53%) of all deaths in USA women with a BMI>29 kg/m² could be directly attributed to obesity (WHO/FAO, 2002:61-62).

Clinical problems associated with obesity can be categorized as: (1) those associated with excess adipose tissue; and (2) those associated with the metabolic effects of increased adiposity. Diseases associated with the first category include osteoarthritis, sleep apnea and psychological problems (Goedecke et al., 2005:67); while the metabolic effects of increased adiposity are the CDLs; including CHD, hypertension, type 2 DM and cancer (Goedecke et al., 2005:68). The distribution of adipose tissue influences disease risk. Accumulation of fat in the abdominal area, particularly in the visceral fat compartment (i.e. central obesity), as measured by waist circumference (Lee & Nieman, 2010:180; WHO/FAO, 2002:69; Mollentze et al., 1995:90) and WHR (Gibson, 2005:281;Lee & Nieman 2010:180; Lee & Nieman, 2003:183, Table 6.8; Centers for Disease Control and Prevention (CDCP), 2009:online), is associated with insulin resistance, DM, hypertension, dyslipidemia, stroke and atherosclerosis (Gee et al., 2008:533, 541; Lee & Nieman, 2010:181; Goedecke et al., 2005:68). Central adiposity is also the cornerstone of the metabolic syndrome, characterized by a clustering of factors, including: insulin resistance; hyperinsulinemia, glucose intolerance; hypertension, and elevated plasma triglycerides and low high-density lipoprotein cholesterol (HDL) (Gee et al., 2008:533, 541; Goedecke et al., 2005:68; Kruger et al., 2005:494; Laquatra, 2004:568; WHO/FAO, 2002:73-74).

Today, metabolic syndrome or syndrome X can occur as early as childhood and adolescence, and is associated with atherosclerosis in young adulthood and an increased risk for later CVD (WHO/FAO, 2002:36). Metabolic syndrome in childhood and adolescence is clustered with obesity, especially central obesity and is found in children and adolescents with excessive intakes of saturated fats, cholesterol and salt, and inadequate intake of fibre (WHO/FAO, 2002:41). Childhood obesity has a greater effect on the development of the metabolic syndrome than becoming obese as an adult (Goedecke et al., 2005:70).

Table 2.17: Differential manifestations of the features of metabolic syndrome in South Africa
(Van der Merwe, 2001:5, Table 1)

Black population	**White population**
Overweight and obesity (BMI >25 kg/m^2) (64%)	Overweight and obesity (BMI >25 kg/m^2) (50%)
Type 2 diabetes (8-12%)	Type 2 diabetes (4%
Accelerated hypertension	Hypertension
Cerebral hemorrhage (3%)	Infarcts (15%)
IHD low prevalence eight in a hundred	IHD very prevalent sixty in 100 000
Insulin resistance (high degree) (associated with brisk lipolysis)	Insulin resistance (lesser degree) (euglycemic hyperinsulinemic clamp studies)
More favourable lipid profiles	Adverse lipid profile
Visceral fat mass lower	Visceral fat mass higher
Gluto-femoral area displays insulin resistance with brisk lipolytic rate and a high subcutaneous blood flow	Regional adipose tissue metabolism in keeping with classical metabolic syndrome

Table 2.17 compares the different features of the metabolic syndrome manifested in the white and black South African population. It is interesting to note that type-2 DM presents differently in South Africa among black South Africans as opposed to white American and African-Americans. Obesity-related type-2 DM is traditionally characterized by hyperinsulinemia, as seen in white Americans and African-Americans (Van der Merwe & Pepper, 2006:5). Obesity-related type-2 DM among black South-

Africans presents with insulinopenia, leading to the metabolic syndrome (Van der Merwe & Pepper, 2006:5).

The WHO's Comparative Risk Assessment Study estimated that an increase in BMI >21 kg/m^2 in adults older than thirty years, was associated with type 2 DM (58% of cases); IHD (21%); hypertension (39%); ischemic stroke (23%), colon cancer (12%); postmenopausal breast cancer (8%), endometrial cancer (32%); and osteoarthritis (13%) (Joubert et al., 2007:684). Also see Figure 2.2.

Jinabhai (2003:online) states that overweight and obesity are preventable conditions and early signs of increasing prevalence offer opportunities to develop preventive strategies. The prevalence of obesity may in part be attributed to rapid changes in diet, urbanization, and changes in physical activity (Goedecke et al., 2005:67; Kruger et al., 2005:491; Sawaya & Roberts, 2003:online; Vorster et al., 2005:online; WHO/FAO, 2002:13). The factors that might promote or prevent weight gain and obesity are summarized in Table 2.18.

Table 2.18. Summary of strength of evidence on factors that might promote or prevent weight gain and obesity (WHO/FAO, 2002:63, Table 7).

Evidence	***Decreased risk***	***No relationship***	***Increased risk***
Convincing	Regular physical activity High intake of dietary fibre		Sedentary lifestyle High intake of energy-dense micronutrient-poor foods
Probable	Home and school environments that support healthy food choices for children Breastfeeding		Heavy marketing of energy-dense foods and fast-food outlets High intake of sugar-sweetened soft drinks and juices Adverse socio-economic conditions (in developed countries, especially for women)
Possible	Low GI foods	Protein content of the diet	Large portion sizes High proportion of food prepared outside the home (developed countries) "Rigid restraint/periodic disinhibition" eating patterns
Insufficient	Increased eating frequency		Alcohol

(i) In the world

The occurrence of obesity at present is unparalleled in the history of mankind (WCRF/AICR, 2007:13). Globally in 2005, an estimated 1,1 billion adults globally (26% of the world population) were overweight (including 312 million obese adults) (Joubert et al., 2007:683), In 2002, Chinese people accounted for approximately one-fifth of the one billion overweight or obese people worldwide. Historically, China had a lean population, but the prevalence of underweight in adults has decreased, and overweight and obesity has risen up to the point where 184 million Chinese people were overweight and 31 million were obese in 2002 (out of a population of 1,3 billion) (WCRF/AICR, 2007:13).

It was estimated that between 1989 and 1997, the percentage of overweight or obese men rose from 6,4% to 14,5% and from 11,5% to 16,2% in women. Between 1989 and 2000, the proportion of overweight and obesity increase by 13,7% in men and 7,9% in women. In the same period, there was only an average decrease of two percent in underweight men and women (WCRF/AICR, 2007:13).

During a ten year period in the 1980s and 1990s, the WHO's MONICA project monitored ten million adults in 21 countries. It was found that the mean BMI increased in most populations, with the largest increases found in Australia and the USA. Overall average BMI increased by 1,5. An average BMI

decrease was found in Russia, Central Europe, and in certain regions of Italy and Switzerland. During 1980 and 2003, overweight and obesity increased threefold in the UK, with 65% of men and 56% of women being overweight, and a further 22% of men and 23% of women being obese (WCRF/AICR, 2007:13).

Between 1992 and 2000, overweight exceeded underweight in most middle- and low-income countries (in North Africa, the Middle East, Central Asia, China and Latin America). Both the incidence and prevalence of obesity among lowest-income groups in most countries has increased "disproportionately". Being overweight was found to be most likely in urban residents. Countries with a higher GDP had a higher ratio of overweight to underweight women (WCRF/AICR, 2007:14).

Very high rates of overweight and obesity have been reported among the middle- and low-income countries of North Africa and the Middle East, often being higher in women than men. Since the mid-1970s, the rise in overweight and obesity has been much faster in lower-income countries. The prevalence has only raised by 0,3-0,5% per annum in Europe and the USA, but the increase has been two- to four-fold higher in many low-income countries (WCRF/AICR, 2007:14).

As can be seen from Figure 2.3, by 2015 the occurrence of obesity could be as high as fifty percent in the USA; between thirty to forty percent in the UK and Australia; and more than twenty percent in Brazil. It was estimated that more than 12 million adults would be obese by 2010 in England, while 25% of children living with obese parents, would themselves become obese (WCRF/AICR, 2007:14).

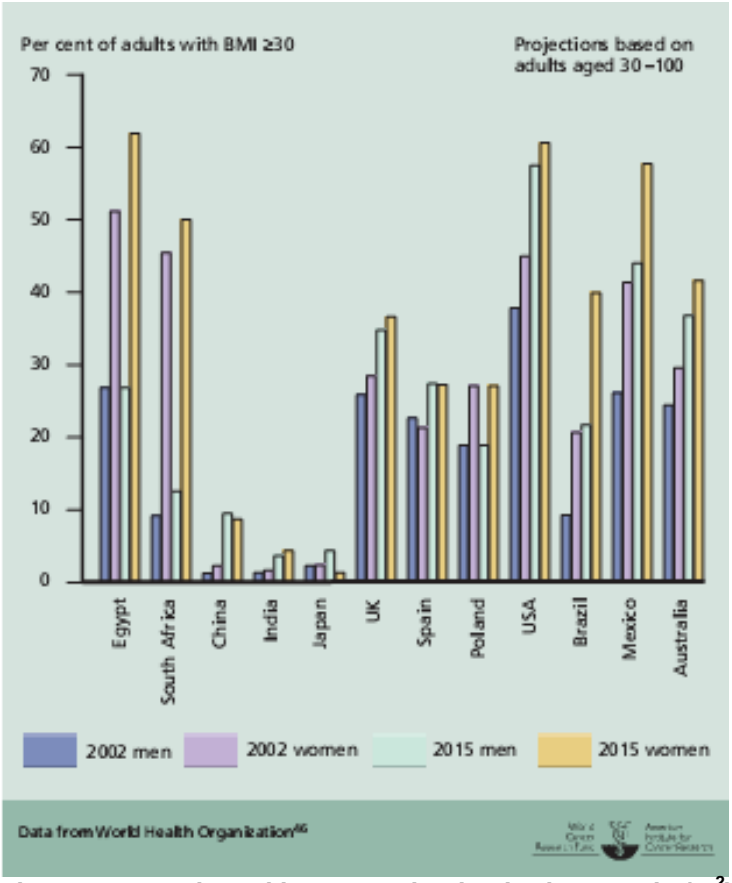


Figure 2.3 Projected increases in obesity (BMI≥30 kg/m^2) in selected countries 2002-2015 (WCRF/AICR, 2007:14, Figure 1.4)

Puoane et al. (2004:2) state that this worldwide problem can be considered to be a result of social-, economic- and cultural problems being encountered by developing- and newly industrialized countries, as well as ethnic minorities and the disadvantaged in developed countries.

Weight gain, overweight, and obesity are now generally much more common than in the 1980s and 1990s. Rates of overweight and obesity doubled in many high-income countries between 1990 and 2005. In most countries in Asia and Latin America, and some in Africa, chronic diseases, including obesity, are now more prevalent than nutritional deficiencies and infectious diseases (WCRF/AICR, 2007:xvii).

(a) In children

Overweight in childhood and early life is liable to be followed by overweight and obesity in adulthood (WCRF/AICR, 2007:xvii). Obesity not only persists from infancy into adulthood, but it has also been associated with an unfavourable cardiovascular risk profile in the first twenty years of life (Mukuddem-Petersen & Kruger, 2004:online). Childhood obesity is associated with several risk factors for CDLs, including heart disease, in later life (Jinabhai et al., 2003:online). The increase in overweight and obesity in the adult population has resulted in an increase in the prevalence of CDLs (Mamabolo et al., 2005:online). Childhood is therefore a key period in which to identify the risk of becoming obese in adulthood (Mukuddem-Petersen & Kruger, 2004:online).

In 2005, 22 million children under age five years were classified as overweight. The prevalence of overweight in American children tripled between the 1960s and 1990s. In Chile, between 1987 and 2000, obesity prevalence in six year olds increased from 6,5% to 7,8% in boys and from 17% to 18,6% in girls (Goedecke et al., 2005:67). The prevalence of overweight has also increased in children over the past twenty years in developing countries such as India, Mexico, Nigeria and Tunisia (WHO/FAO, 2002:8).

In a study carried out on children living in informal settlements in Sao Paulo, Brazil, it was found that low energy expenditure may be the most important risk factor for weight gain in susceptible populations (Hoffman et al., 2000:1030). This may explain the increased risk of excess weight gain leading to obesity among girls from this population as compared to the boys (Mukuddem-Petersen & Kruger, 2004:online). Obesity in children is also becoming a significant problem in industrialized countries, including those in South-East Asia (Darnton-Hill & Coyne, 1998:24).

About twenty to 25% of Mexican children aged between two and six years old, are overweight or obese (Fernald & Neufeld, 2007:623). There is a higher prevalence in girls (Fernald & Neufeld, 2007:624). Overweight in children is more common in urban areas, and particularly in girls (Steyn et al., 2005:12). The risk of overweight and obesity in childhood increased with maternal overweight (Fernald & Neufeld, 2007:629).

High blood pressure in children is strongly associated with obesity, in particular central obesity, together with an adverse serum lipid profile (especially in LDL and total cholesterol) and glucose intolerance. The thinner the child was, the more obese the adult becomes; with a greater subsequent risk of developing CDLs (WHO/FAO, 2002:36-37). The later the weight gain in childhood and adolescence, the greater the persistence of being overweight. More than sixty

percent of overweight children aged five to ten years, have at least one additional risk factor for CVD, such as hypertension, dyslipidemia or hyperinsulinemia, and more than 25% have two or more risk factors (Goedecke et al., 2005:70; WHO/FAO, 2002:37). A large increase in type 2 DM in children and adolescents has been documented. In the USA the incidence of type 2 DM increased tenfold between 1982 and 1994 (Goedecke et al., 2005:70). In 2002, the amount of DM cases were estimated to be around 150 million worldwide. This number was expected to double by 2025, with the largest proportion to be in China and India (WHO/FAO, 2002:72).

Intra-abdominal adipose tissue in obese children has a significant relationship with dyslipidemia and glucose intolerance. Childhood obesity also results in the development of co-morbidities of obesity in adulthood, since childhood obesity often persists into adulthood. Childhood obesity has a greater effect on developing the metabolic syndrome than becoming obese as an adult (Goedecke et al., 2005:70; Vanhala et al., 1999:657-658; Vanhala et al., 1998:319-320).

(b) In adults

In the USA, between the 1960s and the 1990s, the increase in obesity among adults was 1,6 fold (Goedecke et al., 2005:67). According to Darnton-Hill and Coyne (1998:24), almost half of North American adults were overweight in 1997; and more than a third of Australian- and American populations are overweight. The greatest increase in obesity has been found in developing countries (Goedecke et al., 2005:67).

In the USA, obesity prevalence is as high as 26,6% in men and 32,2% in women above twenty years of age (Goedecke et al., 2005:65). The prevalence of overweight ranges from nine percent to 31% in the UK and USA (Jinabhai, 2003:online). The prevalence of overweight in China ranges from 0,2% to 11%. Russia and Spain are approaching a prevalence rate similar to the UK and USA. When looking at black, Indian and mixed race populations of Trinidad and Tobago, the pattern appears similar to the UK's (Jinabhai, 2003:online). Obesity is also becoming an increasing problem in countries undergoing epidemiological transition, such as South Africa, Mexico and South American countries (Goedecke et al., 2005:65). In Mexico, the prevalence of obesity in women increased from 9,4% in 1988, to 24,4% in 1999 (Goedecke et al., 2005:66). Also in Mexico, 17,1% of mothers were obese and 35,5% were overweight in 2003 (Fernald & Neufeld, 2007:627). In Brazil, the prevalence of overweight tripled from 4,1% in 1974, to 13,9% in 1997 (Goedecke et al., 2005:66). According to Mendez et al. (2005:720), overweight in Brazilian adults appears to have replaced undernutrition as a public health problem. In Brazil, it was found that the prevalence of obesity increased with increased income in men, whereas in women obesity increased with poverty (Sawaya & Roberts, 2003:online). In the Federated States of Micronesia, over fifty percent of women between forty and 49 years of age were obese and eighty percent were overweight in 1984. A high prevalence of obesity has also been described in many populations undergoing transition in the Pacific and Indian Oceans (Darnton-Hill & Coyne, 1998:25). According to Darnton-Hill and Coyne (1998:25) obesity is increasingly seen, mainly in urban areas. In Mauritius, similar results were found between 1987 and 1992 with a prevalence of overweight and obesity increasing from 26,1% to 35,7% in men and from 37,9% to 47,7% in women (Goedecke et al., 2005:67).

According to Mendez et al. (2005:720), the overweight burden is generally higher than that of underweight in young women. In low-income countries, obesity is more common in middle-aged women, people with a high socio-economic status and urban residents (WHO/FAO, 2002:61). In developed countries, overweight was high in women with low socio-economic status from both rural (38%) and urban areas (51%) (Mendez et al., 2005:714). In comparison with low-income countries, obesity seems to be more common in developed countries among middle-aged people, younger adults and children. Obesity also tends to be associated more with lower socio-economic status, especially in women, and the differences between rural- and urban residents are diminished or reversed (WHO/FAO, 2002:61).

(ii) In Africa

In most African countries studied in the International Obesity Task Force: Global Burden of Disease Analyses 2002, women had a higher BMI than men. In Cameroon, the average BMI for ages 15 to 59 was 24 kg/m² for males and 24,8 kg/m² for females. In the Seychelles, the average BMI for males aged between 15 and 69 years was 23,2 kg/m², but for women of the same age it was 25,9 kg/m². In South Africa, the average BMI in males for the same age group was 24 kg/m² and for females 27,9 kg/m² (Goedecke et al., 2005:66, Table 7.1).

Van der Merwe and Pepper (2006:4) state that the prevalence of obesity is increasing in most African countries, especially in urban areas. The impact of obesity on CDLs varies and the impact of obesity on type 2 DM is unclear. In the African population with type 2 DM, the prevalence of obesity varies between 15% to seventy percent. In Sub-Saharan Africa, type 2 DM is accompanied by "severe insulinopenia" combined with varying levels of insulin resistance (Van der Merwe & Pepper, 2006:4).

In Ghana, BMI did not differ between natives with normal fasting glucose and those with DM, but biceps and triceps skinfolds were significantly larger in the diabetic population. Interestingly, hyperinsulinemia, insulin resistance and abdominal obesity were found in hypertensive subjects (Van der Merwe & Pepper, 2006:4).

Globally, the prevalence of childhood obesity is a serious problem, but there is insufficient data for most African countries (Mukuddem-Petersen & Kruger, 2004:online).

(i) In South Africa

The high number of overweight and obesity among women can be attributed to the fact that weight loss is associated with HIV and AIDS and that moderately overweight women are thought of as attractive, affluent and well-cared-for by their husbands (Joubert et al., 2007:688; WCRF/AICR, 2007:9).

The Burden of Disease study found in 2000 that excess body weight accounted for one in every ten female deaths in South Africa (Joubert et al., 2007:687). Urban black women are at greatest risk for obesity (Goedecke et al., 2005:66).

Childhood obesity is a strong predictor of obesity in adulthood (Joubert et al., 2007:688). SASOM (2006c:online) states that twenty percent of children under the age of six in South Africa are overweight. These children have a high likelihood of growing into overweight adults.

(a) Incidence, prevalence and epidemiology

In the 1990s, three adult studies found the prevalence of obesity in black women as 34,4%, 53,1% and 53,4% respectively. Lower prevalence of obesity was found in black men with 7,9%, 23% and 32,9% respectively (Bourne et al., 2002:159).

According to WHO data, in 1998 South African men aged between 15 to 24 years old, had an average BMI of 21,1 kg.m^2. For the group between 35 to 65 years of age, the average was 25 kg/m^2. Only 7,8% of men aged between 25 and 34 years old had a BMI >30 kg/m^2, compared to 17,3% of men aged between 45 and 54 years old (WCRF/AICR, 2007:9).

In 1998, women between 15 and 24 years old had an average BMI of 23,7 kg/m^2. For the 35 to 64 years age group, the average BMI was 29 kg/m^2. For women aged between 25 and 34 years old, 27% had a BMI >30 kg/m^2, compared with 45% of women aged 45 to 64 years old. Overall, 21,1% of men and 25,9% of women were overweight (BMI $\geq$25 kg/m^2), and 10,1% of men and 27,9% of women were obese (BMI $\geq$30 kg/m^2) (WCRF/AICR, 2007:9). Refer to Figure 2.3 to ascertain the projections for 2015.

In the Cape Peninsula study (Steyn et al., 1998:35), the prevalence of overweight was 36,4% in women and 22% in men. In the Free State study (Mollentze et al., 1995:93), the prevalence of obesity among women ranged from 27,5% to 49% in QwaQwa (rural area), and from 31,1% to 54,3% in Mangaung (urban area). The prevalence among men was only in the range of 7,4% to 19,2% for QwaQwa men, and from three percent to 24% for Mangaung men. The MRC Technical Report (Steyn et al., 2006, 20) stated that overweight/obesity was more prevalent in urban areas, which is indicative of nutrition transition. Higher levels of BMI were found in older men, those living in the city, and white men. Vorster et al. (2005:online) found in the North West Province that overweight and obesity increased with an improvement in micronutrient intakes and micronutrient status. Vorster and Kruger (2007:323) indicate the rate of overweight and obesity in men was 25,4% in blacks, 30,8% in coloureds, 32,7% in Indians, and 54,5% in whites. In women, the figures were 58,4% in blacks, 52,2% in coloureds, 48,9% in Indians, and 49,2% in whites. Black female students were more likely to be either underweight or overweight/obese than their white counterparts, with 26,8% underweight; 18,2% overweight and 6,5% obese as compared to 7,2% underweight, ten percent overweight and 0,8% obese. In a postal survey it was found that 56,4% of white men, 49,3% of black men and 74,6% of black women were overweight or obese. The prevalence was lower in men (45,7%) and women (66%); 35,5% for Indian men, 37% for white men and 42,2% for white women (Senekal et al., 2003:112).

Goedecke et al. (2005:69) state that a study in South Africa confirmed what has already been found in America: black women have less visceral adipose tissue than white women, but were more insulin resistant. However, the 1998 SADHS found that more than twice as many black women (35,3%) than white women (17,4%) had a waist-hip-ratio greater than 0,85 cm (Goedecke et al., 2005:69). In 1995,

Mollentze et al. (1995:93) found that WHR for men exceeded that for women. It also increased with age in both rural and urban areas in the Free State. The mean BMI for both rural and urban men were below 25 kg/m^2, whereas the mean BMI for women in both rural and urban Free State exceeded 25 kg/m^2 (Mollentze et al., 1995:93). Forty-two percent of women had central obesity (android fat pattern) and this was more prevalent in urban black women and coloured women (Goedecke et al., 2005:65). About nine percent of men had central obesity, with higher levels in older and white men (Goedecke et al., 2005:66). However, in the 1998 SADHS, seven percent of men and 32% of women had a WHR above the cut-off point, with the ratio increasing with age for men and women. In women, the prevalence for a WHR above the cut-off point was higher in the rural areas (DoH, et al., 2002:245).

In 1998, the SADHS showed an adult South African population with a malnutrition pattern consisting predominantly of overnutrition, particularly in black women (DoH et al., 2002:21). Ten percent of women were already obese at 15 to 24 years of age (Goedecke et al., 2005:66; Bourne et al., 2002:159) and forty percent of women were found to be obese by age 35 (Steyn et al., 2006:21). In the 1998 SADHS, the mean BMI was 23,4 kg/m² for men and 27,3 kg/m² for women. According to WHO standards, 29% of men and 56% of women were overweight and obese (Joubert et al., 2007:683; Steyn, 2005b:249; Mukuddem-Petersen & Kruger, 2004:online; DoH et al., 2002:244). This is higher than reported in other African countries (Goedecke et al., 2005:65). Interestingly, less than five percent of men and 15% of women perceived themselves as obese (Bourne et al., 2002:159). Nearly thirty percent of South African women aged between thirty and 59 years were obese (Goedecke et al., 2005:65). Only about ten percent of men are obese. Overweight and obesity were highest in the Western Cape, KwaZulu-Natal and Gauteng. The highest prevalence of overweight and obesity was among black women with 26,7% being overweight and 31,8% obese with a total of 58,5% (Vorster & Kruger, 2007:323; Goedecke et al., 2005:65). Fifty-two percent of coloured women, 49,2% of white women and 48,9% of Indian women were overweight/obese. The prevalence of obesity was particularly high among women in general (30%), with 33% of urban- and 25% of rural women presenting with obesity (Joubert et al., 2007: 638; Goedecke et al., 2005:65)

In men, a different pattern was noted. The prevalence of overweight and obesity was the highest in white men (54,5%), while 32,7% of Indian men and 31% of coloured men were overweight or obese. The lowest prevalence was seen in black men (total of 25%), with only six percent being obese and 19,4% being overweight (Goedecke et al., 2005:66). Urban men and older men had the highest BMIs (Goedecke et al., 2005:66; DoH et al., 2002:244). Women were five to ten times more obese than men, and the percentage of men with low to normal BMI (<18.0 to 24.9 kg/m^2) was higher than in women (Vorster et al., 2005:online). In 1998, the prevalence of obesity among adults, particularly in women ranged from 21% to 31% in different population groups. In white males it was 21%, while it was less than ten percent in other population groups. The prevalence amongst all males was twenty percent (Steyn et al., 2006:34).

The second SADHS conducted in 2003, provides information of a five year trend. Minimal changes in the percentage of overweight men were noted. There was a decline in the proportion of obese men as well as obese women (DoH, 2004:25-26). The 2003 SADHS found that 21% of adult men were overweight and nine percent obese. In women, 29% were overweight and 23% obese. Obesity was more prevalent in older men and women and in the urban areas. Obesity in men was most common in

the provinces of the Western Cape, Gauteng and KwaZulu-Natal. Obese women were more common in the provinces of Eastern Cape, Gauteng and Western Cape (DoH, 2004:24-25). The MRC's Burden of Disease study found that the mean BMI for both genders was "well above" 21 kg/m^2 and that it declined with increasing age. The mean BMI for adults older than 30 years was 28,7 kg/m^2 for women and 24,1 kg/m^2 for men. When using the WHO classifications, 27,3% of men and 29,1% of women older than 30 years were overweight and another 11,0% of men and 38,6% of women older than thirty years were obese (Joubert et al., 2007:685).

In the Burden of Disease study, for type 2 DM and cardiovascular outcomes, the risk was highest in the thirty to 44-year age group, with risk decreasing with age. Cancer-related outcomes and osteoarthritis in males peaked in the 45 to 59 years group and in females peaked in the sixty to 69 years groups (Joubert et al., 2007:685).

The Burden of Disease study also found that the largest proportion of deaths related to excess body weight occurred between 45 and 59-years of age (Joubert et al., 2007:687).

In 1998, combined figures for overweight and obese South African adults across all ethnic groups were 57% for women and 29% for men (Van der Merwe & Pepper, 2006:1). In 2006, it was estimated that the prevalence of obesity in black women was between 31% and 34%, in white women between 18% and 24%; in Indian women between 20% and 22%; and in coloured women between 26% and 28%. The prevalence of obesity in black men was estimated at eight percent, in Indian men between three- and nine percent; in coloured men between six- and nine percent; and the highest prevalence was estimated for white men (between 15% to 20%) (Van der Merwe & Pepper, 2006:1-2). The combined figures for overweight and obesity in the different ethnic groups were: 75% black women; 49% black men; 66% coloured women; 45,7% coloured men; 37% Indian women; 36% Indian men; 42% white women; and 56% white men (Van der Merwe & Pepper, 2006:6). Determinants associated with an increased risk for overweight include: black race; inactivity; and at least one overweight parent (Steyn et al., 2005:12).

As stated previously, the NFCS reported that in 1999 the prevalence of combined overweight and obesity (17,1%) among children between one and nine years of age at the national level was as high as that for stunting (19,3%) (Steyn et al., 2006:34; Steyn, 2005a:43-44; Steyn et al., 2005:4; Goedecke et al., 2005:67; Labadarios et al., 2005:536; Mamabolo et al., 2005:online; Steyn, 2005b:249). The prevalence of overweight in the Free State province (6,2%) as well as in urban areas nationally (7,7%) exceeded the national average (6%) (Labadarios & Nel, 2000:175). Nationally, the prevalence for overweight (6%) remained constant in all age groups (Labadarios & Nel, 2000:169).Ten percent of South Africa women aged between 15 and 24 years old are already obese (Goedecke et al., 2005:66). In the Youth Risk Behaviour Survey, 17% of adolescents were found to be overweight and 4,2% were obese (Goedecke et al., 2005:66; Kruger et al., 2005: 492).

There seems to be some contradictory results when prevalence of obesity according to race is investigated. In a regional school survey, the prevalence of obesity in black girls aged 18 was projected to be 37%, as compared to ten percent for white girls and twenty percent for coloured girls (Goedecke et al., 2005:66). However, the THUSA BANA study conducted in the North West Province

found the BMI in black children to be lower than those of white and Indian children. The same was true for body fat percentage. Iin children aged ten to 15 years old, the highest prevalence of overweight and obesity was found among white children (14,2%), compared to black (7,1%), Indian children (6,4%) and coloured children (2,9%). Body fat was significantly higher in girls of all races, than in boys (Mukuddem-Petersen & Kruger, 2004:online).

In 1996, Popkin et al. (1996:3012) analyzed the nutritional status of South African pre-school children (between three and six years of age) using the WHO NHANES III definition. They found that the prevalence of overweight was 25% for boys and 20,3% for girls.

In the THUSA-BANA study, Kruger et al. (2005:492) found that approximately ten percent of children were overweight, but that overweight was almost non-existent in younger, rural boys. Obesity may start during the adolescent growth spurt (Mukuddem-Petersen & Kruger, 2004:online). Kruger et al. (2005:492) reports that females were more prone to obesity, with overweight occurring more frequently before the growth spurt at ten years of age, as well as after menarche. None of the rural boys were overweight before the age of 15 years, after which body fat increased to a peak at seventeen years. In girls, body fat increased after menarche, also peaking at 17 years of age with 11% of girls being classified as “over-fat.” In one to nine year olds, 6,7% were overweight and 3,7% were obese. The highest overweight prevalence was among urban children (Kruger et al., 2005:492; Steyn et al., 2005:8).

At the national level, the NFCS found that 12,4% of all children were overweight and 6,6% were obese. The combined rate for overweight and obesity was again the highest in urban areas in all age groups (20,7%) (Steyn et al., 2005:8; Labadarios & Nel, 2000:176). Also of interest was that 17,1% of children had a BMI of at least 25 kg/m^2. The highest prevalence of overweight was in urban areas (18,6%) and in those children aged one to three years. Only 13,4% of rural children were overweight (Steyn et al., 2005:9,10,12). According to weight-for-age data, 6,7% of children nationally were overweight and 3,7% were obese (10,4% together). The highest prevalence was in urban areas. According to weight-for-height data, 12,4% were overweight and 6,6% were obese (19% together), again with the highest prevalence in the urban areas (Steyn et al., 2005:8).Urbanization appears to influence the prevalence of obesity in South African children (Goedecke et al., 2005:67). However, rates of overweight and obesity higher than those previously reported were found by Mamabolo et al. (2005:online) in 2003, who reported a high prevalence of overweight (22%) and obesity (24%) amongst rural children in Limpopo. This may be indicative of a nutrition transition.

(b) Etiology

Overweight and obesity result from the energy consumed from fat, carbohydrate, protein and alcohol exceeding energy needs (Vorster & Kruger, 2007:323). Household income, total energy intake, fat intake and low physical activity are the major determinants of obesity (Vorster et al., 2005:online).

Kruger et al. (2005:492) postulates that the obesity epidemic in South Africa is indicative of globalization, which compels the nutrition transition. Obesity and its co-morbidities affect South Africans negatively and the burden of disease contributes to the increasing healthcare costs, both in the private and public sector (Goedecke et al., 2005:65).

The difference in obesity among different ethnic groups is influenced by many factors, including the environment and genetics. However, there has been limited progress on the identification of genetic components. A few single-gene disorders have assisted in identifying significant components involved with metabolic pathways where gene disruption can lead to obesity, e.g. leptin; pro-opiomelanocortin and prohormone convertase. In South Africa, the only genetic data available refers to the gene which encodes for the ß3 subunit of heterotrimeric G proteins. The 825T allele is significantly linked to obesity in blacks and is also linked to hypertension. There are similar allele frequencies found both in rural and urban populations. When genotyping was conducted among 5254 individuals from 55 population groups from Africa, the Americas, Europe, Asia, Australia and New Guinea, the highest 825T allele frequencies were found in black Africans (82%) (Van der Merwe & Pepper, 2006:5).

It is speculated that greater visceral fat mass may have been promoted by higher cortisol levels and may lead to higher lipid profiles (Kruger et al., 2005:495).

2.6.3.2 Diabetes mellitus

According to the WDF (2006:online), DM is one of the major causes of premature death worldwide. Worldwide, a person dies from DM-related causes every ten seconds (WDF, 2006:online). DM contributes to a considerable increase in both morbidity and mortality (Bradshaw et al., 2007:700; Franz, 2004:794). Complications of type 2 DM include: blindness; kidney failure; foot ulcers (which may lead to gangrene and amputations); CHD; stroke; and increased infection risk. DM is responsible for over one million amputations annually; as well as a large percentage of cataracts and at least five percent of blindness worldwide. DM is also the largest cause of kidney failure in developed countries (WDF, 2006:online). Type 2 DM is also now starting to occur in children, where before it was only found in adults (SASOM, 2006c:online).

Obesity-related type-2 DM among black South-Africans presents with insulinopenia, leading to the metabolic syndrome. An insulin resistance is often also noted in these persons (due to free fatty acid toxicity). The insulin resistance together with the insulinopenia increase the risk for keto-acidosis. It has been found that even if young persons in good physical condition are matched for body composition and weight, black South Africans are relatively more insulinopenic than their white counterparts (Van der Merwe et al., 1999:910).

(i) Incidence and prevalence

Type 2 DM (resulting from insufficient production of insulin in order to overcome the underlying insulin resistance) (WDF, 2006:online; Franz, 2008; 769; Franz, 2004:797), accounts for most cases of DM (between 90% to 95% worldwide) (WDF, 2006:online). In the early stages of type 2 DM, insulin is overproduced, but later on insulin levels may fall as a result of partial failure of the ß-cells of the pancreas (WDF, 2006:online).

In 2000, DM was estimated to be the fifth leading cause of death worldwide, accounting for 5,2% of global deaths, and the tenth leading cause of death in South Africa, accounting for an estimated 2,6% of deaths (Bradshaw et al., 2007:700). The lowest mortality (2,4%) for DM is reported for the poorest African- and Western Pacific countries, and the highest mortality (+9%) is found in the Arabian Peninsula, East Mediterranean region, Canada and the USA (Bradshaw et al., 2007:703).

The prevalence of DM increases with age. It affects 18,4% of people older than 65 years of age (Franz, 2004:794). However, type 2 DM no longer only affects mainly older adults. Between 1990 and 1998, the prevalence increased (by 76%) among people in their thirties and has also increased dramatically amongst children (up to 45% among certain racial groups) (Franz, 2008:766).

Whereas DM was previously a disease of the middle-aged and elderly, it has recently escalated in all age groups; including adolescents and children, especially in high-risk populations (WHO/FAO, 2002:72), and where major changes in diet, reduction in physical activity, and increase in overweight and obesity have occurred (WHO/FAO, 2002:73).

In 1985, an estimated thirty million people had DM worldwide (WDF, 2006:online). The WHO estimated that 135 million had DM in 1998 (Bradshaw et al., 2007:700). In 2002, the WHO estimated the incidence of DM worldwide at around 150 million (WHO/FAO, 2002:72) and the prevalence was estimated around 171 million in 2000 (Bradshaw et al., 2007:700). The prevalence increased to 220,5 million worldwide in 2004, of which 9,7 million (4,4%) were from Africa. The Western Pacific countries had the highest prevalence with 56 million or 25,4% (WHO, 2008:32, Table 7). In 2006, DM affected about six percent of the world's adult population (230 million people). In 2006, India had the world's largest DM population (35 million people) (WDF, 2006:online). It is also particularly prevalent in the minorities of the USA, including: African-Americans; Hispanics, Native Americans, Alaska natives; Asian-Americans and Pacific Islanders (Franz, 2004:794). In many countries in Asia, the Middle East, Oceania and the Caribbean, DM affected twelve to twenty percent of the adult population (WDF, 2006:online).

By 2025, the number of DM cases is predicted to double, with most cases expected in China and India (WHO/FAO, 2002:72). The WHO predicts that developing countries will be affected the most by the DM epidemic, with eighty percent of all new cases appearing in developing countries by 2025, mostly among people aged between 45 and 64 years old (WDF, 2006:online). DM prevalence is projected to increase to 366 million worldwide by 2030 (Bradshaw et al., 2007:700). The projected increase in developing countries is partly due to an ageing and growing population, but also attributable to urbanization and the nutrition- and physical activity transition. Increasing prevalence of obesity globally will exacerbate this situation (Bradshaw et al., 2007:700)

Bourne et al. (2002:161) state that the prevalence of DM in the black South African population is considerably higher than 25 years ago, ranging from five percent in the Gauteng province to eight percent in Cape Town. In the early 1990s, Mollentze et al. (1995:93) found DM prevalence to be at 4,8% in rural Free State and six percent in urban Free State. The condition was more frequent in men older than 45 and women older than 35 years of age. The Indian population of South Africa has the highest DM prevalence, followed by the coloured- and black populations (Bradshaw et al., 2007:700). About one to 1,5 million South Africans are considered to have DM, which amounted to 5,5% of the population older than thirty years (Bradshaw et al., 2007:700). The prevalence increased with age in both genders, although there was a marked difference over the age of sixty years, with the prevalence being much higher among females of all population groups except Indian, where there was a marked increase for the age group 45 to 49 years in both genders (Bradshaw et al., 2007:702). After taking various factors into consideration, Bradshaw et al. (2007:702) reported that 4,3% of all deaths were

attributed to DM in South Africa in 2000, which makes it the seventh leading cause of death (Bradshaw et al., 2007:702), unlike the figure they mentioned earlier of 2,6%, which excluded excess deaths resulting from increased mortality risk from DM-associated CVD or –renal failure (Bradshaw et al., 2007:700). Almost half (47,2%) of the 4,3% DM-related deaths occurred in persons younger than 65 years. The peak burden was for men aged between 45 and 59 years, and for women aged sixty to 69 years. More South African women (5,7%) than men (3,0%) died from DM in 2000 (Bradshaw et al., 2007:702).

(ii) Etiology

Bradshaw et al. (2007:703) states that the high mortality rate in comparison to the prevalence, might be linked to: suboptimal access to health care; inadequate blood glucose- and blood pressure control; infrequent examinations for DM-related complications; and lack of access to hypercholesterolemia treatment.

Among the Indian South African population, peripheral insulin resistance and type-2 DM are more prevalent than in white South Africans. There is a high degree of acquired insulin resistance among obese black urban women, and insulin resistance is also an independent feature of essential hypertension among black South Africans. This type of hypertension is usually partly corrected by administration of long-acting angiotensin-converting enzyme (ACE) inhibitors (Van der Merwe & Pepper, 2006:3). Insulinopenia is related to a reduced β-cell mass, whereas insulin resistance is more a consequence of obesity. Both insulinopenia and insulin resistance are involved in type 2 diabetic black South Africans (see Table 2.17) (Van der Merwe, 2001:5, Table 1). Insulin resistance appears to be an important factor in the black South African population, especially in those presenting with hypertension and obesity, together with DM (Kruger et al., 2005:495). As mentioned earlier, the elevated FFA in the black population may be due to the reduced anti-lipolytic effect of insulin on adipocytes, which in turn may down-regulate the insulin receptors (Van der Merwe & Pepper, 2006:3). Taking all these metabolic effects into account may explain why obese black patients are predisposed to type 2 DM. It is interesting to note that the pathogenesis and clinic presentation of type 2 DM among black South Africans resembles type 1 DM, which explains why this population can present with diabetic ketoacidosis by age forty (Van der Merwe & Pepper, 2006:3-4).

The risk of type 2 DM increases with the presence of maternal DM, gestational DM and intra-uterine growth retardation, especially when a rapid catch-up growth occurs (WHO/FAO, 2002:73). Babies born to mothers after they have developed DM have a three-fold higher risk of developing DM themselves (WHO/FAO, 2002:74).

Overweight and obesity are associated with an increased risk of type 2 DM, especially with central adiposity (Goedecke et al., 2007:69; WHO/FAO, 2002:73). Central adiposity is an important determinant of insulin resistance. Waist-to-hip ratio and waist circumference are more powerful determinants of subsequent risk of type 2 DM than BMI (WHO/FAO, 2002:74). Voluntary weight loss improves insulin sensitivity and reduces risk of progression from impaired glucose tolerance to type 2 DM (WHO/FAO, 2002:74). In the Nurses Health Study undertaken in the USA, the risk for DM increased forty-fold when BMI increased from 22 to 35 kg/m². In the Health Professional Follow-Up Study, the relative risk of developing DM was 42 in men with a BMI > 35 kg/m², as compared to those

with a BMI<23 kg/m². In the UK, the relative risk is 5,2 in men and 12,7 in women with a BMI>30 kg/m² (Goedecke et al., 2007:69).

Factors that may affect the risk of developing type 2 DM are summarized in Table 2.19. Less than half of people with DM are diagnosed in developing countries. The amount of complications and morbidities can rise more rapidly if diagnoses and adequate treatment are not done timely. Due to cultural misconceptions and chronic shortages, insulin is also not used adequately in many developing countries (WDF, 2006:online).

Although there is no convincing evidence for lifestyle modification to reduce the risk of developing type 1 DM, since it is associated with an absolute deficiency of insulin, resulting from an auto-immune destruction of the ß-cells of the pancreas, lifestyle modification is the cornerstone of both treatment and prevention of type 2 DM (WHO/FAO, 2002:72). According to the WDF (2006:online), eighty percent of type 2 DM cases are preventable with lifestyle modifications. Modifiable risk factors include: obesity and physical inactivity, while non-modifiable risk factors are: a family history of DM; older age; gestational DM; and ethnicity (Franz, 2004:797).

Table 2.19 Summary of strength of evidence on lifestyle factors and risk of developing type 2 DM (WHO/FAO, 2002:77, Table 9).

Evidence	Decreased risk	Increased risk
Convincing	Voluntary weight loss in overweight and obese people Physical activity	Overweight and obesity Abdominal obesity Physical inactivity Maternal diabetes
Probable	Fibre	Saturated fats Intra-uterine growth retardation
Possible	Omega 3 fatty acids Low glycemic index (GI) foods Exclusive breastfeeding	Total fat intake Trans fatty acids
Insufficient	Vitamin E Chromium Magnesium Moderate alcohol	Excess alcohol

The Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA) (2009:2) recommends weight loss of all overweight or obese individuals who have DM. An achievable and maintainable weight loss goal should be set. Even a moderate weight loss of five percent of body weight can produce significant health benefits. SEMDSA (2009:2) also recommends either a low-carbohydrate or low-fat energy restricted diet for a short period (up to one year). Regular physical activity of thirty to 45 minutes of moderate intensity, three to five times per week improves insulin resistance, lipid profile, glycemic control and blood pressure control, and is recommended to maintain weight loss and prevent weight gain. A gradual increase in duration and frequency is recommended.

2.6.3.3 Cardiovascular diseases

Cardiovascular diseases, (including hypertension, coronary heart disease (CHD), IHD, heart failure, and stroke) are one of the major contributors to the global burden of disease among the CDLs.

It should also be kept in mind that the large South African population with HIV/AIDS receiving anti-retroviral treatment have a high risk of developing metabolic syndrome, because of insulin resistance leading to lipodystrophy, which in turn leads to central adiposity or lipo-atrophy. These patients present with high triglycerides and low HDL, with an increased CVD risk, specifically IHD. Severe cases can

even present with chylomicronemia syndrome, compared with similar factors as seen in diabesity (Norman et al., 2007b:714).

(i) Incidence, prevalence and epidemiology

One-third of all deaths worldwide (15,3 million) are attributable to CVD (WHO/FAO, 2002:81). In the 1990s, the leading causes of DALYs in terms of CDLs were: IHD; cerebrovascular disease; COPD; and pulmonary disease (Murray & Lopez, 1997b:1498). In 1998, developing countries, low-income and middle-income countries accounted for 86% of the DALYs lost to CVD. Ten years ago the WHO/FAO predicted that an increased burden of CVD would mostly be on developing countries within the following two decades (WHO/FAO, 2002:81), especially among people aged thirty to 64 years of age (Seedat, 2007:317). In 2004, CVD was the leading cause of death in the world, especially among women; with CVD causing almost 32% of deaths in women and 27% in men (WHO:2008:8). Around 2007, 29,2% (or 16,7 million) of the total global deaths were from CVD. Around eighty percent of the deaths took place in low- and middle-income countries. In the world, at least twenty million people survive heart attacks and strokes annually (Seedat, 2007:317).

The CVD prevalence in America is highest in non-Hispanic blacks (about 41% for men and 45% for women). The prevalence is 34% in white men and 24% in white women. In comparison, the prevalence in Mexican-Americans (i.e. Hispanic) is the same for both genders (29%) . The prevalence also increases with age; with rates doubling between 35 and 44 years of age, and between 65 and 74 years of age (Krummel, 2008:834). The majority (82%) of coronary events in women are attributable to lifestyle factors, including: an unbalanced diet; inactivity; smoking and overweight (Krummel, 2004a:861).

Heart disease and stroke cause the most deaths in both sexes and in all races. CHD, expressed as MI, is the main form of heart disease responsible for CVD deaths (Krummel, 2008:834). In 1983, O'Keefe (1983:683) found that while there was an increased prevalence of MI in urbanized South African blacks, the rate was still only two percent of that of the white population.

Twenty-five percent of men and 26% of women were found to be hypertensive in the 1998 SADHS. The prevalence of obesity in hypertensive men was 19% and in hypertensive women it was 45%. The prevalence of obesity in hypertensive persons included in the 1998 SADHS was much higher than in the general population, where only nine percent men and 26% of women were obese. Hypertension was two-fold higher in obese participants than in normal weight participants and hypertension was more prevalent in black obese women (Steyn et al., 2008:376-377). This occurrence identifies the need to investigate the ever increasing rates of obesity in South Africans and, in particular, in black women (Steyn et al., 2008:376; DoH et al., 2002:215).

During 1990, stroke accounted for 7,35% of total deaths in South Africa, while IHD accounted for 4,86%. In 1995, stroke was the fourth highest cause of premature death among men aged between 45 and 59 years (6,3%) and the second highest cause among women of the same age (10,1%). IHD was the fifth highest cause of death in men (5,6%) and seventh highest in women (3,8%). Globally, this order is reversed (Vorster, 2002:240). High levels of LDL and/or low levels of HDL increase the risk of developing IHD. The most powerful lipid-profile predictors for IHD are apo-lipoproteins ApoA1 and

ApoB. Total cholesterol measurement is usually more effective as a dyslipidemic marker for risk of IHD, ischemic stroke, and other CVD. Levels of total cholesterol usually increase with age in westernized populations. A diet high in fat (particularly saturated fat from animal origin) as well as high in dietary cholesterol, and low in fiber, is associated with high total cholesterol levels (Norman et al., 2007b:708).

It was already noted in 1977, that atheroma with myocardial infarction (MI) was one of the "Western" diseases appearing most commonly among urban blacks in South Africa. The patients who presented with MI were all "Westernized" according to their diet; occupation; degree of physical activity; and the high prevalence of obesity; DM; hypertension and dyslipidemia (Seftel, 1977:122).

Currently, the predominant CVD among the black South African population is cerebrovascular disease (i.e. stroke). But, as total cholesterol levels increase, the pattern shifts to IHD. It is already reported that the incidence of IHD is increasing among the black population, especially in urban hospitals (Norman et al., 2007b:708-709).

Vorster (2002:239) postulates that black South Africans may be protected against IHD because of their serum lipid profiles (low total cholesterol and high HDL cholesterol), as well as their low homocysteine values. IHD was still rare among urban black South Africans in 1985, with an incidence of only ten per 100 000 in Soweto (Walker & Walker, 1985:1410). Mollentze et al. (1995:93) speculated that since IHD has a long incubation period of thirty to forty years, the development of hypercholesterolemia then noted in the black population of the Free State may point to an impending CVD epidemic in this black population in years to come. The risk factors for CVD are already prevalent in South Africa and are predicted to increase with growing urbanization of the black population (Norman et al., 2007b:708).

Steyn (2005b:253) also states that two recent studies have challenged the notion that black people in Africa are "immune" to the development of IHD. In the first study, the African data of the Interheart study compared first-time acute MI patients with controls similar in age and gender. It was found that more than eighty percent of the African participants were South African. The black, coloured and white subgroups were compared to the findings in the overall Interheart study (conducted in 52 countries) and it was found that the degree of association for each of the major risk factors with an acute MI among the African population, were consistent with that found in the global study. Five modifiable risk factors could be attributed to 89,2% of the African participants, most of whom were South African (Seedat, 2007:317; Steyn, 2005b:250). Acute MI risk increased with higher income and education levels in the black population (Seedat, 2007:317). The second study compared black African MI or angina patients with similar age and gender controls living in the same township outside Pretoria. The data of this study, which was collected two decades earlier than the Interheart study, showed an association between IHD, major CVD risk factors and a family history of CVD among urban black South Africans. The same risk factors and family histories were also associated with eye -, kidney - and peripheral damage (Steyn, 2005b:253).

However, the Burden of Disease study found that there is a "striking" difference between hypercholesterolemia prevalence rates in the black South African population as compared to the other three population groups (i.e. white, Indian, coloured). This was consistent with the lower levels of

urbanization among blacks, together with an incomplete adoption of the Western lifestyle. However, it was interesting to note that the differences were larger in older black participants than in the younger ones, which may suggest that the younger black participants were already moving towards adopting a Westernized diet and lifestyle (Norman et al., 2007b:712-713).

(ii) Etiology

Vorster (2002:240) predicts that IHD will remain the highest cause of death in the world by 2020 (as it was since 1990), while stroke will remain in second place. The South African Society of Obesity and Metabolism (SASOM) (2006a:online) predicts that by 2020, heart disease and depression caused by obesity will be the world's greatest disabilities. Seedat (2007:318) predicts that there will be a rise in CVD-related deaths over the next few years, including in Sub-Saharan Africa, due to: industrialization and urbanization; low fibre intake; and increased tobacco use; LBW in babies (producing metabolic abnormalities, i.e. the Barker theory); and a decline in infection-related deaths in infancy, childhood and adolescence. Murray and Lopez (1997b:1498) predict that CDL-related mortality will increase from 28,1 million deaths in 1990, to 49,7 million by 2020.

Overweight, central obesity, high blood pressure, dyslipidemia, DM and low cardio-respiratory fitness are all factors contributing to an increased risk for CVD (WHO/FAO, 2002:81). A high intake of saturated fats, salt and refined carbohydrates, together with a low intake of fruits and vegetables increase the risk for CVD (WHO/FAO, 2002:81). The consumption of myristic and palmitic acids, trans fatty acids, and high alcohol intake also contribute to an increased risk (WHO/FAO, 2002:82). Intake of dietary cholesterol and unfiltered boiled coffee may probably increase the risk (WHO/FAO, 2002:82).

According to Seedat (2007:317), the most important risk factors for CVD are: hypertension; dyslipidemia DM and tobacco use. The risk factors which are the most modifiable are: high salt intake; increased body weight; and physical inactivity (Seedat, 2007:317). Category I risk factors for which interventions have been proven to lower CVD risk are: cigarette smoking; LDL cholesterol; high fat or cholesterol intake; hypertension; left ventricular hypertrophy; and thrombogenic factors. Category II risk factors for which interventions are likely to lower CVD risk are; DM; physical inactivity; HDL cholesterol; triglycerides; obesity; and postmenopausal status. Category III risk factors, which are associated with increased CVD risk and may lower risk if modified are: psycho-social factors; lipoprotein (a); homocysteine; oxidative stress; and no alcohol consumption. Category IV risk factors associated with increased CVD risk, which are non-modifiable, are: age; male gender; low socio-economic status; and family history of early-onset CVD (Krummel, 2004a:873, Table 35-6).

A number of cardiovascular risk factors are associated with obesity (Vorster & Kruger, 2007:323; Vorster et al., 2005:online). The strength of evidence on lifestyle factors and the risk of developing CVD as set out by the WHO and FAO, are summarized in Table 2.20.

As far as the prevalence of risk factors for CVD in South Africans is concerned: 10,3% of black men and 13% of black women have hypertension. Almost eight percent of black men are obese as opposed to 30,5% of black women (Seedat, 2007:318; Vorster, 2002:241, Table 1). As for dyslipidemia, there is a prevalence of 4,2% in black men aged between 45 to 54 years, and 4,7% of black women in the same age group (Vorster, 2002:241, Table 1).

Table 2.20. Summary of strength of evidence on lifestyle factors and risk of developing CVD (WHO/FAO, 2002:88, Table 10).

Evidence	*Decreased risk*	*No relationship*	*Increased risk*
Convincing	Regular physical activity Linoleic acid Fish and fish oils (EPA and DHA) Vegetables and fruit (incl. berries) Potassium Low to moderate alcohol intake (for CHD)	Vitamin E supplements	Myristic and palmitic acid Trans fatty acids High sodium intake Overweight High alcohol intake
Probable	Alpha linolenic acid Oleic acid Fibre Wholegrain cereals Nuts Plant sterols/stanols Folate	Stearic acid	Dietary cholesterol Unfiltered boiled coffee
Possible	Flavonoids Soy products		Fats rich in lauric acid Impaired fetal nutrition Beta-carotene supplements
Insufficient	Calcium Magnesium Vitamin C		Carbohydrates Iron

The high prevalence of hypertension, obesity and hypercholesterolemia among South Africans may explain the higher prevalence of CVD (Steyn et al., 2006:25). In the THUSA study, the effects of urbanization on some CVD risk factors could clearly be seen. Men in rural areas had a mean BMI of 20,7 kg/m², whereas in urban men the mean BMI was 23,1 kg/m². For the women, the same trend could be seen, where the BMI of 25,6 kg/m² for rural women increased to 28,1 kg/m² in urban women. Total serum cholesterol showed the same trend, with a mean value of 3,9 mmol/l in rural men as opposed to 4,7 mmol/l in urban men. Again, the same was seen for women with an increase from 4,0 mmol/l in rural women to 4,8 mmol/l in urban women (Vorster, 2002:241, Table 2). The total serum cholesterol values were still within the limit of <5 mmol/l, but an increase of between 0,6 and 0,8 mmol/l was already present. An even higher increase in serum cholesterol can be anticipated with a longer periods of urbanization.

Table 2.21 sets out the Interheart study's comparison of the three ethnic groups' risk factor profiles compared with the black group. This shows that the black group is at an earlier epidemiological transition than the other groups. Self-reported hypertension was much higher in the MI group of black participants (50,4%), than the controls (13,1%). For the white ("European") or Indian ("other Africans") group, hypertension was self-reported in 34,4% of MI cases as opposed to only 13,3% of the controls. The occurrence was not so pronounced among coloured Africans, where self-reported hypertension was about double in the MI cases (41,7%) as compared with the control group (26,5%). DM was also more pronounced among the black population, with 23,6% of MI cases having DM in comparison to only four percent of the control group. Again, the difference was less pronounced among the coloured group (23,4% vs. 11,5%) than the white/Indian ("European/other African") group (24,0% vs. 7,6%).

Interestingly, there was minimal difference for fruit and vegetable intake among the control groups and MI cases, except for the European/other Africans, where fruit and vegetable consumption was lower in the MI cases group (43,86%) as compared to the controls (50%).

Table 2.21. Comparison of CVD risk factors between patients with acute MI and controls in three ethnic groups participating in the African countries (Steyn, 2005b:251, Table 18.1 - amended)

	Blacks		***Coloureds***		***Whites and other***	
Characteristics	**Controls %**	**MI cases %**	**Controls %**	**MI cases %**	**Controls %**	**MI cases %**
Male gender	61.9	63.9	63.9	62.5	76.2	79.5
Self-reported hypertension	13.1	50.4	6.5	41.7	13.3	34.4
Diabetes	4.0	23.5	11.5	23.4	7.6	24.0
Current smoker	29.8	26.6	46.2	64.6	39.1	51.6
Current/former smoker	38.4	45.3	74.9	83.6	55.2	75.4
Physical activity	15.9	17.7	13.3	11.2	32.4	20.6
Alcohol	27.3	32.4	24.2	18.0	33.3	20.8
Fruits and vegetables daily	39.4	37.0	36.2	35.0	50.0	43.9

Continuous variables	**Controls** Mean (SD)	**MI cases** Mean (SD)	P value	**Controls** Mean (SD)	**MI cases** Mean (SD)	P value	**Controls** Mean (SD)	**MI cases** Mean (SD)	P value
Age	50.6	53.0	0.03	53.5	54.6	0.21	53.3	54.9	0.28
BMI	26.8	28.4	0.003	26.9	27.4	0.31	26.2	27.2	0.14
Waist-to-hip ratio	0.9	0.92	0.01	0.92	0.96	<0.0001	0.92	0.98	<0.0001

It is important to note that the only tools that are currently available to predict the probability of a person suffering a CVD event over a ten year period, are various formulae (like the Framingham total risk formula). The drawbacks of these formulae are that they were developed in mostly Western populations from long-term cohort studies and may not be applicable to people from Africa. These formulae also require blood testing for CDL risk factors, which are costly and frequently cannot be afforded by the public healthcare services in South Africa (Steyn, 2005b:250).

Higher intake of fruits; vegetables; fish and fish oils (rich in eicosapentanoic acid and docosahexanoic acid); foods high in linoleic acid and potassium; and moderate alcohol intake may reduce the risk of CVD (WHO/FAO, 2002:81). Intake of alpha-linolenic acid, oleic acid, fibre, wholegrain cereals, nuts, folate, and plant sterols and stanols may all probably decrease the risk. There is a possible association of lower risk with intake of flavonoids and soy products, while there is a possible association for higher risk within intake of fats rich in lauric acid, ß-carotene supplements and impaired fetal nutrition (WHO/FAO, 2002:81). Intake of trans fatty acids from hydrogenated oils and fats should be very low (less than one percent of total energy) and this is especially relevant in developing countries where low-cost hydrogenated fat is frequently consumed (WHO/FAO, 2002:89). Daily total fat intake should fall within the recommendations as set out by the 2010 dietary guidelines for Americans, as discussed earlier.

2.6.3.4. Hypertension

In 1977, hypertension was already extremely common among the South African population. After violence, it appeared to be the next important cause of death in black urban adults. At that time, hypertension-related deaths were the highest among adults in their thirties or forties. Severe hypertension appeared to be very rare in rural black population in 1977. The severity of hypertension in urban blacks were compared to that of the black American population, in whom the disease was known to be major public health problem and also more common and serious than in their white counterparts (Seftel, 1977:122).

Hypertension is responsible for about sixty percent of the burden of CVD and about half of the CHD burden globally (Seedat, 2007:317). High blood pressure is a major risk factor for CHD and stroke (ischemic and hemorrhagic) (WHO/FAO, 2002:85). According to Seedat (2007:318), congestive heart

failure is the most common cardiac manifestation of hypertension in the Sub-Saharan African population, although CHD is still relatively uncommon, due to low serum cholesterol levels and high HDL levels. Atheroma (of the aorta and cerebral arteries) and renal failure (from malignant hypertension) are common (Seedat, 2007:318).

(i) Incidence, prevalence and epidemiology

Steyn (2005b:250) states that it is not surprising that a quarter of South Africans have hypertension, since there are high rates of risk factors for hypertension, including: obesity; excessive alcohol use; high intake of sodium and low intake of potassium. In the 1998 SADHS, it was found that 13% of the men in South Africa have hypertension and 16% of women. Of these, only ten percent of men and 18% of women have their blood pressure under control (DoH et al., 2002:264). In the 1998 SADHS, 27% of men and 32% of women reported a family history of hypertension (DoH et al., 2002:168). The national prevalence rate of 21%, is equivalent to other industrialized countries and greater than many developing countries. It is likely that this rate will increase in the black population (currently 23,5% in men and 25% women) because of a more Westernized lifestyle (Bourne et al., 2002:161). It appears that the incidence of hypertension has increased dramatically in the black population, as well as stroke (Steyn et al., 2006:26).

Van der Merwe and Pepper (2006:3) state that hypertension prevalence is highest among black South Africans, where it is a risk factor for stroke. The inverse was found among data from three different studies: the Cape Peninsula study among the coloured community; the CORIS study conducted in rural Western Cape; and the BRISK study (black urban Cape Peninsula community). These studies showed that the differences in hypertension prevalence were not based on race, but on differences in "other socio-demographic parameters" (Steyn et al., 2008:378).

Hypertension is more common in urban than in rural South Africa. This also seems to be the case in Kenya, where hypertension was associated with a rise in blood pressure in migrants moving from rural to urban areas (Seedat, 2007:317). It was found in Kenya that rural blacks' hypertension risk was significantly lower than that of urban black, coloured and white participants (independent of other socio-demographic and risk factors) due to urbanization and the nutrition transition. Rural Kenyan men migrating to the cities were exposed to factors associated with developing hypertension, including: consuming more salt; less physical activity; and having more stressful life (Steyn et al., 2008:378). The urban-rural differences were more pronounced among black women. Rural black women appear to be more protected against having hypertension than black men (Steyn et al., 2008:378).

Obesity significantly increases the risk of hypertension (Goedecke et al., 2005:69). Obesity is associated with hypertension in the black South African population, where eight percent of black males and 30,5% of black females are obese (Seedat, 2007:318; Vorster, 2002:241, Table 1; DoH et al., 2002; 244). However, USA data shows that obesity has a lesser effect on black American women than white American women. Obese black American women had a lower prevalence of risk factors for cerebrovascular disease and lower mortality than white American women. Obese black women have fewer health disadvantages than obese white women, although there is no difference in risk for hypertension, CHD or breast cancer among the races (Steyn et al., 2008:379). Steyn et al. (2008:379) found no significant differences in hypertension risk between obese black Africans and other obese

women compared with women of normal weight. However, obese blacks are predisposed to hypertension, glucose intolerance and DM, since 35-47% of type 2 diabetic black South African women and 15-16% of black men, who were attending primary care services in both rural and urban areas, were obese (Nthangeni et al., 2002:329). In Kwa-Zulu Natal, 37% of the diabetic patients attending a district public health service were obese (Rotchford & Rotchford, 2002:538).

In America, obesity was associated with a relative adjusted risk of 3,5 for hypertension (Goedecke et al., 2005:69). Among the black South African population, Steyn (2005b:250) found high rates of risk factors for hypertension, including: obesity; excessive alcohol use; high sodium intake; and low potassium intake. Seedat (2007:317) reports that the high prevalence of hypertension in urban black Kenyan subjects was due to: obesity; increased peripheral vascular resistance; sodium hypersensitivity; cellular abnormalities; increased epithelial sodium channels; different genes controlling the renin-angiotensin-aldosterone system; low plasma rennin values; low socio-economic status and the "underweight phenotype." Krummel (2004b:905, Box 36-2) and Krummel (2008:868, Box 33-1) state that risk factors for developing hypertension, include: family history of hypertension; black race; youth; male gender; persistent diastolic pressure >115 mmHg; smoking; DM; hypercholesterolemia; overweight/obesity; excessive salt consumption; physical inactivity; excessive alcohol consumption and evidence of end organ damage.

In 1995, Mollentze et al. (1995:93) found that the rates of high blood pressure were higher in the urban Free State than in the rural areas, as the population aged. They found that the prevalence of hypertension was 29% in rural Free State and 30,3% in the urban Free State, according to WHO guidelines. More rural women (34,5%) than rural men (22,1%) were hypertensive, and the same was true for urban women (36,3%) as compared to urban men (22,8%). The prevalence of high risk hypertension (≥160/95 mmHg) increased with age, peaking at 43,4% for elderly QwaQwa (rural) men and 66% for women; and sixty percent for elderly Mangaung (urban) men and 78,1% for women (Mollentze et al., 1995:93).

The 1998 SADHS found the lowest prevalence of hypertension in black men (20,2%) and the highest in white men (38%). Coloured women, white women and Indian men had a high prevalence (close to 30%). White women and Indians controlled their hypertension far better than black men and coloured women, of which less than ten percent had their hypertension under control (Steyn et al., 2006:25). In 1998, the prevalence of hypertension in blacks was 24,4% (Bourne et al., 2002:157; DoH et al., 2002:210), with a 23,5% prevalence amongst men and 25% prevalence amongst women (Bourne et al., 2002:160; DoH et al., 2002:210). In the THUSA study, Vorster (2002:240) found hypertension prevalence to be the highest among black women and white men; more than twenty percent of "apparently healthy" subjects had hypertension. The highest prevalence (32,9%) was seen in informal-settlements in urban areas (Vorster, 2002:241).

In 1998, approximately 21% of adult South Africans (all races together) had hypertension (Seedat, 2007:319; Goedecke et al., 2005:69). Only about twenty percent of black men were aware that they had hypertension. Fourteen percent were on treatment and seven percent had their hypertension under control. Forty-seven percent of females were aware of their hypertension. Twenty-nine percent were on treatment and 15% had their hypertension under control (Seedat, 2007:319). Coloured men

had a lower risk of developing hypertension than black or white men, while rural black women had a significantly decreased risk of hypertension, as compared to white women (Goedecke et al., 2005:69).

The estimated prevalence of hypertension in 2000 in Sub-Saharan Africa was about 41,6 million men and 38,2 million women. Globally it is about 950 million people out of a population of 6,2 billion, with 65% coming from the developing world (Seedat, 2007:318).

(ii) Etiology

A study conducted in the USA compared the underlying factors that contributed to the higher prevalence of hypertension in black African-Americans with those in white Americans. Hypertension prevalence among blacks increased with age. Genetic factors played a lesser role than environmental factors. Even though 75% of Jamaican and African-American blacks' ancestry are from African populations (mostly similar to Nigerians), the hypertension prevalence in blacks was only 16% in West Africa, 26% in the Caribbean, and 33% in the USA. The higher prevalence in the USA could be attributed to: obesity; a high salt/low potassium diet; psycho-social stress; physical inactivity and high alcohol intake (Steyn et al., 2008:379).

The Dietary Approaches to Stop Hypertension (DASH) requires many dietary behavioural changes, including: eating twice the average amount of fruits, vegetables and dairy daily; reducing the intake of meat to one-third of usual intake; halving the use of fats; and reducing to one-quarter the amount of snacks and sweets (Krummel, 2004b:905). The DASH diet was found to be very effective in black Americans, but poverty and cultural factors may hinder the implementation of this high-fruit, high-vegetable, low-salt diet in South Africa (Seedat, 2007:318). To achieve eight to ten servings a day, two to three fruit- and vegetable servings should be consumed with every meal (Krummel, 2004b:905).

2.6.3.5 Stroke

In the 1984, the incidence of stroke in the urban South African black population was 1,01 per 1000 annually with a peak of 10,31 per 1000 annually for men aged 65 to 74 years. Hypertension was present in 69,8% of stroke patients (Rosman, 1986:668, Table 1).

In 2000, there were about 79,8 million people with stroke in Sub-Saharan Africa. This incidence is projected to rise to 150,9 million by 2025 (Seedat, 2007:318).

Stroke seems to be the main contributor to CVD in black South Africans, as has also been seen in black Americans, and is a major public health problem for black South Africans. When comparing available mortality figures for 1984 to 1986 for some CDLs in South Africa, stroke had the highest mortality rate in the black population, followed by hypertension, DM and IHD (Vorster, 2002:239-240). In the coloured population, there was high mortality from stroke and IHD, followed by DM and hypertension. Vorster (2002:239) suggests that urbanization in blacks may be characterized by early emergence of stroke. Even in rural areas, the prevalence of stroke was relatively high, with a rate approaching that of the high-income countries (Seedat, 2007:318).

2.6.3.6 Coronary heart disease and dyslipidemia

Atherosclerosis describes the disease of “thickened and narrowed arterial walls in arteries, due to inflammation, accompanied by accumulation of oxidized cholesterol,” due to hypercholesterolemia (i.e. dyslipidemia) (Krummel, 2008:833).

(i) Incidence, prevalence and epidemiology

In 1992, about 4,8 million South African presented with hypercholesterolemia, including 3,1 million presenting with raised LDL levels. Both of these conditions increase the risk for CHD (Goedecke et al., 2005:69).

Hypercholesterolemia caused about 4,6% of all deaths in South Africa in 2000. It contributed to only 1,4% of all DALYs because cholesterol-related CVD events usually occurred more in middle or old age. There were more hypercholesterolemia-related deaths among females than males. The deaths from ischemic stroke were double in females than in males. Mortality rates from hypercholesterolemia were higher in Indian- and white males than females, but the reverse was true for the black and coloured groups (Norman et al., 2007b:708, 712).

The cut-off points for blood lipid levels are: total cholesterol <5.2 mmol/l; LDL-cholesterol <3.4 mmol/l; HDL-cholesterol >1.2 mmol/l (females) and >1.0 mmol/l (males); and total triglycerides <1.7 mmol/l (Mayo Foundation for Medical Education and Research, 2011:online). In a study conducted in the Free State, Mollentze et al. (1995:92-93) found that mean total cholesterol levels for rural men aged 25 to 34 years was 4,7 mmol/l. This value remained about the same with increasing age. In urban men the mean level was also 4,7 mmol/l, but this value increased with age to 5,3 mmol/l (by age 65 years and older). In women, the mean total cholesterol was 4,4 mmol/l for both rural (QwaQwa) and urban (Mangaung) areas. However, there was an increase in both areas with an increase in age. For the rural area the value increased to 5,2 mmol/l and for the urban area it was 5,7 mmol/l. HDL levels were 1,2 mmol/l for rural and 1,4 mmol/l for urban men aged between 25 and 34 years, and did not increase with age. This pattern was also evident in women. However, an increase in LDL levels was noted in middle-aged women in both rural and urban areas. Fasting triglyceride levels peaked at 1,7 mmol/l for rural men aged 35 to 44 years. For urban men, the value peaked at 1,8 mmol/l for men aged 45 to 54 years. Values also tended to increase for women in both urban and rural areas.

In the 1998 SADHS, white men had the highest mean total cholesterol, and black women the lowest. Men of all other racial groups had mean total cholesterol above 5,2 mmol/l, except black men. Thus, all the races of South Africa have a high risk for developing CHD, except blacks (Steyn et al., 2006:25).

When comparing the results of the South African Burden of Disease in 2000 with worldwide values, 56% of IHD mortality and disease burden worldwide was attributable to hypercholesterolemia (>3,8 mmol/l) as compared to 59% in South Africa. Worldwide, 32% of ischemic stroke cases were attributed to hypercholesterolemia, compared to 29% in South Africa (26% in blacks and 48% in whites). The highest amount of hypercholesterolemic-related deaths and DALYs worldwide were found in European sub-regions, where the highest mean cholesterol levels were found (Norman et al., 2007b:712).

(ii) Etiology

Catch-up growth between birth and seven years of age is associated with an increased risk for the development of CHD (Forsen et al., 2000:180). The risk for CHD increases with obesity, with a relative risk of approximately 2,8 for men and 3,4 for women. The risk of developing CHD amongst American women increased by 3,3-fold when their BMI was >29 kg/m² (Goedecke et al., 2005:69).

Numerous modifiable factors affect serum cholesterol levels, such as: age; diets high in saturated fats and cholesterol; genetics; endogenous and exogenous hormones; body weight, glucose tolerance; physical activity levels and the presence of other CDLs (like DM) (Krummel, 2008:837-838; Krummel, 2004a:867). Dyslipidemia among the black population of Sub-Saharan Africa is still low. Thus it is not a major risk factor for CVD yet in this population, but it is on the increase (Seedat, 2007:317). Black South Africans are less likely to develop hypercholesterolemia and raised LDL levels, even though they have a higher prevalence of obesity. In 1993, the prevalence of CHD was only 2,4% in an urban Zulu population. In a 2004 study among peri-urban South Africans with "mixed ancestry," hypercholesterolemia was present in almost half of the population (47% of men and 46% of women) (Goedecke et al., 2005:69). In 2000, the Burden of Disease study estimated that about 59% of IHD and 29% of ischemic stroke burden in adult males and females older than thirty years were attributable to hypercholesterolemia (≥ 3.8 mmol/l) (Norman et al., 2007b:708, 712).

The slower emergence of IHD among black South Africans (when compared to stroke), could be explained by their advantageous lipid profiles (low total cholesterol, high HDL and low homocysteine levels) (Van der Merwe & Pepper,2006:2; Vorster, 2002:242). Black South African men and women had low mean serum total cholesterol levels (Vorster, 2002:240). Almost all of the black population had HDL levels above twenty percent of total cholesterol, which indicates a protective effect against IHD (Seftel et al., 1995:63). The cholesterol values in women were higher than those in men, probably because they had higher BMIs (Vorster, 2002:241). Between 1984 and 1986, the highest IHD mortality rates were among the white and Indian populations, followed by stroke, DM and hypertension (Vorster, 2002:239). Serum cholesterol increased in the black population with urbanization, but HDL levels remained high. Low homocysteine levels in black South Africans due to genetics, may have a protective effect against CHD (Vorster, 2002:242). However, Van der Merwe and Pepper (2006:2) state that genetic predisposition remain to be established.

When taking the above into consideration, it is clear why white South Africans have such a high prevalence of hypercholesterolemia, since they are mostly from European descent. As mentioned earlier, it should be kept in mind that there are genetic disorders that contribute to CVD, including essential hypertension and familial hypercholesterolemia. Familial hypercholesterolemia is an autosomal dominant disease where affected persons develop CHD by age forty to fifty years. South Africans have the highest reported frequency of this genetic disorder in the world (1 in 71 persons). In Europe, Japan and the USA, the frequency is one in 500. In regions of Quebec, Canada, the frequency is one in 122 (Cummings, 2006:110). By extrapolating the Burden of Disease study's data to all South Africans older than thirty years, it is estimated that about eight million people have a risk for CDLs due to hypercholesterolemia (Norman et al., 2007b:713). This amounts to about one in every

five South Africans; about 17,9% of the total South African population of 44,8 million (Statistics South Africa, 2003).

2.6.3.7 Cancer

Cancer is a largely preventable disease, since patterns of cancer incidence are altered mainly by environmental factors, and mainly genetic factors (WCRF/WICR, 2007:4).

(i) Etiology and risk factors

According to the WCRF/AICR (2007:2-3), cancer is a disease of the genes, although epidemiological and experimental evidence show that only a small amount of cancers are inherited. Mutations in genes (either harmful or beneficial) are caused by different factors during a lifespan. Nutrition plays a large role in determining the likelihood of some mutations. Other modifiable environmental factors, including smoking; tobacco use; infectious agents; radiation; industrial chemicals; pollution; medication; and physical activity, also play a large role in mutations and, therefore, cancer risk.

As figure 2.4 demonstrates, there are multiple factors involved in the development and progression of cancer. Dietary intake is not the only risk factor for cancer (Steyn, 2005b:253). The most important established environmental cause of cancer is tobacco smoking. Other important determinants of cancer risk are: diet (especially alcohol and salted fish), body weight, physical activity, infections, hormonal factors and radiation (Table 2.22). Cancer as a cause of death is increasing, because the proportion of older people is increasing (WHO/FAO, 2002:95). Diet is estimated to account for approximately thirty percent of cancers in industrialized countries (second only to tobacco use) and about twenty percent in developing countries (Eldridge, 2004:1000, WHO/FAO, 2002:95).

Certain lifestyle factors that are linked to the risk of developing cancer are set out in Table 2.22. There is convincing evidence that there is an increased cancer risk with overweight and obesity, high consumption of alcoholic beverages, aflatoxins, and some forms of salted and fermented fish (WHO/FAO, 2002:95).

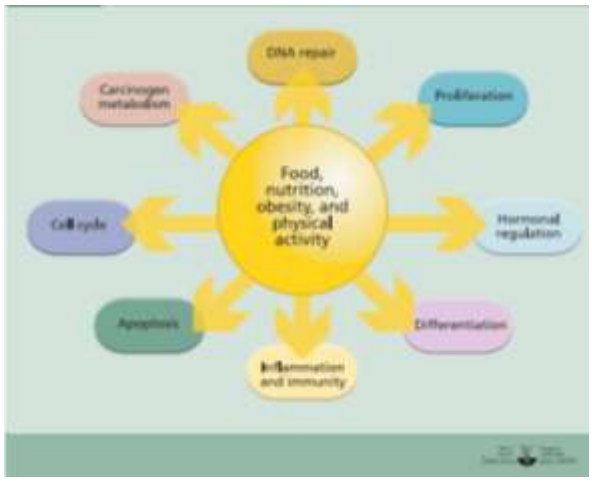


Figure 2.4 Factors which may promote or inhibit cancer development and progression (WCRF/AICR, 2007:33, Figure 2.2).

Salted meats usually contain nitrates and nitrites, which are associated with nitrosamines (potent carcinogens) (Grant, 2008:966; Eldridge, 2004:1003). Dietary factors which probably increase a risk of

cancer include: high intake of preserved meats; salt-preserved foods; salt; very hot drinks and food. Consumption of fruit and vegetables are probable protective factors (WHO/FAO, 2002:96).

Table 2.22 Summary of strength of evidence on lifestyle factors and the risk of developing cancer (Adapted from WHO/FAO, 2002:100, Table 11).

Evidence	***Decreased risk***	***Increased risk***
Convincing	Physical activity (colon)	Overweight and obesity (Esophageal, colorectal, breast in post-menopausal women, endometrium, kidney) Alcohol (oral, pharynx, larynx, esophagus, liver, breast) Aflatoxin (liver) Chinese-style salted fish (naso-pharynx)
Probable	Fruits and vegetables (oral, esophageal, stomach, colorectal) Physical activity (breast)	Preserved meat (colorectal) Salt-preserved foods and salt (stomach) Very hot drinks and food (oral, pharynx and esophagus)
Possible/insufficient	Fibre Soya Fish Omega 3 fatty acids Carotenoids Vitamin B2, B6, folate, B12, C, D, E Calcium, zinc and selenium Non-nutrient plant constituents (allium compounds, flavonoids, isoflavones and lignans)	Animal fats Hetero-cyclic amines Polo-cyclic aromatic hydrocarbons Nitrosamines

Overweight and obesity appear to be the most important avoidable risk factor for cancer, after tobacco use (WHO/FAO, 2002:96). People carrying genes that predispose them to obesity, have a higher risk for obesity-related cancers (WCRF/WICR, 2007:37; Goedecke et al., 2005:69), especially cancers of the colon; rectum; esophagus; gall bladder; breast; endometrium; pancreas; and kidney (Eldridge, 2004:1001). Overweight and obesity may account for up to thirty percent of kidney cancers in both men and women (WHO/FAO, 2002:100). Maintenance of a healthy weight throughout life may be one of the most important ways to protect against cancer. As also previously discussed, a healthy weight also protects against a number of other common CDLs (WCRF/AICR, 2007:xvii).

Obesity in women is especially associated with cancer in the reproductive system, since excess body fat causes overproduction of estrogen in adipose cells (Goedecke et al., 2005:69). An association between glycemic load (GL) of food and fructose intake with pancreatic cancer risk was seen in women with a BMI>25kg/m^2; with low physical activity and impaired glucose metabolism. A high GL in the diet may increase pancreatic cancer risk in women if they already have an underlying degree of insulin resistance (Eldridge, 2004:1001).

Obesity in men is associated with an increased risk for developing colorectal- and prostate cancer (Goedecke et al., 2005:69), as well as cancer in the esophagus; stomach; small intestine; colon; rectum; gallbladder; breast; prostate; bladder; and thyroid; as well as melanoma; multiple myeloma and leukemia (Grant, 2008:963).

As discussed earlier, lower birth weight, followed by rapid growth is associated with the development of metabolic syndrome in adulthood, of which type 2 DM forms a part. Type 2 DM has been associated with increased breast cancer (Augustin et al., 2001:1533). Metabolic syndrome and the inflammatory state of obesity may also increase a person's risk for developing cancer (WCRF/AICR, 2007:34).

Individuals who possess certain genotypes are predisposed to certain cancers, including: breast-; prostate-; lung-; and colon cancers, with nutrition playing an influential role. During cancer, mutations occur both in oncogenes and tumour suppressor genes. Activation of oncogenes, together with inactivation of tumour suppressor genes, has a large influence on cancer progression (WCRF/AICR, 2007:35). For example, a single nucleotide polymorphism (SNP) in the methylenetetrahydrofolate reductase (MTHFR) gene influences folate metabolism by reducing activity of MTHFR, which reduced colorectal cancer risk. Other SNPs in the N-acetyltransferase gene influence enzyme activity during the activation of heterocyclic amines from meat cooked at high temperatures, which may increase colon cancer risk (WCRF/AICR, 2007:37). People who eat large amounts of red- and processed meats who also have the CYP2E1 gene, may have a greater risk of rectal cancer. Eating cruciferous vegetables protects individuals lacking the STM1 gene against lung cancer (WCRF/AICR, 2007:37).

Migration contributes to the increase in incidence of certain cancers and the decrease of other cancers (WCRF/AICR, 2007:24). When people migrate from a low-risk to a high-risk area of the world, the incidence of colorectal cancer increases quickly, e.g. the incidence rate for colorectal cancer is higher in Japanese people born in USA than white people born there (WCRF/AICR 2007:20). Breast cancer rates change when women move to a new country (WCRF/AICR, 2007:21). More recent studies show that cancer incidence among second-generation immigrants becomes similar to those of people in the adopted country (WCRF/AICR, 2007:25). Therefore, environmental factors (food patterns, nutrition and physical activity) obviously have an impact on incidence during migration. Since the genetic pool of any population does not change dramatically within two generations, environmental exposure can influence deoxyribonucleic acid (DNA) damage and gene expression in a relatively short period, influencing cancer incidence (WCRF/AICR, 2007:25).

(ii) Incidence, prevalence and epidemiology

In 2000, over six million cancer deaths and an estimated ten million new cases occurred globally (WHO/FAO, 2002:95). In 2002, ten million new cancer cases were recorded worldwide (excluding non-melanoma skin cancer) and nearly seven million people died from cancer (WCRF/AICR, 2007:17).

In lower-income countries, like Africa, Asia and Latin America, the most common cancers are found in the: upper digestive tract; stomach; liver and cervix (WCRF/AICR, 2007:2-4). Liver cancer is the second leading cause of cancer death in Africa (WHO, 2008:12). Cancers of the mouth and oro-pharynx are the second leading cause of cancer deaths in South East Asia (WHO, 2008:12). Breast cancer is the most common cancer among women from low-income countries (WCRF/AICR, 2007:17). Prostate cancer is the leading cause of cancer deaths among men in Africa and in America's low- and middle-income countries (WHO, 2008:12).

In 2000, the South African National Burden of Disease Study found that cancer was the fourth leading cause of death for all South Africans, and the second leading cause of death among persons older than sixty years (Steyn et al., 2006:29). Steyn (2005b:253) states that neoplasms were the second most common cause of death in South Africa. In males, the leading cause of cancer death was trachea/bronchi/lung cancer (22,5%), followed by esophagus cancer (17,2%). In women, cervix was the leading cause of cancer deaths (17,9%), followed by breast- (15,7%) and lung (10,9%) cancer (Steyn et al., 2006:29).

As populations migrate around the world, and as countries become more urbanized and industrialized, patterns of cancer change. In general, cancer rates are expected to increase (WCRF/AICR, 2007:2). The total number of cancer cases in the developing world is predicted to increase by 73% between 2000 and 2020, and in the developed world by 29%, mostly because of the increase in number of older people (WHO/FAO, 2002:95). Cancer figures are estimated to rise to over 16 million new cases and ten million deaths in 2020, and more than twenty million new cases in 2030. Seventy percent of cancer deaths will be from low-income countries (WCRF/AICR, 2007:4, 17).

Various factors are taken into account when projecting these figures, including: increased global population; the aging world population; increase in tobacco smoking; the amount of people with HIV/AIDS; as well as improved screening tests and treatment for cancer, which will increase the number of cancer survivors (WCRF/AICR, 2007:17).

Although there are many different cancers, only cancers related to the nutrition transition will be briefly discussed.

(a) Esophagus

Worldwide, esophageal cancer is the seventh most common type of cancer. More than 460 000 new cases were recorded in 2002 (WCRF/AICR, 2007:17). It has a poor survival rate and therefore, is the fifth most common cause of cancer death (nearly 390 000 deaths in 2002) (WCRF/AICR, 2007:18).

Southern- and Eastern Africa, South-Central Asia, and some South-American countries all have a high risk for esophageal cancer (WCRF/AICR, 2007:18). This can be partly explained by geographical variability of exposure to carcinogens (WCRF/AICR, 2007:18-19). High rates of squamous cell esophageal carcinoma (SCEC) are found in China, central Asia, the Caspian littoral region of northern Iran, and the Eastern Cape region of South Africa (Churma & Horrell, 2005:45). WHO/FAO (2002:96) state that around sixty percent of esophageal-, oral- and pharyngeal cancers are thought to be due to micronutrient deficiencies from a diet low in fruit, vegetables and animal products. Churma and Horrell (2005:46) state that low levels of selenium, zinc, and low intakes of especially citrus fruit are associated with increased risk. Consuming very hot food and beverages also increases the risk of these cancers (WHO/FAO, 2002:96). Exposure to indoor smoke from solid fuels, and undernutrition contribute to the high rates of esophagus cancer in South African men (Steyn, 2005b:253).

The incidence of esophageal cancer in South Africa has increased since the 1950s. The risk is highest for the Eastern Cape population, particularly in rural areas. Since the mid-1980's, esophageal cancer incidence has decreased, but the reason is unclear. Unlike in the USA, the highest incidence of esophageal cancer is in the black population of South Africa. In 1995, esophageal cancer was the leading cause of cancer in black males, but was replaced by prostate cancer between 1996 and 1999, with esophageal cancer then being the second leading cancer with a lifetime risk of one in 59. Esophageal cancer was the third leading cancer in black women (10%) in 1999, and the lifetime risk was one in 113 (Steyn et al., 2006:30).

In 2003, mortality rates for esophageal cancer in South Africa were high, with 17,2% of all males dying of this cancer and ten percent of all females. It was the leading cause of cancer death in black South

African males and the second leading cause in black South African females. Rates steadily increased from age 35 to 44 years (Steyn et al., 2006:30).

(b) Colorectal

Colorectal cancer (CRC) is one of the most common occurring cancers in the Western world (Masino, 2005:29). CRC is the second most common malignancy in affluent societies in developing countries (Steyn et al., 2006:31). It is the third most common cancer worldwide, with over one million new cases in 2002 (WCRF/AICR 2007:20).

In the white South African population, CRC was the second leading cause of cancer deaths, with the age-standardized death rate being five times greater than in the black population It is the sixth leading cause of cancer deaths among South African men (5,3%) and the fifth leading cause in South African women (6,6%). In 2003, the rates among the white population were about 25,4 in 100 000 in men (more than eight times the rate of the black men) and 17,5 in 100 000 in women (seven time the rate of the black women). However, an increased risk was seen in younger black South Africans, because of a change in lifestyle and diet (Steyn et al., 2006:31).

There is no obvious cause for an estimated 65% to 85% of CRC cases. Another ten to thirty percent of cases are related to a family history of polyps or CRC. Another small percentage (6%) of cases are related to inherited syndromes like: non-polyposis CRC; familial adenomatous polyposis; and other rare syndromes (Masino, 2005:29). Smoking, meat intake and alcohol consumption are known risk factors. Consumption of fruit and vegetables and physical activity are protective (Steyn et al., 2006:31). Overweight/obesity, high energy intake and physical inactivity increase the risk of CRC in men and women (Masino, 2005:29-30; Eldridge, 2004:1000). Overweight and obesity are the best established diet-related risk factors (WHO/FAO, 2002:97). Hyperinsulinism, associated with obesity, may stimulate the growth of CRC (Masino, 2005:30). Some aspects of the Westernized diet are major determinants of risk. High intakes of meat, alcohol and fat may increase risk, and high intakes of fruits, vegetables, dietary fibre, folate and calcium may decrease the risk (Masino, 2005:30; WHO/FAO, 2002:97).

(c) Liver

Worldwide, liver cancer is the sixth most common type of cancer (625 000 new cases in 2002). Due to its poor prognosis, it is the third most common cause of cancer death (600 000 deaths in 2002) (WCRF/AICR, 2007:20). Seventy-five percent (WHO/FAO, 2002:98) to eighty percent (WCRF/AICR, 2007:20) of cases occur in developing countries. Fifty-five percent of all new cases are in China (WCRF/AICR, 2007:20). Rates are much higher in Sub-Saharan Africa and South-East Asia (WCRF/AICR, 2007:20; WHO/FAO, 2002:98) than in North America, Europe (WHO/FAO, 2002:98) and Latin America (WCRF/AICR, 2007:20). Japan and areas of southern Europe have intermediate incidence levels (WCRF/AICR, 2007:20).

The major risk factor for liver cancer is chronic infection with the hepatitis B or C viruses (WCRF/AICR, 2007:20; James & Kulakowski, 2005:117; WHO/FAO, 2002:98). Excessive alcohol consumption is the main diet-related risk factor in industrialized countries (WHO/FAO, 2002:98). Overweight and obesity possibly increase the risk. High intake of meat increases the risk, while high intakes of vegetables

reduces the risk (WHO/FAO, 2002:98). Ingestion of foods contaminated with mycotoxin (an aflatoxin), or aflatoxin B1 (a product of the *Aspergillus* fungus), increase the risk (James & Kulakowski, 2005:118; WHO/FAO, 2002:98). Aflatoxin is also found in water and stored food grains in countries with hot, humid climates (James & Kulakowski, 2005:118).

(d) Breast

The WRCF/AICR (2007:21) state that breast cancer is the third most common cancer in the world, while the WHO/FAO (2002:99) state that breast cancer is the second most common cancer in the world. However, they both agree that it is most common type of cancer in women (WCRF/AICR, 2007:21; WHO/FAO, 2002:99). It is the sixth most common cause of cancer deaths overall, but the second most common cause of cancer death in women, with 410 000 deaths in 2002 (WCRF/AICR, 2007:21).

In South Africa, black females aged between 35 and 44 years, have very similar age-specific death rates than those of white, coloured and Indian women. It is only the older aged black women that have much lower rates. This also applies to incidence (Steyn et al., 2006:30). In 1999, the incidence amongst black women was 18,4 per 100 000, as compared to 76,5 in 100 000 for coloured and white South African women (Steyn et al., 2006:31). In 2004, rural black South African women had a very low incidence of breast cancer (about ten in 100 000), whereas the incidence was higher in urban black women (15,1 in 100 000) (Goedecke et al., 2005:69). Breast cancer risk is associated with high socio-economic status (usually among women with high education or -income) (Steyn et al., 2006:31).

Inherited cancers only account for about five to ten percent of all breast cancer cases. In 1994 and 1995, two tumour suppressor genes (BRCA1 and BRCA2) were identified, which increase a woman's risk for breast cancer substantially. Women with these genetic mutations have a lifetime breast cancer risk of about 82% (Fletcher & Hayward, 2005:17). In these women, increased activity as a teenager and normal weight at menarche and at age 21 were associated with later onset of breast cancer (Fletcher & Hayward, 2005:17). Other risk factors for breast cancer not mentioned earlier include: smoking; and physical inactivity (Steyn et al., 2006:31). Therefore, diet and exercise are important risk reduction factors for these and other high-risk women (Fletcher & Hayward, 2005:17).

Obesity increases breast cancer risk in post-menopausal women by around fifty percent (WHO/FAO, 2002:99). As mentioned earlier, adipose tissue synthesizes estrogen and extended exposure to estrogen increases the risk of breast cancer (Fletcher & Hayward, 2005:16; Goedecke et al., 2005:69). Adipose tissue becomes the strongest contributor of estrogen following menopause (Fletcher & Hayward, 2005:16). A positive association with breast cancer is seen in post-menopausal women who gain weight, but an increased risk is also seen in relative leanness in pre-menopausal women (Eldridge, 2004:1000). The WHO/FAO (2002:99) state that obesity and alcohol intake are the only dietary factors which have been shown to increase breast cancer risk. There is a small increased risk with increasing alcohol consumption. An average consumption of one alcoholic drink per day increases the risk by about ten percent.

High fruit and vegetable intake has been shown to protect against cancer in general, but research specific to breast cancer is inconsistent. Apparently several studies found an increase in breast cancer

risk in vegetarians (Fletcher & Hayward, 2005:17). A recent meta-analysis study concluded that total dietary fat has a positive association with breast cancer risk. High intake of saturated fat, especially from red meat, contributed to an increased relative risk of cancer (Fletcher & Hayward, 2005:18).

(e) Endometrium

Endometrial cancer is the most commonly diagnosed gynecologic cancer in the USA, with the average age of diagnosis at sixty years. The highest incidence in the USA is among white women (Hamilton & Roche, 2005:87).

Extreme obesity, which leads to adipose retention of estrogen (as discussed under breast cancer) is a risk factor (Hamilton & Roche, 2005:88-89). Obese women have a three-fold higher risk for endometrial cancer (WHO/FAO, 2002:99). There is also an increased incidence of endometrial cancer in women with hypertension and coexisting DM (Hamilton & Roche, 2005:89). High consumption of fruits and vegetables may reduce risk; and diets high in saturated- and total fat increase risk (WHO/FAO, 2002:99).

(f) Prostate

Prostate cancer is the sixth most common cancer in the world, and the third most common cancer in men, with 680 000 new cases and 220 000 cancer deaths in 2002. It is the eighth most common cause of cancer deaths globally. The majority of cases are found among men older than 65 years. It is more common in high-income countries (except Japan). Screening programs have increased diagnosis rates, which consequently have influenced incidence rate (WCRF/AICR, 2007: 21), resulting in increased number of cases recorded in the USA in recent years, even though the incidence has declined in several high-income countries since the 1990s. Increased awareness and screening might also explain the increase in incidence in low-income countries, particularly in Latin American countries (Costa Rica, Columbia and Ecuador) and China (WCRF/AICR, 2007: 22).

Death from prostate cancer is about ten times more common in North America and Europe than in Asia (WCRF/AICR, 2007:22; WHO/FAO, 2002:99). However, it still only contributes to 5,8% of all cancer deaths in men (WCRF/AICR, 2007:22). Prostate cancer is positively associated with a Westernized diet. Inconsistent data exists for the influence of diets high in red meat, dairy and animal fat on the development of prostate cancer. Consistent evidence show that β-carotene supplements do not alter the risk of prostate cancer (WHO/FAO, 2002:99), but suggest that vitamin E and selenium might be protective (Eldridge, 2004:1003; WHO/FAO, 2002:99). Lycopene has been associated with a reduced risk, but again, data is inconsistent (WHO/FAO, 2002:99).

2.7. The cost of disease

The costs of CDLs are tremendous, not only in monetary terms, but also in mortality and quality of life; even more so in developing countries such as South Africa (Steyn et al., 2006:21; Goedecke et al., 2005:65; Kruger et al., 2005:492; WHO/FAO, 2002:4).

A disease can have a low incidence, but cause death and disability, which then results in a high burden of disease (with many life years lost) (WHO, 2008:29). The WHO/FAO (2002:61) estimated that the direct costs of obesity in the USA in 1995 accounted for 6,8% of total health care costs (US$70 billion or R560

billion) and physical inactivity for a further US$24 billion (R192 billion). The direct costs in other industrialized countries were slightly lower, but they still made out a noticeable part of national healthcare budgets. Indirect costs are usually far greater than direct costs and include: workdays lost; doctor visits; disability pensions; and premature mortality. Hidden costs were also enormous and include impaired quality of life.

Seedat (2007:316) has stated that the healthcare costs in many of the 54 countries in Sub-Saharan Africa are only around US$10 (R80) per person annually. This amount is completely insufficient to address the needs posed by the double burden of CDLs and infectious diseases, including AIDS (Seedat, 2007:319). In industrialized Western countries, the difference is noticeable, with costs amounting to between US$2 000 (R16 000) and US$5 000 (R40 000) annually. This is explained by the fact that Africa has a huge debt and interest on this debt is equal to twice the amount spent on healthcare and education (Seedat, 2007:316).

South Africa's financial health care resources are comparable to that of Brazil, Mexico and Thailand, but even though these countries spend similar amounts of their gross domestic product (3,5%) in the public sector, they have far better health outcomes than South Africa if life expectancy and maternal mortality are considered. South Africa's health expenditure is expected to rise on average by 7,1% per fiscal year between 2009 and 2013, with costs estimated around R100 billion for G70

2009/2010. Eighty-five percent of South Africa's population is dependent on the public sector. Currently the public sector expenditure is approximately R1600 (US$200) per person per annum, which is approximately four times the amount expended in other sub-Saharan countries. To save maternal and neonatal lives, South Africa has to spend two to four times more money than Cameroon or Southern Nigeria, due to a lack of input costs and human resources. South Africa's per capita expenditure on non-hospital primary care varies considerably by province and by district, ranging between R220 to R505 per person. Of course, these figures do not include the out-of-pocket expenses and direct costs to families and patients themselves. Nearly sixty percent of all poor South African households are found in rural areas, where access to health care is compromised. It was found that in 2006 that in a rural South African province, up to sixty percent of a patient's monthly income was spent on seeking health care, a large portion of which was used for transport costs (Hofman & Tollman, 2010:798). Therefore, the burden of disease is much higher on the poor.

Obesity can affect quality of life in many ways. A 55-year follow-up study of previously obese adolescents, showed that women who had been obese as adolescents were eight times more likely to report difficulty with activities of daily living than those who were lean in adolescence. Obesity in adolescence also had a negative impact on socio-cultural and economic factors, such as household income, self-esteem, marital status and education level, particularly in women (Goedecke et al., 2005:70). The health care expense for obesity and related illnesses is five- to eight percent of the health budget in most developed countries. The impact on indirect cost is considerable. For obese workers, long-term sick leave is up to 2,4 times higher than for their thinner counterparts (SASOM, 2006a:online). In South Africa, the rising prevalence of obesity causes serious concerns because of increased risk of DM and CVD. The direct costs may be as high as 6,8% of healthcare costs, and indirect and intangible costs should also be considered (Steyn et al., 2006:21).

The HIV/AIDS epidemic also has a high impact on health resources on services and health statistics in South Africa (Vorster & Kruger, 2007:321). CDLs dominate the mortality statistics for five out of six regions of the WHO. Africa is the only region where HIV/AIDS dominates instead (WHO/FAO, 2002:4). Hofman and Tollman (2010:799) suggest that the current, predominantly vertical HIV programs in South Africa should be changed to include more broadly the other chronic disease also, since the infectious illnesses are paralleled by a rising burden of CVD, DM and cancer.

The WHO estimates that it would take US$136 million (R1 088 million) per country annually to achieve eighty percent of coverage of basic healthcare. Currently there is a critical shortage in doctors, nurses and midwives. To achieve the eighty percent coverage rate, an average increase in healthcare expenditure of US$2,80 (R22,40) per person annually must occur. However, payment of salaries and emigration of trained workers are not included in these costs, which would amount to an additional US$311 million (R2 488 million). The staff shortage in Sub-Saharan Africa occurs due to a number of reasons, including: underproduction of qualified healthcare workers; internal mal-distribution of professionals; and emigration of trained workers (Seedat, 2007:317).

CDLs related to diet (obesity, DM, CVD, cancer, osteoporosis and dental diseases) present the greatest public health burden, in terms of direct cost to society and government, as well as DALYs (WHO/FAO, 2002:4). According to the WHO/FAO (2002:5), the public health implications of the increasing rate of overweight and obesity annually in most developing countries are “staggering.” Currently, almost half of the total CDL-related deaths are attributable to CVD, but obesity and DM are also showing concerning trends, since they affect a large proportion of the population, and already appear earlier in life. Developing countries are constantly exposed to increasing levels of public health problems related to CDLs (WHO/FAO, 2002:4).

The WHO/FAO estimates that in 2001, CDLs contributed to approximately sixty percent of the 56,5 million deaths in the world and approximately 46% of the global burden of disease. The proportion of the burden of CDLs is expected to increase to 57% by 2020, and will by then account for almost 75% of all deaths worldwide. It is predicted that the number of people in developing countries with DM will increase more than 2,5 fold by 2025 and that sixty percent of the burden of CDLs will occur in developing countries (WHO/FAO, 2002:4-5).

DM is an expensive chronic disease, because of the necessary means to control and prevent the severe complications. According to the WDF (2006:online), the economic and social costs for type 2 DM in the world are “enormous and escalating.” In most developing countries, mechanisms for financing health care are non-existent and health expenses are typically for the public’s own expense. Because the public is left with the choice between health care and food or clothing, financial constraints result in under-consumption of health services. About eighty percent of developing countries’ population pay for some or all of their medication. In Latin America, forty to sixty percent of DM costs are for the public’s own pocket. In India, as much as 25% of a family’s income may be spent on DM care. In 2002, DM costs amounted to US$132 billion (R1 056 billion) in the USA, of which direct costs were US$ 91,8 billion (R734,4 billion) and indirect costs US$39,8 billion (R318,4 billion). The total direct costs of medical care for a person with DM would be around US$13 243 (R105 944) annually, as compared to US$2 560 (R20 480) for a person without DM (Franz, 2008:766; Franz, 2004:794). It was estimated that in 2007, the world would spend

between US$215 billion (R1 720 billion) and US$375 billion (R3 000 billion) for DM care. This figure will rise to between US$234 billion (R1 872 billion) and US$411 billion (R3 288 billion), if nothing is done over the next twenty years (WDF, 2006:online). Those populations in developing countries that cannot afford health care, or who do not have access to health care facilities, are likely to be diagnosed late and suffer DM-related complications. Many people with type 1 DM may even die before they are diagnosed, due to inadequate access to treatment (WDF, 2006:online).

Attempts to reduce both the risk of developing DM, and the cost of managing the established disease, could be advantageous (WHO/FAO, 2002:72). The most cost-effective, affordable and sustainable course of action to cope with the chronic disease epidemic worldwide is the public health approach of primary prevention (WHO/FAO, 2002:5). By preventing disease in large populations, small reductions in blood pressure or blood cholesterol can dramatically reduce health costs. By improving lifestyles, the risk of progression to DM can be reduced by 58% over four years. Up to eighty percent of CHD and up to ninety percent of cases of type 2 DM can be potentially avoided through lifestyle changes (WHO/FAO, 2002:43).

2.8. The influence of diet on health

In general, nutrition is a major modifiable determinant of CDLs (WHO/FAO, 2002:2). Significant changes in diet have occurred in the entire world since the second half of the twentieth century (WHO/FAO, 2002:6). Dietary changes can have both positive and negative effects on health throughout the life-cycle. Culturally varied diets may not only influence present health, growth and development, but may also determine whether an individual will develop certain diseases like cancer, CVD or DM later in life (WHO/FAO, 2002:2,30). A high energy intake in childhood may increase the risk for cancer and other CDLs in later life (WHO/FAO, 2002:35). Dietary influences in the womb have a strong effect on the subsequent manifestation of CDLs, as previously discussed under section 2.2 (Victora et al., 2008:340; WHO/FAO, 2002:30). Genetics, as well as the environment, including the quantity and quality of food ingested, all regulate physical growth (Mamabolo et al., 2005:online).

The WHO's goals for various nutrient intakes are set out in Table 2.23.

Table 2.23. Ranges of population nutrient intake goals (WHO, 2012:online, Table 6).

Dietary factor	Goal (% of total energy, unless otherwise stated)
Total fat	15-30%
Saturated fatty acids	<10%
Poly-unsaturated fatty acids (PUFAs)	6-10%
Omega 6 PUFAs	5-8%
Omega 3 PUFAs	1-2%
Trans fatty acids	<1%
Mono-unsaturated fatty acids (MUFAs)	By difference
Total carbohydrates	55-75%
Free sugars	<10%
Protein	10-15%
Cholesterol	<300 mg per day
Sodium chloride / sodium	<5 g per day / <2 g of sodium per day
Fruits and vegetables	≥400 g per day
Total dietary fibre	From foods
Non-starch polysaccharides	From foods

Excessive energy intake is primarily responsible for the development of obesity (Goedecke et al., 2005:72). Snacking; high eating frequency; binge-eating; and dining out are all factors linked to overweight and obesity (WHO/FAO, 2002:62). High-fat diets promote fat accumulation significantly more

than high-carbohydrate diets, because of their higher energy density. Their high palatability combined with weak satiety effect can also cause higher intake, which leads to poor regulation. With urbanization in South Africa the Westernized diet was adopted, with its higher fat and lower carbohydrates and fibre content (Goedecke et al., 2005:71). However, a high intake of fat alone cannot account alone for weight gain. A lower physical activity level (Kruger et al., 2005:491) and an inability to increase fat oxidation (Goedecke et al., 2005:72) may also contribute to obesity.

2.8.1. Breastfeeding

In South Africa in 1998, about 16% of babies younger than two months were exclusively breastfed, whereas this number decreased to 0,3% by age six to seven months. Seventy percent of babies younger than six months received complementary feeds and 17% were not breastfed at all (DoH et al., 2002:134). The 2003 SADHS found little change in breastfeeding practices overall, but an increased proportion of children younger than four months who were not breastfed (20,1%), and there was a large decrease in the proportion of children who were fed with a bottle (40%) (DoH, 2004:1). The 2003 SADHS also found that supplementation of breast milk starts early, and that exclusive breastfeeding is uncommon in South African mothers. Plain water or other liquids are the most common supplementary feeds given. In 2004, six percent of babies under four months and 27% of babies between four and six months were given mushy or semi-solid foods. By seven to nine months, almost fifty percent of babies received complementary foods (DoH, 2004:19). The 2003 SADHS (DoH, 2004:ii) stated that 12% of South African infants younger than three months were exclusively breastfed (as opposed to 10% in 1998). Only 1,5% of infants aged between four and six months were exclusively breastfed, about the same amount when compared to the 1998 SADHS (DoH, 2004:ii; DoH et al., 2002:267).

There is strong evidence that breastfeeding lowers the risk of obesity, which is directly related to the length of exclusive breastfeeding. Current evidence also indicates that formula milk adversely affects the development of CVD, with increased mortality in older adults who were formula-fed infants (WHO/FAO, 2002:32). Short term breastfeeding or consumption of formula milk is associated with an increased risk for type 1 DM, celiac disease, inflammatory bowel disease and childhood cancers (WHO/FAO, 2002:33). Breastfeeding is further associated with significantly lower blood pressure levels in childhood. Consumption of formula milk instead of breast milk in infancy may increase diastolic and mean arterial blood pressure in later life.

High cholesterol feeding in early life may regulate cholesterol and lipoprotein metabolism later in life. Mean plasma total cholesterol by age four months in breastfed infants were lower than in those receiving formula milk (WHO/FAO, 2002:33). Apparently the fatty acid content, rather than the cholesterol, in the diet regulates cholesterol homeostasis (WHO/FAO, 2002:34).

The WHO/FAO (2002:34) reported that growth rates in infants in Bangladesh, most of whom had chronic intra-uterine undernourishment and were breastfed, were similar to growth rates of breastfed infants in industrialized countries, but catch-up growth was limited and weight at 12 months was relative to weight at birth.

Exclusive breastfeeding protects against overweight or obesity (WHO/FAO, 2002:62). The protective factor of breastfeeding against weight gain has been examined in at least twenty studies. Two of the

largest studies found that the reduction in the risk of developing obesity was substantial (20% to 37%) (WHO/FAO, 2002:66).

Introduction of supplemental foods and breastfeeding practices are important determinants of children's nutritional status, especially for those younger than two years of age. It is unnecessary to supplement breast milk before four months of age, because it contains all the nutrients needed by children in the first four to six months of life (DoH, 2004:18). Supplementation of breast milk is also discouraged because of the risk of contamination, leading to diarrhea (DoH, 2004:19). In exclusively breastfed children younger than five months, the risk for diarrhea-related mortality was 6-fold less and the risk for pneumonia-related mortality was 2,5-fold less (Nannan et al., 2007:738). In babies whose mothers are HIV-negative, the contribution of exclusive breastfeeding to health is substantial. The "older" WHO/UNICEF guidelines stated that HIV-positive women should avoid breastfeeding if it is acceptable, feasible, affordable, sustainable and safe to give formula milk. If formula milk is not viable, exclusive breastfeeding for three months was less risky than mixed feeding (Nannan et al., 2007:737). However, the current 2009 WHO guidelines state that even when anti-retrovirals (ARVs) are unavailable, mothers who still choose to breastfeed should be counselled to exclusively breastfeed in the first six months of life and ***continue breastfeeding thereafter*** "unless environmental and social circumstances are suitable to sustain safe formula feeding (WHO, 2010b:4). Furthermore, the WHO states that HIV positive mothers with infants who are either HIV-negative or who are of unknown HIV status, should exclusively breastfeed for the first six months, where after they should introduce appropriate complementary foods; and should further continue breastfeeding until 12 months of life. After 12 months, breastfeeding should only stop "once a nutritionally adequate and safe diet without breast milk can be provided." Exclusive breastfeeding is therefore still recommended for HIV-positive mothers for the first six months of life unless replacement feeding is acceptable, feasible, affordable, sustainable and safe for them and their infants before that time. When the mother decides to stop breastfeeding at any time, she should stop gradually within one month. If either the mother or infant is receiving ARV prophylaxis, this intake of these ARVs should continue for one week after breastfeeding has been fully stopped. Therefore, abrupt cessation of breastfeeding is not advised (WHO, 2010b:6).

As stated by WCRF/AICR (2007:xxi), sustained, exclusive breastfeeding is protective for the mother as well as the child against cancer, as well as other diseases. Breastfeeding plays a large role in the development of the baby's immature immune system; protects the child against infections in infancy as well as against other childhood diseases. Breastfeeding is especially important where water supply is considered unsafe and among low socio-economic status families where there may not be money to buy infant formula (WCRF/AICR, 2007:xxi).

2.8.2. Fruits and vegetables

Currently only a small amount of the world's population consume the recommended amount of fruits and vegetables daily (i.e. 400 g per capita per day – see Table 2.23) (WCRF/AICR, 2007:12; WHO/FAO, 2002:24).

The low consumption of fruits and vegetables in many developing (i.e. low-income) countries has been confirmed by food consumption surveys (WCRF/AICR, 2007:12; WHO/FAO, 2002:24). Vegetables are

usually more available than fruit (WCRF/AICR, 2007:12). Representative surveys in India found a consumption of only 120 g to 140 g per capita per day (WCRF/AICR, 2007:12; WHO/FAO, 2002:24). In contrast, in 1992 China consumed about 369 g per capita per day (WHO/FAO, 2002:23,56). The WCRF/AICR (2007:12) state that there are high levels of fruit and vegetable intake in Japan, North America and in Australia (300 g/day). The average daily consumption in Europe is about 250 g to 350 g. The consumption is often even much higher in Mediterranean countries, with an average daily consumption of 550 g per day in Spain. Scandinavian countries also have "particularly high" fruit intakes (WCRF/AICR, 2007:12). South Africa is estimated to have an average per capita intake of about 200g daily (similar to intake in the UK). The intake is greater than that of India (120 g to 140 g daily), it is considerably less than that of China (369 g daily) and Spain (600 g daily) (Schneider et al., 2007:718). Even in countries like Italy and Israel, the daily consumption for adults rarely go above 500 g daily day and never above 550 g daily. Greece has the highest mean daily intake of fruit and vegetables (about 700 g to 800 g per person per day) (Schneider et al., 2007:718). National food consumption studies in South Africa show that fruit and vegetable intake was higher in urban areas (168 g per day) than rural areas (137g per day), which can be related to easier access and availability of fruit and vegetables in urban areas (Schneider et al., 2007:721). On the other hand, increasing urbanization decreases the poor urban community's autonomy in their own food production, which may influence the availability of adequate fruits and vegetables (WHO/FAO, 2002:24).

Since the 1960s, countries in Europe, Latin America, North America and South-East Asia have seen an increase in fruit and vegetable availability. However, eastern and central Africa has shown a decrease since the mid-1960s (WCRF/AICR, 2007:12-13). The global average vegetable supply in 2000 was 102 kg per capita per year. The highest supply was in Asia with 116 kg, and the lowest levels were in Africa (52 kg) and South America (48 kg) (WHO/FAO, 2002:24). It should be kept in mind that wild and indigenous vegetables were not taken into account in these production or consumption statistics (WHO/FAO, 2002:24).

The annual per capita fruit intake in South Africa increased from 67,6 kg in 1962 to 80,2 kg by 2001, while vegetable intake remained more or less the same at 120 g per day. Thus, an increase in combined fruit and vegetable intake per capita per day has been seen (from 185 g in 1962 to 220 g by 2001) (Schneider et al., 2007:718). Globally it was estimated that 4,9% of deaths and 1,8% DALYs per year were attributed to low fruit and vegetable intake in 2000. Worldwide low intake of fruit and vegetables was linked to about 31% of IHD; 19% of ischemic stroke; 20% of esophageal cancer; 19% of gastric cancer; 12% of lung cancer and two percent of colorectal cancer (Schneider et al., 2007:717).

The Burden of Disease study found that the mean fruit and vegetable intake was markedly lower for males aged thirty to 44 years than in any of the other age groups of either males or females (Schneider et al., 2007:717). Mean per capita intake over all the age groups was 235 g per day for males and 226 g per day for females, (just under three servings per day). It was estimated that about eighty percent of adults 15 years and older ate less than five fruits and vegetables per day (400 grams per day). This translated to about 11,1 million males and 12,5 million females older than 15 years of age eating too little fruit and vegetables in South Africa in 2000. The largest proportion of total years of life lost in both genders (attributed to low fruit and vegetable intake) accounted for proportionate years of life lost due to IHD in 60,6% of men and 52,2% of women. Ischemic stroke contributed to 17,8% of attributable DALYs in men

and 32,7% in women. Esophageal cancer accounted for 9,8% attributable DALYs in men and 7,0% in women, with lung cancer accounting for 7,8% of all DALYs attributable to low fruit and vegetable consumption in men and 4,7% in women (Schneider et al., 2007:720). Low fruit and vegetable intake ranked 10th to 11th on the list of 17 selected risk factors (as set out in Tables 2.8 and 2.9) in the Comparative Risk Assessment study for South Africa in 2000. South Africa's values were lower than global values with 3,2% of deaths vs. 4,9% of deaths globally, and with 1,1% of DALYs vs. 1,8% global DALYs (Schneider et al., 2007:721).

Increased consumption of fruit and vegetables have been shown to lower the risk of cancers of the mouth; pharynx; larynx; esophagus; lung; kidney; stomach; colon; rectum; ovary; and bladder; especially when consumption include: raw and fresh fruit and vegetables; green leafy vegetables; cruciferous vegetables; lettuce; and carrots (Grant, 2008:965; Eldridge, 2004:1002).

Fruit and vegetables contain various vitamins, minerals, phytochemicals and fibre which may, in different combinations, or even individually, protect against CVD and certain cancers (Schneider et al., 2007:717). The WHO also states that consumption of fruits and vegetables has a significant protective effect for CHD and stroke (WHO/FAO, 2002:86), as well high blood pressure and obesity (Schneider et al., 2007:717), through their phytonutrient, fibre (WHO/FAO, 2002:89) and potassium content (Schneider et al., 2007:717; WHO/FAO, 2002:89). According to the WHO, there is probable evidence for decreased risk for certain cancers (oral cavity, esophagus, stomach, colon and rectum) via increased fruit and vegetable intake through the blocking and suppressing of carcinogen action and prevention of oxidative DNA damage by anti-oxidants, various micronutrients and other substances. There is also probable evidence for decreased risk for type 2 DM with increased fruit and vegetable intake. Daily intake of berries, green leafy- and cruciferous vegetables, and legumes totalling 400 g to 500 g per day is recommended to reduce the risk of CHD, stroke and hypertension (WHO/FAO, 2002:89). The WHO recommends an intake of five portions of eighty grams each fruit/vegetables (totalling 400g) to prevent CVD and certain cancers (Schneider et al., 2007:717).

The risk for atherosclerosis in older women was found to be thirty percent lower in women who ate five to ten servings of fruits and vegetables per day, compared to those who only ate two to five servings per day (WHO/FAO, 2002:40). Dietary plant sterols reduce serum cholesterol by inhibiting cholesterol absorption (WHO/FAO, 2002:84). Fiber from fruit and vegetables also lowers blood cholesterol levels. Eating "plenty" of fruit and vegetables also reduces the risk of fractures, cataracts, age-related macular degeneration, and birth defects, and influences the response to infections, through the function of vitamin A, which maintains the lymphocyte pool and is involved in T-cell mediated response (Schneider et al., 2007;717).

An inverse association exists between potassium intake and the risk of stroke (WHO/FAO, 2002:86). Higher intakes of potassium are associated with lower blood pressure (Couch & Krummel, 2008;872-873; Krummel, 2004b:910). Adequate dietary intake lowers blood pressure and protects against stroke and cardiac arrhythmias. Intake should be at a level where a ratio of 1,0 is kept with sodium intake (about seventy to eighty mmol or 2730 mg to 3120 mg potassium per day) (WHO/FAO, 2002:90). With a sodium intake of around one hundred mmol (2300 mg) less than normal and a potassium intake of up to seventy mmol (2730 mg) daily, a ratio of around 1,0 is achieved, which results in a reduction of 3,4 mmHg in systolic blood pressure (Krummel, 2004b:910; *conversions from Mahan & Escott-Stump, 2004a:1176,

Appendix 4). It has also been suggested that an intake of no more than seventy mmol (or 1,7g) of sodium per day also reduces blood pressure. The recommended levels of intake for fruits and vegetables (Table 2.23) ensure an adequate intake of potassium (WHO/FAO, 2002:86,90). By consuming the recommended amounts of fruits and vegetables recommended by the DASH diet, an intake of around ninety mmol (3510 mg) can be achieved per day (Couch & Krummel, 2008:873; Krummel, 2004b:911).

While potassium supplements have protective effects on blood pressure and CVD, there is no evidence that long-term potassium supplements should be administered to reduce the risk of CVD (WHO/FAO, 2002:86). The equivalent intake of one to two extra servings of fruit, citrus juice, vegetables or potatoes, contributed to a forty percent decrease in the incidence of stroke-related deaths (Krummel, 2004b:911). A higher potassium intake was also linked to a 38% reduction in stroke risk (Couch & Krummel, 2008:873).

Deficiencies in folic acid, vitamin B_{12} and pyridoxine (vitamin B_6) increase levels of homocysteine and free radicals, which result in oxidative damage to endothelial cells, leading to the aggregation of monocytes and platelets which cause vasoconstriction (Schneider et al., 2007:717). High fruit and vegetable consumption can therefore protect against atherosclerosis. A common single gene mutation that reduces the activity of an enzyme that is involved in folate metabolism, is associated with a moderate (20%) increase in serum homocysteine, which raises the risk of both IHD and deep vein thrombosis (DVT) (WHO/FAO, 2002:42). One would assume that persons with this gene mutation should consume even more fruits and vegetables than the general population, to reduce the risk of IHD and DVT.

A diet that protects against cancer has been shown to mainly contain foods of plant origin. It is probable that higher consumption of several plant-based foods probably protects against cancer in various sites. Emphasis should be placed on plant-based foods which are high in nutrients and fiber and low in energy. It is probable that fruit and non-starchy vegetables, including green, leafy vegetables; broccoli; okra; eggplant and bok choy; as well as non-starchy roots and tubers, including carrots; Jerusalem artichokes; celery root, rutabaga and turnips, protect against some cancers, as well as weight gain (because they are typically low in energy-density) (WCRF/AICR, 2007:xviii). Cruciferous vegetables alter the metabolism of carcinogens because they contain glucosinolates, which are transformed into isothiocyanates during food preparation. Indoles and isothiocyanates weaken the effects of polycyclic aromatic hydrocarbons and nitrosamines via the induction of glutathione-S-transferases and the inhibition of cytochrome P450 (WCRF/AICR, 2007:37).

One of the public health goals of the WCRF/AICR (2007:xviii) is that the average consumption non-starchy vegetables and fruits by a population should be at least 600g per day. Different colours of red, green, yellow, white, purple and orange vegetables and fruit, including tomato-based products and allum vegetables (e.g. garlic) should be included daily. Unprocessed cereals and grains and/or legumes should contribute to at least 25 g non-starchy polysaccharides per day. The personal recommendations of the WCRF/AICR (2007:xviii) are to eat at least five portions/servings (equal to at least 400g) of a variety of non-starchy fruit and vegetables daily; to eat relatively unprocessed cereals and grains and to limit refined starchy foods. If starchy roots or tubers are consumed as a staple food, sufficient intake of non-starchy vegetables, fruit and legumes should also be ensured.

2.8.3. Meat and meat alternatives

A remarkable increase in consumption of animal products has been seen in Brazil and China, although levels are still below the levels of North America and most other industrialized countries (WHO/FAO, 2002:21). The per capita supply of vegetable protein is slightly higher in developing countries, while the supply of animal protein is three times higher in industrialized countries (WHO/FAO, 2002:16). Protein, especially animal protein, has a selective effect in promoting height growth. When protein intake is adequate, adult height is expressed as determined by genes (phenotype) (WHO/FAO, 2002:35). Excessive consumption of animal products can, however, lead to excessive intakes of fat (WHO/FAO, 2002:21) and childhood obesity is related to excess protein intake (WHO/FAO, 2002:35).

A strong positive relationship exists between the level of income and the consumption of animal protein. As income increases, so does consumption of meat, milk and eggs, at the expense of staple foods. Urbanization creates opportunities for improved infrastructure. The availability of refrigeration stimulates trade in perishable goods. The urban diet therefore becomes rich in animal proteins and fats, with a high consumption of meat, poultry, milk and other dairy products (WHO/FAO, 2002:20).

2.8.3.1 Red meat

Intake of red or processed meats shows convincing or probable cause of some cancers. High intake of animal fats often equate to relatively high intake of energy, increasing the risk of weight gain (WCRF/AICR, 2007:xix). High intake of red meat has been associated with an increased risk of colon- and prostate cancer (Grant, 2008:964). Preserved meat is associated with an increased risk for colorectal cancer, but fresh meat is not (WHO/FAO, 2002:97). High intake of red meat may result in a higher absorption of haem iron, which may cause greater oxidative stress and increase the risk for potential DNA damage. High red meat intake is also associated with N-nitroso compounds formation, which is associated with the formation of DNA adducts in colon cells and increased levels of nitrogenous residues in the colon. Iron overload can activate oxidative transcription factors and inflammation in the colon (WCRF/AICR, 2007:37).

Bourne et al. (2002:158) states that higher levels of atherosclerotic factors were found amongst 11-year old urban black children in Cape Town, compared to their rural counterparts. It was also found an increase in dietary atherogenicity occurred with increased urban exposure. Intake of protein from meat sources also increased significantly for the urban areas. Therefore, high intake of meat is related to higher atherogenicity. Improvements in socio-economic status do not lead to improved nutritional status, but to a diet pattern associated with atherosclerosis, including high consumption of high-fat and refined foods (Bourne et al., 2002:159).

2.8.3.2. Fish

Fish consumption reduces the risk of CHD. If forty- to sixty grams of fish is consumed by high-risk populations per day, an approximate reduction of fifty percent in deaths from CHD would occur (WHO/FAO, 2002:86). Regular fish consumption of one to two servings a week, protects against CHD and ischemic stroke (WHO/FAO, 2002:90). When survivors of a first MI received advice to consume fatty fish at least twice weekly, two-year mortality was reduced by 29% (WHO/FAO, 2002:86).

Servings should provide an equivalent of 200 mg to 500 mg of eicosapentanoic acid (EPA) and docosahexanoic acid (DHA) (WHO/FAO, 2002:90).

Currently, about 13,8% to 16,5% of animal protein intake in the world is derived from fish, crustaceans and molluscs. In the early 1960's per capita fish supply in low-income countries with a food deficit was thirty percent of that of the richest countries. This gap has gradually narrowed until in 1997, the supply was seventy percent of that of the richest countries. Thus, despite the low consumption of fish in low-income food-deficit countries, the contribution of fish to total animal protein intake is still considerable (WHO/FAO, 2002:22). The recommendation for increased fish consumption should be balanced against the sustainability of marine stocks and the potential for depletion (WHO/FAO, 2002:23).

2.8.3.3 Soy

Soy is rich in isoflavones, the intake of which may provide protection against CHD (WHO/FAO, 2002:87). Soy lowers total cholesterol, LDL and triglycerides (Krummel, 2004a:882). However, to decrease LDL by a few percent would require a soy intake of at least half of a person's daily protein intake (Krummel, 2008:858). An average consumption of 47g of soy per day led to a nine percent decline in total cholesterol and a 13% decline in LDL cholesterol in subjects free of CHD (WHO/FAO, 2002:87). Krummel (2004a:882) states that an intake of 25 grams of soy per day lowers LDL cholesterol by four- to eight percent. Plant sterols and stanols made from soybean oil (and pine tree oil), now found in margarines, lower blood cholesterol by nine- to twenty percent, if two to three grams are consumed per day (Krummel, 2008:858; Krummel, 2004a:882). A daily serving size of about one to two ounces (28 g to 56 g) of soy is recommended (Krummel, 2008:858).

If soy is consumed before reaching adulthood, it may be protective against breast cancer. However, commercially prepared soy supplements should be limited in women with estrogen receptor positive-type breast cancer (Grant, 2008:964).

2.8.4. Fibre

Soluble fibres lower serum cholesterol and LDL (Krummel, 2008:858; Krummel 2004b:881). Soluble fibres include: pectins; gums; mucilages; and some hemi-celluloses (Krummel, 2008:858; Krummel, 2004a:881; WHO/FAO, 2002:84). Insoluble fibres, like cellulose and other hemicelluloses, have no effect on serum cholesterol levels (Krummel, 2008:858; Krummel, 2004a:881; WHO/FAO, 2002:84). The total recommended intake of fibre (25 g to 30 g per day) should contain about six to ten grams of soluble fibre, which is easily achieved by consuming five or more servings of fruit and vegetables per day and six or more servings of grains per day (Krummel, 2008:858; Krummel, 2004a:881). The WHO/FAO (2002:77) recommends a minimum daily intake of 20g fibre from wholegrain cereals, legumes, fruit and vegetables to reduce the risk of type 2 DM. The recommended intake of fruits and vegetables of $\geq$400g per day and the consumption of wholegrain foods is likely to provide >25 g of total dietary fibre per day (WHO/FAO, 2002:58).

The infrequency of DM in rural Africa may result from the consumption of substantial amounts of fibre in the diet, from minimally-processed or unprocessed carbohydrates. High intakes of fibre have been shown to reduce blood glucose and insulin levels in people with type 2 DM and impaired glucose tolerance

(WHO/FAO, 2002:75). High intake of dietary fibre also promotes weight loss (WHO/FAO, 2002:63), lowers blood pressure and protects against CHD. Most fibres reduce plasma total and LDL cholesterol (WHO/FAO, 2002:84).

A fibre-rich diet is associated with a protective effect in colon cancer, but genetics should also be considered (Grant, 2008:964; Eldridge, 2004:1001). When consuming 4,5 servings or more of whole grains daily, a 35% lower risk of colon cancer has been reported, when compared to eating less than 1,5 servings of whole grains daily (Grant, 2008:964).

2.8.5. Sugar

The WHO/FAO (2002:57) recognizes that their recommendation for the intake of free sugars to be less than ten percent of total energy, is controversial. However, they maintain their opinion that high intakes of free sugars threaten the nutrient quality of the diet by providing significant energy without specific nutrients. They maintain their opinion that restricting the amount of free sugars in the diet, reduces the risk of weight gain (WHO/FAO, 2002:57). Processed foods contain substantial amounts of fat or sugar and tend to be more energy-dense than fresh foods (WCRF/AICR, 2007:xviii). It is speculated that the global increase in obesity may be related to the increased consumption of energy-dense foods and sugary drinks worldwide (Van den Berg, 2011:259; WCRF/AICR, 2007:xviii). However, it should be kept in mind that eating any food or beverage in excessive amounts, thereby exceeding energy intake above recommended daily requirements, will cause weight gain (Van den Berg, 2011:258).

Sugar-rich drinks increase overall energy intake by reducing appetite control, which leads to an increased body weight, as compared with consumption of artificially sweetened drinks (WHO/FAO, 2002:57). Sugary drinks are postulated to promote weight gain because they provide energy without inducing satiety or reducing subsequent energy intake, which then promotes overconsumption of energy (WCRF/AICR, 2007:xviii). Children with a high consumption of soft drinks rich in free sugars are more likely to be overweight and to gain weight (WHO/FAO, 2002:57). Every extra can or glass of sugar-sweetened drink taken by children per day, increases their obesity risk by sixty percent. Many fruit drinks and cordials are equally energy-dense and may promote weight gain if drunk in large quantities (WHO/FAO, 2002:66), especially since sugar-sweetened drinks may contain from 170 kJ to 200kJ per 100 ml (Van den Berg, 2011:259).

When adults with metabolic syndrome replaced high glycemic index (GI) carbohydrates with low GI carbohydrates, a beneficial change in weight and metabolic indices was observed (WHO/FAO, 2002:57). Hyperinsulinemia and insulin resistance, which form part of the metabolic syndrome, are associated with increased triglyceride and LDL levels (Van den Berg, 2011:259). Drinking one to two sugar-sweetened drinks per day increases the risk for metabolic syndrome, insulin resistance, type 2 DM, CVD, and hypertension (Van den Berg, 2011:257). Grant (2008:965) states that blood glucose and triglyceride levels can increase when there is high consumption of simple sugars on a regular basis. This can raise the levels of insulin and other hormones that may stimulate cancer cell growth (Grant, 2008:965).

According to Augustin et al. (2001:1533) high GI or GL carbohydrates (such as refined carbohydrates) increase glucose and insulin levels more than legumes or whole grains. Raised insulin levels may raise insulin-like growth factor levels, which may increase breast cancer risk. There have been various

hypotheses that high insulin levels, dietary GI and GL have been directly associated with increased risk of several CDLs, including colorectal cancer, type 2 DM, CHD and obesity. The study by Augustin et al. (2001:1533) supported the hypothesis that there is an association between moderate, direct GI or GL and the risk of breast cancer, as well as an association between hyperinsulinemia or insulin resistance and breast cancer. However, a less clear association between sugar (categorized as a medium-high GI food) and breast cancer was found (Augustin et al., 2001:1535). Potischman et al. (2002:937) found that there was an increased risk for breast cancer in their study group who consumed high amounts of sweet items, particularly soda drinks and desserts. Those participants who consumed sweet foods more than 2,8 times per week had an increased risk of breast cancer. After adjusting for energy intake were made, the association was even greater. Grant (2008:965) states that the consumption of high GI foods may be associated with an increased risk of ovary-; endometrium-; breast-; colorectal-; pancreas-; and lung cancer. Sugar has an intermediate GI and, when consumed together with a low-GI meal, does not exaggerate insulin response (Van den Berg, 2011:260).

When sugar is consumed in moderate quantities, there is no detrimental effect on dental health, weight management, or CDLs. However, excessive consumption of sugar-sweetened beverages is associated with weight gain and obesity (Van den Berg, 2011:257). It is recommended to limit intake of refined and processed sugars and to emphasize whole grains or complex carbohydrates as part of a balanced diet (Grant, 2008:965). Excessive intake of added sugars should be avoided, and especially products sweetened with fructose (Van den Berg, 2011:257). Frequent brushing with fluoride toothpaste and good dental care prevents dental caries more effectively than avoiding sugary foods (Van den Berg, 2011:258).

2.8.6. Fat

According to the WHO/FAO (2002:56), a total fat intake of twenty percent of total energy is consistent with good health. However, highly active groups with diets rich in vegetables, legumes, fruits and wholegrain cereals may have a total fat intake of up to 35% without the risk of weight gain.

Energy from fat is no more fattening than the same amount of energy from carbohydrates or proteins, but diets that are high in fat tend to be energy-dense. By comparing the intake of high-fat diets versus low-fat diets over a two month-period, a reduction in fat intake by ten percent corresponded to about a 1000 kJ reduction in energy intake and a loss of three kilograms of body weight. This equates to about one BMI unit or about a five percent difference in obesity prevalence (WHO/FAO, 2002:64).

Populations that consume high amounts of saturated fatty acids (SFAs), tend to have increased blood cholesterol levels, which increases CHD incidence and mortality (Krummel, 2008:837). A high saturated fat intake impairs glucose tolerance and is associated with higher fasting glucose and insulin levels. Higher SFA levels in serum and muscle are associated with higher fasting insulin levels, lower insulin sensitivity and a higher risk for developing type 2 DM (WHO/FAO, 2002:74). High intake of saturated- and animal fat may relate to an increased risk of breast-, colon-, lung- and prostate cancer (Eldridge, 2004:1001). Myristic acid is the most hypercholesterolemic, followed by palmitic and lauric acid (Krummel, 2008:855). Myristic and palmitic acids are abundant in diets rich in dairy products and meat (WHO/FAO, 2002:82). Palm oil is becoming an important edible oil in diets of much of South East Asia (WHO/FAO, 2002:20). To reduce the risk of DM and CVD, saturated fat intakes should not exceed ten

percent of total energy (WHO/FAO, 2002:77) as recommended in Table 2.23; and <7% of total energy in high risk groups (WHO/FAO, 2002:88). SFAs raise total- and LDL cholesterol. The most effective replacement for SFAs, in terms of CHD outcome, is poly-unsaturated fatty acids (PUFAs), especially linoleic acid (WHO/FAO, 2002:82).

Trans fatty acids elevate LDL and decrease HDL, thus contributing to the development of CHD. To a large degree, trans fatty acids have been reduced or eliminated from retail fats and spreads, but deep fried foods and baked goods are still major sources (WHO/FAO, 2002:82). According to Krummel (2004a:880), fifty percent of fat intakes come from SFA sources, such as beef, butter and milk. The trans-isomer of oleic acid (elaidic acid) raises blood cholesterol as compared with PUFAs, but less than myristic or lauric acid.

Both mono-unsaturated fatty acids (MUFAs) and omega 6-PUFAs lower plasma total -and LDL cholesterol, when substituted for SFAs. PUFAs are more effective than MUFAs (WHO/FAO, 2002:83). Good sources of omega 6-PUFAs are vegetable oils, salad dressings and margarine containing linoleic acid (Krummel, 2004a:879). Linoleic acid is abundant in soybean and sunflower oil (WHO/FAO, 2002:83). If carbohydrates in the diet are replaced by linoleic acid (omega 6-PUFA), LDL is lowered and HDL is raised. If SFAs are replaced with PUFAs, both LDL and HDL are lowered (Krummel, 2004a:879).

Omega 3-PUFAs have the potential to increase LDL and lower triglycerides (Krummel, 2004a:879; WHO/FAO, 2002:83). They are also precursors of prostaglandins that interfere with blood clotting (Krummel, 2008:857; Krummel, 2004a:879; WHO/FAO, 2002:83). Thus, overconsumption of omega 3-PUFAs (either via intake of fish or supplements) can prolong bleeding times, as shown by the Eskimo population who have a high omega-3 dietary intake and low CHD incidence (Krummel, 2008:857; Krummel, 2004a:879). The most important omega 3-PUFAs, are EPA and DHA (found in fatty fish, fish oil and –capsules), and α-linolenic acid (found in plants) (Krummel, 2004a:879; WHO/FAO, 2002:83). For persons with CVD, a daily combined intake of one gram of EPA and DHA, preferably from fish, is recommended. For persons with hypertriglyceridemia, an intake of two to four grams of EPA and DHA is required for a lowering effect (Krummel, 2008:867). Omega 3 fatty acids suppress or decrease the mRNA of interleukin, which is elevated in atherosclerosis, arthritis and other auto-immune diseases (WHO/FAO, 2002:42).

Replacing SFAs with MUFAs lowers serum cholesterol levels, LDL and triglycerides to about the same amount as with PUFAs (Krummel, 2008:855). The only important source of MUFAs is oleic acid, abundant in olive oil (Krummel, 2004a:880; WHO/FAO, 2002:83); as well as canola oil and nuts (WHO/FAO, 2002:83). Olive oil is an important edible oil consumed largely in the Mediterranean region (WHO/FAO, 2002:20).

A proportionate higher intake of unsaturated fatty acids is associated with a reduced risk of type 2 DM. Higher proportions of long-chain PUFAs in muscle are associated with increased insulin sensitivity (WHO/FAO, 2002:74). However, when total fat intake is high (>37% of total energy), altering the quality of the fat has little effect on decreasing glucose intolerance (WHO/FAO, 2002:75).

Dairy fat and meat are major sources of cholesterol. Egg yolk is also rich in cholesterol, but unlike dairy and meat, it does not contain saturated fats. Evidence for an association between dietary cholesterol and

CVD is contradictory (WHO/FAO, 2002:83), since specific genotypes in humans raise cholesterol levels more than others (WHO/FAO, 2002:42). The WHO/FAO (2002:83) is of the opinion that if the intake of dairy fat and meat is controlled, there is no need to severely restrict egg yolk intake.

The incidence of hypertension is lower in vegans, because they tend to consume more PUFAs and less total fat, saturated fat and cholesterol. The prostaglandins produced by PUFAs affect renal sodium excretion and relax vascular muscles, which can have an effect on blood pressure. An olive oil-enriched diet can decrease the use of anti-hypertensive drugs by 48% (Couch & Krummel, 2008:873; Krummel, 2004b:911).

2.8.7. Nuts

Nuts contain a high amount of arginine, which is an amino acids of which nitric oxide, a vasodilator, is synthesized. This may explain the positive association between nut intake and a reduced prevalence of CVD (Krummel, 2004a:865).

Nuts are high in unsaturated fatty acids and low in saturated fats. Frequent consumption of nuts decreases the risk of CHD. Because of the high energy content of nuts, dietary intake should be moderate to achieve the desired energy balance (WHO/FAO, 2002:87).

2.8.8. Alcohol

In 1998, 45% of South African men and 17% of women consumed alcohol at the time of the SADHS (Schneider et al., 2007:665; DoH et al., 2002:238). Schreider et al. (2007:664) indicates that the figure approximated more to fifty percent of men and twenty percent of women, and that this might even be underestimated. The 1998 SADHS found the rate was 28% for the general South African population in 1998, which at that time translated to 8,3 million South Africans. In males, the highest drinking levels (50% or more) were reported in the Free State and Gauteng provinces. The lowest levels (28%) were in the Northern Province. For females, the highest levels (23% to 24%) were in the Free State, Western Cape and Northern Cape, and the lowest levels (9%) were in the Northern Province (DoH et al., 2002:238). The observed rates of fetal-alcohol syndrome in the Western Cape's poorer communities were between 18 to 141 times greater than those found in the USA (Schneider et al., 2007a:665). In a study conducted in the Western Cape, about a quarter (23,7%) of pregnant women reported alcohol intake sufficient to place their unborn child at risk (Schneider et al., 2007a:666).

Alcohol forms an integral part of the South African economy. The wine and brewing industries contributed to South Africa becoming an important player in the global alcohol market. Alcohol taxes has raised R4,2 billion in 2002/3, but the costs of alcohol abuse were estimated at around R9 billion, amounting to about one percent of the GNP (Schneider et al., 2007a:664).

In 2000, adult per capita alcohol consumption was estimated around 10,2 litres of pure alcohol annually, but if adjusted for unrecorded consumption (especially home brews), the estimated amount increases to 12,4 litres. Although these figures are low compared to most developed countries, because a large part of the South African population does not drink, this means that the amount consumed per person should be nearer to about twenty litres of absolute alcohol per year (Schneider et al., 2007a:665). The Comparative

Risk Assessment study found that alcohol harm ranked 3rd when looking at percentages of total DALYs relating to the 17 risk factors included in the study as listed earlier in Tables 2.8 and 2.9 (Schneider et al., 2007a:670).

Alcohol is a significant contributor to the burden of disease, since it is a leading risk factor for mortality and morbidity (Pisa et al., 2010:S4). High alcohol consumption is a risk factor for CDLs, including: stroke; DM; esophageal cancer; as well as breast- and liver cancer (Steyn et al., 2006:18). The alcohol-related health outcomes are listed in Table 2.24. Chronic alcohol abuse can result in liver cirrhosis. Misuse during pregnancy can result in fetal brain damage, leading to long-term developmental- and social consequences, as well as fetal-alcohol syndrome.

Alcohol consumption also increases the risk of high blood pressure (Pisa et al., 2010: S6-S7; Schneider et al., 2007a:665). In older children and adolescents, habitual alcohol use can contribute to high blood pressure and increased risk for CVD later in life (WHO/FAO, 2002:41). In the Prospective Urban and Rural Epidemiology (PURE) study, it was found that alcohol increased blood pressure even after a low consumption (Pisa et al., 2010a:S36).

Table 2.24. Alcohol-related health outcomes (Schneider et al., 2007a:666, Table I)

Health outcomes
Cancers (neoplasms)
Mouth/oropharynx
Esophagus
Liver
Larynx
Breast
Cardiovascular diseases
Hypertensive disease
Ischemic heart disease
Ischemic stroke (cerebral infarction)
Hemorrhagic stroke (intra-cerebral hemorrhage)
Other chronic diseases
Diabetes (non-insulin dependent)
Cirrhosis of liver
Effects of prenatal alcohol exposure
Fetal alcohol syndrome
Low birth weight
Neuropsychiatric conditions
Depression (unipolar major depression)
Epilepsy
Alcohol dependence
Acute adverse effects
Intentional injuries
Unintentional injuries

Alcohol is also associated with increased aggressive behaviour, which can lead to crime, disrupted family life, domestic violence, child neglect, and unsafe sexual practices, increasing the risk of HIV (Schneider et al., 2007a:665). Augustin et al., (2001:1536) states that alcohol consumption increases breast cancer risk, but decreases the risk of diabetes. However, they found that alcohol does not significantly modify the effect of the GL of food, thereby not affecting insulin levels as much as expected.

Alcohol is classified as a Group 1 carcinogen by the WHO. Studies indicate that alcohol is associated with increased risk of cancer in the mouth; larynx; pharynx; esophagus; lung; colon; rectum; liver and breast

(Grant, 2008:966). The risk is 35 times greater for mouth, tracheal or esophageal cancer if individuals both smoke and drink alcohol (Pisa et al., 2010:S8).

In the 1998 SADHS, it was found that alcohol use was higher among persons with hypertension (DoH et al., 2002:265). Seftel (1977:122) already found in 1977 that alcoholism in the black population of South Africa could lead to congestive cardio-myopathy or esophageal cancer. Home-brewed alcoholic drinks, made in iron pots, may contain high amounts of iron, which can lead to hepatic cirrhosis, sometimes accompanied by DM.

According to the Burden of Disease study, alcohol harm accounted for about 7,1% of all deaths and 7.0% of total DALYs in 2000. CVD incidents ranked 2nd in alcohol-related deaths, after injuries (Schneider et al., 2007a:664). The ratio of alcohol-related deaths per gender was one female for every four males, mostly because of the high amount of fatal injuries among young adult men. The benefits of alcohol use could be seen among the amounts of preventable deaths from stroke and DM, especially in older men and women, and were particularly noticeable for stroke in older women. Apart from the beneficial effects, there was more than 1.1. million alcohol-related DALYs in 2000 (Schneider et al., 2007a:668). Alcohol-attributed DALYs as found by cause are depicted in Figure 2.5 below.

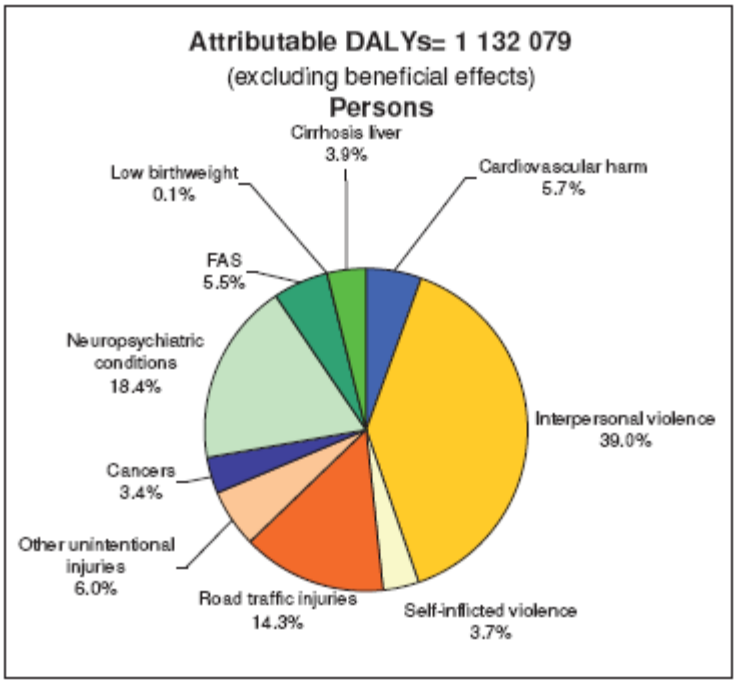


Figure 2.5. Alcohol-related DALYs by cause (Schneider et al., 2007a:668, Figure 2).

Globally, alcohol contributed to 3,2% of deaths and 4,0% of DALYs. When looking at the burden for developing countries, the DALY burden was around 1,6% as compared to the 9,2% burden in developed regions. South Africa's burden is more similar to the developed countries' figures. The WHO's global Comparative Risk Assessment study found that 28% of the unintentional injury burden and 12% of the intentional injury burden was related to alcohol, whereas in South Africa the unintentional injury burden was 20,2% and intentional injury burden 40,9% (Schneider et al., 2007a:670).

Regular light to moderate drinking among older persons reduces blood clotting and plaque deposit in arteries, thereby reducing the risk of heart attacks, IHD and cerebrovascular disease/stroke (Schneider et al., 2007a; 665). Moderate levels of alcohol consumption are associated with decreased risk of MI and

CHD mortality in white men. Wine contains resveratrol, an antifungal compound found in grape skins, which increases HDL cholesterol and inhibits LDL oxidation. The French paradox describes high-fat diet and lower rates of CVD, because of the French population's consumption of red wine (Krummel, 2004a:881).

Alcohol has both beneficial and harmful effects on health, but on the whole the impact of alcohol consumption on health is more negative than positive (Schneider et al., 2007a:664). The WHO states that general recommendations do not favour the use of alcohol, because of its other health risks (WHO/FAO, 2002:87).

Pisa et al. (2010a:S36) suggests that there is not enough scientific evidence to promote any alcohol consumption among persons who do not drink. The WCRF/AICR (2007:xix) state that the evidence on cancer justifies the avoidance of alcohol altogether because no clear level of consumption is shown below which there is no increase in cancer risk. However, other evidence shows that modest intake of alcohol may likely reduce the risk of CHD, and that all alcoholic drinks have the same effect. The WCRF/AICR panel did emphasize that children and pregnant women should not consume alcoholic drinks at all.

Alcohol intake should be less than two drinks per day in men and one drink per day in women and lighter-weight men, to prevent high blood pressure (Couch & Krummel, 2008:872; Krummel, 2004b:910) and cancer (Grant, 2008:966).

2.8.9. Salt

Sodium intake is directly associated with blood pressure (WHO/FAO, 2002:85). In 1998, South African young people consumed more salty foods than older people, suggesting that hypertension prevalence may increase as this generation ages (DoH et al., 2002:265).

According to the WHO (2008a:65), 8,5 million lives could be saved in a decade in the 23 developing countries that comprise eighty percent of the global chronic disease burden, if manufacturers reduce dietary salt intake by 15% through manufacturing practices; and if a mass-media campaign to encourage dietary change is sustained.

In societies where 9g to 12g of salt (150 mmol to 200 mmol sodium) is consumed per day, hypertension and stroke is more prevalent (Krummel, 2004b:907). A reduction of sodium intake by 50 mmol per day may lead to a fifty percent reduction in the number of people requiring anti-hypertensive therapy; as well as a 22% reduction in deaths resulting from strokes; and a 16% reduction in deaths resulting from CHD. A difference in sodium intake of one hundred mmol per day is associated with an average difference in systolic blood pressure of 5 mmHg in 15 to 19 year olds and 10 mmHg in sixty to 69 year olds (WHO/FAO, 2002:85). An intake of no more than seventy mmol sodium per day is beneficial in reducing blood pressure (WHO/FAO, 2002:90). A two grams salt-restricted diet is equal to about 34 mmol sodium (Krummel, 2004b:910). Total sodium intake should take into account all sources, including monosodium-glutamate and preservatives (WHO/FAO, 2002:90).

The WCRF/AICR (2007:xx) state that salt and salt-preserved foods probably increase the risk of developing stomach cancer. Although salt is necessary in the human body, much lower levels are necessary than those typically consumed in most parts of the world.

2.8.10 Coffee

High coffee intake has been associated with CHD. Coffee beans contain a terpenoid lipid called cafestol, which raises total and LDL cholesterol, if consumed in large amounts (WHO/FAO, 2002:87). Heavy consumption of regular coffee (approximately equal to 720 ml daily), minimally increases total cholesterol, LDL and HDL (Krummel, 2004a:881). Boiled, unfiltered coffee contains high amounts of cafestol (WHO/FAO, 2002:87). According to Krummel (2004a:881), boiled coffee causes a greater elevation in plasma lipids than filtered coffee. Thus, drinking filtered coffee instead of boiled coffee, contributes to a significant decline in serum cholesterol (WHO/FAO, 2002:87).

Nel and Steyn (2002:53-58, Table 22) found that coffee was consumed by 7,2% of children between one to five years old, 9,4% of children aged six to nine years, and between 16,2% and 34% of South Africans who were ten years and older. In comparison, tea was consumed by 56,8% of children age one to five years; 60,6% of children aged six to nine years; and between 61,9% and 68,2% of South Africans who were ten years and older.

Chapter 3 - Methodology

3.1. Introduction

The purpose of the AHA FS study was to determine how urban and rural lifestyles of populations predispose them to the double burden of disease, i.e. chronic diseases of lifestyle related to obesity (such as DM and CVD) as well as to undernutrition and to investigate factors that may play a role in these health challenges (such as diet).

The rural leg of the AHA study was undertaken during a two week period in March 2007, in Trompsburg, Springfontein and Philippolis (as part of the Free State Rural Development Partnership Programme (FSRDPP)). The urban leg of the AHA study was undertaken during a two week period between 2 and 13 March 2009, at the Mangaung University Community Partnership Programme (MUCPP) clinic.

3.2. Ethical considerations

Approval for the study was obtained from the Ethics Committee of the Faculty of Health Sciences at the University of the Free State (ETOVS NR 21/07A), Free State Department of Health, local municipalities and community leaders.

Informed consent was signed by each participant (Appendix A). In the case of the children, a parent or guardian gave consent. The study was explained to the participant before he/she was required to sign the consent form. Each participant received a copy of the consent form, together with an information document (Appendix B). Illiterate persons could make a cross in the presence of a witness on the consent form. Consent forms were available in English, Afrikaans and Sotho. All information was kept confidential and participants' privacy was respected.

The community leaders were contacted in each of the specific communities via community health workers. The reason for the study was explained as well as the type of data that would be collected, and the method in which data would be collected. Benefits to the community were also explained. Community health workers visited selected households, explained the study and obtained informed consent. If community members refused to participate, they were not discriminated against. Participants could also withdraw from the study at any time. In addition to the information document, community members received a participation letter (Appendix C), which the participant could present to his/her employer to enable him/her to be granted leave from work to participate in the study on a specific day.

3.3. Study design

The baseline phase of the AHA FS study comprised a descriptive cross-sectional design.

3.4. Sample selection

The AHA FS study included both a rural leg, undertaken during 2007 and an urban leg, undertaken during 2009. The current study formed part of both the rural and the urban baseline surveys of the AHA FS study.

3.4.1. Population

The MRC Technical Report states that the black population is the only ethnic group still undergoing a nutrition transition from a traditional to a Western diet, since the other population groups (white, coloured and Indian) mostly already follow the westernized dietary patterns (Steyn et al., 2006:5). For this reason, it was decided to include areas where mostly black population groups resided in an urban and rural area of the Free State. In rural areas, black and coloured communities often resided in the same area.

3.4.2. Sample

For the rural part of the study in 2007, three rural communities in the Southern Free State (part of the Free State Rural Development Partnership Programme) were selected, namely Trompsburg, Philippolis and Springfontein. All households in the black and coloured communities in these three towns were included in the study.

For the urban part of the study in 2009, six urban communities (serviced by MUCPP) in Mangaung were selected, namely an area of Rocklands indicated as “Buffer” on the municipal map, as well as the communities of Freedom Square, Kagisanong, Chris Hani, Namibia and Turflaagte.

The number of plots in the MUCPP service areas was counted on a municipal map, and an estimate was made of additional squatter households in open areas. A stratified proportional cluster sample was selected, stratified by area and formal plot/squatter households in open areas. Using randomly selected X and Y coordinates, hundred starting points were selected in this way. From each starting point five adjacent households were approached. In the Rocklands buffer community, three formal households from a total of 345 were included. In Freedom Square, forty formal households from a total of 4791 and three squatter households from a total of 300 were included. In Kagisanong, six formal households from a total of 651 formal (and fifty squatter) households were included. In Chris Hani, nine formal households from a total of 1126 and one squatter household from a total of seventy were included. In Namibia, 16 formal households from a total of 2011 and one squatter household from a total of sixty were included. In Turflaagte, 18 formal households from a total of 2216 and three squatter households from a total of 300 were included.

3.4.2.1. Inclusion criteria

Every household in both the rural and urban communities, which met the following inclusion criteria, was eligible to participate:

- If informed consent was given;
- Adults between the ages of 25 and 64 years; and
- Preschool children (0-7 years old).

3.4.2.2. Exclusion criteria

People with mental disabilities were excluded from the study.

3.5. Operational definitions

For the purpose of the bigger AHA FS study, the following information was gathered:

- Blood variables
- Anthropometric measurements (for adults and children separately)
- Medical examinations
- Questionnaires related to the following:
    - Reported health;
    - Knowledge, attitude and practices related to nutrition and eating behaviour;
    - Socio-demographic status;
    - Household food security;
    - Dietary intake; and
    - Physical activity.

For the purpose of this particular part of the study, information related to dietary intake and anthropometry was included.

3.5.1. Dietary intake

A 24-hour recall of reported usual intake (Appendix D) and adjusted food frequency questionnaire (Appendix E) were used to determine dietary intake during individual interviews with each participant. For the purpose of this study, dietary intake referred to the total intake of foods, energy and macronutrients. A 24-hour recall of reported usual intake (Appendix D) and an adjusted food frequency questionnaire (FFQ) reflecting common foods usually consumed by the target population (Appendix E) were completed for each participating adult and preschool child (Appendix F) from every household.

The exchange lists, based on the American Dietetics Association (ADA) Food Guide Pyramid (USDA), 1992:online), classify food into seven groups according to their energy, carbohydrate, fat, and protein content, and these were used to quantify the energy and macronutrient content of the dietary intake of participants. Cut off points were followed as set out in Table 3.1 below. Food intake less than the recommendations of the Food Guide Pyramid (USDA, 1992:online) were regarded as inadequate or below requirements; intake within the guidelines, as adequate or within requirements; and intake higher than the guidelines, as high or above requirements.

Table 3.1. Cut off points for analysis of food intake in portions, according to the recommendations of the ADA Food Guide Pyramid (USDA, 1992:online; Center for Nutrition Policy and Promotion (CNPP), 1999:online)

Food groups	***Adequate adult portions***	***Adequate child portions***
Milk	2 – 3	2
Meat and meat alternatives	2 – 3	2
Vegetables	3 – 5	3
Fruit	2 – 4	2
Bread, cereal and starches	6 – 11	6
Fat	$\leq$ 4	$\leq$ 4
Sugar	$\leq$ 4	$\leq$ 4

3.5.1.1 Adults

The 24-hour recall of reported usual intake (Appendix D) and adjusted FFQ forms (Appendix E) were completed during individual interviews with each participant. Foods regularly consumed, but not necessarily during the previous 24-hours, were noted. Calculations of portions consumed from each food group in adults were indicated in the food exchange calculations for adults. Portion sizes for adults were used as set out in Table 3.2.

Table 3.2. Portion sizes for adults (USDA, 1992:online)

Grain group	**Fruit group**	**Meat group**
1 slice of bread	1 piece of fruit or melon wedge	30g of cooked lean meat, poultry, or fish
½ cup of cooked rice or pasta	½ cup of juice	1 egg = 30 g
½ cup of cooked porridge	½ cup of canned fruit	½ cup of cooked dry beans = 30 g
1 cup of ready-to-eat cereal	½ cup of dried fruit	2 tablespoons of peanut butter (add one fat exchange) = 30 g
Vegetable group	**Milk group**	**Fats and sweets**
½ cup of chopped raw or cooked vegetables	1cup of milk or ½ yogurt	2 teaspoons sugar
1 cup of raw leafy vegetables	45g of cheese	2 hardboiled sweets
		5ml oil or margarine

Intake of one fruit or vegetable exchange rich in vitamin A or β-carotene per day, one fruit or vegetable exchange rich in vitamin C per day, and at least one other fruit or vegetable exchange per day was considered as adequate intake. Less than these amounts was considered inadequate or below requirements. When considering the 5-a-day challenge, i.e. an intake of a total of five fruit and vegetables a day, participants were divided into two groups, i.e. those who consumed less than a total of five fruit and vegetables a day, and those who consumed five or more per day.

Potatoes and other starchy vegetables, such as corn and sweet potato, were counted as a starch/grain exchange and not a vegetable exchange, as consistent with WHO international recommendations (WHO/FAO, 2002:58).

A separate exchange list was designed for legumes as listed in Table 3.3.

Table 3.3. Exchange list for legumes and soy beans.

ONE LEGUME PORTION PROVIDES:	
21 grams of carbohydrates, 7 grams of protein, 0,7 grams of fat, and 500 kJ.	
Split peas (Cooked)……..	½ cup (85g)
Chick peas (Dried & cooked)……	½ cup (85g)
Lentils (Whole; cooked)……..	½ cup (90g)
Lentils (Split; cooked)……	½ cup (90g)
Sugar beans (Fried & cooked)…….	½ cup (100g)
Kidney beans (White; Dried & cooked)……..	½ cup (90g)
Canned:	
Baked beans in tomato sauce……..	⅓ cup (90g)
Kidney beans (White; solids & liquids)……	⅓ cup (90g)

Soy beans	½ cup (80g)
Provides: 8 grams of carbohydrates, 13 grams of protein, 7 grams of fat, and 630 kJ.	

According to the Food Guide Pyramid (USDA, 1992:online), legumes form part of the meat and meat alternatives group. However, legumes contain more carbohydrates than a meat alternative portion;

therefore the carbohydrate intake is underestimated if it is included in the meat group. Soy beans are an exception as they contain much less carbohydrates and more protein. Soy beans were thus included in the meat and meat alternative group. Intake of legumes once a week was classified as adequate intake/ within recommendations.

Energy and macronutrient content of alcoholic beverages were calculated using the guidelines set out in Table 3.4. The content of home-brewed beer was set equal to regular beer.

Table 3.4. Energy and macronutrient content of select alcoholic beverages (Mahan & Escott-Stump, 2008a:1276, Appendix 38; Mahan & Escott-Stump, 2004b:1241, Appendix 44).

Beverage	Serving (ml)	Energy (kJ)	Carbohydrates (g)	Fat (g)
Beer, regular	360	630	13	10
Beer, light	360	420	5	10
Cider	360	252	12	-
Wine, dry	120	336	-	10
Wine, red or rosé	120	357	2	10
Wine, sweet	120	441	5	10
Wine, sparkling	120	420	6,5	10
Whiskey, brandy, etc.	45	420	-	10

After the 24-hour recall of reported usual intake was analyzed according to all items consumed and the items were described in terms of exchanges; the total exchanges were transferred to the calculation table as shown in Table 3.5, to determine total macronutrient intakes. The adjusted FFQ (Appendix E) was used to cross-check the usual dietary intake and also to determine the twenty most frequently consumed foods by comparing it to each participant's 24-hour recall of reported usual intake. Intake of each food group was also coded by this Masters student as below, within or above requirements. Food intake less than the recommendations of the Food Guide Pyramid (USDA, 1992:online) were regarded as inadequate or below requirements; intake within the guidelines, as adequate or within requirements; and intake higher than the guidelines, as high or above requirements.

Table 3.5. Calculation of dietary intake using recommended daily servings

	Quantity	Energy	Protein	CHO	Fat	Below requirement 1	Within requirement 2	Above requirement 3
Milk and milk products		530	8	12	5			
Meat and meat alternatives		315	7		5			
Legumes		500	7	21	1			
Soy beans		630	13	8	7			
Fruit β-carotene		250		15				
Vegetables β-carotene								
Fruit vit C		250		15				
Vegetables vit C								
Fruit other		250		15				
Vegetables B		150	2	7				
Bread and cereal		285	3	15				
Fats and oils		190			5			
Sweets/Sugar		170		10				
Alcohol								
TOTAL								

After performing the calculation in Table 3.5, estimated values of total energy, carbohydrates, protein and fat intake were calculated and coded.

Table 3.6: Prudent dietary guidelines (recommended macronutrient proportions) (USDA & USDHHS, 2010:15,Table 2-4)

Population age group	Carbohydrates	Protein	Fat
Children 1-3 years	45-65%	5-20%	30-40%
Children 4-18 years	45-65%	10-30%	25-35%
Adults 19 years and older	45-65%	10-35%	20-35%

Results were also compared to prudent dietary guidelines (Table 3.6), as well as to the recommended dietary reference intakes for different age groups (Table 3.7).

Table 3.7: Dietary Reference Intakes (DRIs): recommended daily intakes for macronutrients and acceptable distribution ranges [adapted to kJ] (Food & Nutrition Board et al., 2002:1324)

Age group	0-6 months	7-12 months	1-3 years	4-8 years	Men 19-70 years	Women 19-70 years
Energy (kJ)	**2394**♂ **2184**♀	**3121**♂ **2839**♀	**4393**♂ **4166**♀	**7316**♂ **6896**♀	**12881**	**10093**
CHO* (g)	**60**	**95**	**130**	**130**	**130**	**130**
Protein (g)	**9.1**	**11.0**	**13**	**19**	**56**	**46**
Fat (g)	**31**	**30**	**Not determined**			

* carbohydrates

3.5.1.2 Children

The Food Guide Pyramid for young children two to six years old (CNPP), 1999:online) together with information set out in Tables 3.1, 3.3, and 3.7 were used for the analysis of the dietary intake for children between two and six years of age. Portion sizes for children were used as set out in Table 3.8.

Table 3.8. Portion sizes for children, depending on age (Earl, 2004:378, Figure 15-4; Lucas & Feucht, 2008:233, Table 7-4; Lucas, 2004:273, Table 10-3; CNPP, 1999:online)

Grain group	**Fruit group**	**Meat group**
½ to 1 slice of bread	1 piece of fruit or melon wedge	30 to 90g of cooked lean meat, poultry, or fish
½ cup of cooked rice or pasta	¾ cup of juice	1 egg = 30 g
¼ to ½ cup of cooked porridge	½ cup of canned fruit	½ cup of cooked dry beans = 30 g
½ to 1 cup of ready-to-eat cereal	¼ cup of dried fruit	2 tablespoons of peanut butter (add one fat exchange) = 30 g
Vegetable group	**Milk group**	**Fats and sweets**
½ cup of chopped raw or cooked vegetables	½ to1 cup of milk or yogurt	2 teaspoons sugar
1 cup of raw leafy vegetables	60g of cheese	2 hardboiled sweets
		5ml oil or margarine

Energy and macronutrients were calculated based on the food exchange lists and compared to the recommendations of the Food Guide Pyramid for children (CNPP, 1999:online). Food intakes less than the recommendations of the Food Guide Pyramid for children were regarded as inadequate or below requirements; intake within the guidelines, as adequate or within requirements; and intake higher than the guidelines, as high or above requirements.

For children younger than two years, the guidelines as set out by LeLeiko et al. (1995:218-219, Table 13.4 and Table 13.5) and listed here in Tables 3.9. and 3.10, were used to categorize the dietary intake of children. A different dietary intake form was completed for children 0-2 years (Appendix F). If the child also consumed foods other than breast milk or infant formula, a 24-hour recall form of

reported usual intake was completed (Appendix D). If a child was older than two years only a 24-hour recall form of reported usual intake was completed (Appendix D).

Table 3.9. Guidelines for suggested servings for infants from birth through eight months to meet the Recommended Daily Allowance (LeLeiko et al., 1995:218, Table 13.4)

Age	***Foods***	***Portions***
Birth to two months	Breast milk with supplemental vitamin D and iron after four months or commercially prepared iron-fortified formula	6 - 8 bottles (60 – 90 ml each) i.e. 360 – 720 ml per day
Three months	Breast milk or commercially prepared iron-fortified formula	5 - 6 bottles (120 – 180 ml each) i.e. 600 – 1080 ml per day
Four months	Breast milk or commercially prepared iron-fortified formula. Iron-fortified infant cereal	5 bottles (180 – 210 ml each) i.e. 900 – 1050 ml per day Start with ½ teaspoon mixed with formula, increase to 4 tablespoons
Five months	Breast milk or commercially prepared iron-fortified formula. Iron-fortified infant cereal Strained fruit and vegetable	5 bottles (180 - 240 ml each) i.e. 900 – 1200 ml per day 4 - 5 tablespoons ½ - 1 jar of each
Six months	Breast milk or commercially prepared iron-fortified formula. Iron-fortified infant cereal Strained fruit and vegetable Strained meat	4 – 5 bottles (180 – 210 ml each) i.e. 720 – 1050 ml per day 4 – 5 tablespoons 1 jar of each ½ jar
Seven to eight months	Breast milk or commercially prepared iron-fortified formula. Iron-fortified infant cereal Junior or soft-cooked fruit and vegetable Junior meat or finely chopped table food	4 bottles (210 – 240 ml each) i.e. 840 ml – 960 ml ¼ cup (i.e. 62,5 ml) 1 jar each 1 jar

It was difficult to determine the amount of breast milk consumed per day. Thus, it was only noted whether children were either breastfed or not. The child's age was taken into consideration when looking at the amounts of times the mother breastfed per day and the intervals. If the mother breastfed about six to eight times a day for a period of about fifteen to twenty minutes, this was considered adequate intake (Trahms, 2004:205, 227).

Table 3.10. Suggested daily servings for infants and children (ages eight months+) to meet the Recommended Daily Allowance (LeLeiko et al., 1995:219, Table 13.5)

Food group	***Infant 8-12 months***	***Infant 1-2 years***	***Toddler 2-4 years***	***Pediatric (5 years+)***
Milk	2 ½ - 3 cups (iron-fortified infant formula)	2 cups	3 cups	3 or more cups
Meat, protein	60 – 120 g, strained or finely chopped	120 – 150 g, finely chopped	150 – 180 g, chopped or whole	180 g or more
Vegetables (at least one good source of vitamin A)	2 servings (⅛ cup)	2 servings (⅛ cup)	2 servings (¼ cup)	2 servings (½ cup)
Fruit (at least one good source of vitamin C)	3 servings (⅛ cup)	3 servings (⅛ cup)	3 servings (¼ cup)	3 servings (½ cup)
Bread, cereal, starch	4 servings (½ slice bread, ¼ cup cereal)	4 servings (½ slice bread, ½ cup cereal)	4 servings (1 slice bread, ½ cup cereal)	5 servings (1 slice of bread, ¾ cup cereal)
Fat	2 teaspoons or to meet energy needs	1 teaspoon or to meet energy needs	1 tablespoon or to meet energy needs	1 tablespoon or to meet energy needs
Sweets	To meet energy needs	To meet energy needs	To meet energy needs	To meet energy needs

Intake of one fruit or vegetable exchange rich in vitamin A or β-carotene per day, one fruit or vegetable exchange rich in vitamin C per day, and at least one other fruit or vegetable exchange per day was considered as adequate intake. Less than these amounts was considered inadequate or below requirements. When considering the 5-a-day challenge, i.e. an intake of a total of five fruit and

vegetables a day, participants were divided into two groups, i.e. those who consumed less than a total of five fruit and vegetables a day, and those who consumed five or more per day.

Starchy vegetables and legumes were classified under the same, i.e. starchy vegetables were counted as a starch exchange and not a vegetable exchange.

3.5.2 Anthropometry

For the purposes of this study, the term "malnutrition" included both undernutrition, as well as overnutrition (classified as either overweight or obesity). Anthropometric variables for adults included: height; weight; BMI; waist circumference; hip circumference; wrist circumference to estimate frame size; and mid-upper arm circumference. All measurements were recorded on an anthropometry form (Appendix G). Demi-span and knee height were only measured in patients who were unable to stand. The following skin fold thicknesses were also measured to calculate participants' body fat percentages: biceps; triceps; supra-iliac; subscapular; calf and thigh. Anthropometric variables for children included: height; weight; body-mass-index and head circumference. Data was analyzed according to the guidelines as set out by Tables 3.11, 3.15, 3.16, and 3.17.

3.5.2.1 Adults

(i) Body-mass-index (BMI)

A participant's weight and height were used to calculate body-mass-index. Weight was taken in kilograms to the value of one decimal point. Height was taken in centimeters to the value of one decimal point. A participant's body-mass-index (BMI) was calculated to determine whether adults were obese (BMI >30 kg/m^2), overweight (BMI from 25 kg/m^2 to 29,99 kg/m^2), normal weight (BMI from 18,5 to 24,99 kg/m^2), or underweight (BMI <18,5 kg/m^2) (Gibson, 2005:263, Table 10.11; CDCP, 2009:online).

BMI was calculated by using the average of the weight measurement (in kilograms) and average of the height measurement (in meters) previously measured and inserting the values into the following formula:

BMI = weight in kg ÷ (height in m x height in m).

(ii) Waist circumference

Waist circumference was also used to determine a participant's risk of developing CDLs as set out in Table 3.11. A measurement of between 94,0 cm and 101,9 cm in men and between 80,0 cm and 87,9 cm in women indicated an increased risk for CDLs. A measurement $\geq$102 cm in men and $\geq$88 cm in women indicated a substantial risk for CDLs (Gibson, 2005:284; Lee & Nieman, 2010:180, Table 6.7; CDCP, 2009:online; WHO/FAO, 2002:69).

Table 3.11 Guidelines for interpretation of waist circumference and risk for chronic diseases of lifestyle (Lee & Nieman, 2010:180, Table 6.7; Gibson, 2005:284; Lee & Nieman, 2003:183, Table 6.8).

	Ideal	**Increased risk**	**High risk**
Men	< 94cm	94,0 - 101,9cm	≥ 102 cm
Women	< 80cm	80,0 - 87,9cm	≥ 88cm

(iii) Estimated weight and height

For adult participants who could not stand, demi-span, mid-upper arm circumference and knee height were measured, where after the values were applied to formulae according to the participant's age, race and gender, as set out in Table 3.12 and Table 3.13 to calculate estimated weight and height respectively.

Table 3.12. Equations for estimating body weight (W) from knee height (KH) and mid-arm circumference (MAC) for various groups (Lee & Nieman, 2010:225, Table 7.4; Lee & Nieman, 2003:227, Table 7.4)

Age	Race	Equation
Females		
6-18	Black	W = (KH X 0,71) + (MAC X 2,59) – 50,43
6-18	White	W = (KH X 0,77) + (MAC X 2,47) – 50,16
19-59	Black	W = (KH X 1,24) + (MAC X 2,97) – 82,48
19-59	White	W = (KH X 1,01) + (MAC X 2,81) – 66,04
60-80	Black	W = (KH X 1,50) + (MAC X 2,58) – 84,22
60-80	White	W = (KH X 1,09) + (MAC X 2,68) – 65,51
Males		
6-18	Black	W = (KH X 0,59) + (MAC X 2,73) – 48,32
6-18	White	W = (KH X 0,68) + (MAC X 2,64) – 50,08
19-59	Black	W = (KH X 1,09) + (MAC X 3,14) – 83,72
19-59	White	W = (KH X 1,19) + (MAC X 3,21) – 86,82
60-80	Black	W = (KH X 0,44) + (MAC X 2,86) – 39,21
60-80	White	W = (KH X 1,10) + (MAC X 3,07) – 75,81

Table 3.13. Equations for estimating height (S) from knee height (KH) for various groups (Lee & Nieman, 2010:219, Table 7.1; Gibson, 2005:152; Lee & Nieman, 2003:221, Table 7.1)

Age (A)	Equation
Black females	
> 60	S = 58,72 + (1,96 KH)
19-60	S = 68,10 + (1,86 KH) – (0,06 A)
6-18	S = 46,59 + (2,02 KH)
White females	
> 60	S = 75,00 + (1,91 KH) – (0,17 A)
19-60	S = 70,25 + (1,87 KH) – (0,06 A)
6-18	S = 43,21 + (2,14 KH)
Black males	
> 60	S = 95,79 + (1,37 KH)
19-60	S = 73,42 + (1,79 KH)
6-18	S = 39,60 + (2,18 KH)
White males	
> 60	S = 59,01 + (2,08 KH)
19-60	S = 71,85 + (1,88 KH)
6-18	S = 40,54 + (2,22 KH)

(v) Total body fat percentage

The six skinfolds, namely: triceps; biceps; subscapula; supra-iliac; thigh; and calf, were measured with a calibrated calliper and classified into three categories, i.e. <15th percentile, between 15th and 75th percentile, or >75th percentile. The six skinfolds were then used in the six-skinfold formulae as set out in Table 3.14, to determine the total percentage body fat in each participant.

Table 3.14. Six-skinfold formulae (Lee & Nieman, 2003:169)

Gender	Formula
Female	[(Sum of 6 skinfolds) X 0,1548] + 3,58
Male	[(Sum of 6 skinfolds) X 0,1051] + 2,585

The resulting answer was then used to categorize fat percentage of participants according to the cut-offs in Table 3.15.

Table 3.15. Body fat ranges for persons 18 years of age or older
(Lee & Nieman, 2010:193, Table 6.11; Lee & Nieman, 2003:195, Table 6.12)

Classification	Males	Females
Unhealthy range (too low)	≤ 5%	≤ 8%
Acceptable range (lower end)	6 – 15%	9 – 23%
Acceptable range (upper end)	16 – 24%	24 – 31%
Unhealthy (too high)	≥ 25%	≥ 32%

3.5.2.2 Children

In children, weight-for-age, height/length-for-age; weight-for-height; BMI-for-age, and head circumference were interpreted by means of the WHO standards (WHO, 2010:online).

The WHO based their standards for children from birth to six years old on primary data collected through the WHO Multicentre Growth Reference Study (MGRS). The MGRS was a population-based study conducted between 1997 and 2003 in the countries of: Brazil; Ghana; India; Norway; Oman; and the USA. The MGRS combined a longitudinal follow-up from birth to 24 months of age with a cross-sectional component of children aged 18–71 months (De Onis et al., 2007: 144)

The WHO recommends the use of Z-scores for evaluating anthropometric data from low-income countries, because Z-scores can be calculated accurately beyond the limits of the original reference data. This is an advantage in low-income countries because individuals with indices below the extreme percentiles of the reference data can then be classified accurately. The method measures the deviation of the anthropometric measurement from the reference mean or median in terms of standard deviations or Z-scores. The score is a measure of an individual's value with respect to the distribution of the reference population. The exact values for the Z-score of an individual can be calculated using selected reference standard deviation (SD) values for the WHO reference population (Gibson, 2005:337, Table 13.1). The WHO found that their new growth reference chart for 5-19 year olds, "fill the gap in growth curves and provide an appropriate reference for the five to 19 years age group" (WHO, 2010a:online).

Z scores of -2 were designated as indicating risk of severe malnutrition, whereas scores above +2 were taken to indicate obesity (Gibson, 2005:338). Stunting was classified as a height-for-age below minus two standard deviations (<-2SD) from the reference median (Gibson, 2005:256). Underweight was classified as weight-for-age below minus two standard deviations (<-2SD) from the reference median (Gibson, 2005:254). Wasting was classified as weight-for-height below minus two standard deviations (<-2SD) from the reference median (Gibson, 2005:255). A high weight-for-height (>+2 SD or BMI >95th percentile) in children was termed overweight (Gibson, 2005:255, 338; Lucas, 2004a:276). Severely underweight was classified as weight-for-age below minus three standard deviations (<-3SD) from the reference median. Severely wasted was classified as weight-for-height below minus three standard deviations (<-3SD) from the reference median. Severe stunting was classified as height-for-age below minus three standard deviations (<-3SD) from the reference median (Torûn, 2006:890).

Weight-for-age, height-for-age and weight-for-height data was analyzed according to the guidelines set out in Tables 3.16 and 3.17.

Table 3.16 Categories of classification of all data in children

Standard deviation range	***Classification***		
	Weight-for-age	**Height-for-age**	**Weight-for-height**
> + 3SD	Severely overweight	Very tall	Severely high
+2.1 to +3 SD	Moderately overweight	Moderately tall	Moderately high
+1.1 to +2 SD	Mildly overweight	Mildly tall	Mildly high
-1SD to +1 SD	Normal	Normal height	Normal
-1.1 SD to -2SD	Mildly underweight	Mildly stunted	Mildly low
-2.1SD to -3SD	Moderately underweight	Moderately stunted	Moderately low
< -3SD	Severely underweight	Severely stunted	Severely low

Table 3.17 Classification of malnutrition (Torûn, 2006:890)

Standard deviation range	***Classification***
-1SD to +1 SD	Normal
-1.1 SD to -2SD	Mild malnutrition
-2.1SD to -3SD	Moderate malnutrition
< -3SD	Severe malnutrition

3.6. Pilot Study

A pilot study was conducted one month prior to the main study, on a similar group of participants in the same rural and urban communities. The pilot study was conducted to determine whether all questions could be clearly understood and to standardize the taking of anthropometric measurements. Five participants were interviewed from the rural and five participants from the urban area. The questionnaires were then revised, where necessary.

As a result of the pilot study a need was identified to clearly indicate the portion sizes of certain foods that were not available in the food models. For this reason, wax food models were made where a need was identified for portion sizes of cultural foods (e.g. mealie meal porridge). Food examples were also bought from street vendors who were frequented by participants, to use as models in the main study.

Anthropometric measurements and interviewing techniques were also practiced on five participants, but no bloods were drawn. The time duration to complete each interview was determined. It was found that it was impractical and expensive to include the use of bioelectrical-impedance measurements, and therefore they were not included in the main study. The necessary formulae calculations and analysis of data were also done in order to identify discrepancies or inaccuracies. None were found.

3.7. Data collection process

The study was planned to coincide with the service learning functions of the University of the Free State. Therefore, two key community service delivery sites (flagship partnerships) were included, namely the FSRDPP for the rural baseline study; and the MUCPP for the urban baseline study. For the purpose of this sub-study, only results pertaining to dietary intake and anthropometry were included. The data collection process proceeded as follows:

- All necessary approvals for the study were obtained (Ethics Committee; Department of Health; municipalities; and community leaders).

- Participants were identified according to the sample selection via maps, as discussed previously under Section 3.4.2.

- A pilot study was also conducted as discussed previously under Section 3.6.

- Field workers were appointed in each community (with the aid of the community health committee) and received training to explain the purpose of the study to eligible participants and to obtain written informed consent before data collection (Appendix A).

- Students were trained on how to complete questionnaires correctly. Written informed consent was obtained from selected household members and parents or caretakers of children in the language of their choice (Sotho, English or Afrikaans). Parents or guardians signed consent forms on behalf of any pre-school children.

- Each participant also received an information document, detailing all relevant information regarding the study (Appendix B).

- A participation letter (Appendix C) was given to each selected household member in the language of their choice (Sotho, English or Afrikaans) by field workers to inform them of the date that they should be at the research venue to participate in the study as well as the procedures that were to be followed (e.g. no eating from the previous evening). This letter could also be used to inform employers of the study and the reason for not being able to attend work on that day. Any questions the participants had were answered by the field workers.

- On the days of data collection participants were introduced to the researchers, and a checklist card was issued to each participant. All checkpoints that needed to be visited by the participant were listed on the card. The field workers assisted with Sotho interpretation, orienting participants as to all the information points that they needed to visit (i.e. bloods and medical examination; anthropometry; health questionnaire; knowledge, attitude and practices of eating behaviour; socio-demographic; household food security; dietary intake; and physical activity)

- Questionnaires were completed by trained 4th year students and this Masters degree student during interviews with participants, under the supervision of the study leader (Appendices D, E, F and G).

- After the data collection process, the completed dietary intake questionnaires (Appendices D, E and F) and anthropometry forms (Appendix G) were processed and coded by this Masters degree student, whereafter they were sent to the Department of Biostatistics of the Faculty of Health Sciences for data entry and statistical analysis as further discussed under Section 3.9.

3.8. Techniques

3.8.1 Dietary intake

3.8.1.1 24-hour recall of reported usual intake

The information gathered from the dietary intake of the last 24 hours (as in a 24-hour recall), is not representative of an individual's usual intake (Langenhoven & Wolmarans, 1997:250). For this reason a 24-hour recall of reported usual intake (Appendix D) and adjusted food frequency questionnaire (Appendix E) were used to determine dietary intake for a typical week during individual interviews with

each participant. Reported usual intake for weekends was also recorded, which may have been different from intake during the week. Therefore, the adjusted FFQ (Appendix E) was also completed to ascertain a more typical weekly intake. However, with all dietary data gathered, over- and underreporting could still have occurred.

Participants were requested by trained students to report their usual dietary intake for a 24 hour period, by means of a multi-pass 24-hour recall, consisting of (1) a quick list of uninterrupted recall by the respondent from time of arising in the morning until retiring at night; (2) a detailed interview elaborating on the quick list, where questions were asked about portion sizes, cooking methods and types of foods (e.g. low fat- vs. full cream milk / brown- vs. white bread); and (3) a thorough review of the detailed interview, whereby the interviewer read back the data to the participant for confirmation and/or clarification. The dietary intakes of children were determined by interviewing the parent or caregiver most often responsible for their meals.

Where possible, portion sizes were quantified using the food models designed by Life-form® (Nesco, USA). Where food models were not available for cultural foods, regular household utensils, cups and plates used by the population were used to determine portion sizes. Where participants bought food items from vendors in the vicinity of the study, examples were purchased from the vendors, weighed, set equal to exchanges and presented to the participants as food models. Quantified wax models (e.g. ½ cup, 1 cup, etc.) were also made of cooked mealie-meal porridge, since this was one of the identified staple-foods of the population during the pilot study.

Researchers completed all questionnaires. A Sotho translator was used if the individual could not understand English or Afrikaans. The reported intake was converted to the number of servings as listed on the exchange lists, in order to quantify energy and macronutrient content in usual dietary intake.

After the 24-hour recall of reported usual intake was analyzed according to all items consumed and the items were described in terms of exchanges; the calculation table as shown in Table 3.5, was completed. Intake of each food group was also coded as below, within or above requirements.

The exchange lists were used to quantify the energy and macronutrient content of the dietary intakes of all participants. The exchange lists were used to quantify the 24-hour recall of reported usual intake retrospectively. It must be noted that potatoes and other starchy vegetables, such as corn and sweet potato, were counted as a starch/grain exchange and not a vegetable exchange. Legume exchanges were also counted separately, and entered into the exchange list calculation form (Table 3.5).

3.8.1.2 FFQ

In adults and children older than two years, the adjusted FFQ (Appendix E) was used to cross-check the usual dietary intake and also to determine the twenty most frequently consumed foods. The food frequency questionnaire was used to ensure that patients had reported all additional drinks and other foods not reported in the 24 hour recall. The adjusted FFQ was completed by researchers in a detailed interview with each participant during which the researchers asked the participant to report as to the frequency of consumption of twenty selected food items, shown to be most often consumed. This

questionnaire was also of particular significance to confirm more accurately the typical weekly intake of participants and to determine the most commonly consumed foods by frequency of intake..

3.8.2. Anthropometric measurements

As discussed previously under Section 3.5.2., height, weight, waist circumference and hip circumference were taken of non-disabled adults. Although there were few disabled participants, demi-span, knee height and mid-upper arm circumference were included for disabled adults.

3.8.2.1 Weight

Weight was taken in kilograms to the value of one decimal point. The floor surface where the scale was placed was firm and level and no measurements were taken on a carpeted floor. Weight was taken with a calibrated digital electronic SECA scale. The participant wore a minimum amount of clothes and no shoes. The scale was zeroed before measurement and calibration was accurate. The participant stood in the middle of the scale without support. Weight was evenly distributed between both feet. The participant stood upright and looked straight ahead (Lee & Nieman, 2010:164; Lee & Nieman, 2003:167-168). Three consecutive measurements were taken in kilogram, accurate to one decimal point. After all three measurements were taken, the average of the three values was calculated and noted on the anthropometry form (Appendix G) where indicated.

All infants were weighed with the minimum amount of clothing and without a nappy to the nearest 0,01 kg on an infant scale (Lee & Nieman, 2010:164; Lee & Nieman, 2003:167; ADA, 2000:48).

If a child could stand, he/she was weighed on an adult scale. The scale was placed on a hard flat surface and adjusted to zero before each measurement. The child wore a minimal amount of clothing, and no shoes. The child stood in the centre of the platform and looked straight ahead, standing unassisted and relaxed (Lee & Nieman, 2010:164; Gibson, 2005:253; Lee & Nieman, 2003:167; ADA, 2000:12).

Where it was difficult to weigh a child alone, the mother and subject were weighed together using a calibrated scale, with an accuracy to one decimal point (0,1 kg). Thereafter, the mother was weighed alone. The subject's weight was then calculated by subtracting the two values (Gibson, 2005:252).

3.8.2.2 Height

Height was taken in centimeters to the value of one decimal point. The participant stood with feet together, with the participant's heels, buttocks and upper part of the back touching the stadiometer. The head was placed in the Frankfort horizontal plane, by the person taking the measurement placing her hands under the participant's jaw and her fingertips on the mastoid process. The participant then took a deep breath while the measurer delicately placed the head in the Frankfort plane. Then the head board was moved down firmly onto the vertex of the head, with all hair pressed down. The measurer ensured that the participant did not lift his/her feet from the floor and that the head was in the Frankfort plane before taking the reading. The reading was taken at the end of the deep breath (Lee & Nieman, 2010:161-162; Lee & Nieman, 2003:166-167; ADA, 2000:11). The value (in centimeters) was converted to meters before applying the value to the BMI calculation.

Height/length of children up to three years of age was taken by the ruled board method, if the stadiometer method was impossible. Measurement was taken by two measurers. One measurer held the child's head, while the other moved the foot board and took the measurement. The child was laid on the board and stretched out on the board for the most accurate measurement. The moveable flat end was placed against the bottom of the child's feet and the result was read up to an accuracy of one decimal point in millimeters. If the subject was restless only the left leg was positioned for the measurement (Gibson, 2005:246; ADA, 2000:48).

If a child was older than two years and taller than 85cm, standing height was measured to the nearest 0,1 cm with a stadiometer (ADA, 2000:48) via the same method as described above for adults. The child's heels were held by the measurer to ensure they did not leave the ground.

3.8.2.3 Circumferences

(i) Waist circumference

The umbilicus level was used as the site of measuring the waist circumference in this study. The measurer stood in front of the participant. The participant stood straight with abdomen relaxed. Feet were together and arms hung at the sides. The participant was asked to take a normal breath and the waist circumference was recorded when the participant breathed out. The measuring tape followed a level and straight circumference. The tape was snug against the skin, but not so tight as to compress the skin. Three consecutive measurements were taken in centimeters, with accuracy to one decimal point (Lee & Nieman, 2010:180; Lee & Nieman, 2003:183).

(ii) Hip circumference

Hip circumference was measured at the widest point of the posterior protrusion of the buttocks. The measurer stood on the right side of the participant to ensure that the measuring tape followed a horizontal circumference when taking the measurement. The participant's feet were together and the gluteal muscles relaxed. Arms hung at the sides. The measuring tape was placed around the subject's widest part of the hip and the value recorded. Three consecutive measurements were taken, with accuracy to the nearest millimeter (Gibson, 2005:281).

(iii) Head circumference

Measurements were made just above the eyes to include the maximum circumference of the head, with the child supported in an upright position and looking straight ahead (Shaw & Lawson, 2001:5). The child's head was held still. The measuring tape was placed over the most prominent part of the forehead, just above the supra-orbital ridge. It was ensured that the measuring tape was not loose and the measurement was taken in centimeters, to the nearest decimal point (Lee & Nieman, 2010:163; Gibson, 2005:246).

(iv) Mid-upper arm circumference

Mid-upper arm circumference was used, together with the knee height value in order to determine the participant's weight. Three consecutive measurements were taken. Mid-upper arm circumference was only taken if the adult could not stand.

The circumference was taken of the right arm, unless the participant's right arm had been amputated. Circumference was measured in centimeters using a standardized, calibrated, flexible, non-stretch measuring tape. The subject stood erect and sideways to the measurer, with the head in the Frankfort plane, arms relaxed, and legs apart. If the subject was wearing a sleeved garment, it was removed or the sleeves rolled up. The measurement was taken at the midpoint of the upper arm, between the acromion process and the tip of the olecranon. The distance halfway between the acromion process of the scapula and the radial point of the elbow was measured. The acromion process was found when the measurer palpitated the upper point of the scapula towards the corner of the acromion. The corner represented the uppermost point of the upper arm (A). The radial point of the elbow was represented by the lowest point of the upper arm (B). The radial point was found by lightly moving the elbow, while the point was palpitated. To determine the middle point of the upper arm, the arm had to be in the anatomical position. The middle point was between points A and B. The middle point was correctly projected to the triceps and biceps. Mid-upper arm circumference was measured at this middle point. After locating the midpoint, the arm was extended so that it was hanging loosely at the side, with the palm facing inward. The tape was then wrapped gently but firmly around the arm at the midpoint, care being taken to ensure that the arm was not squeezed (Gibson, 2005:290).

(v) Wrist circumference

The participant's right arm was flexed at the elbow with the palm facing upward and the hand muscle relaxed. A non-stretch measuring tape, no wider than 0,7 cm, was placed around the participant's right wrist just distal to the styloid processes of the radius and ulna. The measurer also ensured that the tape was perpendicular to the long axis of the forearm, and that the tape only touched the skin, not compressing the soft tissue. Measurement was recorded to the nearest 0,1cm (Lee & Nieman, 2010:175; Lee & Nieman, 2003:178).

3.8.2.4 Knee height

Knee height was taken in the seated or reclining position. Knee height was measured with a sliding calliper consisting of an adjustable measuring stick with a blade attached to each end at a ninety degree angle. The measurement was taken using the left leg, which was bent at the knee at a ninety degree angle. One blade of the broad-blade calliper was placed under the heel of the left foot flexed at ninety degrees. The second blade was placed over the anterior surface of the left thigh above the condules of the femur and just proximal to the patella. The shaft of the calliper was held parallel to the shaft of the tibia and gentle pressure was applied to the blades of the calliper. At least two consecutive measurements were made and they agreed within five millimeter, where after the mean was then calculated (Gibson, 2005:251; Mahan & Escott-Stump, 2008b:1213, Appendix 20; Mahan & Escott-Stump; 2004c:1184, Appendix 15; Lee & Nieman, 2010:219-220; Lee & Nieman, 2003:167). The average value was then noted on the form where indicated (Appendix G).

3.8.2.5 Skinfolds

Proper measurement of skinfolds required careful attention to site selection and strict adherence to the protocol as set out by Lee and Nieman (2010:183; 2003:186) . Measurements were all noted on the form attached as Appendix G.

(i) Triceps

The triceps skinfold was taken at the midpoint of the upper right arm, between the acromion process and the tip of the olecranon, with the arm hanging relaxed. To mark the midpoint, the participant's right arm was bent ninety degrees at the elbow, and the forearm was placed palm down across the body. The tip of the acromion process of the shoulder blade at the outermost edge of the shoulder, and the tip of the olecranon process of the ulna, were located and marked. The distance between these two points was measured using a non-stretch measuring tape, and the midpoint was marked with a soft pen or indelible pencil, directly in line with the point of the elbow and acromion process. The right arm was then extended to hang loosely by the side. The measurer grasped a vertical fold of skin plus the underlying fat, at a point two centimeter above the marked midpoint, in line with the tip of the olecranon process, using both the thumb and the forefinger. The skinfold was gently pulled away from the underlying muscle tissue, and then the calliper jaws were applied at right angles, exactly at the marked midpoint. The skinfold remained held between the fingers while the measurement was taken (Lee & Nieman, 2010:186; Gibson, 2005:276). Three consecutive measurements were taken.

(ii) Biceps

The mid-upper-arm point was located and the participant relaxed the right arm. A vertical skinfold at one centimeter above the midpoint of the biceps was grasped and the calliper was applied. The measurement was recorded directly after the calliper reading stabilized (Gibson, 2005:277). Three consecutive measurements were taken.

(iii) Supra-iliac

The supra-iliac skinfold was measured just above the iliac crest at the mid-axillary line. The participant stood erect with feet together and arms hanging at the sides. The measurer grasped the skinfold about one centimeter posterior to the mid-axillary line and measured the skinfold at the mid-axillary line (Lee & Nieman, 2010:187-188; Lee & Nieman, 2003:189). Three consecutive measurements were taken.

(iv) Subscapular

The subscapular site is one centimeter below the lowest angle of the scapula. The long axis of the skinfold is on a 45° angle directed down and to the right side. The site was located by gently feeling for the inferior angle of the scapula or by having the participant place the arm behind the back. It was measured with the participant standing with arms relaxed to the sides. The skin was grasped one centimeter above and medial to the site along the axis (Lee & Nieman, 20103:187; Lee & Nieman, 2003:187). Three consecutive measurements were taken.

(v) Calf

With the participant sitting, the right leg was flexed about ninety degrees at the knee with the sole of the foot flat on the floor. The point of maximum calf circumference was marked at the inner aspect of the calf. A vertical skinfold was grasped about one centimeter proximal to the marked site and measured at the site (Lee & Nieman, 2010:189; Lee & Nieman, 2003:191). Three consecutive measurements were taken.

(vi) Thigh

This site is a vertical skinfold along the midline of the anterior aspect of the thigh midway between the junction of the midline and the inguinal crease and the upper border of the patella. The participant was required to shift the weight to the left foot and to relax the leg being measured by slightly flexing the knee with the foot flat on the floor (Lee & Nieman, 2010:188; Lee & Nieman, 2003:190). Three consecutive measurements were taken.

3.9. Statistical analysis

Adequacy of diets was evaluated by comparing the intake of each participant to the intake recommended by the Food Guide Pyramid. The Food Guide Pyramid (Earl, 2004:368, Table 15-2; CNPP, 1999:online; USDA, 1992:online) was used to determine whether participants' dietary intake of each food group fell below requirements (inadequate); within the guidelines (adequate); or above requirements.

Food, energy and macronutrient intake; were described by means of descriptive statistics. Descriptive statistics, namely frequencies and percentages for categorical data; and means and standard deviations or medians and percentiles for continuous data, were calculated. Differences between urban and rural groups were assessed using 95% confidence intervals or p-values, as applicable, for the difference in rural and urban proportions.

An association between anthropometric status of children and adults of the same household was also investigated.

Data was analyzed with the assistance of the Department of Biostatistics of the Faculty of Health Sciences, University of the Free State using *SAS*® software (SAS, 2001).

3.10. Validity and reliability

The validity of a test or instrument refers to its ability to measure the phenomenon it intends to measure. The reliability of a test or instrument is determined by the consistency of results when applied repeatedly by either the same or different persons (Monsen, 1992:13).

3.10.1. Dietary intake questionnaires

A dietary assessment method is considered precise (i.e. reliable/reproducible) if it gives very similar results when repeated in the same situation (Koh & Owen, 2000:182).

3.10.1.1 Validity

The performance of any dietary assessment method is markedly influenced by the motivation and compliance of the respondents (Gibson, 2005:161).

A 24-hour recall of reported usual intake was used to determine the usual intake of foods and drinks by participants. Participants were asked to report usual dietary intake for a usual 24 hour period. Information related to food preparation methods were also obtained. Because the information related to dietary intake was only used to determine the intake of different foods and not nutrients, this method was considered valid.

An adjusted FFQ was used to ensure that participants had reported all additional drinks and other foods not reported in the 24 hour recall of reported usual intake. Careful attention was given to the choice of foods, the clarity of the questions and the format of the frequency response section, when constructing the FFQ (Koh & Owen, 2000:187). By using an adjusted FFQ, which listed commonly consumed foods of the target population, results could be more representative of the population's actual intake of cultural foods.

3.10.1.2 Reliability

A dietary assessment method is considered reliable if it gives very similar results when used repeatedly in the same situation. Replication observations in dietary assessment are impossible, and therefore true reproducibility is very difficult to determine (Gibson, 2005:129).

Single 24-hour recalls can be used to assess actual intakes of foods and nutrients, even though any estimate of an individual's usual intake, based on a single 24-hour recall has low reproducibility because of relatively large within-subject variation in food intake. The reproducibility of a 24-hour recall can be improved by obtaining several 24-hour recalls for the same individual, preferably on non-consecutive days (Gibson, 2005:131) or by employing a 24-hour recall of reported usual intake, as was done in this study, for practical reasons.

For practical reasons, the adjusted FFQ questionnaire used in this study was also only administered once to each participant and thus no measure of reliability was included.

3.10.2. Anthropometry

Validity and reliability of anthropometric results were ensured in the following ways:

3.10.2.1 Validity

Validity was ensured by including variables and methods recommended in the literature to measure anthropometric parameters. By using these variables and measures, results could be compared to the results of other studies that had used the same variables and techniques.

3.10.2.2 Reliability

Researched were extensively trained by a certified anthropometrist to ensure accuracy of measurements. Measurements were repeated three times on each study participants and the average used to ensure reliability. Researchers used the same, standardized techniques, the appropriate measuring tools and calibrated equipment.

Chapter 4 - Results

The dietary intake and anthropometric status of the children and adults will be described hereafter.

4.1 Dietary intake

Dietary intake of children younger than two years, children two to seven years and adults (25 to 64 years old) will be described in the following sections.

4.1.1 Early feeding

Sample sizes for children younger than two years of age for both rural and urban participants were relatively small, and this should be kept in mind when interpreting the following results.

In the rural areas, 16 children in the sample were younger than two years and in urban areas 54 participants were younger than two years. Of the 16 rural participants, eight were male (50%) and eight were female (50%). The median age was 13,5 months. The youngest participant was four months old and the oldest 24 months (2 years). Of the 54 urban participants, 29 were male (53,7%) and 25 were female (46,3%). The median age was nine months. The youngest participant was one month old and the oldest was 24 months (2 years).

Results on breastfeeding prevalence, period of breastfeeding, time of introduction of solids foods and the use of formula milk were obtained from the dietary intake questionnaire for children up to two years old (Appendix F). If a child ate solid foods at the time of the study, dietary intake information was also obtained from a 24 hour recall of reported usual intake (Appendix D).

Where possible, 95% confidence intervals for median differences in categorical data were used to indicate significance of differences between urban and rural young children.

4.1.1.1 Breastfeeding

As reflected in Table 4.1, of all participants younger that two years of age, only 5 (31,25%) rural children and 22 (40,7%) urban children were being breastfed at the time of the study.

Table 4.1: Breastfeeding prevalence

	Rural (n=16)		***Urban (n=54)***		***95% CI on median difference***
Status	**N**	**%**	**N**	**%**	
Breastfeeding	5	31.25	22	40.7	[-31.1; 17.7]
Not breastfeeding	11	68.75	32	59.3	

Of the rural participants who were breastfeeding at the time of the study, one had already been breastfeeding for 12 weeks, one for twenty weeks and two for 52 weeks (i.e. 1 year). In urban participants, the median period of breastfeeding was thirty weeks. As indicated by the 95% CI, there was no significant difference in breastfeeding prevalence between rural and urban children.

The largest percentage (18,2%) of urban participants had already been breastfeeding for twenty weeks. The longest period that an urban participant had been breastfed was 88 weeks (1 year 9 months) (Table 4.2).

Table 4.2: Period of current breastfeeding

	Weeks	11	12	16	20	24	28	32	44	50	52	58	64	72	86	88
Rural n=4		-	1	-	1	-	-	-	-	-	2	-	-	-	-	-
	%	-	25	-	25	-	-	-	-	-	50	-	-	-	-	-
Urban n=22		1	1	1	4	2	2	2	1	1	2	1	1	1	1	1
	%	4.5	4.5	4.5	18.2	9.1	9.1	9.1	4.5	4.5	9.1	4.5	4.5	4.5	4.5	4.5

When looking at results for children younger than two years who were not breastfeeding at the time of the study (Table 4.3), ten (90,9%) rural children and 12 (37,5%) urban children had been previously breastfed. Compared to rural children, a significantly higher percentage of urban children were not previously breastfed (Table 4.3).

Table 4.3: Previous breastfeeding

	Rural (n=11)		***Urban (n=32)***		***95% CI on median difference***
Status	**N**	**%**	**N**	**%**	
Previously breastfed	10	90.9	12	37.5	[20.0; 69.8]*
Not breastfed	1	9.1	20	62.5	

The shortest period that a child was previously breastfed (either exclusively or not) was three weeks in rural participants and two weeks in urban participants (Table 4.4). The longest period that a child was previously breastfed was 54 weeks (1 year and 2 weeks) for rural participants and 76 weeks (1,5 years) for urban participants. The median period of breastfeeding for rural participants was 16 weeks and for urban participants 18 weeks.

Table 4.4: Period (in weeks) of previous breastfeeding

	Weeks	2	3	4	8	12	16	20	24	28	32	52	54	56	76
Rural n=10			1	1	1	1	1	1		1		1	1		
	%		10	10	10	10	10	10		10		10	10		
Urban n=12		1		2	1	2			2		1	1		1	1
	%	8.3		16.7	8.3	16.7			16.7		8.3	8.3		8.3	8.3

When looking at data related to exclusive breastfeeding (both for children previously breastfed and children breastfeeding at the time of the study (Table 4.5), the shortest period that a child was exclusively breastfed was three weeks in rural participants and two weeks in urban participants. The longest period was 32 weeks (8 months) for both rural and urban participants.

Table 4.5: Period (in weeks) of exclusive breastfeeding (currently and previously breastfed)

	Weeks	2	3	4	8	10	11	12	16	20	24	32
Rural n=15			2	2	2	1		4		1	1	1
	%		13.3	13.3	13.3	6.7		26.7		6.7	6.7	6.7
Urban n=34		1		3	4		1	9	2	1	10	3
	%	2.9		8.8	11.8		2.9	26.5	5.9	2.9	29.4	8.8

The median period of exclusive breastfeeding for rural participants was 11 weeks and for urban participants 12 weeks. About a quarter (26,5%) of urban participants were exclusively breastfed to 12 weeks, and about another third (29,4%) were exclusively breastfed for 24 weeks. In comparison, only

28,6% of rural participants were exclusively breastfed for 12 weeks and only 7,1% were exclusively breastfed for 24 weeks.

4.1.1.2 Formula feeding

When comparing the incidence of bottle- and breastfeeding at the time of the study (Table 4.6), it was found that about a third (31,3%) of the rural participants were predominantly bottle fed and another third (31,3%) were breast fed (either exclusively or not). The remainder (37,5%) of rural participants were neither bottle fed nor breast fed, probably since they were already eating complementary solid foods. No mixed feeding (bottle and breast) was reported in rural children. However, among urban participants, 16,7% of participants received mixed feeding. Another 20,4% were neither receiving breast, nor bottle feeds. Twenty-four percent (24,1%) of the urban participants were breastfeeding and 38,9% were bottle feeding.

Table 4.6: Predominant method of feeding

	Rural (n=16)		*Urban (n=54)*	
Method of feeding	**N**	**%**	**N**	**%**
Only breastfeeding	5	31.3	13	24.1
Only bottle feeding	5	31.3	21	38.9
Both	-	-	9	16.7
Neither	6	37.5	11	20.4

In Table 4.7, data for only two children in the rural group and thirty urban children younger than two years old were available. For urban participants who were formula fed, the feeding regimens of about two-thirds (63,3%) fell within recommendations (Table 4.7).

Table 4.7: Does child's formula feeding regimen fall within recommendations?

	Rural (n=2)		*Urban (n=30)*		***95% CI on median difference***
Status	**N**	**%**	**N**	**%**	
Within recommendations	-	-	19	63.3	
Not within recommendations	2	100	11	37.7	[-4.8; 78.1]

The rest of both the urban and rural participants' formula feeding regimens did not fall within recommendations, in other words, formula was either incorrectly prepared or incorrect amounts of feeds were given per day. Of all urban participants, one-third (30%) received only three bottles per day (data not shown). The median amount of time that one tin (400 g) of formula lasted among urban participants was seven days (21,4%) (data not shown). There was no significant difference between the rural and urban participants as to whether their formula feeding regimen fell within recommendations.

As can be seen from Table 4.8, the formula milk most frequently used by rural carers was Nestlé Nan 1 ® (60%), while Nestlé Pelargon ® was most frequently used by urban carers (63,3%). However, it should be kept in mind that the rural data consisted of only five participants, which makes it difficult to draw accurate conclusions.

Table 4.8: Type of formula milk used by carer

	Rural (n=5)		*Urban (n=32)*	
Name of formula	**N**	***%***	**N**	***%***
Nestlé Pelargon ®	1	30	19	63.3
Nestlé Nan 1 ®	3	60	5	16.7
Nestlé Nan 2 ®	-	-	1	3.3
Cow's milk	1	20	-	-
Nestlé Lactogen ®	-	-	2	6.7
Aspen Infacare ®	-	-	2	6.7
Nestlé Nan 3 ®	-	-	1	3.3

More than half of urban carers (53,3%) indicated that they bought the children's formula milk themselves (Table 4.9). A further forty percent indicated that they received the milk from the clinic, presumably as part of the Nutrition Supplementation Programme. Only 6,7% of carers indicated both buying milk and receiving formula from the clinic. Unfortunately, rural data was too small to draw any conclusions.

Table 4.9: Where does the carer get the formula?

	Rural		*Urban (n=30)*	
Location	**N**	***%***	**N**	***%***
Buys herself	1	100	16	53.3
Clinic	-	-	12	40
Both	-	-	2	6.7

4.1.1.3 Solids

When looking at Table 4.10, it can be seen that the earliest age at which solids were introduced was eight weeks for both rural and urban participants.

Table 4.10: Age (in weeks) at introduction of solids

	Rural (n=16)		*Urban (n=54)*		*95% CI on median difference*
Weeks	**N**	***%***	**N**	***%***	
0	-	-	12	22.2	
2	-	-	1	1.9	
8	1	6.7	3	5.6	[-10.2; 23.1]
12	1	6.7	10	18.5	[-25.6; 11.3]
16	-	-	5	9.3	
19	-	-	1	1.9	
20	4	26.7	2	3.7	[4.0; 45.9]
24	4	26.7	12	22.2	[-16.7; 28.9]
28	1	6.7	2	3.7	[-7.7; 24.8]
32	2	13.3	5	9.3	[-10.7; 27.3]
36	2	13.3	1	1.9	[-1.3; 34.2]

Solids were introduced fairly later among rural participants, with more than a quarter (26,7%) only receiving solids around twenty weeks (5 months) and another quarter (26,7%) only at 24 weeks (6 months). In comparison, about one-fifth (18,5%) of urban participants had already been introduced to solid foods by 12 weeks (3 months), with about another quarter (22,2%) at 24 weeks (6 months). However, almost another quarter (22,2%) of urban participants had not been introduced to solids yet at the time that the study was undertaken. The latest that solids were introduced in both rural and urban participants were 36 weeks (9 months). The median age of introduction to solids was 24 weeks (6

months) for rural participants and 19 weeks (4 months) for urban participants. Numbers of children in each group are very small, and thus results are not representative of all children in those communities.

4.1.2 Reported usual intake of different food groups

Reported usual intakes of food groups for both rural and urban children and adults were obtained from the results of the 24-hour recall of reported usual intake. Fourteen food groups were included, i.e.: alcohol; bread and cereals; fats and oils; fruit containing β-carotene; fruit containing vitamin C; other fruit (rich in vitamins other than β-carotene or vitamin C); legumes; meat and meat products; milk and milk products; soy; sweets/sugar; vegetable B (starchy vegetables; vegetables containing β-carotene; and vegetables containing vitamin C.

4.1.2.1 Children younger than two years

Of all children younger than two years of age, ten rural (4 male and 6 female) and 34 urban (16 male, 18 female) consumed solids. Since a 24 hour recall was completed for all children younger than two who also consumed solids, results do not only reflect dietary intake from six months of age onwards, but from the earliest age the participants consumed solids (i.e. 8 weeks in rural and 0 weeks in urban). No significant differences were found between rural and urban boys or rural and urban girls.

Guidelines for dietary intake of children younger than two years set out by LeLeiko et al. (1995:219, Tables 13.4 and 13.5) were used as set out in Tables 3.9 and 3.10 in Chapter 3. The recommended daily servings used for children between eight and 12 months were: 2½ to three cups of iron-fortified infant formula; sixty to 120 g of meat, two servings of ⅛ cup of vegetables (of which one should be a rich source of vitamin A); three servings of ⅛ cups of fruit (of which at least one should be a good source of vitamin C); four servings of bread, cereals or starch (½ slice bread or ¼ cup cereal); two teaspoons of fat; and two teaspoons of sugar. For children between one and two years of age, the recommended daily servings were: two cups of iron-fortified infant formula; 120 to 150 g of meat; two servings of ⅛ cup of vegetables (of which one should be a rich source of vitamin A); three servings of ⅛ cups of fruit (of which at least one should be a good source of vitamin C); four servings of bread, cereals or starch (½ slice bread or ¼ cup cereal); one teaspoon of fat; and two teaspoons of sugar (Table 3.10).

Most boys from both the rural- and urban areas consumed within their daily recommended servings of fats and oils; milk and milk products; and sweets and sugar (Table 4.11). The largest percentage of urban boys (68,75%) exceeded their recommended daily servings of bread and cereals, whereas a hundred percent of rural boys were within daily recommendations. More rural boys (75%) than urban boys (37,5%) consumed adequate daily amounts of milk and milk products; whereas more urban boys (31,25%) than rural boys (25%) exceeded the recommended daily servings and another 31,25% of urban boys did not eat enough milk and milk products.

As far as fruits and vegetables were concerned, 62,5% of urban boys consumed enough “other fruit,” which excluded fruits rich in vitamin C and β-carotene, whereas 75% of rural boys consumed enough daily servings of starchy vegetables (vegetable B). The largest number of the remaining boys from

both areas did not consume enough of any of the other fruit (containing β-carotene or vitamin C) or vegetables (containing β-carotene or vitamin C).

The largest percentage of both rural- and urban boys also consumed less than the recommended daily servings of: legumes; meat and meat products; and soy.

Table 4.11: Reported usual intake of different food groups for rural- and urban boys (<2 years old)

Rural (n=4)							*Urban (n=16)*							*P-value*
Food group	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	**Food group**	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	
Alcohol	0	0.0	4	100.0	0	0.0	Alcohol	0	0.0	16	100.0	0	0.0	
Bread and cereals	0	0.0	4	100.0	0	0.0	Bread and cereals	1	6.25	4	25.0	11	68.75	
Fats and oils	0	0.0	3	75.0	1	25.0	Fats and oils	0	0.0	15	93.75	1	6.25	
Fruit β carotene	4	100.0	0	0.0	0	0.0	Fruit β carotene	15	93.75	1	6.25	0	0.0	1.0
Fruit other	4	100.0	0	0.0	0	0.0	Fruit other	6	37.5	10	62.5	0	0.0	0.09
Fruit vitamin C	4	100.0	0	0.0	0	0.0	Fruit vitamin C	15	93.75	1	6.25	0	0.0	1.0
Legumes	4	100.0	0	0.0	0	0.0	Legumes	15	93.75	1	6.25	0	0.0	1.0
Meat and meat products	3	75.0	0	0	1	25.0	Meat and meat products	12	75.0	2	12.5	2	12.5	1.0
Milk and milk products	0	0	3	75.0	1	25.0	Milk and milk products	5	31.25	6	37.5	5	31.25	0.28
Soy	4	100.0	0	0.0	0	0.0	Soy	14	87.5	2	12.5	0	0.0	1.0
Sweets / sugar	0	0.0	4	100.0	0	0.0	Sweets / sugar	0	0.0	16	100.0	0	0.0	
Vegetable B	1	25.0	3	75.0	0	0.0	Vegetable B	11	68.75	5	31.25	0	0.0	0.5
Vegetable β carotene	4	100.0	0	0.0	0	0.0	Vegetable β carotene	11	68.75	5	31.25	0	0.0	1.0
Vegetable vitamin C	4	100.0	0	0.0	0	0.0	Vegetable vitamin C	12	75.0	3	18.75	1	6.25	0.25

Half (50%) of urban girls exceeded their intake of daily recommended servings for bread and cereals and half of rural girls consumed too many servings of sweets and sugar (Table 4.12).

The largest percentage of both rural- and urban girls consumed within daily recommended servings of fats and oils, whereas half of the rural girls and 94,4% of urban girls stayed within their daily recommended servings of sweets and sugar.

Table 4.12: Reported usual intake of different food groups for rural- and urban girls (<2 years old)

Rural (n=6)							*Urban (n=180*							*P-value*
Food group	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	**Food group**	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	
Alcohol	0	0.0	6	100.0	0	0.0	Alcohol	1	5.6	17	94.4	0	0.0	
Bread and cereals	4	66.7	2	33.3	0	0.0	Bread and cereals	3	16.7	6	33.3	9	50.0	
Fats and oils	0	0.0	6	100.0	0	0.0	Fats and oils	1	5.6	17	94.4	0	0.0	
Fruit β carotene	6	100.0	0	0.0	0	0.0	Fruit β carotene	15	83.3	3	16.7	0	0.0	0.55
Fruit other	3	50..0	3	50.0	0	0.0	Fruit other	13	72.2	4	22.2	1	5.6	0.3
Fruit vitamin C	5	83.3	1	16.7	0	0.0	Fruit vitamin C	12	66.7	6	33.3	0	0.0	0.62
Legumes	6	100.0	0	0.0	0	0.0	Legumes	12	66.7	6	33.3	0	0.0	0.27
Meat and meat products	3	50.0	2	33.3	1	16.7	Meat and meat products	11	61.1	7	38.9	0	0.0	1.0
Milk and milk products	5	83.3	0	0.0	1	16.7	Milk and milk products	9	50.0	6	33.3	3	16.7	0.28
Soy	6	100.0	0	0.0	0	0.0	Soy	12	66.7	6	33.3	0	0.0	0.27
Sweets / sugar	0	0.0	3	50.0	3	50.0	Sweets / sugar	1	5.6	17	94.4	0	0.0	
Vegetable B	2	33.3	4	66.7	0	0.0	Vegetable B	8	44.4	8	44.4	2	11.1	0.53
Vegetable β carotene	6	100.0	0	0.0	0	0.0	Vegetable β carotene	10	55.6	4	22.2	4	22.2	0.28
Vegetable vitamin C	6	100.0	0	0.0	0	0.0	Vegetable vitamin C	13	72.2	5	27.8	0	0.0	0.6

Two-thirds (66,7%) of rural girls and 44,4% of urban girls also consumed adequate amounts of starchy vegetables (vegetable B).

The largest percentage of both rural- and urban girls did not consume enough daily servings of all the fruit groups and remaining vegetable groups. They also did not consume enough legumes, meat and meat products, and soy.

These findings agree with the data in Tables 4.17 and 4.18, which shows that cooked porridge; sugar; full cream milk; tea and margarine/oil/fat mostly fell within the top ten of the most frequently consumed food items among these children.

4.1.2.2 Children older than two years

In the rural areas, there were 26 boys and 24 girls older than two years; and in the urban areas there were 64 boys and 48 girls older than two years. P values were calculated for the differences in the percentage of rural and urban children that had intakes that fell in the "within recommendation" ranges for the different food groups.

Recommended daily servings for children older than two years were used as set out in Table 3.1, i.e. two servings of milk or milk products; two servings of meat or meat products; six servings of bread or cereals; two servings of fruit of which one should be a good source of vitamin C; three servings of vegetables of which one should be a good source of β-carotene; four servings of fat or less; and four servings of sugar or less. Portion sizes of servings varied according to the child's age as set out by Table 3.10.

As with children younger than two years old, most of both rural- and urban boys older than two years ate little fruit, legumes, soy, vegetables, milk and milk products (Table 4.13). Most of both rural- and urban boys ate within the daily requirements of fats and oils. A significantly larger percentage of urban boys consumed fats and oils ($p=0.002$) and β-carotene rich vegetables ($p=0.003$) within the daily recommended number of servings than rural boys.

Urban boys exceeded their daily requirements for bread and cereals more often than the rural boys. A significantly larger percentage of rural boys consumed bread and cereals within daily recommendations ($p=0.001$) than rural boys. A significantly larger percentage of urban boys consumed sugar and sweets within recommendations ($p<0.0001$) than rural boys.

Half (50%) of the urban boys consumed more than the required number of six servings from the bread and cereals group, while a further 39,1% consumed the required six daily servings. In comparison, 76,9% of rural boys consumed bread and cereals within required amounts and 19,2% consumed less than the required six servings.

Intakes of fat and oil intake were mostly within requirements for both rural (57,7%) and urban (87,5%) groups, although 42,3% of the rural group also consumed amounts above requirements (more than four daily servings) compared to only 12,5% of urban boys.

Fruit consumption was mostly low, with only 11,5% and 23,1% of rural boys meeting their requirements for one vitamin C-rich fruit or one other fruit daily. In urban areas 15,6% of boys ate enough vitamin-C rich fruit, 40,6% consumed enough other fruit and 12,5% consumed more than the required serving of one other fruit.

Table 4.13: Reported usual intake of different food groups for rural- and urban boys (>2 years old)

Rural (n=26)							*Urban (n=64)*							*P value*
Food group	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	**Food group**	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	
Alcohol	0	0.00	26	100.0	0	0.00	Alcohol	0	0.0	64	100.0	0	0.0	
Bread and cereals	5	19.2	20	76.9	1	3.9	Bread and cereals	7	10.9	25	39.1	32	50.0	0.001*
Fats and oils	0	0.0	15	57.7	11	42.3	Fats and oils	0	0.0	56	87.5	8	12.5	0.002*
Fruit β carotene	26	100.0	0	0.0	0	0.0	Fruit β carotene	62	96.9	1	1.6	1	1.6	
Fruit other	20	76.9	6	23.1	0	0.0	Fruit other	30	46.9	26	40.6	8	12.5	
Fruit vitamin C	23	88.5	3	11.5	0	0.0	Fruit vitamin C	53	82.8	10	15.6	1	1.6	
Legumes	26	100.0	0	0.0	0	0.0	Legumes	60	93.8	4	6.3	0	0.0	
Meat and meat products	5	19.2	8	30.8	13	50.0	Meat and meat products	38	59.4	13	20.3	13	20.3	0.3
Milk and milk products	16	61.5	8	30.8	2	7.7	Milk and milk products	33	51.6	12	18.8	19	29.7	
Soy	26	100.0	0	0.0	0	0.0	Soy	59	92.2	4	6.3	1	1.6	
Sweets / sugar	0	0.0	13	50.0	13	50.0	Sweets / sugar	0	0.0	57	89.1	7	10.9	<0.0001*
Vegetable B	17	65.4	9	34.6	0	0.0	Vegetable B	39	60.9	24	37.5	1	1.6	
Vegetable β carotene	25	96.2	1	3.9	0	0.0	Vegetable β carotene	37	57.8	22	34.4	5	7.8	0.003*
Vegetable vitamin C	21	80.8	5	19.2	0	0.0	Vegetable vitamin C	47	73.4	11	17.2	6	9.4	

Intake of legumes was mostly low. Rural boys tended to eat more meat and meat products, with fifty percent consuming more than the required two daily servings and a further 30,8% consuming within requirements of two daily servings. In comparison, 59,4% of urban boys did not meet their daily requirements for meat intake. However, 20,3% of urban boys ate adequate servings of meat and meat products and another 20,3% consumed more than the required two servings of meat and meat products per day.

Almost two-thirds (61,5%) of rural boys and half (51,6%) of urban boys did not consume the required number of two servings of milk and milk products per day. However, almost a third (29,7%) of urban boys consumed more than their required number of two servings per day. Soy intake was low, with only 6,3% of urban boys consuming enough soy and 1,6% eating more than the required one serving per day.

Half (50%) of the rural boys (n=13) ate four servings or less of sugar, while the other half consumed more than the recommendation of four or less servings per day. Urban boys stayed more within their requirements (89,1%), with only 10,9% exceeding their requirements for sugar of four servings or less.

It seems that as a whole, urban boys consumed comparatively more fruit and vegetables than rural boys, although the majority still did not meet their daily requirements. Almost two-thirds of both rural (65,4%) and urban (60,9%) boys did not consume enough starchy vegetables (Vegetable B). However, the other one-third of both rural (34,6%) and urban (37,5%) boys did meet their requirements of one serving per day. This was however not the case for beta-carotene-rich vegetables. Most rural boys (96,2%) did not eat one serving a day, with only 3,9% meeting their daily requirement of one serving. The urban picture looks a little different, with only over half (57,8%) of the boys not meeting their daily requirements of one serving, a third (34,4%) meeting their requirements and a further 7,8% exceeding their requirements. Vitamin-C rich vegetable intake was about the same among both groups with about three-quarters of both rural (80,8%) and urban (73,4)% boys eating less than one daily serving and only 19,2% of rural and 17,2% of urban boys eating one daily serving. A further ten percent of urban boys consumed more than one daily serving.

As expected, none of the boys consumed alcohol.

Table 4.14: Reported usual intake of different food groups for rural and urban girls (>2 years old)

Rural (n=24)							*Urban (n=48)*							*P-value*
Food group	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	**Food group**	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	
Alcohol	1	4.2	23	95.8	0	0.0	Alcohol	1	2.1	47	97.9	0	0.0	
Bread and cereals	5	20.8	17	70.8	2	8.3	Bread and cereals	12	25.0	10	20.8	26	54.2	<0.0001*
Fats and oils	1	4.2	16	66.7	7	29.2	Fats and oils	2	4.2	41	85.4	5	10.4	
Fruit β carotene	24	100.0	0	0.0	0	0.0	Fruit β carotene	44	91.7	3	6.3	1	2.1	
Fruit other	18	75.0	6	25.0	0	0.0	Fruit other	19	39.6	24	50.0	5	10.4	0.04*
Fruit vitamin C	20	83.3	4	16.7	0	0.0	Fruit vitamin C	37	77.1	10	20.8	1	2.1	
Legumes	24	100.0	0	0.0	0	0.0	Legumes	41	85.4	7	14.6	0	0.0	
Meat and meat products	10	41.7	8	33.3	6	25.0	Meat and meat products	25	52.1	15	31.3	8	16.7	
Milk and milk products	14	58.3	7	29.2	3	12.5	Milk and milk products	25	52.1	10	20.8	13	27.1	
Soy	24	100.0	0	0.0	0	0.0	Soy	40	83.3	7	14.6	1	2.1	
Sweets / sugar	1	4.2	15	62.5	8	33.3	Sweets / sugar	1	2.1	44	91.7	3	6.3	0.002*
Vegetable B	14	58.3	10	41.7	0	0.0	Vegetable B	28	58.3	17	35.4	3	6.3	
Vegetable β carotene	24	100.0	0	0.0	0	0.0	Vegetable β carotene	28	58.3	13	27.1	7	14.6	0.003*
Vegetable vitamin C	18	75.0	6	25.0	0	0.0	Vegetable vitamin C	39	81.3	9	18.8	0	0.0	

Urban girls exceeded their daily requirements for bread and cereals more often than the rural girls. A significantly larger percentage of rural girls consumed bread and cereals within recommendations ($p<0.0001$) than urban girls. On the other hand, a significantly larger percentage of urban girls consumed sweets and sugar ($p=0.002$) and β-carotene rich vegetables ($p=0.003$) within recommendations than rural girls.

Overall, both rural- and urban girls consumed inadequate amounts of legumes, soy, vegetables, meat and meat products, and milk and milk products (Table 4.14). Urban girls consumed more other fruit than rural girls, but intake of vitamin C-rich or ß-carotene–rich fruit remained low among both rural- and urban girls. About half (54,2%) of urban girls consumed breads and cereals in excess, whereas seventy percent of rural girls stayed within their requirements of six servings a day Sugar and sweets intake as well as intake of fats and oils were mostly within limits among both the rural- and urban girls, but about a third of rural girls also exceeding their daily limits of four servings of sugar, sweets, fats and oils in comparison with a very small amount of urban girls.

More than half (54,2%) of the urban girls consumed above their requirements of six daily servings of bread and cereals as compared to only 8,3% of rural girls. About three-quarters (70,8%) of rural girls did, however, meet their daily requirements, with only 20,8% consuming too little. In comparison, a quarter of urban girls (25%) did not meet their requirements of six daily servings, but a further 20,8% did.

Consumption of fats and oils were sufficient (four daily servings) in two-thirds (66,7%) of rural girls and 85,4% of urban girls, with 29,2% of rural girls and 10,4% of urban girls exceeding their daily requirements of four servings.

Fruit intake was low, with only 16,7% of rural girls eating enough (one daily serving) of vitamin C-rich fruit and 25% eating enough (one daily serving) of other fruit. Fruit intake was higher in urban girls, with 20,8% consuming enough vitamin C-rich fruit and fifty percent eating enough other fruit. A further 10,4% of urban girls ate more than the required one daily serving of other fruit. All the rural girls

(100%) and almost all the urban girls (91,7%) did not meet their daily requirement of one serving of beta-carotene-rich fruit per day.

Legume intake was low for all rural girls and most urban girls (83,3%), but 14,6% of urban girls did reach their intake goal of one daily serving.

About half of both rural (41.2%) and urban girls (52,1%) did not eat sufficient daily servings from the meat and meat products group. However, about one-third of both rural (33.3%) and urban (31.3%) girls did meet their daily requirements of two servings and a further quarter (25%) of rural girls and 16,7% of urban girls ate more meat and meat products than required.

More than half of both the rural (58,3%) and the urban (52,1%) girls consumed too little dairy (less than two servings per day). About a quarter of the rural girls (29,2%) consumed adequate amounts compared to 20,8% of urban girls. In comparison, more than a quarter of urban girls (27,1%) consumed more than two daily servings compared with only 12,5% of rural girls. As was found amongst the boys, all the rural girls consumed too little soy (less than one daily serving), whereas 14,6% of urban girls consumed adequate amounts of soy and 2,1% consumed above their required daily amounts.

Almost two-thirds (62,5%) of the rural girls consumed within their daily requirements of four servings of sugar and sweets, and a further third (33,3%) consumed above this daily requirement. In comparison, almost all the urban girls (91,7%) did not eat more than four servings of sugar and sweets per day. Only 6,3% of urban girls consumed too much sugar and sweets.

More than half of both rural (58,3%) and urban (58,3%) girls consumed less than one serving of starchy vegetables (vegetable B). Another 41,7% of rural girls and 35,4% of urban girls consumed one serving of starchy vegetables per day and a further 6,25% of urban girls consumed more than one daily serving. All of the rural girls did not eat at least one serving of beta-carotene-rich vegetable per day, whereas only about half (58,3%) of urban girls did not. Another 27,1% of urban girls met their requirements and 14,6% ate more than one daily serving of beta-carotene-rich vegetables. Three-quarters (75%) of rural girls did not eat enough vitamin C-rich vegetables, and a quarter (25%) ate enough (one daily serving). In comparison, the majority of urban girls (81,3%) did not eat one daily serving, and only 18,8% did.

Only 14 urban children (12%) (N=117), consumed the recommended five portions of fruit and vegetables per day (data not shown).

4.1.2.3 Adults

Of the 549 rural participants, 160 were men and 389 women. Of the 419 urban participants, 99 were men and 320 were women. P values were calculated for the differences in the percentage of rural and urban adults that had intakes that fell in the “within recommendation” ranges for the different food groups.

The daily recommended servings guidelines were: two to three servings of milk or milk products; two to three servings of meat or meat products; six to eleven servings of bread or cereals; two to four

servings of fruit, of which one should be vitamin C-rich and one β-carotene rich; three to five servings of vegetables, of which one should be vitamin C-rich and one β-carotene rich; one weekly serving of legumes; four servings or less of fat/oil; and four servings or less of sugar/sweets. The alcohol cut-offs were two servings per day for men and one serving per day for women (USDA & USDHHS, 25010:13).

Most rural- and urban men ate less than their daily requirements of fruit; legumes; milk and milk products; soy; and vegetables (Table 4.15). Most rural men (54,4%) and 42,4% ofurban men ate too many servings of bread and cereals (more than 11 daily servings); meat and meat products (more than three daily servings). A significantly larger percentage of rural men consumed the required amount of milk and milk products than urban participants (p<0.0002), while a significantly larger percentage of urban men consumed soy within the daily recommendations (p=0.01).

Table 4.15: Reported usual intake of different food groups for rural and urban men

Rural							*Urban*							*P-value**
n=160	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	**n=99**	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	
Alcohol	0	0.0	160	100.0	0	0.0	Alcohol	0	0.0	94	95.0	5	5.1	0.004*
Bread and cereals	1	0.6	35	21.9	124	77.5	Bread and cereals	11	11.1	23	23.2	65	65.7	
Fats and oils	0	0.0	71	44.4	89	55.6	Fats and oils	0	0.0	76	76.8	23	23.2	<0.0001*
Fruit β carotene	160	100.0	0	0.0	0	0.0	Fruit β carotene	98	99.0	0	0.0	1	1.0	
Fruit other	135	84.4	25	15.6	0	0.0	Fruit other	77	77.8	19	19.2	3	3.0	
Fruit vitamin C	153	95.6	7	4.4	0	0.0	Fruit vitamin C	88	88.9	5	5.1	6	6.1	
Legumes	160	100.0	0	0.0	0	0.0	Legumes	96	97.0	3	3.0	0	0.0	0.03*
Meat and meat products	32	20.0	39	24.4	89	55.6	Meat and meat products	42	42.4	15	15.2	42	42.4	
Milk and milk products	101	63.1	56	35.0	3	1.9	Milk and milk products	84	84.9	14	14.1	1	1.0	<0.0002*
Soy	160	100.0	0	0.0	0	0.0	Soy	90	90.9	4	4.0	5	5.1	0.01*
Sweets / sugar	0	0.0	73	45.6	87	54.4	Sweets / sugar	0	0.0	66	66.7	33	33.3	0.001*
Vegetable B	119	74.4	41	25.6	0	0.0	Vegetable B	59	59.6	26	26.3	14	14.1	
Vegetable β carotene	156	97.5	4	2.5	0	0.0	Vegetable β carotene	68	68.7	10	10.1	21	21.2	0.009*
Vegetable vitamin C	127	79.4	33	20.6	0	0.0	Vegetable vitamin C	72	72.7	14	14.1	13	13.1	

*Chi square / Fischer exact

Rural men also exceeded their daily requirements for fats and oils, as well as sweets and sugar, more often than the urban men. A significantly larger percentage of urban men consumed fats and oils within daily recommendations (p<0.0001) and also consumed sugar and sweets within recommendations (p=0.001) than rural men.

All rural men used alcohol within limits. However, 5% of urban men exceeded their daily recommendation of two drinks per day. Compared to urban men, a significantly larger percentage of rural men consumed alcohol within the daily recommendations (p=0.004).

The majority of participants of both groups ate more bread and cereals than their daily requirements of six to 11 servings (77,5% of the rural participants and 65,7% of urban participants). In comparison, only 11,1% of urban participants and 0,63% of rural participants did not meet their daily requirements for bread and cereals.More than half (55,6%) of the rural participants consumed above their daily requirements of four servings of fats and oils, as compared to only about a quarter (23,2%) of urban participants. Most of the other urban participants (75,8%) consumed amounts of fats and oils within their daily limits, as opposed to only 44,4% of rural participants.

Most participants of both groups did not consume enough fruit (three daily servings). Only 15,6% of rural and 19.2% of urban participants consumed enough (one daily serving) from the “other fruit” category, and only 4,4% of rural and 5,1% of urban participants consumed enough (one daily serving) vitamin C-rich fruit.

Consumption of legumes was also low, with only 3% of urban participants eating the recommended servings (one weekly serving) of legumes. A significantly larger percentage of urban men consumed legumes within their daily recommendations (p=0.03) than rural men.

About half (55,6%) of rural and 42,4% of urban participants ate more than the required two to three daily servings of meat and meat products than required. In contrast, another twenty percent of rural- and 42% of urban participants did not eat enough meat and meat products daily. Only 24,4% of rural- and 15,2% of urban participants met their daily recommendations of two to three daily servings of meat and meat products.

About two-thirds (63,1%) of rural men and 84,9% of urban men did not consume the required two to three daily servings of dairy products. All rural men did not consume a daily serving of soy, but 4% of urban men consumed adequate amounts and 5,1% of urban men consumed amounts above daily requirements.

More than half of the rural men (54.4%) and one-third (33,3%) of the urban men consumed more than four servings of sugar daily.

Vegetable consumption was low for most rural- and urban men, with only a quarter of both the rural- (25,6%) and urban men (26,3%) eating one daily serving of starchy vegetables (Vegetable B). Among urban men, vegetable consumption was above one daily serving in 14% of participants for starchy vegetables, 21,2% for ß-carotene rich vegetables and 13,1% for vitamin C-rich vegetables. None of the rural men ate more than three to five daily servings of vegetables. Compared to rural men, a significantly larger percentage of urban men consumed β-carotene rich vegetables within daily recommendations (p=0.009).

The majority of rural- and urban women consumed inadequate amounts of fruit; legumes; milk and milk products; soya; and vegetables (Table 4.16). Most rural- and urban women consumed excessive servings of bread and cereals; and meat and meat products. Urban women mostly consumed within their limits of fats and oils and sugar and sweets, but most rural women exceeded their intake of these foods. Compared to rural women, a significantly larger percentage of urban women consumed sweets and sugar within daily recommendations (p<0.0001).

Alcohol intake was mostly within limits for both rural (100%) and urban women (99,4%). Intake of bread and cereals was above six to eleven daily servings for more than half of both the rural (57,1%) and urban women (52,5%). Only 2,6% of rural women and 8,8% of urban women did not meet their daily requirements. A higher percentage of rural women (52,1%) exceeded their intake of four daily servings of fats and oils compared to urban women (29,6%). Compared with rural women, a significantly larger percentage of urban women consumed within their daily recommendations of fats and oils (p<0.0001).

Table 4.16: Reported usual intake of different food groups for rural and urban women

Rural							*Urban*							*P-value**
n=389	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	**n=320**	**Below**	**% of total**	**Within**	**% of total**	**Above**	**% of total**	
Alcohol	0	0.0	389	100.0	0	0.0	Alcohol	0	0.0	318	99.4	2	0.6	
Bread and cereals	10	2.6	157	40.4	222	57.1	Bread and cereals	28	8.8	124	38.8	168	52.5	
Fats and oils	0	0.0	184	47.3	205	52.7	Fats and oils	2	0.6	223	69.7	95	29.7	<0.0001*
Fruit β carotene	388	99.7	1	0.3	0	0.0	Fruit β carotene	313	97.8	2	0.6	5	1.6	
Fruit other	295	75.8	94	24.2	0	0.0	Fruit other	228	71.3	78	24.4	14	4.4	
Fruit vitamin C	362	93.1	26	6.7	1	0.3	Fruit vitamin C	277	86.6	10	3.1	33	10.3	0.03*
Legumes	384	98.7	5	1.3	0	0.0	Legumes	305	95.3	9	2.8	6	1.9	
Meat and meat products	71	18.3	122	31.4	196	50.4	Meat and meat products	114	35.6	83	25.9	123	38.4	
Milk and milk products	265	68.1	119	30.6	5	1.3	Milk and milk products	275	85.9	36	11.3	9	2.8	<0.0001*
Soy	389	100.0	0	0.0	0	0.0	Soy	295	92.2	11	3.4	14	4.4	<0.0001*
Sweets / sugar	0	0.0	175	45.0	214	55.0	Sweets / sugar	2	0.6	214	66.9	104	32.5	<0.0001*
Vegetable B	235	60.4	154	39.6	0	0.0	Vegetable B	157	49.1	126	39.4	37	11.6	
Vegetable β carotene	371	95.4	18	4.6	0	0.0	Vegetable β carotene	176	55.0	54	16.9	90	28.1	<0.0001*
Vegetable vitamin C	261	67.1	128	32.9	0	0.0	Vegetable vitamin C	216	67.5	48	15.0	56	17.5	<0.0001*

*Chi square/Fischer exact

As found in the other groups discussed previously, fruit intake was low, with only about a quarter of both rural (24,2%) and urban (24,4%) women consuming one daily serving of “other fruit.” A significantly larger percentage of rural women consumed vitamin C rich fruit within daily recommendations (p=0.03) than urban women.

Legumes intake was mostly low, with only 2,8% of urban women eating enough legumes and a further 1,9% exceeding the recommended one weekly serving. About half (50,4%) of rural women and a third (38,4%) of urban women ate more than two to three daily servings of meat- and meat products. In comparison, 18,3% of rural women and a third (35,6%) of urban women did not consume enough meat- and meat products.

Results for dairy products are comparable to the men’s intakes, with two-thirds (68,1%) of rural women and 85,9% of urban women not consuming two to three daily servings of milk- and milk products. Only 1,3% of rural and 2,8% of urban women exceeded their daily requirements. Compared to urban women, a significantly larger percentage of rural women consumed milk and milk products within daily recommendations (p<0.0001).

All rural women ate less than one serving of soy. In contrast, 3,4% of urban women met and 4,4% of urban women exceeded their daily requirements for soy. A significantly larger percentage of urban women consumed soy within daily recommended servings (p<0.0001), compared to rural women.

More than half (55%) of rural women and a third (32,5%) of urban women used more than four daily servings of sugar/sweets. More than a third of both rural- (39,6%) and urban (39,4%) women consumed one daily serving of starchy vegetables (vegetable B) with a further 11,6% of urban women exceeding the daily requirement. Intake of one daily serving of ß-carotene-rich vegetables was comparatively low in rural women, whereas 16,9% of urban consumed adequate amounts and another 28,1% consumed more than one daily serving. A significantly larger percentage of urban women consumed β-carotene-rich vegetables within daily recommendations than rural women (p<0.0001).

Only one-third (32,9%) of rural women consumed one daily serving of vitamin C-rich vegetables as opposed to 15% urban women meeting and another 17,5% exceeding their daily requirements for vegetables from the vitamin C-rich group. Compared to urban women, a significantly larger percentage of rural women consumed vitamin C-rich vegetables within daily recommendations ($p<0.0001$).

These findings agree with data in Tables 4.21 and 4.22, which shows that cooked porridge; sugar; full cream milk; tea; bread; and margarine/oil/fat mostly fell within the top ten of the most frequently consumed food items among these adults.

4.1.3 Reported usual intake of different food items

Reported usual intake of different food items were obtained by means of a food frequency questionnaire. There were 26 different food items ranked in order of frequency consumed per month, with the most frequently consumed item ranked as first. Results were calculated for three groups, namely: children younger than two years; children older than two years; and adults (25 to 64 years old).

4.1.3.1 Children younger than two years

Reported usual intake of different food items were calculated for ten rural children (4 boys and 6 girls) and 34 rural children (16 boys and 18 girls) in the group younger than two years old. Since the sample sizes were small, caution should be taken when interpreting the results as set out in Table 4.17 and Table 4.18. Median amounts instead of means were used, because data was not always distributed normally. No confidence intervals for differences between urban and rural children were calculated, since the sample sizes were too small.

Tea, cooked porridge, margarine/oil, salt/stock, bread, fruit, and vegetables were all ranked in the top ten items consumed by both rural and urban boys younger than two years old. Rural boys consumed full cream milk more often than boys, whereas urban boys consumed more fruit than rural boys. Rural boys also consumed coffee creamer and cakes/biscuits more often than urban boys.

Cooked porridge, tea, margarine/oil, salt/stock, vegetables, bread, and fruit were within the top ten items most frequently consumed by both rural and urban girls younger than two years old. Rural girls consumed fruit and chips/crisps more often than urban girls, whereas urban girls consumed full cream milk more often than rural girls.

Table 4.17: Frequency of intake of different food items for rural and urban boys (times per month) (<2 years)

Rank	*Rural (n=4)*				Rank	*Urban (n=16)*			
	Food item	**Mean**	**Median**	**Std Dev**		***Food intake***	**Mean**	**Median**	**Std Dev**
1	Sugar	37.5	30	37.8	**3**	Sugar	32	30	19.6
2	Tea	30	30	24.5	**1**	Tea	36.9	30	48.5
3	Full cream milk	22.5	30	15	**8**	Full cream milk	20.8	30	17.2
4	Porridge, cooked	22.5	30	15	**2**	Porridge. Cooked	35	30	25
5	Margarine/oil/fat	22.5	30	15	**5**	Margarine/oil/fat	24	30	15.6
6	Salt/stock	22.5	30	15	**6**	Salt/stock	23.4	30	15.6
7	Bread	24.5	19	26.9	**7**	Bread	21	30	12.4
8	Fruit	24.5	19	26.9	**4**	Fruit	24.4	30	20.9
9	Vegetables	17	19	15.4	**10**	Vegetables	16.8	12	12
10	Cool drinks	16	17	16.3	**12**	Cool drinks	14	8	13.8
11	Coffee creamer	30	15	42.4	**23**	Coffee creamer	3.8	0	10.2
12	Cake/biscuits	22.5	15	28.7	**18**	Cake/biscuits	8.2	4	9.6
13	Fruit juice	15	15	17.3	**17**	Fruit juice	12.9	6	16.5
14	Sweets/chocolates	12.5	10	12.7	**16**	Sweets/chocolates	7.9	8	7.3
15	Samp/mealie rice	10.5	6	13.4	**21**	Samp/mealie rice	2.8	4	2.8
16	Soy mince/legumes	6	6	5.2	**19**	Soy/Legumes	5.1	4	7.5
17	Fish	6	6	5.2	**22**	Fish	2.5	3.5	2.4
18	Cereals	6	6	6.9	**14**	Cereal	13.6	8	13.6
19	Chips/crisps	9.75	4.5	14	**11**	Chips/crisps	12.3	10	11.4
20	Chicken	5	4	5	**15**	Chicken	11.3	8	8.5
21	Red meat	5	4	6	**20**	Red meat	4.4	4	3.8
22	Peanut butter/peanuts	4	4	4.6	**13**	Peanut butter	14	8	13.8
23	Eggs	3	2	3.8	**9**	Eggs	16.8	14	11.6
24	Low fat/skim milk	7.5	0	15	**24**	Low fat/skim milk	0.5	0	2
25	Coffee	0	0	0	**25**	Coffee	0.3	0	1
26	Alcohol	0	0	0	**26**	Alcohol	0	0	0

Table 4.18: Frequency of intake of different food items for rural and urban girls (times per month) (<2 years)

Rank	*Rural (n=6)*				Rank	*Urban (n=18)*			
	Food item	**Mean**	**Median**	**Std Dev**		***Food item///***	**Mean**	**Median**	**Std Dev**
1	Sugar	75	75	67.8	**18**	Sugar	17.1	0	25.4
2	Porridge, cooked	40	45	36.3	**1**	Porridge. Cooked	36.6	30	25.5
3	Fruit	21.3	19	23.4	**9**	Fruit	10.9	6	15.3
4	Chips/crisps	15.7	17	15.8	**15**	Chips/crisps	6.6	3	9.6
5	Tea	25	15	35.1	**2**	Tea	24.2	30	23.5
6	Bread	10.3	10	11.1	**7**	Bread	15.6	12	16.3
7	Vegetables	8	8	8	**5**	Vegetables	22	12	24.6
8	Margarine/oil/fat	22	6	35.3	**3**	Margarine/oil/fat	19.6	30	12.4
9	Salt/stock	22	6	35.3	**4**	Salt/stock	24.1	23	27.3
10	Chicken	6.7	4	9.4	**14**	Chicken	4.4	4	3.6
11	Samp/mealie rice	4.7	4	5.3	**22**	Samp/mealie rice	3.3	0	7.6
12	Peanut butter/peanuts	4.5	3.5	5.2	**21**	Peanut butter	3.3	0	4.6
13	Cool drinks	15.7	2	24.7	**13**	Cool drinks	6.4	4	9.5
14	Sweets/chocolate	6.3	2	11.8	**16**	Sweets/chocolates	7.5	2.5	14.9
15	Soy mince/legumes	3.3	2	4.7	**11**	Soy/Legumes	10.1	4	20.7
16	Fruit juice	6.8	1.5	11.8	**10**	Fruit juice	16.4	4	24.6
17	Red meat	3.5	0.5	5.2	**23**	Red meat	1.6	0	3
18	Full cream milk	27	0	60.5	**6**	Full cream milk	20.8	12	23.9
19	Cake/biscuits	10	0	15.5	**12**	Cake/biscuits	9.6	4	15.6
20	Eggs	3.3	0	6.4	**8**	Eggs	11.4	8	14.2
21	Fish	3.3	0	5.3	**17**	Fish	1.6	1	2.9
22	Coffee creamer	0	0	0	**24**	Coffee creamer	0.2	0	0.9
23	Coffee	0	0	0	**20**	Coffee	4.2	0	14.2
24	Low fat/skim milk	0	0	0	**25**	Low fat/skim milk	0	0	0
25	Cereal	0	0	0	**19**	Cereal	13	0	20.5
26	Alcohol	0	0	0	**26**	Alcohol	0	0	0

4.1.3.2 Children older than two years

Reported usual intakes of different food items were calculated for fifty rural children older than two years, of which 26 (52%) were boys and 24 (48%) were girls. Intakes were also calculated for 112 urban children, of which 64 (57,1%) were boys and 48 (42,9%) were girls. There were no significant differences between the intake of food items among either rural and urban boys or rural and urban girls, except for median intake of cake/biscuits among boys (CI [-30;-1].)

When describing the dietary intake according to foods most frequently consumed by rural and urban boys older than two years old (Table 4.19), it can be seen that rural and urban boys ate comparatively similar foods most frequently, with the exception of vegetables and cake/biscuits. Sugar; porridge; full cream milk; salt/stock; tea; fruit; margarine/oil/fat; bread and cool drinks all fell within the ten most frequently consumed items for both rural and urban boys. In the urban group, vegetables were more frequently consumed (9th ranking) than in the rural group (13th ranking). Rural boys used coffee creamer (17th ranking) more frequently than urban boys (24th ranking). Rural boys also ate cake/biscuits (3rd ranking) more frequently than urban boys (15th ranking).

Table 4.19: Frequency of intake of different food items for rural and urban boys (times per month) (>2 years)

Rank	*Rural (n=26)*				Rank	*Urban (n=64)*				95% CI for difference in medians
	Food item	Mean	Median	Std Dev		*Food item*	Mean	Median	Std Dev	
1	Sugar	59	60	33.1	**2**	Sugar	45.4	30	28.6	[-30; 0]
2	Porridge, cooked	50.1	45	27.3	**1**	Porridge, cooked	49	30	31.2	[-14; 0]
3	Cake/biscuits	39	30	38.5	**15**	Cake/biscuits	12.4	8	12.4	[-30; -1]*
4	Full cream milk	37.9	30	31.2	**5**	Full cream milk	31.5	30	31.7	[-26; 0]
5	Salt/ stock	37.7	30	24.9	**3**	Salt/ stock	36.3	30	23	[0; 0]
6	Tea	37.5	30	24.8	**4**	Tea	31.9	30	31.3	[-30; 0]
7	Fruit	35	30	34.7	**7**	Fruit	26.8	30	23.8	[-18; 0]
8	Margarine/ oil/ fat	28.1	30	25.9	**6**	Margarine/ oil/ fat	29.6	30	18.8	[0; 0]
9	Bread	26.2	30	18.7	**8**	Bread	23	30	17.7	[-8; 0]
10	Cool drinks	20.5	30	17.2	**10**	Cool drinks	21.2	16	25	[-4; 0]
11	Chips/crisps	19.4	21	17.6	**11**	Chips/crisps	16.7	12	12	[-4; 2]
12	Sweets/chocolates	17.6	12	19.1	**12**	Sweets/chocolates	14.2	8	12.8	[-4; 2]
13	Vegetables	12.9	8	10.3	**9**	Vegetables	23.3	27	19.1	[0; 18]
14	Chicken	11.5	8	9.9	**18**	Chicken	8.4	8	6.2	[-4; 0]
15	Eggs	9.6	8	9.7	**16**	Eggs	11.4	8	10.4	[-3; 4]
16	Cereal	8.2	8	8.4	**13**	Cereal	13.7	8	16.2	[0; 8]
17	Coffee creamer	24.6	4.5	30.4	**24**	Coffee creamer	4.5	0	14	[-30; 0]
18	Fruit juice	11.7	4	15.5	**14**	Fruit juice	13	8	19.3	[-2; 4]
19	Soy mince/ legumes	6.5	4	6.1	**19**	Soy mince/ legumes	6.8	4	7.1	[-4; 1]
20	Fish	5.3	4	6	**22**	Fish	3	3.5	3.3	[-4; 0]
21	Samp/ mealie rice	5	4	5.7	**21**	Samp/ mealie rice	4	4	3.7	[-1; 0]
22	Red meat	5	4	3.8	**20**	Red meat	4.4	4	5.3	[-4; 0]
23	Peanut butter/peanuts	6.7	2.5	9.5	**17**	Peanut butter/peanuts	10.8	8	12.8	[0; 7]
24	Low fat/skim milk	4.6	0	13.9	**25**	Low fat/skim milk	0.1	0	1	[0; 0]
25	Coffee	2.4	0	6.6	**23**	Coffee	6.6	0	19.4	[0; 0]
26	Alcohol	0.3	0	1.6	**26**	Alcohol	0.02	0	0.125	[0; 0]

When comparing frequent intake of food items for rural and urban girls older than two years older (Table 4.20), sugar; cooked porridge; full cream milk; salt or stock; tea; fruit; margarine/oil/fat; and bread fell in the top ten list for both rural and urban girls.

As with the boys, sugar and cooked porridge fell within the three most consumed items. Rural girls consumed full cream milk a little more often (3rd) than urban girls (5th) (the opposite was true among

girls younger than two years). As with boys, fruit consumption was frequent, ranking 10th among rural and 7th among urban girls. As found among rural boys, cake and biscuit intake also fell within the ten most frequently consumed foods by rural girls. Intake of cool drinks was also frequently reported.

As with boys, cake/biscuits were consumed more frequently by urban girls (11th) than rural girls (18th), although not significantly so. Chips/crisps were consumed more frequently by rural girls (8th rural vs. 13th urban) and vegetables were consumed more often by urban girls (9th) than rural girls (12th).

Table 4.20: Frequency of intake of different food items for rural and urban girls (times per month) (>2 years)

Rank	*Rural (n=24)*				Rank	*Urban (n=48)*				95% CI for difference in medians
	Food item	**Mean**	**Median**	**StdDev**		***Food item***	**Mean**	**Median**	**Std Dev**	
1	Sugar	60.8	60	47.5	**3**	Sugar	33.5	30	27.4	[-60; 0]
2	Porridge, cooked	46.6	60	30.1	**1**	Porridge, cooked	45.5	30	28.6	[-30; 8]
3	Full cream milk	42	30	47.6	**5**	Full cream milk	28.5	30	23.2	[-30; 8]
4	Salt/ stock	36.2	30	35	**2**	Salt/ stock	34	30	25.3	[-14; 18]
5	Tea	35.3	30	29.9	**6**	Tea	27.1	30	23.5	[-30; 0]
6	Margarine/ oil/ fat	26.8	30	24.6	**4**	Margarine/ oil/ fat	28.6	30	20.3	[0; 8]
7	Bread	26.4	30	22.8	**8**	Bread	22.7	30	16.1	[-10; 0]
8	Chips/crisps	21.8	30	21.5	**13**	Chips/crisps	15.5	12	13	[-10; 0]
9	Cool drinks	27.2	21	27.1	**14**	Cool drinks	16.7	8	25.7	[-26; 0]
10	Fruit	29.2	16	31	**7**	Fruit	25.1	30	19.6	[-8; 14]
11	Cake/biscuits	28.8	16	37.4	**18**	Cake/biscuits	11	4	13.3	[-22; 0]
12	Vegetables	14.6	8	18.9	**9**	Vegetables	23	18	18.6	[0; 18]
13	Eggs	12.8	8	15.3	**12**	Eggs	16.2	12	15.4	[0; 11]
14	Chicken	8.7	8	9.2	**19**	Chicken	8.1	4	6.7	[-4; 4]
15	Fruit juice	17.2	6	23.9	**10**	Fruit juice	21.9	12	26.6	[0; 12]
16	Sweets/chocolates	14.5	4	18.5	**15**	Sweets/chocolates	14.3	8	18.6	[-3; 4]
17	Samp/ mealie rice	9	4	14	**20**	Samp/ mealie rice	4.7	4	6.7	[-4; 0]
18	Fish	7.4	4	8.5	**21**	Fish	3.3	1.5	3.9	[-4; 0]
19	Soy mince / legumes	7	4	8.3	**17**	Soy mince/ legumes	7.4	6	12.9	[-4; 4]
20	Red meat	5.5	4	5.5	**22**	Red meat	3.2	1	5.1	[-4; 0]
21	Peanut butter/peanuts	6.6	3	8.8	**16**	Peanut butter/peanuts	11.5	8	13.9	[0; 8]
22	Cereal	9.5	2.5	12.6	**11**	Cereal	16.4	12	17.5	[0; 12]
23	Coffee creamer (Cremora)	20.5	0	35.9	**24**	Coffee creamer (Cremora)	2	0	5.7	[-4; 0]
24	Coffee	5.7	0	14.4	**23**	Coffee	5.5	0	15.5	[0; 0]
25	Low fat/skim milk	1.3	0	6.1	**25**	Low fat/skim milk	0.1	0	0.6	[0; 0]
26	Alcohol	0.3	0	1.6	**26**	Alcohol	0	0	0	[0; 0]

4.1.3.3 Adults

Reported usual intakes of different food items were calculated for 550 rural participants, consisting of 161 men (29,3%) and 389 women (70,7%); and 419 urban participants, consisting of 99 men (23,7%); and 320 women (76,4%). There were no significant differences in intake of food items between rural and urban women or rural and urban men (95% CI) (Table 4.21 and Table 4.22).

When comparing the frequent median intake of certain food groups for rural and urban men (Table 4.21), sugar; cooked porridge; tea, salt or stock; full cream milk; coffee, margarine/oil/fat; bread; fruit; and vegetables fell in the top ten list. Rural men consumed coffee creamer more frequently than urban men (14th rural vs. 24th urban). Urban men consumed alcohol a little more frequently than rural men (15th rural vs. 17th men).

Table 4.21: Frequency of intake of different food items for rural and urban men (times per month)

Rank	*Rural*				Rank	*Urban*				95% CI for difference in medians
	n=161	Mean	Median	Std Dev		*n=99*	Mean	Median	Std Dev	
1	Sugar	62	60	43.7	1	Sugar	63.6	60	44.2	[0; 0]
2	Porridge, cooked	53.2	60	26.2	2	Porridge, cooked	49.4	60	26.6	[0; 0]
3	Salt/ stock	42.6	30	32	4	Salt/ stock	36.7	30	23.7	[0; 0]
4	Tea	37.3	30	42.8	3	Tea	42.7	30	36	[0; 14]
5	Full cream milk	36.8	30	33.1	7	Full cream milk	23.8	12	27.2	[-18; 0]
6	Coffee	29.9	30	34.8	5	Coffee	27.5	30	31.6	[0; 0]
7	Margarine/ oil/ fat	26.7	30	25	6	Margarine/ oil/ fat	25.7	30	22.5	[0; 0]
8	Bread	22.6	16	21.7	8	Bread	20	12	24.4	[-4; 0]
9	Fruit	26.3	12	34.3	10	Fruit	17.8	8	20.1	[-4; 0]
10	Vegetables	17.2	12	16.1	9	Vegetables	19.9	12	20.2	[0; 0]
11	Cool drinks	19.3	8	29.5	11	Cool drinks	12	8	17.6	[-4; 0]
12	Chicken	10.4	8	9.8	13	Chicken	8.4	8	7.3	[-3; 0]
13	Eggs	10.5	5	17	12	Eggs	10.5	8	15.2	[0; 2]
14	Coffee creamer (Cremora)	26.7	4	36.5	24	Coffee creamer (Cremora)	12.5	1	21.8	[-4; 0]
15	Cake/biscuits	14.2	4	27.8	14	Cake/biscuits	14.2	4	60.8	[-2; 0]
16	Chips/crisps	9.4	4	13.2	18	Chips/crisps	7.2	4	9.7	[-3; 0]
17	**Alcohol**	**9.1**	**4**	**27.6**	15	**Alcohol**	**10.9**	**4**	**29.2**	[0; 0]
18	Fruit juice	8.4	4	16.9	20	Fruit juice	6.9	4	12.8	[-1; 0]
19	Fish	6.8	4	7.6	23	Fish	4.5	2	7.7	[-3; 0]
20	Soy mince/ legumes	6.8	4	7.2	16	Soy mince/ legumes	9	4	12.7	[0; 1]
21	Red meat	6.6	4	6.9	22	Red meat	4.7	4	5.6	[-3; 0]
22	Samp/ mealie rice	6.2	4	6.8	19	Samp/ mealie rice	7	4	9.4	[0; 0]
23	Sweets/chocolates	8.8	2	18	21	Sweets/chocolates	6.1	4	9.3	[0; 1]
24	Peanut butter/peanuts	6.5	1	13	17	Peanut butter/peanuts	8	4	10.8	[0; 1]
25	Cereal	3.7	0	7.1	25	Cereal	7.9	0	14.1	[0; 0]
26	Low fat/skim milk	1.5	0	11	26	Low fat/skim milk	1.6	0	12.2	[0; 0]

Table 4.22: Frequency of intake of different food items for rural and urban women (times per month)

Rank	*Rural*				Rank	*Urban*				95% CI for difference in medians
	n=389	Mean	Median	StdDev		*n=320*	Mean	Median	StdDev	
1	Sugar	66.4	60	43.8	1	Sugar	73	60	51.2	[0; 0]
2	Porridge, cooked	49.8	60	26.6	3	Porridge, cooked	47.3	30	26.4	[0; 0]
3	Tea	44.1	30	34.9	2	Tea	53.8	60	41.7	[0; 4]
4	Salt/ stock	43.9	30	32.3	4	Salt/ stock	38.7	30	25.6	[0; 0]
5	Margarine/ oil/ fat	36.5	30	29.5	5	Margarine/ oil/ fat	34.7	30	24.1	[0; 0]
6	Full cream milk	32.4	30	30.7	7	Full cream milk	25.2	30	26.4	[-4; 0]
7	Bread	26.6	30	24.8	6	Bread	28	30	26.7	[0; 0]
8	Fruit	28.4	12	32.6	9	Fruit	19.5	12	23.5	[-4; 0]
9	Vegetables	18.7	12	17.4	8	Vegetables	21.5	16	19.4	[0; 0]
10	Coffee	20.2	8	26.9	22	Coffee	18.7	0	28.3	[0; 0]
11	Cool drinks	20	8	28.3	10	Cool drinks	13.7	8	19.3	[-3; 0]
12	Cake/biscuits	19.8	8	35.6	15	Cake/biscuits	8.4	4	13.3	[-3; 0]
13	Chicken	12.3	8	13.4	11	Chicken	9.8	8	10.7	[-3; 0]
14	Coffee creamer (Cremora)	22.8	4	31.2	23	Coffee creamer (Cremora)	12.9	0	25.9	[0; 0]
15	Chips/crisps	10.2	4	15.4	14	Chips/crisps	8.6	4	11.3	[0; 0]
16	Eggs	9	4	13	12	Eggs	9	8	11.6	[0; 0]
17	Soy mince/ legumes	8.5	4	9	17	Soy mince/ legumes	7.7	4	9.7	[0; 0]
18	Samp/ mealie rice	6.6	4	7.1	18	Samp/ mealie rice	5.2	4	7.3	[-1; 0]
19	Fish	6	4	9.3	20	Fish	3.9	2.5	6.7	[-1; 0]
20	Red meat	5.7	4	7.6	19	Red meat	3.9	4	4.7	[-1; 0]
21	Sweets/chocolates	11.6	3	24.5	13	Sweets/chocolates	10.2	4	17.7	[0; 1]
22	Fruit juice	8.7	2	16.1	21	Fruit juice	8.2	2	13.4	[0; 0]
23	Peanut butter/peanuts	6.6	1	12.1	16	Peanut butter/peanuts	8	4	11.2	[0; 0]
24	Cereal	7.8	0	13.2	24	Cereal	8.3	0	12.1	[0; 0]
25	**Alcohol**	**3.9**	**0**	**14.9**	25	**Alcohol**	**1.4**	**0**	**3.2**	[0; 0]
26	Low fat/skim milk	1.4	0	7.6	26	Low fat/skim milk	1.4	0	8.2	[0; 0]

The median frequency of food consumption for women (Table 4.22) looks similar to that of the men, with consumption of sugar, porridge; tea and salt/stock ranking the highest (top four items). The top ten items consumed frequently by both urban- and rural women were: sugar; cooked porridge; tea, salt or stock; margarine/oil/fat; full cream milk; fruit; bread; and vegetables. Coffee creamer was consumed

more often among rural women (14th rural vs. 23rd urban), as was also the case among rural men. Rural women also consumed coffee more frequently than urban women (10th rural vs. 22nd urban). Alcohol consumption was listed much less frequently among women when compared to men.

4.1.4 Mean/median macronutrient intakes

Mean and median macronutrient intakes for both children and adults were calculated from the median number of food groups. The food items eaten were described in terms of exchanges; whereupon total exchanges were transferred to the calculation table as shown in Chapter 3 (Table 3.5), to estimate total macronutrient intakes. The dietary reference intakes (DRIs) for energy and macronutrients and the acceptable distribution ranges were used for comparison as set out by Table 3.6 and Table 3.7 in Chapter 3.

4.1.4.1 Children younger than two years

Mean and median macronutrient intakes were calculated for ten rural (4 boys; 6 girls), and 34 urban (16 boys; 18 girls) children younger than two years old. Dietary reference intakes used for energy were between 2394 kJ and 4393 kJ for boys and between 2184kJ and 4166 kJ for girls (Food & Nutrition Board et al., 2002:1324). Acceptable distribution ranges of macronutrients were 45-65% for carbohydrates; 5-20% for protein; and 30-40% for fat (USDA & USDHHS, 2010:15,Table 2-4) (Table 3.6 and Table 3.7). Confidence intervals for the median percentage differences between urban and rural participants were calculated.

Total mean energy intake for both rural and urban boys younger than two years fell within the recommendation. Energy intake was significantly higher in rural boys (4952,5 kJ) than in urban boys (3862,8kJ) (p=0.04) (Table 4.23). The energy distribution was more towards carbohydrates in urban boys (63,5%) than in rural boys (56,2%), and protein intake was almost the same for rural- (18,9%) and urban (19,5%) boys. Percentage energy from fat intake for urban boys was significantly lower (21,1%) than for rural boys (27,8%) (p=0.01).

Table 4.23: Mean macronutrient intake for rural and urban boys (<2 years) (N=20)

Variable	***Macronutrient intake***								**P value (on median)**
	Rural				**Urban**				
Boys (R=4; U=16)	**Min**	**Median**	**Mean**	**Max**	**Min**	**Median**	**Mean**	**Max**	
Carbohydrate (g)	146.0	159.5	163.8	190.0	30.0	135.5	144.3	291.0	0.16
% of total energy		*54.6*	*56.2*			*63.6*	*63.5*		
Protein (g)	39.0	57.0	55.0	67.0	4.0	41.5	44.3	154.0	0.18
% of total energy		*19.5*	*18.9*			*19.5*	*19.5*		
Fat (g)	20.0	35.0	36.3	55.0	3.0	17.5	21.4	76.0	0.01*
% of total energy		*26.8*	*27.8*			*18.3*	*21.1*		
Energy (kJ)	3945.0	4970.0	4952.5	5925.0	677.0	3624.0	3862.8	10199.0	0.04*

The picture looks similar for the girls younger than two years (Table 4.24), with rural girls (3788,3kJ) also consuming more energy than urban girls (2785,4kJ); and with total energy from fat being less in urban- (19,2%) than rural girls (24,3%). The difference in protein intake is more pronounced with 19,6% in urban girls, compared to 16,2% in rural girls.

Notwithstanding the differences between rural and urban children, total carbohydrate and protein intake, as a percentage of total energy, was within recommendations for both rural and urban boys and girls

younger than two years old. However, total fat intake, as a percentage of total energy, fell below recommendations for both rural and urban boys and girls in all areas.

Table 4.24: Mean macronutrient intake for rural and urban girls (<2 years) (N=24)

Variable	*Macronutrient intake*								95% CI for difference in medians
	Rural				Urban				
Girls (R=6; U=18)	**Min**	**Median**	**Mean**	**Max**	**Min**	**Median**	**Mean**	**Max**	
Carbohydrate (g) *% of total energy*	70.0	130.5 *64.7*	127.8 *57.4*	172.0	26.0	104.0 *71.0*	106.2 *64.8*	219.0	[-63;17]
Protein (g) *% of total energy*	17.0	37.0 *18.3*	36.0 *16.2*	48.0	3.0	34.0 *23.1*	32.1 *19.6*	58.0	[-18;9]
Fat (g) *% of total energy*	10.0	25.0 *27.7*	24.4 *24.3*	40.0	0.0	15.0 *22.8*	14.1 *19.2*	28.0	[-20;0]
Energy (kJ)	3135.0	3430.0	3788.3	5150.0	496.0	2497.5	2785.4	5254.0	[-1910;25]

4.1.4.2 Children older than two years

Mean and median macronutrient intakes were calculated for fifty rural (26 boys; 24 girls), and 112 urban (64 boys; 48 girls) children between two and seven years old. Dietary reference intakes used for energy were between 4393 kJ and 7316 kJ for boys and between 4166 kJ and 6896 kJ for girls (Food & Nutrition Board et al., 2002:1324). Acceptable distribution ranges of macronutrients were 45-65% for carbohydrates; 10-30% for protein; and 25-35% for fat (USDA & USDHHS, 2010:15,Table 2-4) (Table 3.6 and Table 3.7).

Total mean energy intake among rural boys (5926,5kJ) was higher than among urban boys (4743,4 kJ) (Table 4.25), but fell within recommendations. When looking at percentage of total energy from the macronutrients, a higher percentage of energy (66,3%) came from carbohydrates in urban boys, than rural boys (55,3%), but still met recommendations. Due to the higher carbohydrate intake among urban boys, fat intake was lower (20%) in comparison to rural boys (30,8%). Although the rural boys consumed fat within recommendations, the fat intake for urban boys fell below recommendations. On average, rural- and urban boys consumed almost equal amounts of energy from protein (17,2% and 17,6% respectively), which fell within recommendations.

Table 4.25: Mean macronutrient intake for rural and urban boys (>2 years) (N=90)

Variable	*Macronutrient intake*								95% CI for difference in medians
	Rural				Urban				
Boys (R=26; U=64)	**Min**	**Median**	**Mean**	**Max**	**Min**	**Median**	**Mean**	**Max**	
Carbohydrate (g) *% of total energy*	65.0	178.0 *53.7*	192.9 *55.3*	367.0	30.0	177.0 *67.7*	185.1 *66.3*	427.0	[-46;29]
Protein (g) *% of total energy*	20.0	62.0 *18.7*	60.0 *17.2*	111.0	4.0	45.5 *17.4*	49.1 *17.6*	154.0	[-22;-4]*
Fat (g) *% of total energy*	20.0	47.5 *32.0*	48.1 *30.8*	80.0	3.0	23.0 *19.7*	24.9 *20.0*	76.0	[-32;-17]*
Energy (kJ)	2170	5635.0	5926.5	10445	677	4444.0	4743.4	10199	[-2100;-334]*

Rural girls consumed more energy (5441 kJ) in total when compared with their urban counterparts (3959,4 kJ) (Table 4.26). Energy intake of urban girls did not meet the minimum recommendation of 4166kJ. When comparing the macronutrient intake of rural- and urban girls, the same pattern is seen as for the boys, where there is a higher percentage energy intake from carbohydrates under the urban girls (65,1% urban vs. 58,4% rural) and a lesser intake of fat than the rural girls (20,5% urban vs. 27,8% rural). Protein intakes fell within recommendations and were similar in urban girls (18,1%) and rural girls

(16,8%). Carbohydrate intakes also fell within recommendations. Urban girls met their recommendations for fat intake, whereas rural girls did not.

Table 4.26: Mean macronutrient intake for rural and urban girls (>2 years) (N=72)

Variable	*Macronutrient intake*								95% CI for difference in medians
	Rural				Urban				
Girls (R=24; U=48)	**Min**	**Median**	**Mean**	**Max**	**Min**	**Median**	**Mean**	**Max**	
Carbohydrate (g) *% of total energy*	70	171.5 *55.0*	186.8 *58.4*	379	26	138.5 *64.4*	151.6 *65.1*	490	[-67;-3]*
Protein (g) *% of total energy*	17	53.5 *17.2*	53.7 *16.8*	85	3	39.0 *18.1*	42.1 *18.1*	120	[-24;-3]*
Fat (g) *% of total energy*	10	35.0 *25.1*	39.8 *27.8*	85	0	18.0 *18.7*	21.4 *20.5*	66	[-25;-10]*
Energy (kJ)	3135	5300.0	5441.0	10720	496	3658.0	3959.4	12123	[-2514;-675]*

4.1.4.3 Adults

The median and mean macronutrient intakes were calculated for 550 rural (161 men; 389 women) and 319 urban participants (99 men; 320 women) (Table 4.27). Recommended daily energy intakes were 12881 kJ for men and 10093 kJ for women (Food & Nutrition Board et al., 2002:1324) of which 45-65% were to come from carbohydrates; 10-35% from protein and 20-35% from fat (USDA & USDHHS, 2010:15,Table 2-4) (Table 3.6 and Table 3.7).

Table 4.27: Mean macronutrient intake for rural and urban men (N=260)

Variable	*Macronutrient intake*								95% CI for difference in medians
	Rural				Urban				
Men (R=161; U=99)	**Min**	**Median**	**Mean**	**Max**	**Min**	**Median**	**Mean**	**Max**	
Carbohydrate (g) *% of total energy*	0	297.0 *59.4*	305.4 *60.2*	297	23	260.0 *68.4*	259.0 *62.2*	730	[-77;-20]*
Protein (g) *% of total energy*	0	85.0 *17.0*	88.7 *17.5*	85	0	68.0 *17.9*	76.1 *18.3*	223	[26;-6]
Fat (g) *% of total energy*	0	50.0 *22.4*	57.3 *25.2*	50	0	30.0 *17.6*	43.3 *23.3*	166	[-26;-10]*
Energy (kJ)	0	8495.0	8630.1	8495	900	6463.0	7078.9	21620	[-2630;-1020]*

Rural men consumed significantly more energy than their urban counterparts (8630,1 kJ rural vs. 7078,9 kJ urban) (Table 4.27). However, both rural and urban men consumed less than their daily recommendation. Carbohydrate intakes fell within recommendations with both rural- (60,2%) and urban (62,2%) men. Protein intakes also fell within recommendations in both urban (18,3%) and rural men (17,5%). Fat intakes were also within recommendations among both rural (25,2%) and urban (23,3%) men.

Table 4.28: Mean macronutrient intake for rural and urban women (N=709)

Variable	*Macronutrient intake*								95% CI for difference in medians
	Rural				Urban				
Women (R=389; U=320)	**Min**	**Median**	**Mean**	**Max**	**Min**	**Median**	**Mean**	**Max**	
Carbohydrate (g) *% of total energy*	32	260.0 *57.4*	275.1 *60.3*	769	0	242.5 *65.5*	246.5 *63.3*	629	[-43;-14]*
Protein (g) *% of total energy*	8	76.0 *16.8*	77.2 *16.9*	161	0	66.0 *17.8*	68.8 *17.7*	202	[-14;-5]*
Fat (g) *% of total energy*	0	50.0 *24.7*	52.9 *25.9*	165	0	33.0 *19.9*	39.7 *22.8*	146	[-20;-12]*
Energy (kJ)	1820	7705.0	7754.9	19020	0	6294.0	6620.8	15905	[-1597;-855]*

Rural women consumed marginally more energy (7754,9 kJ) than their urban counterparts (6620,8 kJ) (Table 4.28). However, neither met their daily recommendations for energy intake. Carbohydrate intake

fell within recommendations among both urban (63,3%) and rural (60,3%) women. Fat intake was higher among rural women (25,9%) when compared to urban women (22,8%), and was within recommendations. Protein intake was similar in both groups (rural 16,9% vs. Urban 17,7%), which fell within recommendations.

4.2 Anthropometric variables

Where applicable, 95% confidence intervals for median differences between urban and rural participants were calculated to determine significance of differences.

4.2.1 Children

Anthropometric data for children aged zero to seven years were compared to WHO standards (Z scores), except for the head circumference data, where both the CDC charts (up to three years old) and the WHO charts (up to two years old) were used for comparison. Results are reported for weight-for-age, height-for-age and weight-for-height, as well as BMI-for-age and head circumference-for-age as set out in Table 4.29 to Table 4.37 below.

4.2.1.1 Weight-for-age

Weight-for-age of 32 rural- and 69 urban boys, as well as 28 rural- and 47 urban girls was determined and compared to the WHO standard (Table 4.29).

When comparing the weight-for-age data for rural and urban participants, about two-thirds (62,6%) of rural boys were all in various stages of underweight as compared to only 39,2% of urban boys (Table 4.29). There were significantly more rural boys who were moderately underweight than urban boys. A larger percentage of underweight rural boys were moderately underweight (34,4%), as opposed to the largest percentage of underweight urban boys who were mildly underweight (20,3%). Only 14,5% (more than half the amount of rural boys) were moderately underweight. None of the 32 rural boys were overweight, whereas 16% of the urban boys were in various stages of overweight, with the largest percentage (10,1%) being mildly overweight. Almost 45% (44,9%) of urban- and 37,5% of rural boys were of normal weight.

Table 4.29: Weight-for-age

	Boys				95% CI for difference in medians	Girls				95% CI for difference in medians
	Rural		*Urban*			*Rural*		*Urban*		
	N = 32		N = 69			N = 28		N = 47		
	N	%	N	%		N	%	N	%	
>+3SD (Severely overweight)	0	0	1	1.5	[-9.3; 7.8]	1	3.6	0	0.0	[-17.7; 4.5]
+2.1 to +3SD (Moderately overweight)	0	0	3	4.4	[-6.7; 12.0]	0	0.0	2	4.3	[-8.2; 14.2]
+1.1 to +2SD (Mildly overweight)	0	0	7	10.1	[-1.7; 19.5]	2	7.1	6	12.8	[-11.3; 19.1]
-1SD to +1SD (Normal)	12	37.5	31	44.9	[-13.1; 26.1]	11	39.3	21	44.7	[-17.2; 26.5]
-1.1SD to -2SD (Mildly underweight)	7	21.9	14	20.3	[-20.2; 13.8]	8	28.6	11	23.4	[-26.1; 14.0]
-2.1SD to -3SD (Moderately underweight)	11	34.4	10	14.5	[-38.4; -2.6]*	5	17.9	2	4.3	[-31.6; 0.5]
<-3SD (Severely underweight)	2	6.3	3	4.4	[-16.1; 7.0]	1	3.6	5	10.6	[-8.3; 19.4]

There were no significant differences between the urban and rural results for girls, a similar percentage of rural (39,3%) and urban (44,7%) girls were of normal weight. Half of the rural girls (50,1%) and 38,3% of urban girls were in various stages of underweight, with a larger percentage of both rural- (28,6%) and urban girls (23,4%) being classified as mildly underweight. One-tenth (10,7%) of rural and 17,1% of urban girls were in various stages of overweight, with 7,1% of rural girls being mildly overweight and

3,6% being severely overweight. No urban girls were severely overweight, but 12,8% were found to be mildly overweight and a further 4,3% were moderately overweight.

4.2.1.2 Height-for-age

Height for age of 32 rural- and 69 urban boys, as well as 28 rural- and 47 urban girls were determined and compared to the WHO standards (Table 4.30). There were no significant difference between rural and urban boys or rural and urban girls. Rural boys were generally shorter than urban boys. About two-thirds (65,7%) of both rural- and urban (62,3%) boys were shorter than the standard. A larger percentage of short rural boys (25%) were severely stunted as opposed to 29% of urban boys who were moderately stunted. However, about another third of both rural (31,3%) boys and urban (29,0%) boys were of normal height. Only 3,1% of rural boys were mildly tall as opposed to 5,8% of urban boys. A small percentage of urban boys (1,5%) were moderately tall and another 1,5% were very tall.

Table 4.30: Height-for-age

	Boys				95% CI for difference in medians	Girls				95% CI for difference in medians
	Rural		*Urban*			*Rural*		*Urban*		
	N = 32		N = 69			N = 28		N = 47		
	N	%	N	%		N	%	N	%	
>+3SD (Very tall)	0	0	1	1.5	[-9.3; 7.8]	1	3.8	1	2.1	[-15.7; 8.0]
+2.1 to +3SD (Moderately tall)	0	0	1	1.5	[-9.3; 7.8]	0	0.0	1	2.1	[-10.1; 11.1]
+1.1 to +2SD (Mildly tall)	1	3.1	4	5.8	[-10.4; 11.2]	0	0.0	1	2.1	[-10.1; 11.1]
-1SD to +1SD (Normal height)	10	31.3	20	29.0	[-22.0; 15.4]	12	42.9	16	34.0	[-30.4; 12.9]
-1.1SD to -2SD (Mildly stunted)	6	18.8	10	14.5	[-23.8; 11.2]	7	25.0	10	21.3	[-24.3; 14.7]
-2.1SD to -3SD (Moderately stunted)	7	21.9	20	29.0	[-12.2; 23.0]	4	14.3	13	27.7	[-6.9; 29.9]
<-3SD (Severely stunted)	8	25.0	13	18.8	[-24.8; 9.8]	4	14.3	5	10.6	[-21.9; 11.0]

As in the case of boys, rural girls were also generally shorter than urban girls The largest percentage of both rural (42,9%) and urban (34,0%) girls were of normal height. More than half of both rural (53,6%) and urban (59,6%) girls were shorter than normal. A quarter (25,0%) of rural girls were mildly stunted as opposed to 21,3% of urban girls. A larger percentage of urban girls (27,7%) were moderately stunted. When looking at the data for the girls, 3,8% of rural girls were very tall, as opposed to 6,3% of urban girls who were either mildly- (2,1%), moderately- (2,1%), or very tall (2,1%), respectively.

4.2.1.3 Weight-for-height

The weight-for-height data of 28 rural- and 58 urban boys, as well as 23 rural- and 41 urban girls are categorized in Table 4.31.There were significantly more rural boys who were mildly wasted than urban boys. A larger percentage of rural boys (46,4%) had a lower than normal weight-for-height when compared to only 15,5% of urban boys; 17,5% of rural girls; and 19,6% of urban girls. A larger percentage of both urban boys (39,6%) and urban girls (39,0%) had a higher weight-for-height than their rural counterparts, although the percentage rural girls did not fall far behind (30,4% of rural girls). Only 17,9% rural boys had a higher than normal weigh-for-height.

Table 4.31: Weight-for-height

	Boys				95% CI for difference in medians	Girls				95% CI for difference in medians
	Rural		*Urban*			*Rural*		*Urban*		
	N = 28		N = 58			N = 23		N = 41		
	N	%	N	%		N	%	N	%	
>+3SD (Severely high)	0	0	5	8.6	[-4.4; 18.6]	0	0.0	1	2.4	[-12.0; 12.6]
+2.1 to +3SD (Moderately high)	1	3.6	6	10.3	[-8.4; 17.6]	2	8.7	5	12.2	[-15.9; 18.3]
+1.1 to +2SD (Mildly high)	4	14.3	12	20.7	[-12.8; 21.2]	5	21.7	10	24.4	[-20.1; 21.9]
-1SD to +1SD (Normal)	10	35.7	26	44.8	[-12.9; 28.8]	12	52.2	17	41.5	[-33.8; 13.8]
-1.1SD to -2SD (Mildly low)	9	32.1	3	5.2	[-45.8; -10.2]*	2	8.7	4	9.8	[-18.0; 15.3]
-2.1SD to -3SD (Moderately low)	3	10.7	5	8.6	[-19.3; 10.1]	1	4.4	2	4.9	[-16.5; 12.3]
<-3SD (Severely low)	1	3.6	1	1.7	[-16.1; 6.1]	1	4.4	2	4.9	[16.5; 12.3]

There were no significant differences between the percentage of rural and urban girls that fell within each of the various weight-for-height categories. Almost a third (35,7%) of rural boys and 44,8% of urban boys fell within the normal weight-for-height category. About a third (32,1%) of rural boys and 5,2% of urban boys had a mildly low weight-for-height (mildly wasted); 10,7% of rural- and 8,6% of urban boys had a moderately low weight-for-height (moderately wasted); and 3,6% of rural- and 1,7% of urban boys had a severely low weight-for-height (severely wasted). More urban boys (39,6%) than rural boys (17,9%) had above normal weight-for-height, with the largest percentage of both urban- (20,7%) and rural (14,3%) boys having mildly high weight-for-height.

More than half of rural girls (52,2%) and 41,5% of urban girls were of normal weight-for-height. Only 17,5% of rural girls and 19,6% of urban girls were below normal for weight-for-height; with 8,7% of rural and 9,8% of urban girls being mildly wasted; 4,4% of rural-and 4,9% of urban girls being moderately wasted and 4,4% of rural- and 4,9% of urban girls being severely wasted. A total of 30,4% of rural girls and 39,0% of urban girls had higher than normal weight-for-height. About a fifth (21,7%) of rural girls and about a quarter (24,4%) of urban girls had mildly high weight-for-height; 8,7% of rural- and 12,2% of urban girls had moderately high weight-for-height; 2,4% of urban girls and no rural girls had a severely high weight-for-height.

4.2.1.4 BMI-for-age

The BMI-for-age was categorized according to age for sixty rural children (32 boys and 28 girls) and 116 urban children (69 boys and 47 girls) (Table 4.32). No significant differences were found between the percentage of rural and urban boys or rural and urban girls that fell within each of the various categories for BMI-for-age.

Table 4.32: BMI for age

	Boys				95% CI for difference in medians	Girls				95% CI for difference in medians
	Rural N = 32		*Urban* N = 69			*Rural* N = 28		*Urban* N = 47		
	N	%	N	%		N	%	N	%	
>+3SD	0	0	6	8.7	[-3.0; 17.7]	0	0.0	1	2.1	[-10.1; 11.1]
+2.1 to +3SD	2	6.3	8	11.6	[-9.6; 16.0]	2	7.1	3	6.4	[-16.8; 11.2]
+1.1 to +2SD	5	15.6	15	21.7	[-12.0; 20.2]	5	17.9	13	27.7	[-10.9; 27.1]
-1SD to +1SD (Normal)	14	43.8	26	37.7	[-26.0; 13.5]	14	50.0	20	42.6	[-29.2; 15.0]
-1.1SD to -2SD (Mildly low)	8	25.0	8	11.6	[-31.4; 1.8]	4	14.3	5	10.6	[-21.9; 11.1]
-2.1SD to -3SD (Moderately low)	0	0.0	5	7.3	[-4.2; 15.9]	2	7.1	4	8.5	[-15.0; 13.9]
<-3SD (Severely low)	3	9.4	1	1.5	[-22.8; 0.9]	1	3.6	1	2.1	[-15.7; 8.0]

Almost half (43,8%) of rural boys were of normal BMI in comparison with 37,7% of urban boys. According to the BMI-for-age, there were more rural boys (34,4%) than urban boys (20,4%) with a BMI-for-age lower than normal. Almost a tenth (9,4%) of rural boys were found to have a severely low BMI, with the remaining 25% having a mildly low BMI. Among urban boys, 11,6% had a mildly low BMI, 7,3% had a moderately low BMI, and only 1,5% had a severely low BMI. Overall, urban boys were fatter than their rural counterparts. A total of 42% of urban boys were classified as having a BMI-for-age above normal, whereas only half as many of the rural boys (21,9%) were found to have a BMI-for-age above normal. Most urban boys (21,7%) only had a mildly high BMI, a further 11,6% had a moderately high BMI and 8,7% had a severely high BMI. In comparison, only 15,6% of rural boys had a mildly high BMI, and a further 6,3% had a moderately high BMI.

Half (50%) of rural girls were of normal BMI in comparison with 42,6% of urban girlss. According to the BMI-for-age, there were more rural girls (25%) than urban girls (21,2%) with a BMI-for-age lower than

normal. A total of 36,2% of urban girls were classified as having a BMI-for-age above normal, whereas only 25% of the rural girls were found to have a BMI-for-age above normal.

The median BMI-for-age was calculated for 37 rural boys, 76 urban boys, 37 rural girls and 54 urban girls (Table 4.33). The median BMI for both rural- and urban boys was in the normal range (overweight was between 17,42 kg/m^2 and 18,41 kg/m^2 in boys aged two to seven years old) as well as for rural- and urban girls (between 17,15 kg/m^2 and 18,02 kg/m^2 in girls aged two to seven years old), according to the cut-off points for overweight in children of different ages suggested by Cole et al. (2000:1-6).

Table 4.33: Median BMI for age

	Boys		Girls	
	Rural	***Urban***	***Rural***	***Urban***
	N = 37	N = 76	N = 37	N = 54
Median (kg/m^2)	15.2	16.4	15.8	16.5

4.2.1.5 Head circumference

Head circumference data was compared with both the WHO charts (CDCP, 2010:online), which went up to two years of age (Table 4.34), and the CDC charts (Mahan & Escott-Stump, 2004d:1178, Appendix 7; Mahan & Escott-Stump, 2004e:1181, Appendix 10), which went up to three years of age (Table 4.35).

Table 4.34: Head circumference of boys and girls 0-2 years old (WHO charts)

	Boys				Girls			
	Rural		***Urban***		***Rural***		***Urban***	
	N=9		N=29		N=10		N=22	
	N	%	N	%	N	%	N	%
Larger than normal	2	22.2	3	10.3	3	30	7	31.8
Normal	3	33.3	13	44.8	5	50	9	40.9
Smaller than normal	4	44.4	13	44.8	2	20	6	27.3

Head circumference data of 9 rural- and 29 urban boys, as well as 10 rural- and 22 urban girls were categorized for children younger than two years old (Table 4.34). The head circumference data was compared to the WHO standards. A large percentage of urban boys had either a normal head circumference (44,8%) or smaller than normal head circumference (44,8%). Only 10,3% of urban boys had a larger than normal head circumference in comparison with 22,2% of rural boys. One-third or rural boys (33,3%) had a normal head circumference. About the same percentage of rural boys (44,4%) than urban boys (44,8%) had a smaller than normal head circumference.

In contrast to the boys, half of rural girls (50%) had a normal head circumference as opposed to only 40,9% of urban girls. Of the remaining rural girls, thirty percent had heads larger than normal and twenty percent had heads smaller than normal. In the urban girls, there was about the same percentage of girls (31,8%) with larger than normal head circumferences, but there were more urban girls (27,3%) who had smaller than normal head circumferences.

Head circumference data of 17 rural- and 42 urban boys, as well as 13 rural- and 31 urban girls were categorized for children younger than three years old (Table 4.35). As expected, the percentage distribution stayed similar for boys when compared with CDC standards, with urban boys having marginally more boys with normal head circumferences (40,5%) versus rural boys (35,3%). However, for girls the results obtained when compared to the CDC standards were different to the WHO

standards, with more urban girls (51,6%) having normal head circumferences as compared to rural girls (46,2%).

Table 4.35: Head circumference of boys and girls 0-3 years old (CDC charts)

	Boys				Girls			
	Rural		***Urban***		***Rural***		***Urban***	
	N=17		N=42		N=13		N=31	
	N	%	N	%	N	%	N	%
Larger than normal	3	17.6	5	11.9	5	38.5	7	22.6
Normal	6	35.3	17	40.5	6	46.2	16	51.6
Smaller than normal	8	47.1	20	47.6	2	15.4	8	25.8

When the CDC charts were used as a standard, there were more boys with smaller than normal head circumferences than with the WHO charts, with 47,8% of urban boys and 47,1% of rural boys now having smaller than normal heads, and only 11,9% of urban boys having larger than normal heads versus 17,6% of rural boys. More rural girls had larger heads (38,5%) versus urban girls (22,6%) and more urban girls had smaller heads (25,8%) than rural boys (15,4%).

4.2.2 Adults

Results of anthropometric assessment for adults aged 25 to 64 years were categorized for BMI; waist circumference; upper arm circumference; triceps skinfold; upper arm fat area; upper arm muscle area; and body fat percentage, as set out in Table 4.36 to Table 4.43 below.

4.2.2.1 BMI

BMI of 163 rural and 100 urban men, as well as 390 rural- and 319 urban women were categorized in Table 4.36.

Table 4.36: BMI

	Males				***p-value***	Females				***p-value***
	Rural		***Urban***			***Rural***		***Urban***		
	N=163		N=100			N=390		N=319		
	N	%	N	%		N	%	N	%	
<18.5 kg/m^2 (Underweight)	54	33.1	23	23.0	0.09	36	9.2	28	8.8	0.9
18.5 – 24.9 kg/m^2 (Normal)	71	43.6	61	61.0	0.007*	98	25.1	80	25.1	0.9
25 – 29.9 kg/m^2 (Overweight)	25	15.3	12	12.0	0.5	87	22.3	80	25.1	0.4
≥30 kg/m^2 (Obese)	13	8.0	4	4.0	0.2	169	43.3	131	41.1	0.6
Median BMI (kg/m^2)	20.2		19.7			28.0		27.9		

The median BMI for urban men (19,7 kg/m^2) was only slightly lower than that of rural men (20,2 kg/m^2) with median BMI of both groups falling in the normal range. The median BMI for urban women (27,9 kg/m^2) was similar to the BMI for rural women (28,0 kg/m^2), with median BMI of both groups falling in the overweight range.

Overall, more urban men (61,0%) were of normal weight than rural men (43,6%). Consequently more rural men were both underweight and overweight/obese than the urban men. A third of rural men (33,1%) and 23% of urban men were underweight. As far as overweight was concerned, 15,3% of rural men and 12% of urban men were overweight and a further 8% of rural men and 4% of urban men were obese.

Only a quarter of both rural (25,1%) and urban (25,1%) women had a normal BMI and only 9,2% of rural women and 8,8% of urban women were found to be underweight. The remaining 22,3% of rural women and 25,1% of urban women were overweight and a further 43,3% of rural women and 41,1% of urban women were obese.

4.2.2.2 Waist circumference

Waist circumference of 163 rural and 100 urban men, as well as 382 rural- and 319 urban women are noted in Table 4.37. Data was analyzed using the cut-off points listed in Table 3.11 under section 3.5.2.1.

Compared to their rural counterparts, significantly more urban men had a normal waist circumference (p=0.002). Significantly more rural than urban men thus had a waist circumference that placed them at risk for developing CDLs (p=0.01). Significantly fewer rural women (24,4%) than urban women (32%) had a normal waist circumference (p=0.03). About one-fifth (17,8%) of rural and 19,4% of urban women were at risk for developing CDLs. More rural women were significantly at high risk of developing CDLs as a result of high waist circumference (57,9%) than urban women (48,6%) (p=0.02). Median waist circumference for rural women was 92 cm (high risk) and for urban women 87cm (at risk).

Table 4.37: Waist circumference

	Males				***p-value***	**Females**				***p-value***
	Rural		***Urban***			***Rural***		***Urban***		
	N=163		N=100			N=382		N=319		
	N	%	N	%		N	%	N	%	
Normal wc (Male: <94cm; Female: <80cm)*	130	79.8	94	94.0	0.002*	93	24.4	102	32.0	0.03*
At risk wc (Male: ≥94cm; Female: ≥80cm)*	17	10.4	2	2.0	0.01*	68	17.8	62	19.4	0.6
High risk wc (Male: ≥102cm; Female: ≥88cm) *	16	9.8	4	4.0	0.09	221	57.9	155	48.6	0.02*
Median wc	78.5		76.0			92.0		87.0		

* cut-off points as set out by Lee & Nieman 2010:180, Table 6.7 and Gibson, 2005:284

Most men from both rural (79,8%) and urban (94%) areas had a normal waist circumference. Only 10,4% of rural men and 2% of urban men were at risk for developing CDLs and only 9,8% of rural and 4% of urban men were at high risk for developing CDLs, according to waist circumference. The median waist was 78,5cm (normal) for rural men and 76 cm (normal) among urban men.

4.2.2.3 Triceps skinfold

Triceps skinfold data of 164 rural and 100 urban men, as well as 379 rural and 319 urban women are categorized in Table 4.38.

Table 4.38: Triceps skinfold

	Males				***p-value***	**Females**				***p-value***
	Rural		***Urban***			***Rural***		***Urban***		
	N=164		N=100			N=379		N=319		
	N	%	N	%		N	%	N	%	
<15th percentile	47	28.7	40	40.0	0.053	93	24.5	57	17.9	0.05*
15th -75th percentile	78	47.6	47	47.0		161	42.5	145	45.5	
>75th percentile	39	23.8	13	13.0		125	33.0	117	36.7	

Almost half of both rural (47,6%) and urban (47%) men had triceps skinfolds between the 15th to 75th percentile. Significantly more urban men (40%) had a triceps skinfold below the 15th percentile than

rural men (p=0.05). On the other hand, another quarter of rural men (23,8%) had triceps skinfolds above the 75th percentile.

The amount of women with normal triceps skinfolds was similar to the men, with 42,5% of rural women and 45,5% of urban women falling between the 15th to 75th percentile range. About the same amount (24,5%) of rural women compared to rural men (28,7%) had triceps skinfolds below 15th percentile, but only 17,9% or urban women had triceps skinfolds lower than the 15th percentile. There were marginally more urban women (36,7%) than rural women (33%) who had a triceps skinfold above the 75th percentile.

4.2.2.4 Upper arm fat area

Upper arm fat area data of 161 rural and 100 urban men, as well as 375 rural and 319 urban women are categorized in Table 4.39.

Table 4.39: Upper arm fat area

	Males				*p-value*	Females				*p-value*
	Rural		***Urban***			***Rural***		***Urban***		
	N=161		N=100			N=375		N=319		
	N	%	N	%		N	%	N	%	
<15th percentile (low)	68	42.2	56	56.0	0.02*	84	22.4	70	21.9	0.8
15th -75th percentile (normal)	62	38.5	34	34.0		141	37.6	138	43.3	
>75th percentile (high)	31	19.3	10	10.0		150	40.0	111	34.8	

More rural- and urban men had normal (15th to 75th percentile) to low (<15th percentile) upper arm fat areas when compared to rural and urban women, who had a larger percentage with normal (15th to 75th percentile) to high (>75th percentile) upper arm fat areas. Significantly fewer rural men (42,2%) than urban men (56%) had an upper arm fat areas under the 15th percentile (p=0.02). More rural men (19,3%) had an upper arm fat area above the 75th percentile when compared to urban men (only 10%). Only 22,4% of rural and 21,9% of urban women had an upper arm fat area below the 15th percentile. Almost the same percentage of rural men (38,5%) and rural women (37,6%) had an upper arm fat area in the normal range (15th to 75th percentile). However, more urban women (43,3%) had a normal upper arm fat area when compared with urban men (34,%). Forty percent of the rural women and 34,8% of urban women had an upper arm fat area larger than the 75th percentile.

4.2.2.5 Upper arm muscle area

The data of 161 rural and 100 urban men, as well as 375 rural and 319 urban women were analyzed as to upper arm muscle area (Table 4.40).

Table 4.40: Upper arm muscle area

	Males				*p-value*	Females				*p-value*
	Rural		***Urban***			***Rural***		***Urban***		
	N=161		N=100			N=375		N=319		
	N	%	N	%		N	%	N	%	
<15th percentile	82	50.9	79	79.0	<0.0001*	19	5.16	52	16.3	<0.0001*
15th -75th percentile	65	40.4	19	19.0		118	31.5	176	55.2	
>75th percentile	14	8.7	2	2.0		238	63.5	91	28.5	

The majority of both rural (50,9%) and urban (79%) men had a low (<15th percentile) upper arm muscle area. A significantly higher percentage of urban men had a low upper arm muscle area than rural men (p<0.0001). Most rural females (63,5%) had a high (>75th percentile) upper arm muscle area and most urban females (55,2%) had a normal (between 15th to 75th percentile) upper arm muscle area.

More than forty percent of rural men (40,4%) and one-fifth (19%) of urban men had a normal upper arm muscle area. Only 8,7% of rural men and 2% of urban men had upper arm muscle areas above the 75th percentile.

Almost two-thirds (63,5%) of rural women had an upper arm muscle area above the 75th percentile, as opposed to only 28,5% of urban women. More than half (55,2%) of urban women and 31,5% of rural women had a normal upper arm muscle area. Significantly more urban women (16,3%) than rural women (5,2%) had an upper arm muscle area below the 15th percentile (p<0.0001).

4.2.2.6 Body fat percentage

Body fat percentage was determined in 161 rural and 99 urban men (Table 4.41). Significantly more urban men (90,9%) than rural men (78,3%) had an acceptable low (6-15%) body fat percentage (p=0.001), while significantly more rural (17,4%) than urban men (6,1%) had an acceptable high (16-24%) body fat percentage (p=0.001). Few rural (3,1%) and urban (3%) men had a high body fat percentage. No urban men and only 1,2% of rural men had a body fat percentage below 5%.

Table 4.41: Body fat percentage rural and urban men

	Rural		***Urban***		***p-value***
	N=161		N=99		
	N	%	N	%	
≤5% (low)*	2	1.2	0	0.0	0.5
6-15% (acceptable low)*	126	78.3	90	90.9	0.001*
16-24% (acceptable high)*	28	17.4	6	6.1	0.001*
≥25% (high)*	5	3.1	3	3.0	0.9

* Cut-off points as defined by Lee & Nieman, 2010:193, Table 6.11)

Body fat percentage was determined in 375 rural and 316 urban women (Table 4.42). No rural women and only 0,3% of urban women had a low body fat percentage. About one-third of rural (32,8%) and urban (30,4%) women had an “acceptable low” body fat percentage. Another quarter of rural (22,9%) and urban (26,9%) women had an “acceptable high” body fat percentage. About 44,3% of rural and 42,4% of urban women had a high body fat percentage.

Table 4.42: Body fat percentage of rural and urban women

	Rural		***Urban***		***p-value***
	N=375		N=316		
	N	%	N	%	
≤8% (low)	0	0.0	1	0.3	
9-23% (acceptable low)	123	32.8	96	30.4	0.4
24-31% (acceptable high)	86	22.9	85	26.9	0.2
≥32% (high)	166	44.3	134	42.4	0.7

* Cut-off points as defined by Lee & Nieman, 2010:193, Table 6.11)

4.2.3 Children vs. caregivers

Data was also categorized as to how many malnourished children resided in the same household as an overweight/obese caregiver. Among rural children, the prevalence of stunting (height-for-age <-2SD) was 42,2% (n=19). A third (33,3%; n=15) were underweight (weight-for-age <-2SD) and 13,3% (n=6) were wasted (weight-for-height <-2SD). In urban children fifty percent (n=39) were stunted; 19,2% were underweight (n=15) and 10,3% (n=8) were wasted.

Table 4.43: Prevalence of malnourished children residing with an overweight/obese caregiver

Adults	Children											
BMI	**Height-for-age (N=123) (rural n=45; urban n=78)**				**Weight-for-age (N=123) (rural n=45; urban n=78)**				**Weight-for-height (N=123) (rural n=45; urban n=78)**			
	< -2 SD **Stunted**		≥ -2 SD		< -2 SD **Underweight**		≥ -2 SD		< -2 SD **Wasted**		≥ -2 SD	
	R n=19	***U n=39***	***R n=26***	***U n=39***	***R n=15***	***U n=15***	***R n=30***	***U n=63***	***R n=6***	***U n=8***	***R n=39***	***U n=70***
Underweight and normal weight BMI < 25 kg/m²	7	11	5	10	5	5	7	16	0	2	12	19
Overweight and obese BMI ≥ 25 kg/m²	**12**	**28**	21	29	**10**	**10**	23	47	**6**	**6**	27	51

Of the 19 rural and 30 urban children who were stunted, 12 rural children (63,2%) and 28 urban children (71,8%) resided with an overweight/obese caregiver (Table 4.43). Of the 15 rural and 15 urban children who were underweight, ten rural children (66,7%), as well as ten (66,7%) urban children, lived with an overweight/obese caregiver. All of the six wasted rural children (100%) had an overweight/obese caregiver. Of the eight wasted urban children, six children (75%) had an overweight/obese caregiver. Therefore, less rural than urban children who were stunted had an overweight/obese caregiver, but more rural than urban children who were wasted had an overweight/obese caregiver.

When looking at the total percentage of children, 26,7% (n=12) of rural children and 35,9% (n=28) of urban children were stunted and also lived with an overweight/obese caregiver. Another 22,2% (n=10) of rural children and 12,8% (n=10) of urban children were underweight and also lived with an overweight/obese caregiver. A further 13,3% (n=6) of rural children and 7,7% (n=6) of urban children were wasted and lived with an overweight/obese caregiver.

Chapter 5 - Discussion of results

5.1 Introduction

The main aim of this study was to determine the diet and anthropometric status of adults (between 25 and 64 years old) and pre-school children (zero to seven years old) in rural and urban areas and to describe anthropometric status of children and adults in the same household.

In this chapter, data for both dietary intake and anthropometry is divided into three age groups: children younger than two years old; children aged between two to seven years old (older than two); and adults aged 25 to 64 years old. Results of the various groups were compared to similar studies conducted in South Africa among similar age groups.

5.2 Limitations of the study

5.2.1 Study population

Sample sizes for children were small, especially for those younger than two years of age. There were also more urban than rural children, and was consistently kept in mind when interpreting results.

With respect to the adult sample, only about one in four participants were men in both rural and urban participants, which means that the male gender was under-represented. Because data was only gathered during weekdays (not weekends), it is possible that a portion of employed male participants who were eligible to participate in study interviews, could not attend due to work obligations.

The median age for both male and female participants was between 45 and 50 years of age. The fact that a medical examination was performed on each participant, could have resulted in persons with illnesses being more likely to attend, especially in rural areas where medical services are usually limited. This could also be the reason why the median age of rural participants (men: 50 years; women: 49 years) was older than that of urban participants (men: 45 years; women: 45 years), which could have impacted on results. This bias is acknowledged, and taken into account in the interpretation of results.

In some instances, a language barrier occurred. In order to overcome this limitation, students that were fluent in the language of choice of participants assisted with completing those interviews. All consent forms and information documents were available in the language of choice (English, Afrikaans and Sotho).

It should also be kept in mind that HIV status, although not determined in this study, may have had an influence on nutritional status.

5.2.2 Dietary intake

For practical reasons, only one 24-hour recall of reported usual intake was included in this study. The use of a short qualitative FFQ was included to increase the likelihood of obtaining good information related to usual food intake.

In open-ended methods, like the 24-hour recall of usual intake, persons may consciously or unconsciously tend either to deny or to exaggerate their food intake (Koh & Owen, 2000:178). Consumption of fruits and vegetables may be over-reported and consumption of high fatty junk foods and alcohol may be under-reported. Overweight persons tend to under-report their food intake (Koh & Owen, 2000:180).

Over-reporting can also occur when administering a FFQ with an overly long list of foods. On the other hand, under-reporting could be due to failure of the interviewer to ask the right questions about food preparation or the use of a cultural food (Koh & Owen, 2000:181). In an effort to obtain good quality information in this study, the short FFQ included foods commonly consumed by the target population.

The food frequency questionnaire used in this study was only qualitative and not quantitative. It could thus indicate the ***frequency*** of food consumption, but not the ***amounts*** in which food items were consumed.

The choice of techniques used to determine dietary intake meant that nutrient content of the diet could not be determined, only intake of foods and food patterns. In order to ensure that participants answered as honestly as possible, they were informed of the benefits of the study to the community at large and encouraged to answer as comprehensively and truthfully as possible. Confidentiality of results was also emphasised.

Intensive training of interviewers and coders; standardization of interviewing techniques and questionnaires; and pretesting of questionnaires prior to the main surveys were all implemented in an effort to obtain the most valid and reliable information possible using the chosen tools and techniques.

5.2.3 Anthropometrical measurements

It is possible that mothers were more likely to bring their children to participate in the study if they were ill (a free medical examination was performed), which could have had an impact on the results related to growth status that were obtained.

5.3 Dietary intake

Dietary intake for children younger than two was compared to similar studies in terms of breastfeeding; introduction of solids; formula feeding; as well as reported usual intake of different food groups; food items and macronutrients.

Dietary intake for children older than two, as well as for adults, was compared to similar studies in terms of reported usual intake of different food groups; food items and macronutrients.

Results for dietary intake in the children will be discussed first, whereafter the dietary intake amongst the adults will be discussed.

5.3.1 Children younger than two years

Data for children younger than two years was mostly compared to the results of the National Food Consumption Survey (NFCS) (Labadarios et al., 2005:533; Nel & Steyn, 2002:48, 59, Table 23)..

5.3.1.1 Breastfeeding

It should be kept in mind that the amount of total participants younger than two years of age was small and further separating data into age groups (e.g. 1-3 age group, etc.) was not feasible.

About one-third (31,24%) of rural children were breastfed at the time of the study and 40,7% of urban children. These percentages are higher than that found in the Free State group of children from the NFCS (1–3 age group), who reported a second highest percentage of children (13%) who were still being breastfed at the time of the study (Northern Cape was first with 19%)(MacIntyre & Labadarios, 2000:320;391, Table 6.13).

Almost all rural participants were previously breastfed (90,9%) as compared to only 37,5% of urban participants. In comparison, the SADHS of 1998 found that 84,3% of their urban- and 89% of their rural participants were ever breastfed. Of the Free State participants in the SADHS, 89,5% were ever breastfed (DoH et al., 2002:133, Table 8.1). The NFCS reported that 89% of Free State participants were previously breastfed (MacIntyre & Labadarios, 2000:392, Table 6.16). However, the NFCS made no distinction between rural and urban areas.

The longest period a rural child was previously breastfed was 54 weeks (1 year) (median=16 weeks/4 months) and the shortest was three weeks. The longest period an urban child was previously breastfed was 76 weeks (1½ years) (median=24 weeks/6 months) and the shortest was two weeks. From this data one might be led to deduce that urban children were breastfed for longer periods compared to rural children, but sample sizes of children younger than two years were small, which makes it difficult to interpret results. The NFCS found that 55% of all urban participants were previously breastfed for more than a year as opposed to 71% of all rural participants (MacIntyre & Labadarios, 2000:394, Table 6.20). The NFCS also found that 64% of all Free State participants (rural and urban) were previously breastfed for more than a year (MacIntyre & Labadarios, 2000:393-394, Table 6.19).

When looking at the data for exclusive breastfeeding (both for children previously breastfed and children breastfeeding at the time of the study), 32 weeks (8 months) was the longest period of exclusive breastfeeding for both rural and urban participants. The median period of exclusive breastfeeding for rural participants was 11 weeks and for urban participants 12 weeks (3 months), which would lead one to assume that, in general, solids were introduced by three months. Again, sample sizes were small, which makes results difficult to interpret. However, the recommended age for introduction of solids is six months of age (WHO, 2002:2). The WHO (2002:2) recommends introduction of “nutritionally adequate, safe and appropriate complementary foods, in conjunction with continued breastfeeding” after six

months of age. The South African Food-Based Dietary Guidelines (SAFBDG) for children recommend that breastfeeding is best for the first six months of life (Bourne, 2007:227).

Dewey (2006:168) indicated that promotion of exclusive breastfeeding for the first six months of life may promote weight loss in mothers, especially considering the high rates of obesity among women in many countries. By only introducing solids at six months in developing countries, the risk for gastrointestinal illness in infants is reduced. Furthermore, it can be beneficial for motor development in the infant if solids are only introduced after six months. No benefit was found in introducing solids at an earlier age. Parsons et al. (1999:S21), in their systematic review, found that that there was no relationship between the type of infant feeding; the duration of breast feeding; or the age when solids were introduced, with obesity in adulthood. However, Wilson et al. (1998:21, 24) found that children who received solids before 15 weeks of age had a higher body fat percentage at age seven years.

5.3.1.2 Formula feeding

The small sample size of rural children younger than two years was too small to draw any conclusions from the results. Two-thirds (63,3%) of urban participants used enough formula that was also correctly prepared (Table 4.8). A large percentage of participants, however, used formula that was incorrectly prepared or given in insufficient amounts.

About a third (31,3%) of rural children and 38,9% of urban children were exclusively formula fed at the time of the current study. A further 16,7% of urban children received mix feeding (breast and bottle). In the 1998 SADHS, 28% of infants younger than 5 months were receiving formula feeds together with breast milk (DoH et al., 2002:138). In comparison, only three percent of children in the NFCS were receiving infant formula (Labadarios & Nel, 2000:396, Table 6.23). It should, however, be kept in mind that the age range for the NFCS only started at one years old.

Nestlé Pelargon® was the most frequently used formula milk among urban carers (63,3%). If it is considered that forty percent of the urban carers indicated that they received their supply of milk from the clinic, it is understandable that Nestlé Pelargon® should be the predominant feed, since it formed part of the NSP at the time of the study. The predominant feeds in the NFCS were Nan® (25%), Nespray® (11%); Lactogen® (10%) and Lactogen 2® (10%) (MacIntyre & Labadarios, 2000:321,396, Table 6.24).

There is some concern regarding the high intake of coffee creamer, which might indicate that it was possibly used as a milk substitute.

5.3.1.3 Introduction of solids

Food groups mostly consumed in adequate- or more than adequate amounts by most children younger than two years of age were: fats and oils; sweets and sugar, and bread and cereals; reflecting that these would be the food groups to be introduced first as solids to these children, together with some meat and meat products and vegetables, and to a lesser extent fruit, legumes and soy. Milk and milk products were also more likely to be consumed in adequate- or more than adequate amounts, probably because these foods were still given as a staple food at this age, together with porridge; sugar and tea.

The WHO also states that all children need complementary foods from six months of age (WHO, 2010:7,27,35). The SAFBDG for children recommend that from six months small amounts of solid foods should be given to children (Bourne, 2007:228, Table 1). The median age of introduction of solids was 24 weeks (6 months) for rural participants and 19 weeks (+5 months) for urban participants (Table 4.7). The age of introduction of solids in rural children in the present study therefore complied with the WHO guidelines, but urban children were weaned too early.

5.3.2 Reported usual intake of different food items

Intake of different food items for both adults and children was obtained by means of a food frequency questionnaire listing 26 different food items. Results were compared to various studies, which will be discussed in more detail below. Results were also compared to the report of Nel and Steyn (2002:48-103, who conducted a secondary data-analysis of existing dietary databases obtained from surveys undertaken in South Africa between 1983 and 2000. They included data from the following studies that used the 24 hour recall method: National Food Consumption Survey (NFCS); Lebowa study; Dikgale study; Black Risk Factor study (BRISK) and Coronary Risk Factor study (CORIS). They also included data from the following studies that used the food frequency questionnaire method: NFCS; the Transition and Health during Urbanisation of South Africans Study (THUSA); First Year Female Students Project (FYFS): and Weight and Risk Factor Study (WRFS).

5.3.2.1 Children younger than two years

Frequency of consumption of certain food items in children younger than two years was determined for four rural boys, 16 urban boys, 16 rural girls and 18 urban girls (the rest were still being breastfed or formula fed). Table 5.1 sets out the current study's results as compared to the results from various other studies. However, it must be kept in mind that there were small sample sizes of children younger than two years in this study, and results should therefore be interpreted with caution.

The following studies' data for one-to-five years old age groups were used for comparison to the current study's data for children younger than two years: the BRISK study conducted between 1983 and 1990 in the urban Western Cape (Nel & Steyn, 2002:103); the Lebowa study conducted in 1991 in the rural Limpopo province (previously named Northern Province) (Nel & Steyn, 2002:97); and the national results of the NFCS (Labadarios et al., 2005:533; Nel & Steyn, 2002:48). The study conducted by Faber and Benadé in 2000 in rural Kwa-Zulu Natal was the only study used as comparison where the age group was limited to between four and 24 months (Faber and Benadé, 2001).

Nel and Steyn (2002) conducted a meta-analysis of all dietary intake studies conducted in South Africa between 1983 and 1990 and their extrapolations were also noted in Table 5.1 for comparison with the current data. It should be noted that the data from the national NFCS was used exactly as is for Nel and Steyn's (2002) national meta-analysis data and this should be kept in mind when reference is made to the national NFCS data in this discussion. However, for the rural and urban sections of the meta-analysis, data was extrapolated from more than one reference source (e.g. BRISK, Lebowa and NFCS) and this is the reason why that data is different. The BRISK and Lebowa studies used the 24 hour recall as their measuring tool, while the NFCS used both the 24 hour recall and the food frequency questionnaire.

Table 5.1 Comparison of ten most frequently consumed food items with ranked results from other studies - children <2 years old

Reference source					*Nel & Steyn, 2002:103*	*Nel & Steyn, 2002:97*	*Faber and Benadé, 2001:364, Table 3)*	*Labadarios et al., 2005:533; Nel & Steyn, 2002:48,59, Table 23*	*Nel & Steyn, 2002:48, 59-60, Table 23*	*Nel & Steyn, 2002:66,75-76, Table 26*	*Nel & Steyn, 2002:80,89-90, Table 29*
Study	***AHA-FS (Current study)***				***BRISK***	***Lebowa***		***NFCS***	***Meta-analysis***	***Meta-analysis***	***Meta-analysis***
Time period of data	***2006***		***2009***		***1983-1990***	***1991***	***2000***	***1999***	***1983-2000***	***1983-2000***	***1983-2000***
Ethnic group	***Mostly black***				***Black***	***Black***	***Black***	***All***	***All***	***All***	***All***
Age groups (years)	***0-2***				***1 - 5***	***1 - 5***	***4-24 months***	***1 - 5***	***1-5***	***1-5***	***1-5***
Area	***Free State - Rural boys***	***Free State - Rural girls***	***Free State - Urban boys***	***Free State - Urban girls***	***Western Cape - urban***	***Northern Province (Limpopo) - rural***	***KwaZulu-Natal - rural***	***South Africa - all***	***South Africa - all***	***South Africa -rural***	***South Africa - urban***
Method used (24 hr recall or FFQ)	***FFQ***				***24 hr***	***24 hr***	***24 hr***	***Both***	***Both***	***Both***	***Both***
Bread and cereals											
Bread	7	6	7	7	8 & 9	4 & 8		5 & 7	5 &7	5 & 8	5 & 8
Cake/biscuits	12			12							
Chips/crisps	19	4	11	15							
Porridge cooked	4	2	2	1	1	1	2 (soft) & 6 (stiff)	1	1	1	2
Potatoes					6			9	9	7	9
Rice					4		5	6	6	6	6
Milk and milk products											
Full cream milk	3	18	8	6	5		8	4	4	4	3
Meat, meat products and other protein											
Chicken		10						10	10		
Eggs			9	8		7					

Table 5.1 Comparison of ten most frequently consumed food items with ranked results from other studies - children <2 years old (continued)

Reference source					*Nel & Steyn, 2002:103*	*Nel & Steyn, 2002:97*	*Faber and Benadé, 2001:364, Table 3)*	*Labadarios et al., 2005:533; Nel & Steyn, 2002:48,,59, Table 23*	*Nel & Steyn, 2002:48, 59-60, Table 23*	*Nel & Steyn, 2002:66,75-76, Table 26*	*Nel & Steyn, 2002:80,89-90, Table 29*
Study	***AHA-FS (Current study)***				***BRISK***	***Lebowa***		***NFCS***	***Meta-analysis***	***Meta-analysis***	***Meta-analysis***
Time period of data	***2006***		***2009***		***1983-1990***	***1991***	***2000***	***1999***	***1983-2000***	***1983-2000***	***1983-2000***
Ethnic group	***Mostly black***				***Black***	***Black***	***Black***	***All***	***All***	***All***	***All***
Age groups (years)	***0-2***				***1 - 5***	***1 - 5***	***4-24 months***	***1 - 5***	***1-5***	***1-5***	***1-5***
Area	***Free State - Rural boys***	***Free State - Rural girls***	***Free State - Urban boys***	***Free State - Urban girls***	***Western Cape - urban***	***Northern Province (Limpopo) – rural***	***KwaZulu-Natal - rural***	***South Africa - all***	***South Africa - all***	***South Africa -rural***	***South Africa - urban***
Method used (24 hr recall or FFQ)	***FFQ***				***24 hr***	***24 hr***	***24 hr***	***Both***	***Both***	***Both***	***Both***
Fruit and vegetables											
Fruit	8	3	4	9							
Fruit juice				10							
Legumes							4				
Pumpkin							9				
Vegetables	9	7	10	5							
Wild green leaves						6				9	
Fats and oils											
Gravy											
Margarine/ oil/ fat	5	8	5	3	3		1 & 7	8	8	10	7
Non-dairy creamer (Cremora)	11					5					
Other											
Cool drinks	10	13			10						10
Salt/ stock	6	9	6	4							
Sugar	1	1	3	18	2	2	3	2	2	2	1
Tea	2	5	1	2	7	3	10	3	3	3	4

(i) Breads and cereals

When describing dietary intake according to foods most frequently consumed, cooked porridge was consumed frequently by all children in the current study; similar to rural children in the Lebowa study (Nel & Steyn, 2002:97); rural children in the meta-analysis study of Nel and Steyn (2002:66,75-76); rural children in the study by Faber and Benadé (2001:364, Table 3); urban children in the meta-analysis by Nel and Steyn (2002:80, 89-90); urban children in the BRISK study (Nel & Steyn, 2002:103); and all children in both the NFCS (Labadarios et al., 2005:533) and the meta-analysis by Nel and Steyn (2002: 48, 59-60).According to the SAFBDG for children from one to seven years, starchy foods should form the basis of most meals (Bourne, 2007:228, Table 1). Children younger than two years in the current study did consume porridge, a starch, as the basis of most of their meals. Cooked porridge was one of the most frequently introduced solids to this age group.

For bread intake, the current study's findings were similar to the urban BRISK study (Nel & Steyn, 2002:103); the rural children in the Lebowa study (Nel & Steyn, 2002:97); the NFCS (Labadarios et al., 2005:533) and rural meta-analysis (Nel & Steyn, 2002:66, 75-76, Table 26); the urban meta-analysis (Nel & Steyn, 2002:66, 75-76); and the national meta-analysis (Nel & Steyn, 2002:80, 89-90, Table 29). Bread is a also starchy food, which should form the basis of most meals as recommended by the SAFBDG (DoH, 2004:12; Vorster & Nell, 2001:S17). Therefore, most children in the current study complied with this guideline. The dietary reference intakes also recommend that carbohydrates should form 45% to 65% of a child's energy intake after the age of one year. By consuming bread and cooked porridge frequently, this recommendation was complied with (Food & Nutrition Board et al., 2002:1324).

(ii) Fats and oils

In this study, margarine, oil and fat were used frequently by rural and urban boys and urban girls. In comparison, they were also used often among urban children in the BRISK study (Nel & Steyn, 2002:103). Oil was listed as the first item in the study by Faber and Benadé (2001:364, Table 3), and margarine was listed as seventh in the same study.

Rural girls consumed chips and crisps 4th most frequently and urban boys consumed chips and crisps 11th most frequently. None of the other studies found this food item to be consumed within the ten most frequently consumed food items.

Rural boys in the current study consumed non-dairy creamer more frequently (11th) than the other groups. The only study that found non-dairy creamer to be frequently consumed, was the rural Lebowa study (Nel & Steyn, 2002:97), where it was found to be the fifth most frequently consumed food item.

In the current study, caregivers sometimes referred to non-dairy-creamer as milk, which may partially explain why non-dairy creamer was found as a frequently consumed food item on further investigation. Similar results were reported by Faber et al. (2001:408).This finding is of concern, due to the fact that non-dairy creamer may have been given to children younger than two years of age as a milk substitute.

Even though fats and oil were consumed frequently, it was not to say that they were consumed in large amounts, since amounts were not quantified in the food frequency questionnaire. According to dietary reference intakes, children younger than two years can consume between thirty to forty percent of energy from fats per day, therefore frequently consumption of margarine, oil and fats are necessary to meet this recommendation (Food & Nutrition Board et al., 2002:1324). There are no recommendations in the SAFBDG for children with regards to fat intake.

(iii) Fruit and vegetables

Fruit was more frequently consumed among urban- than rural boys and rural- than urban girls in the current study. Fruit was not listed among the top ten in any of the studies used for comparison.

The SAFBDG recommend that vegetables and fruit should be given to children daily, but no serving amounts are specified (Bourne, 2007:228, Table 1). Frequent consumption of fruit among children in the current study did, however, meet this requirement.

(iv) Milk and milk products

Full cream milk was consumed frequently by this study's rural boys and urban girls in this study, as was the case among urban children in the meta-analysis by Nel and Steyn (2002:80,89-90, Table 29); urban children in the BRISK study (Nel & Steyn, 2002:103); the national NFCS (Labadarios et al., 2005:544) and the rural meta-analysis (Nel & Steyn, 66, 75-76, Table 26). The current study's urban boys consumed full cream milk a little less frequently (8^{th}), but this results compares with the findings of the study by Faber and Benadé (2001:364, Table 3).

When looking at the SAFBDG recommendations for children which states that children need to drink milk daily (Bourne, 2007:228, Table 1), the current study's participants mostly complied with this guideline.

(v) Sweets and sugar

In the current study, sugar was consumed frequently, similar to the rural and urban children in the meta-analysis of Nel & Steyn (2002: 66, 75-75, Table 26, 80, 89-90); the rural children in the study by Faber and Benadé (2001:364, Table 3), the BRISK (Nel & Steyn, 2002:103), Lebowa (Nel & Steyn, 2002:97), and NFCS (Labadarios et al., 2005, 533).

The frequent consumption of sugar occurred most often added to cooked porridge and tea. However, the SAFBDG for children recommend that sugar should be offered in small amounts with meals (Bourne, 2007:228, Table 1). There are concerns that if sugar is consumed in excess it may displace other foods that are good sources of vitamins and minerals in the diet (Van den Berg, 2011:260; DoH, 2004:32). However, the current study's results only found that sugar was consumed frequently and not what the quantities of consumption were.

Various articles state that consumption of too much sugar can contribute to dental caries (DoH, 2004:33; Lucas, 2004a:278; Touger-Decker, 2004:670-671; Vorster & Nell, 2001:S23). However, a

systematic review concluded that frequent brushing with fluoride toothpaste and good dental care prevents dental caries more effectively than avoiding sugary foods (Van den Berg, 2011:258).

(vi) Tea

Results for tea consumption were similar to consumption by: rural children in the Lebowa study (Nel & Steyn, 2002:97); rural children in the meta-analysis (Nel & Steyn, 2002:80, 89-90, Table 26). the NFCS (Labadarios, et al., 2005:533; Nel & Steyn, 2002:48); and the urban children in the meta-analysis (Nel & Steyn, 2002:80, 89-90, Table 29).

The frequent intake of tea, especially during meal times, can affect iron status, since tea inhibits the absorption of iron (Faber et al., 2001:409). Also, since tea was reported as a frequently consumed fluid more often than milk in some instances, it can be assumed that tea replaced milk in the diet of some children. However, when looking at intake of different food groups (Table 4.11 and Table 4.12), all rural boys, 68,8% of urban boys, and fifty percent of urban girls took within the recommended number of servings of milk and milk products.

(vii) Conclusion

Tea, cooked porridge, margarine/oil, salt/stock, bread, fruit, and vegetables were all ranked in the top ten items consumed by both rural and urban boys younger than two years old. Rural boys consumed full cream milk more often than boys, whereas urban boys consumed more fruit than rural boys. Rural boys also consumed coffee creamer and cakes/biscuits more often than urban boys.

Cooked porridge, tea, margarine/oil, salt/stock, vegetables, bread, and fruit were within the top ten items consumed by both rural and urban girls younger than two years old. Rural girls consumed fruit and chips/crisps more often than urban girls, whereas urban girls consumed full cream milk more often than rural girls.

5.3.2.2 Children older than two years

Frequently consumed food items were determined for 26 rural boys, 64 urban boys, 24 rural girls and 48 urban girls. Table 5.2 sets out the current study's results as compared to the results from various other studies. The following studies were used for comparison to the data for children older than two years: the BRISK study conducted between 1983 and 1990 in the urban Western Cape (Nel & Steyn, 2002:103); the Lebowa study conducted in 1991 in the rural Limpopo province (previously named Northern Province) (Nel & Steyn, 2002:97); the study by MacKeown et al. (1998:307) conducted in the urban area of Johannesburg/Soweto and rural area of previously-named Bophuthatswana in 1984; the national results of the NFCS (Labadarios et al., 2005:533; Nel & Steyn, 2002:48); and the results of the NFCS for the Free State (MacIntyre & Labadarios, 2000). The results of Nel and Steyn's meta-analysis (2002) were also included.

The BRISK and Lebowa studies used the 24 hour recall as their measuring tool, while the NFCS used both the 24 hour recall and the food frequency questionnaire. The current study used a single 24 hour recall of reported usual intake.

Table 5.2 Comparison of ten most frequently consumed food items with ranked results from other studies - children >2 years old

Reference source					*MacIntyre & Labadarios,2000: 469,Table 6.122*	*Nel & Steyn, 2002:103*		*Nel & Steyn, 2002:97*		*MacKeown et al., 1998:307*	*Faber et al., 2001:407, Table 4*	*Labadarios et al., 2005:533; Nel & Steyn, 2002:48,66***		*Nel & Steyn, 2002:48, 59-60, Table 23*		*Nel & Steyn, 2002:66,75-76, Table 26*		*Nel & Steyn, 2002:80,89-90, Table 29*	
Study	***AHA-FS (Current study)***				***NFCS***	***BRISK***		***Lebowa***		***Birth-to-Ten (1995)***		***NFCS***		***Meta-analysis***		***Meta-analysis***		***Meta-analysis***	
Time period of data	***2006***		***2009***		***1999***	***1983-1990***		***1991***		***1984 & 1995***	***2000***	***1999***		***1983-2000***		***1983-2000***		***1983-2000***	
Ethnic group	***Mostly black***				***All***	***Black***		***Black***		***All***	***Black***	***All***		***All***		***All***		***All***	
Age groups (years)	***2 - 7***				***1-9***	***1 - 5***	***6 - 9***	***1 - 5***	***6 - 9***	***4 - 5***	***2-5***	***1 - 5***	***6 - 9***	***1-5***	***6-9***	***1-5***	***6-9***	***1-5***	***6-9***
Area	***Free State - Rural boys***	***Free State - Rural girls***	***Free State - Urban boys***	***Free State - Urban girls***	***Free State - all***	***Western Cape - urban***		***Northern (Limpopo) Province - rural***		***Gauteng - all (1984), urban only (1995)***	***KwaZulu-Natal - rural***	***South Africa - all***		***South Africa - all***		***South Africa - rural***		***South Africa - urban***	
Method used (24 hr or FFQ)	***FFQ***				***Both***	***24 hr***		***24 hr***		***24 hr (1984) & FFQ (1995)***	***Both***	***Both***		***Both***					
Bread and cereals																			
Bread	9	7	8	8		8 & 9	4 & 6	4 & 8	4 & 9	5	3	5 &7	4 &6	5 &7	4 &6	5 &8	4 &5	5 &8	5 &6
Cake/biscuits	3	11								-									
Porridge, cooked	2	2	1	1	1	1	3	1	1	3	1,7	1	2	1	2	1	1	2	2
Potatoes					5	6	5				8	9	9	9	9	7	8	9	
Rice						4					5	6	8	6	8	6	7	6	8
Samp							9												
Milk and milk products																			
Full cream milk	4	3	5	5	2	5	7			2		4	5	4	5	4	9	3	3
Meat, meat products and other protein																			
Chicken					7							10	10	10	10				10
Eggs					8			7	10										
Fats and oils																			
Gravy							10												
Margarine/ oil/ fat	8	6	6	4		3	2					8	7	8	7	10	6	7	7
Non-dairy creamer (Cremora)								5	5		9						10		

Table 5.2 Comparison of ten most frequently consumed food items with ranked results from other studies' data - children >2 years old (continued)

Reference source					*MacIntyre & Labadarios,2000: 469,Table 6.122*	*Nel & Steyn, 2002:103*		*Nel & Steyn, 2002:97*		*MacKeown et al., 1998:307*	*Faber et al., 2001:407, Table 4*	*Labadarios et al., 2005:533; Nel & Steyn, 2002:48,66***		*Nel & Steyn, 2002:48, 59-60, Table 23*		*Nel & Steyn, 2002:66,75-76, Table 26*		*Nel & Steyn, 2002:80,89-90, Table 29*	
Study	*AHA-FS (Current study)*				*NFCS*	*BRISK*		*Lebowa*		*Birth-to-Ten (1995)*		*NFCS*		*Meta-analysis*		*Meta-analysis*		*Meta-analysis*	
Time period of data	*2006*		*2009*		*1999*	*1983-1990*		*1991*		*1984 & 1995*	*2000*	*1999*		*1983-2000*		*1983-2000*		*1983-2000*	
Ethnic group	*Mostly black*				*All*	*Black*		*Black*		*Black*	*Black*	*All*		*All*		*All*		*All*	
Age groups (years)	*2 - 7*				*1-9*	*1 - 5*	*6 - 9*	*1 - 5*	*6-9*	*4 - 5*	*2-5*	*1 - 5*	*6 - 9*	*1-5*	*6-9*	*1-5*	*6-9*	*1-5*	*6-9*
Area	*Free State - Rural boys*	*Free State - Rural girls*	*Free State - Urban boys*	*Free State - Urban girls*	*Free State - all*	*KwaZulu-Natal - rural*		*Northern (Limpopo) Province - rural*		*Gauteng - all*	*KwaZulu-Natal - rural*	*South Africa - all*		*South Africa - all*		*South Africa - rural*		*South Africa - urban*	
Method used (24 hr or FFQ)	*FFQ*				*Both*	*24 hr*		*24 hr*		*24 hr (1984) & FFQ (1995)*	*Both*	*Both*		*Both*					
Fruit and vegetables																			
Banana											10								
Cabbage					4				7		6								
Fruit	7	10	7	7															
Fruit juice				10															
Legumes											4								
Tomato and onion sauce									6										
Vegetables			9	9															
Wild green leaves					9 & 10			6	8							9			
Other																			
Cool drinks	10	9	10			10				4								10	9
Salt/ stock/ Royco	5	4	3	2															
Sugar	1	1	2	3	3	2	1	2	2			2	1	2	1	2	2	1	1
Tea	6	5	4	6	6	7	8	3	3	1	2	3	3	3	3	3	3	4	4

(i) Breads and cereals

When describing the dietary intake according to foods most frequently consumed, both urban boys and girls consumed cooked porridge (including both maize meal- and sorghum porridge) as their first most frequently consumed food item and both rural boys and girls consumed it as their second most frequently consumed food item.

Maize porridge was also consumed most frequently among urban one-to-five-year olds in the BRISK study (Nel & Steyn, 2002:103); rural one-to-five-year olds in the Lebowa study (Nel & Steyn, 2002:97); rural one-to-five-year olds in the meta-analysis study conducted by Nel & Steyn (2002:66,75-76, Table 26); rural six-to-nine-year olds in the Lebowa study (Nel & Steyn, 2002:97); and rural six-to-nine-year olds in the meta-analysis study (Nel & Steyn, 2002:66,75-76, Table 26).

Maize porridge was also most frequently consumed by the Free State data group of the NFCS (MacIntyre & Labadarios, 2000:469, Table 6.122) and the one-to-five-year olds nationally in the NFCS and meta-analysis study (Labadarios et al., 2005:533; Nel & Steyn, 2002:48,59-60, Table 23).

Maize porridge was ranked as the second most frequently consumed food item among both the urban one-to-five-year old and the six-to-nine-year old groups in the meta-analysis (Nel & Steyn, 2002:80,89-90, Table 29) as well as the national group of six-to-nine-year-olds of the NFCS (Labadarios et al., 2005:533; Nel & Steyn, 2002:48,59-60, Table23). Faber and Benadé, 2001:364, Table 3) found that soft maize porridge was eaten second most frequently and stiff maize porridge sixth most frequently among their 4-to-24-month old rural children.

Maize porridge was consumed third most frequently among urban six-to-nine-year olds in the BRISK study (Nel & Steyn, 2002:103) as well as four-to-five-year olds in the study of MacKeown et al. (1998:307).

These findings reflect that porridge is the staple food of the majority of children older than two years, and is in compliance with SAFBDG for children, which states that starches (of which porridge forms part) should form the basis of the main meals for children (Bourne, 2007:228, Table 1).

Bread also fell within the top ten most frequently consumed items, as was found by most of the other studies, except for the NFCS. Bread is a starchy food, which should form the basis of most meals as stated by the SAFBDG for children (Bourne, 2007:228, Table 1). Frequent intake of bread (preferably brown bread) is necessary to meet this guideline.

(ii) Fats and oil

When describing the dietary intake according to foods most frequently consumed, margarine, oil or fat was a frequently consumed food item, especially amongst rural and urban girls and urban boys. In comparison, it was the third most frequently consumed item among urban one-to-five-year olds in the BRISK study, and second among urban six-to-nine-year olds in the BRISK study (Nel & Steyn, 2002:103). Faber and Benadé (2001:364, Table 3) found that oil was the most frequently consumed food item among 4-to-24-month olds and margarine was the seventh most frequently consumed food item.

Urban boys also had a close to significantly more frequent intake of coffee creamer in comparison to rural boys (95% CI [-30;0]).

Frequent consumption of fats and oils does not necessarily mean that they were consumed in excessive amounts. There are no recommendation as to fat intake from the SAFBDG for children. According to the DRIs, children between one and three years may consume between thirty to forty percent of energy from fats per day and children aged four to 18 years old may consume 25-35% of their total energy from fat (Food & Nutrition Board et al., 2002:1324).

However, consuming fats and oil in excessive amounts may lead to weight gain, which may increase the risk for chronic diseases of lifestyle (DoH, 2004:27). However, they are good sources of essential fatty acids and fat-soluble vitamins, which are essential for growth and development in infants (Wolmarans & Oosthuizen, 2001:S48).

A significantly larger percentage of rural boys consumed cake/biscuits/cookies frequently (95% CI [-30;-1]) than urban boys. However, the NFCS found that only 5,3% of their rural one-to-five-year-olds (Nel & Steyn, 2002:.60, Table 23) and 6,9% of their rural six-to-nine-year-olds consumed cookies/biscuits and/or cake (Nel & Steyn, 2002:.61, Table 23). More urban children in the NFCS consumed cookies/biscuits and/or cake, with 7,9% among one-to-five-year-olds (Nel & Steyn, 2002:.89, Table 29) and 11,3% of all six-to-nine-year-olds (Nel & Steyn, 2002: 91, Table 29). Cookies are high in saturated fat and sugar and frequent consumption can lead to weight gain (USDA & USDHHS, 2010:13,24,30; Potischman et al., 2002:946). In addition, the micronutrient content of cookies and biscuits are very low.

(iii) Fruit and vegetables

All children consumed fruit and vegetables seventh most frequently except rural girls (10th ranking). Fruit was consumed almost as frequently as bread among most of the groups, except in the rural girls. Faber et al. (2001:407, Table 4) found in their children in rural Kwa-Zulu Natal that bananas were the tenth item within the top ten most frequently consumed food items. Consumption of fruit in children met the recommendations of the SAFBDG for children, which states that South African children should eat vegetables and fruit daily (Bourne, 2007:228, Table 1). However, frequent consumption does not necessarily mean that sufficient amounts of servings were consumed. Only 12% of urban children consumed five servings or more of fruit and vegetables per day. Frequent consumption of fruit and vegetables is associated with numerous health benefits (Love & Sayed, 2001:S26).

(iv) Milk and milk products

Full cream milk was the third most frequently consumed item by rural girls, the fourth item by rural boys and the fifth item by both urban boys and girls. In the Free State group of the NFCS and in the study conducted by MacKeown et al. (1998:307), it was the second most frequently consumed food item. Nel and Steyn (2002, 80,89-90, Table 29) found it the third most frequently consumed item among urban children between one and nine years old, and it was the fourth most frequently consumed food item nationally among one-to-five-year olds in the NFCS (Labadarios et al., 2005:533; Nel & Steyn, 2002:48), as well as the rural one-to-five year olds in the meta-analysis (Nel & Steyn, 2002, 66, 75-76, Table 26). It was the fifth most frequently consumed food item among urban one-to-

five-year olds in the BRISK study (Nel & Steyn, 2002:103) as well as the six-to-nine-year olds in the national NFCS (Labadarios et al., 2005:533; Nel & Steyn, 2002:48).

The SAFBDG for children recommend that children should drink milk daily (Bourne, 2007:228, Table 1), and frequent consumption of milk in the current study's children possibly ensured that children met this requirement, although the amounts of milk consumed were not specified.

(v) Sweets and sugar

There was a close to significant difference in the frequency of consumption of sugar, with rural boys consuming sugar more frequently than urban boys (95% CI [-30;0]). The significant difference was even closer in girls, with rural girls also consuming sugar more frequently than urban girls (95% CI [-60;0]).

In comparison with other studies, sugar was also consumed most frequently by: urban one-to-five-year olds and six-to-nine-year olds in the meta-analysis by Nel and Steyn (2002:80,89-90, Table 29); as well as six to nine year old urban children in the BRISK study (Nel & Steyn, 2002:103), and six-to-nine-year olds nationally in the NFCS (and meta-analysis) (Labadarios et al., 2005:533; Nel & Steyn, 2002:48,59-60, Table 23).

Sugar was second most frequently consumed by urban one-to-five-year old groups in the BRISK study (Nel & Steyn, 2002:103); as well as both rural one-to-five-year olds and six- to-nine-year-olds in the Lebowa study (Nel & Steyn, 2002:97); the rural one-to-five-year olds and six-to-nine-year olds in the meta-analysis (Nel & Steyn, 2002: 66,75-76, Table 26), and the national NFCS (Labadarios et al., 2005:533; Nel & Steyn, 2002:48).

Sugar was the third most frequently consumed food in the Free State group of children in the NFCS (MacIntyre & Labadarios, 2000:469, Table 6.122) as well as the rural 4-to-24-month old South African children (Faber & Benadé, 2001:364, Table 3).

The frequent consumption of sugar most probably stems from the fact that sugar was frequently added to the frequently consumed cooked porridge and to tea.

What is of more concern in the current study is the frequent consumption of sugar sweetened beverages (cool drinks) among children older than two years, since excessive consumption of sugar-sweetened beverages is associated with weight gain and obesity (Van den Berg, 2011:257). Both rural and urban boys consumed it tenth most frequently. Rural girls consumed it ninth most frequently and urban girls consumed it 14th most frequently.

The SAFBDG for children recommend that sugary treats and drinks should be given sparingly to children and only at meal times (Bourne, 2007:228, Table 1). Replacing soft drinks (that contain sugar) with milk or water, can help with weight management in obese children (Bourne & Seager, 2001:S66).

(vi) Salt

Salt (together with beef stock) was listed as a food item on the FFQ. Results should be interpreted with caution, since only frequent consumption of salt was reported and not the amount of added salt.

Salt was the second most frequently consumed item among urban girls and the third item among urban boys, whereas it was the fourth and fifth item respectively among rural girls and rural boys.

None of the comparison studies investigated salt/stock as a frequently consumed food item, and therefore there were no studies available for comparison. The NFCS found that 57% of Free State respondents used iodized salt in their child's food (MacIntyre & Labadarios, 2000:432, Table 6.80) and 17% used flavoured salts (e.g. Beefstock, Aromat, Bisto, etc.) in their child's food (MacIntyre & Labadarios, 2000:433, Table 6.83).

The SAFBDG (for adults) recommends that salt should be used sparingly (DoH, 2004:30). However, no recommendation is made for children The SAFBDG for children are in the process of being revised.

(vii) Tea

There was a close to significant difference in frequency of tea intake among both boys (95% CI [-30;0]). and girls (95% CI [-30;0]). Tea was consumed more frequently by urban boys (4th) than rural boys (6th) and rural girls consumed tea more frequently (5th) than urban girls (6th). This corresponds well with the Free State group of the NFCS, where tea was ranked sixth (MacIntyre & Labadarios, 2000:469, Table 6.122) and the BRISK study, where it was ranked seventh for one-to-five-year olds, and eighth for six-to-nine-year olds (Nel & Steyn, 2002:103). However, it was more frequently consumed in the Lebowa study and NFCS nationally, where it was ranked third. It was ranked first in the study by MacKeown et al. (1998:307).

(viii) Conclusion

Food groups mostly consumed in adequate- or more than adequate amounts by most children older than two years of age were: sweets and sugar; fats and oils; and bread and cereals, with the fourth most commonly consumed food group being meat and meat products, followed by milk and milk products, reflecting that children tend to consume more meats and less dairy as they age.

These findings agree with data in Tables 4.19 and 4.20, which show that cooked porridge; sugar; full cream milk; tea; bread; and margarine/oil/fat mostly fell within the top ten of the most frequently consumed food items among these children. Intake of cool drinks (which were mostly sugar-sweetened) was also frequently reported.

There were no significant differences between the frequent intake of food items among either rural and urban boys or rural and urban girls, except for a higher frequent median intake of cake/biscuits among rural boys (95%CI [-30;-1]).

A nutrition transition from a traditional diet to a Western diet has occurred in both rural and urban boys and rural and urban girls, when looking at consumption of food groups and the top ten frequently consumed food items.

5.3.2.3 Adults

A ranking of food items most frequently consumed were determined for 161 rural men, 99 urban men, 389 rural women and 320 urban women. Results of the current study were compared with the results

from various studies and summarized in Table 5.3. The following studies were used for comparison: the BRISK study conducted between 1983 and 1990 among black urban residents of the Western Cape aged between three and 60+ years old (Nel & Steyn, 2002:21, Table 4); the Coronary Risk Factor Study (CORIS) conducted in 1979 among white urban adults aged 15 to 64 years in the Western Cape (Steyn et al., 2006:15, Table 2.21; Steyn et al., 2003:636, Table 3; Nel & Steyn, 2002:21, Table 4); the Dikgale study conducted among rural black adults of Northern/Limpopo Province aged 19 years and older (Steyn et al., 2006:15, Table 2.2.1, Steyn, 2005a:35, Table 4.2a; Steyn et al., 2003:636, Table 3; Nel & Steyn, 2002:23, Table 4); the Lebowa study conducted on 19 to 24 year olds in 1991 in the rural Northern (Limpopo) province (Nel & Steyn, 2002:97); the FYFS Project conducted in 1994 among black female aged from 18 to 34 years old in Gauteng (Steyn et al., 2003:636, Table 3; Nel & Steyn, 2002:23, Table 5); the THUSA Study conducted between 1996 and 1998, in the North West Province among urban and rural blacks aged 14 years and older (Steyn et al., 2006:244, Table 2, Vorster et al., 2005:485, Table 4; Steyn et al., 2003:636, Table 3; Nel & Steyn, 2002:23, Table 5); the Weight and Risk Factor (WRFS) study conducted through a postal survey among all South Africans aged between 18 and 55 years old (Steyn et al., 2003:636, Table 3; Nel & Steyn, 2002:23, Table 5).

(i) Breads and cereals

In the current study, cooked porridge was consumed second most frequently by rural men and women, and urban men. Urban women consumed it third most frequently. In comparison, it was found to be the first most frequently consumed food item in: the meta-analysis studies for rural and all South Africans (Nel & Steyn, 2002:62, Table 23, Nel & Steyn, 2002:77, Table 26); the Dikgale study (rural Northern/Limpopo Province)(Nel & Steyn, 2002:101); and the THUSA study (rural women; men and women from farms; men from urban middle- and upper class) (MacIntyre et al., 2002:247-248, Tables 5-6). In the THUSA study, it was the second most frequently consumed food item among all participants overall; as well as in rural men, men and women from informal settlements, and women from urban middle class (MacIntyre et al., 2002:247-248, Tables 5-6).

Eating cooked porridge, as part of starchy foods, is recommended by the SAFBDG, since it may be a good source of fibre and starch, in sufficient quantities. Vorster and Nell (2001:S17) state that there is convincing evidence that foods rich in carbohydrates influence health and prevent CDLs directly via digestion, absorption, fermentation and metabolism; and indirectly by adding micronutrients and phytochemicals to the diet, as well as replacing fat and animal protein in the diet. Vorster & Nell (2001:S17, S20-S23) in their systematic review of the scientific evidence linked to the SAFBDG of “make starchy foods the basis of most meals” state that a high-carbohydrate diet lowers the risk for obesity, DM, CVD, stroke, CHD, cancer and gastrointestinal diseases (Vorster & Nell, 2001:S17,S20-S23). Since it is also a fortified staple food (Labadarios et al., 2005:542), higher intake can be of benefit to an individual’s micronutrient status. However, it is possible to become obese on a low-fat, high-carbohydrate diet, especially if physical activity is also low (Vorster & Nell, 2001:S20).

Table 5.3 Comparison of ten most frequently consumed food items with ranked results from other studies – adults 25 – 64 years

Reference source					*Nel & Steyn, 2002:62, Table 23*	*Nel & Steyn, 2002:77, Table 26*	*Nel & Steyn, 2002:92, Table 29*	*Nel & Steyn, 2002:101*	*Nel & Steyn, 2002:97*	*Nel & Steyn, 2002:103*	*Nel & Steyn, 2002:126-129, Table 37*	*Nel & Steyn, 2002:118*	*MacIntyre et al., 2002:247-248, Table 5 & Table 6*								*Nel & Steyn, 2002:110*	*Nel & Steyn, 2002:122*
Study	*AHA-FS (Current study)*				*Meta-analysis*			*Dikgale*	*Lebowa*	*BRISK*	*CORIS*	*FYFS*	*THUSA*									*WRFS*
Time period of data	*2006*		*2009*		*1983-2000*			*1998*	*1991*	*1983-1990*	*1979 &1983*	*1994*	*1996 - 1998*									
Ethnic group	*Mostly black*				*All*			*Black*	*Black*	*Black*	*White*	*Black*	*Black*									*All*
Age groups (years)	*25-64*				*≥10*			*>19*	*19 - 24*	*25-60+*	*≥25*	*≥25*	*14-60+*									*≥25*
Area	*Rural*		*Urban*		*South Africa - all*	*South Africa - rural*	*South Africa - urban*	*Northern (Limpopo) Province - rural*	*Northern Province (Limpopo) - rural*	*Western Cape (Cape Town) - urban*	*Western Cape - urban and rural*	*Gauteng and Northern_ Province - urban and rural*	*North West - rural*		*North West - farm*		*North West - informal settlement*		*North West - middle class urban*		*North West - upper class urban*		*North West - urban and rural*	*South Africa*
Method used (24 hr recall or FFQ)	*FFQ*				*Both*			*24 hr*		*24 hr*	*24 hr*	*FFQ*	*FFQ*										*FFQ*
	♂	♀	♂	♀									♂	♀	♂	♀	♂	♀	♂	♀	♂	♀		
Bread and cereals																								
Baked wheat												5												
Bread	8	7	8	6	4&5	4&7	4&5	4	4& 6	3	7&10	1	7	8	6	7	9	9	9	6	8		4&5	
Porridge, cooked	2	2	2	3	1	1	2	1		10		4	2	1	1	1	2	2	1	2	1	6	2	
Rice							10			5	9	3	6	4	3	3	4	4	3	4	2	3	3	1
Samp														10									6	
Vetkoek																							7	
Milk and milk products																								
Cheese																								8
Full cream milk	5	6	7	7	9		6			2	3		10		5					10	6	5		
Legumes																								
Beans (legumes)								6																
Fats and oils																								
Margarine/ oil/ fat	8	5	6	5	7		7			4	5		3& 8	6& 9	4	6& 9	5& 8	5	5 & 7	3 & 7	9	4& 10		
Non-dairy creamer (Cremora)	14	14	24	23	6	5		5	5															

Table 5.3 Comparison of ten most frequently consumed food items with ranked results from other studies – adults 25 – 64 years (continued)

Reference source					*Nel & Steyn, 2002:62, Table 23*	*Nel & Steyn, 2002:77, Table 26*	*Nel & Steyn, 2002:92, Table 29*	*Nel & Steyn, 2002:101*	*Nel & Steyn, 2002:97*	*Nel & Steyn, 2002:103*	*Nel & Steyn, 2002:126-129, Table 37*	*Nel & Steyn, 2002:118*	*MacIntyre et al., 2002:247-248, Table 5 & Table 6*								*Nel & Steyn, 2002:110*	*Nel & Steyn, 2002:122*
Study	***AHA-FS (Current study)***				***Meta-analysis***			***Dikgale***	***Lebowa***	***BRISK***	***CORIS***	***FYFS***	***THUSA***										***WRFS***
Time period of data	***2006***		***2009***		***1983-2000***			***1998***	***1991***	***1983-1990***	***1979 &1983***	***1994***	***1996 - 1998***										
Ethnic group	***Mostly black***				***All***			***Black***	***Black***	***Black***	***White***	***Black***	***Black***										***All***
Age groups (years)	***25-64***				***≥10***			***>19***	***19 - 24***	***25-60+***	***≥25***	***≥25***	***14-60+***										***≥25***
Area	***Rural***		***Urban***		***South Africa - all***	***South Africa - rural***	***South Africa - urban***	***Northern (Limpopo) Province - rural***	***Northern Province (Limpopo) - rural***	***Western Cape (Cape Town) - urban***	***Western Cape - urban and rural***	***Gauteng and Northern_ Province - urban and rural***	***North West - rural***		***North West - farm***		***North West - informal settlement***		***North West - middle class urban***		***North West - upper class urban***		***North West - urban and rural***	***South Africa***
Method used (24 hr recall or FFQ)	***FFQ***				***Both***			***24 hr***		***24 hr***	***24 hr***	***FFQ***	***FFQ***										***FFQ***
	♂	♀	♂	♀									♂	♀	♂	♀	♂	♀	♂	♀	♂	♀		
Fruit and vegetables																								
Apple													9				7	8	8		7	7		
Banana																10		10						
Cabbage						10			8					5		4	10	7		8				
Fruit	9	8	10	9								7,8												
Fruit juice																								3,4,5,6
Maize									1															
Onion													4		7	5	3	3	4	5	4	1		
Potatoes					10		9			6	6													10
Pumpkin																						8		
Tomato													5	7		8	6	6	6	9	10	9		
Tomato and onion sauce						8		7	7					3										
Vegetables	10	9	9	8																				
Wild green leave						6		8	11															
Meat, meat products and other protein																								
Beef												9												7
Boerewors																					5			
Chicken					8	9			9			10												
Eggs									10															9

Table 5.3 Comparison of ten most frequently consumed food items with results from other studies – adults 25 – 64 years (continued)

Reference source					*Nel & Steyn, 2002:62, Table 23*	*Nel & Steyn, 2002:77, Table 26*	*Nel & Steyn, 2002:92, Table 29*	*Nel & Steyn, 2002:101*	*Nel & Steyn, 2002:97*	*Nel & Steyn, 2002:103*	*Nel & Steyn, 2002:126-129, Table 37*	*Nel & Steyn, 2002:118*	*MacIntyre et al., 2002:247-248, Table 5 & Table 6*									*Nel & Steyn, 2002:110*	*Nel & Steyn, 2002:122*
Study	***AHA-FS (Current study)***				***Meta-analysis***			***Dikgale***	***Lebowa***	***BRISK***	***CORIS***	***FYFS***	***THUSA***										***WRFS***
Time period of data	***2006***		***2009***		***1983-2000***			***1998***	***1991***	***1983-1990***	***1979 &1983***	***1994***	***1996 - 1998***										
Ethnic group	***Mostly black***				***All***			***Black***	***Black***	***Black***	***White***	***Black***	***Black***										***All***
Age groups (years)	***25-64***				***≥10***			***>19***	***19 - 24***	***25-60+***	***≥25***	***≥25***	***14-60+***										***≥25***
Area	***Rural***		***Urban***		***South Africa - all***	***South Africa - rural***	***South Africa - urban***	***Northern (Limpopo) Province - rural***	***Northern Province (Limpopo) - rural***	***Western Cape (Cape Town) - urban***	***Western Cape - urban and rural***	***Gauteng and Northern_ Province - urban and rural***	***North West - rural***		***North West - farm***		***North West - informal settlement***		***North West - middle class urban***		***North West - upper class urban***		***North West - urban and rural***	***South Africa***
Method used (24 hr recall or FFQ)	***FFQ***				***Both***			***24 hr***		***24 hr***	***24 hr***	***FFQ***	***FFQ***										***FFQ***
	♂	♀	♂	♀									♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	
Meat, meat products and other protein (continued)																							
Fish																						8	2
Meat & offal																							
Mutton											8												
Savoury sausage																		10					
Other																							
Alcohol																						1	
Atchar												6											
Coffee	6	10	5	22	13		8			7	1												
Cool drinks	11	11	11	10						9													
Salt/ stock/ Royco	3	4	4	4																			
Sugar	1	1	1	1	2	2	1	3	2	1	2	2	1	2	2	2	1	1	2	1	3	2		
Tea	4	3	3	2	3	3	3	2	3	8	4											9& 10	

(ii) Fats and oils

Margarine, oil and fat were frequently consumed, but the amounts consumed were not determined. Rural and urban women both consumed margarine/oil/fat fifth most frequently. The BRISK study (Nel & Steyn, 2002:103) found it to be consumed fourth most frequently. The CORIS study (Nel & Steyn, 2002:126-129, Table 37) also found it to be the fifth most frequently consumed item. The THUSA study found it to be in the third to fifth most frequently consumed range (see Table 5.3). Brick margarine was consumed by 61% of participants in the CORIS study (6th most frequently consumed); 57% of Coronary Risk Factor Study (CRISIC) participants (6th most frequently consumed); and forty percent of BRISK participants (8th most frequently consumed) (Wolmarans & Oosthuizen, 2001:S52, Table III).

The SAFBDG recommend that fats should be eaten sparingly, which means that fats should form part of a balanced diet, but should be consumed in limited amounts. Fats are important sources of the essential fatty acids: linoleic and alpha-linolenic acids, and are also precursors for eicosanoids, phospholipids and hormones, but overconsumption may lead to CHD, obesity and breast-, colon- or prostate cancer. To prevent CDL, it is recommended that intake of fried foods, fat from animal sources and non-dairy creamers should be lowered, and that unsaturated margarines (spread thinly on bread) and oils should preferably be consumed (Wolmarans & Oosthuizen, 2001:S48-S49).

Providing generic medication to high-risk CVD individuals to lower blood pressure and cholesterol is cost-effective (Rodgers et al., 2006:online). However, such a strategy remains to be extensively researched (Norman et al., 2007b:714).

(iii) Fruit and vegetables

Overall, women more frequently consumed vegetables and fruit than men (Table 21 and Table 22). The SAFBDG recommend that South Africans should consume “plenty” of fruit and vegetables daily, based on the five-a-day for better health strategy in the United States (Love & Sayed, 2001: S24, S30). Convincing evidence exists that fruit and vegetables protect against cancers of the stomach, esophagus and lungs (Love & Sayed, 2001:S24). Vegetable and fruit also play a protective role against CVD, CHD, and stroke, due to their high content of phytonutrients; flavonoids; potassium; folate and fibre (Schneider et al., 2007:717; WHO/FAO, 2002:86, 89; Love & Sayed, 2001:S24, S27-S28). Increased intake of fruits and vegetables also decreases serum homocysteine, thereby decreasing the risk of developing CVD (Carlson, 2004:449). Fruits and vegetables also have a significant protective effect on high blood pressure and obesity (Schneider et al., 2007:717). The South African burden of disease study found that esophageal cancer accounted for 9,8% attributable DALYs attributable to low fruit and vegetable consumption in men and 7,0% in women, with lung cancer accounting for 7,8% of all DALYs in men and 4,7% in women (Schneider et al., 2007:720).

(iv) Milk and milk products

Full cream milk was consumed frequently by all participants (between 5th and 7th ranking) (Table 5.3). Milk was the third most frequently consumed food item in the THUSA study, but overall intake was low (MacIntyre et al., 2002:252). In the BRISK study, 58% of participants consumed milk and milk products (Scholtz et al., 2001:S43). Many South Africans consume milk, but in a lesser amount than the recommended intake (400 ml or more per day) (Scholtz et al., 2001:S43).

Dairy products are considered expensive in South Africa and intake is also influenced by culture, tradition and religion (MacIntyre et al., 2002: 252; Scholtz et al., 2001:S42). A large portion of the South African black population also struggle with lactose intolerance (MacIntyre et al., 2002:252; Scholtz et al., 2001:S43). This may partly explain why most of the current study's participants did not meet their daily recommended servings of milk and milk products, since they may avoid dairy products. Only 45% of participants in the BRISK study consumed full cream milk (Wolmarans & Oosthuizen, 2001:S53).

In the current study, full cream milk was frequently used in beverages and porridge, but not in large quantities. If two to three servings of dairy are not consumed per day, a person's diet may be deficient in vitamin D, calcium , magnesium and zinc (Scholtz et al., 2001:S43). By consuming inadequate amounts of milk and milk products, the requirement of 1000mg of calcium per day cannot be met (Scholtz et al., 2001:S43). There are unfounded concerns that dairy may increase the risk of CVD, due to increased saturated fat intake. However, saturated fatty acids do elevate blood cholesterol if they are substituted for carbohydrates or other fatty acids in the diet (Krummel, 2004a:878). However, consumption of fermented milk may decrease blood cholesterol concentrations (Scholtz et al. 2001:S43). The Dietary Approaches to Stop Hypertension (DASH) diet recommends increased intake of low fat milk and milk products (USDA & USDHHS, 2010:17; Krummel, 2004b:906).

(v) Sweets and sugar

Sugar was the most frequently consumed food item among all male and female participants in the current study. In comparison it was also found to be the most frequently consumed food item among the following studies: Nel and Steyn's (2002:92, Table 92) urban meta-analysis results; BRISK study (urban Western Cape) (Nel & Steyn, 2002:103); and THUSA study (in the North West province: rural men; men and women living in informal settlements; and women from the middle class urban area)(MacIntyre et al. 2002:247-248, Tables 5-6). It was the second most frequently consumed food item in the following studies: the rural and national meta-analysis results from Nel and Steyn (2002:62, Table 23; 2002:77, Table 26); Lebowa study (rural Limpopo province) (Nel & Steyn, 2002:97); CORIS study (urban and rural Western Cape) (Nel & Steyn, 2002:126-129, Table 37); FYFS project (urban Gauteng and rural Northern province) (Nel & Steyn, 2002:118); and the THUSA study (North West province's rural women; men and women from farms; men from urban middle class and women from urban upper class)(MacIntyre et al., 2002:247-248, Tables 5-6).

One of the trademarks of the nutrition transition is higher consumption of sugar (Joubert, 2007:683). If sugar is consumed in excess it may displace other foods that are good sources of vitamins and minerals in the diet (Van den Berg, 2011:260; DoH, 2004:32). However, eating any food or beverage in excessive amounts, thereby exceeding energy intake above recommended daily requirements, will cause weight gain (Van den Berg, 2011:258).

Therefore, frequent consumption of added sugar in the current study is not necessarily a concern, except where it is consumed in conjunction with high intakes of fat (i.e. the Westernized diet) (Steyn et al., 2006:5,14; Steyn, 2005b:249).

What is also of concern is the frequent consumption of cool drinks, which are sweetened with sugar, both among rural (11th) and urban (11th) men and rural (11th) and urban (10th) women. Drinking one to two sugar-sweetened drinks per day increases the risk for metabolic syndrome, insulin resistance, type 2 DM, CVD, and hypertension (Van den Berg, 2011:257).

When sugar is consumed in moderate quantities, there is no detrimental effect on dental health, weight management, or CDLs (Van den Berg, 2011:257). However, excessive consumption of sugar-sweetened beverages is associated with weight gain and obesity (Van den Berg, 2011:257). It is recommended to limit intake of refined and processed sugars and to emphasize whole grains or complex carbohydrates as part of a balanced diet (Grant, 2008:965). Excessive intake of added sugars should be avoided, and especially products sweetened with fructose (Van den Berg, 2011:257).

The WHO recommends a population nutrient intake goals of less than ten percent of total energy from free sugars (WHO, 2012:online, Table 6).

(vi) Salt

Salt/stock was consumed third most frequently by rural men and fourth most frequently by rural women; urban men and urban women in the current study. None of the comparison studies investigated salt/stock as a frequently consumed food item, and therefore there are no studies available for comparison to salt intake per se. However, again, it should be kept in mind that the current findings do not quantify the amount of salt used, only the frequency.

The SAFBDG recommends that salt should be used sparingly (Charlton & Jooste, 2001:S55). Black hypertensive South Africans are more salt sensitive and more likely to have an increased risk of developing cardiovascular disease. Salt can also influence kidney function (Schutte et al., 2003:343). The SADHS found that 48,3% of black urban men and 43,9% of black urban women consumed salty snacks three or more times per week (Charlton & Jooste, 2001:S59; Bourne et al., 1993:241). A study conducted in the Cape Metropole found that 34% of participants wrongly believed that flavour enhancers that contain monosodium glutamate (e.g. stock) can be used instead of salt to lower salt intake in the diet (Steyn, 2005a:44).

(vii) Tea

Tea was second most frequently consumed by urban Free State women; third most frequently consumed by the urban men and rural women; and fourth most frequently consumed by rural men in the current study. In comparison, it was also second most frequently consumed in the Dikgale study (Nel & Steyn, 2002:101), and third most frequently in the Lebowa study (Nel & Steyn, 2002:97) as well as in all areas of the meta-analysis study (Nel & Steyn, 2002:62,77,92, Tables 23, 26 and 29). It was fourth most frequently consumed in the CORIS study (Nel & Steyn, 2002:126-129, Table 37). The THUSA study found that next to maize products and milk, tea was consumed in large amounts (Scholtz et al., 2001:S43).

The SAFBDG recommends that two litres of safe, clean water should be consumed, in the form of tap water, tea, coffee or other water-based drinks (Bourne & Seager, 2001:S69). Some studies suggest that flavonoids may protect against heart disease (Love & Sayed, 2001:S26). Tea is a good source of flavonoids (Love & Sayed, 2001:S26). Therefore, one would assume that consumption of tea would bear health benefits. However, one must also consider the amount of sugar that is added to the tea, as well as whether non-dairy creamer or milk is added. High intakes of tea can inhibit iron absorption due to its high tannin content (Anderson, 2004:139), which can be disadvantageous to health (Anderson, 2004:140). The NFCS found that iron intake was low in all age groups, and was especially low in the Free State (Labadarios et al., 2005:539). High intakes of tea concurrently with low iron intake, can therefore be detrimental to health.

(viii) Coffee

Men consumed coffee more frequently (5th urban, 6th rural) than women (10th in both rural and urban areas). Coffee consumption in rural and urban children was infrequent (Tables 4.17 to 4.20). It was consumed in similar frequency in the meta-analysis study reported by Nel and Steyn (2002:62, Table 23).

Drinks that contain caffeine, such as coffee, can cause diuresis (Bourne & Seager, 2001:S65). Consuming more than 720 ml of regular coffee per day can increase total cholesterol (Krummel, 2004a:861).

High intakes of coffee also increases homocysteine levels (Verhoef et al., 2002:1246), which has a negative effect on cardiovascular health, since it is linked with several major risk factors for CVD, e.g. blood pressure and hypercholesterolemia (Carlson, 2004:449; Krummel, 2004a:876). Even with a slightly elevated homocysteine, risk for CHD, stroke, peripheral vascular disease increases significantly (Carlson, 2004:449). For these reasons, the SAFBDG recommends that tea and coffee should be drunk in moderation (DoH, 2004:9).

(ix) Alcohol

There was a significant difference in alcohol intake between urban and rural men, with a significantly larger percentage or rural men consuming alcohol within their daily recommendations

(Table 4.15). Five percent of urban men exceeded their daily recommendation of two drinks per day. In the THUSA study, no relationship between the level of urbanisation and amount of consumption was found (Gopane et al., 2010:S16).

The SADHS also found that more men (45%) than women (17%) consumed alcohol (Van Heerden & Parry, 2001:S75). According to Van Heerden and Parry (2001:S75), more urban men (47%) and urban women (19%) usually consume alcohol than rural men (41%) and women (13%). Drinking prevalence was also highest in males of the Free State and Gauteng (50%) (Van Heerden & Parry, 2001:S75). Excessive alcohol intake has been linked with cancer; hypertension (Charlton & Jooste, 2001:S55-S56; Love & Sayed, 2001:S28; Van Heerden & Parry, 2001:S72, Table II), stroke (Charlton & Jooste, 2001:S56; Van Heerden & Parry, 2001:S72, Table II); liver disease; obesity; malnutrition and gastro-intestinal side effects (Van Heerden & Parry, 2001:S72, Table II)

Alcohol and tobacco smoke are some of the carcinogens responsible for esophageal cancer (WCRF/AICR, 2007:19; Churma & Horrell, 2005:45; WHO/FAO, 2002:96), In 2003, mortality rates for esophageal cancer in South Africa were high, with 17,2% of all males dying of this cancer and ten percent of all females. It was the leading cause of cancer death in black South African males and the second leading cause in black South African females (Steyn et al., 2006:30).

Even though men have a higher risk for esophageal cancer than women, both genders should be encouraged to drink alcohol sensibly, as recommended by the SAFBDG (Van Heerden & Parry, 2001:S77).

(x) Conclusion

When comparing frequentintake of certain food **items** for rural and urban men (Table 4.21), there were no significant differences. The following food items fell within the top ten list: sugar; cooked porridge; tea, salt or stock; full cream milk; coffee, margarine/oil/fat; bread; fruit; and vegetables fell in the top ten list. Rural men consumed coffee creamer more frequently than urban men (14th rural vs. 24th urban). Urban men consumed alcohol a little more frequently than rural men (15th rural vs. 17th men).

The frequent intake of food items for women (Table 4.22) looks similar to that of the men. The top ten items consumed frequently by both urban- and rural women were: sugar; cooked porridge; tea, salt or stock; margarine/oil/fat; full cream milk; fruit; bread; and vegetables. As was also the case among rural men, coffee creamer was consumed more frequently by rural women (rural 14th; urban 23rd), as was coffee (rural 10th; urban 22nd). Alcohol consumption was listed much less frequently among women when compared to men.

When comparing the intake of different food **groups**, the majority of all participants (both men and women, rural and urban) ate less than their daily requirements of fruit; legumes; milk and milk products; soy; and vegetables (Table 4.15 and Table 4.16). The majority of the male and all of the

female participants consumed alcohol within limits. However, 5% of urban men exceeded their daily recommendation of two drinks per day. Most participants consumed excessive servings of bread and cereals; and meat and meat products. The majority of rural men and women exceeded their daily intake of fats and oils and sweets/sugar, whereas the majority of urban men and women consumed within their limits of fats and oils; and sweets/sugar. This seems to indicate that the nutrition transition is present in both rural and urban areas, but is more pronounced in rural participants than in urban participants, when looking at consumption of food groups and the top ten frequently consumed food items.

5.3.3 Mean macronutrient intakes

Mean macronutrient intakes were calculated from the 24 hour recall of reported usual intake data and compared to results from similar studies conducted in South Africa. Data was also compared to the current prudent dietary guidelines and recommended dietary reference intakes (Table 3.6) and (Table 3.7).

5.3.3.1 Children younger than two years

The results for the children younger than two years old were compared to the results for the NFCS (Steyn et al., 2006:17, Table 2.4.1; Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000:444-449, Tables 6.97-6.101) and the study conducted by Faber and Benadé (2001:364, Table 3) in rural KwaZulu-Natal. Different methods were used to determine dietary intake. The 24 hour recall was used in the current study as well as in the study of Faber and Benadé (2001:359), whereas both the 24 hour recall and food frequency questionnaire were used in the NFCS (Nel & Steyn, 2002:9, Table 1). Comparative data was summarized in Table 5.4. Acceptable distribution ranges of macronutrients were 45-65% for carbohydrates; 5-20% for protein; and 30-40% for fat (Table 3.6 and Table 3.7) (USDA & USDHHS, 2010:15,Table 2-4).

(i) Total energy intake

As reflected by Table 5.4, the average total energy intake of all children younger than two in the current study was 3510,9 kJ. In comparison, the NFCS found it to be higher at 4690 kJ for all children in the Free State (Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000:444, Table 6.97) and 4200kJ nationally (Steyn et al., 2006:17, Table 2.4.1). The average total energy intake for the current study's rural children was 4254 kJ compared to the national average of 3992 kJ in the rural NFCS (Steyn et al., 2006:17, Table 2.4.1). Faber and Benadé (2001:364, Table 3) found it to be 3691 kJ for their rural children. The average total energy intake for the current study's urban children was 3292,4 kJ, far less than the 4403kJ found in the urban NFCS nationally (Steyn et al., 2006:17, Table 2.4.1).Therefore, the current study's total energy intake for rural children was similar to both the rural NFCS and Faber and Benadé's study. However, the urban children's intake in the current study was than that of the urban NFCS participants.

Table 5.4 - Comparison of macronutrient intakes with other studies' data – children < 2 years old (from 24 hour recall)

Reference source								*Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000:444-448, Table 6.97-6.101*	*Steyn et al., 2006:17, Table 2.4.1*			*Faber and Benadê, 2001:364, Table 3)*
Study	**AHA-FS (Current study)**							***NFCS***				
Time period of data	***2006***		***2009***					***1999***				***2000***
Ethnic group	***Mostly black***							***All***				***Black***
Age groups (years)	***0-2***							***1-3***				***4-24 months***
Area	***Free State - rural BOYS***	***Free State - rural GIRLS***	***Free State - urban BOYS***	***Free State - urban GIRLS***	***Free State - all RURAL***	***Free State - all URBAN***	***Free State - all***	***Free State - all***	***National - rural***	***National - urban***	***National - all***	***KwaZulu-Natal - rural***
n=	***4***	***6***	***16***	***18***	***10***	***34***	***44***					
Energy (kJ)	4952.5	3788.3	3862.75	2785.4	4254.0	3292.4	3510.9	4690	3992	4403	4200	3691
CHO (g)	163.8	127.8	144.3	106.2	142.2	124.1	128.2	178				
CHO (% energy)	56.2	57.4	63.5	64.8	56.9	64.2	62.5	64.5	64.3	59.5	61.5	57
Protein (g)	55.0	36.0	44.3	32.1	43.6	37.8	39.2	32				
Protein (% energy)	18.9	16.2	19.5	19.6	17.2	19.5	19.0	11.6	12.4	12.7	12.6	8
Fat (g)	36.3	24.2	21.4	14.1	29.0	17.5	20.2	25				
Fat (% energy)	27.9	24.3	21.1	19.2	25.7	20.1	21.4	20.3	20.9	25.0	22.6	37

The current study's overall total energy intake results for children younger than two years (3510,9 kJ) was sufficient if compared to the DRI for children 7-12 months (2839 kJ ♀; 3121 kJ ♂), but it was insufficient when compared to the DRI for 1-to-3 year olds (4166 kJ ♀; 4393 kJ ♂) (Food & Nutrition Board et al., 2002:1324). The current study's rural children (4254 kJ) met the DRI for 1-to-3 year olds, but the urban children (3292,4 kJ) did not.

(ii) Carbohydrates

The average percentage energy from carbohydrates for all the current study's children together was 62,5%. In comparison, it was found to be 64,5% in the Free State section of the NFCS (Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000: 446, Table 6.99) and 61,5% in the NFCS nationally (Steyn et al., 2006:17, Table 2.4.1). The total percentage energy intake from carbohydrates was 56,9% for rural children and 64,2% for urban children included in this study. In comparison, it was 64,3% in the rural NFCS nationally and 59,5% in the urban NFCS nationally (Steyn et al., 2006:17, Table 2.4.1). Faber and Benadé (2001:364, Table 3) found carbohydrate intake to be 57% of total energy in their study. The NFCS found that intake was higher in rural areas than in urban areas. However, the current study found the opposite to be true. The current findings for rural children are therefore closer to Faber and Benadé's findings for rural children in KwaZulu-Natal than the NFCS finding.

Intake for the current study's findings for percentage energy from carbohydrates (rural: 56,9%; urban: 64,2%; overall:62,5%) fell within the prudent dietary guidelines (Table 3.6) of 45% to 65% (USDA & USDHHS, 2010:15, Table 2.4). The average grams of carbohydrate consumed in the current study was 128,2g, which almost falls within the DRI recommendation of 130g per day (Food & Nutrition Board et al., 2002:1324)(Table 3.7). The current study's rural children (142,2 g) consumed amounts within the DRI recommendations, but the urban children (124,1g) did not.

(iii) Protein

The mean percentage energy from protein for all children in the current study was 19% (in rural children it was 17,2% and in urban children it was 19,5%). The current findings are much higher than the finding of both the NFCS for the Free State (11,2%) (Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000:445, Table 6.98); and the study by Faber and Benadé (8%) (Faber & Benadé, 2001:364, Table 3). The current study's finding still fall within prudent dietary guidelines of between five and twenty percent energy from protein for one-to-three-year olds (USDA & USDHHS, 2010:15, Table 2-4).

When looking at total grams of protein consumed, (rural: 43,6 g; urban: 37,8g; overall: 39,2g), intakes are far higher than the DRI recommendation of 13 g for 1-to-3 year olds (Food & Nutrition Board et al., 2002:1324). This may indicate that the first solids introduced to children may include a larger portion of protein-based foods, rather than carbohydrate-based foods. When looking again at the intake of food groups (Table 4.16), and specifically meat and meat products, 50,4% of rural women and 38,4% of urban women consumed above their recommended daily servings of meat and meat products.

(iv) Fat

When comparing grams of fat consumed by the current study's children younger than two years (rural 29g; urban 17,5g; overall: 20,2 g), the current study's findings fell below the DRIs for both 0-6 month old children (31 g) and 7-12 month old children (30 g) (Food & Nutrition Board et al., 2002:1324). However, the lower fat intake may be due to the higher proportionate intake of protein found in the current study.

The percentage energy from fat was 21,4% for children younger than two years in the current study (rural: 25,7%; urban: 20,1%). In comparison, the NFCS found it to be quite similar at 20,3% in their Free State group (Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000:448, Table 6.101) and 22,6% nationally (Steyn et al., 2006:17, Table 2.4.1). The rural children in the NFCS nationally consumed 20,9% and the urban children consumed 25% (Steyn et al., 2006:17, Table 2.4.1). Intake was even higher among rural children in KwaZulu-Natal at 37% of total energy (Faber & Benadé, 2001:364, Table 3). Again, the current study's results differ from the NFCS inasmuch as the current study's rural children consumed a higher percentage energy from fat than urban children, as opposed to the NFCS's urban children who consumed more than the rural children. However, keep in mind that sample sizes in especially the rural sample were very small.

When looking at prudent dietary guidelines for macronutrient proportions in children one-to-three years old (30-40%)(USDA & USDHHS, 2010:15, Table 2-4), the current study's children consumed too little energy from fat. This low intake of fat is of concern, and may be related to the a high prevalence of underweight (Table 4.29) and stunting found in children in this study (Table 4.30).

5.3.3.2 Children older than two years

The current results for children older than two years were compared with the following studies: NFCS with different age groups (i.e. one to three years old; four to six years old, as well as one to nine years old) and different areas (i.e. Free State, rural or urban national areas) (Steyn et al., 2006:17, Table 2.4.1; Labadarios et al., 2005:538; MacIntyre & Labadarios, 2000:444-448, 450. Table 6.97-6.101, 6-103); the BRISK study in urban Western Cape (Steyn et al., 2003:636, Table 3; Nel & Steyn, 2002:21, Table 4); the Lebowa study rural Limpopo province (Steyn et al., 2003:636, Table 3; Nel & Steyn, 2002:22,Table 4); the study conducted by MacKeown et al. in rural and urban Gauteng (1998:301, Table 2); and the study conducted by Faber et al. (2001:405, Table 2) in rural KwaZulu-Natal. Comparative data was summarized in Table 5.5. Again, methods for determination of dietary intake were different in the studies. The 24 hour recall was used in: the current study; the Lebowa study (Nel & Steyn, 2002:9, Table 1); and the study by Faber et al. (2001:402). The food frequency questionnaire was used in the study by MacKeown et al. (1998:300, Table 1). Both methods were used in the NFCS (Labadarios et al., 2005:533: Nel & Steyn, 2002:9, Table 1), and the BRISK study (Nel & Steyn, 2002:9, Table 1).

(i) Total energy intake

Average total energy intake combined for all the current Free State children between two and seven years old was 4976,5 kJ. In the NFCS, it was very similar in all Free State children aged

Table 5.5 - Comparison of macronutrient intakes with other studies' data – children > 2 years old

Reference source									*Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000:444-448, Table 6.97-6.101*		*Steyn et al., 2006:17, Table 2.4.1*				*MacIntyre & Labadarios, 2000:450, Table 6.103*			*MacKeown et al., 1998:301,Table 2*		*Faber et al. (2001:405, Table 2)*	*Steyn et al.,2003:636, Table 3; Nel & Steyn, 2002:21, Table 4*		*Steyn et al.,2003:636, Table 3; Nel & Steyn, 2002:22, Table 4*	
Study		**AHA-FS (Current study)**							**NFCS**												**BRISK (Black risk factor)**		**Lebowa**	
Time period of data		*2006*		*2009*		*Mean*			*1999*									*1984*	*1995*	*?1999-2001*	*1983 - 1990*		*1991*	
Ethnic group		*Mostly black*							*All*									*Black*		*Black*	*Black*		*Black*	
Age groups (years)		*2 - 7*							*1-3*	*4-6*	*1-3*		*4-6*		*1 - 9*			*4 - 5*	*5*	*2 - 5*	*1 - 5*	*6 - 9*	*1 - 5*	*6 - 9*
Method		*24 hour recall*							*24 hour recall and FFQ*									*Dietary history*	*FFQ*	*24 hour recall*	*24 hour recall and FFQ*		*24 hour recall*	
Area		*Free State - rural BOYS*	*Free State - rural GIRLS*	*Free State - urban BOYS*	*Free State - urban GIRLS*	*Free State - all RURAL*	*Free State - all URBAN*	*Free State - all*	*Free State - all*		*National - rural*	*National - urban*	*National - all*	*National - rural*	*National - urban*	*National - all*	*National - boys*	*National - girls*	*Free State - all*	*Jhb/Soweto - urban and Bophuthat-swana - rural*	*Jhb/Soweto - urban*	*KwaZulu Natal - rural*	*Cape Town (Western Cape) - urban*		*Northern Province (Limpopo) - rural*	
n		26	24	64	48	50	112	162																
Energy	kJ	5927	5441	4743	3959	5693.7	4407.0	4804.1	4690	5388	3992	4403	4200	4963	5614	5271				6543.6	8799	5085	5271	6426	5211	7755
CHO	g	192.9	186.8	185.1	151.6	190.0	170.7	176.7	178.0	202.0	151.0	154.0	152.0	193.0	192.0	103.0				220.0	259.0					
	%	55.3	58.4	66.3	65.1	56.8	65.8	63.0	64.5	63.7	64.3	59.5	61.5	66.1	58.1	62.2	61.8	60.9	65.0	57.2	50	67.0				
Protein	g	60.0	53.7	49.1	42.1	57.0	46.1	49.5	32.0	37.0	29.0	33.0	31.0	36.0	43.0	39.0				47.0	57.0					
	%	17.2	16.8	17.6	18.1	17.0	17.8	17.6	11.6	11.7	12.4	12.7	12.6	12.3	13.0	12.6	12.0	12.2	11.8	12.2	11.0	11.0				
Fat	g	48.1	39.8	24.9	21.4	44.1	23.4	29.8	25.0	30.0	22.0	29.0	25.0	42.0	38.0	31.0				52.0	95.0					
	%	30.8	27.8	20.0	20.5	29.4	20.2	23.0	20.3	21.2	20.9	25	22.6	32.2	25.7	22.4	22.9	23.7	20	30.2	41.0	27.0				

between one and three years old (4690 kJ), but lower than in the Free State children aged between four and six years old (5388 kJ) (Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000:444, Table 6.97). Nationally in the NFCS it was lower among one-to-three-year olds (4200 kJ) and higher among four-to-six- year olds (5271 kJ)(Steyn et al., 2006:17, Table 2.4.1).

The average total intake for the current study's rural participants was 5693,7 kJ. In the NFCS it was less, at 3992 kJ for one-to-three-year old rural children and 4963 kJ for the rural four-to-six-year olds (Steyn et al., 2006:17, Table 2.4.1). In rural KwaZulu-Natal it was also less with 5085 kJ among two-to-five-year olds (Faber et al., 2001:405, Table 2). Energy intake was higher among four-to five-year olds from rural and urban Gauteng in 1984 (6543,6 kJ)(MacKeown et al. , 1998:301, Table 2). In rural Limpopo (Lebowa study) it was less with 5211 kJ in children aged one to five years old, and higher with 7755 kJ in children aged six to nine years old (Steyn et al.,2003:636, Table 3; Nel & Steyn, 2002:22, Table 4).

Among the current study's urban children the average intake was less (4407 kJ) than in rural children (5693,7 kJ); less in comparison to the national intake among urban children aged between four to six years old (5614 kJ); but similar to the national NFCS for urban children aged between one and three years old (4403 kJ) (Steyn et al., 2006:17, Table 2.4.1). It was higher in the BRISK study for both one-to-five-year olds (5271 kJ) and six-to-nine-year olds (6426 kJ)(Steyn et al., 2003:636, Table 3; Nel & Steyn, 2002:21, Table 4). Intake was even higher in five-year-olds in urban Gauteng in 1995 (8799 kJ)(MacKeown et al., 1998:301, Table 2).

When comparing the current study's results for energy intake (rural 5693,7kJ; urban:4407 kJ; overall 4804,1 kJ), with the DRIs (Table 3.7); participants consumed sufficient quantities if the DRIs for 1-3 years old were used (4166 kJ ♀; 4393 kJ ♂), but were insufficient if DRIs for 4-8 years old (6895 kJ ♀; 7316 kJ ♂) were used (Food & Nutrition Board et al., 2002:1324). Our samples were too small to divide children who were two years and older into smaller groups (e.g. one-to-three years old and four-to-eight years old).

(ii) Carbohydrates

The average percentage energy from carbohydrates was 61,1%. These results compared well to the national NFCS's findings among one-to-three-year olds (61,5%), and four-to-six-year olds (62,2%) (Steyn et al., 2006:17, Table 2.4.1). In the NFCS for the Free State, it was 64,5% among one-to-three-year olds and 63,7% among four-to-six-year olds (Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000:446, Table 6.99). It was also similar among one-to-nine-year olds in the Free State NFCS (65%) (MacIntyre & Labadarios, 2000:450, Table 6.103). MacKeown et al. (1998:301, Table 2) found percentage intake to be lower among four-to-five-year olds in Gauteng (57,2%).

In rural areas, children consumed an average percentage energy from carbohydrate of 56,8%. It was higher nationally in rural areas of the NFCS among one-to-three-year olds (64,3%) and four-to-six-year olds (66,1%)(Steyn et al., 2006:17, Table 2.4.1). It was also higher (67%) among two-to-five-year olds of rural KwaZulu-Natal (Faber et al., 2001:405, Table 2).

Among the current urban children percentage energy from carbohydrates was 65,8%. In comparison, it was found to be less in the urban NFCS among both one-to-three-year olds (59,5%) and four-to-six-year olds (58,1%) (Steyn et al., 2006:17, Table 2.4.1). It was also lower in urban five-year olds in Gauteng (50%) (MacKeown et al., 1998:301, Table 2).

Again, among children older than two years in the current study, intake was higher in urban than rural children, whereas the opposite was true for the NFCS findings. Despite differences between above-mentioned study results, all intakes were still within prudent dietary guidelines of 45-65% energy from carbohydrates (USDA & USDHHS, 2010:15, Table 2-4).

When looking at the amount of carbohydrates consumed in grams (rural: 190g; urban 170,7g; overall:176,7 g), intakes exceed the DRI recommendation of 130g per day (Food & Nutrition Board et al., 2002:1324).

(iii)Protein

For children between two and seven years, the average energy from protein was 17,4%. Among rural children it was 16,9% and among urban children it was 17,9% (Table 5.5). As was the case among the children younger than two years old in the current study; other studies found lower results for protein intake. In the Free State section of the NFCS it was 11,6% in all one-to-three-year olds; and 11,7% in four-to-six-year olds (Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000:445, Table 6.98). In the national section of the NFCS it was 12,6% among one-to-three-year olds and four-to-six-year olds (Steyn et al., 2006:17, Table 2.4.1); and 11,8% among one-to-nine-year olds in the Free State (MacIntyre & Labadarios, 2000:450, Table 6.103). It was also lower (12,2%) in MacKeown et al.'s study (1998,301, Table 2).

In the current study's rural children, percentage energy from protein was on average 17,0%. This was higher than the 12,4% among the rural national NFCS in one-to-three-year olds; and 12,3% in the four-to-six-year olds (Steyn et al., 2006:17, Table 2.4.1). Faber et al. (2001:405, Table 2) found it at 11% in rural KwaZulu-Natal two-to-five-year olds.

Percentage energy from protein for the urban children in the current study was a little higher (17,8%) than the rural children. It was also higher than the national urban NFCS for both one-to-third-year-olds (12,7%) and four-to-six-year olds (13,0%) (Steyn et al., 2006:17, Table 2.4.1), as well as the urban Gauteng five-year olds (MacKeown et al., 1998,301, Table 2).

Protein intakes from all studies, albeit different, still fell within the prudent dietary guidelines for macronutrient proportions of 5-20% for children 1-3 years old and 10-30% of children 4-18 years old (USDA & USDHHS, 2010:15, Table 2-4).

Average intake in grams (rural: 57,0g; urban: 46,1g; overall 49,6g) fell far above the DRI recommendations for both 1-3 year olds (13 g) and 4-8 year olds (19 g) (Food & Nutrition Board et al., 2002:1324).

A reason for the higher protein intake in the current study may be because legumes (which are high in protein and carbohydrates) were analysed as a separate food group on their own in this

study (1 exchange=500 kJ, 7 g protein, 21 g carbohydrates), which could have contributed more protein to the analysis than if they were classified as part of the breads and cereals group (285kJ, 3 g protein; 15 g carbohydrates).

(iv)Fat

On average, children in the current study consumed about 25% of their energy from fat. Other studies found a lower intake of energy from fat: 20,3% among one-to-three-year olds and 21,2% among four-to-six-year olds in the Free State results of the NFCS (Labadarios et al., 2005:538, Table 3; MacIntyre & Labadarios, 2000:448, Table 6.101); 22,6% among one-to-three-year olds and 22,4% in four-to-six-year olds in the national results of the NFCS (Steyn et al., 2006:17, Table 2.4.1); and twenty percent among one-to-nine-year olds in all Free State children of the NFCS (MacIntyre & Labadarios, 2000:450, Table 6.103). The only study that found an intake higher (30,2%) than the current study, was among four-to-five-year olds in the study by MacKeown et al. (1998:301, Table 2).

Fat intake was 29,4% of total energy among rural children in this study, which fell within prudent dietary guidelines of 30-40% (USDA & USDHHS, 2010:15, Table 2-4). The NFCS found a lower intake among one-to-three-year olds nationally (20,9%) and a higher intake among four-to-six-year olds nationally (32,3%), which was closer to the current results (Steyn et al., 2006:17, Table 2.4.1). Faber et al. (2001:405, Table 2) also found a similar intake (27%) in their study.

Among the current urban participants, the average percentage energy from fat intake was 20,2%, again falling within prudent dietary guidelines (USDA & USDHHS, 2010:15, Table 2-4). All other urban studies found it to be higher: the NFCS found it as 25% nationally among one-to-three-year olds and 25,7% among four-to-six-year olds (Steyn et al., 2006:17, Table 2.4.1) and MacKeown et al (1998:301, Table 2) found it to be 41%. It may be higher in the findings by MacKeown et al. since they used the food frequency questionnaire method, whereas the other studies either used the 24 hour recall method or a combination of the 24 hour recall and food frequency questionnaire.

It is interesting to note that the national NFCS results found, similarly to the current results, that percentage energy from fat intake was higher in urban four-to-six-year olds than among their rural counterparts (Steyn et al., 2006:17, Table 2.4.1). When looking at the amount of grams consumed in the current study, the rural participants consumed about double the amount in grams (rural: 44,1g; urban: 23,4 g).

5.3.3.3 Adults

The current results for energy- and macronutrient intake have been compared with results from the following studies: BRISK (Bourne et al., 1993:241, Table II); CORIS (Wolmarans et al., 1988:12-15); Dikgale (Steyn et al., 2006, Table 2.2.1); THUSA (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:240,243, Table 1); VIGHOR (Oosthuizen et al., 1998:1214, Table I); Indians urban (Wolmarans et al., 1998:56-57, Tables 1&2); CRISIC (Steyn et al., 2006, Table 2.2.1). The current results for energy intake alone have been compared with the results from the following studies; which only have results for energy intake (not for

macronutrients): First Year Female Students Project (Steyn et al., 2003:656, Table 3); Lebowa (Steyn et al., 2003:656, Table 3); WRFS (Steyn et al., 2003:656, Table 3; Nel & Steyn, 2002:23, Table 5). Table 5.6 sets out the current study's results in comparison to the THUSA study's findings. All other studies' results in comparison with the current results are set out in Table 5.7.

The following studies used a 24 hour recall to obtain dietary intake information: BRISK; CORIS, Indians urban; Lebowa and Dikgale. The following studies used food frequency questionnaires: THUSA, FYFS and WRFS.

MacIntyre et al. (2002:240) divided the THUSA study's data into two groups. One group contained the rural area, the farms, informal settlements and middle class urban. The second group comprised only the upper class urban. Their argument was that the results for the areas contained in group one was similar, whereas the results for the upper class urban participants were different (Table 5.6).

(i) Total energy intake

The current study's **rural males** consumed an average of 8630 kJ per day. It was less than that of the THUSA study, which found for males: 9597 kJ in rural villages and 8913 kJ on farms (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1). However, it must be kept in mind that the THUSA study used the food frequency questionnaire to determine their values, whereas the current study used the 24 hour recall. Currently, **rural women** consumed on average 7754,9 kJ in comparison with 7906 kJ in the THUSA study for women in rural villages and 7973 kJ on farms (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1). Average energy intake **for all rural** participants in the current study was 8011,1kJ. The THUSA study's rural result was 9333 kJ (Table 5.6). Average intake in the Dikgale study was between 6000kJ and 6700 kJ (Steyn et al., 2006:15, Table 2.2.1; Steyn, 2005a:35, Table 4.2).

Average energy intake for **urban men** was 7078,9 kJ, in comparison with a higher intake in the THUSA study of 9333 kJ for men in squatter camps; 9897 kJ in townships and 9818 kJ in upper class urban men (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1) and also a higher intake among men in the VIGHOR study; 10 700 kJ (Oosthuizen et al., 1998:1214, Table I) and the BRISK study (8500 kJ) (Bourne et al., 1993:241, Table II).

Urban women's average energy intake was 6621 kJ. The THUSA study reported higher values, with 7893 kJ in squatter camps; 8010 kJ in townships and 8523kJ among the upper class (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1). Results from the VIGHOR study (6300kJ) (Oosthuizen et al., 1998:1214, Table I) and the BRISK study (Bourne et al., 1993:241, Table II) were similar to the current study. Average energy intake for **all urban** participants (male and female together) was 6729 kJ. The THUSA study's urban group intake was 8300 kJ, which is again higher than the current study's findings (Steyn, 2005a:35, Table 4.2; MacIntyre et al., 2002:240). The average intake for all **female adults** in the current

Table 5.6 Comparison of mean total energy and percentage macronutrient intake - adults 25-64 years - with THUSA study results

Reference source	*Current study*									*Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1*										*Steyn, 2005a:35, Table 4.2; MacIntyre et al., 2002:240*	
Study	**AHA-FS**									***THUSA***										***THUSA***	
Time period of data	*2006*		*2009*		*Average*					*1996-1998*										*1996-1998*	
Ethnic group	*Mostly black*									*Black*										*Black*	
Age groups (years)	*25 - 64*									*25 - 60+*										*25 - 60+*	
Method	*24 hour recall*									*FFQ*										*FFQ*	
Area	*Free State - rural*		*Free State - urban*		*Free State - all*					*North West - rural villages*		*North West - farms (rural)*		*North West - squatter camps (urban)*		*North West - township/middle class (urban)*		*North West -upper class urban*		*Northwest*	
	♂	♀	♂	♀	**All ♂**	**All ♀**	**All rural**	**All urban**	**Average**	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	**rural, farm, informal settlement, middle class urban**	**upper class urban**
n	161	389	99	320	260	709	550	419	969												
Energy	8630.1	7754.9	7078.9	6620.8	8039.5	7243.0	8011.1	6729.0	7456.7	9597	7906	8913	7973	9333	7893	9897	8010	9818	8523	9300	8300
Carbohydrates (% energy)	60.2	60.3	62.2	63.3	61.0	61.7	60.3	63.0	61.5	67.4	67.0	67.2	68.3	65.5	64.1	64.0	61.5	57.3	55.6	65	57.3
Protein (% energy)	17.5	16.9	18.3	17.7	17.8	17.3	17.1	17.8	17.4	11.6	11.4	12.1	11.3	12	12.1	11.8	12.1	13.2	13.4	12	13.2
Fat (% energy)	25.2	25.9	23.3	22.8	24.3	24.5	25.7	22.9	24.5	22.9	23.6	22.8	22.6	24.3	27.7	26.0	27.7	30.6	31.8	22	30.6

Table 5.7 Comparison of mean total energy and percentage macronutrient intake - adults 25-64 years - with other South African studies

Reference source	*Current study*									*Bourne et al., 1993:241, Table II*	*Wolmarans et al., 1988:3, Table II*	*Steyn et al., 2006:15, Table 2.2.1; Steyn, 2005a:35, Table 4.2a*	*Oosthuizen et al., 1998:1214, Table I*	*Wolmarans et al.1998:56-57, Tables 1&2*	*Steyn et al., 2006:15, Table 2.2.1; Steyn, 2005a:35, Table 4.2a*	*Steyn et al., 2003:636, Table 3*	*Steyn et al., 2003:636, Table 3*	*Steyn et al., 2003:636, Table 3; Nel & Steyn, 2002:23,Table 5*			
Study	**AHA-FS**									*BRISK*	*CORIS*	*Dikgale*	*VIGHOR*	*Indians urban*	*CRISIC*	*FYFS*	*Lebowa*	*WRFS**			
Time period of data	*2006*		*2009*		*Average*					*1990*	*1979 & 1983*	*1998*				*1994*	*1991*	*?1997*			
Ethnic group	*Mostly black*									*Black*	*White*	*Black*	*White*	*Indian*	*Coloured*	*Black*	*Black*	*All*			
Age groups (years)	*25 - 64*									*19 – 64*	*20 - 64*	*20 - 65*	*15 - 64*	*15 - 69*	*20 - 34*	*≥25*	*19 - 24*	*25-34*	*35 - 44*	*45-54*	*55-64*
Method	*24 hour recall*									*24 hour recall*	*24 hour recall*	*24 hour recall*		*24 hr recall*		*FFQ*	*24 hour recall*	*FFQ*			
Area	*Free State – rural*		*Free State - urban*		*Free State - all*					*Cape Town (Western Cape) - urban*	*Western Cape - urban and rural*	*Northern (Limpopo) Province - rural*	*Gauteng - urban*	*KwaZulu-Natal - Urban*	*Urban*	*Gauteng (Northern Province) - urban and rural*	*Northern Province (Limpopo) - rural*	*South Africa*			
	♂	♀	♂	♀	All ♂	All ♀	All rural	All urban	Mean									*Postal survey			
Energy	8630.1	7754.9	7078.9	6620.8	8039.5	7243.0	8011.1	6729.0	7456.7	8500 (♂) 6400 (♀)	10300-12000(♂) 6300-7100(♀)	6000 - 6700	10700(♂) 6300(♀)	5600-8100 (♂) 5000-5600 (♀)	7100-10300	9904	7815	7624	7388	6624	6398
Carbohydrates (% energy)	60.2	60.3	62.2	63.3	61.0	61.7	60.3	63.0	61.5	59.2-61.3(♂) 62.0-62.7(♀)	44.8-46.6(♂) 45.5-47.4(♀)	62.4 - 70.8	45.3(♂) 48.4(♀)	45.6-49.7 (♂) 48.8-50.4 (♀)	45-46.5						
Protein (% energy)	17.5	16.9	18.3	17.7	17.8	17.3	17.1	17.8	17.4	15.1-15.3(♂) 14.3-14.5(♀)	15.1-15.7(♂) 15.5-16.6(♀)	14.2 - 15.6	15.6(♂) 15.9(♀)	12.2-13.8 (♂) 11.9-13.3 (♀)	14.9-15						
Fat (% energy)	25.2	25.9	23.3	22.8	24.3	24.5	25.7	22.9	24.5	23.8-25.9(♂) 26.1-27.0(♀)	35.0-35.6(♂) 35.3-36.5(♀)	15.7 - 17.1	39.3 (♂) 38.1(♀)	33.3-36.6 (♂) 34.0-34.8 (♀)	37.3-38						

study was 7243 kJ. In the FYFS it was much higher (9904 kJ)(Steyn et al., 2003:636, Table 3). Intake for all women and men in the current study was below DRIs for both active adults (men: 12881 kJ; women: 10093 kJ) (Food & Nutrition Board et al., 2002:1324) and sedentary adults (men:10143 kJ; women: 7947 kJ – active DRIs / 1.27 physical activity coefficient).

(ii) Carbohydrates

In rural areas, the average intake of energy from carbohydrates in men was 60,2% and in women was 60,3%. In the men included in the THUSA study, it was higher at 67,4% of total energy in rural villages and 67,2% of total energy in farms. Among rural THUSA women it was also higher at 67% in rural villages and 68,3% in farm areas (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1).

Average intake for **all rural** participants (men and women combined) in the current study was 60,3% in comparison to 65% in the THUSA study (Steyn, 2005a:35, Table 4.2; MacIntyre et al., 2002:240).

In the current study, the average percentage of total energy from carbohydrates for **urban** men was 62,2% and for women 63,3%. The BRISK study found similar values of 59,2 to 61,3% for men and 62 to 62,7% for women (Bourne et al., 1993:241, Table II), but the THUSA study reported higher values of 67,2% in squatter camp men; 64% in township men and lower values of 57,3% in upper class urban men. In the VIGHOR study, a lower value of 45,3% was reported among urban men (Oosthuizen et al., 1998:1214, Table I). Among THUSA women it was also higher with 64,1% of total energy from carbohydrates in squatter camps, 61,5% in townships, and lower (55,6%) in upper class urban women (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1). As with energy, the VIGHOR study reported a lower value of 48,4% among their women (Oosthuizen et al., 1998:1214, Table I).

Intake for **all urban** participants (men and women combined) in the current study was higher (63%) in comparison to the THUSA study's average (57%) (Steyn, 2005a:35, Table 4.2; MacIntyre et al., 2002:240).

Carbohydrate intake for rural and urban **men** in the current study was 61% of total energy, which was lower than the THUSA study's 64,4% of total energy in men. In the current study, 61,7% of total energy for **all women** were comparable to the THUSA study's 62,4% in women (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1).

Average intake for **all participants** (men and women combined) in the current study was 61,5% of total energy. The current study's value falls in the same range found by the BRISK study, but is higher than the average values found by the CORIS, VIGHOR, Indians urban, and CRISIC studies (all urban studies, except for CORIS that covered rural and urban); and lower than the average value in the Dikgale study, which was conducted in a rural area (Steyn et al., 2006:15, Table 2.2.1; Steyn, 2005a:35, Table 4.2a).]

Vorster and Nell (2001:S19) argue that in order for women and men to meet their carbohydrate intake of at least 55% of total energy (8000kJ in women; 10 000kJ in men), women should consume at least 259 g of carbohydrate per day and men 325 g per day. Men in the current study did not meet this requirement, since median intake for men was 297g and for urban men was 259g. Rural women met it with a median intake of 260g, but not urban women (242,5g) (Table 4.27 and Table 4.28). According to the DRIs, at least 130g carbohydrates need to be consumed; an amount that the average participants in the current study met quite easily (USDA & USDHHS, 2010:15:Table 2-4).

The current study's results for both rural and urban participants also fell within the prudent dietary guidelines (Food & Nutrition Board et al., 2002:1324).

(iii) Protein

In the current study, the average percentage energy from protein in **rural** men was 17,5% and 16,9% in women**.** The THUSA study found it to be lower for men at 11,6% of total energy in rural villages and 12,1% of total energy in farm areas. For women, it was also lower: 11,4% in rural villages and 11,3% in farm areas (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1). The average for **all rural** participants in the current study was 17,1% and in the THUSA study it was 12% (Steyn, 2005a:35, Table 4.2; MacIntyre et al., 2002:240).

Urban males in the current study had a protein intake of 18,3% of total energy and for women it was 17,7% of total energy. In comparison, the THUSA study reported lower protein intakes of 12% of total energy in squatter camps; 11,8% of total energy in townships and 13,2% of total energy in upper class urban areas; and for women: 12,1% of total energy in squatter camps; 12,1% of total energy in townships and 13,4% of total energy in upper class urban (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1). It was 15,6% for urban men and 15,9% for urban women in the VIGHOR study (Oosthuizen et al., 1998:1214, Table I) and in the BRISK study it was between 15,1% and 15,3% in men and 14,3 to 14,5% in women (Bourne et al., 1993:241, Table II). The overall average percentage intake for **all urban participants** in the current study was 17,8% in comparison to the THUSA study's 13,2% (Steyn, 2005a:35, Table 4.2; MacIntyre et al., 2002:240).

The average intake for **all men** in the current study was 17,8%. For **all women** it was 17,3%. The average intake **for all participants** was 17,4% in the current study. This is higher than the ranges found by all the other comparative studies (see Table 5.7), except for the female participants of the CORIS study (ranging between 15,5% and 16,6%) (Wolmarans et al., 1988:13, Table II).

The reason for the higher protein intake found in the current study, may be partially due to the fact that the exchange list that was devised for legumes, which sets out that one legume portion contains 7 grams of protein (equal to the amount contained in a meat exchange), also finds that soy products contain 13 grams of protein, and this was taken into consideration when protein intake was calculated. Urban participants also frequently purchased and consumed peanuts from a

street vendor in the vicinity where the study was conducted, which also contributed to their daily protein intake. As can be seen from Tables 4.19 to 4.22, peanuts were consumed more frequently by urban participants.

The prudent dietary guidelines recommend a protein intake of 10-35% of total energy and the DRIs recommend a protein intake of 56g for men and 46g for women (USDA & USDHHS, 2010:15:Table 2-4). The current study's results fell within all of these guidelines (Food & Nutrition Board et al., 2002:1324)

(iv) Fat

In the current study, total average percentage energy intake from fat was 25,2% for **rural** men and 25,8% for rural women. The THUSA study found it to be a little less at 22,9% for rural village men and 23,6 for rural village women. For men from farms it was 22,8% and for women it was 22,6% (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1). **Average rural** intake in the current study was 25,7% in comparison to the THUSA study's 22% (Steyn, 2005a:35, Table 4.2; MacIntyre et al., 2002:240).

For **urban** participants in the current study percentage energy from protein was 23,3% for men and 22,8% for women. The BRISK study found similar results of 26,1% to 27% for women and 23,8% to 25,9% for men (Bourne et al., 1993:241, Table II). The THUSA study found it to be higher at 24,3% (men) and 27,7% (women) in squatter camps; 26% (men) and 27,7% (women) in townships; and 30,6% (men) and 31,8% (women) in upper class urban areas (Steyn et al., 2006:15, Table 2.2.2; Steyn, 2005a:35, Table 4.2b; Vorster et al., 2005:485, Table 4; MacIntyre et al., 2002:243, Table 1). The VIGHOR study found a much higher value of 39,3% in men and 38,1% in women (Oosthuizen et al., 1998:1214, Table I). The average for **all urban** participants in the current study was 23,1% in comparison to 30,6% in the THUSA study (Steyn, 2005a:35, Table 4.2; MacIntyre et al., 2002:240).

The average intake for **all** current **male** participants was 24,3% and for **women** was 24,5%. The average for **all participants** in the current study was 24,3%. Interestingly, neither the current study's results nor the THUSA study's results were found to be above thirty percent energy intake from fat, except for the upper class urban participants in the THUSA study.

The current study's results again correlate well with the BRISK study's range of values (Bourne et al., 1993:241, Table II). However, all other studies found fat intake to be higher than thirty percent intake (Table 5.7).

In studies where the prevalence of overweight and obesity was high, fat intake was usually more than thirty percent of total energy intake (Wolmarans & Oosthuizen, 2001:S48). A positive association was found between obesity and the percentage energy consumed from fat. However, decreased physical activity also plays a role (Wolmarans & Oosthuizen, 2001:S51).

The prudent dietary guidelines recommend 20-35% of total energy intake from fat (USDA & USDHHS, 2010:15:Table 2-4). The current study's results fell within these guidelines.

5.4 Anthropometric variables

5.4.1 Children younger than seven years

The anthropometric findings of the current study were compared with those of the NFCS (nationally)(Labadarios et al., 2005:536-537, Tables 1-2; Steyn et al., 2005:7-10, Tables 2-6) and the SAVACG (Labadarios & Nel, 2000:215-216, Tables 4.31- 4.32). These results are set out by Tables 5.8 to 5.11 below.

The anthropometric measurements were compared to the WHO standards, whereas the NFCS used the CDC charts. When using the WHO charts, the prevalence of underweight is lower after six months of age in comparison with the CDC charts. Stunting rates are also higher when based on the WHO standard; estimates of overweight and obesity will be higher; and estimates of wasting and severe wasting will be lower after the seventy centimeters height cut-off (De Onis et al., 2007:145).

5.4.1.1. Weight-for-age

In the current study, 26,7% of **rural** children younger than seven were moderately underweight (weight-for-age <-2SD of WHO median) and 5% were severely underweight (weight-for-age <-3SD of WHO median) (Table 5.8). In the NFCS, there were only about half as many children (12,8%) moderately underweight (weight-for-age <-2SD of NCHS median) and about one-fifth (1,8%) as many severely underweight (weight-for-age<-3SD of NCHS median) children (Steyn et al., 2005:8, Table 3).

Among the **urban** children, 10,4% of children were moderately underweight (weight-for-age <-2SD of WHO median) and 6,9% severely underweight (weight-for-age <-3SD of WHO median). This was more in line with the NFCS as to moderately underweight (weight-for-age <-2SD of NCHS median) children (7,7%), but the severely underweight (weight-for-age<-3SD of NCHS median) children were far less (1,1%) in the NFCS (Steyn et al., 2005:8, Table 3).

Overall, 15,9% of **all** children in the current study were moderately underweight {weight-for-age <-2SD of WHO median] in comparison to the Free State section of the NFCS 14,3% (Labadarios & Nel, 2000:174; Labadarios & Nel, 2000:202, Table 4.14). Nationally, there was a prevalence of underweight of 10,3% among NFCS children and 9,3% among SAVACG children. In the current study 6,3% of all children were severely underweight (weight-for-age <-3SD of WHO median) in comparison to only 1,4% of NFCS children nationally, 1,4% of SAVACG children and only 1% in the Free State section of the NFCS (Steyn et al., 2005:8, Table 3; Labadarios & Nel, 2000: 174, 202, Table 4.14; Labadarios & Nel, 2000:215-216, Tables 4.31- 4.32).

One would expect that using the WHO standards, the occurrence of underweight would be lower (after six months of age) than when using the CDC charts (De Onis et al., 2007:145). However, a higher percentage of children were underweight than reported in the national and Free State NFCS.

Table 5.8 Comparison of current study's weight-for-age results with NFCS and SAVACG results

Reference														Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:8, Table 3; Labadarios & Nel, 2000:194, Table 4.2; Labadarios & Nel, 2000:202, Table 4.14			Labadarios & Nel, 2000: 174, 202, Table 4.14	Labadarios & Nel, 2000: 215, Table 4.31 Labadarios & Nel, 2000: 216, Table 4.32
Study	**Current study (AHA)**													**NFCS (National)**			**NFCS**	**SAVACG**
Year of study	***2006 (rural); 2009 (urban)***													***1999***			***1999***	***1995***
Group	**Boys**				**Girls**				***All rural***	***All urban***	***All boys***	***All girls***	***All***	***Rural***	***Urban***	***National***	***Free State***	***National***
	Rural		***Urban***		***Rural***		***Urban***											
Age range (years)	***0 - 7***		***0 - 7***		***0 - 7***		***0 - 7***		***0 - 7***		***0 – 7***		***0 - 7***	***1 - 8***			***1-9***	***1-6***
N	32		69		28		47		60	116	101	75	176	1304	1309	2570	203	11238
	N	%	N	%	N	%	N	%	%	%	%	%	%	%	%	%	%	%
>+3SD (Severely overweight)	0.0	0.0	1.0	1.5	1.0	3.6	0.0	0.0	1.7	0.9	1.0	1.3	1.2					
+2.1 to +3SD (Moderately overweight)	0	0.0	3.0	4.4	0.0	0.0	2.0	4.3	0.0	4.4	3.0	2.7	2.9	2.2	4.9	3.7	6.4	
+1.1 to +2SD (Mildly overweight)	0	0.0	7.0	10.1	2.0	7.1	6.0	12.8	3.3	11.2	6.9	10.7	8.5	3.5	9.3	6.7		
-1SD to +1SD (Normal)	12.0	37.5	31.0	44.9	11.0	39.3	21.0	44.7	38.3	44.8	42.6	42.7	42.6	53.6	53.7	53.7		
-1.1SD to -2SD (Mildly underweight)	7.0	21.9	14.0	20.3	8.0	28.6	11.0	23.4	25.0	21.6	20.8	25.3	22.7	29.7	24.9	27.0		
-2.1SD to -3SD (Moderately underweight)	11.0	34.4	10.0	14.5	5.0	17.9	2.0	4.3	26.7	10.4	20.8	9.4	15.9	12.8	7.7	10.3	14.3	9.3
<-3SD (Severely underweight)	2.0	6.3	3.0	4.4	1.0	3.6	5.0	10.6	5.0	6.9	5.0	8.0	6.3	1.8	1.1	1.4	1.0	1.4

Table 5.9 Comparison of current study's height-for-age results with NFCS results

Reference														Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:7, Table 2; Labadarios & Nel, 2000:194, Table 4.2; Labadarios & Nel, 2000:202, Table 4.14			Labadarios & Nel, 2000: 174, 202, Table 4.14	Labadarios & Nel, 2000: 215, Table 4.31 Labadarios & Nel, 2000: 216, Table 4.32
Study	**Current study (AHA)**													**NFCS**			**NFCS**	**SAVACG**
Year of study	***2006 (rural); 2009 (urban)***													***1999***			***1999***	***1995***
Group	**Boys**				**Girls**				***All rural***	***All urban***	***All boys***	***All girls***	***All***	***Rural***	***Urban***	***All***	***Free State***	***National***
	Rural		***Urban***				***Urban***											
Age range (years)	***0 - 7***		***0 - 7***		***0-7***		***0 - 7***		***0 - 7***		***0 – 7***		***0 - 7***	***1 - 8***			***1-9***	***1-6***
N	32		69		28		47		60	116	101	75	176	1304	1309	2570	203	10819
	N	%	N	%	N	%	N	%	%	%	%	%	%	%	%	%	%	%
>+3SD (Very tall)	0	0	1	1.5	1	3.8	1	2.1	1.8	1.7	1.0	2.7	1.8					
+2.1 to +3SD (moderately tall)	0	0	1	1.5	0	0	1	2.1	0.0	1.7	1.0	1.3	1.1	2.4	3.3	2.9		
+1.1 to +2SD (mildly tall)	1	3.1	4	5.8	0	0	1	2.1	1.7	4.3	4.9	1.3	3.4	4.9	8.2	6.7		
-1SD to +1SD (Normal height)	10	31.3	20	29	12	42.9	16	34	36.7	31.0	29.7	37.3	33.0	40.7	44.4	42.8		
-1.1SD to -2SD (mildly stunted)	6	18.8	10	14.5	7	25	10	21.3	21.7	17.3	15.9	22.7	18.8	28.0	28.5	28.3		
-2.1SD to -3SD (moderately stunted)	7	21.9	20	29	4	14.3	13	27.7	18.4	28.5	26.8	22.7	25.0	26.5	16.7	21.6	29.6	22.9
<-3SD (severely stunted)	8	25	13	18.8	4	14.3	5	10.6	20.0	15.5	20.8	12.0	17.0	8.4	4.7	6.5	10.8	6.6

When comparing **boys with girls**, twice as many boys (20,8%) were moderately underweight (weight-for-age <-2SD of WHO median) than girls (9,4%). Less boys (5%) were severely underweight (weight-for-age <-3SD of WHO median) than girls (8%). In total, 25,8% of boys were moderately or severely underweight (weight-for-age <-2SD of WHO median plus weight-for-age <-3SD of WHO median) and 17,4% of girls (Table 5.8).

Overall, rural children (especially boys) were far more likely to be underweight than their urban counterparts. Barker and his colleagues have shown that children who were underweight at one year of age had a greater tendency to store fat in the visceral area as adults (Popkin et al., 1996:3009), which increases the risk for CDLs in adulthood (Vorster & Kruger, 2007:323) Children who are underweight are at increased risk of infections, e.g. diarrhea and pneumonia, with an increased mortality risk (WHO, 2008:14; Nannan et al., 2007:733). Undernourished children younger than two years who gain weight rapidly later in childhood (after two years of age up to adolescence) have a higher risk for CDLs (Victora et al., 2008:340).

The NFCS combined the values for their moderately and severely **overweight** children; all children who had a weight-for-age ≥2SD of NCHS median were termed “obese” (Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:8, Table 3). In the current study, there were no rural children found to be moderately overweight (>2SD of WHO median) and there were 1,7% of children who were severely overweight (>3SD of WHO median) (total 1,7% of children = “obese”). The current study had more urban children (4,4%) who were moderately overweight (>2SD of WHO median), but less children (0,9%) who were severely overweight(>3SD of WHO median) (total 5,3% = “obese”). In comparison, the NFCS found 2,2% of their rural children to be “obese” and 4,9% of their urban children to be “obese.” These values are comparative to the current study’s results. Overall, from all the children in the current study, 2,9% of children were moderately overweight (>2SD of WHO median) and 1,2% were severely overweight (>3SD of WHO median), totalling 4,1% with a weight-for-age ≥2SD (Table 5.8). These results again compare well with the NFCS findings of 3,7% nationally (Labadarios et al., 2005:536-537, Tables 1-2; Steyn et al., 2005:7-10, Tables 2-6) and 6,4% in the Free State (Labadarios & Nel, 2000:175; Labadarios & Nel, 2000:202, Table 4.14).

The same percentage of girls in the current study (4%) were found to be overweight/obese than the boys (4%), which was a very small percentage of the total sample.

Overall, the current study’s findings correlate well with the results of the NFCS in terms of overweight/obese children, even after taking into account that the WHO standard analysis may find more children to be overweight/obese in comparison with a NCHS chart analysis (De Onis et al., 2007:145).

Urban children had a higher prevalence of overweight/obesity. Overweight in childhood and early life is liable to be followed by overweight and obesity in adulthood (WCRF/AICR, 2007:xvii). The later the weight gain in childhood and adolescence, the greater the persistence. Victora et al. (2008:340) found in their systematic review that rapid weight gain among in the first two years of life in children with poor fetal growth did not increase the risk of CDLs, However, undernourished children younger than two years who gained weight rapidly later in their childhood or adolescence did have a high risk for CDLs

(Victora et al., 2008:340). More than sixty percent of overweight children aged five to ten years, have at least one additional risk factor for CVD, such as hypertension, hyperlipidemia or hyperinsulinemia, and more than 25% have two or more risk factors (Goedecke et al., 2005:70; WHO/FAO, 2002:37). Childhood obesity also results in the development of co-morbidities of obesity in adulthood, since childhood obesity often persists into adulthood. Childhood obesity has a greater effect on developing the metabolic syndrome than becoming obese as an adult (Goedecke et al., 2005:70; Vanhala et al., 1999:657-658; Vanhala et al., 1998:319-320).

5.4.1.2. Height-for-age

Among **rural children** in the current study, 18,4% were moderately stunted (height-for-age <2SD of the WHO median) and twenty percent were severely stunted (height-for-age <3SD of the WHO median) (total 38,3%) (see Table 5.9). In comparison, the NFCS found 26,5% of their rural children were moderately stunted (height-for-age <2SD of NCHS median) and 8,4% were severely stunted (height-for-age <3SD of NCHS median) (total 34,9%) (Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:7, Table 2). About a quarter (28,5%) of the current study's **urban** children, were moderately stunted (height-for-age <2SD of the WHO median) and 15,5% were severely stunted (height-for-age <3SD of the WHO median) (total 43,9%); in comparison to the NFCS's 16,7% of moderately stunted (height-for-age <2SD of NCHS median) and 4,7% severely stunted (height-for-age <3SD of NCHS median) (total 21,4%) (Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:7, Table 2). Thus, the rural population's finding were equivalent to the NFCS findings for rural children, but the current study's urban population's values for stunting were two-fold those of the NFCS's findings for urban children.

About a quarter (26,8%) of the current study's **boys** were moderately stunted and a further 20,8% were severely stunted. About one-fifth (22,7%) of **girls** were moderately stunted, and a further 12% were severely stunted. This amounts to almost half (47,6%) of the boys and a third (34,7%) of girls being either moderately or severely stunted.

Overall, 25% of children were moderately stunted (height-for-age <2SD of the WHO median) in comparison with the NFCS's findings of 21,8% (height-for-age <2SD of NCHS median); and another 17% of all children in the current study were severely stunted (height-for-age <3SD of the WHO median) in comparison to 6,5% for the NFCS (height-for-age <3SD of NCHS median) (Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:7, Table 2). The SAVACG found it to be 22,9% moderately stunted, and 6,6% severely stunted nationally (Nel & Labadarios, 2000; 215-216, Tables 4.31 – 4.32). Using WHO standards (current study) may have found a higher occurrence of stunting in comparison with the use of NCHS charts (NFCS). However, Labadarios et al. (2000:870) does state that in the NFCS, stunting was the highest in the Free State province (29,6%), together with the Northern Cape (29,6%) (Labadarios & Nel, 2000:174; Labadarios & Nel, 2000:202, Table 4.14). The prevalence of severe stunting was second highest in the Free State (10,8%), after the Northern Cape (14%) (Labadarios & Nel, 2000:174; Labadarios & Nel, 2000:202, Table 4.14).

Schrimpton and Kachondham (2003:3) state that stunted children are more likely to fall ill and die, compared to underweight or wasted children. Short stature is associated with an increased risk for

Table 5.10 Comparison of current study's weight-for-height results with NFCS and SAVACG results

Reference														Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:9, Table 4, Labadarios & Nel, 2000:194, Table 4.2; Labadarios & Nel, 2000:202, Table 4.14			Labadarios & Nel, 2000: 174, 202, Table 4.14	Labadarios & Nel, 2000: 215, Table 4.31 Labadarios & Nel, 2000: 216, Table 4.32
Study	**Current study (AHA)**													**NFCS**			**NFCS**	**SAVACG**
Year of study	***2006 (rural); 2009 (urban)***													**1999**			**1999**	**1995**
Group	**Boys**				**Girls**				***All rural***	***All urban***	***All boys***	***All girls***	***All***	***Rural***	***Urban***	***National***	***Free State***	***National***
	Rural		***Urban***		***Rural***		***Urban***											
Age range	***0 - 7***		***0 - 7***		***0 - 7***		***0 - 7***		***0 - 7***		***0 - 7***		***0 - 7***	***1 - 8***			***1-9***	***1-6***
N	28		58		23		41		51	99	86	64	150	1304	1309	2570	203	10819
	N	%	N	%	N	%	N	%	%	%	%	%	%	%	%	%	%	
>+3SD (Severely high)	0	0	5	8.6	0	0	1	2.4	0.0	6.0	5.8	1.5	4.0					
+2.1 to +3SD (Moderately high)	1	3.6	6	10.3	2	8.7	5	12.2	5.9	11.1	8.1	10.9	9.3	4.54	7.97	6.6		
+1.1 to +2SD (Mildly high)	4	14.3	12	20.7	5	21.7	10	24.4	17.6	22.2	18.6	23.4	20.7	11.99	12.75	12.4		
-1SD to +1SD (Normal)	10	35.7	26	44.8	12	52.2	17	41.5	43.1	43.4	41.8	45.3	43.3	63.18	63.95	63.6		
-1.1SD to -2SD (Mildly low)	9	32.1	3	5.2	2	8.7	4	9.8	21.5	7.1	14.0	9.4	12.0	15.13	13.24	14.1		
-2.1SD to -3SD (Moderately low)	3	10.7	5	8.6	1	4.4	2	4.9	7.9	7.1	9.3	4.7	7.3	4.9	2.4	3.7	3.4	2.6
<-3SD (Severely low)	1	3.6	1	1.7	1	4.4	2	4.9	4.0	3.0	2.3	4.7	3.3	1.3	0.4	0.8	2.0	0.4

Table 5.11 Comparison of current study's BMI results with NFCS results

Reference										Steyn et al., 2005:9,Table 5		
Study	**Current study (AHA)**									**NFCS**		
Group	**Boys**		**Girls**		***All rural***	***All urban***	***All boys***	***All girls***	***All***	***Rural***	***Urban***	***All***
	Rural	***Urban***	***Rural***	***Urban***								
N	37	76	37	54	74	130	113	91	204	982	1218	2200
Median (kg/m^2)	15.2	16.4	15.8	16.5	15.5	16.4	16.0	16.2	16.1	15.7	15.9	15.8

CHD, stroke and DM in adulthood (WHO/FAO, 2002:35). Stunting is linked to an increased risk of delayed motor and mental development as well as of morbidity and mortality (Labadarios et al., 2000:870). Victora et al. (2008:345) found in their systematic review that height-for-age predicts cognitive performance later in life. Stunting in childhood can persist into adulthood, leading to decreased work capacity (Labadarios et al., 2000:870).

5.4.1.3. Weight-for-height

In the current study, 7,9% of all **rural** children were moderately wasted (weight-for-height <2SD of the WHO median) and 4% were severely wasted (weight-for-height <3SD of the WHO median), whereas 4,9% of rural children in the NFCS were moderately wasted (weight-for-height <2SD of NCHS median) and a further 1,3% were severely wasted (weight-for-height <3SD of NCHS median) (see Table 5.10). Among **urban** children in the current study, 7,1% were moderately wasted (weight-for-height <2SD of the WHO median) and another 3% were severely wasted (weight-for-height <3SD of the WHO median), compared to 2,4% of urban children in the NFCS being moderately wasted (weight-for-height <2SD of NCHS median) and only 0,4% severely wasted (weight-for-height <3SD of NCHS median) (Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:9, Table 4).

The results for rural and urban children in the current study are more or less the same, whereas there were more wasted children in the rural areas of the NFCS than in the urban areas. The current study's rural values (11,7%) are about forty percent more than those reported in the NFCS (6,2%). The urban values (10,1%) are about five times more than the NFCS's values (2,8%).

More **boys** (11,6%) than **girls** (9,4%) were wasted in the current study. Almost one-tenth (9,3%) of boys were moderately wasted vs. half the amount of girls (4,7%). Furthermore, another 2,3% of boys were severely wasted vs. 4,7% of girls.

Overall, 7,3% of children in the current study were moderately wasted (weight-for-height <2SD of the WHO median) and 3,3% were severely wasted (weight-for-height <3SD of the WHO median) (total 10,6%). In comparison, the NFCS found only 3,7% of children were moderately wasted (weight-for-height <2SD of NCHS median) and only 0,8% were severely wasted ((weight-for-height <3SD of NCHS median) nationally (total 4,5%) (Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:9, Table 4). For the Free State part of the NFCS, 3,4% of children were moderately wasted, and 2% were severely wasted (Labadarios & Nel, 2000: 174, 202, Table 4.14). In the SAVACG, 2,6% of children were moderately wasted in 1995, and 0,4% were severely wasted (Labadarios & Nel, 2000:215-216, Tables 4.31 – 4.32). The current study's occurrence of wasting was about double that found in the NFCS (nationally). Nel and Labadarios (2000:178) state that, when comparing the results of the NFCS to the SAVACG survey (conducted in 1995), the prevalence of wasting appeared to have decreased at the time of their study (1999). However, it seems that values for the Free State have increased since 1999, since when basing estimates on the WHO standard; estimates of wasting and severe wasting were supposed to decrease in comparison with the NCHS estimates (De Onis et al., 2007:145). However, this was not the case in the current study. Although wasting prevalence in the current study was low in comparison to stunting and underweight values, as was also the case in the NFCS (Labadarios et al., 2005:536) wasting is a sign of acute current undernutrition, and is of concern, since

the mortality rate in children younger than five with severe wasting ranging from thirty percent to fifty percent (WHO, 2012a:online).

5.4.1.4. BMI

The findings for median BMI among rural children (15,5 kg/m^2) were similar to the NFCS (15,7 kg/m^2) (see Table 5.11). However, the urban children's median BMI (16,4 kg/m^2) was higher than that found in the NFCS nationally (15,9 kg/m^2). Overall, the mean BMI for all children in the current study (16,1 kg/m^2), was comparative to the 15,8 kg/m^2 for the NFCS (Steyn et al., 2005:9,Table 5).

5.4.2 Adults

Anthropometric findings of the current study results were compared with results from the 1998 SADHS (Bourne et al., 2002:160, Table 1); the 2003 SADHS (DoH, 2004a:25-26, Tables 19.1 & 19.2); the CORIS study (Mollentze et al., 1995:92-93, Tables II & III); the VIGHOR study (Oosthuizen et al., 1998:1213) and the THUSA study (Vorster et al., 2005:486, Table 5; Vorster, 2002:240, Table 1). Comparison of the current study's results with the results from other studies are set out in Tables 5.15 through 5.18.

5.4.2.1. Underweight

In the current study, 33,1% of **rural** men and 9,2% of rural women were underweight (BMI<18,5 kg/m^2) (Table 5.12). In the THUSA study, 86,7% of rural men and 52% of rural women in tribal villages; and 90,6% of rural men and 46,9% of women on commercial farms had a BMI between <18,0 kg/m^2 and 24,9 kg/m^2 (Vorster et al., 2005:486, Table 5). In the 2003 SADHS, 13,8% of rural men and 6,8% of rural women were underweight (DoH, 2004a:25-26, Tables 19.1 & 19.2). The current study's findings for women are similar to the 2003 SADHS (nationally), but more men had a BMI<18,5 kg/m^2 than in the SADHS.

When taking the prevalence of HIV into consideration among the male study participants (17% rural, 41% urban) (found in separate sub-study; unpublished), as well as the high levels of physical activity (also found in separate sub-study; unpublished) (two-thirds of rural men were either active or very active - Table 5.7), it is not surprising that a third of rural men (33,1%) and about a quarter of urban men (23%) were underweight.

Among **urban** participants in the current study, 23% of men and 8,8% of women were underweight. In the THUSA study, 93,3% of men from squatter camps; 87,3% of township men; and 66,7% of upper class urban men had a BMI between <18,0 kg/m^2 and 24,9 kg/m^2 (Vorster et al., 2005:486, Table 5). The 2003 SADHS found nationally, that 11,7% of urban men and 5,6% of urban women were underweight (DoH, 2004a:25-26, Tables 19.1 & 19.2). Again, the percentage of men with a BMI<18,5kg/m^2 in the current study was higher than those of the SADHS (Table 5.13) and for women the findings are almost similar.

Of **all men** (rural and urban) in the current study, 29,3% were underweight, whereas only 9% of all women were (Table 5.12). In the 1998 SADHS, 12,9% of men and 4,8% of women were underweight

Table 5.12 Comparison of current study's BMI results with THUSA results

	Current study (AHA)									THUSA (Vorster et al., 2005:486, Table 5)									
Group	**Males**		**Females**		***All rural***	***All urban***	***All men***	***All women***	***All***	**Group 1**		**Group 2**		***Group 3***		***Group 4***		***Group 5***	
	Rural	***Urban***	***Rural***	***Urban***						♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
<18.5 kg/m^2 (Underweight)	33.1	23.0	9.2	8.8	16.2	12.2	29.3	9.0	14.5	86.7	52	90.6	46.9	93.3	52.8	87.3	38.6	66.7	38.7
18.5 – 24.9 kg/m^2 (Normal)	43.6	61.0	25.1	25.1	30.6	33.7	50.2	25.1	31.9										
25 – 29.9 kg/m^2 (Overweight)	15.3	12.0	22.3	25.1	20.2	22.0	14.0	23.6	21.0	8.7	25.3	6	27.9	3.7	18.8	8.5	25	30.9	32.1
≥30 kg/m^2 (Obese)	8.0	4.0	43.3	41.1	32.9	32.2	6.5	42.3	32.6	4.6	22.7	3.4	25.1	3	28.4	4.2	36.3	2.4	29.2

Table 5.13 Comparison of current study's BMI results with SADHS results

Reference	**Current study (AHA)**													**Bourne et al., 2002:160, Table 1**		**DoH, 2004a:25, Table 19.1**				**DoH, 2004a:26, Table 19.2**			
Study														**1998 SADHS**		**2003 SADHS**				**2003 SADHS**			
Group	**Males**				**Females**				***All rural***	***All urban***	***All ♂***	***All ♀***	***All***			**Men**				**Women**			
	Rural		***Urban***		***Rural***		***Urban***							♂	♀	***Rural***	***Urban***	***All***	***Free State***	***Rural***	***Urban***	***All***	***Free State***
N	163		100		390		319		553	419	263	709	972	4006	5897	1093	2214	3307	237	1601	2878	4480	326
	N	%	N	%	N	%	N	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%
<18.5 kg/m^2 (Underweight)	54	33.1	23	23.0	36	9.2	28	8.8	16.2	12.2	29.3	9.0	14.5	12.9	4.8	13.8	11.7	12.4	18.0	6.8	5.6	6.0	7.7
18.5 – 24.9 kg/m^2 (Normal)	71	43.6	61	61.0	98	25.1	80	25.1	30.6	33.7	50.2	25.1	31.9	61.7	36.7	58.1	57.7	57.8	60.0	43.9	36.3	41.3	42.5
25 – 29.9 kg/m^2 (Overweight)	25	15.3	12	12.0	87	22.3	80	25.1	20.2	22.0	14.0	23.6	21.0	19.4	26.7	22.8	20.2	21.1	13.4	28.4	27.9	29.0	23.3
≥30 kg/m^2 (Obese)	13	8.0	4	4.0	169	43.3	131	41.1	32.9	32.2	6.5	42.3	32.6	6.0	31.8	5.3	10.7	8.7	8.6	20.9	31.0	23.3	26.2
Waist circumference (men≥ 102 cm; women≥88 cm)	16	9.8	4	4.0	221	57.9	155	48.6	43.7	38.0	7.6	53.7	41.2	6.4	43.3								

(Bourne et al., 2002:160, Table 1) and in the 2003 SADHS, 12,4% of men nationally and 6% of women nationally were underweight, compared with 18% of Free State men and 7,7% of Free State women (DoH, 2004a:25-26, Tables 19.1 & 19.2). The current study's findings are similar to the SADHS's findings for women, but for men the findings are almost two-fold that of the findings for men in the SADHS (see Tables 5.12 and 5.13). The FYFS (Steyn et al., 2000:149, Table III) used different cut off points in their study for underweight (BMI <20 kg/m^2), therefore their findings of 26,8% of underweight women cannot be compared with the current study (Table 5.15).

There seems to be a pattern of more men (rural and urban) being underweight than women in the current study, as was also found in other studies. This may be related to alcohol intake, especially since there was a higher than recommended intake of alcohol among 5% of urban men, but also due to higher physical activity levels among men (Table 5.7), with a significantly larger percentage of rural men (rural: 42,1%; urban: 12,6%) being classified as very active (p<0.0001) (found in separate sub-study; unpublished).

However, taking into account that about ten percent of both rural and urban women were underweight, the possibility of these women bearing low-birth-weight infants, which increases these infants' mortality risk, as well as risk for infections in childhood as well as CDLs in adulthood, cannot be discounted (Victora et al., 2008:346; WHO, 2008:14; Nannan et al., 2007:733, 734, 737; Levitt et al., 2005:58; WHO/FAO, 2002:31). Almost four percent (3,5%) of the burden from LBW is attributable to maternal underweight. In 2000, the burden attributable to underweight in both women and children accounted for 12,3% of deaths and 2,7% of all DALYs in South Africa (Nannan et al., 2007:733,736).

5.4.2.2. Overweight and obesity

In the current study, 15,3% of **rural** men and 22, 3% of rural women were overweight (BMI=25 to 29,9 kg/m^2), whereas 8% of rural men and 43,3% of rural women were obese (BMI≥30 kg/m^2) (Table 5.12). A significantly larger percentage of urban men (urban 61,0%; rural: 43,6%) were of a normal BMI (18.5 to 24.9 kg/m^2) than rural men (p=0.007) (Table 4.36). The Free State study by Mollentze et al. (1995:93) found that obesity prevalence ranged between 27,5% and 49% among rural women of different age groups and between 7,4% and 19,2% among rural men. It should be noted that the urban population data from the Mollentze et al. study (1995) came from the same area (namely Mangaung) than the current study. However, the rural population sample in the study conducted by Mollentze et al. (1995) was from the Eastern Free State (namely Qwa Qwa), and not the Southern Free State as in the current study.

In the THUSA study, 8,7% of tribal men and 6% of farm dwelling men were overweight, whereas 25,3% of women from tribal villages and 27,8% from farms were overweight; 4,6% of tribal men and 3,4% of farming men were obese, as well as 22,7% of women from tribal villages and 25,1% of women from commercial farms (Vorster et al., 2005:486, Table 5). In the 2003 SADHS, 22,8% of rural men and 28,4% of rural women were overweight; and 5,3% of men and 20,9% of women were obese (DoH, 2004a:25-26, Tables 19.1 & 19.2).

Of all **urban** men in the current study, 12% were overweight as opposed to 25,1% of urban women. Four percent of urban men and 41,1% of women were obese. In the THUSA study, 3,7% of men from squatter camps; 8,5% of men from townships and 30,9% of upper class urban men were overweight, whereas 18,8% of women from squatter camps; 25% of women from townships and 32,1% of upper class urban women were overweight (Vorster et al., 2005:486, Table 5). As far as obesity was concerned: 3% of men from squatter camps; 4,2% of men from townships; and 2,4% of upper class urban men were obese, whereas 28,4% of women from squatter camps; 36,3% of women from townships and 29,2% of upper class urban women were obese (Vorster et al., 2005:486, Table 5). The Free State study by Mollentze et al. (1995:93) found that the prevalence of obesity ranged from 31,1% to 54,3% among urban women and from 3% to 20,4%. among urban men.

Only 14,1% of all **men** in the current study were overweight and 6,5% were obese. This compares well with the Free State result of the 2003 SADHS (Table 5.13) (13,4% overweight men; 8,6% obese men)(DoH, 2004a:26, Table 19.1); the 1998 SADHS (19,4% overweight nationally and 6% obese) (Bourne et al., 2002:160, Table 1); the 2003 SADHS (21,1% overweight nationally and 8,7% obese (DoH, 2004a:26, Table 19.1) and the THUSA study (9,1% obese) (Vorster, 2002:240).

In the current study, 23,6% of **females** were overweight, which compares well with results from the 2003 SADHS's Free State section (23,3%)(DoH, 2004a:26, Table 19.2). The SADHS (26,7%) found the prevalence to be a little higher (Bourne et al., 2002:160, Table 1). There were more obese female participants in the current study (42,3%) in comparison with the 1998 SADHS (31,8%) (Bourne et al., 2002:160, Table 1); the Free State 2003 SADHS (26,2%); the national 2003 SADHS (23,3%)(DoH, 2004a:26, Table 19.2); and the THUSA study (29,4%)(Vorster, 2002:240). The FYFS found that 18,2% of their female students older than 24 years old were overweight and 6,5% were obese (total 24,7% with BMI$\geq$ 25 kg/m^2) (Steyn et al., 2000:149, Table III). In the CORIS study, the amount of overweight respondents increased as they aged, i.e. in the group of males aged twenty to 42 years the prevalence of respondents with BMI $\geq$25 kg/m^2 was fifty percent and for females 39,9%. By age 45 to 64 years, the prevalence was 76,7% in males and 75,2% in females (Wolmarans et al., 1988:13, Table I).

There is an overall pattern in all South African studies where more women than men are found to be overweight or obese. In the current study, the occurrence of obesity was much higher in women than in men (Tables 5.12 and 5.13). As Vorster and Nell (2001:S20) state, women may become obese due to a low-fat, high-carbohydrate diet, especially if they are also inactive. However, Vorster and Nell (2001:S20) also state that it is more likely to become overweight through overconsumption of fat, rather than carbohydrates, because fat is more energy-dense. It should be kept in mind that the mean energy intake of women in the current study was relatively low in comparison to some other studies, but there was a high consumption from the bread and cereals group among both urban and rural women, with more than half of urban (52,5%) and rural (57,1%) women consuming above the amount of recommended daily servings (Table 4.16).

Even though participants consumed less than the DRIs for energy, a large portion of participants, especially women, were overweight or obese (rural: 65,6%; urban: 66,2%) (Table 4.36). However,

when looking at physical activity results found in a separate leg of the AHA study, it was found that 23,4% of rural women were either sedentary or had low activity levels as opposed to 66,9% of urban women, with a significantly larger percentage of urban women classified as sedentary ($p<0.0001$) or with low activity levels ($p<0.0001$). Therefore, although rural women had higher intakes of energy, their activity levels were also higher. Also, in another separate study forming part of the AHA study, HIV-infection prevalence was much higher among urban participants (41%) than rural participants (17%). This finding could also have influenced BMI.

Kruger et al. (2002:426) also showed that overweight and obesity in black women of the North West Province in South Africa was not only related to diet, but also to inactivity, which illustrates that other lifestyle factors are also involved in increasing risk for CDLs.

There is clear evidence of a relationship between onset of obesity and cancer risk (WHO/FAO, 2002:35). Overweight and obesity are associated with an increased risk of type 2 DM, especially with central adiposity (Goedecke et al., 2007:69; WHO/FAO, 2002:73). Obesity-related type-2 DM among black South-Africans presents with insulinopenia, leading to the metabolic syndrome (Van der Merwe & Pepper, 2006:5). Obesity is associated with hypertension in the black South African population (Seedat, 2007:318; Vorster, 2002:241, Table 1; DoH et al., 2002; 244). Overweight, specifically central obesity, together with high blood pressure, dyslipidemia, DM and low cardio-respiratory fitness all increase the risk for CVD (WHO/FAO, 2002:81). The Interheart study found that among black participants the BMI was significantly higher ($p=0.003$) in the MI cases as opposed to controls (Table 2.21)(Steyn, 2005b:251, Table 18.1). The risk for CHD also increases with obesity, with a relative risk of approximately 2,8 for men and 3,4 for women (Goedecke et al., 2005:69).

The direct costs of obesity in South Africa, may be as high as 6,8% of healthcare costs, and indirect and intangible costs should also be considered (Steyn et al., 2006:21). Because the risk for CDLs rise continuously with an increase in weight, the prevention of obesity can also lead to the prevention of CDLs (WHO/FAO, 2002:61).

When looking at the difference in prevalence of overweight and obesity in the current study between rural and urban men; and between rural and urban women, there is more similarity than difference. Urban women are slightly more overweight and slightly less obese than rural women.

Table 5.14 Comparison of median BMI values

	Men		Women		*Rural*	*Urban*	*Men*	*Women*	*All*
	Rural	***Urban***	***Rural***	***Urban***					
N	***163***	***100***	***390***	***319***	***553***	***419***	***263***	***709***	***972***
Current study	20.2	19.7	28.0	27.9	25.7	25.9	20.0	28.0	25.8
VIGHOR (mean)	-	26.9	-	25.4					
FYFS (mean) >24 yrs old								22.8	

The current study's median BMI values were lower than the values from the VIGHOR study's findings (mean BMI)(Table 5.14) (Oosthuizen et al., 1998:1213), whereas the FYFS's mean findings were lower (Table 5.15)(Steyn et al., 2000:149, Table III). The WHO's Comparative Risk Assessment Study estimated that an increase in BMI >21 kg/m^2 in adults older than thirty years, was associated with type

2 DM (58% of cases); IHD (21%); hypertension (39%); ischemic stroke (23%), colon cancer (12%); postmenopausal breast cancer (8%), endometrial cancer (32%); and osteoarthritis (13%) (Joubert et al., 2007:684). In the study by Mollentze et al. (1995:93), the mean BMI for men in both rural and urban areas was within normal ranges (20 to 25 kg/m^2), whereas the mean BMI for women exceeded 25 kg//m^2 in both rural and urban areas. Therefore the women in the current study, but not the men, had an increased risk for CDLs when looking at median BMI values.

Table 5.15 Comparison of current study's BMI results with FYFS results

	Current study (AHA)									Steyn et al., 2000:149, Table III	
										FYFS	
Group	**Males**		**Females**		***All rural***	***All urban***	***All ♂***	***All ♀***	***All***	***Females***	
	Rural	***Urban***	***Rural***	***Urban***							
<18.5 kg/m^2 (Underweight)	33.1	23.0	9.2	8.8	16.3	12.2	29.3	9.0	19.2	<20 kg/m^2 (underweight)	26.8
18.5 – 24.9 kg/m^2 (Normal)	43.6	61.0	25.1	25.1	30.6	33.7	50.2	25.1	37.7	20 – 24.9 kg/m^2 (normal)	48.5
25 – 29.9 kg/m^2 (Overweight)	15.3	12.0	22.3	25.1	20.3	22.0	14.1	23.6	18.9	25 – 29.9 (overweight)	18.2
≥30 kg/m^2 (Obese)	8.0	4.0	43.3	41.1	32.9	32.2	6.5	42.3	24.4	≥ 30 (obese)	6.5

5.4.2.4 Waist circumference

The percentage of men with a waist circumference ≥102 cm was 7,6% in the current study in comparison to 6,4% nationally in the 1998 SADHS (Bourne et al., 2002:160, Table 1). More than half (53%) of all women in the current study had a waist circumference ≥88 cm, compared to 43,3% of women nationally in the 1998 SADHS (Bourne et al., 2002:160, Table 1), whereas only 4,4% of the FYFS's female students older than 24 years had a waist circumference of ≥ 88cm (Steyn et al., 2000:149, Table III). The increasing waist circumferences among both men and women in the current study in comparison to the SADHS results, may infer an increasing risk for type 2 DM; hypertension; dyslipidemia, stroke and atherosclerosis (Gee et al., 2008:533, 541; Lee & Nieman, 2010:181; Goedecke et al., 2005:68; WHO/FAO, 2002:74). Central obesity is associated with insulin resistance, DM, hypertension, dyslipidemia, stroke and atherosclerosis (Gee et al., 2008:533, 541; Lee & Nieman, 2010:181; Goedecke et al., 2005:68). Central adiposity is also the cornerstone of the metabolic syndrome (Gee et al., 2008:533, 541; Goedecke et al., 2005:68; Kruger et al., 2005:494; Laquatra, 2004:568; WHO/FAO, 2002:73-74).

There was a far larger percentage of women in the current study who had a high waist circumference than men, which means that they carry a far higher risk for developing CDLs. A larger percentage of urban participants (men: 94%; women: 32%) had a normal waist circumference in comparison to rural participants (men: 79,8%; women: 24,4%).

5.5 The double burden

The high prevalence of underweight in children and obesity in adults represents a co-existence of over- and undernutrition, sometimes seen in the same household in developing countries. Caregivers or mothers may be obese, while the children are undernourished (Vorster & Kruger, 2007:323; Goedecke et al., 2005:71; Steyn et al., 2005:10; WHO/FAO, 2002:61; Faber et al.,

2001:410). This phenomenon includes the double burden of undernutrition-related infections and the overnutrition-related CDLs (Vorster & Kruger, 2007:323). The co-existence of undernutrition and obesity among the poor places a burden on social-, economic- and health care systems (Sawaya & Roberts, 2003:online).

5.5.1 Underweight

The double burden was present, since 66,7% of rural underweight children and 66,7% of urban underweight children lived with an overweight/obese caregiver. A high prevalence of overweight and obesity amongst caregivers was also found in the same household as underweight or stunted children in the Lebowa study (Steyn et al., 1995:53-59), as well in the study conducted by Faber et al. (2001:410) in KwaZulu-Natal.

In comparison, about one-third (31%) of rural underweight children in the Limpopo province were found to live with an overweight or obese caregiver, and nearly fifty percent of caregivers of stunted or underweight children in rural North West province were overweight or obese (Steyn et al., 2003:10).

5.5.2 Stunted

Again, the double burden was present, since 63,2% of rural stunted children and 71,9% of urban stunted children lived with an overweight/obese caregiver.

In previous studies, the relative risk for overweight if stunted, was found to be 2,6 among South African children (Steyn et al., 2005:12; Jinabhai et al., 2003:online; Faber et al., 2001:410; Popkin et al., 1996:3012). A high prevalence of overweight and obesity amongst caregivers was also found in other studies in the same household as underweight or stunted children (Goedecke et al., 2006:71; Sawaya et al., 2003:170; Faber et al., 2001:410). In the rural Northwest Province, nearly fifty percent of overweight mothers and/or caregivers lived with a stunted or underweight child (Steyn et al., 2005:10).

Stunted South African children are often found in the same households with overweight or obese caregivers, especially in rural areas (Vorster & Kruger, 2007:323; Goedecke et al., 2005:71; Steyn et al., 2005:5, 10; WHO/FAO, 2002:61; Faber et al., 2001:410).

5.5.3 Wasted

All of the wasted rural children and 75% of wasted urban children resided with overweight/obese caregivers. The high prevalence of wasting in these households can be indicative of a high level of food insecurity among children in these households.

Chapter 6 – Conclusions and Recommendations

6.1 Introduction

The main aim of this study was to investigate the diet and anthropometric status of adults (between 25 and 64 years old) and pre-school children (zero to seven years old) in rural and urban areas in order to determine whether a nutrition transition and double burden of undernutrition in children and overweight/ obesity in mothers or caretakers was present.

6.2 Conclusions

The nutrition transition in South Africa entails a shift from a traditional diet consisting of >60% to 65% of total energy from carbohydrates and <50% to 55% of total energy from fat to a Westernized diet consisting of <50 to 55% total energy from carbohydrates and >30% to 35% of total energy from fat (Joubert et al., 2007:684; Steyn et al., 2006:13,14; Steyn, 2005a:36; Bourne et al., 2002:157,159; WHO/FAO, 2002:19). As a traditional eating pattern is exchanged for a more Western eating pattern, diets usually become denser in energy, with fewer starchy foods and more fats; oils; sugars; and additives, as well as alcoholic drinks being ingested. At the same time, physical inactivity increases, with the need for energy decreasing and the rate of overweight and obesity increasing (WRCF/AICR, 2007:4). The traditional diet is associated with a low prevalence of degenerative diseases, whereas the Western diet is associated with an increased risk for developing CDLs (Bourne et al., 2002:157).

A nutrition transition was identified in the study population of both the rural and urban areas among children and adults. This was manifested in the dietary intake and anthropometric profiles of the samples. As found by a separate AHA sub-study, levels of physical activity differed significantly between urban and rural participants, indicating that a physical activity transition was not yet evident in rural populations (not published). As far as diet (of children and adults) and anthropometry (of adults) is concerned, a nutrition transition was confirmed in both rural and urban populations, with rural participants (especially women) often having a higher risk of CDLs than their urban counterparts. Although a number of factors could have played a role in this finding (older median age of rural participants and the possibility that rural participants who already had CDL were more likely to participate in the study due to the free medical examination that was performed), the most likely explanation would be the higher prevalence of HIV found in the urban sample (41%) compared to the rural sample (17%) (separate unpublished sub-study) and its impact on anthropometric variables.

6.2.1 Dietary intake

The intake of food groups, and the frequent intake of certain food items, reflect the presence of a nutrition transition in both rural and urban communities included in this study. Energy intake and macronutrient distribution in the diets were within prudent dietary guidelines.

6.2.1.1 Food groups

Dietary intake, as reflected by the intake of adequate numbers of foods from the different food groups, of both children and adults was mostly inadequate. According to this reference, the nutrition transition was more pronounced in the rural population, with both rural adults and children consuming above their daily recommended servings of fats and oils; sweets and sugar; and meat and meat products and eating fewer fruit and vegetables than their urban counterparts. However, keep in mind that results were only from a single 24 hour recall of reported usual intake.

On the other hand, urban adults and children tended to consume above their daily recommended servings of bread and cereals more often than their rural counterparts.

The intake of legumes and soy was low in both rural and urban participants.

6.2.1.2 Frequent intake of food items

In most instances sugar was the most frequently consumed food item, especially among rural participants. Cooked porridge was the most frequently consumed starchy food, followed by bread.

Tea was the most frequently consumed fluid. Sugar-sweetened beverages (cool drink) were also frequently consumed, especially by children. Men consumed coffee more frequently than the women and children.

Fruit and vegetables were consumed frequently, but fruit more so than vegetables. Fruit and vegetable intakes were, however, mostly below the recommended number of servings per day as evidenced by the intake of foods from the different food groups.

Salt and stock powders were used frequently during food preparation and chips and crisps were consumed frequently by children, but adults consumed them less frequently. Sweets were consumed frequently by urban women, as well as rural and urban boys. Margarine, oil and fat were frequently added during food preparation, and margarine was frequently spread onto bread. Full cream milk was the most frequently consumed dairy product.

Alcohol was consumed excessively by five percent of urban men, while rural men consumed alcohol less frequently.

6.2.1.3 Macronutrients

The average total energy intake of rural adults and children was higher than that of urban adults and children. In addition, the average percentage energy from protein and from fat was higher in rural adults and children than it was in their urban counterparts.

Children younger than two years consumed sufficient energy if compared to the DRI for children 7-12 months, but not when compared to the DRI for 1-to-3 year olds (Food & Nutrition Board et al., 2002:1324). The current study's rural children met the DRI for 1-to-3 year olds, but the urban children did not.

Children older than two years consumed sufficient energy if the DRIs for 1-3 years old were used, but were insufficient if DRIs for 4-8 years old were used (Food & Nutrition Board et al., 2002:1324). The sample sizes of children in this study were too small to divide children who were two years and older into smaller groups (e.g. one-to-three years old and four-to-eight years old).

Even though adult participants reported consuming less than the DRIs for energy, a large portion of participants, especially women, were overweight or obese.

The percentages of energy from macronutrients for carbohydrates fell within the recommended 45% to 65% for all groups. However, adults and rural children older than two years old consumed fat within the recommendations (25% to 35% for children; 20% to 35% for adults). The remaining participants consumed less than the recommended percentage range. Average percentage energy intake from protein was similar in urban and rural areas, and fell within recommendations for all groups.

Despite the relatively prudent energy intake and macronutrient distribution, the intake of foods from different food groups and the intake of specific foods (as determined by the FFQ) of a large percentage of both the rural and urban samples did not fall within recommendations for prudent intake.

6.2.1.4 Breastfeeding

The majority of babies were not breastfed at the time of the study. However, the majority of rural children (90,9%) were previously breastfed as opposed to only 37,5% of urban children.

The median period of exclusive breastfeeding was around four months for both rural and urban children, which was lower than the WHO recommendation of six months.

The median age for introduction of solid foods was six months in rural participants and four months in urban participants. The rural group thus generally waited till the recommended six months to introduce solid foods, but the urban children were weaned onto solids earlier than recommended.

6.2.2 Anthropometry

The numbers of adults included in the study were large, but due to the small numbers of children included in this sample (rural: n=60; urban: n=116) it was no possible to draw representative conclusions from the results of the children.

6.2.2.1 Undernutrition

Undernutrition can have long-lasting consequences on children's health, delay motor development, as well as impair cognitive function and school performance. In adults, it reduces work capacity and influences reproductive health (Victora et al., 2008:343,345; Nannan et al., 2007:733; Vorster & Kruger, 2007:322).

Keeping in mind that the impact of HIV status on nutritional status was not determined as part of the study, undernutrition was present in a large percentage of children and adult men, as reflected by the large number of stunted or wasted children; and underweight adults and children. In general, there was a larger percentage of boys that were underweight, stunted and wasted than girls. This finding is

supported by the results of other studies who have also found the prevalence of undernutrition to be higher in boys than girls. A higher prevalence of stunting in boys has occasionally been reported in Sub-Saharan Africa. In ten countries (including Cameroon, Ghana, Kenya, Malawi, Namibia, Nigeria, Tanzania, Uganda, Zambia and Zimbabwe) boys younger than five years were more likely to become stunted than girls. This sex difference was more pronounced in lower socio-economic groups, and might suggest that boys are more vulnerable to illness (Wamani et al., 2007:online). Epidemiological studies have also previously shown that morbidity and mortality was consistently higher in boys in early life, but the underlying mechanisms are unclear (Wamani et al., 2007:online).

When comparing the rural children to urban children, rural children were more often underweight (rural 31,5% vs. urban 17,2%) than urban children, but urban children were more often stunted than rural children (rural 38,3% vs. urban 43,9%). Prevalence of wasting was similar in rural and urban children (rural 11,7% vs. urban 10,1%). These findings are in contrast to the NFCS that have reported that rural children tend to be more chronically malnourished and stunted than urban children (Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:7, Table 2). As seen in the adult sample, the current study did not support the notion that rural communities in developing countries are worse off than urban communities.

In comparison to the NFCS's national findings, a larger percentage of children in the current study were stunted, underweight and wasted. The percentages of overweight children were similar to that reported in the NFCS (the very small number of overweight children is acknowledged). In comparison to the NFCS's findings for the Free State, the children in the current study tended to be more malnourished, which may indicate that in approximately ten years' time the nutritional status of children in the Free State may have deteriorated. It is also possible that mothers were more likely to bring their children to participate in the study if they were ill (a free medical examination was performed), which could have had an impact on the results related to growth status that were obtained.

(i) Stunting

Stunting, is a sign of chronic undernutrition and the result of an overall poor diet (The Save the Children Fund (SCF), 2012: 2). Stunting, as reflected by a low height-for-age, can have a significant effect on a child's health in both the short and the long-term (SCF, 2012:xiii; Victora et al., 2008:340). If a child was stunted in the first 1000 days of life, the damage done is largely irreversible, even if the child's diet improves after two years of age (SCF, 2012:xiii; Victora et al., 2008:340).

About half of all boys (47,6%) and a third of all girls (34,7%) were either moderately or severely stunted. Moderate stunting was present in about one in four urban children and one in five rural children. A further twenty percent of rural children and 15,5% of urban children were severely stunted. This finding is in contrast to the NFCS that have reported that rural children tend to be more chronically malnourished and stunted than urban children (Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:7, Table 2).

(ii) Wasting

Wasting is a sign of acute and severe undernutrition and is often the result of a sudden lack of food or illness in an already malnourished child (SCF, 2012:2). Wasting is reflected by a low weight-for-height.

A similar percentage (about 7%) of both rural and urban children were moderately wasted. There was also a similar percentage of severely wasted children in both rural (4%) and urban (3%) areas. Slightly more boys (11,6%) than girls (9,4%) were either moderately or severely wasted.

The results for rural and urban children in the current study are more or less the same, whereas there were more wasted children in the rural areas of the NFCS than in the urban areas (Labadarios et al., 2005:536, Table 1; Steyn et al., 2005:9, Table 4, Labadarios & Nel, 2000:194, Table 4.2; Labadarios & Nel, 2000:202, Table 4.14). The current study's rural values (11,7%) are about four percent higher than those reported in the NFCS (6,2%). The urban values (10,1%) are about five times higher than the NFCS's values (2,8%).

(iii) Underweight

Underweight individuals do not consume enough energy, in relation to their energy expenditure, to maintain a healthy body weight (Food & Nutrition Board et al., 2002:111). In children underweight is reflected by a low weight-for-age, and in adults by a BMI<18,5 kg/m^2. Undernourished women have a higher risk of having low birth weight babies, which increases both mortality risk and the risk of developing CDLs in adulthood. This problem can perpetuate even further into the next generation (Victora et al., 2008:346).

About one in four rural children and one in ten urban children were moderately underweight. Another 5% of rural children and 7% of urban children were severely underweight.

In the current study, 33,1% of rural men (one in every three rural men) and 23% of urban men (one in every four urban men) were underweight as opposed to 9,2% of rural women and 8,8% of urban women (almost one in every ten women). Although the results pertaining to physical activity were not included in the initial objectives of this study, the higher percentage of underweight rural men can be explained by the fact that a significantly larger percentage of rural men (rural: 42,1%; urban: 12,6%) were classified as very active ($p<0.0001$). Since a large number of rural men are farm labourers, the high percentage of underweight in rural men may be due to the high physical activity required by the occupation. In the case of urban men, the higher prevalence of HIV-infection and alcohol use in urban compared to rural participants may be responsible for the large percentage of underweight urban men.

6.2.2.2 Overnutrition

Overnutrition, as reflected by overweight and obesity, was present in a large percentage of both rural and urban women.

(i) Overweight and obesity

In overweight individuals, energy intakes are higher than energy expenditures (Food & Nutrition Board et al., 2002:111). On the one hand, childhood obesity is a strong predictor of obesity in adulthood (Joubert et al., 2007:688; WCRF/AICR, 2007:xvii); and on the other hand, undernutrition in children also predisposes them to overweight and obesity as adults. Overweight and obesity has far reaching consequences, both in childhood and in adulthood, including an increased risk of developing CDLs (Goedecke et al., 2005:70; WHO/FAO, 2002:37). In children, the prevalence of overweight and obesity (reflected by a high weight-for-age and high BMI-for-age) was very low in this sample with only four percent of both boys and girls in the urban area being overweight or obese. A high BMI reflected overweight or obesity in adulthood.

Almost two-thirds of both rural and urban women were either overweight or obese, as opposed to less than one in five rural or urban men. The MRC's Burden of Disease study, the 1998 SADHS and the THUSA study all found that a higher percentage of women than men were overweight or obese (Joubert et al., 2007:638, 685; Goedecke et al., 2005:65; Vorster, 2002:241, Table 2). The high percentage of participants that ate above recommended number of servings of fats and oils; sweets and sugar and meat and meat products can possibly explain this finding.

(ii) Central adiposity

Central obesity, as reflected by a high waist circumference, is associated with insulin resistance, DM, hypertension, dyslipidemia, stroke and atherosclerosis (Gee et al., 2008:533, 541; Lee & Nieman, 2010:181; Goedecke et al., 2005:68). Central adiposity is also the cornerstone of the metabolic syndrome (Gee et al., 2008:533, 541; Goedecke et al., 2005:68; Kruger et al., 2005:494; Laquatra, 2004:568; WHO/FAO, 2002:73-74). Increasing obesity due to an inactive lifestyle and a Westernized diet can contribute to the increasing prevalence of CDLs that was previously identified in the participants of this study (Van Zyl et al., 2012:online). Compared to the urban participants, rural participants had a higher prevalence of risk factors for metabolic syndrome (Van Zyl et al., 2012:online). The results related to diet and anthropometry obtained in this study can explain these findings.

As expected, a higher percentage of both rural and urban women than men had a waist circumference that placed them at risk for CDLs (central adiposity was present in two-thirds of both rural and urban women). This corresponded with the high percentage of women that were classified as either overweight or obese according to BMI. One in five rural men also had a high risk waist circumference which confirms a nutrition transition. Only six percent of urban men had central adiposity, which may be related to the higher occurrence of HIV infection and alcohol abuse in this group, compared to the rural men.

6.2.3 The double burden

The high prevalence of underweight in children and obesity in adults points to a co-existence of over- and undernutrition (i.e. the double burden), placing the children at a higher risk for CDLs in later life. This phenomenon has been reported in various other studies (Joubert et al., 2007:688; Vorster & Kruger,

2007:323; Goedecke et al., 2005:71; Steyn et al., 2005:10; WHO/FAO, 2002:61; Faber et al., 2001:410). Caregivers or mothers may be obese, while the children are undernourished. Where there is a high prevalence of obesity among black women, a high prevalence of stunting in children is found, particularly in rural areas (Steyn et al., 2005:5). The co-existence of undernutrition and obesity among the poor places a burden on social-, economic- and health care systems (Sawaya & Roberts, 2003:online).

Cameron (2003:38) states that stunting in infancy and childhood is linked to later obesity. Stunting itself confers an increased risk for overweight in children (Levitt et al., 2005:59). The association between short stature and a higher risk of CDL has been detailed earlier in this dissertation.

A double burden of overnutrition and undernutrition in the same household was confirmed in the current study. Two-thirds of both rural and urban children who were underweight resided with an overweight or obese caregiver. About two-third of rural- and 72% of urban children who were stunted resided with an overweight or obese caregiver. All the wasted rural children and three-quarters of the urban wasted children resided with an overweight or obese caregiver.The large percentage of stunted children in the study is of concern, since stunted children have a higher risk of being overweight or obese, either in childhood and/or adulthood (Goedecke et al., 2005:71; Mendez et al., 2005:720; Steyn et al., 2006:20; Faber et al., 2001:410; Popkin et al., 1996:3012).

6.3 Recommendations

To prevent CDLs in adulthood, it is critical that undernutrition in childhood be either prevented or addressed. In addition, relevant interventions to address existing overweight and obesity in adults also need to be emphasized.

To prevent or address the double burden of disease, the following challenges should receive attention:

- Undernutrition in children, particularly stunting; and
- Overweight and obesity in adults.

SCF (2012:77) mentions six steps to achieving nutrition transformation, which include:

- Make malnutrition visible;
- Invest in direct interventions;
- Fill the health worker gap;
- Protect families from poverty;
- Harness agriculture to help tackle malnutrition; and
- Galvanize political leadership.

With this in mind the following practical recommendations are made:

6.3.1 Healthier lifestyles

Because dietary intake among both rural and urban participants reflects the nutrition transition, nutrition interventions (emphasizing healthy food choices) should be implemented in both rural and urban areas.

Broadly, the interventions to address food intake patterns should entail reducing consumption of:

- saturated fats;
- unhealthy snack foods;
- sugar-sweetened beverages;
- salt; and
- alcohol;

and increasing consumption of:

- fruits and vegetables; and
- legumes.

In an effort to encourage healthier food choices, the SAFBDG, as they are currently available to health care workers in a printed format by the Department of Health, should be fundamental to nutrition education of all members of the community. Nutrition education programmes need to be implemented in community settings as part of the FSRDPP and the MUCPP, both of which are ideal forums through which the communities included in this study can be reached.

There are various practical recommendations that can assist in making communities aware of healthy lifestyle options:

- Printed media, e.g. the pamphlets and brochures that have been developed by organisations such as the Sugar Association of South Africa, which discuss the FBDG and specifically focus on how healthy eating can be made practical and relevant in the treatment or prevention of obesity, diabetes, hypertension and HIV/AIDS. These aids can be used in the clinic setting as well as by community health workers that do home visits.

- Health care workers can be trained on how to make scientific information understandable and easier to apply in communities where a large percentage of members are often illiterate. This can be done by giving talks in the clinics; placing posters with the key messages in visible areas; and also distributing the pamphlets in homes where a specific problem has been identified. Community health workers and community service dieticians can be of immense value in this regard. These dieticians can also play an important role in developing printed media that bring the message across in laymen's terms and can be used as a reminder of the guidelines that have been given during nutrition education.

- Planting of fruit trees and vegetable gardens that can improve the intake of fruit and vegetables can make a significant contribution to improving household food security. In addition, the sale of vegetables from community gardens can have a unique income generating potential. Dieticians

should collaborate with staff from other sectors, such as agriculture, to make sure that challenges to planting gardens (e.g. water shortages) are addressed and that communities are taught how to successfully plant gardens taking these challenges into account. Provision of vegetable seed, and education on how to make a garden with limited space, are important considerations. Children who are involved with planting and maintaining a vegetable garden will also be more likely to eat these vegetables.

- Other health care professionals, such as physiotherapists and biokineticists can also be recruited to become involved at clinics and in the workplace to counsel on healthy and safe exercises, especially to obese adults who also struggle with comorbidities such as diabetes and hypertension. Groups can be convened on specific days where community members can attend a thirty minute training session, to encourage sustainability.

- Special focus should be placed on the importance of health care workers setting a good example to the community. Health care workers should be encouraged to be role models, in order to more successfully convey the ideal lifestyle and relevant information to patients and people in the communities in a culturally acceptable way (teach by example

6.3.2 Stopping the vicious circle of undernutrition

Since the double burden of disease has been confirmed in the current study group, action is required to address and prevent stunting in these and other rural and urban communities. According to Vorster and Kruger (2007:322), nutrition- and other interventions that address poverty-related causes of undernutrition have the potential to improve child growth and development, especially if instituted in the first three years of life. According to the WHO/FAO (2002:68), nutrition programmes designed to control or prevent undernutrition, need to assess stature in combination with weight to prevent providing excess energy to children with a low weight-for-age but a normal weight-for-height. Global targets, specifically measuring stunting, should be implemented to ensure that nutrition is included in major political processes – linking directly to the Millennium Development Goals, and specifically the forty percent reduction in childhood stunting by 2022 (proposed by the WHO) (Rehman et al., 2009:online). In this regard, regular and reliable growth monitoring in the community setting needs to receive attention.

Nutrition as a basic human right, is reflected by the Constitution (Nannan et al., 2007:738) and addressing undernutrition successfully is essential in meeting the Millennium Development Goals. Ideally, the immediate, underlying and basic causes of undernutrition should all be addressed, but in reality this is not so easy to do in a fragmented manner and will require a multidisciplinary and inter-sectoral approach. One of the WHO's millennium development goals is to halve (from 1990 levels) the proportion of children younger than five who are stunted, wasted or underweight by 2015 (Rehman et al., 2009:online). The first Millennium Development Goal aims to reduce poverty and hunger between 1990 and 2015 (Nannan et al., 2007:733).

The 13 low-cost interventions to reduce stunting in children by 36% and child mortality by 25%, as set out by the SCF (2012:xiv), can go a long way to addressing undernutrition. These interventions

include: interventions to encourage change in behaviour to improve nutrition; micronutrient interventions; and therapeutic feeding interventions, e.g. ready-to-use foods (RUTFs) for severely malnourished children (SCF, 2012:20). The interventions to encourage change in behaviour to improve nutrition include: promoting breastfeeding for newborns at birth; promoting exclusive breastfeeding; promoting complementary feeding in addition to continued breastfeeding from six to 24 months of age; and encouraging better hygiene (e.g. encouraging hand washing). Micronutrient interventions include: increasing supplementation of zinc in infants and children; giving zinc to manage diarrhea; providing vitamin A either via fortification or supplementation; and iodization of salt. Micronutrient interventions in pregnant and breastfeeding mothers include: implementing multiple micronutrient supplementation by way of iodized salt; iron, folate and calcium supplements (SCF, 2012:20).

6.3.2.1 Getting the message on nutrition out

Children can also be provided with information on healthy living by:

- Incorporating topics on nutrition and exercise into school curricula, including basic knowledge of good nutrition, as well as how it can be incorporated into the family's nutrition practices and limited resources. The current emphasis on nutrients needs to be replaced with a more "food-based" approach that is easier to implement.

Nutrition week, which is held annually for a week, raises public awareness of nutrition and provides a good opportunity to get the message on nutrition across to everyone in communities. Information, especially on child nutrition, can be disseminated by various means:

- Posters with matching pamphlets containing the necessary information, which children and parents can take home. These can be placed in visible areas, such as entrances to shopping malls, hospitals, government offices, schools, etc.

Thinking more broadly:

- Emphasizing the message in the media, e.g. a government funded broadcasting campaign on television and radio to advertise and endorse the FBDG. Briefly focusing on each FBDG at different time slots can also be an option.

- Dieticians, community members or celebrities that are role models can become spokespersons in the media. These persons can promote the FBDG in the media

- Articles discussing the FBDG with interesting recipes related to the dietary guidelines can be published in various popular magazines and newspapers. Competitions can be arranged to encourage community participation.

Dieticians employed by the Department of Health, community service dieticians and community health workers can all play an important role in ensuring that the Integrated Nutrition Programme is implemented to its full potential by supporting the following:

- The Nutrition Supplementation programme.
- Growth monitoring.
- Vitamin A supplementation.
- School feeding schemes.
- Vegetable gardens.
- Promotion of breastfeeding and support of the Breastfeeding Hospital Initiative (BFHI), as well as the introduction of complementary feeding and continued breastfeeding after six months of age.
- Food fortification. Fortified food items should be affordable (either in the private or public sector); accessible and culturally acceptable.
- Provision of RUTF-type supplements to severely malnourished children.

By empowering women in communities, the health of especially children can be improved:

- Training should be provided to social workers and community service dieticians (among others) on how to teach women (especially in female-headed households) how to budget and spend available resources wisely.
- Information should also be provided to these women on what economical food choices entail and how they can make the most of the little money and food sources they have.
- Support and training of community health workers by nutrition experts (e.g. dieticians) in implementation and maintenance of nutrition programmes are important. Qualified dieticians who have the knowledge and expertise should be appointed in management positions where an in-depth knowledge of nutrition is required (e.g. as INP managers).

6.3.2.2 Government involvement

Government should be involved in making nutrition a priority in the country:

- Nutrition should be given the priority that it deserves for interventions to be successful (SCF, 2012:62). If governments support a programme, it will be much more likely to have the desired impact.
- The necessary support should be provided to other sectors that also support nutrition, such as agriculture.
- The viability of Social Protection Programmes can be investigated. In such programmes persons receiving support are expected to perform certain tasks in the community for a certain amount of hours in the month in exchange for food or money.
- Nutrition programmes, should be monitored and evaluated regularly to determine the impact of these interventions.

- Comprehensive nutrition screening and growth monitoring programmes need to be implemented to ensure the timely identification of problems and necessary nutrition support.
- Financial support of nutrition interventions should be assured, depending on the evidence-based impact of these programmes.
- Sustainable employment opportunities need to be supported by government in an effort to address the basic poverty-related causes of malnutrition.

6.4 Suggestions for further research

Based on the findings of this study, it is recommended that the associations between diet and anthropometry with other variables such as HIV/AIDS, levels of physical activity, and alcohol use be further investigated in the AHA FS dataset.

Summary

Stunting in childhood predisposes to obesity, increasing the risk for chronic diseases of lifestyle in adulthood (i.e. the double burden of disease). To plan relevant nutrition interventions, the burden of disease, and lifestyle factors that contribute to these, need to be determined. The aim of this sub-study of the Assuring Health for All in the Free State (AHA FS) study was to gain insight into the dietary intake and anthropometric nutritional status of pre-school children and adults aged 25 to 64 years in the rural and urban Free State. Rural areas included Trompsburg, Philippolis and Springfontein. Urban areas included communities located around the Mangaung University Community Partnership Program (MUCPP) Clinic.

Dietary intake was measured in 60 rural- and 116 urban children; and 553 rural- and 419 urban adults using a 24-hour recall of reported usual intake and qualitative food frequency questionnaire. Intake of foods was compared with the recommendations of the Food Guide Pyramid and categorised as above, below or within recommendations. Intakes of energy and macronutrients were estimated from the number of foods eaten from each food group and compared to prudent dietary guidelines. Anthropometric data was collected using standard measuring techniques and categorised using World Health Organization (WHO) standards.

At the time that the study was conducted, about one-third (31,24%) of rural children and 40,7% of urban children were breastfed. A significantly larger percentage (90,9%) of rural participants (95% confidence interval (CI) [20,0; 69,8]) were previously breastfed, compared to 37,5% of urban participants.

Rural adults and children consumed above daily recommended servings of fats and oils; sweets and sugar and meat and meat products and ate fewer fruit and vegetables than their urban counterparts. On the other hand, urban adults and children consumed bread and cereals above their daily recommended servings more often than their rural counterparts. The intake of legumes and soy was low in both rural and urban participants.

Sugar was the most frequently consumed food item, especially among rural participants. Cooked porridge was the most frequently consumed starchy food, followed by bread. Tea was the most frequently consumed fluid. Sugar-sweetened beverages (cool drinks) were also frequently consumed, especially by children. Men consumed coffee more frequently than women and children. Salt, stock powders, margarine, oil and fat were used frequently during food preparation and margarine was frequently spread onto bread. Fruit and vegetables were consumed frequently, but fruit more so than vegetables. Fruit and vegetable intake was, however, mostly below the recommended number of servings per day. Chips and crisps were consumed frequently by children. Sweets were consumed frequently by urban women, as well as rural and urban boys. Full cream milk was the most frequently consumed dairy product. Alcohol was consumed excessively by five percent of urban men.

Mean energy intake was 4254 kJ for rural children younger than two years (56,9% carbohydrates; 17,2% protein; 25,7% fat) and 3292kJ for urban children younger than two years (64,2% carbohydrates; 19,5% protein; 20,1% fat). The percentage of energy from carbohydrates and protein were within prudent dietary guidelines (carbohydrates (CHO): 45-65%; protein: 5-20%), while the percentage energy from fat was lower than the recommended 30-40%.

Mean energy intake for rural children older than two years was 5581kJ (57,5% carbohydrates; 16,9% protein; 28,7% fat) and 4220kJ for urban children (65,5% carbohydrates; 17,9% protein; 20,3% fat). As in the younger children, the percentage of energy from carbohydrates and protein were within prudent dietary guidelines (CHO:

45-65%; protein: 10-30%; fat: 25-35%) except for fat intake which was lower than recommended among urban participants.

The average energy intake for all men was 8040 kJ (61% carbohydrates; 17,8% protein; and 24,3% fat) and for all women in the current study was 7243 kJ (61,7% carbohydrates; 17,3% protein and 24,5% fat). Macronutirent distributions were thus within prudent guidelines (CHO: 45-65%; protein: 10-35%; fat: 20-35%). The energy intake was below the estimated energy requirements (EER) range of 10143 kJ for sedentary men and 7947 kJ for sedentary women.

More than half (65,6%) of rural females and two-thirds (66,2%) of urban females were overweight or obese (body-mass-index (BMI) >25kg/m^2). Fewer men were overweight or obese (23,3% rural men and 16% urban men). A significantly larger percentage of urban than rural men (urban: 61,0%; rural: 43,6%) had a normal BMI (18,5 to 24,9 kg/m^2) (p=0.007). A third (33,1%) of rural men and 23% of urban men were underweight (BMI <18,5kg/m^2). Mean BMI for men was within the normal range at 20 kg/m^2. For women mean BMI fell in the overweight range at 28 kg/m^2.

Significantly more urban than rural men had a normal waist circumference (<94 cm) (p=0.002) and similarly, significantly more urban (32%) than rural women (24,4%) had a normal waist circumference (<80 cm) (p=0.03). Significantly more rural than urban men had a waist circumference >94 cm (p=0.01), placing them at risk for developing chronic diseases of lifestyle (CDLs). About one-fifth (17,8%) of rural and 19,4% of urban women were at risk (>80 cm). Significantly more rural women (57,9%) were at high risk of developing CDLs (>88cm) than urban women (48,6%) (p=0.02). Median waist circumference for rural women was 92 cm (high risk) and for urban women 87cm (at risk). The median waist circumference for rural men was 78,5 cm (normal) and for urban men 76 cm (normal).

Rural children were more often underweight (weight-for-age <-2 standard deviations (SD))(rural: 31,7%; urban: 17,3%) than urban children. In contrast to what was expected, urban children were more often stunted (height-for-age <-2SD) than rural children (rural: 38,4%; urban: 44,0%). Prevalence of wasting (weight-for-height <-2SD) was similar in rural and urban children (rural: 11,9%; urban: 10,1%).

A double burden of undernutrition in children and overweight in caretakers was confirmed in this sample, with 63,2% of stunted- and 66,7% of underweight rural children and 71,9% of stunted- and 66,7% of underweight children in urban areas living with an overweight/obese caregiver.

Although energy intake and macronutrient composition of the diets were within prudent guidelines, information related to the types of foods eaten as well as the foods most frequently consumed confirm that a nutrition transition was present in both urban and rural communities included in this study.

Although not initially included in the objectives of this sub-study, possible reasons for differences in anthropometric status of rural and urban adult participants include a higher prevalence of human immunodeficiency virus (HIV)-infection amongst urban participants (41% in urban adults and 17% in rural participants) as well as higher alcohol consumption in urban men and higher levels of physical activity in rural men. Associations between these variables need to be investigated further in this dataset.

Relevant and culturally acceptable nutrition interventions aimed at addressing both undernutrition in children and overweight and obesity in adults need to be implemented in an effort to address the high risk of CDLs as well as the poor quality of diet identified in this sample.

Opsomming

Groei-inkorting in kinderjare voorbestem hul vir vetsug, wat die risiko vir chroniese leefstylsiektes in volwassenheid verhoog (d.i. die dubbele las van siekte). Om die toepaslike voedingsintervensies te beplan moet die las van siekte, en die leefstylfaktore wat daartoe bydra, bepaal word. Die doel van hierdie substudie, wat deel gevorm het van die "Assuring Health for All in the Free State" (AHA FS) studie, was om 'n insig te kry in die dieetinname en antropometriese voedingstatus van voorskoolse kinders en volwassenes tussen die ourderdomme van 25 en 64 jaar in landelike en stedelike areas van die Vrystaat. Die landelike areas het bestaan uit Trompsburg, Philippolis en Springfontein. Die stedelike areas het bestaan uit gemeenskappe rondom die "Mangaung University Community Partnership Program" (MUCPP) kliniek.

Dieetinname was bepaal vir 60 landelike en 116 stedelike kinders, en 553 landelike en 419 stedelike volwassenes deur middel van 'n 24-uur herroep van gewoonlike inname en 'n kwalitatiewe voedselfrekwensie-vraelys. Inname van voedsels was met die voedselpiramide vergelyk en gekategoriseer as meer as, minder as, of binne aanbevelings. Inname van energie en makrovoedingstowwe was bepaal vanaf die aantal voedsels wat ingeneem is van elke voedselgroep, waarna dit met verstandige ("prudent") dieetriglyne vergelyk is. Antropometriese data was versamel deur middel van standaard meettegnieke en gekatagoriseer volgens die Wêreld Gesonheidsvereniging (WHO) se standaarde.

Tydens die tydperk van die studie was ongeveer 'n derde (31,24%) van landelike kinders en 40,7% van stedelike kinders op borsvoeding. 'n Betekenisvolle groter persentasie (90,9%) van landelike deelnemers (95% vertrouensinterval (VI) [20,0; 69,8]) was voorheen geborsvoed, in vergelyking met 37,5% van stedelike deelnemers.

Oor die algemeen het landelike volwassenes en kinders meer as die aanbevole daaglikse hoeveelhede van vet en olie; lekkergoed en suiker; en vleis en vleisproduke ingeneem, asook minder vrugte en groente as die stedelike deelnemers. Daarteenoor het stedelike volwassenes en kinders meer gereeld brood en graanprodukte bo die daaglikse aanbevole hoeveelhede ingeneem. Die inname van peulgroente en soja was laag in beide landelike en stedelike deelnemers.

Suiker was die voedselitem wat mees gereeld ingeneem was, veral onder landelike deelnemers. Gekookte pap was die mees gereelde stapelvoedsel wat ingeneem was, gevolg deur brood. Tee was die mees gereelde vloeistof wat ingeneem was. Suiker-versoete drankies (koeldranke) was ook gereeld ingeneem, veral deur kinders. Mans het koffie meer gereeld gedrink in vergelyking met vrouens en kinders. Sout, boeljonpoeiers, margarien, olie en vet was gereeld in voedselbereiding gebruik en margarien was gereeld op brood gesmeer. Vrugte en groente was gereeld ingeneem, maar vrugte meer as groente. Vrugte en groente-inname was egeter oor die algemeen minder as die aanbevole daaglikse hoeveelhede. Skyfies en slap aartappelskyfies was gereeld deur kinders ingeneem. Landelike en stedelike seuns, sowel as stedelike vroue, het gereeld lekkergoed ingeneem. Volroommelk was die mees gereelde suiwelproduk wat ingeneem is. Alkohol was oormatig ingeneem deur vyf persent van stedelike mans.

Gemiddelde energie-inname was 4254 kJ vir landelike kinders jonger as twee jaar (56,9% koolhidrate; 17,2% proteïen; 25,7% vet) en 3292kJ vir stedelike kinders jonger as twee jaar (64,2% koolhidrate; 19,5% proteïen; 20,1% vet). Die persentasie energie van koolhidrate en proteïen was binne verstandige dieetriglyne (CHO: 45-65%; proteïen: 5-20%), terwyl die persentasie energie van vet laer as aanbevelings (30-40%) was.

Gemiddelde energie-inname vir landelike kinders ouer as twee jaar was 5581kJ (57,5% koolhidrate; 16,9% proteïen; 28,7% vet) en 4220kJ vir stedelike kinders (65,5% koolhidrate; 17,9% proteïen; 20,3% vet). Die persentasie energie vanaf koolhidrate en proteïen was binne die verstandige dieetriglyne, (koolhidrate (CHO): 45-65%; proteïen: 10-30%; vet: 25-35%) soos gevind was by kinders jonger as twee jaar. Die vet inname was egter laer as aanbevelings onder stedelike deelnemers.

Die gemiddelde energie-inname vir alle mans was 8040 kJ (61% koolhidrate; 17,8% proteïen; en 24,3% vet). Vir vroue in die huidige studie was dit 7243 kJ (61,7% koolhidrate; 17,3% proteïen and 24,5% vet). Die verspreiding van makrovoedingstowwe was binne verstandige dieetriglyne (CHO: 45-65%; proteïen: 10-35%; vet: 20-35%). Die energie-inname was minder as die benaderde energiebehoeftes (EER) van 10143 kJ in onaktiewe mans en 7947 kJ in onaktiewe vrouens.

Meer as helfde (65,6%) van landelike vroue en twee derdes (66,2%) van stedelike vroue was oorgewig of vetsugtig (liggaamsmassaindeks (LMI) >25kg/m^2). Minder mans was oorgewig of obees (23,3% landelike mans en 16% stedelike mans). Daar was ‘n betekenisvolle groter persentasie van stedelike mans as landelike mans (stedelik: 61,0%; landelik: 43,6%) met ‘n normale LMI (18,5 tot 24,9 kg/m^2) (p=0.007). ‘n Derde (33,1%) van landelike mans en 23% van stedelike mans was ondermassa (LMI <18,5kg/m^2). Die gemiddelde LMI vir mans (20 kg/m^2) was binne normale reikwydtes. Vir vroue het die gemiddelde LMI (28 kg/m^2) in die oormassa reikwydte geval.

Betekenisvol meer stedelike as landelike mans het ‘n normale middelomtrek gehad (<94 cm) (p=0.002) en soortgelyk, het betekenisvol meer stedelike (32%) as landelike vroue (24,4%) ‘n normale middelomtrek gehad (<80 cm) (p=0.03). Betekenisvol meer landelike as stedelike mans het ‘n middelomtrek >94 cm gehad (p=0.01), wat hul geplaas het binne ‘n risiko grens vir die ontwikkeling van chroniese leefstylsiektes. Ongeveer ‘n vyfde (17,8%) van landelike en 19,4% stedelike vroue was binne die risiko grens (>80 cm). Betekenisvol meer landelike vroue (57,9%) was in die hoë risiko grens vir die ontwikkeling van chroniese leefstylsiektes (>88cm) as stedelike vroue (48,6%)(p=0.02). Mediaan middelomtrek vir landelike vroue was 92 cm (hoë risiko) en vir stedelike vroue 87cm (risiko). Die median middelomtrek vir landelike mans was 78,5 cm (normaal) en vir stedelike mans 76 cm (normaal).

Landelike kinders was meer gereeld ondermassa (massa-vir-ouderdom <-2 standaardafwykings)(landelik: 31,7%; stedelik: 17,3%) as stedelike kinders. In kontras tot verwagtinge het stedelike kinders meer gereeld groei-inkorting (lengte-vir-ouderdom <-2 standaardafwykings) as landelike kinders getoon (landelike: 38,4%; stedelike: 44,0%). Die voorkomsyfer van wegkwyning (massa-vir-lengte <-2 standaardafwykings) was soortgelyk in landelike en stedelike kinders (landelik: 11,9%; stedelik: 10,1%).

‘n Dubbele las van ondermassa in kinders en oormassa in versorgers was bevestig in hierdie studiegroep, met 63,2% van groei-ingekorte landelike kinders en 66,7% van ondermassa landelike kinders, sowel as 71,9% van groei-ingekorte stedelike kinders en 66,7% van ondermassa stedelike kinders wat woonagtig was saam met ‘n oormassa of vetsugtige versorger.

Alhoewel energie-inname en makrovoedingstof-samestellings van die diëte binne verstandige dieetriglyne geval het, het inligting verwant aan die tipes voedsels wat ingeneem was, asook die voedsels wat mees gereeld ingeneem was, die voedingsoorgang (“nutrition transition”) bevestig in beide stedelike en landelike gemeenskappe wat in hierdie studie ingesluit was.

Alhoewel moontlike oorsake nie oorspronklik ingesluit was in die doelwitte van hierdie substudie nie, kan verskille in antropometriese status van landelike en stedelike volwassenes verwant wees aan die hoë voorkomsyfer van HIV-infeksie onder stedelike deelnemers (41% in stedelike volwassenes en 17% in landelike volwassenes), asook ‘n hoër alkoholinname in stedelike mans en hoër vlakke van fisiese aktiwiteit in landelike mans. Verwantskappe tussen hierdie veranderlikes moet verder in hierdie datastel ondersoek word.

Toepaslike en kultureel aanvaarbare voedingsintervensies wat gemik is daarop om beide ondermassa in kinders en oormassa en obesiteit in volwassenes aan te spreek, moet geïmplementeer word in ‘n poging om die hoë risiko van chroniese leefstylsiekstes, sowel as die swak kwaliteit van die dieet wat in hierdie studiegroep geïdentifiseer is, aan te spreek.

References

American Dietetics Association (ADA). 2000. Manual of clinical dietetics. 6th edition. London: Yale University Press.

Anderson JJB. 2004. Minerals, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia:WB Saunders Company:121-163.

Augustin LS, Dal Maso L, La Vecchia C, Parpinel M, Negri E, Vaccarella S, Kendall CW, Jenkins DJ and Francesch S. 2001. Dietary glycemic index and glycemic load, and breast cancer risk: a case-control study. Annals of Oncology, 12(11):1533-1538.

Barker DJP. 1990. Fetal and infant origins of adult diseases. British Medical Journal, 301(6761):1111.

Bihl GR. 2003. Intra-uterine growth and disease in later life – Barker and beyond. South African Medical Journal, 93(10):757-760.

Bourne LT and Seager JR. 2001. Water – the neglected nutrient. South African Journal of Clinical Nutrition, 14(3):S64-S70.

Bourne LT, Lambert EV and Steyn K. 2002. Where does the black population of South Africa stand on the nutrition transition? Public Health Nutrition, 5(1A):157-162.

Bourne LT, Langenhoven ML, Steyn K, Jooste PL, Laubscher JA, Van der Vyver E. 1993. Nutrient intake in the urban African population of the Cape Peninsula, South Africa. The Brisk study. Central African Journal of Medicine, 39(12):238-247.

Bourne LT. 2007. South African paediatric food-based dietary guidelines. Maternal and Child Nutrition, 3:227-229.

Bradshaw D, Groenewald P, Laubscher R, Nannan N, Nojilana B, Norman R, Pieterse D and Schneider M. 2003. Initial Burden of Disease Estimates for South Africa, 2000. Cape Town: South African Medical Research Council. [Online]. Available from: http://www.mrc.ac.za/bod/initialbodestimates.pdf [Accessed June 19th, 2012].

Bradshaw D, Norman R, Pieterse D, Levitt NS and the South African Comparative Risk Assessment Collaborating Group. 2007. Estimating the burden of disease attributable to diabetes in South Africa in 2000. South African Medical Journal, 97(7):700-706.

Brink AJ. 1988. People for research and development 1988. Proceedings of a conference held at the CSIR Conference Centre, Pretoria, 23-24 August 1988.

Cameron N. 2003. Physical growth in a transitional economy: the aftermath of South African apartheid. Economics and Human Biology, 1: 29–42.

Carlson TH. 2004. Laboratory data in nutrition assessment, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia:WB Saunders Company:436-454.

Center for Nutrition Policy and Promotion (CNPP). 1999. Food Guide pyramid for young children 2 to 6 years old. Washington, DC: US Department of Agriculture. [Online]. Available from: http://www.cnpp.usda.gov/fgp.htm [Accessed June 19th, 2012].

Centers for Disease Control and Prevention (CDCP). 2009. Healthy weight – it's not a diet, it's a lifestyle! [Online]. Available from: http://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html [Accessed June 19th, 2012].

Centers for Disease Control and Prevention (CDCP). 2010. Growth Charts. [Online]. Available from: http://www.cdc.gov/growthcharts/who_charts.htm [Accessed March 22nd, 2012].

Charlton KE and Jooste PL. 2001. Eat salt sparingly – sprinkle, don't shake! South African Journal of Clinical Nutrition, 14(3):S55-S64.

Churma SA and Horrell CJ. 2005. Esophageal and gastric cancers, In Nutritional issues in cancer care. Ed. by VJ Kogut and SL Luthringer. Pittsburgh, Pennsylvania:Oncology Nursing Society:45-63.

Couch SC and Krummel DA. 2008. Medical nutrition therapy in hypertension, In Krause's food and nutrition therapy. Ed. by Mahan KL and Escott-Stump S. 12th ed. Philadelphia:WB Saunders Company:865-883.

Cummings, MR. 2006. Human heredity, 7th edition. Belmont, California:Thomson Learning Inc.

Darnton-Hill I and Coyne ET. 1998. Feast and famine: socioeconomic disparities in global nutrition and health. Public Health Nutrition, 1(1):23-31.

De Onis M, Garza C, Onyango AW and Borghi E. 2007. Comparison of the WHO child growth standards and the CDC 2000 growth charts. The Journal of Nutrition, 137:141-148.

De Villiers A and Senekal M. 2002. Determinants of growth failure in 12-24 month-old children in a high-density urban slum community in East London, South Africa. European Journal of Clinical Nutrition, 56:1231-1241.

Department of Health, South African Medical Research Council and Measure DHS+. 2002. South African Demographic and Health Survey - 1998. [Online]. Available from: http://www.hst.org.za/sites/default/files/SADHS_1998_Full.pdf [Accessed June 19th, 2012].

Department of Health. 2001. National food consumption survey in children aged 1-9 years: South Africa 1999. Part I: Methodology, sociodemographic and anthropometric data. Medical Update Number 37, Directorate: Nutrition, Department of Health.

Department of Health. 2004. South African guidelines for healthy eating. Directorate Nutrition, Pretoria.

Department of Health. 2004a. South African Demographic and Health Survey 2003 – Preliminary Report. Pretoria:South Africa. [Online]. Available from: http://www.hst.org.za/sites/default/files/SADHS_2003.pdf [Accessed June 19th, 2012].

Dewey KG. 2006. What is the optimal age for introduction of complementary foods? In Protein and Energy Requirements in Infancy and Childhood - Nestlé Nutrition Workshop Series Pediatric Program, Ed. By Ziegler RJ. Nestec Ltd., 58:161-175.

Earl R. 2004. Guidelines for dietary planning, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia:WB Saunders Company, Table 15-2:368, Figure 15-4:378 .

Eldridge B. 2004. Medical nutrition therapy for cancer prevention, treatment and recovery, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia: WB Saunders Company:1000-1003.

Faber M and Benadé AJS. 2001. Perceptions of infant cereals and dietary intakes of children aged 4-24 months in a rural South African community. International Journal of Food Sciences and Nutrition, 52:359-365.

Faber M, Jogessar VB and Benadé AJS. 2001. Nutritional status and dietary intakes of children aged 2-5 years and their caregivers in a rural South African community. International Journal of Food Sciences and Nutrition, 52:401-411.

Fernald LC and Neufeld LM. 2007. Overweight with concurrent stunting in very young children from rural Mexico: prevalence and associated factors. European Journal of Clinical Nutrition, 61(5):623-632.

Fletcher DM and Hayward MC. 2005. Breast cancer, In Nutritional issues in cancer care. Ed. by VJ Kogut and SL Luthringer. Pittsburgh, Pennsylvania:Oncology Nursing Society:15-28.

Food and Nutrition Board, Institute of Medicine and National Academies. 2002. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids.. Washington DC:The National Academies Press. [Online]. Available from: http://fnic.nal.usda.gov/dietary-guidance/dri-reports/energy-carbohydrate-fiber-fat-fatty-acids-cholesterol-protein-and-amino#overlay-context=dietary-guidance/dietary-reference-intakes/dri-reports [Accessed June 19th, 2012].

Forsen T, Eriksson J, Tuomilehto J, Reunanen A, Osmond C, Barker D. 2000. The fetal and childhood growth of persons who develop type 2 diabetes. Annals of Internal Medicine, 133:176-182.

Franz MJ. 2004. Medical nutrition therapy for diabetes mellitus and hypoglycemia of nondiabetic origin, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia: WB Saunders Company: 794, 797.

Franz MJ. 2008. Medical nutrition therapy for diabetes mellitus and hypoglycemia of nondiabetic origin, In Krause's food and nutrition therapy. Ed. by Mahan KL and Escott-Stump S. 12th ed. Philadelphia:WB Saunders Company:764-809.

Gee M, Mahan LK and Escott-Stump S. 2008. Weight management, In Krause's food and nutrition therapy. Ed. by Mahan KL and Escott-Stump S. 12th ed. Philadelphia:WB Saunders Company:532-562.

Gibson RS. 2005. Principles of nutritional assessment. 2nd edition. New York: Oxford University Press.

Goedecke JH, Jennings CL and Lambert EV. 2005. Obesity in South Africa. Chapter 7 – Chronic Diseases of Lifestyle in South Africa since 1995-2005. [Online]. Available from: http://www.mrc.ac.za/chronic/cdlchapter7.pdf [Accessed June 19th, 2012].

Gopane RE, Pisa PT, Vorster HH, Kruger A and Margetts BM. 2010. Relationships of alcohol intake with biological health outcomes in an African population in transition: the Transition and Health during Urbanisation in South Africa (THUSA) study. South African Medical Journal, 23(32)(Supplement1):S16-S21.

Grant B. 2008. Medical nutrition therapy for cancer, in Krause's food and nutrition therapy. Ed. by Mahan KL and Escott-Stump S. 12th ed. Philadelphia:WB Saunders Company:959-990.

Hales CN, Barker DJP. 1992. Type 2 (non-insulin dependent) diabetes mellitus: the thrifty phenotype hypothesis. Diabetologia;35:595-601.

Hamilton K and Roche SM. 2005. Gynecologic cancers, In Nutritional issues in cancer care. Ed. by VJ Kogut and SL Luthringer. Pittsburgh, Pennsylvania:Oncology Nursing Society:79-102.

Harris NS, Crawford PB, Yangzom Y, Pinzo L, Gylatsen P and Hudes M. 2001. Nutritional and health status of Tibetan children living at high altitudes. New England Journal of Medicine; 344(5): 341–347.

Hoffman DJ, Sawaya AL, Coward WA, Wright A, Martins PA, De Nascimento C, Tucker KL and Roberts SB. 2000. Energy expenditure of stunted and non-stunted boys and girls living in the shantytowns of São Paulo, Brazil. American Journal of Clinical Nutrition, 72:1025-1031.

Hofman KJ and Tollman SM. 2010. Health Policies and Practice: Setting priorities for health in 21st-century South Africa. South African Medical Journal, 100(12):798-800.

James HC and Kulakowski KP. 2005. Hepatobiliary cancer, In Nutritional issues in cancer care. Ed. by VJ Kogut and SL Luthringer. Pittsburgh, Pennsylvania:Oncology Nursing Society:117-125.

Jinabhai CC, Taylor M, Sullivan KR. 2003. Implications of the prevalence of stunting, overweight and obesity amongst South African primary school children: a possible nutritional transition? European Journal of Clinical Nutrition [Online], 57:358–365 Available from: http://www.nature.com/ejcn/journal/v57/n2/full/1601534a.html [Accessed June 19th, 2012].

Joubert J, Norman R, Bradshaw D, Goedecke JH, Steyn NP, Puoane T and the South African Comparative Risk Assessment Collaborating Group. 2007, Estimating the burden of disease attributable to excess body weight in South Africa in 2000. South African Medical Journal, 97(8):683-690.

Kanjilal B, Mazumdar PG, Mukherjee M and Rahman MH. 2010. Nutritional status of children in India: household socio-economic condition as the contextual determinant. International Journal Equity in Health, [Online], 9:19, Available from: http://www.equityhealthj.com/content/9/1/19 [Accessed June 19th, 2012].

Koh ET and Owen WL. 2000. Introduction to nutrition and health research. Massachusetts:Kluwer Academic Publishers.

Kruger HS, Puoane T, Senekal M and Van der Merwe MT. 2005. Obesity in South Africa: challenges for government and health professionals. Public Health Nutrition, 8(5):491-500.

Kruger HS, Venter CS, Vorster HH, Margetts BM. 2002. Physical inactivity is the major determinant of obesity in black women in the North West Province, South Africa: the THUSA study. Transition and Health During Urbanisation of South Africa. Nutrition;18:422-427.

Krummel DA. 2004a. Medical nutrition therapy in cardiovascular disease, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia: WB Saunders Company:861-899.

Krummel DA. 2004b. Medical nutrition therapy in hypertension, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia: WB Saunders Company:910-911.

Krummel DA. 2008. Medical nutrition therapy for cardiovascular disease, In Krause's food and nutrition therapy. Ed. by Mahan KL and Escott-Stump S. 12th ed. Philadelphia:WB Saunders Company:833-864.

Labadarios D and Nel JH. 2000. Chapter 4: Anthropometric status. The National Food Consumption Survey, [Online], 162-187. Available from: http://www.sahealthinfo.org/nutrition/food4anthrostatus.pdf [Accessed June 19th, 2012].

Labadarios D, Mauner E, Swart R, Bagriansky J, Steyn NP, MacIntyre U, Gericke G, Huskisson J, Dannhauser A, Vorster HH and Nesamvuni AE. 2000. Chapter 9: general discussion and recommendations. The National Food Consumption Survey, [Online], 856-946. Available from: http://www.sahealthinfo.org/nutrition/food9discussion.pdf [Accessed June 19th, 2012].

Labadarios D, Steyn NP, Maunder E, MacIntyre U, Gericke G, Swart R, Huskisson J, Dannhauser A, Vorster HH, Nesamvuni AE and Nel JH. 2005. The National Food Consumption Survey (NFCS): South Africa, 1999. Public Health Nutrition, 8(5):533-543.

Langenhoven M and Wolmarans P. 1997. Assessment of dietary intake, In Epidemiology: a manual for South Africa. Ed. by Katzenellenbogen JM, Joubert G, Abdool K. Cape Town:Oxford University Press:250.

Laquatra I. 2004. Nutrition for weight management, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia:WB Saunders Company, Table 24-1:565.

Lee RD and Nieman DC. 2003. Nutritional Assessment. 3rd edition. New York: McGraw Hill.

Lee RD and Nieman DC. 2010. Nutritional Assessment. 5th edition. New York: McGraw-Hill.

LeLeiko N, Rollinson MS and Sockolow RE. 1995. Nutrition in infancy and childhood, In Total Nutrition: The only guide you'll ever need, Ed. by Herbert V and Subak-Sharpe GJ. New York:St Martin's Press, Tables 13-4 and 13-5:218-219.

Levitt NS, Lambert EV and Norris SA. 2005. Early life origins of adult chronic diseases: a South African perspective. Chapter 6 – Chronic Diseases of Lifestyle in South Africa since 1995-2005. [Online], 58-64. Available from: http://www.mrc.ac.za/chronic/cdlchapter6.pdf [Accessed June 19th, 2012].

Li L, Manor O and Power C. 2004. Early environment and child-to adult growth trajectories in the 1958 British birth cohort. American Journal of Clinical Nutrition, 80:185-1992.

Love P and Sayed N. 2001. Eat plenty of vegetables and fruits every day. South African Journal of Clinical Nutrition, 14(3):S24-S31.

Lucas BL. 2004. Nutrition in childhood, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia:WB Saunders Company, Table 10-1:267.

Lucas BL. 2004a. Nutrition in childhood, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia:WB Saunders Company:259-283.

MacIntyre U and Labadarios D. 2000. Chapter 6: dietary intake: quantitative food frequency method. The National Food Consumption Survey, [Online], 312-367. Available from: http://www.sahealthinfo.org/nutrition/

food6dietaryintake.pdf [Accessed June 19th, 2012].

MacIntyre UE, Kruger HS, Venter CS and Vorster HH. 2002. Dietary intakes of an African population in different stages of transition in the North West Province, South Africa: the THUSA study. Nutrition Research, 22(3):239-256.

MacKeown JM, Cleaton-Jones PE, Edwards AW and Turgeon-O'Brien H. 1998. Energy, macro-and micronutrient intake of 5-year-old urban black South African children in 1984 and 1995. Paediatric and Perinatal Epidemiology, 12:297-312.

Mahan KL and Escott-Stump S. 2004a. Nutritive values for alcoholic beverages and mixes, In Krause's food, nutrition and diet therapy. 11th ed. Philadelphia: WB Saunders Company, Appendix 44:1241.

Mahan KL and Escott-Stump S. 2004b. Indirect methods for measuring height, In Krause's food, nutrition and diet therapy. 11th ed. Philadelphia: WB Saunders Company, Appendix 15:1184.

Mahan KL and Escott-Stump S. 2004c. Milliequivalents and milligrams of electrolytes, In Krause's food, nutrition and diet therapy. 11th ed. Philadelphia: WB Saunders Company, Appendix 4:1176.

Mahan KL and Escott-Stump S. 2004d. Birth to 36 months: boys head circumference-for-age and weight-for-length percentiles, In Krause's food, nutrition and diet therapy. 11th ed. Philadelphia: WB Saunders Company, Appendix 7:1178.

Mahan KL and Escott-Stump S. 2004e. Birth to 36 motnhs: girls head circumference-for-age and weight-for-length percentiles, In Krause's food, nutrition and diet therapy. 11th ed. Philadelphia: WB Saunders Company, Appendix 10:1181.

Mahan KL and Escott-Stump S. 2008a. Nutritional facts on alcoholic beverages, In Krause's food and nutrition therapy. 12th ed. Philadelphia: WB Saunders Company, Appendix 38:1276.

Mahan KL and Escott-Stump S. 2008b. Indirect methods for measuring height, In Krause's food and nutrition therapy. 12th ed. Philadelphia: WB Saunders Company, Appendix 20:1213.

Mamabolo RL, Alberts M, Steyn NP, Delemarre-van de Waal HA and Levitt NS. 2005. Prevalence and determinants of stunting and overweight in 3-year old black South African children residing in the Central Region of Limpopo Province, South Africa. Public Health Nutrition, [Online], 8(5):501-508. Available from: http://journals.cambridge.org/action/displayAbstract?fromPage=online&aid=585132 [Accessed June 19th, 2012].

Masino K. 2005. Colorectal cancer, In Nutritional issues in cancer care. Ed. by VJ Kogut and SL Luthringer. Pittsburgh, Pennsylvania:Oncology Nursing Society:29-43.

Mayo Foundation for Medical Education and Research. 2011. Cholesterol levels: What numbers should you aim for? [Online]. Available from: http://www.mayoclinic.com/health/cholesterol-levels/CL00001/METHOD=print [Accessed on June 19th, 2012].

Mendez MA, Monteiro CA and Popkin BM. 2005. Overweight exceeds underweight among women in most developing countries. American Journal of Clinical Nutrition, 81:714-721.

Mollentze WF, Moore AJ, Steyn AF, Joubert G, Steyn K and Oosthuizen GM. 1995. Coronary heart disease risk factors in a rural and urban Orange Free State population. South African Medical Journal, 85:90-96.

Monsen ER. 1992. Research. 1st edition. Chicago: The American Dietetic Association.

Mukuddem-Petersen J and Kruger HS. 2004. Association between stunting and overweight among 10-15 year old children in the North West Province of South Africa: the THUSA BANA Study. International Journal of Obesity, 28:842-851.

Murray CJ and Lopez AD. 1997a. Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. Lancet, 349(9063):1436-1442.

Murray CJ and Lopez AD. 1997b. Alternative projections of mortality and disability by cause 1990-2020: Global Burden of Disease Study. Lancet, 349(9064):1498-1504.

Murray CJ and Lopez AD. 1997c. Mortality by cause for eight regions of the world: Global Burden of Disease Study. Lancet, 349(9061):1269-1276.

Nannan N, Norman R, Hendricks M, Dhansay MA, Bradshaw D and the South African Comparative Risk Assessment Collaborating Group. 2007. Estimating the burden of disease attributable to childhood and maternal undernutrition in South Africa in 2000. South African Medical Journal, 97(8):733-739.

Nel JH and Steyn NP. 2002. Report on South African food consumption studies undertaken amongst different population groups (1983 – 2000): Average intakes of foods most commonly consumed. Department of Health. Pretoria:South Africa.

Norman R, Bradshaw D, Schneider M, Joubert J, Groenewald P, Lewin S, Steyn K, Vos T, Laubscher R, Nannan N, Nojilana B, Pieterse D and the South African Comparative Risk Assessment Collaborating Group. 2007c. A comparative risk assessment for South Africa in 2000: Towards promoting health and preventing disease, South African Medical Journal, 97(7):637-641.

Norman R, Bradshaw D, Schneider M, Pieterse D and Groenewald P. 2006. Revised burden of disease estimates for the Comparative Risk Factor Assessment, South Africa 2000.

Norman R, Bradshaw D, Schneider M, Pieterse D and Groenewald P. 2010. How does South Africa compare with other countries? [Online]. Available from: http://www.mrc.ac.za/bod/faqcompare.htm [Accessed on June 19th, 2012].

Norman R, Bradshaw D, Schneider M, Pieterse D and Groenewald P. 2010a. What are the top causes of death due to cardiovascular disease in SA? [Online]. Available from: http://www.mrc.ac.za/bod/faqcardio.htm [Accessed on June 19th, 2012].

Norman R, Bradshaw D, Schneider M, Pieterse D and Groenewald P. 2010b. What are the leading causes of death in children under 5 years of age? [Online]. Available from: http://www.mrc.ac.za/bod/faqchildren.htm [Accessed on June 19th, 2012].

Norman R, Bradshaw D, Steyn K, Gaziano T and the South African Comparative Risk Assessment Collaborating Group. 2007b. Estimating the burden of disease attributable to high cholesterol in South Africa in 2000, South African Medical Journal, 97(7):708-715.

Norman R, Gaziano T, Laubscher R, Steyn K, Bradshaw D and the South African Comparative Risk Assessment Collaborating Group. 2007a. Estimating the burden of disease attributable to high blood pressure in South Africa in 2000. South African Medical Journal, 97(8):692-698.

Nthangeni G, Steyn NP, Alberts M, Steyn K, Levitt NS, Laubscher R, Bourne L, Dick J, Temple N. 2002. Dietary intake and barriers to dietary compliance in black type 2 diabetic patients attending primary health-care services. Public Health Nutrition, 5: 329–338.

O'Keefe SJD, Thusi D and Epstein S. 1983. The fat and the thin – a survey of nutritional status and disease patterns among urbanized Black South Africans. South African Medical Journal, 63:679-683.

Oosthuizen W, Van der Merwe AM, Kotzé JP, Vorster HH and Steyn HS. 1998. Dietary composition and body mass index at different levels of added sugar consumption – the VIGHOR study. South African Medical Journal, 88 (9):1212-1217.

Parsons TJ, Power C, Logan S and Summerbell CD. 1999. Childhood predictors of adult obesity:a systematic review. International Journal of Obesity, 23, Supplement 8:S1-S107.

Pisa PT, Kruger A, Vorster HH, Margetts BM and Loots DT. 2010a. Alcohol consumption and cardiovascular disease risk in an African population in transition: the Prospective Urban and Rural Epidemiology (PURE) study. South African Medical Journal, 23(3)(Supplement 1):S29-S37.

Pisa PT, Loots DT and Nienaber C. 2010. Alcohol metabolism and health hazards associated with alcohol abuse in a South African context: a review. South African Medical Journal, 23(3)(Supplement 1):S4-S10.

Popkin BM, Richards MK and Montiero CA. 1996. Stunting is associated with overweight in children of four

nations that are undergoing the nutrition transition. The Journal of Nutrition, 126:3009-3016.

Potischman N, Coates RJ, Swanson CA, Carroll RJ, Dalings JR, Brogan DR, Gammon MD, Midthune D, Curtin J and Brinton LA. 2002 .Increased risk of early-stage breast cancer related to consumption of sweet foods among women less than age 45 in the United States. Cancer Causes and Control, 13:937.

Puoane T, Steyn K, Bradshaw D, Laubscher R, Fourie J, Lambert V and Mbananga N. 2002. Obesity in South Africa: the South African demographic and health survey. Obesity Research, 10: 1038–1048

Rehman AM, Gladstone BP, Verghese VP, Muliyil J, Jaffar S and Kang G. 2009. Chronic growth faltering amongst a birth cohort of Indian children begins prior to weaning and is highly prevalent at three years of age. Nutrition Journal, [Online], 8:44. Available from: http://www.nutritionj.com/content/8/1/44 [Accessed on June 19th, 2012].

Richardson, BD, 1978. Growth patterns of South African children. South African Medical Journal, 74:246–249.

Rodgers A, Lawes CMM, Gaziano T, Vos T. 2006. The growing burden of risk from high blood pressure, cholesterol and bodyweight. In Disease Control Priorities in Developing Countries. Ed by DT Jamison, JG Breman, AR Measham, G Alleyne, M Claeson, DB Evans, P Jha, A Mills and P Musgrove. 2nd ed. Washington (DC): World Bank. [Online] Available from: www.ncbi.nlm.nih.gov/books/NBK11738/#A6551 [Accessed September 9th, 2012].

Rosman KO. 1986. The epidemiology of stroke in an urban black population. Stroke, 17:667-669.

Rotchford AP and Rotchford KM. 2002. Diabetes in rural South Africa – an assessment of care and complications. South African Medical Journal, 92:536–541.

SAS. 2001. SAS Institute Inc., Cary, North Carolina.

Save the Children Fund (SCF). 2012. A life free from hunger. London:Save the Children UK.

Sawaya AL and Roberts S. 2003. Stunting and future risk of obesity: principal physiological mechanisms Cadernos de Saúde Pública [Online], 19(S1). Available from: http://www.scielosp.org/scielo.php?script=sciarttext&pid=S0102-311X2003000700003&lng=en&nrm=iso [Accessed July 26th, 2009].

Sawaya AL, Martins P, Hoffman D and Roberts SB. 2003. The link between childhood undernutrition and risk of chronic disease in adulthood: a case study of Brazil. Nutrition Reviews, 61(5):168-173.

Schneider M, Norman R, Parry C, Bradshaw D, Plüddemann A and the South African Comparative Risk Assessment Collaborating Group. 2007a. Estimating the burden of disease attributable to alcohol use in South Africa in 2000. South African Medical Journal, 97(8):664-672.

Schneider M, Norman R, Steyn N, Bradshaw D and the South African Comparative Risk Assessment Collaborating Group. 2007. Estimating the burden of disease attributable to low fruit and vegetable intake in South Africa in 2000, South African Medical Journal, 97(8):717.

Scholtz SC, Vorster HH (jun), Matshego L and Vorster HH. 2001. Foods from animals can be eaten every day – not a conundrum! South African Journal of Clinical Nutrition, 14(3):S39-S47.

Schrimpton R and Kachondham Y. 2003. Analysing the causes of child stunting in Democratic People's Republic of Korea. [Online], 1-35. Available from: http://www.unicef.org/dprk/further_analysis.pdf [Accessed June 19th, 2012].

Schutte AE, Van Rooyen JM, Huisman HW, Kruger HS and De Ridder JH. 2003. Factor analysis of possible risks for hypertension in a black South African population. Journal of Human Hypertension, 17:339-348.

Seedat YK. 2007. Impact of poverty on hypertension and cardiovascular disease in sub-Saharan Africa. Cardiovascular Journal of Africa, 18(5):316-320.

Seftel HC, Raal FJ, Joffe BI. 1995. Dyslipidaemia in South Africa. In Chronic Diseases of Lifestyle in South Africa: Medical Research Council (MRC) Technical Report. Ed. by J Fourie and K Steyn K. Tygerberg: MRC, 61–71.

Seftel HC. 1977. Diseases in urban and rural black populations. South African Medical Journal, 51:121-123.

Senekal M, Steyn NP and Nel JK. 2003. Factors associated with overweight/obesity in economically active South African populations. Ethnicity and Disease, 13:109-116.

Shaw V and Lawson M. 2001. Clinical paediatric dietetics. 2nd ed. Oxford:Blackwell Science Ltd.

Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA). 2009. Guidelines for diagnosis and management of type 2 diabetes mellitus for primary health care.

South African Society for Obesity and Metabolism (SASOM). 2006a. SA Obesity Statistics. [Online]. Available from: http://www.sasom.co.za/index.php?p=sa_obesity_statistics.php [Accessed July 26th, 2009].

South African Society for Obesity and Metabolism (SASOM). 2006b. An estimated 400 million adults around the world are clinically obese; 2 billion are overweight and the numbers are rising each year. [Online]. Available from: http://www.sasom.co.za/index.php [Accessed July 26th, 2009].

South African Society for Obesity and Metabolism (SASOM). 2006c. Obesity in African Women. [Online]. Available from: http://www.sasom.co.za/index.php?p=obesity_in_african_women.php [Accessed July 26th, 2009].

Statistics South Africa. 1999. Population Census, 1996: Primary Tables Report 03-01-17 (1996). Pretoria: Statistics South Africa.

Statistics South Africa. 2003. Census 2001. [Online]. Available from: www.hst.org.za/publications/census-2001 [Accessed March 14th, 2012].

Steyn K, Bourne LT, Jooste PL, Fourie J, Rossouw K, and Lombard C. 1998. Anthropometric profile of a black population of the Cape Peninsula in South Africa. East African Medical Journal, 75:35-40.

Steyn K, Bradshaw D, Norman R and Laubsher R. 2008. Determinants and treatment of hypertension in South Africans: the first demographic and health survey. South African Medical Journal, 98(5):376-380.

Steyn K, Jooste PL, Bourne L, Fourie J, Badenhorst CJ, Bourne DE, Langenhoven ML, Lombard CJ, Truter H, Katzenellenbogen J, Marais M and Oelofse A. 1991. Risk factors for coronary heart disease in the Black population of the Cape Peninsula. The BRISK Study. South African Medical Journal, 79:480-485.

Steyn K. 2005b. Overview and conclusions: a perspective on dealing with chronic diseases of lifestyle in South Africa. Chapter 18 – Chronic Diseases of Lifestyle in South Africa since 1995-2005. [Online], 249-267. Available from: http://www.mrc.ac.za/chronic/cdlchapter18.pdf [Accessed June 19th, 2012].

Steyn NP, Bradshaw D, Norman R, Joubert J, Schneider M, Steyn K, and Fourie J. 2006. The Medical Research Council Technical Report on dietary changes and the health transition in South Africa: implications for health policy. [Online]. Available from: http://www.sahealthinfo.org/lifestyle/dietaccess.pdf [Accessed June 19th, 2012].

Steyn NP, Burger S, Monyeki KD, Alberts M and Nthangeni G. 2001. Variation in dietary intake of the adult population of Dikgale. South African Journal of Medical Nutrition, 14(4):140-145.

Steyn NP, Labadarios D, Maunder E, Nel J, Lombard C and Directors of the National Food Consumption Survey. 2005. Secondary anthropometric data analysis of the national food consumption survey in South Africa: the double burden. Nutrition, 21(1):4-13.

Steyn NP, Nel JH and Casey A. 2003. Secondary data analyses of dietary surveys undertaken in South Africa to determine usual food consumption of the population. Public Health Nutrition: 6(7), 631–644.

Steyn NP, Nel JH, Dhansay MA, Tichelaar HY, Kriek JA, Prinsloo JF. Differences in nutritional status between caretakers of underweight and normal weight Pedi pre-school children. South African Food Science and Nutrition, 7:53-59.

Steyn NP, Senekal M, Brits S, Alberts M, Mashego T and Nel JH. 2000. Weight and health status of black female students. South African Medical Journal, 90(2):146-152.

Steyn NP. 2005a. Chapter 4. Nutrition and the chronic diseases of lifestyle in South Africa since 1995-2005. [Online], 33-47. Available from: http://www.mrc.ac.za/chronic/cdlchapter4.pdf [Accessed June 19th, 2012].

Subramanyam MA, Kawachi I, Berkman LF, and Subramanian SV. 2010. Socioeconomic inequalities in childhood undernutrition in India: analyzing trends between 1992 and 2005. PLoS One, 5(6):1-9.

The World Bank and United Nations Children's Fund (UNICEF). 2002. Background Papers: World Bank/UNICEF Nutrition Assessment.

Torûn B. 2006. Protein-energy malnutrition, In Modern nutrition in health and disease, Ed. by ME Shils, M Shike, AC Ross, B Caballero and RJ Cousins. 10th edition. Baltimore: Lippincott Williams & WIlkins:890.

Touger-Decker R. 2004. Nutrition for oral and dental health, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia:WB Saunders Company:667-683.

Trahms CM. 2004. Nutrition during infancy, In Krause's food, nutrition and diet therapy. Ed. by Mahan KL and Escott-Stump S. 11th ed. Philadelphia:WB Saunders Company:214-233.

United Nations Children's Fund (UNICEF). 2008. The state of the world's children 2009: Maternal and newborn health. New York: United Nations Children's Fund.

United States Department of Agriculture (USDA) and United States Department of Health and Human Services (USDHHS). 2010. Dietary Guidelines for Americans, 2010. 7th Edition, Washington, DC:U.S. Government Printing Office.

United States Department of Agriculture (USDA). 1992. The Food Guide Pyramid. Home and Garden Bulletin, no. 252. Washington: US Government Printing Office. [Online]. Available from: http://www.mypyramid.gov/ [Accessed June 19th, 2012].

Van den Berg VL. 2011. Current opinion: Is added dietary sugar detrimental to health? South African Family Practice, 53(3):257-261.

Van der Merwe MT and Pepper MS. 2006. National Prevalence of Obesity: Obesity in South Africa. The International Association for the Study of Obesity: Obesity reviews:1-8.

Van der Merwe MT, Panz VR, Crowther NJ, Schlaphoff GP, Gray IP, Froguel P, Joffe BI and Lönnroth PN. 1999. Free fatty acids and insuling levels-relationship to leptin levels and body composition in various patient groups from South Africa. International Journal of Obesity, 23:909-917.

Van der Merwe MT. 2001. Adipose tissue lipolysis, metabolic indices and insulin resistance in South African ethnic groups. PhD Thesis. Faculty of Health Sciences, University of the Witwatersrand: Johannesburg.

Van Heerden IV and Parry CDH. 2001. If you drink alcohol, drink sensibly. South African Journal of Clinical Nutrition, 14(3):S71-S77.

Van Zyl S, Van der Merwe LJ, Walsh CM, Groenewald AJ, and Van Rooyen FC. 2012. Risk factor profiles for chronic diseases of lifestyle and metabolic syndrome in an urban and rural setting in South Africa. African Journal of Primary Health Care and Family Medicine, 4(1):Art.#346.

Vanhala M, Vanhala P, Kumpusalo E, Halonen P and Takala J. 1998. Relation between obesity from childhood to adulthood and the metabolic syndrome: population based study. British Medical Journal, 317(7154):319-320.

Vanhala MJ, Vanhala PT. Keinänen-Kiukaanniemie SM, Kumpusalo EA and Takala JK. 1999. Relative weight gain and obesity as a child predict metabolic syndrome as an adult. International Journal of Obesity and Related Metabolic Disorders, 23(6):656-659.

Verhoef P, Pasman WJ, Van Vliet T, Urgert R and Katan MB. 2002. Contribution of caffeine to the homocysteine-raising effect of coffee: a randomized controlled trial in humans. The American Journal of Clinical Nutrition, 76:1244-1248.

Victora CG, Adair L, Fall C, Hallal PC, Martorell R, Richter L, and Sachdev HS. 2008. Maternal and child

undernutrition: consequences for adult health and human capital. Lancet, 371:340-357.

Vorster HH and Kruger A. 2007. Poverty, malnutrition, underdevelopment and cardiovascular disease: a South African perspective. Cardiovascular Journal of Africa, 18(5):321-324.

Vorster HH and Nell TA. 2001. Make starchy foods the basis of most meals. South African Journal of Clinical Nutrition, 14(3):S17-S24.

Vorster HH, Jerling JC, Oosthuizen W, Becker P, Wolmarans P. 1995. Nutrient Intakes of South Africans. An analysis of the literature (SANNSS Group Report). Isando: Roche.

Vorster HH, Venter CS, Wissing MP and Margetts BM. 2005. The nutrition and health transition in the North West Province of South Africa: a review of the THUSA (Transition and Health during Urbanisation of South Africans) study. Public Health Nutrition [Online], 8:480-490. Available from: http://journals.cambridge.org/action/displayFulltext?type=6&fid=631528&jid=&volumeId=&issueId=05&aid=585100&bodyId=&membershipNumber=&societyETOCSession=&fulltextType=RA&fileId=S1368980005000674 [Accessed June 19th, 2012].

Vorster HH. 2002. The emergence of cardiovascular disease during urbanization of Africans. Public Health Nutrition, 5(1A):239-243.

Walker ARP and Walker BF. 1985. Coronary disease in blacks in underdeveloped populations (Letter). American Heart Journal, 109:1410.

Wamani H, Astrøm AN, Peterson S, Tumwine JK and Tylleskär T. 2007. Boys are more stunted than girls in sub-Saharan Africa: a meta-analysis of 16 demographic and health surveys. BMC Pediatric [Online], 7(17). Available from: http://www.ncbi.nlm.nih.gov/m/pubmed/17425787/http://pubmedcentral.nih.gov/articlerender.fcgi?tool=pubmed&pubmedid =17425787 [Accessed June 19th, 2012].

Waterlow JC. 1994. Causes and mechanisms of linear growth retardation (stunting). European Journal of Clinical Nutrition; 481 (Supplement): S1–S4

Wilson AC, Forsyth JS, Greene SA, Irvine L, Hau C, Howie PW. 1998. Relation of infant diet to childhood health: seven year follow up of cohort of children in Dundee infant feeding study. British Medical Journal, 316(7): 21-25.

Witten C, Jooste P, Sanders D and Chopra M. 2002. Successful micronutrient programmes: South Africa case study. Draft – February 2002. Unpublished. [Online]. Available from: http://www.dogpile.com/clickserver/_iceUrlFlag=1?rawURL=http%3A%2F%2Fwww.tulane.edu%2F~internut%2FIUNSpresentations%2FSouth%2520Africa%2FSouthAfrica.doc&0=&1=0&4=67.63.50.146&5=196.207.47.60&9=b6ea5e5027714999a14b1eb1be0dca01&10=1&11=info.dogpl&13=search&14=867530&15=main-title&17=4&18=1&19=0&20=2&21=2&22=Th7aNhJqNkg%3D&23=0&40=0jBKynNwjm8tRm2O%2FdrBRg%3D%3D&_IceUrl=true [Accessed June 19th, 2012].

Wolmarans P and Oosthuizen W. 2001. Eat fats sparingly – implications for health and disease. South African Journal of Clinical Nutrition, 14(3):S48-S55.

Wolmarans P, Langenhoven ML, Benadé AJS, Swanepoel ASP, Koue TJvW and Rossouw JE. 1988. Intake of macronutrients and their relationship with total cholesterol and high-density lipoprotein cholesterol: the Coronary Risk Factor Study, 1979. South African Medical Journal, 73: 12-15.

Wolmarans P, Seedat YK, Mayet FGH, Joubert G, Wentzel E. 1998. Dietary intake of Indians living in the metropolitan area of Durban. Public Health Nutrition, 2(1):55-60.

World Cancer Research Fund (WCRF) / American Institute for Cancer Research (AICR). 2007. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington DC: American Institute for Cancer Research.

World Diabetes Foundation (WDF). 2006. Diabetes facts. [Online]. Available from: http://worlddiabetesfoundation.org/composite-35.htm [Accessed June 19th, 2012].

World Diabetes Foundation (WDF). 2009. WHO launches Action Plan for NCDs. [Online]. Available from: http://worlddiabetesfoundation.org/composite-2447.htm [Accessed June 19th, 2012].

World Health Organization (WHO) and Food and Agriculture Organization (FAO). 2002. Joint Expert Consultation

on Diet, Nutrition and the Prevention of Chronic Diseases. WHO Technical Report Series No. 916. World Health Organization:Geneva, Switzerland.

World Health Organization. 2002. Report of the expert consultation on the optimal duration of exclusive breastfeeding. Geneva, Switzerland.

World Health Organization. 2008. The Global Burden of Disease: 2004 update. Geneva:Switzerland. [Online]. Available from: http://www.who.int/healthinfo/global_burden_disease/2004_report_update/en/ [Accessed June 19th, 2012].

World Health Organization. 2008a. The World Health Report 2008 – Primary Health Care. Geneva:Switzerland. [Online]. Available from: http://www.who.int/entity/whr/en/index.html [Accessed June 19th, 2012].

World Health Organization. 2010. Growth reference data for 5-19 years. [Online]. Available from: http://www.who.int/entity/growthref/en/ [Accessed June 19th, 2012].

World Health Organization. 2010a. Development of a WHO growth reference for school-aged children and adolescents. [Online]. Available from: http://www.who.int/entity/growthref/growthref_who_bull/en/index.html [Accessed June 19th, 2012].

World Health Organization. 2010b. Guidelines on HIV and infant feeding 2010: principles and recommendations for infant feeding in the context of HIV and a summary of evidence. Geneva, Switzerland.

World Health Organization. 2012. Population nutrient intake goals for preventing diet-related chronic diseases. [Online}. Available from: http://www.who.int/nutrition/topic/5_population_nutrient/en/index.html [Accessed June 19th, 2012].

World Health Organization. 2012a. Severe acute malnutrition [Online]. Available from http://www.who.int/nutrition/topics/malnutrition/en/index.html [Accessed June 19th, 2012].

Yach D, Cameron N, Padayachee N, Wagstaff L, Richter L and Fonn S. 1991. Birth to ten: child health in South Africa in the 1990s. Rationale and methods of a birth cohort study. Paediatric and Perinatal Epidemiology, 5:211-233.

Appendices

Appendix A

Assuring Health for All (AHA) in the Free State

CONSENT TO PARTICIPATE IN RESEARCH

You have been asked to participate in a research study.

You have been informed about the study by …….. .

You may contact Prof Corinna Walsh at 083 297 6030 at any time if you have questions about the research or if you are injured as a result of the research.

You may contact the Secretariat of the Ethics Committee of the Faculty of Health Sciences, UFS at telephone number (051) 4052812 if you have questions about your rights as a research subject.

Your participation in this research is voluntary, and you will not be penalized or lose benefits if you refuse to participate or decide to terminate participation.

If you agree to participate, you will be given a signed copy of this document as well as the participant information sheet, which is a written summary of the research. You are also giving permission that some of the same blood can be retained in storage for possible future research related to the present research question.

The research study, including the above information has been verbally described to me. I understand what my involvement in the study means and I voluntarily agree to participate.

_____ _____

Signature of Participant Date

For children:

_____ _____

Name/ Signature of Child Date

_____ _____

Signature of Parent Date

Assuring Health for All (AHA) in the Free State

TOESTEMMING TOT DEELNAME AAN NAVORSING

U is versoek om aan ‘n navorsingstudie deel te neem.
U is oor die studie ingelig deur …… .

U kan Prof Corinna Walsh enige tyd kontak by 083 297 6030 indien u vrae oor die navorsing het of as gevolg van die navorsing beseer is.

U kan die Sekretariaat van die Etiekkomitee van die Fakulteit Gesondheidsweteskappe, UV by telefoonnommer (051) 4052812 kontak indien u enige vrae het oor u regte as ‘n proefpersoon.

U deelname aan hierdie navorsing is vrywillig, en u sal nie gepenaliseer word of voordele verbeur as u weier om deel te neem of besluit om deelname te staak nie.

As u instem om deel te neem, sal ‘n ondertekende kopie van hierdie dokument sowel as die deelnemerinligtingsblad, wat ‘n geskrewe opsomming van die navorsing is, aan u gegee word .

Die navorsingstudie, insluitend die bogenoemde inligting is verbaal aan my beskryf. Ek begryp wat my betrokkenheid by die studie beteken en ek stem vrywillig in om deel te neem.

_____ _____

Handtekening van deelnemer Datum

Vir kinders:

_____ _____

Naam/ Handtekening van kind Datum

_____ _____

Handtekening van ouer Datum

Assuring Health for All (AHA) in the Free State

(Tshepiso ya Bophelo ho bohle ba Foreisitata)

TUMELLO YA HO NKA KAROLO DIPATLISISONG

O kopuwe ho nka karolo dipatlisisong.

O tsebisitswe ka dipatlisiso tsena ke .....

O ka ikopanya le Prof Corinna Walsh ho 083 297 6030 nako e nngwe le e nngwe ha o na le dipotso ka dipatlisiso kapa ha o ka wa lematseha ka lebaka la dipatlisiso.

O ka ikopanya le mongodi wa komiti ya Ethics ho Faculty ya Health Sciences, Yunivesithing ya Foreisitata nomorong ya (051) 4052812 ha o ena le dipotso ka ditokelo tsa hao jwalo ka motho ya nkang karolo dipatlisisong.

O nka ka karolo dipatlisisong ka ho ithaopa, ka hoo o ke ke wa ahlolwa kapa wa lahlehelwa ke di letho ha o ka wa hana ho nka karolo kapa wa tlohella ho nka karolo.

Ha ebe o dumela ho nka karolo, o tla fuwa lengolo le tshwanang le lena le saenuweng hammoho le lengolo le fanang ka tlhahiso leseding, eo e leng tlhaloso e ngotsweng ya dipatlisiso tsena.

Ke hlaloseditswe sepheo sa dipatlisiso, hammoho le tlhahiso leseding ena e ka hodimo ka molomo. Ke utlwisisa ho nka karolo ha ka dipatlisisong mme ke dumela ho nka karolo ka ho ithaopa, ntle le ho qobellwa.

_____ _____

Signature ya ya nkang karolo Letsatsi

Bakeng sa bana

_____ _____

Signature ea ngoana Letsatsi

_____ _____

Signature ea motsoali Letsatsi

Assuring Health for All (AHA) in the Free State

INFORMATION DOCUMENT

Study title: Assuring Health for All (AHA) in the Free State

Thank you for being willing to help us in this very important project. We are sure that the project will contribute to improving the health of all the people of the Free State.
We, the University of the Free State, Faculty of Health Sciences, are doing research on determining the factors involved in causing disease and disability in the Southern Free State. Research is just the process to learn the answer to a question. In this study we want to learn what factors need to be addressed in health programmes in the Free State. The study involves research and is not part of routine medical care.

Invitation to participate: We are asking/inviting you to participate in this research study, or/and asking for your permission to include your child in this research study.

What is involved in the study: The aim of the project is to get enough information regarding the development of chronic diseases like diabetes, stroke, high blood pressure and heart disease as well as HIV/AIDS to plan appropriate health and nutrition intervention strategies for the people of the Free State. Trompsburg, Philippolis and Springfontein have been chosen as the rural areas and Mangaung as the urban area.

For this study we need households whom we can follow for 12 years. The baseline survey will be done during March 2009 in Mangaung. You will be asked to visit the MUCPP clinic for one day to take the necessary measurements and to complete the questionnaires. After the baseline survey has been completed, we will implement a nutrition intervention in your community to address the problems identified in the baseline survey. This intervention will form part of the service learning interventions of the University. In addition to the services that we will render in the community, we will visit your community again after three to six years to repeat the measurements.

All the questionnaires will be filled out at MUCPP clinic by students from the University of the Free State. Respondents from the chosen households will be asked to complete the following questionnaires in an interview with the students:
Socio-demographic and household questionnaire,
Household food security and food procurement questionnaire,
Health questionnaire,
Knowledge, practices and attitudes (KPA) about nutrition questionnaire,
Diet and physical activity questionnaire.

We will also take some measurements such as weight, height, skinfold thicknesses, blood pressure, blood samples and a urine sample. With your permission we will draw 60ml of blood in adults and this will only be done once. In adults blood and urine samples will be used to determine the following: Full blood count; HbA1c; Glucose; Insulin;

Lipogram; Homocysteine; Red cell Folate; Serum Vitamin B12; Fibrinogen; Gamma glutermyl transferases (GGT); Carbohydrate-deficient transferrin (CDT); Ferritin; Uric acid; Creatinin; C-reactive protein; Albumin; Pre-albumin; Transferrin; Retinol-binding protein; TSH; Iodine (urine); Leptin; Tumour Necrosis Factor alpha; Interleuken 6; Melatonin; Brain natriuretic peptide; ACTH; Cortisol; Orexin; Urotensin-11; Endothelin 1; Plasminogen Activation Inhibitor (PAI-1); Adiponectin; Micro-albuminuria (urine); Glucose tolerance (sub-sample); FFA (sub-sample).

A short medical examination will be performed on all participants members to identify any serious health problems.

We would like to retain some of the same blood in storage for possible future research related to the present research question. Blood samples will be stored anonymously for a period of five years at the Department of Chemical Pathology at the University of the Free State. If you are unhappy to have your blood stored for future research, it will be disposed of at the end of the study, once the sample storage and record-keeping requirements of good research practice have been met.

It is very important that we gather quality data and knowledge. Because HIV/AIDS is a devastating illness and affects almost all aspects of health, it is necessary to know if HIV is absent before we analyse the data. It will be to your benefit as well as the benefit of the research to determine your HIV status. Therefore we will also ask permission to draw blood to determine your HIV status and ask questions about your HIV status which you are allowed not to answer. You will be asked to sign a separate consent form for the HIV test. You will receive pre- and post-testing counselling by a medical practitioner and all results will be kept strictly confidential in accordance with the guidelines of the Health Professions Council of South Africa (HPCSA). You will only be informed of your HIV result if you choose to be. All respondents who choose to be informed of the results will be informed by a medical doctor and referred for relevant management. None of the researchers (other than one doctor) will know the HIV status of any participants.

Blood tests will involve an analysis of the genetic composition of red blood cells and are aimed at increasing the understanding of the causes and behaviour of chronic diseases of lifestyle such as obesity, diabetes and heart disease. Genes are what you inherit from your parents. They are found in every part of your body and therefore they will be present in the blood that we draw. The findings may benefit/eventually benefit others in terms of prevention or treatment of diseases. You are free to refuse consent and you do not have to give reasons for doing so. The following arrangements have been made to ensure privacy and confidentiality of your genetic information: All blood samples will be stored anonymously. Your genetic material and information will be used in an identifiable form. The research may reveal information of potential importance to the future health of an identifiable or potentially identifiable participant or the participant's offspring.

Researchers will endeavour to provide information about the outcome of the research. If research generates information about you which may be of relevance to the health of other family members, your consent will be sought before offering to disclose such information to the family members concerned. Your material and information will not be released for other uses without consent, unless required by law.

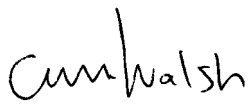
Risks of being involved in the study: Medical doctors and registered nurses will be responsible for safely drawing blood samples. In the unlikely event that an adverse event results from the procedure, you will be compensated for any expenses.

Benefits of being in the study: By participating in the study you will help us to develop health and nutrition strategies that will benefit the people of the Free State. You will be given pertinent information on the study while involved in the project and after the results are available.

Participation is voluntary, and refusal to participate will involve no penalty or loss of benefits to which you are entitled; you may discontinue participation at any time without penalty or loss of benefits to which you are otherwise entitled.

Confidentiality: Efforts will be made to keep personal information confidential. Absolute confidentiality cannot be guaranteed. Personal information may be disclosed if required by law. Organizations that may inspect and/or copy your research records for quality assurance and data analysis include groups such as the Ethics Committee for Medical Research and the Medicines Control Council. If results are published, this may lead to individual/cohort identification.

Kind regards



PROF CORINNA WALSH

Contact details: 083 297 6030 / 051 4013818(W)

INLIGTINGSDOKUMENT

Studietitel: Assuring Health for All (AHA) in the Free State

Dankie dat u bereid is om ons te help met hierdie baie belangrike projek. Ons is seker dat die projek sal bydra om die gesondheid van alle persone in die Vrystaat te verbeter. Ons, die Universiteit van die Vrystaat, Fakulteit Gesondheidswetenskappe, doen navorsing oor die faktore wat betrokke is by die oorsake van siekte in die Vrystaat. Navorsing is slegs die proses waardeur die antwoord op 'n vraagstuk verkry word. In hierdie studie wil ons leer watter faktore aangespreek moet word in gesondheidsprogramme in die Vrystaat. Die studie behels navorsing en is nie deel van roetine mediese behandeling nie.

Uitnodiging om deel te neem: Ons versoek/nooi u uit om aan 'n navorsingstudie deel te neem of/en vra u toestemming om u kind by die navorsingstudie in te sluit.

Wat behels die studie – Die doelwit van hierdie projek is om genoeg inligting in te samel oor die ontwikkeling van chroniese siektes soos diabetes, beroerte, hoë bloeddruk en hartsiektes sowel as MIV/VIGS om toepaslike gesondheids- en voedingintervensie strategieë te kan beplan vir die mense van die Vrystaat. Trompsburg, Philippolis en Springfontein is as die plattelandse areas gekies en Mangaung as die stedelike area.

Vir die studie benodig ons huishoudings wat ons vir 12 jaar kan opvolg. Die basislyn opname sal in Mangaung gedoen word tydens Maart 2009. U sal gevra word om die MUCPP kliniek vir een dag te besoek waar die nodige metings gedoen sal word en vraelyste voltooi sal word. Nadat die basislynopname voltooi is, sal ons 'n voedingintervensieprogram in u area implementeer om die probleme wat in die basislynopname identifiseer is aan te spreek. Hierdie intervensie vorm deel van die diensleer intervensies van die universiteit. Tesame met die intervensie sal ons ook die gemeenskap elke drie jaar tot ses jaar besoek om die metings te herhaal.

Al die vraelyste sal by MUCPP voltooi word deur studente van die Universiteit van die Vrystaat. Respondente van die gekose huishoudings sal gevra word om die volgende vraelyste te voltooi in 'n onderhoud met die student:

- Sosio-demografiese en huishoudelike vraelys,
- Huisehoudelike voedselsekuriteit en voedselverkrygings vraelys,
- Gesondheids vraelys,
- Kennis, praktyke en houding teenoor voeding vraelys,
- Dieet en fisiese aktiwiteit vraelys.

Ons sal ook sekere metings soos gewig, lengte, velvoudiktes, bloeddruk, bloed monsters en uriene monsters neem. Met u toestemming sal ons in volwassenes 60ml bloed trek en dit sal slegs een keer geskied. In volwassenes sal bloed en uriene monsters gebruik word om die volgende te bepaal: Volbloedtelling; HbA1c; Glukose; Insulien; Lipogram; Homositeïen; Rooisel Folaat; Serum Vitamien B12; Fibrinogeen; Gamma glutermyl transferases (GGT); Carbohydrate-deficient transferrin (CDT); Ferritin; Uriensuur; Kreatinien; C-reactiewe proteïen; Albumien; Pre-albumien; Transferrien; Retinol-binding proteïen; TSH; Jodium (uriene); Leptien; Tumor Nekrosis Faktor alfa; Interleuken 6; Melatonien; Brain natriuretic peptide; ACTH; Kortisol; Orexin; Urotensien-11; Endothelien

1; Plasminogen Activation Inhibitor (PAI-1); Adiponektien; Mikro-albumienuria (uriene); Glukose toleransie (sub-sample); FFA (sub-sample).

'n Kort mediese ondersoek sal ook gedoen word op sekere lede van die huishouding om ernstige gesondheidsprobleme te identifiseer.

Ons wil graag van die bloed bêre vir moontlike toekomstige navorsing wat verband hou met die huidige navorsingsvrae. Bloed monsters sal anoniem gestoor word vir 'n periode van vyf jaar. As u ongelukkig daaroor voel dat u bloed vir toekomstige navorsing geberg word sal daar aan die einde van die studie daarmee weggedoen word sodra die monsterbergings- en aantekeningsvereistes van goeie navorsingspraktyk nagekom is.

Dit is baie belangrik dat ons inligting van 'n hoë kwaliteit versamel. Omdat MIV/VIGS 'n siekte is wat amper alle aspekte van gesondheid beïnvloed, is dit nodig dat ons weet of MIV afwesig is voordat ons die data ontleed. Dit sal tot voordeel van uself en die navorsing strek indien u HIV status bepaal kan word. Dus sal ons toestemming vra om bloed te trek om u MIV status te bepaal en vrae oor HIV vra wat u nie hoef te antwoord indien u nie wil nie. U sal gevra word om 'n aparte teostemmingsvorm te voltooi vir die HIV toets. U sal voor en na die toets berading ontvang deur 'n mediese dokter en alle uitslae sal streng vertroulik hanteer word volgens die riglyne van die Health Professions Council of South africa (HPCSA). U sal slegs van u MIV uitslae in kennis gestel word indien u kies om dit te ontvang. Alle respondente wat kies om van hulle uitslae in kennis gestel te word sal deur 'n mediese dokter in kennis gestel word en verwys word vir die relevante hantering. Die navorsers (met uitsluiting van een dokter) sal nie weet wat u uitslae is nie.

Bloedtoetse sal die analise van die genetiese samestelling van rooibloedselle insluit en is gemik daarop om die oorsake en gevolge van chroniese siektes soos vetsug, diabetes en hartsiektes beter te verstaan. Gene is dit wat u van u ouers erf en word in elke deel van u liggaam aangetref. Daarom sal dit in enige weefsel of bloed wat deur ons verwyder word teenwoordig wees. Die bevindings kan tot ander se voordeel strek met betrekking tot voorkoming en behandeling van die toestand. Dit staan u vry om toestemming te weier en u hoef geen redes daarvoor te verstrek nie. Die volgende reëlings is getref om privaatheid en vertroulikheid van u genetiese inligting te verseker: Alle bloedmonsters sal anoniem geberg word. U genetiese materiaal en inligting sal in 'n identifiseerbarevorm gebruik word. Die navorsing mag inligting openbaar wat van potensiële belang mag wees vir die toekomstige gesondheid van 'n identifiseerbare of potensieel identifiseerbare deelnemer of die deelnemer se nakomelinge.

Navorsers sal poog om inligting oor die uitkoms van die navorsing te verskaf. As navorsing inligting aan die lig bring wat van belang mag wees vir die gesondheid van u familielede, sal u toestemming verkry word voordat sodanige inligting aan die betrokke familielede bekend gemaak word. U bloed en inligting sal nie sonder toestemming vir ander gebruike beskikbaar gestel word nie tensy vereis deur die wet.

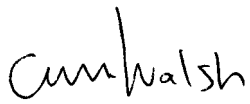
Risikos van deelname aan die studie: Mediese dokters of geregistreerde verpleegkundiges sal verantwoordelik wees vir die veilige neem van bloedmonsters. In die onwaarskynlike geval dat 'n negatiewe gevolg ontstaan as gevolg van die prosedure sal u vir enige onkostes vergoed word.

Voordele van deelname aan die studie: Deur aan die studie deel te neem sal u ons help om gesondheids- en voedingstrategieë te ontwikkel wat die mense van die Vrystaat sal baat. Die proefpersoon sal pertinente inligting oor die studie ontvang tydens betrokkenheid by die projek en agterna wanneer die resultate beskikbaar is.

Deelname is vrywillig, en weiering om deel te neem sal geen boete of verlies van voordele waarop die deelnemer andersins geregtig is behels nie; die proefpersoon kan te eniger tyd aan deelname onttrek sonder boete of verlies van voordele waarop die proefpersoon andersins geregtig is.

Vertroulikheid: Daar sal gepoog word om persoonlike inligting vertroulik te hou. Volkome vertroulikheid kan nie gewaarborg word nie. Persoonlike inligting kan bekend gemaak word as die wet dit vereis. Organisasies wat u navorsingsrekords mag ondersoek en/of kopieer vir kwaliteitsversekering en data-analise sluit groepe soos die Etiekkomitee vir Mediese Navorsing en die Medisynebeheerraad in. As resultate gepubliseer word kan dit lei tot individuele/groepsidentifikasie.

Vriendelike groete



Prof CORINNA WALSH

Kontakbesonderhede: 083 297 6030 / 051 4013818(W)

Assuring Health for All (AHA) in the Free State

(Tshepiso ya Bophelo ho bohle ba Foreisitata)

LENGOLO LA TLHAHISO LESEDING

Sehloho sa dipatlisiso: Assuring Health for All in the Free State

Re leboha ha o dumetse ho re thusa dipatlisisong tsena tse bohlokwa. Re tshepa ha dipatlisiso di tla re thusa ho ntlafatsa maphelo a batho bohle ba Foreisitata.
Rona, re le Yunivesithi ya Foreisitata, Lefapha la tsa Maphelo, re etsa dipatlisiso ho shebana le dintho tse bakang mafu le boqhwala mona Foreisitata e Borwa.
Dipatlisiso ke mokgwa wa ho ithuta karabo ya potso e itseng. Ka dipatlisiso tsena re batla ho ithuta hore na ke dintho dife tse hlokang hore di amuwe ditsamaisong tsa tsa bophelo mona Foreisitata. Tsena ke dipatlisiso feela, mme ha se karolo ya tshebeletso ya tsa bongaka.

Sememo sa ho nka karolo: Re a o kopa/ re a o mema ho nka karolo dipatlisisong tsena, ebile/ kapa re kopa tumello ya hao ho sebedisa ngwana wa hao dipatlisisong tsena.
Dipatlisiso tsena di kenyeleditse eng: Sepheo sa dipatlisiso tsena ke ho fumana tlhahiso leseding e lekaneng mabapi le tswello pele ya mafu a kang diabetes, stroke, high blood pressure le mafu a pelo hammoho le HIV/AIDS hore ho tle ho thalwe mekgwa ya tsamaiso ya tsa bophelo le phepo bakeng sa batho ba Foreisitata. Trompsburg, Phillippolis le Springfontein di kgethilwe jwalo ka dibaka tsa mahaeng mme Mangaung e kgethilwe e le sebaka sa ditoropong tse tla sebediswang dipatlisisong.
Bakeng sa dipatlisiso tsena re hloka ho sebetsa le malapa ohle motseng wa lona bakeng sa dilemo tse 12. Dipatlisiso tsa pele di tla qala ka Hlakubele (March) 2009. O tla kopuwa ho etela MUCPP letsatsi le le leng hore o tle o tsebe ho tlatsa diforomo le hore o methuwe. Kamora dipatlisiso tsena tsa pele, ho kenywa tshebetsong mekgwa ya tlhokomelo mabapi le tsa phepo sebakeng seo o dulang ho sona hore ho tle ho lokisanwe le mathatha a tla be a hlahelletse dipatlisisong tsa pele. Tlhokomelo ena e tla ba karolo ya ho ithuta ho bile ho fanwa ka ditshebeletso ke baithuti ba Yunivesithi. Hammoho le ditshebetso tseo re tla be re fana ka tsona motseng, re tla etela motse wa hao ka mora dilemo tse tharo hoisa ho tse tseletseng hore ho phethwe ho metha.

Diforomo tsohle di tla tlatswa sebakeng seo liphuputso li tla tswareloa teng ke baithuti ba tswang Yunivesithing ya Foreisitata kapa ke bathusi ba kwetlisitsweng ba tswang motseng wa hao. Batho ba nkang karolo ho tswa malapeng a kgethilweng ba tla kopuwa ho tlatsa diforomo tse latelang ho ya ka dipotso tseo ba tla beng ba di botswa ke moithuti kapa mosebeletsi wa setjhaba:

- Dipotso ka maemo a hao le ka lelapa la hao,
- Theko ya dijo le ‘food security’,
- Tsebo, tshebetso le mekgwa e amanang le phepo,
- Tsela ya ho ja le boikwetliso.

Re tla o metha boima, botelete, botenya ba momeno wa letlalo (skinfold) le kgatello ya madi (blood pressure), o tla nkuwa madi hammoho le metsi. Ho batho ba baholo re tla hula madi a kana ka dimililitara tse 60 hona ho tla etswa hanngwe feela. Ho tla etswa dihlahlobo tsa bophelo ho batho ba itseng ba lelapa ho shebana le mathata a

bophelo a tshosetsang. Ka tumello ea hau re tla ntsa madi a ka bang dimelimetara tse mashome a tseletseng (60ml) ho motho e moholo le tse leshome le metso e mehlano, mme hona ho tla etwa hangoe feela. Madi le metsetse e nkuoeng ho batho ba baholo e tla sebediswa ho hlalhoba matsoai le matsooeana a fumanehang ho tsona a kenyeletsang tse latelang: Full blood count; HbA1c; Glucose; Insulin; Lipogram; Homocysteine; Red cell Folic acid; Serum Vitamin B12; Fibrinogen; Gamma glutermyl transferases (GGT); Carbohydrate-deficient transferrin (CDT); Ferritin; Uric acid; Creatinin; C-reactive protein; Albumin; Pre-albumin; Transferrin; Retinol-binding protein; TSH; Iodine (urine); Leptin; Tumour Necrosis Factor alpha; Interleuken 6; Melatonin; Brain natriuretic peptide; ACTH; Cortisol; Orexin; Urotensin-11; Endothelin 1; Plasminogen Activation Inhibitor (PAI-1); Adiponectin; Micro-albuminuria (urine); Glucose tolerance (sub-sample); FFA (sub-sample). Tse ding tsa matsoai le matsoaeana ana di tla hlahlojoa mading le metsetseng ea bana

Re ka rata ho boloka a mang a madi hore a tle a tsebe ho sebediswa ka nako e tlang dipatlisisong tse tshwanang le tsena. Madi ana a tla bolokwa kantle le ho ngolwa mabitso bakeng sa dilemo tse hlano. Ha o sa rate ha madi a hao a bolokwa bakeng sa dipatlisiso tse ding, a tla lahlwa ha ho qetwa ka dipatlisiso le hang feela ha a ile a bolokwa hantle e bile dintho tse hlokahalang bakeng sa dipatlisiso di ile tsa fumanwa ka mokgwa o nepahetseng.

Ho bohlokwa haholo hore tlhahiso leseding eo re e fumanang ke e hlwahlwa. Hobane lefu la HIV e le le hlokofatsang haholo, ebile le ama maphelo a rona, ho a hlokahala hore re tsebe hore kokwana-hloko ena e teng kapa tjhe. Ke molemong oa hau le oa mofuputse ho fumana boemo ha hau ba HIV. Ka hona re tla kopa tumello ea ho ntsa madi ho uena ho ea hlahloba boemo ba hau ba HIV mme re tla u botsa lipotso tse ling tse amanang le boemo ba hau ba HIV tseo u sa qobelloeng ho li araba. U ka li araba ka ho rata ha hau. U tla kopuoa ho saena foromo eo u re fang tumello ea ho hlahloba boemo ba hau ba HIV. Ka hoo re tla hula madi le ho o botsa dipotso mabapi le maemo a hao a HIV. O dumelletswe ho se arabe dipotso tsena. Ho tla buisanwa le wena ho tebesitswe maikutlo pele le ka mora dihlahlobo tse tla etswa ke ngaka, mme diphetho tsohle di tla ba sephiri ho ya ka ditaelo tsa Health Professions Council ya South Africa (HPCSA). O tla tsebiswa ka maemo a hao a HIV feela ha o kgetha hore o tsebiswe. Batho bohle ba batlang ho tsebiswa ka sephetho sa dihlahlobo tsa HIV, ba tla tsebiswa ke ngaka, mme ba tla romellwa ho batho ba tla tseba ho ba fa thuso. Babatlisisi ba bang (ka ntle ho dingaka) ba ke ke ba tseba maemo a ba nka karolo a HIV.

Ho hulwa ha madi ho kenyelleditse ho hlahloba ‘genetic composition’ e ho di ‘red blood cells’, mme hona ho tla thusa ho phahamisa kutlwisiso ya dintho tse bakang mafu a kang monono, diabetes le mafu a pelo. Di ‘genes’, ke dintho tseo o di futsang ho tswa ho batswadi ba hao. Di fumanwa dikarolong tsohle tsa mmele kahoo di a fumaneha le ho madi a tla hulwa. Dintlha tse tla fumanwa di tla thusa batho ba bang ka ho thusa ho thibela, kapa ho phekola mafu a fapaneng. O na le tokelo ya ho hana ho nka karolo ebile ha ho hlokahale hore o fane ka lebaka. Ho netefatsa hore dintlha tse tla fumanwang ho wena di dula e le sephiri, ho entswe dintho tsena tse latelang: Madi ohle a tla bolokwa ntle le ho ngolwa mabitso. ‘Genetic material’ ya hao e tla sebediswa ka mokgwa o tsebahalang. Dipatlisiso tsena di ka nna tsa fana ka tlhahiso leseding e bohlokwa bakeng sa bokamoso ba monka karolo kapa ba bana ba hae.

Babatlisisi ba tla leka ho fana ka lesedi mabapi le sephetho sa dipatlisiso. Ha dipatlisiso di ka fana ka dinthla tse leng bohlokwa bakeng sa maphelo a ba bang ba lelapa, tumello e tla kopuwa ho wena pele batho ba amehang ba tsebiswa. Dintlha tsohle tsa hao di ke ke tsa sebediswa nqeng tse ding kantle ho tumello ya hao, kantle le ha ho ka hlokeha hore ho etswe jwalo ho ya ka molao.

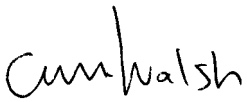
Kotsi tse ka bang teng dipatlisisong: Dingaka le baoki ba tla ikarabella ho huleng ha madi ka mokgwa o bolokehileng. Ha ho ka etsahala hore ho be le se o etsahallang se sa lokang o tla lefuwa ditshenyehelo tsa hao tsohle.

Ditholwana tsa ho nka karolo: Ha o nka karolo o tla thusa ho tsweletsa pele metjha ya tsa bophelo le phepo ho thusa batho ba Foreisitata. O tla fuwa tlhahiso leseding e bohlokwa tsamaong ya dipatlisiso le ha sephetho sa dipatlisiso se fumaneha.

O nka karolo ka ho ithaopa, mme ha o hana ho nka karolo o ke ke wa lahlehelwa ke letho; o ka tlohella ho nka karolo nako e nngwe le e nngwe ntle le ho lahlehelwa ke letho.

Sephiri: Ho tla etswa maleba-leba a hore dintlha tsa hao di dule di le lekunutu. O tshepiswa lekunutu ka hohle-hohle. Dintlha tsa hao di phatlalatswa feela ha molao o re jwalo. Mekgatlo e tla hlahloba kapa e tla kopisa dintlha tsa hao ho lekola boleng e kenyeleditse e kang Ethics Committee ya Medical Research hammoho le Medicine Control Council.

Ha diphetho di ka phatlalatswa, hona ho ka lebisa ho tsebiswa ha motho kapa sehlopha.



Ka boikokobetso

Prof Corinna Walsh

Mohala: 083 297 6030 / 051 401 3818

Assuring Health for All (AHA) in the Free State
Participation letter

Dear Participant

Thank you for being willing to help us in this very important project. We are sure that the project will contribute to improving the health of all the people of the Free State.

At the time you receive this letter you would have already been visited by a fieldworker and you have already signed consent to give a blood sample. This letter serves to inform you of the date and time the blood sample and other measurements will be taken at the MUCPP clinic (basement).

IMPORTANT INFORMATION

1. You must be at MUCPP on **……** by 0….h00.
2. You **MUST NOT EAT OR DRINK** anything after ten o'clock of the previous night (10 pm of the night before). This is necessary for the glucose test to be accurate.
3. You **MUST BRING YOUR ID DOCUMENT** with you
4. Bring along a list of people that live in the household with you and their birth dates.
5. You will receive something to eat and drink after the blood sample is taken.
6. After participating in the project, you will receive an amount of R10 to pay for your transport costs.
7. If you are employed, please show this letter to your employer.

Dear Employer

This serves to ask you to give one day's paid leave to……. in order to allow him/her to attend his appointment with the research team of the Faculty of Health Sciences at the University of the Free State.

Thank you for your cooperation. For any further information please contact Prof Corinna Walsh at 083 297 6030.

Prof C Walsh (project leader)

Assuring Health for All (AHA) in the Free State
Deelname brief

Beste Deelnemer

Dankie dat u bereid is om ons te help met hierdie belangrike projek. Ons is seker dat die projek sal bydra tot die verbetering van gesondheid van al die mense in die Vrystaat.

Teen die tyd wat u hierdie brief ontvang het ‘n veldwerker u al reeds besoek en u het toestemming gegee om deel te neem aan die projek en ‘n bloed monster te gee Met hierdie brief wil ons u graag in kennis stel van die datum en tyd wat die bloed getrek sal word en ander mates geneem sal word by die navorsingseenheid (saal)naaste aan u woning.

BELANGRIKE INLIGTING

1. U moet by MUCPP wees op **……** teen 0….h00.
2. U **MOET NIKS EET OF DRINK** na tien uur die vorige aand (10 pm van die aand voor die toets). Dit is nodig vir die glukose toets om betroubaar te wees.
3. U **MOET U ID DOKUMENT** saam met u kliniek toe bring
4. Bring asseblief ‘n lys van persone wat saam met u in die huis woon saam met hule geboortedatums.
5. U sal ‘n ligte ete en iets om te drink ontvang nadat die bloedtoets voltooi is.
6. Nadat u deelgeneem het , sal u ‘n bedrag van R10 ontvang om te betaal vir u vervoerkostes.
7. Indien u werk, moet u asseblief hierdie brief aan u werkgewer wys.

Geagte Werkgewer

Met hierdie brief vra ons dat u een dag betaalde verlof toestaan aan……. om dit vir haar/hom moontlik te maak om hierdie afspraak met die navorsingsspan van die Fakulteit Gesondheidswetenskappe by die Universiteit van die Vrystaat by te woon.

Dankie vir u samewerking. Vir verdere inligting kontak asseblief vir Prof Corinna Walsh by 083 297 6030.

Prof C Walsh (projekleier)

Assuring Health for All in the Free State

(Tshepiso ya Bophelo ho bohle ba Foreisitata)

Lengolo la ho nka karolo

Ho ya nkang karolo

Re leboha ha o dumetse ho re thusa mosebetsing ona o bohlokwa. Re tshepa ha mosebetsi ona o tla thusa ho ntlafatsa maphelo a batho bohle ba Foreisitata.

Nako eo o fumanang lengolo lena, o tla be o se o etetswe ke e mong wa bathusi ba rona ebile o tla be o se o saenile ho fana ka tumello ya ho hula madi. Lengolo lena ke le o tsebisang ka letsatsi le nako eo tla methwang le ho hulwa madi tliliniking e haufi le lelapa la hao.

DINTLHA TSA BOHLOKWA

1. O tlamehile ho ba MUCPP ka di ..... nako e le 0.....h00.
2. **O SE KE WA JA KAPA WA NWA** letho ka mora hora ya leshome(10:00) bosiu bo ka pele ho fihlela o hlahlobuwa tliliniking. Hona ho bohlokwa hore dihlahlobo tsa tswekere e be tse nepahetseng.
3. O **TSHWANETSE HO TLA LE BUKANA YA HAO YA BOITSEBISO (ID)** ha o tla
4. O tle o tshwere le wena lenane la batho bohle bao o dulang le bona ka tlung hammoho le matsatsi a bona a tswalo
5. O tla fumana dijo kamora hore o hulwe madi.
6. O tla fuwa tjhelete e e kana ka R10 bakeng sa transport ha o nka karolo dipatlisisong.
7. Ha e be o sebetsa, re kopa o bontshe monga hao lengolo lena.

Monga mosebetsi

Lengolo lena le kopa hore of fane ka letsatsi le leng leng le patallwang ho ..... hore a tle a tsebe ho ba teng ka nako eo a e beetsweng bakeng sa sehlopha se etsang dipatlisiso sa Lefapha la tsa maphelo Yunivesithing ya Foreisitata.

Re lebohela tshebedisano mooho ya hao. Ha ebe o hloka tlhalosetso e fetang mona o ka ikopanya le Dr Corinna Walsh ho 083 297 6030.

Prof C Walsh (moetelledipele)

Assuring Health for All (AHA) in the Free State
Dietary intake questionnaire
24-hour recall

Town: _____ 1

Household number: 2-5

Member number (as on socio-demo form): 6-7

Interviewer: _____ 8-9

Food and fluid intake (expressed in exchanges):

Food/Drinks and amounts	Milk and milk products	Meat and meat alternatives	Legumes	Fruit ß-carotene	Vegetables ß-carotene	Fruit Vit C	Vegetables Vit C	Fruit other	Vegetables other	Bread and cereals	Fats and oils	Sweets/Sugar	Alcohol
Breakfast and mid-morning													
Lunch and mid afternoon													
Supper and late night													
Total:													

Evaluation of dietary intake

	Quantity	Energy	Protein	CHO	Fat	Below requirement 1	Within requirement 2	Above requirement 3	
Milk and milk products		530	8	12	5				10
Meat and meat alternatives		315	7		5				11
Legumes		500	7	21	1				12
Soy beans		630	13	8	7				13
Fruit β-carotene		250		15					14
Vegetables β-carotene									15
Fruit vit C		250		15					16
Vegetables vit C									17
Fruit other		250		15					18
Vegetables B		150	2	7					19
Bread and cereal		285	3	15					20
Fats and oils		190			5				21
Sweets/Sugar		170		10					22
Alcohol									23
TOTAL									

Calculated estimated total values for:

Carbohydrate (g): 24-26

Protein (g): 27-29

Fat (g): 30-32

Energy (kJ): 33-37

Food frequency questionnaire

Number of times per day, per week or per month (only use one option)

Food	/day	/week	/month	
Sweets/ chocolates				38-43
Chips (crisp)				44-49
Cake/ biscuits				50-55
Cool drinks				56-61
Cremora				62-67
Coffee				68-73
Tea				74-79
Sugar				1-6
Full-cream milk				7-12
Low fat/ skim milk				13-18
Eggs				19-24
Peanut butter				25-30
Soya mince/ legumes (baked beans, dried beans/peas, lentils)				31-36
Chicken				37-42
Red meat				43-48
Fish				49-54
Bread				55-60
Porridge, cooked				61-66
Cereal (eg. Morevite/ Pronutro)				67-72
Samp/ mielie rice				73-78
Margarine/ oil/ fat				1-6
Fruit juice				7-12
Fruit				13-18
Vegetables				19-24
Salt/ stock/ Royco				25-30
Alcohol _____				31-36

Assuring Health for All (AHA) in the Free State
Dietary intake questionnaire
Children 0-2 years

Town: _____ 1
Household number: 2-5
Member number: 6-7
Interviewer: _____ 8-9

Is the child currently being breast fed? 1 = yes 2 = no 10
Was the child previously breast fed? 1 = yes 2 = no 11
If so for how long (weeks)? _____ 12-13
At what age were solid foods introduced into the child's diet (weeks)? _____ 14-15
How long was the child exclusively breast fed? _____ 16-17
Is the child currently being formula fed? 1 = yes 2 = no 18
If the child is not breast fed, what formula/ milk is used? _____ 19-20
How many bottles does the child receive per day? _____ 21-22
What is the size of the bottle? _____ 23
How many scoops of formula are used to mix one bottle? _____ 24-25
What size tin of formula is bought? 1 = 400g 2 = 900g 26
How long does the tin of formula last (days)? _____ 27-28
Does this fall within recommendations (office use)? 1 = yes 2 = no 29

Where does the mother/ caregiver get the formula milk? 30
1. Buys it at store
2. At clinic

NB: If the child eats solid foods, complete the 24 hour recall.

TYPES OF FORMULAS
1. Pelargon
2. Nan 1
3. Nan 2
4. Nan Alfare'
5. Lactogen
6. S26
7. SMA
8. Infamil
9. Infasoy
10. Isomil
11. Cow's milk
12.
13.
14.

REQUIREMENTS FULFILLED:
Calculate (no of bottles) ..... X (size of bottles).....ml = ... ml (total amount/ day)

REQUIREMENTS

Age:	RDA
0-2 mo	360-720ml
3mo	600-1080ml
4mo	900-1050ml
5 mo	900-1200ml
6 mo	720-1050ml (+ solids)
7-8 mo	840-960ml (+ solids)

Assuring Health for All (AHA) in the Free State

Anthropometry

Area in Mangaung:_____ | | 1

Household number: | | 2-4

Member number (as on socio-demographic form): | | 5-6

Interview Date: | **D D M M Y Y Y Y** | 7-14

Measurer (interviewer):_____ | | 15-16

Weight (kg):_____ | | 17-21

Height (cm): _____ | | 22-26

If height cannot be measured:

Knee height (cm):_____ | | 27-31

Demispan (cm):_____ | | 32-36

Circumferences (cm):

Upper-arm (adults and children):_____ | | 37-40

Waist (adults): _____ | | 41-45

Hip (adults): _____ | | 46-50

Wrist (adults):_____ | | 51-54

Head circumference (children):_____ | | 55-58

Bio-impedance fat percentage (adults):_____ | | 59-62

Skinfold thicknesses (mm):

Triceps (adults and children):	_____	_____	_____	63-64
Biceps (adults):	_____	_____	_____	65-66
Supra-ileac (adults):	_____	_____	_____	67-68
Subscapular (adults):	_____	_____	_____	69-70
Thigh (adults):	_____	_____	_____	71-72
Calf (adults):	_____	_____	_____	73-74