

**Changes in dietary intakes and cardiovascular
disease risk factors in black South Africans from
2005 to 2015: The PURE-SA-NW study**

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PREFACE

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ABSTRACT

Background:

The diets of populations are constantly changing, and the South African population is no exception. Over the last couple of decades, the diets of rural and urban South Africans have been undergoing a rapid nutrition transition, changing from prudent traditional diets that have been typically low in fat and energy, to Westernised diets containing foods high in fat and energy. This shift in dietary patterns and nutrient intakes is referred to as the nutrition transition. Dietary changes associated with a nutrition transition are linked to an increase in non-communicable diseases (NCDs) globally, but also in South Africa in particular. The World Health Organisation (WHO) has identified NCDs as the leading cause of death worldwide. The 2018 NCD report of the WHO revealed that the majority (19%) of NCD deaths in South Africa could be attributed to cardiovascular disease (CVD). The emergence of cardiovascular diseases (CVDs) has been linked to several preventable risk factors, including hypertension, overweight, obesity, diabetes and high cholesterol levels. Lowering the prevalence of these preventable risk factors would be key to addressing the ongoing public health dilemma. Research evidence has shown that dietary and nutrient intake is associated with CVD risk. A growing body of evidence also suggests that the type of dietary fat rather than the total amount of dietary fat consumed drives CVD risk.

The primary aim of this thesis is to examine the changes in dietary intake from 2005 to 2015, as well as the associations between the changes in dietary fat intake and the changes in CVD risk from 2005 to 2010, of rural and urban South African groups amid a nutrition transition.

Methods:

This study is a sub-study of the South African leg in the North-West province of the multinational Prospective Urban and Rural Epidemiological (PURE) study (PURE-SA-NWP). Data for PURE-SA-NWP were collected from two communities, one urban and one rural. Baseline data were collected in 2005, with two follow-up collections conducted in 2010 and 2015. Dietary data were collected using a 145-item culturally sensitive quantified food frequency questionnaire (QFFQ). Only participants with complete dietary data were included in the analysis, resulting in a total of 1154 participants in Chapters 3 and 4, and 722 participants in Chapter 5.

In article 1 (Chapter 3) Wilcoxon's two-sample test was used to test for differences between the rural and urban participants (by gender) in terms of their nutrient intakes, as well as the changes in nutrient intakes between 2005 and 2010. Wilcoxon's signed-rank test was used to test for changes in nutrient intakes between 2005 and 2010, for each of the four groups (by gender and

locality). In article 2 (Chapter 4), stepwise regression models were estimated to test for associations between the change in fat intake and the change in CVD risk factors. In article 3 (Chapter 5), a linear mixed-model approach was used to assess the trends in energy and nutrient intake of urban and rural men and women over time (2005-2010-2015).

Results

In 2010, rural participants consumed the amount of energy (men 9·7 MJ/d; women 9·1 MJ/d) that urban participants had consumed in 2005 (men 9·9 MJ/d; women 9·0 MJ/d). Despite the higher energy intake, not all the participants met total micronutrient needs in 2010. Among rural men, an increase in the change in waist circumference from 2005 to 2010 was associated with a decrease in the change in poly-unsaturated fat ($\beta=-0.280$; $p=0.002$) and an increase in the change in mono-unsaturated fat ($\beta=0.200$; $p=0.027$) intake.

For rural men and women, trend analysis (2005, 2010, 2015) indicated significant upward trends in total energy ($p < 0.001$) (men: 7.60 MJ to 13.26 MJ, women: 6.60 MJ in 2005 to 11.89 MJ), and most macro- ($p < 0.001$) and micronutrients ($p < 0.001$) from 2005 to 2015. Upward trends were also observed in the proportion of total energy intake from all macronutrients (except alcohol) ($p < 0.001$). The observed trends for rural and urban participants differed significantly ($p < 0.001$ for interaction effect) for total energy intake as well as intakes of most micro- and macronutrients.

Discussion and conclusion:

Findings from the 2005 to 2010 data clearly showed that the nutrition transition was occurring at a rapid pace in rural and urban groups. Despite the significant increases in energy and fat intake from 2005 to 2010, changes in fat intake were, overall, not strongly associated with the changes in CVD risk factors.

From the in-depth trend analysis of energy and nutrient intakes of urban and rural participants of the PURE-SA-NWP study (2005-2015), it is evident that rural and urban groups differ significantly in terms of dietary changes occurring during the nutrition transition. The rural groups were experiencing a nutrition transition so rapid that they not only caught up with the urban groups in terms of energy and nutrient intake but had surpassed them by 2015. Energy and nutrient intakes in the urban areas, on the other hand, seemed to have flattened down by 2015. Our results highlight the importance of timeous monitoring of the various drivers of the nutrition transition and the urgent need to steer the nutrition transition in a more positive direction to prevent the progressive worsening of our NCD burden.

Key terms: Risk factors, Dietary intake, Nutrition transition, Dietary fat, Cardiovascular disease, trend analysis

OPSOMMING

Agtergrond:

Die dieet van bevolkings verander gedurig en die Suid-Afrikaanse bevolking is geen uitsondering nie. Gedurende die afgelope paar dekades het die diëte van landelike en stedelike Suid-Afrikaners 'n versnelde voedingsomwenteling ondergaan. Voedsame tradisionele diëte, gewoonlik laag in vet en energie, is vervang met verwesterde diëte wat voedselsoorte hoog in vet en energie insluit. Hierdie verskuiwing in dieetpatrone en nutrientinnames staan bekend as die voedselomwenteling. Dieetveranderinge wat met die voedselomwenteling gepaard gaan, word verbind met 'n toename in nie-oordraagbare siektes (NOS) wêreldwyd en ook spesifiek in Suid-Afrika. NOS is deur die Wêreld Gesondheidsorganisasie (WGO) geïdentifiseer as die vernaamste oorsaak van sterftes wêreldwyd. Die 2018 NOS-verslag van die WGO het aan die lig gebring dat kardiovaskulêre siekte (KVS) die NOS is wat vir die grootste persentasie sterftes verantwoordelik is (19% van alle NOS sterftes). Die opkoms van van kardiovaskulêre siektes (KVS) word verbind met verskeie voorkombare risikofaktore soos hipertensie, oorgewig, diabetes, vetsug en cholesterolvlakke. Om die voorkoms van hierdie voorkombare risikofaktore te verlaag, sal 'n sleutelrol speel in die voortslepende openbare gesondheidsdilemma. Daar is toenemende wetenskaplike getuienis wat aandui dat die tipe vet eerder as die hoeveelheid totale vet wat ingeneem word, geassosieer word met KVS-risiko.

Die primêre doelstelling van hierdie proefskrif is eerstens om die veranderinge in die energie- en nutrientinnames van 2005 tot 2015 in landelik- sowel as stedelike groepe te ondersoek. Verder poog dit ook om die assosiasies tussen die veranderinge in vet innames en die veranderinge in KVS-risiko van 2005 tot 2010 in hierdie groepe te midde van 'n voedingsomwenteling te ondersoek.

Metodes:

Hierdie studie is 'n substudie van die Suid-Afrikaanse been, in die Noordwes-provinsie, van die multinasionale *Prospective Urban and Rural Epidemiological* (PURE) studie (PURE-SA-NWP). Data vir PURE-SA-NWP is in twee gemeenskapsgroepe ingesamel, een landelike en een stedelike gebied. Basislyndata is in 2005 versamel, met twee opvolgingsamplings in 2010 en 2015. Dieetdata is ingesamel deur middel van 'n 145-item kultureel-sensitiewe gekwantifiseerde voedselfrekwensie-vraelys. Slegs deelnemers met volledige data is in die ondersoek ingesluit. Dit het tot gevolg gehad dat 1154 deelnemers in hoofstuk 3 en 4, en 722 deelnemers in hoofstuk 5 ingesluit is.

In artikel 1 (Hoofstuk 3) is Wilcoxon se tweesteekeproef-prosedure gedoen om te toets vir verskille tussen landelike en stedelike deelnemers (volgens geslag) ten opsigte van hulle nutrientinnames, sowel as vir die veranderinge in nutrientinnames van 2005 tot 2010. Wilcoxon se betekenderang-toets is gebruik om te toets vir veranderinge in nutrientinnames van 2005 tot 2010, vir elk van die vier groepe (volgens geslag en lokaliteit). In artikel 2 (Hoofstuk 4) is stapsgewyse regressiemodelle beraam om te toets vir verbande tussen die veranderinge in vetinnames en die verandering in KVS risikofaktore. In artikel 3 (Hoofstuk 5) is 'n linieêr-gemengde model benadering gevolg om die tendense in energie- en nutrientinnameveranderinge van landelike en stedelike mans en vroue oor tyd te beoordeel (2005-2010-2015).

Resultate

In 2010 was die landelike deelnemers se energie-inname (mans 9.7 MJ/d; vroue 9.1 MJ/d) gelykstaande aan die hoeveelheid wat stedelike deelnemers in 2005 ingeneem het (mans 9.9 MJ/d; vroue 9.0 MJ/d). Ten spyte van die hoër energieinname, het nie alle deelnemers hulle mikronutrient-behoefte in 2010 bevredig nie. Ten opsigte van landelike mans is die toename in die verandering in die middellyfomtrek van 2005 – 2010 geassosieer met 'n afname in die verandering in poli-onversadigde vet ($\beta = -0.280$; $p = 0.002$) asook 'n toename in die verandering in mono-onversadigde vet ($\beta = 0.200$; $p = 0.027$) inname.

Ten opsigte van landelike mans en vroue toon tendens-analise (2005-2010-2015) beduidende opwaartse tendense in die innames van energie ($p < 0.001$) (mans: 7.60 MJ to 13.26 MJ, vroue: 6.60 MJ in 2005 to 11.89 MJ), en meeste makro- ($p < 0.001$) en mikronutriente ($p < 0.001$) van 2005 tot 2015. Opwaartse tendense is ook waargeneem ten opsigte van die persentasie energie afkomstig van alle makronutriente (behalwe alkohol) ($p < 0.001$). Die waargenome tendense vir landelike en stedelike groepe het beduidend van mekaar verskil ($p < 0.001$ vir interaksie-effek) in terme van totale energie-inname sowel as die innames van meeste mikro- en makronutriente.

Bespreking en gevolgtrekking:

Die bevindinge van die 2005 tot 2010 data toon duidelik dat 'n voedingsomwenteling steeds teen 'n snelle tempo in landelike en stedelike bevolkings voortgegaan het. Ten spyte van die beduidende veranderinge in energie en vetinnames van 2005 tot 2010, het die verandering in vetinname oor die algemeen nie sterk verbande met die verandering in KVS risiko faktore getoon nie.

Die indiepte tendensanalise van energie en nutrientinnames van landelike en stedelike mans en vroue in die PURE-SA-NWP studie (2005-2015) dui duidelik aan dat landelike en stedelike groepe beduidend van mekaar verskil met betrekking tot die dieetveranderinge wat gedurende die

voedselomwenteling plaasvind. Die landelike groepe het so 'n snelle voedselomwenteling ervaar dat hulle nie net die stedelike groepe ingehaal het wat energie- en nutriëntinnames betref nie, maar hulle teen 2015 verbygesteek het. Daarteenoor het die energie- en nutriëntinnames van die stedelike groepe, skynbaar afgeplat teen 2015. Ons resultate beklemtoon die belangrikheid van tydigse monitering van die van die verskillende dryfvere van die voedselomwenteling en die feit dat die voedselomwentelinge dringend in 'n meer positiewe rigting gestuur moet word om te verhoed dat ons NOS-las progressief swaarder raak.

Sleuteltermes: Risikofaktore, Dieetinname, Voedingsomwenteling, dieetvet, kardiovaskulêre siekte, tendens analise

LIST OF ABBREVIATIONS

ABBREVIATION	EXPLANATION
AGEs	Advanced glycation end products
AI	Adequate intake
AIDS	Acquired immunodeficiency syndrome
AMI	Acute myocardial infarction
ART	Anti-retroviral therapy
BMI	Body mass index
BP	Blood pressure
CAD	Chronic artery disease
CHD	Chronic heart disease
CHF	Congestive heart failure
COPD	Chronic obstructive pulmonary disease
CRP	C-reactive protein
CVD	Cardiovascular disease
DALYs	Disability-adjusted life-years
DASH	Dietary Approaches to Stop Hypertension
DBP	Diastolic blood pressure
EAS	European Atherosclerosis Society
EMM	Estimated marginal means
ESC	European Society of Cardiology
ES	Effect size
FAO	Food and Agriculture Organisation
FBDG	Food based dietary guidelines
FFAs	Free fatty acids
FFQ	Food frequency questionnaire
Hb1Ac	Haemoglobin A1C
HDL	High-density lipoprotein
HIV	Human immunodeficiency virus
IHD	Ischaemic heart disease
IL-6	Interleukin-6
LDL	Low-density lipoprotein
MCH	Mean corpuscular haemoglobin
MI	Myocardial infarction
MJ	Mega joule
MRC	Medical Research Council
MUFA	Mono-unsaturated fatty acids
NCD	Non-communicable disease
NDoH	National Department of Health
NO	Nitric oxide
NS	Not significant
NT	Nutrition transition
NUGAG	Guidance expert advisory group
PAI-1	Plasminogen activator inhibitor
PCOS	Polycystic-ovarian syndrome
PUFA	Poly-unsaturated fatty acid
PURE	Prospective Urban and Rural Epidemiological study
PURE-SA-NWP	South African leg of the PURE study in the North West province.

ABBREVIATION	EXPLANATION
QFFQ	Quantified food frequency questionnaire
RCT	Randomised controlled trail
SAFBDG	South African Food Based Dietary Guidelines
SAHDS	South African Demographic and Health Survey
SANHANES-1	The South African National Health and Nutrition Examination Survey
SBP	Systolic blood pressure
SD	Standard deviation
SE	Standard error
SFA	Saturated fatty acids
SSB	Sugar-sweetened beverages
TE	Total energy
TC	Total cholesterol
THUSA	Transition and health during urbanisation of South Africans
TIA	Transient ischaemic attack
tPA	Tissue plasminogen activator
VCAM	Vascular cell adhesion molecule
VLDL	Very low-density lipoproteins
WC	Waist circumference
WHO	World Health Organisation

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CHAPTER 1 INTRODUCTION

1.1 Background and motivation

In their most recent data on non-communicable diseases (NCDs), the World Health Organisation (WHO) reported that in 2016, 71% of the 56,9 million deaths globally could be attributed to NCDs. NCDs include cancers, diabetes, cardiovascular disease (CVD) and lung diseases. The WHO reported that the majority of all NCD-related deaths (85%) occur in low- and middle-income developing countries, (WHO, 2018d) of which South Africa is one.

Of the total NCD deaths in 2016, an alarming 44% have been due to CVD (WHO, 2018e), making it the leading cause of death worldwide. In 2015 this figure was 37% (WHO, 2017a), translating into a disturbing estimated 19% increase in only one year. Owing to genetic factors (to be discussed in more detail in chapter 2), black Africans have previously been described as genetically protected against CVD (Ubbink *et al.*, 1995), resulting in a low prevalence of CVD in this part of the population (Seftel, 1978). Over the last few decades this genetic advantage has shown signs of fading as a result of an increase in obesity, smoking habits, hypertension and hyperfibrinogenaemia during the different stages of urbanisation (Ayeni *et al.*, 2019; Koegelenberg *et al.*, 2013; NDoH *et al.*, 2017; Shisana *et al.*, 2014; Vorster, 2002).

Several risk factors can trigger NCDs (and consequently CVDs), of which some are avertible (Benziger *et al.*, 2016); these include hypertension, diabetes, physical inactivity, overweight and obesity. As with any global health problem, countries around the globe should try to ascertain the underlying causes for this global epidemic through sound research and should then act by developing guidelines with the purpose of addressing the problem at hand by establishing measures for both prevention and cure in order to reduce the prevalence of NCD- (and by implication, CVD-) related deaths. In a prospective study by Hulsege *et al.* (2016), it was shown that persons who maintained four to five healthy lifestyle factors (diet, smoking, physical activity, favourable change in body mass index (BMI) and alcohol consumption) were 2.5 times less likely to develop CVDs than those adhering to only one or none of the healthy lifestyle factors. The effect of dietary fat consumption (and in particular the type of fat consumed) on CVD risk has been investigated extensively (Chen *et al.*, 2016; Guasch-Ferré *et al.*, 2019; Ho *et al.*, 2020; Hooper *et al.*, 2015; Hooper *et al.*, 2012; Li *et al.*, 2015; Zong *et al.*, 2016). Findings from prospective studies on the subject consistently indicate that replacing saturated fat in the diet with poly-unsaturated fat, mono-unsaturated fat or carbohydrates is associated with lower CVD risk, incidence or mortality (Chen *et al.*, 2016; Guasch-Ferré *et al.*, 2019; Ho *et al.*, 2020; Li *et al.*, 2015; Zong *et al.*, 2016).

One major contributor to the NCD crisis in the developing world is the nutrition transition. The nutrition transition can be defined as the phenomenon occurring when the diets of persons in the developing world change from healthy, low calorie, traditional low-fat diets to Westernised diets high in fat and energy. At the same time, these people in transition also tend to become physically less active. These changes happen as a result of demographic, economic and epidemiological changes and are associated with an increased rate in the incidence of NCDs and NCD-related deaths worldwide (Bourne *et al.*, 2002; Pisa *et al.*, 2012; Ronto *et al.*, 2018).

Popkin (2002a) identified five distinct stages through which countries progress when undergoing the nutrition transition. First there is the hunter-gatherer stage, when communities are physically active and are consuming traditional prudent diets high in complex carbohydrates and low in fat; this is followed by periods of food shortage resulting in lower dietary variation – the famine stage. During the third stage, the receding famine stage, income increases and famine decreases. Dietary intake patterns are now increasingly switching from traditional to Westernised diets and communities enter the degenerative disease stage (stage 4), where dietary intake changes lead to diseases of lifestyle. In the final stage, the behavioural change stage, communities change their dietary intake habits and physical activity levels in order to be healthier, resulting in improved health.

Previously, various researchers established that South Africa is undergoing a rapid nutrition transition (Bourne *et al.*, 2002; MacIntyre *et al.*, 2012; Steyn *et al.*, 2005; Vorster, 2002). At that time it was also determined, that urban South Africans were in the third stage of the nutrition transition (characterised by an increase in the consumption of animal protein, fruit and vegetables and a drop in staple cereal intake), while their rural counterparts were still in stage 1 (the hunter-gatherer stage) (MacIntyre *et al.*, 2002; Popkin, 2002a). There were also indications of some urban groups progressing to stage four (the degenerative disease phase) (Abrahams *et al.*, 2011; MacIntyre *et al.*, 2002)

NCD prevention and treatment has a common agenda with the Sustainable Development Goals (to be achieved by 2030) as set by the general assembly of the United Nations in 2015. The third goal in this collection of 17 goals aims to ensure good health and well-being for people of all ages (SDG, 2015). As part of this particular goal, NCDs are recognised as a key challenge for sustainable development and the aim is to reduce premature mortality as a result of NCDs by one third.

Prospective nutrition data (cohort studies) assist in the development of evidence-based dietary guidelines; however these should be revisited on a regular basis in the light of the current situation to ensure relevance.

The study presented in this dissertation is a sub-study of the South African leg of the multinational Prospective Urban and Rural Epidemiological (PURE) study (PURE-SA-NWP). PURE is an international epidemiological prospective (cohort) study, coordinated by the Population Health Research Institute in Ontario, Canada. It is intended to track the progression of chronic diseases of lifestyle in urban and rural subjects. The study is being conducted in 27 countries/territories in various stages of economic development. Data for PURE-SA-NWP were collected from two communities within the North West Province of South Africa. Baseline data were collected in 2005, with two follow-up collections conducted in 2010 and 2015. The South African leg was conducted in two areas within the North West Province, one urban and one rural.

1.2 Title of the PhD thesis

Changes in dietary intakes and cardiovascular disease risk factors in black South Africans from 2005 to 2015: The PURE-SA-NW study

1.3 Problem statement and purpose of the study

From studies concluded in the early 2000s, we know that the nutrition transition has been taking place at a rapid pace in rural and urban South African groups (Bourne *et al.*, 2002; MacIntyre *et al.*, 2012; Nyepi *et al.*, 2015; Steyn *et al.*, 2005; Vorster, 2002). South African groups have been observed to be in different stages of the nutrition transition, underlining the importance of regular follow-ups and updates of the progression of the nutrition transition to adjust the dietary recommendations to appropriately and effectively combat NCD, and, by implication, also CVD.

Such an update has not been made in recent years and there is thus a need to assess the more recent dietary changes in South African rural and urban groups in order to get a clear picture of where these groups are in terms of the nutrition transition, as well as the pace at which dietary changes are taking place. This will be measured in the present study by assessing the changes in energy intake, micronutrient and macronutrient intake, macronutrient distribution and energy density in rural and urban participants.

Dietary fat intake (and more specifically the type of fat consumed) has consistently been shown to be associated with CVD risk and mortality (Chen *et al.*, 2016; Guasch-Ferré *et al.*, 2019; Ho *et al.*, 2020; Hooper *et al.*, 2015; Hooper *et al.*, 2012; Li *et al.*, 2015; Zong *et al.*, 2016). Few of the existing studies were done in developing countries and the association between the consumption of different types of fat and CVD risk has not yet been investigated in sub-Saharan Africans. Consequently, limited data regarding dietary fat intake and CVD risk in sub-Saharan Africans are available for the formulation of global recommendations for the prevention of CVD and other NCDs. Global recommendations regarding dietary intakes thus far could not take the contextual

drivers of African groups into account, simply because data on the effect of the nutrition transition and risk factors for CVD amongst African groups have not been available. The current study will aim to fill this gap by investigating the associations between the changes in different types of fat (saturated fat, MUFA and PUFA) and the changes in CVD risk factors (waist circumference, body mass index (BMI), total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein HDL, plasma glucose levels, systolic blood pressure and diastolic blood pressure).

1.4 Purpose statement

This study will serve as an in-depth update on the rapid progression of the nutrition transition in the urban and rural groups residing in the North West Province of South Africa. Through investigating the progression of the nutrition transition, dietary recommendations can be streamlined to be in line with the current stage of the nutrition transition of the groups. The progress will be investigated through various methodologies centred around the changes in dietary intake over time.

Given the fact that there is limited research investigating the association of the changes in the intake in different types of dietary fat with the CVD risk of sub-Saharan Africans, with no prospective data on the subject in this region, this study will be the first of its kind in South Africa. It will contribute to the formulation of dietary recommendations regarding CVD, specifically aimed at the black South African population and also the broader sub-Saharan African population.

1.5 Objectives and hypotheses

These are the **objectives** of the thesis followed by the **hypothesis** to be tested for each.

1. To investigate the changes in energy-, micronutrient and macronutrient intakes as well as the macronutrient distribution and energy density of rural and urban black South Africans residing in the North West province from 2005 to 2010 (PURE-NWP-SA study).

Hypothesis: Statistically significant changes in the nutrient intakes of rural and urban black South Africans residing in the North West province, between 2005 and 2010, are observed.

2. To investigate the association between the changes in dietary fat intake (total fat, saturated fat, poly-unsaturated fat, mono-unsaturated fat) and the changes in cardiovascular disease risk factors (waist circumference, BMI, TC, LDL, HDL, plasma glucose levels, systolic blood pressure and diastolic blood pressure) in rural and urban black South Africans residing in the North West province, from 2005–2010 (PURE-NWP-SA study).

Hypothesis: *The changes in the cardiovascular risk factors of rural and urban groups of PURE-NWP-SA are associated with the changes in their dietary fat intake.*

3. To measure the trends in energy-, micronutrient and macronutrient intakes as well as the macronutrient distribution and energy density of rural and urban black South Africans residing in the North West Province from 2005 to 2015 (PURE-NWP-SA study) and report these changes as follow-up longitudinal evidence.

Hypothesis: Statistically significant trends in the nutrient intakes of rural and urban black South-Africans residing in the North West province between 2005 and 2015 are observed.

Based on the information from early years and the research question, aims and objectives of this study, the conceptual framework in Figure 1-1 was compiled.

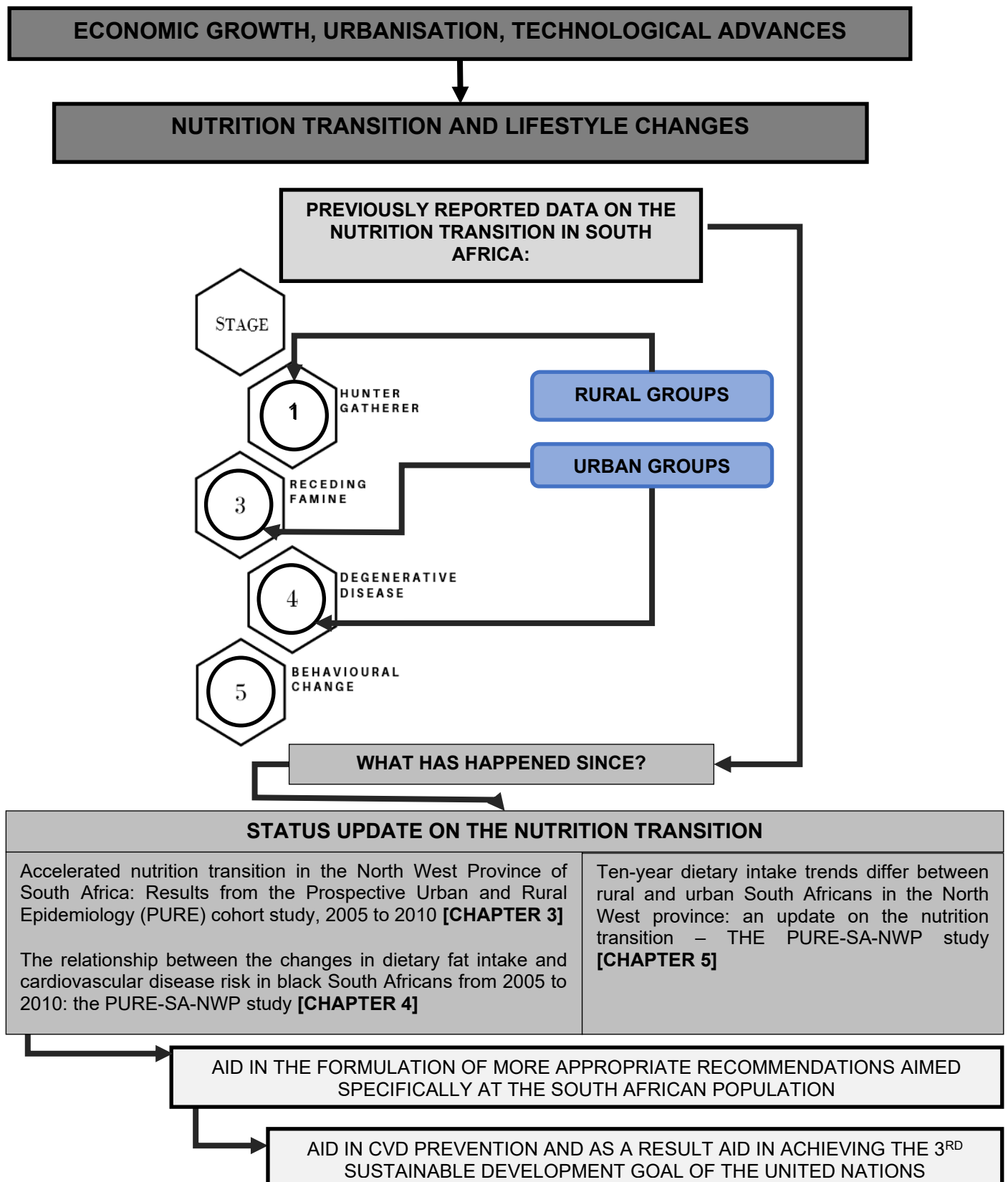


Figure 1-1: Conceptual framework of this study.

1.6 Methods and procedures

1.6.1 Participant recruitment and follow-up

Participant recruitment for the PURE-SA-NWP study is discussed in the respective articles presented in this thesis. A brief summary of participant recruitment follows.

After baseline data collection in 2005, the cohort was closed, and consisted of 2010 participants (urban=1006 and rural=1004), with a full data set. A total of 722 individuals were lost during follow-up in 2010. In addition, subjects with unreliable dietary data based on their reported energy intakes were excluded (energy < 3000 kJ/day and > 30 000 kJ/day). For Chapters 3 and 4 only participants with complete dietary data for both 2005 and 2010 were included in the analysis, leading to a total number of participants of 1154 (rural=388 and urban=766). For Chapter 5, only participants with data for 2005, 2010 and 2015 were included in the analysis, resulting in a total of 722 participants (urban=207 and rural=515).

1.6.2 Questionnaires

Standardised and semi-structured questionnaires of the international PURE study were used in the South African leg, including demographic, socioeconomic, lifestyle, and physical activity questionnaires (Corsi *et al.*, 2013). All the administered questionnaires are discussed in the method sections of Chapters 3, 4 and 5. Dietary data were collected using a 145-item culturally sensitive quantified food frequency questionnaire (QFFQ) that recorded foods eaten over the previous 30 days. This was developed and validated in this population group and it has demonstrated good reproducibility (MacIntyre *et al.*, 2001a; MacIntyre *et al.*, 2001b; Wentzel-Viljoen *et al.*, 2011). A photograph book, specifically developed for use in African groups in the North West Province was used in order to estimate portion sizes (Venter *et al.*, 2000). The portion sizes were reported using household measures, whereafter standard tables were used to convert it to weights (Langenhoven, 1991). Coding of the food intake was done according to the South African Food Composition Database System of the South African Medical Research Council. Hereafter it was analysed to obtain the energy and nutrient intake of the participants (Wentzel-Viljoen *et al.*, 2011).

1.6.3 Statistical analysis

Herewith follows a brief summary of the statistical analysis conducted according to articles in this thesis. The statistical analysis for each article is discussed at length in Chapters 3, 4 and 5.

Article 1 (Chapter 3): Wilcoxon's two-sample test was used to test for differences between the rural and urban participants (by gender) in terms of their nutrient intakes, as well as the changes

in nutrient intakes between 2005 and 2010. Wilcoxon's signed-rank test was used to test for changes in nutrient intakes between 2005 and 2010, for each of the four groups (by gender and study site).

Article 2 (Chapter 4): Stepwise regression models were constructed to test for associations between the change in fat intake and the change in cardiovascular risk factors.

Article 3 (Chapter 5): A linear mixed-model approach was used to assess the trends in energy and nutrient intake changes of urban and rural men and women over time (2005-2010-2015).

1.7 Structure of the thesis

This thesis is in article format. This introductory chapter is followed by Chapter 2, providing an overview of the relevant literature. Chapter 2 focusses on the following topics: the nutrition transition, non-communicable diseases, cardiovascular disease, the risk factors associated with cardiovascular disease, nutrition epidemiology and dietary methodology and the role of the diet in the prevention and treatment of cardiovascular disease. The information in this chapter lays the foundation for the interpretation of the data presented in the articles.

Chapter 3 includes the first article, titled: "Accelerated nutrition transition in the North West Province of South Africa: Results from the Prospective Urban and Rural Epidemiology (PURE) cohort study, 2005 to 2010". The purpose is to investigate the changes in dietary intake of black South Africans in the Prospective Urban and Rural Epidemiological (PURE) study from 2005 to 2010. This article was published in the journal Public Health Nutrition: "Wentzel-Viljoen E, Lee S, Laubscher R, Vorster H.H. Accelerated nutrition transition in the North West Province of South Africa: results from the Prospective Urban and Rural Epidemiology (PURE-NWP-SA) cohort study, 2005 to 2010. Public Health Nutrition. 2018;21(14):2630-41."

Chapter 4 includes the second article, titled: "The relationship between the dietary fat intake and cardiovascular disease risk in black South Africans: The PURE study". This article aims to investigate the association between the change in dietary fat intake (total fat, saturated fat, poly-unsaturated fat, mono-unsaturated fat) of black South Africans, from 2005–2010, and the change in the following cardiovascular disease risk factors: TC, LDL, HDL, plasma glucose levels, systolic blood pressure and diastolic blood pressure. This article was submitted to The Cardiovascular Journal of Africa on 12 November 2020 and is currently under review.

Chapter 5 is an article with the title: "Ten-year dietary intake trends differ between rural and urban South Africans in the North West province: an update on the nutrition transition – The PURE-SA-NWP STUDY". This article aims to investigate the trends in the energy and nutrient intakes of

black South Africans between 2005 and 2015. This article will be submitted to Public Health Nutrition.

In the concluding chapter, Chapter 6, the results are discussed, followed by conclusions and final recommendations concerning the research topic.

With regard to the references, it is essential to note the following: The references of Chapters 1, 2 and 6 were done following the mandatory referencing style of the North-West University and appear as a combined reference list at the end of the document. The references of the article chapters, Chapters 3, 4 and 5, were done following the prescribed referencing style of the respective journals to which these articles were submitted. The references of Addendum A were also done following the mandatory referencing style of the North-West University and appears at the end of the Addendum.

1.8 The research team and author contribution

Table 1-1: The research team for this PhD

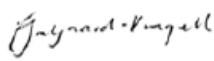
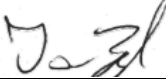
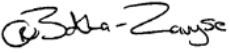
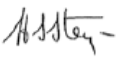
Name and signature*	Affiliation	Role in the study
Mrs S Lee (PhD Candidate)	CEN in the School for Physiology, Nutrition and Consumer Science of the NWU	PhD student Responsible for the planning and conceptualisation of this PhD. Assisted in training the fieldworkers prior to 2015 data collection. Supervising the 2015 dietary data collection. Responsible for the coding and cleaning of the dietary data of 2010 and 2015. Contributions to the different articles are highlighted in a separate table.
Dr C Taljaard-Krugell (Supervisor)	CEN in the School for Physiology, Nutrition and Consumer Science of the NWU	Supervisor of S. Lee Dr Taljaard-Krugell was involved in the planning and conceptualisation of this PhD thesis. Critically revised the manuscript for important intellectual content. Contribution to the different articles are highlighted in a separate table.
Dr C Botha-Ravyse (Co-supervisor)	CEN in the School for Physiology, Nutrition and Consumer Science of the NWU	Co-supervisor of S. Lee Dr Botha-Ravyse was involved in the planning and conceptualisation of this PhD thesis. Critically revised the manuscript for important intellectual content. Contributions to the different articles are highlighted in a separate table.
Dr T van Zyl (Co-supervisor)	CEN in the School for Physiology, Nutrition and Consumer Science of the NWU	Co-supervisor of S. Lee Dr van Zyl was involved in the planning and conceptualisation of this PhD thesis. Critically revised the manuscript for important intellectual content. Contributions to the different articles are highlighted in a separate table.

Table 1-2: Authors contributions to the articles of this PhD

Article	Title	Author contribution
Article 1	Accelerated nutrition transition in the North West Province of South Africa: results from the Prospective Urban and Rural Epidemiology (PURE) cohort study, 2005 to 2010.	E. Wentzel-Viljoen and H. Vorster: Responsible for the conception and design, analysis and interpretation of the data and writing of the manuscript. S.J. Lee: Made contributions to the conception, design, analysis and interpretation of the data and critically revised the manuscript for important intellectual content. R. Laubscher: Was responsible for the statistical analysis of the data and made contributions to the interpretation of the data and critically revised the manuscript for important intellectual content.
Article 2	The relationship between dietary fat intake and cardiovascular disease risk in black South Africans: The PURE-NWP- study.	S. Lee: Responsible for the conception and design, analysis and interpretation of the data and writing of the manuscript. C. Taljaard-Krugell, C. Botha-Ravyse and T. van Zyl: Made contributions to the design, analysis and interpretation of the data and critically revised the manuscript for important intellectual content. E. Wentzel-Viljoen, L. Kruger: Made contributions to the interpretation of the data and critically revised the manuscript for important intellectual content. F. Steyn: Was responsible for the statistical analysis of the data and made contributions to the interpretation of the data.
Article 3	Ten-year dietary intake trends differ between rural and urban South Africans in the North West province: an update on the nutrition transition – The PURE-SA-NWP STUDY	S. Lee: Responsible for the conception and design, analysis and interpretation of the data and writing of the manuscript. C. Taljaard-Krugell: Made contributions to the conception and design, analysis and interpretation of the data and critically revised the manuscript for important intellectual content. C. Botha-Ravyse and T. van Zyl: Made contributions to the design, interpretation of the

Article	Title	Author contribution
		data and critically revised the manuscript for important intellectual content. E. Wentzel-Viljoen: Contributed to the interpretation of the data and critically revised the manuscript for important intellectual content.

Table 1-3: Signatures of all contributors mentioned in Tables 1-1 and 1-2

Name of contributor	Signature
Mrs. Sarie Lee	
Dr Christine Taljaard-Krugell	
Dr Tertia van Zyl	
Dr Chrisna Botha-Ravyse	
Prof. Lanthe Kruger	
Prof. Edelweiss Wentzel-Viljoen	
Prof. Faans Steyn	
Mrs. Ria Laubscher	 Ria Laubscher Digitally signed by Ria Laubscher DN: cn=Ria Laubscher, o=University of Pretoria, ou=Faculty of Health Sciences, email=ria.laubscher@up.ac.za, c=ZA Date: 2019.11.26 08:55:04 +02'00'

CHAPTER 2 LITERATURE REVIEW

2.1 Introduction

In this chapter, the relevant and updated research related to the nutrition transition and its effect on specific health outcomes on the black South African population will be presented. Furthermore, this literature review will shed light on the dietary intake changes of the black South African population, giving an indication of where this population is in terms of the nutrition transition. The five stages of the nutrition transition as explained by Popkin and Gordon-Larsen (2004) will also be discussed. This information will be followed by the latest evidence indicating that the nutrition transition and subsequent changes in the diets of the individuals in transition lead to the increased risk and prevalence of non-communicable disease (NCD) globally, but more specifically, in the South African context. Cardiovascular disease (CVD) as an NCD and its risk factors will be discussed in depth. Each of these topics will be discussed as a separate section of the literature review.

2.2 The nutrition transition

The demographics of developing countries are shifting noticeably from rural to urban areas (MacIntyre *et al.*, 2002; Peters *et al.*, 2019). As a result of demographic and economic changes, the dietary and physical activity patterns of communities have changed from traditional patterns (where persons tend to be more active and consume a prudent (low-fat, low-energy) diet) to patterns typically associated with urban societies (where physical activity tends to be lower and Westernised diets high in energy, fat, sodium and refined carbohydrates are consumed) (MacIntyre *et al.*, 2002; Popkin, 2002a; Ronto *et al.*, 2018). These changes in dietary patterns and nutrient intakes are referred to as the “*nutrition transition*”. The nutrition transition typically occurs during times of rapid urbanisation, and acculturation, as well as social and economic progression. Simultaneously, an increased prevalence of NCDs in developing countries undergoing the nutrition transition has been observed (Vorster, 2002).

The nutrition transition takes place in five distinctive phases, with most developing countries being between the third and fifth phases (Popkin, 2002a; Popkin, 2002b; Popkin & Gordon-Larsen, 2004; Vorster *et al.*, 2011). Popkin (2002a) describes the five stages as follows: The first stage is the hunter-gatherer stage where the population typically consumes food high in complex carbohydrates (and thus fibre) and low in fat (particularly saturated fat). In addition to this prudent diet the population is highly active. The second stage is the famine stage where variety in the diet shrinks with periods of food scarcity. Although the types of physical activity may change, the activity levels in this stage are still high.

The third, fourth and fifth stages are illustrated in detail in Figure 2-1. In short, the third stage is characterised by an increased income and a subsequent decrease in famine. Then follows the fourth stage, where the diet and physical activity patterns change to such an extent that new diseases and increased disability start to emerge. In the final stage, the fifth stage, behavioural change, in terms of food intake and physical activity, leads to a reduction of the negative effects of the previous stages, resulting in “successful ageing” and a decrease in nutrition-related NCDs (Popkin, 2002a).

Although dietary changes are not a new concept, Popkin and Gordon-Larsen (2004) state that the modern dietary changes occur very rapidly and in the early stages of the social and economic development of developing countries, when one would not yet be expecting it. These changes are particularly rapid with regard to increases in dietary intake of fat, caloric sweeteners (added sugars in the form of sucrose and all other monosaccharides and disaccharides in honey, syrups and fruit concentrates), and animal-source foods (Popkin, 2002b; Popkin & Du, 2003). Consequently, developing countries suffer from the global burden of obesity and other NCDs, originally associated with affluent societies (Peters *et al.*, 2019; Popkin & Gordon-Larsen, 2004; Ronto *et al.*, 2018; Vorster *et al.*, 2011).

Since 2002, researchers have reported that South Africa is undergoing an extremely rapid nutrition transition (Bourne *et al.*, 2002; MacIntyre *et al.*, 2012; Steyn *et al.*, 2005; Vorster, 2002). In isolation, a nutrition transition already poses immense challenges to the public health sector of any given country, but the uniqueness of the South African context adds to the complexity faced by this population in a rapid lifestyle and nutrition transition. Bradshaw *et al.* (2003) have described South Africa’s problem as a quadruple burden of disease. This quadruple burden means that the country is facing a mixture of NCDs, pre-transitional illnesses (e.g. poverty-related malnutrition), injuries and the human immunodeficiency virus (HIV). Based on the mortality trends, the second South African National Burden of Disease study (1997–2012) confirmed the persistence of South Africa’s quadruple burden of disease. A recent review on post-apartheid health care in South Africa also still recognised its existence (Maphumulo & Bhengu, 2019).

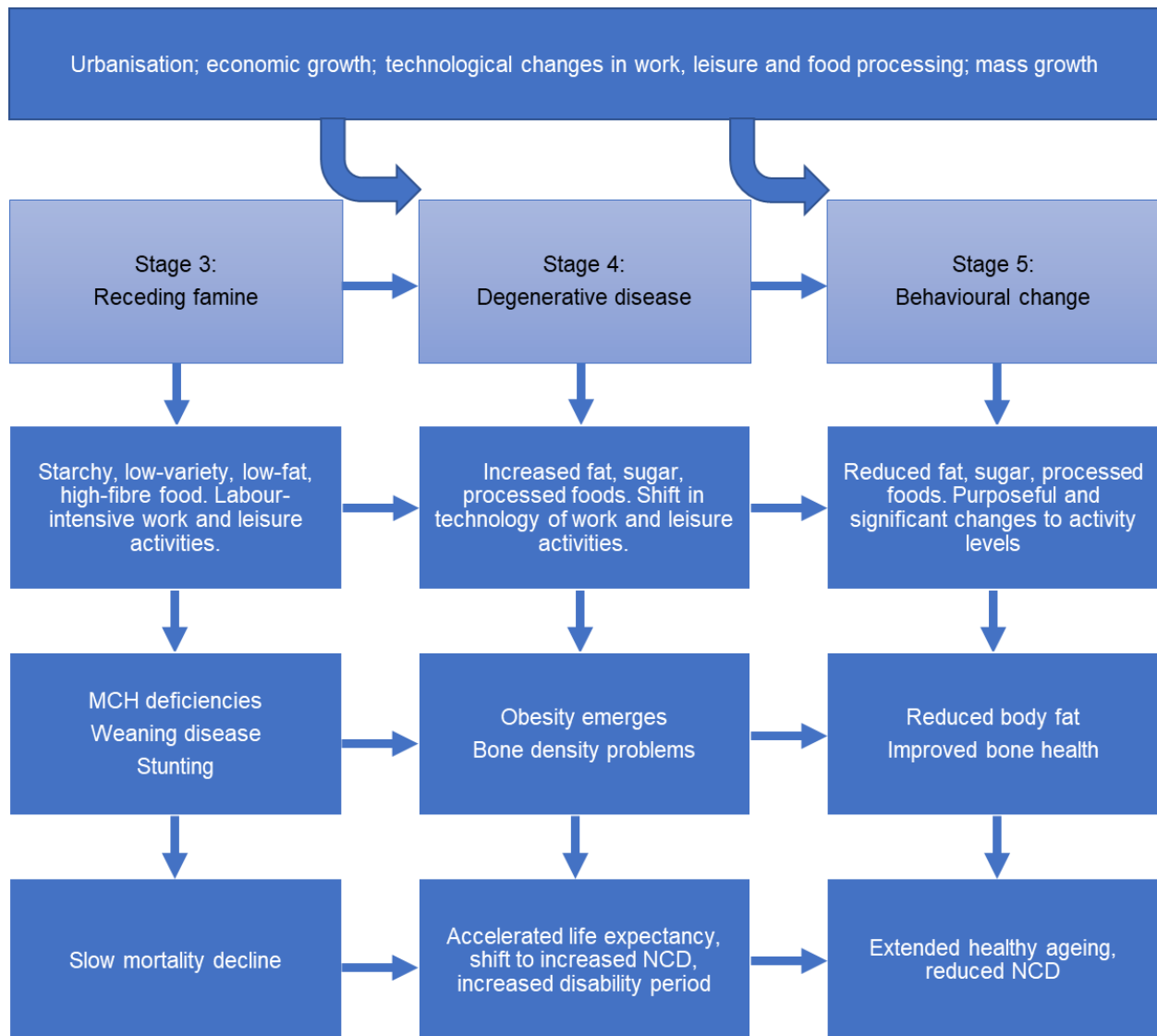


Figure 2-1: A schematic representation of the different stages of the nutrition transition

Adapted from (Popkin, 2002a)

NCD, non-communicable disease; MCH, mean corpuscular haemoglobin

Vorster (2002) reported that, as a result of the rapid migration of black South Africans, there was a decrease in the number of rural inhabitants and a subsequent increase in the number of urban inhabitants. Furthermore, Pisa *et al.* (2012) have reported that urbanisation in the North West province of South Africa sped up dramatically from 1996 to 2005. This was characterised by large numbers of people migrating from rural to urban areas, an increase in the number of industrial companies, and enhanced economic activity. This observation is supported by data from the World Bank, indicating that the percentage of South Africans residing in rural areas decreased from 53% in 1960 to 48% in 1990, resulting in a decrease of 5 percentage points over 30 years.

It then decreased by another 10 percentage points over the next 23 years to 38% in 2013. In 2016 the rural population was measured at 35%. It is clear that the shift from rural to urban areas is a current trend in South Africa, and is happening faster as time passes (Worldbank, 2017).

Amid economic and epidemiological changes, the minds of affected people are preoccupied with their social instability and lack of sufficient income and thus healthy eating tends to not be a priority (Bourne *et al.*, 2002). This may be aggravated by the lack of knowledge of the consequences of poor nutrition (Bourne *et al.*, 2002). During urbanisation, populations which used to consume prudent (low-fat, low-energy) traditional diets, have changed their dietary and nutrient intakes to Westernised, imprudent (high-fat, high-energy) diets, including large quantities of convenience and fast foods. This change in the nutritional intake of communities is often also coupled with a decrease in energy expenditure through higher physical inactivity (Bourne *et al.*, 2002; Vorster, 2002; Vorster *et al.*, 2011; Vorster *et al.*, 2005). In a review by Bourne *et al.* (2002), existing data regarding the changes in the diets of black South Africans over several years were examined. The earliest urban data date back to 1940, while data for rural groups have been available only since 1969. Initial data indicated that rural as well as urban groups showed increased proportional intakes of dietary fat and decreased carbohydrate proportion, over 30–60 years. They concluded that the combined evidence from the data indicated that, in both the urban and rural groups, the diets have shifted from a traditional diet to a more Westernised diet. In a review of the dietary intake of adult South Africans from 2000–2015, Mchiza *et al.* (2015) reported that dietary data in South Africa were limited and that energy and nutrient intakes, according to the available studies, ranged from low in rural areas to very high in urban areas.

In a cross-sectional study including 1751 respondents (743 males and 1008 females), aged between 15 and 80 years in the North West province of South Africa, observations regarding the nutrition transition were noted (MacIntyre *et al.*, 2002). It was reported that the bulk of the population found themselves in the same stage of the nutrition transition, namely the receding famine stage (third stage). This stage is typically characterised by an increase in the consumption of animal protein, fruit and vegetables and a drop in staple cereal intake. Although their cereal intake was still high, higher intakes of animal protein and fat were observed. In addition, a portion of the population, mainly those living in rural areas, were still in the hunter-gatherer stage of the nutrition transition (first stage), which may be accompanied by famine as the pattern is characterised by long periods of food insecurity. Those respondents living on farms and rural settlements had low micronutrient intakes, leading the researchers to conclude that they may still be in stage 2 of the nutrition transition. As previously stated, the receding famine stage of the nutrition transition is followed by the degenerative phase (stage 4), characterised by increases in fat, sugar and processed food consumption. When looking at the data of the upper-class urban

group, it would seem that some were starting to move from the third to the fourth stage as they had higher fat and refined starch intakes (MacIntyre *et al.*, 2002). Abrahams *et al.* (2011) have developed a six-point score to determine the stage in which 40 Sub-Saharan African countries were in terms of the nutrition transition. South Africa, Ghana, Gabon, Cape Verde and Senegal have been reported to show the highest nutrition transition scores. These countries were found to show signs of groups well into the fourth stage of the nutrition transition, where dietary changes start to affect health outcomes. The changes in demographic areas and the ongoing nutrition transition are associated with the growing rate of NCDs not only globally, but also in South Africa (Bourne *et al.*, 2002; Pisa *et al.*, 2012).

The traditional diet of black South Africans used to be low in fat and sugar and high in fibre (Akinboboye *et al.*, 2003). More recently, the diet has been characterised by greater amounts of refined grains, animal products, fats, sugar and salt, while fibre intake is lower (Sliwa *et al.*, 2008). Using data from the Food and Agriculture Organisation, Ronquest-Ross *et al.* (2015) reported that, in a comparison of the food intake of South Africans from 1994 to 2009, the following could be noted: increases in energy intake, the intake of sugar-sweetened beverages and caloric sweeteners, the proportion of processed and packaged foods and animal-derived foods, while the intake of vegetables decreased. Furthermore, South Africans were observed to consume 18 kg more meat per capita per year in 2009 than in 1994. The South African National Health and Nutrition Examination Survey (SANHANES-1) reported fat and sugar consumption in terms of calculated fat and sugar scores based on the use of fat and sugar in the diet. The South African population as a whole was reported to have a medium consumption of fat. In the rural groups' fat consumption was low, while it was high in urban areas. Sugar intake was moderate for the total population as well as for rural and urban groups, respectively. Four or more fruits were consumed by 4,6% of adults, while most participants (52,2%) consumed between one and three fruits per day (Shisana *et al.*, 2014). Diets may have changed since the time when this was previously reported; however, no more recent research is available to shed light on the current situation. Hopefully, this study will be able to remedy that.

2.3 Non-communicable diseases: Overview over the last few decades

Non-communicable diseases include metabolic diseases such as obesity, diabetes and CVD, as well as cancer and chronic respiratory diseases (Dalal *et al.*, 2011). Taking the lives of 41 million people worldwide and being responsible for 71% of all deaths annually, NCDs are without question the primary cause of death worldwide (WHO, 2018d). NCDs have been described as a rising epidemic as they affect more and more people each year. Results from the most recent Global Burden of Disease report (of 2017) indicated that from 2007 to 2017 the NCD-related

deaths increased by 22,7 percentage points, representing a staggering 7,61 million additional deaths (Roth *et al.*, 2018).

The development of more than 75% of all newly diagnosed NCDs can be attributed to one or more of the following modifiable risk factors: the consumption of a Westernised diet high in saturated fats, sugar and salt; sedentary lifestyles; over-consumption of alcohol; and smoking (Wagner & Brath, 2012; WHO, 2002). According to the World Health Organisation (WHO), the consumption of a healthy diet, cessation of smoking and being regularly active may prevent as much as 40% of cancer occurrence, and no less than 80% of all instances of stroke, type 2 diabetes and heart disease (WHO, 2002; WHO, 2018c).

NCDs are often regarded as being diseases exclusively impacting the lives of the wealthy (Alwan, 2011; Benziger *et al.*, 2016; Dalal *et al.*, 2011; Mathers *et al.*, 2008). The WHO has, however, reported that the majority of all NCD-related deaths (85%) occur in low- and middle-income developing countries, including South Africa (WHO, 2018d). This increase in NCD-related deaths is due to an increase in sedentary lifestyles and the adoption of a Westernised diet in developed populations (Alwan, 2011; Benziger *et al.*, 2016; Mathers *et al.*, 2008). In many low- and middle-income countries, including Sub-Saharan Africa, an epidemiological transition from a situation where communicable diseases are the primary cause of death to one where NCDs are the primary cause of death, is well underway (Benziger *et al.*, 2016; Dalal *et al.*, 2011). For instance, in 2017, NCDs were reported to be the second leading cause of death in Sub-Saharan Africa (Yuyun *et al.*, 2020). Statistics-SA's latest report (the 2017 report, published on the 20th of March 2020) on the causes of death in South Africa, indicated that, in 2007, communicable disease was the leading cause of death (responsible for 46,9% of deaths), followed by NCDs (44.1% of deaths). However, in the 10 years from 2007 to 2017 the number of NCD-related deaths in South Africa increased by 13,7 percentage points, making NCDs the leading cause of death (57,8%), while communicable disease accounted for only 30,7% of deaths (Statistics-SA, 2020). This shift can in part be attributed to the nutrition transition and accompanying factors, but it is important to acknowledge that this was not the only independent variable. South Africa's response to the HIV epidemic gained traction in the early 2000's when anti-retroviral therapy (ART) was made freely available through the public health sector (Simelela & Venter, 2014), and this would undoubtedly have played a major role in the decline of communicable disease deaths.

2.4 Cardiovascular disease

CVDs are defined by the WHO as a group of disorders related to the heart and blood vessels, and include: cerebrovascular disease (stroke), coronary heart disease (CHD), rheumatic heart disease, deep vein thrombosis and pulmonary embolism, as well as ischaemic heart disease

(IHD) (WHO, 2017a). The European guidelines on CVD prevention in clinical practice (2012) describe CVD as a chronic illness that progresses stealthily during the course of an individual's life and has usually already developed to an advanced stage by the time it becomes symptomatic and is diagnosed (Perk *et al.*, 2012).

As with other NCDs, the prevalence of CVD is on the increase in developing countries (WHO, 2018d). This can be credited to urbanisation and the subsequent nutrition transition (Vorster *et al.*, 2011; Vorster *et al.*, 2005; WHO, 2018d).

The fight against CVD is not a new one. In 1969 the Executive Board of the WHO described CHD as "Mankind's greatest epidemic" (WHO, 1969) and issued the following warning: "Mankind's greatest epidemic: CHD has reached enormous proportions striking more and more at younger subjects. It will result in coming years in the greatest epidemic humanity has faced, unless we can reverse the trend by concentrated research into its cause and prevention" (WHO, 1974).

The WHO reports that in 2016, 83% of the 17 million premature deaths due to NCDs (of persons younger than 70 years of age), occurred in low- to middle-income countries. Of these deaths in low- to middle-income countries, 37% were due to CVDs (WHO, 2017a; WHO, 2018e). The two most common CVDs are cerebrovascular disease (stroke) and CHD (WHO, 2017a; WHO, 2018e). According to data from the most recent Global Burden of Disease Study, cerebrovascular disease and CHD ranked as the two most common causes of death in low- to middle-income countries (figure 2-2) (Naghavi *et al.*, 2017). According to the WHO's estimations, CVDs were responsible for 17,9 million global deaths in 2016, which accounted for 31% of all deaths globally (WHO, 2017a). A total of 9,4 million of these deaths could be attributed to CHD, while 5,7 million were due to cerebrovascular disease (WHO, 2018e).

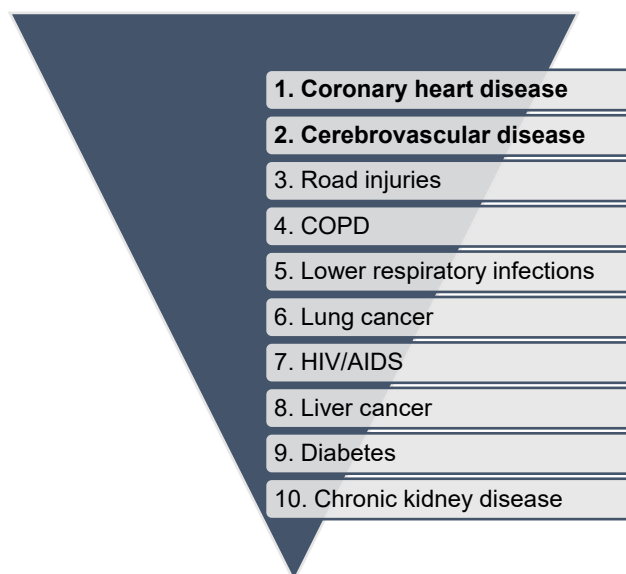


**Figure 2-2: The ten most common causes of death in low-middle income countries
Global Burden of Disease Study 2016 (Naghavi et al., 2017)**

COPD, chronic obstructive pulmonary disease; HIV, human immunodeficiency syndrome; AIDS, acquired immunodeficiency syndrome

In 2016, cerebrovascular disease and CHD accounted for 26.8% of all deaths worldwide (WHO, 2018e). The bulk of deaths due to CHD occur in low- to middle-income countries (80%) (Perk *et al.*, 2012). This increase in the prevalence of CVDs has also been reported in black South Africans, a group which previously had a very low prevalence of CVDs (Akinboboye *et al.*, 2003; Seftel, 1978).

Ubbink *et al.* (1995) reported that black Africans have genetically determined low homocysteine levels, which may genetically protect them against CVDs. However, the Transition and health during urbanisation of South Africans (THUSA) study established that even though total cholesterol (TC) levels of other population groups in South Africa were higher than those of urban black South Africans, the TC levels of urban black South Africans were significantly higher than those of their rural equivalents (Vorster, 2002). The same study confirmed that stroke is a critical public health problem in the black South African group. This is mainly because of an increase in obesity, smoking habits, hypertension and hyperfibrinogenaemia during the different stages of urbanisation (Vorster, 2002). Vorster (2002) suggested that it could be that the more black South Africans come into contact with Westernised lifestyles and privileged circumstances for prolonged periods of time, the more the prevalence and severity of obesity and dyslipidaemia may escalate. Consequently, the protective genetic predisposition of the black South African group (Ubbink *et al.*, 1995) may no longer be effective.

Cerebrovascular disease and CHD will now be discussed in more depth.

2.4.1 Cerebrovascular disease (stroke)

Being ranked as the second most common cause of death worldwide, as well as in low-to-middle income countries, and the third most common cause of decreased disability-adjusted life-years (DALYs) (Lozano *et al.*, 2013; Murray *et al.*, 2012; WHO, 2018e), cerebrovascular disease (stroke) is clearly a public health concern worldwide.

Cerebrovascular disease refers to a group of disorders (including transient ischaemic attacks, ischaemic stroke and haemorrhagic stroke) occurring when there are abnormalities in the blood flow to the brain (Warlow, 1998). These abnormalities may be caused by modifiable risk factors such as lipid profile abnormalities (arteriosclerosis), high blood pressure and obesity (Warlow, 1998).

The onset of stroke is typically sudden; it may continue for more than 24 hours, and may result in death (Warlow, 1998). Symptoms may include the following: a) Loss of balance or dizziness, coupled with difficulty in walking; b) having trouble speaking or understanding others and/or confusion; c) severe headache; d) having difficulty in seeing well, in one or both eyes; and e) loss of feeling (numbness or weakness) in one or more body parts, such as the face, arms or legs, frequently affecting only one side of the body. Survivors of stroke often require abiding rehabilitation and care. Although some survivors may have a full recovery, many individuals are left with severe life-long disabilities, seriously affecting their quality of life (Warlow, 1998).

Inadequate dietary intake is a crucial contributing factor in the development of this cerebrovascular disease. In a meta-analysis including 20 prospective studies (682 149 participants), Chen *et al.* (2019) found that persons adhering to the Mediterranean diet (characterised by a high intake of plant proteins, whole grains, fish, and mono-unsaturated fat; a low intake of refined grains, sweets and red meat; and a modest alcohol intake) had a 16% lower risk of suffering from a stroke. This finding is also supported by a review article of 25 studies, which concluded that adherence to the prudent Mediterranean diet is associated with a lower risk of stroke incidence (Saulle *et al.*, 2019). In another meta-analysis, including 25 studies and more than two million participants, it was shown that overweight and obese individuals had a 22% and 64% respectively progressively increasing risk of developing cerebrovascular disease (Strazzullo *et al.*, 2010).

Feigin *et al.* (2009) conducted a systematic review of population-based studies of the incidence and early-case fatality of stroke (21 days to one month), using data from studies published from 1970 to 2008. Statistically significant findings with regard to the trends of stroke incidence over the past 40 years in both high-income and middle- to low-income countries have indicated

increased stroke incidence. The trend reported for the two income groups differed significantly and, for the first time, the incidence level of stroke in low- and middle-income countries has exceeded that of high-income countries by 20%. Over the past 40 years, the stroke incidence in high-income countries has decreased by 42%, while it has increased by more than 100% in low- to middle-income countries.

According to Statistics-SA's latest report (Statistics-SA, 2020) released in March 2020, cerebrovascular disease was the third most prevalent natural cause of death in South Africa (second most prevalent cause of death among females). According to this report it also accounted for 5% of all natural deaths and 5,1% of natural deaths in the black population. For people older than 65 years of age, cerebrovascular disease is the most prevalent natural cause of death, responsible for 8,5% of all natural deaths in this age group.

2.4.2 Coronary/Ischaemic heart disease

CHD, often also referred to as ischaemic heart disease (IHD) or coronary artery disease (CAD), occurs when the supply of oxygenated blood to the myocardium (muscular wall of the heart) due to atherosclerosis and later, arteriosclerosis of the coronary arteries, becomes insufficient (Ambrose & Singh, 2015; Jinnouchi *et al.*, 2020). As soon as the supply of oxygen to the myocardium cannot meet the demand, the myocardium becomes ischaemic. At this point, the cardiac pumping becomes dysfunctional, leading to abnormal heart rhythms. The severity of the ischaemic episode may vary, but in cases where it is prolonged or severe, it may result in irreparable damage to the myocardial cells, resulting in myocardial infarction (heart attack) (Ambrose & Singh, 2015; Copstead & Banasik, 2013).

Atherosclerosis occurs when fats, cholesterol, and other elements build up in the artery walls to form plaques. This leads to the restriction of blood flow and thus, of oxygen to the myocardium. (Gadgil *et al.*, 2014; Rocha & Libby, 2009). Figure 2-3 illustrates the interaction between the various risk factors associated with IHD.

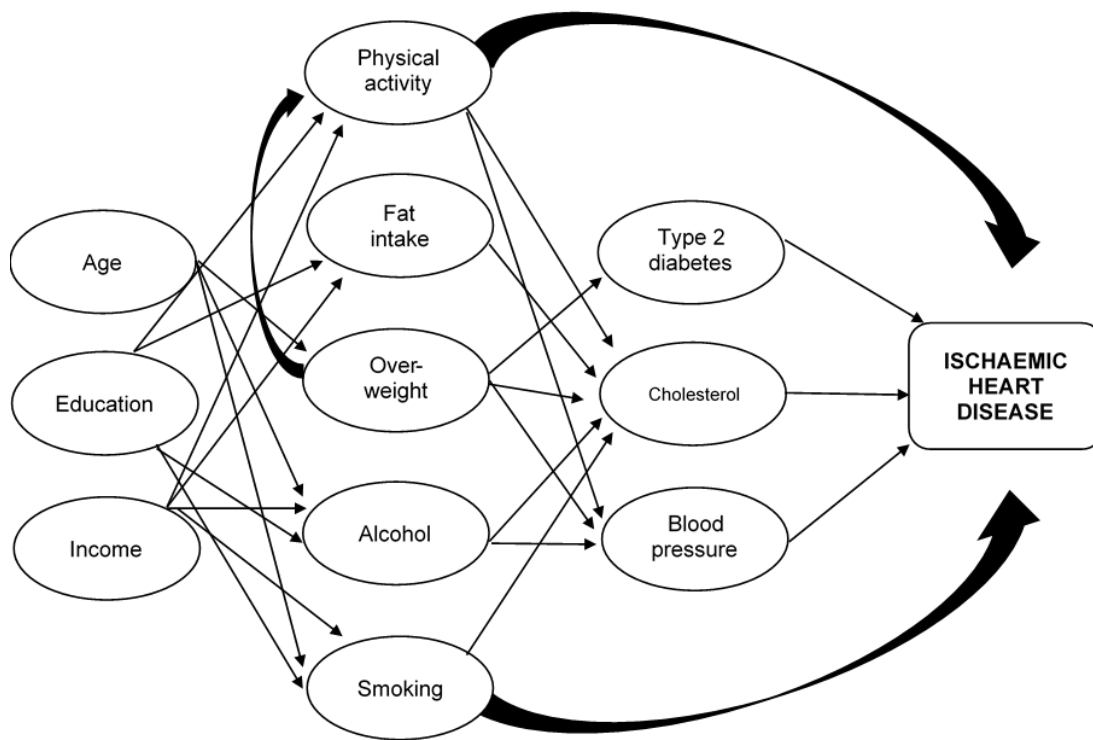


Figure 2-3: The causal chain: Significant causes of ischaemic heart disease
Taken from (WHO, 2009)

For many decades, ischaemic heart disease was not regarded as particularly prevalent in Sub-Saharan Africa, especially in the black population (Brock & Gordon, 1959). Owing to Westernisation and the subsequent nutrition transition, this picture has changed drastically over the last few decades (Vorster, 2002). The increase in sedentary lifestyles and the adoption of a Westernised diet in developed groups as a result of the nutrition transition lead to an increase in obesity, hypertension, hyperfibrinogenaemia and smoking habits, thus increasing their CVD risk (Alwan, 2011; Mathers *et al.*, 2008; Peters *et al.*, 2019; Ronto *et al.*, 2018; Vorster, 2002; Weerahewa *et al.*, 2020). Statistics-SA reports that, in 2017, CHD was the ninth leading cause of all natural deaths, being responsible for 2,9% of deaths. For adults above 65 years of age it was reported to be the fifth leading cause of all natural deaths (4.8%) (Statistics-SA, 2020).

2.5 Risk factors of cardiovascular disease

Numerous risk factors can contribute to the development of CVDs, of which some are avoidable, including: hypertension (global prevalence of 19%), diabetes (8,5%), physical inactivity (25%), overweight (39%) and obesity (13%) (Benziger *et al.*, 2016; WHO, 2018a; WHO, 2018f; WHO, 2018g; WHO, 2019) (Benziger *et al.*, 2016). Data from the 2017 Global Burden of Disease Study showed that most CVD deaths could be attributed to high systolic blood pressure, unhealthy diet, high fasting plasma glucose, and high low-density lipoprotein (LDL) levels (Jagannathan *et al.*,

2019). In the South African population, the CVD risk factors are present in all sectors of the population, be it to a lesser or greater extent. According to the most recent South African Demographic and Health Survey (SADHS), 23,5% of all South Africans 15 years and older are overweight, 45% have raised blood pressure, 34% have elevated cholesterol levels, 26% are obese, and 10,5% are diabetic (NDoH *et al.*, 2017).

According to the results of SANHANES-1, 19,8% of black Africans had elevated blood pressure, 20,6% had elevated cholesterol levels, 16,9% had an HbA1c level of more than 6.1 mmol/mol (the cut-off point for diagnosing diabetes) and 46,7% were overweight or obese (Shisana *et al.*, 2014).

Each one of these risk factors, on its own or in combination with one or more other risk factors, has inevitable biological consequences that may cause an individual to be more prone to the risk of developing cardiovascular disease (Mathers *et al.*, 2008; Mayosi *et al.*, 2009; Schutte *et al.*, 2017; Yusuf *et al.*, 2020). In the sections to follow, all these risk factors will be explained in depth together with their degree of preventability.

2.5.1 Hypertension

According to the latest South African hypertension practice guideline (Seedat *et al.*, 2014), hypertension is diagnosed when the blood pressure (BP) is elevated $\geq 140/90$ mmHg. BP is considered as optimal if the value is $< 130/85$ mmHg. Individuals with a diastolic BP level from 130–139 mmHg and a systolic BP of 85–89 mmHg are classified as pre-hypertensive and have a higher CVD risk but are not yet required to receive drug treatment (Seedat *et al.*, 2014). The risk of death from CHD increases by 7% with every 2 mmHg increase in systolic blood pressure. With the same increase in systolic blood pressure there is also a 10% increase in the risk of death from stroke (Krause *et al.*, 2011).

The WHO regards hypertension as a key risk factor for ischaemic stroke, coronary heart disease and haemorrhagic stroke. Hypertension is a major public health problem worldwide. According to the WHO, 1,13 billion people in the world have hypertension (WHO, 2019). More than 66% of these are people from low- to middle-income countries. The WHO further reports that the highest prevalence of hypertension is in the WHO African region, with 27% of the population being hypertensive. The 2012 SANHANES-1 by Shisana *et al.* (2014) reported 26% of South Africans to be hypertensive, while the 2016 SADHS reported a prevalence of 44% among men and 46% among women (NDoH *et al.*, 2017). The SANHANES-1 further reports that a total of 9,9% of black participants were classified as having pre-hypertension with a BP of 120–139/80–89 mmHg. However, when examining the systolic and diastolic values independent of each other, the figures

differed. The prevalence of pre-hypertensive systolic blood pressures and pre-hypertensive diastolic blood pressures were 38,2% and 18,8%, respectively. As with the pre-hypertensive figures, the picture also changes somewhat when looking at the systolic and diastolic values independent of each other. A total of 23,2% and 12,2% had hypertensive systolic and diastolic blood pressures, respectively. These data are presented in Table 2-1.

Chronic hypertension has a whole array of potential effects on different organs in the body (Copstead & Banasik, 2013). Figure 2-4 gives a schematic representation of these potential effects. According to the 2017 Global Burden of Disease Study, hypertension was found to be the leading risk factor for early death in females and the second leading risk factor for males (IHME, 2018). Statistics-SA reported hypertension to be the sixth leading cause of all natural deaths (4,5%) in South Africa and the third leading cause of all natural deaths in the female group (5,8%) (Statistics-SA, 2020).

Table 2-1: Percentage prevalence of pre-hypertension and hypertension among different groups of participants in SANHANES-1, 2012 (mmHg)

Group	N	Pre-hypertensive blood pressure Pre-hypertension			Hypertensive blood pressure Hypertension		
		SBP 120–139 mmHg	DBP 80–89 mmHg	Pre- hypertensio n 120- 139/80–89	SBP ≥ 140 mmHg	DBP ≥ 90 mmHg	Hypertension BP > 140/90mmHg
All races	7029	38,2	20,0	10,4	24,6	12,2	10,2
Black population	4826	38,2	18,8	9,9	23,4	12,2	9,9
Informal urban	805	38,1	16,9	9,6	15,9	10,2	7,8
Informal rural	1860	37,5	17,2	8,4	23,4	10,2	8,4

SBP, systolic blood pressure; DBP, diastolic blood pressure; BP, blood pressure
Adapted from (Shisana *et al.*, 2014)

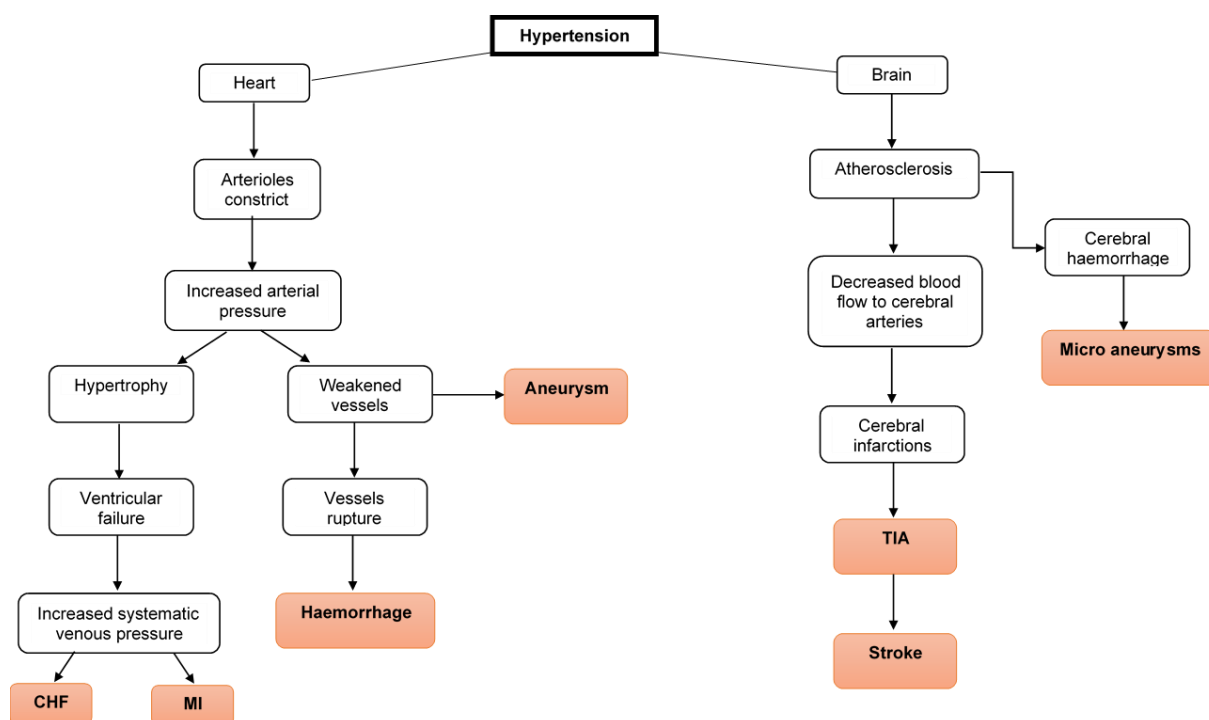


Figure 2-4: The potential cardiovascular effects of chronic hypertension on significant target organs

CHF, Congestive heart failure; MI, myocardial infarction, TIA, transient ischaemic attack.

Adapted from Copstead and Banasik (2013)

In a South African study using data from the 2017 National Income Dynamics Study (n=23 605), the factors contributing to hypertension prevalence were found to be age, body mass index (BMI), alcohol intake and locality (rural vs urban) (Wandai *et al.*, 2020). Nutrition plays an

essential role in the prevention and management of hypertension. Sodium intake, and therefore salt intake, is a crucial factor in the dietary approach to the management and prevention of hypertension. In studies done in collaboration with Sasko Milling and Baking and UNILEVER, the amount of sodium reduction that was possible in the production of various products, without affecting texture or taste, was determined (Charlton, 2007; Charlton *et al.*, 2007). A group of South African researchers have used these proposed reductions to show that a decrease in the sodium content of powdered soup, seasoning, margarine and bread, would lead to a decrease in sodium intake of 0,85g/person/day (Bertram *et al.*, 2012). According to their impact analysis, such a decrease would lead to 7 400 fewer deaths due to CVD and 4 300 fewer non-fatal stroke cases per year. The cost savings amount to R300 million per year, not taking the home-based cost savings into account. This approach to a reduction in salt intake is also supported by the Food-Based Dietary Guidelines of South Africa (SAFBDG) (Wentzel-Viljoen *et al.*, 2013).

In addition, there are other approaches, such as the “Dietary Approaches to Stop Hypertension” (DASH) diet, which is a dietary plan intended to regulate the blood pressure levels of hypertensive patients (Appel *et al.*, 1997; Bhupathiraju & Tucker, 2011). This pattern includes plenty of vegetables, fruits, and dairy products; furthermore, the diet also includes poultry, fish and grains. Foods such as red meat, saturated fat, sugar-containing drinks and sweets, as well as foods with a high salt content are limited (Appel *et al.*, 1997; Bhupathiraju & Tucker, 2011). The effectiveness of the DASH diet in lowering blood pressure levels has been confirmed by numerous studies, including various robust meta-analyses conducted on the subject (Filippou *et al.*, 2020; Salehi-Abargouei *et al.*, 2013; Schwingshackl *et al.*, 2019; Soltani *et al.*, 2020). A meta-analysis on 30 randomised-controlled trials by Filippou *et al.* (2020) found the DASH diet to be effective in lowering blood pressure, even in those participants who did not suffer from hypertension. In a meta-analysis, including six studies on the effects of the DASH diet on the incidence of CVDs, it was shown that participants following a DASH-like diet had a significant reduction in their risk of developing CVD, CHD, stroke and heart failure, by 20%, 21%, 19% and 29%, respectively (Salehi-Abargouei *et al.*, 2013). These findings were confirmed by a recent meta-analysis by Soltani *et al.* (2020) including 17 cohort studies. This study found even the slightest adherence to the DASH diet to be linked to a lower risk of all-cause mortality as well as CVD mortality. This effect escalated with an increase in adherence to the DASH diet.

In the South African hypertension guideline (Seedat *et al.*, 2014), the South African Department of Health states that a healthy lifestyle is the foundation of the management and prevention of hypertension. As an attempt to promote a healthy lifestyle, the Department of Health included six lifestyle guidelines to reduce/prevent high blood pressure:

- (i) Having or achieving an ideal body weight with a BMI from 18,5 and 24,9 kg/m².

- (ii) Sodium intake should be limited to 2400 mg per day, resulting in the consumption of less than a teaspoon of salt per day.
- (iii) Alcohol intake should be limited to two standard drinks per day for men and one for women (Vorster *et al.*, 2013).
- (iv) Emphasis should be placed on following a diet with plenty of fruits and vegetables (5 a day), regular use of low-fat dairy products, increased intake of wholegrain foods, reduction in the total fat of meals, fish or poultry rather than red meat, consumption of foods low in saturated fat, salt and sugar. This is also supported by the SAFBDG (Vorster *et al.*, 2013).
- (v) Moderate-intensity exercise lasting for at least 30 minutes at a time should be engaged in regularly, preferably on most days of the week.
- (vi) Cessation of smoking.

2.5.2 Overweight and obesity

A person that is overweight or obese has an excessive accumulation of abdominal adipose tissue and subcutaneous fat. In a person with healthy body weight, the adipose tissue is considered passive, and its primary function is energy storage, while in the overweight and especially the obese person, it becomes an active tissue in regulating physiological and pathological processes (Lee *et al.*, 2013).

To classify an individual as overweight or obese, a calculation based on the height and weight of the person, called the BMI, is made. This calculation was formerly called the Quetelet Index. It is calculated by dividing a person's weight by his/her height squared (WHO, 2006).

$$BMI = \frac{\text{kilograms}}{\text{meters}^2}$$

For most adults, except in the case of athletes and the elderly, BMI is a dependable indicator of body fat (Rigby, 2006). BMI is regarded as the easiest and most suitable indicator by which a person's weight to height ratio can be correlated with certain health outcomes. Furthermore, as Rigby (2006) states, a small increase in the BMI of a person may lead to higher disease risk, starting as low as 21 kg/m². The WHO classifies BMI as follows: < 18,5 kg/m² as underweight; 18,5 – 24,9 kg/m² as normal weight; 25,0 – 29,9 kg/m² as overweight; 30,0 – 34,9 kg/m² as obese class I; 35,0 – 39,9 kg/m² as obese class II; and ≥ 40.0 kg/m² as obese class III (WHO, 2006).

It is important to note that what is generally regarded as a healthy BMI (18,5–24,9 kg/m²) may not be regarded as healthy for all individuals from different ethnicities, especially for some individuals

or population groups in developing countries. For example, after reviewing the evidence brought before them, the WHO recommended that the upper range of the standard classification for normal weight be modified to 23 kg/m² for Asian populations (Rigby, 2006) as Asian populations were more at risk of developing chronic disease with less body fat. In a cross-sectional study among 435 urban black women in the North-West province of South Africa, it was shown that significant misclassification of individuals in terms of BMI had occurred (Kruger *et al.*, 2017). This study concluded that it may be necessary to redefine BMI categories for South African black women, but that more research was needed. Presently however, no unique recommendations exist for black Africans, and thus the general standard is used.

The prevalence of overweight and obesity has nearly tripled since 1975. Globally in 2016, 39% of adults 18 years or older were overweight, while 13% were obese (WHO, 2017c). The prevalence of overweight and obesity in South Africa has also been on the rise. In comparing the data from two SADHS (NDoH, 1998; NDoH *et al.*, 2017) the following observations are made: the percentage of women who were overweight or obese increased from 56% in 1998 (NDoH, 1998) to 68% in 2016 (NDoH *et al.*, 2017), while the increase in prevalence among men was less dramatic and increased from 29% to 31%. In 1998, the mean BMI of South African women was 27,3 kg/m² compared to 29,2 kg/m² in 2016. No change was noted in the mean BMI of men.

Various other studies conducted in South Africa reflect the above findings (Ayeni *et al.*, 2019; Kruger *et al.*, 2012a; Prinsloo *et al.*, 2011; Shisana *et al.*, 2014). Data from more than 2500 South African participants presented in the SANHANES-1 have illustrated the following findings: the prevalence of overweight and obesity throughout the whole population across all sexes and races was 20,1% and 10,6% respectively; of the black African sub-group of the study group, 12,9% were overweight, while 9,4% were obese (Shisana *et al.*, 2014). In a cross-sectional study by Kruger *et al.* (2012b) amongst 2403 black women aged 16 to 35 years, it was shown that 26,6% were overweight, while 24,9% were obese. Furthermore, a significantly higher prevalence of obesity was noted in individuals in the urban areas as opposed to their rural counterparts (26,8% vs 22,5%). In another cross-sectional study amongst a total of 304 urban black women in Bloemfontein between the ages of 16 and 50 years, 32,2% were overweight, while 44,1% were obese (Prinsloo *et al.*, 2011). A case-controlled study on breast cancer conducted among 798 black South African women in 2017 has found that 81.2% of the participants in the study were overweight or obese (Ayeni *et al.*, 2019). The evidence stated above underlines the magnitude of the overweight/obesity problem that South Africa is facing.

Overweight and obesity, remain a global pandemic and a significant risk factor for various NCDs, including cardiovascular disease, hypertension, diabetes and hypercholesterolaemia (WHO, 2015c; Zhang *et al.*, 2013). A pooled analysis of ten large US cohort studies by Khan *et al.* (2018)

found BMI to be associated with an increased risk of developing CVD. The 2016 SAHDS found that 42,7% of overweight and 58,4% of obese women in South Africa had hypertension. For men these figures are 59,8% and 67,8% respectively. This is in line with findings in Bangladesh, where women (especially those living in urban areas) were shown to have a significantly higher risk of hypertension when overweight or obese (Bishwajit *et al.*, 2017). In Iranian adults, BMI was also strongly associated with blood pressure, with obese participants being three times as likely to have elevated blood pressure (Kalani *et al.*, 2015). This association has also been documented by various studies in other developing countries (Dua *et al.*, 2014; Hariharan *et al.*, 2019; Hwang *et al.*, 2012; Mishra *et al.*, 2006; Patel *et al.*, 2016; Warsy *et al.*, 2015; Zhang *et al.*, 2011).

A sedentary lifestyle and diet patterns high in energy, fat and sugar and low in fibre, fruits and vegetables are the leading causes of the development of overweight and obesity (Lee *et al.*, 2013). In the South African context, these are both consequences of urbanisation and the subsequent nutrition transition (Vorster *et al.*, 2011; Vorster *et al.*, 2005).

2.5.3 Waist circumference

Abdominal adiposity leads to a spill-over of surplus energy due to the saturation of subcutaneous adipose tissue (Ross *et al.*, 2020). Consequently, in a process called ectopic fat deposition, lean organs and tissue such as the liver, pancreas, skeletal muscles and the heart become fat storage locations, subsequently leading to an increased risk of CVDs (Ross *et al.*, 2020). While BMI is the most widely used diagnostic tool for overweight and obesity, it measures general adiposity and it should be noted that in isolation it comes up short as a biomarker of abdominal adiposity (Gearon *et al.*, 2018; Ross *et al.*, 2020). This is in contrast to waist circumference (WC), which is regarded as a good biomarker of abdominal adiposity (Tran *et al.*, 2018).

In a recent register-based observational cohort study with a mean follow-up of 3.8 years, Mohammadi and colleagues (2020) demonstrated that an increase in WC was associated with an increased CVD risk, independent of BMI. Similar findings were observed in a recent randomised controlled trial with a median follow-up of 9.6 years by Olson *et al.* (2020) who aimed to investigate the effects of long-term weight loss interventions. It was shown that the CVD risk of participants increased with an increase in WC, independent of whether they lost or gained weight. What is highlighted by the findings of these studies is the fact that, if overweight and obesity are determined solely by using BMI, a significant number of persons at risk of CVD may be overlooked in the process. A person with a normal BMI may for instance, when including WC as a diagnostic tool, have to be classified as overweight and may need intervention to reduce CVD risk.

Measuring WC is simple, inexpensive and can be standardised effortlessly (Ross *et al.*, 2020; WHO, 2011b). According to the WHO recommendations, WC is measured by first locating the mid-point between the iliac crest and the rib which is least palpable. Measurement is conducted using a measuring tape resistant to stretch, with a constant 100 g tension, while the person being measured distributes his/her weight equally by standing with feet close together and arms at the side (WHO, 2011b).

The WHO classifies people as being at increased risk of developing NCDs when their WC exceeds 94 cm in the case of men and 80 cm in the case of women (WHO, 2011b), while being at a substantially increased risk when WC exceeds 102 cm and 88 cm for men and women, respectively. While the WHO recognises that different cut-off points may be necessary for different population groups and have implemented this for certain populations, they do not yet have separate evidence-based cut-off points for black South African populations. Owolabi *et al.* (2018) argued that it may indeed be necessary to implement specific cut-off points applicable to black South African populations by showing that the current recommendations may be overestimating the risk of metabolic complications in black South African men and women. The cut-off points suggested by this study were ≥ 95.25 cm for men and ≥ 89.45 cm for women. This is supported by the findings of a USA study among Africans living in the USA, which concluded that cut-off points of > 96 cm for men and > 91 cm for women would be more applicable (Kabakambira *et al.*, 2018).

The SADHS of 2016 reported that 9% of men older than 15 years were classified as being at risk of developing NCDs with a WC of between 94 cm and 102 cm, while 10% had a substantially increased risk, with WC exceeding 102 cm. The prevalence among women was dramatically higher with 19% between 80 cm and 88 cm; and 45% exceeding 88 cm. The mean WC of women increased from 85.8 cm in the 1998 SADHS to 87.4 cm in 2016. According to current WHO recommendations, this puts the South African population at significant risk of developing metabolic complications.

2.5.4 Dyslipidaemia

Several chemical compounds present in food and the body are classified as lipids, of which the most important are triglycerides, phospholipids, and cholesterol (Hall & Guyton, 2006). Along with carbohydrates, and in almost equal quantities, triglycerides are used primarily as a source of energy to fuel the various metabolic processes of the body (Hall & Guyton, 2006). Phospholipids and cholesterol, on the other hand, are used in the formation of all cell membranes. A small number of triglycerides are also used for the latter (Hall & Guyton, 2006). The human body needs cholesterol for the synthesis of hormones as well as for bile production to digest fats. Cholesterol

is produced by the body itself, but can also be found in various animal products (meat, milk, cheese and yoghurt) (Copstead & Banasik, 2013). Dietary intake of cholesterol differs from one person to another, depending on each specific diet and the inclusion of animal products (Copstead & Banasik, 2013).

After cholesterol, triglycerides and phospholipids are absorbed, most of the lipids (95%) remaining in the plasma are in the form of lipoproteins to be transported through the body (Grundy *et al.*, 2004). Lipoproteins are tiny particles, with a similar composition to chylomicrons (cholesterol, phospholipids, triglycerides, and protein), but smaller. There are four main types of lipoproteins: i) very-low-density lipoproteins (VLDL), with high concentrations of triglycerides and moderate phospholipid and cholesterol concentrations; ii) intermediate-density lipoproteins, that are VLDL particles from which some of the triglyceride content has been removed, increasing its concentrations of phospholipids and cholesterol; iii) low-density lipoproteins (LDL), that are intermediate-density lipoproteins with practically no triglyceride content left, leaving them with extremely high concentrations of both phospholipids and cholesterol; iv). high-density lipoproteins (HDL), a lipoprotein vastly different from the other three types as about half its content consists of protein, leaving it with much smaller concentrations of phospholipids and cholesterol (Grundy *et al.*, 2004). HDL and LDL are also functionally different from each other in that LDL transports cholesterol to the peripheral parts of the body, while HDL transports cholesterol away from the heart to the liver (Grundy *et al.*, 2004).

Dyslipidaemia occurs when the cholesterol concentration, in the form of LDL, becomes elevated above normal levels (defined later in this section), placing the individual at risk of developing atherosclerosis and eventually CVDs (Hall & Guyton, 2006). Although genetics may play a role in the development of dyslipidaemia, poor dietary practices are mainly to blame (Copstead & Banasik, 2013; Hall & Guyton, 2006; Imamura *et al.*, 2009; Jakobsen *et al.*, 2009). Dietary patterns high in saturated fats (e.g. diets high in animal products), obesity and a sedentary lifestyle are the most common causes, and to a lesser extent also the consumption of cholesterol itself (Copstead & Banasik, 2013; Hall & Guyton, 2006; Imamura *et al.*, 2009; Jakobsen *et al.*, 2009). Coupled with the increase in LDL, there may also be a decrease in the HDL concentrations of the plasma, which is of concern as HDL protects a person against the development of atherosclerosis by absorbing cholesterol crystals that have started to accumulate on the walls of arteries (Copstead & Banasik, 2013; Hall & Guyton, 2006).

The current recommendations for Europe and the United States for lipid levels are presented in Table 2-2 (Lichtenstein *et al.*, 2006; Perk *et al.*, 2012). The European guideline is used in South Africa (Klug *et al.*, 2013; Van Schoor, 2010). However, in patients where the cardiovascular risk

remains high despite lifestyle changes, it might be advisable to lower the targets to 4.4 mmol/L for total cholesterol and 2.5 mmol/L for LDL cholesterol (Klug *et al.*, 2013; Van Schoor, 2010).

In the most recent Global Burden of Disease Study (of 2017), it was found that 4,3 million (7.7%) of global deaths can be attributed to elevated LDL levels (Mattiuzzi *et al.*, 2020). The same study also reported that the mortality rate in low-middle income countries has increased significantly (by 138%). Norman *et al.* (2007) conducted a study to estimate the burden of disease attributable to an abnormal lipid profile, using data from small community studies in South Africa across all race groups. In the black population, 58,1% of CHD and 41,9% of ischaemic stroke was attributable to an unfavourable lipid profile. Similar findings were noticed across all race groups. This led to the conclusion that an unfavourable lipid profile observed in the South African population should be a reason for concern as it is, without a doubt, a significant risk factor for cardiovascular disease.

The SANHANES-1 (n=5458) has reported the following results with regard to blood lipid levels of South Africans: the results on blood lipid levels indicated that abnormally high serum total cholesterol and LDL cholesterol could be found in one out of every four participants (23.9% and 24.6%, respectively) older than 15 years of age, while almost half (47.9%) of them had an abnormally low serum HDL-cholesterol. (Shisana *et al.*, 2014).

To combat the high incidence of abnormal lipid metabolism in South Africa is particularly challenging. As with other risk factors, management of lipid metabolism requires both medical and lifestyle changes. What separates cholesterol management from other risk factors, in terms of how challenging it is to manage, is the impact of the HIV epidemic on the lipid levels of the South African population. It has repeatedly been shown that antiretroviral agents may intensify or even cause abnormal lipid levels (Belury *et al.*, 2017; Calza *et al.*, 2004; Koutkia & Grinspoon, 2004; Vos *et al.*, 2018).

Table 2-2: The current European and United States guidelines in terms of the lipid profile.

	European guideline	United States guideline
Total cholesterol	Less than 5.0 mmol/l	Less than 240 mg/dl (6.2 mmol/l)
LDL	Less than 3.0 mmol/l	Less than 160 mg/dl (3.8 mmol/l)
HDL	1.0 mmol/l or more in males	40 mg/dl (1 mmol/l) or more
	1.2 mmol/l or more in females	
Triglycerides	Less than 1.7 mmol/l	Less than 150 mg/dl (1.7 mmol/l)
<i>LDL, Low-density lipoprotein; HDL, High-density lipoprotein</i> <i>Compiled using data from Lichtenstein et al. (2006) and Perk et al. (2012)</i>		

The South African guidelines for dyslipidaemia was revised in 2018 (Klug et al., 2018). These guidelines are based on the European guidelines. Klug et al. (2018) recommends the following:

- Total cholesterol < 5 mmol/L
- LDL cholesterol < 3 mmol/L for patients with low CVD risk < 2.6 mmol/L for those with moderate risk and < 1.8 mmol/L for patients with a high risk of CVD.
- HDL cholesterol > 1.2 mmol/L for women and > 1.0 mmol/L for men.
- Triglycerides < 1.7 mmol/L

In 2020 the recommendations in terms of LDL cholesterol were amended (Klug & Raal, 2020) following an amendment to the European guidelines. According to the amendment an LDL goal of < 1.4 mmol/L are recommended for patients with type 2 diabetes mellitus with a very high CVD risk, and < 1.8 mmol/L for patients with a high CVD risk. It is also recommended that the LDL levels of high and very high-risk patients be lowered by at least 50% from baseline.

The European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS) guidelines for the management of dyslipidaemia recommend a fat-modified diet where trans-fat in the diet is completely avoided, saturated fat intake is limited to less than 10% of the total energy intake (or below 7% for persons already diagnosed with hypercholesterolaemia), total fat intake is limited to below 30% (mainly from unsaturated fatty acids) and cholesterol in the diet restricted to below 300 mg/day. These guidelines further recommend diets high in dietary fibre (i.e., diets containing a variety of fruit and vegetables as well as whole grains) because of their hypocholesterolaemic effect, providing 25–40 g of dietary fibre per day (Mach et al., 2020). These guidelines are in line with what is also recommended by the WHO, the FBDGs as well as the South African dyslipidaemia guidelines (Klug et al., 2018; Smuts & Wolmarans, 2013; Vorster et al., 2013; WHO, 2018b).

2.5.5 Blood glucose levels

Type 2 diabetes can be defined as a progressive chronic disease characterised by insulin resistance generally due to obesity and relative insulin deficiency as a result of the progressive deterioration of beta-cell function (Copstead & Banasik, 2013; WHO, 2015a). Type 2 diabetes results from a combination of one or more of the following risk factors: environmental factors, genetic predisposition and behavioural factors, which may include factors such as obesity, prior gestational diabetes, race and ethnicity (Tuomilehto *et al.*, 2001; WHO, 2018a).

Insulin plays an essential role in glucose metabolism in the following target tissues: liver, adipose tissue, and skeletal muscle. In the liver, insulin acts as an inhibitor of the expression of key gluconeogenic enzymes (Lee *et al.*, 2006; Pickersgill *et al.*, 2007; Yu *et al.*, 2002). This means that when the body becomes resistant to insulin, this will lead to an increase in the hepatic production of glucose, consequently leading to increased blood glucose levels. In the adipose tissue, insulin signalling prevents free fatty acids (FFAs) from flowing out of adipocytes by decreasing the hormone-sensitive lipase activity (Lee *et al.*, 2006; Pickersgill *et al.*, 2007; Yu *et al.*, 2002). In the event where insulin signalling is impaired, there will be an increase in the levels of circulating FFAs. Free fatty acids promote increased triglyceride synthesis in the liver, which can lead to the secretion of VLDL and thus dyslipidaemia. By stimulating the translocation of the glucose transporter to the plasma membrane in the skeletal muscle, insulin encourages the uptake of glucose. Should this process be impaired, a decreased glucose clearance from circulation will occur, resulting in increased blood glucose (Lee *et al.*, 2006; Pickersgill *et al.*, 2007; Yu *et al.*, 2002)

Obesity is the driving force behind insulin resistance, promoting the development of CVDs (De Luca & Olefsky, 2008; Rader, 2007). A growing body of evidence suggests that overweight and obesity leads to chronic low-grade inflammation and that inflammation is what leads to insulin resistance (Asghar & Sheikh, 2017; Rocha & Libby, 2009; Saltiel & Olefsky, 2017; Schenk *et al.*, 2008; Wu & Ballantyne, 2020). Figure 2-5 gives a visual representation of the association of obesity with insulin resistance and the eventual development of cardiovascular risk factors and atherosclerosis. The causes and effects of insulin resistance are illustrated in Figure 2-6.

Type 2 diabetes is diagnosed when the blood glucose levels of individuals are routinely tested in a fasting state (Shisana *et al.*, 2014). Routine glucose tests have their shortcomings as they can only be used for short-term diagnostic and monitoring purposes. In order to monitor blood glucose management over a prolonged period of time, a glycated haemoglobin (HbA1c) test is done, the

current gold standard (WHO, 2011a). The WHO has suggested that HbA1c be used as a diagnostic tool to diagnose undiagnosed persons since HbA1c is an indication of an individual's glucose control over the previous 8 to 12 weeks and therefore no fasting is required prior to the test (WHO, 2011a). The recommended cut-off point for HbA1c is 6.5% for the diagnosis of diabetes (WHO, 2011a).

As mentioned above, diabetes is turning into a worldwide epidemic (WHO, 2018a). This becomes especially evident when comparing the number of people suffering from diabetes globally in 1980 in that in 2017: In the 37 years between 1980 and 2017, the number of diabetics have more than tripled from 153 million to 432 million (Danaei *et al.*, 2011; Khan *et al.*, 2019). Not only can diabetes be a killer on its own, but it is also a major risk factor for CVD, increasing the patient's risk of developing a CVD together with his existing diabetes (Danaei *et al.*, 2011; WHO, 2017a). In 2017, type 2 diabetes was ranked as the ninth leading cause of death globally by the Global Burden of Disease Study, taking the lives of more than 1 million people (Khan *et al.*, 2019).

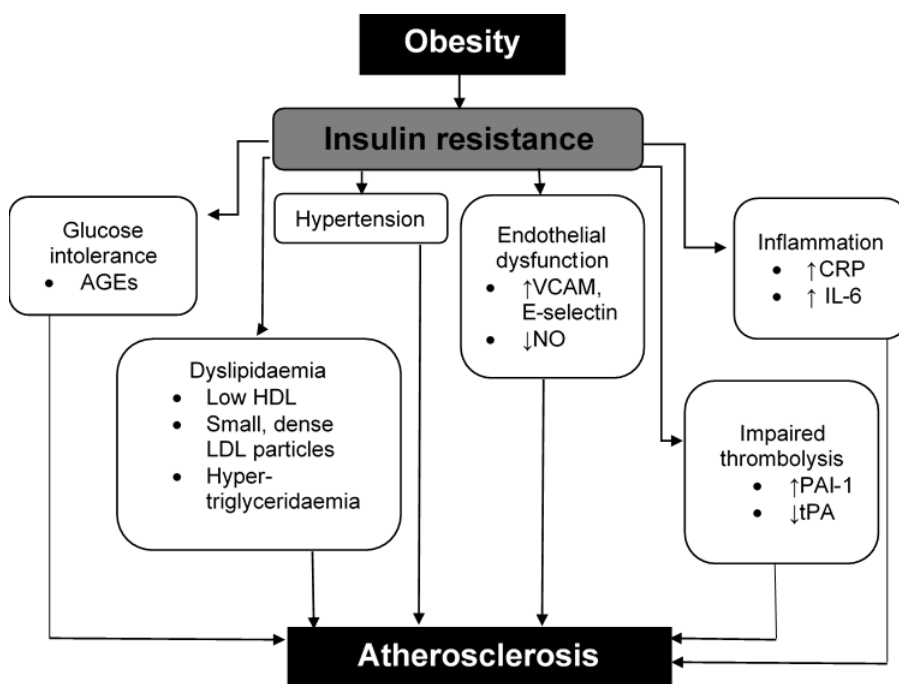


Figure 2-5: Association of insulin resistance with cardiovascular risk factors and atherosclerosis

*CHF, chronic heart failure; AGEs, advanced glycation end products; CRP, C-reactive protein; HDL, high-density lipoprotein; IL-6, interleukin-6; LDL, low-density lipoprotein; NO, nitric oxide; PAI-1, plasminogen activator inhibitor-1; tPA, tissue plasminogen activator; VCAM, vascular cell adhesion molecule. Adapted from McFarlane *et al.* (2001)*

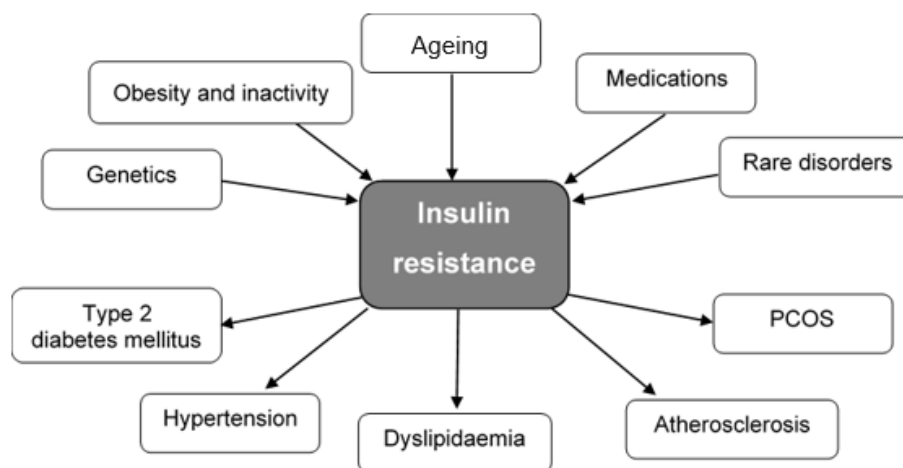


Figure 2-6: Causes and conditions associated with insulin resistance
PCOS, Polycystic ovarian syndrome. Adapted from Rader (2007)

The SADHS of 2016 reported that in the black South African population, 13.1% of women and 7% of men had HbA1c levels of more than 6.5%, classifying them as diabetic. In the case of both black men and women, most of the population (67% and 65%, respectively) were classified as being pre-diabetic (HbA1c of 5.7%–6.4%)

The cardiovascular risk factors have been described in depth in this literature review. The following section will address the role that dietary intake plays in prevention of these risk factors and ultimately the prevention of cardiovascular disease as well.

2.6 The role of the diet in CVD prevention

Even though the relationship between diet and health has been formally studied only for the past few decades, the importance of a nutritious diet has been known for ages (Michels, 2003). This is proven by the sound advice given by the father of Western medicine himself, Hippocrates (460-377 BC): “If we could give every individual the right amount of nourishment and exercise, not too little and not too much, we would have found the safest way to health” (Adams & Brock, 1955).

According to the WHO, sufficient lifestyle changes (e.g. healthy dietary habits, smoking cessation, increased physical activity) may prevent more than 75% of all CVD mortality (Mathers *et al.*, 2008; WHO, 2017a). The risk factors for CVD can be positively influenced if dyslipidaemia (increased LDL cholesterol, triglycerides, TC and low HDL cholesterol levels), hypertension, overweight and obesity are reduced and physical activity is increased (Van Horn *et al.*, 2008; WHO, 2017a).

During urbanisation, the risk factors for CVDs seem to be influenced by the type of diet that is feasible (Vorster, 2002). According to Bourne *et al.* (2002), an increase in socio-economic status does not automatically mean that individuals affected will have an improved nutritional status. On the contrary, these groups are often predisposed to the development of arteriosclerosis because a higher socio-economic status may be linked to unhealthy dietary patterns (Bourne *et al.*, 2002). Strategies should be implemented and managed to promote healthy eating habits and increase physical activity. To successfully implement strategies for the prevention of NCDs, and in particular CVDs, as a consequence of the nutrition transition, we need to fully understand how changes in nutrient intakes influence the preventable risk factors of CVDs.

Many researchers have investigated the effect of different types of fat in the diet instead of reducing overall all-type fat intake as a strategy to reduce NCD and CVD risk (Bender *et al.*, 2014; Hooper *et al.*, 2015; Jiao *et al.*, 2019; Li *et al.*, 2015; Ricci *et al.*, 2018; Sacks *et al.*, 2017). Data from two large independent cohort studies involving US men and women, indicated that the replacement of energy derived from saturated fat sources with sources rich in either mono-unsaturated fatty acids (MUFA) or poly-unsaturated fatty acids (PUFA) or lowered intake of carbohydrates was connected to a decreased risk of CVD (Li *et al.*, 2015). Additional analysis of data from the above-mentioned cohorts confirmed PUFA intake to be associated with lower CVD mortality as well as total mortality in participants with type 2 diabetes (Jiao *et al.*, 2019). This is in line with the findings of a cross-sectional study among US adults by Ricci *et al.* (2018), who found that iso-energetic substitution of saturated fat with MUFA and PUFA reduced all-cause mortality. This topic has also been explored in a Cochrane meta-analysis on 15 randomised controlled trials by Hooper *et al.* (2015), who found that, where dietary saturated fat intake was replaced with PUFA, it was associated with a 27% decrease in cardiovascular events. An earlier meta-analysis by Bender *et al.* (2014) showed that persons consuming fish and n-3 PUFAs (daily dosage varied from 157 mg to 3 360 mg) lost 0,59 kg body weight more than the control groups, while also decreasing waist circumference by 0,81 cm, as opposed to the control groups (Bender *et al.*, 2014). The Presidential Advisory from the American Heart Association was recently led to conclude that the replacement of saturated fat with PUFA lowers the incidence of CVD (Sacks *et al.*, 2017). In light of the strong body of evidence supporting the replacement of saturated fat with healthier types of fat, the South African Food Based Dietary Guidelines (SAFBDGs) recommends that South Africans replace saturated fat in their diet with PUFAs (Smuts & Wolmarans, 2013; Vorster *et al.*, 2013).

Findings from a few related studies that have been carried out in developing countries support the research in developed countries. For example, in Tanzania it was shown that participants who consumed palm oil (rich in saturated fat) had higher mean cholesterol levels compared with those

who consumed vegetable oil (rich in PUFA) (Kakarmath *et al.*, 2017). In Taiwan, a cohort study among 1835 women showed that higher n-6 PUFA consumption was associated with a 48% reduction in CVD risk. Additionally, with sufficient n-6 consumption, 20.7% of CVD events could be prevented (Yang *et al.*, 2019). In a double-blind clinical trial in Singapore, it was reported that PUFA supplementation could improve insulin sensitivity and consequently, insulin resistance. As result a decrease in fasting blood glucose levels may occur (Sarbolouki *et al.*, 2013). This association was also found in a randomised controlled trial conducted among elderly patients in Japan (Ogawa *et al.*, 2013). A prospective Iranian study conducted over a median of 5.8 years (Baseline: 2006-2008; Follow-up: 2012–2014), has found PUFA intake to be associated with a lower risk of type 2 diabetes mellitus (Mirmiran *et al.*, 2018).

Plant-based diets (typically with lower intakes of total fat, saturated fat, and alcohol, and higher intakes of fibre) are another dietary intervention strategy that has received an increasing amount of attention in recent years. In a US prospective study of 12 168 participants (with no CVD at baseline), followed up from 1987 to 2016, greater adherence to healthy plant-based diets were found to be associated with a reduced risk of developing CVD, lower CVD mortality as well as lower all-cause mortality (Kim *et al.*, 2019). This research is supported by Heianza *et al.* (2020), who followed 156 148 British adults free of CVD at baseline in a prospective study from 2006 to 2010, and found that adherence to a healthy plant-based diet was associated with lower CVD incidence regardless of genetic predisposition to CVD.

The Mediterranean diet is a plant-based diet for which a large body of evidence exists to demonstrate its role in lowering CVD risk (Saulle *et al.*, 2019). In recent years various meta-analyses on observational studies, randomised controlled trials as well as prospective studies have been performed on the subject (Asbaghi *et al.*, 2020; Becerra-Tomás *et al.*, 2020; Grosso *et al.*, 2017; Liyanage *et al.*, 2016; Rosato *et al.*, 2019; Shannon *et al.*, 2020). In general, these studies are in agreement that adherence to a Mediterranean diet has a protective effect against the development of CVD and is associated with lower CVD mortality.

A group of South African nutrition experts developed the SAFBDG for South Africans. It was first published in 2003 and revised in 2013. The aim of the SAFBDG is to provide the general population with science-based, easily understandable nutritional messages, referring to food items eaten and known by most of the population (Vorster *et al.*, 2013). These messages are not only looking to decrease or even eradicate starvation, famine and famine-related deaths and nutritional deficiencies, but are also aiming to reduce the NCD burden attributable to nutrition practices. The SAFBDGs consist of ten nutritional messages, each with a solid scientific basis (Vorster *et al.*, 2013), presented in Table 2-3. Since these guidelines have been based on the

WHO guidelines for the prevention of chronic disease, they can be regarded as a good standard of what a healthy diet should look like.

Table 2-3: Revised general food-based dietary guidelines for South Africans, 2013.

1. Enjoy a variety of foods.
2. Be active!
3. Make starchy foods part of most meals.
4. Eat plenty of vegetables and fruit every day.
5. Eat dry beans, split peas, lentils and soya regularly.
6. Have milk, maas or yoghurt every day.
7. Fish, chicken, lean meat or eggs can be eaten daily.
8. Drink lots of clean, safe water.
9. Use fats sparingly. Choose vegetable oils, rather than hard fats.
10. Use sugar and foods and drinks high in sugar sparingly

2.7 Nutrition epidemiology and dietary methodology

Epidemiology is defined in the epidemiological dictionary by Last *et al.* (2001) as follows: “Epidemiology is the study of the distribution and determinants of health-related states or events (including disease) in specified populations, and the application of this study to the control of diseases and other health problems.” It is important to note that epidemiology concerns itself with populations i.e. not single individuals but groups of people (whole communities, entire populations), and this is what sets it apart from clinical medicine (Dos Santos Silva, 1999). Dos Santos Silva (1999) aptly describes it: “Epidemiology is the only source of direct scientific evidence about exposure effects and the preventability of disease within human populations.”

Everything that can be said about epidemiology can also be said about nutrition epidemiology as the latter is but a branch of the bigger science of epidemiology, concerned with studying the diet as a determinant of health-related states with the purpose of controlling health outcomes (Michels, 2003). For the purposes of observational research, diet and physical activity are perhaps the most difficult factors to assess. To a certain extent this is because measurement errors are extremely likely because the measurements are based on the ability of participants to recollect previous meals and levels of activity (Michels, 2001).

Although challenging, nutrition epidemiology is possible with sound dietary methodology. In most instances, except with weighed records where the respondent weighs all the food he/she consumes, dietary methodology relies on the memory of the respondent and, if not executed well enough, may result in an unreliable set of data (Thompson & Subar, 2017).

In this study, quantified food frequency questionnaires (QFFQs) have been used to assess dietary intake. QFFQs have been commonly used to assess dietary intake since the 1990s (Shim *et al.*, 2014). QFFQs are comprehensive questionnaires used to determine habitual dietary intake by asking a person to provide specific and qualitative details regarding their food intake over a specific period (e.g. 30 days). A wide range of food items will be included (100–150 items) and respondents will be asked to provide quantities of food consumed using household measures (Willett, 1998). This method is cost-effective, easy to administer, can be administered in a relatively short period of time (± 30 minutes) and provides detailed information about food intake (Ocke *et al.*, 1997; Rodrigo *et al.*, 2015; Shim *et al.*, 2014). This method has, however, been criticised because of reservations regarding their level of accuracy (Freedman *et al.*, 2006; Schatzkin *et al.*, 2003). In order to ensure the accuracy of a QFFQ, it should be validated within the group where the larger study will take place. After being administered for the purposes of validation, it will be compared to another form of nutrition assessment also administered to the same respondents. It is also desirable for it to be compared to certain biological markers such as urine nitrogen (Coates & Monteilh, 1997; Hankin & Wilkens, 1994; Thompson & Subar, 2017). This will give the researcher an idea of the validity of the method and whether the selected assessment was measuring what it was supposed to. Based on the results, the questionnaire can be adapted to be valid for use in the larger study. Furthermore, all questionnaires should be administered by well-trained fieldworkers in the language with which the respondent feels most comfortable (Thompson & Subar, 2017).

After administering the validated questionnaire in the main study, the data obtained should be converted from food items and quantities to nutritional values with a nutrient composition database containing the foods of the community in question (Coates & Monteilh, 1997; Hankin & Wilkens, 1994; Thompson & Subar, 2017). Only now can the data be used to investigate correlations between dietary intake and health outcomes.

An in-depth explanation of the dietary methodology of the PURE study (international and PURE-SA-NWP) can be found in Addendum A.

2.8 Conclusion

From the evidence presented in this chapter it is clear that South Africa and, in particular, the black population, is in the midst of a nutrition transition due to gradual transition from traditional diets and lifestyles to Westernised diets and lifestyles. This is leading to a definite decline in the health of South Africans. The increasing prevalence of obese and overweight South Africans is also of great concern. It is clear that CVDs (and other NCDs) are on the rise and diseases which the black population was once perceived to be protected against, have been emerging at a

growing rate in recent years. Cardiovascular disease is responsible for more and more fatalities each year, costing the public health sector large amounts of money.

It cannot be denied that South Africa's chronic disease burden needs to be addressed urgently. Each year billions of rands are spent on the treatment and management of nutrition-related chronic diseases, placing an intolerable economic burden on developing countries (Bloom *et al.*, 2012). Furthermore, and more importantly, millions of lives fall victim to chronic disease. As stated in Part IV of the Bellagio Declaration (Popkin, 2002b), the only reasonable way to approach this problem is through prevention.

Prospective nutritional data (Cohort studies) are extremely valuable in the development of evidence-based dietary guidelines. The WHO nutrition Guidance Expert Advisory group (NUGAG) advises the WHO on nutrition guidelines for the prevention of NCDs for developing countries. The recommendations made by the WHO are used in making recommendations to prevent NCDs in South Africa and the FBDGs are also based on WHO guidelines (personal communication with Prof. HH Vorster, April 2015). The WHO Nutrition Guidance Expert Advisory Group (NUGAG) decided that only data from randomised control trials (RCTs) and/or cohort (prospective epidemiological) studies can be used for meta-analyses aimed at developing global evidence-based nutrition guidelines (personal communication with Prof. HH Vorster, February 2015).

Therefore, a thorough investigation into the progression of the nutrition transition and influence of dietary change on the risk factors for CVD using prospective (Cohort) data from the PURE study is an important step in the development of evidence-based dietary guidelines regarding CVD. As far as is known, the PURE study is the only cohort study on nutrition and NCDs in Sub-Saharan Africa.

Even though we are aware of the nutrition transition and its effect on health, it remains an essential public health issue that needs to be continuously measured and monitored to understand the behaviour changes that have taken place with intervention and education initiatives and where these programmes need modification. The success of these will be seen as soon as the nutrition transition has stopped or slowed down and if, regardless of demographic transition, diets remain prudent.

CHAPTER 3 ACCELERATED NUTRITION TRANSITION IN THE NORTH WEST PROVINCE OF SOUTH AFRICA: RESULTS FROM THE PROSPECTIVE URBAN AND RURAL EPIDEMIOLOGY (PURE) COHORT STUDY, 2005 TO 2010



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Accelerated nutrition transition in the North West Province of South Africa: results from the Prospective Urban and Rural Epidemiology (PURE-NWP-SA) cohort study, 2005 to 2010

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Abstract

Objective: South Africa (SA) is in the midst of a health transition characterized by a quadruple burden of diseases and a nutrition transition. The existing nutrition transition in SA, accompanied by the coexistence of under- and overnutrition in the population, motivated the present study. Its objectives were to measure and report the changes in nutrient intakes of rural and urban black Africans over time to assess the impact of urbanization and modernization of lifestyles on dietary intakes and non-communicable disease (NCD) risk.

Design: The PURE-NWP-SA study recruited 2000 black South African volunteers aged 35–70 years in 2005, of which detailed nutrient intakes from 1858 participants were available. In 2010 nutrient intakes of a cohort of 1154 participants were measured.

Results: Median energy intake increased over time. In 2010, rural participants consumed the amount of energy (men 9.7 MJ/d; women 9.1 MJ/d) that urban participants consumed in 2005 (men 9.9 MJ/d; women 9.0 MJ/d). The nutrition transition was characterized by increases in the percentage of energy from animal protein, total fat (rural men and women), saturated (not urban women) and monounsaturated fat, as well as added sugar. Despite the higher energy intake, not all the participants met total micronutrient needs in 2010.

Conclusions: The PURE nutrient intake data confirmed that the nutrition transition in the North West Province of SA is extremely rapid in rural areas. The shift towards higher energy intakes, an animal food-based diet, higher intakes of fat and lower intake of fibre, at the cost of lower plant protein and starchy food intakes, could increase the risk of NCD.

Keywords
South Africa
PURE study
Quantified FFQ
Nutrient intake
Nutrition transition

It is recognized that South Africa (SA), a low-middle-income country, is in the midst of a health transition characterized by a quadruple burden of communicable diseases, non-communicable diseases (NCD), perinatal and maternal and injury-related disorders⁽¹⁾, as well as experiencing a nutrition transition⁽²⁾. Sharp increases in overweight, obesity, hypertension, diabetes and CVD, without substantial improvements in undernutrition among children, have been reported for all population groups^(3–5). The South African Medical Research Council⁽⁶⁾ identified both under- and overnutrition as risk factors for disability-adjusted life years and mortality in the South African population. We have previously shown that the diet followed by black Africans in rural areas of the North West Province (NWP) does not meet micronutrient requirements^(7–10). However, with urbanization and 'modernization' of the diet, increases

(but not optimization) of micronutrient intakes have been observed⁽⁷⁾. Simultaneously, macronutrient intakes have been characterized by increases in animal protein, total fat and especially added sugar^(7,11) while total carbohydrate intake has decreased⁽⁷⁾, despite increased intake of added sugar⁽¹¹⁾. These changes in nutrient intakes have been described since the 1970s⁽²⁾. To address micronutrient deficiencies, the South African Department of Health (Directorate Nutrition) instituted a mandatory micronutrient fortification of the staples, maize meal and bread flour, in 2003⁽¹²⁾ that came into effect in 2004. The objectives of the present study were to measure and report the changes in nutrient intakes of rural and urban black African men and women over time (from 2005 to 2010) to assess the impact of urbanization and modernization of lifestyles on dietary and therefore nutrient intakes and NCD risk.

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Participants and methods

The PURE study

The Prospective Urban and Rural Epidemiology (PURE) study is a prospective cohort study tracking changing lifestyles, risk factors and chronic disease in urban and rural areas of seventeen countries in transition^(13,14). One of the legs of the PURE study in SA is being conducted in two areas (urban and rural) of the NWP (PURE-NWP-SA). The province is experiencing a typical epidemiological transition⁽⁷⁾. The baseline study was conducted in 2005 and the follow-up in 2010.

The design of the PURE-NWP-SA study has been described elsewhere^(11,14,15). Briefly, participants were recruited from four different sites (two rural and two urban) in the NWP of SA. A census profile of 6000 households (1500 in each area) was completed from which 4000 individuals were identified as meeting the inclusion criteria and visited at home. The study was explained to the individuals and after voluntary and informed consent was obtained, the questionnaires were completed. Questionnaires for 3750 participants were completed. These participants were invited to the study sites and after giving informed consent, blood samples were drawn and other measurements taken. The dietary questionnaires were also completed at this stage. In total, 2000 participants were tested (target of 500 in each community) in 2005. In the 5-year follow-up survey conducted in 2010, 1233 participants were included. Figure 1 provides a summary of the recruitment and follow-up procedures (adapted from Vorster *et al.*⁽¹¹⁾).

Measurements

Questionnaires

Standardized structured demographic, socio-economic, lifestyle and physical activity questionnaires of the

international PURE study were used⁽¹³⁾. These questionnaires were adapted for the South African study where necessary. Sixteen volunteer fieldworkers, from the communities where the study was undertaken, were recruited and intensively trained to complete the questionnaires at the study site or participant's home in the language of choice (Setswana, Afrikaans or English). A 145-item, culture-sensitive, quantified FFQ (QFFQ) was used to obtain dietary intakes covering the previous month. The QFFQ was previously developed and extensively validated for this population^(16,17). Reproducibility was tested again for the present study⁽¹⁸⁾. Portion sizes were reported by the participants using a food-portion photograph book⁽¹⁹⁾ specifically developed and tested for this population, as well as other suitable tools as previously described⁽¹⁸⁾. Portion sizes reported in grams (based on the photographic portion book) or household measures were converted to weights using standard tables⁽²⁰⁾. Nutrient intake was calculated using the South African food composition database⁽²¹⁾. Food items not in the database were either analysed or sourced from other databases, added to the database and then coded as previously described⁽¹⁸⁾.

Other measurements

The methodology used for the anthropometric measures (height, weight and waist circumference), blood collection, biochemical analyses (HDL-cholesterol) and HIV testing have been described previously^(11,14).

Statistical analyses

The statistical software package Stata version 14 was used for the analyses. Due to the skewness of the nutrient intake data, results are reported as median and 25th percentile–75th percentile. Wilcoxon's two-sample test was used to test for differences between the rural and urban participants (by gender) in terms of their nutrient intakes, as well as the changes in nutrient intakes between 2005 and 2010. Wilcoxon's signed-rank test used to test for changes in nutrient intakes between 2005 and 2010 for each of the four groups (by gender and study site). Participants with energy intakes $\geq 30\,000$ or ≤ 3000 kJ/d in either 2005 or 2010 were excluded from the data set⁽¹¹⁾.

To evaluate adequacy of intakes as well as compliance to recommendations for the prevention of NCD, reported nutrient intakes were compared with international recommendations⁽²²⁾, WHO recommendations⁽²³⁾ as well as the South African food-based dietary guidelines⁽²⁴⁾, as shown in the online supplementary material, Supplemental Table 1.

Baseline mean characteristics of the cohort in 2005 and of those participants lost to follow-up in 2010 are shown in Table 1.

In addition to intakes reported in their respective units, intakes of macronutrients were also expressed as a percentage of total energy, and intakes of micronutrients and dietary fibre per 4.2 MJ (1000 kcal; see online

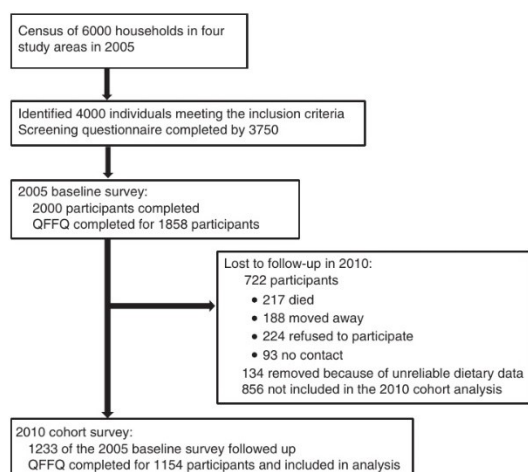


Fig. 1 Participant recruitment and follow-up from years 2005 to 2010 (adapted from Vorster *et al.*⁽¹¹⁾). QFFQ, quantified FFQ

**Table 1** Baseline characteristics in 2005 of participants followed up from 2005 to 2010 (the cohort), compared with subjects lost to follow-up and compared with 2010; PURE-NWP-SA study

Variable	Baseline data in 2005 of participants followed up in 2010			Baseline data in 2005 of subjects lost to follow-up in 2010			P value*	Cohort data of participants in 2010			
	n	Mean	SD	n	Mean	SD		n	Mean	SD	P value†
Age (years)	1154	50.8	10.3	856	48.6	10.4	<0.001	1154	–	–	–
Sex (male; %)	1154	33.6		856	41.8		<0.001	1154	–	–	–
Locality (rural; %)	1154	51.7		856	47.8		0.080	1154	–	–	–
Education attainment (no formal education; %)	1154	36.0		856	32.1		0.073	1154	–	–	–
HIV positive (%)	1154	17.1		856	22.2		0.004	1154	4.9 % new cases		–
BMI (kg/m ²)‡	1154	25.1	7.0	853	24.1	7.0	<0.001	1154	25.5	7.3	<0.001
Waist circumference (cm)‡	1147	80.3	12.8	843	79.0	13.3	0.007	1147	81.9	13.0	<0.001
HDL-cholesterol (mmol/l)‡	1087	1.54	0.61	805	1.48	0.66	0.006	1087	1.42	0.59	<0.001
Energy intake (MJ/d)	n	Median	P25–P75	n	Median	P25–P75	P value*	n	Median	P25–P75	P value†
	1154	7.4	5.6–10.1	796	6.9	4.7–9.8		1154	10.5	7.5–13.9	

PURE-NWP-SA, Prospective Urban and Rural Epidemiology in the North West Province of South Africa; P25, 25th percentile; P75, 75th percentile.

*P value for significance of differences between subjects followed up in 2010 and those lost to follow-up (categorical variables: χ^2 test; continuous variables: two-sample Wilcoxon test).†P value for significance of differences between the cohort in 2005 and 2010 (categorical variables: χ^2 test; continuous variables: two-sample Wilcoxon test).

supplementary material, Supplemental Table 2; Tables 2 and 3). The percentages of participants not reaching 100 % of recommended intakes were also calculated and are shown in Tables 4 and 5.

Results

Comparison of the cohort with those lost from the study and between 2005 and 2010

Dietary intake data of 1858 participants were measured in 2005. Table 1 shows the characteristics (in 2005) of the 1154 cohort participants who could be followed up in 2010 compared with the baseline characteristics of the 856 participants lost to follow-up. The latter were slightly (but significantly) younger, with a higher proportion of men, more HIV-infected participants, with lower BMI, waist circumference, HDL-cholesterol and total energy intake. There were small (but significant) increases in BMI and waist circumference from 2005 to 2010 that could probably be explained by the increase in energy intake and ageing.

Nutrient intakes, macronutrient distribution and micronutrient density of all participants in 2005

Supplemental Table 2 (see online supplementary material) shows that in 2005 the nutrient intakes of urban men and women were consistently higher than those of their rural counterparts.

Changes in nutrient intakes of men from 2005 to 2010

In Table 2, nutrient intakes of the male cohort (186 rural men and 202 urban men) in 2005 are compared with the intakes in 2010. Rural men consumed significantly more energy and therefore significantly more of all nutrients

($P < 0.05$; except Mn) in 2010 than in 2005. However, the percentage of energy contributed by plant protein and the contribution of total carbohydrate to energy intake were lower in 2010 than in 2005, while the energy contributions from total and animal protein, total fat, saturated fat, monounsaturated fat and added sugar were higher in 2010 than in 2005. Energy from alcohol decreased significantly from 2005 to 2010. There were also significant increases in some micronutrient intakes (Fe, niacin and vitamin C), even when these micronutrients were expressed in terms of energy intake to 'correct' for increased energy intakes in 2010.

Urban men also showed a significant increase in total energy intake from 2005 to 2010 ($P < 0.001$) and therefore an increased intake of all other nutrients. A non-significant decrease in the percentage of energy from alcohol was measured. No change in the percentage contribution of total protein to energy intake was observed, but similar to rural men, significant increases in the percentage contributions of animal protein, saturated and monounsaturated fats, as well as added sugar, to energy were seen. The percentage of energy from plant protein and total carbohydrate decreased significantly in rural and urban men from 2005 to 2010. Significant increases in micronutrient density for niacin and vitamin C and significant decreases for fibre and Zn were measured.

Changes in nutrient intakes of women from 2005 to 2010

Table 3 compares the changes in nutrient intakes over time in the female cohort (411 rural women and 355 urban women). Both rural and urban women consumed more energy in 2010 compared with 2005, with the consequence that intakes of all nutrients were higher in 2010 in the respective groups. However, as in the case of men,

Table 2 Daily nutrient intake, energy distribution and nutrient density of the male cohort by rural/urban residence in 2005 and 2010; PURE-NWP-SA study

Energy and nutrients	Rural men (n 186)					Urban men (n 202)					
	2005			2010		2005			2010		
	Median	P25–P75	Median	P25–P75	P value*	Median	P25–P75	Median	P25–P75	P value*	P value†
Energy (MJ)	6.9	5.6–8.8	9.7	6.95–13.8	<0.001	9.9	7.2–12.6	13.7	10.5–17.89	<0.001	0.035
Total protein (g)	44.4	35.3–61.2	66.2	46.79–91.5	<0.001	72.8	52.5–95.8	100.0	75.0–138.4	<0.001	0.042
Plant protein (g)	30.4	22.2–39.7	33.8	24.4–51.5	0.008	36.9	27.6–47.3	43.5	32.9–63.5	<0.001	0.087
Animal protein (g)	12.6	8.0–19.5	25.8	16.4–45.2	<0.001	30.0	20.8–43.5	51.0	37.1–75.3	<0.001	0.058
Total fat (g)	31.2	24.8–43.1	54.6	36.4–79.4	<0.001	61.6	44.4–86.2	94.4	65.9–131.9	<0.001	0.337
Saturated fat (g)	6.9	4.8–9.7	15.6	10.2–23.5	<0.001	15.1	10.6–21.5	24.9	17.3–35.4	<0.001	0.877
Monounsaturated fat (g)	7.3	4.7–10.7	15.5	9.6–22.9	<0.001	16.7	11.99–24.8	28.1	20.6–40.7	<0.001	0.392
Polysaturated fat (g)	10.3	7.3–14.3	16.3	9.99–26.3	<0.001	18.0	12.5–25.7	25.6	16.8–39.2	<0.001	0.694
Cholesterol (mg)	111	72–171	210	87–377	<0.001	249	163–355	408	279–657	<0.001	0.027
Total carbohydrate (g)	257.5	199.0–334.96	333.4	231.6–458.6	<0.001	330.8	238.6–431.6	419.4	306.7–584.2	<0.001	0.100
Added sugar (g)	23.9	14.27–33.06	45.3	23.9–82.5	<0.001	34.7	20.9–56.1	59.3	36.5–99.7	<0.001	0.365
Dietary fibre (g)	18.5	14.2–25.2	20.6	14.2–31.1	0.003	27.3	17.5–35.4	31.4	23.1–43.1	<0.001	0.066
Ca (mg)	229	156–345	339	192–552	<0.001	397	292–573	596	432–800	<0.001	0.073
Fe (mg)	12.2	9.1–15.9	16.0	11.4–23.1	<0.001	16.0	11.2–21.9	22.2	16.2–30.1	<0.001	0.012
Mg (mg)	297	214–452	402	285–81	<0.001	389	288–539	493	369–745	<0.001	0.208
P (mg)	847	646–1113	1206	839–1638	<0.001	1107	856–1477.2	1642	1166–2229	<0.001	0.011
K (mg)	1394	1089–1759	2441	1711–3243	<0.001	2078	1526–2809	3326	2420–4378	<0.001	0.267
Zn (mg)	8.6	6.7–11.3	13.3	9.5–20.2	<0.001	12.5	9.0–17	18.4	14.1–26.5	<0.001	0.038
Cu (mg)	1.1	0.8–1.5	1.4	0.9–1.9	<0.001	1.5	1.1–1.9	1.9	1.4–2.6	<0.001	0.019
Mn (µg)	1749	1110–3259	2061	1329–3124	0.690	2647	1899–3866	3132	2219–4188	0.008	0.156
Vitamin A (µg)	4267	274–712	941	535–1727	<0.001	890	536–1447	1742	1042–2572	<0.001	0.019
Thiamin (mg)	1.6	1.2–2.1	1.9	1.3–2.8	<0.001	1.9	1.3–2.7	2.5	1.8–3.7	<0.001	0.030
Riboflavin (mg)	0.9	0.7–1.4	1.4	0.97–1.97	<0.001	1.5	1.1–2.1	2.3	1.5–3.1	<0.001	<0.001
Niacin (mg)	12.2	9.4–17.6	24.1	17.0–34.8	<0.001	18.5	14.0–25.1	34.4	25.2–49.7	<0.001	<0.001
Vitamin B ₆ (mg)	1.2	0.94–1.6	2.9	1.9–4.3	<0.001	1.8	1.2–2.7	4.4	3.0–6.4	<0.001	<0.001
Folate (µg)	356.9	268.9–467.5	467.7	300.8–744.9	<0.001	443.0	307.3–626.5	675.9	485.3–1022.8	<0.001	0.003
Vitamin B ₁₂ (µg)	1.8	0.9–2.95	2.73	1.3–5.5	<0.001	4.8	2.4–7.7	6.4	3.8–11.9	<0.001	0.136
Pantothenic acid (mg)	2.9	2.2–4.1	4.6	3.1–6.5	<0.001	4.9	3.6–6.6	8.61	6.2–11.4	<0.001	<0.001
Biotin (µg)	25.0	18.2–31.9	30.8	19.2–48.4	<0.001	45.8	29.5–61.7	62.5	41.9–88.7	<0.001	0.025
Vitamin C (mg)	12	7–16	29	14–56	<0.001	33	20–57	62	36–97	<0.001	0.447
Vitamin D (mg)	1.7	0.9–2.7	2.4	0.96–5.1	<0.001	3.2	1.98–4.8	5.0	2.8–8.2	<0.001	0.040
Vitamin E (mg)	8.4	5.2–11.9	11.9	6.1–19.6	<0.001	12.4	8.3–17.4	16.0	10.9–24.2	<0.001	0.742
Alcohol (g)	4.3	0–32	0.7	0–18.8	0.012	11.4	0–27.7	7.1	0–26.5	0.587	0.187
Energy distribution											
% of TE from protein	10.6	9.8–11.6	11.3	9.5–13.5	0.005	12.5	11.6–13.5	12.7	11.3–14.3	0.483	0.088
% of TE from animal protein	3.1	2.0–4.2	4.6	2.9–7.5	<0.001	5.4	4.3–6.9	6.8	5.1–8.5	<0.001	0.109
% of TE from plant protein	7.5	6.7–8.1	6.2	5.2–7.2	<0.001	6.5	5.7–7.2	5.7	4.8–6.3	<0.001	0.014
% of TE from total fat	17.8	13.6–23.1	21.5	15.7–28.9	<0.001	24.9	21.6–29.4	26.3	22.1–31.3	0.329	0.003
% of TE from saturated fat	3.9	2.5–5.1	6.2	4.1–8.5	<0.001	6.4	5.1–7.6	6.9	5.7–8.8	0.001	<0.001
% of TE from monounsaturated fat	3.9	2.7–5.5	6.3	4.3–8.6	<0.001	7.1	5.8–8.4	8.0	6.5–9.8	0.001	<0.001
% of TE from polysaturated fat	5.9	4.1–7.9	6.1	4.2–8.9	0.021	7.2	5.7–8.8	7.2	5.7–9.1	0.943	0.106
% of TE from total carbohydrate	63.8	58.1–69.7	59.5	51.6–66.5	<0.001	56.1	52.0–60.6	54.3	48.4–59.7	0.011	0.113
% of TE from added sugar	5.8	2.9–8.3	8.1	4.5–14.2	<0.001	6.5	4.5–9.2	7.9	4.5–12.0	0.001	0.048
% of TE from alcohol	2.0	0–12.5	0.3	0–6.0	<0.001	3.5	0–7.9	1.4	0–4.9	0.012	0.204



Table 2 Continued

Energy and nutrients	Rural men (n 186)				Urban men (n 202)			
	2005		2010		2005		2010	
	Median	P25–P75	Median	P25–P75	Median	P25–P75	Median	P25–P75
Nutrient density								
Dietary fibre (g/4.2 MJ)	11.4	8.8–13.2	9.6	7.8–10.9	11.0	9.5–12.9	9.9	8.3–11.2
Ca (mg/4.2 MJ)	130.1	98.5–167.8	140.1	99.8–214.2	179.9	139.5–222.3	179.1	134.2–233.9
Fe (mg/4.2 MJ)	5.3	4.8–5.7	6.9	6.2–7.7	5.4	4.9–5.9	6.9	6.1–7.8
Zn (mg/4.2 MJ)	7.2	6.6–8.3	6.1	5.2–6.9	6.8	6.0–7.6	6.1	5.0–6.9
Thiamin (mg/4.2 MJ)	0.9	0.8–1.1	0.9	0.7–1.0	0.8	0.7–0.9	0.8	0.7–0.9
Riboflavin (mg/4.2 MJ)	0.6	0.5–0.8	0.6	0.5–0.7	0.7	0.6–0.8	0.7	0.6–0.8
Niacin (mg/4.2 MJ)	7.5	6.4–8.6	10.6	9.4–12.1	7.8	7.1–8.8	11.0	9.7–12.5
Vitamin C (mg/4.2 MJ)	6.3	3.9–9.4	12.4	6.5–22.8	14.6	9.9–21.6	19.3	13.2–28.7

PURE-NWP-SA, Prospective Urban and Rural Epidemiology in the North West Province of South Africa; P25, 25th percentile; P75, 75th percentile; TE, total energy.

*P value for significance of differences in intakes between 2005 and 2010 for rural men or urban men (two-sample Wilcoxon test).

†P value for significance of differences in the change in intakes from 2005 to 2010 between rural men and urban men (Wilcoxon signed-rank test).

the changes in macronutrient distribution and micro-nutrient density showed distinct patterns.

In rural women, significant increases in the percentage of energy from animal protein, total, saturated, mono-unsaturated and polyunsaturated fats, as well as added sugar, were observed. Significant decreases in the percentage of energy from plant protein and total carbohydrate were seen. Furthermore, significant improvements in the nutrient density of Ca, Fe, niacin and vitamin C occurred, but the density of dietary fibre and thiamin decreased.

In urban women, percentage of energy from animal protein increased, while that from plant protein decreased. The change in percentage of energy from total fat was not significant. There was a significant decrease in the energy contribution from total carbohydrate between 2005 and 2010, but a significant increase from added sugar. Significant improvements in the nutrient density of Fe, niacin and vitamin C were seen, while that of Zn decreased.

Comparison of the differences in change of nutrient intakes between rural and urban participants

A possible question is whether the changes in nutrient intakes from 2005 to 2010 were significantly greater or smaller in rural than in urban participants. We therefore calculated the significance of the effect of time, shown in the final column of Tables 2 and 3.

Differences in change of nutrient intakes from 2005 to 2010 between rural and urban men

The significance of the differences in the change of nutrient intakes from 2005 to 2010 between rural and urban men is shown in Table 2. Because the change in energy intake from 2005 to 2010 was 1172 kJ/d greater in urban men, the changes in intakes of total protein, cholesterol and most micronutrients were also greater in urban men. However, when macronutrient intakes are expressed as a percentage of total energy and micro-nutrients intakes are expressed per 4.2 MJ, the changes in rural men were actually larger than those in urban men. These differences were significant for percentage of energy from plant protein, total, saturated and mono-unsaturated fats, and added sugar. The change in nutrient density was significantly higher in the rural men compared with the urban men for Fe.

Differences in change of nutrient intakes from 2005 to 2010 between rural and urban women

Table 3 shows that the median change in energy from 2005 to 2010 was slightly more in rural women (2.6 v. 2.3 MJ/d) than urban women ($P=0.049$). There were also other significant differences, for example the change in total fat intake was significantly greater in rural women.

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Energy and nutrients	Rural women (n 411)				Urban women (n 355)						
	2005		2010		P†	2005		2010			
	Median	P25–P75	Median	P25–P75		Median	P25–P75	Median	P25–P75		
Energy (MJ)	6.2	5.0–7.6	9.1	6.9–12.8	<0.001	9.0	6.5–11.6	11.7	8.9–14.9	<0.001	0.049
Total protein (g)	40.4	31.9–51.0	60.4	44.6–82.5	<0.001	63.2	47.4–87.4	86.5	64.2–113.9	<0.001	0.808
Plant protein (g)	27.1	20.9–33.2	33.6	24.3–48.9	<0.001	31.1	22.8–40.4	36.5	26.7–49.3	<0.001	0.039
Animal protein (g)	12.5	7.3–18.5	24.1	14.1–37.1	<0.001	29.2	21.4–40.9	46.7	30.9–65.9	<0.001	0.055
Total fat (g)	32.1	23.2–42.4	56.6	36.6–86.7	<0.001	64.7	45.9–88.4	83.5	58.3–112.4	<0.001	<0.001
Saturated fat (g)	7.0	4.6–9.8	15.4	9.6–24.5	<0.001	16.6	11.3–23.0	22.9	15.3–31.4	<0.001	<0.001
Monounsaturated fat (g)	7.1	4.9–10.7	15.9	9.8–24.6	<0.001	18.1	12.2–26.2	25.7	16.7–36.5	<0.001	0.026
Polysaturated fat (g)	10.3	7.0–14.7	17.7	10.9–27.7	<0.001	18.3	12.6–25.8	22.6	16.0–33.7	<0.001	0.002
Cholesterol (mg)	102	60–157	167	83313	<0.001	234.9	152.9–334.9	342.4	215.1–509.8	<0.001	0.018
Total carbohydrate (g)	243.5	191.3–295.6	322.0	240.9–468.8	<0.001	294.6	208.8–376.2	368.3	274.9–477.7	<0.001	0.103
Added sugar (g)	23.9	12.8–36.5	46.6	24.2–83.6	<0.001	40.6	24.1–62.1	67.6	32.6–98.5	<0.001	0.201
Dietary fibre (g)	17.3	13.8–22.1	20.7	14.9–31.3	<0.001	22.8	15.1–30.6	27.5	19.6–37.8	<0.001	0.659
Ca (mg)	198	129–277	344	200–514	<0.001	402	282–622	526	372–740	<0.001	0.050
Fe (mg)	11.2	8.9–13.9	15.5	11.2–21.6	<0.001	13.8	9.5–18.7	18.7	13.8–25.2	<0.001	0.526
Mg (µg)	240	188–310	365	270–522	<0.001	331	237–424	402	300–548	<0.001	0.004
P (mg)	694	544–876	1054	788–1413	<0.001	1018	744–1361	1292	976–1751	<0.001	0.063
K (mg)	1223	991–1546	2322	1742–3213	<0.001	2088	1394–2581	2948	2208–3898	<0.001	0.302
Zn (mg)	7.9	6.2–9.6	12.4	9.0–17.3	<0.001	10.8	7.5–14.8	15.7	11.8–20.9	<0.001	0.275
Cu (mg)	1.0	0.8–1.3	1.4	1.0–1.9	<0.001	1.4	1.0–1.9	1.7	1.3–2.4	<0.001	0.112
Mn (mg)	1221	783–1876	1820	1296–2524	<0.001	2252	1532–3152	2650	1804–3618	<0.001	0.241
Vitamin A (µg)	476	336–704	959	609–1575	<0.001	995	560–1563	1724	985–2588	<0.001	0.843
Thiamin (mg)	1.4	1.1–1.8	1.8	1.3–2.6	<0.001	1.5	1.1–2.2	2.0	1.6–2.9	<0.001	0.605
Riboflavin (mg)	0.7	0.6–1.0	1.2	0.9–1.7	<0.001	1.4	0.9–2.0	1.9	1.4–2.7	<0.001	0.788
Niacin (mg)	10.4	8.4–13.6	22.2	15.7–30.6	<0.001	16.1	11.3–22.3	28.2	21.0–38.9	<0.001	<0.001
Vitamin B6 (mg)	1.2	0.9–1.5	2.2	1.5–3.8	<0.001	1.6	1.1–2.3	3.4	2.5–4.7	<0.001	0.054
Folate (µg)	335	253–429	424	260–663	<0.001	374	272–531	533	375–781	<0.001	0.876
Vitamin B12 (µg)	1.7	0.8–3.0	2.4	1.2–5.2	<0.001	4.6	2.7–7.5	5.6	3.1–10.6	<0.001	0.003
Pantothenic acid (mg)	2.6	2.0–3.5	4.3	3.1–6.6	<0.001	4.7	3.4–6.2	6.8	5.4–9.5	<0.001	0.002
Biotin (µg)	22.2	16.9–31.7	29.2	20.1–44.5	<0.001	42.8	29.8–58.3	58.7	38.6–78.7	<0.001	0.132
Vitamin C (mg)	13	8–19	32	18–55	<0.001	40	21–59	64	40–107	<0.001	0.045
Vitamin D (mg)	1.6	0.8–2.6	1.9	0.9–0.9	<0.001	2.8	1.6–4.7	4.2	2.5–6.6	<0.001	0.307
Vitamin E (mg)	7.9	5.6–12.1	11.2	6.9–16.7	<0.001	12.4	8.4–16.9	14.7	9.6–21.7	<0.001	0.825
Alcohol (g)	0.0	0.0–0.0	0.0	0.0–0.0	0.017	0.0	0.0–14.3	0.0	0.0–6.1	0.092	0.315
Energy distribution											
% of TE from protein	10.9	9.9–12.0	11.1	9.6–12.9	0.326	12.5	11.3–13.5	12.5	11.1–14.3	0.025	0.817
% of TE from animal protein	3.3	2.2–4.8	4.5	2.8–6.7	<0.001	5.8	4.7–7.2	6.8	5.1–9.2	<0.001	<0.001
% of TE from plant protein	7.5	6.6–8.0	6.3	5.4–7.4	<0.001	6.1	5.3–6.8	5.5	4.6–6.2	<0.001	<0.001
% of TE from total fat	20.3	15.6–24.5	22.9	17.7–30.0	<0.001	28.4	23.7–31.9	27.7	22.8–32.3	0.335	<0.001
% of TE from saturated fat	4.3	2.9–5.7	6.5	4.6–8.5	<0.001	7.0	5.8–8.6	7.5	5.9–9.0	0.130	<0.001
% of TE from monounsaturated fat	4.5	3.2–5.9	6.2	4.6–8.7	<0.001	7.9	6.4–9.8	8.4	6.6–10.5	0.019	<0.001
% of TE from polysunsaturated fat	6.5	4.8–8.6	7.3	4.9–9.9	<0.001	8.0	6.4–9.7	7.6	6.1–9.3	0.125	<0.001
% of TE from total carbohydrate	66.7	61.3–71.9	61.7	53.8–67.7	<0.001	55.7	51.1–60.2	54.1	49.5–59.8	0.039	<0.001
% of TE from added sugar	6.4	3.7–10.2	8.9	4.3–14.9	<0.001	8.2	5.7–10.8	9.0	5.9–14.5	<0.001	0.055
% of TE from alcohol	0.0	0.0–0.0	0.0	0.0–0.0	<0.001	0.0	0.0–4.4	0.0	0.0–5.5	0.002	0.731



Table 3 Continued

Energy and nutrients	Rural women (n 411)					Urban women (n 355)				
	2005		2010			2005		2010		
	Median	P25-P75	Median	P25-P75	P†	Median	P25-P75	Median	P25-P75	P value*
Nutrient density										
Dietary fibre (g/4.2 MJ)	11.9	9.8–13.9	9.7	8.1–11.7	<0.001	10.7	8.9–12.3	10.2	8.7–11.8	0.193
Ca (mg/4.2 MJ)	124.1	96.4–163.7	154.7	96.8–224.3	<0.001	198.4	157.2–261.0	195.0	147.9–248.0	0.134
Fe (mg/4.2 MJ)	5.4	4.8–5.9	6.9	6.2–7.8	<0.001	5.2	4.6–5.8	6.8	6.0–7.7	0.003
Zn (mg/4.2 MJ)	7.7	6.7–8.7	5.8	4.9–6.6	<0.001	6.5	5.6–7.5	5.6	4.9–6.6	<0.001
Thiamin (mg/4.2 MJ)	0.9	0.8–1.2	0.8	0.7–1.0	<0.001	0.8	0.7–0.9	0.8	0.6–0.9	0.725
Riboflavin (mg/4.2 MJ)	0.5	0.4–0.6	0.5	0.5–0.7	0.007	0.7	0.6–0.8	0.7	0.6–0.9	0.006
Niacin (mg/4.2 MJ)	7.2	6.3–8.3	10.1	8.6–11.6	<0.001	7.4	6.7–8.6	10.4	9.1–12.1	<0.001
Vitamin C (mg/4.2 MJ)	8.8	5.6–12.7	13.8	8.2–22.8	<0.001	17.8	12.8–24.4	23.9	16.3–34.8	<0.001

PURE-NWP-SA, Prospective Urban and Rural Epidemiology in the North West Province of South Africa; P25, 25th percentile; P75, 75th percentile; TE, total energy.

*P value for significance of differences in intakes between 2005 and 2010 for rural women or urban women (two-sample Wilcoxon test).

†P value for significance of differences in the change in intakes from 2005 to 2010 between rural women and urban women (Wilcoxon signed-rank test).

When expressed as a percentage of energy, the differences in change of macronutrients were significant for plant protein, total, saturated, monounsaturated and polyunsaturated fats, and total carbohydrate, with the rural women showing the largest change. Significant differences in change of micronutrients expressed per energy are also shown in Table 3. For dietary fibre, these changes showed a decrease in rural women but an increase in urban women. Therefore, the direction of change in rural and urban women was not always the same when nutrients were expressed in terms of energy intake.

Evaluation of dietary quality

We also measured how well the cohort participants met their nutrient needs and dietary recommendations for the prevention of NCD. The recommendations in Supplemental Table 1 (see online supplementary material) were used to determine whether a particular nutrient was under- or overconsumed by each participant. Furthermore, the percentage of participants in specific groups who did not meet their recommended intakes was also calculated. Table 4 gives values for men and Table 5 those for women, both by date (2005 and 2010) and rural *v.* urban participants.

Table 4 shows that in 2005 more than 50% of the rural men did not meet the recommendation for sixteen of the twenty-three nutrients presented. More than 50% of the urban men did not meet the recommendations for only six of the twenty-three nutrients in 2005. Although the situation improved in 2010 for the rural men, more than 50% of them did not meet the recommendations for cholesterol, dietary fibre, Ca, K, pantothenic acid, vitamin C, vitamin D and vitamin E. Despite a higher energy intake (meaning that more food was eaten), still more than 50% of the urban men had low Ca, K, vitamin C and vitamin D intakes in 2010 which did not meet the recommendations. In addition, the energy distribution was not optimal. There was a significant improvement in intake compared with the recommendation for rural and urban men for total protein, cholesterol, Zn, Cu, Mn, vitamin A, riboflavin, niacin, vitamin B₆, pantothenic acid, biotin, vitamin C, vitamin D and vitamin E.

Table 5 shows that in 2005, 50% of the rural women did not meet the recommendation for seventeen of the twenty-three nutrients presented. Although there was a significant improvement from 2005 to 2010, more than 50% of the rural women still had an intake not meeting the recommendation for nine nutrients (cholesterol, fibre, Ca, K, pantothenic acid, biotin, vitamin C, vitamin D and vitamin E) in 2010. The nutrient quality of the diet of the urban women was better than that of the rural women, with more than 50% of the urban women not meeting the recommendation for only seven of the twenty-three nutrients in 2005 and for only three nutrients (Ca, K and vitamin D) in 2010.

Table 4 Nutrient intakes of the male cohort, expressed as a percentage of the recommendation, and the percentage who did not meet recommendations by urban/rural residence in 2005 and 2010; PURE-NWP-SA study

Nutrients	Rural men (n 186)					Urban men (n 202)				
	2005					2005				
	Median	P25–P75	%*	Median	P25–P75	%*	Median	P25–P75	%*	P value†
Total protein	79.3	63.1–109.2	69.9	118.2	83.6–163.3	37.6	129.9	93.8–171.0	30.7	<0.001
Cholesterol	37	24–57	95.2	70.1	29.1–125.7	64.5	83.1	54.4–118.3	66.8	<0.001
Dietary fibre	73.9	56.8–100.7	74.7	82.4	56.7–124.6	64.0	109.0	70.1–141.6	47.0	<0.001
Ca	28.4	19.3–42.8	98.9	42.0	24.0–66.2	94.1	49.2	36.0–71.5	89.6	<0.001
Fe	203.6	151.1–265.0	6.5	265.9	189.5–385.7	4.3	266.3	186.8–364.2	6.4	<0.001
Mg	84.8	61.3–129.1	61.3	114.8	81.4–166.0	38.7	111.1	82.2–153.9	38.1	<0.001
P	146.0	111.4–191.8	100.0	208.0	144.6–282.4	9.1	190.9	147.6–254.7	9.4	<0.001
K	29.7	23.2–37.4	100.0	51.9	36.4–69.0	94.1	44.2	32.5–59.8	97.5	<0.001
Zn	91.9	70.7–119.8	55.4	141.6	100.5–214.9	24.7	133.3	95.3–181.6	27.2	<0.001
Cu	153.4	114.3–215.9	14.0	198.1	133.8–278.6	9.1	211.4	151.5–289.7	10.9	<0.001
Mn	76.1	48.2–141.7	63.4	89.6	57.8–135.8	53.2	115.1	82.6–168.1	42.6	<0.001
Vitamin A	68.3	43.9–113.8	70.4	150.5	85.6–276.3	30.7	142.4	85.7–231.5	33.2	<0.001
Thiamin	158.7	119.4–206.2	14.5	190.3	128.1–283.7	10.8	190.9	134.1–273.4	12.9	<0.001
Riboflavin	84.4	61.9–128.8	63.4	131.0	88.4–179.1	32.8	136.3	95.6–186.4	26.2	<0.001
Niacin	101.4	78.0–146.5	48.9	200.7	142.0–290.1	7.0	154.3	116.8–209.4	19.3	<0.001
Vitamin B ₆	101.5	78.1–128.8	46.8	202.0	124.9–305.2	8.6	157.1	95.1–210.7	28.2	<0.001
Folate	111.6	84.0–146.1	39.8	146.1	94.0–232.8	29.0	138.4	96.0–195.8	26.2	<0.001
Vitamin B ₁₂	88.0	45.3–147.6	59.1	129.6	65.8–275.9	39.8	238.8	118.2–385.1	17.3	<0.001
Pantothenic acid	57.4	44.8–81.5	86.0	92.0	62.9–130.2	55.4	98.5	71.3–131.1	51.0	<0.001
Biotin	83.4	60.8–106.4	67.7	102.7	64.1–161.4	48.4	152.6	98.3–205.7	25.3	<0.001
Vitamin C	15.3	9.0–22.0	99.5	39.1	18.1–74.8	85.0	43.8	26.5–76.5	84.2	<0.001
Vitamin D	17.1	8.9–26.5	98.9	23.8	9.6–51.2	90.3	32.0	19.8–48.1	97.5	<0.001
Vitamin E	69.8	43.0–99.3	75.8	98.0	48.8–162.7	51.6	103.4	68.9–145.4	48.5	<0.001
Energy distribution										
% of TE from fat	59.3	45.2–76.9	94.6	71.6	52.2–96.6	78.0	83.1	72.1–98.1	77.2	0.056
% of TE from saturated fat	38.5	25.1–50.6	96.2	62.1	41.3–84.5	90.3	64.5	45.0–91.8	93.6	0.034
% of TE from polyunsaturated fat	99.7	69.4–131.9	51.6	101.6	70.6–148.8	49.5	119.4	95.5–148.2	29.2	1.000
% of TE from added sugar	58.3	29.9–82.9	85.0	80.7	45.4–142.4	57.0	63.6	51.1–75.5	80.2	<0.001

PURE-NWP-SA, Prospective Urban and Rural Epidemiology in the North West Province of South Africa; P25, 25th percentile; P75, 75th percentile; TE, total energy.

*Percentage of participants not meeting the recommendations.

†P value for significance of differences between 2005 and 2010 for rural men or urban men not meeting the recommendations (Wilcoxon two-sample test).



Table 5 Nutrient intakes of the female cohort, expressed as a percentage of the recommendation, and percentage who did not meet recommendations by urban/rural residence in 2005 and 2010; PURE-NWP-SA study

Nutrients	Rural women (n 411)					Urban women (n 355)				
	2005			2010		2005			2010	
	Median	P25-P75	%*	Median	P25-P75	Median	P25-P75	%*	Median	P25-P75
Total protein	87.9	69.4-110.9	63.3	131.4	97.0-179.3	137.6	103.1-190.0	22.3	188.1	139.6-247.7
Cholesterol	33.9	20.1-52.3	93.2	55.8	27.8-104.5	78.3	51.0-111.6	66.8	114.1	71.7-169.9
Dietary fibre	69.3	55.0-88.4	87.6	83.0	59.9-125.0	91.0	60.4-122.5	56.9	109.9	78.3-151.1
Ca	22.9	14.7-31.6	98.5	38.0	22.5-57.5	46.0	30.9-70.7	93.0	56.2	39.1-82.4
Fe	166.0	123.8-216.8	10.5	250.3	176.8-352.0	206.0	144.7-305.9	9.6	316.1	226.8-431.4
Mg	90.5	71.0-116.9	59.9	137.8	101.8-196.8	125.0	89.4-159.9	31.0	151.8	113.1-206.7
P	119.7	93.9-151.0	29.7	181.7	135.9-243.6	175.5	128.2-234.7	10.7	222.7	168.3-301.8
K	26.0	21.1-32.9	99.8	49.4	37.1-68.4	44.4	29.7-54.9	98.9	62.7	47.0-82.9
Zn	115.8	91.5-141.8	33.6	182.0	132.8-254.3	159.5	110.9-217.4	18.0	231.3	173.4-308.1
Cu	141.6	112.0-178.6	73.7	198.5	140.6-269.4	196.2	143.3-272.8	34.4	243.3	178.2-345.3
Mn	67.8	43.5-104.2	16.6	101.1	72.0-140.2	125.1	85.1-175.1	9.6	147.2	100.2-201.0
Vitamin A	95.2	67.2-140.8	53.3	192.0	121.8-315.1	198.9	112.1-312.6	21.4	344.8	197.1-517.6
Thiamin	158.7	126.0-200.9	8.0	200.0	141.9-287.0	170.0	124.4-248.7	12.7	226.5	172.0-317.2
Riboflavin	80.9	62.1-112.5	68.1	135.9	99.4-193.4	156.4	108.9-223.1	21.7	221.1	155.2-298.4
Niacin	94.6	76.5-123.4	55.2	201.3	142.9-278.6	146.1	102.5-202.7	22.8	256.0	191.3-353.7
Vitamin B ₆	98.6	77.8-129.0	50.6	184.8	125.2-297.8	130.0	86.5-190.3	30.7	289.8	198.0-394.6
Folate	104.6	79.1-134.0	45.7	132.6	81.1-207.1	116.9	84.8-165.9	36.6	166.5	117.1-244.0
Vitamin B ₁₂	82.9	40.5-150.7	56.9	120.1	62.2-258.7	229.8	133.7-374.6	17.8	280.7	152.3-532.2
Pantothenic acid	52.1	40.0-70.5	92.0	85.8	61.4-131.0	94.2	67.6-123.1	56.1	136.8	108.3-190.9
Biotin	74.1	56.3-105.5	72.0	97.3	66.9-148.2	142.6	99.5-194.3	25.1	195.7	128.7-262.4
Vitamin C	20.9	13.7-31.5	98.1	52.6	29.4-90.9	66.3	35.0-98.6	75.2	106.8	67.1-178.5
Vitamin D	15.9	8.4-25.6	98.8	19.5	9.3-45.7	27.6	15.8-47.3	98.3	41.5	24.8-66.2
Vitamin E	66.4	46.8-100.6	74.5	93.1	58.0-139.1	103.6	70.4-141.0	46.2	122.2	80.0-181.1
Energy distribution										
% of TE from fat	67.5	52.0-81.7	92.0	76.2	58.9-100.0	94.6	78.9-106.5	62.8	92.2	76.1-107.6
% of TE from saturated fat	64.2	36.5-102.0	97.8	64.6	45.6-85.2	81.8	56.9-108.2	88.2	75.0	58.8-90.1
% of TE from polyunsaturated fat	107.8	80.4-143.2	43.1	121.7	83.2-166.5	133.5	107.4-161.7	18.6	126.9	101.9-154.2
% of TE from added sugar	42.8	29.9-56.8	74.2	89.8	42.7-149.0	70.2	57.5-86.7	67.9	90.3	59.4-144.7

PURE-NWP-SA, Prospective Urban and Rural Epidemiology in the North West Province of South Africa; P25, 25th percentile; P75, 75th percentile; TE, total energy.

*Percentage of participants not meeting the recommendations.

†P value for significance of differences between 2005 and 2010 for rural women or urban women not meeting the recommendations (Wilcoxon two-sample test).



The energy distribution of the rural and urban women was not optimal. Because of overconsumption, more than 50% of the participants in 2005 and 2010 did not meet the restrictive recommendations for total fat, saturated fat and added sugar.

Discussion

The objectives of the present part of the PURE-NWP-SA study were to measure and report the changes in nutrient intakes of rural and urban black African men and women over time, to assess the impact of urbanization and modernization of lifestyles on dietary and therefore nutrient intakes and NCD risk.

Energy intakes

The first important result was the substantial increase in energy intake from 2005 to 2010 in all groups. Increases in median values were 2.3, 3.4, 2.6 and 2.3 MJ/d for rural and urban men and rural and urban women, respectively. In 2010 (within 5 years) the rural subjects 'caught up' with the energy intake of the urban subjects measured in 2005. In 2010 the urban men consumed 13.3 MJ daily and women 11.3 MJ daily.

The question arises whether these increases are real or a result of methodological error. The facts argue against the latter. The same team of fieldworkers and researchers used in 2010 the same validated QFFQ that was used in 2005 to measure intakes. The reproducibility of the QFFQ was satisfactorily validated in the PURE-NWP-SA study in 2005⁽¹⁸⁾. Because the median energy values of rural men and women in 2010 were similar to those of urban men and women in 2005, this suggests that 'environmental' or lifestyle factors were responsible for the increased intakes in 2010. There are several known contenders: (i) after the 2005 measurements, a large grocery and food store opened in the rural area which increased the availability and affordability of many foods, processed food products and beverages; (ii) social support in the form of grants in both rural and urban areas, and thus income, also increased over this time; and (iii) the rural area experienced a severe drought in 2005 which limited the availability of fresh products and income to buy them.

It seems reasonable to conclude that the observed rapid increases in energy intake in both rural and urban participants were real and part of an accelerated nutrition transition. The increases in energy intake were of course accompanied by expected increases in intakes of all other nutrients because of greater food consumption.

Similarly, a consistent rise in total energy supply (between 1970 and 2005) has been reported in the Middle East and North African countries⁽²⁵⁾, while the Chinese population is consuming more energy-dense food than previously⁽²⁶⁾. In Brazil, dietary deficit is changing to dietary excess⁽²⁷⁾. It was recently reported in Nepal that there has been a change from an agricultural staple-based

foods diet to a modern processed-foods diet with higher total energy, total fat and sugar⁽²⁸⁾.

Macronutrient distribution

Another salient observation is the changes in macronutrient intakes and macronutrient distribution (expressed as a percentage of total energy) over time. These changes followed the same pattern described as part of the nutrition transition in the NWP when rural and urban Africans were compared in a cross-sectional study^(7,8). The differences in intakes between rural and urban participants in 2005 were similar to the changes observed in both rural and urban participants over time (in 2010). The urban participants ate more in 2005 and 2010 than their rural counterparts. With a few exceptions, the changes included the following: (i) the percentage of energy contributed by total protein (except urban men), animal-derived protein, total fat (except urban women), saturated fat, mono-unsaturated fat and added sugar all increased significantly and reflected increased intakes of mainly meat, eggs and milk; (ii) the percentage of energy from plant protein decreased significantly in all groups; and (iii) the percentage of energy from total carbohydrate decreased significantly in rural participants despite the increase in added sugar (a carbohydrate which forms part of total carbohydrate), therefore this decrease probably reflects the decreased intake of starchy staple foods. Because these changes occurred mostly in rural participants it suggests that the diet followed by urban participants in 2005 (except for energy intake) was already modernized to a 'stable' level and that rural participants still had 'room to move' towards the energy distribution seen as a result of the nutrition transition. The change from a high-fibre-rich carbohydrate to a lower-carbohydrate diet with more sugar and sugar-sweetened beverages is also taking place in other countries⁽²⁸⁻³⁰⁾. In addition, a dramatic increase in meat consumption has been reported for China and Brazil⁽³¹⁾.

An interesting finding is the significantly lower alcohol intake in rural and urban men in 2010 (both in grams and as a percentage of energy intake). A possible explanation could be that due to the nutrition transition, modernization and moving away from traditional home-brew methods, men consumed commercial beer rather than home-made beer. Commercial beer is more expensive and the higher price could have lowered its consumption.

Although we did not test for significance between men and women, it is interesting to note that women consumed more added sugar (in grams and as a percentage of energy; except for the rural women in 2005) than men. We previously showed⁽¹¹⁾ that more women than men consumed sucrose-sweetened beverages in 2005 and 2010 and that the quantities were also larger. It could also be that the women consumed more sugar in their tea/coffee.

**Micronutrient intakes: nutrient adequacy**

One would have hoped and expected that the fortification of maize meal and bread flour, two main staples in these populations, would correct micronutrient deficiencies of most people in SA. The present study, however, showed that intakes of the rural men and women in 2005 did not meet recommended values for most minerals and vitamins. The situation was better in the urban groups and improved over time in both rural and urban participants. Therefore, the nutrition transition resulted in improved, but still not optimal micronutrient intakes. The South African food composition tables incorporated new analytical data of the fortified staple foods. However, it was reported in 2012⁽³²⁾ that many of the samples of maize meal and white bread flour across SA that were analysed were insufficiently fortified. To assess the real extent of micronutrient deficiencies, suitable biological markers of the micronutrients will have to be measured in future. Nevertheless, the data showed that micronutrient needs in the PURE-NWP-SA participants were not met despite a high intake of energy, probably a result of more energy-dense but nutrient-poor foods chosen.

Protective dietary effects

The low intakes of dietary fibre and many of the antioxidant micronutrients are of concern. The low-fat, high-fibre traditional diet of the indigenous people of Africa has been regarded as a major factor protecting them against NCD^(2,33,34). The nutrient intake changes observed here are indicative of a loss of this protection of the diet. Schutte and co-workers⁽¹⁴⁾ already reported that 24% of participants of the PURE-NWP-SA study who had normal blood pressure in 2005 presented with hypertension in 2010, while we showed the massive increased consumption of nutrient-poor sugar-containing beverages over this time⁽¹¹⁾. Together with other changes during the epidemiological transition, the increase in NCD among the Africans in SA is not surprising.

Limitations

One of the major limitations of the PURE-NWP-SA study, namely the moderately high proportion of subjects lost to follow-up, of whom 30% died, was discussed previously⁽¹¹⁾. Those lost to follow-up were significantly younger, more often males, a higher percentage were HIV-infected, with lower BMI, waist circumference, HDL-cholesterol and energy intake than those followed up. We suspect that the rest of the participants lost to follow-up, being younger and male, probably moved away to bigger cities where there are more opportunities for work.

Conclusions

It is concluded that the results of the current analysis of the PURE-NWP-SA nutrient data confirm a rapid nutrition transition in the NWP, characterized by large increases in

total food energy without correcting fully for micronutrient deficiencies despite fortification of staple foods. Micronutrient intakes could even be lower because of insufficient fortification. Furthermore, the shift towards an animal food-based diet at the cost of a lower plant protein food intake could increase the risk of NCD. The data also confirmed that low dietary fibre and micronutrient intakes were prevalent, further contributing to a double burden of under- and overnutrition in this population.

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Conflict of interest: The authors declare that they have no competing interests. *Authorship:* E.W.-V. and H.H.V. were responsible for the conception and design, analysis and interpretation of the data and wrote the manuscript. S.L. made contributions to the design, analysis and interpretation of the data and critically revised the manuscript for important intellectual content. R.L. was responsible for the statistical analysis of the data and made contributions to the interpretation of the data and critically revised the manuscript for important intellectual content. *Ethics of human subject participation:* The Ethics Committee of the North-West University approved the study (04M10 and NWU-0016-10-A1). The study complied with all applicable requirements of international regulations, including the Helsinki Declaration of 1975 (as revised in 2008). Permission for the study was also obtained from the North-West Provincial Department of Health, local government authorities, and tribal chiefs in the rural areas. Volunteers gave written informed consent. The participants were transported to and from the study sites in both the urban and rural areas. They received lunch on site after blood sampling was completed. The study is registered at ClinicalTrials.gov (ID NCT03225586, <https://clinicaltrials.gov>).

**Supplementary material**

To view supplementary material for this article, please visit
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References

- Pillay-van Wyk V, Msemburi W, Laubscher R *et al.* (2016) Mortality trends and differentials in South Africa from 1997 to 2012: second National Burden of Disease Study. *Lancet Glob Health* **4**, e642–e653.
- Vorster HH, Kruger A & Margetts BM (2011) The nutrition transition in Africa: can it be steered into a more positive direction? *Nutrients* **3**, 429–441.
- Mayosi BM, Flisher AJ, Lalloo UG *et al.* (2009) The burden of non-communicable diseases in South Africa. *Lancet* **374**, 934–947.
- Nojilana B, Bradshaw D, Pillay-van Wyk V *et al.* (2016) Persistent burden from non-communicable diseases in South Africa needs strong action. *S Afr Med J* **106**, 436–437.
- Shisana O, Labadarios D, Rehle T *et al.* (2013) *South African National Health and Nutrition Examination Survey (SANHANES-1)*, pp. 135–144. Cape Town: HSRC Press.
- Norman R, Bradshaw D, Schneider M *et al.* (2007) A comparative risk assessment for South Africa in 2000: towards promoting health and preventing disease. *S Afr Med J* **97**, 637–641.
- Vorster H, Venter C, Kruger H *et al.* (2000) The impact of urbanization on physical, physiological and mental health of Africans in the North West Province of South Africa: the THUSA study. *S Afr J Sci* **96**, 505–514.
- MacIntyre U, Kruger H, Venter C *et al.* (2002) Dietary intakes of an African population in different stages of transition in the North West Province, South Africa: the THUSA study. *Nutr Res* **22**, 239–256.
- Serfontein M, Venter C, Kruger A *et al.* (2010) Alcohol intake and micronutrient density in a population in transition: the Transition and Health during Urbanisation in South Africa (THUSA) study. *S Afr J Clin Nutr* **23**, 22–28.
- MacIntyre U, Venter C, Kruger A *et al.* (2012) Measuring micronutrient intakes at different levels of sugar consumption in a population in transition: the Transition and Health during Urbanisation in South Africa (THUSA) study. *S Afr J Clin Nutr* **25**, 122–130.
- Vorster HH, Kruger A, Wentzel-Viljoen E *et al.* (2014) Added sugar intake in South Africa: findings from the Adult Prospective Urban and Rural Epidemiology cohort study. *Am J Clin Nutr* **99**, 1479–1486.
- South African Department of Health (2013) *Foodstuffs, Cosmetics and Disinfectants Act, 1972 (Act No. 54 of 1972). Regulations Relating to the Reduction of Sodium in Certain Foodstuffs and Related Matters*. Pretoria: Government Gazette, Republic of South Africa; available at <http://www.greengazette.co.za/.../index-for-20171006-national-gazette-no-41164>
- Corsi DJ, Subramanian S, Chow CK *et al.* (2013) Prospective Urban Rural Epidemiology (PURE) study: baseline characteristics of the household sample and comparative analyses with national data in 17 countries. *Am Heart J* **166**, 636–646.e634.
- Schutte AE, Schutte R, Huisman HW *et al.* (2012) Are behavioural risk factors to be blamed for the conversion from optimal blood pressure to hypertensive status in Black South Africans? A 5-year prospective study. *Int J Epidemiol* **41**, 1114–1123.
- Sotunde O, Kruger H, Wright HH *et al.* (2015) Lean mass appears to be more strongly associated with bone health than fat mass in urban black South African women. *J Nutr Health Aging* **19**, 628–636.
- MacIntyre U, Venter C & Vorster H (2001) A culture-sensitive quantitative food frequency questionnaire used in an African population: 1. Development and reproducibility. *Public Health Nutr* **4**, 53–62.
- MacIntyre U, Venter C, Vorster H *et al.* (2001) A combination of statistical methods for the analysis of the relative validation data of the quantitative food frequency questionnaire used in the THUSA study. *Public Health Nutr* **4**, 45–51.
- Wentzel-Viljoen E, Laubscher R & Kruger A (2011) Using different approaches to assess the reproducibility of a culturally sensitive quantified food frequency questionnaire. *S Afr J Clin Nutr* **24**, 143–148.
- Venter C, MacIntyre U & Vorster H (2000) The development and testing of a food portion photograph book for use in an African population. *J Hum Nutr Diet* **13**, 205–218.
- Langenhoven M, Conradie P, Wolmarans P *et al.* (1991) *MRC Food Quantities Manual*. Cape Town: South African Medical Research Council.
- Wolmarans P, Danster N, Dalton A *et al.* (2010) *Condensed Food Composition Tables for South Africa*. Cape Town: South African Medical Research Council.
- Otten JL, Hellwig JP and Meyers LD, (editors) (2006) *Dietary Reference Intakes: The Essential Guide to Nutrient Requirements*. Washington, DC: The National Academies Press.
- Nishida C, Uauy R, Kumanyika S *et al.* (2004) The joint WHO/FAO expert consultation on diet, nutrition and the prevention of chronic diseases: process, product and policy implications. *Public Health Nutr* **7**, 245–250.
- Vorster HH, Badham J & Venter C (2013) An introduction to the revised food-based dietary guidelines for South Africa. *S Afr J Clin Nutr* **26**, 3 Suppl., S5–S12.
- Sibai AA, Nasreddine L, Mokdad AH *et al.* (2010) Nutrition transition and cardiovascular disease risk factors in Middle East and North Africa countries: reviewing the evidence. *Ann Nutr Metab* **57**, 193–203.
- Popkin BM, Lu B, Zhai F *et al.* (2002) Understanding the nutrition transition: measuring rapid dietary changes in transitional countries. *Public Health Nutr* **5**, 947–953.
- Monteiro CA, Mondini L, de Souza AL *et al.* (1995) The nutrition transition in Brazil. *Eur J Clin Nutr* **49**, 105–113.
- Subedi YP, Marais D & Newlands D (2017) Where is Nepal in the nutrition transition? *Asia Pac J Clin Nutr* **26**, 358–367.
- Malik VS, Walter CW & Frank BH (2013) Global obesity: trends, risk factors and policy implications. *Nat Rev Endocrinol* **9**, 13–27.
- Mattei J, Malik V, Wedick NM *et al.* (2015) Reducing the global burden of type 2 diabetes by improving the quality of staple foods: the Global Nutrition and Epidemiologic Transition Initiative. *Global Health* **11**, 23–23.
- Vranken L, Avermaete T, Petalios D *et al.* (2014) Curbing global meat consumption: emerging evidence of a second nutrition transition. *Environ Sci Policy* **39**, 95–106.
- Yusufali R, Sunley N, de Hoop M *et al.* (2012) Flour fortification in South Africa: post-implementation survey of micronutrient levels at point of retail. *Food Nutr Bull* **33**, 4 Suppl., S321–S329.
- Walker A (1994) Highlights of nutrition and health/ill-health research in Africa: how my interest started and developed. *S Afr Med J Suppl.*, 2–4.
- Popkin BM (2002) An overview on the nutrition transition and its health implications: the Bellagio meeting. *Public Health Nutr* **5**, 93–103.

PROOF OF SUBMISSION TO THE CARDIOVASCULAR JOURNAL OF AFRICA (CHAPTER 4)

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CHAPTER 4: THE RELATIONSHIP BETWEEN THE CHANGES IN DIETARY FAT INTAKE AND CARDIOVASCULAR DISEASE RISK IN BLACK SOUTH AFRICANS FROM 2005 TO 2010: THE PURE-SA-NWP STUDY

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4.1 Abstract

Background: This study aims to investigate the association between the change in dietary fat intakes of rural and urban individuals in the North West province of South Africa and the change in CVD risk.

Methods: For this prospective study (as part of PURE-SA-NWP), baseline data were collected among black South African participants in 2005 and followed up in 2010 (n=1154).

Results: Dietary energy, PUFA, MUFA and saturated fat intakes increased in all groups. Among rural men, an increase in the change in WC was associated with a decrease in the change in PUFA ($\beta=-0.280$; $p=0.002$) and an increase in the change in MUFA ($\beta=0.200$; $p=0.027$) intake. In all groups it was associated with a change in BMI.

Conclusion: Despite the significant differences in energy and fat intake, changes in fat intake were, overall, not strongly associated with the changes in CVD risk factors. BMI emerged as a significant role player in CVD risk as a confounding factor in the analysis of other risk factors.

Keywords: Nutrition transition, cardiovascular disease, PURE-SA-NWP, urban, rural, waist circumference

4.2 Introduction

In the process of urbanisation, the dietary intakes of communities change from prudent diets (low-fat, low-energy diets) typically followed by people in rural areas to diets that are more westernised

and higher in fat and energy. This shift in dietary patterns and nutrient intakes is referred to as the nutrition transition (1-3). Since the early 2000s, the nutrition transition has been extensively reported on in certain demographic pockets in South Africa (1, 4-8). In a more recent paper investigating the change in nutrient intakes of rural and urban men and women from 2005 to 2010 the researchers reported that the rate at which the nutrition transition is occurring in rural areas is still very rapid (9).

The nutrition transition also shows associations with an increase in non-communicable diseases (NCDs) (4, 10). In light of the rapid nutrition transition in South Africa (8, 9), together with the disease burden of NCDs, this topic remains a relevant one. NCDs are reported to be the second leading cause of death in sub-Saharan Africa (11). Many low- and middle-income countries are experiencing an epidemiological transition, where the leading cause of death shifts from communicable diseases to non-communicable diseases (12, 13). In their latest report (the 2017 report, published on the 20th of March 2020), Statistics-SA reported that the causes of death in South Africa had shifted dramatically in the 10 years between 2007 and 2017. In 2007, communicable diseases were the leading cause of death in South Africa (accounting for 46.9% of deaths), followed by NCDs (44.1% of deaths), but NCD-related deaths increased by 13,7 percentage points from 2007 to 2017, making NCDs the leading cause of death (57.8%) in South Africa, while communicable diseases were responsible for only 30.7% of deaths in 2017 (14). The 2018 NCD report of the World Health Organisation (WHO) (15) revealed that the majority (19%) of NCD deaths in South Africa could be attributed to cardiovascular disease (CVD).

Several risk factors, some of which are preventable, can trigger CVDs (12). Preventable risk factors include hypertension (global prevalence of 19%); raised total cholesterol (39%), diabetes (8,5%); physical inactivity (25%); overweight (39%); and obesity (13%) (16-19). The evidence paints a worrisome picture within the South African population, with these risk factors present in all sectors of the population to a lesser or greater extent. The most recent South African (2016) has reported that 23,5% of South Africans 15 years and older are overweight, 45% have elevated blood pressure, 34% have elevated cholesterol levels, 26% are obese, and 10.5% have diabetes (20).

A five-year cohort study in the Netherlands has shown that persons who adopted four to five healthy lifestyle factors (the factors considered were diet, smoking, physical activity and alcohol consumption) were 2.5 times less likely to develop CVDs than those adhering to only one or none of the healthy lifestyle factors (21). Dietary fat intake, more specifically the type of dietary fat, has repeatedly been shown to be associated, be it positive or negative, with CVD risk (22-31). Various prospective studies over the years have noted a reduction in CVD risk when replacing saturated fat in the diet with mono-unsaturated fat (MUFA), poly-unsaturated fat (PUFA) or complex

carbohydrates (22-25, 31). These findings are also supported by meta-analysis and systematic reviews on the subject (27, 29). A recent meta-analysis found that an increase of 5% in the percentage energy contribution of PUFA was associated with a 5% decrease in CVD mortality. They also found an increased intake of saturated fat to be associated with a greater risk of death. An earlier systematic review by Hooper *et al.* (27) found that a reduction in saturated fat intake for at least two years resulted in a 17% drop in cardiovascular events and a 27% drop with the replacement of saturated fat with PUFAs.

While these studies represent a growing body of evidence supporting the idea that the type of fat consumed may be associated with CVD risk, there is a lack in prospective data in the South African context. This study was conducted over five years, from 2005 to 2010, among a cohort of rural and urban men and women in the North West province of South Africa. It formed part of the large international Prospective Urban and Rural Epidemiological (PURE) study conducted in 27 countries. In a cross-sectional analysis of the baseline (2005) data of this cohort, Dolman *et al.* (32) explored the associations between dietary fat intakes and CVD risk factors by using predefined diet quality scores and found urbanisation (particularly in urban women) to be associated with an increased CVD risk. Richter *et al.* (33) found that specific dietary fatty acids may impact the blood lipid levels of men and women differently (regardless of locality). In an earlier prospective analysis from the same cohort, individuals with a higher added sugar intake presented with a greater BMI and waist circumference (WC) and a lower high-density lipoprotein (HDL) cholesterol level compared with those whose added sugar intake contributed to less than 10% of their total energy intake (34).

Our study aims to investigate the association between the change in dietary fat intakes of individuals in rural and urban groups in the North West province of South Africa and the change in their cardiovascular disease risk (from 2005 to 2010).

4.3 Methods

4.3.1 Study design

This study is a sub-study of the South African leg of the multinational PURE study in the North West province (PURE-SA-NWP). PURE is an international epidemiological prospective (cohort) study coordinated by the Population Health Research Institute in Ontario, Canada. The study aims to track the progression of chronic diseases of lifestyle in urban and rural participants. This international study is being conducted in 27 countries/territories in various stages of economic development. The study design has been previously described (34-36).

PURE-SA-NWP baseline data were collected in 2005, with follow-up data collection in 2010. The study was conducted in two areas, one urban and one rural. The current study is a prospective (cohort) study over five years and will use the baseline data collected in 2005 and the 2010 follow-up data collection.

4.3.2 Participant recruitment

Participants were recruited from the two communities mentioned above (urban and rural). These communities were further stratified into four geographical areas (two within each community). A household census was completed within 6000 randomly selected households (1500 households from each of the geographical areas). Household census forms were completed by fieldworkers, who were extensively trained in research methodology and ethics.

A family questionnaire and a household questionnaire were included during the household census. This was done to ensure that all members eligible to participate in the study could be identified. Signed, informed consent was obtained from every head of each household questioned. This process was repeated until 1500 households from each specific community could be included.

Using the specific eligibility criteria previously described (34) and the data from the household census, 4000 potential participants were identified as eligible (rural=2000 and urban=2000). The aim was to recruit 1000 rural and 1000 urban participants. Eventually, a total of 2010 participants had a complete set of data after the baseline collection in 2005. In the five years between 2005 and 2010, 217 participants passed away, 188 relocated, 224 refused to continue participation, 93 could not be reached for follow-up, and 134 were removed because of unreliable dietary data (34). This resulted in a total number of 1154 participants in the cohort for the present study.

4.3.3 Measurements

4.3.3.1 Questionnaires

Standardised and semi-structured questionnaires of the international PURE study were used in the PURE-SA-NWP. These included demographic, socio-economic, lifestyle, and physical activity questionnaires (37). Semi-structured questionnaires were administered by fieldworkers selected from the communities that were part of the research. The fieldworkers were trained extensively by a group of experts. All questionnaires were administered in the language the participant preferred (Setswana, Afrikaans or English).

Dietary data were collected using a 145-item culturally sensitive quantified food frequency questionnaire (QFFQ) that recorded foods eaten over the previous 30 days. The specific QFFQ

used in the PURE-SA-NWP study was developed (38) and validated (39) in a similar group of participants from the same culture and demographic region, and has shown good reproducibility (38, 40). A photograph book specifically developed for use in African groups in the North West Province was used in order to assist in estimating portion sizes (41). The portion sizes were reported using household measures, whereafter standard tables were used to convert them to weights (42). Coding of the food intake was done according to the South African Food Composition Database of the South African Medical Research Council (43). Hereafter, it was analysed to obtain the energy and nutrient intake of the participants (9, 40).

The Physical Activity Index questionnaire has been discussed extensively elsewhere (44, 45). It was used to obtain self-reported physical activity data and was developed and validated for the groups studied in the PURE-SA-NWP study (44).

4.3.3.2 Other measurements

In terms of cardiovascular risk, the following measurements were used: total cholesterol, HDL cholesterol, low-density lipoprotein (LDL) cholesterol, BMI, WC, systolic blood pressure, diastolic blood pressure and fasting blood glucose.

Blood sampling, blood pressure measurement and anthropometric measurements were obtained using standard methods, as already described previously (10, 34, 35, 46). The analysis of serum cholesterol and HDL cholesterol was done by means of 2 sequential multiple analyzers [Konelab 20i (Thermo Scientific) and Cobas Integra 400 Plus (Roche)]. Blood glucose level was measured using Vitros DT6011 Chemistry Analyser, an Ortho-Clinical Diagnostics tool (Rochester, New York, USA).

BMI was calculated using the following formula: $\text{weight (kg)} / \text{height (m)}^2$.

4.3.4 Ethical and legal considerations

Ethical approval for the South African leg of the PURE study in the North-West Province was obtained from the Health Research Ethics Committee of the North-West University, Potchefstroom, South Africa (ethics number: 04M10 and NWU-00016-10-A1) and signed informed consent forms were received from all participants. This study complies with the Helsinki Declaration as revised in 2004. Permission for the study to be conducted was granted by the North-West Provincial Department of Health, local government authorities and tribal chiefs in the rural areas. The participants were transported to and from the study sites in both the urban and rural areas. Participants received lunch on site after blood sampling was completed.

4.3.5 Statistical analysis

IBM SPSS statistics version 26 (47) were used as statistical software for this analysis. Paired-samples t-tests were conducted to determine whether the changes from 2005 to 2010 with respect to the following variables were significant; cardiovascular risk factors (HDL cholesterol, low-density lipoprotein (LDL) cholesterol, total cholesterol, BMI, WC, systolic blood pressure, diastolic blood pressure and fasting blood glucose) and dietary fat intakes (total fat, saturated fat, PUFA and MUFA) in each of the four different groups of participants (rural men, rural women, urban men and urban women).

Stepwise linear regression models were fitted on the change in each risk factor from 2005 to 2010 (e.g. change in BMI, change in total cholesterol, etc.) as the dependent variable and the change in fat intakes as independent variables. The following confounding factors were added as independent variables: age; employment status; level of education; tobacco use, alcohol use, physical activity (only 2005 due to poor availability of 2010 data); change in BMI (when it was not the dependent variable); and change in energy intake. Only 2010 data were available for the additional confounding factors, which included aspirin use and use of blood pressure, cholesterol and diabetic medication.

The dichotomous variables (employment, education, tobacco use and alcohol use) had available data for 2005 and 2010. Interpreting changes in these variables called for additional analysis to determine whether a 'positive change' had occurred. A positive change was seen as a person not being employed in 2005, now being employed in 2010, a person smoking in 2005 not smoking in 2010, a person using alcohol in 2005 not using in 2010, and having no education in 2005 to gaining some education in 2010. Age, change in BMI, physical activity and change in energy intake were continuous variables, while the remaining confounding factors were dichotomous (i.e. values 1 and 0).

Contingency (2 x 2) frequency tables and McNemar's test were applied to determine the proportion of participants for whom a positive change occurred from 2005 to 2010. Variables in which more than 10% of the participants experienced a positive change were coded as 1, and as 0 where there was no change or negative change. This dichotomous variable was included in the regression model. In all other cases, the 2005 state (e.g. smoking; not smoking) was included.

This is shown in more detail in Figure 4-1.

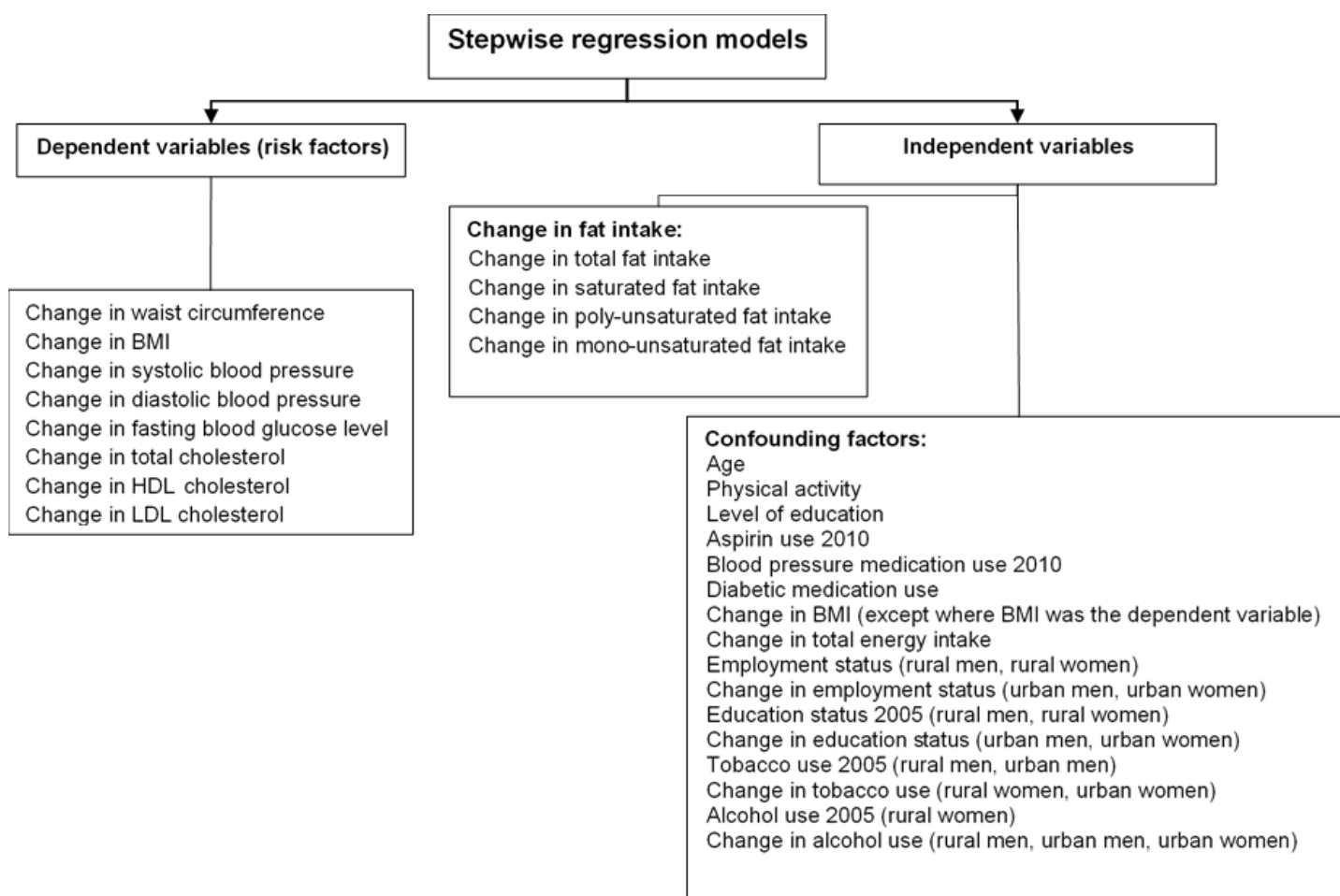


Figure 4-1: Variables entered into the stepwise regression models

Participants with available data for both 2005 and 2010 were included in the analysis and therefore the n-values differ for the various risk factors, nutrients and confounding factors. Participants with a total energy intake of ≥ 30000 kJ or ≤ 3000 kJ were excluded from the analysis. Similar exclusion criteria for analysis based on energy intake were applied in other PURE-SANWP publications (9, 34).

Supplementary Table 1 provides the baseline characteristics of the cohort and those lost to follow-up in 2010. To determine the significance of differences between participants followed up in 2010 and those lost to follow-up, two-samples t-tests were done for continuous variables and the Chi-square (χ^2) test for categorical variables. Phi-values (see Steyn (48)) were reported as effect sizes for locality, gender, education, employment status, alcohol use and tobacco use, while d-values (49) were reported for all other variables. To determine the significance of the differences between the cohort in 2005 and 2010, paired t-tests were used for continuous variables and the χ^2 test for categorical variables. Here, the odds ratio was reported as effect size for education,

employment status, tobacco use and alcohol use, while d-values were reported for all other variables.

A *p*-value of ≤ 0.05 was considered statistically significant.

4.4 Results

4.4.1 Comparison of the cohort with those lost to follow-up between 2005 and 2010

The baseline characteristics are shown in Supplementary Table 1. At baseline in 2005, the group lost to follow-up were younger ($p < 0.001$), with a higher percentage male ($p < 0.001$) and more urban participants ($p < 0.001$). Furthermore, the lost to follow-up group also had a higher percentage of alcohol users ($p = 0.006$) and unemployed participants ($p = 0.045$). In terms of their cardiovascular risk factors at baseline in 2005, this group had a lower mean BMI ($p = 0.010$) and blood glucose concentration ($p = 0.030$), as well as lower total cholesterol ($p < 0.001$), HDL cholesterol ($p = 0.004$) and LDL cholesterol ($p = 0.017$) levels. However, the differences mentioned above were of small effect and can be considered clinically irrelevant.

4.4.2 Changes in cardiovascular risk factors

Paired-samples *t*-tests indicated that there were significant changes in specific cardiovascular risk factors between 2005 and 2010 in the various groups (urban women, urban men, rural women, rural men) and these are briefly summarised below (Supplementary Table 2).

Rural men and women

A significant increase in BMI, systolic and diastolic blood pressure was observed for both rural men and women from 2005 to 2010. A significant decrease in HDL-C was also observed for both rural men and women. There was also an increase in WC and fasting blood glucose in rural women. All these significant differences were of small effect (< 0.3).

Urban men and women

For both urban men and women, an increase was noted in WC from 2005 to 2010. A decrease was observed in HDL-C, LDL-C and total cholesterol for both urban men and women. The urban women also revealed increased BMI and fasting blood glucose levels and decreased systolic and diastolic blood pressure. All these significant differences were of small effect (< 0.3).

These changes in cardiovascular risk factors were further explored through regression models, as depicted in Tables 4-1 to 4-4.

4.4.3 Changes in dietary intake between 2005 and 2010

Changes in dietary intake are presented in Supplementary Table 3.

In 2010, an increase in their overall energy intake was observed for all rural and urban groups when compared with their 2005 baseline values. The same was observed in terms of their total fat intake as well as their intake of saturated fat, MUFA and PUFA. When looking at the percentage energy contribution of each type of fat, rural men and women showed increases in the percentage contribution of total fat as well as the percentage contributions of saturated fat, MUFA and PUFA. In contrast, urban men and women showed increases of only saturated fat and MUFA in their contributions to energy intake.

4.4.4 Regression models for cardiovascular risk

Rural men and women

The regression models for rural men and women are depicted in Table 4-1 and 4-2, respectively. The following observations were made for models that explained more than 10% of the total variance (an adjusted R^2 value of ≥ 0.1):

Rural men

Change in WC:

An increase in the change in WC was associated with a decrease in the change in PUFA intake ($\beta=-0.280$; $p=0.002$) and an increase in the change in MUFA intake ($\beta=0.200$; $p=0.027$). Additionally, it was associated with an increase in the change in BMI ($\beta=0.616$; $p<0.001$) and with smoking in 2005 ($\beta=0.163$; $p=0.004$). The change in BMI explained most of the variance (37.1%, a large effect), while the other variables explained percentage variances of small effect.

Change in total cholesterol:

An increase in the change in total cholesterol was associated with an increase in the change in BMI ($\beta=0.295$; $p<0.001$). Furthermore, an increase in the change in total cholesterol was also associated with a change in alcohol consumption, from drinking alcohol in 2005 to not drinking alcohol in 2010 ($\beta=0.213$; $p=0.004$), with a small %variance. The change in BMI explained most of the variance (8.5%), of medium effect.

Rural women:

Change in WC

An increase in the change in WC was associated with an increase in the change in BMI ($\beta=0.200$; $p<0.001$). In addition, it was also associated with a lower level of education in 2005 ($\beta=0.-0.111$;

$p=0.007$); %variance was small. As in the case of rural men, BMI explained a large amount of variance (40.4%), of large effect.

Table 4-1: Stepwise regression models for rural men for the change (from 2005 - 2010) in each of the cardiovascular risk factors.

Variable	N	Adjusted R ²	B	SE B	β	p-value	Semi-partial correlation	% Variance explained
Change in diastolic blood pressure	169	0.023*						
Change in alcohol consumption (From user in 2005 to non-user in 2010)			-6.209	2.780	-0.170	0.027	-0.170	2.9
Change in WC	177	0.462**						
Change in poly-unsaturated fat intake			-0.072	0.023	-0.280	0.002	-0.173	3.0
Change in mono-unsaturated fat intake			0.050	0.022	0.200	0.027	0.124	1.5
Change in BMI			1.451	0.132	0.616	<0.001	0.609	37.1
Tobacco use 2005			1.395	0.474	0.163	0.004	0.163	2.7
Change in blood glucose concentration	171	0.028*						
Diabetic medication use 2010			1.854	0.764	0.183	0.016	0.183	3.3
Change in total cholesterol	174	0.102**						
Change in BMI			0.233	0.058	0.295	<0.001	0.291	8.5
Change in alcohol consumption (From user in 2005 to non-user in 2010)			0.811	0.278	0.213	0.004	0.210	4.4
Change in LDL cholesterol	173	0.093**						
Change in BMI			0.154	0.044	0.257	0.001	0.253	6.4
Change in alcohol consumption (From user in 2005 to non-user in 2010)			0.696	0.215	0.238	0.001	0.235	5.5
Change in BMI	178	0.024*						
Change in alcohol consumption (From user in 2005 to non-user in 2010)			-0.735	0.350	-0.157	0.037	-0.157	2.5
BMI, Body mass index; LDL, Low-density lipoprotein; HDL, High-density lipoprotein; WC, waist circumference *p-value statistically significant (<0.05); **p-value statistically significant (<0.01) For this group, it was not possible to fit models for Change in systolic blood pressure, Change in HDL cholesterol								

Table 4-2: Stepwise regression models for rural women for the change (from 2005 - 2010) in each of the cardiovascular risk factors.

Variable	N	Adjusted R ²	B	SE B	β	p-value	Semi-partial correlation	% Variance explained
Change in systolic blood pressure	346	0.035**						
Aspirin use 2010			-14.239	3.868	-0.195	<0.001	-0.195	3.8
Change in diastolic blood pressure	346	0.075**						
Change in BMI			0.921	0.239	0.200	<0.001	0.199	4.0
Aspirin usage 2010			-9.028	2.438	-0.192	<0.001	-0.192	3.7
Change in WC	357	0.409**						
Change in BMI			1.450	0.039	0.637	<0.001	0.636	40.4
Level of education 2005			-1.464	0.538	-0.111	0.007	-0.111	1.2
Change in blood glucose concentration	335	0.028**						
Diabetic medication use 2010			1.592	0.485	0.177	0.001	0.177	3.1
Change in total cholesterol	336	0.065**						
Cholesterol medication use 2010			-3.638	1.243	-0.161	0.004	-0.155	2.4
Age 2005			0.017	0.007	0.130	0.014	0.130	1.7
Diabetic medication use 2010			-0.790	0.366	-0.119	0.031	-0.114	1.3
Change in tobacco use (From user in 2005 to non-user in 2010)			0.464	0.221	0.111	0.037	0.111	1.2
Change in HDL cholesterol	337	0.019**						
Change in tobacco use (From user in 2005 to non-user in 2010)			0.279	0.102	0.147	0.007	0.147	2.2
Change in LDL cholesterol	332	0.091**						
Cholesterol medication use 2010			-3.471	1.041	-0.182	0.001	-0.181	3.3
Age 2005			0.017	0.006	0.152	0.004	0.158	2.5
Diabetic medication use 2010			0.029	0.011	0.210	0.010	-0.141	2.0
Change in BMI			0.146	0.074	0.158	0.038	0.114	1.3
Change in BMI	362	0.027**						
Employment status 2005			-2.392	0.927	-0.134	0.010	-0.134	1.8
Change in tobacco use (From user to non-user)			1.098	0.508	0.112	0.031	0.112	1.3
BMI, Body mass index; LDL, Low-density lipoprotein; HDL, High-density lipoprotein; WC, waist circumference *p-value statistically significant (<0.05); **p-value statistically significant (<0.01)								

Table 4-3: Stepwise regression models for urban men for the change (from 2005 - 2010) in each of the cardiovascular risk factors.

Variable	N	Adjusted R ²	B	SE B	β	p-value	Semi-partial correlation	% Variance explained
Change in systolic blood pressure	195	0.036**						
Change in BMI			2.742	0.959	0.201	0.005	0.201	4.0
Change in diastolic blood pressure	195	0.044**						
Change in BMI			1.243	0.597	0.146	0.039	0.146	2.1
Aspirin use 2010			-7.726	3.182	-0.171	0.016	-0.170	2.9
Change in WC	196	0.540**						
Change in BMI			2.388	0.161	0.744	<0.001	0.733	53.7
Change in employment status (From unemployed in 2005 to employed in 2010)			-1.942	0.797	-0.118	0.016	-0.118	1.4
Change in blood glucose concentration	188	0.056**						
Diabetic medication use 2010			-3.038	0.875	-0.247	0.001	-0.247	6.1
Change in total cholesterol	184	0.030**						
Diabetic medication use 2010			-1.262	0.473	-0.191	0.008	-0.191	3.6
Change in BMI			0.129	0.049	0.186	0.010	0.186	3.5
Change in LDL cholesterol	181	0.050**						
Diabetic medication use 2010			-1.240	0.385	-0.234	0.002	-0.234	5.5
Change in BMI	200	0.090**						
Tobacco use 2005			-1.053	0.264	-0.270	<0.001	-0.270	7.3
Physical activity level 2005			0.118	0.052	0.154	0.024	0.154	2.4
BMI, Body mass index; LDL, Low-density lipoprotein; HDL, High-density lipoprotein; WC, waist circumference *p-value statistically significant (<0.05); **p-value statistically significant (<0.01) For this group, it was not possible to fit models for Change in HDL cholesterol								

Urban men and women

The regression models for urban men and women are depicted in Tables 4-3 and 4-4, respectively. The following observations were made for models that explained more than 10% of the total variance (an adjusted R² value of ≥ 0.1):

Urban men

Change in WC

A greater change in WC from 2005 to 2010 was associated with an increase in the change in BMI ($\beta=0.744$; $p<0.001$) from 2005 to 2010. It was further associated with a change in employment status from being unemployed in 2005 to being employed in 2010 ($\beta=-0.118$; $p=0.016$), of small %variance. BMI explained a large amount of variance (53.7%), of large effect.

Urban women:

Change in systolic blood pressure

An increase in the change in systolic blood pressure was associated with an increase in the change in BMI ($\beta=0.133$; $p=0.009$) and an increase in the change in energy intake ($\beta=0.139$; $p=0.006$). In addition, an increase in systolic blood pressure was associated with non-usage of aspirin ($\beta=-0.212$; $p=0.000$) and blood pressure ($\beta=-0.215$; $p=0.000$) medication in 2010. All variables explained small %variance, with the use of blood pressure medication and aspirin in 2010 explaining most of the variance (3.8% each).

Change in diastolic blood pressure

An increase in the change in diastolic blood pressure was associated with an increase in the change in BMI ($\beta=0.169$; $p=0.001$). It was also associated with non-use of blood pressure ($\beta=-0.175$; $p=0.001$) and diabetes medication in 2010 ($\beta=-0.171$; $p=0.001$). Furthermore, it was associated with a change in alcohol consumption, from not drinking alcohol in 2005 to drinking alcohol in 2010 ($\beta=-0.139$; $p=0.006$), and a change in employment, from unemployed in 2005 to employed in 2010 ($\beta=0.119$; $p=0.018$). All variables explained a small %variance, with the use of blood pressure medication in 2010 explaining most of the variance (2.9%)

Change in WC

An increase in the change in WC was associated with an increase in the change in BMI ($\beta=0.571$; $p=0.000$). It was further associated with non-use of cholesterol medication in 2010 ($\beta=-0.129$; $p=0.003$); %variance was small. BMI explained 32.6% of the total variance, of large effect.

Table 4-4: Stepwise regression models for urban women for the change (from 2005 - 2010) in each of the cardiovascular risk factors.

Variable	N	Adjusted R ²	B	SE B	β	p-value	Semi-partial correlation	% Variance explained
Change in systolic blood pressure	343	0.162**						
Aspirin use 2010			-13.799	3.526	-0.212	0.000	-0.194	3.8
Blood pressure medication use 2010			-10.841	2.756	-0.215	0.000	-0.195	3.8
Change in total energy intake			0.001	0.000	0.139	0.006	0.138	1.9
Change in BMI			1.156	0.437	0.133	0.009	0.131	1.7
Change in diastolic blood pressure	343	0.144**						
Blood pressure medication use 2010			-5.087	1.509	-0.175	0.001	-0.169	2.9
Change in BMI			0.846	0.255	0.169	0.001	0.166	2.8
Diabetic medication use 2010			-9.548	2.878	-0.171	0.001	-0.166	2.8
Change in alcohol intake (From user in 2005 to non-user in 2010)			-5.670	2.047	-0.139	0.006	-0.139	1.9
Change in employment status (From unemployed in 2005 to employed in 2010)			5.219	2.203	0.119	0.018	0.119	1.4
Change in WC	343	0.342**						
Change in BMI			1.489	0.114	0.571	<0.001	0.571	32.6
Cholesterol medication use 2010			-6.501	2.203	-0.129	0.003	-0.129	1.7
Change in blood glucose concentration	304	0.053**						
Cholesterol medication use 2010			2.426	0.785	0.174	0.002	0.173	3.0
Change in BMI			0.085	0.041	0.117	0.038	0.116	1.3
Age 2005			0.027	0.011	0.137	0.016	0.135	1.8
Change in total cholesterol	313	0.051**						
Change in BMI			0.086	0.026	0.185	0.001	0.187	3.5
Change in total energy intake			3.121E-05	0.000	0.163	0.003	0.165	2.7
Change in LDL cholesterol	312	0.064**						
Change in BMI			0.049	0.019	0.143	0.010	0.142	2.0
Change in total energy intake			2.167E-05	0.000	0.151	0.006	0.151	2.3
Physical activity level 2005			0.118	0.039	0.167	0.003	0.166	2.8
Change in BMI	350	0.035**						
Physical activity level 2005			0.219	0.104	0.111	0.036	0.110	1.2
Blood pressure medication use 2010			-0.936	0.306	-0.161	0.002	-0.161	2.6
BMI, Body mass index; LDL, Low-density lipoprotein; HDL, High-density lipoprotein; WC, waist circumference								
*p-value statistically significant (<0.05); **p-value statistically significant (<0.01)								
For this group it was not possible to fit models for: Change in HDL cholesterol								

Supplementary Table 1: Baseline characteristics in 2005 of participants followed up in 2010 compared with participants lost to follow-up

Variable	Baseline data in 2005 of subjects followed up in 2010				Baseline data in 2005 of subjects lost to follow-up in 2010				p-value ¹	ES ²	Cohort data of participants in 2010			p-value ³	ES ⁴
	n	Mean	%	SD	n	Mean	%	SD			n	Mean	SD		
Age (y)	1193	50.6		10.2	666	48.8		10.7	<0.001	0.16	-			-	
Sex (men) %	1192		34.1		666		43.2		<0.001	0.09	-			-	
Locality (rural) %	1192		52.0		666		40.5		<0.001	-0.11	-			-	
Education attainment (no formal education) %	1159		33.0		792		36.5		NS	0.04	1087	99/105 ⁵		0.726	1.06
Employment status (unemployed) %	1192		90.5		666		87.5		0.045	-0.05	1143	49/121		<0.001	2.47
Tobacco use (non-user) %	1186		45.6		814		41.8		NS	-0.04	1109	110/147		0.025	1.34
Alcohol use (non-user) %	1184		58.4		813		52.2		0.006	-0.06	1079	149/95		0.002	0.66
PA Index (Physical activity)	1148	7.3		1.9	784	7.2		2.0	NS	0.39					
WC (cm)	1185	80.2		12.8	653	79.2		13.3	NS	0.07	1140	81.9	13.0	<0.001	
BMI (kg/m ²)	1192	25.0		7.0	663	24.2		7.0	0.010	0.12	1149	25.7	7.4	<0.001	0.09
Systolic blood pressure (mmHg)	1185	133.5		26.7	662	134.3		26.0	NS	-0.03	1109	135.2	24.2	0.022	0.07
Diastolic blood pressure (mmHg)	1185	87.8		13.9	662	87.7		15.6	NS	0.00	1109	88.4	13.6	NS	0.04
Blood glucose concentration (mmol/l)	1102	5.0		1.8	602	4.9		1.1	0.030	0.10	1052	5.3	1.9	<0.001	0.14
Total cholesterol (mmol/l)	1121	5.1		1.3	628	4.9		1.4	0.001	0.16	1064	4.9	1.2	<0.001	-0.15
HDL cholesterol (mmol/l)	1121	1.5		0.6	628	1.4		0.7	0.004	0.10	1067	1.4	0.6	<0.001	-0.19
LDL cholesterol (mmol/l)	1112	3.0		1.1	625	2.8		1.3	0.017	0.11	1053	2.9	1.1	0.003	-0.08
Saturated fat intake (g)	1192	12.5		8.8	666	12.9		9.0	NS	-0.41	1154	21.6	14.1	<0.001	1.03
Mono-unsaturated fat intake (g)	1192	13.9		10.3	666	14.3		10.2	NS	-0.04	1154	23.4	16.4	<0.001	0.92
Poly-unsaturated fat intake (g)	1192	15.8		10.6	666	15.7		9.4	NS	0.02	1154	22.8	16.1	<0.001	0.65
Cholesterol intake (g)	1192	194.9		159.8	666	195.6		145.2	NS	0.00	1154	337.1	311.2	<0.001	0.88
Energy intake (MJ/day)	1192	8.2		3.6	666	8.2		3.7	NS	0.00	1154	11.3	5.1	<0.001	0.86

BMI, Body mass index; LDL, Low-density lipoprotein; HDL, High-density lipoprotein; WC, waist circumference; NS, not significant

¹ P-value for significance of differences between subjects followed up in 2010 and those lost to follow-up. For categorical variables: χ^2 test (in bold); continuous variables: two-sample t-test.

² ES (effect size): For Sex - Alcohol use (in bold): phi-values; for Age, PA Index - Energy intake: d-values.

³ P-value for significance of differences between the cohort in 2005 and 2010. For categorical variables: χ^2 test (in bold); continuous variables: paired t-test.

⁴ ES: For Education attainment - Alcohol use, Odds ratio (in bold); PA Index - Energy intake: d-values.

⁵ 99/105: 99 persons who attained education in 2005, but had not attained it in 2010; 105 persons who had not attained education in 2005, but attained it in 2010.

Supplementary Table 2: Cardiovascular risk factors in participants at baseline and follow-up by urbanisation and gender

	Rural men						Urban men				
Cardiovascular risk factors	n	2005	2010	p-value ¹	ES ²	n	2005	2010	p-value ¹	ES ³	
		Mean	Mean				Mean	Mean			
		(SD)	(SD)				(SD)	(SD)			
WC (cm)	184	77.49 (10.80)	77.75 (10.27)	NS	0.02	197	76.41 (9.94)	77.93 (11.04)	0.001	0.15	
Fasting blood glucose (mmol/L)	178	4.87 (0.97)	4.92 (1.30)	NS	0.05	190	4.93 (2.31)	4.9 (1.04)	NS	-0.01	
BMI (kg/m ²)	186	20.97 (4.41)	21.38 (4.54)	0.001	0.09	200	20.92 (3.99)	21.06 (4.53)	NS	0.04	
Systolic blood pressure (mm Hg)	177	133.38 (24.47)	139.28 (25.73)	0.002	0.24	196	137.99 (23.54)	138.47 (23.62)	NS	0.02	
Diastolic blood pressure (mm Hg)	177	85.41 (15.08)	88.01 (13.23)	0.014	0.17	196	88.08 (13.87)	88.32 (13.53)	NS	0.02	
HDL (mmol/L)	183	1.55 (0.62)	1.40 (0.54)	0.001	-0.24	188	1.69 (0.67)	1.57 (0.71)	0.037	-0.18	
LDL (mmol/L)	181	2.77 (1.12)	2.71 (1.15)	NS	-0.05	183	2.72 (1.17)	2.41 (0.98)	<0.001	-0.26	
Serum total cholesterol (mmol/L)	183	4.83 (1.31)	4.70 (1.19)	NS	-0.10	186	5.02 (1.34)	4.58 (1.13)	<0.001	-0.33	
	Rural women						Urban women				
WC (cm)	402	80.67 (13.71)	81.35 (12.9)	0.037	0.05	348	83.45 (13.06)	86.89 (13.94)	<0.001	0.26	
Fasting blood glucose (mmol/L)	375	5.01 (1.66)	5.4 (2.27)	<0.001	0.23	309	5.24 (2.03)	5.6 (2.08)	0.003	0.18	
BMI (kg/m ²)	421	26.27 (6.97)	27.25 (7.28)	<0.001	0.14	355	28.25 (7.35)	28.8 (7.75)	0.001	0.07	
Systolic blood pressure (mm Hg)	390	127.68 (21.27)	132.68 (22.88)	0.001	0.24	346	137.61 (24.47)	134.04 (24.64)	0.010	-0.15	
Diastolic blood pressure (mm Hg)	390	86.77 (13.71)	88.76 (12.74)	0.014	0.15	346	90.12 (13.63)	88.23 (14.61)	0.017	-0.14	
HDL (mmol/L)	378	1.48 (0.56)	1.35 (0.52)	0.001	-0.23	318	1.52 (0.58)	1.44 (0.60)	0.019	-0.14	
LDL (mmol/L)	372	3.05 (1.12)	3.1 (1.07)	NS	0.04	317	3.1 (1.15)	2.93 (1.15)	0.004	-0.15	
Serum total cholesterol (mmol/L)	377	5.1 (1.30)	5.06 (1.14)	NS	-0.03	323	5.28 (1.34)	5.02 (1.25)	<0.001	-0.19	
BMI, Body mass index; LDL, Low-density lipoprotein; HDL, High-density lipoprotein; WC, waist circumference; NS, not significant Statistical test: Paired samples t-test p-value statistically significant (<0.05). ¹ P value for significance of differences in cardiovascular risk factors between 2005 and 2010 for rural men/women or urban men/women ² ES (effect size): d-values											

Supplementary Table 3: Mean intake of energy and fat of participants at baseline and follow-up, by urbanisation and gender

	Rural men (n=186)				Urban men (n=202)			
ENERGY AND FAT	2005	2010	p-value ¹	ES ²	2005	2010	p-value ¹	ES ²
	Mean	Mean			Mean	Mean		
	(SD)	(SD)			(SD)	(SD)		
Energy intake (MJ)	7.6 (3.33)	10.2 (5.00)	<0.001	0.87	10.22 (34.36)	14.23 (50.51)	<0.001	0.97
Total fat (g)	35.42 (15.91)	64.33 (44.54)	<0.001	1.82	69.27 (9.36)	101.05 (13.63)	<0.001	0.92
Saturated fat (g)	7.78 (4.52)	17.82 (12.96)	<0.001	2.22	17.22 (11.28)	26.90 (17.17)	<0.001	1.03
MUFA (g)	8.39 (5.03)	18.66 (16.92)	<0.001	2.04	19.80 (10.60)	31.14 (17.94)	<0.001	1.00
PUFA (g)	11.60 (6.24)	18.27 (14.48)	<0.001	1.07	19.94 (10.60)	28.42 (17.94)	<0.001	0.80
% of TE from total fat	18.71 (7.09)	23.50 (8.51)	<0.001	0.68	25.88 (6.40)	26.92 (7.30)	NS	0.16
% of TE from saturated fat	4.19 (2.38)	6.56 (2.98)	<0.001	0.99	6.46 (2.11)	7.24 (2.32)	<0.001	0.37
% of TE from MUFA	4.44 (2.47)	6.68 (3.44)	<0.001	0.90	7.39 (2.54)	8.28 (2.89)	<0.001	0.35
% of TE from PUFA	6.07 (2.68)	6.60 (3.58)	0.015	0.20	7.50 (2.53)	7.51 (3.22)	NS	<0.00
	Rural women (n=411)				Urban women (n=355)			
Energy intake (MJ)	6.56 (2.23)	10.00 (4.84)	<0.001	1.51	9.35 (3.76)	12.12 (5.19)	<0.001	0.74
Total fat (g)	35.00 (17.16)	66.53 (42.27)	<0.001	1.84	69.87 (34.20)	90.79 (49.85)	<0.001	0.61
Saturated fat (g)	7.73 (4.44)	18.71 (14.34)	<0.001	2.47	17.93 (9.18)	24.84 (14.40)	<0.001	0.75
MUFA (g)	8.21 (4.75)	18.4 (13.99)	<0.001	2.14	20.49 (10.87)	28.16 (16.54)	<0.001	0.73
PUFA (g)	11.9 (8.51)	20.26 (15.07)	<0.001	0.98	20.49 (11.88)	26.00 (16.81)	<0.001	0.46
% of TE from total fat	20.46 (6.79)	24.75 (8.30)	<0.001	0.63	28.20 (6.71)	27.90 (7.04)	NS	-0.04
% of TE from saturated fat	4.53 (2.23)	6.88 (3.16)	<0.001	1.05	7.30 (2.30)	7.68 (2.53)	0.023	0.17
% of TE from MUFA	4.80 (2.24)	6.77 (3.30)	<0.001	0.88	7.29 (2.30)	8.65 (2.84)	0.008	0.19
% of TE from PUFA	6.85 (3.27)	7.54 (3.81)	0.004	0.21	8.17 (2.64)	7.85 (2.91)	NS	-0.12
TE, total energy; MUFA, mono-unsaturated fatty acids, PUFA, poly-unsaturated fatty acids Statistical test: Paired samples t-test p-value statistically significant (<0.05). ¹ P value for significance of differences in cardiovascular risk factors between 2005 and 2010 for rural men/women or urban men/women ² ES (effect size): d-values								

4.5 Discussion

This study aimed to investigate the association between the change in dietary fat intake and the change in CVD risk factors in black South Africans in the North West province over five years from 2005 to 2010. Our main findings indicate that, despite the significant differences observed in energy and dietary fat intake over 5 years in all groups, observed changes in dietary fat intake were, overall, not strongly associated with the observed changes in cardiovascular risk factors. In rural men, an increase in the change in WC was associated with a decrease in the change in PUFA intake and an increase in the change in MUFA intake for the same period, with the percentage variance explained, small (3% and 1.5% respectively).

An increase in BMI, systolic and diastolic blood pressure and a decrease in HDL-C were observed in rural men from 2005 to 2010. Similarly, the same observations were made for the risk factors for rural women, with an increase in WC, fasting blood glucose, BMI, SBP, DBP and a decrease in HDL-C being observed. The observed changes in cardiovascular risk factors within the rural group is indicative of an increased risk of cardiovascular diseases and support the previous findings by Wentzel-Viljoen and co-workers (9) that the nutrition transition is still taking place at a rapid pace within the rural groups. The observed increase in cardiovascular disease risk is in line with what is expected within a group in transition (4, 10).

Results from urban men and women show a decrease in HDL-C, LDL-C and serum total cholesterol, with an increase in WC from 2005 to 2010. The increases observed in dietary fat intake as well as in WC gave way to the question of the use of cholesterol medication and whether it has a potential influence on these results within the urban groups. Furthermore, Richter (50) has previously reported that, at baseline, 42.8% of rural participants and 47% of urban participants of the PURE-SA-NWP had elevated total cholesterol levels (≥ 5.0 mmol/L), while 52.2% and 55.4% presented with elevated LDL-C levels (≥ 3.0 mmol/L). For these reasons, medication usage was included as a possible confounding factor in the regression analyses that are discussed shortly.

Dietary energy intake, PUFA, MUFA and saturated fat intake increased in all groups from 2005 to 2010. Global estimates of the Food and Agriculture Organisation (FAO) show that the per capita energy consumption of Sub-Saharan African countries has increased from 8530 kJ/person/day to 9400 kJ/person/day over the course of 35 years (51). The FAO also reports a projected increase to around 10600 kJ/person/day by the year 2030. Similar findings with regard to dietary fat intake have been reported in India. Dietary fat intake in both rural and urban Indian groups was reported to have increased significantly over a time period of more than 30 years, from 1973 to 2005 (52).

The rural groups had increased proportions of all types of fat (expressed as a percentage of total energy). For the urban groups, smaller increases in fat proportion have been noted, with only the proportion of saturated fat and MUFA increasing. Despite the changes observed, the rural as well as urban groups were well within the recommendations set out in the South African Food-Based Dietary Guidelines (FBDG) ("Eat and use the right type of fats and oils in moderation") (53), as well as WHO recommendations (saturated fat <10% of total energy, PUFA = 6-11% of total energy) (54).

Numerous studies have investigated the association between dietary fat intake and CVD risk, to the extent that robust systematic reviews and meta-analysis are available (27, 28, 55, 56). These studies have shown that substituting dietary saturated fat with PUFA intake reduced cardiovascular events (27), the incidence of CVD (56) as well all-cause mortality in US adults even when substituting dietary saturated fat with PUFA and MUFA (28). While robust evidence exists for developed countries, evidence in developing countries is scarce. Recent evidence from Tanzania indicated that participants who consumed palm oil (high in saturated fat) had higher mean total cholesterol levels than those who used vegetable oil instead (high in polyunsaturated fat) (57). Based on convincing international evidence of the effect the type of fat on CVD risk, the South-African dietary guideline on the importance of the quality or type of fat in the diet recommends replacing animal and plant sources of saturated fatty acids with PUFAs (58) and this shows that this is an element not ignored in developing countries.

The results of this study indicate that among rural men, an increased WC was associated with a decrease in PUFA intake and an increase in MUFA intake. In a meta-analysis, it was shown that individuals consuming fish and omega 3 PUFAs (daily dosage varied from 157 to 3 360 mg) lost 0,59 kg more body weight than the control groups, while also decreasing WC by 0,81 cm, as opposed to the control groups (59). The same relationship between the change in WC and the change in MUFA intake was, however, not observed in this study. Instead, the increase in the change in WC was associated with an increase in the change in MUFA intake. In China, MUFA from plant sources was associated with lower mortality, while MUFA from animal sources showed no associations (60). Two large prospective studies among US groups showed that the increased intake of MUFA from animal sources was associated with more significant weight gain (61) and higher mortality than MUFA from plant sources (62). Wentzel-Viljoen *et al.* (63) have previously reported a significant increase in the intake of food from animal sources (from 48–97g/day) from 2005 to 2010 for the rural men in the current study group. Unfortunately, no data on the MUFA sources were available to give more insight into this association. Miller, Sorkin (64) found PUFA to be the preferred unsaturated fat in managing the metabolic syndrome when compared with

MUFA. We found no associations between the change in WC and the changes in either saturated fat or total fat intake of rural men.

For rural women, urban men and urban women, no associations were found between any of the risk factors and PUFA, MUFA, saturated fatty acids or total fat intake. However, the confounding factors instead explained the changes in some of the CVD risk factors. BMI emerged as a major role player in CVD risk when entered as a confounding factor in the analysis of other risk factors. Findings of a pooled analysis (using data from 10 large US cohorts) by Khan, Ning (65) support these results, where BMI was associated with an increased risk of developing CVD. Our data indicated that, across all groups of gender and locality (rural men, rural women, urban men and urban women), the change in all risk factors (where the regression models explained more than 10% of variance) was associated with a change in BMI.

As can be expected, the change in WC was associated with an increase in the change in BMI for all groups, with BMI explaining a large amount of variance in the change in WC (>25%). BMI and WC were previously shown to be decisively correlated with each other (66, 67).

An increase in the change in both systolic and diastolic blood pressure of urban women was associated with an increase in BMI. Women in Bangladesh, especially those living in urban areas, were shown to have a significantly higher risk of hypertension when overweight or obese (68). In Iranian adults, BMI was also strongly associated with blood pressure, with obese participants (BMI >30) being three times more likely to have elevated blood pressure (69). This association has also been documented by various studies in other developing countries (70-76).

Our results further showed that an increase in the change in the total cholesterol of rural men was associated with an increase in the change in BMI. Overweight and obese rural adults in India also reported having higher total cholesterol levels in comparison with participants of normal weight (77). Khan *et al.* (65), further found that BMI was associated with an increased risk of developing CVD.

In terms of other confounding factors, the percentages variance explained remained small (<5%). Of these, most variance was explained by associations between the change in systolic and diastolic blood pressure and medication use of urban women. In addition to BMI, an increase in the change in the systolic blood pressure was associated with an increase in total energy intake, the non-use of blood pressure medication and the non-use of aspirin. The change in their diastolic blood pressure was associated with the non-use of blood pressure medication and the non-use of diabetic medication. The association with non-use of blood pressure medication is expected. The reasons why these associations are not present in any of the other groups could be related

to the fact that medical services were more accessible in the urban communities of the PURE-SA-NWP study, in comparison with their rural counterparts. Women were also more likely than men to go to the clinic or doctor to receive treatment (78).

In addition to the association with change in BMI, the increase in the change in total cholesterol of rural men was also associated with a change in alcohol consumption, from drinking alcohol in 2005 to not drinking alcohol in 2010 (explaining 4.4% of variance). This association is unexplained, and to the authors' knowledge, has not been investigated.

Limitations and strengths of this study

One of the limitations of this study is that it was conducted in only one of the nine provinces of South Africa and is therefore not representative of the entire South African population. The use of QFFQs as a dietary data collection method may also be a limitation as it has been criticised owing to reservations regarding its level of accuracy (79, 80). The QFFQ used in the THUSA study was found to under-report energy and fat intakes in comparison with 7-day weighted records (39). It does, nevertheless, remain the preferred dietary epidemiological method for evaluating the associations between health outcomes and dietary intake (40, 81). Another limitation is the relatively small sample sizes of both rural and urban men.

The specific QFFQ used in the PURE-SA-NWP study was developed (39) and validated (38) in a similar group of participants from the same culture and demographic region, and has shown good reproducibility (38, 40). Additionally, the same team of fieldworkers, trained and re-trained by the same team of professionals, administered the questionnaires for both the baseline as well as the follow-up data collections. For these reasons, we regard the observed dietary changes as accurate and as the result of lifestyle, behavioural or environmental influences and not the result of methodological error.

4.6 Conclusion

Our findings indicate that a decrease in PUFA intake may increase the CVD risk of the rural men by increasing their WC. Our findings indicate that an increase in BMI rather than changes in dietary fat intake may be associated with an unfavourable change in CVD risk, with BMI emerging as a major determinant of various CVD risk factors. In terms of rural men our results showed that a decrease in PUFA intake over the years may increase CVD risk in that it is associated with a higher WC. Our results also point toward PUFA as the preferred fat substitute as opposed to MUFA for WC decline. For rural women, urban men, and urban women, there were no associations between the change in the different CVD risk factors and any of the changes in fat intakes. Instead, the variance was explained rather by various confounding factors of which BMI

was the most notable. More research is warranted to determine the association between the source of MUFA intake (plant vs animal) and CVD risk.

4.7 Acknowledgements:

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None.

4.10 Authorship:

S. Lee: Responsible for the conception and design, analysis and interpretation of the data and writing of the manuscript.

C. Taljaard-Krugell, C. Botha-Ravyse and T. van Zyl: Made contributions to the design, analysis and interpretation of the data and critically revised the manuscript for important intellectual content.

E. Wentzel-Viljoen, L. Kruger (National Leader: South Africa): Contributed to the interpretation of the data and critically revised the manuscript for important intellectual content.

F. Steyn: Responsible for the statistical analysis of the data and made contributions to the interpretation of the data.

4.11 References

1. MacIntyre U, Kruger H, Venter C, Vorster H. Dietary intakes of an African population in different stages of transition in the North West Province, South Africa: the THUSA study. *Nutr Res.* 2002;22(3):239-56.
2. Popkin BM. An overview on the nutrition transition and its health implications: the Bellagio meeting. *Public Health Nutr.* 2002;5(1A):93-103.
3. Ronto R, Wu JH, Singh GM. The global nutrition transition: trends, disease burdens and policy interventions. *Public Health Nutr.* 2018;21(12):2267-70.
4. Bourne LT, Lambert EV, Steyn K. Where does the black population of South Africa stand on the nutrition transition? *Public Health Nutr.* 2002;5(1a):157-62.
5. MacIntyre UE, Venter CS, Kruger A, Serfontein M. Measuring micronutrient intakes at different levels of sugar consumption in a population in transition: the Transition and Health during Urbanisation in South Africa (THUSA) study. *South Afr J Clin Nutr.* 2012;25(3):122-30.
6. Vorster H. The emergence of cardiovascular disease during urbanisation of Africans. *Public Health Nutr.* 2002;5(1a):239-43.
7. Vorster HH, Kruger A, Margetts BM. The nutrition transition in Africa: can it be steered into a more positive direction? *Nutrients.* 2011;3(4):429-41.
8. Vorster HH, Venter CS, Wissing MP, Margetts BM. 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 Nutr.* 2005;8(05):480-90.
9. Wentzel-Viljoen E, Lee S, Laubscher R, Vorster HH. Accelerated nutrition transition in the North West Province of South Africa: results from the Prospective Urban and Rural Epidemiology (PURE-NWP-SA) cohort study, 2005 to 2010. *Public Health Nutr.* 2018;21(14):2630-41.
10. Pisa P, Behanan R, Vorster H, Kruger A. Social drift of cardiovascular disease risk factors in Africans from the North West Province of South Africa: the PURE study. *Cardiovasc J Afr.* 2012;23(7):371.
11. Yuyun MF, Sliwa K, Kengne AP, Mocumbi AO, Bukhman G. Cardiovascular Diseases in Sub-Saharan Africa Compared to High-Income Countries: An Epidemiological Perspective. *Glob Heart.* 2020;15(1):15.
12. Benziger CP, Roth GA, Moran AE. The global burden of disease study and the preventable burden of NCD. *Glob Heart.* 2016;11(4):393-7.
13. Dalal S, Beunza JJ, Volmink J, Adebamowo C, Bajunirwe F, Njelekela M, et al. Non-communicable diseases in sub-Saharan Africa: what we know now. *Int J Epidemiol.* 2011;40(4):885-901.
14. Statistics-SA. Mortality and causes of death in South Africa, 2017: Findings from death notification Statistics-SA; 2020 [Available from: <http://www.statssa.gov.za/publications/P03093/P030932016.pdf>].

15. WHO. Fact sheet: Noncommunicable diseases Geneva: WHO; 2018 [Available from: <https://www.who.int/en/news-room/fact-sheets/detail/noncommunicable-diseases>.
16. WHO. Hypertension: Fact Sheet Geneva: World Health Organisation; 2019 [updated 13 September 2019. Available from: <https://www.who.int/news-room/fact-sheets/detail/hypertension>.
17. WHO. Diabetes: Fact sheet Geneva: World health organization; 2018 [updated February 2018. Available from: <http://www.who.int/mediacentre/factsheets/fs312/en/>.
18. WHO. Physical activity: Fact Sheet Geneva: World HEalth Organisation; 2018 [updated February 2018. Available from: <https://www.who.int/news-room/fact-sheets/detail/physical-activity>.
19. WHO. Obesity and overweight: Fact Sheet Geneva: Word Health Organisation; 2018 [updated February 2018. Available from: <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>.
20. NDoH, Statistics-SA, SA-MRC, ICF. South African Demographic and Health Survey 2016: Key Indicators. In: Statistics-SA, editor. Pretoria, South Africa: Statistics South Africa; 2017.
21. Hulsegge G, Looman M, Smit HA, Daviglus ML, van der Schouw YT, Verschuren WM. Lifestyle changes in young adulthood and middle age and risk of cardiovascular disease and all-cause mortality: The Doetinchem Cohort Study. *J. Am. Heart Assoc.* 2016;5(1):e002432.
22. Chen M, Li Y, Sun Q, Pan A, Manson JE, Rexrode KM, et al. Dairy fat and risk of cardiovascular disease in 3 cohorts of US adults. *Am. J. Clin. Nutr.* 2016;104(5):1209-17.
23. Guasch-Ferré M, Babio N, Martínez MA, Corella D, Ros E, Martín S, et al. Dietary fat intake and risk of cardiovascular disease and all-cause mortality in a population at high risk of cardiovascular disease. *Am J Clin Nutr.* 2015;102:1563-73.
24. Li Y, Hruby A, Bernstein AM, Ley SH, Wang DD, Chiuve SE, et al. Saturated fats compared with unsaturated fats and sources of carbohydrates in relation to risk of coronary heart disease: a prospective cohort study. *J Am Coll Cardiol.* 2015;66(14):1538-48.
25. Zong G, Li Y, Wanders AJ, Alsema M, Zock PL, Willett WC, et al. Intake of individual saturated fatty acids and risk of coronary heart disease in US men and women: two prospective longitudinal cohort studies. *BMJ.* 2016;355:i5796.
26. Schwab U, Lauritzen L, Tholstrup T, Haldorsson TI, Riserus U, Uusitupa M, et al. Effect of the amount and type of dietary fat on cardiometabolic risk factors and risk of developing type 2 diabetes, cardiovascular diseases, and cancer: a systematic review. *Food Nutr Res.* 2014;58(1):25145.
27. Hooper L, Martin N, Abdelhamid A, Davey Smith G. Reduction in saturated fat intake for cardiovascular disease. *Cochrane Database Syst Rev.* 2015(6).
28. Ricci C, Baumgartner J, Zec M, Kruger HS, Smuts CM. Type of dietary fat intakes in relation to all-cause and cause-specific mortality in US adults: An iso-energetic substitution analysis from the American National Health and Nutrition Examination Survey linked to the US mortality registry. *Br J Nutr.* 2018;119(4):456-63.

29. Kim Y, Je Y, Giovannucci EL. Association between dietary fat intake and mortality from all-causes, cardiovascular disease, and cancer: A systematic review and meta-analysis of prospective cohort studies. *Clin Nutr*. 2020.
30. Tindall AM, Petersen KS, Skulas-Ray AC, Richter CK, Proctor DN, Kris-Etherton PM. Replacing Saturated Fat With Walnuts or Vegetable Oils Improves Central Blood Pressure and Serum Lipids in Adults at Risk for Cardiovascular Disease: A Randomized Controlled-Feeding Trial. *J. Am. Heart Assoc*. 2019;8(9):e011512.
31. Ho FK, Gray SR, Welsh P, Petermann-Rocha F, Foster H, Waddell H, et al. Associations of fat and carbohydrate intake with cardiovascular disease and mortality: prospective cohort study of UK Biobank participants. *BMJ*. 2020:m688.
32. Dolman RC, Wentzel-Viljoen E, Jerling JC, Feskens EJ, Kruger A, Pieters M. The use of predefined diet quality scores in the context of CVD risk during urbanization in the South African Prospective Urban and Rural Epidemiological (PURE) study. *Public Health Nutr*. 2014;17(8):1706-16.
33. Richter M, Baumgartner J, Wentzel-Viljoen E, Smuts CM. Different dietary fatty acids are associated with blood lipids in healthy South African men and women: The PURE study. *Int J Cardiol*. 2014;172(2):368-74.
34. Vorster HH, Kruger A, Wentzel-Viljoen E, Kruger HS, Margetts BM. Added sugar intake in South Africa: findings from the Adult Prospective Urban and Rural Epidemiology cohort study. *Am. J. Clin. Nutr*. 2014;99(6):1479-86.
35. Schutte AE, Schutte R, Huisman HW, Van Rooyen JM, Fourie CM, Malan NT, et al. Are behavioural risk factors to be blamed for the conversion from optimal blood pressure to hypertensive status in Black South Africans? A 5-year prospective study. *Int J Epidemiol*. 2012;41(4):1114-23.
36. Sotunde O, Kruger H, Wright HH, Havemann-Nel L, Kruger I, Wentzel-Viljoen E, et al. Lean mass appears to be more strongly associated with bone health than fat mass in urban black South African women. *J Nutr Health Aging*. 2015;19(6):628-36.
37. Corsi DJ, Subramanian S, Chow CK, McKee M, Chifamba J, Dagenais G, et al. Prospective Urban Rural Epidemiology (PURE) study: baseline characteristics of the household sample and comparative analyses with national data in 17 countries. *Am Heart J*. 2013;166(4):636-46.
38. MacIntyre U, Venter C, Vorster H. A culture-sensitive quantitative food frequency questionnaire used in an African population: 1. Development and reproducibility. *Public Health Nutr*. 2001;4(1):53-62.
39. MacIntyre U, Venter C, Vorster H. A culture-sensitive quantitative food frequency questionnaire used in an African population: 2. Relative validation by 7-day weighed records and biomarkers. *Public Health Nutr*. 2001;4(1):63-71.
40. Wentzel-Viljoen E, Laubscher R, Kruger A. Using different approaches to assess the reproducibility of a culturally sensitive quantified food frequency questionnaire. *South Afr J Clin Nutr*. 2011;24(3):143-8.
41. Venter C, MacIntyre U, Vorster H. The development and testing of a food portion photograph book for use in an African population. *J Hum Nutr Diet*. 2000;13(3):205-18.

42. Langenhoven, M., Conradie, P., Wolmarans, P. & Faber, M. 1991. MRC food quantities manual. Cape Town: South African Medical Research Council, 2.
43. Wolmarans P, Danster N, Dalton A, Rossouw K, Schönfeldt H. Condensed food composition tables for South Africa. Cape Town: Medical Research Council. 2010:1-126.
44. Kruger H, Venter C, Steyn H. A standardised physical activity questionnaire for a population in transition: the THUSA study. *Afr J Phys Health Educ Recreat Dance*. 2000;6:54-64.
45. Oyeyemi AL, Moss SJ, Monyeki MA, Kruger HS. Measurement of physical activity in urban and rural South African adults: a comparison of two self-report methods. *BMC Public Health*. 2016;16(1):1004.
46. Lohman TG, Roche AF, Martorell R. Anthropometric standardization reference manual: Human kinetics books; 1988.
47. Statistics IS. IBM SPSS Statistics software: SPSS Statistics V26. 2019.
48. Steyn H. Manual for the determination of effect size indices and practical significance. Potchefstroom, North-West University. 2012.
49. Cohen J. Statistical power analysis for the behavioral sciences, 2nd edn. Á/L. Erbaum Press, Hillsdale, NJ, USA; 1988.
50. Richter M. Dietary fat intake and blood lipid profiles of South African communities in transition in the North–West Province: the PURE study: North-West University; 2010.
51. Alexandratos N, Bruinsma J. World agriculture towards 2030/2050: the 2012 revision. ESA Working paper FAO, Rome; 2012.
52. Misra A, Singhal N, Sivakumar B, Bhagat N, Jaiswal A, Khurana L. Nutrition transition in India: Secular trends in dietary intake and their relationship to diet-related non-communicable diseases. *J Diabetes*. 2011;3(4):278-92.
53. Smuts CM, Wolmarans P. The importance of the quality or type of fat in the diet: a food-based dietary guideline for South Africa. *South Afr J Clin Nutr*. 2013;26(3):S87-S99.
54. WHO. Draft guidelines on saturated fatty acid and trans-fatty acid intake for adults and children. WHO [https://extranet.who.int/dataform/upload/surveys/666752/files/Draft%20WHO%20SFA-TFA%20guidelines_04052018%20Public%20Consultation \(1\).pdf](https://extranet.who.int/dataform/upload/surveys/666752/files/Draft%20WHO%20SFA-TFA%20guidelines_04052018%20Public%20Consultation%20(1).pdf). 2018.
55. Hooper L, Summerbell C, Thompson R, Sills D, Roberts F, Moore H, et al. Reduced or modified dietary fat for preventing cardiovascular disease *Cochrane Database. Syst Rev*. 2012;16(5).
56. Sacks FM, Lichtenstein AH, Wu JH, Appel LJ, Creager MA, Kris-Etherton PM, et al. Dietary fats and cardiovascular disease: a presidential advisory from the American Heart Association. *Circulation*. 2017;136(3):e1-e23.
57. Kakarmath SS, Zack RM, Leyna GH, Fahimi S, Liu E, Fawzi WW, et al. Dietary determinants of serum total cholesterol among middle-aged and older adults: a population-based cross-sectional study in Dar es Salaam, Tanzania. *BMJ Open*. 2017;7(5):e015028.

58. Vorster HH, Badham J, Venter C. An introduction to the revised food-based dietary guidelines for South Africa. *South Afr J Clin Nutr.* 2013;26(3):S5-S12.
59. Bender N, Portmann M, Heg Z, Hofmann K, Zwahlen M, Egger M. Fish or n3-PUFA intake and body composition: a systematic review and meta-analysis. *Obes Rev.* 2014;15(8):657-65.
60. Mao L, Zhang Y, Wang W, Zhuang P, Wu F, Jiao J. Plant-sourced and animal-sourced monounsaturated fatty acid intakes in relation to mortality: a prospective nationwide cohort study. *Eur J Nutr.* 2020;59(5):1989-98.
61. Liu X, Li Y, Tobias DK, Wang DD, Manson JE, Willett WC, et al. Changes in types of dietary fats influence long-term weight change in US women and men. *J. Nutr.* 2018;148(11):1821-9.
62. Guasch-Ferré M, Zong G, Willett WC, Zock PL, Wanders AJ, Hu FB, et al. Associations of Monounsaturated Fatty Acids From Plant and Animal Sources With Total and Cause-Specific Mortality in Two US Prospective Cohort Studies. *Circ. Res.* 2019;124(8):1266-75.
63. Wentzel-Viljoen E, Laubscher R, Vorster HH. Changes in food intake from 2005 to 2010 by a cohort of black rural and urban African men and women in the North West Province of South Africa: the PURE-NWP-SA study. *Public Health Nutr.* 2018;21(16):2941-58.
64. Miller M, Sorkin JD, Mastella L, Sutherland A, Rhyne J, Donnelly P, et al. Poly is more effective than monounsaturated fat for dietary management in the metabolic syndrome: The muffin study. *J Clin Lipidol.* 2016;10(4):996-1003.
65. Khan SS, Ning H, Wilkins JT, Allen N, Carnethon M, Berry JD, et al. Association of Body Mass Index With Lifetime Risk of Cardiovascular Disease and Compression of Morbidity. *JAMA Cardiol.* 2018;3(4):280.
66. Gierach M, Gierach J, Ewertowska M, Arndt A, Junik R. Correlation between body mass index and waist circumference in patients with metabolic syndrome. *Int Sch Res Notices.* 2014;2014:1-6.
67. Flegal KM, Shepherd JA, Looker AC, Graubard BI, Borrud LG, Ogden CL, et al. Comparisons of percentage body fat, body mass index, waist circumference, and waist-stature ratio in adults. *Am. J. Clin. Nutr.* 2008;89(2):500-8.
68. Bishwajit G, Yaya S, Seydou I. Diabetes mellitus and high blood pressure in relation to BMI among adult non-pregnant women in Bangladesh. *Diabetes Metab Syndr.* 2017;11:S217-S21.
69. Kalani Z, Rafiei M, Salimi T. Comparison of obesity indexes BMI, WHR and WC in association with Hypertension: results from a Blood Pressure Status Survey in Iran. *J Cardiovasc Dis Res.* 2015;6(2).
70. Zhang L, Zhang W-H, Zhang L, Wang P-Y. Prevalence of overweight/obesity and its associations with hypertension, diabetes, dyslipidemia, and metabolic syndrome: a survey in the suburban area of Beijing, 2007. *Obes Facts.* 2011;4(4):284-9.
71. Mishra V, Arnold F, Semenov G, Hong R, Mukuria A. Epidemiology of obesity and hypertension and related risk factors in Uzbekistan. *Eur J Clin Nutr.* 2006;60(12):1355-66.

72. Hwang L-C, Bai C-H, Sun C-A, Chen C-J. Prevalence of metabolically healthy obesity and its impacts on incidences of hypertension, diabetes and the metabolic syndrome in Taiwan. *Asia Pac J Clin Nutr.* 2012;21(2):227.
73. Warsy A, El-Hazmi M, Al-Hazmi A. Prevalence of co-existing hypertension and obesity in Saudis. *Biomed. Pharmacol. J.* 2015;4(2):269-74.
74. Dua S, Bhuker M, Sharma P, Dhall M, Kapoor S. Body mass index relates to blood pressure among adults. *N Am J Med Sci.* 2014;6(2):89-95.
75. Hariharan A, Shanmugam M, Priya L. A cross-sectional study on newly detected hypertensive individuals and its correlation to BMI. *IAIM,* 2019; 6(11): 47-51.
76. Patel SA, Ali MK, Alam D, Yan LL, Levitt NS, Bernabe-Ortiz A, et al. Obesity and its relation with diabetes and hypertension: a cross-sectional study across 4 geographical regions. *Glob Heart.* 2016;11(1):71-9.
77. Ranganathan S, Krishnan TU, Radhakrishnan S. Comparison of dyslipidemia among the normal-BMI and high-BMI group of people of rural Tamil Nadu. *Med J DY Patil Vidyapeeth.* 2015;8(2):149.
78. Van der Hoeven M, Kruger A, Greeff M. Differences in health care seeking behaviour between rural and urban communities in South Africa. *Int. J. Equity Health.* 2012;11(1):31.
79. Schatzkin A, Kipnis V, Carroll RJ, Midthune D, Subar AF, Bingham S, et al. A comparison of a food frequency questionnaire with a 24-hour recall for use in an epidemiological cohort study: results from the biomarker-based Observing Protein and Energy Nutrition (OPEN) study. *Int J Epidemiol.* 2003;32(6):1054-62.
80. Freedman LS, Potischman N, Kipnis V, Midthune D, Schatzkin A, Thompson FE, et al. A comparison of two dietary instruments for evaluating the fat–breast cancer relationship. *Int J Epidemiol.* 2006;35(4):1011-21.
81. Thompson FE, Subar AF. Dietary assessment methodology. *Nutrition in the Prevention and Treatment of Disease: Elsevier;* 2017. p. 5-48.

CHAPTER 5 TEN-YEAR DIETARY INTAKE TRENDS DIFFER BETWEEN RURAL AND URBAN SOUTH AFRICANS IN THE NORTH WEST PROVINCE: AN UPDATE ON THE NUTRITION TRANSITION – THE PURE-SA-NWP STUDY

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5.1 Abstract

Objective: Over the past few decades rural and urban populations in South Africa have been undergoing a rapid nutrition transition, with diets changing from traditional, prudent diets to Westernised, imprudent diets, and consequently increasing NCD risk prevalence. The aim of this study is to present the results of in-depth trend analysis of energy and nutrient intakes of urban and rural groups of the PURE-SA-NWP study over the period 2005 to 2015.

Design: The South African leg of the Prospective Urban and Rural Epidemiological study was conducted in rural and urban populations in the North West province of South Africa. Baseline measurements were obtained in 2005, with follow-up measurements in 2010 and 2015. This article reports on 722 participants with complete nutrient intake data from 2005 to 2015.

Results: For rural men and women, trend analysis indicated significant upward trends in total energy ($p < 0.001$) (men: 7.60 MJ to 13.26 MJ, women: 6.60 MJ to 11.89 MJ), and most macro- ($p < 0.001$) and micronutrients ($p < 0.001$) over the study period. Trend analysis also indicated upward trends, from 2005 to 2015, for the proportion of total energy intake from all macronutrients (except alcohol) ($p < 0.001$). In exploring the interaction effect between rural and urban men and women, the observed trends differed significantly ($p < 0.001$ for interaction effect) for total energy intake as well as intakes of most micro- and macronutrients.

Conclusion: From this in-depth trend analysis of energy and nutrient intakes of urban and rural participants of the PURE-SA-NWP study (2005-2015), it is evident that rural and urban groups differ significantly in terms of dietary changes occurring during the nutrition transition. The rural groups were experiencing a nutrition transition so rapid that they not only caught up with the urban groups in terms of energy and nutrient intake but actually surpassed them by 2015. Energy and nutrient intakes in the urban areas on the other hand, seemed to have equalised by 2015.

Keywords: Nutrition transition, rural, urban, PURE-NWP-SA, non-communicable disease, dietary intake trends

5.2 Introduction

The nutrition transition is a phenomenon which has been receiving increased attention over the last few decades, especially in the developing world ⁽¹⁻⁹⁾. The nutrition transition is described as the changes in dietary intake and energy expenditure occurring with demographic, economic and epidemiological changes in populations. Communities move from traditional dietary intakes (prudent, low-fat, low-energy) to dietary intakes typical of Western societies (high-fat, high-energy) ^(8, 9). The nutrition transition takes place in five fairly distinct stages, with most developing countries, including South Africa, being between the third and fifth phases ^(4, 10-13). Stage 3 is characterised by an increased income and a subsequent decrease in famine. Then follows stage 4, where the diet and activity patterns change to such an extent that new diseases and increased disability start to emerge. In the final stage (stage 5), behavioural change, in terms of food intake and physical activity leads to a reduction of the negative effects of the previous stages, leading to “successful ageing” and a decrease in nutrition-related non-communicable diseases (NCDs) ⁽¹²⁾.

Dietary changes associated with a nutrition transition are associated with an increase in NCDs globally, but also in South Africa in particular ⁽¹⁴⁻²⁰⁾. The WHO reported that the majority of all NCD-related deaths (80%) occur in low- and middle-income developing countries ⁽²¹⁾, including South Africa. Statistics-SA’s latest report (the 2017 report, published in March 2020) on the causes of death in South Africa indicated that in 2007 communicable disease was the leading cause of death (responsible for 46,9% of deaths), followed by NCDs (46,9% of deaths). However, in the 10 years from 2007 to 2017, the number of NCD-related deaths in South Africa increased by 13,7% percentage points, making NCDs the leading cause of death (57,8%), while communicable disease accounted for only 30,7% of deaths ⁽²²⁾.

The nutrition transition in South Africa has previously been reported on ^(4, 7, 8, 11, 14, 23). It is, however, extremely important that regular updates are made with regard to where South African rural and urban groups stand in terms of the nutrition transition as each of the different stages of the nutrition transition poses its own unique challenges – challenges that will need specific nutritional recommendations and interventions.

The dietary changes of the PURE-SA-NWP (The Prospective Urban and Rural Epidemiological study in the North West province of South Africa) study from 2005 to 2010 indicated that the nutrition transition was still happening at a rapid pace in both the rural and urban groups⁽⁴⁾ This

data is also included in the current study. The aim of this study is to present the results of in-depth trend analysis of energy and nutrient intakes of urban and rural groups of the PURE-SA-NWP study over the period from 2005 to 2015.

5.3 Methods

5.3.1 Study design

This study is a sub-study of the South African arm of the transnational Prospective Urban and Rural Epidemiological (PURE) study conducted in the North West Province of South Africa (PURE-SA-NWP). PURE is an international epidemiological prospective (cohort) study, coordinated by the Population Health Research Institute in Ontario, Canada. The purpose of the study is to track chronic disease progression in urban and rural subjects. The study is being conducted in 27 developing countries/territories in numerous stages of economic development. Data collection for PURE-SA-NWP took place in two communities, one urban and one rural. In 2005, baseline data were collected, followed by two follow-up collections in 2010 and 2015. The study design has previously been described ⁽²⁴⁻²⁶⁾. This article focuses on the data collected in 2005, 2010 and 2015.

5.3.2 Participant recruitment

Participants were recruited from two communities (urban and rural). These communities were further stratified into four geographical areas (two within each community). A household census was completed in 6000 randomly selected households (1500 households from each of the geographical areas). Household census forms were completed by fieldworkers from the communities being researched, who were extensively trained in research methodology and ethics.

A family questionnaire and a household questionnaire were included during the household census. This was done so that all members eligible to participate in the study could be identified. Signed informed consent was given by every head of each household. This process was repeated until 1500 households from each specific community were included.

Applying the specific eligibility criteria previously described ⁽²⁵⁾, as well as the household census data, 4000 potential participants were identified as eligible (rural=2000 and urban=2000). Subject recruitment has previously been described ^(24, 25). Only participants with dietary data for all three data collection points (2005, 2010 and 2015) have been included in our analysis, resulting in 722 participants.

5.3.3 Measurements

5.3.3.1 Questionnaires

The standardised and structured questionnaires (including questionnaires on demographics, socioeconomics, lifestyle and physical activity) ⁽²⁷⁾ of the international PURE study were used in PURE-SA-NW. As was the case during the recruitment phase of the study, trained fieldworkers from the communities that were part of the research were used for the administration of these semi-structured questionnaires. Participants could exercise a choice of language in which they preferred to be questioned (Setswana, Afrikaans or English). The same fieldworkers conducted the questionnaires in 2010 and 2015. (data was collected from August to September in the rural groups and from October to November in the urban groups.)

Dietary data were collected using a 145-item culturally-specific quantified food frequency questionnaire (QFFQ). This QFFQ was developed and validated for this population group and proved to have good reproducibility ⁽²⁸⁻³⁰⁾. Participants were asked about their habitual dietary intake over the previous 30 days. Dietary intake frequency included the number of times foods were consumed per day/week/month or seldom/never. In order to estimate portion sizes, life-sized photographs in a book depicting 37 foods (in three portion sizes) were used. This book was specifically developed to be used amongst African groups in the North West province of South-Africa ⁽³¹⁾. Dietary intakes of different foods included in the QFFQ were calculated using a stepwise approach. The amounts of each serving consumed were converted to grams (g) using standardised tables ⁽³²⁾ according to the South African Food Composition Database System of the South African Medical Research Council ⁽³³⁾. Finally, daily consumption was determined by multiplying the frequency of dietary intakes (days per month) by the portion sizes divided over 30 days. Food intake was coded according to the South African Food Composition Database System of the South African Medical Research Council ⁽³³⁾.

In 2010, dietary questionnaires (QFFQs) were administered by the same fieldworkers who had administered the questionnaires in 2005. In 2015, a new group of fieldworkers was trained by nutrition experts for the administration of the dietary questionnaires (QFFQs). New fieldworkers (2015) received training identical to that of the previous fieldworkers of 2005 and 2010 and were trained by the same nutrition experts used in 2005 and 2010.

5.3.3.2 Other measurements

The anthropometric measures and collection of physical activity data have been described previously ^(11, 12). For this paper, results from the following measurements will be used: waist circumference, height, weight and the physical activity index.

5.3.4 Statistical analysis

IBM SPSS statistics version 26 were used as statistical software for this analysis ⁽³⁴⁾. Only participants with an energy intake > 3000 kJ/day and < 30 000 kJ/day have been included in the analysis ⁽²⁵⁾. Paired t-tests were conducted to determine whether changes in 2015 differed significantly from the baseline in 2005, with respect to the following variables: locality, education, BMI, obesity (BMI > 30 kg/ m²), waist circumference, physical activity index and energy intake. The baseline data for age and gender were reported in table 1.

A linear mixed-model approach was used to assess the energy and nutrient intake changes of urban and rural men and women over time (2005-2010-2015). An unstructured covariance matrix was specified, and restricted maximum likelihood estimation was used. Time, location, and gender were treated as fixed effects and the subjects as a random effect in the model. The results were reported as estimated marginal means (EMM) and standard errors (SE).

Macronutrient intakes of participants have been reported in their respective units and as a percentage of total energy. Micronutrient and fibre intake were also explored in terms of their nutrient density by reporting them per 4.2 MJ.

5.4 Results

5.4.1 Summary of the study group

Figure 5-1 provides a summary of the participant recruitment and follow-up from the baseline in 2005 to the final data collection in 2015.

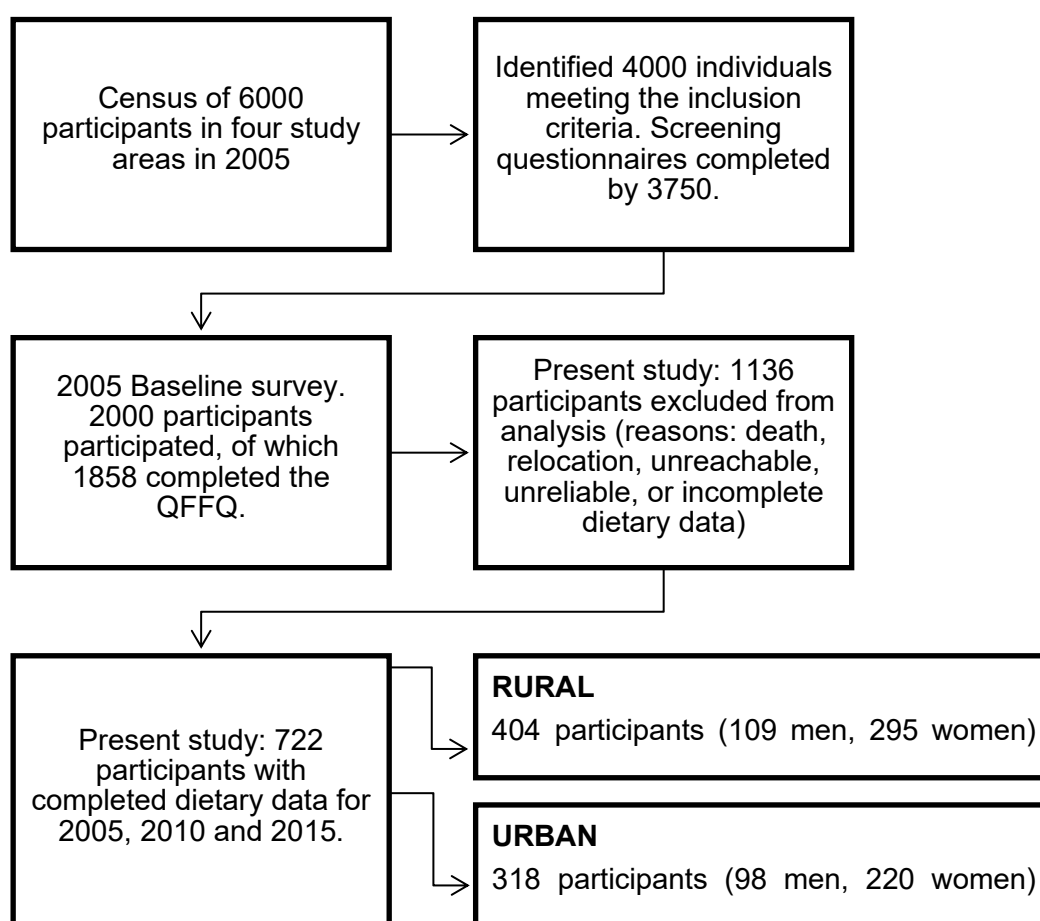


Figure 5-1: Summary of participant recruitment and follow-up from the baseline in 2005 to cohort used in the present study
QFFQ, quantified food frequency questionnaire

5.4.2 Comparison of the baseline characteristics of the 2015 cohort

In Table 5-1, the 2005 baseline characteristics for participants and the data followed up in 2015 (n=722) are compared. The 2015 cohort had a higher mean energy intake ($p < 0.005$) and was less active ($p < 0.005$) in 2015 compared with baseline. In 2015, the cohort presented with a higher BMI ($p < 0.005$) and waist circumference ($p < 0.005$). The percentage of obese participants ($\text{BMI} \geq 30\text{kg/m}^2$) increased significantly ($p < 0.005$).

Table 5-1: Comparison of the baseline data and the 2015 data of the cohort

Variable	Baseline data in 2005 of participants followed up in 2015			Cohort data of participants in 2015			p-value ¹
	n	mean	SD	n	mean	SD	
Age (Years)	722	50.1	9.4	722	-	-	-
Sex (male, %)	722	29%		722	-	-	-
Locality (rural, %)	722	56%		722	-	-	-
Education (no formal education, %)	722	35.20%		722	-	-	-
Body mass index (BMI) (kg/m ²)	707	25.6	7.1	707	26.5	7.5	< 0.005
Obesity (BMI ≥ 30kg/m ²)	153	24.70%		153	30.60%		< 0.005
Waist Circumference (cm)	707	80.8	13	707	88.7	15.2	< 0.005
Physical activity index	686	7.4	1.9	686	5	1.7	< 0.005
Energy intake (MJ)	722	8.2	3.6	722	11.4	5.5	< 0.005
¹ P-value for significance of differences between the baseline data and the 2015 data of the cohort							

5.4.3 Trends in energy and macro- and micronutrient intakes of men from 2005 to 2015

The trends in mean energy and nutrient intake of rural and urban men from 2005 to 2015 are presented in Table 5-2.

For rural men, trend analysis indicated significant upward trends in total energy ($p < 0.001$) (from 7.60 MJ in 2005 to 13.26 MJ in 2015) and macro- ($p < 0.001$) and micronutrients ($p < 0.001$) over the study period. Alcohol intake remained unchanged.

With regard to urban men, different results emerged, and total energy intake did not change significantly over the study period. Regarding macro- and micronutrient intake, significant upward trends were observed for MUFA ($p < 0.041$), added sugar ($p < 0.019$; up from baseline to 2015), manganese ($p < 0.034$), niacin ($p = 0.001$), vitamin B6 ($p < 0.001$) and vitamin D ($p < 0.008$). All other nutrient intakes, including alcohol, remained unchanged.

In exploring the interaction effect between rural and urban men, the observed trends differed significantly ($p < 0.001$ for interaction effect) for total energy intake as well as intakes of most micro- and macronutrients, apart from the intake trends of the following few nutrients: added sugar, vitamin D and alcohol.

Table 5-2: Trends in mean energy and nutrient intake of rural and urban men from 2005 to 2015

Energy and nutrients	Locality	MEN (Rural n=109; Urban n=98)							
		2005		2010		2015		Time effect ¹	Time*Locality interaction effect ²
		EMM	SE	EMM	SE	EMM	SE		
Energy (MJ)	Urban	10.90	498.14	14.40	498.14	11.36	498.14	0.511	<0.001
	Rural	7.60	472.88	10.22	472.88	13.26	472.88	<0.001	
Total protein (g)	Urban	79.74	4.30	111.25	4.30	83.10	4.30	0.587	<0.001
	Rural	48.91	4.63	72.85	4.63	103.49	4.63	<0.001	
Plant protein (g)	Urban	41.19	1.86	48.71	1.86	39.56	1.86	0.530	<0.001
	Rural	33.32	2.19	39.74	2.19	53.22	2.19	<0.001	
Animal protein (g)	Urban	35.71	3.08	60.89	3.08	43.183	3.08	0.091	<0.001
	Rural	14.91	3.56	33.00	3.56	50.14	3.56	<0.001	
Total fat (g)	Urban	73.17	4.60	102.88	4.60	79.52	4.60	0.314	<0.001
	Rural	35.78	4.15	62.93	4.15	91.37	4.15	<0.001	
Saturated fat (g)	Urban	18.37	1.36	28.05	1.36	21.43	1.36	0.101	<0.001
	Rural	7.79	1.26	17.71	1.26	24.15	1.26	<0.001	
Monounsaturated fat (g)	Urban	20.53	1.53	31.17	1.53	24.77	1.53	0.041	<0.001
	Rural	8.36	1.54	17.63	1.54	28.85	1.54	<0.001	
Polyunsaturated fat (g)	Urban	21.07	1.48	27.61	1.48	22.00	1.48	0.647	<0.001
	Rural	11.82	1.29	17.75	1.29	25.19	1.29	<0.001	
Cholesterol (mg)	Urban	275.31	25.92	463.08	25.92	346.38	25.92	0.060	0.001
	Rural	124.51	26.34	257.44	26.34	357.18	26.34	<0.001	
Total carbohydrate (g)	Urban	364.13	17.31	499.17	17.31	389.97	17.31	0.310	<0.001
	Rural	287.52	17.49	388.02	17.49	476.11	17.49	<0.001	
Added sugar (g)	Urban	47.27	6.04	76.67	6.04	66.08	6.04	0.019	0.235
	Rural	30.13	4.84	64.50	4.84	61.20	4.84	<0.001	
Dietary fibre (g)	Urban	29.67	1.43	37.50	1.43	27.30	1.43	0.252	<0.001
	Rural	20.80	1.59	27.30	1.59	35.78	1.59	<0.001	
Calcium (mg)	Urban	508.64	37.20	705.75	37.20	607.68	37.20	0.060	<0.001
	Rural	272.13	35.97	376.90	35.97	655.53	35.97	<0.001	
Iron (mg)	Urban	17.78	0.97	26.14	0.97	20.33	0.97	0.079	<0.001
	Rural	13.40	0.95	19.27	0.95	25.34	0.95	<0.001	
Magnesium (mg)	Urban	449.42	22.36	515.23	22.36	454.51	22.36	0.865	<0.001
	Rural	360.53	23.73	405.47	23.73	545.61	23.73	<0.001	
Phosphorus (mg)	Urban	1277.12	64.18	1673.21	64.18	1378.64	64.18	0.265	<0.001
	Rural	929.55	65.63	1172.61	65.63	1652.29	65.63	<0.001	
Potassium (mg)	Urban	2423.42	131.27	3565.28	131.27	2780.41	131.27	0.061	<0.001
	Rural	1494.31	118.62	2480.34	118.62	3334.37	118.62	<0.001	
Zinc (mg)	Urban	14.06	0.81	20.95	0.81	15.39	0.81	0.265	<0.001
	Rural	9.57	0.83	14.72	0.83	19.78	0.83	<0.001	
Copper (mg)	Urban	1.68	0.11	2.25	0.11	1.92	0.11	0.107	<0.001
	Rural	1.18	0.09	1.61	0.09	2.38	0.09	<0.001	
Manganese (µg)	Urban	3206.21	211.95	4373.21	211.95	3847.66	211.95	0.034	0.002
	Rural	2500.46	254.68	3467.83	254.68	4595.78	254.68	<0.001	

Energy and nutrients	Locality	MEN (Rural n=109; Urban n=98)							
		2005		2010		2015		Time effect ¹	Time*Locality interaction effect ²
		EMM	SE	EMM	SE	EMM	SE		
Vitamin A (µg)	Urban	1160.78	148.21	1933.25	148.21	1525.91	148.21	0.076	0.004
	Rural	582.56	107.32	1065.82	107.32	1667.25	107.32	<0.001	
Thiamine (mg)	Urban	2.20	0.11	2.94	0.11	2.22	0.11	0.875	<0.001
	Rural	1.76	0.11	2.20	0.11	2.83	0.11	<0.001	
Riboflavin (mg)	Urban	1.76	0.11	2.60	0.11	2.02	0.11	0.117	<0.001
	Rural	1.11	0.12	1.55	0.12	2.28	0.12	<0.001	
Niacin (mg)	Urban	20.27	1.35	36.61	1.35	27.22	1.35	0.001	<0.001
	Rural	13.72	1.50	24.27	1.50	32.90	1.50	<0.001	
Vitamin B ₆ (mg)	Urban	2.09	0.20	5.10	0.20	3.46	0.20	<0.001	0.008
	Rural	1.37	0.22	3.41	0.22	3.91	0.22	<0.001	
Folate (µg)	Urban	513.65	32.84	723.61	32.84	596.62	32.84	0.075	0.001
	Rural	397.40	31.65	515.15	31.65	685.87	31.65	<0.001	
Vitamin B ₁₂ (µg)	Urban	6.72	1.04	9.94	1.04	8.71	1.04	0.163	0.009
	Rural	2.47	1.12	4.41	1.12	10.01	1.12	<0.001	
Pantothenic acid (mg)	Urban	5.52	0.40	9.51	0.40	6.68	0.40	0.050	<0.001
	Rural	3.40	0.46	5.25	0.46	8.11	0.46	<0.001	
Biotin (µg)	Urban	53.02	4.90	83.16	4.90	62.50	4.90	0.171	0.001
	Rural	27.85	3.22	44.24	3.22	64.79	3.22	<0.001	
Vitamin C (mg)	Urban	52.48	6.43	87.89	6.43	57.53	6.43	0.590	<0.001
	Rural	13.69	5.82	45.01	5.82	77.48	5.82	<0.001	
Vitamin D (mg)	Urban	3.60	0.41	6.53	0.41	5.21	0.41	0.008	0.088
	Rural	1.91	0.39	3.79	0.39	4.86	0.39	<0.001	
Vitamin E (mg)	Urban	14.27	1.04	18.87	1.04	15.58	1.04	0.382	0.001
	Rural	9.38	0.92	13.63	0.92	17.00	0.92	<0.001	
Alcohol (g)	Urban	17.27	3.23	19.56	3.23	22.18	3.23	0.182	0.103
	Rural	17.83	2.95	13.63	2.95	14.27	2.95	0.331	
Values are expressed as estimated marginal means (EMM) and standard error (SE). TE, Total energy									
¹ Type III tests of fixed effects with time as continuous variable were performed to determine significant trends over time within groups, values significant p < 0.05									
² Type III tests of fixed effects with time as continuous variable were performed to determine significant differences between rural and urban groups, values significant p < 0.05									

5.4.4 Trends in energy and macro- and micronutrient intakes of women from 2005 to 2015

The trends in mean energy and nutrient intake of rural and urban women from 2005 to 2015 are presented in table 5-3.

Trend analysis for rural women reflected the above observations for rural men, with increases in the intakes of total energy (from 6.60 MJ in 2005 to 11.89 MJ in 2015), as well as in all macro- and micronutrients (p < 0.001) over the study period. In addition, a significant downward trend in alcohol intake was observed for this group (p < 0.001).

As in the case of urban men, no trend was observed in terms of the total energy intake of urban women as it did not change significantly from 2005 to 2010 to 2015, showing an upward trend in 2010 and then a downward trend in 2015. Trend analysis showed significant upward trends in the macronutrient intake of urban women for protein ($p=0.044$), animal protein ($p < 0.001$), MUFA ($p=0.042$), total carbohydrate ($p=0.005$) and added sugar (< 0.001). In terms of the micronutrient intake of urban women, trend analysis again showed significant upward trends for the intakes of calcium, iron, potassium, zinc, copper, manganese, vitamin A, niacin, vitamin B6, folate, pantothenic acid and vitamin C over the study period ($p < 0.05$). Urban women presented with a significant downward trend in alcohol intake ($p < 0.003$).

When comparing the observed trends over time by looking at the interaction effect between rural and urban women, most trends in energy and nutrient intake differed significantly ($p < 0.001$ for interaction effect) except for added sugar and alcohol intake.

Table 5-3: Trends in mean energy and nutrient intake of rural and urban women from 2005 to 2015

Energy and nutrients	Locality	WOMEN (Rural n=295; Urban n=220)							
		2005		2010		2015		Time effect ¹	Time*Locality interaction effect ²
		EMM	SE	EMM	SE	EMM	SE		
Energy (MJ)	Urban	9.36	299.29	12.20	299.29	9.84	299.29	0.261	<0.001
	Rural	6.60	256.93	9.93	256.93	11.89	256.93	<0.001	
Total protein (g)	Urban	67.43	2.51	94.55	2.51	74.69	2.51	0.044	<0.001
	Rural	42.81	2.10	67.48	2.10	88.66	2.10	<0.001	
Plant protein (g)	Urban	33.18	1.08	38.56	1.08	32.27	1.08	0.545	<0.001
	Rural	28.35	1.14	37.04	1.14	46.37	1.14	<0.001	
Animal protein (g)	Urban	32.12	1.84	54.73	1.84	42.08	1.84	<0.001	<0.001
	Rural	14.10	1.42	30.06	1.42	42.12	1.42	<0.001	
Total fat (g)	Urban	70.23	2.92	93.43	2.92	73.73	2.92	0.381	<0.001
	Rural	35.25	2.33	67.76	2.33	82.90	2.33	<0.001	
Saturated fat (g)	Urban	18.06	0.81	25.82	0.81	19.98	0.81	0.095	<0.001
	Rural	7.75	0.70	19.00	0.70	21.90	0.70	<0.001	
Monounsaturated fat (g)	Urban	20.31	0.96	28.88	0.96	23.00	0.96	0.042	<0.001
	Rural	8.26	0.77	18.59	0.77	24.51	0.77	<0.001	
Polyunsaturated fat (g)	Urban	20.67	1.02	26.46	1.02	21.25	1.02	0.677	<0.001
	Rural	12.07	0.88	20.87	0.88	25.80	0.88	<0.001	
Cholesterol (mg)	Urban	250.03	16.16	418.35	16.16	278.91	16.16	0.212	<0.001
	Rural	127.60	11.84	224.83	11.84	304.26	11.84	<0.001	
Total carbohydrate (g)	Urban	302.96	10.18	421.67	10.18	345.45	10.18	0.005	<0.001
	Rural	254.08	9.59	381.10	9.59	441.00	9.59	<0.001	
Added sugar (g)	Urban	46.94	3.64	82.17	3.64	74.28	3.64	<0.001	0.449
	Rural	29.45	3.48	69.87	3.48	62.24	3.48	<0.001	
Dietary fibre (g)	Urban	23.36	0.86	32.47	0.86	25.11	0.86	0.160	<0.001
	Rural	18.39	0.87	26.45	0.87	32.87	0.87	<0.001	
Calcium (mg)	Urban	462.99	21.85	639.52	21.85	583.64	21.85	<0.001	<0.001
	Rural	230.44	17.83	420.19	17.83	630.29	17.83	<0.001	
Iron (mg)	Urban	14.25	0.54	21.45	0.54	17.47	0.54	<0.001	<0.001
	Rural	11.87	0.49	18.00	0.49	22.39	0.49	<0.001	
Magnesium (mg)	Urban	349.73	10.72	402.41	10.72	348.64	10.72	0.941	<0.001
	Rural	280.48	11.16	370.12	11.16	466.54	11.16	<0.001	
Phosphorus (mg)	Urban	1079.27	35.59	1364.23	35.59	1126.52	35.59	0.343	<0.001
	Rural	758.31	31.48	1094.93	31.48	1408.02	31.48	<0.001	
Potassium (mg)	Urban	2120.31	80.86	3233.46	80.86	2570.33	80.86	<0.001	<0.001
	Rural	1356.98	73.41	2561.58	73.41	3295.65	73.41	<0.001	
Zinc (mg)	Urban	11.33	0.43	16.25	0.43	13.06	0.43	0.005	<0.001
	Rural	8.26	0.39	13.09	0.39	16.63	0.39	<0.001	
Copper (mg)	Urban	1.49	0.06	2.06	0.06	1.69	0.06	<0.001	<0.001
	Rural	1.05	0.06	1.57	0.06	2.38	0.06	<0.001	
Manganese (µg)	Urban	2501.55	102.20	3480.68	102.20	2942.26	102.20	0.003	<0.001

Energy and nutrients	Locality	WOMEN (Rural n=295; Urban n=220)							Time*Locality interaction effect ²
		2005		2010		2015		Time effect ¹	
		EMM	SE	EMM	SE	EMM	SE		
	Rural	1692.01	107.72	2835.76	107.72	3393.35	107.72	<0.001	
Vitamin A (µg)	Urban	1139.00	104.64	2151.73	104.64	1504.95	104.64	0.014	<0.001
	Rural	581.03	88.00	1109.79	88.00	1919.57	88.00	<0.001	
Thiamine (mg)	Urban	1.69	0.06	2.21	0.06	1.83	0.06	0.102	<0.001
	Rural	1.53	0.06	2.01	0.06	2.42	0.06	<0.001	
Riboflavin (mg)	Urban	1.55	0.08	2.39	0.08	1.76	0.08	0.059	<0.001
	Rural	0.87	0.06	1.42	0.06	2.05	0.06	<0.001	
Niacin (mg)	Urban	17.22	0.79	28.70	0.79	23.32	0.79	<0.001	<0.001
	Rural	11.51	0.72	21.57	0.72	28.75	0.72	<0.001	
Vitamin B ₆ (mg)	Urban	1.70	0.12	3.93	0.12	3.11	0.12	<0.001	0.002
	Rural	1.24	0.10	2.83	0.10	3.36	0.10	<0.001	
Folate (µg)	Urban	398.81	19.44	569.81	19.44	474.82	19.44	0.005	<0.001
	Rural	349.08	18.87	436.96	18.87	568.34	18.87	<0.001	
Vitamin B ₁₂ (µg)	Urban	6.74	0.68	10.36	0.68	7.38	0.68	0.728	<0.001
	Rural	2.43	0.54	3.86	0.54	9.66	0.54	<0.001	
Pantothenic acid (mg)	Urban	4.88	0.25	8.30	0.25	6.31	0.25	<0.001	<0.001
	Rural	2.95	0.21	5.19	0.21	7.28	0.21	<0.001	
Biotin (µg)	Urban	44.81	3.46	77.25	3.46	54.43	3.46	0.052	<0.001
	Rural	26.15	1.72	39.90	1.72	60.24	1.72	<0.001	
Vitamin C (mg)	Urban	48.42	4.66	93.62	4.66	71.86	4.66	0.001	<0.001
	Rural	16.68	4.21	43.88	4.21	121.72	4.21	<0.001	
Vitamin D (mg)	Urban	3.33	0.24	5.59	0.24	3.92	0.24	0.097	0.003
	Rural	2.11	0.19	3.48	0.19	3.93	0.19	<0.001	
Vitamin E (mg)	Urban	13.53	0.69	17.70	0.69	14.03	0.69	0.600	<0.001
	Rural	9.41	0.60	14.31	0.60	17.69	0.60	<0.001	
Alcohol (g)	Urban	10.20	1.27	7.27	1.27	5.40	1.27	0.003	0.794
	Rural	7.05	0.87	2.52	0.87	1.75	0.87	<0.001	

Values are expressed as estimated marginal means (EMM) and standard error (SE). TE, Total energy

¹ Type III tests of fixed effects with time as continuous variable were performed to determine significant trends over time within groups, values significant p < 0.05

² Type III tests of fixed effects with time as continuous variable were performed to determine significant differences between rural and urban groups, values significant p < 0.05

5.4.5 Trends in macronutrient distribution of rural and urban men and women

Table 5-4 and Table 5-5 depict the trends in the macronutrient distribution of rural and urban men and women, respectively.

Proportion of total energy from fats

Trend analysis indicated an upward trend from 2005 to 2015 for the proportion of total energy intake from total fat over all data collection points in rural men (p < 0.001) and rural women (p <

0.001). A similar significant trend was observed for rural men and women for percentage contributions of saturated fat, MUFA and PUFA (all p-values, $p < 0.001$).

In terms of the trend analysis for the urban group, an upward trend in the percentage of energy from total fat was noted for urban men ($p=0.034$). Trend analysis further indicated an upward trend for the percentage contribution of MUFA for both men ($p=0.041$) and women ($p=0.042$). The percentage contributions from PUFA and saturated fat intake did not change significantly.

Proportion of total energy from protein and carbohydrates

Again, trend analysis indicated a significant upward trend in the percentages of energy from animal protein, plant protein and total carbohydrates in both rural men and women (all p-values, $p < 0.001$) (see also Figure 5-3).

No such trends were observed for urban men as they showed no significant trends for the proportions of energy contributed by either protein or carbohydrate. Urban women presented with significant upward trends in energy from total protein ($p=0.044$), animal protein ($p < 0.001$) and total carbohydrates ($p=0.005$).

Proportion of total energy from alcohol

The trends in the proportion of energy from alcohol are similar for rural and urban women, indicating a downward trend over the study period (rural women: $p < 0.001$; urban women: $p=0.003$). Although the proportion of energy from alcohol increased consistently from 2005 to 2015 for urban men, the trend was not significant.

The dissimilarity of the observed trends of the rural versus urban groups in terms of percentage contributions by the different macronutrients is confirmed by the interaction effect being significant for all macronutrients (except added sugar and alcohol) in both men and women ($p < 0.001$ for interaction effect).

Table 5-4: Trends in macronutrient distribution and nutrient density of rural and urban men from 2005 to 2015

Macronutrient distribution	Locality	MEN (Rural n=109; Urban n=98)							
		2005		2010		2015		Time effect ¹	Time*Locality interaction effect ²
		EMM	SE	EMM	SE	EMM	SE		
% of TE from protein	Urban	13.56	0.73	18.91	0.73	14.13	0.73	0.587	<0.001
	Rural	8.31	0.79	12.39	0.79	17.59	0.79	<0.001	
% of TE from animal protein	Urban	6.07	0.52	10.35	0.52	7.34	0.52	0.091	<0.001
	Rural	2.53	0.61	5.61	0.61	8.52	0.61	<0.001	
% of TE from plant protein	Urban	7.00	0.32	8.28	0.32	6.73	0.32	0.053	<0.001
	Rural	5.66	0.37	6.76	0.37	9.05	0.37	<0.001	
% of TE from total fat	Urban	27.81	1.75	39.09	1.75	30.22	1.75	0.314	<0.001
	Rural	13.60	1.58	23.91	1.58	34.72	1.58	<0.001	
% of TE from saturated fat	Urban	6.98	0.52	10.66	0.52	8.14	0.52	0.101	<0.001
	Rural	2.96	0.48	6.73	0.48	9.18	0.48	<0.001	
% of TE from monounsaturated fat	Urban	7.80	0.58	11.84	0.58	9.41	0.58	0.041	<0.001
	Rural	3.18	0.59	6.70	0.59	10.96	0.59	<0.001	
% of TE from polyunsaturated fat	Urban	8.00	0.56	10.49	0.56	8.36	0.56	0.647	<0.001
	Rural	4.49	0.49	6.74	0.49	9.57	0.49	<0.001	
% of TE from total carbohydrate	Urban	61.90	2.94	84.86	2.94	66.30	2.94	0.310	<0.001
	Rural	48.88	2.97	65.96	2.97	80.94	2.97	<0.001	
% of TE from added sugar	Urban	8.04	1.03	13.03	1.03	11.23	1.03	0.019	0.235
	Rural	5.12	0.82	10.97	0.82	10.40	0.82	<0.001	
% of TE from alcohol	Urban	5.01	0.94	5.67	0.94	6.43	0.94	0.182	0.103
	Rural	5.17	0.85	3.95	0.85	4.14	0.85	0.331	
Nutrient density									
Dietary fibre (g/4.2MJ)	Urban	7.07	0.34	8.93	0.34	6.50	0.34	0.252	<0.001
	Rural	4.95	0.38	6.50	0.38	8.52	0.38	<0.001	
Iron (mg/4.2MJ)	Urban	4.23	0.23	6.22	0.23	4.84	0.23	0.079	<0.001
	Rural	3.19	0.23	4.59	0.23	6.03	0.23	<0.001	
Zinc (mg/4.2MJ)	Urban	3.35	0.19	4.99	0.19	3.66	0.19	0.265	<0.001
	Rural	2.28	0.20	3.51	0.20	4.71	0.20	<0.001	
Calcium (mg/4.2MJ)	Urban	121.10	8.86	168.04	8.86	144.69	8.86	0.060	<0.001
	Rural	64.79	8.56	89.74	8.56	156.08	8.56	<0.001	
Thiamine (mg/4.2MJ)	Urban	0.52	0.03	0.70	0.03	0.53	0.03	0.158	<0.001
	Rural	0.42	0.03	0.52	0.03	0.67	0.03	<0.001	
Riboflavin (mg/4.2MJ)	Urban	0.42	0.03	0.62	0.03	0.48	0.03	0.117	<0.001
	Rural	0.26	0.03	0.37	0.03	0.54	0.03	<0.001	
Niacin (mg/4.2MJ)	Urban	4.83	0.32	8.72	0.32	6.48	0.32	0.001	<0.001
	Rural	3.27	0.36	5.78	0.36	7.83	0.36	<0.001	
Vitamin C (mg/4.2MJ)	Urban	12.50	1.53	20.93	1.53	13.70	1.53	0.590	<0.001
	Rural	3.26	1.39	10.72	1.39	18.45	1.39	<0.001	

Values are expressed as estimated marginal means (EMM) and standard error (SE). TE, Total energy

¹ Type III tests of fixed effects with time as continuous variable were performed to determine significant trends over time within groups, values significant p < 0.05

Macronutrient distribution	Locality	MEN (Rural n=109; Urban n=98)							
		2005		2010		2015		Time effect ¹	Time*Locality interaction effect ²
		EMM	SE	EMM	SE	EMM	SE		
² Type III tests of fixed effects with time as continuous variable were performed to determine significant differences between rural and urban groups, values significant p < 0.05									

Table 5-5: Trends in macronutrient distribution and nutrient density of rural and urban women from 2005 to 2015

Macronutrient distribution	Locality	WOMEN (Rural n=295; Urban n=220)							Time*Locality interaction effect ²
		2005		2010		2015		Time effect ¹	
		EMM	SE	EMM	SE	EMM	SE		
% of TE from protein	Urban	11.46	0.43	16.07	0.43	12.70	0.43	0.044	<0.001
	Rural	7.28	0.36	11.47	0.36	15.07	0.36	<0.001	
% of TE from animal protein	Urban	5.46	0.31	9.30	0.31	7.15	0.31	<0.001	<0.001
	Rural	2.40	0.24	5.11	0.24	7.16	0.24	<0.001	
% of TE from plant protein	Urban	5.64	0.18	6.56	0.18	5.49	0.18	0.545	<0.001
	Rural	4.82	0.19	6.30	0.19	7.88	0.19	<0.001	
% of TE from total fat	Urban	26.69	1.11	35.50	1.11	28.02	1.11	0.381	<0.001
	Rural	13.39	0.89	25.75	0.89	31.50	0.89	<0.001	
% of TE from saturated fat	Urban	6.86	0.31	9.81	0.31	7.59	0.31	0.095	<0.001
	Rural	2.94	0.27	7.22	0.27	8.32	0.27	<0.001	
% of TE from monounsaturated fat	Urban	7.72	0.37	10.98	0.37	8.74	0.37	0.042	<0.001
	Rural	3.14	0.29	7.06	0.29	9.31	0.29	<0.001	
% of TE from polyunsaturated fat	Urban	7.85	0.39	10.06	0.39	8.07	0.39	0.677	<0.001
	Rural	4.59	0.33	7.93	0.33	9.80	0.33	<0.001	
% of TE from total carbohydrate	Urban	51.50	1.73	71.68	1.73	58.73	1.73	0.005	<0.001
	Rural	43.19	1.63	64.79	1.63	74.97	1.63	<0.001	
% of TE from added sugar	Urban	7.98	0.62	13.97	0.62	12.63	0.62	<0.001	0.441
	Rural	5.01	0.59	11.88	0.59	10.58	0.59	<0.001	
% of TE from alcohol	Urban	2.96	0.37	2.11	0.37	1.57	0.37	0.003	0.794
	Rural	2.04	0.25	0.73	0.25	0.51	0.25	<0.001	
Nutrient density									
Dietary fibre (g/4.2MJ)	Urban	5.56	0.20	7.73	0.20	5.98	0.20	0.160	<0.001
	Rural	4.38	0.21	6.30	0.21	7.83	0.21	<0.001	
Iron (mg/4.2MJ)	Urban	3.39	0.13	5.11	0.13	4.16	0.13	<0.001	<0.001
	Rural	2.83	0.12	4.29	0.12	5.33	0.12	<0.001	
Zinc (mg/4.2MJ)	Urban	2.70	0.10	3.87	0.10	3.11	0.10	0.005	<0.001
	Rural	1.97	0.09	3.12	0.09	3.96	0.09	<0.001	
Calcium (mg/4.2MJ)	Urban	110.24	5.20	152.27	5.20	138.96	5.20	<0.001	<0.001
	Rural	54.87	4.24	100.05	4.24	150.07	4.24	<0.001	
Thiamine (mg/4.2MJ)	Urban	0.40	0.01	0.53	0.01	0.44	0.01	0.102	<0.001
	Rural	0.36	0.01	0.48	0.01	0.58	0.01	<0.001	
Riboflavin (mg/4.2MJ)	Urban	0.37	0.02	0.57	0.02	0.42	0.02	0.059	<0.001
	Rural	0.21	0.01	0.34	0.01	0.49	0.01	<0.001	
Niacin (mg/4.2MJ)	Urban	4.10	0.19	6.83	0.19	5.55	0.19	<0.001	<0.001
	Rural	2.74	0.17	5.14	0.17	6.85	0.17	<0.001	
Vitamin C (mg/4.2MJ)	Urban	11.53	1.11	22.29	1.11	17.11	1.11	<0.001	<0.001
	Rural	3.97	1.00	10.45	1.00	28.98	1.00	0.001	

Values are expressed as estimated marginal means (EMM) and standard error (SE). TE, Total energy

¹ Type III tests of fixed effects with time as continuous variable were performed to determine significant trends over time within groups, values significant p < 0.05

Macronutrient distribution	Locality	WOMEN (Rural n=295; Urban n=220)								Time effect ¹	Time*Locality interaction effect ²
		2005		2010		2015					
		EMM	SE	EMM	SE	EMM	SE				

² Type III tests of fixed effects with time as continuous variable were performed to determine significant differences between rural and urban groups. values significant p < 0.05

5.4.6 Differences in macronutrient distribution of rural and urban men and women by year

To explore the macronutrient distribution in more detail, further analysis (fixed effect models) was done to investigate the changes in macronutrient distribution by year. These results are presented below and should be viewed together with Figures 5-2 and 5-3 and Supplementary Tables 5-1 to 5-4.

Proportion of total energy from fats

Figure 5-2 clearly illustrates that the observed trends for the urban and rural groups differ (see interaction p-values in Tables 5-4 and 5-5). For rural groups, an upward sloping line is observed from 2005 to 2015, and both the 2005 to 2010 comparison as well as the 2005 to 2015 comparison indicate significant differences.

However, when looking at the urban groups, one can observe a similar upward sloping line from 2005 to 2010, but from 2010 to 2015, the slope decreased again to a mean value similar to where it started in 2005. A comparison of 2005 and 2015 values indicate a non-significant difference, whereas the comparison between 2005 and 2010, and 2010 and 2015 show significant differences. The only exception to the above is found in urban groups for proportion of total energy from MUFA, where the slope from 2010 to 2015 does not decrease to the extent that the 2015 mean value returns to the 2005 level, and the difference, therefore, between the 2005 and 2015 mean value is significant.

This, in part, explains the absence of significant trend analysis observed for the urban group over time, as presented by the p-trend value in Table 5-3 and 5-4.

In terms of dietary recommendations, the rural groups were well below the recommended maximum proportion of < 30% ⁽³⁵⁻³⁹⁾ for total fat in 2005 and 2010 but surpassed that by 2015 (Figure 5-2). The urban groups, on the other hand, were already just below the recommendation in 2005, surpassed it in 2010, but returned to its baseline state in 2015. Similar observations were made for the percentage contributions of saturated fat and PUFA in the urban groups. For the rural groups, the percentage contributions of saturated fat and PUFA were considerably lower

than the respective recommendation of <10%⁽³⁵⁻³⁹⁾ in 2005, followed by a continuous steep increase that brought it noticeably closer to the recommendation in 2015.



Figure 5-2: Percentage contribution of fat to total energy intake of rural and urban men and women

Percentages are reported as estimated marginal means

TE, Total energy; MUFA, mono-unsaturated fatty acids; PUFA, polyunsaturated fatty acids.

Recommendations are based on global recommendations and the South African Food - Based Dietary Guidelines⁽³⁵⁻³⁹⁾

Proportion of total energy from protein and carbohydrates

At first glance, the figures presented for protein and carbohydrates look similar to the figures presented above for fat, and visually illustrate that the trends for the urban and rural groups differ. As above, the graph lines for rural groups are marked by an upward slope from 2005 to 2015. The urban groups, again, show an upward slope from 2005 to 2010, but a downward slope from 2010 to 2015. For urban men, the slope decreases again to a mean value similar to where it started in 2005, with no significant changes observed between the 2005 and 2015 mean values for total protein, plant protein, animal protein and carbohydrate proportions of total energy. For urban women, however, the mean value of the proportion of total energy from protein, animal protein and total carbohydrates differed significantly between 2005 and 2010, 2010 and 2015 and

2005 and 2015. In other words, even though there is a downward slope that is observed, the slope is attenuated and does not decrease to the mean value of 2005.

In terms of recommendations, Figure 5-3 shows that all groups were well within the recommended range (10–35%)⁽⁴⁰⁾ for the percentage contribution of total protein to total energy intake. In the case of total carbohydrates, the rural groups were well below the recommended upper limit of 65%⁽⁴⁰⁾ but surpassed it by 2015. The urban groups had already surpassed it in 2010, but their trend indicates that they might be moving below the recommendation if the trend were to continue.

Proportion of total energy from added sugar

The graph for added sugar stands out from the others, as similar trends are seen for rural and urban men and women and no significant interaction effect between urban and rural groups was found (see Table 5-4 and 5-5). In all groups the proportion of total energy from added sugar rose from 2005 to 2010 and then slightly decreased to 2015, with the 2005 mean value differing significantly from the 2010 and 2015 mean values in all cases.

Both rural men and women were on the recommended 5% ⁽⁴¹⁾ for added sugar in 2005, moved well beyond it in 2010 and remained above the recommendation in 2015. The consumption of urban men and women was already at 8% in 2005 and remained above the recommendation throughout the study period (2005–2015).

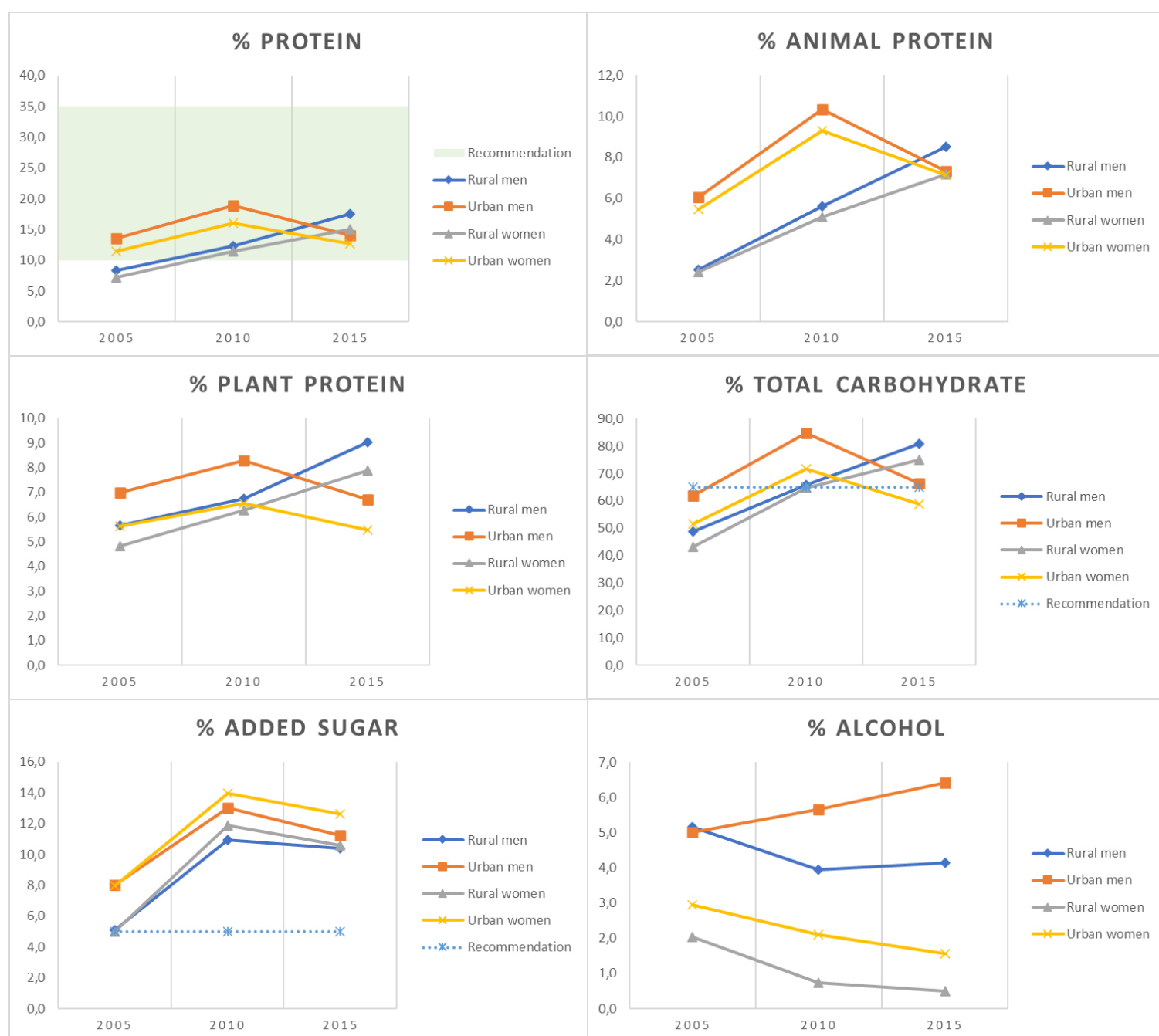


Figure 5-3: Percentage contribution of protein, carbohydrates and alcohol to total energy intake of rural and urban men and women

Percentages are reported as estimated marginal means

TE, total energy

Recommendations: % protein (Dietary reference intakes)⁽⁴⁰⁾, % total carbohydrate (Dietary reference intakes)⁽⁴⁰⁾, % added sugar (WHO)⁽⁴¹⁾.

5.4.7 Trends in nutrient density for rural and urban men and women

The trends in nutrient density of rural and urban men and women are presented in Tables 5-4 and 5-5.

Rural men showed improved nutrient density with regard to dietary fibre, iron, zinc, calcium, thiamine, riboflavin, niacin and vitamin C ($p < 0.001$ for all), while only the nutrient density of niacin improved for urban men ($p=0.001$).

Both rural and urban women showed improvements in the nutrient density of iron, zinc, calcium, niacin, vitamin C ($p < 0.005$ for all trends), while the nutrient density of dietary fibre, thiamine and riboflavin improved only for rural women ($p=0.001$ for all).

The interaction effect was significant for all nutrient density trends comparing rural and urban men, as well as rural and urban women ($p < 0.001$ for interaction effect).

Supplementary table 5-1: Differences in mean energy and nutrient intake of rural and urban men, by year

Energy and nutrients	Locality	MEN (Rural n=109; Urban n=98)							
		2005		2010		2015		p-value for change between 2010-2015	p-value for change between 2005-2015
		EMM	SE	EMM	SE	EMM	SE		
Energy (MJ)	Urban	10.90	498.14	14.40	498.14	11.36	498.14	<0.001	0.478
	Rural	7.60	472.88	10.22	472.88	13.26	472.88	<0.001	<0.001
Total protein (g)	Urban	79.74	4.30	111.25	4.30	83.10	4.30	<0.001	0.530
	Rural	48.91	4.63	72.85	4.63	103.49	4.63	<0.001	<0.001
Plant protein (g)	Urban	41.19	1.86	48.71	1.86	39.56	1.86	<0.001	0.651
	Rural	33.32	2.19	39.74	2.19	53.22	2.19	<0.001	<0.001
Animal protein (g)	Urban	35.71	3.08	60.89	3.08	43.183	3.08	<0.001	0.065
	Rural	14.91	3.56	33.00	3.56	50.14	3.56	<0.001	<0.001
Total fat (g)	Urban	73.17	4.60	102.88	4.60	79.52	4.60	<0.001	0.284
	Rural	35.78	4.15	62.93	4.15	91.37	4.15	<0.001	<0.001
Saturated fat (g)	Urban	18.37	1.36	28.05	1.36	21.43	1.36	<0.001	0.079
	Rural	7.79	1.26	17.71	1.26	24.15	1.26	<0.001	<0.001
Monounsaturated fat (g)	Urban	20.53	1.53	31.17	1.53	24.77	1.53	0.001	0.030
	Rural	8.36	1.54	17.63	1.54	28.85	1.54	<0.001	<0.001
Polyunsaturated fat (g)	Urban	21.07	1.48	27.61	1.48	22.00	1.48	0.005	0.638
	Rural	11.82	1.29	17.75	1.29	25.19	1.29	<0.001	<0.001
Cholesterol (mg)	Urban	275.31	25.92	463.08	25.92	346.38	25.92	0.001	0.046
	Rural	124.51	26.34	257.44	26.34	357.18	26.34	0.004	<0.001
Total carbohydrate (g)	Urban	364.13	17.31	499.17	17.31	389.97	17.31	<0.001	0.270
	Rural	287.52	17.49	388.02	17.49	476.11	17.49	<0.001	<0.001
Added sugar (g)	Urban	47.27	6.04	76.67	6.04	66.08	6.04	0.175	0.016
	Rural	30.13	4.84	64.50	4.84	61.20	4.84	0.613	<0.001
Dietary fibre (g)	Urban	29.67	1.43	37.50	1.43	27.30	1.43	<0.001	0.220
	Rural	20.80	1.59	27.30	1.59	35.78	1.59	<0.001	<0.001
Calcium (mg)	Urban	508.64	37.20	705.75	37.20	607.68	37.20	0.056	0.054
	Rural	272.13	35.97	376.90	35.97	655.53	35.97	<0.001	<0.001
Iron (mg)	Urban	17.78	0.97	26.14	0.97	20.33	0.97	<0.001	0.056
	Rural	13.40	0.95	19.27	0.95	25.34	0.95	<0.001	<0.001
Magnesium (mg)	Urban	449.42	22.36	515.23	22.36	454.51	22.36	0.041	0.864
	Rural	360.53	23.73	405.47	23.73	545.61	23.73	<0.001	<0.001
Phosphorus (mg)	Urban	1277.12	64.18	1673.21	64.18	1378.64	64.18	0.001	0.241
	Rural	929.55	65.63	1172.61	65.63	1652.29	65.63	<0.001	<0.001
Potassium (mg)	Urban	2423.42	131.27	3565.28	131.27	2780.41	131.27	<0.001	0.040
	Rural	1494.31	118.62	2480.34	118.62	3334.37	118.62	<0.001	<0.001
Zinc (mg)	Urban	14.06	0.81	20.95	0.81	15.39	0.81	<0.001	0.218
	Rural	9.57	0.83	14.72	0.83	19.78	0.83	<0.001	<0.001
Copper (mg)	Urban	1.68	0.11	2.25	0.11	1.92	0.11	0.022	0.097
	Rural	1.18	0.09	1.61	0.09	2.38	0.09	<0.001	<0.001

Energy and nutrients	Locality	MEN (Rural n=109; Urban n=98)							
		2005		2010		2015		p-value for change between 2010-2015	p-value for change between 2005-2015
		EMM	SE	EMM	SE	EMM	SE		
Manganese (µg)	Urban	3206.21	211.95	4373.21	211.95	3847.66	211.95	0.075	0.030
	Rural	2500.46	254.68	3467.83	254.68	4595.78	254.68	0.001	<0.001
Vitamin A (µg)	Urban	1160.78	148.21	1933.25	148.21	1525.91	148.21	0.042	0.068
	Rural	582.56	107.32	1065.82	107.32	1667.25	107.32	<0.001	<0.001
Thiamine (mg)	Urban	2.20	0.11	2.94	0.11	2.22	0.11	<0.001	0.867
	Rural	1.76	0.11	2.20	0.11	2.83	0.11	<0.001	0.010
Riboflavin (mg)	Urban	1.76	0.11	2.60	0.11	2.02	0.11	<0.001	0.099
	Rural	1.11	0.12	1.55	0.12	2.28	0.12	<0.001	<0.001
Niacin (mg)	Urban	20.27	1.35	36.61	1.35	27.22	1.35	<0.001	<0.001
	Rural	13.72	1.50	24.27	1.50	32.90	1.50	<0.001	<0.001
Vitamin B ₆ (mg)	Urban	2.09	0.20	5.10	0.20	3.46	0.20	<0.001	<0.001
	Rural	1.37	0.22	3.41	0.22	3.91	0.22	0.091	<0.001
Folate (µg)	Urban	513.65	32.84	723.61	32.84	596.62	32.84	0.005	0.063
	Rural	397.40	31.65	515.15	31.65	685.87	31.65	<0.001	<0.001
Vitamin B ₁₂ (µg)	Urban	6.72	1.04	9.94	1.04	8.71	1.04	0.384	0.160
	Rural	2.47	1.12	4.41	1.12	10.01	1.12	<0.001	<0.001
Pantothenic acid (mg)	Urban	5.52	0.40	9.51	0.40	6.68	0.40	<0.001	0.026
	Rural	3.40	0.46	5.25	0.46	8.11	0.46	<0.001	<0.001
Biotin (µg)	Urban	53.02	4.90	83.16	4.90	62.50	4.90	0.002	0.152
	Rural	27.85	3.22	44.24	3.22	64.79	3.22	<0.001	<0.001
Vitamin C (mg)	Urban	52.48	6.43	87.89	6.43	57.53	6.43	0.001	0.579
	Rural	13.69	5.82	45.01	5.82	77.48	5.82	<0.001	<0.001
Vitamin D (mg)	Urban	3.60	0.41	6.53	0.41	5.21	0.41	0.023	0.006
	Rural	1.91	0.39	3.79	0.39	4.86	0.39	0.037	<0.001
Vitamin E (mg)	Urban	14.27	1.04	18.87	1.04	15.58	1.04	0.026	0.374
	Rural	9.38	0.92	13.63	0.92	17.00	0.92	0.005	<0.001
Alcohol (g)	Urban	17.27	3.23	19.56	3.23	22.18	3.23	0.477	0.183
	Rural	17.83	2.95	13.63	2.95	14.27	2.95	0.861	0.332

Values are expressed as estimated marginal means (EMM) and standard error (SE). TE, Total energy

Supplementary table 5-2: Differences in mean energy and nutrient intake of rural and urban women, by year

Energy and nutrients	Locality	WOMEN (Rural n=295; Urban n=220)							
		2005		2010		2015		p-value for change between 2010-2015	p-value for change between 2005-2015
		EMM	SE	EMM	SE	EMM	SE		
Energy (MJ)	Urban	9.36	299.29	12.20	299.29	9.84	299.29	<0.001	0.233
	Rural	6.60	256.93	9.93	256.93	11.89	256.93	<0.001	<0.001
Total protein (g)	Urban	67.43	2.51	94.55	2.51	74.69	2.51	<0.001	0.031
	Rural	42.81	2.10	67.48	2.10	88.66	2.10	<0.001	<0.001
Plant protein (g)	Urban	33.18	1.08	38.56	1.08	32.27	1.08	<0.001	0.536
	Rural	28.35	1.14	37.04	1.14	46.37	1.14	<0.001	<0.001
Animal protein (g)	Urban	32.12	1.84	54.73	1.84	42.08	1.84	<0.001	<0.001
	Rural	14.10	1.42	30.06	1.42	42.12	1.42	<0.001	<0.001
Total fat (g)	Urban	70.23	2.92	93.43	2.92	73.73	2.92	<0.001	0.360
	Rural	35.25	2.33	67.76	2.33	82.90	2.33	<0.001	<0.001
Saturated fat (g)	Urban	18.06	0.81	25.82	0.81	19.98	0.81	<0.001	0.078
	Rural	7.75	0.70	19.00	0.70	21.90	0.70	<0.001	0.033
Monounsaturated fat (g)	Urban	20.31	0.96	28.88	0.96	23.00	0.96	0.001	0.030
	Rural	8.26	0.77	18.59	0.77	24.51	0.77	<0.001	<0.001
Polyunsaturated fat (g)	Urban	20.67	1.02	26.46	1.02	21.25	1.02	0.005	0.670
	Rural	12.07	0.88	20.87	0.88	25.80	0.88	<0.001	<0.001
Cholesterol (mg)	Urban	250.03	16.16	418.35	16.16	278.91	16.16	0.001	0.002
	Rural	127.60	11.84	224.83	11.84	304.26	11.84	0.004	<0.001
Total carbohydrate (g)	Urban	302.96	10.18	421.67	10.18	345.45	10.18	<0.001	0.270
	Rural	254.08	9.59	381.10	9.59	441.00	9.59	<0.001	<0.001
Added sugar (g)	Urban	46.94	3.64	82.17	3.64	74.28	3.64	0.108	0.016
	Rural	29.45	3.48	69.87	3.48	62.24	3.48	0.113	<0.001
Dietary fibre (g)	Urban	23.36	0.86	32.47	0.86	25.11	0.86	<0.001	0.132
	Rural	18.39	0.87	26.45	0.87	32.87	0.87	<0.001	<0.001
Calcium (mg)	Urban	462.99	21.85	639.52	21.85	583.64	21.85	0.065	<0.001
	Rural	230.44	17.83	420.19	17.83	630.29	17.83	<0.001	<0.001
Iron (mg)	Urban	14.25	0.54	21.45	0.54	17.47	0.54	<0.001	<0.001
	Rural	11.87	0.49	18.00	0.49	22.39	0.49	<0.001	<0.001
Magnesium (mg)	Urban	349.73	10.72	402.41	10.72	348.64	10.72	<0.001	0.940
	Rural	280.48	11.16	370.12	11.16	466.54	11.16	<0.001	<0.001
Phosphorus (mg)	Urban	1079.27	35.59	1364.23	35.59	1126.52	35.59	0.001	0.323
	Rural	758.31	31.48	1094.93	31.48	1408.02	31.48	<0.001	<0.001
Potassium (mg)	Urban	2120.31	80.86	3233.46	80.86	2570.33	80.86	<0.001	<0.001
	Rural	1356.98	73.41	2561.58	73.41	3295.65	73.41	<0.001	<0.001
Zinc (mg)	Urban	11.33	0.43	16.25	0.43	13.06	0.43	<0.001	0.003
	Rural	8.26	0.39	13.09	0.39	16.63	0.39	<0.001	<0.001

Energy and nutrients	Locality	WOMEN (Rural n=295; Urban n=220)							
		2005		2010		2015		p-value for change between 2010-2015	p-value for change between 2005-2015
		EMM	SE	EMM	SE	EMM	SE		
Copper (mg)	Urban	1.49	0.06	2.06	0.06	1.69	0.06	<0.001	0.015
	Rural	1.05	0.06	1.57	0.06	2.38	0.06	<0.001	<0.001
Manganese (µg)	Urban	2501.55	102.20	3480.68	102.20	2942.26	102.20	<0.001	0.002
	Rural	1692.01	107.72	2835.76	107.72	3393.35	107.72	<0.001	<0.001
Vitamin A (µg)	Urban	1139.00	104.64	2151.73	104.64	1504.95	104.64	<0.001	0.042
	Rural	581.03	88.00	1109.79	88.00	1919.57	88.00	<0.001	<0.001
Thiamine (mg)	Urban	1.69	0.06	2.21	0.06	1.83	0.06	<0.001	0.088
	Rural	1.53	0.06	2.01	0.06	2.42	0.06	<0.001	<0.001
Riboflavin (mg)	Urban	1.55	0.08	2.39	0.08	1.76	0.08	<0.001	0.044
	Rural	0.87	0.06	1.42	0.06	2.05	0.06	<0.001	<0.001
Niacin (mg)	Urban	17.22	0.79	28.70	0.79	23.32	0.79	<0.001	<0.001
	Rural	11.51	0.72	21.57	0.72	28.75	0.72	<0.001	<0.001
Vitamin B ₆ (mg)	Urban	1.70	0.12	3.93	0.12	3.11	0.12	<0.001	<0.001
	Rural	1.24	0.10	2.83	0.10	3.36	0.10	<0.001	<0.001
Folate (µg)	Urban	398.81	19.44	569.81	19.44	474.82	19.44	0.005	0.004
	Rural	349.08	18.87	436.96	18.87	568.34	18.87	<0.001	<0.001
Vitamin B ₁₂ (µg)	Urban	6.74	0.68	10.36	0.68	7.38	0.68	0.001	0.458
	Rural	2.43	0.54	3.86	0.54	9.66	0.54	<0.001	<0.001
Pantothenic acid (mg)	Urban	4.88	0.25	8.30	0.25	6.31	0.25	<0.001	<0.001
	Rural	2.95	0.21	5.19	0.21	7.28	0.21	<0.001	<0.001
Biotin (µg)	Urban	44.81	3.46	77.25	3.46	54.43	3.46	<0.001	0.042
	Rural	26.15	1.72	39.90	1.72	60.24	1.72	<0.001	<0.001
Vitamin C (mg)	Urban	48.42	4.66	93.62	4.66	71.86	4.66	0.001	<0.001
	Rural	16.68	4.21	43.88	4.21	121.72	4.21	<0.001	<0.001
Vitamin D (mg)	Urban	3.33	0.24	5.59	0.24	3.92	0.24	<0.001	0.081
	Rural	2.11	0.19	3.48	0.19	3.93	0.19	0.061	<0.001
Vitamin E (mg)	Urban	13.53	0.69	17.70	0.69	14.03	0.69	<0.001	0.591
	Rural	9.41	0.60	14.31	0.60	17.69	0.60	<0.001	<0.001
Alcohol (g)	Urban	10.20	1.27	7.27	1.27	5.40	1.27	0.244	0.003
	Rural	7.05	0.87	2.52	0.87	1.75	0.87	0.497	<0.001
Values are expressed as estimated marginal means (EMM) and standard error (SE). TE, Total energy									

Supplementary table 5-3: Differences in mean macronutrient distribution and nutrient density of rural and urban men, by year

Macronutrient distribution	Locality	MEN (Rural n=109; Urban n=98)							
		2005		2010		2015		p-value for change between 2010-2015	p-value for change from between 2005-2015
		EMM	SE	EMM	SE	EMM	SE		
% of TE from protein	Urban	13.56	0.73	18.91	0.73	14.13	0.73	<0.001	0.554
	Rural	8.31	0.79	12.39	0.79	17.59	0.79	<0.001	<0.001
% of TE from animal protein	Urban	6.07	0.52	10.35	0.52	7.34	0.52	<0.001	0.065
	Rural	2.53	0.61	5.61	0.61	8.52	0.61	<0.001	<0.001
% of TE from plant protein	Urban	7.00	0.32	8.28	0.32	6.73	0.32	<0.001	0.516
	Rural	5.66	0.37	6.76	0.37	9.05	0.37	<0.001	<0.001
% of TE from total fat	Urban	27.81	1.75	39.09	1.75	30.22	1.75	<0.001	0.284
	Rural	13.60	1.58	23.91	1.58	34.72	1.58	<0.001	<0.001
% of TE from saturated fat	Urban	6.98	0.52	10.66	0.52	8.14	0.52	<0.001	0.079
	Rural	2.96	0.48	6.73	0.48	9.18	0.48	<0.001	<0.001
% of TE from monounsaturated fat	Urban	7.80	0.58	11.84	0.58	9.41	0.58	0.001	0.030
	Rural	3.18	0.59	6.70	0.59	10.96	0.59	<0.001	<0.001
% of TE from polyunsaturated fat	Urban	8.00	0.56	10.49	0.56	8.36	0.56	0.005	0.638
	Rural	4.49	0.49	6.74	0.49	9.57	0.49	<0.001	<0.001
% of TE from total carbohydrate	Urban	61.90	2.94	84.86	2.94	66.30	2.94	<0.001	0.270
	Rural	48.88	2.97	65.96	2.97	80.94	2.97	<0.001	<0.001
% of TE from added sugar	Urban	8.04	1.03	13.03	1.03	11.23	1.03	0.175	0.016
	Rural	5.12	0.82	10.97	0.82	10.40	0.82	0.613	<0.001
% of TE from alcohol	Urban	5.01	0.94	5.67	0.94	6.43	0.94	0.477	0.183
	Rural	5.17	0.85	3.95	0.85	4.14	0.85	0.861	0.332
Nutrient density									
Dietary fibre (g/4.2MJ)	Urban	7.07	0.34	8.93	0.34	6.50	0.34	<0.001	0.220
	Rural	4.95	0.38	6.50	0.38	8.52	0.38	<0.001	<0.001
Iron (mg/4.2MJ)	Urban	4.23	0.23	6.22	0.23	4.84	0.23	<0.001	0.056
	Rural	3.19	0.23	4.59	0.23	6.03	0.23	<0.001	<0.001
Zinc (mg/4.2MJ)	Urban	3.35	0.19	4.99	0.19	3.66	0.19	<0.001	0.218
	Rural	2.28	0.20	3.51	0.20	4.71	0.20	<0.001	<0.001
Calcium (mg/4.2MJ)	Urban	121.10	8.86	168.04	8.86	144.69	8.86	<0.001	0.054
	Rural	64.79	8.56	89.74	8.56	156.08	8.56	<0.001	<0.001
Thiamine (mg/4.2MJ)	Urban	0.52	0.03	0.70	0.03	0.53	0.03	0.158	0.867
	Rural	0.42	0.03	0.52	0.03	0.67	0.03	<0.001	<0.001
Riboflavin (mg/4.2MJ)	Urban	0.42	0.03	0.62	0.03	0.48	0.03	<0.001	0.099
	Rural	0.26	0.03	0.37	0.03	0.54	0.03	<0.001	<0.001
Niacin (mg/4.2MJ)	Urban	4.83	0.32	8.72	0.32	6.48	0.32	<0.001	<0.001
	Rural	3.27	0.36	5.78	0.36	7.83	0.36	<0.001	<0.001
Vitamin C (mg/4.2MJ)	Urban	12.50	1.53	20.93	1.53	13.70	1.53	0.001	0.579
	Rural	3.26	1.39	10.72	1.39	18.45	1.39	<0.001	<0.001
Values are expressed as estimated marginal means (EMM) and standard error (SE). TE, Total energy									

Supplementary table 5-4: Differences in mean macronutrient distribution and nutrient density of rural and urban women, by year

Macronutrient distribution	Locality	WOMEN (Rural n=295; Urban n=220)							
		2005		2010		2015		p-value for change between 2010-2015	p-value for change between 2005-2015
		EMM	SE	EMM	SE	EMM	SE		
% of TE from protein	Urban	11.46	0.43	16.07	0.43	12.70	0.43	<0.001	0.031
	Rural	7.28	0.36	11.47	0.36	15.07	0.36	<0.001	<0.001
% of TE from animal protein	Urban	5.46	0.31	9.30	0.31	7.15	0.31	<0.001	<0.001
	Rural	2.40	0.24	5.11	0.24	7.16	0.24	<0.001	<0.001
% of TE from plant protein	Urban	5.64	0.18	6.56	0.18	5.49	0.18	<0.001	0.536
	Rural	4.82	0.19	6.30	0.19	7.88	0.19	<0.001	<0.001
% of TE from total fat	Urban	26.69	1.11	35.50	1.11	28.02	1.11	<0.001	0.360
	Rural	13.39	0.89	25.75	0.89	31.50	0.89	<0.001	<0.001
% of TE from saturated fat	Urban	6.86	0.31	9.81	0.31	7.59	0.31	<0.001	0.078
	Rural	2.94	0.27	7.22	0.27	8.32	0.27	0.002	<0.001
% of TE from monounsaturated fat	Urban	7.72	0.37	10.98	0.37	8.74	0.37	<0.001	0.033
	Rural	3.14	0.29	7.06	0.29	9.31	0.29	<0.001	<0.001
% of TE from polyunsaturated fat	Urban	7.85	0.39	10.06	0.39	8.07	0.39	<0.001	0.670
	Rural	4.59	0.33	7.93	0.33	9.80	0.33	<0.001	<0.001
% of TE from total carbohydrate	Urban	51.50	1.73	71.68	1.73	58.73	1.73	<0.001	0.002
	Rural	43.19	1.63	64.79	1.63	74.97	1.63	<0.001	<0.001
% of TE from added sugar	Urban	7.98	0.62	13.97	0.62	12.63	0.62	0.108	<0.001
	Rural	5.01	0.59	11.88	0.59	10.58	0.59	0.113	<0.001
% of TE from alcohol	Urban	2.96	0.37	2.11	0.37	1.57	0.37	0.244	0.003
	Rural	2.04	0.25	0.73	0.25	0.51	0.25	0.497	0.183
Nutrient density									
Dietary fibre (g/4.2MJ)	Urban	5.56	0.20	7.73	0.20	5.98	0.20	<0.001	0.132
	Rural	4.38	0.21	6.30	0.21	7.83	0.21	<0.001	<0.001
Iron (mg/4.2MJ)	Urban	3.39	0.13	5.11	0.13	4.16	0.13	<0.001	<0.001
	Rural	2.83	0.12	4.29	0.12	5.33	0.12	<0.001	<0.001
Zinc (mg/4.2MJ)	Urban	2.70	0.10	3.87	0.10	3.11	0.10	<0.001	0.003
	Rural	1.97	0.09	3.12	0.09	3.96	0.09	<0.001	<0.001
Calcium (mg/4.2MJ)	Urban	110.24	5.20	152.27	5.20	138.96	5.20	<0.001	0.065
	Rural	54.87	4.24	100.05	4.24	150.07	4.24	<0.001	<0.001
Thiamine (mg/4.2MJ)	Urban	0.40	0.01	0.53	0.01	0.44	0.01	<0.001	0.088
	Rural	0.36	0.01	0.48	0.01	0.58	0.01	<0.001	<0.001
Riboflavin (mg/4.2MJ)	Urban	0.37	0.02	0.57	0.02	0.42	0.02	<0.001	0.044
	Rural	0.21	0.01	0.34	0.01	0.49	0.01	<0.001	<0.001
Niacin (mg/4.2MJ)	Urban	4.10	0.19	6.83	0.19	5.55	0.19	<0.001	<0.001
	Rural	2.74	0.17	5.14	0.17	6.85	0.17	<0.001	<0.001
Vitamin C (mg/4.2MJ)	Urban	11.53	1.11	22.29	1.11	17.11	1.11	0.001	<0.001
	Rural	3.97	1.00	10.45	1.00	28.98	1.00	<0.001	<0.001

Values are expressed as estimated marginal means (EMM) and standard error (SE). TE, Total energy

5.5 Discussion

This study presents the results of in-depth trend analysis of energy and nutrient intakes of urban and rural groups of the PURE-SA-NWP study, over the period 2005 to 2015. Our findings indicate that the nutrition transition and its direction differ significantly between urban and rural groups. Furthermore, it highlights the rapidness of dietary intake changes that occurred in the rural participants. The discussion will focus on key issues emerging from the results as well as framing the above within the context of the South African nutrition transition.

Trends in energy and nutrient intakes and macronutrient distribution

In general, the trends reported for energy and nutrient intakes differed significantly between the rural and urban participants. Over the study period, the observed trends reflected continuous increases in the total energy intake of both rural men and women (respective increases of 5.7 MJ and 5.29 MJ), while no significant p-trend was observed for either urban men or women. When considering the mean differences in energy intake by year (as presented in Supplementary Tables 5-1 and 5-2), it becomes evident that the absence of a significant trend in energy intake of urban men and women was the result of a significant decrease in the energy intake for both men and women from 2010 to 2015. Similar findings were reported in China, where prospective data showed significant decreases in the energy intake of urban groups ⁽⁴²⁾ and Chinese urban participants were shown to consume less energy than rural dwellers ⁽⁴³⁾.

The decrease in energy intake from 2010 to 2015 by the urban groups observed in our own study meant that the rural groups not only “caught up” with their urban counterparts in terms of energy intake but surpassed them by 2015. This higher intake of total energy subsequently led to higher intakes in all macro- and micronutrients (except for the trend of alcohol intake that decreased for rural women and remained unchanged for rural men).

In 2015, Mchiza and colleagues ⁽⁴⁴⁾ published a review of dietary surveys in adult South African groups from 2000 to 2015 to address the lack of national dietary intake data at the time. They reported that, given the available data, energy intakes varied between being low, particularly in rural areas, to being very high in urban areas. At the time, the PURE 2015 data were not yet available and only PURE 2005 and PURE 2010 data were included in the analysis. While the above review did not include only PURE data, the results presented in our article highlight the suddenness with which changes can happen within a specific group in a relatively short time frame. It also emphasises the importance of prospective studies being done at timeous intervals in order to capture dietary changes.

Possible explanations for the continuous increase in energy intake observed in the rural groups may be the improved road access that the rural community has gained to markets or to the development of a local shopping centre during this period ⁽⁴⁾ and the overall food environment that has changed significantly over a 10-year period. D'Haese & Van Huylbroeck reasoned that the importance of supermarkets for household food should not be underestimated, given that, in addition to providing ultra-processed foods high in total fat, sugar and salt, and low in dietary fibre, they often provide good quality products at lower prices ⁽⁴⁵⁾. In addition, through buying food instead of producing food, more time is made available to invest in other income-generating activities. Unfortunately, with ease of access, consumption often increases. In Kenya, supermarket purchases were shown to contribute to several nutrition-related NCD risk factors, including overweight and obesity, as people who buy in supermarkets tend to consume more energy through dietary intake ⁽⁴⁶⁾.

Key concerns with regard to macronutrient distribution

Our results also indicate a disparity in the trends observed in terms of macronutrient distribution between urban and rural groups. Overall, the trends in the percentages of energy from fat (total fat and all types of fat), protein (total protein, animal- and plant protein) and total carbohydrates continuously increased over the entire study period for both rural men and women. In contrast, the trends in the percentage energy from fat remained largely unchanged in the urban groups, with only the trend in MUFA intake showing a significant increase. In our study, meat and meat products contribute to 35% of MUFA intake through consumption of chicken, chicken feet, red meat and pork (data not shown). In a recent study by Zong and colleagues ⁽⁴⁷⁾, it was reported that different associations in relation to CVD risk exist in terms of MUFAs from animal sources and those from plant sources. They furthermore stated that plant-based foods were the preferable sources of MUFAs for the prevention of cardiovascular heart diseases. This is also in line with a Chinese study that found MUFAs from plant sources, but not animal sources, to be associated with lower mortality ⁽⁴⁸⁾. Trevisan *et al.* ⁽⁴⁹⁾ found MUFA from animal sources to be associated with higher mortality, while the opposite association was found for plant-sourced MUFAs.

In terms of dietary recommendations, the total fat intake of the rural groups as well as urban men remains worrisomely above the global recommendation of 30% ⁽³⁵⁻³⁹⁾ and is probably also an indication that high fat meat sources were being purchased at lower prices. Urban women are just slightly below the recommendation at 28%. The upward trend for the percentage contribution to TE from saturated fat for the rural groups should be reason for concern as it will soon be in excess of the recommendation of <10%⁽³⁵⁻³⁹⁾.

Alarmingly high added sugar intake continues in urban and rural groups

Added sugar intake amongst South Africans has been alarmingly high in recent years. In the South African Breast Cancer Study, urban Soweto women had a mean added sugar intake of 12% of total energy (control group) ⁽¹⁷⁾, while the Cardiovascular Risk in Black South Africans study reported mean added sugar intakes above 10% for all groups, except for men younger than 44 years, who had intake of 9.5% ⁽²⁰⁾.

Similar to the above studies, our findings indicate that by 2010 and 2015, added sugar intake was above the latest 2015 WHO ⁽⁴¹⁾ recommendation of <5% for all groups. For now, our results still agree with the South Africa National Health and Nutrition Examination Survey (SANHANES-1) study that reported lowest sugar scores in rural areas and in places where the least urbanisation took place, such as the North West province ⁽⁵⁰⁾. While a downward slope is observed for the percentage contribution of added sugar intake to TE from 2010 to 2015, the change is not significant and the percentage contribution of added sugar to TE remains worryingly high.

In Mexico, sugar-sweetened beverage (SSB) consumption has previously been identified as the major contributor to added sugar intake ^(51, 52). Wentzel-Viljoen *et al.* ⁽⁵³⁾ reported sugar-sweetened beverages (SSB) to be the main contributor to the increase in added sugar intake of both rural and urban participants from 2005 to 2010 in PURE-SA-NWP. For the 2015 PURE-SA-NWP data, SSB and sugar were the main contributors to added sugar, contributing 23% and 52% respectively (data not shown). Other South African studies have also reported on the high intake of SSB by both urban and rural lower-income communities ^(54, 55). Cheap SSB are chosen above portions of fruits and vegetables in the diet, and often undermine the consumption of the latter ⁽⁵⁵⁾.

The nutrition transition in South Africa

These sudden dietary intake changes should not come as a surprise. From the early 2000s, Popkin has alluded several times to the uniqueness of the experiences of the nutrition transition as well as the rapidness of change in low- and middle- income countries ^(12, 13, 56-58). Popkin specifically refers to an accelerated time period in low- and middle-income countries from the “receding famine pattern” to the one dominated by nutrition-related NCDs ⁽¹³⁾. The above has also played out in other developing countries. Rural groups in India underwent dietary changes so rapid that they “caught-up” with those of urban groups as a result of rural villages urbanising while urban intakes were equalising ⁽⁵⁹⁾.

The different stages of the nutrition transition have been thoroughly reported for rural and urban groups in South Africa ⁽⁸⁾. It seems that the rural participants were always a stage behind the urban participants in terms of the nutrition transition. In the early 2000s, rural groups were

reported to be in the hunter-gatherer stage of the nutrition transition while urban groups progressed to the receding famine stage and the degenerative disease stage ^(7, 8, 14). The differences observed between rural and urban groups indicate that, while both urban and rural groups were still undergoing a rapid nutrition transition from 2005 to 2010⁽⁴⁾, by 2015 the rural group continued to transition at a rapid pace, while the urban group seemingly reached some sort of turning point in 2010. This does not, of course, necessarily point toward healthier eating habits as their dietary intake was still far from prudent. It may, however, be indicative of a slower progression of the nutrition transition and may in time lead to healthier eating patterns, as these groups transition from the degenerative disease stage of the nutrition transition to the behavioural change stage.

While the association between dietary intake and CVD risk fall outside the scope of this article, the sharp increase in obesity and WC and the decrease in physical activity observed from 2005 to 2015 may be indicative of the typical nutrition transition dynamics.

Limitations of this study

One limitation of this study is that QFFQs rely on information recall, which may be increasingly difficult as participants age. To effectively limit possible recall bias, our fieldworkers were well trained and used various aids, such as example products (such as a potato and an apple), a picture book, household measuring cups, spoons and crockery, to assist participants in quantifying their food consumption.

Although the PURE-SA-NWP study did collect household income data, these data could not be used prospectively as the income brackets on the questionnaires differed between the various data collections and could thus not be accurately compared. Information regarding changes in household income could have been valuable in explaining some of our results.

5.6 Conclusion

From this in-depth trend analysis of energy and nutrient intakes of urban and rural participants of the PURE-SA-NWP study (2005-2015) it is evident that rural and urban groups differ significantly in terms of dietary changes occurring during the nutrition transition. Most prominent and surprising are the changes observed in the rural groups, which experienced dietary changes so rapid that they not only caught up with the urban groups but surpassed them by 2015. This is in contrast with the urban areas, where energy and nutrient intake seemed to have equalised by 2015.

Disparities, as observed in our results, pose unique health challenges to the various groups. The research group intends to further investigate the impact of the observed changes in urban and rural groups and the association with obesity and other non-communicable disease factors.

5.7 Acknowledgements

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5.9 Conflict of interest

None.

5.10 Author contributions

S. Lee: Responsible for the conception and design, analysis and interpretation of the data and writing of the manuscript.

C. Taljaard-Krugell: Made contributions to the conception and design, analysis and interpretation of the data and critically revised the manuscript for important intellectual content.

C. Botha-Ravyse and T. van Zyl: Made contributions to the design, interpretation of the data and critically revised the manuscript for important intellectual content.

E. Wentzel-Viljoen, contributed to the interpretation of the data and critically revised the manuscript for important intellectual content.

5.11 Ethical standards disclosure

This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and the Ethics Committee approved all procedures involving research study participants of the North-West University and approved the PURE-SA-NWP study, as well as the current study (04M10 and NWU-0016-10-A1). Permission for the study was also obtained from the North-West Provincial Department of Health, local government authorities, and tribal chiefs in the rural areas. Written informed consent was obtained from all volunteers. The participants were transported to and from the study sites in both the urban and rural areas. They received lunch on site after blood sampling was completed. The study is registered at ClinicalTrials.gov (ID NCT03225586, <https://clinicaltrials.gov>).

5.12 References

1. Weerahewa J, Korale-Gedara P Wijetunga CS (2020) Nutrition Transition in Sri Lanka: A Meta-Analysis of the Nutrition Profile. In *Agricultural Research for Sustainable Food Systems in Sri Lanka*, pp. 13-61: Springer.
2. Siddiqui MZ, Donato R Jumrani J (2019) Looking past the Indian calorie debate: What is happening to nutrition transition in India. *The Journal of Development Studies* 55, 2440-2459.
3. Auma CI, Pradeilles R, Blake MK *et al.* (2019) What can dietary patterns tell us about the nutrition transition and environmental sustainability of diets in Uganda? *Nutrients* 11, 342.
4. Wentzel-Viljoen E, Lee S, Laubscher R *et al.* (2018) Accelerated nutrition transition in the North West Province of South Africa: results from the Prospective Urban and Rural Epidemiology (PURE-NWP-SA) cohort study, 2005 to 2010. *Public Health Nutr* 21, 2630-2641.
5. Hawkes C, Harris J Gillespie S (2017) Changing diets: Urbanization and the nutrition transition. *IFPRI book chapters*, 34-41.
6. Rodrigues LPF, Carvalho RC, Maciel A *et al.* (2016) Food insecurity in urban and rural areas in Central Brazil: Transition from locally produced foods to processed items. *Ecology of food and nutrition* 55, 365-377.
7. Abrahams Z, Mchiza Z Steyn NP (2011) Diet and mortality rates in Sub-Saharan Africa: stages in the nutrition transition. *BMC Public Health* 11, 801.
8. MacIntyre U, Kruger H, Venter C *et al.* (2002) Dietary intakes of an African population in different stages of transition in the North West Province, South Africa: the THUSA study. *Nutr Res* 22, 239-256.
9. Popkin BM (2002) An overview on the nutrition transition and its health implications: the Bellagio meeting. *Public Health Nutr* 5, 93-103.

10. Peters R, Amugsi DA, Mberu B *et al.* (2019) Nutrition transition, overweight and obesity among rural-to-urban migrant women in Kenya. *Public Health Nutr* 22, 3200-3210.
11. Vorster HH, Kruger A Margetts BM (2011) The nutrition transition in Africa: can it be steered into a more positive direction? *Nutrients* 3, 429-441.
12. Popkin BM & Gordon-Larsen P (2004) The nutrition transition: worldwide obesity dynamics and their determinants. *International journal of obesity* 28, S2-S9.
13. Popkin BM (2002) The shift in stages of the nutrition transition in the developing world differs from past experiences! *Public Health Nutr* 5, 205-214.
14. Vorster H (2002) The emergence of cardiovascular disease during urbanisation of Africans. *Public Health Nutr* 5, 239-243.
15. Pisa P, Behanan R, Vorster H *et al.* (2012) Social drift of cardiovascular disease risk factors in Africans from the North West Province of South Africa: the PURE study. *Cardiovasc J Afr* 23, 371.
16. Ayeni O, Joffe M, Cubasch H *et al.* (2019) Prevalence of comorbidities in women with and without breast cancer in Soweto, South Africa: Results from the SABC study. *SAMJ: South African Medical Journal* 109, 264-271.
17. Jacobs I, Taljaard-Krugell C, Ricci C *et al.* (2019) Dietary intake and breast cancer risk in black South African women: the South African Breast Cancer study. *Br J Nutr* 121, 591-600.
18. Chikowore T, Pisa PT, van Zyl T *et al.* (2017) Nutrient Patterns Associated with Fasting Glucose and Glycated Haemoglobin Levels in a Black South African Population. *Nutrients* 9, 9.
19. Pisa PT, Pedro TM, Kahn K *et al.* (2015) Nutrient Patterns and Their Association with Socio-Demographic, Lifestyle Factors and Obesity Risk in Rural South African Adolescents. *Nutrients* 7, 3464-3482.
20. Steyn N, Jaffer N, Nel J *et al.* (2016) Dietary Intake of the Urban Black Population of Cape Town: The Cardiovascular Risk in Black South Africans (CRIBSA) Study. *Nutrients* 8, 285.
21. WHO (2018) Fact sheet: Noncommunicable diseases. <https://www.who.int/en/news-room/fact-sheets/detail/noncommunicable-diseases> (accessed 27 October 2019 2019)
22. Statistics-SA (2020) Mortality and causes of death in South Africa, 2017: Findings from death notification <http://www.statssa.gov.za/publications/P03093/P030932016.pdf> (accessed 3 October 2020)
23. Vorster HH, Venter CS, Wissing MP *et al.* (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 Nutr* 8, 480-490.
24. Schutte AE, Schutte R, Huisman HW *et al.* (2012) Are behavioural risk factors to be blamed for the conversion from optimal blood pressure to hypertensive status in Black South Africans? A 5-year prospective study. *Int J Epidemiol* 41, 1114-1123.

25. Vorster HH, Kruger A, Wentzel-Viljoen E *et al.* (2014) Added sugar intake in South Africa: findings from the Adult Prospective Urban and Rural Epidemiology cohort study. *The American journal of clinical nutrition* 99, 1479-1486.
26. Sotunde O, Kruger H, Wright HH *et al.* (2015) Lean mass appears to be more strongly associated with bone health than fat mass in urban black South African women. *J Nutr Health Aging* 19, 628-636.
27. Corsi DJ, Subramanian S, Chow CK *et al.* (2013) Prospective Urban Rural Epidemiology (PURE) study: baseline characteristics of the household sample and comparative analyses with national data in 17 countries. *Am Heart J* 166, 636-646. e634.
28. MacIntyre U, Venter C Vorster H (2001) A culture-sensitive quantitative food frequency questionnaire used in an African population: 2. Relative validation by 7-day weighed records and biomarkers. *Public Health Nutr* 4, 63-71.
29. MacIntyre U, Venter C Vorster H (2001) A culture-sensitive quantitative food frequency questionnaire used in an African population: 1. Development and reproducibility. *Public Health Nutr* 4, 53-62.
30. Wentzel-Viljoen E, Laubscher R Kruger A (2011) Using different approaches to assess the reproducibility of a culturally sensitive quantified food frequency questionnaire. *South Afr J Clin Nutr* 24, 143-148.
31. Venter C, MacIntyre U Vorster H (2000) The development and testing of a food portion photograph book for use in an African population. *J Hum Nutr Diet* 13, 205-218.
32. Langenhoven M, Conradie P, Wolmarans P *et al.* (1991) MRC food quantities manual. *Cape Town: South African Medical Research Council* 2.
33. Wolmarans P, Danster N, Dalton A *et al.* (2010) Condensed food composition tables for South Africa. *Cape Town: Medical Research Council*, 1-126.
34. Statistics IS (2019) IBM SPSS Statistics software: SPSS Statistics V26.
35. Italian Ministry of Agriculture Food and Forestry (2019) *Linee guida per una sana alimentazione italiana. Revisione 2018*. Rome: CREA.
36. Scientific Advisory Committee on Nutrition (2019) Saturated fats and health: SACN report [Public Health England, editor]. London: TSO.
37. Wolfram G, Bechthold A, Boeing H *et al.* (2015) Evidence-based guideline of the German Nutrition Society: fat intake and prevention of selected nutrition-related diseases. *Ann Nutr Metab* 67, 141-204.
38. Dietary Guidelines Advisory Committee (2015) *Dietary guidelines for Americans 2015-2020*: Government Printing Office.
39. Smuts CM & Wolmarans P (2013) The importance of the quality or type of fat in the diet: a food-based dietary guideline for South Africa. *South Afr J Clin Nutr* 26, S87-S99.
40. Meyers LD, Hellwig JP Otten JJ (2006) *Dietary reference intakes: the essential guide to nutrient requirements*: National Academies Press.
41. WHO (2015) *Guideline: sugars intake for adults and children*: World Health Organization.

42. Yu AYL, Lopez-Olmedo N Popkin B (2017) Dietary trends in energy, carbohydrate, protein, fat, sodium and potassium intake by urbanicity index in Chinese adolescents from 1991 to 2011. *The FASEB Journal* 31, 639.653-639.653.
43. Streeter JL (2017) Socioeconomic Factors Affecting Food Consumption and Nutrition in China: Empirical Evidence During the 1989–2009 Period. *The Chinese Economy* 50, 168-192.
44. Mchiza ZJ, Steyn NP, Hill J *et al.* (2015) A review of dietary surveys in the adult South African population from 2000 to 2015. *Nutrients* 7, 8227-8250.
45. D’Haese M & Van Huylenbroeck G (2005) The rise of supermarkets and changing expenditure patterns of poor rural households case study in the Transkei area, South Africa. *Food Policy* 30, 97-113.
46. Demmler KM, Ecker O Qaim M (2017) *Supermarket shopping and nutritional outcomes: a panel data analysis for urban Kenya.*
47. Zong G, Li Y, Wanders AJ *et al.* (2016) Intake of individual saturated fatty acids and risk of coronary heart disease in US men and women: two prospective longitudinal cohort studies. *BMJ* 355, i5796.
48. Mao L, Zhang Y, Wang W *et al.* (2020) Plant-sourced and animal-sourced monounsaturated fatty acid intakes in relation to mortality: a prospective nationwide cohort study. *Eur J Nutr* 59, 1989-1998.
49. Trevisan M, Krogh V, Grioni S *et al.* (2020) Mediterranean diet and all-cause mortality: A cohort of Italian men. *Nutrition, Metabolism and Cardiovascular Diseases* 30, 1673-1678.
50. Shisana O, Labadarios D, Rehle T *et al.* (2015) The South African National Health and Nutrition Examination Survey, 2012: SANHANES-1: the health and nutritional status of the nation.
51. Sánchez-Pimienta TG, Batis C, Lutter CK *et al.* (2016) Sugar-sweetened beverages are the main sources of added sugar intake in the Mexican population. *The Journal of nutrition* 146, 1888S-1896S.
52. Braverman-Bronstein A, Camacho-García-Formentí D, Zepeda-Tello R *et al.* (2020) Mortality attributable to sugar sweetened beverages consumption in Mexico: an update. *International Journal of Obesity* 44, 1341-1349.
53. Wentzel-Viljoen E, Laubscher R Vorster HH (2018) Changes in food intake from 2005 to 2010 by a cohort of black rural and urban African men and women in the North West Province of South Africa: the PURE-NWP-SA study, vol. 21, pp. 2941-2958.
54. Okop KJ, Ndayi K, Tsolekile L *et al.* (2019) Low intake of commonly available fruits and vegetables in socio-economically disadvantaged communities of South Africa: influence of affordability and sugary drinks intake. *BMC Public Health* 19.
55. Okop KJ, Lambert EV, Alaba O *et al.* (2019) Sugar-sweetened beverage intake and relative weight gain among South African adults living in resource-poor communities: longitudinal data from the STOP-SA study. *International Journal of Obesity* 43, 603-614.

56. Popkin BM & Du S (2003) Dynamics of the nutrition transition toward the animal foods sector in China and its implications: a worried perspective. *The Journal of nutrition* 133, 3898S-3906S.
57. Popkin BM (2003) The Nutrition Transition in the Developing World. *Development Policy Review* 21, 581-597.
58. Popkin BM (2002) Part II. What is unique about the experience in lower-and middle-income less-industrialised countries compared with the very-highincome industrialised countries?: The shift in stages of the nutrition transition in the developing world differs from past experiences! *Public Health Nutr* 5, 205-214.
59. Tripathy JP, Thakur JS, Jeet G *et al.* (2016) Urban rural differences in diet, physical activity and obesity in India: are we witnessing the great Indian equalisation? Results from a cross-sectional STEPS survey. *BMC Public Health* 16.

CHAPTER 6 CONCLUSIONS AND RECOMMENDATIONS

6.1 Introduction

The primary aim of this thesis was to examine the changes in dietary intake from 2005 to 2015, as well as the associations between the changes in dietary fat intake and the changes in cardiovascular disease (CVD) risk from 2005 to 2010, of rural and urban South African groups amid a nutrition transition.

This chapter presents a summary of our main findings according to the hypotheses stated in Chapter 1. Furthermore, it draws conclusions, elaborates on the limitations of the present study and makes recommendations for further research.

6.2 Main findings according to the hypotheses provided in Chapter 1

HYPOTHESIS 1

Statistically significant changes in the nutrient intakes of rural and urban black South Africans residing in the North West province, between 2005 and 2010, are observed.

The amount of energy consumed by rural groups in 2010 (men 9.7 MJ/d; women 9.1 MJ/d), was comparable to the amount of energy consumed by urban groups in 2005 (men 9.9 MJ/d; women 9.0 MJ/d). The median energy intake increased substantially from 2005 to 2010 in all groups investigated: rural men, rural women, urban men, and urban women. These increases in median energy values were 2.3, 3.4, 2.6 and 2.3 MJ/d for rural and urban men and rural and urban women, respectively. The changes in macronutrient distribution from 2005 to 2010 indicate increased percentages of energy from animal protein, saturated fat, added sugar and MUFA, while the percentage contributions of plant protein and total carbohydrate decreased in all groups. The percentage of energy from total fat increased only in the rural groups, while the percentage of energy from total protein increased only in rural men. A decreased percentage of energy from alcohol was noted in all groups except for urban men. Furthermore, the dietary intakes of the rural men and women in 2010 did not meet the recommended values for most minerals and vitamins.

Therefore Hypothesis 1 is accepted

HYPOTHESIS 2

The changes in the cardiovascular risk factors of rural and urban populations of PURE-NWP-SA are associated with the changes in their dietary fat intake.

Previously, researchers reported that changes in cardiovascular risk factors, especially in rural groups, were indicative of a population in transition (Bourne *et al.*, 2002; Pisa *et al.*, 2012). In our study the changes in dietary fat intake measured as total fat, saturated fat, poly-unsaturated fat (PUFA) and mono-unsaturated fat (MUFA) were not strongly associated with the observed changes in the cardiovascular risk factors (measured as low-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, total cholesterol, BMI (body mass index), waist circumference, systolic blood pressure, diastolic blood pressure and fasting blood glucose) of rural and urban men and women. This was notwithstanding significant observed increases in energy intake and the intake of all types of dietary fats from 2005 to 2010 in all groups. An increase in the change in waist circumference of rural men was associated with a decrease in the change in PUFA intake ($\beta=-0.280$; $p=0.002$) and an increase in the change in MUFA intake ($\beta=0.200$; $p=0.027$). Rural women, urban men, and urban women lacked any associations between the change in the different CVD risk factors and any of the changes in fat intakes. Instead, the variance was explained rather by various confounding factors (BMI, employment status, age, level of education, tobacco use, change in alcohol consumption, aspirin use, cholesterol medication use, diabetic medication use and physical activity) while BMI surfaced as a key determinant of various CVD risk factors.

Hypothesis 2 is therefore accepted for rural men.

HYPOTHESIS 3

Hypothesis: Statistically significant trends in the nutrient intakes of rural and urban black South-Africans residing in the North West province between 2005 and 2015 are observed.

The trends in energy and nutrient intakes, as well as macronutrient distribution, were significantly different between rural and urban groups. In the rural groups, the observed trends showed a continuous upward trend in total energy intake (respective increases of 5.7 MJ and 5.29 MJ for men and women), while the trends for the energy and nutrient intakes of the urban groups were not significant. When bearing in mind the mean differences in energy and nutrient intake per year (2005, 2010, 2015), it becomes evident that the lack of significant trends in the urban groups was caused by decreases in energy and nutrient intakes from 2010 to 2015. This meant that the rural groups not only “caught up” with their urban counterparts in terms of energy intake but surpassed them by 2015. This higher intake of total energy subsequently led to higher intakes in all macro-

and micronutrients (except for the trend of alcohol intake that decreased for rural women and remained unchanged for rural men). In terms of dietary recommendations, the total fat proportion of the rural groups as well as urban men remains worryingly above the global recommendation of less than 30% (Nishida *et al.*, 2004; Vorster *et al.*, 2013), while urban women were just below the recommendation at 28%. Alarmingly high added sugar intake continues in urban and rural populations with the added sugar intake for all groups being above 10% in 2015, and therefore also above the WHO recommendation (WHO, 2015b) of less than 5%.

Hypothesis 3 is thus accepted.

6.3 Conclusions

Chapter 1 identified two distinct problems, the first being the progression of the nutrition transition. Research evidence on the progress of the nutrition transition in South Africa since the early 2000s was limited and there was an urgent need for up-to-date research. This thesis added to the data in presenting findings for 10 years from 2005 to 2015. Article 1 (Chapter 3) and article 3 (Chapter 5) investigated the dietary intake changes from 2005 to 2015.

Dietary changes from 2005 to 2010 confirmed that the nutrition transition in the North West province of SA was taking place at a rapid pace in rural as well as urban groups. The observed nutrient intakes were high in animal-based proteins, energy, and fat and lower in fibre. Despite the increase in nutrient intakes, low micronutrient intakes prevailed, irrespective of fortification of staple foods. The intake of plant protein and starchy food suffered as a result, which may in turn increase the NCD risk of these groups in the long run.

With the 2015 data available, and analysed in Chapter 5, more insight has been obtained on the direction of the nutrition transition in the urban and rural groups under investigation. The observed changes in the trends from 2005 to 2015 have confirmed that the nutrition transition is still occurring at a rapid pace among the rural participants of PURE-SA-NWP, while among the urban participants it was flattening down to some extent. If the rapid increase in energy and nutrient intake of the rural group were to continue it would have a detrimental effect on the health and well-being of this group by further increasing its risk of developing NCD (Hawkes *et al.*, 2017; Peters *et al.*, 2019; Weerahewa *et al.*, 2020). This could place an even heavier burden on South Africa's quadruple burden of disease (Pillay-van Wyk *et al.*, 2016)

The second problem identified was the possible relationship between observed changes in dietary fat intake and the changes in cardiovascular disease risk. Our findings indicate that an increase in BMI rather than changes in dietary fat intake may be associated with an unfavourable change in CVD risk. Although the changes in fat intake of all groups largely lacked significant associations

with the changes in cardiovascular disease risk, our findings did show that a decrease in PUFA intake over the years may increase CVD risk in that it is associated with a higher WC. In terms of the decline in the waist circumference of rural men, PUFA also emerged as the fat substitute preferable to MUFA. This is probably due to the fact that it is possible that animal products were the main contributor to MUFA intake (as evidenced by the sharp increase in animal protein from 2005 to 2010), accompanied by increased saturated fat intakes. It comes as no surprise that PUFA emerged as a risk protector as the replacement of saturated fat with PUFA and MUFA from plant sources is highly recommended by the South African Food Based Dietary Guidelines (SAFBDG) (Smuts & Wolmarans, 2013). Our results seem to follow the latest thinking (Liu *et al.*, 2018; Mirmiran *et al.*, 2018; Ricci *et al.*, 2018; Schwab *et al.*, 2014; Wang *et al.*, 2016) that the amount of fat consumed is less of a driver of CVD than the type or quality of fat. This is also supported by the SAFBDG for fat intake (Smuts & Wolmarans, 2013).

Change in BMI was also identified as an important role player in the change of several CVD risk factors. Another salient observation was that the observed changes in the cardiovascular risk factors were indicative of a group in transition.

Figure 6-1 is a conclusive framework of the findings of this study and highlights the contributions and their importance in the field of nutrition.

Firstly, this framework gives an indication of the existing evidence regarding the nutrition transition in rural and urban groups in South Africa prior to this study. It then summarises the findings of the research presented in this thesis, indicating how the findings of each of the articles link back to the nutrition transition and where they place rural and urban groups in the various stages of the nutrition transition. We see that, whereas previously the rural and urban groups were shown to be in different stages of the nutrition transition (rural in stage 1, and urban in stages 3 and 4), the findings of this study place both rural and urban groups in stage 4, the degenerative disease phase. This is also supported by the emergence of non-communicable diseases in all sectors of the South African population (NDoH *et al.*, 2017). The framework concludes by indicating that this study will aid in CVD prevention while also aiding in the achievement of the third sustainable development goal of the United Nations.

FINDINGS OF THE PRESENT STUDY

PREVIOUSLY REPORTED DATA ON THE NUTRITION TRANSITION

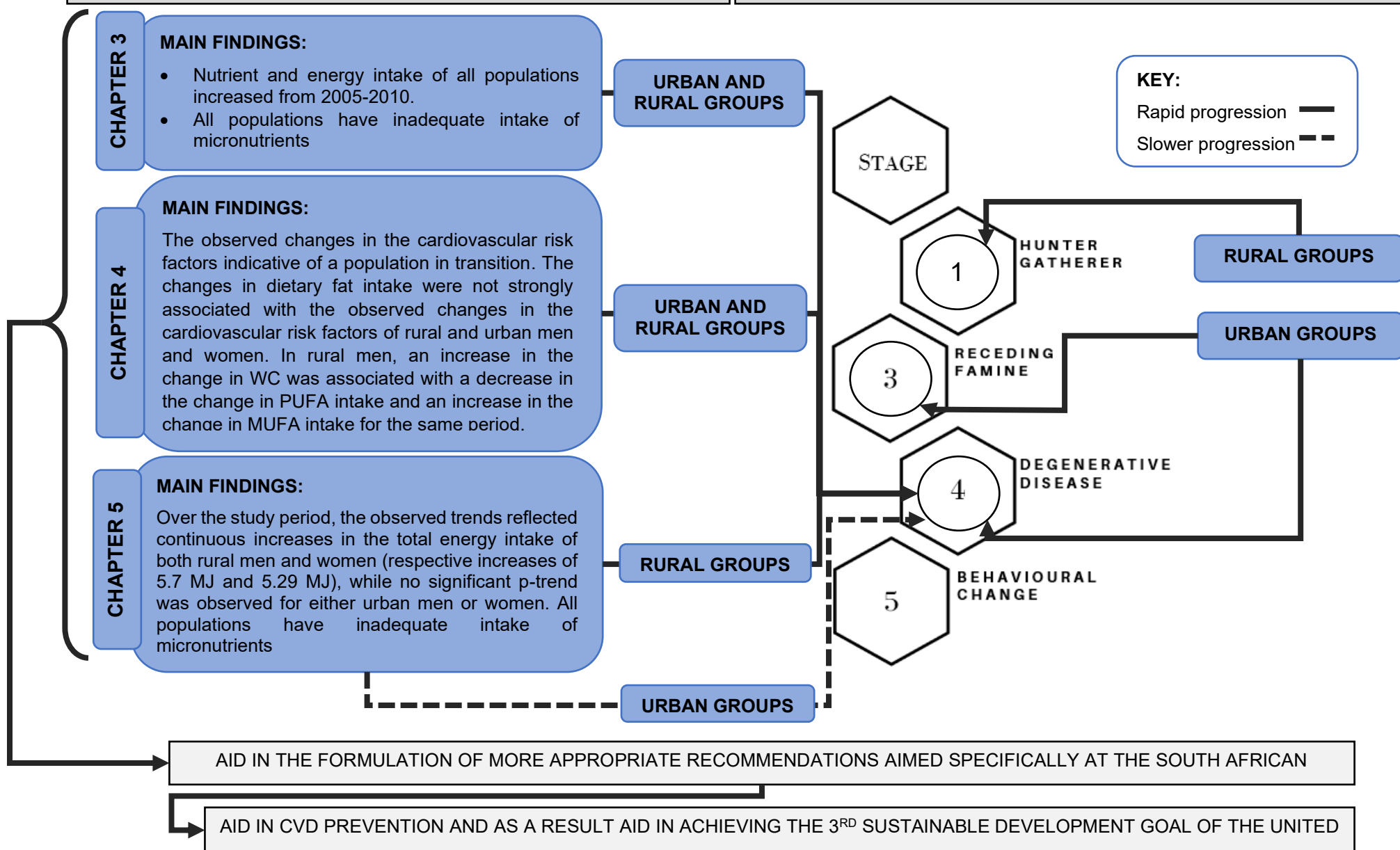


Figure 6-1: Concluding framework of the findings of the study

6.4 Limitations of this study

The current study was conducted in one of the nine provinces of South Africa and is therefore not representative of the South African population. In addition, the use of quantified food frequency questionnaires (QFFQs) as a dietary data collection method has been criticised owing to reservations regarding their level of accuracy (Freedman *et al.*, 2006; Schatzkin *et al.*, 2003). Previously, the QFFQ used in the The Transition and Health During Urbanisation of South Africans (THUSA) study was found to under-report energy and fat intakes in comparison with 7-day records (MacIntyre *et al.*, 2001b). However, the QFFQ used in PURE-SA-NWP was validated for this particular group and showed good reproducibility (MacIntyre *et al.*, 2001a; MacIntyre *et al.*, 2001b). Additionally, the same team of fieldworkers was trained and re-trained by the same team of professionals who administered the questionnaires for both the baseline as well as the follow-up data collections.

The observational study design is another limitation. Since observational studies lack randomisation and are influenced by the circumstances of the participant (as opposed to being under the control of the researcher as in experimental studies), they are much more prone to bias and confounding (Hess & Abd-Elseyed, 2019). The collection of dietary data could result in information bias since it relies on the ability of the participants to recall their dietary intake. In a longitudinal study, such as PURE-SA-NWP, the chance of recall bias has the potential to increase exponentially because of the possibility that the ageing participants may find information recall increasingly difficult. To effectively limit possible recall bias, our fieldworkers were well trained and used various aids such as example foodstuffs, a picture book, household measuring cups, spoons and crockery to assist participants in quantifying their food consumption. In terms of confounding bias, accurate data were collected for a variety of possible confounding factors to minimise the risk of confounding.

Although the PURE-SA-NWP study did collect household income data, these data could not be used prospectively as the income brackets on the questionnaires differed between the various data collections and could thus not be accurately compared. Information regarding changes in household income could have been valuable in explaining some of our results.

6.5 Policy implications

The SAFBDG forms part of existing science-based policy by the Department of Health as an important step toward alleviating South Africa's NCD burden by educating South Africans in modifying their dietary behaviour towards the consumption of more prudent diets (Vorster *et al.*, 2013). It was first developed and adopted as policy in 2001 (Vorster *et al.*, 2001) and revised in

2013 (Vorster *et al.*, 2013). The SAFBDG will achieve its goal only if it is successfully implemented in the communities (Vorster *et al.*, 2013). The changes in the energy and nutrient intakes of our study groups do not, however, reflect the dietary changes advocated by the SAFBDG and serve as proof that their implementation in communities is lacking. It is essential that the Department of Health and its stakeholders make implementing and advocating of the SAFBDG a priority. Targeted educational and promotional material has already been developed to aid in the successful implementation of the guidelines (Vorster *et al.*, 2013) and this should be used to facilitate successful implementation of the guidelines in communities. Previous implementation failures should be identified, and new or improved programmes should be developed and implemented in communities to promote the SAFBDG and empower communities to adopt the guidelines in their lifestyles. The 2020 Global Nutrition Report also highlights the importance of the implementation, monitoring and evaluation of evidence-based nutritional policies to provide healthy, sustainable and affordable food systems (Micha *et al.*, 2020).

From own observations while moving around the communities, we noted that many rural households in our study were still growing their own gardens and thus had better access to affordable fruits and vegetables, while this was less common in the urban communities. We further observed that the plots in our urban communities were often smaller than the rural plots, not allowing much space for household agricultural activities. This could in part explain the dissimilarities in terms of micronutrient intake as well as nutrient density between rural and urban groups. We therefore propose the implementation of community gardens as a strategy to implement the SAFBDG in communities. Government could assist communities by making plots of land available and aiding communities by providing means to irrigate and maintain these gardens. Community members must be encouraged to take part in the community gardening effort and those participating would be rewarded by having access to affordable fresh produce. In an unpublished study on large-scale food gardens, participating community members came up with the idea that coupons be issued to those contributing to allow them to collect food from the facility once a week (personal communication with the researcher, Dr Chrisna Ravyse). Excess produce can be sold to provide funds to maintain the gardens in the long run. In high-density urban areas in Sao Paulo, this method proved to be effective in improving the food quality of communities (Pedro *et al.*, 2020). Projects like these can also create jobs by employing community members as managers or permanent gardeners of these gardens. Community members are further empowered by acquiring new skills (Pedro *et al.*, 2020). Other studies have also reported on the effectiveness and nutritional benefit of well managed community gardens (Gerber *et al.*, 2017; Kou *et al.*, 2019). It is especially important that the community members receive education and training that is fit for purpose on the types of vegetables to plant so that they will be able to consume a variety of foods (as advocated by the SAFBDG). Furthermore, they

should be educated on the importance of fruit and vegetable consumption as well as on the amount that should ideally be consumed. These gardens can be expanded to include chicken coops to produce eggs, or cows to produce milk. The expansion of the community gardens should be accompanied by education to link the products being produced with their respective SAFBDG. We believe that the successful implementation of community gardens on a large scale will aid in implementing and advocating the SAFBDG to empower South Africans to successfully change their dietary habits, reducing the adverse effects of the nutrition transition and lowering their risk of developing NCDs.

In dealing with a double-burden of malnutrition, governments traditionally implement separate policies to address over- and under-nutrition (Hawkes *et al.*, 2020). Over- and under-nutrition, often co-exist in communities and may therefore have shared drivers (WHO, 2017b). Consequently, it is important that governments rather implement integrated policies (double-duty actions) to simultaneously address both, as one-sided approaches to address over-nutrition may hinder or harm progress made in terms of addressing under-nutrition, and vice versa (Hawkes *et al.*, 2020; WHO, 2017b). The SAFBDGs were developed with the purpose of being such a double-duty action that addresses both under- and over-nutrition in equal measure (Vorster *et al.*, 2013). It is important, however, that advocacy and implementation of the SAFBDG in communities be approached holistically so that the double-duty properties of the guidelines are maintained.

The food environment, especially in the rural communities in our research group, underwent dramatic changes 2005 to 2015, allowing them access to a large variety of supermarket foods they did not have access to before. We support the policies the South African government has been implementing with the purpose of providing or promoting healthier food environments, in line with several of the SAFBDGs: limiting trans-fats to a minimum of 2% of the oil content (South Africa, 2010), reducing the salt content of processed foods (South Africa, 2012) and tax on sugar-sweetened beverages (National Treasury, 2018). Another such policy was the implementation of the use of the South African Nutrient Profiling model (SANPM) (developed and validated for South Africans (Lee, 2013; Wentzel-Viljoen *et al.*, 2012; Wicks, 2012)) to regulate nutrient and/or health claims on a scientific basis. According to this policy, foodstuffs can carry a claim only if classified as healthy enough to do so by SANPM. An important consideration in this regard is that food labels are not user-friendly for the illiterate (Solomons *et al.*, 2019) and also meaningless to those without a basic understanding of food labelling and claims (which would be lacking in a community to whom local supermarkets, let alone food labelling, are very new). We therefore want to emphasise that nutrition education on community level should include knowledge about food labelling as well as health and nutrient claims to cultivate an understanding of these and encourage people to use them as tools to adhere to the SAFBDGs and make healthier choices.

Beyond food labelling, the Department of Health has published draft regulations to use the SANPM to identify unhealthy foods that may not be marketed to children (South Africa, 2014). Wicks *et al.* (2016) showed that, although the SANPM was developed for food labelling, it was sensitive enough to be used as a tool to identify high-fat, -sugar and -salt foods that may not be advertised to children. We propose that these regulations be amended to also control the foods that can be advertised to adults. This is supported by a review of 18 studies by Vukmirovic (2015) which reported a significant positive association between food advertising and the food choices of adults. Such a policy change will allow the opportunity to align food advertising and marketing with the SAFBDG to facilitate healthier food choices and steer the nutrition transition and its consequent development of NCDs into a more positive direction.

6.6 Suggestions for future research

The important contribution of descriptive studies such as ours should not be underestimated. These studies form the basis for 1) understanding and tracking dietary intake changes in populations and 2) further analysis to be conducted. Our results pose three questions. First, what are the key factors contributing to the continuous increase in energy and nutrient intake observed in the rural group? Second, what are the key factors contributing to the changes observed in the urban group? Third, what is the impact of the changes occurring in questions one and two on obesity and other non-communicable disease risk factors. Disparities, as observed in our results, pose unique health challenges to the various groups. This research team intends to further pursue these questions.

We further recommend that similar prospective research be conducted in other parts of South Africa to broaden the knowledge base on the nutrition transition in South Africa and for it to be more representative of the entire population. Conducting similar prospective research that is representative of the country's diverse population groups could provide much needed evidence indicating whether other rural groups are following similar trends. Further research on dietary patterns and the specific foods that were consumed to bring about the energy and nutrient changes we observed from 2005 to 2015 will provide much needed insight into the drivers of the nutrition transition in these groups. Wentzel-Viljoen and colleagues (2018) have already conducted analysis on the types of food consumed from 2005 to 2010. Such research would assist in the development of dietary recommendations and interventions to slow the progression of the nutrition transition, to prevent and limit the development of NCD caused by deteriorating dietary intake and to steer the dietary intake of South Africans towards an intake indicative of the behavioural change stage of the nutrition transition.

As discussed earlier and in agreement with earlier research (Liu *et al.*, 2018; Mao *et al.*, 2020), our findings pointed toward the possibility that an increase in MUFA in the diet may be detrimental in terms of CVD risk if it comes mainly from animal sources. More research is warranted to determine the association between the source of MUFA intake (plant vs animal) and CVD risk.

6.7 Concluding statement

The research presented in this PhD thesis gives insight into the dietary changes from 2005 to 2015 and the associations of the change in fat intake with the change in the CVD risk from 2005 to 2010 of rural and urban South African groups. The nutrition transition was still progressing at a rapid pace for rural as well as urban participants during the first five years of the PURE-SA-NWP study. During the last five years, the nutrition transition continued to progress rapidly within the rural groups, while slowing down in the urban group. We have also shown that the CVD risk of all groups worsened from 2005 to 2010 and that there were associations between the changes in MUFA and PUFA intake and the waist circumference of rural men.

Our results highlight the importance of timeous monitoring of the various drivers of the nutrition transition and the urgent need to steer the nutrition transition in a more positive direction to prevent the progressive worsening of our NCD burden.

BIBLIOGRAPHY FOR CHAPTERS 1, 2 AND 6

- Abrahams, Z., Mchiza, Z. & Steyn, N.P. 2011. Diet and mortality rates in Sub-Saharan Africa: stages in the nutrition transition. *BMC Public Health*, 11(1):801. <https://doi.org/10.1186/1471-2458-11-801>
- Adams, F. & Brock, A.J. 1955. Hippocratic writings: Encyclopaedia Britannica.
- Akinboboye, O., Idris, O., Akinboboye, O. & Akinkugbe, O. 2003. Trends in coronary artery disease and associated risk factors in sub-Saharan Africans. *Journal of Human Hypertension*, 17(6):381-387. <https://doi.org/10.1038/sj.jhh.1001562>
- Alwan, A. 2011. Global status report on noncommunicable diseases 2010: World Health Organization.
- Ambrose, J.A. & Singh, M. 2015. Pathophysiology of coronary artery disease leading to acute coronary syndromes. *F1000prime reports*, 7. <https://doi.org/10.12703/P7-08>
- Appel, L.J., Moore, T.J., Obarzanek, E., Vollmer, W.M., Svetkey, L.P., Sacks, F.M., . . . Windhauser, M.M. 1997. A clinical trial of the effects of dietary patterns on blood pressure. *New England journal of medicine*, 336(16):1117-1124. <https://doi.org/10.1056/NEJM199704173361601>
- Asbaghi, O., Choghakhori, R., Ashtary-Larky, D. & Abbasnezhad, A. 2020. Effects of the Mediterranean diet on cardiovascular risk factors in non-alcoholic fatty liver disease patients: A systematic review and meta-analysis. *Clinical Nutrition ESPEN*. <https://doi.org/10.1016/j.clnesp.2020.03.003>
- Asghar, A. & Sheikh, N. 2017. Role of immune cells in obesity induced low grade inflammation and insulin resistance. *Cellular Immunology*, 315:18-26. <https://doi.org/10.1016/j.cellimm.2017.03.001>
- Ayeni, O., Joffe, M., Cubasch, H., Rinaldi, S., Taljaard, C., Vorster, E., . . . Norris, S. 2019. Prevalence of comorbidities in women with and without breast cancer in Soweto, South Africa: Results from the SABC study. *South African Medical Journal*, 109(4):264-271. <https://doi.org/10.7196/SAMJ.2019.v109i4.13465>
- Becerra-Tomás, N., Blanco Mejía, S., Viguiouk, E., Khan, T., Kendall, C.W., Kahleova, H., . . . Salas-Salvadó, J. 2020. Mediterranean diet, cardiovascular disease and mortality in diabetes: A systematic review and meta-analysis of prospective cohort studies and randomized clinical trials. *Critical reviews in food science and nutrition*, 60(7):1207-1227. <https://doi.org/10.1080/10408398.2019.1565281>
- Belury, M.A., Bowman, E., Gabriel, J., Snyder, B., Kulkarni, M., Palettas, M., . . . Sieg, S.F. 2017. Prospective analysis of lipid composition changes with antiretroviral therapy and immune activation in persons living with HIV. *Pathogens & immunity*, 2(3):376. <https://doi.org/10.20411/pai.v2i3.218>
- Bender, N., Portmann, M., Heg, Z., Hofmann, K., Zwahlen, M. & Egger, M. 2014. Fish or n3-PUFA intake and body composition: a systematic review and meta-analysis. *Obesity Reviews*, 15(8):657-665. <https://doi.org/10.1111/obr.12189>

- Benziger, C.P., Roth, G.A. & Moran, A.E. 2016. The global burden of disease study and the preventable burden of NCD. *Global heart*, 11(4):393-397. <https://doi.org/10.1016/j.gheart.2016.10.024>
- Bertram, M.Y., Steyn, K., Wentzel-Viljoen, E., Tollman, S. & Hofman, K.J. 2012. Reducing the sodium content of high-salt foods: effect on cardiovascular disease in South Africa. *South African Medical Journal*, 102(9):743-745. <https://doi.org/10.7196/SAMJ.5832>
- Bhupathiraju, S.N. & Tucker, K.L. 2011. Coronary heart disease prevention: nutrients, foods, and dietary patterns. *Clinica Chimica Acta*, 412(17):1493-1514. <https://doi.org/10.1016/j.cca.2011.04.038>
- Bishwajit, G., Yaya, S. & Seydou, I. 2017. Diabetes mellitus and high blood pressure in relation to BMI among adult non-pregnant women in Bangladesh. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, 11:S217-S221. <https://doi.org/10.1016/j.dsx.2016.12.034>
- Bloom, D.E., Cafiero, E., Jané-Llopis, E., Abrahams-Gessel, S., Bloom, L.R., Fathima, S., . . . Mowafi, M. 2012. The global economic burden of noncommunicable diseases, PGDA Working papers 8712, Program on the Global Demography of Aging. Geneva: World Economic Forum. https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=&ved=2ahUKEwjO3baEhdfAhVtQUEAHUrKB5MQFjAAegQIAxAC&url=http%3A%2F%2Fwww3.weforum.org%2Fdocs%2FWEF_Harvard_HE_GlobalEconomicBurdenNonCommunicableDiseases_2011.pdf&usq=AOvVaw1eoVrqy34bGutp_GI8owOb Date of access: 13 July 2020.
- Bourne, L.T., Lambert, E.V. & Steyn, K. 2002. Where does the black population of South Africa stand on the nutrition transition? *Public Health Nutrition*, 5(1a):157-162. <https://doi.org/10.1079/PHN2001288>
- Bradshaw, D., Groenewald, P., Laubscher, R., Nannan, N., Nojilana, B., Norman, R., . . . Timæus, I.M. 2003. Initial burden of disease estimates for South Africa, 2000. *South African Medical Journal*, 93(9):682-688.
- Brock, J. & Gordon, H. 1959. Ischaemic heart disease in African populations. *Postgraduate Medical Journal*, 35(402):223. <https://doi.org/10.1136/pgmj.35.402.223>
- Calza, L., Manfredi, R. & Chiodo, F. 2004. Dyslipidaemia associated with antiretroviral therapy in HIV-infected patients. *Journal of Antimicrobial Chemotherapy*, 53(1):10-14. <https://doi.org/10.1093/jac/dkh013>
- Charlton, K. 2007. The development of a dietary intervention to modify dietary cation content and the evaluation of its effects on blood pressure in hypertensive black South Africans. *Cape Town: University of Cape Town*.
- Charlton, K.E., Macgregor, E., Vorster, N.H., Levitt, N.S. & Steyn, K. 2007. Partial replacement of NaCl can be achieved with potassium, magnesium and calcium salts in brown bread. *International journal of food sciences and nutrition*, 58(7):508-521. <https://doi.org/10.1080/09637480701331148>
- Chen, G.-C., Neelakantan, N., Martín-Calvo, N., Koh, W.-P., Yuan, J.-M., Bonaccio, M., . . . Van Dam, R.M. 2019. Adherence to the Mediterranean diet and risk of stroke and stroke subtypes. *European Journal of Epidemiology*, 34(4):337-349. <https://doi.org/10.1007/s10654-019-00504-7>
- Chen, M., Li, Y., Sun, Q., Pan, A., Manson, J.E., Rexrode, K.M., . . . Hu, F.B. 2016. Dairy fat and risk of cardiovascular disease in 3 cohorts of US adults. *The American Journal of Clinical Nutrition*, 104(5):1209-1217. <https://doi.org/10.3945/ajcn.116.134460>

- Coates, R.J. & Monteilh, C.P. 1997. Assessments of food-frequency questionnaires in minority populations. *The American journal of clinical nutrition*, 65(4):1108S-1115S. <https://doi.org/10.1093/ajcn/65.4.1108S>
- Copstead, L.E. & Banasik, J.L. 2013. Pathophysiology. St. Louis, Mo.: Elsevier.
- Corsi, D.J., Subramanian, S., Chow, C.K., McKee, M., Chifamba, J., Dagenais, G., . . . Kruger, A. 2013. Prospective Urban Rural Epidemiology (PURE) study: baseline characteristics of the household sample and comparative analyses with national data in 17 countries. *American Heart Journal*, 166(4):636-646. e634. <https://doi.org/10.1016/j.ahj.2013.04.019>
- Dalal, S., Beunza, J.J., Volmink, J., Adebamowo, C., Bajunirwe, F., Njelekela, M., . . . Adami, H.-O. 2011. Non-communicable diseases in sub-Saharan Africa: what we know now. *International Journal of Epidemiology*, 40(4):885-901. <https://doi.org/10.1093/ije/dyr050>
- Danaei, G., Finucane, M.M., Lu, Y., Singh, G.M., Cowan, M.J., Paciorek, C.J., . . . Stevens, G.A. 2011. National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2·7 million participants. *The Lancet*, 378(9785):31-40. [https://doi.org/10.1016/S0140-6736\(11\)60679-X](https://doi.org/10.1016/S0140-6736(11)60679-X)
- De Luca, C. & Olefsky, J.M. 2008. Inflammation and insulin resistance. *FEBS Letters*, 582(1):97-105. <https://doi.org/10.1016/j.febslet.2007.11.057>
- Dos Santos Silva, I. 1999. Cancer epidemiology: principles and methods. Lyon: IARC.
- Dua, S., Bhuker, M., Sharma, P., Dhall, M. & Kapoor, S. 2014. Body mass index relates to blood pressure among adults. *North American Journal of Medical Sciences*, 6(2):89-95. <https://doi.org/10.4103/1947-2714.127751>
- Feigin, V.L., Lawes, C.M., Bennett, D.A., Barker-Collo, S.L. & Parag, V. 2009. Worldwide stroke incidence and early case fatality reported in 56 population-based studies: a systematic review. *The Lancet Neurology*, 8(4):355-369. [https://doi.org/10.1016/S1474-4422\(09\)70025-0](https://doi.org/10.1016/S1474-4422(09)70025-0)
- Filippou, C.D., Tsioufis, C.P., Thomopoulos, C.G., Mihos, C.C., Dimitriadis, K.S., Sotiropoulou, L.I., . . . Tousoulis, D.M. 2020. Dietary Approaches to Stop Hypertension (DASH) Diet and Blood Pressure Reduction in Adults with and without Hypertension: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *European Heart Journal*, 41(S2), ehaa946.2765. <https://doi.org/10.1093/ehjci/ehaa946.2765>
- Freedman, L.S., Potischman, N., Kipnis, V., Midthune, D., Schatzkin, A., Thompson, F.E., . . . Carroll, R. 2006. A comparison of two dietary instruments for evaluating the fat–breast cancer relationship. *International Journal of Epidemiology*, 35(4):1011-1021. <https://doi.org/10.1093/ije/dyl085>
- Gadgil, M.D., Anderson, C.A.M., Kandula, N.R. & Kanaya, A.M. 2014. Dietary Patterns in Asian Indians in the United States: An Analysis of the Metabolic Syndrome and Atherosclerosis in South Asians Living in America Study. *Journal of the Academy of Nutrition and Dietetics*, 114(2):238-243. <https://doi.org/10.1016/j.jand.2013.09.021>
- Gearon, E., Tanamas, S.K., Stevenson, C., Loh, V.H.Y. & Peeters, A. 2018. Changes in waist circumference independent of weight: Implications for population level monitoring of obesity. *Preventive Medicine*, 111:378-383. <https://doi.org/10.1016/j.ypmed.2017.11.030>

- Gerber, M.M., Callahan, J.L., Moyer, D.N., Connally, M.L., Holtz, P.M. & Janis, B.M. 2017. Nepali Bhutanese refugees reap support through community gardening. *International Perspectives in Psychology: Research, Practice, Consultation*, 6(1):17. <https://doi.org/10.1037/ipp0000061>
- Grosso, G., Marventano, S., Yang, J., Micek, A., Pajak, A., Scalfi, L., . . . Kales, S.N. 2017. A comprehensive meta-analysis on evidence of Mediterranean diet and cardiovascular disease: are individual components equal? *Critical reviews in food science and nutrition*, 57(15):3218-3232. <https://doi.org/10.1080/10408398.2015.1107021>
- Grundty, S.M., Brewer Jr, H.B., Cleeman, J.I., Smith Jr, S.C. & Lenfant, C. 2004. Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation*, 109(3):433-438. <https://doi.org/10.1161/01.CIR.0000111245.75752.C6>
- Guasch-Ferré, M., Zong, G., Willett, W.C., Zock, P.L., Wanders, A.J., Hu, F.B. & Sun, Q. 2019. Associations of Monounsaturated Fatty Acids From Plant and Animal Sources With Total and Cause-Specific Mortality in Two US Prospective Cohort Studies. *Circulation Research*, 124(8):1266-1275. <https://doi.org/10.1161/CIRCRESAHA.118.313996>
- Hall, J.E. & Guyton, A.C. 2006. Guyton & Hall physiology review. Philadelphia: Elsevier Saunders.
- Hankin, J.H. & Wilkens, L.R. 1994. Development and validation of dietary assessment methods for culturally diverse populations. *The American journal of clinical nutrition*, 59(1):198S-200S. <https://doi.org/10.1093/ajcn/59.1.198S>
- Hariharan, A., Shanmugam, M. & Priya, L. 2019. A cross-sectional study on newly detected hypertensive individuals and its correlation to BMI. *IAIM Journal*, 6(11): 47-51
- Hawkes, C., Harris, J. & Gillespie, S. 2017. Changing diets: Urbanization and the nutrition transition. *IFPRI book chapters*:34-41.
- Hawkes, C., Ruel, M.T., Salm, L., Sinclair, B. & Branca, F. 2020. Double-duty actions: seizing programme and policy opportunities to address malnutrition in all its forms. *The Lancet*, 395(10218):142-155. [https://doi.org/10.1016/S0140-6736\(19\)32506-1](https://doi.org/10.1016/S0140-6736(19)32506-1)
- Heianza, Y., Zhou, T., Sun, D., Hu, F.B., Manson, J.E. & Qi, L. 2020. Genetic susceptibility, plant-based dietary patterns, and risk of cardiovascular disease. *The American Journal of Clinical Nutrition*, 112(1):220-228. <https://doi.org/10.1093/ajcn/nqaa107>
- Hess, A.S. & Abd-Elseyed, A. 2019. Observational Studies: Uses and Limitations: Springer International Publishing. p. 123-125). https://doi.org/10.1007/978-3-319-99124-5_31
- Ho, F.K., Gray, S.R., Welsh, P., Petermann-Rocha, F., Foster, H., Waddell, H., . . . Celis-Morales, C. 2020. Associations of fat and carbohydrate intake with cardiovascular disease and mortality: prospective cohort study of UK Biobank participants. *BMJ*:m688. <https://doi.org/10.1136/bmj.m688>
- Hooper, L., Martin, N., Abdelhamid, A. & Davey Smith, G. 2015. Reduction in saturated fat intake for cardiovascular disease. *Cochrane Database of Systematic Reviews*, (6). <https://doi.org/10.1002/14651858.CD011737>

- Hooper, L., Summerbell, C., Thompson, R., Sills, D., Roberts, F., Moore, H. & Davey Smith, G. 2012. Reduced or modified dietary fat for preventing cardiovascular disease Cochrane Database. of *Systematic Reviews*, 16(5). <https://doi.org/10.1002/14651858.CD002137.pub3>
- Hulsegge, G., Looman, M., Smit, H.A., Daviglus, M.L., van der Schouw, Y.T. & Verschuren, W.M. 2016. Lifestyle changes in young adulthood and middle age and risk of cardiovascular disease and all-cause mortality: The Doetinchem Cohort Study. *Journal of the American Heart Association*, 5(1):e002432. <https://doi.org/10.1161/JAHA.115.002432>
- Hwang, L.-C., Bai, C.-H., Sun, C.-A. & Chen, C.-J. 2012. Prevalence of metabolically healthy obesity and its impacts on incidences of hypertension, diabetes and the metabolic syndrome in Taiwan. *Asia Pacific Journal of Clinical Nutrition*, 21(2):227.
- Imamura, T., Doi, Y., Arima, H., Yonemoto, K., Hata, J., Kubo, M., . . . Kiyohara, Y. 2009. LDL Cholesterol and the Development of Stroke Subtypes and Coronary Heart Disease in a General Japanese Population The Hisayama Study. *Stroke*, 40(2):382-388. <https://doi.org/10.1161/STROKEAHA.108.529537>
- Institute for Health Metrics and Evaluation (IHME). 2018. Findings from the global burden of disease study 2017. *Seattle, WA IHME* 2018.
- Jagannathan, R., Patel, S.A., Ali, M.K. & Narayan, K.V. 2019. Global updates on cardiovascular disease mortality trends and attribution of traditional risk factors. *Current Diabetes Reports*, 19(7):44. <https://doi.org/10.1007/s11892-019-1161-2>
- Jakobsen, M.U., O'Reilly, E.J., Heitmann, B.L., Pereira, M.A., Bälter, K., Fraser, G.E., . . . Liu, S. 2009. Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies. *The American journal of clinical nutrition*, 89(5):1425-1432. <https://doi.org/10.3945/ajcn.2008.27124>
- Jiao, J., Liu, G., Shin, H.J., Hu, F.B., Rimm, E.B., Rexrode, K.M., . . . Sun, Q. 2019. Dietary fats and mortality among patients with type 2 diabetes: analysis in two population based cohort studies. *BMJ*:l4009. <https://doi.org/10.1136/bmj.l4009>
- Jinnouchi, H., Kolodgie, F.D., Romero, M., Virmani, R. & Finn, A.V. 2020. Pathophysiology of Coronary Artery Disease. *Vessel Based Imaging Techniques*. Springer. p. 211-227). https://doi.org/10.1007/978-3-030-25249-6_11
- Kabakambira, J.D., Baker Jr, R.L., Briker, S.M., Courville, A.B., Mabundo, L.S., Dubose, C.W., . . . Sumner, A.E. 2018. Do current guidelines for waist circumference apply to black Africans? Prediction of insulin resistance by waist circumference among Africans living in America. *BMJ Global Health*, 3(5):e001057. <https://doi.org/10.1136/bmjgh-2018-001057>
- Kakarmath, S.S., Zack, R.M., Leyna, G.H., Fahimi, S., Liu, E., Fawzi, W.W., . . . Danaei, G. 2017. Dietary determinants of serum total cholesterol among middle-aged and older adults: a population-based cross-sectional study in Dar es Salaam, Tanzania. *BMJ Open*, 7(5):e015028. <https://doi.org/10.1136/bmjopen-2016-015028>
- Kalani, Z., Rafiei, M. & Salimi, T. 2015. Comparison of obesity indexes BMI, WHR and WC in association with Hypertension: results from a Blood Pressure Status Survey in Iran. *Journal of Cardiovascular Disease Research*, 6(2). <https://doi.org/10.5530/jcdr.2015.2.5>
- Khan, M.A.B., Hashim, M.J., King, J.K., Govender, R.D., Mustafa, H. & Al Kaabi, J. 2019. Epidemiology of Type 2 Diabetes – Global Burden of Disease and Forecasted Trends. *Journal of Epidemiology and Global Health*, 10(1):107. <https://doi.org/10.2991/jegh.k.191028.001>

- Khan, S.S., Ning, H., Wilkins, J.T., Allen, N., Carnethon, M., Berry, J.D., . . . Lloyd-Jones, D.M. 2018. Association of Body Mass Index With Lifetime Risk of Cardiovascular Disease and Compression of Morbidity. *JAMA Cardiology*, 3(4):280.
<https://doi.org/10.1001/jamacardio.2018.0022>
- Kim, H., Caulfield, L.E., Garcia-Larsen, V., Steffen, L.M., Coresh, J. & Rebholz, C.M. 2019. Plant-Based diets are associated with a lower risk of incident cardiovascular disease, cardiovascular disease mortality, and All-Cause mortality in a general population of Middle-Aged adults. *Journal of the American Heart Association*, 8(16):e012865.
<https://doi.org/10.1161/JAHA.119.012865>
- Klug, E.Q., Raal, F., Marais, A., Taskinen, M., Dalby, A., Schamroth, C., . . . Catsicas, R. 2013. South African Dyslipidaemia Guideline Consensus Statement: A joint statement from the South African Heart Association (SA Heart) and the Lipid and Atherosclerosis Society of Southern Africa (LASSA). *South African family practice*, 55(1):9-18.
<https://doi.org/10.1080/20786204.2013.10874296>
- Klug, E., Raal, F., Marais, A., Smuts, C., Schamroth, C., Jankelow, D., Blom, D. & Webb, D. 2018. South African dyslipidaemia guideline consensus statement: 2018 update a joint statement from the South African Heart Association (SA Heart) and the Lipid and Atherosclerosis Society of Southern Africa (LASSA). *South African medical journal*, 108(11b):973-1000. <https://doi.org/10.7196/SAMJ.2018.v108i11.13383>
- Klug, E. & Raal, F. 2020. New cholesterol targets for patients at high or very high cardiovascular risk and the indications for PCSK9 inhibitors. *SAMJ: South African Medical Journal*, 110(11):1059-1059. <http://dx.doi.org/10.7196%2FSAMJ.2020.v110i11.15191>
- Koegelenberg, A.S., Kruger, A., Towers, G.W. & Schutte, A.E. 2013. Lack of association of glycated haemoglobin with blood pressure and subclinical atherosclerosis in black South Africans: a five-year prospective study. *Journal of Endocrinology, Metabolism and Diabetes of South Africa*, 18(3):148-153. <https://doi.org/10.1080/22201009.2013.10844553>
- Kou, H., Zhang, S. & Liu, Y. 2019. Community-Engaged Research for the Promotion of Healthy Urban Environments: A Case Study of Community Garden Initiative in Shanghai, China. *International journal of environmental research and public health*, 16(21):4145.
<https://doi.org/10.3390/ijerph16214145>
- Koutkia, P. & Grinspoon, S. 2004. HIV-associated lipodystrophy: pathogenesis, prognosis, treatment, and controversies. *Annual reports in medical chemistry*, 55:303-317.
<https://doi.org/10.1146/annurev.med.55.091902.104412>
- Krause, T., Lovibond, K., Caulfield, M., McCormack, T., Williams, B. & Group, G.D. 2011. Management of hypertension: summary of NICE guidance. *BMJ*, 343:d4891.
<https://doi.org/10.1136/bmj.d4891>
- Kruger, H.S., Botha-Ravyse, C., Havemann-Nel, L., Doubell, M. & Van Rooyen, J.M. 2017. Agreement between specific measures of adiposity and associations with high blood pressure in black South African women. *American Journal of Human Biology*, 29(6):e23042.
<https://doi.org/10.1002/ajhb.23042>
- Kruger, H.S., Steyn, N.P., Swart, E.C., Maunder, E.M., Nel, J.H., Moeng, L. & Labadarios, D. 2012a. Overweight among children decreased, but obesity prevalence remained high among women in South Africa, 1999–2005. *Public Health Nutrition*, 15(4):594-599.
<https://doi.org/10.1017/S136898001100262X>

- Kruger, H.S., Steyn, N.P., Swart, E.C., Maunder, E.M., Nel, J.H., Moeng, L. & Labadarios, D. 2012b. Overweight among children decreased, but obesity prevalence remained high among women in South Africa, 1999–2005. *Public Health Nutrition*, 15(04):594-599. <https://doi.org/10.1017/S136898001100262X>
- Langenhoven, M.L., Kruger, M., Gows, E., Faber, M. 1991. MRC food composition tables: South African Medical Research Council, Research Institute for Nutritional Diseases.
- Last, J.M., Abramson, J.H. & Freidman, G.D. 2001. A dictionary of epidemiology. Vol. 141: Oxford University Press.
- Lee, H., Lee, I.S. & Choue, R. 2013. Obesity, inflammation and diet. *Pediatric gastroenterology, hepatology & nutrition*, 16(3):143-152. <https://doi.org/10.5223/pghn.2013.16.3.143>
- Lee, J.S., Pinnamaneni, S.K., Eo, S.J., Cho, I.H., Pyo, J.H., Kim, C.K., . . . Watt, M.J. 2006. Saturated, but not n-6 polyunsaturated, fatty acids induce insulin resistance: role of intramuscular accumulation of lipid metabolites. *Journal of Applied Physiology*, 100(5):1467-1474. <https://doi.org/10.1152/japplphysiol.01438.2005>
- Lee, S.J. 2013. Using existing dietary data for evaluating the construct validity of a nutrient profiling model. Potchefstroom: North-West University (MSc Thesis).
- Li, Y., Hruby, A., Bernstein, A.M., Ley, S.H., Wang, D.D., Chiuve, S.E., . . . Willett, W.C. 2015. Saturated Fats Compared With Unsaturated Fats and Sources of Carbohydrates in Relation to Risk of Coronary Heart Disease: A Prospective Cohort Study. *Journal of the American College of Cardiology*, 66(14):1538-1548. <https://doi.org/10.1016/j.jacc.2015.07.055>
- Lichtenstein, A.H., Appel, L.J., Brands, M., Carnethon, M., Daniels, S., Franch, H.A., . . . Howard, B. 2006. Diet and lifestyle recommendations revision 2006 A scientific statement from the American Heart Association nutrition committee. *Circulation*, 114(1):82-96. <https://doi.org/10.1161/CIRCULATIONAHA.106.176158>
- Liu, X., Li, Y., Tobias, D.K., Wang, D.D., Manson, J.E., Willett, W.C. & Hu, F.B. 2018. Changes in types of dietary fats influence long-term weight change in US women and men. *The Journal of nutrition*, 148(11):1821-1829. <https://doi.org/10.1093/jn/nxy183>
- Liyanage, T., Ninomiya, T., Wang, A., Neal, B., Jun, M., Wong, M.G., . . . Perkovic, V. 2016. Effects of the Mediterranean diet on cardiovascular outcomes—a systematic review and meta-analysis. *PloS One*, 11(8):e0159252. <https://doi.org/10.1371/journal.pone.0159252>
- Lozano, R., Naghavi, M., Foreman, K., Lim, S., Shibuya, K., Aboyans, V., . . . Ahn, S.Y. 2013. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *The Lancet*, 380(9859):2095-2128. [https://doi.org/10.1016/S0140-6736\(12\)61728-0](https://doi.org/10.1016/S0140-6736(12)61728-0)
- Mach, F., Baigent, C., Catapano, A.L., Koskinas, K.C., Casula, M., Badimon, L., . . . Ference, B.A. 2020. 2019 ESC/EAS guidelines for the management of dyslipidaemias: Lipid modification to reduce cardiovascular risk. *European Heart Journal*, 41:111-188. <https://doi.org/10.1093/eurheartj/ehz455>
- MacIntyre, U., Kruger, H., Venter, C. & Vorster, H. 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. [https://doi.org/10.1016/S0271-5317\(01\)00392-X](https://doi.org/10.1016/S0271-5317(01)00392-X)

- MacIntyre, U., Venter, C. & Vorster, H. 2001a. A culture-sensitive quantitative food frequency questionnaire used in an African population: 1. Development and reproducibility. *Public Health Nutrition*, 4(1):53-62.
- MacIntyre, U., Venter, C. & Vorster, H. 2001b. A culture-sensitive quantitative food frequency questionnaire used in an African population: 2. Relative validation by 7-day weighed records and biomarkers. *Public Health Nutrition*, 4(1):63-71. <https://doi.org/10.1079/PHN200040>
- MacIntyre, U.E., Venter, C.S., Kruger, A. & Serfontein, M. 2012. Measuring micronutrient intakes at different levels of sugar consumption in a population in transition: the Transition and Health during Urbanisation in South Africa (THUSA) study. *South African Journal of Clinical Nutrition*, 25(3):122-130. <https://doi.org/10.1079/PHN200041>
- Mao, L., Zhang, Y., Wang, W., Zhuang, P., Wu, F. & Jiao, J. 2020. Plant-sourced and animal-sourced monounsaturated fatty acid intakes in relation to mortality: a prospective nationwide cohort study. *European Journal of Nutrition*, 59(5):1989-1998. <https://doi.org/10.1007/s00394-019-02048-8>
- Maphumulo, W.T. & Bhengu, B.R. 2019. Challenges of quality improvement in the healthcare of South Africa post-apartheid: A critical review. *Curationis*, 42(1):1-9. <https://doi.org/10.4102/curationis.v42i1.1901>
- Mathers, C., Fat, D.M. & Boerma, J.T. 2008. The global burden of disease: 2004 update: World Health Organization. Geneva: World Health Organisation.
- Mattiuzzi, C., Sanchis-Gomar, F. & Lippi, G. 2020. Worldwide burden of LDL cholesterol: Implications in cardiovascular disease. *Nutrition, Metabolism and Cardiovascular Diseases*, 30(2):241-244. <https://doi.org/10.1016/j.numecd.2019.09.008>
- Mayosi, B.M., Flisher, A.J., Lalloo, U.G., Sitas, F., Tollman, S.M. & Bradshaw, D. 2009. The burden of non-communicable diseases in South Africa. *The Lancet*, 374(9693):934-947. [https://doi.org/10.1016/S0140-6736\(09\)61087-4](https://doi.org/10.1016/S0140-6736(09)61087-4)
- McFarlane, S.I., Banerji, M. & Sowers, J.R. 2001. Insulin Resistance and Cardiovascular Disease. *The journal of clinical endocrinology & metabolism*, 86(2):713-718. <https://doi.org/10.1210/jc.86.2.713>
- Mchiza, Z.J., Steyn, N.P., Hill, J., Kruger, A., Schönfeldt, H., Nel, J. & Wentzel-Viljoen, E. 2015. A review of dietary surveys in the adult South African population from 2000 to 2015. *Nutrients*, 7(9):8227-8250. <https://doi.org/10.3390/nu7095389>
- Micha, R., Mannar, V., Afshin, A., Allemandi, L., Baker, P., Battersby, J., . . . Di Cesare, M. 2020. 2020 Global nutrition report: action on equity to end malnutrition. *Technical Report. Behrman, Nina, ed. Development Initiatives*, Bristol, UK. ISBN 978-1-9164452-7-7.
- Michels, K.B. 2001. A renaissance for measurement error. *International Journal of Epidemiology*, 30(3):421-422. <https://doi.org/10.1093/ije/30.3.421>
- Michels, K.B. 2003. Nutritional epidemiology—past, present, future. *International Journal of Epidemiology*, 32(4):486-488. <https://doi.org/10.1093/ije/dyg216>
- Mirmiran, P., Esfandyari, S., Moghadam, S.K., Bahadoran, Z. & Azizi, F. 2018. Fatty acid quality and quantity of diet and risk of type 2 diabetes in adults: Tehran Lipid and Glucose Study. *Journal of Diabetes and Its Complications*, 32(7):655-659. <https://doi.org/10.1016/j.jdiacomp.2018.05.003>

- Mishra, V., Arnold, F., Semenov, G., Hong, R. & Mukuria, A. 2006. Epidemiology of obesity and hypertension and related risk factors in Uzbekistan. *European Journal of Clinical Nutrition*, 60(12):1355-1366. <https://doi.org/10.1038/sj.ejcn.1602465>
- Mohammadi, H., Ohm, J., Discacciati, A., Sundstrom, J., Hambraeus, K., Jernberg, T. & Svensson, P. 2020. Abdominal obesity and the risk of recurrent atherosclerotic cardiovascular disease after myocardial infarction. *European Journal of Preventive Cardiology*: 204748731989801. <https://doi.org/10.1177/2047487319898019>
- Murray, C.J., Vos, T., Lozano, R., Naghavi, M., Flaxman, A.D., Michaud, C., . . . Abdalla, S. 2012. Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *The Lancet*, 380(9859):2197-2223. [https://doi.org/10.1016/S0140-6736\(12\)61689-4](https://doi.org/10.1016/S0140-6736(12)61689-4)
- Naghavi, M., Abajobir, A.A., Abbafati, C., Abbas, K.M., Abd-Allah, F., Abera, S.F., . . . Murray, C.J.L. 2017. Global, regional, and national age-sex specific mortality for 264 causes of death, 1980–2016: a systematic analysis for the Global Burden of Disease Study 2016. *The Lancet*, 390(10100):1151-1210.
- National Treasury. 2018. Budget review. <https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=&cad=rja&uact=8&ved=2ahUKEwiSlqTjkdftAhVUQkEAHR-QCR8QFjAAegQIAxAC&url=http%3A%2F%2Fwww.treasury.gov.za%2Fdocuments%2Fnational%2520budget%2F2018%2Freview%2FFullBR.pdf&usq=AOvVaw0cSeG30QCmMi6aF37KcK7b>
Date of access: 13 December 2020.
- NDoH, Statistics-SA, SA-MRC & ICF, Statistics-Sa. 2017. South African Demographic and Health Survey 2016: Key Indicators. Pretoria, South Africa. <https://dhsprogram.com/pubs/pdf/FR337/FR337.pdf> Date of access: 1 November 2020.
- NDoH. 1998. South African Demographic and Health Survey (SADHS); 1998. Pretoria: National Department of Health, Medical Research Council of South Africa, MEASURE, DHS. <https://dhsprogram.com/pubs/pdf/FR131/FR131.pdf> Date of access: 14 November 2020.
- Nishida, C., Uauy, R., Kumanyika, S. & Shetty, P. 2004. The joint WHO/FAO expert consultation on diet, nutrition and the prevention of chronic diseases: process, product and policy implications. *Public Health Nutrition*, 7(1a):245-250. <https://doi.org/10.1079/PHN2003592>
- Norman, R., Bradshaw, D., Steyn, K., Gaziano, T. & Collaboration, S.A.C.R.A. 2007. Estimating the burden of disease attributable to high cholesterol in South Africa in 2000. *South African Medical Journal*, 97(8):708-715.
- Nnyepi, M.S., Gwisai, N., Lekgoa, M. & Seru, T. 2015. Evidence of nutrition transition in Southern Africa. *Proceedings of the Nutrition Society*, 74(4):478-486. <https://doi.org/10.1017/S0029665115000051>
- Ocke, M.C., Bueno-de-Mesquita, H.B., Goddijn, H.E., Jansen, A., Pols, M.A., van Staveren, W.A. & Kromhout, D. 1997. The Dutch EPIC food frequency questionnaire. I. Description of the questionnaire, and relative validity and reproducibility for food groups. *International Journal of Epidemiology*, 26(suppl_1):S37. https://doi.org/10.1093/ije/26.suppl_1.s37
- Ogawa, S., Abe, T., Nako, K., Okamura, M., Senda, M., Sakamoto, T., . . . Group, D.S. 2013. Eicosapentaenoic acid improves glycemic control in elderly bedridden patients with type 2 diabetes. *The Tohoku journal of experimental medicine*, 231(1):63-74. <https://doi.org/10.1620/tjem.231.63>

- Olson, K.L., Neiberg, R.H., Espeland, M.A., Johnson, K.C., Knowler, W.C., Pi-Sunyer, X., . . . Wing, R.R. 2020. Waist Circumference Change During Intensive Lifestyle Intervention and Cardiovascular Morbidity and Mortality in the Look AHEAD Trial. *Obesity*, 28(10):1902-1911. <https://doi.org/10.1002/oby.22942>
- Owolabi, E.O., Ter Goon, D., Adeniyi, O.V. & Ajayi, A.I. 2018. Optimal waist circumference cut-off points for predicting metabolic syndrome among low-income black South African adults. *BMC Research Notes*, 11(1). <https://doi.org/10.1186/s13104-018-3136-9>
- Patel, S.A., Ali, M.K., Alam, D., Yan, L.L., Levitt, N.S., Bernabe-Ortiz, A., . . . Gutierrez, L. 2016. Obesity and its relation with diabetes and hypertension: a cross-sectional study across 4 geographical regions. *Global heart*, 11(1):71-79. e74. <https://doi.org/10.1016/j.gheart.2016.01.003>
- Pedro, A.A., Görner, A., Lindner, A. & Wende, W. 2020. " More than fruits and vegetables": Community garden experiences from the Global North to foster green development of informal areas in Sao Paulo, Brazil. *Research in Urbanism Series*, 6:219-242.
- Perk, J., De Backer, G., Gohlke, H., Graham, I., Reiner, Ž., Verschuren, M., . . . Cifkova, R. 2012. European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). *European Heart Journal*, 33(13):1635-1701. <https://doi.org/10.1093/eurheartj/ehs092>
- Peters, R., Amugsi, D.A., Mberu, B., Ensor, T., Hill, A.J., Newell, J.N. & Elsey, H. 2019. Nutrition transition, overweight and obesity among rural-to-urban migrant women in Kenya. *Public Health Nutrition*, 22(17):3200-3210. <https://doi.org/10.1017/S1368980019001204>
- Pickersgill, L., Litherland, G.J., Greenberg, A.S., Walker, M. & Yeaman, S.J. 2007. Key role for ceramides in mediating insulin resistance in human muscle cells. *Journal of Biological Chemistry*, 282(17):12583-12589. <https://doi.org/10.1074/jbc.M611157200>
- Pillay-van Wyk, V., Msemburi, W., Laubscher, R., Dorrington, R.E., Groenewald, P., Glass, T., . . . Prinsloo, M. 2016. Mortality trends and differentials in South Africa from 1997 to 2012: second National Burden of Disease Study. *The Lancet Global Health*, 4(9):e642-e653. [https://doi.org/10.1016/S2214-109X\(16\)30113-9](https://doi.org/10.1016/S2214-109X(16)30113-9)
- Pisa, P., Behanan, R., Vorster, H. & Kruger, A. 2012. Social drift of cardiovascular disease risk factors in Africans from the North West Province of South Africa: the PURE study. *Cardiovascular Journal of Africa*, 23(7):371. <https://doi.org/10.5830/CVJA-2012-018>
- Popkin, B.M. 2002a. An overview on the nutrition transition and its health implications: the Bellagio meeting. *Public Health Nutrition*, 5(1A):93-103. <https://doi.org/10.1079/PHN2001280>
- Popkin, B.M. 2002b. The shift in stages of the nutrition transition in the developing world differs from past experiences! *Public Health Nutrition*, 5(1A):205-214. <https://doi.org/10.1079/PHN2001295>
- Popkin, B.M. & Du, S. 2003. Dynamics of the nutrition transition toward the animal foods sector in China and its implications: a worried perspective. *The Journal of nutrition*, 133(11):3898S-3906S. <https://doi.org/10.1093/jn/133.11.3898S>
- Popkin, B.M. & Gordon-Larsen, P. 2004. The nutrition transition: worldwide obesity dynamics and their determinants. *International journal of obesity*, 28:S2-S9. <https://doi.org/10.1038/sj.ijo.0802804>

- Prinsloo, E.A., Joubert, G., Mohale, M., Nyindi, N., Matu, N., Ntechane, L. & Struwig, M.C. 2011. The prevalence and perception of obesity and its association with the lifestyle of women at the Mangaung University Community Partnership Project healthcare centre, Bloemfontein. *South African family practice*, 53(4):366-372. <https://doi.org/10.1080/20786204.2011.10874116>
- Rader, D.J. 2007. Effect of Insulin Resistance, Dyslipidemia, and Intra-abdominal Adiposity on the Development of Cardiovascular Disease and Diabetes Mellitus. *The American journal of medicine*, 120(3, Supplement 1):S12-S18. <https://doi.org/10.1016/j.amjmed.2007.01.003>
- Ricci, C., Baumgartner, J., Zec, M., Kruger, H.S. & Smuts, C.M. 2018. Type of dietary fat intakes in relation to all-cause and cause-specific mortality in US adults: An iso-energetic substitution analysis from the American National Health and Nutrition Examination Survey linked to the US mortality registry. *British Journal of Nutrition*, 119(4):456-463. <https://doi.org/10.1017/S0007114517003889>
- Rigby, N. 2006. Commentary: Counterpoint to Campos et al. *International Journal of Epidemiology*, 35(1):79-80. <https://doi.org/10.1093/ije/dyi261>
- Rocha, V.Z. & Libby, P. 2009. Obesity, inflammation, and atherosclerosis. *Nature Reviews Cardiology*, 6(6):399. <https://doi.org/10.1038/nrcardio.2009.55>
- Rodrigo, C.P., Aranceta, J., Salvador, G. & Varela-Moreiras, G. 2015. Food frequency questionnaires. *Nutricion Hospitalaria*, 31(3):49-56.
- Ronquest-Ross, L.-C., Vink, N. & Sigge, G.O. 2015. Food consumption changes in South Africa since 1994. *South African Journal of Science*, 111(9-10):01-12. <https://doi.org/10.17159/sajs.2015/20140354>
- Ronto, R., Wu, J.H. & Singh, G.M. 2018. The global nutrition transition: trends, disease burdens and policy interventions. *Public Health Nutrition*, 21(12):2267-2270. <https://doi.org/10.1017/S1368980018000423>
- Rosato, V., Temple, N.J., La Vecchia, C., Castellan, G., Tavani, A. & Guercio, V. 2019. Mediterranean diet and cardiovascular disease: a systematic review and meta-analysis of observational studies. *European Journal of Nutrition*, 58(1):173-191. <https://doi.org/10.1007/s00394-017-1582-0>
- Ross, R., Neeland, I.J., Yamashita, S., Shai, I., Seidell, J., Magni, P., . . . Després, J.-P. 2020. Waist circumference as a vital sign in clinical practice: a Consensus Statement from the IAS and ICCR Working Group on Visceral Obesity. *Nature Reviews Endocrinology*, 16(3):177-189. <https://doi.org/10.1038/s41574-019-0310-7>
- Roth, G.A., Abate, D., Abate, K.H., Abay, S.M., Abbafati, C., Abbasi, N., . . . Murray, C.J.L. 2018. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: a systematic analysis for the Global Burden of Disease Study 2017. *The Lancet*, 392(10159):1736-1788. [https://doi.org/10.1016/S0140-6736\(18\)32203-7](https://doi.org/10.1016/S0140-6736(18)32203-7)
- Sacks, F.M., Lichtenstein, A.H., Wu, J.H., Appel, L.J., Creager, M.A., Kris-Etherton, P.M., . . . Robinson, J.G. 2017. Dietary fats and cardiovascular disease: a presidential advisory from the American Heart Association. *Circulation*, 136(3):e1-e23. <https://doi.org/10.1161/CIR.0000000000000510>
- Salehi-Abargouei, A., Maghsoudi, Z., Shirani, F. & Azadbakht, L. 2013. Effects of Dietary Approaches to Stop Hypertension (DASH)-style diet on fatal or nonfatal cardiovascular

diseases—Incidence: A systematic review and meta-analysis on observational prospective studies. *Nutrition*, 29(4):611-618. <https://doi.org/10.1016/j.nut.2012.12.018>

Saltiel, A.R. & Olefsky, J.M. 2017. Inflammatory mechanisms linking obesity and metabolic disease. *The Journal of clinical investigation*, 127(1):1-4. <https://doi.org/10.1172/JCI92035>

Sarbolouki, S., Javanbakht, M.H., Derakhshanian, H., Hosseinzadeh, P., Zareei, M., Hashemi, S.B., . . . Djalali, M. 2013. Eicosapentaenoic acid improves insulin sensitivity and blood sugar in overweight type 2 diabetes mellitus patients: a double-blind randomised clinical trial. *Singapore Medical Journal*, 54(7):387-390. <https://doi.org/10.11622/smedj.2013139>

Saulle, R., Lia, L., De Giusti, M. & La Torre, G. 2019. A systematic overview of the scientific literature on the association between Mediterranean Diet and the Stroke prevention. *Clinica Terapeutica*, 170(5):e396-e408.

Schatzkin, A., Kipnis, V., Carroll, R.J., Midthune, D., Subar, A.F., Bingham, S., . . . Freedman, L.S. 2003. A comparison of a food frequency questionnaire with a 24-hour recall for use in an epidemiological cohort study: results from the biomarker-based Observing Protein and Energy Nutrition (OPEN) study. *International Journal of Epidemiology*, 32(6):1054-1062. <https://doi.org/10.1093/ije/dyg264>

Schenk, S., Saberi, M. & Olefsky, J.M. 2008. Insulin sensitivity: modulation by nutrients and inflammation. *The Journal of clinical investigation*, 118(9):2992-3002. <https://doi.org/10.1172/JCI34260>

Schutte, A.E., Botha, S., Fourie, C.M.T., Gafane-Mateman, L.F., Kruger, R., Lammertyn, L., . . . Huisman, H.W. 2017. Recent advances in understanding hypertension development in sub-Saharan Africa. *Journal of Human Hypertension*, 31(8):491-500. <https://doi.org/10.1038/jhh.2017.18>

Schwab, U., Lauritzen, L., Tholstrup, T., Haldorsson, T.I., Riserus, U., Uusitupa, M. & Becker, W. 2014. Effect of the amount and type of dietary fat on cardiometabolic risk factors and risk of developing type 2 diabetes, cardiovascular diseases, and cancer: a systematic review. *Food & Nutrition Research*, 58(1):25145. <https://doi.org/10.3402/fnr.v58.25145>

Schwingshackl, L., Chaimani, A., Schwedhelm, C., Toledo, E., Püsch, M., Hoffmann, G. & Boeing, H. 2019. Comparative effects of different dietary approaches on blood pressure in hypertensive and pre-hypertensive patients: A systematic review and network meta-analysis. *Critical Reviews in Food Science and Nutrition*, 59(16):2674-2687. <https://doi.org/10.1080/10408398.2018.1463967>

SDG. 2015. RES/70/1. Transforming our world: the 2030 agenda for sustainable development. *Seventieth United Nations General Assembly, New York*, 25. Available at: <https://www.refworld.org/docid/57b6e3e44.html> Date of access: 8 October 2020.

Seedat, Y., Rayner, B. & Veriava, Y. 2014. South African hypertension practice guideline 2014. *Cardiovascular Journal of Africa*, 25(6):288. <https://doi.org/10.5830/CVJA-2014-062>

Seftel, H. 1978. The rarity of coronary heart disease in South African blacks. *South African Medical Journal*, 54(3):99-105.

Shannon, O.M., Mendes, I., Köchl, C., Mazidi, M., Ashor, A.W., Rubele, S., . . . Siervo, M. 2020. Mediterranean diet increases endothelial function in adults: a systematic review and meta-analysis of randomized controlled trials. *The Journal of nutrition*, 150(5):1151-1159. <https://doi.org/10.1093/jn/nxaa002>

- Shim, J.S., Oh, K. & Kim, H.C. 2014. Dietary assessment methods in epidemiologic studies. *Epidemiol Health*, 36:e2014009. <https://doi.org/10.4178/epih/e2014009>
- Shisana, O., Labadarios, D., Rehle, T., Simbayi, L., Zuma, K., Dhansay, A., . . . Naidoo, P. 2014. The South African National Health and Nutrition Examination Survey, 2012: SANHANES-1: the health and nutritional status of the nation.
- Simelela, N. & Venter, W.D.F. 2014. A brief history of South Africa's response to AIDS. *SAMJ: South African Medical Journal*, 104(3):249-251. : <https://doi.org/10.7196/SAMJ.7700>
- Sliwa, K., Wilkinson, D., Hansen, C., Ntyintyane, L., Tibazarwa, K., Becker, A. & Stewart, S. 2008. Spectrum of heart disease and risk factors in a black urban population in South Africa (the Heart of Soweto Study): a cohort study. *The Lancet*, 371(9616):915-922. [https://doi.org/10.1016/S0140-6736\(08\)60417-1](https://doi.org/10.1016/S0140-6736(08)60417-1)
- Smuts, C.M. & Wolmarans, P. 2013. The importance of the quality or type of fat in the diet: a food-based dietary guideline for South Africa. *South African Journal of Clinical Nutrition*, 26(3):S87-S99.
- Solomons, N., Kruger, H.S. & Pouane, T.R. 2019. Addressing non-communicable diseases in the Western Cape, South Africa. *Journal of Public Health Research*, 8(2). <https://doi.org/10.4081/jphr.2019.1534>
- Soltani, S., Arablou, T., Jayedi, A. & Salehi-Abargouei, A. 2020. Adherence to the dietary approaches to stop hypertension (DASH) diet in relation to all-cause and cause-specific mortality: a systematic review and dose-response meta-analysis of prospective cohort studies. *Nutrition Journal*, 19:1-13. <https://doi.org/10.1186/s12937-020-00554-8>
- South Africa. 2010. Regulation 249 of the Foodstuffs, Cosmetics and Disinfectants Act (54/1972): Regulations: Trans fat in foodstuffs Pretoria. https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=&cad=rja&uact=8&ved=2ahUKEwjW1Z_TmNftAhUTV8AKHW52CW0QFjABegQIARAC&url=https%3A%2F%2Fwww.foodstuffs.co.za%2Fdraft-regulations-on-trans-fat-published-2%2F&usq=AOvVaw1zwgc22J4f-0wiiHNkokpR Date of access: 10 November 2020.
- South Africa. 2012. Regulation under the Foodstuffs, Cosmetics and Disinfectants Act 1972 (Act 54 of 1972), 'Regulations relating to the reduction of sodium in certain foodstuffs and related matters'. Pretoria. https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=&cad=rja&uact=8&ved=2ahUKEwiavbvmNftAhUHfMAKHv9Cv9QFjABegQIAhAC&url=https%3A%2F%2Fgain.fas.usda.gov%2FRecent%2520GAIN%2520Publications%2FRegulations%2520relating%2520to%2520the%2520reduction%2520of%2520sodium%2520in%2520certain%2520foodstuff_Pretoria_South%2520Africa%2520-%2520Republic%2520of_7-26-2016.pdf&usq=AOvVaw0CaBy5YCCHS-pFDN4D4Naa Date of access: 12 November 2020.
- South Africa. 2014. Regulation 429 Foodstuffs, Cosmetics and Disinfectants Act (54/1972): regulations relating to the labelling and advertising of foods: Amendment. Pretoria. https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=&cad=rja&uact=8&ved=2ahUKEwjQv8qOmdftAhXHSEEAHTp7CFkQFjACegQIARAC&url=https%3A%2F%2Fwww.green gazette.co.za%2Fnotices%2Ffoodstuffs-cosmetics-and-disinfectants-act-54-1972-regulations-relating-to-the-labelling-and-advertising-of-foods-amendment_20140529-GGR-37695-00429.pdf&usq=AOvVaw1zTe7uAID-bTM8qmnpuKGe Date of access: 13 November 2020.

Statistics-SA. 2020. Mortality and causes of death in South Africa, 2017: Findings from death notification <https://www.statssa.gov.za/publications/P03093/P030932017.pdf> Date of access: 3 October 2020.

Steyn, K., Sliwa, K., Hawken, S., Commerford, P., Onen, C., Damasceno, A., . . . Yusuf, S. 2005. Risk factors associated with myocardial infarction in Africa the INTERHEART Africa study. *Circulation*, 112(23):3554-3561. <https://doi.org/10.1161/CIRCULATIONAHA.105.563452>

Strazzullo, P., D'Elia, L., Cairella, G., Garbagnati, F., Cappuccio, F.P. & Scalfi, L. 2010. Excess body weight and incidence of stroke meta-analysis of prospective studies with 2 million participants. *Stroke*, 41(5):e418-e426. <https://doi.org/10.1161/STROKEAHA.109.576967>

Thompson, F.E. & Subar, A.F. 2017. Dietary assessment methodology. *Nutrition in the Prevention and Treatment of Disease*. Elsevier. p. 5-48). <https://doi.org/10.1016/B978-0-12-802928-2.00001-1>

Tran, N.T.T., Blizzard, C.L., Luong, K.N., Truong, N.L.V., Tran, B.Q., Otahal, P., . . . Callisaya, M. 2018. The importance of waist circumference and body mass index in cross-sectional relationships with risk of cardiovascular disease in Vietnam. *PloS One*, 13(5):e0198202. <https://doi.org/10.1371/journal.pone.0198202>

Tuomilehto, J., Lindström, J., Eriksson, J.G., Valle, T.T., Hämäläinen, H., Ilanne-Parikka, P., . . . Rastas, M. 2001. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *New England journal of medicine*, 344(18):1343-1350. <https://doi.org/10.1056/NEJM200105033441801>

Ubbink, J.B., Vermaak, W., Delport, R., van der Merwe, A., Becker, P. & Potgieter, H. 1995. Effective homocysteine metabolism may protect South African blacks against coronary heart disease. *The American journal of clinical nutrition*, 62(4):802-808. <https://doi.org/10.1093/ajcn/62.4.802>

Van Horn, L., McCoin, M., Kris-Etherton, P.M., Burke, F., Carson, J.A.S., Champagne, C.M., . . . Sikand, G. 2008. The evidence for dietary prevention and treatment of cardiovascular disease. *Journal of the American Dietetic Association*, 108(2):287-331. <https://doi.org/10.1016/j.jada.2007.10.050>

Van Schoor, J. 2010. South African cholesterol guidelines compared. *South African pharmaceutical journal*, 77(9):46-52. <https://doi.org/10.1080/20786204.2010.10873941>

Venter, C., MacIntyre, U. & Vorster, H. 2000. The development and testing of a food portion photograph book for use in an African population. *Journal of Human Nutrition and Dietetics*, 13(3):205-218. <https://doi.org/10.1046/j.1365-277x.2000.00228.x>

Vorster, H. 2002. The emergence of cardiovascular disease during urbanisation of Africans. *Public Health Nutrition*, 5(1a):239-243. <https://doi.org/10.1079/PHN2001299>

Vorster, H., Love, P. & Browne, C. 2001. Development of food-based dietary guidelines for South Africa: the process. *South African Journal of Clinical Nutrition*, 14(3).

Vorster, H.H., Badham, J. & Venter, C. 2013. An introduction to the revised food-based dietary guidelines for South Africa. *South African Journal of Clinical Nutrition*, 26(3):S5-S12.

Vorster, H.H., Kruger, A. & Margetts, B.M. 2011. The nutrition transition in Africa: can it be steered into a more positive direction? *Nutrients*, 3(4):429-441. <https://doi.org/10.3390/nu3040429>

- Vorster, H.H., Venter, C.S., Wissing, M.P. & Margetts, B.M. 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*, 8(05):480-490. <https://doi.org/10.1079/PHN2005784>
- Vos, A.G., Chersich, M.F., Klipstein-Grobusch, K., Zuithoff, P., Moorhouse, M.A., Lalla-Edward, S.T., . . . Barth, R.E. 2018. Lipid levels, insulin resistance and cardiovascular risk over 96 weeks of antiretroviral therapy: a randomised controlled trial comparing low-dose stavudine and tenofovir. *Retrovirology*, 15(1):77. <https://doi.org/10.1186/s12977-018-0460-z>
- Vukmirovic, M. 2015. The effects of food advertising on food-related behaviours and perceptions in adults: A review. *Food Research International*, 75:13-19. <https://doi.org/10.1016/j.foodres.2015.05.011>
- Wagner, K.-H. & Brath, H. 2012. A global view on the development of non communicable diseases. *Preventive Medicine*, 54, Supplement:S38-S41. <https://doi.org/10.1016/j.ypmed.2011.11.012>
- Wandai, M.E., Norris, S.A., Aagaard-Hansen, J. & Manda, S.O. 2020. Geographical influence on the distribution of the prevalence of hypertension in South Africa: a multilevel analysis. *Cardiovascular Journal of Africa*, 31(1):47-54. <https://doi.org/10.5830/CVJA-2019-047>
- Wang, D.D., Li, Y., Chiuve, S.E., Stampfer, M.J., Manson, J.E., Rimm, E.B., . . . Hu, F.B. 2016. Association of Specific Dietary Fats With Total and Cause-Specific Mortality Association of Dietary Fats and Total and Cause-Specific Mortality Association of Dietary Fats and Total and Cause-Specific Mortality. *JAMA Internal Medicine*, 176(8):1134-1145. <https://doi.org/10.1001/jamainternmed.2016.2417>
- Warlow, C. 1998. Epidemiology of stroke. *The Lancet*, 352:S1-S4. [https://doi.org/10.1016/S0140-6736\(98\)90086-1](https://doi.org/10.1016/S0140-6736(98)90086-1)
- Warsy, A., El-Hazmi, M. & Al-Hazmi, A. 2015. Prevalence of co-existing hypertension and obesity in Saudis. *Biomedical and Pharmacology Journal*, 4(2):269-274. <https://doi.org/10.13005/bpj/293>
- Weerahewa, J., Korale-Gedara, P. & Wijetunga, C.S. 2020. Nutrition Transition in Sri Lanka: A Meta-Analysis of the Nutrition Profile. *Agricultural Research for Sustainable Food Systems in Sri Lanka*. Springer. p. 13-61). https://doi.org/10.1007/978-981-15-2152-2_2
- Wentzel-Viljoen, E., Jerling, J. & Vorster, E. 2012. Report: Testing and Software Development of a Nutrient Profiling Model for South Africa. Report to the National Department of Health, Directorate: Food Control, contract no.
- Wentzel-Viljoen, E., Laubscher, R. & Kruger, A. 2011. Using different approaches to assess the reproducibility of a culturally sensitive quantified food frequency questionnaire. *South African Journal of Clinical Nutrition*, 24(3):143-148. <https://doi.org/10.1080/16070658.2011.11734366>
- Wentzel-Viljoen, E., Laubscher, R. & Vorster, H.H. 2018. Changes in food intake from 2005 to 2010 by a cohort of black rural and urban African men and women in the North West Province of South Africa: the PURE-NWP-SA study (Vol. 21. pp. 2941-2958). <https://doi.org/10.1017/S1368980018001878>

Wentzel-Viljoen, E., Steyn, K., Ketterer, E. & Charlton, K. 2013. "Use salt and foods high in salt sparingly": a food-based dietary guideline for South Africa. *South African Journal of Clinical Nutrition*, 26(3):S105-S113.

WHO. 1969. Mankind's greatest epidemic: heart disease: Resolution and Press Release, Division of Public Information World Health Organization, Geneva, Switzerland (February 1969).

WHO. 1974. The Prevention and Control of Major Cardiovascular Diseases: Report on a Conference Convened by the Regional Office for Europe of the World Health Organization, Brussels, 1973: Regional Office for Europe WHO.

WHO. 2002. Joint FAO expert consultation: Diet, nutrition and the prevention of chronic diseases. <http://health.euroafrica.org/books/dietnutritionwho.pdf> Date of access: 1 August 2019.

WHO. 2006. BMI classifications. <http://www.euro.who.int/en/health-topics/disease-prevention/nutrition/a-healthy-lifestyle/body-mass-index-bmi> Date of access: 26 February 2019.

WHO. 2009. Global health risks: mortality and burden of disease attributable to selected major risks. http://www.who.int/healthinfo/global_burden_disease/GlobalHealthRisks_report_full.pdf Date of access: 5 January 2019.

WHO. 2011a. Use of glycated haemoglobin (HbA1c) in diagnosis of diabetes mellitus: abbreviated report of a WHO consultation. 2011: Organization, W.H.

WHO. 2011b. Waist circumference and waist-hip ratio: report of a WHO expert consultation, Geneva, 8-11 December 2008.

WHO. 2015a. Diabetes: Fact sheet. <http://www.who.int/mediacentre/factsheets/fs312/en/> Date of access: 12 October 2020.

WHO. 2015b. Guideline: sugars intake for adults and children: World Health Organization.

WHO. 2015c. Obesity and overweight: fact sheet. <http://www.who.int/mediacentre/factsheets/fs311/en/> Date of access: 20 November 2020.

WHO. 2017a. Cardiovascular disease: Factsheet. <http://www.who.int/mediacentre/factsheets/fs317/en/> Date of access: 9 November 2020.

WHO. 2017b. Double-duty actions for nutrition: policy brief.

WHO. 2017c. Obesity and overweight: fact sheet. <http://www.who.int/mediacentre/factsheets/fs311/en/> Date of access: 9 November 2017.

WHO. 2018a. Diabetes: Fact sheet. <http://www.who.int/mediacentre/factsheets/fs312/en/> Date of access: 21 October 2019.

WHO. 2018b. Draft guidelines on saturated fatty acid and trans-fatty acid intake for adults and children. *WHO [https://extranet.who.int/dataform/upload/surveys/666752/files/Draft%20WHO%20SFA-TFA%20guidelines_04052018%20Public%20Consultation%20\(1\).pdf](https://extranet.who.int/dataform/upload/surveys/666752/files/Draft%20WHO%20SFA-TFA%20guidelines_04052018%20Public%20Consultation%20(1).pdf)* Date of access: 14 July 2020.

WHO. 2018c. Fact sheet: Healthy diet. https://www.who.int/nutrition/publications/nutrientrequirements/healthy_diet_fact_sheet_394.pdf?ua=1 Date of access: 27 October 2019.

WHO. 2018d. Fact sheet: Noncommunicable diseases. <https://www.who.int/en/news-room/fact-sheets/detail/noncommunicable-diseases> Date of access: 27 October 2019.

WHO. 2018e. Global health estimates 2016: disease burden by cause, age sex, by country and by region, 2000-2016. http://www.who.int/gho/mortality_burden_disease/en/index.htm Date of access: 15 October 2019.

WHO. 2018f. Obesity and overweight: Fact Sheet. <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight> Date of access: 21 October 2019.

WHO. 2018g. Physical activity: Fact Sheet. <https://www.who.int/news-room/fact-sheets/detail/physical-activity> Date of access: 21 October 2019.

WHO. 2019. Hypertension: Fact Sheet. <https://www.who.int/news-room/fact-sheets/detail/hypertension> Date of access: 21 October 2019.

Wicks, M. 2012. The validation of a suitable nutrient profiling model for South Africa. Potchefstroom: North-West University. (MSc Thesis).

Wicks, M., Wright, H. & Wentzel-Viljoen, E. 2016. Restricting the marketing of foods and non-alcoholic beverages to children in South Africa: are all nutrient profiling models the same? *British Journal of Nutrition*, 116(12):2150-2159. <https://doi.org/10.1017/S0007114516004244>

Willett, W. 1998. Nutritional Epidemiology Monographs in Epidemiology and Biostatistics, vol. 30: Oxford: Oxford University Press. <https://doi.org/10.1093/acprof:oso/9780195122978.001.0001>

Worldbank. 2017. Rural populations (as % of total population), World Bank Staff estimates based on United Nations, World Urbanization Prospects. <http://data.worldbank.org/indicator/SP.RUR.TOTL.ZS> Date of access: 10 November 2019.

Wu, H. & Ballantyne, C.M. 2020. Metabolic inflammation and insulin resistance in obesity. *Circulation Research*, 126(11):1549-1564. <https://doi.org/10.1161/CIRCRESAHA.119.315896>

Yang, W.-S., Chen, Y.-Y., Chen, P.-C., Hsu, H.-C., Su, T.-C., Lin, H.-J., . . . Chien, K.-L. 2019. Association between Plasma N-6 Polyunsaturated Fatty Acids Levels and the Risk of Cardiovascular Disease in a Community-based Cohort Study. *Scientific Reports*, 9(1). <https://doi.org/10.1038/s41598-019-55686-7>

Yu, C., Chen, Y., Cline, G.W., Zhang, D., Zong, H., Wang, Y., . . . Cooney, G.J. 2002. Mechanism by which fatty acids inhibit insulin activation of insulin receptor substrate-1 (IRS-1)-associated phosphatidylinositol 3-kinase activity in muscle. *Journal of Biological Chemistry*, 277(52):50230-50236. <https://doi.org/10.1074/jbc.M200958200>

Yusuf, S., Joseph, P., Rangarajan, S., Islam, S., Mente, A., Hystad, P., . . . Wielgosz, A. 2020. Modifiable risk factors, cardiovascular disease, and mortality in 155 722 individuals from 21 high-income, middle-income, and low-income countries (PURE): a prospective cohort study. *The Lancet*, 395(10226):795-808. [https://doi.org/10.1016/S0140-6736\(19\)32008-2](https://doi.org/10.1016/S0140-6736(19)32008-2)

Yuyun, M.F., Sliwa, K., Kengne, A.P., Mocumbi, A.O. & Bukhman, G. 2020. Cardiovascular Diseases in Sub-Saharan Africa Compared to High-Income Countries: An Epidemiological Perspective. *Global Heart*, 15(1):15. <https://doi.org/10.5334/gh.403>

Zhang, L., Zhang, W.-H., Zhang, L. & Wang, P.-Y. 2011. Prevalence of overweight/obesity and its associations with hypertension, diabetes, dyslipidemia, and metabolic syndrome: a survey in

the suburban area of Beijing, 2007. *Obesity facts*, 4(4):284-289.
<https://doi.org/10.1371/journal.pone.0070893>

Zhang, Z.-q., Deng, J., He, L.-p., Ling, W.-h., Su, Y.-x. & Chen, Y.-m. 2013. Comparison of various anthropometric and body fat indices in identifying cardiometabolic disturbances in Chinese men and women. *PloS One*, 8(8):e70893.
<https://doi.org/10.1371/journal.pone.0070893>

Zong, G., Li, Y., Wanders, A.J., Alsema, M., Zock, P.L., Willett, W.C., . . . Sun, Q. 2016. Intake of individual saturated fatty acids and risk of coronary heart disease in US men and women: two prospective longitudinal cohort studies. *BMJ*, 355:i5796.
<https://doi.org/10.1136/bmj.i5796>

ADDENDUM A DIETARY INTAKE ASSESSMENT METHODOLOGY

1. Introduction

The PURE-SA-NWP study is part of the international Prospective Urban and Rural Epidemiological study (PURE), the largest international study on the influences of the environment, lifestyle, and biology on NCDs such as cardiovascular disease, diabetes, and cancer. The PURE study is coordinated by the Population Health Research Institute in Ontario, Canada, conducted in 27 countries/regions with more than 150 000 participants globally (Teo et al., 2009). Internationally the PURE study resulted in more than a hundred publications up to date, 58 of which included data from the South African leg of the study. Most of these publications focussed on various aspects of cardiometabolic health.

This addendum describes the dietary intake assessment methodology that was used during the PURE-SA-NWP study from 2005 to 2015 to ensure data quality. The following elements are included: i) The use and development of the Food Frequency Questionnaire (QFFQ), ii) Training of the fieldworkers, iii) Dietary data collection process, iv) Development of the photobook, v) quality control, vi) Dietary data and analysis.

2. Dietary data collection: Methods and data analysis

(i) The use and development of the Quantified Food Frequency Questionnaire (QFFQ)

Food composition of individuals can be measured by using two general groups of methods. Firstly, there is the method where food consumption quantities are recorded over a short term such as a 1-day period. Additional assessment days can be included to estimate the habitual intake of an individual. These methods include 24-hr recalls, repeated 24-hr recalls, estimated food records and weighted food records. The second group of methods records retrospective habitual food intake over a longer time period. These methods include the dietary history method, food frequency questionnaires (FFQ) and quantified food frequency questionnaires (QFFQ) (Gibson, 2005).

As mentioned in Chapter 1 of this thesis, the PURE-SA-NWP study is part of the international Prospective Urban and Rural Epidemiological study (PURE), coordinated by the Population Health Research Institute in Ontario, Canada, conducted in 27 countries/regions (Teo *et al.*, 2009). To ensure comparability of dietary intake between countries, it was important that all countries/regions used the same dietary data collection method. Based on the virtues of the QFFQ (as discussed below) and the aim of the PURE study, the researchers of the international

PURE study selected the QFFQ as the dietary data collection method to be used in all regions of the study (Teo *et al.*, 2009).

An FFQ is a set list of foods and beverages that measures how often these items are consumed in a pre-determined retrospective period. When originally designed, the FFQ aimed to provide qualitative descriptive data of food and beverage consumption (Gibson, 2005). It does however become quantified when estimated portion sizes are added, resulting in a QFFQ (Gibson, 2005). A QFFQ does not merely provide descriptive data, but the data is now quantifiable, and the habitual nutrient and energy intake of individuals can be calculated. The use of QFFQs as a dietary intake data collection method has its shortcomings and has been criticised due to reservations regarding its level of accuracy (Freedman *et al.*, 2006; Schatzkin *et al.*, 2003). In the Transition and Health During Urbanisation of South Africans (THUSA) study, for instance, QFFQs were found to under-report energy and fat intakes in comparison with 7-day weighted records (MacIntyre *et al.*, 2001b).

Important strengths of the QFFQ are that it is relatively inexpensive to administer, it measures dietary intake over a longer period of time (circumventing any recent changes to the respondent's diet) and it places a significantly lower burden on the respondents than do other methods, such as 7-day weighted records (Thompson & Subar, 2017). Because the QFFQ remains the preferred dietary epidemiological method of investigating associations between health outcomes and dietary intake (Gibson, 2005; Thompson & Subar, 2017; Wentzel-Viljoen *et al.*, 2011), it was selected as the preferred method of dietary data collection in the PURE study (Teo *et al.*, 2009).

The project office of the international PURE study either developed and validated country-specific QFFQs for countries, or used existing, validated QFFQs (Teo *et al.*, 2009). The PURE-SA-NWP study falls into the latter group. Dietary intake data for the PURE-SA-NWP study were collected using a 145-item culturally sensitive QFFQ assessing food consumption over a 30-day period (Addendum B). The specific QFFQ used in the PURE-SA-NWP study was developed (MacIntyre *et al.*, 2001a) and validated (MacIntyre *et al.*, 2001b) in a similar group of participants from the same culture and demographic region, and has shown good reproducibility (MacIntyre *et al.*, 2001a; Wentzel-Viljoen *et al.*, 2011).

(ii) Training of the fieldworkers

Dietary intake data were collected by the same team of fieldworkers in 2005 and 2010. These fieldworkers were community members from the respective urban and rural areas where the study took place. They were comprehensively trained in administering the QFFQs by a team of experts in the field of dietary intake assessment methodology. During the training of all fieldworkers, skills

in administering QFFQs effectively and accurately were taught and each fieldworker was provided with a training manual to use as reference when needed. Additionally, the fieldworkers were trained to have a clear understanding of portion sizes and how to use different portion size aids to determine portion sizes accurately. The training also included a practical component where fieldworkers could practise their newly obtained knowledge, closely monitored and guided by the team of experts. These fieldworkers were retrained prior to the 2010 data collection.

In 2015, new fieldworkers were recruited from the urban community and travelled with the research team to conduct the questionnaires in the rural as well as urban communities. These fieldworkers received the same comprehensive training to administer the QFFQs, by the same group of experts who trained the first group of fieldworkers in 2005 and 2010. The fieldworkers were appointed as temporary employees for the duration of the study and were thus motivated to be accurate in the collection of data.

Dietary data collection process

In 2005, the dietary data collection did not take place at the research facility as part of the larger data collection process. The fieldworkers were transported to the homes of the participants, where they administered the QFFQs.

In 2010 and 2015, dietary data collection was administered at the research facility over several weeks. Each day, groups of participants would arrive at the facility in a fasting state. After blood collection and a light breakfast, participants visited several data collection stations where questionnaires or tests were administered.

The QFFQs were printed in English; however, the fieldworkers were competent to conduct each questionnaire in the language the participant preferred (English, Setswana, or Afrikaans).

The following is applicable to all three data collection (2005, 2010 and 2015) periods: To estimate portion sizes, fieldworkers used a photobook specifically developed for use in a similar group living in the North West Province (Venter *et al.*, 2000). Each fieldworker was equipped with a collection of portion size aids, such as spoons of different sizes, measuring apparatus, bowls of different sizes and other tools to help participants identify the correct portion sizes. An additional file with examples of food products often consumed by the rural and urban groups of participants (compiled through the course of the study) was also included. The file included photos of a selection of foodstuffs and the different sizes in which they were available (e.g. chocolate bars of different brands and sizes or different brands of margarine). In cases where the participants reported the consumption of a product that was not familiar to either the fieldworkers or the quality controller, the participant was questioned about the specific retail outlet or street vendor where

the products were purchased. Samples of the product were subsequently bought from this retail outlet/vendor to be correctly identified.

(iii) Development of the photobook

One of the aids to estimate portion sizes in the PURE-SA-NWP study was a photobook developed by Venter *et al.* (2000) for use in the earlier THUSA study, also conducted in the North-West province. To determine the most commonly consumed foods, portion sizes and preparation methods, the developers of the photobook conducted in-depth interviews and focus group discussions. Hereafter they prepared the food accordingly and photographed it. In addition, photos of common household measures, such as spoons, mugs, classes etc., were also included. The photos were printed to be as close to life-size as possible.

The photograph book was tested to determine its usefulness as a visual aid for the administration of the QFFQ (Venter *et al.*, 2000). It proved to be useful as well as convenient. Because of this and the fact that it was developed for a group similar in culture and locality, it was included as a visual aid in the PURE-SA-NWP data collection.

(iv) Quality control

In 2005, 2010 and 2015, real-time quality control during the data collection process was done by a registered dietitian with extensive knowledge and experience/skills/expertise in dietary intake assessment methodology. During the 2015 data collection period this quality control was done by the PhD candidate (also a registered dietitian), who previously had received extensive training before commencement of the data collection. Quality control took place immediately after the fieldworkers conducted the QFFQ interview with each participant while the participant was still on the data collection site. This enabled the fieldworkers to rectify any problems that were identified by the quality control process.

Daily fieldworker meetings were held to discuss any concerns or problems that arose during the day. New/unfamiliar products were discussed at these meetings, ensuring that all fieldworkers recorded the consumption of such products in the same way. Overall, the daily fieldworker meetings ensured that the quality of questionnaire administration stayed consistent.

(v) Dietary data and analysis

During the data collection interviews for all three data collection (2005, 2010 and 2015) periods, the portion sizes were captured in household measures onto the QFFQ. After all data collections, these measures were converted to grams, using standard South African tables (Langenhoven *et al.*, 1991) and captured in the designated column on the QFFQ. Hereafter, the consumed foods

were coded according to the South African Food Composition Database of the South African Medical Research Council (MRC) – in 2005, the 1991 version of the database was used (Langenhoven, 1991), while the updated 2010 version of the database (Wolmarans *et al.*, 2010) was used in both 2010 and 2015. The PhD candidate was trained in coding the QFFQs and coded the 2010 as well as the 2015 data. In cases where food products were reported by study participants for which no code existed in the database, a request was made to the MRC to add the food product to the database. Once the food product was added to the database, a code was assigned to it and it was used for further data capturing. Because combined dishes are often complex informed assumptions regarding the ratios of ingredients were made, using the recipes of the South African food composition tables (again, the 1991 version was used in 2005 and the 2010 version in 2010 and 2015) (Langenhoven, 1991; Wolmarans *et al.*, 2010). Assumptions made in 2005 were applied again in both 2010 and 2015. Likewise, new assumptions made in 2010 were applied again in 2015.

The following steps are applicable to 2005, 2010 and 2015: Once all food items were coded and portion sizes converted to grams, the data for each individual participant was transferred from the QFFQ onto an Excel spreadsheet. After compiling a spreadsheet containing all of the participants' data, the data were cleaned to correct any coding or capturing errors. A biostatistician at the MRC used the South African Food Composition Database of the MRC to calculate the nutrient and energy intake of each participant. Before starting with statistical analysis, the dataset was again systematically screened to ensure that all data were entered and correctly calculated. Participants with the highest and lowest intakes of energy and nutrients (the top- and bottom-20 participants for each nutrient) were flagged and their QFFQs were examined for possible mistakes. This process entailed checking that the correct food codes and accurate amounts were captured.

3. Conclusion

This addendum presented the dietary assessment methodology that was followed during the 2005, 2010 and 2015 data collection periods. Dietary assessment methods do have their limitations; however, to ensure the quality of data in this study, proper training and various control steps were implemented in the manner in which dietary intake data were collected, monitored, collated and analysed. The PURE study team is confident in the quality of the dietary data that were captured for this study.

4. References

- Freedman, L.S., Potischman, N., Kipnis, V., Midthune, D., Schatzkin, A., Thompson, F.E., . . . Carroll, R. 2006. A comparison of two dietary instruments for evaluating the fat–breast cancer relationship. *International Journal of Epidemiology*, 35(4):1011-1021. <https://doi.org/10.1093/ije/dyl085>
- Gibson, R.S. 2005. Principles of nutritional assessment: Oxford university press, USA.
- Langenhoven, M., Conradie, P., Wolmarans, P. & Faber, M. 1991. MRC food quantities manual. *Cape Town: South African Medical Research Council*, 2.
- Langenhoven, M.L., Kruger, M., Gows, E., Faber, M. 1991. MRC food composition tables: South African Medical Research Council, Research Institute for Nutritional Diseases.
- MacIntyre, U., Venter, C. & Vorster, H. 2001a. A culture-sensitive quantitative food frequency questionnaire used in an African population: 1. Development and reproducibility. *Public Health Nutrition*, 4(1):53-62. <https://doi.org/10.1079/PHN200040>
- MacIntyre, U., Venter, C. & Vorster, H. 2001b. A culture-sensitive quantitative food frequency questionnaire used in an African population: 2. Relative validation by 7-day weighed records and biomarkers. *Public Health Nutrition*, 4(1):63-71. <https://doi.org/10.1079/PHN200041>
- Schatzkin, A., Kipnis, V., Carroll, R.J., Midthune, D., Subar, A.F., Bingham, S., . . . Freedman, L.S. 2003. A comparison of a food frequency questionnaire with a 24-hour recall for use in an epidemiological cohort study: results from the biomarker-based Observing Protein and Energy Nutrition (OPEN) study. *International Journal of Epidemiology*, 32(6):1054-1062. <https://doi.org/10.1093/ije/dyg264>
- Teo, K., Chow, C.K., Vaz, M., Rangarajan, S. & Yusuf, S. 2009. The Prospective Urban Rural Epidemiology (PURE) study: examining the impact of societal influences on chronic noncommunicable diseases in low-, middle-, and high-income countries. *American Heart Journal*, 158(1):1-7. e1. <https://doi.org/10.1016/j.ahj.2009.04.019>
- Thompson, F.E. & Subar, A.F. 2017. Dietary assessment methodology. *Nutrition in the Prevention and Treatment of Disease*. Elsevier: 5-48. <https://doi.org/10.1016/B978-0-12-802928-2.00001-1>
- Venter, C., MacIntyre, U. & Vorster, H. 2000. The development and testing of a food portion photograph book for use in an African population. *Journal of Human Nutrition and Dietetics*, 13(3):205-218. <https://doi.org/10.1046/j.1365-277x.2000.00228.x>
- Wentzel-Viljoen, E., Laubscher, R. & Kruger, A. 2011. Using different approaches to assess the reproducibility of a culturally sensitive quantified food frequency questionnaire. *South African Journal of Clinical Nutrition*, 24(3):143-148. <https://doi.org/10.1080/16070658.2011.11734366>
- Wolmarans, P., Danster, N., Dalton, A., Rossouw, K. & Schönfeldt, H. 2010. Condensed food composition tables for South Africa. *Cape Town: Medical Research Council*:1-126.

ADDENDUM B QUANTIFIED FOOD FREQUENCY QUESTIONNAIRE

PURE/South Africa 2015 Quantitative Food Frequency Questionnaire

Subject Code

Name of fieldworker: _____

Subject ID

Centre #

Community #

Household #

Subject #

Subject Initials

F M L

Today's date: Day of the week: _____
year month day

1. Name of the PARTICIPANT: _____

2. Not applicable in South Africa

3. National identity # or equivalent _____ N/A ☐

4. DOB: AND Age years

5. Sex: ☐ Female ☐ Male

Please think carefully about the food and drink you have consumed during the **PAST MONTH** (four weeks). We have divided the foods into different groups for example all the porridges and cereals together. I will go through a list of food groups and drinks with you and I would like you to tell me:

- Which foods you eat in each of the different food groups
- How the food is prepared
- How much of the food you eat at a time
- How many times a day you eat it and if you do not eat it everyday, how many times a week or a month you eat it.

To help you to describe the amount of a food you eat, I will show you pictures of different amounts of the food as well as other food models, containers, etc.

There are no right or wrong answers.

Everything you tell me is confidential. Only your subject number appears on the form.

Is there anything you want to ask now?

Are you willing to go on with the questions?

Before we start I would like to find out what type of margarine, oil and milk you **USUALLY** use in your home.

1. What type of **MARGARINE** do you USUALLY use in your home? Give brand name if possible

☐ Tub/Soft margarine (brand name) _____

☐ Brick/Hard margarine (brand name) _____

☐ Do not use margarine in home

2. What type of **OIL** do you USUALLY use in the preparation of food in your home?

☐ Sunflower oil (give brand name) _____

☐ Canola oil (give brand name) _____

☐ Olive oil (give brand name) _____

☐ Other (give brand name) _____

☐ Oil previously used _____

☐ Do not use OIL ever in the home

3. What type of **MILK** do you USUALLY use in your home? Mark only ONE

☐ Full cream milk / Fresh cow's milk/ Box milk full cream

☐ Low fat milk / 2% milk / Box low fat or 2% milk

☐ Fat free milk / Skim milk / Box fat free or skim milk

☐ Powder milk (eg Elite; give brand name) _____

☐ Do not use milk

4. What type of **CREAMER** do you USUALLY use in your home?

☐ Cremora, Ellis Brown, Coffee Mate, Tea Mate etc

☐ Cremora Lite

☐ Do not use creamer

QUANTIFIED FOOD FREQUENCY QUESTIONNAIRE 2010

INSTRUCTIONS: Circle the subject's answer. Fill in the amount and times eaten in the appropriate columns.

I shall now ask you about the type and the amount of food you have been eating in the **LAST MONTH**. Please tell if you eat the food, how much you eat and how often you eat it. We shall start with maize meal porridge.

<u>PORRIDGE AND BREAKFAST CEREALS</u>								
FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom /Never		
Maize-meal porridge	Stiff (pap)						4401	
Maize-meal porridge	Soft (slappap)						4400	
Maize-meal porridge	Crumbly (phutu)						4402	
Ting	Maize meal						4429	
	Mabella						4427	
	Other							
Mabella	Stiff						3437	
	Soft							
Morvite	Soft						4404	
Oats							3239	
Tastee wheat	Soft						3240	
Other cooked porridge	Type							
Breakfast cereals	All bran flakes						3242	
	Corn flakes plain						3243	
	Weetbix						3244	
	Rice crispies plain						3252	
	Other							

OTHER STARCH								
FOOD	DESCRIPTION	AMOUNT	TIMES EATEN 2 columns to be completed				CODE	AMOUNT / WEEK
			Per day	Per week	Per month	Seldom /Never		
Samp	Bought Self ground						3250	
Samp and beans	Give ratio of samp:beans						3402 (1:1)	
Samp and peanuts	Give ratio of samp:peanuts						3250 (samp) 3458	
Rice	White						3247	
	Brown						3315	
	Maize Rice						3250	
	Any fat added?							
Pasta	Macaroni, plain						3262	
	Spaghetti, plain						3262	
	Spaghetti, canned in tomato sauce						3258	
	Macaroni & cheese Milk: Fat:							
	Other specify							
Pizza	Home made: Specify topping						3353 (base+ch)	
	Bought: Specify topping						3353 (base+ch)	

You are being very helpful. Can I now ask you about meat?

CHICKEN, MEAT, FISH

How many times do you eat meat (beef, mutton, pork, chicken, fish) per week? _____

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN 2 columns to be completed				CODE	AMOUNT / WEEK
			Per day	Per week	Per month	Seldom /Never		
Chicken	Meat & skin, boiled						2926	
	Meat & skin, roasted/grilled/fried in chicken fat						2925	
	Meat ONLY, boiled (no skin)						1559	
	Meat ONLY, roasted (no skin)						1545	
	White meat only, cooked (NO Skin)						2964	
	Kentucky / Chicken Licken (Fried in batter/crums)						3018	
	Nando's						2925	
	Other							
Do you eat chicken skin?		Always	1	Sometimes	2	Never	3	
Chicken stew	With potato and onion WITH skin						4413	
	With tomato and onion WITH skin						2985	
	With vegetables WITH skin						3005	
	With tomato and onion NO skin						4379	
	With vegetables NO skin						4378	
Chicken BONE stew	With potato and onion and tomato						4414	
	Other							
Chicken feet	Nothing added						2997	
	Stew with potato, onion and tomato						4415	
Chicken head							2999	
Chicken offal	Stew with tomato and onion and sunflower oil						4416	
	Liver, cooked						2970	
	Other							

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom /Never		
RED MEAT	How do you like your meat?		With fat	OR	Fat trimmed			
Red meat BEEF	BRISKET, boiled/fried without added fat						4363	
	CHUCK, boiled/fried without added fat						2945	
	BRISKET, fried in added fat Type of fat:						4363	
	CHUCK, fried in added fat Type of fat:						2945	
	Beef, stewed with cabbage						3006	
	Beef, stewed with potato and onion and tomato						4417	
	Beef, stewed with vegetables						3020	
	Mince , nothing added						2921	
	Mince, tomato & onion added						2987	
	Beef BONE stew with potato and onion and oil						4419	
	Other							
	MUTTON	Meat, with fat, cooked						2947
Mutton, no fat, cooked							3036	
Mutton, chop, grilled							2927	
Mutton, stewed with vegetables							2916	
Other								

BRISKET: more fat and cheaper
 CHUCK: less fat than the brisket, but more expensive

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom /Never		
Beef/mutton Offal	Offal, cooked						3003	
	Stewed with vegetables							
	Liver, beef, fried/cooked						2920	
	Liver, sheep, fried/cooked						2955	
	Kidney, beef, cooked						2923	
	Kidney, sheep, cooked						2956	
	Brain, sheep, cooked						2952	
	Lung, beef, cooked						3019	
	Lung, sheep, cooked						4337	
	"Germalde" (lung & fat)						4409	
	Heart, beef, cooked						2968	
	Heart, sheep, cooked						2969	
	Other							
	Goat meat	Grilled/roasted/cooked						4281
Stewed with vegetables								
Other								
Venison/ Wild buck							2913	
Horse/Donkey							4407	
Rabbit							4327	
Other type of meat	Specify							
What type of vegetables is usually put into meat stews?								

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom / Never		
Wors / Sausage							2931	
Bacon							2906	
Patties	Beef, fried						2984	
	Chicken, fried						3011	
Cold meats AND Processed meats	Polony						2919	
	Ham						2967	
	Vienna						2936	
	Frankfurter, beef & pork						2937	
	Frankfurter/Sausage, chicken						3012	
	Russian/Salami						2948	
	Other							
Canned meat	Bully beef, plain						2940	
	Bully beef with potato & onion & oil						2994	
	Other							
Meat pie BOUGHT Or HOMEMADE	Beef						2939	
	Steak and kidney						2957	
	Sausage roll						2939	
	Cornish						2953	
	Chicken						2954	
	Other							
Hamburger	Bought						4418	
	Other							
Biltong	Beef (with fat OR without fat)						3021	
Dried wors Dried sausage	Beef						2949	

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom / Never		
Dried beans	Baked beans in tomato sauce						3176	
	Bean salad / Sousbone						3174	
	Soup with dried beans, beef and vegetables						3145	
	Sugar beans, cooked						3205	
	Other							
Lentils	Whole, cooked						3203	
	Lentil soup with beef and vegetables						3153	
Soya products eg. Imana, Knorr, Jileleke, Toppers	Cooked						3196	
	Soup/Gravy						4431	
	Stewed with extra potato, onion and tomato						4430	
	Other							

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom / Never		
Pilchards in tomato sauce or chilli or brine	Whole						3102	
	Mashed with fried onion						3102 (70%) 3730 (30%)	
	With tomato and onion						4420	
	Other							
Fish	Hake, fried with batter/crums						3072	
	Hake, fried in oil						3060	
	Hake, steamed						4373	
	Moddervis / Yellow fish fried in oil						3084	
	Moddervis / Yellow fish baked with onion (NO oil added)						3089	
	Other							
Other canned fish	Tuna in oil						3056	
	Other							
Fish cakes	Bought: Fried						3080	
	Home made with potato						3098	
Fish fingers	Bought						3081	
Eggs	Boiled/poached						2867	
	Scrambled (full cream milk & oil)						2890	
	Scrambled (NO milk, ONLY oil added)						2869	
	Scrambled (NO oil, ONLY full cream milk)						2872	
	Fried in oil						2869	
	Fried in brick margarine						2877	
	Other							

VEGETABLES								
FOOD	DESCRIPTION	AMOUNT	TIMES EATEN 2 columns to be completed				CODE	AMOUNT / WEEK
			Per day	Per week	Per month	Seldom /Never		
Cabbage	How do you cook cabbage?							
	Boiled, nothing added						3756	
	Boiled with potato and onion and oil						3815	
	Boiled with potato and onion and brick margarine						3813	
	Fried in oil						3812	
	Fried in brick margarine						3810	
	Boiled with potato, onion and tomato and oil						4421	
	Raw with nothing added						3704	
	Other							
Spinach or morogo or beetroot leaves or other green leafy	How do you cook spinach?							
	Boiled, nothing added						3913	
	Boiled with oil added							
	Boiled with brick margarine added						3898	
	Boiled with tub margarine added						3899	
	Boiled with potato, onion and tomato and oil						4422	
	Other							
Tomato and onion gravy	With oil						4423	
	Without fat						3925	
	Canned						4192	
	Thickened with packet soup powder						4432	
	Other							

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom /Never		
Pumpkin (yellow) Butternut Hubbard squash Table Queen Etc	Boiled, nothing added						4164	
	Boiled with sugar only (NO fat)						3728	
	Boiled with brick margarine & sugar						3893	
	Boiled with tub margarine and sugar						4433	
	Boiled with oil and sugar						4428	
	Other							
Carrots	Boiled, nothing added						3757	
	Boiled with oil added							
	Boiled with brick margarine added						3816	
	Boiled with tub margarine added						3817	
	Boiled with sugar only						3818	
	Boiled with oil and sugar							
	Boiled with brick margarine and sugar						3819	
	Boiled with tub margarine and sugar						3820	
	Boiled with potato, onion and oil						3824	
	Boiled with potato, onion and brick margarine						3822	
	Boiled with potato, onion and tub margarine							
	Chakalaka						4412	
	Raw, nothing added						3709	
	Other							
Mealies/ Sweet corn	On cob – fat added Fat						3725	
	On cob – no fat added						3725	
	Creamed sweet corn / canned						3726	
	Whole kernel/canned						3942	
	Whole kernel, frozen, boiled						4132	
	Other							

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FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom /Never		
Beetroot	Salad						3699	
	Boiled, nothing added						3698	
How do you cook potatoes?								
Potatoes	Boiled/baked with skin						4155	
	Boiled/baked without skin						3737	
	Boiled with oil added						3873	
	Boiled with brick margarine added						3867	
	Boiled with tub margarine added						3868	
	Mashed with whole milk and brick margarine						3876	
	Mashed with whole milk and oil							
	Roasted in beef fat						3878	
	Roasted in oil						3979	
	French fries (chips) / Fried potatoes						3740	
	Other							
Sweet potatoes	How do you cook sweet potatoes?							
	Boiled/baked with skin						3748	
	Boiled/baked without skin						3903	
	Boiled with sugar and oil added						4434	
	Boiled with sugar and brick margarine added						3749	
	Other							

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom /Never		
Broccoli	Boiled						3701	
	Raw						3702	
Cauliflower	Boiled						3716	
Green beans	Boiled, nothing added						3696	
	Cooked with potato, onion and oil						3794	
	Cooked with potato, onion and brick margarine						3792	
	Other							
Mixed vegetables	Canned						4264	
	Frozen, boiled						3727	
	Other							
Salad vegetables	Mixed salad: tomato, lettuce and cucumber						3921	
	Raw tomato						3750	
	Cucumber, raw						4119	
	Coleslaw (cabbage) (mayonnaise)						3705	
	Coleslaw (cabbage) (commercial)						3707	
	Potato salad (mayonnaise)						3928	
	Baked bean salad						4424	
	Other salad vegetables							
Mayonnaise / salad dressing	Mayonnaise						3488	
	Vinegar, oil						3487	
	Other: Specify							
Other vegetables, specify + preparation								

Now we come to fruit								
FRUIT								
Do you like fruit?			☐ Yes 1 ☐ No 2					
FOOD	DESCRIPTION	AMOUNT	TIMES EATEN 2 columns to be completed				CODE	AMOUNT / WEEK
			Per day	Per week	Per month	Seldom /Never		
Apples							3592	
Banana							3540	
Pears							3582	
Oranges							3560	
Naartjie							3558	
Grapes							3550	
Peaches	Fresh						3565	
	Canned						3567	
Apricots	Fresh						3534	
	Canned						3535	
Mangoes							3556	
Guavas	Fresh						3551	
	Canned						3553	
Watermelon	Fresh						3576	
Fruit salad	Fresh						3588	
	Canned						3580	
Fig (Vye)							3544	
Avocado							3656	
Wild fruit/berries	Specify type							

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom /Never		
Dried fruit	Apple, dried, raw						3600	
	Peach, dried, raw						3568	
	Mixed fruit, dried, raw						3593	
	Mixed fruit, dried and cooked with sugar						3590	
	Fruit roll, dried (all types)						3655	
	Other							
Other fruit								

If subject eats canned fruit: Do you have custard with the canned fruit or fresh fruit salad?

☐ Yes 1

☐ No 2

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom /Never		
Custard	Homemade, full cream milk or fresh cow's milk						2716	
	Homemade, lowfat milk						2779	
	Homemade, skim milk						2717	
	Commercial eg Ultramel						2716	
	Other							

BREAD AND BREAD SPREADS								
FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom /Never		
Bread / Bread rolls	White						3210	
	Brown						3211	
	Whole wheat						3212	
Do you spread anything on the bread?			Always	1	Sometimes	2	Never	3
Margarine	What brand do you have at home now?							
	Tub, regular						3496	
	Tub, medium fat						4406	
	Tub, light/low fat						3524	
	Brick, regular						3484	
	Brick, medium fat						4405	
	Brick, lite/low fat						3528	
	Other							
Peanut butter						3485		
Jam/syrup/honey						3985		
Marmite / Fray bentos / Oxo						4058		
Fish/meat paste						3109		
Cheese	Cheddar						2722	
	Gouda						2723	
	Other							
Sandwich spread						3522		
Achaar						3117		
Other spreads	Specify							

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom /Never		
Dumpling	White flour						3210	
	Whole wheat flour						3212	
Vetkoek	White flour						3257	
	Whole wheat flour						3324	
Provita, crackers, etc	Provita						3235	
	Cream crackers						3230	
	Other savoury biscuits like Bacon kips, wheat crackers, etc						3331	

DRINKS								
FOOD	DESCRIPTION	AMOUNT	TIMES EATEN 2 columns to be completed				CODE	AMOUNT / WEEK
			Per day	Per week	Per month	Seldom / Never		
Tea	English (normal)						4038	
	Rooibos						4054	
Coffee							4037	
White sugar	Tea						3989	
	Coffee						3989	
Brown sugar	Tea						4005	
	Coffee						4005	
Milk per cup of TEA	Do you use milk in your TEA? ☐ Yes ☐ No		If YES, What type of milk do you use in <u>TEA</u> ?					
	Fresh / long life whole/full cream						2718	
	Fresh/long life: 2%/low fat						2772	
	Fresh/long life: fat free / skim milk						2775	
	Creamer/whitener like Ellis Brown / Cremora						2751	
	Cremora Lite							
	Condensed milk						2714	
	Evaporated milk						2715	
	Other							
	None							
Milk per cup of COFFEE	Do you use milk in your COFFEE? ☐ Yes ☐ No		If YES, What type of milk do you use in <u>COFFEE</u> ?					
	Fresh/long life: whole/full						2718	
	Fresh/long life: 2%/low fat						2772	
	Fresh/long life: fat free						2775	
	Creamer/whitener like Ellis Brown						2751	
	Cremora Lite							
	Condensed milk						2714	
	Evaporated milk						2715	
	Other							
	None							

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom / Never		
Milk as such	What type of milk do you drink milk as such?							
	Fresh/long life: whole / full cream milk						2718	
	Fresh/long life: 2% milk / low fat milk						2772	
	Fresh/long life: fat free / skim milk						2775	
	Condensed milk						2714	
	Sour/maas						2787	
	Other							
Milk drinks	Flavoured milk						2774	
	Milo made with full cream milk						2735	
	Milo made with skim milk						2747	
	Drinking chocolate made with water						4287	
	Other							
Yoghurt	Drinking yoghurt low fat						2756	
	Plain low fat						2734	
	Low fat sweetened with fruit						2732	
Squash	Sweet O						4027	
	Six O							
	Oros/Lecol – with sugar or other						3982	
	- artificially sweetener						3990	
	KoolAid						4027	
	Other							
Fizzy drinks	Sweetened						3981	
Coke, fanta, etc	Diet							

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom / Never		
Fruit juice	Fresh/Liquifruit/Ceres						2866	
	Tropica (Dairy –fruit juice mix)						2791	
	Other							
Mageu/Motogo							4056	
Home brew							4039	
Tlokwe							4039	
Beer							4031	
Cider	Sweet						4057	
Spirits Eg Brandy, gin, vodka, whisky, cane, etc							4035	
Wine red							4033	
Wine White							4033	
Other specify								
WATER	Tap, borehole, dam, river, etc						4042	
	Bottled						4042	

SNACKS AND SWEETS								
FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom / Never		
Potato crisps							3417	
Peanuts	Raw						4285	
	Roasted						3458	
Cheese curls, Niknaks, etc							3267	
Raisins							3552	
Peanuts and raisins								
Chocolates	Milk chocolate, plain						3987	
	Kit Kat etc						4024	
	Chocolate coated bars like Bar One, TV bar, etc						3997	
	Other							
Candies/Sweets	Sugus, gums, hard sweets, etc						4000	
Toffees / Fudge / caramels							3991	
Biscuits/cookies	Potch cookies						4425	
	Homemade, plain						3233	
	Commercial, plain						3216	
	Commercial, with filling						3217	
	Other							
Cakes	Butter cake, homemade with whole milk and brick margarine NO icing						3288	
	Chocolate cake, homemade with whole milk and brick margarine NO icing						3289	
	Icing for cake made with brick margarine						4014	
	Other							

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FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom / Never		
Tarts	Apple tart with a batter made with whole milk and brick margarine						3327	
	Other							
Scones	Plain made with whole milk and brick margarine						3237	
	Other							
Muffin	Bran						3407	
	Plain						3408	
	Other							
Rusks	Buttermilk, commercial						3329	
	Homemade, white						3222	
	Other							
Savouries	Sausage rolls						2939	
	Samoosas: Meat filling						3355	
	Samoosas: Vegetable filling						3414	
	Biscuits eg bacon kips						3331	
	Other							

FOOD	DESCRIPTION	AMOUNT	TIMES EATEN				CODE	AMOUNT / WEEK
			2 columns to be completed					
			Per day	Per week	Per month	Seldom / Never		
Jelly							3983	
	Custard added made with whole milk						2716	
	Other							
Baked pudding	Baked in a syrup						3312	
	Baked without a syrup						3429	
	Custard added made with whole milk						2716	
	Other							
Instant pudding	Made with whole milk						3266	
	Made with low fat milk						3395	
	Other							
Ice cream	Regular						3483	
	Soft serve						3518	
	Other							
Sorbet							3491	
Other specify								

SAUCES, GRAVIES AND CONDIMENTS								
FOOD	DESCRIPTION	AMOUNT	TIMES EATEN 2 columns to be completed				CODE	AMOUNT / WEEK
			Per day	Per week	Per month	Seldom / Never		
Tomato sauce							3139	
Worcester sauce							4309	
Chutney							3168	
Pickles							3866	
White sauce	Made with whole milk and brick margarine						3142	
Packet soups	Dry powder (all types)						3158	
	Made with water (all types)						3165	
Gravy	Made from meat and thickened						3120	
Other								

WILD FRUITS, WILD BIRDS, ANIMALS OR INCECTS (hunted in rural areas or on farms)								
FOOD	DESCRIPTION	AMOUNT	TIMES EATEN 2 columns to be completed				CODE	AMOUNT / WEEK
			Per day	Per week	Per month	Seldom / Never		
MISCELLANEOUS: Please mention <u>ANY OTHER FOODS</u> used more than once/two times a week which we have NOT talked about								
INDIGENOUS/TRADITIONAL FOODS/PLANTS/ANIMALS								
Please tell me if you use any indigenous plants OR other indigenous foods like mopani worms, locusts ect to eat PLEASE GIVE FETAELS								