

**MALE AND FEMALE CARDIOVASCULAR RISK IN AN URBAN, BLACK  
WORKING POPULATION**

**BY**

**LINDSAY MAY JACKSON**

**THESIS**

**Submitted in fulfillment of the requirements for the Degree  
Master of Science**

**Department of Human Kinetics and Ergonomics  
Rhodes University, 2010**

**Grahamstown, South Africa**

## ABSTRACT

The aim of this research project was to assess and compare cardiovascular disease (CVD) risk in black males and females from an urban, working population in the Makana (Grahamstown) region of the Eastern Cape, South Africa.

Two-hundred and ninety one individuals (males:  $n = 143$ , females:  $n = 148$ ) with a mean age of  $42.6 (\pm 8.1)$  years were voluntarily recruited from the greater urban Makana (Grahamstown) area. Eight Cardiovascular disease (CVD) risks were assessed: stature and mass were obtained in order to calculate body mass index (BMI) ( $\text{mass}/\text{stature}^2$ ). Obesity, defined as a morphological risk, was classified according to the World Health Organisation (WHO) BMI criteria ( $\text{BMI} > 30 \text{ kg} \cdot \text{m}^{-2}$ ), as well as according to measures of waist circumference (WC) and body composition. Hypertension, hypercholesterolemia and type II diabetes, were grouped as cardiovascular (CV) risks. Hypertension was defined as a blood pressure greater than 140/90mmHg (JNC-7); hypercholesterolemia, as total cholesterol greater than  $6.2 \text{ mmol} \cdot \text{L}^{-1}$  (NCEP); and type II diabetes, as total glucose greater than  $12 \text{ mmol} \cdot \text{L}^{-1}$  (WHO). Physical activity, diet, tobacco use, and alcohol consumption and dependence were grouped as lifestyle-related risks. These were assessed by means of self-reporting through the use of various validated questionnaires. Finally, self-reporting of obesity, hypertension, hypercholesterolemia and type II diabetes was assessed, in addition to perception questions on individuals' perceived body shape and size (Ziebland figures). Self-reported and perceived responses were then compared to actual measures.

Females were significantly ( $p < 0.001$ ) heavier than the males ( $92.7 \text{ kg}$  compared to  $72.1 \text{ kg}$ ) and had significantly ( $p < 0.001$ ) higher BMIs than their male counterparts ( $37.6 \text{ kg} \cdot \text{m}^{-2}$  compared to  $25.7 \text{ kg} \cdot \text{m}^{-2}$ ). They also recorded significantly ( $p < 0.001$ ) higher waist circumference (WC) values and had significantly ( $p < 0.001$ ) higher percentage and total body fat. Significantly ( $p < 0.001$ ) more females were obese (81%) compared to males (17%). While a higher percentage of males (25 % compared to 22%) presented with stage I hypertension ( $\geq 140/90 \text{ mmHg}$ ,  $< 160/95 \text{ mmHg}$ ), significantly ( $p < 0.05$ ) more females (14% compared to 8%) presented with stage II hypertension ( $> 160/95 \text{ mmHg}$ ). The prevalence of hypercholesterolemia at a high level of risk ( $> 6.2 \text{ mmol} \cdot \text{L}^{-1}$ ) was relatively low (2.1 % of males, 3.4% of females), but notably more participants (22% of males and 26% of females) presented with the condition at a moderate level of risk ( $> 5 \text{ mmol} \cdot \text{L}^{-1}$ ). Type II

diabetes was the least prevalent CV risk factor, with no males and only 3% of females presenting with the condition. Males consumed significantly ( $p<0.05$ ) more in terms of total energy intake (9024 vs. 7234 kJ) and were significantly ( $p<0.05$ ) more active (3315 compared to 2660 MET-mins.week). A significantly ( $p<0.05$ ) higher percentage of males smoked (51.1% compared to 3.4%), consumed alcohol (73.4% compared to 46.6%) and were alcohol dependent (40% compared to 33.5%). Both males and females tended to be ignorant of their health status, with both samples under-reporting obesity, hypertension and hypercholesterolemia, while over-reporting type II diabetes. Furthermore, obesity was significantly ( $p<0.05$ ) underestimated, with both male and female individuals perceiving themselves to be notably smaller than they actually were.

Physical activity and diet were important determinants of CVD risk in this black urban sample of individuals. Obesity, in particular central adiposity, was the most notable risk (particularly in females), followed by hypertension (particularly in males). Although some risks presented at a moderate level of risk, a clustering of risk factors was evident in both samples, with 12.6% and 41.2% of males and females presenting with two risk factors, and 2.8% and 8.1% of males and females respectively presenting with three risks.

## **ACKNOWLEDGEMENTS**

This thesis would not have been possible without the assistance and contributions of many individuals:

First and foremost I would like to extend my sincere thanks and gratitude to my supervisor, Dr Candice Christie for her help and guidance throughout the entire research process. Her continual assistance and encouragement have made this experience a thoroughly rewarding one, and one that would not have been possible without her.

Secondly, to Dr Sarah Radloff, for her assistance with the statistics used in this research project. She was extremely helpful and patient and I thank her for all the hours dedicated to helping me, despite a very busy schedule of her own.

To my research assistants, Mat, Janet, Jono, Jess, Ntombi, Lauren and Evi – thank you all for your many hours of assistance in the field. This project would not have been possible without you. In particular, my sincere thanks to Jessica Stack for helping at almost all testing sessions and for being my ‘right hand lady’ and to Eva-Maria Burford to her invaluable help with data entry.

To June McDougall for her assistance with finances and printing, and to Simon Wright, Janine Harris, Mark Hazell and Lunga Twaku for their assistance with subject recruitment and logistics with regards to testing sessions. Thanks also to all the supervisors who were extremely helpful during all testing sessions.

Thank you to my partner, Jono, for his love, support and encouragement throughout this last year. His guidance and support, particularly during the stressful times, have been amazing, and I thank him from the bottom of my heart for all he does and continues to do for me.

Finally my sincere thanks to my parents, John and Gail, and siblings, Hayley and Bradley, for their continued love and support. Thanks to them for the weekly catch-ups, the Sunday dinners, for always being interested in my work, and for being willing ears to share ideas with. Thanks especially to my dad, John Jackson, for the help with the proof-reading and input towards the end, amidst a busy and stressful exam period.

# TABLE OF CONTENTS

<b>CHAPTER I .....</b>	<b>1</b>
<b>INTRODUCTION .....</b>	<b>1</b>
BACKGROUND .....	1
STATEMENT OF THE PROBLEM .....	3
RESEARCH HYPOTHESES .....	3
STATISTICAL HYPOTHESES .....	3
DELIMITATIONS.....	4
LIMITATIONS.....	5
<b>CHAPTER II .....</b>	<b>7</b>
<b>REVIEW OF LITERATURE .....</b>	<b>7</b>
INTRODUCTION: CARDIOVASCULAR DISEASE GOBALLY .....	7
South Africa.....	7
THE EASTERN CAPE .....	9
POLITICAL CHANGES SHAPING CHANGES IN HEALTH.....	10
What is an ‘epidemiological transition’?.....	11
THE QUADRUPLE BURDEN OF DISEASE .....	12
SEX AND RACIAL CONSIDERATIONS .....	14
PROVINCIAL CONSIDERATIONS .....	16
THE EASTERN CAPE .....	16
NON-MODIFIABLE RISKS FOR CVD.....	17
Age, genetics, sex.....	17
MODIFIABLE RISKS FOR CVD.....	19
MORPHOLOGICAL RISK .....	19
OBESITY.....	19
“Healthy Obese” and other considerations specific to black South African women..	21
CARDIOVASCULAR RISKS .....	23
HYPERTENSION.....	23
HYPERCHOLESTEROLEMIA .....	25
TYPE II DIABETES .....	27
LIFESTYLE-RELATED RISKS.....	30
POOR DIET .....	30
The ‘nutrition transition’ .....	31
PHYSICAL INACTIVITY.....	35
TOBACCO USE .....	38

ALCOHOL CONSUMPTION .....	41
PERCEPTIONS OF HEALTH RISKS.....	43
CONCLUSION AND AIMS OF THIS RESEARCH .....	44
<b>CHAPTER III .....</b>	<b>46</b>
<b>METHODS.....</b>	<b>46</b>
EXPERIMENTAL DESIGN.....	46
Dependent and independent variables of interest .....	46
THE WORLD HEALTH ORGANISATION (WHO) STEP-WISE APPROACH .....	47
Use of the steps approach in the current study .....	48
MORPHOLOGICAL RISK .....	49
OBESITY.....	49
Body Mass Index (BMI) .....	50
Waist circumference (WC) .....	50
Body composition: Bioelectrical Impedance Analysis (BIA).....	52
CARDIOVASCULAR RISKS .....	53
HYPERTENSION.....	53
HYPERCHOLESTEROLEMIA .....	53
TYPE II DIABETES .....	55
LIFESTYLE-RELATED RISKS.....	56
DIET .....	56
24-hour dietary recall.....	56
PHYSICAL ACTIVITY .....	57
Physical activity questionnaires.....	57
Global Physical Activity Questionnaire (GPAQ) .....	58
TOBACCO USE .....	58
ALCOHOL CONSUMPTION AND DEPENDENCE .....	59
ETHICAL CONSIDERATIONS.....	61
PILOT RESEARCH AND TRAINING .....	61
EXPERIMENTAL PROCEDURES: EQUIPMENT AND MEASUREMENT PROCEDURES.....	62
MORPHOLOGICAL RISK .....	62
OBESITY.....	62
Stature.....	62
Body Mass .....	63
Body Mass Index (BMI) .....	63
Waist circumference (WC) .....	63
Body composition: Bioelectrical Impedance Analysis (BIA).....	63

PERCEPTUAL RESPONSES .....	63
Body perception illustrations .....	63
CARDIOVASCULAR (CV) RISKS .....	64
HYPERTENSION .....	64
Blood pressure measurement: The auscultatory technique .....	64
Medical Conditions Questionnaire.....	64
HYPERCHOLESTEROLEMIA .....	65
Blood cholesterol measurement: the 'pin-prick' method .....	65
TYPE II DIABETES .....	66
Blood glucose measurement: the 'pin-prick' method.....	66
Medical Conditions Questionnaire.....	66
LIFESTYLE RELATED RISKS .....	66
DIET .....	66
24-hour dietary recall.....	66
Dietary Intake Questionnaire.....	67
FoodFinder.....	67
PHYSICAL ACTIVITY .....	67
Global Physical Activity Questionnaire (GPAQ) .....	67
TOBACCO USE .....	69
Adapted STEP-wise questionnaire.....	69
ALCOHOL CONSUMPTION AND DEPENDENCE .....	70
Adapted questions for the assessment of alcohol use; The CAGE questionnaire....	70
STUDY POPULATION .....	70
VOLUNTEER CHARACTERISTICS.....	71
SAMPLE SIZE.....	71
EXPERIMENTAL PROCEDURES: PHASE 1 .....	71
EXPERIMENTAL PROCEDURES: PHASE 2 .....	73
DATA ANALSYES.....	74
<b>CHAPTER IV .....</b>	<b>75</b>
<b>RESULTS.....</b>	<b>75</b>
MORPHOLOGICAL RISK .....	76
OBESITY .....	76
Stature, mass and Body Mass Index (BMI) .....	76
Waist circumference (WC) .....	78
Body Composition .....	79
CARDIOVASCULAR (CV) RISKS.....	80
BLOOD PRESSURE .....	81

TOTAL BLOOD CHOLESTEROL .....	82
BLOOD GLUCOSE .....	83
LIFESTYLE-RELATED CHARACTERISTICS.....	84
PHYSICAL ACTIVITY .....	84
Total physical activity .....	84
Work-related physical activity .....	86
Leisure-time physical activity.....	87
Transport-related physical activity.....	88
DIET .....	88
SMOKING .....	92
ALCOHOL CONSUMPTION .....	93
SELF REPORTED AND PERCEIVED RISKS IN COMPARISON TO ACTUAL MEASURES.....	96
Self reported versus actual measures of hypercholesterolemia .....	96
Self reported versus actual measures of type II diabetes.....	97
Self reported versus actual measures of hypertension.....	98
Perceived versus actual prevalence of obesity .....	99
HYPOTHESES.....	101
Hypothesis 1: .....	101
Hypothesis 2: .....	101
Hypothesis 3: .....	102
Hypothesis 4: .....	102
<b>CHAPTER V.....</b>	<b>104</b>
<b>DISCUSSION .....</b>	<b>104</b>
MORPHOLOGICAL RISK .....	104
OBESITY.....	104
Lifestyle factors .....	108
Socio-demographic considerations .....	109
Waist circumference.....	110
Body Composition .....	111
CARDIOVASCULAR RISKS .....	114
HYPERTENSION.....	114
Factors influencing the development of hypertension .....	116
Hypertension and other CVD risks .....	117
HYPERCHOLESTEROLEMIA .....	118
TYPE II DIABETES .....	120
LIFESTYLE-RELATED RISKS.....	121



PHYSICAL ACTIVITY .....	121
DIETARY INTAKE .....	125
SMOKING .....	129
ALCOHOL CONSUMPTION .....	132
PERCEIVED VERSUS ACTUAL MEASURES OF RISKS .....	133
Self-reported versus actual hypercholesterolemia .....	133
Self-reported versus actual type II diabetes .....	134
Self-reported versus actual hypertension .....	134
Perceived versus actual prevalence of obesity .....	135
<b>CHAPTER VI.....</b>	<b>138</b>
<b>CONCLUSIONS AND RECOMMENDATIONS.....</b>	<b>138</b>
SUMMARY AND CONCLUSIONS OF PRESENT RESEARCH .....	138
RECOMMENDATIONS .....	142
<b>REFERENCES .....</b>	<b>145</b>
<b>APPENDICES .....</b>	<b>171</b>
APPENDIX 1: SUBJECT INFORMATION AND CONSENT FORM .....	171
CONSENT FORM .....	173
APPENDIX 2: QUESTIONNAIRES AND DATA SHEETS.....	175
APPENDIX 3: STATISTICAL TABLES.....	188

## LIST OF FIGURES

Figure 1: Map of South Africa showing the nine different provinces (shaded area represents the Eastern Cape) (sourced from Stats SA, 2006) .....	8
Figure 2: Map of the Eastern Cape, South Africa. Area 1 (small map, highlighted in red) represents the Eastern Cape and Area 2 (big map, highlighted in red) represents the Makana area in which Grahamstown is located. (Sourced from Wikipedia Commons: URL: commons.wikimedia.org).....	10
Figure 3: Factors contributing to diabetes and obesity (Sourced and adapted from Candib, 2007).....	29
Figure 4: Stages of the nutrition transition (Sourced and adapted from Popkin & Gordon-Larsen, 2004).....	32
Figure 5: Male subject having his stature measured by one of the research assistants .....	62
Figure 6: Blood pressure measurement of a male participant .....	65
Figure 7: Mean ( $\pm$ standard deviation) BMI classification of male and female participants.....	77
Figure 8: Percentage of males and females falling within different BMI classifications .....	77
Figure 9: Mean ( $\pm$ standard deviation) waist circumference of males and females ..	78
Figure 10: Classification of WC according to cut off points: percentage of males and females within each category .....	79
Figure 11: Mean ( $\pm$ standard deviation) percentage body fat of male and female participants.....	79
Figure 12: Total lean and fat mass of male and female participants .....	80
Figure 13: Classification of hypertension in male (a) and female (b) participants ....	81
Figure 14: Mean ( $\pm$ standard deviation) total cholesterol of male and female participants.....	82
Figure 15: Total cholesterol classification of male and female participants .....	83

Figure 16: Mean ( $\pm$ standard deviation) total non-fasting blood glucose of male and female participants .....	83
Figure 17: Mean ( $\pm$ standard deviation) total physical activity (MET-minutes/week) of male and female participants .....	85
Figure 18: Classification of physical activity for male and female participants .....	85
Figure 19: Mean ( $\pm$ standard deviation) total, vigorous and moderate activity of male and female participants per day .....	86
Figure 20: Percentage contributions of different domains of activity to total daily physical activity in female and male participants.....	87
Figure 21: Mean ( $\pm$ standard deviation) total energy intake (kJ) of male and female participants.....	88
Figure 22: Dietary composition of (a) males and (b) females.....	89
Figure 23: Mean ( $\pm$ standard deviation) sodium (Na) intake of males and females..	91
Figure 24: Self-reported fast food intake of male and female participants (KFC refers to Kentucky Fried Chicken). .....	92
Figure 25: Percentage of males and females who currently smoke .....	92
Figure 26: Percentage of male and female participants exposed to environmental and tobacco smoke at home and at the workplace .....	93
Figure 27: Mean ( $\pm$ standard deviation) number of alcoholic drinks consumed by male and female participants .....	94
Figure 28: Percentage of males and females classified as 'risky drinkers'.....	95
Figure 29: Reported versus actual cholesterol classification of (a) male and (b) female participants .....	96
Figure 30: self reported versus actual measures of type II diabetes in (a) males and (b) females .....	97
Figure 31: Reported versus actual classification of hypertension in (a) males and (b) females.....	98
Figure 32: Perceived and actual body size of female (a) and male (b) participants .	99

Figure 33: Body images by sex (adapted from Ziebland <i>et al.</i> , 2002): illustrations for body shapes used for women A) and men B). .....	100
Figure 34: BMI classification of urban black males and females: comparison between the 1998 and 2003 DHS and current data.....	106
Figure 35: Contributions of different domains of exercise to total physical activity: comparison of current study trends with 2003 national statistic for urban black individuals (DHS, 2003).....	123
Figure 36: Smoking prevalence in black urban males and females, comparison of national statistics and current findings (1998-2010) .....	130
Figure 37: Perceived and actual prevalence of overweight and obesity in black urban males and females. Comparison of national data from 1998 and 2003, and current Eastern Cape data (from the present study). .....	136
Figure 38: Combined risks in male and female participants (risks considered included obesity, hypertension, type II diabetes and hypercholesterolemia).....	142

## LIST OF TABLES

Table I: Age-standardised death rates (per 100 000) across the different population groups of South Africa, 2000 (Adapted from Bradshaw <i>et al.</i> , 2006 and Steyn <i>et al.</i> , 2006) .....	15
Table II: Dependent and independent variables of interest .....	47
Table III: STEPS framework for the assessment of selected CVD risk factors including self-reports, objective measures & blood samples .....	49
Table IV: BMI classification of overweight and obesity (WHO 1995, 2000, 2004) ....	50
Table V: Waist circumference guidelines (WHO, 2000) (James <i>et al.</i> , 2001; James, 2004).....	51
Table VI: Hypertension classification (Adapted from JNC-7, Chobanian <i>et al.</i> , 2003) .....	53
Table VII: Total cholesterol guidelines for the classification of hypercholesterolemia and associated risk. (Adapted from NCEP).....	55
Table VIII: Domain-specific MET classification (GPAQ) .....	68
Table IX: Classification of levels of physical activity (adapted from IPAQ classification of physical activity) .....	68
Table X: “Cut-off points” for the classification of self reported physical activity data (DHS, 2003) .....	69
Table XI: General background characteristics of male and female participants .....	75
Table XII: Mean ( $\pm$ standard deviation) anthropometric and morphological characteristics of participants .....	76
Table XIII: Mean ( $\pm$ standard deviation) cardiovascular (CV) measures of participants .....	80
Table XIV: Self reported lifestyle-related risks.....	84
Table XV: Selected dietary components (mean $\pm$ standard deviation) consumed by males and females .....	89

Table XVI: Mean ( $\pm$ standard deviation) number of drinks consumed by males and females on a weekly basis .....	95
Table XVII: Comparison of morphological data from 1998-2010 for urban black males and females within South Africa.....	105
Table XVIII: Obesity prevalence across different provinces of South Africa within urban black males and females (1991-2010) .....	107
Table XIX: Blood pressure of urban black individuals (35-54) within different provinces of South Africa (1991-2010).....	115
Table XX: Mean total cholesterol (mmol.L <sup>-1</sup> ) of urban black individuals (35-54 years) within different provinces of South Africa, 1992-2010. ....	118
Table XXI: Dietary composition of black urban males and females from various provinces of South Africa (1994-2010).....	128

# CHAPTER I

## INTRODUCTION

### BACKGROUND

South Africa is comprised of a vast mix of people and cultures, ranging from those at the top end of the socio-economic scale, to those who live below the poverty line and who make up the majority of the population (Steyn *et al.*, 2006). As a result, South Africans face a broad spectrum of diseases: non-communicable cardiovascular diseases, historically associated with first world countries and affecting largely the wealthy upper class, as well as those associated with poverty, including communicable and infectious diseases and violence-related trauma (Bradshaw & Steyn, 2001; Steyn *et al.*, 2000). In addition, South Africans also face the burden of the HIV/AIDS virus, which accounts for the leading cause of mortality in the country (Booyesen, 2003). Thus, unique to the South African setting, are the combined risks of communicable diseases, HIV/AIDS, infectious diseases, and violence-related injuries and trauma, all of which form the 'quadruple burden' of disease (Bradshaw & Steyn, 2001; Bradshaw *et al.*, 2003). Research has shown that the heaviest burden of disease falls on poor communities in urban areas (Mayosi *et al.*, 2009). Since three-quarters of the South African population is comprised of black Africans (Mayosi *et al.*, 2009), and these individuals thus form the majority of those living in impoverished urban areas, it is black males and females who bear the brunt of the 'quadruple burden' within the country (Bourne *et al.*, 2002; Mayosi *et al.*, 2009), and who were the focus of this research.

In addition to this 'quadruple burden' of disease, within different population groups, a variety of specific disease risks exists. From a historical perspective, cardiovascular disease (CVD) predominantly affects white urban males – those exposed to, and living, sedentary lifestyles and consuming diets rich in fats (Rossouw *et al.*, 1983; Seftel, 1978). Indeed, research has shown white men to be at the top end of the scale in terms of obesity and hypercholesterolemia, in comparison to other South African population groups (Seftel, 1978). In contrast to this, black males and females have amongst the lowest cardiovascular disease risk, despite black females having a predisposition towards being overweight and obese (Rush *et al.*, 2007; Bourne *et al.*, 2002). National statistics demonstrate a prevalence of obesity in 59% of black women, in comparison to 54% and 52% in coloured and white women respectively (Rush *et al.*, 2007; Punyadeera *et al.*, 2001). Literature has linked this to

the consumption of diets high in carbohydrates, and relatively low levels of physical activity in relation to energy consumed (Schutte *et al.* 2006; Bourne *et al.* 2002; Sparling *et al.*, 1994). In addition, cultural beliefs (particularly among the rural Xhosa women) command a very different view of being overweight and obese – namely that women of this disposition are seen to represent good health, prosperity and lack of HIV infection (Puoane *et al.*, 2002). By contrast only 8% of black men are obese, as opposed to 3-9% and 6-9% of indian and coloured men (van der Merwe & Pepper, 2006). Aligned with these notable differences in disease risk, diagnosis and knowledge of individual health status are often limited, and thus perceived and self reported health risks and knowledge thereof tend to be very different to measured values, ignorance which, to date, has not been fully explored.

To date the only Eastern Cape statistics that exist with regards to cardiovascular disease risk, are those gained from two large-scale demographic and health surveys, which were carried out in 1998 and 2003. While research has been undertaken in the Northern Province, the Western Cape and Gauteng (Senekal *et al.*, 2003; Kruger *et al.*, 2002; Vorster *et al.*, 2000; Venter *et al.*, 2000; Steyn *et al.*, 1998; Steyn *et al.*, 1992) these demographic and health surveys were the first to include the Eastern Cape. They served to create an initial data set of the health status of South Africans at a national level, encompassing aspects of poverty and socio-demographics to explain different disease and injury risks across the country, as well as encompassing perceptions and self reporting of various CVD risk factors. However, due to the large-scale nature of the surveys, they were for the most part questionnaire-based and only very basic cardiovascular measures of body mass index (BMI), waist to hip ratio (WHR), and blood pressure were taken. No other physical measurements, such as physical measures of blood glucose or cholesterol (both important cardiovascular risk factors) were included. Therefore, while the information gained was extremely beneficial in terms of creating an initial dataset for the Eastern Cape, the data obtained relied for the most part on self reporting of cardiovascular and health risks. Furthermore, not all areas of the Eastern Cape were included within the survey, and therefore there remain some regions within the province, including the Grahamstown area, where data on cardiovascular disease risk is non-existent.

While the majority of the province live below the poverty line and face poverty related infections and HIV/AIDS, the Eastern Cape also presents high mortality rates from non-



communicable diseases: Cardiovascular disease-related mortalities within the province have in fact been shown to be *higher* than the *national* prevalence rate (Bradshaw *et al.*, 2000).

## STATEMENT OF THE PROBLEM

The cardiovascular disease risk of individuals residing in the Eastern Cape has to date not been fully elucidated. While broad statistics of the province as a whole exist, there remains limited knowledge of risks within all areas of the province, including the Grahamstown area, for which there are currently no data available. Urban black individuals appear to bear the brunt of the 'quadruple burden' of disease due to urbanization and the associated 'nutrition transition' and yet are perhaps ignorant of this disease risk (Bradshaw *et al.*, 2000). Accordingly the aims of this research project were as follows: To assess and compare cardiovascular disease risk, and to compare these findings to perceived/self reported measures, within black males and females comprising an urban working population in Grahamstown, Eastern Cape.

## RESEARCH HYPOTHESES

It was expected that cardiovascular disease risk would be different between male and female participants. More specifically, it was expected that there would be a higher prevalence of cardiovascular risk factors in females compared to male participants. In addition, it was expected that there would be a difference in the reported and perceived prevalence of CVD risks in comparison to actual measures in both samples.

## STATISTICAL HYPOTHESES

1. The first null hypothesis proposed was that there would be no difference in morphological characteristics between the two samples, as defined by a) BMI, b) waist circumference and c) body composition.

$H_0: \mu_{\text{Males MORPH (a,b,c)}} = \mu_{\text{Females MORPH (a,b,c)}}$

$H_a: \mu_{\text{Males MORPH (a,b,c)}} \neq \mu_{\text{Females MORPH (a,b,c)}}$

2. The second null hypothesis proposed was that there would be no difference with regards to cardiovascular risk (CV) characteristics between the two samples, as defined by d) blood pressure, e) total blood cholesterol, and f) blood glucose.

$$H_0: \mu_{\text{Males}}_{CV(d,e,f)} = \mu_{\text{Females}}_{CV(d,e,f)}$$

$$H_a: \mu_{\text{Males}}_{CV(d,e,f)} \neq \mu_{\text{Females}}_{CV(d,e,f)}$$

3. The third null hypothesis was that lifestyle-related habits would be similar between the two samples, as defined by g) dietary intake, h) physical activity, i) tobacco use, j) alcohol consumption, and k) alcohol dependence.

$$H_0: \mu_{\text{Males}}_{LIFESTYLE(g,h,i,j,k)} = \mu_{\text{Females}}_{LIFESTYLE(g,h,i,j,k)}$$

$$H_a: \mu_{\text{Males}}_{LIFESTYLE(g,h,i,j,k)} \neq \mu_{\text{Females}}_{LIFESTYLE(g,h,i,j,k)}$$

4. The fourth null hypothesis was that self reported and perceived measures of risk would be similar to observed measures of risk, as defined by comparisons between observed and actual measures of l) obesity, m) hypertension, n) type II diabetes and o) hypercholesterolemia.

$$H_0: \mu_{\text{Reported}}_{(l,m,n,o)} = \mu_{\text{Actual}}_{(l,m,n,o)}$$

$$H_a: \mu_{\text{Reported}}_{(l,m,n,o)} \neq \mu_{\text{Actual}}_{(l,m,n,o)}$$

## DELIMITATIONS

One hundred and forty-eight urban black females and one hundred and forty-three urban black males participated in this study. Individuals were recruited from various businesses and institutions throughout the greater urban Grahamstown area within the Eastern Cape, South Africa. Various questionnaires were completed by each participant – these were carried out in interview form with trained research assistants. These allowed for self-reported measures of dietary intake (24-hour dietary recall), physical activity (GPAQ and IPAQ), tobacco use (adapted WHO STEP-wise questionnaire), alcohol consumption (adapted WHO questionnaire of monitoring alcohol use) and alcohol dependence (CAGE questionnaire) to be assessed. Three body perception questions were also completed, in addition to questions about self reported prevalence of various risks (hypertension, hypercholesterolemia and Type II diabetes), perceived body size and habitual intake of various food items (these to

complement the 24-hour dietary recall analysis). All questionnaires were carried out in the form of an interview, with a translator present to overcome any language barriers. Stature, mass and waist circumference were also measured and recorded as well as body composition, which was assessed via Bioelectrical Impedance Analysis (BIA). Thereafter measures of blood pressure, blood cholesterol and blood glucose were taken.

## **LIMITATIONS**

Variations limitations of this research project reduce the accuracy and applicability of data obtained. As the questionnaires used in this research were largely qualitative in nature and therefore, for the most part, dependent on the honesty of the participants, there were certain parameters which could have affected the research outcomes. In addition to this, it is acknowledged that some of the measurement techniques used were limited in their accuracy and reliability. The following factors were taken into consideration:

Due to time and financial constraints, testing a large number of individuals was not possible. The sample size in the current study was relatively small, and was therefore not encompassing and entirely representative of the Makana (Eastern Cape) population as a whole. Conclusions drawn from this sample specifically may therefore not be indicative of trends for the whole community or province. However, power analyses revealed that this sample size would be sufficient to yield statistically sound results reflective of the population.

The use of questionnaires and self reporting of health risks required participants to answer as honestly as possible. However, the honesty of subjects was something that could not be controlled. Despite this limitation, it is accepted that physical activity questionnaires and survey instruments remain the most practical when assessing large samples in epidemiological studies (Richardson *et al.*, 2001).

It is accepted that the 24 hour dietary recall is not the most accurate dietary assessment tool – however due to time, financial and logistical constraints, the use of more accurate methods was not possible. In an attempt to overcome this, additional dietary questions regarding general dietary habits were included. These provided more detailed information as regards habitual dietary intake as well as dietary habits – data which was not captured within the 24-hour recall.

Language barriers, due to the researcher being an English mother tongue speaker and the majority of participants being Xhosa mother tongue speakers, may have hindered the accuracy of the results obtained, and may have resulted in some details being omitted or falsely interpreted. However, use was made of a Xhosa-speaking interpreter, who was present and assisted during all testing sessions. Questionnaires were also translated into Xhosa in an attempt to overcome language barriers.

It is acknowledged that the 'pin-prick' method is not the most accurate with regards to blood cholesterol and glucose assessment. However, due to financial constraints, full blood samples were not possible. Reflectance photometers provide a less accurate means of assessment, but are practical when testing large numbers of individuals. Therefore use was made of them. Care was taken to ensure that measures were taken as accurately as possible, and given the lack of any existing data, it was agreed that these measures would suffice.

Diet and physical activity were not absolutely controlled for, prior to Bioelectrical Impedance Analysis (BIA) or blood glucose and cholesterol testing. This may have affected the accuracy of the results obtained. However, in all cases, all activity and food consumed over the previous 24 hours was recorded to account for possible effects. In addition, while fasting measures were not possible, individuals were tested at least two hours after food consumption, and a recognized conversion factor was used when classifying glucose readings to account for this lack of control with regards to diet.

Since no research has assessed CVD risk in this area prior to this research project, it is proposed that data acquired and analysed will prove useful in cross-provincial comparisons, despite the limitations to the methods.

## CHAPTER II

### REVIEW OF LITERATURE

#### INTRODUCTION: CARDIOVASCULAR DISEASE GLOBALLY

In 1998 it was estimated that approximately 31% of global mortality was attributable to cardiovascular disease (CVD) (Yusuf *et al.*, 2001a). More recent literature suggests that CVD and chronic diseases of lifestyle now represent the *leading* cause of mortality in *westernised* countries, a finding which is expected to be mirrored *globally* over the next 15-20 years (Marijon *et al.*, 2007; Poulter, 1999). Aligned with this, between 2006 and 2012, deaths from non-communicable diseases (half of which will be due to CVD) are expected to increase by 17%, accounting for 37% of all deaths globally by 2020. As the global epidemic of CVD increases, it has been suggested that the 'social gradient' of individuals affected has *reversed* such that impoverished and more vulnerable population groups are now falling victim to this epidemic (Yusuf *et al.*, 2001a). As a result, increasingly the burden of CVD is being borne by developing countries, such as South Africa (Lim *et al.*, 2007): while currently it is estimated that almost half the disease burden in low- and middle-income countries, is *already* due to non-communicable chronic disease, by 2020 it is expected that mortality from ischemic heart disease (IHD) will increase by 137% in men and 120% in women (Ghoziladeh & Davidson, 2008; Yach *et al.*, 2004). In light of this, CVD was put on the global public health agenda in 2000 (Prentice, 2006; Chopra *et al.*, 2002).

#### South Africa

The population of South Africa, at approximately 46 million people, comprises a vast mix of cultures and ethnicities. With 11 official languages, and various additional dialects, the population is divided up roughly as follows: 77-79% black/African, 9% coloured (individuals of mixed ancestry), 2.5% asian/indian and 10-11% white (Steyn *et al.*, 2006; Faber & Kruger, 2005).

The political history of South Africa has to a large extent governed the *geographic composition* of the country: While previously divided up into four provinces, South Africa is now made up of *nine* provinces (Figure 1), which are host to a variety of different social and

living conditions – including wealthy and middle-income suburban areas, semi-impooverished peri-urban areas (largely made up of informal settlements), farms and under-developed rural areas (Steyn *et al.*, 2006). These areas differ according to demographic location as well as cultural influences and living standards.

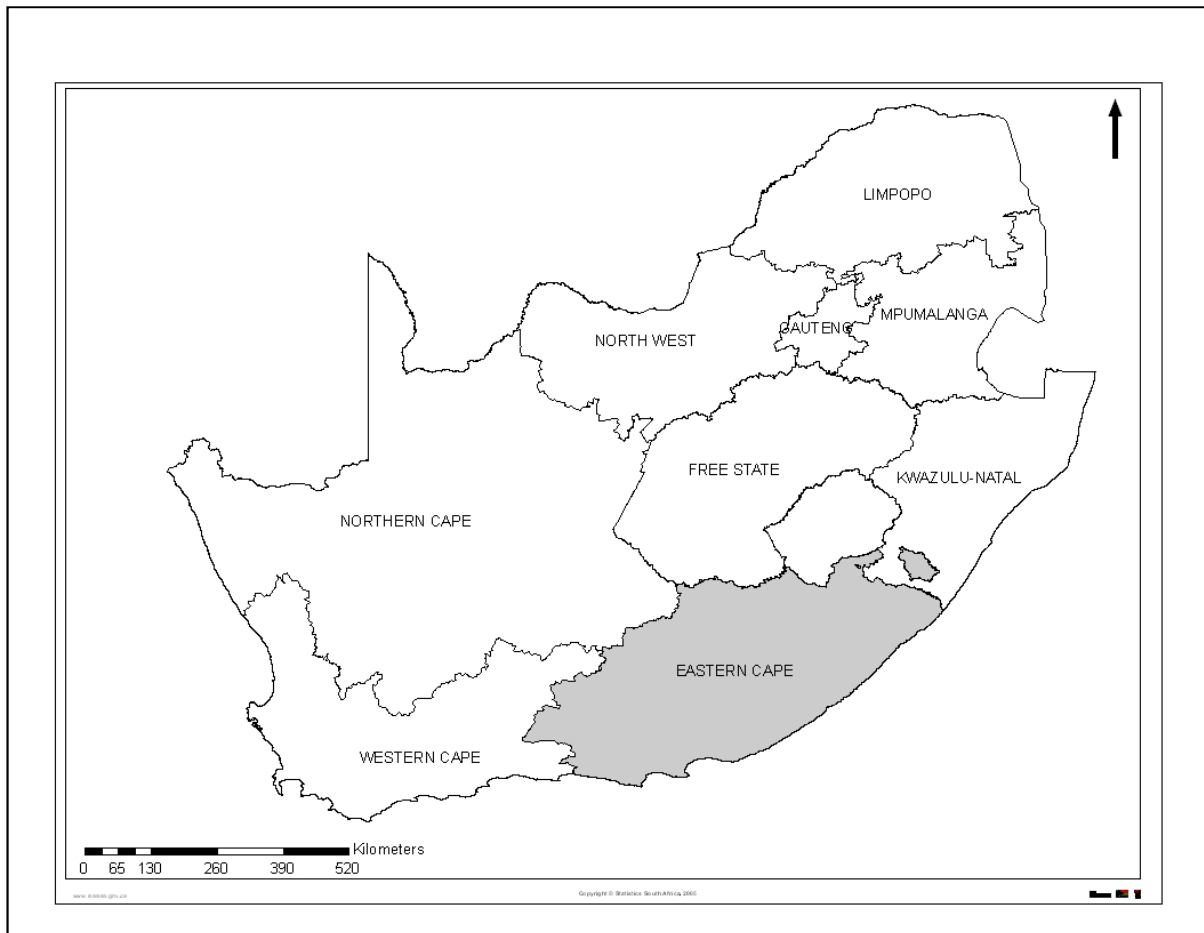


Figure 1: Map of South Africa showing the nine different provinces (shaded area represents the Eastern Cape) (sourced from Stats SA, 2006)

A large portion of the country's population resides in rural areas: on farms and in rural villages. Within these settings individuals live off the land and thus lead traditional lifestyles typical of rural environments. Juxtaposed with these areas, are urban cities in which infrastructure is more advanced, and lifestyles tend to be more westernised and sedentary. Since South Africa is a developing country, cities are also typified by urban slums and 'townships', which are home for many lower-income impoverished individuals (Steyn & Schneider, 2001). These areas are characterised by poor living conditions, inadequate access to clean water and sanitation, and urban squalor and violence. Indeed, it is within

these areas that many previously rural individuals reside –those who have moved off farms and rural villages in search of work and opportunities not afforded to them in the past due to historical prejudices in the country.

## THE EASTERN CAPE

In line with national changes in the geographic division of the country as a whole, the division of the Eastern Cape has also been shaped by the political history of the country. Prior to the election of a democratic government in 1994, what is now the Eastern Cape province consisted of two 'national states', Transkei and Ciskei, and an area which fell under the jurisdiction of the Cape Province (Bradshaw *et al.*, 2000) (Figure 2). Now a single province, the Eastern Cape comprises an area of 169 580 km<sup>2</sup>, making it the second largest province in the country in terms of surface area, and representing 14% of South Africa's surface area (Stats SA, 2006; Stats SA, 2003). After Gauteng and Kwa-Zulu Natal, it is the third most populated province in the country, home to 14.4% of the total population of the country (Stats SA, 2006). In 2002, about 63% of all individuals within the province lived in *rural* areas. In addition, 68% of the population of the province lived below the poverty line, with 47% living in formal housing, and 11% of individuals living in informal housing and 38% in traditional structures. Furthermore, with an average of 4.1 individuals per house, 31% of houses had no toilet facility and only 28% of households had access to electricity for cooking purposes (Bradshaw *et al.*, 2000). Hence, the Eastern Cape is one of the poorest provinces in the country, and according to the 2001 population census, only 60% of the population are literate (Stats SA, 2006).

The population structure of the Eastern Cape mirrors that of the country as a whole, with the majority of individuals within the province being black/African (87%), followed by coloured (7.4%) and white (4.7%) (Stats SA, 2006). The Xhosa people make up the majority of the province, and aligned with this, isiXhosa is the main language spoken. The Eastern Cape is home to many industries, including a large automotive contingent, dairy, chicory and pineapple farming, and more recently the safari industry, which has seen an increase in foreign revenue from tourism (Bradshaw *et al.*, 2000). The province itself is divided into 38 local and 5 district municipalities. The main economic centres are Port Elizabeth and East London, while Grahamstown represents the largest urban settlement within the Makana local municipality (Stats SA, 2006).



Figure 2: Map of the Eastern Cape, South Africa. Area 1 (small map, highlighted in red) represents the Eastern Cape and Area 2 (big map, highlighted in red) represents the Makana area in which Grahamstown is located. (Sourced from Wikipedia Commons: URL: [commons.wikimedia.org](https://commons.wikimedia.org))

The area of Makana is about 4221km<sup>2</sup> and comprises a population of about 74 500 people. Only half of the Makana population are employed, and poverty is high with 67% of households earning less than R2000 per month. The most common language spoken is isiXhosa (Stats SA, 2006). Grahamstown itself is largely an educational centre, home to Rhodes University and a host of private and government schools. It is also the seat of the high court within the province, and is the main economic centre within the greater Makana area.

## **POLITICAL CHANGES SHAPING CHANGES IN HEALTH**

South Africa has seen much political and demographic change over the last 15 years. With the election of a new democratic government in 1994, many individuals who were previously discriminated against and confined to certain areas were allowed freedom of choice and movement (Talip *et al.*, 2003). The result has been the movement of many previously



disadvantaged black African individuals from rural into urban areas. And hence associated urban 'westernised' lifestyles have been adopted (Talip *et al.*, 2003). Traditional active lifestyles accompanied by high fibre, carbohydrate-rich and low fat diets have been abandoned in favour of urbanized lifestyles typified by sedentary living, and high fat, high sugar, and low fibre diets (Talip *et al.*, 2003; Puoane *et al.*, 2002; Sparling *et al.*, 1994). Associated with this change in lifestyle has been an increase in CVD prevalence within previously disadvantaged population groups, particularly black African individuals (Loock *et al.*, 2006; Talip *et al.*, 2003; Christie, 2001; Vorster *et al.*, 2000), a disease and health risk traditionally associated with first world countries and affecting predominantly the wealthier sectors of society (Tibazarwa *et al.*, 2009). This is in addition to existing malnutrition and the existence of infectious diseases which already pose a serious threat to the health of such population groups (Tibazarwa *et al.*, 2009; Sliwa *et al.*, 2008; Boutayeb, 2006; Yusuf *et al.*, 2001b; Coutsooudis & Coovadia, 2001).

### **What is an 'epidemiological transition'?**

The term 'epidemiological transition' has been broadly described as referring to a series of complex and interrelated changes in health and disease patterns that occur in populations over a long period of time (Popkin, 1998; Frenk *et al.*, 1989), usually related to social, economic and demographic changes which are happening in such populations (Lim *et al.*, 2007; Steyn & Schneider, 2001). Linked with these changes, is a set sequence of events, beginning with the predominance of infectious diseases, and followed by an era in which chronic disease predominates (Steyn & Schneider, 2001; Yusuf *et al.*, 2001a; Popkin, 1998; Frenk *et al.*, 1989). Populations adopting unhealthy lifestyles typified by smoking, alcohol dependence, physical inactivity, and the consumption of westernised diets are increasingly at risk of chronic disease - which tends to predominate in such communities, resulting in high levels of obesity, hypertension, diabetes and hypercholesterolemia (Doak *et al.*, 2005; Steyn & Schneider, 2001).

At any given time, different countries and indeed different regions within a particular country can be at different stages of the epidemiological transition (Yusuf *et al.*, 2001a). Within South Africa specifically, historically CVD and chronic diseases of lifestyle used to be associated with the wealthier sectors of society, those able to afford rich food and live lavish lifestyles. In the last quarter century, however, these conditions have been shown to occur much more

frequently in poor population groups, those undergoing industrialisation, urbanization and development, and thus adopting more westernised lifestyles. It is hence these population groups that have undergone an epidemiological transition (Steyn & Schneider, 2001; Yusuf *et al.*, 2001a; Christie, 2001; Vorster *et al.*, 2000; Popkin, 1998).

South Africa is defined as a middle-income country – one in which the average income is reasonable but concurrently there are high levels of poverty (Steyn & Schneider, 2001). Furthermore, given the variety of living conditions, South Africa fits the protracted-polarised model of epidemiological transition – a model characterized by the *coexistence* of infectious and chronic diseases within one population (Steyn & Schneider, 2001). While partly associated with development and associated changes in health, past prejudices and inequality lie at the base of these issues within South Africa (Gilbert, 1996). At 0.58 South Africa has the second highest Gini-coefficient in the world, highlighting the high level of inequality that exists within the country, and associated with this, the coexistence of poverty-related and westernised disease risks (Steyn & Schneider, 2001; Coutsooudis & Coovadia, 2001).

## **THE QUADRUPLE BURDEN OF DISEASE**

The situation in South Africa is further made unique by the co-existence of violence-related trauma as well as HIV/AIDS, which, in addition to CVD and infectious diseases, serves to create a '*quadruple burden of disease*' (Mayosi *et al.*, 2009). Many population groups, particularly those living below the poverty line, currently face this burden (Steyn *et al.*, 2004; Coutsooudis & Coovadia, 2001). In 2000, a Burden of Disease study was carried out in South Africa, the findings of which highlighted AIDS as the leading cause of mortality, accounting for 30% of all deaths (Bradshaw *et al.*, 2003). This was followed by CVD at 17%. Furthermore HIV/AIDS and CVD accounted for a higher proportion of deaths in females than males (34% vs. 26%; 19% vs. 14%, respectively) (Bradshaw *et al.*, 2003). A follow up study was carried out in 2006, results of which pointed to an increase in mortality from chronic diseases of lifestyle (CDL), while HIV/AIDS accounted for almost 30% of all mortality and represented the single largest cause of years of life lost (Bradshaw *et al.*, 2006). Accompanying the presence of HIV/AIDS and non-communicable diseases, was the co-existence of low birth weight, as well as under- and malnutrition – which simultaneously

topped the scale in terms of leading causes of mortality within the country (Steyn *et al.*, 2006).

Thus there are various attenuating factors which exist within the South African context, and simultaneously contribute to the quadruple burden of disease. Firstly, low socio-economic status and associated living conditions serve to create stress, and related risks: there is strong evidence suggesting that *socio-economic stress* plays a significant role in the development of CVD (Brydon *et al.*, 2006; Todaro *et al.*, 2003; Steptoe *et al.*, 2003). This is highlighted by findings that the primary risk factors for CVD account for only 50% of all overt cases of Coronary Heart Disease (CHD) (Vieweg *et al.*, 1998), and 60% of patients with CVD have been shown to have only one or no major risk factors (Brydon *et al.*, 2000; Hlatky *et al.*, 1995). This finding reflects an increasingly pertinent and serious situation within South Africa, whereby the effects of urbanization mean that more individuals are leading increasingly fast paced, stressful lives (Striegel-Moore *et al.*, 2006). In addition, it is thought that lower status individuals experience greater stress in their lives, based on increased financial strain, poor living conditions and minimal (if any) social support (Steptoe *et al.*, 2003). This is an important consideration in South Africa, based on the fact that individuals of low socio-economic status typify the majority of the population groups within the country (Ndaba & O'Keefe, 1985).

In part, associated with high levels of poverty, is the presence of HIV/AIDS - both of which co-exist in a vicious circle. Shown to be the single highest cause of mortality in South Africa (Booyesen, 2003), the profound effect of the AIDS epidemic can be seen in the significant decline in the life expectancy of South Africans, which dropped from 61.6 years in 1992 to 49.7 years in 2006 (Steyn *et al.* 2006). HIV infection is also associated with a reduction in BMI of  $1.9\text{kg.m}^{-2}$  and in systolic blood pressure of 3 mmHg, in addition to which antiretroviral treatment is associated with an increase in insulin resistance and dyslipidaemia (Mayosi *et al.*, 2009). Not only are impoverished individuals often malnourished, but they do not have access to adequate health care (Charlton & Rose, 2001), and therefore on top of HIV/AIDS infection, other chronic and acute infections develop. Immense strain is thus often placed on older retired individuals within the household earning a minimal pension, who while not only having to take care of Aids orphans, are often physically and emotionally abused for their monthly pension (Booyesen, 2003). Together with the combined risks of CVD and infectious diseases, this places additional strain on households in the face of limited health care

resources (Booyesen, 2003). In South Africa, infectious diseases account for 28% of years of life lost, in comparison to 25% of years lost accounted for by chronic disease (Yach *et al.*, 2004).

With much of the population living under impoverished conditions (40-50% of the population categorized as poor, 25% as ultra-poor) (Steyn *et al.*, 2006), the heaviest burden of disease in fact falls on *poor communities* within *urban areas* – which tend, for the most part, to consist of black urban individuals (Mayosi *et al.*, 2009; Vorster *et al.*, 2000). Aligned with this, a study of age-standardised mortality rates in Cape Town found that mortality rates attributable to non-communicable diseases (including CVD) within the poor sub-district of Khayelitsha were *double* those in the wealthier northern and southern sub-districts. In addition, in relation to other diseases and disease-risks including injuries and HIV/AIDS, the mortality rates of the poor were found to be twice to three times more than those for the rich – a concerning statistic given the fact that 75% of black households and 6.7% of white households in the country live below the poverty line (Mayosi *et al.*, 2009; Gilbert, 1996). Furthermore, given the plethora of diseases, health services are required to cater for a variety of health risks. Since the symptoms of chronic diseases are less immediate, and cannot compete with the more acute and urgent symptoms of infectious diseases, active infections and trauma, resource allocation is not prioritized in favour of CVD, despite increasing levels within population groups already at risk (Steyn & Schneider, 2001).

## **SEX AND RACIAL CONSIDERATIONS**

A particularly important consideration within the South African context, aligned with the quadruple burden of disease which affects the population as a *whole*, is the *variation in risk* which affects different sexes and population groups in different ways. Thus, while national findings highlight a population at risk, *specific risks* affect different population groups in varying degrees. Indeed, the varied population composition of South Africa allows for research examining the relationship between lifestyle and CVD in light of very different cultural and lifestyle disease profiles (Bradshaw *et al.*, 2006). In 2006, Bradshaw *et al.* reported on findings from the 2000 National Burden of Disease study. Herein, they highlighted the very different risk profiles across the different population groups of the country: *overall mortality* was reported to be *highest* in *black* individuals (nearly double the figure reported for whites), while mortality from Chronic Diseases of Lifestyle (CDL) was

slightly higher in indian and coloured individuals. Indians in particular, demonstrated the highest mortality from *CVD*, and also presented high mortality rates associated with diabetes, IHD and strokes (Bradshaw *et al.*, 2006) (Table I). In contrast to this, research has shown that blacks tend to present with high mortality rates associated with *hypertensive heart disease*, demonstrating the highest rates compared to other population groups (Bradshaw *et al.*, 2006) (Table I). Furthermore, mortality from diabetes is shown to be higher in *females*, while *males* present with higher mortality associated with IHD and hypertensive heart disease (Bradshaw *et al.*, 2006).

Table I: Age-standardised death rates (per 100 000) across the different population groups of South Africa, 2000 (Adapted from Bradshaw *et al.*, 2006 and Steyn *et al.*, 2006)

	BLACKS	WHITES	COLOUREDS	INDIANS	SOUTH AFRICA
<b>ALL CAUSES</b>	1613	937	1304	1172	1468
<b>CVD</b>	375	384	406	607	361
<b>DIABETES</b>	59	23	64	111	49
<b>IHD</b>	70	230	171	392	123
<b>STROKES</b>	143	72	139	392	124
<b>HHD</b>	88	10	37	29	68
<b>OTHER CDL</b>	116	91	82	96	108
<b>Total CDL</b>	769	767	876	1000	750

Where: CVD represents cardiovascular disease; IHD Ischaemic heart disease and HHD Hypertensive heart disease. Values in italics represent highest death rates for each risk within different population groups

In comparison to mortality trends from the 1940s, notable increases in CVD risks are evident – as seen in the high mortality rates from IHD and strokes, as well as hypertensive heart disease and diabetes (Steyn *et al.*, 2006). These findings highlight both a population in transition, and the *differing risk profiles* according to both *race* and *sex* - representative of different groups of individuals who are at different stages of the health transition – with indians and whites being at the later stages and blacks at the earlier stages of the transition according to the disease risks they currently face.

## PROVINCIAL CONSIDERATIONS

Due to the cultural and socio-demographic diversity in South Africa, mortality estimates also differ across the different provinces of the country – these tending to differ according to the level of development and wealth of the particular provinces: the Western Cape and Gauteng, being the two wealthiest provinces, are reported to have higher mortality rates associated with cancer and IHD (Steyn *et al.*, 2006), while the poorer provinces such as the Northern and *Eastern Cape*, reportedly have higher mortality rates from stroke and respiratory diseases, generally associated with poverty and infectious diseases such as Tuberculosis (TB) (Bradshaw *et al.*, 2006).

## THE EASTERN CAPE

The *Eastern Cape* specifically has been shown to have amongst the *lowest IHD mortality*, and amongst the *highest mortality* rates associated with *chronic bronchitis* (Bradshaw *et al.*, 2006). HIV/AIDS is the leading cause of mortality (20% of all deaths in men and women), although this is lower than the national prevalence rate of 30%. This is followed by CVD at 17%. Furthermore it has been shown that HIV/AIDS, CVD, respiratory infections and diabetes account for more deaths in *females* compared to males (Bradshaw *et al.*, 2000). Non-communicable disease accounts for 43% of mortality within the province, indeed constituting a higher proportion of mortality than the national average of 38%. Similarly, tuberculosis (TB), diarrhoeal diseases and other diseases associated with poverty and under-development constitute a higher proportion of mortality (38%) in comparison to national findings (27%) (Bradshaw *et al.*, 2006). Therefore, as with racial and sex differences in mortality rates, provincial data also point to different provinces being at different stages of the health transition – with the more developed provinces being at the later stages of the transition and the poorer provinces embedded within the earlier stages of the transition (Steyn *et al.*, 2006; Bradshaw *et al.*, 2006).

While mortality estimates for the Eastern Cape exist, there are limited data on the disease risks which the population of the province currently faces, in particular, for CVD. While the 1998 and 2003 Demographic and Health Surveys (DHS) included the Eastern Cape, data were only presented on the province as a whole. Thus, very little is known about *sex- and racial-specific risks*. Comparisons therefore can only be made with *national statistics* and

*other provincial findings*, since those presented for the Eastern Cape encompass of all population groups, including both children and older individuals, and lack any specific information on black urban individuals. Broad CVD risk factor categories are therefore discussed *generally*, and where possible, references are made to the Eastern Cape specifically.

## **NON-MODIFIABLE RISKS FOR CVD**

### **Age, genetics, sex**

Research has illustrated that there are genetic, sex and age factors which influence the development and onset of various CVD risks (Bray & Champagne, 2005). Firstly, it has demonstrated that the development of risk factors for chronic disease tends to increase with age (Fodor & Tzerovska, 2004). In particular, men over 45 and women over 55 years of age have been shown to be at higher risk (Bray & Champagne, 2005; Fodor & Tzerovska, 2004), not only for CVD, but indeed for myocardial infarction and strokes, both of which increase exponentially with age (Bray & Champagne, 2005). In females specifically, although there is a paucity of literature to date, research has shown that the risk of CVD increases significantly after menopause (Hu *et al.*, 1999). This is thought to be related to a reduced amount of estrogen in the body, which serves to promote atherosclerosis (Hu *et al.*, 1999). Menopause is associated with changes in body composition and an increased android fat distribution, which alters an individual's lipid profile (Tremollieres *et al.*, 1999). Overall, the effect of age on the development of biological risk factors is largely thought to be influenced by biological maturation as well as changes in lifestyle which accompany the aging process (Twisk *et al.*, 2001). As individuals age, there is a tendency to become less active and increasingly sedentary. The consequences thereof are increased body mass, changes in body composition and, in turn, the development of various associated risk factors (Bray & Champagne, 2005; Twisk *et al.*, 2001), including hypertension, diabetes (Abbott *et al.*, 2002), as well as obesity and hypercholesterolemia (Twisk *et al.*, 2001).

In addition to age, research has highlighted that certain CVD risk factors have a genetic predisposition (Ellsworth *et al.*, 1999), in particular, hypertension and hypercholesterolemia which often occur regardless of diet or other lifestyle factors (Fodor & Tzerovska, 2004). While only a few studies have looked at any genetic impacts on risks for CVD within the

South African context, interestingly those that have been carried out have found similar trends, particularly amongst black individuals (Vorster *et al.*, 2005). Studies have shown that various hormones, levels of which appear to be higher in black men and women than in other population groups, are evidenced to exert a protective effect (Vorster *et al.*, 2005): The THUSA (Transition and Health during the Urbanisation of South Africans) study in the North West province of the country demonstrated that black individuals had lower levels of homocysteine. It is traditionally accepted that increased homocysteine within the body increases an individual's risk for CVD (Vorster *et al.*, 2005). In accordance with this, the study concluded that the low levels of homocysteine, most likely genetically determined, offered these individuals a protective effect against CVD risk (Vorster *et al.*, 2005). Furthermore, studies have also shown that lower plasma fibrinogen levels in African populations in general may also exist as a protective factor – demonstrating that a cluster of protective factors may, in part, explain the reduced prevalence of CVD seemingly evident in this population group (Cappuccio, 1997).

While women share similar risk factors to men, some risk factors play greater or lesser individual roles in terms of overall risk profile. High blood triglycerides, for example, as well as low high-density lipoprotein cholesterol (HDL) increase CVD risk to a greater extent in females, in addition to smoking which has been shown to place women at relatively higher risk for CVD in comparison to their male counterparts (Fodor & Tzerovska, 2004; Ghazizadeh & Davidson, 2008). Given these statistics it is concerning that women, in general, are largely under-represented in studies related to chronic diseases of lifestyle (Ghazizadeh & Davidson, 2008).

Looking to South Africa specifically, variety and difference in the prevalence of risk factors amongst different sexes and races has been demonstrated (Kalk & Joffe, 2007). Historically, chronic disease was highest amongst *white males*, who were also shown to be at the top end of the scale in terms of obesity and hypercholesterolemia (Rossouw *et al.*, 1983). This was thought to be largely related to lifestyle, with many white males traditionally leading sedentary lifestyles and consuming diets high in fats (Kalk & Joffe, 2007). In contrast to this, hypertension and smoking are thought to pose the greatest threat to *black* South Africans (Seftel, 1978). Indeed hypertension is proposed to be the main predictor of CVD in black populations, specifically men (Cappuccio, 1997). Obesity, on the other hand, has been shown to be much more prevalent in females, specifically *black females* (Seftel, 1978). In



fact, obesity represents the single most common and important CVD risk factor amongst black South African women, something which has been linked largely to over-nutrition (Bourne *et al.* 2002). The significantly lower rate of obesity in black males is thought to be related to their involvement in manual labour, thus resulting in notably higher energy expenditure levels (Sparling *et al.*, 1994).

In terms of worldwide statistics, males are at increased risk for CVD, yet it is important to note that huge variation exists between different races and population groups, and it is vital to acknowledge the fact that despite the known sex differences in CVD risk, the correlation of three or more risk factors predisposes all individuals to significantly greater risk of disease, regardless of sex (Fodor & Tzerovska, 2004).

## **MODIFIABLE RISKS FOR CVD**

As one acknowledges the varying risk profiles within South Africa, illustrative of a very diverse population group undergoing a health transition, it is important to consider the contributions of different risk factors, specifically relating to black males and females of the Eastern Cape, and to compare the findings with those in other provinces and South Africa as a whole. It must be noted throughout, that while discussion is centered on black urban individuals, the only data available for the Eastern Cape represent the province as a whole, and do not involve sex or race. Since both the latter are important aspects of the current study, the extent to which comparisons can be made of the province, is limited.

## **MORPHOLOGICAL RISK**

### **OBESITY**

Obesity can be described as an energy imbalance such that energy consumed and stored exceeds energy expended (Livingston & Black, 2003; Nicklas *et al.*, 2003). It is defined as a chronic, relapsing, neurochemical disease and, due to its association with excess body fat, is a strong independent risk factor for CVD (James, 2004; McArdle *et al.*, 2001; Rippe *et al.*, 1998). Linked primarily to poor diet and physical inactivity, it is associated with, and strongly related to, other major cardiovascular risks including hypertension, hypercholesterolemia and type II diabetes (Henderson, 2005; Mciza *et al.*, 2005; Chopra *et al.*, 2002). The etiology of obesity is multifactorial, with a combination of genetic, metabolic, environmental and

behavioural factors contributing to the development and progression of the disease (Bray & Champagne, 2005; Rippe *et al.*, 1998). Research has demonstrated that while 75% of obesity is attributable to lifestyle factors, 25-35% is attributable to genetic factors (Walker *et al.*, 2001; Rippe *et al.*, 1998).

While past research has shown obesity (particularly abdominal) to be highest amongst white males (Bourne *et al.*, 2002; Vorster, 2002), present statistics reveal a changing trend, such that black females now present with amongst the highest obesity levels in the country (Tibazarwa *et al.*, 2009; Malhotra *et al.*, 2008; Puoane *et al.*, 2002). Country wide statistics on overweight and obesity were presented in the first DHS carried out in 1998. These indicated that 29% of males and 56% of females in South Africa were overweight – these figures largely comprised urban individuals. Furthermore, the *highest rates* of obesity were found in white males and *black females*, with *black males* and asian females presenting with the *lowest rates*. Within the *Eastern Cape* specifically it was highlighted that 20.5% of males and 25.7% of females were overweight; and a further 10% of males and 29.7% of females were obese. Additionally, 39.6% of females presented with a waist circumference (WC) greater than 880mm. In the follow up survey carried out in 2003, in addition to a national increase in the prevalence of obesity in black women, notable increases were evident in the Eastern Cape in comparison to 1998 data, with the percentage of obese women increasing by 2.2% (DHS, 2003). With 31.9% of females being classified as obese, the Eastern Cape presented with the *highest obesity figures* out of all the other provinces of the country. These findings are encompassing of all racial groups, and therefore conclusions about black males and females specifically cannot be drawn, yet these findings do highlight the high prevalence of obesity within females – and since the majority of the province is comprised of black individuals, it is likely that many of the females making up this statistic would have been black.

Looking back to studies from the 1990s until the present, a notable prevalence of obesity can be seen in black females, in comparison to their male counterparts – a trend which has been evidenced country-wide. In 1991, Steyn *et al.* in a study of the black population of the Cape Peninsula, found that 44.4% of the females had a BMI  $\geq 30\text{kg.m}^{-2}$ , and were therefore classified as obese. This is in contrast to findings from the males, who demonstrated a prevalence of only 4%. These findings have been confirmed by various other studies: Seedat *et al.* (1992) illustrated obesity prevalence in 22.6% of women versus 3.7% of men,

while Rossouw *et al.* (1983) highlighted the occurrence of obesity in 31.7% of women over 54 years. Mollentze *et al.* (1995), in a study carried out in the Free State, went on to propose that raised obesity levels amongst black women contributed to higher levels of hypertension evident in their sample – which was consistent with the findings of Seedat *et al.* in 1992. Some years later, Bourne colleagues demonstrated a prevalence of 53.1% and 23% in women and men respectively (Bourne *et al.*, 2002), while in 2005, Alberts *et al.* demonstrated a prevalence of obesity in 29% of black women. Indeed, 50% of these women demonstrated waist to hip ratios *above* normative cut off points for overweight and obesity – suggesting a high prevalence of central obesity, shown to predispose these individuals to an even greater extent to hypertension, diabetes, strokes and CHD (Alberts *et al.*, 2005)

The most recent statistics for South Africa as a whole suggest that the current prevalence of overweight and obesity is 57% for women and 29% for men (Evans *et al.*, 2007). In addition to this, studies have found that central obesity (measured by a waist circumference of >880mm) is now evident in 43.4% of black women throughout South Africa (Evans *et al.*, 2007), with 60% of black women in South Africa diagnosed as obese - 5 times the prevalence rate seen in men (Case & Menendez, 2009; Steyn *et al.*, 2000). This high prevalence rate has largely been attributed to a shift in the calorie-intake-expenditure balance, whereby urbanization has led to a change in lifestyle and dietary habits typified by low physical activity levels, and the increased consumption of cheap high caloric fast foods (Case & Menendez, 2009). Indeed, the latter trend represents a shift away from the traditional high fibre diets accompanied by higher physical activity levels (Case & Menendez, 2009). As a result, urban black women are regarded as the ‘most at risk’ for the development of obesity related consequences and disorders (Dugas *et al.*, 2009a).

### **“Healthy Obese” and other considerations specific to black South African women**

Given the fact much of the literature within South Africa has focused on the rising levels of obesity, particularly amongst black women, various attenuating factors have been identified, which serve to make combating this problem more interesting and challenging, particularly given the wide range of cultural, social, and biological factors which come into play. Perhaps of foremost importance when assessing the health implications of obesity within this population group, is the finding that black women have not appeared to demonstrate many, if any, additional risk factors for CVD (Walker *et al.*, 2001). Although the literature clearly

illustrates abnormally high levels of obesity within this population group, and highlights the potential associated health risks, some studies have not found any associated health risks in black females (Weinsier *et al.*, 2001). Hypertension, hypercholesterolemia and type II diabetes - all traditionally shown to increase with increased obesity levels - have been shown to remain low – and thus black women have been referred to as “healthy obese”. In a study carried out among rural black women in Kwa-Zulu-Natal, 40% were found to be overweight, and 31.6% obese (Walker *et al.*, 2001). Yet even with these findings, these women did not present with any additional risk factors for CVD: hypertension was low, present in only 13.7% of subjects, and therefore the authors concluded the following:

“given the low occurrence of hypertension and of other degenerative diseases, such as diabetes and heart disease, it can be speculated that this group of rural Zulu women, with their traditional diet and near-traditional lifestyle, represent an example of ‘healthy’ obese”. (Walker *et al.*, 2001).

A similar study carried out in Soweto (Johannesburg) showed that while obesity was by far the most prevalent risk factor, in 55% of black women (versus 23% of black men), those regarded as obese were not adversely affected by hypertension, hypercholesterolemia or hyperglycaemia (Weinsier *et al.*, 2001). In Cape Town, while the prevalence of obesity was significantly higher in black women, prevalence of hypertension amongst both men and women was virtually the same (Weinsier *et al.*, 2001; Punyadeera *et al.*, 2001a). And finally, in a study carried out in Durban, while obesity was significantly higher in women than men (22.6% vs. 3.7%), the prevalence of hypertension was similar across both groups, and type II diabetes was actually lower in women, despite the presence of obesity in a large proportion of the sample (Walker *et al.*, 2001; Weinsier *et al.*, 2001; Punyadeera *et al.*, 2001a). Despite these findings, more recent studies have highlighted a changing trend, and have found associations between obesity and other CVD risks, thus refuting the concept of ‘healthy obese’ and highlighting an increased risk for CVD within this population group (Malhotra *et al.*, 2008). Thus the situation among black South African women appears to be approaching that which exists in western cultures, in particular African-American women whose risk profile in the presence of obesity has been shown to increase dramatically (Walker *et al.*, 2001).

## CARDIOVASCULAR RISKS

### HYPERTENSION

Hypertension is defined as the pathological elevation of blood pressure to a level  $\geq 140/90$  mmHg, and is a major risk factor for the development of various cardiovascular and renal diseases (Galobardes *et al.*, 2003; Sankaranarayan, 1999). Blood pressure is affected by various functional and structural characteristics of the cardiovascular, endocrine, nervous and renal systems, as well as by inherited genetic variation (Galobardes *et al.*, 2003). More importantly, lifestyle factors including increased body weight, central obesity, the excessive consumption of alcohol, and insufficient physical activity - further promote and facilitate the development thereof (Sankaranarayanan, 1999).

Hypertension contributes to mortality in the form of strokes, heart attacks and kidney disease or failure (DHS, 1998). It is claimed that within middle- and low-income countries, hypertension is one of the leading causes of diseases, accounting for approximately 50% of strokes globally (Norman *et al.*, 2007; Norman *et al.*, 2006). Interestingly, research globally has shown that hypertension is 3-4 times more prevalent in individuals of African than those of white ancestry – a finding thought to be linked to the high incidence of strokes within this population group (Cappuccio, 1997).

It is estimated that over six million South Africans suffer from hypertension (Maseko *et al.*, 2006). In addition, in a burden of disease study carried out in 2000, it was estimated that 47000 deaths in South Africa were attributed to hypertension, representing the second leading risk factor in terms of deaths after sexually transmitted diseases from unsafe sex (Norman *et al.*, 2007). According to the 1998 DHS, 26% of males and 51% of females in South Africa were diagnosed as hypertensive. In terms of race and sex specifics, prevalence in males was shown to be highest in whites and coloureds, while prevalence was *lowest* in *non-urban blacks*. With females, whites and coloureds also presented with the highest prevalence rates, but the lowest was amongst asians. Although there were no race and sex specific statistics presented for the *Eastern Cape*, overall it was shown that *men* presented with the *3<sup>rd</sup> highest prevalence of hypertension in the country* (14%), while *females* of the Eastern Cape only had the *5<sup>th</sup> highest prevalence* (16%) out of the nine provinces.

Looking at country-wide hypertension data, despite hypertension being largely non-existent within black population groups in the 1940s and remaining lower in this group compared to other racial groups (Tibazarwa *et al.*, 2009), at present hypertension represents the *single most prevalent CVD risk factor in rural and urban adult black South Africans* (Marijon *et al.*, 2007). With a national prevalence rate of 21%, hypertension is thought to represent the main and most important predictor for the development of CVD within these individuals (Bourne *et al.*, 2002; Cappuccio, 1997; Molletze *et al.*, 1995). This has been validated by various provincial studies across the country: prevalence of hypertension in the adult Zulu population in Kwa-Zulu Natal was demonstrated at 25%, while over 14% of the population of the Cape Peninsula have been shown to be at increased risk of CHD due to moderately or highly elevated blood pressure (Seedat *et al.*, 1992; Steyn *et al.*, 1991). The 'Heart of Soweto' Study found that 33% of urban subjects were hypertensive, while the INTERHEART Africa studies reported hypertension to be the most important risk factor for abdominal obesity as well as myocardial infarction (Marijon *et al.*, 2007). Further research has identified hypertension as being an especially common risk factor for stroke in urban black South Africans, with hypertension being present in 69.8% of stroke victims (Mollentze *et al.*, 1995). The THUSA study, carried out in the North West province, demonstrated that blood pressure was significantly higher in urban and transitional groups as opposed to their rural counterparts, with more than 20% of 'apparently healthy' subjects demonstrating blood pressure levels of  $\geq 140/90$  mmHg. A concerning finding was that many of the individuals identified with hypertension were untreated and were unaware that they suffered from the condition (Vorster, 2002).

Research has demonstrated that hypertension within black individuals, may have a *genetic cause*. Studies have in fact demonstrated a strong link between hypertension and family history thereof or family history of strokes (Steyn, 2006a). Aligned with this, it has been reported that a family history of hypertension is found 4 times more frequently in those with IHD than in individuals without the condition (Steyn, 2006a). However, research on the particular candidate genes responsible for this relationship and the development of hypertension has to date been limited, and the exact mechanisms governing the genetic link with hypertension within black individuals remains poorly understood (Steyn, 2006a).

The increased rate of hypertension within urban individuals has been linked largely to the consumption of westernised diets generally typified by high salt levels. These, along with

sedentary living, help to raise blood pressure levels (White & Dalby, 2008; Schutte *et al.*, 2006) and indeed facilitate the development of hypertension. While the prevalence of hypertension amongst different South African population groups has been fairly well documented, literature has shown that the disease, due to poor health care resources, is usually only identified and treated after major damage has been done. Thus, people with hypertension are usually unaware of the condition, unless they have had their blood pressure measured at a health care institution. For this reason, hypertension is often referred to as the “silent epidemic” in South Africa (Steyn, 2006a). While IHD remains low within the black population group, hypertension predisposes these individuals to increased risk of stroke, representing one of the leading causes of mortality within black South African individuals (Norman *et al.*, 2007; Steyn, 2006a). Since hypertension represents a major, yet modifiable risk factor for CHD, efforts need to be made to educate individuals on the potential health risks as well as lifestyle modifications which can generally improve individuals’ overall risk profile (Schutte *et al.*, 2006).

## **HYPERCHOLESTEROLEMIA**

As with other CVD risk factors, cholesterol levels vary according to both genetic and environmental factors: genetics are shown to be associated with 60% of the phenotypic variation in total cholesterol concentration (Sankaranarayanan, 1999). Environmental influences, including inactivity, overeating and the subsequent development of associated risk factors such as obesity and hypertension, further serve to promote the development of hypercholesterolemia (Poulter, 1999). Since westernised lifestyles and their associated reliance on fast food and sedentary living are fast becoming the norm throughout the world, hypercholesterolemia is becoming an ever conspicuous disease globally, in both developed and developing countries (Broxmeyer, 2004). Studies have proposed that 56% of global CHD is attributed to hypercholesterolemia, a level which is set to increase further in the presence of, and associated with, other compounding risk factors (Ghoziladeh & Davidson, 2008; Broxmeyer, 2004).

In South Africa, 62 000 hypercholesterolemia sufferers have been identified and a further 1100 are diagnosed each year (Hitzeroth, 1996). Although no national studies have been carried out on cholesterol levels, various provincial studies provide an indication of the prevalence of the condition within different population groups across the country. Research

has shown that hypercholesterolemia was historically a disease seen mainly in white urban males (Hitzeroth, 1996), with rates shown to be highest in white males, slightly lower in indian and coloured men, and *lowest* in *black males*. (Hitzeroth, 1996). Historical studies from the 1980s and early 1990s have highlighted the predominance of hypercholesterolemia affecting the white urban population group. Rossouw *et al.* (1983), in a study of a white Afrikaans population group in what was then known as the south-west Cape, demonstrated a prevalence of hypercholesterolemia in 30.1% of males and 34.4% of females, a value associated with increased risk of CHD. In addition to this, 70% of males and 80% of females demonstrated not only hypercholesterolemia, but indeed a combination of risk factors. In contrast to this, Seedat *et al.* (1992) demonstrated notably lower levels of hypercholesterolemia within black individuals. In their study of a group of Durban blacks, a prevalence of only 4% was demonstrated in both males and females. Similarly, Steyn *et al.* (1991) found hypercholesterolemia in only 1.1% and 2.3% of males and females respectively. Interestingly, in addition to this, was the finding that 96% of males and females demonstrated an increased high-density lipoprotein (HDL)/total cholesterol (TC) ratio, which they proposed served as a protective mechanism thus reducing overall CHD risk (Steyn *et al.*, 1991). More recently, studies have demonstrated a slight shift in hypercholesterolemia. In 1995, in a study carried out in the Free State, it was found that a large number of black individuals were at moderate risk for hypercholesterolemia (Mollentze *et al.*, 1995). This was especially prevalent in younger individuals who it is claimed were exposed to urbanized living typified by high levels of saturated fats and animal products (Mollentze *et al.*, 1995). Similarly in 2005, in Limpopo, in a study of rural black individuals, participants were found to have unexpectedly high levels of cholesterol (Alberts *et al.*, 2005), while finally in 2009, in the “Heart of Soweto” study, 14% of individuals had elevated blood cholesterol levels (Tibazarwa *et al.*, 2009). This slight increase in total cholesterol levels is likely to be associated with the demographic and nutritional transition, which has seen many black previously rural individuals move into cities and become part of the urban sector.

The 1998 and 2003 DHS are the only two studies to present any data on hypercholesterolemia in the *Eastern Cape*, however the data presented are based on self-reporting of the condition and are thus not likely to be entirely accurate. In 1998, 1.2% of men and women of the province reported the condition – the third lowest reported prevalence out of all the provinces in the country. This figure increased slightly to 2.2% of males and females in the 2003 DHS, potentially associated with transitional trends in nutritional and living habits.



It must be acknowledged however that total cholesterol levels remain higher amongst the white population (Rush *et al.*, 2007; Sparling *et al.*, 1994). While hypercholesterolemia prevalence rates range from 0-12% in black individuals (the rate is higher in urban individuals), prevalence is shown to be 17% for coloureds, and 25-25% for whites (Senekal *et al.*, 2003). Despite the high levels of obesity amongst black women, and the significant presence of hypertension amongst both black men and women, cholesterol levels appear to have remained lower than for whites and coloureds, even in the presence of urbanization and changes in dietary and nutritional habits (Mayosi *et al.*, 2009). And it is this that is thought to be of primary importance in explaining the low incidence of CHD and IHD amongst the black population group of South Africa (Alberts *et al.*, 2005; Sparling *et al.*, 1994). Furthermore, it has been found that black individuals tend to have markedly favourable total cholesterol in association with very high levels of protective HDL cholesterol (Mayosi *et al.*, 2009). In more than 80% of black South Africans, it is this which is thought to serve as a protective mechanism and keep the level of heart disease low within this population group (Mayosi *et al.*, 2009).

## **TYPE II DIABETES**

*Diabetes mellitus* covers a heterogeneous group of disorders, which all share the trait of elevated blood glucose. Collectively, diabetes is one of the most common chronic diseases, afflicting 5-10% of individuals particularly in westernised countries (Sankaranarayanan, 1999). Approximately 90-95% of diabetes is diagnosed as type II diabetes, a condition also associated with other chronic diseases including obesity (Narayan *et al.*, 2000). Statistics from the International Diabetes Federation in 2003 illustrated that the number of diabetics worldwide is expected to increase from 194 million found in 1993, to 330 million, predicted in 2030, with three in four of these individuals living in developing countries (Boutayeb, 2006). Indeed, by 2020, it is predicted that diabetes will contribute to 70% of all mortality (Ghoziladeh & Davidson, 2008).

Early studies carried out in the 1960s demonstrated that diabetes was virtually absent in black South African individuals leading traditional lifestyles: In accordance with this, in 1969, in a study carried out in Cape Town, a prevalence of only 3% was found in black individuals, a prevalence which was entirely hospital based (Vorster, 2002). Indeed, the first studies carried out, assessing the prevalence of diabetes among black South Africans, were based

on hospital outpatient populations (Levitt, 1996). However, in the face of increasing urbanization, it is predicted that the prevalence of this condition will increase by two to three times in the next 20 years (Aspray *et al.*, 2000; Narayan *et al.*, 2000). Indeed, diabetes occurrence within various different South African population groups is thought to be hinged primarily on the effects of urbanization and the adopting of an unhealthy lifestyle. This is evidenced by the fact that prevalence of diabetes amongst rural population groups has been shown to be low. Research has illustrated a prevalence of only 1% in rural Africans in the Western Cape as well as the Limpopo province, as opposed to 8% in those living in urban areas (Alberts *et al.*, 2005; Riccardi *et al.*, 2003). Further research in the Free State revealed a far more frequent occurrence of diabetes in both urban and rural communities, highlighting the western influence associated with urbanization and the promotion of associated chronic diseases of lifestyle (Mollentze *et al.*, 1995).

While a few provincial studies have been carried out, limited national surveys have assessed the prevalence of type II diabetes within black urban individuals. In 1998 the first national DHS carried out in South Africa found that self-reported prevalence of the disease, while highest in the Indian community, was in fact *lowest* in *black African groups*, with black urban males and females demonstrating a prevalence rate of 1.6% and 3.7% respectively. This low prevalence was confirmed in the self-reported findings from the 2003 survey. Within the *Eastern Cape* specifically, although figures for black urban males and females are not reported, low self-reported prevalence rates for diabetes were recorded in both 1998 and 2003, with females reporting a slightly higher prevalence of the condition. Findings from these surveys highlight the fact that in the face of urbanization, diabetes levels appear to have remained low in black individuals (Mollentze & Levitt, 2006) – a finding which has been attributed to different underlying mechanisms for diabetes that exist in black and white individuals. White population groups tend to demonstrate high levels of hypercholesterolemia, in addition to a central distribution of adiposity and a high waist to hip ratio – which in turn indicates and promotes a condition of insulin resistance (Cappuccio, 1997). In contrast to this, black population groups, despite the presence of raised glucose intolerance, tend to present with low levels of cholesterol and a different distribution of adiposity – thus predisposing these individuals to lower levels of insulin resistance and thus lower levels of type II diabetes (Cappuccio, 1997). Furthermore, it has been suggested that type II diabetes has a strong genetic component within black, particularly *Xhosa* individuals (most of whom live in the Eastern Cape) (Mollentze & Levitt, 2006). Erasmus *et al.* (2001) found that diabetic

individuals reporting an earlier onset of the condition, tended have a positive family history of the condition, tending to report it on paternal (31%), maternal (60.6%) or both sides of the family (6.6%).

Although these genetic influences may have an association with the low prevalence of type II diabetes within black South Africans, the profound influence of lifestyle factors, regardless of fat distribution, insulin resistance or heredity, cannot be ignored. Increasingly these factors are coming into play within the South African context, and will likely impact on disease prevalence in the future. Particularly pertinent to the South Africa context, it is important to note that the factors associated with and promoting obesity, are traditionally also associated with type II diabetes. Aligned with this, those individuals forming part of the nutrition transition, are exposed to lifestyle and environmental factors which simultaneously promote the development of both conditions - as illustrated in Figure 3 (Candib, 2007). This is a concerning statistic given the already high obesity prevalence in black South Africa women, in whom it is likely that diabetes will follow a similar trend.

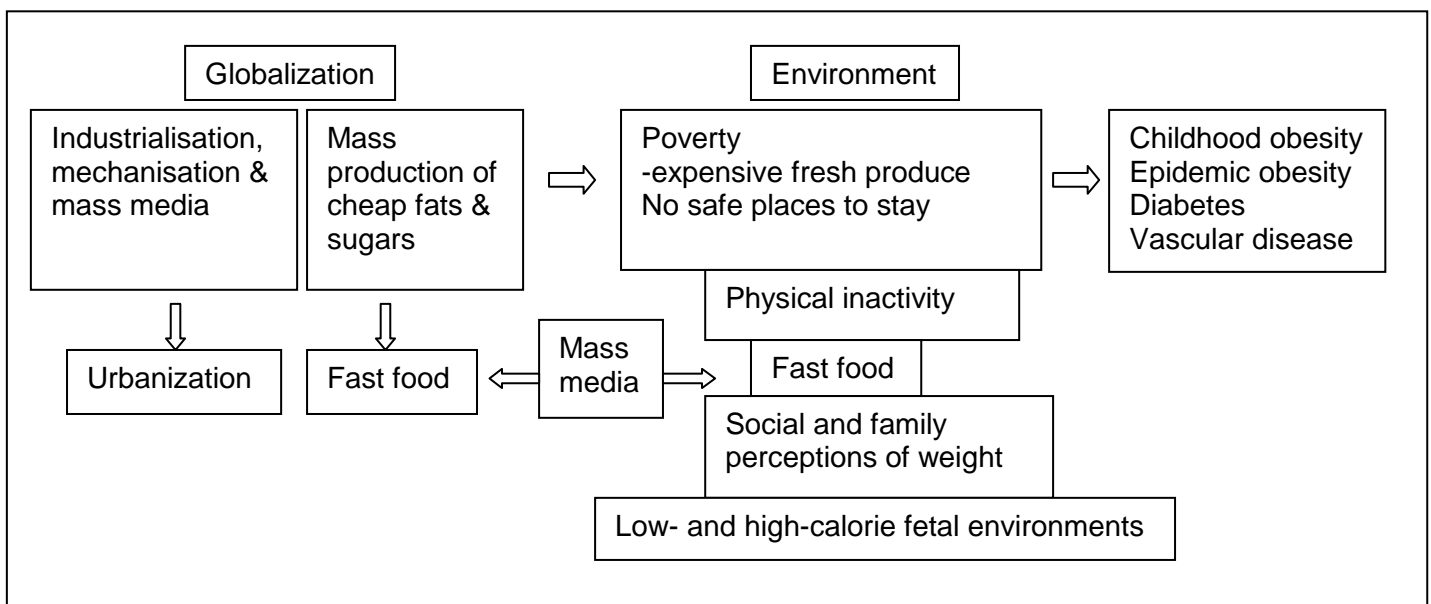


Figure 3: Factors contributing to diabetes and obesity (Sourced and adapted from Candib, 2007)

In addition, aligned with the fact that South African is a middle income country with a high prevalence of poverty, diabetes as a disease is not only expensive to manage and treat, but there are several indirect costs to society, including lost productivity due to sickness, absence, disability, early retirement and premature mortality (Narayan *et al.*, 2000). This is of

particular importance with regard to manual labour, since physical limitations associated with diabetes lead to lost productivity - a vital component of a developing economy (Narayan *et al.*, 2000). Hence it is important to implement prevention strategies, including changes in lifestyle, diet, and the promotion of healthier living overall (Riccardi *et al.*, 2003; Narayan *et al.*, 2000).

## **LIFESTYLE-RELATED RISKS**

### **POOR DIET**

Diet, or more specifically dietary composition, has a direct impact on body weight and percentage body fat (Krauss *et al.*, 2000). A poor diet, or one that is high in saturated fats, and low in fibre, thus plays an extremely important role in predisposing individuals to chronic diseases of lifestyle, including obesity, type II diabetes, hypertension, hypercholesterolemia and CHD (Walker *et al.*, 2003; Riccardi *et al.*, 2003).

The impact of diet on CVD risk is primarily related to the relationship between various different food stuffs, and the proportion of energy derived from each individual food source in relation to overall energy consumed (Walker *et al.*, 2003; Sparling *et al.*, 1994). It is generally agreed that diets rich in fruit and vegetables and relatively lower in carbohydrates (CHO) and fat, promote healthy living and help reduce the risk of CVD (Boutayeb, 2006; Kesa & Oldewage-Theron, 2005); while the converse is true for diets rich in saturated fats and sugar and containing lower levels of fibre – hence facilitating and encouraging the development of various health risks (Krauss *et al.*, 2000). Research has in fact shown that if total fat consumed is above the threshold level of 35-40% of total energy intake, the risk of type II diabetes is significantly increased, as are various associated risks, including hypertension, hypercholesterolemia and obesity (Riccardi *et al.*, 2003).

The role of diet in relation to disease risk in South Africa presents an interesting scenario – this, due to the combined prevalence of both under-and over-nutrition associated with a very diverse population, comprising individuals at both the top and bottom ends of the socio-economic scale. The 2003 DHS assessed the diet of all population groups across the country, and whether individuals were meeting their respective recommended daily allowances (RDAs) and consuming sufficient essential nutrients (DHS, 2003). Overall it was

reported that individuals in urban areas consumed notably more micronutrients, and micronutrient scores were also better in individuals with higher levels of education. Interestingly, whites and *urban blacks* had the best micronutrient scores, while *non-urban blacks* and indians had the worst (DHS, 2003). However, blacks were also shown to have the highest prevalence of deficiency for all nutrients compared to all other population groups (DHS, 2003). This survey presents the only available data on the nutritional status of the *Eastern Cape* population. Out of the 9 provinces of the country, males were found to have the third worst micronutrient score, and females the fourth worst. Additionally, 43% of individuals had a poor total dietary score. Since the Eastern Cape is one of the poorest provinces in the country, it is not surprising that these findings highlight the prevalence of malnutrition in these individuals, despite the country-wide 'nutrition transition' which is thought to be fundamental in the changing health profile of South Africans over the last decade.

### **The 'nutrition transition'**

A 'nutrition transition' is a term describing the changes in dietary patterns which occur when a population moves from a predominantly rural lifestyle to that which typifies an urban industrialised one (MacIntyre *et al.*, 2000; Vorster *et al.*, 2000). It is thought that many of the factors that explain this transition concurrently explain similar shifts in physical activity and body composition (Popkin, 2002; Popkin, 1998).

The 'nutrition transition' is associated with two historic processes which occur either simultaneously or precede the transition (Popkin & Gordon-Larsen, 2004). The first is a 'demographic transition' in which there is a shift within populations from a pattern of high fertility and mortality, to one of low fertility and mortality (more typical of first world, industrialised countries). The second is an 'epidemiological transition', in which there is a shift from a pattern of infectious diseases to one where chronic diseases associated with urbanization become more prevalent (Popkin & Gordon-Larsen, 2004). The 'nutrition transition' is closely linked to both these transitions, particularly with regard to changes in diet and physical activity patterns (Figure 4).

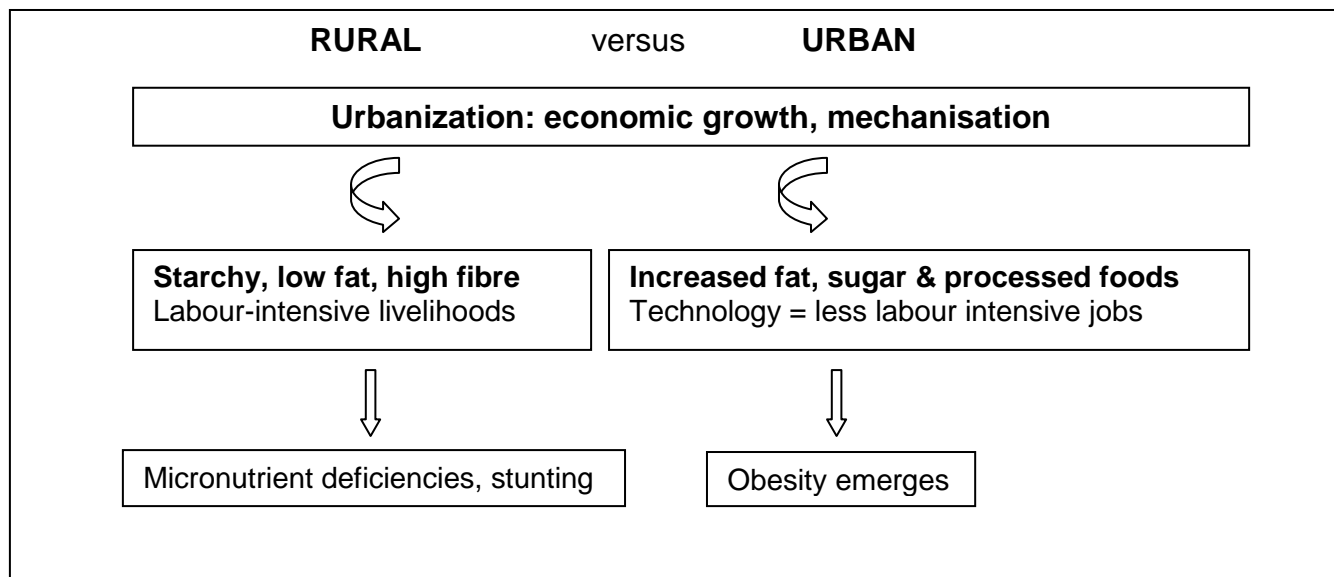


Figure 4: Stages of the nutrition transition (Sourced and adapted from Popkin & Gordon-Larsen, 2004)

Indeed, associated with this shift to westernised nutrition, is an associated westernised lifestyle including more sedentary living and physical inactivity. As seen in figure 4, parallel with the nutrition transition, are changes in diet and activity, and as a result, the emergence of related disease risks. The effects of the ‘nutrition transition’ within the South African context are evidenced in the large numbers of obese individuals within both the adult and child population of South Africa, a finding which is becoming more apparent despite the presence of stunting and under-nutrition which still exists within the country due to high levels of poverty (Steyn *et al.*, 2006; Labadarios & Steyn, 2005).

Within the country as a whole, numerous dietary studies have documented this nutrition transition, particularly within black individuals (Joubert *et al.*, 2007a; Bourne *et al.*, 2002; Kruger *et al.*, 2002; Vorster *et al.*, 2000; Popkin, 1998; Levitt, 1996; Bourne *et al.*, 1996). At a very basic level, changes in dietary patterns have been linked with changes in food availability and provision, and hence, changes in dietary composition (Popkin, 2002). This is particularly so in population groups which have become more urbanized over the last few years, and which are increasingly exposed to new technologies, increased access to western media and the accelerated introduction of western technology (Popkin, 2002). Within South Africa, per capita food supply has also increased notably: From 1961 to 2001, available protein supplies increased from 68.4 to 75.1 g, fat from 61.2 to 79 g and CHO supplies from 445 to 478 g. While the availability of food does not directly correlate with consumption, the

mere increase in food availability and supply implies that the risk of over consumption – indeed the increase particularly in fat availability per capita – may have had serious implications in terms of chronic disease risk (Steyn, 2006b).

A comparative study assessed the change in diet in a group of black women from 1969 to 1989. Findings illustrated that the percentage of energy from dietary fat increased from 19 to 24%, protein from 12 to 14%, and that there was an overall 8% increase in total energy consumed (Walker *et al.*, 2001). These figures may help to explain another finding of the study, that the percentage of obese individuals within the group increased significantly from 9.3-19.9% (Walker *et al.*, 2001). Similar findings were demonstrated in a study by Bourne *et al.* in 1996. Dietary intakes of black South Africans living in the Western Cape (Cape Town) were analysed, and dietary consumption was correlated with time spent living in the city. Findings revealed that within this period carbohydrate intake (as a percentage of total energy intake) decreased from 61.4% to 52.8%, fibre decreased from 20.7g to 16.7 g, protein intake remained similar and fat intake (as a percentage of total daily energy intake) increased from 23.8% to 31.8% (Walker *et al.*, 2001). These findings were confirmed by Vorster *et al.* (2000) who similarly demonstrated increased fat intake associated with urbanization, as well as Vorster *et al.* (2005) who assessed the impacts of urbanization on nutrition (THUSA study). When comparing urban and rural women, they found that percentage energy from carbohydrates (CHO) dropped (67.4% to 57.3%), while the percentage energy from protein and fat increased (protein = 11.4% to 13.4%, fat = 23.6% to 31.8%). Associated with this were increased levels of obesity within the more urbanized group. These dietary changes clearly illustrate a population group in transition (Steyn, 2006a; Popkin, 1998;). Accompanying this, is a general lack of physical activity, placing such individuals at enhanced risk for CVD, and associated risk factors including diabetes, hypertension, and obesity (Talip *et al.*, 2003; Fried & Rao, 2003; Ndaba & O’Keefe, 1985).

Aligned with the prevalence of over-nutrition and changes in diet associated with the nutrition transition, a compounding factor, specific to the South African context, is the role of mal- and under-nutrition. *Under-nutrition*, while affecting mainly poor rural individuals involved in manual labour, is characterised by the consumption of diets in which energy obtained is insufficient to meet daily energy requirements (O’Keefe *et al.*, 1983). Indeed, alongside the high prevalence of adult female obesity is under-nutrition among the working rural blacks and malnourishment amongst many of the impoverished youth (Joubert *et al.*, 2007a; Popkin,

2002). This is particularly a problem in South Africa, due the fact that rural black individuals (particularly males) make up the majority of the manual work force (Christie, 2001; Ndaba & O'Keeffe 1985), and in fact largely fall below 2/3rds of the recommended daily allowance (RDA) (Bourne *et al.*, 2002).

Another important consideration within the South Africa context, associated with under-nutrition, is the role of early food deprivation leading to stunting but linked with the development of obesity later in life (Kruger *et al.*, 2010; Steyn *et al.*, 2005). Research has shown that associated with this, living conditions during early childhood can play a major role in appetite regulation, eating behaviours and patterns of body weight gains (Kruger *et al.*, 2006). Research has suggested that fetal growth retardation results in metabolic changes which occur *in utero*, changes which can, in turn, lead to increased insulin resistance and the development of obesity later in life (Prentice, 2006; Punyadeera *et al.*, 2001b; Popkin, 1998). It has been suggested that early food deprivation may alter the regulatory mechanisms for energy intake, leading to increased levels of fat storage and obesity later in life (Case & Menendez, 2009; Kruger *et al.*, 2001; Popkin, 1998). This has been evidenced in various studies, including one by Case and Menendez (2009), who reported that childhood deprivation, measured as a childhood hunger index, was positively and significantly associated with obesity in women during adulthood. Furthermore, women who went to bed or school hungry, or were forced to eat at other people's houses because of food shortage, were 15% more likely to be obese than those who reported none of these factors (Case & Menendez, 2009). In a further study by Kruger *et al.* (2001), it was found that stunted girls were at risk of having excessive fat stores and appeared to be at greater risk of accumulating fat on the trunk – both of which are associated with increased CVD risk.

Nutrition knowledge, explained as knowledge about correct and healthy nutrition practices, also plays a role in what and how individuals eat. Ignorance of healthy eating practices can lead to the consumption of unhealthy foodstuffs, which in turn serves to promote the onset of obesity and various related risks (Steyn *et al.*, 2000). Nutrition knowledge has been found to be particularly low amongst black women within South Africa, who have been found to be, for the most part, ignorant of the principals of healthy eating and living (Steyn *et al.*, 2000). This finding was demonstrated in a study of older black women (over 45 years) as well as black students (Steyn *et al.*, 2000).



In addition to nutritional ignorance with regard to healthy eating, the role of the media and the availability of fast, palatable foodstuffs, are also factors to be considered (Alm-Roijer *et al.*, 2004). Associated with westernised sedentary lifestyles, the food industry serves to compound the problem somewhat: Ever increasing food portions, incorrect listing of kilocalories on food labels, and the increased use of high fructose corn syrup as a caloric sweetener in most soft drinks and most solid foods - all these things promote weight gain. What is more, fast foods, which also contain high levels of both fat and sugar, are incredibly palatable and often inexpensive (Tibazarwa *et al.*, 2009; Goedecke, 2006; Haslam & James, 2005; Speakman, 2004). The *novelty* of fast food for many previous rural and disadvantaged individuals results in a desire to emulate the lifestyles and foodstuffs that were previously associated with the wealthy white urban population who, for many years, were notably better circumstanced - this, despite the potentially very dangerous health risks (Cappuccio, 2007; Walker *et al.*, 2001). The consumption thereof, despite the potential health risks, is therefore, for the most part, encouraged (Walker *et al.*, 2001; Rippe *et al.*, 1998).

## **PHYSICAL INACTIVITY**

The American College of Sports Medicine (ACSM, 2000) proposes that there is a dose-response effect between greater amounts of physical activity and lower CVD risk (Balady, 2000). Aligned with this, an increasing number of prospective epidemiological studies support the notion that a physically active lifestyle and at least 30 minutes of moderate intensity exercise on most and preferably all days of the week independently lowers an individual's risk for CHD (Dalleck *et al.*, 2009; Balady, 2000; Sparling *et al.*, 1994). In this connection, being physically active serves to improve endothelial function due to enhanced vasodilation and vasomotor function in the blood vessels (Fang *et al.*, 2003). It also contributes to weight loss and glycaemic control, improves blood pressure, lipid profile and insulin sensitivity, increases myocardial oxygen control, and reduces plasma fibrinogen (Fang *et al.*, 2003; Chopra *et al.*, 2002; Popkin, 1998). Physical activity therefore plays an important role in reducing most CVD risk factors – including obesity, hypertension, type II diabetes and hypercholesterolemia (Riccardi *et al.*, 2003; Lambert *et al.*, 2001; Popkin, 1998) – the result being that mortality from CVD in both males and females, associated with regular physical activity is significantly reduced (Fodor & Tzerovska 2004; Walker *et al.*, 2003).

Lack of physical activity, and the leading of sedentary lifestyles, is a trend which has increased dramatically in the last few years throughout the world (Ghoziladeh & Davidson, 2008). Research has shown that 60-85% of the world's population do not undertake sufficient daily physical activity for the health benefits to take effect (Ghoziladeh & Davidson, 2008). This is thought to be partly related to the explosion in technological advances, which has decreased the need for physical activity: most products and services are quickly and easily available either online, or by delivery, which encourages laziness and an increased trend towards sedentary living (Rippe *et al.*, 1998). The impacts of technology are particularly prevalent in urban environments, as are the effects on lifestyle. Based on these factors, it has been estimated that physical *inactivity* is responsible for a third of all deaths due to coronary heart disease and type II diabetes, and is one of the leading causes of cardiovascular disease risk (Joubert *et al.*, 2007b; Walker *et al.*, 2001; Manson *et al.*, 1999; Popkin, 1998).

Within South Africa, although studies have been limited, research has demonstrated a shift in physical activity levels as a result of urbanization and a period of transition which the population is currently undergoing (Lambert & Kolbe-Alexander, 2006); and thus South Africa appears to be following the global trend of increasing levels of inactivity. While specific trends, particularly within the different provinces and population groups, are hard to come by (Lambert & Kolbe-Alexander, 2006), recent literature has reported an increasing prevalence of physical inactivity across all population groups (DHS, 2003), with the whites and indians shown to be the most inactive, and *black individuals* shown to be the *least inactive* (Lambert & Kolbe-Alexander, 2006). The *Eastern Cape* specifically was reported to have the *highest* percentage of sufficiently active males (41.3%) and the *second highest* percentage of sufficiently active females (23.7%), potentially due to the large rural component within the province.

Historically (and still the case with rural individuals), levels of physical activity have been shown to be significantly higher within the rural setting. While partly related to livelihood and having to farm and perform hard manual labour to make a living and survive, physical location also dictates that individuals have to commute long distances on foot, thus ensuring that rural lifestyles on the whole are more active (Christie, 2001; Sparling *et al.*, 1994). This is in contrast to the experiences of individuals forming part of the urban sector – for whom the necessity to engage in physical activity is reduced (Walker *et al.*, 2001). Various studies have documented this change: In a study carried out in the North West province, levels of physical

activity were significantly reduced within a group of urban (previously rural) individuals – which in turn was associated with reported higher levels of obesity as a result (Bourne *et al.*, 2002; Kruger *et al.*, 2002; Lambert *et al.*, 2001). In 2000 physical inactivity was reported to account for 3.3% (17 037 deaths) of all mortality within the country – with the majority of deaths being attributed for IHD (Joubert *et al.*, 2007b). More recent literature suggests that 20-30% of IHD, type II diabetes and ischemic strokes can be attributed to physical inactivity – a concerning statistic given the fact that 43-49% of South Africans are reportedly inactive (Cook *et al.*, 2010; Cook, 2007). It is reported that over 40% of historically disadvantaged individuals living in urban impoverished informal settlements do not participate in any leisure or occupational physical activity (Sparling *et al.*, 2000). Aligned with the fact that 43% of black South Africans are now living in urban environments, research has demonstrated a significant decrease in physical activity levels as a result of this transition (Bourne *et al.*, 2002) – resulting in 49% of men and 43% of women reportedly taking part in insufficient physical activity to achieve any health benefits (Joubert *et al.*, 2007b).

In addition to urbanization, a disparity in physical activity levels between black women and men is also evident. On the whole, black males are shown to be more active than their female counterparts, due, for the most part, to their involvement in manual labour which forms the bulk of the work productivity within the country (Christie, 2001). Various studies have demonstrated this disparity: Dugas *et al.* (2009b) demonstrated within a sample of black males and females from Khayelitsha (an urban informal settlement in Cape Town), that both total daily energy expenditure as well as activity daily energy expenditure was significantly higher amongst male subjects, which in turn correlated with these subjects being leaner. Interestingly it was further evidenced that men involved for longer periods of time in vigorous activities had lower levels of adiposity than female subjects, who all in fact demonstrated BMI values of  $31\text{kg.m}^{-2}$  – a value higher than the national average of  $27.1\text{kg.m}^{-2}$ . In accordance with this Cook *et al.* (2009) reported that a reduction in subsistence tasks (typically light to moderate in intensity and carried out throughout the duration of the day) by black females, has seen increases in obesity within this group, in addition to various other chronic diseases of lifestyle (Cook *et al.*, 2009). In contrast Seftel (1978) and Walker (1991) demonstrated high levels of physical activity, particularly amongst black males in rural and transitional environments, and a parallel reduced risk for CVD (Sparling *et al.*, 1994). In accordance with these findings, there are concomitant and large discrepancies in obesity levels between these population groups. Black females are shown to be at the top of the scale in terms of

obesity prevalence, and males at the bottom – and it is thought that physical inactivity may play an important role in this regard (Dugas *et al.*, 2009b; Lambert & Kolbe-Alexander, 2006). In line with this, various initiatives by the Ministries of Health, Sport and Education have been put in place to encourage healthy lifestyles within all population groups, and promote mass participation in sport and recreation activities (Joubert *et al.*, 2007b; Lambert & Kolbe-Alexander, 2006). It is hoped that these initiatives, in addition to further research on the benefits of physical activity within different South African population groups, will serve to reduce the prevalence of physical inactivity and consequent health risks within the population as a whole.

## **TOBACCO USE**

The smoking of tobacco is regarded as an important and reversible risk factor for CVD (Fodor & Tzerovska, 2004), the magnitude of which relates directly to the number of cigarettes smoked daily, or in an individual's lifetime (Fordor & Tverovska, 2004; McArdle *et al.*, 2001). It is reported that the smoking of tobacco contributes to a notable share of premature mortality globally, accounting for 4.8 million deaths in 2000 – a figure that is expected to increase to 10 million by 2025 (Groenewald *et al.*, 2007). While high rates of smoking are demonstrated globally, research has illustrated a smoking prevalence of nearly 50% in *developing countries*, a figure which is thought to be increasing by 3.4% per year (Boutayeb, 2006). Furthermore, in developing countries, CVD accounts for the highest proportion of tobacco-attributable deaths (Gaziano, 2007), in addition to chronic respiratory disease and tuberculosis (TB) (Groenewald *et al.*, 2007). Indeed, most smoking attributable deaths are the result of other CVD risks, which increase as a result of tobacco use and thus increase mortality associated with CVD (Critchley & Capewell, 2003). Aligned with this, research has shown that smoking cessation can result in a 36% reduction in CHD mortality (Critchley & Capewell, 2003).

Since the mid 1980s, although a few studies have investigated the prevalence of smoking in different population groups in South Africa, these have largely been provincial, encompassing small province specific population groups. In 1983, Rossouw *et al.* found that 33.4% of males and 12.9% of females comprising a rural white Afrikaans community in the Free State smoked, while Steyn *et al.* (1991), in a study of the urban black population of the Cape

Peninsula, demonstrated smoking prevalence in 52% of males, and 8.4% of females; 24.7% of the males smoked 10 or more cigarettes per day.

In 1992, the first national statistics became available, which reported that 32% of South Africans smoked (Groenewald *et al.*, 2007). Smoking was more prevalent in males of all population groups (52% of males smoked versus 17% of females) and smoking rates among *black* individuals were higher than those among whites (Sitas *et al.*, 2004). However, notably lower smoking rates were reported for black *females*, 10% of whom reportedly smoked, in comparison to 27% of white and 59% of coloured females (Sitas *et al.*, 2004).

In 1995, the percentages of men and women smokers were the same as in 1992, but in 2000 these figures increased to 60% and 17% respectively (Vorster *et al.*, 2000). In 1998 smoking prevalence across the country was assessed in the national DHS. This survey provided the first available *Eastern Cape* data, and while neither race nor sex specific, smoking rates for the province were presented that were absent previously: it was reported that 50% of men within the Eastern Cape smoked, compared to 11% of women. For *males*, this represented the *third highest* prevalence rate in the country, while for *females*, this was the *third lowest* prevalence rate (DHS, 1998). According to race and sex specific findings, it was shown that coloured males presented with the highest smoking prevalence (57%), while prevalence was lowest in *black males* (40%), while females followed a similar trend (highest in coloureds = 40%, lowest in blacks = 5%).

From 2003, national statistics reported that smoking prevalence had *decreased* across the board (Groenewald *et al.*, 2007): Indeed, findings from the 1998 and 2003 DHS demonstrated a decline in smoking among adults from 24.6% in 1998, to 21.4% in 2003, including 35.8% of men and 8.1% of women (Saloojee, 2006). Despite this general decline, it was acknowledged that there remained large discrepancies between the different population groups and sexes across the country (Groenewald *et al.*, 2007). In general, smoking prevalence is shown to be significantly higher in males compared to females. Specifically, prevalence has been shown to be highest amongst coloured males and females, lowest in black individuals (particularly females), with white individuals demonstrating intermediate rates (Groenewald *et al.*, 2007). The 1998 DHS confirmed this, reporting that more than 39% of black, white, coloured and indian adult males (older than 15 years) smoked daily or occasionally, while prevalence was lowest in rural black males and females. The discrepancy

in smoking prevalence between males and females is particularly evident within the black population: For example, in 1998, while smoking prevalence was lowest in the black population group as a whole, 33.9% of black males smoked, in comparison to only 4.2% of black females (Peer *et al.*, 2009). The fact that 30% fewer females smoked may be linked to cultural beliefs and ideals in which smoking is perceived as taboo for black women of reproductive age, and is not recognised as being socially acceptable (Peer *et al.*, 2009; Saloojee, 2006). In addition to sex differences, smoking has also been shown to be lower in individuals of lower socio-economic status (Mezei *et al.*, 2005; Blakely *et al.*, 2005). This suggests that higher cigarette prices tend to lower smoking prevalence among these individuals (Groenewald *et al.*, 2007). Aligned with this, smoking decreases across the board have been attributed to stringent tobacco control legislation and taxation (Groenewald *et al.*, 2007). Over the last decade, various smoking laws and legislation policies have been implemented, including health warnings on cigarette packets, increased taxes on the retail price of cigarettes, and the prohibition of smoking in public places and the sale of tobacco products to minors (Saloojee, 2006). According to the South African Advertising and Research Foundation surveys, 2.5 million individuals stopped smoking during this period.

In addition to statistics on smoking prevalence, mortality estimates provide a useful means by which tobacco-related mortality can be estimated. Before 1999 there were limited data available on smoking-related mortality. From the sketchy age-standardised mortality data covering the period 1949-1985, it was estimated that 27.9 out of 100 000 deaths were attributable to lung cancer in black urban males – just over half the figure of 48.7/100 000 for white males (Groenewald *et al.*, 2007). While this had increased slightly to 31.1 per 100 000 for black males in 2000, the rate of black *females* was only 6.3/100 000. Of concern however, was the finding that cancer in *younger black* individuals (within the 35-44 and 45-54 age groups) was notably *higher* than in *whites* of similar age, despite being notably lower in the older age groups. Based on this finding it is proposed that lung cancer in black individuals may exceed rates in white individuals if tobacco use increases (Groenewald *et al.*, 2007). Furthermore, it has been reported that smoking causes one in five deaths from TB (Sitas *et al.*, 2004), a concerning statistic given the prevalence of TB within the country. Therefore, while smoking prevalence has declined, the risks of tobacco use remain apparent within the country, and therefore consistent intervention programs reducing tobacco consumption are essential in order to ensure that the health risks associated with smoking are not underestimated, and smoking-related mortality is reduced (Groenewald *et al.*, 2007).

## ALCOHOL CONSUMPTION

While the inverse association between moderate alcohol consumption and CVD is well established (Rimm, 1996), a high intake of alcohol is associated with liver cirrhosis and certain cancers, while a low intake has been shown to have a protective effect against certain cancers as well as CHD (Walker *et al.*, 2002; Walker, 1995). Alcohol consumption, in addition to its association with liver disease and various cancers, is also associated with hypertension (Schneider *et al.*, 2007).

Within South Africa, historically, different patterns of alcohol consumption have been evident in different population groups. Traditionally, rural black individuals were known to consume intoxicating drinks made from plants, fruits and grains, while wine and beer were typically consumed by white individuals (Schneider *et al.*, 2007). However, while the consumption of traditional alcoholic drinks such as sorghum beer remains high, increasingly these drinks are being replaced by western alcoholic drinks, as black individuals become more urbanized (Walker, 1995). Viticulture has become a prominent part of the South African way of life, and is thought to have had a notable impact on drinking behaviours, particularly within the Northern and Western Cape (Walker *et al.*, 2002). The supply of crude wine as part of black farm workers wages is thought to have contributed to the culture of heavy drinking that currently exists within the country (Schneider *et al.*, 2007), such that alcohol consumption has been termed 'the country's most abused drug' (Walker *et al.*, 2002).

In 1998, alcohol consumption was assessed at a national level (DHS, 1998). Results from the survey indicated that 28% of South Africans, accounting for 8.3 million individuals, consumed alcohol. Within this group, prevalence rates were reported to be highest in white men (71%) and women (51%) and lowest in *black* (12%) and asian women (9%). Findings from the *Eastern Cape* specifically demonstrated that 47.4% of males and 16.2% of females consumed alcohol. In 2003, the follow up DHS indicated that alcohol consumption had decreased. These findings were mirrored across the different provinces including the *Eastern Cape*. The latest estimates suggest that 50% of South African men and 20% of women consume alcohol (Schneider *et al.*, 2007). This statistic is based on household surveys, however, and is therefore likely to be an underestimation (Schneider *et al.*, 2007). Consumption is reported to be particularly marked in *black males*, who have been shown to

consume substantially higher levels of alcohol than their female counterparts (Schneider *et al.*, 2007).

Associated with this, beer halls and shebeens form a significant part of impoverished urban environments, and serve to encourage the culture of heavy drinking, particularly among urban black males (Schneider *et al.*, 2007). In addition, the South African population structure is slanted towards young people who tend to drink heavily at weekends, resulting in increased morbidity and mortality from accidents, crime and violence (Schneider *et al.*, 2007). Aligned with this, research has shown that 46% of all cases of mortality due to non-natural causes demonstrate blood alcohol concentrations of greater than or equal to 0.05g/100ml, which is the legal limit for driving (Schneider *et al.*, 2007). A survey carried out between 1999 and 2001 in fact found that of patients treated in trauma units in the South African cities, 17-67% of patients had blood alcohol concentrations of greater than or equal to 0.05g/100ml (Schneider *et al.*, 2007).

Alcohol consumption is not only associated with long term health complications, it is also associated with aggressive behaviour which often leads to violent behaviour, crimes and domestic violence (Schneider *et al.*, 2007). Alcohol use is also associated with unsafe sex and in turn an increased risk of contracting HIV – something which is particularly concerning in South Africa given the fact that almost one in five HIV patients studied at a large infectious disease clinic in Cape Town met the criteria for an alcohol use disorder (Schneider *et al.*, 2007). In addition, of the 20 countries which comprise the WHO AFR-E sub-region, it is reported that South Africa has the seventh highest level of all 14 WHO regions, consuming an estimated 7.1 litres of alcohol per adult (Schneider *et al.*, 2007). With the relatively high level of abstainers in this region, this translates to a rate of 16.6 litres per drinker, which is thought to be close to the highest rate in the world (Schneider *et al.*, 2007). Alcohol abuse results in a considerable burden of disease throughout the country as a whole, including alcohol-related homicide and violence, alcohol-related road traffic accidents, alcohol use disorders and fetal alcohol syndrome (Schneider *et al.*, 2007). To counter these problems, The National Liquor Act of 2003, while promoting a sustainable liquor industry, aims to encourage responsible drinking and thereby to reduce the social and economic costs associated with alcohol abuse (Schneider *et al.*, 2007).



## PERCEPTIONS OF HEALTH RISKS

An important consideration within the South Africa context, is how individuals perceive their health, and aligned with this, how aware they are of the potential health risks they may face. Although research is limited, both the 1998 and the 2003 DHS presented findings on the self reporting and perception of various CVD risks and compared these findings to measured values. These findings suggest that South Africans, particularly black South Africans, are largely ignorant of their health status. Hypertension, in particular was, shown to be largely under-reported and therefore to go undiagnosed and treated. In the 1998 DHS it was found that black males in particular were ignorant of their blood pressure, with large discrepancies between self-report and actual measures. Within the Eastern Cape specifically, although data is for the province as a whole and therefore encompassing all population groups, it was found that less than half all hypertensive males were aware of the condition (19% knew their blood pressure, only 8% reported it to be high), while only 12.2% of female hypertensives were aware that their blood pressure was high – a finding confirmed by the 2003 survey.

Obesity is another CVD risk which has been shown to be largely under-perceived, something which has been linked to different social and cultural ideologies. Within different cultural settings, specific social and psychological factors are associated with obesity. Within 'westernised' culture, where a slim figure is viewed as the ideal, obesity can lead to stress and trauma associated with the negative stigma attached to obesity (Bray & Champagne, 2005). Obese individuals often face prejudice in social and economic settings, the result thereof being that their overall quality of life is lowered (Goedecke, 2006; Bray & Champagne, 2005). However, perceptions of obesity in South Africa are slightly different, wherein notable differences have been found in perceptions of the 'ideal' body weight within different cultural groups (Prentice, 2006). Xhosa culture in particular (which predominates in the Eastern Cape) commands a very different viewpoint in which the opposite is preferred. In Xhosa and Zulu cultures, overweight is perceived to be a sign of health and prosperity. It is thought to represent happiness, beauty, affluence and wealth (Mciza *et al.*, 2005), and to reflect a husband's ability to care for his wife (Joubert *et al.*, 2007; Goedecke, 2006; Puoane *et al.*, 2002). Such perceptions govern a preconceived idea that 'bigger is better' and thus larger BMIs become 'ideal'. Various studies have demonstrated differences between perceived and actual prevalence of obesity: Case and Mendez (2009) found that within a sample of women from Khayelitsha, a BMI of  $30\text{kg.m}^{-2}$  was perceived as 'ideal'. This was in

comparison to a BMI of  $25\text{kg.m}^{-2}$  perceived as ideal in males (Case & Menendez, 2009); Mvo *et al.* (1999) reported that while 9.7% of black men and 22.1% of women perceived themselves to be overweight, in reality 29.2% and 56.6% were actually shown to be so. Furthermore, with the HIV/AIDS explosion, being overweight is thought to demonstrate an individual's status as being free from infection (Prentice, 2006; Puoane *et al.*, 2002; Clark *et al.*, 1999). This places additional strain on attempts to promote healthy living, and makes efforts to curb the increase in CHD that much more challenging (Prentice, 2006).

## **CONCLUSION AND AIMS OF THIS RESEARCH**

The quadruple burden of disease, unique to the South African setting, sees many different population groups within the country facing a broad spectrum of disease risk. From a health care perspective, this means that resource allocation has to be carefully considered such that numerous disease risks are treated, and ideally prevented. From a research point of view, challenges arise due to the specific risks that different population groups within different provinces face. Indeed, these differ according to socio-demographic location, level of poverty, as well as cultural norms and belief systems. The Demographic and Health Surveys (DHS), carried out in 1998 and 2003, together represent the most conclusive surveys at a national level to date: not only because of the large-scale nature of these surveys, but indeed because additional disease risks (including communicable diseases), issues of violence, level of poverty, and perceptions of health, were taken into account – thus providing an overall indication of the quadruple burden of disease at a country-wide level. In addition, and specific to this research project, these surveys represent the only literature available on chronic disease risk within the Eastern Cape Province. This is despite the fact that mortality estimates placed CVD as the second leading cause of mortality within the province (Bradshaw *et al.*, 2000).

According to the 2001 census, the Eastern Cape contains the second highest number of households in the country, with 650 095 urban and 862 570 rural households. Therefore, for the purposes of the DHS within the Eastern Cape, 18 urban and 45 rural sample points were selected. Within each household, a Household Questionnaire was administered in addition to an Adult Health Questionnaire, which was administered to all adults over 15 years. Anthropometric measures of stature, mass, waist circumference, blood pressure and peak pulmonary flow were taken, with the aim of using hypertension and chronic respiratory

disease as indicator conditions of chronic disease. At the time of the survey it was agreed that the survey should be conducted every 5 years to enable the Department of Health to monitor trends in health services. Since 2003, *no* national surveys have been done, and although studies have been conducted in various other provinces of the country, *no research has been undertaken in the Eastern Cape*. In light of this, the aim of this research project was to bridge a gap which exists in the literature, with regard to Eastern Cape literature specifically. It aimed to build on the work done in the 2003 DHS and provide a more conclusive indication of cardiovascular disease risk within the province. Indeed, it is hoped that findings from this research will enable inter-provincial comparisons to be made, and thereby for country-wide CVD risk to be monitored and controlled.

## CHAPTER III

### METHODS

Research has assessed CVD risk within the Eastern Cape, but as it is limited, there remains paucity within the literature with regard to the prevalence and extent of CVD risk factors within urban black individuals. Therefore the central aim of this research project was to assess and compare the presence and prevalence of cardiovascular disease risks within an urban black population in the Grahamstown region of the Eastern Cape, South Africa. More specifically, it aimed to identify which CVD risks predominate within males and females in this population, and to compare risk profiles between the sexes. This chapter aims to critically discuss various methods of CVD risk assessment, including those chosen for this research project.

#### EXPERIMENTAL DESIGN

##### **Dependent and independent variables of interest**

Various morphological, cardiovascular (CV) and lifestyle-related factors were assessed in order to provide an indication of the following risk factors, each defined as a dependent variable of interest: obesity – representing a morphological risk; hypertension, hypercholesterolemia and type II diabetes – all three representing cardiovascular (CV) risks; and finally lifestyle-related risks, including diet, physical activity, tobacco use and alcohol consumption and dependence. According to these criteria, CVD risk profiles of *males* and *females* forming an *urban working population* were compared, thus sex and demographic location (urban) represented independent variables of interest. In addition, self reported measures of hypercholesterolemia, type II diabetes and hypertension, and perceived measures of obesity, were compared to actual values (Table II).

Table II: Dependent and independent variables of interest

URBAN WORKING POPULATION	RISK				
	MORPHOLOGICAL		CARDIOVASCULAR		LIFESTYLE-RELATED
<b>Males</b>	BMI, WC, BIA	Actual vs. perceived obesity	BP, TC, TG	Actual vs. self reported measures	DIET, PA, SMOKING, ACL CONSUMP & DEP
<b>Females</b>	BMI, WC, BIA		BP, TC, TG		DIET, PA, SMOKING, ACL CONSUMP & DEP

Where: BMI represents body mass index; WC: waist circumference; BIA: bioelectrical impedance analysis; BP: blood pressure; TC: total cholesterol; TG: total glucose; PA: physical activity; ACL CONSUMP: alcohol consumption and ACL DEP: alcohol dependence

## THE WORLD HEALTH ORGANISATION (WHO) STEP-WISE APPROACH

Because reliable information on chronic disease risks is important for identifying population groups which are at risk, for monitoring risk and, in turn, for reducing future burden of disease, (Strong & Bonita, 2004), the World Health Organisation (WHO) developed a surveillance tool called the STEP-wise approach (STEPs) – a methodological tool which has been recommended for the assessment and surveillance of CVD risk (Bonita *et al.*, 2001). With reference to the use of this tool within the South African setting, STEP's was developed as an 'entry point' for low and middle income countries to capture data and begin surveillance on CVD risks within different population groups. As such, the concept behind this tool is based on the idea that the use of standardised tools and methods of data collection allows comparability of data over time and across different locations (Bonita *et al.*, 2001). Globally, it therefore allows for cross-country comparisons to be made, and within South Africa itself, allows for comparisons between provinces to be made without methodological discrepancies hindering such comparisons.

Within the context of this research, the DHS (2003) made use of questionnaires based on this STEP-wise approach. To date this survey contains the *only available* information on CVD risk within Eastern Cape population groups, and therefore within this research project, use of a similar tool allows for comparisons to be made, in addition to ensuring reliability of the instrument within a similar population group (DHS 2003; Bonita *et al.*, 2001). A further advantage of the STEP-wise approach, is the fact that it allows for CVD risks to be assessed in fairly large samples of individuals, taking into account financial and logistical constraints, which often hinder the implementation of objective measures: While objective measures for the assessment of CVD risks are preferable, these are, for the most part, time consuming

and expensive and therefore least appropriate or applicable to large scale epidemiological studies or field-based research (Armstrong & Bull, 2006; Pitta *et al.*, 2006).

According to Bonita *et al.* (2001), a “risk factor” refers to “any attribute, characteristic, or exposure of an individual, which increases the likelihood of developing a non-communicable disease”. Based on, and using this definition, the STEP-wise approach proposes that “risk” be assessed according to the following three steps (Strong & Bonita, 2004; Bonita *et al.*, 2001): Step 1: questionnaire-based information; Step 2: standardised physical measurements; and Step 3: blood samples for biochemical analyses (Strong & Bonita, 2004; Bonita *et al.*, 2001).

### **Use of the steps approach in the current study**

Within the current study, Steps 1, 2 and 3 were utilised in the assessment of each risk factor. Although step 3 usually requires the use of blood samples for biochemical analyses, only the ‘pin prick’ method was used due to financial and logistical constraints associated with the use of blood samples when testing large groups. A summary of techniques used, within the STEPS framework is presented in Table III.

Table III: STEPS framework for the assessment of selected CVD risk factors including self-reports, objective measures & blood samples

<b>RISK</b>	<b>STEP 1: QUESTIONNAIRE-BASED</b>	<b>STEP 2: PHYSICAL MEASUREMENTS</b>	<b>STEP 3: BLOOD SAMPLES</b>
<b>Diet</b>	-24-hour dietary recall -Selected questions on habitual intake		
<b>PA</b>	-Adapted Global Physical Activity Questionnaire (GPAQ) -International Physical Activity Questionnaire (IPAQ)		
<b>TU</b>	-Adapted WHO STEP-wise surveillance questionnaire (WHO, 2001)		
<b>ALC CONSUMP. &amp; DEP.</b>	-Standard set of 8 questions proposed by the WHO for the Monitoring of Alcohol Consumption and Related Harm (DHS, 2003; WHO,2000) - CAGE questionnaire		
<b>Obesity</b>	-Zieblands figures on perceived body shape and size (Ziebland <i>et al.</i> , 2002)	-Stature & mass (from which BMI was calculated) -Waist circumference (WC) -Bioelectrical impedance analysis (BIA)	
<b>HYPTEN</b>	-self-reporting of hypertension	-Measured blood pressure	
<b>TIID</b>	-self reporting of type II diabetes		-Measured total 'non-fasting' blood glucose ('pin-prick' analysis)
<b>HYPCHOL</b>	-self reporting of hypercholesterolemia		-Measured total blood cholesterol ('pin-prick' analysis)

Where: PA represents Physical activity; TU: tobacco use; ALC CONSUMP: Alcohol consumption; ACL DEP: Alcohol dependence; HYPTEN: Hypertension; HYPCHOL: Hypercholesterolemia; and TIID: Type II diabetes

## MORPHOLOGICAL RISK

### OBESITY

Various measurements exist pertaining to the assessment of obesity. These include stature and body mass (from which body mass index is calculated), and waist circumference measurements (Baumgartner & Jackson, 1999). All are thought to be good indicators of body size and shape and therefore level of fatness and obesity (McArdle *et al.*, 2001). In addition to these measures, bioelectrical impedance analysis (BIA) has been deemed acceptable to provide a fairly accurate indication of body composition, findings of which can be compared

to normative values to give an indication of level of fatness or obesity (Houtkooper *et al.*, 1996).

### **Body Mass Index (BMI)**

BMI is a well known measurement in the assessment of fatness in relation to height, and, despite its limitations in terms of its relative inability to distinguish between fat and lean muscle, is accepted as one of the standard measures of obesity (Rush *et al.*, 2007). In addition to being applicable to field based research, due to its ease of application and calculation (Baumgartner & Jackson, 1999), it is thought to provide a reliable measure of 'normalcy' for an individual's body mass in relation to his or her stature (McArdle *et al.* 2001). Since both body mass and stature are taken into account, some indication is provided of the linearity or fatness of a subject in relation to height (Baumgartner & Jackson, 1999). Along with waist circumference, an estimation of body composition can also be inferred which, in turn, for the purposes of this research, was related to lifestyle factors.

The BMI guidelines proposed by the WHO (1995, 2000, 2004) provide an effective and comprehensive means of identifying and classifying overweight and obesity as well as associated co-morbidities (Table IV).

Table IV: BMI classification of overweight and obesity (WHO 1995, 2000, 2004)

MEASURE (kg.m <sup>-2</sup> )	CLASSIFICATION	RISK OF CO-MORBIDITIES
< 18.5	Underweight	Low
18.5-24.9	Normal range	Average
25.9-29.9	Overweight (Pre-obese)	Increased
30.0-34.9	Obese class I	Moderate
35.0-39.9	Obese class II	Severe
≥40.0	Obese class III	Very severe

### **Waist circumference (WC)**

It has been suggested that waist circumference, as a criterion for central fatness, is one of the strongest predictors of disease within population groups, and is a convenient indicator of



abdominal adipose tissue (Zhu *et al.*, 2002). Waist circumference (WC) measurements therefore help to identify those at risk of obesity (Punyadeera *et al.*, 2001b), and to predict subsequent coronary artery disease (Hickey, 2003). WC correlates well with intra-abdominal adipose tissue, tissue which is associated with increased metabolic abnormalities and health risks (Weinsier *et al.*, 2001; Punyadeera *et al.*, 2001b). In turn, a greater and more central distribution of body fat is associated with increased risk of various risk factors including type II diabetes, hypertension, obesity and Coronary Heart Disease (CHD) (Rush *et al.*, 2007). Specific to the South African context, research has shown that, in the abdominal region, black women tend to have less visceral fat (van der Merwe & Pepper, 2006) but increased subcutaneous abdominal tissue (Puoane *et al.*, 2002; Punyadeera *et al.*, 2001b). Some of the highest rates of abdominal obesity have in fact been found in urban black women, making waist circumference an important measurement and indicator of obesity, particularly within this population group (Puoane *et al.*, 2002).

Please note, that discussion regarding obesity within the contexts of WC and BIA measures and relating specifically to visceral and subcutaneous fat relies on the following definitions: *Total adipose tissue* refers to all adipose tissue in the body excluding that in the head, hands and feet, and bone marrow tissue; *Subcutaneous adipose tissue* refers to the layer of fat found between the dermis and fascia of the muscles; and *Visceral adipose tissue* refers to adipose tissue distributed in the three body cavities, namely *intrathoracic*, *intraabdominal* and *intrapelvic* (Shen *et al.*, 2003).

Although WC cut off's and classification vary according to various population groups, the WHO (2000) WC guidelines are based on numerous global studies and therefore provide useful criteria for the identification of individuals at risk (Table V).

Table V: Waist circumference guidelines (WHO, 2000) (James *et al.*, 2001; James, 2004).

RISK CLASSIFICATION	WAIST CIRCUMFERENCE (mm)	
	FEMALES	MALES
Above action level 1	≥ 800	≥ 940
Above action level 2	≥ 880	≥ 1020

## Body composition: Bioelectrical Impedance Analysis (BIA)

Bioelectrical impedance analysis (BIA) is accepted as a reliable measure of body composition: although not the most accurate, its appeal lies in its ease of use and thus applicability within the field (Dehghan & Merchant, 2008). BIA is defined as the opposition of a conductor to the flow of an alternating current (Dehghan & Merchant, 2008), and involves the application of a 50kHz current through the body, via electrodes which are placed on the hands and feet (Gray *et al.*, 1989). Impedance is measured as the amount of dropped voltage that occurs as the current passes through the body. Lean body tissue is rich in water and electrolytes and therefore poses minimal impedance. However, impedance increases to a maximum in the presence of fat/adipose tissue (Dehghan & Merchant, 2008). As a result, lean body mass and fat mass can be calculated from the difference in conductivity, and is further based on the principle that the resistance to this current is related to the volume of the conductor and the square of its length (Gray *et al.*, 1989), as expressed in the following equation (adapted from Houtkooper *et al.*, 1996):

$$V = p \times S^2/R$$

Where:

V = conductive volume (assumed to represent total body water or fat free mass)

P = specific resistivity of the conductor

S = stature (an estimate of the length of the conductor)

R = whole body resistance (measured with four surface electrodes placed on the wrist and ankle)

BIA is a non-invasive relatively inexpensive technique for the measurement of body composition, and due to its portability is ideal for field based research (Kyle *et al.*, 2004). Therefore, in the context of this research, BIA was used in order to assess the body composition of all participants. Specifically, it was used as a means of identifying abnormally high levels of body fat and thus provided an indication of level of obesity within subjects in comparison to normative data.

## CARDIOVASCULAR RISKS

### HYPERTENSION

Accurate measurement of blood pressure is essential in order to classify individuals and identify blood-pressure related risk (Pickering *et al.*, 2005). The auscultatory technique, which involves a trained observer using a mercury sphygmomanometer and the Korotkoff sound technique, represents the most accurate measurement technique, and represents the gold standard with regards blood pressure measurement (Pickering *et al.*, 2005).

Although various guidelines have been developed for the classification of hypertension, those proposed by the Joint National Committee on the Prevention, Detection, Evaluation and Treatment of High Blood pressure (JNC) appear to be the most conclusive since they were based on numerous studies and clinical trials on hypertension and hypertension management throughout the world (Chobanian *et al.*, 2003). The Seventh Report of the JNC (JNC 7) combined evidence from these studies, and simplified the classification of blood pressure levels in order to maximize guideline implementation. It therefore represents the most reliable and conclusive, yet simple set of guidelines available for the classification and diagnosis of hypertension (Wang & Wang, 2004). The JNC 7 defines hypertension as starting at 140/90 mm Hg in adults 18 years and older (Table IV). Classification is based on at least two seated measurements, properly measured with well-maintained equipment, and according to the following criteria (Pickering *et al.*, 2005; Milne & Pinkney-Atkinson, 2004).

Table VI: Hypertension classification (Adapted from JNC-7, Chobanian *et al.*, 2003)

SBP mmHg	DBP mmHg	BP CLASSIFICATION
< 120	< 80	Normal
120-139	80-89	Pre-hypertensive
140-159	90-99	Stage 1 hypertension
≥ 160	≥ 100	Stage 2 hypertension

BP indicates blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure

### HYPERCHOLESTEROLEMIA

The association between total cholesterol (TC) and CVD is known to be significant (Nauck *et al.*, 2002). Blood plasma cholesterol level is thought to be one of the strongest predictors for

the development of heart disease and is associated with many other cardiovascular risks, including hypertension, obesity and stroke (Sankaranarayanan, 1999).

Accurate and precise measurements of blood cholesterol are essential for appropriate and effective diagnosis of blood lipid disorders such as hypercholesterolemia (Nakamura *et al.*, 2003). When assessing blood cholesterol in the determination of cardiovascular risk, the different kinds of cholesterol within the body need to be considered. Low-density Lipoprotein (LDL) cholesterol is the main form of cholesterol in circulation, and has been shown to be primarily responsible for the association with chronic disease risk (Nauck *et al.*, 2002). Therefore, ideally, and for the most accurate results, both TC and LDL should be assessed in order to get an appropriate indication of level of risk (Warnick *et al.*, 1995). A blood test, in which a blood sample is drawn from the participant, represents the most accurate means of assessment, allowing one to assess TC, in addition to High-density Lipoproteins (HDL), LDL as well as triglycerides. However, due to financial and logistical constraints, this was not possible, and the 'pin prick' method was used as an alternative. Despite the reduced accuracy of this technique due to its inability to distinguish between the different cholesterol components in the blood, its appeal lies in its simplicity, ease of use and therefore applicability within the field setting (Bhatnagar & Durrington, 1993). This technique is also relatively non-invasive (in comparison to full blood samples) and allows for large numbers of subjects to be tested. Research has also shown that reflectron analysers are accurate enough in assessing total cholesterol and provide a simple and effective diagnostic tool for the identification of hypercholesterolemia (Reimer *et al.*, 1991).

High cholesterol was defined according to guidelines proposed in the Third Report of the National Cholesterol Education Programme (NCEP, Table VII) Expert Panel on the Detection, Evaluation & Treatment of High Cholesterol in Adults (Adult Treatment Panel III, 2002). These are the most recent guidelines to be developed. Based on numerous epidemiological studies, these guidelines serve to compile and interpret the available information of blood lipids and present this information in a simplified and clinically useful way, thus allowing for the identification and classification of hypercholesterolemia for research purposes and within clinical settings (WHO, 2005).

Table VII: Total cholesterol guidelines for the classification of hypercholesterolemia and associated risk. (Adapted from NCEP)

TOTAL CHOLESTEROL (mmol.L <sup>-1</sup> )	CLASSIFICATION
< 5.2	Desirable
5.2-6.2	Borderline High
> 6.2	High

## TYPE II DIABETES

Type II diabetes is a strong independent risk factor for CVD (Drucker *et al.*, 1983). Abnormally high levels of blood glucose provide an indication of type II diabetes, and the measurement thereof allows for estimations of cardiovascular risk according to increased blood glucose to be made.

As with blood cholesterol testing, the most accurate means of assessing blood glucose is in the laboratory via a blood sample (Drucker *et al.*, 1983). However, while accurate, this is expensive and time-consuming, and therefore not the most appropriate means of assessment in field-based research and when testing large numbers of individuals. The use of reflectance photometers provided a simple and accurate means of assessment in the context of this research (Drucker *et al.*, 1983).

The World Health Organisation (WHO, 2007) classifies people with type II diabetes according to the following criteria: individuals with a *fasting* plasma glucose concentration of >7.0mmol.L<sup>-1</sup>, or a postprandial (approximately 2 hours after main meal) plasma glucose concentration of >11.1mmol.L<sup>-1</sup>. These represent the most recent criteria and are based on numerous epidemiological studies which have been carried out worldwide (WHO, 2007).

Research has also suggested that different methods of assessment, in addition to an inability to control diet or liquid consumption prior to assessment, can lead to misinterpretation of data and misdiagnosis (D'Orazio *et al.*, 2006). Although attempts were made to only test individuals at least two hours after the ingestion of food, this was something which could not be absolutely controlled. For this reason, a conversion factor of 0.94 was used to give a measure of 12 mmol.L<sup>-1</sup> in order to approximate the equivalent of raised blood glucose. This was done in a similar study by Tibazarwa *et al.* (2009) who were unable to ascertain the time

of last ingestion for most participants and therefore used the same conversion factor. This follows the approved International Federation of Clinical Chemistry (IFCC) recommendation on reporting results for blood glucose (Tibazarwa *et al.*, 2009; D'Orazio *et al.*, 2006; Burnett *et al.*, 2001).

## **LIFESTYLE-RELATED RISKS**

### **DIET**

#### **24-hour dietary recall**

Diet remains one of the strongest predictors of cardiovascular risk factors, such as obesity, hypertension and type II diabetes (Boutayeb, 2006). Various techniques exist for the assessment of diet and habitual energy intake. These include food frequency questionnaires as well as the 24-hour dietary recall method (Thompson & Byers, 1994). Food frequency questionnaires require participants to recall all food and beverages consumed on a daily, weekly or monthly basis. While these questionnaires are accurate in that they reflect an extended period of time and therefore allow for more precise analyses of habitual diet, the limitation of this method is that individuals tend to over-report dietary intakes (Nel & Steyn, 2002; Bingham *et al.*, 1994). In comparison to this, the 24-hour dietary recall method, which requires participants to recall all food and beverages consumed over a 24 hour period, is limited in that it is only a reflection of one day rather than habitual dietary intake. It also tends to under-report dietary intake (Nel & Steyn, 2002). However, despite its limitations, studies have shown that the 24-hour dietary recall compares well with weighted records as well as food frequency questionnaires, especially when repeated measures are used (Bingham *et al.*, 1994). Due to logistical and time constraints, repeated measures were not possible in this research project. However, in addition to carrying out a 24-hour recall, additional questions regarding the habitual intake of various foods and beverages were carried out. These were validated and based on similar questions in the Adult Health Questionnaire used in the 2003 DHS (DHS, 2003). This recognises the importance of understanding the frequency at which certain types of foods and beverages are habitually consumed (Bingham *et al.*, 1994). Additional advantages associated with the use of the 24-hour dietary recall method are its speed, simplicity and ease of application. The interview approach associated with this technique allows individuals to respond verbally and thus partly eliminates potential literacy barriers, which may hinder the accuracy of items recorded. The immediacy of this technique

also ensures accuracy, since most subjects are able to recall, with a fair amount of accuracy, all they have consumed in the last 24 hours (Thompson & Byers, 1994).

## **PHYSICAL ACTIVITY**

The link between physical activity and cardiovascular risk is a significant one, based on a dose-response relationship (Joubert *et al.*, 2007b; Walker *et al.*, 2001). The regular participation in physical activity (recommended to be 30 minutes of moderate intensity exercise, five times a week), be it during leisure or work time, is associated with a reduction in cardiovascular risk, while the opposite is true for individuals who take part in little or no exercise (WHO, 2007; Wolf *et al.*, 1994).

There are various methods which exist pertaining to the measurement of physical activity. In the past, exercise tended to be defined as a 'planned, structured' exercise session aimed at improving physical health (Joubert *et al.*, 2007b), and thus objective tools of measurement were the norm. These include calorimetry, the Doubly Labelled Water (DLW) technique, pedometers and accelerometers amongst others. These techniques and tools allow for the accurate assessment of energy expenditure (Laporta *et al.*, 1985). However, the former two techniques are expensive and time-consuming, while the latter two are logistically difficult to implement in field based research. In addition, more recently it has been recognized that physical activity happens equally in various domains of life, and thus self-reported measures of time spent within different lifestyle domains of activity have become more pertinent, particularly in large-scale epidemiological research (Joubert *et al.*, 2007b). Physical activity questionnaires have therefore become an increasingly used tool for the assessment of habitual physical activity, and have the advantage of being inexpensive, and easy to apply within the field setting (Pitta *et al.*, 2006; Laporta *et al.*, 1985).

### **Physical activity questionnaires**

The accuracy and reliability of physical activity questionnaires, however, rests on various factors: Information needs to be accurately perceived and recalled by the subject; the design of the questionnaire needs to be considered such that responses are accurate – questionnaires with an interval response for example tend to show higher self-reported amounts of physical activity when compared to 'open' questions; and finally individual and

population characteristics need to be considered, such as age, cultural factors and cognitive capacity (Pitta *et al.*, 2006). Various validated physical activity questionnaires have been developed in the past. These include the Baecke Physical Activity Questionnaire, Follick's Diary, the Minnesota Leisure Time Physical Activity Questionnaire (MLTPAQ), the Physical Activity Scale for the Elderly (PASE), the Zutphen Physical Activity Questionnaire (ZPAC) and the Stanford Seven-Day Physical Activity Questionnaire, amongst others (Pitta *et al.*, 2006; Florindo & Latorre, 2003 and Baecke *et al.*, 1982). These vary in reliability and have been tested on a range of different population groups, however, none specifically on lower income populations, typical of developing countries such as in South Africa.

### **Global Physical Activity Questionnaire (GPAQ)**

The International Physical Activity Questionnaire (IPAQ) has been validated in both rural and urban South African population groups (Jourbert *et al.*, 2007b). The Global Physical Activity Questionnaire (GPAQ) was developed by the WHO as an instrument to assess physical activity patterns in *developing* country contexts, as part of a project aimed at monitoring risks for chronic disease in developing countries (Armstrong & Bull, 2006). During its development numerous existing physical activity questionnaires were reviewed and scrutinized in terms of their usefulness and application within developing countries. Following this, research was carried out to test the reliability and validity of GPAQ in a diverse set of countries, particularly those with low levels of education (South Africa was included in this research). Results found the questionnaire to be applicable to developing country populations (Armstrong & Bull, 2006). For this reason the GPAQ was used within this research project, in order to assess the physical activity levels of participants.

### **TOBACCO USE**

Tobacco use represents a strong and modifiable risk factor for chronic disease (Striegel-Moore *et al.*, 2006). There are various means of assessing smoking or tobacco use, including the number of people who use the product, as well as the amount of tobacco consumed. Within South Africa tools for monitoring tobacco use countrywide have only been in place for a few years (Swart & Panday, 2010). Since 2004, questions on tobacco use have been included within household surveys – surveys which were conducted twice a year on blacks 18 years and older until 1996 (Swart & Panday, 2010). These surveys defined smoking as



the smoking of one or more cigarettes, pipes or cigars per day. All Media and Product Surveys (AMPS) have also been carried out annually within the adult population of South Africa (all individuals over 16 years). According to these surveys, smokers are defined as individuals who spend money on cigarettes. Since 1998, smoking status has also been included within mortality/death data, allowing for an indication of tobacco-related mortality to be assessed (Swart & Panday, 2010).

In 1998 the first Demographic and Health survey was carried out, and this provided a platform for the assessment of tobacco use throughout different population groups and across different provinces of the country. Tobacco-related questions included were based on and derived from the WHO guidelines for the controlling and monitoring of the tobacco epidemic (Swart & Panday, 2010; DHS, 2003; Chopra *et al.*, 2002; Bradshaw & Steyn, 2001). Similar questions were used again in the 2003 DHS, however these were adapted to form a more rigorous questionnaire based on the STEPS programme proposed by the WHO (2001). Research has highlighted the applicability and advantages of these questions, in providing a useful tool for the assessment of tobacco use. This is due to the fact that questions used are standardised, which therefore allows for cross-country and indeed, cross-provincial comparisons to be made (Nawi *et al.*, 2006). Herein, the importance of using such a tool in surveillance assessments of individuals is vitally important, especially with regards to monitoring changes in population groups over time, associated with lifestyle and behavioural changes.

## **ALCOHOL CONSUMPTION AND DEPENDENCE**

In addition to smoking, alcohol abuse has also been associated with increased CVD risk (Rimm, 1996), with research indicating that those who drink large amounts of alcohol have higher death rates, as well as higher mortality from all causes of CVD (Chopra *et al.*, 2002). For this reason, it was deemed important to assess alcohol consumption and compare and relate consumption to CVD risk factors. Of the methods developed for the assessment of alcohol consumption, self reports are widely used because that they are, for the most part, reliable and have increased applicability for field-based research (Del Boca & Darkes, 2003). While various questionnaires have been developed, the AUDIT questionnaire developed by the WHO represents one of the most widely used and accepted questionnaires (Babor *et al.*, 2001). Not only has it been tested and validated on a range of different population groups,

but it is recognized as the only screening tool designed specifically for international use. It allows to comparisons to be made between countries and for trends within different population groups to be identified. Furthermore, the questionnaire itself is simple and therefore applicable within a wide range of settings. The questionnaire has also been validated across a range of different cultures internationally (Babor *et al.*, 2001). However, despite the wide applicability of the WHO AUDIT questionnaire across different population groups, the applicability of it is predominantly as a *screening tool*. The STEP-wise questionnaire developed by the WHO for the surveillance of non-communicable diseases (Bonita *et al.*, 2001) is a more appropriate tool for the assessment of habitual alcohol intake and therefore has a wider range of applicability. Other questionnaire methods focus on how much and how often individuals drink (Quantity-Frequency questionnaires) or on how often individuals drink specified amounts in one day (Graduated Quantity-Frequency questionnaires); the 7 Days method requires individuals to complete a 'diary' illustrating how much alcohol consumption they have consumed over the previous 7 days (WHO, 2000). The STEP-wise questionnaire however, encompasses *all* of these aspects. It includes questions on habitual alcohol consumption, with regard to both present and past consumption (within the last week, 30 days, 12 months and lifetime), as well as frequency of alcohol use (Bonita *et al.*, 2001). Perhaps most pertinent to the current research project is the fact that alcohol consumption within the latest national Demographic and Health survey (2003) was assessed using a set of questions which was based on the WHO STEP-wise questionnaire and proposed by the WHO for the Monitoring of Alcohol Consumption and Related Harm (2000). These questions have therefore been tested and validated on Eastern Cape individuals, which allows for comparisons with current data to be made.

As regards risky drinking, various tools have also been used for the assessment of alcohol dependence, defined as a 'cluster of behavioural, cognitive and physiological phenomena' which can develop following repeated use of alcohol (Babor *et al.*, 2001). The assessment of alcohol dependence is important in identifying individuals and population groups at risk – something which is particularly important with regard to research on cardiovascular health due to the association between alcohol dependence and abuse and CVD risk (WHO, 2000). The CAGE questionnaire is known to serve as a screening instrument for possible alcohol dependence (Schneider *et al.*, 2007). CAGE represents "Cutting down", "Annoyance by criticism", "Guilty feeling" and "Eye-openers". Similarly, the CAGE questionnaire was also

used in the 2003 DHS, and therefore has been validated not only nationally, but within different population groups within the Eastern Cape.

## **ETHICAL CONSIDERATIONS**

Prior to testing, the study protocol was approved by the Ethical Standards Committee of the Department of Human Kinetics and Ergonomics, Rhodes University. Detailed information regarding the risks as well as benefits involved in the study was also submitted, in addition to clarity on issues of confidentiality, anonymity and informed consent (Appendix 1). All participant data were coded and all individuals remained anonymous. Any potential risks involved as part of the study were clearly explained, and accounted for, while benefits to the participants included feedback on their health status and risk of cardiovascular disease.

## **PILOT RESEARCH AND TRAINING**

Prior to commencement of testing, all test procedures were performed on a random sample to ensure accuracy and precision of techniques and methods, as well as efficacy of the investigators. This included the completion of the questionnaires, as well as the 24-hour dietary recall. All testing procedures, including anthropometric measures of stature, mass and waist circumference were taken. Bioelectrical impedance analysis was also carried out, in addition to the assessment of blood pressure, glucose and cholesterol. As the majority of the participants tested were mother-tongue Xhosa speakers, both questionnaires were translated into isi-Xhosa and completed by a group of Xhosa speaking individuals. Data from the pilot testing of the questionnaires was captured and edited to ensure that it was entered and stored correctly for further analysis.

Five research assistants (all Masters Students in the same field of research) were trained to assist with the data collection: each assistant was provided with an overview of the questionnaires as well as the design and objectives of the study, and was involved in two training workshops centered on familiarization with the questionnaires and the administration of an interview, as well as guidance on gathering anthropometric and cardiovascular measurements. Following this each research assistant was required to interview and collect physical measures on two individuals representative of an urban black population group (one male and one female). They were then required to re-test these individuals to ensure that

data obtained was similar. This method of testing and then retesting individuals was done to ensure that data was obtained and recorded correctly, that questions were asked and interpreted appropriately, and that physical measures were carried out according to standardised procedures so that the readings recorded would be accurate and reliable. Pilot testing allowed for the research assistants to familiarise themselves with all equipment and test procedures before the data collection began.

## **EXPERIMENTAL PROCEDURES: EQUIPMENT AND MEASUREMENT PROCEDURES**

### **MORPHOLOGICAL RISK**

#### **OBESITY**

##### **Stature**

As measurements were taken *in situ*, the stature of all participants was obtained using a portable stadiometer, which was placed up against a wall. During measurement, subjects were required to remove shoes and stand upright against the wall with head erect and facing forwards. Stature was measured from the floor to the vertex in the mid-sagittal plane, to the nearest 0.1cm (Figure 5).



Figure 5: Male subject having his stature measured by one of the research assistants

## **Body Mass**

A portable Seca scale was used for the assessment of body mass. During measurement participants were required to wear light clothing and remove their shoes. Readings were taken to the nearest 0.5kg.

## **Body Mass Index (BMI)**

BMI was determined from measurements of stature and body mass using the following formula:  $\text{body mass (kg)} / \text{stature (m)}^2$ .

## **Waist circumference (WC)**

Standing at the side of the participant, and with a tape measure held horizontally to the body, waist circumference was measured midway between the costal margin and the anterior superior iliac spine. Subjects were asked to relax, and breathe in and out a few times before the measurement was taken.

## **Body composition: Bioelectrical Impedance Analysis (BIA)**

Measurement involved the placement of surface electrodes on the right side of the body: two on the dorsal surfaces of the hands and feet proximal to the metacarpal-phalangeal and metatarsal-phalangeal joints respectively, and two between the distal prominences of the radius and ulna, and between the medial and lateral malleoli at the ankle. Measurement was taken with participants supine, arms slightly abducted and legs separated so they were not in contact with one another using a LipoTrak<sup>TM</sup> bioelectrical impedance analyzer.

## **PERCEPTUAL RESPONSES**

### **Body perception illustrations**

As a final consideration, individuals were asked to answer three questions about their perceptions of their current body shape and size. Individuals were given pictures of eight different body shapes, with 1 representing 'bone thin' and 8 representing 'morbidly obese'

(see Appendix 2). Participants then had to identify (a) which body shape reflected their current shape and size most accurately, and b) which body shape represented their 'ideal'. These pictures were taken from a study done by Ziebland *et al.* in 2002 and were also used in the Cape Area Panel study in 2006.

## **CARDIOVASCULAR (CV) RISKS**

### **HYPERTENSION**

#### **Blood pressure measurement: The auscultatory technique**

Blood pressure measurements were taken after participants had been seated for ten minutes and using a stethoscope and mercury column sphygmomanometer (Figure 6) (McArdle *et al.*, 2001). Participants were asked to remain as relaxed as possible to eliminate the possibility that anxiety or physical exertion would influence the measurement. The blood pressure of each participant was measured three times on the left arm. If the second blood pressure reading (both systolic and diastolic) differed by more than 5mmHg, the first reading was excluded. Systolic readings were only accepted if they were  $\geq 80$ mmHg and at least 15mmHg higher than diastolic. The mean systolic and diastolic blood pressure from two separate readings was recorded (Bradshaw & Steyn, 2001).

#### **Medical Conditions Questionnaire**

In addition to the physical measurement of blood pressure, self-reported hypertension was also assessed (Appendix 2). This was done by means of structured questions in which individuals were asked if they had ever been told by a health professional that they were hypertensive. In general, research has found few discrepancies between reported and actual values for hypertension (Kehoe *et al.*, 1994)



Figure 6: Blood pressure measurement of a male participant

## **HYPERCHOLESTEROLEMIA**

### **Blood cholesterol measurement: the 'pin-prick' method**

Total blood cholesterol of subjects was recorded using an Accutrend ® Plus reflectance photometer (© 2007, Roche Diagnostics). This instrument is applicable for professional use, and allows for the measurement of both glucose and cholesterol. A lancing device was used to prick the side of the fingertip, from which the drop of blood formed was applied to a test pad. In order to ensure accuracy and eliminate the possibility of extraneous factors affecting the blood sample, the first drop of blood to form on the fingertip was wiped away and the second drop used for analysis. The test pad was then inserted into the photometer for analysis. As with blood pressure, measurements were taken with individuals seated and relaxed.

## **TYPE II DIABETES**

### **Blood glucose measurement: the 'pin-prick' method**

Abnormally high levels of blood glucose provide an indication of type II diabetes, therefore the total blood glucose of all participants was assessed. As with blood cholesterol, blood glucose was measured using an Accutrend® Plus reflectance photometer. A lancing device was used to prick the side of the finger: once the first drop of blood formed was wiped away, a reading was taken by placing the second drop of blood onto a test pad which was inserted into the photometer for analysis. The measurement range of the photometer ranged from 1.1 to 33.3mmol.L<sup>-1</sup> for blood glucose and between 3.88 and 7.76mmol.L<sup>-1</sup> for cholesterol.

### **Medical Conditions Questionnaire**

Self reported prevalence of both hypercholesterolemia and type II diabetes was also assessed (Appendix 2). Individuals were asked structured questions about whether they had been told by a health professional that they had either of these conditions – this, order to gain an understanding of self-reported prevalence of these conditions in comparison to measured values. The appropriate terminology was used within the questionnaire to ensure that participants understood the various concepts discussed: the terms 'blood fat' and 'blood sugar' were used to describe hypercholesterolemia and type II diabetes – these both represent terms used by African individuals to describe these conditions (Appendix 2).

## **LIFESTYLE RELATED RISKS**

### **DIET**

#### **24-hour dietary recall**

24-hour dietary recall was used to assess the habitual energy intake of individuals over a period of 24 hours. This method requires that participants record everything they eat for a period of 24 hours. Thereafter an interview is carried out during which time participants are required to verbally confirm specific quantities and types of foods consumed. Visual aids of various typically South African food types were also included to assist with understanding, explanation and accuracy of foodstuffs recorded (see Appendix 2).



## **Dietary Intake Questionnaire**

Individuals were also asked various additional questions regarding the type of foodstuffs habitually consumed by participants, how often these foodstuffs were consumed, as well as the amount of salt used on food. These questions were included in order to gain an understanding of the types of foodstuffs habitually consumed (Appendix 2).

## **FoodFinder**

Dietary intake and macronutrient composition for each subject were analysed using the software programme *FoodFinder 3* for Windows <sup>TM</sup> (Microsoft Corporation). This involved entering data recorded into the programme, following which dietary intake was analysed.

## **PHYSICAL ACTIVITY**

### **Global Physical Activity Questionnaire (GPAQ)**

Individuals were asked various questions on habitual physical activity. These were based on and formed part of the Global Physical Activity Questionnaire (GPAQ) (also included within the WHO STEP-wise questionnaire). Questions included related activity at work, activity involved in getting to and from places, and finally activity related to recreational activities. Therefore, an overall indication of physical activity was provided, based on duration and intensity, as well as according to different lifestyle domains. For example:

- 1) *Occupation-related* physical activity during a *usual week* and *usual day*
- 2) *Travel –related* physical activity during a *usual week* and *usual day*
- 3) *Non-work related* and *leisure time* physical activity during a *usual week* and *usual day*.

Physical activity levels were categorised according to a scoring system in which volume of activity is computed by weighting each type of activity by its energy requirements. This was computed by using METs (multiples of the resting metabolic rate), to derive a score in MET-minutes: calculated by multiplying a MET score by minutes performed. 1 MET-minute represents 3.5ml of oxygen per kg of body weight per minute (GPAQ). Data were therefore collected as a continuous measure and reported as median MET-minutes computed for

walking (W), moderate-intensity activities (M), and vigorous-intensity activities (V) within each domain (work, transport, and leisure). Physical activity was calculated using the following MET values (Table VIII):

Table VIII: Domain-specific MET classification (GPAQ)

DOMAIN	MET VALUE
Work	Moderate MET value = 4.0 Vigorous MET value = 8.0
Transport	Walking MET value = 4.0
Leisure	Moderate MET value = 4.0 Vigorous MET value = 8.0

Physical activity was defined according to the definitions proposed by the International Physical Activity Questionnaire (IPAQ) Scoring Committee, which defines overall reported activity as follows (Table IX):

Table IX: Classification of levels of physical activity (adapted from IPAQ classification of physical activity)

CATEGORY	CRITERIA
1: Low/Inactive or insufficiently active	< 600 MET-minutes/week
2: Moderate/Minimally active	600 up to 3000 MET-minutes/week
3: High/Sufficiently active	> 3000 MET-minutes/week

Within all physical activity questions and criteria, only activity lasting longer than ten minutes was included within analyses. Furthermore, maximum duration ‘cut-off points’ were put in place, similar to those devised and used in the DHS (2003). Any individual who reported physical activity which exceeded the indicated ‘cut-off’ was excluded from final analysis. This is due to the known fact that individuals tend to over-report when self reporting level of physical activity (DHS, 2003). “Cut-off points” were defined as follows (Table X):

Table X: “Cut-off points” for the classification of self reported physical activity data (DHS, 2003)

DOMAIN	TIME
Occupational	720 minutes of work time 480 minutes of moderate or vigorous occupational activity
Leisure	240 minutes of vigorous activity 480 minutes of moderate activity
Transport	300 minutes of transport time
Sedentary	720 minutes of inactive time (sitting, sleeping)

## TOBACCO USE

### Adapted STEP-wise questionnaire

Questions on tobacco use were completed in order to ascertain smoking prevalence amongst participants. These were based on, and adapted from, the STEP-wise questionnaire developed by the WHO (2001). Questions included related to past and current smoking prevalence, as well as opinions regarding exposure to environmental tobacco smoke at home and at work, as well as exposure to dust and fumes.

Based on these questions, smokers were classified according to the following criteria: One tobacco equivalent (containing approximately one gram of tobacco) was defined as: one manufactured cigarette; one hand rolled cigarette; one pipe smoked; one cigar; one cheroot; one cigarillo.

Smokers were classified as those individuals who reported that they currently smoked either occasionally or daily. Individuals were further classified as light smokers if they smoked 1-14 tobacco equivalents per day, or heavy smokers if they smoked  $\geq 15$  tobacco equivalents per day (Bradshaw & Steyn, 2001).

Individuals were also assessed with regard to their exposure to environmental tobacco smoke (ETS), both at the work place as well as at home. In addition to this, exposure to

environmental fumes in the workplace was also assessed, and the number of years of exposure recorded.

## **ALCOHOL CONSUMPTION AND DEPENDENCE**

### **Adapted questions for the assessment of alcohol use; The CAGE questionnaire**

Alcohol use and dependence was assessed by means a set of questions based on those used in the DHS AHQ (2003), and adapted from a questionnaire designed by the WHO to monitor and assess alcohol use (WHO, 2000). Through the use of these questions (see Appendix 2), information was obtained regarding frequency of use, as well as quantities of alcohol consumed each day during a typical week. Individuals were considered risky drinkers if they consumed over five drinks per day (in the case of males) or three (in the case of females) (Schneider *et al.*, 2007). In addition, the CAGE questionnaire was used, which is based on the following 4 questions.

*'Have you ever felt that you should cut down on your drinking?'*

*'Have you been annoyed by being criticised for drinking?'*

*'Have you ever felt guilty about drinking', and*

*'Have you ever had a drink first thing in the morning?'*

Individuals who answered affirmatively to two or more questions were classified as being alcohol dependent (Schneider *et al.*, 2007).

## **STUDY POPULATION**

The study population was limited to *black* males and females from an *urban working* population in Grahamstown, Eastern Cape. This was in cognizance of the fact that different population groups face different disease risks according to demographic location, socio-demographic status, ethnicity and sex; and the fact that province specific research, particularly within the Eastern Cape, has to date been limited. Subjects were recruited from various 'working' populations throughout the Grahamstown area, including institutions, as well as local businesses within the greater urban area.

## **VOLUNTEER CHARACTERISTICS**

Volunteers were recruited according to racial group, health status and age. In addition to limiting the study sample to black urban individuals, only individuals who were not pregnant or lactating, or on any treatment for chronic diseases (like hypertension and type II diabetes) were included. It is acknowledged that this may have created a certain bias within the sample, and potentially lead to an under-estimation of the prevalence of chronic disease risks within this population, however, for the purposes of this research, the aim was to control for as many variables as possible so as to ensure that similar comparisons between male and females individuals could be made. Although onset of menopause can often occur at the age of 50 years, it was decided to include females up to 55 years so as to ensure a large enough sample representative of an urban black population. As there was no significant difference in any of the risk factors (morphological, CV or lifestyle-related) between females under and over the age of 55 years ( $p>0.05$ ), females up to that age were included in the analyses. Only individuals over 18 years were assessed, due to the ethical considerations associated with testing individuals under the age of 18, and the fact that most working individuals are over 18 years. Subjects were age matched (mean age = 41.6 years), with males demonstrating a mean age of 40.5 years and females, 42.6 years.

## **SAMPLE SIZE**

A statistical power analysis was carried out in order to determine the sample size for this study. In accordance with this a total of 291 participants were assessed: 148 females and 143 males. This was deemed sufficient to yield a statistical power of 90%.

## **EXPERIMENTAL PROCEDURES: PHASE 1**

Since the central aim of this project was to assess cardiovascular risk within the Grahamstown area of the Eastern Cape, the priority was to find enough subjects residing in the greater urban area of Grahamstown who were both willing and able to participate in the study, and fulfilled the requirements for inclusion.

The initial stage of the project therefore involved contacting local businesses and institutions within the Grahamstown area. In early 2010, various meetings were set up, during which time detailed information pertaining to the project was given to managers and supervisors in each

instance. This included information regarding the aims of the project, the requirements of participants, as well as any aspects of the project which might benefit the participants.

Following these initial meetings, all supervisors were asked to distribute the project information to their employees. Posters with additional information and contact details of the researcher were also put up around each working area. In addition, information sessions with various employees were held, in which more information was given, and any questions answered. Participants were recruited on a voluntary basis: any interested black individuals (fulfilling the necessary criteria) were introduced to the research concept and were given written and verbal information about the background and aims of the study. Thereafter, the personal particulars of those interested in taking part were collected by the supervisors concerned.

Follow up meetings with each supervisor were held, in which the names of the volunteers for the project were collated. Logistics with regard to when and where testing could take place were also discussed. Following careful assessment of the volunteers' work schedules, it was decided that two testing sessions per day would be held: Session 1 in the morning, from 9:00-12:30, and session 2 in the afternoon, from 15:00-17:30. These times were decided upon for the following reasons: individuals would not have consumed food or drink for at least two hours prior to testing, in addition to the fact that these testing times gave individuals at least two hours to get settled into their work routine before testing commenced. This was important from a work perspective, in that taking a few individuals each hour to be tested was less disruptive once work teams had been given a bit of time to get work processes in place.

Since many of the individuals who had volunteered for the project were Xhosa speaking, a mother-tongue Xhosa speaking individual was recruited to assist with translation and interpretation during testing. Two meetings were set up during which time she was introduced to the research topic, given a thorough explanation of the project, as well as details pertaining to data collection and procedures to be followed. She was given the questionnaires to translate into isi-Xhosa, as well the 24-hour dietary recall sheet, and various visual aids to be used to assist with the explanation of concepts. These were duly translated into isi-Xhosa and made available to subjects during the testing protocol.

## **EXPERIMENTAL PROCEDURES: PHASE 2**

Data collection commenced and lasted for a period of two months. It took place at the participants' place of work, where a private room was allocated to the researchers. Participants were assigned testing times. On arrival, each participant was introduced to the researcher and research assistants. A maximum of three participants per hour were assessed, each in a different area of the testing room. This was to ensure the privacy of those being tested and to make sure each individual felt as comfortable as possible. Assessment of each individual took about an hour to complete, following which they returned to their work duties as normal. Although no financial remuneration was given, the benefits associated with increased awareness of issues of health and wellness, were emphasized as incentives to encourage individuals to take part in the study, and feedback workshops will take place early next year.

Although the participants had received information and a thorough explanation of the project during the recruitment phase, time was again taken prior to testing to explain the background to the project, as well as the testing procedures. Individuals were given time to ask any questions, following which, if still happy with the protocol, they signed informed consent documents (see Appendix 1). The interpreter was present at all times during testing. Although not involved in the actual data collection, she was in the room to assist with translation and explanation where necessary.

To start with, the questionnaires on self-reported CVD risks, tobacco use and alcohol consumption were completed, along with the perceptual questions and the 24-hour dietary recall. These were carried out randomly. The decision was made to administer the questionnaires first, to ensure that by the time the physical measures were taken, that individuals were rested and relaxed. This is particularly important for the assessment of blood pressure, as any anxiety or physical exertion prior to measurement can impact on readings recorded and thus hinder the accuracy of findings.

Participants were given verbal feedback as each physical measure was assessed. This was to ensure that subjects remained informed about the protocol and remained comfortable throughout. Once all physical measures had been completed, individuals were thanked for

their time, and were reminded of their subject codes, which were used to ensure the anonymity of those tested.

## **DATA ANALSYES**

All results were analysed using the Statistica software package Version 8 (Statistica®, Statsoft Inc). Descriptive statistics were carried out initially in order to obtain mean and standard deviation responses for all variables, as well as to test for normality and homogeneity of the variances. Independent T-tests were conducted on all variables in order to determine any significant differences between males and females with regard to all variables assessed. In instances where homogeneity of the variances between males and females failed, non-parametric tests (Mann-Whitney) were carried out. For variables which contained response data (from questionnaires) and percentages as opposed to continuous numerical measurements, chi-square tests (McNemar for dependent samples and Pearson for independent samples) were carried out to test for any significant differences in responses between males and females for all variables. Correlation analyses were also carried out to assess for any significant relationships between different variables. All statistical hypothesis tests were set at a 5% level of significance.



## CHAPTER IV

### RESULTS

The aim of this chapter is to compare CVD risk profiles of black males and females forming a working population in the urban area of Grahamstown. Risk factors have been organised into three categories: Obesity is classified as a 'morphological risk'; hypertension, hypercholesterolemia and type II diabetes are grouped as 'cardiovascular (CV) risks', and finally, diet, physical activity, tobacco use, and alcohol consumption and dependence, are categorised as 'lifestyle-related risks'. Initially background information about the subject pool is presented, following which CVD risks are assessed and compared. Finally self reported and perceived risks are compared to actual measures.

In addition to being 'urban working' individuals, participants were also age-matched and thus there was no significant difference ( $p>0.05$ ) in age between males and females. Males had a mean age of 40.5 ( $\pm 10.8$ ) years and females a mean age of 42.6 ( $\pm 8.1$ ) years (Table XI). In terms of background demographic information, 82.4% of males and 87.4% of females had electricity at home, and most participants were educated (97% of individuals had been to school). Males had completed up to an average of Grade 10 and females Grade 11 (Table XI).

Table XI: General background characteristics of male and female participants

	FEMALES	MALES
Age (mean $\pm$ standard deviation)	42.6 ( $\pm 8.1$ )	40.5 ( $\pm 10.8$ )
Electricity at home (%)	82.4	87.4
Been to school (%)	97	97
Highest level of school completed (Grade)	11	10

## MORPHOLOGICAL RISK

### OBESITY

#### Stature, mass and Body Mass Index (BMI)

With the exception of stature, females demonstrated significantly higher values for both mass and BMI (Table XII). Males were significantly ( $p<0.0001$ ) taller than the females with a mean stature of 1671 ( $\pm 60.1$ ) mm in comparison to 1567 ( $\pm 63.6$ ) mm within the female sample (Table XII). In contrast to this, females were significantly ( $p<0.0001$ ) and over 20.5kg heavier than their male counterparts (72.1  $\pm$ 16.4 kg).

Table XII: Mean ( $\pm$  standard deviation) anthropometric and morphological characteristics of participants

MEASURE	FEMALES	MALES
Stature (mm)	1567.0 ( $\pm$ 63.6)	1671.0 ( $\pm$ 60.4)*
Mass (kg)	92.7 ( $\pm$ 22.1)	72.1 ( $\pm$ 16.4)*
BMI (kg.m <sup>-2</sup> )	37.6 ( $\pm$ 8.2)	25.7 ( $\pm$ 5.5)*
WC (mm)	1023.4 ( $\pm$ 184.0)	887.4 ( $\pm$ 120.7)*
BF (%)	51.8 ( $\pm$ 8.3)	26.8 ( $\pm$ 6.8)*
BF(kg)	48.8 ( $\pm$ 17.7)	20.2 ( $\pm$ 9.9)*
LBM (%)	48.8 ( $\pm$ 7.9)	73.1 ( $\pm$ 6.6)*
LBM (kg)	43.9 ( $\pm$ 6.4)	52.1 ( $\pm$ 8.5)*

\* denotes significant difference ( $p<0.0001$ )

Where: BMI represents body mass index; WC: waist circumference; BF: body fat; LBM: lean body mass

Not surprisingly, females had significantly ( $p<0.0001$ ) higher BMI values (Figure 7), with a mean BMI of 37.6 ( $\pm$ 8.2) kg.m<sup>-2</sup>. Thus the female sample as a whole was classified as class II obese ('morbidly obese'). In comparison, with a mean BMI of 25.7 ( $\pm$ 5.5) kg.m<sup>-2</sup> the males were slightly over the 'normal' classification and were classified as overweight (Figure 7).

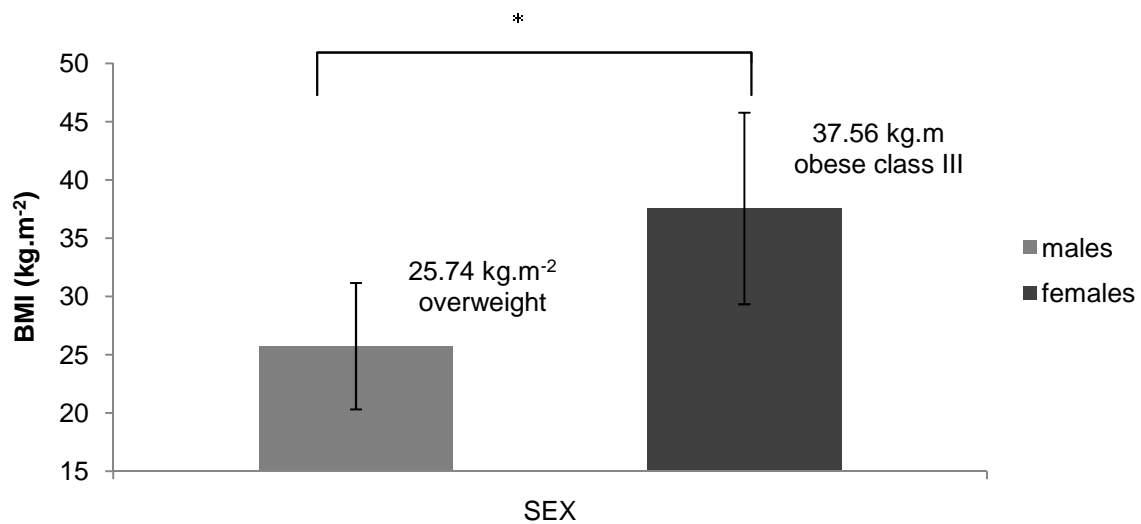


Figure 7: Mean ( $\pm$ standard deviation) BMI classification of male and female participants

Overall BMI classifications for females were significantly ( $p < 0.0001$ ) higher than those for males. While just over half (55%) of the male individuals presented with a BMI within the 'normal' range ( $18.5\text{--}24.9\text{kg.m}^{-2}$ ), only 7% of females fell within this range. In contrast, 39% of females were classified as obese class III, with a BMI greater than  $40\text{kg.m}^{-2}$ . This was the case in only 1% of males. In total, 93% of females were overweight/obese, in comparison to 45% of males (Figure 8).

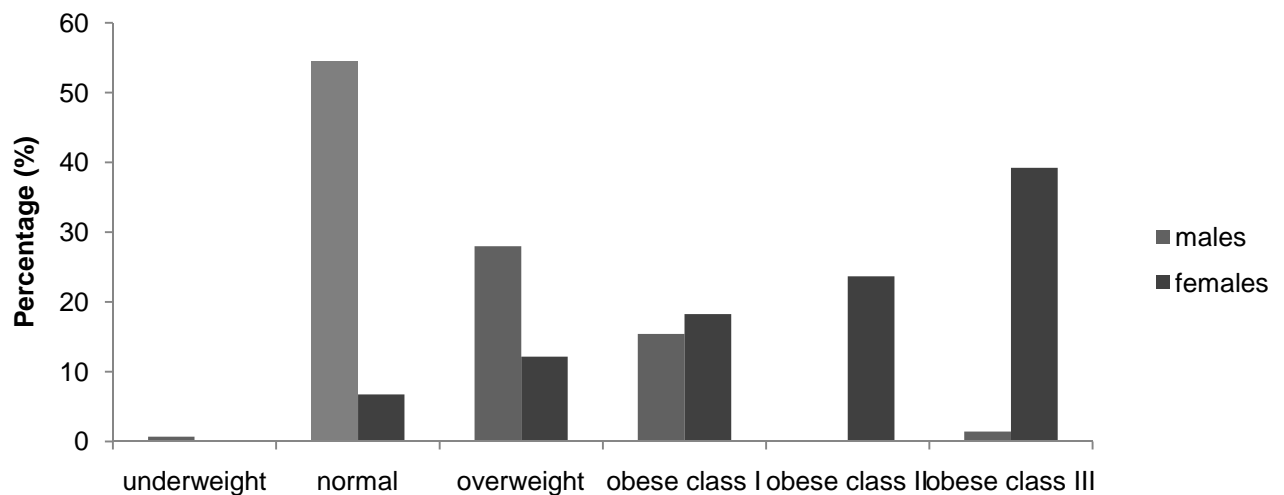


Figure 8: Percentage of males and females falling within different BMI classifications

## Waist circumference (WC)

Particularly noteworthy was the volume of central adiposity prevalent in the female sample. This was evident in the significantly ( $p < 0.0001$ ) larger waist circumferences ( $1023.4 \pm 184$  mm) of the females, compared to the males ( $887 \pm 120.7$  mm): a 136mm difference (Figure 9).

According to WC cut-off criteria, this categorises females above risk category 2, while males fall below category 1 and therefore fall into the 'no risk' category (Figure 9). This difference was significant ( $p < 0.0001$ ).

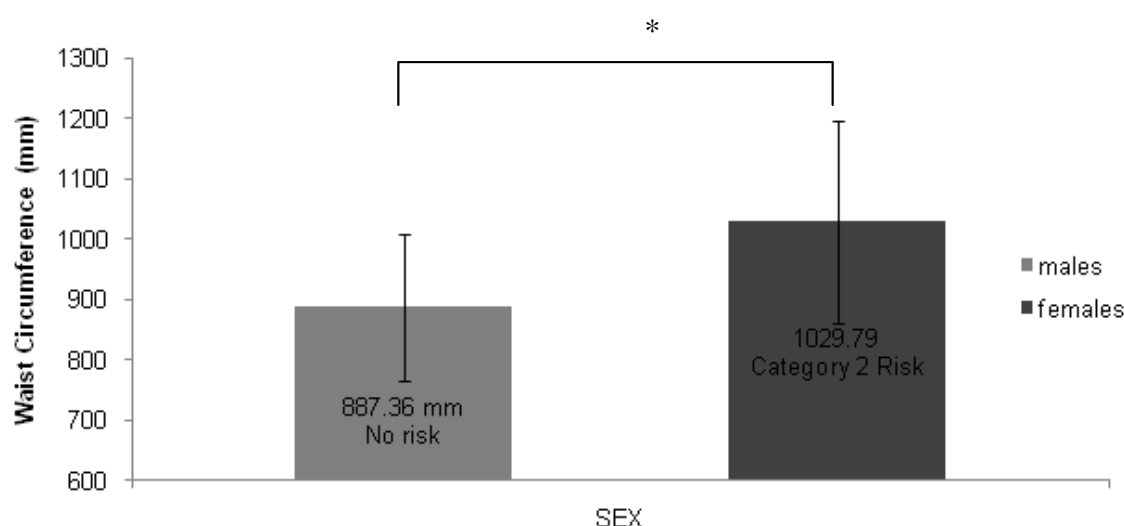


Figure 9: Mean ( $\pm$  standard deviation) waist circumference of males and females

Most males fell within the normal WC category with 74% classifying below action level 1 of risk ( $< 940$ mm). In contrast to this, only 9% of the females fell below this category ( $< 800$ mm). In addition, while only 15% of males had values over 1020mm, representing action level 2 risk, 82% of females fell within this category ( $> 880$ mm) (Figure 10).

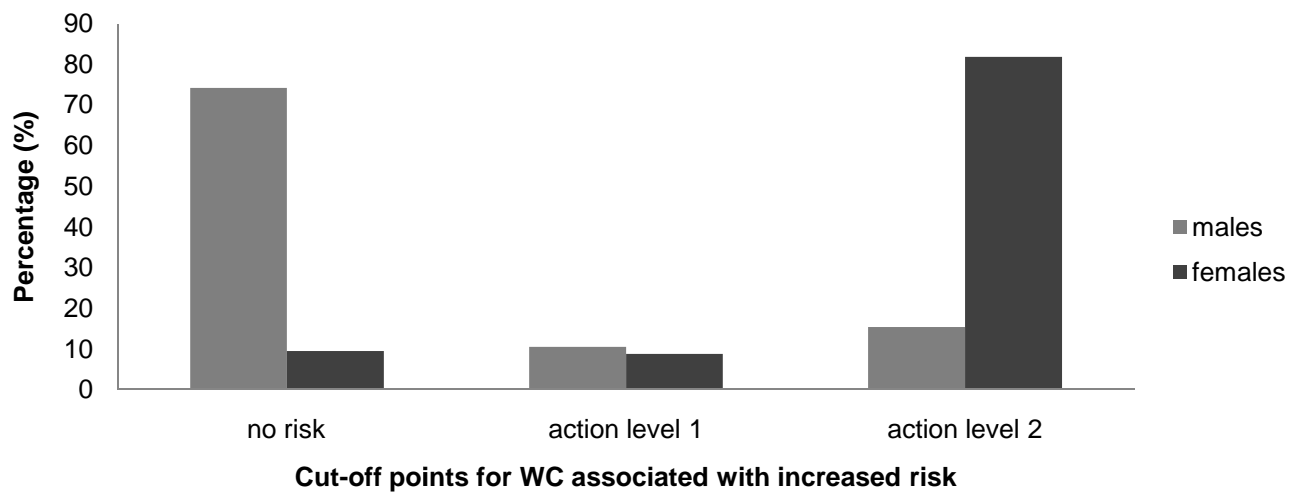


Figure 10: Classification of WC according to cut off points: percentage of males and females within each category

### Body Composition

The female sample had significantly ( $p < 0.0001$ ) higher body fat percentage (Figure 11). In the case of the females, 51.8% body mass was comprised of fat, 25% more than the male sample which demonstrated a mean body fat percentage of 26.8%. Accompanying this, females had significantly ( $p < 0.0001$ ) less lean body mass (48.8%) compared to their male counterparts (73.1%).

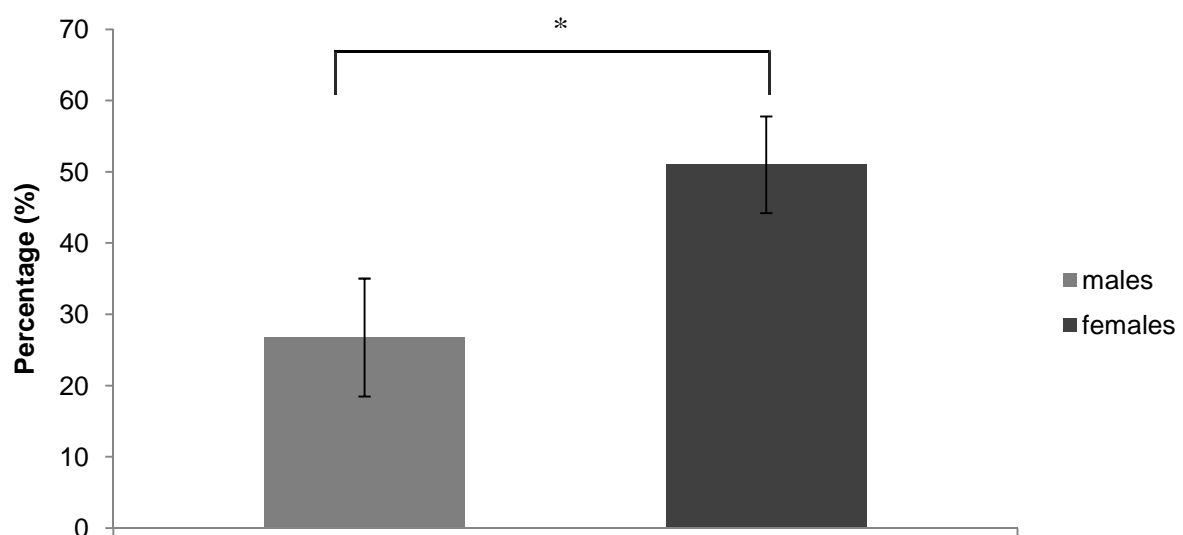


Figure 11: Mean ( $\pm$  standard deviation) percentage body fat of male and female participants

Therefore, encompassing both fat and lean body mass, the body composition profiles of male and female participants were very different (Figure12), with a greater a share of body mass within the female sample made up of fat mass (48.84kg), while lean body mass comprised more than half (52.05kg) of the body mass within the male sample (Figure 12).

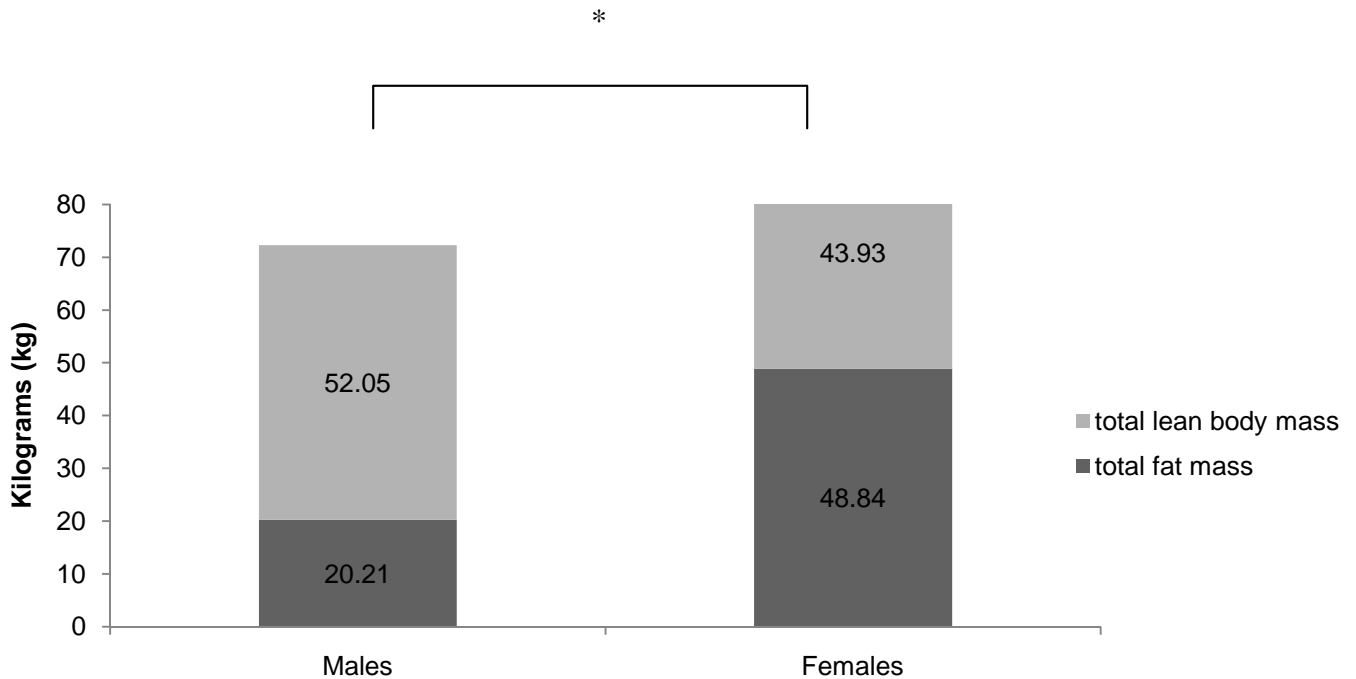


Figure 12: Total lean and fat mass of male and female participants

## CARDIOVASCULAR (CV) RISKS

Although females demonstrated slightly higher values for all CV risks, these were not significant ( $p > 0.05$ ) (Table XIII).

Table XIII: Mean ( $\pm$  standard deviation) cardiovascular (CV) measures of participants

MEASURE	FEMALES	MALES
SBP (mmHg)	132.9 ( $\pm 19.3$ )	130.6 ( $\pm 15.5$ )
DBP (mmHg)	84.9 ( $\pm 12.6$ )	82.3 ( $\pm 12.4$ )
Total blood glucose (mmol.L <sup>-1</sup> )	4.3 ( $\pm 2.1$ )	4.0 ( $\pm 1.2$ )
Total blood cholesterol (mmol.L <sup>-1</sup> )	4.4 ( $\pm 0.9$ )	4.3 ( $\pm 0.9$ )

Where: SBP represents systolic blood pressure; DBP: diastolic blood pressure; \* represents  $p < 0.05$

# BLOOD PRESSURE

While mean blood pressure was similar in both samples, both diastolic and systolic responses were slightly higher within the female sample (Table XIII). Although more females classified as ‘Normal’ according to their systolic response, the opposite trend was apparent with regard to their diastolic response (Figure 13). Furthermore, while a higher percentage of males classified as ‘Pre-hypertensive’ (according to both SBP and DBP), and ‘Stage I hypertensive’ (SBP) (Figure 13a) significantly ( $p<0.05$ ) more females classified with ‘Stage II hypertension’ (SBP and DBP) ( $\geq 160/100\text{mmHg}$ ) (Figure 13b).

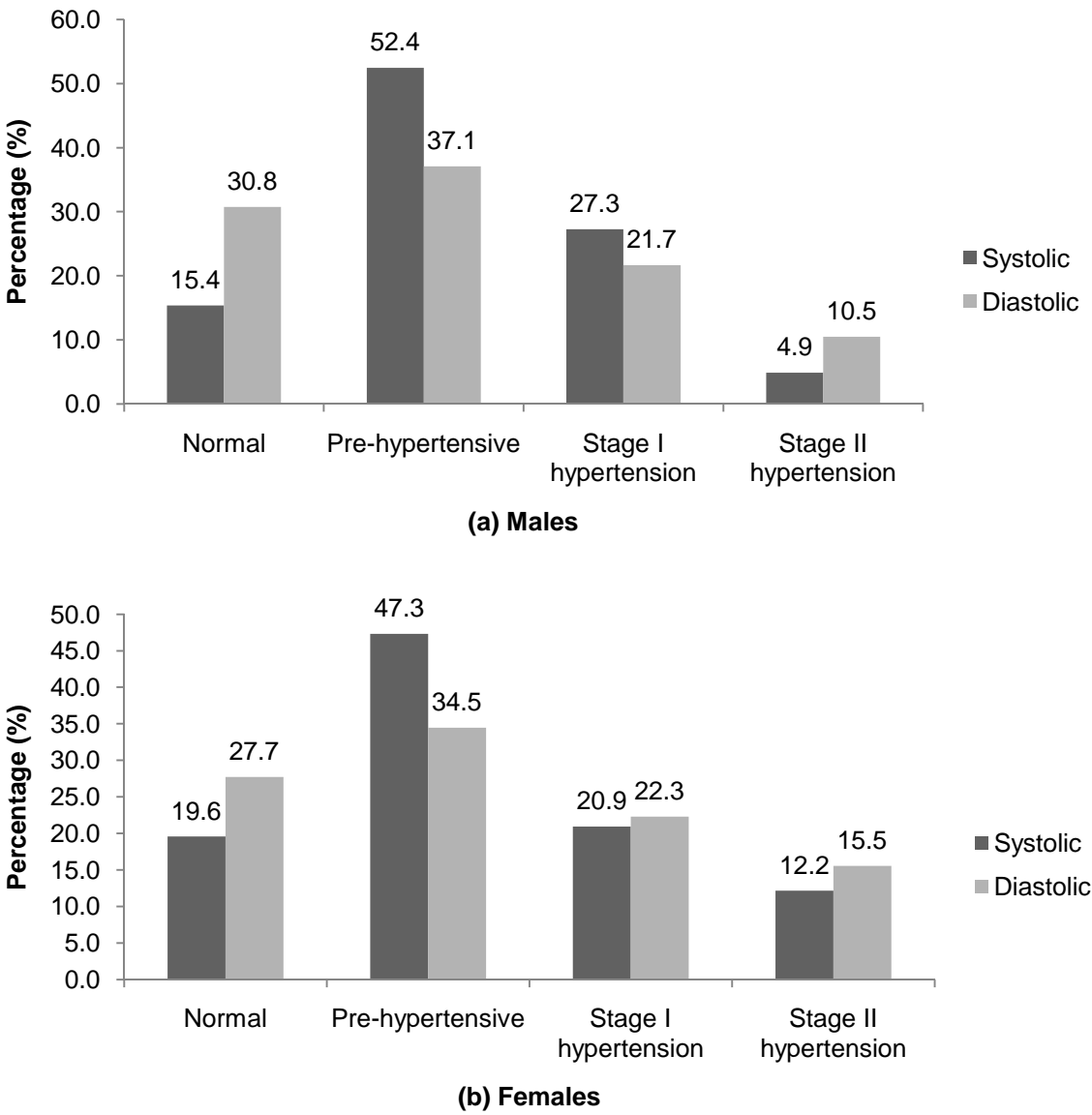


Figure 13: Classification of hypertension in male (a) and female (b) participants

## TOTAL BLOOD CHOLESTEROL

The prevalence of hypercholesterolemia was notably low in both males and females. With a mean total cholesterol of  $4.43 (\pm 0.9) \text{ mmol.L}^{-1}$  (females) and  $4.26 (\pm 0.9) \text{ mmol.L}^{-1}$  (males), both groups fell within the 'desirable' range (classified as anything lower than  $5.2 \text{ mmol.L}^{-1}$ ). However, the standard deviations for both samples were high, indicating a high degree of inter-individual variation (Figure 14).

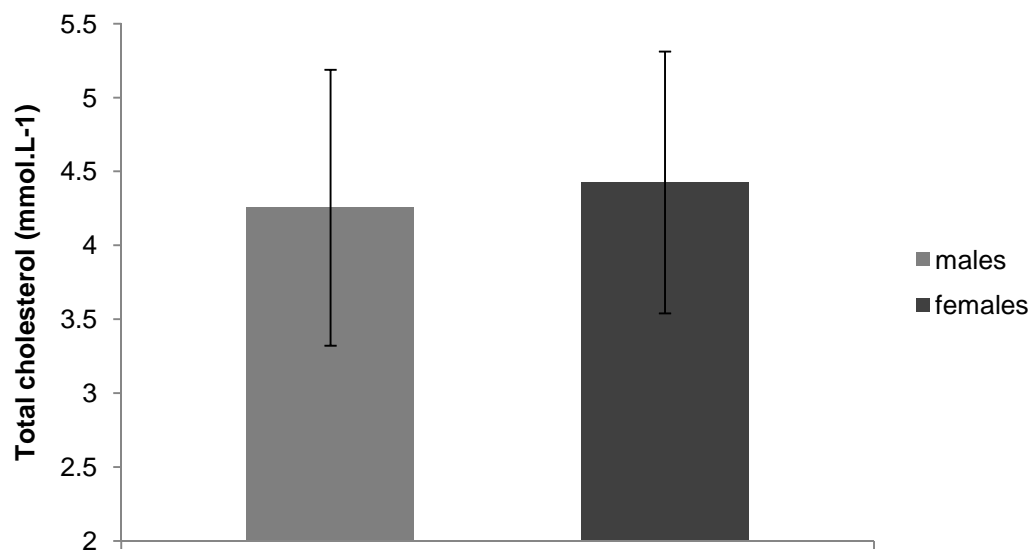


Figure 14: Mean ( $\pm$  standard deviation) total cholesterol of male and female participants

However, despite the fact that the majority of individuals had desirable blood cholesterol levels, 12% of females and 9% of males had cholesterol readings that were borderline high ( $5.2\text{-}6.2 \text{ mmol.L}^{-1}$ ), while 3.4% and 2.1% of females and males had high total cholesterol ( $>6.2 \text{ mmol.L}^{-1}$ ) (Figure 15).



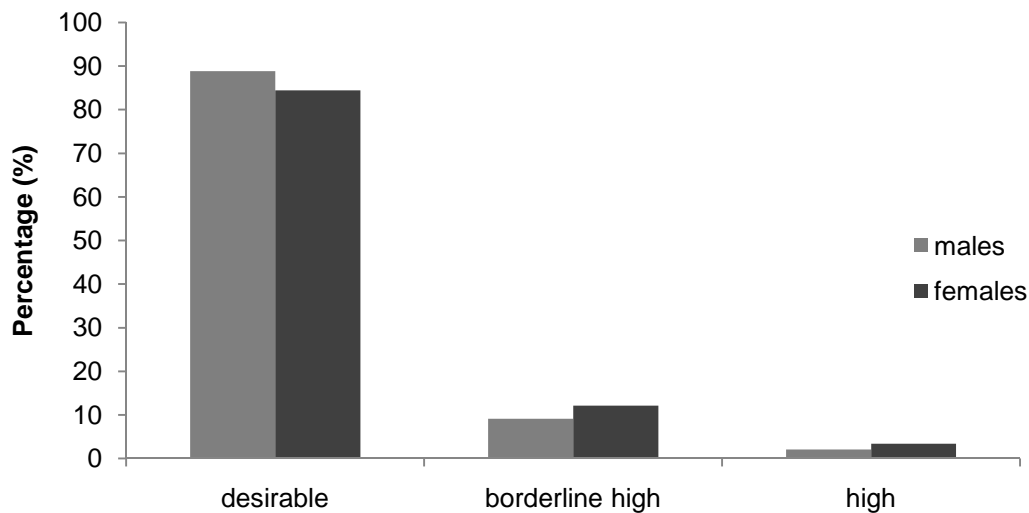


Figure 15: Total cholesterol classification of male and female participants

## BLOOD GLUCOSE

Prevalence of type II diabetes was notably low amongst both male and female participants (Figure 16). No males, and only 3% of females, were measurably Type II diabetic. Females recorded a mean, non-fasting blood glucose reading of  $4.3 (\pm 2.1) \text{ mmol.L}^{-1}$  and males,  $4.0 (\pm 1.2) \text{ mmol.L}^{-1}$ , placing both groups within the 'normal' range. However, as with total cholesterol levels the standard deviations within both groups were high. This was particularly notable within the female sample, where a large amount of variance was evident (Figure 16).

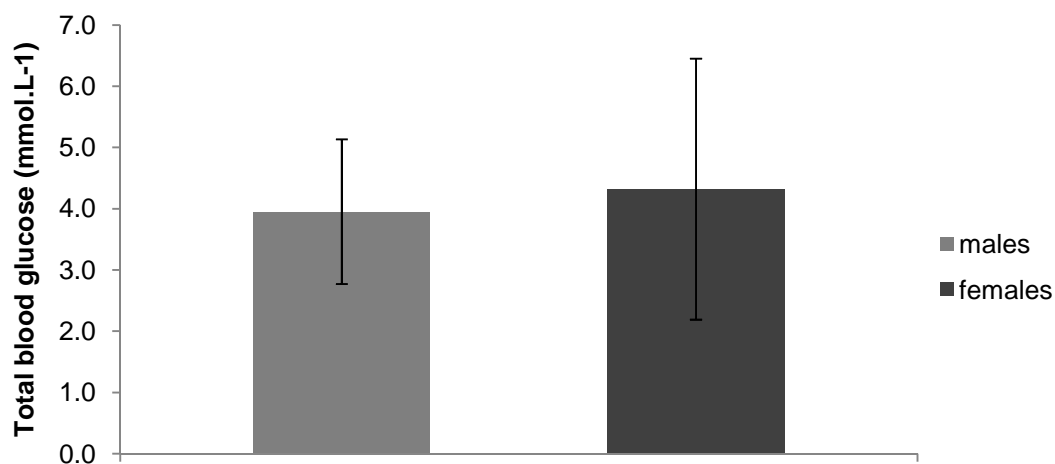


Figure 16: Mean ( $\pm$  standard deviation) total non-fasting blood glucose of male and female participants

## LIFESTYLE-RELATED CHARACTERISTICS

In contrast to the morphological and CV measures, males presented with significantly higher values for all lifestyle-related variables (Table XIV). They reported significantly ( $p<0.05$ ) higher levels of physical activity and consumed significantly ( $p<0.05$ ) more total energy (kJ). In addition, a significantly ( $p<0.01$ ) higher percentage of the male sample smoked, consumed alcohol and reported alcohol dependence.

Table XIV: Self reported lifestyle-related risks

MEASURE	FEMALES	MALES
Physical activity (MET-minutes/week)	2659.57 ( $\pm 1592.7$ )	3315.26 ( $\pm 1774.3$ )*
Diet: Total energy intake (kJ)	7233.859( $\pm 3467.2$ )	9023.62 ( $\pm 3761.1$ )*
Smoking (%)	3.4	51.1**
Alcohol consumption (%)	46.6	73.4**
Alcohol dependence (%)	33.3	40*

\* denotes significant difference ( $p<0.05$ ), \*\* denotes significant difference ( $p<0.01$ ); kJ refers to kilojoules

## PHYSICAL ACTIVITY

### Total physical activity

Males were significantly ( $p<0.05$ ) more active than females (Figure 17), taking part in a mean total of 3315.26 ( $\pm 1774.3$ ) MET-minutes of activity per week, in comparison to females who undertook 2659.57 ( $\pm 1592.7$ ) MET-minutes weekly (Figure 17). However, the standard deviations within the sample as a whole were relatively high, indicating a large variation in physical activity levels (Figure 17).

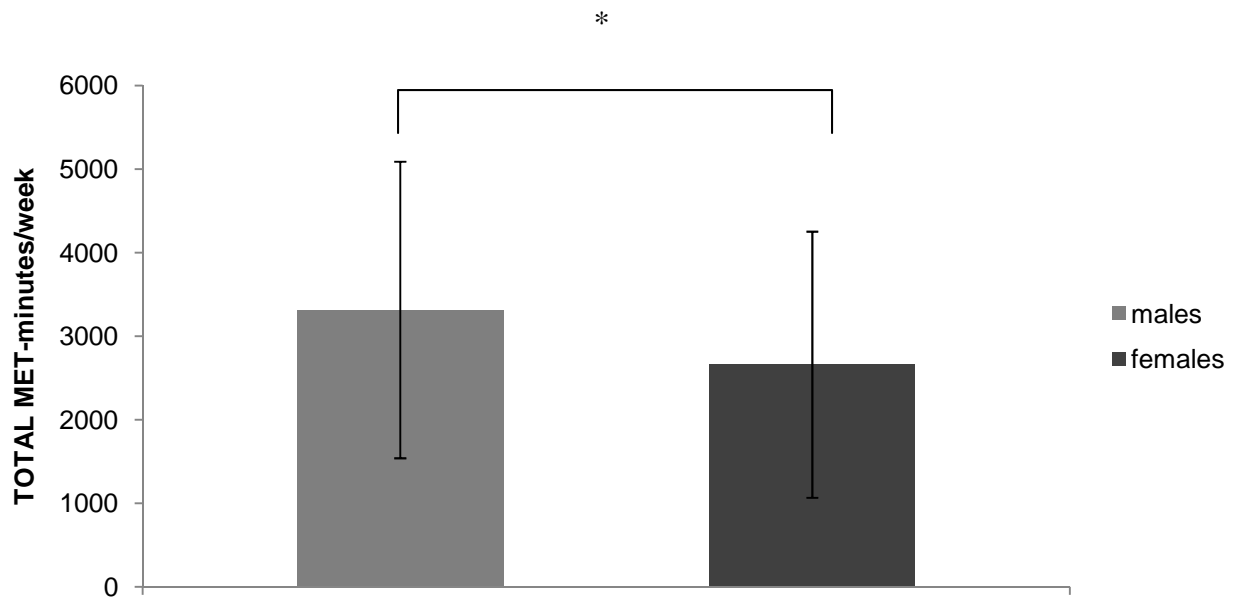


Figure 17: Mean ( $\pm$  standard deviation) total physical activity (MET-minutes/week) of male and female participants

Sufficient physical activity was defined in terms of those individuals expending >3000 MET-minutes per week. According to these criteria, 51.3% of males and 42.4% of females were sufficiently active, while 43.2% and 44.5% of females and males respectively took part in 'minimal' activity (Figure 18). In contrast, 14.4% and 4.2% of females and males respectively, were inactive, taking part in less than 600 MET-minutes of activity per week (Figure 18).

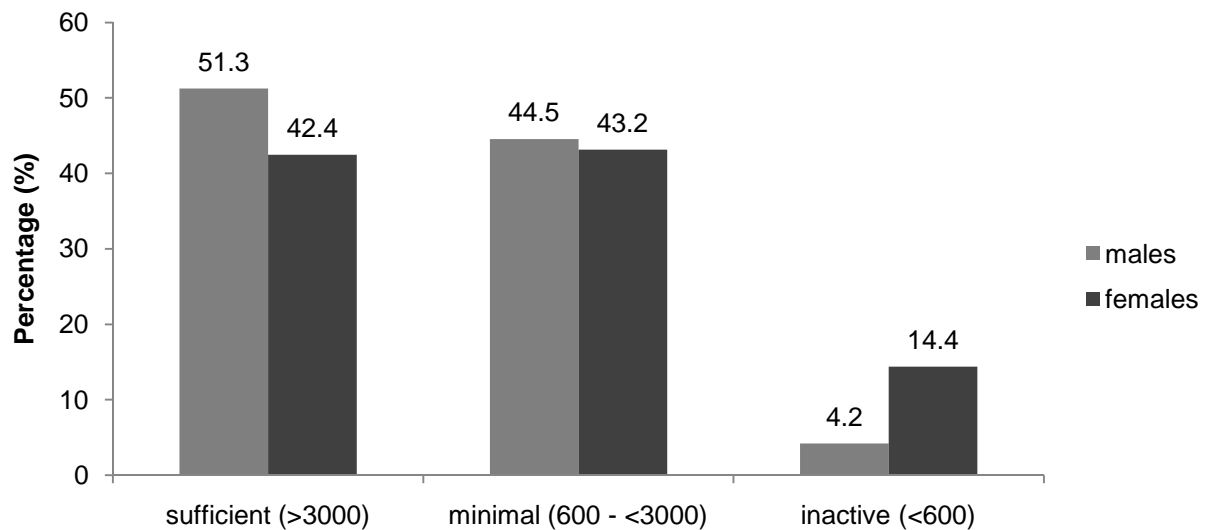


Figure 18: Classification of physical activity for male and female participants

The significantly ( $p < 0.05$ ) higher total physical activity (MET-minutes per week) demonstrated in the males, was for the most part related to the intensity of the activity. Despite the fact that females were active on more days of the week (4.45 days per week compared to 3.91 days for the males) and took part in slightly more 'moderate' activity weekly (3 hours 16 minutes compared to 3 hours 3 minutes for males), males reported significantly ( $p < 0.005$ ) higher levels of *vigorous* activity compared to females (a mean total of 65 minutes of vigorous activity,  $2.6 \pm 2.8$  days a week, compared to 21 minutes,  $1.4 \pm 2.3$  days per week by females). There was however a large amount of inter-individual variation, evidenced in the high standard deviations within both samples (Figure 19).

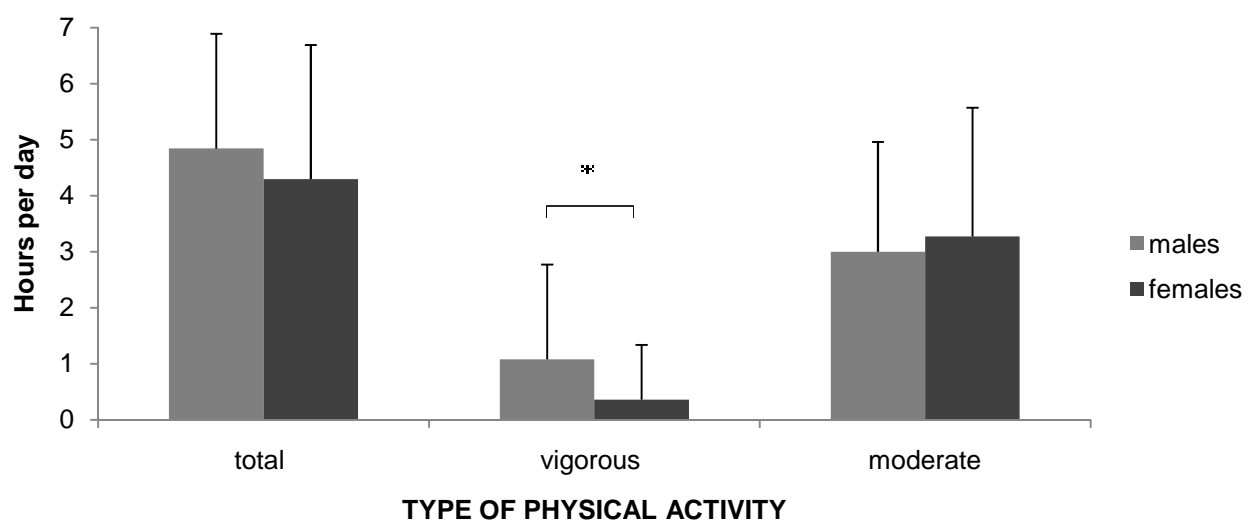


Figure 19: Mean ( $\pm$  standard deviation) total, vigorous and moderate activity of male and female participants per day

### Work-related physical activity

It was evident that for both samples, the most physical activity took place in the work place, and hence contributed the most to total weekly activity (Figure 20). Among the females, work-related physical activity accounted for 69% of total daily physical activity (179 minutes), and in males 76% (221 minutes). Males took part in significantly ( $p < 0.05$ ) more days of *vigorous* activity (mean total of 1.9 days of vigorous activity at work, compared to 1.1 days by female participants) and for significantly ( $p < 0.05$ ) longer per day (57 minutes per day versus 19 minutes for females). Males were also more *moderately* active, taking part in 163 minutes of moderate activity, compared to 159 minutes by the females (Figure 20).

**Leisure-time physical activity**

Second to activity in the workplace, leisure activity contributed 17% of total activity in the female sample and 15% in the male. In contrast to work-related activity, females were more active at home than males, taking part in significantly ( $p<0.0001$ ) more days of *vigorous* activity (2.7 days versus 0.6 days for males). Despite this, exercise duration within both samples was similar, with females only taking part in 6.4 minutes of activity per day and males, 7.4 minutes. There was therefore no significant difference in the mean total MET-minutes of vigorous activity per week between the samples (Figure 20).

Females also engaged in significantly more *moderate* activity at home: taking in part in activity significantly ( $p<0.0001$ ) more regularly (2.2 days per week versus once a week in males) and for significantly ( $p<0.05$ ) longer duration (36 minutes per day versus 19 minutes for males). Accordingly females expended significantly ( $p<0.01$ ) more MET-minutes of activity per week ( $1029.9 \pm 1125.7$  minutes versus  $550.1 \pm 823.7$  minutes for males). The standard deviations for both samples were high, again indicating a high degree of variance within individual participants (Figure 20).

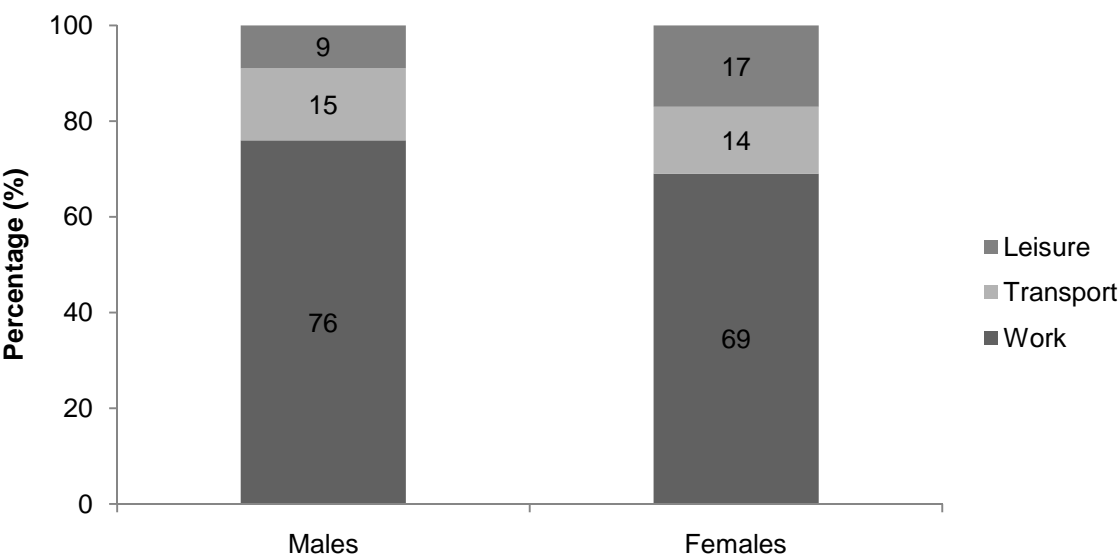


Figure 20: Percentage contributions of different domains of activity to total daily physical activity in female and male participants

## Transport-related physical activity

Since walking appeared to be the main form of transport for the majority of individuals, this was defined as 'transport-related physical activity'. In terms of volume of activity, there were very few differences between male and female participants, with walking contributing 14% and 15% of total activity in females and males respectively (Figure 20). Out of a seven day week, while both samples walked on four days of the week, females walked for 42 minutes, compared to males who walked for 37 minutes. Males therefore expended slightly more MET-minutes related to walking, however this was not significant (1200 MET-minutes for males versus 1053 MET-minutes for females).

## DIET

Males consumed significantly ( $p < 0.0001$ ) more kilojoules ( $9003.9 \pm 1043$  kJ) per day than females ( $7193 \pm 3277$  kJ) (Figure 21).

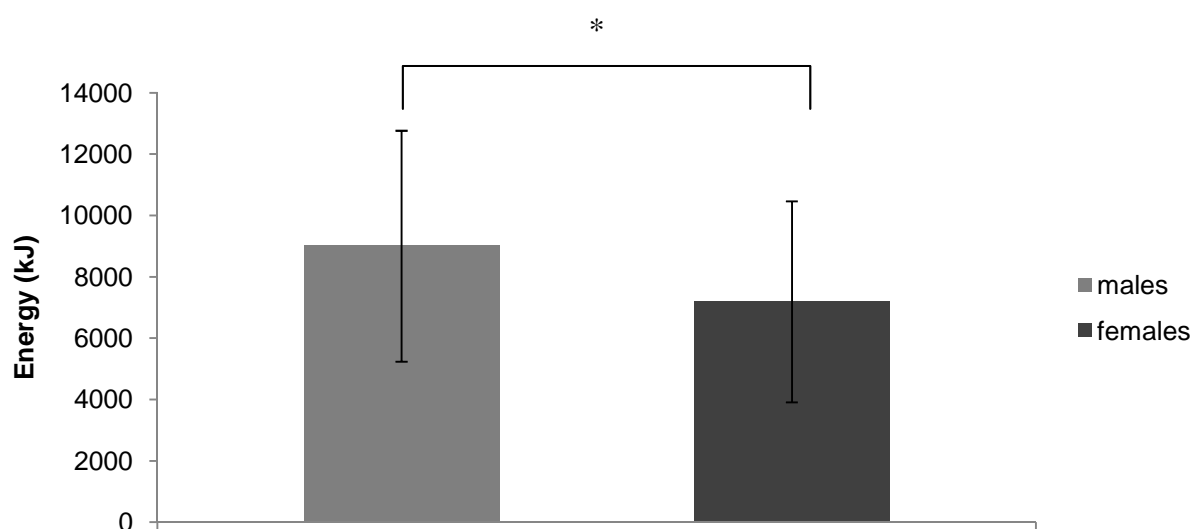


Figure 21: Mean ( $\pm$  standard deviation) total energy intake (kJ) of male and female participants

In addition, males consumed significantly ( $p < 0.0001$ ) more total protein (including animal and plant protein), carbohydrates (CHO), starch, total dietary fibre, and cholesterol (Table XV).

Table XV: Selected dietary components (mean  $\pm$  standard deviation) consumed by males and females

	FEMALES	MALES
<b>TOTAL PROTEIN</b>	59.7 ( $\pm$ 28.7)	84.1( $\pm$ 52.9)**
<b>Animal protein (g)</b>	32.2 ( $\pm$ 23.5)	52.3 ( $\pm$ 49.8)**
<b>Plant protein (g)</b>	18.75( $\pm$ 11.2)	30.1 ( $\pm$ 17.1)**
<b>CARBOHYDRATES (CHO) (g)</b>	211.1 ( $\pm$ 99.2)	264.3 ( $\pm$ 107)**
<b>Starch (g)</b>	27.5 ( $\pm$ 41.6)	57.7 ( $\pm$ 62.8)**
<b>TOTAL FAT (g)</b>	62.1 ( $\pm$ 41.6)	71.0 ( $\pm$ 45.8)
<b>Total dietary fibre (g)</b>	16.8 ( $\pm$ 8.9)	20.9 ( $\pm$ 13.8)*
<b>Cholesterol (mg)</b>	197.2 ( $\pm$ 160.4)	279.6 ( $\pm$ 264.4)*
<b>Total sugar (g)</b>	62.4 ( $\pm$ 37.1)	51.4 ( $\pm$ 38.2)*
<b>Added sugar (g)</b>	68 ( $\pm$ 49.4)	56.4 ( $\pm$ 41.2)*

\*denotes  $p < 0.05$ , \*\* denotes  $p < 0.01$

Females, on the other hand, consumed significantly ( $p < 0.05$ ) more total sugar (62.4 versus 51.4 grams) as well as added sugar (68 grams versus 56.4 grams in males) although, the variance in the sample was substantial.

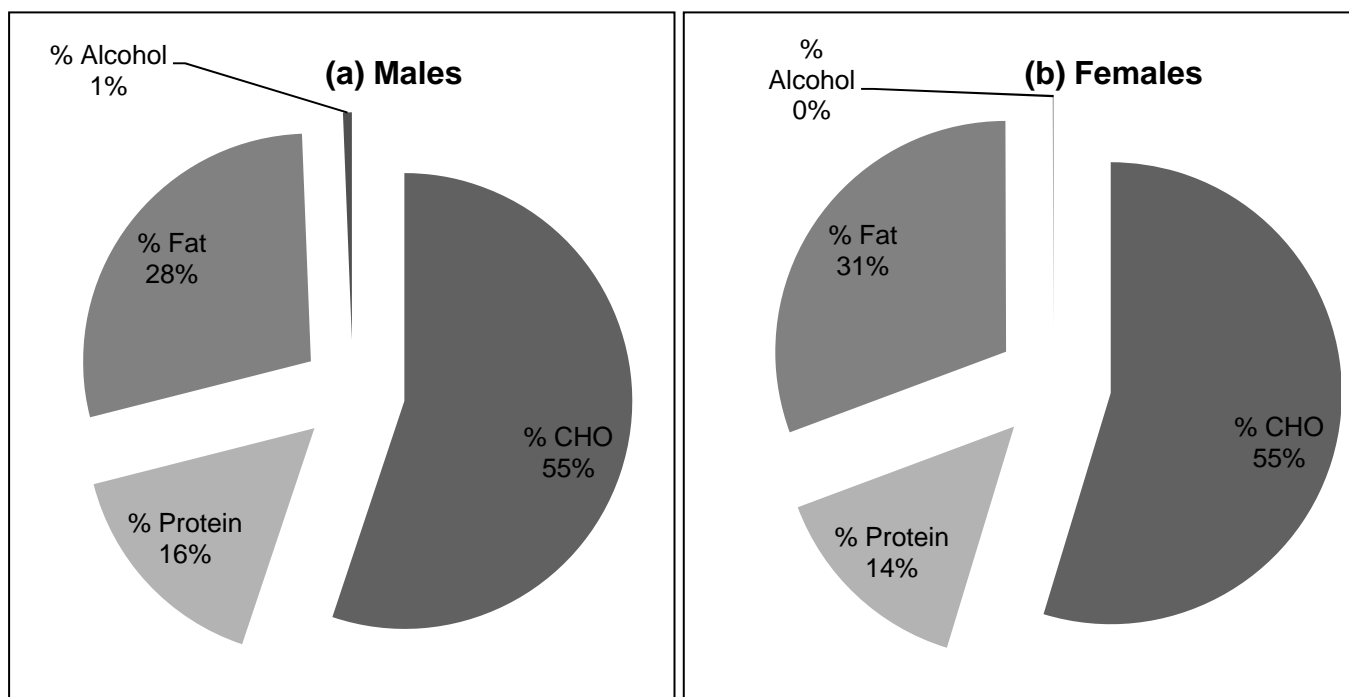


Figure 22: Dietary composition of (a) males and (b) females

When analysing the dietary composition of males and females, similar trends were observed (Figure 22). For both males and females, carbohydrates (CHO) contributed to the bulk of total dietary intake: over half of individuals' daily intake was composed of CHO, with both samples consuming 55%.

Males, on the other hand, consumed slightly more protein (16% versus 14% for the females), and linked with this, reported higher intakes of chicken and red meat consumption. Of these individuals, 76.2% of males ate chicken with the skin, compared to 64.2 % of the females. In contrast, more females ate lean chicken meat – with 34.5% of females consuming chicken without skin (compared to 23.8% of males). In addition, notably more males ate red meat: 95.1% of males compared to 84.5% of females. Furthermore, the majority of individuals, within both samples, tended to eat fatty red meat, with 73.4% and 52% of males and females consuming red meat with fat, while only 21.7 and 32.5% of males and females eating lean red meat. Particularly marked, was the daily consumption of processed meat, particularly within the male sample. Within the male sample, 18.9% ate processed meat every day, compared to less than half of this with the female sample (8.1%). Despite this, there was a higher prevalence of females who consumed processed meat on a weekly basis (56.8% of females compared to 44.8% of males), while slightly more males (36.4% versus 35.1%) reported consuming processed meat occasionally.

Females consumed slightly more fat – 31% compared to 28% consumed by the males. Linked with this was the slightly higher consumption of full fat dairy products within this sample, with a higher percentage of females using margarine (35.8% versus 30.8% in males), and consuming full cream milk (80.4% versus 72.7%). Only 12.6% and 12.2% of males and females consumed low fat milk, 8.4% and 2.7% consumed skim milk, while a very small number of individuals consumed no milk whatsoever – 4.2% of males and 4.1% of females. The higher intake of fat by female individuals may also have been related to the intake of fried foods, consumed on a weekly basis by 60.1% of females compared to 56.6% of males, and on a *daily* basis by 24.3% and 20.3% of females and males respectively.

Although Sodium (Na) intake was significantly ( $p<0.05$ ) higher within the male sample ( $1979.3 \pm 1512.4$  mg versus  $1525.2 \pm 1108.0$  mg for females) (Figure 23), there were minimal differences in the reported intake of salt between males and females. In fact, a higher percentage of males reported eating food lightly salted (70.6% of males compared to 58.1% of



females), and a lower percentage consumed 'very salted' food (24.5% of males versus 30.4% of females). However, notably more males reported adding salt even before tasting food (10.5%) compared to females (4.7%). Most subjects, within both samples, did not appear to eat salty snacks: 21.6% of females and 21.6% of males reported eating salty snacks, while 78.4% and 79% of females and males did not consume salty snacks.

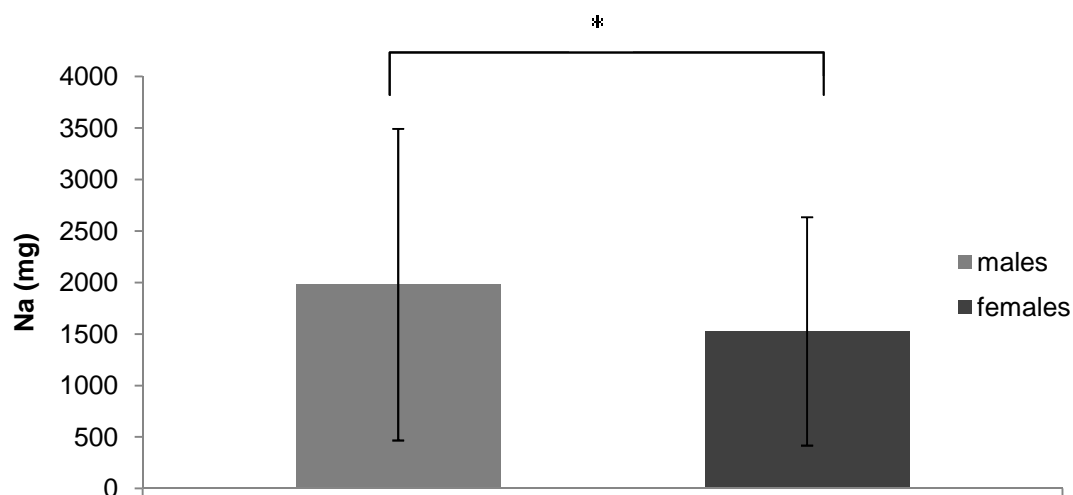


Figure 23: Mean ( $\pm$  standard deviation) sodium (Na) intake of males and females

Interestingly, most individuals (81.1% of males and 77% of females) did not consume fast food (Figure 24). Of those individuals that did consume fast foods, most consumed Kentucky Fried Chicken (KFC), however, this was a very small percentage of the sample as a whole (15.4% of males and 21.6% of females).

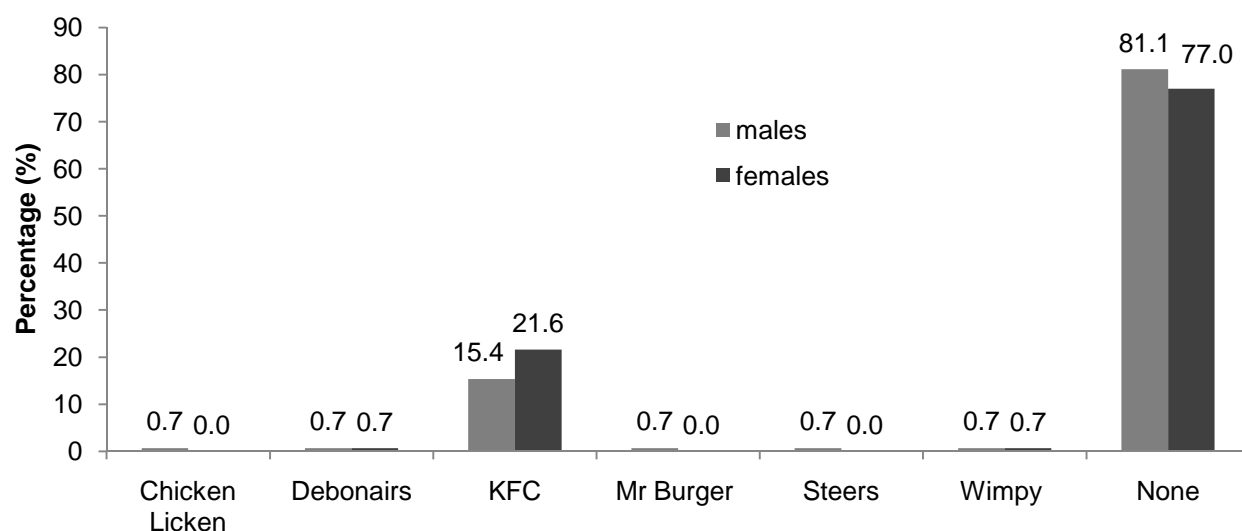


Figure 24: Self-reported fast food intake of male and female participants (KFC refers to Kentucky Fried Chicken).

## SMOKING

Significantly ( $p < 0.001$ ) more males (47.6%) smoked in comparison to females (2.7%) (Figure 25). In addition, significantly more males reported having smoked previously (21.05%) compared to females (1.4%).

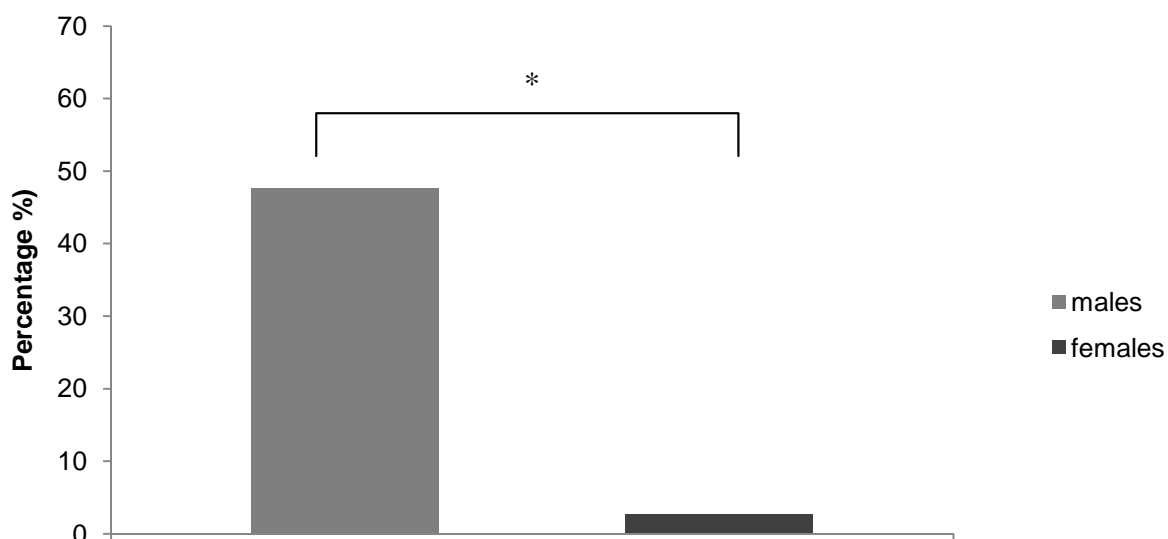


Figure 25: Percentage of males and females who currently smoke

For both males and females, tobacco equivalents smoked took the form of cigarettes. Females smoked an average of 8.5 ( $\pm 7.6$ ) cigarettes per day, compared to males who smoked just over half of this ( $5.1 \pm 3.8$ ) daily. This finding is however based on data from only four females. In addition there was a high degree of inter-individual variation, as evidenced in the high standard deviation within the sample.

In addition to smoking prevalence, exposure to environmental smoke (encompassing both tobacco smoke and environmental fumes, both in the work place and at home) was also assessed. While there was no significant ( $p > 0.05$ ) difference in exposure to environmental tobacco smoke (ETS) at home, significantly ( $p < 0.0001$ ) more males were exposed to smoking at work as well as fumes at the workplace (49.65% of males compared to 18.92% of females) (Figure 26).

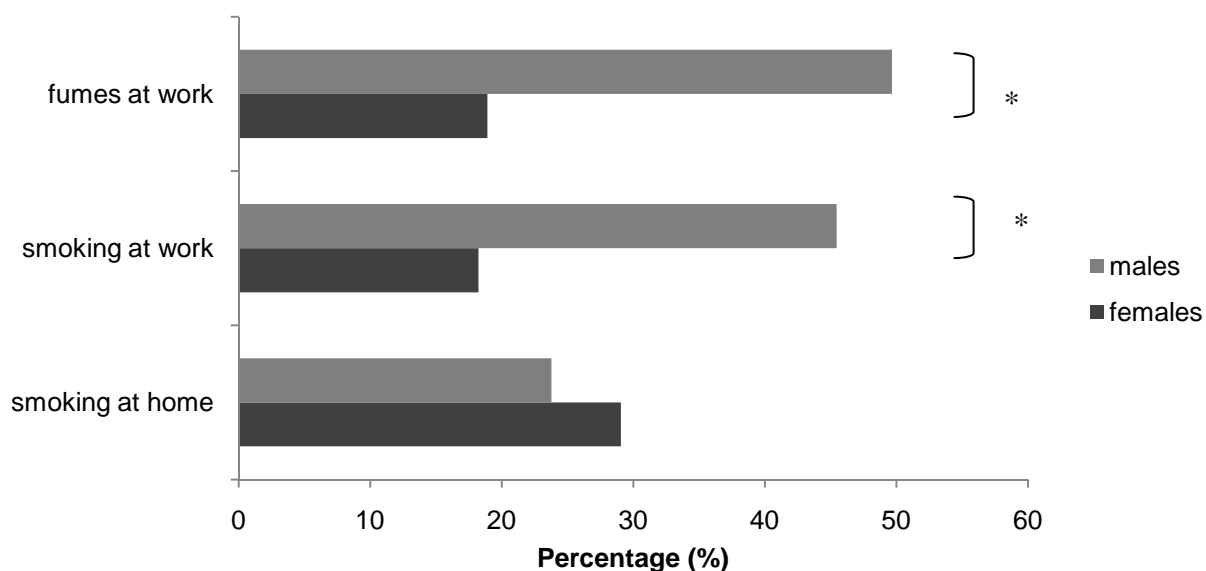


Figure 26: Percentage of male and female participants exposed to environmental and tobacco smoke at home and at the workplace

## ALCOHOL CONSUMPTION

The consumption of alcohol was significantly ( $p < 0.0001$ ) higher in the male sample. While 73.4% of males had consumed an alcoholic drink in the previous 12 months, this was the case for only 46.6% of females. There was also a significant ( $p < 0.0001$ ) difference in the average number of drinks consumed per day, with males consuming more than double ( $6.8 \pm 4.97$  drinks) the number of drinks than females ( $3.2 \pm 1.8$  drinks) on a daily basis (Figure 27).

The standard deviation within both samples was however high, highlighting a high degree of variation within individual participants.

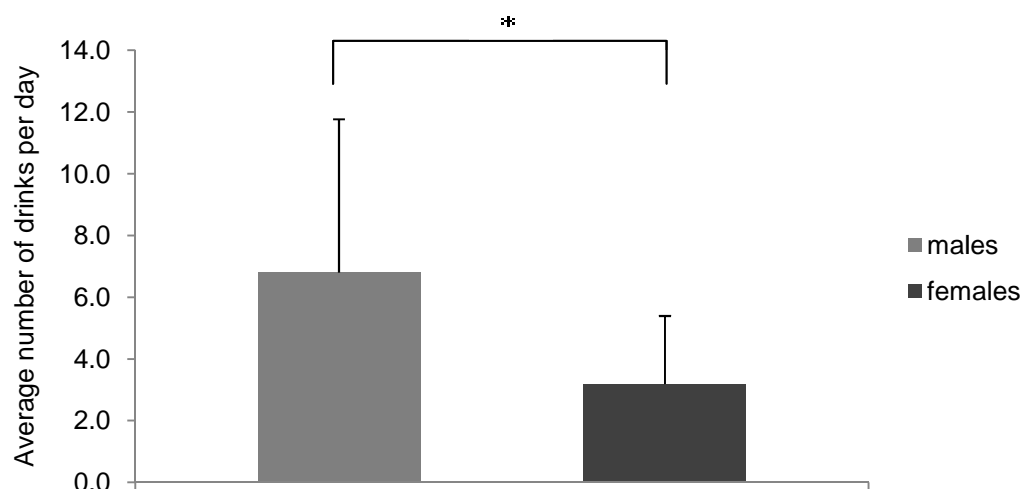


Figure 27: Mean ( $\pm$  standard deviation) number of alcoholic drinks consumed by male and female participants

Males also drank significantly ( $p < 0.0001$ ) more frequently, with 66.7% of males compared to only 13% of females drinking 1-4 days per week. Females were more inclined to drink less than once a month (40.6% compared to 10.5% of males). Aligned with this increased frequency of drinking, was the occurrence of binge drinking, which was particularly noteworthy within the male sample: while individuals tended not to drink during the week, they drank excessively on the weekend (Table XVI).

While the mean values do not clearly indicate this, there were notably high standard deviations for weekend days, particularly within the male sample. For example, while the mean number of drinks consumed by males on Saturday was 4.1, the standard deviation was 5.24. This means that some individuals were consuming up to 10 alcoholic drinks – more than double the average value. Similarly, on Sunday, while the mean number of drinks consumed was 2.07, with a standard deviation of 3.98, once again, some individuals were consuming almost double the volume of the group average consumption (Table XVI).

Table XVI: Mean ( $\pm$  standard deviation) number of drinks consumed by males and females on a weekly basis

AVERAGE NUMBER OF DRINKS PER WEEK (mean $\pm$ standard deviation)		
DAYS OF THE WEEK	FEMALES	MALES
Monday	0	0.34 ( $\pm 1.57$ )
Tuesday	0.01 ( $\pm 0.12$ )	0.02 ( $\pm 0.14$ )
Wednesday	0	0.01 ( $\pm 0.10$ )
Thursday	0	0.02 ( $\pm 0.14$ )
Friday	0.06 ( <b><math>\pm 0.34</math></b> )	2.22 ( <b><math>\pm 3.98</math></b> )
Saturday	0.55 ( <b><math>\pm 1.62</math></b> )	4.14 ( <b><math>\pm 5.24</math></b> )
Sunday	0.23 ( <b><math>\pm 1.2</math></b> )	2.07 ( <b><math>\pm 3.98</math></b> )

(Figures in bold represent notably high standard deviations)

Not surprising - given the significantly higher average alcohol consumption, the increased frequency of drinking per week, and the prevalence of binge drinking on the weekend - was the increased number of 'risky drinkers' within the male sample. More than half of the males (52.38%) that confirmed drinking within the previous 12 months were classified as 'risky drinkers' (>5 alcoholic drinks per day), compared to only 39.13% of females who were classified as such (>3 alcohol drinks per day) (Figure 28).

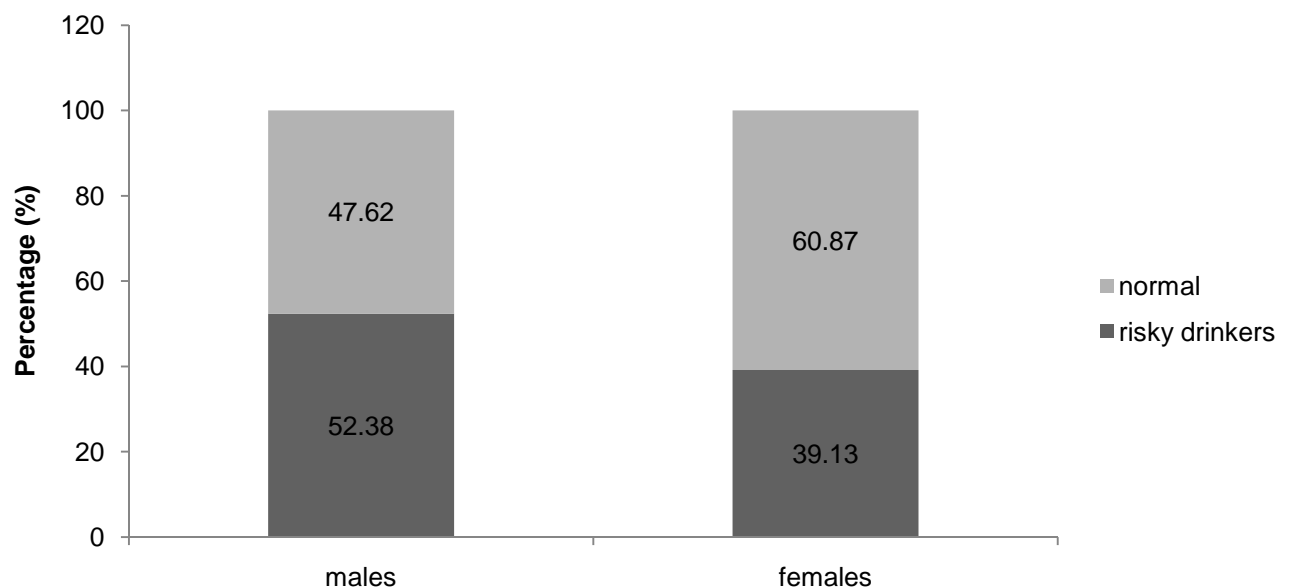


Figure 28: Percentage of males and females classified as 'risky drinkers'

In addition to this, 40% and 33.3% of males and females respectively were classified as alcohol dependent according to the CAGE questionnaire (**C**utting down, **A**nnoyance by criticism, **G**uilty feeling and **E**ye-openers.).

**SELF REPORTED AND PERCEIVED RISKS IN COMPARISON TO ACTUAL MEASURES**

For all risk factors, there were notable discrepancies between self reported and actual risk.

**Self reported versus actual measures of hypercholesterolemia**

With reference to self reported levels of hypercholesterolemia, of concern, was the fact that those found to be hypercholesterolemic were, for the most part, not aware of this condition. There was a significant difference ( $p<0.0001$ ) between self reported and actual levels of blood cholesterol in both samples (Figure 29).

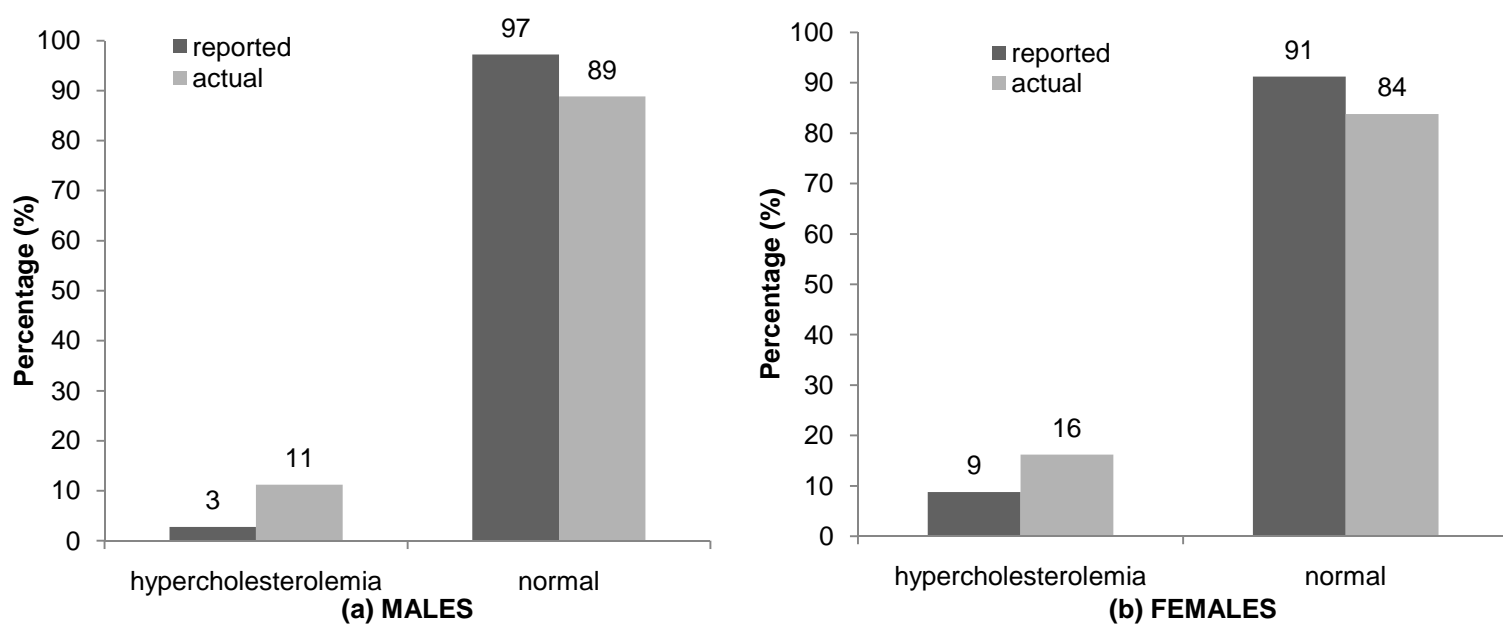


Figure 29: Reported versus actual cholesterol classification of (a) male and (b) female participants

Within the female sample, 9% of individuals reported having hypercholesterolemia, but 16% were in fact measurably hypercholesterolemic (Figure 29b). This finding was similar within the males where 11% were hypercholesterolemic, while only 3% reported this (Figure 29a). Despite this, however, prevalence of hypercholesterolemia was low within both samples, and this was confirmed by reported findings. Of the 84% of females with normal blood cholesterol levels, 91% reported this to be the case (Figure 29); and while 89% of males demonstrated readings within a normal range, 97% of them reported normal levels (Figure 29).

**Self reported versus actual measures of type II diabetes**

There was a significant difference ( $p<0.0001$ ) between self-reported and actual prevalence of the condition within the sample as a whole (Figure 30).

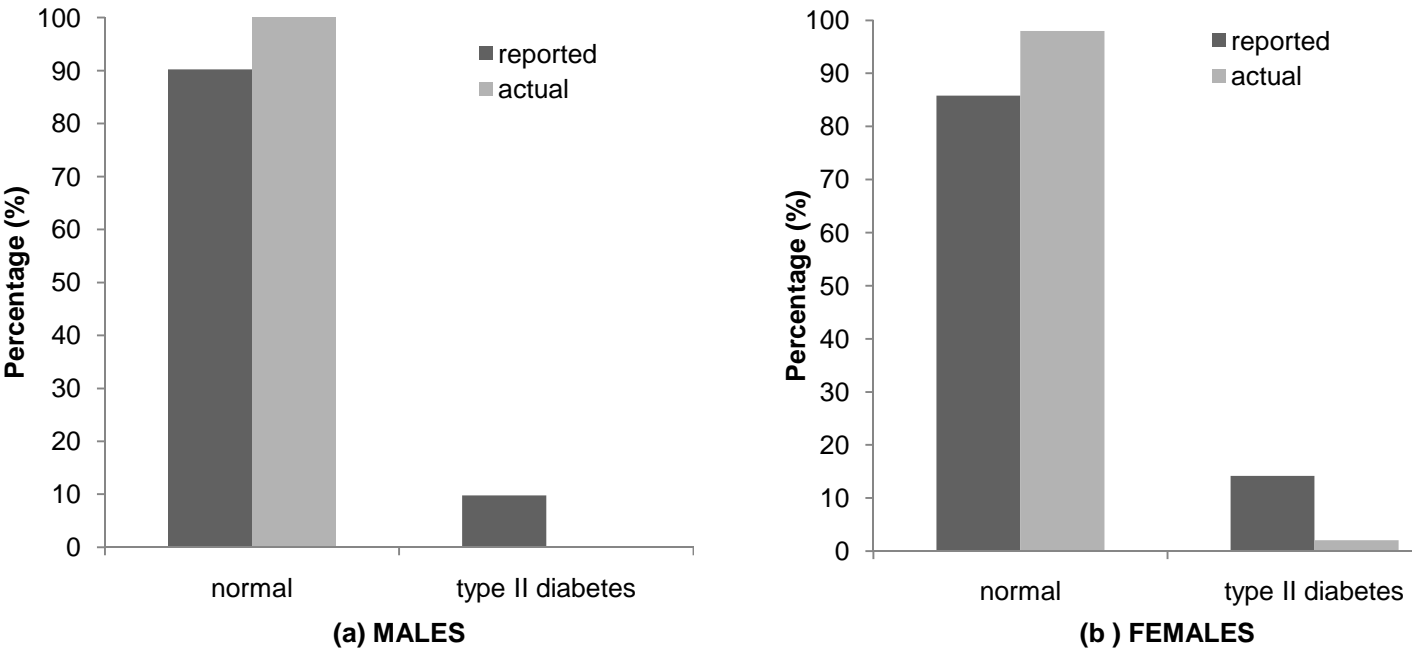


Figure 30: self reported versus actual measures of type II diabetes in (a) males and (b) females

Although 10% of males reported having elevated blood glucose, all participants (100%) had blood glucose within normal levels (Figure 30a). This was the case in 98% of females, with only 2% presenting with blood glucose levels classifying them as type II diabetic. This was significantly ( $p<0.0001$ ) lower than the self reported prevalence amongst female participants 14% of whom reported to have abnormally high blood glucose levels (Figure 30b).

### Self reported versus actual measures of hypertension

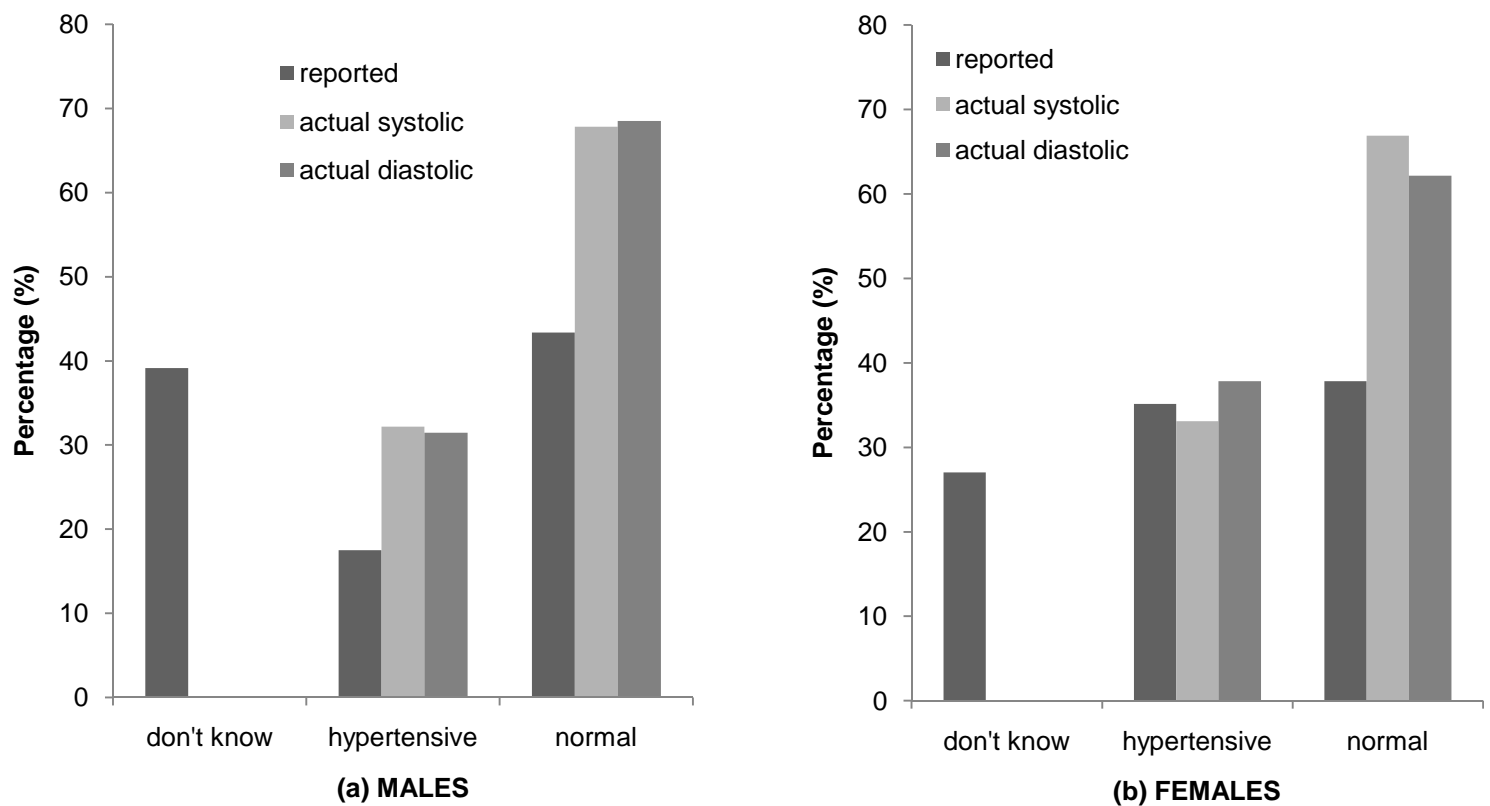


Figure 31: Reported versus actual classification of hypertension in (a) males and (b) females

The females' self reporting of hypertension was fairly accurate: 35% reported being hypertensive, while 33% (SBP) and 38% (DBP) were classified as such. While 67% and 62% of females demonstrated normal systolic and diastolic blood pressure readings respectively, only 38% of females reported their blood pressure to be within normal range. Additionally 27% of individuals did not know what their blood pressure was or if they were hypertensive or not (Figure 31b).

Within males, self reported responses were slightly different. Only 17% of participants reported being hypertensive, while 32% (SBP) and 31% (BDP) were classified as such, indicating an underestimation of the condition. While just under half of all males (43%) reported normal blood pressure, 68% (SBP) and 69% (DBP) were within normal range



(Figure 31a). On the whole, more males were ignorant of their blood pressure status, with 39% of them not knowing if they were hypertensive or not.

**Perceived versus actual prevalence of obesity**

Overall, there were significant ( $p<0.0001$ ) differences between individuals' perceived and actual body size (defined according to BMI classification). This was particularly marked for females. While 41.2% of females perceived themselves to be normal weight in reality, only 6.7% were classified as 'normal' (Figure 32).

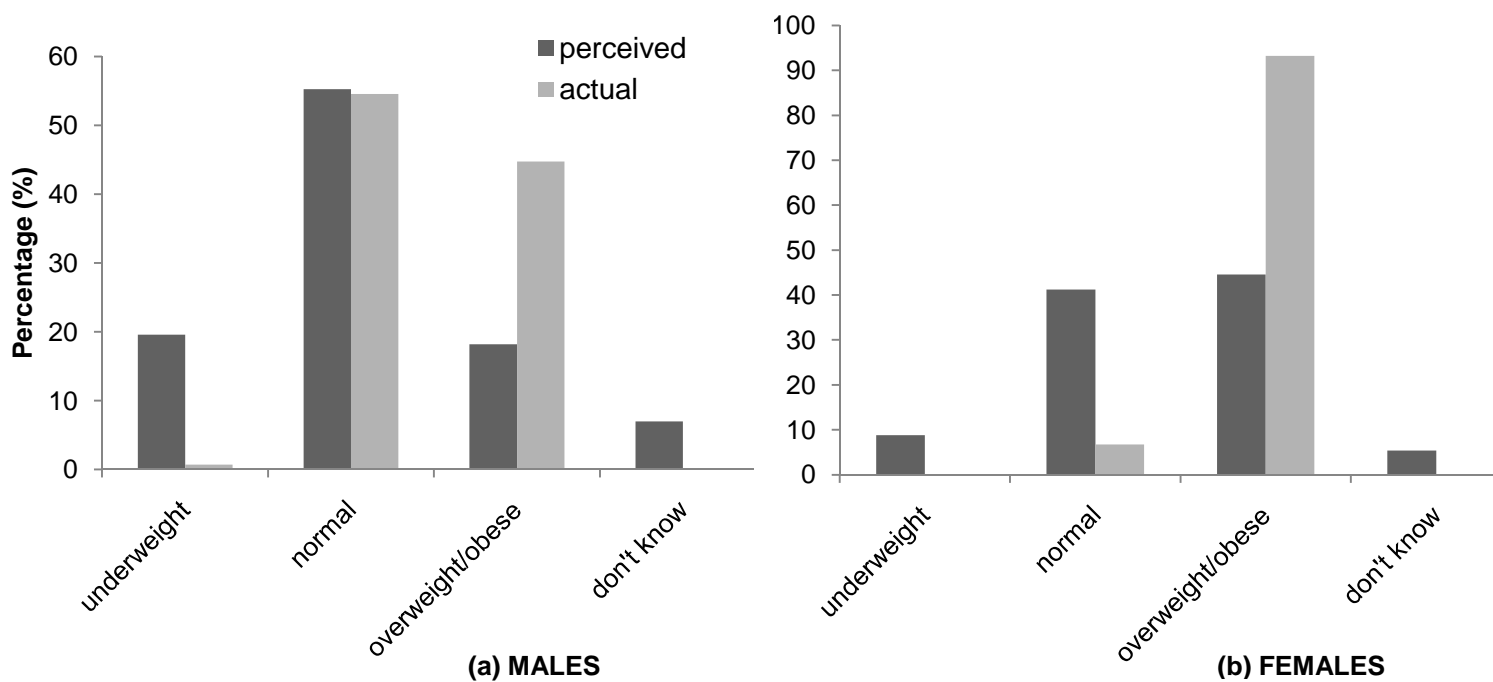


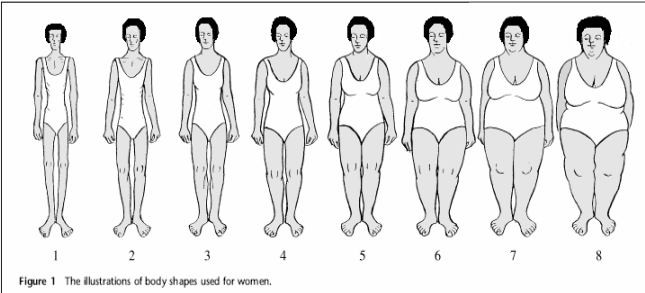
Figure 32: Perceived and actual body size of male (a) and female (b) participants

In addition, while 44.9% of females perceived themselves to be overweight, the actual prevalence was more than double this (93.2%) (Figure 32b). In comparison, differences between perceived and actual body weight were less distinct within the male sample, particularly with regard to the males' perception of 'normal' body weight: Ninety-eight percent of the men who were classified as normal, did in fact perceive themselves to be of normal body weight (55.2% perceived vs. 54.6% actual). In contrast to this, significant differences ( $p<0.0001$ ) were found between perceived and actual levels of overweight and obesity. Only

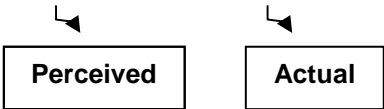
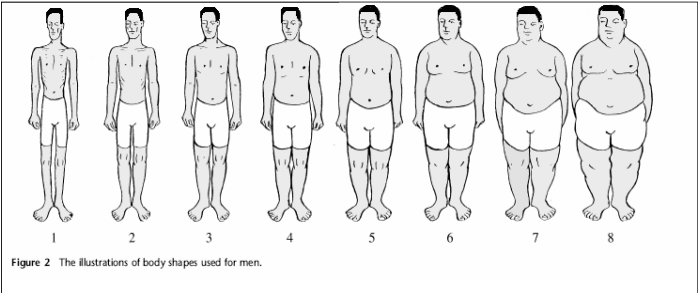
18.2% of the male participants perceived themselves to be overweight, while 44.8% were classified as overweight/obese (Figure 32a).

Overall, 8.8% of the females and 19.6% of the males perceived themselves to be underweight. This was the case for 0.7% of males, while no females were classified as underweight. In addition to inaccuracies with regard to perception of body weight, some individuals were in fact ignorant of their body size, and answered ‘I don’t know’ when questioned. Aligned with this, 5.4% of females and almost 7% of males had no perception of body size.

Referring to the illustrations presented to the samples (Figure 33), the females, with a BMI of  $37.6\text{kg.m}^{-2}$  were classified as ‘morbidly obese’, yet they rated themselves as 4.8 ( $\pm 1.4$ ), which is within ‘normal’ range (in comparison to a rating of 8, which represents ‘morbidly obese’). Therefore, at a very basic level, they perceived themselves to be half the size they were. Interestingly, they rated individual 3 as the most attractive. Within the male sample, while the group was classified as overweight, they rated themselves as 3.5 ( $\pm 0.9$ ), which again is slimmer than they were in reality (Figure 33).



(A)



(B)

Figure 33: Body images by sex (adapted from Ziebland *et al.*, 2002): illustrations for body shapes used for women (A) and men (B).

## **HYPOTHESES**

The research hypothesis proposed that the prevalence of CVD risk factors would be higher in females than males. It was further proposed that there would be a difference between perceived and self reported risk in comparison to actual measures.

### **Hypothesis 1:**

The first hypothesis proposed that there would be no difference in morphological risk between males and females as defined by a) BMI, b) waist circumference and c) body composition.

$$H_0: \mu_{\text{Males MORPH (a,b,c)}} = \mu_{\text{Females MORPH (a,b,c)}}$$

Females demonstrated significantly higher BMIs and larger waist circumferences, and had significantly different body compositions (significantly higher percentage and total body fat and significantly lower percentage and total lean body mass). Therefore for these variables the null hypothesis is rejected and the alternative hypothesis is accepted.

### **Hypothesis 2:**

In the second hypothesis it was proposed that there would be no difference between males and females with regards to cardiovascular (CV) risks as defined by d) blood pressure, e) total cholesterol and f) total blood glucose

$$H_0: \mu_{\text{Males CV (d,e,f)}} = \mu_{\text{Females CV (d,e,f)}}$$

Both systolic and diastolic blood pressure was similar between the two sexes, therefore for this response, the null hypothesis is accepted and the alternative hypothesis is rejected.

Mean total cholesterol and blood glucose were similar in both males and females, as was the prevalence of hypercholesterolemia and type II diabetes. Therefore the null hypothesis is accepted and the alternative hypothesis rejected for these values.

### Hypothesis 3:

The third hypothesis proposed that lifestyle-related risks would be similar between males and females with regard to g) dietary intake, h) physical activity, i) tobacco use, j) alcohol consumption and k) alcohol dependence.

$$H_0: \mu_{\text{Males LIFESTYLE (g,h,i,j,k)}} = \mu_{\text{Females LIFESTYLE (g,h,i,j,k)}}$$

Since males consumed significantly more in terms of total energy intake than females and were significantly more active (MET-mins.week), the null hypothesis for these two variables is rejected and the alternative hypothesis is accepted. The same applies to tobacco use, alcohol consumption and dependence, for which significantly more males smoked, drank and were alcohol dependent.

### Hypothesis 4:

The fourth hypothesis considered the difference between perceived/self reported and actual measures of l) obesity, m) hypertension, n) type II diabetes and o) hypercholesterolemia. It proposed that there would be no difference between perceived/self-reported prevalence and actual measures for these risks.

$$H_0: \mu_{\text{Reported (l,m,n,o)}} = \mu_{\text{Actual (l,m,n,o)}}$$

With regard to obesity there were significant differences in perceived and actual measures, with both males and females underestimating their level of obesity. Therefore for this variable the null hypothesis is rejected and the alternative hypothesis is accepted.

Hypertension presented with a higher degree of accuracy in terms of self-reporting in the *females*, however males under-reported the condition. Therefore the null hypothesis is accepted for females and rejected for the males, for whom the alternative hypothesis is accepted.

The prevalence of type II diabetes was significantly *over-reported* in both samples, while hypercholesterolemia was significantly *under-reported*. Therefore for these variables the null hypothesis is rejected and the alternative hypothesis is accepted.

## CHAPTER V

### DISCUSSION

While it is well established that CVD is on the increase, both globally as well as in developing countries such as South Africa, the degree to which different CVD risks affect various population groups within the 9 provinces of the country, and the interaction between these risks within different individuals remains unclear. To date the Eastern Cape has only been included within two national surveys with respect to demographics and health, and thus there is paucity in the literature on specific health risks among urban black individuals within this province. The aim of this chapter is to assess, compare and attempt to explain CVD risk within this population group, in relation to other provincial and national findings over the last few years. While the chapter follows roughly the same format as the Chapter IV, in some instances the order changes due to the interlinking of various risk factors and the combined discussion thereof.

#### MORPHOLOGICAL RISK

##### OBESITY

The current data followed the same trend as provincial and national statistics, with females demonstrating significantly ( $p < 0.05$ ) higher BMI values (as well as a higher percentage of obesity) in comparison to their male counterparts (Figure 7: Pg. 77; Table XII: Pg. 76). This supports the notion that obesity is one of the leading CVD risks within South African females (van der Merwe & Pepper, 2006). Recent literature suggests that 31-34% of black South African women are obese ( $BMI > 30 \text{ kg.m}^{-2}$ ) compared to only 8% of black males (van der Merwe & Pepper, 2006). In this study, 81% of females were obese, compared to 17% of males (Figure 8: Pg. 77).

National statistics taken from the last two Demographic and Health surveys reveal a trend of *increasing* obesity levels within *both male and female* individuals over the last few years (Table XVII: Pg. 105). In comparison with data from the 1998 and 2003 DHS, participants from the current study appear to be heavier, and to have higher BMIs and waist circumferences. This is particularly marked in female individuals (Table XVII). It must be noted however, that the findings from the DHS (1998 and 2003) represent national statistics

on black urban males and females, while the current data represents black urban males and females from the *Eastern Cape* only. However, province-specific data was not available for comparisons to be made.

Table XVII: Comparison of morphological data from 1998-2010 for urban black males and females within South Africa

	FEMALES			MALES		
RISK FACTORS	DHS		CURRENT STUDY	DHS		CURRENT STUDY
	1998	2003	2010	1998	2003	2010
Stature (mm)	1580	1590	1576	1680	1690	1670
Mass (kg)	70.6	70.4	92.7	65.3	65.4	72.1
BMI (kg.m <sup>-2</sup> )	28.4	28.1	37.5	23.6	23.1	25.7
WC (mm)	869	846	1023.4	808	780	887.4
>WC cut off <sup>1</sup> (%)	44.6	39.1	82	6.8	3.1	15

Where BMI represents: Body Mass Index (mean values); WC: waist circumference;

<sup>1</sup> >1020mm for males, >880mm for females. Columns highlighted in red represent current data

Thus, in comparison to statistics countrywide, black females in this study were approximately 20 kilograms heavier, had a mean waist circumference that was approximately 200mm greater, and about 40% more were above the WC cut-off level of 880mm. Differences, although not as marked, are also evident within male individuals, with current data identifying urban black males from this Eastern Cape population group as being approximately 7 kilograms heavier and 80mm larger around the waist than the national average, with 8-12% more individuals with a WC above the cut-off of 1020mm.

Similar discrepancies can be observed when comparing BMI classification data (Figure 34: Pg. 106). In this study, 12% of the females were overweight and **81%** were obese. According to national statistics, in 1998, 25.5% of urban black females were overweight, and **36.3%** were obese ( $\geq 30\text{kg.m}^{-2}$ ); in 2003, 27.1% of urban black females were overweight, and **33.8%** obese. (van der Merwe & Pepper, 2006; DHS, 2003).

In contrast, the prevalence of obesity within the male sample was significantly lower than within the female sample. In this study 28% of males were overweight and **17%** were obese.

However, as with the results for the female sample, this prevalence rate is notably higher than the national prevalence rate of obesity, which was shown to be **12%** in 1998 and **13%** in 2003 (DHS, 2003; van der Merwe & Pepper, 2006).

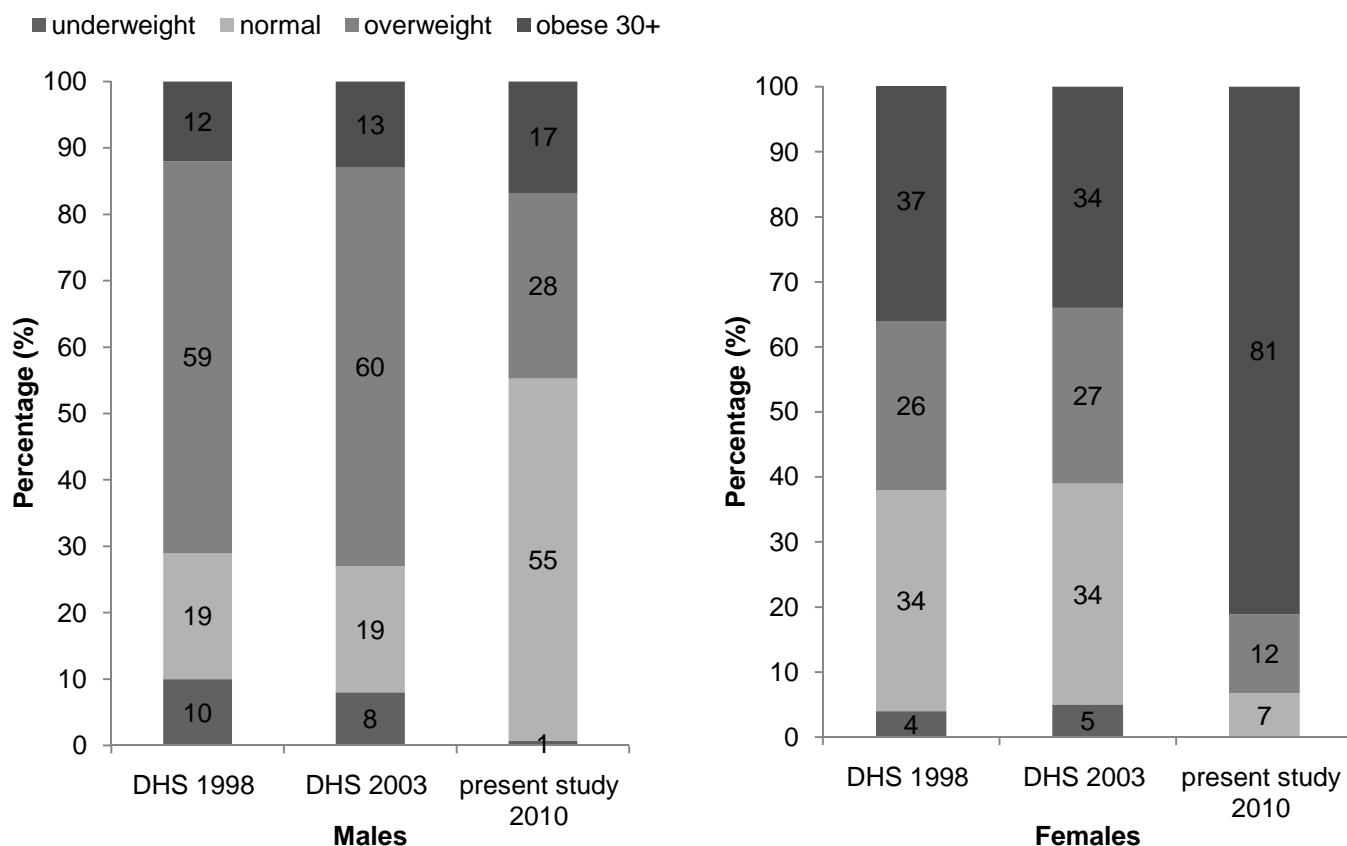


Figure 34: BMI classification of urban black males and females: comparison between the 1998 and 2003 DHS and current data

In contrast to national statistics, *no definitive trends* are apparent in the *provincial findings* (Table XVIII: Pg. 107). While it is evident that obesity within males has increased slightly, from around  $21\text{kg.m}^{-2}$  in 2000 and 2001 to  $25\text{-}26\text{kg.m}^{-2}$  from 2003-2008; within females, there appears to be no trend at all, with BMI values remaining in the range of  $28\text{-}30\text{kg.m}^{-2}$ . Of concern, however, is the notably higher prevalence of *severe obesity* within the female sample in the current study (Figure 8: Pg. 77), which is markedly higher than other provincial findings (Table XVIII: Pg. 107): Only Punyandeera *et al.* (2001) demonstrated a mean BMI that is comparable to current findings. In contrast, most other provincial studies reported mean BMIs within the range of  $28\text{kg.m}^{-2}$  to  $32\text{kg.m}^{-2}$ , substantially lower than the current findings (mean BMI =  $38\text{kg.m}^{-2}$ ). In addition, 30-60% more females within the current sample were obese, highlighting the notably high prevalence of Stage III obesity. In comparison, the



findings in the male sample (mean BMI of 26kg.m<sup>-2</sup>), were more comparable to provincial findings, with most studies indicating a mean BMI range of 21kg.m<sup>-2</sup> to 26kg.m<sup>-2</sup>.

Table XVIII: Obesity prevalence across different provinces of South Africa within urban black males and females (1991-2010)

AUTHORS	YEAR	PROVINCE	FEMALES			MALES		
			Mean BMI	% OW	% obese	Mean BMI	% OW	% obese
Current Study	2010	Eastern Cape	38	12	81	26	28	17
Tibazarwa <i>et al.</i>	2009	Gauteng	32		55	26		23
Sliwa <i>et al.</i>	2008	Gauteng	30			25		
Malhotra <i>et al.</i>	2008	Western Cape		25	53		15	19
Schutte <i>et al.</i>	2006	North West,	28					
Vorster <i>et al.</i>	2005	North West		25	23		8.7	5
Senekal <i>et al.</i>	2003	National urban	30	35	40	26	29	20
Kruger <i>et al.</i>	2001	North West	28			21		
Punyadeera <i>et al.</i>	2001	Gauteng	37					
Kruger <i>et al.</i>	2001	North West		31	39			
Vorster <i>et al.</i>	2000	North West	28			21		
Mollentze <i>et al.</i>	1995	Orange Free State	30		54	24		20
Seedat <i>et al.</i>	1992	Kwa-Zulu natal			23			4
Steyn <i>et al.</i>	1991	Western Cape	28		44	23		9

Where: BMI: Body Mass Index; OW: Overweight; %: Percentage. Row highlighted in red represents current data.

Both the provincial findings and the current results definitively confirm the notable difference in obesity prevalence between males and females with the latter, for the most part, demonstrating BMI values at least 5-7kg.m<sup>-2</sup> higher than their male counterparts (Table XVIII). It is likely these significant differences between the sexes are related to lifestyle factors. In this study, although males consumed significantly more daily in terms of total energy intake (Figure 21: Pg. 88), they were also significantly more active (Figure 17: Pg. 85). It is well established that obesity is related to, and strongly associated with, diet (energy intake that exceeds expenditure and one that is rich in fats) and physical inactivity (Case & Menendez, 2009; Malhotra *et al.*, 2008; Bray & Champagne, 2005; Walker *et al.*, 2001).

## Lifestyle factors

The fact that individuals in this study were *urban* may account, in part, for the high obesity and WC findings, consistent with national trends. Vorster *et al.* (2000) similarly found that urbanization was associated with increased body mass and a higher prevalence of obesity. It is well established that increases in body mass, waist circumference and BMI are generally attributed to urbanization (Kruger *et al.*, 2005), in addition to the fact that urban living generally dictates that individuals experience higher stress levels and lifestyles are more sedentary (Ghoziladeh & Davidson, 2008; Malhotra *et al.*, 2008; Walker *et al.*, 2003; Hlatky *et al.*, 1995).

Many participants within the current study were employed by an institution which provided meals for employees. Thus, many of these individuals were eating three large meals per day, in addition to some individuals snacking in between. This may have accounted for higher total energy intake, which may in turn have translated into higher levels of obesity within both sexes compared to national prevalence rates (DHS, 1998; DHS, 2003). Interestingly, while there was no significant correlation between total energy intake and body mass within the females ( $r=0.1$ ,  $p>0.05$ ), within the male individuals, there was a significant ( $p<0.05$ ) *negative* correlation between these variables ( $r=-0.2$ ) as well as between total energy intake and BMI ( $r=-0.2$ ). Interestingly, this suggests that males who consumed *more*, had a significantly *lower* body mass and BMI – a relationship which is almost certainly related to physical activity levels in the male sample, in which a high level of physical activity necessitated the need to eat more, and yet simultaneously resulted in these males being lighter and with lower BMIs.

A weak relationship between energy intake and BMI has been confirmed by other research, and has been linked to the tendency for obese individuals to under-report dietary intake, thus confounding any relationship or correlations between these variables (Kruger *et al.*, 2006). This was likely the case in the female sample where significant relationships were absent between dietary intake and BMI as well as between overall intake and body weight, despite the high obesity levels in the sample. Interestingly, significant *negative* correlations ( $p<0.05$ ,  $r=-0.2$ ) were found between *CHO intake* and *body mass* as well as *CHO intake* and *BMI* in both *males and females*, indicating that those who ate more CHO had significantly lower body mass and BMIs. Again, this may have been related to physical activity levels promoting

the need to consume more CHO, yet simultaneously resulting in lower body mass and BMI values in these individuals.

With regard to obesity and level of physical activity, males were significantly more active. At a very basic level, it is proposed that this may have accounted for lower obesity levels in this sample in general. This is supported by a significant ( $p < 0.05$ ) negative correlation ( $r = -0.2$ ) between total physical activity and body mass, as well as between total physical activity and BMI ( $r = -0.2$ ;  $p < 0.05$ ) – relationships which were absent in the female group. This highlights the negative impacts of reduced physical activity associated with increased levels of obesity among these individuals, a finding which has been highlighted in other provincial studies within the country (Dugas *et al.*, 2009a; Kruger *et al.*, 2002; Sparling *et al.*, 1994).

### **Socio-demographic considerations**

Within the current sample, most individuals were domestic workers, kitchen assistants or gardeners: very few were involved in heavy manual labour, which has been shown to have a notable impact on nutritional and health status (Seftel, 1978). In comparison to this, national statistics for urban males and females include a wide range of individuals - from those who are unemployed, to those involved in manual labour as well as those who are sedentary (DHS, 2003). In addition, the majority of individuals assessed in the current study were better circumstanced and earning more than the average black South African. Aligned with this, research has demonstrated higher levels of obesity linked with higher socio-economic status and among individuals who are better circumstanced (Senekal *et al.*, 2003; James *et al.*, 2001). In a study by Senekal *et al.* (2003), which assessed *economically active* urban black individuals throughout the country, 40% of black women were obese. Although the mean BMI of  $29.76 \text{ kg.m}^{-2}$  (compared to the mean BMI of  $37.56 \text{ kg.m}^{-2}$ ) found in the female sample in this study) was notably lower, the male BMIs were comparable ( $25.57 \text{ kg.m}^{-2}$  compared to  $25.7 \text{ kg.m}^{-2}$  found in the present study for males).

Although a link between level of education and BMI has been claimed in some past research, within the current sample, no correlations between these two factors were found within either sex (Table XI: Pg. 75) ( $r = 0.03$  for females and  $0.2$  for males). This is in line within findings of Malhotra *et al.* (2008), who similarly found no association between educational status and risk of being overweight/obese. This remains a relationship which requires more research,

due to the inconsistencies which exist in the literature: while the current study demonstrated no relationship between these variables, other studies have presented conflicting results, with some indicating that *lower* education levels are associated with higher BMIs (Kruger *et al.*, 2005), while others reporting the opposite to be the case (Senekal *et al.*, 2003).

## Waist circumference

Females' WC (mean = 1023mm) was significantly ( $p < 0.001$ ) larger than the males' (887mm), placing them within category 2 risk ( $> 880\text{mm}$ ) (Figure 9: Pg. 78; Figure 10: Pg. 79). This result is slightly higher than values recorded in other South African studies, where black women have been found to have WC values of 948mm (Punyandeera *et al.*, 2001), 863mm (Alberts *et al.*, 2005) and 816mm (Schutte *et al.*, 2006). This confirms the prevalence of abdominal obesity among women in this population group and the finding that black women tend to have greater proportion of subcutaneous adipose tissue than women in other population groups. Consistent with findings from other South African studies (Kruger *et al.*, 2001; Mollentze *et al.*, 1995), a strong correlation was found between WC and BMI in both males ( $r = 0.9$ ,  $p < 0.0001$ ) and females ( $r = 0.7$ ,  $p < 0.0001$ ). This confirms the notion that waist circumference is an appropriate measure and predictor of abdominal obesity. Interestingly, research, while confirming the high prevalence of abdominal obesity in black women, has not shown any linkage to insulin sensitivity or type II diabetes. While obese, white women tend to have more visceral fat which predisposes them to metabolic disorders and impaired glucose function, this tendency has not been found in black women: it has been linked to an altered distribution of fat and a tendency for fat to be subcutaneous abdominal in area (Punyadeera *et al.*, 2001a). The results for the females in this study are in contrast to this: there was a *significant positive* correlation between WC and total blood glucose ( $r = 0.2$ ,  $p < 0.05$ ). Interestingly, among male individuals, the relationship between WC and blood glucose was even stronger ( $r = 0.3$ ,  $p < 0.01$ ), indicating that WC may play an important role in blood glucose disorders such as diabetes in male individuals. In accordance with this Wang *et al.* (2005) suggested that WC was a strong predictor of type II diabetes in males. They also reiterated that WC was a significantly better predictor of the condition than BMI.

In addition to the significant relationship between WC and blood glucose, WC was significantly associated with all other CV variables. In comparison, although BMI (usually accepted as a CVD risk predictor) was significantly associated with most CVD risk factors,

there was no significant relationship between BMI and total cholesterol in males or between BMI and total blood glucose in females. Similar findings were demonstrated by Levitt *et al.* (1999), who demonstrated that a WC larger than 920mm was independently associated with type II diabetes, while Charlton *et al.* (2001) found the number of CVD risk factors was significantly higher in individuals with a WC greater than 920mm. Both studies included individuals of mixed ancestry. Findings such as these highlight the importance of WC as a CVD measure and disease predictor, particularly in population groups already at risk. This result is consistent with past research, which has proposed that WC, in addition to being a good measure of abdominal obesity (Punyadeera *et al.*, 2001a), may be a *better predictor* of CVD than BMI (Rush *et al.*, 2007), due to the fact that risk factors tend to cluster in individuals with larger waist circumference values, in particular those above 920mm (Charlton *et al.*, 2001).

## **Body Composition**

With a mean body fat percentage of 26.8% recorded for males and 51.8% for females (Figure 11: Pg. 79; Figure 12: Pg. 80), both samples are notably above the suggested norms proposed by the ACSM (ACSM, 2000). This association suggests that between the ages of 30 and 59 years, males should have a body fat percentage of 19-22% and females of 25-30%. In comparison with these values, not only are both sexes in the current research notably above the recommended values, but females have approximately 20% more body fat than is desired. This finding is almost certainly related to the high level of obesity within the female sample, 93% of whom were classified as overweight/obese, within which 39% were classified as obese, class III (BMI  $\geq 40$ ). Similar values were found in a study by Punyandeera *et al.* 2001b, who demonstrated a body fat percentage of 47.2% in obese black women, while Dugas *et al.* (2009a) reported a similar body fat percentage of 46% in this group.

However, these results should be viewed with caution due to the known limitations associated with Bioelectrical Impedence Analysis (BIA). These include the tendency of BIA to *over-predict* body fat percentage, particularly in obese individuals (Deurenberg, 1996). It has been proposed that this has to do with the amount of total body water, as well as different body geometries, which result in different measurement errors. Neither factors are constant and therefore, when placed in a standardised BIA equation ( $V = p \times S^2/R$ ), present very different results (Deurenberg, 1996).

The body proportions of an obese individual are very different from those of a lean individual (Deurenberg, 1996). Obese individuals tend to have much larger areas making up their trunk and extremities, resulting in disproportionate impedance values for these areas (Deurenberg, 1996). The trunk itself contributes relatively little to total impedance (10-20%) due to the fact that it is short with a large diameter (Deurenberg, 1996). In obese individuals, relatively more water (and therefore fat-free mass, FFM) is located in the trunk. Since, however, the effect of the trunk on total body impedance is low, this is not detected and therefore the prediction of total body water and FFM generally tends to be falsely *low*, and consequently fat mass, inaccurately *high* (Deurenberg, 1996). The result is an overestimation of body fat percentage. This is reportedly especially the case in subjects with abdominal obesity – which most of the female subjects presented with (mean WC = 1023mm = category 2 risk).

For BIA to be accurate, certain measurement protocols should be standardised and controlled, protocols which in the present research were not absolutely adhered to owing to logistical constraints. These include: no eating or drinking at least 4 hours prior to assessment, no moderate or vigorous activity within the previous 12 hours, no alcohol consumption within the previous 48 hours and no caffeine prior to assessment (McArdle *et al.*, 2001). Lack of control over all these factors could have influenced the accuracy of the findings. In addition, since the Impedance Analyzer used computed body fat and lean body mass percentages independently, there was no control over the specific equation used. Therefore fatness-specific BIA equations that predict body fat for obese individuals as well as individuals of different ethnicities, which are available (McArdle *et al.*, 2001), were not an option and were not used in the analyses.

According to Hill *et al.* (1995), body weight and *composition* are determined by both current environmental conditions (lifestyle, diet and physical activity) as well as the functional phenotype of an individual (behavioural and metabolic characteristics and past experiences) (Senekal *et al.*, 2003). Therefore, in theory, there should be a strong link between these lifestyle and behavioural factors such as diet and physical activity, and an individual's body composition.

The impact of *diet* on body composition was notably more evident within the male sample, in which significant relationships were found between dietary variables and body composition –

relationships which were not as clear within the female group. Within the males, there were significant ( $p < 0.05$ ) *negative* correlations between total energy intake (kJ) and % body fat ( $r = -0.3$ ), and between total energy intake and total body fat (kg) ( $r = -0.2$ ), and a significant ( $p < 0.05$ ) *positive* correlation between total energy intake (kJ) and total lean body mass (kg) ( $r = 0.3$ ). The significant negative correlations within the male sample indicate that individuals who ate *more*, had significantly *less* body fat, and simultaneously significantly *more* lean body mass. None of these relationships were evident within the females – again, suggesting the potential role of physical activity in causing the males to eat more, yet simultaneously resulting in significantly lower levels of body fat. Despite the fact that fat contributed more to total dietary intake in the female group, there were no significant relationships between fat intake and body composition (Figure 22: Pg. 89).

Interestingly, the impacts of sugar on body composition were more apparent: females consumed significantly ( $p < 0.05$ ) more sugar than males, and while there was no significant relationship between total added sugar and body composition within the *males*, there was a significant ( $p < 0.05$ ) *negative* correlation ( $r = -0.2$ ) demonstrated in the female sample. This, specifically relating to total *lean* body mass: This suggested that those females who consumed more total sugar, has significantly less lean body mass. Interestingly, a higher intake of sugar was also significantly related to *higher SBP* in this group ( $r = 0.2$ ;  $p < 0.05$ ), suggesting the dangerous effect of higher sugar intake translating into raised blood pressure levels.

The lack of association between increased fat intake and body composition within the females may be again be related to under-reporting. Livingston & Black (2003) suggest that if total energy intake is under-reported, then components of total energy intake are likely to be under-reported as well, which may in turn lead to distortions between nutrient intakes and disease risks and outcomes. In confirmation of this, inaccurate and under-reporting of dietary fat and sugar intake may have compounded any relationships with body composition within the female sample.

Body composition is a more accurate measure of body fatness than mass or BMI, as it indicates the percentage contribution of fat-mass to total body mass. In contrast to this, BMI classifications can often be inaccurate since they do not make this distinction (McArcle *et al.*, 2001). Not surprisingly, within both the male and female samples, those who were classified

as overweight and obese according to their BMI, were in fact over fat as well. There was a strong positive and significant relationship between both body mass and % body fat ( $r=0.7$ ,  $p<0.01$ ), as well as between BMI and % body fat ( $r=0.7$ ,  $p<0.01$ ) for males as well as for females (body mass and % body fat:  $r=0.8$ ,  $p<0.01$ ; BMI and % body fat:  $r=0.9$ ,  $p<0.01$ ). In addition, there were strong correlations between % body fat and other CVD risks: in females % body fat was significantly correlated with SBP ( $r=0.3$ ,  $p<0.01$ ), DBP ( $r=0.3$ ,  $p<0.01$ ) and total cholesterol ( $r=0.3$ ,  $p<0.01$ ), while in males similar correlations were found in all these variables in addition to total blood glucose ( $r=0.3$ ,  $p<0.01$ ). This supports the well established theory that excess body fat is associated with various other CVD risks, including hypertension, hypercholesterolemia and type II diabetes (ACSM, 2000).

## **CARDIOVASCULAR RISKS**

### **HYPERTENSION**

In the present study, no significant differences were found between males and females with regard to mean blood pressure (Table XIII: Pg. 80). However, females presented with higher values for both diastolic and systolic responses. This is most likely related to the higher obesity levels in this sample, and associated with this, significantly higher percentage and total body fat. This is an important finding due to the association between raised blood pressure and strokes in the South African black population (Norman *et al.*, 2007). As with the morphological data, the mean blood pressure levels recorded were notably higher than in the national statistics given for black urban males and females. In 1998, black urban males had a mean blood pressure of 122/75mmHg - very similar to the mean of 123/73mmHg found in 2003. In contrast, black urban females were found to have a mean blood pressure of 118/75mmHg in 1998 and 121/75mmHg in 2003. Therefore, in comparison to current findings, not only are the female values *higher* than the male values (the opposite was found in the DHS data), but the blood pressure values for the sample as a whole are notably higher. When comparing the blood pressure of individuals, with values from other provinces, *similar* findings were recorded, and while there are no notable differences in mean blood pressure values between the sexes, females tend to present with slightly higher values (Table XIX: Pg. 115). Consistent with present findings, Tibazarwa *et al.* (2009), Alberts *et al.* (2005), Vorster *et al.* (2002 & 2000) and Mollentze *et al.* (1995) all reported slightly higher mean SBP and DBP values for black females (Table XIX).



Table XIX: Blood pressure of urban black individuals (35-54) within different provinces of South Africa (1991-2010)

AUTHORS	YEAR	PROVINCE	FEMALES		MALES	
			SBP	DBP	SBP	DBP
Current study	2010	Eastern Cape	133	85	131	82
Tibazarwa <i>et al.</i>	2009	Gauteng	133	84	133	83
Schutte <i>et al.</i>	2006	North West	136	78		
Alberts <i>et al.</i>	2005	Limpopo	136	86	133	86
Vorster	2002	North West	135	83	132	79
Kruger <i>et al.</i>	2001	North West (M & F))			138	86
Punyandeera <i>et al.</i>	2001	Gauteng	133	84	133	83
Vorster <i>et al.</i>	2000	North West	135	83	132	79
Mollenze <i>et al.</i>	1995	Orange Free State	135	81	128	79
Steyn <i>et al.</i>	1991	Western cape	117	77	124	81

Where: SBP represents systolic blood pressure; DBP: diastolic blood pressure; M: males; F: females. Column highlighted in red represents current findings

In terms of levels of hypertension, notably higher levels than in national statistics for urban black individuals were also evident. In 1998, 21.5% of urban black males were classified as hypertensive, while this decreased to 11% in 2003 (DHS, 2003; DHS, 1998). In the current study **32%** were classified as hypertensive according to their systolic blood pressure (SBP) and **31%** according to their diastolic blood pressure (DBP) (Figure 13: Pg. 81). The female data presents a similar trend, with 25% and 20% of urban black females classified as hypertensive in 1998 and 2003 respectively. In the current study **32%** of females were classified as hypertensive according to their SBP and **37%** according to their DBP (Figure 13: Pg. 81).

In contrast to the findings of the THUSA study (North West province), in which the prevalence of hypertension was similar in both black urban males (11.1%) and females (11.8%), in the current study females presented with significantly higher levels of stage II hypertension (females = 14% males = 8%). These results are similar to the findings of Steyn *et al.* (1991), that while approximately 24% of females (35-54 years) presented with hypertension  $\geq 160/95\text{mmHg}$ , this was the case in only 10% of the males within the same age group. However, more males than females presented with moderate hypertension ( $\geq 140/90\text{mmHg}$  but less than  $160/95\text{mmHg}$ ) – again, similar to present findings (stage I (moderate)

hypertension: females = 22%, males = 25%). Also consistent with present results were the findings of Mollentze *et al.* (1995), that 36.3% of urban black women, compared to 22.8% of men, presented with stage II hypertension ( $\geq 160/95$  mmHg). Seedat *et al.* (1992) in a study of urban blacks in Kwa-Zulu Natal, found that there was a *similar* prevalence of hypertension at the high level of risk (12.6% of males and 13.6% of females), but confirmed the finding that males had increased stage I risk (32% of males versus 25% of females).

Schutte *et al.* (2006) suggest that obesity is a powerful contributor to hypertension in black women, and therefore it is postulated that the raised obesity levels within the current sample may account for the higher prevalence of hypertension, particularly severe (stage II), within the female sample. Interestingly there were significant ( $p < 0.05$ ;  $r = 0.3$ ) correlations between SBP and DBP and BMI in both *males and females*, although the relationships themselves were weak. These relationships are consistent with past research (Tibazarwa *et al.*, 2009). Various studies throughout South Africa have highlighted hypertension as being the most prominent and indeed the most prevalent risk factor in black individuals, particularly males (van der Merwe & Pepper, 2006). Despite the fact that hypertension was found to be lowest in black males in the 1998 DHS, many community based studies have reported that hypertension is, for the most part, inadequately diagnosed and poorly treated in South Africa, particularly in this population group (DHS, 2003).

### **Factors influencing the development of hypertension**

The fact that the individuals assessed in the current study form part of an urban community may have impacted on the levels of hypertension recorded. Aligned with this, the development of hypertension has been linked in part to urbanization, and the associated lifestyle changes, including changes in diet and activity levels (Steyn *et al.*, 2006; Van Rooyen *et al.*, 2000). In accordance with present findings, Schutte *et al.* (2003) found greater levels of hypertension in more urbanized individuals. Dietary composition also has been shown to have an impact on levels of hypertension. In confirmation of this, within the present study, males who consumed less total fat had significantly ( $p < 0.05$ ) lower SBP ( $r = -0.2$ ) and DBP ( $r = -0.2$ ). Similarly, males who consumed less total protein, had significantly lower SBP and DBP ( $r = -0.2$  for both). Present findings confirm those of Schutte *et al.* (2003), that hypertensive individuals consumed a significantly higher percentage of dietary saturated fat and animal protein, while consuming significantly less dietary plant protein. Interestingly, in

the present study, in contrast to the findings in the male group, there were *no* significant relationships between either total fat or total protein intake and blood pressure within the female group. This suggests that diet had little effect on hypertension prevalence within this sample, and therefore other mechanisms such as obesity may have played a more important role.

Black hypertensive individuals have also been shown to be more sodium sensitive (Steyn, 2006; Schutte *et al.*, 2003), and associated with this, hypertension has been linked with increased sodium intake within this population group. Interestingly, in the present research there were no significant correlation between sodium intake and blood pressure in either males or females, despite the fact that 25% of males and 30% of females reported enjoying their food 'very salted', and both males and females consumed slightly more than the recommended 1.5g of Na<sup>+</sup> per day (Maseko *et al.*, 2006) (males = 2 grams, females = 1.53 grams) (Figure 23: Pg. 91). This is consistent with earlier findings that black individuals, in addition to having an abnormal transport mechanism for sodium, report a high intake of sodium, which is used by many individuals to preserve or flavor different foods (Steyn, 2006b; Seedat, 1996). Furthermore, South African bread reportedly has a much higher level of salt than that produced in other industrialised countries (DHS, 1998).

### **Hypertension and other CVD risks**

Within the current study, there were significant correlations between blood pressure (both SBP and DBP) and body mass in both males ( $r=0.3$ ,  $p<0.001$ ) and females ( $r=0.3$ ,  $p<0.05$ ), confirming the established relationship between body weight and raised blood pressure, which are proposed to be directly associated (Hankey & Leslie, 2001). Consistent with present findings, in a study by Schutte *et al* (2003) within a similar population group in the North West province, it was found that hypertensive individuals had significantly higher percentage body fat, while various other studies have noted a relationship between hypertension and obesity (Van Rooyen *et al.*, 2000; Steyn *et al.*, 1996; Mollentze *et al.*, 1995).

Similar findings were also presented in both the 1998 and 2003 DHS in which obesity prevalence was notably higher in hypertensive individuals - thus highlighting the association between the two (DHS, 1998). Interestingly, despite the significantly higher prevalence of

obesity within the current female sample, the relationship between obesity and hypertension did not appear to be any stronger. This may be linked to the suggestion that the harmful effects of obesity are less evident in black individuals than they are in individuals of other population groups. The exact reasons for this remain unclear, and to date little research has been carried out which has examined this relationship. However, it is thought that it may be linked to the fact that hypertension within black individuals is associated to a much greater degree than within other population groups, with a high sodium intake, excessive alcohol consumption (alcohol dependence), and a family history of hypertension or stroke (Steyn, 2006).

## HYPERCHOLESTEROLEMIA

The low prevalence of hypercholesterolemia in both males and females found within the current sample (Figure 15: Pg. 83), is consistent with provincial statistics; and while no national statistics are available, it has been reported that the prevalence of hypercholesterolemia is low within urban black individuals (Maritz, 2006; Seedat *et al.*, 1992). The mean total cholesterol values recorded are very similar to previous findings (Table XX: Pg. 118) and indicate that despite increases in urbanization, and the associated changes in lifestyle, this appears not to be impacting blood cholesterol levels. If anything, the cholesterol levels of the current study are slightly lower than values from 5 years back, however this difference is not that large. In addition, it can be seen that there are no notable differences in total cholesterol levels between the sexes, despite the high incidence of obesity within the female group. For the most part, blood cholesterol levels are shown to be *slightly* lower in males, and this is supported by the finding that black males have the lowest total cholesterol levels out of all population groups in the country (Maritz, 2006).

Table XX: Mean total cholesterol (mmol.L<sup>-1</sup>) of urban black individuals (35-54 years) within different provinces of South Africa, 1992-2010.

AUTHORS	YEAR	PROVINCE	FEMALES	MALES
Current study	2010	Eastern Cape	4.4	4.3
Vorster <i>et al.</i>	2005	North West	4.5	4.2
Oelofse <i>et al.</i>	1996	Western Cape	4.5	4.2
Mollenze <i>et al.</i>	1995	Free State	5.2	5
Seedat <i>et al.</i>	1992	Kwa-Zulu Natal	4.8	4.9

Only 2.1% of males and 3.4% of females had elevated total blood cholesterol levels, which categorised them as hypercholesterolemic ( $>6.2\text{mmol.L}^{-1}$ ). These results are very similar to findings from the 2003 DHS, in which self reported prevalence of hypercholesterolemia was notably low: in only 1.3% of males and 1.7% of females. Although no physical measurements of blood cholesterol were taken, it is likely that they would have followed this trend (DHS, 2003). This is supported by literature, which has suggested that it is indeed the low prevalence of dyslipidaemia within black South Africans that is paramount in establishing the concept of 'healthy obese' – particularly noteworthy in black women, who despite demonstrating a high prevalence rate of obesity, appear for the most part to be free of additional risk factors such as hypercholesterolemia or Type II diabetes (van der Merwe & Pepper, 2006). Specifically, this has been linked with a favourable serum lipid profile (low total cholesterol and high levels of high-density lipoprotein cholesterol) in black individuals (Kruger *et al.*, 2005), which may have a protective function against IHD. In light of this, black individuals are shown to have a less atherogenic fasting lipid profile than their white counterparts (Punyadeera *et al.*, 2001a). A limitation of this study was the fact that only total cholesterol was assessed, which means that a full lipid profile of individuals and any protective effects of HDL could not be ascertained. For this reason, the cholesterol findings should be viewed and interpretations of the data made with caution.

In the current study, a total cholesterol of  $5.2\text{--}6.2\text{mmol.L}^{-1}$  classified participants as 'borderline high', however, a total cholesterol  $>5\text{mmol.L}^{-1}$  is also used as a cut-off for the classification of hypercholesterolemia. Using this cut-off, 22% of males, and 26% of females were classified as hypercholesterolemic. This is similar to the reported prevalence of 20% and 30% in black males and females (30 years and older) respectively (Maritz, 2006), and demonstrates notable increases compared to findings from a decade ago, in which prevalence rates of around 12% were reported (Seedat *et al.*, 1992). Therefore, while high cholesterol ( $>6.2\text{mmol}$ ) appears to be rare, *moderately high* cholesterol ( $>5\text{mmol}$ ) seems to have increased. This is a worrying statistic given the fact that most CVD is reportedly associated with cholesterol and blood pressure levels *below thresholds* set for hypercholesterolemia and hypertension or obesity (Norman *et al.*, 2007).

Previous South African studies have reported positive correlations between elevated cholesterol and elevated blood glucose in black males and females (Tibazarwa *et al.*, 2009). In contrast to this, in the present study only *females* demonstrated a significant relationship

between total blood cholesterol and total blood glucose ( $r=0.3$ ,  $p<0.05$ ), indicating that those with elevated blood cholesterol had significantly elevated blood glucose as well. Although the relationship itself was weak, the significant relationship between these variables in female subjects points to a clustering of risks within these individuals. Since total cholesterol was also significantly associated with mass ( $r=0.2$ ,  $p<0.05$ ), BMI ( $r=0.2$ ,  $p<0.05$ ) and WC ( $r=0.2$ ,  $p<0.05$ ), in addition to total ( $r=0.2$ ,  $p<0.05$ ) and percentage body fat ( $r=0.3$ ,  $p<0.01$ ), it is likely that obesity played a significant role in predisposing these individuals to higher levels of total cholesterol, and in so doing, increasing their risk of CVD. Furthermore, in both sexes, there were significant relationships between total blood cholesterol and blood pressure (both SBP and DBP) – a finding which emphasizes that those who had higher total blood cholesterol levels, simultaneously had higher blood pressure. This again highlights the potential for risk factors for CVD to cluster in individuals, exponentially increasing their risk of disease.

## TYPE II DIABETES

The prevalence of Type II diabetes was notably low in the present study, in which no males and only 3% of females (Figure 16: Pg. 83) presented with elevated blood glucose levels (non-fasting blood glucose  $>12\text{mmol.L}^{-1}$ ). Despite the fact that no large scale national study has been conducted on the risk of type II diabetes in black South Africans, provincial research has demonstrated that despite rising levels of urbanization, particularly within black individuals, diabetes within this population group has remained low (Colditz *et al.*, 1995). The current results confirm this finding. Other provincial studies suggest similar results to the current findings, and show no increasing trend within the last 10 years. In 1992 Seedat *et al.* demonstrated a prevalence rate of 5% and 3% in males and females respectively of Kwa-Zulu Natal; and in 1995 Mollentze *et al.* found that only 6% of males and females from the Orange Free State were diabetic. A decade later, Alberts *et al.* (2005) demonstrated prevalence rates of 5.8% in males and 8.8% in females of the Limpopo and finally Vorster *et al.* (2005) found that only 0.8% of males and 5.1% of females from the North West presented with diabetes. Although Levitt *et al.* (1993) suggested an association between level of urbanization and diabetes, this appeared not to be the case in the current sample.

The low prevalence of type II diabetes within this population group has been linked to altered insulin resistance in black individuals. Literature has suggested that among black South African women, obesity does not significantly affect glucose tolerance, in contrast to non-

obese individuals (van der Merwe & Pepper, 2006). Aligned with this, black individuals are reported to have lower insulin resistance linked with lower levels of serum triglycerides, and thus tend to present with lower levels of diabetes (Seedat *et al.*, 1992). Interestingly, while there was no significant correlation between total blood glucose and BMI among women, there was a *significant correlation* between these variables in males ( $r=0.2$ ,  $p<0.05$ ). Kruger *et al.* (2001) found that total glucose correlated significantly with BMI ( $r=0.3$ ,  $p<0.05$ ) as well as with WC ( $r=0.4$ ,  $p<0.001$ ), while Omar *et al.* (1993) in a study of urban Zulu individuals found significant relationships between *fasting* glucose and BMI in women ( $r=0.2$ ,  $p<0.05$ ) and between *non fasting* glucose and BMI in men ( $r=0.2$ ,  $p<0.05$ ) (Alberts *et al.*, 2005). Levitt *et al.* (1999) reported that WC was an independent risk factor for type II diabetes – a finding that was confirmed by the results of Kruger *et al.* (2001). The findings of the present study are consistent with this: there were significant correlations between total glucose and WC in both males ( $r=0.3$ ,  $p<0.001$ ) and females ( $r=0.2$ ,  $p<0.05$ ), although this was not as marked among the females. Interestingly, glucose was significantly associated with SBP in females, however this was not the case in males. This is an important finding, given the understanding that SBP represents an important predictor of CVD, in addition to the finding that 48% of strokes in black women in SA can be attributed to raised SBP  $> 115\text{mmHg}$  (Norman *et al.*, 2007).

## LIFESTYLE-RELATED RISKS

### PHYSICAL ACTIVITY

As reflected in national statistics on urban black individuals from the 2003 DHS, as well as some provincial findings (Dugas *et al.*, 2009b), in the present study males were significantly more active than females (Table XIV: Pg. 84; Figure 17: Pg. 85). It is most likely because of the kinds of work that males are typically involved in - that more manual labour results in higher physical activity levels within these individuals. Despite the similar sex differences in physical activity, both *males and females* appeared to be notably *more active* in comparison to national findings. In **2003**, it was reported that **49.4%** and **66%** of urban black males and females were **inactive**, while only 22.2% of males and 12.4% of females were sufficiently active. In contrast to this, in the **current sample**, over half the males (51.3%) and 42.4% of the females were sufficiently active, while only **4.2%** and **14.4%** of males and females respectively were **inactive** (Figure 18: Pg. 85). The DHS (2003) further reported that physical

inactivity was higher in urban individuals, and linked this to increased risk of CVD within this population group. Although the current data do not appear to match this trend, it needs to be noted that urban national data encompass individuals from both the business as well as the informal sector. This inclusion of office-bound individuals within the 'urban black' cohort may have impacted on physical activity scores, as opposed to the current data in which most individuals were involved in more active job types (cleaning, gardening, operating of machinery, kitchen assistance). In addition, it has been reported that individuals who live close to the coast are more physically active (DHS, 2003). It is postulated that this 'coastal effect' may have had an impact on raised physical activity levels within this sample, given the fact that Grahamstown is only 30-40 minutes from the coast.

Further inconsistencies are noticeable when comparing domains of exercise with national findings (Figure 35: Pg. 123). Within the current sample, the majority of physical activity took place in the workplace (69% and 76% of total activity in males and females respectively) (Figure 20: Pg. 87), whereas in the DHS (2003) work-related physical activity contributed the *least* to total activity (19.9% for males and 19.7% for females). This is likely to be related to the fact that most participants were involved in occupations requiring movement throughout the course of the day, as opposed to office-based professions, which typify most urban occupations. This confirms the finding of Sparling *et al.* (1994) that black South African men, in particular, tend to engage in jobs that are physically demanding and require high levels of walking and physical activity: this they attribute to socio-economic conditions. In contrast, according to national statistics, leisure activity formed the bulk of daily physical activity (45.8% for males and 49.8% for females). While leisure activity was the second highest contributor to total activity in females (17%), it contributed the least in males (9%) within the current sample. Furthermore, while transport activity constituted 34.3% and 30.6% in males and females respectively in 2003, only 15% of total activity within the current sample (both males and females) was travel-related. Since national statistics take into account data from urban cities that are likely to be a lot larger than Grahamstown, it is likely the small distances required to travel in a small town may have accounted for these notable discrepancies.



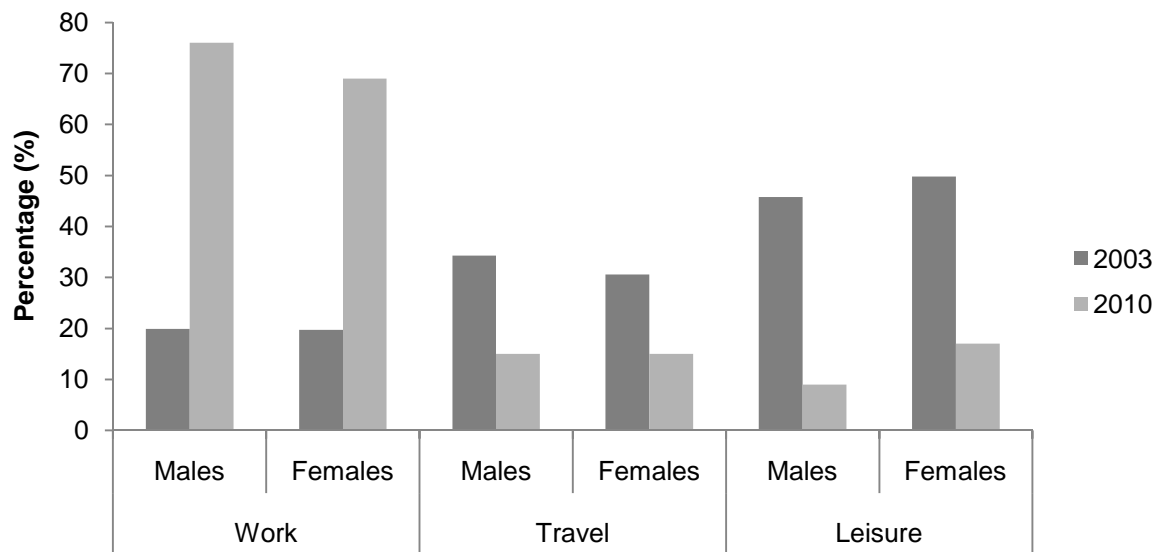


Figure 35: Contributions of different domains of exercise to total physical activity: comparison of current study trends with 2003 national statistic for urban black individuals (DHS, 2003)

Although it is claimed that GPAQ, IPAQ and other subjective measures of physical activity have under-estimated physical activity levels (as may have been the case in the 2003 DHS), and have lacked sensitivity in their measurement of daily habitual physical activity levels (Cook *et al.*, 2010), it is unlikely that this was the case in the current study. In fact, it is postulated that physical activity levels may indeed have been *over-estimated* or over-reported, given the high prevalence of obesity yet seemingly high physical activity levels. The concomitant lack of significant correlations between total physical activity (MET-mins/week) and BMI, mass or WC within the female sample in particular, highlights the seemingly absent effects of physical activity on these health measures, and thus possible over-reporting. Similar disassociations between physical activity and CVD risk factors were found by Malhotra *et al.* (2008), who reported no association between level of physical activity and risk of being overweight/obese in black township dwelling males and females. Similarly, Charlton *et al.* (2001), in a study of older South African individuals of mixed ancestry from the West Coast of the country, reported that while physical activity levels were higher in *females* compared to males, there were no significant relationships with any other CVD risks factors. However, they did report a significant negative relationship between past physical activity (in particular total moderate activity) and SBP, and therefore concluded the importance of assessing historical physical activity levels as opposed to current habitual levels. In terms of type of activity, it was reported that increased physical activity, in the form of housework, contributed to the increased physical activity levels within the female sample. Similarly,

Walker *et al.* (1989) noted that the physically active lifestyles of rural black women were surprisingly similar and comparable to those of urban township residents (Tshabangu & Coopoo, 2001), and this was linked to the lack of modern amenities within townships settings, which require subsistence activity (Cook *et al.*, 2009). This is confirmed in the results from the current study, in which females participated in significantly more physical activity at home than their male counterparts (contributing 17% to total activity compared to 9% in males). However, it is proposed that since this physical activity was mainly light to moderate in nature (as is typical of household chores) (Cook *et al.*, 2009), the health effects thereof may not have been felt, as evidenced in the high obesity levels paralleled with the presence of higher activity levels. Accordingly, it is proposed that the lack of vigorous activity may have induced the same effect generally typical of physical inactivity, evidenced in the high obesity prevalence within the female sample. Physical inactivity is a well established risk factor for chronic disease. It contributes to chronic energy imbalances, which form the foundation of chronic diseases such as obesity (Sparling *et al.*, 2000).

The predominance of moderate and light physical activity within the females, aligned with an apparent lack of *any* health benefits, and thus the importance of exercise *intensity* in reaping any CVD-related effect, is illustrated in part by the notably different findings among the males. Males were significantly ( $p<0.05$ ) more active, in addition to taking part in significantly ( $p<0.05$ ) more *vigorous* activity (Figure 19: Pg. 86). Accordingly, there were significant relationships between total physical activity (MET-mins/week) and reduced body fat, body fat percentage, BMI and WC, in addition to a significantly higher percentage lean body mass. *None* of these relationships was present in the female sample. In addition, total vigorous activity, was significantly associated with lower total blood cholesterol ( $r=-0.3$ ,  $p<0.05$ ), percentage body fat ( $r=-0.4$ ,  $p<0.01$ ) and total body fat ( $r=-0.3$ ,  $p<0.01$ ) in addition to higher percentage lean body mass ( $r=0.4$ ,  $p<0.01$ ). Interestingly, there was no significant relationship between total physical activity (MET-mins/week) or vigorous or moderate activity, and blood pressure (SBP or DBP), a finding that is supported by past research on black South Africans (Charlton *et al.*, 1997). The beneficial effects of *vigorous* activity on other CVD risks, are however supported in similar results by Dugas *et al.* (2009b) who found that males who engaged in notably more vigorous activity compared to their female counterparts, simultaneously had notably lower BMIs (males =  $21.6\text{kg.m}^{-2}$ , females =  $31\text{kg.m}^{-2}$ ), body mass (males = 64.1kg, females = 77.1kg) and percentage body fat (23.6% versus 43.5% for females). They subsequently suggested that vigorous activity might play a crucial role in

weight control within South Africa. In this regard, it is thought that while moderate intensity activities, such as walking, serve to reduce CVD risk, this reduction is even more pronounced when walking is combined with vigorous exercise (Manson *et al.*, 1999). Furthermore, it has been suggested that exercise of a higher intensity had a 'graded relation' to improved lipid concentrations in addition to improved insulin resistance (Manson *et al.*, 1999).

In 1983 Rossouw *et al.* reported that, compared to other population groups, the black population were the least inactive. The consensus since then has been that physical inactivity within this population group has increased. Aligned with this, Senekal *et al.* (2003), in a study of 550 economically active South Africans, found that self-reported inactivity constituted a major risk factor for being overweight and obese. These findings were confirmed by Kruger *et al.* (2002), who proposed that physical inactivity was a contributing factor to overweight and obesity in black females of the North West province. However, these authors also found a weak but *significant* relationship ( $r=0.054$ ,  $p=0.04$ ) between total energy intake and BMI (also evidenced in this study), highlighting the impact of *diet* on obesity, and tentatively emphasising the *more important role of diet* as a way of explaining the prevalence of obesity within this population group. It must be noted however, that since both diet and physical activity relied on self-reporting, which is known to have limitations in terms of accuracy and reliability (Pitta *et al.*, 2006), particularly in obese individuals, causal relationships may have been missed or misinterpreted.

## DIETARY INTAKE

Healthy eating forms the basis for achieving and maintaining cardiovascular health (Krauss *et al.*, 2000). McArdle *et al.* (2001) recommend that females should consume approximately 2000kcal per day and males, 3000kcal per day. According to these criteria, both males and females consumed notably *less* than the recommended daily amount (RDA). In fact in this study, males (2144kcal) consumed almost 1000kcal less than their RDA, and females (1713kcal) about 300kcal less (Figure 21: Pg. 88). This was an unexpected result, due to the high prevalence of obesity within both samples, particularly the females. Since obesity, at a very basic level, is the result of an energy imbalance, such that energy consumed exceeds that which is expended (Popkin *et al.*, 2006), it was expected that individuals would report dietary intakes that exceeded that which is recommended. Additionally, it was expected that there would be a strong relationship between energy intake and levels of obesity. However,

while there was a significant ( $p < 0.05$ ) negative correlation between total energy intake and mass ( $r = -0.2$ ) as well as between total energy intake and BMI ( $r = -0.2$ ) in *males*, no significant relationships were found within the female group, despite the significantly higher obesity levels.

These discrepancies with regard to reported total energy intake and obesity levels, may be due to *inaccurate and under-reporting* of energy intake, which is known to pose challenges for research into associations between dietary factors and health outcomes (van Dam & Seidell, 2007; Mendez *et al.*, 2004) and has been illustrated in other South African studies (Charlton *et al.*, 2005). Research has documented that Food Frequency Questionnaires (FFQs) and 24-hour dietary recalls (as were used in the current research project) tend to under-report energy intake. In addition, it has been shown that this under-reporting is often related to characteristics such as obesity, and that under-reporters are more likely to estimate low intake of food perceived as unhealthy compared to food perceived as healthy (Mendez *et al.*, 2004). Consistent with this, Charlton *et al.* (2001) found that 25% of mixed ancestry older women (over 55 years) reported consuming below two-thirds of their RDA. This was despite the high incidence of obesity within the sample (46%). Similarly, in a study by Mendez and colleagues (2004) on under- and over-reporting in Jamaican adults, it was found that under-reporting was more prevalent in obese individuals. Indeed, among normal, overweight and obese women, it was found that 25.3%, 36.7% and 57.1% under-reported, while in the same categories of men, under-reporting was found in 17.3%, 34.1% and 38.1% respectively. In addition it was found that women significantly under-reported intakes of sweetened dairy products, snack items and alcohol. Similarly, Kruger *et al.* (2002), in a study of urban black women in the North West province, found that obese black women tended to under-report to the greatest extent. Furthermore, when under-reporters were excluded from analyses, the power of the positive correlations between BMI and total energy intake increased (Kruger *et al.*, 2002). In accordance with this, it is proposed that in the present study the lack of any relationship between dietary intake and obesity within the females may be related to under-reporting, particularly within obese individuals who made up the majority of the female sample. This being said, however, it should also be acknowledged that there is a strong interplay between diet and physical activity: those who exercise more simultaneously need to eat more in order to have the required energy for physical activity. Therefore, while the males consumed significantly more than the females, they were also significantly more active, thus resulting in a significant negative correlation between total energy intake and BMI, and

similarly a significant negative correlation between total physical activity and BMI – relationships which were absent in females.

In terms of dietary composition, it is recommended that carbohydrates (CHO) should make up 55-65% of total calories, fat intake 15-30% (of which saturated fat should be limited to less than 10%), and protein intake 10-15% (Nishida *et al.*, 2004; McArdle *et al.*, 2001; Kraus *et al.*, 2000). In the present study both males and females reported dietary intakes which roughly resembled this composition, with males consuming slightly more protein (16%) and females slightly more fat (31%) than is recommended (Figure 22: Pg. 89). This is, for the most part, consistent with findings from other provincial studies (Table XXI: Pg. 128). Overall it can be seen that females tend to consume a higher percentage of fat than males. In addition, the females in the current study consumed a notably higher percentage of fat compared to females from other studies, a finding which may have influenced the higher obesity rates compared to provincial statistics. Both males and females, while within the RDA for CHO intake, appeared to consume a notably *lower* percentage compared to findings from other provinces, while alternatively consuming a slightly *higher* percentage of protein. The higher intake of protein, particularly within the male individuals, may be related to the finding that males tend to *over-report* protein intake: while women have been shown to randomly over-report protein intake, over-reporting in males is suggested to be common (Charlton *et al.*, 2001). Overall, unfortunately most dietary studies have taken place within the North West province and therefore it is difficult to make different inter-provincial comparisons or have a clear understanding of national trends.

Table XXI: Dietary composition of black urban males and females from various provinces of South Africa (1994-2010)

AUTHORS	YEAR	PROVINCE	F	M	F	M	F	M
			% FAT		% CHO		% Protein	
Current Study	2010	Eastern Cape	31	28	55	55	14	16
Vorster <i>et al.</i>	2005	North West	26	24	64	66	12	12
MacIntyre <i>et al.</i>	2002	North West (THUSA)	24	25	67	66	11	12
Kruger <i>et al.</i>	2002	North West (women)	26		63			10
Vorster	2002	North West (THUSA)	28	26	62	64	12	12
Vorster <i>et al.</i>	2000	North West	28	26	62	64	12	12
Bourne <i>et al.</i>	1994	Western Cape (M & F)	24-28		59-64		13-15	

Where: M refers to Males; F: Females; CHO: Carbohydrates

Looking to specific aspects of dietary intake (Table XV: Pg. 89), literature had linked raised body fat and level of obesity to the intake of high fat food items (Kruger *et al.*, 2002). Interestingly, in the present study *no correlation* was found between body weight or BMI and total fat intake in either sample. This is consistent with findings by Senekal *et al.* (2003). However, it is in contrast to the findings of Kruger *et al.* (2002), who demonstrated a significant correlation ( $r=0.05$ ,  $p=0.05$ ) between BMI and fat intake. However, it was highlighted that this correlation, while significant, was weak, thus supporting the notion that within South Africa fat intake does not promote obesity independently of total energy intake (Kruger *et al.*, 2002). In accordance with this, within the South African context specifically, it is proposed that the terms 'healthy obese' and 'unhealthy' obese are related to two different types of obesity: namely carbohydrate-induced and fat-induced obesity. Based on these distinctions, it has been suggested that severe obesity in black women is far less detrimental to overall health than in white women due to the fact that black women in general eat more CHO than fat, and vice versa among white women (Kruger *et al.*, 2001).

Interestingly, in the current study, within the *male* sample there was a significant ( $p<0.05$ ) negative correlation between total fat intake and both systolic and diastolic blood pressure ( $r=-0.2$ ), indicating a significant relationship between lower blood pressure values and higher fat intake. In contrast to this, there were no significant correlations between total fat intake and blood pressure in females - although fat constituted a higher percentage of total daily intake within this group. Females, on the other hand, consumed significantly more total and added sugar ( $p<0.05$ ) in comparison to their male counterparts. This is consistent with findings from

the BRISK study (Steyn, 2006b), in addition to a study by Steyn *et al.* (2000), both of which reported that urban black females consumed significantly more sugar than their rural counterparts, in addition to oil/fats (Steyn, 2006b). According to Fried and Rao (2003), increased dietary sugar (particularly sucrose and fructose) increases serum triacylglycerol concentrations by approximately 60%, which can lead to increased CVD risk. In the present study, in contrast to expectations, apart from SBP there was *no significant correlation* between increased sugar intake and any other CVD risk factor - highlighting the fact that while it is well established that sugar intake adds to weight gain and associated risk factors (van Dam & Seidell, 2007), this was not obviously the case in the current sample. This is similar to findings of Charlton *et al.* (2005), and may be linked to the high variance within the sample, or under-reporting of sugar intake, which may have confounded any significant relationships.

Fast food intake on the whole was low, with only 15% of males and 22% of females reportedly consuming fast foods (Figure 24: Pg. 92). This is similar to the findings of Kruger *et al.* (2006), who found that urban black children also reported a low intake of fast food. Unhealthy eating may be linked, in part, firstly to the role of the media, which promote the consumption of high fat fast foods, and secondly, to a nutritional ignorance which appears to exist among black urban individuals. Charlton *et al.* (2003) reported marked misconceptions as regards knowledge of healthy and unhealthy food types in urban black South African women and found that over half of the subjects classified polony (a high fat processed meat product) as a 'low fat' food, and rated potatoes as having no fat. Subjects also thought that eating more cheese (high in fat) would help them lose weight. Although perceptions of different foodstuffs were not assessed in the current research, it is postulated that ignorance with regard to healthy eating may have come into play, given the ignorance which existed in individuals about various health risks and a general lack of awareness in both males and females of general health.

## **SMOKING**

Consistent with national and provincial findings (DHS 1998 & 2003; Seedat, 1992), in this study there was a significant ( $p<0.01$ ) difference in smoking prevalence between the sexes, with a significantly higher percentage of males currently smoking (48% versus 3% of females) (Figure 25: Pg. 92). Although findings from the last two DHS indicated that smoking

prevalence had decreased, findings from the current study showed this to be the case with females only. However, the notable sex differences are clearly evident (Figure 36)

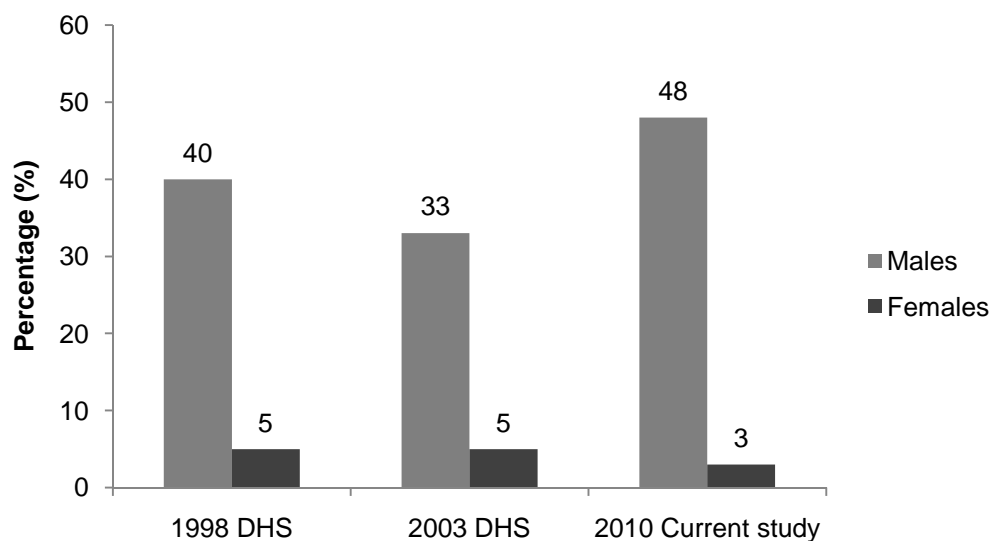


Figure 36: Smoking prevalence in black urban males and females, comparison of national statistics and current findings (1998-2010)

Provincial studies have similarly noted a sex difference in smoking prevalence among black individuals. In 1991 Steyn *et al.* found that within urban black individuals of the Cape Peninsula (Western Cape), 52% of males smoked in comparison to only 8.4% of females. A year later Seedat *et al.* (1992) demonstrated similar findings in urban Kwa-Zulu Natal with 28% of males and no females reportedly smoking. A decade later, Vorster *et al.* (2000) found that 28% of males and no females, from an urban population in the North West, smoked, while in 2005 Alberts *et al.* reported a smoking prevalence in 50.4% of males compared to 18% of females. The notable sex difference in smoking prevalence has been attributed to certain cultural norms and ideologies which exist within different African cultures in South Africa. For the most part, it is thought to be culturally unacceptable for black females to smoke, and therefore it is discouraged and even forbidden (Saloojee, 2006). This ideal appears to have remained within this population group despite westernised influences coming into play as these individuals become more urbanized and embedded within urban westernised society.

The smoking of more than 10 cigarettes per day is generally accepted as a strong risk factor for CVD (Rossouw *et al.*, 1983). In the present study, although just under half (48%) of the male sample smoked on a daily basis, they smoked a mean of only 5 cigarettes per day. In



addition, there were no significant correlations between the average number of cigarettes smoked and any of the other CVD risks. Therefore, while it is acknowledged that smoking is an important risk for CVD, within the current sample, it appeared that other risk factors such as diet and physical activity had more notable impacts on the cardiovascular health of this sample of individuals. A lack of heavy smoking has been confirmed by various studies in other provinces, in which smoking prevalence in males was relatively high, however the average number of cigarettes smoked was low (Alberts *et al.*, 2005; Mollentze *et al.*, 1995; Steyn *et al.*, 1991). Only one study found that a notable portion of males (28%) smoked heavily. Associated with this, they reported that smoking was the most frequent risk factor in black males (Seedat *et al.*, 1992).

In contrast to smoking prevalence, exposure to environmental smoke may in fact have constituted more of a health risk for these individuals. Self reported exposure to environmental smoke both at home and at the workplace was recorded, using similar questions as those used in the 2003 DHS (Figure 26: Pg. 93). If the data are compared, there was a similar amount of exposure to smoking at home for males and females (24% of males and 29% of females - compared to 26% of males and 25% of females in 2003). However, significantly ( $p < 0.05$ ) more males were exposed to smoke as well as fumes at the workplace (smoke at work = 45% of males and 18% of females, fumes at work = 50% of males and 19% of females). These findings are notably higher than those recorded in 2003 (smoke at work = 23% of males and 8% of females; fumes at work = 24% of males, 7% of females). This may be attributed to the fact that many of the males within the current sample were employed within a brick making company in which workers were exposed to a lot of dust and fumes. However, the high percentage of exposure within the male sample is a matter of concern, due to the associated risk of respiratory diseases, reported to be common in black working populations, particularly those involved in Manual Materials Handling (MMH) (Christie, 2001). Respiratory diseases, in addition to their individual health consequences, place additional strain on individuals, many of whom have existing CVD risk factors. The harmful effects of smoke and fume exposure on respiratory disease within South Africa are confirmed by the finding within the 1998 DHS that, among subjects who reported occupational exposure, there was a moderately strong correlation with limited airflow and chronic bronchitis (DHS 1998).

## ALCOHOL CONSUMPTION

Significantly ( $p < 0.05$ ) more males than females reported having consumed a drink in the previous 12 months. This is similar to findings from various other South African studies: Alberts *et al.* (2005) found that in a rural black population in the Limpopo province, more than 57% of the males drank, in comparison to only 16.5% of females, while similarly in the 2003 DHS it was shown that 38% of males compared, to only 14.2% of females, had consumed an alcoholic drink in the previous 12 months. This confirms the finding that alcohol consumption is notably higher amongst black South African males than females (DHS, 2003). In addition, findings from the current study confirmed the reported prevalence of binge drinking at the weekend (DHS, 2003) (Table XVI: Pg. 95). However, this was only the case within the *male* sample. During the week (Monday to Thursday) males consumed a mean of 0.1 drinks per day, compared to 2.8 drinks per day at the weekend (Friday to Sunday). These mean values appear low, however the standard deviations within the male sample were notably high, indicating that some individuals drank substantially more (Table XVI: Pg. 95). In the 2003 DHS it was reported that 28.6% of males and 29.2% of females were involved in hazardous drinking at the weekend (>6 drinks for males, >4 drinks for females). Thus, while the trend of increased alcohol consumption on the weekend is similar, not only do males within the current sample appear to drink notably less than according to national statistics, but females on the whole appear to drink notably less both during the week and during the weekend. In contrast, a higher percentage of females in the 2003 DHS, took part in weekend binge drinking. It is proposed that the notably lower prevalence of binge drinking in this study may be related to the manner in which individuals were interviewed. While interviews took place in private, the venue was a room on the worksite premises. This may have led individuals to inaccurately report lower alcohol intakes for fear of information getting to employers. In addition, excessive alcohol consumption in females is culturally unacceptable within many African cultures, including Xhosa. This is particularly the case for females, in whom it is reportedly seen as unattractive to consume alcohol.

A high intake of alcohol is strong risk factor for chronic diseases including strokes, diabetes and certain types of cancer (Schneider *et al.*, 2007). More than half of males (52%) and 39% of females were classified as alcohol dependent (CAGE questionnaire) (Figure 28: Pg. 95). This is notably higher than the prevalence of alcohol dependence reported by Schneider *et al.* (2007) who noted that 30% of males and 10% of females were alcohol dependent.

Findings from the current study are also notably higher than the alcohol dependence findings from the 2003 DHS (22.4% of males and 7.5% of females). In addition to various other CVD risks, it is well established that excessive alcohol intake leads to increased blood pressure (Temple, 2002). In this study, despite the high prevalence of alcohol dependence, there was no relationship between alcohol consumption and SBP or DBP or any other CVD risk. This may be related to a possible tendency of most individuals to feel guilty about drinking at all, and therefore responding positively (answering 'yes') to many of the alcohol dependence questions. It is proposed therefore, that individuals' responses may not have been a true reflection of their drinking habits in reality, and that there may have been an *over-reporting* of alcohol dependence – highlighted by the lack of association between alcohol intake and CVD risk.

### **Perceived versus actual measures of risks**

In addition to physical measures, self-reported prevalence of each risk was compared to measured values. In addition, individuals were asked about their perceptions of body weight; their responses were then compared to measured BMI classifications.

### **Self-reported versus actual hypercholesterolemia**

For the most part, it was found that individuals tended to under-report and underestimate their level of risk: when assessing levels of hypercholesterolemia, although prevalence of the condition was overall very low, self-reported prevalence was significantly lower than actual measures in both males and females (Figure 29: Pg. 96). In females, 16% were measurably hypercholesterolemic, while only 9% of individuals reported this to be the case. In males, although not as noticeable, 11% were measurably hypercholesterolemic, compared to only 3% who self-reported the condition. The low prevalence of hypercholesterolemia is consistent with findings of both the 1998 and 2003 DHS, although since no physical measures were actually taken (figures based entirely on self-reporting), it is likely that these values may in fact also be an underestimation of the condition. In 2003, 1.3% of males and 1.7% of females reported hypercholesterolemia, slightly more than was reported in 1998 (0.2% of males and 0.3% of females). It is conjectured that had total cholesterol been measured, similar trends of under-reporting would have been observed, particularly given the notably lower self-reporting of the condition in comparison to present findings.

## Self-reported versus actual type II diabetes

Consistent with the findings of Tibazarwa *et al.* (2009), individuals tended to *over-report* the prevalence of Type II diabetes (Figure 30: Pg. 97). Tibazarwa *et al.* (2009) reported that while 11% of individuals reported the condition, only 3.5% of males and 3% of females had measurably high total blood glucose levels. Consistent with this, in the current study while 10% of the male participants reported that they had been told by a health professional that they had elevated blood glucose levels, in no case was this measurably so. In addition, while only 2% of the females had elevated blood glucose levels, 14% of individuals reported that this was so. The fact that the rate of self-reporting of type II diabetes was higher among females than males is consistent with the findings of both the 1998 and 2003 DHS: In 1998, 3.7% of females reported the condition, compared to 1.6% of males, and similarly in 2003, while only 1.4% of males reported diabetes, 4% of females reported the condition. As with hypercholesterolemia, since no actual measures were taken, it is unclear whether these self-reports are an under- or over-estimation, although given current and previous findings, it is conjectured that the latter is the case.

## Self-reported versus actual hypertension

Hypertension presented the highest level of accuracy in terms of knowledge of the condition among the *females*. While 36% were classified as hypertensive, 35% reported this condition (Figure 31: Pg. 98). In contrast, males appeared more ignorant, with 17% reporting to be hypertensive in comparison to 32% who were measurably so. Within the DHS (1998 and 2003), similar underestimations were found. In 1998, while 21.5% of urban black males were hypertensive, 19.9% reported to be so; and in 2003, while only 7.6% of individuals reportedly were hypertensive, this was in fact the case in 11% of individuals. As with the current study, females appeared to be more accurate in their self-reporting of the condition. In contrast to this, according to the findings of Tibazarwa *et al.* (2009), hypertension was largely *overestimated*: while 80% of subjects reported being hypertensive, fewer than half of these individuals were measurably so (33% of males and 34% of females).

This underestimation and misconception of hypertension is not limited to black urban individuals, but is reportedly a national trend across all population groups (DHS, 1998). It has also been linked to an international concept known as the 'Rule of Halves', which was

proposed by Bannan *et al.* in 1981. This proposes that in most community surveys, half of all hypertensive patients are not known to health services, and of those individuals, half are not treated at all and half are not treated adequately (DHS, 1998). This may indeed account for the prevalence of under-reporting, particularly in males, highlighting the fact that either these individuals were misinformed or misdiagnosed. Either way, these notable misconceptions are a matter of concern, given the fact that hypertension is thought to represent the single most important CVD risk in black South African males (Marijon *et al.*, 2007).

### **Perceived versus actual prevalence of obesity**

Perhaps most notable was significant ignorance with regard to obesity levels. Obesity prevalence, while higher than the national prevalence rate, was largely not perceived (Figure 32: Pg. 99). In males, while more than half were classified as 'normal' according to their BMI, 44% were classified as overweight/obese. This stands in contrast to only 18.2% of individuals who perceived themselves as overweight. This misconception with regard to body weight was even more apparent in the female sample, in which 93.2% of individuals were classified as overweight/obese according to their BMI. Of concern was the fact that less than half (44/9%) of individuals perceived this to be the case.

These significant differences in perception of overweight and obesity, and the underestimation of body weight, have been highlighted by various other studies: In the 1998 DHS, nationally, while 15% of urban black women perceived themselves as being overweight/obese, this was in fact the case with 57% of individuals. In the follow-up survey, carried out in 2003, 18.8% of urban black women perceived themselves to be overweight, while in fact 60.9% were classified as overweight/obese (Figure 37: Pg. 136). Among male individuals, in 1998 6% of males perceived themselves to be overweight, while this was the case with more than double the number of individuals (19% were overweight and 10% obese), while in 2003, although the prevalence of overweight/obesity remained similar among urban black males (18.7% overweight, 8.1% obese), individuals' perceptions of being overweight decreased to 3.3%. In a study looking at nutrition knowledge of black urban females in the Western Cape and Gauteng, similar results were found, in which only 27% of the women who were classified as overweight and obese, perceived themselves to be so. In fact, most of these women considered their body size to be 'just right' (Charlton *et al.*, 2004). Furthermore, only a third of the participants agreed with the statement 'black women are

generally fatter than white women’, suggesting a distinct lack of awareness of the obesity problem within this population group (Charlton *et al.*, 2004).

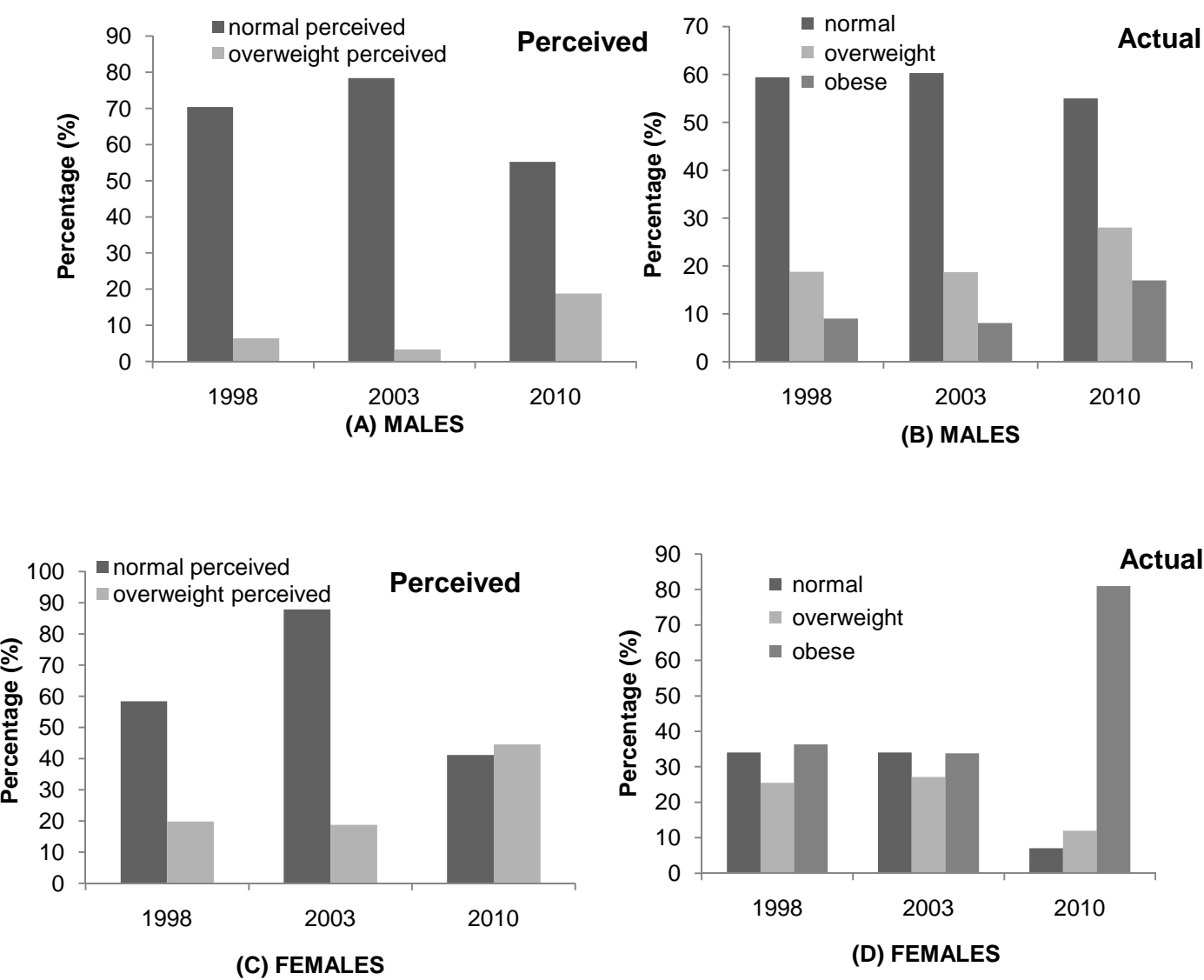


Figure 37: Perceived and actual prevalence of overweight and obesity in black urban males and females. Comparison of national data from 1998 and 2003, and current Eastern Cape data (from the present study).

The underestimation of body size was again evident when individuals were asked to identify which body shapes best illustrated their current body size (Figure 33: Pg. 100). Both males and females underestimated their body size and claimed to be smaller than their measured BMI values reflected. This is consistent with previous research findings, that larger body sizes are often seen to be more attractive (Ziebland *et al.*, 2002; Faber & Kruger, 2005; Case

& Menendez, 2009), and is thought to be largely associated with a mindset within African culture (particularly Xhosa) with regard to body weight and size which is very different from a mindset which prevails in western culture (Puoane *et al.*, 2002). For the most part, there are no negative connotations associated with overweight/obesity. In fact, the opposite appears to be true, whereby 'overweight' is seen to represent good health, prosperity, happiness, a husband's ability to care for his wife, and (in more recent times) the lack of HIV/AIDS infection (Case & Menendez, 2009; Charlton *et al.*, 2004). This mindset translates into the perception that overweight/obesity is 'healthy', and it is this misconception that influences subsequent behaviour (Faber & Kruger, 2005).

Interestingly, however, in this study while both males and females perceived themselves to be slimmer than they were in reality, females did not perceive this to be the most attractive body size. In contrast, they actually rated illustration 3 as the most attractive, which represented an even slimmer individual (Figure 33: Pg. 100). This is in contrast to findings from Case and Menendez (2009), who found that women rated larger body shapes than their own as 'ideal'. However, findings among the males were similar, with males in both studies rating their self-perceived size as being equal to the ideal size.

In contrast to the western mindset of slim being attractive, this ideal of 'bigger being better' which exists within South Africa makes attempts to promote healthy eating and living habits that much harder to implement. This is a matter of concern, given the increasing prevalence of obesity, particularly among black females (Senekal *et al.*, 2003). Perhaps future research and indeed health policy makers need to look into what mechanisms govern such belief systems and misinterpretations and find ways of educating and informing individuals about what is indeed healthy. Furthermore, attempts should be made to correctly diagnose individuals at risk, so that risk profiles within different population groups can be monitored and if at all possible controlled.

## CHAPTER VI

### CONCLUSIONS AND RECOMMENDATIONS

#### SUMMARY AND CONCLUSIONS OF PRESENT RESEARCH

Within the Eastern Cape Province specifically, research has shown CVD to be the second leading cause of mortality, accounting for 17% of all mortality within the province (Bradshaw *et al.*, 2000). Despite this, limited research has taken place in the province, in particular in the Makana area (in which Grahamstown is situated) for which there are currently *no* data available. Thus the aim of the current study was to assess CVD risk within a black urban working population in this area: more specifically to compare risk profiles between males and females, and compare these findings with self-reported risk. This chapter aims to summarise and conclude the findings of the current research project, and based on these, to propose recommendations for future research.

The current findings confirm and highlight the severity of obesity within black South African women. This is evidenced in the high percentage of female individuals who were classified as obese, in particular severely obese. In addition, the high mean waist circumference of the females highlights the prevalence of central adiposity within this population group. Although the males presented with significantly lower levels of obesity, noteworthy was the higher prevalence of obesity and central adiposity in comparison to national statistics for urban males. While BMI is criticised for its inability to distinguish between fat and fat-free mass (McArdle *et al.*, 2001), in this sample it provided a good indication of obesity prevalence - as evidenced in the significant and strong correlation between BMI and WC. Indeed, WC proved to be an accurate tool for the identification of central obesity, and due to its significant association with all other CVD risk factors in both samples, was a good indicator of overall CVD risk. Despite both samples eating less than their respective RDAs, and taking part in notably more activity than was reported in the latest DHS (DHS, 2003), the prevalence of obesity within this Eastern Cape sample of urban black individuals was notably higher than in national findings.

The figures for all other CVD risk factors assessed were fairly comparable with provincial and national figures (DHS, 1998, DHS, 2003; Alberts *et al.*, 2005; Tibazarwa *et al.*, 2009).



Hypertension, traditionally accepted as a prominent risk factor in black urban males (Schutte *et al.*, 2003), was present within this male sample. However, this was at a *moderate* level of risk. It is reported that hypertension, while being one of the most important and prevalent CVD risk factors, is associated for the most part with stroke among black individuals, which is one of the leading causes of mortality within this population group (Norman *et al.*, 2007). Despite this, its presence goes largely undetected and therefore is often untreated. It has therefore been coined ‘the silent epidemic’ within South Africa. While few associations between hypertension and other CVD risk factors such as obesity and hypercholesterolemia were found, of concern was the understanding that most disease associated with hypertension, particularly IHD and stroke, occurs in individuals with *suboptimal levels* of risks, below thresholds set for hypertension, hypercholesterolemia and obesity (Norman *et al.*, 2007). Therefore, individuals with only *moderately high* levels of any of these factors, may in fact be at substantially higher risk, risk that appears to go unnoticed and untreated. More females than males presented with *high risk* hypertension. This was almost certainly related to the high prevalence of obesity within the females, evidenced in the significant correlation between blood pressure and body weight. Females also presented with higher DBP levels, a known and important predictor of stroke in these individuals. The prevalence of hypertension in this sample (both males and females) was higher than in the national statistics reported for urban black individuals, reported in the last DHS. Since hypertension and obesity prevalence followed a similar trend, it is likely that these two risk factors were strongly associated and drivers of each other.

Hypercholesterolemia followed a similar trend to that found nationally (DHS, 2003), with findings demonstrating a low prevalence of the condition. However, there was a notably higher prevalence of *moderate* hypercholesterolemia, particularly among the females. This is most likely associated with increased obesity levels within the current sample. Like WC, hypercholesterolemia at a moderate level of risk appeared to be a significant indicator of CVD risk. This was illustrated by the significant relationships between total blood cholesterol and almost all other CVD risks. Type II diabetes was the least pertinent risk observed within this sample. Prevalence of the condition was low in both groups – consistent with national findings (DHS, 2003; DHS, 1998).

Smoking and alcohol consumption were not associated with any CVD risks and therefore within this specific sample appeared to be less important determinants of risk. In contrast,

diet and physical activity were the main two behavioural risks associated with increased CVD risk in both males and females. Diet was largely under-reported and therefore any relationships with CVD risks were compromised, but this phenomenon is not uncommon in obese individuals (van Dam & Seidell, 2007) and therefore was not entirely unexpected. While there were no associations between CVD risk factors and dietary intake within the female sample, in *males* there were significant *negative* relationships between total energy intake and BMI, as well as between total energy intake and percentage and total body fat – highlighting the role of physical activity necessitating the need for the males to consume more, yet simultaneously resulting in these individuals weighing less and demonstrating lower BMI values.

Similarly, physical activity did not appear to have any association with CVD risk in the female sample, yet within the males, who were significantly more active, positive health effects associated with exercise were evident: both total- and vigorous weekly physical activity were significantly associated with reduced BMI and body mass – again, relationships absent within the females. This lack of association is postulated to be due to the nature of the activity undertaken, which was for the most part light to moderate in nature (typical of domestic and household tasks) and thus insufficient in intensity to yield any health benefits. Similarly, within the literature it is suggested that while the positive effects of physical activity are well established, there remains inconsistency within the literature with regard to the *nature* of physical activity needed to achieve these effects (Malhotra *et al.*, 2008; Haapenen *et al.*, 1997). As evident within the current findings, while physical activity levels were notably higher than those reported nationally (DHS, 2003), there was little or no association with obesity levels within the female sample, in whom the prevalence rate was notably high. In this regard, it is proposed that in addition to the limitations of physical activity questionnaires in only being able to classify physical activity broadly, it is the *nature* and *intensity* of physical activity that play an important role in determining CVD risk. As evidenced in the male sample, which self reported significantly more total and *vigorous* activity, BMI, body mass and body fat percentage were significantly lower. The lack of any significance within the female sample suggests that the higher prevalence of light and moderate activity may have negated the positive effects of physical activity and these health variables. Aligned with present findings, it has been suggested that exercise intensity, in particular *vigorous* activity may play an important role in weight control in South Africa (Dugas *et al.*, 2009b). Furthermore, given the lack of association between physical activity and obesity in females, but the tentative link

between obesity and diet (CHO intake specifically), it has been proposed that physical activity alone may not be sufficient to yield any health benefits, and thus perhaps nutritional interventions may be more influential and effective (Malhotra *et al.*, 2008).

A worrying finding was the under-reporting and underestimation of almost all CVD risks. While obesity and hypertension were both higher in the current sample than in national statistics for black urban individuals (DHS, 2003), these were largely under-reported and underestimated. There appeared to be a distinct ignorance with regard to health risks, and this was similarly evidenced in the under-reporting of hypercholesterolemia and over-reporting of type II diabetes. In addition, many individuals appeared to have no opinion of their health status at all, and answered 'I don't know' to many questions on specific health risks. Since an individual's mindset and preconceived ideas tend to govern subsequent behaviour, it is these aspects that should be targeted so that positive change can be implemented. Educating individuals about healthy eating and living practices is thus crucial, particularly because, in addition to perception that 'overweight' represents good health and prosperity, many black women, in particular, believe that healthy food is expensive and therefore unobtainable. This in turn prevents healthy diets and promotes unhealthy eating practices which, in turn, facilitate and accentuate the existing obesity problem within this population group (Charlton *et al.*, 2004).

A particularly noteworthy finding in the current research was the prevalence of risks factors at a *moderate* level of risk. Stage I hypertension (according to SBP response) was more prevalent within the male sample, while more females presented with stage II hypertension (according to their SBP and DBP). Furthermore, a notable number of both males and females presented with moderate risk ( $>5\text{mmol.L}^{-1}$ ) hypercholesterolemia. Aligned with this, and of concern, is the understanding that *most* CVD risk is associated with cholesterol and blood pressure levels *below* thresholds set for hypercholesterolemia and hypertension – highlighting the fact that while individuals within the current sample may have presented with moderate levels of risk, their underlying risk of CVD may have been substantially increased. Furthermore, if these risk factors are combined, although some presented at a moderate level, this points to a clustering of risks within individuals. As illustrated in Figure 38, a clustering of risks is particularly notable in the females: While 38.5% of females had one risk factor, just under half (41.2%) had two risk factors (which tended to be obesity and hypertension). Within the males, just over half (51.7%) had no risks, while substantially fewer

had one (32.9% - which tended to be hypertension) or two (12.6%) combined risks. In addition, notably more females than males had three or more combined risks (8.1% versus 2.8%).

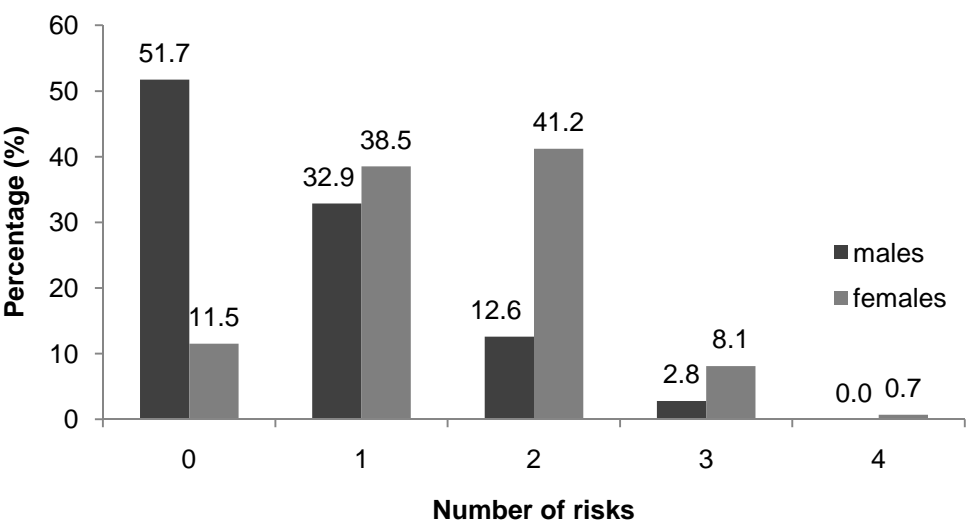


Figure 38: Combined risks in male and female participants (risks considered included obesity, hypertension, type II diabetes and hypercholesterolemia)

In this connection, it is well established that risk factors work synergistically rather than additively, and therefore it is the duration of exposure, particularly during childhood, which is important (Seedat *et al.*, 1992). Thus, while the prevalence of combined risk factors at a high level of risk may, at this stage, be relatively low, a clustering of risks is certainly notable, particularly within the females. Herein it is proposed that in a few generations time, when individuals currently forming part of the nutrition transition are fully embedded within the urbanized environment, it is likely that these risk profiles will change substantially towards multiple risks co-existing within individuals and mortality from CVD increasing.

### RECOMMENDATIONS

Since only limited data exist for the Eastern Cape, and there are currently *no data* for the Makana (Grahamstown) region, this current research project bridges a notable gap that currently exists in the literature. In this light it is proposed that findings from the current study are particularly important, given the notably higher prevalence of obesity illustrated in the

results in comparison to national trends. Aligned with these findings, it is recommended that future province-specific research be carried out (particularly in areas from which there are currently limited or no data available), allowing for specific trends across different population groups to be analysed - thus providing an overall perspective of CVD risk in the country as a whole. Small-scale studies such as these allow more for detailed sex/race specific information to be obtained, which large-scale epidemiological studies often cannot obtain due to financial and logistical constraints. Furthermore, it is recommended that where possible, accurate objective measures and blood samples should be *combined* with self-reported measures, so that the reliability and validity of data may be ensured. In addition, it is recommended that similar methodological tools be used (such as the STEP-wise questionnaires and methodology), to enable for cross-provincial comparisons to be made with ease, without methodological issues hindering comparisons and the identification of health trends. In addition, it is recommended that more detailed blood analyses should be carried out to enable a more conclusive understanding of the specific lipid and glucose related factors responsible for, and associated with, different risks. Indeed, it is proposed that future research should look at the *mechanisms* behind various CVD risks, particularly those associated with obesity within black women. Since obesity was particularly high in this sample of Eastern Cape men and (especially) women, it is recommended that future studies look into what possible inherent genetic traits may exist within these Xhosa individuals, which perhaps predispose them to higher adiposity levels in comparison to other South African population groups.

Results from this study, in addition to drawing attention to the problem and prevalence of obesity (particularly abdominal), highlight the importance of *lifestyle-related* and *behavioural drivers* of risk (particularly *diet* and physical *inactivity*), playing a key role in the promotion of overall CVD risk within black urban individuals. Herein it is recommended that studies assessing which *specific* behavioural aspects drive risk, would yield more conclusive information on behavioural modifications, which in turn could play a pivotal role in alleviating the burden of risk within these individuals. Implicit in this term, '*behavioural* induced CVD risk' is the idea that it can be modified, changed and indeed reduced, through educated and informed behavioural changes – changes which will only happen through comprehensive and informative education programs and intervention policies (Bradshaw *et al.*, 2007). Herein, a further recommendation for future research is to assess the effectiveness of educational intervention programs in alleviating the burden of risk within this population group.

Finally, given the notable ignorance with regard to health status, it is recommended that preconceived ideals of health also need to be challenged. In this connection, a greater understanding of Xhosa culture and ideals may aid in understanding perceptions of health, particularly obesity, and allow for interventions geared at changing perceptions to be implemented. It should be noted that the individuals in question are also facing a 'quadruple burden' of disease - a burden which plays a substantial role in altering perceptions, and shifting the focus of disease, from CVD (consequences and symptoms of which are often not immediately felt) to more pressing and immediate health risks, which other tiers of the burden (such as infectious diseases and HIV/AIDS) represent.

It is hoped that the knowledge and insights gained from this research project will help to identify which specific CVD risks pose the most serious threats to urban black individuals in the Eastern Cape, to create some understanding of the behavioural mechanisms governing and underpinning different risks, and to inform future research, so that in the face of many other disease risks and research challenges, the health status of these individuals may improve.

## REFERENCES

**Note:** Asterisked citations \* are secondary sources. These were not directly consulted and are referenced as fully primary sources, indicated in brackets, permit.

Abbott RD, Curb D, Rodriguez BL, Masaki KH, Yano K, Schatz IJ, Ross GW and Petrovitch H (2002). Age-Related Changes in Risk Factor Effects on the Incidence of Coronary Heart Disease. *Annals of Epidemiology*, 12: 173-181

Alberts M, Urdal P, Steyn K, Stensvold I, Tverdal A, Nel JH and Steyn NP (2005). Prevalence of cardiovascular disease and associated risk factors in a rural black population of South Africa. *European Journal of Cardiovascular Prevention and Rehabilitation*, 12: 347-354

Alm-Roijer C, Stagmo M, Uden G and Erhardt L (2004). Better knowledge improves adherence to lifestyle changes and medication in patients with coronary heart disease. *European Journal of Cardiovascular Nursing*, 3: 321-330

Armstrong T and Bull F (2006). Development of the World Health Organization Global Physical Activity Questionnaire (GPAQ). *Journal of Public Health*, 14: 66-70

Aspray TJ, Mugusi F, Rashid S, Whiting D, Edwards R, Alberti KG and Unwin NC (2000). Rural and urban differences in diabetes prevalence in Tanzania: the role of obesity, physical inactivity and urban living. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 94: 637-644

Baecke JA, Burema J and Frijters JE (1982). A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *American Journal of Clinical Nutrition*, 36: 936-942

Babor TF, Higgins-Biddle JC, Saunders JB and Monteiro M (2001). AUDIT. The Alcohol Use Disorders Identification Test. Guidelines for Use in Primary Care. Second Edition. *World Health Organisation*. Department of Mental Health and Substance Dependence

Balady GJ, Berra KA, Golding LA, Gordon NF, Mahler DA, Myers JN and Sheldahl LM (2000). American College of Sports Medicine. *ACSM's Guidelines For Exercise Testing and Prescription*. Sixth Edition. USA: Lippincott Williams & Wilkins

Bannan LT, Beevers DG and Jackson SH (1981). ABC of blood pressure management: Detecting hypertensive patients. *British Medical Journal*, 282: 1211-1213.

Baumgartner TA and Jackson AS (1999). *Measurement for Evaluation*. Sixth Edition. New York: The McGraw-Hill Companies, Inc.

Bhatnagar D and Durrington PN (1993). An evaluation of the Reflectron for the determination of plasma cholesterol in capillary blood. Effect of operator variability. *Occupational Medicine*, 43(2): 69-72

Bingham SA, Gill C, Welch A, Day K, Cassidy A, Khaw KT, Sneyd MJ, Key TJA, Roe L and Day NE (1994). Comparison of dietary assessment methods in nutritional epidemiology: weighted records v. 24 h recalls, food-frequency questionnaires and estimated-diet records. *British Journal of Nutrition*, 72: 619-643

Blakely T, Hales S, Kieft C, Wilson N and Woodward A (2005). The global distribution of risk factors by poverty level. *Bulletin of the World Health Organisation*, 83(2): 118-126

Bonita R, de Courten M, Dwyer T, Jamrozik K, Winkelmann R (2001). Surveillance of risk factors for noncommunicable diseases: The WHO STEPwise approach. Summary. *World Health Organisation*: Geneva

Booyesen F (2003). Chronic and transitory poverty in the face of HIV/AIDS-related morbidity and mortality: Evidence from South Africa. Paper presented at the *International Conference on "Staying Poor: Chronic Poverty and Development Policy"*, University of Manchester, 7-9 April 2003, United Kingdom

Bourne LT, Langenhoven ML, Steyn K, Jooste PL, Laubscher JA and Van der Vyver E (1993). Nutrient intake in the urban African population of the Cape Peninsula, South Africa. The Brisk Study. *Central African Journal of Medicine*, 39(12): 238-247



\* Bourne LT (1996). Dietary intake in an urban African population in South Africa – with special reference to the nutrition transition. *Ph.D Thesis*. University of Cape Town, Cape Town (see Steyn *et al.*, 2006)

Bourne LT, Lambert EV and Steyn K (2002). Where does the black population of South Africa stand on the nutrition transition? *Public Health Nutrition*. 5(1A): 157-162

Boutayeb A (2006). The double burden of communicable and non-communicable diseases in developing countries. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 100: 191-199

Bradshaw D, Nannan N, Laubscher R, Groenewald P, Joubert J, Nojilana B, Norman R, Pieterse D and Schneider M (2000). Mortality Estimates for Eastern Cape Province: South African National Burden of Disease Study. *Medical Research Council*: Cape Town

Bradshaw D and Steyn K (2001). Poverty and Chronic Diseases in South Africa. Technical report. *Medical Research Council*: Cape Town.

Bradshaw D, Groenewald P, Laubscher R, Nannan N, Nojilana B, Norman R, Pieterse D and Schneider M (2003). Initial Burden of Disease Estimates for South Africa, 2000. *South African Medical Research Council*: Cape Town

Bradshaw D, Schneider M, Norman R and Bourse D (2006). In: Steyn K, Fourie J and Temple N (2006). Chronic Disease of Lifestyle in South Africa: 1995-2005. Technical Report. *South African Medical Research Council*: Cape Town, South Africa

Bradshaw D, Norman R, Lewin S, Joubert J, Schneider M, Nannan N, Groenewald P, Laubscher R, Matzopoulos R, Nojilana B, Pieterse D, Steyn K, Vos T and the South African Comparative Risk Assessment Collaborating Group (2007). Strengthening public health in South Africa: Building a stronger evidence base for improving the health of the nation. *South African Medical Journal*, 97 (8): 643-649

Bray GA and Champagne CM (2005). Beyond Energy Balance: There Is More to Obesity than Kilocalories. *Journal of the American Dietetic Association*, 1 (5): S17-S23

Broxmeyer L (2004). Heart disease: the greatest 'risk' factor of them all. *Medical Hypotheses*, 62: 773-779

Brydon L, Magid K and Steptoe A (2006). Platelets, coronary heart disease, and stress. *Brain, Behavior, and Immunity*, 20: 113-119

Burnett RW, D'Orazio P, Fogh-Andersen N, Kuwa K, Kulpmann WR, Larsson L, Lewnstrom A, Maas AHJ, Mager G and Spichiger-Keller U (2001). International Federation of Clinical Chemistry and Laboratory Medicine. IFCC Recommendation on reporting results for blood glucose. *International Journal of Clinical Chemistry*, 307: 205-209

Candib LM (2007). Obesity and Diabetes in Vulnerable Populations: Reflection on Proximal and Distal Causes. *Annals of Family Medicine*, 5(6): 547-556

Cappuccio FP (1997). Ethnicity and cardiovascular risk: variations in people of African ancestry and South Asian origin. *Journal of Human Hypertension*, 11: 571-576

Case A and Menendez A (2009). Sex differences in obesity rates in poor countries: Evidence from South Africa. *Economics and Human Biology*, 7: 271-282

Charlton KE, Lambert EV and Kreft J (1997). Physical activity, change in blood pressure and predictors of mortality in older South Africans – a 2-year follow-up study. *South African Medical Journal*, 87 (9): 1124-1130

Charlton KE and Rose D (2001). Nutrition among Older Adults in Africa: the Situation at the Beginning of the Millenium. *Journal of Nutrition*, 131: 2424S-2428S

Charlton KE, Schloss I, Visser M, Lambert EV, Kolbe T, Levitt NS and Temple N (2001). Waist circumference predicts clustering of cardiovascular risk factors in older South Africans. *Cardiovascular Journal of South Africa*, 12(3): 142-150

Charlton KE, Brewitt P and Bourne LT (2004). Sources and credibility of nutrition information among black urban South African women, with a focus on messages related to obesity. *Public Health Nutrition*, 7 (6): 801-811

Charlton KE, Kolbe-Alexander TL and Nel JH (2005). Micronutrient dilution associated with added sugar intake in elderly black South African women. *European Journal of Clinical Nutrition*, 59: 1030-1042

Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, Jones DW, Materson BJ, Oparil S, Wright JT and Rocella EJ (2003). The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: The JNC 7 Report. *Journal of the American Medical Association*, 289 (19): 2560-2572

Chopra M, Galbraith S and Darnton-Hill I (2002). A global response to a global problem: the epidemic of overnutrition. *Bulletin of the World Health Organisation*, 80(12): 952-958

Christie CJ (2001). Consideration of the effect of nutritional status and disease patterns on work output amongst black South African workers involved in manual materials handling (MMH) tasks. *Ergonomics SA*, 13(1): 32-36

\* Clark RA, Niccolai L, Kissinger PJ, Peterson Y, Bouvier V (1999). Ethnic differences in body image attitudes and perceptions among women infected with human immunodeficiency virus. *Journal of the American Dietetic Association*, 99: 735–7.

Colditz GA, Willett WC, Rotnitzky A and Manson JE (1995). Weight gain as a risk factor for clinical diabetes mellitus in women. *Annals of Internal Medicine*, 122: 481-486

Cook I (2007). Physical activity in rural South Africa – are current surveillance instruments yielding valid results? *South African Medical Journal*, 97 (11): 1072-1073

Cook I, Alberts M and Lambert EV (2009). Development of a four-item physical activity index from information about subsistence living in rural African women: a descriptive, cross-sectional investigation. *International Journal of Behavioral Nutrition and Physical Activity*, 6:75

Cook I, Alberts M, Brits JS, Choma SR and Mkhonto SS (2010). Descriptive Epidemiology of Ambulatory Activity in Rural, Black South Africans. *Medicine and Science in Sports & Exercise*, 42 (7): 1261-1268

Coutsoudis A and Coovadia HM (2001). Nutrition activities in South Africa. *Nutrition Research*, 21: 459-463

Critchley J and Capewell S (2003). Smoking cessation significantly reduces overall mortality in people with coronary heart disease. *Evidence-based Healthcare*, 8: 36-37

Dalleck LC, Allen BA, Hanson BA, Borresen EC, Erickson ME and De Lap SL (2009). Dose-Response Relationship between Moderate-Intensity Exercise Duration and Coronary Heart Disease Risk Factors in Postmenopausal Women. *Journal of Women's Health*, 18 (1): 105-113

Dehghan M and Merchant AT (2008). Is bioelectrical impedance accurate for use in large epidemiological studies? *Nutrition Journal*, 7: 26 (<http://www.butritionj.com/content/7/1/26>)

Del Boca FK and Darkes J (2003). The validity of self-reports of alcohol consumption: state of the science and challenges for research. *Addiction*, 98 (Suppl 2): 1-12

Department of Health, South African Medical Research Council, Measure DHS+. 2002. *South Africa Demographic and Health Survey 1998*, Full Report. Pretoria: Department of Health.

Department of Health, Medical Research Council, OrcMacro. 2007. *South Africa Demographic and Health Survey 2003*. Pretoria: Department of Health.

Deurenberg P (1996). Limitations of the bioelectrical impedance method for the assessment of body fat in severe obesity. *American Journal of Clinical Nutrition*, 64 (suppl) 449S-452S

Doak CM, Adair LS, Bentley M, Monteiro C and Popkin BM (2005). The dual burden household and the nutrition transition paradox. *International Journal of Obesity*, 29: 129-136

D'Orazio P, Burnett RW, Fogh-Andersen N, Jacobs E, Kurwa K, Kulpmann WR, Larsson L, Lewenstam A, Maas A, Mager G, Naskalski JW and Okorodudu AO (2006). Approved IFCC

recommendations on reporting results for blood glucose. *Clinical Chemistry and Laboratory Medicine*, 44(12): 1486-1490

Drucker RF, Williams DR and Price CP (1983). Quality assessment of blood glucose monitors in use outside the hospital laboratory. *Journal of Clinical Pathology*, 36: 948-953

Dugas LR, Cohen R, Carstens MT, Schoffelen PFM, Luke A, Durazo-Arvizu RA, Goedecke JH, Levitt NS and Lambert EV. Total daily energy expenditure in black and white, lean and obese South African women (2009a). *European Journal of Clinical Nutrition*, 63: 667-673.

Dugas LR, Carstens MA, Ebersole K, Schoeller DA, Durazo-Arvizu RA, Lambert EV and Luke A. Energy expenditure in young adult urban informal settlement dwellers in South Africa (2009b). *European Journal of Clinical Nutrition*, 63: 805-807.

Ellsworth DL, Sholinsky P, Jaquish C, Fabsitz R and Manolio TA (1999). Coronary Heart Disease. At the Interface of Molecular Genetics and Preventive Medicine. *American Journal of Preventive Medicine*, 16(2): 122-133

Erasmus RT, Blanco E, Okesina AB, Mesa Arana J, Gqweta Z and Masha T (2001). Importance of family history in type 2 black South African diabetic patients. *Postgraduate Medical Journal*, 77: 323-325

Evans J, Collins M, Jennings C, van der Merwe L, Soderstrom I, Olsson T, Levitt N, Lambert EV and Goedecke JH (2007). The association of interleukin-18 genotype and serum levels with metabolic risk for cardiovascular disease. *European Journal of Endocrinology*, 157: 633-640

Faber M and Kruger S (2005). Dietary Intake, Perceptions regarding Body Weight, and Attitudes Toward Weight Control of Normal Weight, Overweight, and Obese Black Females in a Rural Village in South Africa. *Ethnicity & Disease*, 15: 238-245

Fang J, Wylie-Rosett J, Cohen HK, Kaplan RC and Alderman MH (2003). Exercise, Body Mass Index, Caloric Intake, and Cardiovascular Mortality. *American Journal of Preventive Medicine*, 25 (4): 283-289

Florindo AA and Latorre MDRDO (2003). Validation and reliability of the Baecke questionnaire for the evaluation of habitual physical activity in adult men. *Brazilian Society of Sports Medicine*, 9 (3): 129-135

Fodor JG and Tzerovska R (2004). Coronary heart disease: is gender important? *The Journal of men's health & gender*, 1 (1): 32-37

Foodfinder Dietary Analysis Software (2002). Release 1.10. Parrow: *Medical Research Council*: Cape Town

Frenk J, Bobadilla JL, Sepulveda J and Cervantes ML (1989). Health transition in middle-income countries: new challenges for health care. *Health Policy and Planning*, 4(1): 29-39

Fried SK and Rao SP (2003). Sugars, hypertriglyceridemia, and cardiovascular disease. *American Journal of Clinical Nutrition*, 78(suppl): 873S-880S

Galobardes B, Costanza MC, Bernstein MS, Delhumeau CH and Morabia A (2003). Trends in Risk Factors for the Major "Lifestyle-related Diseases" in Geneva, Switzerland, 1993-2000. *Annals of Epidemiology*, 13 (7): 537-540

Gaziano T (2007). Reducing The Growing Burden of Cardiovascular Disease In The Developing World. *Health Affairs*, 26 (1): 13-24

Ghoziladeh L and Davidson P (2008). More Similarities Than Differences: An International Comparison of CVD Mortality and Risk Factors in Women. *Health Care for Women International*, 29: 3-22

Gilbert L (1996). Urban violence and health – South Africa 1995. *Social Science and Medicine*, 43 (5): 873-886

Global Physical Activity Questionnaire (GPAQ). Department of Chronic Diseases and Health Promotion. Surveillance and Population-Based Prevention. *World Health Organisation*: Geneva, Switzerland. Accessed at [www.who.int/chp/steps](http://www.who.int/chp/steps)

Goedecke JH, Jennings CL and Lambert EV (2006). Obesity in South Africa. In: Steyn K, Fourie J and Temple N. Chronic Diseases of Lifestyle in South Africa: 1995-2005. Technical Report. *South African Medical Council: Cape Town*

Gray DS, Bray GA, Gemayel N and Kaplan K (1989). Effect of obesity on bioelectrical impedance. *American Journal of Clinical Nutrition*, 50: 255-260

Groenewald P, Vos T, Norman R, Laubscher R, van Walbeek C, Saloojee Y, Sitas F, Bradshaw D and the South African Comparative Risk Assessment Collaborating Group (2007). Estimating the burden of disease attributable to smoking in South Africa in 2000. *South African Medical Journal*, 97 (8): 674-681

Hankey CR and Leslie WS (2001). Nutrition and coronary heart disease. *Coronary Health Care*, 5: 194-201

Haslam DW and James WP (2005). Obesity. *The Lancet*, 366: 1197-1209

Henderson RM (2005). The bigger the healthier: Are the limits of BMI risk changing over time? *Economics and Human Biology*, 3: 339-366

Hickey J (2003). Waist circumference and cardiovascular risk. *Canadian Family Physician*, 49: 1287

Hill JO, Melby C, Johnson SL and Peters JC (1995). Physical activity and energy requirements. *American Journal of Clinical Nutrition*, 62 (suppl): 1059S-1066S

Hitzeroth HW (1996). Familial hypercholesterolemia in South Africa: to screen or not to screen? A national perspective. *Screening*, 4: 233-245

Hlatky MA, Lam LC, Lee KL, Clapp-Channin NE, Williams RB, Pryor DB, Califf RM and Mark DB (1995). Job Strain and the Prevalence and Outcome of Coronary Artery Disease. *Circulation*, 92: 327-333

Houtkooper LB, Lohman TG, Going SB and Howell WH (1996). Why bioelectrical impedance analysis should be used for estimating adiposity. *American Journal of Clinical Nutrition*, 64 (suppl): 436S-448S

Hu FB, Grodstein F, Hennekens CH, Colditz GA, Johnson M, Manson JE, Rosner B and Stampfer MJ (1999). Age at Natural Menopause and Risk of Cardiovascular Disease. *Archives of Internal Medicine*, 159: 1061-1066

IPAQ Committee Report (2002). *International Physical Activity Questionnaire: Long Last 7 Days Self-Administered Format*. Accessed online at <http://www.ipaq.ki.se>

IPAQ Committee Report (2005). *International Physical Activity Questionnaire*. Accessed online at <http://www.ipaq.ki.se>

James PT, Leach R, Kalamara E and Shayeghi M (2001). The Worldwide Obesity Epidemic. *Obesity Research*, 9 (suppl): 228S-233S

James PT (2004). Obesity: The Worldwide Epidemic. *Clinics in Dermatology*, 22: 276-280

Joubert J, Norman R, Bradshaw D, Goedecke JH, Steyn NP, Puoane T and the South African Comparative Risk Assessment Collaborating Group (2007a). Estimating the burden of disease attributable to excess body weight in South Africa in 2000. *South African Medical Journal*, 97 (8): 683-690

Joubert J, Norman R, Lambert EV, Groenewald P, Schneider M, Bradshaw D and the South African Comparative Risk Assessment Collaborating Group (2007b). Estimating the burden of disease attributable to physical inactivity in South Africa in 2000. *South African Medical Journal*, 97 (8): 725-731

Kalk WJ and Joffe BI (2007). Differences in coronary heart disease prevalence and risk factors in African and White patients with type 2 diabetes. *Diabetes Research and Clinical Practice*, 77: 107-112



Kehoe R, Wu S-Y, Leske C and Chylack LT (1994). Comparing Self-reported and Physician-reported Medical History. *American Journal of Epidemiology*, 139 (8): 813-818

Kesa H and Oldewage-Theron (2005). Anthropometric indications and nutritional intake of women in the Vaal Triangle, South Africa. *Public Health*, 119: 294-300

Krauss RM, Eckel RH, Howard B, Appel LJ, Daniels SR, Deckelbaum RJ, Erdman JW, Kris-Etherton P, Goldberg, IJ, Kotchen TA, Lichtenstein AH, Mitch WE, Mullis R, Robinson K, Wylie-Rosett J, St Joer, S, Suttie J, Tribble DL and Bazarre TL (2000). AHA Dietary Guidelines: Revision 2000: A Statement for Healthcare Professionals From the Nutrition Committee of the American Heart Association. *Stroke*, 31: 2751-2766

Kruger HS, Venter CS and Vorster HH (2001). Obesity in African women in the North West Province, South Africa is associated with an increased risk of non-communicable diseases: the THUSA study. *British Journal of Nutrition*, 86: 733-740

Kruger HS, Venter CS, Vorster HH and Margetts BM (2002). Physical inactivity is a major determinant of obesity in black women in the North West Province, South Africa: the THUSA study. *Nutrition*, 18(5): 422-427

Kruger HS, Puoane T, Senekal M and van der Merwe M-T (2005). Obesity in South Africa: challenges for government and health professionals. *Public Health Nutrition*, 8(5): 491-500

Kruger R, Kruger HS and MacIntyre (2006). The determinant of overweight and obesity among 10- to 15-year-old schoolchildren in the North West Province, South Africa – the THUSA BANA (Transition and Health during Urbanisation of South Africans; BANA, children) study. *Public Health Nutrition*, 9 (3): 351-358

Kruger HS, Pretorius R and Schutte AE (2010). Stunting, adiposity, and low grade inflammation in African adolescents from a township high school. *Nutrition*, 26: 90-99

Kyle UG, Bosaeus I, De Lorenzo AD, Deurenberg P, Elia M, Gomez JM, Heitmann BL, Kent-Smith L, Melchior J, Pirlich M, Scharfetter H, Schols, AMWJ and Pichard C (2004).

Bioelectrical impedance analysis – part II: utilization in clinical practice. *Clinical Nutrition*, 23: 1430-1453

Labadarios D and Steyn NP (2005). Nutritional disorders in Africa: The triple burden. *Nutrition*, 21: 2-3

Lambert EV, Bohlmann I, Kolbe-Alexander T (2001). 'Be Active' – Physical Activity for Health in South Africa. *South African Journal of Clinical Nutrition*, 14 (3 Suppl): S12-S16

Lambert EV and Kolbe-Alexander (2006). Physical Activity and Chronic Diseases of Lifestyle in South Africa. In: Steyn K, Fourie J and Temple N. Chronic Diseases of Lifestyle in South Africa: 1995-2005. Technical Report. *South African Medical Council*: Cape Town

Laporte RE, Montoye HJ and Caspersen CJ (1985). Assessment of Physical Activity in Epidemiological Research: Problems and Prospects. *Public Health Reports*, 100 (2): 131-146

Levitt NS (1996). Diabetes Mellitus in Black South Africans. *International journal of diabetes in developing countries*, 16: 41-44

Levitt NS, Katzenellenbogen JM, Bradshaw D, Hoffman MN and Bonnici F (1993). The prevalence and identification of risk factors for NIDDM in urban Africans in Cape Town, South Africa. *Diabetes Care*, 16: 601-607

Levitt NS, Steyn K, Lambert EV, Reagon G, Lombard CJ, Fourie JM, Rossouw K and Hoffman M (1999). Modifiable risk factors for Type 2 diabetes mellitus in a peri-urban community in South. *Diabetic Medicine*, 16(11): 946-950

Lim SS, Gaziano E, Gakidou E, Reddy KS, Fazarfar F, Lozano R and Rodgers A (2007). Prevention of cardiovascular disease in high-risk individuals in low-income and middle-income countries: health effects and costs. *Lancet*, 370: 2054-2062

Livingston MBE and Black AE (2003). Biomarkers of Nutritional Exposure and Nutritional Status. Markers of the Validity of Reported Energy Intake. *Journal of Nutrition*, 133: 895S-920S

Loock M, Steyn K and Becker P (2006). Coronary Heart Disease and Risk Factors in Black South Africans. *Ethnicity & Disease*, 16: 872-879

MacIntyre UE, Venter CS and Vorster HH (2000). A culture-sensitive qualitative food frequency questionnaire used in an African population: 2. Relative validation by 7-day weighted records and biomarkers. *Public Health Nutrition*, 4(1): 63-71

MacIntyre UE, Kruger HS, Venter CS and Vorster HH (2002). Dietary intakes of an African population in different stages of transition in the North West Province, South Africa: the THUSA study. *Nutrition Research*, 22: 239-256

Malhotra R, Ostbye T, Hughes G, Schwartz D, Tsolekile L, Zulu J and Puoane T (2008). Determinants of obesity in an urban township of South Africa. *South African Journal of Clinical Nutrition*, 21 (4): 315-320

Manson JE, Hu FB, Rich-Edwards JW, Colditz GA, Stampfer MJ, Willett WC, Speizer FE and Hennekens CH (1999). A prospective study of walking as compared with vigorous exercise in the prevention of Coronary Heart Disease in Women. *The New England Journal of Medicine*, 341: 650-658

Marijon E, Trinquart L, Jani D, Jourdier H, Garbarz E, Mocumbi AO, Celermajer DS and Ferreira B (2007). Coronary heart disease and associated risk factors in sub-Saharan Africans. *Journal of Hypertension*, 21: 411-414

Maritz F (2006). Diabetes Mellitus and Impaired Glucose Tolerance in South Africa. In: Steyn K, Fourie J and Temple N (2006). Chronic Disease of Lifestyle in South Africa: 1995-2005. Technical Report. *South African Medical Research Council*: Cape Town, South Africa

Maseko MJ, Majane HO, Milne J, Norton GR and Woodiwiss AJ (2006). Salt intake in an urban, developing South African community. *Cardiovascular Journal of South Africa*, 14 (4): 186-191

Mayosi BM, Fisher AJ, Lalloo UG, Sitas F, Tollman SM and Bradshaw D (2009). The burden of non-communicable diseases in South Africa. *Lancet*, 374: 934-947

McArdle WD, Katch FI and Katch VL (2001). *Exercise Physiology*. Fifth Edition. Pennsylvania: Lippincott Williams and Wilkins

Mciza Z, Goedecke JH, Steyn NP, Charlton K, Puoane T, Meltzer S, Levitt NS and Lambert EV (2005). Development and validation of instruments measuring body image and body weight dissatisfaction in South Africa mothers and their daughters. *Public Health Nutrition*, 8(5): 509-519

Mendez MA, Wynter S, Wilks R and Forrester T (2004). Under- and Overreporting of energy is related to obesity, lifestyle factors and food group intakes in Jamaican adults. *Public Health Nutrition*, 7(1): 9-19

Mezei G, Cher D, Kelsh M, Edinboro C, Chapman P and Kavet R (2005). Occupational Magnetic Field Exposure, Cardiovascular Disease Mortality, and Potential Confounding by Smoking. *Annals of Epidemiology*, 15: 622-629

Milne FJ and Pinkney-Atkinson VJ (2004). Hypertension Guideline 2003 Update. *South African Medical Journal*, 94: 209-226

Mollentze WF, Moore AJ, Steyn AF, Joubert G, Steyn K and Oosthuizen GM (1995). Coronary heart disease risk factors in a rural and urban Orange Free State black population. *South African Medical Journal*, 85: 90-96

Mollentze WF and Levitt NS (2006). Diabetes Mellitus and Impaired Glucose Tolerance in South Africa. In: Steyn K, Fourie J and Temple N (2006). Chronic Disease of Lifestyle in South Africa: 1995-2005. Technical Report. *South African Medical Research Council: Cape Town, South Africa*

\* Mvo Z, Dick J and Steyn K (1999). Perceptions of overweight African women about acceptable body size of women and children. *Curationis*, 22: 27-31.

Nakamaru N, Sato S and Shimamoto T (2003). Improvements in Japanese Clinical Laboratory Measurements of Total Cholesterol and HDL-cholesterol by the US Cholesterol

Reference Method Laboratory Network. *Journal of Atherosclerosis and Thrombosis*, 10(3): 145-153

Narayan KMV, Gregg EW, Fagot-Campagna A, Engelgau MM and Vinicor F (2000). Diabetes – a common, growing, serious, costly, and potentially preventable public health problem. *Diabetes Research and Clinical Practice*, 50: S77-S84

Nauck M, Warnick G and Rifai N (2002). Methods of Measurement of LDL-Cholesterol: A Critical Assessment of Direct Measurement by Homogenous Assays versus Calculation. *Clinical Chemistry*, 48 (2): 236-254

Nawi NG, Van Minh H, Tesfaye F, Bonita R, Byass P, Stenlund H, Weinehall L and Wall S (2006). Combining risk factors and demographic surveillance: Potentials of WHO STEPS and INDEPTH methodologies for assessing epidemiological transition. *Scandinavian Journal of Public Health*, 34: 199-208

Ndaba N and O'Keefe SJD (1985). The nutritional status of black adults in rural districts of Natal and Kwazulu. *South African Medical Journal*, 68: 588-590

Nel JH and Steyn NP (2002). Report on South African food consumption studies undertaken amongst different population groups (1983-2000): Average intakes of foods most commonly consumed. Directorate: Food Control, *Department of Health*: Pretoria, South Africa

Nicklas BJ, Dennis KE, Berman DM, Sorkin J, Ryan AS and Goldberg AP (2003). Lifestyle Intervention of Hypocaloric Dieting and Walking Reduces Abdominal Obesity and Improves Coronary Heart Disease Risk Factors in Obese, Postmenopausal, African-American and Caucasian Women. *Journal of Gerontology*, 58A (2): 181-189

Nishida C, Uauy R, Kumanyika S and Shetty P (2004). The Join 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

Norman R, Bradshaw D, Schneider M, Pieterse D, Groenewald P (2006). Revised Burden of Disease Estimates for the Comparative Risk Factor Assessment, South Africa 2000. Methodological Note. *South African Medical Research Council*: Cape Town

Norman R, Gaziano T, Laubser R, Steyn K and Bradshaw D (2007). Estimating the burden of disease attributable to high blood pressure in South Africa in 2000. *South African Medical Journal*, 97(8): 692-698

Oelofse A, Jooste PL, Steyn K, Badenhorst CJ, Lombard C and Bourne L (1996). The lipid and lipoprotein profile of the urban black South Africa population of the Cape Peninsular – the BRISK study. *South African Medical Journal*, 86: 162-166

O'Keefe SJD, Thusi D and Epstein S (1983). The fat and the thin – a survey of nutritional status and disease patterns among urbanized Black South Africans. *South African Medical Journal*, 63: 679-683

Omar MA, Seedat MA, Motala AA, Dyer RB and Becker P (1993). The prevalence of diabetes mellitus and impaired glucose tolerance in a group of urban South African blacks. *South African Medical Journal*, 83 (9): 641-643

Peer N, Bradshaw D, Laubscher R and Steyn K (2009). Trends in adult tobacco use from two South African demographic and health surveys conducted in 1998 and 2003. *South Africa Medical Journal*, 99 (10): 744-749

Pickering TG, Hall JE, Appel LJ, Falkner BE, Graves J, Hill MN, Jones DW, Kurtz T, Sheps SG and Roccella EJ (2005). Recommendations for Blood Pressure Measurement in Humans and Experimental Animals: Part 1: Blood Pressure Measurement in Humans: A Statement for Professionals From the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Circulation*, 111: 697-716

Pitta F, Troosters T, Probst VS, Spruit MA, Decramer M and Gosselink R (2006). Quantifying physical activity in daily life with questionnaires and motion sensors in COPD. *European Respiratory Journal*, 27: 1040-1055

Popkin BM (1998). The nutrition transition and its health implications in lower-income countries. *Public Health Nutrition*, 1(1): 5-21

Popkin BM (2002). Part II. What is unique about the experience in lower- and middle-income less-industrialised countries compared with the very-high-income industrialised countries? *Public Health Nutrition*, 5(1A): 205-214

Popkin BM and Gordon-Larsen P (2004). The nutrition transition: worldwide obesity dynamics and their determinants. *International Journal of Obesity*, 28: S2-S9

Popkin BM, Kim S, Rusev ER, Du S and Zizza C (2006). Measuring the full economic costs of diet, physical activity and obesity-related chronic diseases. *Obesity Reviews*, 7: 271-293

Prentice AM (2006). The emerging epidemic of obesity in developing countries. *International Journal of Epidemiology*, 35: 93-99

Poulter N (1999). Coronary Heart Disease is a Multifactorial Disease. *American Journal of Hypertension*, 12: 92S-95S

Puoane T, Steyn K, Bradshaw D, Laubscher R, Fourie J, Lambert V and Mbananga N (2002). Obesity in South Africa: The South African Demographic and Health Survey. *Obesity Research*, 10 (10): 1038-1048

Punyadeera C, van der Merwe M-T, Crowther NJ, Toman M, Schlaphoff GP and Gray IP (2001a). Ethnic differences in lipid metabolism in two groups of obese South African women. *Journal of Lipid Research*, 42: 760-767

Punyadeera C, van der Merwe M-T, Crowther NJ, Toman M, Schlaphoff GP and Gray IP (2001b). Weight-related differences in glucose metabolism and free fatty acid production in two South African population groups. *International Journal of Obesity*, 25: 1196-1295

Reimer HL, Elford RW and Shumak S (1991). Detecting Elevated Cholesterol Levels: how accurate is the reflectron? *Canadian Family Physician*, 37: 2361-2365

Riccardi G, Giacci R and Parillo (2003). Lifestyle modification to prevent type 2 diabetes. *International Congress Series*, 1253: 231-236

Richardson MT, Ainsworth BE, Jacobs DR and Leon AS (2001). Validation of the Stanford 7-Day Recall to Assess Habitual Physical Activity. *Annals of Epidemiology*, 11(2): 145-153.

Rimm EB (1996). Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine or spirits? *British Medical Journal*, 312: 731-736

Rippe JM, Crossley S and Ringer R (1998). Obesity as a chronic disease: Modern medical and lifestyle management. *Journal of the American Dietetic Association*, 98 (10 Suppl 2): S9-S15

Rossouw E, Du Plessis JP, Benade AJS, Jordaan PCJ, Kotze JP, Jooste PL and Ferreira JJ (1983). Coronary risk factor screening in three rural communities: The Coris baseline study. *South African Medical Journal*, 64(12): 430-436

Rush EC, Goedecke JH, Jennings C, Micklesfield L, Dugas L, Lambert EV and Plank LD (2007). BMI, fat and muscle differences in urban women of five ethnicities from two countries. *International Journal of Obesity*, 31: 1232-1239

Saloojee Y (2006). Tobacco Control in South Africa. In: Steyn K, Fourie J and Temple N (2006). Chronic Disease of Lifestyle in South Africa: 1995-2005. Technical Report. *South African Medical Research Council*: Cape Town, South Africa

Sankaranarayanan K, Chakraborty R and Boerwinkle (1999). Ionizing radiation and genetic risks VI. Chronic multifactorial diseases: a review of epidemiological and genetic aspects of coronary heart disease, essential hypertension and diabetes mellitus. *Mutation Research*, 436: 21-57

Schneider M, Norman R, Parry CDL, Bradshaw D, Pluddemann A (2007). Estimating the burden of alcohol abuse in South Africa in 2000. Methodological Note. *South African Medical Research Council*: Cape Town



Schutte AE, van Rooyen JM, Huisman HW, Kruger HS and de Ridder JH (2003). Factor analysis of possible risks for hypertension in a black South African population. *Journal of Human Hypertension*, 17: 339-348

Schutte AE, van Vuuren D, van Rooyen JM, Huisman HW, Schutte R, Malan L and Malan NT (2006). Inflammation, obesity and cardiovascular function in African and Caucasian women from South Africa: the POWIRS study. *Journal of Human Hypertension*, 20: 850-859

Seedat YK, Mayet FGH, Latif GH and Joubert G (1992). Risk factors and coronary heart disease in Durban blacks – the missing links. *South African Medical Journal*, 82: 251-256

Seedat YK (1996). Is the pathogenesis of Hypertension different in black patients? *Journal of Human Hypertension*, 3 (Suppl): S35-S37

Seftel HC (1978). The Rarity of Coronary Heart Disease in South African Blacks. *South African Medical Journal*, 54: 99-105

Senekal M, Steyn NP and Nel JH (2003). Factors associated with Overweight/Obesity in Economically Active South African Populations. *Ethnicity & Disease*, 13: 109-116

Shen W, Wang Z, Punyanita M, Lei J, Sinav A, Kral JG, Imielinska C, Ross R and Heymsfield SB (2003). Adipose tissue quantification by imaging methods: a proposed classification. *Obesity Research*, 11 (1): 5-16

Sitas F, Urban M, Bradshaw D, Kielkowski D, Bah S and Peto R (2004). Tobacco attributable deaths in South Africa. *Tobacco Control*, 13: 396-399

Sliwa K, Wilkinson D, Hansen C, Ntyintyane L, Tibazarwa K, Becker A and 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: 915-922

Sparling PB, Noakes TD, Steyn K, Jordaan E, Jooste PL, Bourne LT and Badenhorst C (1994). Level of physical activity and CHD risk factors in black South African men. *Medicine and Science in Sports and Exercise*, 26 (7): 896-902

Sparling PB, Owen N, Lambert EV and Haskell WL (2000). Promoting physical activity: the new imperative for public health. *Health Education Research*, 15(3): 367-376

Speakman JR (2004). Obesity. The Integrated Roles of Environment and Genetics. *Journal of Nutrition*, 134: 2090S-2105S

Statistics South Africa. 2003. Census 2001: Census in Brief. Report No. 03-02-03 (2001). *Statistics South Africa*: Pretorie

Statistics South Africa. 2006. Provincial Profile 2004. Eastern Cape. Report No. 00-91-02 (2004). *Statistics South Africa*: Pretoria

Steptoe A, Magid K, Edwards S, Brydon L, Hong Y and Erusalimsky J (2003). The influence of psychological stress and socioeconomic status on platelet activation in men. *Atherosclerosis*, 168: 57-63

Steyn K, Jooste PL, Bourne L, Fourie J, Badenhorst CJ, Bourne DE, Langenhoven ML, Lombard CJ, Truter H, Katzenellenbogen J, Marais M and Oelofse A (1991). Risk factors for coronary heart disease in the black population of the Cape Peninsular. The BRISK study. *South African Medical Journal*, 79: 480-485

Steyn K, Fourie J and Bradshaw D (1992). The impact of chronic diseases of lifestyle and their major risk factors on mortality in South Africa. *South African Medical Journal*, 82: 227-231

Steyn K, Fourie JM, Lombard CJ, Katzenellenbogen J, Bourne LT and Jooste PL (1996). Hypertension in the black community of the Cape Peninsula. The BRISK study. *East African Medical Journal*, 73: 756-760

Steyn K, Bourne L, Jooste P, Fourie JM, Rossouw K and Lombard C (1998). Anthropometric profile of a black population of the Cape Peninsula in South Africa. *East African Medical Journal*, 75: 35-40

Steyn NP, Senekal M, Brits S and Nel J (2000). Urban and rural differences in dietary intake , weight status and nutrition knowledge of black female students. *Asia Pacific Journal of Clinical Nutrition*, 9 (1): 53-59

Steyn K and Schneider M (2001). Overview on Poverty in South Africa. In: Bradshaw D and Steyn K. *Poverty and Chronic Diseases in South Africa*. Technical Report. 2001. ISBN: 1-919809-17-1. MRC online document

Steyn K, Levitt NS, Hoffman M, Marais AD, Fourie JM, Lambert EV, Gaziano TA, Kepe L and Lombard CJ (2004). The Global Cardiovascular Disease Risk Pattern in a Peri-Urban Working Class Community in South Africa. The Mamre Study. *Ethnicity & Disease*, 14: 233-242

Steyn NP, Labadarios D, Maunder E, Nel J and Lombard C (2005). Secondary anthropometric data analysis of the national food consumption survey in South Africa: The double burden. *Nutrition*, 21: 4-13

Steyn NP, Bradshaw D, Norman R, Joubert JD, Schneider M and Steyn K (2006). Dietary Changes and the Health Transition in South Africa: Implications for Health Policy. *South African Medical Research Council*: Cape Town, South Africa

Steyn K (2006a). Hypertension in South Africa. In: Steyn K, Fourie J and Temple N (2006). Chronic Disease of Lifestyle in South Africa: 1995-2005. Technical Report. *South African Medical Research Council*: Cape Town, South Africa

Steyn NP (2006b). Nutrition and Chronic Diseases of Lifestyle in South Africa. In: Steyn K, Fourie J and Temple N (2006). Chronic Disease of Lifestyle in South Africa: 1995-2005. Technical Report. *South African Medical Research Council*: Cape Town, South Africa

Striegel-Moore RH, Thompson DR, Affrenito SG, Franko DL, Barton BA, Schreiber GB, Daniels SR, Schmidt M and Crawford PB (2006). Fruit and vegetable intake: Few adolescent girls meet national guidelines. *Preventive Medicine*, 42 (3): 223-228

Strong KL and Bonita R (2004). Investing in surveillance: a fundamental tool of public health. Surveillance for Information and Policy Team, Chronic Disease and Health Promotion Department, Noncommunicable diseases and Mental Health Cluster, *World Health Organisation*: Geneva

Swart D and Panday S (2010). The Surveillance and Monitoring of Tobacco Control in South Africa. National Health Promotion R & D Group, Medical Research Council, *World Health Organisation*: Geneva

Talip W, Steyn NP, Visser M, Charlton KE and Temple N (2003). Development and Validation of a Knowledge Test for Health Professionals Regarding Lifestyle Modification. *Nutrition*, 19: 760-766

Temple NJ (2002). Nutrition and Disease: Challenges of Research Design. *Nutrition*, 18: 343-347

Third Report of the National Cholesterol Education Programme (NCEP) Expert Panel on the Detection, Evaluation & Treatment of High Cholesterol in Adults (Adult Treatment Panel III). Final Report (2002). *Circulation*, 106: 3143-3421

Thompson FE and Byers T (1994). Dietary Assessment Resource Manual. *Journal of Nutrition*, 124: 2245S-2317S

Tibazawa K, Ntyintyane L, Sliwa K, Gerntholtz T, Carrington M, Wilkinson D and Stewart S (2009). A time bomb of cardiovascular risk factors in South Africa: Results from the Heart of Soweto Study “Heart Awareness Days”. *International Journal of Cardiology*, 132: 233-239

Todaro JF, Shen B-J, Niaura R, Sprio III A and Ward KD (2003). Effect of Negative Emotion of Frequency of Coronary Heart Disease (The Normative Aging Study). *The American Journal of Cardiology*, 92: 901-906

Tremolleires FA, Pouilles IM, Cauneille C and Ribot C (1999). Coronary Heart Disease risk factors and menopause. A study in 1684 French women. *Atherosclerosis*, 142 (2): 415-423

\* Tshabangu EL and Coopoo Y (2001). Physical activity levels and health profiles of adult women living in informal settlements. *South African Journal for Research in Sport, Physical Education and Recreation*, 23: 27-36

Twisk JWR, Kemper HCG, Van Mechelen W and Post GB (2001). Clustering of Risk Factors for Coronary Heart Disease: The Longitudinal Relationship with Lifestyle. *Annals of Epidemiology*, 11 (3): 157-165

Van Dam RM and Seidall JC (2007). Carbohydrate intake and obesity. *European Journal of Clinical Nutrition*, 61 (Suppl 1): S75-S99

van der Merwe MT and Pepper MS (2006). Obesity in South Africa. *Obesity Reviews*, 7: 315-322

Van Rooyen JM, Kruger HS, Huisman HW, Wissing MP, Margetts BM and Venter CS (2000). An epidemiological study of hypertension and its determinants in a population in transition: the THUSA study. *Journal of Human Hypertension*, 14: 779-787

Venter CS, MacIntyre UE, Vorster HH (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): 205–18

Vieweg WVR, Tucker R, Bernardo NL and Dougherty LM (1998). *Medical Update for Psychiatrists*, 3(2): 49-52

Vorster HH (2002). The emergence of cardiovascular disease during the urbanization of Africans. *Public Health Nutrition*, 5 (1A): 239-243

Vorster HH, Wissing MP, Venter CS, Kruger HS, Kruger A, Malan NT, de Ridder JH, Veldman FJ, Steyn HS, Margetts BM and MacIntyre U (2000). The impact of urbanization on physical, physiological and mental health of Africans in the North West Province of South Africa: the THUSA study. *South African Journal of Science*, 96: 505-514

Vorster HH, Venter CS, Wissing MP and Margetts B (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(5): 480-490

\* Walker AR, Walker BF, Walker AJ and Vorster HH (1989). Low frequency of adverse sequelae of obesity in South African rural black women. *International Journal for Vitamin and Nutrition Research*, 59: 224-228

\* Walker AR (1991). Interethnic physiological and pathological diversities in southern African populations. *The Journal of the Royal College of Physicians*. 25(1): 16-20. In: Sparling PB, Noakes TD, Steyn K, Jordaan E, Jooste PL, Bourne LT and Badenhorst C (1994). Level of physical activity and CHD risk factors in black South African men. *Medicine and Science in Sports and Exercise*, 26 (7): 896-902

Walker ARP (1995). Nutrition-Related Diseases in Southern Africa: With Special Reference to Urban African Populations in Transition. *Nutrition Research*, 15 (7): 1053-1094

Walker ARP, Adam F and Walker BF (2001). World pandemic of obesity: the situation in Southern African populations. *Public Health*, 115: 368-372

Walker ARP, Walker BF and Adam F (2002). Variations in Occurrences of Nutrition-Related Diseases in Sub-Saharan African in Stages of Transition: What of the future? *Nutrition*, 18: 71-74

Walker ARP, Walker BF and Adam F (2003). Nutrition, Diet, Physical Activity, Smoking, and Longevity: From Primitive Hunter-Gatherer to Present Passive Consumer – How Far Can We Go? *Nutrition*, 19: 169-173

Wang Y and Wang QJ (2004). The Prevalence of Pre-hypertension and Hypertension Among US Adults According to the New Joint National Committee Guidelines. *Archives of Internal Medicine*, 164: 2126-2134

Wang Y, Rimm EB, Stampfer MJ, Willett WC and Hu FB (2005). Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. *American Journal of Clinical Nutrition*, 81 (3): 555-563

Warnick GR and Wood PD (1995). National Cholesterol Education Program Recommendations for Measurement of High-Density Lipoprotein Cholesterol: Executive Summary. *Clinical Chemistry*, 41 (10): 1427-1433

Weinsier RL, Hunter GR, Gower BA, Schulz Y, Darnell BE and Zuckerman PA (2001). Body fat distribution in white and black women: different patterns of intraabdominal and subcutaneous abdominal adipose tissue utilization with weight loss. *American Journal of Clinical Nutrition*, 74: 631-636

White HD and Dalby AJ (2008). Heart disease in Soweto: facing a triple threat. *The Lancet*, 371: 876-877

Wolf WM, Hunter DJ, Colditz GA, Manson JE, Stampfer MJ, Corsano KA, Rosner B, Kriska A and Willett WC (1994). Reproducibility and Validity of a Self-Administered Physical Activity Questionnaire. *International Journal of Epidemiology*, 23 (5): 991-999

World Health Organisation (1995). World Health Organization Expert Committee. Physical Status: The use and interpretation of anthropometry. (WHO Technical Report Series No. 854), *World Health Organisation*: Geneva

World Health Organisation (2000). International guide for monitoring alcohol consumption and related harm. Department of Mental Health and Substance Dependence, Noncommunicable Disease and Mental Health Cluster, *World Health Organisation*: Geneva

World Health Organisation. The WHO STEPwise approach to chronic disease risk factor surveillance (STEPS) (2001). *The World Health Organisation*: Geneva, Switzerland. Accessed at [www.who.int/chp/steps](http://www.who.int/chp/steps)

World Health Organisation (2004). Report on the Consultation on establishing regional guidelines on dyslipidaemia, obesity and diabetes. *World Health Organization: Regional Office for the Eastern Mediterranean*

World Health Organisation (2005). Report on establishing regional guidelines on dyslipidaemia, obesity and diabetes. *World Health Organisation: Regional Office of the Eastern Mediterranean, Cairo.*

World Health Organisation (2007). Prevention of Cardiovascular Disease: Guidelines for Assessment and Management of Cardiovascular Risk. *World Health Organisation: Geneva, Switzerland.*

Yach D, Hawkes C, Gould CL and Hofman KJ (2004). The Global Burden of Chronic Diseases. Overcoming Impediments to Prevention and Control. *Journal of the American Medical Association, 291(21): 2616-2622*

Yusuf S, Reddy S, Ounpuu S and Anand S (2001a). Global Burden of Cardiovascular Diseases: Part I: General Considerations, the Epidemiologic Transition, Risk Factors, and Impact of Urbanisation. *Circulation, 104: 2749-2753*

Yusuf S, Reddy S, Ounpuu S and Anand S (2001b). Global Burden of Cardiovascular Diseases: Part II: Variations in Cardiovascular Disease by Specific Ethnic Groups and Geographic Regions and Prevention Strategies. *Circulation, 104: 2855-2864*

Zhu S, Wang Z, Heshka S, Heo M, Faith MS and Heymsfield SB (2002). Waist circumference and obesity-associated risk factors among whites in the third National Health and Nutrition Examination Survey: clinical. Action thresholds. *American Journal of Clinical Nutrition, 76: 743-749*

Ziebland S, Robertson J, Jay J and Neil A (2002). Body image and weight change in middle age: a qualitative study. *International Journal of Obesity, 26: 1083-1091*



## **APPENDICES**

### **APPENDIX 1: SUBJECT INFORMATION AND CONSENT FORM**

#### **CARDIOVASCULAR RISK IN A BLACK URBAN POPULATION IN THE EASTERN CAPE, SOUTH AFRICA**

##### **Masters Research Project**

**Principal researcher: Lindsay Jackson**

Dear subject

Thank you for expressing an interest in this study. The aim of this project is to assess the prevalence of chronic disease within black women in the Eastern Cape, an area of research which has received very little attention in the past. Information gained from this project will therefore be invaluable in not only creating awareness of cardiovascular disease risk within the Eastern Cape women, but also help you in understanding your health status better.

Participation in this project will require you to initially have a few measurements taken, followed by an interview, in which you will be asked various questions about your lifestyle as well as dietary habits.

Measurements taken will include the following: Firstly your stature (height), mass (how much you weigh) and waist circumference. Following this we will take 3 measurements of your blood pressure, and thereafter measurements of your blood cholesterol (blood fat) and glucose (blood sugar). Measurements of blood fat and blood sugar will be done by making a small prick on your finger with a lancing device. A reading from the drop of blood formed will then be taken. This will aid in assessing how much fat and sugar you have in your blood, and in turn will help us determine how healthy your body is.

Once these measurements have been taken, you will be asked various questions regarding your lifestyle – the kind of work you do, the kinds of activities you are involved in, and the kinds of food

you eat. Please answer these questions as honestly as possible and feel free to ask at any time if you do not understand anything. Although this interview will be carried out in English, a copy of the questions will be available in both Xhosa and Afrikaans, and an interpreter will be present to ensure that you understand all questions fully.

Please note, that all information received from you will be treated confidentially, and will be coded for use. At no point in time will your name be used, and your information will not be released to any other individual without your written consent. Once we have analysed your information/data, this will become available to you. This will include detailed information in the form of a written report as to your cardiovascular health, and well as lifestyle habits which may currently impact negatively on your health. We trust that you will find this information helpful and educational, and that it will help you lead a healthy and risk free lifestyle going forward!

**Lindsay Jackson**

**Supervisor: Dr Candice Christie**

**Principal researcher**

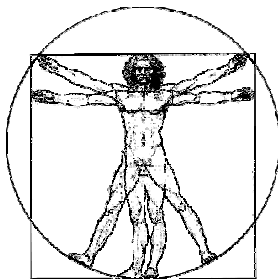
Tel [w]: 046 603 8470

Tel: 083 465 9869

Email: [c.christie@ru.ac.za](mailto:c.christie@ru.ac.za)

Email: [lindsayjac@gmail.com](mailto:lindsayjac@gmail.com)

## CONSENT FORM



I, ..... have been fully informed of the research project entitled:

### **CARDIOVASCULAR RISK IN A BLACK URBAN POPULATION IN THE EASTERN CAPE, SOUTH AFRICA**

I have read the information sheet and understand the testing procedure that will take place. I have been told about the risks as well as benefits involved, as well as what will be expected of me as a subject. I understand that all information gained from this project will be treated confidentially, that I will remain anonymous at all times and that data obtained may be used and published for statistical or scientific purposes. All testing procedures, associated risks and the benefits from partaking in this study have been verbally explained to me as well in writing. I have had ample opportunity to ask questions and to clarify any concerns or misunderstandings. I am satisfied that these have been answered satisfactorily.

In light of this, and in agreeing to participate in this study, I accept joint responsibility together with the Human Kinetics and Ergonomics Department, in that should any accident or injury occur as a direct result of the protocols being performed during the study, the Human Kinetics and Ergonomics Department will be liable for any costs with may ensure and will reimburse the subject to the full amount. I.e. doctors consultation, medication etc. The department will, however, waiver any legal recourse against the researchers of Rhodes University, from any and all claims resulting from personal injuries sustained whilst partaking in the investigation due

negligence on the part of the subject or from injuries not directly related to the study itself. This waiver shall be binding upon my heirs and personal representatives.

I have read and understood the above information, as well as the information provided in the letter accompanying this form.

***I therefore consent to voluntarily participate in this research project.***

**SUBJECT (OR LEGAL REPRESENTATIVE):**

_____	_____	_____
(Print name)	(Signed)	(Date)

**PERSON ADMINISTERING INFORMED CONSENT:**

_____	_____	_____
(Print name)	(Signed)	(Date)

**WITNESS:**

_____	_____	_____
(Print name)	(Signed)	(Date)

## APPENDIX 2: QUESTIONNAIRES AND DATA SHEETS



### **PROJECT TITLE: CARDIOVASCULAR RISK IN A BLACK 'URBAN' WORKING POPULATION IN THE EASTERN CAPE, SOUTH AFRICA**

#### **BACKGROUND QUESTIONNAIRE**

Please answer the following questions honestly and to the best of your understanding

<b>Age</b>		<b>Sex *</b>		<b>Business/institution</b>	
<b>Home language of respondent **</b>				<b>Language of interview **</b>	

\* Sex codes: 01 MALE 02 FEMALE

\*\* Language codes: 01 ENGLISH 02 AFRIKAANS 03 isiXHOSA

	<b>EDUCATION</b>					
	I'm first going to ask you some questions about your education – so if you went to school or are currently attending school					
1	Have any people living in your house ever been to school, or are currently attending school?					
	Yes (1)		No (2)		Don't know (8)	
2	If <b>yes</b> , what is the highest level of school completed?					
	Less than 1 year completed (00)			Grade 1 (01)		Grade 2 (02)
	Grade 4 (04)		Grade 5 (05)		Grade 6 (06)	Grade 7 (07)
	Grade 9 (09)		Grade 10 (10)			Grade 11 (11)
	Further studies incomplete (13)			Diploma/other post school complete (14)		
	Further degree complete (15)			Don't know (98)		
	<b>DEMOGRAPHIC INFORMATION</b>					
	The next three questions are about your job and your house					
3	Do you work for payment?				Yes (1)	No (2)
4	How many people live in your house?				Number	
5	Do you have electricity at home?				Yes (1)	No (2)

MEDICAL CONDITIONS							
						Yes(1)	No (2)
Have you had your blood pressure measured in the past 12 months?							
Do you know what your blood pressure is?							
Is it high, normal or low? Please tick the relevant box.							
Normal (2)			Low (3)			Don't know (8)	

Do you personally think that you are underweight, normal weight or overweight?							
Underweight (1)			Normal weight (2)			Overweight (3)	
						Don't Know (8)	
Would you say your health is poor, average, good, or very good/excellent?							
Poor (1)			Average (2)			Good (3)	
						Very good/Excellent (4)	
Has a <b>doctor</b> or <b>nurse</b> or <b>health worker</b> at a <b>clinic</b> or <b>hospital told you</b> that you have or have had any of the following conditions?						Yes (1)	No (2)
High blood pressure?							
High blood cholesterol or fats in the blood?							
Diabetes or blood sugar?							

DIETARY INTAKE											
Now I would like to ask you some questions about the foods you eat. Please try and answer the questions as honestly as possible Which of the following do you <b>usually</b> eat?											
15A	<b>Chicken/Poultry</b>			With skin (1)			Without skin (2)			None (3)	
15B	<b>Red Meat</b>			Fatty meat (1)			Lean meat (2)			None (3)	
15C	<b>Spread (butter/margarine)</b>	Butter (1)		Hard margarine (brick) (2)			Soft margarine (3)			None (4)	
15D	<b>Milk/Milk products</b>	Full cream (1)		2%/low fat (2)			Skim/Fat free (3)			Blends (4) None (5)	
How often do you <b>usually</b> eat the following?											
16A	<b>Fried foods</b> (chips, fish, potatoes, doughnuts, eggs)			Occasionally/never (1)			At least once a week (2)			Daily (3)	
16B	<b>Chips</b> (chips/crisps, niknaks, or other salty snacks)			Occasionally/never (1)			At least once a week (2)			Daily (3)	
16C	Processed meat, e.g. polony, viennas, meat pies, sausage rolls			Occasionally/never (1)			At least once a week (2)			Daily (3)	
16D	How salty do you usually eat your food?		Very salted (1)			Lightly salted (2)			Not salted (3)		Don't know (8)
Do you usually add salt or <b>Aromat/Fondor</b> to your food? If <b>yes</b> , do you add this before or after tasting the food?											
		No, I never add salt/aromat (1)			Yes, but I taste first and then add (2)			Yes, even before having tasted food (3)			Don't know (8)
17	Do you eat <b>salty snacks</b> (such as chips, niknaks, salted peanuts, salty biscuits, biltong, dried sausage, dried fish) more often than <b>3 times per week</b> ?							Yes (1)			No (2)
18	Do you eat <b>fast foods</b> – or any of the following, more than once per week?										
	KFC(1)		Steers (2)		Mr Burger (3)		Nandos (4)		Debonairs (5)		
			Chicken Licken (6)		Whimpy (7)		MacDonalds (8)		None (9)		

**DIETARY INTAKE:** Now I would like to ask you some questions about the foods that you eat. Please think back over the last 24 hours, and try and tell me as accurately as possible, everything that you ate:

Time	Type of food and/or drink and how you made it	Quantity	Eaten at Home (H) or at Work(W)
<b>BEFORE BREAKFAST</b>			
<b>BREAKFAST</b>			
<b>MID-MORNING: Between breakfast and lunch</b>			
<b>LUNCH</b>			
<b>MID-AFTERNOON: Between lunch and dinner</b>			
<b>DINNER</b>			
<b>AFTER DINNER</b>			

Name: \_\_\_\_\_

Day of the week \_\_\_\_\_

Is this a typical day? \_\_\_\_\_ (Yes or No)



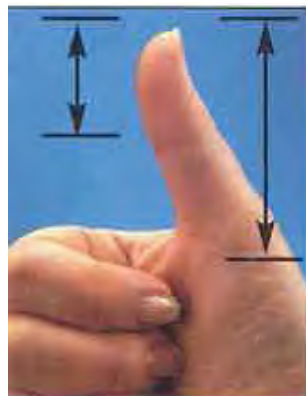
## PORTION SIZES



The size of your fist refers to a medium sized fruit or vegetable  
(i.e. apple, orange, potato)



The size of your fingertip refers to one teaspoon  
(i.e. sugar, butter on bread)



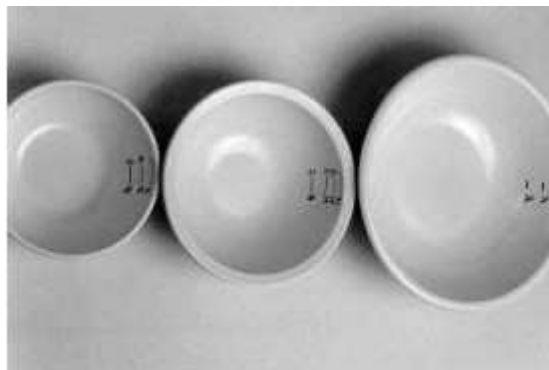
The size of your thumb refers to one tablespoon



Your cupped hand refers to approximately 45 g  
(i.e. a cupped hand of chopped vegetables or rice)



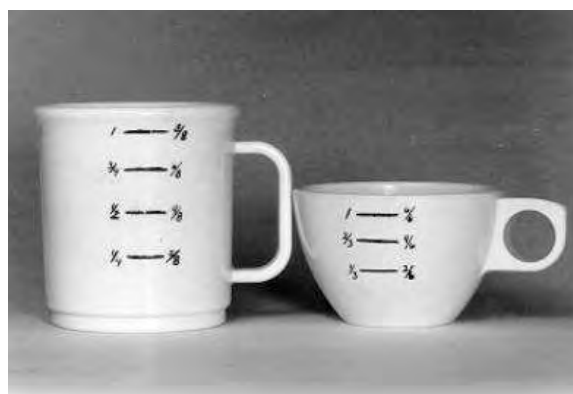
The palm of your hand refers to a medium portion of cooked meat,  
poultry or fish



Small

Medium

Large



Mug

Cup

ALCOHOL USE									
I'm now going to ask you a few questions about any alcohol you may drink. Alcoholic drinks include things like beers, wine and spirits.									
								Yes (1)	No (2)
1	Have you <b>ever consumed</b> a drink that contains alcohol such as beer, wine, spirits or sorghum beer? IF NO GO TO NEXT QUESTIONNAIRE								
2	Was this within the <b>past 12 months</b> ?								
3	In the <b>past 12 months, how frequently</b> have you had at least 1 drink?								
	5 or more days a week (1)			1-4 days per week (2)			1-3 days per month (3)		
	Less than once a month (4)								
4	When you drink alcohol, <b>on average</b> , how many drinks do you have during one day?								
					Drinks		Don't know (98)		
5	During the <b>past 7 days</b> , how many standard drinks of any alcoholic drink did you have each day?								
	Monday		Tuesday		Wednesday		Thursday		Friday
					Saturday		Sunday		
	<b>ALCOHOL DEPENDENCE : CAGE</b>								
								Yes (1)	No (2)
1	Have you ever thought that you should <b>cut down</b> on your drinking?								
2	Have people <b>annoyed</b> you by criticizing your drinking?								
3	Have you ever felt <b>guilty</b> about your drinking?								
4	Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover?								

TOBACCO USE									
I'm now going to ask you some questions about your smoking habits, Please answer the questions as honestly as possible									
								Yes (1)	No (2)
1	Do you currently smoke any tobacco products such as cigarettes, cigars or pipes? IF NO, GO TO Q6								
2	Do you currently smoke tobacco products <b>daily</b> ? IF NO, GO TO Q6								
3	How old were you when you first started smoking daily?	Years old			Don't remember (98)				
				Weeks ago (1)	Months ago (2)		Years ago (3)		
4	Do you remember how long ago it was when you first started smoking daily?								
5	On average, how many of the following items do you smoke each day?								
	Manufactured cigarettes		Hand-rolled cigarettes			Pipes full of tobacco			
				Cigars/cheroots/cigarillos					
6	In the past, did you ever smoke daily? IF NO, GO TO Q9				Yes (1)			No (2)	
7	How old were you when you stopped smoking daily?	Years old			Don't remember/not sure (98)				
8	Do you remember how long ago it was when you stopped smoking daily?								
	Weeks ago (1)			Months ago (2)			Years ago (3)		
					Yes (1)		No (2)		
9	Do you currently use any smokeless tobacco, such as snuff or chewing tobacco? IF NO GO TO Q12								
10	Do you currently use smokeless tobacco <b>daily</b> ? IF NO GO TO Q12								
11	On average, how many times do you use each of the following items per day								
	Snuff (by mouth)		Snuff (by nose)			Chewing tobacco			
					Yes (1)		No (2)		
12	In the past, did you ever use smokeless tobacco such as snuff or chewing tobacco daily?								
13	Do you live in a house where other people smoke cigarettes regularly?								
14	Do you currently work in a job where other people smoke cigarettes around you?								
15	Have you ever worked in a job where you were regularly exposed to smoke, dust, fumes or strong smells?								
16	Is yes, how long did you work in that job? (IF LESS THAN 1 YEAR WRITE 00)				Years				

**HABITS AND LIFESTYLE – PHYSICAL ACTIVITY :** I'm now going to ask you a few questions about time you spend doing different types of physical activities. This includes activities you do **at home, at work, traveling from place to place and at home**. Please try and answer the questions as honestly as possible.

**OCCUPATION-RELATED PHYSICAL ACTIVITY (PAID OR UNPAID WORK):** When answering the following questions, think back over the **past 12 months** and think of **a usual week**.

1	Does your work involve <b>mostly</b> sitting or standing still, <b>OR</b> walking for very short periods (less than 10 minutes) or doing moderate/vigorous activity (less than 10 minutes) IF 1,2 OR 3 GO TO Q7, IF 5 GO TO Q9				
	Mostly sitting (1)		Mostly standing still (2)		Mostly walking for very short periods (3)
	Mostly doing moderate/vigorous activity (4)			None of the above (5)	
					Yes (1) No (2)
2	Does your work involve <b>vigorous</b> activities (like heavy lifting, digging or heavy construction) for <b>at least 10 minutes</b> at a time? IF NO GO TO Q6				
	In a <b>usual week</b> , how many days do you do <b>vigorous</b> activities as part of your work?				Days (1)
3	On a <b>usual day</b> on which you do <b>vigorous</b> activities, how much time do you spend doing such work?			Hours (1)	Minutes (2)
					Yes (1) No (2)
4	Does your work involve <b>moderate-intensity</b> activities (like fast walking or carrying light loads) for <b>at least 10 minutes</b> at a time? IF NO GO TO Q7				
5	In a <b>usual week</b> , how many days do you do <b>moderate-intensity</b> activities as part of your work?				Days (1)
6	On a <b>usual day</b> on which you do <b>moderate-intensity</b> activities, how much time do you spend doing such work?			Hours (1)	Minutes (2)
7	How long is your usual workday?				
8	Please tick which of the following best describes your work?				
	Working behind a computer (1)		Seated industry work (2) (eg. Sewing, assembling, packing, teller)		
	Teaching (3)	Nursing (4)	Cleaning (5) (domestic work)	Child minding (6)	
			Physical labour (7)	Other (9) (specify)	

**Travel-related physical activity:** Other than activities we've spoken about, I would now like to ask you about the way you travel to and from places (to work, to the shops, to church, etc.)

		Yes (1)	No (2)
9	Do you walk or use a bicycle for <b>at least 10 minutes</b> at a time to get to and from places? IF NO GO TO Q12		
10	In a <b>usual week</b> , how many days do you walk or cycle for at least 10 minutes to get to and from places?		Days
11	On a <b>usual day</b> , how much time do you spend walking or cycling for travel?	Hours (1)	Minutes (2)

<b>Non-work related and leisure time physical activity:</b> The next questions I'm going to ask you are about activities you do in your leisure or spare time, for recreation or fitness, or at home. These do not include activities we've already spoken about					
		Yes (1)	No (2)		
12	In your leisure or spare time do you do any vigorous or moderate-intensity physical activity lasting <b>more than 10 minutes</b> at a time? IF NO GO TO Q19				
13	In your leisure or spare time, do you do any <b>vigorous</b> activities (like running or playing strenuous sport) for <b>at least 10 minutes</b> at a time? IF NO GO TO Q16				
14	In a <b>usual week</b> , how many days do you do <b>vigorous</b> activities as part of your leisure or spare time?	Days			
15	How much time do you spend doing this on a <b>usual day</b> ?	Hours (1)		Minutes (2)	
		Yes (1)	No (2)		
16	In your leisure or spare time, do you do any <b>moderate-intensity</b> activities (like brisk walking, cycling or swimming) for <b>at least 10 minutes</b> at a time? IF NO GO TO Q19				
17	In a <b>usual week</b> , how many days do you do <b>moderate-intensity</b> activities as part of your leisure or spare time?	Days			
18	How much time do you spend doing this on a <b>usual day</b> ?	Hours (1)		Minutes (2)	
<b>Sitting/resting activity:</b> Now I would like to ask you about time you spend sitting or resting, not including sleeping, <b>in the past 7 days.</b> This includes things like sitting at a desk, visiting friends, reading or sitting and watching TV <b>during working hours and leisure or spare time.</b>					
19	Over the <b>past 7 days</b> , how much time did you spend sitting or lying down on a <b>usual day (excluding sleeping)</b> ?	Hours (1)		Minutes (2)	

### PERCEPTIONS OF BODY SHAPE AND SIZE

The last few questions I'm going to ask you are about how you perceive your body. Please answer the questions as honestly as possible

P1	Sometimes we have ideas about how we look and how we might like to look. Among this set of people, which best describes your body shape?	Record the number	
P2	Among this set of people, which best describes the body shape you would most like to have?	Record the number	
P3	Which of these people do you find the most attractive?	Record the number	

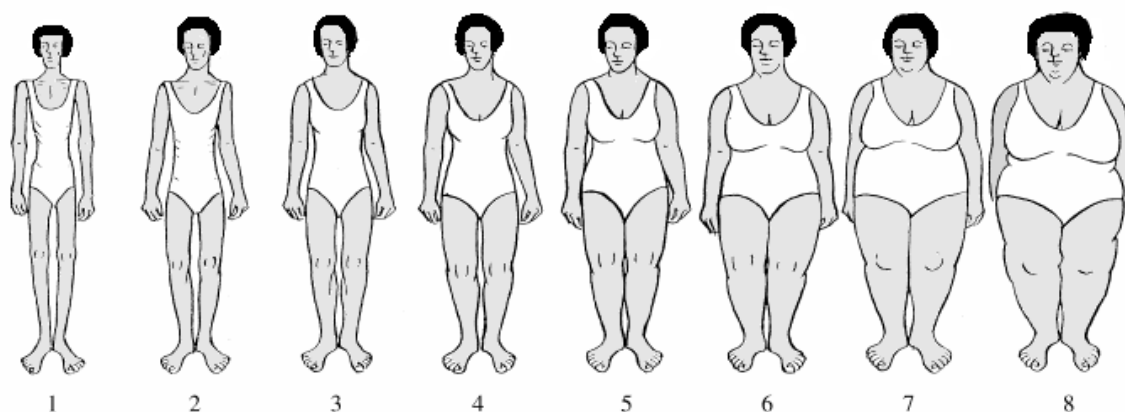


Figure 1 The illustrations of body shapes used for women.

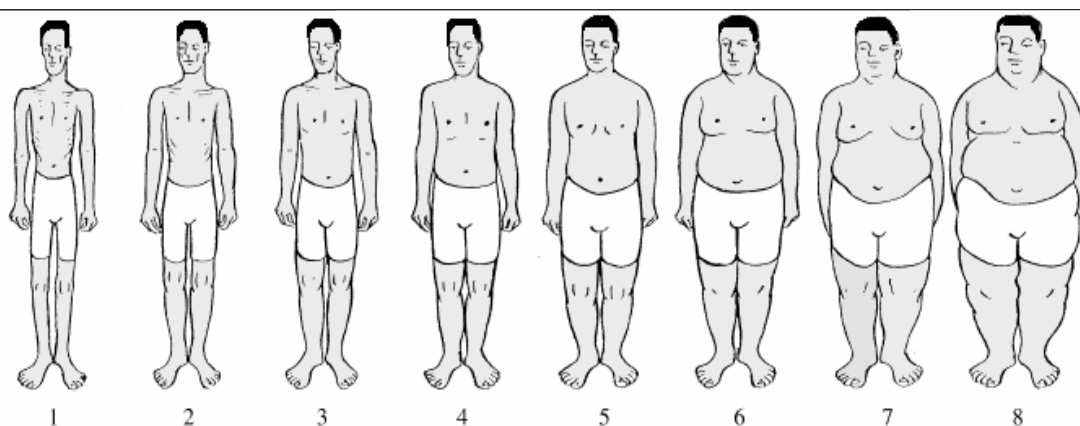


Figure 2 The illustrations of body shapes used for men.

## ANTHROPOMETRIC DATA SHEET

<b>AGE</b>	
<b>SEX</b>	
<b>DATE</b>	
<b>WEIGHT (kg)</b>	
<b>HEIGHT (mm)</b>	
<b>WAIST CIRCUMFERENCE (mm)</b>	
<b>SYSTOLIC BLOOD PRESSURE 1 (mmHg)</b>	
<b>DIASTOLIC BLOOD PRESSURE 1 (mmHg)</b>	
<b>SYSTOLIC BLOOD PRESSURE 2 (mmHg)</b>	
<b>DIASTOLIC BLOOD PRESSURE 2 (mmHg)</b>	
<b>SYSTOLIC BLOOD PRESSURE 3 (mmHg)</b>	
<b>DIASTOLIC BLOOD PRESSURE 3 (mmHg)</b>	
<b>TOTAL BLOOD CHOLESTEROL (mmol.L<sup>-1</sup>)</b>	
<b>BLOOD GLUCOSE (mmol.L<sup>-1</sup>)</b>	



## **BIOELECTRICAL IMPEDANCE DATA**

<b>% BODY FAT</b>	
<b>BODY FAT(kg)</b>	
<b>% LEAN BODY MASS</b>	
<b>LEAN BODY MASS (kg)</b>	
<b>REE</b>	
<b>BMI</b>	

**WHEN DID THE SUBJECT EAT THEIR LAST MEAL?**

<b>APPROXIMATELY 2 HOURS AGO</b>	
<b>MORE THAN 2 HOURS AGO</b>	

## APPENDIX 3: STATISTICAL TABLES

### MORPH AND CV RISKS: COMPARISON TESTS BETWEEN MALES AND FEMALES: INDEPENDENT T-TESTS

Var.	T-tests; Grouping: sex (all cv data age adjusted.sta)											
	Group 1: males					Group 2: females						
	Mean males	Mean females	t-value	df	p	Valid N males	Valid N females	Std.Dev. males	Std.Dev. females	Levene F(1,df)	df Levene	p Levene
age	40.5385	42.554	-1.8084	289	0.071580	143	148	10.7584	8.1125	11.75418	289	0.000695
stature	1.6714	1.567	14.3306	289	0.000000	143	148	0.0604	0.0636	0.63484	289	0.426238
mass	72.0783	92.599	-8.9619	289	0.000000	143	148	16.4091	22.1256	15.69593	289	0.000094
BMI	25.7454	37.592	-14.4713	289	0.000000	143	148	5.4514	8.1926	25.68812	289	0.000001
WC	887.3566	1023.405	-7.4316	289	0.000000	143	148	120.7062	183.9679	10.75215	289	0.001169
SBP	130.6336	132.865	-1.0907	289	0.276312	143	148	15.5026	19.1374	6.44006	289	0.011683
DBP	82.3392	84.878	-1.7374	289	0.083388	143	148	12.3509	12.5725	0.30024	289	0.584154
Gluc.	3.9535	4.322	-1.8130	289	0.070875	143	148	1.1827	2.1316	7.06561	289	0.008295
Chol.	4.2561	4.427	-1.5995	289	0.110794	143	148	0.9333	0.8854	0.29798	289	0.585575
%BF	26.7776	51.039	-27.3065	289	0.000000	143	148	6.7806	8.2737	4.70555	289	0.030880
kg BF	20.2133	48.843	-16.9510	289	0.000000	143	148	9.8775	17.7097	43.14228	289	0.000000
%LBM	73.0636	48.796	28.2418	289	0.000000	143	148	6.6434	7.9336	4.58467	289	0.033096
kg LBM	52.0538	43.932	9.2338	289	0.000000	143	148	8.4808	6.4131	9.80405	289	0.001920

### MORPH AND CV RISKS: NON-PARAMETRIC TESTS: MANN-WHITNEY TESTS

variable	Mann-Whitney U Test (all cv data age adjusted.sta)								
	By variable sex Marked tests are significant at p <.05000								
	Rank Sum males	Rank Sum females	U	Z	p-value	Z adjusted	p-value	Valid N males	Valid N females
age	19559.00	22927.00	9263.00	-1.8373	0.066166	-1.8385	0.065993	143	148
stature	28914.50	13571.50	2545.50	11.1980	0.000000	11.1986	0.000000	143	148
mass	14999.50	27486.50	4703.50	-8.1909	0.000000	-8.1909	0.000000	143	148
BMI	12646.00	29840.00	2350.00	-11.4704	0.000000	-11.4704	0.000000	143	148
WC	15391.50	27094.50	5095.50	-7.6446	0.000000	-7.6451	0.000000	143	148
systolic BP	20682.00	21804.00	10386.00	-0.2724	0.785295	-0.2739	0.784182	143	148
diastolic BP	19860.50	22625.50	9564.50	-1.4172	0.156435	-1.4330	0.151860	143	148
glucose	20462.00	22024.00	10166.00	-0.5790	0.562596	-0.5794	0.562345	143	148
cholesterol	19568.50	22917.50	9272.50	-1.8241	0.068143	-1.8267	0.067750	143	148
%BF	10801.50	31684.50	505.50	-14.0407	0.000000	-14.0409	0.000000	143	148
kg BF	11741.50	30744.50	1445.50	-12.7308	0.000000	-12.7310	0.000000	143	148
%LBM	31059.50	11426.50	400.50	14.1870	0.000000	14.1872	0.000000	143	148
kg LBM	26719.50	15766.50	4740.50	8.1393	0.000000	8.1395	0.000000	143	148

PA: COMPARISON BETWEEN MALES AND FEMALES USING T-TESTS FOR INDEPENDENT SAMPLES  
(PARAMETRIC TEST)

Variable	T-tests; Grouping: Gender (physical activity GPAQ Leisure final.sta)							Std.Dev. M	Std.Dev. F	Levene F(1,df)	df Levene	p Levene
	Mean M	Mean F	t-value	df	P	Valid N M	Valid N F					
days vigorous activity work	1.975	1.050	3.49908	256	0.000550	119	139	2.323	1.920	30.82393	256	0.000000
mins vig activity work	91.134	32.662	3.95067	256	0.000101	119	139	146.739	87.417	46.10189	256	0.000000
METs vig activity work	3205.378	1067.626	3.90440	256	0.000121	119	139	5439.561	3217.675	43.96830	256	0.000000
days mod activity work	4.193	4.080	0.49129	255	0.623642	119	138	1.772	1.911	1.84811	255	0.175205
mins mod activity work	233.109	237.194	-0.20973	256	0.834042	119	139	152.448	158.890	0.06565	256	0.797983
METs mod activity work	4571.765	4468.201	0.25390	256	0.799774	119	139	3107.992	3395.167	1.68885	256	0.194921
days walking transport	3.941	4.036	-0.30424	256	0.761195	119	139	2.498	2.492	0.05219	256	0.819473
minutes walking	58.824	52.302	0.95646	256	0.339741	119	139	52.164	56.589	0.07186	256	0.788858
METs walking	1200.504	1053.669	1.02660	256	0.305578	119	139	1125.099	1162.202	0.39815	256	0.528607
days vig leisure	0.630	2.722	-5.24841	135	0.000001	119	18	1.478	2.137	4.81799	135	0.029874
mins vig leisure	16.387	16.317	0.01149	256	0.990843	119	139	41.287	54.394	0.19069	256	0.662712
MET mins vig leisure	418.151	359.252	0.35961	256	0.719433	119	139	1083.894	1478.484	0.10350	256	0.747932
days mod leisure	1.000	2.194	-5.78969	215	0.000000	119	98	1.396	1.641	2.83518	215	0.093672
mins mod leisure	86.681	149.137	-3.57997	256	0.000411	119	139	118.751	155.372	20.00945	256	0.000012
MET mins mod leisure	550.084	1029.928	-3.85012	256	0.000149	119	139	823.691	1125.716	13.81483	256	0.000248
TOTAL PAMETs	9945.882	7978.676	3.12744	256	0.001967	119	139	5323.004	4777.980	0.57052	256	0.450749
total days vig	2.605	1.403	3.80785	256	0.000176	119	139	2.805	2.264	20.23883	256	0.000010
total mins vig	47.079	43.234	0.39678	115	0.692268	65	52	53.102	50.780	0.00006	115	0.993646
total METs vig	3623.529	1426.878	3.77003	256	0.000203	119	139	5681.810	3573.918	34.89508	256	0.000000
total days mod	9.134	9.633	-1.09943	256	0.272613	119	139	3.539	3.709	0.04921	256	0.824620
total mins mod	45.936	51.366	-1.24599	254	0.213916	118	138	28.575	39.277	3.50914	254	0.062179
total METs mod	6322.353	5498.129	1.80646	256	0.072021	119	139	3392.564	3862.352	4.33634	256	0.038300

PA: COMPARISON BETWEEN MALES AND FEMALES USING NON-PARAMETRIC ROCEDURES: MANN-WHITNEY TEST

variable	Mann-Whitney U Test (physical activity GPAQ Leisure final.sta)					Z adjusted	p-value	Valid N M	Valid N F
	Rank Sum M	Rank Sum F	U	Z	p-value				
days vigorous activity work	17019.50	16391.50	6661.500	2.69204	0.007102	3.11366	0.001848	119	139
mins vig activity work	17145.00	16266.00	6536.000	2.90208	0.003707	3.33646	0.000849	119	139
METs vig activity work	17232.00	16179.00	6449.000	3.04768	0.002306	3.50314	0.000460	119	139
days mod activity work	15596.00	17557.00	7966.000	0.41148	0.680722	0.52055	0.602679	119	138
mins mod activity work	15269.00	18142.00	8129.000	-0.23598	0.813447	-0.23773	0.812089	119	139
METs mod activity work	15551.50	17859.50	8129.500	0.23515	0.814096	0.23649	0.813051	119	139
days walking transport	15277.50	18133.50	8137.500	-0.22176	0.824504	-0.23253	0.816126	119	139
minutes walking	16194.50	17216.50	7486.500	1.31129	0.189761	1.32713	0.184467	119	139
METs walking	16148.00	17263.00	7533.000	1.23347	0.217403	1.24176	0.214326	119	139
days vig leisure	7408.50	2044.50	268.500	-5.10994	0.000000	-6.31567	0.000000	119	18
mins vig leisure	15889.50	17521.50	7791.500	0.80083	0.423229	1.25851	0.208207	119	139
MET mins vig leisure	15986.50	17424.50	7694.500	0.96317	0.335460	1.52921	0.126214	119	139
days mod leisure	9772.00	13881.00	2632.000	-6.94900	0.000000	-7.24725	0.000000	119	98
mins mod leisure	13509.00	19902.00	6369.000	-3.18157	0.001465	-3.27010	0.001075	119	139
MET mins mod leisure	13228.50	20182.50	6088.500	-3.65103	0.000261	-3.74938	0.000177	119	139
TOTAL PA METs	16913.00	16498.00	6768.000	2.51379	0.011944	2.51391	0.011941	119	139
total days vig	17271.50	16139.50	6409.500	3.11379	0.001847	3.40797	0.000655	119	139
total mins vig	3977.50	2925.50	1547.500	0.77890	0.436041	0.77966	0.435590	65	52
total METs vig	17385.50	16025.50	6295.500	3.30459	0.000951	3.61274	0.000303	119	139
total days mod	14674.50	18736.50	7534.500	-1.23096	0.218340	-1.23883	0.215408	119	139
total mins mod	14704.00	18192.00	7683.000	-0.77640	0.437516	-0.77651	0.437451	118	138
total METs mod	16471.00	16940.00	7210.000	1.77405	0.076056	1.77438	0.076001	119	139

# COMPARISON BETWEEN MALES AND FEMALES USING CROSSTABULATIONS: CHI-SQUARE TESTS (ALC. CONSUMP & SMOKING)

Statistic	Statistics: sex(2) x had a drink in the last 12 months(2)		
	Chi-square	df	p
Pearson Chi-square	23.37070	df=1	p=.00000
M-L Chi-square	23.80836	df=1	p=.00000
Yates Chi-square	22.22285	df=1	p=.00000
Fisher exact, one-tailed			p=.00000
two-tailed			p=.00000
McNemar Chi-square (A/D)	3.396739	df=1	p=.06533
(B/C)	9.752381	df=1	p=.00179

Statistic	Statistics: sex(2) x currently smoke(2)		
	Chi-square	df	p
Pearson Chi-square	82.92248	df=1	p=0.0000
M-L Chi-square	94.83945	df=1	p=0.0000
Yates Chi-square	80.51352	df=1	p=0.0000
Fisher exact, one-tailed			p=0.0000
two-tailed			p=.00000
McNemar Chi-square (A/D)	21.70892	df=1	p=.00000
(B/C)	53.63514	df=1	p=.00000

Statistic	Statistics: sex(2) x exposed to smoking at work(2)		
	Chi-square	df	p
Pearson Chi-square	24.90855	df=1	p=.00000
M-L Chi-square	25.45475	df=1	p=.00000
Yates Chi-square	23.66583	df=1	p=.00000
Fisher exact, one-tailed			p=.00000
two-tailed			p=.00000
McNemar Chi-square (A/D)	16.26344	df=1	p=.00006
(B/C)	23.80952	df=1	p=.00000

Statistic	Statistics: sex(2) x exposed to smoking at home(2)		
	Chi-square	df	p
Pearson Chi-square	1.041111	df=1	p=.30757
M-L Chi-square	1.043212	df=1	p=.30708
Yates Chi-square	.7875468	df=1	p=.37485
Fisher exact, one-tailed			p=.18748
two-tailed			p=.35269
McNemar Chi-square (A/D)	35.25180	df=1	p=.00000
(B/C)	27.79605	df=1	p=.00000

Statistic	Statistics: sex(2) x exposed to fumes at work(2)		
	Chi-square	df	p
Pearson Chi-square	30.59989	df=1	p=.00000
M-L Chi-square	31.35573	df=1	p=.00000
Yates Chi-square	29.24611	df=1	p=.00000
Fisher exact, one-tailed			p=.00000
two-tailed			p=.00000
McNemar Chi-square (A/D)	12.06283	df=1	p=.00051
(B/C)	18.49000	df=1	p=.00002

Statistic	Statistics: sex(2) x how frequently drink(4)		
	Chi-square	df	p
Pearson Chi-square	54.90186	df=3	p=.00000
M-L Chi-square	60.52382	df=3	p=.00000

Statistic	Statistics: sex(2) x smoked in past(3)		
	Chi-square	df	p
Pearson Chi-square	27.59387	df=2	p=.00000
M-L Chi-square	27.58319	df=2	p=.00000

# PERCEIVED VS ACTUAL: **MALES ONLY:**

## Systolic BP

Statistic	Statistics: perceived BP(2) x actual systolic BP(2): v2="M"		
	Chi-square	df	p
Pearson Chi-square	5.702340	df=1	p=.01694
M-L Chi-square	5.642593	df=1	p=.01753
Yates Chi-square	4.606967	df=1	p=.03184
Fisher exact, one-tailed			p=.01631
two-tailed			p=.02857
McNemar Chi-square (A/D)	11.85965	df=1	p=.00057
(B/C)	2.700000	df=1	p=.10035
Kendall's tau b & c	b=.2560159	c=.2272427	

## Diastolic BP

Statistic	Statistics: perceived BP(2) x actual diastolic BP(2): v2="M"		
	Chi-square	df	p
Pearson Chi-square	7.400228	df=1	p=.00652
M-L Chi-square	7.366400	df=1	p=.00665
Yates Chi-square	6.149499	df=1	p=.01315
Fisher exact, one-tailed			p=.00670
two-tailed			p=.00855
McNemar Chi-square (A/D)	10.77586	df=1	p=.00103
(B/C)	3.448276	df=1	p=.06332
Kendall's tau b & c	b=.2916506	c=.2600079	

## High cholesterol

Statistic	Statistics: perceived high cholesterol(2) x actual high cholesterol(2): v2="M"		
	Chi-square	df	p
Pearson Chi-square	.7899356	df=1	p=.37412
M-L Chi-square	.6161049	df=1	p=.43250
Yates Chi-square	.0071197	df=1	p=.93276
Fisher exact, one-tailed			p=.38122
two-tailed			p=.38122
McNemar Chi-square (A/D)	119.0720	df=1	p=0.0000
(B/C)	6.722222	df=1	p=.00952
Kendall's tau b & c	b=.0743238	c=.0154531	

## BMI

Statistic	Statistics: perceived body weight(2) x actual BMI(2): v2="M"		
	Chi-square	df	p
Pearson Chi-square	22.37819	df=1	p=.00000
M-L Chi-square	24.44663	df=1	p=.00000
Yates Chi-square	20.35188	df=1	p=.00001
Fisher exact, one-tailed			p=.00000
two-tailed			p=.00000
McNemar Chi-square (A/D)	20.54445	df=1	p=.00001
(B/C)	29.16667	df=1	p=.00000
Kendall's tau b & c	b=.4117423	c=.3269054	

## PERCEIVED VS ACTUAL: FEMALES ONLY

### Systolic BP

Statistic	Statistics: perceived BP(2) x actual systolic BP(2): v2="F"		
	Chi-square	df	p
Pearson Chi-square	12.15087	df=1	p=.00049
M-L Chi-square	12.40469	df=1	p=.00043
Yates Chi-square	10.80049	df=1	p=.00102
Fisher exact, one-tailed			p=.00046
two-tailed			p=.00066
McNemar Chi-square (A/D)	3.125000	df=1	p=.07710
(B/C)	3.361111	df=1	p=.06676
Kendall's tau b & c	b=.3354222	c=.3237311	

### Diastolic BP

Statistic	Statistics: perceived BP(2) x actual diastolic BP(2): v2="F"		
	Chi-square	df	p
Pearson Chi-square	6.123244	df=1	p=.01334
M-L Chi-square	6.176947	df=1	p=.01294
Yates Chi-square	5.199759	df=1	p=.02259
Fisher exact, one-tailed			p=.01110
two-tailed			p=.01948
McNemar Chi-square (A/D)	.9552239	df=1	p=.32840
(B/C)	.3902439	df=1	p=.53217
Kendall's tau b & c	b=.2381107	c=.2359396	

### High cholesterol

Statistic	Statistics: perceived high cholesterol(2) x actual high cholesterol(2): v2="F"		
	Chi-square	df	p
Pearson Chi-square	1.554229	df=1	p=.21252
M-L Chi-square	2.779547	df=1	p=.09548
Yates Chi-square	.5531822	df=1	p=.45702
Fisher exact, one-tailed			p=.25050
two-tailed			p=.35828
McNemar Chi-square (A/D)	110.0089	df=1	p=0.0000
(B/C)	5.633333	df=1	p=.01762
Kendall's tau b & c	b=-.104620	c=-.034914	

### Type II diabetes

Statistic	Statistics: perceived type II diabetes(2) x actual type II diabetes(2): v2="F"		
	Chi-square	df	p
Pearson Chi-square	24.32277	df=1	p=.00000
M-L Chi-square	13.67953	df=1	p=.00022
Yates Chi-square	16.05034	df=1	p=.00006
Fisher exact, one-tailed			p=.00117
two-tailed			p=.00117
McNemar Chi-square (A/D)	116.3769	df=1	p=0.0000
(B/C)	11.07692	df=1	p=.00087
Kendall's tau b & c	b=.4124188	c=.0745269	

### BMI

Statistic	Statistics: perceived body weight(2) x actual BMI(2): v2="F"		
	Chi-square	df	p
Pearson Chi-square	5.005249	df=1	p=.02527
M-L Chi-square	5.741395	df=1	p=.01657
Yates Chi-square	3.579268	df=1	p=.05851
Fisher exact, one-tailed			p=.02533
two-tailed			p=.03578
McNemar Chi-square (A/D)	42.01389	df=1	p=.00000
(B/C)	60.13636	df=1	p=.00000
Kendall's tau b & c	b=.1904466	c=.0938878	

# INDEPENDENT T-TESTS: DIETARY VARIABLES

Variable	T-tests; Grouping: gender (dietary intake males and females 2 ex						Valid N Female	Valid N Male	Std.Dev. Female	Std.Dev. Male	Levene F(1,df)	df Levene	p Levene
	Mean Female	Mean Male	t-value	df	p								
energy (kJ)	7193.034	9003.993	-4.38131	289	0.000017		148	143	3277.259	3764.29	2.59705	289	0.108154
total protein (g)	59.713	84.096	-4.91289	289	0.000002		148	143	28.694	52.85	22.29491	289	0.000004
plant protein (g)	18.753	30.052	-6.68998	289	0.000000		148	143	11.191	17.11	10.59930	289	0.001266
animal protein (g)	32.186	52.308	-4.42769	289	0.000014		148	143	23.529	49.84	29.25347	289	0.000000
total fat (g)	62.147	71.009	-1.72800	289	0.085056		148	143	41.629	45.81	1.43825	289	0.231406
carbohydrate (g)	211.096	264.302	-4.40019	289	0.000015		148	143	99.213	107.01	1.76280	289	0.185324
starch (g)	27.510	57.697	-4.85045	289	0.000002		148	143	41.582	62.79	28.91970	289	0.000000
total sugars (g)	62.436	51.369	2.50609	289	0.012757		148	143	37.094	38.24	0.25240	289	0.615772
added sugar (g)	67.967	56.427	2.15838	289	0.031721		148	143	49.441	41.24	1.08143	289	0.299247
total dietary fibre (g)	16.832	20.928	-3.01809	289	0.002770		148	143	8.916	13.80	11.30496	289	0.000877
na (mg)	1525.243	1979.350	-2.92879	289	0.003674		148	143	1108.014	1512.43	6.98472	289	0.008669
Cholesterol (mg)	197.162	279.577	-3.22427	288	0.001408		148	143	160.418	264.35	20.09818	289	0.000011
% Energy -Protein	0.146	0.159	-1.86588	289	0.063070		148	143	0.049	0.07	9.55796	289	0.002185
% Energy - Fat	0.306	0.283	1.88189	289	0.060856		148	143	0.098	0.11	1.46645	289	0.226896
% Energy - CHO	0.547	0.551	-0.29463	289	0.768491		148	143	0.117	0.13	1.38995	289	0.239383
% Energy - Alcohol	0.000	0.006	-1.84026	289	0.066755		148	143	0.004	0.04	13.99477	289	0.000221

# NON-PARAMETRIC PROCEDURE

variable	Mann-Whitney U Test (dietary intake males and females						p-value	Valid N Female	Valid N Male
	Rank Sum Female	Rank Sum Male	U	Z	p-value	Z adjusted			
energy (kJ)	18075.50	24410.50	7049.50	-4.92177	0.000001	-4.92177	0.000001	148	143
total protein (g)	18575.00	23911.00	7549.00	-4.22572	0.000024	-4.22575	0.000024	148	143
plant protein (g)	17048.00	25438.00	6022.00	-6.35357	0.000000	-6.35364	0.000000	148	143
animal protein (g)	19264.50	23221.50	8238.50	-3.26492	0.001095	-3.26502	0.001095	148	143
total fat (g)	20223.50	22262.50	9197.50	-1.92857	0.053785	-1.92859	0.053783	148	143
carbohydrate (g)	18013.50	24472.50	6987.50	-5.00816	0.000001	-5.00817	0.000001	148	143
starch (g)	18921.00	23565.00	7895.00	-3.74358	0.000181	-3.74529	0.000180	148	143
total sugars (g)	23574.50	18911.50	8615.50	2.73958	0.006152	2.73959	0.006152	148	143
added sugar (g)	23325.50	19160.50	8864.50	2.39260	0.016730	2.39287	0.016718	148	143
total dietary fibre (g)	19946.50	22539.50	8920.50	-2.31457	0.020637	-2.31464	0.020634	148	143
na (mg)	19699.50	22786.50	8673.50	-2.65876	0.007843	-2.65876	0.007843	148	143
Cholesterol (mg)	19803.50	22391.50	8777.50	-2.42334	0.015379	-2.42339	0.015377	148	142
% Energy -Protein	20813.50	21672.50	9787.50	-1.10642	0.268545	-1.10643	0.268543	148	143
% Energy - Fat	22907.00	19579.00	9283.00	1.80943	0.070385	1.80944	0.070384	148	143
% Energy - CHO	21417.50	21068.50	10391.50	-0.26476	0.791194	-0.26476	0.791193	148	143
% Energy - Alcohol	21376.00	21110.00	10350.00	-0.32259	0.747006	-1.21543	0.224202	148	143

# ALL CV DATA CORRELATIONS:

MALES (N=143)

Var.	Correlations (all cv data) N=143: v1="males"											
	stature	mass	BMI	WC	SBP	DBP	Gluc.	Chol.	%BF	kg BF	%LBM	kg LBM
stature	1.0000	.3710	.0586	.1999	.0976	.1461	.0726	.0445	-.0452	.1344	.0651	.5076
	p= ---	p=.000	p=.487	p=.017	p=.246	p=.082	p=.389	p=.598	p=.592	p=.110	p=.440	p=.000
mass	.3710	1.0000	.9467	.8998	.3507	.3359	.2284	.1411	.6742	.9036	-.6794	.8440
	p=.000	p= ---	p=0.00	p=0.00	p=.000	p=.000	p=.006	p=.093	p=0.00	p=0.00	p=0.00	p=0.00
BMI	.0586	.9467	1.0000	.8968	.3372	.3066	.2140	.1322	.7345	.9227	-.7462	.7323
	p=.487	p=0.00	p= ---	p=0.00	p=.000	p=.000	p=.010	p=.116	p=0.00	p=0.00	p=0.00	p=0.00
WC	.1999	.8998	.8968	1.0000	.3766	.3700	.3132	.1814	.7124	.8639	-.7191	.7144
	p=.017	p=0.00	p=0.00	p= ---	p=.000	p=.000	p=.000	p=.030	p=0.00	p=0.00	p=0.00	p=0.00
SBP	.0976	.3507	.3372	.3766	1.0000	.7874	.1081	.1756	.2860	.3422	-.2895	.2768
	p=.246	p=.000	p=.000	p=.000	p= ---	p=0.00	p=.199	p=.036	p=.001	p=.000	p=.000	p=.001
DBP	.1461	.3359	.3066	.3700	.7874	1.0000	.0939	.2444	.3199	.3626	-.3337	.2345
	p=.082	p=.000	p=.000	p=.000	p=0.00	p= ---	p=.265	p=.003	p=.000	p=.000	p=.000	p=.005
Gluc.	.0726	.2284	.2140	.3132	.1081	.0939	1.0000	.0951	.3148	.2578	-.3079	.1328
	p=.389	p=.006	p=.010	p=.000	p=.199	p=.265	p= ---	p=.259	p=.000	p=.002	p=.000	p=.114
Chol.	.0445	.1411	.1322	.1814	.1756	.2444	.0951	1.0000	.2696	.2188	-.2833	.0428
	p=.598	p=.093	p=.116	p=.030	p=.036	p=.003	p=.259	p= ---	p=.001	p=.009	p=.001	p=.612
%BF	-.0452	.6742	.7345	.7124	.2860	.3199	.3148	.2696	1.0000	.8563	-.9756	.2407
	p=.592	p=0.00	p=0.00	p=0.00	p=.001	p=.000	p=.000	p=.001	p= ---	p=0.00	p=0.00	p=.004
kg BF	.1344	.9036	.9227	.8639	.3422	.3626	.2578	.2188	.8563	1.0000	-.8939	.6091
	p=.110	p=0.00	p=0.00	p=0.00	p=.000	p=.000	p=.002	p=.009	p=0.00	p= ---	p=0.00	p=.000
%LBM	.0651	-.6794	-.7462	-.7191	-.2895	-.3337	-.3079	-.2833	-.9756	-.8939	1.0000	-.2692
	p=.440	p=0.00	p=0.00	p=0.00	p=.000	p=.000	p=.000	p=.001	p=0.00	p=0.00	p= ---	p=.001
kg LBM	.5076	.8440	.7323	.7144	.2768	.2345	.1328	.0428	.2407	.6091	-.2692	1.0000
	p=.000	p=0.00	p=0.00	p=0.00	p=.001	p=.005	p=.114	p=.612	p=.004	p=.000	p=.001	p= ---

FEMALES (N=148)

Variable	Correlations (all cv data) N=148:											
	stature	mass	BMI	WC	SBP	DBP	Gluc.	Chol.	%BF	kg BF	%LBM	kg LBM
stature	1.0000	.4200	.0805	.2776	.0673	.0525	.0882	.0391	.0472	.2900	-.0234	.6186
	p= ---	p=.000	p=.331	p=.001	p=.416	p=.526	p=.286	p=.637	p=.569	p=.000	p=.778	p=.000
mass	.4200	1.0000	.9355	.7522	.2597	.2530	.1522	.2009	.8154	.9733	-.8160	.7377
	p=.000	p= ---	p=0.00	p=0.00	p=.001	p=.002	p=.065	p=.014	p=0.00	p=0.00	p=0.00	p=0.00
BMI	.0805	.9355	1.0000	.7227	.2575	.2556	.1354	.2002	.8773	.9529	-.8852	.5775
	p=.331	p=0.00	p= ---	p=0.00	p=.002	p=.002	p=.101	p=.015	p=0.00	p=0.00	p=0.00	p=.000
WC	.2776	.7522	.7227	1.0000	.2101	.2231	.1880	.1625	.6743	.7541	-.6725	.4761
	p=.001	p=0.00	p=0.00	p= ---	p=.010	p=.006	p=.022	p=.048	p=0.00	p=0.00	p=0.00	p=.000
SBP	.0673	.2597	.2575	.2101	1.0000	.7872	.2805	.2388	.3203	.2801	-.3065	.0797
	p=.416	p=.001	p=.002	p=.010	p= ---	p=0.00	p=.001	p=.003	p=.000	p=.001	p=.000	p=.336
DBP	.0525	.2530	.2556	.2231	.7872	1.0000	.1560	.1941	.3063	.2753	-.3011	.0901
	p=.526	p=.002	p=.002	p=.006	p=0.00	p= ---	p=.058	p=.018	p=.000	p=.001	p=.000	p=.276
Gluc.	.0882	.1522	.1354	.1880	.2805	.1560	1.0000	.2773	.1580	.1581	-.1587	.0786
	p=.286	p=.065	p=.101	p=.022	p=.001	p=.058	p= ---	p=.001	p=.055	p=.055	p=.054	p=.342
Chol.	.0391	.2009	.2002	.1625	.2388	.1941	.2773	1.0000	.2703	.2408	-.2601	.0054
	p=.637	p=.014	p=.015	p=.048	p=.003	p=.018	p=.001	p= ---	p=.001	p=.003	p=.001	p=.948
%BF	.0472	.8154	.8773	.6743	.3203	.3063	.1580	.2703	1.0000	.8747	-.9738	.3119
	p=.569	p=0.00	p=0.00	p=0.00	p=.000	p=.000	p=.055	p=.001	p= ---	p=0.00	p=0.00	p=.000
kg BF	.2900	.9733	.9529	.7541	.2801	.2753	.1581	.2408	.8747	1.0000	-.8939	.5847
	p=.000	p=0.00	p=0.00	p=0.00	p=.001	p=.001	p=.055	p=.003	p=0.00	p= ---	p=0.00	p=.000
%LBM	-.0234	-.8160	-.8852	-.6725	-.3065	-.3011	-.1587	-.2601	-.9738	-.8939	1.0000	-.3164
	p=.778	p=0.00	p=0.00	p=0.00	p=.000	p=.000	p=.054	p=.001	p=0.00	p=0.00	p= ---	p=.000
kg LBM	.6186	.7377	.5775	.4761	.0797	.0901	.0786	.0054	.3119	.5847	-.3164	1.0000
	p=.000	p=0.00	p=.000	p=.000	p=.336	p=.276	p=.342	p=.948	p=.000	p=.000	p=.000	p= ---



# PHYSICAL ACTIVITY CORRELATIONS:

MALES (N=119)

Var.	Correlations (physical activity correlations data.sta) N=119: v2=1				
	total MET-mins of activity/week	total mins of activity/week	total mins of vigorous activity/week	total mins of moderate activity/week	total mins transport activity/week
Mass	-.2031 p=.027	-.1192 p=.197	-.2360 p=.010	.1365 p=.139	-.1681 p=.068
BMI	-.2177 p=.017	-.1314 p=.154	-.2485 p=.006	.1429 p=.121	-.1927 p=.036
WC	-.2307 p=.012	-.1771 p=.054	-.2176 p=.017	.0843 p=.362	-.2390 p=.009
SBP	-.0342 p=.712	.0244 p=.793	-.0936 p=.311	.1112 p=.228	-.0145 p=.876
DBP	-.0092 p=.921	.1150 p=.213	-.1567 p=.089	.2513 p=.006	.0123 p=.894
Chol.	-.0900 p=.331	.0518 p=.576	-.2314 p=.011	.2519 p=.006	.0059 p=.949
Gluc.	-.1514 p=.100	-.1544 p=.094	-.0964 p=.297	-.0591 p=.524	-.0563 p=.543
%BF	-.2461 p=.007	-.0905 p=.328	-.3516 p=.000	.2305 p=.012	-.0633 p=.494
Total BF (kg)	-.2163 p=.018	-.0820 p=.375	-.3059 p=.001	.2280 p=.013	-.1456 p=.114
% LBM	.2483 p=.006	.0830 p=.369	.3646 p=.000	-.2529 p=.006	.0731 p=.429
Total LBM (kg)	-.1558 p=.091	-.1361 p=.140	-.1269 p=.169	.0281 p=.762	-.1783 p=.052

FEMALES (N=138)

Variable	Correlations (physical activity correlations data.sta) N=138: v2=2				
	total MET-mins of activity/week	total mins of activity/week	total mins of vigorous activity/week	total mins of moderate activity/week	total mins transport activity/week
Mass	-.0494 p=.565	-.0174 p=.840	-.0940 p=.273	.0496 p=.563	-.0810 p=.345
BMI	-.0350 p=.684	-.0055 p=.949	-.0929 p=.279	.0593 p=.490	-.0909 p=.289
WC	-.0071 p=.934	.0421 p=.624	-.1516 p=.076	.0814 p=.342	.0462 p=.591
SBP	.0465 p=.588	.0645 p=.452	-.0162 p=.851	.0563 p=.512	.0632 p=.462
ABP	-.0563 p=.512	-.0407 p=.635	-.0748 p=.383	-.0326 p=.704	.0604 p=.481
Chol.	-.0282 p=.743	-.0207 p=.809	-.0446 p=.603	.0047 p=.956	-.0419 p=.626
Gluc.	-.1501 p=.079	-.1342 p=.117	-.1009 p=.239	-.1114 p=.193	.0667 p=.437
%BF	-.0145 p=.866	-.0102 p=.906	-.0220 p=.798	-.0007 p=.993	-.0080 p=.926
Total BF (kg)	-.0379 p=.659	-.0157 p=.855	-.0786 p=.360	.0421 p=.624	-.0920 p=.283
% LBM	.0291 p=.735	.0192 p=.823	.0427 p=.619	-.0081 p=.925	.0390 p=.650
Total LBM (kg)	-.0523 p=.543	-.0034 p=.968	-.1373 p=.108	.0819 p=.340	-.0808 p=.346

## DIETARY DATA CORRELATIONS

MALES (N=143)

Variable	Correlations (dietary data correlations.sta) N=143: v2=1							
	total energy intake (kJ)	total sugars (g)	total added sugar (g)	total protein (g)	total fat (g)	carbohydrate (g)	saturated fatty acids (g)	cholesterol (mg)
Mass	-.1921	-.0205	.0824	-.1383	-.1003	-.2243	-.0314	-.0702
	p=.022	p=.808	p=.328	p=.099	p=.233	p=.007	p=.709	p=.405
BMI	-.1774	-.0103	.1093	-.1132	-.1240	-.1822	-.0945	-.0910
	p=.034	p=.903	p=.194	p=.178	p=.140	p=.029	p=.261	p=.280
WC	-.2277	-.0782	.0335	-.1287	-.1412	-.2454	-.1035	-.1327
	p=.006	p=.353	p=.691	p=.126	p=.092	p=.003	p=.219	p=.114
SBP	-.1652	.0168	-.0027	-.2217	-.1922	-.0628	-.1606	-.0952
	p=.049	p=.842	p=.974	p=.008	p=.021	p=.456	p=.055	p=.258
DBP	-.1591	.0497	-.1337	-.1654	-.1719	-.1006	-.1179	-.1412
	p=.058	p=.556	p=.112	p=.048	p=.040	p=.232	p=.161	p=.093
Chol.	-.0461	-.0116	-.0475	-.0089	-.0412	-.1098	-.0404	-.0398
	p=.584	p=.890	p=.573	p=.916	p=.625	p=.192	p=.632	p=.637
Gluc.	.0160	-.0339	.0261	.0210	.0863	-.0512	.0625	-.0413
	p=.849	p=.687	p=.757	p=.804	p=.305	p=.544	p=.458	p=.624
% BF	-.2501	.0223	.0652	-.1004	-.2485	-.2388	-.2034	-.1616
	p=.003	p=.791	p=.439	p=.233	p=.003	p=.004	p=.015	p=.054
Total BF (kg)	-.2443	-.0078	.0699	-.1593	-.1987	-.2378	-.1582	-.1456
	p=.003	p=.926	p=.407	p=.057	p=.017	p=.004	p=.059	p=.083
% LBM	.2875	-.0094	-.0680	.1581	.2724	.2657	.2378	.2031
	p=.000	p=.911	p=.420	p=.059	p=.001	p=.001	p=.004	p=.015
Total LBM (kg)	-.0642	-.0198	.0715	-.0880	.0687	-.1402	.1234	.0481
	p=.446	p=.815	p=.396	p=.296	p=.415	p=.095	p=.142	p=.568

FEMALES (N=148)

Variable	Correlations (dietary data correlations.sta) N=148: v2=2							
	total energy intake (kJ)	total sugars (g)	total added sugar (g)	total protein (g)	total fat (g)	carbohydrate (g)	saturated fatty acids (g)	cholesterol (mg)
Mass	-.1458	-.0933	-.0755	.0333	-.1159	-.1826	-.0296	-.0596
	p=.077	p=.260	p=.362	p=.688	p=.161	p=.026	p=.721	p=.472
BMI	-.1581	-.0952	-.0646	-.0023	-.1278	-.1830	-.0718	-.0577
	p=.055	p=.250	p=.435	p=.978	p=.122	p=.026	p=.386	p=.486
WC	-.1603	-.0894	-.0681	.0376	-.1427	-.1872	-.0707	-.0239
	p=.052	p=.280	p=.411	p=.650	p=.084	p=.023	p=.393	p=.773
SBP	-.0037	.0394	.1682	-.0440	-.0854	.0761	-.0788	-.0723
	p=.964	p=.634	p=.041	p=.595	p=.302	p=.358	p=.341	p=.383
DBP	-.0555	.0242	.1479	-.0996	-.1009	.0059	-.0661	-.0562
	p=.503	p=.771	p=.073	p=.229	p=.222	p=.943	p=.425	p=.497
Chol.	.0105	.0290	.0846	.0355	-.0467	.0485	-.0203	.0896
	p=.900	p=.727	p=.307	p=.668	p=.573	p=.558	p=.806	p=.279
Gluc.	.0483	-.1534	.0157	.1179	.0752	-.0077	.0614	.0310
	p=.560	p=.063	p=.850	p=.153	p=.364	p=.926	p=.458	p=.708
% BF	-.1105	-.0220	.0761	-.0557	-.1142	-.0922	-.0644	-.1304
	p=.181	p=.790	p=.358	p=.501	p=.167	p=.265	p=.437	p=.114
Total BF (kg)	-.1229	-.0582	-.0225	.0178	-.1033	-.1455	-.0375	-.0493
	p=.137	p=.483	p=.786	p=.830	p=.211	p=.078	p=.651	p=.552
% LBM	.1114	.0063	-.0690	.0645	.1175	.0870	.0766	.1211
	p=.178	p=.939	p=.405	p=.436	p=.155	p=.293	p=.355	p=.143
Total LBM (kg)	-.1560	-.1586	-.2082	.0646	-.0992	-.2241	-.0005	-.0509
	p=.058	p=.054	p=.011	p=.436	p=.230	p=.006	p=.995	p=.539