

**CARDIO-METABOLIC DISEASE AND  
ASSOCIATED RISK FACTORS IN THE  
JOHANNESBURG HEALTH DISTRICT**

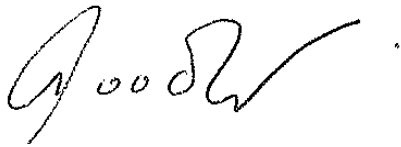
**Nishila Moodley**

**A research report submitted to the  
Faculty of Health Sciences, University of Witwatersrand, South Africa  
in partial fulfillment of the requirements for the Masters in Medicine  
in the branch of Community Health  
Johannesburg, 2011**

Supervisor:  
Dr Debashis Basu

## **DECLARATION**

I, Nishila Moodley, declare that this research report is my own work. It is being submitted for the Masters in Medicine, Community Health. It has not been submitted before for any degree or examination.

A handwritten signature in black ink, appearing to read 'Nishila Moodley', with a small dot at the end of the signature.

18th day of July, 2011

## DEDICATION

To

My husband, Jonathan Sheldon & my sister, Camintha Moodley

*If I have seen further it is only by standing on the shoulders of giants.*

Sir Isaac Newton, 5th day of February, 1675

## **PRESENTATIONS ARISING FROM THIS STUDY**

1. Moodley N, Basu D, Wing J, Kaye-Petersen E. The Metabolic Syndrome and Associated Risk Factors in the Johannesburg Health District. IDF 20th World Diabetes Congress, Montreal, Canada. 18 – 22 October 2009. (Poster Presentation)
  
2. Moodley N, Basu D, Wing J, Kaye-Petersen E. The Metabolic Syndrome and Associated Risk Factors in the Johannesburg Health District. 5th Public Health Association of South Africa (PHASA) Conference, Durban, South Africa. 30 November – 2 December 2009. (Poster Presentation)

## **ABSTRACT**

**Introduction:** The global burden of non-communicable diseases (NCDs) has long been neglected, with the omission of NCDs from the Millennium Development Goals (MDGs) bearing testament to this. The growing prevalence of chronic cardio-metabolic diseases in South Africa places huge demands on the health system. This study sought to determine the community prevalence of these cardio-metabolic diseases and associated risk factors in Chiawelo, Soweto – a township undergoing rapid urbanization in the Johannesburg Health District.

**Methods:** The study comprised 337 participants: 124 male and 213 female. This was a community based cross sectional survey using questionnaires, anthropometric and biochemical measurement of HbA<sub>1c</sub>. Cluster sampling techniques identified eligible adult participants. Regression models were performed to identify factors associated with disease. Ethical approval to conduct the study was obtained from the University of the Witwatersrand and written informed consent was obtained from the participants.

**Results:** The study population was black with middle to higher socio-economic status and education levels below Grade 12 mostly. The prevalence of diabetes mellitus (DM) in this study population was 14%, with many undiagnosed and those with disease poorly controlled. More than half the study population had hypertension (HPT) (58%) and most were poorly controlled. This was a markedly obese population (39%) with 54% of women having a body mass index (BMI) categorised as obese (BMI  $\geq$  30 kg/m<sup>2</sup>).

**Conclusions:** The burden of chronic cardio-metabolic diseases in the Johannesburg Health District has been grossly underestimated. The prevalence of HPT and DM was high and both diseases were poorly controlled with obesity reaching epidemic proportions. Countering the burden of disease involves targeting females as a high risk priority group, engaging the community in health promotion and developing a NCD surveillance system. Clinically, it is the findings of this study to support the screening of cardio-metabolic diseases from as early as 30 years of age in males and 40 years of age in females.

## ACKNOWLEDGEMENTS

1. Dr Debashis Basu, my supervisor, I am indebted to you for your patience and guidance throughout this project.
2. I must acknowledge the residents of Chiawelo, who allowed us to proceed with this study in their community, and graciously assisted us with data collection.
3. Brenda, Gugu, Mpho and Noku, my research assistants, for your tireless work and extra effort in completing the field work.
4. Mr. Michael Brown and Novo-Nordisk (Pty) Ltd. for the use of the Changing Diabetes<sup>®</sup> Bus and on-board facilities.
5. The Faculty Research Committee Grant 2009 awarded by the University of the Witwatersrand has greatly assisted in covering the costs of the biochemical testing.
6. Finally, to Jacqueline Mendes and Elvira Singh, I humbly thank you for supporting me through this project, my rotations and my time as a registrar.

## TABLE OF CONTENTS

DECLARATION .....	ii
DEDICATION .....	iii
PRESENTATIONS ARISING FROM THIS STUDY .....	iv
ABSTRACT .....	v
ACKNOWLEDGEMENTS .....	vii
LIST OF FIGURES .....	xi
LIST OF TABLES .....	xiii
LIST OF ABBREVIATIONS .....	xiv
GLOSSARY OF TERMS .....	xvi
<b>CHAPTER 1</b> .....	1
INTRODUCTION .....	1
1.1 BACKGROUND .....	1
1.2 JUSTIFICATION FOR THE STUDY .....	6
1.3 AIM AND OBJECTIVES .....	7
1.4 SUBSEQUENT CHAPTERS OF THIS REPORT .....	8
1.5 SUMMARY OF THE CHAPTER .....	9
<b>CHAPTER 2</b> .....	10
LITERATURE REVIEW .....	10
2.1 INTRODUCTION .....	10
2.2 THE IMPACT OF CARDIO-METABOLIC DISEASES .....	11
2.3 CARDIO-METABOLIC DISEASES AND ASSOCIATED RISK FACTORS .....	13
2.4 SOUTH AFRICAN MANAGEMENT OF CHRONIC DISEASES .....	24
2.5 THE EPIDEMIOLOGICAL TRANSITION .....	25
2.6 THE INDIVIDUAL IN THE CONTEXT OF THEIR ENVIRONMENT .....	26
2.7 DEVELOPING NON-COMMUNICABLE DISEASE SURVEILLANCE .....	31
2.8 SUMMARY OF THE CHAPTER .....	33



<b>CHAPTER 3</b> .....	34
STUDY METHODS AND MATERIALS .....	34
3.1    STUDY DESIGN.....	34
3.2    STUDY SETTING AND SCOPE.....	34
3.3    STUDY POPULATION AND SAMPLING .....	36
3.4    STUDY PERIOD .....	38
3.5    DATA COLLECTION.....	39
3.6    DATA MANAGEMENT AND MEASUREMENT.....	41
3.7    DATA ANALYSIS .....	51
3.8    PILOT STUDY .....	55
3.9    ETHICS .....	56
3.10   SUMMARY OF THE CHAPTER .....	56
<b>CHAPTER 4</b> .....	57
RESULTS .....	57
4.1    SOCIO-DEMOGRAPHIC FACTORS .....	57
4.2    CARDIO-METABOLIC DISEASES AND ASSOCIATED RISK FACTORS	62
4.2.1   CARDIOVASCULAR RISK FACTOR PROFILE.....	62
4.2.2   DIABETES MELLITUS .....	63
4.2.3   HYPERTENSION .....	71
4.2.4   OBESITY .....	80
4.3    ASSOCIATIONS BETWEEN RISK FACTORS.....	86
4.3.1   BMI vs. SBP .....	86
4.3.2   BMI vs. DBP.....	90
4.3.3   BMI vs. HbA1c.....	93
4.3.4   HbA1c vs. SBP .....	97
4.3.5   HbA1c vs. DBP.....	100
4.4    THE METABOLIC SYNDROME .....	101
4.5    SUMMARY OF THE CHAPTER.....	103

<b>CHAPTER 5</b> .....	104
DISCUSSION .....	104
5.1 INTRODUCTION.....	104
5.2 DIABETES MELLITUS .....	104
5.3 HYPERTENSION.....	108
5.4 OBESITY .....	112
5.5 ASSESSMENT OF CARDIOVASCULAR RISK FACTORS .....	113
5.6 THE METABOLIC SYNDROME .....	115
5.7 THE ROLE OF DIET AND PHYSICAL ACTIVITY .....	117
5.8 SOCIO-DEMOGRAPHIC FACTORS .....	119
5.9 PUBLIC HEALTH IMPLICATIONS OF THIS STUDY .....	122
5.10 LIMITATIONS.....	125
5.11 SUMMARY OF THE CHAPTER .....	127
<b>CHAPTER 6</b> .....	128
CONCLUSIONS AND RECOMMENDATIONS .....	128
6.1 CONCLUSIONS.....	128
6.2 RECOMMENDATIONS .....	132
6.3 SUMMARY AND CONCLUSIONS .....	142
REFERENCES .....	143
APPENDIX A: SAMPLING STRATEGIES .....	156
APPENDIX B: MEASUREMENT TOOLS.....	158
APPENDIX C: PERMISSIONS .....	167
APPENDIX D: PATIENT DOCUMENTS .....	169

## LIST OF FIGURES

<b>Figure</b>		<b>Page</b>
2.1	The role of insulin resistance and obesity in the MS	23
2.2	Understanding the individual in the context of their environment	26
2.3	The concept of the STEPwise approach	32
3.1	Regional map of Johannesburg indicating Chiawelo	35
3.2	Map of Chiawelo indicating recruitment sites	38
3.3	Participants outside the Changing Diabetes <sup>®</sup> Bus	39
3.4	Diagram showing values identified by Cook's distance	55
4.1	Age distributions by gender	59
4.2	Self reported DM	63
4.3	Prevalence of DM by gender	65
4.4	Prevalence of DM by age and gender	67
4.5	Levels of glycaemic control among diagnosed diabetics	68
4.6	Self reported HPT	71
4.7	Prevalence of HPT	73
4.8	Prevalence of HPT by category and gender	74
4.9	Prevalence of HPT by age and gender	76
4.10	Levels of control among diagnosed hypertensives	78
4.11	Prevalence of obesity by gender (n=337)	80
4.12	Categories of obesity by gender	81
4.13	Prevalence of obesity by age and gender	83
4.14	Scatter plot of SBP vs. BMI	86
4.15	Normality plots of residuals of SBP	87
4.16	Plot of studentized residuals vs. SBP	88
4.17	Plot of leverage values against squared residuals of SBP	89
4.18	Scatter plot of DBP vs. BMI	90
4.19	Normality plot of residuals of DBP	91
4.20	Plot of studentized residuals and DBP	92

<b>Figure (cont)</b>	<b>Page</b>
4.21 Scatter plot of HbA <sub>1c</sub> vs. BMI	93
4.22 Normality plot of residuals of HbA <sub>1c</sub> before and after removal of outliers	94
4.23 Scatter plot of HbA <sub>1c</sub> vs. SBP	97
4.24 Normality plot of residuals of HbA <sub>1c</sub> before and after removal of outliers	98
4.25 Scatter plot of HbA <sub>1c</sub> vs. DBP	100
6.1 Prevalence of HPT identifying target ages for screening	136
6.2 Prevalence of DM identifying target ages for screening	138

## LIST OF TABLES

<b>Table</b>	<b>Page</b>
2.1 Obstacles to adherence	27
3.1 Inter-operator variability for measurements	41
3.2 Description of variables used in the analyses	43
3.3 Level of DM disease control described by HbA <sub>1c</sub> levels	49
3.4 Level of HPT disease control described by BP readings	49
3.5 Indications for statistical tests	51
4.1 Demographic characteristics of the study population (n=337)	58
4.2 Cardiovascular risk factor profile (n=337)	62
4.3 Reporting of DM by gender (n=326)	64
4.4 Prevalence of DM by age and gender (n=326)	66
4.5 Factors associated with DM on bivariate and multivariate regression (n=326)	70
4.6 Reporting of HPT by gender (n=337)	72
4.7 Prevalence of HPT by age and gender (n=337)	75
4.8 Factors associated with HPT on bivariate and multivariate regression (n=337)	79
4.9 Prevalence of categories of BMI by age and gender (n=337)	82
4.10 Factors associated with obesity on bivariate and multivariate regression (n=337)	85
4.11 Prevalence of the MS and associated risk factors (n=337)	102
4.12 Factors associated with the MS on bivariate and multivariate analysis (n=337)	103

## LIST OF ABBREVIATIONS

ADA	American Diabetes Association
AIDS	Acquired Immuno-Deficiency Syndrome
aOR	Adjusted odds ratio
BMI	Body mass index
BP	Blood pressure
CAD	Coronary artery disease
CARDIA	Coronary Artery Development in Young Adults Study
CHC	Community health centre
CVA	Cerebro-vascular accident
CVD	Cardio-vascular disease
DALY	Disability adjusted life year
DBP	Diastolic blood pressure
DHIS	District Health Information System
DM	Diabetes mellitus (Type 2 DM specifically)
HAART	Highly Active Anti-Retroviral Therapy
HDL	High density lipoprotein
HIV	Human Immuno-deficiency Virus
HPT	Hypertension
IDF	International Diabetes Federation
IGT	Impaired glucose tolerance
IHD	Ischaemic heart disease
IQR	Inter-quartile range
JNC7	The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
MDG	Millennium Development Goals
MI	Myocardial infarction
MRFIT	Multiple Risk Factor Intervention Trial
MS	Metabolic syndrome
NCD	Non-communicable disease

### **Abbreviations (cont)**

NCEP (ATP) III	National Cholesterol Education Programme Adult Treatment Panel III
NHANES III	National Health and Nutrition Examination Survey III
NHLS	National Health Laboratory Service
OR	Odds ratio
PHC	Primary health care
PI	Principal investigator
POWIRS	Profiles of Obese Women with the Insulin Resistance Syndrome
RA	Research assistant
SADHS	South African Demographic and Health Survey
SANAS	South African National Accreditation System
SBP	Systolic blood pressure
SD	Standard deviation
SEMSDA	Society for Endocrinology, Metabolism and Diabetes of South Africa
SES	Socio-economic status
UKPDS	United Kingdom Prospective Diabetes Study
UN	United Nations
WC	Waist circumference
WHA	World Health Assembly
WHO	World Health Organization

## GLOSSARY OF TERMS

**Diabetes mellitus:** For the purposes of this study, diabetes mellitus was classified using the American Diabetes Association classification of glycated haemoglobin (HbA<sub>1c</sub>) (1):

- Normal: < 5.7%
- Impaired glucose tolerance (IGT): 5.7 – 6.4%
- Diabetes: ≥ 6.5%

Those with IGT have been referred to as having pre-diabetes, indicating the relatively high risk for the future development of diabetes.

**Dyslipidaemia:** Refers to a clinically significant alteration in the circulating lipids and lipoproteins predisposing to coronary artery disease (2). The 2000 South African clinical guideline for the classification of hypercholesterolaemia (mmol/l) is as follows (3):

	Desirable lipid profile	Hypercholesterolaemia	
		Moderate	Severe
Total cholesterol	≤ 5.0	5.0 – 7.5	> 7.5
LDL-cholesterol	≤ 3.0	3.0 – 5.0	> 5.0
HDL-cholesterol	≥ 1.2	variable	variable
Triglycerides	≤ 1.5	< 1.5	< 1.5



**Epidemiological transition:** The term ‘epidemiologic transition’ describes the change in disease patterns associated with economic development (4). There has been a drastic shift in mortality from infectious disease and malnutrition before the 1900s to CVD and cancer currently in most high-middle income countries (5).

**Hypertension(6):** The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure was used to define hypertension:

<b>BP classification</b>	<b>SBP (mmHg)</b>	<b>DBP (mmHg)</b>
Normal	<120	and < 80
Prehypertension	120 - 139	or 80 - 89
Stage 1 hypertension	140 - 159	or 90 - 99
Stage 2 hypertension	≥ 160	or ≥ 100

Patients with prehypertension are at increased risk for progression to hypertension; those in the 130–139/80–89 mmHg BP range are at twice the risk to develop hypertension as those with lower values. The classification “prehypertension” signals the need for increased education of health care professionals and the public to reduce BP levels and prevent the development of hypertension in the general population.

**Insulin resistance (7):** Resistance to insulin-mediated glucose uptake is characteristic of individuals with impaired glucose intolerance or non-insulin-dependent diabetes. It also occurs commonly in patients with high blood pressure.

**Metabolic syndrome:** Represents a constellation of inter-related risk factors of metabolic origin that include diabetes mellitus, hypertension, obesity and dyslipidaemia (8). The International Diabetes Federation defines the metabolic syndrome as (9):

Central obesity (defined as waist circumference of  $\geq 94$ cm for males and  $\geq 80$ cm for females) plus any two of the following four factors:

Raised triglycerides	– $\geq 1.7$ mmol/l
	– Or specific treatment for this lipid abnormality
Reduced HDL cholesterol	– $< 1.03$ mmol/l (males) or $< 1.29$ mmol/l (females)
	– Or specific treatment for this lipid abnormality
Raised blood pressure	– SBP $\geq 130$ mmHg or diastolic BP $\geq 85$ mmHg
	– Or treatment of previously diagnosed hypertension
Raised fasting plasma glucose	– $\geq 5.6$ mmol/l
	– Or previously diagnosed type 2 diabetes

**Obesity and overweight:** BMI is defined as weight in kilograms divided by the square of height in metres ( $\text{kg/m}^2$ ) (10). The World Health Organisation defines the following weight categories (11):

<b>Classification</b>	<b>BMI (<math>\text{kg/m}^2</math>)</b>
Underweight	$< 18.50$
Normal range	18.50 - 24.99
Overweight	$\geq 25.00$
Obese	$\geq 30.00$
- Obese class I	30.00 - 34.99
- Obese class II	35.00 - 39.99
- Obese class III	$\geq 40.00$

Source: Adapted from WHO 1995, WHO 2000 and WHO 2004

**Residual values:** The difference between the observed and predicted values in a regression model.

**Risk factor:** Refers to an attribute, characteristic, or exposure of an individual, which increases the likelihood of developing a non-communicable disease (12).

**Studentized residual values:** This is the residual value divided by the standard error/standard deviation of the value.

# **CHAPTER 1**

## **INTRODUCTION**

This chapter outlines the background and justification for the study. The aim and objectives of the study are described and the subsequent chapters are detailed.

### **1.1 BACKGROUND**

#### **1.1.1 The global impact of chronic diseases**

The United Nations (UN) member states will gather in New York in September 2011 for the first UN High-level Summit (UN Resolution 64/265) on non-communicable diseases (NCDs) (13). High on the agenda for discussion is the prevention, early detection, diagnosis and management and importantly, the investment in effective and measurable national control programmes for these diseases (13).

The Millennium Development Goals (MDGs) were launched in 2000. Three of these goals focus specifically on health issues (14):

- MDG 4: Reduce child mortality
- MDG 5: Improve maternal health
- MDG 6: Combat Human Immuno-deficiency Virus (HIV) and Acquired Immuno-Deficiency Syndrome (AIDS), malaria and other diseases

Non-communicable diseases, which are a substantial cause of death and disability globally, are a very apparent omission from this list of ambitious targets intended to improve the lives of people, particularly the poorest, all over the world. Health and development are inextricably linked; one cannot be achieved without the other. The exclusion of NCDs from the MDGs has been a major barrier to securing funding for NCDs as many donors exclusively fund the health priorities contained within the MDGs (15).

Attempting to prioritise NCDs has been on the global health agenda for a more than a decade. From as early as May 2000, the resolution on *Prevention and Control on non-communicable diseases* (WHA53.17) was adopted at the 53rd World Health Assembly (WHA) in recognition of the growing epidemic of these chronic diseases (16;17). The fundamental outcomes of this assembly called for establishing programmes for the prevention and control of NCDs, assessing and monitoring morbidity attributable to NCDs, promoting the effectiveness of secondary and tertiary prevention and supporting the development of guidelines for cost-effective screening, diagnosis and treatment for NCDs (16).

The 2011 Summit represents the biggest and best opportunity to put NCDs on the global agenda. The Summit has the potential to secure commitment from heads of government for a coordinated global response to NCDs through substantial increases in financial resources allocated to NCDs (15;18). This broad political and financial commitment will save millions from premature death and debilitating health complications.

Chronic cardio-metabolic diseases, particularly cardiovascular disease (CVD) and diabetes mellitus (DM), represent 43% of the global burden of disease and account for 50% of all mortality worldwide (19); with an increase in mortality to 73% anticipated by 2020 (20). Given this enormous burden of disease, it is not surprising that NCDs are being considered in global development targets, most notably the successor goals to the current MDGs that are set to expire in 2015 (13).

The metabolic syndrome (MS) is an important concept in chronic disease management and control. Defined as *'multiple, interrelated risk factors of metabolic origin that appear to directly promote the development of atherosclerotic cardiovascular disease'*, the MS represents a constellation of diseases that include DM, hypertension (HPT), obesity and dyslipidaemia (8). There has been a striking increase in the global prevalence of the MS with obesity and DM being the major contributing culprits (21). The underlying risk factors (defined in: Glossary of terms) to each of these diseases are well documented and their prevention and control, through comprehensive population-based programmes, have time and again proven to be the most cost-effective interventions to counter the emerging epidemic (12). For the purposes of this study, the MS is defined as described in Chapter 3: 3.6.1.2 Clinical variables (Metabolic syndrome).

### **1.1.2 Chronic diseases in developing countries**

The overall age-specific rates of NCDs are higher in sub-Saharan Africa than in populations in Established Market Economies (22). Developing countries have competing health demands that restrict the capacity of their resource-limited health services from curbing the dramatic increases in the burden of NCDs (12). This translates to a growing prevalence of diseases as HPT and DM, particularly in urban areas, placing additional huge demands on the health care system (22).

Much of this increase in NCDs in developing countries result from the ‘epidemiological transition’ (defined in: Glossary of terms), adoption of poor diet and sedentary lifestyles (20) and high levels of illiteracy (23). Studies have shown the need for health care providers to incorporate patient education and evidence-based measures to reduce the risk of CVD and DM through global risk assessment and screening complimented by lifestyle modification (24).

### **1.1.3 Chronic diseases in South Africa**

South Africa is in the midst of a health transition that is characterised by the simultaneous, quadruple burden of poverty related infectious diseases (including HIV infection) and a rise in NCDs, in a population facing a profound level of perinatal and maternal disorders, injury, and violence (25). Health system strengthening, globally and in South Africa, focuses much attention on maternal and child health and infectious

diseases with a lesser emphasis placed on chronic diseases (26;27). The fact remains that South Africa constitutes one of the 23 countries globally that are responsible for 80% of the burden of chronic diseases in all middle-income and low-income countries combined (26). Aside from urbanisation and its perils, there is a growing prevalence of chronic disease risk factors in the rural areas of South Africa as well, placing this population at increased risk of developing a CVD event in the following ten years (28).

HIV infection, and not cardiovascular disease, is the leading cause of mortality in South Africa with the infection rate being the highest in the world (5.4 million people infected in 2006) (29;30). The complex interaction between HIV and CVD (the second largest contributor to mortality in South Africa) should not be under-emphasized as HIV infection and highly active anti-retroviral therapy (HAART) are clearly implicated in the pathogenesis of CVD adding to the overwhelming burden of NCDs in South Africa (29).



## **1.2 JUSTIFICATION FOR THE STUDY**

Leading a multi-sectoral response to NCDs requires mapping the emerging epidemiology of NCDs, reducing the levels of exposure of individuals to the modifiable risk factors and strengthening the health system to combat NCDs through the development of evidence-based standards and norms (31). It is well documented that low-income and middle-income countries lack the health system capacity and surveillance infrastructure to adequately reduce the disease burden and both these structures need to be urgently strengthened (32). Most of the available NCD data have been generated outside of Africa and it is unclear how transferable this causation and prevention data is to Africans given their differing social, economic and cultural structures (22). Reliable data on the burden and causes of NCDs in sub-Saharan Africa is known to be lacking (22). Thus, the logical first step to any NCD surveillance programme would be to conduct a baseline survey of the target population. The surveillance data generated would allow for more accurate prediction of incidence and prevalence rates within the community which is essential for interventions aimed at reducing morbidity and mortality.

Efforts to scale-up interventions in low-income and middle-income countries generally tend to concentrate on a single disease resulting in a service that is fragmented and vertical (33). The South African health care system adopts a similar approach. These efforts represent continued missed opportunities to providing comprehensive chronic disease care. Screening for a cluster of related diseases thus makes more sense, as it would allow for earlier diagnosis and management of NCDs.

This study will provide broad insights into the prevalence of CVD risk factors in the South African black population as the urban community of Soweto represents the largest township in sub-Saharan Africa with the highest concentration of black Africans on the continent (34). The study will also provide the initial surveillance data for NCDs in this community – an initiative that would contribute to the planning and organisation of health services tailored to the needs of the community.

### **1.3 AIM AND OBJECTIVES**

#### **1.3.1 Aim**

To determine the prevalence of cardio-metabolic diseases and the associated risk factors in the community of Chiawelo, Johannesburg Health District, Gauteng Province with a view to reducing the burden of disease in terms of long term management and development of complications.

### **1.3.2 Objectives**

1. To describe the study population in terms of socio-demographic factors
2. To explore the prevalence and risk factors associated with the development of the following cardio-metabolic diseases:
  - a. Diabetes mellitus
  - b. Hypertension
  - c. Obesity
3. To examine the associations among these major risk factors
4. To determine the community prevalence of the MS and the major risk factors that predispose individuals in this community to the development of the MS (as defined in Chapter 3: 3.6.1.2 Clinical variables (Metabolic syndrome))

## **1.4 SUBSEQUENT CHAPTERS OF THIS REPORT**

Thus far, the background to the research has been explained and the objectives have been defined. The subsequent chapters will focus on:

Chapter 2: Literature review

The purpose of the literature review is to discuss key concepts and examine potential solutions to the research problem.

### Chapter 3: Study methods and materials

This chapter describes the research methodology used to conduct this study.

### Chapter 4: Results

This chapter presents the data analysis according to the aim and objectives.

### Chapter 5: Discussion

The literature review is integrated with the study findings from the preceding chapter in view of the aim and objectives of the study.

### Chapter 6: Conclusions and recommendations

In this chapter, conclusions are drawn from the report, and suggestions are formulated considering the current literature and the findings of the study.

## **1.5 SUMMARY OF THE CHAPTER**

This introductory chapter described the background to the study, the justification for the research and the objectives of the study. A summary of the subsequent chapters were then briefly described.

## **CHAPTER 2**

### **LITERATURE REVIEW**

This chapter examines the current literature on cardio-metabolic diseases in the context of the individual, the prevalence of these diseases and the South African health system. The epidemiological transition in South Africa is explored and finally the components of a NCD surveillance strategy are discussed.

#### **2.1 INTRODUCTION**

*‘Governments have a responsibility to support their citizens in their pursuit of a healthy, long life. It is not enough to say, we have told them not to smoke, we have told them to eat fruit and vegetables, we have told them to take regular exercise. We must create communities, schools and workplaces and markets that make these healthy choices possible. We must tackle this problem step by step and we must start now.’ ~ Nigerian President Olusegun Obasanjo (35).*

Chronic cardio-metabolic diseases are a growing cause of death and disability in South Africa. As the country moves through an ‘epidemiological transition’, the determinants and risk factors for disease development changes (36). It is vital to understand the inherent risk factors within the community to understand the context in which disease develops.

The relationship between the community and the health services in managing these conditions also needs to be explored. This understanding serves as a foundation to plan appropriate interventions and allocate resources.

## **2.2 THE IMPACT OF CARDIO-METABOLIC DISEASES**

### **2.2.1 Global impact**

Cardio-metabolic diseases are a matter of global concern. It is predicted that the incidence of DM will double by 2025 and this will be paralleled by an inevitable rise in CVD (9). The complications of DM and CVD and the resultant blindness, amputation and renal failure account for much of the social and financial burden (9).

The nine million deaths that NCDs are responsible for in people younger than 60 years of age annually bears testament to the magnitude of the problem and the earlier onset of disease (13). There are increasing numbers of children afflicted with these diseases, especially obesity and DM (37) and it must be appreciated that interventions targeting children have many health gains, albeit in the longer term (38). The impact that this will have on global healthcare systems is phenomenal.

### **2.2.2 Developing countries**

It is globally predicted that mortality attributable to NCDs will increase by 77% from 1990 – 2020 (17). Four African countries, including South Africa, feature in the list of the 23 countries that account for 80% of the total chronic disease mortality burden in developing countries (39) where the majority of these deaths will occur (17). Regional World Health Organization (WHO) data from 2005 indicated that chronic metabolic diseases accounted for 23% of all deaths in the African region (40). If no effective measures are put in place, an estimated US \$ 84 billion of national income will be lost from heart disease, CVA, and DM alone in these 23 countries between 2006 – 2015 (39).

In the poorest of sub-Saharan countries, chronic metabolic diseases are becoming more prevalent despite communicable diseases continuing to predominate. Evidence of DM and HPT are on the increase in some urban African settings, but also in rural African settings (28). As urbanization increases, so too does the burden of chronic diseases. By 2015, it is estimated that the mortality in Africa from communicable diseases will be exceeded by chronic metabolic diseases (4). It is therefore imperative that greater emphasis is placed on the prevention and control of these diseases.

### **2.2.3 South Africa**

The South African health services are grappling with the quadruple burden of disease: pre-transitional diseases of poverty and underdevelopment, the chronic diseases of lifestyle due to urbanization, injuries (intentional and unintentional) and HIV/AIDS (41). South Africa has limited resources but multiple demands are being placed on the health services. Chronic metabolic diseases do not pose a significant priority when competing with acute conditions such as trauma and communicable diseases (36). As a result, provisions made for these diseases are unlikely to be adequate and programmes aimed at combating the emergence of associated risk factors probably receive the least attention in South Africa's health related activities (36).

## **2.3 CARDIO-METABOLIC DISEASES AND ASSOCIATED RISK FACTORS**

### **2.3.1 Cardiovascular disease**

Cardiovascular mortality increases substantially with higher, more poorly controlled levels of blood glucose, accounting for 1 490 000 deaths from ischaemic heart disease (IHD), 709 000 deaths from CVA and 959 000 deaths directly associated with DM (42). A global CVD epidemic is evolving with twice as many deaths in developing countries as in developed countries (43). High blood pressure (BP), high cholesterol levels, tobacco and alcohol use and poor diet are emerging as major risk factors for CVD (5).



In addition to the morbidity and mortality, CVD poses a great financial burden to developing countries to both the health care system and to the national economy (5). Figures from as early as 1991 showed that South Africa devoted two to three percent of the country's gross national income or approximately 25% of the health expenditure on the treatment of CVD (44). The fact that a high proportion of CVD falls on adults of working age has a further impact on the economy. Conservative estimates indicate that in five countries (including South Africa), at least 21 million years of future productive life are lost due to CVD annually (5).

Numerous previous cross sectional studies conducted in South Africa had decisively shown that people living a larger proportion of their lives in an urban setting had significantly higher rates of DM and HPT than those who had spent a smaller proportion of their lives in the city (45;46). This picture is quickly changing. The effects of urbanization on the development of these chronic diseases are starting to be felt with the constant migration of people into the urban areas. The aetiology of the higher levels of CVD and mortality in the black population than the white population seems to multi-factorial with obesity, urbanisation and adoption of a Westernised lifestyle having a huge contributory role (47).

### **2.3.2 Diabetes mellitus**

Diabetes mellitus is a global epidemic which drastically impacts on the quality of life of those affected. By 2008, the global prevalence of DM was estimated at 171 million, with an expected increase to 366 million by 2030 (48). The rapid rates of urbanisation and poor lifestyle choices made are largely responsible for this (49). Historically, DM had been a disease restricted to affluent urban areas but the effects of Westernisation have resulted in the disease being more and more prevalent in the rural black populations (50). Sub-Saharan Africa has an escalating burden of DM mostly resulting from lifestyle factors encompassing low levels of physical activity, poor dietary choices laden with fat and energy and high levels of obesity (49;51;52).

The burden of DM in South Africa is also unacceptably high with rates higher than the African average (53). In 2000, mortality from DM was estimated at 20 000 which accounted for 4.3% of all deaths in South Africa – the 7th commonest cause of death in the country (53). Diabetes mellitus prevalence rates of between 3 – 10% have been noted urban and peri-urban areas in South Africa, a stark contrast to the less than 1% noted between the 1960s – 1980s (49). The Framingham Heart Study also demonstrated a doubling of DM incidence between the 1970s-1990s in 3104 participants aged between the 40 – 55 years who were initially free of DM (52). To complicate matters further, the INTERHEART study found DM was a prominent risk factor for MI in black South Africans (42).

The consequences of DM are sobering. Those with DM are at an increased risk of CVD, blindness and renal failure (54). This highlights the importance of the MS as it is a recognised precursor to DM and CVD (55). There is limited research on the incidence of DM in South Africa, yet despite this, there is clear evidence suggesting that under-diagnosis of DM is occurring (56).

### **2.3.3 Hypertension**

Hypertension is a commonly occurring chronic disease that is universally under-diagnosed and poorly treated resulting in extensive target organ damage and death (36). Hypertension is responsible for a growing burden of disease both of established importance in middle-income countries and of emerging importance in low-income countries (57). In the year 2000, estimates suggest that HPT accounted for 47 000 deaths in South Africa which represents 9% of all mortality and 2.4% of total disability adjusted life years (DALY) (57).

Hypertension is the most common CVD risk factor among the black population with a prevalence of 59% (58). The INTERHEART study reinforced the predilection that cardio-vascular complications associated with HPT has for the black population (42). Urbanisation predisposes the black population to developing HPT, a trend that will continue to grow as urbanisation continues to flourish (57). People living in Gauteng report higher prevalence of HPT than the national figures. This is likely due to urban environments, such as Gauteng, tending to have higher salt consumption, increased alcohol consumption and tobacco use and unhealthier diets (56).

In a study conducted in Gauteng of 40 black patients with established CAD, it was found that 95% had previously diagnosed HPT with 50% having elevated glucose concentrations (59). Research has shown that the rural/urban divide in HPT prevalence is not as apparent anymore, with levels similar to those in urban areas being seen in rural areas (56).

#### **2.3.4 Obesity**

The WHO estimates that globally about one billion adults are overweight and about 300 million are clinically obese (5). This is paralleled by increases in childhood obesity that result in increased levels of DM and HPT (5). Accumulation of abdominal fat, particularly in the visceral compartment, is associated with an increased risk of insulin resistance, DM, HPT, dyslipidaemias and atherosclerosis (60). This visceral adiposity is the cornerstone of the MS.

Despite South Africa's pressing concerns of under-nutrition, poverty and infectious diseases such as HIV and tuberculosis, more than 29% of males and 56% of females were classified as obese or overweight (61). The 1998 South African Demographic and Health Survey (SADHS) analysed data from 7726 South African women aged 15 – 95 years old and found the highest prevalence of overweight and obesity in the female black population (59%) (61). The report also showed associated higher BMI with women from urban areas and increasing age with central obesity particularly prevalent in urban black females (42%) (61).

Health promotion and prevention strategies are becoming increasingly more important when considering the growing levels of childhood obesity and chronic diseases. The 1999 South Africa National Food Health Consumption Survey showed that the national average of being overweight in children aged 1 - 9 years was 6% while only 5% were classified as 'wasted' and almost 25% as 'stunted' (37). Children who are obese at six years of age have a probability exceeding 50% of obesity in adulthood, particularly if the parents are overweight (62).

There are many social considerations to obesity in South Africa. Many black women perceive being overweight as desirable (63), despite Western ideas to the contrary. This belief is promulgated by the idea that being thin is associated with having HIV/AIDS infection, and thus being thin stigmatises the individual as such (63). Larger body sizes in the black community is also associated with affluence and happiness (63). Obesity or overweight in females is thought to reflect on a husband's ability to care for his wife and family (64). These factors culminate in obesity being a growing epidemic in South Africa. Primordial prevention should be addressed, especially from an early age, by promoting physical activity and encouraging healthy diets.

### **2.3.5 Dyslipidaemia**

Dyslipidaemia includes hypertriglyceridaemia, reduced HDL cholesterol and usually hypercholesterolaemia and forms part of the insulin resistance syndrome (65;66). Dyslipidaemia remains a major risk factor for CVD in the South African population and is increasing with the progressive urbanisation of the black population (56). In 2000, South African data showed that 59% of IHD and 29% of ischaemic stroke was attributable to raised cholesterol levels (67).

Previous South African studies have documented that 28% of black African people aged 30 years and over had cholesterol levels in excess of the clinical cut-off point of 5mmol/l (67). Higher cholesterol levels have been found in younger black South Africans implicating the earlier adoption of a Westernised lifestyle (67). This finding suggests that screening for high cholesterol in the black population begin as early as 30 years of age (67). Individuals resident in Gauteng report higher levels of hyperlipidaemia prevalence than national rates, again largely attributable to the adoption of a Westernised lifestyle (56). Females seem to be more afflicted than the male population; but this probably reflects the better health-seeking behaviour in females (67).

The HIV epidemic has to be considered when evaluating hyperlipidaemia as HAART is directly responsible for metabolic changes resulting in truncal obesity and changes in the composition of lipids (56).

Again, dyslipidaemia is inextricably linked to other chronic diseases with at least 30 – 50% of patients with DM having abnormal lipid patterns and similar rates reported among the hypertensives (65;67). Current data suggests that dyslipidaemia in South Africa is not a commonly recognized entity. As such, it is rarely diagnosed and poorly treated (67). If the South African health system is to have some semblance of control over the IHD and CVA rates, dyslipidaemia recognition and management needs to be drastically improved.

### **2.3.6 Physical inactivity and poor dietary choices**

Evidence points to the growing prevalence of CVD and DM being largely attributed to lifestyle-dependant risk factors such as poor levels of physical activity, transition in diets that are associated with NCDs (68) and rising levels of obesity (69). Interventions directed towards a global strategy on diet, physical activity and health featured significantly on the 2008 61st WHA agenda (70) on endorsing plans to prevent and control NCDs (71;72).

There are no specific studies in Gauteng documenting the levels of physical activity of adults (56). Data emanating from other parts of the country show that most individuals did not engage in physical activity, particularly females and residents of rural areas (56). Twenty different cohort studies strengthen the claim that physical activity decreases the risk of DM by between 20-30% (51). Physical activity modifies the risk of DM through the prevention of obesity, but impacts on DM risk in the presence or absence of obesity

(51). These findings of reduced incidence of DM achieved without weight loss but with physical activity is substantiated by the Indian Diabetes Prevention Programme and the Da Qing IGT and Diabetes Study (73;74). Lifestyle modification has also been shown to be beneficial in BP management (75;76).

Many studies have shown that the rapid urbanisation that the black population has been subjected to has resulted in an important change in their food consumption from a traditional high carbohydrate / low fat diet to a diet high in saturated fats and sugars (29). In the USA alone, poor diet and physical inactivity accounted for 20% of adult deaths in 2000 (20). In South Africa, poor diet, defined by low fruit and vegetable intake, accounted for 3% of mortality and 1% of the 16.2 million attributable DALYs in 2000 (77).

### **2.3.7 The metabolic syndrome**

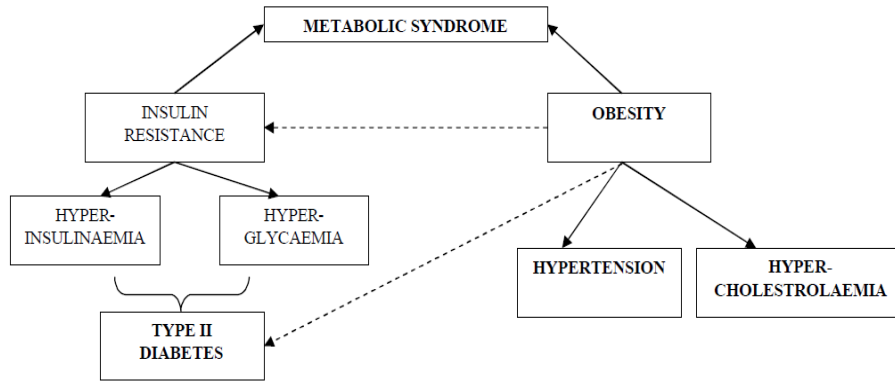
The MS cluster represents the most critical risk factors for the development of a myocardial infarction (MI) including DM, raised fasting plasma glucose, abdominal obesity, dyslipidaemia (defined in: Glossary of terms) and HPT (9). The diagnosis of the MS substantially increases the risk of DM, coronary artery disease (CAD) and premature mortality (78). The relationship between the MS and CVD, in some cases described as almost double the risk (79), is well established irrespective of the definition used for the MS (80). Estimates suggest that 20 – 25% of the world's adult population have the MS (9). Those individuals with the syndrome are twice as likely to die from and three times as likely to have a MI or a cerebro-vascular accident (CVA) compared to those without



the syndrome (9). Additionally, those with the MS are five times more likely to develop DM (81). The clustering of diseases comprising the MS is considered to be the driving force of a new cardiovascular disease epidemic (9). In fact, the Strong Heart Study, a ten year longitudinal follow up of close to 4000 participants, found that the MS was highly associated with incident CVD in those known to have DM (82).

Data from Finland found the prevalence of the MS to range from 4% in 24 year olds to 25% in 39 year olds, with most of the increased prevalence attributed to obesity (83). In the USA, the prevalence of being overweight or obese in children has tripled between 1970 – 2000, with a corresponding increase in the MS in this young group (84). Other studies confirm these findings with the MS rates in adolescents at 12% using the modified National Cholesterol Education Programme Adult Treatment Panel III [NCEP (ATP) III] criteria and a prevalence of 10% in children aged 12-19 years of age from the National Health and Nutrition Examination Survey (NHANES) III cohort using the same criteria (85).

The underlying causes of the MS remains unclear though insulin resistance (defined in: Glossary of terms) and central obesity have been significantly implicated (86;87). Insulin resistance results in hyperinsulinaemia and hyperglycaemia (Type 2 diabetes-[DM]) (9). Obesity is associated with insulin resistance and DM (9). Central obesity has been found to be more indicative of the MS than body mass index (BMI) measurements (88). Obesity contributes to HPT, hypercholesterolemia, low high density lipoprotein (HDL) and hyperglycaemia, and is an independent risk factor for CVD (86;89;90). Figure 1 reflects the complex interplay of these factors in the causation of MS.



**Figure 2.1: The role of insulin resistance and obesity in the MS**

In South Africa, the burden of the MS seems to fall predominantly on black women. The NHANES III findings support the data that women, of African-American descent, have higher prevalence of the MS than their male counterparts with the prevalence in white males exceeding that of black males (91;92). The findings in Johannesburg are similar, with females having more severe MS (characterised by the presence of four or five criteria) and males having lower prevalence rates across both black and white populations (93).

## 2.4 SOUTH AFRICAN MANAGEMENT OF CHRONIC DISEASES

Chronic cardio-metabolic diseases have historically been poorly managed in South Africa. Studies conducted in the Cape Peninsula reviewed the care received by 923 patients with HPT and 455 with DM attending 18 community health centres (CHC) (94). There was 23% level of control among hypertensives and of those with diabetes as well, only 21% had a BP below the recommended level of 130/80 mmHg (94). In the DM patients, 42% had a random blood glucose above 11.1 mmol/l and 76% had a HbA<sub>1c</sub> level  $\geq 1\%$  of the upper limit of normal (94). Numerous audits of patient folders have revealed that patients attending CHCs receive poor quality routine care for DM and HPT. The already poor levels of hypertensive and glycaemic control is exacerbated by the limited assessment of patients for target organ damage as reflected in their patient notes (36).

Community health centres in Cape Town that had formed 'chronic care teams' that meet regularly to discuss their goals and plan improvements through more structured and systematic approaches to patient care have showed vast improvement in the annual review of their diabetic patients in PHC (95). Current management principles are targeted at prevention and early treatment to minimise the development of complications (96). Various other factors are implicated in the inadequate clinical examinations and care that these patients receive at clinic level. These include human resource issues, erratic drug supplies and complex tertiary cases from tertiary centres referred back to the clinic for management (94).

## 2.5 THE EPIDEMIOLOGICAL TRANSITION

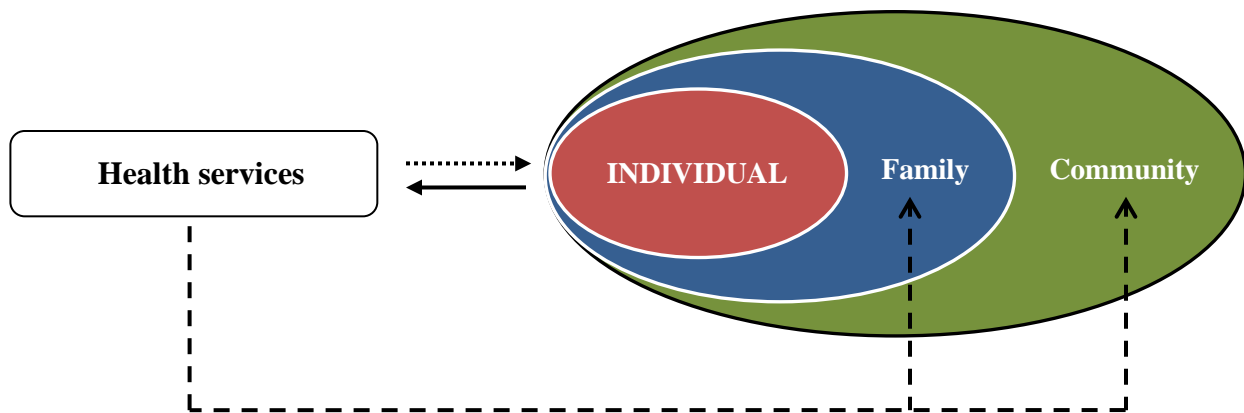
The term ‘epidemiologic transition’ describes the change in disease patterns associated with economic development (4). There has been a drastic shift in mortality from infectious disease and malnutrition before the 1900s to CVD and cancer currently in most high-middle income countries (5). This can be attributed to the economic and social transformations resulting from industrial and technological revolutions that have transpired over the last two centuries (5).

Developing nations have to contend with the emergence of chronic disease borne out of industrialization and the adoption of a Westernised lifestyle against a backdrop of poverty related illnesses (16). This double burden of disease is crippling to the economy and healthcare demands of the country. It is vital that such economies utilize their limited resources optimally and implement cost-effective health promotion interventions (16).

Most of Africa is undergoing an epidemiological transition (35). By 2004, the World Bank still considered sub-Saharan Africa to be in the stage of pestilence and famine (Stage 1) despite CVD being the leading cause of death in those greater than 30 years of age in the region (5). South Africa appears to be approaching the age of degenerative man-made diseases (Stage 4) characterised by *‘lifestyle changes in diet, activity levels, and smoking that set the stage for the emergence of atherosclerosis’* (5). Current trends suggest that many developed countries are entering the unnamed Stage 5 of epidemiological transition characterised by epidemics of obesity and DM (5).

## 2.6 THE INDIVIDUAL IN THE CONTEXT OF THEIR ENVIRONMENT

Given the massive burden of communicable and NCDs that the public health services contends with, the sustainability of interventions aimed at prevention and control of chronic diseases directed purely from the health system seems less likely. Tackling obesity and chronic diseases should become a ‘family matter’ as the lifestyle changes would prove beneficial to the entire family especially considering the prevalence in adults and children alike. The health system has an important role in facilitating these processes. Figure 2.2 describes the interaction of the individual in the context of his environment.



**Figure 2.2: Understanding the individual in the context of their environment**

### 2.6.1 The contribution of the individual to his disease

Health reform post apartheid has seen increases in the numbers of peripheral clinics and screening programmes reaching the masses and yet disease rates remain high (50). Poor adherence to treatment is largely responsible for uncontrolled HPT (97) but the factors implicated are easily applicable to most chronic diseases and are described in Table 2.1.

**Table 2.1: Obstacles to adherence**

<b>Treatment characteristics</b>	<b>Patient and illness characteristics</b>
<ul style="list-style-type: none"><li>▪ Long duration of therapy</li><li>▪ Complicated regimens</li><li>▪ Expensive medications</li><li>▪ Side-effects of medications</li><li>▪ Lack of specific appointment times</li><li>▪ Long waiting period at clinic or office</li><li>▪ Lack of consistent and continuous primary care</li><li>▪ Instructions not understood</li><li>▪ Organic brain syndrome (e.g. memory deficit)</li><li>▪ Medicines not available</li></ul>	<ul style="list-style-type: none"><li>▪ Asymptomatic nature of the condition leaves people feeling that they are not ill</li><li>▪ Chronic conditions require constant attention</li><li>▪ There are no immediate consequences of stopping, e.g. one does not feel sick</li><li>▪ Social isolation</li><li>▪ Disrupted home situation</li><li>▪ Psychiatric illnesses</li></ul>

Source: South African Hypertension Guideline 2006 (97)

Much of the answer lies with the individual. Attaining optimal disease control hinges on the individual in the community having an understanding of the disease (50). Patients understanding their disease processes allows them to assess their risk of disease, motivates them to seek appropriate treatment and, to some degree, to take control of the disease in their lifetime (50;98;99).

The point of educating patients about their disease is not to increase their knowledge concerning the disease but rather to adapt the management and control of the disease to the constant changes occurring in the patients' life (100). While this data refers specifically to the self-management of DM, the principle is applicable to all diseases of lifestyle.

Another important concept that emerges with progressive urbanisation and Westernisation of lifestyles is that of affluence. Affluence has rendered the higher-income black South African more susceptible to MI than the high-income white or other non-black counterparts presumably due to being in a different stage of the epidemiological transition (35).

### **2.6.2 The role of the community**

Community resources serve as an invaluable adjunct to health care services in the management of chronic diseases. Patients spend most of their time within a community and not linked to a health system. Thus, communities are ideally placed to raise the profile of diseases, generate awareness about their associated risk factors and reduce stigma through involvement of leaders, non-governmental organisations and local support groups (101).

In fact, the large numbers of PHC clinic visits related to DM in Gauteng for 2006/07 (472 847 visits in total) were attributed in part to the 73 support groups for DM in the province (56). The community thus plays a vital role in health promotion and prevention campaigns and training of community health workers (101).

### **2.6.3 The impact of health services**

Given the overwhelming burden of infectious diseases in both low-income and middle-income countries, it is not surprising that the chronic diseases are often treated as serial acute episodes and not with a continuum of care (102). The repeated management of the acute phase of chronic diseases may improve to the detriment of the prevalence of the disease resulting in soaring expenses due to the increased burden of disease (102). Interventions targeting chronic diseases are highly dependent on well functioning health systems as they require long term co-ordination and inter-sectoral collaboration across a continuum of care (33).

South Africa is better resourced and has a better health structure than anywhere else in sub-Saharan Africa and it is thus conceivable that impetus can be given to these interventions (53). Feasible interventions such as optimising blood glucose levels, adequate BP control and simple measures such as foot care have been shown to be highly cost-effective (53).



However, the reality is that many South African studies have reported sub-optimal HPT and DM control, infrequent examinations for complications despite their high rate of occurrence and poor supplies of lipid lowering medication in the PHC facilities (53).

When considering individual diseases, the high mortality comparative to the prevalence of DM might well reflect the sub-optimal level of care by health services in this country (53). A Kwa-Zulu Natal study also showed that patients who were informed by clinic staff of their blood glucose levels had better DM knowledge possibly translating to improved self management (50). Patients receiving counselling at the PHC on diet, exercise and their medication had much better knowledge on the disease and its management resulting in better glycaemic control (50).

The situation appears to be similar for HPT control. The HiHi Study showed that only 26% of patients were informed of their BP readings at their last visit and on average, only a single change was made to their HPT medication in the last year despite their control being far from adequate (58). Failure to inform patients of their BP measurements had been shown to be associated with poor compliance and having a negative influence of patient empowerment (58). Problems associated with poor HPT control are still compounded by inadequate medication stocks at PHC level (58). These inherent flaws in the health care system sends a potent message to patients and their families that HPT control is not important (58).

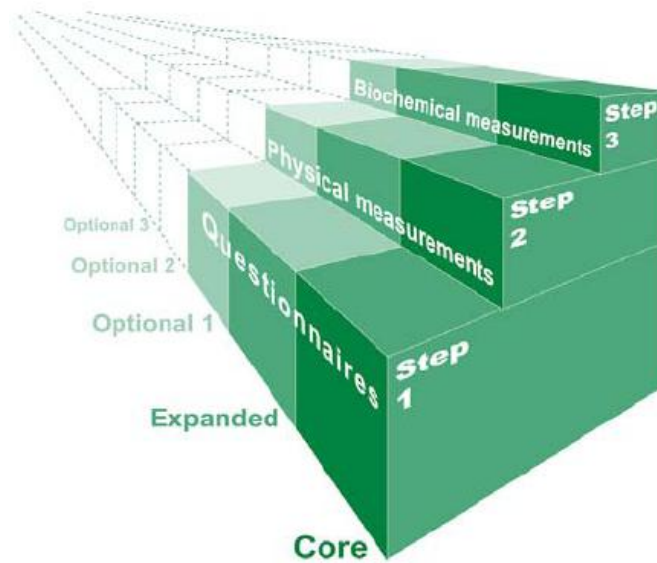
Interestingly, the HiHi Study also showed that patients receiving private health care had better BP control than those attending PHC facilities despite those in private having a higher prevalence of DM and being more obese (103), a finding that reaffirms the 1998 SADHS (104). In many ways, this serves as an indictment on the quality of care of services provided in the public sector.

## **2.7 DEVELOPING NON-COMMUNICABLE DISEASE SURVEILLANCE**

An operational NCD surveillance system, similar to programmes conducted with communicable diseases, is an integral cog in the public health response to NCDs as information generated informs decision-making and priority setting (12).

The basic principle is that countries are able to collect and integrate data at the most basic level, and allow for more sophisticated information systems as resources allow (22). The WHO STEPwise chronic disease risk factor surveillance approach argues that NCD surveillance should be an essential national public health function (12). In Gauteng , the planning for the implementation of this approach began in 2006 (56) with the focus now being shifted to setting up surveillance systems for the continuous monitoring and evaluation of NCDs.

Figure 2.3 describes the sequential approach of STEPS based on a of information gathering (Step 1), acquiring simple anthropometry (Step 2) and only then drawing blood for biochemical assessment (Step 3) (12).



**Figure 2.3: The concept of the STEPwise approach**

By standardising the data collection tools, data collected within a country, and indeed, between countries become comparable (12). This ideal forms the basis of this research. This pilot study concentrates on the core modules (12) of the WHO STEPwise.

Many factors implicated in the development of NCDs are not amenable to change (e.g. genetic). The emphasis of a surveillance programme should be placed on those core risk factors that are amenable to modification (105;106) and can provide measurable successes through intervention. This makes sense considering that inappropriate diet, physical inactivity and concurrent tobacco use together explain about 75% of CVD (107).

## **2.8 SUMMARY OF THE CHAPTER**

The global prevalence of cardio-metabolic diseases is increasing with much of the burden attributable to urbanisation and poor lifestyle choices: physical inactivity and unhealthy diets. Low-income and middle-income countries face increasing cardio-metabolic disease associated morbidity and mortality placing huge demands on the health systems already overrun with the competing needs of infectious diseases and maternal and child health. In South Africa, optimal management of these diseases (particularly DM, HPT and obesity) extends beyond the disease with individual and health system factors impacting considerably of delivery and use of services. Surveillance of NCDs plays an integral part of the national NCD programme. Information generated guides policy development and tailors services to the specific needs of the community.

## **CHAPTER 3**

### **STUDY METHODS AND MATERIALS**

The study methodology was defined by the aim and objectives of the study. The chapter describes the study design, setting and sampling strategy. The data collection, management and measurement are discussed followed by the analysis plan.

#### **3.1 STUDY DESIGN**

A community based cross sectional survey was conducted.

#### **3.2 STUDY SETTING AND SCOPE**

##### **3.2.1 Study setting**

Soweto is the largest township in sub-Saharan Africa with the highest concentration of black Africans on the continent representing the major black ethnic groups in South Africa (34). The study population included the residents of Chiawelo, a suburb in Soweto located in Region D of the Johannesburg Health District (indicated by the arrow in Figure 3.1). Chiawelo is representative of various ethnic (Nguni, SothoSwana, Tshangaan and Venda speaking) and socio-economic groups found in the Johannesburg Health District, thus represents a homogenous black population with heterogeneous ethnicity.



**Figure 3.1: Regional map of Johannesburg indicating Chiawelo**

The Chiawelo CHC which caters for this population ranked second in the Johannesburg Health District with regard to the number of diabetic patients seen according to the District Health Information System (DHIS) data from April 07– March 08. Additionally, the community of Chiawelo was selected given their high numbers of diabetic patients which translates to increased disease prevalence in the area (108).

### **3.2.1 Scope**

The study involved primary data collection.

### **3.3 STUDY POPULATION AND SAMPLING**

#### **3.3.1 Study populations**

The study population comprised residents of the Chiawelo community that had been resident for 5 years or more. Residents aged 18 years or older during the study period were included in the study. Those younger than 18 years of age, had refused blood testing or had dementia or severe psychiatric illness were excluded.

#### **3.3.2 Sample size calculation**

STATCALC was used to calculate the sample size (109). A sample size of 288 was calculated as appropriate for this study using a confidence interval of 95%. The prevalence of cardio-metabolic diseases in South Africa varies between 1 – 30%. In light of this prevalence, a pre-test probability of 25% was used with a worst acceptable rate at 20%.

As the exact prevalence is unclear, the decision was made to over-sample the population as this would additionally increase the generalizability of the study results. The 20% increase on the initial calculated sample size (n=345) still had randomly selected participants using the cluster sampling technique. The final sample size was 337, as nine participants were excluded based on the exclusion criteria described in Section 3.3.1: Study populations.

### **3.3.3 Sampling strategy**

A cluster sampling technique was used for this study. The study sample represented a single stratum with clusters. Second stage sampling of people from the clusters was then performed (multi-stage cluster sampling). The schema for the clustering and selection process was adapted from a previous study conducted in the area (Appendix A: Sampling strategies) (110). Using a map, Chiawelo was divided into 20 clusters, five of which were randomly chosen for the study (Figure 3.2). Seventy subjects were chosen from each of these five clusters.

A point marked X was placed on the map. The research assistants (RA) started at this point X and then selected the first yard on the left side of the street. They then went to every third yard till 70 participants were recruited per each of the five clusters. RAs employed were trained in the above technique. If no one was available at a chosen house or if the occupant refused participation then three yards from that point was calculated to maintain the sampling strategy.





**Figure 3.2: Map of Chiawelo indicating recruitment sites**

### **3.4 STUDY PERIOD**

The study ran from 18 May – 5 June 2009. The chosen study period had 15 full working days and did not coincide with any public or religious holidays. Data was collected between the hours of 8am – 4pm and participants were not recruited on weekends.

## 3.5 DATA COLLECTION

### 3.5.1 Data collection

The principal investigator (PI) and five research assistants (RA) collected the primary data. Once written informed consent was obtained, the questionnaires were completed by the RAs at the participants' home. The time required to complete the questionnaire was no more than 30 minutes. The participants' were then brought to the Changing Diabetes<sup>®</sup> Bus where the anthropometric measurements and blood drawing was done. The Changing Diabetes<sup>®</sup> Bus is a mobile clinic operated by Novo Nordisk in collaboration with the Gauteng Department of Health (Figure 3.3) that provides free screening for DM, HPT and anthropometric measurements (height, weight and waist circumference [WC]).



**Figure 3.3: Participants outside the Changing Diabetes<sup>®</sup> Bus**

### **3.5.2 Measurement tools**

Various documents were developed to facilitate the ethical conduct of the study and data collection (Appendix B: Measurement tools):

Tool 1: Patient information sheet

Tool 2: Patient informed consent

Tool 3: Patient questionnaire

#### **3.5.2.1 Validity of measurement tools**

The tools used in this study were adapted from a 2008 study conducted by the Department of Community Health (108). The patient questionnaire also incorporated the Type 2 diabetes risk assessment form developed by the Finnish Diabetes Association (111).

#### **3.5.2.2 Reliability of measurement tools**

The reliability of the tools was verified through a pilot study (Section 3.8: Pilot Study).

### **3.5.3 Reproducibility of measurements**

Training of the RAs served to reduce the likelihood of intra-operator variability. The RAs selected were fluent in the local languages and conducted structured interviews in participants' home languages. All measurements were conducted consistently by the same individuals (intra-operator variability). Each measurement was taken twice to reduce the likelihood of inter-operator variability.

The Bland-Altman analysis is a methods comparison approach that assesses the agreement between two methods of clinical measurement (112). The mean ( $\bar{D}$ ) differences and the standard deviation (SD) of differences between the two measurements were calculated and the limits of agreement represented by: Limits of agreement =  $\bar{D} + 2SD$ . The inter-operator variability was calculated (113) and is presented in Table 3.1.

**Table 3.1: Inter-operator variability for measurements**

Variable	$\bar{D}$	SD	Limit of agreement	
			Upper	Lower
Waist (cm)	-0.09	0.56	1.03	-1.21
Weight (kg)	0.75	0.46	0.99	-0.84
Height (mm)	0.00	0.00	0.01	-0.01
SBP (mm/Hg)	5.33	15.90	37.13	-26.46
DBP (mm/Hg)	2.66	14.06	30.77	-25.45

Patients with persistently high BP recordings were asked to return the next day to repeat the measurement and verify the initial figures obtained. Appropriately sized sphygmomanometers cuffs were selected according to the size of the patients' arm. Blood drawings were conducted after the BP measurements to avert the stress of the drawing impacting on the BP measurement.

### 3.6 DATA MANAGEMENT AND MEASUREMENT

Data was entered into Microsoft Excel 2003 and transferred to Stata release 10 (114) for statistical analysis. The data were checked for errors by the PI. In addition, 15 questionnaires from each week were randomly selected, and data entry was double-checked in each field.

Data from the questionnaire (Appendix B: Measurement tools) was categorized into two sections, as described below and is followed by a detailed description of the individual variables.

1. *Socio-demographic factors* - examined factors such as the age, educational level and socio-economic status (SES) of the participants. It also addressed the impact of fresh fruit and vegetable consumption and levels of physical activity by measuring energy expenditure.
2. *Clinical factors* – assessed the anthropometric (e.g. height, weight), clinical (e.g. BP, BMI) and biochemical measurements (e.g. HbA<sub>1c</sub> levels) of participants. The section included previously reported histories of DM or HPT.

### **3.6.1 Description of variables**

This section describes how the data was organized to meet the objectives of the study. Table 3.2 describes the variables used in the bivariate and multivariate analyses. The table describes the type of variable, unit of measurement and generation of variables. The variables generated to meet the objectives are then described.

**Table 3.2: Description of variables used in the analyses**

<b>Variable</b>	<b>Type</b>	<b>(Abbreviation) / Description</b>
<b>Socio-demographic</b>		
Gender	Categorical	Male or female
Age (years)	Continuous Non-normal	Calculated from South African ID document and presented as integers. Range: 18-93.
Marital status	Categorical	Six level variable: single, married, widowed, divorced, cohabit, other.
Education	Categorical	Six level variable dichotomised into < Grade 12 and ≥ Grade 12.
Work	Categorical	Eight level variable coded into four levels: unemployed, employed, students, retired.
Socio-economic status	Continuous Non-normal	(SES). Scale derived from Asset Index Score used in a previous South African study assessing chronic diseases and poverty (115). A four point Likert scale was used to score each of the 13 items. (13-items, Cronbach's $\alpha = 0.74$ ).
Fruit and vegetable consumption	Categorical	Combining two variables: fruit consumption/wk, vegetable consumption/wk. Three level variable generated: consumption – never, 1-4 days/wk, 5-7 days/wk.
Alcohol use	Categorical	Self reported alcohol use by participants. Two level variable: yes, no.
Tobacco use	Categorical	Self reported tobacco use by participants. Two level variable: yes, no.
Energy expenditure (kcal)	Continuous Non-normal	Self reported duration of mild, moderate and intense activity per week (116;117). Range: 0 – 61128.
Family history	Categorical	Self reported history of DM and/or HPT in parents, siblings or grandparents. Two level variable: yes, no.
Social grants	Categorical	Three level variable: child, disability, old age.
<b>Clinical</b>		
Waist circumference (cm)	Continuous Normal	(WC). Average of two readings taken using paper tape measures. Range: 58 – 145.
Height (mm)	Continuous Normal	Average of two readings taken using a manual, vertical scale. Range: 115 – 190.
Weight (g)	Continuous Normal	Average of two readings taken using an electronic scale calibrated daily. Range: 45 – 141.
Body mass index (kg/m <sup>2</sup> )	Continuous Non-normal	(BMI). Calculated using the Quetelet Index [BMI = weight (kg) / height(m) <sup>2</sup> ](10). Range: 17 – 55.
Previous history of HPT	Categorical	(Prev. diag. – HPT). Self reported HPT by participants.
Systolic blood pressure (mm/Hg)	Continuous Normal	(SBP). Average of two readings taken using a sphygmomanometer calibrated daily. Range: 93 – 238.
Diastolic blood pressure (mm/Hg)	Continuous Normal	(DBP). Average of two readings taken using a sphygmomanometer calibrated daily. Range: 57 – 144.
Previous history of DM	Categorical	(Prev. diag. – DM). Self reported DM by participants.
Glycated haemoglobin (%)	Continuous Non-normal	(HbA <sub>1c</sub> ). Biochemical measurement of glycaemic control over the preceding 2-3 months (1). Range: 4.4 – 15.3.
Metabolic syndrome	Categorical	(MS). Defined using the IDF criteria (9).

### **3.6.1.1 Socio-demographic variables**

#### **Gender**

The analysis was stratified by gender. This is because the literature points to appreciable differences in chronic disease epidemiology between females and males.

#### **Education**

Education was stratified into those with  $<$  Grade 12 and those with  $\geq$  Grade 12. Grade 12 represents a landmark educational level in the South African education system and was hence used as a cut-off point in this study. Learners who successfully complete Grade 12 have the potential to enter a Bachelors degree programme at a university, and can thus pursue tertiary education (118).

#### **Socio-economic status**

The socio-economic questionnaire was based on a validated questionnaire used in a Soweto study with a similar setting to this study (110). The questionnaire collected information on various topics such as: housing, access to electricity, indoor water source and toilets, material possessions (such as television sets, radio, music system, video machine, fridge, washing machine, microwave, land-line telephone, motor vehicle), the number of residents and household income. An asset indicator score (115) was then calculated using a 4 point Likert scale for each of the 13 variables mentioned. The asset indicator score was then used to divide the subjects into quartiles ranging from lower to higher SES.

### **Energy expenditure**

A questionnaire (long version) designed and tested by the ‘International Consensus Group for the Development of an International Physical Activity Questionnaire’ was used to collect information from the subjects about their physical activities (116;117). The questionnaire had been tested in 12 countries and found to have acceptable measurable properties for monitoring of physical activity among participants (18 to 65 years old) in diverse settings (119). The questionnaire was designed to collect self-reported information on occupational, household and garden/yard work, transportation and leisure-time physical activities during the previous 7 days. Computation of the total scores requires summation of the duration (in minutes) and frequency (days) of walking, moderate-intensity and vigorous-intensity activity. A MET-minute is computed by multiplying the MET score (METS are multiples of the resting metabolic rate) by the duration of minutes, the activity is performed (119).

Kilocalories may then be calculated from MET-minutes using the following equation:

$$\text{Kcal} = \text{MET-min} \times (\text{weight in kg} / 60 \text{ kg})$$

### **Social grants**

Social grants, in particular disability and child support grants and old age pensions, are discussed in this study in lieu of the large percentage of the South African population relying on these remittances as their sole source of income. Close to 15 million South Africans obtain social grants from the government, 10 million of whom are children who receive the child support grant, linked to school attendance (120).



### **3.6.1.2 Clinical variables**

#### **Waist circumference**

It was measured in the horizontal plane at the level of the natural waist (narrowest part of the torso) as seen from the anterior aspect of the body (121).

#### **Height**

Standing height was measured using a fixed stadiometer to the nearest mm.

#### **Weight & body mass index**

Weight (in grams) was measured using the same scale for all subjects and the BMI was calculated from the height and weight. The BMI is a commonly used indicator to assess the nutritional status of adults. It correlates highly with adiposity. Body Mass Index was calculated from the height and weight using following formula (10):  $BMI = \text{Weight in kg} / (\text{Height in m})^2$

Subjects were then classified into following weight categories (defined in: Glossary of terms) (11):

- Under-weight (< 18.5)
- Normal-weight (18.5 to 24.9)
- Over- weight (25 to 29.9) and
- Obese (30 or more).

## **Diabetes mellitus & hypertension**

The primary outcome measure remained the presence of disease. Prevalence rates for HPT and DM were calculated by adding the self-reported cases to the new cases identified by measurement in the study. Self-reporting of HPT and DM was used to interpret disease control parameters and levels of undiagnosed disease in the study population. The information gauged from those reporting *yes* to having the disease were used as a proxy to determine the control of the disease. Similarly, those who reported *no* to having a disease served as a proxy for the numbers of undiagnosed cases of the disease in the study population.

- **Diabetes mellitus**

The analysis of HbA<sub>1c</sub> was conducted through the National Health Laboratory Service (NHLS) laboratories which are accredited by the South African National Accreditation System (SANAS). The accreditation involves assessing the implementation of quality standards as well as the competence of the laboratory to perform designated tests. This ensures that tests performed in these laboratories are comparable to the same test performed anywhere else in the world using the same methodology.

The Cobas Integra analyser (Roche, South Africa) was used for analysis of HbA<sub>1c</sub> specimens. This is a completely automated system that determines HbA<sub>1c</sub> using whole blood samples. The system is designed to immediately flag blood samples unsuitable for analysis (clotted) while internal quality control software programmes monitor precision and accuracy to ensure reliable results.(122)

Those individuals with HbA<sub>1c</sub> results were considered in this analysis. Individuals with outstanding results were excluded. The HbA<sub>1c</sub> levels were categorised according to the American Diabetes Association (ADA) guidelines (defined in: Glossary of terms) (1). The ADA advocates the use of HbA<sub>1c</sub> for the diagnosis and management of DM as it is more convenient than tests requiring fasting samples, has greater pre-analytical stability and is subject to less day-to-day fluctuations during periods of stress and illness (1). The Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA) does not advocate the use of the HbA<sub>1c</sub> for diagnosing DM, mostly because of limited capacity to perform the test in some parts of the public sector, increased costs and because the test may not be useful in the presence certain co-morbidities (e.g. chronic malaria, iron deficiency anaemia) (123;124).

Despite these valid reservations, HbA<sub>1c</sub> was used in this study as it allowed for ease of collection and based on the reasons postulated by the ADA. A bivariate variable was generated using these guidelines and risk factors associated with the development of disease was assessed in those with the disease compared to those without. This variable was used in the prevalence, bivariate and multivariate analyses of the DM.

Table 3.3 describes the levels of DM disease control designated in accordance with the ADA classification using HbA<sub>1c</sub> (1). This was used to classify the disease control in individual participants based on their HbA<sub>1c</sub> results.

**Table 3.3: Level of DM disease control described by HbA<sub>1c</sub> levels**

<b>ADA classification</b>	<b>HbA<sub>1c</sub> level</b>	<b>Level of control</b>
Normal	< 5.7%	Good
Impaired glucose tolerance	5.7 – 6.4%	Sub-optimal
Diabetes mellitus	≥ 6.5%	Poor

- **Hypertension**

The SBP and DBP levels were categorised using The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC7) definition of HPT (defined in: Glossary of terms) (6). The SBP and DBP categories described were combined to generate a bivariate variable of participants with HPT and those without. The risk factors associated with the development of disease was assessed using this generated variable.

Table 3.4 describes the levels of HPT disease control designated in accordance with the JNC7 classification (6). This was used to classify the disease control in individual participants based on their BP recordings at the time of the interview.

**Table 3.4: Level of HPT disease control described by BP readings**

<b>BP classification</b>	<b>SBP (mmHg)</b>	<b>DBP (mmHg)</b>	<b>Level of control</b>
Normal	<120	and < 80	Good
Prehypertension	120 - 139	or 80 - 89	} Sub-optimal
Stage 1 hypertension	140 - 159	or 90 - 99	
Stage 2 hypertension	≥ 160	or ≥ 100	Poor

## **Obesity**

Body weight was categorized according to the WHO Global Database on BMI (11). A bivariate variable was generated comparing obese individuals ( $\text{BMI} \geq 30 \text{ kg/m}^2$ ) with the rest of the study population. This variable was used in the prevalence, bivariate and multivariate analyses of the obesity.

## **Metabolic syndrome**

The primary outcome measure was the diagnosis of MS. This was generated independently by gender to accommodate for the variation in WC criteria between males and females (9). A bivariate variable was generated consolidating the male and female information but differentiating those with the syndrome from those without using the IDF definition of the MS (defined in: Glossary of terms) (9). The IDF criteria were specifically used for this study as the definition tends to result in higher prevalence rates of the syndrome than other definitions (125), provides clear guidelines for identifying at risk populations worldwide (29) and uses specific WC cut-off levels for males and females in different ethnic groups (126). The IDF criteria for the diagnosis of the MS were modified for the purposes of this study. Firstly,  $\text{HbA}_{1c}$  results were used as a surrogate for fasting plasma glucose samples. This was done to facilitate ease of collection and given the financial and human resources constraints of the project. The use of  $\text{HbA}_{1c}$  was deemed to be acceptable in the diagnosis of DM in keeping with the current ADA guidelines (1). Secondly, blood samples collected for lipid profile analysis were incorrectly stored, and were unable to be analysed. Thus, the diagnosis of the MS is

based on the criteria defining waist circumference, HPT and DM using HbA1c as a surrogate.

### 3.7 DATA ANALYSIS

Stata 10 (114) was used to perform bivariate and multivariate regression analysis. Descriptive statistics and multivariate regression was used to answer the study objectives. Statistical tests used in the analysis and their indications are described in Table 3.5. The differences were considered to be statistically significant if  $p < 0.05$ . All analyses accounted for the effects of clustering in the study design.

**Table 3.5: Indications for statistical tests**

<b>Dependent variable</b>	<b>Independent variable (IV)</b>	<b>Distribution</b>	<b>Test</b>	<b>Example</b>
Categorical	1 IV with 2 levels (independent group)		Chi-square test	Education and gender
Continuous	One sample, one variable	Normal	Mean, standard deviation (SD)	WC
		Non-normal	Median, inter quartile range (IQR)	Age
	1 IV with 2 levels (independent groups)	Normal	Student's t-test	WC and gender
		Non-normal	Wilcoxon-Mann Whitney test	BMI and gender
	1 IV with 2 or more levels (independent groups)	Non-normal	Kruskal-Wallis	Age and work
Categorical or continuous	1 IV with 2 or more levels	Normal or non-normal	Multivariate regression	HPT with age and gender

Distribution of the variables was assessed. Data were assessed according to their distribution (as described in Table 3.5). Chi-square testing ( $X^2$ ) was used to test the association between categorical variables in the bivariate analysis.

For the parametric data, Student's t-tests were performed for the analysis of continuous variables and described in terms of means and SD. For the non-parametric data, the Wilcoxon-Mann Whitney test was used for continuous data and results were expressed with medians and inter-quartile ranges (IQR). Kruskal-Wallis testing was performed when several independent groups were being compared to a continuous variable. This provided evidence of a difference between the groups. When a significant value was found, the *kwallis2* command was used to perform post-hoc paired comparison testing to determine which groups were different.

Some variables were dichotomized to simplify the statistical analysis and allow for ease of interpretation and presentation of results. However, it was appreciated that in dichotomizing variables, much information could be lost, so the statistical power to detect a relationship between the variable and outcome is reduced. One of the variables dichotomized was the education level into below Grade 12 and  $\geq$  Grade 12.

All factors found to be significant in the bivariate analysis at p-values  $<0.1$  were considered for the multivariate logistic regression analysis. Stepwise backward multivariate logistic regression was performed and non-significant factors were sequentially removed from the model. Explanatory variables with consistent p-values

<0.05 remained in the model. The multivariate models found to be significant were tested for multicollinearity (this is multiple regression where the predictor variables themselves are highly correlated). As the degree of multicollinearity increases, the regression model estimates of the coefficients become unstable and the standard errors for the coefficients can possibly be inflated. Variance inflation factor (*vif*) specific to survey data was used to test for this.

Various associations between factors purported to predispose to the MS were assessed by Spearman's correlation and the relationship quantified by linear regression. The strength of the associations between various factors was tested by residual analyses. This tested the model for normality, common variance, linearity and assessment of outliers as described below:

- Normality:

The distribution of the data was tested using the residual values (difference between the observed and predicted values in the model) and the studentized residual values (residual value divided by the standard error/standard deviation of the value). If the residual is normally distributed, an increase (or decrease) in one variable would result in a proportional change in the second variable (e.g. a unit increase in BMI would result in a unit increase in the SBP). The Shapiro-Wilk test was used to statistically test the distribution of the data.

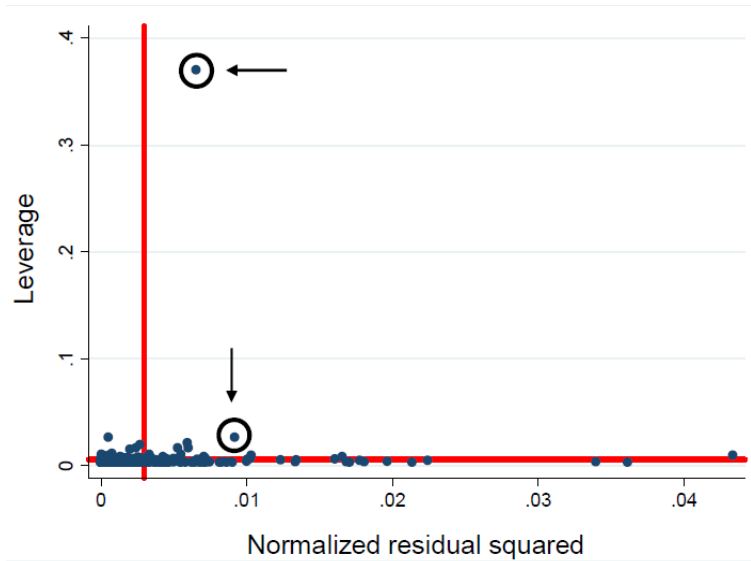


- Common variance and linearity:

Plotting the studentized residuals against the observed values should show a random pattern in the residuals. This assesses linearity and tests for homogeneity of variance. The Breusch-Pagan test was used to test for heteroskedasticity in the linear regression model testing whether the estimated variance of the residuals (e.g. the difference between the observed and predicted values in the SBP from a regression) are dependent on the values of the independent variables (e.g. BMI)

- Outliers & Cook's distance:

These tests were conducted to assess for outliers (variables with large residual values). Values identified as outliers by Cook's Distance (which combine the leverage and residual values) would be deleted and the model reanalyzed. When plotting the leverage against the normalized residuals squared, points above the horizontal line have higher than average leverage; points to the right of the vertical line have larger than average residual values (Figure 3.4) (e.g. the outliers of SBP in the above regression model would be removed and the model reanalyzed). If the same strength of association is yielded irrespective of removal of the outliers, then the outliers had no impact on the initial model and the finding (e.g. a unit increase in BMI would result in a unit increase in the SBP) is generalizable.



**Figure 3.4: Diagram showing values identified by Cook's distance**

### **3.8 PILOT STUDY**

The questionnaire was piloted among ten staff members of the University of the Witwatersrand School of Public Health prior to the commencement of the study. The findings of the pilot study revealed possible limitations to the study, and based on these findings, the tools utilized were modified accordingly.

### **3.9 ETHICS**

The study was submitted to the Committee for Research on Human Subjects of the University of the Witwatersrand, Faculty of Health Sciences, for consideration (Ethics number: M090831) (Appendix C: Permissions). The study was conducted in an ethical manner and information obtained was treated with the strictest of confidence. Written informed consent was obtained from the participants. Disease information pamphlets were distributed to the participants as they awaited clinical assessment. Participants received immediate referral to their local clinic if their clinical assessments warranted this. These documents are presented in Appendix D: Patient documents.

The study was conducted with the permission of the Non-communicable Disease Directorate: Gauteng Department of Health as service delivery project to the province as part of the Registrar training programme. The findings of the study could potentially influence current NCD policy.

### **3.10 SUMMARY OF THE CHAPTER**

This study was a community based cross sectional survey conducted in Chiawelo, Soweto in the Johannesburg Health District. The study describes the baseline descriptive socio-demographics of the community. Multiple regression models were performed to identify factors associated with disease causation. Ethical approval was obtained from the Committee for Research on Human Subjects at the University of the Witwatersrand.

## **CHAPTER 4**

### **RESULTS**

This chapter outlines the main results of the study. Demographic characteristics of the study population are presented, followed by data on the individual cardio-metabolic diseases. Associations between the risk factors are then explored. Finally, the MS is briefly discussed.

#### **4.1 SOCIO-DEMOGRAPHIC FACTORS**

The demographic characteristics of the study population are set out in Table 4.1. The study comprised 337 participants.

**Table 4.1: Demographic characteristics of the study population (n=337)**

Variable		Total		Female		Male		p-value
		n=337	(%)	n=213	(%)	n=124	(%)	
Age <sup>#</sup>		44	(30-56)	45	(32-57)	39	(26-56)	0.033*
Marital status	Single	138	(41)	83	(25)	55	(16)	0.024*
	Married	127	(38)	76	(23)	51	(15)	
	Divorced	19	(6)	14	(4)	5	(1)	
	Widowed	37	(11)	32	(10)	5	(1)	
	Cohabit	15	(4)	8	(2)	7	(2)	
	Other	1	(0)	0	(0)	1	(1)	
Education	< Grade 12	195	(59)	136	(41)	59	(18)	0.004*
	≥ Grade 12	139	(41)	75	(22)	64	(19)	
Work	Unemployed	168	(50)	120	(36)	48	(14)	0.001*
	Employed	88	(26)	42	(12)	46	(14)	
	Student	19	(6)	9	(3)	10	(3)	
	Retired	62	(18)	42	(12)	20	(6)	
SES <sup>#</sup>		44	(30-56)	45	(32-57)	39	(26-56)	0.115
Social grants	Yes	106	(31)	88	(27)	18	(5)	<0.001*

\*statistically significant

<sup>#</sup> median and (IQR)

#### 4.1.1 Gender

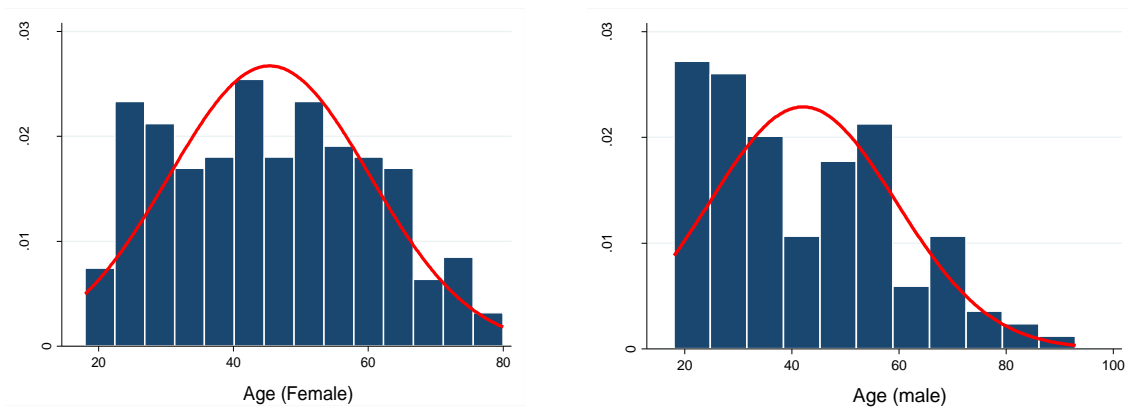
The study comprised 337 participants: 213 female (63%), 124 male (37%).

#### 4.1.2 Race

Most study participants were black (336/337) with only one coloured female.

### 4.1.3 Age

The median age of participants was 44 years (IQR 30-56). The ages ranged from 18 to 93 years. The age distribution was right skewed and platykurtic. Figure 4.1 describes the age distribution of the population sample by gender: the female age distribution was platykurtic while the males appeared bi-modal. While age and gender were significantly associated ( $p=0.033$ ), there was no evidence of collinearity.



**Figure 4.1: Age distributions by gender**

### 4.1.4 Marital status

There was a statistically significant association between marital status and gender (chi square test,  $p=0.024$ ). Specifically, widowed participants were identified as highly likely to be female than male (OR 4.24, 95% CI 1.56-11.55).

#### **4.1.5 Education**

The majority of participants did not complete high school (124/334, 37%). Only 26% (88/334) had matriculated. Three percent (9/334) had no prior education.

#### **4.1.6 Work**

The association between gender and work remained significant when adjusted for age ( $p=0.017$ ). The statistically significant relationship between age and work (Kruskal-Wallis,  $p<0.001$ ) was then further described by gender. Among genders, the following relationships showed significant differences in median ages (kwallis2):

- Unemployed and students (Female:  $p<0.001$ , male:  $p<0.001$ )
- Employed and students (Female:  $p=0.001$ , male:  $p<0.001$ )
- Retired and unemployed (Female:  $p<0.001$ , male:  $p<0.001$ )
- Retired and employed (Female:  $p<0.001$ , male:  $p<0.001$ )
- Retired and students (Female:  $p<0.001$ , male:  $p<0.001$ )

There were no significant differences in the ages between those employed and those unemployed.

#### 4.1.7 Social grants

Females were mostly dependant on the child support grants (47/88, 53%) and the median age of the women accessing these grants was 32 years (IQR 29-40). Most males accessing grants accessed the old age pensions (16/18, 89%). This was at the median age of 69 years in males (IQR 68-78) and 66 years (IQR 64-72) in females.

The association between social grants and gender was significant when adjusted for age ( $p < 0.001$ ). Among the genders, the following relationships had significant differences in median ages (kwallis2):

- Child support and old age (Female:  $p < 0.001$ )
- Child support and disability (Male:  $p = 0.012$ )



## 4.2 CARDIO-METABOLIC DISEASES AND ASSOCIATED RISK FACTORS

In this section, the cardio-metabolic diseases and associated risk factors individual risk factors in the study sample will be described.

### 4.2.1 CARDIOVASCULAR RISK FACTOR PROFILE

Table 4.2 shows the CVD risk factor profile of the study population stratified by gender. Thirty eight percent (38%) of females had reported a previous history of HPT with 24% in males (30/124) (chi square test,  $p=0.031$ ). Females had a markedly elevated BMI level compared with males (Mann-Whitney,  $p<0.001$ ). Though not statistically significant, both genders had elevated mean systolic BP (SBP) and diastolic BP (DBP). There was a significant difference between HbA<sub>1c</sub> levels in males and females ( $p<0.001$ ) and both these values were within normal limits.

**Table 4.2: Cardiovascular risk factor profile (n=337)**

Risk factors	Female		Male		p-value
	n = 213	(%)	n = 124	(%)	
<b>Socio-demographic</b>					
Age #	45	(32-57)	39	(26-56)	0.032*
Uses tobacco	34	(10)	18	(5)	0.724
<b>Clinical</b>					
BMI #	31	(27-36)	25	(21-28)	<0.001*
Prev. diag. - HPT	81	(24)	30	(9)	0.031*
SBP ##	143	(24)	148	(21)	0.070
DBP ##	89	(14)	91	(15)	0.333
Prev. diag. - DM	20	(6)	17	(5)	0.439
HbA <sub>1c</sub> #	5.5	(5.2-5.9)	5.3	(5.1-5.6)	<0.001*

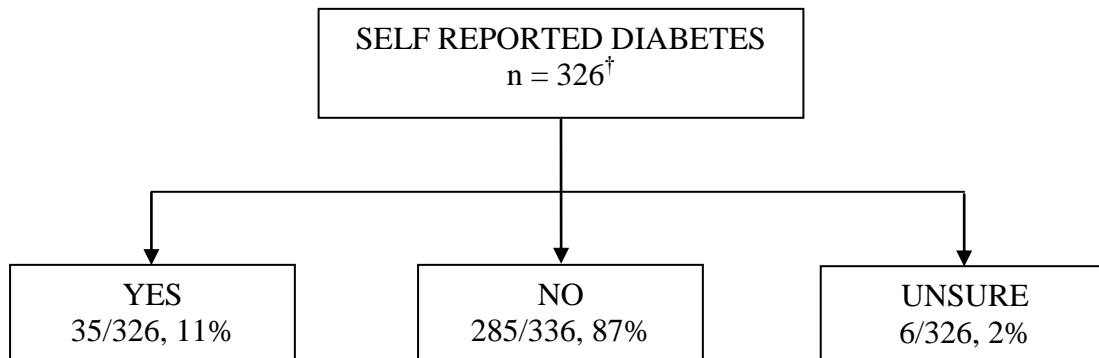
\* statistically significant

## mean and (SD)

# median and (IQR)

#### 4.2.2 DIABETES MELLITUS

Figure 4.2 describes the self reported prevalence of DM. Those participants without HbA<sub>1c</sub> results were excluded (11/337, 3%). From the remaining participants, 35 (11%) reported *yes*, 285 (87%) reported *no* and 6 (2%) were *unsure*.



<sup>†</sup>11 HbA<sub>1c</sub> blood samples were outstanding and were excluded

**Figure 4.2: Self reported DM**

The findings in Figure 4.2 are elaborated on in Table 4.3 according to the HbA<sub>1c</sub> measurements and then stratified by gender. The self reported rates of DM in the study population (35/326, 11%) were 9% in females and 14% in males.

**Table 4.3: Reporting of DM by gender (n=326)**

		HbA <sub>1c</sub> <sup>††</sup> (%)	Total n=326 (%)	Female n=203 (%)	Male n=123 (%)	p-value
<b>Reported yes</b> ( <i>control</i> ) n = 35 <sup>†</sup>	Normal	< 5.7	8 (2)	3 (9)	5 (14)	0.659
	IGT	5.7 < 6.4	4 (1)	2 (6)	2 (6)	
	DM	≥ 6.4	23 (7)	13 (36)	10 (29)	
	<b>Total</b>			<b>18 (51)</b>	<b>17 (49)</b>	
<b>Reported no</b> ( <i>undiagnosed</i> ) n = 285 <sup>†</sup>	Normal	< 5.7	221 (68)	130 (46)	91 (32)	0.003*
	IGT	5.7 < 6.4	52 (16)	44 (15)	8 (3)	
	DM	≥ 6.4	12 (4)	8 (3)	4 (1)	
	<b>Total</b>			<b>182 (64)</b>	<b>103 (36)</b>	
<b>Reported don't know</b> n = 6 <sup>†</sup>	Normal	< 5.7	6 (2)	3 (50)	3 (50)	
	<b>Total</b>			<b>3 (50)</b>	<b>3 (50)</b>	

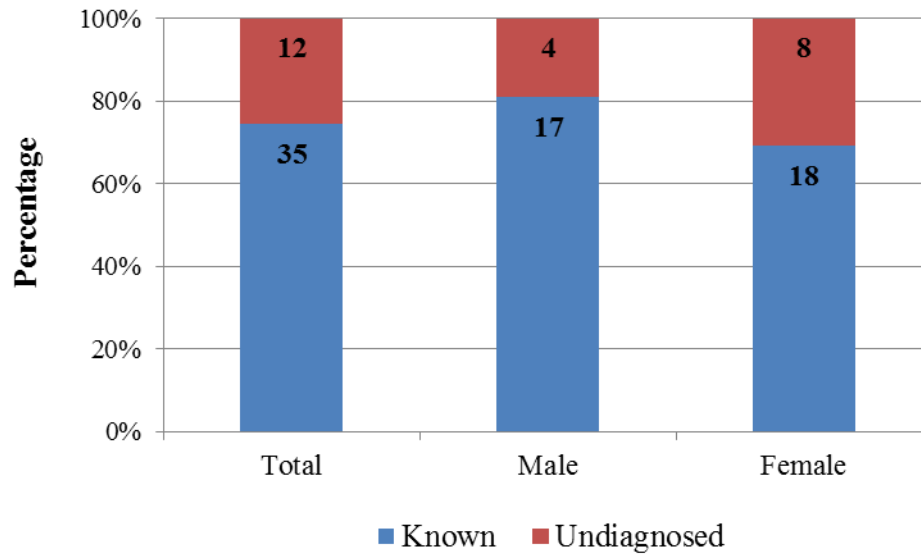
<sup>††</sup> Classification of DM as per the ADA (1)

<sup>†</sup> 11 HbA<sub>1c</sub> blood samples were outstanding and were excluded

\* statistically significant

#### 4.2.2.1 Prevalence of DM

The prevalence of DM among the study participants included those previously diagnosed and individuals identified with high HbA<sub>1c</sub> among those who reported not having DM (Table 4.3). The prevalence was 14% (47/326) with similar distributions amongst males and females (Figure 4.3).



**Figure 4.3: Prevalence of DM by gender**

The statistically significant relationship between age and categories of DM (Kruskal-Wallis,  $p < 0.001$ ) was further described by gender. Normal HbA<sub>1c</sub> levels had significantly lower median ages from (kwallis2):

- IGT (Female:  $p < 0.001$ , male:  $p = 0.004$ )
- DM (Female:  $p < 0.001$ , male:  $p < 0.001$ )

The prevalence estimates for DM in this study are presented in Table 4.4. The prevalence estimates for DM remained significant when adjusted for gender ( $p < 0.010$ ), age ( $< 0.001$ ) and gender and age together ( $p < 0.001$ ). Table 4.4 describes the median ages per category of HbA<sub>1c</sub> classification, while Figure 4.4 graphically represents the prevalence of DM by age categories.

**Table 4.4: Prevalence of DM by age and gender (n=326)**

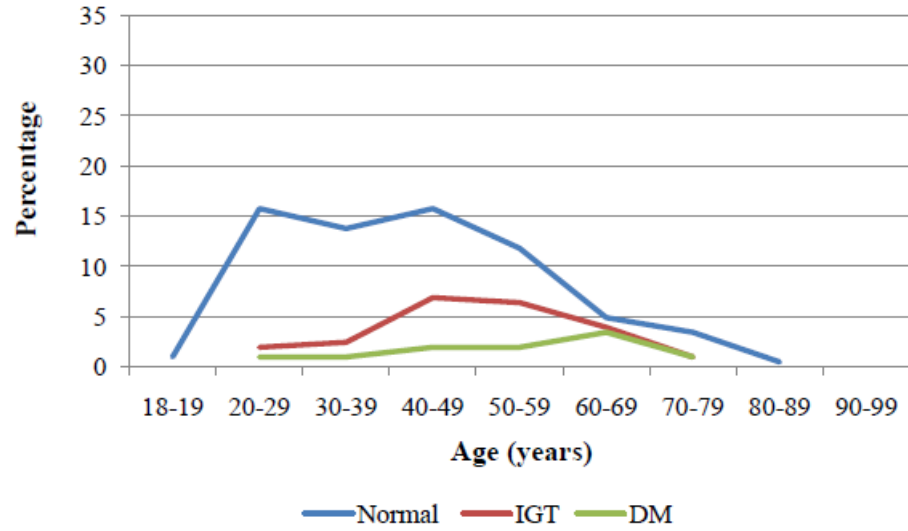
		<b>Normal</b>	<b>IGT</b>	<b>DM</b>
<b>Females</b> (n=203)	Prevalence <sup>+</sup>	136 (67)	46 (23)	21 (10)
	Age <sup>#</sup>	42 (30-53)	50 (43-59)	55 (43-64)
<b>Males</b> (n=123)	Prevalence <sup>+</sup>	99 (81)	10 (8)	14 (11)
	Age <sup>#</sup>	35 (25-51)	55 (52-61)	57 (43-68)
<b>Total</b> (n=326)	Prevalence <sup>+</sup>	235 (72)	56 (17)	35 (11)
	Age <sup>#</sup>	39 (27-52)	52 (44-59)	57 (43-66)

<sup>+</sup> number and (percentage)

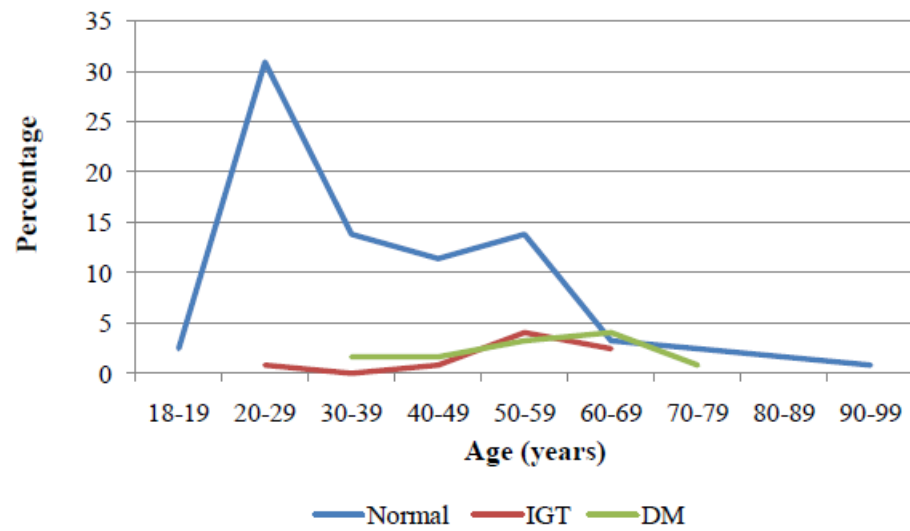
<sup>#</sup> median and (IQR)

Most participants in this study had normal glycaemic control (235/326, 72%). The prevalence of IGT among females was high (23%) compared to males (8%), with females presenting at a younger median age than males. The prevalence of DM was similar in males and females, with females presenting at a younger median age. Among females, both impaired glucose tolerance (IGT) and DM were present in participants as young as 20-29 years [Figure 4.4(a)]. While males presented at an older age with DM, IGT was found at a younger age [Figure 4.4(b)].

**a. Female**



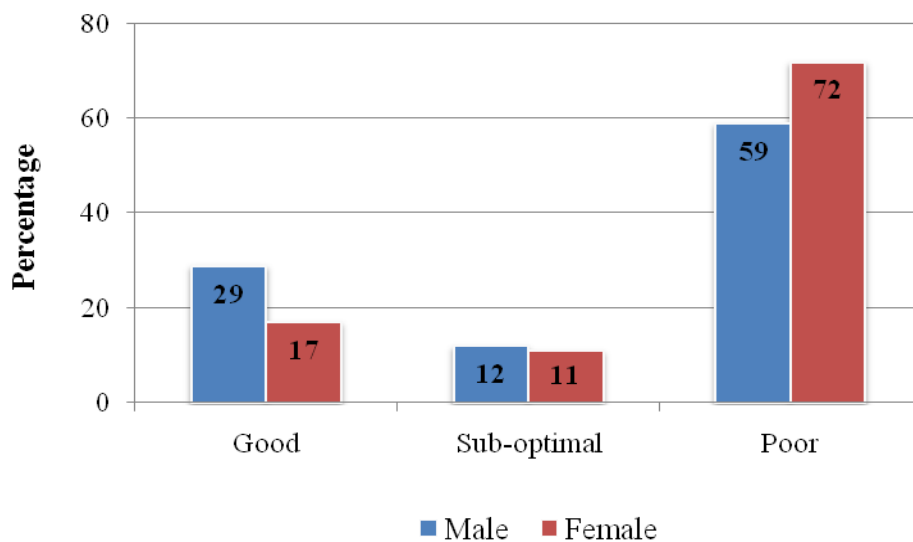
**b. Male**



**Figure 4.4: Prevalence of DM by age and gender**

#### 4.2.2.2 Control of DM

Glycaemic control among diabetics was classified using the ADA criteria for the diagnosis of DM (1) as a proxy for control (as described in Chapter 3: Table 3.3). Figure 4.5 describes the glycaemic control among those who reported having DM (35/326, 11%). Few recorded optimal glycaemic control (8/35, 23%). The majority were poorly controlled (23/35, 66%). Those diagnosed with DM have been shown to have received education on diet, exercise and diabetic care (chi square test,  $p < 0.001$ ). This has, however, not translated to improved health practices with few exercising (Kruskal-Wallis,  $p = 0.183$ ) and poor intake of fresh food and vegetables (chi square test,  $p = 0.447$ ) among those with DM.



**Figure 4.5: Levels of glycaemic control among diagnosed diabetics**

#### **4.2.2.3 Prevalence of pre-diabetes**

Those participants who reported not having DM (285/326, 87%) (Table 4.3) comprised a high number that would be classified as 'pre-diabetic'. These individuals have IGT and are likely to develop DM as well as CVD (1). Fifty two (52/285, 18%) participants are at risk of developing DM, with females being predominantly at risk (44/182, 24%). Both females and males presented with IGT from as young as 20-29 years of age (Figure 4.4).

#### **4.2.2.4 Factors associated with DM**

Table 4.5 describes the bivariate and multivariate factors associated with DM. The bivariate analysis found that DM was associated with increasing age and WC. There was a significant difference in the median ages of those with DM (57 years [IQR 43-66]) and those without (43 years [IQR 28-55]) (Mann-Whitney,  $p < 0.001$ ). Similar differences were recognised in WC where those with disease had markedly larger WC ( $105 \pm 13$  cm) than those without disease ( $97 \pm 17$  cm) (Student's t-Test,  $p = 0.003$ ). Higher educational attainment was protective against disease (OR 0.38, 95% CI 0.17-0.88). Further categorization of work found that those retired were highly associated with having DM (OR 3.39, 95% CI 1.54-7.47).

Increasing age remained significant, with those with a previous diagnosis of HPT also having higher odds of having DM (aOR 2.23, 95% CI 1.19-4.19). There was no evidence of multicollinearity in the multivariate model.



**Table 4.5: Factors associated with DM on bivariate and multivariate regression**

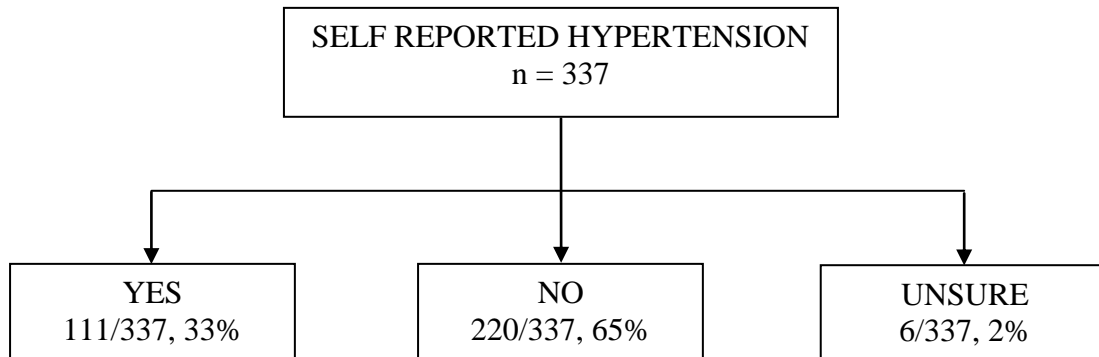
(n=326)

	Bivariate			Multivariate (p<0.001)		
	Odds ratio	95% CI	p-value	Adjusted Odds Ratio	95% CI	p-value
<b>Socio-demographic</b>						
Gender	1.11	0.54 – 2.29	0.770			
Age	1.05	1.02 – 1.07	<0.001*	1.03	1.01-1.06	0.012*
Marital status	1.29	1.04 – 1.61	0.022*			
Education	0.38	0.17 – 0.88	0.024*			
Work	1.50	1.10 – 2.04	0.011*			
SES	0.62	0.34 – 1.14	0.123			
Fruit & vegetable consumption	1.28	0.64 – 2.57	0.477			
Alcohol use	1.16	0.57 – 2.36	0.487			
Tobacco use	0.87	0.32 – 2.36	0.777			
Energy expenditure	0.99	0.99 – 1.00	0.744			
Family history	0.77	0.40 – 1.49	0.439			
<b>Clinical</b>						
WC	1.03	1.01 – 1.05	<0.001*	1.02	1.00-1.04	0.044*
Weight	1.02	1.00 – 1.03	0.048			
BMI	1.01	0.99 – 1.04	0.283			
Prev. diag. - HPT	3.32	1.84 – 5.99	<0.001*	2.23	1.19-4.19	0.013*
SBP	1.01	1.00 – 1.02	0.048			
DBP	1.00	0.98 – 1.02	0.911			

\* statistically significant

### 4.2.3 HYPERTENSION

Figure 4.6 describes the self reported prevalence of HPT. From the 337 participants, 111 (33%) reported *yes*, 220 (65%) reported *no* and 6 (2%) were *unsure*.



**Figure 4.6: Self reported HPT**

The findings in Figure 4.6 are elaborated on in Table 4.6 according to the combined SBP and DBP measurements. The self reported rates of HPT in the study population (111/337, 33%) were 38% in females and 24% in males.

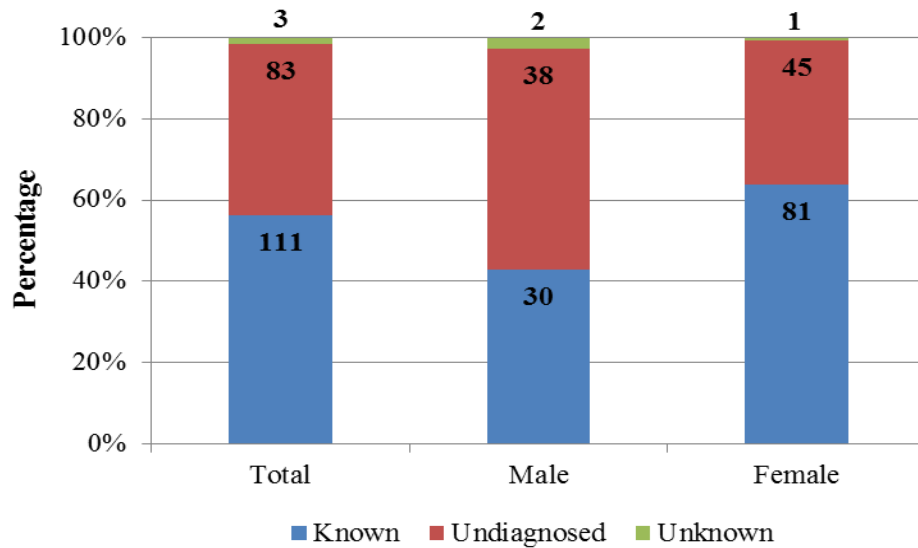
**Table 4.6: Reporting of HPT by gender (n=337)**

		<b>Total</b>		<b>Female</b>		<b>Male</b>		<b>p-value</b>
		<b>n=337</b>	<b>(%)</b>	<b>n=213</b>	<b>(%)</b>	<b>n=124</b>	<b>(%)</b>	
<b>Reported yes</b>								0.411
<i>(control)</i> n = 111	Normal	6	(2)	6	(6)	0	(0)	
	Pre-HPT	37	(11)	28	(25)	9	(8)	
	<b>Stage 1</b>	<b>39</b>	<b>(12)</b>	<b>27</b>	<b>(24)</b>	<b>12</b>	<b>(11)</b>	
	<b>Stage 2</b>	<b>29</b>	<b>(9)</b>	<b>20</b>	<b>(18)</b>	<b>9</b>	<b>(8)</b>	
	<b>Total</b>	<b>111</b>	<b>(34)</b>	<b>81</b>	<b>(73)</b>	<b>30</b>	<b>(27)</b>	
<b>Reported no</b>								0.026*
<i>(undiagnosed)</i> n = 220	Normal	25	(7)	20	(9)	5	(2)	
	Pre-HPT	112	(33)	64	(29)	48	(22)	
	<b>Stage 1</b>	<b>57</b>	<b>(17)</b>	<b>35</b>	<b>(16)</b>	<b>22</b>	<b>(10)</b>	
	<b>Stage 2</b>	<b>26</b>	<b>(8)</b>	<b>10</b>	<b>(5)</b>	<b>16</b>	<b>(7)</b>	
	<b>Total</b>	<b>220</b>	<b>(65)</b>	<b>129</b>	<b>(59)</b>	<b>91</b>	<b>(41)</b>	
<b>Reported don't know</b>								0.606
n = 6	Normal	1	(0)	1	(17)	0	(0)	
	Pre-HPT	2	(0.5)	1	(17)	1	(17)	
	<b>Stage 1</b>	<b>2</b>	<b>(0.5)</b>	<b>1</b>	<b>(17)</b>	<b>1</b>	<b>(17)</b>	
	<b>Stage 2</b>	<b>1</b>	<b>(0)</b>	<b>0</b>	<b>(0)</b>	<b>1</b>	<b>(17)</b>	
	<b>Total</b>	<b>6</b>	<b>(1)</b>	<b>3</b>	<b>(50)</b>	<b>3</b>	<b>(50)</b>	

\* statistically significant

#### 4.2.3.1 Prevalence of HPT

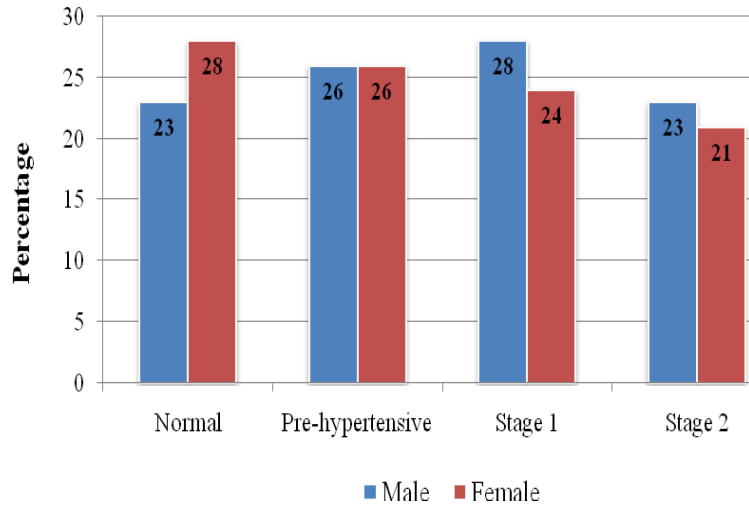
The calculated prevalence of HPT among the study participants included those previously diagnosed and newly identified individuals with elevated BP measurements among those who reported not having HPT and among those who were unsure about the diagnosis (Table 4.6). Figure 4.7 demonstrates the prevalence at 58% (197/337) with similar distribution in females (60%) and males (56%).



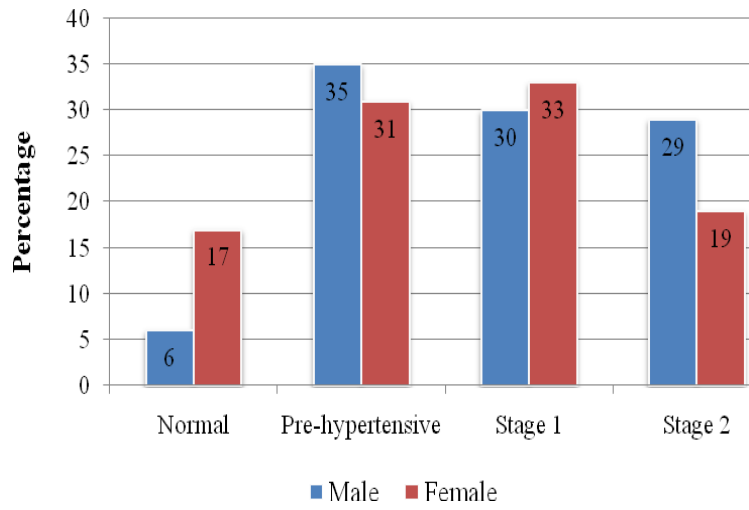
**Figure 4.7: Prevalence of HPT**

Figure 4.8 classifies the prevalence of HPT by (a) DBP, (b) SBP and then a combination of the two (c). Systolic blood pressure and DBP were found to be correlated ( $r=0.769$ ,  $p<0.001$ ). There are no significant differences between males and females when comparing the means of SBP ( $p=0.070$ ) or DBP ( $p=0.333$ ). Only six percent of the female population recorded normal SBP, with 35% identified as prehypertensive. Combining the data yielded approximately 10% of participants with normal BP with the majority in both genders being prehypertensive [Figure 4.8(c)].

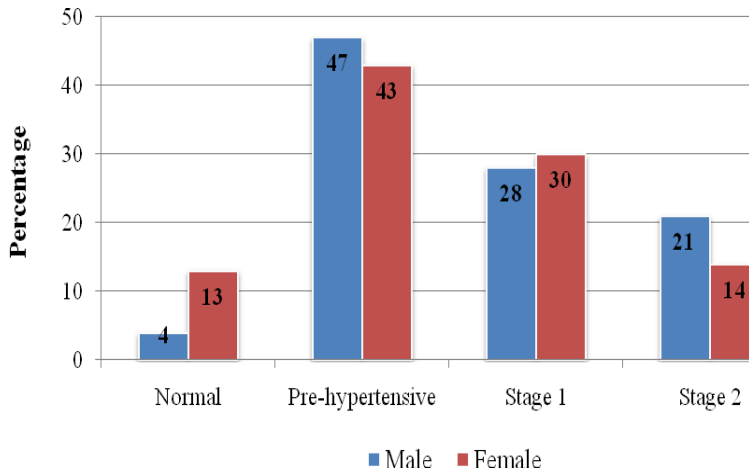
**a. Diastolic blood pressure**



**b. Systolic blood pressure**



**c. Combined blood pressure measurements**



**Figure 4.8: Prevalence of HPT by category and gender**

The statistically significant relationship between age and classes of HPT (Kruskal-Wallis,  $p < 0.001$ ) was then further described by gender. Among the genders, the following relationships showed significant differences in median ages (kwallis2):

- normotensive and Stage 1 (Female:  $p = 0.002$ )
- normotensive and Stage 2 (Female:  $p < 0.001$ )
- prehypertension and Stage 2 (Female:  $p < 0.001$ , male:  $p = 0.002$ )

The prevalence of HPT is described Table 4.7. The prevalence estimates for HPT remained significant when adjusted for age ( $p = 0.002$ ), gender ( $p = 0.014$ ) and age and gender together ( $p = 0.002$ ). Table 4.7 describes the median ages in each category of HPT by gender while Figure 4.9 graphically represents the prevalence of disease by age categories.

**Table 4.7: Prevalence of HPT by age and gender (n=337)**

		<b>Normal</b>	<b>Prehypertension</b>	<b>Stage 1</b>	<b>Stage 2</b>
<b>Females</b> (n=213)	Prevalence <sup>+</sup>	27 (12)	93 (44)	63 (30)	30 (14)
	Age <sup>#</sup>	33 (26-47)	43 (30-57)	46 (35-55)	55 (47-64)
<b>Males</b> (n=124)	Prevalence <sup>+</sup>	5 (4)	58 (47)	35 (28)	26 (21)
	Age <sup>#</sup>	46 (23-51)	33 (25-52)	36 (27-55)	53 (36-61)
<b>Total</b> (n=337)	Prevalence <sup>+</sup>	32 (9)	151 (45)	98 (29)	56 (17)
	Age <sup>#</sup>	33 (26-55)	40 (27-56)	45 (32-55)	55 (43-63)

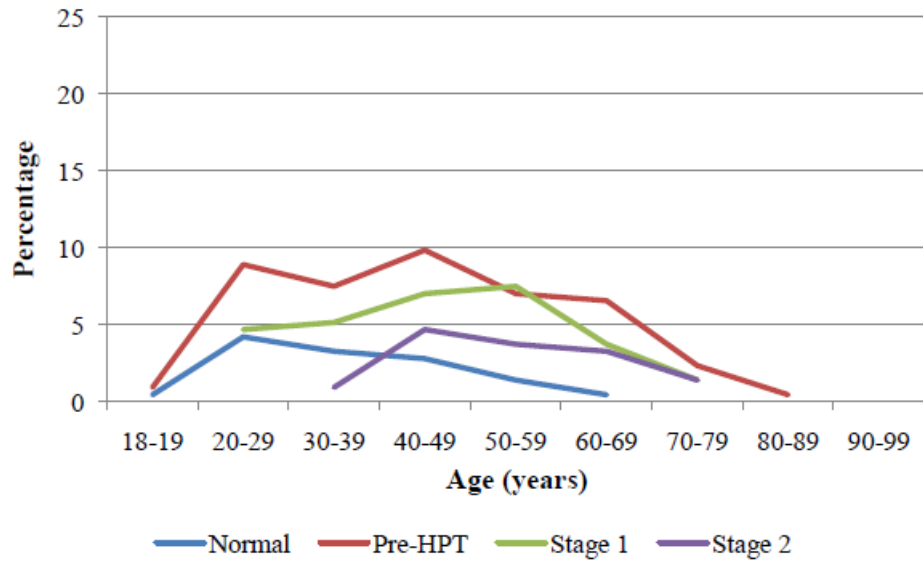
<sup>+</sup> number and (percentage)

<sup>#</sup> median and (IQR)

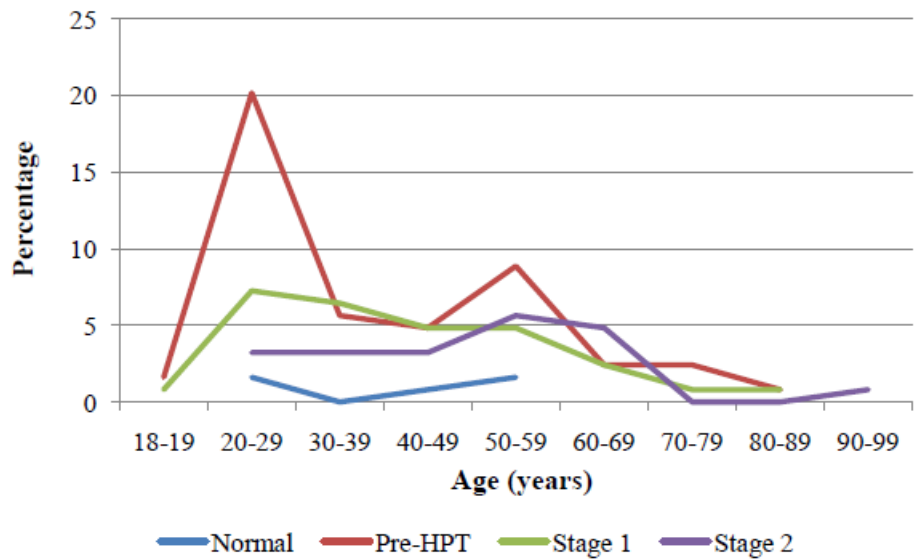
The prevalence of prehypertension in both males and females are high, with males (33 years [IQR 25-52]) presenting at a considerably younger median age than females (43 years [IQR 30-57]). Males presented with disease at earlier median ages for all categories of disease.

Figure 4.9 represents the prevalence of HPT by age and gender. Most participants were classed as prehypertensive which presented as early as 18-19 years of age. Females presented at an earlier age with Stage 1 disease (20-29 years) but later with Stage 2 disease [Figure 4.9(a)]. Among males, Stage 1 disease presented around the same age as in females, but Stage 2 diseases presented earlier than in females [Figure 4.9(b)].

**a. Female**



**b. Male**



**Figure 4.9: Prevalence of HPT by age and gender**

#### **4.2.3.2 Prehypertension**

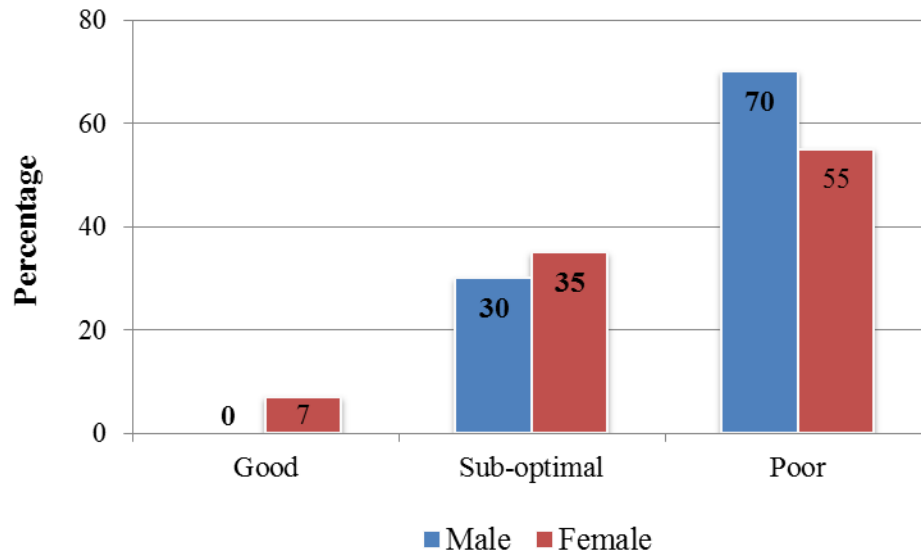
There were a high number (112/220, 51%) of ‘prehypertensive’ candidates among those participants who reported not having HPT (Table 4.6). Prehypertension is not a disease category but rather identifies a group at high risk of developing HPT. The combined data table [Figure 4.8(c)] identifies 150 participants (45%) at risk of developing HPT with males being predominantly at risk (58/124, 47%).

#### **4.2.3.3 HPT control**

Blood pressure control among hypertensives was classified using the JNC7 criteria for the diagnosis of HPT (6) as a proxy for control (as described in Chapter 3: Table 3.4).

Few participants reporting HPT recorded optimal hypertensive control (6/111, 5%). Figure 4.10 describes the majority as being poorly controlled (68/111, 61%). Those diagnosed with HPT have been shown to have received education on diet and exercise (chi square test,  $p < 0.001$ ). This has not, however, translated to improved health practices with few exercising (Kruskal-Wallis,  $p = 0.168$ ) and poor intake of fresh food and vegetables (chi square test,  $p = 0.429$ ).





**Figure 4.10: Levels of control among diagnosed hypertensives**

#### 4.2.3.4 Factors associated with HPT

Table 4.8 describes the bivariate and multivariate factors associated with HPT. The bivariate analysis found that increasing age, WC, weight, BMI and HbA1c levels is associated with having HPT. There was a significant difference in the median ages of those with HPT (48 years [IQR 35-58]) and those without (40 years [IQR 27-54]) (Mann-Whitney,  $p < 0.001$ ). Similar differences were recognised in WC where those with disease had markedly larger WC ( $101 \pm 15$  cm) than those without disease ( $96 \pm 17$  cm) (Student's t-Test,  $p = 0.003$ ). Correspondingly, higher BMI levels were associated with HPT ( $30$  kg/m<sup>2</sup> [IQR 26-35] vs.  $27$  kg/m<sup>2</sup> [IQR 23-33]) (Mann-Whitney,  $p = 0.003$ ). HbA1c levels were also slightly higher in those with disease ( $5.5\%$  [IQR 5.2-5.9]) than those without ( $5.4\%$  [IQR 5.1-5.7]) (Mann-Whitney,  $p = 0.036$ ). Being single, married or widowed, but not divorced was associated with developing disease. Higher educational attainment was protective against having HPT (OR 0.55, 95% CI 0.35-0.86).

Factors identified on multivariate analysis account for 4% of the variation in HPT. Age remained significant, with increasing weight (aOR 1.02, 95% CI 1.00-1.03) also being associated with disease. There was no evidence of multicollinearity in the multivariate model.

**Table 4.8: Factors associated with HPT on bivariate and multivariate regression (n=337)**

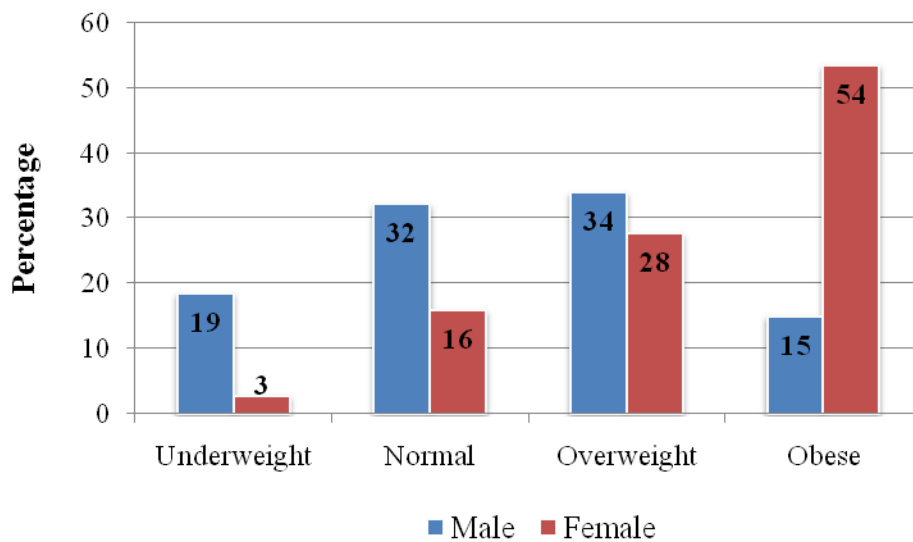
	Bivariate			Multivariate (p<0.001)		
	Odds ratio	95% CI	p-value	Adjusted Odds Ratio	95% CI	p-value
<b>Socio-demographic</b>						
Gender	1.25	0.80 – 1.95	0.326			
Age	1.03	1.01 – 1.04	<0.001*	1.02	1.01 – 1.04	0.002*
Marital status	1.40	1.13 – 1.73	0.002*			
Education	0.55	0.35 – 0.86	0.009*			
Work	1.02	0.85 – 1.24	0.816			
SES	0.91	0.60 – 1.38	0.378			
Fruit & vegetable consumption	1.14	0.75 – 1.74	0.528			
Alcohol use	1.22	0.78 – 1.90	0.374			
Tobacco use	1.34	0.74 – 2.44	0.329			
Energy expenditure	1.00	0.99 – 1.00	0.319			
Family history	1.06	0.70 – 1.61	0.776			
<b>Clinical</b>						
WC	1.02	1.01 – 1.04	0.003*			
Weight	1.02	1.01 – 1.03	0.004	1.02	1.00 – 1.03	0.015*
BMI	1.05	1.01 – 1.08	0.004*			
Prev. diag. - DM	0.98	0.58 – 1.65	0.935			
HbA <sub>1c</sub>	1.25	1.02 – 1.53	0.033*			

\* statistically significant

## 4.2.4 OBESITY

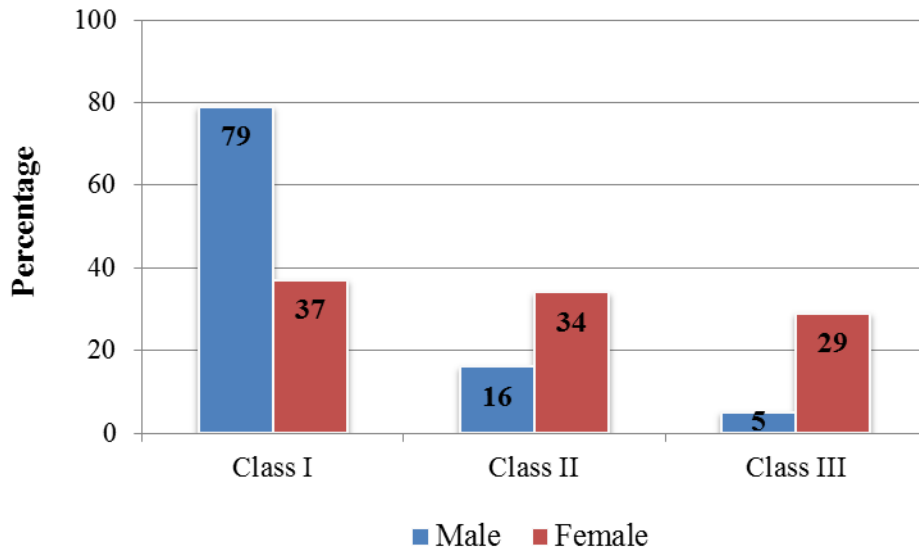
### 4.2.4.1 Prevalence of obesity

Forty two males (34%) and 59 females (28%) are classified as overweight ( $BMI \geq 25$   $kg/m^2$   $<30$   $kg/m^2$ ) in this study population (Figure 4.11). The obesity levels ( $BMI \geq 30$   $kg/m^2$ ) are marked (133/337, 39%). Particularly concerning is the level of obesity among females reaching 54% (114/213) described in Figure 4.12.



**Figure 4.11: Prevalence of obesity by gender (n=337)**

Figure 4.12 categorizes the obesity among 114 female participants and the 19 male participants with the most being Class I disease (57/133, 43%). The relationship between obesity and gender was statistically significant (chi-square test,  $p < 0.001$ ).



**Figure 4.12: Categories of obesity by gender**

The association between classes of BMI and gender remained significant when adjusted for age ( $p=0.003$ ). The statistically significant relationship between age and classes of BMI (Kruskal-Wallis,  $p<0.001$ ) was then further described by gender. Among the genders, the following relationships showed significant differences in median ages (kwallis2):

- Underweight and obese (Male:  $p<0.001$ )
- Normal and obese (Male:  $p=0.003$ , female:  $p=0.003$ ,)
- Underweight and overweight (Male:  $p<0.001$ )

The prevalence of overweight and obesity in this study is described in Table 4.9. The prevalence of categories of BMI remained significant when adjusted for age ( $p < 0.001$ ), gender ( $p < 0.001$ ) and age and gender together ( $p < 0.001$ ). Table 4.9 explains the median age per category of BMI while Figure 4.13 graphically represents the BMI by age categories.

**Table 4.9: Prevalence of categories of BMI by age and gender (n=337)**

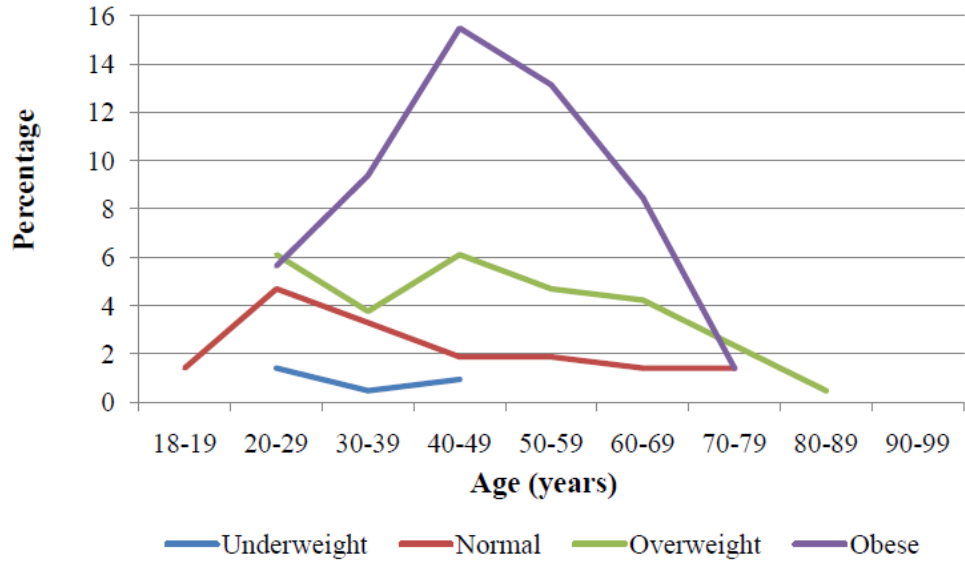
		<b>Underweight</b>	<b>Normal</b>	<b>Overweight</b>	<b>Obese</b>
<b>Females</b> (n=213)	Prevalence <sup>+</sup>	6 (3)	34 (16)	59 (28)	114 (54)
	Age <sup>#</sup>	31 (25-41)	34 (24-55)	47 (31-60)	48 (38-57)
<b>Males</b> (n=124)	Prevalence <sup>+</sup>	23 (19)	40 (32)	42 (34)	19 (15)
	Age <sup>#</sup>	25 (22-40)	32 (25-49)	49 (32-58)	52 (43-56)
<b>Total</b> (n=337)	Prevalence <sup>+</sup>	29 (9)	74 (22)	101 (30)	133 (39)
	Age <sup>#</sup>	26 (23-40)	33 (24-51)	48 (32-59)	48 (39-57)

<sup>+</sup> number and (percentage)

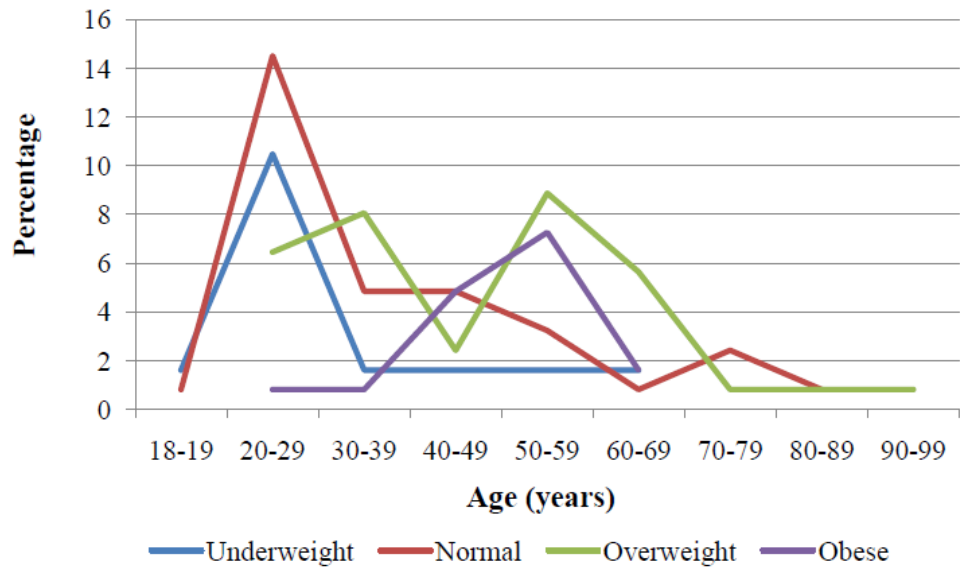
<sup>#</sup> median and (IQR)

Obesity was marked among females with 34% of males being classified as overweight. Figure 4.13 graphically represents the prevalence of obesity by gender and age group. The levels of obesity among females were pronounced with females being overweight and obese from as young as 20-29 years of age [Figure 4.13(a)]. Males presented at the same age as females but most were overweight rather than obese aged between 20-29 years of age [Figure 4.13(b)].

**a. Female**



**b. Male**



**Figure 4.13: Prevalence of obesity by age and gender**

#### 4.2.4.2 Factors associated with obesity

Table 4.10 describes the bivariate and multivariate factors associated with obesity. The bivariate analysis found increasing age, WC, SBP and DBP to be associated with obesity. Obesity was significantly associated with the female gender (OR 6.36, 95% CI 3.63-11.14). Higher educational attainment was protective against being obese (OR 0.30, 95% CI 0.19-0.49). Further categorisation of work found that being retired was protective against being obese. Being married, widowed or divorced is also associated with obesity. Obesity is also associated with a previous diagnosis of HPT.

There was a significant difference in the median ages of those obese (48 years [IQR 39-57]) than those who were not (38 years [IQR 26-56]) (Kruskal-Wallis,  $p < 0.001$ ). Similar differences were recognised in WC where obese individuals had markedly larger WC ( $111 \pm 13$  cm) than those without disease ( $90 \pm 12$  cm) (Student's t-Test,  $p < 0.001$ ). Obese individuals had higher SBP ( $148 \pm 25$  mmHg vs.  $142 \pm 22$  mmHg) (Student's t-Test,  $p = 0.036$ ) and DBP ( $92 \pm 15$  mmHg vs.  $88 \pm 14$  mmHg) (Student's t-Test,  $p = 0.004$ ).

Factors identified on multivariate analysis accounted for 52% of the variation in obesity. Female gender remained markedly significant (aOR 7.58, 95% CI 3.46-16.61) together with WC (aOR 1.19, 95% CI 1.13-1.25) and education levels (aOR 0.42, 95% CI 0.21-0.86). There was no evidence of multicollinearity in the multivariate model.

**Table 4.10: Factors associated with obesity on bivariate and multivariate regression**

(n=337)

	Bivariate			Multivariate (p<0.001)		
	Odds ratio	95% CI	p-value	Adjusted Odds Ratio	95% CI	p-value
<b>Socio-demographic</b>						
Gender	6.36	3.63 – 11.14	<0.001*	7.58	3.46 – 16.61	<0.001*
Age	1.02	1.01 – 1.04	0.001*			
Marital status	1.33	1.08 – 1.63	0.007*			
Education	0.30	0.19 – 0.49	<0.001*	0.42	0.21 – 0.86	0.018*
Work	0.80	0.65 – 0.99	0.037*	0.55	0.41 – 0.74	<0.001*
SES	0.99	0.92 – 1.06	0.795			
Fruit & vegetable consumption	0.82	0.53 – 1.26	0.359			
Alcohol use	0.71	0.45 – 1.12	0.139			
Tobacco use	0.86	0.46 – 1.60	0.639			
Energy expenditure	1.00	0.99 – 1.00	0.206			
Family history	1.48	0.96 – 2.27	0.071			
<b>Clinical</b>						
WC	1.16	1.12 – 1.20	<0.001*	1.19	1.13 – 1.25	<0.001*
Prev. diag. - HPT	1.77	1.14 – 2.76	0.011*			
SBP	1.01	1.00 – 1.02	0.039*			
DBP	1.02	1.01 – 1.04	0.006*			
Prev. diag. - DM	1.07	0.63 – 1.80	0.812			
HbA <sub>1c</sub>	1.17	0.93 – 1.48	0.173			

\* statistically significant



### 4.3 ASSOCIATIONS BETWEEN RISK FACTORS

Trends in communicable diseases can be determined by using coefficients of determination ( $r$ -squared); however NCD's generally have a multi-factorial aetiology which explains why many of the associations have a low  $r$ -squared value with a correspondingly high  $p$ -value. Often, outlying (or extreme values) of a variable affects the relationship between two variables resulting in biased regression coefficients and corresponding low  $r$ -squared values. Removing these outlying values can improve the model and increase the  $r$ -squared value.

#### 4.3.1 BMI vs. SBP

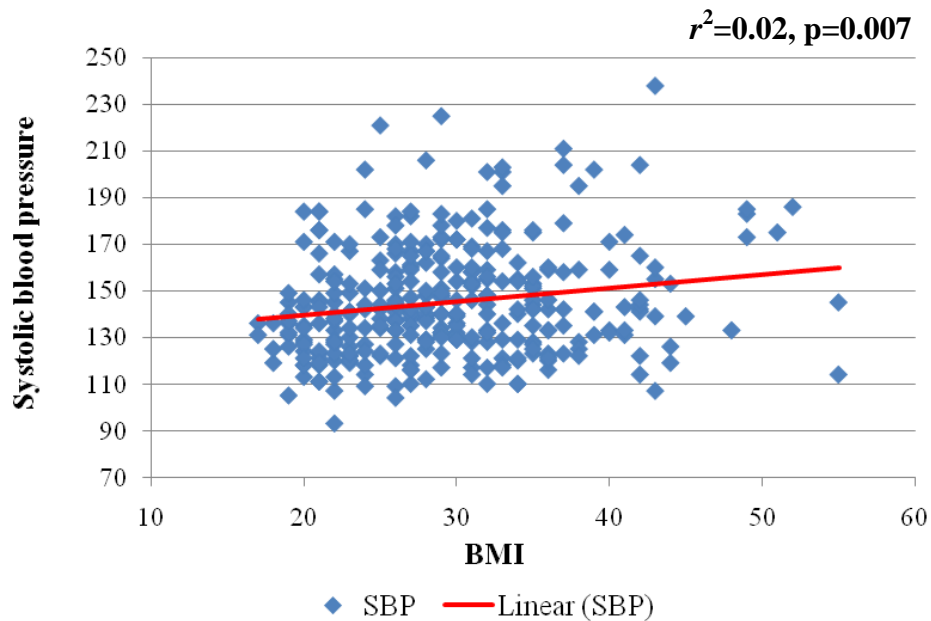
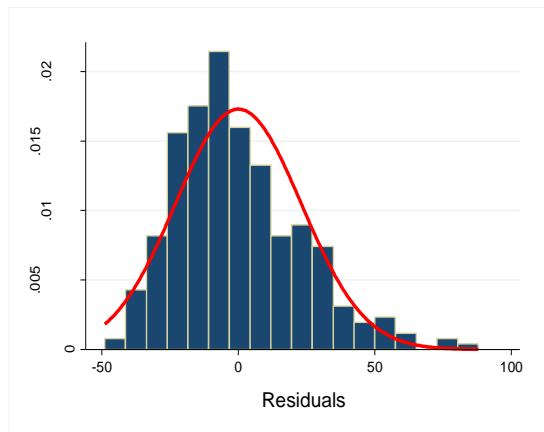


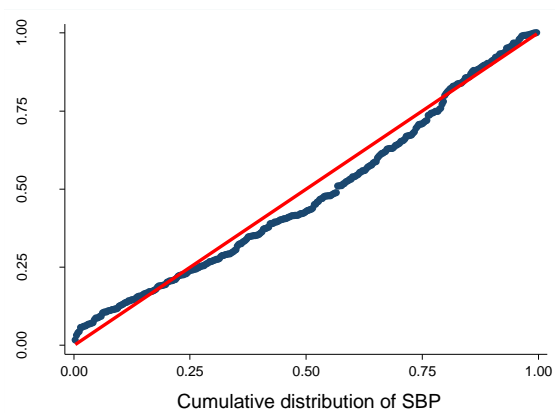
Figure 4.14: Scatter plot of SBP vs. BMI

Systolic blood pressure and BMI are correlated ( $r=0.168$ ,  $p=0.002$ ). The relationship appears to be a linear and significant ( $p<0.001$ ) (Figure 4.14). The  $r$ -squared value only explains about 2.19% of the variation in SBP by BMI ( $r^2=0.0219$ ). This is probably due to the multi-factorial aetiology of HPT. Therefore, a residual analysis was done to fully explore the association.

**Normality:** When testing the studentized residuals of SBP, the mean value was close to 0 (mean = 0.0004) and the median value was slightly smaller than the mean (median = -0.18), suggesting a normal distribution. Ninety percent of all studentized SBP values of lie between -1.36 to 1.84 (95% CI -1.08 – 0.11). This suggests that there was little difference between the observed and predicted values of SBP.



(a) Histogram of the residuals

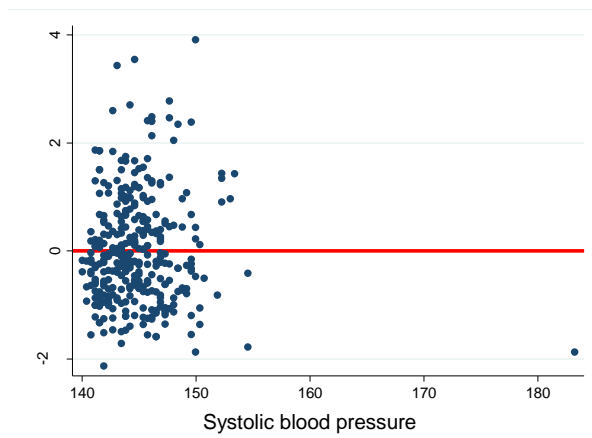


(b) Probability-probability plot of residuals

**Figure 4.15: Normality plots of residuals of SBP**

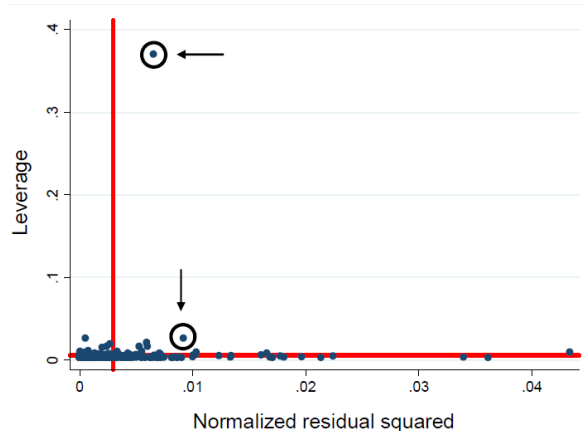
The histogram of the residuals of SBP in Figure 4.15(a) appears approximately normally distributed. Figure 4.15(b), the normal probability-probability (normal PP plot) plot plotting the cumulative distribution of the residuals against the cumulative distribution of the SBP, confirms this. On statistical testing for normality (Shapiro Wilk test,  $p < 0.001$ ) however, the p-value was less than 0.05, thus the null hypothesis that the data are normally distributed was rejected. This data suggests that while SBP and BMI were associated, the increase in SBP is not accompanied by a consistent proportional change in the BMI.

**Common variance and linearity:** Plotting the studentized residuals against the SBP values should show a random pattern in the residuals (Figure 4.16). In this case, the evidence is against the null hypothesis that the variance is homogeneous (Breusch-Pagan test,  $p = 0.008$ ). This plot suggests that SBP has a heterogeneous distribution confirming the variability described under *Normality*.



**Figure 4.16: Plot of studentized residuals vs. SBP**

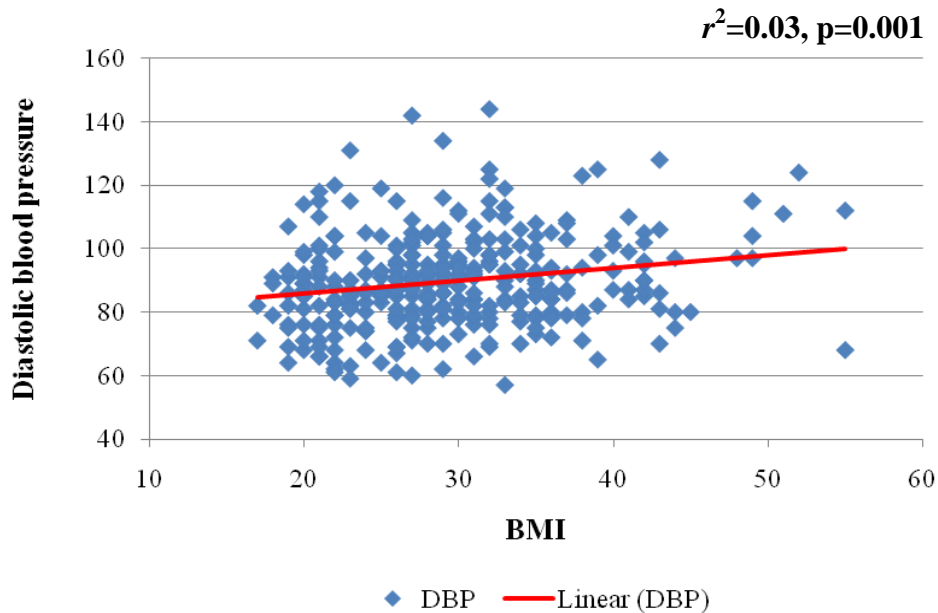
**Outliers & Cook's distance:** Values identified as outliers by Cook's Distance (which combine the leverage and residual values) were deleted (indicated by arrows in Figure 4.17); and the models run again.



**Figure 4.17: Plot of leverage values against squared residuals of SBP**

**Relationship once the outliers were removed:** The model did not improve with removal of the outliers ( $r^2=0.01$ ). This implies that the curve describing the initial relationship between SBP and BMI in Figure 4.14 holds true. These findings have two important clinical implications. Firstly, despite the multiple variables implicated in the aetiology of NCDs, increasing levels of SBP can be expected as the BMI increases. The amount of change in BMI was variable and the relationship between SBP and BMI persists at even extreme values. This persistence makes these study findings generalizable.

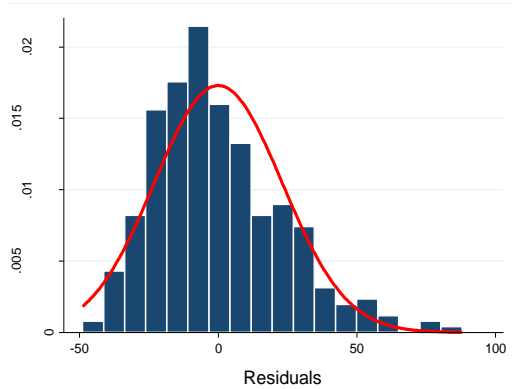
### 4.3.2 BMI vs. DBP



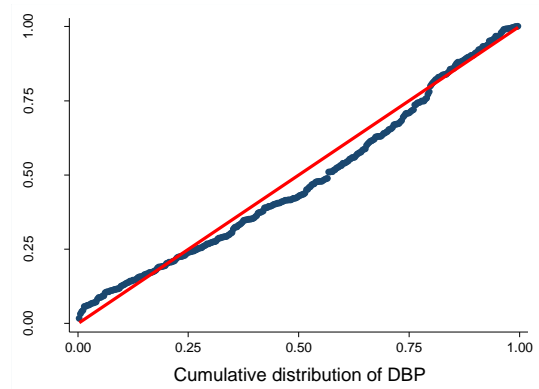
**Figure 4.18: Scatter plot of DBP vs. BMI**

Diastolic blood pressure and BMI are correlated ( $r=0.20, p<0.001$ ). The relationship appears to be a linear and significant ( $p=0.001$ ) (Figure 4.18). The  $r$ -squared value explains about 3.10% of the variation in DBP by BMI ( $r^2=0.0310$ ). This is probably due to the multi-factorial aetiology of HPT. Therefore, a residual analysis was done to fully explore the association.

**Normality:** When testing the studentized residuals of DBP, the mean was close to 0 (mean = -0.0001) and the median was slightly smaller than the mean (median = -0.52), suggesting a normal distribution. Ninety percent of all studentized DBP values of lie between -1.52 to 1.91 (95% CI -0.11 – 0.11). This suggests that there was little difference between the observed and predicted values of DBP.



(a) Histogram of the residuals

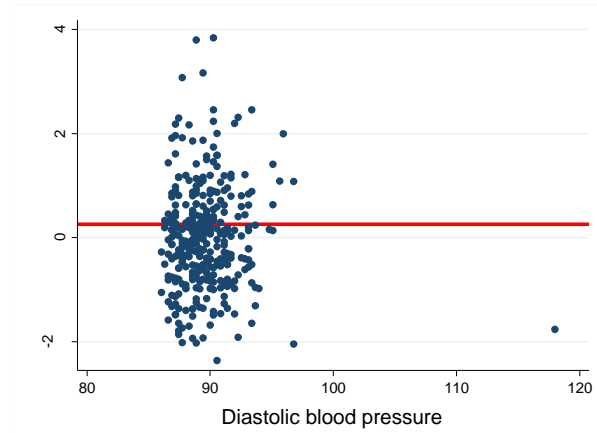


(b) Probability-probability plot of residuals

**Figure 4.19: Normality plot of residuals of DBP**

The histogram of the residuals in Figure 4.19(a) appears approximately normally distributed. The normal PP plot in Figure 4.19(b) also suggests this. On statistical testing for normality (Shapiro Wilk test,  $p < 0.001$ ) however, the p-value was less than 0.05, thus the null hypothesis that the data are normally distributed was rejected. This data suggests that while DBP and BMI were associated, the increase in SBP is not accompanied by a consistent proportional change in the BMI.

**Common Variance & Linearity test:** The variance of the residuals for DBP were homogenous (Breusch-Pagan test,  $p = 0.360$ ), though plotting the residuals against the DBP appears to be a random distribution (Figure 4.20). This plot suggests that DBP has a heterogeneous distribution confirming the variability described under *Normality*.

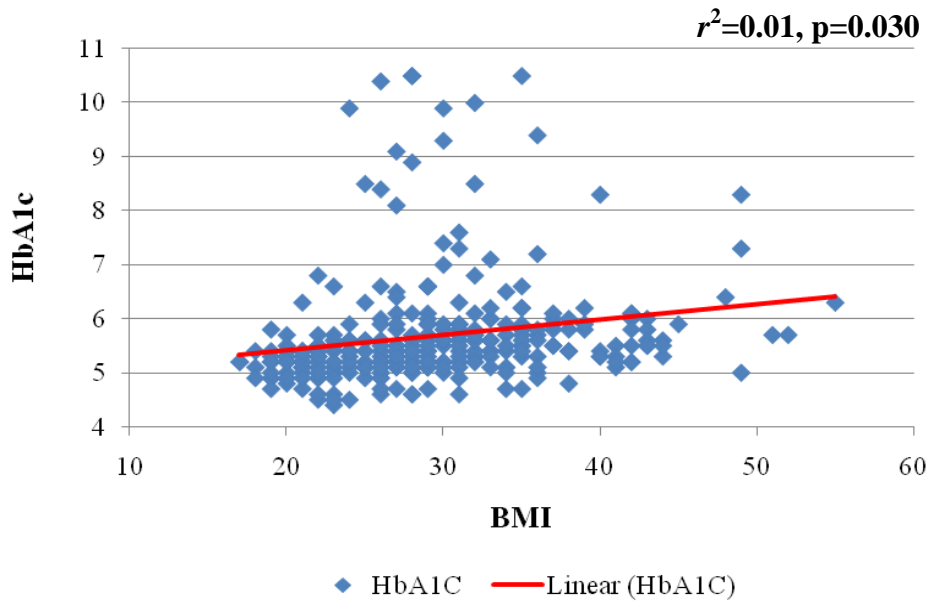


**Figure 4.20: Plot of studentized residuals and DBP**

**Outliers & Cook's distance:** Outliers identified by Cook's Distance were deleted; and the models run again.

**Relationship once the outliers were removed:** The model did not improve with removal of the outliers ( $r^2=0.03$ ). The initial model of the relationship between DBP and BMI in Figure 4.18 remains valid. Despite the multiple variables implicated in the aetiology of NCD's, the evidence suggests that increasing levels of DBP can be expected as the BMI increases. Again, these findings have two important clinical implications. Firstly, despite the multiple variables implicated in the aetiology of NCDs, increasing levels of DBP can be expected as the BMI increases. The amount of change in BMI was variable and the relationship between BMI and DBP persists at even extreme values. This persistence makes these study findings generalizable.

### 4.3.3 BMI vs. HbA1c



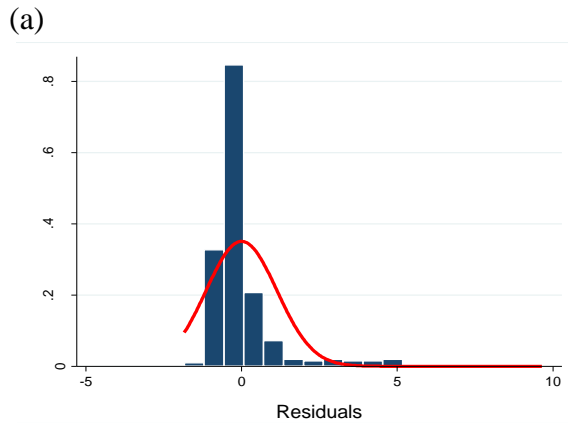
**Figure 4.21: Scatter plot of HbA<sub>1c</sub> vs. BMI**

HbA<sub>1c</sub> levels and BMI are correlated ( $r=0.38, p<0.001$ ). The relationship appears to be a linear and significant ( $p=0.030$ ) (Figure 4.21). The  $r$ -squared value explains about 1.45% of the variation in HbA<sub>1c</sub> by BMI ( $r^2=0.0420$ ). This is probably due to the multi-factorial aetiology of DM. Therefore, a residual analysis was done to fully explore the association.

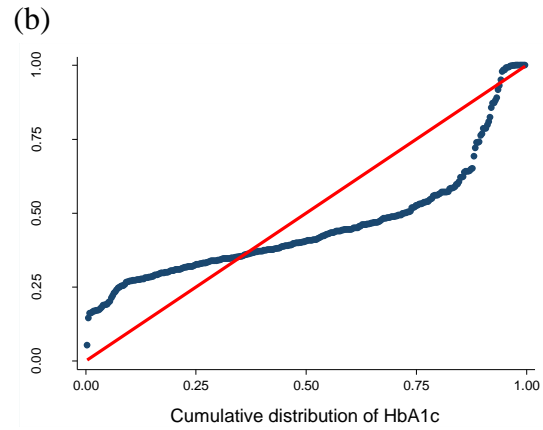
**Normality:** The distribution of the HbA<sub>1c</sub> data was tested using the residual values and the studentized residual values. When testing the studentized residuals of HbA<sub>1c</sub>, the mean value was close to 0 (mean = 0.005) and the median value was slightly smaller than the mean (median = -0.24), suggesting a normal distribution. Ninety percent of all studentized HbA<sub>1c</sub> values of lie between -0.85 and 2.15 (95% CI -1.09 – 0.12). This suggests that there was little difference between the observed and predicted values of HbA<sub>1c</sub>.



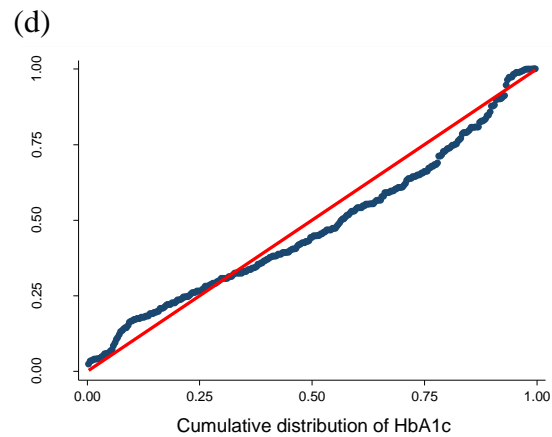
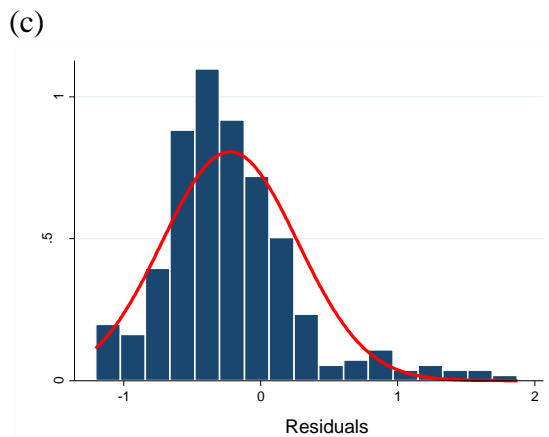
**Histogram of the residuals**  
*Before removal of outliers*



**Probability-probability plot of residuals**



*After removal of outliers*



**Figure 4.22: Normality plot of residuals of HbA<sub>1c</sub> before and after removal of outliers**

The residuals of HbA<sub>1c</sub> in Figure 4.22(a) and (b) have a non-normal distribution. This was statistically confirmed (Shapiro Wilk test,  $p < 0.001$ ) with a p-value less than 0.05 rejecting the null hypothesis of normal distribution. This data suggests that while HbA<sub>1c</sub> and BMI were associated, the increase in HbA<sub>1c</sub> is not accompanied by a consistent proportional change in the BMI.

**Common Variance & Linearity test:** The variance of the residuals was homogenous (Breusch-Pagan test,  $p=0.848$ ). This suggests that HbA<sub>1c</sub> has a heterogeneous distribution confirming the variability described under *Normality*.

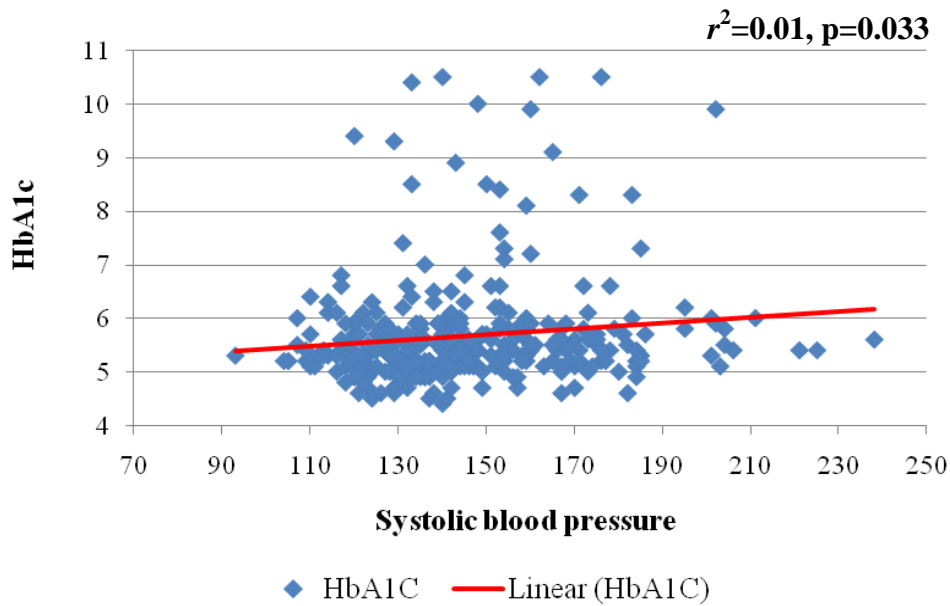
**Outliers & Cook's distance:** Outliers identified by Cook's Distance (which combines leverage and residual values) were deleted; and the models run again.

**Relationship once the outliers were removed:** The model improved with removal of the outliers. The outliers represent aberrations that do not reflect the characteristics of the population being explored. Twelve percent of the variation in HbA<sub>1c</sub> can now be attributed to BMI levels ( $r^2=0.12$ ). The improvement in the model is graphically represented in Figure 4.22(c) and (d) though statistical testing still did not show normality in the distribution (Shapiro Wilk test,  $p<0.001$ ). This evidence shows that increasing levels of BMI was significantly associated with increases in HbA<sub>1c</sub> levels, though the amount of change could not be reliably predicted.

The reason why outlying variables impact on the association between DM and BMI and not with the association between HPT and BMI (discussed in Section 4.4.1: BMI vs. SBP and Section 4.4.2: BMI vs. DBP) can only be postulated. Perhaps, it has to do with the difference in testing between DM and HPT, or that DM may present with intractable disease at an earlier age in this population.

The differences between the outlying values and the rest of the study population were explored further. The participants identified as outliers were older (55 vs. 43 years) (Mann-Whitney,  $p=0.001$ ) and 57% were married (chi square test,  $p<0.001$ ) with 78% have lower education levels (chi square test,  $p=0.003$ ). Clinically, the outliers had significant histories of DM (chi square test,  $p<0.001$ ) and HPT (chi square test,  $p<0.001$ ) and had larger WC ( $104 \pm 12\text{cm}$  vs.  $98 \pm 16\text{ cm}$ ) (Student's t-test,  $p=0.043$ ), BMI values ( $31\text{ kg/m}^2$  [IQR 27-36] vs.  $28\text{ kg/m}^2$  [IQR 23-33]) (Mann-Whitney,  $p=0.026$ ) and HbA<sub>1c</sub> ( $9.3\%$  [IQR 8.4-10.4] vs.  $5.4\%$  [IQR 5.1-5.7]) (Mann-Whitney,  $p<0.001$ ) levels than the rest of the study population. Apart from the impact of increased age and lower education levels on poor glycaemic control, it is possible that the significant history of HPT may have played a role in the extreme values demonstrated. The significant history of DM among the outliers alludes to the poor control describes in Section 4.3.2.2: Control of DM.

#### 4.3.4 HbA1c vs. SBP

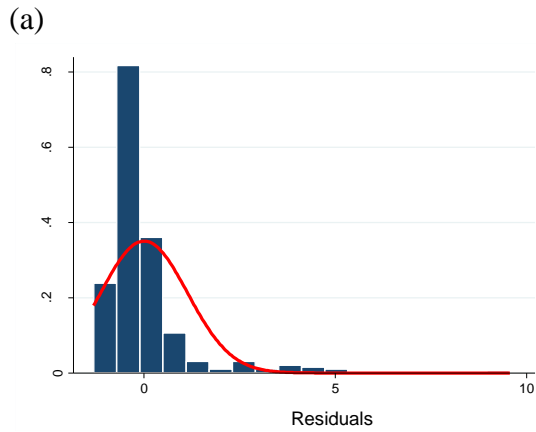


**Figure 4.23: Scatter plot of HbA<sub>1c</sub> vs. SBP**

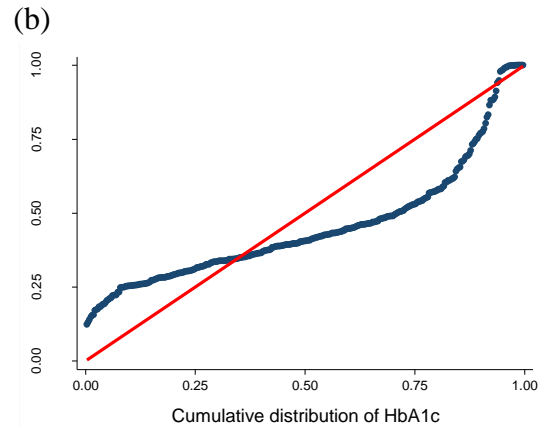
Systolic blood pressure and HbA<sub>1c</sub> are correlated ( $r=0.15, p=0.006$ ). The relationship appears to be a linear and significant ( $p=0.033$ ) (Figure 4.23). The  $r$ -squared value explains about 1.40% of the variation in HbA<sub>1c</sub> by SBP ( $r^2=0.0140$ ). This is probably due to the multi-factorial aetiology of DM. Therefore, a residual analysis was done to fully explore the association.

**Normality:** When testing the studentized residuals of HbA<sub>1c</sub>, the mean was close to 0 (mean = 0.006) and the median was slightly smaller than the mean (median = -0.24), suggesting a normal distribution. Ninety percent of all studentized HbA<sub>1c</sub> values of lie between -0.82 and 2.10 (95% CI -0.11 – 0.12). This suggests that there was little difference between the observed and predicted values of HbA<sub>1c</sub>.

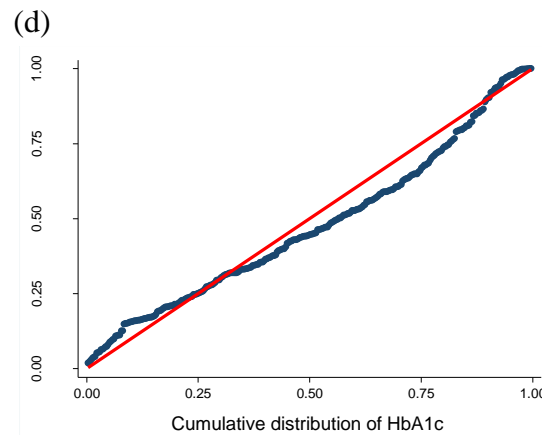
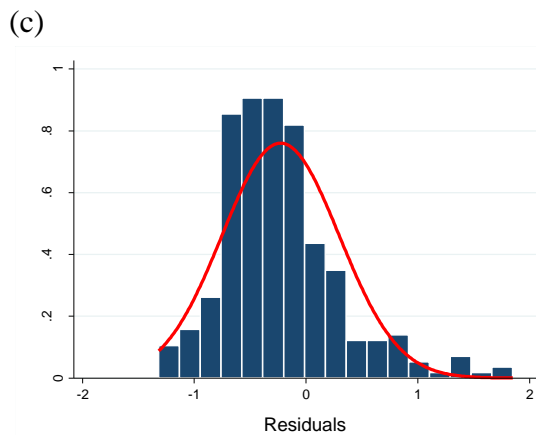
**Histogram of the residuals**  
*Before removal of outliers*



**Probability-probability plot of residuals**



*After removal of outliers*



**Figure 4.24: Normality plot of residuals of HbA<sub>1c</sub> before and after removal of outliers**

The residuals of HbA<sub>1c</sub> in Figure 4.24(a) and (b) have a non-normal distribution. This was statistically confirmed (Shapiro Wilk test,  $p < 0.001$ ) with a p-value less than 0.05 rejecting the null hypothesis of normal distribution.

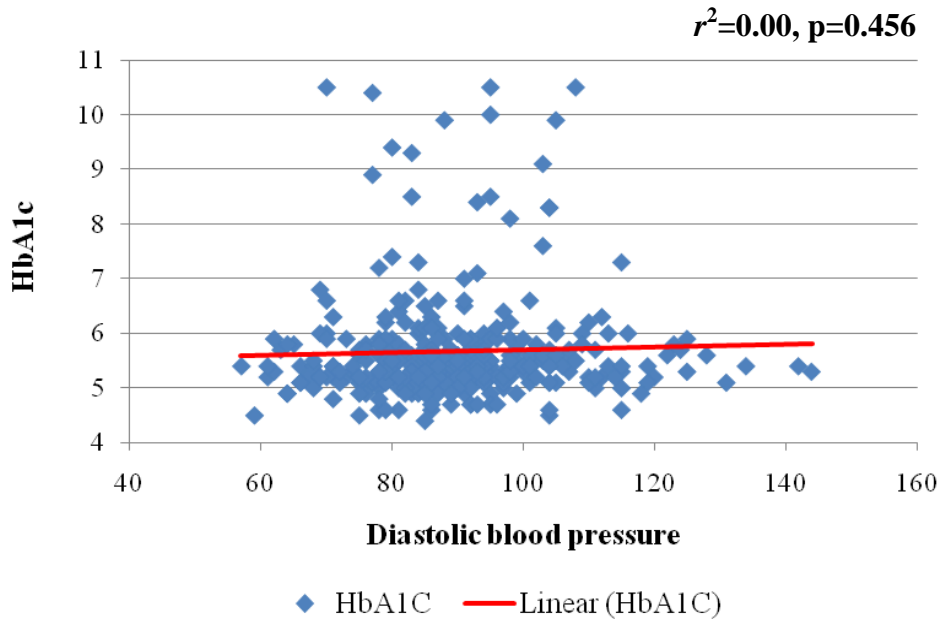
This data suggests that while HbA<sub>1c</sub> and SBP were associated, the increase in HbA<sub>1c</sub> is not accompanied by a consistent proportional change in the SBP. This suggests that HbA<sub>1c</sub> has a heterogeneous distribution confirming the variability described under *Normality*.

**Common Variance & Linearity test:** In this case, the evidence is against the null hypothesis that the variance is homogeneous (Breusch-Pagan test,  $p < 0.001$ ).

**Outliers & Cook's distance:** Outliers identified by Cook's Distance (which combines leverage and residual values) were deleted; and the models run again.

**Relationship once the outliers were removed:** The model did not improve with removal of the outliers ( $r^2=0.01$ ,  $p=0.09$ ). Despite the model improving graphically [Figure 4.24(c) and (d)], the distribution of the data remained non-normal (Shapiro Wilk test,  $p < 0.001$ ). In this study population, no significant relationship could be found between HbA<sub>1c</sub> levels and SBP.

### 4.3.5 HbA1c vs. DBP



**Figure 4.25: Scatter plot of HbA<sub>1c</sub> vs. DBP**

Diastolic blood pressure and HbA<sub>1c</sub> levels were not correlated ( $r=0.08, p=0.136$ ) despite there being an appearance of a linear relationship (Figure 4.25). The p-value for the overall regression model was not significant ( $p=0.456$ ) implying no relationship between DBP and HbA<sub>1c</sub>.

#### **4.4 THE METABOLIC SYNDROME**

In this section, factors related to the development of the MS are discussed. The blood samples collected for the lipid profiles were incorrectly stored making analysis of these samples impossible. Financial constraints did not allow for recollection of these samples. Additionally, HbA<sub>1c</sub> results (based on the ADA criteria for the diagnosis of DM) were used as a surrogate for the fasting plasma glucose samples. This was done considering that HbA<sub>1c</sub> blood sample collection represented a less labour intensive method given the financial and resource constraints of this project. The findings relating to the MS presented in this analysis is thus limited as it is based only on the waist circumference, hypertension and glucose criteria (using HbA<sub>1c</sub> as a surrogate) of the IDF definition of the MS (defined in: Glossary of terms).

##### **4.4.1 Prevalence of the MS**

Participants were assessed for the MS by gender. One hundred and ninety five females (92%) met the criteria of WC  $\geq$  80cm for the MS as stipulated by the IDF (chi square test,  $p < 0.001$ ). Only 64 males (52%) had WC  $\geq$  94cm. Raised HbA<sub>1c</sub> levels (HbA<sub>1c</sub>  $\geq$  5.6%) was found in 117 participants (89 female, 28 male). Seventy nine females and 25 males were diagnosed with the MS (chi square test,  $p < 0.001$ ). The prevalence of the MS in the study population was 31% (104/337). Fifty one females (24%) and 24 males (19%) met all three criteria for the diagnosis of hypertension (chi square test,  $p = 0.329$ ) for the diagnosis of the MS. Similarly, 18 females (8%) and 12 males (10%) fulfilled both



criteria for DM (chi square test,  $p=0.703$ ) (data not shown). Table 4.11 demonstrates the prevalence of the MS and the associated risk factors.

**Table 4.11: Prevalence of the MS and associated risk factors (n=337)**

Variable	Total		Female		Male		p-value
	n=337	(%)	n=213	(%)	n=124	(%)	
<b>Diagnosed with MS</b>	104	(31)	79	(23)	25	(7)	0.001*
<b>Risk factors for MS</b>							
WC‡	259	(77)	195	(58)	62	(19)	<0.001*
History of hypertension	111	(33)	81	(24)	30	(9)	0.032*
SBP $\geq$ 130mmHg	243	(72)	145	(43)	98	(29)	0.030*
DBP $\geq$ 85 mmHg	213	(63)	129	(38)	84	(25)	0.188
History of DM	37	(11)	20	(6)	17	(5)	0.439
HbA <sub>1c</sub> $\geq$ 5.6%	117	(35)	89	(26)	28	(8)	<0.001*

\* statistically significant

‡ meeting the IDF criteria for WC ( $\geq$  94cm-males,  $\geq$  80cm-females) (9)

#### 4.4.2 Factors associated with the MS

The analysis in Table 4.12 reflects the significant association of female gender, older age with the MS on both bivariate and multivariate analysis. Lower levels of education were again associated with disease. The MS had no associations with SES or with levels of consumption of alcohol or use of cigarettes.

**Table 4.12: Factors associated with the MS on bivariate and multivariate analysis (n=337)**

	Bivariate			Multivariate (p<0.001)		
	Odds ratio	95% CI	p-value	Adjusted Odds Ratio	95% CI	p-value
<b>Socio-demographic</b>						
Gender	2.33	1.38 – 3.94	0.002*	2.32	1.33 – 4.06	0.003*
Age	1.06	1.04 – 1.08	<0.001*	1.06	1.04 – 1.08	<0.001*
Marital status	1.36	1.10 – 1.67	0.004*			
Education	0.27	0.16 – 0.46	<0.001*			
Work	1.48	1.20 – 1.82	<0.001*			
SES	1.23	0.78 – 1.96	0.502			
Fruit & vegetable consumption	1.04	0.66 – 1.64	0.849			
Alcohol use	1.22	0.76 – 1.96	0.406			
Tobacco use	0.89	0.46 – 1.72	0.733			
Energy expenditure	1.30	0.48 – 3.50	0.600			
Family history	1.18	0.76 – 1.83	0.468			

\*statistically significant

#### 4.5 SUMMARY OF THE CHAPTER

This chapter outlined the key results of the study. The study population was black with middle to higher SES and education levels below Grade 12 mostly. The prevalence of DM in this study population was 14%, with many undiagnosed individuals and those with disease having poor control. More than half the study population had HPT (58%) and most were sub-optimally or poorly controlled. This was a markedly obese population (39%) with 54% of women having a BMI categorised as obese (BMI  $\geq$  30 kg/m<sup>2</sup>). There is a high prevalence of the MS (31%) with the syndrome affecting mostly females (79/213, 37%).

## **CHAPTER 5**

### **DISCUSSION**

This chapter describes the findings of the study in the context of the available literature. The clinical information will be discussed first followed by the socio-demographic data.

#### **5.1 INTRODUCTION**

The study aims to determine the prevalence of cardio-metabolic diseases and the associated risk factors in the community of Chiawelo, Johannesburg Health District, Gauteng Province. This data will hopefully contribute to reducing the burden of disease through early screening and identification of cases, improving long term management and minimising the development of complications by targeting the at-risk populations.

#### **5.2 DIABETES MELLITUS**

##### **5.2.1 Prevalence of DM**

The self-reported prevalence of DM is 11% (35/326). This differs from the calculated prevalence of DM of the study population classified according to the HbA<sub>1c</sub> results in the current ADA guidelines (1). According to this classification, the prevalence is 14% (47/326) with similar distributions amongst males and females. When considering DM prevalence, data from rural areas in South Africa suggest rates of 8.8% in females and 8.5% in males (28). In Gauteng, the prevalence has steadily increased from 28% in 2005

to a staggering 73% in 2007 according to the DHIS data (127), though the reliability of this data may be brought into question. In 2006/07, the incidence of DM is thought to have stabilised in Gauteng Province, with approximately 112 new cases per 100 000 people 45 years and older, diagnosed annually (56).

While SEMDSA does not advocate the use of the HbA<sub>1c</sub> for diagnosing DM (123;124), the prevalence of 14% calculated in this study based on HbA<sub>1c</sub> data, in the background of previously reported prevalence of 5.5% among South Africa adults aged 30 years and over in 2001 (128) and 5-8% in the black population in 2009 (129), cannot be ignored.

From those participants not reporting DM, there is a high number that are classified as 'pre-diabetic' (52/285, 18%) with females predominating (44/182, 24%). These individuals have IGT, are likely to develop DM, and render themselves susceptible to the complications that are associated with poor glycaemic control. Data from the rural Ubombo district in Kwa-Zulu Natal confirms the epidemic proportions of glucose intolerance in that community, which far exceeds other rural communities in Africa (130). The Framingham Heart Study also highlights pre-diabetes as a key risk factors for the development of DM (52). These pre-diabetic conditions have a 25-50% lifetime risk of developing DM and this group should be specifically targeted for primary preventative strategies (131). Both the Finnish Diabetes Prevention Study and the Chinese Da Qing Study have conclusively shown that that the progression to DM from a pre-diabetic state can be prevented through dietary modification to promote weight loss and increasing levels of physical activity (73;132).

The Framingham Offspring Study and NHANES 2001-2004 found HbA<sub>1c</sub> to be positively associated with age in non-diabetic subjects (133). Globally there has been a foreboding decline in the age of diagnosis of DM with between 8-45% of all new cases diagnosed in children and teenagers (134). While the 2004 National programme for control and management of Diabetes Type 2 at primary level identified those older than 60 years at risk of DM (96), findings from this study suggest earlier age of onset (Chapter 4: Table 4.7) for both IGT and DM.

### **5.2.2 Identifying undiagnosed cases**

The testing of HbA<sub>1c</sub> levels in the study identifies 12 individuals with undiagnosed DM (12/326, 4%) from those who did not report having DM. These are similarly distributed between the males (3%) and females (4%). However, many studies have reported that DM is a stronger risk factor for CVD in females than in males, with a 50% higher relative risk in females than in males (125). Bearing this in mind, current data suggests that more than half of women and three quarters of men in requiring intervention for HPT and DM in South Africa are unaware that they are suffering from these conditions (135), emphasizing the need for early diagnosis.

### 5.2.3 Management and control of DM

Deranged glycaemic control together with all other cardio-metabolic disease parameters support the fact that comprehensive diabetic care appears to be lacking in the three major academic hospitals in Johannesburg (129). Few participants in this study have optimal glycaemic control (8/35, 23%) from those who report having the disease (35/326). The majority are poorly controlled (23/35, 66%). Levitt et al showed that black South African patients had a poor ability to manage their disease (136). Self management of diabetes is knowledge dependant and this reflects a poor understanding of the disease by the black South African population (50) which is in keeping with the educational level of this population and its association with age.

Rural studies conducted in Limpopo Province found that black patients with DM had poor levels of glycaemic control but also presented with related conditions as dyslipidaemia, obesity and elevated BP levels (137). Many studies have shown that DM has sub-optimal control in South Africa, with education having a positive, but not very impressive, impact on glycaemic levels (138). This is important because health literacy must be considered in formulating diabetes education. Perhaps the answer lies in engaging the patient more in the management of their condition. Evidence from controlled clinical trials point to the fact that programmes focussing on self-management skills have proven to be more effective than information only patient education in improving clinical outcomes (139).

#### **5.2.4 Factors associated with developing DM**

Increasing age and a previous diagnosis of HPT are associated with developing DM on multivariate analysis. Lower education levels are associated with disease on bivariate analysis and this cross-cuts the discussion on the interplay between age and education level. Increasing weight and a history of HPT (all factors inextricably linked) are associated with DM.

### **5.3 HYPERTENSION**

#### **5.3.1 Prevalence of HPT**

The self-reported prevalence of HPT is 33% (111/337). This differs from the calculated prevalence of HPT of the study population classified according to the current guidelines (6). According to this classification, the prevalence is 58% (197/337) with similar distribution in females (60%) and males (56%). This is in keeping with the prevalence rates of HPT identified in the Heart of Soweto Study at 33% for either SBP or DBP elevation (34). Many South African studies from as early as 1998 have confirmed the high levels of BP and HPT in black people (35;45;140) but have rates significantly higher in obese, black women (104) while this research report shows comparable rates in males and females. The prevalence of HPT in this study far exceeds the self-reported rates in Gauteng adults in 2006/07 (9% for males, 21% for females) (56). Rural South African data seems more feasible with a HPT prevalence of 26% in females and 22% in males

(28). Data from the 1996 Mamre Study looking at a coloured community in Cape Town showed HPT prevalence rates of 22% in men and 16% in females (141).

From those participants not reporting having HPT, there is a high number that are classified as 'prehypertensive' (112/220, 51%) with males predominating (58/124, 47%). These individuals form a group at high risk of developing HPT. The identification of this group is vitally important as it is known that HPT occurs at an earlier age, is more severe and is more likely to result in early end-organ damage in the black population (34). Males present at an earlier age with prehypertension and HPT in this study. This is supported by South African data showing mean SBP increases with age and this was higher in men than in women at all ages (57).

There does appear to a clearly defined age at which screening for HPT should occur in South Africa, probably because BP measurements are routinely conducted at most PHC visits (opportunistic screening). The 2006 South African Hypertension Guideline consider DM (in females > 65 years and males > 55 years) and family history of early onset of CVD (in females aged < 65 years and males aged < 55 years) as major risk factors for the development of HPT (97). The ages documented in this study for prehypertension and HPT were low, particularly among males. The median age of prehypertension was 43 years (IQR 30-57) among females and 33 years (IQR 25-52) among males with increasing ages in both genders as they reached more severe levels of disease.



### **5.3.2 Identifying undiagnosed cases**

Measuring BP in this study identifies 83 individuals with undiagnosed HPT (83/337, 25%) from those not reporting HPT. These undiagnosed cases represent 42% of males and 35% of females. This is a major proportion of the study population reflecting the high burden of disease even for this sample.

### **5.3.3 Management and control of HPT**

Few recorded optimal hypertensive control (6/111, 5%) from those reporting having the disease (111/337). The majority are poorly controlled (68/111, 61%). These findings are significantly different from data at three primary care sites servicing three townships in Cape Town that showed optimal control in 33% of men and 44% of women (58). It is vital that the poor control in this study be addressed as higher levels of SBP and DBP have been documented in black women (29) and the general black population has been shown to have twice the CVA rates than their white counterparts (35). Many large epidemiological studies have shown that a simple 2 mm Hg reduction in mean BP will decrease the risk of CVD by 10% (23).

Barriers to optimum HPT care exist at the level of the patient, provider and organisation and include lack of awareness of the dangers of untreated HPT or the benefits of control, poor patient-provider relationships, unemployment rates, cost of care and medications and side effects (58). Improved HPT control can be achieved with active participation of the patient through greater awareness and knowledge and explanation of their actual BP measurements (58).

#### **5.3.4 Factors associated with developing HPT**

Increasing age and weight are associated with developing HPT. Family history did not impact on disease prevalence in this population. Again, the importance of education is highlighted with lower levels of education being associated with developing HPT. The HiHi Study found that significant predictor of lower levels of SBP, DBP or BP control included: fewer medications prescribed, better compliance to HPT recommendations, younger age, female respondents, higher levels of education and attending private health care (58). In addition to these, the 1998 SADHS also quoted family history and obesity as significantly associated with the risk of HPT (104).

## **5.4 OBESITY**

### **5.4.1 Prevalence of obesity**

The change in diet to more saturated fats and sugars is largely responsible for high rates of obesity characterising the emergence of NCDs in South Africa, particularly in urban black women (29;56). The obesity levels ( $\text{BMI} \geq 30 \text{ kg/m}^2$ ) are marked (133/337, 39%) in this research. Particularly concerning is the level of obesity among females reaching 54% (114/213). The Heart of Soweto Study identified obesity as the most prevalent CVD risk factor (43%) with 55% of women classified as obese (34). Data from rural South Africa identified 52% of females as overweight or obese (28). The 2003 SADHS demonstrated that approximately 10% of adult men and 30% of adult women in Gauteng were obese, while 20% of adult men and 28% of adult women are overweight (142). This study found much higher rates of being overweight in males (42/124, 34%) but similar rates in females (59/231, 28%).

The HiHi study showed that 85% of participants were overweight or obese despite these participants perceiving themselves to be of normal or under-weight (58). These perceptions were highly associated with education levels among black South Africans with the least educated black males and females having the greatest discrepancies between actual and perceived BMI (58;61). Being overweight and/or obese poses a significant risk to health, especially among females, with 87% of patients with DM, 68% of hypertensives and 38% of those with IHD having a BMI greater than  $21 \text{ kg/m}^2$  in the

1998 SADHS (143). Despite the perceptions of weight and the possible sensitivity surrounding the issue of being overweight or obese, the epidemic has to be addressed.

#### **5.4.2 Factors associated with developing obesity**

Obesity was associated with WC on both bivariate and multivariate assessment. In the Gauteng study, WC is also the parameter that differentiated between the presence and absence of the MS in patients with both HPT and DM (59). Lower levels of education are again associated with disease. Apart from WC, gender was also associated with the MS on multivariate analysis. High levels of obesity among females is a recurring theme in the literature (29;34;56).

### **5.5 ASSESSMENT OF CARDIOVASCULAR RISK FACTORS**

Unlike communicable diseases, NCDs generally have a multi-factorial aetiology which explains why many of the associations discussed have a low r-squared value with a correspondingly high p-value. The residual analyses are being done to better interpret the relationship between these risk factors.

In this study population, increasing values of SBP and DBP can be expected as the BMI increases. However, similar increases in BMI are not associated with increases in HbA<sub>1c</sub> in this population. This negative finding is to the contrary of existing data that supports an inter-relationship between all risk factors. Increased body weight is a highly prevalent

and highly implicated component of the MS, but it also has strong associations with DM, HPT, dyslipidaemia and atherosclerosis (80). Data from the UK show that mortality from CVD increases incrementally as the presence of the MS features increase (144).

This study also demonstrated increasing levels of BMI to be significantly associated with increases in HbA<sub>1c</sub> levels. Many studies have identified obesity as an important independent modifiable predictor of DM (8;51), with almost 80% of people overweight at the time of diagnosis of DM (8). The United Kingdom Prospective Diabetes Study (UKPDS) observed that the mean BMI at the time of DM diagnosis was 28 – 29 kg/m<sup>2</sup> (145). Most cases, however, will present at a BMI or between 25 – 30 kg/m<sup>2</sup> (146). There are probably differences in disease development based on ethnicity with Asians developing disease at lower BMI values than the white population (51). Again, data on the black population is lacking.

Smoking accounted for between 41 632 – 46 656 deaths in South Africa in 2000, with three times more deaths in males than females, mostly attributable to CVD (147). The high mortality in males is likely due to the fact that male smoke more tobacco than females. The prevalence of tobacco use was higher in males (57%) than in females (36%) in studies conducted in the Limpopo Province (28). Perhaps the lower levels of tobacco use in this study (52/337, 15%) could be the result of the stringent anti-tobacco laws currently in place.

## **5.6 THE METABOLIC SYNDROME**

The MS was defined using the IDF criteria for WC, HPT and DM but using HbA<sub>1c</sub> levels as a surrogate for fasting plasma glucose levels (Section 3.6.1.2 Clinical variables: Metabolic syndrome). Lipid profile results were unavailable.

### **5.6.1 Prevalence of the MS**

The prevalence of the MS in the study population is 31% (104/337) with 37% female (79/213) and 20% male (25/124). The rate of disease in the study population is significantly higher than the 25% prevalence reported among black South African women in 2009 also using the IDF criteria (148). The prevalence of MS was 60% in a group of 40 patients with established CAD and no previous history of DM in Johannesburg (59). Most participants (257/337, 76%) met the IDF criteria for WC for the diagnosis of the MS with the majority being female (92%) (1;9). These rates are particularly higher than South African studies conducted among students that found only 24% of females and 14% of males exhibited WCs above the acceptable range (149). The POWIRS (Profiles of Obese Women with the Insulin Resistance Syndrome) study found that 48% of black women exceed the IDF criteria for WC (29). Perhaps this reflects the more sedentary lifestyle of the study population compared to that of a student population.

### **5.6.2 Factors associated with the MS**

Female gender and increasing age are associated with having the MS on multivariate analysis. Both this research report and preceding studies have associated female gender with disease (29;149). In Johannesburg, increased WC with HPT and elevated glucose levels were found to be the most recurring combination of risk factors implicated in the development of the MS in a study of 40 black subjects with established CAD but these individuals did not report a previous diagnosis of DM (59).

Significant associations are found with age and the development of the MS on bivariate analysis. This is important as data from the Coronary Artery Development in Young Adults (CARDIA) study found the MS to be associated with increasing age and appeared to be more prevalent in the black population (150). There are no associations between the MS and physical activity in this study even though higher levels of physical activity confer protection against the syndrome (150).

## **5.7 THE ROLE OF DIET AND PHYSICAL ACTIVITY**

Intensive interventions combining drug therapy and behaviour modification have shown sustained benefits in respect vascular complications and mortality rates in high risk DM patients (151). Behaviour modification encourages increased levels of physical activity coupled with appropriate dietary choices. Participants identified as hypertensive and diabetic both reported receiving education on diet and exercise at PHC facilities. This did not translate into improved consumption of fruit and vegetables or increased levels of physical activity. This could be due to the patients not heeding the advice given or perhaps, the distinction between health education and health promotion on the part of the health services needs to be clarified.

The Finnish Diabetes Prevention Study showed that moderate-vigorous activity and low intensity activity both resulted in reduced incidence of DM in a dose-dependent manner and independent of changes in diet and BMI (51). Data from the US based Diabetes Prevention Programme also showed that moderate activity for 30 minutes/day coupled with a 10% reduction in body weight translated to a 50% reduction in diabetes in high-risk IGT patients (131). Despite the convincing evidence to the contrary, this study did not demonstrate any impact of reported levels of physical activity on disease development or control. This may be due to inaccurate reporting of physical activity, as the questionnaire did call for recall of activities over a period of time. Few reported high activity levels, and it is likely that the majority were not engaging in physical activity.



Four participants (4/330, 1%) report never eating fresh fruit and vegetables at all while 61% report consumption five or more times per week (200/330). This information about diet is not associated with disease development or control. Data regarding intake of proteins (e.g. red meat), starch saturated fats is not available and might be the reason for the fresh fruit and vegetable consumption not being significant. This is an important concept as few diabetic patients (58%) identified maize as a food with high carbohydrate content despite it being a staple food in South Africa (50). It is suggested that this lack of knowledge compounded by poor dietary choices could predict for co-morbidities later (50).

## **5.8 SOCIO-DEMOGRAPHIC FACTORS**

### **5.8.1 Gender and race**

The study comprises 337 participants; 336 of whom are black. Identifying the study population as black is an important component of this study. Previous studies report that apart from the rural-urban gradient, there exists a gradient across population groups with DM being highly prevalent in the Indian community, followed by the coloured and then black populations (53) but the prevalence is growing in the black communities with ageing and lifestyle changes (50). Epidemiological information among the black population in Southern Africa reflects not just an increase in DM rates, but in other chronic diseases as well that are known to be more prevalent in the urban black populations (152). It is the measure of this change in South Africa that needs to be ascertained. Another important consideration in terms of race is that 90% of the black South African diabetic population access public health facilities with only 8% able to access private health care (50). Thus, this data should be able to provide an accurate estimate of the care received by those attending PHC facilities.

The majority of participants are female (213/337, 63%). The predominance of the female participants in the study population (63%) is in keeping with previous South African studies assessing chronic diseases, particularly DM, in the public clinics and the community (50;153). The apportionment of the study by gender is important because women are known to have better health seeking behaviour and the study sample is representative of what would be expected in a PHC facility.

### **5.8.2 Age and educational level**

There is a wide variation in the ages of those interviewed with a median age of 44 years (IQR 30-56) and a range of 18 – 93 years. The wide age band is an important consideration in self monitoring of chronic diseases. Research has shown that there is a 3% reduction in the knowledge score of diabetics for every ten year increase in age (50) so this is likely to explain poor levels of control in the older participants. Moodley and Rambiritch showed that the highest levels of diabetes knowledge was in the 40-59 year age group, which is the majority of this study population, and the lowest among the 60-79 year old patients in a Kwa-Zulu Natal clinic survey assessing DM knowledge among patients (50).

Many participants have attended high school (124/334, 37%) with 26% (88/334) having a matriculation certificate. A poor understanding of DM has been suggested as the reason for the poor glycaemic control in the black population (136). This may be attributable to the inequalities of education under apartheid that saw the white population having better facilities in both the private and public sector and a system of education that permeated through the clinics, schools and media (50). Most of this study population is older and have been schooled prior to the political changes of 1994, and thus subject to poor standards of education.

While only a small number received no formal education (9/334, 3%), it is important to consider the impact of this on disease development. Schutte and Olckers show in a study comparing South African women that unschooled black women have a 4.8 times higher risk of having the MS than schooled women (29). If the well documented inverse relationship between education levels and the development of CVD events hold true (154), it is not unanticipated to have high levels of disease in this study population.

### **5.8.3 Employment status**

The unemployment rate in the study population is high (168/337, 50%) with 56% of females unemployed. While unemployment does not automatically equate to a sedentary lifestyle, it is likely to be so and may afford an explanation as to the high levels of obesity particularly among females. Twenty percent of females (42/213) and 37% of males (46/124) are employed.

### **5.8.4 Social grants remittances**

Females are mostly dependant on the child support grants (47/88, 53%) and the median age of the women accessing these grants is 32 years (IQR 29-40). The introduction of the child support grant by the South African government in 1998 was intended as a poverty-alleviation measure though the poorest households are unlikely to access them (155). The SES of this community is middle to high income, and many have recourse to these grants (155).

## **5.9 PUBLIC HEALTH IMPLICATIONS OF THIS STUDY**

The public health implications of this study extend beyond the clinical diagnosis and management of this cluster of diseases. This section is presented considering the ‘Grand Challenges in Chronic Non-communicable Diseases’ that were formulated to reduce the burden of disease through finance, debate and evidence based guiding policies (156;157).

### **5.9.1 Raising public awareness**

Public education and awareness has been shown to impact on disease control and development. Health education needs to be encouraged in schools and is essential in all health care facilities. The message that is conveyed needs to be culturally sensitive and in an appropriate language and medium of communication to reach the entire community and not just the clinics and schools. For a public health intervention to be successful, the programme must simultaneously account for all CVD risk factors in an integrated, intervention involving co-ordination from policy makers, mass media messaging and commitment from the food industry to reduce the salt content of staple foods and provide correct labelling of food through policy and legislative development (57).

Interventions aimed at chronic disease prevention can take years to yield benefits. Changing behaviours is difficult and requires time, effort and considerable motivation. Perhaps these would be more readily achieved if the process was initiated through clinically based primary care treatments targeting those at risk for chronic diseases, particularly CVD and DM (19).

### **5.9.2 Modifying risk factors**

Comprehensive community-based lifestyle interventions, even in the developing countries, can alter the environment of the entire community to support healthier lifestyle choices which would translate to limiting health risk behaviours and ultimately, reducing the morbidity and mortality attributable to NCDs (20).

Enabling environments for such changes must be encouraged in schools, clinics and the community such that the entire community is reached, irrespective of whether they have a disease. Strategies such as increased availability of health foods, especially at schools; promoting physical activity and addressing misperceptions about weight have a vital role to play in reducing burden of disease.

### **5.9.3 Generation of accurate information**

National, provincial and local government, together with policy-makers has a pivotal role in the generation of information to help guide the development of preventative and treatment programmes, especially for those living in poverty. Early identification of those at risk and those with the disease through the generation of current, accurate and reliable data will serve as the first step in the process of effective management. Besides informing policies, surveillance data plays a vital role in evaluating health policies and preventative interventions. The information collected must be analysed and fed back into the health system to advise policy and allow for priority setting tailored to the needs of the community. It is vital that chronic disease surveillance be more comprehensively

integrated into the district and national health information systems with regular monitoring of the data guiding policy. To achieve this, it is imperative that the initiative secure broad political commitment.

#### **5.9.4 Reorientating the health system**

The health system, in many ways, has several constraints to the delivery of chronic disease services. In order to achieve improved quality and access to services, all the building blocks of the health system including service delivery, workforce, informatics, equipment and technologies, finance and leadership need to be strengthened (33;102;158). The chronic care model needs to be developed at primary health care level incorporating adequate training of health care workers and support staff, provision of the necessary equipment and maintaining a supply of appropriate drug therapy.

Besides political will and dedicated non-governmental organisations, the success of NCD programme is incumbent on the health care worker (35). The ‘brain drain’ deprives Africa of invaluable and medical and technical management and support (35). Having skilled and motivated medical and support staff in sufficient numbers, particularly in areas where access poses a significant problem, is intrinsic to delivering appropriate health services and ultimately improving health outcomes (159). The patient-provider relationship has been emphasised with patients who are actively engaged by the health care worker in terms of treatment, results of measurements and patient education having better health outcomes. Self management must form an integral part of quality primary

care for chronic diseases and this can only be achieved through comprehensive education and involvement.

## **5.10 LIMITATIONS**

The study was affected by the following limitations:

- a) Study participation depended on the availability of members of the community within the allocated times of the study period. The results, as such, are not generalizable to the greater community outside of this group.
- b) Participants refusing to have blood samples drawn have affected the sampling strategy as they were excluded from the study on this basis.
- c) All behaviours and practices were self-reported by the study participants. There was no means of verifying the data collected. The information reported was also subject to recall bias. Some areas covered by the questions were highly sensitive. Though the interviewers were trained to deal with this issue, this did not guarantee open and honest answers.
- d) Blood samples drawn were not fasting samples. Abnormal results warranted clinic referrals for either further investigation or for formal fasting blood samples if these were necessitated.



- e) The blood collected for the lipid profile testing had been incorrectly stored and the laboratory was unable to analyse these samples. HbA<sub>1c</sub> was used as a surrogate for fasting plasma glucose samples given resource constraints to the project. Eleven samples drawn for HbA<sub>1c</sub> testing were unaccounted for by the laboratory. As a result of this, the prevalence of the MS considering the cholesterol criteria (9) could not be ascertained. It is likely that the prevalence data of the MS presented in this study is under-estimated because of this.
- f) Some variables (e.g. Education levels of the participant, alcohol consumption) were dichotomized to simplify the statistical analysis and allow for ease of interpretation and presentation of results. However, it is appreciated that in dichotomizing variables, much information could be lost, so the statistical power to detect a relation between the variable and outcome is reduced.
- g) HIV testing was not conducted and no questions regarding the use of HAART were asked. This was not done because of the concern that people would decline participation as a result of the stigma associated with HIV testing and this would affect the sampling strategy. In doing so, the disease of burden attributable to the complex interaction between HIV and CVD could not be estimated.

## **5.11 SUMMARY OF THE CHAPTER**

The analysis describes the huge, and probably underestimated, burden of chronic diseases in the Johannesburg Health District, South Africa. Low levels of education have consistently been shown to be associated with disease. Females have emerged as a high risk priority group with levels of obesity exceeding 50%. Community-based screening programmes have been shown to be an effective tool in detecting disease in minority and low income populations. The findings of this study support the screening of cardio-metabolic diseases from as early as 30 years of age in males and 40 years of age in females.

## **CHAPTER 6**

### **CONCLUSIONS AND RECOMMENDATIONS**

This chapter summarizes the key findings of the study. Possible recommendations are then discussed.

#### **6.1 CONCLUSIONS**

The growing prevalence of cardio-metabolic diseases continue to pressurise limited health finances, to the point where their collective impact cannot be ignored (8). Health care workers and policy makers alike are facing what is tantamount to an epidemic. The findings of this study indicate that the extent of the burden of chronic diseases in the Johannesburg Health District has been grossly underestimated.

##### **6.1.1 Objective 1**

**To describe the study population in terms of socio-demographic factors**

The study population is predominantly black with most participants being female (63%). The median age was 44 years (IQR 30-56). Most participants are unemployed (50%) and most had less than Grade 12 education (59%). Thirty one percent of the study population is dependent on social remittances from the government as a source of income.

### **6.1.2 Objective 2**

**To explore the prevalence and risk factors associated with the development of the following cardio-metabolic diseases:**

- a. Diabetes mellitus**
- b. Hypertension**
- c. Obesity**

Female gender is highly associated with being overweight and obese. The development of the DM, HPT and obesity is strongly associated with lower levels of education.

#### **a. Diabetes mellitus**

The prevalence of DM is 14% (47/326) with similar distributions amongst males and females. Most diabetics are poorly controlled (66%). Almost 20% of participants interviewed are classified as pre-diabetic.

#### **b. Hypertension**

The prevalence of HPT is 58% (197/337) with similar distribution in females (60%) and males (56%). There is a high number (112/220, 51%) of ‘prehypertensive’ participants. Most diagnosed with HPT are poorly controlled (61%) with only 5% recording BP within the normal range.

### **c. Obesity**

Thirty percent of participants (101/337) are classified as overweight in this study. The levels of obesity is marked (133/337, 39%) with predominance among females (114/213, 54%).

### **6.1.3 Objective 3**

#### **To examine the associations among these major risk factors**

The diseases discussed are risk factors for the development of other cardio-metabolic diseases (e.g. a previous history of HPT has been associated with DM). This demonstrates that these diseases are very much linked and draws attention to the clinical value in screening for all cardio-metabolic diseases. An increase in the BMI is associated with an increase in both SBP and DBP but the rate of increase is not consistent. Removal of outlier values yielded the same relationship implying the generalizability of the study findings. BMI values are also associated with an increase in HbA<sub>1c</sub> levels – an association that improved from 1.45% to 12% with removal of the outlying values of HbA<sub>1c</sub>. The outliers represented older, married individuals of both genders with lower educational levels and a clinical history of DM and HPT.

#### **6.1.4 Objective 4**

**To determine the community prevalence of the MS and the major risk factors that predisposes individuals in this community to the development of the MS (as defined in Chapter 3: 3.6.1.2 Clinical variables (Metabolic syndrome))**

The prevalence of the MS in the study population is 31%. One hundred and ninety five females (92%) and 64 males (52%) met the IDF criteria for WC. On multivariate analysis, female gender and a previous history of DM are strongly associated with the development of the MS. Increasing SBP and WC was also implicated in the development of the MS.

## **6.2 RECOMMENDATIONS**

### **6.2.1 Engage the community in health promotion strategies**

Raising public awareness among the community influences early diagnosis, brings risk factors to the fore and allows the families and communities of the patients to participate more actively in the patients' management. These strategies need to extend beyond the clinic environment.

Health messages that are developed should be culturally appropriate and reach the community through a wide array of media including television, radio and print. Encouraging behaviour change can prove quite challenging. Community programmes can be used to provide messaging on safe and effective methods for prevention and management of diseases including walking, jogging and ball games. Having said this, environments would need to be created to allow for these activities to occur safely. Nutrition and physical activity needs to be integrated into the core curriculum at schools and food snack sold or provided should be nutritious. Community or school food gardens are an ideal way to ensure healthy foods while engaging large sectors of the community.

Understandably, the presence of the Changing Diabetes<sup>®</sup> Bus in the community piqued the interest of the residents. All residents who wished to be tested received a simple screening on the bus. Apart from receiving health education, the residents had their BP, random glucose and BMI measured. The findings were explained to them and were accompanied by a clinical referral in the case of abnormal findings. This implies, to some

degree, the level of interest that the community has in knowing and understanding their health status and alludes to the success that future screening programmes of this nature may enjoy.

### **6.2.2 Identify females as a high priority group**

Females need to be targeted primarily for future interventions. The data suggests that females have a disproportionately higher risk of developing associated CVD risk factors. Further, the high levels of obesity among women is confirmed by this study and substantiated by preceding studies. Females are known to have better health seeking behaviour patterns than males. Thus, every opportunity that a female presents herself to the health services should represent an opportunity to screen and educate her about these diseases but more importantly, to reinforce positive lifestyle choices.

### **6.2.3 Strengthening the health system**

The population of Chiawelo appears to be in an epidemiological transition. As such, greater emphasis needs to be placed on screening programmes and follow up, as well as endorsing lifestyle modification to avert the development of these chronic metabolic conditions. Integration of health services are needed at clinic level so that the individual cardio-metabolic diseases are not addressed in isolation. This can be facilitated through education and training of health care workers and clinic personnel to optimally diagnose and manage these conditions as a single entity. Cost-effective interventions tackling



obesity through improved diets and increased physical activity need to form part of a package addressing chronic diseases (38).

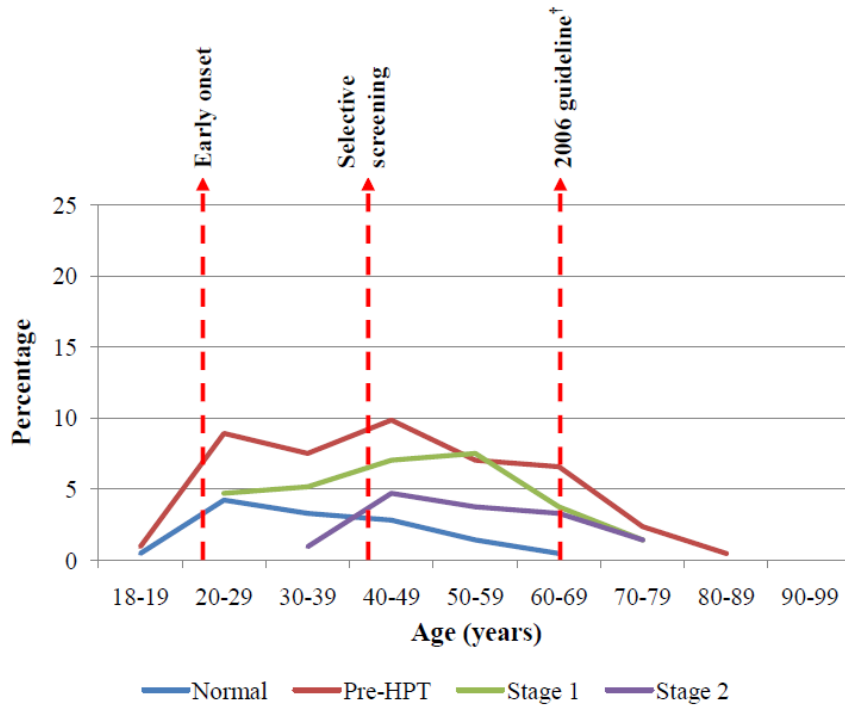
Low levels of education have consistently been linked to all chronic diseases within the scope of this report. To the patient, understanding the disease process gives more meaning to the management and promotes a greater desire to be involved in treatment strategies. The literature shows that health care workers engaging patients result in better health outcomes.

#### **6.2.4 Clinical implications on screening practices**

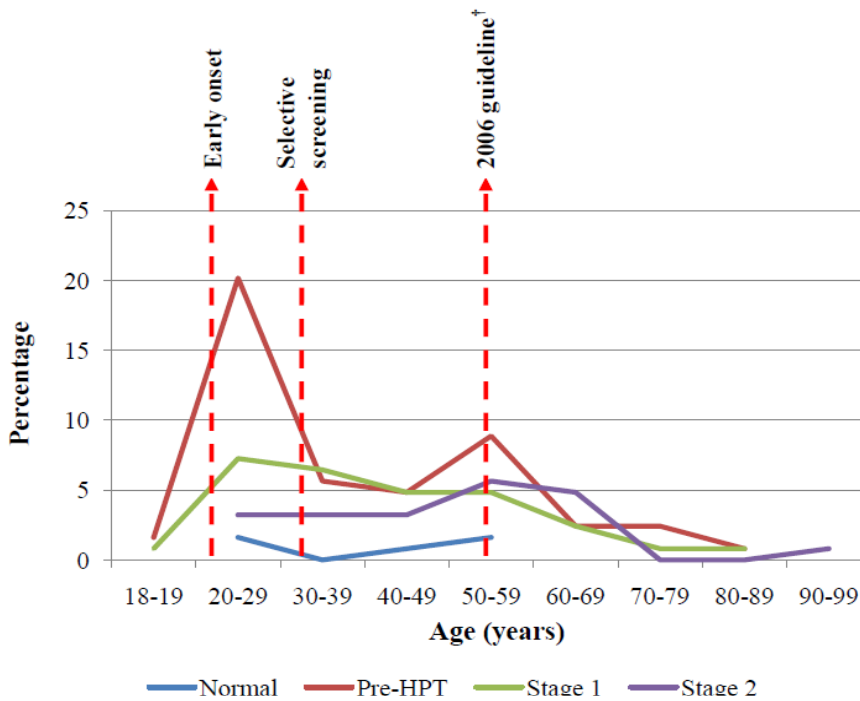
BP measurements reaching levels of prehypertension or above (SBP  $\geq$  130 mmHg and/or DBP  $\geq$  85 mmHg) requires treatment including weight loss and exercise followed by drug therapy after an appropriate trial of lifestyle modification (97). This statement has massive ramifications for this population as this study found prehypertension levels of 51%. Given the median ages described in Table 4.10, selective screening for HPT in this population should occur as early as 40 years in females [Figure 6.1(a)] and 30 years in males [Figure 6.1(b)]. Hypertension was identified among both males and females as young as 20-29 years of age. It will not prove cost effective to reduce the screening age to this level, but perhaps factors identified on multivariate analysis in this population (weight) could be considered together with clinical presentation and the presence of other recognised risk factors (97) when deciding whether to screen for HPT or not.

When considering DM, there does not appear to be a prescribed age at which screening is recommended. The findings of this study point to selective screening for DM as early as 50 years of age in females [Figure 6.2(a)] and 55 years of age in males [Figure 6.2(b)]. As with HPT, both genders presented as early as 20-29 years of age with IGT and DM. Screening for a previous diagnosis of HPT would be an additional consideration for screening at this age group, over and above the presence of clinical judgment and recognised risk factors.

**a. Females**



**b. Males**



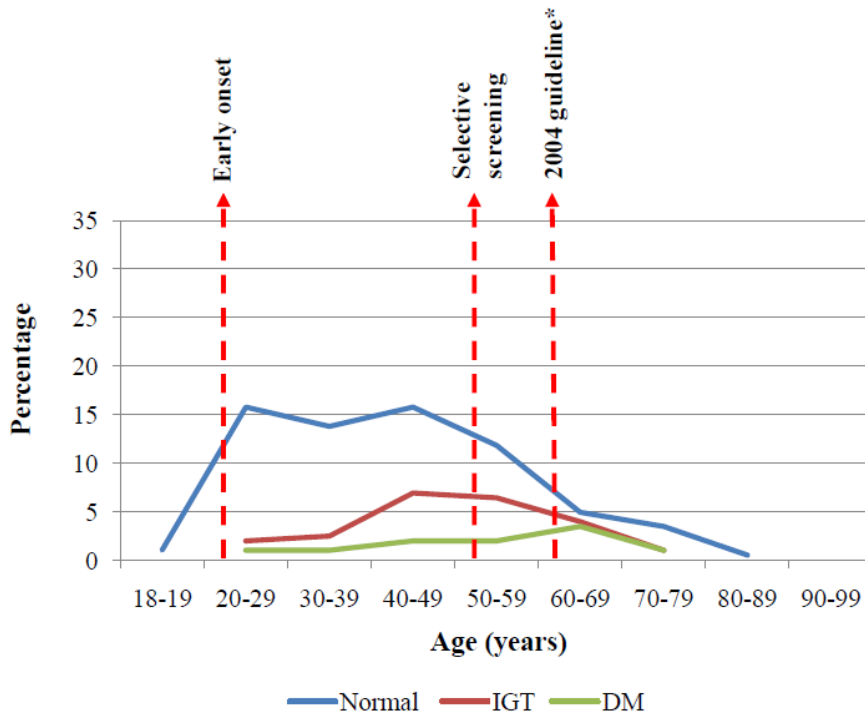
†South African Hypertension Guideline 2006

**Figure 6.1: Prevalence of HPT identifying target ages for screening**

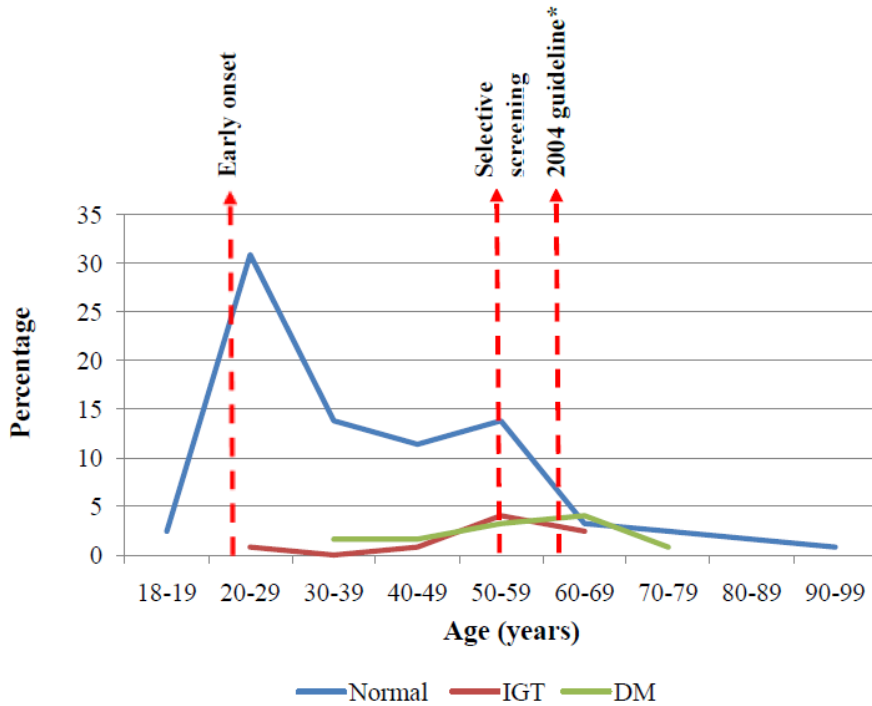
People with chronic diseases of lifestyle such as HPT, DM and CVD have long been identified as high risk target groups for primary and secondary prevention of obesity (64). This study identified 47 years of age in females and 49 years in males as selective screening ages for being overweight. This is a guide only and patients should be screened based on clinician presentation. Obesity was further identified as a particular concern in the study population with 54% of females being classified as obese.

Community-based screening programmes have been shown to be an effective tool in detecting disease in minority and low income populations (160). The findings of this study support the screening of cardio-metabolic diseases from as early as 30 years of age in males and 40 years of age in females.

**a. Female**



**b. Male**



\*National Programme for the control and management of diabetes type 2 at primary level (96)

**Figure 6.2: Prevalence of DM identifying target ages for screening**

### **6.2.5 Improving screening and surveillance strategies**

Clinical decisions and policy implementation is guided by the ‘local’ data on disease prevalence. It is thus imperative that this information be as accurate as possible. Community-based screening programmes have been shown to be an effective tool in detecting disease in minority and low income populations. While remaining cognisant of the current HPT and DM guidelines in use in South Africa, it is the findings of this study to support the screening of the cardio-metabolic diseases from as early as 30 years of age in males and 40 years of age in females.

Prior to initiating a system of surveillance, buy-in by those tasked with collecting the information would be advisable, as this would assist with sustainability of the intervention. The tools used for this purpose should be easy to understand, simple to complete and allow for comparison between clinics and provinces alike. The use of the WHO STEPwise tool is a useful starting block and should be modified in according to the local requirements of a surveillance programme. The tool can be implemented at a single clinic and be extended to other clinics, all the while identifying strengths and weaknesses of implementation and acting on these accordingly.

Data collected over time allows for analysis of trends and helps assess the effectiveness of interventions. The clinic staff that collects the data must be privy to the findings and witness the translation to clinic practice through policy evaluation and changes. In doing so, they will remain motivated to collect the data, as they will be able to track the positive, or negative, influence of their service provision.

Apart from improvements in clinical management, the surveillance data should identify gaps in the quality of service at PHC facilities. The additional value of this data is that it would guide health care worker training and subsequently, service provision. The training should be conducted regularly.

#### **6.2.6 Further research**

The following research areas are vital as the findings would provide insight into the screening, management and surveillance of NCDs particularly pertaining to South Africa:

- a. Evaluation of the clinical and cost effective utilisation of resources dedicated to the care of patients with NCDs and the use of these to direct preventative measures
- b. Identification and the assessment of the most cost-effective and culturally appropriate strategies tackling unhealthy diets, physical inactivity and obesity in South Africa.
- c. Investigation into the impact and effectiveness of food labelling
- d. Evaluation of strategies to integrate the management of NCDs and communicable diseases
- e. Understanding the development and provision of culturally appropriate resources with which to train health care-workers
- f. Determining the levels of physical activity among Gauteng residents using a standardized tool

- g. Understanding the effects of malaria, iron-deficiency anaemia, HIV and HAART on HbA<sub>1c</sub> levels among the various ethnic groups of South Africa
- h. Assessment of the Metabolic Syndrome in the Johannesburg Health District



### **6.3 SUMMARY AND CONCLUSIONS**

The global prevalence of NCD is increasing with much of the burden attributable to urbanisation and poor lifestyle choices. South Africa suffers a similar fate. In South Africa, optimal management extends beyond the disease with individual and health system factors impacting considerably of delivery and use of services. This study was a community based cross sectional survey conducted in Chiawelo, Soweto in the Johannesburg Health District. The findings of this study indicate that the extent of the burden of chronic cardio-metabolic diseases in the Johannesburg Health District has been grossly underestimated. The prevalence of HPT and DM are high, with poor control and obesity levels are reaching epidemic proportions, particularly among females. Low levels of education seem to a recurring factor in disease development. Countering the burden of chronic disease involves the identification and targeting of females as a high risk priority group, engaging the patients and community alike in health promotion programmes and developing an adequate, accurate and reproducible system of NCD surveillance. The entire process hinges on the strengthening of the health system through retraining of staff, sensible following of clinical guidelines and health care workers engaging patients in their clinical management. Clinically, it is the findings of this study to support the screening of cardio-metabolic diseases from as early as 30 years of age in males and 40 years of age in females.

## REFERENCES

- (1) American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care* 2010;33:S62-S69.
- (2) Butler N. National Guidelines at a glance: Hypercholesterolaemia. *South African Pharmaceutical Journal* 2009;7:28-33.
- (3) No authors listed. Diagnosis, management and prevention of the common dyslipidaemias in South Africa--clinical guideline, 2000. South African Medical Association and Lipid and Atherosclerosis Society of Southern Africa Working Group. *South African Medical Journal* 2000;90:164-78.
- (4) Unwin N, Mugusi F, Aspray T, Whiting D, Edwards R, Mbanya JC, et al. Tackling the emerging pandemic of non-communicable diseases in sub-Saharan Africa: The essential NCD health intervention project. *Public Health* 1999;113:141-6.
- (5) Gaziano TA. Cardiovascular Disease in the Developing World and Its Cost-Effective Management. *Circulation* 2005;112:3547-53.
- (6) National Institutes of Health. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. US Department of Health and Human Services; 2004.
- (7) Reaven GM. Role of Insulin Resistance in Human Disease (Syndrome X): An Expanded Definition. *Annual Review of Medicine* 1993;44:121-31.
- (8) Smyth S, Heron A. Diabetes and obesity: twin epidemics. *Nature Medicine* 2005;12(1):75-80.
- (9) Alberti G, Zimmet P, Shaw J, Grundy SM. The IDF consensus worldwide definition of the Metabolic Syndrome. International Diabetes Federation; 2006.
- (10) Garrow JS, Webster J. Quetelet's index (W/H<sup>2</sup>) as a measure of fatness. *International Journal of Obesity* 1985;9(2):147-53.
- (11) WHO. Global Database on Body Mass Index. WHO . 2006.  
Ref Type: Internet Communication
- (12) Bonita R, de Courten M, Dwyer T, Jamrozik K, Winkelmann R. Surveillance of risk factors for noncommunicable diseases: The WHO STEPwise approach. Summary . Geneva, Switzerland: World Health Organisation; 2001.
- (13) Mbanya JC, Squire SB, Cazap E, Puska P. Mobilising the world for chronic NCDs. *Lancet*. In press 2010.
- (14) UNESCO. South Africa. Millennium Development Goals. Mid-term Country Report. 2007.

- (15) The NCD Alliance. NCD Alliance Plan for the United Nations High Level Summit on Non- Communicable Diseases. 2010.
- (16) Steyn K, Bradshaw D. Non-communicable disease surveillance in developing countries. *Scandinavian Journal of Public Health* 2001;29:161-5.
- (17) WHO. Global strategy for the prevention and control of non-communicable diseases. Fifty-third World Health Assembly: World Health Organization; 2000. Report No.: A53/14.
- (18) Prevention and control of non-communicable diseases, Resolution 64/265, United Nations General Assembly, (2010).
- (19) Yach D, Leeder SR, Bell J, Kistnasamy B. Editorial: Global Chronic Diseases. *Science* 2005;307:317.
- (20) Sarrafzadegan N, Kelishadi R, Esmailzadeh A, Mohammadifard N, Rabiei K, Roohafza K. Do lifestyle interventions work in developing countries? Findings from the Isfahan Healthy Heart Program in the Islamic Republic of Iran. *Bulletin of the World Health Organisation* 2009;87:39-50.
- (21) Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. *Lancet* 2005;365:1415-26.
- (22) Unwin N, Setel P, Rashid S, Mugusi F, Mbanya JC, Kitange H. Noncommunicable diseases in sub-Saharan Africa: where do they feature in the health research agenda? *Bulletin of the World Health Organization* 2001;79(10):947-53.
- (23) Chockalingam A. Cardiovascular Diseases India: Let's not make the same mistakes. *The Lancet* 2000;356:S9.
- (24) Rosenzweig JL, Ferrannini E, Grundy SM, Haffner SM, Heine RJ, Horton ES, et al. Primary Prevention of Cardiovascular Disease and Type 2 Diabetes in Patients at Metabolic Risk: An Endocrine Society Clinical Practice Guideline. *Journal of Clinical Endocrinology and Metabolism* 2008;93(10):3671-89.
- (25) Coovadia H, Jewkes R, Barron P, Sanders P, McIntyre D. The Health and Health System of South Africa: Historical Roots of Current Public Health Challenges. *Lancet* 2009;374:817-34.
- (26) Beaglehole R, Horton R. Chronic diseases: global action must match global evidence. *Lancet* 2010;376:1619-21.
- (27) Mayosi BM, Flisher AJ, Lalloo UG, Sitas F, Tollman SM, Bradshaw B. The burden of non-communicable diseases in South Africa. *Lancet* 2009;374:934-47.

- (28) Alberts M, Urdal P, Steyn K, Stensvold I, Tverdal A, Nel JH, et al. Prevalence of cardiovascular diseases and associated risk factors in a rural black population of South Africa. *European Journal of Cardiovascular Prevention and Rehabilitation* 2005;12(4):347-54.
- (29) Schutte AE, Olckers A. Metabolic Syndrome Risk in Black Women Compared to Caucasian Women. *Hormone and Metabolic Research* 2007;39:651-7.
- (30) Ntintyane LM, Panz VR, Raal FJ, Gill GV. Post prandial lipaemia, metabolic syndrome and LDL particle size in urbanised South African blacks with and without coronary artery disease. *QJM: An International Journal of Medicine* 2008;101:111-9.
- (31) WHO. 2008 - 2013 Action Plan for the Global Strategy for the Prevention and Control of Noncommunicable Diseases. Geneva: World Health Organisation; 2008.
- (32) Alwan A, MacLean DR, Riley LM, d'Espaignet ET, Mathers CD, Stevens GA, et al. Monitoring and surveillance of chronic non-communicable diseases: progress and capacity in high-burden countries. *Lancet* 2010;376:1861-8.
- (33) Samb B, Desai N, Nishtar S, Mendis S, Bekedarn H, Wright A, et al. Prevention and management of chronic disease: a litmus test for health-systems strengthening in low-income and middle-income countries. *Lancet* 2010;376:1785-97.
- (34) Tibazarwa K, Ntintyane LM, Sliwa K, Gerntholz T, Carrington M, Wilkinson D, et al. A time bomb of cardiovascular risk factors in South Africa: Results from the Heart of Soweto Study "Heart Awareness Days". *International Journal of Cardiology* 2009;132:233-9.
- (35) Opie LH, Mayosi BM. Cardiovascular Disease in Sub-Saharan Africa. *Circulation* 2005;112:3536-40.
- (36) Steyn K, Fourie N, Temple N. *Chronic Diseases of Lifestyle in South Africa: 1995 - 2005*. Cape Town: South African Medical Research Council; 2006.
- (37) du Toit G, van der Merwe M-T. The epidemic of childhood obesity. *South African Medical Journal* 2003;93(1):49-50.
- (38) Cecchini M, Sassi F, Lauer JA, Lee YY, Guajardo-Barron V, Chisholm D. Tackling of unhealthy diets, physical inactivity, and obesity: health effects and cost-effectiveness. *Lancet* 2010;376:1775-84.
- (39) Abegunde DO, Mathers CD, Adam T, Ortegón M, Strong K. The burden and costs of chronic diseases in low-income and middle-income countries. *The Lancet* 2007;370:1929-38.
- (40) Meusel D, Hoyer C, Perez-Rodrigo C, Aranceta J, Cavill N, Armstrong T. A framework to monitor and evaluate implementation: Global strategy on diet, physical activity and health. Geneva: WHO; 2008.

- (41) Bradshaw D, Groenewald P, Laubscher N, R Nannan, Nolijsana B, Norman R. Initial burden of disease estimates for South Africa, 2000 . South African Medical Journal 2003;93:682-8.
- (42) Steyn K, Sliwa K, Hawken S, Commerford P, Onen C, Damasceno A. Risk factors associated with myocardial infarction in Africa: the INTERHEART Africa Study. Circulation 2005;112:3554-2561.
- (43) Beaglehole R, Irwin A, Prentice T. World Health Report 2003: Shaping the Future. Geneva, Switzerland: World Health Organization; 2003.
- (44) Pestana JA, Steyn K, Leiman A, Hartzenburg GM. The direct and indirect costs of cardiovascular disease in South Africa in 1991. South African Medical Journal 1996;86:679-84.
- (45) Steyn K, Katzenellenbogen J, Bradshaw D, Lombard C, Bourne LT. Urbanisation and the risk for chronic diseases of lifestyle in the black population of the Cape Peninsula. Journal of Cardiovascular Risk 1997;4:135-42.
- (46) Levitt NS, Katzenellenbogen J, Bradshaw D, Hoffman MN, Bonnici F. The prevalence and identification of risk factors for NIDDM in urban Africans in Cape Town, South Africa. Diabetes Care 1993;16(4):601-7.
- (47) Gaillard T, Schuster D, Osei K. Metabolic Syndrome in Black people of the African diaspora: The paradox of current classification, definition and criteria. Ethnicity & Diseases 2009;19:S2-1-S2-7.
- (48) Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care 2004;27(5):1047-53.
- (49) Mbanya J, Motala AA, Sobgnwi E, Assah FK, Enoru ST. Diabetes in sub-Saharan Africa. Lancet 2010;375:2254-66.
- (50) Moodley LM, Rambiritch V. An assessment of the level of knowledge about diabetes mellitus among diabetic patients in a primary health care setting. South African Family Practice 2007;49(10):16-20.
- (51) Gill JMR, Cooper AR. Physical Activity and Prevention of Type 2 Diabetes Mellitus. Sports Medicine 2008;38(10):807-24.
- (52) Fox CS. Cardiovascular Disease Risk Factors, Type 2 Diabetes Mellitus, and the Framingham Heart Study. Trends in Cardiovascular Medicine 2010;20:90-5.
- (53) Bradshaw D, Norman R, Pieterse D, Levitt NS. Estimating the burden of disease attributable to diabetes in South Africa in 2000. South African Medical Journal 2007;97(7):700-6.

- (54) Rheeder P. Type 2 diabetes: the emerging epidemic. *South African Family Practice* 2006;48(10):20.
- (55) Mash RJ, De Vries E, Abdul I. Diabetes in Africa: the new pandemic. Report on the 19th World Diabetes Congress, Cape Town, December 2006. *South African Family Practice* 2007;49(6):44-50.
- (56) Elgoni A, Hunter J, Kaye-Petersen E, Naidu V. Health in Gauteng. Status Report 2006 / 2007. Johannesburg: Gauteng Department of Health; 2008.
- (57) Norman R, Gaziano T, Laubscher R, Steyn K, Bradshaw D. Estimating the burden of disease attributable to high blood pressure in South Africa in 2000. *South African Medical Journal* 2007;97(8):692-8.
- (58) Dennison CR, Peer N, Steyn K, Levitt NS, Hill MN. Determinants of Hypertension Care and Control among Peri-Urban black South Africans: The HiHi Study. *Ethnicity & Disease* 2007;17:484-91.
- (59) Ntintyane LM, Panz VR, Raal FJ, Gill GV. Metabolic syndrome, undiagnosed diabetes mellitus and insulin resistance are highly prevalent in urbanised South African blacks with coronary artery disease. *Cardiovascular Journal of South Africa* 2006;17(2):50-5.
- (60) Despres JP. Health consequences of visceral obesity. *Annals of Medicine* 2001;33:534-41.
- (61) Puoane T, Steyn K, Bradshaw D, Laubscher R, Fourie N, Lambert V. Obesity in South Africa: the South African demographic and health survey. *Obesity Research* 2002;10(10):-1038.
- (62) Steyn NP. Managing childhood obesity: a comprehensive approach. *Continuing Medical Education* 2005;23(11):540-4.
- (63) Puoane T, Bradley H, Hughes G. Obesity among South African women. *Human Ecology Special* 2005;13:91-5.
- (64) Southern African Society for the Study of Obesity. Guidelines for the Prevention and Management of Overweight and Obesity in South Africa. 2003.
- (65) Vezi ZB, Naidoo DP. Dyslipidaemia among black patients with type 2 diabetes. *Cardiovascular Journal of South Africa* 2005;16(4):194-8.
- (66) Joffe BI, Distiller LA, Kalk WJ. Lipid levels in Black South Africans with Type 2 Diabetes: Dispelling misconceptions. *Diabetes Care* 2004;27(7):1839-40.
- (67) Norman R, Bradshaw D, Steyn K, Gaziano T. Estimating the burden of disease attributable to high cholesterol in South Africa in 2000. *South African Medical Journal* 2007;97(8):708-15.

- (68) Lock K, Smith RD, Dangour AD, Keogh-Brown M, Pigatto G, Hawkes C. Health, agricultural, and economic effects of adoption of healthy diet recommendations. *Lancet* 2010;376:1699-708.
- (69) Lakerveld J, Bot SDM, Chinapaw MJ, van Tulder MW, van Oppen P, Dekker JM. Primary prevention of diabetes mellitus type 2 and cardiovascular diseases using a cognitive behavior program aimed at lifestyle changes in people at risk: Design of a randomized controlled trial. *BMC Endocrine Disorders* 2008;8(6).
- (70) Prevention and Control of Noncommunicable Diseases: Implementation of the Global Strategy, Resolution WHA 61.14, World Health Assembly, (2008).
- (71) WHO. A Framework to Monitor and Evaluate Implementation: Global Strategy on Diet, Physical Activity and Health. Geneva: World Health Organisation; 2008.
- (72) WHO. Global Strategy on Diet, Physical Activity and Health. Geneva: World Health Organisation; 2004.
- (73) Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance: the Da Qing IGT and Diabetes Study. *Diabetes Care* 1997;20(4):537-44.
- (74) Ramachandran A, Snehalatha C, Mary S, Yamuna A, Ping Z. The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). *Diabetologia* 2006;49(2):289-97.
- (75) Appel J, Champagne CM, Harsha DW, Cooper LS, Obarzanek E, Elmer PJ. Effects of comprehensive lifestyle modification on blood pressure control: main results of the PREMIER clinical trial. *Journal of the American Medical Association* 2003;289:2083-93.
- (76) Elmer PJ, Obarzanek E, Vollmer WM, Simons-Morton D, Stevens VJ, Young DR. Effects of comprehensive lifestyle modification on diet, weight, physical fitness, and blood pressure control. *Annals of Internal Medicine* 2006;144:485-95.
- (77) Schneider M, Norman R, Steyn N, Bradshaw D. Estimating the burden of disease attributable to low fruit and vegetable intake in South Africa in 2000. *South African Medical Journal* 2007;97(8):717-23.
- (78) Katzmarzyk PT, Leon AS, Wilmore JH, Skinner JS, Rao DC, Rankinen T, et al. Targeting the Metabolic Syndrome with Exercise: Evidence from the HERITAGE Family Study. *Medicine & Science in Sports & Exercise* 2003;35(10):1703-9.
- (79) Cornier MA, Dabelea D, Hernandez TL, Lindstrom RC, Steig AJ, Stob NR. The Metabolic Syndrome. *Endocrine Reviews* 2008;29(777):822.

- (80) Assmann G, Schulte H, Seedorf U. Cardiovascular assessment in the metabolic syndrome: results from the Prospective Cardiovascular Munster (PROCAM) Study. *International Journal of Obesity* 2008;32:S11-S16.
- (81) Stern M, Williams K, Gonzalez-Villpando C, Hunt KJ, Haffner SM. Does the metabolic syndrome improve identification of individuals at risk of Type 2 diabetes and/or cardiovascular disease? *Diabetes Care* 2004;27(11):2676-81.
- (82) De Simone G, Devereux RB, Chinali M, Best LG, Lee ET, Galloway JM. Prognostic Impact of Metabolic Syndrome by Different Definitions in a Population With High Prevalence of Obesity and Diabetes: The Strong Heart Study. *Diabetes Care* 2007;30(7):1851-6.
- (83) Mattsson N, Ronnema T, Juonala M, Viikari JSA, Raitakari OT. The presence of the metabolic syndrome in young adults. The Cardiovascular Risk in Young Finns Study. *Journal of Internal Medicine* 2007;261:159-69.
- (84) Harrell JS, Jessup A, Greene N. Obesity and the Metabolic Syndrome in Children and Adolescents. *Journal of Cardiovascular Nursing* 2006;21(4):322-30.
- (85) Zimmet PZ, Alberti KGMM, Kaufman F, Tajima N, Silink M, Arslanian S, et al. The metabolic syndrome in children and adolescents. *Lancet* 2007;369:2059-61.
- (86) Hu G, Qiao Q, Tuomilehto J. Plasma insulin and cardiovascular mortality in non-diabetic European men and women: a meta-analysis of data from eleven prospective studies. The DECODE Insulin Study Group. *Diabetologia* 2004;47(7):1245-56.
- (87) Carr DB, Utzschneider KM, Hull RL, Kodama K, Retzlaff BM, Brunzell JD. Intra-Abdominal Fat Is a Major Determinant of the National Cholesterol Education Program Adult Treatment Panel III Criteria for the Metabolic Syndrome. *Diabetes* 2004;53(8):2087-94.
- (88) Pouliot MC, Despres JP, Lemieux S. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *The American Journal of Cardiology* 1994;73:460-8.
- (89) Zimmet P, Alberti KGMM, Shaw J. Global and societal implications of the diabetes epidemic. *Nature* 2001;414:782-7.
- (90) Carey VJ, Walters EE, Colditz GA. Body fat distribution and risk of non-insulin dependent diabetes in women: The Nurses' Health Study. *American Journal of Epidemiology* 1997;145:614-9.
- (91) Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *Journal of the American Medical Association* 2002;287:356-9.



- (92) Ford ES. Prevalence of the metabolic syndrome defined by the International Diabetes Federation among adults in the U.S. *Diabetes Care* 2005;28:2745-9.
- (93) Kalk WJ, Joffe BI. The Metabolic Syndrome, Insulin Resistance, and Its Surrogates in African and White Subjects with Type 2 Diabetes in South Africa. *Metabolic Syndrome and Related Disorders* 2008;6(4):247-55.
- (94) Steyn K, Levitt NS, Patel M, Fourie J, Gwebushe N, Lombard C. Hypertension and diabetes: Poor care for patients at community health centres. *South African Medical Journal* 2008;98(8):618-22.
- (95) Mash R, Levitt NS, Van Vuuren U, Martell R. Improving the annual review of diabetic patients in primary care: an appreciative inquiry in the Cape Town Health Services. *South African Family Practice* 2008;50(5):50a-d.
- (96) National Department of Health. National programme for control and management of diabetes type 2 at primary level. 2004.
- (97) Joint National Hypertension Guideline Working Group 2006, Seedat YK, Croasdale MA, Milne FJ, Opie LH, Pinkney-Atkinson VJ, et al. South African Hypertension Guideline 2006. *South African Medical Journal* 2006;96(4):337-62.
- (98) Gossain VV, Bowman KA, Rovner DR. The actual and self perceived knowledge of diabetes among staff nurses. *The Diabetes Educator* 1993;19(6):497-502.
- (99) Peyrot M, Rubin RR. Modelling the effect of diabetes education on glycaemic control. *The Diabetes Educator* 1994;20(2):143-8.
- (100) Assal J-P, Jacquement S, Morei Y. The Added Value of Therapy in Diabetes: The Education of Patients for Self-Management of Their Disease. *Metabolism* 1997;46(12):61-4.
- (101) WHO. The Innovative Care for Chronic Conditions (ICCC) Framework. Geneva: World Health Organisation; 2002.
- (102) Allotey P, Reidpath DD, Yasin S, Chan CK, de-Graft Aikins A. Rethinking health-care systems: a focus on chronicity. *Lancet*. In press 2010.
- (103) Dennison CR, Peer N, Lombard CJ, Kepe L, Levitt NS, Steyn K, et al. Cardiovascular risk and Comorbid conditions among Black South Africans with hypertension in public and private health care settings: The HiHi Study. *Ethnicity & Disease* 2007;17:477-83.
- (104) Steyn K, Bradshaw D, Norman R, Laubscher R. Determinants and treatment of hypertension in South Africa: The first Demographic and Health Survey. *South African Medical Journal* 2008;98(5):376-80.

- (105) Stamler J, Stamler R, Neaton JD, Wentowrth D, Daviglus ML, Garside D. Low risk-factor profile and long-term cardiovascular and noncardiovascular mortality and life expectancy: findings for 5 large cohorts of young adult and middle-aged men and women. *Journal of the American Medical Association* 1999;282(21):2012-8.
- (106) Engstrom G, Jerntorp I, Pessah-Rasmussen H, Hedblad B, Berglund, G, et al. Geographic Distribution of Stroke Incidence Within an Urban Population: Relations to Socioeconomic Circumstances and Prevalence of Cardiovascular Risk Factors. *Stroke* 2001;32(5):1098-103.
- (107) Magnus P, Beaglehole R. The real contribution of the major risk factors to the coronary epidemics: time to end the "only 50%" myth. *Archives of Internal Medicine* 2001;161(21):2657-60.
- (108) Timothy GA, Moorman J. Factors influencing glycaemic control in diabetics, in three community health centres, in the city of Johannesburg. Johannesburg: University of the Witwatersrand; 2008.
- (109) Epi Info [computer program]. Version 3.4.1 2007.
- (110) Basu D. Endometrial thickness in post-menopausal black women in Soweto: a study of associated factors. Johannesburg: University of the Witwatersrand; 2002.
- (111) National Diabetes Programme DEHKO. Type 2 Diabetes Risk Assessment Form. Finnish Diabetes Association . 2009.  
Ref Type: Internet Communication
- (112) Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986;1(8476):307-10.
- (113) Stoddard GJ. Biostatistics and Epidemiology Using Stata: A Course Manual. University of Utah School of Medicine, editor. 2010.  
Ref Type: Unpublished Work
- (114) Stata Statistical Software: Release 10 [computer program]. College Station, TX: StataCorp LP; 2007.
- (115) Medical Research Council (South Africa). Poverty and Chronic Diseases in South Africa. Cape Town; 2001.
- (116) Kriska AM, Caspersen CJ. Introduction to a Collection of Physical Activity Questionnaires. *Medicine and Science in Sports and Exercise* 1997;29(6):S5-S9.
- (117) Ainsworth BE, Haskell WL, Leon AS, Jacobs DR, Montoye HJ, Sallis JF, et al. Compendium of Physical Activities: classification of energy costs of human physical activities. *Medicine and Science in Sports and Exercise* 1993;25(1):71-80.

- (118) Action Plan 2014: Towards the realisation of Schooling 2025, Department of Basic Education, (2010).
- (119) Craig CL, Marshall AL, Sjöström M, Bauman AE, Booth ML, Ainsworth BE, et al. International physical activity questionnaire: 12-country reliability and validity. *Medicine and Science in Sports and Exercise* 2003;35(8):1381-95.
- (120) Zuma J. Statement of the National Executive Committee of the African National Congress on the occasion of the 99th Anniversary of the ANC. 2011.
- (121) Lohman TG, Roche AF, Martorell R. Anthropometric standardization reference manual. Champaign, IL: Human Kinetic Books; 1988.
- (122) MedWrench. Roche Diagnostics Cobas Integra. 2011.  
Ref Type: Internet Communication
- (123) Amod A. Position statement on the use of HbA1c assays for the diagnosis of diabetes mellitus. *Journal of Endocrinology, Metabolism and Diabetes of South Africa* 2010;15(2):71-3.
- (124) Dave JA, Weinrich C. Diagnosis of Diabetes in 2010. *Continuing Medical Education* 2010;28(10):470-3.
- (125) Nilsson PM, Engström G, Hedblad B. The metabolic syndrome and incidence of cardiovascular disease in non-diabetic subjects - a population-based study comparing three different definitions. *Diabetic Medicine* 2007;24:464-72.
- (126) Ntyintyane LM, Panz VR, Raal FJ. The metabolic syndrome using the National Cholesterol Education Program and International Diabetes Federation definitions among urbanised black South Africans with established coronary artery disease. *Journal of Endocrinology, Metabolism and Diabetes of South Africa* 2007;12(1):6-12.
- (127) Vundle Z. Report on Diabetes Surveillance in Gauteng Province: April 2005 - February 2008. 2008. Johannesburg.  
Ref Type: Unpublished Work
- (128) Butler N. National Guidelines at a glance: Type 2 Diabetes Mellitus. *South African Pharmaceutical Journal* 2009;11-12:32-43.
- (129) Klisiewicz AM, Raal F. Sub-optimal Management of Type 2 Diabetes Mellitus - A Local Audit. *Journal of Endocrinology, Metabolism and Diabetes in South Africa* 2009;14(1):13-6.
- (130) Motala AA, Pirie FJ, Esterhuizen T, Omar MAK, Gouws E. Diabetes and Other Disorders of Glycemia in a Rural South African Community: Prevalence and associated risk factors. *Diabetes Care* 2008;31(9):1783-8.

- (131) Joshi P, Joshi S. Type 2 diabetes: Primary health care approach for prevention, screening and diagnosis in South Africa. *South African Family Practice* 2008;50(4):14-20.
- (132) Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Ilanne-Parikka P. Prevention of Type 2 diabetes by changes in lifestyle among subjects with impaired glucose tolerance. *New England Journal of Medicine* 2001;344:1343-50.
- (133) Pani LN, Korenda L, Meigs JB, Driver C, Chamany S, Fox CS, et al. Effects of Aging on A1C Levels in Individuals Without Diabetes. Evidence from the Framingham Offspring Study and the National Health and Nutrition Examination Survey 2001-2004. *Diabetes Care* 2008;31:1991-6.
- (134) Gali RM, Mshelia DS, Mamza YP, Adeonote J. Is screening for type 2 diabetes worthwhile in adolescents and young adults in developing countries? *African Journal of Diabetes Medicine* 2009;11:12-4.
- (135) National Department of Health. Negotiated Service Delivery Agreement. 2010.
- (136) Levitt NS, Bradshaw D, Zwarenstein MF, Bawa AA, Maphumulo S. Audit of public sector primary diabetes care in Cape Town, South Africa: high prevalence of complications, uncontrolled hyperglycaemia, and hypertension. *Diabetic Medicine* 1997;14:1073-7.
- (137) Nthangeni G, Steyn NP, Alberts M, Steyn K, Levitt NS, Laubscher R, et al. Dietary intake and barriers to dietary compliance in black type 2 diabetic patients attending primary health-care services. *Public Health Nutrition* 2001;5(2):329-38.
- (138) van de Sande M, Dippenaar H, Rutten GEHM. The relationship between patient education and glycaemic control in a South African township. *Primary Diabetes Care* 2007;1:87-91.
- (139) Bodenheimer T, Lorig K, Holman H, Grumbach K. Patient Self-management of Chronic Disease in Primary Care. *Journal of the American Medical Association* 2002;288(19):2469-75.
- (140) Seedat YK. Hypertension in black South Africans. *Journal of Human Hypertension* 1999;13:96-103.
- (141) Steyn K, Levitt NS, Hoffman M, Marais AD, Fourie JM, Lambert EV, et al. The Global Cardiovascular Risk Pattern in a Peri-Urban Working-Class Community in South Africa. The Mamre Study. *Ethnicity & Disease* 2004;14:233-42.
- (142) South African Department of Health. South African Demographic and Health Survey 2003. Pretoria: South African Department of Health; 2003.

- (143) Joubert J, Norman R, Bradshaw D, Goedecke JH, Steyn NP, Puoane T. Estimating the burden of disease attributable to excess body weight in South Africa in 2000. *South African Medical Journal* 2007;97(8):683-90.
- (144) Guzder RN, Gatling W, Mullee MA, Byrne CD. Impact of the metabolic syndrome criteria on cardiovascular disease risk in people with newly diagnosed type 2 diabetes. *Diabetologia* 2006;49:49-55.
- (145) Lean ME, Powrie JK, Anderson AS, Garthwaite PH. Obesity, weight loss, and prognosis in type 2 diabetes. *Diabetic Medicine* 1990;7:228-33.
- (146) van der Merwe M-T. The importance and predictive value of BMI and waist circumference in the development of Type 2 Diabetes. *South African Family Practice* 2004;46(6):10-4.
- (147) Groenewald P, Vos T, Norman R, Laubscher R, van Walbeek C, Saloojee Y, et al. Estimating the burden of disease attributable to smoking in South Africa in 2000. *South African Medical Journal* 2007;97(8):674-81.
- (148) Schutte AE, Schutte R, Huismann HW, Rooyen JM, Malan L, Olckers A. Classifying Africans with the metabolic syndrome. *Hormone and Metabolic Research* 2009;41(2):79-85.
- (149) Smith C, Essop MF. Gender differences in metabolic risk factor prevalence in a South African student population. *Cardiovascular Journal of Africa* 2009;20(3):178-82.
- (150) Carnethon MR, Sidney S, Loria CM, Savage PJ, Hill JO, Liu K. Risk factors for the Metabolic Syndrome. The Coronary Artery Risk Development in Young Adults (CARDIA) study, 1985-2001. *Diabetes Care* 2004;27(11):2707-15.
- (151) Gaede P, Lund-Andersen H, Parving H-H, Pedersen O. Effect of a Multifactorial Intervention on Mortality in Type 2 Diabetes. *New England Journal of Medicine* 2008;358:580-91.
- (152) Reeve PA, Faulkner MJ. Disease patterns in a rural black population. *South African Medical Journal* 1986;69(9):551-2.
- (153) Omar MAK, Dyer RB, Rajput MC, Motala AA, Joubert SM. The prevalence of diabetes mellitus in a large group of South African Indians. *South African Medical Journal* 1985;67(23):924-6.
- (154) Silventoinen K, Pankow J, Jousilahti P, Hu G, Tuomilehto J. Educational inequalities in the metabolic syndrome and coronary heart disease among middle-aged men and women. *International Journal of Epidemiology* 2005;34:327-34.

- (155) Twine R, Collinson MA, Polzer TJ, Kahn K. Evaluating access to a child-orientated poverty alleviation intervention in rural South Africa. *Scandinavian Journal of Public Health* 2007;69:118-27.
- (156) Daar AS, Singer PA, Persad DL, Pramming SK, Matthews DR, Beaglehole R, et al. Grand challenges in chronic non-communicable diseases: the top 20 policy and research priorities for conditions such as diabetes, stroke and heart disease. *Nature* 2007;450(22):494-6.
- (157) Kengne AP, Amoah AGB, Mbanya J. Cardiovascular Complications of Diabetes Mellitus in Sub-Saharan Africa. *Circulation* 2005;112:3592-601.
- (158) WHO. Everybody's business:strengthening the health systems to improve health outcomes: WHO's framework for action. Geneva: World Health Organisation; 2007.
- (159) WHO. Increasing access to health workers in remote and rural areas through improved retention: Global policy recommendations. Geneva: World Health Organization; 2010.
- (160) Grahams GN, Kim S, James B, Reynolds G, Buggs G, Hunter M, et al. Benefits of Standardized Diabetes and Hypertension Screening Forms at Community Screening Events. *Health Promotion Practice* 2011;7(1):26-33.

## APPENDIX A: SAMPLING STRATEGIES

### 1. Study enrolment detail

#### THE METABOLIC SYNDROME AND ASSOCIATED RISK FACTORS IN THE JOHANNESBURG HEALTH DISTRICT

##### STUDY ENROLMENT DETAIL

CLUSTER NO: \_\_\_\_ PARTICIPANT NO: \_\_\_\_ STUDY NO: \_\_\_\_

##### Research assistant:

Use the map of Chiawelo. Start at the point marked X for each chosen cluster. You have to go to every 3rd yard until you have 70 adult participants from each of the 5 clusters.

Select the first yard next to the X on the left side of the street.

Select all the adults who are older than 18 years of age who permanently live in this yard and write their names on the sheet named "How to choose the respondent". Follow the instructions on the sheet in order to select a person for the interview.

Write down the cluster and the yard number on the enrolment sheet.

If there are no eligible adults who permanently live in this yard, or if there are people who refuse to participate, move clockwise in the street and skip 2 yards. Try the next yard.

Explain the study details and request selected participant's signed consent.

Address visited: \_\_\_\_\_

Name of research assistant: \_\_\_\_\_

Date of enrolment: \_\_\_\_/\_\_\_\_/ 2009

Comments by research assistant:

---

---

---

## 2. Choosing the respondent

### THE METABOLIC SYNDROME AND ASSOCIATED RISK FACTORS IN THE JOHANNESBURG HEALTH DISTRICT

#### HOW TO CHOOSE THE RESPONDENT

House number: \_\_\_\_\_

C

A Names of all adult participants 18 years or older in descending sequence of age	B									
	1	2	3	4	5	6	7	8	9	10
1	1	1	1	1	1	1	1	1	1	1
2	2	2	1	1	2	1	1	2	1	2
3	1	2	3	2	1	2	3	1	3	3
4	3	1	2	4	1	4	3	2	1	2
5	4	2	3	1	5	5	3	1	4	2
6	2	5	4	6	6	3	1	4	2	5
7	6	7	5	4	2	1	7	3	2	5
8	7	1	4	6	5	3	2	8	1	7
9	4	8	5	9	7	1	2	3	6	9
10	8	10	1	6	7	5	3	9	4	2

#### Research Assistant:

- List all the adults 18 years or older who are permanent members of the yard in column A, starting with the oldest and ending with the youngest. List only those present.
- Draw a line under the name of the youngest adult and extend it across the grid.
- Place the last digit of the house number in the block provided at C.
- Find the column of the same number (as the one you have written at C) in B.
- Draw a line vertically down this column until it meets the horizontal line you have drawn from column A.
- These lines will cross at a number. Whatever that number is, will show which adult you should enroll into the study. For example, if the lines cross at number 3 column 5 you will enroll adult number 1.



## APPENDIX B: MEASUREMENT TOOLS

### 1. Patient information sheet

#### THE METABOLIC SYNDROME AND ASSOCIATED RISK FACTORS IN THE JOHANNESBURG HEALTH DISTRICT

#### PARTICIPANT INFORMATION SHEET

Good day. My name is Dr Nishila Moodley and I am a Registrar at the WITS School of Public Health. You are being invited to participate in a research study to determine the prevalence of the Metabolic Syndrome and the associated risk factors in the Johannesburg Metro District, Gauteng Province. This form is to help you decide whether you wish to participate in the study or not.

#### What is the Purpose of the Study?

The research project, to be conducted from January 2009 will involve interviews with patients to determine what influences the development of hypertension, Diabetes Mellitus, high cholesterol and obesity.

#### Why have you been chosen?

You have been chosen to participate in this study because we are interested to find out how many people in this community have these diseases. I would like to conduct an interview with you requiring about 10-15 minutes of your time, answering various questions about yourself and your health. We also would like your permission to do a blood test to determine whether you have these diseases and whether they require treatment. Lastly we wish to do a few measurements on you - i.e. height, weight, waist and hip.

**Participation is voluntary:** I would like to stress that this is voluntary, and you may choose not to participate or to discontinue participation at any time.

**Risks:** There are no foreseeable risks to your participating. Very minor discomfort might be experienced whilst taking your blood for testing. This will be exactly the same amount of discomfort you may be accustomed to if you have had blood drawn previously.

**Benefits:** The benefits of taking part in this study include having a role in improving the control of these diseases in this community. Diseases that we may diagnose in you that you may have been unaware of may be treated.

#### Confidentiality

Your identity will not be revealed and your confidentiality will be protected in any reviews and reports of this study which may be published.

#### Contact details

If you have any questions or concerns or would like more information about this study please contact: Nishila Moodley, Principal Investigator on the study, at 082 927 1547 or e-mail [nishila.moodley@wits.ac.za](mailto:nishila.moodley@wits.ac.za)

If you are unhappy with the way this research is conducted, you are welcome to contact the Chair of the Wits Human Ethics Committee Prof P Cleaton-Jones through his secretary Ms Anisa Keshav on 011-717-1234.

*Thank you for taking time to read this sheet.*

**Dr Nishila Moodley**

## 2. Patient consent form

### THE METABOLIC SYNDROME AND ASSOCIATED RISK FACTORS IN THE JOHANNESBURG HEALTH DISTRICT

INFORMED CONSENT
------------------

DATE: dd / mm / yy

The research project- *The Metabolic Syndrome and associated Risk Factors in the Johannesburg Health District*

1. Has been explained to me. I understand that it involves a face to face questionnaire administered by a research assistant. I do not mind giving information about myself, my general health.
2. I understand that a blood test is required. I do not mind having this test done on me.
3. I understand that while the study may not have any direct benefits for me, it will help researchers to understand how the management of Diabetes Mellitus, hypertension, high cholesterol and obesity can be improved in the Gauteng province.
4. I understand that my name, address, and other personnel information will not be recorded on the questionnaire forms and thus ensuring my confidentiality.
5. I understand that I do not have to take part in this project. If I choose not to take part or decide not to answer a question, this will not affect the way, in which I will be treated at the health facility. Similarly, if I choose to withdraw from this project at any stage, this will not prejudice me in anyway in the future.

Name of Patient participant

Name of Research Assistant

\_\_\_\_\_

\_\_\_\_\_

Signature of Patient-participant

Signature of Research assistant

\_\_\_\_\_

\_\_\_\_\_

### 3. Patient questionnaire

#### THE METABOLIC SYNDROME AND ASSOCIATED RISK FACTORS IN THE JOHANNESBURG HEALTH DISTRICT

<b>PATIENT QUESTIONNAIRE</b>
------------------------------

DATE: dd / mm / yy

INTERVIEWER: \_\_\_\_\_

#### I. SOCIO-DEMOGRAPHY

1. Age: \_\_\_\_\_
2. Sex: Male  Female
3. Ethnicity:  
Black  White  Coloured  Indian  Other  State: \_\_\_\_\_
4. Marital status:  
Single  Married  Divorced  Widowed  Cohabit
5. Educational level:  
None  Primary  High school   
Matric  Post matric  University
6. Which of the following best describes your work status over the past 1 year?

<input type="checkbox"/> Unemployed (but able to work)	<input type="checkbox"/> Student
<input type="checkbox"/> Unemployed (unable to work)	<input type="checkbox"/> Government employee
<input type="checkbox"/> Retired/pensioner	<input type="checkbox"/> Non government employee
<input type="checkbox"/> Homemaker	<input type="checkbox"/> Voluntary work (unpaid)
<input type="checkbox"/> Self employed	<input type="checkbox"/> Other, specify _____
7. Do you receive a social grant?  
Yes  No   
If YES, what type? \_\_\_\_\_

## II. ASSET INDEX SURVEY

8. How would you describe your home?

Shack / zozo  Flat /cottage  House   
 Hostel  Shared house  Room / Garage

9. How do you describe your floors of your house?

Mud uneven  Mud even  Plastered Tiles   
 Other (describe) \_\_\_\_\_

10. Household water: Do you have access to?

Indoor water  Only outside tap water  Other water source

11. What type of toilet do you have?

Flush inside  Only flush outside  Pit/bucket  Other type

12. Which of the following do you have in your household at the present time?

	YES	NO
Electricity		
Television		
Radio		
Motor vehicle		
Fridge		
Washing machine		
Telephone		
Video machine		
Microwave		

### III. METABOLIC DISEASE PROFILE

13. Have you been told by the clinic staff / any medical staff that you are overweight?

Yes  No

14. Have you been diagnosed with any of the following?

Yes No

14.1 Diabetes Mellitus

14.2 Hypertension

14.3 Dyslipidaemia

		DM		HPT		DYSLIPID	
15	When were you diagnosed?						
16	Where were you diagnosed?						
17.1	Which clinic do you attend?						
17.2	Why this clinic? (Choose 1 or more options) a = convenient                      d = medication always available b = referred here                      e = other, please state c = staff treats you well						
18	Are you on treatment?	Yes	No	Yes	No	Yes	No
19.1	Do you forget to take your medication?	Yes	No	Yes	No	Yes	No
19.2	How often do you forget? (Choose 1 option) a = Every day                      d = >3 times a week b = At least once a week      e = a few times a month c = 2-3 times a week              f = very rarely						
20	Have you received education about your condition?						
21	Have you received education about your diet?						
22	Have you been told to exercise?						
23	When where you last checked at the clinic?						
24	Was the value high during the last year?						
25	Have you visited a traditional healer about this?						
26	Have you taken traditional medications / homeopathic treatment or herbal medication for this?						

27. Have you been hospitalized in the last year?

Yes  No

27.1 If YES, what was the diagnosis? \_\_\_\_\_

#### IV. RISK FACTOR ANALYSIS

28. HISTORY

28.1 Do you have a family member (sibling and /or a parent) with diabetes and / or hypertension? Yes  No

28.2 **FEMALE PATIENTS ONLY:**

Have you had diabetes during your pregnancy?

Yes  No

29. ALCOHOL CONSUMPTION

29.1 Have you consumed alcohol within the past 1 year?

Yes  No

29.2 If YES, how often do you drink?

- Daily
- 5-6 times a week
- 1-4 times a week
- 1-3 times a month
- Less than once a month

29.3 When you do drink, how many standard drinks do you have at one go?  
i.e. on one day?

\_\_\_\_\_

30. SMOKING

- 30.1 Do you smoke cigarettes? Yes  No
- 30.2 How many cigarettes do you smoke in a day? \_\_\_\_\_
- 30.3 How many years have you smoked? \_\_\_\_\_
- 30.4 Do you use snuff? Yes  No
- 30.5 How many times a day do you use snuff? \_\_\_\_\_
- 30.6 How many years have you used snuff? \_\_\_\_\_

31. DIET

- 31.1 In a typical week, how many days do you eat fruit?  
Never  1-2 days  3-4days  5-7 days  Unsure
- 31.2 In a typical week how many days do you eat vegetables?  
Never  1-2 days  3-4days  5-7 days  Unsure

32. PHYSICAL ACTIVITY

- 32.1 During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, digging, aerobics, or fast bicycling?  
\_\_\_\_\_ **days per week**

No vigorous physical activities → *Skip to question 32.3*

- 32.2 How much time did you usually spend doing vigorous physical activities on one of those days?

\_\_\_\_\_ **hours per day**  
\_\_\_\_\_ **minutes per day**

Don't know/Not sure

Think about all the **moderate** activities that you did in the **last 7 days**. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

- 32.3 During the last 7 days, on how many days did you do moderate physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

\_\_\_\_\_ **days per week**

No moderate physical activities → *Skip to question 32.5*

- 32.4 How much time did you usually spend doing moderate physical activities on one of those days?

\_\_\_\_\_ **hours per day**

\_\_\_\_\_ **minutes per day**

Don't know/Not sure

Think about the time you spent **walking** in the **last 7 days**. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure.

- 32.5 During the last 7 days, on how many days did you walk for at least 10 minutes at a time?

\_\_\_\_\_ **days per week**

No walking → *Skip to question 32.7*



32.6 How much time did you usually spend walking on one of those days?

\_\_\_\_\_ **hours per day**  
\_\_\_\_\_ **minutes per day**

Don't know/Not sure

The last question is about the time you spent **sitting** on weekdays during the **last 7 days**. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

32.7 During the last 7 days, how much time did you spend sitting on a week day?

\_\_\_\_\_ **hours per day**  
\_\_\_\_\_ **minutes per day**

Don't know/Not sure

**END**

## APPENDIX C: PERMISSIONS

### 1. Ethical clearance

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

R14/49 Dr Nishila Moodley

CLEARANCE CERTIFICATE

M090831

PROJECT

The Metabolic Syndrom and Associated Risk Factors in the Johannesburg Health District

INVESTIGATORS

Dr Nishila Moodley.

DEPARTMENT

School of Public Health

DATE CONSIDERED

09.08.28

DECISION OF THE COMMITTEE\*

Approved unconditionally

Unless otherwise specified this ethical clearance is valid for 5 years and may be renewed upon application.

DATE

30.08.09

CHAIRPERSON .....

  
(Professor PE Claton-Jones)

\*Guidelines for written 'informed consent' attached where applicable

cc: Supervisor : Dr D Basu

---

DECLARATION OF INVESTIGATOR(S)

To be completed in duplicate and **ONE COPY** returned to the Secretary at Room 10004, 10th Floor, Senate House, University.

I/We fully understand the conditions under which I am/we are authorized to carry out the abovementioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee. I agree to a completion of a yearly progress report.

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES...

## 2. Post-graduate approval letter



Dr N Moodley  
P.O. Box 6932  
Westgate  
1734  
South Africa

Faculty of Health Sciences  
Medical School, 7 York Road, Parktown, 2193  
Fax: (011) 717-2119  
Tel: (011) 717-2745

Reference: Ms Tania Van Leeve  
E-mail: tania.vanleeve@wits.ac.za  
02 July 2009  
Person No: 9602853F  
PAG

Dear Dr Moodley

### **Master of Medicine in the specialty of Community Health: Approval of Title**

We have pleasure in advising that your proposal entitled "*The metabolic syndrome and associated risk factors in the Johannesburg Health District*" has been approved. Please note that any amendments to this title have to be endorsed by the Faculty's higher degrees committee and formally approved.

Yours sincerely

A handwritten signature in black ink, appearing to read 'Sandra Benn', with a horizontal line underneath.

Mrs Sandra Benn  
Faculty Registrar  
Faculty of Health Sciences

## APPENDIX D: PATIENT DOCUMENTS

### 1. Clinic referral letter

# novo nordisk changing diabetes

Dear Doctor,

Date.....

I visited the Novo Nordisk Changing Diabetes Mobile Clinic and have these findings:

Age:	years
Sex:	Male/female
Race:	
Waist Circumference:	
Blood Pressure	
Random Blood Glucose (Finger Prick):	
Weight:	
Height:	

Brought to you in the best interests of Changing Diabetes

**novo nordisk, world leaders in diabetes care.**

Novo Nordisk (Pty) Ltd. Reg. 1959/000833/07  
10A Achter Road, Paulshof, Sandton 2056.  
P.O. Box 783155, Sandton. 2146  
Tel: (011) 202-0500. Toll Free 0800 11 6941  
www.novonordisk.co.za  
08/08



2. Example of disease information pamphlet

# PLANNING YOUR MEAL

DIABETES EDUCATION

CARBOHYDRATES

PROTEIN

VEGETABLES

FRUITS

2/5

1/5

2/5

2/5

novo nordisk  
changing diabetes

Novo Nordisk (Pty) Ltd Reg. 1959/000833/07, 10A Achter Rd. Paulshof, Sandton, 2096, P.O. Box 783155, Sandton, 2146.  
Tel: (011) 202 0500 Fax: (011) 807 7989 www.novonordisk.co.za • www.d4pro.com