A COMPARATIVE EVALUATION OF BODY MASS INDEX, WAIST CIRCUMFERENCE AND WAIST-TO-HIP RATIO AS CORRELATES OF GLUCOSE INTOLERANCE AMONG URBAN ADULTS IN JOS METROPOLIS, NIGERIA

A DISSERTATION SUBMITTED TO THE NATIONAL POSTGRADUATE MEDICAL COLLEGE OF NIGERIA IN PART FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF THE FMCP IN INTERNAL MEDICINE (ENDOCRINOLOGY AND METABOLISM)

BY

ANIEKWENSI EZECHUKWU

MBBS, 1998

DEPARTMENT OF MEDICINE

JOS UNIVERSITY TEACHING HOSPITAL

MAY 2010

DECLARATION

It is hereby declared that this work is original unless otherwise acknowledged. The work has not been presented to any other college for fellowship nor has it been submitted elsewhere for publication.

_____

ANIEKWENSI EZECHUKWU

MBBS, 1998

CERTIFICATION 1

The study reported in this dissertation was done by Dr. Aniekwensi Ezechukwu of the Department of Internal Medicine, JUTH, under our supervision. We have also supervised the writing of the dissertation to our satisfaction.

SUPERVISORS

1. **DR F. H. PUEPET (FMCP)**
   Consultant Physician/Endocrinologist
   Department of Medicine
   Jos University Teaching Hospital, Jos

   Signature:_____

   Date:_____

2. **DR E. K. CHUHWAK (FWACP)**
   Consultant Physician/Endocrinologist
   Department of Medicine
   Jos University Teaching Hospital, Jos

   Signature:_____

   Date:_____

CERTIFICATION II

I certify that this work was done by Dr. Aniekwensi Ezechukwu under the supervision of Dr. F. H. Puepet and Dr. E. K. Chuhwak of the Department of Internal Medicine, Jos University Teaching Hospital.

_____ _____

DR. E. N. OKEKE Date
Head, Department of Internal Medicine
Jos University Teaching Hospital

DEDICATION

This dissertation is dedicated to my wife Chinwe, my daughter Chinememma, and niece Oluebube, for the support I enjoyed during the field survey and in the writing of this work.

To my parents Ogenna and Unoego, my Uncles Pius and Michael, all of whom laid the foundation for my academic pursuits.

And to God the Almighty father from whom all good things come.

ACKNOWLEDGEMENTS

I wish to express my profound gratitude to my trainers and supervisors, Dr. Puepet and Dr. Chuhwak, for their tireless efforts in teaching and training me. Their insistence on excellence in this work is highly appreciated. For all the support during the period of training, I am exceedingly grateful.

I thank all my teachers of the Department of Medicine for all their contribution to my postgraduate training. I am particularly grateful to Prof. Okeahialam for his encouragement and support.

I appreciate Drs. Gezawa and Adegite, colleagues in the same unit for the mutual encouragement we gave each other, Drs. Adegbe and Onuh for their assistance in data analysis. I also thank my younger colleagues who assisted me in ensuring a smooth data collection, and the staff of Tadam Hospital Jos for their assistance with Radioimmunoassay.

LIST OF ABBREVIATIONS

ADA	-	American Diabetes Association
AUC	-	Area under the Curve
BMI	-	Body Mass Index
CT	-	Computed Tomography Scan
CVD	-	Cardiovascular disease
DECODE	-	Diabetes Epidemiology: Collaborative analysis of Diagnostic criteria in Europe
DESIR	-	Data from an epidemiological study on the Insulin resistance syndrome
DM	-	Diabetes Mellitus
EPIC Potsdam	-	European Prospective Investigation into Cancer and Nutrition-Potsdam
FSIGTT	-	Frequently sampled intravenous glucose tolerance test
GI	-	Glucose Intolerance
HOMA	-	Homeostasis Model Assessment
HOMA-IR	-	HOMA INDEX for Insulin Resistance
IAAT	-	Intra-abdominal Adipose Tissue
IDF	-	International Diabetes Federation
IFG	-	Impaired Fasting Glycemia
IGT	-	Impaired Glucose Tolerance
IR	-	Insulin Resistance
MRI	-	Magnetic Resonance Imaging
NCEP	-	National Cholesterol Education Program
NGT	-	Normal Glucose Tolerance
OD	-	Optical Density

OGTT	-	Oral Glucose Tolerance Test
QUICKI	-	Quantitative insulin sensitivity check index
RIA	-	Radioimmunoassay
ROC	-	Receiver Operator Characteristic
SAAT	-	Subcutaneous Abdominal Adipose Tissue
SAD	-	Sagittal Abdominal Diameter
SD	-	Standard deviation
WC	-	Waist Circumference
WHR	-	Waist- to- Hip Ratio
WHtR	-	Waist- to- Height Ratio
WHO	-	World Health Organisation
2HPGL	-	2 hours post-75g post glucose load.

TABLE OF CONTENTS

EXPANDED TABLE OF CONTENTS

CHAPTER TWO

LITERATURE REVIEW

CHAPTER THREE

SUBJECTS, MATERIALS AND METHODS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

BACKGROUND

Body fat distribution has been reported as a significant risk factor for the development of type 2 diabetes mellitus and other forms of Glucose Intolerance. Anthropometric Indices including BMI, WC, and WHR are used as surrogate measures of body fat distribution. Definitive values of BMI, WC, and WHR are used to characterize individuals into general or central obesity. Few studies have evaluated the relationship between anthropometric indices and Glucose Intolerance in Nigerian population. This study was carried out to compare the discriminatory ability of these indices in correctly identifying individuals with glucose intolerance among Urban adults in Jos.

OBJECTIVES

To evaluate the anthropometric Indices of BMI, WC, WHR as correlates of Glucose Intolerance and to determine the prevalence of IFG, IGT, and DM, in the study population and also the prevalence of obesity in the population.

SUBJECTS, MATERIALS AND METHODS

A three-stage random sampling was used to enroll 800 subjects ≥20 years of age residing in Jos. Ill and pregnant subjects were excluded. Out of the 800 subjects, 709 responded and participated in the study giving a response rate of 88.6%. Demographic and clinical data were obtained using a modified WHO STEPS Questionnaire. Anthropometric indices including weight, height, waist circumference and hip circumference were measured. BMI and WHR were calculated. Fasting plasma glucose (FPG) was estimated after 8-12 hours overnight fast. Oral Glucose tolerance test (OGTT) with 75g glucose was also carried out for all subjects. Fasting plasma Insulin was assayed in a subset of 119 subjects to determine Insulin Resistance using Homeostasis model assessment (HOMA-IR). Data was

analyzed with Epi info 3.6.1.and SPSS 16.0. Receiver-Operator characteristic curve was used to test the prediction of glucose intolerance by the anthropometric indices. All p-values less than 0.05 was considered significant.

RESULTS

The subjects were made up of 43.4% of males and 56.6% of females. The mean (SD) age of subjects was 43.21 (14.74) years with a range of 20-89. There was no significant difference in mean age of male and females. Mean (SD) WC and BMI were higher in females than males [WC: 87.5(12.5)cm vs 85.2(10.8)cm, p=0.009, BMI: 27.4 (5.1)kg/m^2 vs 25.3(3.7)kg/m^2, p=0.001] while WHR was higher in males than females [0.88(0.07) vs 0.86(0.07), p=0.02]. The differences were statistically significant. The overall prevalence of IFG was 2.7% (3.2% in men, 2.2% in women, p=0.41) while that of IGT was 7.5% (5.5% in men, 8.5% in women, p=0.13). The prevalence of DM in the study population was 4.1% (4.5% men, 3.7% in women, p=0.59). The prevalence of IFG and DM were higher in men while that of IGT was higher in women, however none was significant. The prevalence of Overweight and Obesity using BMI was 35% and 21% (10.7% in men, 28.9% in women, p=0.0001) respectively. The prevalence of Central Obesity using WHR was 52.6% (39.3% in men, 63.1% in women, p=0.0001) while it was 30.7% (8.8% in men, 47.6% in women, p=0.0001) using WC (NCEP 111) and 47.8% (21.8% in men, 67.8% in women, p=0.0001) using WC (IDF). Obesity was significantly commoner in women than men.

Receiver – Operator Characteristic Curve showed that all the studied indices had significantly higher AUC than the Null hypothesis true area of 0.5, but WHR had the highest Area under the Curve (AUC) for IFG, IGT and DM in both men and women. It performed better than WC and BMI in that order.

CONCLUSION

The results from this study showed that the prevalence of IGT was higher than that of IFG (7.5% vs 2.7%). There was also higher prevalence of IGT than DM (7.5% vs 4.1%) with implications for the future prevalence of DM. Prevalence of Central Obesity using WHR was higher than using WC and also higher than generalized obesity with BMI (52.6% vs 30.7% vs 21%). Out of the three tested anthropometric indices (BMI, WC, WHR), WHR had the best ability to identify Individuals with Glucose Intolerance (GI) in the studied population. WHR, a measure of central obesity is better correlated to Glucose Intolerance than other anthropometric indices.

CHAPTER ONE

INTRODUCTION

1.1 GLUCOSE HOMEOSTASIS

Glucose homeostasis is the maintenance of plasma glucose levels within normal limit. Plasma glucose is derived principally from intestinal absorption, glycogenolysis and gluconeogenesis.[1] Plasma glucose is utilized by cells and tissues of the body. The muscle, fat and liver require insulin for glucose uptake but the Central Nervous System (CNS) does not require insulin for glucose utilization.

Plasma glucose concentration is regulated by a balance between actions of hormones, enzymes and substrates that act to maintain euglycaemia in normal individuals. Despite periods of feeding and fasting, blood glucose concentration is kept within a narrow range of between 4 – 7mmol/l.[1]

The ability of pancreatic B-cells to secrete adequate amounts of insulin and for the secreted insulin to promote glucose utilization is vital to this normal regulation. This maintains normal glucose tolerance, thus there is a net balance between glucose production and utilization. Whenever there is a disturbance of this balance, glucose intolerance results. Glucose Intolerance describes disorders of glycaemia or disorders of glucose tolerance and includes the categories of Diabetes Mellitus (DM), Impaired Glucose Tolerance (IGT) and Impaired Fasting glycaemia (IFG).[2,3,4]

1.2 OBESITY AND RISK OF GLUCOSE INTOLERANCE

Obesity is associated with increased risk of developing insulin resistance and type 2 diabetes.[5] In obese individuals, adipose tissue releases increased amounts of non-esterified fatty acids, glycerol, hormones, pro-inflammatory cytokines and other factors that are involved in the development of insulin resistance.[6] When insulin

resistance is accompanied by dysfunction of pancreatic islet B-cells – the cells that release insulin – failure to control blood glucose levels results.[5]

Both obesity and type 2 diabetes are associated with insulin resistance, but most obese, insulin-resistant individuals do not develop hyperglycemia, until there is B-cell failure.[5] Under normal conditions, the pancreatic islet B-cells increase insulin release sufficiently to overcome the reduced efficiency of insulin action, thereby maintaining normal glucose tolerance.[7] For obesity and insulin resistance to be associated with type 2 diabetes, B-cells must be unable to compensate fully for decreased insulin sensitivity.[5,7] Since the early 1980s, a number of researchers have reported that body fat distribution (based on anthropometric measurements) is a significant risk factor for the development of diabetes.[8,9] These anthropometric measurements including Waist Circumference (WC), Waist – to – Hip Ratio (WHR), Waist – to Height Ratio (WHtR) and Body Mass Index (BMI) provide important information on cardiovascular risk.[10] While WC, WHR and WHtR are measures of central obesity, BMI is a measure of general obesity. These anthropometric indices do not measure only overweight and obesity but also assess undernutrition. The prevalence of obesity and associated chronic diseases such as hypertension and diabetes are on the increase globally especially in developing countries particularly Africa[11]. Prevalence rates of 17.2% and 4.2% has been reported for overweight and obesity in a survey in Jos.[12] Urbanization and Westernization has been largely responsible for the increasing trend of obesity in most African countries.

1.3 Definition and Prevalence of Diabetes.

Diabetes Mellitus (DM) is a metabolic disorder of multiple aetiology characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action or both.[13,14] The chronic hyperglycaemia of diabetes is associated with long term damage, dysfunction, and failure of various organs, especially the eyes, kidney, nerves, heart and blood vessels.

According to a report by King et al[15], estimated global prevalence of DM in 1995 was put at 135 million representing 5.4% of the world population with a projected rise to 299 million in 2025 representing about 6.3% of the world population. In Nigeria, DM is the commonest endocrine disease.[16] The Federal Ministry of Health National Non-communicable Diseases Survey of 1992 puts the prevalence of DM nationwide at 2.2% with the highest prevalence of 7.2% in Lagos mainland and lowest of 0.6% in rural Mangu, Plateau State.[17] In urban Jos, prevalence is put at 3.1%.[18]

1.4 Definition and Prevalence of Impaired Glucose Tolerance

Impaired Glucose tolerance (IGT) is defined by the World Health Organization (WHO) as two-hour plasma glucose concentration of between 7.8 and 11.0mmol/l after oral administration of 75g glucose.[13]

Impaired Glucose Tolerance has been recognized as a stage in the natural history of diabetes. It is not a type of diabetes. The prevalence of IGT varies among population, race and ethnic groups. In North American and Western European populations, prevalences are 10% to 20% of tolerance test (OGTT), with a quarter to a third of older adults (55 years and above) having the condition.[19] In Singapore, the prevalence rate is less than 1% among the Chinese in Da Qing[20] while the rate is

greater than 20% among Muslims in Dar-Es-Salaam, Tanzania.[21] In Nigeria, a hospital based study done in Lagos using data from two diabetes clinics reports the prevalence at 29.5% among patients.[22]

1.5 Definition of Impaired Fasting Glycaemia

Impaired Fasting Glycaemia (IFG) is defined as fasting plasma glucose (FPG) of between 6.1 and 6.9mmol/l. This category of glucose intolerance was introduced by the American Diabetes Association (ADA) in 1997[14,23]. It recognizes individuals with fasting plasma glucose above normal but below the diabetic level. Individuals with impaired fasting glycaemia are also at risk of developing diabetes though at a lesser risk than for individuals with IGT.[24] However, FPG is easier to perform than the WHO OGTT especially in epidemiological surveys.[25]

1.6 JUSTIFICATION FOR THE STUDY

There is a rising incidence of glucose intolerance (DM, IGT and IFG)[2]. Diabetes has become a public health problem because of the risks associated with the disease. Since obesity increases the risk of IGT and DM, [26] it becomes imperative to determine which of these indices – BMI, WHR, and WC, of estimating body fat is the most suitable for the assessment of glucose intolerance risk in clinical practice. Furthermore, central to the management of DM and IGT is lifestyle change of which weight reduction is a cardinal component. Knowing which of the indices is most sensitive will make it a reference parameter in monitoring changes of lifestyle modification measures for clinicians and patients alike.

Published studies evaluating the mentioned indices and relationships with glucose intolerance in Nigeria are few and none has been carried out in Jos, Nigeria.

1.7 AIMS AND OBJECTIVES

General

To evaluate the relationship between Waist Circumference (WC), Waist – to – Hip Ratio (WHR), Body Mass Index (BMI) and glucose intolerance.

Specific

1) To determine which of the following anthropometric indices: WC, WHR and BMI correlates best with glucose intolerance.
2) To determine the prevalence of pre-diabetes states of IFG, IGT, and combination of both and also the prevalence of DM in the study population.
3) To determine the prevalence of Obesity using the WHO, NCEP ATP 111, IDF criteria in the study population.

CHAPTER TWO

LITERATURE REVIEW

2.1 GLUCOSE INTOLERANCE

This is impaired glucose metabolism which usually results when there is disturbance of normal glucose homeostasis. It describes disorders of glycaemia or disorders of glucose tolerance and includes the categories of Diabetes Mellitus (DM), Impaired Glucose Tolerance (IGT), and Impaired Fasting Glycaemia (IFG).[2,3,4] In extrapolating this definition, it can be deduced thus that glucose intolerance is fasting plasma glucose equal to or greater than 5.6mmol/l and two-hour post glucose load equal to or greater than 7.8mmol/l[13,14].

Impaired Glucose Tolerance (IGT) and Impaired Fasting Glycaemia (IFG) have been referred to as prediabetes mellitus states, a condition that may progress to clinical diabetes if not detected and treated early.[27] Diabetes mellitus is a metabolic disorder that can have a significant adverse impact on health, quality of life and life expectancy of individuals as well as on the health care system.[28] The total annual economic burden of diabetes is believed to approach $100 billion in the United States.[28.]

2.2 DIABETES MELLITUS

2.2.1 Classification of DM[13]

DM is etiologically classified on the basis of the pathogenic process that leads to hyperglycaemia.

Type I

- Autoimmune
- Idiopathic

Type 2

- Predominantly insulin resistance
- Predominantly insulin secretory defect

Other specific types

- Genetic defects of beta-cell function e.g. maturity onset Diabetes of the young, MODY 1 – 6.
- Genetic defects in insulin action e.g. type A insulin resistance.
- Diseases of the exocrine pancreas e.g. fibrocalculous pancreatopathy.
- Drug or chemical induced e.g. glucocorticoids.
- Endocrinopathies e.g. Cushings syndrome.
- Infections e.g. congenital rubella
- Uncommon forms of Immune – mediated diabetes e.g. Stiffman syndrome.
- Other genetic syndromes sometimes associated with diabetes e.g. Down syndrome.

Gestational Diabetes

2.2.2 Diagnosis of DM[13]

The diagnosis of DM is based on the following WHO criteria:

1. Classical symptoms of DM (Polyuria, polydipsia, weight loss) plus random plasma glucose ≥ 11.1 mmol/l
2. Fasting plasma glucose ≥ 7 mmol/l
3. Two-hour plasma glucose ≥ 11.1 mmol/l during an oral glucose tolerance test (OGTT).

Random plasma glucose is without regard to time of last meal while fasting plasma glucose means no caloric intake for between 8 to 12 hours. Oral glucose tolerance test (OGTT) is performed using 75g anhydrous glucose dissolved in water.

2.2.3 Stages of DM

The clinical stages range from normal glucose tolerance through impaired glucose tolerance (IGT) and/or impaired fasting glycaemia (IFG) to frank DM which can be non-insulin requiring, insulin requiring for control or insulin requiring for survival. Normal glucose tolerance is fasting plasma glucose of < 5.6mmol/l or random plasma glucose of < 7.8mmol/l.

2.3 Type 2 Diabetes

The progression from normal glucose tolerance to type 2 Diabetes is characterized by dual defects that include insulin resistance and an insulin secretory defect.[29]

Insulin resistance is characterized by decreased tissue sensitivity to insulin and marked compensatory hyperinsulinaemia. Initially, plasma glucose levels are maintained in the normal range. In patients who will eventually develop diabetes, there is a decline in beta cell secretory capacity. The first glucose abnormality that is detected is a rise in the post-prandial glucose levels because of reduced first-phase insulin secretion.[30] With time, further decline in beta cell function leads to elevation of the fasting glucose levels. Eventually diabetes occurs with more insulin secretory loss.

The pathogenesis of type 2 DM is complex and involves the interaction of genetic and environmental factors such as obesity and physical inactivity.

Type 2 DM currently accounts for between 85 – 90% of diabetes cases worldwide. There has been an increasing prevalence worldwide due to the twin factors of urbanization and industrialization with increased obesity and a more sedentary lifestyle.[31] A WHO report[31] estimates that the number of cases of diabetes in developing countries is likely to increase more than two fold within three decades,

from 115 million in 2000 to 284 million in 2030. Type 2 DM is thus increasingly becoming a problem of developing countries , Nigeria inclusive. The economic implications are colossal.

The risk factors for Type 2 DM are modifiable and non-modifiable. The modifiable ones include Obesity and physical inactivity, Hypertension, Dyslipidaemia, IGT and/or IFG, Prior GDM, Polycystic Ovary Disease. The non-modifiable include Age, Race/Ethnicity (Blacks, Hispanics, Native Americans, and Pacific Islanders), family history of DM.[32]

Type 2 DM currently affects 5 – 10% of the world's populations and is now the most frequently encountered metabolic disorder in the world.[33,34] In sub-Saharan Africa, estimated number of people with diabetes is about 7 million and this is expected to rise to 18 million by 2030.[33]

In Nigeria, prevalence is about 2.2%[17] and in Jos, prevalence of DM is 3.1%.[18]

2.4 IMPAIRED GLUCOSE TOLERANCE

This is a pre-diabetic state. Patients with impaired glucose tolerance or impaired fasting glycaemia are at significant risk for the development of diabetes.[35] The natural history of IGT is variable, with about 25% progressing to diabetes, 50% maintaining the abnormal glucose tolerance and 25% reverting to normal glucose tolerance (NGT) over an observational period of 3-5 years.[36,37] Individuals who are older, overweight and have other diabetes risk factors are more likely to progress. Moreover low insulin secretion and severe insulin resistance identify individuals that are more likely to progress to diabetes.[38]

Impaired Glucose Tolerance is a risk factor for the development of diabetes and cardiovascular disease.[39] The DECODE study (Diabetes Epidemiology: Collaborative analysis of Diagnostic criteria in Europe) showed that raised blood

glucose two hours after 75g glucose load is an independent risk factor for premature death.[40]

Studies have shown that appropriate intervention with lifestyle modification (modest weight loss, good dietary habit and regular physical activity) and with drugs such as metformin and acarbose reduces the risk of progression to diabetes in patients with IGT. These studies include the Diabetes Prevention Program,[41] Finnish Diabetes Prevention Study[42] and the STOP-NIDDM trial.[43]

The International Diabetes Federation (IDF) reports the prevalence of IGT worldwide in persons aged 20-79 years to be 344 million, that is, about 7.9% of the world's population in 2010.[44] This is projected to rise to 472 million, about 8.4% of the world's population by 2030. In parts of the world, prevalence of IGT varies from between 15.6% and 20.3% in the US[19,45], 12% in Denmark[46], 8.2% in France[47],10.2% in China[48] to 11.2% in India.[49] In Africa, prevalence was found to be about 10% in rural Bantu and 20% in Dar-Es-Salaam Tanzania.[21] In Nigeria, a hospital based study reported a prevalence of 29.5% among patients,[22] while Olatunbosun et al[50] reported a prevalence of 2.2% in Ibadan and Bakari et al[51] reported a 7.7% prevalence among Hausa Fulani in Northern Nigeria in earlier studies. This indicates that in Nigeria, a developing country, the prevalence is variable, depending on the ethnicity or the population studied.

2.5 IMPAIRED FASTING GLYCAEMIA

This is another category of prediabetes. It has also been established that individuals with Impaired Fasting Glycaemia (IFG) are also at increased risk of developing diabetes and the risk is greatest for those with combined IFG and IGT but IGT is more strongly associated with Cardiovascular disease (CVD) outcomes.[20] The

DECODE study also showed that IGT is a better predictor of future diabetes, cardiovascular disease and mortality than IFG[44].The natural history of IFG is similar to that of IGT. Over a 3-5 years observation, about 25% will progress to DM, 50% will remain with the abnormality and about 25% will revert to Normal Glucose Tolerance.[36,37]

The determinants of elevated but non-diabetic fasting glucose and elevated but non-diabetic 2 hour plasma glucose in an OGTT differ. Raised hepatic glucose output and a defect in early insulin secretion are characteristic of IFG while peripheral insulin resistance is most characteristic of IGT.

In the US, the prevalence of IFG is 9.7% among US adults 40 to 74 years of age[45] while a prevalence of 12.6% was reported in Denmark[46] and a prevalence of 16.3% was reported in France.[47] Bakari et al,[51] reported a prevalence of 0% among 40 Hausa Fulani subjects in Northern Nigeria while a prevalence of 5.5% was reported among indigenous Ghanian subjects.[52]

In all prevalence studies conducted as at 2002, only half or less of people with IFG have IGT and even a lower proportion (20-30%) of persons with IGT also have IFG.[20]

The ADA criteria for IFG confers certain advantage over the WHO criteria for IGT. It is easier, faster and more acceptable to patients and easier to reproduce than OGTT.

Impaired Fasting Glycaemia and Impaired Glucose Tolerance are frequently associated with metabolic syndrome. The Adult Treatment Panel III of the National Cholesterol Education Program (NCEP ATP 111) has identified metabolic syndrome as a constellation of lipid and non-lipid risk factors for coronary artery disease.[53] The syndrome is characterized by insulin resistance, atherogenic dyslipidaemia [high Triglyceride (TG) level, low High Density Lipoprotein (HDL), high Low Density Lipoprotein (LDL)], hypertension, abdominal obesity and pro-thrombotic and pro-

inflammatory states. In a prospective study in Finland, cardiovascular disease and all-cause mortality were increased in men with metabolic syndrome even in the absence of cardiovascular disease and diabetes.[54]

Data on the outcome of lifestyle intervention in individuals with IFG alone do not exist yet.[20]

2.6 OBESITY, ADIPOSITY, ANTHROPOMETRIC INDICES AND GLUCOSE INTOLERANCE

2.6.1 Obesity

Obesity is an important risk factor for cardiometabolic diseases, including diabetes, hypertension, dyslipidaemia and coronary heart disease (CHD). Obesity is considered the strongest risk factor for type 2 diabetes.[25]

Several leading health institutions including the WHO and the National Institute of Health (U.S.A) have provided guidelines for classifying weight status based on BMI.[55,56] These have agreed that men and women who have a BMI $\geq 30 kg/m^2$ are considered obese and are generally at higher risk for adverse health events than are those who are considered overweight (BMI between 25.0 and $29.9 kg/m^2$) or lean (BMI between 18.5 and $24.9 kg/m^2$). Therefore, BMI has become the gold "standard" for identifying patients at increased risk for adiposity – related adverse health outcomes.[57]

2.6.2 Abdominal Fat and Waist Circumference

Body fat distribution is also an important risk factor for obesity-related diseases. Excess abdominal fat (also known as central or upper body fat) is associated with an increased risk of cardiometabolic disease. The application of Computed Tomography (CT) and Magnetic Resonance Image (MRI) enabled investigators to directly measure abdominal fat distribution and to precisely analyze the relationship

between fat distribution and metabolic abnormalities. Since, precise measurement of abdominal fat content requires the use of expensive radiological imaging techniques, Waist Circumference (WC) is often used as a surrogate marker of abdominal fat mass, because WC correlates with abdominal fat mass (subcutaneous and intra-abdominal)[58] and with cardiometabolic disease risk.[8] Men and Women who have waist circumference greater than 102cm and 88cm, respectively are considered to be at increased risk for cardiometabolic disease.[59] These cut-off-points were derived from a regression curve that identified the waist circumference values associated with a BMI 30kg/m^2 in primarily Caucasian men and women living in North Glasgow.[60]

The IDF recently proposed new waist circumference cut off points as criteria for central obesity.[61,62] The values are ethnic and gender specific. European values are 94cm for men and 80cm for women, for Asians 90cm for men and 80cm for women. Sub-Saharan Africans are to use European values until more specific data are available. Magnetic Resonance Imaging (MRI) and Computed Tomography (CT) are considered the gold-standard methods for determining the quantity of Subcutaneous Abdominal Adipose Tissue (SAAT) and Intra-Abdominal Adipose Tissue (IAAT).[63] Most MRI and CT methods involve acquisition of cross-sectional abdominal images which are then analyzed for fat content. The relationships between WC, BMI and adipose tissue compartments in primarily Caucasian and African-American men and women demonstrate that both BMI and WC are strongly correlated with total body adipose tissue mass but that WC is a better predictor of IAAT than BMI.

The biological mechanism(s) responsible for the relationship between excess abdominal fat distribution and cardiometabolic disease is not known. It is thought that relatively increased lipolytic activity of omental fat cells and the direct portal-venous concentration of free fatty acids, causes the liver to be exposed to a high concentration

of free fatty acids which in turn results in hepatic insulin resistance and dyslipidaemia.[64]

2.7 INSULIN RESISTANCE

Insulin resistance is clinically defined as a state in which a given increase in plasma insulin in an individual causes less than expected effect in lowering plasma glucose than it does in normal population.[65] Insulin resistance can be assessed by several methods all of which estimate the relationship between plasma glucose and plasma insulin. The gold standard however is the Euglycaemic Hyperinsulinaemic clamp technique which measures the effect of constant infusion of insulin on glucose utilization under conditions in which the plasma glucose is kept constant by administration of intravenous glucose. It is a difficult and time consuming technique and has found greater use as a research tool. Currently the most commonly used method in clinical practice and epidemiological studies is the Homeostasis model assessment (HOMA)[65]. This is simple as it requires the estimation of fasting plasma insulin and fasting glucose. . The model is derived from a computed program that assesses the relationship between fasting plasma insulin and fasting plasma glucose. From the model, a simple formula for the calculation of HOMA INDEX for Insulin Resistance (HOMA-IR) is generated:

HOMA-IR = Fasting plasma insulin [μU/l] × Fasting plasma glucose[mmol/l] /22.5[65]

The mean HOMA-IR for normal subjects is between 2.1 and 2.7; between 4.3 and 5.2 for individuals with impaired glucose tolerance and 8.3 to 9.5 for patients with Type 2 DM[65].

Other methods of estimating insulin resistance include the frequently sampled intravenous glucose tolerance test (FSIGTT) analyzed by the minimal model of Bergman, short insulin tolerance test, Quantitative insulin sensitivity check index

(QUICKI), insulin response during an OGTT[66]. The prevalence rates of insulin resistance vary depending on the criteria used for its definition. Arbitrary thresholds such as the 25th percentile (as in QUICKI) and 75th percentile (as in HOMA –IR) values have been used to identify individuals with insulin resistance even among normal glucose tolerant populations[67]. About 25% of the normal population has been found to be insulin resistant using insulin and glucose technique[65]. The prevalence increases with worsening of glucose tolerance status, being about 60-75% in patients with impaired glucose tolerance and about 85% in those with type 2 diabetes[65].

2.8 BMI as a tool for determining Obesity

Body mass index is defined as an individual's body weight divided by the square of the height in meters and is expressed as kg/m^2. It scales the weight of an individual according to the height. BMI has been used to define overweight and obesity using a value of ≥25 kg/m^2 and ≥30 kg/m^2 respectively[55,56]. Its major limitation however is that it does not differentiate between fat mass and muscle mass (lean body mass). Also, BMI does not provide information on body fat distribution and is unable to identify abdominal obesity. BMI performed poorly as an index of obesity when compared to other anthropometric indices in some Nigerian studies.[68, 69]

2.9 WC as a tool for determining Central Obesity

Parameters that have been used to define central obesity include Waist Circumference (WC), Waist-Hip Ratio (WHR), Waist to Height ratio (WHtR), Sagittal abdominal diameter (SAD)[56]. Abdominal or Visceral obesity (upper body fat) has been found to correlate more strongly with obesity related health risks than peripheral (lower body) obesity.[70] Waist circumference ≥102cm in men and ≥88cm in women have been set as global thresholds for determining increased likelihood of

developing weight related disorders[71]. Waist circumference is simple to measure and does not require calculation. WHO recommends the midpoint between the lowest rib and the iliac crest as the site of measurement.

2.10. WHR as a tool for determining Central obesity

Waist to Hip ratio is also used to assess body fat distribution. Waist Circumference expressed relative to hip circumference is Waist to Hip Ratio. The WHO has recommended WHR ≥0.90 in men and ≥0.85 in women as cut-off points for increased likelihood of developing obesity related diseases[11]. Ferland et al in a study of 51 obese women, reported that WHR was a good predictor of intra-abdominal adipose tissue.[72]

2.11. Anthropometric Indices in other Studies

The relationship of these anthropometric indices in predicting glucose intolerance has been studied in some parts of the world. In a cross-sectional study involving 1965 people aged 35years and above, Lopatynski et al,[26] reported that with the increasing WC, WHR and BMI, there is a correlated increase of glycaemia especially after an oral glucose load. WC showed the strongest correlation with fasting glycaemia, BMI and WTH ratio showed the strongest link after the load while WHR showed the weakest correlation.

In the European Prospective Investigation into Cancer and Nutrition (EPIC) – Postsdam study,[73] comparison of anthropometric characteristics in predicting the incidence of Type 2 Diabetes was analyzed by calculating the receiver – operator characteristic (ROC) area under the curve (AUC). Among men, waist – to height ratio had the highest Area under the Curve (AUC) – with WTH ratio = 0.77, WC = 0.76,

BMI = 0.75, WHR =0.74. Among women WC = 0.83, WTH Ratio, 0.83, WHR = 0.81 and BMI 0.8. Thus WTH Ratio and WC had the strongest association.

In a similar study in Nagpur, India, the area under ROC curve for WC, WHR and BMI was 0.75, 0.61, 0.73 respectively. Thus WC and BMI appeared to have the highest predictive accuracy.[74]

In another cross-sectional study in France – The D.E.S.I.R. study (Data from an epidemiological study on the insulin Resistance Syndrome)[75], in men, BMI had a non-significantly higher sensitivity than WC or WHR (77% as against 74% and 66%) while for women, WC had a slightly higher sensitivity than BMI or WHR (82% as against 77% and 77%).

In a prospective study in U.S.A with a 13 year follow-up, the cumulative proportions of Type 2 diabetic cases identified according to medians of BMI (≥24.8), WC (≥94cm) and WHR (≥0.94) were 82.5%, 83.6%, and 74.1% respectively. The ROC analysis indicated that WC and BMI were similar and were better than WHR in predicting Type 2 diabetes.[59]

CHAPTER THREE

SUBJECTS, MATERIALS AND METHODS

3.1 STUDY AREA

Jos is the capital city of Plateau State in North-Central Nigeria. Plateau state lies between latitude 80° 24' north and Longitude 80° 32' and 100° 38' east. It is situated in the middle of the country sharing boundaries with Kaduna, Bauchi, Nasarawa, Taraba, Benue and Kogi States. It is located at an elevation of 1300m above sea level and surrounded by hills, the best known being the Shere hills which reaches 1800m

above sea level. It has an equable climate with its average monthly temperatures ranging between 21° and 25°C , average humidity of 60% and average annual rainfall of 1400mm. It is an urban metropolitan setting. The major ethnic groups found here include the Beroms, Anagutas, Afizere and the Jarawas. Others include Tarok and Angas. The current population of Jos by the 2006 census is 736,016.

3.2 STUDY DESIGN

This was a cross-sectional descriptive study.

3.3 SAMPLE SIZE

The sample size was determined using the formula[76]

$$N = \frac{(Z_1 - a)^2 \, P \, (1 - P)}{d^2}$$

Where

N = Minimum sample size

P = Prevalence of impaired glucose tolerance from review of literature (7.7% will be used for this study-prevalence from a similar community based study[51])

d = Level of precision (5% for this study).

$(Z_1 - a)$ = Standard deviation at 95% confidence interval which is 1.960.

Therefore P = .077

1 – P = 1 - 0.077 = 0.923

$$N = \frac{(1.96)^2 \times 0.077 \times 0.923}{(0.05)^2} = 110.$$

Minimum sample size is 110.

A sample size of 800 was used for the study to make it more representative, and allow for non-response.

3.4 SAMPLING TECHNIQUE/PROCEDURE

The study was carried out in Dagip and Fudawa wards of the metropolis. A multi-stage random sampling technique was used to select 800 eligible subjects for this study. In stage one, two local governments of Jos were selected, Jos North and Jos south LGA's. In stage two, a ward was picked from each of the two local government areas in Jos, Dagip ward was randomly selected out of the 20 wards in Jos south LGA while Fudawa ward was randomly selected out of the 20 wards in Jos North LGA. This was done by simple balloting. In stage three, a household enumeration was conducted to identify eligible residents. A total of 800 subjects from 340 households selected systematically (every second household) were identified and invited to participate in the study. Four hundred subjects from 178 households in Dagip and another 400 subjects from 162 households in Fudawa ward.

The main sources of income of this population include paid employment, unskilled labour (artisans), farming, trading including petty trading. The young adults are mainly students. The areas are mainly residential. The diet of the inhabitants of the areas consists mainly of carbohydrates with little proteins and fats and oil.

The study subjects included persons aged 20 years and above residing in the study area. Most similar studies used subjects ≥ 20 years of age.[19,44]

Permission and co-operation for the study was obtained from the Ward Heads and consent sought individually before being enlisted. Written, informed consent (Appendix A) was obtained from individual participants. Ethical approval was obtained from the Ethics Committee of the Jos University Teaching Hospital (JUTH)- (Appendix B).

Inclusion Criteria

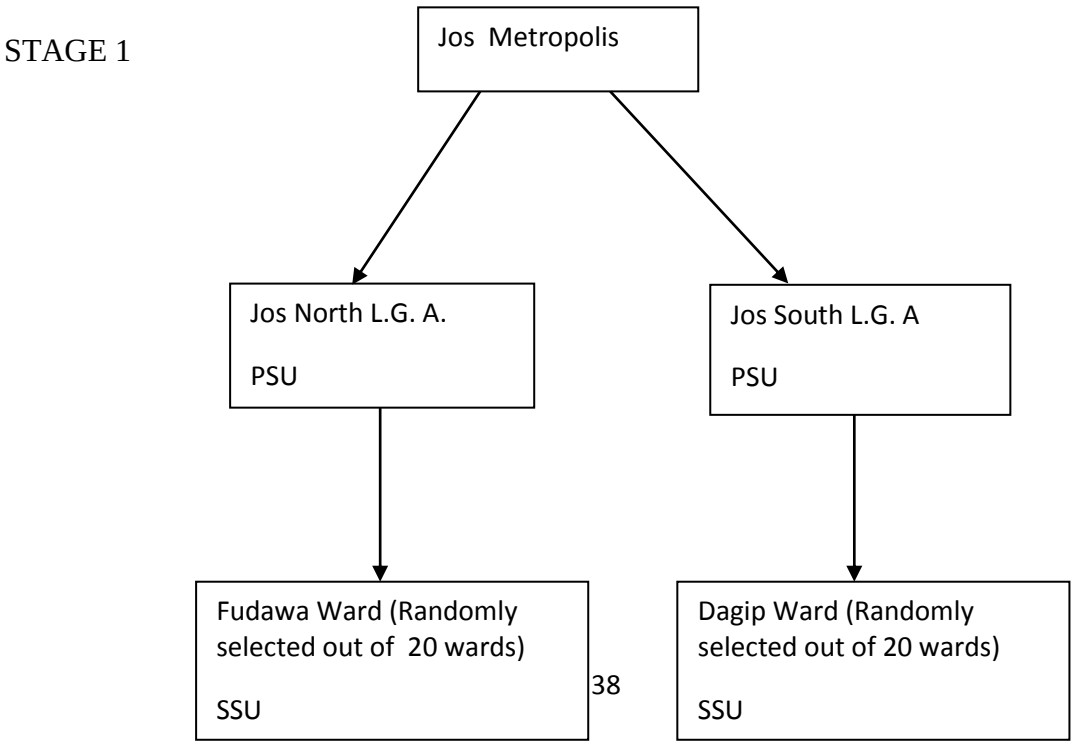
1. Healthy individuals aged 20 years and above.

2. Individuals resident in Jos.
3. Individuals who gave informed consent.

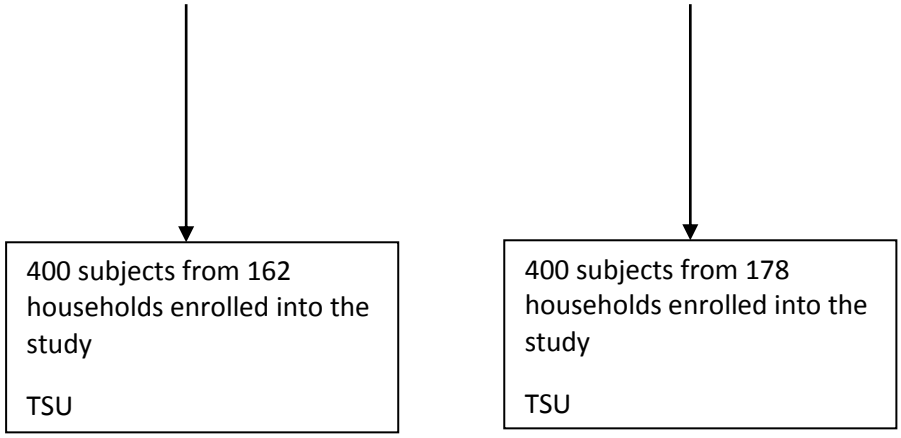
Exclusion Criteria

1 Individuals less than 20 years of age
2. Pregnant women/Lactating mothers
3. Chronic ill-health such as chronic liver disease, chronic kidney disease, chronic heart failure, TB, AIDS, cancer, connective tissue diseases, diarrhoeal disease.
4. Previously diagnosed diabetic patients/patients on anti-diabetic agents.
5. Patients on immunomodulating drugs such as steroids
6. Individuals unable to stand erect.

Fig 1. Flow chart showing the multi-stage sampling technique.



STAGE 2



PSU-Primary sampling unit

SSU- Secondary sampling unit

TSU- Tertiary sampling unit

3.5 STUDY MATERIALS

- Questionnaire (Appendix C)
- Non-stretch metric tape
- Flouride oxalate bottles
- Plain bottles
- Anhydrous glucose
- SWAN Spring water
- Cups and spoons.
- Precision pipettes (Biotech, USA)
- Cotton wool and methylated Spirit

- Syringes and needles (5mls)
- Packets of gloves

3.6 EQUIPMENTS / REAGENTS

- Standard hospital weighing scale with stadiometer (Health Scale Techmel & Techmel Texas USA)
- Accosson's Mercury Sphygmomanometer (Accoson England)
- Vaccine carrier (Cold chain Div.,Nariman Point,Bombay India)
- Deep freezing unit (Westinghouse, Germany)
- Centrifuge (Gallenkamp, England)
- Spectrophotometer (TRSP -721, China)
- Automatic Gamma counter (1470 Wizard, Finland)
- Glucose Oxidase reagent
- RIA kit (MP Biomedicals, Germany) for Insulin assay

3.7 STUDY PROCEDURE

The study was conducted over 20 weeks from January to May 2009. It was a one-day once weekly exercise done on Saturdays and subjects presented to a convenient site (Abbatoir-Giring Catholic Church and Fudawa Cocin Church). Individuals were instructed to present themselves after 8 -12 hours of fasting by 7am. There were 15 people on the survey team, 7 doctors and 8 medical students out of which two were trained for the interviews. On presentation, individuals had a 15-30 minute rest period before measurements and samples were taken, after due consent were given.

The study procedure was explained to all subjects and written informed consent was obtained from each subject. A questionnaire (Appendix C) was administered to obtain relevant demographic, social and medical history. The questionnaire for data collection, physical measurements and biochemical parameters was adapted and modified from WHO STEPS instrument[77]

The following anthropometric measurements were taken:

i. Weight (kg) - Was measured to the nearest 0.1kg with subjects in minimal clothing and without shoes using a standard weighing scale.

ii. Height (m) - Was measured to the nearest 0.1cm with a stadiometer with subjects barefoot and without headgear.

iii. Body Mass Index (BMI) - Was calculated as weight in kg divided by the square of height in meters (m^2) i.e. $kg/m^{2,}$.[56]

iv. Waist circumference (cm) - Was measured to the nearest 0.1 cm using a non-stretch metric tape as the horizontal level at the mid-point between the lowest rib and the iliac crest.[56]

v. Hip circumference (cm) - Was measured to the nearest 0.1cm as the largest circumference of the gluteal region or as the maximal circumference around the buttocks (posteriorly) and the pubic symphysis (anteriorly).[56]

vi Waist – to – Hip Ratio (WHR) - Was calculated as the waist circumference (cm) divided by the hip circumference (cm).

Blood Pressure

Blood pressure was measured using a standard mercury sphygmomanometer (Accoson Variety) on either arm after a 10 minute rest. Korotkoff phases 1 and 5 were recorded as systolic and diastolic blood pressures respectively. A mean of two consecutive readings was recorded as blood pressure.

Blood Sampling Procedure

Blood sample was obtained from a forearm vein by an aseptic procedure for the estimation of laboratory parameters. Subjects were then given 75g anhydrous glucose dissolved in 250ml of SWAN water. Two hours later, another blood sample was drawn for the 2 hour post-glucose load estimation. Blood samples were collected in fluoride oxalate bottles, kept on ice in vaccine carrier and transported within 3 hours of collection to the chemical pathology laboratory of Jos University Teaching Hospital for plasma glucose determination. On arrival at the laboratory, the samples were centrifuged at 3000 rpm for 5 minutes and the plasma separated into plain bottles with pipette and frozen at -20^{o}C till they were analyzed for plasma glucose and plasma insulin.

3.8 Plasma Glucose Assay

Plasma glucose was determined using the glucose oxidase method of Trinder.[78] The details are as described in Appendix D.

Principle of plasma glucose determination

Glucose oxidase is a specific enzyme which promotes the oxidation of glucose to gluconic acid with the production of an equivalent amount of hydrogen peroxide (H_20_2). In the presence of peroxidase, oxygen from H_20_2 is transferred to a suitable acceptor (4-amino phenazone) with the production of a coloured complex, the intensity of which is proportional to the concentration of glucose in the plasma sample. The reaction is as follows:

$$\text{Glucose} + 2H_20 \xrightarrow{\text{Glucose Oxidase}} \text{Gluconic acid} + 2H_20_2$$

$H_2O_2 \xrightarrow{\text{Peroxidase}} H_2O + O$ (nascent oxygen)

O + 4 – Aminophenazone + Phenol $\longrightarrow$ Red-violet solution.

The intensity of the colour formed is measured spectrophotometrically at 520nm.

$$\text{Plasma glucose in sample} = \frac{\text{OD of sample}}{\text{OD of Standard}} \times \text{Concentration of standard.}$$

Precision

For 20 consecutive assay runs, samples of known glucose concentration at three levels of concentration (low, normal and high) was included and these was used to determine the inter and intra assay precision. Co-efficient of variation (CV) of < 5% was used.

3.9 DIAGNOSIS OF GLUCOSE INTOLERANCE[13,14]

1. IFG – Plasma glucose level of 6.0 to 6.9mmol/l after 8 – 12 hours fast.
2. IGT – Plasma glucose level of 7.8 to 11mmol/l after 2 hours of 75g oral anhydrous glucose load.
3. DM – Fasting plasma glucose ≥ 7.0 mmol/l and/or 2 hours plasma glucose ≥11.1 mmol/l after a 75g oral anhydrous glucose load.

3.10. INSULIN ASSAY

Fasting plasma Insulin was assayed in a subset of 119 subjects. Subjects were divided into three groups for the purpose of Insulin assay. The first group were subjects with normal FPG and normal anthropometric indices whom served as controls for the other groups, the second group were subjects with normal FPG and abnormal anthropometric indices, the third group were subjects with abnormal FPG (IFG and DM). Forty subjects were randomly selected from the first and second groups to give 80 subjects. Their selection was by generation of random numbers with Excel computer software from all subjects that fall within the groups. All the 19

subjects that had IFG were selected and all the 20 subjects with diabetic fasting glucose were selected. The frozen fasting plasma samples of selected subjects that have been stored at -20˚C were then analyzed for Insulin estimation. Insulin Resistance was assessed with Homeostasis Assessment (HOMA) model.

The groups were designed such that the first group will serve as controls for the second and third groups with abnormal anthropometric indices and abnormal glycaemic status.

Estimation of Plasma Insulin

Plasma Insulin was estimated by Radioimmunoassay (RIA) technique using commercially available LCC ^{125}I RIA kit. The kit has an intra-assay and inter-assay coefficients of variation of less than 15%. Radioimmunoassays depend on the ability of an antibody to bind its antigen. To quantitate the antigen, the radioactive (containing ^{125}I) and non-radioactive form of the antigen compete for binding sites on the specific antibody. As more of the non-radioactive antigen is added, less radioactive antigen remains bound until equilibrium between the free and antibody bound antigen occurs.

The antibody to the Insulin antigen is covalently bound to a tube wall. Antigen Antibody complex is also bound to the tube wall. At the end of the assay, free antigen is decanted leaving only antibody bound antigen. The coated tube is then counted in a gamma counter calibrated for ^{125}I to determine the level of antibody bound Insulin^{125}I. The details are given in appendix E.

Precision:

The coefficient of variation for all the samples with valid results including standards and quality control were less than 15% and the assay result for quality control were within the range given by the manufacturer.

3.11. DEFINITION OF TERMS

1. **Glucose Intolerance-**was defined to include any one of IFG, IGT, DM.[2,3,4]
2. **Diabetes Mellitus (DM)**-was diagnosed by Fasting plasma glucose ≥ 7.0 mmol/l and/or 2 hours plasma glucose ≥ 11.1 mmol/l after a 75g oral glucose load.[13,14]
3. **Impaired Fasting glycaemia (IFG)**-was defined as fasting plasma glucose concentration of 6.0 to 6.9 mmol/l.[14,23]
4. **Impaired Glucose Tolerance (IGT)**-was defined as plasma glucose concentration of 7.8 to 11.1 mmol/l, [13,14] 2 hours after an OGTT
5. **Fasting-** was defined as no calorie intake for at least 8 hours.

6. **Oral Glucose Tolerance Test (OGTT)**-plasma blood glucose concentration after 75g of oral glucose load with subjects on a diet of at least 150g carbohydrate daily for at least three days prior.

7. **Body Mass Index (BMI)**-was defined as the body weight (kg) of an individual divided by the square of the height in meters, expressed as kg/m^2. Subjects with BMI <18.5 were classified underweight, those with BMI of 18.5-24.9 were classified as having normal weight. Those with BMI of 25.0 - 29.9 and ≥30.0 were classified as overweight and obese respectively.[55,56]

8. **Waist Circumference (WC)-** Waist circumference ≥102 cm in men and ≥88 cm in women was considered abnormal using NCEP 111 Criteria, [59, 74] while waist circumference ≥94 cm in men and ≥80 cm in women was considered abnormal using IDF criteria.[61,62]

9. **Waist to hip ratio (WHR)**- WHR of ≥0.85 in women and ≥0.90 in men were considered abnormal.[11]

10. **Insulin resistance**- Subjects with HOMA-IR values ≥75^{th} percentile value had insulin resistance in this study[67]

3.12. STATISTICAL ANALYSIS

Data was analyzed using both EPI info 3.5.1 and SPSS 16.0 statistical packages. Mean (SD) was used to describe continuous variables and proportion's for categorical data. Two-tailed student t-test was used for comparison of two group means and where groups were more than two, analysis of variance (ANOVA) was used. When comparing groups of subjects, the X^2 (chi-square) test was used to determine the significance of differences. Fischer exact test was employed where cells were less than five.

The Receiver Operator Characteristic (ROC) curve analysis was used to determine which of the anthropometric indices best correlates with glucose intolerance. Pearson correlation was used to determine the direction and strength of association between the anthropometric indices and HOMA-IR. SPSS 16.0 was utilized for these purpose. In all cases, p-values < 0.05 were considered significant.

3.13. LIMITATIONS OF THE STUDY

The following were the identified limitations of the study

1. Sampling – A simple random sampling of a defined population will be the goal of a population study but a multistage sampling is employed instead because of the large size of the population.
2. Insulin could not be assayed in all subjects due to its high cost.

CHAPTER FOUR

RESULTS

4.1 SOCIO- DEMOGRAPHIC CHARACTERISTICS OF THE STUDY SUBJECTS.

4.1.1 Age and Sex distribution of Subjects

Eight hundred subjects were enrolled into the study, of whom 709 responded and participated in the survey giving a response rate of 88.6%. A total of 308 (43.4%) male subjects and 401 (56.6%) female subjects participated in the study giving a male to female ratio of 1:1.3. The mean (SD) age of study subjects was 43.21 (14.73) years with a range of 20 to 89 years. The mean (SD) age of the male subjects was 42.19 (15.3) years while that of female subjects was 43.99 (14.39) years, females being

slightly older, however this difference was not significant,(t=1.62, p=0.10). The age and sex distribution of the study subjects is shown in Table 1.

Table 1: Age and Sex distribution of Study Participants (Responders).

Age	**Sex**			**Percentage**
(Years)	**Males**	**Females**	**Total**	**(%)**
20-29	76	77	153	21.6
30-39	78	91	169	23.8
40-49	50	90	140	19.8
50-59	59	61	120	16.9
60-69	29	54	83	11.7
≥70	16	28	44	6.2
Total	**308(43.4%)**	**401(56.6%)**	**709(100%)**	**100**

X^2=11.26 , P=0.046

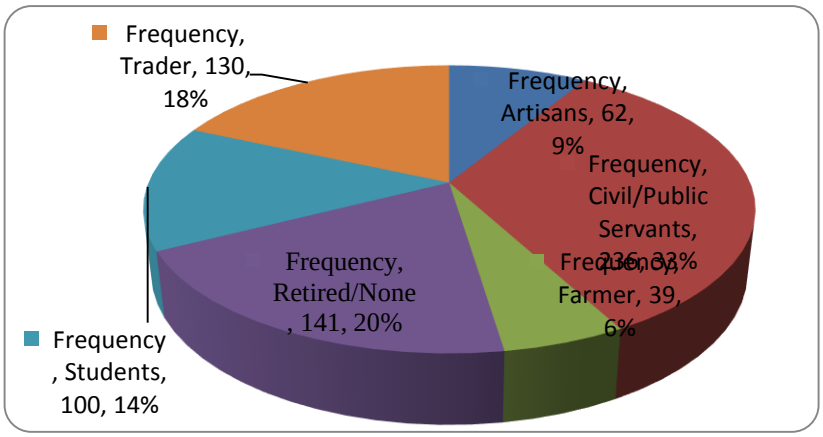
4.1.2 Level of Education

One hundred and fifty three subjects (21.6%) had primary education only, 143 subjects (20.1%) had up to secondary education while two hundred and thirty nine (33.7%) had up to tertiary education. One hundred and seventy four subjects (24.6%) had no formal education.

Overall, majority of the subjects (535 subjects; 75.4%) were educated.

4.1.3 Occupational categories of study subjects

Figure 2: A Pie-chart illustrating the relative frequencies of Occupational categories of subjects.



The categories of occupation of the study subjects is shown in figure 2. The largest proportion were civil/public servants (33%) while the least proportion were farmers (6%).The non-working population made up 20%. Students constituted 14% of the study population.

.

4.2 ANTHROPOMETRIC CHARACTERISTICS OF STUDY POPULATION

4.2.1 Body Mass Index (BMI) of study subjects

The BMI range for all subjects is 16.26 to 42.72 kg/m^2. The range for male subjects is 16.26 to 37.89 kg/m^2 while that for female subjects is 17.09 to 42.72 kg/m^2. The mean (SD) BMI of males and females were 25.3(3.7)kg/m^2 and 27.4(5.1)kg/m^2, respectively (t=5.8,p=0.001) with the difference being statistically significant. The distribution is shown in tables 2 and 3.

Table 2: Distribution of Body mass index (BMI) by age in male and female subjects.

	Mean (SD) BMI (kg/m^2)

Age group (years)	Males	Females	t	P-value
20-29	24.1(3.13)	26.7(4.18)	4.22	0.0001
30-39	24.9(3.15)	27.4(5.02)	3.92	0.0001
40-49	26.2(3.22)	29.3(5.9)	3.35	0.001
50-59	26.6(4.1)	28.0(4.9)	1.65	0.09
60-69	24.0(3.67)	25.7(5.21)	1.58	0.11
≥70	28.0(5.26)	24.7(3.37)	2.55	0.01

Body mass index increased with age in both males and females reaching a peak in the middle ages (40-49,50-59) before declining in the elderly age group. In men, a second peak was seen in the ≥ 70 age group. Females showed higher BMI in all age groups except in the ≥ 70, but significant differences were seen in the age groups 20-29, 30-39, 40-49.

Three hundred and four (42.9%) subjects had normal BMI (BMI=18.5-24.9kg/m^2), two hundred and forty eight (35%) were overweight (BMI 25.0-29.9kg/m^2) while one hundred and forty nine (21%) subjects were obese (BMI≥30.0kg/m^2). The proportion of obese females (28.9%) was significantly higher than that of obese males (10.7%), $p<0.001$.

Table 3: Distribution of BMI categories among subjects by sex.

BMI(kg/m^2)	Males (%)	Females (%)	Total (%)
<18.5	2(0.6)	6 (1.5)	8 (1.1)
18.5-24.9	164 (53.2)	140 (34.9)	304 (42.9)
25.0-29.9	109 (35.4)	139 (34.7)	248 (35.0)

≥30.0	33 (10.7)	116 (28.9)	149 (21.0)
Total	**308 (100)**	**401 (100)**	**709 (100)**

X^2=42.2, p=0.0001.

4.2.2 Waist Circumference (WC) of study subjects

The waist circumference range of all subjects is 62 to 122cm. The range for male subjects is 62 to 119cm while that of female subjects is 63 to 122cm. The mean (SD) waist circumferences of male and female subjects were 85.2(10.8) cm and 87.5(12.5) cm respectively, (t=2.59, p=0.009) with the difference being statistically significant. The distribution is seen in table 4.

Table 4: Distribution of Waist circumference (WC) by age and sex among the study subjects.

	Mean (SD) WC (cm)			
Age Group (years)	**Males**	**Females**	**t**	**P-value**

20-29	79.1(8.26)	80.5 (10.7)	0.94	0.34
30-39	82.4 (8.53)	86.6 (12.0)	2.58	0.01
40-49	86.6 (8.73)	91.0 (13.2)	2.11	0.03
50-59	91.4 (12.3)	91.7 (11.4)	0.11	0.90
60-69	85.64(9.53)	88.35(13.25)	0.95	0.34
≥70	97.18(10.7)	86.78(9.3)	3.37	0.001

Waist Circumference also increased with age in both sexes reaching a peak in the middle age groups (40-49, 50-59) and then declined thereafter. In men it declined and had another peak in the ≥70 age group. Females had higher waist circumference in the age groups except in those ≥70 where males had a higher waist circumference.

Applying NCEP ATP 111 criteria for waist circumference showed that twenty six (8.8%) male subjects had abnormal waist circumference (≥ 102cm) and 191 (47.6%) female subjects had abnormal waist circumference (≥ 88cm) while, 67 (21.8%) male subjects had abnormal waist circumference (≥94cm) and 272 (67.8%) female subjects had abnormal waist circumference (≥ 80cm) applying IDF criteria.

4.2.3 Waist-to-Hip Ratio (WHR) of study subjects

The WHR range of all subjects is 0.7 to 1.28. The range for male subjects is 0.72 to 1.20 while that of females is 0.7 to 1.28. The mean (SD) WHR of male and female subjects were 0.88(0.07) and 0.86(0.07) respectively,(t=2.3,p=0.02) being significantly higher among male subjects. The distribution is seen in table 5.

Table 5: Distribution of WHR by age and sex among study subjects

Age Group(years	**Males**	**Females**	**t**	**P-value**

20-29	0.83(0.04)	0.81(0.05)	1.92	0.05
30-39	0.85(0.04)	0.85(0.05)	0.12	0.89
40-49	0.89(0.08)	0.88(0.08)	1.12	0.26
50-59	0.93(0.07)	0.89(0.06	2.87	0.004
60-69	0.90(0.03)	0.90(0.06)	0.28	0.77
≥70	0.97(0.05)	0.90(0.06)	3.62	0.008

Waist-to-Hip Ratio increased across the age groups with reaching a peak in the elderly in both sexes. Men had significantly higher WHR in the 50-59 and ≥70 age groups.

When WHO criteria for classifying WHR was applied, one hundred and twenty one (39.3%) male subjects had abnormal WHR value equal to or greater than 0.90 and 253(63.1%) female subjects had abnormal WHR values ≥ 0.85.

Table 6: Comparison of mean values of anthropometric indices by sex among subjects.

	Means (SD)		
Anthropometric indices	**Male**	**Female**	**P-value**
BMI	25.3(3.7)	27.4(5.1)	0.001
WC	85.2(10.8)	87.5(12.5)	0.009
WHR	0.88(0.07)	0.86(0.07)	0.02

A comparison of the mean values of the anthropometric indices is seen in table 6. BMI and WC were higher in female subjects while WHR is higher in male subjects. All differences observed were significant.

4.3 PREVALENCE OF GLUCOSE INTOLERANCE

4.3.1 Prevalence of Impaired fasting glycaemia (IFG).

Nineteen subjects had Impaired Fasting Glycaemia giving a prevalence rate of 2.7% (3.2% in men and 2.2% in women). The difference in the prevalence of IFG in males and females was not statistically significant (p=0.41). The range of fasting plasma glucose for male subjects is 3.6 to 15.2 mmol/l while that for female subjects is 3.5 to 15.1 mmol/l. The mean (SD) of fasting plasma glucose for males, 4.7(0.9) mmol/l was not significantly different from that of females, 4.9(1.3)mmol/l, t=1.62, p=0.10. This is shown in Table 7.

4.3.2 Prevalence of Impaired Glucose Tolerance

Fifty-one subjects had Impaired Glucose Tolerance (IGT) giving a prevalence rate of 7.2% (5.5% in men and 8.5% in women). The prevalence of IGT was significantly different in males and females (p=0.13). The range of 2HPGL values in men is 3.2 to 14.8mmol/l while that of females is 3.4 to 19.6mmol/l. The mean (SD) of 2hr post-glucose load value for female and male subjects are 6.36(1.68) and 6.22(1.74) respectively,(t=1.06,p=0.28) being statistically insignificant.

4.3.3 Prevalence of Diabetes Mellitus (DM)

The crude prevalence of DM in the study population was 4.1% (29 subjects). The prevalence rates in male and female subjects were 4.5% and 3.7% respectively,(X^2=0.28,p=0.59) being statistically insignificant. Ten subjects were diagnosed with both fasting plasma glucose and 2hr post glucose load results, ten were diagnosed with fasting glucose only and nine were diagnosed with 2hr post

glucose load only. Table 7 shows the proportion of subjects with IFG, IGT, IFG+IGT, and DM.

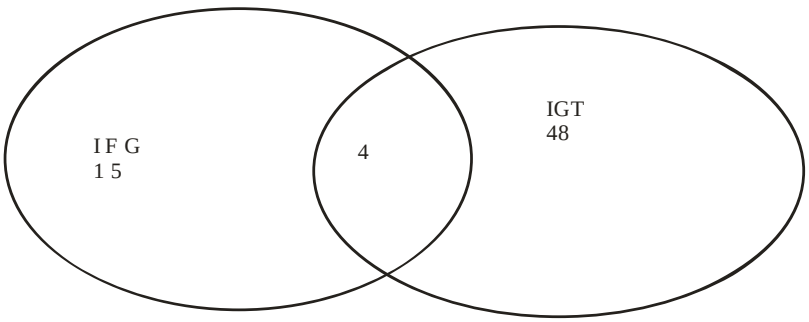
Table 7: Distribution of IFG, IGT and DM among subjects

Glucose Intolerance	**Males n (%)**	**Females n (%)**	**Total n (%)**	**P-value**
IFG	10 (3.2)	9 (2.2)	19 (2.7)	0.41
IGT	18(5.5)	34(8.5)	52(7.2)	0.15
IFG+IGT	0 (0)	4 (1.0)	4 (0.6)	0.07
D.M.	14(4.5)	15(3.7)	29(4.1)	0.59

4.3.4 Prevalence of Pre-Diabetes Mellitus (IFG, IGT, IFG+IGT)

The prevalence of Pre-Diabetes Mellitus in the study population was 9.3% (27 males and 39 females). Four subjects (0.6%) had IFG+IGT (all were female subjects).

Figure 3: A Venn Diagram showing subjects with Pre-Diabetes Mellitus.



The Venn diagram shows the number of subjects with prediabetes mellitus.15 subjects had IFG only, 48 subjects had IGT only, 4 subjects had IFG+IGT. A total of 15+4 had IFG while a total of 48+4 had IGT.

4.4. Prevalence of Overweight and Obesity

The prevalence of Obesity in the study subjects (using BMI ≥30.0 kg/m^2) was 21% (28.9% in females, 10.7% in males, p=0.0001) while the prevalence of overweight was 35% (34.7% in females, 35.4% in males, p=0.73).

The Prevalence of Central Obesity using WHR (WHO) was 52.6%. The difference in the prevalence between males and females was statistically significant [females (63.1%), males (39.3%), p=0.0001].

The prevalence of Central Obesity using WC (ATP III) was 30.7% among subjects. The difference in the prevalence rate between males and females was statistically significant [females (47.6%), males (8.8%), p=0.0001].

The prevalence of Central Obesity using WC (IDF) was 47.8% among subjects .The difference in the prevalence rate between males and females was statistically significant [females (67.8%), males (21.8%), p=0.0001]

Table 8: Prevalence of obesity using different criteria

Indices of obesity	**Obese Males n(%)**	**Obese Females n(%)**	**Obese Total n(%)**
BMI	133(10.7)	116(28.9)	149(21)
WHR	121(39.3)	253(63.1)	374(52.6)
WC (NCEP III)	27(8.8)	191(47.6)	218(30.7)
WC (IDF)	67(21.8)	272(67.8)	339(47.8)

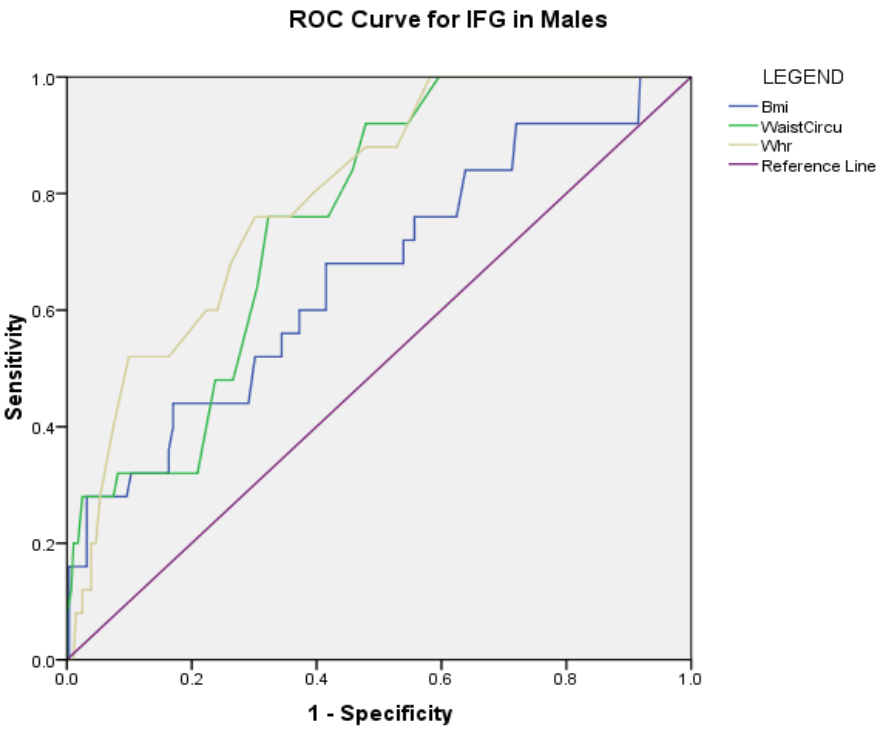
4.5. Anthropometric Indices and Prediction of Glucose Intolerance

The comparative ability of the Indices to correctly identify the three categories of glucose intolerance was tested using the Receiver Operator Characteristic (ROC) Curve analysis.

4.5.1 ROC Curve for IFG in male subjects

The ROC curves of the Anthropometric Indices associated with the presence of IFG in men are shown in Figure 4. The Area under the Curve (AUC) for BMI, WC, WHR were 0.66, 0.75 and 0.79 respectively. Although all the studied Anthropometric Indices had significant abilities to identify IFG in male subjects, WHR was the best in this population. All the Indices showed significantly higher AUC: BMI($p=0.008$), WC($p<0.001$), and WHR ($P<0.001$) than the Null hypothesis true area of 0.5.

FIGURE 4: ROC Curves of the anthropometric Indices associated with the presence of IFG in Male subjects.

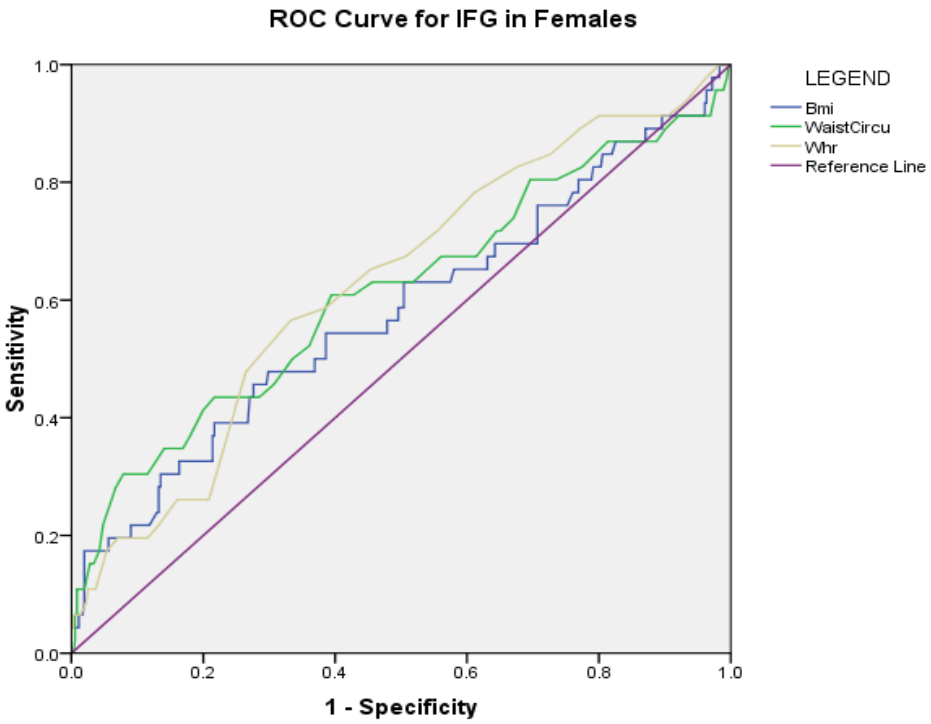


All Indices showed significant AUC compared to the Null hypothesis true area of 0.5. Since WHR had the highest AUC (0.79), as seen in the curves, it has the best predictive ability for IFG in males.

4.5.2 ROC Curve for IFG in Female subjects

The ROC Curves of the Anthropometric Indices associated with the presence of IFG in female subjects are shown in Figure 5. The AUC's for BMI, WC, WHR were 0.57(p=0.08), 0.61(p=0.013), 0.62(p=0.005) respectively showing that although WC and WHR had significant abilities to identify IFG in female subjects, WHR was the best in this population.

FIGURE 5: ROC Curves of the Anthropometric Indices associated with the presence of IFG in female subjects.

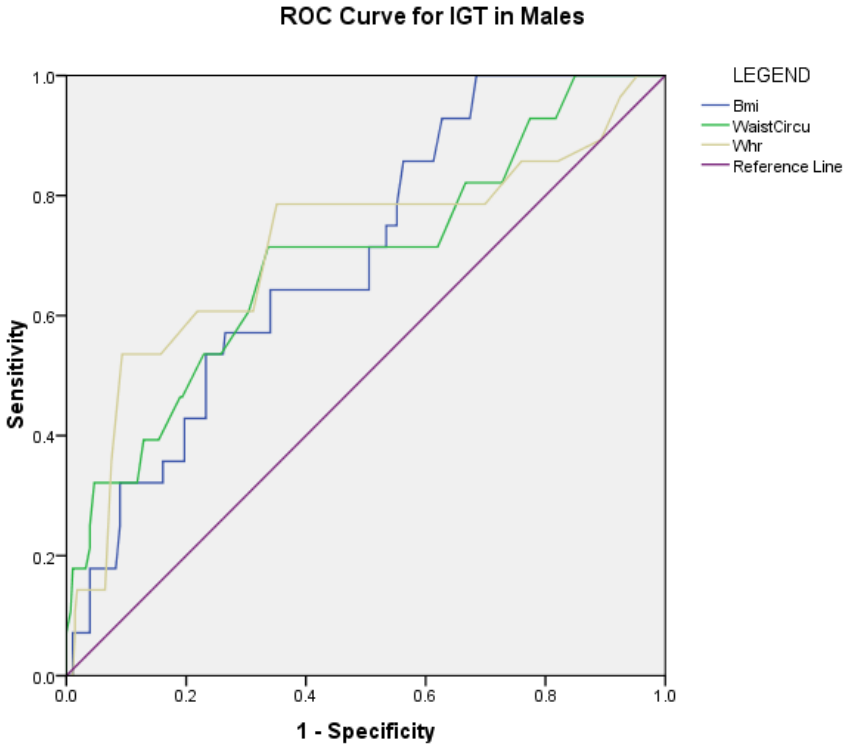


Compared to the Null hypothesis true area of 0.5, only WHR and WC were significant. Since WHR had the highest AUC (0.62), as seen in the curves, it has the best predictive ability for IFG in females. The curves show that the higher the sensitivity, the Indices are no longer discriminatory between subjects with IFG and subjects without IFG.

4.5.3 ROC Curve for IGT in Male subjects

The ROC Curves associated with the presence of IGT in Male subjects are shown in Figure 6. The AUC's for BMI, WC, WHR were 0.69(p=0.001), 0.69(p=0.001), 0.71(p<0.001) respectively. Although all the Indices had significant abilities to identify IGT in male subjects, WHR was the best in this population.

FIGURE 6: ROC Curves of the Anthropometric Indices associated with the presence of IGT in male subjects.

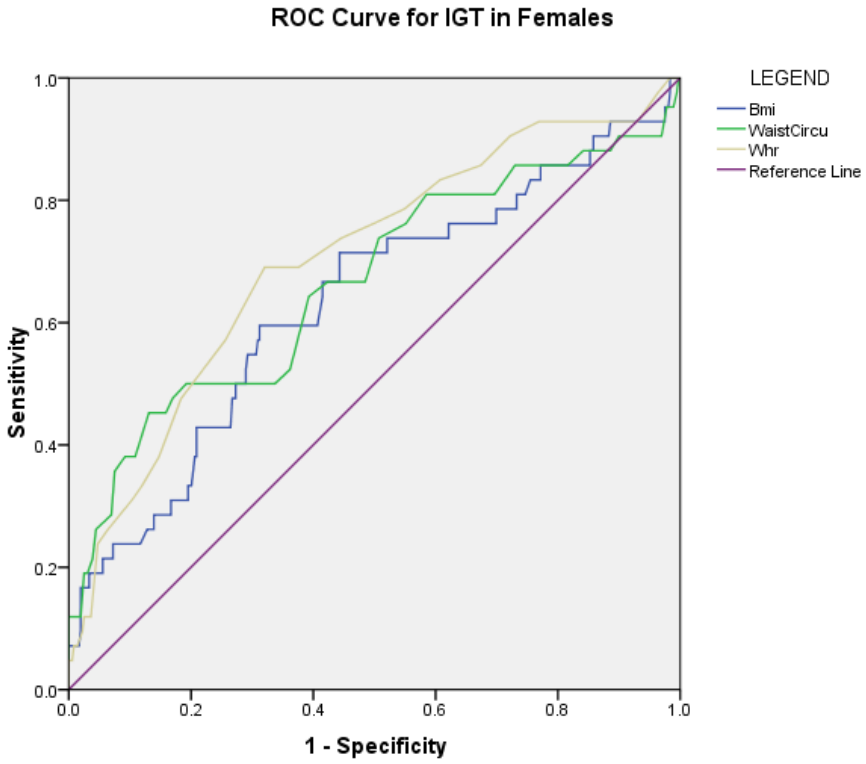


All indices showed significant AUC compared to the Null hypothesis true area of 0.5. Since WHR had the highest AUC (0.71), as seen in the curves, it has the best predictive ability for IGT in males.

4.5.4 ROC Curve for IGT in Female subjects

The ROC Curves of the Anthropometric Indices associated with the presence of IGT in Female subjects are shown in Figure 7. The AUC's for BMI, WC and WHR were 0.63($p=0.005$), 0.66($p=0.001$) and 0.70($p<0.001$) respectively showing that although all the studied indices had significant abilities to identify IGT in female subjects, WHR was the best in this population.

FIGURE 7: ROC Curves of the Anthropometric Indices associated with the presence of IGT in female subjects.

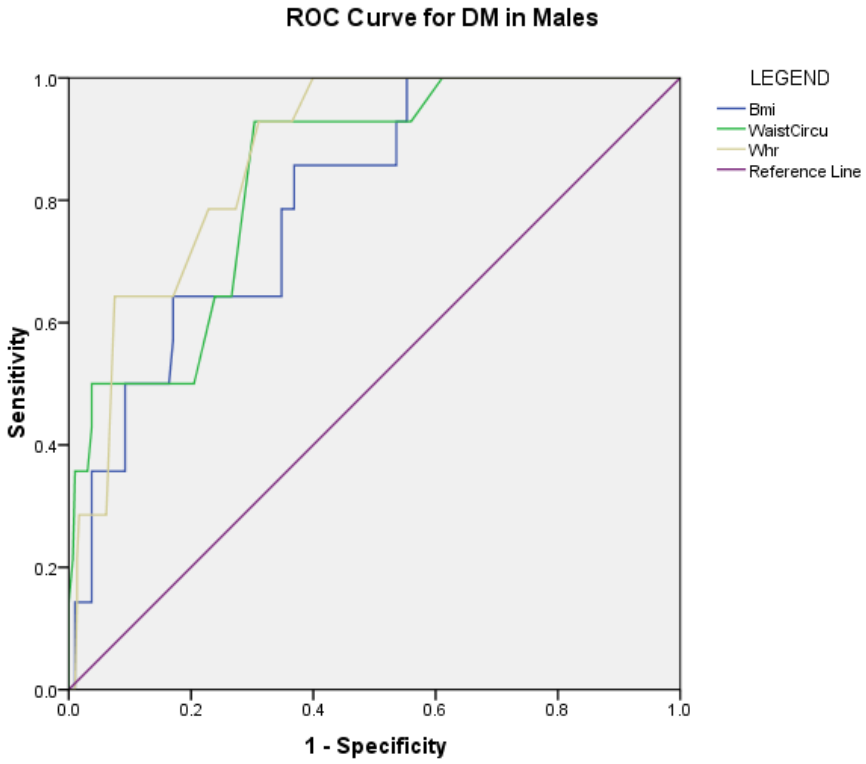


All the indices showed significant AUC compared to the Null hypothesis true area of 0.5. Since WHR had the highest AUC (0.70), as seen in the curves, it has the best predictive ability for IGT in females.

4.5.5 The ROC Curves for DM in Male subjects

The ROC Curves of the Anthropometric Indices associated with the presence of DM in Male subjects are shown in Figure 8. The AUC's for BMI, WC, WHR were 0.79($p<0.001$), 0.83($p<0.001$), 0.87($p<0.001$) respectively showing that although all the studied indices had significant abilities to identify DM in male subjects, WHR was the best in this population.

FIGURE 8: ROC Curves of the anthropometric Indices associated with the presence of DM in male subjects.

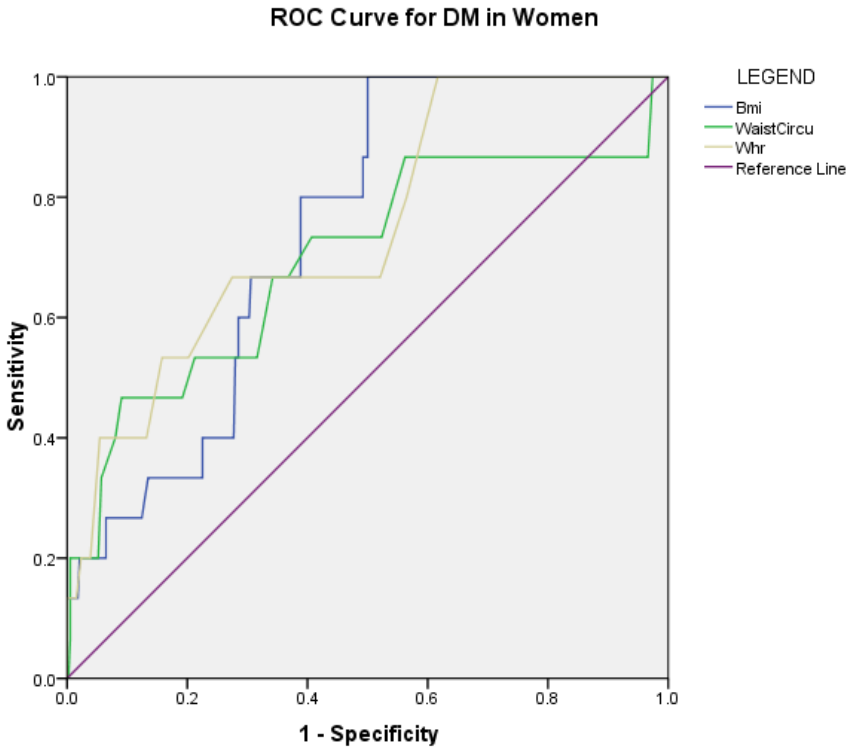


All the indices had significant AUC compared to the Null hypothesis true area of 0.5. Since WHR had the highest AUC (0.87), as seen in the curves, it has the best predictive ability for DM in males.

4.5.6 ROC Curves for DM in Female subjects

The ROC Curves of the Anthropometric Indices associated with the presence of DM in Female subjects are shown in figure 9. The AUC's for BMI, WC, WHR were 0.74(p=0.001), 0.69(p=0.01), 0.74(p=0.001) showing that although all the studied Indices had significant abilities to identify DM in female subjects, WHR and BMI were the best.

FIGURE 9: ROC Curves of the Anthropometric Indices associated with the presence of DM in female subjects.



All the indices had significant AUC's compared to the Null hypothesis true area of 0.5. Since WHR and BMI had similar AUC (0.74), higher than that of WC (0.69), both WHR and BMI had the best predictive ability.

4.6 Anthropometric Indices, Insulin Resistance and Glycaemic Variables.

One hundred and nineteen subjects were selected for estimation of insulin resistance using the Homeostasis Model Assessment Method for Insulin Resistance (HOMA-IR). The selected subjects were in three categories;

1. Subjects with normal Anthropometric Indices and normal fasting glycaemia (40 subjects)

2. Subjects with abnormal Anthropometric Indices and normal fasting glycaemia (40 subjects)
3. Subjects with Abnormal fasting glycaemia (19-IFG; 20-DM)

The three categories are designated Cat1, Cat2, Cat3, in the order listed above. Cat 3 is subdivided into Cat3a (19 Subjects with IFG) and Cat3b (20 subjects with DM).

Thirteen results had gamma count errors and were excluded from analysis. Analysis was therefore based on 106 subjects with valid results. The means (SD) of the Biochemical and Anthropometric variables of these three categories is presented in Table 9, together with their level of significance using ANOVA.

Table 9: Biochemical and Anthropometric variables of 106 subjects selected for Insulin Resistance by HOMA-IR.

Variables	**Cat1 n=38**	**Cat2 n=36**	**Cat3a n=18**	**Cat3b n=14**	**ANOVA f**	**P-value**
BMI(kg/m^2)	22.6(1.47)	31.7(4.96)	30.0(6.35)	32.6(5.50)	34.1	0.0001
WC (cm)	74.3(5.3)	95.5(12.4	98.3(12.5)	101.6(16.0)	33.6	0.0001

WHR	0.81(0.04)	0.89(0.06)	0.91(0.07	0.96(0.11)	19.8	0.0001
FPG (mmol/l)	4.5 (0.4)	4.7 (0.4)	6.3 (0.2)	10.2(2.9)	106.7	0.0001
2hPGL	5.7(0.9)	6.2(1.5)	7.4(2.1)	13.9(3.0)	67.3	0.0001
FPI (μiu/ml)	13.4(5.8)	21.0(10.0)	17.9(8.2)	21.3(16.5)	4.63	0.004
HOMA-IR	2.7(1.2)	4.4 (2.1)	5.0 (2.2)	9.6 (5.6)	17.19	0.0001

Figures are means (SD)

The difference in the means of fasting plasma insulin across the categories was significant (f=4.63,p=0.004) and the difference in the HOMA-IR was also significant (f=17.19, p< 0.0001). There was significant increase in the mean HOMA-IR across the categories. This shows that diabetic subjects are likely insulin resistant more than subjects with IFG who in turn are likely insulin resistant more than subjects with normal fasting glycaemia. Among subjects with normal fasting glycaemia, those with abnormal anthropometric indices (Cat2) are likely to be insulin resistant more than those with normal anthropometric indices (Cat1).

The HOMA-IR threshold that was used for Insulin resistance was the 75th percentile[67] (the upper quartile) and this yielded a cut-off value of 5.64. Applying this HOMA-IR cut-off value of 5.64, 2 subjects (5.3% of subjects in the category) had insulin resistance in Cat1, 9 subjects (25% of subjects in the category) had insulin resistance in Cat2, 6 subjects (33.3% of subjects in the category) had insulin resistance in Cat3a while 9 subjects (64.3% of subjects in the category) had insulin resistance in Cat3b. This shows an increasing prevalence with worsening glycaemic status. There was a positive and strong correlation between HOMA-IR and all three anthropometric indices. Their correlation coefficients (r) were all statistically significant at p<0.001. BMI had a positive and significant correlation with HOMA-IR (r=0.55, p< 0.001), WC had a positive and significant correlation with HOMA-IR (r=0.53,

p<0.001) and WHR also had a positive and significant correlation with HOMA-IR (r=0.34, p<0.001). Overall, BMI had the strongest correlation with HOMA-IR, closely followed by WC while WHR had the least strong correlation.

CHAPTER FIVE

DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

5.1 DISCUSSION

5.1.1 Preamble

Diabetes Mellitus has long been recognized as a public health problem[28], and screening for IFG and IGT has been considered important in recent times since studies have shown that early and appropriate lifestyle intervention or drugs can stop

the development of overt diabetes, the most studied being IGT.[40-42] Obesity has also been recognized as the strongest risk factor for Type 2 Diabetes.[24] Measurements of total body fat and abdominal fat could more precisely predict the impact on health. However, due to high cost and complex instruments involved (Double energy X-ray densitometry, CT and MRI), these instruments are rarely used in epidemiological studies. BMI is by far the most widely used measurement to reflect general obesity, while WHR and WC are used as indices of central obesity. These indices are widely used because of ease of measurement and reliability.

Some authors showed that BMI and WHR were predictors of type 2 diabetes outcome[79] whereas in other studies, WC was a better predictor of type 2 diabetes and was more strongly correlated to intra- abdominal fat than WHR.[80,81] These parameters have ethnic susceptibility and could vary according to age.[82,83]

5.1.2 Prevalence of Impaired Glucose Tolerance (IGT) in study population.

The overall prevalence of IGT in this study was 7.5%. The prevalence rates were 5.9% and 8.8% in males and females respectively. The prevalence is higher in female subjects than in male subjects, however this was not statistically significant. This overall prevalence rate is higher than was previously reported by Olatunbosun et al[50] in Ibadan in 1998. The time interval between the two studies is suggestive that the prevalence of IGT is rising in our environment. However Bakari[51] and co-workers reported 7.7% prevalence among Hausa Fulani subjects in Zaria, though the findings are similar to this study, it may appear that there was no rise in prevalence over the five year interval between the two studies. It has to be noted that the ethnicity of the subjects were different. This study also compares well with 7.9% reported worldwide

by IDF[44] in 2010. It is lower than the prevalence of 15.6% and 20.3% in the US[19,45] 12% in Denmark[46] and 11.2% in India[49],12.7% in China[48],10 to 20% in Tanzania[21]. The lower prevalence rate in our environment supports the fact that sedentary lifestyle, physical inactivity, and other environmental influences are still commoner in the developed world than the developing world. If the trend of westernization continues, our prevalence is expected to continue rising over time. Another reason that may help to explain the observed differences with the US study is that subjects 40 years and above were used in US.

5.1.3 Prevalence of Impaired Fasting Glycaemia (IFG) in study population.

The overall prevalence of IFG was 2.7% in the study (3.2% in male subjects and 2.2% in female subjects). The prevalence was not significantly different between males and females. This prevalence is higher than previously reported from Zaria[51], Northern Nigeria where 0% prevalence was reported. This indicates a rising prevalence in our environment from 0% in Zaria six years ago to the 2.7% obtained in this study. Developing nations are said to be undergoing epidemiological transition,[84] due to the factors of modernization and urbanization with a shift from the traditional African lifestyle to the western lifestyle, change in diet, increasing sedentary lifestyle, reduction in physical activity[85]. As a result, the prevalence of non-communicable diseases is on the increase. In Ghana, a 5.5% prevalence was reported.[52]. Though the prevalence is lower in our study population, the observed difference may portend the involvement of not only environmental factors but also of other factors such as genetic predisposition.[85] Reports from western nations show that our prevalence rate is lower than the prevalence of 9.7% among US adults aged 40-74 years[45] and also

lower than reported by studies from Europe (Denmark 12.6%[46], France 16.3%[47]) and Asia (India 11.0%[49], China 12.7%[48]) .Though the prevalence in our environment is not as high as these, it is on the rise. These are Western countries whose lifestyle we are adopting. If the trend of Westernization continues, our prevalence may be higher over time. Our population is becoming less physically active, more sedentary and consumes more calories, taking after our Caucasian and Asian counterparts. These environmental and lifestyle influences are still commoner in the developed world and may explain why our prevalence though rising, is lower than theirs.

5.1.4 Prevalence of Combined IFG and IGT

The prevalence of Combined IFG and IGT in this study (0.6%) is lower than the 2.8% reported in China[43], 4.4% reported from USA[40], 3.5% from Denmark[41], 3.5% from France[42]. Environmental and other lifestyle influences will help to explain the lower rate seen in our environment. This follows a similar trend to what was discussed in 5.1.1 and 5.1.2. Our prevalence is not as high as in the developed world but it is on the rise.

5.1.5 Prevalence of Diabetes Mellitus (DM) in the study population.

The overall crude prevalence of DM was found to be 4.1%.The prevalence rate was 4.5% and 3.7% in male and female subjects respectively. The prevalence is higher in males than females, however this gender difference was not statistically significant. This is higher than the rates previously reported in some population surveys in Nigeria. In Jos prevalence has risen by 1% from the 3.1% reported by Puepet[18] in 1996 to the 4.1% found in this study. Ohwovoriole[86] et al reported rates of

1.5% and 1.9% in males and females respectively in Lagos metropolis and a national survey by the federal ministry of health in 1992 put the prevalence rate at 2.2% The higher rate in this study is an indication of increasing prevalence of DM in Nigeria as is also being reported in other developing nations[84,87]. Nyenwe[88] and co-workers reported 7.9% prevalence in Port Harcourt. Age range of subjects may account for the observed difference as this study used 20 years and above while older subjects 40 years and above was used in Port Harcourt. The male preponderance seen here agrees with the findings of Puepet[18] and Nyenwe[88] et al. Some surveys reported higher rates in females[86]. Other explanations may be sought for that explains why prevalence rate of DM may be higher in men despite the fact that obesity is higher in women. Generally android obesity is commoner in men as men tend to accumulate intra-abdominal fat which is metabolically active as a result of the activity of the hormone –sensitive lipase which induces insulin resistance[89]. Insulin resistance will cause abnormal glycaemic states.

5.1.6. **Prevalence of Overweight and Obesity in study population**.

The prevalence of Overweight was 35% (35.4% in men and 34.7% in women) while the prevalence of Obesity using BMI was found to be 21% (10.7% in men & 34.7% in women) in the study population .Mean BMI values were higher in female subjects (p=0.001). Body mass Index was also seen to increase across the age categories before declining. The prevalence of overweight and obesity using BMI is higher than previously reported from Lagos and Jos. In an earlier study in

metropolitan Lagos in 1970, Johnson[90] found 4.3% of 440 men and 26.5% of 476 women to be overweight, while 3.1% and 14.9% of men and women were obese. Puepet and Chuhwak[12] reported a 17.2% prevalence of overweight and 4.2% prevalence of obesity among 825 urban adult subjects in Jos in 2002. The time interval between the studies further confirms the rising prevalence of obesity in our environment. The lifestyle factors of increasing sedentary lifestyle and increasing consumption of calorie rich, high fat, low fibre diet as against the traditional African high fibre, low fat diets with reduced levels of physical activity being largely responsible[11].

The prevalence of obesity in this study (10.7% in men and 34.7% in women) showed significant difference in the two sexes (p=0.0001). Several studies in both diabetic and non-diabetic populations have shown that obesity is commoner in women[69,91] In addition to genetic and hormonal differences, this observation has also been linked to lower levels of physical activity in women[92] . In the US, obesity was reported to be 27.6% in men and 33.2% in women, [93] which differs from the 10.7% in men and 28.9% in women in this study. This high degree of obesity seen in developed nations results from abundance of food, excess consumption of calorie dense foods, and physical inactivity due to technological advancement.

A major limitation seen with BMI is that it measures body fat in addition to muscle mass, bone mass, mineral and body water which make up body weight, and so does not provide information on fat distribution and is unable to differentiate abdominal obesity. Cardiovascular risk is known to be related not only to body fat content but also its distribution. In this regard, indices of visceral obesity such as WC and WHR may be superior. The prevalence of central obesity was found to be 52.6% (WHR values), 30.7% (WC- NCEP 111), and 47.8% (WC- IDF) respectively. All the

indices showed significantly higher prevalence in women. Mean WHR was found to be higher in male subjects than in female subjects (p=0.02). On the other hand, mean WC was found to be higher in female subjects (p=009). This is due to the differences in body fat composition between men and women, presence of larger hips in women and the influence of testosterone and oestradiol which promotes fat deposition centrally and peripherally in men and women respectively[89]. Obesity was commonest with WHR. In the UK, prevalence of abdominal obesity using NCEP 111 criteria was 38.8% in men and 51.2% in women.[94] Using the same criteria, the prevalence of abdominal obesity was 8.8% in men and 47.6% in women in this study. The UK findings is higher which further buttresses the fact that obesity is higher in the developed world than in the developing world.

5.1.7 Relationship between Anthropometric Indices and Glucose Intolerance.

The ROC Curve analysis showed that WHR had the highest AUC, followed by WC for all forms of glucose intolerance in both men and women. BMI had the least AUC except for DM in female subjects where it had a higher AUC than WC. Comparing the three anthropometric indices using the ROC analysis, WHR with its highest AUC had the better discriminative ability to identify glucose intolerance among our subjects. This contrasts with Caucasian studies[80,81] where WC performed better than WHR as a predictor of type 2 DM. The ROC curve analysis for type 2

diabetes in men in this study had AUC's of 0.79, 0.83, 0.87 for BMI, WC, WHR respectively. These AUC's are higher than the ones obtained in the EPIC-Potsdam study[73] for type 2 diabetes in men with AUC's of 0.75, 0.76, 0.74 for BMI, WC, WHR respectively. For men, WHR had the highest AUC in this study while WC had the highest AUC in the EPIC-Potsdam study. In women, for type 2 diabetes, the AUC's in this study were 0.74, 0.69, 0.74 which was lower than the AUC's in the EPIC-Potsdam study, 0.80, 0.83, 0.81 for BMI, WC and WHR respectively. BMI and WHR had the highest AUC in this study while WC had the highest AUC in the EPIC-Potsdam study.

In the D.E.S.I.R. study,[75] for type 2 diabetes, in men, BMI had a higher AUC than WC or WHR (0.77 as against 0.74 and 0.66) while in women, WC had a higher AUC than BMI or WHR (0.82 as against 0.77 and 0.77). This differs also from this study which in men, WHR had a higher AUC than BMI or WC (0.87 as against 0.83 and 0.79) and in women, BMI and WHR had higher AUC's than WC (0.74 as against 0.69).

Thus while WC co-relates better and appears to be more sensitive than BMI or WHR in identifying type 2 diabetes in Caucasians, WHR co-relates better and probably more sensitive than BMI or WC in identifying type 2 diabetes in our study population. We observed that in our subjects while some had normal BMI and WC, such subjects may have abnormal WHR. This is reflected in the fact that Obesity was commonest using WHR in our study population. This was in agreement with the general observation that native Africans tend to have smaller physical frame than their Caucasian counterpart. This finding suggests that native Africans deposit fat differently from Caucasians whom appear to have larger waist circumference than Africans. Hoffman et al, found that Caucasians had a greater visceral adipose tissue

mass and smaller subcutaneous adipose tissue mass compared with African-americans respectively[95]. Mechanisms to explain the racial difference in visceral adipose tissue content are lacking[95]. BMI performed least in its discriminative ability to identify categories of glucose intolerance. BMI is unable to differentiate between fat mass and muscle mass and does not identify abdominal obesity which has been shown to correlate more strongly with obesity related health risks than peripheral obesity[70]. Some Nigerian studies also showed that BMI compared to other anthropometric indices performed poorly as an index of obesity among Nigerian adults with diabetes mellitus.[68] In our subjects with glucose intolerance, body fat distribution represented by WHR was more sensitive than WC or BMI in identifying them. WHR may be a better measure of central obesity than WC in this population.

5.2 CONCLUSION

i) The crude estimates of Glucose intolerance from this study is fairly high. This shows that the prevalence of DM has increased in Jos compared to previous finding. It may also be increasing in Nigeria. The prevalence of IFG/IGT was higher than DM (12.8% vs 4.1%). That of IGT was higher than that of IFG (7.5% vs 2.7%). All subjects found to have diabetes had gone undetected prior to the screening which confirms the observation that type 2 diabetes may go undetected and thus untreated for a considerable length of time.

ii) The prevalence of obesity is also on the increase and prevalence is higher among female subjects supporting the findings that obesity is commoner in women.

iii) The study also showed that WHR is the best of the three anthropometric indices studied in identifying individuals with Glucose intolerance in the study population.

5.3 RECOMMENDATIONS

1. The Diabetes Association of Nigeria should formulate a policy for annual screening for DM, IGT, and IFG among our adult population that will effectively cover the entire nation.
2. The Government should allocate funds for more research on measures to reduce obesity and thereafter mount a strong and sustained awareness campaign so as to reverse the trend of increasing prevalence of obesity because of its associated morbidities.

3. Larger multicentre studies should be encouraged and funded to enable us conclude that WHR is the best correlate of glucose intolerance in the Nigerian population.

REFERENCES

1. Kahn CR. Glucose Homeostasis and Insulin action. Principle and Practice of Endocrinology and Metabolism; Second Edition, Philadelphia: J.B. Lippincott 1995; PP 1198 – 1201.
2. Samuel Olatunbosun, Samuel Dagogo-Jack. Glucose Intolerance. Medscape CME. Available at www.medscape.com. Accessed 6th January 2010.

3. Cecilia I, Gabrielle G, Luisa G, Francesco M, Gincarlo V. Prevalence and concomitants of Glucose Intolerance in European Obese children and adults. Diabetes Care 2003; 26: 118-124.

4. Thomas H, Sharina P, Mark P, Kiang L, Carlos I, Catarina K. Active and passive smoking and development of glucose intolerance among young adults in a prospective cohort. BMJ 2006; 332: 1064-1069.

5. Kahn SE, Hull RL, Utzschneider KM. Mechanisms Linking obesity to insulin resistance and type 2 diabetes. Nature 2006; 444: 840-846.

6. Scherer PE. Adipose Tissue: From lipid storage compartment to endocrine organ. Diabetes 2006; 55: 1537 – 1545.

7. Kahn SE. Quantification of the Relationship between insulin sensitivity and B-cell function in human subjects. Evidence for a hyperbolic function. Diabetes 1993; 42: 1663 – 1672.

8. Kissebah AH, Vydelingum N, Murray R, Evans DJ, Hartz AJ, Kalhoff RK, et al. Relation of body fat distribution to metabolic complications of obesity. J Clin Endocrinol Metab 1982; 54:254 – 260.

9. Krotkiewski M, Bjorntorp P, Sjostrom L, Smith U. Impact of Obesity on Metabolism in men and women: importance of regional tissue distribution. J Clin Invest 1983; 72: 1150 – 1162.

10 Berber A, Gomez-Santos R, Fanghanel G, Sanchez-Reyes L. Anthropometric indices in the prediction of Type 2 Diabetes, Hypertension and Dyslipidemia in a Mexican population. Int. J. Obes 2001; 25: 1794 – 1799.

11. World Health Organization Expert Committee: Physical Status ; the use and interpretation of Anthropometry. Report of a WHO Expert committee. Technical report series 845, WHO Geneva, 1995.

12. Puepet FH, Zoakah AI, Chuhwak EK. Prevalence of overweight and obesity among urban Nigerian Adults in Jos. Highland Medical Research Journal. 2002; 1: 13-16.

13 .World Health Organization (W.H.O). Definition, diagnosis and classification of Diabetes Mellitus and its complication. Report of a WHO consultation Part 1. Geneva. WHO, 1999.

14 .American Diabetes Association (ADA); Clinical Practice Recommendations. Report of the Expert Committee on the diagnosis and classification of Diabetes Mellitus. Diabetes care 2002; (23), Suppl 1.

15. King H, Aubert R, Herman W. Global burden of diabetes, 1995 – 2025. Prevalence, numerical estimates and projections. Diabetes care 1998; 21: 1414 – 1431.

16. Famuyiwa O.O. Problems and challenges in the practice of Endocrinology in Developing Country. Nigerian Medical Practitioner 1990; 20: 47 – 52.

17. The National Expert Committee on Non-Communicable Diseases in Nigeria. Report of a National Survey. Federal Ministry of Health, Lagos. 1997: 64 – 90.

18. Puepet F.H. The prevalence of DM and associated risk factors in adults in Jos, Nigeria: Part II FMCP Dissertation. National Postgraduate Medical College of Nigeria; 1996.

19. Harris MI, Eastman RC, Flegal KM, Cowie CC, Eberhardt MS. Comparison of Diabetes diagnostic criteria in the US population according to the 1997 A.D.A. and 1985 W.H.O diagnostic criteria. Diabetes Care 1997; 20: 1859 – 1862.

20. UnWin N, Shaw J, Zimmet P, Alberti KG. Impaired glucose tolerance and Impaired fasting glycemia: the current status on definition and intervention. Report of International Diabetes Federation Expert Consensus Workshop. Diabet Med 2002; 19(9): 708 – 723.

21. Mclarty DG, Swai AB, Kitange HM. Prevalence of diabetes mellitus and Impaired glucose tolerance in rural Tanzania. The Lancet 1989; 1: 871 – 875.

22. Ohwovoriole AE, Olorondu JO, Agboola-Abu CF, Kuku SF. Impact of using criteria of the American Diabetes Association in categorizing glucose tolerance. African Journal of Endocrinology and Metabolism 2001; 2(1): 46-47.

23. Genuth S, Alberti KG, Bennett P, Buse J, Defronzo R, Khan R et al. Expert committee on diagnosis and classification of diabetes mellitus; Follow up report on diagnosis of diabetes mellitus. Diabetes care 2003;26: 3160-3167.

24. American Diabetes Association Position Statement. Standards of Medical Care in Diabetes. Diabetes Care 2006; 29(S1): S4 – S42.

25. WHO Expert Committee on Diabetes Mellitus. Second Report. Geneva. WHO Tech Rep Ser 1980; 646.

26. Lopatynski J, Mardarowicz G, Szczesniak G. A comparative evaluation of waist circumference, waist – to – hip ratio, waist – to – height ratio and Body Mass Index as indicators of Impaired glucose tolerance and as risk factors for Type 2 DM. Ann Univ Mariae Cuire Sklowdoska [Med] 2003; 58(1): 413 – 419.

27. American Association of Clinical Endocrinologists (AACE) Diabetes Mellitus Guidelines. Endocr Pract 2007; 13 (Suppl. 1): 7

28. American Diabetes Association. Direct and Indirect costs of diabetes in the United States in 1992. Alexandria, VA. American Diabetes Association, 1993.

29. Ramlo-Halstead BA, Edelman SV. The natural history of Type 2 diabetes. Implications for Clinical Practice. Prim Care 1999; 26: 771 – 789.

30. Pietropaolo M, Le Roith D. Pathogenesis of diabetes: Our current understanding. Clin Cornerstone 2001; 4: 1 – 16.

31. Zimmet PZ, McCarty PJ, Decourten MP. The global epidemiology of Non-insulin dependent diabetes mellitus and the metabolic syndrome. J Diabet Compli 1997; 11: 60 – 68.

32. American Diabetes Association, Position Statement; Screening for Type 2 Diabetes. Diabetes Care 2004; 27: 11 – 40.

33. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: Estimates for the year 2000 and projection for 2030. Diabetes Care 2004; 27: 1047 – 1053.

34. Amos AF, McCarty DJ, Zimmet P. The rising global burden of diabetes and its complications. Estimates and projections to the year 2010. Diabet Med 1997; 14(5): 1 – 85.

35. Edelstein SL, Knowler WC, Bain RP, Andres R. Predictors of progression from impaired glucose tolerance to NIDDM: An analysis of six prospective studies. Diabetes 1997; 46: 701 – 710.

36. Gabir MM, Hanson RL, Dabetea D, Imperatore G, Roumain J, Bennett PH, et al. The 1997 American Diabetes Association and 1999 World Health Organization criteria for hyperglycemia in the diagnosis and prediction of diabetes. Diabetes Care 2000; 23: 1108 – 1112.

37. Stern MP, Williams K, Haffner SM. Identification of persons at high risk for Type 2 Diabetes Mellitus: Do we need the oral glucose tolerance test? Ann Intern Med 2002; 136:575 – 581.

38. Kahn SE. The relative contributions of insulin resistance and beta – cell dysfunction to the pathophysiology of Type 2 Diabetes. Diabetologia 2003; 46: 3 – 19.

39. Haffner SM, Stern MP, Hazuda HP. Cardiovascular risk factors in confirmed prediabetic individuals: Does the clock for coronary heart disease start ticking before the onset of clinical diabetes? JAMA 1990; 263: 2893 –2899.

40. International Diabetes Federation: Fact sheet, Impaired glucose tolerance. Available from www.idf.org. Accessed 6th January 2010.

41. Diabetes Prevention Program Research Group. Reduction in the incidence of Type 2 Diabetes with lifestyle intervention or metformin. N Engl J Med 2002; 346:393 – 403.

42. Tuomilehto J, Lindstrom J, Erikson JG. Prevention of Type 2 DM by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med 2001; 344: 1343 – 1350.

43. Chiasson JL, Josse RG, Gomis R, Hanefeld M, Karasik A, Laapso M, et al. Acarbose for prevention of Type 2 Diabetes Mellitus: The STOP – NIDDM randomized trial. Lancet 2002; 359: 2077 – 7.

44. International Diabetes Federation: Diabetes Atlas – Prevalence 2005. Available from www.idf.org. Accessed 6th January 2010.

45. Harris MI, Flegal KM, Cowie CC, Eberhodt MS, Goldstein DE, Little RR, et al. Prevalence of diabetes, impaired fasting glucose and IGT in US adults. 1988 – 1994. Diabetes Care 1998; 21: 518 – 524.

46. Glumer C, Jorgensen T, Borch – Johnsen K. Prevalence of diabetes and impaired glucose regulation in a Danish population: The Inter 99 study. Diabetes Care 2003; 26: 2335 – 2346.

47. Balkan B, Eschweye E, Ducimeture P, Richard JL, Warmet JM. The high risk of death by alcohol related disease in subjects diagnosed as diabetic and impaired glucose tolerance. The Paris prospective study after 15 years of follow-up. J Clin Epid 1991; 44: 465 – 470.

48. Dong Y, Gao W, Nan H, for the Qingdao Epidemiology Study Group.The prevalence of diabetes and risk factors in Chinese population in Qingdao city. 18th International Diabetes Federation Congress, Paris, France, 24-29 August 2003.

49. Ramachadran A, Snehalatha C, Kapar A et al. High prevalence of diabetes and IGT in India. National Urban diabetes survey. Diabetologia 2001; 44 :1094 – 1101.

50. Olatunbosun ST, Ojo PO, Finberg NS, Bella AF. Prevalence of Diabetes Mellitus and Impaired glucose tolerance in a group of urban adults in Nigeria. J Nat Med Asso 1998, 90(5): 293 – 301.

51. Bakari AG, Onyemelukwe GC. Glucose Intolerance among apparently healthy Hausa Fulani Northern Nigerians. Ann Afri Med 2004; 3:32 – 34.

52. Amoah AG, Dara PS, Trudy Gaillard, Kwame O. Insulin resistance, beta-cell function and cardiovascular risk factors in Ghanaians with varying degrees of glucose tolerance. Ethnicity and disease 2002; 12: 3-10

53. Executive Summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 2001; 285: 2486 –2497.

54. Lakka HM, Laaksonen DE, Lakka TA. The metabolic syndrome and total and cardiovascular disease mortality in middle – aged men. JAMA 2002; 288: 2709 – 2716.

55. World Health Organization. Obesity: Preventing and managing the Global Epidemic: Report of a WHO consultation on Obesity. Geneva, WHO 1998.

56. National Institute of Health: National Heart, Lung and Blood Institute. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults – the evidence report. Obes Res 1998; 6 (Suppl.2): S51 – S209.

57. Klein S, Allison D, Heymsfield SB, Kelley DE, Liebel RL, Nonas C, Kahn R. Waist circumference and cardiometabolic risk. A consensus statement from shaping America's Health: Association for Weight Management and Obesity Prevention: NAASO, the Obesity Society, the American Society for Nutrition; and the American Diabetes Association. Diabetes Care 2007; 30: 1647 – 1652.

58. Pouliot MC, Depes JP, Lemieux S. Waist circumference and abdominal saggital diameter: Best simple anthropometric indices of abdominal visceral adipose tissue

accumulation and related cardiovascular risk in men and women. AM J Cardiol 1994; 73: 460 – 468.

59. Wang Y, Rimm EB, Meir JS, Willet WC, Hu FB. Comparison of abdominal adiposity and overall obesity in predicting risk of Type 2 diabetes among men. Am J Clin Nutr 2005; 81 : 555 – 563 .

60. Lean ME, Han TS, Morrison CE. Waist Circumference as a measure for indicating need for weight management. BMJ 1995; 311: 158-216.

61. Alberti KG, Zimmet P, Shaw J. IDF Epidemiology Task Force Consensus Group. The Metabolic Syndrome-a new worldwide definition. Lancet 2005; 366: 1059-1062.

62. Alberti KG, Zimmet P, Shaw J. Metabolic Syndrome-a new worldwide definition. A Consensus Statement from the International Diabetes Federation. Diabet Med 2006; 23(5): 469-480.

63. Shen W, Wang Z. Adipose Tissue quantification by imaging methods: a proposed classification. Obes Res 2003; 11: 5 – 16.

64. Bjorntorp P. Metabolic implications of body fat distribution. Diabetes Care 1991; 14: 1132 – 1143.

65. Lebovitz HE. Insulin Resistance: Definition and Consequences. Exp Clin Endocrinol Diabetes 2001;109(Suppl 12): S135-S148.

66. Radzuik J. Insulin sensitivity and its measurements. Structural commonalities among the methods. J Clin Endocrinol Metab 2000; 85: 4426-4433.

67. Ascaso J, Pardo S, Real J, Lorente R, Priego A, Carmena R. Diagnosing insulin resistance by simple quantitative methods in subjects with normal glucose metabolism. Diabetes care 2003; 26: 3320-3325.

68. Okubadejo N, Fasanmade A. Concomitant hypertension and type 2 diabetes mellitus in Nigerians: Prevalence of obesity and its indices compared to normotensive diabetics. Niger Med J 2004; 45:79-83.

69. Bakari A, Onyemelukwe G. Indices of Obesity among type 2 diabetic Hausa –Fulani Nigerians. Int J Diab Metab 2005; 13: 28-29.

70. Kahn S, Prigeon R, Schwartz R, Fujimoto W, Knopp R, Brunzel J. Obesity, body fat distribution, insulin sensitivity and islet-B cell functions as explanations for metabolic diversity. J Nutri 2001;131: 3545-3606.

71. Flegal K, Graubard B, Williamson D, Gail M. Excess death associated with underweight, overweight and obesity. JAMA 2005; 293: 1861-1867.

72. Ferland M, Despres J, Trembley A, Pinault S, Nadeau A, Moorjani S, et al. Assessment of Adipose Tissue distribution by Computed axial tomography in 51 obese women; association with body density and anthropometric measurements.Br J Nutr 1989; 61:139-148.

73. Schuleze SB, Heidemann C, Schienkiewitz A, Bergmann MM, Hoffmann K, Boeing H. Comparison of Anthropometric characteristics in predicting the incidence of Type 2 Diabetes in the EPIC – Potsdam study. Diabetes care 2006; 29: 1921 – 1923.

74. Manju RM, Hemant Kulkarni: Predictive performance of Anthropometric Indices of Central Obesity for the Risk of Type 2 Diabetes. Arch Med Res 2005; 36(5): 581 – 589.

75. Balkau B, Sapinho D, Petrella A, Mhamdi L, Cailleau M, Arondel D, et al and the D.E.S.I.R. Study group: Prescreening tools for diabetes and obesity-associated dyslipidemia: Comparing BMI, Waist and WHR. The D.E.S.I.R Study. Eur J Clin Nutri 2006; 60: 295 – 304 .

76. Oyejide CO. Health Research Methods for developing country scientists. ISBN 978 – 195.044-2 pages 59-63

77. World Health Organisation. WHO STEPwise approach to chronic disease risk factor surveillance- Instrument version 2.0,Questionnaire file. Department of Chronic Diseases and Health promotion. Available at www.who.int/steps (accessed 25th March 2008.)

78. Sacks DB. Carbohydrates. In Tietz Fundamentals of clinical chemistry.5th Ed. WB Saunders Company 2001 pg 427-461.

79. Sekikawa A, Eguchi H, Igarashi K. Waist to hip ratio, body mass index, and glucose intolerance from funagata population based diabetes survey in Japan. Tohoku J Exp Med 1999; 189: 11-20.

80. Wei M, Gaskill SP, Haffner SM, Stern MP. Waist circumference as the best predictor of Noninsulin dependent diabetes mellitus (NIDDM) compared to body mass index, waist hip ratio and other anthropometric measurements in Mexican Americans- a 7-year prospective study. Obes Res 1997; 5: 16-23.

81. Falkenberg M. Check your waist circumference! Overweight, obesity and abdominal obesity risk factors of type 2 diabetes. Lakartidningen 2001;98: 3520- 3522.

82. Okosun IS, Liao Y, Rotimi CN, Choi S, Cooper RS. Predictive values of waist circumference for dyslipidemia, type 2 diabetes and hypertension in overweight White, Black and Hispanic American adults. J Clin Epidemiol 2000; 53: 401-408.

83. Daniel M, Marion SA, Sheps SB, Hertzman C, Gamble D. Variation by body mass index and age in waist-to-hip ratio associations with glycemic status in an aboriginal population at risk for type 2 diabetes in British Columbia, Canada. Am J Clin Nutr 1999; 69:455-460.

84 Omran AR. The epidemiological transition. A theory of the epidemiology of population change. The Milbank Memorial fund quarterly report bulletin of WHO. 2001; 76; 161-170.

85. McKeigue PM, Pierpoin T, Ferrie JE, Marmot MG. Relationship of glucose intolerance and hyperinsulinemia to body fat pattern in South Asians and Europeans. Diabetologia 1992: 35; 785-791.

86. Ohwovoriole A, Kuti J, Kabiawu S. Casual blood glucose levels and prevalence of diabetes mellitus in Lagos metropolis Nigerians. Diab Res Clin Pract 1988;4:153-158

87. Levitt N, Katzenellenbogen J,Bradshaw D, Hoffman M, Bonnia F. The prevalence and identification of risk factors for NIDDM in urban Africans in Cape Town, South Africa. Diabetes Care 1993; 16(4):602-607.

88. Nyenwe E, Osaretin O, Anele I, Aaron O, Seye B. Type 2 diabetes in adult Nigerians: a study of its prevalence and risk factors in Port Harcourt, Nigeria. Diab Res Clin Pract 2003;62: 177-185.

89. Wajchenberg BL. Subcutaneous and Visceral Adipose Tissue: Their relation to the Metabolic Syndrome. Endocrine Reviews 2000;21:697-738

90. Johnson TO. Prevalence of overweight and obesity among Adult subjects of an urban African population sample. British journal Prev. Soc. Med.1970; 24:105-109.

91. Coker A, Fasanmade O, Ohwovoriole A. HOMA-estimated insulin resistance in Nigerians with type 2 diabetes mellitus. Abstract of the scientific sessions of the Nigerian Society of Endocrinology and Metabolism (NSEM); September 26-29,Lagos, Nigeria. Abstract NSEM2007 ABS13.

92 Sobongwi E, Mbanya J, Unwin N. Physical activity and its relationship with obesity, hypertension and diabetes in urban and rural Cameroun. Int J Obes Relat Metab Disorder 2002; 26: 1009-16.

93. Baskin ML, Ard J, Franklin F, Allison DB. Prevalence of obesity in the United States. Obesity Reviews 2005; 6(1): 5-7.

94. Morrell J, Fox KA. Prevalence of abdominal obesity in Primary care: the IDEA UK study. Int J Clin Pract. 2009; 63(9):1301-1307.

95 Hoffman D, Zimian W, Gallagher D, Heymsfield S. Comparison of visceral adipose tissue mass in African Americans and whites. Obesity Research 2005; 13:66-74.

APPENDIX – A

CONSENT FORM

A COMPARATIVE EVALUATION OF BODY MASS INDEX, WAIST CIRCUMFERENCE, WAIST-TO-HIP RATIO AS CORRELATES OF GLUCOSE INTOLERANCE AMONG URBAN ADULTS IN JOS METROPOLIS, NIGERIA

I am Aniekwensi Ezechukwu, a resident Doctor in Jos University Teaching Hospital carrying out a study on the above subject. The study involves participants presenting themselves after 8 -12 hours of fasting which will be in the morning period between 7am and 10am (they would have their normal dinner the previous night but would not eat before coming in the morning). Waist circumference, height, weight and blood pressure will be measured. Blood sample will be taken from a forearm vein, then participants will receive a 75g glucose dissolved in water. Two hours later, another blood sample will be drawn. Participation is voluntary and will be of no financial cost and participants may withdraw from the study at any time. Withdrawal from the study will not affect any medical care to be given to me at the Jos University Teaching Hospital.

Individuals participating in the study found to have glucose intolerance will be referred to the Jos University Teaching Hospital for appropriate treatment.

This is to certify that I …….. have been fully informed about the study and have fully accepted to participate.

Subjects' Signature/Thumbprint	**Date**
Signature/Thumbprint of witness	**Date**
Signature of Investigator	**Date**

APPENDIX B

ETHICAL APPROVAL

JOS UNIVERSITY TEACHING HOSPITAL
JOS, NIGERIA

Phone: 073-450226 - 9
E-mail: juth@infoweb.abs.net

Cables & Telegram: JUTH
P.M.B. 2076
Jos.

Ref: JUTH/DCS/ADM/127//XXVII/2037

Date: 7th July, 2008

Dr. Aniekwensi Ezechukwu,
Medicine Department,
JUTH, Jos.

RE: ETHICAL CLEARANCE/APPROVAL

I am directed to refer to your application dated **21/04/2008** on the research proposal titled: **"A comparative evaluation of body mass index, waist circumference and waist-to-hip ratio in predicting glucose intolerance among urban adults in Jos Metropolis, Nigeria"** and your appearance before the Ethical Committee on 13th June, 2008.

Following recommendation from the Institutional Health Research Ethical Committee, I am to inform you that Management has given approval for you to proceed on your research topic as indicated.

You are however required to obtain a separate approval for use of patients and facilities from the department you intend to use for your research.

The Principal Investigator is required to send a progress report to the Ethical Committee at the expiration of three (3) months after ethical clearance to enable the Committee carry out its oversight function.

Submission of final research works should be made to the Institutional Health Research Ethical Committee through the Secretary in Room 14, Administration Department, please.

On behalf of the Management of this Hospital, I wish you a successful research outing.

Hajia R. Danfillo
for: Chairman, IHREC

DIRECTORATE OF CLINICAL SERVICES & TRAINING
DISPATCHED
09 JUL 2008
Jos University Teaching Hospital
P.M.B 2076, JOS.

APPENDIX C

QUESTIONNAIRE

A COMPARATIVE EVALUATION OF BODY MASS INDEX, WAIST CIRCUMFERENCE, WAIST-TO-HIP RATIO IN PREDICTING GLUCOSE INTOLERANCE AMONG URBAN ADULTS IN JOS METROPOLIS, NIGERIA.

1. SOCIO DEMOGRAPHIC DATA

DATE _____ SERIAL NO.

NAME (INITIALS)_____

AGE _____

SEX (1) MALE ☐ (2) FEMALE ☐

OCCUPATION (SPECIFY) _____

MARITAL STATUS
(1) SINGLE ☐
(2) MARRIED ☐
(3) DIVORCED ☐
(4) WIDOWED ☐

EDUCATIONAL STATUS
(1) INFORMAL ☐
(2) PRIMARY ☐
(3) SECONDARY ☐
(4) TERTIARY ☐

PAST MEDICAL HISTORY (1) DIABETES ☐
(2) OBESITY ☐
(3) HYPERTENSION ☐
(4) OTHERS (SPECIFY) _____

CURRENT MEDICATIONS (SPECIFY)_____

FAMILY HISTORY

Relationship to family member KEY

1. Yes
2. No
3. Don't Know

Disease Condition	Father	Mother	Brother/Sister	Others (Specify)
Diabetes				
Obesity				
Hypertension				
Others (Specify)				

2.PHYSICAL MEASUREMENTS

Height (m) _____

Weight (kg) _____

BMI (kg/m^2) _____

Waist circumference (cm) _____

Hip circumference (cm)_____

Waist-to-Hip Ratio _____

Blood Pressure (mmHg) Systolic …….

Diastolic ……..

3. BIOCHEMICAL PARAMETERS

BLOOD GLUCOSE

Fasting plasma glucose_____

2hr Post glucose load _____

FASTING PLASMA INSULIN(subset only)

……..

APPENDIX D

PLASMA GLUCOSE ASSAY (TRINDER, ANALYTIC METHOD)

This will entail glucose oxidase enzyme buffered in phenoxylate and dissolved in a Colour reagent. A constituted solution will contain:

Potassium dihydrogen sulphate	38.9mls
Disodium hydrogen sulphate	61.1mls
Glucose oxidase	1.0ml
Phenol	0.64ml
4-Aminophenaxone	20mg
Sodium	100mls

This solution is stored at 4ºC.

Procedure:

- Place 2mls of glucose oxidase containing solution inside the test tube
- Add 0.2ml plasma which has been allowed to thaw and warmed to room temperature to the test tube
- Incubate the mixture in a water bath at 37ºCfor 15mins.
- Allow to cool down to room temperature and observe for a Colour change from colourless solutions to a pink solution.
- Read off the absorbance (optical density- O.D) of the pink solutions using a spectrophotometer at the wavelength of 540nm.
- The reading is then compared to that of a standard glucose solution with known glucose concentration.

Glucose estimation is calculated thus:

$$\frac{\text{O.D of test solution}}{\text{O.D of standard}} \times \text{Glucose concentration in the standard}$$

APPENDIX E

PLASMA INSULIN ASSAY PROCEDURE

REAGENTS PROVIDED

A. Anti-insulin Coated Tubes

B. Insulin Standards (STD 1-7)

C. Insulin ^{125}I (lyophilized)[TRACER]

D. Insulin Buffer

E. Insulin controls (Control 1-2)

Procedure

1. Reconstitute the lyophilized tracer insulin with 5mls of distilled water and allow to sit at room temperature for 60 mins. Then mix with the Insulin buffer solution.

2.Bring all standards, coated tubes and tracer to room temperature prior to use

Standards, controls and unknowns should be assayed in duplicate.

3. Place the required number of anti-insulin tubes in attest tube rack.

4. Add 100µL each of insulin standards, controls and unknown samples to their respective tubes.

5. Add 900 µL of TRACER/BUFFER solution to all tubes.

6. Vortex all tubes thoroughly and incubate at room temperature for 18 hours.

7. Aspirate the tubes.

8. Rinse each tube with 4mls of distilled water.

9. Repeat step 7.

10. Count the empty tube in a gamma counter calibrated for ^{125}I for 1min.

11.Calculate the results using the supplied formula

11. A standard curve will be constructed from the standards

    (using commercially available software or using graph paper)

12. The results of the samples will be read from the standard curve.

RAW DATA

Age	Sex	Height	Weight	Bmi	WaistCircu	HipCircum	Whr	Fpg	N2hrpgl	Fpisubset
24	Female	1.69	95	33.26	106	117	0.91	5.8	7.2	
25	Female	1.59	54	21.36	69	92	0.75	5.5	5.8	
36	Female	1.6	58	22.66	80	97	0.82	5.7	6.5	
51	Male	1.66	82	29.76	107	104	1.03	5.3	12.1	14.93
21	Female	1.65	66	24.24	77	101	0.76	4.3	6.2	21.98
59	Male	1.66	58	21.05	90	94	0.96	5.8	3.9	
27	Male	1.57	64	25.96	80	93	0.86	6.7	6.2	
43	Female	1.53	70	29.9	68	53	1.28	7.3	9.1	
39	Female	1.59	86	34.02	102	111	0.92	4.7	5.8	
62	Female	1.6	77	30.08	107	110	0.97	7.8	11.3	20.4
49	Female	1.45	72	34.24	102	114	0.89	5.2	5.9	
30	Male	1.79	75	23.41	79	94	0.84	4.4	5.4	
21	Male	1.73	63	21.05	75	88	0.85	4.3	6.2	
25	Female	1.5	59	26.22	75	98	0.77	3.9	4.3	
70	Female	1.4	43	21.94	78	85	0.92	4.3	9.7	
50	Female	1.62	65	24.77	85	99	0.86	4.2	5.3	
54	Female	1.66	88	31.93	106	118	0.9	3.8	6.4	
40	Male	1.58	65	26.04	73	98	0.74	3.8	6	
21	Male	1.72	70	23.66	79	97	0.81	5.1	4.1	
60	Male	1.75	71	23.18	83	97	0.86	5.9	6.3	
60	Female	1.49	46	20.72	83	84	0.99	4.7	6.4	
50	Female	1.55	66	27.47	95	104	0.91	4.4		
75	Male	1.66	65	23.59	92	92	1	5.7	6.5	
48	Female	1.61	90	34.72	108	119	0.91	4.9	4.7	
70	Female	1.54	60	25.3	88	104	0.85	5.2	4.1	
24	Female	1.53	57	24.35	69	96	0.72	3.8	6.3	
56	Female	1.62	55	21.15	93	95	0.98	3.8	5.3	
65	Female	1.56	46	18.9	75	89	0.84	4.6	7.6	
70	Female	1.49	63	28.38	92	107	0.86	15.1		
21	Male	1.63	67	25.22	74	97	0.76	3.9	5.7	
66	Male	1.65	75	27.55	87	99	0.88	4.1	5.1	29.97
21	Male	1.6	62	24.22	77	95	0.81	4.6	4.8	
21	Male	1.65	55	20.2	72	87	0.83	5.2	5.7	
21	Female	1.52	54	23.37	69	92	0.75	5.1		
33	Female	1.48	47	21.46	71	89	0.8	5.4	6.2	
49	Female	1.66	78	28.31	103	117	0.88	4.9	6.8	
30	Female	1.5	55	24.44	89	99	0.9	5	6.3	
60	Male	1.71	67	22.91	88	100	0.88	4.1	5.7	
27	Female	1.58	93	37.25	99	112	0.88	5.3	4.8	31.51
53	Male	1.73	79	26.4	87	99	0.88	4.5	3.6	
52	Female	1.62	83	31.63	96	111	0.86	4.8	5.4	
48	Female	1.68	76	26.93	85	103	0.83	5.2	4.4	
39	Female	1.7	88	30.45	97	109	0.89	4.4	7	
43	Female	1.48	84	38.35	102	112	0.91	5.8	8.2	
89	Male	1.7	103	35.64	119	108	1.1	7.2	14.4	66.63
75	Female	1.45	44	20.93	82	87	0.94	4.6	7.4	
40	Female	1.54	69	29.09	89	104	0.86	4.8	7.8	
56	Female	1.46	58	27.21	90	100	0.9	4.9	8.3	20.41
75	Male	1.71	83	28.38	94	102	0.92	5.1	5.5	9.93
38	Female	1.53	71	30.33	87	108	0.81	4.8	5.7	
56	Female	1.61	72	27.78	87	103	0.84	4.9	6.1	
56	Female	1.53	83	35.46	106	109	0.97	9.2	19.6	31.08
46	Male	1.68	67	23.74	74	90	0.82	4.8	5.9	
22	Male	1.65	69	25.34	77	94	0.82	3.9	4.9	
65	Female	1.46	50	23.46	83	92	0.9	4.3	5.4	
57	Male	1.65	71	26.08	95	99	0.96	4.5	5.3	
50	Female	1.61	98	37.81	118	126	0.94	6.1	5.4	17.5
38	Female	1.59	107	42.32	121	128	0.95	4.9	8.1	36.12
65	Female	1.54	52	21.93	79	89	0.89	5	6.8	

70	Female	1.53	50	21.36	84	88	0.95	4.8	7.8	
60	Female	1.5	53	23.56	84	91	0.92	4.4	8.1	
70	Female	1.57	53	21.5	79	89	0.89	4.8	6.6	12.13
65	Female	1.42	45	22.32	75	85	0.88	4.7	5.6	
50	Female	1.55	61	25.39	89	95	0.94	4.6	6.3	
49	Female	1.54	85	35.84	104	118	0.88	4.2	4.7	
23	Male	1.68	68	24.09	77	93	0.83	4.2	8.2	
22	Male	1.84	63	18.61	73	87	0.84	4.3	6.4	
60	Female	1.58	59	23.63	93	90	1.03	4.6	7.5	13.54
65	Female	1.59	64	25.32	93	95	0.98	5.5	7.4	
70	Female	1.51	51	22.37	82	93	0.88	5.9	6.7	
27	Male	1.69	93	32.56	97	109	0.89	4.3	6	35.37
60	Female	1.55	60	24.97	87	97	0.9	4.8	6.9	
32	Female	1.53	76	32.47	97	107	0.91	4.2	6.3	
26	Female	1.51	52	22.81	68	89	0.76	4.2	4.4	
50	Female	1.59	67	26.5	84	100	0.84	4.4	5.8	
80	Female	1.51	67	29.38	102	98	1.04	5.1	7.2	
20	Male	1.66	76	27.58	74	94	0.79	4.7	3.3	14.05
31	Male	1.67	74	26.53	81	95	0.85	4.4	4.8	
22	Male	1.6	71	27.73	79	96	0.82	4.8	5.1	
23	Male	1.62	65	24.77	74	93	0.8	5.3	6.2	24.26
36	Female	1.51	73	32.02	101	111	0.91	4.5	5.7	19.88
21	Female	1.54	60	25.3	77	93	0.83	4.6	5.7	
53	Male	1.7	66	22.84	77	92	0.84	5.4	5.8	
70	Female	1.49	57	25.67	74	94	0.79	4.8	6.1	
53	Male	1.73	79	26.4	87	99	0.88	4.5	3.6	
26	Female	1.5	59	26.22	75	98	0.77	3.9	4.5	
29	Female	1.63	85	31.99	96	114	0.84	4.6	6.2	
64	Female	1.41	42	21.13	79	87	0.91	5.8	6.7	
23	Male	1.78	74	23.36	80	96	0.83	4.4	4.3	6.8
27	Female	1.55	54	22.48	66	85	0.78	5.3	5.2	22.4
61	Female	1.49	83	37.39	102	116	0.88	4.8	6.8	
44	Female	1.61	57	21.99	64	85	0.75	4.1	5.2	
60	Male	1.51	46	20.17	70	82	0.85	4.4	5.3	31.25
64	Female	1.49	49	22.07	78	89	0.88	4.8	5.8	
28	Female	1.55	67	27.89	87	100	0.87	4.2	5.2	
60	Female	1.44	53	25.56	76	87	0.87	4.2	4.9	
27	Male	1.7	65	22.49	70	88	0.8	4.4	4	12.76
35	Male	1.76	71	22.92	75	89	0.84	4.6	5.1	
25	Female	1.65	69	25.34	71	94	0.76	4.7	5.6	
27	Female	1.48	68	31.04	78	102	0.76	4.7	6.9	28.06
50	Female	1.55	62	25.81	88	102	0.86	5.3	6.1	
50	Female	1.48	52	23.74	84	90	0.93	5.4	6.9	
26	Male	1.63	67	25.22	79	92	0.86	4.8	5.9	
42	Male	1.68	72	25.51	86	97	0.89	4.5	6.5	
31	Male	1.52	58	25.1	76	89	0.85	4.4	5.9	
27	Male	1.82	109	32.91	109	117	0.93	4.9	5.9	
38	Male	1.81	77	23.5	79	98	0.81	4.3	3.7	
31	Male	1.6	97	37.89	97	108	0.9	5.5	4.9	27.94
65	Female	1.5	52	23.11	97	84	1.15	6.4	5.8	15.53
22	Female	1.45	62	29.49	77	94	0.82	4.6	4.7	
26	Male	1.69	73	25.56	80	90	0.89	7.6	5.4	
30	Female	1.54	70	29.52	76	95	0.8	4.6	5.9	
42	Female	1.57	75	30.43	93	107	0.87	4.2	5.5	13.93
52	Female	1.6	90	35.16	109	122	0.89	5	7	
46	Male	1.69	88	30.81	95	106	0.9	5.2	5.7	
65	Female	1.51	76	33.33	105	113	0.93	8	16.8	15.9
30	Male	1.65	55	20.2	72	89	0.81	4.2	5.3	
34	Male	1.75	69	22.53	76	95	0.8	4.2	4.4	17.82
40	Female	1.55	71	29.55	102	105	0.97	5.4	7.8	25.39
45	Female	1.48	49	22.37	71	82	0.87	4.6	5.8	
34	Female	1.54	56	23.61	78	94	0.83	3.8	5.6	

75	Female	1.52	57	24.67	92	103	0.89	3.9	6.3	
45	Female	1.5	94	41.78	108	128	0.84	4.1	6.4	
25	Female	1.5	68	30.22	88	97	0.91	4.9	6.8	
35	Female	1.71	63	21.55	71	93	0.76	4.1	6.1	
43	Female	1.56	82	33.69	90	106	0.85	4.9	6.6	18.43
30	Male	1.69	76	26.61	86	96	0.9	3.8	4.9	
25	Female	1.49	56	25.22	78	92	0.85	4.8	6.2	
20	Female	1.59	60	23.73	79	91	0.87	3.7	4.7	
59	Male	1.7	102	35.29	108	111	0.97	4.8	7.3	61.98
46	Male	1.76	75	24.21	85	94	0.9	4.3	4.2	
75	Female	1.49	73	32.88	107	112	0.96	5	5.1	
22	Male	1.75	75	24.49	77	97	0.79	4.5	6	6.62
50	Female	1.55	56	23.31	83	91	0.91	4.2	6.2	
35	Female	1.53	71	30.33	95	100	0.95	4.2	6.8	13.55
30	Male	1.64	60	22.31	80	96	0.83	4.3		
35	Male	1.63	69	25.97	80	97	0.82	4.7	5.6	
32	Male	1.88	75	21.22	74	98	0.76	4.5	5.5	
25	Male	1.7	73	25.26	79	94	0.84	4.9	6.3	
53	Male	1.66	84	30.48	105	104	1.01	4.8	7.7	
38	Male	1.65	61	22.41	76	90	0.84	4.5	6.5	
55	Female	1.56	55	22.6	84	92	0.91	4.2	5.6	
55	Male	1.6	84	32.81	99	108	0.92	5	4	12.58
29	Female	1.63	54	20.32	65	93	0.7	3.9	6.1	10.11
43	Male	1.64	72	26.77	88	98	0.9	5	7.4	
39	Male	1.65	77	28.28	91	98	0.93	4.1	5.3	
31	Female	1.54	89	37.53	108	116	0.93	4.9	6.1	
37	Female	1.5	89	39.56	106	120	0.88	4.1	7.4	15.93
40	Female	1.44	52	25.08	77	90	0.86	4.2		
40	Female	1.5	93	41.33	105	123	0.85	4.6	5.9	
50	Female	1.61	74	28.55	91	111	0.82	4.5	5.5	
44	Female	1.64	66	24.54	77	94	0.82	4.5	6.3	
30	Male	1.63	59	22.21	80	96	0.83	4.1		
26	Male	1.63	64	24.09	82	101	0.81	4.2	5.3	
31	Female	1.61	72	27.78	89	109	0.82	4.2	7.1	
25	Male	1.68	62	21.97	77	90	0.86	4.9	4.5	
28	Male	1.69	67	23.46	82	93	0.88	3.9	6.1	
70	Female	1.5	57	25.33	98	97	1.01	4.3	5.5	
40	Female	1.43	56	27.39	91	96	0.95	4.6	5.7	
28	Female	1.59	58	22.94	77	91	0.85	4.3	6.3	13.4
22	Female	1.57	60	24.34	73	94	0.78	5.1	6.5	
33	Male	1.65	71	26.08	90	104	0.87	4.7	6.1	
45	Female	1.53	100	42.72	120	141	0.85	10.7	15.6	33.32
50	Female	1.57	90	36.51	106	111	0.95	5.1	6.1	24.9
44	Female	1.48	74	33.78	96	111	0.86	4.7	7.1	
23	Female	1.61	75	28.93	87	102	0.85	5.1	5.3	
35	Female	1.59	62	24.52	77	96	0.8	4.2	5.4	21.51
25	Male	1.64	63	23.42	77	92	0.84	4.3	5.9	14.46
40	Male	1.65	61	22.41	78	90	0.87	4.6	3.5	
47	Female	1.59	89	35.2	97	113	0.86	5	5.3	18.48
60	Female	1.41	52	26.16	81	87	0.93	3.8	4.6	
65	Female	1.55	64	26.64	92	102	0.9	4.9	6	
60	Male	1.63	62	23.34	91	96	0.95	5.4	7.3	
45	Female	1.59	63	24.92	91	98	0.93	4.8	7.3	
58	Female	1.54	83	35	110	119	0.92	4.9	8.6	
40	Female	1.57	76	30.83	105	111	0.95	4.6	6.5	
30	Male	1.72	63	21.3	73	89	0.82	4.9	6.4	13.3
28	Male	1.7	65	22.49	75	97	0.77	4.6	4.9	
50	Female	1.62	65	24.77	85	99	0.86	4.2	5.4	
48	Female	1.6	85	33.2	103	112	0.92	5.4	5.8	26.99
51	Male	1.52	45	19.48	62	81	0.77	4.9	5.9	19.75
55	Male	1.55	58	24.14	92	98	0.94	5.9	12.1	
25	Female	1.56	63	25.89	78	100	0.78	4.9	5.2	

24	Female	1.5	46	20.44	69	83	0.83	4.1	5.4	
44	Male	1.59	62	24.52	79	88	0.9	4.6	8.5	
60	Male	1.75	71	23.18	83	97	0.86	5.8	6.5	
25	Male	1.59	50	19.78	63	87	0.72	4.1	7	7.43
43	Female	1.48	84	38.35	102	112	0.91	5.8	8.2	
88	Male	1.7	103	35.64	119	108	1.1	7.4	14.8	
75	Female	1.45	44	20.93	82	87	0.94	4.6	7.4	
56	Female	1.46	58	27.21	90	100	0.9	4.9	8.5	
75	Male	1.71	83	28.38	94	102	0.92	5.1	5.5	
56	Female	1.61	72	27.78	87	103	0.84	4.9	6.3	
25	Male	1.67	65	23.31	79	90	0.88	4.2	5.7	17.34
50	Female	1.5	65	28.89	90	100	0.9	4.8	5	
40	Female	1.58	74	29.64	97	104	0.93	4.6	7	
37	Female	1.52	72	31.16	94	107	0.88	5.2	6.4	
42	Female	1.44	66	31.83	92	105	0.88	4.2	7.7	15.9
68	Male	1.57	46	18.66	75	82	0.91	5.3	6.8	
28	Male	1.71	65	22.23	76	95	0.8	4.5	6.5	18.31
24	Male	1.72	62	20.96	72	91	0.79	4.3	7.6	
70	Male	1.63	63	23.71	92	93	0.99	4.1	5.6	
68	Male	1.57	46	18.66	75	82	0.91	5.3	6.8	
29	Male	1.73	60	20.05	70	91	0.77	3.6	4.9	9.25
61	Female	1.69	114	39.91	118	146	0.81	5.6	6.8	
48	Female	1.5	68	30.22	94	102	0.92	4	6.3	
33	Male	1.86	74	21.39	78	96	0.81	4.6	5.2	
32	Male	1.6	63	24.61	76	92	0.83	5.4	6.9	
30	Male	1.72	63	21.3	73	89	0.82	4.9	6.5	
28	Male	1.7	65	22.49	75	97	0.77	4.6	4.9	
50	Female	1.44	73	35.2	100	104	0.96	4.4	4.9	
51	Male	1.52	45	19.48	62	81	0.77	4.9	5.9	
55	Male	1.56	59	24.24	92	98	0.94	5.9	12.3	
38	Male	1.82	111	33.51	110	116	0.95	4.5	5.1	15.22
49	Female	1.68	93	32.95	116	122	0.95	4.4	6.7	12.59
75	Male	1.52	66	28.57	91	96	0.95	4.5	6.3	
30	Male	1.59	72	28.48	84	93	0.9	5.2	6.7	
34	Male	1.68	72	25.51	80	97	0.82	4.7	4.5	
30	Female	1.66	70	25.4	85	98	0.87	4.9	5.2	
62	Male	1.63	75	28.23	90	99	0.91	15.2		7.83
35	Male	1.76	74	23.89	78	96	0.81	4.9	5.5	
20	Male	1.79	66	20.6	73	91	0.8	4.9	7.6	
57	Male	1.55	60	24.97	86	93	0.92	5	4.9	
65	Female	1.54	65	27.41	85	98	0.87	4.9	5.7	
30	Female	1.57	55	22.31	80	90	0.89	5.4	6.1	
48	Female	1.68	110	38.97	114	133	0.86	5.2	6.6	13.44
47	Female	1.5	75	33.33	92	110	0.84	5.3	6.8	
32	Female	1.58	66	26.44	87	102	0.85	5.1	5.8	
26	Male	1.66	56	20.32	70	86	0.81	4.7	6.4	14.7
31	Male	1.74	67	22.13	74	94	0.79	4.2	4.8	11.49
31	Male	1.75	71	23.18	81	94	0.86	4.9	6.1	14.22
43	Male	1.65	75	27.55	86	94	0.91	4	6.3	
45	Female	1.62	68	25.91	88	102	0.86	5.1	5.3	
37	Female	1.58	61	24.44	74	93	0.8	5.1	5.7	12.65
38	Female	1.48	74	33.78	99	113	0.88	4.9	7.6	
38	Female	1.61	76	29.32	103	106	0.97	4.6	5.4	
29	Female	1.53	69	29.48	92	101	0.91	5.4	6.6	
25	Female	1.6	66	25.78	85	97	0.88	4.6	5.7	
56	Female	1.71	97	33.17	110	122	0.9	6.3	7.9	13.54
38	Male	1.82	111	33.51	110	116	0.95	4.6	5.2	
48	Female	1.59	58	22.94	82	95	0.86	4.4	6.1	
32	Female	1.66	72	26.13	85	107	0.79	4.3	4.2	
60	Female	1.53	91	38.87	121	120	1.01	11.4	14.8	8.74
25	Female	1.56	63	25.89	78	100	0.78	4.9	5.4	
24	Female	1.5	46	20.44	69	83	0.83	4.3	5.5	

44	Male	1.59	62	24.52	79	88	0.9	4.6	8.6	
45	Male	1.69	62	21.71	75	87	0.86	4.3	4.7	5.62
45	Female	1.59	69	27.29	89	100	0.89	5.2	6.8	
40	Female	1.58	86	34.45	93	114	0.82	6.1	7.5	7.4
65	Female	1.55	64	26.64	91	95	0.96	4.6	5.4	
39	Female	1.67	67	24.02	80	91	0.88	4.2	5.2	
48	Male	1.68	76	26.93	90	95	0.95	4.8	6.3	
45	Male	1.68	79	27.99	96	100	0.96	5.2	8.5	
35	Male	1.71	71	24.28	81	99	0.82	5.2	6.7	7.67
20	Female	1.55	55	22.89	66	87	0.76	4.6	5.5	
30	Female	1.53	49	20.93	63	84	0.75	4.5	5.1	8.47
65	Female	1.55	75	31.22	98	109	0.9	5.9	6.3	
26	Female	1.46	54	25.33	76	88	0.86	4.7	5.3	
52	Male	1.55	66	27.47	89	92	0.97	4.9	8.5	
57	Male	1.64	80	29.74	99	97	1.02	6.1	4.2	15.63
42	Male	1.75	75	24.49	81	100	0.81	4.3	4.8	
33	Male	1.58	58	23.23	75	86	0.87	3.7	5.4	
48	Male	1.72	83	28.06	95	100	0.95	4.4	6.5	
53	Female	1.51	49	21.49	79	87	0.91	4.7	6.6	
63	Male	1.56	63	25.89	90	93	0.97	4.7	12.2	
32	Female	1.61	67	25.85	76	95	0.8	4.4		
38	Female	1.56	65	26.71	82	104	0.79	4	5.8	
35	Male	1.63	69	25.97	80	97	0.82	4.6	5.6	
46	Female	1.58	55	22.03	66	93	0.71	4.1	5.9	3.17
30	Male	1.69	76	26.61	86	96	0.9	3.7	4.9	
28	Female	1.48	55	25.11	78	92	0.85	4.6	6.1	
20	Female	1.59	60	23.73	79	91	0.87	3.5	4.6	
49	Female	1.55	50	20.81	75	85	0.88	4.4	5.8	
32	Male	1.88	75	21.22	74	98	0.76	4.5	5.5	12.04
23	Male	1.7	73	25.26	79	94	0.84	4.4	6.2	
53	Male	1.66	84	30.48	105	104	1.01	4.6	7.7	
34	Female	1.59	72	28.48	79	103	0.77	4.7	5.4	
39	Male	1.65	61	22.41	76	90	0.84	4.4	6.3	10.92
54	Female	1.56	55	22.6	84	92	0.91	4.2	5.4	
65	Female	1.57	49	19.88	69	84	0.82	4.4	6.2	
31	Female	1.63	89	33.5	95	117	0.81	5.1	6.4	
30	Female	1.46	61	28.62	78	103	0.76	4.2	6.1	
50	Male	1.6	65	25.39	84	94	0.89	4.7	6.4	
28	Male	1.61	55	21.22	72	87	0.83	3.9	5.7	
37	Male	1.76	89	28.73	94	107	0.88	4.2	7.7	
37	Male	1.69	70	24.51	84	95	0.88	4.7	6.3	8.45
40	Female	1.56	80	32.87	96	104	0.92	4.7	4.9	
37	Female	1.52	65	28.13	82	96	0.85	4.4	6.2	
34	Female	1.57	78	31.64	103	108	0.95	4.8	5.2	
55	Male	1.76	80	25.83	92	98	0.94	4.5	5.4	
43	Male	1.75	69	22.53	78	93	0.84	4.3	7.1	8.46
57	Male	1.7	85	29.41	98	97	1.01	4.7	7.6	
28	Female	1.5	75	33.33	93	102	0.91	4.7	5.3	
31	Male	1.66	65	23.59	78	90	0.87	5.2	6.7	10.05
25	Male	1.78	76	23.99	80	96	0.83	4.9	6.4	
65	Female	1.57	63	25.56	88	104	0.85	4.9	6.9	
41	Female	1.55	72	29.97	89	115	0.77	4.5	6.2	
38	Female	1.52	53	22.94	82	93	0.88	4.7	6.3	
50	Female	1.46	50	23.46	84	90	0.93	5.1	6.7	
22	Male	1.63	67	25.22	79	92	0.86	4.9	5.8	
42	Male	1.68	72	25.51	86	97	0.89	4.5	6.5	
31	Male	1.52	58	25.1	76	89	0.85	4.4	5.8	
25	Male	1.82	109	32.91	109	117	0.93	4.7	5.7	
25	Female	1.59	74	29.27	86	105	0.82	5.2	7.5	20.35
38	Male	1.81	77	23.5	79	98	0.81	4.1	3.4	
30	Female	1.56	60	24.65	73	97	0.75	4.8	4.7	
27	Female	1.7	88	30.45	102	112	0.91	4.6	5.8	

38	Female	1.5	65	28.89	85	102	0.83	4.8	6.8	
26	Female	1.65	58	21.3	72	94	0.77	4.9	5.8	
52	Male	1.68	62	21.97	82	93	0.88	4.5	6.1	
24	Male	1.69	69	24.16	82	98	0.84	4.3	3.7	
70	Female	1.61	62	23.92	76	91	0.84	5.2	6.2	20.27
40	Male	1.72	75	25.35	89	101	0.88	4.2	3.8	
30	Male	1.68	72	25.51	83	100	0.83	4.8	4.6	
69	Male	1.66	64	23.23	80	93	0.86	4.7	5.6	
57	Male	1.55	59	24.56	86	90	0.96	4.1	5.8	
27	Female	1.59	72	28.48	82	106	0.77	4.2	5.9	
35	Female	1.5	50	22.22	73	88	0.83	4.3	5.3	
48	Male	1.7	63	21.8	76	91	0.84	4.7	6.4	
45	Male	1.6	80	31.25	111	108	1.03	4.7	3.9	
40	Female	1.5	73	32.44	95	106	0.9	4.7	5.4	
28	Female	1.5	64	28.44	86	103	0.83	4.2	5.9	
37	Male	1.82	96	28.98	103	110	0.94	5.4	6.9	12.09
35	Female	1.55	48	19.98	70	87	0.8	4.5	7.2	
32	Female	1.68	95	33.66	109	115	0.95	4.1	7.4	
28	Female	1.6	89	34.77	100	117	0.85	4.7	6.3	
36	Male	1.67	66	23.67	86	94	0.91	4.7	6.1	
65	Female	1.54	68	28.67	100	103	0.97	4.3	5.7	
29	Male	1.77	66	21.07	76	91	0.84	4.5		
35	Male	1.79	73	22.78	79	94	0.84	4.2	3.7	13.02
31	Male	1.81	73	22.28	81	93	0.87	4.4	3.6	11.73
27	Female	1.52	62	26.84	104	115	0.9	4.7	4.6	
26	Female	1.5	55	24.44	74	92	0.8	4.1	7.3	
33	Male	1.65	72	26.45	94	100	0.94	4.2	6.1	
51	Male	1.72	99	33.46	111	115	0.97	6.6	12.8	16
30	Female	1.64	82	30.49	99	113	0.88	4.6	5.5	
40	Female	1.52	70	30.3	89	100	0.89	4.8	4.3	
55	Male	1.71	75	25.65	90	96	0.94	4.7	3.4	
25	Male	1.64	80	29.74	93	104	0.89	4.1	4.3	
40	Female	1.53	43	18.37	73	82	0.89	4.1	5.4	
50	Female	1.43	72	35.21	100	104	0.96	4.2	4.7	12.76
32	Female	1.64	110	40.9	116	132	0.88	4.9	6.4	
22	Female	1.59	49	19.38	70	89	0.79	3.7	4.8	
22	Male	1.7	65	22.49	77	93	0.83	4.8	4.1	
56	Male	1.73	102	34.08	112	117	0.96	4.6	7.6	
49	Male	1.74	80	26.42	85	99	0.86	4.3	6.3	
32	Female	1.61	69	26.62	79	100	0.79	4.4	5.8	
35	Female	1.62	55	20.96	73	87	0.84	4.8	6.3	
51	Male	1.67	65	23.31	75	94	0.8	4.5	9.1	
40	Female	1.52	72	31.16	95	107	0.89	4.4	5.2	
30	Male	1.69	65	22.76	74	92	0.8	4.1	4.7	
28	Male	1.73	85	28.4	97	103	0.94	4.7	5.7	
23	Female	1.51	71	31.14	77	98	0.79	4.8	5.7	
20	Male	1.71	70	23.94	77	94	0.82	4.3	6.7	
37	Female	1.49	81	36.48	106	109	0.97	4.4	6.2	
25	Female	1.57	71	28.8	85	102	0.83	3.8	4.7	
23	Female	1.56	60	24.65	81	91	0.89	4.8	5.6	
33	Female	1.59	85	33.62	98	109	0.9	4.7	5.1	
35	Female	1.62	73	27.82	92	109	0.84	5.4	7.1	
40	Female	1.69	77	26.96	85	101	0.84	4.7	5.2	11.78
50	Female	1.5	82	36.44	104	116	0.9	4.7	7.4	
39	Female	1.62	75	28.58	89	108	0.82	4.6	7.1	
48	Female	1.41	63	31.69	90	102	0.88	5.3	8.1	
60	Female	1.54	63	26.56	83	95	0.87	4.8	6.8	
31	Male	1.86	74	21.39	78	96	0.81	4.6	5.1	19.09
50	Female	1.59	64	25.32	84	94	0.89	4.5	7.7	
68	Male	1.71	72	24.62	90	99	0.91	4.9	6.1	
29	Male	1.82	103	31.1	94	116	0.81	4.4	5.1	
55	Female	1.63	64	24.09	76	91	0.84	5.7	5.6	

48	Male	1.66	93	33.75	106	112	0.95	5.1	7.3	
54	Female	1.55	63	26.22	88	75	1.17	4.3	5.1	
38	Female	1.61	67	25.85	82	101	0.81	4.4	6.4	
43	Female	1.57	72	29.21	87	102	0.85	4.5	5.8	
38	Female	1.61	72	27.78	86	103	0.83	4.4	8.7	
45	Female	1.57	86	34.89	100	111	0.9	4.9	7.2	
70	Female	1.48	58	26.48	85	93	0.91	13.1		3.1
45	Female	1.53	61	26.06	85	90	0.94	4.4	8.7	
30	Female	1.59	64	25.32	75	92	0.82	4.7	5.3	
50	Female	1.56	67	27.53	90	95	0.95	4.7	7.1	
45	Male	1.71	82	28.04	84	101	0.83	4.6	5.9	
24	Female	1.51	61	26.75	72	95	0.76	5.8	6.6	
30	Male	1.78	87	27.46	89	103	0.86	3.9	5.6	
49	Female	1.58	85	34.05	97	110	0.88	5.1	6.9	
33	Male	1.59	70	27.69	78	92	0.85	4	6.1	
29	Male	1.65	72	26.45	84	96	0.88	6.1	6.9	4.95
43	Female	1.52	72	31.16	95	100	0.95	4.8	7.4	
34	Female	1.61	66	25.46	77	93	0.83	6.1	7.2	17.12
48	Male	1.61	85	32.79	91	103	0.88	5.3	7.1	
24	Male	1.79	79	24.66	84	97	0.87	4.6	4.7	
39	Female	1.6	82	32.03	98	111	0.88	4.6	6.1	
50	Female	1.48	51	23.28	66	84	0.79	4.3	5.4	
40	Female	1.6	90	35.16	104	110	0.95	4.6	5.9	
34	Female	1.68	83	29.41	100	109	0.92	5	7	
53	Male	1.7	66	22.84	77	92	0.84	5.1	5.9	
25	Female	1.69	95	33.26	106	118	0.9	5.8	7.2	
24	Female	1.59	55	21.76	69	92	0.75	5.5	5.8	
52	Male	1.66	82	29.76	108	104	1.04	5.4	12.5	
63	Female	1.62	79	30.1	107	110	0.97	7.8	11.6	23.1
43	Female	1.54	70	29.52	68	53	1.28	7.3	9.2	19.74
23	Female	1.65	66	24.24	77	101	0.76	4.4	6.4	
59	Male	1.66	58	21.05	90	94	0.96	5.8	3.9	
70	Female	1.54	60	25.3	88	104	0.85	5.5	4.5	
65	Female	1.56	46	18.9	75	89	0.84	4.8	7.8	
75	Male	1.66	65	23.59	92	92	1	5.8	6.6	
24	Female	1.53	58	24.78	69	96	0.72	3.8	6.4	
70	Female	1.49	63	28.38	92	107	0.86	14.3		
56	Female	1.62	55	20.96	93	95	0.98	3.7	5.5	
22	Male	1.6	62	24.22	77	95	0.81	4.6	4.8	
21	Male	1.65	55	20.2	72	89	0.81	5.2	5.8	
52	Female	1.62	83	31.63	96	111	0.86	4.8	5.4	
35	Female	1.57	73	29.62	80	100	0.8	4.4	5.4	25.82
75	Female	1.5	73	32.44	107	112	0.96	5	5.2	
21	Female	1.52	54	23.37	69	92	0.75	5.2		
35	Female	1.49	48	21.62	71	89	0.8	5.4	6.2	
30	Female	1.5	55	24.44	89	99	0.9	5	6.4	
60	Male	1.71	67	22.91	88	100	0.88	4.2	5.8	
23	Male	1.72	70	23.66	79	97	0.81	5.2	4.2	
54	Female	1.66	88	31.93	106	118	0.9	3.9	6.4	
27	Male	1.7	72	24.91	78	92	0.85	4.4	5.6	11.61
50	Male	1.53	50	21.36	67	80	0.84	4.7	7	8.4
70	Female	1.4	45	22.96	78	85	0.92	4.5	9.8	
40	Male	1.58	65	26.04	73	98	0.74	3.8	6	
50	Female	1.61	74	28.55	91	112	0.81	4.6	5.6	
27	Male	1.63	64	24.09	83	101	0.82	4.4	5.5	
25	Male	1.68	62	21.97	78	90	0.87	4.8	4.6	
28	Male	1.69	67	23.46	82	93	0.88	3.9	6.4	
70	Female	1.5	57	25.33	98	97	1.01	4.3	5.5	
28	Female	1.59	58	22.94	77	91	0.85	4.5	6.4	
23	Female	1.57	60	24.34	73	94	0.78	5.3	6.8	
47	Female	1.55	100	41.62	120	141	0.85	10.1	14.3	35.97
50	Female	1.57	90	36.51	106	112	0.95	5.1	6.3	

45	Male	1.49	74	33.33	96	112	0.86	4.7	7.2	
27	Male	1.64	63	23.42	78	92	0.85	4.3	5.9	
25	Male	1.59	50	19.78	63	87	0.72	4.2	7.1	
25	Male	1.67	65	23.31	79	90	0.88	4.3	5.9	
50	Female	1.51	66	28.95	90	100	0.9	4.9	5.1	
28	Male	1.72	66	22.31	76	95	0.8	4.5	6.5	
25	Male	1.72	62	20.96	72	91	0.79	4.5	7.7	
29	Female	1.59	70	27.69	80	102	0.78	4.7	5.8	19.32
70	Male	1.66	102	37.02	109	111	0.98	6.8	6.1	28.64
70	Male	1.63	63	23.71	92	93	0.99	4.3	5.8	
29	Male	1.74	61	20.15	70	91	0.77	3.8	4.9	
32	Male	1.59	72	28.48	84	93	0.9	5.2	6.9	
34	Male	1.68	72	25.51	80	97	0.82	4.8	4.7	
33	Male	1.58	58	23.23	75	86	0.87	3.8	5.6	
48	Male	1.72	83	28.06	95	100	0.95	4.6	6.5	
55	Female	1.51	49	21.49	79	87	0.91	4.8	6.8	
65	Male	1.56	63	25.89	90	93	0.97	4.9	12.6	
34	Female	1.61	67	25.85	76	95	0.8	4.8		
50	Male	1.6	65	25.39	84	94	0.89	4.9	6.8	
29	Male	1.61	55	21.22	72	87	0.83	3.9	5.8	
37	Male	1.76	89	28.73	94	107	0.88	4.6	7.7	
38	Male	1.69	70	24.51	84	95	0.88	4.8	6.2	
35	Female	1.57	78	31.64	103	108	0.95	4.8	5.6	
55	Male	1.76	80	25.83	92	98	0.94	4.7	5.8	
43	Male	1.75	69	22.53	78	93	0.84	4.8	7.4	
28	Female	1.56	84	34.52	95	110	0.86	4.4		25.16
58	Male	1.7	85	29.41	98	97	1.01	4.7	7.5	
29	Female	1.5	75	33.33	93	102	0.91	4.7	5.1	
31	Male	1.66	65	23.59	78	90	0.87	5.5	6.9	
26	Male	1.78	76	23.99	80	96	0.83	4.9	6.8	
27	Female	1.7	88	30.45	102	112	0.91	4.6	5.8	
26	Female	1.51	52	22.81	68	89	0.76	4.4	4.6	
51	Female	1.55	61	25.39	89	95	0.94	4.7	6.6	
25	Male	1.68	68	24.09	77	93	0.83	4.5	8.3	
35	Female	1.62	55	20.96	73	87	0.84	4.8	6.5	
51	Male	1.67	65	23.31	75	94	0.8	4.9	9.1	
30	Male	1.69	65	22.76	74	92	0.8	4.1	4.9	
28	Male	1.73	85	28.4	97	103	0.94	4.8	5.8	
49	Male	1.75	81	26.45	85	99	0.86	4.7	6.7	
58	Male	1.73	102	34.08	112	118	0.95	4.8	7.7	
75	Female	1.51	55	24.12	90	99	0.91	6	9.2	6.35
55	Male	1.71	75	25.65	90	96	0.94	4.9	3.7	
52	Male	1.72	99	33.46	111	115	0.97	6.8	12.2	15.92
35	Male	1.65	72	26.45	94	100	0.94	4.5	6.7	
23	Female	1.61	75	28.93	87	102	0.85	5.1	5.6	
40	Male	1.65	61	22.41	78	90	0.87	4.6	3.8	
35	Female	1.49	83	37.39	102	120	0.85	4.3	4.6	29.16
30	Female	1.49	50	22.52	69	86	0.8	4.2	4.8	12.34
24	Female	1.51	71	31.14	77	98	0.79	4.9	5.8	
21	Male	1.72	71	24	77	94	0.82	4.7	6.9	
37	Female	1.49	81	36.48	106	109	0.97	4.5	6.6	
23	Female	1.57	71	28.8	85	102	0.83	3.6	4.8	
46	Male	1.56	60	24.65	81	91	0.89	4.8	5.4	
27	Female	1.64	86	31.98	96	114	0.84	4.5	6.1	
52	Female	1.5	82	36.44	104	116	0.9	4.4	7.3	
50	Female	1.59	64	25.32	84	94	0.89	4.7	7.5	
64	Male	1.72	73	24.68	90	99	0.91	4.8	6.5	
28	Male	1.82	103	31.1	94	116	0.81	4.8	5.4	
55	Female	1.63	64	24.09	76	91	0.84	5.8	5.8	
48	Male	1.66	93	33.75	106	112	0.95	5.3	7.6	
56	Female	1.55	63	26.22	88	95	0.93	4.5	5.4	
37	Female	1.61	72	27.78	86	103	0.83	4.8	8.9	

72	Female	1.49	59	26.58	87	92	0.95	14.9		6.13
50	Female	1.56	67	27.53	90	95	0.95	4.5	7.4	
47	Male	1.71	82	28.04	84	101	0.83	4.7	5.2	
30	Male	1.78	87	27.46	89	103	0.86	3.7	5.8	
33	Male	1.59	70	27.69	78	92	0.85	4.3	6.5	
26	Male	1.65	72	26.45	84	96	0.88	5.6	6.8	
48	Female	1.51	87	38.16	110	126	0.87	5.8	8	
49	Male	1.61	85	32.79	91	103	0.88	5.1	7.2	
27	Male	1.79	79	24.66	84	97	0.87	4.5	4.6	
70	Male	1.68	70	24.8	94	100	0.94	5.2	6	
60	Female	1.45	60	28.54	90	102	0.88	5.9	6.8	
34	Male	1.64	80	29.74	95	108	0.88	4.6	4.3	
56	Male	1.64	68	25.28	89	97	0.92	6.1	6.9	16.13
74	Male	1.54	53	22.35	82	93	0.88	5.1	5.9	
66	Male	1.63	80	30.11	105	112	0.94	4.4	9.2	
68	Male	1.61	79	30.48	106	110	0.96	4.8	8.3	
52	Male	1.61	74	28.55	97	107	0.91	5.1	4.6	
50	Female	1.58	67	26.84	95	106	0.9	5	6.4	
32	Female	1.52	63	27.27	90	97	0.93	4.7	5.6	
70	Female	1.55	50	20.81	80	88	0.91	4.6	6.3	
54	Female	1.64	87	32.35	119	117	1.02	4.6	5.2	
36	Female	1.51	68	29.82	98	107	0.92	4.5	7.2	
40	Female	1.54	77	32.47	100	109	0.92	5.1	6.4	
48	Female	1.52	51	22.07	76	93	0.82	4.8	5.6	
38	Female	1.56	50	20.55	73	87	0.84	4.2	5.7	
73	Male	1.53	67	28.62	91	96	0.95	4.5	6.6	
40	Female	1.56	49	20.13	82	87	0.94	4.4		
36	Female	1.56	51	20.96	76	87	0.87	4.4	5.3	
40	Female	1.48	72	32.87	101	109	0.93	4.4	5.9	
34	Female	1.53	62	26.49	86	95	0.91	3.8	3.7	
47	Female	1.64	85	31.6	110	123	0.89	5.3	6.8	
42	Male	1.78	83	26.2	98	104	0.94	4.6	6.1	
37	Female	1.6	66	25.78	92	100	0.92	4.6	5.6	
70	Female	1.56	58	23.83	89	97	0.92	4.7	5	
33	Female	1.6	80	31.25	96	110	0.87	5	5.6	
39	Male	1.76	68	21.95	83	95	0.87	4.2	4.3	
48	Female	1.5	61	27.11	86	105	0.82	4.9	6.6	
45	Male	1.62	63	24.01	85	95	0.89	4.7	5.2	
44	Female	1.59	68	26.9	90	103	0.87	4.1	5.6	
45	Female	1.6	63	24.61	76	105	0.72	4	7.7	
48	Female	1.48	65	29.67	98	107	0.92	4.9	7	
35	Female	1.62	93	35.44	102	121	0.84	4.8	7.1	
42	Female	1.57	75	30.43	101	110	0.92	4.8	5.8	
31	Male	1.6	63	24.61	76	92	0.83	5.4	6.8	15.86
49	Male	1.61	58	22.38	83	69	1.2	4.9	5.5	
40	Female	1.6	55	21.48	71	94	0.76	4.5	5.6	
60	Female	1.59	63	24.92	84	96	0.88	4.8	5.8	
33	Male	1.73	81	27.06	92	104	0.88	4.4	5.2	
50	Female	1.45	77	36.62	110	127	0.87	4.8	6.2	
65	Female	1.61	82	31.63	104	116	0.9	5.2	8.2	
33	Female	1.62	62	23.62	84	100	0.84	3.9	4.1	
52	Male	1.61	56	21.6	73	89	0.82	4.6	6.1	
41	Female	1.6	89	34.77	111	116	0.96	4.7	6.3	
60	Female	1.6	83	32.42	99	112	0.88	6.1	8.4	30.1
50	Male	1.65	73	26.81	92	98	0.94	5.1	6	
62	Male	1.67	51	18.29	73	84	0.87	4.2	6.4	
27	Female	1.52	57	24.67	74	96	0.77	4.3	6.3	
34	Male	1.67	71	25.46	90	100	0.9	4.4	6.3	
36	Female	1.69	80	28.01	99	106	0.93	4.5	4.5	
41	Female	1.5	56	24.89	82	98	0.84	4.2		
45	Female	1.68	66	23.38	86	99	0.87	4.1	5.5	
38	Female	1.6	74	28.91	84	114	0.74	4.4	6.1	

45	Female	1.58	58	23.23	86	91	0.95	4.2	4.8	
35	Female	1.59	75	29.67	93	107	0.87	4.4	6.7	
40	Female	1.47	80	37.02	122	117	1.04	4.8	8.1	
39	Female	1.67	65	23.31	80	103	0.78	3.6	6.3	
40	Female	1.51	53	23.24	84	98	0.86	4.4	6.3	
46	Female	1.54	68	28.67	92	100	0.92	4.9	6.3	
60	Female	1.49	50	22.52	83	91	0.91	4.9	6.2	
52	Female	1.54	60	25.3	91	101	0.9	4.4	6.2	
22	Male	1.8	71	21.91	80	90	0.89	4.6	5.4	
37	Male	1.66	57	20.69	81	86	0.94	4.5	5.3	
40	Female	1.55	67	27.89	91	102	0.89	4.9	6	
33	Female	1.58	50	20.03	75	91	0.82	4.4	5.3	
70	Female	1.51	46	20.17	82	91	0.9	6.1	6.7	9.75
56	Male	1.69	63	22.06	85	88	0.97	4.4	5.5	
30	Male	1.6	59	23.05	72	91	0.79	3.7	5.6	
46	Female	1.55	80	33.3	95	118	0.81	4.2	5.3	
37	Male	1.67	56	20.08	73	88	0.83	3.9	7.2	
52	Male	1.67	73	26.18	101	103	0.98	4.9	5.2	
42	Female	1.53	91	38.87	104	125	0.83	4.7	5.5	
31	Male	1.66	65	23.59	77	97	0.79	5.1	8.9	
65	Female	1.53	40	17.09	65	78	0.83	4	6.8	
55	Female	1.43	42	20.54	72	89	0.81	4.8	5.8	
36	Female	1.58	55	22.03	72	90	0.8	4.3	6	
65	Female	1.6	71	27.73	105	108	0.97	4.6	10.4	
47	Female	1.52	48	20.78	69	89	0.78	4.4	5.3	
40	Female	1.56	47	19.31	75	84	0.89	4.3	6.2	
28	Male	1.56	67	27.53	85	99	0.86	4.7	6	
60	Female	1.54	59	24.88	88	99	0.89	5.6	7.3	
60	Female	1.55	56	23.31	91	95	0.96	6.2	7.9	24.2
57	Female	1.47	68	31.47	98	105	0.93	7.3	11.4	12.01
60	Female	1.68	75	26.57	90	106	0.85	13.8		15.5
42	Female	1.55	72	29.97	90	102	0.88	4.9	5.5	
57	Male	1.68	66	23.38	97	87	1.11	4.3	5.1	
60	Female	1.65	50	18.37	70	88	0.8	5.2	5.7	
60	Male	1.62	56	21.34	81	89	0.91	4.3	3.2	
29	Female	1.63	85	31.99	96	107	0.9	4.7	5.1	
43	Female	1.6	77	30.08	92	109	0.84	4.7	7.1	
56	Male	1.64	74	27.51	94	105	0.9	3.6	8.2	
43	Female	1.62	73	27.82	97	106	0.92	4.9	5.8	
39	Female	1.61	74	28.55	91	104	0.88	4.5	6.3	
60	Female	1.5	71	31.56	108	109	0.99	6	10.5	23.9
52	Male	1.56	69	28.35	92	97	0.95	4.7	6	
50	Female	1.6	58	22.66	75	91	0.82	4.7	6.1	
65	Female	1.62	65	24.77	98	97	1.01	4.4	3.7	
32	Female	1.61	66	25.46	84	97	0.87	4.8	6.6	
39	Female	1.64	65	24.17	79	98	0.81	4.4	5.1	
60	Female	1.6	53	20.7	76	89	0.85	4.3	5.4	
22	Female	1.67	51	18.29	69	88	0.78	3.8	4.4	
40	Female	1.51	51	22.37	76	90	0.84	5.2	6.5	
45	Female	1.67	48	17.21	69	85	0.81	4.2	4.7	
60	Female	1.59	46	18.2	72	84	0.86	4.8	4.8	
43	Male	1.68	65	23.03	77	95	0.81	4.4	4.8	
28	Female	1.63	68	25.59	77	96	0.8	4.7	6.1	
48	Female	1.61	53	20.45	70	88	0.8	5.1	5.7	
38	Female	1.58	74	29.64	87	105	0.83	4.9	6	
45	Female	1.54	77	32.47	91	111	0.82	4.8	5.2	
50	Female	1.62	74	28.2	94	111	0.85	4.9	3.4	
67	Male	1.7	47	16.26	68	79	0.86	5	5.8	
68	Female	1.61	68	26.23	92	101	0.91	5.3	5.9	
65	Female	1.56	64	26.3	85	105	0.81	5.3	6.2	
51	Male	1.67	64	22.95	84	93	0.9	5	6.4	
45	Female	1.62	75	28.58	90	106	0.85	4.3	5.1	

42	Female	1.49	48	21.62	78	89	0.88	4.8	4.9	
23	Female	1.5	75	33.33	85	104	0.82	5.1		
65	Female	1.53	44	18.8	64	85	0.75	5.6	10.1	
27	Female	1.6	52	20.31	71	86	0.83	3.9	4.3	
25	Female	1.6	53	20.7	68	89	0.76	5.2	5.7	
35	Female	1.64	61	22.68	76	91	0.84	4.8	5.7	
70	Female	1.52	47	20.34	76	90	0.84	4.6	6.9	
49	Female	1.63	63	23.71	86	97	0.89	4.8	5.7	
32	Female	1.64	61	22.68	78	95	0.82	4.9	5.3	
35	Female	1.53	54	23.07	74	93	0.8	4.9	7.6	
50	Female	1.55	67	27.89	94	106	0.89	4.9	6.4	
55	Female	1.49	76	34.23	96	118	0.81	4.8	4.7	
50	Female	1.55	49	20.4	76	90	0.84	4.2	5.2	
30	Female	1.63	66	24.84	77	99	0.78	4.7	4.8	
38	Female	1.58	55	22.03	78	89	0.88	4.8	4.6	
48	Female	1.73	60	20.05	71	92	0.77	5.6	6.7	
55	Female	1.52	50	21.64	72	95	0.76	4.8	9.5	
50	Female	1.67	71	25.46	89	103	0.86	4.7	5.7	
32	Female	1.56	56	23.01	88	90	0.98	5.3	5.7	
60	Female	1.46	57	26.74	87	100	0.87	5.3	6.9	
39	Female	1.57	70	28.4	94	103	0.91	4.5	5.8	
43	Female	1.61	69	26.62	88	103	0.85	4.8	5.2	
42	Female	1.55	56	23.31	78	99	0.79	4.1	4.4	
65	Male	1.61	67	25.85	96	101	0.95	4.6	5.9	
32	Female	1.55	50	20.81	74	85	0.87	4.7	5.9	
60	Female	1.58	82	32.85	103	115	0.9	4.1	6	
75	Male	1.7	68	23.53	90	95	0.95	5.3	5.2	
74	Male	1.68	70	24.8	95	102	0.93	5.5	7	
50	Male	1.64	68	25.28	89	97	0.92	6.2	7.1	
63	Male	1.61	79	30.48	106	110	0.96	4.8	8.6	
71	Female	1.51	57	25	90	99	0.91	6.2	10.2	27.35
45	Male	1.78	83	26.2	98	104	0.94	4.8	6.4	
46	Male	1.62	65	24.77	85	95	0.89	4.9	5.8	
42	Male	1.61	58	22.38	83	70	1.19	5.1	6.5	
68	Female	1.61	82	31.63	105	117	0.9	5.4	8.6	
65	Male	1.67	55	19.72	73	84	0.87	5.2	7.4	
29	Female	1.52	57	24.67	74	96	0.77	4.6	6.6	
50	Male	1.67	77	27.61	101	103	0.98	4.9	5.9	
33	Male	1.66	65	23.59	79	97	0.81	5.3	9.8	
60	Male	1.63	72	27.1	91	96	0.95	5.4	7.4	
59	Male	1.68	68	24.09	97	88	1.1	4.5	5.8	
61	Male	1.62	65	24.77	81	89	0.91	4.8	6.2	
27	Female	1.63	85	31.99	96	107	0.9	4.7	5.4	
55	Male	1.64	74	27.51	94	105	0.9	4.6	8.2	
53	Male	1.56	70	28.76	92	98	0.94	4.6	6.5	
46	Male	1.68	65	23.03	78	95	0.82	4.5	5.8	
66	Female	1.58	49	19.63	65	85	0.76	5.8	9.9	
62	Male	1.61	67	25.85	96	101	0.95	4.9	6.7	
65	Male	1.65	75	27.55	87	99	0.88	4.3	5.3	
22	Male	1.6	72	28.13	80	97	0.82	4.8	5.3	
27	Male	1.62	65	24.77	74	93	0.8	5.1	6	
20	Female	1.56	62	25.48	77	93	0.83	4.7	5.9	
26	Female	1.59	70	27.69	80	102	0.78	4.7	6.8	
71	Male	1.66	100	36.29	109	111	0.98	6.5	7.1	32.39
70	Female	1.51	59	25.88	74	94	0.79	4.8	6.3	
68	Female	1.43	44	21.52	79	87	0.91	6	7.6	
24	Male	1.78	74	23.36	80	96	0.83	4	5.3	
57	Male	1.7	102	35.29	108	111	0.97	4.6	7.5	
48	Male	1.76	75	24.21	85	94	0.9	4.3	4.7	
25	Male	1.75	75	24.49	77	97	0.79	4.6	6.4	
56	Male	1.6	84	32.81	99	108	0.92	5	5.4	
26	Female	1.59	74	29.27	86	105	0.82	5	7.8	

44	Male	1.64	72	26.77	88	98	0.9	5	7.9
38	Male	1.65	77	28.28	91	98	0.93	4.8	5.7
67	Male	1.65	77	28.28	90	99	0.91	12.9	
35	Male	1.76	74	23.89	78	96	0.81	4.9	5.8
57	Male	1.55	60	24.97	86	93	0.92	5	5.9
65	Female	1.54	65	27.41	85	98	0.87	4.6	5.8
39	Male	1.8	95	29.32	103	110	0.94	5.3	7.2
25	Male	1.66	56	20.32	70	86	0.81	4.5	6.6
33	Male	1.74	67	22.13	74	94	0.79	4.5	4.9
34	Male	1.75	71	23.18	81	94	0.86	4.6	6.3
46	Male	1.65	75	27.55	86	94	0.91	4	6.7
48	Male	1.68	76	26.93	90	95	0.95	4.9	6.7
46	Male	1.68	79	27.99	96	100	0.96	5.3	8.9
35	Male	1.71	71	24.28	81	99	0.82	5	7
28	Male	1.7	72	24.91	78	92	0.85	4.5	5.8
52	Male	1.54	51	21.5	67	80	0.84	4.1	7.2
50	Male	1.55	68	28.3	89	93	0.96	5.9	9.5
65	Female	1.55	55	22.89	79	87	0.91	5.8	6.8
36	Male	1.59	59	23.34	76	86	0.88	3.8	5.6
48	Male	1.72	83	28.06	95	100	0.95	3.9	6.3
35	Male	1.65	73	26.81	94	100	0.94	4.6	6.4
54	Male	1.72	99	33.46	112	115	0.97	6.9	11.7
42	Male	1.75	75	24.49	81	100	0.81	4.6	4.7
29	Female	1.59	72	28.48	82	106	0.77	4	6.1
53	Male	1.68	62	21.97	82	93	0.88	4.5	6.1
41	Male	1.72	75	25.35	89	101	0.88	5.2	4.8
30	Male	1.68	72	25.51	83	100	0.83	4.4	4.1
67	Male	1.67	65	23.31	80	93	0.86	4.5	6.5
36	Male	1.67	66	23.67	86	94	0.91	4.8	6.3
27	Female	1.5	64	28.44	86	103	0.83	4.1	6.2