

**PREVALENCE AND CORRELATES OF DIABETES
MELLITUS AND GLUCOSE INTOLERANCE AMONG
ADULTS IN KANO METROPOLIS**

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

BY

DR LABARAN YUSUF ABUBAKAR, BM BCh (Jos) 1995

DEPARTMENT OF INTERNAL MEDICINE

AMINU KANO TEACHING HOSPITAL, KANO NIGERIA.

NOVEMBER, 2012.

DECLARATION

It is hereby declared that this work is original unless otherwise acknowledged. The work has not been submitted in part or in full to any other examining body for an award, nor has it been submitted elsewhere for publication.

CERTIFICATION 1

SUPERVISOR'S CERTIFICATION

The study reported in this dissertation was carried out by Dr Abubakar Labaran Yusuf under our supervision. We also supervised the writing of this dissertation.

SUPERVISORS:

1. DR F H PUEPET, *FMCP, FACE*

Associate professor,
Consultant Physician and Endocrinologist
Department of Medicine
Jos University Teaching Hospital (JUTH)
P M B 2076 Jos
Plateau State, Nigeria

Signature.....

Date.....

2. DR M.M. Borodo, *FMCP*

Associate professor,
Consultant Physician
Department of Medicine
Aminu Kano Teaching Hospital (AKTH)
P M B 3452
Kano State, Nigeria.

Signature.....

Date.....

3 DR A E ULOKO *FMCP, FACE*

Senior lecturer,
Consultant Physician and Endocrinologist
Department of Medicine
Aminu Kano Teaching Hospital (AKTH)
P M B 3452
Kano State, Nigeria.

Signature.....

Date.....

CERTIFICATION II

HEAD OF DEPARTMENT'S CERTIFICATION

I hereby certify that this study was carried out by Dr Abubakar Labaran Yusuf of the department of Medicine , Aminu Kano Teaching Hospital under the supervision of Dr F H Puepet, Dr M M Borodo and Dr A E Uloko.

DR MAHMUD UMAR SANI

HEAD

DEPARTMENT OF MEDICINE

AMINU KANO TEACHING HOSPITAL

KANO STATE

NIGERIA

NOVEMBER 2012

iv

TABLE OF CONTENTS

Title page

cover page

Declaration	ii
Certification I	iii
Certification II	iv
Table of contents	v
Expanded table of contents	vi
Dedication	xi
Acknowledgements	xii
List of Abbreviations	xiii
List of tables	xiv
List of figures	xv
Summary	1
CHAPTER ONE INTRODUCTION	3
CHAPTER TWO LITERATURE REVIEW	11
CHAPTER THREE SUBJECTS, MATERIALS AND METHODS	27
CHAPTER FIVE RESULTS	35
CHAPTER SIX DISCUSSION	70
CONCLUSION AND RECOMMENDATIONS	76
REFERENCES	77
APPENDIX	87

v
EXPANDED TABLE OF CONTENTS

CHAPTER ONE INTRODUCTION	3
1.1 Brief history of Diabetes mellitus	3
1.2 Definition of Diabetes mellitus	4
1.3 Diagnosis of Diabetes mellitus	5
1.4 Classification of Diabetes mellitus	6
1.5 Burden of Diabetes mellitus	8
1.6 Justification for the study	9
1.7 .0 Aims and objectives of the study	10
CHAPTER TWO LITERATURE REVIEW	11
2.0 Introduction	11
2.1 Prevalence of Diabetes mellitus	11
2.1.1 World prevalence	12
2.1.2 Prevalence of Diabetes mellitus in Nigeria	15
2.1.3 prevalence of Diabetes mellitus in Kano	15
2.2.0 Prevalence of Glucose intolerance	15
2.2.1 Prevalence of Glucose intolerance world wide	16
2.3.0 Risk factors for Diabetes mellitus	16
2.4.0 Aetiopathogenesis of Diabetes mellitus	20
2.4.1 Type 1 Diabetes mellitus	20
2.4.1.1 Autoimmune Type 1 Diabetes mellitus	20
2.4.1.2 Idiopathic Type 1 Diabetes mellitus	21
2.4.2 Type 2 Diabetes mellitus	21

2.4.2.1 insulin resistance	21
2.4.2.2 insulin secretory defect	22
2.5.0 Methods of screening for Dm	23
2.5.1 Screening tests	23
Chapter Three	26
3.0 Subjects materials and methods	26
3.1 Study Area	26
3.2 Study design	27
3.3 study population	27
3.4 Sample size determination	27
3.4.1 Inclusion criteria	27
3.4.2 Exclusion criteria	27
3.5. Sampling method	28
3.6 Ethical approval	28
3.7.0 Instrument/ materials/equipment/method of data collection	29
3.8 Limitation of the study	35
3.9 Data analysis and statistics	35
CHAPTER FOUR RESULTS	36
4.1 Quality of the data	36
4.2 Socio-demographic characteristic of the study subjects	36
4.3 Clinical characteristics of the participants	41 4.4
Anthropometric indices of the study subjects	42

4.5 Prevalence of Diabetes and Glucose intolerance	46
4.6 Socio-demographic characteristics of the subjects with diabetes and IGT	54
4.7 Clinical characteristics of the subjects with diabetes and IGT	54
4.8 Risk factors and occurrence of DM in the study	59
4.9 Risk factors among subjects with IGT	65
4.10 Odd ratio estimate of risk factors from multiple regression	66
4.11 Risk factor profile of subjects with DM, IGT and normal subjects	67
CHAPTER FIVE Discussions	68
5.0 Background	68
5.1 prevalence of DM in the study	68
5.2 Type of DM in the study	70
5.3 Prevalence of Glucose Intolerance in the study	70
5.4 Correlates of DM from the study	70
5.4.1 Age and the prevalence of diabetes	71
5.4.2 Obesity and the prevalence of DM	71
5.4.3 Hypertension and the prevalence of DM	71
5.4.4 Physical inactivity and the prevalence of Dm	71
5.4.5 Parental history of DM and the prevalence DM	72
5.4.6 Social history and the prevalence of DM	72
5.4.7 Alcohol use and the prevalence of DM	72
5.4.8 Cigarette smoking and prevalence of DM	72

CHAPTER SIX CONCLUSION AND RECOMMENDATIONS

6.0 conclusion	73
6.1 Recommendations	73
References	74
Appendices	84
Appendix 1: Pretesting of the questionnaire and methodology	84
Appendix 2: Blood pressure measurement	85
Appendix 3: Anthropometric measurements	86
Appendix 4: Blood sample taking and laboratory procedure	88
Appendix 5: Protocol for oral glucose tolerance test	91
Appendix 6: Questionnaire	92
Appendix 7: Consent form	95
Appendix 8: Ethical Approval	96
Appendix 9: Raw Data	97

DEDICATION

This work is dedicated to all the people living with diabetes and the people that positively affect their lives worldwide.

x

ACKNOWLEDGEMENTS

I give honour and praise to Almighty God who in his infinite mercy gave me the opportunity and privilege to complete this work successfully.

I am very grateful to my parents for their unrelenting prayers, support and encouragement. I am grateful to my wife Nafisat A Tukur and my children Saudat and Muhammad for their love, prayers and understanding at all times.

I am very grateful to Dr F. H. Puepet my teacher and mentor, for his guidance, understanding, and kindness.

I am thankful to Dr M. M. Borodo for his tutelage, guidance and his fatherly advice.

I am very grateful to DR A. E. Uloko for his immense help in all my affairs and encouragement, I feel indebted.

A big thank you to Drs A. M. Jibo, M.U. Lawan, A. U. Gajida , and Z. Iliyasu all of the Department of community medicine, Drs K. M. Karaye, B. M. Tijjani, Aliyu Abdu and A. G. Habib all of the department of medicine, for your constructive criticism, guidance and advices, Drs Isa Yahaya, Anthony Gadzama, and Alhaji Dadin Kowa all of chemical pathology Department, for the support and cooperation.

The staff of the department of health Tarauni LGC particularly Alhaji Bello Dandago and others from the five health centers, Cipla-Evans Nigeria LTD, Mega life sciences and Novartis Pharmaceutical Limited need to be mentioned, I am very grateful for assistance and the contributions both in cash and kind.

ABBREVIATION

2HPGL.....	2 Hour post Glucose load
CPG.....	Casual plasma glucose
DM.....	Diabetes mellitus
FPG.....	Fasting plasma glucose
IDDM.....	Insulin Dependant Diabetes mellitus
IFG.....	Impaired Fasting glucose
IGT.....	Impaired glucose tolerance
L G A.....	Local government Area
M O D Y.....	Maturity onset diabetes of the young
M R D M	Malnutrition related diabetes mellitus
NCD.....	Non communicable disease
NIDDM.....	Non insulin-dependent diabetes mellitus
RPG.....	Random plasma glucose
S D.....	Standard Deviation
OGTT.....	Oral glucose tolerance test
UKPDS.....	United Kingdom Prospective Diabetes Study
W H O	World Health Organisation

LIST OF TABLE

Table 1.3.1 Diagnosis of Diabetes Mellitus	5
Table 3.1 Classification of Social Class	34
Table 4.1 Age and sex distribution of the study subjects	37
Table 4.2 Distribution of the subjects by social class	40
Table 4.3 Level of physical activity among the study subjects	41
Table 4.4 Distribution of CPG by sex among the subjects	45
Table 4.5 Standardization of prevalence rate	47
Table 4.6 Age standardised prevalence of Dm by sex	48
Table 4.7 Distribution of CPG among the subjects with diabetes	50
Table 4.8 Age distribution of the subjects with diabetes by sex	52
Table 4.9 Relationship between Age and Diabetes mellitus	59
Table 4.10 Relationship between Weight and Diabetes mellitus	60
Table 4.11 Relationship between physical inactivity and Diabetes mellitus	61
Table 4.12 Relationship between social class and Diabetes mellitus	62
Table 4.13 Prevalence of hypertension among subjects with diabetes mellitus	64
Table 4.14 Odd ratio estimates of risk factors for DM multiple logistic regression	66
Table 4.15 Mean CPG and risk factor profile of subjects with DM, IGT and normal	67

FIGURES

Figure I Occupational distribution of the study subjects	38
Figure II Distribution of study subjects by ethnic group	39
Figure III Distribution of BMI of the study subjects by sex	43
Figure IV Distribution of BMI by sex among the subjects with diabetes	56
Figure V Distribution of BMI by sex among the subjects with IGT	57

SUMMARY

BACKGROUND: Diabetes Mellitus (DM) is assuming a pandemic dimension. The number of cases of diabetes worldwide among adults 20 years and above in 2010 was estimated to be 285 million and the number will increase to 438 million by the year 2030. The major part of this numerical increase will occur in the developing countries⁴, Nigeria included. There is a paucity of data regarding the prevalence of DM in northern Nigeria in particular and the country in general, the latest data for the country was from the Expert committee report on Noncommunicable Disease (NCD) survey of 1997. This study is intended to contribute to data base for the national prevalence of DM.

STUDY OBJECTIVES: To determine the prevalence and correlates of DM and Glucose intolerance among adults in Kano.

SUBJECTS AND METHODS: In a multistage (3 stages) randomized cluster sampling scheme, 684 subjects were screened for diabetes mellitus according to WHO standard using casual plasma glucose. Data obtained included demography, anthropometry, blood pressure measurement and casual plasma glucose. The data was subsequently analysed using simple descriptive statistics with EPI-INFO 3.6 and Mini Tab 12 statistical packages.

Correlates of DM and IGT were determined. P value of less than 0.05 was considered to be statistically significant. IGT was determined through administration of 82g of glucose D to the subjects with CPG between 7.8mmol/l and 11.0mmol/l in fasting state and their 2hour post glucose load (2HPGL) estimated. Subjects with 2HPGL 7.8mmol/l but less than 11.1mmol/l were diagnosed as having IGT.

RESULTS: A total of 700 subjects were recruited of which 684 were evaluated, giving a response rate of 97.7%. Two hundred and fifty (36.5%) were males and 434 (63.5%) females. Of these 684 subjects, 59 subjects were found to have diabetes (19 males and 40 females), giving a crude total prevalence rate of 8.6%, crude male prevalence was 7.6% and crude female prevalence was 9.2 %, giving male : female ratio of 1 : 1.2, when adjusted for age and sex the overall age standardized prevalence was 6.9% (95% CI 5.0% - 8.8%). The male age standardized prevalence dropped to 6.4% (95% CI 2.9% - 9.4%) while that of females dropped to 8.8% (95% CI 4.0%- 13.6%). Twenty eight subjects (4.1%) were known to be living with diabetes before this study, while 31 (4.5%) subjects were newly diagnosed with diabetes during this study. The predominant type of DM was type 2 DM accounting for 95.5%.

Fourty two (42) subjects were found to have IGT given a prevalence of 6.2%, 10 (1.5%) males and 32 (4.7%) females.

Uni-variate analysis indicated that increased body mass index (BMI), waist circumference (WC) , waist hip ratio (WHR), physical inactivity, parental history of DM, and age were significant risk factors for DM and glucose intolerance. However, logistic regression analysis showed that age and BMI were the only independent risk factors for DM after adjusting for the other risk factors. In contrast, social class, alcohol consumption and cigarette smoking did not appear to be associated risk factors for DM or IGT in this study.

Conclusions: The prevalence of DM among adults in Kano appears higher than previously reported with age and higher BMI as independent risk factors. The predominant form of DM in this study was Type 2 DM.

CHAPTER ONE

1.0 INTRODUCTION

1.1 BRIEF HISTORY OF DIABETES MELLITUS

The earliest known record of diabetes was mentioned on the 3rd Dynasty Egyptian papyrus by a physician, Hesy-Ra; who mentioned polyuria (frequent urination) as a symptom in 1552 BC.^{1,2}

In the 1st century Arateus of Cappadocia described the disease as 'the melting down of flesh and limbs into urine and coined the word “diabetes” meaning “to run through” or “a siphon”.¹

In 164 AD, Greek physician Galen of Pergamum mistakenly diagnosed diabetes as an ailment of the kidneys and employed alternative terms for diabetes “diarrhoea urinosa and dipsakos” emphasizing the cardinal symptoms of excessive urination and drinking². Two notable Indian physicians, Susruta and Charuka (5th century AD) associated polyuria with sweet-tasting substance in the urine. The Indian descriptions of that time appeared to distinguish two forms of the diabetes one affecting older, obese people and the other thin and younger people who did not survive long¹.

Up to the 11th century, Diabetes was commonly diagnosed by 'water tasters,' who drank the urine of those suspected of having diabetes; the urine of people with diabetes was thought to be sweet-tasting. “Mellitus” the Latin word for honey (referring to its sweetness) was added to the term diabetes as a result^{1,2}. It was not until the 17th century AD that Thomas Willis (1621-1675) an English physician confirmed that diabetic urine contained sugar. Matthew

Dobson, later in the 18th century AD determined that diabetic serum was sweet to taste and contained sugar.¹

In 1870 French physician, Bouchardat, noticed the disappearance of glycosuria in his patients with diabetes during the rationing of food in Paris while under siege by Germany during the Franco-Prussian War and formulated the idea of individualised diets for his patients with diabetes.²

First chemical tests developed to indicate and measure the presence of sugar in the urine was early in 19th century. Still in 19th century, French researcher, Claude Bernard, studied the workings of the pancreas and the glycogen metabolism of the liver.^{1,2}

In 1869, Paul Langerhans, a German medical student, announced in a dissertation that the pancreas contains two systems of cells. One set secretes the normal pancreatic juice; the function of the other was unknown.^{1,2} Several years later, these cells were identified as the islets of Langerhans.² Jean de Meyer named the hypothetical pancreatic glucose-lowering hormone ‘insulin’. Insulin was finally extracted by Banting and Best in 1921.^{1,2}

In 1955 Oral hypoglycaemic drugs were introduced to help lower blood glucose levels.²

1.2 DEFINITION OF DIABETES MELLITUS

The term Diabetes Mellitus (DM) describes a metabolic disorder of multiple aetiology characterised by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both.³⁻⁵

The effects of diabetes mellitus include long-term damage, dysfunction and failure of various organs. Diabetes mellitus may present with characteristic symptoms such as excessive thirst, polyuria, blurring of vision, and weight loss. In its most severe forms, ketoacidosis or a nonketotic hyperosmolar state may develop and lead to stupor, coma and, in the absence of effective treatment, death.³⁻⁵

The long-term effects of diabetes mellitus include progressive development of the specific complications such as retinopathy with potential blindness, nephropathy that may lead to renal failure, and neuropathy with risk of foot ulcers, amputation, Charcot joints, and features of autonomic dysfunction, including sexual dysfunction. People with diabetes are at increased risk of cardiovascular, peripheral vascular and cerebrovascular disease.³⁻⁵

1.3 Diagnosis of Diabetes Mellitus

The diagnosis of diabetes mellitus according to World Health Organization (WHO)⁶ is based on the criteria in Table 1.3.1 below.

Table 1.3.1 Diagnosis of Diabetes mellitus

Diabetes mellitus

- Fasting plasma glucose(FPG) >126 mg/dl (7.0 mmol/l)
- Two-hour plasma glucose(2HPG) >200mg/dl (11.1 mmol/l) during standard 75g oral glucose tolerance test(OGTT).
- Random plasma glucose(RPG) >200 mg/dl (11.1 mmol/l) plus symptoms of diabetes

Diagnosis of DM is made if any of the above is present with classic symptoms of DM.

In the absence of classic symptoms of DM, the result of FPG, 2hr PG or RPG must be replicated on another day for the diagnosis of DM. **Impaired glucose tolerance (IGT)**

2 hour post parandial plasma glucose(2HPPG) >140 mg/dl
(7.8mmol/l) but less than 200 mg/dl (11.1 mmol/l).

Impaired fasting glucose (IFG)

Fasting plasma glucose >110 mg/dl (6.1 mmol/l) but less than 126 mg/dl (7.0 mmol/l).

The fasting plasma glucose is preferred because of convenience, acceptability to patients and lower cost. A new diagnostic category, impaired fasting glucose (IFG) was added to impaired glucose tolerance. Both of them refer to a stage intermediate between normal glucose homeostasis and diabetes.⁶

For epidemiological surveys, single casual plasma glucose or 2 Hour post glucose load may be used for screening or diagnosis of DM.⁴ The 2hr post glucose value determined by a specific enzymic assay after an oral glucose load of 75g in 300mls of water for adults is more reliable, as fasting state cannot be assured under clinical setting.

[1.4 Classification of Diabetes Mellitus](#)

The first widely accepted classification of diabetes mellitus was published by WHO in 1980 and in a modified form in 1985. The 1980 and 1985 classifications of DM and allied categories of glucose intolerance included clinical classes and two statistical risk classes ⁴. The 1980 Expert Committee proposed two major classes of diabetes mellitus and named them, Insulin

Dependant Diabetes Mellitus (IDDM) or Type 1 DM, and Non Insulin Dependant Diabetes Mellitus (NIDDM) or Type 2 DM⁴.

In the 1985 Study Group Report the terms Type 1 and Type 2 were omitted, but the classes IDDM and NIDDM were retained, and a class of Malnutrition-related Diabetes Mellitus (MRDM) was introduced. In both the 1980 and 1985 reports other classes of diabetes, included Impaired Glucose Tolerance (IGT) as well as Gestational Diabetes Mellitus (GDM). These were reflected in the subsequent International Nomenclature of Diseases (IND) in 1991, and the tenth revision of the International Classification of Diseases (ICD-10) in 1992⁴.

The 1985 classification was widely accepted and used internationally. It represented a compromise between clinical and aetiological classification and allowed classification of individual subjects and patients in a clinically useful manner even when the specific cause or aetiology was unknown⁴. The current recommended classification of DM includes both staging of diabetes mellitus based on clinical descriptive criteria and a complementary aetiological classification. Based on this, the WHO identifies four types of diabetes mellitus: Type 1, Type 2, "other specific types" and gestational diabetes. Each of the type of diabetes mellitus identified extends across a clinical continuum of hyperglycemia and insulin requirements.⁴

A. Type 1 Diabetes mellitus

- Autoimmune mediated Type 1 DM
- Idiopathic Type 1 DM

B. Type 2 DM which is further sub divided into

- Predominantly insulin resistance Type2 DM
- Predominantly insulin secretory defects Type 2 DM C. Other specific types:
- Genetic defects of beta cell function e.g. Maturity onset Diabetic of the young (MODY)
- Genetic defects of insulin action
- Diseases of the exocrine pancreas eg Fibrocalcific pancreatopathy
- Endocrinopathies e.g. Cushing syndrome, Acromegaly.
- Drug induced DM e.g. steroids, Thiazide diuretics
- Infections e.g congenital rubella, CMV
- Uncommon forms of immune-mediated diabetes eg stiff-man
- Other genetic syndromes sometimes associated with diabetes eg Down's syndrome, Turner's syndrome.

D. Gestational diabetes DM

1.5 Burden of Diabetes Mellitus

Diabetes Mellitus is a disease with significant impact on the health of the affected individuals, their quality of life and life expectancy, as well as on the health-care system⁷.

Patients with diabetes have a higher hospitalization rate, longer hospital stay, and increased ambulatory care visits⁸.

The increased morbidity and mortality among diabetics are associated with heavy economic losses⁹. Diabetes is a significant cause of adult blindness in the non-elderly, and a leading

cause of non-traumatic amputation in adults, as well as the third cause of nephropathy requiring dialysis in our environment¹⁰.

Patients with DM are prone to severe forms of infections like gram negative septicaemia, pyelonephritis, perinephric abscess, emphysematous cystitis, and renal papillary necrosis .^{11,12}

Diabetes mellitus patients are also prone to certain infections such as pseudomonas

“malignant” Otitis externa, monilial skin infections, and rhinocerebral mucormycosis.¹³

Cognitive impairment has also been identified in patients with diabetes.^{12, 13}

Diabetes was estimated to cause 3.8 million deaths worldwide in 2007, about 6% of total global mortality, about the same as HIV/AIDS. Using W H O figures on years of life lost per person dying of diabetes, this translates into more than 25 million years of life lost each year⁵. The International Diabetes Federation (IDF) estimates that the equivalent of an additional 23 million years of life are lost to the disability and to reduced quality of life caused by the preventable complications of diabetes⁵.

People living with diabetes and their families feel the impact of diabetes most directly as it decreases the income, increase expenditure on Medicare as well as reduces the quality of life of the patient⁵.

1.6 Justification for the study

There is the need to update data regarding the prevalence of DM in Nigeria, the latest data for the country was from the Expert committee report on Non-communicable Disease (NCD) survey of 1997. Although some studies were carried out in different parts of the

country, most are not community-based and therefore not truly reflective of the reality of the situation in the community. Thus, this study is intended to contribute to the data base.

Determination of the prevalence of the disease and its risk factors in our environment will enable us to detect sub-clinical cases. It is known that some of the risk factors for the disease are modifiable and prevention of the disease is possible for type 2 diabetes which is associated with urbanisation and sedentary life style^{7,8}.

The trend in the epidemiology of DM shows global increase in its prevalence. Moreover, developing nations will be worst hit by the numerical increase in the disease prevalence due to adoption of western life style¹⁴. The community screened was typically urbanizing hence the need for the study.

Lastly it is hoped that this study may provide the basis for a more rational public awareness campaign strategy and may be of use by the government in formulating policies for disease prevention, care and resource allocation.

1.7.0 Aim and Objectives of the study

1.7.1 Main Aim

To determine the prevalence and correlates of Diabetes Mellitus and Glucose intolerance among adults living in Kano metropolis.

1.7.2 Specific objectives

1. To determine the prevalence of Diabetes Mellitus and Glucose Intolerance among adults in Kano metropolis.

2. To assess the risk factors associated with Diabetes Mellitus and Glucose intolerance among adults living in Kano metropolis.

CHAPTER TWO

LITERATURE REVIEW

2.0 Introduction

Diabetes mellitus is a chronic disease that requires long-term medical attention both to limit the development of its devastating complications and to manage them when they do occur.

Diabetes mellitus previously considered a disease of minor significance to world health, is now taking its place as one of the main threats to human health in the 21st century.⁵

It is the fourth or fifth leading cause of death in most developed countries and there is

substantial evidence that it is epidemic in many developing and newly industrialized nations⁵⁻⁸. However, Diabetes Mellitus requires life-long therapy, usually with drugs. This creates a lifelong financial burden on the family, especially in low socio-economic communities in West Africa, where the majority of the population still live on less than one dollar a day.¹⁵ This affects the wellbeing of the entire family, hence the need for early detection, prompt and adequate management of the disease and avoidance of its complications.¹⁵

2.1 Prevalence of Diabetes Mellitus

The number of people with diabetes is increasing due to population growth, aging, urbanization, and increasing prevalence of obesity and physical inactivity¹⁶. Quantifying the prevalence of diabetes and the number of people affected by diabetes now and in the future is important to allow rational planning and allocation of resources¹⁶. The current estimates are based on demographic changes alone with the conservative assumption that other risk factor levels such as obesity and physical activity remain constant (in developed countries)¹⁶.

2.1.1 World prevalence of DM

The number of cases of diabetes worldwide among adults 20 years and above in 1995 was estimated to be 135 million (4% of world population) and 171 million by the year 2000 and 5.4% of world population will be affected by the year 2025⁹.

More recently in 2010 the International Diabetes Federation (IDF) reported that some 285 million (6.6%) people worldwide had diabetes and this figure will increase to 438 million

(7.8%) of world population by 2030⁵. The major part of this numerical increase will occur in the developing countries.

There will be a 42% increase from 51 to 72 million in the developed countries and a 170% increase from 84 to 228 million, in the developing countries⁹. Thus, by the year 2025, 75% of people with diabetes will reside in developing countries as compared with 62% in 1995. The countries with the largest number of people with diabetes are (and will be in the year 2025) India, China, and the United State of America (USA)⁵.

The crude prevalence of total diabetes in the United States (US) in 1999–2002 was 9.3% (19.3 million, 2002 U.S. population), consisting of 6.5% diagnosed and 2.8% undiagnosed¹⁷.

The situation in Europe is also not much different as over 60 million people live with diabetes in the new Europe; many of these people are unaware of their condition¹⁸.

The prevalence of type 2 diabetes in Latin America ranges from 1.2% to 8%, with higher prevalence rates in urban areas¹⁹. The frequency of diabetes in Latin America is expected to increase by 38% over the next 10 years, compared with an estimated 14% increase in the total population. The total number of cases of diabetes is expected to more than double and to exceed the number of cases in the US, Canada, and Europe by 2025¹⁹.

The growing epidemic of diabetes mellitus, type 2 in particular, has not spared the Asia-Pacific region. According to current estimates, this region has the largest diabetic population in the world namely 47.3 million, which is 46% of the global burden of diabetes. Population-based surveys from Asia show a wide range of prevalence rates of diabetes mellitus from 1.3% in Vietnam to 24.2% in certain Indian urban communities.²⁰

The prevalence has increased approximately three-fold from 0.8% to 2.3% between 1986 and 1994 in China. More recent data from certain Chinese cities suggest this increase may be even more pronounced, with a prevalence of as high as 9.2% in Hong Kong.²⁰

Seventeen studies performed in ten African countries since 1984, using the WHO criteria, have shown rates ranging from 0% in Togo to 4.8–8% in South Africa and to 10% in Northern Sudan²¹. Although there is still a paucity of data using standardised criteria, those that are available suggest that prevalence rates are low in sub-Saharan Africa north of the Limpopo River (separating South Africa from Botswana and Zimbabwe) in both urban and rural settings. The prevalence in South and North Africa is comparable to rates in developed countries but high in populations with Egyptian ancestry in Northeast Africa²¹.

The majority of the patients (70-90%) present with typical Type 2 diabetes, and up to 25% of patients with diabetes mellitus are diagnosed with Type 1 diabetes²². Variable proportions present with tropical diabetes (~ 1%), or with ketosis-prone atypical diabetes (~ 10-16%)²⁰. The incidence of type 1 diabetes mellitus varies from 4 to 10/100,000 among the 0-19yr old population in Africa²².

In 1985 prevalence of DM in rural region of Mali was put at 0.92%²³. In 1997, a prevalence of 2.4% in the urban population and 0% in the rural Villages in southern Sierra Leone were reported²⁴.

Ghana reported a crude prevalence of 6.4% in 2000. The study however showed that the prevalence of Diabetes and IGT increased with age. The oldest age group (64 years and above) had the highest diabetes prevalence (13.6%)²⁵.

The International Diabetes Federation estimated that 10.8 million people had diabetes in subSaharan Africa in 2006 and that this would rise to 18.7 million by 2025, an increase of 80%, as

such exceeding the predicted worldwide increase of 55%^{5,26}.

2.1.2 Prevalence of Diabetes Mellitus in Nigeria

The prevalence of DM in Nigeria based on the 1997 National Expert Committee Report on Noncommunicable Disease (NCD) was 2.2% with a male: female ratio of 1.1 :1²⁷.

Figures from different parts of Nigeria includes Jos, North Central Nigeria by Puepet was 3.2% as at 1996²⁸ ; and Ohwovori et al in Lagos a Western Nigerian State put the prevalence of undiscovered diabetes at 1.7% among Nigerians in Lagos metropolis²⁹.

Oyegbade et al¹⁵ put the prevalence at Ife to be 4.62%. Nyenwe et al³⁰ found the prevalence of Type 2 DM among adults in Port Harcourt to be 6.8% crude and 7.9% standardized; they stated that for every three known Type2 diabetic individuals in Port Harcourt, there were two who are unknown. However, the NCD survey of 1997 gave a crude prevalence rate of 7.2% for Lagos main land. Other parts of the country recorded low prevalence e.g. Ibadan an urban center had a

prevalence of 1.5%²⁷

2.1.3 Prevalence of Diabetes Mellitus in Kano

The NCD survey of 1997 found the age adjusted prevalence in urban Kano to be 1.8% and a crude prevalence of 2.5%²⁷.

2.2.0 Prevalence of glucose intolerance

Impaired glucose regulation (IGT and IFG) refers to metabolic state intermediate between normal glucose homeostasis and diabetes. It should be stated unequivocally however, that IFG and IGT are not interchangeable and represent different abnormalities of glucose regulation, one in the fasting state and the other post-prandial³.

IGT, rather than being a class as in the previous classification, is categorized as a stage in the natural history of disordered carbohydrate metabolism. A stage of IFG is also recognised because such subjects, like those with IGT, have increased risks of progressing to diabetes and macrovascular disease, although prospective data are sparse and early data suggest a lower risk of progression than IGT, although a similar CVD risk factor profile has been shown in IFG and IGT subjects.

IGT and IFG are not clinical entities in their own right, but rather risk categories for future diabetes and/or cardiovascular disease. They can occur as an intermediate stage in any of the disease processes. IGT is often associated with the Metabolic Syndrome (Insulin Resistance Syndrome). Thus, IGT may serve as an indicator or marker of enhanced risk by virtue of its correlation with the other elements of the Metabolic Syndrome. Individuals with IGT manifest glucose intolerance only when challenged with an oral glucose load³.

2.2.1 Prevalence of glucose intolerance world wide

The National Health and Nutrition Examination Survey (NHANES) showed that from 1988 to 1994, undiagnosed diabetes comprised approximately one-third of total diabetes

(diagnosed and undiagnosed) in U.S. adults. The prevalence of IFG was nearly as high as the prevalence of total diabetes¹⁷.

In Italy the prevalence of glucose intolerance in those above 45 years of age was 8.9 %³¹.

The prevalence in Australia was 16.4% for both IGT and IFG³.

In Philippines, an increase was also noted in the magnitude of IGT which almost doubled from 4.1% in 1982 to 8.1% in the recent survey (1996-2000)³². Only one in three diabetics reported that they had diabetes. The frequency of diabetes and IGT in urban and rural areas were about the same, although a substantial increase from the earlier survey was noted in rural areas. Women registered a higher prevalence for both conditions than men³².

The 1994 China National Diabetes Survey examination of 224 251 men and women aged 25 to 64 years yielded prevalence estimates for impaired glucose tolerance of 3.2%³³.

2.3.0 Risk factors for Diabetes Mellitus

The identified risk factors for DM can be classified as modifiable and non-modifiable risk factors.

2.3.1 Non modifiable risk factors

- Family history of Diabetes mellitus
- Genetic predisposition
- Advanced Age

2.3.2 Modifiable risk factors

- Urban life style
- Central Obesity
- Heavy alcohol consumption
- Physical inactivity

- Cigarette smoking
- Social class
- Impaired Glucose Tolerance

2.3.3 Age, Race and Family history of Diabetes mellitus

Age, ethnicity and family history of Diabetes are the main non-modifiable determinants of

Type 2 diabetes Mellitus as confirmed by the increasing prevalence with age, and the difference between Indians, Blacks and Caucasians in the United States of America¹⁷.

Indians have the highest predisposition followed by Blacks and Caucasians¹⁷.

In Nigeria a significant increase in prevalence of Diabetes was seen with increase in age in both sexes.²⁷ However, the NCD survey also found the odds of diabetes occurring is greater than that in general population of similar age if one or more parents have diabetes mellitus²⁷.

2.3.4 Obesity

Independent of genetic factors, obesity is associated with poor fitness, low insulin sensitivity, and decreased transcript levels of genes involved in mitochondrial oxidative phosphorylation³⁴. The level of obesity among individuals diagnosed with type 2 diabetes is significantly higher than the rate found among the general population³⁴.

Obesity is an important contributing factor to increased insulin concentrations and decreased insulin sensitivity, almost certainly attributable to increased portal blood free fatty acids from visceral fat. There is strong evidence from prospective studies that the risk of type 2 diabetes increases progressively from a BMI of over 20 kg/m² after adjustment for age, family history of diabetes, physical activity, and smoking¹⁴.

The NCD survey of Nigeria also found that the prevalence of diabetes increases when BMI exceed 30kg/m²²⁷.

2.3.5 Urbanisation

Among the modifiable risk factors, residence seems a major determinant, since urban residents have a 1.5 to 4-fold higher prevalence of diabetes compared to their rural counterparts. This is attributable to lifestyle changes associated with urbanisation and westernization²². Urban lifestyle is characterised by changes in dietary habits involving an increase in consumption of refined sugars and saturated fat, and a reduction in fibre intake. Moreover, there is a reduction in physical activity associated with urban lifestyle. Rural populations trek as transportation means and often have intense agricultural activities as their main occupation²⁰. Rural dwellers therefore have a high physical activity related energy expenditure compared to urban subjects thus explaining the higher rates of obesity in the cities. Obesity is also at least four times higher in urban areas compared to rural²².

2.3.6 Heavy Alcohol consumption

Heavy consumption of alcohol has been positively associated with diabetes. This may be due to hepatic and/or pancreatic damage, which is known to complicate alcoholism^{17,34-36}.

2.3.7 Cigarette smoking

Three large prospective studies suggest that smoking is associated with the development of type 2 diabetes in men and women, consistent with evidence linking smoking and insulin

resistance³⁶⁻³⁸. Smoking cessation is often accompanied by substantial weight gain, and obesity is an important risk factor for development of diabetes³⁸. It is not clear whether the benefits of giving up smoking outweigh the adverse effect of weight gain. The effect of pipe or cigar smoking (primary or secondary) on the development of diabetes is also not known³⁶. A report from the British Regional Heart Study based on 11.8 years of follow-up observed a significant positive relationship between current cigarette smoking and diabetes after adjustment for age and BMI³⁸.

2.3.8 Social class

Studies show that people in the highest social class have a significantly higher prevalence of type 2 diabetes than those in the lower classes³⁵. This finding agrees with the observation by Indian Doctors in 400 BC that diabetes was a disease of the rich³⁵. Fisch²⁷ also noted that diabetes was more common in the upper-class families in the developing Nations of the world. However, the non-communicable disease survey in Nigeria contradicted this observation as a crude prevalence of 4.6% was reported in the lower social class compared with 2.5% in the highest social class²⁷.

2.3.9 Physical inactivity

It has long been suggested that a physically active lifestyle may decrease the risk of developing type 2 diabetes. Recent clinical trials of adult Swedish, Chinese and Finnish men and women with impaired glucose intolerance (IGT) at baseline in which physical activity was part of the intervention strategy demonstrated decreases in diabetes development at follow-up^{39,40}. Evidence of a significant relation between physical activity and glucose tolerance had been supported earlier on by the observation that societies that had abandoned traditional lifestyles (which typically had included large amounts of habitual physical activity) experienced major increases in type 2 diabetes⁴¹. Physical activity alone or in conjunction with dietary changes can reduce the incidence of type 2 diabetes^{35,42-44}. However, the intensity of activity required remains unclear because the independent role of moderately intense activities had not been directly examined in these trials⁴⁵.

2.4.0 AETIOPATHOGENESIS OF DIABETS MELLITUS

2.4.1 Type 1 Diabetes Mellitus

2.4.1.1 Auto immune Type 1 Diabetes Mellitus

Type 1 diabetes results from a cell-mediated autoimmune destruction of the cells of the pancreas by CD4+ and CD8+ T cells and macrophages infiltrating the islets. Markers of the

immune destruction of the cell include islet cell auto antibodies (ICAs), auto antibodies to insulin (IAAs), auto antibodies to glutamic acid decarboxylase (anti-GAD), and auto-antibodies to the tyrosine

phosphatases^{9,46-51}.

One or more of these auto antibodies are present in 85%–90% of individuals when fasting hyperglycemia is initially detected. The more the number of auto anti bodies the higher the chance of developing Type 1 DM and the faster the progression of the disease^{7,45-50}. The disease also has strong HLA associations, with linkage to the DQA and DQB genes, and it is influenced by the DRB genes, and ICA negativity^{9,46-51}.

The rate of cell destruction is quite variable, being rapid in some individuals and slow in others. Some patients, particularly children and adolescents, may present with ketoacidosis as the first manifestation of the disease while others have modest fasting hyperglycemia that can rapidly change to severe hyperglycemia and/or ketoacidosis in the presence of infection or other stress. Still others may retain residual B cell function sufficient to prevent ketoacidosis for many

years^{9,46-51}.

2.4.1.2 Idiopathic Type 1 Diabetes mellitus

Some forms of type 1 diabetes have no known etiologies. Some of these patients have permanent insulinopenia and are prone to ketoacidosis, but have no evidence of autoimmunity. Only a minority of patients with type 1 diabetes fall into this category, of those who do, most are of African or Asian origin^{52,53}. Individuals with this form of

diabetes suffer from episodic ketoacidosis and exhibit varying degrees of insulin deficiency between episodes. This form of diabetes is strongly inherited, lacks immunological evidence for B cell autoimmunity, and is not HLA associated^{52,53}. An absolute requirement for insulin replacement therapy in affected patients may come and go^{52, 53}.

2.4.2 Type 2 Diabetes Mellitus

Type 2 diabetes is a chronic disorder of carbohydrate and lipid metabolism and is known for its heterogeneity. It is caused by impaired insulin action in muscle and adipose tissue, defective insulin secretion and inadequate suppression of hepatic glucose output (HGO)⁵⁴. The major genetic factor is impaired B-cell function and insulin resistance is the major acquired factor.

2.4.2.1 Insulin resistance

Insulin resistance is a hallmark of type 2 diabetes world wide, and is genetic and familial, with an acquired component. Insulin resistance is found predominantly in the skeletal muscle, adipose tissue, and hepatic tissues in several populations, and it precedes the development of IGT and type 2 diabetes by a decade^{55,56}. It has been shown that race and ethnicity,

independent of family history of type 2 diabetes, also determine insulin resistance^{8,9}.

Several population studies have demonstrated that insulin resistance is associated with obesity in a given population^{8,9,22,23,53,55,56}. The consequences of insulin resistance are progressive deterioration in the rate of glucose disappearance or disposal in the peripheral tissues, and perhaps, beta cell exhaustion. The latter, whether genetically inherited (e.g.

apoptosis) or not, could precipitate the development of IGT and type 2 diabetes in individuals susceptible to the disease²¹.

In the Western industrialised countries, the major determinants of insulin resistance in addition to genetics, are obesity or overweight and sedentary lifestyles. It has therefore, been suggested that in populations with lower prevalence rates of obesity, such as native black Africans, the degree of insulin resistance is predominantly determined by genetic inheritance, and further affected by conventional risk factors such as physical inactivity²¹.

2.4.2.2 Insulin secretory defect

One of the most striking observations from the United Kingdom Prospective Diabetes Study was the significant decrease in beta-cell function of up to 50% of normal, in subjects with a recent diagnosis of type 2 diabetes⁵⁷. Extrapolating the data from the same study suggests that this decline starts approximately 10–12 years before the diagnosis of diabetes. It is now universally accepted that beta-cell dysfunction is a hallmark of diabetes and is also related to hyperglycaemia^{4, 57-60}. This dysfunction is manifest through different ways which includes reduced insulin release in response to glucose and other non-glucose secretagogues, changes in pulsatile and oscillatory insulin secretion and an abnormality in the effective conversion of proinsulin to insulin. There is enough evidence to infer that these changes start well before the diagnosis of the disease and most abnormalities have been noted in pre-diabetic individuals. These defects have been identified in high-risk individuals for type 2 diabetes such as subjects with first degree relatives with diabetes and women with a history of gestational diabetes or polycystic ovarian syndrome. Insulin release from the pancreas is biphasic, with the initial brief spike which lasts for 10 minutes called the first

phase insulin release. This is followed by a prolonged second phase which plateau by 2–3 hours.

The vast majority of patients with type 2 diabetes exhibit some degree of insulin resistance, but type 2 diabetes can occur in the absence of insulin resistance. Furthermore, most obese individuals who are insulin resistant do not develop type 2 diabetes. These individuals remain normoglycemic because they compensate for the reduction in insulin sensitivity by increasing their insulin secretion⁶¹.

2.5.0 Methods of screening for diabetes mellitus

The following five criteria define the optimal conditions for screening for a particular disorder:

- The disorder should be an important public health problem
- An early asymptomatic stage should exist
- There should be a suitable screening test
- An accepted treatment should be available
- There is evidence that early treatment improves long-term outcome

Diabetes mellitus fulfill the 1st four criteria but it has not been firmly established that early detection of type 2 diabetes and intervention improve long-term outcome⁶².

Screening for DM in asymptomatic subjects suffers from two important limitations: the lack of a screening test that combines accuracy with practicality, and the absence of adequate evidence that early detection and treatment improve outcome in asymptomatic persons⁶³.

2.5.1 Screening tests — The most commonly used screening tests for type 2 diabetes include measurement of the plasma glucose (FPG), two-hour plasma glucose during an oral glucose tolerance test (2-h OGTT), glycosylated hemoglobin, and the urine dipstick testing for glucose^{62,63}.

1. Urine glucose — Detection of glucose on a semiquantitative urine dipstick (anything regarded as trace positive or more) or Clinitest tablets is an insensitive means of screening for type 2 diabetes. The high rate of false-negative results suggests that the urine dipstick is not adequate as a screening test. Additionally, not all patients with glucosuria have diabetes. Glucosuria can occur with defects in renal tubular function, as seen in Type 2 (proximal) renal tubular acidosis and in familial renal glucosuria, a genetic disorder associated with salt wasting,

polyuria, and volume depletion⁶²⁻⁶⁴

2. Glycosylated hemoglobin — Recently, newer methods of screening have been suggested, using estimation of glycosylated haemoglobin (HbA1c) or fructosamine on a random blood specimen. Although HbA1c has a high predictive value, it can only be use in long-standing hyperglycaemic states, hence it is found to be within the reference range in many patients with newly developed diabetes or other minor abnormalities of glucose tolerance. The advantage of HbA1c estimation is that it reflects the average prevailing blood glucose level, and only a single blood specimen is required. However it is more expensive than glucose estimation, and may be affected by factors such as renal failure and

haemoglobinopathies. The latter is particularly relevant in community surveys due to the relatively high prevalence of certain haemoglobinopathies⁶⁴.

In June 2009, an International Expert Committee recommended using the HbA1C to diagnose diabetes. National and international diabetes organizations, including the American Diabetes Association (ADA), are considering whether and how to implement the recommendation ⁶².

3. Fructosamine estimation- Fructosamine estimation is much cheaper than HbA1 or plasma glucose estimation, and is a fully automated procedure. It is also not affected by uraemia unless associated with a serum albumin of less than 30 g/l. However, it appears to be even less reliable in the detection of subjects with IGT ⁶⁴. Most reports on fructosamine evaluate its use in the monitoring of diabetic control and there are few reports of its sensitivity and specificity in screening in the elderly ⁶⁴.

4. The oral glucose tolerance test: The oral glucose tolerance test (OGTT) has been accepted as the ultimate standard for the diagnosis of diabetes mellitus; nevertheless, it is cumbersome in community screening. It is often difficult to ascertain a proper fasting or an accurate 2-h postprandial blood glucose estimation so that in practice only urine analysis and/or a random blood glucose estimation may be possible⁶²⁻⁶⁴. However, for cultural reasons, some people are often reluctant to accept repeated venepunctures as required for an OGTT⁶⁴.

5. Fasting Plasma glucose- The FPG test is preferred in clinical settings because it is easier and faster to perform, more convenient and acceptable to patients, and less expensive.

However, it is difficult to use at a community level as fasting cannot be guaranteed and not all the participants will comply or agree to fast ⁶⁵.

6) Casual plasma glucose

Plasma glucose testing may be performed on individuals who have taken food or drink shortly before testing. Such tests are referred to as casual plasma glucose measurements and are given without regard to time of last meal. This method is neither sensitive nor specific but it is difficult in community screening to use the more sensitive and more specific tests⁶⁵.

CHAPTER THREE **SUBJECTS, MATERIALS AND METHODS**

3.1 Study Area

The study was carried out in Kano state. It is bordered on the east by Bauchi and Jigawa States, to the south by Kaduna State and to the west and north is by Katsina state. Kano State has a population of 9,383,682 people with an almost equal distribution of male (51%) and female (49%). Majority of the people of Kano State are Hausa/Fulani while other ethnic groups constitute the minority⁶⁶.

Urban Kano is made up of eight local Government Areas (Municipal, Dala, Gwale, Fagge, Nassarawa, Tarauni, Ungogo and Kumbotso LGCs) from which Tarauni L G A was randomly selected for this study by balloting. It is located outside the ancient walls of the Kano city, on latitude 12° N and longitude 8.3° E with a land area of 26km². It shares boundaries with Nassarawa local government area to the north, Kumbotso local government area to the Southeast, and Kano Municipal local government area to the west. It has a population of 221,367 from 2006 population census⁶⁶ and this is spread over ten political

wards: Unguwa Uku, Tarauni, Hausawa, Jao`ji, Yar akwa ,Gyadi-gyadi, Kauyen alu, Kundila, Hotoro and Darmanawa. The people of Tarauni are predominantly Hausa and Fulani ethnic groups but other ethnic groups such as Yoruba and Igbo are present. These people are mainly involved in commerce, trading, farming with others in the civil service. There are small and medium scale industries located within the LGA such as shoe making factories and leatherworks.

3.2 Study Design

The study was a community based cross-sectional descriptive study.

3.3 Study population

The subjects included all males and females aged 15years and above residing within the study area.

3.4 Sample size determination

The minimum sample size was determined by using the formula for estimation of minimum sample size for descriptive study i.e $n = z^2 p q / d^2$ ⁶⁷ Based on the prevalence of diabetes mellitus in a Nigerian urban city similar to Kano (Lagos main land = 7.4%) as reported by the expert committee on non communicable disease survey of 1997 ²⁷ where:

n=minimum sample size **z** = standard normal deviate at 95% confidence

level = 1.96 **p**= prevalence of diabetes mellitus obtained from previous

study (7.4% = 0.074) **q**= Complementary probability to p = 1-p = 1-0.074 =

0.0926 **d**= absolute precision limit required (2%) =0.02

Thus $n = (1.96)^2 (0.074) (0.926) / (0.02)^2 = 658$

700 persons were recruited to accommodate non responders.

3.4.1 Inclusion criteria

- (1) All persons aged 15 years and above within the study area
- (2) Persons that gave informed consent to participate in the study

3.4.2 Exclusion criteria

- (1) All persons below the age of 15 years
- (2) Persons who did not consent to take part in the study
- (3) Persons living outside the study area
- (4) Ill persons and pregnant women

3.5 Sampling method

A multi-stage random cluster sampling method was used to select the participants. In the first stage one local government area (Tarauni) was randomly selected from the eight metropolitan local government areas in Kano, using balloting method. Similarly, random sampling (balloting) was used in the second stage to select five wards out of the ten wards that made up Tarauni local government area. House and House-holds listing was thereafter conducted in the five selected wards. Using the systematic sampling technique a proportionate number of households were obtained in the wards after calculating a sampling interval for each ward (from sampling frame and sample interval).

Finally, eligible respondents from each of the selected houses were enlisted and requested to participate after obtaining an informed consent. The first house hold was selected using table of random numbers while subsequent house-holds were identified by adding the sampling interval (2 i.e every 3rd house) to preceding house hold number. This was continued until the required sample size was obtained.

3.6 Ethical Approval

An approval was obtained from the AKTH Ethical Committee (Appendix 9) and informed consent (Appendix 8) were obtained from all the participants before they were interviewed and examined. Those that were found to have diabetes or had abnormal blood glucose were counseled and referred to health facilities of their choice for further management after the study. The researcher paid for the cost of investigating the participants till completion of the study.

3.7.0 Instruments / Materials / Equipments and Method of Data Collection

3.7.1 The materials/equipments used in the study

- (1) Hanson^R weighing Scales (Bath room type)
- (2) A 9 parameter strips for urinalysis (Medi-Test combi 9)
- (3) Butterfly Tailors tapes non stretch
- (4) Clean universal urine sample bottles
- (5) Portable stadiometers (Surgifriend Medicals England)
- (6) Accuson^R mercury Sphygmomanometer
- (7) Stethoscope (Littman classic II S.E) SN: 16N52897.
- (8) Fluoride Oxalate blood sample bottles
- (9) Cool box and bottle stands
- (10) 5cc syringes and latex gloves
- (11) Glucose D, drinking mugs, and clean drinking water

3.7.2 Instruments of data collection

The instruments of data collection were a pre-tested, adopted interviewer administered questionnaire (Appendix 7). The questionnaire was pre-tested in a community similar to the

study community (Kundila ward) to assess the acceptability of the questions therein and participant's response based on their understanding. This enabled adjustment and corrections of the designed questionnaire as well as enhanced validity. The questionnaire was translated into Hausa language since majority of the participants were Hausas.

3.7.3 Preparation for data collection

A courtesy call was paid to the Local Government Chairman, District Head and various Ward Heads to appraise them about the study and its importance as well as solicited their support. Other influential persons in the community (self help groups, youth vanguards) were identified and their support was also solicited for. Discussion was held with various components of the community to clarify their doubts, answer their questions with regard to the project as much as possible.

Ten research assistants' five males and five females were recruited and trained for two days on how to obtain consent and sample collection as well as the study protocol. They were recruited from the primary health centers located in the chosen wards to enhance community acceptance. Their roles included obtaining subject's personal data and consent, measuring weight and height, collection of urine, and blood samples. They served as a bridge to reach the community as they were from the same community.

3.7.4 Method of data collection (Appendices 1-4)

Data collection was achieved with the aid of interviewer administered pre-tested questionnaires.

The subject's blood pressure (appendix 4) and anthropometric indices (appendix 8) were measured and blood samples taken to AKTH chemical pathology laboratory for analysis. For cultural reasons the researcher and five trained male research assistants interviewed and obtained measurements from male subjects and trained female research assistants performed the same for female subjects. For ease and accuracy of collection of data and measurements, collection centers were formed at the selected ward where the subjects were invited to. Those that chose not come to the centers were met at home for collection of their data, urine and blood samples as well as measurements of their BP and anthropometric indices. Samples from students of higher institutions were collected during the weekends.

The role of the researcher included:

- 1- Training of research assistants for data and blood sample collection as well as obtaining consent from the subjects.
- 2- Measurement of blood pressure
- 3- Measurements of Anthropometric indices
- 4- Collecting and handling of blood samples (venopuncture)
- 5- Administering oral glucose tolerance test (OGTT)
- 6- Transporting and analysis of samples at the chemical pathology laboratory
- 7- Direct analyses of the collected urine and blood sample
- 8- Overall coordination of the study
- 9- Analysis of the collected data
- 10- Final write-up of the study

3.7.5 Blood sample taking and laboratory procedures

Five milliliters (5mls) of venous blood was drawn from a vein in the forearm with the subjects seated and relaxed. The samples were put into fluoride oxalate (glycolytic inhibitor) containers and were kept on ice packed cool box and transported within 3 hours of collection to the chemical pathology laboratory of AKTH (about 2-3.5 km from survey sites) for estimation of casual plasma glucose. Subjects with Casual plasma glucose of ≥ 7.8 mmol/l were categorized

into 3, viz:

- 1- Those that were known to be living with diabetes before the survey.
- 2- Those with classical symptoms of diabetes mellitus and casual plasma glucose of ≥ 11.1 mmol/l and were not known to have diabetes.
- 3- Those with CPG ≥ 7.8 mmol/l but < 11.1 mmol/l without symptoms and were not known to have diabetes.

The 3rd category was invited to chemical pathology laboratory of AKTH on another day in fasting state to define their diabetes status. This same category of subjects were subjected to a standard OGTT to ascertain their status; those with 2Hr PGL of ≥ 11.1 mmol/l were classified as having diabetes while those with 2 Hr PGL between 7.8- 11.0 mmol/l were classified as having impaired glucose tolerance.

3.7.5 Classification of physical activity²⁸

Table 3.1 Adopted classification of physical activity base on subject's work and leisure activity

	Work related activity	Leisure activity

Very active	Eg labouring	Sport ≥ 3 days/week
Moderately active	Eg house work, trade work and nursing work	Gardening, walking and sport 1-2 days/ week
Not active	E.g Sedentary, office work, unemployed	House bound

Definition of terms

- 1- Diabetes mellitus- Fasting plasma glucose >126 mg/dl (7.0 mmol/l) or two hour post-glucose load >200 mg/dl (11.1 mmol/l) plus symptoms of diabetes².
- 2- Impaired glucose tolerance- Normal fasting plasma glucose with 2 hour post-glucose load >140 mg/dl (7.8mmol/l) but less than 200 mg/dl (11.1 mmol/l) ².
- 3- Impaired fasting glycaemia- Fasting plasma glucose >110 mg/dl (6.1 mmol/l) but less than 126 mg/dl (7.0 mmol/l)².
- 4- Body Mass Index category-
 - Underweight- Body Mass Index < 18.5 kg/m².
 - Normal- Body Mass Index 18.5 kg/m² – 24.9 kg/m².
 - Overweight- Body Mass Index >25 kg/m² but <29.9 kg/m².
 - Obesity- Body Mass Index >30 kg/m².
- 5- Central Obesity

- Waist circumference \geq 94cm (men)
- Waist circumference \geq 80cm (women)

6- Hypertension- Blood pressure measurement of and above 140/90mmHg.

7- Sedentary lifestyle – walk of less than thirty minutes per day.

8- Registrar General’s Social Classification⁶⁸

This classification is based on occupation of the populace.

TABLE 3.1 Classification of social class

SOCIAL CLASS	DESCRIPTION	EXAMPLES
I	Professionals Businessmen/Women	Lawyers, Doctors Large employers
II	Lesser professions Trade	Teachers Shopkeepers
III	N	Skilled non-manual Clerical workers
III	M Lorry drivers	Skilled manual Electricians
IV	Semi-skilled manual	Farm workers Machine operators Masons
V	Unskilled manual to Unemployed	Farm workers, Labourers

Adopted from Morris 1975.

3.8 Limitations of the study

1-This study did not subject all the participants to standard OGTT for logistic reasons which may give a clearer picture of the prevalence of both DM and IGT.

3.9 Data Analysis and Statistics

Soft wares used: EPI info 3.2. 2002 and MiniTab 12.

- Means \pm (SD) were used to described continuous variables
- Proportions was used to describe categorical Data
- Student's t-test were used for comparisons of means
- χ^2 (chi square) test for comparism of proportions and determination of linear trend.
- Standardization of prevalence rates using census figure for Tarauni (95% CI 5.0% - 8.8%)
- Multiple logistic regression analysis to determine independent risk factors : Only significant risk factors are applied.

- Linear regression for determination of relationship of significant continuous variables with plasma glucose levels.

CHAPTER FOUR

RESULTS

4.1 QUALITY OF THE DATA

Seven Hundred (700) subjects were enlisted during the house-hold census, sixteen (15 males and 1 female) withdrew their consent during the survey. Data obtained from 684 subjects were analysed giving a response rate of 97.7%.

4.2 SOCIO-DEMOGRAPHIC CHARACTERISTICS OF THE STUDY SUBJECTS

4.2.1 Age and sex distribution of the subjects

There were 434 (63.5%) females and 250 (36.5%) males in this study population.

The age of the participants ranged from 15 to 80 years with a mean \pm (SD) of $35.3 \pm (13.1)$ yrs. The mean age for males was $36.6 \text{ yrs} \pm (14.9)$ and $34.5 \text{ yrs} \pm (11.9)$ for female ($t=2.02$, $p=0.043$) The age distribution of the study subjects is as shown in table 4.1.

Table 4.1: Age and sex distribution of the study subjects

<u>Age (years)</u>	<u>sex</u>		<u>Total</u>
	<u>Males</u>	<u>Females</u>	<u>N(%)</u>
	<u>N (%)</u>	<u>N (%)</u>	
15-24	47(6.9)	87(20)	134(19.6)

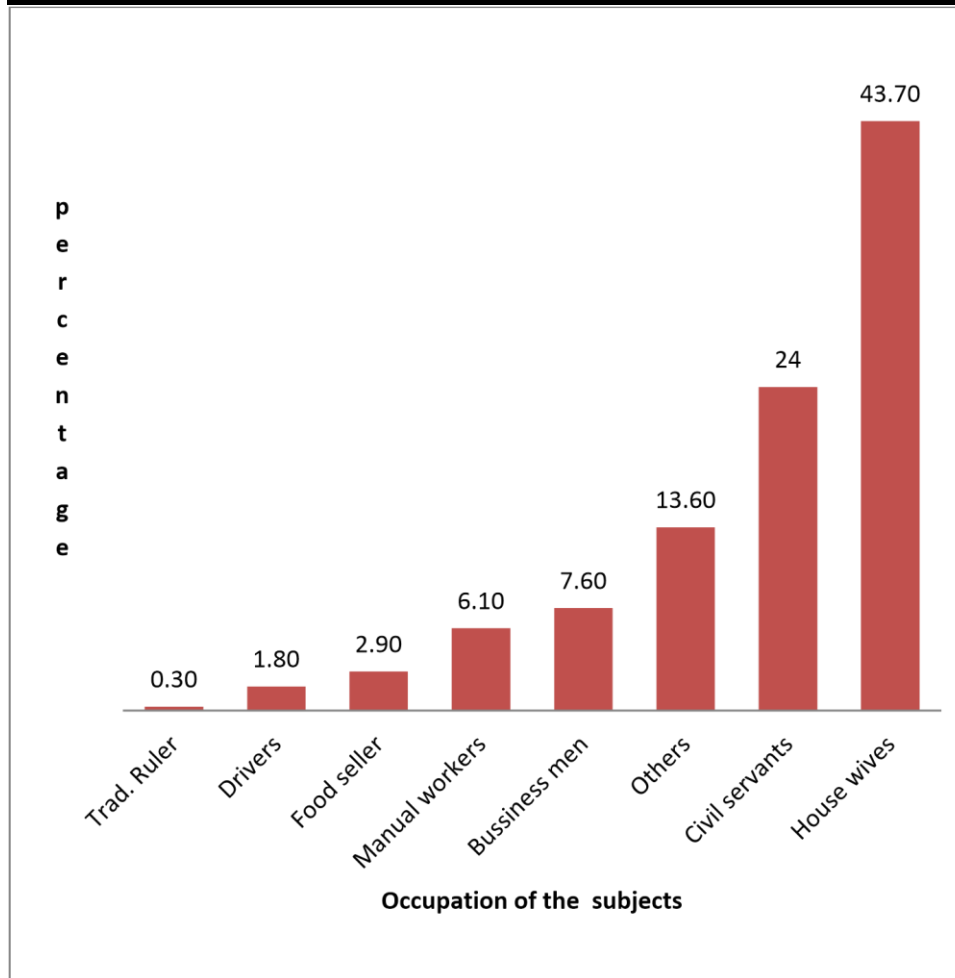
25-34	87(12.7)	131(30.2)	218(31.9)
35-44	48(7.0)	116(26.7)	164(24.0)
45-54	28(4.1)	68(15.7)	96(14.0)
55-64	24(3.5)	28(6.5)	52(7.6)
≥65	16(2.3)	4(0.9)	20(2.9)
<hr/>			
Total	250(36.5)	434(63.5)	684
<u>(100)</u>			

$\chi^2 = 38$, $p = 0.001$ females being more than males.

4.2.2 OCCUPATIONAL DISTRIBUTION OF THE STUDY SUBJECTS

The distribution of the subjects by their occupation is as shown in the figure I which shows that house wives account for 43.7% of total participants followed by the civil servants accounting for 24%, others (unemployed and those under-care) 13.6%.

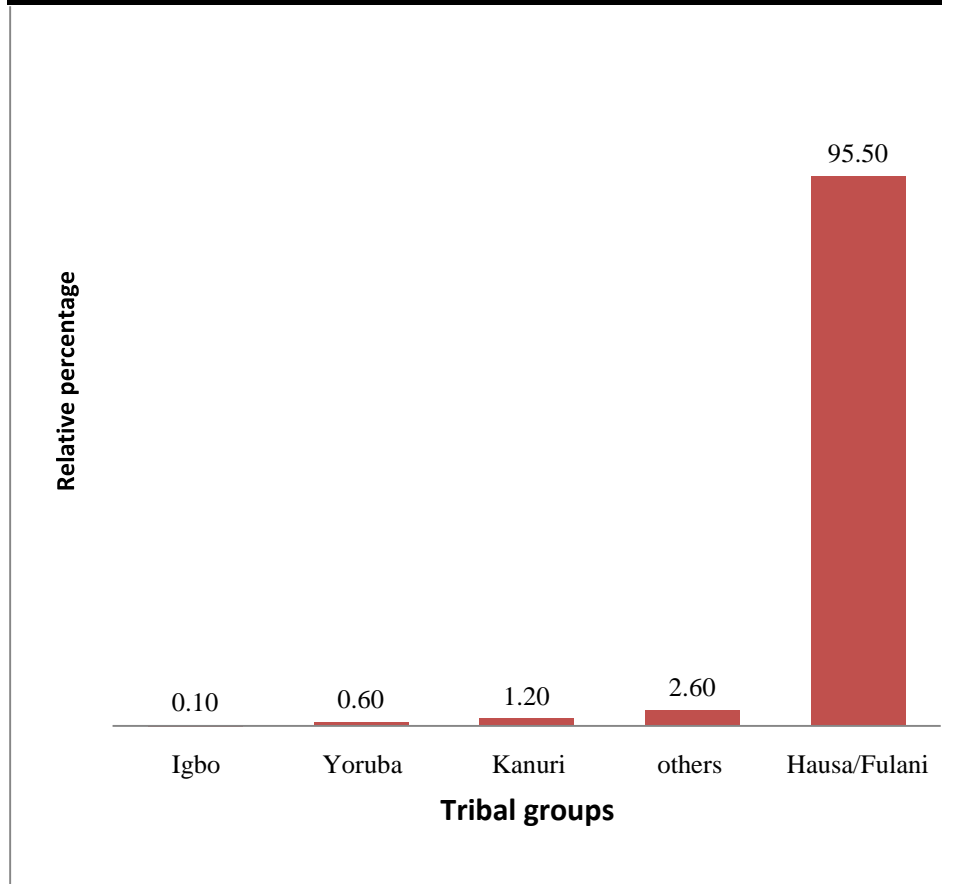
Figure I: Occupational distribution of the study subjects



4.2.3 DISTRIBUTION OF SUBJECTS BY ETHNIC GROUPS

The distribution of the study subjects by ethnic group is as shown in figure II. Hausa/Fulani were the majority (95.5%) and the least were the igbos (0.1%).

Figure II: Distribution of subjects by Ethnic group



4.2.4 Distribution of study subjects by social class

The distribution of the subjects by social class is shown in table 4.2 which shows that majority of the subjects (59.6%) belong to the social class v.

Table 4.2: Distribution of subjects by social class

Social class	frequency (%)		Total (%)
	Male	female	
Class I	7 (1.0)	3(0.5)	10(1.5)
Class II	3(0.4)	1(0.2)	4 (0.6)
Class III	61(8.9)	90(13.2)	151(22.1)
Class IV	39(5.7)	72(10.5)	111(16.2)
Class V	140(20.5)	268(39.2)	408(59.6)
Total	250 (36.5)	434(35.5)	684 (100)

$\chi^2 = 9.31$, $p = 0.053$. There is no statistically significant difference between the males and the females.

4.3. 0 CLINICAL CHARACTERISTICS OF THE STUDY SUBJECTS

4.3.1 The level of physical activity among the study subjects is shown in table, 316 (46.2%) of the subjects were active and 386 (53.8%) were inactive.

Table 4.3: Level of physical activity among the subjects

Level of physical activity	Males	Females	Total
	N (%)	N (%)	N (%)
Very active	87(12.8)	29(4.2)	116(17)
Moderately active inactive	49(7.2)	151(22)	200(29.2)
	114 (16.7)	254(37.1)	368(53.8)
Total	250(36.5)	434(63.5)	684(100)

$\chi^2 = 91.4$, $p = < 0.0001$ there is statistically significant difference with males being more active than females

4.3.2 Family history of DM among the study subjects

Out of the 684 subjects only 82 (12%) had positive family history of DM, while 602 (88%)

either did not have family history of DM or did not know.

4.3.3 Alcohol use among the study subjects

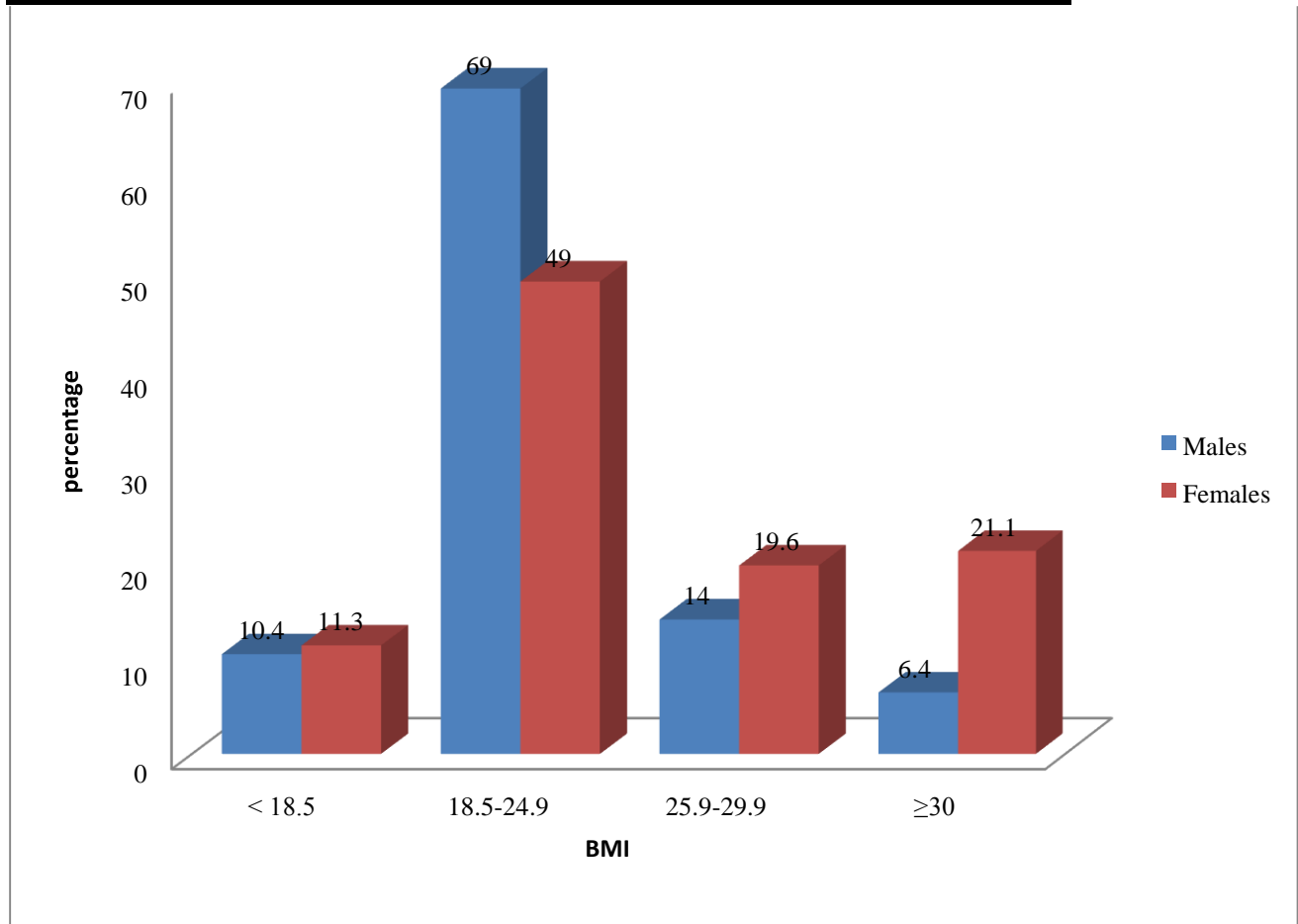
Only 2 (0.3%) of the subjects volunteered history of alcohol consumption.

4.4.0 ANTHROPOMETRIC INDICES OF THE STUDY SUBJECTS

4.4.1 Body Mass Index by sex of the study subjects

The BMI by gender of the study subjects is shown in figure III. The mean BMI of the study subjects was $23.8 \pm (5.8) \text{ kg/m}^2$. The mean BMI for male subjects was $22.1 \pm (5.6) \text{ kg/m}^2$ while that of females was $24.6 \pm (5.9) \text{ kg/m}^2$. The females have higher BMI. ($t= 5.4$; $p=0.001$) this is statistically significant.

Figure III: Distribution of BMI of the study subjects by sex



Majority of the subjects particularly males had BMI in the normal range. There were more overweight and obese females than males (19.6% vs 14% and 21.1% vs 6.4% respectively).

4.4.2 Waist circumference (WC) of the study subjects

The mean WC for the males was 80.9cm \pm 10.9, while for females it was 86.9 \pm 14.0.(t=5.8 ; p=0.001). Using IDF cut off values for different sexes i.e 80cm for females and 94 cm for males,

61.5% of the female subjects had central obesity and 14.8% of the males had central obesity.

4.4.3 Waist to hip ratio (WHR) of the study subjects

WHR were divided into four groups based on Quartiles of distribution in the study

population. In the upper quartiles for males and females were subject with WHR \geq 0.91 and 0.93 respectively; while in the lowest quartiles were subjects WHR < 0.84 and < 0.85

males: females respectively. The mean \pm (SD) WHR for males and females were 0.87 \pm 0.07 and 0.88 \pm 0.07. (t= 1.8, P= 0.06)

4.4.4 Distribution of casual plasma glucose in mmol/l among study subjects

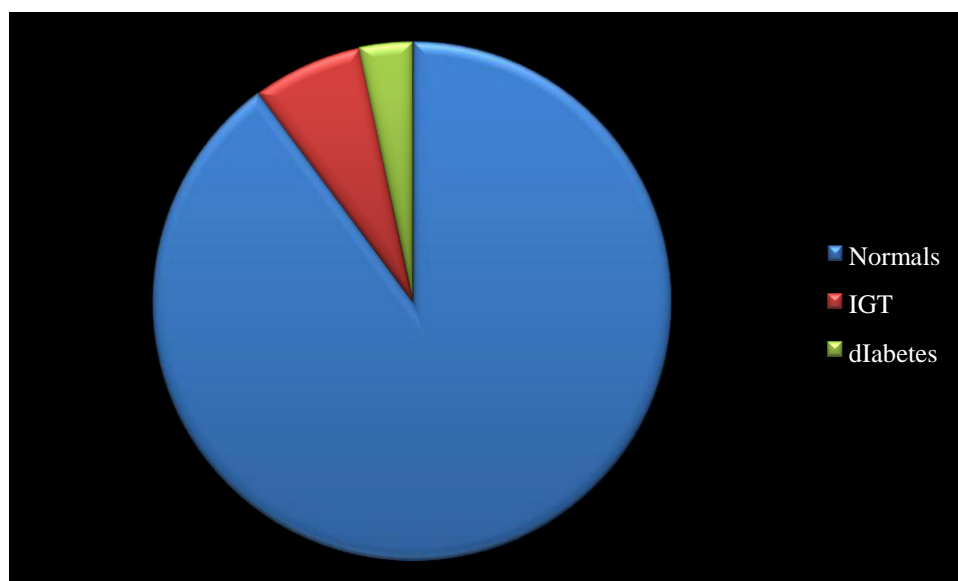
The mean casual plasma glucose of all the subjects was 6.8 \pm 2.6mmol/l, the mean for males was 6.7 \pm 2.6 mmol/l, while it was 6.8 \pm 2.5 mmol/l for the females (no significant difference between the CPG of the males and the females; p=0.6 , t=0.5)The distribution of CPG of all subjects is as shown in table 4.4

Table 4.4: Distribution of CPG among the study subjects by sex

CPG(Mmol/L)	Males N(%)	Females N(%)	Total (%)
-------------	---------------	-----------------	--------------

Normal (3.5-7.8)	225(32.8)	363(53.1)	588(85.9)
I G T (7.9-11.0)	17(2.5)	58(8.5)	75(11)
Diabetes (≥ 11.1)	8(1.2)	13(1.9)	21(3.1)
Total	250 (36.5)	434(63.5)	684(100)

Approximately 86% of the subject's CPG falls within the normal range, the remaining 14% had various degrees of glucose dysregulation.



Distribution of CPG among all the study subjects.

4.5.0 PREVALENCE OF DIABETES MELLITUS AND GLUCOSE INTOLERANCE IN THE STUDY

4.5.1 Prevalence of Diabetes mellitus

The study found twenty eight 28 subjects (4.1%) who were known to be living with diabetes before the study (14 males and 14 females) and 31 (4.5%) newly diagnosed to have diabetes

during the survey (5 males and 26 females) giving a total crude prevalence of 8.6%. The crude male prevalence was 7.6% and crude females prevalence was 9.2% and a male to female ratio of 1:1.2; The total crude prevalence dropped to 6.9% after adjusting for sex and age, so also the prevalence for the males which dropped to 6.4%, and the prevalence for the females dropped to 8.8%. The mean age of all the subjects with diabetes was 42.4 yrs \pm (11.8). The mean age for the male subjects with diabetes was 49.4 yrs \pm (12.2), while it was 41.7 yrs \pm (11.7) for the female counterpart ($t = 2.33$; $p\text{-value: } 0.02$).

The relative frequency of type T2DM compared to likely T1DM was 95.6% to 4.4% respectively all T1DM were known to have the disease before this study.

this was based on age of the subjects and lack of history of insulin use which is characteristic of type 1 DM or else they would have been found ill, as T1DM patients are absolutely insulin dependent.

4.5.1.1 Standardization of prevalence rates of DM in the study, adjusted to Tarauni

population (2006 census) using direct method. Tarauni population 2006 census

221,567

15 years and above

116,660

Table 4.5: Standardization of prevalence rates

Age group(yrs)	population	Diabetics	Crude Prev.	Expected
15-24	42502	2	1.5	673.5
25-34	31655	8	3.7	1171.2
35-44	19259	20	12.2 11.5	2349.6
45-54	11511	11	28.8	1323.8
55-64	5756	15	15	1657.7
≥ 65	5977	3		896.6
Total	116660	59	8.6%	8072

Age standardized prevalence = No. of expected cases/ Tarauni popn. ×100

$8072/116660 \times 100$

=6.9%

95% C I = $P \pm 1.96 \sqrt{pq/n}$

= $P \pm 1.96 \sqrt{0.069 \times (1-0.069)/ 684}$

= (5.0% to 8.8%)

4.5.1.2 Standardization of prevalence rates of DM by Age and sex in the study subject

Tarauni population 2006 census 15 years and above 116,660

Males 60,430

Females 56,230

Table 4.6: Age standardized prevalence of Dm by sex

Age grp (yrs)	Male popn. crude prev. (%) Expected			females crude prev.(%) Expected		
15-24	21349	0	0	20766	2.3	477.6
25-34	14583	2.3		16799	4.6	772.8
35-44	355.4			8866	14.7	1303.3
45-54	10266	6.3		4666	8.8	410.6
55-64	646.8			2567	25.0	642.7
≥65	6766	17.9		2683	50.0	1341
	1448.0					
	3500	33.3				
	1165.5					
	3850	6.25				
	243.0					
Total	60,430	7.6	3859	56,230	9.2	4948

Adjusted prevalence for age for the males = $3859/60430 \times 100$
= 6.4%

Adjusted prevalence for age for the females = $4948/56230 \times 100$
= 8.8%

95% C I = $P \pm 1.96 \times \sqrt{pq/n}$

1) Males = $0.064 \pm 1.96 \times \sqrt{(0.064 \times 1-0.064) / 250}$
= (2.9% to 9.4%)

2) Females = $0.088 \pm 1.96 \times \sqrt{0.088 \times 1- 0.088)/434}$
= (4.0% to 13.6%)

4.5.2 Prevalence of impaired Glucose tolerance post OGTT

This study found a total of 42 (6.2%) subjects with impaired glucose tolerance, 10 (1.5%)

males and 32 (4.7%) females, their mean age was $36.1 \text{ yr} \pm (7.9)$. The mean age for the

male subjects with IGT was $33.0 \text{ yrs} \pm (8.8)$, while it was $37.0 \text{ yrs} \pm (7.5)$ for the females.

($t=1.41$; $p=0.17$) there is no statistically significant difference between the sexes' mean age .

4.5.3 Distribution of plasma glucose among the subjects with diabetes

4.5.3.1 Casual Plasma glucose of the subjects with diabetes

The mean \pm (SD) Casual plasma glucose of all the subjects with diabetes was 11.7 mmol/l .

The mean for male subjects was $12.5 \pm (6.8) \text{ mmol/l}$ while it was $11.3 \pm (5.7) \text{ mmol/l}$ for

their female counterparts ($t=0.71$; $p\text{-value}=0.48$) there is no statistically significant

difference between the CPG of the sexes'.

The distribution of casual plasma glucose among subjects with DM is as shown in table4. 7

Table 4.7: Distribution of casual plasma glucose among the subjects with diabetes

CPG	Males No. (%)	Females No. (%)	Total No. (%)
3.5-4.9	1 (1.7)	2(3.4)	3(5.1)
5.0-7.9	4(6.8)	6(10.2)	10(16.9)
8.0-10.9	6(10.2)	21(35.6)	27(45.8)
11.0-13.9	2(3.4)	4(6.8)	6(10.2)
14.0-16.9	2(3.4)	2(3.4)	4(6.8)
17.0-19.9	2(3.4)	1(1.7)	3(5.1)
20.0-22.9	0(0)	2(3.4)	2(6.8)
≥23	2(3.4)	2(3.4)	4(6.8)
Total	19(32.2)	40(67.8)	59 (100)

Of these 59 subjects with diabetes 13(22%) had CPG ranging between 3.5mmol/l to 7.9mmol/l while 46 (78%) had CPG in the range of 8.0mmol/l to 30mmo/l.

4.5.3.2 Casual Plasma glucose of the subjects with IGT

A) The mean \pm (SD) casual plasma glucose of all the subjects with IGT was $9.1 \pm (1.4)$ mmol/l. The mean for the males was $8.9 \pm (0.7)$ mmol/l, while it was $9.2 \pm (1.4)$ mmol/l for the female pre-diabetics

($t= 0.65$; $p=0.52$) there is no statistically significant difference .

4.5.3.3 Fasting plasma glucose of the subjects with diabetes

The mean \pm (SD) fasting plasma glucose of all the subjects with diabetes was $9.8 \pm (4.4)$ mmol/l with a range of 7.0mmol/l to 22.0 mmol/l. The mean for the males was $9.6 \pm (4.7)$ mmol/l , while it was $9.8 \pm (4.4)$ mmol/l for the females with diabetes. ($t= 17$; $p= 0.86$). There is no statistically significant differences between the sexes'.

B) The mean FBS for all the subjects with IGT was $6.2 \pm (0.5)$ mmol/l. The mean was $6.9 \pm (0.7)$ mmol/l for the male pre-diabetics, while it was $6.9 \pm (0.5)$ mmol/l for their females counterparts. ($t= 0.00$; $p= 1.00$) there is no statistically significant difference between the sexes'.

4.5.3.4 Subjects with IFG

This study did not discover any subject with isolated IFG.

4.6 SOCIO-DEMOGRAPHIC CHARACTERISTIC OF THE SUBJECT WITH DIABETES AND IGT

4.6.1 Age and sex distribution of the subjects with diabetes and those with IGT

There were 59 subjects with diabetes 19(32.2%) males and 40 (67.8%) females. The mean

age of all the subjects with diabetes was 45.2 ± 12.3 years, with age range Of 20-70 years.

The mean age for the males was 49.4 yrs (± 12.2) , while it was $41.7 \pm (11.7)$ yrs for the

females ($t= 2.32$; $p=0.02$) . The males were older than the females and this was statistically

significant ($p=0.02$)

Table 4.8: Age distribution of the subjects with diabetes by sex

Age group(years)	Males N(%)	Females N(%)	Total N(%)
15-24	0(0)	2 (3.4)	2(3.4)
25-34	2(3.4)	6(10.2)	8(13.6)
35-44	3(5.1)	17(28.8)	20(33.9)
45-54	5(8.4)	6(10.2)	11(18.6)
55-64	8(13.6)	7(11.9)	15(25.4)
≥ 65	1(1.7)	2(3.4)	3(5.1)
Total	19(32.2)	40(67.8)	59(100)

4.6.2 Age and sex distribution of the subjects with IGT

There were a total of 42 (6.2%) IGT, 10 (1.5%) males and 32 (4.7%) females. The mean age of all the subjects with IGT was 36.1 ± 7.9 years, with age range between 25 and 54 years. The mean age for the males was 33.0 ± 8.8 years, while it was 37.0 ± 7.5 years ($t = 1.41$; $p = 0.17$). There is no statistically significant difference in their mean age. Majority 35 (83.4%) of the subjects with

IGT were in age group 25-44 years; while 45-59 year age group account for 16.6%.

4.6.3 Social class distribution by sex of the subjects with diabetes

The social class distribution of the subjects with diabetes showed that only 2 (3.4%) of the 59 subjects with diabetes were in class I (1 male and 1 female), another 2 (3.4%) in class II both were males, there were 10 (16.9%) diabetics in class III (3 males and 7 females). However, in class IV were 14 (23.7%) diabetics, 4 males and 10 females, while in class V there were 31 (52.5%) diabetics 9 males and 22 females; 93.1% of all the subjects with diabetes were in classes III to V and only 6.9% were found in classes I and II.

4.6.5 Social class distribution of the subjects with IGT

The social class distribution of the subjects with IGT showed that 2 (4.8%) were in class I (1 male, 1 female), 3 (7.1%) were in class II (2 males, 1 female), 5 (11.9%) were in class III (3 males, 2 females) and 17 (40.5%) were in class IV (2 male and 15 females), while 15 (35.7%) were in class V (2 male and 13 females). This showed that 88.1% of the pre-diabetics were found in between class III and V while only 12.9% were found in classes I and II.

4.7.0 CLINICAL CHARACTERISTICS OF THE SUBJECTS WITH DIABETES AND IGT

4.7.1 Family history of diabetes mellitus among the subjects with diabetes

Of the 59 subjects with diabetes in this study, 28 (47.5%) had positive family history of diabetes mellitus and 31 (52.5%) had no family history of the disease or did not know.

4.7.2 Family history of Diabetes mellitus among the subjects with IGT

This study found that 32(76.2%) of the subjects with IGT did not have family history of Dm while 10 (23.8%) had positive family history of DM.

4.7.3 Physical inactivity among the subjects with diabetes

This study found that 19 (32.2%) of the 59 subjects with diabetes were physically active while 40 (67.8%) were physically inactive. Of these 59 subjects with DM, only 4(6.8%) were found to be very active(2 males ,2 females), 15 (25.4%) were moderately active (7 males and 8 females) and 40(67.8%) were inactive (10 males and 30 females). There was no statistically significant difference between male and female subjects with diabetes ($\chi^2 = 2.97$, $P = 0.22$).

4.7.4 Physical inactivity among the subjects with IGT

Of the 42 subjects with IGT in this cohort, 2(4.8%) were very active(1 male, 1 female), 8 were moderately active (2 males and 6 females) while 32(76.2%) live a sedentary life (7 males 25 females) There is no statistically significant difference between the sexes' ($\chi^2 = 0.83$, $P = 0.66$)

4.7.5 Alcohol use among the subjects with diabetes and the IGT

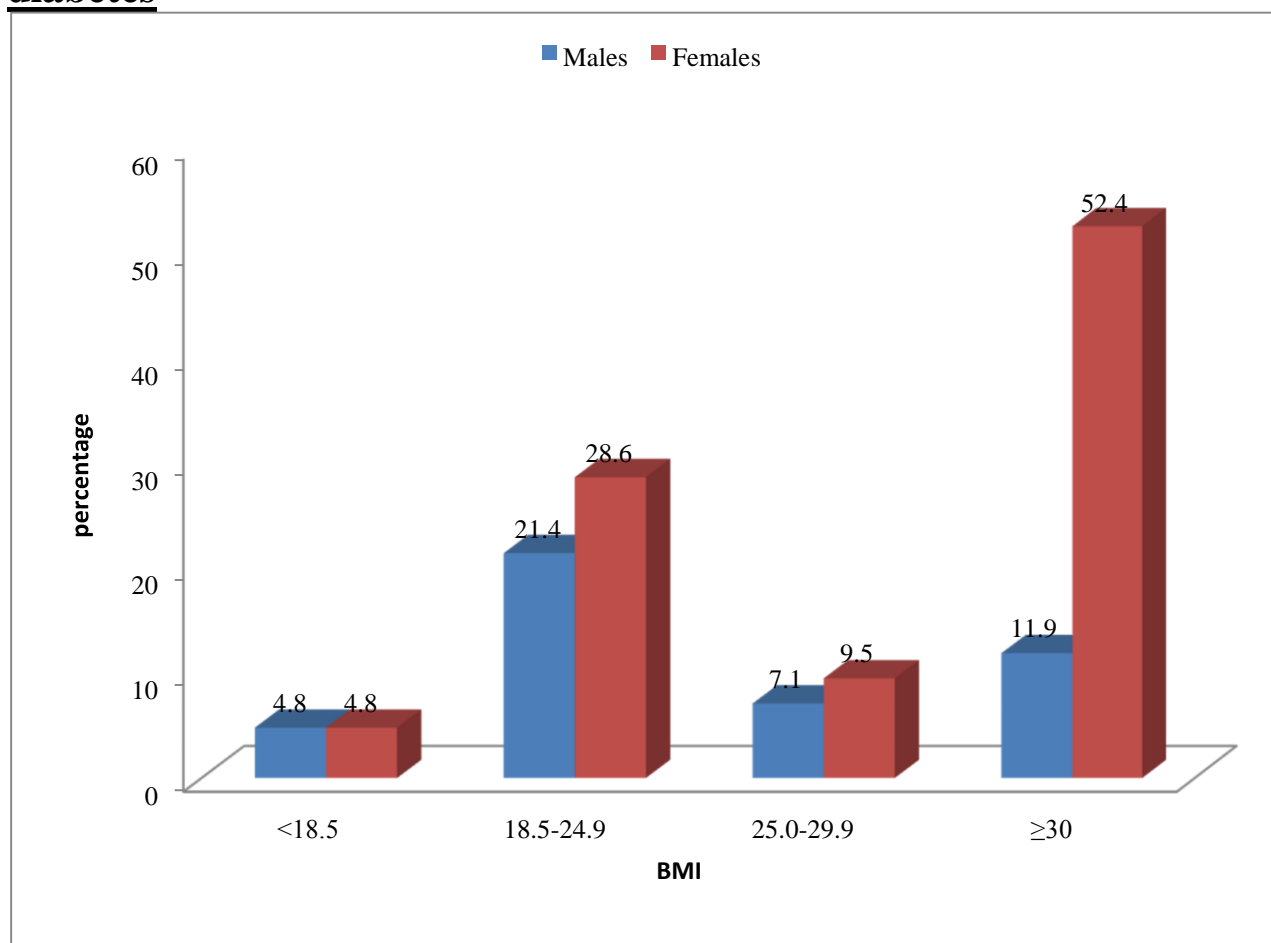
History of alcohol use was not documented in any of the persons with diabetes or those with IGT.

4.7.6 Anthropometric indices of persons with Diabetes and IGT

4.7.6.1 *Distribution of BMI among the subjects with diabetes by gender*

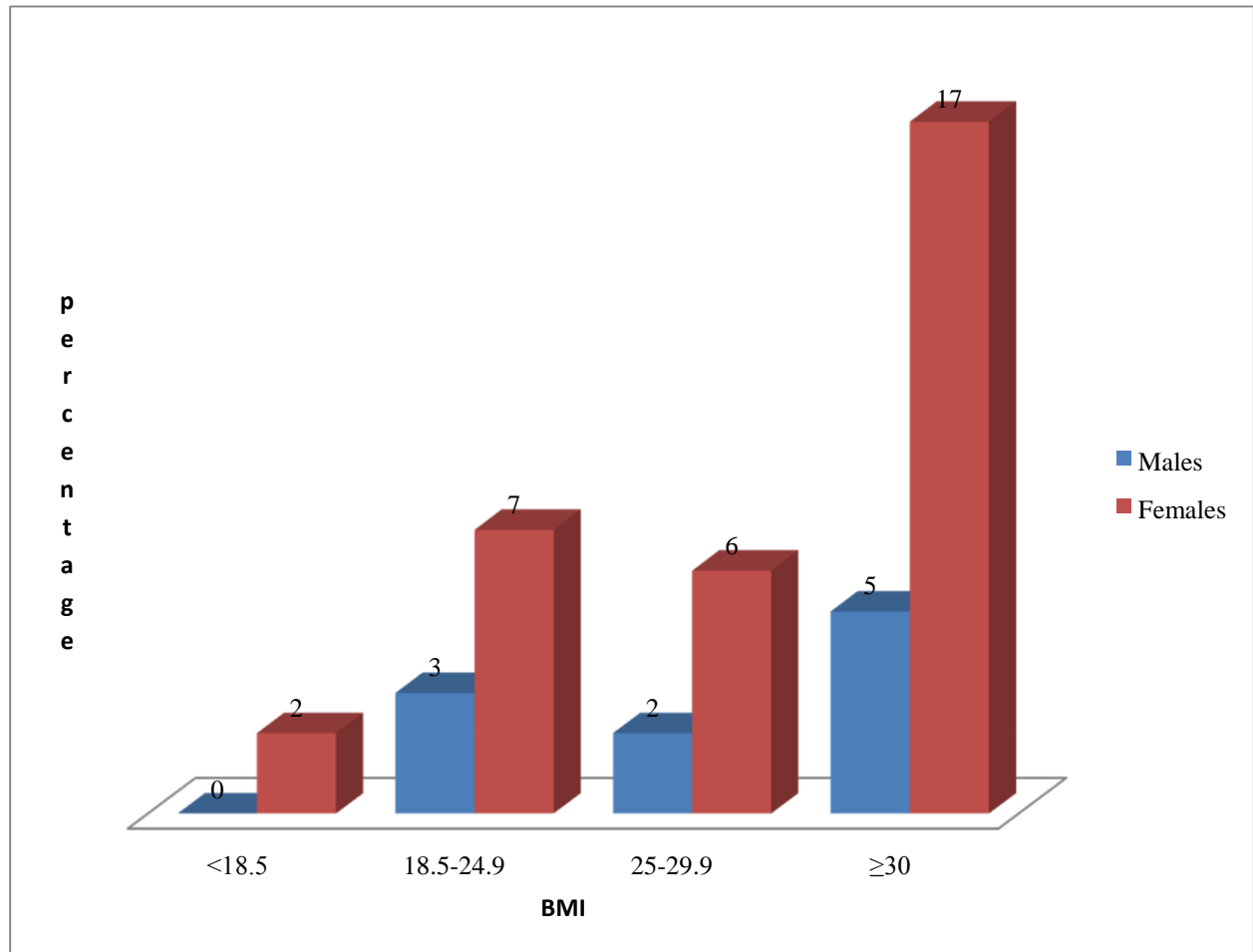
The distribution of BMI among diabetics by gender is as shown in figure IV. The mean BMI of all the subjects with diabetes was $26.2 \pm 5.4 \text{ kg/m}^2$. The mean for the males was $23.7 \pm 4.5 \text{ kg/m}^2$, while it was $28.0 \pm 5.1 \text{ kg/m}^2$ for the females; ($P= 0.002$ $t=3.1$) females have significantly higher BMI.

Figure IV: Distribution of BMI by sex among the subjects with diabetes



The BMI of the females is higher than that of the males in virtually all age groups except the underweight group.

Figure V: Distribution of BMI by sex among the subjects with IGT



Similarly BMI of the female with IGT was higher than their male counterparts.

4.7.6.2 Waist circumference (WC) of the subjects with diabetes and those with IGT using IDF cutoff values.

The mean \pm SD WC of all the subjects with diabetes was 93.2 ± 11.8 cm, it was 86.8 ± 11.6 cm for male subjects with diabetes while the mean for the females was 96.6 ± 10.5 cm. ($P=0.01$, $t=3.24$). The females have statistically significant higher WC.

The mean \pm SD WC for all the subject with IGT was 96.1 ± 17.3 cm, it was $80.6 \pm (15.8)$ cm for the males while it was 98.3 ± 15.3 cm for the females ($P=0.02$, $t=3.17$). The females with IGT have statistically significant higher WC than the males.

4.7.6.3 Obesity based on WC using IDF cutoff value in subjects with diabetes and IGT

It was found that 36 (90%) of the female subjects with diabetes have WC of ≥ 80.0 cm indicating presence of obesity. For their male counterparts only 6 (31.6%) have a WC of ≥ 94 cm showing presence of obese in this group.

Similarly 26 (81.3%) out of 32 female with IGT were found to have increased WC above 80cm; and 3 (30%) out of 10 male with IGT had WC ≥ 94 cm i.e obese.

4.7.7 Hypertension among subjects with Diabetes mellitus

4.7.8 Prevalence of hypertension among the subjects with diabetes and those with IGT

This study found that 44 (74.6%) out of 59 subjects with diabetes were hypertensive while 11 (26.2%) out of all the subjects with IGT were hypertensive.

4.8.0 RISK FACTORS AND OCCURANCE OF DIABETES MELLITUS

Risk factors for DM in subjects with diabetes were compared with 583 normal subjects to assess the significance of those risk factors.

4.8.1 Age and Diabetes mellitus

The prevalence of DM is known to increase with advancing age. The relationship between age and DM in this study is as shown in table 4.9, which showed that 29 (49.1%) of all the subjects with diabetes were 45yrs and above as against 168 (24.5%) of normal subjects found in the same age group.

Table 4.9 : Relationship between age and DM

Age (yrs)	Diabetics	Normal	Total
	N (%)	N (%)	
15-24	2 (0.3)	131(20.4)	133(20.7)
25-34	8 (1.3)	187 (29.1)	195(30.4)
35-44	20(3.1)	130 (20.2)	150 (23.3)
45-54	11(1.7)	77 (12.0)	88 (13.7)
55-64	15 (2.3)	42 (6.5)	57(8.9)
≥65	3 (0.5)	16(2.5)	19(3.0)
Total	59(9.2)	583(90.8)	642(100)

$\chi^2 = 40.7$, df = 5, P = 0.001

The relationship is statistically significant.

4.8.2 RELATIONSHIP BETWEEN WEIGHT AND DIABETES MELLITUS

The prevalence of DM is also known to be higher in person with obesity. The relationship in our cohort is shown in viz:

1- **BMI:** Table 4.10 shows the relationship between higher BMI and development of diabetes mellitus.

Table 4.10: Relationship between weight and Diabetes mellitus

<u>Weight category</u>	<u>Diabetics Normal</u>	
	N (%)	N (%)
<18.5	4 (6.7)	105 (18.0)
18.5-24.9	21 (35.6)	317 (54.4)
25-29.9	10 (16.9)	97(16.63)
≥ 30	24 (40.6)	64(10.97)

Total	59(100)	583 (100)
--------------	----------------	------------------

$$\chi^2 = 42.1, \quad df = 3, \quad P = 0.001$$

There is a statistically significant relationship between overweight and Diabetes mellitus **2-Waist circumference (WC):** The mean WC for the male subjects with DM was 86.6 ± 11.6 and 98.3 ± 15.3 for the female subjects with the diabetes; the mean WC for normal male subjects was 80.9 ± 10.9 while it was 86.9 ± 14.0 for normal female subjects, this shows that subjects with DM have higher mean WC compared to the normal subjects.

4.8.3 PHYSICAL INACTIVITY AND DM

The relationship between physical inactivity and occurrence of diabetes mellitus is shown in table 4.11.

Table 4.11: Relationship between physical inactivity and diabetes mellitus

<u>Physical activity</u>	<u>Diabetic</u>	<u>Normal</u>
	N (%)	N (%)
Very active	4(6.8)	124(21.3)
Moderately active	15 (25.4)	254(43.6)
Inactive	40(67.8)	205 (35.2)
Total	59(100)	583 (100)

$$\chi^2 = 16.3, \quad df = 2, \quad P = 0.001$$

The less active subjects had increased prevalence of DM

4.8.4 Social class and Diabetes Mellitus

Table 4.12 Relationship between social class and DM

Social class	Diabetics	Normal	Total
	N(%)	N(%)	
I	2	23	25
II	2	47	49
III	10	118	128
IV	14	176	190
V	31	219	250
Total	59	583	642

$$\chi^2 = 5.7, \quad P = 0.22$$

There is no statistically significant relationship

4.8.5 Family History and Diabetes mellitus

Twenty eight (28) of the 59 subjects with DM have positive family history of the disease as against 71 out of 583 normal subjects with family history. ($\chi^2 = 51.13$; $df = 1$, $p = 0.00$).

This shows that the relationship is statistically significant.

4.8.6 Tobacco use and Diabetes mellitus

Only one (1.7%) subject with diabetes admitted to the use of tobacco as against 23 (4%) of the normal subjects. ($\chi^2 = 0.75$; $p = 0.39$) There was no statistically significant relationship between tobacco use and occurrence of DM from this study.

4.8.7 Alcohol and Diabetes mellitus

None of the subjects with diabetes admits to the use of alcohol, and only 2 of the normal subjects admit the use of alcohol ($\chi^2 = 0.2$; $p = 0.65$) the relationship is statistically not significant.

4.8.8 Hypertension and the prevalence of Diabetes mellitus

The prevalence of hypertension among the subjects with diabetes is shown in table 4.13

Table 4.13: prevalence of hypertension among the subjects with diabetes

	<u>Hypertension Diabetics</u>	
	<u>Normal</u>	<u>N(%)</u>
	<u>N(%)</u>	
Yes	44(74.5)	150 (25.7)
No	15(25.5)	433(74.3)
Total	59(9.2)	583 (90.8)
$\chi^2 = 60.0$; df=1 p= 0.001		

The relationship between hypertension and DM is statistically significant

4.9.0 Risk factors among the subjects with IGT

4.9.1 Age and IGT

The mean age of all the subjects with IGT was 36.1 ± 7.9 years as against 34.5 ± 13.1 for the normal subjects. ($\chi^2 = 0.78$; p= 0.44) there is no significant difference between the ages of the two groups.

4.9.2 Weight and IGT

1-Mean BMI of the subjects with glucose intolerance was $29.7 \pm 8.6 \text{ kg/m}^2$, it was $23.1 \pm 5.2 \text{ kg/m}^2$ For the normal subjects (t= 7.53; p= 0.001). This shows that subjects with IGT have statistically significant higher weight compared to the normal subjects.

2-The mean WC of the subjects with IGT was 96.1 ± 17.3 , while it was 83.2 ± 12.3 for the normal subjects ($t= 6.36$; $p= 0.01$) this shows that subjects with IGT have statistically significant excess weight compared to the normal subjects.

4.9.3 Family history of DM and IGT

Ten out of 42 subjects with IGT have positive family history of Dm as against 71 out 583 normal subjects ($\chi^2= 4.7$, $p= 0.03$). There is statistically significant relationship between family history of

Dm and IGT.

4.9.4 physical inactivity and IGT

Of the 42 subjects with IGT 10 were active as against 71 out of 583 normal subjects. ($\chi^2 = 4.7$; $p=0.03$). there is statistically significant relation between physical inactivity and IGT

4.10 Odd ratio estimate of risk factors for Dm from multiple logistic regression

Table 4.14: Odd ratio (OR), estimate of risk factors for Dm from

Risk factors	Odds ratio (OR)	95% C I	P value
AGE	0.83	0.68-1.00	0.042*
BMI	0.55	0.30-1.00	0.049*
FAMILY HISTORY	0.97	0.94-1.01	0.88
PHY. INACTIVITY	0.97	0.59-1.57	0.84

multiple logistic regression

*Age and BMI are the independent risk factors for Dm in this study.

4.11 Risk factor profile of subjects with DM, IGT and normal subjects

Table 4.15: Mean CPG and risk factor profile of the subjects with

Diabetes, IGT and normal subjects

<u>Diabetics</u>		<u>Males</u>
<u>Females</u>		
CPG(mmol/l)	12.5 ± 6.8	11.3 ± 5.7
Age (yrs)	49.4 ± 12.2	41.7 ± 11.7
BMI (kg/m ²)	23.7 ± 4.5	28.0 ± 5.1
WHR	0.93 ± 0.05	0.91 ± 0.06
<u>IGT</u>		
CPG(mmol/l)	8.9 ± 0.7	9.2 ± 1.4
Age (yrs)	33.0 ± 8.8	37.0 ± 7.5
BMI (kg/m ²)	30.7 ± 11.4	29.2 ± 7.7
WHR	0.91 ± 0.1	0.89 ± 0.06
<u>Normal subjects</u>		
CPG(mmol/l)	6.1 ± 0.8	6.1 ± 0.7
Age (yrs)	36.2 ± 13.2	33.5 ± 11.9
BMI (kg/m ²)	21.9 ± 3.8	23.9 ± 5.8
WHR	0.88 ± 0.06	0.87 ± 0.07

The subjects with DM and those with IGT have higher values of all the known risk factors for DM compared with the normal subjects.

CHAPTER FIVE

DISSCUSSION

5.0 Back ground:

Diabetes mellitus previously considered a disease of minor significance to world health, is now taking its place as one of the main threats to human health in the 21st century⁵.

Recently (2010) the International Diabetes Federation reports that currently some 285 million people worldwide or 6.6% of the adult population have diabetes at the moment and this figure will increase to 438 million or 7.7% by 2030⁵. The major part of this numerical increase will occur in the developing countries Nigeria included.

It is against this background that this study was conducted to update the prevalence of DM among adults in Urban Kano and it is of particular interest to study the epidemiological transition of the state and to identify the risk factors associated with the development of the disease.

This population-based study that was carried out among a random sample of 684 subjects comprising 250 males and 434 females. The female preponderance observed may be due to the polygamous nature of the society in which the study was carried out.

The method used in the screening was estimation of CPG which is an accepted method of obtaining a crude estimate of the frequency of diabetes in a population and it is less expensive than the standard WHO method (Fasting and 2 hr post 75g glucose load). However, the subset that were detected to have indeterminate status with CPG Of 7.8mmo/l but less 11.1mmol/l on screening day where subjected to the standard OGTT for confirmation of their status.

5.1 Prevalence of Dm in this study.

The crude prevalence of DM in this study was 8.6% (4.1% were living with diabetes before this study while 4.5% was newly diagnosed to have diabetes during this study) and age standardized prevalence of 6.9%. The crude prevalence rate was 7.6% for the males and age standardized prevalence of 6.4%, while the crude prevalence for the females was 9.2% and age standardized prevalence of 8.8%. This shows a dramatic rise from the previously reported prevalence by NCD

survey of 1997 that reported 2.5% as the crude prevalence for urban Kano²⁷.

The time interval between the two surveys may explain the rise in the prevalence. Recently, Gezawa⁶⁸ reported a crude prevalence of 7% in Maiduguri, while Sabir A⁶⁹ found a crude prevalence of DM in urban Sokoto to be 4.6%. This may be because both Sokoto and Maiduguri are less industrialised as compared to Kano; Moreover, Kano is undergoing a more rapid change in social and economic characteristic that is associated with a shift in life style marked by consumption of high calorie, energy dense diet and low level of physical activity. This transition may contribute to the higher prevalence of DM in Kano.

Our finding was similar to Nyenwe's³⁰ and NCDS²⁷ finding of 7.9% standardized prevalence in port in Port Harcourt and 7.2% in Lagos mainland respectively. However a prevalence of 16% among non oil workers and 23.4% among oil workers was found by Nwafor and Owhoji⁷⁰ in Port Harcourt, higher energy dense foods and less physical activity may account for the higher prevalence compared with Kano.

In Tanzania and Cameroon, the only two African countries where repeated local surveys have been undertaken using standard WHO methodology, the prevalence has increased six- to 10-fold within a 10-year period²⁶. This agrees with the IDF assertion that developing countries will be more affected and is also in agreement with significant rise recorded in Kano after 13years of previous survey.

5.2 Type of Dm in the study

In this study 95.6% of the subjects with diabetes are presumed to have type 2 DM, based on age at onset , long duration without medication and majority being obese or overweight. This is consistent with the findings of NCDS²⁷, Puepet²⁸, Ohwovoriole²⁹ and Nyenwe³⁰; it also agrees with, Gezawa⁶⁹

Sabir⁷⁰ and Nwafor and Owhoji ⁷¹

5.3 Prevalence of glucose intolerance in the study

There was no study in Kano to determine the prevalence of glucose intolerance in the past.

The prevalence of glucose intolerance from this study was found to be 6.2%. This is lower than the prevalence of 7.7% reported in Zaria⁷². The difference may be due to a smaller sample size of 50 subjects used in the Zaria study.

The prevalence of both IGT and IFG is much higher in USA than in our environment. A study among

U.S. adults (40 to 74 years of age), 15.6 % (14.9 million) had IGT, and 9.7 % (9.6 million) had IFG⁷⁴. The higher prevalence of IGT/IFG may be due to the older age of their study subjects compared with our cohort, however, differences in life style, more robust economy and industrialization may further explain why they have higher prevalence.

5.4 Correlates of diabetes mellitus

This study found a significant positive correlation between obesity (high BMI, high WHR, and high WC), physical inactivity, advancing age, family history of diabetes and Diabetes Mellitus,

While no significant correlation was found between diabetes mellitus and, social class, alcohol consumption, or cigarette smoking in this study population.

5.4.1 Age and the prevalence of DM

In this study the prevalence of DM increased with advancing age. Analysis by age group showed that 16.9% were 15-34 years of age, 33.9% were in age group 35-44year while 49.2% of all the subjects with diabetes were in age group 45-80 years. This suggests that the risk of developing DM increases particularly after middle age. The NCD survey also found a positive correlation between

DM and increasing age²⁷. Similarly, a positive correlation between DM and older age was also found

in o ther local studies^{28,92,30,69-71}

5.4.2 Obesity and prevalence of DM

In this study 57.6% of the subjects with diabetes were either obese or overweight. The prevalence of DM was found to increase across BMI categories. NCD²⁷, Puepet²⁸, Ohwovori²⁹, Nyenwe ³⁰, Gezawa⁶⁹, Sabir⁷⁰, all reported obesity or overweight are positively associated with the development of DM. Ozturk ⁷⁵ found over 60% of the subjects with diabetes was either obese or overweight in Turkey.

5.4.3 Hypertension and prevalence of DM

This study found more hypertensives (74.6%) in the subjects with diabetes compared to the normal subjects (25.7%) This finding is similar to NCDS²⁷ finding were prevalence of DM increased with rising systolic blood pressure. Similarly, Puepet²⁸, Nyenwe ³⁰, Gezawa⁶⁹ and

Sabir⁷⁰ all found the prevalence of hypertension among the subjects with diabetes to be higher than in normal subjects.

5.4.4 Physical inactivity and the prevalence of DM

Physical inactivity is a recognized risk factor for DM. This study found that the level of physical inactivity was higher in subjects with DM as compared to the normal subjects. A statistically significant relation exists between DM and physical inactivity. A similar finding was reported by Nyenwe³⁰ who also found a significant relationship between prevalence of DM and physical inactivity, this is because of accumulation of excess calories in the body predisposing the individual to insulin resistance. In contrast to NCDS²⁷ and Puepet²⁸ who found a statistically not significant relation between DM prevalence and physical inactivity.

5.4.5 Parental history of DM and prevalence of DM

The odds of diabetes occurring is greater than that in the general public of similar age if one has one or more parents that have DM. This was found to be true in our cohort. This is due the genetic abberation leading to different kind of DM and glucose intolerance.

5.4.6 Social class and the prevalence of Dm

The prevalence of DM increase down the social class with 76.3% of the subjects with diabetes in classes IV and V while only 6.8% were in classes I and II. This is most likely due to ignorance among the lower classes. Similar findings were reported by the NCDS, in contrast Puepet found the prevalence of DM to be higher in the upper social class.

5.4.7 Alcohol use and the prevalence of DM

Alcohol use was not an issue as very few subjects in the study environment consume alcohol hence it is difficult to relate it to the occurrence of DM in this study group. No significant relationship was observed by Puepet as all his discovered subjects with diabetes had stopped using alcohol prior to his study. Similarly, the NCDS did not find consistent relationship

between alcohol and the prevalence of Dm but they found that below the age of 45years, heavy drinkers had a higher prevalence of DM than those drinking mildly or moderately

5.4.8 cigarette smoking and the prevalence of DM

Cigarette smoking was also not a social vice among the study subjects, only one subject with DM admitted the use cigarette as against 23 normal subjects. It is difficult to relate the occurrence of DM with cigarette smoking from this study.

CHAPTER SIX

6.0 CONCLUSION AND RECOMMENDATION

6.1 Conclusion: The overall prevalence rates of diabetes mellitus observed in this study is higher than previously reported by the NCDS for Kano state. It seems that the prevalence is increasing which is in keeping with the disease pandemic. The situation is the same in other parts of the country. The predominant form of the disease in the population studied is Type 2 DM; and all of the newly diagnosed patients were unaware of their condition which shows that the disease can go unnoticed for quite some time.

Advanced age, overweight, family history of the disease, physical inactivity and hypertension were found to be the risk factors associated with the disease in our cohort, these findings are consistent with many other works in Nigeria. However, social class, Alcohol use as well as cigarette smoking were not found to associated with the disease in this study.

6.2 Recommendations

The recommendations from this study includes:

- 1- There is need for extensive public campaign programmes to create awareness among the public with regard to early detection of DM and its risk factors so as to mount prevention program.

- 2- There is the need for frequent updating of the data to monitor disease trend and burden on the population
- 3- There is need to follow up the persons with glucose intolerance to see the effect of life style changes recommended for them.

References

- 1) Macfarlane, A. Gill, G . History of diabetes (review) . *pract. Diabetes Digest*; 1991 2:2, 54-62.
- 2) History of diabetes. Los Angeles Chinese learning centre www.cljhealth.com (assessed march 2010)
- 3) Alberti KGMM, Zimmet PZ, for WHO Consultation, Definition and Classification of Diabetes Mellitus and its Complication. *Diabetic Medical Journal* 1998; 15. 539-553
- 4) The Expert committee Report on Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications. *Diabetes Care*, Jan. 2003 vol. 26, supplements 1
- 5) Diabetic Prevalence, International Diabetes Federation Home page www.eatlas.idf.org (accessed May 2011).
- 6) World Health Organization Definition, Diagnosis and classification of Diabetes Mellitus: Report of a WHO Consultation, Geneva, Switzerland: WHO Publication.

- WHO/NCD/NCS 1999: /99-2.
- 7) Zachary TBG; Type 2 Diabetes Mellitus: its prevalence, causes, and Treatment. *Diabetes Care*, 1998. 21.5 860-865.
 - 8) Wild S, Gojka R, Anders G; Global prevalence of Diabetes ,estimates for the year 2000 and projections for 2030 . *Diabetes Care*, 2004; 27: 1047-1053.
 - 9) King H, Ronald EA, William HH; Global Burden of Diabetes Mellitus. *Diabetes care* 1998; 2. 9: 1414-1430.
 - 10) Bamgboye E L; End Stage Renal Disease in Sub Saharan Africa ; *Ethnicity and Disease*. 2006;16:S2-5 to S2-9.
 - 11) Mac Farlane IA, Brown RM, Smyth RW, et al. Bacteraemia in Diabetics. *Journal of Infections*.1986;213-219.
 - 12) Wheat LJ ; Infection and diabetes Mellitus. *Diabetes Care*. 1980;3:187- 197.
 - 13) David M Nathan. Long Term Complications of Diabetes Mellitus. *NEJM*.1993;328:1676-1685 .
 - 14) Oldroyd J, Banerjee M, Cruikshank K. Diabetes and ethnic minorities; *Postgrad Med J*, 2005;81: 486-490.
 - 15) Oyegbade OO, Abioye-kuti EA , Kolawole BA, Ezeoma I T, Bello IS; Screening for Diabetes Mellitus in a Nigerian Family Practice Population . *SA Fam Prac*.2007;49 (8):15 -15d.

- 16) Wild S, Roglic G , Green A ;Global prevalence of Diabetes . *Diabetes Care*.2004;27: 1047-1053.

- 17) Cowie CC, Rust KF, Bryd-Holt DD, Eberhart MS, Elegal KM: Prevalence of Diabetes and Impaired Fasting Glucose in Adults in the US population. *Diab Care*. 2006; 29:1263-1268.

- 18) Passa P. Diabetes trends in Europe: *Diabetes Metab Res Rev*.2002;18: S3–S8.

- 19) Aschner P. Diabetes trends in Latin America; *Diabetes Metab Res Rev*.2002;18: S27–S31.

- 20) Leung GM, Lam KSL. Diabetic complications and their implications on health care in Asia; *HKMJ* 2000; 6: 61-68.

- 21) Motala AA. Diabetes trends in Africa ; *Diabetes Metab Res Rev*.2002; 18: S14–S20.

- 22) Sobngwi E, Mauvais-javis F , Vexiau P , Mbanya JC, Gautter JF. Diabetes in Africans ; *Diabetes and metabolism*. 2001; 27, 628-634.

- 23) Fisch A, Pichard E, Prazuc T H. Leblanc H, Sidibe Y, Bricker G. Prevalence and risk factors of diabetes mellitus in the rural region of Mali (West Africa): a practical approach; *Diabetologia*. 1987; 30 : 859-862.

- 24) Ceesay MM, Morgan MW, Kamanda MO, Willoughby VR, Lisk D R . Prevalence of diabetes in rural and urban populations in southern Sierra Leone: a preliminary survey; *Trop Med and Int Health*. 1997;2: 3 pp 272–277.

- 25) Albert G.B. Amoah AGB, Owusu SK , Adjei S. Diabetes in Ghana: a community based prevalence study in Greater Accra; *Diab Res and Clin Prac.*2002;56:197–205.

- 26) Levitt N S. Diabetes in Africa: epidemiology, management and healthcare challenges *BMJ.* 2008;94:1376–1382.

- 27) Akinkugbe O O (ed) The National Expert Committee on Non communicable Diseases in Nigeria, Final report on Non communicable disease survey 1997: series 4: 64-90.

- 28) Pupet FH. The Prevalence of Diabetes Mellitus and associated Risk factors in Adults in Jos. National Postgraduate Medical College of Nigeria. 1996. Part 2 FMCP Dissertation.

- 29) Ohwovoriolè, AE; Kuti J.A; Kabiawu S I O. Casual blood glucose levels and Prevalence of Undiscovered diabetes Mellitus in Lagos Metropolis, Nigeria. *Diabetes Research and Clinical Practice.* 1988; 4 : 153-158.

- 30) Nyenwe EA, Odia JO, Anele E I, Aaron O, Seye B. Type 2 Diabetes in Adults Nigerians: A Study of its Prevalence and Risk Factors in Port Harcourt Nigeria. *Diab Res Clin Prac.* 2003; 62:177-185.

- 31) Garancini MP , Calori G , Ruotolo G, Manara E , IzzonA. , Et al . Prevalence of NIDDM and impaired glucose tolerance in Italy: an OGTT-based population study *Diabetologia.*1995;38:306-313.

- 32) Baltazar JC , Ancheta AC, Aban BI, Fernando RE , Baquilod MM. Prevalence and correlates of diabetes mellitus and impaired glucose tolerance among adults in Luzon, Philippines ; *Diab Res and Clin Prac.*2004; 64:107–115.
- 33) Gu D, Reynolds K, Duan X, Xin X; the Inter ASIA Collaborative Group. Prevalence of diabetes and impaired fasting glucose in the Chinese adult population: International Collaborative Study of Cardiovascular Disease in Asia (Inter ASIA) *Diabetologia* 2003; 46:1190-119.
- 34) Mustelin L, Pietilainen KH, Rissanen A, Sovijarvi AR. Acquired obesity and poor physical fitness impair expression of genes of mitochondrial oxidative phosphorylation in monozygotic twins discordant for obesity; *Am J Physiol Endocrinol Metab.*2008;295: E148-E154.
- 35) Ramachandran A , Snehalatha C, Mary S, Mukesh B, Bhaskar AD, Vijay V. The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1): *Diabetologia.*2006; 49: 289–297.
- 36) Rimm EB, Chan J, Stampfer MJ, Colditz GA, Willett WC: Prospective study of cigarette smoking, alcohol use and the risk of diabetes in men. *BMJ.*1995; 310:555–559.

- 37) Kawakami N, Takatsuka N, Shimizu H, Ishibashi H: Effects of smoking on the incidence of non-insulin-dependent diabetes mellitus: replication and extension in a Japanese cohort of male employees. *Am J Epidemiol.* 1997; 145:103–109.
- 38) Facchini FS, Hollenbeck CB, Jeppesen J, Chen YD, Reaven GM: Insulin resistance and cigarette smoking. *Lancet.* 1992; 339:1128–1130.
- 39) Eriksson KF, Lindgarde F: Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise. *Diabetologia.* 1991; 34:891–898.
- 40) Pan X, Li G, Hu YH, Wang J, Yang W; Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance: the Da Qing IGT and Diabetes Study. *Diabetes Care.* 1997; 20:537–544.
- 41) Kriska AM, Hanley JGA, Harris SB, Zinmann B. Physical Activity, Physical Fitness, and Insulin and Glucose Concentrations in an Isolated Native Canadian Population Experiencing Rapid Lifestyle Change; *Diabetes Care.* 2001 24:1787–1792.
- 42) Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, et al Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med.* 2001; 344:1343–1350.
- 43) Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM: Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *NEJM.* 2002; 346:393–403.

- 44) Yates T, Khunti K, Bull F, Gorely T, Davies MJ. The role of physical activity in the management of impaired glucose tolerance: a systematic review; *Diabetologia*.2007; 50:1116–1126.
- 45) Jeon CY, Peter LR, Hu FB, VanDam RM. Physical Activity of Moderate Intensity and Risk of Type 2 Diabetes; *Diabetes Care*;2007;30:3,744-752.
- 46) Nicole AS, Emily BT, Kevan CH. Natural History of *B-cell* function in Type 1 Diabetes. *Diabetes*. 2005;54: pp S32-S38.
- 47) American Diabetes Association. Report of the Expert committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care*.1997;20: 118-197.
- 48) Ingrid M L, Massimo P, Silva AA. Evidence of Heterogeneous Pathogenesis of Insulin Treated Diabetes in Blacks and White children . *Diab Care*. 2003; 26:10. 2876-2881.
- 49) Aldo AR, John PM, Eugene SH, Speculation of Aetiology of Diabetes Mellitus . *Diabetes*.1998;37: 257-261.
- 50) Andrea KS, George SE Genetic Similarities between Latent Autoimmune Diabetes and Type 1 and Type 2 Diabetes. *Diabetes*. 2008;57 : 1160-1162.
- 51) Jennifer MB . Type 1 Diabetes , Associated Auto immunity. Natural History, Genetic Association and Screening. *J clin. Endo metab*. 2006;91(4).1210-1217.
- 52) Zachary T. Bloom G ; Type 2 Diabetes Mellitus: its prevalence, causes, and Treatment. *Diabetes Care*.1998; 21:5 860-865.

- 53) Guillermo E U, Dawn S, EKitabchi AE. Narrative Review: Ketosis-Prone Type 2 Diabetes Mellitus; *Ann Intern Med.* 2006;144:350-357.
- 54) Jayapaul M. K, Walker M: Mechanisms contributing to the development of type 2 diabetes; *British Journal of Diabetes & Vascular Disease* 2004 4: 227
- 55) Bu FH, Manson JE, Stampfer MJ, Colditz G; Diet, Life style and the risk of Type 2 Diabetes Mellitus in women. *NEJM.* 2001;345: No. 11, 790-797.
- 56) Abubakari AR, Bhopal RS. Systematic review on the prevalence of diabetes, overweight/obesity and physical inactivity in Ghanaians and Nigerians. *Public Health* 2008;122 : 173–182.
- 57) UK Prospective Diabetes Study 16. Overview of six years' therapy of type 2 diabetes – a progressive disease. *Diabetes* 1995;44:1249-58
- 58) Laukkanen O. , Lindstro J. , Eriksson J. et al. Polymorphisms in the *SLC2A2* (GLUT2) Gene Are Associated With the Conversion From Impaired GlucoseTolerance to Type 2 Diabetes. *DIABETES*, JULY 2005,VOL. 54.
- 59) Osei K, Rhinesmith S, Gaillard T, Schuster D. Impaired Insulin Sensitivity, Insulin Secretion, and Glucose Effectiveness Predict Future Development of ImpairedGlucose Tolerance and Type 2 Diabetes in Pre-Diabetic African Americans. *Diabetes Care* 2004;27:1439–1446,

- 60) Weyer C, Tatranni P. A, Bogardus C, Pratley R . E, Insulin Resistance and Insulin Secretory Dysfunction Are Independent Predictors of Worsening of Glucose Tolerance During Each Stage of Type 2 Diabetes Development *Diabetes Care* 2000;24:89–94,
- 61) Gerich J.E. Contributions of Insulin-Resistance and Insulin-Secretory Defects to the Pathogenesis of Type 2 Diabetes Mellitus. *Mayo Clin Proc.* 2003;78:447-456
- 62) D K McCulloch. Screening for diabetes mellitus. www.uptodate.com . 2009, June 10.
- 63) Marie-Dominique Beaulieu. *Screening for Diabetes Mellitus in the Non-Pregnant Adult; materials prepared for the U.S. Preventive Services Task Force*
- 64) J. Woo , R. Swaminathan , C. Cockram , C. E Pang , Y.T. Mak et al. The prevalence of diabetes mellitus and an assessment of methods of detection among a community of elderly Chinese in Hong Kong. *Diabetologia* (1987) 30:863-868.
- 65) Position Statement. Screening for Diabetes; American diabetes association: *Diabetes Care*, 2002 Vol. 25, Supplement 1.
- 66) Federal Republic of Nigeria official Gazette, legal notice on publication of details of breakdown of the National and state provisional Totals census. 2007, 15th may.
- 67) Singha P. introductory text on Biostatistics; 2005: 3rd edit pp 205.
- 68) D. Rose. Official Social Classifications in the UK, Social Research Update; July 1995 Issue 9 (accessed November 2009).
- 69) Gezawa I.D . Normative anthropometric values and glucose intolerance among adults in Maiduguri north eastern Nigeria, Dissertation submitted for award of part 2

fellowship of the National postgraduate medical college of Nigeria. 2009, November.

70) Sabir A.A. Glucose tolerance among rural and urban Fulani of northern Nigeria. Dissertation submitted for award of part 2 fellowship of the National postgraduate medical college of Nigeria 2008.

71) Nwafor A, Owhoji A. Prevalence Of Diabetes Mellitus Among Nigerians In Port Harcourt

Correlates With Socio-Economic Status; *J. Appl. Sci. Environ. Mgt.* 2001 vol. 5 (1) 75-77.

72) Bakari A. G. and Onyemelukwe G. C. Glucose intolerance among apparently healthy Hausa-Fulani. *Annals of African Medicine Vol. 3, No. 1; 2004: 32 – 34*

73) Olatunbosun S.T., Ojo P.O., Fineberg N.S., Bella A.F; Impaired glucose tolerance in a group of urban adults I Nigeria. *J.of nat med asso.* Vol. 90, 5.

74) Rao S. S, Disreali P, and Mcgreggo T. Impaired Glucose Tolerance and Impaired Fasting

Glucose ; *American family physician.* April 2004:vol.69;8.

75) Öztürk Y, Mualla A, Fahrettin K, Osman G ,Fevziye Ç, Osman C, Mücahit E.

Prevalence of Diabetes Mellitus and Affected Factors in the District of Kayseri

Health Group Area: *Turk J Med Sci* ; 2000 30 , 181–185

76) Kadiri S and Onwubere BJC. Guidelines for the management of Hypertension in

Nigeria. Recommendation for health care providers prepared by the Nigeria Hypertension society and adopted at the consensus meeting held 28th April 2000 during the 8th Annual and scientific meeting of the society in Lagos and revised by the guidelines committee. 2005, pg 1-4 printed.

77) World Health Organization Expert Committee: Physical status; the use and interpretation of Anthropometry. Report of a WHO expert committee. Technical report series 845. 1995 *WHO Geneva*.

78) World Health Organization. The world Health report 2002. Reducing risk, promoting Healthy life. Geneva Switzerland 2002 .

79) Luke A , Durazo-Arvizu R, Rotimi C, Prewitt T E., et al Relation between Body Mass

Index and Body Fat in Black Population Samples from Nigeria, Jamaica, and the United

States ; Am J Epid 1997; 145, No. 7.

80) Aviles-Santa L, Karin ST, Adams-Huet B, Raskin P; Anthropometric features and cardiovascular risk in young Latin Americans with type 2 diabetes mellitus. *J Diab and Its Complications*. 2006; 20: 69–74.

81) Farrance I. Clin Biochem Reviews. Enzymatic colourimetric test methods 1987; 8: pg 55-68.

82) Glucose tolerance test. www.wikipedia. Wikipedia Foundation Int. the free encyclopedia.

- 83) <http://www.who.int/chp/steps/manual/en/index.html> accessed March, 2009
- 84) Motala AA. Diabetes trends in Africa ; *Diabetes Metab Res Rev.*2002; 18: S14–S20.
- 85)Aksu H, Pala K, Huseyin A. Prevalence and associated risk factors of type 2 diabetes mellitus inNilufer District, Bursa, Turkey : *Int J Diabetes & Metabolism* 2006 14: 98102 .

APPENDIX 1

PRE-TESTING OF THE QUESTIONNAIRE AND METHODOLOGY

The questionnaire designed for the study was pre-tested at kundila to assess the acceptability of the questions therein and participant's response based on their understanding. This enabled adjustment and corrections of the designed questionnaire as well as enhanced validity. The questionnaire was translated into Hausa language since majority of the participants were Hausas.

The blood samples of the participants were taken to the chemical pathology laboratory of Aminu Kano Teaching Hospital located in the same Tarauni Local Government for estimation of casual plasma glucose. Participants found to have Diabetic range of plasma glucose as well as those with glucose dysregulation (levels above normal but not high enough for the diagnosis of DM) were counseled for further investigation and management. This category of participants had their serum insulin level assayed as well as had an OGTT, fasting plasma glucose and 2 hr post glucose load were assayed at AKTH chemical pathology laboratory

APPENDIX 2

BLOOD PRESSURE MEASUREMENT

Blood pressure (BP) was measured with the participants seated for at least five minutes in a calm and quiet place with the arm supported at the level of the heart using a standard Sphygmomanometer (ACCOSON^R) of appropriate size of cuff, both arms were measured at first encounter, and arm that gave higher reading was used subsequently. The cuff was rapidly inflated to determine the pulse obliteration pressure, then quickly deflated, then re-inflated to the same point and slowly deflated to measure the BP. The first appearance of repeated sounds and the disappearance of the sounds correspond to the systolic and diastolic points respectively. The readings were recorded to the nearest 2mmHg and two or more readings separated by interval of at least 2 minutes were averaged and were the first two readings differed by more than 10mmHg additional readings was obtained ⁷⁶.

APPENDIX 3

ANTHROPOMETRIC

MEASUREMENTS

Anthropometry in physical anthropology refers to the measurement of living human individuals for understanding human physical variation. The anthropometric measurements suitable for epidemiological studies as well as for routine clinical assessment of subjects include weight, height, body mass index, waist circumference; hip circumference and waist to hip ratio.¹ Other measurements frequently employed include waist to height ratio, the mid-upper arm circumference and skin fold thickness taken from different sites⁷⁷.

The anthropometric indices are useful not only for the detection of overweight and obesity, but also for the assessment of under nutrition. Obesity has been shown to be associated with an increased risk for chronic non-communicable diseases (NCDs) like diabetes mellitus (DM), hypertension and ischaemic heart disease (IHD)⁷⁷.

Measurement of Height

Standing height was measured without shoes or head gear to the nearest 0.1 cm at all sites using stadiometre with the participant looking straight in a coronas plane⁷⁸.

Measurement of weight

Weights was measured to the nearest 0.1 kg using a calibrated manual weighing scale, with participants wearing light clothes. BMI was calculated as weight (kg)/height (m)² for each participant⁷⁸.

Measurement of hip and waist circumferences

The waist circumference (W. Circ) was measured with inelastic tape as the horizontal level at the mid point between the iliac crest and the lower costal margin with minimal clothes on to the nearest 0.1 cm^{79,80}.

Hip circumference was recorded as the horizontal level of maximum circumference around the buttocks to the nearest 0.5cm⁷⁷. Waist-Hip ratio was calculated as Waist circumference (cm)/ Hip circumference (cm)^{79,80}.

APPENDIX 4

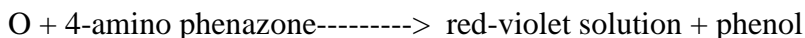
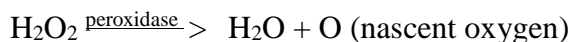
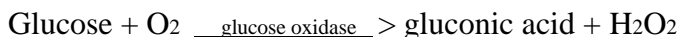
BLOOD SAMPLE TAKING AND LABORATORY PROCEDURES

Blood samples Taking

Blood samples five milliliter (5mls) was drawn from a forearm vein with the participant seated and relaxed. The samples were put into fluoride oxalate (glycolytic inhibitor) containers and were kept on ice packed cool box and transported within 3 hours of collection to the chemical pathology laboratory of AKTH (about 2-3.5 km from survey sites) for plasma glucose estimation.

Plasma Glucose estimation⁸¹

Plasma glucose was estimated, through the glucose-oxidase method of Trinder, using 4-amino phenazone as Oxygen acceptor. The principle of the test is as follows:



Glucose oxidase promotes the oxidation of glucose to gluconic acid with the production of an equivalent amount of hydrogen peroxide. In the presence of peroxidase, O₂ from H₂O₂ is transferred to an acceptor in this case 4-amino phenazone with the production of a colour complex, the intensity of which is proportional to the concentration of glucose concentration in the plasma sample.

Reagents and Equipments

Reagents:

- Glucose oxidase
- Enzyme/ colour reagent
- Phenol reagent
- Standard glucose solution

Equipments:

- Bench centrifuge
- water bath
- spectrophotometer

Analytical test procedure

The blood sample were centrifuged at 2500 rev/min for 2-3 min and plasma were separated and stored at -20° until analysed. Samples pooled on each survey day were analysed within 3 days. Glucose level estimation was carried out thus: 2.5mls of solution containing glucose oxidase, 4-amino phenazone and peroxidase was put in a test tube and mixed with 50ul of plasma warmed up to room temperature and 2.5mls of phenol reagent. The mixture were then incubated in a water bath at 37°C for 20 minutes. The tubes and the mixture contained therein were allowed to cool down to room temperature. A colour change was observed, the intensity of which depended on the glucose concentration. The absorbance (optical density – OD) was then read off on the spectrophotometer at a wave length of 510nm with that of the standard

glucose solution of known concentration which was similarly incubated with glucose oxidase and phenol.

To calculate glucose concentration, the OD of a standard was used as follows:

Plasma glucose concentration = T. OD/ std OD ×

10mmol/L Where T. OD = optical density of

test sample std OD = optical density

of the standard

Precision of plasma Glucose Assays

For each set of samples, the mean OD of duplicated standard solutions was utilised in calculating the plasma glucose level. Every tenth plasma sample was assayed in duplicate.

Inter-assay and intra-assay coefficients of variation at three levels of plasma glucose concentrations (i.e. low, normal and high) were determined for each 10 consecutive assays run.

The coefficient of variation were determine using the formula:

$$CV = SD/Mean \times 100$$

Where CV= coefficient of variation

SD= standard Deviation

Mean = average mean of plasma glucose concentration

APPENDIX 5

PROTOCOL FOR OGTT⁸²

Protocol for Oral Glucose Tolerance Test

Participant's preparation

The participants were instructed not to restrict carbohydrate intake in the days or weeks before the test. The test was not done during an illness, as results may not reflect the participant's glucose metabolism when healthy. Participants weighing less than 43 kg (94 lb), were given 1.75g of glucose per kilogram body weight to avoid exaggerated glucose response and a subsequent false

positive result.⁸²

The participants were asked to fast for the 8-14 hours (water was allowed) and the OGTT was scheduled to begin in the morning (0700-0800) as glucose tolerance exhibits a diurnal rhythm with a significant decrease in the afternoon. Weight and anthropometric measurement were taken before participants were given glucose. A zero time (baseline) blood sample was drawn at zero hour. A glucose solution was constituted at the standard dose of 1.75g of glucose per kilogram of body weight, to a maximum dose of 75g (adjusted to 82g of glucose D). The estimated amount of anhydrous glucose was dissolved in 300mls of clean water, the participants were asked to drink the glucose solution within 5 minutes. Then blood was drawn at 2 hr post glucose load. For simple diabetes screening, the most important sample is the 2 hour sample⁶², so the baseline and 2 hour samples were the only samples collected⁸².

The Oral Glucose Tolerance Test was interpreted according to WHO and definition of diabetes and other glucose intolerance states.

APPENDIX 6

QUESTIONNAIRE(Adopted from WHO steps for the study of non communicable diseases and modified)⁸⁴

Serial No.....

Dear respondent,

This questionnaire is part of a study/research on the prevalence and correlates of Diabetes Mellitus and glucose intolerance among adults in Kano metropolis. It will assist in providing information about Diabetes Mellitus in our environment for further health care planning and management. Kindly assist in providing the information required. The information provided in this questionnaire is for research purpose only, and will be treated confidentially.

I. PERSONAL DATA

- a) Age (years) b) Sex M[] F[]
c) Occupation..... d) Tribe.....

MEDICAL HISTORY

- a) Do you have Diabetes Yes[] No [] I don't know []
b) Do you have Hypertension Yes [] No [] I don't know []
c) Do you have any other disease Yes [] No [] I don't know [] If yes specify:
.....
.....
.....

SYMPTOMS/COMPLICATIONS

- a) Do you pass urine excessively Yes [] No []
- b) Do you take water excessively Yes [] No []
- c) Have you noticed that you are losing weight Yes [] No []
- d) Do you have Eye problem Yes [] No []
- e) Do you have abnormal feelings in your feet or hands Yes [] No []
- f) Have you had any of the following?
Stroke Yes [] No [] I don't know []
Acute chest pain requiring attention Yes [] No []
- g) Have you had a difficult to heal wounds Yes [] No []
- h) Do you have long standing leg ulcer(s) Yes [] No []
- i) Have you suffered from
i recurrent boils Yes [] No [] ii Athletic
foot Yes [] No [] iii Recurrent vaginal itch
and discharge Yes [] No []
- j) Do you have problem getting erection Yes [] No []

RISK OF DIABETES MELLITUS

- a) Family history of diabetes. Is any of the following a diabetic? Father [] Mother
[] Siblings [] others specify.....
- b) Family history of hypertension. Is any of the following hypertensive? Father []
Mother [] Siblings [] others specify.....

c) Do you have history of delivery of large babies ($\geq 4\text{kg}$) in the past Yes [] No []

d) Is there any history of recurrent miscarriages yes [] No []

e) Is there any history of still birth in the past Yes [] No []

f) Is there any history of caesarean section in past Yes [] No []

g) Drug history

Are you on any routine medicine(s) Yes [] No []

if Yes list i).....

ii).....

iii).....

h) Do you smoke Cigarette Yes[] No[] if yes duration of smoking.....pack
year(s).....

i) Do you drink Alcohol Yes [] No []

if yes duration Quantity per week.....

j) Do you exercise

If yes. i)

Type.....

..... ii)

Duration.....

..... iii)

Frequency.....

.....

2 physical examination

Weight in kg..... Height in meter.....BMI..... kg/m^2

Waist circumference in cm.....Hip circumference in cm W/H ratio.....

B/p.....mm/hg

Urinalysis... glucose.....
 Protein.....
Ketones.....
 Nitrite.....
RBS.....
OGTT zero min 2hr post glucose.....

Serum insulin level.....

Thank you for your cooperation.

APPENDIX 7

CONSENT FORM

Dear Participant,

I am Dr Abubakar L. Yusuf from Department of Medicine, Aminu Kano Teaching Hospital, conducting a study in this ward with the aim of determining the prevalence and correlates of DIABETES MELLITUS among adult in Kano Metropolis.

The Study Title is:

PREVALENCE AND CORRELATES OF DIABETES MELLITUS AND GLUCOSE INTOLERANCE AMONG ADULTS IN KANO METROPOLIS.

I am requesting for your consent to take part in this study in providing the required information. This study if conducted successfully will give us the current prevalence of the disease in this community and enable to advice the Government on how to plan for the management of the disease more effectively. However, early detection of the disease will prevent the development of its debilitating complications.

Note that:

- 1- Your participation is voluntary
- 2- You can withdraw from the study when ever you want to
- 3- The result of the tests will be explained to you
- 4- If you are found to have the disease or abnormal result you will be referred to any health facility of your choice after the study.
- 5- Declining to participate will not be used against you incase you need care in future

If you consent to partake in this project please append your signature below

Signature of the participant.....

Date.....

Signature of the Doctor/ Researcher.....

Date.....

Thank you for your cooperation

