## KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY KUMASI

## **COLLEGE OF SCIENCE**

## DEPARTMENT OF BIOCHEMISTRY AND BIOTECHNOLOGY

## THE EFFECT OF NUTRITIONAL STATUS ON THE PREVALENCE OF

## CARDIOVASCULAR DISEASES AMONG DIABETIC PATIENTS

THIS DISSERTATION IS PRESENTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS OF MPhil. DEGREE IN HUMAN NUTRITION AND DIETETICS

BY

**TUVOR ALICE** 

**BSc.** (HONS)

OCTOBER, 2016

WJSANE

CARSH

### DECLARATION

I declare that I have wholly undertaken the study reported herein under the supervision of Dr. Patricia Brown and Dr. Bernard Nkum and that except portions where references have been duly cited, this dissertation is the outcome of my research.

| Tuvor Alice (20285935) |           |       |
|------------------------|-----------|-------|
| (Student Name & ID)    | Signature | Date  |
|                        |           |       |
|                        |           |       |
| Certified By           |           |       |
| Dr. Patricia Brown     |           |       |
| (Supervisor)           | Signature | Date  |
|                        | and 1     |       |
| (32)                   |           | TES . |
| Certified By           | I DI F    | 17    |
| Dr. Bernard Nkum       |           | >     |
| (Co-Supervisor)        | Signature | Date  |
| Rules                  |           |       |
|                        |           |       |
| Certified By           |           |       |
| Dr. Hilary D. Zakpa    |           |       |
| (Head of Department)   | Signature | Date  |
| A.P.                   | Sal       | 2°    |
| W                      |           |       |
| WJSI                   | ANE NO    |       |

## ABSTRACT

Diabetes mellitus is associated with increased incidence of cardiovascular disease (CVD). Poor nutritional status such as under- and over-nutrition accelerates the development of complications and reduces the longevity of life of diabetes patients. The study aimed to assess nutritional status, using anthropometric, biochemical, dietary indicators as well as unhealthy lifestyle factors and finding their association with cardiovascular diseases; mainly stroke, hypertension and heart failure. A cross sectional descriptive study was conducted among 145 diabetes patients selected from three hospitals in the Ashanti Region of Ghana. Nutritional status and lifestyle assessments were done using anthropometric, biochemical and dietary indicators of participants. Out of the participants, 76.6% were females. The mean age of respondents was 55.88  $\pm$  12.75 S.D years. There was a significant association of age with cardiovascular diseases (p<0.05). The prevalence rates of cardiovascular diseases; hypertension, stroke and heart failure among the diabetic patients were 66.2%, 8.28% and 3.45%, respectively. The prevalence of overweight and obesity, assessed by BMI were 42.8%and 22.8%, respectively but there was no significant association of BMI with cardiovascular diseases (p>0.05). The prevalence of abdominal obesity, measured by waist circumference and waist to hip ratio were 61.38% and 65.5%, respectively and were significantly associated with cardiovascular diseases (p<0.05). Dyslipidemia among diabetes patients showed prevalence of hypercholesterolemia (47.58%), hypertriglyceridemia (55.9%), low HDL-C (35.2%) and high LDL-C levels (36.8%). Lipid profile showed that total cholesterol was not significantly associated with cardiovascular diseases, but TG, HDL-C and LDL-C levels were significantly associated with cardiovascular diseases. The study showed that 18.6% had high creatinine levels, but there was no significant association of creatinine levels with cardiovascular diseases (p>0.05). The study showed inadequate intake of fruits and vegetables and there was no significant association of fruit and vegetable intake with cardiovascular diseases (p>0.05). Majority of the respondents, 56.6% reported low salt intake, whilst 9.0% reported high salt intake. Salt intake showed significant association with cardiovascular diseases (p<0.05). The patients lifestyles showed low levels of physical activity, as 61.4% did not exercise, 91.0% diabetics had never smoked, 9.0% were ex-smokers, 33.8% were ex-drinkers, whilst 11.7% were current alcohol drinkers. The lack of exercise and smoking status had significant association with cardiovascular diseases (p<0.05) but alcohol consumption had no significant association with cardiovascular diseases (p>0.05). The study showed independent risk factors significantly associated with cardiovascular diseases incidence among diabetes patients included family history of hypertension with (odds ratio of 6.8), exercise (0.1), salt intake (0.1) and HDL-C level (0.2) with p-values < 0.05. In conclusion, the study has shown that family history of hypertension, moderate exercise, low salt intake and HDL-C level are significantly associated with cardiovascular diseases incidence.

| AIDS    | ABBREVIATIONS<br>Acquired Immune Deficiency Syndrome     |
|---------|--|
| ACCORD  | Action to Control Cardiovascular Risk in Diabetes        |
| ADA     | American Diabetes Association                            |
| ADVANCE | Action in Diabetes and Vascular Disease                  |
| AGE(s)  | Advanced glycated end product(s)                         |
| ANOVA   | Analysis of Variance                                     |
| BMI     | Body Mass Index  |
| BP      | Blood Pressure   |
| CVD     | Cardiovascular disease                                   |
| CVDs    | Cardiovascular diseases                                  |
| CHD     | Coronary Heart Disease                                   |
| CE      | Cholesterol esterase                                     |
| СО      | Cholesterol oxidase                                      |
| DASH    | Dietary Approach to Stop Hypertension                    |
| DCCT    | Diabetes Control and Complications Trial                 |
| DPP     | Diabetes Prevention Program                              |
| DM      | Diabetes Mellitus  |
| DPPOS   | Diabetes Prevention Program Outcomes Study               |
| DSME    | Diabetes Self-Management Education                       |
| DSMEP   | Diabetes Self-Management Education Program               |
| EDIC    | Epidemiology of Diabetes Interventions and Complications |
| FPG     | Fasting Plasma Glucose                                   |
| GDM     | Gestational diabetes                                     |
| HbA1c   | Hemoglobin A1c   |
| HDL-C   | High-density lipoprotein-Cholesterol                     |

| IDF          | International Diabetes Federation                                  |
|--------------|--|
| LDL-C        | Low-Density Lipoproteins-Cholesterol                               |
| NCEP         | National Cholesterol Education Program                             |
| NCHS         | National Center for Health Statistics                              |
| NHLBI        | National Heart, Lung, and Blood Institute                          |
| NCEP ATP III | National Cholesterol Education Programme Adult Treatment Panel III |
| OR           | Odds Ratio   |
| OPD          | Out-Patient Department   |
| PA           | Physical Activity  |
| POD          | Peroxidase   |
| SSA          | Sub-Saharan Africa   |
| T1DM         | Type 1 diabetes mellitus   |
| T2DM         | Type 2 diabetes mellitus   |
| TC           | Total cholesterol  |
| TG           | Triglycerides  |
| UKPDS        | United Kingdom Prospective Diabetes Study                          |
| VEGF         | Vascular endothelial growth factor                                 |
| VLDL         | Very low-density lipoprotein                                       |
| WHO          | World Health Organization  |
| WC           | Waist Circumference  |
| WHR          | Waist-to-Hip-Ratio   |
|              | WJ SANE NO   |

v

#### ACKNOWLEDGEMENT

Much gratitude is given to God Almighty from whom all good things come, for His grace and mercy towards me and for seeing me through the execution of this project work. Without Him this project will not have been successful. I am forever grateful to my mother, Ms Roselyn Gordon, for providing me with the financial support and all the necessary resources needed to complete my degree. I am always grateful to her for making this work possible.

I am highly indebted to my supervisors, Dr. Patricia Brown and Dr. Bernard Nkum, for their directions and valuable contributions to this work. My appreciation goes to Madam Gloria Ankar Brewuo, for her support and guidance throughout.

I would also like to express my profound gratitude to lecturers in the Department of Food Science and Technology and Department of Biochemistry and Biotechnology of the Kwame Nkrumah University of Science and Technology, for their views and comments. I must admit that these interactions have been very useful and I would like to express my deepest appreciation to all those who contributed to this success.

I also wish to acknowledge with profound gratitude the support I received from the laboratory staff of the St. Micheal's Hospital, Kumasi South Hospital and Aninwah Medical Centre, for their support and cooperation during the data collection period of this study, God richly bless ARKSAP J W J SAME them.

BADH

vi

# TABLE OF CONTENTS

| DECLARATIONi   |   |
|--|---|
| ABSTRACTii   |   |
| ABBREVIATIONSiv  | 7 |
| ACKNOWLEDGEMENTv   | i |
| TABLE OF CONTENTSvi                                      | i |
| LIST OF TABLESx  | i |
| L <mark>IST OF FIGURESxi</mark>                          | i |
| CHAPTER ONE  |   |
| 1.1 Introduction   |   |
| 1.2 Aim of the Study                                     | ŀ |
| 1.3 Study Hypothesis                                     | ŀ |
| 1.4 Problem Statement                                    | 5 |
| 1.5 Justification  | 5 |
| CHAPTER TWO  |   |
| LITERATURE REVIEW  | 7 |
| 2.0 Introduction   |   |
| 2.1 Diabetes Mellitus                                    | 7 |
| 2.2 Types of Diabetes Mellitus                           | 7 |
| 2.3 Epidemiology of Diabetes and Cardiovascular Diseases | 3 |
| 2.4 Complications of Diabetes Mellitus                   | ) |
| 2.5 Diabetic Retinopathy                                 | ) |

| 2.6 Diabetes Nephropathy   | 11 |
|--|----|
| 2.7 Diabetic Neuropathy  | 12 |
| 2.8 Macrovascular Complications of Diabetes                                | 12 |
| 2.9 Development of Cardiovascular Diseases in Diabetes                     | 13 |
| 2.10 Nutritional status and Health   | 15 |
| 2.11 Effect of Nutritional Status Indicators on Cardiovascular Diseases    | 16 |
| 2.11.1 Anthropometric Indicators (Generalized Obesity and Central Obesity) | 16 |
| 2.11.2 Effect of BMI on Cardiovascular Diseases                            | 16 |
| 2.11.3 Waist Circumference and Cardiovascular diseases                     | 18 |
| 2.12 Dietary Factors   | 21 |
| 2.12.1 Fruit and Vegetable Intake  | 22 |
| 2.12.2 Sodium Intake   | 24 |
| 2.13 Biochemical Risk Factors  | 25 |
| 2.13.1 Dyslipidemia  | 25 |
| 2.14 Unhealthy Lifestyle and Cardiovascular Disease Risk in Diabetes       | 26 |
| 2.14.1 Smoking   |    |
| 2.14.2 Alcohol Intake  | 27 |
| 2.14.3 Physical Inactivity   | 29 |
| CHAPTER THREE  |    |
| MATERIALS AND METHODS  |    |
| 3.1 Study Design   | 31 |
| 3.2 Study Site   | 31 |
| 3.3 Study Population and Sample Size                                       | 31 |
| 3.4 Inclusion and Exclusion Criteria                                       | 32 |
| 3.4.1 Inclusion Criteria for Control Participants                          | 32 |
| 3.4.2 Inclusion Criteria for Case Participants                             | 32 |
| 3.5 Exclusion Criteria for Participants                                    | 32 |
| 3.6 Sampling Procedure   | 33 |

| 3.7 Questionnaire-Based Data Collection   | 33   |
|---|--|
| 3.8 Pre-testing of Questionnaire  | 33   |
| 3.9 Anthropometric Variables  | 33   |
| 3.9.1 Body Mass Index   | 34   |
| 3.9.2 Weight Measurement  |  |
| 3.9.3 Height Measurement  | 34   |
| 3.9.4 Waist and Hip Circumference   | 34   |
| 3.10 Dietary Intake   | 35   |
| 3.11 Cardiovascular Disease Diagnosis   | 35   |
| 3.12 Blood Pressure Measurement   | 35   |
| 3.13 Biochemical Indicators Measurements  | 36   |
| 3.13.1 Blood Collection and Handling  | 36   |
| 3.13.2 Fasting Blood Glucose Measurement  | 36   |
| 3.13.3 Lipid Profile: (Triglycerides, HDL, LDL and total cholesterol)   | 37   |
|   |  |
| 3.13.4 Serum Creatinine   |  |
| 3.14 Operational Definition of Terms  | 38   |
|   | 38   |
| 3.14 Operational Definition of Terms  | 38<br>39   |
| <ul> <li>3.14 Operational Definition of Terms</li> <li>3.15 Ethical Considerations</li> <li>3.16 Statistical Analysis</li> <li>CHAPTER FOUR</li> </ul>  | 38<br>39<br>39<br><b> 41</b>                                   |
| <ul> <li>3.14 Operational Definition of Terms</li></ul>   | 38<br>39<br>39<br><b> 41</b>                                   |
| <ul> <li>3.14 Operational Definition of Terms</li> <li>3.15 Ethical Considerations</li> <li>3.16 Statistical Analysis</li> <li>CHAPTER FOUR</li> <li>RESULTS</li> <li>4.1 Demographic Characteristics of Respondents</li> </ul>   | 38<br>39<br>39<br><b> 41</b><br><b>41</b><br>41                |
| <ul> <li>3.14 Operational Definition of Terms</li> <li>3.15 Ethical Considerations</li> <li>3.16 Statistical Analysis</li> <li>CHAPTER FOUR</li> </ul>  | 38<br>39<br>39<br><b> 41</b><br><b>41</b><br>41                |
| <ul> <li>3.14 Operational Definition of Terms</li> <li>3.15 Ethical Considerations</li> <li>3.16 Statistical Analysis</li> <li>CHAPTER FOUR</li> <li>RESULTS</li> <li>4.1 Demographic Characteristics of Respondents</li> </ul>   | 38<br>39<br>39<br>41<br>41<br>42                               |
| <ul> <li>3.14 Operational Definition of Terms</li> <li>3.15 Ethical Considerations</li> <li>3.16 Statistical Analysis</li> <li>CHAPTER FOUR</li> <li>CHAPTER FOUR</li> <li>4.1 Demographic Characteristics of Respondents</li> <li>4.2 Cardiovascular Diseases</li> </ul>   | 38<br>39<br>39<br>41<br>41<br>42<br>42                         |
| <ul> <li>3.14 Operational Definition of Terms</li> <li>3.15 Ethical Considerations</li> <li>3.16 Statistical Analysis</li> <li>CHAPTER FOUR</li> <li>CHAPTER FOUR</li> <li>4.1 Demographic Characteristics of Respondents</li> <li>4.2 Cardiovascular Diseases</li> <li>4.3 Anthropometric and Blood Pressure Data of Respondents</li> </ul>  | 38<br>39<br>39<br>41<br>41<br>42<br>42<br>42<br>43             |
| <ul> <li>3.14 Operational Definition of Terms</li> <li>3.15 Ethical Considerations</li> <li>3.16 Statistical Analysis</li> <li>CHAPTER FOUR</li> <li>CHAPTER FOUR</li> <li>4.1 Demographic Characteristics of Respondents</li> <li>4.2 Cardiovascular Diseases</li> <li>4.3 Anthropometric and Blood Pressure Data of Respondents</li> <li>4.4 Blood Pressure of Respondents</li> </ul>   | 38<br>39<br>39<br>41<br>41<br>42<br>42<br>42<br>43<br>45       |
| <ul> <li>3.14 Operational Definition of Terms</li> <li>3.15 Ethical Considerations</li> <li>3.16 Statistical Analysis</li> <li>CHAPTER FOUR</li> <li>CHAPTER FOUR</li> <li>4.1 Demographic Characteristics of Respondents</li> <li>4.2 Cardiovascular Diseases</li> <li>4.3 Anthropometric and Blood Pressure Data of Respondents</li> <li>4.4 Blood Pressure of Respondents</li> <li>4.5 Biochemical Data of Respondents</li> </ul>  | 38<br>39<br>39<br>41<br>41<br>42<br>42<br>42<br>43<br>45       |
| <ul> <li>3.14 Operational Definition of Terms</li> <li>3.15 Ethical Considerations</li> <li>3.16 Statistical Analysis</li> <li>CHAPTER FOUR</li> <li>CHAPTER FOUR</li> <li>4.1 Demographic Characteristics of Respondents</li> <li>4.2 Cardiovascular Diseases</li> <li>4.3 Anthropometric and Blood Pressure Data of Respondents</li> <li>4.4 Blood Pressure of Respondents</li> <li>4.5 Biochemical Data of Respondents</li> <li>4.5.1 Fasting Blood Glucose of Participants</li> </ul> | 38<br>39<br>39<br>41<br>41<br>42<br>42<br>42<br>43<br>45<br>45 |

| 4.6.1 Oral Medication  | 47              |
|--|-----------------|
| 4.7 Family and Medical History   |                 |
| 4.8 Dietary Habits of Respondents                                      |                 |
| 4.9 Participants' Physical Activity Levels and Lifestyle               |                 |
| 4.10 Association between CVD and Some Selected Risk Factors a Patients |                 |
| CHAPTER FIVE   | 57              |
| DISCUSSION   |                 |
| 5.0 Introduction   | 57              |
| 5.1 Baseline Characteristics of Participants                           |                 |
| 5.2 Anthropometric Data of Respondents                                 | 59              |
| 5.3 Dyslipidemia   | 62              |
| 5.4 Creatinine   | 64              |
| 5.5 Fasting Blood Glucose Levels                                       | <mark>64</mark> |
| 5.6 Prevalence of Cardiovascular Disease among DM Patients             |                 |
| 5.7 Dietary Habits of Respondents                                      | 67              |
| 5.8 Fruit and Vegetable Intake   |                 |
| 5.9 Lifestyle of Respondents   | 70              |
| 5.10 Physical Activity Levels  | 72              |
| 5.11 Alcohol Consumption   | 74              |
| 5.12 Blood Pressure  |                 |
| 5.13 Diabetes Medications  |                 |
| 5.14 Herbal Medications Usage among Diabetic Respondents               |                 |
| CHAPTER SIX  | 77              |
| CONCLUSION AND RECOMMENDATIONS   |                 |
| 6.1 Conclusion   | 77              |
| 6.2 Recommendations  | 78              |
| 6.3 Limitations and Further Research                                   | 79              |

| REFERENCES |  |
|------------|--|
| APPENDIX   |  |

## LIST OF TABLES

| Table 4.1 Demographic Characteristics of All Diabetic Respondents                         | . 44 |
|---|------|
| Table 4.2 Comparison of Demographic Characteristics of Respondents with and without       |      |
| CVDs  | . 45 |
| Table 4.3 Differences in BMI of Male and Female Diabetic Respondents                      | . 45 |
| Table 4.4 Prevalence of Cardiovascular Diseases among Diabetics                           | . 46 |
| Table 4.5 Gender and Age of Respondents with and without CVDs                             | . 46 |
| Table 4.6 Anthropometric Data of Diabetic Respondents with and without CVDs               | . 47 |
| Table 4.7 Comparison of Anthropometric and Blood Pressure Data among Diabetic             |      |
| Respondents with and without CVDs   | . 48 |
| Table 4.8 Biochemical Data of Respondents with and without CVD                            | . 48 |
| Table 4.9 Biochemical Data of Respondents with and without CVDs                           | . 49 |
| Table 4.10 Oral Medication and Insulin Use  | . 49 |
| Table 4.11 Family Medical History of Cardiovascular Diseases among All Respondents        | . 50 |
| Table 4.12 Family Medical History among Respondents with and without CVDs                 | . 50 |
| Table 4.13 Duration of Diseases among Respondents with and without CVDs                   | . 51 |
| Table 4.14 Dietary Habits of All Diabetic Respondents                                     | . 52 |
| Table 4.15 Dietary Habits of Diabetic Respondents with and without CVDs                   | . 54 |
| Table 4.16 Lifestyle and Physical Activity Levels of All Diabetic Participants'           | . 54 |
| Table 4.17 Diabetic Participants' Lifestyle and Physical Activity of Respondents with and |      |
| without CVDs  | . 55 |
| Table 4.18 Differences in Smoking Status of Male and Female Diabetic Respondents          | . 55 |
| Table 4.19 Selected Risk Factors associated with CVDs among Diabetic Patients             | . 57 |
| Table 4.20 Significant Risk Factors Independently Associated with CVD Incidence among     |      |
| Diabetic Respondents with and without CVDs in Logistic Model                              | . 57 |
| JANE  |      |

## LIST OF FIGURES

| Figure 1: The Mechanism of Atherosclerosis in Diabetes Mellitus           | 15 |
|---|----|
| Figure 2: Type 2 diabetes mellitus and cardiovascular disease development | 15 |



#### **CHAPTER ONE**

#### **1.1 Introduction**

The increased burden of diabetes mellitus and cardiovascular diseases (CVDs) is the principal cause of death globally (WHO, 2011). Currently, diabetes and heart diseases affect many people in poor populations more than infectious diseases (WHO, 2008). Records from the International Diabetes Federation show that 4.8 million people died of the disease, and there were 371 million diagnosis in 2012 (International Diabetes Federation, Diabetes Atlas, 2012).

Diabetes was a rare disease in sub-Saharan Africa (SSA), but now 12.1 million adults are affected by the disease in this region, which is anticipated to rise to 23.9 million by 2030 (Sicree, 2009). The prevalence of diabetes in adults worldwide and in Africa, stands at 8.5% and 5.1% respectively (Whiting *et al.*, 2011). In Ghana, diabetes mellitus (DM) was reported in 1976 to be between 0.2 to 0.4% (Owusu, 1976). This prevalence has increased to 6.3% (Amoah *et al.*, 2002), with a prevalence of 3.9% in Greater Accra region (Vuvor *et al.*, 2011).

Diabetes mellitus (DM) is a disorder of metabolism, characterized by hyperglycemia, resulting from defects in insulin secretion, insulin action or both (Abou-Seif and Youssef, 2004). The defects lead to distinctive deficiency of insulin or its adequate function, resulting in disturbances in carbohydrate, lipid and protein metabolism, which manifest as prolonged hyperglycemia (Nair, 2007). There are two main types of diabetes type 1 and type 2. Type 1 diabetes, an insulin-dependent form of diabetes results from T cell-mediated autoimmune destruction of pancreatic beta cells (Kaufman *et al.*, 1993). In type 2 diabetes high blood glucose levels occurs due to the body's inefficient use of insulin and it is the more prevalent. Type 2 diabetes mellitus is characterized by a state of long-standing insulin resistance, compensatory hyperinsulinemia and varying degrees of elevated plasma glucose (Faghilimnai *et al.*, 2006). Apart from the two types of diabetes, there is also gestational diabetes, characterized by hyperglycemia, which is recognized firstly during pregnancy, with similar symptoms as in type 2 diabetes (WHO, 2008) and develops in about 2-5% of all pregnancies (ADA, 2002).

Diabetes mellitus is linked with complications including retinopathy, nephropathy and neuropathy. Elevated blood glucose levels in diabetics result in oxidative stress and endothelial damage which lead to diabetic complications (Giunti *et al.*, 2006). Cardiovascular disease (CVD) is the most common complication of Type 2 DM, with a 2 to 3 times occurrence in diabetics than in non-diabetics (Abraham, 2004). Studies by Bauters *et al.* (2003) and Thiam and co-workers have shown increased incidence of heart failure among diabetes patients. In a study in Nigeria among diabetes patients 58% had heart failure (Ola *et al.*, 2006). Diabetes mellitus plays a significant role in the etiology and development of cardiovascular diseases (Solang *et al.*, 1999).

Diabetes mellitus is characterized by hypermetabolism with metabolic and nutritional consequences (Bitz *et al.*, 2004). The metabolism of diabetics differs from the metabolism of people without it. In diabetes mellitus, metabolism of all the main food nutrients are altered (Nyarko *et al.*, 1997; Ryden *et al.*, 2007). Patients with type 2 diabetes (DMT2) suffer from macronutrient overnutrition, with increased fat stores but those with type 1 diabetes (DMT1) tend to be undernourished, by nutrient spillage through glycosuria (Ryden *et al.*, 2007).Nutrition plays a critical role in health status, as poor nutritional status accelerates development of complications and increases mortality rate of diabetic patients (Mitch, 1998).

Nutritional status can be assessed by several factors, including anthropometric, biochemical and dietary indicators, as well as lifestyle factors. The health and life expectancy of diabetes

patients are greatly affected by their nutritional status (Taubert *et al.*, 2007). In order to prevent the dreadful diabetic complications some diabetic patients control hyperglycemia, by restricting diet which sometimes leads to under-nutrition and health complications (Begum *et al.*, 2004). Alternatively, other patients are reluctant to consume the diet as prescribed by nutritional recommendations, hence become overweight, which is undoubtedly harmful to diabetic patients (Begum *et al.*, 2004).

Studies have clearly demonstrated links between obesity, arising from over-nutrition and many chronic diseases, including cardiovascular disease and diabetes (Taubert *et al.*, 2007). Obesity, hypertension and hyperlipidemia associated with over-nutrition increases the risk of stroke and heart diseases (Madonna *et al.*, 2004). Obesity, predominantly central obesity, is a risk factor for heart diseases, largely through insulin resistance and inflammation-mediated pathways (Madonna *et al.*, 2004). Insulin resistance is concomitant with increased lipolysis from the adipose tissue, resulting in the flux of fatty acids in different organs, including the liver, the heart and muscle (McGavock *et al.*, 2006). The high free fatty acids released causes insulin resistance, associated with a clustering of cardiovascular risk factors, including hypertension, dyslipidemia and abnormal fibrinolysis (Fagot-Campagna *et al.*, 1998). It is known that insulin resistance causes DM and the processes by which this occurs has repercussions on CVD.

Chronic diseases have been linked with poor dietary intake for many years (Martin, 2006). Diabetes greatly increases the risk of heart disease and stroke even with well controlled glycemic levels (Candido *et al.*, 2003). Diabetes patients often develop cardiovascular diseases due to the prevalence of conditions such as high blood pressure (hypertension), dyslipidemia, smoking, obesity and lack of physical activity (Candido *et al.*, 2003).

These conditions above, such as poor diet and unhealthy lifestyle, recognized in diabetics have been related with significant risk factors of heart and chronic diseases (Arthur and John, 2000). Diet and nutrition maintained at optimum levels, prevent development of complications among diabetics. Hence, assessment of nutritional status indicators of diabetic patients is vital, as poor nutrition could contribute to the development cardiovascular diseases. This will offer valuable information for more comprehensive and intensive approach to diabetic patient care.

#### 1.2 Aim of the Study

The study aimed to assess nutritional status as well as unhealthy lifestyle factors among diabetic patients and its association with prevalence of cardiovascular diseases mainly stroke, hypertension and heart failure.

The specific objectives were;

- 1. To assess the anthropometric data (body mass index, waist circumference and waist to hip ratio) of diabetic patients above 18 years with or without cardiovascular disease.
- 2. To determine the prevalence of dyslipidemia among diabetic patients.
- 3. To determine the creatinine levels of diabetic patients.
- 4. To assess the dietary intake of diabetic patients.
- 5. To assess the physical activity level and lifestyle of diabetic patients.
- 6. To determine the prevalence of cardiovascular diseases (hypertension, stroke and heart failure) among diabetic patients.

#### **1.3 Study Hypothesis**

The study hypothesis is that there is an association between nutritional status (anthropometric parameters, biochemical parameters, dietary intake) and other lifestyle factors of diabetics and the prevalence of cardiovascular diseases; namely, hypertension, stroke and heart failure.

SANE

#### **1.4 Problem Statement**

The upsurge in prevalence of diabetes in Ghana, especially among young adults has become a public health concern. In the 1950s, the prevalence of diabetes was estimated at less than 0.5% (Dodu, 1958), but increased to 6.3% in 2000 (Amoah *et al.*, 2002). The fact that many people between the ages of 15-59 years are affected by diabetes poses huge financial problem and threatens the economic stability of the nation (GNA report, 2013). Diabetes is a principal risk factor for cardiovascular disease (CVD). Studies have shown that diabetes increases the risk of CVD complications (Stamler *et al.*, 1993; APCSC, 2003). In a study among patients with heart failure in Ghana, 17% had diabetes (Amoah and Kallen, 2000).

Despite the rising prevalence of heart diseases among diabetic patients, the causative link between nutrition and lifestyle on CVD events is difficult to determine. The exposure to risk factors such as obesity, unhealthy lifestyle practices, hyperinsulinemia, elevated serum triglycerides and elevated total cholesterol among diabetics have been implicated in cardiovascular disease incidence (WHF, 2012). The extent to which these risk factors independently contribute to CVD development among diabetics is uncertain. Risk factors for cardiovascular disease in diabetics remain under-studied in sub-Sahara Africa (WHF, 2012).

Therefore, there is paucity of information on the magnitude of these risk factors among diabetic patients in the region. Hence evidence derived from other populations have been used to inform cardiovascular disease management and prevention among diabetics. This presents a faulty baseline, as differences in cultural, socioeconomic as well as genetic factors are not considered. This could lead to a misdiagnosis of the root cause of the disease and a diversion of efforts on other less important risk factors. Researchers continue to find risk factors associated with

various cardiovascular diseases among diabetics but the key problem that remains unanswered is, 'what risk factors, could cause predisposition of some diabetics to cardiovascular disease complications?

#### **1.5 Justification**

Efforts are required to fill the gaps in knowledge on diabetes and heart diseases in sub-Sahara Africa (SSA). As this will transform the current knowledge regarding interventions into useful strategies that will limit the burden of cardiovascular diseases among diabetes patients in this region. The indiscriminate application of recommendations derived from other countries to SSA populations may be unsuitable (Cobayashi *et al.*, 2010). Research is needed to improve knowledge on CVDs in Africa (Beaglehole *et al.*, 2010). The identification of dietary habits and lifestyle patterns among diabetics play an integral part in the management of the condition. Managing cardiovascular risk factors in diabetics will help reduce the progression of complications (American Diabetes Association, 2007).

For countless individuals with diabetes, the most important part of the treatment plan is determining what to eat. Therefore, from a preventive perspective, it would be useful to examine the association between nutritional status and the prevalence of the cardiovascular diseases among diabetics. This will provide data on the role of diet and lifestyle changes in preventing and controlling morbidity and premature mortality from diabetes and cardiovascular diseases.

#### **CHAPTER TWO**

#### LITERATURE REVIEW

#### **2.0 Introduction**

This chapter reviews the work of other researchers in relation to the study. The chapter explains diabetes mellitus (DM), the types and complications of the disease, focusing on cardiovascular diseases. The development of cardiovascular disease in diabetes patients are also elaborated and relevant research findings on the prevalence of cardiovascular disease among diabetes patients are also reviewed. The chapter reviews research on the effect of nutritional indicators on cardiovascular disease among diabetes patients and the process by which nutritional factors such as anthropometric indicators, dietary indicators, biochemical indicators, and lifestyle factors such as smoking, alcohol consumption and physical activity, influence the incidence of cardiovascular diseases.

#### **2.1 Diabetes Mellitus**

Diabetes mellitus (DM) is no longer a rare disorder in the tropics, as its incidence is increasing in many places (Dodu, 1967). The disease is silent and several patients become aware only when its complications develop (Wee *et al.*, 2002). Diabetes mellitus is a chronic progressive metabolic disease, caused by elevated blood glucose levels in the blood, which may be the consequence of defect in insulin secretion, insulin action or a combination of both (American Diabetes Association, 2006; Hill, 2009). Diabetes is mainly classified as type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM), of which type 2 DM is the more prevalent (Alberti *et al.*, 1998).

#### 2.2 Types of Diabetes Mellitus

Type 1 diabetes represents about 10% of all diabetes cases, affecting approximately 20 million people worldwide (Bierman, 2001). Type 1 diabetes mellitus is a condition characterized by a

lack of insulin production due to autoimmune destruction of pancreatic  $\beta$ -cells (Ammari, 2004). For type 1 diabetes diagnosis; autoantibodies to insulin and autoantibodies to the tyrosine phosphatases are the markers of the immune destruction (Keeling, 2012). At present, lifelong insulin therapy is the only management for this type of diabetes. Type 2 diabetes accounts for 90% of all diabetes cases worldwide and it is the most common (Kappala, 2012). Type 2 diabetes is a heterogeneous condition that results from genetic and environmental factors interaction (Litwak *et al.*, 2013). The main pathophysiological features, responsible for hyperglycemia associated with diabetes are inadequate insulin secretion and decreased insulin sensitivity (Kumar, 2002). Another type of diabetes is gestational diabetes mellitus, which is defined as any degree of glucose intolerance with onset or first recognition during pregnancy (World Health Organization, 2012).

#### 2.3 Epidemiology of Diabetes and Cardiovascular Diseases

Globally in 2013, it was estimated that almost 382 million people suffered from diabetes, giving a prevalence of 8.3%, which increased to 9% in 2014 (Kengne *et al.*, 2005). A further 316 million with impaired glucose tolerance are at risk of the disease, set to reach 471 million by 2035. In sub-Saharan Africa (SSA), the overall DM prevalence is 4%, with estimated prevalence of 1% in rural areas, and ranges from 5% to 7% in urban sub-Saharan Africa (Kengne *et al.*, 2005). In urban Ghana, type 2 DM (DM2) affects at least, 6.3% of adults (Amoah *et al.*, 2002) and is associated with advanced age and obesity as 23% of adults are overweight (Amoah, 2003). In Ghana, currently 6.8% of adult admissions at the Korle-Bu Teaching Hospital are as a result of diabetes (Kengne *et al.*, 2005; Okertchiri, 2013). Risk factors including physical inactivity, dietary intake, ageing and obesity have contributed to high incidence of diabetes in Ghana (Amoah, 2003). Heart diseases deaths constitute 8.8% of all deaths and 3.5% of all disability-adjusted life years (DALYs) in SSA (Krishnamurthi *et al.*, 2013).

Cardiovascular disease had been considered uncommon in SSA (Ntsekhe and Damasceno, 2013). However, both population- and hospital-based studies now show evidence for an increasing problem of cardiovascular disease in SSA, with diabetes mellitus as a main contributor (Ntsekhe and Damasceno, 2013). Several epidemiological studies have shown that CVD incidence in people with diabetes is two to four times greater than non-diabetics (Stamler *et al.*, 1993; APCSC, 2003). Diabetes mellitus plays a vital role in the pathogenesis of heart failure (Solang *et al.*, 1999). In a group of patients with heart failure in Nigeria, the fraction of those with diabetes was found to be 58% (Ola *et al.*, 2006). A study of patients with heart failure in Ghana, showed that 17% of those with coronary artery disease had diabetes (Amoah and Kallen, 2000).

#### 2.4 Complications of Diabetes Mellitus

The source of concern with diabetes is the development of complications arising from the injurious effect of chronic hyperglycemia and its metabolic abnormalities (Fong *et al.*, 2004; Rotimi *et al.*, 2004). Most individuals with diabetes suffer complications (Agarwal *et al.*, 2013). Diabetes complications are divided into microvascular and macrovascular complications (Fröhlich-Reiterer & Borkenstein, 2010). Microvascular complications such as retinopathy, nephropathy and neuropathy are specific to DM, whereas macrovascular (coronary artery disease, peripheral arterial disease and stroke can occur in the absence of DM (FröhlichReiterer & Borkenstein, 2010). The proinflammatory microenvironment associated with hyperglycemia leads to the progression of microvascular complications in diabetes (Goh and Tooke, 2002).

High blood glucose levels results in the production of sorbitol, overproduction of superoxide, due to oxidative stress, overproduction of advanced glycation end-products and direct glucosemediated endothelial damage, which synergistically leads to progression of diabetic complications (Giunti *et al.*, 2006; Eid *et al.*, 2004). In diabetes mellitus, metabolism of all the main foodstuffs are altered. As a result of this, blood glucose concentration increases, cell utilization of glucose falls and utilization of fats and proteins increases (Guyton and Hall, 2006; Ismail *et al.*, 2000). These metabolic changes cause changes in endothelial permeability, extravascular protein deposition and coagulation, resulting in organ dysfunction (Giunti *et al.*, 2006).

#### 2.5 Diabetic Retinopathy

The main microvascular complication of diabetes is retinopathy (Yadav *et al.*, 2008). Diabetic retinopathy (DR) is an ocular manifestation of diabetes in which there is damage to the retina, as a result of prolonged hyperglycemia (Mookiah *et al.*, 2015). There are two major types namely non proliferative diabetic retinopathy (NPDR) with no formation of new blood vessels and proliferative diabetic retinopathy (PDR) with formation new blood vessels in the retina (Harney, 2006). Loss of vision in diabetic retinopathy arises as a result of diabetic macular edema (DME) and proliferative diabetic retinopathy (Rema and Pradeepa, 2007).

Numerous processes have been linked with the development of retinopathy in diabetes patients. Increase in glucose levels leads to the build-up of sugar molecules; which causes sorbitol accumulation in cells (Gabbay, 2004). Sorbitol accumulation in the cells leads to osmotic stress which causes the development of diabetic microvascular complications, including diabetic retinopathy (Fong *et al.*, 2004). In diabetic retinopathy, there is the formation advanced glycolysated end products (AGEs) from free radical reactions and production of reactive

oxygen species (Fong *et al.*, 2004). Vascular endothelial growth factor (VEGF), growth hormone and transforming growth factor- $\beta$ , contribute to the progress of diabetic retinopathy. However reduced VEGF levels is associated with delayed progression of retinopathy (Keenan *et al.*, 2007).

#### 2.6 Diabetes Nephropathy

Diabetes can damage blood vessel clusters (glomeruli) in the kidney that filter wastes from the blood which could lead to the development of kidney failure or end-stage renal diseases (Gross *et al.*, 2005). Diabetes causes damage in the kidney, resulting in a condition called diabetic nephropathy; characterized by proteinuria > 500 mg/24 hours in the setting of diabetes or albumin excretion of 30-299 mg/24 hours (Gross *et al.*, 2005). Globally, most of the chronic renal conditions are as a result of diabetic nephropathy (Leung and Lam, 2000). Microalbuminuria is associated with progression of nephropathy which increases the risk of cardiovascular diseases in diabetics (Leung and Lam, 2000).

In Nigeria, a study by Alebiosu *et al.* (2003) among diabetic patients with persistent proteinuria showed a diabetic nephropathy prevalence of 28.4%. Ajayi *et al.* (2014) in another study on chronic kidney disease among newly diagnosed asymptomatic hypertensives and diabetics in Nigeria revealed a total of 242 (38.5%) had CKD stages 3a, 3b and 4. Study by Eghan *et al.* (2007) in Ghana, also showed that prevalence of microalbuminuria in patients with diabetes was 43%. Without intervention, diabetic patients with microalbuminuria typically progress to proteinuria and overt diabetic nephropathy. This progression occurs in both type 1 and type 2 diabetes (Gross *et al.*, 2005).

#### 2.7 Diabetic Neuropathy

Diabetic neuropathy is defined as damage to the peripheral nerve, primarily by high glucose levels (American Diabetes Association, 2007). Many individuals with diabetes will ultimately develop neuropathy, with some populations at risk of undergoing a lower extremity amputation (Abbott *et al.*, 2011). Diabetic neuropathy affects the peripheral nervous system, causing damage to the spinal cord and central nervous system (Singleton and Smith, 2012; Obrosova, 2009). Hyperglycemic-associated conditions such as accumulation of polyol, AGEs and oxidative stress are main causes of damage to the peripheral nerves (Boulton, 2005). Poor glycemic control and duration of diabetes are the major causes of neuropathy (King *et al.*, 1999).

#### 2.8 Macrovascular Complications of Diabetes

Diabetes can lead to complications such as hypertension and atherosclerosis (Donaghue *et al.*, 2009). Conditions such as high blood pressure, elevated serum cholesterol and use of tobacco account for most of the macrovascular complications among diabetes patients (Dokken, 2008). The cells and extracellular matrix of the vascular wall are subject to disturbances mediated by oxidative stress, inflammatory responses and pathogenic mechanisms, and these are accelerated under hyperglycemic conditions (Dokken, 2008). Independent risk factors for vascular complications in diabetics are mostly dyslipidemia and poor glycemic control. Hypertensive heart disease is the cardiac damage related to chronic systemic arterial hypertension (Lavados *et al.*, 2005; WHO, 1997; Bronner *et al.*, 1995). Nowadays, hypertension prevalence among people >65 years of age, is roughly 30-40% in rural West Africa, 50% in semi-urban West Africa (de Ramirez *et al.*, 2010), 50-60% in South Africa and 30-50% in East Africa (de Ramirez *et al.*, 2014).

#### 2.9 Development of Cardiovascular Diseases in Diabetes

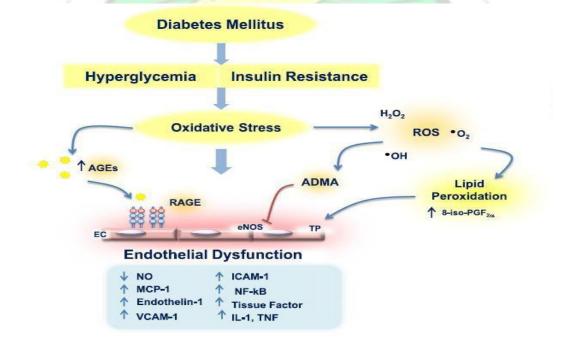
Cardiovascular disease is caused by disorders of the heart and blood vessels, and includes heart attacks, stroke, hypertension, peripheral artery disease, rheumatic heart disease, congenital heart disease and heart failure (WHO, 2014). The major causes of cardiovascular disease are tobacco use, physical inactivity, an unhealthy diet and harmful use of alcohol (WHO, 2014). An understanding of mechanisms underlying the development of CVD in patients with type 2 DM helps in the management of the disease. Atherosclerosis is most important process for cardiovascular disease development. Nutritional intake plays a major role in the development of cardiovascular disease, by influencing the onset of atherosclerosis progression to atherosclerotic plaque, which leads to thrombus formation (De Caterina *et al.*, 2006). CVD occurs through vascular abnormalities such as endothelial dysfunction and arterial stiffness, to the development of atherosclerotic lesions and eventually established end stage disease (Dzau and Braunwald, 1991).

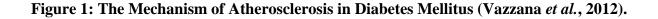
Atherosclerosis is a complex process of numerous cell interactions that lead to "fatty streak" formation, known as atherosclerotic plaques (Ross, 1999). Extracellular deposition of lipids is followed by an inflammatory infiltrate of monocytes and T lymphocytes to form fatty streaks (Ross, 1999). Monocytes develop into macrophages and scavenge modified lipids to form foam cells (Woollard and Geissmann, 2010). The inflammatory infiltrate and foam cells secrete inflammatory mediators and produce reactive oxidative species, resulting in the migration and proliferation of vascular smooth muscle cells (Hansson, 2005). This process continues with further lipid deposition, inflammatory infiltration and vascular smooth muscle cell

proliferation.

As the lesion develops, focal necrosis occurs at the centre; attracting further inflammatory cells and formation of fibrous tissue (Ross, 1999). As the lesion expands reduction in arterial blood flow occurs. The plaque may rupture with thrombus forming, leading to acute occlusion of the vessel (Ross, 1999). Multiple processes are involved in the initial development and progression of atherosclerotic plaques. Important processes in the development of atherosclerosis are endothelial dysfunction, oxidative stress, inflammation and increased arterial stiffness (Wilkinson and McEniery, 2004).

Vascular complications in diabetes mellitus are initiated, to a significant extent, by chronic hyperglycemia. High glucose concentration plays a central role in the progression of these complications, by altering gene expression of growth factors and cytokines, regulating vascular inflammation and activating macrophages and platelets (Hamilton *et al.*, 2004). High glucose mediates this by increasing the production of advanced glycation products, stimulating the polyol pathway, activating protein kinase C and enhancing the generation of reactive oxygen species (Hamilton *et al.*, 2004).





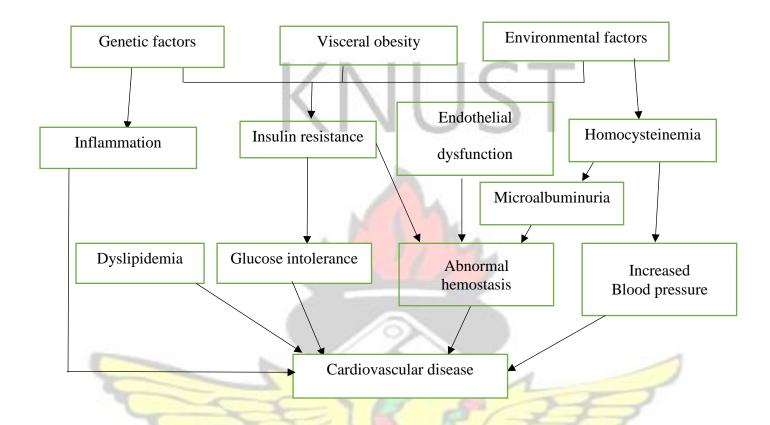


Figure 2: Type 2 diabetes mellitus and cardiovascular disease development (Martín-Timón *et al.*, 2014)

#### 2.10 Nutritional status and Health

Nutrition is the foundation of healthy living, as it is the science of food and its relationship with health (Parks, 2009). The ability of the body to maintain metabolic integrity as influenced by diet and nutrient levels is termed nutritional status (Bender, 2005). Poor dietary intake is closely associated with chronic diseases (Martin, 2006). Adequate nutrition is necessary for cardiac function, muscle strength, immunity, wound healing and psychological well-being (Martin, 2006). Adequate nutrition is a diet that contains the right proportion of macronutrients and micronutrients that are essential for the body's physiological function. Malnutrition refers to either inadequate intake of nutrients, resulting in underweight and nutrient deficiency; or excess intake of nutrients from poor dietary habit, resulting in overweight and obesity (Arthur and

John, 2000). The development of heart diseases have at their core, atherosclerosis and hypertension, both of which are greatly affected by diet and can be approached, from a nutritional point of view (Getz and Reardon, 2007).

The risk factors of cardiovascular diseases are mostly poor dietary intake, including high salt and saturated fat intake and unhealthy lifestyle, entailing smoking, alcohol consumption and physical inactivity (Arthur and John, 2000). Over-nutrition increases the risk of stroke by accelerating the development of obesity, hypertension, hyperlipidemia and diabetes (Hamilton *et al.*, 2004). Hence, assessment of nutritional status of patients with diabetes is important because their nutritional status influences disease progression. The risk factors for CVD include tobacco use, dietary intake and physical inactivity, overweight, obesity and diverse childhood habits (Celermajer and Ayer, 2006; Dong *et al.*, 2004). Studies show that dietary intake, specifically energy-dense diets high in saturated fats and salt have contributed to an increase in CVD incidence in developing countries (Hu, 2008). Excessive and harmful intake of alcohol also clearly increases CVD risk (Lucas *et al.*, 2005).

#### 2.11 Effect of Nutritional Status Indicators on Cardiovascular Diseases

#### 2.11.1 Anthropometric Indicators (Generalized Obesity and Central Obesity)

#### 2.11.2 Effect of BMI on Cardiovascular Diseases

Anthropometric measurements are vital in determining the risk factors for developing chronic diseases (Purnell *et al.*, 2000). The body mass index (BMI), an anthropometric indicator is a useful medical tool in determining whether an individual is underweight, normal weight, overweight or obese (Purnell *et al.*, 2000; Al-Sharafi and Gunaid, 2014). Body mass index (BMI) is a potential indicator of CVD risk factor, throughout the years (Cui *et al.*, 2005; Lobstein *et al.*, 2004). The incidence of cardiovascular diseases increases with body mass index

(BMI), as well as with age (Dexter et al., 2013). Researchers from the University of Oxford

have showed that the incidence of CHD increases with small incremental increases in BMI, hence for every 5 unit increase in BMI, CHD occurrence is increased by 23%, equivalent to the risk conferred by increase in age by 2.5 years (Dexter *et al.*, 2013). The risk for diabetes and hypertension have been shown by American Association of Clinical Endocrinologists (AACE) in 1998, to be about 2-fold in the mildly obese, 5-fold in moderately obese and 10fold in severely obese persons. The relationship between BMI and CVD mortality has been established; however, there is ambiguity about the specific types of CVD sturdily associated with excess weight (Lenz *et al.*, 2009; Pandeya *et al.*, 2012; Kurth *et al.*, 2005).

Increased body-mass index (BMI), is a well-documented risk for cardiovascular disease (Berrington de Gonzalez et al., 2010; Czernichow et al., 2011; Rahmanian et al., 2014; Prospective Studies Collaboration, 2009). A number of epidemiological researches have established a relationship of body mass index (BMI) with cardiovascular disease. In a study by Bogers *et al.* (2007) overweight (BMI > 25 kg/m<sup>2</sup>) and obesity were found to be associated with coronary heart disease (CHD). Observational studies undertaken in England and Asia have shown a relationship between body mass index (BMI) and ischemic heart disease (IHD) (Prospective Studies Collaboration, 2009; Asia Pacific Cohort Studies Collaboration, 2004). Consistently, a positive relationship has been shown between BMI and the risk of IHD and other cardiovascular diseases (Zheng et al., 2011; Song et al., 2004; Zhou et al., 2008). The high incidence of CVDs are associated with risk factors such as generalized and abdominal obesity, determined by BMI and the waist circumference (WC), respectively (Grundy et al., 2005; Lemieux et al., 1994; Chen et al., 2000; Ness-Abramof and Apovian, 2008). BMI correlates well with laboratory-based measures of adiposity in most clinical settings. However, BMI does not account for body fat distribution, body composition and size (World Health Organization, 1998).

#### 2.11.3 Waist Circumference and Cardiovascular diseases

Visceral fat is a relevant risk factor for CVD, diabetes and other metabolic conditions (Goran *et al.*, 2003).Waist circumference (WC) is an appropriate measure of abdominal adipose tissue (Ross *et al.*, 1992; Han *et al.*, 1995) which correlates closely with BMI (Onat *et al.*, 1999) and total body fat (Lean *et al.*, 1996).

Increased adipose tissue in the visceral organs have been shown to be strongly connected with cardiovascular disease than BMI (Zhu *et al.*, 2002; World Health Organization, 2000). Central obesity correlates closely with excessive visceral fat, which is associated with insulin resistance, hypertriglyceridemia, highly atherogenic small LDL particles, and low HDL levels, features considered pro-atherogenic (Navab *et al.*, 2006; Dinarello, 2000; Ho *et al.* 2001). Hence, the heightened inflammatory state, coupled with more atherogenic lipid profile and hypertension leads to an excess of clinical cardiovascular diseases in individuals with visceral and central obesity, regardless of their weight (Navab *et al.*, 2006). The Health Professionals' Follow-up Study, among men older than 65 years revealed that those with highest WHR had a nearly 3-fold increased risk of CHD than those in the lowest WHR (Rimm *et al.*, 1995).

In men, several studies support significant risk associated with abdominal adiposity. Other studies also revealed that, the WHR was associated with increased risk of cardiovascular mortality. Fuchs *et al.* in 2005, found that WC and WHR, but not BMI, were associated with hypertension in all study groups, and Wessel *et al.* in 2004, found that WC and WHR, but not BMI, were associated with cardiovascular events.

Obesity is a nutritional disorder ensuing from disproportion between energy intake and energy expenditure, primarily due to physical inactivity (Boden, 1997). Factors contributing to obesity are heredity, over-eating, altered metabolism, defective or decreased thermogenesis, decreased physical activities without appropriate reduction in food intake and some prescribed medication (WHO/FAO, 2003). Obesity is a significant risk factor for cardiometabolic diseases, including diabetes, hypertension, dyslipidemia, and coronary heart disease (CHD) (World Health Organization, 1998). The fat distribution in the upper body is greatly linked with hypertension than with fat in the lower body (Lemieux *et al.*, 1996; Larsson *et al.*, 1984). The abdominal fat results in increased plasma non-esterified free fatty acids, through the release of non-esterified free fatty acids into the portal vein. This in turn, causes an excess hepatic synthesis of triacylglycerols, leading to insulin resistance and hyperinsulinemia (Maegawa, 2000).

Inflammation mediated pathways are the main mechanism through which obesity causes cardiovascular diseases. Insulin resistance associated with obesity causes increases inflammatory responses, through increased production of inflammatory mediators (Madonna *et al.*, 2004). This affects the production of several pro-inflammatory cytokines (adipokines) and hormones (Madonna *et al.*, 2004). Insulin resistance allows the initiation and perpetuation of vascular inflammation, through the increased gene expression of vascular cell adhesion molecule (VCAM-1), monocyte chemoattractant protein-1 (MCP-1) and macrophage colony stimulating factor, CD-40L (Madonna *et al.*, 2004). Monocyte migrates into the sub endothelial space, matures into a resident macrophage and takes up lipid, through scavenger receptors such as SR-A and CD-36, forming a foam cell (Jialal *et al.*, 2002). Later, smooth muscle cells migrate to the surface and form the fibrous cap of the lesion, and lastly, lipid-laden macrophages release matrix metalloproteinase, causing plaque rupture and unstable angina associated with development of cardiovascular diseases (Brownlee, 2001). Atherosclerosis may proceed many years before the development of diabetes (Zavaroni *et al.*, 1989), through the formation of advanced glycation end products (Basta *et al.*, 2004), which can prolong an inflammatory response in the endothelium (Despres *et al.*, 1996). The atherogenesis begins as an endothelial cell dysfunction when various noxious attacks as dyslipidemia, hypertension, diabetes, smoking, *etc.* induce deficits of nitric oxide (NO) and prostacyclin (Boden, 1997; Grimble, 2000). Therefore, the increase in insulin production and plasma concentration that accompanies the compensated phase of insulin resistance increases atherogenic risk directly in obesity. Several studies have linked insulin resistance to systemic inflammation, as the result of increased concentrations of circulating free fatty acids (Boden, 1997; Grimble, 2002).

When hyperglycemia produces toxic effects on the endothelium, it propagates an inflammatory response in the endothelium (Cosentino and Egidy Assenza, 2004). There is evidence that obesity is associated with macrophage accumulation in the adipose tissue (Xu *et al.*, 2003). A recent study showed that the degree of infiltration of the adipose tissue by activated macrophages closely correlates with the adipocyte and that adipose-tissue-associated macrophage number is directly proportional to adiposity in humans (Weisberg *et al.*, 2003). If a positive energy balance is pro-inflammatory and increases cardiovascular disease risk, caloric restriction reduces inflammation, parallel to and likely reduces cardiovascular disease risk (Fontana *et al.*, 2004). The effect of caloric restriction protocols on risk factors for atherosclerosis has been evaluated, comparing individuals who had been restricting their food intake for 6 years and age-matched healthy individuals consuming a typical American diet (Fontana *et al.*, 2004).

The caloric restriction group had significantly lower body mass indices and percentages of body fat than the group consuming the American diet (Fontana *et al.*, 2004). Total serum cholesterol concentrations, LDL-cholesterol concentrations, triacylglycerol concentrations, fasting glucose, fasting insulin, C-reactive protein (CRP), platelet-derived growth factors A and B, and systolic and diastolic blood pressure were all markedly lower, and HDL cholesterol was higher, in the calorie restriction group than in the American diet group (Fontana *et al.*, 2007). None of the individuals in the caloric restriction group had evidence of atherosclerotic plaques (Fontana *et al.*, 2007). Very low plasma insulin concentrations and serum-derived platelet-derived growth factor A and B concentrations in the calorie restriction group suggest that caloric restriction results in a decreased stimulus for cell proliferation. Overweight, obesity and physical inactivity are major risk factors for stroke and important independent contributors to coronary heart disease, chronic heart failure, and other cardiovascular diseases (Murray and Lopez, 1997).

#### **2.12 Dietary Factors**

Dietary factors play a significant role in the development of cardiovascular disease (CVD) (Din, 2002; Hu & Willett, 2002), and dietary optimization is an important lifestyle intervention for the management of CVD and prevention (Ferrari *et al.*, 2004). Diet, a multi-component mixture of many nutrients, which may interact with one another and play a critical role in the development and prevention of cardiovascular disease (Mozaffarian *et al.*, 2011; Perrin *et al.*, 2002). The major environmental factors that lead to type II diabetes are sedentary lifestyle and over-nutrition, leading to obesity (Harris, 1991). According to World Heart Federation (2012), intake of diets low in saturated fat and rich in fruits and vegetables leads to a 73% reduction in CVDs.

Plasma cholesterol concentration increases with increased amount of cholesterol ingested daily (Arthur and John, 2000). Clear evidence exists that serum cholesterol correlates with the risk of CVDs and coronary death (Arthur and John, 2000). This can also be expressed by the relation between the percentage of daily energy intake from saturated fats, which correlates closely with serum cholesterol concentrations, and the degree of coronary artery disease. A high saturated fat diet increases blood cholesterol concentration by 15 to 25% (Arthur and John, 2000). When cholesterol is ingested, the rising concentration of cholesterol inhibits the most essential enzyme for endogenous synthesis of cholesterol, 3-hydroxy-3-methylglutaryl CoA reductase, thus providing an intrinsic feedback control system to prevent excessive increase in plasma cholesterol concentration (Arthur and John, 2000).

Studies by Kuller *et al.* (2006); Moore *et al.* (2011); Williams & Hoffman (2009) and Swain *et al.* (2008) on macro-and micronutrient diets in heart health, showed that the composition of the diet can be changed and still reach a reduction in a CVD risk by 16% to 21%. Further reduction of the risk was reached by substituting a part of the carbohydrates in the diet with either unsaturated fats or proteins (Swain *et al.*, 2008; Gross *et al.*, 2005).

#### 2.12.1 Fruit and Vegetable Intake

Fruit and vegetables are important components of a healthy diet, and their adequate daily intake could help prevent most diseases, such as cardiovascular diseases and certain cancers (WHO/ FAO, 2003; Bazzano *et al.*, 2003). Fruit and vegetable consumption decreases CVD risk, through the beneficial combinations of micronutrients such as potassium, antioxidants, phytochemicals and fiber in these foods (Tribble and Nutrition Committee, 1999). A diet rich in fruit and vegetables reduces the risk of cardiovascular disease mortality by 15% (Leenders *et al.*, 2013). Furthermore, more than 4% of deaths due to cardiovascular disease could be

prevented by consuming more than 400 grams of fruit and vegetables a day (Leenders *et al.*, 2013). The American Heart Association and other national agencies recommend a diet that includes  $\geq$ 5 servings of fruit and vegetables daily (US Department of Health and Human Services, 1995).

Research shows that fruit and vegetable intake help reduce cardiovascular diseases, through reduction of oxidation, inflammation and cell proliferation (Bonomo *et al.*, 2003; Genkinger *et al.*, 2004; Pereira *et al.*, 2004). Fruit and vegetable consumption prevents cardiovascular events by different mechanisms (Liu *et al.*, 2001; Padayatty *et al.*, 2003; Rasmussen *et al.*, 2006; Bonomo *et al.*, 2003; Genkinger *et al.*, 2004; Pereira *et al.*, 2003; Genkinger *et al.*, 2004; Pereira *et al.*, 2003; Genkinger *et al.*, 2004; Pereira *et al.*, 2003; Rasmussen *et al.*, 2006; Bonomo *et al.*, 2003; Genkinger *et al.*, 2004; Pereira *et al.*, 2004) such as reducing antioxidant stress, improving the plasma lipoprotein profile, lowering blood pressure, improving insulin sensitivity and hemostasis regulation (Van Duyn and Pivonka, 2000; Bazzano *et al.*, 2003, Wannamethee *et al.*, 2006; Samman *et al.*, 2003). Fruits possess antioxidant activity (i.e., scavenging free radicals, reducing oxidative stress, reducing LDL oxidation), cause induction of expression of hepatic LDL receptors, modulation of cholesterol synthesis, regulation of blood pressure, modify lipid profiles, prostanoid synthesis, and nitric oxide production, inhibition of cholesterol absorption, reduction of platelet aggregation and lowering serum Creactive protein and other inflammatory markers (Hertog *et al.*, 1997).

Research on the relationship between vegetable and fruit consumption on cardiovascular disease, established that there is a moderate inverse association between vegetable and fruit consumption on heart attack and stroke risk, with significantly higher, positive effects noted above five servings of vegetables and fruits per day (Murphy *et al.*, 2012; Hu *et al.*, 1999; Hu *et al.*, 2000). Evidence shows that adequate vegetables and fruit intake decrease the risk for obesity (WHO, 1990). Fruit and vegetable consumption contribute to increased feelings of

satiety (fullness), which can help in reducing overall energy intake, an important part of a weight management strategy (Azagba and Sharaf, 2011).

Several prospective studies have directly related fruit and vegetable intake to CVD (Ness and Powles, 1997). Several prospective studies have related higher fruit and vegetable intake to lower CVD mortality (Gaziano *et al.*, 1995) and morbidity (Joshipura *et al.*, 1999). A study of risk behaviors found that inadequate fruit and vegetable consumption has been associated with decreased life expectancy (Joshipura *et al.*, 1999). Nonetheless, there remains a low intake of fruits and vegetables globally (Hall *et al.*, 2009).

#### 2.12.2 Sodium Intake

Reduction in sodium intake is linked with a decrease in blood pressure in hypertensive and normotensive individuals (Hooper *et al.*, 2004; Uzu *et al.*, 2006; Sacks *et al.*, 2001; Hoffmann and Cubeddu, 2007). On the contrary, high dietary sodium intake is linked with increased stroke incidence, and cardiovascular disease mortality (Hooper *et al.*, 2004; Ekinci *et al.*, 2011; Pimenta *et al.*, 2009). There was a systematic review by Hooper *et al.* (2004) on the effects of advice to reduce dietary salt in patients with elevated or normal BP (Hooper *et al.*, 2004). The review found no significant differences in cardiovascular morbidity between low sodium and control groups (Hooper *et al.*, 2004; Elliott *et al.*, 1996; Vedovato *et al.*, 2004; Provenzano *et al.*, 2014). A prospective study in Finland for over 13 years revealed in men, the risk of CHD and CVD was associated with higher sodium excretion in urine (Tuomilehto *et al.*, 2001; Meneton *et al.*, 2005). A Scottish study in women, found an association between sodium excretion and the incidence of coronary events (Tunstall-Pedoe *et al.*, 1997). Among obese people, a high salt intake is associated with increased risk of CVDs. A prospective large study by He *et al.* found that in participants who were overweight, sodium intake was associated with

increased frequency of stroke and mortality from CHD and CVD (He *et al.*, 1999; Huang *et al.*, 1998).

#### 2.13 Biochemical Risk Factors

#### 2.13.1 Dyslipidemia

The most important risk factor for complications in diabetes is dyslipidemia, the second most prevalent cardiovascular risk factor globally (Tekes-Manova *et al.*, 2005; Goldberg, 2001; Krauss, 2004). Dyslipidemia is defined by the presence of one or more than one abnormal serum lipid concentration, according to United State National Cholesterol Education Program Adult Treatment Panel (NCEP-ATP) III guideline (Safeer and Ugalat, 2002). The reasons for the management of lipoprotein abnormalities in diabetic patients are to prevent pancreatitis, due to severe hypertriglyceridemia and to reduce the risk of macrovascular complications (Kako *et al.*, 2002; Jayarama *et al.*, 2012; Haffner *et al.*, 1998).

Dyslipidemia contributes to the development of arteriosclerosis, which progresses even faster in the presence of hypertension and diabetes mellitus (Dunn, 1990). In type 2 diabetes mellitus (T2DM), the most common abnormal lipid pattern is a combination of elevated TG levels and decreased HDL cholesterol (Dunn, 1990). The functions of HDL particles that lead to cardioprotective effects, include promotion of cellular cholesterol efflux and direct antioxidative and anti-inflammatory properties (Lamarche *et al.*, 1996). In the Quebec Cardiovascular Study, HDL<sub>2</sub> particles contributed to the cardioprotective effects of high HDL cholesterol levels, more than HDL<sub>3</sub> particles (Assmann and Schulte, 1992; Manninen *et al.*, 1992).

Diabetic dyslipidemia is linked with quantitative and qualitative lipid abnormalities due to increased hepatic lipase and decreased lipoprotein lipase activity, resulting in decrease in VLDL clearance (Arora *et al.*, 2007). A study conducted in Saudi Arabia, on lipid profile of diabetic patients, showed the prevalence of dyslipidemia among diabetic with high risk levels of TC, TG, LDL-C and HDL-C (Habib, 2006; Marcus, 2001; Lehto *et al.*, 1997). A prospective study has found that plasma triglyceride level is a risk factor for cardiovascular disease in men and women, independent of high-density lipoprotein cholesterol level (Hokanson and Austin, 1996).

The Strong Heart Study, among American Indian patients with diabetes showed that increased LDL-C level by 10 mg/dl, increased the risk of cardiovascular disease by 12% (Howard *et al.*, 2000). Many prospective epidemiologic studies have reported a positive relationship between serum triglyceride levels and incidence of coronary heart disease, especially in patients with diabetes (Tseng *et al.*, 2006).

# 2.14 Unhealthy Lifestyle and Cardiovascular Disease Risk in Diabetes

#### 2.14.1 Smoking

Smoking is connected with weakening in metabolic control in diabetic patients (Bott *et al.*, 1994) which is linked with an increased risk for development of cardiovascular complications (Chase *et al.*, 1991). The mechanism by which smoking causes cardiovascular disease is through impaired insulin sensitivity; due to compromised beta-cell function and hemodynamic dysregulation in capillary vascular bed (Kirschbaum *et al.*, 1992; Wellman and Kamp, 2008). Furthermore, smoking causes bronchitis and pulmonary infections which increase inflammatory markers associated with impaired insulin sensitivity (Kirschbaum *et al.*, 1992; Peters *et al.*, 2008). Direct effects of tobacco smoking are lipotoxicity, resulting from increased triglyceride levels, hypercortisolemia, increase in abdominal fat tissue and elevated sympathetic nervous activation (Kirschbaum *et al.*, 1992).

Smoking leads to nicotine inhalation which results in increase circulating levels of insulinantagonistic hormones (growth hormone, catecholamines and cortisol) and negatively affects the autonomic nervous system (Kirschbaum *et al.*, 1992; Lucini *et al.*, 1996). Nicotine, by these mechanisms, decreases insulin sensitivity, which directly or indirectly leads to the development of cardiovascular disease (Bergman and Ader, 2000; Kirschbaum *et al.*, 1992; Lucini *et al.*, 1992; Lucini *et al.*, 1992;

1996). Also, smoking increases circulating free fatty acid levels (Kershbaum and Bellet, 1996; Bott *et al.*, 1994), and causes an additional negative effect on the insulin-mediated glucose uptake. In a study of patients with diabetes, followed in the UKPDS, smoking was shown to increase the risk of CHD (Turner *et al.*, 1998; Morrish *et al.*, 1991). The expected relative risk incidence of myocardial infarction attributable to smoking was 1.350 (95%CI: 1.11-1.59). This reveals that smoking is an independent and significant risk factor for stroke (Kothari *et al.*, 2002) and peripheral vascular disease (Adler, 2002). A study in 1997 by Chaturvedi *et al.* revealed that smoking cessation decreases mortality risk in diabetes patients.

#### 2.14.2 Alcohol Intake

Alcohol consumption is a major risk factor for diseases (Alwan, 2011). Globally, alcohol causes more deaths than deaths by infectious diseases (Alwan, 2011). Injuries and cardiovascular diseases are the major complications of excessive alcohol (Alwan, 2011). Globally, alcohol causes more deaths in males than in females (Alwan, 2011). The electrical movement of the heart is altered due to excessive alcohol consumption which leads to ventricular fibrillation and cardiac arrest (Cairns *et al.*, 1984; Cooper *et al.*, 2004). Furthermore, long-term excessive alcohol intake weakens the heart muscle and makes it less efficient, through dilated cardiomyopathy and damage in myocardial tissue through oxidative stress (Mukamal *et al.*, 2010). A study by Ormel *et al.* (2007) in Nigerians found that patients who were alcohol abusers were more likely to have heart disease, compared to non-alcohol abusers. Similarly, in Nigerian patients with heart failure in a teaching hospital in Jos, more than 24% of heart failure patients reported regular alcohol intake (Laabes *et al.*, 2008; Moeini *et al.*, 2011). Puepet and Ohwovoriole (2008) conducted a study to identify risk factors for type 2 diabetes in Jos,

Nigeria, and found that alcohol consumption was highly prevalent among the diabetic patients, In a study in Kenya, it was found that excess alcohol consumption was related to increased likelihood of glucose intolerance in men (Christensen *et al.*, 2009). In a prospective cohort study among Cameroonians, alcohol consumption was related to increased likelihood of cardiovascular death (Kengne and Awah, 2009). Alcohol consumption has been associated with a lower risk of CVD in individuals who are light to moderate drinkers (Mukamal *et al.*, 2010; Facchini *et al.*, 1994; Kiechl *et al.*, 1996). People who refrain from alcohol show a higher death rate from CVD than do light drinkers (up to three drinks per day), but heavy drinkers (more than three drinks per day) die more frequently of CVD than do light drinkers (Lands and Zakhari, 1990). Excessive alcohol drinking offsets the benefits of alcohol and has been connected with hypertension, hemorrhagic stroke, and cardiomyopathy (Lands and Zakhari, 1990).

The risk of hypertension is reduced when excessive drinkers decreased their alcohol intake (Lands and Zakhari, 1990; Mahan and Escott-Stump, 2008). Significant association between excessive alcohol consumption and hypertension was observed among females in Munich by Cairns *et al.* (1984). Binge drinking has been associated with increased risk of cerebral hemorrhage and ischemic stroke (Lands and Zakhari, 1990).

#### **2.14.3 Physical Inactivity**

According to World Health Organization (2012), an adequate amount of physical activity decreases the risk of heart diseases (WHO, 2012). The terms physical activity and exercise are frequently used interchangeably. Exercise is a sub-category of physical activity (Caspersen *et al.*, 1991). While physical activity is any movement which is created by skeletal muscles, exercise is always planned, recurring physical activity that has a structure and is undertaken with the purpose of improving the physical fitness (Caspersen *et al.*, 1991).

According to the American Diabetes Association, physical activity helps control blood glucose levels, lowers blood pressure, improves blood fats, as well as reduce the amount of insulin doses or diabetic pills after losing weight (American Diabetes Association, 2004). It also helps maintain the weight a person loses and lowers the risk of health problems, hence helps sustain a healthy life (American Diabetes Association, 2007). Regular physical exercise reduces the risk of cardiovascular diseases (Swartz *et al.*, 2003, Murphy *et al.*, 2002). This evidence is subject to other lifestyle changes that take place together with exercise (for example stopping smoking, a balanced diet, etc. (Shephard and Balady, 1999; Powell *et al.*, 1987).

Patients with DM have a reduced capacity to exercise, due to ageing, overweight and the presence of left ventricular dysfunction (Fang *et al.*, 2005; Gregg *et al.*, 2000). Exercise improves insulin sensitivity in diabetic patients in the same way as it does in non-diabetic patients (Kirwan *et al.*, 2009; Winnick *et al.*, 2008). Patients with diabetes have greater insulin resistance, which improves with physical exercise (Richards *et al.*, 2010; Le Brasseur and Ruderman, 2005). Increased physical activity achieves higher mitochondrial enzyme activity and increases insulin sensitivity (Richards *et al.*, 2010). Multiple studies have shown physical

LI ADOT

exercise improves cardiovascular risk factors (dyslipidemia, hypertension and body composition) in patients with DM (Chudyk and Petrella, 2011).

Different kinds of physical activities do not exert the same influence on CVD risk. Aerobic exercise only or combined with resistance exercise improves glycemic control, BP, the amount of TGs and WC (Sluik *et al.*, 2012). In prospective cohort studies, exercise promoted reduced cardiovascular mortality in patients with T2DM (Sluik *et al.*, 2012). Results from the Nurses' Health Study by Hu *et al.* (2001) reported that women with T2DM who exercised for at least 4 h per week had a 40% lower risk of developing heart diseases, compared to those who did not. This risk improvement remained after adjustments for smoking, BMI and other cardiovascular risk factors. High blood pressure, cholesterol levels, obesity, diabetes, and lack of physical activity can all be positively affected by a regular exercise program (Jenna and Barry, 2012).

Regular exercise improves glycemic control in all types of diabetes (Goodpaster *et al.*, 2010). Regular exercise can reduce free fatty acid load to liver, the main cause of insulin resistance in obesity and thereby reduce hepatic insulin resistance (Haus *et al.*, 2011). Exercise recommended is moderate exercise for 30 minutes a day or moderate physical activity like brisk walking at least 150 minutes per week (Tuomilehto *et al.*, 2001).



#### **CHAPTER THREE**

#### MATERIALS AND METHODS

#### 3.1 Study Design

This study was a descriptive comparative cross-sectional study, carried out from June to November, 2015.

#### 3.2 Study Site

The study was conducted at three sites; St. Michael's Hospital, located at Jachie-Pramso in the Bosomtwe district, Kumasi South Hospital, located at Agogo in Kumasi, and Aninwah Medical Centre located at Emena in Kumasi; all three study sites are located in the Ashanti Region of Ghana.

#### **3.3 Study Population and Sample Size**

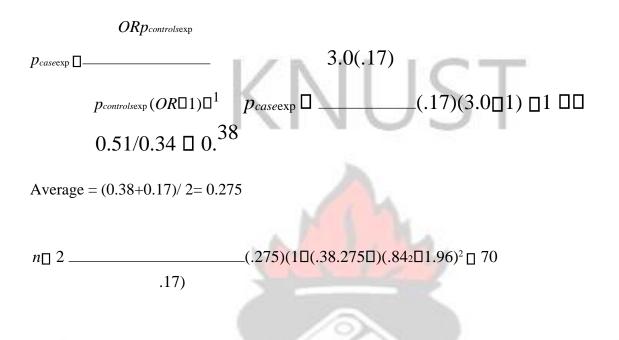
Diabetic patients who visited the St. Michael's Hospital, Kumasi South Hospital and Emena Hospital were used for the study. One hundred and forty five diabetes, patients made up of 97 who had cardiovascular disease; mainly stroke, hypertension and heart failure and 48 without cardiovascular disease, were used for the study. The minimum sample size of 70 diabetic patients used, based on calculations using the following variables: The proportion exposed in the control group 17%, at 80% power,  $Z_{\Box}=0.84$ . At 0.05 significance level,  $Z_{\Box}=1.96$ , Odds Ratio (0R)=3 r=1 EpiTools (*Epitable calculator*, AusVet, power at 80%) (http://epitools. ausvet.com.au/content.php?page=casecontrolSS&P1=0.17&RR=3&Conf=0.95&Power=0.8)

 $n\Box(rr\Box1)(\overline{p})(1\Box(pp_1)(\Box Zp_{\Box 2})\Box_2 Z_{\Box/2})^2$ 

 $n\Box(rr\Box 1)(p)(1\Box(pp_1)(\Box Zp_{\Box 2})\Box_2 Z_{\Box/2})^2$ 

(Charan and Biswas, 2013; Naing, 2003).

To get proportion of cases exposed:



# 3.4 Inclusion and Exclusion Criteria

# **3.4.1 Inclusion Criteria for Control Participants**

- Type1 and 2 diabetes patients without any cardiovascular disease
- Male or female diabetics above 18 years
- Willingness to take part in the study

# **3.4.2 Inclusion Criteria for Case Participants**

- Type1 and 2 diabetes patients
- Diabetics above 18yrs with cardiovascular disease such as hypertension, heart failure and stroke diagnosed by a physician.

ANE

• Willingness to take part in the study

# **3.5 Exclusion Criteria for Participants**

- Gestational diabetes patients
- Diabetes patients below 18yrs

• Diabetics with other cardiovascular complications different from stroke, hypertension and heart failure.

#### **3.6 Sampling Procedure**

Participants were recruited by random sampling in all three hospitals for patients who were willing to participate in the study.

#### 3.7 Questionnaire-Based Data Collection

A structured questionnaire was used to collect information from participants. The questionnaire was divided into the five sections: A: Demographic information (age, gender, place of residence, level of education and employment status).

B: dietary habits

C: physical activity

D: family and medical history, co-morbidities and

E: lifestyle of respondents.

### 3.8 Pre-testing of Questionnaire

The questionnaire used for data collection was piloted on ten participants at the Diabetes center at the St. Michael's Hospital. This was done to ensure that all relevant information needed for this research were captured appropriately. The pre-test results revealed that the data capturing sheets needed some minor modifications. Hence, some questions were eliminated and those which were not clear were modified

#### **3.9** Anthropometric Variables

Anthropometry is the measurement of body size, weight, and proportions (Lee and Nieman, 2003). Anthropometric measurements made included weight, height, waist circumference, hip

SANE

circumference and waist-to-hip ratio. The measurements were compared to reference standards to assess risk for various diseases (Laquatra, 2004).

#### **3.9.1 Body Mass Index**

The BMI was calculated mathematically from the height and weight measures (Tsigos *et al.*, 2008). BMI = weight (kg) / Height (m<sup>2</sup>). The values were used to classify BMI into categories, based on the World Health Organization criteria (2012).

#### 3.9.2 Weight Measurement

Weight was measured with an Omron Body Composition Monitor scale (BF- 506, Omron Healthcare, Inc., Vernon Hills, IL, USA), to the nearest 0.1kg, with subjects in light clothing and standing erect. Each subject stood still, with weight evenly distributed on both feet, with no additional support, while the reading was taken (Heymsfield *et al.*, 1999).

#### **3.9.3 Height Measurement**

Height was measured with a portable Seca stadiometer (Hamburg, Germany) to the nearest 0.1 centimeter. Subjects were made to stand upright on a base plate without shoes, with their head and back straight, feet together and heels touching the back of the plate. The head plate was lowered to touch the top of the head and height noted (Heymsfield *et al.*, 1999).

# 3.9.4 Waist and Hip Circumference

Waist circumference was measured with a Gulick II springloaded measuring tape (Gay Mills, WI) midway between the inferior angle of the ribs and the suprailiac crest and recorded to the nearest 0.1cm (Hammond, 2000). Hip circumference was measured with a Gulick II springloaded measuring tape (Gay Mills, WI) around the point with the maximum circumference of the buttocks and recorded to the nearest 0.1cm (Hammond, 2000).

Waist to hip ratio (WHR) was calculated by dividing the waist circumference by the hip circumference. All measurements were taken with participants dressed in light clothing (Sizer & Whitney, 2003).

#### **3.10 Dietary Intake**

Dietary intake was assessed using a food-frequency questionnaire and 24-hour dietary recall methods, to determine dietary intake and habits over the past year. Food models and household measurements were used in portion size estimation. Handy measures such as ladles, spoons (dessert and tea spoons), milk tins, sardine tins and calibrated cups were also used to help collect information on meal quantities.

#### 3.11 Cardiovascular Disease Diagnosis

The cardiovascular diseases used were stroke, hypertension and heart failure. The diagnosis of cardiovascular disease was based on the American Heart Association criteria and classification, as diagnosed by a physician. Hypertension was defined using the World Health Organization and International Society of Hypertension criteria (WHO and ISH, 2003) as systolic blood pressure (SBP)  $\geq$  140 mmHg and/or diastolic blood pressure (DBP)  $\geq$  90 mmHg.

#### 3.12 Blood Pressure Measurement

Blood pressure (systolic blood pressure) and (diastolic blood pressure) were obtained twice with subjects in sitting position, using digital sphygmomanometer (an Omron Blood Pressure Monitor model BP 785). Three measurements were taken, within a minute interval, in the right arm and the average of the last 2 readings used.

#### **3.13 Biochemical Indicators Measurements**

The biochemical measurements made were fasting blood glucose levels (FBG), serum total cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) levels and serum creatinine.

#### **3.13.1 Blood Collection and Handling**

Five millimeter fasting blood samples (overnight fast between 8-12 hours) were drawn from the antecubital vein on the anterior forearm (the side within the fold of the elbow) into gel separator tubes (BD, Plymouth, PL6 7BP, UK) for total cholesterol, HDL, LDL and TG. Blood samples were taken from participants before 9:00 hours in the morning. Fluoridated blood samples were kept on ice, prior to centrifugation within 15 minutes of blood draw. The fluoridated blood was centrifuged (Zentrifugen, D-78532, Tuttlingen, Germany) at 3000 rpm for 5 minutes to separate the serum from the deposit. All analysers were calibrated before the start of the analysis. Results which were flagged as high or low were repeated to verify their reproducibility.

#### 3.13.2 Fasting Blood Glucose Measurement

The fasting blood glucose (FBG) was measured using the ONETOUCH glucometer (Serial number: DCFVWB3E), using the procedures below. Fasting blood glucose (FBG) reading was measured and recorded for each participant.

- 1. The glucometer was prepared for the test by switching it on and ensuring that it was working properly
- 2. The researcher then washed and sanitized hands and wore the laboratory hand gloves
- 3. The test strip was then inserted into the slot provided on the glucometer
- 4. Alcohol was poured on a cotton ball/cotton wool till soaked with alcohol

- 5. The side of the thumb where the blood sample was to be drawn was then cleaned with the cotton wool soaked with alcohol
- 6. When the glucometer indicated that sample could be dropped on the strip, the researcher used the lancet pricker to prick appropriately at the cleaned area where sample was to be taken
- 7. A drop of blood was then placed directly onto the test strip inserted into the glucometer
- 8. The researcher then waited for the reading to appear in few seconds
- 9. The researcher then read and recorded the fasting blood glucose of the patient.

# 3.13.3 Lipid Profile: (Triglycerides, HDL, LDL and total cholesterol)

The lipid profile - total cholesterol (TC), high density lipoprotein (HDL) and triglycerides (TG) of each patient was measured with Selectra Junior Auto Analyzer (Vital Scientific, N.V. Netherlands) and low density lipoprotein (LDL) calculated by same analyzer. 10µl of serum o was pipetted into the auto analyzer at internal temperature of 37 C and an incubation period of

10 minutes, after which readings for each of the lipid parameters were displayed and recorded.

#### 3.13.4 Serum Creatinine

Serum creatinine was determined with a standard autoanalyzer RA-XT (Bayer, Elkhart, IN). 10µl of serum was pipetted into the auto analyzer at internal temperature of 37 C and an incubation period of 10 minute, after which readings for the creatinine were displayed and recorded.

#### **3.14 Operational Definition of Terms**

Diabetes was diagnosed when fasting blood glucose was 126 mg/dl (6.40 mmol/l) and higher. (The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 2003). BMI

was categorized according to the WHO (Laquatra, 2004):

Underweight-BMI less than 18.5 kg/m<sup>2</sup>

Normal BMI-18.5 to 24.9 kg/m<sup>2</sup>

Overweight BMI-25.0 to 29.9 kg/m<sup>2</sup>

Obesity BMI-30.0 kg/m<sup>2</sup> and

Obese class I:  $30.0 - 34.9 \text{ kg/m}^2$ , Obese class: II  $35.0 - 39.9 \text{ kg/m}^2$ , Obese class III: >  $40 \text{ kg/m}^2$ . Using the waist circumference participants were classified as follows: normal: WC < 80 cm, overweight: WC of 80 - 87.9 cm and obese WC ≥ 88 cm for females (NHLBI Obesity Education Initiative, 2000). Men with waist circumference (WC) < 94 cm, 94-101.9 cm and ≥102 cm were classified as normal, overweight and obese respectively. Men with WHR < 0.90,  $0.90 - 0.99 \text{ and} \ge 1$  and women with WHR < 0.80,  $0.80-0.84 \text{ and} \ge 0.85$  were classified as normal weight, overweight and obese respectively (NHLBI Obesity Education Initiative, 2000).

Hypertension was defined using the World Health Organization and International Society of Hypertension criteria (WHO and ISH, 2003) as systolic blood pressure (SBP)  $\geq$  140 mmHg and/or diastolic blood pressure (DBP)  $\geq$  90 mmHg. Raised total cholesterol defined as total cholesterol  $\geq$  6.50 mmol/L. High serum triglycerides defined as serum triglycerides  $\geq$  1.70 mmol/L. High serum LDL-C defined as LDL-C  $\geq$  4.90 mmol/L. Low serum HDL-C defined as with HDL- Cholesterol < 1.55 mmol/L (Dyslipidemia was diagnosed according to NCEP ATP III). High creatinine levels defined as creatinine  $\geq$  120 µmol/l.

Insufficient physical activity, defined as attaining less than 150 minutes of moderate- intensity physical activity (e.g slow dancing, brisk walking and table tennis) per week or less than 75 mins of vigorous–intensity physical activity (e.g. running and football) per week (Bull *et al.*, 2009). Diabetics currently smoking any tobacco product includes both daily and non-daily or occasional smoking.

#### **3.15 Ethical Considerations**

This study was performed under a protocol that was reviewed and approved by the Committee on Human Research, Publications and Ethics (CHRPE), School of Medical Sciences, Kwame Nkrumah University of Science & Technology (KNUST), Kumasi. Letters were sent to the St. Michael's Hospital, Kumasi South Hospital and Emena Hospital to seek permission, which was duly granted. All the prospective participants in this study were informed about the study. The purpose of the study was thoroughly explained to the participants to select those willing to participate and those who declined to participate in the study were excluded. The patients who met the inclusion criteria and were willing to partake in the study were made to sign or thumbprint on the consent forms before they were enrolled to take part in the study.

#### **3.16 Statistical Analysis**

Anthropometric, biochemical, dietary, demographic and socio-economic data were analysed by using Statistical Package for Social Sciences (SPSS), version 20. Continuous variables were expressed as mean and standard deviation, whereas categorical variables were expressed as percentages. The significance of the differences in the mean values between cases and controls for normally distributed parameters were determined using the independent samples t-test for continuous variables and Chi-square test for categorical variables at 95% confidence level. Binary logistic regression model was used to test for independent association with CVDs. Pvalues <0.05 were reckoned as statistically significant.



#### **CHAPTER FOUR**

#### RESULTS

#### Introduction

Data were obtained from 145 diabetic patients in three different hospitals in the Ashanti region and analyzed.

#### 4.1 Demographic Characteristics of Respondents

Tables 4.1 and 4.2 show the socio-demographic characteristics of respondents. A total of 145 known diabetic patients were recruited. Out of the participants, 76.6% were females. More female respondents (48.3%), had cardiovascular diseases. However, based on gender, the difference between diabetics with and without CVDs showed no statistical significance (p=0.076) (Table 4.2). More female respondents were overweight and obese (31.7% and 17.9%) respectively, with no significant gender differences in BMI (p=0.930) (Table 4.3).

The overall mean ( $\pm$ SD) age was 55.88  $\pm$  12.75, ranging from 26-93 years. Tables 4.2 shows more than half of the patients (53.1%) were in the age group 40-60 years, and diabetics with age above 40 years had significantly higher prevalence of CVDs (p=0.000).

On educational attainment Tables 4.1 shows majority of the diabetics (69.0%), were Junior High School leavers. From Table 4.2, diabetics with CVDs had higher percentage of the lowest level of education, while having the lower percentages of secondary and tertiary education. However, the difference in educational status between the diabetics with and without CVDs was not significant (p=0.277).

On employment status, more diabetics with CVDs were unemployed, compared to those without CVDs. The difference in employment status showed significance (p=0.039).

On income status, diabetics with CVDs had higher proportion of respondents with low income status but lower percentage of those with high income status, with significant differences in the income level (p=0.021). The majority of the respondents, 85.5% were of Akan ethnicity. However, ethnic differences between diabetics with and without CVDs showed no significant differences (p=0.649) (Table 4.2).

#### 4.2 Cardiovascular Diseases

Out of the diabetic respondents, 66.9% reported suffering from at least one of the cardiovascular diseases of study (hypertension, stroke or heart failure), whilst 33.1% of the respondents had no incidence of cardiovascular disease. From Table 4.4, hypertension was the most common cardiovascular disease. According to Table 4.5, more female diabetics had CVDs but the difference was not statistically significant (p=0.076).

#### 4.3 Anthropometric and Blood Pressure Data of Respondents

The anthropometric data and blood pressure of participants are presented in Tables 4.6 and 4.7. The mean BMI of respondents with CVDs and those without CVDs were similar. From Table 4.6, diabetics who were overweight and obese had higher prevalence of CVDs. However, the difference in BMI between the diabetics with and without cardiovascular disease was not significant (p=0.89).

More diabetics with CVDs had higher WC and WHR than those without CVDs. Based on WC, diabetics with CVDs had significantly higher means values (p=0.000). Additionally, waist to hip ratio of diabetics with CVDs was significantly higher (p=0.005) than those without CVDs (Table 4.7).

# 4.4 Blood Pressure of Respondents

Generally, both systolic and diastolic blood pressure were higher in diabetics with CVDs than those without CVDs with significant statistical differences (p=0.00) (Table 4.7).

KNUST

| Variable                  | Diabetic Respondents<br>(N=145) No. (%) |
|---------------------------|---|
| Gender                    |   |
| Male                      | 34 (23.3)                               |
| Female                    | 111 (76.7)                              |
| Age (years)               |   |
| Below 40 Years            | 15 (10.3)                               |
| Between 40 - 60 Years     | 77 (53.1)                               |
| Above 60 Years            | 53 (36.6)                               |
| Educational Level Junior  | 2                                       |
| High School               | 100(69.0)                               |
| SHS/A Level completed     | 24(16.6)                                |
| Tertiary completed        | 21(14.4)                                |
| Income Level              | N/ FFF                                  |
| Low income                | 104 (71.7)                              |
| Middle income             | 34 (23.45)                              |
| High income level         | 7 (4.8)                                 |
| Employment Status         |   |
| Employed                  | 76 (52.4)                               |
| Unemployed                | 69 (47.6)                               |
| Marital Status            |   |
| Married                   | 77 (53.1)                               |
| Single/divorced/separated | 33 (22.8)                               |
| Widowed                   | 35 (24.1)                               |
| Ethnicity                 |   |
| Akan                      | 124 (85.5)                              |
| Ewe<br>Ga                 | 7 (4.8)                                 |
| Ga                        | 5 (3.4)                                 |
| Hausa/Gonja/Dagbani       | 9 (6.2)                                 |

| Variable                  | Diabetics without                   | Diabetics with CVD | Total                 | P-Value |
|---------------------------|-------------------------------------|--------------------|-----------------------|---------|
|                           | CVD (N=48) No.                      | (N=97)             | (N=145)               |         |
|                           | (%)                                 | No. (%)            |                       |         |
| Gender                    | and the second second second second |                    |                       | 0.076   |
| Male                      | 7 (4.8)                             | 27 (18.6)          | 34 (23.3)             |         |
| Female                    | 41 (28.3)                           | 70 (48.3)          | 111 (76.7)            |         |
| Age                       |                                     | US                 |                       | 0.000   |
| Below 40 years            | 12 (8.3)                            | 3 (2.06)           | 15 (10.3)             | 0.000   |
| 40-60 years               | 27 (18.6)                           | 50 (34.5)          | 77 (53.1)             |         |
| Above 60 years            | 9 (6.2)                             | 44 (30.34)         | 53 (36.6)             |         |
| Educational Status        |                                     | × /                | . ,                   | 0.277   |
| JHS Level                 | 34(23.4)                            | 66 (45.5)          | 100(69.0)             |         |
| SSS/A Level               | 5 (3.5)                             | 19 (13.1)          | 24(16.6)              |         |
| Tertiary Level            | 9 (6.2)                             | 12 (8.3)           | 21(14.5)              |         |
| <b>Employment Status</b>  |                                     |                    |                       | 0.039   |
| Employed                  | 31 (21.4)                           | 45 (31.0)          | 76(52.4)              |         |
| Unemployed                | 17 (11.7)                           | 52 (35.9)          | 69(47.6)              |         |
| Marital Status            |                                     |                    |                       | 0.0097  |
| Married                   | 17 (11.7)                           | 60 (41.4)          | 77 (53.10)            |         |
| Single/divorced/separated | 16 (11.0)                           | 17 (11.7)          | <u>33(22.76)</u>      |         |
| Widowed                   | 15 (10.3)                           | 20 (13.9)          | 35(24.13)             |         |
| Income Level              | Sec.                                | Y XX               |                       | 0.021   |
| Low –Income               | 28 (19.4)                           | 75 (51.7)          | 93(64.1)              |         |
| Middle Income Level       | 15 (10.3)                           | 20 (13.8)          | 35(24.1)              |         |
| High Income Level         | 5 (3.4)                             | 2 (1.4)            | 7(4.8)                |         |
| Ethnicity                 | man                                 |                    |                       | 0.649   |
| Akan                      | 43 (29.7)                           | 81 (55.9)          | 124(85.5)             |         |
| Ewe                       | 1 (0.7)                             | <u>6 (4.1)</u>     | 7(4.8)                |         |
| Ga                        | 1 (0.7)                             | 4 (2.8)            | 5(3.5)                |         |
| Hausa/Gonja/Dagbani       | 3 (2.0)                             | <u>6 (</u> 4.1)    | 9( <mark>6.2</mark> ) | 921     |

| Table 4.2 Comparison of Demographic Characteristics of Respondents with and without |
|---|
| CVDs  |

# Table 4.3 Differences in BMI of Male and Female Diabetic Respondents

| Variable  | Males     | Females   | P-value |
|---|-----------|-----------|---------|
| BMI Ranks                                       | IE NO     | 3         | 0.930   |
| Underweight; BMI < 18.5 kg/m <sup>2</sup>       | 0 (0.0)   | 1 (0.7)   |         |
| Normal, BMI 18.5 to 24.9 $kg/m^2$               | 12 (8.3)  | 38 (26.3) |         |
| Above $25.0 - 29.9 \text{ kg/m}^2$ , overweight | 15 (10.3) | 46 (31.7) |         |

| Cardiovascular diseases | Frequency (%) |
|-------------------------|---------------|
| Hypertension            | 96 (66.2)     |
| Stroke                  | 12 (8.3)      |
| Heart failure           | 5 (3.5)       |

#### **Table 4.4 Prevalence of Cardiovascular Diseases among Diabetics**

#### Table 4.5 Gender and Age of Respondents with and without CVDs

| Variable       | Diabetics without CVD | Diabetics with CVD | P-value |
|----------------|-----------------------|--------------------|---------|
|                | N=48 (%)              | N=97 (%)           |         |
| Gender         |                       |                    | 0.076   |
| Male           | 7 (4.8)               | 27 (18.6)          |         |
| Female         | 41 (28.3)             | 70 (48.3)          |         |
| Age            |                       |                    | 0.000   |
| Below 40 years | 12 (8.2)              | 3 (2.2)            |         |
| 40 - 60 years  | 27 (18.6)             | 50 (34.5)          |         |
| Above 60 years | 9 (6.2)               | 44 (30.3)          |         |

# 4.5 Biochemical Data of Respondents

#### 4.5.1 Fasting Blood Glucose of Participants

Tables 4.8 and 4.9 show biochemical data of participants. The mean FBG levels was high for the whole population, with similar FBG levels for diabetics with and without CVDs. According to Table 4.9, high FBG levels was more prevalent among diabetics with CVDs than those without CVDs but the difference was not statistically significant (p=0.905).

#### 4.5.2 Lipid Profile and Creatinine Levels of Respondents

Dyslipidemia was observed in some of the patients. Hypertriglyceridemia was the most prevalent lipid abnormality (55.9%) followed by hypercholesterolemia (47.6%), elevated LDL (37.2%) and reduced HDL (35.2%) (Table 4.9).

Tables 4.8 shows mean TC levels of diabetic respondents with CVDs was higher than those without CVDs; however, the difference was not statistically significant (p=0.109). The mean TG level of respondents with CVDs was significantly higher than those without CVDs (p=0.005) (Table 4.8). Table 4.9 shows CVD was more prevalent among diabetics with high TG levels.

The mean HDL-C of the respondents with CVDs was significantly lower than those without CVDs (p=0.007). Table 4.9 shows more diabetic respondents with low HDL-C levels had CVDs. The mean LDL-C of the respondents with CVDs was significantly higher than those without CVDs (p=0.005). Table 4.9 reveals more diabetics with high LDL-C levels had CVDs.

Table 4.8 shows 18.6% of the diabetic respondents had high creatinine levels. The mean creatinine level of respondents with CVDs was not significantly higher than those without CVDs (p=0.146) (Table 4.9).

| Table 4.6 Anthropometric Data of Diabetic Respondents with and without CVDs |             |             |           |         |  |
|---|-------------|-------------|-----------|---------|--|
| Anthropometric Data   | All         | Diabetics   | Diabetics | P-value |  |
|   | Respondents | without CVD | with CVD  |         |  |
|   | No. (%)     | N=48 (%)    | N=97(%)   | -       |  |
| BMI Ranks   | 7           |             |           | 0.605   |  |
| Underweight; BMI < 18.5 kg/m <sup>2</sup>                                   | 2(1.4)      | 1 (0.7)     | 1 (0.7)   |         |  |
| Normal, BMI 18.5 to 24.9 kg/m <sup>2</sup>                                  | 48(33.1)    | 19(13.1)    | 29 (20)   |         |  |
| Above $25.0 - 29.9 \text{ kg/m}^2$ ,  | 62(42.7)    | 19 (13.1)   | 43 (29.6) |         |  |
| overweight  |             |             | 35        |         |  |
| Above 30.0 kg/m <sup>2</sup> , (obese)                                      | 33(22.8)    | 9 (6.2)     | 24 (16.6) |         |  |
| Waist Circumference   | SANE        | 1           |           | 0.000   |  |
| Less than 80 cm Normal  | 32(22.1)    | 18 (12.4)   | 14 (9.7)  |         |  |
| Between 80 - 87.9 cm overweight   | 24(16.5)    | 1 (0.7)     | 23 (15.9) |         |  |
| Above 88 cm (obese)   | 89(61.4)    | 29 (20)     | 60 (41.3) |         |  |
| Waist to Hip ratio  |             |             |           | 0.005   |  |

#### Table 4.6 Anthropometric Data of Diabetic Respondents with and without CVDs

| Less than 0.90 for male,             | 9(6.2)   | 7 (4.8)   | 2 (1.4)   |
|--------------------------------------|----------|-----------|-----------|
| <0.80 for female, (Normal)           |          |           |           |
| Between 0.90 – 0.99 for male, 0.80 – | 41(28.2) | 14 (9.7)  | 27 (18.6) |
| 0.84 for female, (overweight)        |          |           |           |
| Above 1.0 for male, 0.85 for female  | 95(65.5) | 27 (18.6) | 68 (46.9) |
| (Obese)                              |          |           |           |

 Table 4.7 Comparison of Anthropometric and Blood Pressure Data among Diabetic

 Respondents with and without CVDs

100

| Variable                  | Mean for All               | Diabetics          | Diabetics with               | P-Value |
|---------------------------|----------------------------|--------------------|------------------------------|---------|
|                           | Respondents                | without CVD        | CVD (N=97)                   |         |
|                           |                            | (N=48)             |                              |         |
| BMI (kg/m <sup>2</sup> )  | $27.39 \pm 5.12$           | $27.47 \pm 4.90$   | $27.34 \pm 5.72$             | 0.885   |
| Waist Circumference (cm)  | 92.30±15.87                | 86.31 ±14.30       | $95.26 \pm 15.84$            | 0.001   |
| Hip Circumference (cm)    | $98.75 \pm 13.81$          | $98.98 \pm 15.49$  | $98.63 \pm 12.98$            | 0.895   |
| Waist to Hip Ratio        | $0.93 \pm 0.07$            | $0.91\pm0.08$      | $0.95 \pm 0.064$             | 0.002   |
| Systolic Pressure (mmHg)  | $136.21 \pm 18.17$         | $124.17 \pm 12.69$ | $142.16\pm17.56$             | 0.000   |
| Diastolic Pressure (mmHg) | $\underline{86.29\pm9.82}$ | $78.96 \pm 9.94$   | $\underline{86.29 \pm 9.82}$ | 0.000   |
|                           |                            |                    |                              |         |

# 4.6 Oral Medications and Insulin Use

## 4.6.1 Oral Medications

The type of medications used by the diabetics were grouped as biguanides, sulfonylureas, combined oral medications treatment (which included both sulfonylureas and biguanides), insulin and insulin combined with oral medications (Table 4.10).

From Table 4.10, the most prescribed medication was combined oral diabetic medications (46.9%) and the least prescribed was insulin only (3.4%). Herbal medications was used by

19.3% of diabetics. For the CVD medications, the most prescribed was Calcium-Channel Blockers (43.29%).

| Variable              | Overall mean    | Diabetics without | Diabetics with    | P-value |
|-----------------------|-----------------|-------------------|-------------------|---------|
|                       | mmol/L          | CVDs (N=48)       | CVDs (N= 97)      |         |
|                       |                 | mmol/L            |                   |         |
| Fasting Blood Glucose | 9.92 ± 4.15     | $9.90 \pm 4.08$   | $9.98 \pm \ 4.08$ | 0.905   |
| Total Cholesterol     | $5.85 \pm 1.45$ | $5.57 \pm 1.35$   | $5.98 \pm 1.48$   | 0.109   |
| Triglycerides         | $1.95 \pm 0.91$ | $1.65 \pm 0.84$   | $2.10\pm0.91$     | 0.005   |
| HDL-C                 | $1.20 \pm 0.41$ | $1.33 \pm .042$   | $1.13\pm0.38$     | 0.007   |
| LDL-C                 | 3.83 ± 1.39     | $3.02 \pm 0.88$   | $4.23 \pm 1.43$   | 0.005   |
| Creatinine µmol/l     | 93.88 ± 30.19   | 78.96 ±9.94       | $86.29 \pm 9.82$  | 0.146   |

# Table 4.8 Biochemical Data of Respondents with and without CVD

# Table 4.9 Biochemical Data of Respondents with and without CVDs

| Variable                            | Diabetic    | Diabetics  | Diabetics with           |
|-------------------------------------|-------------|--|--------------------------|
|                                     | Respondents | without CVD  | CVD N=97 (%)             |
|                                     | N=145 (%)   | N=48 (%)   |                          |
| Fasting Blood Glucose               | 16          | 0  | 11                       |
| Below 3.60 mmol/L                   | 1(0.7)      | 1 (0.7)  | 0 (0.0)                  |
| Normal 3.60-6.40 mmol/L             | 32(22.1)    | 15 (10.4)  | 17 (11.7)                |
| Above 6.40 mmol/L                   | 112(77.2)   | 32 (22.1)  | 80 (55.2)                |
| Total Cholesterol                   |             | and I  |                          |
| Below 3.10 mmol/L                   | 1(0.7)      | 0 (0.0)  | 1 (0.7)                  |
| Normal between 3.10- 6.50 mmol/L    | 75(51.7)    | 28 (19.3)  | 47 (32.4)                |
| Above 6.50 mmol/L                   | 69(47.6)    | 20 (13.8)  | 49 (33.8)                |
| Triglyceride                        |             |  |                          |
| Between 0.30-1.70 mmol/L            | 64(44.1)    | 31 (21.4)  | 33 ( <mark>22.8</mark> ) |
| Above 1.70 mmol/L                   | 81(55.9)    | 17 (11.7)  | 64 (44.1)                |
| Low density lipoproteins-C          |             | The second secon | 5                        |
| Below 2.60 mmol/L                   | 27(18.6)    | 10 (6.9)   | 17 (11.7)                |
| Normal Between 2.60-<br>4.90 mmol/L | 64(44.1)    | 37(25.5)   | 27 (18.6)                |
| Above 4.90 mmol/L                   | 54(37.2)    | 1 (0.7)  | 53 (36.6)                |
| High density lipoproteins-C         |             |  |                          |
| Below 1.03 mmol/L                   | 51(35.2)    | 5 (3.5)  | 46 (31.7)                |
| Normal Between 1.03-1.55 mmol/L     | 73(50.4)    | 34 (23.4)  | 39 (26.9)                |
| Above 1.55 mmol/L                   | 21(14.4)    | 9 (6.2)  | 12 (8.3)                 |
| Creatinine                          |             |  |                          |
| Below 60 µmol/l                     | 8(5.5)      | 1 (0.7)  | 7 (4.8)                  |
|                                     |             |  |                          |

| Normal Between 60-120 µmol/l | 110(75.9) | 40 (27.6) | 70 (48.3) |
|------------------------------|-----------|-----------|-----------|
| Above 120 µmol/l             | 27(18.6)  | 7 (4.8)   | 20 (13.8) |

# **Table 4.10 Oral Medications and Insulin Use**

| Diabetic Medications   | Frequency (%)          |
|--|------------------------|
| Combined oral medications (Biguanides and sulphonylureas)      | 68 (46.9)              |
| Biguanides only  | 40 (27.6)              |
| Sulfonylureas only   | 8 (5.5)                |
| Insulin with oral medication                                   | 24 (16.6)              |
| Insulin only   | 5 (3.4)                |
| Herbal Medications Use   |                        |
| Herbal medications   | 28 (19.3)              |
| Cardiovascular Medications                                     | Frequency (%)          |
| Calcium-Channel Blockers                                       | 63 (43.29)             |
| Diuretics 53 (36.08) <u>Angiotensin Converting Enzyme (ACE</u> | 2) inhibitor 29(20.61) |

# 4.7 Family and Medical History

According to Table 4.11 family medical history of hypertension and medical history of diabetes, were the most prevalent with family medical history of hypertension being the more prevalent of the two. There were significant differences for family history of hypertension and family history of diabetes (p=0.001 and p=0.021) respectively between the diabetics with and without CVDs (Table 4.12).

From Table 4.13, most diabetic respondents (65.5%), had been diagnosed with diabetes for not more than 5 years and 15.2% had been diagnosed with diabetes for more than 10 years. More diabetics with longer duration of diabetes had a significantly higher incidence of CVDs (p=

0.050).

# Table 4.11 Family Medical History of Cardiovascular Diseases among All RespondentsFamily Medical HistoryFrequency (%)

WJSANE

Family History of hypertension Family History of Diabetes 68 (46.9) 67 (46.2)

#### Table 4.12 Family Medical History among Respondents with and without CVDs

| Family Medical History          | Diabetics   | Diabetics  | P-value |
|---------------------------------|-------------|------------|---------|
|                                 | without CVI | D with CVD |         |
|                                 | N=48 (%)    | N=97(%)    |         |
| Family History of Hypertension  | 16 (23.5)   | 52 (76.5)  | 0.001   |
| Family History of Diabetes      | 13 (19.4)   | 54 (80.6)  | 0.021   |
| Family History of Stroke        | 6 (22.22)   | 21 (77.8)  | 0.078   |
| Family History of Heart Failure | 0 (0%)      | 6 (100)    | 0.183   |

#### Table 4.13 Duration of Diseases among Respondents with and without CVDs

| Variable              | Diabetic               | Diabetics               | Diabetics with | P-value |
|-----------------------|------------------------|-------------------------|----------------|---------|
|                       | Respondents<br>No. (%) | without CVD<br>N=48 (%) | CVD N=97 (%)   |         |
| Duration of Diabetes  |                        |                         |                | 0.050   |
| Not more than 5 years | 95(65.5)               | 38 (26.3)               | 57 (39.3)      | /       |
| Between 6-10 years    | 32(22.0)               | 6 (4.1)                 | 26 (17.9)      | -       |
| Above 10 years        | 18(15.2)               | 4 (2.8)                 | 14 (9.6)       |         |

#### **4.8 Dietary Habits of Respondents**

Tables 4.14 and 4.15 show the dietary habits of the diabetic participants. Most diabetic respondents (62.1%) ate thrice daily. The diabetics with CVDs who ate thrice daily were more than those without CVDs. The number of times diabetic respondents ate their main meals daily did not show any significant differences (p=0.071) (Table 4.15).

The results on vegetable intake (Tables 4.14), shows most diabetic respondents (62.1%) ate vegetables thrice in a week, whilst 8.3% ate vegetables occasionally. The vegetable intake among the diabetics with CVDs was higher than those without CVDs. However, the difference was not significant (p=0.09) (Table 4.15).

Data on fruit intake (Tables 4.14 and 4.15), reveal majority of the respondents 80 (56.6%) ate fruits less than twice a week. On fruit intake, diabetics with CVDs ate fruit more than those without CVDs. Nonetheless, the difference in fruit intake between the diabetics with and without CVDs was not significant (p= 0.68).

From Table 4.14, most diabetic respondents (56.6%) had low salt intake, whilst 9.0% had high salt intake. Table 4.15 shows all the diabetic respondents with high salt intake had CVDs, whilst none of the diabetics without CVDs had high salt intake. On salt intake, diabetics without CVDs had significantly lower salt intake than those with CVDs (p= 0.00).

Table 4.14 reveals most diabetic respondents (56.6%) consumed canned foods occasionally. The diabetics with CVDs who consumed canned foods were more than those without CVDs but the difference was not statistically significant (p=0.225) (Table 4.15). Table 4.14 shows the type of oil mostly consumed by diabetics was saturated fat (82.1%). From Table 4.15, diabetics who took saturated fat had more CVDs than those without CVDs, but this did not show any significant differences (p=0.460).

| <b>Frequency</b> (N=145) (%) |
|------------------------------|
|                              |
| 90 (62.1)                    |
| 53 (36.6)                    |
| 2 (1.4)                      |
| 100                          |
| 78 (53.8)                    |
| 55 (37.9)                    |
| 12 (8.3)                     |
|                              |
| 33 (22.8)                    |
| 82 (56.6)                    |
| 30 (20.7)                    |
|                              |

| Low                                 | 82 (56.6)  |
|-------------------------------------|------------|
| Moderate                            | 50 (34.5)  |
| High                                | 13 (9.0)   |
| Canned Foods Intake                 |            |
| Daily                               | 15 (10.3)  |
| Weekly                              | 22 (15.2)  |
| Occasionally                        | 108 (74.5) |
| Type of oil Used                    |            |
| Vegetable oil (unsaturated fat)     | 26 (17.9)  |
| Non-vegetable oil (unsaturated fat) | 119 (82.1) |

### 4.9 Participants' Physical Activity Levels and Lifestyle

Tables 4.16 and 4.17 show the lifestyle and physical activity levels of diabetic respondents. The greater majority of the diabetics participants (94.5%) live a sedentary lifestyle (by virtue of; time spent sitting/reclining per day, time spent walking and lack of moderate physical activity at work). On physical activity 61.4% of diabetics did not exercise. Diabetics without CVDs had significantly higher physical activity (p=0.00) (Table 4.17).

Smoking status, showed most diabetics (91%) had never smoked whilst (9%) were ex-smokers (Table 4.16). Ex-smokers were more prevalent among diabetics with CVDs than those without CVDs (Table 4.17). From Table 4.18, significantly higher proportion of males were exsmokers (p=0.00). Moreover, significantly higher prevalence of smoking was observed among diabetics with CVDs than those without CVDs (p= 0.041) (Table 4.17). For alcohol consumption, most diabetic respondents (54.4%) had never drunk alcohol, whilst 11.7% were current drinkers. From Table 4.18, more males than females were current drinkers with significant differences (p=0.00). Alcohol consumption was more prevalent among diabetics with CVDs than those without CVDs (p= 0.041) (Table 4.17).

| Dietary Habits of Respondent    | Diabetics without CVDs<br>N=48 No. (%) | Diabetics with CVDs<br>N=97 No. (%) | P-value      |
|---------------------------------|--|-------------------------------------|--------------|
| Main Meal                       |  |                                     | 0.071        |
| Thrice                          | 26 (17.9)                              | 64 (44.2)                           |              |
| less than thrice                | 20 (13.8)                              | 33 (22.7)                           |              |
| More than thrice                | 2 (1.4)                                | 0 (0)                               |              |
|                                 |  |                                     | 0.091        |
| Vegetable Intake                |  |                                     |              |
| Thrice in a week                | 32 (22.1)                              | 46(31.7)                            |              |
| Twice and below                 | 13 (8.9)                               | 42 (28.9)                           |              |
| Occasionally                    | 3 (2.2)                                | 9(6.2)                              |              |
| Fruit Intake                    | No Maria                               |                                     | 0.6          |
|                                 |  |                                     | <b>98</b>    |
| Thrice in a week                | 11 (7.6)                               | 19 (13.1)                           |              |
| Twice and below                 | 28 (19.3)                              | 54 (37.2)                           |              |
| Occasionally                    | 9 (6.2)                                | 24 (16.6)                           |              |
| Salt Intake                     |  |                                     | <u>0.000</u> |
|                                 |  |                                     | _            |
| Low                             | 40 (27.6)                              | 42 (29.0)                           |              |
| Moderate                        | 8(5.5)                                 | 42(29.0)                            | -            |
| High                            | 0 (0.0)                                | 13 (8.9)                            |              |
| Canned Foods Intake             |  | 17                                  | 0.225        |
| Daily                           | 3(2.1)                                 | 12 (8.3)                            |              |
| Weekly                          | 5(3.4)                                 | 17 (11.7)                           |              |
| Occasionally                    | 40 (27.6)                              | 68 (46.9)                           |              |
| Type of Oil Used                | Time 1                                 |                                     | 0.460        |
| Vegetable oil / unsaturated fat | 7 (4.8)                                | 19 (13.1)                           |              |
| Non vegetable oil / saturated   | 41 (28.3)                              | 78 (53.8)                           |              |

# Table 4.15 Dietary Habits of Diabetic Respondents with and without CVDs

# Table 4.16 Lifestyle and Physical Activity Levels of All Diabetic Participants'

|                          | Frequency (%) |
|--------------------------|---------------|
| Lifestyle of Respondents | (N=145)       |
| Time Sitting             | sh'           |
| More than 30 minutes     | 137 (94.5)    |
| 30 mins                  | 8 (5.5)       |
| Exercise Type            |               |
| Vigorous intensity       | 4 (2.8)       |
| Moderate intensity       | 52 (35.9)     |
| Never                    | 89 (61.4)     |
| Walk for 10 mins         |               |
| Yes                      | 133 (91.7)    |
| No                       | 12 (8.3)      |

| Smoking Status       |           |            |
|----------------------|-----------|------------|
| Never smoked Current |           | 132 (91.0) |
| Smoker               |           | 0 (0.0)    |
| Ex-smoker            |           | 13 (9.0)   |
| Drinking Status      |           |            |
| Never drank alcohol  |           | 79 (54.5)  |
| Current drinker      | 1 2 5 1 1 | 17 (11.7)  |
| Ex-drinker           |           | 49 (33.8)  |

# Table 4.17 Diabetic Participants' Lifestyle and Physical Activity of Respondents with and without CVDs

| Lifestyle of        | Diabetics without | Diabetics with | P-value |
|---------------------|-------------------|----------------|---------|
| Respondents         | CVD No. (%)       | CVD No. (%)    |         |
| Time Spent Sitting  | N N               |                | 0.001   |
| Less than 30mins    | 7 (4.8)           | 1 (0.7)        |         |
| More than 30 mins   | 41 (28.3)         | 96 (66.2)      |         |
| Exercise Type       |                   |                | 0.000   |
| Vigorous intensity  | 4 (2.8)           | 0 (0.0)        |         |
| Moderate intensity  | 36 (24.8)         | 16 (11.1)      |         |
| Never               | 8 (5.5)           | 81 (55.8)      |         |
| Smoking Status      |                   | A la           | 0.041   |
| Never smoked        | 47 (32.4)         | 85 (58.6)      | F       |
| Ex-smoker           | 1 (0.7)           | 12 (8.3)       | 1       |
| Drinking Status     | Cox.              | A AN           | 0.748   |
| Never Drank Alcohol | 25 (17.3)         | 54 (37.2)      | 2       |
| Current drinker     | 7 (4.8)           | 10 (6.9)       |         |
| Ex-drinker          | 16 (11.1)         | 33 (22.7)      |         |

# Table 4.18 Differences in Smoking Status of Male and Female Diabetic Respondents

| Variable         | Males                 | Females              | P-value |
|------------------|-----------------------|----------------------|---------|
| Smoking Status   |                       |                      | 0.000   |
| Never smoked     | 25 (17.2)             | 107 (73.8)           | 21      |
| Ex-smoker        | 9 (6.2)               | 4 (2.8)              | 2       |
| Drinking Status  |                       | 0 1                  | 0.000   |
| Diffiking Status | ANE M                 | -                    | 0.000   |
| Never drinks     | 15 (10.3)             | 64 (44.2)            | 0.000   |
|                  | 15 (10.3)<br>12 (8.3) | 64 (44.2)<br>5 (3.4) | 0.000   |

#### 4.10 Association between CVD and Some Selected Risk Factors among Diabetic Patients

Association between CVD and selected risk factors such as gender, age, BMI, waist hip ratio, waist circumference, systolic blood pressure, diastolic blood pressure, exercise type, smoking status, alcohol intake, family medical history of hypertension, family medical history of diabetes, family medical history of stroke and family medical history of heart failure, FBS, total cholesterol, triglyceride, HDL-C, LDL-C and creatinine levels with CVD was conducted using Pearson chi-square for categorical variables, t-test for continuous variables and crosstabulated comparison of risk among respondents with CVD and those without CVD.

The analysis revealed risk factors such as gender, family history of stroke or heart failure, BMI, total cholesterol, fasting blood glucose, creatinine levels, fruit and vegetables intake and alcohol consumption had insignificant association with cardiovascular disease incidence among diabetics. Therefore these variables were removed from the binary logistic regression analysis.

The significant predictors of CVD among the DM patients were age, waist circumference, WHR, exercise, family medical history of hypertension, family medical history of diabetes, salt intake, smoking status, triglyceride levels, HDL-C and LDL-C. These were used for further analysis by binary logistic regression. The results of the bivariate analysis (Table 4.20), family history of hypertension had an odds ratio (OR) (OR=6.789, 95% CI 1.391- 33.14), exercise (OR=0.104, 95% CI 0.028 – 0.394), salt intake (OR= 0.113; 95% CI 0.022 – 0.589) and HDLC levels (OR=0.181, 95% CI 0.036-0.0912) from the regression model.

Even though individually, some of the independent variables were significantly associated with CVDs, when they were put together, some of the variables had no significant association with

CVDs.

The logistic regression co-efficient (b) from the analysis (Table 4.20) showed that individuals who exercise (either vigorous/ moderate intensity exercise) had decreased incidence of CVDs, compared to those who do not exercise, with logistic regression co-efficient of (b= -2.261) at p<0.05. Individuals with low salt intake had decreased incidence of CVDs, compared to those who had high/moderate intake with logistic regression co-efficient of (b= -2.183) at p<0.05. HDL-C levels show that individuals with normal levels had decreased incidence of CVDs, shown by logistic regression co-efficient of (b= -1.708) at p<0.05. Family medical history showed that individuals with family history of hypertension had increased risk of CVDs incidence compared to individuals without family history of hypertension with logistic regression co-efficient of (b= 1.915) at p<0.05.

| Variables                      | P-value |
|--------------------------------|---------|
| Age (yrs.)                     | 0.000   |
| Salt-Intake                    | 0.000   |
| Exercise Type                  | 0.000   |
| Family History of Diabetes     | 0.021   |
| Family History of Hypertension | 0.001   |
| Smoking Status                 | 0.041   |
| Waist-Circumference (cm)       | 0.0002  |
| Waist Hip Ratio                | 0.005   |
| Triglycerides (mmol/L)         | 0.005   |
| HDL-C (mmol/L)                 | 0.007   |
| LDL-C (mmol/L)                 | 0.005   |

| Table 4.20 Significant Risk Factors Independently Associated with CVD Incidence among |
|---|
| Diabetic Respondents with and without CVDs in Logistic Model                          |

| Variables     | b      | Odds Ratio | 95% Confidence | P-Value |
|---------------|--------|------------|----------------|---------|
|               |        | (OR)       | Interval       |         |
| Age           | 300    | 0.741      | 0.122 - 4.45   | 0.744   |
| Salt-Intake   | -2.183 | 0.113      | 0.022 - 0.589  | 0.010   |
| Exercise Type | -2.261 | 0.104      | 0.028 - 0.394  | 0.001   |

| Family History of Diabetes     | 1.420          | 4.137        | 0.925 - 18.496 | 0.063 |
|--------------------------------|----------------|--------------|----------------|-------|
| Family History of Hypertension | 1.915          | 6.789        | 1.391-33.140   | 0.018 |
| Smoking Status                 | -2.468         | 0.085        | 0.002-3.596    | 0.197 |
| Waist-Circumference            | 886            | 0.412        | 0.079 - 2.143  | 0.292 |
| Waist Hip Ratio                | -1.342         | 0.261        | 0.009 - 7.252  | 0.429 |
| Triglycerides                  | -1.225         | 0.294        | 0.075 -1.154   | 0.079 |
| HDL-C                          | -1.708         | 0.181        | 0.036-0.0912   | 0.038 |
| LDL-C                          | <u>-19.971</u> | <u>0.000</u> | 0.00           | 0.997 |
|                                |                |              |                |       |

#### **CHAPTER FIVE**

#### DISCUSSION

## **5.0 Introduction**

The results revealed that cardiovascular diseases; hypertension, stroke and heart failure were observed among diabetes patients. However, hypertension was the most prevalent. In the study, risk factors including age, central obesity, dyslipidemia, lack of exercise, family history of hypertension or diabetes, salt intake and smoking status had significant association with risk of cardiovascular diseases among diabetes patients. Other risk factors such as gender, general obesity, FBG level, creatinine level, fruit and vegetable intake, family history of stroke or heart failure and alcohol consumption showed no significant association with cardiovascular diseases among diabetes patients.

#### 5.1 Baseline Characteristics of Participants

In this study, data on sociodemographic characteristics (Tables 4.1) revealed, majority (76.6%) of the respondents were females. Correspondingly, similar results of 70.3% females with diabetes was observed by Acheampong (2010) in Ghana. Further similar observations of high proportion of females with diabetes were also made by Wild *et al.* (2004), Titty (2009), Adibe *et al.* (2009) and Cunningham-Myrie *et al.* (2013). This result does not mean that majority of diabetic patients are females but it could be that the females are more concerned of their diabetic

status and may seek medical treatment regularly. Another reason for this observation could be that at the time of the study more females were due for their routine hospital attendance than the males.

The high proportion of female diabetics in this study could be linked to the high incidence of obesity among women observed in the study. Also, women have smaller muscle mass available for the uptake of the glucose than men (Faerch *et al.*, 2010).

High levels of estrogen and progesterone found in females, both of which can reduce insulin sensitivity among females and can also genetically predispose them to diabetes (Franconi *et al.*, 2012). Furthermore, more elderly women than men in most populations could be a contributing factor for this observation (Wild *et al.*, 2004). The significantly high prevalence of diabetes in women in the study is in contrast with data from India by Chow *et al.* (2006) and in China by Yang *et al.* (2010) in which diabetes was more predominant in men than women.

Data on age (Tables 4.1 and 4.2) showed, majority of the respondents with CVDs were above 40 years and this is similar to the finding of Fourlanos *et al.* (2008) and Okereke *et al.* (2008). The results of the study showed a directly proportional association between increasing age and prevalence of diabetes and CVDs among the study subjects, with participants between 51-60 years being the most affected. The increasing prevalence of DM with age, particularly after 40 years observed in this study is in agreement with results from India by Kokiwar *et al.* (2012) and Raghupathy *et al.* (2007). This supports ageing to be a risk factor for diabetes in Africa, which is in line with predictions by Hall *et al.* (2011) and Guariguata *et al.* (2011) that, prevalence of diabetes will shift from the elderly (>65 years) to younger age-groups. In subSaharan Africa, prevalence of chronic diseases such as diabetes and CVDs increase with age (Baldé *et al.*, 2007; Motala *et al.*, 2008). Age is the most powerful unmodifiable risk factor

for atherosclerosis (Jousilahti *et al.*, 1999). As age increases above 45 years the risk of cardiovascular event increases. This is because accumulated risk factors in an individual increase with age (Jousilahti *et al.*, 1999).

The high incidence of diabetes and CVDs among the aged is consistent with lower physical activity, obesity and adoption of unhealthy lifestyles among diabetics, as reported by Kokiwar *et al.* (2012). This shift in age range has deleterious consequences on national economies, hence efforts geared towards addressing this problem will yield economic and public health benefits.

The educational status showed majority of diabetic patients were primary school drop-outs (Tables 4.1). The findings on educational status concur with studies made by Abebe *et al.* (2015), in Turkey, Berhe *et al.* (2014) in Pakistan in which majority of DM patients were found to have only primary school educational level. The presence of large proportion of diabetics who have lower level of education may also reflect a large number of poorly educated people among the study population, hence the level of education should be taken into consideration during nutritional counselling. Although most participants were employed, majority were in the low income class which is similar to results by Gezawa *et al.* (2015), in North-Eastern Nigeria and Cunningham-Myrie *et al.* (2013) in Australia. The low income levels among respondents, as high educational level is associated with better income status.

# **5.2 Anthropometric Data of Respondents**

Anthropometry data (Table 4.6) revealed, majority of the participants were either overweight (42.7%) and obese (22.8%). The anthropometric data showed no association of cardiovascular diseases with BMI. Rahmanian *et al.* (2014) reported similar prevalence of overweight and obesity among diabetic patients in Iran with a prevalence of overweight and obesity as 48%

BADW

and 28% respectively. Al-Sharafi and Gunaid (2014) also showed similar findings among diabetic patients in Yemen, as 58.5% were overweight and 28.8% were obese. In agreement with this study, a study by Pandeya *et al.* (2012) showed that 39.0% and 11.0% of the diabetics to be overweight and obese, respectively. The high proportion of respondents being overweight and obese could be due to non-modifiable factors such as ageing and family history of obesity and also influenced by modifiable factors such as poor diet, sedentary lifestyle of respondents, lack of emphasis on the management of obesity during dietary counselling among diabetics and the use of medications, such as sulphonylureas and insulin (Connor *et al.*, 2003). BMI or obesity increases with age, as reported by Lobstein *et al.* (2004) so the high overall mean BMI of the study participants could be because all respondents were adults above 18 years.

However, the findings on BMI was in marked contrast with the overweight and obesity prevalence reported by Thomas *et al.* (2006), Joseph *et al.* (2004), Shera *et al.* (2004), Esmaillzadeh *et al.* (2007) and Daousi *et al.* (2006). The variations in prevalence of overweight and obesity among diabetics in various studies could be due to factors like environmental differences, lifestyle, employment status and genetic factors of the population (Connor *et al.*, 2003).

Among the respondents overweight and obesity was more prevalent among females than males (Table 4.3). This concurs with a study by Basukala *et al.* (2014) which showed that obesity was found more prevalent among women than men. One reason for this difference could be attributed to sex hormones. In males, testosterone is responsible for the high muscle mass-tofat mass ratio (Powers and Howley, 2007) whereas oestrogen is responsible for more fat distribution in females (Powers and Howley, 2007). Furthermore, the high prevalence of

obesity among African females could reflect their belief that overweight and obesity indicate health status and the wealth of their spouses (Puoane *et al.*, 2005).

Similar to our result, most studies showed higher frequency of overweight and obesity in diabetic women, compared to men. Similarly, the high rate of overweight and obesity found among Ghanaian females in our study was consistent with the findings of Kruger *et al.* (2005) and Croft *et al.* (1995) which stated that anthropometric measures were higher in AfricanAmerican females, compared to Caucasian females.

BMI does not factor the proportion of weight related to muscle or fat distribution in the body. Individuals with similar BMI can vary considerably in their abdominal-fat mass, with premenopausal women characteristically having more abdominal-fat mass than men (Lemieux *et al.*, 1994). Furthermore, BMI has considerable limitations in predicting intra-abdominal fat accumulation (Chen *et al.*, 2000). For this reason data on WHR and waist circumference were also determined, to ascertain CVD risk factors and its correlations with BMI.

The prevalence of abdominal obesity in this study was 61.4% and 65.5% for waist circumference and waist to hip ratio respectively (Table 4.6). Data on waist circumference and waist to hip ratio showed that majority of respondents had abdominal obesity. This means, central obesity as per waist-hip ratio and waist circumference was more prevalent than general obesity in diabetics. The anthropometric data showed significant association of cardiovascular diseases with central obesity. This findings is in agreement with findings of Raimi *et al.* (2015), Ahaneku *et al.* (2011), Ulasi *et al.* (2010). The finding that central obesity was more prevalent than general obesity in diabetics was consistent with study by RezaDerakhshan and Asghar (2010) and Al-Nozha *et al.* (2007) in which 58.2% and 66.5% type 2 diabetics respectively had abdominal obesity.

The prevalence of abdominal obesity (Table 4.6) was however, higher than results from Nigeria by Siminialayi *et al.* (2008) as 31.7% and Sodjinou *et al.* (2008) as 32.0%. The prevalence of abdominal obesity was significantly higher in female than in male respondents, which is in agreement with several studies in other African countries, by Maher *et al.* (2011), Shayo and Mugushi *et al.* (2011) and Wahab *et al.* (2011). This finding may be attributed to low levels of physical activity among females and the notion that being obese is considered as a sign of wellbeing in Africa (Ojofeitimi *et al.*, 2007).

Abdominal obesity was found to be linked with increased risk of CVD among diabetics in the study. Abdominal obesity is connected to insulin resistance and abnormal blood lipid levels (Ness-Abramof and Apovian, 2008). This results in accumulation of pro-inflammatory cytokines and C-reactive protein and reduced adiponectin levels. The combined effects of these metabolic abnormalities thus lead to a higher cardiometabolic risk and subsequently cardiovascular disease (Ness-Abramof and Apovian, 2008; Dinarello, 2000). Ho *et al.* (2001), in a comparative study of several anthropometric measures, proved waist circumference was effective in identifying CVD risk factors.

### 5.3 Dyslipidemia

Studies have shown categorically that lipid abnormalities in patients with diabetes is associated with increased risk of cardiovascular disease (Goldberg, 2001; Krauss, 2004). Findings from this study in Table 4.9, show prevalence of hypercholesterolemia, hypertriglyceridemia, low HDL-C, and high LDL-C levels which are well known risk factors for cardiovascular diseases among diabetic patients. Table 4.9 shows that 47.6% and 55.9% of the diabetic patients had high total cholesterol and high triglyceride levels, putting them in the high risk for cardiovascular disease. The results show that dyslipidemia is more severe and prevalent among

diabetic patients with CVD than diabetic patients without CVD. Diabetics with CVDs had significantly higher triglyceride levels, lower HDL-C and higher LDL-C levels (Table 4.8). The pattern of dyslipidemia observed in the present study was similar to reports by AbdulRamman and Olufunsho, (1995) in Saudi Arabia, where dyslipidemia was observed in the range of 25-60%, among diabetic patients.

The results on prevalence of dyslipidemia in the present study was also consistent with results of Sawant *et al.* (2008) in India, which revealed the prevalence of hypercholesterolemia, hypertriglyceridemia, abnormally high levels of LDL-C and low HDL-C levels among diabetics. The elevated levels of LDL in this study was also observed by Saaddine *et al.* (2002) in USA and in Jordan by Abdel-Aal *et al.* (2008) among diabetics. A study in Nigeria showed similar findings, where hypercholesterolemia and hypertriglyceridemia were 43.5% and 34.8% respectively, among DM patients. The prevalence of hypercholesterolemia and hypertriglyceridemia in this study was consistent with results of Jayarama *et al.* (2012).

The variations in lipid abnormalities can be attributed to different lifestyles among these subjects, including diet and exercise, differences in insulin sensitivity, and other social habits. Diabetic patients can have many lipid abnormalities, including elevated levels of low density lipoprotein cholesterol (LDL-C) and triglycerides, and low levels of high- density lipoprotein cholesterol (HDL-C) (Haffner *et al.*, 1998). In diabetes, it is the impaired glucose metabolism that leads to hyperglycemia and subsequently dyslipidemia. Vascular complications and lipid abnormalities in diabetics can be prevented by controlling elevated blood glucose levels (Marcus, 2001; Lehto *et al.*, 1997).

From Table 4.9, the participants with CVDs had higher levels of LDL-C and TG compared with those without CVDs. This is consistent with the studies of Akintunde *et al.* (2015) and

Ojji *et al.* (2009) in Nigeria, where high levels of LDL-C and TG were reported in patients with hypertension. The increasing prevalence of dyslipidemia among the respondents with CVD and those without CVD in this study could be attributed to urbanization, adoption of western diet and differences in lifestyle, characterized by physical inactivity among diabetics, as reported by Kokiwar *et al.* (2012). Improving the lipid profile among diabetics is important, as it is reported that dyslipidemia is associated with CVD (National Institutes of Health, 2001).

## **5.4 Creatinine**

Plasma creatinine and urea have been reported as potent markers of glomerular filtration rate (GFR). However, plasma creatinine gives a more accurate measure of kidney function, compared to plasma urea level and this is attributed to the fact that creatinine fulfills the requirements for a perfect filtration marker (Perrone *et al.*, 1992). Creatinine levels are usually elevated when the kidneys have lost about 50% of their function (Perrone *et al.*, 1992). The results on creatinine levels (Table 4.9) showed no significant difference among participants with cardiovascular disease and those without cardiovascular disease. Out of the diabetic respondents studied, 18.6% had high creatinine levels (Table 4.9). This observation is in accordance with the reports by Adler *et al.* (2003), Judykay (2007) and Wagle (2010) in which high creatinine levels observed in diabetic patients indicated impaired function of the nephrons. Research by Anjaneyulu and Chopra (2004) found elevated serum creatinine levels in diabetic rats indicated progressive renal damage. Similar findings to the current study were reported by Luepker *et al.* (2003), Antman *et al.* (1996) and Wilmer *et al.* (2000) in which significantly high plasma creatinine levels were observed in diabetic patients, compared with nondiabetics.

### **5.5 Fasting Blood Glucose Levels**

Poor glycemic control was observed in most diabetic patients, even though all of them were on antidiabetic medications. The results on FBG (Table 4.9), shows no significant differences among participants with cardiovascular diseases and those without cardiovascular diseases. Some of the reasons of hyperglycemia among diabetes patients are a carbohydrate-rich local diet, physical inactivity, lack of knowledge about diabetes and inadequate knowledge on treatment protocol, as reported by Eid *et al.* (2004). Poor glycemic control status has also been reported in other studies in developing and developed countries among diabetes patients (Ismail *et al.*, 2000). A study by Ismail *et al.* (2000) in Malaysia and Rotimi *et al.* (2004) in Nigeria also documented poor glycemic control among diabetic patients.

However, the results on FBG levels contrasted with findings by Jifeng in (2006) in USA, Reisig *et al.* (2007) in Germany and Arai *et al.* (2009) in Japan, who reported good glycemic control among diabetes patients. The good glycemic control status in these countries might be because of the higher literacy levels, resulting in better knowledge about DM (Reisig *et al.*, 2007). Other factors associated with good glycemic control can be dosage of medication or insulin, compliance with management regimens, self-monitoring of blood glucose and socio-economic differences in groups, leading to greater improvements in FBG levels (Benoit *et al.*, 2005). Some of the factors that influence diabetes control and management could be socio-economic status which are also associated with access to health care, healthcare utilization, use of medication and access to good nutrition (Corpus *et al.*, 2004).

## 5.6 Prevalence of Cardiovascular Disease among DM Patients

Epidemiological data on cardiovascular disease in diabetics vary throughout the world. The prevalence rate of hypertension, stroke and heart failure in diabetic patients were 66.2%, 8.28% and 3.45% respectively (Table 4.4). The findings in this study highlight the burden of hypertension across the populations in Ghana and also corroborate the documented pattern of rise in the hypertension prevalence in diabetic patients, as 63% found by Okoro and Oyejola in

(2004) among diabetics in Nigeria. The findings in this research is also consistent with findings of other researchers who demonstrated a high prevalence of hypertension among diabetes patients (Unadike *et al.*, 2011; Pancha et *al.*, 2012; Basavegowda *et al.*, 2014) but clearly higher than those previously described in other sub-Saharan African countries by Dembele *et al.* (2000), Asekun-Olarinmoye *et al.* (2013) and Ogah *et al.* (2014). The level was lower than that of United Kingdom with hypertension prevalence of 73% and USA with a hypertension prevalence of 77%, where diet, physical inactivity and obesity have collectively caused this increased prevalence of hypertension (Kearney *et al.*, 2005).

This study's 8.28% overall prevalence of stroke (Table 4.4) is consistent with reported stroke prevalence of 8% reported in SSA and 9.3% in Nigeria by Oni *et al.* (2008). This is also consistent with findings of other researchers (Tseng *et al.*, 2004; Agyeman *et al.*, 2006), but greatly exceed that reported in Nigeria, where stroke constituted 0.36% of the total hospital admissions from 1994 to 1998 (Njoku and Aduleju, 2004).

The stroke prevalence among diabetes patients can be attributed to poor control of blood pressure, a powerful predictor of stroke and responsible for nearly 70% of all stroke cases (Lavados *et al.*, 2005; WHO, 1997; Bronner *et al.*, 1995). The prevalence of stroke among diabetes patients can be attributed to ignorance of common risk factors, non-compliance with diabetes medication and nonattendance to clinic, as reported by some of the respondents and health professionals. This correlates with reports of other studies in Africa by Mohammed *et al.* (2000), Agyemang et *al.* (2006) and Van der Sande *et al.* (2000).

The present study showed a heart failure prevalence of 3.45% among diabetes patients (Table 4.4). The prevalence of heart failure (HF) can be attributed to the high prevalence of hypertension observed among the diabetic respondents. The causes of heart failure in SSA are

mostly hypertension, valvular heart diseases and various cardiomyopathies among adults (Sliwa *et al.*, 2005; Commerford and Mayosi, 2006). In diabetics, risk factors that partly account for the incidence of HF include diabetic-specific cardiomyopathy, accelerated coronary atherosclerosis and other diabetes-related risk factors (Adlerberth *et al.*, 1998). Nevertheless, factors such as dyslipidemia, hypertension, anemia from malnutrition, hypercoagulability, obesity and inflammation in people with diabetes, increase the risk of experiencing cardiovascular complications (Stamler *et al.*, 1993; Steyn *et al.*, 2005). Pathological changes in the myocardium of diabetics can lead to development of heart failure linked with sustained hyperglycemia, impaired insulin resistance and accumulation of collagen and other glycation end-products (Bell, 1995).

In patients with diabetes the prevalence of heart failure is between 9–22%, which is much higher, compared to the general population Adriaan *et al.* (2011). The result of the study was contrary to 19% diabetics with heart failure patients by Amoah and Kallen (2000). It is lower than the prevalence of heart failure among diabetics, reported by Bauters *et al.* (2003), Garty *et al.* (2007) in Israel and Ola *et al.* (2006) in Nigeria. This study shows low prevalence of HF among patients with diabetes in Ghana, compared to other regions in Africa. One of the reasons for the reported low prevalence of HF may be due to a lack of diagnostic facilities, as these are available only in a few urban health centers (Kengne *et al.*, 2005).

# 5.7 Dietary Habits of Respondents

Dietary patterns signify the combination of foods habitually consumed, which together produce synergistic health effects (Mozaffarian *et al.*, 2011; Perrin *et al.*, 2002). For the many risk factors associated with high blood pressure (HBP), the dietary exposure most investigated has been daily salt consumption.

The data on salt intake (Table 4.14) revealed that salt intake among respondents was low. The low intake of sodium among most respondents is likely due to the high prevalence of hypertension among the diabetes patients. Dietary sodium restriction (to <3 grams/day) is an important component of management of any patient with hypertension (Uzu *et al.*, 2006; Sacks *et al.*, 2001; Hoffmann and Cubeddu, 2007).

Excess sodium intake contributes to hypertension directly, by increasing intravascular volume, and indirectly, by blunting the effectiveness of antihypertensive drugs (Ekinci *et al.*, 2011; Pimenta *et al.*, 2009). Subsequently, clinical guidelines recommend a reduced intake of salt as a measure to maintain a blood pressure at or below target levels in patients with diabetes (Elliott *et al.*, 1996; Vedovato *et al.*, 2004; Provenzano *et al.*, 2014).

It has been documented that a reduced sodium intake can prevent hypertension (Huang *et al.*, 1998) and lower BP in patients on antihypertensive medication (Weir *et al.*, 1997; Appel *et al.*, 2001) and can facilitate hypertension control. The data on salt intake (Table 4.15) shows significant differences in salt intake among respondents with cardiovascular disease and those without cardiovascular disease. This indicates that sodium intake is associated with the incidence of cardiovascular disease among diabetes patients. Meneton *et al.* (2005) stated that strong relationship between high salt intake and high blood pressure in human and animal models.

The finding of low salt intake observed among diabetics is consistent with reviews that reported reduced sodium intake decreased blood pressure in adults (Graudal *et al.*, 2012; Dickinson *et al.*, 2006; Dietary Guidelines Advisory Committee, 2010). However, the low sodium intake among diabetics in this study was contrary to high salt intake observed by Provenzano *et al.* 

(2014) in Pennsylvania. This difference is likely due to concerns by other researchers that a reduction in sodium intake might lead to adverse health effects such as increased total cholesterol, low density lipoprotein cholesterol, triglycerides and catecholamine levels, as well as adverse changes in renal function or adverse effects on cardiovascular risk (Alderman and Cohen, 2012).

#### **5.8 Fruit and Vegetable Intake**

Results on fruit and vegetable intake (Table 4.14) reveal inadequate fruit and vegetables intake, compared to the 9 servings/day of fruit and vegetables, provided in a DASH diet. As the majority of respondents consume vegetables thrice or below in a week. The fruit and vegetable intake among respondents with CVD and without CVD showed no significant differences.

A number of compounds may contribute to the cardioprotective effects of fruit and vegetables, including vitamin C, folate, potassium, fiber and phytochemicals (Bazzano *et al.*, 2003). The insufficient intake of fruit and vegetables among respondents is linked with several socioeconomic, demographic, nutritional knowledge, personal and environmental factors (Rasmussen *et al.*, 2006; Bonomo *et al.*, 2003). Other factors responsible for the low consumption of fruit and vegetables, are related to the availability, accessibility, cost and quality of the food (Cummins *et al.*, 2009). High fruit and vegetable intakes are related to a healthy dietary pattern (Hu *et al.*, 1999; Hu *et al.*, 2000).

The results of the study are similar to the results of Hall *et al.* (2009) in which 78.0% of respondents from mainly low-and middle-income countries consumed less than the recommended five daily servings of fruits and vegetables, with Ghana, Bangladesh, Ukraine and Malawi, recording outstandingly low fruits and vegetables consumption (Hall *et al.*, 2009).

The low intake of fruits and vegetables in the current study is similar to a survey in South Africa by Hall *et al.* (2011), Appleton *et al.* (2009) in Northern Ireland and Health Survey in England in which low fruit and vegetable consumption was observed among diabetics. Studies have confirmed that frequent fruit and vegetable consumption was associated with a 6–22% lower risk for coronary heart disease mortality (Van't Veer *et al.*, 2000).

A meta-analysis that combined the results of 11 prospective cohort studies found that people in the 90<sup>th</sup> percentile of fruit and vegetable intake (about 5 servings/day or more) had a 15% lower risk of cardiovascular diseases than those in the 10<sup>th</sup> percentile of intake (Law and Morris, 1998). Studies have shown that consuming more than five daily servings of fruit and vegetables is associated with 17% reduction in risk of cardiovascular disease, compared with consumption of less than three servings daily (He *et al.*, 2007). Fruit and vegetable consumption varies considerably among and within countries, in large part reflecting, the prevailing economic, cultural and agricultural environments, but consumption in many parts of the world remains low (Hall *et al.*, 2009).

# 5.9 Lifestyle of Respondents

The results on lifestyle (Table 4.16) from the study revealed, none of the diabetes patients was currently smoking, which can be attributed to low prevalence of smoking in Ghana, as reported by Pampel (2008). Another likely reason for this can be due to the ban on public smoking in Ghana and also health workers advising patients to desist from smoking. Cultural factors may also have had a strong influence on the pattern of smoking in Ghana. In reality, the low smoking prevalence in Ghana reflects a mix of cultural and political influences (Addo *et al.*, 2008). There is also likelihood that patients will not admit smoking even if they smoked and this could underestimate the effect of smoking.

The low number of female ex-smokers, compared to their male counterparts in our study is a typical finding in African countries (Pampel, 2008; Rudatsikira *et al.*, 2007). This has been attributed to limited opportunities to smoke, coupled with low levels of economic independence among women and sociocultural contexts within which smoking among women is often considered to be immoral (Rudatsikira *et al.*, 2007). Smoking is generally five times more prevalent among males than females; in Nigeria smoking status was found to be a significant risk factor CVDs in males only (Ekpenyong *et al.*, 2012).

However, findings of the study is contrary to the prevalence of cigarette smoking among diabetic patients in Pakistan reported by Khalid *et al.* (2014) and World Bank (2009) as 27% and 34.47% respectively. Contrarily, cigarette smoking in this study was low, compared with 17% in Los Angeles by Johnson (2001) and 12.4% European by Scemama *et al.* (2006) among diabetics. This difference in smoking prevalence is likely due to the lifestyle of the society, cultural differences and health education given by the health professionals. However, being an ex-smoker had significant relationship with risk of cardiovascular disease among diabetes patients.

11 Casto

Smoking accelerates the ageing process and hastens death according to Wellman and Kamp (2008). Smoking has a negative impact on health status, in addition to risks of lung cancer. Smoking is a risk factor for cardiovascular morbidity, mortality and the development of myocardial infarction (Peters *et al.*, 2008). Smoking is linked with deterioration in metabolic control in diabetic patients (Bott *et al.*, 1994) which is associated with an increased risk for development of macrovascular complications (Morrish *et al.*, 1991). Data on lifestyle of smoking (Table 4.17) with p-value of 0.041 showed, that smoking was significantly associated with the CVD incidence among diabetes patients. This is because nicotine intake as result of

smoking, decreases insulin sensitivity, directly or indirectly increases circulating free fatty acid levels, and also affects the autonomic nervous system and this is an additional negative factor for the insulin-mediated glucose uptake which are implicated in cardiovascular disease progression (Bergman and Ader, 2000; Kirschbaum *et al.*, 1992; Lucini *et al.*, 1996).

The prevalence of cigarette smoking among diabetic patients in this survey could not be compared with values from most countries of sub-Saharan African regions because most African countries have not conducted studies in this regard to establish the base line prevalence rate.

#### **5.10 Physical Activity Levels**

The results of the study from Table 4.16, show the physical activity level of the participants was predominantly low as 61.4% did not exercise, whilst 35.9% and 2.8% of the participants engaged in moderate and vigorous intensity exercise respectively. The results above concur to the findings of Oguntibeju *et al.* (2012), in a study in Nigeria in which 62% of the patients showed low physical activity levels, while 34% and 4% of them had moderate and high physical activity levels, respectively. The study findings are also consistent with findings by Moeini *et al.* (2011) in a study in Persia, where 64.9% of the patients did light-intensity or no physical activity. However, 35.1% in this study who engaged in moderate intensity exercise is low, compared to 59%, 52.5% and 64.4% reported by Qidwai and Azam (2004), Arcury *et al.* (2006) and Serour *et al.* (2007), respectively who had moderate physical activity. The study revealed that 94.5% of the respondents reported sedentary leisure time, which is in line with a study by Forghani *et al.* (2000), in which 90% of diabetic women reported sedentary leisure time. Prevalence of physical inactivity among the respondents in this study as 94.5% was higher than

(76.7%) physical inactivity reported by the Ghana STEPS Survey and 34.71% in Vietnam by Pham *et al.* (2009).

The low physical activity levels among diabetes patients is because those with diabetes often have physical disabilities (Gregg *et al.*, 2000), perceive discomfort when exercising or have decreased exercise capacity, compared to non-diabetic patients (Le Brasseur and Ruderman, 2005). Furthermore, the perception that diabetes 'weakened' and 'aged' the body appeared to have a de-motivational effect as far as physical activity was concerned (Lawton *et al.*, 2005). Similarly, decline in physical activity can be attributed to aging, as majority of the respondents were above 40 years, as documented in another study among middle-aged adults by Norman *et al.* (2002).

Physical activity levels showed significant relationship with incidence of CVD among diabetes patients in this study (Table 4.17). The results showed that CVD incidence was absent in those who engaged in vigorous intensity exercise, compared with those who did not exercise. This implies that exercising has an influence on CVD incidence among diabetes patients. Similar findings were reported by Manson *et al.* (1991) among postmenopausal women as walking briskly was associated with a 30% reduction in cardiovascular events. Regular physical activity has beneficial effects on incidence CVD, through regulating body weight, enhancing insulin sensitivity and glycemic control, and reducing blood pressure, atherogenic dyslipidemia, inflammation, fibrinolysis and endothelial dysfunction (Ross, 1999). Without a doubt, physical activity has been reported to improve the metabolic and cardiovascular risk of sedentary individuals (Swartz *et al.*, 2003, Murphy *et al.*, 2002). Studies by Gregg *et al.* (2003) and Hu *et al.* (2001) have shown walking to reduce CVD incidence or CVD mortality among persons with diabetes.

### **5.11 Alcohol Consumption**

Table 4.16 shows 11.7% of the diabetic respondents were current drinkers, which is lower than the 32.5% and 30.5% in the general population reported by GSS (2009) and Ghana Health and ICF Macro Survey (2006) respectively. Alcohol consumption in the study is similar to studies in Los Angeles by Johnson (2001) which showed that alcohol consumption among diabetic patients was 18%. However, the observation from this study is different from alcohol consumption of 50.8% among diabetes patients in USA by Ahmed *et al.* (2006).

The rates of current drinking among diabetics in our study were lower than rates of current drinking in the general population (National Institute on Alcohol Abuse and Alcoholism, 2004). This curtailed alcohol consumption among diabetics observed in this study is likely due to the older age and declining health of respondents (Chartier and Caetano, 2009). Another reason may be due to the perceived dangers associated with alcohol abuse on medical therapy and its influence on disease progression and physicians' advice to limit alcohol intake (Zins *et al.*, 1999; Poikotainen *et al.*, 1996).

Majority of current alcohol drinkers in the study were male respondents. This is consistent with GSS (2009) findings, where 36.7% of men consume alcohol, compared to 17.5% of women. Peltzer and Ramlagan (2009), in their study also had similar findings in Mexico (77% males, 44% females) and Namibia (61% males, 47% females) that males consume more alcohol than females. In Western Africa, high alcohol abstinence has been reported among women in Senegal (97.7%), Mali (95.8%), and Ghana (63.0%) (Martinez *et al.*, 2011). The high intake of alcohol among male respondents can be attributed to the fact that in sub-Saharan Africa, alcohol

is seen as a symbol of power, hence considered something for males and traditionally not consumed by women (Willis, 2002).

However, alcohol consumption showed no significant relationship with CVD among diabetics. This can be due to the fact that moderate alcohol consumption is associated with increased insulin sensitivity (Facchini *et al.*, 1994; Kiechl *et al.*, 1996), hence reduction in the progression of diabetes complications. Alcohol consumption has been associated with a lower risk of CVD in individuals who are light to moderate drinkers (Mukamal *et al.*, 2010). Moderate alcohol intake is associated with increases in the level of HDL-C which is associated with reduced risk of cardiovascular disease (Cooper *et al.*, 2004) by consuming two drinks a day for men and one drink a day for women. There is a significant decrease in cardiovascular risk, due to the ability of alcohol to raise HDL and reduce fibrinogen (Mahan and Escott-Stump, 2008).

# 5.12 Blood Pressure

From Table 4.7, most diabetic respondents, had high blood pressure readings above 120/80 mmHg. This implies that the majority of diabetic patients do not have control of their BP. Blood pressure in patients with diabetes is known to be 1.5–3 times higher than in non-diabetics (Pacheco-Alvarez *et al.*, 2002).

The high blood pressure prevalence among diabetics in our study as 49.7% was lower than 64.1% diabetics with high blood pressure reported by Gudina *et al.* (2011) in Ethiopia. The proportion of diabetes patients (50.3%) who attained optimal blood pressure control was higher than 21% reported by Gu *et al.* (2013) in sub-Sahara Africa and 17% reported by Chineye *et al.* (2012) in Nigeria. This means compared with other sub-Sahara Africa countries, diabetics in Ghana had better control of their BP. Arterial hypertension is present in more than 60% of DM patients (Nilsson *et al.*, 2011). This is directly linked to increased renin-

angiotensinaldosterone system activity; hyperinsulinemia associated with increased renal reabsorption of sodium; and increased sympathetic tone (Narkiewicz *et al.*, 2009).

### **5.13 Diabetes Medications**

According to Table 4.10, most respondents were on the combined oral medication (sulfonylureas and biguanides (mostly metformin), representing 46.9%. This may be ascribed to the poor glycemic control observed among diabetes patients in the study. Evidence suggest that combination therapy, using oral antidiabetic agents with different mechanisms of action is more effective in achieving and maintaining target blood glucose levels, as shown by poor glycemic control among respondents (CDA 2008, NIHCE 2005 and Weissman *et al.*, 2006). A combination of oral antidiabetic drugs is recommended if patients do not achieve a HbA1c level lower than 6.5% with monotherapy (CDA 2008, NIHCE 2005; Weissman *et al.*, 2006).

The majority of patients need multiple therapies to attain these glycemic target levels in the longer term. The fact that metformin combined with other oral medication were the most prescribed drugs complies with its endorsement as the preferred antidiabetic agent by current clinical guidelines (CDA 2008, NIHCE 2005; Weissman *et al.*, 2006). Metformin combined oral medication was the commonly prescribed anti-diabetic drug observed in the present study, which is in line with findings of Upadhyay *et al.* in (2007) and Johnson in (2006). This contrasts with the report of Sudha *et al.* (2008) that showed metformin as the most prescribed anti-diabetic medication.

## 5.14 Herbal Medications Usage among Diabetic Respondents

Table 4.10 reveals that 19.3% of those interviewed admitted using herbal remedies as part of their management of diabetes, whilst 80.7% admitted never to have used herbal medications as part of treatment. This finding in our study is consistent with 12.4% use of herbal remedies

observed by Mwangi and Gitonga in (2014). The results of herbal medication use of the study was however, lower than 65% reported by Dannemann *et al.* (2008) in Germany, 35.5% reported by Hasan *et al.* (2009) in Malaysia, 41.0% reported by Ceylan *et al.* (2009) in Turkey, among diabetics. However, herbal medication had no influence on the incidence of CVD among diabetes patients. Despite insufficient data on herbal medications safety and effectiveness, the fact remains that people with diabetes do and will continue to use herbal medications (Bastaki, 2005). The reason for the use of herbal medications among diabetics can be attributed to unwanted side effects such as allergic reactions, nutrients and drug-nutrient interactions, resulting in long-term adverse effects, arising from the use of conventional treatments (Palmer and Howland, 2001; Palmer and Betz, 2002; Halat and Dennehy, 2003).

#### CHAPTER SIX

### **CONCLUSION AND RECOMMENDATIONS**

## 6.1 Conclusion

In conclusion, the prevalence rates of cardiovascular diseases; namely; hypertension, stroke and heart failure among diabetic patients were 66.2%, 8.28% and 3.45% respectively. Anthropometric data revealed prevalence of overweight and obesity as 42.8% and 22.8%, respectively, with central obesity prevalence of 61.38% and 65.5% for waist circumference and waist to hip ratio respectively. Dyslipidemia among diabetes patients showed prevalence of hypercholesterolemia, hypertriglyceridemia, low HDL-C and high LDL-C levels as 47.58%, 55.9%, 35.2% and 36.8%, respectively. Results on creatinine levels among diabetes patients revealed 18.6% had high creatinine levels.

Dietary habits among respondents showed inadequate vegetable and fruits intake among diabetes patients, low salt intake, high intake of saturated fats with no significant association of dietary intake with cardiovascular diseases (p>0.05) except salt intake which showed

SANE

significant association with cardiovascular disease incidence (p<0.05). The physical activity levels of participants revealed low levels of physical activity, as majority (61.4%) did not exercise, with significant association of exercise with cardiovascular disease incidence (p<0.05) among diabetics. Lifestyle of the respondents revealed most (91.0%) diabetics had never smoked before with few (9.0%) being ex-smokers, with significant association of smoking status with cardiovascular disease incidence (p<0.05) among diabetics. On alcohol consumption, of the subjects, 11.7% were currently drinking whilst 54.4% had never consumed alcohol, with no significant association of alcohol consumption with cardiovascular disease incidence (p>0.05) among diabetics.

The study further showed that risk factors such as gender, family history of stroke or heart failure, BMI, total cholesterol, fasting blood glucose, creatinine levels, fruit and vegetables intake and alcohol consumption had no significant association with cardiovascular disease incidence among diabetics.

Logistic regression analysis showed that independently, risk factors such as family history of hypertension, exercise, low salt intake and HDL-C levels are the only factors that had significant association with CVD among diabetes patients with p-values < 0.05.

# 6.2 Recommendations

In the light of the findings of this study, the following recommendations are made: Further studies using cardiac profile assays, genetic analysis and other nutritional biomarkers such as sodium, potassium should be conducted to identify risk factors significantly associated with CVDs among diabetics. Further studies using cohort studies on the predictive effects of total cholesterol, triglycerides and LDL should be conducted to identify the lipid profile parameters significantly associated with CVDs among diabetics.

Nutritional intervention for diabetes should emphasize weight management in order to prevent the development of CVDs among diabetics.

Routine public health education on the adequate consumption of fruits and vegetables should be organized by Regional/District health Directorates as the study showed inadequate consumption of fruits and vegetables.

Periodic monitoring of diabetic patients lipid profile, especially HDL-C levels should be integrated into the diabetes management program, to offset the incidence of CVDs.

The Ghana Health Service should consider establishing health facility-based CVD screening programs among diabetics, especially among those with family history of hypertension.

Studies should be conducted on the usefulness of nutritional counselling and education in nutritional management of diabetes patients.

Health care practitioners, especially doctors and dieticians should be encouraged to intensify diet prescription and lifestyle modification for individuals with CVDs especially among diabetics, to help them limit or avoid certain foods.

## **6.3 Limitations and Further Research**

There were several limitations to the current study:

No cause-effect relationship can be inferred from cross-sectional data. The cross sectional design of the study may underestimate the true effects, as data obtained was from patients' ability to recall. Cohort or prospective studies are needed to identify relationships between risk factors and CVDs among diabetics.

Biases and sampling challenges occurred due to time constraints and patients not willing to consent. This might have caused challenges in establishing relationship between risk factors and CVDs.

Due to time constraint and some hospitals unwillingness to permit their facility to be used for the study we therefore used facilities which did not have diabetic or hypertensive clinics. This affected sampling and proportional distribution of cases and control in the study.

Among respondents, waist circumference was measured with their dresses on, as it was difficult to get a separate room for measurement of their waist. This is likely to cause a variation in WC measurements and WHR of respondents.

CORSULA BADY WJSAN

#### REFERENCES

- **Abbott**, C. A., Malik, R. A., van Ross, E. R., Kulkarni, J. & Boulton, A. J. (2011). Prevalence and characteristics of painful diabetic neuropathy in a large community-based diabetic population in the UK. *Diabetes Care*, *34*(10): 2220-2224.
- Abdel-Aal, N. M., Ahmad, A. T., Froelicher, E. S., Batieha, M., Hamza, M. M. & Ajlouni, K. M. (2008). Prevalence of dyslipidemia in patients with type 2 diabetes in Jordan. *Saudi Medical Journal*, 29(10): 1423-1428.
- Abdul-Ramman A.N. and Olufunsho F. (1995). Hyperlipidemia among Saudi diabetic patients-pattern and clinical characteristics. *Ann Saudi Med*, 15:240-3.
- Abebe, S. M., Berhane, Y., Worku, A., Alemu, S. & Mesfin, N. (2015). Level of sustained glycemic control and associated factors among patients with diabetes mellitus in Ethiopia: A hospital-based cross-sectional study. *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy*, 8: 65-79.
- **Abou-Seif** M. A. and Youssef A. A. (2004). Evaluation of some biochemical changes in diabetic patients. *Diabetes Medicine*, *346*:161–170.
- Abraham, W.T. (2004) Preventing cardiovascular events in patients with diabetes mellitus. *Am J Med*, 116 (supplsa): 39s-46s.
- Acheampong, A.Y. (2010). The Relation between diabetes with hypertension and other cardiovascular risk factors using logit and probit model: A thesis submitted to the department of Mathematics, KNUST in fulfilment of the requirement for the degree of Master of Philosophy in Mathematics.
- Addo, J., Smeeth, L. & Leon, D. A. (2009). Smoking patterns in Ghanaian civil servants: changes over three decades. *International Journal of Environmental Research and Public Health*, 6(1): 200-208.
- Adibe, M. O., Aguwa, C. N., Ukwe, C. V., Okonta, J. M. & Udeogaranya, O. P. (2009).
   Diabetes self-care knowledge among type 2 diabetic outpatients in south-eastern Nigeria. Int J Drug Dev Res, 1(1):85-104.
- Adler, A. I., Stevens, R. J., Manley, S. E., Bilous, R. W., Cull, C. A. & Holman, R. R. (2003). Development and progression of nephropathy in type 2 diabetes: the United Kingdom Prospective Diabetes Study (UKPDS 64). *Kidney International*, 63(1): 225-232.
- Adler, A. I., Stevens, R. J., Neil, A., Stratton, I. M., Boulton, A. J. & Holman, R. R. (2002). UKPDS 59: hyperglycemia and other potentially modifiable risk factors for peripheral vascular disease in type 2 diabetes. *Diabetes Care*, 25(5): 894-899.

- Adlerberth, A. M., Rosengren, A. & Wilhelmsen, L. (1998). Diabetes and long-term risk of mortality from coronary and other causes in middle-aged Swedish men: a general population study. *Diabetes Care*, 21(4): 539-545.
- Adriaan A., Voors G., Iwan C. C. and van der H. (2011). Education in Heart Diabetes: a driver for heart failure. *Heart*; 97: 774-780.
- Agarwal P.R., Ranka, M. and Beniwal, R. (2013). Prevalence of microvascular complications in newly diagnosed patients with type 2 diabetes. *Int J Diab Dev Countries* 21(2):2532.
- Agyemang, C., Bruijnzeels, M. A. & Owusu-Dabo, E. (2006). Factors associated with hypertension awareness, treatment, and control in Ghana, West Africa. *Journal of Human Hypertension*, 20(1): 67-71.
- Ahaneku, G. I., Osuji, C. U., Anisiuba, B. C., Ikeh, V. O., Oguejiofor, O. C. & Ahaneku, J. E. (2011). Evaluation of blood pressure and indices of obesity in a typical rural community in eastern Nigeria. *Annals of African Medicine*, 10(2): 55-72.
- Ahmed, A. T., Karter, A. J. & Liu, J. (2006). Alcohol consumption is inversely associated with adherence to diabetes self-care behaviours. *Diabetic Medicine*, 23(7): 795-802.
- Ajayi, S., Mamven, M. & Ojii, D. (2014). eGFR and chronic kidney disease stages among newly diagnosed asymptomatic hypertensives and diabetics seen in a tertiary health center in Nigeria. *Ethnicity & Disease*, 24(2): 220-225.
- Akintunde, A. A., Akintunde, T. S. & Opadijo, O. G. (2015). Knowledge of heart disease risk factors among workers in a Nigerian University: A call for concern. *Nigerian Medical Journal*, *56*(2):91-125.
- Alberti, K. G. M. M. & Zimmet, P. F. (1998). Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus. Provisional report of a WHO consultation. *Diabetic Medicine*, 15(7): 539-553.
- Alderman, M. H. & Cohen, H. W. (2012). Dietary sodium intake and cardiovascular mortality: controversy resolved? *American Journal of Hypertension*, 25(7): 727-734
- Alebiosu, C. O., Odusan, O. & Jaiyesimi, A. (2003). Morbidity in relation to stage of diabetic nephropathy in type-2 diabetic patients. *Journal of the National Medical Association*, 95(11): 1042-1092.
- Al-Nozha, M. M., Al-Hazzaa, H. M., Arafah, M. R., Al-Khadra, A., Al-Mazrou, Y. Y., AlMaatouq, M. A., ... & Al-Shahid, M. S. (2007). Prevalence of physical activity and inactivity among Saudis aged 30-70 years: a population-based cross-sectional study. *Saudi Medical Journal*, 28(4): 559-568.

- **Al-Sharafi**, B. A. & Gunaid, A. A. (2014). Prevalence of obesity in patients with type 2 diabetes mellitus in Yemen. *International Journal of Endocrinology and Metabolism*, *12*(2): e13633-e13633.
- Alwan, A. (2011). Global status report on noncommunicable diseases 2010. World Health Organization. *Am J Med*, 116 (supplsa): 39s-46s.
- American Diabetes Association (ADA). (2002). Expert committee report on diagnosis and classification of diabetes. *Diabetes Care*, 25: suppl 1:S1-14.
- American Diabetes Association (ADA). (2004). Physical activity/exercise and diabetes. *Diabetes Care*, 27: s58-80.
- American Diabetes Association (ADA). (2006). Diagnosis and classification of diabetes mellitus. *Diabetes Care*, 29(1): s43-57.
- American Diabetes Association. (2012). Standards of medical care in diabetes--2012. *Diabetes Care*, 35, S11-30.
- American Heart Association. (2007). The heart of diabetes. Available at: http://www. americanheart.org/presenter.jhtml?identifier=3044762. Accessed December 3.
- Ammari, F. (2004). Long-term complications of type 1 diabetes mellitus in the western area of Saudi Arabia. *Diabetologia Croatica*, 200(33): 59-63.
- Amoah, A. G. (2003). Obesity in adult residents of Accra, Ghana. *Ethnicity & Disease:13* (2 Suppl 2), S97-101.
- Amoah, A. G. B., & Kallen, C. (2000). Aetiology of heart failure as seen from a National Cardiac Referral Centre in Africa. *Cardiology*, 93(1-2): 11-18.
- Amoah, A. G., Owusu, S. K. & Adjei, S. (2002). Diabetes in Ghana: a community based prevalence study in Greater Accra. *Diabetes Research and Clinical Practice*, 56(3):197-205.
- Anjaneyulu, M. & Chopra, K. (2004). Quercetin, an anti-oxidant bioflavonoid, attenuates diabetic nephropathy in rats. *Clinical and Experimental Pharmacology and Physiology*, 31(4): 244-248.
- Antman, E. M., Tanasijevic, M. J., Thompson, B., Schactman, M., McCabe, C. H., Cannon, C. P., ... & Braunwald, E. (1996). Cardiac-specific troponin I levels to predict the risk of mortality in patients with acute coronary syndromes. *New England Journal of Medicine*, 335(18): 1342-1349.
- **Appel**, L. J., Espeland, M. A., Easter, L., Wilson, A. C., Folmar, S. & Lacy, C. R. (2001). Effects of reduced sodium intake on hypertension control in older individuals: results

from the Trial of Nonpharmacologic Interventions in the Elderly (TONE). *Archives of Internal Medicine*, *161*(5): 685-693.

- Appleton, K. M., McGill, R. & Woodside, J. V. (2009). Fruit and vegetable consumption in older individuals in Northern Ireland: levels and patterns. *British Journal of Nutrition*, 102(07): 949-953.
- Arai, K., Hirao, K., Matsuba, I., Takai, M., Matoba, K., Takeda, H., ... & Terauchi, Y. (2009). The status of glycemic control by general practitioners and specialists for diabetes in Japan: a cross-sectional survey of 15,652 patients with diabetes mellitus. *Diabetes Research and Clinical Practice*, 83(3): 397-401.
- Arcury, T. A., Snively, B. M., Bell, R. A., Smith, S. L., Stafford, J. M., Wetmore-Arkader, L. K. & Quandt, S. A. (2006). Physical activity among rural older adults with diabetes. *The Journal of Rural Health*, 22(2): 164-168.
- Arora, M., Koley, S., Gupta, S. & Sandhu, J. S. (2007). A study on lipid profile and body fat in patients with diabetes mellitus. *Anthropologist*, 9(4): 295-298.
- Arthur C.G., John E.H. (2000). Textbook of Medical Physiology. 10th ed. Philadelphia: WB Saunders Company, pp 657-884
- Asekun-Olarinmoye, E. O., Akinwusi, P., Adebimpe, W., Isawumi, M., Hassan, M., Olowe, O., ... & Adewole, T. (2013). Prevalence of hypertension in the rural adult population of Osun State, southwestern Nigeria. *Int J Gen Med*, 6: 317-22.
- Asia Pacific Cohort Studies Collaboration (APCSC). (2003). The effects of diabetes on the risks of major cardiovascular diseases and death in the Asia-Pacific region. *Diabetes Care*, 26(2): 360-366.
- Asia Pacific Cohort Studies Collaboration (APCSC). (2004). Body mass index and cardiovascular disease in the Asia-Pacific Region: an overview of 33 cohorts involving 310 000 participants. *International Journal of Epidemiology*, *33*(4): 751-758.
- Assmann, G. & Schulte, H. (1992). Relation of high-density lipoprotein cholesterol and triglycerides to incidence of atherosclerotic coronary artery disease (the PROCAM experience). *The American Journal of Cardiology*, 70(7): 733-737.
- Awosan, K. J., Ibrahim, M. T. O., Essien, E., Yusuf, A. A. & Okolo, A. C. (2014). Dietary pattern, lifestyle, nutrition status and prevalence of hypertension among traders in Sokoto Central market, Sokoto, Nigeria. *International Journal of Nutrition and Metabolism*, 6(1): 9-17.
- Azagba, S. & Sharaf, M. F. (2011). Disparities in the frequency of fruit and vegetable consumption by socio-demographic and lifestyle characteristics in Canada. *Nutrition journal*, *10*(1): 1-17.

- **Bakari**, A. G., Onyemelukwe, G. C., Sani, B. G., Aliyu, I. S., Hassan, S. S. & Aliyu, T. M. (1999). Prevalence of diabetes in suburban northern Nigeria: results of a public screening survey. *Diabetes International*, *9*: 56-60.
- Baldé, N. M., Diallo, I., Baldé, M. D., Barry, I. S., Kaba, L., Diallo, M. M., ... & Sangaré-Bah, M. (2007). Diabetes and impaired fasting glucose in rural and urban populations in Futa Jallon (Guinea): prevalence and associated risk factors. *Diabetes & Metabolism*, 33(2): 114-120.
- Basavegowda, M., Shankarappa, K. H., Channabasappa, A. N., Marulaiah, S. K., & Hathur, B. (2014). Magnitude and pattern of hypertension among diabetics; risk prediction for stroke and myocardial infarction. *Journal of Mahatma Gandhi Institute of Medical Sciences*, 19(1): 51-74.
- **Basta**, G., Schmidt, A. M. & De Caterina, R. (2004). Advanced glycation end products and vascular inflammation: implications for accelerated atherosclerosis in diabetes. *Cardiovascular Research*, 63(4): 582-592.
- Bastaki, A. (2005). Diabetes mellitus and its treatment. *International Journal of Diabetes and Metabolism*, *13*(3): 111-115.
- **Basukala**, A., Sharma, M., & Pandeya, A. (2014). Prevalence of overweight and obesity among patients with type 2 diabetes mellitus in Kathmandu. *Age (years)*, *36*(85.00): 57-91.
- Bauters, C., Lamblin, N., Mc Fadden, E. P., Van Belle, E., Millaire, A. & De Groote, P. (2003). Influence of diabetes mellitus on heart failure risk and outcome. *Cardiovasc Diabetol*, 2(1): 1-32.
- Bazzano, L. A., He, J., Ogden, L. G., Loria, C. M., Vupputuri, S., Myers, L. & Whelton, P. K. (2002). Fruit and vegetable intake and risk of cardiovascular disease in US adults: the first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study. *The American Journal of Clinical Nutrition*, 76(1): 93-99.
- **Bazzano**, L. A., Serdula, M. K. & Liu, S. (2003). Dietary intake of fruits and vegetables and risk of cardiovascular disease. *Current Atherosclerosis Reports*, 5(6):492-499.
- Beaglehole R., Bonita R., Horton R., Adams C. & Alleyne G. (2010). Priority actions for the non-communicable disease crisis. *Lancet*, 377:1438–1447.
- Begum, M. A., Gilgs, W. W., Mould, W. W., Laing, R., Peggs, R.,.... & Krings, B. A. K. (2004). Glycemic control in young diabetic patients. *Diabetes Research and Clinical Practice*, 27(15): 35-72.
- **Bell**, D. S. (1995). Diabetic Cardiomyopathy: A unique entity or a complication of coronary artery disease? *Diabetes Care*, *18*(5): 708-714.

- Bender, D., A. (2005). "Nutritional status." A Dictionary of Food and Nutrition. Retrieved from Encyclopedia.com: <u>http://www.encyclopedia.com/doc/1039nutritionalstatus.</u> <u>html</u>. 27/02/2016. 3:30pm.
- Benoit, S. R., Fleming, R., Philis-Tsimikas, A. & Ji, M. (2005). Predictors of glycemic control among patients with Type 2 diabetes: a longitudinal study. *BMC Public Health*, 5(1): 1-15.
- Bergman, R. N. & Ader, M. (2000). Free fatty acids and pathogenesis of type 2 diabetes mellitus. *Trends in Endocrinology & Metabolism*, 11(9): 351-356.
- **Berhe**, K. K., Gebru, H. B., Kahsay, H. S. & Kahsay, A. A. (2014). Assessment of diabetes knowledge and its associated factors among Type 2 diabetic patients in Mekelle and Ayder Referral Hospitals, Ethiopia. *Journal of Diabetes & Metabolism*, *34*(11): 121276.
- Berrington de Gonzalez, A., Hartge, P., Cerhan, J. R., Flint, A. J., Hannan, L., MacInnis, R. J., ... & Beeson, W. L. (2010). Body-mass index and mortality among 1.46 million white adults. *New England Journal of Medicine*, 363(23): 2211-2219.
- **Bierman**, E. L. (2001). American Diabetes Association Clinical Practice Recommendations 2001. *Diabetes Care*, 24: S1-S133.
- Bitz, C., Toubro, S., Larsen, T. M., Harder, H., Rennie, K. L., Jebb, S. A. & Astrup, A. (2004). Increased 24-h energy expenditure in type 2 diabetes. *Diabetes Care*, 27(10): 24162421.
- Boden, G. (1997). Role of fatty acids in the pathogenesis of insulin resistance and NIDDM. *Diabetes*, 46(1): 3-10.
- Bogers, R. P., Bemelmans, W. J., Hoogenveen, R. T., Boshuizen, H. C., Woodward, M., Knekt, P., ... & Thorpe, R. J. (2007). Association of overweight with increased risk of coronary heart disease partly independent of blood pressure and cholesterol levels: a metaanalysis of 21 cohort studies including more than 300 000 persons. Archives of Internal Medicine, 167(16): 1720-1728.
- Bonomo, É., Caiaffa, W. T., César, C. C., Lopes, A. C. S. & Lima-Costa, M. F. (2003). Food intake according to socioeconomic and demographic profile: the Bambuí Project. *Cad. Saúde Pública*, *19*(5): 1461-1471.
- **Bott**, U., Jörgens, V., Grüsser, M., Bender, R., Mühlhauser, I. & Berger, M. (1994). Predictors of glycaemic control in type 1 diabetic patients after participation in an intensified treatment and teaching programme. *Diabetic Medicine*, *11*(4): 362-371.
- **Boulton**, A. J. (2005). Management of diabetic peripheral neuropathy. *Clinical Diabetes*, 23(1): 9-15.
- Bronner, L. L., Kanter, D. S. & Manson, J. E. (1995). Primary prevention of stroke. *New England Journal of Medicine*, *333*(21): 1392-1400.

- **Brownlee**, M. (2001). Biochemistry and molecular cell biology of diabetic complications. *Nature*, *414*(6865): 813-820.
- **Bull** F.C., Martin T.S. and Armstrong T. (2009). Global Physical Activity Questionnaire (GPAQ): nine country reliability and validity. *J Phys Act Health*, 6:790-804.
- Cairns, V., Keil, U., Kleinbaum, D., Doering, A. & Stieber, J. (1984). Alcohol consumption as a risk factor for high blood pressure. Munich Blood Pressure Study. *Hypertension*, 6(1): 124-131.
- **Canadian Diabetes Association (CDA)**. (2008). Clinical Practice Guidelines for the Prevention and Management of Diabetes in Canada. Canadian Diabetes Association.
- Candido, R., Srivastava, P., Cooper, M. E. & Burrell, L. M. (2003). Diabetes mellitus: cardiovascular disease. *Current opinion in investigational drugs (London, England:* 2000), 4(9): 1088-1094.
- Caspersen, C. J., Bloemberg, B. P., Saris, W. H., Merritt, R. K. & Kromhout, D. (1991). The prevalence of selected physical activities and their relation with coronary heart disease risk factors in elderly men: the Zutphen Study, 1985. American Journal of Epidemiology, 133(11): 1078-1092.
- Celemajer, D., S. and Ayer J., G. (2006). Childhood risk factors for adult cardiovascular disease and primary prevention in childhood. Heart. 92(11): 1701-1706.
- Ceylan, S., Azal, Ö., Taşlipinar, A., Türker, T., Açikel, C. H. & Gulec, M. (2009). Complementary and alternative medicine use among Turkish diabetes patients. *Complementary Therapies in Medicine*, 17(2): 78-83.
- Chartier, K. & Caetano, R. (2009). Ethnicity and health disparities in alcohol research. Alcohol research & health: the journal of the National Institute on Alcohol Abuse and Alcoholism, 33(1-2): 152-160.
- Chase, H. P., Garg, S. K., Marshall, G., Berg, C. L., Harris, S., Jackson, W. E., & Hamman, R. E. (1991). Cigarette smoking increases the risk of albuminuria among subjects with type I diabetes. *JAMA*, 265(5): 614-617.
- Chaturvedi, N., Stevens, L., Fuller, J. H., & World Health Organization Multinational Study Group. (1997). Which features of smoking determine mortality risk in former cigarette smokers with diabetes? The World Health Organization Multinational Study Group. *Diabetes Care*, 20(8): 1266-1272.
- **Chawan**, J., & Biswas, T. (2013). How to calculate sample size for different study designs in medical research? *Indian journal of psychological medicine*, *35*(2): 121-253.

- **Chen**, C. H., Lin, K. C., Tsai, S. T., & Chou, P. (2000). Different association of hypertension and insulin-related metabolic syndrome between men and women in 8437 nondiabetic Chinese. *American Journal of Hypertension*, *13*(7):846-853.
- Chinenye, S., Uloko, A. E., Ogbera, A. O., Ofoegbu, E. N., Fasanmade, O. A., Fasanmade, A. A., & Ogbu, O. O. (2012). Profile of Nigerians with diabetes mellitus-Diabcare Nigeria study group (2008): Results of a multicenter study. *Indian Journal of Endocrinology and Metabolism*, 16(4): 558.
- Choi, Y. J., Kim, H. C., Kim, H. M., Park, S. W., Kim, J., & Kim, D. J. (2009). Prevalence and Management of Diabetes in Korean Adults Korea National Health and Nutrition Examination Surveys 1998–2005. *Diabetes Care*, 32(11): 2016-2020.
- Chow, C. K., Raju, P. K., Raju, R., Reddy, K. S., Cardona, M., Celermajer, D. S., & Neal, B. C. (2006). The prevalence and management of diabetes in rural India. *Diabetes Care*, 29(7): 1717-1718.
- Christensen, D. L., Friis, H., Mwaniki, D. L., Kilonzo, B., Tetens, I., Boit, M. K., ... & BorchJohnsen, K. (2009). Prevalence of glucose intolerance and associated risk factors in rural and urban populations of different ethnic groups in Kenya. *Diabetes Research* and Clinical Practice, 84(3): 303-310.
- Chudyk, A., & Petrella, R. J. (2011). Effects of exercise on cardiovascular risk factors in Type 2 diabetes: A meta-analysis. *Diabetes Care*, *34*(5): 1228-1237.
- Cobayashi, T., Farrell, M.A., Chaput, L.A., Rocha, D.A. & Hernandez, M. (2010). Lifestyle Intervention, Behavioral Changes, and Improvement in Cardiovascular Risk Profiles. *Journal of Women's Health*, 19(6):1129-1138.
- **Commerford**, P., & Mayosi, B. (2006). An appropriate research agenda for heart disease in Africa. *The Lancet*, *367*(9526): 1884-1886.
- **Connor,** H., Annan F., Bunn E., Frost G., McGough N., Sarwar T., Thomas B. (2003). The implementation of nutritional advice for people with diabetes. Nutrition Subcommittee of the Diabetes Care Advisory Committee of Diabetes UK *Diabet Med. Oct;* 20(10):786-807.
- Cooper, K. A., Chopra, M., & Thurnham, D. I. (2004). Wine polyphenols and promotion of cardiac health. *Nutrition Research Reviews*, 17:111-129.
- Corpus, R. A., George, P. B., House, J. A., Dixon, S. R., Ajluni, S. C., Devlin, W. H., ... & O'Neill, W. W. (2004). Optimal glycemic control is associated with a lower rate of target vessel revascularization in treated type II diabetic patients undergoing elective percutaneous coronary intervention. *Journal of the American College of Cardiology*, 43(1): 8-14.
- Cosentino, F., & Egidy Assenza, G. (2004). Diabetes and inflammation. Herz, 29(8): 749-759.

- **Costa-Font**, J., & Gil, J. (2005). Obesity and the incidence of chronic diseases in Spain: a seemingly unrelated probit approach. *Economics & Human Biology*, *3*(2): 188-214
- Croft, J. B., Keenan, N. L., Sheridan, D. P., Wheeler, F. C., & Speers, M. A. (1995). Waisttohip ratio in a biracial population: measurement, implications, and cautions for using guidelines to define high risk for cardiovascular disease. *Journal of the American Dietetic Association*, 95(1), 60-64.
- Cui, R., Iso H., Toyoshima, H., Date, C., Yamamoto, A., and Kikuchi, S. (2005). JACC study group. Body mass index and mortality from cardiovascular disease among Japanese men and women: the JACC study. *Stroke*. 36:1377–82
- Cummins, S., Smith, D. M., Taylor, M., Dawson, J., Marshall, D., Sparks, L., & Anderson, A. S. (2009). Variations in fresh fruit and vegetable quality by store type, urban–rural setting and neighbourhood deprivation in Scotland. *Public Health Nutrition*, 12(11): 2044-2050.
- Cunningham-Myrie, C., Younger-Coleman, N., Tulloch-Reid, M., McFarlane, S., Francis, D., Ferguson, T., ... & Wilks, R. (2013). Diabetes mellitus in Jamaica: sex differences in burden, risk factors, awareness, treatment and control in a developing country. *Tropical Medicine & International Health*, 18(11): 1365-1378.
- Czernichow, S., Kengne, A. P., Huxley, R. R., Batty, G. D., De Galan, B., Grobbee, D., ... & Neal, B. (2011). Comparison of waist-to-hip ratio and other obesity indices as predictors of cardiovascular disease risk in people with type-2 diabetes: a prospective cohort study from ADVANCE. *European Journal of Cardiovascular Prevention & Rehabilitation*, *18*(2): 312-319.
- Dai, W. S., Laporte, R. E., Hom, D. L., Kuller, L. H., D'antonio, J. A., Gutai, J. P., ... & Wohlfahrt, B. (1985). Alcohol consumption and high density lipoprotein cholesterol concentration among alcoholics. *American Journal of Epidemiology*, 122(4): 620-627.
- Dannemann, K., Hecker, W., Haberland, H., Herbst, A., Galler, A., Schäfer, T., ... & Kapellen, T. M. (2008). Use of complementary and alternative medicine in children with type 1 diabetes mellitus-prevalence, patterns of use, and costs. *Pediatric Diabetes*, 9(3pt1): 228-235.
- **Daousi** C., Casson I.F., Gill G.V., MacFarlane I.A., Wilding J.P. & Pinkney J.H. (2006). Prevalence of obesity in type 2 diabetes in secondary care: association with cardiovascular risk factors. *Postgrad Med J*.;82 (966):280-284.
- **De Caterina**, R., Zampolli, A., Del Turco, S., Madonna, R. & Massaro, M. (2006). Nutritional mechanisms that influence cardiovascular disease. *The American Journal of Clinical Nutrition*, 83(2): 421S-426S.
- **de Ramirez**, S. S., Enquobahrie, D. A., Nyadzi, G., Mjungu, D., Magombo, F., Ramirez, M., ... & Willett, W. (2010). Prevalence and correlates of hypertension: a cross-sectional

study among rural populations in sub-Saharan Africa. *Journal of Human Hypertension*, 24(12): 786-795.

- Dembele, M., Sidibé, A. T., Traoré, H. A., Tchombou, H. I. C., Zounet, B., Traore, A. K., .... & fongoro, S. (2000). Association HTA-Diabète sucré dans le service de Médecine interne de l'hôpital du Point G-Bamako. *Med Afr Noire*, 47(6) :120-174.
- **Després**, J. P., Lamarche, B., Mauriège, P., Cantin, B., Dagenais, G. R., Moorjani, S. & Lupien, P. J. (1996). Hyperinsulinemia as an independent risk factor for ischemic heart disease. *New England Journal of Medicine*, *334*(15): 952-958.
- **Dexter,** C., Cairns, B. J., Balkwill, A., Wright, F. L., Green, J., Reeves, G. & Beral, V. (2013). Body mass index and incident coronary heart disease in women: a population-based prospective study. *BMC Medicine*, *11*(1):1-121.
- **Dickinson**, H. O., Mason, J. M., Nicolson, D. J., Campbell, F., Beyer, F. R., Cook, J. V., ... & Ford, G. A. (2006). Lifestyle interventions to reduce raised blood pressure: a systematic review of randomized controlled trials. *Journal of Hypertension*, 24(2): 215-233.
- **Dietary Guidelines Advisory Committee**. (2010). Adults and sodium: what is the relationship between sodium and blood pressure in adults aged 19 years and older? Department of Health and Human Services and Department of Agriculture.
- Din, R. (2002). Diet and cardiovascular disease. American Journal of Public Health, 92(7): 1050-1051.
- Dinarello, C. A. (2000). Proinflammatory cytokines. Chest Journal, 118(2): 503-508.
- **Dodu**, S. R. (1958). The incidence of diabetes mellitus in Accra (Ghana); a study of 4,000 patients. *The West African Medical Journal*, 7(3): 129-134.
- **Dokken**, B. B. (2008). The pathophysiology of cardiovascular disease and diabetes: beyond blood pressure and lipids. *Diabetes Spectrum*, 21(3): 160-165.
- **Donaghue**, K. C., Chiarelli, F., Trotta, D., Allgrove, J. & Dahl-Jorgensen, K. (2009). Microvascular and macrovascular complications associated with diabetes in children and adolescents. *Pediatric Diabetes*, *10*(s12): 195-203.
- **Dong** M., Giles W. H., Felitti V. J., Dube S. R., Williams J. E., Chapman D. P. & Anda R. F. (2004). Insights into causal pathways for ischemic heart disease: Adverse childhood experiences study. *Circulation*, 110(13): 1761-1798.
- **Dunn**, F. L. (1990). Hyperlipidemia in diabetes mellitus. *Diabetes/Metabolism Reviews*, 6(1): 47-61.

- **Dzau**, V. & Braunwald, E. (1991). Resolved and unresolved issues in the prevention and treatment of coronary artery disease: a workshop consensus statement. *American Heart Journal*, *121*(4): 1244-1263.
- Eghan, B. A., Frempong, M. T. & Adjei-Poku, M. (2007). Prevalence and predictors of microalbuminuria in patients with diabetes mellitus: a cross-sectional observational study in Kumasi, Ghana. *Ethnicity and Disease*, 17(4): 726-765.
- Eid, M., Mafauzy, M., & Faridah, A. R. (2004). Non-achievement of clinical targets in patients with type 2 diabetes mellitus. *Medical Journal of Malaysia*, *59*(2): 177-184.
- Ekinci, E. I., Clarke, S., Thomas, M. C., Moran, J. L., Cheong, K., MacIsaac, R. J., & Jerums, G. (2011). Dietary salt intake and mortality in patients with type 2 diabetes. *Diabetes Care*, *34*(3):703-709.
- **Ekpenyong**, C. E., Akpan, U. P., Ibu, J. O., & Nyebuk, D. E. (2012). Gender and age specific prevalence and associated risk factors of type 2 diabetes mellitus in Uyo metropolis, south eastern Nigeria. *Diabetol Croat*, *41*(1):17-28.
- Elliott, P., Stamler, J., Nichols, R., Dyer, A. R., Stamler, R., Kesteloot, H., & Marmot, M. (1996). Intersalt revisited: further analyses of 24 hour sodium excretion and blood pressure within and across populations. *BMJ*, *312*(7041): 1249-1253
- Esmaillzadeh, A., Kimiagar, M., Mehrabi, Y., Azadbakht, L., Hu, F. B., & Willett, W. C. (2007). Dietary patterns, insulin resistance, and prevalence of the metabolic syndrome in women. *The American Journal of Clinical Nutrition*, 85(3): 910-918.
- Facchini, F., Chen, Y. I., & Reaven, G. M. (1994). Light-to-moderate alcohol intake is associated with enhanced insulin sensitivity. *Diabetes Care*, *17*(2): 115-119.
- Faerch, K., Borch-Johnsen, K., Vaag, A., Jørgensen, T., & Witte, D. R. (2010). Sex differences in glucose levels: a consequence of physiology or methodological convenience? *Diabetologia*, 53(5): 858-865.
- **Faghilimnai,** S., Hashemipour M., Kelishadi B. (2006). The lipid profile of children with type 1 diabetes as compared to the controls. *ARYA*. *J*, 2(1):36-38.
- Fagot-Campagna, A., Pettitt, D. J., Engelgau, M. M., Burrows, N. R., Geiss, L. S., Valdez, R.,
  ... & Narayan, K. V. (1998). Type 2 diabetes among adolescents: An epidemiologic health perspective. *The Journal of Pediatrics*, 136(5): 664-672.
- Fang, Z. Y., Sharman, J., Prins, J. B. & Marwick, T. H. (2005). Determinants of exercise capacity in patients with type 2 diabetes. *Diabetes Care*, 28(7): 1643-1648.
- Ferrari, R., Merli, E., Cicchitelli, G., Mele, D., Fucili, A. & Ceconi, C. (2004). Therapeutic effects of 1-carnitine and propionyl-1-carnitine on cardiovascular diseases: A review. *Annals of the New York Academy of Sciences*, 1033(1): 79-91.

- Fong, D. S., Aiello, L., Gardner, T. W., King, G. L., Blankenship, G., Cavallerano, J. D., ... & Klein, R. (2004). Retinopathy in diabetes. *Diabetes Care*, 27(suppl 1): s84-s87.
- Fontana, L., Eagon, J. C., Trujillo, M. E., Scherer, P. E., & Klein, S. (2007). Visceral fat adipokine secretion is associated with systemic inflammation in obese humans. *Diabetes*, *56*(4): 1010-1013.
- **Fontana**, L., Meyer, T. E., Klein, S., & Holloszy, J. O. (2004). Long-term calorie restriction is highly effective in reducing the risk for atherosclerosis in humans. *Proceedings of the National Academy of Sciences of the United States of America*, *101*(17): 6659-6663.
- **Forghani** B, Kasaeyan N, Faghih-Imani B, Hosseinpour M, Amini M. (2000). The assessment of physical activity in non-insulin dependent diabetic patient reffered to Esfahan Endocrine & Metabolism Research Center. *Jundishapur Scientific Medical Journal*. 31:41-5.
- **Fourlanos**, S., Varney, M. D., Tait, B. D., Morahan, G., Honeyman, M. C., Colman, P. G., & Harrison, L. C. (2008). The rising incidence of type 1 diabetes is accounted for by cases with lower-risk human leukocyte antigen genotypes. *Diabetes Care*, *31*(8): 1546-1549.
- Franconi, F., Campesi, I., Occhioni, S., & Tonolo, G. (2012). Sex-gender differences in diabetes vascular complications and treatment. *Endocrine, Metabolic & Immune Disorders-Drug Targets (Formerly Current Drug Targets-Immune, Endocrine & Metabolic Disorders), 12*(2): 179-196.
- Fröhlich-Reiterer, E. E., & Borkenstein, M. H. (2010). Type-1-Diabetes mellitus: Spätkomplikationen im Kindes-und Jugendalter. *Wiener Medizinische Wochenschrift*, *160*(15-16), 414-418.
- Fuchs, F. D., Gus, M., Moreira, L. B., Moraes, R. S., Wiehe, M., Pereira, G. M., & Fuchs, S. C. (2005). Anthropometric indices and the incidence of hypertension: a comparative analysis. *Obesity Research*, 13(9), 1515-1517.
- **Gabbay**, K. H. (2004). Aldose reductase inhibition in the treatment of diabetic neuropathy: where are we in 2004? *Current Diabetes Reports*, *4*(6): 405-408.
- Garty, M., Shotan, A., Gottlieb, S., Mittelman, M., Porath, A., Lewis, B. S., ... & Zimlichman, R. (2007). The management, early and one year outcome in hospitalized patients with heart failure: a national Heart Failure Survey in Israel--HFSIS 2003. *The Israel Medical Association Journal: IMAJ*, 9(4): 227-233.
- Gaziano, J. M., Manson, J. E., Branch, L. G., Colditz, G. A., Willett, W. C., & Buring, J. E. (1995). A prospective study of consumption of carotenoids in fruits and vegetables and decreased cardiovascular mortality in the elderly. *Annals of Epidemiology*, 5(4): 255260.

- Genkinger, J. M., Platz, E. A., Hoffman, S. C., Comstock, G. W., & Helzlsouer, K. J. (2004). Fruit, vegetable, and antioxidant intake and all-cause, cancer, and cardiovascular disease mortality in a community-dwelling population in Washington County, Maryland. *American Journal of Epidemiology*, 160(12): 1223-1233.
- Geraldes, P. & King, G. L. (2010). Activation of protein kinase C isoforms and its impact on diabetic complications. *Circulation Research*, *106*(8): 1319-1331.
- Getz, G. S. & Reardon, C. A. (2007). Nutrition and cardiovascular disease. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 27(12): 2499-2506.
- Getz, G. S. & Reardon, C. A. (2010). High-density lipoprotein function in regulating insulin secretion: possible relevance to metabolic syndrome. *Arteriosclerosis, Thrombosis, and Vascular Biology*, *30*(8): 1497-1499.
- Gezawa, I. D., Puepet, F. H., Mubi, B. M., Uloko, A. E., Bakki, B., Talle, M. A. & Haliru, I. (2015). Socio-demographic and anthropometric risk factors for Type 2 diabetes in Maiduguri, North-Eastern Nigeria. *Sahel Medical Journal*, *18*(5): 1-25.
- **Ghana News Agency-Health News**, (2013). Ghana's NHIS excludes 64 percent of population. Ghana web News. 15/07/2013.
- Ghana Statistical Service (GSS), Ghana Health Service (GHS), & ICF Macro. (2009). Ghana Demographic and Health Survey 2008. Accra, Ghana. pp. 60–64.
- Giunti, S., Barit, D., & Cooper, M. E. (2006). Diabetic nephropathy: from mechanisms to rational therapies. *Minerva Medica*, 97(3): 241-262.
- Glovannucci, E., Colditz, G., Stampfer, M. J., Rimm, E. B., Litin, L., Sampson, L., & Willett,
  W. C. (1991). The assessment of alcohol consumption by a simple self-administered questionnaire. *American Journal of Epidemiology*, 133(8): 810-817.
- Goh, K., & Tooke, J. (2002). Abnormalities of the microvasculature. Oxford Textbook of Endocrinology and Diabetes. Oxford University Press, Oxford, pp1749-1755.
- Goldberg, I. J. (2001). Diabetic dyslipidemia: causes and consequences. *The Journal of Clinical Endocrinology & Metabolism*, 86(3): 965-971.
- Goodpaster, B. H., Kelley, Mihalik, S. J., D. E., Chace, D. H., Vockley, J., Toledo, F. G., & DeLany, J. P. (2010). Increased levels of plasma acylcarnitines in obesity and type 2 diabetes and identification of a marker of glucolipotoxicity. *Obesity*, *18*(9):1695-1700.
- Goran, M. I., Ball, G. D., & Cruz, M. L. (2003). Obesity and risk of type 2 diabetes and cardiovascular disease in children and adolescents. *The Journal of Clinical Endocrinology & Metabolism*, 88(4): 1417-1427.
- Graudal, N. A., Hubeck-Graudal, T., & Jürgens, G. (2012). Effects of low-sodium diet vs.

high-sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride (Cochrane Review). *American Journal of Hypertension*, 25(1), 1-15.

- Gregg, E. W., Beckles, G. L., Williamson, D. F., Leveille, S. G., Langlois, J. A., Engelgau, M. M., & Narayan, K. M. (2000). Diabetes and physical disability among older US adults. *Diabetes Care*, 23(9): 1272-1277.
- Gregg, E. W., Gerzoff, R. B., Caspersen, C. J., Williamson, D. F. & Narayan, K. V. (2003). Relationship of walking to mortality among US adults with diabetes. *Archives of Internal Medicine*, 163(12), 1440-1447.
- **Grimble**, R. F. (2002). Inflammatory status and insulin resistance. *Current Opinion in Clinical Nutrition & Metabolic Care*, *5*(5): 551-559.
- **Gross**, M. L., Dikow, R. & Ritz, E. (2005). Diabetic nephropathy: recent insights into the pathophysiology and the progression of diabetic nephropathy. *Kidney International*, 67: S50-S53.
- Grundy, S. M., Cleeman, J. I., Daniels, S. R., Donato, K. A., Eckel, R. H., Franklin, B. A., ... & Spertus, J. A. (2005). Diagnosis and management of the metabolic syndrome an American Heart Association/National Heart, Lung and Blood Institute scientific statement. *Circulation*, 112(17): 2735-2752.
- Gu, H., Yang, J., Li, W., Teo, K., Liu, L., & Yusuf, S. (2013). Physical activity and its relationship with obesity hypertension and diabetes in urban and rural China: the pure China study. *Journal of the American College of Cardiology*, 61(10\_S) :230-252.
- **Guariguata**, L., Whiting, D., Weil, C., & Unwin, N. (2011). The International Diabetes Federation diabetes atlas methodology for estimating global and national prevalence of diabetes in adults. *Diabetes Research and Clinical Practice*, 94(3): 322-332.
- Gudbjörnsdottir, S., Cederholm, J., Nilsson, P. M., & Eliasson, B. (2003). The National Diabetes Register in Sweden An implementation of the St. Vincent Declaration for Quality Improvement in Diabetes Care. *Diabetes Care*, 26(4): 1270-1276.
- Gudina, E. K., Amade, S. T., Tesfamichael, F. A., & Ram, R. (2012). Assessment of quality of care given to diabetic patients at Jimma University Specialized Hospital diabetes follow-up clinic, Jimma, Ethiopia. *BMC Endocrine Disorders*, 11(1): 1-75.
- Guyton, A. C., & Hall, J. E. (2006). Dietary balances; regulation of feeding; obesity and starvation; vitamins and minerals. *Textbook of Medical Physiology (Guyton AC, Hall JE, eds)*. *Elsevier Saunders Inc. Philadelphia, PN*, pp. 876-890.
- Habib, A. N., Baird, B. C., Leypoldt, J. K., Cheung, A. K., & Goldfarb-Rumyantzev, A. S. (2006). The association of lipid levels with mortality in patients on chronic peritoneal dialysis. *Nephrology Dialysis Transplantation*, 21(10): 2881-2892.

- Habib, S. S. (2006). Frequency distribution of atherogenic dyslipidemia in Saudi type 2 diabetic patients. *Pak J Physiol*, 2(2): 20-23.
- Haffner, S. M., Lehto, S., Rönnemaa, T., Pyörälä, K., & Laakso, M. (1998). Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. *New England Journal of Medicine*, 339(4): 229-234.
- Halat K.M., and Dennehy C.E. (2003). Botanicals and dietary supplements in diabetic peripheral neuropathy. *J Am Board Family Pract.*; 16:47–57.
- Hall, J. N., Moore, S., Harper, S. B., & Lynch, J. W. (2009). Global variability in fruit and vegetable consumption. *American Journal of Preventive Medicine*, *36*(5): 402-409.
- Hall, V., Thomsen, R. W., Henriksen, O., & Lohse, N. (2011). Diabetes in sub-Saharan Africa 1999-2011: epidemiology and public health implications. A systematic review. BMC Public Health, 11(1): 1-13.
- Hamilton, C. A., Miller, W. H., Sammy, A. B., Brosnan, M. J., Drummond, R. D., McBride, M. W., & Dominiczak, A. F. (2004). Strategies to reduce oxidative stress in cardiovascular disease. *Clinical Science*, 106(3): 219-234.
- Hammond K.A. (2000). Dietary and clinical assessment. *Krause's Food, Nutrition and Diet Therapy*. Tenth edition. Philadelphia: W.B. Saunders Company. pp 17-174.
- Han, T. S., Kelly, I. E., Walsh, K., Greene, R. M. E., & Lean, M. E. J. (1997). Relationship between volumes and areas from single transverse scans of intra-abdominal fat measured by magnetic resonance imaging. *International Journal of Obesity & Related Metabolic Disorders*, 21(12): 49-124.
- Han, T. S., Van Leer, E. M., Seidell, J. C., & Lean, M. E. J. (1995). Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. *BMJ*, 311(7017): 1401-1405.
- Hansson, G. K. (2005). Inflammation, atherosclerosis, and coronary artery disease. *New England Journal of Medicine*, *352*(16): 1685-1695.
- Harney, F. (2006). Diabetic retinopathy. Diabetes Care 34 (3): 95-98.
- Harris, M. I. (1991). Hypercholesterolemia in diabetes and glucose intolerance in the US population. *Diabetes Care*, *14*(5): 366-374.
- Hasan, S. S., Ahmed, S. I., Bukhari, N. I., & Loon, W. C. W. (2009). Use of complementary and alternative medicine among patients with chronic diseases at outpatient clinics. *Complementary Therapies in Clinical Practice*, 15(3): 152-157.

- Haus, J. M., Solomon, T. P., Lu, L., Jesberger, J. A., Barkoukis, H., Flask, C. A., & Kirwan, J. P. (2011). Intramyocellular lipid content and insulin sensitivity are increased following a short-term low-glycemic index diet and exercise intervention. *American Journal of Physiology-Endocrinology and Metabolism*, 301(3): E511-E516.
- He, F. J., Nowson, C. A., Lucas, M., & MacGregor, G. A. (2007). Increased consumption of fruit and vegetables is related to a reduced risk of coronary heart disease: meta-analysis of cohort studies. *Journal of Human Hypertension*, 21(9): 717-728.
- He, J., Ogden, L. G., Vupputuri, S., Bazzano, L. A., Loria, C., & Whelton, P. K. (1999). Dietary sodium intake and subsequent risk of cardiovascular disease in overweight adults. *JAMA*, 282(21): 2027-2034.
- Hertog, M. G., Sweetnam, P. M., Fehily, A. M., Elwood, P. C., & Kromhout, D. (1997). Antioxidant flavonols and ischemic heart disease in a Welsh population of men: the Caerphilly Study. *The American Journal of Clinical Nutrition*, 65(5): 1489-1494.
- **Heymsfield**, S. B., Baumgartner, R. N., & Pan, S. F. (1999). Nutritional assessment of malnutrition by anthropometric methods. *Modern Nutrition in Health and Disease*, 9: 903-922.
- Hills, C. E. (2009). Cellular and physiological effects of C-peptide. *Clinical Science*, *116*(7): 565-574.
- Ho, S. C., Chen, Y. M., Woo, J. L. F., Leung, S. S. F., Lam, T. H., & Janus, E. D. (2001). Association between simple anthropometric indices and cardiovascular risk factors. *International Journal of Obesity & Related Metabolic Disorders*, 25(11): : 1-24.
- Hoffmann, I. S. & Cubeddu, L. X. (2007). Increased blood pressure reactivity to dietary salt in patients with the metabolic syndrome. *Journal of Human Hypertension*, 21(6): 438444.
- Hokanson, J. E., & Austin, M. A. (1996). Plasma triglyceride level is a risk factor for cardiovascular disease independent of high-density lipoprotein cholesterol level: a metaanalysis of population-based prospective studies. Journal of Cardiovascular Risk, 3(2): 213-219.
- Hooper, L., Bartlett, C., Davey, S. G., & Ebrahim, S. (2004). Advice to reduce dietary salt for prevention of cardiovascular disease. *Cochrane Database Syst Rev, 1*(1).

SANE

Howard, B. V., Robbins, D. C., Sievers, M. L., Lee, E. T., Rhoades, D., Devereux, R. B., ... & Howard, W. J. (2000). LDL cholesterol as a strong predictor of coronary heart disease in diabetic individuals with insulin resistance and low LDL the Strong Heart Study. *Arteriosclerosis, Thrombosis, and Vascular biology*, 20(3): 830-835.

- Howard, W. J. (2000). LDL cholesterol as a strong predictor of coronary heart disease in diabetic individuals with insulin resistance and low LDL the Strong Heart Study. *Arteriosclerosis, Thrombosis, and Vascular biology*, 20(3): 830-835.
- Hu F. B. (2008). Globalization of food patterns and cardiovascular disease risk. *Circulation*. *118*(19): 1913-1914.
- Hu F.B., Manson J.E., Stampfer M.J., Colditz G., Liu S., Solomon C.G., Willett W.C. (2001). Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. *N. Engl. J. Med.* 345(11):790-7.
- Hu, F. B., & Willett, W. C. (2002). Optimal diets for prevention of coronary heart disease. *JAMA*, 288(20): 2569-2578.
- Hu, F. B., Rimm, E. B., Stampfer, M. J., Ascherio, A., Spiegelman, D., & Willett, W. C. (2000). Prospective study of major dietary patterns and risk of coronary heart disease in men. *The American Journal of Clinical Nutrition*, 72(4): 912-921.
- Hu, F. B., Stampfer, M. J., Manson, J. E., Ascherio, A., Colditz, G. A., Speizer, F. E., ... & Willett, W. C. (1999). Dietary saturated fats and their food sources in relation to the risk of coronary heart disease in women. *The American Journal of Clinical Nutrition*, 70(6): 1001-1008.
- Huang, Z., Willett, W. C., Manson, J. E., Rosner, B., Stampfer, M. J., Speizer, F. E., & Colditz, G. A. (1998). Body weight, weight change, and risk for hypertension in women. *Annals of Internal Medicine*, 128(2): 81-88.
- International Diabetes Federation (IDF). (2012). International Diabetes federation sixth edition.
- Ismail, I. S., Nazaimoon, W. W., Mohamad, W. W., Letchuman, R., Singaraveloo, M., Pendek, R., ... & Khalid, B. A. K. (2000). Socioedemographic determinants of glycaemic control in young diabetic patients in peninsular Malaysia. *Diabetes Research and Clinical Practice*, 47(1): 57-69.
- Jayarama, N., Reddy, M., & Lakshmaiah, V. (2012). Prevalence and pattern of dyslipidemia in type 2 diabetes mellitus patients in a rural tertiary care centre, southern India. *Glob. J. Med. Public Health*, 1: 24-27.
- Jenna Brinks, M.S., and Barry F. (2012). Risk factors for cardiovascular disease: Where do you fall? *America College of Sports Medicine*, 401:202-233.
- **Jialal**, I., Devaraj, S., & Venugopal, S. K. (2002). Oxidative stress, inflammation, and diabetic vasculopathies: the role of alpha tocopherol therapy. *Free Radical Research*, *36*(12) 1: 1331-1336.

- **Jifeng,** M. (2006). Diabetes care quality indicators. *The Medicare Quality Improvement Organization for Florida*, 12(5): 56-134.
- Johnson, J. A., Pohar, S. L., Secnik, K., Yurgin, N., & Hirji, Z. (2006). Utilization of diabetes medication and cost of testing supplies in Saskatchewan, 2001. *BMC Health Services Research*, *6*(1): 1-20.
- Johnson, N. (2001). Tobacco use and oral cancer: a global perspective. *Journal of Dental Education*, 65(4): 328-339.
- Joseph, E. U., Fadupin, G. T., & Keshinro, O. O. (2004). Prevalence of obesity among type 2 diabetics in Nigeria a case study of patients in Ibadan, Oyo State, Nigeria. *African Journal of Medicine and Medical Sciences*, *33*(4): 381-384.
- Joshipura, K. J., Ascherio, A., Manson, J. E., Stampfer, M. J., Rimm, E. B., Speizer, F. E., ... & Willett, W. C. (1999). Fruit and vegetable intake in relation to risk of ischemic stroke. *JAMA*, 282(13): 1233-1239.
- Jousilahti, P., Vartiainen, E., Tuomilehto, J., & Puska, P. (1999). Sex, age, cardiovascular risk factors, and coronary heart disease A prospective follow-up study of 14 786 middleaged men and women in Finland. *Circulation*, *99*(9): 1165-1172.
- Judykay, T. (2007). Nutrition for reducing urea and cratinine in the blood. *Diabetes Care*, 27: 2191-2192.
- Kako, Y., Huang, L. S., Yang, J., Katopodis, T., Ramakrishnan, R., & Goldberg, I. J. (1999). Streptozotocin-induced diabetes in human apolipoprotein B transgenic mice: effects on lipoproteins and atherosclerosis. *Journal of Lipid Research*, 40(12): 2185-2194.
- Kako, Y., Massé, M., Huang, L. S., Tall, A. R., & Goldberg, I. J. (2002). Lipoprotein lipase deficiency and CETP in streptozotocin-treated apoB-expressing mice. *Journal of Lipid Research*, 43(6): 872-877.
- **Kappala,** S. S. (2012). *Risk factors and Blood borne-biochemical markers in type 2 diabetes mellitus Journal of Diabetes Research*, 26(2): 236-313.
- Kaufman, D. L., Clare-Salzler, M., Tian, J., Forsthuber, T., Ting, G. S., Robinson, P., ... & Lehmann, P. V. (1993). Spontaneous loss of T-cell tolerance to glutamic acid decarboxylase in murine insulin-dependent diabetes. *Nature: International Weekly Journal of Science*, 366(6450): 69-72.
- Kearney, P. M., Whelton, M., Reynolds, K., Muntner, P., Whelton, P. K., & He, J. (2005). Global burden of hypertension: analysis of worldwide data. *The Lancet*, *365*(9455): 217-223.
- **Keeling**, A. (2011). The UN Summit and beyond: a new era for diabetes. *Diabetes Research and Clinical Practice*, *94*(1): 163-203.

- Keeling, A. (2012). The United Nations on non-communicable diseases—One year on. *Diabetes Research and Clinical Practice*, 98(1): 169-171.
- Keenan, H. A., Costacou, T., Sun, J. K., Doria, A., Cavellerano, J., Coney, J., ... & King, G. L. (2007). Clinical factors associated with resistance to microvascular complications in diabetic patients of extreme disease duration the 50-year medalist study. *Diabetes Care*, 30(8): 1995-1997.
- Kengne, A. P., & Awah, P. K. (2009). Classical cardiovascular risk factors and all-cause mortality in rural Cameroon. *QJM*, *102*(3): 209-215.
- Kengne, A. P., Amoah, A. G., & Mbanya, J. C. (2005). Cardiovascular complications of diabetes mellitus in sub-Saharan Africa. *Circulation*, 112(23): 3592-3601.
- Kershbaum, A., & Bellet, S. (1966). Smoking as a factor in atherosclerosis. A review of epidemiological, pathological, and experimental studies. *Geriatrics*, 21(12): 155.
- Khalid, N., Khan, E. A., Saleem, S., Tahir, A., Mahmood, H., & Saleem, S. (2014). Prevalence and associated factors of cigarette smoking among Type 2 Diabetes Patients in Pakistan. *International Journal of Collaborative Research on Internal Medicine & Public Health*, 6: 73-88.
- Kiechl, S., Willeit, J., Poewe, W., Egger, G., Oberhollenzer, F., Muggeo, M., & Bonora, E. (1996). Insulin sensitivity and regular alcohol consumption: large, prospective, cross sectional population study (Bruneck study). *BMJ*, 313(7064): 1040-1044.
- King, P., Peacock, I., & Donnelly, R. (1999). The UK Prospective Diabetes Study (UKPDS): clinical and therapeutic implications for type 2 diabetes. *British Journal of Clinical Pharmacology*, 48(5): 643-648.
- Kirschbaum, C., Wüst, S., & Strasburger, C. J. (1992). 'Normal'cigarette smoking increases free cortisol in habitual smokers. *Life Sciences*, *50*(6): 435-442.
- Kirwan, J. P., Solomon, T. P., Wojta, D. M., Staten, M. A., & Holloszy, J. O. (2009). Effects of 7 days of exercise training on insulin sensitivity and responsiveness in type 2 diabetes mellitus. *American Journal of Physiology-Endocrinology and Metabolism*, 297(1), E151-E156.
- Kokiwar, P. R., Gupta, S. S., & Durge, P. M. (2012). Prevalence of hypertension in a rural community of central India. *J Assoc Physicians India*, 60(2): 25-68.
- Kothari, V., Stevens, R. J., Adler, A. I., Stratton, I. M., Manley, S. E., Neil, H. A., ... & UK Prospective Diabetes Study Group. (2002). UKPDS 60 risk of stroke in type 2 diabetes estimated by the UK Prospective Diabetes Study risk engine. *Stroke*, *33*(7): 176-178.
- **Krauss**, R. M. (2004). Lipids and lipoproteins in patients with type 2 diabetes. *Diabetes Care*, 27(6): 1496-1504.

- Krishnamurthi, R. V., Feigin, V. L., Forouzanfar, M. H., Mensah, G. A., Connor, M., Bennett, D. A., ... & O'Donnell, M. (2013). Global and regional burden of first-ever ischaemic and haemorrhagic stroke during 1990–2010: findings from the Global Burden of Disease Study 2010. *The Lancet Global Health*, 1(5): e259-e281.
- **Kruger** H.S., Venter CS, Vorster HH. (2001). Obesity in African women in the North-West province, South Africa is associated with an increased risk of non-communicable diseases: the THUSA study. *Br J Nutr*. 86:733–740.
- **Kruger**, H. S., Puoane, T., Senekal, M., & van der Merwe, M. T. (2005). Obesity in South Africa: challenges for government and health professionals. *Public Health Nutrition*, 8(05): 491-500.
- Kuller, L. H., Kinzel, L. S., Pettee, K. K., Kriska, A. M., Simkin-Silverman, L. R., Conroy, M. B., ... & Johnson, B. D. (2006). Lifestyle intervention and coronary heart disease risk factor changes over 18 months in postmenopausal women: the Women On the Move through Activity and Nutrition (WOMAN Study) clinical trial. *Journal of Women's Health*, 15(8): 962-974.
- **Kumar**, A. (2013). Prevalence of glycemic status, obesity and waist circumference in Punjabi type 2 diabetics. *Journal of Exercise Science and Physiotherapy*, 9(1): 1-40.
- Kumar, P., & Clark, M. (2002). Diabetes mellitus and other disorders of metabolism. *Clinical Medicine*: 2:1069-1071.
- Kurth, T., Gaziano, J. M., Rexrode, K. M., Kase, C. S., Cook, N. R., Manson, J. E. & Buring, J. E. (2005). Prospective study of body mass index and risk of stroke in apparently healthy women. *Circulation*, 111(15): 1992-1998.
- Laabes, E. P., Thacher, T. D. & Okeahialam, B. N. (2008). Risk factors for heart failure in adult Nigerians. *Acta Cardiologica*, 63(4): 437-443.
- Lamarche, B., Després, J. P., Moorjani, S., Cantin, B., Dagenais, G. R. & Lupien, P. J. (1996). Triglycerides and HDL-cholesterol as risk factors for ischemic heart disease. Results from the Quebec cardiovascular study. *Atherosclerosis*, *119*(2): 235-245.
- Lands, W. E. & Zakhari, S. (1990). Alcohol and cardiovascular disease. *Alcohol Research and Health*, *14*(4): 304-350.
- Laquatra, I. (2004). Nutritional assessment for weight management. Krause's Food & Nutrition Therapy 11<sup>th</sup> ed Saunders/Elsevier. pp 65-224.
- Larsson, B., Svärdsudd, K., Welin, L., Wilhelmsen, L., Björntorp, P. & Tibblin, G. (1984). Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. Br Med J (Clin Res Ed), 288(6428): 1401-1404.

- Lavados, P. M., Sacks, C., Prina, L., Escobar, A., Tossi, C., Araya, F., ... & Alvarez, G. (2005). Incidence, 30-day case-fatality rate, and prognosis of stroke in Iquique, Chile: a 2-year community-based prospective study (PISCIS project). *The Lancet*, 365(9478): 22062215.
- Law, M. R. & Morris, J. K. (1998). By how much does fruit and vegetable consumption reduce the risk of ischaemic heart disease? *European Journal of Clinical Nutrition*, 52(8): 549556.
- Lawton, J., Ahmad, N., Hallowell, N., Hanna, L., & Douglas, M. (2005). Perceptions and experiences of taking oral hypoglycaemic agents among people of Pakistani and Indian origin: qualitative study. *BMJ*, *330*(7502), 1247-1267.
- Le Brasseur, N. K. & Ruderman, N. B. (2005). Why might thiazolidinediones increase exercise capacity in patients with type 2 diabetes? *Diabetes Care*, 28(12): 2975-2977.
- Lean, M. E., Han, T. S. & Deurenberg, P. (1996). Predicting body composition by densitometry from simple anthropometric measurements. *The American Journal of Clinical Nutrition*, 63(1): 4-14.
- Lee, R. D. & Nieman, D. C. (2003). Biochemical assessment of nutritional status. Nutritional Assessment, 3rd ed. New York: McGraw-Hill pp 20-124.
- Leenders, M., Sluijs, I., Ros, M. M., Boshuizen, H. C., Siersema, P. D., Ferrari, P., ... & ClavelChapelon, F. (2013). Fruit and vegetable consumption and mortality european prospective investigation into cancer and nutrition. *American Journal of Epidemiology*, 34(17): 2175-2477
- Lehto, S., Rönnemaa, T., Haffher, S. M., Pyörälä, K., Kallio, V., & Laakso, M. (1997). Dyslipidemia and hyperglycemia predict coronary heart disease events in middle-aged patients with NIDDM. *Diabetes*, *46*(8): 1354-1359.
- Lemieux, S., Despres, J. P., Moorjani, S., Nadeau, A., Theriault, G., Prud'Homme, D., ... & Lupien, P. J. (1994). Are gender differences in cardiovascular disease risk factors explained by the level of visceral adipose tissue? *Diabetologia*, *37*(8): 757-764.
- Lemieux, S., Prud'homme, D., Nadeau, A., Tremblay, A., Bouchard, C., & Després, J. P. (1996). Seven-year changes in body fat and visceral adipose tissue in women. Association with indexes of plasma glucose-insulin homeostasis. *Diabetes Care*, *19*(9): 983-991.
- Lenz, M., Richter, T., & Mühlhauser, I. (2009). The morbidity and mortality associated with overweight and obesity in adulthood. *Dtsch Arztebl Int*, *106*(40): 641-8.
- Leung, G. M., & Lam, K. S. (2000). Diabetic complications and their implications on health care in Asia. *Hong Kong Medical Journal*, 6(1): 61-68.

- Litwak, L., Goh, S. Y., Hussein, Z., Malek, R., Prusty, V., & Khamseh, M. E. (2013). Prevalence of diabetes complications in people with type 2 diabetes mellitus and its association with baseline characteristics in the multinational A 1 chieve study. *Diabetology & Metabolic Syndrome*, 5(1): 1-30.
- Liu, S., Lee, I. M., Ajani, U., Cole, S. R., Buring, J. E., & Manson, J. E. (2001). Intake of vegetables rich in carotenoids and risk of coronary heart disease in men: The Physicians' Health Study. *International Journal of Epidemiology*, 30(1): 130-135.
- Lobstein, T., Baur, L., & Uauy, R. (2004). Obesity in children and young people: a crisis in public health. *Obesity Reviews*, 5(s1): 4-85.
- Lucas, D. L., Brown, R. A., Wassef, M. and Giles, T. D. (2005). Alcohol and the cardiovascular system researchchallenges and opportunities. *J. Am Coll Cardiol*. 45(12):1916-1924.
- Lucini, D., Bertocchi, F., Malliani, A., & Pagani, M. (1996). A controlled study of the autonomic changes produced by habitual cigarette smoking in healthy subjects. *Cardiovascular Research*, *31*(4): 633-639.
- Luepker, R. V., Apple, F. S., Christenson, R. H., Crow, R. S., Fortmann, S. P., Goff, D., ... & Levy, D. (2003). Case definitions for acute coronary heart disease in epidemiology and clinical research studies a statement from the AHA council on epidemiology and prevention; AHA Statistics Committee; World Heart Federation Council on Epidemiology and Prevention; the European Society of Cardiology Working Group on Epidemiology and Prevention; Centers for Disease Control and Prevention; and the National Heart, Lung, and Blood Institute. *Circulation*, 108(20): 2543-2549.
- Lupien, P. J. (1994). Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *The American Journal of Cardiology*, 73(7): 460-468.
- Madonna, R., Pandolfi, A., Massaro, M., Consoli, A., & De Caterina, R. (2004). Insulin enhances vascular cell adhesion molecule-1 expression in human cultured endothelial

cells through a pro-atherogenic pathway mediated by p38 mitogen-activated proteinkinase. *Diabetologia*, 47(3):532-536.

- Maegawa, H. (2000). Impairments of insulin receptor function in insulin resistant states. *Nihon Hinsho. Japanese Journal of Clinical Medicine*, *58*(2): 304-309.
- Mahan, L. K. & Escott-Stump, S. (2008). Krause's Food & Nutrition Therapy 11<sup>th</sup> ed Saunders/Elsevier. pp. 120-127.
- Maher, D., Waswa, L., Baisley, K., Karabarinde, A., Unwin, N. & Grosskurth, H. (2011). Distribution of hyperglycaemia and related cardiovascular disease risk factors in

lowincome countries: a cross-sectional population-based survey in rural Uganda. *International Journal of Epidemiology*, 40(1): 160-171.

- Manninen, V., Tenkanen, L., Koskinen, P., Huttunen, J. K., Mänttäri, M., Heinonen, O. P. & Frick, M. H. (1992). Joint effects of serum triglyceride and LDL cholesterol and HDL cholesterol concentrations on coronary heart disease risk in the Helsinki Heart Study. Implications for treatment. *Circulation*, 85(1): 37-45.
- Manson, J. E., Stampfer, M. J., Colditz, G. A., Willett, W. C., Rosner, B., Hennekens, C. H., ... & Krolewski, A. S. (1991). Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *The Lancet*, 338(8770): 774-778.
- Manuel, D. G., Perez, R., Bennett, C., Rosella, L., Taljaard, M., Roberts, M., ... & Tanuseputro, P. (2012). Seven more years: The impact of smoking, alcohol, diet, physical activity, and stress on health and life expectancy in Ontario, un rapport ICES/PHO, Toronto. *Institute for Clinical and Evaluative Sciences et Santé publique Ontario 30*(1): 54-97.
- **Marcus**, A. O. (2001). Lipid disorders in patients with Type 2 diabetes: meeting the challenges of early, aggressive treatment. *Postgraduate Medicine*, *110*(1): 111-123.
- Martin, A.C. (2006). Clinical Chemistry and Metabolic Medicine. 7th ed. London, UK: Edward – Arnold (Publishers) Ltd. pp. 12-343.
- Martinez, P., Røislien, J., Naidoo, N. & Clausen, T. (2011). Alcohol abstinence and drinking among African women: data from the World Health Surveys. *BMC Public Health*, 11(1): 160-175.
- Martín-Timón I., Sevillano-Collantes C., Segura-Galindo A., Francisco J. and Cañizo-Gómez D. (2014). Type 2 diabetes and cardiovascular disease: Have all risk factors the same strength? World Journal of Diabetes 5(4): 444-470.
- Mathenge, W., Bastawrous, A., Peto, T., Leung, I., Yorston, D., Foster, A. & Kuper, H. (2014). Prevalence and correlates of diabetic retinopathy in a population-based survey of older people in Nakuru, Kenya. *Ophthalmic Epidemiology*, 21(3): 169-177.
- Mathenge, W., Foster, A. & Kuper, H. (2010). Urbanization, ethnicity and cardiovascular risk in a population in transition in Nakuru, Kenya: a population-based survey. *BMC Public Health*, *10*(1): 1-23.
- McGavock J.M, Victor R.G, Unger R.H. & Szczepaniak L.S., (2006). American College of Physicians and the American Physiological Society. Adiposity of the heart, revisited. *Ann Intern Med*, 144(7):517–524.
- Meneton, P., Jeunemaitre, X., de Wardener, H. E. & Macgregor, G. A. (2005). Links between dietary salt intake, renal salt handling, blood pressure, and cardiovascular diseases. *Physiological Reviews*, 85(2): 679-715.

- Mitch, W. E. (1998). Robert H Herman Memorial Award in Clinical Nutrition Lecture. Mechanisms causing loss of lean body mass in kidney disease. *The American Journal of Clinical Nutrition*, 67(3): 359-366.
- Moeini, B., Hazavehei, S. M. M., Jalilian, M., Moghimbeigi, A. & Tarigh Seresht, N. (2011). Factors affecting physical activity and metabolic control in type 2 diabetic women referred to the diabetes research center of Hamadan: Applying trans-theoretical model. *Scientific Journal of Hamadan University of Medical Sciences*, 18(2): 31-37.
- Mohammed, M. R., Shafek, M., El Damaty, S. & Seoudi, S. (2000). Hypertension control indicators among rural population in Egypt. *The Journal of the Egyptian Public Health Association*, 75(5-6): 391-401.
- Mookiah, M. R. K., Acharya, U. R., Fujita, H., Tan, J. H., Chua, C. K., Bhandary, S. V., ... & Tong, L. (2015). Application of different imaging modalities for diagnosis of Diabetic Macular Edema: A review. *Computers in Biology and Medicine*, 66: 295-315.
- Moore, S. M., Hardie, E. A., Hackworth, N. J., Critchley, C. R., Kyrios, M., Buzwell, S. A. & Crafti, N. A. (2011). Can the onset of type 2 diabetes be delayed by a group-based lifestyle intervention? A randomised control trial. *Psychology and Health*, 26(4): 485499.
- Morrish, N. J., Stevens, L. K., Fuller, J. H., Jarrett, R. J. & Keen, H. (1991). Risk factors for macrovascular disease in diabetes mellitus: the London follow-up to the WHO Multinational Study of Vascular Disease in Diabetics. *Diabetologia*, 34(8): 590-594.
- Motala, A. A., Esterhuizen, T., Gouws, E., Pirie, F. J. & Omar, M. A. (2008). Diabetes and other disorders of glycemia in a rural South African community prevalence and associated risk factors. *Diabetes Care*, *31*(9): 1783-1788.
- Mozaffarian, D., Appel, L. J. & Van Horn, L. (2011). Components of a cardioprotective diet new insights. *Circulation*, *123*(24): 2870-2891.
- Mukamal, K. J., Chen, C. M., Rao, S. R. & Breslow, R. A. (2010). Alcohol consumption and cardiovascular mortality among US adults, 1987 to 2002. *Journal of the American College of Cardiology*, 55(13):1328-1335.
- Murphy, M. H., Nevill, A. M., Neville, C., Biddle, S. & Hardman, A. E. (2002). Accumulating brisk walking for fitness, cardiovascular risk, and psychological health. *Journal of Nutrition*, 25(2): 123-229.
- Murphy, M. M., Barraj, L. M., Herman, D., Bi, X., Cheatham, R. & Randolph, R. K. (2012). Phytonutrient intake by adults in the United States in relation to fruit and vegetable consumption. *Journal of the Academy of Nutrition and Dietetics*, *112*(2): 222-229.
- Murray, C. J. & Lopez, A. D. (1997). Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. *The Lancet*, *349*(9063): 1436-1442.

- Mwangi, J., & Gitonga, L. (2014). Perceptions and use of herbal remedies among patients with diabetes mellitus in Murang'a North District, Kenya. *Open Journal of Clinical Diagnostics*, 4(03): 152-170.
- Naing, T. (2003). Hyperglycemia and diabetes complications. *Diabetes Care*, 59: 171-195.
- Nair, M. (2007). Diabetes mellitus, part 1: physiology and complications. *British Journal of Nursing*, *16*(3), 184-194.
- Narkiewicz, K., Redon, J., Cifkova, R., Laurent, S., Nilsson, P., S. & Mancia, G. (2009). Mechanisms of hypertension in the cardiometabolic syndrome. *Journal of hypertension*, 27(3): 441-451.
- National Heart, Lung and Blood Institute (NHLBI) Obesity Education Initiative (2000). Report of the Joint Committee: the use and interpretation of anthropometry, pp. 10-21.
- National Institute for Health and Clinical Excellence (NIHCE). (2005). Type 2 diabetes: The management in primary and secondary care.
- **National Institute for Health and Clinical Excellence NIHCE** (2010). Type 2 diabetes: The management in primary and secondary care.
- National Institute on Alcohol Abuse and Alcoholism (NIAAA) (2004). Alcohol use and alcohol use disorders in the United States: Main findings from the 2001-2002 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) 1. Vol. 8. National Institutes of Health; Bethesda, MDU.S. Alcohol Epidemiologic Data Reference Manual. 1(8): 23-56.
- National Institute on Alcohol Abuse and Alcoholism (NIAAA). (2009). Alcohol Alert, No. 67 "Underage Drinking," Available at: <u>http://pubs.niaaa.nih.gov/publications/ AA67/ AA 67. htm</u>
- National Institutes of Health. (2001). Third Report of the National Cholesterol Education Program Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *Executive Summary. Bethesda, MD, National Institutes of Health, National Heart, Lung and Blood Institute (NIH publ. no. 01-3670).*
- Navab, M., Anantharamaiah, G. M. & Fogelman, A. M. (2005). The role of high-density lipoprotein in inflammation. *Trends in Cardiovascular Medicine*, *15*(4): 158-161.
- Navab, M., Anantharamaiah, G. M., Reddy, S. T., Van Lenten, B. J., Ansell, B. J. & Fogelman, A. M. (2006). Mechanisms of disease: proatherogenic HDL—an evolving field. *Nature Reviews Endocrinology*, 2(9): 504-511.
- **Ness**, A. R. & Powles, J. W. (1997). Fruit and vegetables, and cardiovascular disease: a review. *International Journal of Epidemiology*, *26*(1): 1-13.

- **Ness-Abramof**, R. & Apovian, C. M. (2008). Waist circumference measurement in clinical practice. *Nutrition in Clinical Practice*, 23(4): 397-404.
- Nilsson, P. M. & Cederholm, J. (2011). Diabetes, hypertension, and outcome studies: overview 2010. *Diabetes Care*, 34(Supplement 2):S109-S113.
- Njoku, C. H. & Aduloju, A. B. (2004). Stroke in Sokoto, Nigeria: A five year retrospective study. *Nig Med Pract*, *32*(5/6): 54-62.
- Norman, A., Bellocco, R., Vaida, F. & Wolk, A. (2002). Total physical activity in relation to age, body mass, health and other factors in a cohort of Swedish men. *International Journal of Obesity & Related Metabolic Disorders*, 26(5): 307-384.
- Ntsekhe, M. & Damasceno, A. (2013). Recent advances in the epidemiology, outcome, and prevention of myocardial infarction and stroke in sub-Saharan Africa. *Heart*, 99(17): 1230-1235.
- Nyarko, A., Adubofour, K., Ofei, F., Kpodonu, J. & Owusu, S. (1997). Serum lipid and lipoprotein levels in Ghanaians with diabetes mellitus and hypertension. *Journal of the National Medical Association*, 89(3): 191-230.
- **Obrosova**, I. G. (2009). Diabetic painful and insensate neuropathy: pathogenesis and potential treatments. *Neurotherapeutics*, 6(4): 638-647.
- Ogah, O. S., Stewart, S., Falase, A. O., Akinyemi, J. O., Adegbite, G. D., Alabi, A. A.,.... & Sliwa, K. (2014). Contemporary profile of acute heart failure in Southern Nigeria: data from the Abeokuta Heart Failure Clinical Registry. *JACC: Heart Failure*, 2(3): 250259.
- **Oguntibeju**, O. O., Odunaiya, N., Oladipo, B. & Truter, E. J. (2012). Health behaviour and quality of life of patients with type 2 diabetes attending selected hospitals in south western Nigeria. *West Indian Medical Journal*, *61*(6): 619-626.
- **Ojji**, D. B., Ajayi, S. O., Mamven, M. H. & Atherton, J. (2009). Prevalence of dyslipidemia in normoglycemic subjects with newly diagnosed high blood pressure in Abuja, Nigeria. *Journal of Clinical Lipidology*, *3*(1): 51-56.
- Ojofeitimi, E. O., Adeyeye, A. O., Fadiora, A. O., Kuteyi, A. O., Faborode, T. G., Adegbenro, C. A., ... & Towobola, K. S. (2007). Awareness of obesity and its health hazard among women in a university community. *Pakistan Journal of Nutrition*, 6(5): 502-505.
- Okereke, O. I., Kang, J. H., Cook, N. R., Gaziano, J. M., Manson, J. E., Buring, J. E. & Grodstein, F. (2008). Type 2 Diabetes mellitus and cognitive decline in two large cohorts of community-dwelling older adults. *Journal of the American Geriatrics Society*, 56(6): 1028-1036.

- Okertchiri J.A. (2013). Ghana needs more Diabetic Educators. Daily Guide. 2013. Nov 16, [Last accessed on 2013 Jan 04]. Available from: <u>http://www.dailyguideghana.</u> <u>com/?p=67402</u>.
- **Okon** E.B., Chung A.W., Rauniyar P., Padilla E., Tejerina T., McManus B.M., Luo H. & van Breemen C. Compromised arterial function in human type 2 diabetic patients. *Diabetes* 54: 2415–2423, 2005.
- **Okoro**, E. O. & Oyejola, B. A. (2004). Inadequate control of blood pressure in Nigerians with diabetes. *Ethnicity and Disease*, *14*(1): 82-86.
- **Ola**, B. A., Adewuya, A. O., Ajayi, O. E., Akintomide, A. O., Oginni, O. O. & Ologun, Y. A. (2006). Relationship between depression and quality of life in Nigerian outpatients with heart failure. *Journal of Psychosomatic Research*, *61*(6): 797-800.
- **Onat**, A., Sansoy, V. & Uysal, Ö. (1999). Waist circumference and waist-to-hip ratio in Turkish adults: interrelation with other risk factors and association with cardiovascular disease. *International Journal of Cardiology*, *70*(1): 43-50.
- **Oni**, A. O., Eweka, A. O., Otuaga, P. O. & Prefa, V. (2009). The incidence and pattern of stroke in Bayelsa State, Nigeria. *Internet J Third World Med*, 8: 23-90.
- Ormel J., Von Korff M., Burger H., Scott K., Demyttenaere K., Huang Y., Posada-Villa J., Pierre-lepine J., Angermeyer M.C.,....& Levinson D. (2007). Mental disorders among persons with heart disease-results from World Mental Health surveys. *Gen Hosp Psychiatry*, 29(4):325-334.
- **Owoaje**, E. E., Rotimi, C. N., Kaufman, J. S., Tracy, J., & Cooper, R. S. (1997). Prevalence of adult diabetes in Ibadan, Nigeria. *East African Medical Journal*, 74(5): 299-302.
- Owusu, S. K. (1976). Diabetes in Ghana –A 10 year study. *Ghana Medical Journa*, 15(2): 93 -96.
- Pacheco-Alvarez D., Solórzano-Vargas R.S., Del Río A.L. (2002).Biotin in metabolism and its relationship to human disease. *Arch. Med. Res.* 33:439–447.
- Padayatty, S. J., Katz, A., Wang, Y., Eck, P., Kwon, O., Lee, J. H., ... & Levine, M. (2003). Vitamin C as an antioxidant: evaluation of its role in disease prevention. *Journal of the American College of Nutrition*, 22(1): 18-35.
- **Palmer**, M. and Betz J. (2002). In: Goldfrank's toxicologic emergencies. 7th edn.. New York: McGraw-Hill; pp. 1150–1182.
- **Palmer**, M.E. and Howland, M.A. (2001). Herbals and dietary supplements. In: Ford M, editors. Clinical toxicology. Philadelphia: WB Saunders; pp. 316–331.

- **Pampel**, F. (2008). Tobacco use in sub-Sahara Africa: estimates from the demographic health surveys. *Soc Sci Med.* 66(8):1772-83.
- Pancha, O., Koona, A. K., Yiagnigni, E. & Ndobo, P. (2012). The prevalence and management of hypertension in a population of adults with type 2 diabetes in the Adamawa Region (Cameroon): A retrospective analysis. *International Journal of Medicine and Medical Sciences*, 4(7): 147-151.
- Pandeya, A., Sharma, M., Regmi, P., Basukala, A. & Lamsal, M. (2012). Pattern of dyslipidemia and evaluation of non-HDL cholesterol as a marker of risk factor for cardiovascular disease in type 2 diabetes mellitus. *Nepal Med Coll J*, 14(4): 278-282.
- Park, K. (2009). Park's Textbook of Preventive and Social Medicine. 20th ed. Jabalpur, India; pp. 316–331.
- Peltzer, K. & Ramlagan, S. (2009). Alcohol use trends in South Africa. J Soc Sci, 18(1): 1-12.
- Pereira, M. A., O'Reilly, E., Augustsson, K., Fraser, G. E., Goldbourt, U., Heitmann, B. L., ... & Spiegelman, D. (2004). Dietary fiber and risk of coronary heart disease: a pooled analysis of cohort studies. *Archives of Internal Medicine*, 164(4): 370-376.
- Perrin, A. E., Simon, C., Hedelin, G., Arveiler, D., Schaffer, P. & Schlienger, J. L. (2002).
   Original Communications-Ten-year trends of dietary intake in a middle-aged French population: Relationship with educational level. *European Journal of Clinical Nutrition*, 56(5): 393-401
- **Perrone**, R. D., Madias, N. E. & Levey, A. S. (1992). Serum creatinine as an index of renal function: new insights into old concepts. *Clinical Chemistry*, *38*(10): 1933-1953.
- Peters, R., Poulter, R., Warner, J., Beckett, N., Burch, L. & Bulpitt, C. (2008). Smoking, dementia and cognitive decline in the elderly, a systematic review. *BMC Geriatrics*, 8(1): 36-50.
- Pham, L. H., Au, T. B., Blizzard, L., Truong, N. B., Schmidt, M. D., Granger, R. H. & Dwyer, T. (2009). Prevalence of risk factors for non-communicable diseases in the Mekong Delta, Vietnam: results from a STEPS survey. *BMC Public Health*, 9(1): 1-21.
- Pimenta, E., Gaddam, K. K., Oparil, S., Aban, I., Husain, S., Dell'Italia, L. J. & Calhoun, D. A. (2009). Effects of dietary sodium reduction on blood pressure in subjects with resistant hypertension results from a randomized trial. *Hypertension*, 54(3): 475-481.
- **Poikotainen**, K., Vartiainen, E., & Korhonen, H. J. (1996). Alcohol intake and subjective health. *American Journal of Epidemiology*, *144*(4): 346-350.
- **Powell**, K. E., Thompson, P. D., Caspersen, C. J., & Kendrick, J. S. (1987). Physical activity and the incidence of coronary heart disease. *Annual Review of Public Health*, 8(1), 253287.

- **Powers** S.K. and Howley E.T. (2007). Exercise Physiology: Theory and Application to Fitness and Performance. 6th edn. Boston: McGraw-Hill; pp. 540-690.
- **Prospective Studies Collaboration.** (2009). Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. *The Lancet*, *373*(9669):1083-1096.
- **Provenzano**, L. F., Stark, S., Steenkiste, A., Piraino, B. & Sevick, M. A. (2014). Dietary sodium intake in Type 2 diabetes. *Clinical Diabetes*, *32*(3): 106-112.
- **Puepet**, F. H. & Ohwovoriole, A. E. (2008). Prevalence of risk factors for diabetes mellitus in a non-diabetic population in Jos, Nigeria. *Nigerian Journal of Medicine*, *17*(1):71-74.
- Puoane, T., Fourie, J. M., Shapiro, M., Rosling, L., Tshaka, N. C., & Oelefse, A. (2005). \'Big is beautiful\'-an exploration with urban black community health workers in a South African township. South African Journal of Clinical Nutrition, 18(1):6-15.
- Purnell, J. Q., Kahn, S. E., Albers, J. J., Nevin, D. N., Brunzell, J. D. & Schwartz, R. S. (2000). Effect of weight loss with reduction of intra-abdominal fat on lipid metabolism in older men. *The Journal of Clinical Endocrinology & Metabolism*, 85(3): 977-982.
- Qidwai, W. & Azam, S. I. (2004). Knowledge, attitude and practice regarding obesity among patients, at Aga Khan University Hospital, Karachi. *J Ayub Med Coll Abbottabad*, *16*(4):93-102.
- **Raghupathy**, P., Antonisamy, B., Fall, C. H., Geethanjali, F. S., Leary, S. D., Saperia, J., ... & Richard, J. (2007). High prevalence of glucose intolerance even among young adults in south India. *Diabetes Research and Clinical Practice*, *77*(2): 269-279.
- Rahmanian, M., Haghighi, F. S., & Namiranian, N. (2014). Management of diabetes in Ramadan fasting. *Iranian Journal of Diabetes & Obesity (IJDO)*, 6(4):22-35.
- Raimi, T. H., Odusan, O., & Fasanmade, O. (2015). High prevalence of central obesity in rural South-Western Nigeria: Need for targeted prevention. *Journal of Diabetes and Endocrinology*, 6(3): 12-18.
- Rasmussen, M., Krølner, R., Klepp, K. I., Lytle, L., Brug, J., Bere, E., & Due, P. (2006). Determinants of fruit and vegetable consumption among children and adolescents: a review of the literature. Part I: quantitative studies. *International Journal of Behavioral Nutrition and Physical Activity*, 3(1): 22-43.
- Reisig, V., Reitmeir, P., Döring, A., Rathmann, W., Mielck, A., & KORA Study Group. (2007). Social inequalities and outcomes in type 2 diabetes in the German region of Augsburg. A cross-sectional survey. *International Journal of Public Health*, 52(3): 158-165.
- **Rema**, M., & Pradeepa, R. (2007). Diabetic retinopathy: an Indian perspective. *Indian Journal* of Medical Research, 125(3): 297-337.

- **RezaDerakhshan**, D., & Asghar, K. (2010). Evaluation of abdominal obesity prevalence in diabetic patients and relationships with metabolic syndrome factors. *International Journal of Endocrinology and Metabolism*, 2010(3): 143-146.
- Richards, J. C., Johnson, T. K., Kuzma, J. N., Lonac, M. C., Schweder, M. M., Voyles, W. F., & Bell, C. (2010). Short-term sprint interval training increases insulin sensitivity in healthy adults but does not affect the thermogenic response to β-adrenergic stimulation. *The Journal of Physiology*, 588(15): 2961-2972.
- **Rimm**, E. B., Chan, J., Stampfer, M. J., Colditz, G. A., & Willett, W. C. (1995). Prospective study of cigarette smoking, alcohol use, and the risk of diabetes in men. *BMJ*, *310*(6979): 555-559.
- Ross, R. (1999). Cell biology of atherosclerosis. Annu Rev Physio57: 791-804,
- **Ross**, R., Leger, L., Morris, D., de Guise, J. & Guardo, R. (1992). Quantification of adipose tissue by MRI: relationship with anthropometric variables. *Journal of Applied Physiology*, 72(2), 787-795.
- Rotimi, C. N., Chen, G., Adeyemo, A. A., Furbert-Harris, P., Guass, D., Zhou, J.,... & Acheampong, J. (2004). A genome-wide search for Type 2 diabetes susceptibility genes in West Africans The Africa America Diabetes Mellitus (AADM) Study. *Diabetes*, 53(3): 838-841.
- **Rudatsikira** E., Abdo A., Muula A.S. Prevalence and determinants of adolescent tobacco smoking in Addis Ababa, Ethiopia. BMC *Public Health*, 7(12):176-221.
- Rydén, L., Standl, E., Bartnik, M., Van den Berghe, G., Betteridge, J., De Boer, M. J., ... & Priori, S. (2007). Guidelines on diabetes, pre-diabetes, and cardiovascular diseases: *European Heart Journal Supplements*, 9(suppl C): 3-74.
- Saaddine, J. B., Cadwell, B., Gregg, E. W., Engelgau, M. M., Vinicor, F., Imperatore, G. & Narayan, K. V. (2006). Improvements in diabetes processes of care and intermediate outcomes: United States, 1988–2002. Annals of Internal Medicine, 144(7): 465-474.
- Sacks, F. M., Svetkey, L. P., Vollmer, W. M., Appel, L. J., Bray, G. A., Harsha, D., ... & Karanja, N. (2001). Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *New England Journal of Medicine*, 344(1): 3-10.
- Safeer, R. S. & Ugalat, P. S. (2002). Cholesterol treatment guidelines update. *American Family Physician*, 65(5): 871-882.
- Samman, S., Sivarajah, G., Man, J. C., Ahmad, Z. I., Petocz, P. & Caterson, I. D. (2003). A mixed fruit and vegetable concentrate increases plasma antioxidant vitamins and folate and lowers plasma homocysteine in men. *The Journal of Nutrition*, 133(7): 2188-2193.

- Sawant, A. M., Shetty, D., Mankeshwar, R. & Ashavaid, T. F. (2008). Prevalence of dyslipidemia in young adult Indian population. *JAPI*, *56*(2): 99-102.
- Scemama, O., Hamo-Tchatchouang, E., Le Faou, A. L. & Altman, J. J. (2006). Difficulties of smoking cessation in diabetic inpatients benefiting from a systematic consultation to help them to give up smoking. *Diabetes & Metabolism*, 32(5): 435-441.
- Serour, M., Alqhenaei, H., Al-Saqabi, S., Mustafa, A. R. & Ben-Nakhi, A. (2007). Cultural factors and patients' adherence to lifestyle measures. *Br J Gen Pract*, *57*(537): 291-295.
- Shayo, G. A. & Mugusi, F. M. (2011). Prevalence of obesity and associated risk factors among adults in Kinondoni municipal district, Dar es Salaam Tanzania. *BMC Public Health*, 11(1): 1-20.
- Shephard, R. J. & Balady, G. J. (1999). Exercise as cardiovascular therapy. *Circulation*, 99(7): 963-972.
- Shera, A. S., Jawad, F., Maqsood, A., Jamal, S., Azfar, M. & Ahmed, U. (2004). Prevalence of chronic complications and associated factors in type 2 diabetes. *JPMA*. *The Journal of the Pakistan Medical Association*, *54*(2): 54-59.
- Sicree, R. A. (2009). Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Research and Clinical Practice*, 87(1): 4-14.
- Siminialayi, I. M., Emem-Chioma, P. C. & Dapper, D. V. (2008). The prevalence of obesity as indicated by BMI and waist circumference among Nigerian adults attending family medicine clinics as outpatients in Rivers State. *Nigerian Journal of Medicine*, 17(3): 340-345.
- Singleton, J. R. and Smith, A. G. (2012). Diabetic neuropathy. *Continuum: Lifelong Learning In Neurology*, *18*(1, Peripheral Neuropathy): 60-84.
- Sizer, F. and Whitney, E. (2003). Nutrition Concepts and Controversies 9th ed. Australia: Wardswoth Thomson Learning. pp 25-184.
- Sliwa, K., Damasceno, A. & Mayosi, B. M. (2005). Epidemiology and etiology of cardiomyopathy in Africa. *Circulation*, *112*(23), 3577-3583.
- Sluik, D., Buijsse, B., Muckelbauer, R., Kaaks, R., Teucher, B., Johnsen, N. F., ... & Ardanaz, E. (2012). Physical activity and mortality in individuals with diabetes mellitus: a prospective study and meta-analysis. *Archives of Internal Medicine*, 172(17): 12851295.
- **Sodjinou**, R., Agueh, V., Fayomi, B. & Delisle, H. (2008). Obesity and cardio-metabolic risk factors in urban adults of Benin: relationship with socio-economic status, urbanisation, and lifestyle patterns. *BMC Public Health*, 8(1): 1-43.

- **Soläng**, L., Malmberg, K. & Ryden, L. (1999). Diabetes mellitus and congestive heart failure. *European Heart Journal*, 20(11): 789-795.
- Song, Y. M., Sung, J., Smith, G. D. & Ebrahim, S. (2004). Body mass index and ischemic and hemorrhagic stroke: A Prospective Study in Korean Men. *Stroke*, *35*(4): 831-836.
- Stamler, J., Vaccaro, O., Neaton, J. D., Wentworth, D. & Multiple Risk Factor Intervention Trial Research Group. (1993). Diabetes, other risk factors, and 12-yr cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial. *Diabetes Care*, 16(2): 434-444.
- Steyn, K., Sliwa, K., Hawken, S., Commerford, P., Onen, C., Damasceno, A., ... & Yusuf, S. (2005). Risk factors associated with myocardial infarction in Africa the INTERHEART Africa study. *Circulation*, 112(23): 3554-3561.
- Stout, R. W. (1990). Insulin and atheroma: 20-yr perspective. *Diabetes Care*, 13(6): 631-654.
- Sudha, V., Shukla P. and Patidar P. (2008). Prescribing Pattern of Antidiabetic Drugs in Indore City Hospital Indian. *Journal of Pharmaceutical Sciences*, 70 (5):637-640.
- Swain, J. F., McCarron, P. B., Hamilton, E. F., Sacks, F. M., & Appel, L. J. (2008). Characteristics of the diet patterns tested in the optimal macronutrient intake trial to prevent heart disease (OmniHeart): options for a heart-healthy diet. *Journal of the American Dietetic Association*, 108(2): 257-265.
- Swartz, A. M., Strath, S. J., Bassett, D. R., Moore, J. B., Redwine, B. A., Groër, M., & Thompson, D. L. (2003). Increasing daily walking improves glucose tolerance in overweight women. *Preventive Medicine*, 37(4), 356-362.
- Taubert, K. A., Clark, N. G., & Smith, R. A. (2007). Patient-centered prevention strategies for cardiovascular disease, cancer and diabetes. *Nature Clinical Practice Cardiovascular Medicine*, 4(12): 656-666.
- Tekes-Manova, D., Israeli, E., Swartzon, M., Gordon, S., Heruti, R., Ashkenazi, I., & Justo, D. (2006). The prevalence of reversible cardiovascular risk factors in Israelis aged 25–55 years. *New England Journal of Medicine*, 34(7.1): 34-71.
- **Tekes-Manova**, D., Tirosh, A., Shai, Israeli, E., Pereg, D., Shochat, T., ... & Rudich, A. (2005). Normal fasting plasma glucose levels and type 2 diabetes in young men. *New England Journal of Medicine*, 353(14): 1454-1462
- **Thiam**, M. and co- workers (2003). Cardiac insufficiency in the African cardiology milieu. *Bulletin de la Societe de pathologie exotique 96*(3): 217-218.
- **Thomas** M.C., Zimmet P. & Shaw J.E. (2006). Identification of obesity in patients with type 2 diabetes from Australian primary care: the NEFRON-5 study. *Diabetes Care*. 29(12):2723-2725.

- **Titty**, K.F. (2009).Clinical, Metabolic and Immunological Characteristics of Ghanaian patients with diabetes mellitus: A thesis submitted to the department of Molecular Medicine, KNUST in fulfilment of the requirement for the degree of Doctor of Philosophy in Chemical Pathology.
- Tribble, D. L. & Nutrition Committee. (1999). Antioxidant Consumption and Risk of Coronary Heart Disease: Emphasis on Vitamin C, Vitamin E, and β-Carotene A Statement for Healthcare Professionals From the American Heart Association. *Circulation*, 99(4): 591-595.
- **Tseng**, C. H., Chong, C. K., Sheu, J. J., Wu, T. H. & Tseng, C. P. (2005). Prevalence and risk factors for stroke in Type 2 diabetic patients in Taiwan: a cross-sectional survey of a national sample by telephone interview. *Diabetic Medicine*, 22(4): 477-482.

The second

- **Tseng**, C. H., Tseng, C. P., Chong, C. K., Cheng, J. C. & Tai, T. Y. (2006). Independent association between triglycerides and coronary artery disease in Taiwanese type 2 diabetic patients. *International Journal of Cardiology*, *111*(1): 80-85.
- Tsigos, C., Hainer, V., Basdevant, A., Finer, N., Fried, M., Mathus-Vliegen, E., ... & Toplak, H. (2008). Management of obesity in adults: European clinical practice guidelines. *Obesity Facts*, 1(2): 106-116.
- Tunstall-Pedoe, H., Woodward, M., Tavendale, R., A'Brook, R., & McCluskey, M. K. (1997). Comparison of the prediction by 27 different factors of coronary heart disease and death in men and women of the Scottish Heart Health Study: cohort study. *BMJ*, 315(7110): 722-729.
- **Tuomilehto**, J., Jousilahti, P., Rastenyte, D., Moltchanov, V., Tanskanen, A., Pietinen, P., & Nissinen, A. (2001). Urinary sodium excretion and cardiovascular mortality in Finland: a prospective study. *The Lancet*, *357*(9259): 848-851.
- Turner, R. C., Millns, H., Neil, H. A. W., Stratton, I. M., Manley, S. E., Matthews, D. R. & Holman, R. R. (1998). Risk factors for coronary artery disease in non-insulin dependent diabetes mellitus: United Kingdom Prospective Diabetes Study (UKPDS: 23). BMJ, 316(7134): 823-828.
- Ulasi, I. I., Ijoma, C. K., & Onodugo, O. D. (2010). A community-based study of hypertension and cardio-metabolic syndrome in semi-urban and rural communities in Nigeria. *BMC Health Services Research*, 10(1): 1-30.
- **Unadike**, B. C., Eregie, A., & Ohwovoriole, A. E. (2011). Prevalence of hypertension amongst persons with diabetes mellitus in Benin City, Nigeria. *Nigerian Journal of Clinical Practice*, *14*(3): 300-302.
- **Upadhyay**, D. K., Palaian, S., Ravi Shankar, P., Mishra, P., & Sah, A. K. (2007). Prescribing pattern in diabetic outpatients in a tertiary care teaching hospital in Nepal. *Journal of Clinical and Diagnostic Research*, *1*(4): 248-255.

- **US Department of Health and Human Services**. (1995). US Department of Agriculture: Nutrition and Your Health: Dietary Guidelines for Americans. pp 1-13.
- Uzu, T., Kimura, G., Yamauchi, A., Kanasaki, M., Isshiki, K., Araki, S. I., ... & Haneda, M. (2006). Enhanced sodium sensitivity and disturbed circadian rhythm of blood pressure in essential hypertension. *Journal of Hypertension*, 24(8): 1627-1632.
- Van der Sande M.A., Milligan P.J., Nyan O.A. (2000). Blood pressure patterns and cerebrovascular risk factors in rural and urban Gambian communities. *J Hum Hypertens*;14: 489-96.
- Van Duyn, M. A. S., & Pivonka, E. (2000). Overview of the health benefits of fruit and vegetable consumption for the dietetics professional: selected literature. *Journal of the American Dietetic Association*, 100(12), 1511-1521.
- van't Veer, P., Jansen, M. C., Klerk, M. & Kok, F. J. (2000). Fruits and vegetables in the prevention of cancer and cardiovascular disease. *Public Health Nutrition*, 3(01), 103107.
- Vazzana, N., Ranalli, P., Cuccurullo, C. & Davì, G. (2012). Diabetes mellitus and Thrombosis. *Thrombosis Research*, *129*(3): 371-377.
- Vedovato, M., Lepore, G., Coracina, A., Dodesini, A. R., Jori, E., Tiengo, A., ... & Trevisan, R. (2004). Effect of sodium intake on blood pressure and albuminuria in Type 2 diabetic patients: the role of insulin resistance. *Diabetologia*, 47(2): 300-303.
- Vuvor, F., Steiner-Asiedu, M., Armar-Klemesu, M. & Armah, S. (2011). Population-based study of diabetes mellitus prevalence and its associated factors in adult Ghanaians in the Greater Accra region. *International Journal of Diabetes in Developing Countries*. 31: 149-154
- Wagle, T. J. (2010). Genderwise comparison of serum creatinine and blood sugar levels in type 2 diabetic patients. *Bombay Hosp J*, 52: 64-68.
- Wahab, K. W., Sani, M. U., Yusuf, B. O., Gbadamosi, M., Gbadamosi, A. & Yandutse, M. I. (2011). Prevalence and determinants of obesity-a cross-sectional study of an adult Northern Nigerian population. *International Archives of Medicine*, 4(1): 1-45.
- Wannamethee, S. G., Lowe, G. D., Rumley, A., Bruckdorfer, K. R. & Whincup, P. H. (2006). Associations of vitamin C status, fruit and vegetable intakes, and markers of inflammation and hemostasis. *The American Journal of Clinical Nutrition*, 83(3): 567574.
- Wee, H. L., Ho, H. K. & Li, S. C. (2002). Public awareness of diabetes mellitus in Singapore. *Singapore Medical Journal*, *43*(3): 128-134.

- Weir, M. R., Hall, P. S., Behrens, M. T. & Flack, J. M. (1997). Salt and blood pressure responses to calcium antagonism in hypertensive patients. *Hypertension*, *30*(3): 422442.
- Weisberg, S. P., Hunter, D., Huber, R., Lemieux, J., Slaymaker, S., Vaddi, K., ... & Ferrante Jr, A. W. (2006). CCR2 modulates inflammatory and metabolic effects of high-fat feeding. *The Journal of Clinical Investigation*, 116(1): 115-124.
- Weisberg, S. P., McCann, D., Desai, M., Rosenbaum, M., Leibel, R. L., & Ferrante, A. W. (2003). Obesity is associated with macrophage accumulation in adipose tissue. *The Journal of Clinical Investigation*, 112(12):1796-1808.
- Weissman, A. J., Ross, P. S., Nathan, D. M., Genuth, S., Lachin, J., & Cefalu, W. T. (2006). Intensive diabetes treatment and cardiovascular disease. *Mass Med Soc*, 354:17511785.
- Wellman, N. S., & Kamp, B. J. (2008). Nutrition in aging. Krause's Food and Nutrition Therapy, 12th ed., Saunders Elsevier, Toronto. pp 45-309.
- Wessel, T. R., Arant, C. B., Olson, M. B., Johnson, B. D., Reis, S. E., Sharaf, B. L., ... & Pepine, C. J. (2004). Relationship of physical fitness vs body mass index with coronary artery disease and cardiovascular events in women. *JAMA*, 292(10): 1179-1187.
- Whiting, D. R., Guariguata, L., Weil, C. & Shaw, J. (2011). IDF diabetes atlas: global estimates of the prevalence of diabetes for 2011 and 2030. *Diabetes Research and Clinical Practice*, 94(3): 311-321.
- Wild, S., Roglic, G., Green, A., Sicree, R. & King, H. (2004). Global prevalence of diabetes estimates for the year 2000 and projections for 2030. *Diabetes Care*, 27(5), 1047-1053.
- Wilkinson, I. B. & McEniery, C. M. (2004). Arterial stiffness, endothelial function and novel pharmacological approaches. *Clinical and Experimental Pharmacology and Physiology*, 31(11): 795-799.
- Williams, P. T. & Hoffman, K. M. (2009). Optimal Body Weight for the Prevention of Coronary Heart Disease in Normal-weight Physically Active Men. *Obesity*, *17*(7): 1428-1434.
- Willis, J. (2002). Potent Brews: A Social History of Alcohol in East Africa 1850-1999.
- Wilmer, W. A., Hebert, L. A., Lewis, E. J., Rohde, R. D., Whittier, F., Cattran, D., ... & Bain, R. P. (2000). Remission of nephrotic syndrome in type 1 diabetes: long-term follow-up of patients in the Captopril Study. *American Journal of Kidney Diseases*, 34(2): 308314.
- Winnick, J. J., Sherman, W. M., Habash, D. L., Stout, M. B., Failla, M. L., Belury, M. A. & Schuster, D. P. (2008). Short-term aerobic exercise training in obese humans with type 2 diabetes mellitus improves whole-body insulin sensitivity through gains in peripheral, not hepatic insulin sensitivity. *The Journal of Clinical Endocrinology & Metabolism*, 93(3), 771-778.

- Womack, L., Peters, D., Barrett, E. J., Kaul, S., Price, W. & Lindner, J. R. (2009). Abnormal skeletal muscle capillary recruitment during exercise in patients with type 2 diabetes mellitus and microvascular complications. *Journal of the American College of Cardiology*, 53(23), 2175-2183.
- Woollard, K. J., & Geissmann, F. (2010). Monocytes in atherosclerosis: subsets and functions. *Nature Reviews Cardiology*, 7(2): 77-86.
- World Bank (2009). World bank list of economies (July 2009). Washington DC, USA: World Bank.
- World Health Organization (WHO) (2008). Diet, nutrition and the prevention of chronic disease. A report of joint expert consultation, Geneva. WHO Technical Report series 916.
- World Health Organization (WHO), J., & Consultation, F. E. (1990). Diet, nutrition and the prevention of chronic diseases. Geneva: World Health Organization. pp 57-152.
- World Health Organization (WHO). Global Diabetes Surveillance. (1997). Source: The National Expert Committee on NCD. Non-communicable diseases in Nigeria. Final report of a national survey. Federal Ministry of Health and Social Services, pp. 25–26.
- World Health Organization WHO (2013). Obesity and overweight. WHO Fact Sheet. World Health Organization. Geneva, Switzerland. Available at: <u>http://www.who.int</u> / media centre/factsheet/
- World Health Organization. (1998). Obesity: preventing and managing the global epidemic: report of a WHO consultation on obesity, Geneva, 3-5 June 1997.
- World Health Organization. (2000). Diabetes Fact sheet N°312
- World Health Organization. (2011). Global status report on non-communicable diseases. pp 145-298.
- World Health Organization. (2012). Obesity: Preventing and Managing the Global Epidemic (No. 894).
- World Health Organization. (2014). Global status report on non-communicable diseases. pp 1-298.
- World Health Organization/Food Agriculture Organization (WHO/FAO) (2003). Diet, Nutrition and the prevention of chronic Disease. A report of joint WHO/FAO expert consultation, Geneva, 28 January – 1<sup>st</sup> February 2002.
- World Health Organization/International Society of Hypertension (WHO/ISH) (2003). Guidelines Sub-Committee Guidelines for the management of hypertension. J Hypertens, 17 1983-1992.

World Heart Federation. (2012). Diet and cardiovascular diseases. pp 1-2.

- Xu, H., Barnes, G. T., Yang, Q., Tan, G., Yang, D., Chou, C. J., ... & Chen, H. (2003). Chronic inflammation in fat plays a crucial role in the development of obesity-related insulin resistance. *The Journal of Clinical Investigation*, 112(12): 1821-1830.
- Yadav, R., Tiwari, P. & Dhanaraj, E. (2008). Risk factors and complications of type 2 diabetes in Asians. *CRIPS*, *9*(2): 8-12.
- Yang, W., Lu, J., Weng, J., Jia, W., Ji, L., Xiao, J., ... & Zhu, D. (2010). Prevalence of diabetes among men and women in China. New England Journal of Medicine, 362(12): 109011011.
- Zavaroni, I., Bonora, E., Pagliara, M., Dall'Aglio, E., Luchetti, L., Buonanno, G., ... & Reaven, G. (1989). Risk factors for coronary artery disease in healthy persons with hyperinsulinemia and normal glucose tolerance. *New England Journal of Medicine*, 320(11):702-706.
- Zheng, W., McLerran, D. F., Rolland, B., Zhang, X., Inoue, M., Matsuo, K., ... & Irie, F. (2011). Association between body-mass index and risk of death in more than 1 million Asians. *New England Journal of Medicine*, 364(8): 719-729.
- **Zhou**, M., Offer, A., Yang, G., Smith, M., Hui, G., Whitlock, G., ... & Chen, Z. (2008). Body mass index, blood pressure, and mortality from stroke a nationally representative prospective study of 212 000 Chinese men. *Stroke*, *39*(3): 753-759.
- Zhu, S., Wang, Z., Heshka, S., Heo, M., Faith, M. S. & Heymsfield, S. B. (2002). Waist circumference and obesity-associated risk factors among whites in the third National Health and Nutrition Examination Survey: clinical action thresholds. *The American Journal of Clinical Nutrition*, 76(4): 743-743.
- Zins, M., Carle, F., Bugel, I., Leclerc, A., Orio, F. D. & Goldberg, M. (1999). Predictors of change in alcohol consumption among Frenchmen of the GAZEL study cohort. *Addiction*, 94(3): 385-400.

W J SANE

### APPENDIX

### **QUESTIONNAIRE FOR: IMPACT OF NUTRITIONAL STATUS ON THE**

# PREVALENCE OF CARDIOVASCULAR DISEASE AMONG DIABETES

MELLITUS IN GHANA IDENTIFICATION

BADH

NUMBER.....

FOLDER NO...... DATE...... (DD/MM/YYYY) TIME....... (HH:MM) PLEASE TICK  $f\sqrt{J}$  OR CIRCLE AS APPROPRIATE

### **OBJECTIVES:**

- 1. To assess the anthropometric data of diabetes mellitus patients
- 2. To determine the prevalence of dyslipidemia among diabetes mellitus patients
- 3. To assess the dietary intake of diabetes mellitus patients
- 4. To assess the physical activity level of diabetes mellitus patients
- 5. To determine prevalence cardiovascular disease in diabetes mellitus patients

# SECTION A: DEMOGRAPHICS CHARACTERISTICS

- 1. Age .....
- 2. Gender
  - a. Male
  - b. Female
- 3. Highest Educational Level
  - a. No formal Schooling
  - b. Basic School
  - c. SSS/A Level completed
  - d. Tertiary completed
  - e. Other (Specify): .....
- 4. Employment status
  - a. Employed
  - b. Unemployed
- 5. Type of employment.....
- 6. Residence:
  - a) Urban
  - b) Semi
  - c) Rural
- 7. Ethnicity:
  - a) Akan

|    | <ul> <li>b) Ew</li> <li>c) Ga</li> <li>d) Hav</li> <li>e) Gru</li> <li>f) Dag</li> </ul> | Isa                      |  |                       |                  |
|----|--|--------------------------|--|-----------------------|------------------|
|    | g) Oth<br>8. Marita  | ers Specify<br>l status: |  | TCT                   |                  |
|    | a) Single  | b.) Married              | c.) Separated  | d.) Divorced          | e.) Widowed      |
| 9. | Level of i   | ncome:                   |  |                       |                  |
| 10 | . How do y   | ou pay your medical      | bill?  |                       |                  |
|    | a) Sel   | f                        |  |                       |                  |
|    | b) Re  | atives                   |  | 14.                   |                  |
|    | c) En  | ployer                   |  |                       |                  |
|    | d) NH  | IS                       | A COLORADO AND A COLO |                       |                  |
|    | e) Otl   | ers specify              |  |                       |                  |
| SE | ECTION B   | DIETARY HABIT            | rs in the second s   |                       |                  |
|    | 11. How 1  | nany times do you us     | ually eat your main  | meal in a day?        |                  |
|    |  |                          |  | ] d) More than T      | Thrice [ ] 12.   |
|    |  | ls do usually prepare    |  |                       |                  |
|    |  |                          |  | pper [] d)            | Nil [ ] 13.      |
|    |  | y times do you usuall    |  |                       |                  |
|    |  |                          |  | ] d) More than Thr    | ice [] e) Nil [] |
|    | 14. In a t   | pical week how man       | iy days do you take f  | ruits?                |                  |
|    |  |                          |  | d) More than Thrice [ | ] e) Nil [ ] 15. |
|    | •  | times do you usually t   |  |                       |                  |
|    |  | When do you eat fruit    |  | More than Thrice [ ]  | e)               |
|    |  |                          |  | ng [] d) Nil []       | 17.              |
|    |  | week how many times      |  |                       | 121              |
|    |  |                          |  | ] d) More than Thr    | ce e) Nil [ ]    |
|    |  | ten do you eat vegetab   | •  |                       | ~/               |
|    |  |                          |  | d) More than Thrice   | e[]e) Nil What   |
| ,  | • -  | fat is often used for    |  |                       |                  |
|    |  | oil (unsaturated) oliv   |  |                       |                  |
| b) | -  | · · · ·                  |  | ut oil name           |                  |
|    | -  |                          | ien cooking? a) Y  | es [ ] b) No          | [ ] If no go to  |
|    | questi   |                          | 4 dagarihan wave asl4  | intolvo 9             |                  |
|    |  | of the following bes     | •  |                       |                  |
|    | a) L(  | w[] b) M                 | oderate [ ]  | c) High [ ]           |                  |

- 21. Do you add table salt to already served meal cooked food at table? a) Yes [ ] b) No [ ]
- 22. How often do you consume canned foods?

a) Never b) Daily [ ] c) Weekly [ ] d) Monthly [ ] e) Occasionally [ ]

### SECTION C: PHYSICAL ACTIVITY

- 23. How often do you exercise in a day?
- a) Once [] b) Twice [] c) Thrice [] d) More than Thrice [] e) Nil [] 25. What type of exercise do you do?
- a.) vigorous-intensity e.g. running, basketball, bicycling, swimming and football

b) moderate-intensity (slow dancing, brisk walking, bicycling at a regular pace or table tennis)

24. In a typical week, on how many days do you do exercise state No. of Days

a) One day [ ] b) Two days [ ] c)Three days [ ] d) Four days [ ] e) All days [ ]

- 25. How much time do you spend exercising On a typical day? Number of hr / min: ...
- 26. Does your work involve vigorous-intensity activities (e.g. chopping wood, lifting heavy loads, farming, digging or construction work) that causes large increase in breathing or heart rate for at least 10 minutes continuously?
  - a) Yes [ ] b) No [ ]
- 27. In a typical week, on how many days do you vigorous- intensity activities (e.g. carrying light loads, gardening/yard work) as part of your work? [.....]
- 28. How long do you spend doing vigorous-intensity activities on a typical day?
- 29. Does your work involve moderate-intensity activities (e.g. carrying light loads, gardening/yard work) that causes small increase in breathing or heart rate for at least 10 minutes continuously?
  - a)Yes [

1

b) No [ ]

- 30. In a typical week, on how many days you do moderate-intensity activities exercise (e.g. carrying light loads, gardening/yard work) as part of your work?.....
- 31. How long do you spend doing moderate-intensity activities on a typical day?
- 32. Do you walk or use a bicycle for at least 10 minutes continuously to get to and from places? Yes [ ] No [ ]
- 33. In a typical week, on how many days do you walk or bicycle for at least 10 minutes continuously to get to and from places? Number of days [\_\_\_\_\_]
- 34. How much time do you spend walking or bicycling for travel on a typical day? Hours : minutes
- 35. How much time do you usually spend sitting or reclining on a typical day? Number of hr / min .....

#### SECTION D: FAMILY AND MEDICAL HISTORY

- 36. Does any member of your family have Diabetes Mellitus?
  - a) Yes [ ] b) No [ ] c) Don't know [ ]

|      | If yes, how do you relate to the person?  |
|------|---|
| 38.  | Does your family know about your condition Diabetes? a) Yes [ ] b) No [ c) Don't know [ ] |
| 39.  | If yes, do you receive any support from them? a) Yes [ ] b) No [ ]                        |
| 40.  | How long have you been diagnosed with diabetes?   |
| 41.  | Do you currently use any form of diabetic medication? a)Yes [ ] b) No [                   |
|      | 42. If yes which of these do you use?   |
| a)   | Glineperide only  |
| b)   | Metformin only  |
| c)   | Glibenclamide only  |
| d)   | Insulin only  |
| e)   | Glibenclamide and Metformin   |
| f)   | Gliclazide and Metformin  |
| g)   | Insulin with oral medication  |
| h)   | Others specify  |
| 43.  | If yes how long have you been using it?   |
| 44.  | Are you using any herbal medication?  |
|      | a) Yes [ ] b)No [ ]   |
|      | If yes how long? <u>CO-MORBIDITY</u>  |
| 45.  | Do you have any of these diseases?  |
|      | a) Hypertension [ ] b) Stroke [ ] c) Heart failure/attacks                                |
|      | c) If others, state type  |
| 46.  | When was/ were these diseases diagnosed?  |
| a.   | Strokewhere   |
| b.   | Heart failure/attack  |
| c.   | Hypertension where  |
| 47.  | Are you on any medication for any of these diseases?                                      |
| 48.  | If yes state the medication   |
| 49.  | Duration of treatment   |
|      | 7777  |
| FF 9 | STYLE: SMOKING STATUS   |

| 50. | Which of the following best describes your smoking status?                   |      |     |
|-----|--|------|-----|
|     | a) Never smoked [ ] b) Current smoker [ ] c) Ex-smoker [                     | [ ]  | ]   |
|     | If never smoked go to 56, if ex-smoker go to 55                              |      |     |
| 51. | Do you smoke any of the tobacco products? Such as pipes or cigarettes daily? | a)Ye | s [ |
|     | ] b)No [ ]   |      |     |
| 52. | If yes, how long have you been smoking?                                      |      |     |

**53.** What is the average number of cigarettes Smoked per day? .....

54. If you quit for how many years did you smoke?

# LIFE STYLE: ALCOHOL CONSUMPTION

55. Which of the following best describes your drinking status?

- a) Never drink [ ] b) Current drinker [ ] c) Ex –drinker [ ] **If** never drink skip to FFQ checklist, if ex-drinker go to 61
- - a) Once [ ] b) Twice [ ] c) Thrice [ ] d) More than thrice
- 58. Have you consumed alcohol such as beer, wine, spirits akpeteshi, palm wine, pito, fermented cider, other alcoholic bitters or any other drink within the past seven days?a) Yes [ ]b) No [ ]
- 59. In the past 3 months, how frequently have you had at least one drink?
- a) Nil [ ] b)Daily [ ] b) 5-6 dys per week [ ] c) 1-4 dys per week [ ] d)1-3 dys per month [ ]
- 60. Which of the following describes your drinking pattern?
- a) Daily [ ] b) Weekly [ ] c) Monthly [ ] d) Occasionally [ ] 61. What age did you quit drinking?

#### SECTION F: ANTROPOMETRY AND MEASUREMENT

| ANTHROPOMETRY             | READING | UNIT        |
|---------------------------|---------|-------------|
| Weight                    |         |             |
| Height                    |         |             |
| Waist circumference       | 7       |             |
| Hip circumference         |         |             |
| BODY COMPOSITION          |         | 1 3 3 3     |
| Percentage body fat       |         | 1373        |
| Visceral fat              | 3-6     | 200         |
| BLOOD PRESSURE            | 730 X-W | 224         |
| Systolic                  | 2       | and and and |
| Diastolic                 | 11      |             |
| LIPID PROFILE             |         |             |
| Total cholesterol         |         |             |
| Triglycerides             | 1000    |             |
| High density lipoproteins | 20      |             |
| Low density lipoproteins  |         |             |

### FOOD FREQUENCY CHECKLIST

I am going to read out a list of various foods. Please tell me how many times you eat them on

| High Fibre Starches | Daily | 3-5 times | Weekly | Monthl | Occasio | Never |  |
|---------------------|-------|-----------|--------|--------|---------|-------|--|
|                     |       | per week  |        | у      | nally   |       |  |
| Oats                |       |           |        |        |         |       |  |
| Tom brown/weanimix  |       |           |        |        |         |       |  |

#### an average every week.

| Wheat bread/Wheat     |         |         |      |     |       |
|-----------------------|---------|---------|------|-----|-------|
| Kenkey                |         |         |      |     |       |
| Others                |         |         |      |     |       |
|                       |         |         |      |     |       |
|                       |         |         |      |     |       |
| Low Fibre Diets       |         |         |      |     |       |
| Banku                 |         |         | 10   |     |       |
| Fufu                  |         |         |      | . I |       |
| Konkonte              |         | VI      | 1    |     |       |
| Plain rice/Rice balls | - N. II |         |      |     |       |
| Waakye                |         | 2       |      |     |       |
| Sugar bread           |         |         |      |     |       |
| Tea bread             |         |         | 6    |     |       |
| Butter bread          |         | 110     |      |     |       |
| Boiled plantain       |         |         |      |     |       |
| Boiled ripe Plantain  | 57      | 1       | - 4  |     |       |
| Boiled yam            | 4       |         |      |     |       |
| Gari                  |         |         |      |     |       |
|                       |         | 10      |      |     |       |
| Hausa koko            |         |         |      |     |       |
| Koko (corn)           | 1       | ALC NO. |      |     | 1     |
| Tuo-zaafi             |         |         |      | 1   | <br>5 |
| Others                |         |         | -8-1 | 0   |       |
|                       | -       |         |      |     |       |

|     |               | 1   | 1    |     | 1 |
|-----|---------------|-----|------|-----|---|
| -   | 15            | -5- | 0    | 1   |   |
| -   |               | 51  | 30   | -   |   |
| 2   |               |     | Cal- |     |   |
| 1-1 |               | 5   | 0    | 1   |   |
| 8-  |               | an  | -    | 1   |   |
| CM. | 1             |     | -    |     |   |
| LAS | 151           |     |      |     |   |
|     | 111           | 1   |      | 1.2 |   |
| /   |               |     |      | /   |   |
| 2   |               |     |      |     |   |
| 5   | 2             |     | ÷    | 13  | / |
|     |               |     |      | 54/ |   |
|     |               | -   | 0    | /   |   |
|     |               | N   | 8    |     |   |
| 20  |               | 50  | 2    |     |   |
|     | ANE           |     |      |     |   |
|     |               |     |      |     |   |
|     |               |     |      |     |   |
|     |               |     |      |     |   |
|     |               |     |      |     |   |
|     |               |     |      |     |   |
|     | S MARKEN NAME |     |      |     |   |

| Fried chicken with skin          |          |     |     |    |   |   |
|----------------------------------|----------|-----|-----|----|---|---|
| Grilled/boiled chicken with skin |          |     |     |    |   |   |
| orphals                          |          |     |     |    |   |   |
| Crabs                            |          |     |     |    |   |   |
| Shrimps                          |          |     |     |    |   |   |
| Domedo                           | niae tae |     |     |    |   |   |
| Others                           |          |     | 6   |    |   |   |
|                                  |          |     |     |    |   |   |
| Foods Low in                     |          | VU  |     |    |   |   |
| Saturated Fats and               |          |     |     |    |   |   |
| Cholesterol                      |          | 1.2 |     |    |   |   |
| Grilled/boiled chicken without   |          |     |     |    |   |   |
| skin                             |          |     | 6   |    |   |   |
| Smoked/Grilled fish              |          |     |     |    |   |   |
| Boiled egg                       | N        |     | NA. |    |   |   |
| Skimmed meat                     | 1        | 1   | 13  |    |   |   |
| Cooked lean meat                 |          |     |     |    |   |   |
| Others                           |          |     |     |    |   |   |
|                                  | 1        | 10X | 100 |    |   |   |
| Nuts And Seeds                   |          | -   |     |    |   | 1 |
| Cooked beans                     | 4        |     | 1   | 1  | _ |   |
| Roasted Groundnuts               | 1        | V   | 3   | 5  | 5 | 2 |
|                                  | -        | 0   | DI  | 57 | - | J |

| Roasted cashew nuts    | 2~~ |        | ~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~ |     | 2 |     |
|------------------------|-----|--------|---|-----|---|-----|
| Agushie                | 2   |        | R                                       | S   |   |     |
| Others                 | 1   |        | 3                                       | 1   | × |     |
|                        | 1   |        |   |     |   |     |
| High Sodium Foods      | S   | 5      |   |     |   |     |
| Kobi                   |     |        |   |     |   |     |
| Kako                   |     |        |   | 27  |   |     |
| Salted pig feet        | 6   |        |   |     |   | - 1 |
| Toolo beef             | -   |        |   |     | X |     |
| Momoni                 |     |        | -                                       |     | 5 |     |
| Sardine                |     |        |   | 10  | 2 |     |
| Corned beef            | 1   |        | V                                       | and |   |     |
| Maggi                  |     |        |   |     |   |     |
| Jumbo                  | 25  | ANE    | 202                                     | >   |   |     |
| Royco                  | _   | 11 11- |   |     |   |     |
| Onga                   |     |        |   |     |   |     |
| Tinned vegetable salad |     |        |   |     |   |     |
| Others                 |     |        |   |     |   |     |
|                        |     |        |   |     |   |     |
| Fruits                 |     |        |   |     |   |     |

| Pawpaw                      |        |       |   |       |     |   |
|-----------------------------|--------|-------|---|-------|-----|---|
| Orange                      |        |       |   |       |     |   |
|                             |        |       |   |       |     |   |
| Mango<br>Banana             |        |       |   |       |     |   |
|                             |        |       |   |       |     |   |
| Water melon                 |        |       |   |       |     |   |
| Pineapple                   | 1 10   |       | 1.2                                     | -     |     |   |
| Apple                       | / P    |       |   | S     |     |   |
| Others                      |        |       |   | N -   |     |   |
|                             |        | NC    | 1 -                                     | 2 -   |     |   |
| Vegetables                  |        |       |   |       |     |   |
| Cabbage                     |        |       |   |       |     |   |
| Carrot                      |        |       |   |       |     |   |
| Cucumber                    |        |       | (                                       |       |     |   |
| Fresh tomatoes              |        |       | 1                                       |       |     |   |
| Kotonmire                   | - A. I |       |   |       |     |   |
| Garden eggs                 |        | 11    | 1                                       |       |     |   |
| Okro                        |        |       | Same Sec                                |       |     |   |
| Bra                         |        |       |   |       |     |   |
| Ауоуо                       |        | 6     |   |       |     |   |
| Pumpkin leaves              |        |       |   |       |     |   |
| Alefu                       | × .    |       |   | 1.00  |     | 1 |
| Bitter leaf                 |        |       | 1                                       | 1     |     | 1 |
| Foods High in Refined Sugar |        | 12    | -8-1                                    | 2     | 1   |   |
|                             | -1     |       | DI.                                     | 37    | -   |   |
| Coca cola                   | 3-6    |       | ~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~ | Color | 7   |   |
| Biscuits                    | 1      |       | La l                                    | 0     |     |   |
| Cakes                       | 1      |       | حدد                                     | -     | ~   |   |
| Sprite                      | 1      | 10    | 1.0                                     |       |     |   |
| Fanta                       | 100    | 10    |   |       |     |   |
| Sugar cane                  |        | 227   | 1                                       |       | 1.2 |   |
| Don Simon/Ceres Minute      | /      | 7     | -                                       |       | 1   |   |
| maid/ etc.                  | -      |       |   |       | 1   | 1 |
| Malt                        | . ~    |       | 1                                       |       | 12  | 1 |
| E JE                        |        |       |   |       | 2   |   |
| Others                      |        |       |   |       | 21  |   |
| AN                          |        |       | 1                                       | all   | /   |   |
| Alcoholic Beverages         |        |       |   | -     |     |   |
| Bear                        | 25     | A LUT | NO                                      | >     |     |   |
| Guinness                    |        | ALLE  | -                                       |       |     |   |
| Spirit                      |        |       |   |       |     |   |
| Wine                        |        |       |   |       |     |   |
| Punch                       |        |       |   |       |     |   |
|                             |        |       |   |       |     |   |
| Others                      |        |       |   |       |     |   |