

**PREVALENCE OF OBESITY, HYPERGLYCAEMIA AND DYSLIPIDAEMIA AND
ASSOCIATED FACTORS AMONG SENIOR STAFF OF THE UNIVERSITY
COLLEGE HOSPITAL, IBADAN**

BY

OMOLARA OLUTOSIN, POPOOLA

BMLS (Ekpoma), MSc Chemical Pathology (Ibadan)

MATRIC NO: 136554

A Dissertation in the Department of Epidemiology and Medical Statistics

Submitted to the Faculty of Public Health

In partial fulfilment of the requirements for the degree of

MASTER OF SCIENCE (EPIDEMIOLOGY)

of the

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CERTIFICATION

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DEDICATION

This research is dedicated to the Almighty God and the participants of this study.

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LIST OF ABBREVIATIONS

BMI- Body mass index

% BF- percentage body fat

CHD- Coronary heart disease

CVD- Cardiovascular disease

CVDs- Cardiovascular diseases

DM- Diabetes mellitus

HDL-C- High density lipoprotein cholesterol

LDL-C- Low density lipoprotein cholesterol

TG- Triglycerides

TC- Total cholesterol

WC- Waist circumference

IFG- Impaired fasting glucose

IGT-Impaired glucose tolerance

WHO- Who health organisation

ADA- American diabetic association

GH-Growth hormones

VLDL- very low density lipoproteins

FPG- Fasting plasma glucose

NHS-National health scheme insurance

MLS-Medical Laboratory Scientists

HRO- Health Records Officer

MSW- Medical Social Worker

MSE-Medical scientific equipment

NCDs- Non-communicable diseases

UCH- University College Hospital

UI- University of Ibadan

IRB-Institutional Review Board

NCEP- National Cholesterol Education Program

HND – Higher National Diploma

RN- Registered Nursing Certificate

RM- Registered Midwife

BSc- Bachelor of Science

MSc- Masters of Science

PhD- Doctor of Philosophy

NCEP- National Cholesterol Education Program

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ABSTRACT

Cardiovascular diseases (CVDs) are the leading causes of deaths due to non-communicable diseases worldwide. Obesity, dyslipidaemia and diabetes mellitus are diseases associated with high risk for CVDs. Parallel changes in lifestyle and diet in developing countries have led to a greatly increased burden. Epidemiological data on the prevalence and associated factors of obesity, dyslipidaemia and diabetes mellitus in senior public officers in Nigeria is rare. This study aimed to determine the prevalence and associated factors of obesity, hyperglycaemia and dyslipidaemia among senior staff of the University College Hospital, Ibadan as well as determine the pattern of clustering of obesity, hyperglycaemia, dyslipidaemia and hypertension.

A cross sectional study was carried on 371 senior staff at the University College hospital, Ibadan. A self-administered semi-structured questionnaire was used to obtain information on demographic characteristics, occupational data, physical activity, lifestyle/diet, medical drug history, anthropometric measurements, blood pressure and laboratory investigations. Data was analysed using SPSS 19.0 to determine descriptive statistics, Chi-square test and logistic regression at $p < 0.05$.

The mean age of the participants was 41.5 ± 7.6 years. The prevalence of obesity was 34.8%, abdominal obesity, 70.9%, impaired fasting glucose, 5.1%, diabetes, 1.6%, hypercholesterolaemia, 33.2%, hypertriglyceridaemia, 2.7%, decreased high density lipoprotein cholesterol (HDL), 45.3% and increased low density lipoprotein cholesterol (LDL), 39.4%. Gender, age, marital status, first degree relatives of diabetics and cadre were factors found to be associated with obesity. Gender, age, marital status and cadre were factors found to be associated with abdominal obesity. Age, obesity and abdominal obesity showed significant associations with impaired fasting glucose (IFG). The associated factors of diabetes include age, physical stress, hypertension, family history of diabetes and first degree relative history of diabetes. Age, obesity, abdominal obesity, IFG, alcohol consumption, hypertension and cadre were observed to be associated with hypercholesterolaemia and high LDL. Age was the only factor associated with the occurrence of hypertriglyceridaemia in this study. Marital status, obesity, abdominal obesity, being engaged in physical activity, sports activity and sedentary lifestyle were found to be

associated factors associated with low HDLC. Approximately 64% of the participants of this study had a combination of 2 or more of the factors (obesity, hyperglycaemia, dyslipidaemia and hypertension). The most common pattern of clustering was obesity and dyslipidaemia.

The high prevalence of obesity, dyslipidaemia, physical inactivity and low level of sports activity in health workers who are supposed to be role models suggest a negative potential impact on diffusion of knowledge and measures to control cardiovascular diseases in the general population.

Keywords: obesity, hyperglycaemia, dyslipidaemia, associated factors

Word count: 425

CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND

Obesity, dyslipidaemia and diabetes mellitus are associated with high risk for cardiovascular diseases (Yusuf et al., 2004; Powers, 2007). Cardiovascular disease is any disorder, abnormality or failure to function well, relating to the heart and blood vessels or the circulation (Boon et al., 2002). Cardiovascular diseases (CVDs) are prevalent conditions which impose significant negative impacts on the healthcare system (Singh et al., 2008). Over the past decade, CVDs have become the single largest cause of death worldwide, representing nearly 30% of all deaths and about 50% of non-communicable disease deaths (WHO, 2011a). Cardiovascular diseases which were noted as being the leading cause of morbidity and mortality only in the western world are now emerging public health challenges in developing countries (WHO 2002a). Presently, about 80% of the CVD deaths are from low- and middle-income countries (WHO, 2011a). Parallel changes in lifestyle and diet in developing countries have led to an increase in life expectancy and a greatly increased burden of cardiovascular disease and other chronic diseases including obesity and diabetes (Popkin et al., 2001; Yusuf et al., 2001; Wu et al., 2001).

Obesity is recognized worldwide as a serious health problem (WHO, 1998). The World Health Organization (WHO) defines it as a condition with excessive fat accumulation in the body, to the extent that health and well-being are adversely affected (WHO, 1998). In 2008, more than 1.4 billion adults were overweight worldwide, and of these over 200 million men and nearly 300 million women were obese (WHO, 2011). Overweight and obesity are very common in Europe (Bianchini et al., 2002) and are one of the most important health problems in the USA (Gutiérrez-Fisac et al., 2000; Flegal et al., 2002). The prevalence of overweight and obesity is increasing at an alarming rate in the developed industrialized world and also in the developing countries (Ariyo, 2002; Cameron et al., 2003). Suggested reasons for increasing obesity include reduction in physical activity, increased consumption of energy-dense diet, increased television viewing hours, and individuals transitioning from traditional lifestyle to rapid urbanization (Salmon et al., 2000; Cameron et al., 2003). The transitions have occurred in developing countries as the economy becomes more resourceful, causing significant shifts in dietary and physical activity patterns (Misra and Kharango, 2008). In 2005, the WHO reported that the prevalence of obesity in sub-Saharan African

countries ranges between 3.3% and 18.0%, and that obesity has become a leading risk factor for diabetes mellitus and cardiovascular diseases in the urban areas of Africa (WHO, 2005). Studies from the United States and Europe have shown that obesity is closely associated with increased risks of disease and reduced life expectancy (Flegal et al., 2002). The burden of the medical complications of obesity threatens to overwhelm health services (WHO, 1998a). Worldwide, an estimated 35.8 million (2.3 per cent) of global disability adjusted life years (DALYs) are caused by overweight or obesity and at least 2.8 million people die each year as a result of being overweight or obese (WHO, 2011). Obesity has been shown to be an independent risk factor for coronary heart disease and the insulin resistance associated with obesity contributes to the development of other cardiovascular risk factors, including type 2 diabetes, dyslipidaemia and hypertension (McFarlane et al., 2001; Peppa et al., 2003). Overweight and obesity have been related to increased morbidity and mortality rates due to diabetes mellitus, hypertension, dyslipidaemia, several forms of cancer, digestive diseases and coronary heart disease (National Institute of Diabetes and Digestive and Kidney Diseases, 2000).

Diabetes prevalence is on the rise globally. The rising prevalence in diabetes is ascribed to lifestyle changes and urbanization, with the data now showing that the strongest and more consistent risk factors are obesity and weight gain (Idemayor, 2011). Of all the obesity-associated co-morbidities diabetes may be the most closely linked to obesity and its prevalence appears to increase as the prevalence of obesity increases (Janghorbani et al., 2007). Even though the prevalence of such infectious diseases as HIV/AIDS, malaria, and tuberculosis is galvanizing the health economy, diabetes seems to be the world's most threatening epidemic, which is beginning to submerge the developing world (Azevedo and Alla, 2008). The number of people with diabetes is increasing worldwide due to population growth, aging, urbanization, and increasing prevalence of obesity and physical inactivity (Wild et al., 2004). For a long time Africa was considered safe from diabetes (Azevedo and Alla, 2008) however epidemiological studies carried in the last decade of the 20th century have provided evidence of a different picture (Mbanya and Gill, 2004) as there are reports that there is a global trend towards the increase of the incidence and prevalence of diabetes in African populations (Sobngwi et al., 2001). With rising rates of obesity consequences of obesity other than diabetes are showing increasing prevalence.

Dyslipidaemia, abnormalities in the levels of plasma lipids, is indicated by total cholesterol greater than 200mg/dl (hypercholesterolemia) or triglycerides greater than 150mg/dl

(hypertriglyceridemia) or HDL cholesterol less than 40 mg/dl in men and postmenopausal women or less than 50 mg/dl in premenopausal women or LDL-cholesterol greater than 130 mg/dl (NCEP, 2001). Dyslipidaemia is a common health problem worldwide (WHO, 2002; He, 2004), the prevalence of which is rising steadily (Schaefer, 2002) even in developing countries (Souza et al., 2003). Dyslipidaemia is an independent and modifiable risk factor for cardiovascular disease (WHO, 2002). It is the second most prevalent cardiovascular risk factor after sedentary lifestyle (Tekes-Manova et al., 2006). Research over the past four decades has consistently shown the burden of dyslipidaemia to be very high in terms of morbidity, mortality, and medical costs (WHO, 2002). WHO estimates in 2002 showed that dyslipidaemia accounted for 18% of ischaemic heart disease, 56% of stroke and more than 4 million deaths per year.

Dyslipidemia was previously thought to be rare in Africa and recent findings show that the current state of dyslipidaemia clearly contradicts previous perceptions (Oguejiofor et al., 2012). The gap in prevalence of dyslipidaemia in Africa compared to Caucasians is not only closing, the high prevalence values currently obtained is comparable to Caucasian values (Oguejiofor et al., 2012). This abnormal lipid metabolism has been associated with urbanisation and westernisation (Njeleka et al., 2002). Recent studies in Nigeria have reported dyslipidaemia in the range of 59.3 to 60.5% (Agboola-Abu and Onabolu, 2000; Odenigbo et al., 2008; Osuji et al., 2010; Sani et al., 2010). Odenigbo et al., in a study on the prevalence of dyslipidaemia in apparently healthy professionals in Asaba reported that 5% of the study population had hypertriglyceridemia, 23% elevated total serum cholesterol, 51% elevated LDL-cholesterol and 60% low HDL-cholesterol. Another study, in Naze, Owerri- North Local Government Area of Imo State, on women of different socioeconomic classes found that the prevalence of hypertriglyceridemia to be 34.1%, hypercholesterolemia, 31.4% and low HDL, 37.6% (Osuji et al., 2010).

1.2 PROBLEM STATEMENT

In 2008, 9.8 % of men and 13.8 % of women, globally, were obese compared to 4.8 % of men and 7.9 % of women in 1980 (WHO, 2011). The increasing epidemic of obesity throughout the world is creating concern because of its negative impact on morbidity and mortality. Obesity has a direct correlation with related co-morbidities; dyslipidaemia, type 2 diabetes mellitus, hypertension, the metabolic syndrome, and cardiovascular disease (Gupta et al. 2002; Gupta and Misra, 2007). Worldwide at least 2.8 million annual deaths and an estimated 35.8 million (2.3 per cent) disability adjusted life years are attributed to overweight and obesity (WHO, 2011). The prevalence of diabetes worldwide was approximately 2.8% in 2000 and will be 4.4% in 2030 (Wild et al., 2004; Danaei et al., 2011) and was responsible for 1.3 million deaths globally in 2008 (WHO, 2011). Dyslipidaemia is associated with more than half of global cases of ischemic heart disease and more than 4 million deaths per year (WHO, 2002). Urbanization is usually associated with increased sedentary lifestyle and prevalence of CVD risk factors such as hypertension, obesity and dyslipidaemia (Vorster, 2002).

Significant changes in the form of rapid urbanization have led to automobile dominance for the workplace, surplus availability of energy dense calorie foods, satellite TV, increased reliance on computers and telecommunication technology as well as decreased occupational-work demands (Jones et.al, 2007). These changes have had a remarkable lifestyle transformation by reducing the daily life physical requirements and encouraging sedentary lifestyles which consequently lead to an epidemic of non communicable diseases and contributes substantially to the global burden of disease, disability and death (U.S. Department of Health and Human Services ,1996).

Studies have shown individuals can spend more than half of their waking hours in sedentary activities (Matthews et. al., 2008). Population research using self-report of sitting (Katzmarzyk et.al., 2009) or television viewing times (Dunstan et.al., 2004) has found that as sitting or television viewing time increases there is an increased cardio-metabolic risk, independent of moderate/vigorous physical activity (Sedentary Behaviour Research Network, 2012). A greater proportion of the workforce is now employed in low activity occupations such as office work (Parry and Straker, 2013). With the evolution of the "technology age", sedentary time is reported to be increasing (Borodulin et.al., 2007; Brownson et.al., 2005; Matthews et.al., 2008).

. There is increasing prevalence of sedentary occupations and the potential contribution of work to sedentary risk (Straker and Mathiassen, 2009). Work is an important contributor to overall weekly sedentary exposure for office workers and thus to their associated health risks (Healy et.al., 2008; 2011)

Nigeria is one of the countries that have experienced rapid urbanisation (Oladunjoye, 2005; Jiboye, 2009; Olotuah and Bobadoye, 2009) with consequent lifestyle changes (Ekezie et al., 2011; Akarolo-Anthony and Adebamwo, 2012) however little is known about the prevalence of obesity and associated co-morbidities such as hyperglycaemia and dyslipidaemia of individuals of high socioeconomic status with a predisposition to a sedentary lifestyle (Odenigbo et al., 2008; Ugwuja et al., 2013).

1.3 JUSTIFICATION OF STUDY

The problems of obesity, hyperglycaemia and dyslipidaemia have emerged as pressing public health issues that the global community will face for the next several decades (Nguyen and El-Serag, 2010). Obesity and diabetes are easy medical condition to recognize but the most difficult to control, early intervention is therefore necessary (Davies, 2008). The fundamental causes of the obesity, hyperglycaemia and dyslipidaemia epidemic are sedentary lifestyles and high-fat energy-dense diets, both resulting from the profound changes taking place in society and the behavioural patterns of communities as a consequence of increased urbanization and industrialization and the disappearance of traditional lifestyles (WHO, 2000). Ibadan is one of the cities in Nigeria that have experienced rapid urbanisation and industrialisation (GKW, 2003).

Majority of civil servants do not engage in heavy occupational activities with senior staff tending to have a lower level of occupational activities than junior staff (Forrest et al., 2001). Senior staff are often administrators and their job description requires that they sit for long stretches of time, hold meetings and assess documents (Forrest et al., 2001; Banwat et al., 2012). They therefore have the propensity to be overweight and have associated diseases as the activities required by them in the office require expenditure of low calories (Banwat et al., 2012). Civil servants also engage in snacking, eat foods rich in carbohydrate, protein and fat, eat between meals, prefer fried to boiled foods but consume less of fruits and vegetables (Adegun 2003; Talabi 2005; Oyerinde and Owojaiye, 2008). Despite low activities of daily living civil servants do not engage in physical activities (Oyerinde and Owojaiye, 2008). Federal establishments are the highest paying government institutions in Nigeria. Furthermore, the monthly income of senior staff is high enough and have improved

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overtime making them to be able to afford 'fast foods' and other calorie dense meals which increase the risk of obesity and overweight (Nammi et al., 2004; Bakari et al., 2007). Epidemiological data on the prevalence and risk factors of cardiovascular diseases such as obesity, hyperglycaemia and dyslipidaemia in public officers in Nigeria is rare.

1.4 SIGNIFICANCE OF STUDY

There appears to be not much epidemiological data on obesity, hyperglycemia and dyslipidaemia among allied health workers in Nigeria. This study sets out to identify the prevalence and factors related to obesity, hyperglycaemia and dyslipidaemia in senior staff of UCH. Assessing the prevalence, risk factors, and predictors of these conditions in the study population is of paramount importance for preventing and controlling the sequelae.

1.5 RESEARCH QUESTIONS

This study aims at answering the following questions:

1. What is the prevalence of obesity, hyperglycaemia and dyslipidaemia among senior staff of the University College Hospital, Ibadan?
2. What are the prevalent factors of obesity, hyperglycaemia and dyslipidaemia among senior staff of the University College Hospital, Ibadan?
3. What is the pattern of clustering of the cardiovascular risk factors (obesity, hyperglycaemia, dyslipidaemia and hypertension) among senior staff of the University College Hospital, Ibadan?

1.6 OBJECTIVES OF THE STUDY-

Broad objectives

- ❖ To determine the prevalence of obesity, hyperglycaemia and dyslipidaemia and the associated factors among senior staff of University College Hospital (SSUCH), Ibadan.

The Specific Objectives are to:

- 1) Determine the prevalence of obesity, hyperglycaemia and dyslipidaemia among SSUCH.
- 2) Identify factors associated with obesity among senior staff of the University College Hospital, Ibadan
- 3) Identify factors associated with hyperglycaemia among senior staff of the University College Hospital, Ibadan
- 4) Identify factors associated with dyslipidaemia among senior staff of the University College Hospital, Ibadan
- 5) Determine the pattern of clustering of cardiovascular risk factors (obesity, hyperglycaemia, dyslipidaemia and hypertension) among senior staff of the University College Hospital, Ibadan

CHAPTER TWO

LITERATURE REVIEW

2.1 CARDIOVASCULAR DISEASES

Cardiovascular disease is any disorder, abnormality or failure to function well, relating to the heart and blood vessels or the circulation (Boon et al., 2002). Cardiovascular diseases (CVD) are prevalent conditions which impose significant negative impact on the healthcare system (Singh et al., 2008). Over the past decade, CVDs have become the single largest cause of death worldwide, representing nearly 30% of all deaths and about 50% of NCD deaths (WHO, 2011a). Over 82% of the mortality burden is caused by ischaemic or coronary heart disease (IHD), stroke (both hemorrhagic and ischaemic), hypertensive heart disease or congestive heart failure (CHF). In 2008, CVD caused an estimated 17 million deaths and led to 151 million DALYs (representing 10% of all DALYs in that year). About 80% of the CVD deaths are from low- and middle-income countries (WHO, 2011a). In fact, it has been reported that CVD in developing countries causes twice as many deaths as HIV, malaria and tuberculosis combined (Lopez et al., 2006). Cardiovascular diseases (CVDs) used to be the leading cause of morbidity and mortality in the western world but are now emerging public health challenges in developing countries (WHO 2002a). Parallel changes in lifestyle and diet in developing countries have led to an increase in life expectancy and a greatly increased burden of cardiovascular disease and other chronic diseases (Popkin et al., 2001; Yusuf et al., 2001; Wu et al., 2001). Biological and behavioural variables, such as diabetes, obesity, smoking, hypertension, dyslipidaemia and physical inactivity are robust risk factors for the development of CVD and mortality (Yusuf et al., 2004; Barr et al., 2007; Powers, 2007). Obesity has been shown to be an independent risk factor for coronary heart disease and the insulin resistance associated with obesity contributes to the development of other cardiovascular risk factors, including dyslipidaemia, hypertension, and type 2 diabetes (McFarlane et al., 2001; Peppa et al., 2003). The burden of cardiovascular disease that has increased in nearly all developing countries seems to be associated with urbanisation (Boutayeb and Boutayeb, 2005).

2.2 OBESITY: DEFINITION AND DESCRIPTION

Obesity is defined as a condition characterized by excessive accumulation and storage of fat in the body (Caterson and Gill, 2002) to the extent that health and well-being are adversely affected (WHO, 2000). In normal adult men 15 to 22% of the body is composed of fat while in healthy adult women 18 to 33% of the body is composed of fat. Technically, men with more than 22% fat and women with more than 33% body fat are regarded as obese (Billington, 2004).

2.3 ASSESSMENT OF OBESITY

A number of methods are used to determine overweight and obesity. The appropriate index of obesity is by measurement of percentage body fat (Caterson and Gill, 2003). There are many direct and indirect methods of assessing body composition and body fat (Genton et al., 2002). Techniques used for the accurate estimation of body fat include underwater weighing, dual energy X-ray absorptiometry (DEXA), total body water, total body electrical conductivity, total body potassium, computerized tomography and magnetic resonance imaging. However, the use of most of these methods is limited to research because of their complexity and cost (Taylor et al., 2000; Lindsay et al., 2001). Moreover, because increased body fat is usually accompanied by increased total body mass, anthropometric indices have replaced the classical approach (Aronne, 2002). The most frequently used tools in public health evaluations and clinical screening, therefore, are anthropometric-based measurements such as body mass index [BMI: $\text{wt} \ (\text{kg}) / \text{ht}^2 \ (\text{m}^2)$], waist circumference, waist to hip ratio and waist to height ratio (WHO, 1995).

2.4 CLASSIFICATION OF OBESITY

2.4.1 General Obesity: This is characterized by excess total body fat without any particular concentration of fat in a given area of the body. The WHO Obesity Task Force in 1998 developed a graded classification system for categorizing overweight and general obesity in adults according to body mass index and was adopted by the Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults (USA NIH, 1998). In the WHO system, in adults 20 years of age or older; BMI less than 18.5 kg/m^2 is regarded as underweight, between 18.5 and 24.9 kg/m^2 is classified as normal weight, overweight is defined as a $\text{BMI} \geq 25$ and obesity is defined as a $\text{BMI} \geq 30$. Obesity, based on the grade of the BMI, is further subdivided into class I ($\text{BMI}, 30.0$ to 34.9 kg/m^2), class II ($\text{BMI}, 35.0$ to 39.9 kg/m^2), and class III or extreme obesity ($\text{BMI} \geq 40 \text{ kg/m}^2$) (WHO, 1998).

2.4.2 Abdominal Obesity

The distribution of body fat is important. Upper body or abdominal obesity constitutes excess accumulation of fat and subcutaneous fat on the trunk and in the visceral compartment (WHO, 2005). Abdominal obesity is associated with more medical problems than lower body obesity. In clinical practise abdominal obesity is measured by waist circumference (Billington, 2004). Waist circumference of $>102 \text{ cm}$ in men and $> 88 \text{ cm}$ in women is regarded as abdominal obesity (WHO, 1997).

2.5 AETIOLOGY OF OBESITY

The aetiology of obesity is a chronic imbalance between energy intake and that expended (Bray, 2004). Increasing energy consumption, decreasing energy expenditure, or a combination of both, leads to a positive energy balance and a marked increase in body weight (Stein and Colditz, 2004). It is a complex, heterogeneous group of disorders which arises from the interactions of multiple genes and physiological, behavioural, social, cultural and environmental factors (Racette et al., 2003; Yang et al., 2007). Behavioural and environmental factors (sedentary lifestyles combined with excess energy intake) are primarily responsible for the dramatic increase in obesity during the past two decades (WHO, 1998).

2.6 EPIDEMIOLOGY OF OBESITY

The World Health Organization (WHO) in 2000 estimated that over one billion people were overweight globally, and that if current trends continue, the number will increase to 1.5 billion by 2015. Obesity in the past was regarded predominantly as a problem of Westernized societies (Likitmaskul et al., 2003; Adeyemo et al., 2003). In recent years it has become an increasing problem throughout most of the world including the developing world (Popkin, 2001; Campbell and Campbell, 2007). Indeed in many developing countries, overweight and obesity are so common that they are replacing more traditional problems such as undernutrition and infectious diseases, the most significant causes of ill health (WHO, 2000). WHO in year 2000 described obesity as one of the most blatantly visible, yet most neglected public-health problems that threaten to overwhelm both more and less developed countries. The global prevalence of obesity is 10% in men and 14% in women (WHO, 2008). The worldwide prevalence of obesity has been found to range from less than 5% in rural China, Japan and some developing countries to as high as 55% amongst the adult population of urban Samoa (WHO, 2005c). In the United States and elsewhere, obesity has increased dramatically since 1980 (Ogden et al., 2007). Between 1980 and 2004 the prevalence of obesity increased from 15% to 33% among US adults (Flegal et al., 2002; Ogden et al., 2006). Also, obesity is relatively common in Europe. A marked trend toward increasing levels of adult overweight and obesity can be found throughout Europe (Turconi and Cena, 2012). Data from WHO, 2005 suggest that obesity prevalence in European countries in 2002 ranges from 2.5 % - 26.2% for men and up to 33% for women (WHO, 2005c). The problem of obesity is growing in many developing countries (WHO, 2015). Rates of obesity have tripled in the last 20 years in the developing world with 10% of the world's children currently overweight or obese. The Middle East, Pacific Islands, Southeast Asia, and China are facing the greatest challenges (Hossain et al., 2007). It is currently estimated that as much as 20-50% of urban populations in Africa are classified as either overweight or obese (Kamadjeu et al., 2006; Sodjinou et al., 2008). These dramatic increases in the prevalence of obesity raise serious concerns about a future characterized by increasing rates of diabetes and other obesity-associated disorders around the globe (Boschiero, 2008). It is logical to link the increasing obesity rates in developing countries with a progressive introduction of factors associated with obesity in developed societies such as sedentary lifestyle, high fat and fast foods (Caballero, 2001).

2.7 OBESITY: MORBIDITY AND MORTALITY.

2.7.1 Obesity and Morbidity

Obesity is becoming a major public health issue due to its association with chronic diseases such as prediabetes, diabetes mellitus, hypertension, dyslipidaemia, sleep apnea, osteoarticular disease, cancer, cardiovascular and cerebrovascular diseases (Poirier et al., 2006). Obese people have a higher incidence of prediabetes, diabetes, hypertension and dyslipidaemia compared to non-obese people (Mokdad et al., 2003; Qian et al., 2010). The risks of diabetes, hypertension, and dyslipidaemia increase from a BMI of about 21.0 kg/m^2 , thereby greatly increasing the health and societal economic burden (James et al., 2004). Moreover, obesity accounts for 20–33% of the risk for breast, oesophageal, endothelial and kidney cancer (IARC. 2002).

2.7.2 Obesity and Mortality

Even though the optimum body weight for health is a subject of debate (Mitchell, 2003) obesity has been shown repeatedly to be associated with a modestly increased risk of increased mortality (Engeland et al., 2003). A body mass index of between 30.0 and 34.9 kg/m^2 is associated with a two-fold increase in mortality (Freedman et al.. 2006). In the Framingham Heart Study life expectancy was shown to decrease by 5.8 and 9.1% in obese men and women respectively (Peeters et al., 2003). In another study based on US national data, it was demonstrated that obesity shortened life expectancy, especially among younger adults (Fontaine et al., 2003). The number of deaths per year attributable to obesity is roughly 30.000 in the UK (UK National Audit Office. 2001) .The main cause of excess mortality in obesity, relative to normal weight, usually has been found to be cardiovascular disease (Dorn et al., 1997).

2.8 RISK FACTORS OF OBESITY

The factors associated with obesity are increasing age, female gender, family history of obesity, urban residence, parity of a woman, being literate, having a high socio-economic status, being stressed, alcohol consumption, tobacco use, contraceptive use, exposure to television, high intake of meat, taking lunch outside home, taking snacks between meals, eating tea, physical inactivity and sedentary life style (Jafar et al.. 2006; Mahmood et al., 2010; Agbeko et al., 2013).

Age has a positive significant association with overweight and obesity (Agbeko et al., 2013). The most significant predictors of overweight and obesity by fat percentage are age,

and the number of assets in the household (Mbochi et al., 2013). Overweight and obesity appear to be positively associated with age ranging from 14% for women aged 15-24 to 43% for those aged 35-44.

Moreover, obesity is associated with gender (Aekplakorn and Mo-suwan 2009). Women are more likely (14%) to be obese than men (10%) (Aekplakorn and Mo-suwan 2009). Family history of obesity is present in 44% of obese individuals (Mahmood et al., 2010)

Educational and wealth status have a positive significant association with overweight and obesity (Amoah, 2003). People with high level of education and the employed working class are more likely to be overweight or obese (Amoah, 2003). Women with higher education were about two times more likely to be overweight or obese compared with those without education (Agbeko et al., 2013). Women in the richest wealth category were more than five times to develop obesity than those in the poorest category (Agbeko et al., 2013).

Moreover, overweight or obesity increases with parity (Agbeko et al., 2013). A woman's risk of overweight or obesity increases by 7% per child (Martorell et al. 2000). Also, during pregnancy, women gain weight but after giving birth some find it hard to lose the weight gained resulting in obesity (Abubakari et al., 2008).

Alcohol consumption, tobacco use, contraceptive use and exposure to television are positively associated with overweight and obesity (Mahmood et al., 2010; Agbeko et al., 2013). Women who consume alcohol are about 1.4 times more likely to be overweight or obese than those who did not consume alcohol (Agbeko et al., 2013). There is higher probabilities of overweight or obesity among women who used contraceptive methods, irrespective of whether such methods were modern or traditional (Agbeko et al., 2013). The odds of women who were exposed to television being overweight or obese are about 1.3 times more than those who were not exposed (Agbeko et al., 2013). Nearly 18% of obese consume tobacco (Mahmood et al., 2010).

Stress is significantly and strongly predictive of physician overweight, as are particular eating behaviours, including eating food provided at the medical office (Mahmood et al., 2010). There is evidence that people who are stressed are more likely to be overweight than those who are not (Laitinen et al. 2002).

Similarly, physical activity is also found to be a protective factor against obesity (Aronne, 2003; Dugan, 2008). There is a significant increase in mean BMI, %BF and WC as physical activity levels decrease with the highest mean BMI, %BF and WC found at the lowest level of physical activity levels (Mbochi et al., 2013).

Most of the variance in body mass index is explained by age, total physical activity, percentage dietary reference intake of fat consumed, parity and socio-economic group, in that order, together accounting for 18.0% of the variance in BMI (Mbochi et al., 2013). With regard to WC, age, parity and the number of rooms in the houses where the women resided, were the most significant predictors of abdominal fat deposition (Mbochi et al., 2013).

2.9 IMPAIRED FASTING GLUCOSE (IFG)

2.9.1 DEFINITION AND DESCRIPTION

Prediabetes is typically defined as blood glucose concentrations higher than normal, but lower than diabetes thresholds (ADA, 2007; Tabák, 2012). Prediabetes is characterized by impaired glucose metabolism which includes the conditions of impaired fasting glucose {IFG} and impaired glucose tolerance {IGT} (WHO, 2006; ADA, 2007). IFG and IGT represent intermediate states of abnormal dysglycaemia (ADA, 2008). IFG is identified by a fasting plasma glucose (FPG) measurement of between 100 and 125 mg/dl. Prediabetes is associated with the simultaneous presence of insulin resistance and β -cell dysfunction: abnormalities that start before glucose changes are detectable (Tabak et al., 2012).

2.9.2 EPIDEMIOLOGY

The prevalence of prediabetes ranges from 20 to 40% (Benjamin et al. 2003; The Expert Committee on the diagnosis and Classification of Diabetes Mellitus, 2003; Qiao et al., 2003; Johnsen et al., 2004). Prevalence of prediabetes is increasing worldwide and experts have projected that more than 470 million people will have prediabetes by 2030 (Wild et al., 2004). Prediabetes is very common in the USA (Cowie et al., 2009) and affects nearly 79 million Americans (CDC, 2011). The population-based US National Health and Nutrition Examination Survey (NHANES) suggests that 35% of US adults older than 20 years and 50% of those older than 65 years had prediabetes between 2005 and 2008 (CDC, 2011). The prevalence of IFG and IGT varies widely, with recent data from the U.S. indicating the prevalence of IFG to be approximately 26% and somewhat older data showing a 15% prevalence of IGT (CDC, 2005).

The pre-diabetes phase can last up to seven years in those who develop diabetes (Wild et al., 2004). In a meta-analysis of prospective studies published between 1979 and 2004, annualised incidence rate of progression to diabetes in patients with isolated IFG was 6.9% and for isolated IGT was 4.6% and these values are lower than in those with both IFG and

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IGT which had prevalence rates of between 15 and 19% (Gerstein et al., 2007). The annualized relative risk of people with isolated IFG progressing to diabetes is 4.7-fold compared with people with normal glucose tolerance (Santaguida et al., 2005). The annualized relative risk of a person with IGT progressing to diabetes is increased 6-fold when compared with people with normal glucose tolerance (Santaguida et al., 2005) while individuals with both IFG and IGT experience a 12-fold relative risk (Santaguida et al., 2005).

2.9.3 PREDIABETES AND MORBIDITY

Prediabetes significantly increases the risk of type 2 diabetes (NIDDK, 2007), cancers (Jee et al., 2005), fatal and non-fatal cardiovascular disease (Levitin et al., 2004.). Observational evidence also shows associations between prediabetes and early forms of nephropathy, chronic kidney disease, small fibre neuropathy and retinopathy (Tabak et al., 2012). The development of chronic complications of diabetes, either micro or macrovascular, can begin earlier in the pre-diabetes phase as demonstrated by UKPDS and Diabetes Prevention Program (DPP), with increasing prevalence from IFG to IGT (Diabetes Prevention Program Research Group, 2002). The highest prevalence is observed in patients with both conditions (The DECODE Study Group, 2003).

2.9.4 PREDIABETES AND MORTALITY

Prediabetes is increasingly recognized as being associated with an increased risk of premature mortality though it was initially defined as a category of glycaemia associated with an increased risk of developing diabetes (WHO, 2006). People with impaired glucose tolerance have 40% increased mortality, regardless whether they progress to diabetes or not (Qiao et al., 2003). Because impaired glucose tolerance is a common condition affecting 15–40% of adults (DECODE, 2003; DECODE, 2003) and is an independent predictor of mortality, the impact of hyperglycemia on mortality is larger than that associated with diabetes alone (Roglic et al., 2005). The risk of premature mortality is however less than that of developing diabetes (WHO, 2006).

Prediabetes is associated with fatal cardiovascular disease (Levitin et al., 2004.) and all-cause mortality (Kakigami et al., 2004; Sorkin et al., 2005). Cardiovascular mortality are increased in individuals in the prediabetic state who have either impaired glucose tolerance (IGT) or impaired fasting glucose (IFG); however, those with IGT appear to be at greater

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risk (Nathan et al.. 2007; Barr et al., 2009). The relative risk of a fatal cardiovascular outcome was 1.7-fold higher for people with IGT and 1.2-fold higher for those with IFG than in normal individuals (Santaguida et al., 2005). Individual-level evidence from prospective studies suggests that fasting hyperglycaemia and postload glucose are all robust predictors of vascular mortality (Barr et al., 2001; Sarwar et al., 2010) and after multivariable adjusted analyses, these associations are independent of vascular risk factors such as obesity, blood pressure, triglyceride, and lipoproteins (Brunner et al., 2006 ; Seshasai et al., 2011).

IFG and IGT differ significantly in their age and sex distribution however both disorders are more common in people older than 40 years (Cowie et al., 2009). Prevalence of isolated IFG increased in the DECODE study from 5.2% in 30- to 39-year-old men up to 10.1% in 50- to 59-year-old men and then decreased to 3.2% in 80- to 89-year-old men, whereas in women, the prevalence increased from 2.6% in 30 to 39 year olds to 5.9% in 70- to 79-year-old individuals (DECODE Study Group, 2003).

In the Diabetes Epidemiology Collaborative analysis of Diagnostic criteria in Europe (DECODE) study, 2003, the prevalence of isolated IGT increased from 2.9% in 30- to 39-year-old men to 15.1% in 70- to 79-year-old men and from 4.5% 30- to 39-year-old women to 16.9% in 70- to 79-year old women. A similar pattern (with some exceptions) was observed in Asian populations, with the prevalence of IGT increasing with age up to 70 to 89 years (Qiao et al., 2003).

IFG is more prevalent in men than in women, although the reasons for this difference are poorly understood (Cowie et al., 2009). In Asian populations, the prevalence of isolated IFG generally increases with age, although again with few exceptions (Qiao et al., 2003). IGT, on the other hand is typically more common in women than in men (DECODE Study Group, 2003). Prevalences of IFG and IGT vary between ethnic groups (Cowie et al., 2009).

2.10 DIABETES MELLITUS (DM)

2.10.1 DEFINITION AND DESCRIPTION

Diabetes mellitus is a group of chronic metabolic conditions, all of which are characterized by elevated blood glucose levels resulting from the body's inability to produce insulin or resistance to insulin action, or both (ADA, 2006). Because DM is often asymptomatic in the early stages, it is frequently undiagnosed for many years. Insulin resistance and hyperglycemia develop gradually, while pathological and functional changes in various target tissues may not be severe enough for patients to notice any of the classical symptoms of the disease (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 2003). It represents one of the most challenging public health problems of the 21st century and is reaching epidemic levels globally (Mao et al., 2010). Diabetes mellitus is one of the most common chronic diseases in nearly all countries, and continues to increase in number and significance, as changing lifestyles lead to reduced physical activity and increased obesity (Chen et al., 2009; Shaw et al., 2010). Diabetes mellitus remains an important component of non-communicable diseases because of its prevalence and the related complications worldwide (Li et al., 2010).

It is a common condition that leads to considerable morbidity and premature mortality and long-term complications include foot and leg ulcers, visual and renal complications, heart disease, stroke and peripheral vascular disease (Newnham et al., 2002). The results of a study using the UK General Practice Research Database showed that patients with type 2 diabetes have a mortality rate almost twice as high as those without diabetes (Mukherjee et al., 2006). Diabetes itself is not a high-mortality condition but it is a major risk factor for other causes of death and has a high attributable burden of disability (Wilson et al., 2011). The late diagnosis of diabetes and lack of regular monitoring of patients in sub-Saharan Africa coupled with poor facilities in managing care, leads to early presentations of diabetic complications (Ogundipe et al., 2009; Tsof, 2008). It exerts a heavy economic burden on society and the economy with a considerable drain on healthcare and financial resources although in Africa, a prevention, population-based cohort study found that diabetic individuals with complications were twice as likely not to be in the labour force compared to non-diabetic individuals (Ong et al., 2009). Although the related costs differ there is variety of factors, which contribute towards economic burden. The main direct costs of the total cost of diabetes management are:

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2.10.2 AETIOLOGY OF DIABETES

The two main types of diabetes mellitus are type 1 (formerly known as insulin-dependent diabetes), and type 2 (formerly known as non-insulin-dependent diabetes). Type 1 diabetes is caused by the autoimmune destruction of the β -cells of the pancreatic islets whereas type 2 diabetes results from both impaired insulin secretion and resistance to the action of insulin. T1D occurs mainly in children and adolescents (Liese et al., 2006). Type 2 diabetes accounts for 90-95% of all diabetes cases and usually affects overweight individuals, and most cases are diagnosed in those over 40 years old (Jonsson, 2002). However, the demographics of this disease are changing, and it is now becoming increasingly common in children and young adults (American Diabetes Association, 2000; Simmons et al., 2011).

2.10.3 RISK FACTORS FOR DIABETES MELLITUS

A number of factors that increase the risk for diabetes mellitus have been identified (Deshpande et al., 2008). Risk factors for type 1 diabetes include family history, race and certain viral infections during childhood (Deshpande et al., 2008). Risk factors for type 2 diabetes are more diverse; some are modifiable, others are not (Deshpande et al., 2008).

Non-modifiable risk factors for type 2 diabetes include age, race or ethnicity, family history, history of gestational diabetes, and low birth weight (Deshpande et al., 2008). Diabetes incidence and prevalence increases with age (CDC, 2005). African Americans are more likely to develop diabetes than whites (Egede and Dagogo-Jack, 2005). In addition, for Native Americans, the rates of diagnosed diabetes range from 5% to 50% in different tribes and population groups. Little difference exists by sex. Genetic factors also play a role, but non-genetic or lifestyle risk factors (such as diet and physical activity) appear to be the primary culprits (Schulz et al., 2006).

Modifiable or lifestyle risk factors include increased overweight, obesity, physical inactivity, poor nutrition, hypertension, pre-diabetic states (impaired fasting glucose and impaired glucose tolerance), smoking, and alcohol use, delivery of macrosomic babies ($HDL \leq 35\text{mg/dl}$ and/ or triglyceride greater than 250mg/dl) and polycystic ovarian syndrome among others (Braunwald et al., 2001; Zimmet, 2001; Egede and Dagogo-Jack, 2005). Increased BMI is consistently shown to be one of the strongest risk factors for development of diabetes (Hu et al., 2001). In addition, distribution of body fat (Rewers and Hamman, 1995) and specifically an increased waist-to-hip ratio, lower levels

of physical activity (Deshpande et al., 2008), hypertension (Gress et al., 2000), high total caloric intake (Deshpande et al., 2008) all increase a person's risk for diabetes .Consistent findings from various studies show that moderate alcohol use may reduce the risk for developing diabetes (Narayan et al., 2006) but smoking has been shown to be an independent risk factor for diabetes (Will et al., 2001). Impaired fasting glucose and impaired glucose tolerance are strong predictors of type 2 diabetes (2).Individuals with impaired fasting glucose (IFG) have a 20–30% chance of developing diabetes over the next 5–10 years (Meigs, 2003; Tirosh et al., 2005). The risk is even greater for individuals who have combined IFG and impaired glucose tolerance (Sinha et.al. 2002).

Psychosocial factors such as depression, increased stress, lower social support, and poor mental health status also are associated with an increased risk for the development of diabetes (Arroyo et al., 2004; Strodl and Kenardy, 2006). Recently, adverse housing conditions were found to be independently associated with the development of self-reported diabetes (Schoolman et al., 2007).

2.10.4 EPIDEMIOLOGY

Diabetes is an important chronic disease which incidence is rapidly increasing globally and therefore considered as an epidemic (Wild et al., 2004). According to the US Centers for Disease Control and Prevention, rates of type 2 diabetes mellitus (T2DM) have tripled in the past 30 years. The World Health Organization (WHO) estimated there were 30 million people who had diabetes worldwide in 1985. This number increased to 135 million by 1995 and reached 217 million in 2005. By the year 2030 WHO predicts this number will increase to at least 366 million (Wild et al., 2004). The prevalence of diabetes mellitus for all age-groups world-wide was 2.8% in 2000 and estimated to be 4.4% in 2030 (Wild et al., 2004). This growth in diabetes prevalence, driven principally by an increased prevalence of type 2 diabetes is occurring in both developing and developed countries (Wild et al., 2004). Factors responsible for the rise in diabetes include unhealthy diet, population growth, aging, urbanization and increase in prevalence of overweight/obesity and physical inactivity (WHO, 2004; Wild et al., 2004).

The prevalence of diabetes in the UK is 6.1% (Kanavos et.al 2012). Africa, 3.1% (IDF Diabetes Atlas, 2007), Nigeria(3.8%).(IDF Diabetes Atlas, 2007) while the crude prevalence of total diabetes in the US is 9.0% (CDC, 2011).Globally, diabetes prevalence is increasing and is responsible for 5% of all deaths annually (World Health Organisation 2011). Given

current projections, without urgent action, mortality due to diabetes is expected to increase by 50% in the next 10 years (World Health Organisation 2011). Developing countries, including Africa, will bear the brunt of the rapid diabetes increases (Wild et al., 2004).

2.10.5 DIABETES AND LIFESTYLE

Worldwide there are over 250 million diabetics, and projections for 2025 are disquieting: more than 330 million people will suffer from diabetes if prevention measures are not taken (Jacobson et al., 2011).

Furthermore, prospective studies have demonstrated that lifestyle modification in the form of diet and regular moderate exercise sharply decreases the likelihood of developing type 2 diabetes in high-risk individuals with impaired glucose tolerance or impaired fasting glucose (Knowler et al., 2002).

An unhealthy lifestyle featuring a lack of physical activity and excessive eating initiates and propagates the majority of type 2 diabetes (Fowler 2010). Regular physical activity (Manson et al., 1991; Lynch et al., 1996), maintenance of an optimal body weight (Schienkiewitz, 2006; Biggs et al., 2010) a healthful diet (Salmero'n et al., 2001; Hodge et al., 2004), avoiding smoking (Nakanishi et. al., 2000) and moderate alcohol use (Ajani et al., 2000; Wei et al., 2000) have each been associated with a lower risk for diabetes. An overall healthy lifestyle that incorporates more than one of these factors may be more effective in lowering risk for diabetes than any single factor (Hu et al., 2001; Mozaffarian et al., 2009). Individuals with low-risk lifestyle profile that included not smoking, engaging in regular physical activity, consuming a healthful diet, using alcohol in moderation, and having an optimal body weight have a dramatically lower risk for incident diabetes than those without such a profile (Reis et al., 2011). Each additional lifestyle factor in the low-risk category is associated with a 31% lower risk for diabetes among men and a 39% lower risk among women (Reis et al., 2011). The combined influence of these factors had a slightly stronger association with a lower risk for diabetes in a study among women (Reis et al., 2011).

Reis et al. 2011 also observed a 19% lower risk for diabetes among men and a 37% lower risk among women with moderate alcohol consumption, compared with those who were not moderate drinkers. Insulin resistance is an important factor the development of diabetes, and light to moderate alcohol consumption has been associated with enhanced insulin sensitivity in several observational studies (Kiechl et al., 1996; Lazarus et al., 1997). In controlled trial in non-diabetic postmenopausal women consumption of 30 g/day of alcohol for 8 weeks resulted in decrease in fasting insulin levels, as well as increases in triacylglyceride

concentration and insulin sensitivity (Davies et al., 2002). Moderate alcohol consumption also has anti-inflammatory effects which could further decrease the risk for diabetes (Imhof et al., 2001; Sierksma, 2002).

Several prospective cohort studies have shown an association between active smoking and risk for diabetes (Willi et al., 2007). In recent study diabetes risk was higher in persons who had recently quit smoking than in never-smokers, but gradually decreased to 0 after 12 years (Yeh et al., 2010). Smoking negatively affects insulin sensitivity and pancreatic β -cell functioning (Somm, et al., 2008), has proinflammatory effects (Asthana, 2010) and increases central obesity (Barrett-Connor, 1989) all of which have been implicated in the development of diabetes (Reis et.al., 2011).

Lifestyle interventions are not only beneficial before the development of diabetes (Fowler, 2010). Several studies have clearly demonstrated the benefits of control over diet, exercise, and weight loss in individuals already diagnosed with diabetes (Fowler, 2010). Dietary restriction to 1,100 kcal/day has been shown to decrease fasting blood glucose levels in obese patients with diabetes and even those without diabetes in as few as 4 days. This improvement is likely the result of decreased hepatic glucose output. After 28 days of calorie restriction, there is further decline in fasting glucose levels of obese diabetic subjects, and insulin sensitivity is significantly improved. It is also noteworthy that improvement in insulin sensitivity correlates well with decrease in fasting glucose (Markovic et al., 1998). These changes occurred with an average weight loss of only 6 kg (Maggio and Pi-Sunyer, 1997; Markovic et.al., 1998).

Physical exercise is a key component of lifestyle modification that can help individuals prevent or control type 2 diabetes (Fowler, 2010). Although diet is probably more important in the initial phases of weight loss, incorporating exercise as part of a weight-loss regimen helps maintain weight loss and prevent regaining of weight (Klein et al., 2004). Mild to moderate activity levels have been associated with a lower risk of developing diabetes or pre-diabetes. Men with low degrees of cardio-respiratory fitness may possess up to a 1.9-fold increased risk of developing impaired fasting glucose compared to men with high degrees of fitness (Wei et al.. 1999). The amount of exercise that produces a beneficial effect on health is not large: as little as 30 minutes of moderate physical activity daily may offer protection from diabetes (Klein et al., 2004; Bassuk and Manson, 2005). Walking was associated with reduced risk of mortality and walking pace was inversely associated with CVD, fatal CVD, and total mortality, independent of walking hours (Tarnescu et al., 2003). In contrast, low levels of physical fitness are consistently associated with higher

cardiovascular and all-cause mortality rates. Moreover, midlife increases in physical activity, through change in occupation or recreational activities, are associated with a decrease in mortality rates.

As with the lowering of HbA_{1c} levels, there is a gradient of benefit with higher levels of exercise and activity. Greater levels of physical activity are associated with lower risks of developing diabetes in women compared to lesser levels of activity (Hu et al., 1999). These studies indicate that exercise should be a mainstay of primary prevention of diabetes (Fowler, 2010). As further evidence regarding the benefit of exercise, in men with diabetes, the degree of physical fitness correlated with overall mortality, and this association was independent of BMI (Church et al., 2004; Klcin et al., 2004). A recent review of 10 prospective cohort studies investigating moderate-intensity physical activity and diabetes provides evidence that people who achieve recommended levels of even moderate activity are about 30% less likely to develop diabetes than their inactive counterparts (Jeon et al., 2007). Tanasescu et al., in 2003 add to the evidence that physical activity reduces cardiovascular mortality in those with existing diabetes. Despite the scientific evidence and the organizational efforts, these messages have not reached the public, because physical activity is vastly underutilized in the management of diabetes, and the majority of individuals remain sedentary or do too little exercise to achieve health benefits (Fletcher et al., 2001).

2.10.6 DIABETES AND MORBIDITY

Diabetes is a risk factor for other diseases, often termed "complications" that impose major health problems (Begg and Schulzer, 2001). Diabetics have higher rates of morbid events, hospitalization and disability than non-diabetic persons (Naslafkikh and Sestier, 2003). Diabetes accounts for the majority of patients with adult-onset retinopathy, blindness, end-stage renal failure, and non-traumatic limb amputations. Furthermore, diabetes is the leading underlying cause of coronary heart disease, stroke and peripheral vascular disease (American Diabetes Association, 2000; Center for Disease Control, 2000). Diabetes can affect many different organ systems in the body and, over time, can lead to serious complications. Complications from diabetes can be classified as microvascular or macrovascular. Microvascular complications include nervous system damage (neuropathy), renal system damage (nephropathy) and eye damage (retinopathy) (ADA, 2006). Macrovascular complications include cardiovascular disease, stroke, and peripheral vascular

disease. Peripheral vascular disease may lead to bruises or injuries that do not heal, gangrene, and, ultimately, amputation (Deshpande et.al., 2008). Also patients with diabetes aggregate other co-morbidities such as obesity, hypertension, and dyslipidaemia which also contribute to increase the risk for CVD (Ghatrif, 2011). It has been stated that patients with T2D without a previous history of myocardial infarction have the same risk of coronary artery disease (CADs) as non-diabetic subjects with a history of myocardial infarction (Haffner, 1998); this has led the National Cholesterol Education Program to consider diabetes as a coronary heart disease risk equivalent (NCEP, 2002).

2.10.7 DIABETES AND MORTALITY

Hyperglycaemia is a serious illness with multiple complications and premature mortality (Diabetes Atlas, 2003). Diabetes ranks among one of the leading causes of death worldwide. Globally, it was the fifth leading cause of death in 2000 after communicable diseases, cardiovascular disease, cancer and injuries (Roglic et al., 2005). The World Health Organisation estimated that mortality from diabetes in the world for the year 2002 was 1.7% of total world mortality (WHO, 2003). Furthermore, global excess mortality attributable to diabetes in the year 2000 was estimated to be 5.2% of world all-cause mortality (Roglic et al., 2005). The number of excess deaths attributable to diabetes is similar in magnitude to numbers reported for HIV/AIDS in the year 2000 (WHO, 2003). In most developing countries, almost one in ten deaths in economically productive individuals aged 35–64 years can be attributed to diabetes (Roglic et al., 2005). Even in the poorest countries, at least one in twenty adult (35–64 years of age) deaths is diabetes related, and in most countries, the proportion is substantially higher (Roglic et al., 2005).

Type 2 diabetes is associated with an unequivocal increase in mortality risk regardless of the age at diagnosis when compared to the general population (Nwaneri et al., 2013). People with diabetes have an increased mortality and a reduced life expectancy compared with those without diabetes (Morgan et al., 2000). An India study reported that the mean age at death of diabetics is significantly lower than the life expectancy at birth (Zargar et al., 2009). Zargar et al., 2009 also documented that diabetes-related death was attributed to a single cause in 52.9%, two causes in 36.3%, and three or more causes in 8.0% of cases (Zargar et al., 2009). This association with an increase in mortality rate is independent of traditional cardiovascular risk factors (Saydah et al., 2001; The DECODE Study Group, 2001; Nakagami, 2004).

Studies have reported that individuals with diabetes have an excess risk of mortality compared with those without diabetes especially at the peak of their working career (Morgan et al., 2000; Mohan et al., 2006). Recent studies have suggested that the rate of death in diabetics is twice that seen in the population without diabetes (Guzder et al., 2007; Nwanaeri et al., 2010). Nwanaeri et al., 2010 reported a relative risk of 1.85 (95% CI 1.79–1.92) for all-cause mortality and 1.76 (95%CI 1.66–1.88) for CVD related mortality and 2.26 (95% CI 1.7–3.02) for stroke.

Independent predictors of diabetes-related death include age, micro and/or gross albuminuria, gender, smoking, hypertension, previous cardiovascular disease, peripheral vascular disease, lipid abnormalities , poor glycaemic control and duration of diabetes (Biderman et al., 2000; Ostgren et al., 2002; Nwaneri et al., 2003).

2.11 DYSLIPIDAEMIA

2.11.1 Definition

Dyslipidaemia is defined as an abnormal plasma lipid status (NCEP, 2001). It is indicated by elevated levels of total cholesterol, LDL cholesterol, triglycerides and low levels of HDL cholesterol (NCEP, 2001). Dyslipidaemia is defined as total cholesterol >200 mg/dl, LDL-C >130, HDL-C <40 mg/dl for males, <50 mg/dl for females and TG > 150 mg/dl. (NCEP, 2001). These abnormalities can be found alone or in combination (Ballantyne et.al., 2009).

2.11.2 Epidemiology of Dyslipidaemia

Dyslipidaemia is the second most prevalent cardiovascular risk factor (Tekes-Manova et al. 2006). WHO estimates in 2002 showed that dyslipidaemia accounted for 18% of ischemic heart disease, 56% of stroke (cerebrovascular disease) and more than 4 million deaths per year globally. Dyslipidaemia is common even among persons without CVD (Goff et.al.. 2006). The total cost associated with cardiovascular diseases and stroke in the United States—many of which are related to dyslipidaemia—is estimated to exceed \$400 billion in 2006 (Thom et. al. 2006). Dyslipidaemia develops through a number of mechanisms including heredity, advancing age, a diet high in cholesterol and saturated fatty acids, insulin resistance, and obesity (White, 2008).It may also be secondary to other diseases such as diabetes, hypothyroidism, nephritic syndrome and chronic renal failure (White, 2008).

Dyslipidaemia is common in persons without CVD however the quality of care for dyslipidaemia is suboptimal (Goff et.al., 2006). Data from the U.S. National Health and Nutrition Examination Survey conducted from 1999 to 2000 found that 25 percent of adults had total cholesterol greater than 239.4 mg per dl or were taking a lipid-lowering medication (Ford et.al., 2003).

Dyslipidaemia was previously thought to be rare in Black Africa, including Nigeria (Oguejiofor et.al., 2012). Early reports suggested that blacks have lower prevalence of dyslipidaemia possibly due to genetic, nutritional, and environmental factors (Kesteloot et.al., 1989). Dyslipidaemia is now highly prevalent in Nigeria and Black Africa at a rate currently comparable with Caucasian values (Oguejiofor et.al., 2012).

2.11.3 PREDICTORS OF DYSLIPIDAEMIA

Dyslipidaemia is significantly associated with co-morbidity from obesity, diabetes and hypertension (Al-Kaabba et.al., 2012). Obesity (Zhao et.al., 2007), hypertension (Akintunde et al., 2010) and diabetes mellitus (Ogbera et al., 2009) are associated with all forms of dyslipidaemia. Several other factors have been shown to be related to lipid levels in adults, including dietary habits and the intake of certain foods, sedentary lifestyle, education and occupation, gender, age, physical activity, routine physical exercise and smoking (Mahley et.al., 2005; Song et.al., 2012).

Migration to urban centers and adoption of a sedentary lifestyle is likely related to rising lipid levels and prevalence of dyslipidaemia (Bener et.al., 2009). Previous studies have also shown that dyslipidaemia is associated with education and occupation (Ogbeide et.al., 2004; Al-Nozha et.al., 2008). A higher education level was strongly associated with hypercholesterolemia and high LDL-C levels, even after adjusting for age and body mass index (Pradeepa 2003).

Dyslipidaemia is more common in men than in women and controlled less often in men than in women (Goff et.al., 2006; Al-Kaabba et.al., 2012). Men are approximately 30% more likely than women to have dyslipidaemia; adjustment for risk factors and socioeconomic and healthcare access variables had no substantive effect on these findings (Goff et.al., 2006).

Prevalence of dyslipidaemia increases with advancing age (Al-Kaabba et.al., 2012). There is a greater prevalence of dyslipidaemia in the older age groups, a finding that is significant after adjustment for gender, ethnicity, clinic site, risk factors, and socioeconomic and healthcare access variables (Goff et.al., 2006).

Dyslipidaemia prevalence differed little across ethnic groups, with the exception of a lower prevalence in Chinese subjects (Goff et.al., 2006). Among persons with dyslipidaemia, control of dyslipidaemia was achieved less commonly in blacks and Hispanics than in non-Hispanic whites (Goff et.al., 2006).

Dyslipidaemia is affected by lifestyle (Al-Kaabba et.al., 2012). Moderate physical activity at least 30-60 minutes 5 days a week or longer will help to raise HDL-C, lower TG levels, total and LDL-C. A meta-analysis indicated that regular aerobic exercise modestly increases HDL-C level resulting in a 2.53-mg/dL elevation of net HDL-C change (Kodama et. al., 2007). Every 1-mg/dL (0.026-mmol/L) increment in HDL-C level was reported to be associated with a 2% and 3% decreased risk of CVD in men and women, respectively (Maron, 2000). The increases in HDL-C level by exercise result in a CVD risk that is reduced by approximately 5.1% in men and 7.6% in women (Kodama et. al., 2007). Intervention studies have demonstrated improvements in lipid profile with increases in exercise (Erem et. al., 2008; Kang et.al., 2009), as physical activity mainly results in a reduction in triglyceride levels and an increase in HDL-C (Polychronopoulos et.al., 2005).

Smoking also has been reported to have atherogenic effect through abnormal lipid metabolism (Sharma et.al., 2005). Also, smokers have significantly higher levels of TC, TG and LDL-C but lower HDL- C concentration than non-smokers (Mouhamed et.al., 2013). These differences remain significant after adjustment for BMI, gender, and age (Mouhamed et.al., 2013).

2.11.3.1 OBESITY

Atherogenic dyslipidaemia is strongly associated with obesity, is common and is an important risk factor for cardiovascular outcomes (Bamba and Daniel, 2007). The primary dyslipidaemia related to obesity is characterized by increased triglycerides, decreased HDL levels, and an abnormal small LDL composition (Taskinen, 2005) and no doubt plays a major role in the development of atherosclerosis and cardiovascular disease (Taskinen, 2005).

2.11.3.1.1 Elevated Triglycerides In Obesity.

Obesity is always associated with increases in plasma triglycerides (Howard, 2003). Analysis of the Second National Health and Nutrition Educational Survey (NHANES II) data, which represent a random sample of US adults within a wide range of age and socioeconomic status, shows an increase in triglyceride levels with increasing obesity in white men and women of all ages (Howard, 2003).

2.11.3.1.2 Increased Small, Dense Low-density Lipoprotein Particles in Obesity

In contrast to the consistent changes in triglycerides and HDL with obesity, a less consistent effect on low-density lipoprotein (LDL) concentrations has been observed (Howard, 2003). Despite the lack of consistent changes in LDL concentration with obesity, a change in LDL composition occurs in obese individuals whose LDL particles are smaller and more dense, consistent with their higher triglyceride and lower HDL concentrations (Gray *et. al.*, 2000).

2.11.3.1.3 Decreased High-density Lipoprotein Cholesterol in Obesity

Low levels of HDL-C have been shown to be well correlated with obesity (Ashen and Blumenthal, 2005). A longitudinal study confirmed this association, with an 8-year increase in BMI of 1 unit associated with a decrease in HDL cholesterol of approximately 3 mg/dl. Weight loss studies of longer than 1 year in duration show consistent increases in HDL (Wood *et. al.*, 1991; Karvetti and Hakala 1992).

2.11.3.2 DIABETES MELLITUS

Closely related to dyslipidaemia is DM (Smith, 2007). The term “atherogenic dyslipidaemia” refers to the triad of elevated triglycerides, low levels of high-density lipoprotein (HDL) cholesterol, and a preponderance of small, dense low-density lipoprotein (LDL) particles (Reasner, 2006). Atherogenic dyslipidaemic profile is typically associated with type 2 diabetes mellitus (Mooradian, 2009). Dyslipidaemia is a major risk factor for macrovascular complications in patients with type 2 diabetes mellitus (Mooradian, 2003). Approximately 80% of deaths in diabetic patients are attributable to cardiovascular disease (CVD), which in turn is highly correlated with dyslipidaemia (O’Keefe *et.al.*, 1999). Most common form of dyslipidaemia found in T2DM patients is low HDL (59.3%). Hypertriglyceridemia is found in approximately 51% of diabetics, raised LDL is found in 51% patients (Karim *et. al.*, 2013) while 21% is found to have raised total cholesterol (Karim *et. al.*, 2013). Dyslipidaemia in terms of at least one lipid abnormality as found in T2DM patients is as high as 84.4% (Karim *et. al.*, 2013).

Around 55.7% of the patients had two or more abnormality, 33% had three or more abnormality, and 9% had all four lipid parameters abnormal (Karim *et. al.*, 2013).

Poor glycaemic control is a predictor of all types of dyslipidaemia (Abdel-Aal *et.al.*, 2008, Karim *et. al.*, 2013). Duration of T2DM is associated with increased risk of having higher TC, LDL and lower HDL and does not seem to affect triglyceride (Karim *et. al.*, 2013). A national cross-sectional chart audit study of 2473 Canadian patients with type 2 diabetes

showed 55% of patients with a diagnosis of diabetes of 2 years had dyslipidaemia. This proportion rose to 66% in patients with diabetes for 15 years (Harris et. al., 2005). T2DM with co-morbid hypertension seems to predict hypertriglyceridemia and lower HDL (Karim et. al., 2013)

Persons with DM have average LDL-C levels in excess of 140 mg/dl ((Erdman et.al., 2002). Hypertriglyceridemia is associated with insulin resistance in type 2 diabetes mellitus (Singh et.al., 2011). There is a good correlation between insulin resistance and plasma TG concentration. as TG may influence an early step in insulin action pathway; alternatively, insulin resistance may cause hypertriglyceridemia (Ravi et.al., 2004). With the growing prevalence of obesity, insulin resistance, and type 2 diabetes in our communities, prevention and management of this dyslipidemic state is critically important for the prevention of coronary artery and macrovascular disease (Koba et. al., 2000).

2.11.3.3 CARDIOVASCULAR DISEASES

Cardiovascular disease (CVD) due to atherosclerosis is the leading cause of morbidity (Burnett, 2004). Atherosclerotic lesions form *de novo* from focal accumulation of lipoproteins, monocyte-derived macrophages, and lymphocytes within the arterial wall (Burnett, 2004). Atherosclerosis is a form of arterial disease that manifests in the coronary circulation as coronary heart disease (CHD), in the carotid arteries as cerebrovascular disease (otherwise called stroke), and in the aorta and lower extremity arteries as peripheral arterial disease (PAD). Dyslipidaemia is recognized as a prominent risk factor for cardiovascular disease (Yusuf et. al., 2004). Furthermore, increasing evidence points to insulin resistance, which results in increased levels of plasma triglycerides and low-density lipoprotein cholesterol (LDL-C) and a decreased concentration of high-density lipoprotein cholesterol (HDL-C) as an important risk factor for peripheral vascular disease (Wild et. al. 2009), stroke and coronary artery disease (Rodriguez-Colon et. al . 2009).

The Atherosclerosis Risk in Communities (ARIC) study after 10-year follow-up of 12,339 middle-aged participants with resultant 725 coronary heart disease events, showed strong associations of total cholesterol, LDL-C, and TG with increased CHD risk and of HDL-C with decreased risk (Sharrett, et.al., 2001).

ARIC study reported that CHD risk is elevated by approximately 40% for every mmol/L increment in LDL-C (Sharrett, et.al., 2001). The lowest incidence of CHD is observed in

those at the lowest LDL cholesterol (LDL-C) quintile, with medians of 88 mg/dL in women and 95 mg/dL in men, and risk accelerates at higher levels, with RRs for the highest quintile of 2.7 in women and 2.5 in men (Sharrett, et.al., 2001). Evidence from primary prevention trials in predominantly male populations shows that CHD incidence can be reduced 31% to 37% by 25% LDL-C reductions from levels of 4 to 5 mmol/L (Goto, 2000). Low blood levels of high-density lipoprotein cholesterol (HDL-C) is a strong, independent risk factor for CVD (Boden, 2000; Franceschini, 2001). HDL-C associations with CHD are continuous and strong, and the associations, like LDL-C, are independent of other lipids (Sharrett, et.al., 2001). HDL-C is associated with CHD protection with top quintile RRs of 0.16 in women and 0.36 in men (Sharrett, et.al., 2001).

Triglyceride level is a significant CHD risk factor (Sarwar et al., 2007). There is a continuous, positive association between usual triglyceride levels and the risk of CHD that persisted after adjustment for age, sex, blood pressure, smoking, and total-to-HDL cholesterol ratio (Asia Pacific Cohort Studies Collaboration, 2007). Moreover, analysis of 29 prospective studies in Western populations consistently indicates moderate and highly significant associations between triglyceride values and coronary heart disease risk (Sarwar et al., 2007). An 88 mg/dl increase in TG levels significantly increases the relative risk of CHD by up to 30% in men and 75% in women (Cullen, 2000). The combined odds ratio for CHD in Western populations, adjusted for several established risk factors, is approximately 1.7 in individuals with usual triglyceride values in the top third of the population compared with those in the bottom third (Sarwar et al., 2007).

Ischemic stroke subjects have higher total cholesterol levels compared to control subjects even after adjustment for confounders (Tirschwell et. al., 2004). Each 50 mg/dl rise in total cholesterol increases the risk of ischemic stroke 22% (Tirschwell et. al., 2004). The prevalence of stroke increases with rising of LDL-C level (Xing et. al., 2009). The prevalence of stroke in LDL-C \geq 3.32 mmol/L group increased 4.2 times as compared with that in LDL-C < 2.00 mmol/L group (Xing et.al., 2009). After adjustment for age, sex and smoking LDL-C level was positively associated with the development of stroke with the odds ratio of 2.35 (Xing et. al., 2009). Compared with those in the lowest fifth of triglycerides, the risk of fatal or nonfatal stroke among individuals in the highest fifth was increased by 50% (Asia Pacific Cohort Studies Collaboration, 2007). The hazard ratio for the risk of fatal or nonfatal ischemic stroke comparing the highest and lowest fifths of triglyceride was 1.97 (Asia Pacific Cohort Studies Collaboration, 2007). Epidemiological

studies have supported an association between elevated HDL-C level and decreased risk of stroke (Koren-Morag, *et. al.*, 2002; Soyama *et. al.*, 2003). Higher levels of HDL cholesterol are associated with a significant decrease in risk of nonfatal stroke even after adjustment for potential confounders (Wannamethee *et.al.*, 2000). Prospective cohort studies reporting on relative risk per unit increase in HDL-C have shown an 11–15% decreased stroke risk per 10-mg/dl increase in HDL-C (Koren-Morag, *et. al.*, 2002; Walldius, 2006). The association between low HDL-C and increased MI risk is strong and consistent in older adults (Psaty *et.al.*, 2004).

2.11.4 DYSLIPIDAEMIA AND MORTALITY

Dyslipidaemia is one of the most important risk factors for many chronic non-communicable diseases resulting in serious and mortality, and medical costs worldwide (Stamler *et.al.*, 2000; WHO, 2002). Men with unfavourable serum cholesterol levels have strong gradients of relative mortality risk (Stamler *et.al.*, 2000). For men with serum cholesterol levels of 240 mg/dl or greater CHD mortality risk was 2.15 to 3.63 times greater than that of men with serum cholesterol favourable levels of 200 or lower (Stamler *et.al.*, 2000) ; CVD disease mortality risk was 2.10 to 2.87 times greater; and all-cause mortality was 1.31 to 1.49 times greater (Stamler *et.al.*, 2000). Hypercholesterolemic men had age-adjusted absolute risk of CHD death of 59 per 1000 men in 25 years in the Chicago Heart Association Detection Project in Industry (CHA) cohort; 90 per 1000 men in 34 years in the Peoples Gas Company Study (PG) cohort ; and 15 per 1000 men in 16 years in the Multiple Risk Factor Intervention Trial (MRFIT) cohort (Stamler *et.al.*, 2000). Absolute excess risk was 43.6 per 1000 men (CHA), 81.4 per 1000 men (PG), and 12.1 per 1000 men (MRFIT) (Stamler *et.al.*, 2000). Men with favourable baseline serum cholesterol levels had an estimated greater life expectancy of 3.8 to 8.7 years (Stamler *et.al.*, 2000).

There is a significant, positive, continuous graded relationship of serum cholesterol level to long-term mortality from all causes (Stamler *et.al.*, 2000). Substantially longer estimated life expectancy of 3.8 to 8.7 years longer is observed for younger adult men with favourable serum cholesterol levels (<200 mg/dl) than those with unfavourable levels (Stamler *et.al.*, 2000).

There is a continuous, graded, strong, independent relationship of serum cholesterol level to long-term risk of CHD and CVD death (Stamler et.al., 2000). With high serum cholesterol levels in young adulthood, there is substantial absolute risk and absolute excess risk of CHD and CVD death over the decades from young adulthood through middle age (Stamler et.al., 2000).

Compared with those individuals grouped in the lowest fifth of usual triglyceride values, the risk of fatal CHD was 70% greater for participants with usual triglyceride levels in the highest fifth (Asia Pacific Cohort Studies Collaboration, 2007). Triglycerides appears to predict CHD mortality in older women but not in men (Mazza, et.al., 2005). In women, the predictive role of triglycerides remains significant after adjustment for diabetes, obesity, alcohol consumption and history of CHD, and even increases after including low HDL-C in the model (Mazza et.al., 2005).

Cross-sectional and prospective studies have identified high HDL-C levels as a characteristic of individuals who survive to exceptionally old age (Barzilai et.al., 2003; Weverling-Rijnsburger et.al., 2003; Koropatnick et.al., 2008). In a cohort of Physician health study men who survived to age 90 had significantly higher HDL-C levels at baseline than those who died before age 90 (Rahilly-Tierney et.al., 2012). A significant association exists between increasing HDL-C levels and decreasing risk for all-cause mortality before age 90 even after adjustment for potential confounders (Rahilly-Tierney et.al., 2012). HDL-C also has a protective effect of on CVD, the most common cause of death among older persons (Rahilly-Tierney et.al., 2012).

There is a continuous, graded, strong, independent relationship of serum cholesterol level to long-term risk of CHD and CVD death (Stamler et.al., 2000). With high serum cholesterol levels in young adulthood, there is substantial absolute risk and absolute excess risk of CHD and CVD death over the decades from young adulthood through middle age (Stamler et.al., 2000).

Compared with those individuals grouped in the lowest fifth of usual triglyceride values, the risk of fatal CHD was 70% greater for participants with usual triglyceride levels in the highest fifth (Asia Pacific Cohort Studies Collaboration.2007). Triglycerides appears to predict CHD mortality in older women but not in men (Mazza, et.al., 2005). In women, the predictive role of triglycerides remains significant after adjustment for diabetes, obesity, alcohol consumption and history of CHD, and even increases after including low HDL-C in the model (Mazza et.al., 2005).

Cross-sectional and prospective studies have identified high HDL-C levels as a characteristic of individuals who survive to exceptionally old age (Barzilai et.al., 2003; Weverling-Rijnsburger et.al., 2003; Koropatnick et.al., 2008). In a cohort of Physician health study men who survived to age 90 had significantly higher HDL-C levels at baseline than those who died before age 90 (Rahilly-Tierney et.al., 2012). A significant association exists between increasing HDL-C levels and decreasing risk for all-cause mortality before age 90 even after adjustment for potential confounders (Rahilly-Tierney et.al., 2012).HDL-C also has a protective effect of on CVD, the most common cause of death among older persons (Rahilly-Tierney et.al., 2012).

CHAPTER THREE

METHODOLOGY

3.1 STUDY AREA:

The study was carried out at the University College Hospital, Ibadan, Oyo state.

Oyo State consists of 33 local government areas (Oyo State website, 2013). The capital of the state is Ibadan, the most populous city in black Africa with about two and a half million people (Oyo State website, 2013). Ibadan had been the centre of administration of the old Western Region, Nigeria since the days of the British colonial rule (Oyo State website, 2013). There are eleven local governments in Ibadan Metropolitan area consisting of five urban local governments in the city and six semi-urban local governments in the less city (Tomori and Owoeye, 2013).

The city of Ibadan is known to be the third largest metropolitan area in Nigeria, after Lagos and Kano (NPC, 2006). Ibadan is one of the cities in Nigeria that have experienced rapid urbanisation and industrialisation (GKW, 2003). The increase in urbanization is attributed to the provision of better economic opportunities due to setting up of factories and industries, which has led to migration of population from rural areas to the city (Owoeye, 2013).

Industries in Ibadan city engage in processing of agricultural products, brewing, food products and the manufacture of cigarettes. The city is the site of several major research institutes like International Institute of Tropical Agriculture (IITA), Cocoa Research Institute (CRI), Forestry Research Institute (FRI), National Horticultural Research Institute (NHRI), and Nigerian Institute of Social and Economic Research (NISER). Most of Nigeria's leading publishing companies are also based in the city (GKW, 2003).

A number of important public institutions that were established in Ibadan have contributed immensely to its urban growth. They include the University of Ibadan, the University College Hospital (UCH), Moor Plantation, Premier Hotel, Nigerian Institute of Social and Economic Research (NISER), International Institute of Tropical Agriculture (IITA), Central Bank of Nigeria, the Army and Police Command Headquarters, various agricultural research institutes, television and radio stations such as the NTA and several others. (Central Bank of Nigeria, 1999).

The University College Hospital (UCH) located in Ibadan North Local Government Area is the premier teaching hospital in Nigeria. UCH, Ibadan is an 850-bed tertiary care hospital with staff strength of over 5,233. The hospital comprises 34 departments which are made up of clinical and non-clinical departments. The clinical departments are 25 while the non-clinical are 9. There are different health professional groups working within these departments and include Medical and Dental Doctors, Nurses, Physiotherapists, Medical Social Workers, Pharmacists, Radiographers, Medical Laboratory Scientists, Dieticians, Medical Record Officers, Accountants and Administrators. The combination of Nurses, Physiotherapists, Medical Social Workers, Pharmacists, Radiographers, Medical Laboratory Scientists, Medical Record Officers and Dieticians are often referred to as Allied Health workers.

The workers within the hospital are also classified into senior and junior staff based on their salary grade level. Junior staff of UCH are of cadre, CONHESS 1 to 6 while senior staff are of cadre, CONHESS 7 to 15. The senior and junior staff constitutes about 40% and 60% of the total staff respectively.

UCH has a staff clinic department which take care of staff that are ill. The clinic used to be funded by UCH itself until the Obasanjo Presidential regime when National Health Insurance Scheme (NHIS) was established. NHIS services however do not fund the screening of health for staff. The hospital used to have gymnasium and swimming pool in the senior staff club to cater for the recreational activities of the staff however these facilities are now dilapidated.

3.2 STUDY DESIGN

A cross sectional study was employed in order to answer the research questions of determining the prevalence and identifying the risk factors of obesity, hyperglycaemia and dyslipidaemia among UCH, Ibadan senior staff.

3.3 STUDY POPULATION: Allied health workers (Nurses, Physiotherapists, Medical Social Workers, Radiographers, Health record officers, Medical Laboratory Scientists, Dieticians, and Medical Record Officers) and Accountants as well as Administrators working in UCH are the subjects of this study.

3.3.1 Inclusion Criteria: The study population constitutes senior cadre staff (of between CONHESS 8 and 13; from basic senior staff to Assistant Directors) of UCH who are Nurses, Physiotherapists, Medical Social Workers, Radiographers, Health record officers, Medical Laboratory Scientists, Dieticians, Accountants and Administrators

3.3.2 Exclusion Criteria: Pregnant women, breastfeeding mothers and individuals with lower limbs deformities in whom body mass index cannot be measured

Study Duration: The study was carried out for over three months

3.4 SAMPLE SIZE DETERMINATION

Formula for single proportion was used in calculating the sample size (Cochran, 1963):

$$n = z_{\alpha/2}^2 p (1-p)/d^2,$$

Where:

n= minimum sample size to be determined

$z_{\alpha/2}$ = The standard normal deviate, usually set at 1.96 which corresponds to 95% confidence interval (two-sided test).

Prevalence of diabetes among senior staff of Oyo State Secretariat Civil Servants, Ibadan according to Ojewale and Adejumo, 2014 was 4.7% giving rise to a sample size of 69.

P= Prevalence of dyslipidaemia amongst apparently healthy professionals in Asaba is 60% (Edo and Enofe, 2013) giving rise to a sample size of 369.

p= Prevalence of obesity among Nurses in Akwa Ibom State was 62.2% giving rise to a sample size of 369.

Therefore the sample size for this study is 369

$$z = 1.96$$

$$1-p = 0.4$$

$$d = \text{margin of error of } 5\% = 0.05$$

$$n = (1.96)^2 \cdot 0.6(0.4) = 369$$

$$0.05^2$$

Adjusting for non-response rate (Ns) of 10%,

$$Ns = n \times 1/1-r, \text{ where } r = \text{proportion of non-response}$$

$$= 369(1/1-0.1) = 369/0.9 = 410$$

3.5 SAMPLING TECHNIQUE

The participants recruited (371 subjects) for this study make up 27.7 % (1,338 subjects) of the total senior staff of UCH between CONHESS 8 and 13..

The senior staff list of UCH, according to the professional groups, was obtained at the Human Resources Department of UCH. The list served as the sampling frame. Stratified random sampling technique was employed in the selection of participants to ensure that the sample is representative of the senior staff of UCH (Table 3.1). The senior staff were stratified according to the professional groups namely; (1) Nurses (2) Medical laboratory Scientists, (3) Administrators (4) Physiotherapists (5) Accountants (6) Health record officers (7) Medical Social Workers (8) Radiographers and (9) Dieticians. The sample size, 371, was proportionally allocated to all the professional groups.

Simple random sampling was used in the selection of subjects (Appendix IV) for each of the professional groups by cadre. The names of subjects according to their professional group were arranged by identification number in each cadre. Table of random numbers was used to select the sample of 371 subjects. When a subject is not eligible the next individual according to table of random numbers was selected. Nurses made up 81.1 % of the senior staff recruited, 7.3% were Medical Laboratory Scientists, 3.8% were Administrators, 2.7% were Physiotherapists, 1.3% were Accountants, 1.3% , were Health record officers, 1.1% were Medical Social Workers, 0.8% were Radiographers while 0.5% were Dieticians

Table 3.1 Proportional allocation of Senior Staff on CONHESS 8 to 13

Professional Category	Number of staff on CONHESS 8 to 13	Number of sample	% of total
Nurses	1083	301	81.1 %
Medical Laboratory Scientists (MLS)	98	27	7.3%
Administrators	68	14	3.8%
Physiotherapists	35	10	2.7%
Accountants	34	5	1.3%
Health Record officers (HRO)	21	5	1.3%
Medical Social Workers (MSW)	16	4	1.1%
Radiographers	23	3	0.8
Dieticians	10	2	0.5%
Total	1388	371	100%

3.6 DATA COLLECTION

The data collection commenced after taking approval from the UI/UCII ethical committee. Research assistants were recruited and the protocol of this study was followed to train them on blood collection and the measurement of the anthropometric indices. The selected participants were visited at their departments by research assistants and the principal investigator. The participants visited were evaluated for eligibility to participate in the study using the exclusion criteria. The next person on the list of randomised subjects was recruited when a potential participant refused to participate. The procedures of the study was explained to the prospective subject of the study and written, informed consent was obtained from those who met the inclusion criteria and agrees to participate in the study. An appointment was fixed with consenting participants for another day for administration of questionnaire, collection of fasting plasma sample, blood pressure and physical body (weight, height, waist circumference and hip circumference) measurements.

3.6.1 QUESTIONNAIRE

The questionnaire was semi-structured (Appendix III) and was self-administered by the participants. The questions were borrowed from the WHO STEPwise approach to non-communicable disease risk factor surveillance (STEPS) questionnaire. The questionnaire was face-validated by my supervisors (an expert epidemiologist and medical statistician) and another Physician/Epidemiologist. They reviewed the questions to ensure they addressed the objectives of the study.

The questionnaire had five sections:-

- Section A: Socio-demographic data such as age, sex, and educational level
- Section B: Occupational data like job specification and duration on the job
- Section C: Physical Activity, Lifestyle/Diet, Medical and Drug History
- Section D: Anthropometric measurements (height, weight, BMI) and blood pressure
- Section E: Laboratory investigations(Fasting plasma glucose and fasting lipid profile)

3.6.2 PRETEST

Pretest of the questionnaire was done at the Adeoyo Maternity Teaching Hospital, Ibadan on January, 2014. The hospital is a 255 bed setting with staff strength of 1,510. Wooden measuring rod (stadiometer), a non-expandable measuring tape and OMRON BF-400 weighing scale, OMRON Blood Pressure Monitor were also tested along with the questionnaire.

The questionnaire was pre-tested on 25 senior staff of Adeoyo Maternity Teaching Hospital, Yemetu, Ibadan to determine its acceptability, comprehensibility and ease of administration among the study participants. The pretest was carried out in order to identify potential problems, to test the accuracy of the instruments as well as to revise the methods and logistics of data collection before starting the actual fieldwork. The questions were found easy to comprehend by the subjects.

3.6.3 MEASUREMENTS

Anthropometric measurements taken include height (in centimetre), weight (in kilogram), percentage body fat (%), waist circumference (in centimetre) and hip circumference (in centimetre). The height measurement was taken using a wooden measuring rod, the weight and BMI were taken with OMRON BF-400 digital weighing scale, while the waist and hip circumferences were measured with a measuring tape. Blood Pressure was also measured with Omron (M2Basic) Blood Pressure Monitor. One research assistant was assigned to the measurements.

3.6.3.1 Height measurement: A wooden rod which had been graduated up to 2m was placed against a wall. Each of the participants was made to stand barefooted with his or her back (scapulae), buttocks and both heels together touching the rod. The participant was made to stand with his or her head erect, the arms in relaxed position and hanging loosely at the sides and the shoulders were made to relax. The participant was asked to look straight ahead and stand tall. The participant was also asked to inhale and the measurement will be recorded before the subject exhales. The participants were reminded not to alter their position by, for example, raising the heels off the floor as they breathe in. A ruler was placed on the head to flatten the hair, the measurement was taken and recorded as centimetres and converted to meters. Height was measured to the nearest 0.1 cm (US NIH, 1998).

3.6.3.2 Weight and BMI measurement

Omron weighing scale (OMRON Healthcare) was used in taking the weight and body mass index (BMI) measurements. The age, sex and height of a participant were entered into the digital weighing scale after it was powered on. The machine was allowed auto-calibrate and the participant in light clothing was asked to step onto the scale. The scale displayed the BMI, percentage body fat and weight after a minute of the participant's standing on the scale and the values were recorded in the appropriate units. Weight was measured to the nearest 0.1kg

BMI is an index of obesity that assesses body weight relative to a person's height. It is calculated as the weight in kilograms divided by the square of the height in meters (kg/m^2). (WHO 2000; 2004). This study classified BMI of participants into four groups: underweight ($\text{BMI} < 18.5$), normal weight ($\text{BMI } 18.5\text{-}24.9$), overweight (BMI of between 25.0 and 29.9) and obesity ($\text{BMI} \geq 30$) (WHO, 2000).

3.6.3.3 Waist and Hip Circumference Measurements

These measurements were made according to US NIH specifications. The waist circumference (WC) was measured at the level of the umbilical cord while the hip circumference (HC) was measured at the broadest part of the buttocks.

Waist circumference was measured while the participant was standing and asked to hold up his or her dress around the level of the waist. The measuring tape was placed around the trunk in a horizontal plane at the level of umbilicus. The tape was ensured to be snug and parallel to the floor, but did not compress the skin. The measurement is then made at minimal respiration to the nearest 0.1 cm (US NIH, 1998). Waist circumference of >102 cm in men and > 88 cm in women is regarded as abdominal obesity (WHO, 1997).

Hip circumference was measured while the participant was standing. The participant was made to stand erect with the feet together and weight evenly distributed on both feet while holding up his or her dress. The measuring tape was held snug but not tight at the maximum extension of the buttocks. The tape was adjusted and the front and sides checked so that the plane of the tape was horizontal. The zero end of the tape was held under the measurement value. The measurement was taken from the right side and the value recorded to the nearest 0.1cm (US NIH, 1998).

3.6.3.4 Waist to Hip Ratio (WHR)

It was derived from the ratio of waist circumference to that of hip circumference.

$$\text{WHR} = \frac{\text{Waist circumference (cm)}}{\text{Hip circumference (cm)}}$$

Waist to hip ratio of >0.95 in men and > 0.85 in women is regarded as abdominal obesity

3.6.3.7 Blood Pressure Measurement

Omron Blood Pressure Monitor, M2 Basic, (OMRON Healthcare) was used in taking the blood Pressure measurements. The participant was made to relax for five minutes after which he or she was made to be in a relaxed, seated position and asked not to talk or move during measurement. Tight fitting clothing was removed, the participant was assisted to sit upright, the arm on a table in front of him or her, the cuff was applied around the arm and made to be at the level of the heart. The cuff was connected to the monitor and powered. The result was displayed and recorded. Two measurements were made at two minutes interval.

Hypertension: a person was said to have hypertension if he/she is on medication or has a systolic blood pressure (SBP) of ≥ 140 mmHg and /or a diastolic blood pressure (DBP) of ≥ 90 mmHg.

Prehypertension: a person was said to have prehypertension if he/she has a systolic blood pressure (SBP) of between 130 and 139 mmHg and /or a diastolic blood pressure (DBP) of between 80 and 89 mmHg.

3.6.4 BLOOD COLLECTION

Eight (8) millilitres of blood samples were aseptically collected from all participants after an overnight fast of between 10 and 12hours. 4mls were dispensed into EDTA- containing anticoagulant tubes for plasma lipid profile determination and the remaining 4mls into fluoride oxalate- anticoagulant tubes for the determination of fasting plasma glucose. Blood samples were centrifuged within 10 minutes of collection at 4,000r.p.m for 5 minutes in Measurement Scientific Equipment (MSE), United Kingdom, centrifuge machine. The plasma was harvested, stored frozen at -20°C and analyzed within one week (Chessbrough, 2002).

3.6.5 BIOCHEMICAL ANALYSIS

Fasting lipid profile comprises of total cholesterol, triglycerides, HDL- cholesterol and LDL-cholesterol. Total cholesterol, triglyceride and glucose were estimated by enzymatic methods. HDL-cholesterol was estimated as for total cholesterol after the precipitation of the other lipoproteins while LDL-cholesterol was calculated from the friedwald's equation (Baker, 2003). For details on biochemical analysis see Appendix V

Impaired fasting glucose (IFG): when fasting plasma glucose is between 100 and 125mg/dl

Diabetes (DM): refers fasting plasma glucose greater than 126mg/dl or two hours post prandial greater than 200 mg/dl

Dyslipidaemia: is an abnormal blood lipid. triglycerides greater than 130mg/dl(hypertriglyceridemia) or total cholesterol greater than 200mg/dl (hypercholesterolaemia) or HDL cholesterol less than 40 mg/dl in men and postmenopausal women or less than 50 mg/dl in premenopausal women or LDL-cholesterol greater than 150 mg/dl. Dyslipidemia was defined according to guidelines from the US NCEP ATP III diagnostic criteria.

3.6.6 DATA QUALITY ASSURANCE: Questionnaires were checked for omissions and errors at the end of each day and corrections were effected appropriately. The weighing scale and the stadiometer were checked for accuracy and precision from time to time. Standard weights Blood samples were collected using standard procedures, quality control materials were included in laboratory tests and standard operating procedures were adhered to.

3.7 DATA ANALYSIS

The data were entered, cleaned and analysed using SPSS 19.0. Univariate analyses for descriptive statistics such as frequencies, percentages, proportions, means and standard deviation were generated to describe the distribution of variables such as socio-demographic factors, anthropometric indices, biochemical parameters, blood pressure, physical activity and sports activity.

Bivariate analysis (Chi square and Fisher's exact tests) were used to test for significant associations between categorical independent (socio-demographic, occupational data) and dependent variables (Obese, hyperglycaemia and dyslipidaemia). All statistical tests of significance were two-sided, at 5% level of significance.

Subsequently, independent variables that were significant at 10% level of significance were included into multiple logistic regression analysis to identify the independent determinants of obesity, hyperglycaemia and dyslipidaemia. Crude odds ratio, adjusted odds ratio and 95% C.I were used to calculate the strength of the association.

3.7 OPERATIONAL DEFINITION OF TERMS

3.7.1 Alcohol consumption: refers to a participant's behaviour of consumption of beverages containing ethyl alcohol (Encyclopedias Britannica, 2014). The universally acceptable measure of alcohol consumption is called a standard drink. The net alcohol content of a standard drink is approximately 10g of ethanol (WHO). 1 standard drink is equivalent to 1 standard bottle of regular beer (285ml) or 1 single measure of spirits (30ml), 1 medium size glass of wine (120ml) or 1 measure of aperitif (60ml).

In this study four components of alcohol consumption and drinking patterns were measured. These include 'drinker' & 'non-drinker', the frequency of drinking (i.e. days per week/month), the number of drinks consumed on a typical drinking occasion, and the type of alcohol consumed. From these measures an overall estimate of total pure alcohol consumed weekly was determined. 'Ex-drinker' questions included reasons why they stopped drinking. 'Drinkers' and 'non-drinkers' were analysed separately. The 'non-drinkers' group consisted of i) 'never-drinkers', ii) 'ex-drinkers', and iii) 'occasional drinkers, not monthly'.

3.7.2 Physical Activity Status

There are 3 levels of physical activity; low, moderate and severe.

Low physical Activity:

- No activity is reported **OR**
- Some activity is reported but not enough to meet categories 2 or 3.

2- Moderate physical Activity:

Either one of the following 3 criteria

- 3 or more days of vigorous activity of at least 20 minutes per day **OR**
- 5 or more days of moderate intensity activity and / or walking of at least 30minutes per day **OR**
- 5 or more days of any combination of walking, moderate intensity or vigorous intensity activities achieving a minimum or at least 600 Metabolic Equivalent Task (MET) minutes/week.

3- High physical Activity:

Any one of the following 2 criteria

- Vigorous intensity activity on at least 3 days and accumulating at least 1500 MET-minute/week **OR**
- 7 or more days of any combination of walking, moderate or vigorous intensity activity accumulating at least 3000 MET-minutes/week.

Formula for computation of MET-minutes/week:

Walking MET-minutes/week = $3.3 * \text{walking minutes} * \text{walking days}$

Moderate MET-minutes/week = $4.0 * \text{moderate-intensity activity minutes} * \text{moderate days}$

Vigorous MET-minutes/week = $8.0 * \text{vigorous-intensity activity minutes} * \text{vigorous intensity days}$

Total physical activity MET-minutes/week = sum of Walking + Moderate + Vigorous MET minutes/ week scores (WHO STEPS questionnaire).

3.7.3 Sports activity: involvement in sports and was classified as Yes or No

3.7.4 Sedentary time: sitting or reclining time on a typical day.

Normal- ≤ 4 hours/day, High- >4 hours/day

3.8 Ethical considerations

Ethical approval was obtained from the UI/UCH ethical committee / IRB committee (Appendix I). Research procedure was explained to the participants and written informed consent was obtained (Appendix II). In addition, subjects were informed that they could withdraw from the study any time they wanted. All data were recorded and stored in a locked cabinet. Questionnaires were made anonymous (no name was written) and only identified by codes. The blood samples were collected by competent phlebotomists so as to minimize stress and pain. Participants who desired to know the results of the blood tests were given their results and those who had abnormal results were referred to the staff clinic of the University College Hospital, Ibadan for appropriate intervention.

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CHAPTER FOUR

RESULTS

A total of 410 subjects were approached to participate in the study out of which 371 returned fully completed questionnaire giving a response rate of 90%. The results of this study are presented in this chapter.

4.1 Characteristics of the participants

The socio-demographic characteristics of respondents are presented in Table 4.1. Majority, 323 (87.1%), of the participants were females while 48 (12.9%) were males. Over 90% were married, 347 (93.5%), 21 (5.7%) were single and only 3 (0.8%) were widowed. Most participants, 316 (85.2%), were Yorubas, 42 (11.3%) were Igbos, 13 (3.5%) were of other tribes. Thirty-two (8.6%) of participants had Higher National Diploma (HND), 2 (0.5%) had Registered Nursing (RN), 130 (35%) had Registered Midwifery (RM), 148 (39.9%) had Bachelor of Science (BSc), 50(13.5%) had Masters of Science (MSc), 2(0.5%) had Doctor of Philosophy(PhD) certificates while 7(1.9%) had other types of certificates .Participants who had post secondary school education other than PhD constitute 319 (86%) while only 52 (14%) had postgraduate certificates. The mean age of the participants was 41.5 ± 7.6 years, the youngest being 27 years old and the oldest was 60 years old. A small proportion of participants 11 (3%) were aged less than 30 years old, over 155 (40.0%) aged 30-39, 138 (37.2%) aged 40-49, 66 (17.8%) aged 50-59 and only one (0.3 %) aged 60 years old. Participants that are less than 40 years old make up 166 (44.7%) while those older than or equal to 40 years old constitute 205 (55.3%).

Only, 18 (4.9%) of the study subjects consumed alcohol and one (0.3%) used tobacco.

Table 4.1: Socio-demographic characteristics of the Participants (N = 371)

Variables	n (%)
Gender	
Male	48(12.9)
Female	323(87.1)
Marital status	
Married	347 (93.5%)
Single	21(5.7%)
Widow	3 (0.8%)
Ethnicity	
Yoruba	316(85.2%)
Ibo	42(11.3%)
Others	13(3.5%)
Educational status	
HND	32(8.6%)
RN	2(0.5%)
RM	130(35%)
BSc	148(39.9%)
MSc	50(13.5%)
PhD	2(0.5%)
Others	7(1.9%)
Age group (years)	
<30	11(3%)
30-39	155 (41.8%)
40-49	138(37.2%)
50-59	66(17.8%)
≥60	1(0.3%)
Current alcohol consumption	
No	334(90%)
Yes	38(10%)
Tobacco Use	
No	370(99.7%)
Yes	1(0.3%)

4.2 Distribution of Physical activity, Physical Stress, Sports Activity, Hypertension and Prehypertension

Frequency distribution according to level of Physical activity, Physical Stress, Sports Activity, Hypertension and Prehypertension among participants are as displayed in Table 4.2

Majority of the participants, 346 (94.3%), engaged in little or no physical activity, 20 (5.4%) practised moderate physical activity and one (0.3%) practised vigorous physical activity. The proportion of participants that experienced physical stress was 167 (46.0%) while 200 (54.5%) did not. Three hundred and thirteen (84.4%) did not engage in sports activity compared to 54 (14.6%) who did. The proportion of participants who had high sedentary time was 228 (61.5%) while 136 (36.7%) had normal sedentary time. Forty-two (11.3 %) of study participants had hypertension while 329 (88.7%) did not. Thirty-one (8.4%) participants were on anti-hypertensive drugs. Also, 87 (23.5%) of participants had prehypertension compared to 76.5% who did not.

Table 4.2: Frequency Distribution of Physical activity, Physical Stress, Sports Activity, Hypertension and Prehypertension, N=371

Variable	n (%)
Physical activity, N = 371	
No or Low Physical activity	346(94.3%)
Moderate Physical Activity	20(5.4%)
Vigorous physical activity	1(0.3%)
Physical stress, N = 371	
Yes	167(45.5%)
No	200(54.5%)
Engaged in sports activity , N = 371	
Yes	54(14.6%)
No	313(84.4%)
Sedentary Time, N= 364	
Normal (≤ 4 hours /day)	136(36.7%)
High (>4 hours/day)	228(61.5%)
Hypertension	
Yes	42(11.3%)
No	329(88.7%)
Prehypertension	
Yes	87(23.5%)
No	284(76.5%)

4.3 Anthropometric measurements of the participants

Table 4.3 shows the means of the anthropometric measurements of the participants. The mean BMI for all subjects was $28.4 \pm 4.5\text{kg/m}^2$. The mean percentage body fat (% BF) was 22.0 ± 6.1 % and 40.7 ± 5.6 % for men and women respectively. Also the mean waist circumference was $92.1 \pm 12.3\text{cm}$ for men and 93.7 ± 9.2 for women. The mean hip circumference was 101.5 ± 9.8 for men and 105.8 ± 8.6 for women. Waist to hip ratio was 0.90 ± 0.1 for men and 0.0 ± 0.1 for women.

Table 4.3: Mean of anthropometric measurements of participants.

Anthropometric measurements	Mean \pm SD
Body mass index(kg/m^2)	28.35 ± 4.53
% Body Fat	
Men	21.95 ± 6.12
Women	40.65 ± 5.64
Waist Circumference(cm)	
Men	92.07 ± 12.32
Women	93.67 ± 9.21
Hip circumference(cm)	
Men	101.51 ± 9.75
Women	105.77 ± 8.58
Waist to hip ratio	
Men (n=48)	0.90 ± 0.05
Women (n=323)	0.89 ± 0.05

4.4 Biochemical and Blood Pressure measurements of study participants

Table 4.4. shows the mean of biochemical and blood pressure measurements of participants. The mean fasting plasma glucose was 86.9 ± 18.2 mg/dl, mean plasma total cholesterol was 185.0 ± 37.1 mg/dl, mean plasma triglycerides was 73.9 ± 29.2 mg/dl while the mean plasma LDL-C was 121.3 ± 36.2 . The mean HDLC in men and women was 44.5 ± 8.0 and 49.5 ± 9.7 respectively. The mean systolic and diastolic blood pressure was 113.8 ± 10.4 and 72.7 ± 8.9 , respectively.

Table 4.4: Mean of the biochemical and blood pressure measurements of participants.

Parameters	Mean ± SD
Fasting plasma glucose	86.93 ± 18.16
Total Cholesterol	184.95 ± 37.11
Triglycerides	73.88 ± 29.21
LDL-C	121.34 ± 36.17
HDL-C	
Men	44.47 ± 7.97
Women	49.48 ± 9.74
Blood Pressure	
Systolic	113.82 ± 10.41
Diastolic	72.70 ± 8.90

4.5: Prevalence of obesity, abdominal obesity, hyperglycaemia and dyslipidaemia among participants.

Table 4.5 depicts the prevalence of obesity, abdominal obesity, hyperglycaemia and dyslipidaemia of the participants.

One (0.3%) participant was underweight, 81 (21.8%) were of normal weight, 160 (43.1%) were overweight, 129 (34.8%) were obese and 263 (71.0%) of the participants had abdominal obesity.

With respect to hyperglycaemic status about 19 (5.0%) had impaired fasting glucose, 6 (1.6%) had diabetes and 346 (93.3%) were normoglycaemic. Newly diagnosed diabetes was 2 (0.5%).

The participants who had hypertriglyceridaemia were 10 (2.7%), 123 (33.2%) had hypercholesterolemia, 168 (45.3%) had low HDL cholesterol, 146 (39.4%) had high LDL cholesterol and overall 241 (65.0%) had dyslipidaemia.

Table 4.5: Prevalence of obesity, abdominal obesity, hyperglycaemia (IFG and type 2 DM) and dyslipidaemia among participants, N = 371

Parameters	n (%)
General obesity status	
Underweight	1(0.3%)
Normal	81(21.8%)
Overweight	160(43.1%)
Obese	129(34.8%)
Abdominal obesity status	
Yes	263(70.9%)
No	108(29.1%)
Hyperglycaemic Status	
IFG(>100mg/dl)	19(5.1%)
Newly diagnosed T2DM	2(0.5%)
T2DM(>200mg/dl)	6(1.6%)
Normal(50-100mg/dl)	346(93.3%)
Triglycerides	
Normal (<150 mg/dl)	361(97.3%)
High(>150 mg/dl)	10(2.7%)
Total Cholesterol	
Normal (<200mg/dl)	248(66.8%)
High(>200mg/dl)	123(33.2%)
HDL-C status	
Low	168(45.3%)
Normal	203(54.7%)
LDL-C status	
Normal (<130mg/dl)	225(60.6%)
High (>130mg/dl)	146(39.4%)
Dyslipidaemia	
No	130(35%)
Yes	241(65%)

4.6: Factors associated with obesity among participants.

Tables 4.6a & b show the association between obesity and selected characteristics of the participants. Gender, age, marital status, first degree relatives of diabetics and cadre were factors significantly associated with obesity. A higher proportion of women 119 (36.8%) compared to men 9 (18.8%) were obese. Women were 2.5 times more likely to be obese than men. Crude OR, CI= 1.2-5.4, p=0.014.

A significantly higher proportion of participants who were over 40 years old 84 (41%) were at more risk of developing obesity compared to those under 40 years old 44 (26.5%). Subjects who were over 40 years old were 1.9 times more likely to be obese when compared those under 40 years old. Crude OR, CI= 1.2-3.0, p= 0.004.

A higher proportion of subjects who were married 125 (35.7%) were more obese compared to subjects who were not married 3 (14.3%). Married subjects were 3.33 times more likely to be obese than subjects who were not married, Crude OR, CI= 1.0-11.5, p = 0.045.

Among subjects who were first degree relatives of diabetics 35 (47.9%) were obese, this proportion was higher than those who were not first degree relatives of diabetics 91 (30.8%). Participants who were first degree relatives of diabetics were 2.1 times more likely to be obese than non-obese, Crude OR, CI= 1.2-3.4, p = 0.006.

A higher proportion of participants in higher cadre were obese 87 (39.9%) compared with subjects in the lower cadre 41 (27%). Individuals in the higher cadre were 6.6 times more likely to develop obesity than those in the lower cadre, Crude OR, CI= 1.1-2.8. p = 0.010

Relating tribe to obesity a higher proportion of subjects who were not Yoruba 23 (41.8%) compared to Yoruba 105 (33.2%) though this was not statistically significant, p= 0.216.

With respect to religion a higher proportion of Muslims 12 (36.4%) were obese compared to Christians 116 (34.3%) however this association was not statistically significant, (p= 0.814).

Educational status (p = 0.994), tobacco use (p = 1.000), alcohol use p= 0.254, physical stress (p = 0.115), physical activity (p = 0.921), sports activity (p = 1.000), sedentary lifestyle (0.833) and family history of diabetes (p = 0.175) did not show significant association with obesity.

4.7: Independent factors associated with obesity

Table 4.7 shows the results of multiple logistic regression of the association between obesity and participants characteristics significant at bivariate analysis. The analysis showed that being a first degree relative of diabetics was an independent risk factor for obesity (AOR CI. 1.0-4.8)

Table 4.7: Multivariate analysis of factors associated with obesity

Factors	AOR	95% CI	P-value
Gender			
Female	2.083	0.953-4.553	0.066
Male	1	Ref.	
Age			
≥40 (Ref)	1.499	0.849- 2.647	0.163
<40	1	Ref.	
Marital status			
Married (Ref)	2.101	0.565-7.816	0.268
Not married	1	Ref.	
First Degree			
Relative of diabetic			
Yes	2.144	1.253- 3.671	0.005*
No	1	Ref.	
Cadre			
Principal to	1.276	0.714-2.281	0.410
Assistant Directors	1	Ref.	
Basic to Senior	1	Ref.	

*= statistically significant, AOR= Adjusted odds ratio, Ref. = Reference

4.8: Factors associated with abdominal obesity among participants.

Tables 4.8a & b show the association between abdominal obesity and selected participants characteristics. Gender, age, marital status and cadre of staff were factors found to be associated with abdominal obesity. Tribe, religion, tobacco use, alcohol consumption, physical stress, physical activity sports activity, family history of diabetes and first degree relatives of diabetes were not significantly associated with abdominal obesity.

A higher proportion of women 252 (78.0%) compared to men 11 (22.9%) had abdominal obesity, $p < 0.001$. Women were approximately 12 times more likely to be obese than men. Crude OR, CI= 1.1- 5.4,

Subjects who were over 40 years old, 163 (79.5%), were at more risk of developing abdominal obesity compared to those under 40 years old 100 (60.2%), $p < 0.001$. Subjects who were over 40 years old were 2.6 times more likely to have abdominal obese when compared those under 40 years old, Crude OR, CI=. 1.6-4.1

Relating to marital status, a higher proportion of those married 259 (74%) had abdominal obesity in comparison to those who were not married 4 (19%), $p < 0.001$. Married subjects were 12 times more likely to be obese than subjects who were not married, Crude OR, CI= 4.0-36.9.

A higher proportion of staff in the higher cadre 172 (78.9%) had abdominal obesity when compared with those with lower cadre 91 (59.9%), $p<0.001$. Participants of high cadre were 2.5 times more likely to have abdominal obesity compared with those who are not, Crude OR, CI= 1.6-4.0

Relating tribe to abdominal obesity a higher proportion of participants who were not Yoruba 41(74.5%) compared to those who were Yoruba 222 (70.3%) had abdominal obesity, however this was not statistically significant, , $p= 0.518$. With respect to religion a higher proportion of Christians 242 (71.6%) compared to Muslims 21 (63.6%) had abdominal obesity but this association was not significant, ($p= 0.337$). Tobacco use ($p = 0.291$), alcohol consumption ($p = 0.128$) physical stress ($p = 0.670$), physical activity ($p = 0.664$), sports activity ($p = 0.464$), family history of diabetes ($p = 0.305$), being a first degree relative of diabetics ($p = 0.119$) and educational status ($p = 0.776$) did not have significant association with obesity

Table 4.8a: Association between abdominal obesity and socio-demographic characteristics

Characteristics	Abdominal obesity		Crude OR	p-value	95% CI
	Yes	No			
Gender					
Female	252(78%)	71(22%)	11.939	<0.001*	5.795-24.595
Male	11(22.9%)	37(77.1%)			
Age					
≥40	163(79.5%)	42(20.5%)	2.561	<0.001*	1.617-4.058
<40	100(60.2%)	66(39.8%)			
Tribe					
Others	41(74.5%)	14(25.5%)	1.240	0.518	0.645-2.382
Yoruba	222(70.3)	94(29.7%)			
Marital status					
Married	259(74%)	91(26%)	12.096	<0.001*	3.966-36.891
Not married	4(19%)	17(81%)			
Religion					
Christian	242(71.6%)	96(28.4%)	1.440	0.337	0.682-3.042
Muslim	21(63.6%)	12(36.4%)			
Education					
Graduate	227(71.2%)	92(28.8%)	1.097	0.776	0.580-2.073
Postgraduate	36(69.2%)	16(30.8%)			

*= statistically significant association

Table 4.8b: Association between abdominal obesity and lifestyle factors

Lifestyle factors	Abdominal Obesity		Crude OR	p-value	95% CI
	Yes	No			
Tobacco Use					
Yes	0(0%)	1(100%)	-	0.291*	-
No	263(71.1%)	107(28.9%)			
Alcohol consumption					
Drinker	7(50%)	7(50%)	0.395	0.128*	0.135-1.153
Non-drinker	256(71.7%)	101(28.3%)			
Physical Stress					
Yes	116(69.5%)	51(30.5%)	0.907	0.670	0.578-1.422
No	143(71.5%)	57(28.5%)			
Physical activity					
Yes	14(66.7%)	7(33.3%)	0.813	0.664	0.319-2.074
No	246(71.1%)	100(28.9%)			
Sports activity					
Yes	36(66.7%)	18(33.3%)	0.795	0.464	0.429-1.472
No	224(71.6%)	89(28.4%)			
Sedentary					
Yes	37(27.2%)	69(30.3%)	0.861	0.535	0.537-1.380
No	99(72.8%)	159(69.7%)			
Family history of diabetes					
Yes	61(75.3%)	20(24.7%)	1.342	0.305	0.764-2.358
No	200(69.4%)	88(30.6%)			
First degree relatives of diabetes					
Yes	57(78.1%)	16(21.9%)	1.615	0.119	0.880-2.962
No	203(68.8%)	92(31.2%)			
Cadre					
Principal 10	172(78.9%)	46(21.1%)	2.506	<0.001**	1.583-3.968
Assistant Director					
Basic and Senior officers	91(59.9%)	61(40.1%)			

*= Fisher's exact test, **= statistically significant association, - = not valid

4.9: Independent factors associated with abdominal obesity

Table 4.9 shows the results of multivariate analysis of independent factors associated with abdominal obesity. Gender (AOR, CI = 5.3-24.2) and marital status (AOR, CI= 1.9-21.5) remained significant after multiple logistic regression

Table 4.9: Multivariate analysis of factors associated with abdominal obesity.

Factors	AOR	95% CI	P-value
Gender			
Female	11.307	5.289-24.174	<0.001*
Male	1	Ref.	
Age			
≥40 (Ref)	1.859	0.976-3.539	0.059
<40	1	Ref.	
Marital status			
Married (Ref)	6.392	1.899-21.518	0.003*
Not married	1	Ref.	
Cadre			
Principal to Assistant Directors	1.424	0.748- 2.709	0.289
Basic to Senior	1	Ref.	

*= statistically significant association, Ref.= Reference, AOR= Adjusted Odds Ratio

4.10: Factors associated with impaired fasting glucose among participants

Table 4.10a & b show the association between impaired fasting glucose (IFG) participants' characteristics. Age, obesity and abdominal obesity showed significant associations with diabetes. There were no significant associations between IFG and other characteristics [gender, age, tribe, marital status, religion, tobacco use, alcohol consumption, physical stress, engaged in sports activity, presence of hypertension, presence of prehypertension, family history of diabetes, education and cadre of staff], ($p>0.05$) .

A higher proportion of participants who were 40 years old or higher 15 (7.3%) had IFG compared to those who were below 40 years old 4 (2.4%), $p = 0.033$. Participants who were 40 years old and over were 3.2 times more likely to have IFG compared to those who were not Crude OR, CI= 1.0-9.8. Similarly with respect to obesity a significant proportion of obese subjects 11 (8.6%) had IFG as compared to those who were not obese 8 (3.3%) $p, = 0.028$. Obese subjects were 2.8 times more likely to have IFG than non-obese, Crude OR, CI = 1.0-7.0. Approximately 18 (6.8%) of participants who had abdominal obesity had IFG compared to one (0.9%) who did not, $p = 0.019$. Participants who were abdominally obese were 7.8 times more likely to have IFG compared with those with normal waist, Crude OR, CI= 1.0-59.6

Table 4.10a: Association between impaired fasting glucose and socio-demographic characteristics of participants

Characteristics	Impaired fasting glucose		OR	p-value	95% CI
	Yes	No			
Sex					
Male	2(4.2%)	46(95.8%)	0.783	1.000*	0.175-3.499
Female	17(5.3%)	306(94.7%)			
Age					
≥40	15(7.3%)	190(92.7%)	3.197	0.033**	1.040-9.826
<40	4(2.4%)	162(97.6%)			
Tribe					
Yoruba	19(6%)	297(94%)	-	0.090*	-
Others	0(0%)	55(100%)			
Obesity					
Yes	11(8.6%)	117(91.4%)	2.762	0.028**	1.082-7.051
No	8(3.3%)	235(96.7%)			
Abdominal obesity					
Yes	18(6.8%)	245(93.2%)	7.861	0.019**	1.036-59.643
No	1(0.9%)	107(99.1%)			
Marital status					
Married	19(5.4%)	331(94.6%)	-	0.614*	-
Not married	0(0%)	21(100%)			
Religion					
Christian	19(5.6%)	319(94.4%)	-	0.396	-
Muslim	0(0%)	33(100%)			
Education					
Graduate	16(5%)	303(95%)	0.862	0.738*	0.242-3.070
Postgraduate	3(5.8%)	49(94.2%)			

* = Fishers exact test, ** = statistically significant association, - = not valid

Table 4.10b: Association between impaired fasting glucose and lifestyle factors of participants

Lifestyle factors	Impaired fasting glucose		Crude OR	p-value	95% CI	Lifestyle factors
	Yes	No				
Tobacco use						
Yes	0(0%)	1(100%)	0.054	1.000*	-	-
No	19(5.1%)	351(94.9%)				
Alcohol consumption						
Drinker	1(7.1%)	13(92.9%)	-	0.527*	1.449	0.179-11.695
Non-drinker	18(5.0 %)	339(95%)				
Physical Stress						
Yes	8(4.8%)	159(95.2%)	0.093	0.760	0.864	0.339-2.202
No	11(5.5%)	189(94.5%)				
Physical activity						
Yes	1(4.8%)	20(95.2%)	-	1.000*	0.968	0.123-7.643
No	17(4.9%)	329(95.1%)				
Engaged in sports activity						
Yes	4(7.4%)	50(92.6 %%)	0.423	0.501	1.589	0.507-4.984
No	15(4.8%)	298(95.2%)				
Sedentary						
Yes	10(4.4%)	218(95.6%)	0.858	0.354	0.647	0.256-1.635
No	9(6.6%)	127(93.4%)				
Presence of hypertension						
Yes	3(7.1%)	39(92.9%)	-	0.463*	1.505	0.420-5.397
No	16(4.9%)	313(95.1%)				
Presence of prehypertension						
Yes	3(3.4%)	84(96.6%)	-	0.581*	0.598	0.170-2.103
No	16(5.6%)	268(94.4%)				
Family history of hypertension						
Yes	12(6.1%)	184(93.9%)	0.857	0.480	1.565	0.602-4.069
No	7(4%)	168(96%)				
Family history of diabetes						
Yes	5(6.2%)	76(93.8%)	-	0.580*	1.29	0.450-3.688
No	14(4.9%)	274(95.1%)				
First degree relatives of diabetes						
Yes	5(6.8%)	68(93.2%)	-	0.553*	1.476	0.514-4.238
No	14(4.7%)	281(95.3%)				
Gender						
Principal to Assistant Director	14(6.4%)	204(93.6%)	1.804	0.179	2.018	0.711-5.725
Basic and Senior officers	5(3.1%)	147(96.7%)				

* = Fishers exact test, - = not valid

4.11: Independent factors associated with IFG

Table 4.11 shows the results of multivariate analysis of independent factors for IFG. None of the associated factors was found to be independently associated with IFG when those significant at bivariate analysis were put into regression model.

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Table 4.11: Multivariate analysis of factors associated with IFG

Risk factors	AOR	95% CI	P-value
Age			
≥40 (Ref)	2.808	0.897-8.790	0.076
<40	1	Ref.	
Tribe			
Others	0.000	0.000	0.997
Yoruba	1	Ref.	
BMI			
Obese	1.836	0.680-4.959	0.231
Not Obese	1	Ref.	
Waist Circumference			
High	4.741	0.565- 39.800	0.152
Normal	1	Ref.	

AOR= Adjusted Odds Ratio, Ref. = Reference

4.12: Factors associated with diabetes among participants

Table 4.12 a & b depict the association between diabetes and participants' characteristics. Significant associations were found between age, physical stress, presence of hypertension, family history of diabetes and first degree relative history of diabetes and diabetes ($p<0.05$). On the other hand no significant associations was found between diabetes and the remaining characteristics of the participants [gender, ($p= 0.784$), tribe ($p= 0.219$), obesity ($p= 0.419$), religion ($p = 0.430$), marital status ($p= 1.000$), current tobacco use (1.000), alcohol ($p= 0.207$), engaged in sports activity (1.000), history of prehypertension ($p= 0.156$), education ($p= 0.2$) and cadre of job, $p=1.000$]

A higher proportion of subjects who were 40 years old and above 6 (2.9%) were diabetic compared with those under 40 years old 0 (0%). $p = 0.035$. Relating physical stress to diabetes, 6 (3%) of subjects who experienced physical stress had diabetes however none of the subjects who did not experience physical stress 0 (0%) had diabetes, $p = 0.034$. Similarly, a higher proportion of subjects found to be hypertensive 5 (11.9%) had diabetes in comparison to one (0.3%), who did not have hypertension, $p< 0.001$. Among subjects who had family history of diabetes 6 (7.4%) turned out to be diabetic whereas none of those without family history 0 (0%) had diabetes, $p< 0.001$. Out of those who were first degree relatives of diabetics 6 (8.2%) had diabetes but none 0 (%) of those who were not first degree relatives had diabetes, $p< 0.001$.

Table 4.12a: Association between diabetes and socio-demographic characteristics of participants

Characteristics	Diabetes		Crude OR	p-value	95% CI
	Yes	No			
Gender					
Male	1(2.1%)	47(97.9%)	1.353	0.784*	0.155-11.837
Female	5(1.5%)	318(98.5%)			
Age					
≥40	6(2.9%)	199(97.1%)		0.035*	
<40	0(0%)	166(100%)			
Tribe					
Others	2(3.6%)	53(96.4%)	2.943	0.219*	0.526-16.473
Yoruba	4(1.3%)	312(98.7%)			
Obesity					
Yes	3(2.3%)	125(97.7%)	1.920	0.419*	0.382-9.652
No	3(1.2%)	240(98.8%)			
Abdominal obesity				0.187*	
Yes	6(2.3%)	257(97.7%)			
No	0(0%)	108(100%)			
Marital status					
Married	6(1.7%)	21(100)		1.000*	
Single	0(0%)	344(98.3%)			
Religion					
Islam	1(3%)	32(97%)	2.081	0.430*	0.236-18.365
Christianity	5(1.5%)	333(98.5%)			
Education					
Graduate	4(1.3%)	315(98.7%)	0.317	0.200*	0.057-1.779
Postgraduate	2(3.8%)	50(96.2%)			

* = Fishers exact test, Bold= statistically significant association, - = not valid

Table 4.12b: Association between diabetes and lifestyle factors of participants

Lifestyle factors	Yes	No	Crude OR	p-value	95% CI	Lifestyle factors
Tobacco Use						
Yes	0(0%)	1(100%)	-	1.000*	-	
No	6(1.6%)	364(98.4%)				
Alcohol Consumption						
Drinker	1(7.1%)	13(92.9%)	-	0.207*	5.415	0.59-49.724
Non-drinker	5(1.4%)	352(98.6%)				
Physical Stress						
Yes	6(3%)	194(97%)	-	0.034*	-	
Yes	0(0%)	167(100%)				
Physical activity						
Yes	1(4.8%)	20(95.2%)	-	0.300*	3.410	0.380-30.587
No	5(1.4%)	341(98.6%)				
Sports activity						
Yes	1(1.9%)	53(98.1%)	-	1.000*	1.162	0.133-10.146
No	5(1.6%)	308(98.4%)				
Sedentary						
Yes	4(1.8%)	224(98.5%)	-	1.000*	1.196	0.216-6.620
No	2(1.5%)	134(98.5%)				
Presence of Hypertension						
Yes	5(11.9%)	37(88.1%)	-	0.000*	44.32	5.042-389.672
No	1(0.3%)	318(99.7%)				
Presence of Prehypertension						
Yes	1(1.1%)	86(98.9%)	-	1.000*	0.649	0.075-5.630
No	5(1.8%)	279(98.2%)				
Family history of diabetes						
Yes	6(7.4%)	75(92.6%)	-	0.000*	-	
No	0(0%)	288(100%)				
First relative degree history of diabetes						
Yes	6(8.2%)	67(91.8%)	-	0.000*	-	
No	0(0%)	295(100%)				
Cadre						
Principal to Assistant Director	4(1.8%)	214(98.2%)	-	1.000	1.402	0.254-7.752
Basic and Senior officers	2(1.3%)	150(98.7%)				

*= Fishers exact test, Bold= statistically significant association, DM= diabetes, - = not valid

4.13: Independent factors associated with diabetes

Table 4.13 shows the results of multivariate analysis of factors associated with diabetes in bivariate analysis. after adjusting for confounding only hypertension was observed to be an independent risk factor of diabetes among the studied population.

Table 4.13: Multivariate analysis of factors associated with diabetes

Factors	AOR	95% CI	P-value
Age			
≥40 (Ref)	2.083	0.000	0.995
<40	1	Ref.	
Physical Stress			
Yes	0.000	0.000	0.994
No	1	Ref.	
Presence of hypertension			
Yes	18.333	1.508-222.875	0.022*
No	1	Ref.	
Family history of diabetes			
Yes	4.355	0.000	1.000
No	1	Ref.	
First Degree			
Relative of a diabetic			
Yes	3.784	0.000	0.996
No	1	Ref.	

AOR= Adjusted Odds Ratio, Ref.= Reference, *= statistically significant

4.14: Factors associated with hypercholesterolaemia among participants

Table 4.14 a & b show the association between hypercholesterolaemia and participants characteristics. Age, obesity, abdominal obesity, IFG, alcohol consumption, presence of hypertension and cadre of staff were observed to be associated with hypercholesterolaemia.

A greater proportion of subjects who were 40 years old and over 86 (42%) had hypercholesterolaemia compared to those who were under 40 years old 37 (22.3%), $p < 0.001$. Individuals who were 40 years and older were 2.5 times more likely to have hypercholesterolaemia than those under 40 years of age, AOR, CI = 1.6-4.0. A higher percentage of the obese 53 (41.4%) had hypercholesterolaemia as compared to non-obese 70 (28.8%), $p < 0.001$. Obese participants were 1.75 times more likely to have hypercholesterolaemia as compared to non-obese, AOR, CI = 1.1-2.7. A significant higher proportion of participants who had abdominal obesity 99 (37.6%) had hypercholesterolaemia compared to those who had normal waist circumference 24 (22.2%), $p = 0.004$. The abdominally obese were 2.1 times more likely to have hypercholesterolaemia compared those who did not have abdominal obesity, AOR, CI = 1.3-3.5. A higher proportion of participants who had IFG 11 (57.9%) had hypercholesterolaemia compared to those who did not have IFG 11 (31.8 %), $p = 0.0019$. Participants who had IFG were 2.9 times more likely to have hypercholesterolaemia than those with normal plasma glucose values, AOR, CI = 1.153-7.527. A higher proportion of participants who consumed alcohol 106 (31.7%) had hypercholesterolaemia than those who did not consume alcohol 64 (18.3%). A higher proportion of participants who had hypertension had 23 (54.8%) hypercholesterolaemia compared to those who did not have hypertension 100 (30.4%), $p = 0.002$. Hypertensives were 2.7 times more likely to have hypercholesterolaemia than non-hypertensives, AORCI, 1.445-5.318. Relating hypercholesterolaemia with cadre of staff a higher proportion of subjects 91 (41.7%) in the higher cadre had hypercholesterolaemia as compared to those in the lower cadre 32 (21.1%), $p < 0.001$.

No association was found between hypercholesterolaemia and characteristics such as gender ($p = 0.764$), tribe ($p = 0.702$), marital status ($p = 0.349$), diabetes ($p = 0.377$), religion ($p = 0.236$), education ($p = 0.130$), tobacco use ($p = 0.332$), physical stress ($p = 0.326$), physical activity ($p = 0.640$), sports activity ($p = 0.603$), presence of prehypertension ($p = 0.109$) and family history of diabetes ($p = 0.835$).

Table 4.14a: Association between hypercholesterolaemia and socio-demographic characteristics

Characteristics	HCE		Crude OR	p-value	95% CI
	Yes	No			
Gender					
Female	108(33.4%)	215(66.6%)	1.105	0.764	0.575-2.122
Male	15(31.2%)	33(68.8%)			
Age					
≥40	86(42.0%)	119(58%)	2.520	<0.0001	1.592-3.987
<40	37(22.3%)	129(77.7%)			
Tribe					
Others	17(30.9%)	38(69.1%)	0.886	0.702	0.478-1.664
Yoruba	106(33.5%)	210(66.5%)			
Marital status					
Married	118(33.7%)	232(66.3%)	1.628	0.349	0.582-4.551
Not married	5(23.8%)	16(76.2%)			
Obesity					
Yes	53(41.4%)	75(58.6%)	1.746	0.014	1.116-2.734
No	70(28.8%)	173(71.2%)			
Abdominal obesity					
Yes	99(37.6%)	164(62.4%)	2.113	0.004	1.259-3.546
No	24(22.2%)	84(77.8%)			
Diabetes					
Yes	3(50%)	3(50%)	2.042	0.403*	0.406-10.267
No	120(32.9%)	245(67.1%)			
IFG					
Yes	11(57.9%)	8(42.1%)	2.946	0.019	1.153-7.527
No	112(31.8%)	240(68.2%)			
Religion					
Muslim	14(42.4%)	19(57.6%)	1.548	0.236	0.748-3.203
Christian	109(32.2%)	229(67.8%)			
Education					
Graduate	101(31.7%)	218(69.3%)	0.632	0.130	0.347-1.150
Postgraduate	22(42.3%)	30(57.7%)			

HCE= Hypercholesterolaemia, * = Fisher's exact test, Bold= statistically significant

Table 4.14b: Association between hypercholesterolaemia and lifestyle factors

Lifestyle factors	Yes	No	Crude OR	p-value	95% CI
Tobacco Use					
Yes	1(100%)	0(0%)	-	0.332	-
No	122(33%)	248(67%)			
Alcohol consumption					
Drinker	9(64.3%)	5(35.7%)	1.828	0.018	0.920-3.632
Non-drinker	106(31.7%)	228(68.3%)			
Physical Stress					
Yes	59(35.3%)	108(64.7%)	1.245	0.326	0.804-1.928
No	61(30.5%)	139(69.5%)			
Engaged in Physical activity					
Yes	6(28.6%)	15(71.4%)	0.793	0.640	0.300-2.098
No	116(33.5%)	230(66.5%)			
Engaged Sports activity					
Yes	16(29.6%)	38(70.4%)	0.846	0.603	0.451-1.588
No	104(33.2%)	209(66.8%)			
Sedentary					
Yes	77(33.8%)	151(66.2%)	1.031	0.894	0.657-1.618
No	45(33.1%)	91(66.9%)			
Presence of Hypertension					
Yes	23(54.8%)	19(45.2%)	2.772	0.002	1.445-5.318
No	100(30.4%)	229(69.6%)			
Presence of Prehypertension					
Yes	35(40.2%)	52(59.8%)	1.499	0.109	0.912-2.464
No	88(31%)	196(69%)			
Family history of diabetes					
Yes	26(32.1%)	55(67.9%)	0.945	0.835	0.558-1.601
No	96(33.3%)	192(66.7%)			
Family history of hypertension					
Yes	70(35.7%)	126(64.3%)	1.279	0.268	0.828-1.976
No	53(30.3%)	122(69.7%)			
Cadre					
Principal to Assistant Director	91(41.7%)	127(58.3%)	2.687	P<0.001	1.673-4.316
Basic and Senior officers	32(21.1%)	120(78.9%)			
HCE - Hypercholesterolaemia, * = Fisher's exact test, Bold= statistically significant					

4.15: Independent factors associated with hypercholesterolaemia

Table 4.15 shows the multivariate analysis of factors that were found to be significant in bivariate analysis. After multiple regression alcohol consumption and cadre were found to be the independent predictors of hypercholesterolaemia.

Table 4.15: Multivariate analysis of factors associated with hypercholesterolaemia

Risk factors	AOR	95% CI	P-value
Age			
>40 (Ref)	1.448	0.805-2.604	0.216
<40	1	Ref.	
BMI			
Obese	1.180	0.697-2.000	0.538
Not obese	1	Ref.	
Waist circumference			
High	1.544	0.829-2.876	0.171
Normal	1	Ref.	
IFG			
Yes	2.243	0.831-6.053	0.111
No	1	Ref.	
Alcohol consumption			
Yes	5.608	1.633-19.268	0.006*
No	1	Ref.	
Hypertension			
Yes	1.933	0.968-3.859	0.062
No	1	Ref.	
Cadre			
Yes	2.060	1.152-3.865	0.019
No	1	Ref.	

*= statistically significant, AOR= Adjusted odds ratio, Ref.= Reference

Table 4.16a: Association between triglycerides and socio-demographic characteristics

HTG					
Characteristics	Yes	No	OR	p-value	95%CI
Gender					
Female	7(2.2%)	316(97.8%)	0.332	0.127*	0.083-1.332
Male	3(6.2%)	45(93.8%)			
Age					
≥40	9(4.4%)	196(95.6%)	7.577	0.027*	0.950-60.424
<40	1(0.6%)	165(99.4%)			
Tribe					
Others	0(0%)	55(100)	-	0.181	
Yoruba	10(3.2%)	306(96.8%)			
Marital status					
Married	10(2.9%)	340(97.1%)	-	1.000	-
Not married	0(0%)	21(100%)			
Obesity					
Yes	4(3.1%)	124(96.9%)	1.274	0.711	0.353-4.600
No	6(2.5%)	237(97.5%)			
Abdominal obesity					
Yes	9(3.4%)	254(96.6%)	3.791	0.292	0.474-30.296
No	1(0.9%)	107(99.1%)			
Diabetes					
Yes	0(0%)	6(100%)	-	1.000	-
No	10(2.7%)	355(97.3%)			
IFG					
Yes	2(10.5%)	17(89.5%)	5.059	0.088	0.997-25.670
No	8(2.3%)	344(97.7%)			
Religion					
Muslim	1(3%)	32(97%)	1.142	1.000	0.140-9.307
Christian	9(2.7%)	329(97.3%)			
Education					
Graduate	8(2.5%)	311(97.5%)	0.643	0.581	0.133-3.116
Postgraduate	2(3.8%)	50(96.2%)			

HTG = Hypertriglyceridaemia, * = statistically significant

Table 4.16b: Association between triglycerides and lifestyle factors

Lifestyle factors	HTG Yes	HTG No	Crude OR	p-value	95% CI
Tobacco Use					
Yes	10(2.7%)	360(97.3%)	-	1.000	-
No	0(0%)	1(100%)	-	-	-
Alcohol Consumption					
Drinker	0(0%)	14(100%)	-	1.000	-
Non-drinker	10(2.8%)	347(97.2%)	-	-	-
Physical Stress					
Yes	4(2%)	196(98%)	1.512	0.737	0.400-5.725
No	5(3%)	162(97%)	-	-	-
Physical activity					
Yes	1(4.8%)	20(95.2%)	1.872	0.449	0.226-15.515
No	9(2.6%)	337(97.4%)	-	-	-
Sports activity					
Yes	2(3.7%)	52(96.3%)	1.681	0.626	0.340-8.317
No	79(2.2%)	306(97.8%)	-	-	-
Sedentary					
Yes	6(2.6%)	222(97.4%)	1.811	0.715	0.360-9.101
No	2(1.5%)	134(98.5%)	-	-	-
Presence of hypertension					
Yes	0(0%)	42(100%)	-	0.611	-
No	10(3.0%)	319(97%)	-	-	-
Presence of prehypertension					
Yes	5(5.7%)	82(94.3%)	3.402	0.059	0.961-12.041
No	5(1.8%)	279(98.2%)	-	-	-
Family history of diabetes					
Yes	1(1.2%)	80(98.8%)	0.502	1.000	0.061-4.139
No	7(2.4%)	281(97.6%)	-	-	-
Family history of hypertension					
Yes	2(1%)	194(99%)	0.215	0.051	0.045-1.027
No	8(4.6%)	167(95.4%)	-	-	-
Cadre					
Principal	9(4.1%)	209(95.9%)	6.502	0.052*	0.815-51.870
Assistant Director	1(0.7%)	151(99.3%)	-	-	-
Basic and Senior officers					

HTG= Hypertriglyceridemia, Bold = statistically significant, * = Fisher's exact test, - = not valid.

Table 4.17: Multivariate analysis of factors associated with plasma triglycerides

Risk factors	AOR	95% CI	P-value
Age			
≥40 (Ref)	3.112	0.296-32.709	0.344
<40	1	Ref.	
IFG			
Yes	4.817	0.814-28.496	0.083
No	1	Ref.	
Family history of hypertension			
Yes	0.177	0.036-0.876	0.034*
No	1	Ref.	
Prehypertension			
Yes	2.649	0.696-10.085	0.153
No	1	Ref.	
Cadre			
Yes	3.324	0.324-34.133	0.312
No	1	Ref.	

*= statistically significant, AOR= Adjusted odds ratio, Ref.= reference

Table 4.18a: Association between plasma LDLC values and socio-demographic characteristics

Characteristics	LDLC		χ^2	p-value	OR	95% CI
	High	Normal				
Gender						
Female	126(39%)	197(61%)	0.124	0.753	0.895	0.484-1.658
Male	20(41.7%)	28(58.3%)				
Age						
≥ 40	95(46.3%)	110(53.7%)	9.375	0.002*	1.947	1.268-2.991
<40	51(30.7%)	115(69.3%)				
Tribe						
Others	21(38.2%)	34(61.8%)	0.037	0.847	0.944	0.524-1.701
Yoruba	125(39.6%)	191(60.4%)				
Marital status						
Married	139(39.7%)	211(60.3%)	0.338	0.561	1.318	0.519-3.347
Not married	7(33.3%)	14(66.7%)				
Obesity						
Yes	66(51.6%)	62(48.4%)	12.20	0.000*	2.169	1.400-3.361
No	80(32.9%)	163(67.1%)	6			
Abdominal obesity						
Yes	115(43.7%)	148(56.3%)	7.239	0.007*	1.930	1.191-3.128
No	31(28.7%)	77(71.3%)				
Diabetes						
Yes	3(5.0%)	3(50%)	0.290	0.590	1.552	0.309-7.798
No	143(39.2%)	222(60.8%)				
IFG						
Yes	12(63.2%)	7(36.8%)	4.755	0.029*	2.789	1.071-7.259
No	134(38.1%)	218(61.9%)				
Religion						
Muslim	15(45.5%)	18(54.5%)	0.565	0.452	1.317	0.641-2.703
Christian	131(38.8%)	207(61.2%)				
Education						
Graduate	124(38.9%)	195(61.1%)	0.221	0.638		0.479-1.571
Postgraduate	22(42.3%)	30(57.7%)				

* = statistically significant

Table 4.18b: Association between plasma LDLC values and lifestyle factors

Lifestyle factors	LDL				p-value	95% CI
	Normal	High	Crude OR			
Tobacco Use						
Yes	0(0%)	1(100%)	-	0.394	-	-
No	225(60.8%)	145(39.2%)				
Alcohol consumption						
Drinker	4(28.6%)	10(71.4%)	6.272	0.012*	1.250-13.208	
Non-drinker	221(61.9%)	136(38.1%)				
Physical Stress						
Yes	100(59.9%)	67(40.1%)	0.172	0.678	0.718-1.665	
No	124(62%)	76(38%)				
Engaged in physical activity						
Yes	14(66.7%)	7(33.3%)	0.326	0.568	0.300-1.938	
No	209(60.4%)	137(39.6%)				
Engaged in sports activity						
Yes	35(64.8%)	19(35.2%)	0.380	0.537	0.453-1.512	
No	189(60.4%)	124(39.6%)				
Sedentary						
Yes	137(60.1%)	91(39.9%)	0.032	0.859	0.673-1.607	
No	89(61%)	53(39%)				
Presence of Hypertension						
Yes	18(42.9%)	24(57.1%)	6.280	0.012*	1.180-4.337	
No	207(62.9%)	122(37.1%)				
History of Prehypertension						
Yes	48(55.2%)	39(44.8%)	1.427	0.232	0.827-2.185	
No	177(62.3%)	107(37.7%)				
Family history of diabetes						
Yes	50(61.7%)	31(38.3%)	0.046	0.831	0.570-1.570	
No	174(60.4%)	114(39.6%)				
Family history of hypertension						
Yes	118(60.2%)	78(39.8%)	0.034	0.853	0.685-1.579	
No	107(61.1%)	68(38.9%)				
Cadre						
Principal to Assistant Director	117(53.7%)	101(46.3%)	10.487	0.001*	1.324-3.182	
Basic and Senior officers	107(70.4%)	45(29.6%)				

* = statistically significant

Table 4.19: Multivariate analysis of factors associated with plasma LDLC

Risk factors	AOR	95% CI	P-value
Age			
≥40 (Ref)	1.237	0.706-2.168	0.458
<40	1	Ref.	
BMI			
Obese	1.678	1.008-2.794	0.047*
Not obese	1	Ref.	
Waist circumference			
High	1.261	0.706-2.253	0.433
Normal	1	Ref.	
IFG			
Yes	2.128	0.786-5.765	0.137
No	1	Ref.	
Alcohol consumption			
Yes	4.894	1.380-17.355	0.014*
No	1	Ref.	
Hypertension			
Yes	1.692	0.848-3.380	0.136
No	1	Ref.	
Cadre			
Yes	1.676	0.949-2.957	0.075
No	1	Ref.	

*= statistically significant, AOR= Adjusted odds ratio, Ref.= reference

4.20: Factors associated with low plasma HDLC among participants

Table 4.20 a & b shows the association between low HDLC and participants characteristics. Marital status, obesity, abdominal obesity, being engaged in physical activity, sports activity and sedentary lifestyle were found to be associated factors associated with the presence of low LDLC.

A higher proportion of married women had low HDLC163 (46.6%) compared to those who were not married 5 (23.8%), $p = 0.042$. A higher proportion of obese participants had low HDLC 73 (57%) compared to the non-obese 95 (39.1%), $p = 0.001$. Subjects with abdominal obesity 133 (50.6%) had a higher prevalence of low HDLC compared to those who had normal waist circumference 35 (32.4%), $p = 0.001$. The proportion of those with low HDLC was higher among those who were physically inactive 162 (46.8%) compared to those who were physically active 5 (23.8%), $p = 0.040$. A higher proportion of participants who did not engage in sports 149 (47.6%) had low HDLC compared to those who engaged in sports 17 (31.5%), $p = 0.028$.

On the other hand no association was found between low HDLC and factors such as gender ($p = 0.141$), age = 0.221, tribe ($p = 0.539$), diabetes($p = 1.000$), IFG (0.775) , religion ($p = 0.730$), education ($p = 0.892$), tobacco use ($p = 0.453$), alcohol consumption ($p = 0.145$), physical stress ($p = 0.292$), presence of hypertension ($p = 0.737$), presence of prehypertension ($p = 0.922$), family history of diabetes ($p = 0.160$) and cadre ($p = 0.116$).

Table 4.20a shows the association between low plasma HDLC and socio-demographic characteristics, N = 371

Characteristics	HDLC Normal	Low	Crude OR	p-value	95% CI
Gender					
Female	172(53.3%)	151(46.7%)	1.601	0.141	0.852-3.008
Male	31(64.6%)	17(35.4%)			
Age					
≥40	118(57.6%)	87(42.4%)	0.774	0.221	0.513-1.168
<40	85(51.2%)	81(48.8%)			
Tribe					
Other tribes	28(50.9%)	27(49.1%)	1.197	0.539	0.675-2.123
Yoruba	175(55.4%)	141(44.6%)			
Marital status					
Married	187(53.4%)	163(46.6%)	2.789	0.042	1.000-7.781
Not married	16(76.2%)	5(23.8%)			
Obesity					
Yes	55(43%)	73(57%)	2.068	0.001	1.339-3.193
No	148(60.9%)	95(39.1%)			
Abdominal obesity					
Yes	130(49.4%)	133(50.6%)	2.134	0.001	1.334-3.414
No	73(67.6%)	35(32.4%)			
Diabetes					
Yes	3(50%)	3(50%)	1.212	1.000*	0.241-6.085
No	200(54.8%)	165(45.2%)			
IFG					
Yes	11(57.9%)	8(42.1%)	0.873	0.775	0.343-2.222
No	192(54.5%)	160(45.5%)			
Religion					
Muslim	19(57.6%)	14(42.4%)	0.880	0.730	0.427-1.814
Christian	184(54.4%)	154(45.6%)			
Education					
Graduate	175(54.9%)	144(45.1%)	0.960	0.892	0.533-1.729
Post graduate	28(53.8%)	24(46.2%)			

*= Fisher's exact test, Bold= statistically significant

Table 4.20b shows the association between low plasma HDLC and lifestyle factors

Lifestyle factors	HDL Normal	Low	Crude OR	p-value	95% CI
Tobacco Use					
Yes	0(0%)	1(100%)	-	0.453	-
No	203(54.9%)	167(45.1%)			
Alcohol consumption					
Drinker	5(35.7%)	9(64.3%)	2.242	0.145	0.737-6.822
Non-drinker	198(55.5%)	159(44.5%)			
Physical Stress					
Yes	114(57%)	86(43%)	1.249	0.292	0.826-1.887
No	86(51.5%)	81(48.5%)			
Engaged in Physical activity					
Yes	16(76.2%)	5(23.8%)	0.355	0.040*	0.127-0.990
No	184(53.2%)	162(46.8%)			
Engaged in Sports activity					
Yes	37(68.5%)	17(31.5%)	0.506	0.028*	0.273-0.936
No	164(52.4%)	149(47.6%)			
Sedentary					
Yes	116(50.9%)	112(49.1%)	1.609	0.031*	1.043-2.482
No	85(62.5%)	51(37.5%)			
Presence of hypertension					
Yes	24(57.1%)	18(42.9%)	0.895	0.737	0.468-1.712
No	179(54.4%)	150(45.6%)			
Presence of Prehypertension					
Yes	48(55.2%)	39(44.8%)	0.976	0.922	0.602-1.582
No	155(54.6%)	129(45.4%)			
Family history of DM					
Yes	39(48.1%)	42(51.9%)	1.424	0.160	0.869-2.335
No	164(56.9%)	124(43.1%)			
Cadre					
Principal to Assistant Director	127(56.3%)	91(41.7%)	0.717	0.116	0.472-1.087
Basic and Senior officers	76(50%)	76(50%)			

* = statistically significant

4.21: Independent factors associated with low plasma HDLC

Table 4.21 shows multivariate analysis of risk factors for low HDLC. After multiple regression analysis, body mass index and sedentary time remain significantly associated with low HDLC.

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Table 4.21: Multivariate analysis of factors associated with low HDLC

Risk factors	AOR	95% CI	P-value
Age			
≥40 (Ref)	1.237	0.706-2.168	0.458
<40	1	Ref.	
BMI			
Obese	1.679	1.012-2.786	0.045*
Not obese	1	Ref.	
Waist circumference			
High	1.549	0.882-2.723	0.128
Normal	1	Ref.	
Marital status			
Married	2.472	0.833-7.339	0.103
Not married	1	Ref.	
Physical activity			
Yes	0.513	0.163-1.617	0.254
No	1	Ref.	
Sports			
Yes	0.630	0.313-1.269	0.196
No	1	Ref.	
Sedentary			
Yes	1.762	1.119-2.777	0.015*
No	1	Ref.	

* = statistically significant, AOR= Adjusted odds ratio, Ref. = Reference

4.22 Prevalence of multiple factors

Table 4.22 depicts the prevalence of multiple factors, 14.8% had none of the factors, 29.9% had 1 factor, 43.4% had 2 factors, 10.2% had 3 factors and 1.6% had all the 4 factors.

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Table 4.22: Prevalence of multiple factors

Number of CV factors	Frequency %
None	14.8
One	29.9
Two	43.4
Three	10.2
Four	1.6

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4.23: Clustering of factors among participants

Table 4.23 shows the pattern of clustering of multiple factors. The most common cluster of factors was obesity and dyslipidaemia (65%), followed by hyperglycaemia and dyslipidaemia (27%) and by obesity and hypertension (10%).

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Table 4.23: Clustering of factors among participants

Cluster of variables	% Yes No	
	Yes	No
Obesity and Hyperglycaemia	3.8	96.2
Hyperglycaemia and dyslipidaemia	5.7	94.3
Obesity and dyslipidaemia	65.0	35.0
Obesity and hypertension	10.0	90.0
Hypertension and hyperglycaemia	2.2	97.8
Hypertension and dyslipidaemia	8.6	91.4
Obesity, hyperglycaemia and dyslipidaemia	3.2	96.8
Obesity, dyslipidaemia and hypertension	7.5	92.5
Obesity, hyperglycaemia and hypertension	2.2	97.8
Hypertension, hyperglycaemia and dyslipidaemia	1.6	98.4
Dyslipidaemia, hypertension, obesity and hyperglycaemia	0.8	99.2

CHAPTER 5

DISCUSSION, CONCLUSION AND RECOMMENDATIONS

5.1: DISCUSSION

There are limited studies on prevalence of obesity, hyperglycaemia and dyslipidaemia among health workers in Nigeria and Africa. Similar results of higher prevalence of cardiovascular risk factors in health workers compared to that of the general population have been reported across the globe.

5.1.1: Prevalence of obesity and associated risk factors.

Obesity has been recognized as a major public health problem worldwide that requires preventive action (Al-Muraikhi, 2010). Nowadays, increased prevalence of obesity is noted among all age groups in both genders all over the world (Shanab and Al -Sabbah, 2011). Information on prevalence and associated risk factors is essential in its control.

Based on BMI classification of nutritional weight status, findings from this study indicated that the majority of the participants were overweight (43.1%), obese (34.8 %) and had abdominal obesity (70.9%). The obesity prevalence of this study is similar to the prevalence of obesity among hospital personnel in Littoral region in Cameroon (Tachang et.al., 2012) in which a prevalence of 38.4% was reported. It was however lower than the prevalence of obesity of 62.2% found among Nurses in Akwa Ibom (Ogunjimi et.al., 2011). Prevalence of obesity in this study was higher than that reported by the World Health Organization in its 2013 World Health Statistics (6.5%) for Nigeria. It was also higher than the worldwide prevalence of 10% (WHO, 2008). However, unlike this study which was based on an urban population, the WHO survey included both rural and urban populations. The comparatively higher prevalence of overweight and obesity in this study population to that of the general Nigerian population may be explained by the possibly higher socioeconomic status in the study population compared to the average Nigerian population or perhaps because the study population is largely female dominated, as these attributes have been known to be associated with overweight and obesity (Aryee et.al., 2013; Benkeser et al., 2012).

The prevalence of abdominal obesity in this study is comparable to the prevalence of 68.2% found among Nurses in South Africa (Goon et.al., 2014), higher than 47% found among senior civil servants in Asaba (Odenigbo et.al., 2012) but lower than a prevalence of 80% reported among middle and upper class individuals in Abia (Okasor et.al., 2014).

Differences in defining abdominal obesity, genetic factors, age, type of diet and extent of physical activity might contribute to variations (Goon et.al., 2014) comparable to those found in this study. The high prevalence of obesity among health workers of a tertiary care hospital who should serve as role models teaching about the health implications of excessive weight gain and fat deposit is of concern and might predispose them to risks of hypertension, diabetes mellitus, cardiovascular disease and stroke. This suggests serious lack of awareness or non-application of information regarding physical activity and diet (Sharma et. al., 2012). The finding in this study of obesity levels that can be compared to those of affluent countries, coupled with the rates at which such countries are experiencing the fall outs from this epidemic, may buttress the important point of Ogunjiuni *et al.*, (2010) that if corrective measures are not put in place our healthcare providers sooner than later would become care receivers. Again, if these healthcare providers do not respond to obesity interventions, it may be unrealistic to expect the general public to do so (Miller et.al., 2008).

The women in this study showed a higher prevalence of both general and abdominal obesity than the men. This is in agreement with the earlier studies carried out among health workers in Jos (Ojomu and Kuranga,2013) and South Africa (Skaal and Pengpid, 2011) but in contrast to the study of Palacios-Rodriguez et.al., 2006 among health workers in Mexico in which the male gender instead was strongly associated with obesity in health workers. Higher obesity in women as compared to men has been attributed to some reasons. It could be related to the influence of cultural factors (Agbary et.al., 2014). Female obesity is seen as a sign of wealth and beauty in some African cultural context. Similarly, the influence of behavioural and psychosocial factors has been demonstrated to explain the importance of obesity in women (Amoah, 2003b). Moreover, there are gender differences in carbohydrate metabolism that cause a greater increase in triglyceride levels in women (Knopp et. al., 2005). Therefore, the increased refined carbohydrate intake in developing countries may affect excess weight gain in women more than in men (Kanter and Caballero, 2012).

Age has frequently been reported as a prognostic factor contributing to the development of obesity in both genders (Hu et al., 2008). Several community-based cross sectional studies reported higher prevalence of obesity with increasing age (Sidik & Rampal, 2009). In consistence with studies done in Hospital Staff in Botswana (Garrido et. al., 2009), and Iran (Sahebi et.al., 2014) this study demonstrates that age is positively associated with obesity. An Indian study however showed that age was not associated with obesity in health workers (Sharma et al., 2010). The association between obesity and age in this study may be

probably explained, in part, by a decrease in the level of physical activity with increasing age in both men and women (Martinez-Ros *et al.*, 2001). Also, age effects on body weight are attributed to physiological changes that occur with ageing such as decreased energy requirements at rest (Nooyens *et. al.*, 2008).

This study did not observe association between tribe and obesity. The finding in this study is in agreement with the findings of Aryee *et.al.*, 2013 in Ghana who reported no association between tribe and obesity but in disagreement with a study which showed that women of the Igbo ethnic group in Nigeria were likely to be obese than other women (Okoh, 2013). The inability to find an association may be due to low population of the other tribes in this study. A significant social characteristic that is reported to exhibit gender and ethnic differential to weight status is marriage (Claire, 2013). Married subjects were more obese than singles and widows in this study. Our study is supported by that of Piricci *et.al.*, 2009 who reported that being married was found to be a risk factor for obesity in academic staff of a Turkish University. This is however in contradiction to the study of Sahebi *et.al.*, 2014 among health workers in Iran which showed that widowers were more likely to have obesity compared to the married and singles. The positive relationship between marital status and weight has been explained by many hypotheses including social obligations leading to increased food consumption and decreased time for physical activity, and less focus on body image related to the lack of concerns to attract a potential marital partner (Janghorbani *et al.*, 2008; Tzotzas *et al.*, 2010). Pregnancy and changes in diet following marriage have also been proposed as factors for weight gain in married women (Claire, 2013).

This study did not find an association between religion and obesity. This is in accordance with the outcome of a study of geographic variation of overweight and obesity among Nigerian women (Kandala and Stranges, 2014). Religious denomination was, however, shown to be related to body weight in US men (Kim *et.al.*, 2003). Absence of association between religion and obesity in this study may be due to the fact that the religious activities of our environment are not energy-intensive.

Prevalence of obesity was not associated with education in this study. This is contrary to the findings of studies conducted among health workers in Italy (Abbate *et.al.*, 2006) and civil servants in Kaduna (Oladimeji *et.al.*, 2014). The education of participants of these two studies ranged from primary education to university degree. The least education in the participants of this present study was higher national diploma. So it is possible that postgraduate education does not have additional effect on obesity.

Concurrent with the findings of this study obesity was not associated with smoking in urban Kastina (Wahab et.al., 2014) and among Academic Staff of a Turkish University (Pirincci et.al., 2009). However, it appeared to influence obesity in urban adults of Cameroon (Pasquet et.al., 2003). The prevalence of tobacco use in this study was 0.3% and this may be responsible for lack of association.

There was no association between obesity and alcohol consumption in this study. The finding of this study is similar to that of a study done among civil servants in urban Ghana (Mogre et.al., 2001) but contrary to a study among adults in urban Kastina (Wahab et.al., 2014). Cross-sectional studies found a positive association between alcohol consumption and body weight or measures of abdominal adiposity especially in heavy and binge drinkers (Arif and Rohrer, 2005; Breslow and Smothers, 2005).

This study did not detect any association between obesity and physical activity. This is in accordance with studies done in urban Abuja (Akaralo-Anthony et.al., 2014) however studies among hospital staff in Jos (Ojomu and Kuranga, 2013) and the urban population of Abeokuta (Afolabi et.al., 2004) found an association. The high prevalence of physical inactivity among women in this study may be one of the factors that could be responsible for their high prevalence of obesity.

Engaging in physical exercise did not protect against obesity in participants of this study. Kapilan, 2010, reported the same finding in South African Nurses. However the outcome of our study is not in concordance with a study done among healthcare workers in Ghana (Kasu et.al., 2015) and Turkey (Oguz et.al., 2008). The obese in this study engaging in sports may have not lost weight to the extent of being of normal weight (Akaralo Anthony.). This study did not observe an association between obesity and sedentary lifestyle. This is in agreement with the studies which have demonstrated that sitting time was not associated with obesity (Pulsford et.al., 2013) and abdominal obesity (Hassapidou et.al., 2013). Other studies have, however, shown significant associations for obesity (Hu et.al., 2003; Parsons et.al., 2008) and abdominal obesity (Healy et.al., 2008).

Cadre was shown to influence general and abdominal obesity in our study. This is in keeping with other Nigerian studies which demonstrated that obesity prevalence was higher in senior cadre than junior cadre civil servants (Oladimeji et.al., 2014) and University staff (Onyeka et.al., 2014,) in Northern part of Nigeria. This may be explained by the fact that senior cadre jobs are essentially at the decision-making level, with less frequent physically demanding routines (Oladimeji et.al., 2014). In addition the higher socio-economic status

among the senior staff brings about increasing prevalence of obesity as lifestyle may be different due to affordability (Derek et al., 2006)

5.1.2: Prevalence of hyperglycaemia (IFG and diabetes) and associated risk factors

Hyperglycaemia is a group of metabolic disorders that threatens to reach pandemic levels by 2030 (IDF, 2006). IFG is a pre-diabetic state and have a high risk of conversion to diabetes. Studies have indicated that both pre-diabetic and diabetic states are at high risk for cardiovascular disease (Novoa et. al., 2005). It is important to screen for IFG and diabetes so that prevention measures can be implemented at the earliest stage thereby preventing or postponing the conversion to diabetes and cardiovascular diseases (Deepthi et.al., 2013).

The overall prevalence of hyperglycaemia in this study was found to be 6.7%; the prevalence of IFG and diabetes being 5.1% and 1.6 respectively. The prevalence of newly diagnosed diabetes was 0.5%. A study among health employees in a city in Mexico(Padierna-Luna et.al., 2007) found higher prevalence of IFG (19%) when compared with our study however a lower rate of 3.3% was reported among senior civil servants of the State Secretariat in Ibadan (Ojewale and Adejumo, 2010). The reason for the difference between this study and that of Ojewale and Adejumo may be because they used a lower cut-off point of FPG.

The prevalence of diabetes in our study population is far more lower than that reported among the staff of University of Ibadan, 11% (Ige et.al., 2013) and senior civil servants at the state secretariat, Ibadan, 4.7% (Ojewale and Adejumo, 2010) as well as in health workers in Cameroon, 4.8% (Tachang, 2012), South Africa, 8.8% (Kapita . 2010) and 5.8% in India (Sharma et.al., 2012). Studies in Nigeria which reported prevalence of diabetes as low as that obtained in this study did so in the last two decades. Earlier, in 1998, Olaiunbosun et al had reported a prevalence of 0.8% of DM among civil servants in Ibadan. Also, Onoaje et al reported a prevalence of 2.8% in an adult population in Ibadan a year before. Njewu et. al., reported a prevalence of 2.2% in residents of Port Harcourt in 2003. The prevalence of 1.6% found in this study is also lower than the 2008 national prevalence (WHO, 2014) of 2%. Majority of the participants of this study are Nurses. Duties of Nurses include patient care, stooping activities, lifting and transferring of patients and stock, and pushing of patients in beds and or wheelchairs (Kapitan, 2010). These activities improve insulin sensitivity and glycemia even in the absence of considerable weight loss (Thompson et. al., 2001; Dipietro et.al., 2006).

This study did not observe an association between gender and hyperglycaemia. This is in accordance with studies done as a survey in Nigeria (Abubakari and Bhopal, 2007) as well as in urban China (Wang et.al., 2009). Other studies have, however, observed association between gender and hyperglycaemia in Kastina city, rural China (Li et. al., 2015) and the USA (Flegal et.al., 2002). This result may indicate that the body's mechanism of handling glucose is not different in Nigerian men and women.

The present study shows increase in prevalence of IFG with increase in age. Similar results were observed by other studies (Chow et.al., 2006; Kokiwar et.al., 2007; Raghupathy et.al., 2007) but contradicted by the study of Sahai et.al., 2011 in India. Globally, the greatest numbers of people with diabetes are aged between 40 and 59 years (IDF, 2011) as observed in this study. All the diabetics in this study were all over 40 years of age. This result is close to that of study in Uyo metropolis which reported that the prevalence of diabetes was highest in the 46- 60 age group (Ekpeyong et.al., 2012). In contradiction to our study Nkwoka et.al., 2014 observed that age was not associated with diabetes among University Staff in Sokoto. The worsening of insulin resistance with age, increased inactivity and longevity of diabetes patients due to improved care have been proposed as likely reasons for the rising prevalence of IFG and T2DM with age (Burattini et.al., 2006; Di Nardo et.al., 2009).

There was no association between tribe and diabetes in this study. This finding is in agreement with a survey on factors associated with diabetes across the 36 states of Nigeria which showed that ethnicity was not associated with diabetes (Kyari et.al., 2014). It was opined in a study of factors associated with diabetes in Port Harcourt that being Ibibio, Hausa or Fulani were associated with significantly higher prevalence of type 2 diabetes (Nyewa et.al., 2003). Only one Ibibio was a participant of this study moreover individuals of Hausa and Fulani ethnicity were not recruited for this study; this may be responsible for why this study could not record an association.

Marital status was not associated with hyperglycaemia in this study. This finding is in accordance with a study in a rural Vietnam population (Quang Binh et.al., 2012). In agreement with this study Okonkwo et.al., 2013 reported an association in Bukuru Metropolis, Jos.

In this study higher prevalence of IFG was found among participants who had obesity compared to those who did not. This finding is consistent with the outcome of a study done among health workers in Brazil (Almeida et.al., 2011). Moreover, compared to those with

normal FPG levels, the participants of this study with IFG tended to have relatively higher waist. This is in keeping with the results by previous studies (Khambalia et.al., 2011; Chen and Yeh, 2013). A study done in India found no association between BMI and IFG (Kumar et.al., 2008). BMI was also not associated with diabetes in Guinea (Baldé et.al., 2007) as was also observed in our study. Obesity could result in higher insulin concentration, secretion and resistance causing IFG (Sainaghi et.al., 2008). The finding of no association between diabetes and obesity in this study may be due to the fact that weight loss is a feature of diabetes (ADA, 2010a)

Religion was not associated with diabetes in our study. An Indian study however detected an association (Valliyot et.al., 2013). The reason for our finding may be because our religious activities are of low physical activity type.

Contrary to our study, studies in Uyo (Ekpeyong et.al., 2012) and Abuja(Kyari et.al., 2014) reported that education was associated with diabetes. All the subjects of this study are graduates, additional certificates may therefore not have effect on diabetes.

This study found no significant association between hyperglycaemia and tobacco use. This observation is consistent with the study done in Port Harcourt (Nyewe et.al., 2003) and rural India (Prabhakaran et.al., 2005, Majgi et.al., 2010) but is in disagreement with the outcome of studies done in the city of Jos (Okonkwo et.al., 2013) .The prevalence of smoking is very low in this study and may therefore be inconclusive. Low smoking among health workers may be a reflection of their understanding of the effect.

This study did not find any association between alcohol usage and hyperglycaemia. Okonkwo et.al 2013 reported the same finding. Studies have however found association between alcohol consumption and IFG in Koreans (Kim et.al., 2006) as well as diabetes among Fulani in Jos (Puepet and Ohwovoriole, 2008). Alcohol consumers in this study are not heavy drinkers and this may be responsible for the lack of association.

There exists an association between diabetes and physical stress in this study. Our study is opposed by On'Kin et. al., 2008 in Kinshasa but contrary to another study among Brazilian miners which observed that there was no greater chance of developing diabetes among mining workers who reported work stress when compared to those that did not report it (Cavadas et.al., 2010). Stress activates the hypothalamo-pituitary-adrenal (HPA) axis and the central sympathetic system which leads to production of hormones that increase glucose production and insulin resistance. This mechanism has been proposed to be responsible for

the development of type 2 diabetes in stressed individuals (Rosmond and Björntorp, 2000; Agardh et.al., 2003).

Hyperglycaemia was not associated with physical activity in this study. In studies done in Jos (Puepet and Ohwovoriole, 2008) and India (Mohan et al 2006; Gupta et.al., 2010) diabetes was significantly observed among participants who did not engage in physical activity.

Hyperglycaemia was not associated with sedentary time in this study. Previous studies have, however, found an association (Higgins et.al., 2009; Wilmot et.al., 2012).

Hypertension was significantly associated with diabetes prevalence in our study. Studies conducted among Nurses in Brazil (Vilarinho et.al., 2010), women in Nnewi (Osuji et.al., 2010) and bank employees in India (Parashar et. al., 2009) have reported similar finding. In contrast studies have demonstrated that hypertension was not associated with diabetes in Kinshasa (On'Kin et.al., in 2008) and Guinea (Balde et.al., 2008). The aetiological factors linking diabetes and hypertension include genetic factors, insulin resistance, inflammation, the renin-angiotensin-aldosterone system (RAAS), sodium retention, and hyperglycemia (Stump et.al., 2005; Sharma et.al., 2006; Shoelson et.al., 2006).

Family history of diabetes showed a significant association with the occurrence of diabetes in our subjects however this is not so with IFG. Earlier studies have shown that the risk of IFG and diabetes was higher among individuals with family history of diabetes. (Wang et.al., 2009; Abebe et.al., 2014). Nadeeni et al and Gupta et al from their studies done in India and Colagiuri et. al., 2002 in their study on the prevalence of diabetes in the kingdom of Tonga reported similar findings for diabetes. Family history of diabetes in a first degree relative also presents a risk that is more than double (Chege, 2010). We found important relationship between DM and history of DM among first degree relatives like other previous studies (Nazmul Ahsan et.al., 2011, Sayeed et.al., 2003, 2007, Shera, 2007). Genetic studies have demonstrated the susceptibility of individuals with family or ethnic history of diabetes to increased (McCarthy and Froguel, 2002) or decreased risk to type2 diabetes (Altshuler et. al., 2000).

Cadre of job was not associated with diabetes in this study. Parashar et. al., 2009 reported similar finding. The low prevalence of diabetes may obscure possible association.

5.1.3: Prevalence and risk factors associated with dyslipidaemia

Dyslipidaemia (high TC, high LDL, low HDL, and high TG) is associated with increased CVD risk (WHO, 2010). Estimation of the prevalence of dyslipidaemia as well as knowledge of patterns of association with its risk factors ensures proper planning of health actions for both primary and secondary prevention of cardiovascular diseases. As reported in other studies the prevalence of overall dyslipidaemia in our study is high; about two-thirds of the subjects had dyslipidaemia. This is consistent with the finding of a review study on dyslipidaemia in Nigeria (Oguejiofor et.al., 2012) as well as that of a study among the working population in Spain (Sanchez-Chaparro et.al., 2006). It is however contradictory to the study of dyslipidaemia in rural Edo State which found a prevalence of 37.1% (Okaka et.al., 2013). It has been postulated that high prevalence of dyslipidaemia in developing countries may be closely linked to rapid urbanization and western diet with most urban cities saturated with fast food outlets and increasing sedentary lifestyle which contrasts with our previous highly active agrarian based lifestyle (Oguejiofor et.al., 2012). The magnitude of the problem of dyslipidaemia in this population is surprising since it involves health workers. We would expect to find a lower prevalence of disease than that found in population studies, due to the so-called health worker effect.

The most frequent form of dyslipidaemia was low HDLC and the least frequent hypertriglyceridaemia. This is consistent with the findings of studies done among residents in the city of Campos dos Goytacazes in Brazil (Souza, 2010) and apparently healthy professionals in Asaba (Odenigbo and Oguejiofor, 2008). In contrast to this finding Okaka et. al., 2013 demonstrated that total cholesterol and not HDLC was the most frequent dyslipidaemia in a rural community in Edo state.

The prevalence of high total cholesterol, hypertriglyceridaemia, high LDLC and low HDLC in this study was 33.2%, 2.7%, 39.4% and 45.3%, respectively. The prevalence of high total cholesterol in this study population is similar to that obtained in a study among government employees (31.3%) in urban population of India (Sekhri et. al., 2014). Our result is, however, lower than the prevalence of 51% found among health workers in Benin city (Eno and Enofe, 2013).

Previous studies among health workers in Botswana reported prevalence of hypertriglyceridaemia to be 14% (Garrido et.al., 2003). The observed prevalence was higher than that found in this study. Moreso, the finding of this study is also lower than the prevalence of 8.7% found among rural Edo community (Okaka et.al., 2013).

The results of the prevalence of HDLC and LDLC of our study can be compared with that found in Turkish adults. In the study low HDLC was present in 41.5% subjects and LDLC in 36.2% (Bayram et.al. 2014). This finding however differ from that reported for Angolan government employees which found a higher rate of 50.1% for HDLC and a lower rate of 19.8% for LDLC (Capingana et. al., 2013).

This study did not observe an association between dyslipidaemia and gender. A study among health workers in Benin city could also not uncover an association in the prevalence of high TC and high LDL-C between men and women (Edo and Enofe, 2013). This study is however in disagreement with the results of a study among civil servants in Abakaliki which reported that females were more likely to have dyslipidaemia than men (Ugwuja et.al., 2013).

A study of prevalence of dyslipidaemia among civil servants in Abakaliki revealed that unfavourable plasma lipids was associated with age (Ugwuja et.al., 2013). These findings were also observed in this present study except for HDLC. The study of Erem et.al., 2008 among Turkish adults however showed that age was not associated with total cholesterol and LDLC. Some evidence demonstrates that the causes of age-related disruption of lipid homeostasis include the gradual decline in fractional clearance of LDL with increasing age, the progressively reduced ability to remove cholesterol through conversion to bile acids, and the decreased activity of the rate-limiting enzyme in bile acid biosynthesis, cholesterol 7-hydroxylase (Trapani and Pallottini, 2010). Moreover, an interesting hypothesis states that critical changes in cholesterol and lipoprotein metabolism depend on the progressive decrease in growth horinone (GH) secretion, a characteristic feature of aging. Growth hormone plays an important role in cholesterol homeostasis by either modulating the expression of hepatic LDL_r (Matasconiet.al., 2005) or controlling the activity of cholesterol 7-hydroxylase (Rudling et.al., 1997). Hypertriglyceridemia associated with old age are related to a profound reduction in the liver expression and activity of PPAR α , and several of its target genes (Sanguino et al., 2004).

Marital status was associated with HDLC in this study. A study done in rural Chinese reported similar finding however a study done among Malaysian women did not (Sun et.al., 2014).

This study did not observe an association between dyslipidaemia and education. Sun et al reported association between education and TC as well as LDLC. The high education level of all subjects in this study may explain the disparities.

The findings of our study suggested that obesity and abdominal obesity are predictors of hypercholesterolaemia and low HDL. The results of this study are comparable with the findings of other studies which showed that LDLC (Souza et.al., 2003) and TC (Khader et.al., 2010) increases while HDLC decreases with body mass index and waist circumference (Khader et.al., 2010). Erem et. al., 2008, in contrast, demonstrated that BMI and WC were not associated with TC and LDLC. Obesity is the main cause of the resistance to insulin-mediated glucose disposal and compensatory hyperinsulinemia, which are in turn responsible for most, if not all, of the associated lipoprotein abnormalities (Howard and Robins, 2003). Insulin resistance causes overproduction of very low density lipoproteins (VLDL) particles and defective lipoprotein lipase (LPL)-mediated lipolysis which lead to increased fasting and postprandial TRL (Triglycerides-rich Lipoproteins) concentrations. The increased small, dense LDL and decreased HDL cholesterol concentrations appear to be secondary to the delayed metabolism of TRLs (Howard and Robins, 2003).

In this study IFG was positively associated with hypercholesterolaemia and high LDLC. This observation is in agreement with the finding of the study of Williams et.al., 2005 done among US adults. The dysregulation of lipid and lipoprotein in IFG state may be a manifestation of underlying insulin resistance, which is a central pathophysiological feature of type 2 diabetes and abdominal obesity (Qian et.al., 2010). Insulin resistance results in hyperinsulinemia, enhanced hepatic gluconeogenesis and glucose output. It also reduces suppression of lipolysis in adipose tissue, leading to high free acid flux, and it increases hepatic very low density lipoprotein secretion leading to hypertriglyceridemia, high LDLC and reduced levels of HDL-C (Avramoglu et.al., 2006).

Diabetes mellitus was not associated with dyslipidaemia. This is in disagreement of the study of Al-Kaaba which showed that only HDLC was not associated with diabetes. Two-thirds of the diabetics of this study were aware of their disease condition. Treatment in known diabetics may obscure the relationship.

Smoking was not associated with dyslipidaemia in this study. This contradicts the study of Grabauskas et.al., 2013 which reported an association in rural Lithuanian population.

Total cholesterol and high LDLC were also positively associated with alcohol consumption in this study. This is in agreement with other reports (Erem et.al., 2008). During ethanol metabolism, large amounts of reduced nicotinamide-adenine dinucleotid (NADH) is generated, thus, the Krebs cycle and oxidation of fatty acid are inhibited, which favours liver steatosis and serum hypercholesterolaemia (Lieber, 2004; Rukkumani et.al., 2002).

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In agreement with other studies (Polychronopoulos et.al., 2005; Dancy et.al., 2008) this study reported a positive association between physical activity and HDL-C. AL-Kaabba et.al., 2012 in contrast found that HDLC was not associated with physical activity. Increased physical activity improves insulin sensitivity; this dampens lipid abnormalities (Grundy, 2006).

(Grundy et.al., 2004).

This study observed an association between physical activity and plasma HDLC. This is in keeping with other studies (Polychronopoulos et.al., 2005; Dacy et.al., 2008). Al-Kaabba et.al., 2012 in their study among Saudi Arabian adults in contrast did not.

Individuals who engaged in sports activity had improved plasma HDL-C but not TC, LDL and triglycerides in this study. Other studies have demonstrated that aerobic (Kelly et.al. 2004) and endurance exercise training (Halverstadt et al., 2007) were efficacious for increasing HDL-C .Physical exercise has been shown to increase LPL and hepatic lipase activity, which stimulates TG lipolysis leading to increased HDLC (Thomas et.al., 2001).

This study found an association between sitting time and HDLC but with not total cholesterol, triglycerides and LDLC. Previous studies have demonstrated that sedentary time was associated with high triglycerides and HDLC only in women (Healy et.al., 2008) and hypercholesterolaemia in men but not in women (Hassapidou et.al., 2013). It has been suggested that a possible pathway may be through the effect of inactivity on lipoprotein lipase (LPL) that regulates plasma triglycerides and HDL cholesterol (Hamilton et.al., 2007).

Hypertensives tended to have more risk for hypercholesterolaemia and high LDLC than non-hypertensives in this study. This observation is supported by the studies carried out by Souza et.al., 2003 among residents in the city of Campos dos Goytacazes, Brazil and among a resettled colony in India (Sharma et.al., 2014) who reported similar findings. Prehypertension however was not associated with dyslipidaemia in this study. It may be that blood pressure in prehypertension range is not able to exert dyslipidaemia. Studies have linked lipids and hypertension through a mechanism of angiotensin I overexpression (Nickenig and Harrison, 2002a&b). Activation of Renin-Angiotensin System leads to release of reactive oxygen species and transcriptional up-regulation of LDL and increased ox-LDL uptake in macrophages, smooth muscle cells, and endothelial cells (Singh and Mehta, 2003; Stump et.al., 2005).

Our study indicated that cadre is associated with total and LDL cholesterol. This is in accordance with a study done among staff of Federal University of Technology Owerri, Imo and adults of Saudi Arabia (Al-Kaaba et.al, 2012) but contradictory to the study of Yamell et.al, 2005 among Northern Ireland men who reported that there was no significant association between occupational status and dyslipidaemia.

5.1.4: Clustering of risk factors

Studies have shown that individuals with who have clustering of CV factors are at a considerably higher risk for CVD (Eberly et.al., 2006 ; Taylor et al., 2008). A small proportion of participants (15%) in this study had no factors whereas 64.2% had at least two factors and 11.8% had at least 3 factors. The most common clustering in this study is obesity and dyslipidaemia. These findings are similar to that of Akintunde et.al., 2013 who found out that about 72.3% of staff of Ladoke Akintola University, Ogbomoso had at least two CV risk factors clustered together and that the most frequent clustering were low HDL and obesity. These findings are quite alarming and warrant a substantial public health effort to prevent and/or control these potentially hazardous factors among this set of health workers.

5.3: PUBLIC HEALTH IMPLICATIONS

Obesity, dyslipidaemia and type 2 diabetes mellitus are associated with high risk for cardiovascular diseases which in turn is the single largest cause of death worldwide .The contribution of obesity to diabetes and dyslipidaemia is immense. Also, obesity and diabetes once established are difficult to control. It would be expected that the prevalence of these preventable diseases would be low among health workers. The custodians of health in this community are the ones having very high prevalence. This has serious implications on public health because hospital staff are supposed to serve as role models to the general public in the global fight to reduce cardiovascular risk factors. This warrants urgent intervention so as to prevent epidemic of cardiovascular diseases in our community.

5.4: LIMITATIONS

Majority of the participants did not disclose their income and found it hard to recall sitting as well as walking time. The cross-sectional design of this study limits conclusions about the direction of causality. This study is also limited by its setting in senior staff of the UCH, Ibadan thus the results cannot be generalized to junior staff and the general population. The fasting plasma glucose of subjects who had impaired fasting glucose was not repeated because participants did not want to be bled a second time. Oral glucose tolerance tests

(OGTTs) were not conducted, which indicates that the prevalence of diabetes could have been underestimated.

5.5: CONCLUSION

Obesity, impaired fasting glucose and dyslipidaemia are prevalent among senior staff of University College Hospital (UCH), Ibadan. The prevalence of obesity and dyslipidaemia was higher than expected but that of diabetes was acceptable. The data support the notion that prevalence of these metabolic disorders increase with urbanization and aging of the population. This study indicates there is a high risk of lifestyle-associated conditions within a group of people who have good access to medical facilities and information and are responsible for promoting health, healthy values and behaviours among the population.

Modifiable factors such as obesity, alcohol consumption and cadre are important factors.

Factors associated with obesity include gender, age, marital status, being a first degree relative of diabetics, and cadre. Being a first degree relative of a diabetic was an independent factor associated with obesity. Being a female, of age greater than or equal to 40 years old, married and of a higher cadre were factors that were associated with abdominal obesity. Gender and marital status were the independent risk factors associated with abdominal obesity.

Being of age greater than or equal to 40 years old, obese, abdominally obese were associated with impaired fasting glucose. There was no independent association between IFG and any socio-demographic or lifestyle factors. Physical stress, age greater than or equal to 40 years older, hypertension and family history of diabetes were factors associated with diabetes. Hypertension was an independent risk factor for diabetes.

Hypercholesterolaemia and high LDLC were associated with age, obesity, abdominal obesity, impaired fasting glucose, alcohol consumption, presence of hypertension and cadre. The independent factors of hypercholesterolaemia were alcohol consumption and cadre while body mass index and alcohol consumption were independent factors found to be associated with increased LDLC. Age was the only factor associated with hyperglycaemia in this study. Marital status, obesity, abdominal obesity, physical activity and sports activity are factors associated with low HDLC. Obesity and sedentary time were the independent factors associated with obesity. The most common clustering was obesity and dyslipidaemia.

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Modifiable factors such as obesity, alcohol consumption and cadre are important factors. Factors associated with obesity include gender, age, marital status, being a first degree relative of diabetics, and cadre. Being a first degree relative of a diabetic was an independent factor associated with obesity. Being a female, of age greater than or equal to 40 years old, married and of a higher cadre were factors that were associated with abdominal obesity. Gender and marital status were the independent risk factors associated with abdominal obesity.

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APPENDIX I

Ethical Considerations:

Research protocol as well as potential benefit will be explained to the participants with a view to obtaining informed written consent.

(i) Confidentiality of data

All information and data collected in this study will be given code numbers and no name will be recorded. This cannot be linked to any participant in anyway and names or any identifier will not be used in any publication or reports from this study.

(ii) Translation of protocol to Language of the participant

The participants of this study will all be educated and therefore there will be no need of translation into local language.

(iii) Beneficence to participants

In case of any abnormality determined, the subject will be referred to the Staff Clinic of University College hospital, Ibadan for appropriate intervention.

(iv) Non-maleficence of participants

The blood samples will be collected by competent phlebotomists so as to minimize stress and pain

(v) Voluntariness: Informed consent will be obtained from all participants without coercion and they would be made to know that they have the right to withdraw from the study if they wish.



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UI/UICH EC Registration Number: NHREC/05/01/2008a

NOTICE OF FULL APPROVAL AFTER FULL COMMITTEE REVIEW

Re: Prevalence and Risk Factors Associated with Obesity, Hyperglycaemia and Dyslipidaemia among Senior Staff of the University College Hospital, Ibadan

UI/UICH Ethics Committee assigned number: UI/EC/13/0229

Name of Principal Investigators: Omolara O. Popoola

Address of Principal Investigators: Department of Epidemiology & Medical Statistics,
College of Medicine,
University of Ibadan, Ibadan

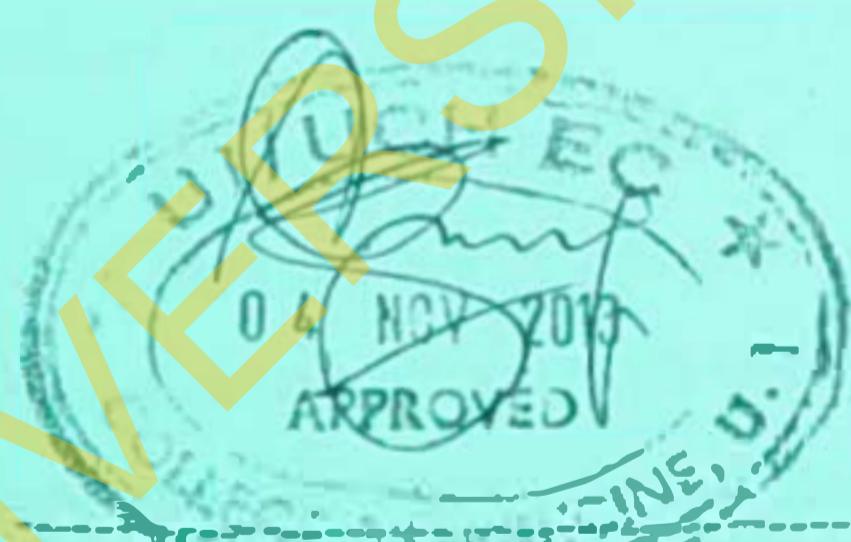
Date of receipt of valid application: 03/07/2012

Date of meeting when final dateetermination ethical approval was made: N/A

This is to inform you that the research described in the submitted protocol, the consent forms, and other participant information materials have been reviewed and given full approval by the UI/UICH Ethics Committee.

This approval dates from 04/11/2013 to 03/11/2014. If there is delay in starting the research, please inform the UI/UICH Ethics Committee so that the dates of approval can be adjusted accordingly. Note that no participant accrual or activity related to this research may be conducted outside of these dates. All informed consent forms used in this study must carry the UI/UICH EC assigned number and duration of UI/UICH EC approval of the study. It is expected that you submit your annual report as well as an annual request for the project renewal to the UI/UICH EC early in order to obtain renewal of your approval to avoid disruption of your research.

The National Code for Health Research Ethics requires you to comply with all institutional guidelines, rules and regulations and with the tenets of the Code including ensuring that all adverse events are reported promptly to the UI/UICH EC. No changes are permitted in the research without prior approval by the UI/UICH EC except in circumstances outlined in the Code. The UI/UICH EC reserves the right to conduct compliance visit to your research site without previous notification.



Professor A. Ogunniyi
Director, IAMRAT
Chairman, UI/UICH Ethics Committee
E-mail: uiuchire@yahoo.com

APPENDIX II
INFORMED CONSENT FORM

IRB Research Approval Number:

This approval will elapse on:

Title of research:

Prevalence and risk factors of Obesity, Hyperglycaemia and Dyslipidaemia among Senior Staff of University College Hospital, Ibadan

Name and Affiliation of Researcher:

This study is being conducted by Omolara Olutosin Popoola of the Department of Epidemiology and Medical Statistics, College of Medicine, University of Ibadan.*email: larrylabs@yahoo.co.uk phone: +234-8056738867*

Sponsor of Research:

The research is self-sponsored

Purpose of Research:

The purpose of this research is to determine the prevalence and risk factors of obesity, hyperglycaemia and dyslipidaemia among senior staff of University College Hospital, Ibadan

Procedure of research:

A total of four hundred and ten individuals who are senior staff of University College Hospital, Ibadan will be recruited. The subjects will be chosen using Multistage Sampling method. Written informed consent will be obtained from all participants. Anthropometric Indices such as height and weight will be measured using standard procedures and from these two, body mass index (BMI) will be calculated. Socio-demographic and socioeconomic characteristics of the study population will be obtained through a well structured, self-administered pre-tested and validated, mailed questionnaire. Fasting venous blood samples will be aseptically collected from all subjects of the study for the determination of fasting plasma glucose and lipid profile and another blood sample will be collected two hours after eating.

Expected Duration of study and participants involvement:

The study is expected to be concluded within a period of 3 months and blood samples will be collected from each participant only once throughout the period of study.

Risks:

The process of collection of blood samples will pose no threat to the participant as the blood sample will be collected aseptically and competent individuals. The needle prick will cause some short lived minor pain to the participants.

Benefits:

The results of the study will be given to participants and those requiring medical attention will be referred to the staff clinic. The research will provide evidence-based information to help advocate for appropriate interventions to prevent or reduce cardiovascular risk among staff of the hospital.

Confidentiality:

All information provided by the participants will be kept confidential and not passed to a third party. The participants will be identified with numbers and there will be no connection between them and the findings of the research.

Voluntariness:

Your participation in this research is entirely voluntary

Alternative to participation:

The participants have full rights to decline or withdraw from this study

Due inducement:

You will not be paid any fees for participating in this research.

Consequences of participants' decisions to withdraw from research and procedure for orderly termination of participation:

You can choose to withdraw from the research anytime however all your information will be kept confidential.

Modality of providing treatments and action(s) to be taken in case of injury or adverse event(s):

In case of injury the injured will be referred to Staff Clinic of the University College Hospital, Ibadan and the researcher will be responsible for the cost.

What happens to participants when research is over?

The researchers will provide results of the laboratory investigation to individuals and hand over those requiring further treatment or monitoring to staff clinic and also provide summary of research results to hospital management to consider intervention implementation.

Statement of sharing benefits among researcher and whether this will include or exclude research participants:

The participant that so desires will collect his or her result.

Conflict of Interest: We are not aware of any other information that may cause the researchers not to do their work without fear or favour.

Statement of person obtaining informed consent:

I have fully explained this research to
and have given sufficient information, including about risks and benefits, to make an informed decision.

Date:.....

Signature:.....

Name:.....

Statement of person giving consent:

I have read the description of the research or have it translated in the language I understand. I understand that my participation is voluntary .I know enough about the purpose, methods, risks and benefits of the research study to judge that I want to take part in it. I have received a copy of this consent form and additional information sheet to keep for my self

Date:..... **Signature:**.....

Name:.....

Witness signature (if applicable):.....

Witness name (if applicable):.....

Detailed contact information including contact address, telephone, fax, e-mail and any other contact information of researcher(s), institutional HREC and head of the institution:

This research has been approved by the Ethics Committee of the University of Ibadan and the Chairman of this committee can be contacted at Biode building room T10 , 2nd Floor, Institute for Advanced Medical Research and Training (IMRAT), College of Medicine, University of Ibadan, Ibadan Ext 2451, E- mail: uiuchirc@yahoo.com.

In addition, if you have any questions about your participation in this research you can contact the principal Investigator. Name: Omolara Olutosin Popoola. Department: Epidemiology and Medical Statistics. Phone: 08056738867. E-mail: larrylabs@yahoo.co.uk
PLEASE KEEP A COPY OF THE SIGNED INFORMED CONSENT

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APPENDIX III
QUESTIONNAIRE
ON
PREVALENCE OF OBESITY, HYPERGLYCAEMIA AND DYSLIPIDAEMIA AND
ASSOCIATED FACTORS AMONG SENIOR STAFF OF UNIVERSITY COLLEGE
HOSPITAL, IBADAN

SECTION A: Socio-Demographic Data

Please fill your response in the space provided

1. Serial Number
2. Date of birth (day/month/year)

Please CIRCLE the appropriate option for the following set of questions:

3. Sex: 1. Male 2. Female
4. Tribe: 1. Yoruba 2.Ibo 3. Hausa 4.Others (Please specify)
5. Marital Status: 1. Single 2.Married 3.Widow 4.Divorced 5.Separated 6. Co-habiting
6. Highest Educational Level Attained:
1. OND 2.HND 3.RN 4.RM 5.BSc 6. MBBS 7.MSc
- 8.PhD
9. Others (please specify)
7. Professional Qualification(s)
8. Religion: 1.Christian 2. Muslim 3.Traditional 4. Others, please specify

SECTION B: Occupational Data

Please fill your response in the space provided for the following set of questions:

9. Profession:
10. Discipline/Specialty (if applicable):
11. Current Salary Grade Level/Step:
12. Present Position at Work:
13. How long have you spent in your present position?

14. How long have you been working in this hospital? _____

15. Average Monthly income

SECTION C: Medical and Drug History

Please CIRCLE the appropriate option for the following questions:

i. History of Hypertension

16. Have you ever had your blood pressure measured by a doctor or other health worker?

1. Yes 2. No (If no please skip to question 22)

17. Have you ever been told by a doctor or other health worker that you have raised blood pressure or hypertension?

- 1. Yes 2. No** **(If No Please Skip to Question 22)**

18. If yes to question 17 have you been told in the past 12 months?

1. Yes 2. No

19. In the past two weeks, have you taken any drugs (medication) for raised blood pressure prescribed by a doctor or other health worker?

1. Yes 2. No

20. Have you ever seen a traditional healer for raised blood pressure or hypertension?

1. Yes 2. No

21 Are you currently taking any herbal or traditional remedy for your raised blood pressure?

1. Yes 2. No

22. Is any of your parents hypertensive? 1. Yes 2. No

23. If yes to (22) who of them? 1. Father 2. Mother 3. Both parents

24. Do you have any other relations with hypertension? 1. Yes 2. No

(If No, please skip to question 26)

(If No please skip to question 26)

25. If yes to (24) what is the relationship of the person to you?

1. Brother 2. Sister 3. Grandmother 4. Grandfather 5. Cousin 6. Uncle 7. Auntie

8. Step-siblings

ii) History of Diabetes

26. Have you ever had your blood sugar measured by a doctor or other health worker?

1. Yes 2. No

(If No please skip to question 33)

27. Have you ever been told by a doctor or other health worker that you have raised blood sugar or diabetes? 1. Yes 2. No (If No please skip to question 33)
28. If yes to (27) have you been told this in the past 12 months? 1. Yes 2. No
29. In the past two weeks, have you taken any drugs (medication) for diabetes prescribed by a doctor or other health worker? 1. Yes 2. No
30. Are you currently taking insulin for diabetes prescribed by a doctor or other health worker? 1. Yes 2. No
31. Have you ever seen a traditional healer for diabetes or raised blood sugar? 1. Yes 2. No
32. Are you currently taking any herbal or traditional remedy for your diabetes? 1. Yes 2. No
33. Is any of your parents' diabetic? 1. Yes 2. No
34. If yes to (33) who of them? 1. Father 2. Mother 3. Both parents
35. Do you have any other relative with diabetes? 1. Yes 2. No (If No please skip to question 37)
36. If yes to (35) what is the relationship of the person to you?
1. Brother 2. Sister 3. Grandmother 4. Grandfather 5. Cousin 6. Uncle 7. Auntie
8. Step-siblings
- iii) History of Raised Cholesterol
37. Have you ever had your cholesterol (fat levels in your blood) measured by a doctor or other health worker? 1. Yes 2. No (If No please skip to question 43)
38. Have you ever been told by a doctor or other health worker that you have raised cholesterol? 1. Yes 2. No (If No please skip to question 43)
39. If yes to (38) have you been told in the past 12 months? 1. Yes 2. No
40. In the past two weeks, have you taken any oral treatment (medication) for raised total cholesterol prescribed by a doctor or other health worker? 1. Yes 2. No
41. Have you ever seen a traditional healer for raised cholesterol? 1. Yes 2. No
42. Are you currently taking any herbal or traditional remedy for your raised cholesterol? 1. Yes 2. No

43. Has any of your relative ever been diagnosed of high cholesterol or other blood lipids?

1. Yes 2. No

44. If yes to (43) who of them? 1. Father 2. Mother 3. Brother 4. Sister 5. Grandmother

6. Grandfather 7. Cousin 8. Uncle 9. Auntie 10. Step-siblings

v. Other Medical Conditions

45. Do you have other medical conditions? 1. Yes 2. No

46. If yes to (54) please state the condition(s)

.....
47. Do you think you are undergoing physical stress (stress caused by overwork)?

1. Yes 2. No

SECTION D: Physical Activity, Lifestyle and Dietary Assessment

Please circle the appropriate option or fill in the space when required for the following set of questions:

i) Tobacco Use:

48. Do you currently smoke any tobacco products, such as cigarettes, cigars or pipes?

1. Yes 2. No (If No please go to question 55)

49. Do you currently smoke tobacco products daily? 1. Yes 2. No

50. On average, how many of the following products do you smoke each day/week?

(If less than daily please record weekly)

Daily

Weekly

a. Manufactured cigarettes

b. Hand-rolled cigarettes

c. Pipes full of tobacco

d. Cigars

e. Others (specify)

51. In the last 24 hours how many cigarettes did you smoke?
52. During any visit to a doctor or other health worker in the past 12 months, were you advised to quit smoking tobacco? 1. Yes 2. No 3. No visit during the past 12 months.
53. If you have stopped, how long ago did you stop smoking?
 1. years ago 2.months ago 3. weeks ago

54. Do you currently use any smokeless tobacco products such as snuff, chewing tobacco?
 1. Yes 2. No

(If No please go to question 57)

55. Do you currently use smokeless tobacco products daily? 1. Yes

56. On average, how many times a day/week do you use

Daily

Weekly

- a. Snuff, by mouth?
- b. Snuff, by nose?
- c. Chew tobacco?
- d. Other

ii) Alcohol

The next questions ask about consumption of alcohol.

57. Have you ever consumed any alcohol such as beer, wine, spirits or other local alcohol?
 1. Yes 2. No (If No please go to question 66)

58. Have you consumed any alcohol within the past 12 months?

1. Yes 2. No

59. Have you stopped drinking due to health reasons, such as a negative impact on your health or on the advice of your doctor or other health worker? 1. Yes 2. No

60. During the past 12 months, how frequently have you had at least one standard alcoholic drink?

(one standard alcoholic drink = 1 standard bottle of regular beer{285 ml}, or 1 single measure of spirits{30ml} or 1 medium size glass of wine {120ml} or 1 measure {60ml} of an alcoholic beverage drink before a meal)

- a. Daily
- b. 5-6 days per week
- c. 3-4 days per week
- d. 1-2 days per week
- e. 1-3 days per month
- f. Less than once a month

61. Have you consumed any alcohol within the past 30 days? 1. Yes 2. No

(If no please go to question 66)

62. During the past 30 days, on how many occasions did you have at least one standard alcoholic drinking occasion? i. occasion (s) ii. don't know

63. During the past 30 days, when you drank alcohol, how many standard drinks on average did you have during one drinking occasion? i) drinks ii) don't know

64. During each of the past 7 days, how many standard drinks did you have each day?

1. Monday 2. Tuesday 3. Wednesday 4. Thursday
..... 5. Friday 6. Saturday 7. Sunday

65. On average, how many standard drinks of the following did you consume during the past 7 days?

Homebrewed spirits, e.g. moonshine drinks

Homebrewed beer or wine, e.g. beer, palm or fruit wine drinks

Alcohol brought over the border/from another country drinks

Alcohol not intended for drinking, e.g. alcohol-based medicines, perfumes, after shaves

iii) Physical Activity

Next you will answer questions about the time you spend doing different types of physical activities in a typical week. Please answer these questions even if you do not consider yourself to be a physically active person.

Think first about the time you spend doing work. Think of work as the things that you have to do such as paid or unpaid work, study/training, household chores, harvesting food/crops, fishing or hunting for food. In answering the following questions 'vigorous-intensity activities' are activities that require hard physical effort and cause large increases in breathing or heart rate, 'moderate-intensity activities' are activities that require moderate physical effort and cause small increases in breathing or heart rate

66. Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like *carrying or lifting heavy loads, digging or construction work* for at least 10 minutes continuously? 1. Yes 2. No (If No please go to question 69)

67. In a typical week, on how many days do you do vigorousintensity activities as part of your work? days

68. How much time do you spend doing vigorous-intensity activities at work on a typical day?

i). minutes ii) hours

69. Does your work involve moderate-intensity activity that causes small increases in breathing or heart rate such as *brisk walking or carrying light loads* for at least 10 minutes continuously?

1. Yes 2. No (If No please go to question 72)

70. In a typical week, on how many days do you do moderate-intensity activities as part of your work? days

71. How much time do you spend doing moderate-intensity activities at work on a typical day?

i). minutes ii) hours

The next questions exclude the physical activities at work that you have already mentioned. Now you would be asked about the usual way you travel to and from places. For example to work, for shopping, to market, to place of worship.

72. Do you walk or use a bicycle (*pedal cycle*) for at least 10 minutes continuously to get to and from places? 1. Yes 2. No (If No please go to question 75)

73. 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? days

74. How much time do you spend walking or bicycling on a typical day?
i) minutes ii) hours

The next questions exclude the work and transport activities that you have already mentioned. Now you will be required to answer questions about sports, fitness and recreational activities (*leisure*)

75. Do you do any vigorous-intensity sports, fitness or recreational (*leisure*) activities that cause large increases in breathing or heart rate [like *running or football*] for at least 10 minutes continuously?

1. Yes 2. No (If No please go to question 78)

76. In a typical week, on how many days do you do vigorous-intensity sports, fitness or recreational (*leisure*) activities? days

77. How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?
i) minutes ii) hours

78. Do you do any moderate-intensity sports, fitness or recreational (*leisure*) activities that cause a small increase in breathing or heart rate such as brisk walking, *cycling, swimming, volleyball* for at least 10 minutes continuously?

1. Yes 2. No (If No please go to question 81)

79. In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (*leisure*) activities? days

80 How much time do you spend doing moderate-intensity sports, fitness or recreational (leisure) activities on a typical day?

i) minutes

ii) hours

iv. Sedentary behaviour

The following question is about sitting or reclining at work, at home, getting to and from places, or with friends including time spent sitting at a desk, sitting with friends, travelling in car, bus, train, reading, playing cards or watching television, using a computer, doing hand craft like knitting, resting etc. but do not include time spent sleeping.

81. How much time do you usually spend sitting or reclining on a typical day?

i) Minutes

ii) Hours

v) Diet

The next questions ask about the fruits and vegetables that you usually eat. As you answer these questions please think of a typical week in the last year.

82. In a typical week, on how many days do you eat fruit?

(A typical week means a "normal" week when the diet is not affected by cultural, religious, or other events).

i) days

ii) don't know

(If zero days, please go to question 84)

83. How many servings of fruit do you eat on one of those days?

i) Servings

ii) don't know

(A serving of fruit {80g} is one medium sized whole fruit or one-half cup of chopped, cooked or canned fruit or one-half cup fruit juice not artificially flavoured)

84. In a typical week, on how many days do you eat vegetables?

i) days

ii) don't know (If zero days, please go to question 86)

85. How many servings of vegetables do you eat on one of those days?

i) Servings

ii) don't know

(A serving is one-half of a cup of cooked or chopped raw vegetable or one cup of raw green leafy vegetable). One standard serving = 80 grams

86. Are you pregnant (for women only)?

1. Yes

2. No

87. Are you breastfeeding (for women only)?

1. Yes

2. No

88. LMP (for women only)

89. Anthropometric Measurements

Weight kg

Height m

BMI kg/m²

WC cm

HC cm

90. Blood pressure mmHg

91. Laboratory Investigations

Total Cholesterol mg/dl

Triglycerides mg/dl

HDL-C mg/dl

LDL-C mg/dl

APPENDIX IV: TABLE ON SELECTION OF PARTICIPANTS

NURSES

Medical Laboratory S

Cadre	Number	Proportional Allocation		Cadre	Number		
		Allocation 1					
		Allocation 1	Allocation 2				
NO	121		31	Med. Lab Scientist(MLS)	1		
SNO	278		71	Snr Med. Lab Scientist	18		
PNO	242		61	Prin Med. Lab Scientist	14		
CNO	302	275	77	Chief Med. Lab Scientist	22		
ADN	135		34	Assistant Director, MLS	14		
DON	5		1	DD, MLS	4		
Subtotal	1083		275	Subtotal	98		

Dieticians

Physiotherapists

Cadre	Number	Allocation	Cadre	Number	P
					F
Dietician1	5		Physiotherapist 1	7	
Principal Dietician	3		Snr Physiotherapist	9	
Chief Dietician	1	2.5	Principal Physiotherapist	7	
Assistant Director	1		Chief Physiotherapist	8	
Subtotal	10	2.5	Assistant Director	3	
			DD	1	
			Subtotal	35	

Records

Radiographer

Number	Proportional Allocation 1	Proportional Allocation 2		Number	Proportional Allocation 1
HRO 1	6		1.1	Radiographer 1	4
SHFO	11		1.9	Senior Radiographer	4
Prin HRO	1		0.2	Prin Radiographer	6
CHRO	2		0.4	Chief Radiographer	3
DOHRO	1		0.2	AD Radiographer	5
Subtotal	21		1.7	Subtotal	23

APPENDIX IV: TABLE ON SELECTION OF PARTICIPANTS

NURSES				Medical Laboratory Scientists			
Cadre	Number	Proportional Allocation		Cadre	Number	Proportional Allocation	
		Allocation 1	Allocation 2			Allocation 1	Allocation 2
NO	121			31 Med. Lab Scientist(MLS) 1	26		11
SNO	278			71 Snr Med. Lab Scientist	18		3
PNO	242			61 Prin Med. Lab Scientist	14		2
CNO	302	275		77 Chlef Med. Lab Scientist	22		4
ADN	135			34 Assistant Director, MLS	14		2.4
DDN	5			1 DD, MLS	4		0.7
Subtotal	1083		275	Subtotal	98		23

Dieticians			Physiotherapists		
Cadre	Number	Allocation	Cadre	Number	Proportional Allocation 1
Dietician 1	5		Physiotherapist 1	7	1.2
Principal Dietician	3		Snr Physiotherapist	9	1.6
Chief Dietician	1	2.5	Principal Physiotherapist	7	1.2
Assistant Director	1		Chief Physiotherapist	8	9
			Assistant Director	3	0.5
			DD	1	0.2
Subtotal	10	2.5	Subtotal	35	6.2

Records			Radiographer		
	Number	Proportional Allocation 1		Number	Proportional Allocation 1
MRO 1	6		1.1 Radiographer 1	4	0.7
SMAO	11		1.9 Senior Radiographer	4	0.7
Prin MRO	1	5	0.2 Prin Radiographer	6	1.1
CMRO	2		0.4 Chief Radiographer	3	0.5
ODHRO	1		0.2 AD Radiographer	5	0.9
			DD Radiographer	1	0.2
Subtotal	28	1.7	Subtotal	23	5.8

Soc.Med Worker				ACCOUNTANTS			
Cadre	Number	Proportional Allocation 1	Proportional Allocation 2	Cadre	Number	Proportional Allocation 1	Proportional Allocation 2
SMSW	7		1.1	Accountant I	8		1.4
PMSW	4		0.7	Snr Accountant	8		1.4
CMSW	3	3		Prin Accountant	7		1.2
ADMSW	1		0.5	Chief Accountant	5	6	0.8
DD	1		0.2	Assistant Director	5		0.8
			0.2	DD	1		0.2
Subtotal	16	3	3	Subtotal	34	6	

ADMINISTRATORS				
Cadre	Number	Proportional Allocation 1	Proportional Allocation 2	
Administrator I	39			7
Snr Administrator	9			2
Prin Administrator	11			2
Chief Administrator	3	12		1
Assistant Director	5			0.8
Director	1			0.2
Subtotal	68	12		12

APPENDIX V: BIOCHEMICAL ANALYSIS

QUANTITATION OF TOTAL CHOLESTEROL BY ENZYMATIC METHOD

(Trinder, 1969; Allain *et. al.*, 1974; Roeschlau *et. al.* 1974).

This is done by using a combination of the enzymes: cholesterol esterase, cholesterol oxidase and the peroxidase-phenol-4-aminoantipyrine system. Allain *et. al.* 1974 and Roeschlau *et. al.* 1974 were able to constitute cholesterol esterase, cholesterol oxidase into a single enzymatic reagent for the determination of cholesterol. DIALAB cholesterol CHOD-PAP with ATCS single reagent kit was used. This combines the use of these enzymes with the peroxidase- phenol-4- aminoantipyrine system of Trinder for the measurement of total cholesterol (Trinder, 1969).

DIALAB Cholesterol Reagent Composition

COMPONENTS

Good's Buffer (PH 6.7)

Phenol

4-Aminoantipyrine

Cholesterol Esterase

Cholesterol Oxidase

Peroxidase

Standard

FINAL CONCENTRATIONS

50 mmol/L

5 mmol/L

0.3 mmol/L

$\geq 200 \text{ U/L}$

$\geq 50 \text{ U/L}$

$\geq 3 \text{ KU/L}$

200mg/dl

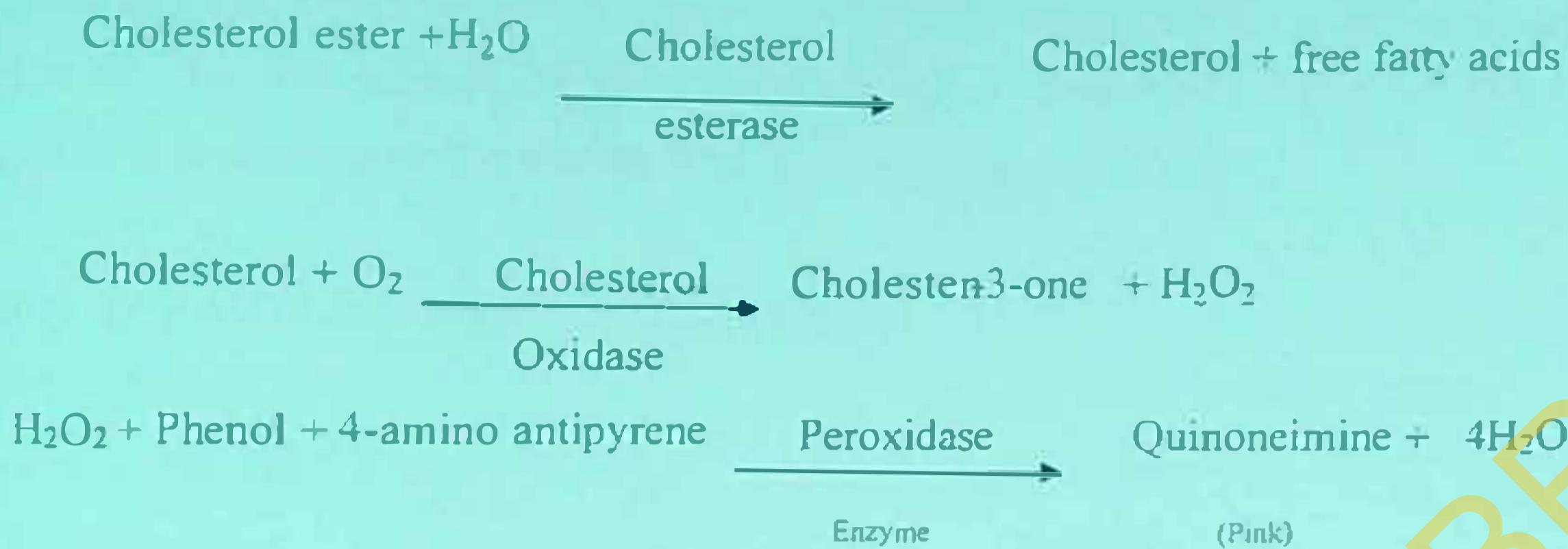
Reagent Preparation

The reagent and cholesterol standard were ready for use.

Principle of the Test:

Cholesterol esterase hydrolyses the cleavage of cholesterol esters to cholesterol and free fatty acids. The free cholesterol in the presence of cholesterol oxidase and oxygen is oxidised to cholesten-3-one with simultaneous production of hydrogen peroxide. The hydrogen peroxide formed oxidatively couples with 4-aminoantipyrine and phenol in the presence of peroxidase enzyme to yield a coloured solution which absorbs light maximally at 500nm. The intensity of the pink colour formed is directly proportional to the cholesterol concentration in the sample.

THE STEPWISE REACTION



PROCEDURE:

	Blank	Sample	Standard	Control
Distilled water	10 μl	-	-	-
Sample	-	10 μl	-	-
Standard	-	-	10 μl	-
Control	-	-	-	10 μl
Reagent	1000 μl	1000 μl	1000 μl	1000 μl

The mixture was incubated at room temperature for twenty minutes and the absorbance was read at 500nm using DU 520 Ultraviolet/Visible Spectrophotometer.

Calculations: $\frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \times \text{concentration of cholesterol standard used}$

Concentration of cholesterol standard = 200mg/dl

TRIGLYCERIDE^S ESTIMATION BY ENZYMATIC METHOD (Trinder, 1969)

Triglycerides are generally determined by hydrolysis of triglycerides to glycerol and free fatty acids. The glycerol concentration is then estimated. DIALAB Triglycerides GPO- PAP with ATCS single reagent kit was used for the estimation of triglycerides.

TRIGLYCERIDE REAGENT COMPOSITION

COMPONENT

Good's Buffer, PH 7.2

4-Chlorophenol

Mg²⁺

ATP

4-Aminoantipyrine

Glycerol kinase

Peroxidase

Lipoprotein lipase

Glycerol-3- phosphate-oxidase

Standard

FINAL CONCENTRATIONS

50mmol/L

4mmol/L

15mmol/L

2mmol/L

0.5 mmol/L

≥ 0.4KU/L

≥ 2 KU/L

≥ 2KU/L

≥ 0.5KU/L

200mg/dl

Reagent Preparation

The reagent and triglycerides standard were ready for use

Principle of test:

Triglycerides are hydrolysed by lipoprotein lipase to yield glycerol and fatty acids. The glycerol produced is phosphorylated to yield glycerol-3- phosphate. The reaction is catalysed by glycerol kinase and the enzyme requires magnesium ions as cofactor. The product, glycerol-3-phosphate is oxidized to dihydroacetone phosphate under the catalysis of glycerol peroxidase with an accompanying production of hydrogen peroxide. Hydrogen peroxide oxidatively couples with 4- aminoantipyrine and 4-chlorophenol to yield the chromogen, quinoneimine. This reaction is catalysed by peroxidase. The coloured complex formed absorbs light maximally at 500nm and the intensity of the colour is directly proportional to the concentration of triglyceride present in the sample.

REACTIONS



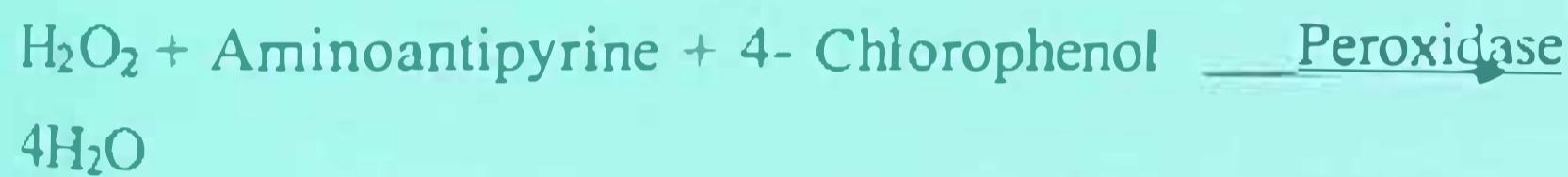
Lipase



Kinase, Mg^{2+}



oxidase



Quinoneimine + HCl +

*DHAP= Dihydroxyacetone phosphate

PROCEDURE:

	Blank	Sample	Standard	Control
Distilled water	10 μl	-	-	-
Sample	-	10 μl	-	-
Standard	-	-	10 μl	-
Control	-	-	-	10 μl
Reagent	1000 μl	1000 μl	1000 μl	1000 μl

The mixture was incubated at room temperature for twenty minutes and the absorbance was read at 500nm using DU 520 Ultraviolet /Visible Spectrophotometer.

Calculations: $\frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \times \text{concentration of triglycerides standard used}$

Concentration of standard = 200mg/dl

ESTIMATION OF HIGH DENSITY LIPOPROTEIN (HDL) CHOLESTEROL.

FOTRESS diagnostic HDL-cholesterol precipitant kit was used for the precipitation of apo-B containing lipoproteins.

Principle:

Low density lipoprotein (LDL), very low density lipoprotein (VLDL) and chylomicron fractions are quantitatively precipitated by the addition of phosphotungstic acid in the presence of magnesium ions. HDL cholesterol subfraction remains in the supernatant after centrifugation and this is assayed for using enzymatic method.

HDL-C Precipitant Composition

COMPONENT	CONCENTRATION
Phosphotungstic acid	0.55 mmol/L
Magnesium Chloride	25.00 mmol/L

Standard

Cholesterol

50 mg/dl

Reagent Preparation

Reagent and Standard were ready for use.

PRECIPITATION PROCEDURE: A dilution of four to one ratio of the precipitant with water was made. The following was pipetted into test tubes

Sample	200 µl
Diluted precipitant	500 µl

The mixture was incubated for ten minutes at room temperature after which it was centrifuged at 4,000 rpm for another ten minutes. The resultant clear supernatant was separated out and analysed for HDL cholesterol using the enzymatic method for cholesterol as follows;

PROCEDURE:

	Blank	Sample	Standard	Control
Sample	-	100µl	-	-
Distilled water	100µl	-	-	-
Standard	-	-	100 µl	-
Control	-	-	-	100 µl
Reagent	1000µl	1000µl	1000µl	1000µl

The mixture was incubated at room temperature for twenty minutes and the absorbance was read at 500nm using DU 520 Ultraviolet /Visible Spectrophotometer.

Calculations: $\frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \times \text{concentration of cholesterol standard used} \times 3.5$

Concentration of cholesterol standard = 50mg/dl and 3.5 equals the dilution factor of the HDL precipitant.

LOW DENSITY LIPOPROTEIN CHOLESTEROL DETERMINATION

(Friedwald *et. al.*, 1972)

Friedwald's equation was used to calculate the LDL- cholesterol by substituting in the triglyceride, HDL- cholesterol and total cholesterol values.

The formula is given by:

$$\text{LDL cholesterol (mg/dl)} = \frac{\text{TC} - \frac{\text{TG}}{5} - \text{HDL-C}}{2}$$

ESTIMATION OF FASTING PLASMA GLUCOSE (Barham and Trinder, 1972;

Thomas, 1998)

DIALAB glucose GOD-PAP diagnostic reagent kit was used for the estimation of glucose.

Principle:

Glucose present in the sample is oxidised to gluconic acid and hydrogen peroxide. The hydrogen peroxide generated reacts with phenol and 4-aminophenazone in the presence of peroxidase to form a quinoneimine dye which absorbs light maximally at 500nm. The intensity of the pink colour formed is directly proportional to the glucose concentration in the sample.

REAGENT COMPOSITION

COMPONENTS

Phosphate Buffer, pH 7.5

Phenol

4-Aminoantipyrine

Glucose Oxidase

Peroxidase

FINAL CONCENTRATIONS

250 mmol/L

5 mmol/L

0.5mmol/L

> 10 KU/L

> 1KU/L

REAGENT PREPARATION

Reagent and Standard were ready for use

PROCEDURE:

	Blank	Standard	Sample	Control
Reagent	1000µl	1000µl	1000µl	1000µl
Sample	-	-	10µl	-
Standard	-	10µl	-	-
Control	-	-	-	10µl

The mixture was incubated at 37°C for 10 minutes. The resultant coloured solution was read at 500nm using DU 520 Ultraviolet Visible Spectrophotometer.

Calculations: $\frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \times \text{concentration of glucose standard used}$

Concentration of standard = 100mg/dl