CASTELLI RISK INDEX AND LIPID PROFILE VARIATIONS AMONGST A POPULATION OF YOUNG OBESE FEMALE STUDENTS IN THE UNIVERSITY OF BENIN

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CERTIFICATION

This is to certify that this project on **CASTELLI RISK INDEX AND LIPID PROFILE VARIATIONS AMONGST A POPULATION OF YOUNG OBESE FEMALE STUDENTS IN THE UNIVERSITY OF BENIN** was carried out by **ENOBAKHARE PRECIOUS EGHOSA** with matriculation number **BMS1702215** in partial fulfilment for the award of Bachelor of science (B.Sc.) Degree in the department of Physiology, School of Basic Medical Sciences, College of Medical Sciences, University of Benin.

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DEDICATION

This project work is dedicated to almighty God who has been my strength and my parents for their financial, moral and spiritual support and finally my siblings and friends for their love and support.

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ABSTRACT

In Africa, despite the high prevalence of under nutrition, the prevalence of overweight and obesity is increasing at an alarming rate. This leaves us with a double burden of malnutrition and obesity with a need for prevention. Children and young adults who are obese are more likely to have a clustering of cardiovascular risk factors such as dyslipidemia, hypertension and type-2 diabetes mellitus, which persist into adulthood. Unsaturated FAs include mono-unsaturated FAs and poly-unsaturated FAs. Mono-unsaturated FAs are considered to be the healthiest types of FAs because they help to reduce harmful low-density lipoproteins (LDLs) that are closely associated with coronary heart disease. They are mainly found in olive oil, rapeseed oil, nuts and seeds. This study is aimed at examining the castelli risk index and lipid profile variation amongst a population of young obese female students in the University of Benin. The study was carried out across the university environment in ovia north east local government area. 60 participants were employed for this study with 30 obese subjects as test subjects and 30 slim subjects as control. 5mls of blood were collected into lithium heparin bottles and centrifuged for 15mins. The supernatant was separated using a pasteur pipette and placed in plain bottles after which they were refrigerated at -20^{o}C for lipid profile analysis (total cholesterol/HDL cholesterol and LDL/HDL cholesterol ratios). Castelli Risk Index, CRI was calculated as the ratio of TC/HDLc and LDLc/HDLc respectively while Atherogenic Index of Plasma, AIP is a logarithmically transformed molar ratio of TG to HDLc i.e. Log10 (TG/HDLc) ratio (where, TG=Triglyceride, TC=Total Cholesterol). Our results revealed that there was no significant difference in triglyceride, LDL, and total cholesterol concentration between the control (slim) and test (obese) subjects ($p>0.05$) but there was significant reduction in HDL concentration in the obese subjects when compared with the control subjects ($p<0.05$). In conclusion, obesity leads to an unfavorable lipid pattern, characterized by high TC, TG, LDL (“bad lipoprotein”) levels and low HDL (“Good lipoprotein”) levels that elevates the values of AIP and CRI I-II ratios which takes account of the proportion between pro-atherogenic and anti-atherogenic fractions hence considered more effective as diagnostic and prognostic alternatives in cardiovascular risk assessment unlike the conventional lipid parameters shown to be inadequate, especially in persons with intermediate risk.

CHAPTER ONE

1.0 INTRODUCTION

1.1 BACKGROUND OF STUDY

In Africa, despite the high prevalence of under nutrition, the prevalence of overweight and obesity is increasing at an alarming rate (WHO, 2008). With the risk of the double burden of malnutrition, there is a need to target prevention in developing countries (Ukegbu *et al.,* 2017). Children and young adults who are obese are more likely to have a clustering of cardiovascular risk factors such as dyslipidemia, hypertension and type-2 diabetes mellitus, which persist into adulthood (Verçoza *et al.,* 2009; Demerath *et al.,* 2003). Some studies have related the lifestyle of youths to risk factors for overweight and obesity. These include smoking, alcoholism, commuting to school by car/taxi, being from a rich family, as well as excess energy consumption (Baalwu *et al.,* 2011; Bouwari *et al.,* 2010). All over the world, the prevalence of overweight and obesity has been on the increase (WHO, 2008). Among university students in developing countries, the prevalence of overweight/obesity is reported to be 10–20.7% in Nigeria (Nwachukwu *et al.,* 2010; Ejike and Ijeh, 2012), 10.8–24% in South Africa (Cilliers *et al.,* 2006; Peltzer *et al.,* 2014), 11–37.5% in India (Seo *et al.,* 2009; Pengpid and Peltzer, 2014) and 20–30% in Malaysia (Gopalakrishnan *et al.,* 2012; Boo *et al.,* 2010).

Research done by Ukuegbu *et al.* (2017) reported that there is a higher obesity rates in females which may be attributed to gender differences whereby females accumulate more fat than their male counterparts. Furthermore, other researchers noted that cultural norms in Africa predispose more women to increased body weight since being fat is associated with beauty, affluence and evidence of good living and health (Ejike and Ijeh, 2012). It was reported that a gradual build-up of body weight at early adulthood could increase the chances of young adults becoming obese with advancement in age (Chinedu and Emiloju, 2014).

Results of the study done by Ukuegbu *et al.* (2017) showed that the prevalence of overweight and obesity is rising in this population group (young adults) and thus does not bode well for the burden of non-communicable diseases linked to obesity. The study shows the prevalence of obesity to be 6.5%, a value within the range (5–10%) reported by one study in Nigeria (Nwachukwu *et al.,* 2010). The figure is however higher than the 1.3% and 3% recorded for undergraduates in two different universities in Nigeria (Ejike and Ijeh, 2012; Chinedu and Emiloju, 2014) but consistent with the

6.0% and 9.8% recorded in cross-sectional surveys of a group of Nigerian and Ghanaian undergraduates, respectively (Ukegbu *et al.,* 2014; Mogre *et al.,* 2014). A higher prevalence (10.8-24%) has been reported for South African undergraduates (Cilliers *et al.,* 2006; Peltzer *et al.,* 2014). The prevalence of central obesity (8.9%) obtained in the study was lower than 20% reported in a university in Nigeria (Huxley *et al.,* 2010) but consistent with 9.8% obtained for Ghanaian undergraduates.

Obesity is a condition of excessive fat deposition in the body to an extent that adversely affects health (WHO, 1999). Studies have shown that obesity adversely alters the lipid profile which in turn poorly reflects on the cardiovascular health (Lichtash *et al.,* 2013; de Oliviera, 2010; Njelekela *et al.,* 2009). The burden of obesity has assumed pandemic proportions worldwide, with estimates of deaths due to overweight and obesity being 2.8 million each year (WHO, 2011).

The serum lipid profile is a test panel carried out to assess the quantitative deviation of various lipid and lipoprotein traits across a biochemical threshold in the serum or plasma (Bora *et al.,* 2015). Abnormalities in the lipid profile or dyslipidemia may manifest as one or more of the following: increased total cholesterol (TC), increased triglycerides (TGL), increased low density lipoprotein cholesterol (LDL-C) or decreased high density lipoprotein cholesterol (HDL-C) (Anne Carol Goldberg, 2008). Dyslipidemia is a major risk factor as well as a causal factor for cardiovascular diseases (most notably, coronary artery disease and stroke) (Truswell, 2010).

Association of lipid profile is reported with lifestyle, intra-abdominal adiposity, obesity and BMI (Omotoye and Fadupin, 2016). Studies have shown a direct relationship between increasing BMI and raised TC, LDL-C, and TG and an inverse correlation with HDL-C. This correlation between BMI and lipoprotein levels, especially LDL-C, has been proposed to be a strong contributing risk factor for cardiovascular diseases in obese individual (Hussain *et al.,* 2019).

Glew *et al.* (2001) studied Fulani pastoralists living in Jos plateau of north central Nigeria consuming a high fat diet, that have low serum total cholesterol and triglyceride concentrations in which the researchers concluded that a low energy intake, healthy life style and genetic features unique to the Fulani may help to account for why their diet does not result in elevated total or LDL-Cholesterol concentrations.

In a study done by Ugwuja *et al.* (2013) showed that male subjects were found to have more favorable plasma lipid profile (lower LDL- C and higher HDL- C) than the females. Although a significant percentage of the population were involved in social habits that may predispose them to

CVD, such as cigarette smoking (5.9%) and alcohol consumption (23.9%), few were involved in physical activities (9.3%). Plasma lipids were positively correlated with BMI and artherogenic indices, except for HDL- C, which was negatively correlated with artherogenic indices and LDL- C but positively correlated with BMI.

Study done by Edo and Enofe (2013) on apparently healthy staff of University of Benin Teaching Hospital (UBTH), Benin City using 202 females and 102 males found that High-density lipoprotein cholesterol dyslipidemias was found in 12.9% of female subjects and in 10.8% of male subjects. Total cholesterol dyslipidemia, low-density lipoprotein cholesterol dyslipidemia and triglyceride dyslipidemia were found in 51.0%, 26.3% and 4.9% of the subjects respectively.

Similarly, Abubakari *et al.* in their meta-analysis of the prevalence and time trends in obesity among adult West African populations noted that obesity in West Africa had doubled over the previous 15 years (114 %) as at 2008 and that women accounted entirely for this increase (Case and Menendez, 2009). Two of the reasons for the high prevalence of obesity in women include the fact that urban African women perceive the obese body type as what the ideal female body should be, another reason is that females who were nutritional deprived as children have a higher likelihood to be obese compared to men (Schneider *et al.,* 2010).

Hence, this study is designed to examine the lipid profile variation amongst a population of obese female students in the University of Benin.

1.2 JUSTIFICATION OF STUDY

Obesity has become a global problem of public health over the past few decades with its high prevalence (ranging between 11-15%) and medical burden in both developed and developing countries. Non-communicable diseases are the major health problems in the world today. Majority of premature deaths are due to these diseases, the most common cause been cardiovascular disease. Atherogenic index of plasma has been reported to be associated with cardiovascular diseases (Dobiasova and Frohlich, 2001). However, no study has yet systemically evaluated the association between Atherogenic index of plasma and obesity with its advantage in obesity prediction compared to conventional lipid components in the University of Benin.

1.3 AIM OF STUDY

This study is aimed at examining the castelli risk index and lipid profile variation amongst a population of young obese female students in the University of Benin.

1.4 RESEARCH QUESTIONS

I. Are there any variations in the BMI of the test and control subjects?
II. Does total Cholesterol concentration differ significantly between the test and control subjects?
III. Does total Triglyceride concentration differ significantly between the test and control subjects?
IV. Does low density lipoprotein concentration differ significantly between the test and control subjects?
V. Does high density lipoprotein concentration differ significantly between the test and control subjects?
VI. Does total Castelli risk index differ significantly between the test and control subjects?
VII. Does Atherogenic index of plasma differ significantly between the test and control subjects?

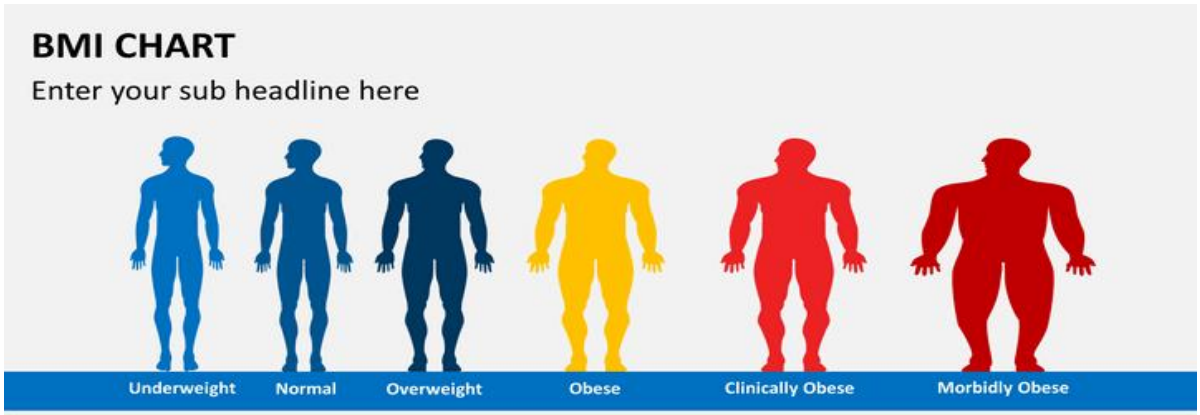
1.5 SPECIFIC OBJECTIVES

I. To determine the BMI variations in test and control subjects
II. To determine the concentration of total cholesterol using enzymatic assay method
III. To determine if Triglyceride concentration differ significantly between the test and control subjects
IV. To determine if Low density lipoprotein concentration differ significantly between the test and control subjects
V. To determine if high density lipoprotein concentration differ significantly between the test and control subjectS
VI. To determine the castelli risk index by calculating the ratio of plasma concentration of TG, LDL and non-HDL to HDL and examine if it differ significantly between the test and control subjects
VII. To determine the Atherogenic index of plasma (AIP) by calculating the logarithm (log) of the ratio of plasma concentration of TG to HDL and examine if it differ significantly between the test and control subjects.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 OBESITY, OVERWEIGHT AND BODY MASS INDEX



Risk of Associated Disease According to BMI and Waist Size

BMI		Waist < 40 in. (men) 35 in. (women)	Waist > 40 in. (men) 35 in. (women)
18.5 or less	Underweight	--	N/A
18.5 – 24.9	Normal	--	N/A
25.0 – 29.9	Overweight	Increased	High
30.0 – 34.9	Obese	High	Very High
35.0 – 39.9	Obese	Very High	Very High
40 or greater	Extremely Obese	Extremely High	Extremely High

Fig. 1: Obesity, Overweight on Body Mass Index Chart (www.sketchbubble.com).

Overweight and obesity are common conditions that are defined as the increase in size and amount of fat cells in the body. Overweight and obesity are caused by many factors including behaviors like eating patterns, lack of sleep or physical activity, and some medicines, as well as genetics and family history. Obesity is a chronic health condition that raises the risk for heart disease and is linked to many other health problems, including type 2 diabetes and cancer. Overweight and obesity can lead to serious health issues for people of all ages.

Healthcare providers use body mass index (BMI) to screen for overweight and obesity in adults. Body Mass Index (BMI) is an important anthropometric index that is commonly used for body fat storage status assessment and is used for body fat determination (Paknahad *et al.,* 2008). It is measures body fat using the height and weight parameters, hence defined as the body mass (in kilograms) divided by the square of the body height (in meters) and expressed in units of kg/m^2, but there is more to obesity

than BMI. Unhealthy lifestyle habits, such as not getting enough physical activity and eating high-calorie, low-nutrient foods and beverages, can raise your risk of overweight and obesity. Some people find that their weight goes up when they start taking medicine for another health condition such as diabetes, depression, or high blood pressure.

Another indicator is the waist circumference or abdominal adiposity, which is associated with excess abdominal fat and total body fat. Abdominal adiposity is defined as a waist circumference of ≥ 102 cm for men and ≥ 88 cm for women (Odili *et al.,* 2014). The risk of cardiovascular disease (CVD) and non-insulin dependent diabetes is high in men and women with abdominal adiposity (Zimmet *et al.,* 2005). Okosun *et al*. (1999) in their study of six populations of West African descent including Nigeria reported that the prevalence of hypertension was closely linked to abdominal adiposity. They further showed that the prevalence of abdominal adiposity and hypertension was more common in women than in men.

Body mass index (BMI) can be calculated as weight (kg) divided by the square of height (m^2) (Odili *et al.,* 2014). BMI is frequently used to categorize individuals as underweight, normal, overweight and obese (WHO Working Group, 1986). BMI was divided into four categories according to the WHO cut-off points with corresponding interpretations (<18.5kg/m^2, underweight; 18.5 to 24.9kg/m^2, normal weight; 25.0 to 29.9kg/m^2, overweight; ≥30.0kg/m^2, obese respectively).

The World Health Organization (WHO) regards obesity as the accumulation of excess fat that poses a direct risk to health (Al Shehri *et al.,* 2013). Results from comprehensive studies are now causing great concern as they are showing a global increase in the rates of overweight children worldwide, including Saudi Arabia (Al Shehri *et al.,* 2013; Wang and Lobstein, 2006; Misra and Khurana, 2008; Kelishadi, 2007). It was estimated that by the year 2020, three quarters of all deaths would be attributable to non-communicable diseases, of which obesity is a major risk factor (WHO, 2008).

Studies of anthropometric measures and prevalence of overweight and obesity among young adults in African countries particularly in Nigeria are limited (Odili *et al.,* 2014). Olufemi and Abiodun (2013) in their study of prevalence of overweight and obesity in an institutionalized multiethnic based male adult sample, reported the prevalence of overweight and obesity among Lagos State University undergraduate male adults to be 14.0% (BMI: 25-29) and 2.3% (BMI: 30-40) respectively. Akinpelu *et al.* (2008) found that prevalence of overweight was 0-8.1% and 1.3-8.1% in males and females respectively while that of obesity was 0-2.7% and 0-1.9% still in males and females respectively in a sample of Nigerian adolescents in an urban community aged 12-18 years.

Similarly, another study of a small sample of students in the University of Port Harcourt reported a prevalence of overweight and obesity of 6.8% respectively (Buowari, 2010). One study found an alarmingly high prevalence rate of 21% among undergraduate students in the halls of residence of the University of Nigeria, Nsukka campus (Onyechi and Okolo, 2018).

Several studies have confirmed the link between dyslipidemia in the setting of obesity and high BMI (Milyani and Al-Agha, 2019). Stefanig *et al.* (1996) demonstrated that BMI relates directly to total cholesterol, VLDL (very low-density lipoprotein) and LDL-C (Low-density lipoprotein-cholesterol), though; the present findings revealed that normal weights reflect a low risk cardiovascular disease (Shuaibu *et al.,* 2014). However, as the BMI increases, deposition of lipoprotein especially LDL-C increases in the cardiovascular system leading to their concomitant atherosclerosis, a potential cardiac destroyer (Shuaibu *et al.,* 2014).

Cardiovascular diseases (CVDs) are an example of the most predominant non-communicable diseases, widely known as the number one cause of mortality worldwide. They are associated with both obesity and dyslipidemia (Pituelli Saurez *et al.,* 2008; American Academy Pediatrics, 1998; Nascimento *et al.,* 2012). The most common dyslipidemias that have been associated with obesity are elevated triglycerides (TGs) and low-density lipoprotein (LDL) and decreased high-density lipoprotein (HDL) levels (Milyani and Al-Agha, 2019).

2.2 LIPID METABOLISM AND THE LIVER

Lipids, represented by phospholipids, cholesterol, triglycerides (TG) and fatty acids, are considered essential to the human body, both by making up of the basic structure of cell membranes (phospholipids), and by acting as a precursor to steroid hormones, bile acids and vitamin D, as well as being a constituent of cell membranes, acting on the fluidity of the latter and in the activation of the enzymes located there (cholesterol) (Sociedade Brasileira de Cardiologia, 2007).

Lipids are one of the necessary components which control cellular functions and homeostasis (Ghadir *et al.,* 2010). Liver plays an essential role in lipid metabolism, several stages of lipid synthesis and transportation. Therefore, it is reasonable to expect an abnormal lipid profile in those with sever liver dysfunction. There is prominent decline in plasma cholesterol and triglyceride (TG) levels in patients with severe hepatitis and hepatic failure because of reduction of lipoprotein biosynthesis. For reduced liver biosynthesis capacity, low levels of TG and cholesterol is usually observed in chronic liver diseases (Halsted, 2004).

Obesity and obesity-associated diseases are major issues in the twenty-fist century in adults as well as children and, as a result, fatty liver is considered to be the hepatic component of the metabolic syndrome (Angulo, 2002). Indeed, sedentary work and reduced physical activity combined with high calorie ingestion lead to morbid obesity (Kopelman, 2002). In the westernized countries, diets are highly caloric and particularly rich in saturated fatty acids (FAs) and monosaccharides (sucrose and fructose), thus resulting in a positive energy balance with increasing body mass (Bray *et al.,* 2004; Bray, 2004). The liver is an altruistic metabolic organ that plays a central role in synthesizing molecules that are utilized to support homeostasis, in converting molecules of one type into another, and in regulating energy balances (Canbay *et al.,* 2007). The liver's major metabolic functions can be subdivided into several major categories (e.g., carbohydrate, protein and lipid metabolism). Indeed, the liver has a central role in various aspects of lipid metabolism. First, the liver produces bile, which is required for efficient intestinal fat absorption. Secondly, biliary secretion of cholesterol in the form of bile salts and phospholipids which is of major importance for the lipid metabolism. Additionally, the liver is not only the major producer of plasma lipoproteins, but also the major site of clearance of these circulating proteins (Canbay *et al.,* 2007). Due to the increasing prevalence of obesity and its association with non-alcoholic fatty liver disease (NAFLD) as the most common liver disease in the developed countries experimental research has emerged to understand liver lipid metabolism (Angulo, 2002). For example, NAFLD may be caused by increased uptake of lipids by the liver, increased hepatic synthesis of FAs, decreased b-oxidation and/or decreased synthesis of very low density lipoproteins (VLDLs) (Canbay *et al.,* 2007). Detailed knowledge of alterations in the hepatic lipid metabolism might help to develop new therapeutic strategies in fatty liver diseases (FLD) such as NAFLD, or alcoholic fatty liver disease (AFL). In addition to injuries that are induced due to fatty liver, fat is required for the proliferation of hepatocytes and for liver regeneration (Canbay *et al.,* 2007).

Lipid metabolism involves several pathways that are at least in part, inter-dependent and 'cross-regulated'. Fatty acids are the most commonly stored and circulating forms of energy, and triacylglycerols are the most common non-toxic form of fatty acids. Fatty acids/triacylglycerols may originate from four sources (pool input): De novo lipogenesis, cytoplasmic triacylglycerol stores, fatty acids derived from triacylglycerols of lipoprotein remnants directly taken up by the liver, and plasma non-esterified fatty acids (NEFA) released by adipose tissue (Nguyen *et al.,* 2008).

2.2.1 DIETARY LIPIDS

Lipid-rich diets with an increased content of saturated fats are major causes of metabolic disorders such as obesity, diabetes, coronary heart disease and FLD (fatty liver disease) (Canbay *et al.,* 2007). Most of the lipids found in food are provided in the form of triacylglycerols (TAGs; triglycerides) that represent fatty esters of glycerol. Other types of dietary lipids are cholesterol and phospholipids (Canbay *et al.,* 2007). As is well known, some fats are essential to our health. Fatty acids fall into two categories: saturated FAs and unsaturated FAs. Saturated FAs are found in meat, eggs, and dairy products and are not essential to health; in fact, if consumed excessively, they can be difficult to metabolize (causing weight gain) and may lead to serious sequelae, such as cardiovascular disease (Clarke, 2001; Timlin *et al.,* 2005). Unsaturated FAs include mono-unsaturated FAs and poly-unsaturated FAs. Mono-unsaturated FAs are considered to be the healthiest types of FAs. They are mainly found in olive oil, rapeseed oil, nuts and seeds. The high consumption of olive oil in the Mediterranean population is considered to be one of the reasons why these countries have a lower prevalence of coronary heart disease. This is because mono-unsaturated FAs help to reduce harmful low-density lipoproteins (LDLs) that are closely associated with coronary heart disease (Canbay *et al.,* 2007). Recent investigations indicate that poly-unsaturated FAs may reduce the amount of protective high-density lipoproteins (HDLs) as well as the harmful LDLs (Clarke, 2001). However, the group of polyunsaturated FAs comprises two very important essential fatty acids, i.e., ω-6 FAs (e.g., linoleic acid, sunflower oils) and ω-3 FAs (e.g., a-linolenic acid) found in oily fish. Essential FAs regulate mental health, growth, and vitality and are believed to assist the transport and uptake of oxygen throughout the body (Clarke, 2001). However, once the consumption of energy considerably exceeds the combustion of calories, excess energy is conserved in the form of fat and, in particular, as TAGs within the adipose tissue, hence leading to obesity (Reddy and Rao, 2006).

2.2.2 DIGESTION AND ABSORPTION OF DIETARY LIPIDS

The assembly of intestinal lipoprotein is essential for the absorption of dietary lipids. Absorption consists of three major steps and is defined as the transport of dietary lipids from the intestinal lumen to the plasma. First, dietary lipids are emulsified and hydrolyzed within the intestinal lumen. Second, hydrolyzed fat components are absorbed by intestinal epithelial cells. Third, fat is re-synthesized in intestinal epithelial cells and packaged into lipoprotein particles (chylomicrons) (Canbay *et al.,* 2007).

FAs and TAGs are both constituents of dietary lipids. However, both can be synthesized from glucose and other carbon sources within the liver (Canbay *et al.,* 2007). Indeed, hepatocytes are the main site

of FA synthesis and also play a central role in the distribution of lipids to other organs. Adipocytes are a major recipient of dietary as well as synthesized TAGs within in liver. During lipolysis, FAs are released from the adipose tissue and provide an important source of substrate for hepatocellular TAG synthesis (Bradbury and Berk, 2004). The neighboring omental and mesenteric adipose tissue can directly release FAs via lipolysis into the portal vein and provide therefore, a rapid source of energy (Bradbury and Berk, 2004).

2.2.3 TRANSPORT MECHANISMS FOR FATTY ACIDS

TAGs are emulsified in the small intestine by bile salts and hydrolyzed to FAs and 2-monoacylglycerol (2-MAG) by pancreatic lipase (PL) (Canbay *et al.,* 2007). Pancreatic enzymes also release FAs from other dietary lipids such as cholesterol esters or phospholipids. FAs are absorbed by the intestinal epithelium (Bradbury and Berk, 2004; Pohl *et al.,* 2004a). The absorption of FAs occurs in the distal jejunum and ileum, and the FAs enter the lymphatic system and bloodstream as chylomicrons via facilitated transport (Canbay *et al.,* 2007). The cellular uptake of FAs is a critical step in the movement of lipids from the intestinal tract to the liver. Recent investigations indicate that FAs enter cells by two distinct pathways: the protein-mediated uptake and the passive, transmembraneous flip-flop of protonated FAs. FAs are highly insoluble in aqueous media, and are kept in solution by binding to albumin. At least 90% of the uptake of FAs into hepatocytes and adipocytes occurs by this process (Bradbury and Berk, 2004; Pohl *et al.,* 2004a). Capillary-bound lipoprotein lipase (LPL), produced by liver, heart, adipose, and other tissues mediate the lipolysis of lipoprotein-bound TAGs on the luminal surface of capillary endothelial cells, or within adipocytes during hormone-sensitive lipase (HSL)-mediated lipolysis of intracellular TAGs (Merkel *et al.,* 2002; Heeren *et al.,* 2002). This may be associated with higher proportions of FA movement across the plasma membrane into or out of cells occurring by the passive non-saturable pathway (Pohl *et al.,* 2004a; Pohl *et al.,* 2004b; Hubbard *et al.,* 2006). In the past, it was believed that FAs enter cells by diffusion throughout the bilayer lipid membrane. However, the uptake mechanism of FAs is complex and sophisticated. These mechanisms include dissociation of FAs from albumin or intestinal micelles, transport across the plasma membrane, binding to intracellular proteins, and/or esterification to acyl-CoA (Begriche *et al.,* 2006). Several plasma membrane protein transporters are involved in the FA uptake process (Fig.1) (Canbay *et al.,* 2007). However, the main translocation process across the plasma membrane due to the saturable transport process involves members of the fatty acid transport protein (FATP) family (Pohl *et al.,* 2004b; Hubbard *et al.,* 2006; Ehehalt *et al.,* 2006; Goldberg and Ginsberg, 2006).

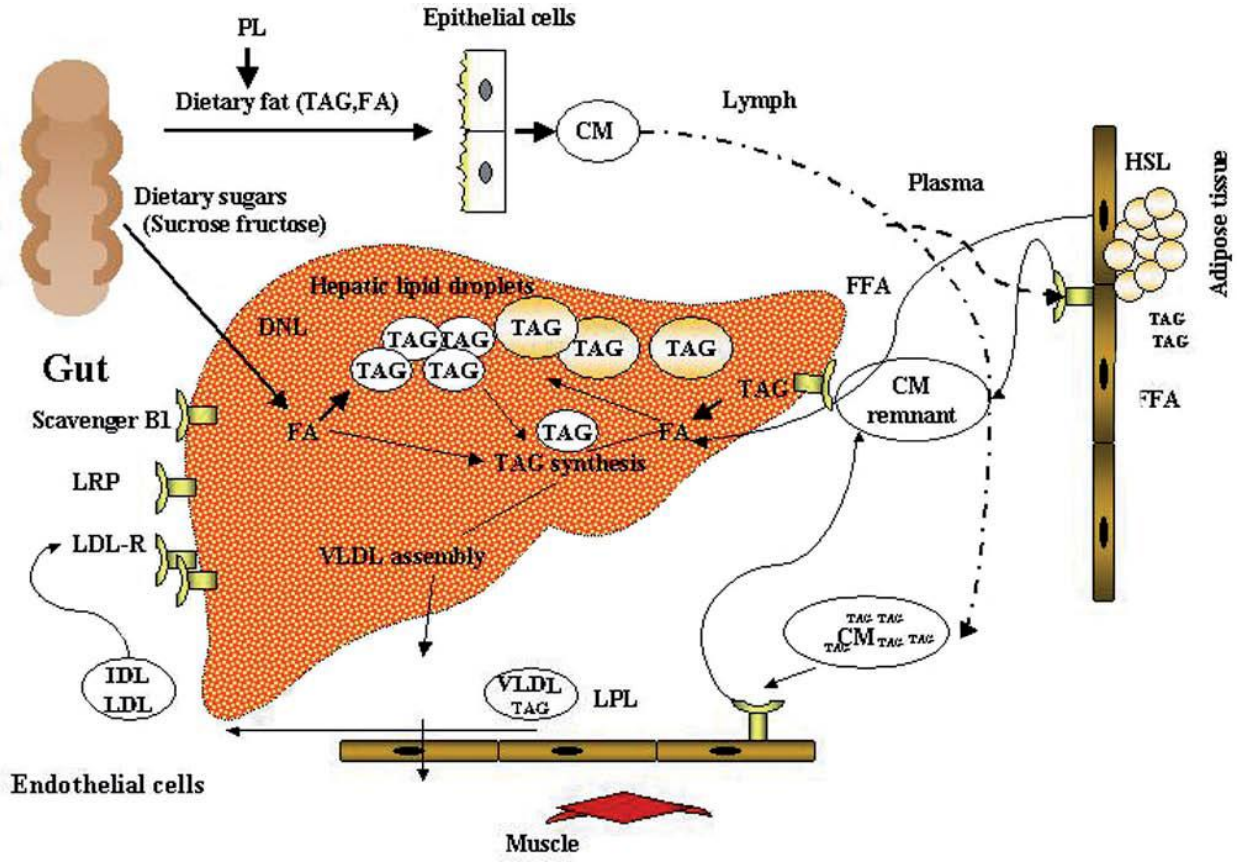


Fig. 2: Cellular fatty acid uptake and possible mechanisms involved in TAG storage by hepatocytes.

(Transport proteins, e. g., fatty acid transport proteins (FATP), fatty acid translocase (FAT/CD36), caveolin-1 and fatty acid binding protein (FABP) are involved in FA-uptake, in particular, of long-chain FAs (LCFAs). Alterations in these transport proteins potentially result in steatotic hepatocytes. Increased insulin and glucose levels upregulating FA synthesis and thus triacylglycerols (TAGs) through the induction of sterol regulatory element-binding protein 1c (SREBP-1c), carbohydrate responsive element binding protein (ChREBP), peroxisome proliferator-activated receptor g (PPARg), and forkhead box 01 (Fox01). These factors increase the expression of lipogenic enzymes such as acetyl-coenzyme A carboxylase (ACC) and fatty acid synthase (FASN), which results in increased hepatic FAs and causes excessive hepatocyte TAGs storage) (Canbay *et al.,* 2007).

When expressed, they facilitate the uptake of long-chain (C_{12}–C_{20}) FAs. FATP is highly expressed in hepatocytes and adipocytes that reveal high-level FA uptake for both metabolism and storage. So far, six members of this protein family (FATP1 to 6) have been identified; their isoforms show distinct organ-specific distribution patterns (Ehehalt *et al.,* 2006; Frohnert and Bernlohr, 2000). For example, FATP1 is found in adipose tissue and in the heart. In contrast, FATP2 and FATP5 are expressed in the

liver, while FATP4 is expressed in the intestine (Pohl *et al.,* 2004b; Stahl *et al.,* 2001). Overexpression of FATP5 in cultured cells has been shown to increase FFA uptake (Ehehalt *et al.,* 2006). Thus, FATP5 is an important membrane protein involved in FA accumulation by the liver (Hubbard *et al.,* 2006; Goldberg and Ginsberg, 2006). In contrast, in mice and humans, FATP4 serves as the major FA transporter on the apical surface of enterocytes (Pohl *et al.,* 2004b).

2.2.4 INTESTINAL EPITHELIAL CELLS: RESYNTHESIS OF TAGS AND CHYLOMICRONS

Absorbed FAs are activated to fatty acyl CoA that, combined with 2-MAG, forms diacylglycerol, which reacts with the second fatty acyl CoA thus leading to the resynthesis of TAGs (Bradbury and Berk, 2004). Dietary lipids such as TAGs, cholesterol esters, phospholipids, absorbed fat-soluble vitamins, and lesser amounts of cholesterol and FAs are packed into nascent chylomicrons (lipoprotein particles). The major protein component of the chylomicrons, apoprotein (apo) B-48, is synthesized by epithelial cells (Tennyson *et al.,* 1989). Interestingly, both apoB-48 and apoB-100 are encoded by the same gene (Canbay *et al.,* 2007). In contrast, the main protein component of VLDLs, apoB-100, is produced in hepatocytes (Tennyson *et al.,* 1989). Nascent chylomicrons also contain apoA-I and apoA-IV, as successively secreted by intestinal epithelial cells into the lymphatic channels, thereby passing the liver to enter the systemic circulation via the thoracic duct (Timlin *et al.,* 2005; Bradbury and Berk, 2004).

Chylomicrons are highly enriched with TAGs and reach the general circulation within one to two hours after consuming a meal and continue to circulate for many successive hours as the dietary lipids are digested and adsorbed (Timlin *et al.,* 2005). In the circulation process, chylomicrons lose their apoA-I and apoA-IV components, but receive additional apoproteins (apoE and apoC-II) from HDLs, thereby converting them from nascent to mature chylomicrons (Bradbury and Berk, 2004). Lipoprotein-bound apoC-II activates LPL, which results in the digestion of chylomicron-bound TAGs, with the subsequent release of FAs for local cellular uptake. The activity of LPL differs from that of pancreatic lipase (PL). Specifically, LPL releases three FA molecules and one glycerol from each TAG, while PL yields only two FAs, and one molecule of 2-MAG (Bradbury and Berk, 2004). In adipose tissue, the activity of LPL reaches its highest level after finishing a meal, which correlates with the level of circulating chylomicrons (Merkel *et al.,* 2002; Heeren *et al.,* 2002). Glycerol released during LPL-mediated lipolysis of lipoprotein TAGs is taken up by the liver for glycolysis or gluconeogenesis (Lavoie and Gauthier, 2006). After the digestion of chylomicron TAGs, residual so-

called chylomicron remnants can be recognized by their apoE content that interacts with the LDL receptor, LDL receptor-related protein (LRP) and scavenger receptor B-1 leading to endocytotic uptake by hepatocytes (Goldberg and Ginsberg, 2006).

Chylomicron remnants are thereupon digested within lysosomes, and their glycerol, FAs, cholesterol, amino acids, and phosphate residues are recycled by incorporation into new VLDL particles for export (Canbay *et al.,* 2007).

2.2.5 FATTY ACID OXIDATION

FA oxidation is the predominant source of energy during fasting periods (Canbay *et al.,* 2007). Under such conditions, the glucagon level rises, thus mobilizing FAs from adipose tissue, which successively leads to FA influx into the liver. Therefore, FA oxidation plays a pivotal role in fat metabolism and disturbances in FA oxidation contribute to the state of fatty liver (Canbay *et al.,* 2007). After conversion to their CoA derivatives, FA oxidation occurs mainly within mitochondria and peroxisomes (Reddy and Rao, 2006). The key enzymes of these oxidation systems are regulated by PPAR-α (peroxisome proliferator-activated receptor). PPAR-α knockout in mice fail to upregulate FA oxidation and it leads to development of fatty liver (Hashimoto *et al.,* 2000). In line with these observations, PPAR-α agonists prevent steatosis induced by diets that are devoid of methionine and choline (Ip *et al.,* 2003). Unlike long-chain FFAs, short-chain and medium-chain FFAs freely enter mitochondria, without requiring carnitine palmitoyltransferase I (CPT-I), an outer membrane enzyme whose activity is inhibited by malonyl-CoA. Indeed, malony-CoA regulates the entry of FAs into mitochondria, and its inhibition increases FA oxidation and reduces TAGs in hepatocytes (Goldberg and Ginsberg, 2006; Savage *et al.,* 2006). After a carbohydrate-rich meal, high glucose and insulin levels cause hepatic FA synthesis (Begriche *et al.,* 2006). Glycolysis generates pyruvate, which is transformed by mitochondria into acetyl-CoA and then into citrate. In the cytosol, citrate regenerates acetyl-CoA, which is used for malonyl-CoA and then FA synthesis, as catalyzed by fatty acid synthase (FAS). High malonyl-CoA levels inhibit CPT-I and decrease FA oxidation (Begriche *et al.,* 2006; McGarry, 1980). Therefore, FFAs are not degraded, but are instead shuttled towards the formation of TAGs that are secreted as VLDL (Canbay *et al.,* 2007).

2.3 DYSLIPIDAEMIA

Dyslipidaemia refers to abnormal concentrations of lipids and/or lipoproteins in the blood and may result from defects in the production, transport and/or degradation of lipoproteins (Couch and Daniels, 2008). It may be classified as primary or secondary to other diseases, such as HIV infection, insulin resistance or chronic kidney disease (Couch and Daniels, 2008). The reported prevalence of dyslipidaemia in adolescence ranges from 19.7 to 34.3%, depending on the lifestyle and the cut-off values applied in the definition (Tomeleri *et al.,* 2015; Yang *et al.,* 2012; Taheri *et al.,* 2015).

Two separate school-based studies, one in India and the other in Brazil, reported dyslipidaemia prevalence rates as high as 62.3% and 66.7%, respectively (Nijaguna *et al.,* 2015; de Carvalho, 2007). The results of several studies indicate that the prevalence of dyslipidaemia in adolescence is increasing in parallel with the dramatic rise in prevalence of adolescent obesity in various countries (Kuo *et al.,* 2014; Ding *et al.,* 2016; Agirbasli *et al.,* 2011). Some recent studies involving children and adolescents revealed that high body mass index (BMI) is closely associated with dyslipidaemia (Horta *et al.,* 2009; Meral *et al.,* 2015). Dyslipidaemia is a modifiable, metabolic risk factor for development of cardiovascular disease (CVD) (Couch and Daniels, 2008) and insulin resistance (Asato *et al.,* 2006). It is estimated that 40-55% of children with dyslipidaemia will have hyperlipidemia during adulthood (Hatami *et al.,* 2012). Elevated levels of serum triglyceride and low-density lipoprotein cholesterol (LDL-C) are linked metabolically and this combination, is known as “atherogenic dyslipidaemia”, a strong risk factor for CVD (Onyiriuka *et al.,* 2021). Low HDL-C level was the most common abnormality in dyslipidaemia (Onyiriuka *et al.,* 2021).

2.4 CASTELLI RISK INDEX (CRI) AND ATHEROGENIC INDEX OF PLASMA (AIP)

It has been suggested that the different combinations of these lipid profile parameters can be used to identify such high risk individuals. Castelli Risk Index (CRI) and Atherogenic Index of Plasma (AIP) are the ratios we studied in predicting the risk of CAD. These are the calculated fractions which can be used in the clinical setting for assessing the risk of cardiovascular disease beyond the routinely done lipid profile. Atherogenic Index of Plasma (AIP) is based on two important parameters TG and HDLc, both of which are independent risk factors for CAD. Castelli Risk Index (CRI-I) calculated as (TC/HDLc) and (CRI-II) as (LDLc/HDLc) is another fraction which involves independent risk factors for CAD. Thus, the present study was conducted with the objective of assessing the significance of lipid ratios like Castelli Risk Index (CRI) and Atherogenic Index of Plasma (AIP) in identification of at-risk individuals for CAD beyond the routinely done lipid profile especially in insufficient resource situations. CRI reflects the formation of coronary plaques with a diagnostic value as good as the

determination of total cholesterol. CRI is based on three important lipid profile parameters i.e. TC, LDLc and HDLc and it is categorized into two; CIR-1 and CIR-11.

- CRI-I: CRI-1 is the ratio of TC and HDLc. i.e. CRI-I= TC/HDLc,
- CRI-II: CRI-II is the ratio of LDLc to HDLc. i.e. CRI-II= LDLc/HDLc (Adedokun et al., 2017).

AIP is a logarithmically transformed molar ratio of TG to HDLc. AIP= Log10 (TG/HDLc) ratio. It Was proposed by Dobiasova and Frohlich in 2001. Atherogenic index of plasma (AIP) is a novel index composed of triglycerides and high-density lipoprotein cholesterol. It has been used to quantify blood lipid levels and commonly used as optimal indicator of dyslipidemia and associated diseases (e.g., cardiovascular diseases) (Zhu *et al*., 2018).

CHAPTER THREE

3.0 MATERIALS AND METHOD

3.1 MATERIALS

The following are the materials and equipment used for this research:

i. Digital weighing scale
ii. ELISA kits
iii. Centrifuge
iv. Test tubes/racks
v. Timer
vi. Cotton wool
vii. Plain sample bottle
viii. Lithium heparin sample bottles
ix. Questionnaires
x. 70% alcohol
xi. Syringes and needles
xii. Hand Gloves
xiii. Tourniquet.

3.2 METHOD

A) LIPID PROFILE

i) Total Cholesterol Assay by Enzymatic Method

To 1000 µL of reagent present in test tubes labeled blank, sample tubes, control tube and standard tube 10 µL of' water, sample, control serum and standard was added respectively, mixed thoroughly and incubated for 10 minutes at 37 °C. The absorbance of test. standard and control were read against reagent blank using a spectrophotometer at 500 nm (Allain *et al.,* 1974).

Calculation:

Concentration of cholesterol in sample = ΔA_{sample} X Concentration of Standard $\Delta A_{standard}$

ii) Triglyceride Assay by Enzymatic Method

To 1000 µL of reagent present in test tubes labeled blank, sample tubes, control tube and standard tube 10 11L or water, sample, control scrum and standard was added respectively mixed thoroughly

and incubated for 10 minutes at 37°C. The absorbance of test, standard and control were read against reagent blank using a spectrophotometer at 500 nm (Buccolo and David. 1973).

Calculation:

Concentration of Triglyceride = $\Delta \mathbf{A}_{sample}$ x Concentration of Standard (mg/di) $\Delta \mathbf{A}_{standard}$

HDL-Cholesterol Assay by Precipitation

Stage 1: Precipitation

Test tubes were labeled sample and control and 200 11L or sample and 200 11L of control scrum were added to the tubes respectively. 500 11L of precipitant was added to all the tubes and were mixed thoroughly. The tubes were Jell to stand for 10 minutes at room temperature and spun at 4000 rpm for 10 minutes (Benzie, 1979).

Stage 2: Enzymatic Assay

A 50 µL each of water. supernatant of test. supernatant of control sera and standard solution were added to the appropriate lest tube labeled reagent blank, sample, control and standard tube respectively. 1000 µL of reagent were added to all the tubes, mixed thoroughly and incubated for 10 minutes at 37°C and read at 500 nm (Benzie, 1979).

Calculation:

HDL Cholesterol:

Concentration HDL Cholesterol in supernatant = ΔA_{sample} X Concentration of Standard (mg/dl) $\Delta A_{standard}$.

LDL Cholesterol (Friedewald *et al.,* 1972)

LDL Cholesterol = Total Cholesterol - (Triglyccridcs/5-HDLCholesterol).

B) CASTELLI RISK INDEX (Vascular Risk Index).

Total cholesterol/HDL cholesterol and LDL/HDL cholesterol ratios

Subjects were placed on 8-h fasting before fasting blood tests were performed.

The lipid-ratio-related studies included:

Total cholesterol = LDL + HDL + (triglycerides/5).

Total CHL /high-density lipoprotein (HDL) cholesterol (Dobiásová, 2004).

LDL/HDL cholesterol (Walldius and Junger, 2006).

C) ATHEROGENIC INDEX OF PLASMA (AIP).

The Atherogenic Index of Plasma was calculated as follows:

Atherogenic Index of Plasma (AIP) = log TG/ high-density lipoprotein (HDL) (Dobiásová, 2004).

3.3 STUDY AREA

This study was carried out across Ovia North East local government area and in the University of Benin community.

3.4 STUDY POPULATION

A total of 120 apparently healthy obese and non-obese subjects (Females) were employed in this study with age range between 18 and 30 years.

3.5 INCLUSION CRITERIA

This study incorporated apparently healthy obese subjects with age range between 18 and 26 years.

3.6 EXCLUSION CRITERIA

Subjects with any form of cardiovascular abnormalities, pulmonary disorders, Psychiatric illness, aging individuals, pregnant and lactating women, women on contraceptives or any other medications that may affect lipid profile were excluded from the study.

3.7 ETHICAL CONSIDERATION

Approval and clearance for this study was sought and obtained from Ministry of Health, Benin City, Edo State. (HM.1208/88).

3.8 STUDY DESIGN

The study protocol was explained to the subjects and oral and written informed consent was obtained to participate in the study.

3.8.1 PHASE 1 (Administration of questionnaire)

Anthropometric data were collected by oral interviews and questionnaires also other related data collection equipment.

3.8.2 PHASE 2 (Identification of relevant subjects for the study)

A total of 60 subjects were employed in this study; categorized into 2 groups:

1. Control group (A): Consists of 30 female subjects who were recorded as “normal weight”
2. Test group (B): Consists also of 30 female subjects recorded to be “obese”.

Body Mass Index (BMI) was calculated to ascertain the condition of the patient using the weight and height parameter of individuals. It is Mathematically represented as the weight in kilograms divided by the square of the height in meters (kg/m2). BMI within 18.5-24.9 kg/m2 was considered as normal weight while a BMI of ≥30 kg/m2 was recorded as obesity according to WHO, (2021).

3.8.3 SAMPLE COLLECTION AND LABORATORY ANALYSIS

After an overnight fast (minimum 12 hours), 5ml of blood was collected through venepuncture via the median cubital vein into lithium heparin bottles following standard phlebotomy guidelines.

Samples were centrifuged for 15mins at 4000rpm. The supernatant was separated into plain bottles and refrigerated at - 20°C for lipid profile analysis. All analysis was carried out in the university of Benin and the university of Benin teaching hospital.

3.9 STATISTICAL ANALYSIS

The statistical package used for the experiment was graph pad prism version 8.0. and Data obtained were presented as mean ± standard error of the mean (SEM) and analyzed for Statistical significance by student T-test. Results of the analysis was represented in graphs. Confidence limit was set at 95% and probability value of $p < 0.05$ was regarded as statistically significant.

CHAPTER FOUR

4.0 RESULTS

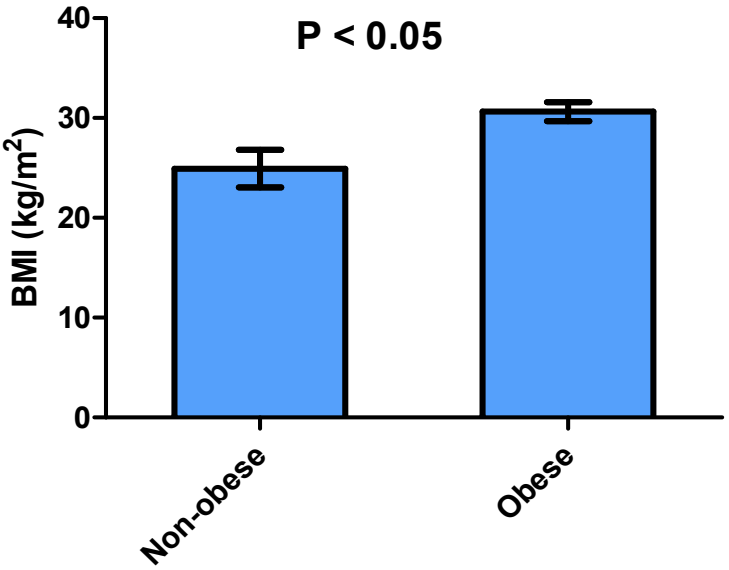


Fig I: Body Mass Index variations in obese and non-obese individuals.

The result shows a significant difference in obese compared with non-obese individuals. P<0.05; N= 30 ± SEM.

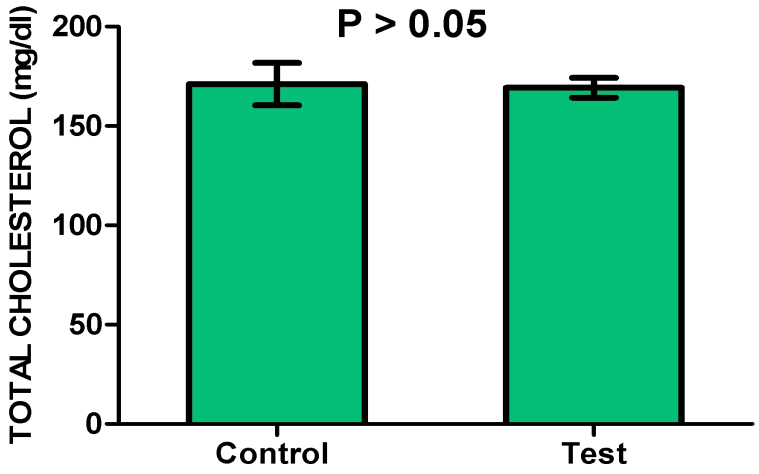


Fig. II: Total cholesterol concentration between Obese and Non-Obese subjects.

The result showed that there was no significant difference in total cholesterol level between test group compared with control. P>0.05; N= 30 ± SEM.

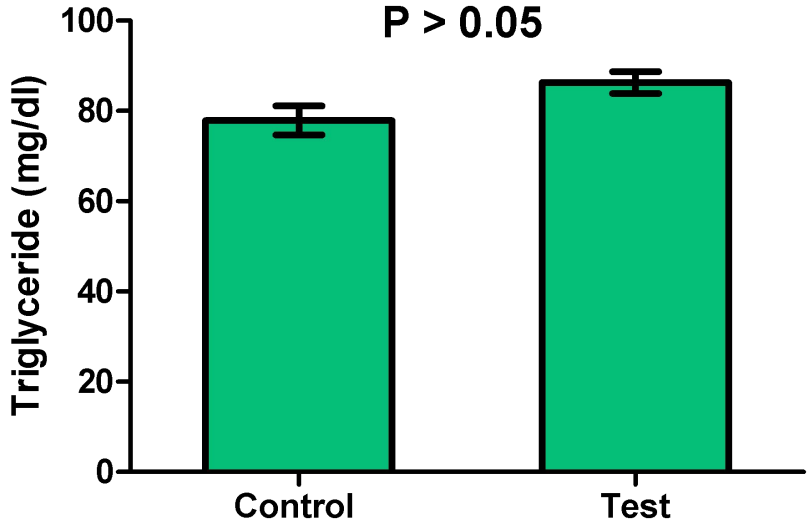


Fig. III: Triglyceride concentration between Obese and Non-Obese subjects.

The result showed that there was no significant difference in test compared with control. P>0.05; N= 30 ± SEM

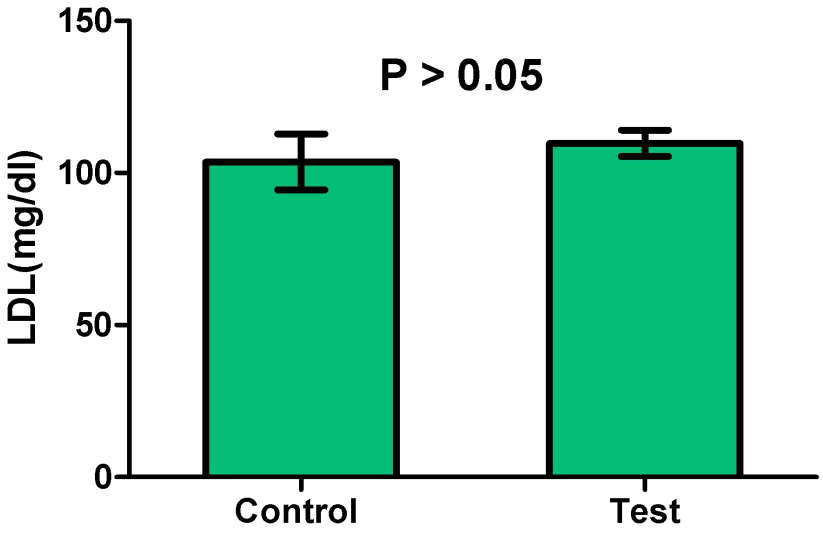


Fig. IV: LDL Concentration between Obese and Non-Obese subjects.

The result showed there was no significant difference in test compared with control. P>0.05; N= 30 ± SEM.

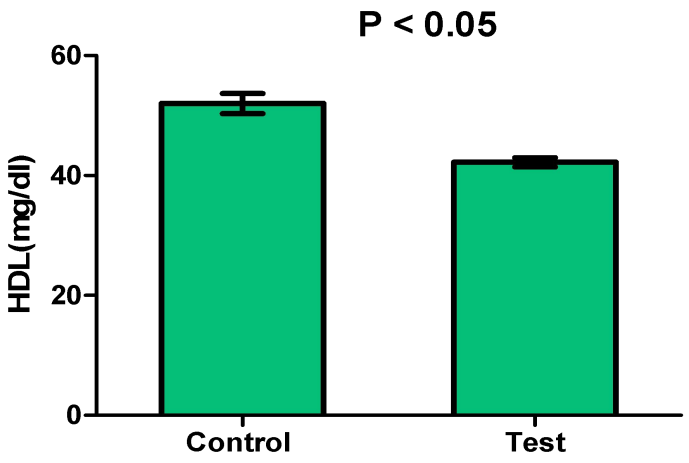


Fig. V: HDL Concentration between Obese and Non-Obese subjects.

The result showed there was a significant decrease in test compared with control. P<0.05; N= 30 ± SEM.

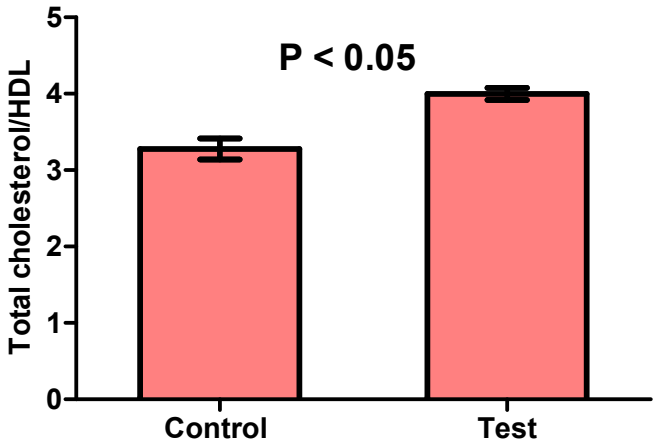


Fig VI: Total cholesterol-HDL ratio (CRI-I) variations in obese and non-obese individuals.

There was a significant increase in test compared with control. P<0.05; N= 30 ± SEM.

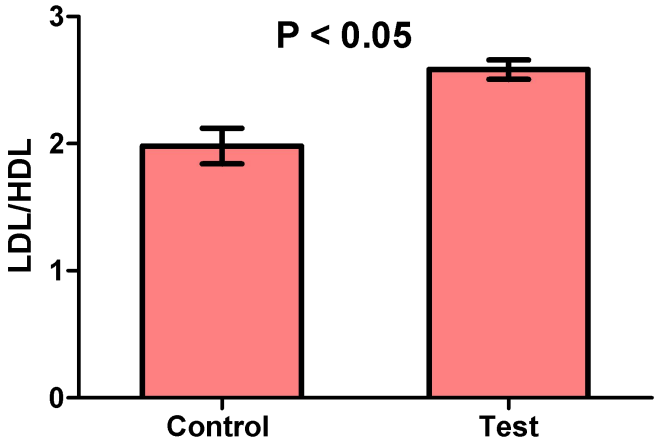


Fig VII: *LDL-HDL ratio* (CRI-II) between Obese and Non-Obese subjects.

The result showed there was a significant increase in test compared with control. P<0.05; N= 30 ± SEM.

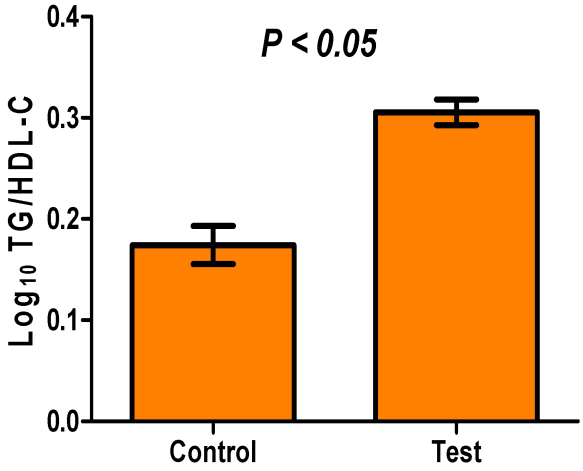


Fig VIII: Atherogenic index of plasma in obese young individuals.

The result showed there was a significant increase in obesity over control. P<0.05; N= 30 ±SEM

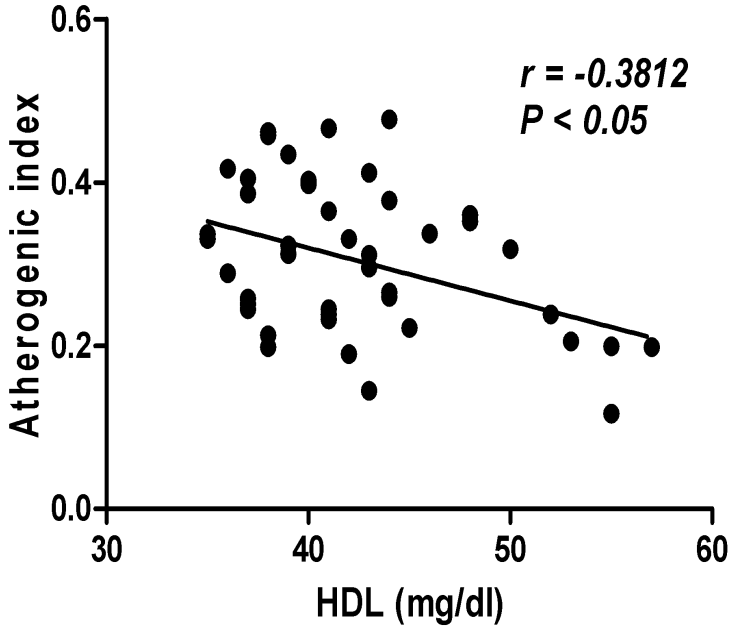


Fig IX: Relationship between atherogenic index and high density lipoprotein in obese subjects.

The result showed there was a significant negative correlation between atherogenic index and high-density lipoprotein. $P<0.05$; $N= 30 \pm SEM$.

CHAPTER FIVE

5.0 DISCUSSION

Our research involved the assessment of some major lipid parameters amongst apparently healthy young female subjects in the university and from our results we observed in Fig I: Body Mass Index variations in obese and non-obese individuals.

The result shows a significant difference in obese compared with non-obese individuals. P<0.05; N= 30 ± SEM. This may be attributed to the accumulation of fat in adipose tissues of these obese subjects as the result corresponds to a study on 'Obesity' published by World Health Organization in 2012.

Fig. II from our research showed Total cholesterol concentration between Obese and Non-Obese subjects. There was no significant difference in total cholesterol level between test group compared with control. P>0.05; N= 30 ± SEM. In contrast with some previous researches, increased serum total cholesterol is associated with Obesity. However, our finding is in tandem with the work by Nichols,2017 who in his research revealed that Diet, exercise or genetics could play a role in this or it may be a matter of timing. He also said 1 in 7 Obese People Has Normal Cholesterol. It could also be due to the fact that the participants in this research were apparently young healthy subjects who are not morbidly nor severely obese.

From our work, Fig. III: Triglyceride concentration between Obese and Non-Obese subjects. The result showed that there was slightly elevated levels of triglyceride in the test when compared with the control but this was not statistically significant. P>0.05; N= 30 ± SEM. This may be because these test subjects are very young and apparently healthy; though obese, their body system's mechanism of equilibrium is unscathed and homeostasis is ensured. In a research work done by Bhardwaj *et. al.,* in 2013 also, Obesity was stated to be characterized by insulin resistance causing enhanced fatty acid esterification due to elevated insulin levels and serum TG levels raises.

Fig. IV in our results revealed LDL Concentration between Obese and Non-Obese subjects. The result showed there was slightly elevated levels of LDL in the test when compared with the control but this was not statistically significant. P>0.05; N= 30 ± SEM. However, increase in serum lipids has been established to be a cause of obesity in previous researches which corroborates with the elevated lipid levels of TG and LDL seen in Fig III and IV in this study. Low-density lipoprotein

(LDL) cholesterol is the most common type of cholesterol found in blood. Although it's often known as the "bad" cholesterol, LDL cholesterol isn't inherently unhealthy as it helps to protect the nerves also produce cells and hormones. At higher levels, LDL cholesterol builds up as plaque in the walls of blood vessels. Over time, this buildup can result in atherosclerosis, or hardening of the arteries. Atherosclerosis can raise the risk of heart attack, stroke, and death. When plaques form on the arteries supplying blood to your brain, abdomen, arms, and legs, they can lead to intestinal damage and peripheral arterial disease. This result is in line with a research study by Gordons *et al.,* 1997.

From our research Fig. V revealed HDL Concentration between Obese and Non-Obese subjects. The result showed that there was a significant decrease in test compared with control. $P<0.05$; N= 30 ± SEM. Decrease in HDLc levels may be due increased HDL2 uptake by adipocytes and enhanced catabolism of apolipoprotein A-l on HDL particles. The most important anti-atherogenic function of HDL is believed to be its ability to drive reverse cholesterol transport, a series of reactions by which HDL is able to interact with cells in the systemic vasculature and deliver excess cholesterol back to the liver for disposal as bile salts. HDL reverses endothelial cell dysfunction, stimulates prostacyclin production (which is both vasodilatory and antithrombotic), inhibits endothelial cell apoptosis, decreases platelet aggregation, and inhibits LDL oxidation, among other functions, Unfortunately, these HDLc ("Good Lipoprotein") levels are decreased in our test (obese) subjects. This result is in tangent with a study carried out by Rashid and Gernest, 2007.

Our result in Fig. VI (*Total cholesterol-HDL* ratio, CRI-I) AND Fig. VII: (*LDL-HDL ratio, CRI-II)* showed there was a significant increase in the test(obese) subjects when compared with the control subjects ($P<0.05$; N= 30 ± SEM). Castelli's Risk Ratio (CRI) is based on three important lipid profile parameters i.e. TC, LDLc and HDLc. CRI-I calculated as the ratio of {TC/HDLc} and CRI-II as {LDLc/HDLc}, was found to be significantly higher in the test subjects compared to the control. In our study, we could not observe a significant difference in TC and LDLc levels between the two study groups whereas, the ratio based on these parameters showed a significant difference between the two groups. This clearly suggests the relevance of ratios over individual lipid parameters especially in situations where the drug management might be affected and in cases of intermediate risk. This result is in line with a study research done by Tyagi *et al.*, 2013.

In our research, Fig **VIII showed the Atherogenic index of plasma in obese young individuals.** The result revealed that there was a significant increase in obesity over control. $P<0.05$; N= 30 ±SEM. Atherogenic index of plasma (AIP) is a logarithmically transformed ratio of molar concentrations of triglycerides to HDL-cholesterol, and was first described by Dobiášová and Frohlich in 2001. AIP has a stronger sensitivity that reflects the interaction between atherogenic and protective lipoprotein. A previous study showed that the value of AIP was inversely associated with the diameter of LDL-C particles. In a cross-sectional study conducted in Iran by Cai *et al*., 2017, the value of AIP was positively associated with waist circumference and body mass index and was inversely associated with physical activity. The strong correlation of AIP with lipoprotein particle size may explain its high predictive value. Our result did not only reveal a significant increase in the value of the atherogenic risk index of plasma in the test subjects compared to the slim subjects, it also revealed a significant reduction in HDL in our test subjects. AIP is being used by some practitioners as a significant predictor of atherosclerosis. As HDLc is significantly reduced in our test group, AIP is significantly raised. Studies have shown its role in predicting cardiovascular risk and effectiveness of therapy.

Fig IX: Relationship between atherogenic index and high density lipoprotein in obese subjects.

The result showed there was a significant negative correlation between atherogenic index and high-density lipoprotein. $P<0.05$; N= 30 ± SEM. AIP, the logarithm of the molar ratio of TG to HDL-C, according to a study by Hartopo *et al*.,2016, AIP was inversely correlated with age, hence we decided to use young obese subjects to eliminate the influence of age related issues on our findings which agrees with our study that shows a strongly statistically negative relationship between the AIP and the HDL concentration. This means the more the increase in HDL concentration the lesser the risk of CAD.

CONCLUSION

Obesity leads to an unfavorable lipid pattern, characterized by high total cholesterol, triglyceride concentration, low density lipoprotein cholesterol levels and low high density lipoprotein cholesterol levels which elevates the values of Castelli Risk Index and Atherogenic Index of Plasma. These lipid ratio parameters take account of the proportion between pro-atherogenic and anti-atherogenic fractions hence considered more effective as diagnostic and prognostic alternatives in cardiovascular risk assessment unlike the conventional lipid parameters shown to be inadequate, especially in persons with intermediate risk.

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