EFFECTS OF INTERVAL TRAINING ON SERUM LIPIDS, LIPOPROTEINS AND BODY FAT PATIERNS OF NIGERIAN ADULT MALES

BY

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AUGUST, 2015

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BY

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DEPARTMENT OF PHYSICAL AND HEALTH EDUCATION, FACULTY OF EDUCATION, AHMADU BELLO UNIVERSITY, ZARIA, NIGERIA

AUGUST, 2015

DECLARATION

I hereby declare that the research work in this dissertation titled "Effects of Interval Training on Serum Lipids, Lipoproteins and Body Fat Patterns of Nigerian Adult Males" is written by me and that this dissertation has not been presented for the award of any degree to the best of my knowledge. All sources of publication and other related literature cited in this research work are duly acknowledged in the references.

…… …….

DANIEL, Philip Lekwot **Date**

CERTIFICATION

This dissertation titled "Effect of Interval Exercise Training on Serum Lipid, Lipoprotein and

Body Fat Patterns of Nigerian Adult Males" by Daniel, Philip Lekwot meets the regulation governing the award of the degree of Doctor of Philosophy (Ph.D.) in Exercise and Sport Science in the Department of Physical and Health Education, Abmadu Bello University, Zaria and is approved for it contribution to knowledge.

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DEDICATION

This research work is dedicated to the Almighty God for His grace, mercy and love.

It is also dedicated to my late parents (Mr. and Mrs. Daniel Lekwot) and late daughter (Comfort Philip).

ACKNOWLEDGEMENTS

The researcher wishes to appreciate the Almighty God for His love, Grace and Mercy throughout the period of academic work. The researcher wishes to acknowledge the great contributions of his supervisors Professor J. A. Gwani, Professor Professor M. A. Chado and Prof. Venkateswarlu K. who despite their tight schedules found time to read this work and offered useful suggestions that helped in improving the quality of this research work. The researcher also appreciates Dr. B. A. Gunen and Professor C. B. Dikki who contributed greatly in improving the quality of this research work by offering useful suggestions that assisted the researcher to complete this work successfully. The researcher appreciates all his research assistants, Mr. Daniel Gerji, Mr. Timon Gunen the participants in the study and all the Chemical Pathologists and Hematologists whose contributions made this work a good one. Special thanks go to the researcher's family members for their prayers and encouragement throughout the period of this study. The researcher appreciates Mrs. Mary Daniel who took time to type this work successfully. The God Almighty would bless everyone that contributed to the success of this work, Amen.

ABREVIATIONS

AAPHERD-America Alliance for physical, Health Education, Recreation and dance.

AC SM- American College of sports medicine

ASRM- Anthropometric standardization Reference Manual

BMI-Body mass index

CHD- Coronary Heart disease

HDL-C- High density lipoprotein cholesterol

LDL-C-Low density lipoprotein cholesterol

T.C-Total cholesterol

TG-Triglycerides

WHR- waist - hip - ratio

CVD- cardiovascular disease

JAR-Institute for Agricultural Research

C.I — Conicity index

VAT-Visceral adipose tissue

ABSTRACT

This study investigated the effect of a 12 week programme of moderate intensity interval training on serum total cholesterol (TC), serum triglycerides (TG), low density lipoprotein cholesterol (LDL-C), High density lipoprotein cholesterol (HDL-C), TCIHDL-C, TC/HDL-C LDLC/HDL-C ratios and body fat variables which include Body Mass Index, Conicity Index, Waist to hip circumferences, and waist to hip ratio (WHR) in an untrained adults ages 18-25 years. The participants were randomly assigned to experimental group (N18) or a control group (N18). The participants in the experimental group underwent a 12 week interval training in brisk walk, jogging and running (1:1iwork-rest ratio) 3 times for 12 weeks at a moderate intensity of 70% maximal heart rate. Results showed that there were significant reduction in serum total cholesterol (TC), (P < 0.01), triglycerides (P> 0.01), TG/HDL-C ratio (P 0.01) waist circumference (P 0.01) and significant increase in high density lipoprotein cholesterol (P> 0.05) but no changes were shown in low density lipoprotein cholesterol (LDL-C), waist to hip ratio, Body Mass Index and Conicity Index with interval training. Therefore, based on the findings of this study, the researcher recommended that Nigerian Adult males should always participate in regular interval training programmes of low to moderate intensity to reduce the chances of cardiovascular disease risk factors and to maintain health and fitness for productivity in the general population.

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OPERATIONAL DEFINITION OF TERMS

Technical terms used in this study are operationally defined to avoid confusion.

1. Serum-refers to a clear liquid contained in blood which can be separated from blood that clots.

2. Cholesterol-Refers to a fatty substance that is produced in the liver and circulated in plasma to perform important physiological functions. It is measured in milligram deciliter (mg/dl) or millimol per litre (mmol/L). A value > 240mg/di is consider a risk factor forcardiovascular disease.

3. Triglycerides-Soluble lipids that are energy source and are measured in milligram per decilitre or millimol per litre with a normal range of < 1 50mg/dl in plasma. A value greater than 150mg/dl is consider a risk factor for cardiovascular disease.

4. Low Density Lipoprotein Cholesterol (LDL-C). refers to a complex lipid and lipoprotein molecule that forms a fatty substance on the blood vessels to cause obstruction to blood flow. It is measured in milligram per deciliter or millimol per litre with a value less than l00mg/di in plasma. A level above 100 mgldl is considered a cardiovascular risk factor.

5. High Density Lipoprotein Cholesterol (HDLC) Refers to a complex lipid and lipoprotein molecule that is circulated in plasma. It is measured in milligram per deciliter (mg/dl) or in millimol per litre (mmol/L). a level below 40 mg/dl) or bçlow O9mmolfL is considered a risk factor for cardiovascular disease. It protects cardiovascular events.

6. Waist circumference-Refers to a measure that assess intra abdominal fat a value > 107cm in men and > 88cm in women indicates cardiovascular risk factor.

7. Waist - hip - ratio-refers to a figure which describes the distribution of fat around the abdominal region of a person that can be determined the use of the formula$\frac{waist\ circumference}{Hip\ circumference}$ a value of 09 for men and08 for women is considered safe while a value of 1.0 and above is considered a risk factor to cardiovascular disease.

8. Body mass index-refers to the ratio of the total body weight that is measured in kilogram and divided by the height of the individual in metre square.

9. Obesity-refers to an excessive body fat in a given normal body weight that can result in degenerative metabolic malfunction.

l0. Hypercholesterolemia-refers to a condition in which there is over production of cholesterol in the body that can be linked with hypothy roidism, diabet es mellitus, nephritic syndrome, coronary thrombosis and obstructive jaundice.

11. Hypocholesterolemia: refers to a condition in which there is overproduction of triglycerides in the body which can be associated with cardiovascular disease, livers disease, Nephrotic syndrome, pancreatitis, neural degeneration and myocardial infaretion and stroke.

12. Hypotriglyceridemia-refers to a condition which there is under production of triglycerides in the blood.

13. Familial hypercholesterolemia-refers to an inherited or genetic disorder in which there is over production of cholesterol in the blood.

14. Hyperlipidemia —refers to a condition in which there is over production or elevated lipid level in the blood.

15. Exercise-refers to a single and accurate bouts of bodily movement which requires energy expenditure above resting levels resulting in voluntary movement of the whole body of the individual from one position to the other.

16. Aerobic exercise-refers to an exercise training that requires the utilization of oxygen in order to keep large muscle groups moving continuously at an intensity that can be maintained for a period of 30 minutes or above such as walking, jogging, cycling, running and swimming.

17. Interval training-refers to a series of repeated bouts of exercise is alternated within periods.

CHAPTER ONE

1.1 INTRODUCTION

Background to the Study

The prevalence of lipids, lipoprotein cholesterol including body fat and its distribution in populations has increased over the years and has become a major health problem, thus increasing the risk of coronary heart disease which is a leading cause of death worldwide (Cutler, 2006; Yang, 2005; Drygas, 2000; Franceschini, 2001). For example, it has been generally suggested that elevated levels of serum cholesterol are the primary and the most independent risk factor in the development of both atherosclerosis and coronary heart diseases both in the developing and developed countries (Cutler, 2006, Yang, 2005 and Drygas 2000). High levels of high density lipoprotein cholesterol have been generally accepted to show a protective effect against coronary heart disease (CHO) due to its role in the reverse transport of cholesterol from the peripheral vascular compartment and tissue to the liver to excretion of bile. This mechanism has been linlced or associated with the activity of hepatic lipase (HL) an enzyme that facilitates the role of high density lipoprotein cholesterol in reverse cholesterol transport from the arterial wall to help keep the arteries open to reduce the risk of coronary heart disease (CHD). (Kodama, 2007; William, 1999; Drygas, 2000; Cutler, 2006 and Kelley, 2006). Similarly, research evidence (William, 1999; Kelley and Kelley, 2006) suggest that today fat patterns and its distribution

correspondents to obesity and body mass index (BMI) values of 27.8 for men and 27.3 for women respectively indicate an increased rises of coronary heart disease (CHD). Individuals with a large waist circumference compared to hip circumference have been shown to have a higher risk of coronary heart disease, onset diabetes mellitus, menstrual abnormalities, arthritis, gall bladder disease, atherosilerosis, hypertension and sudden death while high tonicity value has been associated with poor health (Kodama, 2007; William, 1999; Kelley, 2006).

Research evidence has demonstrated that a variety of factors affect blood lipids and lipoprotein composition and body fat variables such as age, gender, dietary intake, alcohol consumption, cigarette smoking, medication, menopause status, heredity, rare and physical activity (Drygas, 2000; Kelley, 2006; Cutler, 2006). Physical activity at moderate intensity has shown significant increase in high density lipoprotein cholesterol levels (HDL-C), decrease in total cholesterol levels (IC), decrease in low density lipoprotein cholesterol levels (LDL-C), decrease in triglycerides levels (TG) and improvement in body fat patterns in both men and women to enhance health and fitness compared with inactive control typically 20-30% (Durstine, 1994; Kelley, 2006). Furthermore, it has been generally accepted that there is a dose-response relationship between the amount of exercise performed and the increase in high density lipoprotein cholesterol levels (LDL-C), decrease in total cholesterol levels (TL), decrease in triglycerides (TG) levels and

improvement in body fat patterns and as well as the exercise intensity, duration and frequency (Cutler, 2006, Drygas 2000; Stem, 1990 and Kelley, 2006). Despite the well known beneficial effects of aerobic training on blood lipid profiles, the effects of other mocks of training programme on blood lipid profiles have been adequately examined. For example, interval training as a mode of training which alternates between moderate intensity work and period of rest has been suggested to be the most popularly used method of physical training in young men and women. Studies suggest that intervals 1.3 or 1.2.5 have shown little or no effect on blood lipid profile (Drygas, 2000;

Frey 1982; Perry, 1986; and Thomas 1984), but it is not clear whether longer work intervals at moderate intensity and with prolonged physical activity would show more favourable alterations of blood lipid profiles and improvement in body fat variables. Furthermore, independent effect of interval training on blood lipids and body fat patterns has not been well established.

The purpose of this present investigation was to determine whether 12 week programme of moderate intensity interval training would significantly increase high density lipoprotein cholesterol levels (HDL-C), reduce total cholesterol levels (TC) reduce triglycerides (TG-S) levels and improve body fat patterns of untrained Nigerian Adult males age 18-25 years. If different modes of interval training can be shown to cause favourable alteration on

blood lipid profiles and improve body fat patterns, it could be suggested as a method of exercise to reduce the risk of coronary heart disease (Cl-It)).

1.2 Statement of the Problem

Lipid and lipoprotein abnormality including body fat patterns are common in the general population and one suggested as a highly modifiable risk factors for cardiovascular disease while some forms may predispose to acute pancreates. One of the most clinically relevant lipid substances is cholesterol which is closely associated with the development of athenoselerosis and coronary heart disease as a leading cause of death worldwide (Durrington, 2003). Chronic diseases including cardiovascular disease result of excess lipid and body fat accumulation in the peripheral tissues has been well explained and account for 24% of all deaths in Nigeria (WHO, 2005). For example, obesity has been suggested to be the most prevalent nutrition related disorder. Investigation conducted in the western world has shown that about 23% of adult, females are now clinically obese BMI 30 while a further 1/3 of the population are overweight (BMI 25.29.9) (Jebb, 2005).

It has been well established over the years (Gonzalez, 2007) that the specific aspect of obesity that best explains the development of cardiovascular disease is the amount of fat concentrated inside the abdomen. The studies of (lean, Han, Marrison, 1995, Onat, 1999) suggest that waist circumference is an independent risk factor for cardiovascular disease independent of BMI

(U.S Department of Health and human services, 2000). The studies of lean, Han, and Morrison, 1998, Liao and Rotimi (2000) indicated that waist circumference may be an effective clinical method for measuring the risk of cardiovascular disease. Waist circumference is considered to be a good measure of intra abdominal fat, and increased in waist circumference is correlated with other health problems such as deceased pulmonary function, increased disability in older adults, increased chances of arthritis increased likelihood of asthma, increased (Rasouli, Molari, Elbein and Keru, 2007).

The risk factors for cardiovascular disease have been well established while the effects of exercise on these factors have been well examined (Drygas 2000; Cutler, 2006; Kodama, 2007). Furthermore, studies have confirmed that cardiovascular healthy fitness and physical activity are closely associated with favourable alternative in blood lipid and body fat patterns following regular participation in moderate bouts of physical activity documented as beneficial to cardiovascular health (U.S Department of Health and Human Service, 1996). Prevailing evidence supports the concept that physical activity as an independent environmental factor can help slow the progression of coronary heart disease (Kelley and Kelley, 2006; Cutley 2006; and Kodama, 2007). For example, physical activity has been shown as an effective mode of exercise intervention to improve cardiovascular protection following reduction in total cholesterol levels (TC), reduction in triglycerides levels (TG), reduction in low density lipoprotein cholesterol levels (LDL-C),

increased High Density lipoprotein cholesterol levels, improvement in lipdlipoprotein ratio and decreased levels of body fat patterns (Kelley and Kelley, 2006). Dooly-Manning and white, (1991) found significant reduction in total cholesterol, low density lipoprotein cholesterol (LDL-C) while no significant changes were recorded in blood high density lipoprotein cholesterol (HDL-C) and triglycerides following physical activity. The inconsistency in their study was attributed to small sample size of 6-14 participants and short duration of training ranging from 6-10 weeks. V

The independent effect of physical activity on total cholesterol levels, triglycerides levels, low density lipoprotein cholesterol levels, high density lipoprotein cholesterol levels, lipid/lipoprotein cholesterol ratio and body fat pattern has not been fully confirmed on Nigerian adult males. Despite the well benefits of aerobic exercise on blood lipid profiles and body fat patterns, effects of other modes of physical activity on blood lipid profile and body fat patterns has not been suggested to provide protective effects. Interval training for example, and which alternates between high intensity work load and period of rest is one of the most popularly used method for physical training for young men and women, but it isnot clear whether longer work intervals at moderate intensity with prolonged periods of physical activity would show more favourable effects on blood lipid profiles and body fat patterns to improve cardiovascular health and fitness. Therefore, this study was conducted to find out the effect of interval training on total cholesterol levels.

(TC), triglycerides levels (TG), low density lipoprotein cholesterol levels (HDL-C) lipid and lipoprotein ratios and body fat patterns. If internal training can show positive effect on blood lipid profile and body fat patterns of Nigerian young adult. The study would suggest alternative intervention strategies to improve cardiovascular health and fitness.

1.3 Research Questions

This study was designed to answer the following research questions:

i. Can interval training cause any change on total cholesterol level (TC) of Nigerian adult males?

ii. Can interval training cause any change on triglycerides levels of Nigerian young adult males!

iii. Can interval training cause any change on low density lipoprotein cholesterol levels of Nigerian adult males?

iv. Can interval training cause any change on high density lipoprotein cholesterol levels of Nigerian adult males?

v. Can interval training cause any change on TC/HDL-C, TG/HDL-C, and LDL-C/HDL-C ratios of blood lipids lipoprotein cholesterol of Nigerian adult males?

vi. Can interval training cause any change on body fat patterns of Nigerian adult males?

1.4 Basic Assumptions

On the basis of research evidence, the following basic assumptions are made for the purpose of this study

i. Serum lipids and lipoproteins have detrimental effects on health and fitness.

ii. Sedentary lifestyle is a risk factor for cardiovascular events

iii. Body fat patterns are precursors of metabolic disorders

iv. Different exercise training programme have different effects on on serum lipids, lipoproteins and body fat patterns

v. Effects of exercise training programme on serum lipid, lipoprotein and body fat patterns can be objectively measured.

1.5 Hypotheses

On the basis of the research questions, the following hypothesis are made for the purpose of this study.

Major Hypothesis

There are no significant effects of interval training on serum lipids, lipoproteins cholesterol and body fat patterns of Nigerian adult males.

Sub-hypothesis

i. There is no significant effect of interval training on total cholesterol levels (TC) of Nigerian adult males

ii. There is no significant effect of interval training on triglycerideslevels (TC) of Nigerian adult males.

iii. There is no significant effect of interval training on low density lipoprotein cholesterol levels (LDL-C) of Nigerian adult males.

iv. There is no significant effect of interval training on high density lipoprotein levels (HDL-C) of Nigerian adult males.

v. There is no significant effect of interval training on serum lipids/lipoprotein cholesterol ratios of Nigerian adult males.

vi. There is no significant effect of interval training on body fat patterns of Nigerian young adult males.

1.6 Significance of the Study

This study was conducted to find out the effects of interval training on serum lipids, lipoprotein and body fat patterns of young adult males in Nigeria. Studies have shown that serum lipids, lipoprotein and body fat distribution are the determinants of obesity, overweight, artheroselsis, diabetes mellitus, hypertension and cardiovascular disease. (Gorais, 1999; Cutler, 2006; Kelley and Kelley, 2006). Previously, eveidence supports the comncept that physical activity helps slow the progression of coronary heart disease (Durrington, 2003). Therefore, the result of this study will provide useful information on the benefits of interval training cardiovascular health and fitness in the general population.

However, different exercise training programmes have been shown with favourable results in respect of their effect in alteration of cardiovascular risk factors. This study showed that interval training reduced cardiovascular risk

factors such as total cholesterol levels (TC), triglycerides levels (TG) waist circumference and an increase in High density lipoprotein cholesterol level of Nigerian adult males.

The result of this study will contribute to the body of knowledge with reference to the effects of interval training on cardiovascular risk factors to improve health and fitness.

1.7 Delimitation of the Study

This study was limited to the following:

a. The study was conducted on sedentary Nigerian adult males age 18-25 years at Federal College of Education, Pankshin.

b. The study investigated the effects of interval training on the following blood lipids, low density lipoproteins, total cholesterol, triglycerides, low density lipoprotein cholesterol, high density lipoprotein cholesterol and serum lipid/lipoprotein cholesterol ratio and anthropometric factors which include waist circumference, hip circumference, body mass index and conicity index to determine body fat patterns.

1.8 Limitation of the Study

This study has the following limitations which was considered while interpreting the results

1. Adherence to training is the major problem that is associated with experimental training programmes. Therefore, to reduce this, the

training programme was limited to 3 training sessions per week for 12 weeks at moderate intense to.

2. There are many different groups of serum lipids and lipoproteins but only those mentioned in this study were used.

3. Field method was used to measure the anthropometric variables to determine body fat patterns of the participants. This method has been shown to correlate highly with laboratory method of measurement, so it was considered very effective.

CHAPTER TWO

2.0 REVIEW OF RELATED LITERATURE

2.1 Introduction

This chapter review literature on the effect of interval training on serum lipids, lipoproteins and body fat patterns of Nigerian adult males. To achieve the purpose of the study available related literature was reviewed and presented in this chapter under the following subheadings.

2.2 Serum lipids lipoproteins and health

2.2.1 Total cholesterol

2.2.2 Triglycerides

2.2.3 Low density lipoprotein cholesterol

2.2.4 High density lipoprotein cholesterol

2.3 Body Fat Patterns and Health

2.3.1 Body mas index

2.3.2 Conicity index

2.3.3 Hip circumference

2.3.4 Waist circumference

2.3.5 Waist to hip ratio

2.4 Biochemical measurement

2.4.1 Total cholesterol

2.4.2 Triglycerides

2.4.3 Low density lipoproteins cholesterol

2.4.4 High density lipoprotein cholesterol

2.5 Measurement of body fat patterns

2.6 Summary

2.2 Serum Lipids Lipoproteins and Health

Serum has been described as the clear yellowish fluid or liquid which is neither a blood cell nor a clothing factor but blood plasma, without fibrinogen. Blood is centrifuged in order to take away the cellular components and anti-coagulated blood yields serum without fibrinogen (Elizabeth, 2007). According to Elizabeth (2007), serum contains proteins electrolytes, antibodies, antigens, lipids, hormones and exogenous substances such as drug and micro organisms. In health and fitness, the study of serum (Serology) is important because it is used in numerous diagnostic tests and as well as blood typing. Serum is used to describe any normal or pathological fluid. For example, serum and antitoxins are used in the treatment of diaphtheria (Elizabeth, 2007). Epidemiological studies confirmed that serum contains lipids and lipoproteins which are described as fatty acids. Those lipids are cholesterol, phospholipids and triglycerides which are transported in the blood to maintain health and fitness in individuals (Cockvil et al, 1995). For

example, triglycerides are long chain fatty acids for long time storage of energy, and they provide energy for different metabolic processes for body functions including the heart (Cockvil et al, 1995; Cutler 2000 and Kelley, (2006).

According to Cockvil (1985); Cutler (2000) and Kelley (2006), cholesterol is produced in the liver for body function which include the following:

a) Synthesis of bile acids which are important for the absorption of dietary fats in the small intestines.

b) Biosynthesis of steroid hormones and provision of structural integrity to cell membrane by phospholipids as health around the nerve cells

c) An essential component of cell membrane that helps the body to absorb fats and fat soluble vitamins, and controls water soluble products in and out of the cell.

The studies of Appel, (1993); Kris, (2003); Mathan, (2005); and Nair, (1997) reported that very long chains fatty acids have significant beneficial effects on cardiovascular health and fitness suggesting that the mechanism underlying this beneficial effect results from decreased cardiac arrhythnosis, improved triglycerides levels in hyper-triglyceridemia individuals, improved blood pressure and decreased platelet aggregation.

Durting (2003) reported that cholesterol packages are the lipoprotein found in serum which are, very low density lipoprotein cholesterol (VLDL-C), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol

(HDL-C) and chyclomicrons. Von (1999) explained that high density lipoprotein cholesterol has a protective effect against cardiovascular event because it picks up excess cholesterol from the walls of the arthries and return to the liver to keep the arteries open to reduce therisk of cardiac attack.

Rye, (1999) added that density lipoprotein cholesterol product re-esterification process of cholesterol and prevents its absorption to increase the synthesis of eicosanoids to cut as antici-plate let aggregating factor to decrease the chances of clot formation, decreases the synthesis of the procures or of very low density lipoprotein and triglycerides and increase clearance of low density lipoprotein cholesterol, promotes male fertility, female conception, muscle contraction, regulation of sleep and clearance of different types of pain to maintain health and fitness in individuals.

2.2.1 Total Cholesterol

Total cholesterol has been described as a fat-like substance that helps build cell membrane, production of some hormones, synthesis of vitamin D, and synthesis bile acids that aids digestion. It is a waxy fat substance found in animal products and eggs (Durington, 2003).

However, for many years, it has been suggested that elevated levels of serum cholesterol are a primary risk factor in the development of both atfieroscferosfs and coronary artery disease (CAD) (Castelli, 1977; Durting, 2003; Cutler,2006; Kelley and Kelley, 2006; Kedama, 2007). Recent data

in the blood may be more critical in the development of coronary artery disease (CAD) than are total blood cholesterol concentrations. There is evidence showing that cholesterol transported by low density lipoproteins cholesterol may infiltrate the arterial intemia and contribute to the atherosclerotic process (Shirom et al, 2011) and thus, have a protective effect on the cardiovascular system.

Epidemiological evidence explained that populations with higher levels of high density lipoprotein cholesterol (HDL-C) and or decreased levels of low density lipoproteins cholesterol have a lower incidence of coronary heart disease (Domanski, 2007; Kelley, 2006; Kodama, 2007). Based on the above evidence it has been suggested that the development of atherosclerosuis may be more successfully prevented by increasingly serum High Density Lipoprotein Cholesterol (FIDL-C) or decreasing serum low density lipoprotein cholesterol than by attempts to decrease total serum cholesterol (Miller, 1979; Cutler, 2006; Williams, 1999).

Durinton, (2003) reported that the presence of high levels of cholesterol in the blood (hypercholesterolemia) is not a disease but a metabolic disorder that can be secondary to many diseases and can contribute to many forms of diseases notably coronary heart disease. Freeman, (2003) and Durington, (2003) suggested significant relationship between hypercholesterolemia with mental depresslon, cancer arid cerebral /raefrrcrthage adding that the effect of cholesterol on the heart may involve more than one artey. Freeman, (2003)

further reported that unhealthy levels of cholesterol may affect the heart muscle and increase the risk of heart failure and can also inhibit the protection that aspirin provides for people with heart disease.

According to Freeman, (2003) the mortability rate associated with coronary artery disease has dropped by over one half during the past 30 years due to better cholesterol management adding that only 4% of people with high cholesterol levels actually die from heart disease. Clinical investigation suggests consistently that a high risk of death from heart disease was due to high levels of cholesterol of 5.6 mmol/L and above.

However, it has been generally accepted that regular exercise has been demonstrated to show significant reduction in total cholesterol to improve cardiovascular health and fitness in the fugal population (Kelley, 2005; Shiron, 2011; and NArayani, (2010). For example Raul, (2010) and Kelley, (2006) reported that interval training produces related adaptation to total cholesterol to reduce the chances of blockage of the arteries which similarly, several clinical investigations have shown significant decrease in total cholesterol following moderate intensity interval training of 50-75 max heart rate to reduce the risk of cardiovascular events (Kelley, 2005; Miller and Williams, 2004; Williams, 2007). This decrease was attributed to body weight and fat reductions (Superko, 1991).

Superko, (1991) reported further that favourable changes in total cholesterol are often observed after training which requires energy expenditure adding

that training programmes that elicit 1200 to 220k/cal/wk in exercise are often effective in inducing significant reduction in total cholesterol levels to enhance cardiovascular health and fitness but Hakkino, (1979), Kraise and Wood (1986) did not record changes in total cholesterol following excercise programme. Furthermore, Silvanpaia Hakkinon et al, (2009) reported that interval training has been suggested as an effective mode of exercise intervention to improve cardiovascular protection following favourable alterations in serum cholesterol. According to a 2000 study, men with total cholesterol levels higher than 5.7 mmol/l have a risk that is 3 times than those whose cholesterol is below 5.7 mmol/l (Freeman, 2003).

2.2.2 Triglycerides

Triglycerides are long chain fatty acids that are primary constituents for long time storage of energy which provide energy for different metabolic processes that derive energy for many body functions including the heart (Allen, 1999). According to Allen, (1999) triglycerides which are blood fat are stored in fat cells above the usual level in saturated fats are transported in the form of chylomicron in the blood plasma as lipoprotein while they increase with age lower in men than in women. Hadiha, (2005) reported that circulating triglycerides increase with weight gain and are higher in post menopausal women and increase with administration of oestagen. Allen,

(1999); Haruna, (1994) reported that circulating triglycerides in the blood is

an independent risk factor for cardiovascular disease that can result in an increased chance for stroke, high blood pressure; low thyroid hormones level (Hypothyroidism) and type 2 diabetes mellitus, adding that triglycerides have the potential to be an independent risk factor for cardiovascular disease.

Palumbo et al, (1978); Cutler, (2006); Williams, (1999) examined individuals with known coronary artery disease (CAD) discovered that diet containing 4 grams of sucrose or grams produced a significant rise in serum triglycerides in patients with coronary artery disease compared with health controlled subsets suggesting that patients with cardiovascular disease are closely associated with high levels of serum triglycerides. Reiser, (1981), Kodama, (2007) and Kelley (2005) in a study of effect of sucrose on blood lipids in carbohydrates sensitive individuals reported that triglycerides levels increase significantly as the level of sucrose increase in the diet. The study concluded that high levels of triglycerides represents the most common form of hyperlipidemia observed in the obese while elevation of triglycerides and cholesterol may decrease with weight loss. According to Leman (1996), triglycerides pose danger regardless of cholesterol levels and are responsible for blood clot that block the arteries and can cause a considerable damage to cells. Connell, (2007) reported that lipids related disorders which include hypertriglyceridemia,

hyperlipoproteinmia, ketosis, cardiovascular disease, fatty liver, obesity, cancer, metabolic disorders, infant respiratory distress syndrome and toad disease and Zwellwegens disease. In fatty liver, lipids accumulate excessively

in the liver while droplets of triglycerides are found in the hepatic cytoplasm to cause impairment in liver function. Increased synthesis of triglycerides and impairment in lipoprotein synthesis may interfere with liver function which associated with the fibrotic changes and cirrhosis, (Connell, 2007). Increased levels of triglycerides occur in disease condition as liver disease, nephritic syndrome, pancreatitis; acute myocardial infection and diabetes mellitus (Allen, 1999).

Epidemiological research evidence indicates that physical activity as an independent factor positively influence triglycerides levels to improve health and fitness (Ochi, 2007); Williams, 1996). Due to exercise training, the activity of several enzymes such as hepatic lipase and protein lipase are modified in order to promote more rapid catabolism of triglycerides (TGS) to result to greater production of High density lipoprotein (Williams and Millers, 2004). Different types of exercise modes have been shown to influence blood lipid proffle positively such as exercise positively such as triglycerides (TGS). (Leon and Sanchez, 2001). Beneficial effects of interval training on triglycerides (TGS) have been reported by several researchers. For example, Guo (2011) reported a significant decrease in triglycerides as a result of 3 months of interval training in Japan workers. Kelley, (2005), confirmed a significant decrease in triglycerides due to 12 weeks interval training in overweight and obese adults. Similar result were reported by Tolfley (2010),on children and adolescents. Habib Zabeh (2010), on post monarchial 13-14

years old girls and Mohammed (2003), on non insulin dependent diabetes regarding the effects of exercise training. The exercise training consisted of 6- 24 weeks jogging or jogging/walking 3-5 days per a week. In a meta analysis of controlled studies, Dursting (2001), reported on the effects of exercise training performed at a volume of 1200-2200 calories per week for 18 weeks said that triglycerides levels were reduced by 0.O8mmol/1. Prong (1993), reported that moderate intensity interval training produced reduction in triglycerides, a favourable effect on blood lipid that has a protective mechanism against coronary heart disease.

Observational data provide strong evidence for low TGS in physicallyactive individuals which favours health and fitness (Durstine, 2000). Triglycerides levels are almost always lower in endurance exercise and moderate intensity interval training and athletes that are aerobically trained when compared with sedentary controls (Durstine, 2000). There is significant TGS difference between these range from 18-77mgldl or 19-50% in over half of all cross several studies (Wood 1977, Mann, 1977, Cutler, 2006; Lakka 1992; Drygas 2000,; Kelley, 2005).

Several investigators have used cross sectional design to quantify dose response relationship between the volume of exercise and changes in bloodlipids. For example Kakkinos, (1995), Lakka, (1992) compared the blood lipid levels of 2906 middle aged men who were portioned into 6 group based

on their self reported kilometers run per week. It was discovered that triglycerides levels HDL-C, total cholesterol (TC) and HDL were improved incrementally with training volume. The triglycerides (TGS), Total cholesterol and High lipoprotein were consistent across activity group. These results suggest that although exercise may have an effect on blood lipid and lipoprotein concentration differences between exercise and per week. The differences between individuals running at this level and the sedentary groups were 5.7mmolll and 0.O7mmol/l for HDL-C and triglycerides (TGS) level respectively, 0.59 for total cholesterol/high density lipoprotein cholesterol ratio.

The effect of interval training on serum lipids and lipoprotein was studied in 13 young medical students who participated in exercise programme for 30mm. Sessions of intense physical activity, there was a significant decrease in serum triglycerides from 11 0-8mgldl (Cutler, 2006).

Kelley (2006); Williams, (1999) and Wood (1976) reported that early cross sectional studies compared middle age male runners of sedentary men and reported that interval exercise has beneficial effects on serum lipid levels. The study reported significantly lower serum levels of triglycerides among the runners.

Mannineu (1992), in his studies reported that triglycerides have been shown to be an independent risk factor for cardiovascular disease or a predict risk

factor for coronary disease. The study suggested that triglycerides (TGS) decrease significantly ($P<0.036$) following acute bout of moderate interval physical activity with participants who have high level but with little impact, in similar finding, Temblay et al (1999) confirmed that physical activity of interval training type increases the activity of both skeletal muscle and adipose tissue lipoprotein lipase to facilitate the circulating triglycerides in the blood. Venkateswarlu (1992) had earlier stated that physical activity such as interval training alone can decrease triglycerides levels adding that the decrease in triglycerides due to interval training was not clear but suggested that it could be due to activity of the regulating enzymes modified positively by physical activity that clears triglycerides (TGS) from the blood.

According to Sarika, Manpreet and Jaspal (2010), interval training reduces triglycerides concentration as greater utilization of fatty acids in the trained state is fueled by increased biolysis of muscle triglycerides. Fabri (2010) reported that moderate intensity interval training results in a sustained reduction in serum triglycerides over 15 days of detraining. The decrease, he said was not attributable solely to weight reduction but it has been shown that serum triglycerides concentration decreases during exercise progression even though the participants increase their caloric intake to compensate for additional caloric expenditure. Lapman, (1977); Kodama, (2007); Drygas, (2010) and Guntelberg, (1977), indicated that interval training has been

shown to lower blood triglycerides in people. Interval based training (Raul, Mannel, 2010), has been suggested as an effective mechanism for improving cardiovascular protection with training resulting in reduction of 2% on total cholesterol and 9% on triglycerides.

Research evidence has shown positive training related adaptations to triglycerides (Raul, Mannel 2010). However, Sillanpaa, Hakkino, Aunnoven, and Laaksone (2009) failed to attain changes on following 21 weeks of high intensity endurance training. Joseph and Benar (1977) reported that training intensity between 50-90% of V02 Max intake can increase interval training power to result to triglycerides reduction while training intensity between 75-90% has been reported most appropriate for young people. A physically active lifestyle may help to prevent the age related rise in triglycerides normally observed in men (Martin, Haskell and wood 1977). They reported that interval training lowers triglycerides levels in individuals having elevated initial base line levels. Lower triglycerides levels concentration in blood has been attributed to increase the skeletal muscle and adipose tissue and lipoprotein lipase activity resulting from intcrval training. Lipoprotein lipase has been shown to be the key enzyme for the breakdown of triglycerides rich lipoprotein (Haskell and wood 1977). According to Martin, Haskell and wood (1977), a long term interval training may be a contributing factor for the lowering effect of triglycerides due to exercise in order to improve health and

fitness. The studies of (Nikkiila, Kun and Mylynen, 1980) explained that lipoprotein lipase accelerates the breakdown of triglycerides resulting in a transfer of cholesterol and other substances to the high density lipoprotein cholesterol physical activity was restricted to bed rest for three to sic weeks because of some type of illness manifest a significant decrease in high density lipoprotein cholesterol.

2.2.3 Low Density Lipoprotein Cholesterol

Low density lipoprotein cholesterol (LDL-C) has been described as a protein complex in the blood that results from the removal of triglycerides from very low density lipoprotein cholesterol (VLDL-C) by the activity of lipoprotein lipase (LP) (Brown and Carrgill, 2007). Low density lipoprotein cholesterol has been shown to be the main transporter of cholesterol in plasma and it is taken up by the peripheral tissues and liver by the specific cells surfaces lipoprotein receptor may result in familiar hypercholesterolemia (Gentic disorder) (Brown and Cargill, 2002). Low density lipoprotein cholesterol (LDL-C) has been observed to be the most atherogenic of all lipoprotein's. For example, epidemiological studies reported that increased plasma levels of low density lipoprotein cholesterol are correlated strongly with manifestation of atherosclerosis adding that it is not clear how low density lipoprotein cholesterol is atherogenic (Brown and Cargill, 2002). The findings of Aszalis, (2004); Chun, (1991) indicated that the potential

mechanism that linkedlow density lipoprotein cholesterol particles to increase the risk of cardiovascular disease include augmented oxidibility and increased permeability through the endothelial barrier. Proctor, (2004), reported that weight loss by means of dietary restriction or influence of exercise has been shown to decrease the proportion of small low density lipoprotein cholesterol particles in individuals resulting in risk reduction of cardiovascular disease.

Furthermore, when levels of low density lipoprotein cholesterol are elevated, cholesterol begins to accumulate in the vessels walls and restrict blood flow (Cutler, 2006). Clinical evidence suggests that low density lipoprotein cholesterol may directly contribute to the cellular alternations of the inner walls of arteries which may altimately lead to the development of atheroscievosis plague (Kodama, 2007) adding that oxidized low density lipoprotein cholesterol plays a dangerous role by reducing levels of nitric oxide, a chemical substance that helps relax the blood vessels to allow blood to flow freely. The studies of Drygas et al, (2006); Kannel (1986); Castell, (1986) and Cutler, (2006) reported that an increased level of low density lipoprotein concentration in plasma is atherogenic and it is associated with incidence of cardiovasacular events. Most studies reported that atherosclerosis is the building up of fatty plaque materials factor in most of cardiovascular disease casca (Drygas 2006).

According to Cutler, (2006), Drygas, (2006), when atherosclerosis plaque blocks one or more of the heart coronary blood vessels, the diagnosis

is coronary heart disease. (CHD), in this disease, a blood clot often forms in the narrowed coronary artery, thus blocking the blood flow to the parts of the heart muscle supplied by that artery. This causes heart attack or myocardial infarction. Atherosclerosis can also block blood vessels in the brain that can lead to stroke or legs that lead to peripheral artery disease or intermitted dandication, before they must rest. Therefore such patients can only walk short distances before they must rest to relieve pains in their legs (Neiman 1998). Low density lipoprotein cholesterol is elevated in metabolic syndrome such as diabete mellitus stroke, hypertension, and bladder disease and insulin resistance disorder (Neiman, 1998). Furthermore, low density lipoprotein has been suggested to be closely associated with atherosclerosis and it is described as the most atherogenic of all lipoproteins. An increased plasma level of low density lipoprotein cholesterol is strongly correlated with the manifestation of atherosclerosis but it is not clear how low density lipoprotein cholesterol is atherogenic (Brow and Cargill, 2002). The study explained that low density lipoprotein cholesterol stimulates growth and politeration to initiate atherosclerosis while patients with familial hypercholesterolemia and their members sustain a myocardial infarction by mid 40s and by 60 years of age. Regular physical exercise training has been suggested to make favourable influence on low density lipoprotein cholesterol to maintain health and fitness in active populations (Kodama, 2007).

Research evidence has explained that regular physical activity as an independent environmental factor induces favourable alterations or changes on low density lipoprotein cholesterol to improve cardiovascular health and fimess (Drygas, 2000). For example, interval training for 6-22 weeks performed on 3-5 days a week at an intensity of 50-75 maximumal heart rate has been shown to induce favourable changes in low density lipoprotein cholesterol (LDL-C) (Linder, 19993; Blessing, 1995; Muhammed, 2003; Kelly, 2012). There is evidence that suggests that those who participate in interval training show low levels of low density lipoprotein cholesterol than those who are less active (Kikkinos, 1995; Tolfrey, 2000; Venkateswarlu, 2009 and Gou, 2011), reported that low density lipoprotein cholesterol levels were lower by 0.084 mmol/l (2-21%) in those who participated in moderate intensity interval training. This suggests that regular interval training has a dramatic influence on low density lipoprotein cholesterol and other lipids variables. Furthermore, regression analysis of data from large scale epidemiological studies has failed to consistently demonstrate a relationship between physical activity and low density lipoprotein cholesterol in normo and hyperlipidemia groups (Gordons, 1988).

Williams, Kraise and Wood, (1986) and Drygas, (2000) have reported no significant effect of physical activity on low density lipoprotein cholesterol levels in active versus inactive people. Krummel, (1993); Stefanice (1994) reported that interval training conducted at moderate intensity 50-75 maximal.

heart rate induces significant changes on low density lipoprotein cholesterol improve health and fitness.

2.2.4 High Density Lipoprotein Cholesterol

High density lipoproteins cholesterol contains high concentration of protein and much smaller concentration cholesterol and phospholipids while small quantity are synthesized in the intestinal epithelium during food absorption of fatty acids (Neiman 1998). Previous studies (Castelli and Gordon, 1971; and Gordon, 1977), suggested that high density lipoprotein has an increase relationship with coronary heart disease offering a protective mechanism against the development of cardiovascular disease (Kannel, Castelli and Gordon (1977). According to Neiman (1998), high density lipoprotein cholesterol is considered to be the most powerful lipid parameter for predicting coronary heart disease on people of all ages adding that the primary function of high density lipoprotein is to transport cholesterol from the tissues to the liver for excretion from the body or synthesized into bile acids. It prevents the uptake of low density lipoprotein at receptor sites in the body and participante in the metabolism of other lipids. Cutler (2006), reported that high density lipoprotein cholesterol is predominantly composed of phospholipids and is separated into several subciasses based on size and particle density such as HDL-2 and HDL-3. HdL2; HDL-2 and HDL-C while HDL-C2 is the major antitherogenic subfraction. The studies of Kodama (2007) confirmed that females have by high contents of high density

lipoprotein cholesterol than males which helps to protect women from developing cardiovascular disease suggesting that this may be partially due to the relatively higher lipoprotein lipase activity in women and also estrogen seems to have a major role in lowering the risk of cardiovascular disease for premenopausal women.

According to Manson et al, (1992) female sex hormones found in oral contraceptives or used in hormones replacement therapies have variable effects on lipoprotein profile. Manson et al, (1992) explained further that women who consume moderate alcohol tend to have higher HL-C levels (Taylor and Ward, 1993). Further explained that high density lipoprotein cholesterol contains a high proportion of proteins, moderate amount of cholesterol, phospholipids and a little triglycerides. Williams (2007) reported that a high ratio of high density lipoprotein to total cholesterol has been suggested to have a protective effect against atherosclerosis and other forms of other heart disease. This view was earlier expressed by stuart, (2004), Werner and Sharon, (2007) that high level of high density lipoprotein cholesterol protects the heart. In disease condition, a low level of high density lipoprtotein has been reported to be the strongest indicator of coronary heart disease at all levels of cholesterol (Sharon and Werner 2007), adding that in every nimol/1 increase in high density lipoprotein cholesterol results in risk reduction of coronary heart disease by 3% in men and 5% in women. The value which is commented to reduce cardiovascular risk is 0.l7mm0l/1

(Werner and Sharon 2007), while physical activity such as interval training improves high density lipoprotein cholesterol (Cutler, 2006).

Since serum lipids and lipoproteins have been reported to influence human health and fitness, numerous clinical reports have explained that serum lipids concentrations are often used to evaluate the risk of cardiovascular disease (CVD) which is the leading course of death worldwide. (Cutler, 2006). For example, previous studies have reported that low serum concentration of high density lipoprotein (HDL) together with high serum concentration of total cholesterol (TC) triglycerides (TGS) and lipoproteins are associated with an increased risk of coronary heart disease (Cl-ID)

(O'Connel 2001) while other clinical findings have indicated that serum concentration apoliporoteins has been shown to be anti-athorogenic potential. Low density lipoproteins (LDL) is suggested to play a significant role in atherogenesis (Sniderman, 2003); O'connel, 2001) adding that APO-B concentration appears and partially useful method of evaluating cholesterol transport as an independent risk factor for coronary heart disease (CND).

Several studies have indicated that moderate jogging, brisk walk for about 30 minutes 3-5 times a week can improve High Density lipoprotein cholesterol. One should spend about 1000 calories per week through moderate to high intensity interval exercise to produce positive changes in blood lipids and lipoprotein to improve health and fitness (Ferber, 2000).

Marrugat et al (1996) reported that moderate intensity interval training with intensity greater than 7kcal/min was significantly associated with a higher level of HDL-C index. Dursting et al, (2002) explained that differences between sedentary and exercise groups become significant in individuals running 1 1-23kmlwk (700-l500kcalJwk) in HDL-C level. Lippo et al, (2006) showed that in professional cross country skiers and road cyclists, the HDL-C concentration was higher where as the total cholesterol, the triacglycerols and the LDL-C levels were lower compared to sedentary control groups. These results were confirmed by Hubner-wozmal et al (2007) in wrestlers and volley ball players, but the findings of Giada et al (1996) had earlier explained that they did not observed any differences in the total cholesterol (TC), the low density lipoprotein (LDL) and the High density lipoprotein cholesterol (HDLC) levels in experienced soccer player and body builders. Imamoglu et al (2005) did not determine any differences in the lipid profile between wrestlers and the sedentary control groups. Significant increase in the high density lipoprotein cholesterol level as a result of moderate intensity interval training has been reported by Leon and Sauchez (2001), Venkateswarlu, (2010), Thompson et al (2001) and Blessing et al (1999). The training protocol used in these studies includes walking, jogging and cycling. The duration of training ranged from 6 — 22 weeks performed at 5 0-70% of maximal heart rate 3-6 times a week for 30-50minutes in a training session.

It appears that the training caused activity of several enzymes like hepatic and a protein lipase to modify and promote a more catabolism of triglycerides to result to a greater production of HDL-C (Williams, 2007). Observational data provide stronger evidence that individuals improve health and fitness following moderate intensity interval training (Durstine 2000, 1994). Blood levels of HDL-C are 0.03 5mmól/L higher in those having physically demanding jobs and individuals engaged in endurance exercise or moderate intensity interval exercise compared with their less active individuals (Lakka, 1992; Martin 1977) suggesting that those who are physically active show good health and fitness because of the protective particularly an increase in HDL-C Hertzberg, (2004), Dallmeiser, (1997) reported that regular moderate intensity interval training has a beneficial effect on blood lipids profile thus resulting in an increase in HDL-C fractions to improve health and fitness. (Prong 1993; USDHHS, 1996).

Woof and Haskell (1970) earlier reported that HDL-C has been significantly increased following different types of physical activities such as running, brisk walk, tennis, cross country and swimming adding that is contrast, athletes who engaged primarily in anaerobic type of physical activity such as sprinting or resistance exercise which involves few repetitions have generally shown no higher HDLC-C or lower triglycerides (TGS). On the other hand, Venkateswarlu (1992) reported that jogging or brisk walking for

about 30 minutes and 3-5 times a week is important for improvement in HDL-C through moderate to high intensity interval exercise which produces positive changes in blood lipids and lipoprotein. Williams (1998) reported higher levels of HDL-C was with 16km increment in weekly running distance in both men and women, and that in men, difference in HDL-C was 0.O6mmol/L and between the least active and next exercise group, the differences in HDL-C were somewhat lower than the relationship between HDL-C and running distance in both pre and postmenopausal women. In a cross sectional design, Drygas et al, (1993) found that significant HDL-C differences between male exercisers and those in the lowest activity category (-0.022mmol/L) occurred with exercise energy expenditures of 1500 to 3000K!cal./wk. In the same study, the investigators observed that the threshold for significant HDL-C difference between middle-aged male exercisers and sedentary controls was 0.02lmmol/L and occurred at an exercise energy expenditure of 2000Kcal/wk. Drygas (2000) reported that in men, HDL-C increased by 0.0l4mmol/L/km. Drygas (2000) reported that in men, HDL-C ratio decreased by 0.02mmol/L and 0.12 per kilometer respectively (Williams (1998), Moor et al (1983) determined that running distance was predictive of greater HDL-C levels in active female recreational joggers and long distance runners. In their study, influence of running distance on HDL-C remained significant after adjusting for group differences in body fat. Durstine (2000) indicated that HDL-C levels were greater in

female recreational runners and were even higher in runners classified in the elite groups versus age matched sedentary controls. The time spent running each week was strongly associated with I{DL-C levels providing additional evidence for a positive relationship between exercise volume and favourable lipids and lipoprotein changes (William 1998, 1993). Interestingly, the study by William (1998) suggests that along with training volume, the intensity of training may be related to exercise induced blood lipid and lipoprotein changes. Kokkinos et al (1995) found significantly better blood lipid and lipoprotein proteins in women who were able to achieve moderate intensity exercise of 6-11 metabolic equivalent (IMETE = 0.01 4mmol/L/min versus of those exhibiting lower intensity levels (> 6 METS). Williams (1995) observed predictive relationship between blood lipids which was related to training.

However, results from these cross sectional studies confirmed that training volume rather than training intensity has shown the greatest influence on favourable lipid changes. This is true for exercise induced changes in HDL-C (Lakka, 1992). Research studies on effect of interval exercise training on High density lipoprotein cholesterol have shown that interval training provides positive results (Lindheim et al 1994)

Research studies have indicated that the volume or amount of exercise performed per week may also influence the magnitude of change in high density lipoprotein cholesterol levels (HDL-C) while most of the exercise

training studies identify a weekly mileage threshold of 7 kilometers a week for significant increases in high density lipoprotein cholesterol. The author suggested that a threshold of running approximately 4 kilometer per week for one year was necessary increase in high density lipoprotein cholesterol levels. In addition, Williams et al (1982) reported that plasma concentration of high density lipoprotein cholesterol generally did not begin to change but until a threshold exercise levels of 10 kilometers per week was maintained for at least 9 months. Kikkinos et al (1995a) reported a significant increase in high density lipoprotein cholesterol levels in runners that averaged 7 kilometers per week. An additional study by Williams (1998) suggested that exercise volume is more important than exercise intensity adding that weekly mileage was more strongly correlated with high density lipoprotein cholesterol levels than exercise provides significant increases in high density lipoprotein cholesterol in a shorter period of time. Williams, (1998) reported that there may be a relationship between exercise volume and length of the training programme. For non runners, a caloric expenditure of above 1 000k/cals per week has also been defined as a threshold close of exercise to increase high density lipoprotein cholesterol levels (Drygas et al, 2000). The authors noted that energy expenditure of 2000k/cals perweek is associated with additional increases in high density lipoprotein cholesterol (HDL-C).

However, exercise training studies attempting to access the role of exercise intensity on high density lipoprotein cholesterol in women are few and have report conflicting results (Spate Douglas and Keyer 1999). They reported that most of the studies suggest that women (pre and postmenopausal) with low levels of high density lipoprotein cholesterol are more likely to respond positively to exercise training. Duncan et al (1991) reported similar increases in high density lipoprotein levels in women (29-40 years) following 24 weeks of walking 4-8 kilometers regardless of intensity. This finding suggests that moderate exercise will raise high density lipoprotein cholesterol levels as much as intense exercise. Spate — Douglas and Keyer (1999) confirmed that moderate intensity interval training over a 12 week period was sufficient to improve the high density lipoprotein cholesterol profile and high intensity training appears to be of no further advantage.

In women, the volume of exercise seems to be more important than the intensity of exercise for influencing high density lipoprotein cholesterol levels (Fernhall 1999). Most studies suggest a large volume HDL-C changes in women, however, the exercise volume threshold has not been defined (Fernhall 1999). Generally, physically active women exhibit higher levels of HDL-C levels when compared to their sedentary counter parts (Kikkinos et al and Famhall 1995). In a study by Kikkinos et al (1995) women who were

categorized in a moderate and high fitness category as accessed by an exercise tolerance test, exhibit higher HDL-C levels than those who were categorized in a low fitness category. Elevated high density lipoprotein cholesterol (HDLC) levels have been reported in women following high volume training programme (Williams, 1996; Williams 1998) but not for those in a low volume training programme.

Williams (1996) reported that HDL-C concentration increased significantly in relation to the number of kilometers (km) run per week in postmenopausal women and pre menopausal women whether they were receiving Hormonal replacement therapy or not. He reported substantial increases in high density lipoprotein cholesterol in women who ran more than 64 kilometers per week (37miles) when compared to those who ran less than 48 kilometers (30 miles). These findings suggest a dose response relationship between exercise and high density lipoprotein levels.

2.2.5 Serum Lipid/Lipoprotein Ratios and Health.

A series of clinical studies suggested that the use of ratio of low density lipoprotein cholesterol to high density lipoprotein cholesterol is superior to the use of high density lipoprotein cholesterol alone (Brown and Cargill, 2002). According to Brown and Cargill, (2002) the ratio of low density lipoprotein cholesterol and high density lipoprotein cholesterol (LDL-C/HDLC) may make better provision for risk assessment for both atherogenic and

protective lipid fractions. Cutler (2006) and Kelly, (2005) reported that the cardio protection effect of High density lipoprotein cholesterol (HDL-C) has been widely attributed to its role in reverse cholesterol transport (RCT) in which excess cholesterol is conveyed from the peripheral tissues to the liver and the steroid organs. However, high density lipoprotein cholesterol has been classified into several groups due to differences in the quantitative and qualitative lipids contents, apolipoproteins and enzymes thus resulting in the presence of various High density lipoprotein cholesterol. Sub classes which are characterized by differences in size, shape, charge and antigencity (Cutler, 2006, Kelly, 2005).

Epidemiological evidence has suggested that increased levels of low density lipoprotein cholesterol and reduced levels of High Density lipoprotein cholesterol have been suggested to be highly atherogenic. The low density lipoprotein cholesterol/high density lipoprotein cholesterol ratio possessed more progrostic value than low density lipoprotein cholesterol and high density lipoprotein cholesterol alone (Kodama, 2007; Drygas, 2000 and William, 1995). In several studies, the low density lipoprotein cholesterol/High density lipoprotein cholesterol ratio has emerged as the best single lipid predictor of coronary heart disease (Brown and Cargill, 2002). Previous reports from the revealed that the low density lipoprotein cholesterol/high density lipoprotein cholesterol (LDL-C/FIDL-C) ratio was

significantly linked or correlated with the progression of both low grade and high grade coronary artery disease (CAD) (Kelly and Kelly, 2006).

The data showed that for every 1% decrease in the low density lipoprotein cholesterol/high density lipoprotein Tholesterol/high density lipoprotein cholesterol ratio, there was 31% reduction in the risk of a major cardiovascular event. According to the assessment from the Quebel Cardiovascular study and calculated odds ratios for Ischemic heart disease (Il-ID), increased ischemic heart disease in the 3.9 to 4.6 and < 4.6 subgroups of the low density lipoprotein cholesterol/high density lipoprotein cholesterol ratio odds ratio for the 2.3 along with the 2.3 to 3.9 low density lipoprotein cholesterol subgroups were 1.0 and 1.9 respectively.

Furthermore, several prospective studies (Taylor, 1993; Chrisstodoubu, 2008) have suggested that a high/low density lipoprotein cholesterol/high density lipoprotein cholesterol (LDL-C/HDL-C) ratio combined with hypertriglyceridemia (HTG) ratio correlated with highest incidence of coronary heart disease.

Taylor, (1993) observed that the average low density lipoprotein cholesterol/high density lipoprtotein cholesterol ratio for people without coronary heart disease (CHD) was below 3.4 while the ratio values for patients with excessive rates of coronary heart disease (CHD) were at least 3.5 suggesting further that this is commonly thought as a desirable ratio of

low density lipoprotein cholesterol/high density lipoprotein cholesterol ratio that has been used as an indicator of coronary atherosclerosis.

To improve health and fitness, epidemiological studies have reported that different modes of physical activity have been shown to make favourable alterations in serum lipids and lipoprotein cholesterol ratios. For example, Taylor, (1993); Dowda, (2000) have reported that initial levels of total cholesterol, total triglycerides/High density lipoprotein cholesterol ratios were strongly correlated with their respective changes due to interval training adding that high initial levels of total cholesterol, total triglycerides and high density lipoprotein cholesterol ratios resulted in greater decreases post exercise and lower initial levels of High density lipoprotein resulted in greater post exercise training programme.

2.3 Body Fat Patterns and Health

Body fat constitutes essential body fat and also fat that is stored in the body. Body fat is present in nerve tissue, bone morrow, organs and all the membranes. All these fats cannot be lost without compromising the physiological functions that are involved. Storage fat presents energy sourcewhich accumulates when excess energy is required than consumed (Lahman, 1993) adding that women are believed to have more essential body fat than men due to childbearing and hormonal functions. Lobman (1993) further

reported that the total body essential fats and storage fat is between 12% 15% for young men and between 25%-28% for young women.

Durmin (1994) reported that the distribution of body fat appears to be associated with metabolic attractions, and that in the middle age women, genetic factors remain the strongest influential factors on the amount and distribution of body fat which accounts to 60%. There is substantial evidence which demonstrated that body fat distribution to the abdominal region during and after menopause in woman may be attributed to the effects of estrogen (Rebute, 1987; Manson, 1995).

In health, fat is understood as the subcutaneous tissue necessary for health and fitness, and too much or little of it is detrimental to health, fitness and performance (Chado, 1992; venkateswarlu, 1990). Fat is used in the body as fuel especially for exercise or body movement of low to moderate intensity (McArdle, 1997; Bar-or1987).

Bar-or (1987) suggested that low fat content is desirable for both men and women for better health and fitness. Fat deposit is controlled by such factors as heredity and dietary habits while fat deposit during childhood is as a result of increase in fat cells, size or an increase in the fat cell member or both. As the individual grows up to adolescent, the body contains more fat due to increase in the size to existing number of fat cells (Pollock et al 1993).

In disease condition, Manson (1995) reported that an increase in central adiposity in woman is a greater predictor for heart disease, diabetes mellitus and death. Mason (1995) added that central obesity is part of metabolic syndrome associated with dyslipidemia, hypertension and glucose intolerance while Venkateswarlu (2000) reported that fat pattern differ in individual which depends on heredity, age and sex. William (1999) indicated that regional fat distribution is a concept that represents the anatomical distribution of fat over the body and may be related with several major health problems. However, classification of different types of obesity has been shown most popular differentiation (Campaign 1990).

Campaign (1990) explained that research evidence has shown that android type of obesity is characterized by accumulation of deep visceral fat in the abdominal region and similarly accumulation of subcutaneous fat. Android type of obesity is commonly called lower trunk obesity. The female type obesity (gyniod) is characterized by fat accumulation in the gluteal formal region, the hip, buttocks and the thighs (Campaign, 1990). Research evidence suggests that both types of obesity have a genetic component (Bourehard, 1991). William (1999) stated that android obesity is commonly known to be a greater health risk than obesity itself. It appears that both android and gynoid type posses different biochemical functions due to differences in lipoproteins lipase activities i.e. an enzyme that regulates fat metabolism and fat cells that are found in the deep visceral depots which are

active metabolically to release free fatty acids into the blood streams when they are stimulated by epinephrine. This may contribute to abnormalities in glucose and lipid metabolism especially in people who experience psychological stress (Radin et al 1989 and Williams, 1999).

Furthermore, body fat pattern distributions have been well explained. For example, several research studies have demonstrated that fat pattern differ from one person and to the other on heredity, age and sex factors (Venkateswarlu 2000). This observation was supported by the studies of Perez et al (2000) which showed that there is increase in the waist to hip ratio (WHR) in boys and girls and waist and hip circumference although an increase in male during adolescent periods. The study explained further that conicity index (CI) and WI-JR has a strong link in females than males and tend to be more greater in adverse age groups.

2.3.1 Body Mass Index (BMI)

Several studies have reported that people with a body mass index of 25- 29.9 are considered over weight and are associated with obesity. Body mass index is a measure of expression of relationship of weight to height and it is associated with body composition (body fat %) with indices of health risk. For example, Mama health.com (2009) stated that in some people, the body mass index is not a reliable indication of health risk that highly muscled individual who is very fit and healthy may have heavy body weight due to

muscle pack which may result to high body mass index (BMI) that improperly places the individuals while other individuals who have a low body weight with very little muscles and high percentage of fat may have a normal body mass index whose weight would be an incorrect indicator of healthiness. Durimin 1984 and Williams (1999) stated that body mass index provides a guide line based and height in order to determine underweight and overweight while children's body fitness changes over the years, and added that interpretation of body mass (BMI) depends on the individual age. Williams, (1994) reported that body mass index is a persons body weight in kilogramme divided by the square of his or her height (W/ht2) and correlated with body fat. Studies have shown that both Body mass index and waist circumstance have more adverse effects on triglycerides and high density lipoprotein cholesterol. In a Heart study population, 60% increase in Body mass index (BMI) from 25-4Okg/m2 was associated with a 0.O76mmol/L increase in triglycerides and 0.O75mmol/L decrease in High density lipoprotein cholesterol in women (Rexode K.M 1996).

Valdez et al, (1992) Perez et al (2000) suggested that a high value of concity index is strongly associated with more central fat distribution and has been used as an alternative method to analyze fat distribution especially in adolescent in field surveys.

Perez et al (2000) reported that a positive association between overwieght and high concity index values and as well as between high conicity values and high level of triglycerides have been reported to influence health and fitness. For example obesity has been linked with overweight and obesity itself has many negative effects on health and fitness because it is associated with diabetes, gallbladder disease hypertension and heart disease (Perez et al 2006).

Knowler, et al (1995) reported that obesity is also associated with increased mortality of various lipid and lipoprotein abnormalities which has been observed in obese individuals including elevated cholesterol, triglycerides and apolipoprotein B (apoB) and lower high density lipoprotein cholesterol (HDL-C) levels Gunnell (1998) suggested that changes in triglycerides (TG) and High density lipoprotein cholesterol are most constituents and pronounced. Some studies have demonstrated that central obesity is more strongly related to lipid and lipoprotein abnormalities than general obesity (Seidell, 1991, Walton, 1995, Wada 1998 and Trials 1997).

The consequences associated with overweight and with abdominal obesity are numerous (Flier, 2001). For example, overweight and obesity constitute health disorders which include coronary heart disease, pulmonary disease, reproductive disorders, gall stone disease, type 2 diabetes mellitus, certain cancers of the colon, prostate cancer, breast cancer, cancer of the

crevix, ovarian hypertension, degeneration joint disease, low back pain, elevated triglycerides and elevated low density lipoprotein and total cholesterol. This finding was supported by the American College of Sport Medicine (2001) that these problems increase when the body mass index in adults (BMI) is greater or above 25. Obesity is excess fat with BMI over 30 in adults (Flier, 2001). Increased body weight has been correlated with an increased risk of mortality with coronary heart disease in the general population (Clamonfava 1996). The National Institute of Health (1998) report that adults who are 1 8years or older and whose body mass index is 25 or more are considered risk for premature death and disability as a consequence of overweight even more as the severity of the individual obesity increases. Venkatesswarlu (1998) confirmed that overweight is associated with genetic factors and that overweight is associated with high blood pressure, diabetes mellitus, arthritis related disabilities and some cancers.

Body mass index (BMI) in kg/m2 has been shown to be widely used for classification of overweight (BMJ 25) and obesity (BMI30) in both men and women (Zhu, Zimian, Stanley, Moonseong and Myles, 2002). BMI has been shown to correlate reasonably with laboratory based measures adiposity for population studies and it has been similarly reported been practiced in most clinical settings according to Zhu, Zimian, Stanley et al (2002). Body mass index does not account for a wide variation in body fat distribution,

nature of obesity across different individuals, population and the joint relation of body composition and body size to health outcomes (WHO, 1997; Micvels; Grenniand, Rosner, 1998). General studies have demonstrated that body fat distribution has been suggested to be .more powerful predictor than body mass index for risk factors and disease mortality (Stanley et al, 2002; Murray et al, 1982; Williams, 1999; Moonsaony and Mylos, 2002; Suter, 1993 and Preveen, 2009). Correlation of abdominal fat distribution, and associated ill health than waist to hip ratio (Lean et al, 1995).

Lean et al, (1995) reported that in a middle aged woman, a wide hip circumference is protective factor for health in later life. Their study confirmed that large hip circumference predicts less incidence of cardiovascular disease and total death in women expressing further that a large hip circumference seems to have independent and positive effects on cardiovascular and coronary heart disease morbility and mortality in women but no protective effect on cardiovascular health in men.

2.3.2 Conicity Index (CI)

Valdez et al, 1992; Perez et al, (2000) reported that a high value of conicity index has been shown to be strongly correlated with more central fat distribution and has been used as an alternative method of analyzing fat distribution especially in adolescents in field surveys. Perez et a! (2000) added that's a positive association between overweight and high conicity index

values and as well as between high conicity values and high level of triglycerides have been reported to influence health and fitness

Waist to hip ratio appears to be superior to body mass index (BMI) in predicting cardiovascular risk (Williams, 1999). According to the World Health Organization (WHO, 2008), this is based on the reason that increased risceval adipose tissue is correlated with a range of metabolic abnormalities including increased glucose tolerance, insulin sensitivity and adverse lipid profiles which are risk factors for type 2 diabetes and cardiovascular disease.

Larbon 1984, Stanley (2002) confirmed that waist to hip ratio is a risk factor for cardiovascular disease and diabetes adding that hip circumstance is calculated on the waist circumference divided by the hip circumference. Further, Larbon (1984) explained that, for most people carrying extra weight around that, middle, increase health risk more than carrying exttra weight around their hips and thigh adding that for both men and women, a waist hip ratio of 1.0 or higher is considered a risk health consequence of heart disease. Stunkard (1993) reported that for men, a waist to hip ratio 0.9 or less which is considered safe while for women, a ratio of 0.8 is. suggested safe.

2.3.3 Hip Circumference

More recently, it has been proposed that hip measurements should be discarded with a concentration on waist circumference and body index (BMI) because waist circumference makes provision for a more practical.

2.3.3 Waist Circumference

The National Institute of Health (1990) reported that waist circumference is a common measure that is used to assess abdominal fat contents. Measurement of waist circumference is useful in individuals who are classified as overweight on the BIvil scale although increased waist circumference is suggested to be marker for increased risk of degenerative disease in patients of normal weight, (Depres 1990)

The National Institute of Health (1990) reported further that the presence of excess body fat in the abdomen when out of proportion to total body fat is considered an independent predictor for risk factor and ailment associated with obesity adding that waist circumference differs for men and women, and that men who are at risk have a waist measuring greater than 100cm.

The studies of Ford and Giles, (2003), Thorton and Bani (2003) confirmed that abdominal fat contains high amount of visceral fat which is produced by the liver and converted into cholesterol and released into the blood stream where it forms plaque on the artery walls. They further reported that this is why an individual is likely to have high cholesterol, high blood

pressure, cardiovascular disease and a strong risk factor for insulin resistance which is a key in metabolic syndrome and a precursor to type 2 diabetes.

Lahman (1998) in the anthropometric standardization reference manual suggested that waist circumstance is measured at the narrowest point of the waist. The World Health Organization guidelines suggest that waist circumference should be measured at the midpoint between the lowest rib and the iliac crest where as the National Institute of Health (NIH) recommends that waist circumference be measured immediately above the iliac crest. Wang, thorton and Bani, (2003) reported that waist circumstance for men that is over 94cm indicates health risk while in women waist circumference over 80cm indicates a substantial increased health risk.

2.3.5 Waist - Hip - Ratio (WIIR)

Larson (1984) reported that waist to hip ratio is used to estimate abdominal adipose tissue distribution. The waist to hip ratio i.e. waist circumference divided by hip circumference was suggested as an additional measure of body fat distribution (WHO, 2008). The ratio can be measured more precisely than the use of skinfold and it makes provision for index of both subcutaneous and abdominal adipose tissue (Bjoratop 1987). The World Health Organization (2008) reported that even though body mass index was linked or correlated with an increased risk of cardiovascular disease, waist to

hip ratio appears to be the strogest independent risk factor for cardiovascular event than the Body mass index.

To improve health and fitness in the general population, physical activity as an independent environmental factor. has been reported to effect positive changes in adipose tissue. For example, interval training has been shown to have positive effect on obesity (William, 1999). He explained that interval training induces increase in caloric expenditure that allows for the accumulation of the subcutaneous adipose tissue. Kelley, (2005) reported that obese people do not show activeness compared to those people with normal weigh. Hill et al (1994) expressed that interval training as an independent factor determines total level of energy expenditure, and that increased in physical activity is a determinant of fat balance and it is an important factor for weight regulation in individuals.

Williams, (1999); Kodama, (2007) and Cutler, (2006) reported that interval training activity offers variety of effects on the individual body weight and body fat and effect changes in percent body fat and fat free mass (FFM). Studies have shown that interval training has no effect on waist to hip ratio (WFIR) in Africa-America men and women (Larson 1997). This suggests a loss in visceral adipose tissue during negative energy balance.

Schwartz et al (1991) reported a greater loss in visceral adipose tissue In young men and old ones following interval training. The finding concluded

that interval training alone can reduce body fat content and it has been suggested to be an important factor for maintaining body fitness and body weight. Hill (1997) explained that interval training is closely linked with body fat distribution. He concluded that both interval training and resistant exercise have been shown to have a positive effect in body weight regulation. Hill (1997) reported that moderate intensity interval exercise plays a greater role than exercise of high intensity in the reduction of weight which in turn induces a substantial reduction of visceral adipose to improve health and fitness in individuals.

This finding is supported by Guttin and Ownes (1999) who confirmed that interval training has been shown to be a major factor that readiness excess adipose tissue and subsequent overweight in individuals.

Leon (1997) suggested that moderate intensity interval physical activity should be carried out for a longer period of time in order to induce much fat oxidation while interval training with high intensity, longer duration and frequency induces positive changes in body mass index (BMI). Two studies using doubly labeled water to assess the energy expended in physical' activity have demonstrated a significant negative association of interval training and body mass index (BI'vH) (Davis et al 1995, Schulz 1994).

Research evidence has shown that both interval and resistant physical activity of moderate intensity have been shown to be effective in weight reduction programme (Wood 1998). He further expressed that of greater

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intensity is linked with lower body mass index (BMI) compared to exercise of moderate intensity.

Davis (1995) Schulz (1995) reported a significant negative effect of interval training on body mass index. The National cholesterol Education Programme (1991) reported that data are available regarding how frequent interval training influences body weight regulation, and explained further that experts recommend that people should exercise 3- 5 days per week. Davis (1995) reported that weight loss produced by interval training include substantial reduction of visceral adipose tissue. Despres (1991) reported that it is not clear whether weight loss produced by interval training combined with energy restriction includes greater production of visceral adipose tissue reduction than weight loss reduction restriction alone. Depres (1993) said that combination of interval training and energy restriction does not result in any greater weight loss than does energy restriction alone while loss of visceral adipose tissue may be influenced by interval training with gender variation. This suggests that it is unclear whether men and women show the same reduction in visceral adipose tissue with weight loss following interval training of moderate intensity.

Wing and Jeffery (1995) reported that waist to hip ratio (WHR) in men and women who engaged in a six month interval training found a greater reduction in waist to hip ratio in men versus women at the end of the training programme. They found that at the end of the 18 months, the result was

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reversed with women showing greater reduction in waist to hip ration (\VHR) than men. There is much research evidence that supports the benefits of interval training for all ages (Williams, 1999). Recent studies have demonstrated that middle aged women who engaged in interval training have shown higher resting metabolic rates and lower percent body fat and fat mass compared to sedentary women of similar age and overall body mass (Gilliat et al 2001). A similar study compared healthy and weight stable premenopausal women age 35-50 years restrictively to assess the influence of regular interval training level on resting metabolic rates and body composition. The participants were grouped into active with Nine hours per week of interval training for ten years, the participants constitutes 18 active and 14 sedentary participants.

All the participants in the two groups were similar in body mass, fat free mass, body mass index (BMI) 27 and age. The activity levels were higher in the active group without difference in age and body size between the two groups. The result of the study showed that the women in the active group had 28.8% body fat. Total weight of fat adipose of the active group was estimated as 11.1 kg and 18.8kg for the sedentary women. The resting metabolic rate was found to be significantly higher in the active women than middle age women who engaged in regular interval training (Gilliat et al 2001). Few studies examined the effects of interval training on fat distribution which is usually assessed as waist to hip ratio (WHR), the studies found negative

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correlation between levels of interval training in African-American men and in Caucasian men. There was no significant effect of interval training on waist to hip ratio (WHR) and waist circumference in European fat distribution (Seidel et al 1991).

2.4 Methods of Measurement of Serum lipid, Lipoprotein and Body Fat Pattern

Investigation in recent years has shown .that serum lipid and lipoprotein cholesterol described as fats are heterogeneous groups of chemical substances that do not exist freely in serum but are present in combination with protein subclasses (Allarn et al, 1974). The development of physical laboratory methods approaches towards elucidation of the nature of those complex substances for the purpose of identification for clinical significance has been fully explained (Tietz, et al, 1987; Allain et al, 1974; Lopez-Virella et al, 1977; Tietz, 1990; WHO, 1999; Kamlesh and Melanic, 2005; Manuel, Sean and Ana, 2010). Also laboratory methods of measuring body fat pattern have been developed which include hydrostatic weighing devices, skinfold measurement, bio electric impedance analysis, ultra sound and dual energy protection methods (Maud and Foster, 1995). For the purpose of this study, field methods of measuring body fat pattern were used because they are not expensive, are easy to administer and correlate with laboratory methods of measuring body fat pattern have been developed which include hydrostatic weighing devices, skinfold measurement, bio electric impedance analysis, ultra sound and dual energy protection method. (Maud and Foster, 1995). Enzymatic methods and the use of phosphogungstic aid magnesium chloride (mg2cl) reagents to estimate total chloestrol (TC),

triglycerides (TG), and High density lipoprotein cholesterol (HDL-C) were used.

The low density lipoprotein cholesterol (LDL-C) was estimated using the Friedwald et al, (1972) equation which relies on the estimation of total cholesterol (TC) triglycerides (TG) and high density lipoprotein cholesterol (HDL-C) without ultra centrifugation. All the biochemical methods used are fully discussed in chapter three in the methodology of this study.

2.5 Summary

Lipids, lipoprtoteins and body fat pattern abnormalities are common in the general population and are regarded as modifiable risk factors for cardiovascular disease while some forms may predispose to acute pancreatitis (Kelly and Kelly, 2006; Drygas, 2000; Kodama, 2007). These factors are associated with abdominal obesity, increased wiast circumference, high values of conicity index, increased body mass index (BMI), elevated serum total density lipoprotein cholesterol (LDL-C) and low levels of high density lipoprotein cholesterol (HDL-C) (Mbanya and Kanshik, 2009; Aberty and Grundy et al, 2009; Zimmet and Show, 2005).

Epidemiological studies have reported that interval training of moderate intensity has been shown to be an effective mechanism for improving

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cardiovascular protection done to its favourable alterations of serum total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-C), increase in levels of high density lipoprotein cholesterol (HDL-C) improvement in serum lipids and lipoprotein ratios and body fat patterns (Williams, 1999; Kelly and Kelly, 2006; Kodama, 2007; and Drygas, 2000).

The studies of Kelly and Kelly, (2006), Grundy et al, (2009) and Kodama, (2007) have confirmed positive training related adaptations on total cholesterol (TC), triglycerides (TGS), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), serum lipid and lipoprotein ratios and body fat pattern to improve cardiovascular health and fitness. However, inconsistent health results exist on the effect of interval training on serum lipid, lipoprotein cholesterol and body fat pattern in both men and women on failing to show positive changes (Drygas, 2000). Generally, regular physical activity has been reported to produce avoura't1e changes on serum lipids, lipoproteins and body fat pattern to improve cardiovascular health and fitness and as well as the whole body (Warren et al, 2003; Eliot and Sale, 2002; Argnes and Carvallo, 2009). Interval training studies have been inadequate on Nigerian population. Therefore this study was conducted to assess the effect of 12 weeks interval training on serum lipids, lipoproteins cholesterol and body fat pattern of Nigerian adult males.

CHAPTER THREE

RESEARCH METHODOLOGY

3.1 Introduction

The purpose of this study was to investigate the effects of 12 weeks interval training on serum lipid, lipoproteins cholesterol and body fat pattern of Nigerian young adult males, serum lipid lipoprotein cholesterol and body-fat pattern are regarded as common risk factors for cardiovascular disease (CVD). To achieve the purpose of which this study was conducted, the research design, population, sample and sampling procedures, the experimental control, sequence of testing, training protocol and the statistical analysis used are discussed in this chapter.

3.2 Research Design

A pretest-post test experimental and control research design was used for this study. The purpose was to compare the effects of moderate intensity interval training on lipid profiles and body fat pattern of Nigerian adult males. The primary dependent variables in this study were total cholesterol ((TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-C), High density lipoprotein cholesterol (HDL-C), TC/HDL-C, TG/HDL-C and LDL-CIHDLC ratios while body fat variables were Body Mass Index (BMI), conicity index (CI), hip and waist circumferences and waist to hip ratio (WHR). All

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the dependent variables were obtained at baseline and after 12 weeks of intensity interval training.

In this design, 36 Nigerian adult males from Federal College of Education Pankshin who had no contra-indication to participate in the study were randomly assigned to either experimental group (N1 8) or control group (N=l 8) before training. All the participants were tested in the selected risk factors for cardiovascular disease before the experimental group was exposed to interval training for 3 times in a week for 12 weeks. The tests were repeated on all the variables for all the participants after 12 weeks of interval training for analysis.

3.3 Population

The population for this study constitute young adult males from Federal College of Education Pankshin.

3.4 Sample and Sampling Procedure

A total of 55 out of 5,000 male students showed willingness to participate in the study, and were screened before the commencement of the exercise training programme. The key inclusion criteria were as follows: Body mass index between 25 and 39.9, kg/rn2, non diabetic, no history of cardiovascular disease, stable weight, sedentary or light activity, not taking lipid and weight altering medication, not smoking cigarettes, not taking alcohol drinks and

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have not participated in any organized exercise training programme. Prior to the test, 36 of the participants met the criteria for inclusion and were randomly assigned to either experimental group or control group. All the participants who volunteered to participate in the study gave their written consent. The experimental group underwent 12 weeks interval training while the control group maintain regular activity. All the participants who volunteered to participate in the study had their data analysed to test the study objectives.

3.5 Research Assistants

The services of research assistants were required for the purpose of collecting data. A total of two (20 physical Education Specialists helped to supervise the training session while two (2) qualified haematologists collected pre and post training blood samples from the omticubital veins of the participants who EDTA contining tubes. Three chemical pathologists performed analysis on both base-line and post exercise blood samples of the participants at the Department of Chemical Pathology, Jos University Teaching Hospital, Jos.

3.6 Instrumentation

In this study, the following instruments were used for the purpose of collecting data.

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3.6.1 Weighing Scale: A precision electronic scale (Nwodel 89584, made in the People Republic of China 2009) for accurate measurement and monitoring of body weight, body mass index (BMI), body metabolic rate (BMI), body fat and body water was used to measuring the participants weight in kilogram as suggested by (AAPERD, 1990). The reading •was taken to the nearest 0.kg.

3.6.2 Stadiometer: The hottain stadiometer (model Njo 7072: Export inc. US.A) was used to measure the participants height in meters. The measurement was taken as the participant stood correct without shoes and againt the instrument as suggested by Rose et al, (1982). The reading was recorded to the nearest 0.01 meters.

3.6.3 Needles and syringes: Seventy two (72) non puragenic 5m1 disposable needles and syringes were used along with tourniguet to bleed the participants as suggested by Gos, Gordon and Mentz (1991).

3.6.4 Stop Watches: Three (3) stop watches manufactured by the Tewelled Sports Company, Britain were used to time the duration of the work outs to tl rest second on each training session (Sharky, 1990).

3.6.5 Measuring tape: Three (3) non elastic horse brands (Model 51542 tape rules, made in China) were used to measure the hip and waist circumstances of the participants in centimeters (plowman and Smith, 1997).

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3.7 Procedure for Data Collection

3.7.1 Order of Testing: Before tests were conducted, formal letters of request seeking for consent were sent and approval were obtained.

(a) The Ethics Committee of the Post Graduate School of Ahmadu Bello University, Zaria.

(b) The sampled participants' willingness to participate in the study.

© The Department of Chemical Pathology, Jos University Teaching Hospital, Jos.

The following sequence of tetsts for body fat variables were conducted.

1. Recording of personal data

ii. Measurement of body mass index (BMI)

iii. Measurement of waist hip ration (WFIR)

iv. Measurement of hip and waist circumferences

All the measurements were conducted between 8.00 and l0.OOam pre and post exercise measurements were taken.

Blood samples were collected from the anticubital veins of the participants between 8.00 and 10.00 after 12 hrs overnight fast anad analysis pathology, Jos University Teaching Hospital, Jos. A base line characteristics and post exercise values of serum lipid and lipoprotein

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cholesterol were analyzed as suggested by Alesa, Barbara and Irena (2000). The following blood lipid and lipoprotein cholesterol were analyzed.

i. Serum Total Cholesterol (TC)

ii. Serum Triglycerides (TG)

iii. Low Density Lipoprotein Cholesterol (LDL-C)

iv. High Density Lipoprotein Cholesterol (HDL-C)

3.8 Experimental Control

Blood samples of the participants were collected in the morning between

8.00 — 10.OOam by haematologigists into EDTA containing tubes following 12 hours overnight fast, and stored at room temperature before analysis were performed at the Department of Chemical Pathology, Jos University teaching Hospital.

The researcher was assisted by physical Education Specialists in the conduct of the test. They helped to supervise the training programme and monitored the training intensity using an age predicted heart rate Max (HRmax) as suggested by Faeze and Sirvan (2011). Body weight in kilogramme and body mass index (kgIm2) were determined using a precision electronic scale (model 8958U manufactured in China). Height of the participants was measured using a wall mounted stadiometer (Model

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NJ07072 Export Inc. U.S.A). All the anthropometric characteristics of the participants were measured by each of the physical education specialists to ensure consistency. To reduce the effects of Pre-test on the results of all tests conducted, the participants were instructed not to engage in any other organized physical training programme rather than this test and not to smoke cigarettes or drink alcohol but can still maintain their normal nutritional habits throughout the course of the trial.

3.9 Exercise Training Methods

Venkateswarles (1992), reported that exercise training methods arer the procedures which involve repetitions of activities either in whole or in part in order to improve upon performance or health benefits. In recent years, several exercise training methods have been designed in every segment of the society for the society for the purpose of developing different components of performance in different sporting activities by enchanting cardiovascular functions to improve health and fitness through improvement in blood lipids, lipoproteins and body fat pattern (Plowman and Smith, 1997; Martin and Daniel, 1993; Heyward, 1998; Sharkey, 1990; Showvahair and Wells, 1995).

Venkateswarlu, (1995) reported further that exercise training methods include continuous training, interval training, circuit training, weight training, endurance training and resistant training. For the purpose of this

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study, interval training was used to achieve the purpose of which this study was conducted.

3.10 Interval Training

Interval training is the use of varying intensities throughout an exercise bout and is primarily associated with endurance activities that enhance cardiorespiratory performance and the maximum ability to remove carbon dioxide and utilize oxygen for health and fitness (Micheal, 1997, Perry, 2008) adding that it involves resting process between sets and work out. They further explained that resting periods between sets within a training session and as well as between work outs affect adaptation while the rest periods vary depending on the goals of the training programme. ‘for example, high intensities and power activities require longer periods of rest to regenerate creatine phosphate levels and re-establish intercellular Ph while less periods of time are required for moderate intensity exercise (Michael, 1997, Perry, 2008). The American College of Sports Medicine (2006), recommended that

2-3days per week using 8-10 exercise utilize most of the body large muscle during training programme, and this would go along way to improve health and fitness level of the individual. Sharon (2007), explained that the need for rest in interval training depends on the periods of rest which is generally determined by the intensity and duration of the exercise. Sharon (2007)

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reported that the basic health improvement of interval training is generally performed 3-5days a week with a day of rest.

Furthermore, interval training has been described as a type of discontinuous physical training that involves a series of low to high intensity exercise work outs interspersed with rest or relief periods. The high intensity periods s are typically close to anaerobic exercise while the recovery periods involve activity that are of lower, intensity (Perry, 2008). Interval training can be described as short period, of work and followed by rest with the aim to improve speed and cardiovascular fitness (Perry, 2008) Perry (2008) further explained that! interval training is referred to as any cardiovascular workout such as cycling, running, rowing and swimming, and is prominent in training routines for many sports adding that it is a technique particularly employed by runners and athletes. From different backgrounds.

Perry (2008), reported that interval training is favourite of coaches due to its effectiveness in cardiovascular build up and its ability to make runners and to exercise as it helps to improve aerobic capacity to exercise longer at varying intensities. For example, long interval workouts have been reported as the best method to reduce fat as it is popularly believed that fatty acid utilization usually occurs after at least 30 minutes of traihing (Tremblay; 1994; Boutcher, 2011).

Interval training can be an effective means of enhancing an athlete's lactate threshold which has been shown to be a significant factor determining performance for long distance running events. This methods of training has moderate intensity level for

the same duration. This is due to the metabolism boosting effects of high intensity intervals (Laursen, 2002, Talamian, 2007).

Interval training has been shown to improve performance of middle and long distance races and useful for rehabilitation purpose in sport medicine (Venkateswarlu, 1992).

Venkateswarlu (1982), early reported that interval training is a method of training that is characterized by formal fas-slow workouts and the work is divided into parts, While each part is performed with a predetermined intensity and followed by specific intensity duration and frequently of training, adding that there are different types of interval training which include the following:

- Fast interval training
- Slow interval training
- Pace interval training
- Sprint interval training
- Repetition interval training.

Venkateswarlu (1982), explained that sprint interval training constitutes work load of more than 80% of max speed at a distance of about 250m and has been useful for sprinters while the fast interval training constitutes a work load of 70-80% of maxspeed at a distance of 650m, and has been shown to be faster than competition speed in sprint endurance and endurance sport

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activities, adding that this type of interval training is used for speed and strength development in athletes.

Heyward (1980), reported that participation in exercise programme of interval training, requires increment in the work accomplished with little time and with less effort to enable a participants that is well fit to maintain a slower rate during work and to recover fast after work.

The American College of Sport Medicine (1991) reported that aerobic interval training should take place 3-5 times a week which should include; walking and jogging which have been shown to be effective in the development of fitness in individ (Franz and Hankey 1996) reported further that running requires more energy than walking while both provide effective activities that are useful in the development of cardio respiratory functions. Costil (1986) explained that training intensity for interval training may range between 70%- 80% of max heart rate but depending on the category of the athletes. Confirming further that interval training of 70% intensity is quite sufficient and effective enough to induce physiological changes that can enhance fitness.

3.11 Manupulation of Variables in Interval Training

According to Venkateswarlu (1982), interval training constitutes five important variables that are manipulated which include the following:

- The duration of work interval (rate and distance)
- The duration of recovery phase or relief interval
- The nature of recovery phase (type of activity)
- The number of repetitions during each training sessions in the interval training programme.
- The frequency of training per week.

Several studies have explained that during interval training, long duration repetitions are performed at a very low intensity and medium duration repetitions are performed at a moderate intensity while short duration

repetitions are carried out at high intensity (Venkateswarlu, 1982).

Venkateswarlu (1982), further explained that the intensity and duration of repetition depend on the particular energy systems that are to be developed in a given sport by the coach. Suggesting that aerobic metabolism should be improved more than the anaerobic metabolism. To this effect, repetitions carried out at low intensities are important for such sports.

Many methods have been used in order to determine the appropriate intensity of repetitions in interval training which include heart rate response during the repetitions (Mathew and Fox, 1976). For example, young men and women under 20 years of age should have their heart rate increased to 190 beats per minute during repetitions, men and women of 29-69 years of age

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should have their heart rate increased to about 140-l8Obpm respectively during each repetition for effective intensity of the repetition (Mathew and Foc; 1976; Sharon, 2008)

Venkateswarlu (1974) had earlier reported that the number of repetitions should depend on the athlete's conditions and it should be determined on the basis of pulse rate. Sharon, 2008; and (1992) had reported that with lower work intervals say 800m and above, 1.1 work relief ratio is recommended emphazing that the rest period (recover interval) should not be less than 30 seconds and not more than 90 seconds due to the dilation effect on the heart which takes place during the first phase of recovery interval thus remains important for the purpose of ensuring that the pulse rate drops to 120-125prn during the recovery period. Suggesting that example, if the purpose of interval training is to develop anaerobic metabolism, then the recovery interval should consist of active rest such as jogging because of the fact that active rest partially block the complete restoration of aerobic source of energy placing the athlete predominantly on anaerobic metabolism in subsequent work interval (Sprececherc, 1972 and Sharon (2007). It is well understood that jogging, provides the most physiological benefits during recovery interval due to the possibility of massaging effect of the heart (Sprececyherc 1972; Sharon, 2007; Nick, 2013; and Michael, 1997).

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3.12 Health and Fitness Benefits of Interval Training

Coe Nick (2013), reported that interval training induces health and fitness benefits which include the following:

- Strengthening the muscles that are involved in respiration in order to facilitate the flow of air in and out of the lungs to maintain health and fitness.
- Strengthening and enlarging the heart muscle to improve its pumping efficiency and reduce the resting heart rate called “aerobic conditioning”.
- Improving circulation efficiently and reducing blood pressure in athletes
- Increasing the total number of red blood cells in the body in order to facilitate oxygen transport.
- Improving mental health including reduction in stress and lowering the incidence of depression.
- Reducing the risk of cardiovascular disease and diabetes mellitus.

Furthermore, Coe Nick (2013), Sharon, (2007), and Micheal (1997) reported that interval training provides numerous performance benefits which include:

- Increased storage of energy molecules such as fats and carbohydrates within the muscles thus allowing for increased endurance.

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- Improving the neurovascularization of the muscle sarbmeres to increase blood flow through the muscles.
- Increasing speed at which aerobic metabolism is activated within the muscles, allowing a greater portion of energy for intense exercise to be generated aerobically.
- Improving the ability of muscles to utilize fats during exercise preserving intra muscular glycogen.
- Enhancing the speed at which muscles recover from high intensity exercise.

Nick (2013) concluded that interval training reduces muscle mass, during long distance running and it is not an effective approach to building lean muscle while it is only effective for fat loss suggesting that it can cause damage to the heart if used recklessly.

In exercise training programme, the use of percentage of maximal I-JR (% HRmax) method is based on the fact that the %HR has been shown to be associated with % V02 max (Heyward, 1998).

Swain, Abernathy and Smith (1994), reported that 69% and 89% HRMax corresponds with exercise intensity of 50% and 80% V02 max respectively, while the ASCM (1996) recommended that the target heart rate between 60% and 90% HRMax depends on the participant's initial fitness level. To use this procedure, the actual HRMax should be known or predicted either from the

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Heart rate response to submaximal workloads or from the formula 220 — age of the participant in years) (Pollock, Wilmore and Fox, 1990).

% Lower limit- This is HRMax x 0.6 and upper limit HR]Vlax x 0.85 as suggested by Hales (2007).

The % HRmax method shows a lower value when the relative intensity is used. To be more accurately used, the ACSM (1996) recommended that a correction factor should be used i.e by multiplying the target HR by 1.15 in order to obtain an accurate heart rate.

Wilmore and Costill (1994), reported that a minimum HR of 40%, 60%, 70% and a maximum of 75%, 85% and 90% exercise intensity has been recommended for individuals with low, average and high fitness levels. In this study, the training intensity started with estimated Heart rate of (40-50% HR max) and was increase to a predetermined level of 70% as the training progressed as suggested by Raul, Manuel, Sean and Ana (2010).

3.13 Training Protocol

Thirty six young adult males from Federal College of Education,

Pankshin participated in the study and were randomly assigned to either an experimental (N=18) or control (N1 8) group before the commencement of the training programme. Exclusion criteria in the study include the following (1) Presence of Diabetes mellitus, (2) hypothyroidism (3) hepatic disorder

(4) presence of cardiovascular event or myocardial infarction (5) Nephrosis (6) history of alcohol abuse (7) cigarette smoking, (9) taking lipid altering medication, (10) participants who had participated in any organized exercise training programme before the test. Informed consent was obtained from each participant upon entry into the study population. This study was approved by the Ethics Committee Board of Ahmadu Bello University, Zaria, and only the exercise group participated in the training intervention. The participants participated in a moderate intensity exercise programme 3 times per week under supervised condition for 12 weeks. The control group maintained their regular level of activity throughout the course of the trial. Each training session was preceded by 10 minutes warn up and stretching exercises and 10 minutes for cod down as suggested by ACSM (1990). The training intensity was established for each participant using an age —predicted heart rate maximum (HRMax) (220-age in years), Faeze and Sirvan, 2011).

At the beginning of the study, each training session was run for 45 minutes that corresponds to 40-50% at the participants HRMax Training duration and intensity was then increased incrementally in the second 4th week by 5 minutes and 5% HRMax. By week 9-12, participant was exercising for 60 minutes at an intensity of 70% HRMax. The participants ran 2 sets of 400m (2 x 400m intervals) with 1.1 work rest ratio including brisk walk and jogging

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at 70% of the participants age predicted max heart rate (HRMax) (Raul, Manuel, Sean and Ana, 2010).

Furthermore, to determine the intensity of the training programme, one repetition max (IRM) was estimated for each participant as suggested by Faeze and Sirvan (2011).

The training intensity consisted of 2 sets with submaximal effort to accomplish 8-12 repetitions per set in 3 days per week for 12 weeks with an interval of (61 workrest ratio) as suggested by Pollock, Franklin and Ballachy, (2000). Sean and Ana (2010).

During the 12 week interval training programme, each participant exercise heart rate was motivated to ensure proper training intensity. However, observation indicated that the training protocol was characterized by fatigue suggesting that the training induced significant physiological adaptation capable of producing health, fitness and performance benefits in the participants who participated in the study for 12 weeks.

3.13.1 Tests

In this study, the following tests were conducted:

(a) Waist and Hip Circumferences

The measurement of waist circumference is useful in individuals who are classified as overweight on the body mass scale (BMI) although increased waist circumference has been suggested to be marker for increased risk of degenerative disease in patients of normal weight (Depres, 1990). In this study, the participants waist and hip circumferences were determined using a tape rule to the nearest centimeter with the participants standing straight while the legs remain parallel wearing light clothing and without shoes. For the measurement of waist circumference, the tape was placed approximately midpoint between the lower margin of the last rib and the top of the iliac crest. The measurement was determined following normal respiration in the participants to the nearest 0.1cm. two measurements were determined and the average was recorded. The waist circumference indicates abdominal fat (Maffeis et a! 2000).

On the other hand, the hip circumference was determined when the tape was placed at the widest part area of the participant's buttocks as suggested by WHO, 2008b; Brian, Joseph and Williams,2007, Stian, Thomas, Wrrik and Stig, 2010. The values obtained for waist circumference measurement was divided by the values obtained for hip circumference in order to determine the waist hip ratio. In men, waist to hip ratio values 0.90 and 0.85 in women respectively have been suggested to be the strongest independent risk factor for cardiovascular disease than the body mass index (WHO, 2008).

3.10.2 Biochemical Measurements

Blood samples for lipid analysis were obtained from the antecubital vein of the participants who participated in the test following a 1 2br overweight fast as suggested by Raul, Manuel, Manuel, Sean and Ana, (2010) Kerry, Anita and Katherine (2005).

The pre training and post training blood samples were drawn in the morning using the technique described by Frankel, Elwood and Sweetnam (1996); Visch, Goss, Gordon and Mertz, (1991).

In this method, a tourniguet was tied around the participant's upper arm to ensure a brief arrest of the blood circulation to the forearm. The participants were instructed to clench their fists for the purpose of increasing the prominence of the participants' antecubital veins from which the blood samples were drawn. About 5m1 of blood was drawn by venupuncture from the antecubital veins of the left forearm of each participant. The blood of each participant was aspirated with syringe and needle under antiseptic condition and was ttransfered into labeled disposable container that contain fluoride oxalate non vaccum tubent that were free from anticoagulants. All the blood samples drawn from the participants antecubital veins were placed in the refrigerator as suggested by Burstein and Sammailk, (1960) before analysis were performed. Serum total cholesterol (TC), total triglycerides (TG), low

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density lipoprotein cholesterol (LDL-C), and High Density Lipoprotein Cholesterol were estimated as explained below:

i) Estimation of Total Cholesterol (TC)

Serum total cholesterol was estimated using enzymatic method as suggested by Allain and Rose Schwald et al (1974).

The total cholesterol (TC) was estimated after enzymatic hydrolysis and oxidation thus resulting to the formation of Quinoyemin as an indicator from hydrogen and 4-amino antipyrine in the presence of perioxide producing the reaction as follows:

- Cholesterol estertH2O $\xrightarrow[\text{Esterase}]{\text{Cholesteroi}}$ Cholesterol + fatty acids

- Cholesterol estertH2O $\xrightarrow[\text{Esterase}]{\text{Cholesterol}}$ Cholesterol -3- corr + H

- H202 + 4 amino antipyramine perioxide quinine +H20

In the procedure, well arranged and labeled test tubes were pipette reagent, black and standard sample. The mixture was mixed and incubated for 10 minutes at 250 C while the absorbance was measured spectophotometrically at 500nm within 60 minutes, and the reagent blank was used to zero the spectrophostometer. The formular below was used to determine the serum total cholesterol concentration.

Concentration of cholesterol (sample).

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Ab (sample) x con. (standard)

Ab (standard)

Serum total cholesterol rises with age and the rise is more marked in men

than in women. In the middle age, it may rise up to 302mg/d/=

(7.0mmol/l. A value above 200mgIdl=

(46mmol/l) is considered a risk factor for cardiovascular event.

ii. Estimation of Triglycerides (TGS)

Estimation of serum triglycerides was carried out using enzymatic method as suggested by Allain and Rosewald, (1974).

The triglycerides were spitted enzymatically by lipoprotein lipase produce glycerid and fatty acids. The glycerol produced was acted upon by glucokinase enzyme in the presence of ATP to form -3-P04 which inturn was oxidized by glycerol 3-P04 oxidase to form dilydroxyn- acephorphate and hydrogen perioxide. The hydrogen perioxide produced reaction with 4 amino anatipyrine, 4 cholesterol to form guinonelomi. In the procedure well arranged and labeled test tubes reagent blank and standard sample were pipette. The mixture was well mixed and incubated for 5-lOminutes at room temperature. The absorbance of sample and standard were measured at 500nm reagent blank to zero the equipment. The formular below was used to estimate the triglycerides concentration serum triglycerides:

$$\text{mgldl} = \frac{\text{AbTest} \times 100}{\text{Ab (standard)}}$$

Normal value is 80— 150mgbll = (0.9 — 1.7mmol)

While value above l5Omgldl or 1.7 mmol/L is consider a risk factor cardiovascular disease (Allain and Rosechwald, 1974; Kelley and Kelley, 2006; Kodama, 2007).

Centrifuged tube and 1ml of precipitating reagent was added to it. After the solution was well mixed, it was allowed to stand for a period of 10 minutes at a room temperature while the mixture was centrifuged twice for 2 minutes each at 4000 rpm and the supernatant represents the High Density Lipoprotein Cholesterol (HDL-C) fraction. The formula as shown below was applied.

HDL-C = Estmated cholesterol x 1.125

Where 1.125 = dilution factor (TieTz .1 990). However, high density lipoprotein cholesterol has been described as a negative risk factor for cardiovascular disease but the risk of coronary heart disease increases as the value falls below 0.9mmol!L and diminishes as levels become higher (Zimmet and Shaw, 2005).

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iii Estimation of Low Density Lipoprotein Cholesterol.

The low density lipoprotein cholesterol was estimated using the fried wald et al (1972) equation which relies on the estimation of total cholesterol (TC) triglycerides (TG) and High density lipoprotein cholesterol (HDL-C) without ultra centrifugation . the formula used for the estimation of the low density lipoprotein cholesterol was

$$\frac{TC - TG - HDL\text{-}C = LDL\text{-}C\ (MMOL/l)}{2.2}$$

This equation suggested that the ration of total cholesterol and triglycerides in very low density lipoprotein is relatively constant and that most of the triglycerides in plasma are contained in very low density lipoprotein cholesterol when the chyclomicrons are not detected. Cutler (2006) reported that an increased level of low density lipoprotein cholesterol concentration in plasma is atherogenic and it is associated with incidence of cardiovascular events.

iv Estimation of High Density Lipoprotein Cholesterol (HDL-C).

Serum high density lipoprotein cholesterol was estimated using phosohogunstic acid magnesium chloride (Mg2Cl) magnet as suggested by Lopez-Virella et al, (1977).

In this method, very low density lipoprotein cholesterol (VLDL-C) were precipitated in serum by the use of reagent after which the high density

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lipoprotein cholesterol (HDL-.C) was estimated in the clear supernatant. A pipette was used to draw O.5m1 of serum into.

Statistical Analyses

The data collected for the purpose of this study were analyzed. All the analysis were performed on an IBM compatible computers using the statistical package of social sciences at the Institute of Agricultural Research (IAR) Ahmadu Bello University, Zaria. The data were analyzed using descriptive statistics the (means, SD), independent samples t-tests fitness indices were determined by analyzing differences between pre and post training values. Independent samples t-tests were used to examine between group differences on the pre training descriptive data while the pre to post training differences for each group were tested with paired sample t-tests. Analysis were performed at a probability alpha level of P 0.05 for statistical significance.

CHAPTER FOUR

RESULTS AND DISCUSSIONS

4.1 Introduction

This study was conducted to assess the effects of interval training programme on serum lipid, lipoprotein cholesterol and body fat pattern of Nigerian adult males. The major serum lipid, lipoprotein cholesterol and body fat pattern variables included in the study were serum total cholesterol (TC), serum triglycerides (TG), low density lipoprotein cholesterol (LDL-C), high density protein cholesterol (HDL-C), serum lipid lipoprotein cholesterol a ratios, Bbody Mass Index (BMI), Conicity Index (CI), waist and hip circumferences and waist to hip ratio (WHR). These variables regarded as risk factors for cardiovascular disease (Kelley and Kelley, 2006; Cutler, 2000; WHO, 2005; Zimmet and Shaw, 2005; Kamlesh and Malamil, 2005). These variables were measured at base line (pre test) and after 12 weeks (post test). The interval training programme lasted for 12 weeks. The data collected before and after training were statistically analysed using the statistical package of social sciences (SPSS) at the institute of Agricultural Research (JAR), Ahmadu Bello University, Zaria.

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4.2 Results

Variable	Mean	SD	SE
Age (in years)	22.11	2.398	.5653
Height (m)	1.66	0.378	.0089
Weight (kg)	63.272	7.327	1.727

Table 4.2.1 above showed the mean, standard deviation (SD) and standard Error (SE) of the participants' physical characteristics of the participants that were used for this study. -

Examination of the table showed that the average age, height and weight of the participants were 22.11 ± 2.398 years, 1.66 ± 0.378 m and 63 .272 ± 7.327ktg.

4.3 Testing Of Hypotheses

Major Hypotheses

There are no significant effects of interval training on serum lipid, lipoprotein and body fat pattern of Nigerian adult males.

In order to test the major hypothesis, the major was decomposed into several subhypothesis which were tested as shown below.

Subhypothesis 1

There is no significant effect of interval training on serum total cholesterol (TC) of Nigerian adult males. To test this sub-hypothesis, the data collected on the post training values of serum total cholesterol for controlled and experimental group were analysed for difference using independent t-test sample statistics as shown in table 4.2.2 below.

Table 4.2.2: Independent t-test sample statistics for difference between post training values of controlled and experimental groups in their serum total cholesterol levels.

Group	Variable	Test period	Mean	SD	SE	df	T
Controlled	Total cholesterol (mmol/l)	Pretest Post	4.75 4.75	.355 .355	.084 .084	34	*2.291
Experimental	Total cholesterol (mmol/l)	Pretest Post test	4.75 4.21	.355 .355	.084 .084		

t(34) = 1.96, P < 0.05 * Significant

Examination of table 4.2.2 above showed post training values of control and experimental groups for serum total cholesterol (TC). The post training values of controlled and experimental groups were compared as shown in the table above. The result showed significant difference in the post training values of controlled and experimental group (t=2.29 1<0.01) suggesting that 12 week interval training significantly decreased serum total cholesterol level of the participants. Serum training total cholesterol did not decrease in the controlled group at post test. Therefore, the null hypothesis which stated that there is no

significant effect of 12 week interval training on serum total cholesterol level is rejected

Sub hypothesis II

There is no significant effect of interval training on serum triglycerides of Nigerian adult males.

To test this subhypothesis, the data collected on the post training values of serum triglycerides for controlled experimental groups were analysed for differences using independent t-test sample statistics as shown in table 4.2.3 below.

Table 4.2.3: Independent t-test sample statistics for differences between post- training values of controlled and experimental group in their serum triglycerides levels.

Group	Variable	Test period	Mean	SD	SE	df	T
Control	Serum TG (mmol/1)	Pretest Post test	1.111 1.111	.461 .1461	.0344 .0344	34	*3.128
Experimental	Serum TG (mmol/1)	Pretest Post test	1.111 .856	.355 .516	.084 .122		

t(34) = 1.96, p? 0.05 *significant

Table 4.2.3 above indicates post training values of control and experimental group for serum triglycerides (TG). The post training values of control and experimental groups were compared as shown in the table above. The result

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indicated significant changes at post training for the experimental group in (t=3 328 S 0.01), while the post training values remained unchanged in the control group suggesting that 12 week interval training significantly reduced serum triglycerides levels of the participants who underwent 12 week interval training. Therefore, the null hypothesis which stated that there is no significant effect of 12 week interval training on serum triglycerides is hereby rejected.

Sub hypothesis III

There is no significant effect of aerobic training on high density lipoprotein cholesterol (LDL-C) of Nigerian adult males.

To test this subhypothesis, the data collected on post training values of low density lipoprotein cholesterol were analyzed for differences between post training values in each of the groups using independent t-test sample statistics as presented in table 4.2.4 below.

Table 4.2.4: independent t-test sample statistics for differences between post training values of controlled and Experimental groups in their serum low density lipoprotein cholesterol.

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Group	Variable	Test period	Mean	SD	SE	df	T
Control	LDLC (mmol/1)	Pretest Post test	2.561 2.261	0.537 0.537	0.1266 0.1266	34	1.215
Experimental	LDLC (mmol/1)	Pretest Post test	2.261 2.261	0.537 0.537	0.1266 0.1266		

t(34), = 1.96, P?: 0.05 Not significant

Examination of the table above showed post training values of control and experimental groups for serum low density lipoprotein cholesterol (LDL-C). The post training values of control and experimental groups were compared as shown in the table above. The result indicated that the experimental group showed no significant changes (t=1.2150.01) suggesting that 12 weeks interval training did not induce changes in serum low density lipoprotein cholesterol levels of the participants who participated in 12 week interval training progrgamme.

Therefore, the null hypothesis which stated that interval training has no effect on low sensity lipoprotein cholesterol is retained.

Subhypothesis IV

That is no significant effect of 12 week interval training on high density lipoprotein cholesterol (LDL-C) of Nigerian adult males.

To test thi subhypothesis, the data collected on the post training values of high density lipoprotein cholesterol for control and experimental groups were

analyzed for differences using independent t-test sample statistics as shown in table 4.2.4 below.

Table 4.2.5: Independent t-test sample statistics for differences between post training values of control and experimental sample in their high density lipoprotein cholesterol levels.

Group	Variable	Test period	Mean	SD	SE	df	T
Control	HDLC (mmol/1)	Pretest Post test	1.078 1.078	.1003 .1003	.0236 .0236	34	*9.295
Experimental	HDLC (mmol/1)	Pretest Post test	1.078 1.078	.1003 .1.003	.0236 .0236		

t(34), = 1.96, P? 0.05 ** significant

Examination of table 4.2.4 above showed post training values of control group and the experimental group for High density lipoprotein cholesterol (HDL-C). the post training values of controlled and experimental groups were compared as shown in the table above. The result showed significant increase (t=9.295 0.01) in high density lipoprotein cholesterol of the participants that underwent interval training for 12 weeks. Therefore, the null hypothesis which stated that 12 week interval training has no significant effect on high density lipoprotein cholesterol of Nigerian adult males is rejected.

Subhypothesis V

There is no significant effect of interval training on serum lipoprotein ratios of

Nigerian adult males. To test this sub-hypothesis, data collected on the post

training values of serum lipid/lipoprotein ratios for control and the experimental groups were analyzed for differences using paired sample t-test statistics as shown in table 4.2.5 below.

Table 4.2.6: Paired sample t-test statistics for differences between post training values of control and experimental groups in their serum lipid/lipoprotein ratios.

Group	Variable	Test period	Mean	SD	SE	df	T
Control	Te/HDL-C (mmol/1)	Pre-test Post test	3.9368 3.9368	.64473 .64473	.15196 15196	34	
Experimental	Te/HDL-C (mmol/1)	Pre-test Post test	3.9368 3.9368	.64473 .64473	0.15196 0.15196		
Control	Te/HDL-C (mmol/1)	Pre-test Post test	1.0356 1.0356	0.12573 0.12573	0.15196 0.15196		
Experimental	Te/HDL-C (mmol/1)	Pre-test Post test	1.0356 0.6137	0.12573 .23084	0.15196 0.15196		
Control	Te/HDL-C (mmol/1)	Pre-test Post test	2.4051 2.4051	.61402 .61402	0.02963 .0544		
Experimental	Te/HDL-C (mmol/1)	Pre-test Post test	2.3122 2.3122	40958 40958	.14475 14475		

t(34), = 1.96, P? 0.05 ** significant

Examination of table 4.2.5 above showed post training values of control group and the experimental group for serum lipid/lipoprotein ratios. The post training values of control and experimental group were compared as shown in

the above. The result showed significant decrease in the triglycerides/high density lipoprotein cholesterol ratios compared to controlled group in those variables suggesting that the null hypothesis which stated that 12 week interval training has no significant effect on serum lip/lipoprotein cholesterol ratio his here by rejected. However, clinic evidence has shown that a strong association of the ratio of triglycerides/high density lip/protein cholesterol ratio is hereby rejected. However, clinical evidence has shown that a strong association of the ratio of triglycerides/high density lipprotein cholesterol with the risk of conoray heart diseases exists. This suggests a metabolic interactions between the triglycerides and the cholesterol ester-richliporin increasing the risk of myocardial infarction while a high ratio may be a good indicator of abonomal cholesterol metabolism (Gariano, 1997; Kelley,2005:

Willams, 1999 and freedom, (2003)

Subhpothesis VI

There is no significant effect of 12 week interval training on body fat pattern of Nigerian adult male.to test this subhypothesis, the data collected on post training values for body fat pattern of the controlled and experiment group were analyzed for difference usingindependent t-test sample statistics shown in table4.2.6 below.

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Table 4.2.7; independence t-test sample for statistics for differences between the post training value of the controlled group and the experimental group in their body fat pattern variables.

Group	Variable	Test period	Mean	SD	SE	df	T
Control	Body mass index	Pre-test Post test	21.61 21.61			34	
Experimental	Body mass index	Pre-test Post test	22.58 22.58				
Control	Conicity index	Pre-test Post test	21.33 21.33				
Experimental	Conicity index	Pre-test Post test	20.612 20.612				
Control	Hip Circumference	Pre-test Post test	34.22 34.22				
Experimental	Hip Circumference	Pre-test Post test	33.33 33.33				
Control	Waist to hip ratio	Pre-test Post test	.0894 0.894				
Experimental	Waist to hip ratio	Post test Post test	.0878 .0878				
Control	Waist circumference	Post test Post test	31.94 31.94				
Experimental	Waist circumference	Pre-test Post test	31.94 30.22				

t(34), = 1.96, P< 0.05 * significant

Table 4.2.6 indicates that post training values of the controlled group and the experimental group were compared. The result showed that there was significant difference in waist circumference between control and experimental group (t=2.532 <0.05) suggesting that 12 week interval training positively influenced only waist circumference of Nigerian adult males. In this study, body fat pattern was determined by five independent variables.

These include body mass index, conicity index, hip circumference, waist to hip ratio and waist circumference. A number of studies have reported that wqaqist circumference is cardiovascular risk factor and more practically correlate of abdominal fat distribution than waist to hip ratio and has been suggested to be the best measure of body fat pattern compared to other variables (Lean, 1995; Williams, 1999, and Cutler, (2000). Therefore, the null hypothesis which stated that there is no significant effect of 12 week interval training on body fat pattern of Nigerian adult males is hereby rejected.

DISCUSSION

It has been clearly explained, suggested and documented that serum lipids and lipoprotein cholesterol have negative effect on cardiovascular health (Blessing, 1995, Cutler, 2006; Kelley, 2005). For example, serum lipid and lipoprotein cholesterol levels are predictive of artherogenic lesion and cardiovascular disease in adult (Kelley and Kelley, 2006). However, the early stages of artherogenic begin in childhood. Fatty streaks are found in arteries of three years old children and both fatty streaks and fibrous plaque appear in the coronary arteries during the second decade of life (Strong et a!, 1962). This view was supported by Kelley et al (2005), who reported that early artherogenic lesions are associated with levels of serum lipids and lipoprotein cholesterol fraction in children and adolescents, adding that there is relationship between serum lipoproteins and, stroke, high blood pressure and early atherosclerosis. Selberg and Strong, (1983), reported that multiple serum lipid and lipoprotein determination at six months to 2 years of age and as well as knowledge of subsequent excessive weight grain may help to identify people likely to have elevated serum lipid and lipoprotein cholesterol levels in later childhood.

Available research evidence suggests that elevated levels of serum cholesterol are the primary risk factors in the development of both artheroscierosis and coronary heart disease (Drygas et a!, 2000; Kelley, 2005;

Tian et al 2010; Castali et al, 1986 and Guo, 2011). Recent evidence, Jatau,

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(2012); Gua, (2011) and Tian (2010) indicated that the manner in which cholesterol is transported in the blood may be more critical in the development of cardiovascular disease than all total blood cholesterol concentration.

Serum studies (Viyutran, 1983, Kelley and Kelley 2006; Williams, 1999), have clearly shown that cholesterol transported by low density lipoprotein cholesterol amy infiltrate the artery and contribute to the artheroscierosis process. Kodama, (2007); Kelley and Kelley, (2006), reported that cholesterol carried by high density lipoprotein may be transported from the arterial wall to the liver for catabolism and excretion and this have protective effect. Epidemiological studies support this concept by indicating that populations with high levels of high density lipoprotein cholesterol have a long incidence of cardiovascular disease (Blessing, 1995; Thomas, 2004; Drygas, 2000; Toifrey, 2000; and Kdama, 2007). Based on this, several studies have shown that regular exercise training has favourable effects on total cholesterol (Kodama, 2007; Drygas, 2000; Kelley and Kelley, 2006 and Morgan, 2004).

This study was conducted lto assess the effect of interval training on total cholesterol of Nigerian adult male. The training lasted about 3 times a week in every training session for 112 weeks. This study clearly indicated significant decease (5%) in total cholesterol levels due to 12 week moderate intensity interval training which was conducted on alternate days at 70% of maximal heart rate. This finding was in agreement with the finding of Superko, (1991;)

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Narayani, (2010) and Guo (2001;) who reported that interval training significantly decreases total cholesterol due to body weight and fat reduction, but William, Kraise and Flood (1986), reported that no significant relationship exist between physical activity and total cholesterol. Furthermore, the decrease in total cholesterol level due to 36 sessions of moderate intensity interval training that lasted for 3 0-60 minutes in very training session was about 5-7 mmol/L. this significant decrease also supports the previous studies which showed a significant decrease in total cholesterol due to 6 or more weeks of moderate to vigorous interval training in both men and women (Comlonid, 2015; Tian, 2010; Kelley, 2011; Jia, 2006; Kelley, 2005; Kelley, (2004;) and Thomas, 2004). Thus, the results of this present study suggests that the improvement of 5% reduction in total cholesterol can be expected due to moderate intensity of interval training programme conducted for 12 weeks on alternate days.

Jatau, (2004); Mohammed, (2003) and Tolfery, (2000), reported significant decrease in total cholesterol following 8—16 weeks of interval training programme such as walking, jogging and cycling. The result of this study confirmed these previous results by showing significant decrease in total cholesterol due to 12 weeks of moderate intensity interval training. Recent meta analytic studies of Kelley, (2005), revealed that more than three studies conclusively found significant improvements in total cholesterol following 8— 16 weeks of interval training, this present study supports this findings.

Triglycerides have been described as natural fat composed of fatty acids and glycerol. High levels of triglycerides are associated with cardio vascular disease (Ochi, 2007). Williams (1996) reported that triglycerides are transported in plasma in the form of chylomicron and very low density lipoprotein (VLDLC) and are present in small amount in low density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C). Several studies have shown that exercise training programme which increase the activity of several enzymes such as hepatic lipase and protein are modified to promote more rapid catabolism of triglycerides to result to greater production of high density lipoprotein which is a protective mechanism for cardiovascular health (Williams and Millers 2004). Several studies have shown beneficial effect of exercise on triglycerides. For example, Gao (2011), reported a significant decrease in triglycerides as a result of 3 months of physical activity on Japan workers while Kelley (2005), study confirmed a significant decrease in triglycerides due to 12 weeks of exercise training in overweight and obese adults. This present study supports the finding of Kelley (2005), and Pong (1993), who reported that moderate intensity interval training produced reduction in triglycerides, a favourable effect on blood lipid that has a protective mechanism against coronary heart disease. The results of this study clearly indicated significant decreases in triglycerides due to 12 weeks moderate intensity interval training which wasconducted on alternate days at 60% of maximal heart rate. The training lasted

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30 — 60 minutes in every training session. The result of the present study suggest that the improvement of 30% in triglycerides can be expected as a result of moderate intensity interval training programme conducted for 12 weeks on qalternate days. This result is in agreement with the study of Dinstine (2000) who reported that triglycerides are always lower in moderate physical activity.

Mannineu (1992), in his studies reported that triglycerides have been shown to be an independent risk factor for cardiovascular disease or a predict risk factor for coronary disease adding that triglycerides decreases significantly following acute bout of moderate physical activity withparticipants who have high level of triglycerides. This present study revealed that 12 week interval training at moderate intensity decrease triglycerides level. This result is in agreement with the finding of Sarika Manpreet Jaspal (2010) who reported that physical training reduces triglycerides concentration. Fahri (2010), reported that moderate intensity physical activity results in sustained reduction in serum triglycerides, the decrease he said was not attributable solely to weight reduction but it has been shown that serum triglycerides concentration decreases during exercise progression while Mannel and Raul (2010), suggested an effective mechanism for improving for improving cardiovascular protection with physical that results in reduction of 90% on triglycerides.

Low density lipoprotein cholesterol (LDL-C) has been described as a protein complex in the blood that facilitates the transport of cholesterol, triglycerides and phospholipids (Brown and Cargill, 2002). Low density lipoprotein cholesterol (LDL-C) is a molecule of very low density lipoprotein with most of the triglycerides removed while almost all of the cholesterol remaining. It is a product of very low density lipoprotein metabolism (Scann, 1978). Studies have shown that from puberty until menopause, women generally have lower cholesterol and low density lipoprotein values than men.

After menopause, their values equal or exceed that of men (Heiss, 1980, Kodama, 2007, Kelley 2005). Low density lipoprotein cholesterol is often called bad cholesterol because it transports or carries cholesterol in the blood circulation to various body cells and deposits it to cause the risk of cardiovascular events. About 50% - 60% of cholesterol is transported to the cells and the desirable level of low density lipoprotein cholesterol is below 3.3. mmol/L with an optimal level of 11.07 mmol/L (Heiss 1989; Kodama 2007; Kelley, 2005). When low density lipoprotein cholesterol (LDL-C) elevated, cholesterol begins to accumulate in vessels walls and restrict blood flow (Scann 1978). Clinical evidence suggests that low density lipoprotein cholesterol may directly contribute to the cellular alterations of the inner walls of arteries which may ultimately lead to the development of atherosclerosis plaque (Scann, 1987), adding that oxidized low density lipoprotein cholesterol plays a dangerous role by reducing the level of nitric oxide, a chemical

substance that helps relax the blood vessels to allow free flow of blood. The studies of Drygas, (2006); Kannel, (1986); Castelli, (1986); Cutler, (2006), reported that an increased level of low density lipoprotein concentration in plasma is atherogenic, and it is associated with incidence of cardiovascular events.

However, studies have demonstrated significant decrease in low density lipoprotein cholesterol level due to exercise training to improve health and fitness. (Cutler, 2006; Kelley, 2006; Kodama 2007) exercise training for 6 -22 weeks performed for 3-5 days a weeks at an intensity of 50-75% of maximal heart rate has been shown to improve low density lipoprotein cholesterol (Linder, 1993; Blessing, 1995; Kelley 200o5; Tolfery, 2000; Guo, 2011). This study was conducted to assess the effect of 12 weeks interval training on low density lipoprotein cholesterol levels of Nigerian adult males. The study clearly indicated that 12 weeks interval training showed insignificant effect on low density lipoprotein cholesterol (LDL-C). These findings are in agreement with the regression analysis of data from large scale epidemiological studies that failed to consistently demonstrate a relationship between physical activity and low density lipoprotein cholesterol (LDL-C) and total cholesterol)TC) (Gordons 1988). Similarly, Williams, Kraise and Wood (1986) reported no significant effect of physical activity on low density lipoprotein cholesterol levels (LDL-C) in active versus inactive people while

Krumel (1993), reported level of small dense low density lipoprotein particles in male runners versus non runners. These findings support the results of this present study which demonstrated insignificant effect on low density lipoprotein cholesterol (LDL-C) following 12 weeks of interval training at moderate intensity for 3 times per week on alternate days.

High density lipoprotein contains high concentration of protein and much smaller concentration cholesterol and phospholipids while small quantity is synthesized in the intestinal epithelium during food absorption of fatty acids (Neiman, 1998). Previous studies (Gordon et al, 1977; Lastelli and Gordon, 1971), suggested that high density lipoprotein cholesterol (HDL-C) offers a protective mechanism against the development of cardiovascular disease. According to Neiman (1998), high density lipoprotein cholesterol is considered to be the most powerful lipid parameter for predicting coronary heart disease in people of all ages adding that the primary function of high density lipoprotein is to transport cholesterol from the tisues to the liver to synthesize bile acids. The studies of wood and Haskell (1979), reported that females have high contents of high density lipoprotein cholesterol than males which help to protect them from developing cardiovascular disease suggesting that this may be partially due to the relatively higher lipoprotein lipase activity in women while estrogen seems to play a major role in lowering the risk of cardiovascular disease in premenopausal women.

Studies have shown that exercise training programmes have indicated improvement in high density lipoprotein cholesterol (Lakka, 1992; Renkatesawrhi, 1992; and Hertzberg, 2004]. For example, Hertzberg (2004), reported that regular moderate intensity physical activity has a beneficial effect on blood lipids profile thus resulting in a increase in high density lipoprotein cholesterol fractions to improve health and fitness. The present study assessed the effect of 12 weeks moderate intensity interval training on high density lipoprotein cholesterol of Nigerian adult males which was conducted 3 times per week. The result of this study revealed that 12 weeks interval training which was conducted 3 times per week showed significant increase [3%) in high density lipoprotein cholesterol of the participants. This finding suggested that engaging in interval training can positively improve high density lipoprotein cholesterol (HDL-C) as an effective mechanism against cardiovascular disease. The significant increase in high density lipoprotein could be due to the activity of the lipoprotein lipase (Kelley, 2005 and Laaksonen 2009). The finding of this study is in agreement with the study of Hertzberg, 2004; Kokkinos et al, 1995 and William (1998), who reported that exercise training programme has shown significant increase in high density lipoprotein cholesterol levels in individuals. The beneficial effect of interval training on high density lipoprotein cholesterol may be attributed to enhanced levels of lipoprotein lipase (LPL) activity (Fieldman and Kinderman, 1989; Goran and Gower, 1999, Heyward, 1998 and Smith 1999

adding that every 10 minutes prolonged exercise persssion was associated with an increase of about 0.06 mmol/L (2%) in high density lipoprotein cholesterol level. Fieldman and Kindemen (1989) and Smith (1999), reported further that participants in exercise training with BMI less than 28, and with total cholesterol level of 5 .7mmol/h as an increase in high density lipoprotein cholesterol level than those with BMI of 28 or more that have total cholesterol level of 5.7mmol/h.

Studies have shown that serum lipids and lipoprotein cholesterol ratios may offer a refined risk assessment by simultaneously considering both anti atherogenic and atherogenic parameters (Tian 2010; Goran and Gower 1999). Elevated fasting triglycerides levels have been shown to be a useful marker for cardiovascular disease especially when high density lipoprotein cholesterol levels are considered (Drygas, 2000; Cutler 2006). The ratio of cholesterol ester rich lipoprotein level is well established prediction of coronary heart disease (CHD) (National Consensus Development Panel on Triglycerides 1993), adding that the high ratio may be a good indicator of normal cholesterol metabolism. Furthermore, increase in triglycerides/high density lipoprotein cholesterol ratio is associated with 49% increase in the risk of myocardial infarction (Drygas, 2000). It appears that total cholesterol/high density lipoprotein cholesterol and triglycerides/high density lipoprotein cholesterol ratios have been suggested to be predictors of arterial stenosis while the

ratio of triglycerides index is a highly significant independent predictor of myocardial infarction (Golbount 1997). Risk for cardiovascular events is significantly higher when the triglycerides/high density lipoprotein cholesterol is greater than 5, therefore the most risk common usage is total cholesterol/high density lipoprotein cholesterol ratio is 2:3 as the cut off point for cardiovascular disease Qia, 2006; Thomas, 1999; Kelley and Kelley, 2006). Increase in the ratio of Total cholesterol/high density lipoprotein cholesterol and triglycerides/high density lipoprotein cholesterol is associated with the shift of high density lipoprotein cholesterol particles to smaller size (Jia, 2006).

Plasma low density lipoprotein cholesterol/high density ratio and high density lipoprotein subclass distribution profiles indicates that an elevated high density lipoprotein cholesterol concentration in plasma is atherogenic where as a high density cholesterol level is cardio protective [Morgan, 2004; Farhan and Michael 2008). Several studies have indicated that the use Of ratio of low lipoprotein cholesterol/high density lipoprotein cholesterol has been shown superior to the use of high density lipoprotein cholesterol above indicating further that the ratio of low density lipoprotein cholesterol is better assessment by concurrently accounting for arthrogenic and protective mechanism as high density lipoprotein subfraction (Ridker; 2005).

Furthermore, low density lipoprotein cholesterol/high density lipoprotein cholesterol ratio has been associated with 50% increased in the risk of myocardial infarction (Morgan, 2004). The studies of (Ridker, 2005; Tian, 2010 and Gaiano, (1997), suggested that with elevation of low density lipoprotein cholesterol/high density lipoprotein cholesterol ratio, the contents of small size high density lipoprotein cholesterol 2, [HDLC2) and (1-IDL- C3b) increase progressively while those of HDL-C2A, 2b decrease progressively.

However, several studies have shown that exercise training influences lipid/lipoprotein ratios. For example, positive changes relatedto exercise training include reduction in triglycerides/high density lipoprotein cholesterol ratio, total cholesterol/high density lipoprotein cholesterol ratio and low density lipoprotein cholesterol/high density lipoprotein cholesterol ratio due to 8 - 16 weeks of walking, jogging and cycling (Thomas et at, 2004); Kelley and Kelley, 2006). The present study was conducted to assess the effect of 12 week interval training on lipid profiles of Nigerian adult males. The study revealed a significant reduction of 20% in triglycerides/high density lipoprotein cholesterol ratio due to 12 weeks interval training, but total cholesterol/high density lipoprotein cholesterol and low density lipoprotein cholesterol/high density lipoprotein cholesterol ratios

remained unchanged. These findings is in agreement with the study of Thomas, (2004); Kelley and Kelley, (2006), who reported that exercise training significantly reduces triglycerides/high density lipoprotein cholesterol ratio. However, clinical evidence has shown that a strong association of the ratio of triglycerides/high density lipoprotein cholesterol with the risk of coronary heart disease exists. This suggests a metabolic interactions between triglycerides and the cholesterol ester rich lipoproteins in increasing the risk of myocardial infarction (Mi) while a high ratio may be good indicator of abnormal cholesterol metabolism (Gazano, 1997). The reduction in triglycerides / high density lipoprotein cholesterol ratio was attributed to the activity of several enzymes such as hepatic lipase and protein lipase which were modified in order to promote more rapid catabolism of triglycerides to result to greater production of High density lipoprotein cholesterol which is cardio protective (Williams and Miller, 2004].

In the other hand, body fat patterns have been reported to influence health and fitness of people in the general population. For example, Manson (1995), reported that an increase in central adiposity in women is a greater predictor for heart disease, diabetes mellitus and death. Similarly William (1999), indicated that regional fat distribution is a concept that represents the anatomical distribution of fat over the body and may be related

with several major health problems in the general population. Studies have demonstrated that body mass index and waist circumference have more adverse effects on triglycerides and high density lipoprotein cholesterol (Durmin 1984), while Knowler, et al, (1995) reported that obesity is also associated with increased mortality of various lipid and lipoprotein abnormalities that influence cardiovascular health. Available research evidence suggests that overweight and abdominal obesity have been shown to constitute health disorders which include coronary heart disease, pulmonary disease, reproductive disorders, gallstone disease, type 2 diabetes mellitus, cancer of the colon, prostate cancer, breast cancer, cancer of the colon, cancer of the cervix, ovarian hypertension, degeneration joint disease, low back pain and elevated triglycerides [Flier, 2001). Epidemiological studies support this concept by indicating that populations with overweight and abdominal obesity have a higher incidence of cardiovascular disease [cutler, 2006; Hill etal 1994).

Epidemiological studies have demonstrated that exercise training positively influences body fat patterns variables to improve health and fitness. For example, Hill et al, (1994), reported that exercise training has been shown to be an independent environmental factor which determines the total level of energy expenditure, and that increased in physical activity is a

determinant of fat balance, and it is an important factor for weight regulation in individuals to improve health. The present study was conducted to assess the effect of 12 weeks interval training on serum lipids, lipoprotein including body fat patterns. The finding of the study revealed significant reduction in waist circumference of the participant while other fat patterns variables remained unchanged, suggesting that waist circumference is the best measure of body fat patterns and it has been found in several studies to correlate better with intra abdominal adipose and showed increased sensitivity and specificity for measuring central fat distribution and assessing metabolic risk proffle related to body flit distribution (Williams, 1999; Kelley and Kelley, 2006; Depres, 1994 and Golboerty, 1997). The finding of this study is in agreement with the study of Schwartz et al, (1991), who reported a greater loss in visceral adipose tissue in both young and old men following interval training. The finding of Schwartz et al, (1991) concluded that interval training alove can reduce body fat patterns, and has been suggested to be an important factor for maintaining body fitness and body weight in the general population. Exercise training in this study has been shown to be protective against the incident of cardiovascular disease.

There has been consideration intervention in recent years on how altered lipid metabolism and body fat patterns could contribute to the

incidence of cardiovascular disease and muscle insulin resistance. For example, several animals and human studies of insulin resistance have shown decreased triglycerides contents due to exercise training indicating possible mechanistic links between lipids accumulation and insulin resistance which go beyond the classic glucose cytrit (Bell, 2000; Hegaty et al, 2003), However, the identification of these relationship between exercise training, lipid, lipoprotein and body fat patterns during growth and development remains unclear. Recent meta analytic studies of Kelley et al, 2005.showed that more than three studies conclusively found significant improvements in total cholesterol (TC), triglycerides (TGs) high density lipoprotein cholesterol and waist circumference but not in low density lipoprotein cholesterol. This improvements equivalent to 2% of total cholesterol (TC), 11% for triglycerides (TG), 3% for high density lipoprotein cholesterol and 3% for waist circumference. These studies further revealed significant positive association between increase in high density lipoprotein cholesterol and increase in V02 max which shows a negative association (Kodama et al, 2007). The main results of these meta analytic of several studies showed an increase of 0.O65mmol/L in high density lipoprotein cholesterol with a minimal exercise of 9.00k/cal energy expenditure per week or 120 minutes per week. They also showed that every 10

minutes prolonged exercise persession was associated with an increase of 0.O6mmo/h in high density lipoprotein cholesterol level. However, there was no significant association between exercise frequency and intensity. But participants with BIV11 less than 28 and total cholesterol level of 5.7mmol!L or more experience about 0.054 mmolIL increased in high density lipoprotein cholesterol level than those with BMI of 28 or more and total cholesterol level of 5.7mmoIJL [Kodama et a!, 2007). The main results of this present study showed significant improvements in total cholesterol (TC), triglycerides (TGs), high density lipoprotein cholesterol and waist circumference. This improvement was equivalent to 2% of total cholesterol (TC), 3% for triglycerides (TGJ 5% for high density lipoprotein cholesterol, 2% for triglycerides I high density lipoprotein cholesterol and 3% for waist circumference.

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATIONS

5.1 Introduction

This chapter presents the summary, conclusion and the finding of the study on effects of interval training on serum lipids, lipoprotein and body fat pattern of Nigerian adult males. Recommendation on the use of training methods to improve health and fitness is also included inthis chapter.

5.2 Summary

The purpose of this investigation was to assess the effect of 12 week interval training on serum lipid lipoprotein cholesterol and body fat pattern of Nigerian adult males. The major lipids and lipoprotein cholesterol included in the study were serum total cholesterol (TC), serum triglycerides (TG), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C),TCIHDL-C, TG/HDL-C and LDL-C/HDL-C ratios while fat pattern variables that were examined were body mass index (BMI), conicity index (CI) hip and waist circumferences, and waist to hip ratio (WHR). These variables are regarded as risk factors of cardiovascular disease (Kelley and Kelley, 2006; Cutler, 2006; Drygas, 2000; and Williams 1999).

In the conduct of study, 36 adult males who were the participants were randomly assigned based on meeting the included criteria to either experimental group (N =18) or control group (N=1 8). The experimental group underwent 12 week interval training while the control group maintained

regular activity. The data obtained on pre and post exercise values were analyze using descriptive statistics of means and standard Deviation (SD). The result obtained were as follows:

1. Twelve week interval training significantly reduced serum total cholesterol of Nigerian adult males. This reduction was significant at the end of the 12 week of training.

2. There was significant reduction in serum triglycerides following 12 week of interval training of Nigerian adult males. The decrease was significant at the end of 12 week of training.

3. Twelve week interval training did not produce changes in low density lipoprotein cholesterol (LDL-C) of Nigerian adult males. This may bedue to lack of excessive weight loss of the participants who participated in the study (Depress, Tzotzas and Varandy, 2007).

4. Twelve week interval training produced significant increase in the high density lipoprotein cholesterol at the end of the training prgramme.

   This significant increase may be attributed to enhanced levels oflipoprotein luipase (LPL) activity and hormous sensitivity which areresponsible for adipose tissue lipolysis (Goran, Naggay and Gower, 1999; Heward, 1998; Plowman and Smith, 1999)

5. Twelve week interval training induced significant reduction in trilycerides/high density lipoprotein cholesterol ratio but did notproduce changes in total cholesterol/high density lipoprotein

cholesterol ratio respectively. This suggests that a general shift toward smaller size high density lipoprotein cholesterol was accompanied by an increase of the LDL-C/HDL-C ratio (Assmann, 1998; Manninan, 1992 and Castelli, 1982).

6. Twelve week interval training showed significant reduction in waist circumference of the participants. Waist circumference has been described as the best measure of body fat pattern and it has been found in several studies to correlate better with intra abdominal adipose and show increased sensitivity and specificity for measuring central fat distribution and assessing metabolic risk profile related to body fat distribution (Williams, 1999; Kelley and Kelley, 2006; Depres, 2007 and Golboerty, 1997).

7. Twelve week interval training did not induce changes in body mass index (BMI), conicity index (CI), hip waist to hip ratio (with) and hip circumference. This may be due to lack of training effect.

5.3 Conclusion

Based on the finding and limitation of this study, the following conclusions were drawn.

1. The present study indicates reduction in total cholesterol (TC), triglycerides (TG) and triglyceride/high density lipoprotein cholesterol ratio of Nigerian adult males following 12 weeks of interval training at moderate intensity. High density of blood cholesterol, triglycerides and

triglycerides/High density lipoprotein cholesterol ratio have been shown to be risk factors of atherosclerosis and many lipids associated with ailment like obesity, heart attacks, stroke and kidney failure.

2. The present study indicates that 12 weeks interval training at moderate intensity significantly increased high density lipoprotein cholesterol of Nigerian adult males. High density lipoprotein cholesterol gives protection against many cardiac events and obesity because it transports cholesterol from the peripheral tissues to the liver. Although genetic factor recline behind these lipid disorders, in most cases it is allied with diet in saturated fats. The increase in high density lipoprotein cholesterol was attributed to lipoprotein cholesterol lipase activity (Kilamura, 1994).

3. The present study revealed that 12 weeks interval training did not produce effect on low density lipoprotein cholesterol. Low density lipoprotein cholesterol (LDL-C) has been suggested to be a major carrier of cholesterol towards tissue having athiagenic potential.

4. The present study indicated a significant reduction in waist circumference of Nigerian adult males following 12 weeks of interval training at moderate intensity, measurement of waist circumference is useful in individuals who are classified as overweight on the BMI scale, although increased waist circumference has been suggested to be

marker for increased risk of degenerative disease in patients of normal weight.

5. The present study did not produce changes in waist to hip ratio (WHR), Body mass index (BMI) and conicity index of Nigerian adult males following 12 weeks interval training. Waist to hip ratio has been suggested to be the strongest independent risk factor for cardiovascular event than the body mass index.

5.4 Recommendations

1. The result of this study showed that 12 week interval training significantly decreased serum total cholesterol (TC), serum triglycerides (TG), triglycerides! high density lipoprotein cholesterol ratio, waist circumference and significantly increased high density lipoprotein cholesterol of Nigerian adult males. This suggests that interval training positively influences health and fitness by inducing favourable changes in serum total cholesterol, triglycerides, triglycerides! high density lipoprotein cholesterol and waist circumference in individuals. It is therefore recommended that Nigerian adult males should always participate in interval training programme to maintain health and fitness.

2. This study did not find any training effect on serum low density lipoprotein cholesterol (LDL-C), total cholesterol! high density lipoprotein cholesterol ratio, (TC!HDL-C), low density lipoprotein cholesterol!high density lipoprotein cholesterol ratio (LDL-C/HDL-C), body mass index (BMI), conicity index (CI), hip circumference and waist - hip - ratio. Therefore it is recommended that Nigerian adult males should participate in regular interval training of intensity. This may induce favourable alterations of these variables as risk factors for cardiovascular disease to improve health and fitness.

3. It is recommended that Nigerian adult males within the range of 18-25 and above in the general population should always participate in interval training progrmme of moderate to high intensity for life time to maintain health and fitness for productivity.

Recommendations for Further Studies

1. Based on the findings of this investigation, it is recommended that future studies should focus attention on the effects of interval training on serum lipid, lipoprotein cholesterol and body fat pattern variables as risk factors for cardiovascular disease in a large randomized trial for both experimental and control groups using different training intensities.

2. Future studies should examine whether healthy individuals with low base line serum lipid profiles can use moderate intensity interval training protocols with a similar rest interval to see if similar result can be obtained.

REFERENCES

Allen, F.M. Stiliman E., Fitz R. (1991). *Total olietary regulation in the treatment of diabetes. Exercise,* New York, Rockfeller Institute of medical research.

Allen, R., Hollmann, Boutellier (1993). *Effect of aerobic and anaerobic training on plasma lipoprotein*. Int. sports med. 14396-400.

Allian C.C., Poon, S.G., Roeschlawl, Richmond W.F. *1974). *Enzymatic Determination of total cholesterol.* Clinical chemistry, 20 :470.

Alterations of *HDL Subclasses in endogenous hypertriglyceridemia* American Heart Journal 150: 1039-1045.

American Alliance of Health. Physical Education Recreation and Dance (1990). The AAPHWED youth manual Washington DC Government press.

American college of sports medicine (2006). Guidelines for exercise Testing and prescription 7th ed. Pheladelphia Lippincott, Williams and Wilkins.

American college of sports medicine (1995). Guidelines for exercise Testing and prescription, 5dth ed. Philadelphia, Febiger 151-176.

American college of sports medicine (1996). Proper and *improper weight loss programmes. Medicine in science and sports exercise 1*8(7): XIX".

American college of sports medicine, position stand (1998). Exercise and physical activity for older adults. Medicine in science sports and exercise, 30:992-1008.

Annuzi G.E., Jansson E, Kaisser L, Holmquist L, Carison L.A. (1987). Increased removal rate of exogenous triglycerides after prolonged exercise in man time course and- effects of exercise duration. Metabolism 36: 438-443.

Appel L.J., E.R., Miller A.J. Seidler and P.K, Wheton (1993), Does Supplementation of Diet with Fish Oil reduce blood pressure? A Meta analysis of controlled Conical Trials ARCH, mt. 153, 153-1438.

Assman G., Schult H. Von Eckardstain H., (1998). The Emergency of triglycerides as a significant Independent Risk factor in coronary artery disease Euheart T. 19 (Suppl M) M8-M4.

Athonia, O.O, (2010). Prevalance and gender distribution of the metabolic syndrome. *Diabetology and metabolic syndrome* 2:1-7.

Austin M.A., Breslow J.L Henekens C.H., Burning J.E., Willet W.C. Kraus R.M (1988) Low density lipoprotein subclass patterns and risk of myocardial in tarition. JAMA 260: 1917-1721.

American College of sports medicine. 1998, ACSM's Resource manual for guidelines for ax. Testing & prescription, Third Edition, Baltimone. Lippincott Williams & Wilkins.

Brownell II-D, P.S. Bachorik & R.S. Ayerle (1982) Chayes in Plasma lipid and lipoprotein levels in men and women after a programme of moderate ax", circulation 65(3): 477-84.

Balk, E.M., A.H. Liehtenstein M. Chung, B. Kupelmick, P. Chew and 3.1, (2006). Effect of Omega 3fattyacid on coronary restenosis, a systematic review. *Atherosclerosis*. 184:237-246.

Ballantyne F.C., Clerk R.S., Simpson H. S., Ballantyne D. (1982). High density and low density lipoprotein subfractions in survivors of myocardial infarction and control subjects. *Metabolism* 31:433-437.

Bar-or (1987). Pediatric sports medicine for the practitioner. *Physiological principles of clinical application*, New York.

Bell K.S., Schmit-peiterchim-Traser, M; Biden T.S., Cooney, G.J. and Kraege, E.W. (2000). Acute reversal of lipids induced muscle insulin resistance is associated with rapid alteration in PVC-thetalocalizaiton. Journal of *physiology, Endocrinology and metabolism.* 279: El 196-E1201.

Bereson, G; Wattihney W., Baow, Srinivasan S. Radhal Crishia marthy BL; (1995). Rationale to study the early natural history of heart disease; the *Bagolusa heart study*. Am J.Med. Sci 30:522.

Blair S.N; Kohi, H.W., Barlow C.E., Paftenbarger, R.S., Jr. Giblons L.W., Macera C.A., (1995). Changes in physical fitness and all cause mortality: A prospective study of healthy and unhealthy. JAMA 2731093-1098.

Blessing, DL, Keith, R.E. and Williford, H.N (1995). Blood lipid and physiological response to Endurance training in adolescents; pediatric *exercise sci*. 7:192-202.

Boden W.E, (2000). High density lipoprotein cholesterol as an independent risk factor in cardiovascular disease. Assessing the data from. Framingham to the veterans affairs high density lipoprotein intervention trials. *AMcardiol*. 86 (12A): 19 1-221.

Boston AG, cupples, LA, Jenner J.L (1996). Lipoprotein levels and risk for coronary heart disease in managed 55 years and younger, a prospective study. *JAMA* 276; 544-548.

Bouchard C, Despres J.P. (1991). Physical activity and health: Atherosclerotic metabolic and hypertensive disease. Exercise sports 4 66:1-8.

Boutcher, SH (2011). High intensity intermittent exercise and fat loss. Journal of obesity 2011: 868305.

Bouzan, C.J.T., Cohen, W.E., Connor. RM Kris' Etherton, GM Gray A. Konig, R.S Lawrence, D.A Saritz and SM Tentsch (2005). A quantitative analysis of fish consumption and stroke. *AMJ. Med*. 29. 347-352.

Boyden, T.W., Pamenter, R.W. and Going, S.B. (1993) Resistance exercise training is associated with decreases in serum low density lipoprotein cholesterol levels in premenopausal women. Achieves of internal medicine vol. 153 (1): 97-100.

Brairaktari E.T., Seferiadia K, and Elisaf M.S (2005). Evaluation of methods to the measurement of low density lipoprotein cholesterol. *J. Cardiol. Pharm*. 10 (1): 45-54.

Castelli WP, Abbot RD; M; Namava, PM (1983). Summary estimates of cholesterol used to predict coronary Heart disease. *Circulation* 67: 730-734.121

Castelli, WP, Garrison, RJ., Wilson P.W.F, Abbott, RD Kalousdian, .S and Kannel, W.B (1986). Indices of coronary heart disease and lipoprotein cholesterol levels: the *Framingham study*. JAJYJA 256: 2835-283 8.

Chado, M.A (1992), Physical Fitness for protective health, Zaria, ABU press Pp 22-36

Commell .E. Darios, F, Broersen K, Gatsby N. Peak-chew, S.Y Rickman C. Davletor (2007). Mechanism of arachidonic acid action on syntax in - Munc. 1 8EMBORep:4 14-92.

Conklin, Martha, et al (2005). College students use of point of selection Nutrition information' Topics in *clinical Nutr*., vol. 204107 pp97 (12)

Crouse SF, OlBrien B.C Graudjean PW,Lowe Rc Rohack 1.1 Green I.S (1997). Effect of training and a single session of exercise on lipids and apolipoprotein in Hypercholesterolaemic men *J App Physical* 83:2019-28.

Cutler J.A, Thom TJ Roccella E., (2006). leading causes of death in the United States *JAMA* 29538-384.

Crique, M, Heiss G., Chn R Cowan!., Suerin CM Bangei waiass kritchersky, Jacob B.O Grady H., Davis C.D (1993). Plasma triglycerides level and mortality from coronary heart disease *N. Engl. I Med* 328.

Chado, M.A. (2011). Physiological Basis of active lifestyle. Fighting obesity. The Natural way. 2nd ed Zaria Sttevawo press.

Darka, R.C Frost, R. Colins P Apple by and R. peto (1997) Dietary lipids and blood cholesterol quantitative meta analysis of metabolic studies BM J. 314.

Davis, PSW, Gregory J White A; (1995) Physical Activity and body fatness in preschool children inter obesity 19-6-10 Department of Human services (1996). Physical Activity and Health: A report of the surgeon General Centre for chronic disease preventing and Health promotion Washington, DC. Us Govt. Printing of file.

Depres I.P (1994). Dys lipidemia and obesity. Bailers clinical *Endocrinology and metabolism* 8: 629-660.

Depres JP, Moorgani S., Tupiann PJ., Tremblay A, Nadeau A., Bouchard C. (1990). i Regional distribution of body fat,' Plasma lipoprotein and cardiovascular disease-*Atherosclerosis*- 10:497-511.

Depres, JP Pouliot, ML Moorjani S. et al (1991). Loss of abdominal fat and metabolic responses to exercise in obese women *AMJ physical* 26. EL59-EL187

Despires J.P, Pouliot M.C. Moorjani S, Nadeau A. Tremblay A. Lupien P.J., Therianult G. Bourchard C. Loss of abdominal fat and metabolic response to exercise training in obese women. *Am S.physiol* 1991: 261. E 159-E167.

Divitis O. Faio, S., Petitto M Maddalena G., Contaldo F. Mancinc M. (1981) obesity and cardiac function *circulation* 64: 477-11.

Dowda M., Ainsworth B.E Addy C.L, Saunders R, Riner W. (2001) Environment influences, physical activity and - weight status in 8-16 years old. Arch Pediator *Adolsc Med* 155: 711-717.

Driller Mathew, Fell James, Gregory John, Shing Cecilia, Williams Andrew (2009) "The effects of high intensity interval Training in well trained vowers; *international journal of sports physiology & performance* 4:1.

Driskell 1., Wolinsky I, Editors, Energy yielding macro: nutrients and Energy *metabolism in sports nutrition,* Vol. 1 Boca Raton (FL) Press: 87-117.

Drygas W, K, T. Jegier, A and Kunski, H (2000) Long terms Effect of different physical activity levels on coronary disease factor in middle- aged men. *Journalof Sport medicine,* 21: 23 5-241.

Duncan J.J., N.F. Gordon, & C.B. Scott (1991) "Women walking for health and fitness. How much is enough?" *Journal of America; Medical Association.* 266(23): 329-9.

Durstin J.C. Crouse, S.F., & Moffort, R. (2000) Lipids in Aercise & sport Energy yielding macronutrients & energy *metabolism in sports nutrition* 1:87-117.

Durstin, J.L., Grandjean, P.W, Lox, A; & Thompson, P.S (2002). Lipids, lipoprotein, *Exercise Journal of Cardio pulmonary Rehabilitation* 22: 2385-398.

Durstine J.L. & W.L Haskell (1994) “affects of exercise training on plasma lipids and lipoprotein exercise and sports science reviews. 22:477-522.

Durstine JL., Haskell W (1994) Effect of Exercise training on plasma lipids and lipoproteins. In: Holloszy 1. aditor exercise and sports science reviews. Philadepeiphia (PA): Williams and Wilkins 273 :402- 7

Durrington P. (2003) Dylipidaemia: Lancet 362 (9385): 717-. 31, doi 10,1016,80140.
Durstine 1 .L, Crouse, SF, Moffant R (2000) Lipids in exercise and sports

Durstine JL, Grand Jean PW Daris PG Ferguson M.A Alderson NL DuBose KD (2001). Blood lipid and lipoprotein adaptation to exercise a quantitative analysis *sports med*: 3 11033-1062.

Sarika, C, Manpreet, K. K., Jaspal, S. (2010). Effects of aerobic exercise on lipids profiles and high molecular weight adiponectin in Japanese workers.

Eimann D.C. (1988). The exercise health connection Champaign IL. *Human Kinetics.*

Eisenmann 1. C., (2003) Secular trends in variables associated with metabolic syndrome; of North American children and adolescent. *AMJ Hnm. Biol* 15:786-794.

Executive Summary of the Third Report of the Natinal cholesterol Educ. Progress (NCEP) (2001) expert panel on Detection, Evaluation & Treatment of High Blood cholesterol in Adults, (Adult Treatment panel III) *The American Medical Association* 285: 2486-2497.

Expert Panel (1993) Summary of the second Report of the National cholesterol Educ. Prog. (NCEP) Expert panel on Detective, Evaluation & Treatment of High Blood chol in Adults. *The America medical Association* 269: 3015-3023.

Executive summary of the third report of the national cholesterol education program (NCEP) (2001) Experts panel on Detection, Evaluation and Treatment of high blood cholesterol in (Adults Treatment panel 11) *JAMA*, 285: 2486-2493

Farhan M and Micheal (2008) Low density lipoprotein cholesterol: an important consideration in coronary heart disease risk assessment: current opinion in Endocrinology, *diabetes and obesity* 15: 175-181.

Fletcher G. Blair, S. Blumenthal C. et al 91992) Benefits and recommendations for physical activity program for all Americans: a statement for health professional by the committee on Exercise and Cardiac Rehabilitation of the council on clinical cardiology. American heart Association. *circulation* 86: 340-4.

Folsorne, A et al (1993). Body fat distribution and 5 year risk of death in order women Journal of the American medical Association, 269- in Williams, M.H (1991) Nutrition for Health fitness and sports fifth Edition WCB/MC Graw-Hill.

Ford E.S (2005). Risk for all-cause mortality; cardiovascular disease and diabetes association with metabolism syndrome: a summary of the evidence, diabetes cave, 28: 1769-1778.

Ford E.S, Giles Wil, Diet WH (2002) prevalence of the metabolic syndrome among U.S adults: Finding from the third National health and National Examination survey. *JAMA287*-356-359

Franceschin G. (2001) Epidemiologic evidence for high density lipoprotein cholesterol as a risk factor for coronary artery disease. A1VI J. Cardiol 88: (12A) 9M-13M Grammer, K and Robert K (2006) Harpers illustrated (8) Biochemistry 2ih Tata McGraw Hill's publication 3.

Frankel S., Elwood, F and Sweetnam P (1996) Birth weight, adult risk factors and incidence of coronary heart disease. New Eng. *Journal of medicine* 327: 13350-13355.

Friedwald WT, Fredrickson, D.S (1974) "Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge clinical chemistry 18:499-5 02.

Garrison R.J, Wilson PW, Castelli WP, Feinleib M., Kannel WB,MC Namara Pm (1980). Obesity and lipoprotein cholesterol in' the Framingham offspring study. Metabolism .29:1053-1060 'cross Ref medicine.

Goldbourt U, Yaari S, Medalie SF1, Isolated low HDL-C as risk factor for coronary heart disease mortality: a 21 year follow up of 800 men. Arterioscier Thromb vase. Biol. 1997: 17: 107-113.

Goran, M.I. Naggay, T.R. & Gower, B.A. (1999) Viscaval fat, insulin sensitivity and lipids in pre-bubertal children. *Diabetes*, 48: 1515-1521.

Gaiziano J.M, Hennekens, C.H, 0' Donnell, C.J and Buring, J,E (1997). Fasting triglycerides high density lipoprotein and risk of myocardial infarction: *circulation* 96: 2520-2525.

Giada, F. Vigna,LB, Vitale, E Ct a! (1995). Effect of age on the response of blood lipids, body composition arid aerobic power to physical conditioning and deconditioning *metabolism* 44: 161.

Golbourt, I Yaari S., and Medalic, JH (1997) isolated high density lipoprotein cholesterol as risk factor for coronary heart disease mortality. 21 year follow up of 8000men. *Art, thrombosis and vascular Biology*, 17:107- 113

Gotsia, Fg stare and J Worcester (1959) Interaction between the kind and amount of obesity fat and dietary cholesterol in Experimental hypercholesterolemia. *Am Jclin Nutr* 7: 5-12.

Griffin, B.A, Freeman DJ, Tait GW, Thonspson J., Caslate M.J Packard C.J Shepherd J. (1994). Role of plasma triglyceride in the regulation of plasma low density lipoprotein subs fractions: Relative contribution of small dense low density lipoprotein to coronary heart disease risk *Atherosclerosis*: 106: 241-253.

Guo, LT, FU, M.D, XU, YN, Tian, L Yan, B.Y and Yang L.C (2005). Guo, W, Kawano, H. Pino, L Itoh, N, Node, K and Sato, T. (2011).

Guttin B, and Owens J. (1999). Role of Exercise intervention in improving body fat distribution and risk profile in children. American Journal of Human Biol. 12: 127-247.

Hardman AE. Pa, obesity & blood lipids. mt. J Obes. 1999: 23 (Supply 3):964-571.

Haruna, F .R (1994)3. The role of exercise program in the treatment of obesity: The*Kinesiologist* Vol NO 1 PP 26-32

Heyward V.H. (1998) Advance fitness Assessment and Exercise prescription. Champaign IL. *Human Kinetics.*

Howley, E.T. (2001). Type of Activity: Resistance, Aerobic and Leisive versus occupational Phy. Act. *Medicine in science, sports and ax.*33(6): 5364-5369.

Hill, 10, Pagliassitti Mj Peters J.e (1994). Non genetic determined of obesity and fat topography. In Bouchard (Ed) Genetic determinants of obesity. Bola Rato CRC Press 35-48.

Haditta, Suwaidij A (2007). Vascular Health risk management. 3(6): 853-76

Halbert 1A Silagy C.A Finucane, P Withers PTHandorf PA (1999). Exercise training and blood lipids in hyperdemic adults: a meta analysis of randomized, controlled trials *Eur.J Clin Nur.* 53514-522.

Hanson,R.L Narayan, K.M Mc (Canoe, D.R et al (1995) Rates of weight gain, weight fluctuation and incidence of MIDDM. Diabetes 44: 261-266 pub med/chemport

Hardman AE, (1999). Interaction of physical activity and diet implications for lipoprotein metabolism Public Health Nutr 2: 369- 376

Havel, RJ, Kane JP, (1982). Therapy of hyperlipidemic states. *Ann Rev. Med*. 33: 417.

Hegarty, B.D, Furter, SM, Ye, J, Cooney, G.S and Kreagen, E.W (2003). The role of intramuscular lipid in insulin, resistance: Acta physiol. of *Scaridinaria* 178:373-383.

Hersey WC, Graves J .E, Pollock Ml (1994) Endurance exercise training improves body composition and plasma insulin responses in 70-79 years old men and women, *metabolism* 43:841-854.

Hinkleman LL Nieman DC (1993). The effect of walking programme on body composition and serum lipids and lipoproteins in overwight women T. *Splorts Med phys. fitness* 3349-58.

Huff, M.W, and D.E. Tefford (1989). Dietary Fish oil increases conversion of very low density lipoprotein apoproteins B to low density lipoprotein **Art**. 9: 58-66.

Hunter et al (1994) intra-abdominal adipose tissue values associated with risk of processing elevated blood lipids and blood pressure, *obese Res*. 2:563-8.

Iawley, V.H, (1991). Advanced fitness assessment and Exercise prescription champolign iL. Human IC Netic Books. Imamoglu G. Atan T., Kishali N.F Barmaoglu G. Akypip yildrinin K. (2005).Comparison of lipid and lipoprotein values in men and women differing in training status *Biol Sport* 22: 261-270

Jatau, R.S, Verikateswarlu,K, Dikki CE and Haruna, F.R (2012).

Jia, LQ, Lon g, S.Y, Fu, Md, Yan, B.Y. Tian Y, XY Y.H, Guo, L.T, (2006) relationship between total cholesterol/High density lipoprotein cholesterol ration, triglycerides/High density lipoprotein subclasses *metabolism* 55:1148.

Journal of Apidemiology. 136 (7): 806-18 seip R.L et al. 1993. Ax. Training decreases plasma cholesterol. Ester transfer protein. Athrosclerosis" 13(9): 1359-67.

King A.C. et al, (1995) Varying intensities and formats of PA on participants rates, fitness and lipoproteins in men, and women aged 5 0-65 years" *American Heart Journal* 9 (10) 2596-04.

Kadoma, S. Tanaka, S., Salto,K Shu M., Sone, Y, Ornitake, F Suzuki, E. Shimano, I-I. Yamamoto, S. Kondoo, K Ohashi Y., Yariada, N., and Sone H, (2007) Effects of aerobic Exercise Training on Cholesterol. *Arch. Intr. Medicine* 167 (10) 999-1008.

Kelley, G.A. Kelley, K.S. (2006) Aerobic exercise and lipids and lipoproteins in men: a meta anal2ysis of randomized control trials *J Men's Health Gend3:61-70.*

Kelley, G.A. Kelley, K.S. (2009) impact of progressive resistance training on Lipids and lipoproteins in women: a metal analysis of randomized controlled trials. *J. Women Health* 13: 1148-1164.

Kelley, G.A., Kelley, K.S. and VU Tran, Z., (2005) The effect of aerobic exercise on lipid and lipoprotein in overweight and obese adults *International journal of Obesity* (Lond:,) 29 (8): 881:8931.

Kelley, G.S. Kelley, K.S. and VU Tran, ZU (2004) Walking, Lipids and lipoproteins: a meta analysis of randomized controlled trials preventive *medicine*, 38:S438-445.

Kennel WB, Neaton JD, Wentworth D Thomas H.E. Stamler J. Hulley SB Kjelsherg M.O (1986) overall factors in 325, 348 men screened for the MRFIT Multiple risk factor intervention.

Keys, A.O Mickelseen, E.O Miller and CB Chapman (1950). The relations in man between cholesterol level in the diet and in the *blood Science* 121: 79-81.

Kiens, B. Lithel H., (1989) lipoprotein metabolism influenced by training induced changes in human skeletal muscles T *clin invest.*

Kikkinos P.F. and B. Fernhall, (1999) physical Activity and high density lipoprotein cholesterol levels. *Sports medicine* 28(5): 307-14.

Kikkinos P.F. et al (1995a) miles run per week and High density lipoprotein cholesterol levels in healthy middle aged men. A dose response relationship. *Archieves of internal medicine* 155 (4): 4 15-20.

Kikkinos P.F. et al (1995b). cardio respiratory fitness and coronary heart disease risk factor association in women. *Journal of American college of cardiology*. 26 (2): 3 58-64.

Knowler, WC, Saad M.F, Pettit D.J, Nelson RG Bennett PH (1993) Determination of diabetes mellitus in the Pima *Indians. DiabetesCave* 16:216-227.

Kokkinos P. Holland J. Narayan P. et al (19995) miles run per week and high density lipoprotein cholesterol levels in healthy middle aged men a close response relationship. *Arch Intern Med* 155:415-20.

Kris-Etherton, PM, W.S Harris and L.J. Aple Nutrition committee (2002) Fish consumption, Fish Oil, Omega 3 fatty acids and cardiovascular dlisease. *circulation*. 106:2747-2757.

Kunnel WE (1985) Lipids, diabetes and coronary heart disease: Insight form the Framingham study *AMHeartJ*. 100: 11 00-1109

Lakka T.A Salonen J (1992) Physical activity and serum lipids, cross sectional population study in men. AMJ. *Epidem.* 36:806-18

Laursen, P.B. Jenkins DG (2002) The Scientific Basis for high intensity interval Treg; optimizing tray profs. A maximizing performance in Highly trained Endurance Athletes. *sports medicine* 32 (1): 53-73.

Lakka, T.A an Laaksone, D.E (2007). Physical activity in prevention and treatment of the metabolic syndrome. *Applied physical in Nutrition and metabolism* 32(1)76-88.

Lammon-Fava S., Wilson, Schaefer EJ (1996) impact of the body mass index on coronary heart disease risk factors in men and women.Arteriosclerosis: *Thromb Vasc.Biol* 16: 1509-1515.

Larson Meyer B. Newcomer BR Heibom, Lk Volaufora 1. Smith SR, Altonson A.J, Lefewe M, Road J.C Williamson DA, Ravussin E. (2008). Effects of 6 month Caloric Restriction and Exercise on serum and liver lipids and markers of liver function obesity, 16:1355-1362.

Lean, M.E. J., Han, T.S, Morrison, CE (1995) waist circumference as a measure for indicating need for weight management. BMj. 311: 158-161.

Leckre .S. Alland c., Talbot J. et al (1985) High density lipoprotein cholesterol, habitual physical activity and physical fitness. *Atherosclerosis* 57: 43-5.

Leon A.S, Connett, J. Jacobs DR Raurama R (1987) Leisure time physical activity level and risk of coronary heart disease and death. The multiple risk factor intervention trial. *J. med Association* 258:2388-2395

Leon, A.S., and Sanche, O.A (2001). response of blood lipids to exercise training alone or combined with dietary intervention: medicine and *Science in sports and exercise* 33 (6Suppl): S502.S5 15.

Levine, D.M Soan, B.I Dunner IE, Lorenz J.D Heinzerting RH (1992) Automated measurement of lipoprotein by immuno turbidmetric analysis Inst. *J. Clin Lab RES 22: 173-8.*

Levy R.J (1981) Cholesterol lipoprotein, Apoprotein and Heart disease: present status and future prospects. *Clin Chern* 27: 653.

Lindhein S.R. et al (1994) “The independent effects of exercise and estrogen on lipids and lipoproteins in postmenopansal women”. *Obstetrics and gynecology*. 83(2): 167-72.

Lippi G. Schena F., Salvagno GL. Montagnana M. Balliestrier F., Guidi G.C (2006) Comparison of the lipid profile and lipoprotein between sedentary and highly trained subjects.*Clin chemistry*. Lab md. 44:322-326.

Lope-virella, M.F Stone P., Ellis S. and Coiwell (1977) cholesterol determination in HDL-C separated by three different methods *Clin chemistry* 23 :882-884.

Manson, J.E. Tosterson, H, Ridker, P.M, Satterfield, S., Hebert, P.T., O.,Buring, J.E, & Henekens, C.H. (1992) the primary prevention of myocardial infaratin. *The newEngland Journal of medicine,* 326, 1406-1416.

Martin, R.P, Haskell, W.L. & Wood, P.D. (1977) Blood chemistry ad lipid profiles of elite distance runners: *Anals of NYAcademic of Science* 301, 346-360.

Martin, R.P, Haskell, W.L. & Wood, P.D. (1977) Blood chemistry ad lipid profiles of elite distance runners: *Anals of NYAcademic of Science* 301, 346-360.

Mahley R. W, (1982) Antherogenic hyperlipoproteinemia. *med.clin* North. Am 66:375.

Mahley, R W (1982) Antherogenic —hyperlipoproteinemia. *Med clin*, North AM 66: 375.

Mamgat J. Elosua, Covas, M.I, Molina L. Rubies Prat (1996) Amount and intensity of physical activity, physical fitness and serum lipids in men *AMJ. Epidem*. 143 ;562-569.

Martin Elizabeth A. (2007) concise medical Dictionary (Ed) Oxford England:
Oxford University Press ISBN 0192806971.

Mary Lynn Hendrix Encarta (2009)

Mathew J. Watt et al (2002) Intra Muscular triacyglycerol, glucogen and acetyl group metabolism during 4h of moderate exercise in men. *J Physiology* 541 (P3): 969-78 PMC 2290362 PMC 1268055.

Maud, P.1 and Foster, C, (1995) Physiological assessment of human fitness, *Human kinetics.*

Mclaren L., and L., Gaurin (2002). Cumulative impact of being over weight and women's body Esteem. A preliminary study" Eating and weighing disorders Vol N04 pp 324.

Medina Urrutia A.; Juarez-Rojas JG Martinez- Alvarado R. Jorge Galaza E., Posadas-Sanchez, R., Cardoso- Saldana G. caracas Podilla N., Mendoza-Perez E. Posdas Romero (2008) High density lipoprotein subclasses distribution and composition in Mexican adolescents with low l-IDL cholesterol and high triglycerides concentration and its association with insulin and creation protein Art 201: 392-397.

Mensick, R.P, PL., Zock., A.D, Kester and M.B, Katan (2003).

Miller G) ,Miller NE (1977) Plasma high density lipoprotein concentration and development of Ischemic heart disease. Lancet 1: 16-18.

Miller, JP, Prathey RE, Golbery et al (1994)., Strength training increase insulin action in Health 50-65years oldmen. *Journal A pp1. Physiol*. 77:7122-7

Morgan,1. Cavey C,Lincoff, A, andCapuzzi, D (2004). High density lipoprotein subfraction and risk of coronary artery disease current *Atherosclerosis* 6:359-365.

Muhammad, M.S., (2003). Effect of aerobic exercise training on selected physiological variables in non-insulin dependent diabetes. (Unpublished) Ph.D Dissertation, Ahmadu Bello University.

Neiman D.C. 1998 "The exercise Health connection. Champaign, IL: *Human Kinetics.*

Nikkila F., Kuusi, T, & Myllunen, P.(1980) High density lipoprotein and apolipoportein A-i during physical inactivity, *Atherosclerosis*, 37, 457-462.

Nair, S.S., J.W Leitch,J. Falconer, and M.L. Garg (1997). Prevention, of cardiac arrrythmia by dietary (n-3) Poly unsaturated fatty acid and their mechanisms of action. *J. Nutr*. 127: 383-393.

National cholesterol Education- program (1993) Second Report of (the Expert Pannel on Detection, Evaluation and Treatment of high blood cholesterol in adults publication No 93-3095 JAma 269: 305-3022.

National Institute of health concensus Development panel on the health implication of obesity (1985). Health implication of obesity. *Ann. Intern. Med*. 103: 1 073-1 077.

Nestel, P.J, W.E. Conor,M.F, Reardon, S. Connors. Wong and R., Boston 91984), suppression by diet in Fish oil of very low, density lipoprotein production in man. 1. *Clin Invest* 74:82-89.

NIH Concensus Development Pannel on triglycerides, High density lipoprotein and coronary Heart disease (1993). NIH concensus conference on Triglycerides, High Density lipprotein and coronary heart Disease. *JAMA*, 269: 505-510.

O'Connell BJ and Genest J. (2001) High density lipoprotein and Endothelial Function: *Circulation*: 104: 1978-83.

Ononogbo, I.C (1979) Serum cholesterol levels In Nigeria population sample (Nsuka) Experiments 35:1428-1429.

Pate R. Pratt M. Blair (1995). Physical activity and public health *JAMA* 273: 402-7.

Pekkanen 1. Linn S. Keiss G et al (1990), Ten year mortality from cardiovascular disease in relation to cholesterol level among men with and without pre existing cardiovascular disease *N. Engl. J med* 322:1700-

Perez, B., Landaeta-Jumenez and Vosquez,M. (2000). Fat distribution in venezualan children and adolescents estimated by conicity index and hip ration. *American Journal of Human Biology* 14: 15-20.

Plowman, S.A and smith DL (1997) Exercise physiology for health fitness performance. *Alliyn and Bacon Boston.*

Powell K.E., Thompson P.D, Caspersen C.J. Kendrick 1.K (1987) Physical activity and the incidence of coronary heart disease. *Ann Rev Public Health* 8: 253-287

Praveen Ganesan (2009). Importance of Regular Exercise for perfect health *wvvw with diet blogspot.com.*

Prong NP (1993). Short term effect of exercise on plasma lipids and lipoprotein in humans sports med 16:431-448.

Perry, A.C. Rosenblatt, E.S. Kempner, L. Feldman, B.B. Paolercio, M.A. & Van Benden, A.L, (2002). The effects an ex. Physiology programme on physical fitness variables, body satisfaction, and physiology knowledge, *journal of strength and conditioning Research,* 16, 219-226.

Perry, Christopher G.R., Heigenbauser, George J.F; Bonen, Arend; spriet Lawrence L. (2008) high Intensity aerobic interval tray increases fat & CH metabolic capacities in human skeletal muscle." *applied physio, Nutrition a metabolism* 33(6): 112-11123.

Reaven, G.M (1988) Bauting lecture," Role of insulin resistance in human disease. *Diabetes* 37:1595-1607.

Rexrode, K.M,manson JE, Hennekens, CH (1996). Obesity and CYD CUD'. *Opin Cardio.Il* :490-495.

Ricker PM, Rifai M. Look NR, Poradwin G. Burning JE (2005). Non-HDL Cholesterol apolipoprotein A-I and B 100 standard lipid measures, lipid ratios and CRP as risk factors for cardiovascular disease in women.*JAMA* 94. 326-333.

Rificind BM (1990) High density lipoprotein cholesterol and coronary artery disease survey for the evidence Am 1. Cardio. 66: (6), 3A-6A Rippe 1M Ward A Porcari TP Fredson PS (1988) Walking for health and fitness-*JAMA.* 2592720-2724

Rohack J.J Green J.S (1997). Effect of training and a single session of Exercise on lipids and apolipoprotein in Hypercholesterolaemic men 1. *App Physiol* 83: 20 19-28.

Rye KA, Clay M.A, Barter P1(1999) overview of plasma lipid transport. In plasma lipids and their role in disease. Edited by Barter P.1. Rye, K.A Advances in vascular Biology. *Harwood Academic Pub*. Imprint.

Sarika C. Manpreet, K.K., and Jaspal, S.S (2010). Aerobic Exercise, Resistance Training Obesity, heart rate and physical fitness. Sports *medicine research centre* 1-7.

Scan, A.M. (1978) Plasma lipoproteins and coronary heart disease annals of clinical & Manson, J.E. Tosterson, H, Ridker, P.M, Satterfield, S., Hebert, P.T., 0., Buring, J.E, & Henekens, C.H. (1992) the primary prevention of myocardial infaratin. –*The new England Journal of medicine,* 326, 1406-1416.

Scan, A.M. (1978) Plasma lipoproteins and coronary heart disease annals of *Clinical&Laboratory Science*, 8. 79-88.

Schartz, R.S 91988). Effect Exercise training on high density lipoprotein and lipoprotein and in old and young men. *metabolism* 37- 1128

Seidell J.C- Verschuren. Van Leer, E.M Kromhout D. (1996) Overweight and mortality a prospective study of 48287 men and women. *Arch Int Med.* 156: 958-963.

Seidell, J.C, Cigolim M. Deshypere JP et al.(1991). Body fat distribution in relation to serum lipoprotein and blood pressure in 38 years old European men. The *European fat distribution study*. Art 86:251-260.

Seidell, J.c., Perusse, L., Depres, J. Bouchard, C. (2001) waist and hip circumferences have independent and opposite effects on Cardiovascular disease risk factors. *The Quebec family Study*. The 74: 315-321

Sharon, A., Plowman, Denise L. Smith (202007) Exercise physiology for health, fitness and performance. *Lippincott Williams* P.61 ISB 978-6-7817-8406-1

Shemmel, R.A (1990) Nutrition physiology and obesity. *CRe Press inc* Bota Ration Florida Silverman, DL, Geofrey SG Pastennak (1993). High density lipoprotein sub fractions. *AM Med*. 94:64:636-645.

Sniderman AD, Furbery C.D Keech A, Rocters van Lennep J.E et al (2003) Apolipoprotein versus lipids as indices of coronary risk and as targets for Statin treatment *Lancet.* 361: 777-80

Stample. r MJ. Sacks, FM. Salvanis Witlet W.C, Hennekare C.H (1991) A prospective study of cholesterol, apoprotein and the risk of myocardial infarction. *NEng*i. *J.* 325-373-381.

Stein R. Micheilli D. Glantz M. et al (1990) Effects different exercise training intensities on lipoprotein cholesterol fraction in healthy middle-aged men. *AMHealth* 1. 119: 277-83.

Stunkard AI.J Wadden T.A (1993). Theory and Therapy 2 Ed. N. Y *Raven Press*.

Superko. H, (1991) Exercise training, Serum lipids and lipoprotein particles. Is there a change threshold? *Med. Sci. Sports exercise*. 231:677- 284.

Tall, A. (2002). Exercise to reduce cardiovascular risk how much is *enough*.N ENGL 1. Med 347: 1522-25.

Taylor, Eric Etal(2005) obesity, weight gain and the risk of kidney stones: *journal of American medical association* vol. 293: No 4 P 459.

Talanian, Jason L; Stuart D. R Galloway, George, J.F. Heigenhauser, Arend Bonen, Lawrence L. Spriet (2007) two weeks of high intensity aerobic interval to increases the capacity for fat oxidation ex. In women *journal of Applied physiol*. 102 (4): 1439-1447.

Taylor, D.A. & ward; A., (1993). Women, high density lipoprotein Cholesterol and exercise. *Archives of internal medicine*, 153, 1178- 1184.

Tremblay A, Simoneau J.A, Bouchard C. (1994) impact of exercise intensity of body fatness and skeletal muscle metabolism." *clinical & Experimental* 43(7): 8 14-8.

Terry, R.B, Wood, P.D, Haskell, W.L Stefanick, M.LKrause R.M (1980) Regional Adiposity Pattern in relation to lipids, lipoprotein cholesterol subfractions Mass in *Men .J. Clin Endocrinol. Metabolism 68:191-199.*

The Trials of Hypertension prevention collaborative Research Group (1997). Effect of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high normal blood pressure: The trial of hypertension prevention: Phase II. *Arch Int. Med.* 157:657-667.

Thomas, T. Adeniran, S. and Etheridge, G. (2004). Effect of Omega- 3 fatty acids supplementation and exercise on low density lipoprotein and high density lipoprotein subtraction. Metabolism, S3:749-754

Thompson P.D Cullinane EM, Sady SP et al (1988) modest changes in high density lipoprotein concentration and metabolism with prolonged exercise training, *circulation* 78: 25

Thompson P.D, Buchner B, Pinia IL et al (2003) Exercise and physical activity in the prevention and treatment of Atherosclerotic cardiovascular disease. A statement from the council on clinical cardiology (Subcommittee) and the council on *Nutrition.*

Thompson, P Cullinane, E and Sady, S (2001). The acute versus the chronic response to exercise. Medicine and Science in *Sports and exercise*33(6): S438-S445.

Tian, J. and FU, M., (2010). The relationship between high density' lipoprotein ssubclasses profile and plasma lipids concentration> Lipids in Health and Disease49(l 18): 1-9

Tietz, N.W(1987. Fundamentals of clinical chemistry 3rd 'Ed W.B *Saunders Company*, US.A.

Toifrey, K. Jones, A.M., and Campbell, 1G. (2000). The effects of aerobic exercise training in the lipid lipoprotein profile of children and adolescents. Sports *Medicine* 29(2):99-l 12.

Tomiyasu K. Ishikawa T. Ikewaki K et al (1996). Effects of exercise on plasma lipases and cholesterol ester transfer protein activities in normallipidemic male subjects. *Nutr, metabolic cardiovascular disease.*

Tremblay, A, J.P, Leblance C; et al (1990) Effects of intensity of physical activity on body fatness and fat distribution. *AM J. Clinical Nutr* 5 1: 153- 157.

Tsinikas. S. Brilakis, Miller ER,MC Connell J.P et al (2005) Oxidised phosphod lipids lipoprotein and coronary artery disease M. *Engl .J. Med*. 353: 46-51.

United States Department of Health and Human Services (1996). Physical activity and Health: a report of the surgeon General. Atlanta, G.A United states Department of health and human Services, cetre for disease control and prevention and National central for chronic disease prevention, Health and promotion.

Valdex, R, Seidell, J.C Aim, Y.I and Weiss K(1992) A newindex of abdominal adiposity as an indicator of risk for cardiovascular diseases. Across population study. *Internal journal of obstetrics* 16: 77-82.

Venkateswardy K. (1982) Training methods. A paper presented at the first kanon state sport council seminar August 12 Kano.

Venkateswarly, k.. (1 992)variation of body mass index body fat percent lipid profiles and trunks and limb skinfold ratio with age: Abstract of the *Olympic scientific congress Baccellona Spain Bose.*

Venkateswarly, K. (1 992)relationship between habitual physical activity level and diseases risk factors and cardio respiratory endurance. Abstracts of Olympic scientific congress baccellona Spain Bose.

Von Eckard stein A, Huang y. Assmann (1994) physiology role and clinical relevance of high density lipoprotein subclasses lurr-opin lipidol 5: 404-416.

Vakatswalu K. (1985) Training methods, Zaria unpublished monograph.

Venkateswarlu, K. (1992) Effects of Different methods of training on biochemical makers-*Journal of Nigerian Association of Sports medicine* 10: 25-34.

Watson, A.W.S (1985). Physical fitness and Athletic performance; A guide for stds, Athletes and coaches. *The Longman Group England.*

Wada, T. Ikeda, Y. (1998). Longitudinal studies to determine the effect of body fat rate reduction on blood pressure 1. *Med. Sys*t 22: 19-25.

Wald N.J. Law M. Watt TIC, WU, et al (1994) Apoliprotein arid Ischemic Heart Disease. Implications for Screening. *Lancet*. 343: 75-9.

Walto!), C Lees B. Crook D. Worthington, M. Godsiand, 1.F. Stevenson, J.C (1995). Body fat distribution rather than over all adiposity ‘influences serum lipids and lipoprotein in healthy men independently of age. *AM 1. Med* 99: 459-465.

Wamick, GR, Ivguyen T. Albers A.A. (1985) comparison of improve precipitation methods for qualification of high density lipoprotein cholesterol. *Clin Chem.* 31:217-222

Whelan I. (2008) (n.6) and (n-3) Poly unsaturated fatty acids and the aging brain. Food for thought. *Abstract.1. Nutr*. 138: 252 1-2522.

WHO (1999) WHO definite, diagnosis & classification of diabetes mellitus & its complication: part Geneva. WHO Department of non-communicable diseases surveillance,

William D. MC Ardle, Frank .1. Katch, Victor L. Katch (2006). Essentials of Exercise physiology. Lippincolt Williams and Wilkins P 204 1SBN978- 0-7817-4991 G.

Williams .P. (1998) Relationship of Heart Disease risk factors to exercise quality and intensity. *Arch Intern. Med* 158: 237-45

Williams P.T. Krause, RM, Wood P.D et al (1996) lipoprotein subfractions of runner and sedentary men-metabolism 35045.

Williams P.T. Mouse D.K (1995) Women runners showed improvement in plasma high density lipoprotein and adiposity at higher exercise levels than currently recommended. *Circulation*: 92

Williams RT Stefanick, ML, Vramizan KM, Wood P D (1994) Effect of weight loss by exercise or by dieting on plasma high density lipoprotein CHDL) levels in men with low, intermediate and normal - to- high HDL-at baseline-metabolism. 43:917.

Williams P.T (1998) “Relationships bf heart Disease risk factors for exercise quantity.

Williams P.T. (1996). “High-density lipoproteins cholesterol and other risk factors for coronary heart disease in female runneis” 334(20) 1298-1298

Wilmore JH. Stanforth P.R. Domenick M.A. Gagnon J. Daw EW, Leon As
Rao D.C. Skinner JS, Bouchard C. Reproducibility of anthropometric & body composition measurement the Heritage family study. mt. Jobes 1997 21:297-303.

Wilmore JR Stanforth P.R. Mandel 5, Rice T. Gagnon J. Leon As Rao D. Skinner JS Bourchards C. Alterations in body weight and composition consequent to 20 role of endurance training: The Heritage family study. *Am J. Chin Nufr*. 1999: 70: 346-32.

Wood P.O. & Haskell, W.L. (1979). The effect of exercise on plasma high density lipoprotein. Lipids, 14, 417-427.

Wood P. Haskell W. Stem Metal (1977). Plasma lipoprotein Distribution in Male and Female runners. *Ann NYAcad*. Sci 301: 748-63.

Wood P.D Stefanick, ML, Williams P.T Haskell W. L. (1991). The effect on plasma lipoprotein of a prudent weight reducing diet, with or without exercise in overweight and women .*N. Engl. 1. Med* 325 :461.

Wood, P.D Haskell, "V. Klein et al (1976) The distribution of plasma lipoproteins in middle. Aged male runner. *metabolism* 25: 1249

World Health organization (2005) Global Data on mass index. World-wide definition: A consensus statement from the Intemational Diabetes federation. *Diabetes* 23: 469-4 80.

World Health Organization (2008b). waist circ. And waist Hip Ratio. Report of a WHO Expert consultation, Geneva, 8-12 December, Pp 5-20.

WUXW, MD, LID, BW (1999). Study on immune detection. Method of HDL Subclasses in Human Serum. Clin J. Art 7: 253-255.

DEPARTMENT OF PHYSICAL AND HEALTH EDUCATION
AHMADU BELLO UNIVERSITY, ZARIA NIGERIA
(OFFICE OF THE HEAD OF DEPARTMENT)
PROFESSOR ABDULLAHI MUSTAPHA,
PROFESSOR C. I. DIKKI,

Date: 15th April, 2013

The Head,
Department of Chemical Pathology,
University of Jos Teaching Hospital,
University of Jos,
Jos.

Dear Sir,

REQUEST TO HELP IN CONDUCTING TEST FOR SERUM LIPIDS AND LIPOPROTEINS ON MY STUDENTS

Mr. Daniel, Philip Lekwot is conducting Research in partial fulfillment on his requirement of his Ph.D programme on the Effect of Aerobic Training on Serum Lipids and Lipoproteins of Nigerian College Students. In this regards, he needs to estimate Lipids and Lipoproteins of the subjects he would study. In view of this, I am writing this letter to request you to kindly help him in getting the test conducted in your well established laboratory.

Your assistance in this regard will be highly appreciated and acknowledged.

Yours faithfully,

Prof. K.Venkateswarlu
(Major Supervisor)

APPENDICS B

Department of Physical and Health Education
Ahmadu Bello University Zaria

Dear Parent/Guardian/Participant,

INFORMED CONSENT FOR DATA COLLECTION AND PERMISSION TO RELEASE WARD TO PARTICIPATE IN RESEARCH DATA COLLECTION.

Your ward ----------has been selected to participate in research data collection tiled " Effect of Aerobic training on Serum lipids, lipoprotein and body fat pattern of Nigeria college male students.

The Procedure

Anthropometric data shall be determined on each participant and indicated on the data collection form. Blood samples also shall be drawn from each participant for lipid analysis as indicated on attached form N0 1-36

Values of the Exercise training

This is purely an independent research work, and it is designed only to contribute to a body of knowledge, therefore all information collected shall be treated confidentially for the purpose of this research work only.

Attestation

I have read the above statement and I have agree/ not agree to participate in the exercise training with due consultation with my parents/guardian that:

(a) I have no history of any contraindication

(b) I can withdrawn without penalty from the exercise at will.

(1) Name of participant ---------- Signature ----------

(2) Name of Parent/Guardian---------- Signature ----------

(3) Name of Researcher ---------- Signature ----------

Adopted from Adeyanju (2000)

APENDIX C

CONTROL GROUP RAW VALUES OF PRE AND POST TRAINING BLOOD SAMPLES IN SUBJECTS FOR INTERNAL TRAINING

PRE TRAINING VALUES OF BLOOD													POST TRAINING VALUES OF BLOOD										
S/N	AGE	WEIGHT KG	HEIGHT (M)	WAIST CIRCUM	HIP CIRCUM	WHR	TC	TG	HDL-C	LDL-C	BMI	I/INDEX	AGE	WEIGHT KG	HEIGHT (M)	WAIST CIRCUM	HIP CIRCUM	WHR	TC	TG	HDL-C	LDL-C	BMI	I/INDEX
1	25	63	1.68	32	36	.08	4.0	1.2	1.2	2.3	22	22	25	64	1.68	32	36	.08	4.0	1.5	1.1	2.0	22	22
2	23	68	1.64	30	34	.08	4.3	0.9	1.2	2.8	25	20	23	66	1.64	30	34	.08	4.3	1.0	1.2	2.7	25	21
3	22	58	1.60	32	36	.08	3.8	1.3	1.0	2.3	22	24	22	59	1.60	32	36	.08	3.8	1.3	1.0	2.2	23	25
4	21	64	1.65	31	35	.08	4.9	1.4	1.0	3.2	23	21	21	65	1.65	31	35	.08	4.9	1.5	1.0	3.1	23	22
5	20	71	1.70	32	36	.08	3.4	1.2	1.2	2.1	24	20	20	72	1.70	30	36	.08	3.9	1.3	1.2	2.1	24	20
6	24	61	1.70	32	36	.08	4.9	1.2	1.1	3.1	21	22	24	61	1.70	32	36	.08	4.9	1.3	1.1	3.1	21	22
7	25	71	1.68	32	36	.08	4.4	1.0	0.9	2.9	225	21	25	71	1.68	33	37	.08	4.4	1.2	0.9	2.7	25	21
8	25	57	1.72	30	34	.08	4.6	1.4	1.2	2.6	19	21	25	59	1.72	31	35	.08	4.6	1.5	1.2	2.5	19	21
9	25	66	1.70	29	33	.08	4.4	1.0	1.0	2.9	22	19	25	68	1.70	30	34	.08	4.4	1.1	1.0	2.8	23	20
10	23	64	1.64	30	34	.08	4.8	1.0	0.9	3.3	23	19	23	65	1.64	29	34	.08	4.8	1.2	0.9	3.1	24	20
11	24	60	1.60	30	34	.08	3.8	1.3	1.2	1.9	23	22	24	62	1.60	31	35	.08	3.8	1.5	1.2	1.7	24	23
12	22	64	1.64	30	34	.08	4.6	1.2	1.2	2.8	23	20	22	65	1.64	31	35	.08	4.6	1.4	1.2	2.6	24	21
13	21	62	1.68	32	36	.08	4.4	1.0	1.1	2.9	21	18	21	64	1.68	32	36	.08	4.4	1.2	1.1	2.7	22	19
14	20	65	1.68	29	35	.08	4.6	1.0	1.1	3.1	23	19	20	67	1.68	30	35	.08	4.6	1.1	1.1	3.0	23	20
15	18	61	1.70	30	33	.08	4.2	1.2	1.1	2.5	21	20	18	63	1.70	31	34	.08	4.2	1.3	1.1	2.4	21	21
16	18	60	1.64	29	34	.08	3.2	1.2	1.0	1.5	22	20	18	62	1.64	30	36	.08	3.2	1.3	1.0	1.4	23	21
17	19	61	1.60	31	35	.08	3.5	1.0	1.1	2.0	23	22	19	62	1.60	32	36	.08	3.5	1.2	1.1	2.8	24	21
18	23	65	1.68	32	36	.08	3.4	1.2	1.0	1.7	23	21	23	66	1.68	32	36	.08	3.4	1.3	1.0	1.6	23	22

RAW VALUES OF PRE AND POST TRAINING BLOOD SAMPLES IN SUBJECTS FOR INTERNAL TRAINING

RESULT OF PRE-EXERCISE TEST RAW DATA

PRE TRAINING VALUES OF BLOOD													POST TRAINING VALUES OF BLOOD										
S/N	AGE	WEIGHT KG	HEIGHT (M)	WAIST CIRCUM	HIP CIRC UM	WHR	TC	TG	HDL-C	LDL-C	BMI	I/INDEX	AGE	WEIGHT KG	HEIGHT (M)	WAIST CIRCUM	HIP CIRC UM	WHR	TC	TG	HDL-C	LDL-C	BMI	I/INDEX
1	23	58	1.68	32	36	.08	4.4	1.2	1.2	2.0	20	20	23	57.5	1.68	29	36	.08	3.8	1.0	1.5	2.5	20	20
2	23	60	1.70	31	36	.08	4.5	2.2	1.3	0.9	20	22	23	59.5	1.70	31	35	.08	3.7	1.2	1.4	2.9	20	22
3	19	70	1.68	32	35	.08	4.8	1.0	1.3	2.8	19	23	19	70	1.68	30	35	.08	4.4	1.0	1.4	3.1	19	23
4	24	62	1.65	31	35	.08	4.4	1.1	1.4	2.0	22	22	24	61	1.65	30	34	.08	3.8	1.0	1.5	2.7	22	22
5	17	55	1.72	30	34	.08	4.8	1.2	1.0	3.3	18	21	17	54.5	1.72	30	34	.08	4.8	1.0	1.1	3.3	18	21
6	24	75	1.74	31	35	.08	4.7	1.2	1.2	3.2	24	18	24	74.5	1.74	30	34	.08	4.3	0.5	1.4	3.7	24	18
7	25	55	1.60	31	35	.08	5.0	1.0	1.2	3.7	21	23	25	54.8	1.60	30	34	.08	5.0	0.7	1.3	3.8	21	23
8	22	572	1.70	30	34	.08	4.8	0.9	1.3	2.1	24	19	22	71.8	1.70	30	34	.08	3.5	0.8	1.5	3.4	24	19
9	21	73	1.68	31	35	.08	4.2	1.0	1.2	3.6	25	21	21	72	1.68	30	36	.08	5.2	0.6	1.5	2.3	25	21
10	25	55	1.72	32	36	.08	5.0	1.0	1.2	3.1	18	23	25	55	1.72	31	35	.08	4.7	1.3	1.3	3.4	18	23
11	24	70	1.74	32	36	.08	4.9	1.3	1.2	2.8	23	21	24	70	1.74	31	37	.08	4.1	0.7	1.5	3.6	23	21
12	21	65	1.64	33	37	.08	4.4	1.0	1.3	3.3	24	23	21	65	1.64	32	36	.08	4.3	0.4	1.5	2.7	24	23
13	24	55	1.70	31	35	.08	4.6	1.1	1.2	2.2	19	22	24	55	1.70	30	35	.08	3.8	1.7	1.4	2.7	19	22
14	22	66	1.68	31	35	.08	4.6	2.5	1.2	2.9	23	20	22	65.5	1.68	30	34	.08	4.5	1.2	1.3	3.0	23	20
15	15	74	1.70	33	33	.08	5.8	1.4	1.2	3.7	25	21	15	74	1.70	32	36	.08	5.2	1.3	1.5	4.2	25	21
16	22	65	1.76	32	37	.08	5.0	1.0	1.2	3.1	22	23	22	65	1.76	32	36	.08	4.9	1.0	1.4	3.3	22	23
17	21	56	1.60	32	36	.08	4.8	1.0	1.2	2.6	21	21	21	56	1.60	30	34	.08	4.9	1.3	1.5	3.0	21	21
18	20	58	1.65	30	34	.08	4.6	1.0	1.0	3.5	21	21	20	57.8	1.65	32	36	.08	4.6	0.5	1.3	3.5	21	21