EFFECTS OF MODERATE INTENSITY INTERVAL TRAINING IN RUNNING ON BODY FAT PATTERNS, BLOOD PRESSURE, SERUM GLUCOSE, LIPIDS AND LIPOPROTEINS OF SEDENTARY NIGERIAN MALE ADULTS.

BY

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NOVEMBER 2012

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BY

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(Ph.D/EDUC/2882/2009-10)

**A DISSERTATION SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES,
AHMADU BELLO UNIVERSITY, ZARIA
NIGERIA**

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF DOCTOR OF PHILOSOPHY IN EXERCISE AND SPORTS SCIENCE,

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

NOVEMBER 2012.

DECLARATION

I hereby declare that this dissertation has been written by me and that it is a record of my own research work. It has not been presented in a previous application for higher degree. All quotations have been indicated and the sources of information are specifically acknowledged by means of references.

_____ _____

Richard S. JATAU **Date**

CERTIFICATION

This dissertation entitled "EFFECTS OF MODERATE INTENSITY INTERVAL TRAINING IN RUNNING ON BODY FAT PATTERNS, BLOOD PRESSURE, SERUM GLUCOSE, LIPIDS AND LIPOPROTEINS OF SEDENTARY NIGERIAN MALE ADULTS" by Richard Shahbogawi JATAU meets the regulations governing the award of the degree of Doctor of Philosophy (Ph.D) in Exercise and Sports Science, Ahmadu Bello University, Zaria, and is approved for its contribution to knowledge and literary presentation.

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DEDICATION

This dissertation is dedicated to my mother Mrs, Bare Wulajoko Jatau, who paid the three shillings which made me start schooling, and my wife Fibi and the children who missed me greatly during the period spent on this programme.

ACKNOWLEDGEMENT

The researcher acknowledges the various steps taken by Professor K. Venkateswarlu whose interest in this work has been particularly tremendous and unequalled. The brilliant and scholarly inputs, energy and the time he devoted to this work are highly appreciated. Professor C.E. Dikki was more than a supervisor as far as this doctoral programme was concerned. The researcher appreciates most sincerely and with all sense of humility, his contribution and direction. Professor F.R. Haruna is being appreciated for his technical observations, criticism, direction and readiness to assist. The cooperation of the Provost, staff and students of the Adamawa State College of Nursing and Midwifery is highly acknowledged and appreciated, particularly those who volunteered as subjects for this research.

Mrs. Fibi R. Jatau and the children who missed the reseacher's companionship during the years spent on this programme, deserve special appreciation.

The staff of the Chemical Pathology Laboratory of the Federal Medical Centre, Yola must be appreciated for their assistance and for taking blood samples and analyzing them.

Lastly and not the least, Dr. E.A. Gunen,, the Postgraduate Studies Coordinator of the Physical and Health Education Department who on a person to person level contributed immensely to this work. The researcher sincerely appreciates his contribution. The academic and non academic staff of the Physical and Health Education Department deserves my gratitude for helping individually and collectively.

ABSTRACT

This study examined the effects of 12 -week moderate intensity interval training in running on body fat patterns, lipids and lipoproteins, blood glucose and cardiovascular efficiency variables of sedentary Nigerian male adults. The body fat pattern variables included weight, waist circumference, hip circumference, Body Mass Index, Waist- Hip-Ratio and Conicity Index. lipids and lipoproteins included Total Cholesterol, Triglycerides, High Density Lipoprotein Cholesterol and Low Density Lipoprotein Cholesterol, fasting blood glucose. The variables covered under cardiovascular efficiency included systolic and diastolic Blood Pressure, Mean Arterial Pressure, Resting Pulse Rate and Double Product. A 2 x 3 factorial design was used for this study in which 30 medically certified healthy sedentary adult men aged, between 20-30 were selected after obtaining their informed consent and were randomly divided into experimental and control groups of 15 subjects each.. The experimental group went through the training using running as an exercise mode performed at 55-68% of maximum heart rate. The training was done 4 times a week for 12 weeks. During the intervention period, three tests were conducted on both groups at baseline, midway and immediately after the termination of training at week 12.The data obtained was analysed using the Statistical Package of the Social Sciences (SPSS) of the Iya Abubakar Computer Center of the Ahmadu Bello University Zaria Nigeria. The descriptive statistic of the mean and standard deviation were used for data description while the independent t-test was employed to determine the difference between the experimental and control groups. The comparison of the difference between changes in test periods week zero ,sixth and twelfth were made using the repeated measure ANOVA. The result of this study indicated significant decreases in waist circumference and cardiovascular efficiency variables. However, 12 week moderate intensity interval training in running performed 4 times weekly was not found to be adequate to produce significant changes in weight, hip circumference, Body Mass Index, Waist-Hip-Ratio, Conicity Index.,Serum glucose, lipids and lipoproteins of sedentary Nigerian male adults. The not significant changes observed in the other variables may be attributed to the short duration of the intervention or the exercise intensity used in this investigation. The changes observed in these variables however, were minimal and were visible especially in week 12. In the light of these findings, it is recommended that moderate intensity interval training in running can be used to improve cardiovascular efficiency or fitness and reduction in waist circumference of sedentary Nigerian male adults. Following the changes observed towards the end of the training, it is also recommended that longer duration and a higher intensity interval training may be used to bring about the desired changes in variables such as hip circumference, BMI, WHR, CI, blood glucose, lipids and lipoproteins of sedentary Nigerian male adults.

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OPERATIONAL DEFINITION OF TERMS:

Moderate intensity: The effort employed in the performance of physical exercise based on percentage of the maximum heart rate.(low, moderate and high) 55-69% maximum heart rate is moderate intensity (Nieman, 2003).

Sedentary: Not engaged in structured exercise training or sports for a period not less than six months.

Lipids: refers to total cholesterol and triglycerides.

Lipoproteins: stands for high- density lipoprotein cholesterol and low –density lipoprotein cholesterol.

Cardiovascular efficiency variables: Systolic blood pressure, diastolic blood pressure, mean arterial pressure, pulse rate and double product.

Body fat patterns: Weight, waist circumference, hip circumference, waist to hip ratio body mass index and conicity index.

Pre- training: Week 'zero'

Mid- training: Week 6 .

Post- training: Week 12 .

ABBREVIATIONS:

TC:	Total Cholesterol.
TG:	Triglycerides.
HDL-C:	High Density Lipoprotein Cholesterol.
LDL-C:	Low Density Lipoprotein Cholesterol.
WHR:	Waist Hip Ratio.
BMI:	Body Mass Index.
C.I.:	Conicity Index.
RPR:	Resting Pulse Rate.
DP:	Double Product.
MAP:	Mean Arterial Pressure.
CVD:	Cardiovascular Disease.

CHAPTER ONE

1.0. INTRODUCTION

1.1 **Background to the** Study

Numerous reports have indicated that abnormal blood lipid levels like total cholesterol (TC), triglycerides (TG), low- density lipoprotein cholesterol (LDL-C), and high density lipoprotein cholesterol (HDL-C) predispose individuals to cardiovascular and metabolic diseases (Ginsberg, 1994; Gotto, 1994; Glew, Corn, Cadena,Crossey and Okolo, 1994; Chrysohoou, Panagiotakes,Pitsavas, Kosmas, Barbetseas,Karagiorga, Ladi, and Stepandis, 2000; Venkateswarlu, 2011). Serum lipid concentrations are often used to evaluate the risk of coronary heart disease (CHD). Low serum concentrations of HDL-C together with high serum concentration of TC, TG and LDL-C are associated with an increased risk of coronary heart disease (Ginsberg, 1994, Ponka 1998). Obesity, which is a product of fat accumulation leads to increased risk for type 2 diabetes and cardiovascular disease (CVD). Abdominal visceral adiposity is strongly linked to insulin resistance, type 2 diabetes mellitus, hypertension and dyslipidaemia, where such association seems to be much less consistent with respect to subcutaneous fat mass (Evans, et al; 1984). Such high levels of lipids are risk factors for hypertension, elevated blood sugar, pulmonary dysfunction, including asthma and sleep apnoea, depression, gastro-intestinal disorders, type 2 diabetes and cardiovascular disease with strong impact on diabetes risk (McArdle, 2009). Similarly, Montague (2000) Keru and Ranganathan (2001), Mokdad and Ford (2001), Marett (2003) and Yusuf, Hawken, Qumpnuus, et al; (2004) observed that excess visceral fat, central obesity (belly fat), where the belly protrudes excessively, correlate strongly with CVD, insulin resistance, inflammatory disease and obesity-related diseases. In addition, abdominal obesity has been very intimately associated with

cardiovascular and metabolic risks (Despres, Moorjani, Nadeau; et al, 1990; Hunter, Synder, Kekes-Szobo; et al; 1994; Love-Joy, Dela Brefonae, Klemperer, et al; 1996, Albu, Murphy, Frager, et al; 1997; Venkateswarlu, 2010). Inra-abdominal adipose tissue accumulation (visceral fat accumulation), which is an indication of centripetal obesity, has been shown to be particularly related to several disease factors (Ross, Dagnone and Jone, 2000, Kamel, McNeil and Van Wijik, 2000, Venkateswarlu, 2010). Other studies also reported a causal relationship between central adiposity and adverse levels of cardiovascular risk factors in both children and adult

(Okosun, Liao, Rotimi, et al; 2000; Okura, Nakata andTanaka, 2003; McCarthy et al; 2005; Yang, Lei, Chen, et al, 2006; Gutin , Johnson and Humphries, 2007)

Following the realization that physical inactivity is one of the principal risk factors of coronary heart diseases and obesity, exercise scientists have focused attention on the use of physical activity as one of the management and preventive strategies. Several experiments, using physical exercise modes for modification, control and prevention of serum lipids and lipoproteins, body fat patterns, BP and blood glucose, have been carried out with encouraging results (Gilliam and Freedson, 1980; Ignigo and Mahon, 1995; Adeyanju, 2002; Muhammad, 2003; Arogbonlo, 2004). The physical exercises used in these studies vary in terms of types, intensity, duration, mode and frequency of exercise and duration of training. Some of them observed significant effects, while others observed little or no differences.

There is an existing relationship between increased physical activity and fitness with reduction in the risk of cardiovascular disease (Blair et al 1989) the type of change that can be brought about by physical activity depends largely on the mode duration and intensity of the physical exercise. An exercise may be classified as vigorous, moderate or low in terms of its

intensity and each may bring about different changes depending on what it was intended for. A statement issued by the Centre for Disease Control (CDC) and the American College of Sports Medicines (ACSM) suggested that exercise may not have to be vigorous or continuous to confer benefits on the participant. Thirty minutes of moderate intensity exercise is recommended for adults to derive full benefits (Pate et al; 1995). For healthy adults to develop and maintain cardiovascular and respiration fitness and proper body composition, the ACSM emphasized that the intensity of exercise needed to be between 50-85% of maximum heart rate n which is approximately the same as 50-85% of maximum oxygen uptake reserve. (Costil 1988; ACSM 200). Maximum heart rate reserve (HRR) and maximum oxygen uptake ($V0_2$ max) are calculated from the difference between resting and maximum heart rate and resting and maximum $V0_2$ max respectively. In estimating training intensity, a percentage of the HRR is added to the resting heart rate. Exercise or training intensity is therefore expressed in percentages. Nieman (2003) classified physical activity intensities into three main groups as low intensity 35-54%, moderate intensity 55-69% and high intensity $\geq$ 70% of maximum heart rate reserve. This intensity can be applied for any form of training and physical exercise mode. Training may be continuous or intermittent depending on choice and what is needed to be achieved. Mc Ardle et al, (2010) stated that with correct spacing of exercise and rest intervals, one can perform extra ordinary amount of intense physical activity, which is not normally possible in continuous type.

In interval training, consideration must be made for intensity of the exercise interval, duration of the exercise interval, length of relief interval and the number of repetitions of the exercise-relief interval. The rationale for interval training is based on sound physiological and energy metabolism mechanism. In a continuous training, anaerobic glycolysis forms the large portion of energy requirement; which within a minute or two, lactate level rises and the runner

gates fatigued. In interval training, however, the relief interval does not allow for lactate built up thereby resulting into minimal fatigue. The systematic day to day approach of this training mode enables one to easily observe and record progress in performance variables. One obvious advantage it has over continuous running is that it provides a means of performing large amounts of high-intensity exercise in a short time. It gives more rapid improvement in energy potential than in other conditioning methods. It also has the advantage of being performed everywhere and requires little or no special equipments. This method may be more effective at inducing fat loss than simply running at a moderate intensity level for the same duration. Work should be selected based on the actual needs of the athletes or patient. Interval training prescriptions are made up of Long duration work intervals performed at low intensities, medium duration intervals performed at moderate intensities, and short work efforts performed at higher intensities. It is important in the choice of exercise or work especially for the treatment or prevention of cardiovascular disease to consider foremost, the type, intensity, and quantity. An activity which reduces cardiovascular disease risk factors and confers many other health benefits does not require a structured or vigorous exercise programme. This implies that the majority of benefits of physical activity can be gained by performing moderate intensity activities (Blair et al, 1995). A moderate level of physical activity confers health benefits, however, it has to be performed regularly to maintain the effects. Moderate intensity activity therefore performed by previously sedentary individuals result in significant improvements in many health related outcomes, and is likely to be sustained as opposed to high intensity activities (Blair et al, 1995).

Exercise intensity, frequency, duration and type play significant role in their effectiveness. For example, high intensity activity is associated with an increased risk of injury, discontinuation of activity or acute cardiac events during activity. On the other hand, a very low

intensity activity without increased duration and frequency may not produce the desired effect. Interval training has been used in several studies. However, records regarding the use of moderate intensity interval training in running, especially in Nigeria, are not handy. This investigation was therefore proposed to investigate the effect of moderate intensity interval training in running on body fat patterns, BP, serum glucose, lipids and lipoproteins of sedentary Nigerian male adults.

1.2 Statement of the Problem

The projections made by Murray and Lopez (1996), Reddy and Yusuf (1998) that by the year 2020, cardiovascular disease will become the leading cause of global health burden, accounting for 73% of total global mortality and 56% of total morbidity is quite threatening and frightening. Africa, before now, maintained a low record in cardiovascular disease prevalence, but the World Health Organization (WHO) in (1999),showed that, in most African countries, CVD is the second most common cause of death after infectious diseases. In this regard, African countries will have to cope with infectious diseases as well as degenerative and chronic abnormalities like those affecting the cardiovascular system. This apart, the socio-economic effects of treating, preventing and controlling CVD will be overwhelming. In a study conducted on CVD and mortality in sub-Saharan Africa (Jamison, Feachem and Makgoba, 2006), it was observed that the financial and social costs of CVD epidemic may have negative impact on development and the alleviation of poverty in Africa.

To cope with this threat, the developed and the developing nations especially African countries, must make efforts to control the epidemic of CVD. Cases of illnesses related to CVD are becoming more rampant in Nigeria, today among both the city and rural dwellers. Lifestyles

such, as high cigarette smoking, alcohol consumption, fast food vending, inactivity, and poor nutritional practices, are already becoming common in Nigeria and many other African countries. Instead of learning from the experiences of countries such as America and Europe and take adequate preventive measures, there are little or no serious efforts being made towards controlling the risk factors of CVD among Africans.

The implication of the above is that efforts should be made towards finding a relatively cheap and effective methods for both prevention and treatment of cardiovascular and metabolic diseases. The use of physical exercise training as modifier of most CVD risk factors is ongoing. The objective of each effort is to provide specificity with respect to the prescription of exercise for any particular condition. Like in other clinical practices, prescription of exercise must be targeted at some specific disease factors and conditions. Consideration should also be made to take care of gender, age and other disparities; among various populations. The attempt in their research was to try the use of moderate intensity interval training in running which is not known to have been used especially on sedentary Nigerian male adults. Since the optimal intensity or type of training necessary for reduction in CVD risk factors is unknown and the recommendations to make for exercise that will confer specific health benefits remains or is still elusive. This research was therefore carried out to investigate the effect of moderate intensity interval training in running on body fat patters, blood pressure serum glucose, lipids and lipoproteins of sedentary Nigerian male adults

1.3 Research Questions

This study was proposed to answer the following specific research questions:

1. Is there any effect of 12- week moderate intensity interval training in running on centripetal and peripheral body fat patterns of sedentary Nigerian male adults?

2. Is there any effect of 12- week moderate intensity interval training in running on blood pressure of sedentary Nigerian male adults?
3. Is there any effect of 12 week moderate intensity interval training in running on serum lipids and lipoproteins of sedentary Nigerian male adults?
4. Is there any effect of 12-week moderate intensity interval training in running on serum glucose levels of sedentary Nigerian male adults?
5. Is there going to be any difference in the magnitude of changes in the variables under investigation between the three testing levels?

1.4 Purpose of the Study

The purpose of this study was to investigate the effect of 12 week moderate intensity interval training in running on:

1. The effect of 12 week moderate intensity interval training in running on body fat patterns of sedentary Nigerian male adults.
2. To investigate the effect of 12 wk moderate intensity interval training in running on blood pressure of sedentary Nigerian male adults.
3. What will be the effect of 12 wk moderate intensity interval training in running on fasting blood glucose of sedentary Nigerian male adults.
4. The effect of 12 week moderate intensity interval training in running on serum levels of lipids and lipoproteins of sedentary Nigerian male adults.

1.5 Basic Assumptions

On the basis of available research evidence, the following assumptions were made for this research.

1. Regular physical exercise affects body weight and body fat patterns.
2. Regular moderate to vigorous physical activity improves body composition, cardiovascular and metabolic health and reduces disease risk.
3. Lipid and lipoprotein, body fat patterns, blood pressure and blood sugar are influenced by physical activity.

1.6 Hypotheses

On the basis of the research questions raised, the following hypotheses were formulated for the purpose of this study.

Major Hypotheses

There is no significant effect of 12 weeks moderate intensity interval training in running on body fat pattern, blood pressure, blood glucose, serum lipids and lipoprotein of sedentary Nigerian male adults.

Sub- Hypotheses:

The following null hypotheses were formulated.

1. There is no significant effect of 12 -week moderate intensity interval training in running on body fat patterns of sedentary Nigerian male adults.
2. There is no significant effect of 12- week moderate intensity interval training in running on lipids and lipoproteins of sedentary Nigerian male adults.

3. There is no significant effect of 12- week of moderate intensity interval training in running on blood glucose of sedentary Nigerian male adults.
4. There is no significant effect of 12-week of moderate intensity interval training in running on cardiovascular efficiency variables of sedentary Nigerians male adults.
5. There are no significant differences in magnitude and direction of changes between pre-mid and post- training tests in body fat patterns, blood pressure, serum glucose, lipids and lipoproteins.

1.7 Significance of the Study

This study was justified on the following bases:

The result of this study will show if Moderate intensity Interval Training can be used to improve fat deposits in the different regions of the body of sedentary Nigerian male adults

This investigation is to prove whether moderate intensity interval training in running will be prescribed for modification of serum glucose, lipids and lipoproteins levels of sedentary Nigerian male adults.

To show if moderate intensity interval training in running can be used to assist people with problems associated with cardiovascular efficiency.

This study may shade light on areas of further research on the effect of different modes and intensity of exercise or training on different disease risk factors

1.8 Delimitation of the Study

Participants were adult males aged between 20 – 30 years final year students of the Adamawa State College of Nursing and Midwifery Yola.

The investigation was limited to the following variables:

Body fat patterns: conicity index, waist circumference, hip circumference, WHR and BMI.

Lipids and lipoproteins, included TC, TG, LDL-C and HDL-C.

Blood glucose, and cardiovascular efficiency variables studied were systolic and diastolic BP, mean arterial pressure, (MAP) resting pulse rate and double product (DP) .

1.9 Limitations of the Study

One of the major constraints during this research was the problem of control and monitoring the subjects for the study both in movement and feeding.. It was a bit better and easier for the experimental group because they meet almost daily with the researcher. The control group on the other hand was hardly seen on daily bases except on days when they meet with the researcher. The College Authority had to give some restrictions to ensure that all the groups observe strict campus rules regarding outings from the campus. Observing and monitoring their feeding .Feeding was virtually of common Northern Nigerian foodstuffs such as rice, beans and corn eaten along with beef and fish.

CHAPTER TWO

2.0. REVIEW OF RELATED LITERATURE

2.1 Introduction

This research was carried out to investigate the effect of 12 weeks of moderate intensity interval training in running on body fat patterns, blood pressure, serum glucose, lipids and lipoproteins of sedentary Nigerian male adults.

To understand the metabolic processes leading to high or low concentration of fat, changes in blood pressure, lipids and glucose accumulation in the body and their reduction by exercise, relevant literature were consulted and reviewed to give the researcher a good understanding of the field under investigation. Included were the following subheadings:

2:2 Nature, Risk Factors and Changing Prevalence of CVD

2.2.1 Risk factors for Cardiovascular Disease

2.2.2 Unalterable Risk Factors

2.2.3 Alterable or modifiable risk factors

2.2.4 Cardiovascular Disease

2:3 Body Fat Distribution Pattern:

2:.4: Anthropometric Assessment of Body fat pattern:

2.4.1 Body Mass Index

2.4.2. Body Circumferences

2.4.3 Waist-Hip-Ratio

2.4.4 Conicity Index**:**

2.5 Blood Pressure, Classification, Measurement and Complications:

2.5 1 Mean arterial pressure

2.6 Hypertension and Exercise:

2.6.1 Exercise in the Prevention of Hypertension:

2.7 Exercise and Cardiovascular Disease Risk Factors:

2.7.1 Effects of exercise on CVD risk factors

2.8 Impact of Physical Activity on Blood Lipids and Lipoproteins:

2.9 Serum glucose homeostasis and physical exercise training.

2.10 Interval Training

2:11 Summary

2.2 Nature, Risk Factors and Changing Prevalence of CVD

The cardiovascular system includes the heart, arteries and veins with a primary responsibility of pumping and distributing blood to and from the body cells and tissues. Like other body organs, this system suffers from various forms of obstructive disorders related mainly to unhealthy lifestyles (United State Department of Health and Human Services, 2000, American Heart Association, 2001). CVD include those diseases of the heart and its blood vessels and there are over twenty known diseases of the cardiovascular system (Nieman, 2003). Atherosclerosis, the building up of fatty plague material in the inner layer of the blood vessels is the underlying factor in 85% of CVD. The physiology is simply the blocking of blood vessels, leading to less or no blood reaching the areas it supplies. This result in heart attack or myocardial infarction. When the blockage occurs in the vessels in the brain, stroke occurs. The atherosclerotic plague, range from small yellowish streaks to advanced lesions with ulceration or blood clot within the blood vessels (Sniderman, Pedersen and Kjekshurs 1997; Vogel, 1997; McGill, McMahan, Hendric et al, 2000).

Atherosclerosis develops gradually and in stages. The arterial wall is first injured by factors such as hypertension, high blood cholesterol levels (hypercholesterolemia) oxidized LDL, cigarette smoking, toxins, viruses and blood flow turbulence (Nieman, 2003). The injury leads to impairment in the normal function of the endothelium and a chronic inflammatory response begins (Vogel, 1997).In reaction to the injury, monocytes and T. cells (immune cells) penetrate through the endothelium into the underlying intima (inner layer of the arterial wall) (Sniderman et al, 1997, Vogel, 1997). This is followed by the conversion of the monocytes into microphages, scavenger cells that ingest oxidized low density lipoprotein and other substances. By accumulating large amounts of cholesterol from oxidized LDL, the macrophages are transformed into foam cells. In addition, oxidized LDL causes further injury to the endothelium, attracting more monocytes, inducing a vicious cycle that leads to the development of a fatty streak, a precursor to plague (Sneiderman, 1997). The injury and impaired endothelial cells attract platelets and begin to release growth factors that stimulate the migration of smooth-muscle cells from the outer layers of the artery wall into the intima, where they proliferate abnormally. The macrophages and the smooth-muscle cells begin to release collagen and other proteins which form the fibrous components of atherosclerosis. The engorged foam cells then die and release cholesterol debris into the artery wall (United State. Department. Of Health and Human Services, 2000). At this stage, the mature plague is made up of a complex mixture of foam cells, smooth muscle cells cholesterol debris and fibrous proteins. With time, the plague may become harden and develop cracks and ulcers thereby prompting the formation of blood clots that can suddenly close up the narrowed artery lumen, causing a heart attack (Vogel,1997, American Heart Association, 2000, McGill et al, 2000). There is increasing evidence that atherosclerosis begins in childhood and progresses from fatty streaks to raised lesions in

adolescence and young adulthood. From the developmental stages of arthrosclerosis, inner layer of artery walls become thick and irregular due to deposits of fat, cholesterol and other substances. This building is sometimes called a plague where the artery wall is lined up with the deposits, which gradually narrow the vessel lumen making blood passage more difficult (blood pressure) ultimately, arterial lumen becomes completely blocked causing either a heart attack or stroke.

2.2.1 Risk factors for Cardiovascular Disease

Risk factors CVD are defined by Nieman (2003) as personal habits or characteristics that medical research has shown to be associated with an increased risk of heart disease. In other terms, risk factors can be described as variables that are associated with an increased risk or probability that someone may develop morbidity or incur mortality from a specific disease. Major risk factors are those that medical research has shown to definitely be associated with a significant increase in the risk of developing morbidity or mortality from specific disease. Among the major factors for CVD are those which can be altered and those which cannot be altered (Nieman, 2003).

2.2.2 Unalterable Risk Factors

McArdle, Katch and Katch (2010) identified the following unalterable risk factor as:

I. Heredity: Those with family history of CHD are more likely to develop CVD than those with no such history.

II. Gender: Men are said to be at greater risk of coronary heart disease than women especially through middle age. Women's estrogen is said to be helpful in preventing CVD. When the level falls however, women become more vulnerable.

III. Age: The vulnerability increases with age especially after the age of 65 years.

IV. Nieman (2003) added the fourth factor as race – Some races are more Vulnerable to CVD than others.

2.2.3 Alterable or modifiable risk factors

i. Hypertension or high BP: The force exerted by blood as it flows through the arteries and arterioles. This is the function of the heart as it forces the blood to flow and the resistance of the arteries to the blood flow. The pressure is grouped as follows:((Nieman 2003).

< 130/< 80 – normal

130 – 139/85-89, High normal

140-159/90-99, Stage I mild hypertension

160-179/100-109, Stage II moderate hypertension.

≥180/ ≥ 10, Stage III severe hypertension.

ii. Smoking tobacco – Smoking tobacco releases nicotine and some 1,200 other toxic compounds into the blood stream and dramatically hastens the process of atherosclerosis. It may also increase the likelihood of blood clot formation. It increases heart rate, which increases blood pressure. It also decreases blood HDL.

iii. Diabetes: Diabetes mellitus is a metabolic disease in which the body fails to maintain normal blood glucose level. This affects cholesterol levels and, therefore becomes a

cardiovascular disease risk. Incidences of cardiovascular disorder are more among people with diabetes (Nieman 2003).

iv. Hyperlipidaemia: Abnormalities in blood lipids and lipoproteins increase stroke risk. Total cholesterol – between 200 and 239 milligrams per deciliter is a borderline high while 240 milligrams/deciliter or higher is regarded as high and dangerous. The desirable level is below 200 mg/dl of blood (MacArdle et al, 2010).

LDL-C

Desirable < 130 mg/dl

Borderline high between 130 and 159 mg/dl.

High, 160 mg/dl.

v. Physical inactivity: Blair, Kampert, Kohl, et al (1996), found that people with low levels of cardiovascular endurance had risks of dying from CDV two to four times higher than those that had better cardiovascular fitness. Being physically active has positive impact on a number of the risk factors identified. It can help lower blood pressure, reduce body fat, decrease cholesterol and triglyceride levels, improve HDL-C levels, control diabetes and manage stress.

vi. Stress or psychological stress.

vii. Alcohol abuse.

viii. Poor diet selection.

ix. Obesity or overweight.

2.2.4 Cardiovascular Disease

The relative and absolute importance of CVD in countries of sub-Saharan Africa is thought to be increasing, but information on morbidity, mortality, prevalence of disease and risk factors is scanty (Muna 1993; Razum 1996). Data from 20 years ago consisted largely of hospital records, which reported that 8 to 12 percent of hospital admissions were due to heart disease in several countries in southern Africa (Vaughan, 1977), hypertensive and rheumatic heart disease (RHD) accounted for 40 to 60 percent of these admissions. There were few recorded episodes of ischaemic cardiac events. By 1977, such causes of death as syphilitic heart disease were rarely recorded, despite the continuing high frequency of acute syphilis. In the 1970s, cardiac disease was common among young people in Kampala, Uganda, where autopsy data demonstrated cardiac disease to be a cause of death in 15.3 percent of males older than 60 years and in 13.1 percent of males younger than 30 years (Drury, 1972). In 1980, the Zambian Ministry of Health report of admissions and causes of death in all government, mining, and mission hospitals listed 2.06 percent of deaths in those under age 14 as cardiac in origin, compared with 14.50 percent in those over age 14. As regards morbidity, 0.62 percent of hospital admissions of children under 14 in that same study were cardiac in origin compared with 4.23 percent of those individuals over age 14 (Hutt, 1990). By 1990, CVD had become the third most common cause of death in a prospective autopsy study in 90 of the 167 deaths in one year at Tshepong Hospital in the North West Province of South Africa (Steenkamp et al, 1992). In patients over age 35 years, CVD was the most common cause of death. CVD accounted for 32 percent of the overall deaths . Among these, intracerebral haemorrhage was found in 50 percent and cerebral infarction in 29 percent of cases. Fifty-seven percent of cardiovascular deaths were due to cardiac conditions, the most common being pulmonary hypertension (31 percent), dilated

cardiomyopathy and chronic rheumatic valvular disease (17 percent each), and hypertensive heart disease (14 percent). Only 3 percent of the examined vessels had signs of severe atherosclerosis. The clinical diagnosis was the same as the final autopsy diagnosis in only 38 percent of cases, emphasizing the importance of performing autopsies to obtain reliable mortality statistics in African countries. In a prospective study among elderly patients in Kenyatta National Hospital, Nairobi, Kenya, in 1991 to 1992, clinical evidence of CVD was present in 40 percent of the patients evaluated; 54 percent were hypertensive, 53 percent had arrhythmia, and 49 percent had congestive cardiac failure (Lodenyo, McLigeyo andOgola 1997). A prospective study of 708 subjects with CVD was conducted between January 1992 and December 1995 in Ghana (Amoah, 2000). Participants were evaluated clinically, with ancillary laboratory tests, chest X-ray, electrocardiography, and two-dimensional echocardiography with doppler and colour flow mapping. Hypertensive heart disease (n = 133), RHD (n = 123), idiopathic cardiomyopathy (n = 103), congenital heart disease (n = 90), and coronary artery disease (CAD; (n = 80) were the major causes of cardiovascular morbidity. The mean age of the subjects was 41.6; peak incidence of CVD occurred during the decile 40–49 years of age.

A retrospective study in 1995 in Cameroon of 312 adult patients with CVD, average age 44 years, revealed high blood pressure (38.5 percent), rheumatic valvular heart diseases (25.6 percent), cardiomyopathies (22.5 percent), and other cardiovascular diseases (13.5 percent) (Kotto and Bouelet, 2000). Rheumatic valvulopathies were predominant among the age group 20 to 39 years, hypertension was predominant from the age of 40 years, and cardiomyopathies were observed in the age range 20 to 60 years.

A Cameroonian study between 1992 and 1997 ranked coronary artery disease eighth among the CVDs, registered with a prevalence of 1.53 percent (2.42 percent in males and 0.45 percent in females). Myocardial infarction was the most frequent clinical form of CAD observed (43 percent), followed by angina pectoris (23 percent), unstable angina (20 percent), and other forms of ischaemic heart disease (13 percent). The cardiovascular risk factors were obesity (80 percent), hypertension (60 percent), dyslipidaemia (43 percent), smoking (36 percent), diabetes/hyperglycemia (26 percent), and hyperuricaemia (20 percent). Seventy-six percent of the patients had at least three cardiovascular risk factors (Mbanya, Minkoulou, Sala, et al, 1998).

In multi-ethnic South Africa, CHD is the major cause of death among white people and South Africans of Indian descent, with incidence rates of 165.3 and 101.2 per 100,000 people, respectively, but only 55.1 per 100,000 among people of mixed descent and 5.3 per 100,000 among black African people. Cerebrovascular disease is the most common cause of CVD death among those of mixed descent, followed by white people and South Africans of Indian descent, and then black African people (73.6, 62.5, and 36.5 per 100,000, respectively) (Bradshaw, Bourne, Schneider, et al, 2003).

In South Africa 90 percent of deaths are certified; of these certifications over 70 percent are by a medical doctor, and cause-specific data are available for most deaths. Gradually shrinking proportions, currently 13 percent, are categorized as "ill-defined." It is quite possible that many deaths from CVD could masquerade as ill-defined deaths, particularly in a country where there is a reluctance to diagnose CVD death in still black people (Mbaya, et al 1998)

2.3 Body Fat Distribution Pattern

In histology, adipose tissue or body fat is loose connective tissue composed of adipocytes. It is technically composed of roughly only 80% fat. Fat in its solitary state exists in the liver and muscle (McArdle et al, 2010). Adipose tissue is derived from lipoblasts. Its main role is to store energy in the form of fat, it cushions and insulates the body. Obesity or being overweight, does not depend on body weight but on the amount of body fat or adipose tissue. This tissue exists as white and brown adipose tissue. Adipose tissue serves as an important endocrine organ by producing hormones such as leptin, resistin and the cytokine (Kershaw and Flier, 2004). Formation is controlled by adipose gene. Adipose tissue was first identified by the Swiss Naturalist Conrad Gessner in 1551 (Cannon and Nedergaard, 2008). In humans fat is located beneath the skin (subcutaneous), around internal organs (visceral fat) in born marrow (yellow) and in breast tissue. It is found in specific locations (adipose depots). The main function of fat is to be a reserve of lipids, which can be burnt to meet the energy needs of the body (Kershaw and Flier, 2004). According to location fat is grouped into (a) abdominal fat, (b) visceral fat, (c) subcutaneous fat and (d) intramuscular fat. Fats in the lower body as in thighs and buttocks are subcutaneous while those in the abdomen are visceral (Bachmanove, Reed,Tordoff, et. al, 2001).

Abdominal Fat: Visceral fat or intra abdominal fat is located inside the abdominal cavity parked in between organs including stomach, liver, intestine and kidney (Bachmanove et al, 2001). Visceral fat is composed of several adipose depots including mesentric, epididymal white adipose tissue and perineal depots. Excess of this type of fat is referred to as central obesity or belly fat (abdomen protrudes excessively). There is a strong correlation between central obesity

and cardiovascular disease (Yusuf et al, 2004). Similarly, it is linked to diabetes, insulin resistance, inflammatory diseases and other obesity- related diseases as observed by Montague and O'Rahily (2000), Keru and Rangnathan (2001), Marett, (2003) and Mokdad, (2003).

In women abdominal fat is suppressed by the hormone estrogen and causes fat deposit to the buttocks, thigh and hips (Mokdad and Ford 2003). After menopause, the ceasation of this hormone returns the fat deposit to the waist. Abdominal fat can be reduced by exercise (Ohkawara, Tanaka, Miyachi, et al, 2007).

Epicardial fat: This is a form of visceral fat deposited around the heart. Such deposit might significantly affect cardiac function (Mazurek, Zhang, Zalewaski, et al, 2003).

Subcutaneous Fat: Most of the non-visceral fat is found below the skin in a region called hypodermis (Marieb and Hoehn, 2008). This fat is not related to many of the classical obesity-related pathologies, such as heart attack or stroke and that there is some evidence that it is protective (Porter,Massaro, Hoffinann, et al, 2009).

The most widely used measure of total or general and abdominal obesity or adiposity are the BMI and waist circumference, respectively. While abdominal obesity is recognized as a major risk factor for CHD, waist circumference and WHR are more strongly associated with metabolic risk factors, incidences of CVD and deaths. (Fouad et al, 2006). Many studies appear to favour waist circumference over BMI in predicting cardiovascular risk and events (Saelens et al , 2007). Visceral and general adiposity have been traced to factors such as poor nutritional habits and practices and physical inactivity (Fouad,et al 2006).

Over the years, a number of scales and measures have been developed to determine the levels of adiposity in man. These measure are meant to predict if an individual is at risk or not of being obese or vulnerable to cardiovascular disease. Among the popular measures are, hip and waist circumferences, BMI, conicity index, waist hip ratio, skinfold, hydrostatic weighing and quetelet index (Okura, et al 2003).

Body fat patterns: Total body fat mass is a product of regional fat deposits found in the body. Fat is found deposited in different parts of the body called regional depots. Fat distribution pattern differs between male and female and even between races (Martin and Daniel, 1993). The regional distribution of fat in humans is referred to as fat patterning. Fat pattern may be observed in the upper and lower regions of the body. Some writers refer to this pattern as central or abdominal and lower or hip locations. Central, upper body or abdominal accumulation of fat is associated with many disease risk factors in adults (Kisseah and Krakower, 1994). It is from the patterning of fat that enables scientist to know the risk of fat accumulation in the body than mere total fat mass.

Bjantrop (1998), observed that increased abdominal or waist accumulation of fat is associated with health risk of hyperlipidaemia, hypertension and coronary heart disease than fat located in the hips and thighs. Fat deposited on the upper abdominal part of the body are also called android obesity and those on the hip as gynoid obesity (Bjantrop 1998).

It was observed by Martin and Daniel (1993) that android obesity is highly associated with cardiovascular health risk than gynoid which is most common in females.

The determination of fat distribution or patterning is being carried out by the use of measurements (not the total body mass). These measures as discussed somewhere in this review, includes height, weight, circumferences and indexes. The commonly used of these include BMI

waist hip ratio (WHR) conicity index (CI) waist circumference, hip circumference and the skinfolds.

2.4.1 Anthropometric Assessment of Body Composition

Owing to lack of highly trained technicians and expensive equipment, laboratory methods are rarely used in field settings. The most common alternative is to use some form of anthropometric methods such as weight – height ratios, body circumferences and skinfold measurements (Baumgartner, Jackson, Mahar, et al, 2007). Among the commonly used are the BMI, WHR, waist circumference, hip circumference, conicity index and the skinfolds. These tools independently or in combination are used to investigate body fat patterns, and their ability to predict disease conditions and proneness. The following discussion attempts to define these tools as measures for assessing body composition, their uses, strength and weakness and the procedure for measurement.

2.4.2 Body Mass Index

The mass of the body refers to the totality of the weight of everything that constitutes the body. BMI is a ratio of body weight and height of an individual. The ratio is used to determine if an individual is overweight, underweight and or obese. Clinicians and researchers use the body mass index derived from body mass and stature (weight and height) to assess normalcy for body weight. The measure exhibits a somewhat higher yet still moderate association with body fat and disease risk than estimates based simply on stature and body mass (McArdle et al, 2010). It categorizes people into underweight or obese. The National Institute of Health,(1998)categorized as follows for adults.

18.5 – 24.9 - normal

25.0 – 29.9 - overweight

30.0 – 34.9 - obesity category I

35.0 – 39.9 - obesity category II

40.0 – above - extreme obesity category III.

In measuring and computation, materials include weighing scale, and standiometer for height or ruler calibrated in kilograms and meters, respectively. Computation is simply done by dividing the weight by height squared ,for example an individual with weight of 97.1 kg and height of 1.753 m,

$BMI = 97.1 \div (1.753)^2$

= 31.6 (obesity category I).

The BMI is said to be most useful for defining the body composition of groups of individuals. It however, lacks accuracy when evaluating the body components of individuals. Its main shortcoming is that the weight factor is not partitioned into fat-free weight and fat height. It is also an insufficient index for disease risk (Powers and Howley, 2007).

2.4.3. Body Circumferences

Certain body circumferences have been used to assess body composition. The approaches followed include body density from combination of body circumference measurements, and using the ratio of hip and waist circumference measurements (Baumgartner, 2007).

i. Waist Circumference: This is a simple measurement of central obesity. It is adjudged to be one of the most accurate anthropometrical indicators of abnormal fat and quite reliable in that respect (Lee, Song and Sung, 2008). Central obesity is fat accumulation in the abdomen due to excess visceral fat. Excess fat in this area can lead to release of fats into

bloodstreams and its buildup is responsible for majority of negative health consequences associated with obesity (McArdle et al, 2001).

In measuring waist circumference, a tape is used to take the measurement as the subject breaths out in a relax manner in a standing position, two measurements may be taken and the average use for accuracy reasons. Waist circumference cut-off values for increased risk of metabolic syndrome are:

Europeans Male, 94 cm; and Female, 80 cm

Asians, Chinese, Japanese, South and Central Americans

Male, 90 cm and female, 80 cm

Africans, Middle East: male, 94 cm and female, 80 cm

>102cm for men and 88 cm for women portains danger of cardiovascular disease (Lee et al, 2008).

Like WHR, waist circumference is strongly associated with risk of artherosclerosis. The major limitation of waist circumference is the great inter-ethnic variability that exists. Cut-off for each ethnic group should be identified and used all the time (Lee et al 2008).

ii. Hip circumference: Hip circumference is measured using a measuring tape. It should be measured as the maximum circumference over the buttocks. Tape must be horizontally placed all round the hip. Seidell, Perusse, Despres, et al (2001) suggested that people with a narrow waist circumference and a large hip circumference have a lower risk of CVD. Large hip circumference is found to be associated with lower risk of diabetes (Snijder, Dekker andVister 2003). Hip circumference gives a picture of low or high fat accumulation in the lower part of the body. This is visible on the buttocks and thighs especially in women. In men, this picture is not so common. Among women, fat

accumulation is more pronounced among young ladies from puberty to premenopausal period. When used in combination with waist circumference, a waist-hip ratio could be calculated for visceral or central obesity. WHR is a good measure for abdominal fat accumulation.

2.4.4 Waist-Hip-Ratio

WHR measures visceral or central obesity. It is a good measure of abdominal fat distribution but is not a measure of absolute abdominal fat mass. Furthermore, WHR is a significant predictor of morbidity and mortality risk, for example, type 2 diabetes, coronary heart disease and death (Ohlson, Larsson and Svardsudd 1985; Ducimeture and Richard, 1989; Lapidus, Bengtsson and Lissner 1990). Follow up studies have confirmed the above and showed that WHR is a stronger predictor of myocardial infarction than BMI or waist circumference alone (Ducimeture and Richard 1989).

Waist, hip ratio is a measure of waist circumference relative to hip circumference. WHR values greater than 0.90 in men and 0.85 in women are proposed to diagnose obesity. WHR is calculated by dividing the waist circumference by the hip circumference.

Limitations include:

1. Not an absolute measure of abdominal fat
    a. When measuring, it is unclear whether an individual has a high WHR due to a high waist circumference or small hip circumference. It should be noted that waist circumference also measures abdominal obesity more than WHR.

2.4.5 Conicity Index:

This is one of the indexes used in the measurement of abdominal adiposity which combines the function of girth, weight and height (Valdezed, Seidal, Ahn, et al, 1993). Conicity index has the advantage of a theoretical range, a build-in adjustment of waist circumference for height and weights, which is said to allow for direct comparison of abdominal adiposity of individuals or between population and does not depend on hip circumference in its computation as used in the waist to hip ratio. Conicity index is positively associated with triglyceride in females. Risk factors such as for cardiovascular and diabetes are visible using this index. Conicity index is computed as abdominal girth or waist circumference, divided by the factor 0.109, multiplied by product of weight divided by height.

$$CI = \frac{\text{waist circumference (cm) x weight/height}}{0.109}$$

Summary of cut-off points for indexes of obesity

Index	**Male**	**Female**
Conicity index	1.25	0.85
Waist-hip-ratio	0.95	1.18
Waist circumference	88.0	83.0
Body mass index	24.0	26.0

2.5 Blood Pressure, Classification, Measurement and ComplicationsBlood pressure: (BP) is the pressure exerted by circulating blood upon the walls of blood vessels. During each heartbeat, BP varies between a maximum (systolic) and a minimum (diastolic) pressure. The

mean BP, due to pumping by the heart and resistance to flow in blood vessels, decreases as the circulating blood moves away from the heart through arteries. BP drops most rapidly along the small arteries and arterioles, and continues to decrease as the blood moves through the capillaries and back to the heart through veins.(Klabunde, 2005) Gravity, valves in veins, and pumping from contraction of skeletal muscles, are some other influences on BP at various places in the body.

The term BP refers to the pressure measured at a person's upper arm. It is measured on the inside of an elbow at the brachial artery, which is the upper arm's major blood vessel that carries blood away from the heart. A person's BP is usually expressed in terms of the systolic pressure over diastolic pressure (mmHg), for example 140/90.

Classification blood pressure for adults:

Category	**systolic, mmHg**	**diastolic, mmHg**
Normal	< 120	< 80
Prehypertension	120 – 139	or 80 – 89
Stage 1 Hypertension	140 – 159	or 90 – 99
Stage 2 Hypertension	160 – 179	or 100 – 109
Hypertensive Crisis	≥ 180	or ≥ 110

The following US classification of blood pressure applies to adults aged 18 and older. It is based on the average of seated BP readings. In the UK, hypertension is considered when a patient's reading is above 140/90 mmHg.

Normal pressure: While average values for arterial pressure could be computed for any given population, there is often a large variation from person to person; arterial pressure also varies in individuals from moment to moment. Additionally, the average of any given population may

have a questionable correlation with its general health, thus the relevance of such average values is equally questionable. However, in a study of 100 subjects with no known history of hypertension, an average blood pressure of 112/64 mmHg was found, (Pesola, Pesola, Nelson et al, 2001).which is the normal value.

Various factors influence a person's average BP and variations. Factors such as age and gender (Rekelhoff and Jane, 2001) influence average values. As adults age, systolic pressure tends to rise and diastolic tends to fall. (Pickering, 2005). In the elderly, BP tends to be above the normal adult range, largely because of reduced flexibility of the arteries. Also, an individual's BP varies with exercise, emotional reactions, sleep, digestion and time of day (Rekelhoff and Jane 2001).

The risk of cardiovascular disease increases progressively above 115/75 mmHg (Appel, Brands, Daniels, et al, 2006). Clinical trials demonstrate that people who maintain arterial pressures at the low end of these pressure ranges have much better long term cardiovascular health. The principal medical debate concerns the aggressiveness and relative value of methods used to lower pressures into this range for those who do not maintain such pressure on their own. Elevations, more commonly seen in older people, though often considered normal, are associated with increased morbidity and mortality. Average blood pressure in (mmHg) (Pickering 2005):

1 year	**6 – 9 years**	**adults**
95/65	100/65	110/65 - 140/90

Measurement: Arterial pressure is most commonly measured via a sphygmomanometer, which historically used the height of a column of mercury to reflect the circulating pressure (Booth,

1977). BP values are generally reported in millimeters of mercury (mmHg), though aneroid and electronic devices do not use mercury. For each heartbeat, BP varies between systolic and diastolic pressures. Systolic pressure is peak pressure in the arteries, which occurs near the end of the cardiac cycle when the ventricles are contracting. Diastolic pressure is minimum pressure in the arteries, which occurs near the beginning of the cardiac cycle, when the ventricles are filled with blood. An example of normal measured values for a resting, healthy adult human is 120 mm Hg systolic and 80 mm Hg diastolic (written as 120/80 mm Hg (Boot 1977).

Systolic and diastolic arterial BPs are not static, but undergo natural variations from one heartbeat to another and throughout the day (in a circadian rhythm). They also change in response to stress, nutritional factors, drugs, disease, exercise, and momentarily from standing up. Sometimes the variations are large. Hypertension refers to arterial pressure being abnormally high, as opposed to hypotension, when it is abnormally low. Along with body temperature, respiratory rate, and pulse rate, BP is one of the four main vital signs routinely monitored by medical professionals.

Palpation method: A minimum systolic value can be roughly estimated by palpation, most often used in emergency situations. Historically, students have been taught that palpation of a radial pulse indicates a minimum BP of 80 mm Hg, a femoral pulse indicates at least 70 mm Hg, and a carotid pulse indicates a minimum of 60 mm Hg. However, at least one study indicated that this method often overestimates patients' systolic BP (Deakin and Low, 2000). The American Heart Association recommends that palpation be used to get an estimate before using the auscultatory method.

Auscultatory method: The auscultatory method (from the Latin word for "listening") uses a stethoscope and a sphygmomanometer. This comprises an inflatable (*Riva-Rocci*) cuff placed around the upper arm at roughly the same vertical height as the heart, attached to a mercury or aneroid manometer. The mercury manometer, considered the gold standard, measures the height of a column of mercury, giving an absolute result without need for calibration and, consequently, not subjected to the errors and drift of calibration which affect other methods. The use of mercury manometers is often required in clinical trials and for the clinical measurement of hypertension in high-risk patients, such as pregnant women (Appel, et al 2006).

A cuff of appropriate size is fitted smoothly and snugly, then inflated manually by repeatedly squeezing a rubber bulb until the artery is completely occluded. Listening with the stethoscope to the brachial artery at the elbow, the examiner slowly releases the pressure in the cuff. When blood just starts to flow in the artery, the turbulent flow creates a "whooshing" or pounding (first Korotkoff sound). The pressure at which this sound is first heard is the systolic BP. The cuff pressure is further released until no sound can be heard (fifth Korotkoff sound), at the diastolic arterial pressure.

Oscillometric method: The oscillometric method was first demonstrated in 1876 and involves the observation of oscillations in the sphygmomanometer cuff pressure, which are caused by the oscillations of blood flow, which is the pulse. The electronic version of this method is sometimes used in long-term measurements and general practice. It uses a sphygmomanometer cuff, like the auscultatory method, but with an electronic pressure sensor (transducer) to observe cuff pressure oscillations, electronics to automatically interpret them, and automatic inflation and deflation of the cuff. The pressure sensor should be calibrated periodically to maintain accuracy.

Oscillometric measurement requires less skill than the auscultatory technique and may be suitable for use by untrained staff and for automated patient home monitoring (Klabunde, 2005).

The cuff is inflated to a pressure initially in excess of the systolic arterial pressure and then reduced to below diastolic pressure over a period of about 30 seconds. When blood flow is nil (cuff pressure exceeding systolic pressure) or unimpeded (cuff pressure below diastolic pressure), cuff pressure will be essentially constant. It is essential that the cuff size is correct: undersized cuffs may yield too high a pressure; oversized cuffs yield too low a pressure. When blood flow is present, but restricted, the cuff pressure, which is monitored by the pressure sensor, will vary periodically in synchrony with the cyclic expansion and contraction of the brachial artery, that is, it will oscillate. The values of systolic and diastolic pressure are computed, not actually measured from the raw data, using an algorithm; the computed results are displayed. Oscillometric monitors may produce inaccurate readings in patients with heart and circulation problems, which include arterial sclerosis, arrhythmia, preeclampsia, *pulses alternans,* and *pulsus paradoxus*.

In practice the different methods do not give identical results; an algorithm and experimentally obtained coefficients are used to adjust the oscillometric results to give readings which match the auscultatory results as well as possible. Some equipment uses computer-aided analysis of the instantaneous arterial pressure waveform to determine the systolic, mean, and diastolic points. Since many oscillometric devices have not been validated, caution must be given as most are not suitable in clinical and acute care settings.

White-coat method: For some patients, BP measurements taken in a doctor's office may not correctly characterize their typical BP. In up to 25% of patients, the office measurement is

higher than their typical BP. This type of error is called white-coat hypertension (WCH) and can result from anxiety related to an examination by a health care professional. The misdiagnosis of hypertension for these patients can result in needless and possibly harmful medication. Debate continues regarding the significance of this effect Some reactive patients will react to many other stimuli throughout their daily lives and require treatment. In some cases a lower BP reading is said to occur at the doctor's office.

Home monitoring method: Ambulatory blood pressure devices that take readings every half hour throughout the day and night have been used for identifying and mitigating measurement problems like white-coat hypertension. Except for sleep, home monitoring could be used for these purposes instead of ambulatory blood pressure monitoring. Home monitoring may be used to improve hypertension management and to monitor the effects of lifestyle changes and medication related to BP. Compared to ambulatory blood pressure measurements, home monitoring has been found to be an effective and lower cost alternative. Aside from the white-coat effect, BP readings outside of a clinical setting are usually slightly lower in the majority of people. The studies that looked into the risks from hypertension and the benefits of lowering BP in affected patients were based on readings in a clinical environment. When measuring BP, an accurate reading requires that one should not drink coffee, smoke cigarettes, or engage in strenuous exercise for 30 minutes before taking the reading. A full bladder may have a small effect on BP readings; if the urge to urinate exists, one should do so before the reading. For 5 minutes before the reading, one should sit upright in a chair with one's feet flat on the floor and with limbs uncrossed. The BP cuff should always be against bare skin, as readings taken over a shirt sleeve are less accurate. During the reading, the arm that is used should be relaxed and kept at heart level, for example by resting it on a table. Since BP varies throughout the day,

measurements intended to monitor changes over longer time frames should be taken at the same time of day to ensure that the readings are comparable. Suitable times are:

- immediately after awakening (before washing/dressing and taking breakfast/drink), while the body is still resting,
- immediately after finishing work.

Automatic self-contained BP monitors are available at reasonable prices, some of which are capable of Korotkoff's measurement in addition to oscillometric methods, enabling irregular heartbeat patients to accurately measure their blood pressure at home.

Invasive method: Arterial blood pressure is most accurately measured invasively through an arterial line. Invasive arterial pressure measurement with intravascular cannulae involves direct measurement of arterial pressure by placing a cannula needle in an artery (usually radial, femoral, dorsalis pedis or brachial). The cannula must be connected to a sterile, fluid-filled system, which is connected to an electronic pressure transducer. The advantage of this system is that pressure is constantly monitored beat-by-beat, and a waveform (a graph of pressure against time) can be displayed. This invasive technique is regularly employed in human and veterinary intensive care medicine, anesthesiology, and for research purposes. Cannulation for invasive vascular pressure monitoring is infrequently associated with complications such as thrombosis, infection, and bleeding. Patients with invasive arterial monitoring require very close supervision, as there is a danger of severe bleeding if the line becomes disconnected. It is generally reserved for patients where rapid variations in arterial pressure are anticipated. Invasive vascular pressure monitors are pressure monitoring systems designed to acquire pressure information for display and processing. There are a variety of invasive vascular pressure monitors for trauma, critical

care, and operating room applications. These include single pressure, dual pressure, and multi-parameter (i.e. pressure / temperature). The monitors can be used for measurement and follow-up of arterial, central venous, pulmonary arterial, left atrial, right atrial, femoral arterial, umbilical venous, umbilical arterial, and intracranial pressures.

2.5. 1 Mean arterial pressure

The mean arterial pressure (MAP) is the most important of the two pressures (systolic and diastolic) because it is the average pressure driving blood into the tissues throughout the cardiac cycle. A higher pressure is dependent upon vascular resistance which may be caused by many factors for example hardening of vessels as in atherosclerosis, vessels becoming more resistant to expansion as blood enters them. They may be stiffer or less distensible. Arterial pressures are important in the assessment of CVD factors. MAP is equal to cardiac output multiplied by total peripheral resistance. That the volume of blood pumped into the arteries per unit of time (cardiac output) is one of the two direct determinants of MAP, should not come as a surprise and that the resistance of blood vessels to flow (total peripheral resistance) is the other determinant, (Vander et al, 1986). It should be emphasized that, it is the total arteriolar resistance which influences arterial blood pressure. When the skeletal muscle arterioles dilate during exercise, the total resistance of all vascular beds could still be maintained if arterioles were to constrict in other organs (the brain remains the same). Due to difference in the pressure between pulmonary and systemic circulations, the mean arterial pressure becomes very important that is MAP = *pulse pressure* + *diastolic* divided by 3 (pressure throughout the cardiac cycle).

Pulse Pressure = systolic pressure – diastolic pressure (average 50 mmHg)

It is important to have the MAP because arterial pressure is not the same throughout the vascular system. The pressure falls very slightly in the large and medium-sized arteries because their resistance to flow is small, but it falls rapidly in the small arteries and arterioles, which are the main sites of the peripheral resistance against which the heart pumps. The mean pressure at the end of the arterioles is 30-38 mm Hg. It declines rapidly to about 5 mm Hg at the end of the arterioles. The magnitude of the pressure drop along the arterioles varies considerably, depending on whether they are constricted or dilated (Ganong, 2005)

Complications of persistent high blood pressure: Arterial hypertension can be an indicator of other problems and may have long-term adverse effects. Sometimes it can be an acute problem, for example hypertensive emergency. All levels of arterial pressure put mechanical stress on the arterial walls. Higher pressures increase heart workload and progression of unhealthy tissue growth (atheroma) that develops within the walls of arteries. The higher the pressure, the more stress that is present and the more atheroma tend to progress and the heart muscle tends to thicken, enlarge and become weaker over time. Persistent hypertension is one of the risk factors for strokes, heart attacks, heart failure and arterial aneurysms, and is the leading cause of chronic renal failure. Even moderate elevation of arterial pressure leads to shortened life expectancy. At severely high pressures, mean arterial pressures of 50% or more above average, a person can expect to live no more than a few years unless appropriately treated (Guyton and Hall, 2006). In the past, most attention was paid to diastolic pressure; but nowadays it is recognized that both high systolic pressure and high pulse pressure (the numerical difference between systolic and diastolic pressures) are also risk factors. In some cases, it appears that a decrease in excessive diastolic pressure can actually increase risk, due probably to the increased difference between

systolic and diastolic pressures. For those with heart valve regurgitation, a change in its severity may be associated with a change in diastolic pressure. In a study of people with heart valve regurgitation that compared measurements 2 weeks apart for each person, there was an increased severity of aortic and mitral regurgitation when diastolic blood pressure increased, whereas when diastolic blood pressure decreased, there was a decreased severity.

Low pressure: Blood pressure that is too low is known as hypotension . Hypotension is a medical concern only if it causes signs or symptoms, such as dizziness, fainting, or in extreme cases, shock (NHLI, 2008).

When arterial pressure and blood flow decrease beyond a certain point, the perfusion of the brain becomes critically decreased (that is the blood supply is not sufficient), causing lightheadedness, dizziness, weakness or fainting. Sometimes the arterial pressure drops significantly when a patient stands up from sitting. This is known as orthostatic hypotension (postural hypotension); gravity reduces the rate of blood return from the body veins below the heart back to the heart, thus reducing stroke volume and cardiac output. When people are healthy, the veins below their heart quickly constrict and the heart rate increases to minimize and compensate for the gravity effect. This is carried out involuntarily by the autonomic nervous system. The system usually requires a few seconds to fully adjust, and if the compensations are too slow or inadequate, the individual will suffer reduced blood flow to the brain, dizziness and potential blackout. Increases in G-loading, such as routinely experienced by aerobatic or combat pilots 'pulling Gs', greatly increases this effect. Repositioning the body perpendicular to gravity largely eliminates the problem.

2.6 Hypertension and Exercise

Hypertension is a widespread health problem effecting nearly 25% of the adult population in the United States (Fang et al, 2005). The importance of treating this "silent killer" lies in its associated risk to cardiovascular disease, the number one cause of death in America, as well as other maladies including renal disease, stroke, heart failure, and peripheral artery disease. Although hypertension is defined as systolic blood pressure (SBP) equal to and greater than 140 and/or diastolic blood pressure (DBP) equal to and greater than 90 mmHg, risk factors of hypertension can be seen in blood pressure (BP) as low as 115/75 mmHg and will begin to double in risk for every 20/10 mmHg increase (Pescatello, et al, 2004). The new classification of "pre-hypertensive" (SBP 120-139 and DBP 80-89 mmHg) has been introduced to identify individuals who are at a higher risk of developing hypertension, pointing out an important fact that hypertension is a modifiable risk factor. Exercise as a lifestyle modification is beneficial to a wide variety of health conditions. Specific to hypertension, the benefits of exercise have been promoted by a number of organizations and agencies including the American Heart Association, the American College of Sports Medicine, the Surgeon General of the United States, The National Institutes of Health, and the Centers for Disease Control (Wallace, 2003). Although hypertension studies are ongoing, there is an ample amount of research support that provides clear evidence of the positive effects of exercise on lowering persons with hypertension.

Exercise Intensity and Duration: Most exercise prescriptions for hypertensive patients include a duration of 20-60 minutes of aerobic exercise at 40-70% VO2max performed 3-5 times a week (Wallace, 2003). Several studies confirm the results of low to moderate training being just as efficient in lowering BP compared to high intensity (>70% VO2max) cardiovascular exercise (Halbert et al, 1997). The fitness level of the individual may also play a central role in

determining optimal intensity. A recent study of 49 middle-aged men with high-normal (SBP=130-139 mmHg; DBP=85-89 mmHg) to Stage 1 (SBP=140-159 mmHg; DBP=90-99 mmHg) hypertension, randomly assigned to a light (40% VO2max) or moderate (60% VO2max) exercise protocol, found reductions in BP from the lower intensity were more prevalent in the older, less fit male subjects; whereas the moderate intensity exercise was more effective in the physically fit men (Pescatello, et al, 2004). Although more research is needed to better specify an optimal exercise intensity to reduce BP, the general guidelines of moderate intensity exercise performed for 30 minutes or more on most days of the week are appropriate for successfully lowering elevated blood pressure levels, and can be readily implemented for many populations of men and women. Moderate exercise programs are also maintained easier and impart less musculoskeletal injury for previously sedentary populations, who are not accustomed to vigorous physical exertion.

Exercise Mode: There is a significant amount of evidence that aerobic training helps to reduce BP. In a meta-analysis (a statistical technique that combines the results of several studies) of 54 clinical trials, findings (in hypertensive men and women) included a reduction in SBP by an average of 3.84 mmHg and 2.58 mmHg for DBP (Whelton et al, 2002). In addition to aerobic exercise, many people are now enthusiastically participating in resistance exercise. Although there is relatively little research on blood pressure and resistance exercise as compared to aerobic training/blood pressure studies, one recent meta-analysis found a decrease of 3.2 mmHg and 3.5 mmHg for systolic and diastolic BP, respectively (Cornelissen and Fagard, 2005). Yet there is debate on the BP benefits of resistance training, as an association has been shown between vigorous resistance training and reduced arterial compliance (meaning the arteries stiffen, and do not expand as well to increased blood flow) (Miyachi et al, 2003). The reduction in arterial

compliance can lead to an increase in systolic BP. However, it has also been shown that aerobic training performed in conjunction with resistance training negated the decrease in arterial compliance (Kawano et al, 2006). This suggests that resistance training be accompanied with aerobic training as a hypertension intervention strategy, which is in agreement with the American College of Sports Medicine recommendations for the prevention, treatment, and control of hypertension (Pescatello, et al, 2004). Aside from traditional aerobic and resistance exercise modalities, a recent Chinese study found that qigong (a series of relaxation, breathing, gentle movement, and walking exercises) also resulted in the reduction of both systolic and diastolic BP (Cheung et al, 2005). This is a notable finding that will hopefully lead to further research in alternative exercise methods that may be beneficial in reducing BP.

Reducing Blood Pressure: Overweight and obesity are associated with many cardiovascular risks including hypertension. Exercise has become a staple in weight management and weight loss programmes, but is mostly effective when concurrently implemented with diet modifications. Specific dietary guidelines that have been shown to lower hypertension are explained in the Dietary Approaches to Stop Hypertension (DASH) diet, which recommends a diet rich in fruits, vegetables and low-fat dairy foods as well as restrictions in alcohol and sodium intake (Bacon et al, 2004). A recent review of lifestyle interventions found that these guidelines as well as fish oil supplements resulted in significant reduction in BP (Dickinson et al, 2006). Although it has been suggested through other studies that potassium, magnesium, and calcium supplementation play a role in BP reduction, this review failed to find strong clinical support for these claims.

There remains controversy over the combined effect of exercise and weight reduction in reducing BP. Both have been shown to help independently, yet a comprehensive review by

Hagberg et al (2000) concluded from the gathered evidence that exercise training and dietary weight loss are independent, and that exercise can be effective in lowering BP without concomitant reductions in body weight. Other reviews maintain this idea but also reinforce the recommendation of the combination of diet and exercise as effective means to facilitate weight loss (Bacon et al, 2004). Therefore, if weight loss is desired in addition with BP pressure reductions, then diet should be modified accordingly.

2.6.1 Exercise in the Prevention of Hypertension:

The encouragement of regular exercise is not only useful as a treatment method for individuals with hypertension, but should also be advocated as a means for prevention. Predictors that may be examined to evaluate the risk of developing hypertension include resting BP, family history, and physical activity levels. Higher physical activity levels have shown an inverse relationship to the development of hypertension. As well, Barengo et al (2005) presented evidence that European men doing equal to and greater than 4 hours per week of leisure-time physical activities (e.g. recreational sports, skiing, gymnastics, heavy gardening, hunting, fishing and walking/jogging) had reduced risk of hypertension. Thus physical activity and regular exercise can protect against hypertension.

Effect of Exercise: The responses to acute bouts of exercises are the physiological changes that occur within only a few exercise sessions, whereas the chronic adaptations are derived from the accumulation of several continuous exercise bouts over a period of time. The underlying mechanisms relating to anti-hypertensive benefits are not completely understood, however several studies have shown connections to specific physiological mechanisms. An immediate (acute) reduction in BP following exercise has been termed ‘post-exercise hypotension’ and is

agreed to be caused by reductions in vascular resistance (the resistance to flow that must be overcome to push blood through the circulatory system) (Hamer, 2006). The mechanisms associated with the chronic adaptations to blood pressure are more complex. A recent meta-analysis supports this chronic role being partially explained by a decreased systemic vascular resistance in which the autonomic nervous system and renin-angiotensin system (a hormone system that helps normalize long-term blood pressure and blood volume in the body) are most likely the underlying regulatory mechanisms (Cornelissen and Fagard, 2005). Another factor contributing to this decrease in vascular resistance is the increase of nitric oxide production (from different sites in the body) causing a vasodilation (increase in the internal diameter of a blood vessel that results from relaxation of smooth muscle within the wall of the vessel) in response to regular aerobic exercise.

As hypertension is associated with an increase in risk for cardiovascular disease, it is vital that effective interventions are advocated to reduce overall morbidity and mortality. Although pharmacological treatments can be costly and necessary for treating some BP conditions, lifestyle modifications should also be implemented whenever possible. The role of exercise has been shown to be consequential in lowering both systolic and diastolic BP. Both aerobic and resistance training have been shown to facilitate anti-hypertensive responses, although aerobic exercise has been more largely studied. Specifics concerning optimal intensity and length of the exercise program are yet to be fully determined, however, moderate intensity exercise performed for at least 30 minutes on most days of the week still remains to be the 'minimal' yet effective recommendation necessary for prevention and treatment of hypertension, as well as for promoting overall health.

2.7. Exercise and Cardiovascular Disease Risk Factors

Exercise is a type of physical activity which is planned, structured and repetitive bodily movement done to improve or maintain one or more components of physical fitness.

Physical activity has been known to provide protection against the development of cardiovascular disease and also favourable in modification of many CVD risk factors including high blood pressure, blood lipid level, insulin resistance and obesity (NIH, 1995) Cardiovascular disease conditions do not just happen but are highly associated with certain conditions which are generally referred to as risk factors. A risk factor is defined as the ground which sufficiently makes the cause possible.

Risk factors: In order to appreciate how exercise help in reducing, preventing or managing CVD risk factors, a chronicle of all known risk factors need to be identified. Nieman (2003), Fox and Mathew (1981) and McArdle et al (2010), identified the following as risk factors for cardiovascular disease. Age, gender, family history, smoking cigarette, high total cholesterol, high triglycerides, high LDL, low HDL, high blood pressure, obesity, diabetes, stress, high consumption of alcohol and physical inactivity.

While, gender, age, family history and race are considered as those that cannot be controlled easily, others are subject to change depending on lifestyle activities.

The prevalence of some factors has been recognized by Happenen et al (1997) Grandy et al (1999) and Vankerswarlu (2010) as follows among people:

i. Cigarette smoking 25%;
ii. High blood pressure 25% (140/90 mmHg)
iii. High blood cholesterol 20% (above 240 mg/dl.)
iv. Physical inactivity 60%
v. Age, heredity, gender or family history 13%

vi. Diabetes 7%

vii. Obesity 33% and

viii. Stress

Cardiovascular disease risk factors predispose persons in different ways

i. Age: as people advance in age so also their cardiovascular fitness goes low especially above 45 years for men and 55 years for women. Losing their physiological vitalities and the increase in physical inactivity, proneness to cardiovascular disease increases too.

ii. Gender differential: Women have greater body fat percent than men 20-25% and 13-15% respectively (Venkateswarlu, 2010). Having a higher body fat percent may predispose a person to cardiovascular ill condition.

iii. Family history – Having a record of cardiovascular disease in a family tends to place one at risk of developing CVD. Similarly, coming from a particular race where CVD is prevalent could cause one to develop CVD.

iv. Cigarette smoking – certain elements in cigarette can increase high blood pressure and stress. It injures blood arteries also.

v. Hypertension: This results from other factors but when it is higher than 140 over 90 it is dangerous.

vi. High cholesterol which can lead to artherosclerosis (stroke may occur).

vii. Diabetes mellitus: More sugar in the system causing ombulus formation leading to heart failure.

viii. Physical inactivity: Physical activity has been known to affect positively majority of the risk factors identified. Failure to exercise exposes one to most of these risk factors

developing. Inactivity in combination with poor choice of diet would lead to obesity, diabetes, hypertension, hyperglycerolema, or hyperlipidaemia.

2.7.1 Effects of exercise on CVD risk factors

Modifications in majority of CVD risk factors have been observed following exercise intervention (Pekkanen et al, 1990). Similarly some studies have shown that habitual physical activity helps in reducing CHD risk factors, particularly in relation to TC, TG and lipoprotein concentration (Dowling, 2000 and Tall et al, 2002), consistent involvement in aerobic type of activities sustained over time have also been found to reduce obesity remarkably, (Pekkanen (1990) and Ventakeswarlu 2010), stated conclusively that lipids such as triglycerides, total cholesterol and high density lipoproteins are lower in people who take part in regular exercises. In the same document, Venkateswarlu observed that regular participation in sustained moderate intensity exercise has been found effective in lowering blood pressure.

Regarding risk factors such as age, gender, family history and race, exercise can be used to reduce or prevent the development of cardiovascular diseases among populations. When people advance in age, their physiologic conditions get reduced generally. Exercise with its longevity effect can assist the elderly to reduce the chances of developing coronary heart disease conditions.

2.8 Impact of Physical Activity on Blood Lipids and Lipoproteins

Physical activity beneficially influences most of the atherosclerotic risk factors. The impact of regular exercise on plasma lipids and lipoproteins has been clearly defined with regard to the interactions among lipids, lipoproteins, apolipoproteins (apo), lipoprotein enzymes, and the

influence of various factors such as aging, body fat distribution, dietary composition, and cigarette smoking status.(Durstin and Thomson, 2001). The importance of physical activity, like nutrition, cannot be underestimated. Unfortunately, healthcare providers are generally not well equipped to educate and support patients in the pursuit of a lifetime physical activity programme. Providers are faced with time constraints, by the resistance of many adults to make physical activity a part of their daily routine; and by a lack of knowledge and experience in behavioral change. A collaborative approach to the care of adults with coronary risk factors through the use of nonphysician healthcare providers such as nutritionists, nurses, and exercise physiologists can help improve patients' success in the adoption of regular physical activity. Cardiac rehabilitation programmes can offer assistance to healthcare providers with exercise education and supervision when indicated.

Exercise training studies usually observe lower plasma TG concentrations; (Grandjean et al, 1998). Large plasma TG reductions after exercise training are reported for previously inactive people with higher baseline concentrations, (Kokkinos et al, 1995), although subjects with low initial TG concentrations have smaller TG reductions after exercise training.(Wood et al 1983). Exercise training studies do not support an exercise-induced change in total cholesterol, rather, total cholesterol reductions are associated with body weight, percentage of body fat, and dietary fat reductions.(Durstin and Thompson, 2001). Very-low-density lipoprotein (VLDL) cholesterol are lower after aerobic exercise training, (Durstin and Grandjean, 2002). After completing 6 months of jogging (20 mi/week at 65% to 80% of aerobic capacity), after 8 months of regular exercise participation, and after 3 weeks of diet and brisk walking, subjects exhibited greater LDL particle sizes with lower LDL-C. Cholesterol was decreased in the more-dense LDL subfractions and increased in the less-dense LDL fractions; these changes correlated with TG

reductions; (Beard et al, 1996). Plasma lipoprotein(a) (Lp a), an LDL subfraction containing apo(a), is highly homologous with plasminogen and competes with plasminogen for fibrin-binding sites, inhibiting fibrinolysis. Lp(a) does not change after regular physical activity participation. (Dustin and Thompson, 2001). Exercise training longer than 12 weeks with good adherence is more likely to increase plasma HDL-C in a dose-dependent manner,(Durstin and Grandjean, 2002). Exercise-induced increases in HDL-C range from 4% to 22%, whereas absolute HDL-C increases are more uniform and range from 2 to 8 mg/dL. Findings show that exercise training without altered body weight and/or composition can increase HDL-C, and this is augmented by body fat loss. The impact of exercise training on apolipoproteins has been reviewed such that apolipoprotein (apo) A-I levels are observed, whereas apoB changes after exercise training usually parallel LDL-C changes. ApoE levels in response to exercise appear to be mediated by many factors such as age and phenotype, with phenotype playing a strong role (Velliqette et al, 2000). Exercise training studies provide direct evidence of a possible interactive effect between apoE polymorphism and exercise training lipoprotein/lipid change. In comparison with endurance training, less information exists to support resistance training as a modifier of plasma lipids. Studies are often contradictory, with some showing positive benefits of resistance exercise on the lipid profile, (Behall et al, 2003) and others finding no benefits (Prabhakaran et al, 1999). A decrease in body fat percentage and an increase in lean body mass after resistance training are associated with decreased total cholesterol and LDL-C. Both total cholesterol and LDL-C may be reduced after circuit resistance training. The magnitude of change found for lipid and lipoprotein/lipid concentrations after a single exercise session is similar to that seen after the completion of a longitudinal exercise training programme. A measurable, beneficial effect on circulating lipids and lipoproteins/lipids may be expected after a single exercise session during

which 350 kcal is expended; (Crouse et al, 1997), whereas trained individuals may require ≥ 800 kcal to elicit comparable changes. Lp (a) concentrations were not changed after short-duration exercise or longer-duration exercise sessions that required 1500 kcal of energy expenditure. To maintain beneficial lipid and lipoprotein/lipid changes, exercise must be performed regularly as contemplated above.

High-intensity exercise and high-energy expenditure that causes depletion of intramuscular TG stores are needed to increase muscle LPL synthesis and release; (Seip and Semenkovich, 1998). Current data support a favorable impact for exercise training on lipid and lipoprotein profiles. Because much is known about the mechanisms responsible for changes in plasma lipid and lipoprotein modifications as a result of exercise training, a comprehensive medical management plan can be developed that optimizes pharmacological and lifestyle modifications. Scientific investigations are focusing on the molecular basis for lipid and lipoprotein change as a result of various interventions. Findings from these studies can provide a better understanding of why some people respond to exercise whereas others do not. Information about the interactive effects between regular exercise participation and pharmacological therapy is however lacking.

With its favorable effect on many blood lipid abnormalities, physical activity/exercise training is a most appropriate intervention to the management of abnormal blood lipids. Activity should be undertaken at moderate to high intensity, 5 to 7 days/week, for at least 30 min/day and for ≥60 min/day by people who need to achieve weight loss. If this is done with an appropriate emphasis on nutrition and adherence, then body weight will likely be reduced and the need for medication

2.9 Serum Glucose Homeostasis and Physical Exercise Training

Glucose is a monosaccharide and is a central molecule in carbohydrate metabolism. Glucose is also known as blood sugar, or fasting blood sugar, it is the transportable form of carbohydrate which reaches the cells. During the process known as glycolysis, glucose is converted to lactic acid with the production of energy in the form of Adenosine Triphosphate (ATP) (Williams, 2007).

Glucose is primarily used by tissues such as the brain, erythrocytes (RBC), renal medulla and the bone marrow. At rest a man uses as much as two thirds of blood glucose to keep the brain working while the remaining one third is used for Red Blood Cells and the skeletal muscles.

There are mainly three sources of blood glucose (Satyanarayana and Chakrapani, 2009).

1. Dietary sources - Carbohydrates.
2. Gluconeogenesis - Degradation of muscles glycogen to glucose.
3. Glycogenolysis - Degradation of glycogen in the liver to glucose. (Glycogen metabolism in the liver regulates the blood glucose level).

Blood glucose is very important in the sustenance of cellular activities of the body. As it is constantly being depleted, so also it is consistently being resynthesized thereby maintaining a tolerant level or homeostasis in a healthy state. Serious fluctuations in blood glucose concentration portains danger to the human body.

Normal fasting blood glucose level is 70-99 mg/dl (3.9 – 5.5mmol/L). above this and below indicate hyperglycemia and hypoglycemia respectively. When blood glucose concentration falls to less than 45mg/dl, symptoms of hypoglycaemia begin to appear such as headache, anxiety,

confusion, sweating blurred speech, seizures and coma. This may lead to death if left unchecked. High blood glucose levels are said to be toxic and can cause serious microvascular and macrovascular damages (Rossini, 2004, Keefe and Bells, 2007).

Dangers of high blood sugar levels include:

- Damage to body cells.
- Increased cancer risk.
- Increase heart disease risk.
- Depressed immune system.
- Lead to obesity which may result into diabetes mellitus.

High blood glucose levels are the primary cause of CVD and nerve disorder in people with diabetes.

The concern in this paper is hinged on the fact that higher blood glucose level constitute a metabolic problem such as diabetes mellitus (Disorder of fuel metabolism) which is characterized by hypoglycemia that leads to several long term complications.

Insulin dependent or type I diabetes (Juvenile diabetes) mainly occurs in childhood (12-15yrs). It is characterized by almost total deficiency of insulin due to destruction of β (beta) cells of the pancreases which secret insulin as soon as blood glucose levels are elevated.

The type II or non-insulin dependent diabetes (adult onset) is the most common and accounts for 80-90% of the diabetes population, its occurrence is more with people that are obese.

Diabetes mellitus is associated with several metabolic alterations. Most important among them are hyperglycemia which is primarily due to reduced glucose uptake by tissues and its

increased production via gluconeogenesis and glycogenolysis. Another metabolic alteration is ketoacidosis. This is an increased mobilization of fatty acids resulting in overproduction of kentone bodies.

Hyperglycemia is said to be directly or indirectly associated with several complications such as atherosclerosis, retinopathy, nephropathy and neuropathy. Diet, exercise, drugs and insulin are the options for diabetic control (Satyanarayana and Chakropani, 2009, Laaksoneu and Sen, 2000).

Regular physical exercise has been reported to be effective in the prevention and delay of outset of type 2 diabetes, increases insulin sensitivity and ameliorate glucose metabolism (Deroniah and Boutayeb, 2002). Similarly, Kelly and Goodpaster (2001), stated that, a relatively small number of studies have suggested that exercise acutely lowers hepatic glucose production. At moderate intensity exercise studies of patterns of substrate utilization during exercise appear to indicate that there is substantial similarity in the metabolism of glucose by peripheral tissues in comparing nondiabetic and type 2 diabetic individuals (Colberg et al, 1995).

Exercise is said to be an effective method of maintaining a healthy blood glucose levels. Along with healthy diet, it is the most effective prevention strategy for avoiding diabetes.

In conclusion however, it must be noted that acute effect of exercise training leads to an increase in blood glucose levels to meet the demand of muscles in use. Similarly insulin secretion increases so as to enable cellular reception and ultimate utilization of glucose for energy metabolism. Glucose concentration in the blood may increase and decrease subject to demand for cellular activities.

An increase in plasma glucose will stimulate pancreatic release of insulin for cellular reception of glucose. The use of glucose by the cells causes a fall in blood glucose levels. It is

this mechanism which is of interest to exercise scientists. can exercise cause there negative feedback mechanism to be sustained?

2.10 Interval Training

Interval training is defined in several ways by different writers Fox and Mathews, (1981) described it as a series of repeated bouts of exercise alternated with periods of relief. Similarly, Nieman, (2003), sees interval training as an exercise session in which the intensity and duration of exercise are consciously alternated between harder and easier work. Powers and Howley (2007), defined interval training as the performance of repeated exercise bouts with brief recovery periods in between. In all three definitions, two features are common, namely exercise bouts and a period of recovery. One of the definitions referred to the recovery period as alternating the exercise intensity. In whichever way, interval training is a consciously planned exercise or training session in which bouts are altered with rest periods till the end of the session duration. The opposite of interval training is continuous training. Interval training can be adapted for all forms of training plans.

To properly understand the mechanism of interval training certain terminologies need to be understood (Powers and Howlay, 2007, Fox and Mathew, 1981, Lawsen and Jenkins, 2002). These terms are;

1. Work interval
2. Relief interval
3. Sets
4. Repetitions
5. Training time or training duration
6. Training distance or load and
7. Frequency, how often per week.

- Work interval: description or prescription of the work e.g. performing 200 meters run in 40 seconds.
- Relief or rest interval – This may be a complete rest period without an activity or a light activity such as jogging or walking or a combination of the two.

It is usually expressed in relationship to the work interval (work relief ratio) which may be 1:½, 1:1, 1:2 or 1:3. 1:1 means work and relief time are same while 1:3 means relief time is three times the work interval.

- Longer work interval – 1:½ or 1:1 (low intensity)
- Middle duration 1:2 (medium intensity)
- Shorter duration 1:3 (high intensity).
- Sets: These are series of work and relief intervals such as 200mts runs in

40 seconds interspersed with 40 seconds relief interval.

- Repetitions: This describes the number of work intervals in a set. For example 200 meters x 6 repetitions.
- Training time- Rate at which work is to be performed for example. 200m run in 40 seconds.
- Training distance for example. 200m.
- Frequency: The number of workouts per week e.g. 3 times (Fox and Mathew 1974; Powers and Howley, 2007).

Depending on the purpose of the training, an interval training must be prescribed for any particular requirement. Fox and Mathews, (1981) illustrated an interval training prescription as follows:

Set 1, 6 x 200 at 33 (1:39).

Where 6 is number of repetitions.

200 is training distance in meters.

0.33 is training time in minutes and seconds.

(1.39) is time of relief interval in minutes and seconds.

In an attempt to design or plan an interval training programme, certain variables must be considered (Powers and Howley 2007; Fox and Mathews, 1981). These include rate and distance of work interval.

- Number of repetitions per workout
- Type of activity during relief interval
- Intensity or the effort
- Frequency of training per week.

Advantages of Interval Training: There is precision in control of stress. The systematic day to day approach, enabling one to easily observe progress. One obvious advantage it has over continuous running is that it provides a means of performing large amounts of high-intensity exercise in a short time. It gives more rapid improvement in energy potential than in other conditioning methods. It also has the advantage of being performed everywhere and requires little or no special equipments. This method may be more effective at inducing fat loss than simply running at a moderate intensity level for the same duration. Work should be selected based on the actual needs of the athletes or patient. Interval training prescriptions are made up of Long duration work intervals performed at low intensities, medium duration intervals performed at moderate intensities, and short work efforts performed at higher intensities. It is important in the choice of exercise or work especially for the treatment or prevention of cardiovascular disease to consider foremost, the type, intensity, and quantity. Activities which reduce cardiovascular disease risk factors and confers many other health benefits does not require a

structured or vigorous exercise programme. This implies that the majority of benefits of physical activity can be gained by performing moderate intensity activities (Blair et al, 1995). A moderate level of physical activity confers health benefits, however, it has to be performed regularly to maintain the effects. Moderate intensity activity therefore performed by previously sedentary individuals results in significant improvements in many health related outcomes, and are likely to be sustained as opposed to high intensity activities (Blair et al, 1995). The duration of such activities should be at least 30 minutes per day as minimal standard and at least 3 days in a week. It should be noted that increases in the intensity or amount of activity produces further benefits but high intensity activity is associated with an increased risk of injury, discontinuation of activity or acute cardiac events during the activity. The frequency, intensity, and duration are interrelated. The number of episodes of activity recommended for health depends on the intensity and or duration of the activity could be performed approximately 3 times weekly and achieve cardiovascular benefits, but low intensity or shorter duration activities should be performed more often to achieve cardiovascular benefits (King et al, 1992).

2.11. Summary.

Cardiovascular Diseases include those diseases of the heart and its blood vessels and there are over twenty known diseases of the cardiovascular system (Nieman, 2003). Atherosclerosis, the building up of fatty plague material in the inner layer of the blood vessels is the underlying factor in 85% of CVD. Atherosclerosis develops gradually and in stages. The arterial wall is first injured by factors such as hypertension, high blood cholesterol levels (hypercholesterolaemia) oxidized LDL, cigarette smoking, toxins, viruses and blood flow turbulence (Nieman, 2003). Fat, depending on where it is deposited is largely the main factor that lead to the causes of CVD. Fat is found deposited in different parts of the body called regional depots. Fat distribution

pattern differs between male and female and even between races (Martin and Daniel, 1993). The regional distribution of fat in humans is referred to as fat patterning. Fat pattern may be observed in the upper and lower regions of the body. Bjantrop (1998), observed that increased abdominal or waist accumulation of fat is associated with health risk of hyperlipidaemia, hypertension and coronary heart disease than fat located in the hips and thighs. Fat deposited on the upper abdominal part of the body are also called android obesity and those on the hip as gynoid obesity The determination of fat distribution or patterning is being carried out by the use of measurements (not the total body mass). These measures as discussed somewhere in this review, includes height, weight, circumferences and indexes. The commonly used of these include BMI waist hip ratio (WHR) conicity index (CI) waist circumference, hip circumference and the skinfolds.

The pressure exerted by the pumping action of the heart on the blood vessels is known as blood pressure (systolic and diastolic) The risk of cardiovascular disease increases progressively when blood pressure goes above 115/75 mmHg (Appel, Brands, Daniels, et al, 2006). Clinical trials demonstrate that people who maintain arterial pressures at the low end of these pressure ranges have much better long term cardiovascular health. . At severely high pressures, mean arterial pressures of 50% or more above average, a person can expect to live no more than a few years unless appropriately treated (Guyton and Hall, 2006). In the past, most attention was paid to diastolic pressure; but nowadays it is recognized that both high systolic pressure and high pulse pressure (the numerical difference between systolic and diastolic pressures) are also risk factors. Hypertension is a widespread health problem effecting nearly 25% of the adult population in the United States (Fang et al, 2005) Although hypertension studies are ongoing, there is an ample amount of research support that provides clear evidence of the positive effects of exercise on

lowering persons with hypertension Most exercise prescriptions for hypertensive patients include a duration of 20-60 minutes of aerobic exercise at 40-70% VO2max performed 3-5 times a week (Wallace, 2003). Several studies confirm the results of low to moderate training being just as efficient in lowering BP compared to high intensity (>70% VO2max) cardiovascular exercise (Halbert et al, 1997). The encouragement of regular exercise is not only useful as a treatment method for individuals with hypertension, but should also be advocated as a means for p0revention.

Physical activity has been known to provide protection against the development of cardiovascular disease and also favourable in modification of many CVD risk factors including high blood pressure, blood lipid level, insulin resistance and obesity (NIH, 1995). Physical activity beneficially influences most of the atherosclerotic risk factors. The impact of regular exercise on plasma lipids and lipoproteins has been clearly defined with regard to the interactions among lipids, lipoproteins, apolipoproteins (apo), lipoprotein enzymes, and the influence of various factors such as aging, body fat distribution, dietary composition, and cigarette smoking status.(Durstin and Thomson, 2001). Exercise training studies usually observe lower plasma TG concentrations; (Grandjean et al, 1998). Large plasma TG reductions after exercise training are reported for previously inactive people with higher baseline concentrations, (Kokkinos et al, 1995), although subjects with low initial TG concentrations have smaller TG reductions after exercise training.(Wood et al 1983). Exercise training studies do not support an exercise-induced change in total cholesterol, rather, total cholesterol reductions are associated with body weight, percentage of body fat, and dietary fat reductions.(Durstin and Thompson, 2001). Very-low-density lipoprotein (VLDL) cholesterol are lower after aerobic exercise training, (Durstin and Grandjean, 2002).

High blood glucose levels are the primary cause of CVD and nerve disorder in people with diabetes. Regular physical exercise has been reported to be effective in the prevention and delay of outset of type 2 diabetes, increases insulin sensitivity and ameliorate glucose metabolism (Deroniah and Boutayeb, 2002). Similarly, Kelly and Goodpaster (2001), stated that, a relatively small number of studies have suggested that exercise acutely lowers hepatic glucose production. At moderate intensity exercise studies of patterns of substrate utilization during exercise appear to indicate that there is substantial similarity in the metabolism of glucose by peripheral tissues in comparing nondiabetic and type 2 diabetic individuals (Colberg et al, 1995). Interval training is defined in several ways by different writers Fox and Mathews, (1981) described it as a series of repeated bouts of exercise alternated with periods of relief. Similarly, Nieman, (2003), sees interval training as an exercise session in which the intensity and duration of exercise are consciously alternated between harder and easier work. Powers and Howley (2007), defined interval training as the performance of repeated exercise bouts with brief recovery periods in between. One obvious advantage it has over continuous running is that it provides a means of performing large amounts of high-intensity exercise in a short time. It gives more rapid improvement in energy potential than in other conditioning methods. It also has the advantage of being performed everywhere and requires little or no special equipments. This method may be more effective at inducing fat loss than simply running at a moderate intensity level for the same duration. Activities which reduce cardiovascular disease risk factors and confers many other health benefits does not require a structured or vigorous exercise programme. This implies that the majority of benefits of physical activity can be gained by performing moderate intensity activities (Blair et al, 1995).

CHAPTER THREE:

3.0 RESEARCH METHODOLOGY

3.1 Introduction

The purpose of this study was to investigate the effect of moderate intensity interval training in running on body fat patterns, blood pressure, serum glucose, lipids and lipoproteins of sedentary Nigerian male adults. To achieve this purpose, the research design, population, sample and sampling technique, measurements procedures, instrumentation, training protocol, research assistants, experimental controls and

Statistical techniques are described in this chapter.

3.2 Research Design

The research design used for this study is the 2x3 factorial research design. In this design, there were two groups, one group as experimental group which engaged in training and the other group as a control. These two groups were tested for all the variables investigated three times during the 12 weeks this research lasted. The first test was conducted at week zero (baseline), the second at week six and the third immediately at the termination of training in week 12.

3.3 Population

The population for this study comprised sedentary male Nigerian students of the College of Nursing and Midwifery, Yola, Adamawa State. They were adults between the ages of 20 and 30 years old and in the final year of their study. At the time of this study the College had 37 male students in the final year..

3.4 Sample and Sampling Technique

A random screening of the subjects was carried by the researcher to meet the inclusion criteria. These included:

1. Must have been certified medically healthy by a medical personnel for the programme .
2. Should be a male between the ages of 20 and 30 years.
3. Must not have been engaged in active physical exercise or sports for at least six months preceding this research.
4. Must have baseline weight of not more than 75 kilograms.
5. Should have a height of between 1.60 and 1.85 meters.

Only those who met the inclusion criteria and had given their informed consent for participation were used in this study. The random sampling technique was used to select 30 subjects for this study (Nwana 1982 ,Ojo, 2005, Olayiwola, 2007). The subjects were finally assigned to either experimental (n-15) or control (n-15) group using a simple random statistical technique (Lucky Deep). All odd numbers were assigned to the experimental group while even numbers to the control group.

3.5 Measurement procedures.

The data for this research included the anthropometric, cardiovascular and biochemical measurements. These measurements were taken in week 'zero' (baseline), after the first six weeks of training (week six') and at the termination of training (week 12').

(a) **Anthropometric and body composition measurements-;** All measurements were taken by the researcher and the research assistants using standard procedures as described below (Ross and Martfell-Jones, 1991).

(i) Weight; The instrument used in measuring the weights of the subjects was the Mechanical Personal Scale, Model BR9012 , Capacity 120 kilogrammes, Made in China. Subjects wore minimal clothing made of knickers and top underwear singlet without foot wears. Subjects were made to stand with both feet on the scale and reading was taken two times to the nearest 0.5 kg. the average of the two measurements was recorded accordingly.

(ii) Height; The instrument used was a standard standiometer, manufactured by Iyketony and Partner made in China. It has a platform of 38.5x 28 cm with a height of 190 cm.

Subjects were made to dress in very light cloths and with bare feet. Each was made to stand on the platform erect and facing directly forward with the buttocks and scapulae protrusions having contact with the machine. The overhead rule was adjusted to the highest point of the head of the subject. The subject was allowed to dismount the machine, while the reading was taken in centimeters.

(iii) Hip Circumference; A flexible measuring tape Model S-1542 made in the People's Republic of China was used in measuring hip circumference of subjects. Measurement was taken by circling the tape at the level of the greatest protrusion of the gluteal muscles. Measurements were recorded in centimeters to the nearest 0.1 cm.

(iv) Waist Circumference; A flexible measuring tape Model S-1542 made in the People's Republic of China was used. The tape passed at the minimal abdominal girth

approximately midway between the xiphoid process and the umbilicus. The reading was taken to the nearest 0.1 cm.

(v) Conicity Index: This was calculated as recommended by Valtez (1992)

$$C = \frac{\text{Waist circumference (cm)}}{\text{Weight/height}}$$

(vi). WHR = Waist circumference (cm) ÷ hip circumference. in (cm)

(vii). Body Mass Index (BMI) was calculated as follows:

BMI = Weight (kg) ÷ height $(m)^2$ as recommended by Plowman and Smith, (1997).

b. Biochemical Measurements

Venous blood samples for the determination of serum concentration of TC TG ,LDL-C, HDL-C and glucose were obtained as follows:

- Subjects were made to have an overnight fast for at least 12 hours preceding the bleeding. All blood samples were taken by the research assistants (laboratory technicians from the Chemical Pathology laboratory in a hall provided by the College of Nursing and Midwifery Yola) between the hours of 8.00 am and 10.00 am. Subjects were required to sit on a chair where a tourniquet was tied around the upper left or right hand just above the elbow to briefly arrest circulation to the forearm.
- By clenching the fist, which increased the prominence of the vein, puncturing with a 10.ml syringe and a 5 ml of blood was drawn. The samples were finally transferred into test tubes and labeled fully for each subject. The samples were allowed to coagulate within two hours of the venopuncturing before they were taken for analysis.

Procedure for the determination of serum levels of glucose, lipids and lipoproteins

a. Fasting blood sugar (Glucose)

Glucose was determined after enzymatic oxidation in the presence of glucose oxidase. The hydrogen peroxide formed reacted, under catalysis of peroxidase, with phenol and 4-aminophenazone to form a red-violet quinoneimine dye as indicator.

- Reaction Principles:-

$$\text{Glocuse} + 0_2 + H_20 \xrightarrow{\text{GOD}} \text{Gluconic acid} + H_2\,0_2$$

$$2H_2\,0_2 + 4\ \text{aminophenozone} + \text{phenol} \xrightarrow{\text{POD}} \text{Quinvnecimine} + 4\ H_20$$

- **Test procedure:**

	Test	**Standard**	**Blank**
Glucose reagent kit	1000 µl	1000 µl	1000 µl
Sample	10 µl	-	-
STD	-	10 µl	-

The contents of the testubes were mixed and incubated at 37^0C for 5 minute, then read at 520 nm. The result was recorded by measuring absorbance of the standard and the sample against the reagent black calculation = $^{T}/_{STD}$ x concentration of standard.

b. Triglyceride

- Principle:- (Levy, 1972)

$$TG + H_20 \xrightarrow{\text{LIPOR}} \text{Glycerol} + \text{fatty acids}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{GK}} \text{Glycerol-3-phosphate} + \text{ADP}$$

$$\text{Glycerol-3-phosphate} + 0_2 \xrightarrow{\text{GPO}} \text{dihydoxyacetone} + \text{phosphate} + H_20_2$$

$2H_20_2$ + 4- aminophenozone + 4 chlorophenol $\xrightarrow{POD}$ Quinoneimine + Hcl + $4H_20$

- Procedure – pipette into testubes as follows.

	Test	Standard	Blank
Sample	10µl	_	_
Standard	_	10 µl	_
Reagent	1000 µl	1000 µl	1000 µl

- Mix and incubate for 5 min at 37^0C or at 20-25^0C for 10 min. Read by measuring the absorbance of the sample and standard against the blank within 60 min.

- Calculation $^T/_5$ x concentration of the standard.

c. Total cholesterol

Blood samples were allowed to stand and clot before they were centrifuged at 2,500 rpm and processed after the separation of the serum and used within 4 hours after collection.

- **Principles:-** Cholesterol was determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4- aminoantipyrine in the presence of phenol and peroxidase.

Cholesterol ester + H_20 $\xrightarrow[\text{esterase}]{\text{Cholesterol}}$ + Fatty acid.

Cholesterol = 0_2 Cholesterol $\xrightarrow[\text{Oxidase}]{}$ – 3- one + H_20_2.

Procedure:- pipette as follows.

	Test	Standard	Blank
Cholesterol reagent kit randox	1000 µl	1000 µl	1000 µl
Sample	10 µl	_	_
Standard	_	10 µl	_

Mixture were incubated for 5mins at 37^0C and results were read within 60mins at 520 nm wave length. Read absorbance of sample and standard against the reagent black calculate = $^T/_{STD}$ x concentration of STD.

d. High-density lipoprotein:

Principle:- The LDL,VLDL and chylomicron fractions were precipitated quantitatively by the addition of phosphotungstic acid in the presence of magnesium iron. After centrifugation, the cholesterol concentration in the HDL fraction remaining in the supernatant was determined.

Procedure:- Pipette into centrifuging tubes as follows.

Step I.

	Test	**Standard**	**Blank**
Sample	2000 µl	_	_
Standard	_	200 µl	_
Reagent kit	500 µl	500 µl	_

After mixing, centrifuging was done at 2,500 rpm after which it was allowed to stand for 10 min at room temperature and the supernatant was ready for use in the analysis.

Step II Pipette into tubes as follows.

	Test	**Standard**	**Blank**
DDH_20	_	_	100µl
STD Supernatant	_	100 µl	_
Sample supernatant	100 µl	_	_
Chol. Reagent	1000 µl	1000 µl	1000 µl

Above tubes were properly mixed and incubated for 5 mins at 37^0C. Rreading of result after incubation was done at 520 wave length.

$$HDL = \frac{\text{sample}}{\text{standard}} \times \text{concentration of standard.}$$

e. Low-Density Lipoprotein Cholesterol

$$LDL = TC - \frac{TG}{5} - HDL$$ (Freiedwald et al, 1997)

Where: TC (Total cholesterol)

TG (Triglyceride)

HDL (High density Lipoprotein).

b. Measurement of cardiovascular variables

i. Systolic and Diastolic blood pressure:

Although there are many methods that can be used to measure blood pressure, the auscultatory method was chosen for this research. This method which requires the use of a stethoscope and sphygmomanometer has a dual advantage of being simple and is commonly used in many health -related fields. Instruments used for this measurement was the KA-112 Anaeroid Sphygmomanometer and Dual Head Stethoscope Model S-223 manufactured by Medicare Instruments WUXD (LTD). No.301 Xixin Road Zhangjing, India.

Subjects were required to sit on a chair close to a table whose height was of the same vertical height as the heart. With the left arm on the table, a cuff was placed around the upper arm and attached to the aneroid manometer. The cuff fitted snugly and inflated manually by repeatedly squeezing a rubber bulb until the artery was completely occluded. With the help of

the stethoscope, the tester listens to the brachial artery at the elbow. The tester slowly released the pressure on the cuff. When the occluded blood started to flow in the artery, the turbulent flow created a "whooshing" or pounding sound. The pressure at which this sound was first heard was the systolic blood pressure. The cuff will further be released until no sound was heard and here, the diastolic pressure is read and recorded accordingly for each subject.

ii. Mean Arterial Pressure

The MAP is the most important than systolic and diastolic pressures. It is the average pressure driving blood into the tissues throughout the cardiac cycle. MAP was calculated as systolic pressure minus diastolic pressure plus diastolic pressure divided by three. Pulse pressure + diastolic pressure /3

iii. Resting pulse rate

Subjects were made to rest by lying on couches for between 15 to 30 minutes on arrival at the testing room. This was done to calm down nerves and anxiety. With the use of a stethoscope placed on the left hand side of the sternum (5 th intercostal space), (Guyton, (1991), the two heart sounds were heard for 60 seconds and recorded as pulse rate per minute.

iv. Double Product (DP)

The measure of the work load of the heart (DP) was determined as systolic blood pressure multiplied by heart rate.

3.6. Instrumentation

The following instruments were used in this study for data collection.

a. Measuring tapes : a flexible non elastic tape Model S-1542 made in China was used in the measurement of distances on the running course in meters, hip and waist circumferences in centimeters (Plowman and Smith, 1977).

b. Standiometer: the Holtain Standiometer model NJ07072 manufactured by Pfister Import-Export Inc. U.S.A. This was used in the measurement of height of subjects

c. Weight Scale: A portable bathroom type Hanson scale, model B180A made in Ireland, was used in measuring the weight of subjects standing bare feet and with minimum clothing in kilograms.

d. Needle and syringe: Disposable needles and syringes along with tourniquet were used in bleeding subjects.

e. Stop watches. SEWAN Professional Stopwatch Shenzhensmarttimes Digital Technology Company, China. They were used in timing the training duration and the rest interval as well as the pulse count.

f. Sphygmomanometer K-112 Aneroid type and the Dual Head stethoscope Model No S-223, manufactured in India, was used. for the measurement of blood pressure.

g. Pulse Rate monitor. Omron RX-M Wrist blood pressure and pulse monitor from Omron Healthcare, Europe B.V. Wigalaan.OZ05315L.

3.7 Training Protocol

The duration of training for this research was 12 weeks and held for four days in every week as detailed below.

1. **Training method**:- interval training.

2. **Exercise mode**:- Running.

3. **Intensity:-** Moderate.

Schedule I. Weeks 1-4.

Workload	Repetitions	Rest interval	Frequency
100 meters	5	$1^1/_2$ -2 minutes	2 per week
200 meters	5	$1^1/_2$ -2 minutes	2per week

Schedule II. Weeks. 5-8

work load	repetition	Rest interval	Frequency
200 meters	5	$1^1/_2$ -2minutes	2 per week
300 meters	5	$1^1/_2$ -2minutes	2 per week

Schedule III. Week 9-12

work load	repetition	Rest interval	Frequency
300 meters	5	$1^1/_2$ -2minutes	2 per week
400 meters	5	$1^1/_2$ -2minutes	2per week

The workload in each schedule was increased so as to allow the subjects to adapt to the stress of training. For example, in schedule I, subjects run 100 meters on Monday and 200 meters on Tuesday. 100meters was repeated on Thursday and 200 meters on Friday.

The same alternation was followed in schedule II and III. The intensity of the training was carefully monitored with wrist digital monitors and brachial pulse counting. At the beginning of each training session, 10minute were allowed for warm up and 5 minutes at the end of each training session for warming down.

3.8 Research Assistants

Three research assistants were drawn from the Federal Medical Centre biochemical laboratory and the College of Nursing and Midwifery (final year nursing students). These, apart from being familiar with the testing procedures and the instruments, were further trained particularly on the methodology adopted for this research. The medical laboratory scientists were scheduled to take the blood samples and carry out the analysis in the laboratory while the student nurses assisted in taking the measurements of the circumferences, weight, heights, blood pressure and pulse rates. Similarly, two training assistants were recruited from the Adamawa State Sports Council, Yola to assist in monitoring the training sessions.

3.9 Experimental Controls

The following experimental controls were observed to eliminate the influence of extraneous and intervening variables on the test results.

1. Subjects were requested to strictly avoid being involved in any form of physical activities like playing games that would be interpreted as active exercising, throughout the duration of this experiment. The control group was also to observe these restrictions.
2. Efforts were made to ensure that all the two groups feed on similar diets. All the students in the chosen college feed on similar traditional northern Nigeria diets (corn flour, rice, gari, beef/fish and beans).
3. Routine sessions were organized to discuss the programme and challenges faced by the subjects. During such sessions, effort was made to encourage participants to realize the importance of exercise in their daily activities and their health as well as the research on hand.

4. Movements out of the college especially during weekends were only permitted when they were absolutely necessary.

3.10 Statistical Techniques

The data collected were analyzed using the statistical package of the social science (SPSS) at the Iya Abubakar Computer Centre of the Ahmadu Bello University Zaria. The descriptive statistics of the mean and standard deviation were used for the purpose of interpretation. The t-test was employed to determine the difference between the experimental and the control groups, while the comparison of the difference between trials (0, 6 and 12) were made using repeated-measure ANOVA and the Scheffe's post hoc tests at an alpha level of 0.05.

CHAPTER FOUR

4.0. RESULTS AND DISCUSSION

4.1. Introduction

The purpose of this study was to find out the effects of 12 week moderate intensity interval training in running on body fat patterns, blood pressure, serum glucose, lipids and lipoproteins of sedentary Nigerian male adults. To achieve this purpose, the data collected were statistically analyzed, the results of which are presented and discussed according to the hypothesis in this chapter.

4.2 Results

4.2A Information regarding the physical characteristics of the subjects is presented in table 1.

Table 1: Physical Characteristics of Subjects

Variable	Group	n.	M	SD	SE
Age (yrs)	Experimental	15	23.33	2.19	0.56
	Control	15	25.00	2.42	0.62
Height (m)	Experimental	15	1.71	0.06	0.016
	Control	15	1.69	0.06	0.015
Body weight	Experimental	15	61.5	6.12	1.58
(kg)	Control	15	63.3	7.30	1.88

Table 1 shows there were 30 subjects made up of 15 in experimental group and 15 in control group. The mean age of the experimental group was 23.33 years and that of the control

group was 25 years. Both the experimental and control groups were similar in height 1.71 m and 1.69 m and body weight of 61.5 kg and 63.3 kg, respectively.

4.2B Descriptive statistics of experimental and control groups regarding the anthropometric, body composition and body fat pattern variables in pre-training, mid-training and post-training periods are presented in table 2 as follows:

Table 2: Descriptive Statistics of Test Results of Subjects in Anthropometric and Body Fat Pattern Variables

Variables	Group	N	WEEKS								
			0 (T1)			6 (T_2)			12 (T_3)		
			M	SD	SE	M	SD	SE	M	SD	SE
Weight	Expt	15	61.46	6.11	1.57	61.26	5.36	1.38	62.86	5.50	1.42
(kg)	Contr	15	63.26	7.29	1.88	62.73	6.31	1.63	62.8	7.98	2.06
Waist cir	Exp	15	73.40	3.15	0.81	74.33	3.51	0.90	72.4	3.09	0.79
(cm)	Contr	15	75.40	4.50	1.16	75.26	4.74	1.22	75.6	4.91	1.26
Hip cir	Exp	15	89.06	3.21	0.83	86.53	4.64	1.19	86.13	3.56	0.91
(cm)	Contr	15	87.26	4.72	1.22	86.4	4.89	1.26	86.93	4.52	1.16
BM 1	Exp	15	21.00	2.13	0.55	20.86	2.09	0.54	21.26	1.86	0.48
	Contr	15	21.53	2.16	0.55	21.53	2.03	0.52	21.26	2.4	0.62
WHR	Exp	15	0.82	0.04	0.01	0.86	0.08	0.02	0.84	0.05	0.01
	Contr	15	0.86	0.05	0.01	0.86	0.05	0.01	0.86	0.04	0.01
CI	Exp	15	19.00	1.36	0.35	19.00	1.36	0.35	18.46	1.55	0.400
	Contr	15	18.86	1.50	0.38	19.26	1.62	0.41	19.26	1.38	0.35

Fig. 1: Changes in weights of experimental and control groups in pre, mid and post training periods.

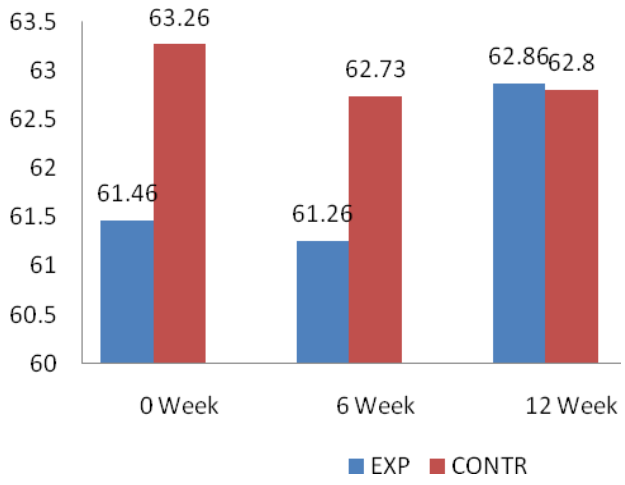


Fig. 2: Changes in waist circumference of experimental and control groups at pre, mid and post training periods.

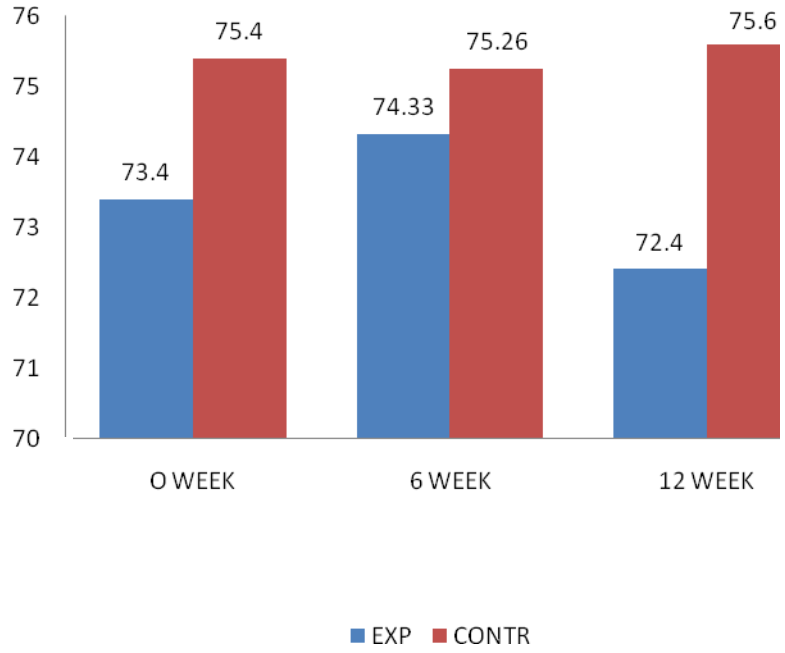


Fig. 3 Changes in hip circumference of experimental and control groups at pre- mid and post training periods.

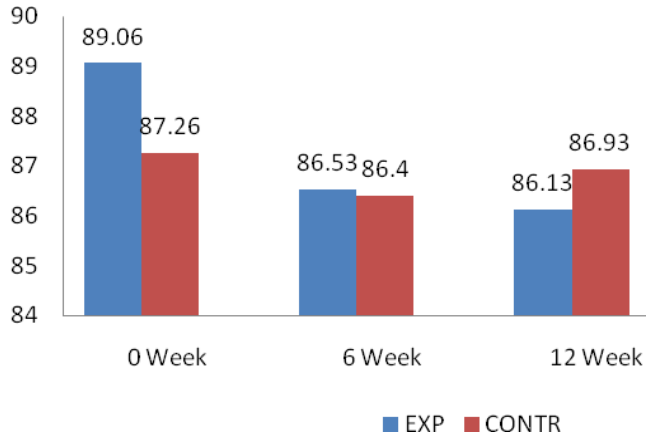


Fig. 4: Changes in body mass index of experimental and control groups at pre- mid and post training periods.

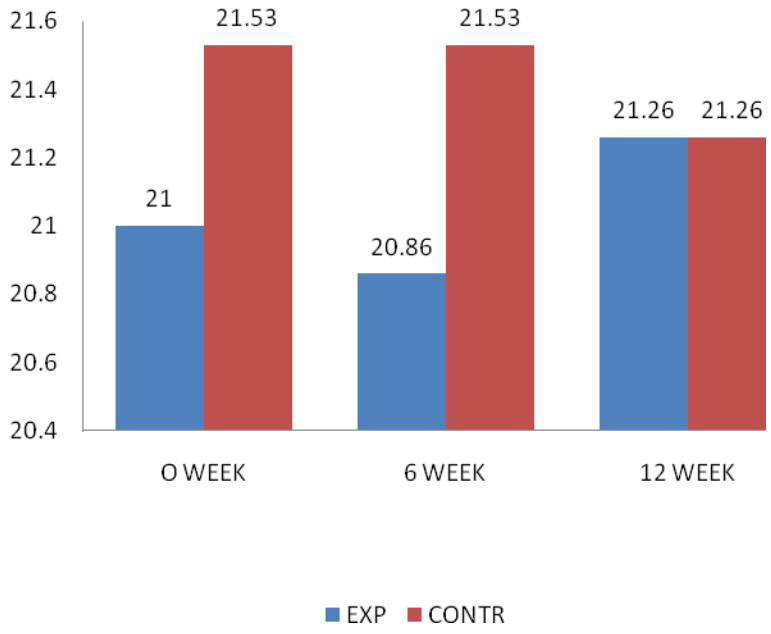
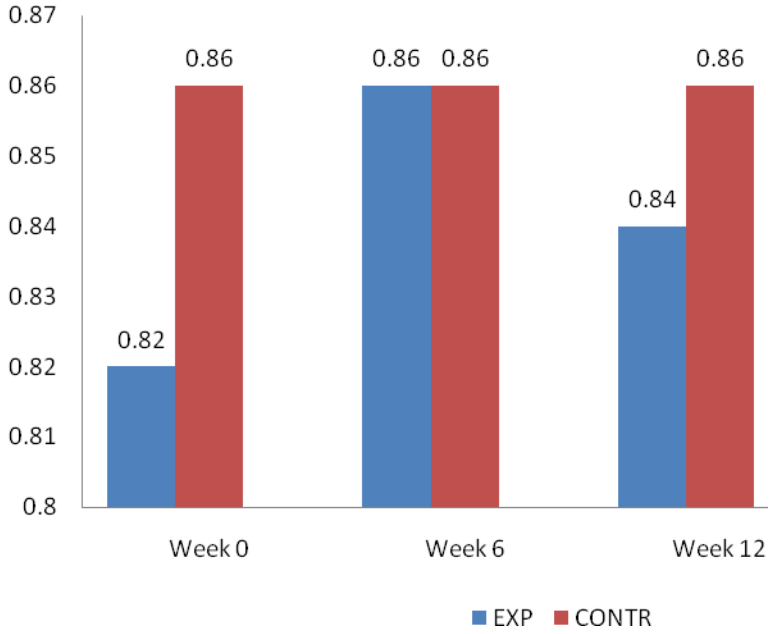
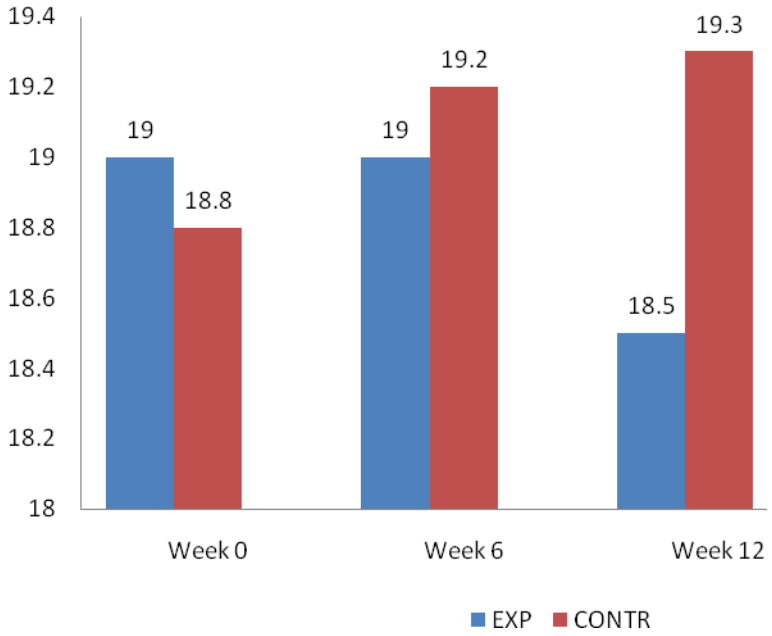


Fig. 5: Changes in waist to hip ratio of experimental and control groups at pre, mid and post training periods.



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Fig.6: Changes in concity index of experimental and control groups at pre, mid and post training periods.



Examination of Table 2 shows that the mean body weight of the experimental group increased slightly from 61.46 ± 6.11kg before starting the training to 62.86 ± 5.50 kg at the end of 12 weeks training. Similarly, the mean body weight of the control group changed very minimally from 62.62 ± 7.29 kg to 62.8kg at the end of week 12. Further observation of Table 2 shows that the changes in hip circumference, body mass index, waist to hip ratio (WHR) and conicity index (CI) from pre- training to post- training levels were very minimal. However, the mean waist circumference of the experimental group changed slightly from 89.06 ± 3.21 cm to 86.13 ± 3.56 cm, which was more than what has been observed in other variables. The change from pre-training level to post- training level in this variable of the control group was not as much as that of the experimental group. These changes are graphically shown in Figure, 4-9.

4.2C Information regarding the changes in biochemical variables of glucose, total cholesterol, triglyceride, high density lipoprotein cholesterol and low density lipoprotein cholesterol during weeks '0' 6 and 12 is shown in Table 3..

Table 3: Descriptive Statistics of Test Results of Subjects in Biochemical Variables.

Variables	Group	N	Weeks								
			0 (T1)			6 (T_2)			12 (T_3)		
			M	SD	SE	M	SD	SE	M	SD	SE
Glucose	Expt	15	93.08	4.85	1.25	83.36	5.40	1.39	81.5	5.7	1.47
(mg/lt)	Contr	15	91.80	7.40	1.91	90.68	6.20	1.60	76.06	7.14	1.84
T.C.	Exp	15	130.23	20.3	5.26	129.1	33.11	8.55	128.5	19.87	8.51
(mg/lt)	Contr	15	140.97	28.9	7.46	135.7	36.28	9.35	136.2	32.99	8.01
TG	Exp	15	58.59	23.29	6.01	78.9	15.4	3.98	57.3	15.65	4.04
(mg/lt)	Contr	15	42.29	9.57	2.47	86.9	32.4	8.38	75.9	28.03	7.23
HDL-C	Exp	15	29.45	6.83	1.76	45.2	17.77	4.58	42.39	6.51	1.68
(mg/lt)	Contr	15	30.14	4.65	1.20	52.8	25.53	6.59	41.22	7.08	1.82
LDL-C	Exp	15	14.12	3.18	0.82	10.45	7.05	1.82	14.45	4.71	1.21
(mg/lt)	Contr	15	19.82	6.15	1.58	6.87	4.23	1.09	12.99	5.27	1.36

Fig. 7: Changes in total chotesterol levels of experimental and control groups at pre, mid and post training periods.

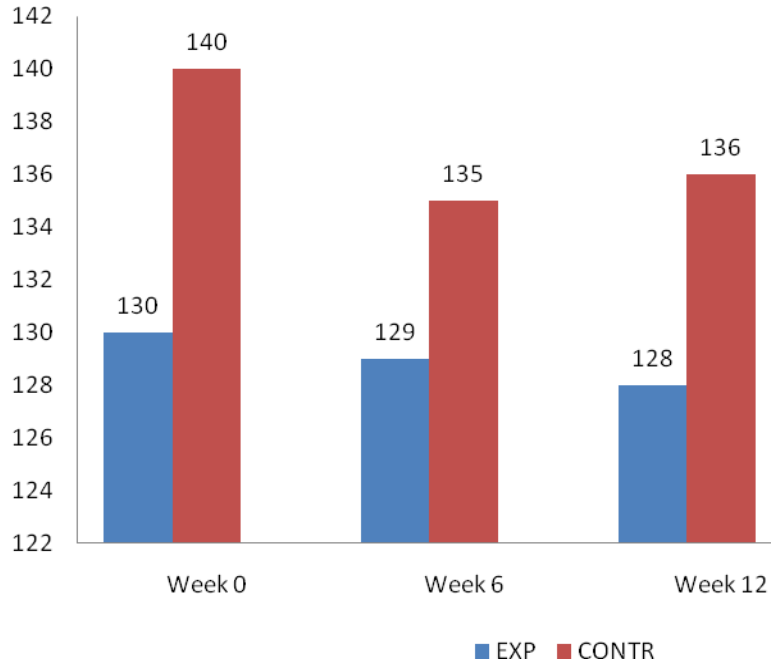


Fig. 8: Changes in triglyceride levels of experimental and control groups at pre, mid and post training periods.

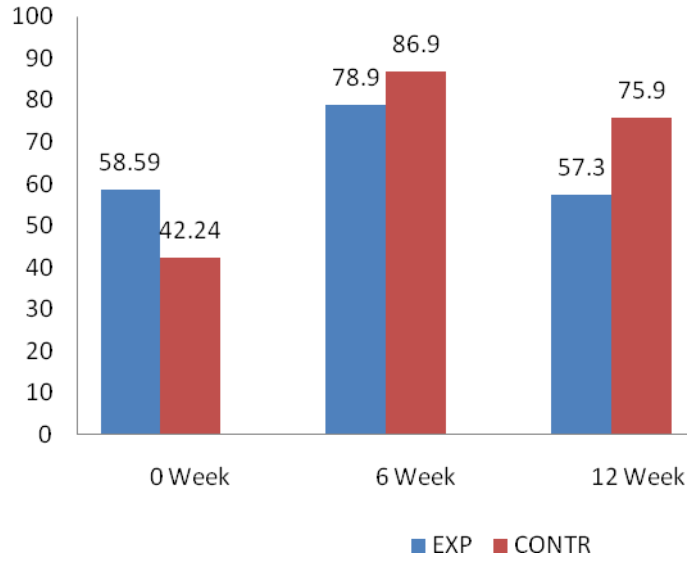


Fig. 9: Changes in high density lipoprotein levels of experimental and control groups at pre, mid and post traing periods.

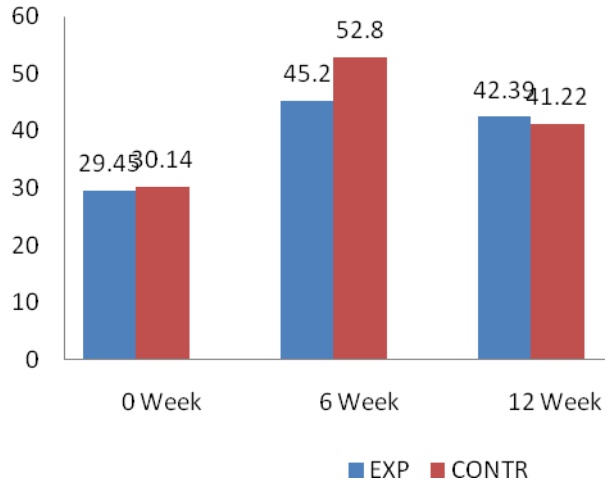


Fig. 10: Changes in low density lipoprotein levels of experimental and control groups at pre, mid and post training periods.

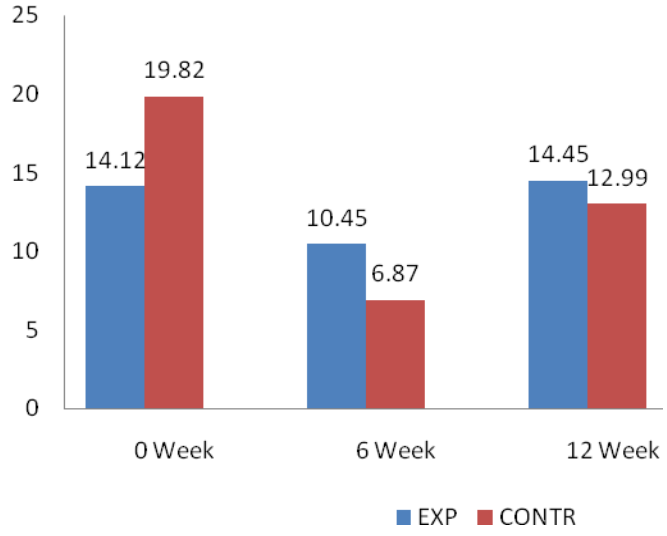
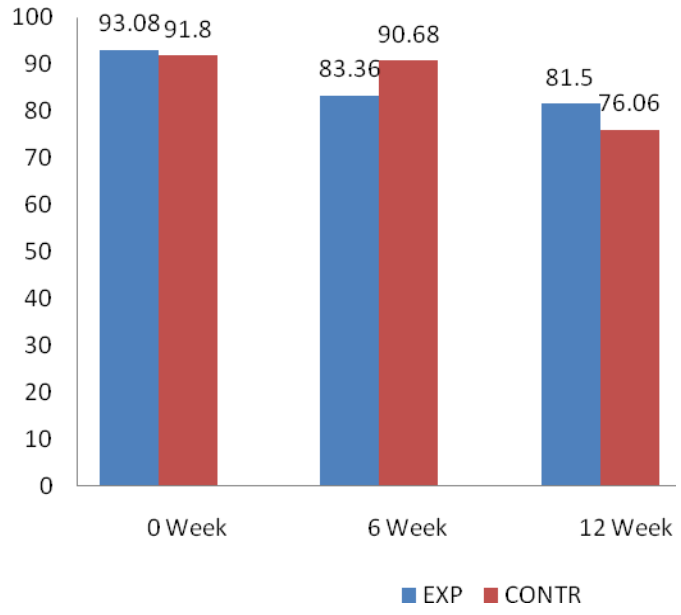


Fig. 11: Changes in fasting blood glucose levels of experimental and control groups at pre, mid and post training periods.



A careful observation of Table 3 shows a decrease in blood glucose level from 93.08 ± 5.7 mg to 81.5 ± 5.7 after training in the experimental group and from 91.80 ± 7.40 mg before starting the training to 76.06 ± 7.14 mg after training period in the control group. Similar decrease in T.C. level of both experimental and control groups was observed during the period of training. There was a minimal decrease in TG level of experimental group from 58.59 ± 23.29 mg before the start of training to 57.3 ± 15.65mg after training period. However, the level of TG rose from 42.29 ± 9.57 mg to 75.9 ± 28.03 after the training period in the control group. Table 3 further shows a considerable increase in HDL-C of experimental group from 29.45 ± 6.83 mg before the start of training to 42.39 ± 6.5 mg after training. Similar increase was found in HDL-C of control group. The changes in LDL-C of both experimental and control groups were minimal during the training period.

4.2D **In**formation regarding the changes in cardiovascular variables (systolic BP, Diastolic BP, Mean Arterial Pressure (MAP) pulse rate and double product (DP) is shown in Table 4.

Table 4: Descriptive Statistics of Test Results of Subjects in Cardiovascular efficiency Variables

Variables	**Group**	**N**	**Weeks**								
			0 (T1)			**6 (T_2)**			**12 (T_3)**		
			M	**SD**	**SE**	**M**	**SD**	**SE**	**M**	**SD**	**SE**
Systolic P	Expt	15	118	6.76	1.74	114	8.28	2.13	111.3	11.25	2.9
	Contr	15	116	8.16	2.10	115	8.12	2.09	116.5	8.79	2.28
Diastolic	Exp	15	72	7.74	2.00	69	10.3	2.66	70.6	8.83	2.27
P	Contr	15	77	6.27	1.61	75	7.5	1.94	75.4	7.53	1.94
MAP	Exp	15	87.2	6.76	1.74	84.4	9.86	2.54	84.3	8.62	2.22
	Contr	15	89.9	5.58	1.44	88.0	6.79	1.75	90.3	6.77	1.75
Pulse R	Exp	15	77.6	4.80	1.24	66.4	4.95	1.27	62.6	3.35	0.86
	Contr	15	80.0	5.37	1.38	77.8	6.23	1.61	78.4	4.64	1.19
Double	Exp	15	15223	23712	6122	7560	692.4	178.7	11516	176.3	4554
Product	Contr	15	9350	987	254	9511	2665	688	9232	958	247

Fig. 12: Changes in systolic blood pressure of experimental and control groups at pre, mid and post training periods.

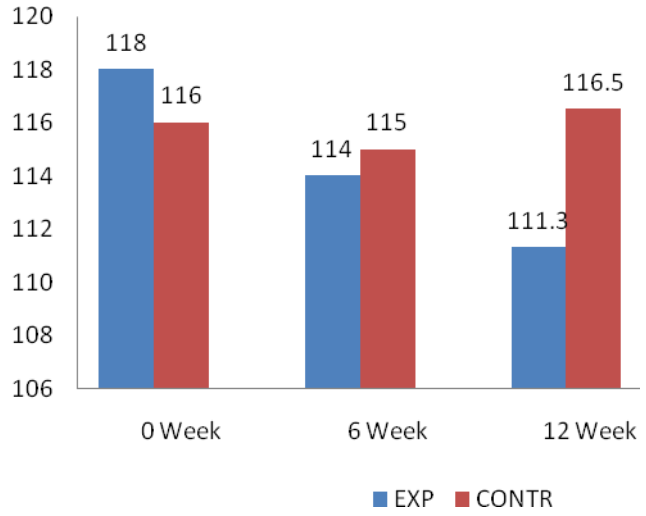


Fig. 13: Changes in diastolic blood pressure of experimental and control groups at pre, mid and post training periods.

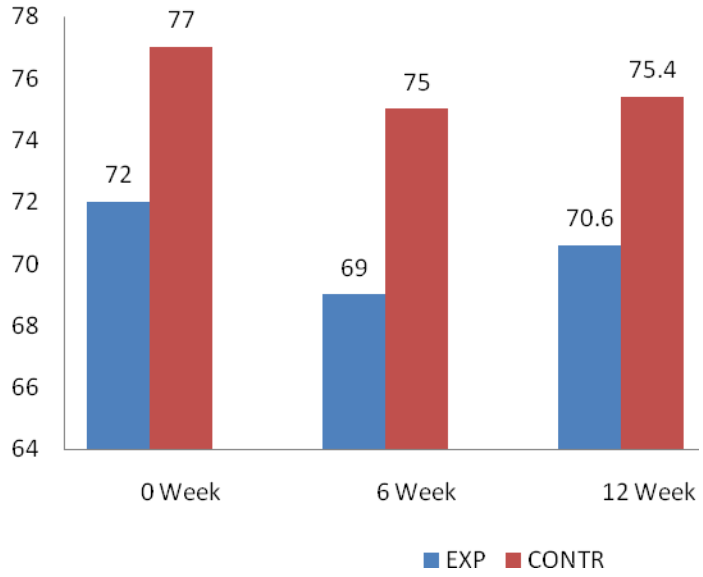


Fig. 14: Changes in mean arterial pressure of experimental and control groups at pre, training periods.

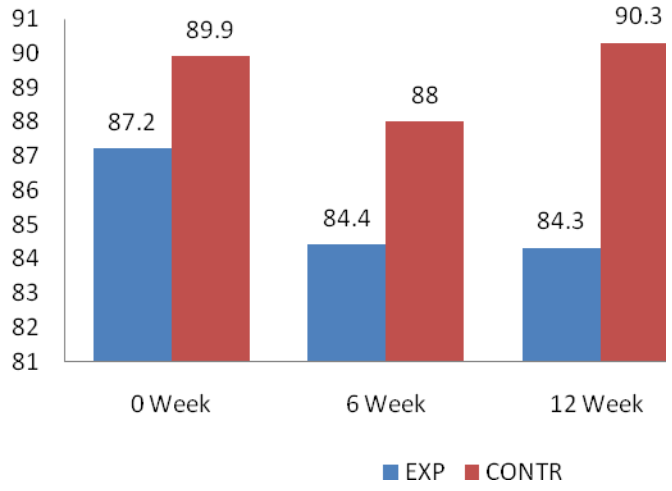


Fig. 15: changes in resting pulse rate of experimental and control groups at pre, mid and post training periods.

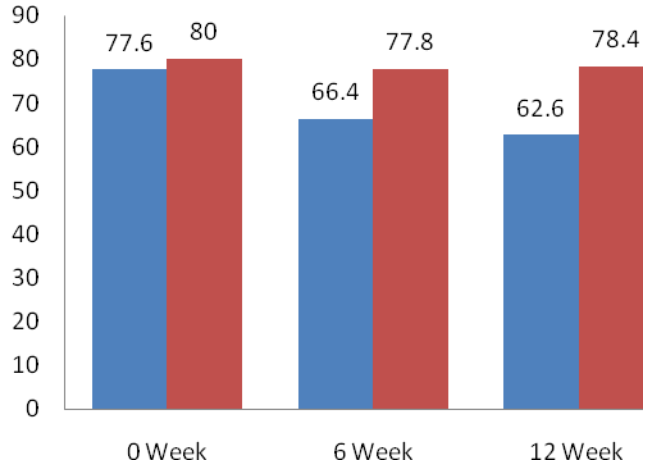
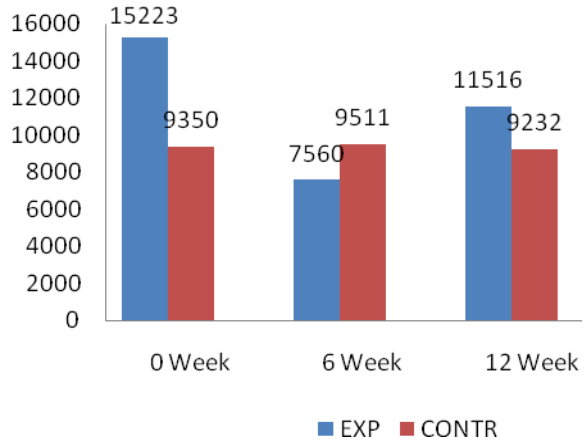


Fig. 16: Changes in double produccct of expperimental and control groups at pre, mid and post training periods.



Observation of the Table 4 shows that the experimental group had decreased systolic pressure from pre training level of 118 ± 6.76 mm Hg to 111.3 ± 11.25 post-training. Similarly there was a slight decrease in mean diastolic pressure from 72 ± 7.74 pre training to 70.6 ± 8.83 post- training. However, the control group had very minimal changes in both systolic and diastolic pressures. Furthermore, the experimental group has shown remarkable decrease in MAP from 87.2 to 84.3, pulse rate from77.6 to 62.6 and double product from 15,223 to 11,516. On the other hand, the control group recorded very minimal changes in MAP, pulse rate and the double product.

4.3 Hypotheses Testing

The major hypotheses for this investigation state that there is no significant effect of 12 week moderate intensity interval training in running on body fat patterns, lipids and lipoproteins, blood glucose and cardiovascular efficiency of sedentary Nigerian male adults. In order to test this hypothesis, it has been decomposed into several sub hypotheses which were tested in two different ways. In the first case, each sub-hypothesis was tested by computing the t- test for the

difference between experimental and control groups that occurred after 12 weeks of training. In the second case, in order to determine the magnitude and direction of the effects of 12 weeks of training in the different variables of the experimental group, repeated measures ANOVA was used. First the results of the computation of the t ratio for experimental and control groups are presented This was followed by the presentation of the results of the repeated measures ANOVA for the magnitude and direction of the effects of the training in the three levels of tests of the experimental group.

Sub hypothesis No. 1

There is no significant effect of 12 weeks moderate intensity interval training in running on body fat patterns of sedentary Nigerian male adults.

Sub hypothesis 1 was tested by computing t- ratio for difference between experimental and control groups in each of the body fat pattern variables, the result of which are shown in Table 5.

Table 5: Independent t-test statistics for the difference between experimental and control groups in body fat patterns after 12 weeks of moderate intensity interval training in running

Fat Pattern Variables	Groups	n	Mean	SD	t
C. i	Experimental	45	18.89	1.41	0.798
	Control	45	19.13	1.48	
Hip Cir.	Experimental	45	87.24	3.99	0.415
	Control	45	86.87	4.62	
Waist cir	Experimental	45	73.40	3.27	2.39
	Control	45	75.42	4.61	
WHR	Experimental	45	0.844	0.64	1.503
	Control	45	0.862	0.48	
BMI	Experimental	45	21.04	1.99	0.912
	Control	45	21.44	2.16	

$t(88) = 1.98 < 0.05$

Careful examination of table 5 shows significant difference between experimental and control groups only in waist circumference, suggesting that 12 week moderate intensity interval training in running caused a significant decrease in the waist circumference of the experimental group, which was not evident in other indices of body fat patterns. In order to appreciate the meaningfulness of the significant difference, Omega squared was computed to determine the percent of variance in waist circumference contributed by the interval training as shown below:

$$\text{Omega}^2 = \frac{t^2 - 1}{t^2 + n_1 + n_2 - 1}$$

Omega square showed that 5% of the total variance of the waist circumference was accounted for by the difference between the experimental and control groups. This shows that

95% of the variance in the waist circumference was accounted for by other factors. This result shows that moderate intensity interval training in running for 12 weeks can reduce waist circumference but is not adequate to bring about same changes in other body fat pattern variables of sedentary Nigerian male adults.

Sub hypothesis No. 2

There is no significant effect of 12 week moderate intensity interval training in running on lipid and lipoprotein of sedentary Nigerian male adults.

Sub- hypothesis was tested by comparing the changes in TC, TG, LDL-C and HDL-C of experimental and control groups after 12 weeks of moderate intensity interval training in running. The result of this is shown in Table 6.

Table 6: Independent t test statistics for the difference between experimental and control groups in lipids and lipoproteins after 12 weeks of moderate intensity interval training in running.

Lipid/Lipo Variable	Groups	n	Mean	SD	t
TC	Experimental	45	137.64	32.1	1.380
	Control	45	129.30	24.8	
TG	Experimental	45	64.83	20.65	0.617
	Control	45	68.39	31.38	
LDL-C	Experimental	45	13.20	5.43	0.162
	Control	45	13.23	2.43	
HDL-C	Experimental	45	39.04	13.31	0.716
	Control	45	41.41	17.84	

t(88) 1.98 < 0.05

Table 6 shows no significant difference between experimental and control groups after 12 weeks of moderate intensity interval training in running in TC, TG, LDL-C and HDL-C levels.

This suggests that the 12- week moderate intensity interval training in running was not adequate to produce significant changes in the selected lipid and lipoproteins variables of sedentary Nigerian male adults.

Sub-hypotheses No. 3

There is no significant effect of 12 moderate intensity interval training in running on blood glucose levels of sedentary Nigerian male adults.

Sub-hypothesis 3 was tested by comparing the changes in the blood glucose levels of experimental and control groups after 12 week, of moderate intensity interval training in running. The results of this are shown in Table 7.

Table 7: Independent t test statistics for the difference between experimental and control groups in blood glucose after 12 weeks of moderate intensity interval training in running

Variable	Groups	n	Mean	SD	t
Glucose	Experimental	45	85.99	7.31	0.102
	Control	45	86.18	9.92	

$t(88) = 1.98 < 0.05$

Table 7 shows no significant difference between experimental and control groups in their glucose levels after 12 weeks of moderate intensity interval training in running. Therefore the moderate intensity interval training in running underwent by experimental group was not adequate to produce significant changes in their blood glucose levels of sedentary Nigerian male adults.

Sub- hypothesis No. 4

There is no significant effect of 12 week, moderate intensity interval training in running on cardiovascular efficiency variables of sedentary Nigerian male adults.

In this study, cardiovascular efficiency was assessed by examining the changes in blood pressure, MAP, resting pulse rate, and double product. Sub-hypothesis No. 4 was therefore tested by computing changes in systolic and diastolic blood pressures, mean arterial pressure, resting pulse rate and double product of experimental and control groups after 12 weeks of moderate intensity interval training in running, the results of which are shown in Table 8.

Table 8: Independent t test statistics for the difference between experimental and control groups in the variables of their cardiovascular efficiency after 12 weeks of moderate intensity interval training in running

Variable	**Groups**	**n**	**Mean**	**SD**	**t**	**Omega Square**
Systolic BP	Experimental	45	114.44	9.18	1.382	
	Control	45	117.02	8.50		
Diastolic BP	Experimental	45	70.66	8.88	2.178	.04
	Control	45	74.53	7.92		
MAP	Experimental	45	95.24	8.43	2.65	.06
	Control	45	89.42	6.34		
Double	Experimental	45	7904	1221	5.50	.25
product	Control	45	9186	145		
Pulse rate	Experimental	45	68.91	7.76	6.99	.35
	Control	45	78.77	5.41		

$t(88) = 1.98 < 0.05$

A careful examination of Table 8 reveals significant difference between experimental and control groups in diastolic BP 70.66 and 74.53 , MAP 95.24 and 89.42, DP 7,904 and 9,186 and Pulse rate of 68.91 and 78.77, respectively after 12 weeks of moderate intensity interval training in running. The significant difference in diastolic pressure was because of lower diastolic pressure of the experimental group compared to the control group. The MAP of experimental group was significantly more than the control group, whereas the DP and PR were much less in

the experimental group compared to control group after 12 weeks of moderate intensity interval training in running. In order to determine the meaningfulness of those significant differences, between experimental and control groups in the various variables of cardiovascular efficiency, Omega square was computed for each variable, which is shown in the last column of Table 8. Table 8 thus shows the difference between experimental and control groups explained only 4% of the variance in diastolic pressure, 6% in MAP, 25% in DP and 35% of the variance in resting pulse rate. Taken together, these modifications due to 12 weeks of moderate intensity interval training in running significantly improved cardiovascular efficiency of sedentary Nigerian male adults, especially the double product and resting pulse rate, suggesting that the load on the heart significantly decreased.

Sub- hypothesis 5

There is no significant deference among the pre-mid and post- training tests in body fat patterns, blood pressure, serum glucose, lipids and lipoproteins of the experimental group. To test this sub-hypothesis, repeated measures ANOVA and Scheffe's post hoc tests were used for each of the variables, the result of which are presented in the following section.The mean and standard deviation of body fat patterns in the three tests are shown in table 9A.

Table 9A: Means and standard deviations of body fat pattern indices of experimental group in pre-, mid- and post-training test

Variable	Test	n.	Mean	SD	SE
Weight	0	15	61.47	6.116	1.57
	6	15	61.27	5.36	1.38
	12	15	62.68	5.50	1.42

Waist cir	0	15	73.40	3.15	.815
	6	15	74.33	3.51	.909
	12	15	72.47	3.09	.798
Hip cir	0	15	89.07	3.21	.831
	6	15	86.53	4.64	1.19
	12	15	86.13	3.56	.920
BMI	0	15	21.00	2.13	.552
	6	15	20.87	2.10	.542
	12	15	21.27	1.87	.483
WHR	0	15	.820	.041	.010
	6	15	.867	.081	.021
	12	15	.847	.051	.013
			.		
Ci.	0	15	19.00	1.36	.352
	6	15	19.20	1.32	.341
	12	15	18.47	1.55	.401

Fig. 17: Magnitude and direction of change in weight of experimental group from pre-mid-post- training.

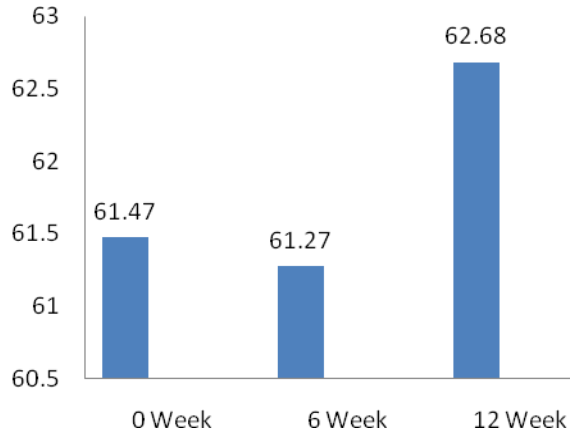


Fig. 18: Magnitude and direction of change in waist air cumterence of experimental group from pre-mid-post- training.

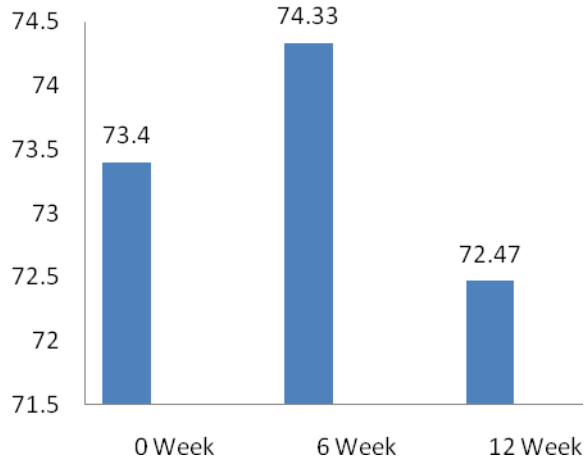


Fig. 19: Magnitude and direction of change in hip circomstance of experimental group from pre-mid-post- training.

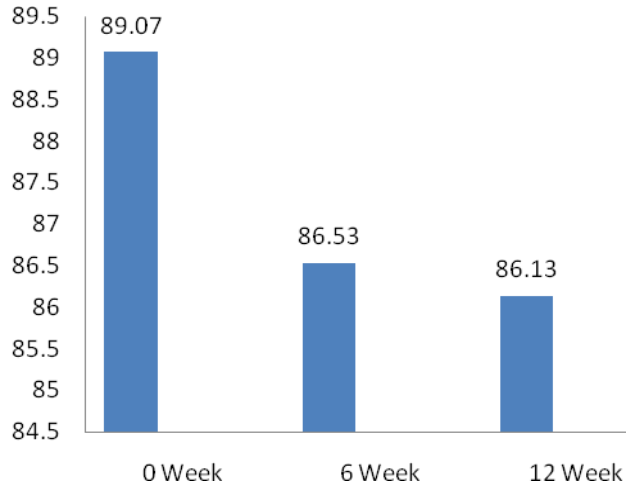


Fig. 20: Magnitude and direction of change in body mass index of experimental group from pre-mid and post- training.

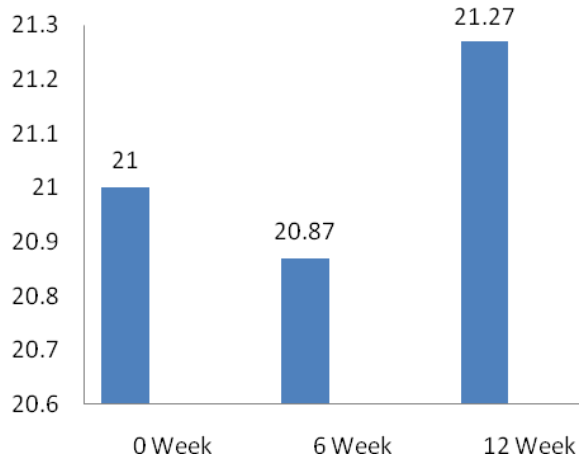


Fig. 21: Magnitude and direction of change weight hip ratio of experimental group from pre-mid-post- training.

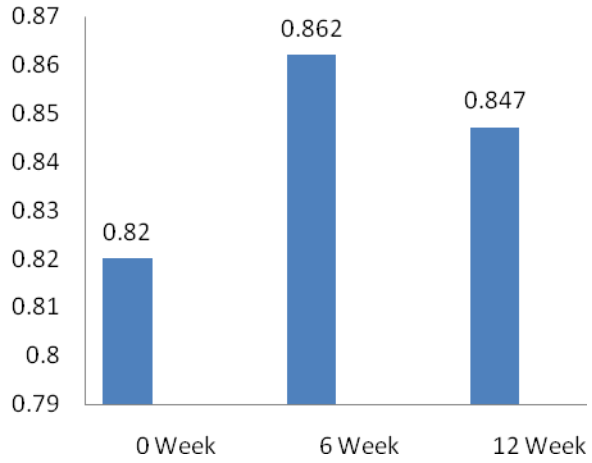


Fig. 22: Magnitude and direction of change in concify index of experimental group from pre-mid-post- training.

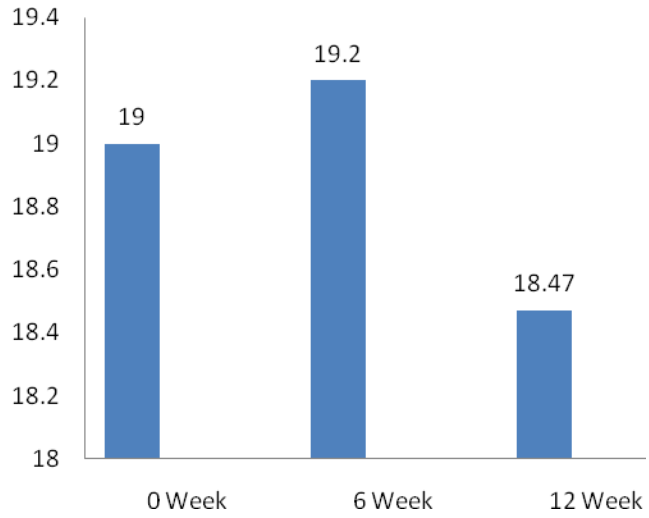


Table 9A shows changes due to 12 week moderate intensity interval training in running in weight, waist circumference, hip circumference, BMI, WHR and C.I. Although these changes were very minimal, it was felt necessary to find out the significance of these changes from test one to test three in these variables. This purpose was achieved by analyzing the data with repeated measures ANOVA, the results of which are shown in table 9B.

Table 9B: Repeated measures ANOVA for difference between tests in body fat patterns.

Variable	Source	SS	Df	Mean Square	F	Sig
Weight	B/groups	22.800	2	11.400	.355	.704
	Within groups	1350.400	42	32.152		
	Total	1373.200	44			
Waist circ.	B/groups	26.133	2	13.067	1.229	.303
	Within groups	446.667	42	10.635		
	Total	472.600	44			
Hip circ.	B/groups	75.911	2	37.956	2.553	.090
	Within groups	624.400	42	14.867		
	Total	700.311	44			
BMI	B/groups	1.244	2	.622	.150	.861
	Within groups	174.667	42	4.159		
	Total	175.911	44			
WHR	B/groups	0.016	2	.008	2.233	.120
	Within groups	0.155	42	.004		
	Total	0.171	44			
C.I	B/groups	4.311	2	2.156	1.076	.350
	Within groups	84.133	42	2.003		
	Total	88.444	44			

$F(2,42) = 3.23 < 0.05$

Table 9B shows no insignificant differences between the 3 tests in hip circumference, weight, waist circumference, BMI, WHR and C.I.

Means and standard deviation of values for lipids and lipoproteins in the 3 tests are shown in table 10A.

Table 10A: Means and Standard Deviation of Lipids and Lipoproteins of Experimental Group in Pre-Mid-Post Training

Variable	Test	n.	Mean	SD	SE
TC	0	15	130.23	20.33	5.26
	6	15	129.10	33.11	8.55
	12	15	123.56	19.87	5.13
TG	0	15	58.59	23.29	6.01
	6	15	78.90	15.43	3.98
	12	15	57.30	15.65	4.04
HDL-C	0	15	29.45	6.83	1.76
	6	15	45.27	17.77	4.58
	12	15	42.39	6.512	1.68
LDL-C	0	15	14.12	3.18	.823
	6	15	10.45	7.05	1.82
	12	15	14.45	4.71	1.21

Fig. 23: Magnitude and direction of change in total cholesterol level of experimental group from pre-mid- post training.

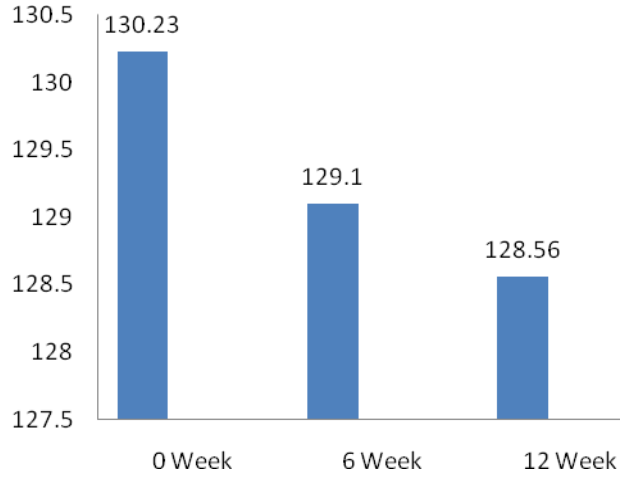


Fig.24: Magnitude and direction of change in triglyceride level of experimental group from pre-mid-post training.

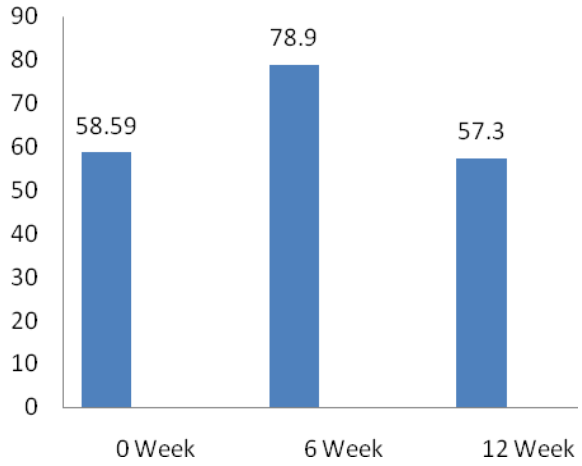


Fig. 25: Magnitude and direction of chanmge in high density hipoprotein levels of experimental group from pre-mid-post training.

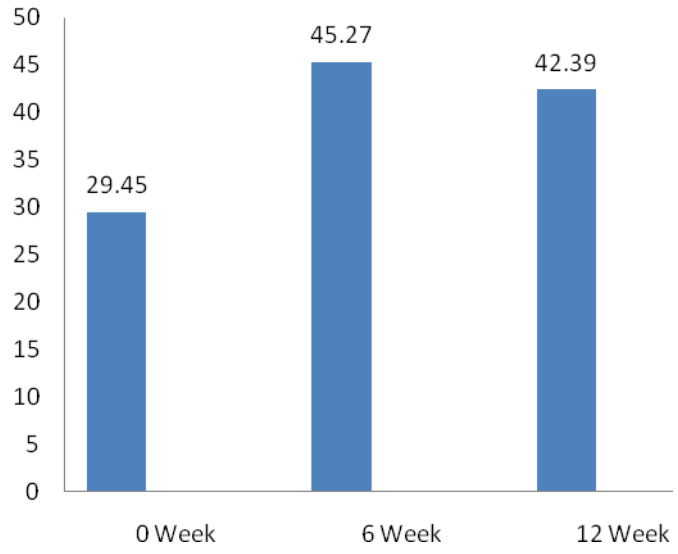


Fig. 26: Magnitude and direction of change in low density. Lipoprotein levels of experimental group from pre-mid-post training.

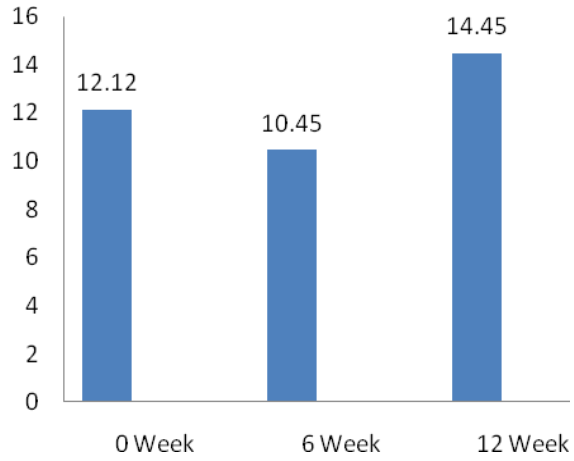


Table 10A shows decrease in T.C., TG and LDL-C from T_1 to T_3. In order to find out the significant level of these changes, the data were analyzed by using repeated measures ANOVA, the result of which is shown in table 10B.

Table 10B: Repeated measure ANOVA for difference between tests in lipids and lipoproteins

Variable	Source	SS	Df	Mean Square	F	Sig
TC	B/groups	21.841	2	10.921	.017	.983
	Within groups	26703.53	42	635.799		
	Total	26725.39	44			
TG	B/groups	4401.441	2	2200.721	6.439	.004
	Within groups	14365.22	42	342.030		
	Total	18766.72	44			
HDL-C	B/groups	2130.05	2	1065.026	7.887	.001
	Within groups	5671.79	42	135.043		
	Total		44			
LDL-C	B/groups	147.778	2	73.889	2.699	.079
	Within groups	1149.599	42	27.371		
	Total	1297.37	44			

$F(2, 42) = 3.23 < 0.05$

Careful observation of Table 10B shows insignificant differences between the 3 tests in the effect of 12 week moderate intensity interval training in running in TC and LDL.C and significant difference in TG and HDL.C. The significant difference in TG was due to greater decrease of TG after 12 weeks of training compared to pre-training level and after 6 weeks. The significant difference in the effects between tests for HDL.C was due to greater increase in HDL.C level after 6 weeks and 12 weeks of training compared to pre-training values.

Table 10C: Scheffe's Post Hoc test for significance of difference between tests in lipids and lipoproteins

Variable	(I) repetitions	(J) repetitions	Mean Difference (I-J)	Std. Error	Sig.
TC exp	Week 0	Week 6	1.1267	9.20723	.993
		Week 12	1.6733	9.20723	.984
	Week 6	Week 0	-1.1267	9.20723	.993
		Week 12	.5467	9.20723	.998
	Week 12	Week 0	-1.6733	9.20723	.984
		Week 6	-.5467	9.20723	.998
TG exp	Week 0	Week 6	-20.3067*	6.75308	.017
		Week 12	1.2867	6.75308	.982
	Week 6	Week 0	20.3067*	6.75308	.017
		Week 12	21.5933*	6.75308	.982
	Week 12	Week 0	-1.2867	6.75308	.017
		Week 6	-21.5933*	6.75308	.010
HDL-C exp	Week 0	Week 6	-15.8200*	4.24331	.002
		Week 12	-12.9400*	4.24331	.015
	Week 6	Week 0	15.8200*	4.24331	.002
		Week 12	2.8800	4.24331	.795
	Week 12	Week 0	12.9400*	4.24331	.015
		Week 6	-2.8800	4.24331	.795
LDL-C exp	Week 0	Week 6	3.6667	1.91037	.171
		Week 12	-.3333	1.91037	.985
	Week 6	Week 0	-3.6667	1.91037	.171
		Week 12	-4.0000	1.91037	.124
	Week 12	Week 0	.3333	1.91037	.985
		Week 6	4.0000	1.91037	.124

Based on observed means.

* The mean difference is significant at the .05 level.

Table 10 C revealed that the significant difference was due to the greater decrease in T_2 and T_3 compared to T_1 and T_3 compared to T_1 in TG. The significant difference between these groups was due to a greater increase in HDL-C in T_2 and T_3 compare to T_1.

Information regarding the mean values of glucose level before, week 6 and week 12 of moderate intensity interval training in running as shown in table 11A.

Table 11A: Means and standard deviations of glucose level of experimental group in pre, mid and post training tests.

Variable	Test	n.	Mean	SD	SE
Glucose	0	15	93.08	4.85	1.25
mg/lt	6	15	83.36	5.40	1.39
	12	15	81.54	5.70	1.47

Fig.27: Magnitude and direction of change in blood glucose levels of experimental group from pre-mid-post training.

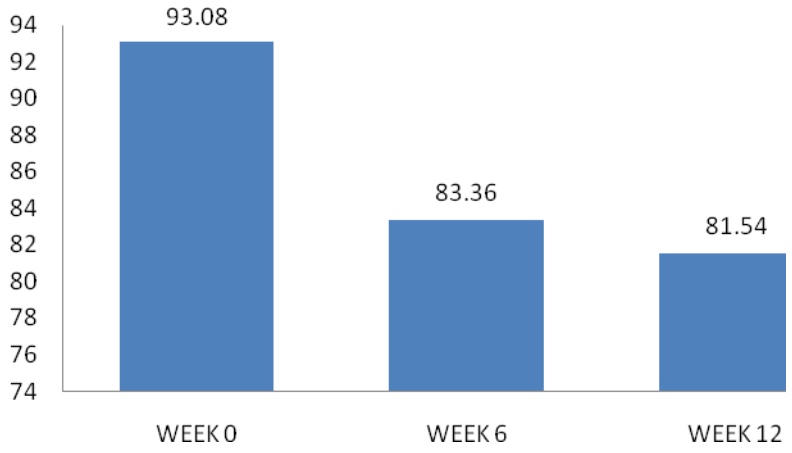


Table 11A shows decrease in glucose level for week 6 and 12 compared to pre-training level. The significant level of the difference between the 3 tests in glucose level was computed using repeated measures ANOVA. The result is presented in table 11B.

Table 11B: Repeated measures ANOVA for difference between tests in blood glucose

Variable	Source	SS	Df	Mean Square	F	Sig
Glucose	B/groups	1156.230	2	578.115	20.313	.000
	Within groups	1195.329	42	28.460		
	Total	2351.559	44			

F (2,42) = 3.23 < 0.05

Table 12B shows significant decrease in glucose between the tests. Scheffe's Post Hoc Test (table 12C) indicates that the significant difference was due to a greater decrease in T_2 and T_3 compared to T_1 in glucose level.

Table 11C: Scheffe's Post Hoc Test for significance of difference between tests in blood glucose

Variable	(I) repetitions	(J) repetitions	Mean Difference (I-J)	Std. Error	Sig.
Glucose mg/lt	Week 0	Week 6	9.726*	1.948	.000
		Week 12	11.546*	1.948	.000
	Week 6	Week 0	-9.726*	1.948	.000
		Week 12	1.820*	1.948	.649
	Week 12	Week 0	-11.546*	1.948	.000
		Week 6	-1.8200	1.948	.649

* Significant at 0.5 level.

Cardiovascular efficiency was examined in this study by studying the effect of 12 week moderate intensity interval training in running on systolic and diastolic BP, MAP, resting pulse rate and double product. Mean values in these different variables in pre, mid and post training tests are shown in Table 12A.

Table 12A: Means and standard deviations of cardiovascular variables of experimental group in pre, mid and post training test

Variable	Test	n.	Mean	SD	SE
Systolic	0	15	118.00	6.76	1.74
BP	6	15	114.00	8.28	2.13
	12	15	111.33	11.25	2.90
Diastolic	0	15	72.000	7.74	2.00
BP	6	15	69.33	10.32	2.66
	12	15	70.66	8.83	2.28
	1				
MAP	0	15	87.200	6.76	1.74
	6	15	84.400	9.86	2.54
	12	15	84.13	8.62	2.22
Pulse R	0	15	77.66	4.80	1.24
	6	15	66.40	4.95	1.27
	12	15	62.66	3.35	.86
DP	0	15	9169.3	846.66	218.6
	6	15	7560.0	692.48	178.7
	12	15	6982.6	861.58	222.4

Fig. 28: Magnitude and direction of change in diastolic blood pressure of experimental group from pre-mid-post training tests

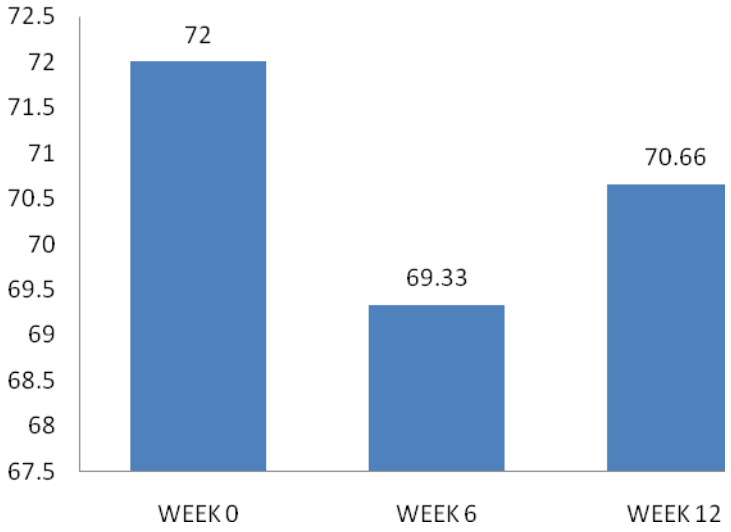


Fig. 29. Magnitude and direction of change in systolic blood pressure of experimental group from pre-mid-post training. Tests

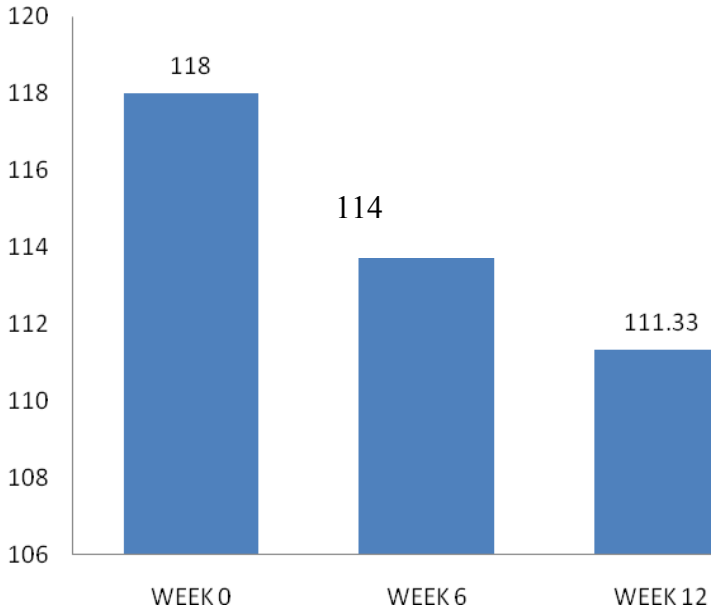


Fig. 30: Magnitude and direction of change in mean arterial pressure of experimental group from pre-mid-post training tests.

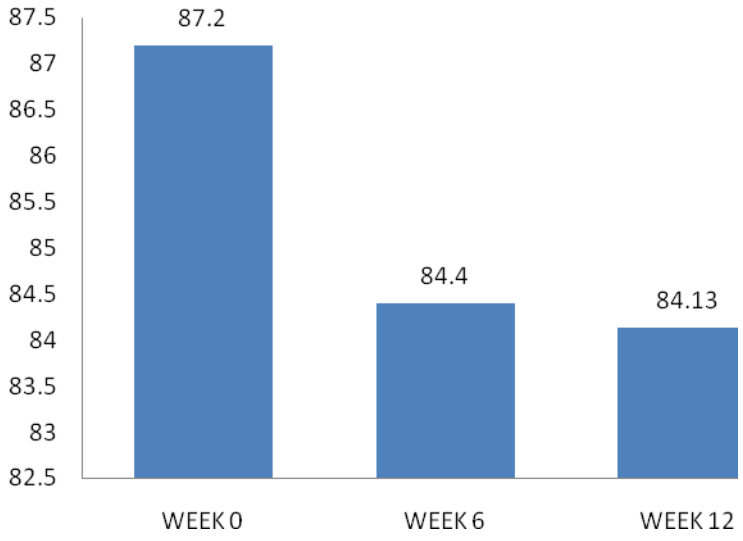


Fig. 31: Magnitude and direction of change in resting pulse rate of experimental group from pre-mid-post training tests.

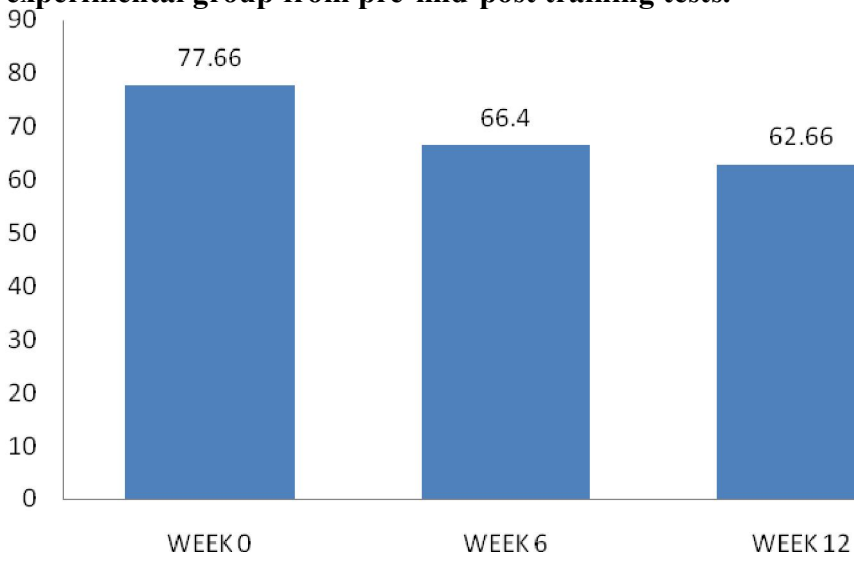


Fig. 32: Magnitude and direction of change in double pressure of experimental group from pre-mid-post training tests.

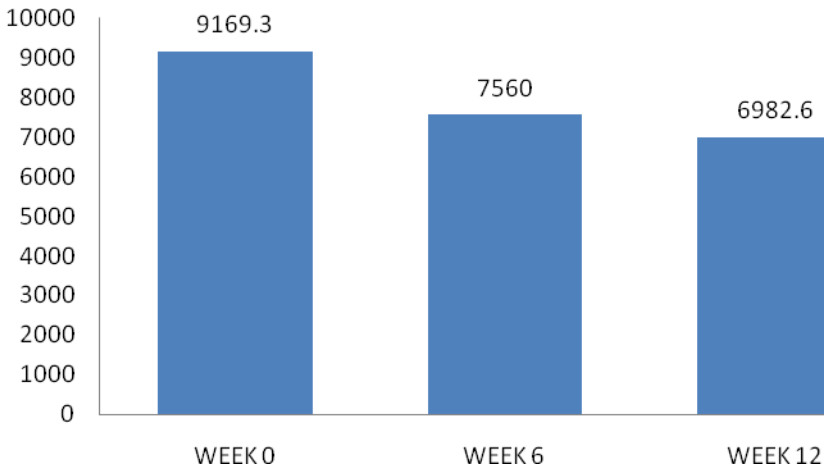


Table 12A shows decrease in systolic, diastolic blood pressures, resting pulse rate, MAP and DP, due to 12 week moderate intensity interval training in running. In order to find out whether the differences between the 3 tests within those variables are significant, repeated measures ANOVA was used to analyze the data as presented in table 12B.

Table 12B: Repeated measures ANOVA for difference between tests in cardiovascular efficiency variables.

Variable	Source	SS	Df	Mean Square	F	Sig
Systolic	B/groups	337.270	2	.889		
BP	Within groups	3373.333	42	80.317	2.103	.135
	Total	2711.111	44			
Diastolic	B/groups	53.333	2	26.667	.327	.723
BP	Within groups	3426.667	42	81.586		
	Total	3480.000	44			
MAP	B/groups	86.578	2	43.289		
	Within groups	3045.733	42	72.517	.597	.555
	Total	3132.311	44			
Pulse R	B/groups	1829.37	2	914.689		
	Within groups	824.267	42	19.626	46.607	.000
	Total		44			
Double	B/groups	38523893	2			
Product	Within groups	27141987	42		29.806	.000
	Total	65665880	44			

$F(2,42) = 3.23 < 0.05$

The decrease due to 12 week moderate intensity interval training in running in resting pulse rate and double product became significant only after 6 weeks of training. However, the decrease in systolic and diastolic blood pressures and MAP was not significant. Scheffe's Post Hoc result in table 12C shows that the significant decrease in resting pulse rate and double product was because of greater decrease in T_2 and T_3 compared to T_1.

Table 12C: Scheffe's Post Hoc test for significance of difference between tests in cardiovascular efficiency variables

Variable	(I) repetitions	(J) repetitions	Mean Difference (I-J)	Std. Error	Sig.
Systolic exp BP	Week 0	Week 6	4.0000	3.27246	.480
		Week 12	6.6667	3.27246	.138
	Week 6	Week 0	-4.0000	3.27246	.480
		Week 12	2.6667	3.27246	.719
	Week 12	Week 0	-6.6667	3.27246	.138
		Week 6	-2.6667	3.27246	.719
Diastolic exp BP	Week 0	Week 6	2.6667	3.29823	.723
		Week 12	1.3333	3.29823	.922
	Week 6	Week 0	-2.6667	3.29823	.723
		Week 12	1.3333	3.29823	.922
	Week 12	Week 0	1.3333	3.29823	.922
		Week 6	1.3333	3.29823	.922
MAP exp	Week 0	Week 6	2.8000	3.10950	.669
		Week 12	3.0667	3.10950	.618
	Week 6	Week 0	-2.8000	3.10950	.669
		Week 12	.2667	3.10950	.996
	Week 12	Week 0	-3.0667	3.10950	.618
		Week 6	-.2667	3.10950	.996
Pulse rate exp	Week 0	Week 6	11.2667*	1.61763	.000
		Week 12	15.0000*	1.61763	.000
	Week 6	Week 0	-11.2667*	1.61763	.000
		Week 12	3.7333*	1.61763	.081
	Week 12	Week 0	-15.0000*	1.61763	.000
		Week 6	-3.7333	1.61763	.081
Double Product exp.	Week 0	Week 6	1609.3333*	293.53882	.000
		Week 12	2186.6667*	293.53882	.000
	Week 6	Week 0	-1609.3333*	293.53882	.000
		Week 12	577.3333	293.53882	.157
	Week 12	Week 0	-2186.6667*	293.53882	.000
		Week 6	-577.3333	293.53882	.157

Based on observed means. * The mean difference is significant at the .05 level.

4.4 DISCUSSION

The purpose of this study was to find out the effects of 12 week moderate intensity interval training in running on body fat patterns, lipids and lipoproteins, blood glucose and blood pressure of sedentary Nigerian male adults. The 12- week training programme, which the subjects went through, produced several significant changes which are discussed in this section.

4.4A Effects of 12 Week Moderate Intensity Interval Training in Running on Body Fat Patterns of Sedentary Nigerian Male Adults.

The finding of this study revealed significant decrease in waist circumference due to 12-week moderate intensity interval training in running. This decrease may be attributed to the fact that moderate intensity interval training in running, four times per week for 12 weeks is adequate to produce a significant decline in waist circumference of sedentary adults. This finding supports the findings of Friendlanda, Cassara and Brooks (1999), Gan, Kriketos, Ellis, et al (2003), Venkateswarlu (2009) and Vesby, Anderson and Sjodin (1998), that moderate intensity training reduces body weight and waist circumference. However, in this study, 12 week moderate intensity interval training in running did not produce any significant effect on hip circumference, weight, BMI, WHR and C.I, which contradicts the findings of Wilmore and Costill (1994), Tremblay, Swimoneau and Bouchard (1994), Ohkowara, et al (2007) and Venkateswarlu (2010), that continuous training for 12 weeks or more, significantly reduces body fat patterns. The discrepancy between this study and previous studies may be explained by the fact that the subjects of this study were neither obese nor overweight, whereas previous studies have shown that significant decrease in fat patterns due to moderate training can be found more among adults who are more obese and overweight (Barbeau, Gutin, Litaker; et al, 1999) and

among those who undergo high intensity training (Tremblay; et al, 1994; Adeyanju, 2002). Had this subjects in this study undergone high intensity interval training, there could have been a significant decrease in body fat patterns. Another important point, this study has observed that significant decrease in waist circumference is an indication that fat deposits at this region are more mobilized than those at other regions, apparently be due to higher percent body fat deposits in that region, Contrary to expectations, the subjects of this study, after 12 weeks of training, showed an increase in their body weight compared to the control group which did not undergo any training. This surprise finding tends to show that moderate intensity interval training in running increases appetite, thereby causing a positive energy balance, which resulted in increase in body weight. Physical exercise training can lead to muscle atrophy which may be responsible for the increase in weight of the experimental group especially in week 12.

4.4B Effects of 12 Week Moderate Intensity Interval Training in Running on Lipids and Lipoproteins of Sedentary Nigerian Male Adults

Moderate intensity interval training in running performed 4 times a week for 12 weeks did not produce significant changes in TC, TG, HDL-C and LDL-C in this study. Although there was increase in HDL-C and decrease in other variables, these were not significant, which suggests that the changes produced in these variables are only directional without any statistical importance . This may be attributed to the fact that moderate intensity training performed 4 times a week for 12 weeks was not adequate to produce significant changes in lipids and lipoproteins. This further supports the failure of this study to find significant changes in body fat patterns. This position is supported by findings of Wood, Haskell, Blair, et al; (1983), Bouchard, et al (1993), Dowlin (2001), Leon and Sanchez (2001), Pekkanen (2009) and

Venkateswarlu (2010), according to which significant change in lipids and lipoprotein can be produced only after a prolonged period of training, especially when the training is of moderate intensity. Alternatively, significant changes can be expected due to moderate intensity training when the subjects are overweight, obese and on high fat diet as reported by Dustin and Thompsen (2001), Adeyanju (2002), Musa (2003), Dustin and Grandjean (2002) and Venkateswarlu (2011). In this study, the subjects are normal in their weight, lipids and lipoproteins and they underwent moderate intensity interval training, 4 times a week and for only 12 weeks. Similarly, Williams ; (2007) observed that subjects with low baseline HDL-C levels will show minimal increase in HDL-C after exercise training. However some differences in changes were observed between test periods especially for triglyceride and HDL.C. This observation further agrees with the earlier position that had this training gone beyond 12 weeks, these variables would have had significant modifications.

4.4C Effects of 12- Week Moderate Intensity Interval Training in Running on Blood Glucose Level

Several studies have shown that moderate to vigorous exercise training performed for a prolonged period of time can reduce fasting blood glucose levels (Bouchard, 1993; Despres, Pouliot, Morjani, et al, 1991; Leon and Sanchez, 2001). This study did not reveal any significant effect on fasting blood glucose level, which is contrary to the findings of previous studies. This may be attributed to two important differences in the present study and the previous studies. First, the main subjects who were used in this study had normal levels of fasting glucose, whereas in the previous studies, subjects had more than normal levels of glucose before the training. Any change in blood glucose can be expected only in individuals who have higher pre-training glucose levels. Secondly, previous studies, involving moderate to vigorous training

were conducted over prolonged period of time which was at least 24-30 weeks, whereas this study involved only moderate intensity training performed in 12 weeks. Had this training been performed for a longer period of time, the results would have been similar to those reported by other studies on effects of exercise on fasting blood glucose levels. The fluctuation observed in the glucose level of the experimental as well as the control groups were all within the normal range. In normal individuals, serum glucose levels are consistently maintained so as to avoid a situation where one will be hypoglycemic or hyperglycemic.

4.4D Effects of 12 week Moderate Intensity Interval Training in Running on Cardiovascular Efficiency

Cardiovascular efficiency was examined in this study by observing changes in systolic and diastolic blood pressures, mean arterial pressure, resting pulse rate and double product. The result of this study showed improvement in cardiovascular efficiency by significant decreases in systolic and diastolic BP, MAP, RPR and DP. Similar findings were reported by Flowman and Smith (1997), Walace (2003) and Cornelissen and Fagard (2005).This is an indication that moderate intensity interval training in running for as short as 12 weeks is enough to bring about substantial positive changes in cardiovascular efficiency variables of sedentary Nigerian male adults. The significant decrease in DP indicates a decrease in the load on the myocardium due to decrease in resting pulse rate and systolic blood pressure. These cumulative effects from moderate intensity interval training improve cardiovascular efficiency and minimize cardiovascular risk factors.

4,4E . The difference in magnitude and direction of change among tests of the experimental group after 12 week moderate intensity interval training in running.

The findings of this study revealed a number of changes in the variables studied after each test period of the experimental group as follows.

(a) Body fat patterns: The changes observed were not significant in all the variables and at each test level. This suggests that changes in body fat patterns due to moderate intensity interval training in running was not adequate to produce significant changes even at the termination of training in week 12.

(b) Lipids and lipoproteins .

Comparison of changes in the three tests for lipids and lipoproteins revealed difference between T1 and T2, T2 compared to T3 as well as between T2 and T3.

In triglyceride, there were significant differences between the first and second tests and between the second test and third suggesting that the training effect could have been more visible for triglyceride if it lasted more than 12 weeks.

HDL-C also showed some changes among the three test periods. There was significant differences when result of the first test is compared to the second test just as the case with test one compared to test three. This finding further confirmed that the changes would have been more if this training had lasted more than 12 weeks. However TC and LDL-C did not show any marked differences when the results of test periods are compared.

(c) Glucose. This study has revealed some decrease in fasting glucose as indicated from the three test periods. Significant decrease was observed during T3 as compared to T1 and

T2.This suggests that moderate intensity interval training could lead to positive changes in fasting blood glucose of previously sedentary adults if it lasts longer than 12 weeks.

(d) Cardiovascular efficiency variables

Cardiovascular efficiency variables were in this study found to have decreased. Comparing the changes from T1 to T3 there were not much differences in systolic, diastolic blood pressure and mean arterial pressure. This suggests that these cardiovascular variables are either stable in normal subjects or that the training duration and or intensity were not adequate to bring about the desired positive changes. However, significant differences were observed in test results for resting pulse rate and double product suggesting that moderate intensity interval training done in 12 weeks is adequate to produce result as early as in week six.

CHAPTER FIVE

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

5.1 Summary

Over the years, physical activity has been found to prevent and help in treating many established CVD risk factors, such as elevated blood pressure, insulin resistance and glucose intolerance, elevated TG concentration, LDL-C, HDL-C concentrations and obesity. Exercise in combination with weight reduction can decrease LDL-C concentrations and limit the reduction in HDL-C (Stelanick, et al, 1998).

The magnitude of the exercise effect is said to be influenced by characteristics of the exercise intervention, individual variation and whether exercise produces concomitant reduction in body weight. The effect on CVD risk factors can be large in some individuals and obviate the need for other interventions. In general, the effects of exercise on CVD risk factors is substantial, especially when done in combination with other lifestyle changes, such as dietary composition and weight loss.

Based on the above facts, this study was conducted to find out the effects of 12 week moderate intensity interval training in running on body fat patterns, lipids and lipoproteins, blood glucose and cardiovascular efficiency of sedentary Nigerian male adults. To achieve this purpose, 30 sedentary male adults were randomly selected and assigned to either experimental (N = 15) or control (N = 15) groups. The experimental group underwent the training 4 times each week for 12 weeks. The training consisted of warm up, main activity and warm down. In the first four weeks, subjects alternated the running distance between 100 meters and 200 meters. In second 4 weeks, the running distance was 200 meters and 300 meters and in the final 4 weeks subjects were alternating between 300 meters and 400 meters. The days involved were

Mondays, Tuesdays, Thursdays and Fridays. This was done to enable subjects to run each alternate course two times in a week.

During the 12 weeks, tests in all the variables under investigation were carried out at base-line, regarded as T_1, after 6 weeks (T_2) and at the termination of training in week 12 (T_3). The results were as follows;.

1. The results of the study indicated significant decrease in waist circumference ,but not in the other body fat pattern variables such as weight, hip circumference, WHR, BMI, and CI of sedentary Nigerian male adults.
2. Lipids and lipoproteins examined were not found to have been significantly affected, by moderate intensity interval training for 12 weeks although there were some minimal decreases in TG, TC, LDL-C and increase in HDL-C. However, these were not statistically significant.
3. 12 week moderate intensity interval training in running performed 4 times a week did not have significant effects on fasting blood glucose.
4 Moderate intensity interval training in running performed 4 times a week for 12 weeks had significant effect on the cardiovascular efficiency variables examined in this research for sedentary Nigerian male adults..

On the whole, the major contribution of this study was that it showed that, training induced changes vary from set of variables to set of variables. Moderate intensity interval training followed in this study showed significant improvements in cardiovascular efficiency and waist circumference .Whereas the intensity and duration followed in this study were inadequate to produce changes in lipids, serum glucose and some body fat patterns of sedentary Nigerian male adults.. This result therefore suggest that cardiovascular efficiency can be improved fast and greater than the modifications of lipids, glucose and body fat patterns that may require

longer training duration preferably of higher intensity. The implication for this finding for clinical practice is that the intensity and duration of any training protocol should be for that specific purpose of training. If the training is to promote health by reducing weight and lipid content of the body, it should be for a longer period of more than 12 weeks. Whereas if it is only to improve body cardiovascular fitness, the training protocol used in this study can be adequate

5.2 Conclusions

On the bases of the findings and in view of the limitations of this study, the following conclusions are drawn.

1. Moderate intensity interval training in running, performed 4 times a week will not produce positive changes in body fat patterns. However, waist circumference can be modified by this mode of exercise training.

2. Modifications in serum levels of TC, TG, HDL-C cannot be achieved by engaging in moderate intensity interval training lasting for 12 weeks.

3. Moderate intensity interval training cannot be used if the desire is to lower blood glucose level.

4. Moderate intensity interval training in running can be prescribed for the improvement of cardiovascular efficiency or fitness.

. 5.3 Recommendations

In the course of the present investigation, the researcher came across several unresolved issues ,which are suggested below for further research.

1. Moderate intensity interval training is recommended to improve cardiovascular efficiency and reduce waist circumference of previously sedentary male adults.
2. High intensity interval training in any mode of exercise is recommended to bring about positive changes in body fat patterns, glucose and lipids and lipoproteins.
3. Studies similar to the present one should be conducted on different age and body weight groups.
4. Studies similar to the present investigation be conducted by increasing both duration and intensity on sedentary adult men and women.
5. In order to minimize attrition in training, moderate intensity training in any mode of exercise for a prolonged period is recommended to produce positive changes in disease risk factors of adults.

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