

RELATIONSHIP BETWEEN SOMATOTYPES AND  
CARDIOVASCULAR DISEASE RISK FACTORS IN NIGERIAN  
UNDERGRADUATE STUDENTS OF KADUNA STATE UNIVERSITY,  
KADUNA, NIGERIA

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APRIL, 2021

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A THESIS SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES,  
AHMADU BELLO UNIVERSITY, ZARIA, NIGERIA  
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD  
OF DOCTOR OF PHILOSOPHY IN HUMAN ANATOMY

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APRIL, 2021

## DECLARATION

I hereby declare that the work in this thesis titled “**Relationship Between Somatotypes and Cardiovascular Disease Risk Factors in Nigerian Undergraduate Students of Kaduna State University, Kaduna, Nigeria**” has been carried out by me in the Department of Human Anatomy, under the supervision of Prof. S.S. Adebisi, Prof. B. Danborno and Prof. S.A. Akuyam.

The information derived from the literature has been duly acknowledged in the text and a list of references provided. No part of this work has been previously presented for another degree or diploma at any institution.

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Signature

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Date

## CERTIFICATION

This thesis entitled “RELATIONSHIP BETWEEN SOMATOTYPES AND CARDIOVASCULAR DISEASE RISK FACTORS IN NIGERIAN UNDERGRADUATE STUDENTS OF KADUNA STATE UNIVERSITY, KADUNA, NIGERIA” by Abdulwaheed Abdulazeez OYEWALE meets the regulations governing the award of the degree of Doctor of Philosophy in Human Anatomy of Ahmadu Bello University, Zaria, and is approved for its contribution to knowledge and literary presentation.

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## **DEDICATION**

This work is dedicated to the memory of my late caring father, Mallam Abdulazeez Oyewale Alayemi. May Allah have mercy on his soul and bless my aged mother, Mrs. Rafatu Abike Oke for her relentless prayer.

## ACKNOWLEDGEMENTS

All praise and gratitude to Almighty Allah for His divine mercy, favour and grace for the pursuit of this degree and completion of the thesis. First and foremost, I want to acknowledge the immense contributions of my professional supervisors: Prof. S.S. Adebisi, Prof. S.A. Akuyam and Prof. B. Danborno for their fatherly guidance, support, concern and taking the pain to read through all the contents of this thesis. I am greatly indebted to my mentor in the field of biological anthropology, Prof. B. Danborno who devoted every hour of his time to see to the success of this work. My sincere appreciation to Prof. S.S. Adebisi for his encouragement and optimistic approach throughout. My endless gratitude to Prof. S. A. Akuyam for his wise, helpful and positive counsel that enabled me to see myself not as I was, but as I could become and for this inspiration, I am deeply grateful.

I express my profound gratitude to Dr. J. A. Timbuak for the invaluable statistical advice/analyses and guidance. Dr. S. A. Musa, your contributions are gratefully acknowledged. I would also like to thank my HODs, Dr. D.S. Amaza (KASU) for his proof reading and data management support and encouragement; and Dr. (Mrs.) Z. M. Bauchi (ABU) for her support and encouragement. Special thanks to my elders and teachers, Dr. W.O. Hamman, Dr. U.E. Umana, Dr. A.U. Dahiru, Dr. Y.N. Abdullahi, Dr. A.A. Buraimoh and others numerous to mention, who always encourage me to persevere and move on. A special word of thanks to my dedicated and enthusiastic research assistants and students: Kabiru Sani, Aminu Musa, Abdurrazzaq Shuaibu, Zainab Aliyu and others. My special appreciation to Mallam Aliyu Garba, Nura Umar and Yaqub Ibrahim of Histology Unit, Department of Human Anatomy, KASU and Mrs. Sarah Kase of Chemical Pathology Department of BDTH, KASU for their support, advice and biochemical analyses.

My most sincere gratitude to all members of staff (academic and non-academic) in the Department of Human Anatomy, Faculty of Basic Medical Sciences, College of Medicine, Kaduna State University, Kaduna and Ahmadu Bello University, Zaria for their contributions in various ways making this research a success. Special thanks to all students that participated in the research.

My heartfelt appreciation to my loving families for their wonderful moral support: my late father, Mallam Abdulazeez Oyewale Alayemi, may Allah have mercy on his soul for his endless upbringing and support; and my mother, Mrs. Rafatu Abike Oke, for her care and loving prayers. My wife (Hajiya Sarat O. Bankole) for her immeasurable support, patience and understanding; and my caring children (Maryam, Abdullahi, Abdurrahman and Aishah). Other family members are appreciated, may Allah reward you all. My friends, too numerous to mention who helped in various capacities to the success of this work, may Almighty bless you beyond. Prof. A. Ajibola, your support and encouragement is well appreciated. I salute all my Islamic scholars for their various

advice and encouragement, thank you all. A number of other people have contributed to the progress and completion of the work presented in this thesis, and their contributions are gratefully acknowledged.

Above all, I thank Almighty Allah (SWT) for the life long, strength and the grace to complete this work. I am extending my special thanks to the dear readers of this thesis, I hope that the contents of this investigation will help them in their pursuit of knowledge.

## ABSTRACT

The variation in human body physique has been a subject of interest to physical anthropologists and human biologists since time immemorial. It is important to understand and analyze the factors associated with body physique, in order to have a better understanding of the morphological structure of the human body. Despite several decades of research, the relevance of body fat distribution to the risk of cardiovascular disease remains unclear. This study aimed to investigate the relationship between the somatotypes and cardiovascular disease risk factors in Nigerian undergraduate students. This is a cross sectional study conducted among the undergraduate students of Kaduna State University, Kaduna, Nigeria. Relationships between somatotype, physiological and biochemical parameters were considered in five hundred and sixty (560) apparently healthy students consisting of females (n = 181) and males (n = 379), aged 17 - 33 years. Somatotype was assessed using the Heath Carter anthropometric somatotype method. Risk factors included systolic and diastolic blood pressures (SBP, DBP), triglycerides (TG), total cholesterol (TC), high density lipoprotein cholesterol fraction (HDL-C), low density lipoprotein cholesterol (LDL-C), very low density lipoprotein cholesterol (VLDL-C) and atherogenic index (TC/HDL-C ratio, AI). Anthropometric assessment included measurement of weight, height, waist and hip circumferences, biepicondylar breadth of humerus and femur and skinfold thickness at different sites. Serum levels of TC, HDL-C and TG were measured. LDL-C was calculated by the Friedewald formula. Statistical analysis was done to examine the associations between anthropometric variables and lipid. One-way analysis of variance (ANOVA) was used to evaluate the statistical significance of differences between sex and age related groups. The comparison of somatotype components within the BMI classification was also analyzed using ANOVA. A significant level at 0.05 was used for all analyses employing IBM SPSS version 20. Pearson's correlation coefficients between each somatotype component and each blood pressure were calculated. Correlations between risk factors and each somatotype component were calculated. Significant correlations ranging from - 0.23 to + 0.23 in males and - 0.20 to + 0.30 in females. The statistical analysis showed that females were significantly more endomorphic and less ectomorphic than males. SBP and DBP showed an upward tendency with age in both sexes. The results suggest that in females, correlations between somatotype and blood pressure tended to be stronger, while in males this pattern was less consistent. The results showed that, the somatotype component ratings of females and males were found to be 2.50 - 3.07 - 2.54 and 2.30 - 3.25 - 2.66 respectively. There was also significant association between somatotype components and BMI, depicting that the increase in endomorphy and mesomorphy components were a risk factor having predisposition towards certain diseases. In general, for males and females, correlation between blood pressure and endomorphy were positive, while between blood pressure and ectomorphy were negative. This suggests that ponderosity and muscularity have the opposite effect; however linearity of physique could offer an



adaptive advantage. For each cardiovascular risk, those with a poorer profile tended to be more endomorphic and mesomorphic and less ectomorphic than those with a better profile who were more ectomorphic and less endomorphic and mesomorphic. The association was more apparent in males than in females and more so in those at 26 - 33 years of age than in the younger age group. Although the correlations suggest that body type is weakly associated with common cardiovascular risk factors in healthy females and males, somatotype associations are more apparent at the extremes of the distributions of specific risk factors. BMI, WC and WHR were similarly and importantly associated with blood pressure and lipids. The findings from this study highlights the importance of these intermediate risk factors on the pathway between excess body fat and cardiovascular disease.

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## LIST OF ABBREVIATIONS

ADP	Adenosine diphosphate
AI	Atherogenic index: TC/HDL
ANOVA	Analysis of variance
Apo-B	Apolipoprotein B
AT	Adipose tissue
ATP	Adenosine triphosphate
BMI	Body mass index
BP	Blood pressure
BW	Birth weight
CAD	Coronary artery disease
CDAH	Childhood determinants of adult health
CHD	Coronary heart disease
CI	Confidence interval
CRP	C-reactive protein
CT	Computed tomography
CVD	Cardiovascular disease
CVRFs	Cardiovascular risk factors
DBP	Diastolic blood pressure
ECTO	Ectomorphy
EDTA	Ethylene diamine tetraacetate
ENDO	Endomorphy
ERFC	Emerging risk factors collaboration
FC	Free cholesterol
FFA	Free fatty acid

FFM	Fat free mass
FM	Fat mass
g/dL	Gram per decilitre
HC	Hip circumference
HDL-C	High density lipoprotein cholesterol
Ht	Height
IAF	Intra-abdominal fat
IHD	Ischaemic heart disease
KASU	Kaduna State University
LDL-C	Low density lipoprotein cholesterol
MESO	Mesomorphy
MetS	Metabolic syndrome
MI	Myocardial infarction
mm Hg	Minimetre of mercury
mmol/L	Millimole per litre
NCEP ATP III	National Cholesterol Education Program Adult Treatment Program III
NHANES	National Health and Nutrition Examination Survey
NHLBI	National Heart, Lung, and Blood Institute
NIH	National Institute of Health
OGTT	Oral glucose tolerance test
PAD	Peripheral artery disease
PDAY	Pathobiological Determinants of Atherosclerosis
ROC	Receiver operating characteristic curves
ROS	Reactive oxygen species

RR	Risk ratio
SADi	Somatotypical attitudinal distance
SAT	Subcutaneous adipose tissue
SBP	Systolic blood pressure
SD	Standard deviation
TC	Total cholesterol
TG	Triglyceride
US	United States
WC	Waist circumference
WHO	World Health Organization
WHR	Waist to hip ratio
WHtR	Waist to height ratio
Wt	Weight
VLDL-C	Very low density lipoprotein cholesterol

# CHAPTER ONE

## 1.0 INTRODUCTION

### 1.1 Background of the Study

The study of the shape of the human body always arouses interest due to its clinical and aesthetics purpose that is related to variations of human growth and its usefulness for the assessment of other anthropometric variables (Carter and Heath, 1990; Carter, 2003; Adebisi, 2008; Sebbane *et al.*, 2009). Scientists are eager to quantify and compare the infinite variations of human morphology and develop many systems to classify these physical variations (Sebbane *et al.*, 2009). Since the sixties, the calculation of the somatotype represent one of the methods mostly used for quantification of the body form, independent of the size. The use of adequate anthropometric measurements made it possible to create a method needed to evaluate the total image of the shape of the human body and to study its plasticity (Carter and Heath, 1990; Carter, 2003).

Anthropometry is the hallmark technique (Leaper, 2004) that deals with the study of body proportion and absolute dimensions that vary widely with age and sex within and between racial groups (Hunt and Mueller, 1994). Over the centuries, there have been remarkable changes in anthropometric measurements due to geographical, cultural, genetic and environmental factors (Leif, 2003) as well as worldwide mingling of races (Webster, 2008).

The morphological and structural differences among human beings are unique and that is why no two humans are alike in body form. Even the identical twins (monozygotic) can be identified from each other although they develop from a single ovum and share exactly similar genetic information (Singh, 2007). These large differences in body form, morphology and physique in humans form the basis for any attempt at classification and analysis of human physique. The somatotype, as conceived of a biological tag to the individual should



remain unaltered throughout life but in the absence of grossly disturbing pathology and malnutrition (Singh, 2007). Humans with different somatotypes demonstrate unique performance capacities during exercise and physical training (Cureton, 1947; Tanner *et al.*, 1969; Tanner and Whitehouse, 1976). As explained by Ramos-Jiménez *et al.* (2017), the concepts of body image, body shape and body composition should not be confounded and must be studied together.

Adult morphological features are determined by the combined influence of genetic and environmental factors and of these environmental factors, both social and cultural factors determine to a major extent the biological interrelationships between growth, health, fertility and morbidity pattern (Singh, 2007). Morphological characteristics of a person are associated not only with the body proportions, but also with features of functional systems of the organism (Martirosov *et al.*, 2006; Radysh *et al.*, 2007; Oleinik, 2013) and are determined even at the biochemical level (Shutova and Potapov, 2009).

The assessment of a patient's bodily structure has now become an important part of clinical investigation in many medical fields. In general medicine, body build has been found to have a significant relationship with such conditions as peptic ulcer and gall-bladder disease (Draper, 1944). In the field of psychiatry, body build has been shown to have an association with the type of mental illness, to influence its progress and to determine response to treatment. It has been shown that the linear type of body build in particular, has an affinity with schizophrenia, whereas the broad or lateral types of body build tend to be associated more with manic depressive psychosis (Kretschmer, 1921). A broad type of body build in schizophrenia has been found to be associated with a shorter duration of illness than in those with narrow types of structure (Tucker and Lessa, 1940a) and is considered to be a factor of good prognostic significance in the insulin treatment of schizophrenia (Akabaliev *et al.*, 2011).

The study of human constitution as one of its major objectives the discovery of stable organic correlations (integrated biological relations between the morphological, psychological and pathological characteristics of the individual) and eventually the precise numerical measurement of such correlations (Tucker and Lessa, 1940a).

The somatotype corresponds to the estimation of the corporeal shape and its composition, which is expressed in three numbers that correspond to the components obtained during the embryological development: endoderm, mesoderm and ectoderm, altogether corresponding to the morphological characteristics of the subject as a whole (Carter, 1996). The somatotype is a morphological characteristic of the body built, that is “a phenotypic entity capable of changes with growth, aging, exercise and nutrition” (Carter, 1996). It is defined by three components: endomorphic, referred to relative adiposity; mesomorphic related to the musculo-skeletal magnitude and ectomorphic based on physical thinness and or its linearity. It is important to recognize that somatotype describes the body in general and does not answer more specific questions related to the specific dimensions of the body (Carter, 1996).

Adolescence is the period in which the somatotype exhibits significant changes. When men begin puberty, their somatotype increases in mesomorphy and ectomorphy but decreases in endomorphy, because the amount of subcutaneous fat tissue of the upper and lower limbs and the lower and dorsal region of the thorax is reduced, unlike women, who increase their endomorphy. Variables such as nutrition and physical activity are critical in modifying each somatotype component (Carter *et al.*, 1997; Mladenova *et al.*, 2010).

The Heath-Carter method derived from modifications and simplifications of Sheldon's system, is designed to provide an objective phenotype method. It has become the most widely used somatotype method. Heath and Carter (1996) gave the method of somatotyping human physique by giving different ratings according to anthropometric measurements. They

defined somatotype as ‘present morphological confirmation’, expressed in a three numeral rating of primary components of physique that identify individual features of morphology and body composition (Carter and Heath, 1990). Biotypology using the Heath - Carter anthropometric somatotype is one of the most widely used methods for the selection of gifted and talented people for sports (Amigoa *et al.*, 2009; Sterkowicz *et al.*, 2019).

Children are typically born with many of the requisite components of ideal cardiovascular health. They generally have healthy blood pressure, lipid and glucose levels and they do not smoke and have the potential for developing an ideal body weight, healthy dietary and physical activity practices. That is, numerous studies have indicated that loss of cardiovascular health often begins in childhood and progresses over the life course (Berenson and Srivivasan, 2005). Those who reach midlife with a favorable constellation of cardiovascular risk factors (example, systolic blood pressure  $\leq 120$  mm Hg, diastolic blood pressure  $\leq 80$  mm Hg, total cholesterol  $< 200$  mg/dL (5.17 mmol/L), body mass index  $< 25$  kg/m<sup>2</sup>, non - smoker and non - diabetic) experience significantly lower lifetime remaining risk for developing cardiovascular disease (CVD) and have increased longevity relative to individuals with unfavorable levels of such factors (Daviglus *et al.*, 2004; Lloyd-Jones *et al.*, 2006; Lloyd-Jones *et al.*, 2007; Lloyd-Jones *et al.*, 2010; Folsom *et al.*, 2011).

However, the vast majority of middle-aged US adults have poor cardiovascular health (Folsom *et al.*, 2011) and once risk factors are elevated, they are difficult to ameliorate (Lloyd-Jones *et al.*, 2010). As such, primordial prevention of CVD or preventing the development of CVD risk factors in the first place has increasingly become of interest to cardiovascular epidemiologists and clinicians to more effectively reduce the burden of CVD in the population (Lloyd-Jones *et al.*, 2010). Because many CVD risk factors such as obesity and atherosclerosis develop early in life (Berenson and Srivivasan, 2005), childhood is a life stage particularly amenable to primordial prevention efforts because cardiovascular risk is not

yet well established. Moreover, childhood may serve as a sensitive period of development whereby exposures occurring early in life, including health-promoting experiences, may impact physical health over the life course (Ben-Shlomo and Kuh, 2002; Shonkoff *et al.*, 2009). Therefore, the identification of childhood factors associated with adult favourable cardiovascular risk may be of great utility in furthering primordial prevention work.

Cardiovascular diseases (CVDs) are responsible for nearly half of all deaths in the developed world and are expected to be the world's number one cause of death or disability by the year 2020 (Gaziano, 2005; Gaziano, 2007; WHO, 2009). It is estimated that there are 2 million patients with coronary disease and that the annual death toll caused by cardiovascular disease increases by approximately ninety to one thousand cases (Onat, 2009). The most important step in improving prevention against cardiovascular disease is altering the habits into ingrained lifestyle. Such a change would be more efficient and cost-effective (Parker and Assaf, 2005). On the other hand if the risk factors are well known, a significant amount of deaths can be prevented by identification of individuals with these factors by implementing preventative programmes accordingly (Arıkan *et al.*, 2009; Ural, 2011).

Early detection, education and life style changes can lead to a reduction of CVDs by increasing awareness of not only what causes CVDs but also of what can prevent them through implementation of healthy habits such as diet, exercise, smoking cessation and other factors (Vale, 2000; Check *et al.*, 2004). The behaviour of individuals regarding healthy lifestyle choices is most probably linked to their "health beliefs, including their perceptions of susceptibility, severity, benefits and barriers" (Hasse *et al.*, 2004; Gilski, 2005; Glanz and Bishop, 2010). According to some studies, awareness on risk factors is not only limited among lay people, but also it depends on the nature of the society under investigation (Jafary *et al.*, 2005; D'Agostino *et al.*, 2008; Ton *et al.*, 2011).

Accordingly, an individual value considered pathologic may be within the physiologic range for that particular body habitus. Since the physical constitution is predetermined to a large degree, it is reasonable to suppose, without postulating a causal relationship that the chemical attributes likewise seem to be predetermined genetically. Hence, an attempt to associate physique and serum lipids has a certain theoretic justification. Cholesterol is important and necessary for mammals, but its high levels in the blood can damage arteries and are potentially linked to diseases such as those associated with the cardiovascular system (Pearson *et al.*, 2003). The determination of cholesterol levels in the blood is essential in the diagnosis and medical management of heart disease such as atherosclerosis, nephrosis, diabetes mellitus, myxedema, obstructive jaundice and cholelithiasis (Levy, 1981).

### **1.2 Statement of the Research Problem**

Several studies in adults suggest that a pattern of exaggerated central body fat deposition (that is, body fat situated in the trunk region), rather than general adiposity (that is, body fat representative of both central and peripheral regions) is more strongly associated with metabolic derangements, cardiovascular risk factors and CVD. Central fat deposition in youth and its relation to cardiovascular risk factors and subsequent development of CVD have received little attention. Because the antecedents of these diseases originate in childhood, the identification of these risk factors in youth presents an important opportunity for their primary prevention.

Central or visceral obesity, with the disproportionate increase of adipose tissue surrounding the internal organs, has been associated with abnormal blood sugar, triglycerides, high-density and low-density lipoproteins, total cholesterol and uric acid levels. These body shape alterations - induce atherosclerosis, high blood pressure and insulin resistance, leading to an end result of increased risk for cardiovascular disease, diabetes mellitus, gout, stroke and other co-morbidities. Metabolic syndrome defined by this cluster of abnormalities starts to

occur more frequently in younger persons, generating calls for earlier screening and exploration of new anthropometric assessment technologies by health care professionals.

The studies in the past relating physique and serum lipids have been suggestive of such a relationship. However, the differences in physique classifications and nomenclature have made it virtually impossible to compare the results. The establishment of correlations of significant value between bodily structure and serum lipids such as cholesterol (total and fractions) could lead to the determination of normal lipid values and thus, detection of abnormal lipid metabolism in the lipodystrophies.

Research evidence regarding the relationship of physiological and biochemical profiles with somatotype is scanty or inconsistent, especially among Nigerian youths. This study would show how physiological and biochemical profiles relates with body somatotype. Hypertension and other cardiovascular diseases are on the increase in sub - Saharan Africa and estimates show that by 2030, cardiovascular diseases will bypass communicable diseases including tuberculosis and malaria as the most important cause of morbidity and mortality in the region (WHO, 2015). Understanding the roots of hypertension is therefore of major public health importance as a step towards primary intervention.

### **1.3 Justification of the Study**

Somatotypes can be assessed as a useful tool for description and comparison of populations and for monitoring growth and aging changes. The present study provided useful information regarding relationship between somatotype components and health related physical, physiological and biochemical variables. The findings from this study could help to overcome some of the lifestyle diseases.

The result could provide a useful database for overweight and obesity which might lead to adverse metabolic effects on blood pressure, cholesterol and triglycerides. It could also

provide a baseline data for students of higher institutions on the indication of increased body mass index (BMI). The findings of the study could also provide guidance to physical educationist and coaches to analyse the body type or somatotype components of the athletes which help to adopt the proper training schedule. The results could also help manufacturers in the garments industry in the accurate determination of the appropriate sizes for adolescents in terms of clothes fitting.

Body mass index and waist circumference are measured in population studies because they can predict Cardiovascular disease (CVD) risk factors. Measurements of cholesterol are used primarily in the diagnosis and treatment of disorders involving excess cholesterol in the blood, lipid and lipoprotein metabolic disorders. Total serum cholesterol analysis has proven useful in the diagnosis of hyperlipoproteinaemia, atherosclerosis, hepatic and thyroid diseases. Total and high density lipoprotein (HDL) cholesterol, in conjunction with a triglyceride determination, provide valuable information for the prediction of coronary artery disease (CAD).

#### **1.4 Aim and Objectives of the Study**

##### **1.4.1 Aim**

The aim of this study was to investigate the relationship between somatotypes and cardiovascular disease risk factors in Nigerian undergraduate students.

##### **1.4.2 Objectives**

The objectives of the present study were to:

- i determine age and sex related variations of somatotype in a cross sectional sample of apparently healthy Nigerian students;
- ii examine the association of somatotype with blood pressure according to age;
- iii investigate the relationship between somatotype, blood pressure and biochemical variables;

- iv evaluate body shape and composition and assess their relationship;
- v determine the relationship between BMI and body fat from skinfold measurements in Nigerian students;
- vi determine the correlations between the levels of serum cholesterol, its fractions and the concentration of triglycerides and some anthropometric features and indices, particularly between the serum lipid profile and somatotype as characterized by endomorphy, ectomorphy and mesomorphy;
- vii assess the relationship in selected anthropometric measurements (BMI and Waist Hip Ratio) among Nigerian students of higher institutions.

### **1.5 Research Hypotheses**

The study test the following hypotheses that:

Alternative hypothesis ( $H_1$ )

- i. there will be relationship between age and sex of subjects anthropometric parameters and somatotype;
- ii. the cardiovascular risk factors and somatotype can predict the health status of an individual;
- iii. there will be relationship among somatotype components on selected physiological and biochemical variables;
- iv. the level of education will influence somatotype and cardiovascular risk factors.

Null hypothesis ( $H_0$ )

- i. there would be no effect of somatotype on biochemical indices in the population.

### **1.6 Limitations of the Study**

This limitation was considered while interpreting the results:



Although percent body fat can be assessed more objectively with greater precision in the use of such sensitive methods such as, underwater weighing or hydrostatic, ultrasonography, Computer Tomography Scan, Lunar Prodigy DXA scanner and Magnetic Resonance Imaging; only skinfold measurement was used to estimate percent body fat in this study because it is simple and easy to use.

## CHAPTER TWO

### 2.0 LITERATURE REVIEW

#### 2.1 Preamble

Monitoring of various anthropometric parameters is due to its non invasiveness and relatively low demands on financial and technical needs, advisable for extensive epidemiological studies. The monitoring and use of anthropometric parameters and derived indices in clinical practice help us to observe their relationships to the function of the organism, with other indices characterizing health hazards (Ivana *et al.*, 2009). The somatotype has proven to be closely related to the physical condition and the identification of the physical characteristics of athletes (Carlson *et al.*, 1994; Carter *et al.*, 2005) with health risk factors (Makgae *et al.*, 2007; Baltadjiev *et al.*, 2009), as well as the body composition (Slaughter and Lohman, 1976). Moreover, many investigators have recognized the value of the somatotype characteristics in population from developing countries: maturity and sexual dimorphism (Toro *et al.*, 1983; Marrodan, 1991; Almagia *et al.*, 1996; Gomez *et al.*, 2002; Mladenova *et al.*, 2010).

This life course perspective is crucial for a better understanding of the health consequences of overweight and obesity and for development of effective prevention strategies. Hereby, anthropometrical characterization of developing and aging populations in terms of body types and of transitions between them constitutes a novel option to investigate onset and progression of obesity and other civilization diseases (Loeffler *et al.*, 2018).

Somatotyping is a unique method for the classification of human physique which was first invented by Sheldon *et al.* (1940) and later modified by Heath and Carter in 1967 (Shilpa and Reeta, 2014). It reflects an overall outlook of the body and conveys a meaning of the totality of morphological features of the human body. Harrison *et al.* (1976) are of the

opinion that certain relationship must exist between body build and endocrine functions and metabolism. To further support this logic they emphasised that it is highly unlikely that persons of different builds have similar basal or habitual functions. Whether physique, constitution and disease are interlinked to each other or not has always been a point of great interest.

Various factors are responsible for the ultimate body shape and size. Somatotype studies from around the world reflect extensive variations suggesting differences due to genetics (Singh and Singh, 2000), sex (Tanner, 1962; Parizkova and Carter, 1976; Carter and Parizkova, 1978), nutrition (Malik *et al.*, 1986), physical activity (Carter, 1970; Parizkova, 1970; Withers *et al.*, 1987) and ageing (Zuk, 1958; Heath and Carter, 1971; Walker, 1978). Handal *et al.* (1995) emphasised that the differences in physique between populations in different regions are of importance, especially to underlie the cultural differences between populations.

Despres (2012) observed that a sedentary lifestyle is a risk factor for a number of diseases that become more prevalent with age in both genders. It was rightly observed from studies that the incidence of fat accumulation due to inactive lifestyle and other tendencies such as positive energy balance and excessive gestational weight gain affect quality of life with serious health implications of high blood pressure or hypertension, ischaemic heart disease, certain cancers, type two diabetes mellitus and cardiovascular disease. Longkumer (2016) observed that the somatotype components changed significantly with age among girls and there were remarkable sex differences with regard to all the three components, more so within the endomorphy and ectomorphy components. There was also significant association between somatotype components and BMI depicting that increase in endomorphy and mesomorphy was a risk factor having predisposition toward certain diseases.

Cardiovascular disease remains the leading cause of death and disability in the United States despite advances in treatment and secondary prevention (Mozaffarian *et al.*, 2008). Recognizing that CVD has roots in early life, the American Academy of Paediatrics now recommends that cardiovascular screening, prevention and treatment should begin in childhood (Daniels and Greer, 2008). Interventions to alter CVD risk in childhood require an understanding of the early social contexts that shape risk trajectories (Shonkoff *et al.*, 2009).

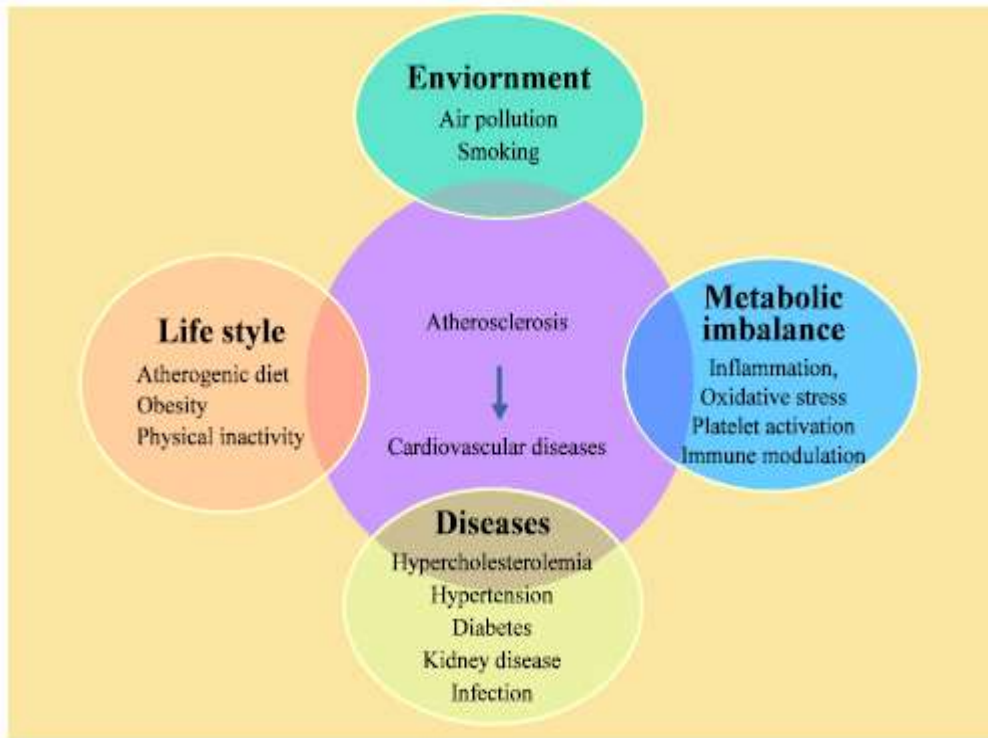
Cardiovascular disease is among the most killer diseases. This disease is more serious than cancer (Bakari *et al.*, 2007). If all forms of CVDs are eliminated, total life expectancy could increase by about ten years while if all forms of cancer are eliminated life expectancy would increase by only three years (Nwabunike, 1992; Brambilla and Pietrobelli, 2007). CVD is not a single disease but is a general term given to more than twenty different diseases of the heart and its blood vessels like hypertension, ischaemic heart disease, coronary heart disease, cerebrovascular disease (stroke), peripheral vascular disease, heart failure and atherosclerosis (Hampton, 1983; Ogundipe, 2006; Roy, 2006; Yang *et al.*, 2006).

The World Health Organization defines obesity as a condition with excessive fat accumulation in the body, to the extent that health and well being are adversely affected (WHO, 2002). Hypertension in children and adolescents is defined as average systolic and/or diastolic blood pressure (BP) levels above or equal to the 95th percentile for age, gender and height, taken on three or more separate occasions at least a week apart (NCEP, 1995). Various lipid/lipoprotein abnormalities have been observed in obese individuals, including elevated cholesterol, triglycerides and reduced high-density lipoprotein (HDL) cholesterol levels (Depres, 2012). Of these indicators, changes in triglyceride and HDL cholesterol levels are most consistent and pronounced (Depres, 2012). These adverse lipid/lipoprotein profiles

in obese individuals are important, because they may be responsible for their increased risk for cardiovascular disease (WHO, 2002).

Atherosclerosis, the building up of fatty plaque materials in the layer of blood vessels and the most important disease of larger and medium sized arteries in the western world, is the underlying factor of most of the CVDs (Hampton, 1983; Kalichman *et al.*, 2006). When atherosclerosis plaque blocks one or more of the heart coronary blood vessels, the diagnosis is coronary heart disease, the major form of CVD (Figure 2.1). In this disease process, a blood clot is often formed in the narrowed coronary artery, blocking the blood supply to the part of the heart muscle supplied by the artery and this causes heart attack or myocardial infarction (MI). Atherosclerosis can also block blood vessels in the brain that leads to stroke or legs that lead to peripheral artery disease, PAD (for instance, gangrene).

Singh (2007) observed that the presence of at least three or more cardiovascular disease risk factors (overweight, central obesity, hypertension either for systolic blood pressure or diastolic blood pressure, hyperglycaemia, hyperlipidaemia and hypertriglyceridaemia) has been considered as a case of cardiovascular disease risk (Table 2.1).



**Figure 2.1:** Risk factors of atherosclerosis. Group of interrelated risk factors influence the development of atherosclerosis including life style, diseases of metabolic disturbances and environment (Sempos *et al.*, 1993).

Table 2.1: Cardiovascular disease risk factor measurements and their respective cut-off points

<b>Risk factors</b>	<b>Name of measurements</b>	<b>Cut – off points</b>	<b>References</b>
Overweight	Body Mass Index (BMI)	$\geq 25 \text{ kg/m}^2$	WHO (2002)
Central adiposity	Waist Circumference (WC)	> 90 cm	Tan <i>et al.</i> , (2004)
Hypertension	Systolic Blood pressure (SBP)	$\geq 140 \text{ mm Hg}$	Wilson <i>et al.</i> , (1998)
	Diastolic Blood pressure (DBP)	$\geq 90 \text{ mm Hg}$	
Hyperglyceamia	Random Blood Glucose	> 126 mg/ dL	ADA (2004)
Hyperlipidemia	Total Cholesterol in Blood	> 200 mg/ dL	NCEP (2001)
Hypertriglyceridemia	Total Triglycerides in Blood	> 150 mg/ dL	NCEP (2001)

Adopted from Arupendra and Subrata, 2008 (Biological Anthropology Unit, India)

High blood pressure, also called hypertension is a sustained elevation of the systemic arterial pressure. It is both a risk factor for coronary heart disease (CHD) and disease by itself. Blood pressure is usually categorised as follows:

(a) Normal category in which systolic blood pressure (SBP) is less than 130 mmHg and diastolic blood pressure (DBP) is less than 85 mmHg.

(b) High normal category in which systolic blood pressure is between 130 mmHg and 139 mmHg and diastolic blood pressure is between 85 mmHg and 89 mmHg.

(c) High or severe category in which systolic blood pressure is 140 mmHg and higher and diastolic blood pressure is 90 mmHg and higher (United States Department of Health and Human Services, 1996; Heyward, 1998; Dudeja *et al.*, 2001; Musa *et al.*, 2001; Varo *et al.*, 2003; Ganong, 2005).

There are millions of people who have high blood pressure, one fourth of whom do not even know that they are hypertensive (asymptomatic). There are many more that are at high risk of developing high blood pressure. High blood pressure is a killer disease because it does not give any warning. It increases the risk of heart disease, stroke and kidney failure. If it is not detected and treated, it can cause: Enlargement of the heart, leading to heart failure; formation of small blisters called aneurism in the blood vessels of the brain, leading to stroke; hardening of arteries faster throughout the body, leading to stroke, heart attack or kidney failure and narrowing of blood vessels in the kidney, leading to kidney failure (Singh, 2007).

There are several factors associated with the incidence of high blood pressure. Some of these factors like heredity, family background, age and sex are non modifiable. There are factors that are modifiable like diet, exercise, sleep, consumption of fatty diet, obesity, overweight and cigarette smoking (Singh, 2007).



## **2.2 Somatotypes and Blood Pressure**

According to Badenhorst *et al.* (2003) somatotype and elevated blood pressure showed associations which indicated that the blood pressure of the endomorphic boys was the highest which increased with an increase in physical activity levels. An increase in physical activity did not lower the resting blood pressure values of endomorphic boys. Relationships between cardiovascular risk factors and Heath-Carter anthropometric somatotype components were investigated by Malina *et al.* (1997) in 642 healthy adults. Risk factors included SBP and DBP, fasting glycaemia and blood lipids. Correlations between risk factors and each somatotype components were calculated after controlling for the effects of the other two somatotype components.

According to a study by Herrera *et al.* (2004), the correlation between ectomorphy and both SBP and DBP showed that as ectomorphy increased the blood pressure decreased, except for the oldest age group. Endomorphy and mesomorphy showed a stable correlation pattern with blood pressure in males indicating a neutral stance of these components in determining the blood pressure, while in females this pattern was more irregular and less consistent.

Kalichman *et al.* (2004) observed that individuals of robust physique (with high endomorphy and mesomorphy) showed high mean values of SBP and DBP, whereas the smallest persons had the lowest blood pressure values. They also suggested the existence of common physiological mechanisms in the development of body physique and blood pressure regulation with the possibility of the involvement of pleiotropic genetic and/or epigenetic mechanisms in this regulation.

## **2.3 Somatotypes and Cardiovascular Disease (CVD)**

Physique or the overall configuration of the body, may also play an important role in health and disease (Damon, 1970; Bailey, 1985). Historically, physique has been assessed most

often using the somatotype, a three component index which includes endomorphy, mesomorphy and ectomorphy (Bailey, 1985). Early methods of somatotyping, such as those of Sheldon *et al.* (1940) and Parnell (1958) relied on the analysis of photographs, while the Heath-Carter (Carter and Heath, 1990) anthropometric method relies completely on anthropometric dimensions. The various types of somatotype (Sheldon, Parnell, Carter and Heath protocols) have been studied in relation to coronary heart disease (Damon *et al.*, 1969; Smit *et al.*, 1979) and risk factors for CHD such as serum lipid levels (Malina *et al.*, 1997; Katzmarzyk *et al.*, 1998), blood glucose levels (Fredman, 1972; Fredman, 1974) and blood pressure (Seltzer, 1966; Malina *et al.*, 1997).

Mueller and Joos (1985), indicated that the Sheldonian photoscopic somatotype was related to the degree of central adiposity in adult males. Sheldon *et al.* (1954) used photographs of 824 men in the Atlas of Men which indicated that android obese men were rated higher on mesomorphy and lower on ectomorphy than gynoid obese men. Among youth, the Heath-Carter (Carter and Heath, 1990) anthropometric somatotype was related to relative fat patterning, those with a centralized subcutaneous fat distribution were higher in endomorphy and mesomorphy and lower in ectomorphy (Carter and Heath, 1990).

According to Manna *et al.* (2010) that investigated the effect of training on selected anthropometric, physiological and biochemical variables of elite field hockey players. A total of 30 Indian male field hockey players (23 - 30 years) volunteered for the study. A significant increase ( $P < 0.05$ ) in back and hand grip strength, serum level of urea, uric acid and HDL-C; and a significant decrease ( $P < 0.05$ ) in body fat, sub-maximal exercise heart rate and recovery heart rate, haemoglobin, total cholesterol, triglyceride and LDL-C were noted. No significant change was noted in stature, body mass, HR max, resting heart rate,  $VO_2$  max and anaerobic power of the players after the training.

Monitoring the trends in CVD risk factors in populations enhances our understanding of mortality trends, since these trends are dynamic and are influenced by the health environment, personal health behaviors and medical care. According to a previous report, 44% of the decline in CVD mortality among US adults between 1980 and 2000 was due to changes in CVD risk factors (Ford *et al.*, 2007). Specifically, reductions in total cholesterol, blood pressure, smoking and physical inactivity contributed 24%, 20%, 12%, and 5% respectively, to the decreased CVD mortality rate (Ford *et al.*, 2007). However, the national obesity epidemic has been associated with increased prevalence of diabetes and elevations in blood pressure and lipid levels (NIH, 1998; Despres *et al.*, 2008). Changes in eating patterns have also been demonstrated (Lichtenstein *et al.*, 2006). There is already evidence that the prevalence of hypertension is increasing in adults and children (Hajjar and Kotchen, 2003; Muntner *et al.*, 2004). Such empirical data on population risk factor levels and trends are required for appropriate public health and medical policy.

Cardiovascular disease occurs earlier and with greater frequency in people with diabetes, a finding particularly striking in women (Nathan, 1993). These observations are especially true for young adults with type I diabetes, in whom coronary artery disease is increased 10-fold or greater (Laing *et al.*, 2003). Much of the risk for CAD in type 1 diabetes lies in the presence and severity of atherosclerosis and its risk factors (dyslipidaemia/ hyperlipidaemia). Blood flow dynamics and arteriosclerosis or arterial stiffening, which are measured in a variety of ways, are also important risk factors for cardiovascular events and mortality (Laurent, 2001).

Beyond increased weight, abdominal obesity is a key feature in cardiovascular risk assessment. It is an essential component of the metabolic syndrome (MetS), which is a cluster of metabolic and clinical abnormalities associated with visceral fat hypertrophy and insulin resistance (Alberti *et al.*, 2006). Lipid abnormalities associated with MetS typically include a high level of plasma triglycerides (TG) and a low high density lipoprotein (HDL)-cholesterol

plasma level. MetS has also been associated with HDL and low-density lipoprotein (LDL) subfraction distribution changes characterised by an increased content of small, dense LDL and HDL (Hulthe *et al.*, 2000; Pascot and Lemieux, 2001; Solymoss *et al.*, 2004). Few studies have specifically analysed the relationship between abdominal obesity and the distribution of HDL and LDL subfractions (Goff Jr *et al.*, 2005; Palaniappan *et al.*, 2007; Medina-Urrutia *et al.*, 2008).

Malina *et al.* (1997) observed that correlations were generally low and at best moderate, with significant correlations ranging from  $-0.23$  to  $+0.23$  in males and  $-0.20$  to  $+0.30$  in females. The relationships were stronger in the older group, 40–49 years, but the pattern of correlations was different in men and women. Endomorphy tended to be positively related to risk factors in older females, whereas ectomorphy tended to be negatively related to risk factors in older males. Comparison of somatotypes of individuals at the extremes of the distributions for each risk factor (upper and lower tertiles) were generally consistent with the direction of the correlations. For each cardiovascular risk, those with a poorer profile tended to be more endomorphic and mesomorphic and less ectomorphic than those with a better profile, who were more ectomorphic and less endomorphic and mesomorphic. The association was more apparent in males than in females and more so in those 40–49 years of age than in the younger age group (Malina *et al.*, 1997). Although the correlations suggest that body type is weakly associated with common cardiovascular risk factors in healthy men and women, somatotype associations are more apparent at the extremes of the distributions of specific risk factors.

## **2.4 Etiology of Cardiovascular Disease**

Cardiovascular disease is a multifactorial disease, with a number of modifiable physiological risk factors such as high blood pressure (Kannel *et al.*, 1980), high total cholesterol (Thomas

*et al.*, 1966), high blood glucose (Kannel and McGee, 1979) and high BMI (Kannel *et al.*, 1967). Also, modifiable behavioural risk factor play a causal role and include increased alcohol use (Friedman and Kimball, 1986), tobacco smoking and unhealthy diet (Doyle *et al.*, 1962) and physical inactivity (Kannel *et al.*, 1967). Finally, familial aggregation of CVD suggests evidence of a genetic predisposition (Lloyd-Jones *et al.*, 2004) and twin studies have reported about 40% heritability of CHD mortality (Zdravkovic *et al.*, 2002).

#### **2.4.1 Cardiovascular disease has been recognised as a global priority**

Cardiovascular diseases are leading cause of morbidity and mortality throughout the world. Largely diseases of lifestyle and affluence, they account for about 50% of deaths in many developed countries (Gwatkin and Guillot, 1999; WHO, 2007b). For example, in the United States and Western European countries, CHD is the leading cause of death, also accounting for approximately 50% of deaths. In the United States alone, the prevalence of CVD in the total population was estimated to be over 71 million in 2003 (NHLBI, 1997; 2006; Nichols *et al.*, 2012).

Cardiovascular disease has modifiable and non-modifiable risk factors. Non-modifiable risk factors such as age and sex are strongly associated with CVDs. The rise of CVD is also attributed to a number of modifiable risk factors. The leading global cardiovascular risk factors for CVD mortality are high blood pressure (accountable for 13% of deaths globally), tobacco use (9%), high blood glucose (6%), physical inactivity (6%), overweight and obesity (5%) (WHO, 2008). These risk factors are responsible for raising the risk of CVDs such as CHD, stroke and IHD. The high prevalence of risk factors for heart diseases and stroke among young and middle-aged adults, combined with the ageing population worldwide, suggest that the prevention and management of CVDs will continue to be a public health priority both in developing and developed countries (WHO, 2008).

Because of high prevalence in the growing population, the United Nation held its first high-level meeting of the General Assembly on chronic non-communicable diseases, mainly CVDs, cancer, diabetes and chronic respiratory disease in New York, September 2010 (Beaglehole and Horton, 2010). This meeting made CVD a global priority among heads of states and governments and extended universal access to essential medicines and technologies for the secondary prevention of CVD (Alleyne *et al.*, 2010). The past declarations and recent global strategies provide a welcome sign that the international community is increasingly aware of the importance of CVD. Moreover, the World Bank (2007) recognised the effects of CVD in the most deprived areas of the world and acknowledged CVD as a development priority.

The vast majority of cardiovascular mortality might be prevented through simple interventions such as smoking cessation, improved diet and increased exercise. In the INTERHEART case control study, Yusuf *et al.* (2004) identified nine modifiable cardiovascular risk factors that accounted for over 90% of the contribution to risk of an initial acute myocardial infarction for young men and women worldwide, these includes:cholesterol, smoking, blood pressure, diabetes, abdominal obesity (waist-to-hip ratio), psychosocial factors (including depression and stress), consumption of fruits and vegetables, drinking of alcohol and physical activity.

Chow *et al.* (2010) surveyed about 19, 000 patients from 41 countries who had undergone percutaneous coronary intervention after myocardial infarction and who had answered questions about their lifestyle. They showed that patients who continued to smoke and did not adhere to diet and exercise regimens were 3.8 times more likely to suffer a myocardial infarction, stroke, or death within 6 months than were non-smokers who modified their diet and increased exercise, considering that both groups complied with their medications.

Prevention and management of CVDs will continue to be a public health priority both in developing and developed countries, but effective preventive care and a healthy lifestyle could lead to reduced prevalence of cardiovascular risk factors and therefore, CVD itself. Current evidence has identified modifiable CVRFs and we know how evidence has translated into population effects, such as sex, socioeconomic status, region and country.

Taking obesity as an example, more than 1.4 billion adults, aged 20 and older, were overweight globally (WHO, 2013). Of these overweight adults, over 200 million men and nearly 300 million women were obese (WHO, 2013). In all WHO regions, women were more likely to be obese than men. In the WHO regions for Africa, Eastern Mediterranean and South East Asia, women had approximately twice as much the obesity prevalence as men. The prevalence of overweight increases with income level of countries up to upper middle income levels. The prevalence of overweight in high income and upper middle income countries was more than twice as high as low and lower middle income countries. For obesity, the difference was more than triples from 7% obesity in both sexes in lower middle income countries to 24% in upper middle income countries. Women's obesity was significantly higher than men, with the exception of high income countries where it was similar. In low and lower middle income countries, obesity among women was approximately double that among men (Global Health Observatory, 2013).

#### **2.4.2 Cardiovascular risk factors**

A cardiovascular risk factor is a condition that is associated with an increased risk of developing CVD. The association is almost a statistical one and so the fact that a particular person has a particular factor merely increases the probability of developing a certain type of cardiovascular disease it does not mean that he or she is certain to develop heart or blood vessel disease. Conversely, the fact that an individual does not have a particular

cardiovascular risk factor (or for that matter, any of the known cardiovascular risk factors) does not guarantee protection against heart disease. Even today, a number of individuals who have heart attacks or strokes have none of the identified risk factors.

a) Risk factors that cannot be changed

Age, gender and heredity

b) Risk factors that can be changed

High blood pressure, elevated serum cholesterol, lipoprotein, cigarette smoking, obesity, glucose intolerance, diabetes, fibrinogen, left ventricular hypertrophy, cocaine, behavioural factors (stress, Type A)

c) Protective factors

HDL cholesterol, exercise, estrogen, moderate alcohol intake.

## **2.5 Lipids Measurements**

The processes involved in the formation and progression (sometime even regression) of atherosclerotic lesions are complex and still not completely known, but ongoing research is constantly refining our understanding. Nevertheless, it is generally accepted that elevated blood lipids play an important role in the genesis of these lesions (Castelli *et al.*, 1986; Hokanson and Austin, 1996), although many heart attacks occur in persons with "normal" blood lipid levels. However, clinical trials with the so called "statin" group of drugs have shown that reducing blood lipid levels decreases the risk for coronary events. The assessment of blood lipid levels is therefore, an important component of risk factor monitoring.

Lipid transport in the circulation occurs in the form of lipoproteins (protein shells surrounding a lipid core). Lipoproteins are classified according to their source, composition and physiological action. The two types considered to be most important for cardiovascular



risk assessment are low density lipoproteins (LDL) and high density lipoproteins (HDL). They are involved in the transport of cholesterol to and away from the body tissues, which led to the suggestion that the ratio LDL/HDL is an important indicator for cardiovascular risk. Besides cholesterol, triglycerides are considered to play an independent role in the formation of atherosclerotic lesions. In a recent review of the epidemiological, clinical, cellular and molecular evidence of the role of triglycerides in atherogenicity, Sprecher (1998) comes to the conclusion that high levels of triglycerides are an important risk factor, especially for mild cases of atherosclerotic lesions. Fasting triglycerides or the postprandial rise of triglycerides in response to a test meal have been found to be risk factors.

Established methods exist for the direct measurement of total cholesterol (TC), HDL and triglycerides (TG), either from plasma or serum samples. There are now also methods available for the direct measurement of the LDL subfraction, but it is still frequently estimated from TC, HDL and fasting triglycerides by the Friedewald formula (Sprecher, 1998). The review of the methods used in the determination of total cholesterol and HDL was prepared based largely on the experience from the *WHO MONICA Project* (Ferrario *et al.*, 1999). The measurement of triglyceride levels was not part of the *WHO MONICA Project*. Therefore, no international comparisons were made as was done for the total cholesterol and HDL. However, some centres have collected blood from fasting subjects and measured triglycerides (Ferrario *et al.*, 1999).

### **2.5.1 Seasonal variation in lipid measurements**

Several studies have shown that total cholesterol levels are increased in winter (Bleiler *et al.*, 1963; Robinson *et al.*, 1992; Frohlich *et al.*, 1997). However, little is known about the reasons for the seasonal variation, and the variation may differ between countries. The difference in total cholesterol levels between June and December has been reported to be around 0.19 mmol/L (Gordon *et al.*, 1987; 1988). The change in season does not seem to

affect significantly HDL cholesterol (Cooper *et al.*, 1992). Several studies have suggested that level of triglycerides are increased during winter months (Gordon *et al.*, 1988; Woodhouse *et al.*, 1993; Frohlich *et al.*, 1997).

In the *WHO MONICA Project*, the time of year of the cholesterol measurements was not standardized between the populations. However, to avoid bias in trend estimation in cholesterol levels that may be caused by seasonal variation, all surveys in a population were supposed to take place at the same time of the year. When the blood samples were collected throughout the entire year, it was recommended to keep the proportion of subjects from of each age and sex group equal throughout the period (WHO MONICA Project, 1998). In 13 populations a seasonal change may have affected the trends in cholesterol levels. In these population the seasonal difference between two surveys is at least 2 months which can cause 0.1 mmol/L difference in total cholesterol levels (Ferrario *et al.*, 1999).

### **2.5.2 Daily variation and fasting status in lipid measurements**

The effect of time of day is estimated to be about 2.5% (coefficient of variation) on total cholesterol and 4.5% on HDL cholesterol (Demacker *et al.*, 1982). Daily variations in cholesterol are detectable if large fluctuations of triglycerides occur during the day (Cooper *et al.*, 1988). In most subjects, the time of blood drawing does not seem to be important. For triglyceride concentration the maximum daily rhythmic variation could reach 63% (Rivera-Coll *et al.*, 1994).

Total cholesterol level is not significantly influenced by fasting, but HDL cholesterol level decreases transiently postprandially (Cooper *et al.*, 1988). Therefore fasting is usually not considered essential before total cholesterol measurements, but 12-14 hours fasting is often recommended for HDL cholesterol measurements (Cooper *et al.*, 1992). The fasting time may make a difference in cholesterol levels for a small percentage of people (Cohn *et al.*,

1988). Triglycerides are influenced by fasting, although their daily fluctuation does not have a simple relation with intake of meals (Cooper *et al.*, 1992). The fasting time may make a difference in cholesterol levels for a small percentage of people (Cohn *et al.*, 1998). It is usually recommended that blood for measurement of triglycerides should be collected after at least 12 hours fasting.

### **2.5.3 Position of the subject in lipid measurements**

Several studies reported on the effect of total cholesterol levels caused by the position of the subject during blood drawing (Felding *et al.*, 1980; Kjeldsen *et al.*, 1983; Hagan *et al.*, 1986). There is evidence that change of posture from 30 minutes supine to 30 minutes standing results in a 9.3% increase of total cholesterol concentration (Hagan *et al.*, 1986). Other studies showed that drawing blood after a transition from standing to supine may produced a 4-6% decrease in total cholesterol compared with drawing blood after a transition from standing to sitting (Stoker *et al.*, 1966; Tan *et al.*, 1973). The effect is brought about by haemodilution when assuming supine position or haemoconcentration as a result of assuming the upright position. The sympathetic nervous system may also play some role (Cooper *et al.*, 1992).

In the *WHO MONICA Project*, it is recommended that venipunctures should be carried out with the subject in a sitting position (WHO MONICA Project, 1998). Nevertheless, in five populations the blood was drawn in supine position (Ferrario *et al.*, 1999).

### **2.5.4 Type of tubes**

Traditionally, syringes and glass tubes were used for collecting blood. In the past decades these have been replaced largely with plastic vacuum sealed tubes, which are more expensive but easier to use. The plastic tubes do not break, and they can be frozen to - 80 °C. In recent years, vacuum sealed tubes which include gel have come on the market. In centrifuging, the

gel settles between the serum and cells making the separation of serum easier and providing a higher yield of serum.

According to the instruction of the *WHO MONICA Project*, either 10 ml vacuum sealed tubes or syringes and glass tubes could be used to collect blood. The use of vacuum tubes containing EDTA was recommended if plasma was used for analysis (WHO MONICA Project, 1998). Two thirds of the centres used vacuum sealed containers in the final MONICA surveys.

### **2.5.5 Tourniquet use**

Prolonged venous occlusion produced by tourniquet use is associated with higher cholesterol values compared to results obtained by blood drawing without tourniquet use. Levels can increase by 10-15% after 10 minutes of venous occlusion and 2-5% after 2 minutes. Tourniquet use up to one minute is not associated with any significant increase in cholesterol levels (Junge *et al.*, 1978). Statland *et al.* (1974) found a significant increase in total cholesterol levels (3.5%) when the tourniquet was used for 3 minutes. According to Naito (1988), well trained technicians need less than one minute for drawing one tube. Tan *et al.* (1973) did not find any significant changes in serum cholesterol concentration when the tourniquet was applied for 0.5 to 1 minute. It has also been demonstrated that the use of a low-pressure tourniquet for 3 minutes does not significantly affect the concentration of serum constituents including total cholesterol (Crombie, 1987). Vacuum sealed containers, if correctly used, should not affect these findings.

In the *WHO MONICA Project*, the use of a tourniquet was to be avoided. If the tourniquet was needed to obtain good flow, it had to be released before the withdrawal of blood (WHO MONICA Project, 1998). In most centres the above instruction was followed and use of tourniquet was limited to less than one minute (Ferrario *et al.*, 1999).

### **2.5.6 Serum or plasma**

The commonly recommended anticoagulant for plasma is disodium ethylene diamine tetraacetate (EDTA). Its use produces a shift of water from red blood cells to plasma, and therefore dilutes the plasma and lowers the concentration of cholesterol. In the 1970s, its use was reported to lower the cholesterol concentration by 3% compared with measurements from serum, and in 1990 a difference of 4.7% was reported. The increase in the difference over the years was explained by a 50% increase in the EDTA concentrations in the commercially manufactured tubes (Laboratory Methods Committee, 1977; Cloey *et al.*, 1990).

In the *WHO MONICA Project*, serum was recommended in preference to plasma. It was, however, recognised that some centres wanted to continue the same methods that had been used in earlier surveys. For most of the populations serum was used. Only 5 populations used plasma, with EDTA, in the final survey (Ferrario *et al.*, 1999).

### **2.5.7 Centrifuging and storing before centrifuging**

The time, temperature and force of centrifuging should be standardised. For serum this concerns also the time allowed for clotting before centrifuging. For serum samples, in general, blood should be allowed to clot at least half an hour and then be centrifuged at room temperature (15-24 °C) and 1500 g or more for at least 10 minutes (Tietz, 1995). The usually recommended upper limit for storage before centrifuging is 2 hours, although even a longer storage does not seem to influence cholesterol levels (Ono *et al.*, 1981). For plasma samples, centrifugation can be done immediately. If it has to be done later, the storage temperature may vary with the length of the storage. When centrifuging, the tubes should be closed tightly to avoid evaporation. This holds for both serum and plasma samples.

The *WHO MONICA Project* recommended that for serum preparation, blood samples are allowed to clot at not more than 20 °C usually for up to one hour before centrifugation. Blood specimens should be centrifuged at a temperature of not more than 20 °C at a minimum of 1500 G for at least 10 minutes. With a refrigerated centrifuge, centrifugation should preferably be done at 4 °C. Whole blood samples must not be frozen during processing (WHO MONICA Project, 1998). For plasma preparation, after thorough mixing of the blood samples with EDTA, they should be cooled on melting ice. Within 3 hours (and preferably within one hour), the tubes should be centrifuged at 4 °C in a refrigerated centrifuge at 1500 G for 30 minutes. If a refrigerated centrifuge is not available within 3 hours of collection, the samples may be centrifuged at room temperature within 1 hour of collection, and the plasma stored at 4 °C (WHO MONICA Project, 1998).

### **2.5.8 Haemolysis**

Haemolysis may occur during blood drawing and handling. It will result in higher cholesterol values, if the direct "Liebermann-Burchard" method is used. For enzymatic methods, only a gross haemolysis has an increasing effect on cholesterol (Tietz, 1995). Lipideamia can affect the triglyceride measurements by interfering with absorbance measurement (Belcher *et al.*, 1991). In the *WHO MONICA Project*, it was recommended that haemolytic samples should be discarded and fresh samples should be drawn from the subjects and analysed (WHO MONICA Project, 1998).

### **2.5.9 Storage after centrifuging**

Storage before isolation of HDL: It is usually recommended that isolation of HDL should be done on the day of blood sample collection. Storage of fresh samples for more than three days at +4 °C leads to a reduction in HDL cholesterol levels of about 8.2% to 14.9%. Storage of frozen samples for more than 14 days at -20 °C leads to a decrease in HDL cholesterol

levels, whereas storage at lower temperatures does not produce such modifications (Bausserman *et al.*, 1994; Evans *et al.*, 1995).

Storage before analysis of cholesterol and triglycerides: Storage using refrigeration or at room temperature between centrifuging and analysis does not seem to be crucial if the material is analysed within a few days and bacterial contamination is avoided. Freezing in appropriate vials is acceptable at a temperature of -20 °C for 1 year or at a temperature of -60 °C for a longer period (Rehak and Chiang, 1988). However, a recent study on long term storage of serum at -70 °C suggest a decrease of 2% per year for total cholesterol over 7 years, 2.8% per year in triglycerides, and 1.3% (not significant) per year for HDL-cholesterol (Shih *et al.*, 2000).

According to the MONICA Manual, isolation of HDL should preferably be done on fresh serum aliquots on the day of blood collection (Cooper *et al.*, 1988). If impossible, the serum or plasma for HDL cholesterol determination should be frozen at -20 °C and isolation should be performed within 14 days. In two thirds of the populations in the final survey, isolation was done on fresh samples (Ferrario *et al.*, 1999).

It was recommended that the total cholesterol and HDL cholesterol levels should be assessed on the day of sample collection. Samples could be stored for up to four days at +4 °C. If analyses of total cholesterol could not be performed within 4 days, the serum or plasma samples should be immediately stored at -20 °C or lower in tightly stoppered glass tubes (WHO MONICA Project, 1998). In about half of the centres in the final surveys, cholesterol was measured from fresh samples, and in about a half from frozen samples (Ferrario *et al.*, 1999).

### 2.5.10 Total cholesterol and triglyceride determination

Enzymatic methods with automatic analysers, which have been in use since the 1980s have become standard methods in cholesterol measurement (Bergmeyer and Grassl, 1990). They allow very good precision, provided that they are used with care and they are calibrated properly (Cooper *et al.*, 1992). Triglycerides are measured enzymatically in serum or plasma by using reactions in which triglycerides are first hydrolysed to produce glycerol (Bergmeyer and Grassl, 1990).

In the *MONICA Project*, local laboratories were used by each collaborating centre. The use of enzymatic cholesterol method was recommended. It was however recognised that some centres may need to use other methods for local reasons (WHO MONICA Project, 1998). In the final MONICA survey, an enzymatic method was used in all laboratories, except one, which used the so-called "direct", "one-step", "Liebermann-Burchard" method (Ferrario *et al.*, 1999). Triglycerides were not part of the MONICA core study, and therefore no instructions for its measurement were given. Some centres measured triglycerides as a local option.

*NHANES III* has reported the percentage of the sample in the following categories (Sempos *et al.*, 1993):

	<b>Total cholesterol</b>	<b>HDL-cholesterol</b>	<b>LDL-cholesterol*</b>
<b>Desirable</b>	<5.2 mmol/L	□ 0.91 mmol/L	<3.36 mmol/L
<b>Borderline-High-Risk</b>	5.2-6.2 mmol/L	<0.91 mmol/L	3.36-4.13 mmol/L
<b>High-Risk</b>	□ 6.2 mmol/L		□ 4.14 mmol/L

\*LDL-cholesterol was calculated using the Friedewald formula:

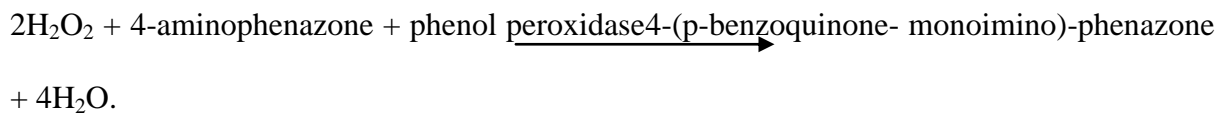
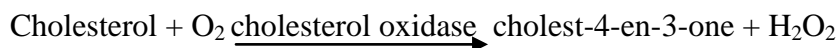


LDL = total cholesterol - HDL - 0.45 x triglycerides.

### 2.5.11 Summary of test principles and clinical relevance

#### A. Total cholesterol

Cholesterol is measured enzymatically in serum or plasma in a series of coupled reactions that hydrolyze cholesteryl esters and oxidize the 3-OH group of cholesterol (Artiss and Zak, 1997). One of the reaction byproducts, H<sub>2</sub>O<sub>2</sub> is measured quantitatively in a peroxidase catalyzed reaction that produces a color. Absorbance is measured at 500 nm. The color intensity is proportional to cholesterol concentration (Artiss and Zak, 1997). The reaction sequence is as follows:

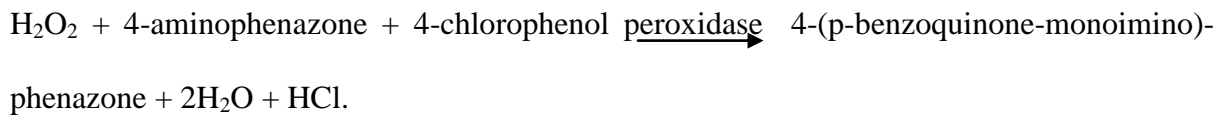
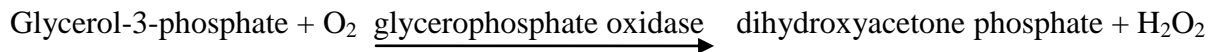
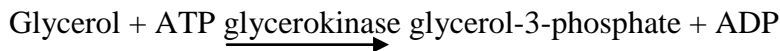
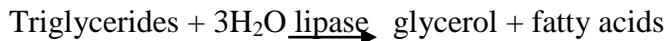


Elevated levels of cholesterol increase the risk for CHD. Cholesterol is measured to help assess the patient's risk status and to follow the progress of patient's treatment to lower serum cholesterol concentrations. Desirable cholesterol levels are considered to be those below 200 mg/dL (5.17 mmol/L) in adults and below 170 mg/dL (4.39 mmol/L) in children (Artiss and Zak, 1997).

#### B. Triglycerides

Triglycerides are measured enzymatically in serum or plasma using a series of coupled reactions in which triglycerides are hydrolyzed to produce glycerol (Cole *et al.*, 1996). Glycerol is then oxidized using glycerol oxidase and H<sub>2</sub>O<sub>2</sub>, one of the reaction products, is

measured as described above for cholesterol. Absorbance is measured at 500 nm. The reaction sequence is as follows:



High levels of serum triglycerides help mark conditions that are associated with increased risk for CHD and peripheral atherosclerosis. High triglycerides are associated with increased risk for CAD in patients with other risk factors, such as low HDL-cholesterol, some patient groups with elevated apolipoprotein B (apo B) concentrations, and patients with forms of LDL that may be particularly atherogenic (Cole *et al.*, 1996). Desirable fasting triglyceride levels are considered to be those below 200 mg/dL (5.17 mmol/L) and are further categorized as borderline (200-400 mg/dL = 5.17 - 10.34 mmol/L), high (400-1,000 mg/dL = 10.34 - 25.85 mmol/L) and Very High (> 1000 mg/dL = > 25.85 mmol/L) (Cole *et al.*, 1996). Very high triglycerides can result in pancreatitis and should be promptly evaluated and treated (Kafonek *et al.*, 1992). Triglycerides are also measured because the value is used to calculate low density lipoprotein (LDL) cholesterol concentrations (Kafonek *et al.*, 1992). In NHANES 2003 - 2004, triglycerides are only measured in specimens from fasting participants (Cole *et al.*, 1996).

### **C. High density lipoprotein (HDL) cholesterol**

Low serum concentrations of HDL-cholesterol are associated with increased risk for CHD. Coronary risk increases markedly as the HDL concentration decreases from 40 to 30 mg/dL (1.03 - 0.78 mmol/L) (Kafonek *et al.*, 1992). A low HDL-cholesterol concentration is

considered to be a value below 35 mg/dL (0.90 mmol/L) and high HDL > 60 mg/dL (> 1.55 mmol/L). HDL-cholesterol values are also used in the calculation of LDL-cholesterol (Belcher *et al.*, 1991).

Direct HDL method: HDL is measured directly in serum. The basic principle of the method is as follows. The apoB containing lipoproteins in the specimen are reacted with a blocking reagent that renders them non-reactive with the enzymatic cholesterol reagent under conditions of the assay. The apoB containing lipoproteins are thus effectively excluded from the assay and only HDL-C is detected under the assay conditions (Belcher *et al.*, 1991).

The method uses sulfated alpha-cyclodextrin in the presence of  $Mg^{+2}$ , which forms complexes with apoB containing lipoproteins, and polyethylene glycol-coupled cholesteryl esterase and cholesterol oxidase for the HDL-C measurement (Belcher *et al.*, 1991). The reactions are as follows:

(1) ApoB containing lipoproteins +  $\alpha$ -cyclodextrin +  $Mg^{+2}$  + dextran  $SO_4$  soluble non-reactive  $\rightarrow$  complexes with apoB-containing lipoproteins

(2) HDL-cholesteryl esters PEG-cholesteryl esterase  $\rightarrow$  HDL-unesterified cholesterol + fatty acid

(3) Unesterified chol +  $O_2$  PEG cholesterol oxidase  $\rightarrow$  cholestenone +  $H_2O_2$

(4)  $H_2O_2$  + 5-aminophenazone + N-ethyl-N-(3-methylphenyl)-N'-succinyl ethylene diamine +  $H_2O$  +  $H^+$  peroxidase  $\rightarrow$  quoneimine dye +  $H_2O$

Absorbance is measured at 600 nm.

#### **D. Low density lipoprotein (LDL)-cholesterol**

Most of the circulating cholesterol is found in three major lipoprotein fractions: very low density lipoproteins (VLDL), LDL and HDL (Belcher *et al.*, 1991).

$$[\text{Total chol}] = [\text{VLDL-chol}] + [\text{LDL-chol}] + [\text{HDL-chol}]$$

LDL-cholesterol is calculated from measured values of total cholesterol, triglycerides and HDL-cholesterol according to the relationship:

$$[\text{LDL-chol}] = [\text{total chol}] - [\text{HDL-chol}] - [\text{TG}]/5$$

where  $[\text{TG}]/5$  is an estimate of VLDL-cholesterol and all values are expressed in mg/dL (or mmol/L).

LDL carries most of the circulating cholesterol in man and when elevated contributes to the development of coronary atherosclerosis. LDL-cholesterol is measured to assess risk for CHD and to follow the progress of patients being treated to lower LDL-cholesterol concentrations. Desirable levels of LDL-chol are those below 130 mg/dL (3.36 mmol/L) in adults and 110 mg/dL (2.84 mmol/L) in children (Kafonek *et al.*, 1992).

- **Reportable range of results**

The expected values for cholesterol, triglyceride and HDL-cholesterol are as follows:

<b>(i) Cholesterol Conc., mg/dL (mmol/L)</b>	<b>Interpretation</b>
< 200 (< 5.17)	Desirable
200-239 (5.17 - 6.12)	Borderline-High
> 240 (> 6.20)	High
<b>(ii) Triglyceride Conc., mg/dL (mmol/L)</b>	<b>Interpretation</b>
< 200 (< 5.17)	Desirable
200- 400 (5.17 - 10.34)	Borderline
400-1,000 (10.34 - 25.85)	High
>1,000 (> 25.85)	Very High
<b>(iii) HDL-C Conc., mg/dL (mmol/L)</b>	<b>Interpretation</b>
< 35 (< 0.90)	Low

>60 (> 1.55)

High

(NCEP, 1995)

## 2.6 Disease and Somatotype

The relationship between somatotype and disease has been first investigated by Sheldon *et al.* (1940, 1954 and 1969). Being a psychologist himself, he wanted to relate somatotype with abnormality of behaviour and function. Sheldon *et al.* (1969) studied psychotic patterns with somatotype and found that paranoid schizophrenic patients were localized towards mesomorphic ectomorphic type of physique where these two components were almost equal and lacking in endomorphy. Catell and Metzner (1993) also found associations of behaviour and somatotype on the much expected lines given by Sheldon. There is a linking of centripetal or abdominal fat with somatotype components as investigated by Rosique *et al.* (1994).

According to a study by Herrera *et al.* (2004), the correlation between ectomorphy and both SBP and DPB showed that as ectomorphy increased the blood pressure decreased, except for the oldest age group. Endomorphy and mesomorphy showed a stable correlation pattern with blood pressure in males indicating a neutral stance of these components in determining the blood pressure, while in females this pattern was more irregular and less consistent. The persons with high levels of SBP and DBP had mean somatotypes, which were closer to those of other male groups characterized by myocardial infarct, coronary heart disease and the risk of hypertension, indicating that these somatotypes may be associated with cardiovascular risk factors. The individuals who had a cardiovascular risk profile are more endomorphic and mesomorphic and less ectomorphic than those with a lower cardiovascular risk profile (Herrera *et al.*, 2004).

Katzmarzyk *et al.* (1999) found that the somatotype has been related to the sum of six skinfolds taken at different sites. In terms of biological risks with a predisposition of disease, the somatotype is a much better predictor than the individual measurements and it was more predictable in males rather than the females. Kalichman *et al.* (2004) observed that individuals of robust physique (with high endomorphy and mesomorphy) showed high mean values of SBP and DBP, whereas the smallest persons had the lowest blood pressure values. They also suggested the existence of common physiological mechanisms in the development of body physique and blood pressure regulation with the possibility of the involvement of pleiotropic genetic and/or epigenetic mechanisms in this regulation.

A longitudinal follow up study of blood pressure was conducted by Harlan *et al.* (1962) in which a group of young men observed for 18-years. Seven hundred and eighty-five (96%) of the surviving members have been re-evaluated, and the mean age at the time of re-examination was 42 years. Significant correlations were observed between the indicators of weight and somatotype. A significantly greater increase in blood pressure was noted in association with increasing weight. Subjects with a predominance of ectomorphic characteristics had a smaller increment of blood pressure over the period of study whereas endomorphic subjects had a greater increment of blood pressure. A significantly greater increase in blood pressure had a predisposition because of the family history in these subjects. The greater increment in blood pressure associated with a positive family history was independent of weight gain and that means if there is a family history the chances of hypertension increase (Harlan *et al.*, 1962).

Katzmarzyk *et al.* (1998) explored the relationship between physique and metabolic fitness from a sample of 413 boys and 343 girls in the age range of 9-18 years from Québec. Physique was assessed using the Heath-Carter anthropometric somatotype. The metabolic fitness was assessed from plasma triglyceride levels (TG), high density lipoprotein

cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), and blood glucose levels (GLY). In both boys and girls the first canonical correlation is significant ( $P < 0.001$ ) and indicates a relationship between the physique and metabolic fitness variables. The Heath Carter anthropometric somatotype explains 8% to 19% of the variance in metabolic fitness. The physique domain highlights a positive relationship for ectomorphy and negative for endomorphy and mesomorphy. The metabolic fitness domain has a positive loading for HDL-C and negative loadings for TG, LDL-C, and GLY. The results indicate that a physique characterized by high endomorphy and mesomorphy has a propensity to have higher blood lipids as these are associated with higher levels of TG, LDL-C, and GLY during 9-18 years.

The somatotype of female patients has been investigated for different categories of genital tract cancer by Eiben *et al.* (2004). The ovarian cancer patients had a somatotype as 6.8-5.3-1.0, whereas those with endometrial cancer had it as 7.9-5.8-0.9. The variance analysis showed that there was no significant difference among majority of the patients who had mesomorphic-endomorph forms where endomorphic elements dominated in their physique and mesomorphy (robusticity) was greater than ectomorphy (linearity). Butova *et al.* (2005) investigated the somatotypic characteristics of healthy women and patients with mammary. Age peculiarities of morphological typing revealed the dominance of athletic type which means a high development of mesomorphy in mature age and mesoplastic type in the elderly one. The anthropometric parameter of women with oncological pathology in the studied periods of ontogenesis has demonstrated a predominance of a mesomorphic vector in shaping their somatotype.

Caldin *et al.* (1959) investigated 400 patients admitted consecutively to a Veterans hospital for pulmonary tuberculosis with the help of photographs for obtaining somatotype evaluation according to Sheldon's method. Analysis of the data revealed that almost of the patients had

mesomorphy and endomorphic-mesomorphy predominant in their body characteristics. Only 14% of the subjects of the study showed the thin, narrow ectomorphic physique which traditionally has been associated with tuberculosis. Clinical impression of the patients, however, suggests that tuberculosis patients may be having weak-looking body physiques. The folk wisdom which seems to relate thin body build and tuberculosis in the past has been attributed by the present authors to confusion between weight loss or emaciation which may be a consequence of the disease and the fundamental physique which existed prior to the onset of the illness. According to Solomon *et al.* (1982) the patients suffering from osteoarthritis (OA) of the hip may be considered a subset of the population because of their greater predisposition to joint failure.

A comparative study of somatotype, bone density, disc degeneration, polyarticular joint degeneration and soft-tissue calcification was carried out by Solomon *et al.* (1982) in 3 groups of individuals: (1) patients presenting with OA of the hip; (2) patients with acute femoral neck fracture; (3) healthy controls. There were significant differences in somatotype in the 2 patient groups; 94% of those with OA were endomorphic mesomorphs. Patients with Down's syndrome seem to have a somatotype which goes much beyond the boundary of the endo-mesomorphic sector and meso-endomorphic sector of the somatochart. Most of these patients possess endomorphy and mesomorphy ratings above 6 each (Buday and Eiben, 1982; Buday, 1990). A study on thalassemia child patients has been conducted by Gaur and Sarkar (1998) which reflect a different set of somatotypes of the patients from those of the control children.

Koleva *et al.*, (2002) examined the association between the somatotype and its main components (endomorph, mesomorph and ectomorph), and the prevalence of several chronic diseases. The data were obtained from a cross-sectional survey designed to assess somatotype and morbidity with special reference to most often diagnosed diseases. In five



different disease groups, the frequency of patients was significantly related to a somatotype. Those with mesomorphic endomorph of physique most frequently suffered from digestive system diseases (40.6%,  $p < 0.05$ ), neuroses (30.1%,  $p < 0.05$ ), and radicalises lumbosacralis (15.4%). The prevalence of arterial hypertension in mesomorph-endomorphs has been found in 37.1% of the cases, endomorphic mesomorphs in 35.5% and mesomorphic endomorphs in 34.3%. Cluster analysis showed that those who suffered most frequently from arterial hypertension and liver disease were having highest endomorphy and mesomorphy and the lowest ectomorphy.

Neni *et al.* (2017) reported that endomorphy and mesomorphy exhibit a small negative association with age at menarche, whereas ectomorphy a small positive association. Significant differences in somatotype were evident for only endomorphy, which exhibit a higher prevalence among urban girls relative to rural girls. Urban girls were heavier and more endomorphic than rural girls. Their findings indicated that age at menarche increases with decreasing endomorphy and mesomorphy components, thus girls with high endomorphy and mesomorphy attained menarche earlier than girls with lower endomorphy and mesomorphy. Furthermore, girls with lower ectomorphy attained menarche earlier than did girls with higher ectomorphy

### **2.7 Somatotype Relationship in Families**

Walker and Tanner (1980) studied the photographs of 82 boys from the Harpenden growth study and assigned somatotype ratings at different ages of 5, 8, 11, 14, and 18 years. These being subjective assessments, inter-judge correlations were obtained for the anthroposcopic ratings of the 18-year-olds which ranged from 0.79 to 0.93 for the three components and these ratings can be considered to be quite reliable. According to them there were very little changes in mean somatotype ratings with age. These findings point towards the direction of the somatotype (especially the Sheldonian) being largely immutable. Damon *et al.* (1962)

studied the somatotype with age and was of the opinion that the prediction of somatotype from earlier ages can be made.

Singh and Singh (2000) reported the somatotypes of father-son duo in Punjabi Sikhs. The mean somatotypes of fathers were 3.87 – 5.07 – 2.12 compared to 3.20 – 4.31 – 2.66 of sons. Mesomorphy is significantly greater in case of fathers whereas sons have significantly larger values of ectomorphy. Endomorphy does not show significant differences between the fathers and sons. Studies on growing children have indicated a change in somatotype from central location towards ectomorphy and showed a wide range between 4 and 20 years of age especially in case of the Gaddi tribe of the Himalayas (Singh and Sidhu, 1980). Rebato *et al.* (2000) analysed the Heath-Carter somatotype to find out familial resemblance in a sample of 1350 siblings (685 males and 665 females) from 634 nuclear families in the province of Biscay (Basque Country, Spain). Maximum likelihood procedure was adopted to estimate sibling correlations for endomorphy, mesomorphy, ectomorphy and somatotypical attitudinal distance (SADi), after adjusting for age and sex. All sibling correlations of the somatotype components were significant, however, these were higher in the case of mesomorphy than in the case of endomorphy. Same sex siblings exhibited significant resemblances for mesomorphic and ectomorphic components. So, the genetic similarity seems to have been reflected in the somatotype components as well.

Heritability estimates of somatotype components based upon familial data from French Canadian families were made by Bouchard *et al.* (1980). Somatotype components were obtained with the help of Heath-Carter method in 239 French-Canadian families from Montreal in the later study. Sibling correlations for somatotype components were 0.40 for endomorphy, 0.30 for mesomorphy, and 0.38 for ectomorphy. Partialling out the effects of 7 socioeconomic indicators permitted an estimate of common familial environment upon co-

variation between relatives. Residual sibling correlations provided broad heritability estimates of 0.50 for endomorphy, 0.42 for mesomorphy and 0.54 for ectomorphy. Familial transmission of Heath-Carter anthropometric somatotype was investigated in a sample of 328 participants from 103 nuclear families in Northern Ontario (Canada) by Katzmarzyk *et al.* (2000). The three somatotype components (endomorph, mesomorph, ectomorph) were subjected to principal components analysis to generate an additional index of physique. Maximal heritabilities observed were 56%, 68%, 56% and 64% for endomorphy, mesomorphy, ectomorphy and PCI respectively, which provided enough proof of familial resemblance for the Heath-Carter anthropometric somatotype. This also highlighted the role of genetic factors in explaining variation in human physique.

Peeters *et al.* (2003) concluded from a review of the available literature that several studies with different designs have attempted to estimate the heritability of somatotype components but ignored the co-variation between the three components as well as possible sex and age effects. This study explores the pattern of genetic and environmental determination from a longitudinal sample of Belgian same-aged twins followed from 10 to 18 years ( $n = 105$  pairs, equally divided over five zygosity groups), with the help of multivariate path analysis. Heritability estimates from 10 to 18 years range from 0.21 to 0.88 for endomorphy, 0.46 to 0.76 for mesomorphy and 0.16 to 0.73 for ectomorphy in boys. In girls, heritability estimates range from 0.76 to 0.89, 0.36 to 0.57 and 0.57 to 0.76 for the respective somatotype components. Sex differences in heritability become significant from 14 years onwards. More than half of the variance in all somatotype components for both sexes at all time points is explained by factors the three components have in common. The finding supports the hypothesis that the variability of somatotype components is influenced by genetic factors. Somatotypes though reflect changes with age during growing years yet these fluctuations do not seem to be reaching extraordinary levels (Hebbelinck *et al.*, 1995). There is a differential

role of genetic and environmental influences on the development of somatotype components as reflected in a family study by Sanchez-Andres (1995).

Kaul *et al.* (1994) found a correlation between the somatotypes among family members in North Indian population and found that significant correlations existed among all the combinations. Kaul *et al.* (1996) reviewed the somatotype literature especially from India and found gaps in information on somatotype and disease. A comparison of Heath-Carter anthropometric somatotype components was attempted in 28 male and 34 female monozygotic (MZ) twin pairs and 19 male and 21 female dizygotic (DZ) twin pairs from 9.3-23.5 years of age by Song *et al.* (1994). The results indicated that there are no differences in mean somatotypes of male twins and female twins although the male twins were significantly more mesomorphic than female twins. Intra-class correlations were consistently higher in MZ than in DZ twins of both sexes. Within-pair variances were consistently lower in MZ than in DZ twins of both sexes.

## **2.8 Somatotype and Cardiovascular Risk Factor**

Cardiovascular disease remains the leading cause of death and disability among women in the United States despite advances in treatment and secondary prevention (Mozaffarian *et al.*, 2008). Recognizing that CVD has roots in early life, the American Academy of Pediatrics now recommends that cardiovascular screening, prevention and treatment begin in childhood (Daniels and Greer, 2008). Interventions to alter CVD risk in childhood require an understanding of the early social contexts that shape risk trajectories (Shonkoff *et al.*, 2009). Therefore, in the Manitoba Study of a cohort of 3,983 men with a mean age at entry of 30.8 years, initial measurements of body weight, represented by body mass index (weight/height<sup>2</sup>), were compared with the 26 year incidence of ischemic heart disease. After adjustment for the effects of age and blood pressure in univariate and multivariate analysis, body mass index was a significant predictor of the 390 cases of ischemic heart disease.

In Herrera *et al.* (2004), it was observed that females were more endomorphic and mesomorphic than males. Males were more ectomorphic than females. SBP showed a downward tendency with age in males, while in females the tendency was for the SBP to increase. Correlations among variables were from low to moderate and ranged from - 0.37 to + 0.34 in males, and from - 0.18 to + 0.32 in females. Correlations tended to be stronger in the younger age group and differences between sexes were found. A negative tendency in the correlation between ectomorphy and both SBP and DBP was found, except for the oldest age group, for which the correlation was positive. Endomorphy and mesomorphy showed a stable correlation pattern with blood pressure in males, while in females this pattern was more irregular and less consistent.

In the study of Badrul *et al.* (1996), relationships between cardiovascular risk factors, body composition and tissue distributions were examined in 10 Indian and 10 Swedish males matched by age, height and weight. The body was divided into 29 compartments by means of a multiscan computed tomography (CT) technique. Fasting glucose, insulin, and triglycerides (TG) were higher in Indians than in Swedes. During the oral glucose tolerance test (OGTT), the glucose area was similar in both groups, whereas the insulin area was 80% larger in Indians. Adipose tissue (AT) and skin volumes were larger and remaining lean tissues were smaller in Indians. Indians had proportionally less muscle and more skeleton in the legs, but no ethnic difference could be demonstrated with respect to AT distribution. The visceral AT to total AT volume ratio was positively related to insulin and TG and with higher risk factors for Indians at any given ratio. TG and glucose were negatively related to the leg muscle to total muscle volume ratio, and this ratio was smaller in Indians. It is concluded that the metabolic disturbances of Indians are not necessarily dependent on a preponderance of visceral AT, and also that an upper-body muscle distribution - recognized as a new

phenotypic companion to the metabolic syndrome - is statistically related to cardiovascular risk factors (Badrul *et al.*, 1996).

Valkov *et al.* (1996), in their study which included 635 practically healthy male subjects aged 20-60 years employed in the Plovdiv region. Using the Heath-Carter method, the following somatotypes are differentiated: endomorphic (135), mesomorphic (157), ectomorphic (26) and subjects not matching any somatotype and lying outside the typological diagram (317). The analysis of the relationship between the somatotypes and some risk factors for ischemic heart disease (IHD) (cholesterol, arterial blood pressure, body mass, and body mass index) revealed the following peculiarities: the subjects lying outside the typological diagram showed the greatest predisposition for developing IHD followed by the endomorphic and mesomorphic groups. The risk for IHD in the ectomorphic group was negligible. Arterial hypertension was found in 48% of the subjects having the type lying outside the typological diagram, 31% in the endomorphic and mesomorphic groups. None of the ectomorphic subjects had elevated arterial pressure. Increased risk due to high cholesterol levels was established in the subjects outside the diagram (56.6%), while in the remaining groups it was 46%. The mean levels of triglycerides and LDL had critical values in the subjects outside the typological diagram while the HDL levels were the most elevated in the subjects of ectomorphic type.

In the study of Mozumdar and Roy (2008), 102 adult male with unilateral lower-extremity amputation residing in Calcutta and adjoining areas were investigated. The anthropometric data for somatotyping and data on cardiovascular risk traits (such as body mass index, blood pressure measurements and blood lipids) have been collected. The somatotyping technique of Carter and Heath (1990) has been followed. The result shows high mean values of endomorphy and mesomorphy components and a low mean value of the ectomorphy

component among the amputated individuals having cardiovascular risks. The results of both discriminant analysis and logistic regression analysis show a significant relationship between somatotype components and CVD risk among the individuals with lower extremity amputation. The findings of the study support the findings of similar studies conducted on the normal population. Diagnosis of CVD risk condition through somatotyping can be utilized in prevention/treatment management for the individuals with lower extremity amputation.

Priebe (2000) suggested that medical or physical fitness best reflects the quality of cardiovascular biological age and a measure of this is exercise tolerance. Poor exercise tolerance indicates severity of underlying disease with a low aerobic capacity indicative of preoperative short and long term cardiac risk. According to Blair (2007), the precise effect of regular aerobic training on the accelerated decline in aerobic capacity remains unresolved. It is clear however, that there is an age related decline in aerobic fitness with increasing age but the decline in fitness occurs nearly twice as fast in sedentary men and women compared with individuals who maintain regular exercise training (Blair, 2007).

In the work of Deshpande *et al.* (2016) on screening of risk factors for cardiovascular diseases among school going adolescent aged 10-18 years. Among 871 adolescents, 462 (53.05%) were male. Mean age  $\pm$  SD was  $13.86 \pm 2.21$  years. Majority of the adolescents 782 (89.78%) consumed inadequate amount of fruits and vegetables. The proportion of adolescents who consumed junk food more than three times a week was 24.34% (212). There were 413 (47.42%) study subjects who consumed added salt while having their meals. Thirty-seven (4.25%) consumed carbonated drinks more than 3 times a week. More than half 478 (54.88%) were not physically active. Most of them i.e. 734 (84.27%) spent three hours or more per day on sedentary activities. Proportion of overweight and obesity was 5.97% (52) and 2.87% (25). Proportion of pre-hypertension was 10.10% (88) and hypertension was

8.27% (72). High proportion of risk factors for CVD is found among the school going adolescents in the urban area.

Paul *et al.* (2016) observed that fat distribution assessments of the patients showed that some of them are at high health risk which might be implicated in non communicable diseases such as diabetes and cardiovascular diseases later in life. It was more prevalent in the adolescent females. The 3 different equations used in their study to measure fat percentage showed no differences which implied that one could be used instead of the other with expectation of similar result in a clinical setting. They suggested periodic assessment of weight and fat distribution of children and adolescents in schools and communities as part of school health program as this is essential for early detection, planning and implementation of intervention programs to reduce morbidity and mortality associated with overnutrition.

Leko *et al.* (2017) clearly showed in their study that endomorphy correlates positively with blood pressure in both sex and this may be a common component of physique that contributes to the development of risk factor for CVD in the paediatric population. In the study conducted by Isezuo *et al.* (2018), BP was found to increase with age among the subjects and was higher in females than males; and higher in upper than lower socioeconomic class subjects. BMI was higher in females than males, and BP increased as the BMI percentile of the subject increased, supporting the premise of its predictive significance.

## **2.9 Skinfolds and Relative Body Fat**

Anthropometric skinfolds measurements have been used to estimate body fatness for many years and in many different populations. Lohman (1981) suggested that approximately 50 to 100% of body fat is stored subcutaneously. As a close inverse relationship exists between whole-body density and relative body fat (Durnin and Womersley, 1974), it is possible to predict fatness from whole body density. The estimation of body fat (relative and absolute)



from skinfold is therefore, based upon the regression of the logarithmic transformation of skinfolds against whole-body density, the relationship between skinfolds and body density varies across populations.

Despite their widespread use, the use of skinfold regression equations to predict body fatness has been severely criticised recently. Much of this criticism emanates from the findings of the Brussels cadaver analysis study (Martin *et al.*, 1985, 1992, 1994; Clarys *et al.*, 1987). When skinfolds are used to predict body fat, several assumptions have to be made (Martin *et al.*, 1985; Heyward and Stolarczyk, 1996; Hawes and Martin, 2001). The study consisted of two separate cadaver dissection studies of men (n = 12) and women (n = 13) ranging in age from 55-94 years. Cadavers were extensively measured and dissected into skin, AT, skeletal muscle, bone, organs and visceral tissues. The study aimed to extend the existing quantitative data on tissues and organs in humans and obtain data to validate existing in vivo body composition methods and develop new anthropometric models of body composition. Skin thickness has been shown to vary between individuals and from site to site (Martin *et al.*, 1992). Furthermore, AT compressibility varies with factors such as age, gender, tissue hydration, anatomical site and cell size (Martin *et al.*, 1992).

The lipid fraction of AT may also be highly variable. Martin *et al.* (1994) suggested a range of 60-85° although an earlier investigation found a much greater variation of 5.2 to 94.17° (Orpin and Scott, 1964). Fat distribution is affected by factors such as age, gender, energy balance and the level of total body fat and local AT biology (Bouchard *et al.*, 1993). In general, for any sum of skinfolds, total body fat is likely to be higher in older individuals because of greater fat internalisation with age (Lemieux *et al.* 1995).

The identification of bony and anatomical landmarks is also more difficult in the obese (Bray & Gray, 1988). For these reasons, alternative anthropometric methods that rely on

circumference of 10 measurements (Weltman *et al.*, 1987; 1988) have been recommended for estimating body composition in the obese (Heyward & Stolarczyk, 1996). An alternative approach is to assess body fatness in relation to the average for the population (McArdle *et al.*, 2001). This value should be considered in relation to the variation that is observed in populations who differ with regard to age, gender and ethnicity.

### **2.10 Anthropometric Girth Measurements**

Fat deposition differences between males and females begin in childhood and become progressively established after maturation (Malina and Bouchard, 1988). Males tend to accumulate more truncal fat, whereas fat deposition in females tends to be at the same rate on the trunk and limbs (Malina and Bouchard, 1988). The study of a large number of obese men and women highlighted the sexual dimorphism that exists with regard to AT distribution in the mature individual (Krotkiewski *et al.*, 1983). When matched for body fat mass, females had a greater fat cell size and number in the gluteal and femoral regions and males a greater fat cell size and number in the abdominal region. Consequently, men had a greater AT thickness in the abdominal region and females a greater thickness in the gluteo-femoral region. Men also tend to have more visceral or intra-abdominal fat (IAF) for any given total body fat, although it increases with age in both genders and in the non-obese as well as the obese (Bouchard *et al.*, 1993; Lemieux *et al.*, 1993). Abdominal obesity, despite being primarily a male characteristic, is also observed in a sub-group of obese women. As IAF is thought to be the principal fat depot responsible for the atherogenic metabolic profile in abdominal obesity (Bjorntorp, 1990), its valid determination is of great importance.

The most frequently used anthropometric indicators of abdominal obesity have been the waist to hip ratio (WHR) and the waist and abdominal girths (Williams *et al.*, 1997). A cadaver dissection of 100 men found that the waist circumference, measured at a level within 1 cm of

the umbilicus, is the best predictor of IAF (Pounder *et al.*, 1997). Based on the incidence of CVD morbidity and mortality in the prospective study of 792 Gothenburg men, Bjorntorp (1985) suggested a WHR cut-off point of 1.00 for men. The same value was also later proposed by Bray (1987). Using the absolute level of visceral fat as the criteria defining increased CVD risk, Lemieux *et al.* (1996) proposed a WHR cut-off point of 0.94 and waist circumferences of 100 cm and 90 cm for men aged 40-years or less and greater than 4% respectively.

Following analysis based on receiver operating characteristic (ROC) curves, Lean *et al.* (1995) proposed two waist circumference "action levels" that could be used to identify individuals at increased CVD risk from being both overweight (BMI > 25 kg/m<sup>2</sup>) and/or abdominally obese (WHR > 0.95). This approach utilises the concepts of sensitivity (the proportion of people with a disease who are correctly identified by a positive test) and specificity (the proportion of people without the disease who are correctly identified by a negative test). Fletcher *et al.* (1996) in their study found that using waist circumference as a tool to screen for individuals at risk of CVD because of hypercholesterolaemia, low HDL-C and/or hypertension would misclassify about 35% of subjects.

Ramamani and Suganya (2018) findings showed a high proportion of obese participants by both the anthropometric assessment methods, namely BMI and WHtR, however, the proportion of participants by both the methods are not same, this is mainly due to the fact that, BMI have been classified into Normal, underweight, Overweight and Obese, whereas , WHtR on the other hand is determined only based on single cut-off value of 0.5, above which the participants are considered to be obese.

## **2.11 Trends in Cardiovascular Disease**

Despite continuing to be the leading cause of morbidity and mortality in modern industrialised nations, CVD death rates have declined over the past 30 years in many developed countries (Ounpuu *et al.*, 2001). In developing countries, the opposite has been the case, where CVD mortality rates have increased (Ounpuu *et al.*, 2001). The incidence of CAD, the most prevalent cardiovascular disease has also declined during recent decades since its peak in the 1960's (Rosamund *et al.*, 1998; Goldberg *et al.*, 1999). Globally, however, it is anticipated that between the years 1990 and 2020 morbidity and mortality rates from CAD will more than double (Ounpuu *et al.*, 2001).

The close association between diabetes and CVD suggests that current predictions of a large increase in the prevalence of type 2 diabetes may well precede a large increase in CVD (James, 2001). Some evidence for this is already available. Hu *et al.* (2000) have recently reported that an increase in BMI among 85,941 females explained an 87% increase in CHD, whilst decreases in cigarette smoking, an improvement in diet and an increase in postmenopausal hormone use explained decreases in CHD of 13%, 16% and 97% respectively. Some biometric indices have been suggested to define body shape and through them the individual's health status may be determined. When those indexes are altered, they are associated with cardiovascular diseases, hypertension, dyslipidaemia, diabetes, cancer and osteoporosis, among others (Urquidez-Romero *et al.*, 2017).

## **2.12 Body Habitus and Coronary Artery Disease (CAD) in Men**

During the last several decades, a great deal of attention has been focused on the identification of potentially modifiable biological, physiological and biochemical risk factors (Leon, 1981) that place the individual at an increased risk of developing atheromatous lesions in the coronary blood vessels. The degree of overweight and obesity are two possible risk

factors that have attracted a great deal of research attention in men. Height has also been studied as a potential marker for ischaemic CAD.

Despite this abundance of information, contrasting findings suggest that the exact position of overweight or obesity in the etiology of CAD remains unclear. One possible explanation for this disparity is that the measurement techniques employed do not satisfactorily estimate body fatness. More recent evidence suggests that these inconsistencies can also be partly explained by the distribution of body fat. As the metabolic complications associated with excess body fat may require a prolonged period of time before their effect on cardiovascular disease mortality is observable, the duration of the obese state may also be an important factor in explaining these inconsistencies (Bjorntorp, 1985). Body habitus include body weight and height, weight for-height, relative weight, total body fat, fat distribution, subcutaneous fat pattern and somatotype. Body weight and height are the simplest, most accessible measurements of body size and are generally reliable with small technical errors of measurement.

### **2.12.1 Body weight and height**

Amongst the earliest investigations of an association between CHD, body weight and height are the classic studies of Harvard and Pennsylvania University students (Paffenbarger *et al.*, 1966a, 1966b). They found that for later coronary decedents, body weight at initial examination was greater than controls. An increased incidence of IHD was reported for shorter London transport workers (height range 151 to 167 cm) compared to their taller counterparts ( $P < 0.1$ ) (Morris *et al.*, 1966). A study of 17,530 London office workers reported an inverse relationship between height and IHD after 7.5 and 10 years follow-up following multivariate adjustment for age and grade of employment (Marmot *et al.*, 1978; 1984).

Further research of nearly 18,000 Civil servants discovered the highest IHD incidence rate was for subjects shorter than 165.1 cm (Morris *et al.*, 1980). A 16-year prospective study of almost 1.8 million Norwegians (approximately 900,000 men) found CVD mortality was clearly reduced for those who were taller (Waalder, 1984). For males shorter than 160 cm, CVD mortality was 50 to 100% greater than the total. For those between 185 and 189 cm, however, CVD mortality was only 70 to 80% of the total mortality. The British Regional Heart Study of 11,135 middle-aged men demonstrated a similar finding (Walker *et al.*, 1989). The mean height of subjects who suffered an IHD event ( $n = 443$ ) was significantly lower than the height of the remaining subjects (111.7 vs 173.3 cm,  $P < 0.001$ ).

Adjustment for age, social class, serum TC, HDL-C, SBP and cigarette smoking weakened the association by over 50%. As height and lung function (forced expiratory volume in one second, FEV) were closely correlated ( $r = 0.44$ ,  $P < 0.002$ ) and lung function is associated with IHD (Cook & Shaper, 1988), expiratory volume was added to the multivariate model. The addition of lung function alone ( $P = 0.25$ ) or in combination with other confounding variables ( $P = 0.70$ ) further weakened the relationship.

After 26-years follow-up of a select cohort of almost 4,000 North American male airline pilots, body weight was significantly greater ( $16.5 + 0.5$  vs  $14.2 + 0.2$  kg,  $P < 0.01$ ) and height shorter ( $175.8 + 0.3$  vs  $176.9 \pm 0.1$  cm,  $P < 0.01$ ) in subjects who developed CHD (Rabkin *et al.*, 1977). Hebert *et al.* (1993) found that among a population of 22,071 US male physicians, the relative risk of myocardial infarction was 35% lower in the tallest men ( $>185.4$  cm) compared to the shortest men ( $< 170.2$  cm).

In a short-term study (3 year follow up) of almost 30,000 US men, Rimm *et al.* (1995) found that, in comparison to men whose height was  $< 173$  cm, the multivariate relative risk of CHD decreased steadily within increasing stature. The relative risk in the highest quintile for height

(> 186.0 cm) was 0.67. In a study that adjusted for age, obesity, smoking status, HDL-C, TC, hypertension, diabetes and education, Parker *et al.* (1998) reported a strong inverse association between height, CHD and stroke. In this study, men taller than 177.45 cm had an 83% lower risk of CHD compared to men shorter than 165.38 cm.

### **2.12.2 Evaluation of body weight and height as predictors of coronary heart disease**

A number of possible explanations have been proposed to give the inverse relationship between height and CHD a biological basis. Inadequate pre-natal, infant and childhood nutrition and the occurrence of illness during the growing years may partly account for some cases of shorter attained adult stature. It is plausible that these factors may also directly affect pulmonary development and therefore, explain the association between height and lung function.

Based on findings from a large number of studies, Barker had suggested that under nutrition of the foetus can lead to permanent changes in structure, physiology and metabolism that predispose to elevated fibrinogen and factor VII, noninsulin dependent diabetes, hypertension, hyperlipidaemia and therefore, to an increased risk of CVD (Barker, 1994). Stern (1996) has supported this hypothesis by suggesting that non-insulin dependent diabetes mellitus and CVD share common genetic and environmental antecedents, including foetal and early life nutritional deficiencies. Inverse relationships between height and TC, HDL-C, SBP and smoking duration have also been reported (Walker *et al.*, 1989). Correlation coefficients are weak, however ( $r = -0.04$  to  $-0.11$ ,  $P < 0.002$ ) and are significant due to the large sample size.

A further possible biological mechanism is that taller individuals have larger coronary arteries than shorter individuals and therefore, have a lessened risk of occlusion (Palmer *et al.*, 1990). Support for this mechanism can be derived from studies that have found a higher

rate of post coronary by-pass surgery mortality in shorter individuals compared to taller individuals (Fisher *et al.*, 1982; Loop *et al.*, 1983). In the Manitoba study (Rabkin *et al.*, 1977), the mean body weight of the CHD subjects ( $76.5 \pm 0.5$  kg) was only moderate and although significant, differed from the body weight of subjects free of CHD by only about 2kg. The striking similarity in the body weight of subjects with significant ( $17.4 \pm 9.6$  kg) and insignificant ( $77.8 \pm 11.3$  kg) arterial disease (Flynn *et al.*, 1993) may be partly accounted for by the insensitivity of the disease classification criteria used. Of interest would be a comparison of the mean body weight of asymptomatic subjects and those with evidence of extreme arterial disease. Contrary to this theory, however, no difference was found in the height and weight of men free from CAD when compared to men with angina and an angiogram showing greater than 50% luminal narrowing (Ley *et al.*, 1994).

### **2.12.3 Weight-for-height ratios**

A number of large-scale population studies examining the association between BMI and CHD have been performed in both North America and Europe. Jooste *et al.* (1988) have examined this relationship in 7,188 white South Africans. Data gathered in these studies have produced inconsistent findings. Dyer *et al.* (1975) found that a U-shaped curve described the relationship between BMI and CHD mortality in 1,233 white middle-aged North American men followed for 14 years. Rhoads and Kagan (1983) reported this phenomenon in 8,006 men aged 45 to 68 years who were subsequently followed for 10 years as part of the Honolulu Heart Program. In this latter study, excess deaths amongst those in the lower BMI category were due primarily to cancer and in the upper BMI groups to CHD. In South Africa, the incidence of CHD in relation to BMI was greater in both the lowest (BMI < 20 kg/m<sup>2</sup>) and highest (BMI 30-35 and > 35 kg/m<sup>2</sup>) (P < 0.01).



In a further multivariate model with age, TC, triglyceride (TG), SBP, cigarette smoking, presence of diabetes and a fat distribution index entered as covariates, BMI was not a predictor of CHD ( $P > 0.05$ ) (Ducimetiere *et al.*, 1986). The Stockholm prospective study of 3,168 men identified smoking and elevated levels of plasma TC and TG as independent risk factors for IHD (Carlson and Bottiger, 1972). After adjustment for subscapular skinfold thickness, the independent effect of BMI on either non-fatal myocardial infarction or death from CHD was not significant ( $P > 0.05$ ) after 12-years follow-up in the Honolulu Heart Program (Donahue and Abbott, 1987). Hargreaves *et al.* (1992) reported that, of an original random sample of 107 Edinburgh men, 11 developed clinical CHD over the subsequent 12 year period. Examination of baseline data revealed the BMI of CHD men ( $26.7 \pm 0.8 \text{ kg/m}^2$ ) was greater ( $P < 0.05$ ) than the men who remained free of the disease ( $24.9 \pm 0.3 \text{ kg/m}^2$ ).

Researchers from the Paris prospective study found increasing BMI was modestly associated with CVD in subjects with a mean blood pressure less than 96 mmHg, but had no effect in men with higher blood pressure ( $> 96 \text{ mmHg}$ ) (Filipovsky *et al.*, 1993). In a few instances, large scale prospective studies have reported a significant independent relationship between BMI and CHD. After adjustment for age and blood pressure, BMI was found to be a significant independent predictor of sudden death ( $P < 0.01$ ), coronary insufficiency or suspected myocardial infarction ( $P < 0.05$ ) and myocardial infarction ( $P < 0.05$ ) (Rabkin *et al.*, 1977). Shaper *et al.* (1997) suggested that risk of cardiovascular death, heart attack and diabetes increases progressively from a BMI of  $< 20.0 \text{ kg/m}^2$ , even after adjusting for age, smoking, social class, alcohol consumption and physical activity. Lindsted and Singh (1998) studied 5,062 Seventh Day Adventists who had never smoked. The lowest risk of cardiovascular mortality was for men with a BMI in the range 14.3 to 22.5  $\text{kg/m}^2$ .

#### **2.12.4 Relative weight**

Relative weight is obtained by expressing the individual's bodyweight as a percentage of some reference weight. This reference data, usually based on a large, random, cross-sectional sample can be obtained from a regression equation or chart (Lieberman and Probart, 1992) or more frequently a set of height-weight tables. Although, relative weight implies no value judgment (Harrison, 1985) and correlations with mortality has led to the application of the concept of "desirable" or "ideal" weight. These terms are used to describe individuals at lowest-risk of premature mortality and as the standard for weight reduction targets. An autopsy study of 127 Framingham decedents found relative weight 9 years prior to death was an independent predictor of heart weight but not left ventricular muscle thickness, percentage luminal involvement or percentage luminal insufficiency (Feinleib *et al.*, 1979). The final report of the Pooling Project Research Group (1978) suggested relative weight was associated with an increased risk of a first coronary event only in younger men aged 40 to 44 years ( $P < 0.01$ ) and 45 to 49 years ( $P < 0.05$ ).

#### **2.12.5 Body fat distribution**

Evidence is accumulating in support of the hypothesis suggesting the anatomical distribution of body fat as a stronger predictor of susceptibility to CHD mortality and morbidity, than measures of overweight or obesity. In the Paris Prospective Study, 6,718 men aged 42 to 53 years were followed for an average of 6.6 years (Ducimetiere *et al.*, 1986). Trunk skinfolds (subscapular, axillary and subumbilicus) were the strongest predictors of CHD ( $P < 0.05$ ), whereas thigh skinfolds (anterior, posterior, internal and external) were not associated with CHD ( $P > 0.05$ ). The trunk-to-thigh skinfolds ratio was a highly significant predictor of angina pectoris ( $P < 0.0001$ ) and to a lesser extent, sudden death and myocardial infarction ( $P < 0.01$ ). The association between the skinfold ratio and total incidence of CHD was also highly significant ( $P < 0.00001$ ).

In multivariate analysis, with TC, cigarette habit, blood pressure, diabetes, age, BMI and TG as co-variables, the skinfold ratio remained a significant predictor ( $P < 0.025$ ). The study of Edinburgh men found baseline abdominal skinfold thickness was significantly greater ( $P < 0.05$ ) in the 11 men who developed CHD than the 96 men who remained free of the disease (Hargreaves *et al.*, 1992). There was no difference in triceps and subscapular skinfold thickness ( $P > 0.05$ ). After adjustment for HDL-C, abdominal skinfold thickness remained an independent predictor of CHD ( $P < 0.05$ ). Waist height ratio and waist circumference can be helpful parameters in identifying school age children with adverse blood-lipids profile where population based screening is considered (Yilgwan *et al.*, 2017).

Mukadas *et al.* (2016) reported in their study that there is a strong relationship between BMI and BF% measured by BIA which is influenced mostly by gender and to a lesser extent by age among adult Nigerians. Also, there is linear relationship between BMI and BF% which implies that as BMI increases there is a corresponding increase in the BF% of participants. Therefore, their findings support that age and gender should be considered when interpreting BMI in a cross sectional studies and either BMI or BF% can be used to assess level of adiposity among the study sample in predicting cardiovascular diseases.

#### **2.12.6 Evaluation of WHR as predictors of CHD**

Rimm *et al.* (1995) reported that after controlling for height and BMI, the relative risk of CHD in men in the highest WHR quintile was 1.42 in comparison to men in the lowest quintile. When separated according to age, WHR was a stronger predictor of CHD in men  $>65$  years than their younger counterparts. Further analysis of waist circumference data showed men in the upper quintile ( $>102$  cm), had a relative risk of 1.44 in comparison to men in the lowest quintile ( $<89$  cm). When variation in height was controlled statistically, the relative risk of men in the upper quintile of waist circumference increased to 1.86. As with

WHR, the association between CHD and waist circumference was stronger among older men. Megnien *et al.* (1999) has recently reported on the 10 year incidence of cardiovascular events in relation to WHR in 552 men. A high WHR ( $> 0.98$ ) was a strong predictor of CAD, and the number of subjects in this group who exceeded a 15% risk of developing a coronary event was more than twice the number in the lowest WHR group ( $< 0.88$ ).

Two notable studies have reported that WHR was significantly related to cardiovascular mortality in Mediterranean populations. In an Italian population characterised by low TC levels and a low incidence of early CHD (Barbagallo *et al.*, 2001). Although there were only a small number of cardiovascular deaths recorded over an 8 year follow-up period, the relative risk for those with a WHR greater than the median was 5.49 in comparison to those with a WHR below the median. Azevedo *et al.* (1999) also found that WHR rather than BMI was associated with a higher risk of a first myocardial infarction. In comparison to men in the first tertile of WHR, the odds ratio of a heart attack in the second and third tertiles were 2.5 and 11.1 respectively. The increased risk of CVD in men with a greater WHR is also apparent among subjects of African-American origin. The atherosclerosis risk in communities study has reported similar positive trends ( $P = 0.06$ ) between WHR and CHD in both black and white men (Folsom *et al.*, 1998).

Flynn *et al.* (1993) reported that both waist-to-thigh circumference ratio (WTR) ( $P < 0.005$ ) and WHR ( $P < 0.05$ ) were independently associated with CAD. Whilst WTR was positively associated with CAD, in contrast to other prospective (Larsson *et al.*, 1984) and angiography studies (Hauner *et al.*, 1990; Hodgson *et al.*, 1994; Thompson *et al.*, 1991) WHR was inversely related to CAD.

### 2.13 Somatotype

The relationship between somatotype and CHD attracted attention in the United States in the 1950's and 1960's (Gertler *et al.*, 1951, 1959; Spain *et al.*, 1953, 1955, 1963; Paul *et al.*, 1963) and later in South Africa (Smith *et al.*, 1979). Of 91 men and 3 women who experienced a non-fatal myocardial infarction before 40 years of age, 42% were found to be dominant mesomorphs, 26% dominant endomorphs, 25% were in the mid range (no dominant component) and only 7% were dominant ectomorphs (Gertler *et al.*, 1951, 1959).

Spain *et al.* (1953), reported the autopsy findings on 111 consecutive white males under 46 years of age. Of these, 38 had suffered death secondary to CAD and 73 had died suddenly and unexpectedly by violent means (suicide, homicide, accident) or some other non-cardiac condition. Of the 38 who died from CAD, 24 were classified as being dominant mesomorphs, 3 endomorphs, 3 ectomorphs and 8 were in the mid range. In the 13 apparently healthy males, the degree of atherosclerosis was found to be distinctly more pronounced in mesomorphic individuals compared to those of ectomorphic dominance. A second post-mortem study also revealed that the extent of coronary atherosclerosis was markedly greater in mesomorphic compared to ectomorphic individuals (Spain *et al.*, 1955). Of 64 consecutive autopsy examinations involving sudden death from coronary occlusion, 44 cases were classified as dominant mesomorphs. In a third study, the incidence of CHD amongst 5000 males aged 36- to 50 years was three times greater for endomorphic-mesomorphs (9.2%) compared to dominant ectomorphs (3.0%) (Spain *et al.*, 1963). This further evidence led to the conclusion that individuals characterised by mesomorphic dominance, were at greater risk of CHD than their ectomorphic counterparts (Spain *et al.*, 1963).

As body fat distribution appears to be particularly important in the relationship between body habitus and CVD, the association between somatotype and fat distribution is of great interest

and may help explain the abundance of CHD amongst mesomorphic individuals. Among 824 men, those classified as android obese (mean somatotype 4.67 - 4.21 - 1.89) were reported to be significantly more mesomorphic and less endomorphic than those with gynoid obesity (mean somatotype 5.91 - 2.16 - 1.84) ( $P < 0.01$ ) (Mueller and Joos, 1985). Mesomorphy is also a masculine characteristic, and as reported for non-insulin-dependent diabetes mellitus, there appears to be an assemblage of male differentiation factors amongst individuals at increased risk of CHD (Mueller and Joos, 1985).

Studies of obese and overweight men have shown a relationship between fat loss and weight reduction and improvements in blood pressure and blood lipids (Reisin *et al.*, 1978; Berchtold *et al.*, 1982; Dustan, 1985; Sopko *et al.*, 1985; Wood *et al.*, 1988; Schotte and Stunkard, 1990). The studies that examined CHD in relation to somatotype revealed very consistent findings. Men with an endomorphic-mesomorphic physique appear to experience a far greater incidence of coronary events than other somatotypes. Ectomorphic dominance appears to be the somatotype least associated with CHD.

In the study conducted by Adodo and Agwubike (2015) among students of college of Health Sciences, Nnamdi Azikiwe University, Nnewi Campus, Anambra State, Nigeria. Their findings indicated that all Nigeria junior handball players as a group were characterised as mesomorphic ectomorphs.

Stojanoska *et al.* (2016) reported the impact of some socioeconomic factors on somatotype components of the population group of adolescents of both males and females in the Republic of Macedonia. According to their study, socioeconomic factors are related to somatotype components, especially with endomorph and mesomorph component of somatotype of Macedonian adolescents. They concluded that parents' educational level and employment status had some influence on body shape and composition.

Umut (2017) reported that generally, endomorphy has low negative and mesomorphy positive relationships with physical fitness tests, whereas ectomorphy correlates significantly with physical fitness measures. Physical fitness elements had low positive correlation with mesomorphy and low negative correlation with endomorphy. The negative association between physical fitness and endomorphy was obvious especially in the activities requiring the whole body movement.

Karol *et al.* (2018) indicated in their study that anthropometric assessment of body build, as well as somatotype analysis, may be key factors in the process of talent identification in basketball. In male adults, there are somatic predispositions for centers (such as the height, weight, arm span and girths) while the body build of forwards tends to be similar to that of the centers. The position with the lowest requirement for body size is the guard.

Ryan-Stewart *et al.* (2018) observed in particular that those who have high mesomorphy values are predisposed to better strength performance and in the lower body, this may also be combined with a higher ectomorphy value. Their overall findings have important implications for both the identification of those predisposed to perform well in sports containing strength-based movements and prescription of training programmes in physically active males.

#### **2.14 Adipose Tissue Morphology and Fasting Lipid and Lipoprotein Levels in Obese and Non-Obese Men and Women**

Studies indicate that fasting TG concentration is associated with subcutaneous abdominal fat-cell size (Stern *et al.*, 1973; Krotkiewski *et al.*,1983) but not gluteal or femoral fat-cell size (Krotkiewski *et al.*,1983) or abdominal fat cell number (Stern *et al.*, 1973). Plasma TC has been reported to be unrelated to either adipocyte size or number, or total body fat (Stern *et al.*, 1973). One study has reported a significant univariate relationship between fat-cell size, determined from a bilateral buttock biopsy and serum TG in women but found no such

relationship in a smaller sample of men (Foster *et al.*, 1977). Fat cell size also related inversely to HDL-C and HDL-C /TC in women but not men.

Despres *et al.* (1988) investigated the independence of the relationship between fat distribution and HDL-C in 429 healthy men after statistically adjusting for TG concentration. The distribution of subcutaneous fat, as reflected by the trunk to extremity skinfold ratio, and abdominal skinfold thickness were significantly related to TG and HDL-C. The relationship between abdominal skinfold and HDL-C remained significant after adjustment for TG and BMI suggesting that a portion of the relationship between HDL-C and subcutaneous abdominal adiposity is independent of obesity and TG.

In a group of healthy sedentary men, WHR had a stronger relationship with fasting TG concentration than either waist girth, subscapular skinfold, relative body fat or BMI (Teny *et al.*, 1989). WHR remained a significant predictor of TG concentration after adjustment for relative body fat. Pouliot *et al.* (1992) also found that WHR, but not relative body fat, was related to fasting TG concentration in obese men.

In the study of Lee *et al.* (1999) a sample of 2,339 adults were considered and those with non-central obesity tended to have blood pressure, lipids and glucose between those of the non-obese and centrally obese. Whilst abdominal adiposity is closely related to elevated TG levels and other lipoprotein-lipid variables associated with elevated CVD risk, investigators studying this relationship in a mixed group of sedentary and exercise-trained men, found that aerobic fitness was a better predictor of TG and HDL (Houmard *et al.*, 1991). Abdominal adiposity was more closely associated with LDL particle diameter and HDL. The importance of physical activity as a lifestyle variable that attenuates the risk of CVD among the obese has received considerable support (Lee *et al.*, 1999).



Abdominal distribution of body fat, particularly an increased deposition of fat in the intra-abdominal cavity, is associated with hypertriglyceridaemia, an elevated number of small, dense LDL particles and apo B, and reduced HDL-C, especially HDL2-C (Kissebah and Krakower, 1997). The traditional lipid markers of CVD risk, TC and LDL-C are not commonly found in subjects with visceral obesity (Despres and Lemieux, 2006).

## **CHAPTER THREE**

### **3.0 MATERIALS AND METHODS**

#### **3.1 Location of the Study**

This study was conducted in Kaduna State University, Kaduna, Nigeria. The University was established in the year 2004 with campuses in Kaduna and Kafanchan. Kaduna State University has a total number of seven Faculties with 39 Departments and students population of about sixteen thousand (16,000).

Kaduna is the capital city of Kaduna State in Northern Nigeria (Figure 3.1). Kaduna State occupies part of the central position of the Northern part of Nigeria and share borders with Zamfara, Katsina, Niger, Kano, Bauchi, Plateau States and Federal Capital Territory.

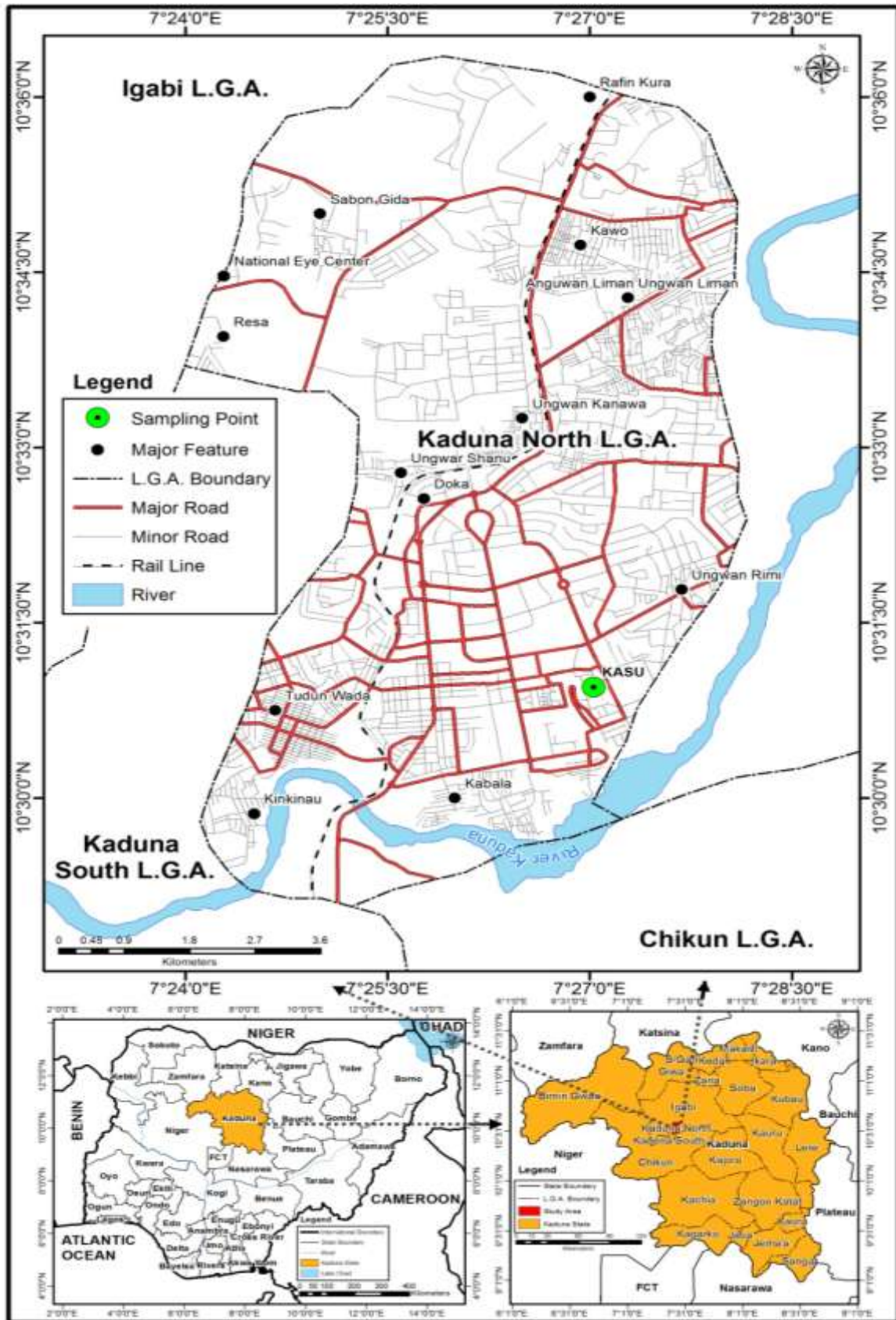


Figure 3.1: Map of Kaduna North L.G.A. showing Sample Point  
 Source: Modified from the Administrative map of Kaduna State, 2014

### 3.2 Study Subjects

The study group which was considered representative of apparently healthy population was a cross – sectional sample of 560 undergraduate students of Kaduna State University, Kaduna.

#### 3.2.1 Sample size determination

The sample size for this study was determined using the formula for calculating sample size (Naing *et al.*, 2006; Prashantand Supriya, 2010; Jaykaran and Tamoghna, 2013):

$$n = (z\alpha^2 pq)/d^2,$$

Where:

n = minimum sample size;  $\alpha$  = level of significance. It will be set at 0.05.

1 -  $\alpha$  = confidence level that the estimate is within distance (d) of the proportion of interest. It is 0.95.

Z $\alpha$  = standard normal deviate; at 95% confidence level; Z $\alpha$  = 1.96 for a two tailed test.

p= proportion in the target population estimated to have a particular characteristic. It was estimated at 50% (0.5).

d = degree of accuracy desired or the distance (or tolerance) – how close to the proportion (p) of interest the estimate is desired to be. It was within 0.05.

$$q = 1 - p$$

$$n = \frac{(1.96)^2 \times 0.5(1 - 0.5)}{(0.05)^2} = \frac{(1.96)^2 \times 0.5(1 - 0.5)}{(0.05)^2}$$

$$n = \frac{(1.96)^2 \times (0.5 \times 0.5)}{(0.05)^2} = 384.16 \quad (384.16 + 10\% = 422)$$

#### 3.2.2 Inclusion criteria

- i. Undergraduate students of Kaduna State University, Kaduna, between the ages of 17 - 33 years within the Main campus (Kaduna) and Kafachan campus.
- ii. Apparently healthy students

- iii. Physically fit without deformity

### **3.2.3 Exclusion criteria**

- i. Postgraduate students of Kaduna State University, Kaduna
- ii. Undergraduate students above the age of 33 years within the Main campus (Kaduna) and Kafachan campus.
- iii. Students with certified health challenges
- iv. Students of Kaduna State University that their course of study were not within the two campuses mentioned above.
- v. Those that decline consent after they were recruited.

### **3.2.4 Informed consent**

All participants were informed about the aim and procedure of the study and given the option of refusal without giving reason. Subjects consented to the above conditions in writing (Appendix II).

### **3.2.5 Ethical approval**

The ethical approval was sought according to World Health Organization (WHO, 2002) and Helsinki declaration (The World Medical Association, 2013) from the Health Research Ethical Committee, Kaduna State Ministry of Health, Kaduna (Appendix I) and Ahmadu Bello University Committee for Use of Human Subjects for Research..

## **3.3 Materials**

### **3.3.1 Measurement tools and devices**

Tools used for the study: weighing machine, portable anthropometer, non-elastic measuring tape, sliding calipers, stethoscope, sphygmomanometer, plain tube, tourniquet, cotton wool, methylated spirit, 10 ml syringe and 22 G needle and reagents test kits.

### **i. Weight and Height Measurements**

A weighing machine with anthropometer (Angel, 150 MA; Istanbul/ Turkey) was used to determine the weight and height of all participants (Plate I).

### **ii. Body Fat Percentage Measurements**

Slim guide skinfold caliper and digital body fat caliper were used to measure skinfold thickness (Super Accurate Digital measurement; AccuFitness, Greenwood Village, CO) (Plate II).

### **iii. Width Measurements**

Width measurements were obtained with a sliding caliper (Digital Caliper; 150mm) (Plate III).

### **iv. Girth Measurements**

Girth measurements were obtained with a non-flexible tape measure (Myotype; AccuFitness, Greenwood Village, Co) (Plate IV).

### **v. Somatotype Assessment**

Somatotype was calculated using the Heath and Carter method (Heath and Carter, 1967) and modified method of Heath and Carter method (Carter and Heath, 1990).

### **vi. Blood Pressure Measurement**

Littmann Brand Classic II S.E. Stethoscope (SEPO120B) and Agary® aneroid Sphygmomanometer (manufactured for: Agary Pharmaceutical Limited, China) (Plate V and VI).

### **vii. Blood Sample Collection**

Plain tube, tourniquet, cotton wool, methylated spirit, 10 ml syringe and 22 G needle, reagents test kits.

### 3.3.2 Research assistants

Six research assistants (males and females) were recruited and trained on the procedures of using the tools for measurements and detailed protocols were explained.

### 3.3.3 Reagent test kits

Reagent diagnostic kits for the measurements of TC, HDL-C and TG were obtained from RANDOX assay kits (Randox Laboratories Ltd, United Kingdom, Cat no. RX MONZA CH 210). These consisted of CHOL Liquicolor reagent, TG Liquicolor reagent, precipitant for HDL-C determination as well as CHOL, HDL-C and TG standards. The content of these reagents were as follows:

#### (i) CHOL Liquicolour reagent

This contained phosphate buffer (pH 6.5, 100 mmol/L), 4-aminophenol (5 mmol/L), 4 – aminophenazone (0.3 mmol/L), phenol (5 mmol/L), peroxidase (>.5 kU/L), cholesterol esterase (>150 U/L), CHOL oxidase (100 U/L) and sodium oxide (0.05%).

#### (ii) TG liquicolour reagent

This contained pipes buffer (pH 7.5, 50 mmol/L), 4-aminoantipyrine (0.25 mmol/L), magnesium ions (4.5 mmol/L), ATP (2 mmol/L), lipases ( $\geq$  1.3 u/ml), peroxidases ( $\geq$  0.5 u/ml), glycerol kinase ( $\geq$  0.4 u/ml) and glycerol-3-phosphate oxidase ( $\geq$  1.5 u/ml).

#### (iii) Precipitant

The precipitant contained phosphotungstic acid (0.55 mmol/L) and magnesium chloride (25.00 mmol/L). The content of one bottle (80 ml) was diluted with 20 ml distilled water.

(iv) **CHOL Standard:** (5.17 mmol/L CHOL)

(v) **HDL-C Standard:** (4.52 mmol/L HDL-C )

(vi) **TG Standard:** (2.28 mmol/L triglycerides)

### **3.3.4 Laboratory equipments**

Econospin centrifuge, Sorval instrument (Federal Republic of Germany) was used to separate the serum from cells. Humalyzer 2000 Spectrophotometer (Human, Germany) was used for the measurements of serum lipids. BCL 1000 DG adjustable micropipette (the Boehringer Corporation Ltd, London) and Labsystems Finn Pipette (Labsystems Oy Pultitie, Helsinki, Finland) were used for the analysis of serum lipids.

## **3.4 Methods**

### **3.4.1 Sampling techniques**

The study sample was selected by a multistage stratified random sampling technique with proportional allocation from all class levels. Data were collected using an interviewer-administered questionnaire which included socio demographic variables and risk factors. The stratified random sampling technique was adopted because the students population had distinct sub-groups (strata) according to age, gender and educational status. This is in accordance with standards set by the International Society for the Advancement of Kinanthropometry (ISAK, 2012).

### **3.4.2 Anthropometric measurements**

The measurements of all parameters were taken in the morning, under the strict observance of all standard conditions for measurements. In order to estimate the anthropometrical somatotype by Heath-Carter, data of height, body weight, skinfold thickness (triceps, subscapular, supraspinale, medial calf, biceps, abdominal, suprailiac and thigh), as well as the humeral and femoral biepicondylar breadth, flexed arm and maximum tension breadth (upper arm girth), and maximum calf breadth (calf girth) was measured through standardized procedures (Carter and Heath, 1990; Norton and Olds, 1996; Ventrella *et al.*, 2008; Sirvent and Garrido, 2009). Measurements were made in bipedestation, barefooted and in light



clothes. All measurements were taken three times, having the average of them as a final result.

In accordance with internationally accepted standards(Heath and Carter, 1967; Tanner, *et al.*, 1969; Heath and Carter, 1996), the following anthropometric measurements were taken according to standard procedures reported by Lohman *et al.* (1988).

- i. Height:** was measured with a portable anthropometer/stadiometer (0.1 cm sensibility) with an accuracy of 1 mm or 0.1 cm. The subjects were asked to inhale deeply and maintain a fully erect position during the measurement. The subject standing straight and the head oriented in the Frankfort plane (the upper border of the ear opening and the lower border of the eye socket on a horizontal line) and the heels together. Subjects were instructed to stretch upward and took a full breath. The headboard was lowered until it firmly touches the vertex. (Plate V).
- ii. Weight:** was measured to the nearest 0.1 kg with a weighing balance (precision scales - 100 grams sensibility) and the subject wearing minimal clothing (Plate V).
- iii. Biepicondylar breadth of the humerus:** the width between the media and lateral epicondyles of the humerus, with the shoulder and elbow flexed to 90 degrees was measured with a sliding caliper to the nearest 0.05 cm (Plate VI).
- iv. Biepicondylar breadth of the femur:** the subjects sat with knee bent at right angle and the greatest distance between the lateral and medial epicondyles of the femur was measured with a sliding caliper to the nearest 0.05 cm (Plate VII).
- v. Upper arm circumference (girth):** the subjects flexed the shoulder to 90 degrees and the elbow to 45 degrees, clenches the hand, and maximally contracts the elbow flexors and extensors. The measurement was taken at the greatest girth of the arm and measured to nearest 0.1 cm with a non-elastic measuring tape (Myotape) (Plate VIII).

- vi. Calf circumference (girth):** the subjects stood with feet slightly apart and the measurement was taken at maximum circumference. This was measured to nearest 0.1 cm with a non-elastic measuring tape (Plate IX). Care was taken to ensure that the subject's weight was equally distributed through both lower limbs.
- vii. Waist circumference:** The participant stands with his/her arms folded across the thorax, breathing normally. The measurement is recorded at the midpoint between the last palpable rib and the tip of iliac crest (Plate X).
- viii. Hip circumference:** The participant assumes a relaxed standing position, with the feet together, the gluteal muscles relaxed and the arms folded across the thorax. The measurement is recorded at the level of the greatest posterior circumference of the buttocks.
- ix. Triceps skinfold:** All the skinfolds measured with a skinfold caliper to the nearest 0.01 cm (Digital body fat caliper) or 0.05 cm other calipers. The subject's arm hanged loosely in anatomical position, a fold was raised at the back of the arm at a level halfway on a line connecting the acromion and the olecranon processes. The subject stands erect with feet together, shoulders relaxed and the arms hanging freely at the sides. A fold of skin and subcutaneous adipose tissue is grasped gently with thumb and fingers with the skinfold parallel to the long axis of the arm (Plate XI).
- x. Biceps skinfold:** The subject's arm hanged loosely in anatomical position, a fold was raised on the anterior of mid arm (at a level bicep) on a line connecting the acromion and the olecranon processes.
- xi. Subscapular skinfold:** the skinfold was raised on a line from the inferior angle of the scapula in a direction that is obliquely downwards and laterally at  $45^{\circ}$ . The jaws of the caliper are placed perpendicular to the length of the fold about 2.0 cm lateral to the

fingers with the top jaw of the caliper over the inferior angle of the scapula (Plate XII).

- xii. Supraspinale skinfold:** the fold was taken above the anterior superior iliac spine on a line to the anterior axillary border and on a diagonal line going downwards and medially at 45° (Plate XIII).
- xiii. Suprailiac skinfold:** the fold was taken above the posterior superior iliac spine on a line to the posterior axillary border and on a diagonal line going downwards and medially at 45°.
- xiv. Medial calf skinfold:** a vertical fold was raised on the medial side of the leg, at the level of the maximum girth of the calf.
- xv. Abdominal skinfold:** The fold was taken 3 cm lateral and 1 cm inferior to the centre of the umbilicus. The measurement was taken vertically at the abdominal site, but placing the finger or the caliper inside the navel was avoided. The measurement was taken after 2 seconds while applying full pressure of the caliper to the skin.
- xvi. Thigh skinfold:** The thigh skinfold is measured in the midline of the anterior aspect of the thigh. The subject stands with weight shifted back on one leg with the other leg forward, knee slightly flexed, foot flat on the floor. A fold of skin and subcutaneous tissue is grasped in the midline and the jaws of the skinfold calipers are placed perpendicular to the length of the fold (Plate XIV).

### 3.4.3 Blood Collection and Processing

Blood samples were collected and analysed in the Department of Human Anatomy, KASU, on each working day from 8 am to 10:30 am, under the supervision by Physicians from the Department.

On arrival of each participant every morning, preliminary exchange of pleasantries was done, after which the participants were asked what time they ate or drank, to confirm the 12 hour fast. This was followed by a brief registration that entailed assignment of a sample number on a questionnaire which was later filled by the participant. The sample number was quoted on each sample collection tube, accordingly. The sample collection began by tying a tourniquet to the upper arm above the cubital fossa (a tourniquet was applied a few centimeters above the antecubital fossa to occlude the veins). The ante-cubital area was cleaned with cotton-wool that was soaked in methylated spirit, and allowed to dry (blood was then taken using a sterilize 10 ml syringe and 22 G needle). The most prominent vein was identified and 5 ml of blood (fasting blood specimens was taken into plain tubes by venipunctures). The blood sample was emptied into labeled plain tubes and left to stand for twenty to thirty minutes, then spun at 3000 g in a centrifuge for 20 minutes to obtain the serum (the blood specimens were centrifuged and the serum carefully drawn into sample bottles and analysed immediately or stored frozen at -20 °C until the time for analysis).

Subjects were each given an apple to break their fast after blood collection, then filled a questionnaire for data about their socio-demographic background. They then proceeded with measurement of blood pressure and other anthropometric measurements.

#### **3.4.4 Physiological measurements**

Blood pressure (BP) was measured by use of a sphygmomanometer and stethoscope (auscultatory method). With participant seated for at least 5 minutes, the cuff was placed around the upper right arm (Plate XV). Two measurements taken after the participant had been seated and rested for 5 and 10 min. A third measurement taken if readings differed by more than 10%. The mean of the two readings or the median of three readings were calculated for both pressures.

The mid-point of the length bladder was placed over the brachial artery, and the mid height of the cuff was at height level. Lower edge of the cuff was placed, with its tubing connections, about 1" above the natural crease across the inner aspect of the elbow. The cuff was wrapped snugly about the arm, with the palm of the participant's hand turned upward.

The cuff was then inflated manually by squeezing a rubber bulb, until the artery is completely occluded, which was indicated by obliteration of the pulse, felt by palpation on the radial artery on the wrist. Then, the pressure in the cuff was slowly released by deflating the cuff. The pressure at which sounds produced by the arterial pulse waves (Korotkoff sounds) appear (systolic pressure) and disappear again as flow through the artery resumes (diastolic pressure), were noted.

### 3.5 Calculating the Anthropometric Somatotype

Anthropometric somatotype was computed according to Carter and Heath (1990). The predictive equations to calculate endomorphy, mesomorphy and ectomorphy were as follows:

$$\text{Endomorphy} = -0.7182 + 0.1451 (X) - 0.00068 (X^2) + 0.0000014 (X^3),$$

Where X is the sum of triceps, subscapular and supraspinale skinfolds multiplied by 170.18/ height (cm).

$$\text{Mesomorphy} = (0.858 \times \text{humerus breadth}) + (0.601 \times \text{femur breadth}) + (0.188 \times \text{corrected arm girth}) + (0.161 \times \text{corrected calf girth}) - (\text{height} \times 0.131) + 4.5.$$

The arm and calf girths will be corrected by subtracting the triceps and calf skinfolds (in cm), respectively.

**Ectomorphy:** three different methods are used to calculate ectomorphy according to Height-

$$\text{Weight Ratios (HWR} = \text{height} / \sqrt[3]{\text{weight}})$$

If HWR is greater than or equal to 40.75, i.e.  $\text{HWR} \geq 40.75$ , then,

Ectomorphy =  $0.732 \times \text{HWR} - 28.58$ .

If HWR is less than 40.75 but greater than 38.25, i.e.  $\text{HWR} < 40.75 > 38.25$ , then, ectomorphy =  $0.463 \times \text{HWR} - 17.63$ .

If HWR is equal to or less than 38.25, i.e.

$\text{HWR} \leq 38.25$ , then Ectomorphy = 0.1

The somatotype calculation composed of: a) somatotype mean, b) the three components of somatotype (endomorph, ectomorph, and mesomorph) (Carter and Heath, 1990).

### **3.5.1 Interpreting anthropometric somatotype result**

Ratings on each component of  $\frac{1}{2}$  to  $2\frac{1}{2}$  are considered low, 3 to 5 are moderate,  $5\frac{1}{2}$  to 7 are high, and  $7\frac{1}{2}$  and above are very high (Carter & Heath, 1990). The rating is phenotypical, based on the concept of geometrical size-dissociation and applicable to both genders from childhood to old age.

#### **3.5.1.1 Plotting the Somatotype**

Traditionally, the three-number somatotype rating is plotted on a two-dimensional somatochart using X,Y coordinates. The coordinates are calculated as follows:

$X = \text{ectomorphy} - \text{endomorph}$ ;

$Y = 2 \times \text{mesomorph} - (\text{endomorph} + \text{ectomorphy})$

These points on the somatochart are called somatoplots.

#### **3.5.1.2 Somatocharts**

The somatoplots on the somatochart provide an important visual display, either for individuals or means, and should be utilized routinely. The shape of the distribution of somatoplots of a sample imparts considerable useful information and should be preliminary to, or accompany, statistical analysis. It is vital to interpreting results.

### 3.5.1.3 *Somatotype categories*

Determine the frequencies of somatotypes in categories, e.g. balanced ectomorph, endomorph. When there are low frequencies in adjacent categories on the somatochart they can be combined for a better summary and analysis.

According to Carter and Heath (1990) somatotypes with similar relationships between the dominance of the components are grouped into categories named to reflect these relationships. The frequencies of somatotypes within categories (or combined categories) can be used to describe the overall distribution of samples or for comparing them using a Chi-square analysis. The definitions of 13 categories are based on the areas of the 2-D somatochart (Carter and Heath, 1990).

- i Central: no component differs by more than one unit from the other two.
- ii Balanced endomorph: endomorphy is dominant and mesomorphy and ectomorphy are equal (or do not differ by more than one-half unit).
- iii Mesomorphic endomorph: endomorphy is dominant and mesomorphy is greater than ectomorphy.
- iv Mesomorph-endomorph: endomorphy and mesomorphy are equal (or do not differ by more than one half unit) and ectomorphy is smaller.
- v Endomorphic mesomorph: mesomorphy is dominant and endomorphy is greater than ectomorphy.
- vi Balanced mesomorph: mesomorphy is dominant and endomorphy and ectomorphy are equal (or do not differ by more than one-half unit).
- vii Ectomorphic mesomorph: mesomorphy is dominant and ectomorphy is greater than endomorphy.
- viii Mesomorph-ectomorph: mesomorphy and ectomorphy are equal (or do not differ by more than one half unit) and endomorphy is smaller.

- ix Mesomorphic ectomorph: ectomorphy is dominant and mesomorphy is greater than endomorphy.
- x Balanced ectomorph: ectomorphy is dominant and endomorphy and mesomorphy are equal (or do not differ by more than one-half unit).
- xi Endomorphic ectomorph: ectomorphy is dominant and endomorphy is greater than mesomorphy.
- xii Endomorph-ectomorph: endomorphy and ectomorphy are equal (or do not differ by more than onehalf unit), and mesomorphy is lower.
- xiii Ectomorphic endomorph: endomorphy is dominant and ectomorphy is greater than mesomorphy.

The 13 categories can be simplified into four larger categories:

- a. Central: no component differs by more than one unit from the other two.
- b. Endomorph: endomorphy is dominant, mesomorphy and ectomorphy are more than one-half unit lower.
- c. Mesomorph: mesomorphy is dominant, endomorphy and ectomorphy are more than one-half unit lower.
- d. Ectomorph: ectomorphy is dominant, endomorphy and mesomorphy are more than one-half unit lower.

### **3.5.2 Body mass index (BMI)**

BMI is a measure of obesity, an index of weight relative to square of height– Quetelet index ( $\text{kg}/\text{m}^2$ ).

BMI normal range:  $18.5 - 24.9 \text{ kg}/\text{m}^2$ ; overweight:  $25.0 - 25.9 \text{ kg}/\text{m}^2$ ; obesity -  $> 30 \text{ kg}/\text{m}^2$ ; underweight –  $< 18.5 \text{ kg}/\text{m}^2$



### 3.6 Laboratory Analytical Methods

#### 3.6.1 Measurement of serum total cholesterol (TC)

Total cholesterol was determined by enzymatic endpoint method by using RANDOX assay kits (Randox Laboratories Ltd, United Kingdom Cat No. RX MONZA CH 210) as described by Allain *et al.* (1974)

##### 3.6.1.1 Principle

The cholesterol was determined after enzymatic hydrolysis and oxidation. CHOL ester was hydrolysed to free cholesterol (FC) and free fatty acid (FFA) by CHOL esterase (CHE) enzyme. The FC formed was oxidized by CHOL oxidase (CHO) to cholestene-3-one and hydrogen peroxide which reacted with 4-aminophenazone and phenol by phenol oxidoreductase (POD) and produced quinoneimine (coloured compound), the intensity of which was proportional to the concentration of TC present in the sample. The colour was read at 500 nm wavelength.

Cholesterol ester + H<sub>2</sub>O  $\xrightarrow{\text{Cholesterol esterase}}$  Cholesterol + Fatty acids

Cholesterol ester + O<sub>2</sub>  $\xrightarrow{\text{Cholesterol oxidase}}$  Cholestene-3-one + H<sub>2</sub>O<sub>2</sub>

##### 3.6.1.2 Procedure

Three test tubes were prepared for blank, test and standard. In each tube, 1000µl CHOL liquicolour reagent was placed and 10 µl distilled water, serum and CHOL standard solution was added to blank, test and standard respectively. The contents of the tubes were thoroughly mixed and incubated at room temperature for 10 minutes. The absorbance was measured against the reagent blank at 500 nm wavelength.

Serum TC concentration was calculated from the formula:

$$C_{(\text{sample})} = \frac{A_{(\text{Sample})} \times C_{(\text{standard})}}{A_{(\text{Standard})}}$$

$$= \frac{A_{(\text{Sample})} \times 5.17 \text{ mmol/L}}{A_{(\text{Standard})}}$$

Where A= absorbance, C = concentration, 5.17 mmol/L = Concentration of CHOL standard.

Normal values in serum:

< 5.17 mmol/L (200 mg/dL) =Desirable blood cholesterol

1.17 - 6.18 mmol/L (200 - 239 mg/dL) = Borderline - high blood cholesterol

≥ 6.20 mmol/L (240 mg/dL) = High blood cholesterol.

### 3.6.2 Measurement of serum HDL-cholesterol (HDL-C)

Serum HDL was measured by precipitation method (Burstein *et al.*, 1970) using RANDOX assay kits (Randox Laboratories Ltd, United Kingdom, Cat, No. CH 203).

#### 3.6.2.1 Principle

The chylomicrons, VLDL-C and LDL-C were precipitated by addition of phosphotungstic acid and magnesium chloride. After centrifugation the supernatant fluid containing HDL was assayed for HDL-C with the HUMAN CHOL liquicolour reagent.

#### 3.6.2.2 Procedure

A diluted precipitant (500 µl) was pipetted into centrifuge tube and 200 µl of sample added. The content was thoroughly mixed and incubated for 10 minutes at room temperature. The mixture was centrifuged for 5 minutes at 3,000 g. After centrifugation, the clear supernatant was separated from the precipitate within one hour and the CHOL concentration.

The results was obtained from the formula.

$$C_{(\text{sample})} = \frac{A_{(\text{Sample})} \times C_{(\text{standard})}}{A_{(\text{Standard})}}$$

$$= \frac{A_{(\text{Sample})} \times 4.52 \text{ mmol/L}}{A_{(\text{Standard})}}$$

Where A= absorbance, C = concentration, 4.52 mmol/L = Concentration of HDL-C standard

### 3.6.3 Measurement of serum triglycerides

Serum triglycerides were measured by colorimetric method (McGowan *et al.*, 2013), using RANDOX assay kits (Randox Laboratories Ltd, United Kingdom, Cat, No. RX MONZA TR 210).

#### 3.6.3.1 Principle

The TG was determined after enzymatic hydrolysis with lipases. Triglyceride was hydrolysed by lipases to glycerol and FFA. The glycerol reacted with adenosine triphosphate (ATP) to form glycerol-3-phosphate and adenosine diphosphate (ADP) under the influence of glycerol kinase (GK). Glycerol-3-phosphate oxidized by glycerol phosphate oxidase (GPO) to dihydroxyacetone and hydrogen peroxide. Hydrogen peroxide then combined with 4-aminoantipyrine to form quinoneimine (coloured compound), the intensity of which was proportional to the concentration of TG present in the sample. The coloured complex was read at 500 nm wavelength.

#### 3.6.3.2 Procedure

Three test tubes were prepared for blank, test and standard. In each tube, 1000 µl CHOL liquicolour reagent was placed and 10 µl distilled water, serum and TG standard solution added to blank, test and standard respectively. The contents of the tubes were mixed and incubated for 10 minutes at room temperature. The absorbance was measured against the reagent blank at 500 nm wavelength.

Serum TG concentration was obtained as follows:

$$\begin{aligned} C_{(\text{sample})} &= \frac{A_{(\text{Sample})} \times C_{(\text{standard})}}{A_{(\text{Standard})}} \\ &= \frac{A_{(\text{Sample})} \times 2.28 \text{ mmol/L}}{A_{(\text{Standard})}} \end{aligned}$$

Where A = Absorbance, C = Concentration, 2.28 mmol/L = Concentration of TG standard.

#### **3.6.4 Estimation of serum LDL-cholesterol**

Serum LDL-C concentrations was calculated from the TC, HDL-C and TG concentrations according to Friedewald formula (Friedewald *et al.*, 1972) as follows:

$$\text{LDL-C} = \text{TC} - (\text{HDL-C} + \text{VLDL-C}), \text{ where}$$

$$\text{VLDL-C} = \text{TG}/2.2 \text{ (mmol/L)}$$

#### **3.6.5 Estimation of serum VLDL-C**

Concentrations of serum VLDL-C was estimated according to Friedewald formula (Friedewald *et al.*, 1972) as shown below:

$$\text{VLDL-C} = \text{TG}/2.2 \text{ (mmol/L), when TG} < 4.0 \text{ mmol/L} \quad \text{or}$$

$$\text{TG}/5.0 \text{ (mmol/L), when TG} > 4.0 \text{ mmol/L}$$

#### **3.6.6 Determination of serum TC/HDL-C ratio**

Serum TC/HDL-C ratios (atherogenic indices) were determined as follows:

$$\text{TC/HDL-C} = \text{TC divided by HDL-C concentrations} \frac{(\text{TC})}{(\text{HDL-C})}$$

### **3.7 Statistical Analyses**

The descriptive statistics of the anthropometric variables were calculated for each group of different sex and age. Statistical analysis results were expressed as mean and standard deviation for continuous variables and as number and percentage (%) for qualitative variables. One way Analysis of Variance (ANOVA) was used to assess the differences in all parameters according to somatotype. Pearson correlation coefficient was used to check relationship in parameters in both males and females. Linear multiple regression was used to generate predictive blood pressure, body composition and biochemical profiles.

Statistical analysis was performed with statistical package for Service Solution (IBM SPSS statistics version 20 for Windows) and somatotype calculation and analysis (Carter, 2002).

Correlation coefficients were Pearson coefficients. ANOVA was performed in order to compare means between LDL and HDL subclasses. Statistical differences were considered significant when  $p$  was  $< 0.05$ .



**Plate I: Weighing balance with anthropometer (Angel, 150 MA; Istanbul/Turkey) for weight (kg) and height (cm) measurement**



**Plate II: Digital body fat caliper (Super Accurate Digital measurement; AccuFitness, Greenwood Village, CO) for skinfolds measurement in mm**



**Plate III: Digital sliding caliper (Holtain Ltd, United Kingdom) for measuring Bipicondylar breadth in mm.**





**Plate IV: Non-elastic measuring tape (Myotape; AccuFitness, Greenwood Village, Co) for circumferences measurement in cm**



**Plate V: Weight (kg) and height (cm) measurements with weighing balance and stadiometer**



**Plate VI: Measurement of biepicondylar breadth of humerus with digital sliding caliper in mm**



**Plate VII: Measurement of biepicondylar breadth of femur with digital sliding caliper in mm**



**Plate VIII: Measurement of upper arm girth (circumference) with non elastic measuring tape in cm**



**Plate IX: Measurement of calf girth (circumference) with non elastic measuring tape in cm**





**Plate X: Measurement of waist circumference with non elastic measuring tape in cm**



**Plate XI: Triceps skinfold thickness measurement with skinfold caliper in mm**





**Plate XII: Subscapular skinfold thickness measurement with skinfold caliper in mm**



**Plate XIII: Supraspinale skinfold thickness measurement with skinfold caliper in mm**



**Plate XIV: Thigh skinfold thickness measurement with skinfold caliper in mm**



**Plate XV: Blood pressure measurements with anaeroid sphygmomanometer and stethoscope (Littmann Brand Classic II S.E. Stethoscope (SEPO120B) and Agary<sup>(R)</sup> Sphygmomanometer mercurial 300 mmHg (Agary Pharmaceutical Limited, China)**

## CHAPTER FOUR

### 4.0 RESULTS

A total of 560 apparently healthy undergraduate students of Kaduna State University participated in the study, 379 (67.7%) males and 181 (32.3%) females, with age range of 17 - 33 years. The mean age for females was  $21.5 \pm 2.60$  years, while males was  $22.6 \pm 3.03$  years. The majority of the students were in age groups of 17 - 25 years. About 48% of the subjects were of Hausa ethnic group. Fig. 4.1 illustrated the strength and spread of ethnic group participants in the study.

#### 4.1 Anthropometric Characteristics of the Study Participants

Mean values and standard deviations of anthropometric variables according to sex are presented in Table 4.1. The mean age of males ( $22.6 \pm 3.03$ ) year and females ( $21.5 \pm 2.60$ ) year as shown in the table. Table 4.2 showed the comparison of anthropometric variables according to sex. Birth weight, body weight, height, biepicondylar breadth of humerus and femur; and upper arm girth had higher mean value in males than females and showed significance differences in various categories ( $p < 0.001$ ,  $p < 0.01$  and  $p < 0.05$ ) while hip circumference showed higher mean value in females than males with significance. The mean values for BMI ( $\text{kg/m}^2$ ) for both sexes were within reference range ( $21.50 \pm 2.03 \text{ kg/m}^2$  and  $21.74 \pm 1.93 \text{ kg/m}^2$  for females and males respectively).

#### 4.2 Body Composition Characteristics of the Study Population

Mean and standard deviations of skinfold thickness for the determination of body composition and according to sex are presented in Table 4.3. Females have higher mean values for skinfold measurements in all the sites measured except at suprailiac point. Table 4.4 presented the comparison of skinfold thickness according to sex. The skinfold thicknesses at the sites of triceps, suprascapular, medial calf and thigh showed significance.

### **4.3 Blood pressure and Lipid Profile of the Study Population**

Overall mean SBP and DBP for females was  $116.23 \pm 9.65$  mmHg and  $78.47 \pm 7.96$  mmHg respectively; and  $117.62 \pm 8.87$  and  $79.44 \pm 6.65$  respectively for males as presented in Table 4.5. The lipid profile shown on the table 4.6 illustrated higher mean values in females than males except the HDL-C. Most were within the range of desirable values.

### **4.4 Somatotypes of the Study Population**

The three somatotype components – endomorphy, mesomorphy and ectomorphy – were calculated according to the Heath-Carter anthropometric somatotyping method (Carter and Heath, 1990). The somatotype values for females were 2.50 - 3.07 - 2.54, while the males have 2.30 - 3.25 - 2.66 as shown on Table 4.7. Endomorphy mean value was significant between sex and higher in females than males as presented in Table 4.8.

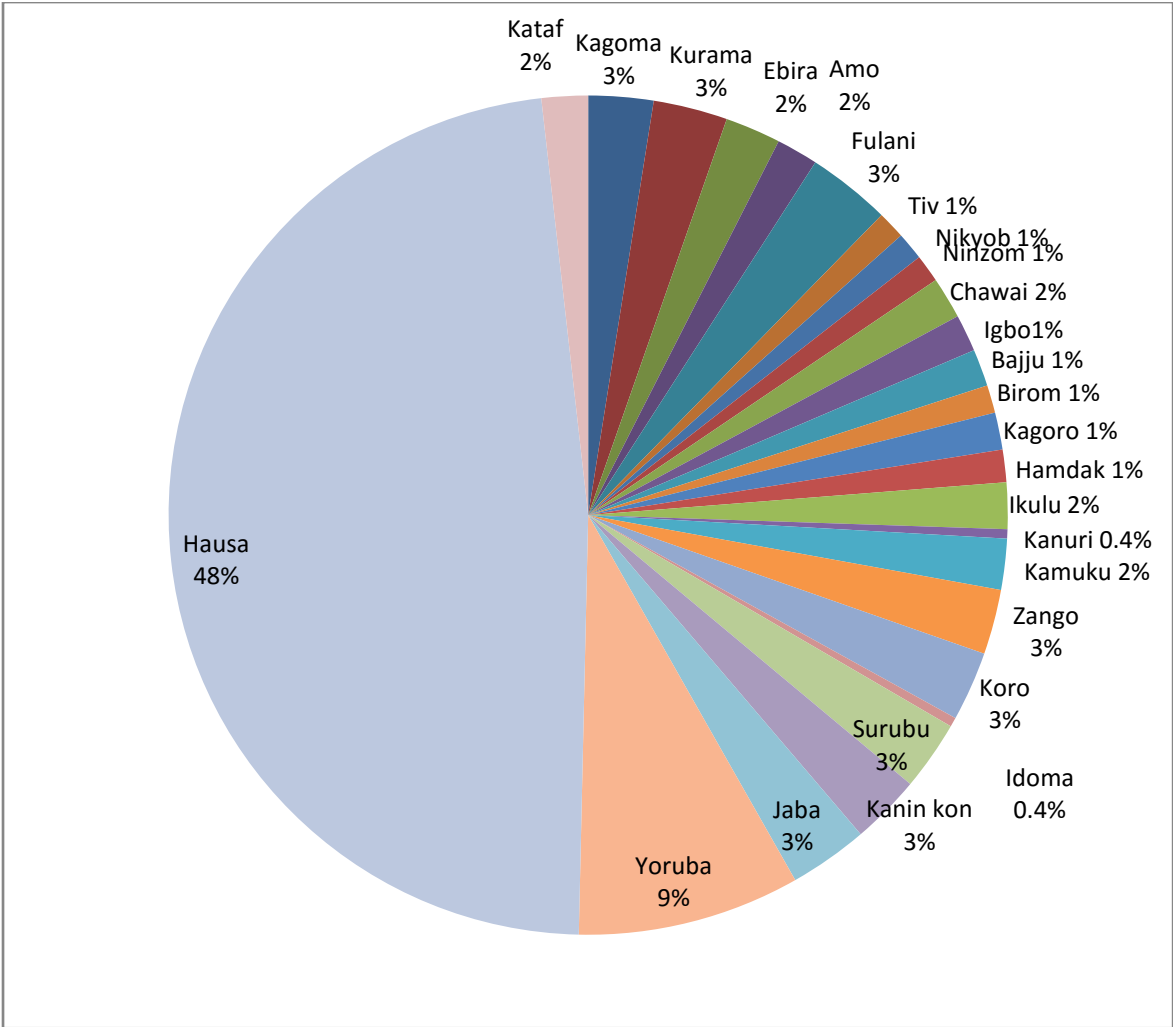


Figure 4.1: Ethnicity of all participants

**Table 4.1: Descriptive statistics of anthropometric variables according to sex**

Variables	Females (n=181)			Males (n=379)		
	Mean $\pm$ S.D	Min.	Max.	Mean $\pm$ S.D.	Min.	Max.
Age (year)	21.5 $\pm$ 2.60	17.00	30.00	22.6 $\pm$ 3.03	17.00	33.00
Birth Weight (kg)	3.09 $\pm$ 0.31	2.50	4.00	3.16 $\pm$ 0.36	2.50	5.00
Weight (kg)	58.64 $\pm$ 7.43	36.00	76.00	62.11 $\pm$ 6.83	47.00	95.00
Height (cm)	165.02 $\pm$ 6.95	141.00	182.00	168.98 $\pm$ 6.44	152.00	185.00
Body Mass Index (kg/m <sup>2</sup> )	21.50 $\pm$ 2.03	17.12	27.58	21.74 $\pm$ 1.93	16.73	31.38
Biepicondylar Breadth of Humerus (cm)	6.21 $\pm$ 0.71	5.08	8.50	6.56 $\pm$ 0.52	4.72	9.10
Biepicondylar Breadth Femur (cm)	8.22 $\pm$ 0.88	6.05	9.92	8.57 $\pm$ 0.71	5.61	11.20
Upper Arm Girth (cm)	26.39 $\pm$ 2.83	20.00	36.80	26.97 $\pm$ 2.24	20.60	33.60
Calf Girth (cm)	32.47 $\pm$ 3.25	24.30	39.70	32.93 $\pm$ 2.39	25.90	39.70
Waist Circumference (cm)	72.73 $\pm$ 6.89	52.10	90.00	72.39 $\pm$ 5.78	28.10	92.00
Hip Circumference (cm)	81.05 $\pm$ 6.88	63.00	105.00	78.46 $\pm$ 8.39	0.88	102.50

n-sample size, S.D. - Standard Deviation



**Table 4.2: Comparison of anthropometric variables according to sex**

Variables	Females (n=181)	Males (n=379)	t-value	p- value
	Mean ± S.D	Mean±S.D.		
Age (year)	21.5 ± 2.60	22.6 ± 3.03	0.32	0.234
Birth Weight (kg)	3.09±0.31	3.16±0.36	-2.25	0.025*
Weight (kg)	58.64±7.43	62.11±6.83	-5.32	0.000***
Height (cm)	165.02±6.95	168.98±6.44	-6.46	0.000***
Body Mass Index (kg/m <sup>2</sup> )	21.50±2.03	21.74±1.93	-1.36	0.174
Biepicondylar Breath of Humerus (cm)	6.21±0.71	6.56±0.52	-5.98	0.000***
Biepicondylar Breath Femur (cm)	8.22±0.88	8.57±0.71	-4.70	0.000***
Upper Arm Girth (cm)	26.39±2.83	26.97±2.24	-2.45	0.015*
Calf Girth (cm)	32.47±3.25	32.93±2.39	-1.71	0.088
Waist Circumference (cm)	72.73±6.89	72.39±5.78	0.58	0.561
Hip Circumference (cm)	81.05±6.88	78.46±8.39	3.88	0.000***

n- sample size, S.D. - Standard Deviation, \*\*\*p<0.001, \*\*p<0.01 and \*p<0.05

**Table 4.3: Skinfold thickness according to sex**

Variable	Females (n=181)			Males (n=379)		
	Mean±S.D	Min.	Max.	Mean± S.D.	Min.	Max.
Age (year)	21.5± 2.60	17.00	30.00	22.6 ± 3.03	17.00	33.00
Triceps skinfold (mm)	8.14±1.41	3.80	13.50	7.79±1.69	3.50	11.30
Subscapular skinfold (mm)	7.97±1.19	3.90	12.60	7.82±1.25	3.50	11.60
Supraspinale skinfold (mm)	8.09±0.97	3.80	10.30	7.45±1.44	3.50	9.90
Medial calf skinfold (mm)	8.61±1.09	5.70	13.70	8.04±1.20	3.80	9.90
Bicep skinfold (mm)	7.62±1.24	3.60	9.80	7.43±1.80	3.70	9.90
Abdominal skinfold (mm)	7.82±1.06	3.70	9.90	7.63±1.39	3.70	9.80
Suprailiac skinfold (mm)	6.91±1.67	3.00	9.60	7.02±1.69	1.00	11.00
Thigh skinfold (mm)	7.69±1.15	4.50	9.80	7.13±1.57	3.40	9.80

n- sample size, S.D. - Standard Deviation

**Table 4.4: Comparison of skinfold thickness according to sex**

Variable	Females (181) Mean±S.D	Males (n=379) Mean± S.D.	t-value	p-value
Age (year)	21.5± 2.60	22.6 ± 3.03	0.32	0.234
Triceps skinfold (mm)	8.14±1.41	7.79±1.69	2.56	0.011**
Subscapular skinfold (mm)	7.97±1.19	7.82±1.25	1.33	0.183
Supraspinale skinfold (mm)	8.09±0.97	7.45±1.44	6.20	0.000***
Medial calf skinfold (mm)	8.61±1.09	8.04±1.20	5.58	0.000***
Bicep skinfold (mm)	7.62±1.24	7.43±1.80	1.48	0.139
Abdominal skinfold (mm)	7.82±1.06	7.63±1.39	1.75	0.081
Suprailiac skinfold (mm)	6.91±1.67	7.02±1.69	-0.78	0.434
Thigh skinfold (mm)	7.69±1.15	7.13±1.57	4.75	0.000***

n- sample size, S.D. - Standard Deviation, \*\*\*p<0.001, \*\*p<0.01 and \*p<0.05

**Table 4.5: Blood pressure and lipid profiles according to sex**

Variable	Females (n=181)			Males (n=379)		
	Mean $\pm$ S.D	Min.	Max.	Mean $\pm$ S.D.	Min.	Max.
Age (year)	21.5 $\pm$ 2.60	17.00	30.00	22.6 $\pm$ 3.03	17.00	33.00
Systolic Blood Pressure(mmHg)	116.23 $\pm$ 9.65	100.00	140.00	117.62 $\pm$ 8.87	100.00	140.00
Diastolic Blood Pressure (mmHg)	78.47 $\pm$ 7.96	60.00	97.00	79.44 $\pm$ 6.65	67.00	96.00
High Density Lipoprotein Cholesterol (mmol/l)	2.59 $\pm$ 0.71	1.15	4.16	2.61 $\pm$ 0.77	0.83	4.52
Low Density Lipoprotein Cholesterol (mmol/l)	0.91 $\pm$ 1.15	-2.31	3.99	0.83 $\pm$ 1.07	-2.66	4.17
Very Low Density Lipoprotein - C. (mmol/l)	1.12 $\pm$ 0.29	0.40	2.32	1.08 $\pm$ 0.31	0.36	3.40
Triglyceride (mmol/l)	2.49 $\pm$ 0.61	1.22	5.11	2.47 $\pm$ 0.61	0.94	7.49
Total Cholesterol (mmol/l)	4.62 $\pm$ 0.84	2.15	7.52	4.52 $\pm$ 0.79	2.38	6.74
Atherogenic Index (TC/HDL-C)	1.95 $\pm$ 0.77	0.79	5.42	1.91 $\pm$ 0.75	0.57	7.04

n- sample size, S.D. - Standard Deviation

**Table 4.6: Comparison of blood pressure and lipid profiles according to sex**

Variable	Females (n=181)	Males (n=379)	t-value	p-value
	Mean ± S.D	Mean ± S.D.		
Age (year)	21.5 ± 2.60	22.6 ± 3.03	0.32	0.234
Systolic Blood Pressure (mmHg)	116.23±9.65	117.62±8.87	-1.64	0.101
Diastolic Blood Pressure (mmHg)	78.47±7.96	79.44±6.65	-1.42	0.157
High Density Lipoprotein Cholesterol (mmol/l)	2.59±0.71	2.61±0.77	-0.27	0.785
Low Density Lipoprotein Cholesterol (mmol/l)	0.91±1.15	0.83±1.07	0.84	0.404
Very Low Density Lipoprotein - C. (mmol/l)	1.12±0.29	1.08±0.31	1.23	0.219
Triglyceride (mmol/l)	2.49±0.61	2.47±0.61	0.51	0.607
Total Cholesterol (mmol/l)	4.62±0.84	4.52±0.79	1.34	0.181
Atherogenic Index (TC/HDL-C)	1.95±0.77	1.91±0.75	0.64	0.525

n- sample size, S.D. - Standard Deviation, \*\*\*p<0.001, \*\*p<0.01 and \*p<0.05

**Table 4.7: Somatotypes according to Sex**

Variable	Females (n=181)			Males (n=379)		
	Mean $\pm$ S.D	Min.	Max.	Mean $\pm$ S.D.	Min.	Max.
Age (year)	21.5 $\pm$ 2.60	17.00	30.00	22.6 $\pm$ 3.03	17.00	33.00
Endomorphy	2.50 $\pm$ 0.28	1.40	3.54	2.30 $\pm$ 0.34	1.37	3.20
Mesomorphy	3.07 $\pm$ 1.40	-0.67	7.52	3.25 $\pm$ 1.08	-0.06	6.38
Ectomorphy	2.54 $\pm$ 1.09	0.10	5.00	2.66 $\pm$ 1.07	-0.39	6.11

n- sample size, S.D. - Standard Deviation

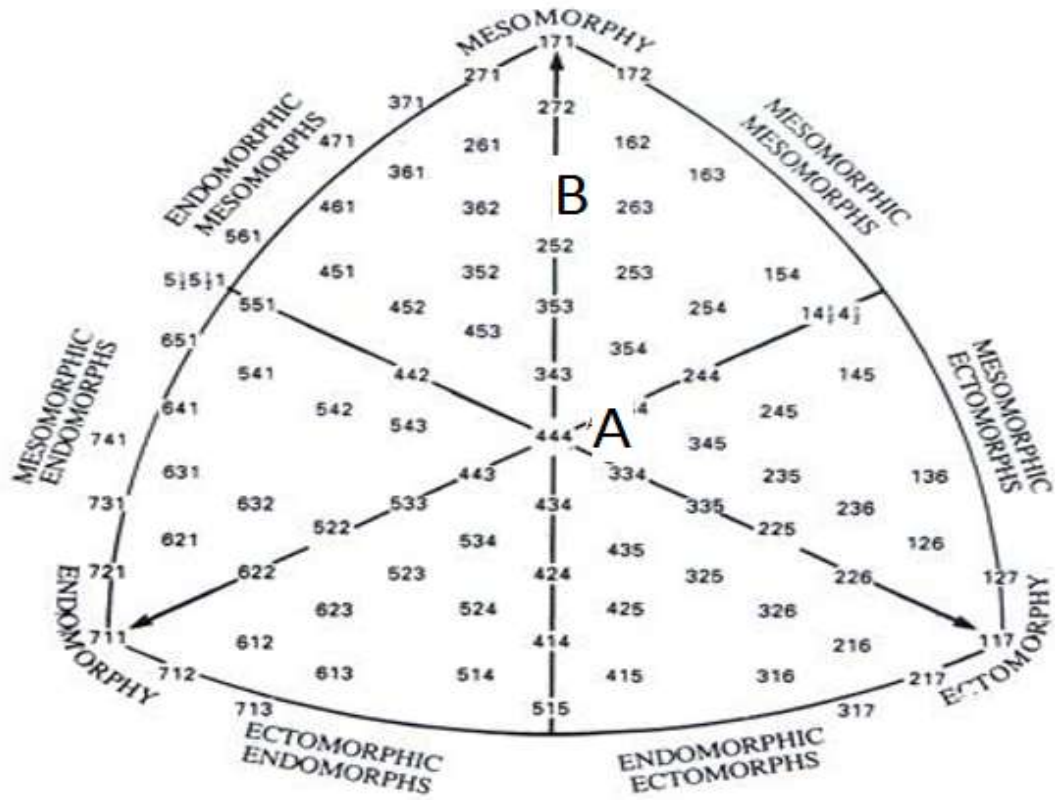


Figure 4.2: Somatotype according to sex  
 A – females, B – males

**Table 4.8: Comparison of somatotypes according to Sex**

Variable	Females (n=181)	Males (n=379)	t-value	p-value
	Mean $\pm$ S.D	Mean $\pm$ S.D.		
Age (year)	21.5 $\pm$ 2.60	22.6 $\pm$ 3.03	0.32	0.234
Endomorphy	2.50 $\pm$ 0.28	2.30 $\pm$ 0.34	7.28	<0.000
Mesomorphy	3.07 $\pm$ 1.40	3.25 $\pm$ 1.08	-1.53	0.127
Ectomorphy	2.54 $\pm$ 1.09	2.66 $\pm$ 1.07	-1.21	0.228

n- sample size, S.D. - Standard Deviation, \*\*\*p<0.001, \*\*p<0.01 and \*p<0.05



#### **4.5 Descriptive Statistic of the Study Population according to Ethnicity**

The descriptive statistics of females and males variables according to ethnic background were presented in Tables 4.9 and 4.10 respectively. Mean value of weight, BMI, calf girth, waist circumference and systolic blood pressure were of significance values in females while weight and BMI recorded significance difference in males. Table 4.11 summarized females and males somatotype according to ethnic groups. The subjects were grouped as central and balanced mesomorph according to the study.

**Table 4.9: Parameters of females population according to ethnicity**

Variable	Hausa (n=105) Mean $\pm$ S.D	Yoruba (n=21) Mean $\pm$ S.D	Others (n=55) Mean $\pm$ S.D.
Age (year)	21.64 $\pm$ 2.47	21.67 $\pm$ 3.10	21.02 $\pm$ 2.64
Birth weight (kg)	3.13 $\pm$ 0.33	3.02 $\pm$ 0.32	3.04 $\pm$ 0.25
Weight (kg)	56.38 $\pm$ 6.67	61.24 $\pm$ 8.35 <sup>c</sup>	61.95 $\pm$ 7.00 <sup>a</sup>
Height (cm)	164.20 $\pm$ 7.44	165.90 $\pm$ 7.65	166.24 $\pm$ 5.43
Body Mass Index, BMI (kg/m <sup>2</sup> )	20.90 $\pm$ 1.97	22.16 $\pm$ 1.69 <sup>b</sup>	22.37 $\pm$ 1.87 <sup>a</sup>
Biepic. Breadth of Humerus (cm)	6.17 $\pm$ 0.69	6.34 $\pm$ 0.84	6.24 $\pm$ 0.68
Biepic. Breadth of Femur (cm)	8.15 $\pm$ 0.82	8.11 $\pm$ 1.08	8.40 $\pm$ 0.89
Upper Arm Girth (cm)	26.04 $\pm$ 2.69	27.23 $\pm$ 3.83	26.72 $\pm$ 2.60
Calf Girth (cm)	31.75 $\pm$ 3.15	33.58 $\pm$ 4.02	33.41 $\pm$ 2.79 <sup>b</sup>
Waist circumference (cm)	71.52 $\pm$ 5.65	69.31 $\pm$ 8.75	76.37 $\pm$ 6.89 <sup>a</sup>
Hip circumference (cm)	80.93 $\pm$ 6.83	78.23 $\pm$ 8.40	82.34 $\pm$ 6.08
Triceps skinfold (mm)	8.15 $\pm$ 1.34	7.75 $\pm$ 1.78	8.25 $\pm$ 1.40
Subscapular skinfold (mm)	7.89 $\pm$ 1.23	8.23 $\pm$ 1.25	8.02 $\pm$ 1.07
Supraspinale skinfold (mm)	8.19 $\pm$ 0.86	7.87 $\pm$ 0.77	7.99 $\pm$ 1.21
Medial calf skinfold (mm)	8.58 $\pm$ 1.10	8.72 $\pm$ 1.40	8.63 $\pm$ 0.94
Bicep skinfold (mm)	7.49 $\pm$ 1.13	7.08 $\pm$ 1.90	8.08 $\pm$ 0.99
Abdominal skinfold (mm)	8.03 $\pm$ 0.86	7.71 $\pm$ 1.19	7.45 $\pm$ 1.25
Suprailiac skinfold (mm)	6.68 $\pm$ 1.81	6.92 $\pm$ 1.71	7.34 $\pm$ 1.26
Thigh skinfold (mm)	7.71 1.11	7.36 0.96	7.76 1.28
Systolic Blood Pressure (mmHg)	114.79 $\pm$ 9.10	115.76 $\pm$ 10.91	119.18 $\pm$ 9.68 <sup>c</sup>
Diastolic Blood Pressure (mmHg)	77.39 $\pm$ 7.81	81.10 $\pm$ 8.94	79.53 $\pm$ 7.61
HDL-C (mmol/l)	2.59 $\pm$ 0.71	2.80 $\pm$ 0.68	2.50 $\pm$ 0.72
LDL-C (mmol/l)	0.84 $\pm$ 1.12	0.79 $\pm$ 1.12	1.09 $\pm$ 1.22
VLDL-C (mmol/l)	1.11 $\pm$ 0.27	1.18 $\pm$ 0.33	1.13 $\pm$ 0.33
Triglyceride (mmol/l)	2.46 $\pm$ 0.55	2.66 $\pm$ 0.63	2.50 $\pm$ 0.69
Total Cholesterol, TC (mmol/l)	4.54 $\pm$ 0.77	4.77 $\pm$ 1.11	4.72 $\pm$ 0.86
Atherogenic index (TC:HDL-C)	1.91 $\pm$ 0.69	1.82 $\pm$ 0.65	2.08 $\pm$ 0.93

Biepic. - Biepicondylar, HDL-C - High Density Lipoprotein Cholesterol, LDL-C -Low Density Lipoprotein Cholesterol, VLDL-C - Very Low Density Lipoprotein Cholesterol, n- sample size, S.D. - Standard Deviation, a- p<0.001, b- p<0.01, c- p<0.05.

**Table 4.10: Parameters of males population according to ethnicity**

Variable	Hausa (n=183) Mean $\pm$ S.D	Yoruba (n=27) Mean $\pm$ S.D	Others (n=169) Mean $\pm$ S.D
Age (year)	22.19 $\pm$ 2.64	21.89 $\pm$ 2.76	23.27 $\pm$ 3.35
Birth weight (kg)	3.14 $\pm$ 0.39	3.18 $\pm$ 0.30	3.18 $\pm$ 0.34
Weight (kg)	60.76 $\pm$ 7.23	64.26 $\pm$ 9.94	63.24 $\pm$ 5.39 <sup>a</sup>
Height (cm)	168.10 $\pm$ 6.19	169.00 $\pm$ 5.65	169.92 $\pm$ 6.71
Body Mass Index, BMI (kg/m <sup>2</sup> )	21.48 $\pm$ 2.05	22.41 $\pm$ 2.56 <sup>c</sup>	21.91 $\pm$ 1.61
Biepic. Breadth of Humerus (cm)	6.56 $\pm$ 0.54	6.63 $\pm$ 0.45	6.56 $\pm$ 0.51
Biepic. Breadth of Femur (cm)	8.53 $\pm$ 0.78	8.79 $\pm$ 1.03	8.59 $\pm$ 0.54
Upper Arm Girth (cm)	26.67 $\pm$ 2.45	27.67 $\pm$ 2.24	27.19 $\pm$ 1.94
Calf Girth (cm)	32.64 $\pm$ 2.60	32.72 $\pm$ 2.32	33.29 $\pm$ 2.11
Waist circumference (cm)	71.65 $\pm$ 6.79	73.95 $\pm$ 6.95	72.94 $\pm$ 4.06
Hip circumference (cm)	78.32 $\pm$ 7.11	81.73 $\pm$ 7.62	78.59 $\pm$ 7.59
Triceps skinfold (mm)	7.78 $\pm$ 1.65	8.48 $\pm$ 1.27	7.69 $\pm$ 1.77
Subscapular skinfold (mm)	7.84 $\pm$ 1.27	7.97 $\pm$ 1.17	7.78 $\pm$ 1.24
Supraspinale skinfold (mm)	7.50 $\pm$ 1.34	7.56 $\pm$ 1.63	7.37 $\pm$ 1.53
Medial calf skinfold (mm)	7.99 $\pm$ 1.07	7.95 $\pm$ 1.16	8.13 $\pm$ 1.33
Bicep skinfold (mm)	7.48 $\pm$ 1.79	7.75 $\pm$ 1.57	7.32 $\pm$ 1.85
Abdominal skinfold (mm)	7.57 $\pm$ 1.43	7.77 $\pm$ 1.24	7.68 $\pm$ 1.37
Suprailiac skinfold (mm)	6.85 $\pm$ 1.69	7.12 $\pm$ 1.82	7.20 $\pm$ 1.65
Thigh skinfold (mm)	7.07 1.56	7.22 1.87	7.16 1.55
Systolic Blood Pressure (mmHg)	116.24 $\pm$ 7.47	119.97 $\pm$ 9.87	118.63 $\pm$ 9.90
Diastolic Blood Pressure (mmHg)	78.59 $\pm$ 6.11	83.07 $\pm$ 6.79	79.78 $\pm$ 6.98
HDL-C (mmol/l)	2.62 $\pm$ 0.75	2.81 $\pm$ 0.80	2.57 $\pm$ 0.79
LDL-C (mmol/l)	0.76 $\pm$ 1.00	0.69 $\pm$ 1.22	0.92 $\pm$ 1.11
VLDL-C (mmol/l)	1.13 $\pm$ 0.35	1.06 $\pm$ 0.22	1.05 $\pm$ 0.27
Triglyceride (mmol/l)	2.54 $\pm$ 0.70	2.33 $\pm$ 0.48	2.40 $\pm$ 0.51
Total Cholesterol, TC (mmol/l)	4.50 $\pm$ 0.76	4.55 $\pm$ 0.99	4.54 $\pm$ 0.79
Atherogenic index (TC:HDL-C)	1.87 $\pm$ 0.69	1.81 $\pm$ 0.93	1.96 $\pm$ 0.78

Biepic. - Biepicondylar, HDL-C - High Density Lipoprotein Cholesterol, LDL-C -Low Density Lipoprotein Cholesterol, VLDL-C - Very Low Density Lipoprotein Cholesterol, n- sample size, S.D. - Standard Deviation, a- p<0.001, b- p<0.01, c- p<0.05.

**Table 4.11: Somatotype according to ethnic group**

Ethnicity	n	Endomorphy Mean $\pm$ S.D	Mesomorphy Mean $\pm$ S.D	Ectomorphy Mean $\pm$ S.D	Category
<b>Females</b>					
Hausa	105	2.52 $\pm$ 0.27	2.91 $\pm$ 1.40	2.81 $\pm$ 1.15	Central
Others	55	2.48 $\pm$ 0.31	3.26 $\pm$ 1.20	2.12 $\pm$ 0.90 <sup>b</sup>	Balanced mesomorph
Yoruba	21	2.44 $\pm$ 0.30	3.34 $\pm$ 1.83	2.30 $\pm$ 0.83	Balanced mesomorph
Total	181				
<b>Males</b>					
Hausa	183	2.32 $\pm$ 0.33	3.23 $\pm$ 1.14	2.78 $\pm$ 1.10	Central
Others	169	2.26 $\pm$ 0.35	3.22 $\pm$ 1.01	2.58 $\pm$ 1.05	Central
Yoruba	27	2.41 $\pm$ 0.25	3.54 $\pm$ 1.10	2.39 $\pm$ 1.03	Balanced mesomorph
Total	379				

n- sample size, S.D. - Standard Deviation, a-  $p < 0.001$ , b-  $p < 0.01$ , c-  $p < 0.05$ .

#### **4.6 Age and Sex Variation of Students Population**

Descriptive statistics of students population according to age groups were presented in Table 4.12 (females), Table 4.13 (males) and Table 4.14 (somatotype and blood pressure according to age groups). The mean and standard deviation of different age groups were shown.

Most of the anthropometric variables had higher mean values in females than males, the skinfold thickness were higher in 30 - 33 years age group of both sexes than any other group except for subscapular skinfold which shown higher mean value in 26 - 29 years age group ( $9.27 \pm 1.57$ ) (Table 4.12 and 4.13). The anthropometric variables of weight, biepicondylar breadth of femur and humerus; and upper arm and calf girth of female in the age range of 26 - 29 years were of higher values than any age group when compared with male (Table 4.12 and Table 4.13).

The mean values of the three somatotype components are presented by age group in Table 4.14. Endomorphy showed similar values in all four age groups and the highest mean value was recorded in 30-33 age group in females and 22-25 in males (Table 4.14). Mesomorphy had a higher mean value in females ( $4.37 \pm 1.92$ ), while the highest mean somatotypic value recorded in males was ectomorphy ( $3.46 \pm 0.37$ ). Endomorphy and mesomorphy increases with age in females and were relatively higher in females than in males. Males appeared to have higher mean ectomorphy values than females. Mesomorphy, had the lowest mean value among the youngest group of females aged 17-21 and the highest was recorded in 26-29 years age group, while 30-33 and 22-25 age group in males respectively (Table 4.14).

Analysis among various age groups in the lipid profile of participants showed that females had significantly higher mean systolic ( $132.00 \pm 3.46$ ; 30 - 33 years age group) and diastolic ( $86.29 \pm 5.35$ ; 26 - 29 years age group) blood pressure than males (Table 4.15). However, SBP in females increase along the age group. Statistically significant difference were found among

the age groups in females along height, biepicondylar breadth of humerus, hip circumference, triceps, subscapular and medial calf skinfolds (Table 4.12); SBP and DBP (Table 4.14). The lipid profiles according to age group were presented in Table 4.15.

Table 4.16 presented the comparison of mean somatotype components within BMI classification (underweight, normal weight and overweight). Among both sexes, endomorphy and mesomorphy components were found to be significantly higher among those with the prevalence of overweight and significantly lower among the underweight children. Ectomorphy, on the other hand, was found to be significantly higher among those with the prevalence of underweight and lower among overweight children. From this Table, there was a significant association between somatotype components and BMI among the studied groups. The proportions of adolescents with underweight, healthy and overweight by BMI categorization were 7.18%, 88.95% and 3.87% respectively among females and 3.69%, 90.50% and 5.80% respectively among males (Table 4.16) and this was statistically significant ( $p < 0.001$ ).

**Table 4.12: Anthropometric variables of female students population according to age group**

Variable	17 - 21 years (n=92)	22 - 25 years (n=79)	26 - 29 years (n=7)	30 - 33 years (n=3)
	Mean $\pm$ S.D	Mean $\pm$ S.D	Mean $\pm$ S.D	Mean $\pm$ S.D
Birth weight (kg)	3.11 $\pm$ 0.29	3.09 $\pm$ 0.33	3.19 $\pm$ 0.27	3.20 $\pm$ 0.20
Weight (kg)	59.12 $\pm$ 7.25	58.77 $\pm$ 6.56	65.71 $\pm$ 5.47	65.33 $\pm$ 5.51
Height (cm)	165.10 $\pm$ 5.78	166.53 $\pm$ 7.22 <sup>c</sup>	170.43 $\pm$ 5.42 <sup>c</sup>	169.00 $\pm$ 3.61
Body mass index (kg/m <sup>2</sup> )	22.42 $\pm$ 2.18	21.17 $\pm$ 1.69	22.73 $\pm$ 1.50	22.84 $\pm$ 1.02
Biepicondylar breadth of humerus (cm)	6.17 $\pm$ 0.63	6.31 $\pm$ 0.77	7.04 $\pm$ 0.80 <sup>b</sup>	6.48 $\pm$ 0.44
Biepicondylar breadth of femur (cm)	8.15 $\pm$ 0.19	8.33 $\pm$ 0.76	8.84 $\pm$ 0.51	8.08 $\pm$ 0.50
Upper arm girth (cm)	26.19 $\pm$ 2.53	26.74 $\pm$ 2.77	28.84 $\pm$ 4.14	27.63 $\pm$ 1.27
Calf girth (cm)	32.53 $\pm$ 3.17	33.13 $\pm$ 2.81	34.94 $\pm$ 3.73	33.17 $\pm$ 1.26
Waist circumference (cm)	74.76 $\pm$ 7.68	72.44 $\pm$ 5.67	75.26 $\pm$ 3.89	79.57 $\pm$ 8.99
Hip circumference (cm)	80.45 $\pm$ 7.62	81.49 $\pm$ 6.02	87.93 $\pm$ 4.31 <sup>c</sup>	83.40 $\pm$ 3.84
Triceps skinfold (mm)	7.85 $\pm$ 1.64	8.58 $\pm$ 1.18 <sup>a</sup>	7.87 $\pm$ 2.02	9.41 $\pm$ 1.91
Subscapular skinfold (mm)	7.93 $\pm$ 1.12	8.13 $\pm$ 1.21	9.27 $\pm$ 1.57 <sup>a</sup>	9.03 $\pm$ 1.31
Supraspinale skinfold (mm)	8.54 $\pm$ 0.89	7.95 $\pm$ 1.06	7.54 $\pm$ 1.96	8.20 $\pm$ 1.04
Medial calf skinfold (mm)	8.79 $\pm$ 0.65	8.47 $\pm$ 1.12	9.76 $\pm$ 2.76 <sup>c</sup>	10.00 $\pm$ 1.38
Bicep skinfold (mm)	8.01 $\pm$ 1.43	7.54 $\pm$ 1.08	7.86 $\pm$ 1.94	8.57 $\pm$ 0.12
Abdominal skinfold (mm)	7.82 $\pm$ 1.27	8.03 $\pm$ 0.97	7.10 $\pm$ 0.89	7.93 $\pm$ 0.68
Suprailiac skinfold (mm)	7.48 $\pm$ 1.51	6.61 $\pm$ 1.82	6.41 $\pm$ 1.52	7.67 $\pm$ 0.12
Thigh skinfold (mm)	7.60 $\pm$ 1.21	7.66 $\pm$ 1.15	8.29 $\pm$ 1.21	8.67 $\pm$ 0.12

S.D. - Standard Deviation, n- sample size, a- p<0.001, b- p<0.01, c- p<0.05

**Table 4.13: Anthropometric variables of male students population according to age group**

Variable	17 - 21 years (n=144) Mean ± S.D	22 - 25 years (n=165) Mean ± S.D.	26 - 29 years (n=66) Mean ± S.D	30 - 33 years (n=4) Mean ± S.D
Birth weight (kg)	3.10± 0.32	3.17± 0.40	3.17± 0.39	2.87± 0.35
Weight (kg)	62.75± 7.21	61.71± 6.32	64.41± 6.05	60.75± 2.22
Height (cm)	166.03± 6.49	168.52± 6.75	171.12± 5.17	172.00± 2.00
Body mass index (kg/m <sup>2</sup> )	21.51± 2.62	21.72± 1.77	22.00± 1.92	20.54± 0.64
Biepicondylar breadth of humerus (cm)	6.72± 0.71	6.59± 0.51	6.69± 0.46	6.67± 0.14
Biepicondylar breadth of femur (cm)	8.63± 0.77	8.56± 0.69	8.66± 0.54	8.08± 0.50
Upper arm girth (cm)	27.14± 2.57	27.18± 2.08	27.18± 2.49	26.88± 0.71
Calf girth (cm)	32.62± 2.40	32.88± 2.32	33.53± 2.39	33.50± 2.04
Waist circumference (cm)	71.99± 5.13	72.33± 4.20	72.99± 9.26	72.60± 2.22
Hip circumference (cm)	77.59± 5.21	79.28± 4.68	77.71± 13.48	80.83± 1.12
Triceps skinfold (mm)	7.88± 1.04	7.69± 1.62	7.79± 2.01	8.43± 0.55
Subscapular skinfold (mm)	7.77± 1.35	7.99± 1.32	7.74± 1.24	6.58± 1.01
Supraspinale skinfold (mm)	7.48± 1.29	7.59± 1.40	7.23± 1.68	8.58± 1.93
Medial calf skinfold (mm)	8.13± 1.30	8.10± 1.23	8.04± 0.99	8.70± 0.35
Bicep skinfold (mm)	7.65± 1.71	7.73± 1.71	7.53± 1.88	5.43± 2.29
Abdominal skinfold (mm)	7.53± 1.41	7.70± 1.29	7.55± 1.60	8.75± 0.99
Suprailiac skinfold (mm)	6.77± 1.42	7.20± 1.47	6.88± 2.24	4.98± 1.94
Thigh skinfold (mm)	7.76± 1.52	7.22± 1.61	6.99± 1.73	7.03± 1.32

S.D. - Standard Deviation, n- sample size, a- p<0.001, b- p<0.01, c- p<0.05



**Table 4.14: Descriptive statistics of somatotype components and blood pressure by age group and sex**

Age groups	N	ENDO	MESO	ECTO	SBP (mmHg)	DBP (mmHg)	
		Mean±SD	Mean±SD	Mean±SD	Mean ± SD	Mean ± SD	
<b>Females</b>							
<b>17 - 21</b>	92	2.47± 0.29	2.83± 1.57	2.35± 1.17	114.76± 9.11	77.45± 7.28	Central
<b>22 - 25</b>	79	2.53± 0.26	3.18± 1.38	2.76± 1.17	117.08± 9.28	79.33± 8.44	Central
<b>26 - 29</b>	7	2.46± 0.61	4.37± 1.92	2.28± 0.82	124.29± 7.87	86.29± 5.35 <sup>c</sup>	Ectomorphic- mesomorph
<b>30 - 33</b>	3	2.70± 0.41	3.02± 0.27	2.16± 0.27	132.00± 3.46 <sup>c</sup>	84.00± 5.29	Mesomorph- endomorph
<b>Males</b>							
<b>17 - 21</b>	144	2.27± 0.22	3.17± 1.33	2.65± 1.11	118.23± 8.88	79.21± 6.71	Central
<b>22 - 25</b>	165	2.33± 0.33	3.35± 1.02	2.64± 1.05	117.48± 8.79	79.33± 6.84	Central
<b>26 - 29</b>	66	2.22± 0.36	3.26± 1.23	2.66± 1.04	117.65± 8.96	79.94± 6.21	Central
<b>30 - 33</b>	4	2.31± 0.14	2.69± 0.42	3.46± 0.37	120.00± 0.00	77.50± 5.00	Balanced ectomorph

S.D. - Standard Deviation, n- sample size, a- p<0.001, b- p<0.01, c- p<0.05, p<0.05 when compared with 17 - 21 year age group, ENDO- Endomorphy, MESO - Mesomorphy, ECTO - Ectomorphy, SBP - Systolic Blood Pressure, DBP - Diastolic Blood Pressure.

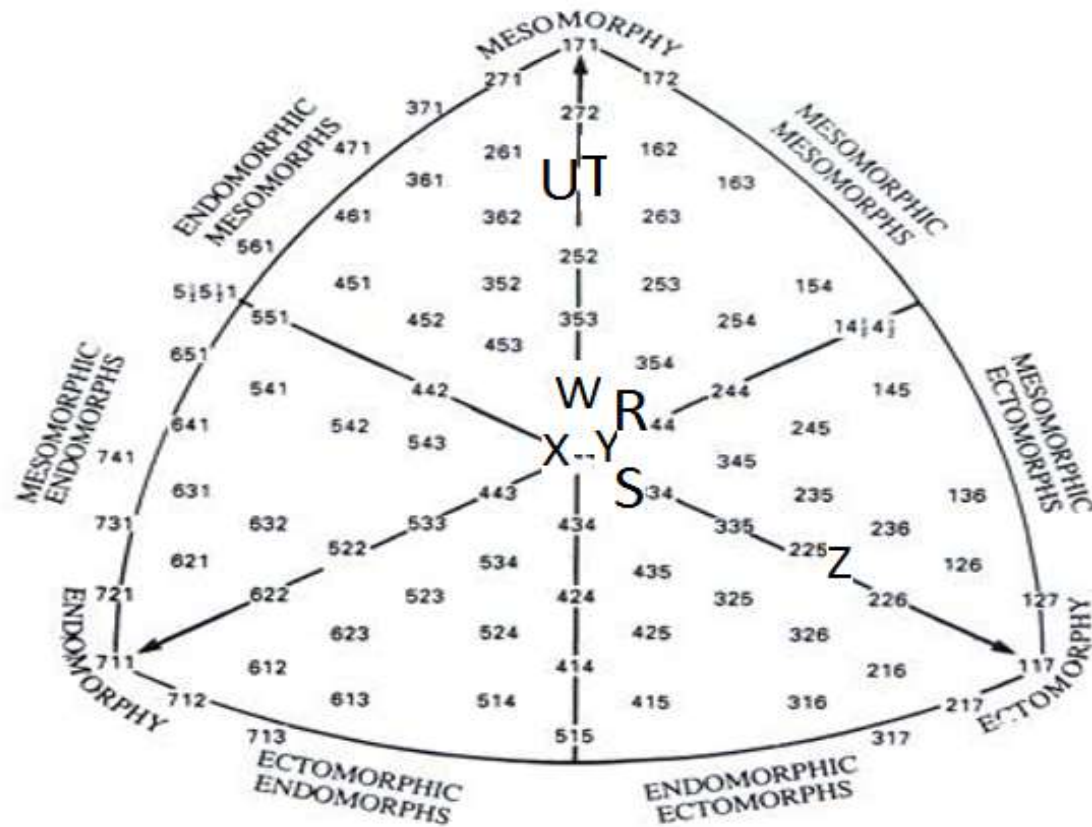


Figure 4.3: Somatotype components by age group and sex

Females – R (18-21yrs), S (22-25yrs), T (26-29yrs) & U (30 -33yrs)

Males – W (18-21yrs), X (22-25yrs), Y (26-29yrs) & Z (30 -33yrs)

**Table 4.15: Lipid profiles according to age group**

Lipid profile	17 - 21 years	22 - 25 years	26 - 29 years	30 - 33 years
	(n=92)	(n=79)	(n=7)	(n=3)
	Mean ± S.D	Mean ± S.D	Mean ± S.D	Mean ± S.D
<b>Females</b>				
HDL-C (mmol/L)	2.63± 0.73	2.56± 0.74	2.58± 0.48	2.04± 0.84
LDL-C (mmol/L)	0.85± 1.21	0.93± 1.08	0.54± 0.67	1.14± 0.87
VLDL-C (mmol/L)	1.18± 0.31	1.09± 0.22	1.30± 0.33	1.11± 0.21
Triglyceride (mmol/L)	2.55± 0.69	2.43± 0.43	2.86± 0.72	2.45± 0.45
TC (mmol/L)	4.74± 0.82	4.58± 0.78	4.42± 1.20	4.30± 0.38
TC/HDL-C	1.93± 0.79	1.96± 0.71	1.71± 0.29	2.37± 0.94
<b>Males</b>				
	(n=144)	(n=165)	(n=66)	(n=4)
HDL-C (mmol/L)	2.61± 0.75	2.61± 0.77	2.59± 0.76	2.64± 0.76
LDL-C (mmol/L)	0.79± 1.01	0.83± 1.10	0.92± 1.15	1.01± 0.74
VLDL-C (mmol/L)	1.09± 0.24	1.09± 0.34	1.14± 0.30	1.18± 0.03
Triglyceride (mmol/L)	2.47± 0.56	2.46± 0.67	2.57± 0.59	2.59± 0.07
TC (mmol/L)	4.45± 0.73	4.53± 0.80	4.64± 0.84	4.83± 0.87
TC/HDL-C	1.81± 0.63	1.90± 0.70	2.03± 1.10	1.92± 0.47

HDL-C - High Density Lipoprotein Cholesterol, LDL-C -Low Density Lipoprotein Cholesterol, VLDL-C - Very Low Density Lipoprotein Cholesterol, n-sample size, S.D. - Standard Deviation

**Table 4.16:** Comparison of mean somatotype components within BMI classification according to sex

BMI classification (kg/m <sup>2</sup> )	n	%	Endomorphy Mean ± S.D.	Mesomorphy Mean ± S.D.	Ectomorphy Mean± S.D.	Somatotype categories
<b>Females</b>						
Underweight (<18.5)	13	7.18	2.25 ± 0.26	1.46 ± 1.14	4.59 ± 0.47	Balanced ectomorph
Normal (18.5 - 24.9)	161	88.95	2.51 ± 0.28	3.12 ± 1.30	2.45 ± 0.91	Central
Overweight (30 &above)	7	3.87	2.65 ± 0.28	4.84 ± 1.34	0.81 ± 0.43	Endomorphic mesomorph
	n (181)	p-value	0.0023	<0.001	<0.001	
<b>Males</b>						
Underweight (<18.5)	14	3.69	2.30 ± 0.29	1.99 ± 0.66	5.09 ± 0.48	Balanced ectomorph
Normal (18.5 - 24.9)	343	90.50	2.29 ± 0.34	3.21 ± 1.00	2.68 ± 0.90	Balanced mesomorph
Overweight (30 &above)	22	5.80	2.49 ± 0.28	4.71 ± 1.06	0.85 ± 0.55	Endomorphic mesomorph
	n (379)	p-value	0.0259	<0.001	<0.001	

S.D. = Standard Deviation, n=number of sample size; BMI=Body Mass Index

#### **4.7 Somatotype Category**

The somatotype category for all subjects were presented in Table 4.17. The majority of the females were centrally placed, while the males were categorized as balanced mesomorph. The overall subjects tend towards mesomorphic somatotype.

The frequency and distribution of 13 categories of somatotype (Carter, 1980) are shown in Table 4.17. The studied participants belonged to four somatotype categories: balanced mesomorph (26.43%), central type (22.32%), balanced ectomorph (17.14%) and endomorphic-mesomorph (13.93%), while other participants belonged to other categories of somatotype (Table 4.17). Males participants belonged predominantly to balanced mesomorph (30.08%), central (20.32%) and balanced ectomorph (19%), while females belonged to central (26.52%), balanced mesomorph (18.78%) and endomorphic mesomorph (17.68%).

**Table 4.17: Frequency table of somatotype category for the studied group**

S/NO	Somatotype category	Female (n=181)	Male (n=379)	Total (560)
1.	Balanced ectomorph	24 (13.26%)	72 (19%)	96 (17.14%)
2.	Balanced mesomorph	34 (18.78%)	114 (30.08%)	148 (26.43%)
3.	Central	48 (26.52%)	77 (20.32%)	125 (22.32%)
4.	Ectomorphic endomorph	1 (0.55%)	1 (0.26%)	2 (0.36%)
5.	Ectomorphic mesomorph	4 (2.21%)	13 (3.43%)	17 (3.04%)
6.	Endomorph-ectomorph	7 (3.87%)	3 (0.79%)	10 (1.79%)
7.	Endomorphic ectomorph	12 (6.63%)	13 (3.43%)	25 (4.46%)
8.	Endomorphic mesomorph	32 (17.68%)	46 (12.14%)	78 (13.93%)
9.	Mesomorph-ectomorph	6 (3.31%)	20 (5.28%)	26 (4.64%)
10.	Mesomorph-endomorph	11 (6.08%)	5 (1.32%)	16 (2.86%)
11.	Mesomorphic ectomorph	2 (1.10%)	14 (3.69%)	16 (2.86%)
12.	Mesomorphic endomorph	0 (0%)	1 (0.26%)	1 (0.18%)
13.	Balanced endomorph	0 (0%)	0 (0%)	0 (0%)
Total		181(100%)	379 (100%)	560 (100%)

n- sample size

#### **4.8 Body Mass Index Ranges**

The frequency distribution of 560 individuals in all BMI ranges are shown in Table 4.18 and there was no female found with BMI above 30 but only one male in this category displayed endomorphic mesomorph somatotype with 2.67 - 4.39 - 0.1.

**Table 4.18: Frequency distribution of females and males in all BMI ranges**

BMI Classification (kg/m <sup>2</sup> )	Category	Female Frequency (n=181)	Male Frequency (n=379)	Total
< 18.5	Underweight	13	14	27
18.5 - 24.9	Normal	161	344	505
25 - 29.9	Overweight	7	20	27
30 & above	Obese	Nil	1	1
	Total	181	379	560

BMI - Body Mass Index, n- sample size



## 4.9 Correlation Analyses

Pearson's correlation coefficient was calculated to determine the relationship between blood pressure, lipid profile and somatotypes in the total studied participants.

In Table 4.19 (correlation analysis of female variables) positive significant correlations were observed between the SBP/DBP and somatotypes (endomorphs and mesomorphs) ( $p < 0.001$ ) and negative correlation with ectomorphs ( $p < 0.05$ ). Significant negative correlations were seen between SBP and HDL-C ( $p < 0.05$ ). HDL-C showed negative correlation between endomorphs and mesomorphs but positive with ectomorphs in females (Table 4.19). LDL-C showed positive correlation between endomorphs and mesomorphs and significant negative correlation with ectomorphs ( $p < 0.05$ ) among females (Table 4.19). TG correlated negatively with endomorphs and ectomorphs and TC showed positive correlation with endomorphs and mesomorphs (Table 4.19).

Table 4.20 (correlation analysis of male variables) positive significant correlations were observed in SBP and somatotypes (endomorphs and mesomorphs) ( $p < 0.001$ ) and negative correlation with ectomorphs in males. The DBP was positively correlated with mesomorphs ( $p < 0.001$ ) and negatively correlated with ectomorphs ( $p < 0.05$ ). Positive correlations were seen between SBP/DBP and HDL-C in males. HDL-C showed positive correlation between endomorphs and ectomorphs but negative with mesomorphs in males (Table 4.20). LDL-C showed negative correlation between endomorphs and ectomorphs; and negative correlation with mesomorphs among males (Table 4.20). TG and TC correlated negatively with endomorphs and mesomorphs and showed positive correlation with ectomorphs (Table 4.20).

Table 4.21 showed correlation analysis between some variables and lipid profile according to sex. In females, BMI was significantly correlated with TG and VLDL-C ( $p < 0.05$ ) and

negatively with AI. WC significantly correlated negatively with LDL-C and HDL-C ( $p < 0.05$ ) and positive with AI ( $p < 0.01$ ). SBP was negatively correlated with LDL-C and HDL-C ( $p < 0.05$ ) and positive with AI significantly ( $p < 0.001$ ) (Table 4.21).

In males, DBP showed significant negative correlation with TG ( $p < 0.01$ ).

Table 4.22 presented correlates of anthropometric variables and blood pressure according to sex. The result of SBP in females showed significant correlation except abdominal and suprailiac skinfold. DBP in females was significantly correlated except supraspinale, bicep and suprailiac skinfold thickness. In males, the result showed significant correlation except subscapular, suprailiac and thigh skinfold thickness. DBP in males was significantly correlated except supraspinale, medial calf, suprailiac and thigh skinfold thickness. All the conventional measures of adiposity (age, height, weight, BMI, waist circumference, hip circumference) significantly correlated positively with SBP and DBP in both sexes (Table 4.22). In addition, suprailiac skinfold thickness not correlated with SBP and DBP in both sexes. (Table 4.22).

The mean DBP was associated with increase in weight, BMI and waist and hip circumferences in the studied population and positively correlated significantly (Table 4.22). Skinfold measurements showed weak correlations with mean DBP.

**Table 4:19: Correlation matrix of female variables (n = 181)**

	SBP(mmHg)	DBP(mmHg)	HDL-C	LDL-C	VLDL-C	TG	TC	TC/HDL	ENDO	MESO	ECTO
SBP(mmHg)	–										
DBP(mmHg)	0.702 <sup>a</sup>	–									
HDL-C	-0.169 <sup>c</sup>	-0.084	–								
LDL-C	0.184 <sup>c</sup>	0.124	-0.632 <sup>a</sup>	–							
VLDL-C	0.088	0.119	0.017	-0.262 <sup>a</sup>	–						
TG	0.058	0.118	0.027	-0.253 <sup>a</sup>	0.959 <sup>a</sup>	–					
TC	0.140	0.139	-0.014	0.741 <sup>a</sup>	0.005	0.011	–				
TC/HDL-C	0.238 <sup>a</sup>	0.145	-0.787 <sup>a</sup>	0.858 <sup>a</sup>	0.027	0.020	0.516 <sup>a</sup>	–			
ENDO	0.217 <sup>a</sup>	0.270 <sup>a</sup>	-0.063	0.073	-0.053	-0.061	0.028	0.064	–		
MESO	0.427 <sup>a</sup>	0.395 <sup>a</sup>	-0.082	0.075	0.148	0.112	0.084	0.124	0.376 <sup>a</sup>	–	
ECTO	-0.149 <sup>c</sup>	-0.145	0.051	-0.020 <sup>c</sup>	-0.120	-0.107	-0.025	-0.066	-0.296 <sup>a</sup>	-0.434 <sup>a</sup>	–

a = p<0.001, b = p<0.01, c = p<0.05

SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure, HDL-C - High Density Lipoprotein Cholesterol (mmol/L), LDL-C -Low Density Lipoprotein Cholesterol (mmol/L), VLDL-C - Very Low Density Lipoprotein Cholesterol (mmol/L), TG-Triglyceride (mmol/L), TC-Total cholesterol (mmol/L), AI-Atherogenic Index (TC/HDL-C), ENDO - Endomorphy, MESO - Mesomorphy, ECTO - Ectomorphy.

**Table 4:20: Correlation matrix of male variables (n = 379)**

	SBP	DBP	HDL-C	LDL-C	VLDL-C	TG	TC	TC/HDL	ENDO	MESO	ECTO
SBP	–										
DBP	0.610 <sup>a</sup>	–									
HDL-C	0.053	0.023	–								
LDL-C	-0.018	-0.001	-0.648 <sup>a</sup>	–							
VLDL-C	-0.045	-0.084	0.006	-0.218 <sup>a</sup>	–						
TG	-0.079	-0.136 <sup>c</sup>	0.069	-0.241 <sup>a</sup>	0.889 <sup>a</sup>	–					
TC	0.010	-0.012	0.101	0.636 <sup>a</sup>	0.103	0.089	–				
TC/HDL-C	-0.026	-0.019	-0.775 <sup>a</sup>	0.840 <sup>a</sup>	0.012	-0.028	0.385 <sup>a</sup>	–			
ENDO	0.148 <sup>a</sup>	0.068	0.092	-0.087	-0.026	-0.017	-0.039	-0.092	–		
MESO	0.168 <sup>a</sup>	0.239 <sup>a</sup>	-0.018	0.000	-0.106 <sup>c</sup>	-0.105 <sup>c</sup>	-0.058	-0.029	0.314 <sup>a</sup>	–	
ECTO	-0.014	-0.108 <sup>c</sup>	0.046	-0.025	0.072	0.080	0.039	-0.011	-0.239 <sup>a</sup>	0.573 <sup>a</sup>	–

a = p<0.001, b = p<0.01, c = p<0.05

SBP-Systolic Blood Pressure (mmHg), DBP-Diastolic Blood Pressure (mmHg), HDL-C - High Density Lipoprotein Cholesterol (mmol/L), LDL-C -Low Density Lipoprotein Cholesterol (mmol/L), VLDL-C - Very Low Density Lipoprotein Cholesterol (mmol/L), TG-Triglyceride (mmol/L), TC-Total cholesterol, AI-Atherogenic Index (TC/HDL-C), ENDO - Endomorphy, MESO - Mesomorphy, ECTO - Ectomorphy.

**Table 4.21: Correlation coefficient analysis between variables and lipid profile according to sex**

	TC (mmol/L)	LDL-C (mmol/L)	HDL-C (mmol/L)	TG (mmol/L)	VLDL-C (mmol/L)	AI
<b>Females</b>						
BMI(kg/m <sup>2</sup> )	0.020	-0.021	-0.017	0.176*	0.181*	-0.041
WC (cm)	0.105	-0.166*	-0.161*	0.037	0.039	0.223**
WHR	0.053	0.107	-0.105	-0.008	-0.014	0.122
SBP(mmHg)	0.140	-0.184*	-0.169*	0.058	0.088	0.238***
DBP(mmHg)	0.139	0.124	-0.084	0.118	0.119	0.145
<b>Males</b>						
BMI(kg/m <sup>2</sup> )	-0.049	-0.023	-0.005	-0.039	-0.030	-0.034
WC (cm)	-0.043	-0.034	0.012	-0.008	-0.021	-0.011
WHR	0.013	0.055	-0.062	0.015	-0.001	0.060
SBP(mmHg)	0.010	-0.018	0.053	-0.079	-0.045	-0.026
DBP(mmHg)	-0.012	-0.001	0.023	-0.136**	-0.084	-0.019

\*\*\*-  $p < 0.001$ , \*\*-  $p < 0.01$ , \*-  $p < 0.05$ . BMI - Body Mass Index, WC -Waist circumference, WHR - Waist Hip Ratio, SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure, HDL-C - High Density Lipoprotein Cholesterol, LDL-C -Low Density Lipoprotein Cholesterol, VLDL-C - Very Low Density Lipoprotein Cholesterol, TG-Triglyceride, TC- Total cholesterol, AI-Atherogenic Index.

**Table 4.22: Correlates of anthropometric variables and blood pressure according to sex**

Variables	Systolic Blood Pressure (mmHg)	Blood Pressure	Diastolic Blood Pressure (mmHg)	Blood Pressure
	Females	Males	Females	Males
	Pearson's correlation	Pearson's correlation	Pearson's correlation	Pearson's correlation
Age (years)	0.269***	0.057 <sup>NS</sup>	0.231**	0.049 <sup>NS</sup>
Weight (kg)	0.443***	0.332***	0.507***	0.307***
Height (cm)	0.575***	0.300***	0.385***	0.188***
Body Mass Index (kg/m <sup>2</sup> )	0.365***	0.155**	0.323***	0.213***
Waist circumference (cm)	0.452***	0.244***	0.336***	0.152**
Hip circumference (cm)	0.388**	0.243***	0.317***	0.193***
Triceps skinfold (mm)	0.315***	0.226***	0.331***	0.210***
Subscapular skinfold (mm)	0.338***	0.019 <sup>NS</sup>	0.411***	-0.007
Supraspinale skinfold (mm)	0.161*	0.191***	0.125 <sup>NS</sup>	0.012 <sup>NS</sup>
Medial calf skinfold (mm)	0.225**	0.133**	0.220**	0.032 <sup>NS</sup>
Bicep skinfold (mm)	0.191*	0.102*	0.129 <sup>NS</sup>	0.154**
Abdominal skinfold (mm)	-0.102 <sup>NS</sup>	0.210***	-0.181*	0.071
Suprailiac skinfold (mm)	0.052 <sup>NS</sup>	0.013 <sup>NS</sup>	0.059 <sup>NS</sup>	0.000 <sup>NS</sup>
Thigh skinfold (mm)	0.221**	0.069 <sup>NS</sup>	0.153*	0.078 <sup>NS</sup>

a- p<0.001, b- p<0.01, c- p<0.05, NS - Non significance

#### **4.10 Socioeconomic status of all participants as determined by Parents' education level**

The socioeconomic status of the students defined by their parents' education level was shown in Table 4.23. Approximately 7% of the mothers had received no formal education and about 48% had received a tertiary education. The majority of the fathers had obtained a tertiary education (approximately 63%).

#### **4.11 Impact of Socioeconomic Factors on Somatotype Components**

Blood pressure and somatotype components in relation to level of education and employment status of parents and socioeconomic status in females and males were presented in Tables 4.24 – 4.27.

Table 4.24 presented the blood pressure and somatotype components related to fathers' educational level. There were significant difference in SBP in females as related to father's educational level ( $p < 0.05$ ). This significant was observed in fathers' that had a tertiary education to those with primary and none education according to post hoc pair wise comparison analysis. There were significant differences of endomorph component in both females and males in relation to all socioeconomic factors. Endomorph component had higher values ( $2.74 \pm 0.37$ ) in females whose father had primary education, while ( $2.35 \pm 0.34$ ) in males whose father had completed higher education (Table 4.24). There were significant differences for mesomorph component in females and males related to parental educational level ( $p < 0.05$  and  $p < 0.001$  respectively). Mesomorph component was dominant in females when the students' mother had completed primary school and in males when their fathers had an higher degree (Table 4.24). Ectomorph component had higher values (Table 4.24) in female students when their father had none education.

completed primary school and in those whose father were unemployed ( $2.97 \pm 1.23$  and  $2.73 \pm 0.97$  respectively), contrary to those with employed parents ( $2.35 \pm 1.10$  and  $2.66 \pm 1.11$  respectively) (Table 4.21).

Table 4.25 presented fathers' employment status in relation to somatotype and blood pressure. Endomorph component had higher values for the employed fathers in both sexes. In relation to socioeconomic status (parentals' employment status) significant difference were found for endomorphy component. Ectomorph component had higher values (Table 4.25) in female and male students whose fathers are artisan/farmers ( $2.97 \pm 1.23$  and  $2.73 \pm 0.97$  respectively), contrary to those with employed parents ( $2.35 \pm 1.10$  and  $2.66 \pm 1.11$  respectively) (Table 4.25).

Table 4.26 showed blood pressure and somatotype components related to mothers' educational level in all participants while Table 4.27 presented the blood pressure and somatotype components related to mothers' employment status. SBP, endomorphy and mesomorphy showed significant across the group.



**Table 4.23: Socioeconomic status of Nigerian undergraduate students by parentals' level of education**

Level of Education	Father's level of education		Mother's level of education			
	Females n = 181 (%)	Males n = 379 (%)	Females n = 181 (%)	Males n = 379 (%)	Father n = 560 (%)	Mother n = 560 (%)
Primary	8 (4.42)	40 (10.55)	15 (8.29)	68 (17.94)	48 (8.57)	83 (14.82)
Secondary	43 (23.76)	94 (24.80)	46 (25.41)	123 (32.45)	137 (24.46)	169 (30.18)
Tertiary	126 (69.61)	229 (60.42)	104 (57.46)	165 (43.54)	355 (63.39)	269 (48.03)
None	4 (2.21)	16 (4.22)	16 (8.84)	23 (6.07)	20 (3.57)	39 (6.96)

n - sample size

**Table 4.24: Blood pressure and somatotype components related to fathers' educational level in all participants.**

	None n (F=4, M=16) Mean ± S.D	Primary n (F=8, M=40) Mean ± S.D	Secondary n (F=43, M=94) Mean ± S.D	Tertiary n (F=126, M=229) Mean ± S.D	P value	Post hoc pair wise comparison
Age (F) (year)	25.5 ± 3.11	23.25±2.87	21.79 ± 2.91	21.10 ± 2.31	P<0.01	
Age (M) (year)	23.75±3.17	23.0 ± 3.40	22.95 ± 3.31	22.39 ± 2.81	NS	
SBP (F) (mmHg)	125 ± 10	121 ± 7.19	118.44±8.31	114.89±9.91	P<0.05	F1&F4, F2&F4
SBP (M) (mmHg)	115.63±9.31	118.08±9.96	117.65±8.39	117.65±8.87	NS	
DBP (F) (mmHg)	76.25±7.50	82.75±7.40	77.88 ± 8.90	78.47± 7.66	NS	
DBP (M) (mmHg)	78.81± 6.63	79.53± 7.15	78.95± 6.69	79.71± 6.55	NS	
ENDO (F)	2.35 ±0.21	2.74 ±0.37	2.46 ±0.29	2.50 ±0.27	NS	**F2&M1,**F4&M1, *F2&M2,*F4&M2, **M3&M4,***F2&M 3, ***F3&M3, ***F4&M3, *F2&M4, ***F4&M4
ENDO (M)	2.21 ±0.35	2.32 ±0.28	2.20 ±0.32	2.35 ±0.34	P<0.01	
MESO (F)	3.17 ±0.09	4.44 ±1.42	2.80 ±1.27	3.07 ±1.42	P<0.05	*M(3&4), *F1&M3,
MESO (M)	3.01 ±0.72	3.20 ±1.16	2.91 ±1.02	3.41 ±1.09	P<0.001	**F(2&3), *F(2&4)
ECTO (F)	2.96 ± 0.82	2.23 ± 0.45	2.64 ±1.03	2.51 ±1.14	NS	
ECTO (M)	2.87 ± 1.03	2.48 ± 0.97	2.91 ± 1.11	2.57 ± 1.07	NS	

S.D. - Stan. Dev., ENDO- Endomorphy, MESO - Mesomorphy, ECTO - Ectomorphy, SBP - Systolic Blood Pressure, DBP - Diastolic Blood Pressure, n- sample size, (F) - Females, (M) - Males, 1-None; 2-Primary; 3-Secondary; 4-Tertiary. \*\*p<0.01, \*p<0.05, NS- Non significance, F1 - Female None education's father, F2 - Female Primary education's father, F3 - Female Secondary education's father, F4 - Female Tertiary education's father, M1 - Male None education's father, M2 - Male Primary education's father, M3 - Male Secondary education's father, M4 - Male Tertiary education's father.

**Table 4.25: Blood pressure and somatotype components related to fathers' employment status in all participants.**

	Civic servant n (F=114, M=215)	Trader n (F=39, M=81)	Artisan n (F=28, M=83)		
	Mean ± S.D	Mean ± S.D	Mean ± S.D	P value	Post hoc pair wise comparison
Age (F) (year)	21.18 ± 2.52	21.05 ± 1.67	23.18 ± 3.33	P<0.001	*F1&F3, ***F1&M1, ***F1&M3, *F2&F3, *F2&M1, **F2&M3
Age (M) (year)	22.63±2.78	22.21 ± 2.98	23.13 ± 3.62	NS	
SBP (F) (mmHg)	115.82±9.52	114.97±7.88	119.6 ± 9.93		
SBP (M) (mmHg)	117.60±8.45	116.81±9.31	118.46±9.50	NS	
DBP (F) (mmHg)	78.33±7.69	78.54 ± 6.76	78.93± 10.48	NS	
DBP (M) (mmHg)	79.41± 6.64	79.86± 6.90	79.08± 6.47	NS	
ENDO (F)	2.51 ±0.28	2.51 ±0.21	2.44 ±0.38	NS	***F1&M1, ***F1&M2,
ENDO (M)	2.32 ±0.33	2.26 ±0.35	2.27 ±0.34	P<0.01	***F1&M3, *F2&M1, **F2&M2, **F2&F3
MESO (F)	3.12 ±1.39	2.73 ±1.14	3.34 ±1.71	NS	
MESO (M)	3.35 ±1.11	3.22 ±1.09	3.03 ±0.97	NS	
ECTO (F)	2.35 ± 1.10	2.78 ±0.82	2.97 ±1.23	P<0.05	
ECTO (M)	2.66 ±1.11	2.59 ±1.10	2.73 ±0.97	NS	

ENDO- Endomorphy, MESO - Mesomorphy, ECTO - Ectomorphy, SBP - Systolic Blood Pressure, DBP - Diastolic Blood Pressure, n- sample size, (F) - Females, (M) - Males, 1-Civic servant; 2-Trader/Businessman; 3- Artisans/farmers. \*\*p<0.01, \*p<0.05, NS- Non significance, F1 - Female Civic servant's father, F2 - Female Trader/Businessman's father, F3 - Female Artisans/farmers's father, M1 - Male Civic servant's father, M2 - Male Trader/Businessman's father, M3 - Male Artisans/farmers's father, S.D. - Standard Deviation.

**Table 4.26: Blood pressure and Somatotype components related to mothers' educational level in all participants.**

	None n (F=16, M=23) Mean ± S.D	Primary n (F=15, M=68) Mean ± S.D	Secondary n (F=46, M=123) Mean ± S.D	Tertiary n (F=104, M=165) Mean ± S.D	P value	Post hoc pair wise comparison
Age (F) (year)	23.94 ± 3.07	21.87±3.18	21.63 ± 2.79	20.94 ± 2.11	P<0.001	**F1&F3, ***F1&F4
Age (M) (year)	23.43±3.19	22.81 ± 2.95	22.72 ± 2.94	22.42 ± 3.10	NS	
SBP (F) (mmHg)	122.06±10.51	112.80 ± 10.84	117.13±9.02	115.42±9.33	P<0.05	*F(1&2)
SBP (M) (mmHg)	112.65±7.94	115.66±9.04	118.20±8.79	118.70±8.69	P<0.05	*M(1&3), *M(1&4),
DBP (F) (mmHg)	78.06±11.73	77.53±6.39	78.35 ± 7.48	78.72± 7.77	NS	
DBP (M) (mmHg)	77.70± 6.63	79.13± 6.16	78.63± 6.80	80.41± 6.64	NS	
ENDO (F)	2.50 ±0.26	2.42 ±0.39	2.54 ±0.32	2.49 ±0.25	NS	**F3&M1, ***F3&M2, ***F3&M3, *F3&M4, **F4&M1, ***F4&M2, ***F4&M3, **F4&M4
ENDO (M)	2.21 ±0.38	2.26 ±0.35	2.27 ±0.37	2.35 ±0.29	NS	
MESO (F)	3.74 ±1.57	2.17 ±1.24	3.01 ±1.21	3.12 ±1.43	P<0.05	**F(1&2), **F2&M1,
MESO (M)	3.52 ±0.74	3.15 ±1.09	3.07 ±1.02	3.39 ±1.15	NS	***F2&M4.
ECTO (F)	2.54 ± 1.58	2.66 ± 1.13	2.66 ±0.95	2.47 ±1.06	NS	
ECTO (M)	2.75 ±0.73	2.59 ±1.01	2.80 ±1.13	2.57 ±1.09	NS	

ENDO- Endomorphy, MESO - Mesomorphy, ECTO - Ectomorphy, SBP - Systolic Blood Pressure, DBP - Diastolic Blood Pressure, n- sample size, (F) - Females, (M) - Males, 1-None; 2-Primary; 3-Secondary; 4-Tertiary. \*\*p<0.01, \*p<0.05, NS- Non significance, F1 - Female None education's father, F2 - Female Primary education's father, F3 - Female Secondary education's father, F4 - Female Tertiary education's father, M1 - Male None education's father, M2 - Male Primary education's father, M3 - Male Secondary education's father, M4 - Male Tertiary education's father.

**Table 4.27: Blood pressure and Somatotype components related to mothers' employment status in all participants.**

	Trader n (F=32, M=95)	Housewife n (F=60, M=107)	Civic servant n (F=81, M=149)	Artisan n (F=8, M=28)	P value	Post hoc pair wise comparison	
	Mean ± S.D	Mean ± S.D	Mean ± S.D	Mean ± S.D			
Age (F) (year)	21.34 ± 2.51	22.30±2.91	20.98 ± 2.23	20.50 ± 2.83	P<0.05	*F2&F3 **F3&M1, ***F3&M3	*F3&M2,
Age (M) (year)	22.73±2.94	22.43 ± 2.92	22.71 ± 3.07	22.89 ± 3.56	NS		
SBP (F) (mmHg)	113.97±9.52	116.60±9.80	116.47±9.87	120.0 ± 5.35	P<0.01	M(2&3), M(2&4)	
(M)	117.11±8.93	115.91±7.50	118.65±9.04	120.46±11.27			
DBP (F) (mmHg)	77.22±7.30	78.45±8.14	79.14 ± 8.18	76.88± 7.47	NS		
DBP (M) (mmHg)	78.79± 7.17	78.59± 6.03	80.15± 6.65	81.07± 6.66	NS		
ENDO (F)	2.44 ±0.22	2.51 ±0.34	2.50 ±0.25	2.57 ±0.44	NS	***F1&M2,	**F1&M4,
ENDO (M)	2.40 ±0.35	2.17 ±0.34	2.36 ±0.27	2.12 ±0.38	P<0.001	***F2&M2, ***F2&M4, *F3&M3, *F4&M2, ***M1&M2, ***M2&M3,	*F2&M3, ***F3&M2, ***F3&M4, **F4&M4, ***M1&M4, **M3&M4
MESO (F)	2.74 ±1.03	2.95 ±1.42	3.33 ±1.47	2.58 ±1.53	NS		
MESO (M)	3.38 ±1.00	3.07 ±1.11	3.36 ±1.16	2.87 ±0.48	NS		
ECTO (F)	2.71 ± 0.95	2.78 ± 1.14	2.30 ±1.09	2.48 ±0.86	NS	***F3&M2,	***M1&M2,
ECTO (M)	2.39 ±1.04	3.03 ±1.12	2.51 ±1.05	2.96 ±0.68	NS	***M2&M3	

ENDO- Endomorphy, MESO - Mesomorphy, ECTO - Ectomorphy, SBP - Systolic Blood Pressure, DBP - Diastolic Blood Pressure, n- sample size, (F) - Females, (M) - Males, 1-Civic servant; 2-Trader/Businessman; 3- Artisans/farmers. \*\*p<0.01, \*p<0.05, NS- Non significance, F1 - Female Civic servant's father, F2 - Female Trader/Businessman's father, F3 - Female Artisans/farmers's father, M1 - Male Civic servant's father, M2 - Male Trader/Businessman's father, M3 - Male Artisans/farmers's father, S.D. - Standard Deviation.

#### **4.12 Relationship between Body build, Somatotype components and Lipid profiles**

The Spearman correlation coefficients (Table 4.28) showed overall cholesterol in serum correlated negatively to body weight, BMI, WC and WHR, while positively to height and ectomorphy. The same relationship exists between the level of LDL-C and the anthropometrical features (except height) and somatotype, that is, a negative correlation of LDL-C to body height and a positive to WHR and mesomorphy. All HDL-C correlation coefficients to body build features were positive. Analysis of the association between content of TG and components of body build showed the most significant correlations. The body height, WHR and the ectomorphy factor showed positive relationship, while weight, BMI, WC, endomorphy and mesomorphy showed negative relationship to all the other parameters.

Blood pressure significantly correlated positively with all other variables except WHR and ectomorphy component which were negatively correlated. AI significantly correlated negatively with endomorphy.

**Table 4.28: Correlation analysis between the content of cholesterol, its fractions and triglycerides in blood serum and selected anthropometric features and body build components of all participants (n=560)**

	TC (mmol/L)	TG (mmol/L)	HDL-C (mmol/L)	LDL-C (mmol/L)	SBP (mmHg)	DBP (mmHg)	AI
Weight (kg)	-0.024	-0.044	0.062	-0.033	0.290**	0.287**	0.047
Height (cm)	0.033	0.015	0.099	-0.044	0.261**	0.185**	-0.066
BMI(kg/m <sup>2</sup> )	-0.062	-0.062	-0.007	-0.016	0.141**	0.195**	0.011
WC (cm)	-0.052	-0.043	0.022	-0.056	0.264**	0.213**	-0.050
WHR	-0.023	0.004	-0.078	0.049	-0.003	-0.079	0.065
ENDO	-0.023	-0.038	0.114*	-0.085	0.121*	0.053	- 0.107*
MESO	-0.037	-0.098	-0.029	0.022	0.167**	0.211**	0.006
ECTO	0.038	0.076	0.051	-0.021	-0.021	-0.084	-0.034

n- sample size, \*\* p < 0.01, \* p < 0.05, BMI - Body Mass Index, WC - Waist Circumference, WHR - Waist to Hip ratio, ENDO - Endomorphy, MESO - Mesomorphy, ECTO - Ectomorphy, TC - Total cholesterol, TG - Triglycerides, HDL-C - High density lipoprotein cholesterol, LDL-C - Low density lipoprotein cholesterol, SBP - Systolic blood pressure, DBP - Diastolic blood pressure, AI - Atherogenic index

#### **4.13 Relationship of WHR and BMI with Blood Pressure of Individual Participants**

Table 4.29 showed the demographic characteristics and the indices of adiposity of the studied participants. From the table, it can be observed that male participants were significantly taller and heavier ( $p < 0.001$ ), while no significant difference existed in age between the male and female participants ( $p > 0.05$ ). The mean values of WHR for all participants (0.92 for males and 0.90 for females) fell below the range for the overweight and obese. The mean BMI for all the participants fell within the healthy weight range (approx.  $22 \text{ kg/m}^2$ ) (Table 4.29).

#### **4.14 Relationship Between Indices of Adiposity and Cardiovascular Variables of Participants**

There were significant correlations between the systolic blood pressure as well as the diastolic blood pressure and the indices of adiposity for all the participants ( $p < 0.05$ ) (Table 4.30).



**Table 4.29: Demographic characteristics and indices of adiposity in participants**

Variables	Female (n=181) Mean $\pm$ S.D.	Male (n=379) Mean $\pm$ S.D.	t-value	p-value
Age (year)	21.46 $\pm$ 2.60	22.65 $\pm$ 3.03	2.697	0.143
Weight (kg)	58.64 $\pm$ 7.43	62.11 $\pm$ 6.83	7.878	<0.001
Height (cm)	165.02 $\pm$ 6.95	168.98 $\pm$ 6.44	8.969	<0.001
WC (cm)	72.74 $\pm$ 6.89	72.39 $\pm$ 5.78	0.7805	0.442
BMI (kg/m <sup>2</sup> )	21.50 $\pm$ 2.03	21.74 $\pm$ 1.93	0.5557	0.686
WHR	0.90 $\pm$ 0.06	0.92 $\pm$ 0.04	0.04335	0.193
WHtR	0.44 $\pm$ 0.04	0.43 $\pm$ 0.03	0.0323	0.134

n- sample size, S.D. - Standard Deviation, WC - Waist circumference, BMI - Body mass index, WHR - Waist Hip Ratio, WHtR - Waist to Height Ratio, ns - non significant

**Table 4.30: Correlation between indices of adiposity and cardiovascular variables for all participants**

	WC (cm)	WHR	BMI (kg/m <sup>2</sup> )	WHtR
<b>Females</b>				
SBP (mmHg)	0.452**	0.115	0.365**	0.265**
DBP (mmHg)	0.336**	0.056	0.323**	0.172*
<b>Males</b>				
SBP (mmHg)	0.244**	0.196	0.155**	0.101*
DBP (mmHg)	0.152**	-0.084	0.213**	0.065

\*\* correlation is significant at the 0.01 level (2 tailed)

\* correlation is significant at the 0.05 level (2 tailed)

SBP - Systolic blood pressure, DBP - Diastolic blood pressure, WC - Waist circumference, BMI - Body mass index, WHR - Waist Hip Ratio, WHtR - Waist to Height Ratio

#### **4.15 Lifestyle Characteristics of Individual Participants**

Alcohol consumption and smoking were higher among males than females. The incidence of hypertension was higher in males than females as shown in Table 4.31. Consumption of alcohol among male participants were higher (18.78%) than females (0.55%).

**Table 4.31: Lifestyle characteristics of Nigerian students**

	<b>Females</b>	<b>Males</b>
<b>Smoking</b>		
Present	1 (0.55%)	6 (1.58%)
Before	1 (0.55%)	15 (3.96%)
Never	179 (98.9%)	358 (94.46%)
<b>Alcohol consumption</b>		
Yes	1 (0.55%)	34 (8.97%)
No	180 (99.45%)	345 (91.03%)
<b>Do you have Diabetes mellitus?</b>		
Yes	0 (0.55%)	0 (0%)
No	159 (87.84%)	322 (84.96%)
Don't know	21 (11.60%)	57 (15.04%)
<b>Are you on any Diabetic drug?</b>		
Yes	0 (0%)	0 (0%)
No	181 (100%)	379 (100%)
<b>Do you have hypertension?</b>		
Yes	0 (0%)	0 (0%)
No	168 (92.82%)	335 (89.45%)
Don't know	13 (7.18%)	40 (10.55%)
<b>Are you on any hypertensive drug?</b>		
Yes	0 (0%)	0 (0%)
No	181 (100%)	379 (100%)

#### **4.16 Comparison of Anthropometric Indices in Predicting the Risk of Hypertension in Individual studied participants.**

Some anthropometric indices were considered in Table 4.32 and correlated in Tables 4.33 and 4.34. Using Pearson correlation, there were positive correlations between the BMI and WC and WHtR. Most of the anthropometric indices were highly correlated.

**Table 4.32: Anthropometric indices according to sex**

Variable	Females (n=181)			Males (n=379)			p-value
	Mean $\pm$ S.D	Min.	Max.	Mean $\pm$ S.D.	Min.	Max.	
Age (year)	21.5 $\pm$ 2.60	17.00	30.00	22.6 $\pm$ 3.03	17.00	33.00	0.234
BMI (kg/m <sup>2</sup> )	21.50 $\pm$ 2.03	17.12	27.58	21.74 $\pm$ 1.93	16.73	31.38	0.174
WC (cm)	72.73 $\pm$ 6.89	52.10	90.00	72.39 $\pm$ 5.78	28.10	92.00	0.561
WHR	0.90 $\pm$ 0.06	0.62	1.16	0.92 $\pm$ 0.04	0.76	1.04	0.345
WHtR	0.44 $\pm$ 0.04	0.34	0.56	0.43 $\pm$ 0.03	0.17	0.54	0.333

n- sample size, S.D. - Standard Deviation, BMI - Body mass index, WC - Waist circumference, WHR - Waist to Hip ratio, WHtR - Waist to Height ratio.

**Table 4.33: Correlation between anthropometric indices for all participants**

	<b>BMI (kg/m<sup>2</sup>)</b>	<b>WC (cm)</b>	<b>WHR</b>	<b>WHtR</b>
<b>Females</b>				
BMI(kg/m <sup>2</sup> )	–	0.456**	0.099	0.477**
WC(cm)		–	0.543**	0.896**
WHR			–	0.485**
WHtR				–
<b>Males</b>				
BMI(kg/m <sup>2</sup> )	–	0.399**	- 0.074	0.465**
WC(cm)		–	0.316**	0.881**
WHR			–	0.299**
WHtR				–

\*\* correlation is significant at the 0.01 level (2 tailed)

\* correlation is significant at the 0.05 level (2 tailed)

BMI - Body mass index, WC - Waist circumference, WHR - Waist to Hip ratio, WHtR - Waist to Height ratio.

**Table 4.34: Correlation between Anthropometric indices and blood pressure for all participants**

	SBP (mmHg)		DBP (mmHg)	
	Female	Male	Female	Male
<b>BMI(kg/m<sup>2</sup>)</b>	0.365**	0.155**	0.323**	0.213**
<b>WC (cm)</b>	0.452**	0.244**	0.336**	0.152**
<b>WHR</b>	0.115	0.019	0.056	- 0.084
<b>WHtR</b>	0.265**	0.101*	0.172*	0.065

BMI - Body mass index, WC - Waist circumference, WHR - Waist to Hip ratio, WHtR - Waist to Height ratio, SBP - Systolic blood pressure, DBP - Diastolic blood pressure.

\*\* Correlation is significant at the 0.01 level (2 tailed)

\* Correlation is significant at the 0.05 level (2 tailed)



#### **4.17 Correlation between Body Components and Anthropometric Indicators of Subjects**

Tables 4.35 (females) and 4.36 (males) showed significant correlation with anthropometric indicators. BMI and UAC had positive and significant correlations with triceps, biceps, subscapular, suprailiac, waist and hip circumferences, whereas WHR showed no significant correlations with the above except waist and hip circumferences.

**Table 4:35: Correlation between skinfold thickness and anthropometric indicators of female participants (n = 181)**

	BMI (kg/m <sup>2</sup> )	UAC (cm)	TSF(mm)	SbS(mm)	BSF(mm)	SiS(mm)	WC(cm)	HC (cm)	WHR	WHtR
BMI (kg/m <sup>2</sup> )	–									
UAC (cm)	0.372**	–								
TSF (mm)	0.259**	0.395**	–							
SbS (mm)	0.192**	0.427**	0.344**	–						
BSF (mm)	0.201**	0.144	0.062	0.155*	–					
SiS (mm)	0.359**	0.059	- 0.086	-0.008	0.212**	–				
WC (cm)	0.456**	0.394**	0.351**	0.167*	0.379**	0.242**	–			
HC (cm)	0.423**	0.385**	0.415**	0.121	0.237**	0.211**	0.650**	–		
WHR	0.099	0.051	-0.020	0.076	0.219**	0.079	0.543**	- 0.280**	–	
WHtR	0.477**	0.179*	0.242**	0.043	0.311**	0.301**	0.896**	0.587**	0.485**	–

BMI - Body mass index, UAC - Upper arm circumference, TSF - Triceps skinfold, SbS - Subscapular skinfold, BSF - Biceps skinfold, SiS - Suprailiac skinfold, WC - Waist circumference, HC - Hip circumference, WHR - Waist to Hip ratio, WHtR - Waist to Height ratio.

\*\* correlation is significant at the 0.01 level (2 tailed), \* correlation is significant at the 0.05 level (2 tailed), n - sample size

**Table 4:36: Correlation between skinfold thickness and anthropometric indicators of male participants (n = 379)**

	BMI (kg/m <sup>2</sup> )	UAC (cm)	TSF(mm)	SbS(mm)	BSF(mm)	SiS(mm)	WC(cm)	HC (cm)	WHR	WHtR
BMI (kg/m <sup>2</sup> )	–									
UAC (cm)	0.508**	–								
TSF (mm)	0.161**	0.292**	–							
SbS (mm)	0.049	0.128*	0.021	–						
BSF (mm)	- 0.111*	- 0.214**	- 0.174**	0.025	–					
SiS (mm)	0.176**	0.210**	- 0.132*	0.179**	- 0.173**	–				
WC (cm)	0.399**	0.412**	0.168**	0.119*	0.046	0.115*	–			
HC (cm)	0.442**	0.453**	0.209**	0.137**	0.073	0.151**	0.889**	–		
WHR	- 0.074	- 0.073	-0.092	- 0.045	- 0.048	- 0.072	0.316**	- 0.146**	–	
WHtR	0.465**	0.258*	0.138**	0.066	0.071	0.050	0.881**	0.778**	0.299**	–

BMI - Body mass index, UAC - Upper arm circumference, TSF - Triceps skinfold, SbS - Subscapular skinfold, BSF - Biceps skinfold, SiS - Suprailiac skinfold, WC - Waist circumference, HC - Hip circumference, WHR - Waist to Hip ratio, WHtR - Waist to Height ratio.

\*\* correlation is significant at the 0.01 level (2 tailed), \* correlation is significant at the 0.05 level (2 tailed), n - sample size

## CHAPTER FIVE

### 5.0 DISCUSSION

The average height and body weight of participants were  $165.02 \pm 6.95$  cm and  $58.64 \pm 7.43$  kg in females and  $168.98 \pm 6.44$  cm and  $62.11 \pm 6.83$  kg in males respectively and comparable to those of Küpper *et al.* (1998) and Gurruci *et al.* (1998, 1999) and indicating no major discrepancy in sizes and shapes across these years. The present study showed that the anthropometric characteristics of Nigerian undergraduate students were in agreement with the standard values according to their various age groups. Comparison studies showed that the males had higher mean values than the females in most of the anthropometric variables except waist and hip circumferences. The differences were statistically significant ( $p < 0.001$ ) in both sexes including the weight, height, biepicondylar breadth of femur and humerus; and hip circumference.

Females (weight,  $58.64 \pm 7.43$  kg) as compared to males (weight,  $62.11 \pm 6.83$  kg) generally perceived themselves as being a little heavier and wish to look thinner, confirming previous reports in university population (El Ansari *et al.*, 2014), in adolescents (Cocca *et al.*, 2016) and in adults (Kiviruusu *et al.*, 2016). Anthropometric measures and body composition changed across age and gender, consistent with some previous studies (Heyward and Wagner, 2004; Baumgartner *et al.*, 2005; Shephard, 2005).

The average BMI in the present study (females:  $21.50 \pm 2.03$  kg/m<sup>2</sup> and male:  $21.74 \pm 1.93$  kg/m<sup>2</sup>) was similar to those reported by Küpper *et al.* (1998) and Gurruci *et al.* (1999), though these results suggested that variations may occur within populations consistent with studies which reported different relationships between BMI among different ethnicities and races (Gurruci *et al.*, 1998; Deurenberg *et al.*, 2000; Deurenberg and Deurenberg, 2002; Rush *et al.*, 2007; Rush *et al.*, 2009). It was noted that the smaller sample sizes in previous studies may account for the differences. Similarly, Gupta *et al.* (2004) calculated the values of BMI  $>23$

kg/m<sup>2</sup> in Punjabi Bhatia community in Jaipur. Also Sargeant *et al.* (2002) observed the cut - off point for BMI lower than the criteria of WHO in Jamaica.

This findings indicated that approximately 89% of normal-weight females and 91% of males (according to BMI) had a central somatotype. On the other hand, approximately 4% and 5% females and males respectively of individuals were identified as overweight by BMI. Both types of individuals may potentially have health risks without being recognized. Cut-off values for adiposity indices may differ between countries. This may be partly due to different ethnicities having different relationships between anthropometric measures and body composition (Gallagher *et al.*, 1996; Molarius and Seidell, 1998; Deurenberg and Deurenberg, 2002; Flegal *et al.*, 2002).

In the present study, the values for WC were observed to be < 100 cm (females, 72.73 ± 6.89 cm and males, 72.39 ± 5.78 cm) in relation with the study of Lopatynski *et al.* (2003) who found the cut - off point for waist circumference as 99 cm in men of Lublin. Waist circumference (WC) is a strong predictor of intra - abdominal adiposity.

In this findings, the cut - off values of hip circumference were observed as 81.05 ± 6.88 cm and 78.46 ± 8.39 cm for females and males respectively. Goh *et al.* (2004) found the cut - off values for hip circumference as 101.5 cm in Singaporian men (Goh *et al.*, 2004). Ketel *et al.* (2007) found the values of hip circumference as 89.3 cm in Caucasian Dutch adults. Fat predominantly deposited around hips and buttocks does not have risk for health complications such as cardiovascular disease as observed by Goh *et al.* (2004).

Females had higher mean values than males in skinfold thicknesses and the differences were statistically significant ( $p < 0.001$ ) in supraspinale, medial calf and thigh skinfolds. Also noticeable in triceps skinfold was a significant difference ( $p < 0.01$ ) in both sexes. This study was in agreement with previous studies reported (Dierkes *et al.*, 1993; Gurruci *et al.*, 1998, 1999a; Küpper *et al.*, 1998; Hastuti, 2009); also compared to Asians, for example,

Japanese (Kagawa *et al.*, 2007) and Chinese (Lu *et al.*, 2011). Body composition is considered as level of fatness, although skinfolds are a more difficult anthropometric procedure than any other, they provide a more accurate estimate of body fatness than those based only on height, weight and circumference (Lohman *et al.*, 1997; Stewart, 2010; Bushman, 2011). Murguía-Romero *et al.* (2015) observed that WC showed between 63% and 83% specificity/sensitivity to determine the prevalence of metabolic syndrome in young Mexicans. In addition, it has been shown that metabolic indexes are better predictors of dyslipidemia in persons with Down syndrome when those are adjusted by height (Kolodziejczyk *et al.*, 2015). All these data suggest a strong association between body shape and physical health.

In addition, females were found to have higher triceps and subscapular skinfold thickness, a measure of subcutaneous fat deposition and greater percent body fat similar to what has been reported by Shaw *et al.* (2007). The prevalence of overweight and truncal obesity was found to be higher in females compared with their male counterparts similar to what has been reported among Nigerians (Ene-Obong *et al.*, 2012; Okpara & Adediran, 2013), Brazillia (De Assis *et al.*, 2005) and Sudanese children (Nagwa *et al.*, 2011).

These results clearly showed that body topology seems to have statistical difference among the group means of somatotyping components. From the study, the somatotype category for females were central while males were categorized as balanced mesomorph. The somatotype of the general population changes in component dominance (Donahue and Abbott, 1987; Singh and Sidhu, 1980; Amigó *et al.*, 2009).

In this study, the cut - off values of WHR were calculated as  $0.90 \pm 0.06$  (females) and  $0.92 \pm 0.04$  (males) respectively. Another ratio, waist to height (WHtR) has the potential to be globally applicable to different ethnic populations. In the present study, the values of WHtR were found as  $0.44 \pm 0.04$  (females) and  $0.43 \pm 0.03$  (males). Schulze *et al.* (2006) concluded

that WHtR was the strongest anthropometric predictor than any other anthropometrical measure in both men and women of Potsdam, Germany. Furthermore, evidence suggested that WHR was more strongly associated with the mortality index than BMI (Yusuf *et al.*, 2005). However, Taylor *et al.* (2010) demonstrated that central adiposity measurements were positively associated with mortality as was BMI, but only when those individuals with BMI less than  $22.5 \text{ kg/m}^2$  were removed from the analysis, indicating that those adiposity measures could not replace BMI in clinical or public health practice. Our findings (as obtained in the present study,  $\text{WHR} \geq 0.92$  for males and  $0.90$  for females) indicated that previous WHR categories were better than WC categories but not as good as BMI or WHtR categories.

The available WHtR classifications showed a higher sensitivity compared to the WHO international BMI cut-off points. The WHtR obtained ( $\text{WHtR} \geq 0.44$  for females and  $0.43$  for males) can improve the sensitivity of the index. Findings of the current study are consistent with some previous reports that WHtR could better predict obesity health risk than WHR and BMI in some populations (Li & McDermott, 2010; Xu *et al.*, 2010). However, our study indicated WHtR as a better predictor than BMI in females only.

The mean values for blood pressure (SBP - females:  $116.23 \pm 9.65 \text{ mmHg}$  and males:  $117.62 \pm 8.87 \text{ mmHg}$ ; DBP - females:  $78.47 \pm 7.96 \text{ mmHg}$  and males:  $79.44 \pm 6.65 \text{ mmHg}$ ). The Lipid profile mean values obtained (Females: HDL-C  $2.59 \pm 0.71 \text{ mmol/L}$ , TG  $2.49 \pm 0.61 \text{ mmol/l}$ , TC  $4.62 \pm 0.84 \text{ mmol/l}$ , Atherogenic Index  $1.95 \pm 0.77$  and Males: HDL-C  $2.61 \pm 0.77 \text{ mmol/L}$ , TG  $2.47 \pm 0.61 \text{ mmol/l}$ , TC  $4.52 \pm 0.79 \text{ mmol/l}$ , Atherogenic Index  $1.91 \pm 0.75$ ). The average SBP and DBP for males and females were within expected reference ranges which is classified as  $\text{SBP} \geq 140 \text{ mmHg}$  and  $\text{DBP} \geq 90 \text{ mmHg}$ . The mean values for cardiovascular related traits included SBP and DBP, lipids (total cholesterol and triglycerides), lipoprotein cholesterol levels (HDL, LDL and VLDL)

and other variables which have significant impact on cardiovascular disease. Males were observed to have significantly higher values for SBP, DBP and HDL-C compared to females. Females had higher values of LDL-C, VLDL-C, TG, TC and atherogenic index than males. Lipid analysis was compared with WHO guidelines. Mean total cholesterol, triglyceride and lipoproteins (HDL and LDL) were within reference recommended limits for both males and females.

This study explored the relationship between anthropometry and lipid parameters. There were significant negative correlations between BMI, WC and triglycerides and negative correlations between BMI and HDL-C in the total study population. Only few studies examined the relationship between anthropometry and lipid profile (Lu *et al.*, 2011). In Asians, the increased risks associated with obesity have been shown to occur at lower BMI and these populations are predisposed to visceral or abdominal obesity. Therefore, the WHO proposes a lower BMI value to define overweight and obesity in Asia-Pacific region and the cut - off points for overweight and obesity given were 23 kg/m<sup>2</sup> and 25 kg/m<sup>2</sup> respectively (WHO, 2008).

Relationship between basic measured parameters including height, weight, BMI and body composition parameters including waist and hip circumferences were explored and the result showed some positive correlation and others correlated negatively. Arnulfo *et al.* (2017) concluded that body shape measured as somatotype, WC and WHR as an excellent determinant of body image in young university students. The higher the endomorphy, WC and WHR and the lower the ectomorphy, the higher the perception of being overweight or obese is, as well as the wish to be thinner.

Poor environmental conditions such as poor housing and hygienic conditions, unsafe drinking water, heavy workloads, lack of preventive and control measures of locally endemic diseases and infections have been found to be the causes of undernutrition in various other studies,



which were the common characteristics of population groups belonging to low socio-economic strata of the society especially in developing countries (De Onis *et al.*, 2000 Khongsdier, 2002; Sterkowicz *et al.*, 2019).

From the study results, females were more endomorphic than males, while males were more mesomorphic than females. Similar to most studies, females are more endomorphic than males, while males appears to be more mesomorphic (Toselli & Gruppioni, 1999; Gakhar and Malik, 2002; Bhasin and Jain, 2007). However, there are conflicting opinions on the differences in ectomorphy between sexes, while some studies maintain that males are more ectomorphic than females, others stated that females are more ectomorphic than males or both are equally ectomorphic (Toselli & Gruppioni, 1999; Herrera *et al.*, 2004; Kalichman *et al.*, 2004; Buffa *et al.*, 2005; Kalichman *et al.*, 2006). On examination of the sex differences in somatotype component in the present study, it was observed that endomorphy exhibited pronounced sexual differences across the ages considered, whereas in few ages, there were significant sex difference in mesomorphy. However, no sex difference was observed in ectomorphy. This observation for sex difference only in endomorphy and mesomorphy was also noted by some authors (Munoz *et al.*, 2007).

The results of this study showed that regardless of the quantitative changes during growth, the correlation between the components keep on changing, mesomorphy component is more dominant than other components. Despite variations in mean values, the mean somatotype in human remains constant. It is the category of central somatotype. Men belonging to this category typically have a solid bone structure, prominent muscles, medium to low height and moderate to high percentage of subcutaneous fats.

This result coincides with the data from the representative national anthropological study of the Bulgarian population carried out in 1989-1993 at the Institute of Experimental Morphology and Anthropology at the Bulgarian Academy of Sciences (Yordanov *et al.*,

2006). According to it, at the end of the last century, the mean male somatotype of men aged 30-40 years belongs to the category endomorphic mesomorph. It should be noted that the youngest men included in our study display a greater variety of individual body types. Apart from the high percentage of the endo-mesomorphic somatotype, their group contains almost all morphotype categories with the exception of endomorph-ectomorph and the balanced endomorph. This results support the data from another population survey conducted in 2000 in Sofia, regarding age-related increase of the percentage of the endo-mesomorphic somatotype in men above the age of 40 (Koleva *et al.*, 2000). The results show that in the years between 30 and 50, men tend to build muscle rather than fat. The 50 year-old men are more mesomorphic than women, but they are shorter and with less elongated body segments.

The present study found that BP increased with age, BMI and social class. Both the mean SBP (females -  $116.23 \pm 9.65$  mmHg and males -  $117.62 \pm 8.87$  mmHg) and DBP ( $78.47 \pm 7.96$  mmHg and males -  $79.44 \pm 6.65$  mmHg) with BMI (females -  $21.50 \pm 2.03$  kg/m<sup>2</sup> and males -  $21.74 \pm 1.93$  kg/m<sup>2</sup>) were found to be higher in males than females. The mean SBP and DBP levels in this study were similar to those reported in the previous studies (Oyewole and Oritogun, 2009; ogboye, 2012; Senbajo *et al.*, 2013) in Sagamu, Lagos and Abeokuta respectively in South West Nigeria and higher than the values reported by Bugaje *et al.* (2005); Mijinyawa *et al.* (2012) and Ujunwa *et al.* (2013) in Zaria, Kano and Enugu respectively.

Studies from South Africa and India showed higher mean levels of DBP (Moselakgomo *et al.*, 2012; Patel *et al.*, 2014). Differences in BMI and social class in other studies, when compared with this study may be responsible for the variation, as well as the devices used for BP measurement. The auscultation method with an aerobic analogue sphygmomanometer used in this study is the gold standard and measures blood pressure more accurately than

automated devices (Nelson *et al.*, 2008). These findings suggest that factors other than BMI may also play important roles in the determination of BP among adolescents.

The mean BP level was higher for the upper socioeconomic class and lowest in those of lower socioeconomic status. A study from Northern India (Mahajan and Negi, 2015) showed a similar pattern of a rise in BP and Ogboye (2012) also demonstrated a similar relationship in their study in Lagos. Reasons for increasing BP levels as socioeconomic status rises include a sedentary lifestyle and indulgence in unhealthy fast foods that have a high energy and high fat content leading to overweight. The differences in mean BP levels according to the family history of hypertension may suggest that genetic factors have a role to play in BP. Studies by Naim *et al.* (2008), Patel *et al.* (2014) and Mahajan and Negi (2015) also demonstrated this. BMI was the only significant predictor for elevated BP. These findings are consistent with the existing literature, as the measure of adiposity used in this study (BMI) was also positively associated with BP.

It is generally known that cigarette smokers have lower body weights than non-smokers (Lissner *et al.*, 1997) as smoking tends to reduce appetite and to elevate metabolic rate by its thermogenic effects (Lean, 1995). Smoking thus, has been associated with thinness and smokers, even pregnant women, have expressed concern that smoking cessation might lead to increased food intake, decreased energy expenditure and thus, eventually overweight and obesity (Lissner *et al.*, 1997). Increased tobacco consumption in developing countries experiencing demographic transition (rural to urban) has been observed, and a large magnitude of health problems associated with smoking has been reported (Baddoura & Chidiac, 2001). All countries of the Eastern Mediterranean region report a higher prevalence of male smokers than female smokers. Alarming high smoking rates in males were reported in Lebanon (46%), Jordan (48%) and Syria (51%). For females, the highest smoking rates were found in Lebanon (35%) (WHO, 2003). Despite various efforts by different

governmental and nongovernmental organizations, anti-smoking legislation is effectively nonexistent in Lebanon.

Another socially and somewhat sensitive element that might exhibit associations with BMI is alcohol consumption. Alcohol is a source of dietary energy providing 28.8 KJ/g (Yeomans *et al.*, 2009) and its constituent ethanol is the least satiating dietary macronutrient (Yeomans *et al.*, 2009; Almiron-Roig *et al.*, 2003). Physiologically, alcohol may be a contributor to excess body weight by providing an extra energy source and by acting as a catalyst to increased food intake by stimulating appetite (de Castro, 2000; Almiron-Roig *et al.*, 2003; Breslow and Smothers, 2005; Ghandour *et al.*, 2009; Yeomans *et al.*, 2009).

In this findings, all the four anthropometric indicators were significantly different between the groups, this is related to the study conducted by Rezende *et al.*(2018) but BMI had a higher relationship than other anthropometric indices (Fuchs *et al.*, 2005). Some study shows WHtR has better predictor of hypertension (Li *et al.*, 2013) and WHR was observed to be good predictor for the risk of hypertension in some study (Dobbelsteyn *et al.*, 2001).

In this study, results showed that BMI, WC and WHtR had a significant relationship with each other indicating similar predictive power. In cross-sectional study that was conducted by Liu *et al.* (2011), the values did not differ much between them. In a meta-analysis study in the world that compared WC, WHtR and BMI indices, WHtR was the best indicator for measuring obesity (Kodama *et al.*, 2012). In a study conducted by Zabetian *et al.* (2009) in Tehran, the cut - off point for waist circumference was 94.5 as predictor for risk of cardiovascular disease. Also, according to the National Committee of Obstetrics (WC  $\geq$  90) had suggested for obesity and this committee recommends waist circumference  $\geq$  95 cm for appropriate medical interventions. Each population, depending on race and ethnicity has a different cut-off point for anthropometric indices related to the risk of developing diseases such as high blood pressure.

These results revealed that many of the body measurements showed significant correlation with anthropometric indicators. BMI and UAC had a positive and significant relation with triceps, biceps, subscapular, suprailiac, waist and hip circumferences, whereas WHR showed no significant correlation with the above except waist and hip circumferences.

Raja and Kaushik (2009) showed that BMI and WC had strongest significant impact on these two measures compared with WHR. It is also worth mentioning that these relationships did not differ across the age groups. Kumari and Chauhan (2012) and Nazni and Bhuvaneshwari (2013) reported that there were significant differences in the gender-adjusted BMI and WHR between diabetic subjects and controls. Norgon (1994) have shown that BMI correlates highly with body composition and is largely independent of height, enabling an unbiased comparison between short and tall population groups. It should however, be kept in mind that BMI is no more than weight adjusted for height, and that BMI is also related to fat free mass and to a lesser extent, also to body build. However, the positive and significant association between increased BMI, WC, WHR and lipids with the development of cardiovascular diseases among population has been reported by Alhamdan (2008), Owiredu *et al.* (2008), Latiffah *et al.* (2008), Badaruddoza and Kumar (2009), Badaruddoza and Sawhney (2009), Badaruddoza *et al.* (2010a, b) and Afoakwah and Owusu (2011).

A positive correlation exists among the four anthropometric markers of cardio-metabolic conditions (BMI, WC, WHR and WHtR). The strongest correlation was between WC and WHtR and a remarkable level of correlation was also observed between BMI and WHtR. The correlation between BMI and WC was also strong, especially among women. The correlations of WHR with waist circumference and WHtR ratio were moderate. WHR generally had lower correlation with the other anthropometric indices. For further understanding of the correlation matrix and better explanation of the existing correlations, A

correlation analysis was carried out among height, weight, waist circumference and hip circumference measurements. Three relationships were highly correlated: waist and hip circumferences as well as body weight with both waist and hip circumference. The correlation between waist circumference and hip circumference was high indicating the drawbacks of WHR in assessing obesity when both waist circumference and hip circumference co-vary.

One of the key findings of this study was that WC was strongly associated with BP and provided better discrimination of hypertension. WC is an indicator of central fat accumulation and the amount of intra-abdominal adipose tissue (IAAT), high levels of which confer an increased risk of cardio - metabolic disease (Nakamura *et al.*, 1994; Miyawaki *et al.*, 2004). Hence, it might be expected that population data on weight or WC (or an index based on WC such as WHR or WHtR) would be more informative than data on BMI. While BMI is strongly correlated with WC (Freiberg *et al.*, 2008; Flegal *et al.*, 2009), it is a general indicator of excess body weight relative to height, and the correlation of WC with IAAT is greater than that of BMI with IAAT (Shen *et al.*, 1985).

It is biologically plausible that men have greater central distribution of fat (as indicated by greater WC, WHR and WHtR) relative to fatmass (as indicated by BMI) than women. In Asian populations as among Caucasians, men are prone to store visceral fat around the abdomen or organs, whereas women typically accumulate fat around the hips, buttocks and thighs (Norgan, 1997; Wells, 2007). This difference in fat distribution and fat storage can be responsible for different associations of WC and BMI with markers of CVD risk.

WC has been endorsed by several leading national and international organisations as a key indicator of obesity-related health risk (Grundy *et al.*, 2004; Alberti *et al.*, 2006). This is supported by research findings for both Western (Janssen *et al.*, 2004; Leitzmann *et al.*, 2012; Ostchega *et al.*, 2012) and Asian (APCSC, 2006; Huxley *et al.*, 2010; Ashwell *et al.*, 2012)

populations. Some previous studies have shown WC to be a stronger predictor and/or better discriminator of CVD risk factors than BMI (Hojgaard *et al.*, 2008; Beydoun *et al.*, 2011; Staiano *et al.*, 2012; Hajian-Tilaki *et al.*, 2014; Lee *et al.*, 2015; Li *et al.*, 2015). Others have found that WC performs better for men in United States (Janssen *et al.*, 2004), Japan (Sakurai *et al.*, 2006), China (Chen *et al.*, 2011; Deng *et al.*, 2013; Cheong *et al.*, 2015) and Taiwan (Li *et al.*, 2013).

WC has been found to be a stronger predictor and/or better discriminator of diabetes mellitus for women than BMI (Meisinger *et al.*, 2006; Schooling *et al.*, 2010; Mohammadifard *et al.*, 2013) but BMI performs better for women in Japan (Sakurai *et al.*, 2006) and China (Zhou *et al.*, 2009; Chen *et al.*, 2011; Deng *et al.*, 2013) and Taiwan (Li *et al.*, 2013) and for Pima Indians, Native Americans from Arizona (Tulloch-Reid *et al.*, 2003). In our present study, WC was more strongly associated with CVD risk whereas BMI was more strongly associated with BP for both sexes.

The result for the Pearson's correlation between age and blood pressure showed that age was positive and significantly correlated with both blood pressure (SBP and DBP) respectively ( $p= 0.01$ ) and this confirmed the results of previous studies (Daniel *et al.*, 2013). For the combined sex, somatotype components (mesomorphy and endomorphy) positively correlated with SBP and DBP, while ectomorphy negatively correlated. However, mesomorphy significantly correlated with both blood pressures. Several authors had observed that there was an association between somatotype and blood pressure for different populations and across varied age groups in adult and children (Toselli *et al.*, 1997; Williams *et al.*, 2000; Badenhorst *et al.*, 2003; Herrera *et al.*, 2004; Kalichman *et al.*, 2004; Makgae *et al.*, 2007; Neni, 2012). The purpose of studying this relationship by various authors is to see how somatotype contributes to the development of cardiovascular disease. Whereas some authors suggested that there may exist a common physiological pathway regulated by pleiotropic or

epigenetic mechanism in explaining the relationship between body physique and blood pressure regulation (Kalichmann *et al.*, 2004), others maintained that the physiologic pathway is obscured as there are indication of several risks clustering in the development of many organic diseases (Sing *et al.*, 2003; WHO, 2007b).

The results from this study revealed that there was a positive relationship between the endomorphic physique and blood pressure. Other studies have reported similar correlation between endomorphy and blood pressure (Hererra *et al.*, 2004). Both blood pressures exhibited an increasing trend with age in both sexes and this is the usual observation reported in most studies (Neni, 2012; Daniel *et al.*, 2013). This increase of blood pressure with age has been attributed to an aggregation of several factors including the decrease in arterial compliance (as a result of atherosclerosis) and increased body weight (Neni, 2012; Daniel *et al.*, 2013). The mean blood pressure was relatively higher in males than in females. However, the difference in blood pressure was significant between both sexes. This is in accordance with some studies (Daniel *et al.*, 2013; Ramoshaba *et al.*, 2013).

In this study, it was observed that among the somatotype components when correlated with other parameters, only mesomorphy (correlated with both) and endomorphy appeared to correlate with blood pressure, which suggest that there is tendency that increased blood pressure may result from an increase in endomorphic mesomorphy in both sexes. However, on examination of the correlation coefficient values between somatotype and blood pressure for both sexes when treated separately, both endomorphy and mesomorphy correlated with SBP in females, while only endomorphy correlated with blood pressure in males.

Analysis of BMI indicated that the studied groups were averagely normal. Waist circumference is another significant indicator for abdominal obesity. With the reference of WHO data, it is assumed that waist circumference increases the risk of cardiovascular disease in males and females with measurements exceeding 101 and



89 cm respectively. The average waist circumference of male (72.39 cm) and female (72.73) were within the recommended range. In the current study, we found females to have an average wider waist line and hips compared with their male counterparts in contrast to what has been generally reported (Shaw *et al.*, 2007). Females have significantly more body fat mass than males, which is an aspect that could influence their sports performance (Gideon and Olamide, 2017; Jorge *et al.*, 2018). Urquídez-Romero *et al.* (2017) agreed that abdominal obesity is strongly associated to blood pressure in young Mexicans studied.

Both WHtR and WC showed significant association with serum lipids compared with BMI which further underscoring their importance as measures of adiposity and supporting the current preference for employing them instead of BMI as screening tools or surrogate indicators of measure of adiposity especially in children (Martin - Calvo *et al.*, 2016; Nawarycz *et al.*, 2016; Zheng *et al.*, 2016). This study revealed the important role of lipoprotein in determining atherosclerotic changes especially as it relates to changes in its main fractions – HDL, LDL and TG (Shaw *et al.*, 2007; Grober-Gratz *et al.*, 2013; Montali *et al.*, 2015).

Apart from observed findings from this study, several other reasons could explain blood pressure gender differences in this population. In the study by Ujunwa *et al.* (2013), findings similar to this study were noted among Nigerian adolescents, hormonal changes occurring at puberty being attributed to the observed differences. The psychosocial stress at menarche and rapid physiological changes that accelerate completion of puberty among girls have been documented to be associated with an increase in blood pressure among adolescent girls (Ujunwa *et al.*, 2013).

The relationship between body composition and blood pressure levels has well been established in epidemiological studies and in adolescents and childhood blood pressure were positively correlated with age, weight, height as well as height/weight measurements (Salman

*et al.*, 2010; Oduwole *et al.*, 2012). Traditionally, body composition has been estimated by BMI as well as WC and these have been shown to positively correlate with levels of BP. This study found height, weight, BMI and waist and hip circumferences to positively and significantly correlate with both SBP and DBP. Skinfold thicknesses were considered differently in various sites, the correlates were rather weak when compared to BMI.

The individual who had cardiovascular risk profile are more endomorphic and mesomorphic and less ectomorphic than those with a lower cardiovascular risk factor. In our study, correlation analysis indicated that relationships were stronger between cardiovascular factors but the association pattern varied depending on sex. Williams *et al.* (2000) suggested that in case of patients with coronary artery disease (CAD), endomorphy was significantly correlated with abdominal circumference, abdomen-to-hip ratio and abdominal sagittal diameter, while Katzmarzyk *et al.* (1998) found that the somatotype has been related to sum of six skinfolds taken at different sites. Some authors pointed out that redistribution of body fat moves from the periphery towards the trunk with age. This fat mobilization could cause an error in the discrimination of fat surrounds muscle and bone, since part of this fat may have been considered as muscularity (Herrera *et al.*, 2004; Wilmore and Costill, 2005).

In general, in male and female groups, correlation between blood pressure and endomorphy and mesomorphy were positive, however, while blood pressure and ectomorphy were negative. Among females, there were positive association between blood pressure and endomorphy and mesomorphy. This suggests that ponderosity and muscularity have the opposite effect in males and females. However linearity of physique could offer an adaptive advantage among both sexes.

## CHAPTER SIX

### 6.0 CONCLUSIONS, RECOMMENDATIONS AND CONTRIBUTION TO KNOWLEDGE

#### 6.1 Conclusions

This study aimed at investigating the relationship between somatotype and cardiovascular disease risk factors in Nigerian undergraduate students. This relationship were examined using different variables and the results indicated strong relationship. The findings from this study suggest that there exist significant differences between the somatotype and cardiovascular disease risk.

Overall, based on the analysis and within the limitations of the study, the results of this study gave much interesting approach. The findings includes:

- (1) Anthropometric and body composition characteristics of Nigerian undergraduate students in the present study were comparable with those previously reported.
- (2) The study clearly shows that endomorphy and mesomorphy positively correlated with blood pressure while ectomorphy was negatively correlated in both sexes. Endomorphic mesomorph may be a common component of physique that contributes to the development of risk factor for CVD in the population.
- (3) It was deduced from the study that the somatotype category for females were central while males were categorized as balanced mesomorph.
- (4) Both WC and BMI were important assessment for estimation of CVD risk. BMI and WC were similarly and importantly associated positively with blood pressure and lipids. This finding highlights the importance of these intermediate risk factors on the pathway between excess body fat and cardiovascular disease.

(5) The study indicates a weak to moderate association between somatotype and blood pressure; and physique characterized by high endomorphy and mesomorphy, and low ectomorphy.

(6) There were significant association between somatotype components and BMI, depicting that increase in endomorphy and mesomorphy was a risk factor.

(7) In this study we confirmed the positive relation of some socioeconomic factors (socioeconomic status, parents' level of education and employment status) and somatotype components in undergraduate students.

(8) Waist height ratio and waist circumference can be helpful parameters in identifying person with adverse blood lipid profile especially where population based screening is considered.

(9) Relationship between measured parameters including height, weight and BMI and body composition parameters including waist circumference and hip circumference showed positive correlation.

## **6.2 Recommendations**

From the findings of this study, the following are recommended:

(1) For better assessment of somatotyping, the anthropometric and photoscopic techniques when combined together may give better results.

(2) Due to a smaller sample size used, particularly participants of only one institution, future studies are needed comprising a larger cohort and including more variables as well as social determinants for better understanding of differences between the groups.

(3) The covered age range should be extended to study changes into overweight categories at later ages.

(4) It is important to educate the populace on health related issues and association of CVDs, where inactive living and unhealthy diet are increasing rapidly as an impact of urbanization, industrialization and expansion of food markets, especially in developing societies.

(5) The body types should be associated with the long-term health status of the participants to estimate their potential risk for selected diseases. This life course perspective is crucial for a better understanding of the health consequences of overweight and obesity and for development of effective prevention strategies. Anthropometrical characterization of developing and aging populations in terms of body types and of transitions between them constitutes a novel option to investigate onset and progression of obesity and other civilization diseases.

(6) Other physical variables such as health-related physical fitness and coordinative abilities are also needed to establish relationships.

(7) A study be conducted on children with their biochemical analysis.

(8) The present study may be replicated among different ethnicities, heritabilities and geographical conditions.

(9) Further studies are needed in this regard to understand the mechanisms by which somatotype may be associated with risk factor for disease.

(10) Individual socioeconomic characteristics should be collected and used in addition to area-based measurements to reflect socioeconomic circumstances fully. Educational level has been noted to be better associated with cardiovascular risk factors as a measurement of individual socioeconomic status.

### **6.3 Contribution to Knowledge**

(i) This study established a relationship between somatotypes and cardiovascular disease risk factors in Nigerian undergraduate students. The average values obtained for undergraduate students of Kaduna State university as follows: Females (weight  $58.64 \pm 7.43$  kg, height  $165.02 \pm 6.95$  cm, BMI  $21.50 \pm 2.03$  kg/m<sup>2</sup>, WC  $72.73 \pm 6.89$  cm, HC  $81.05 \pm 6.88$  cm, SBP  $116.23 \pm 9.65$  mmHg, DBP  $78.47 \pm 7.96$  mmHg, HDL-C  $2.59 \pm 0.71$  mmol/L, TG  $2.49 \pm 0.61$  mmol/l, TC  $4.62 \pm 0.84$  mmol/l, Atherogenic Index  $1.95 \pm 0.77$ , ENDO  $2.50 \pm 0.28$ ,

MESO  $3.07 \pm 1.48$ , ECTO  $2.54 \pm 1.09$ ) and Males (weight  $62.11 \pm 6.83$  kg, height  $168.98 \pm 6.44$  cm, BMI  $21.74 \pm 1.93$  kg/m<sup>2</sup>, WC  $72.39 \pm 5.78$  cm, HC  $78.46 \pm 8.39$  cm, SBP  $117.62 \pm 8.87$  mmHg, DBP  $79.44 \pm 6.65$  mmHg, HDL-C  $2.61 \pm 0.77$  mmol/L, TG  $2.47 \pm 0.61$  mmol/l, TC  $4.52 \pm 0.79$  mmol/l, Atherogenic Index  $1.91 \pm 0.75$ , ENDO  $2.30 \pm 0.34$ , MESO  $3.25 \pm 1.08$ , ECTO  $2.66 \pm 1.07$ ).

(ii) It contributes to the empirical literature by providing somatotypic values for Nigerian undergraduate students of Kaduna state university (females: 2.5 – 3.07 – 2.54 and males: 2.3 - 3.25 - 2.66).

(iii) The values obtained from the study indicates that male students had higher mean value than female in some anthropometric measurements (weight, height, BMI) and in relation to standard values from WHO. All anthropometric indices were within the WHO normal range.

(iv) Added to Scientific knowledge is that BMI should not be used in preference to other methods, but always be used in conjunction with other anthropometric features and indicators such as WC, WHR and WHtR, as screening tools to classify individuals at risk.

(v) This study contributes to the empirical literature by filling the existing gaps on the comprehensive anthropometry, somatotype, blood pressure and lipid profile in Nigerian undergraduate students.

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## APPENDIX I: ETHICAL CERTIFICATE



### MINISTRY OF HEALTH AND HUMAN SERVICES KADUNA STATE, NIGERIA

MOM/ADM/744/VOL.1/61

6TH MARCH, 2017

#### NOTICE OF APPROVAL AFTER FULL COMMITTEE REVIEW


#### **RELATIONSHIP BETWEEN SOMATOTYPES AND CARDIOVASCULAR DISEASE RISK FACTORS IN NIGERIAN UNDERGRADUATE STUDENTS: A CASE STUDY OF KADUNA STATE UNIVERSITY, KADUNA, NIGERIA**

Name of Principal Investigator :	OYEWALE ABDULWAHEED ABDULAZEEZ
Address of Ethical Approval:	DEPARTMENT OF HUMAN ANATOMY, FACULTY OF MEDICINE, KADUNA STATE UNIVERSITY,
Date of receipt of Application:	10th JANUARY, 2017
Date of Ethical Approved:	27th JANUARY, 2017

This is to inform you that the research described in the submitted Protocol, the Consent Forms, advertisements and other participant information materials have been reviewed and given full approval by the the Health Research Ethics Committee (HREC)

If there is delay in starting the research or any change, inform the HREC so that the dates of Approval can be adjusted accordingly.

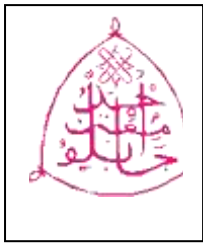
However, Researcher is kindly requested to submit a copy of his/her findings to the State Ministry of Health, please.

  
Dr. BUTAWA NN  
Secretary  
For: Chairman

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Independence way, P.M.B. 2014 Kaduna, Kaduna State Nigeria

## APPENDIX II: INFORMED CONSENT PROTOCOL



### DEPARTMENT OF HUMAN ANATOMY AHMADU BELLO UNIVERSITY, ZARIA



#### INFORMED CONSENT FORM (ICF)

This Informed Consent Form is for Nigerian Undergraduate Students of Kaduna State University, Kaduna. We are inviting you to participate in this research work titled “Relationship between somatotypes and cardiovascular disease risk factors among Nigerian undergraduate students of Kaduna State University, Kaduna, Nigeria”.

**Principal Investigator:** OYEWALE Abdulwaheed Abdulazeez

**Collaborating Investigators:** Prof. S.S. Adebisi  
Prof. B. Danborno  
Prof. S.A. Akuyam

**Name of Organization:** Department of Human Anatomy,  
Ahmadu Bello University, Zaria, Nigeria.

**Name of Sponsor:** Self

**Name of Project:** PhD project

This Informed Consent Form has two parts:

- Information Sheet (to share information about the research with you)
- Certificate of Consent (for signatures if you agree to take part)

You will be given a copy of the full Informed Consent Form

## PART I: INFORMATION SHEET

### **INTRODUCTION**

I am OYEWALE Abdulwaheed Abdulazeez, a postgraduate student (PhD) of the Department of Human Anatomy, Ahmadu Bello University, Zaria, Nigeria, carrying out a research work under the supervision of Prof. S.S. Adebisi, Dr. B. Danborn, and Prof. S.A. Akuyam, from the Department of Human Anatomy, Ahmadu Bello University, Zaria and Department of Chemical Pathology, Ahmadu Bello University Teaching Hospital, Shika – Zaria.

We are carrying out a research work on the “Relationship between somatotypes and cardiovascular disease risk factors among Nigerian undergraduate students of Kaduna State University, Kaduna, Nigeria”. I am informing and inviting you to be part of this research. You do not have to decide today whether or not you will participate in the research. Before you decide, you can talk to anyone you feel comfortable with about the research. There may be some words that you do not understand. Please ask me to stop as we go through the information and I will take time to explain. If you have any questions or concerns about the research, please feel free to contact: Oyewale, Abdulwaheed Abdulazeez, Tel.: 08065639317; Prof. S.S. Adebisi, Tel.: 08100448722; Dr. B. Danborn, Tel: 08139429300; or Prof. S.A. Akuyam, ABUTH Shika, Tel.: 08032889572.

### **PURPOSE OF THE RESEARCH**

The purpose of this study is to investigate the relationship between the somatotypes and cardiovascular disease risk factors in Nigerian undergraduate students of Kaduna State University, Kaduna, Nigeria.

### **WHY ARE YOU BEEN ASKED TO PARTICIPATE?**

You are being invited because you are an undergraduate student of Kaduna State University, within the age range of 17 – 35 year.

### **DESCRIPTION OF THE PROCESS**

It involves collection of certain information about your personal and family characteristics and measurements of some anthropometric parameter: weight, height, biepicondylar breadth, skin fold thicknesses, arm and girth circumferences. Measurements of blood pressure and collection of your blood sample for cholesterol analysis.

### **WHAT WILL HAPPEN DURING THIS STUDY?**

Information pertaining to body composition, blood pressure and cholesterol analysis shall be collected using a questionnaire.

### **POTENTIAL RISKS AND DISCOMFORT**

There is a little risk of pain in blood collection as a result of the syringe. The study does not pose any form of physical, emotional or psychological risks to you.

### **POTENTIAL BENEFITS TO PARTICIPANTS**

The result of this study shall be useful to individual participant to know their body composition and cardiovascular disease risk status. The information collected from you will be used strictly to achieve the objectives of this study and for scientific publication.

### **WILL THERE BE ANY COST FOR PARTICIPATING?**

Aside from your time, there is no cost in taking part in this study.

### **REMUNERATION FOR PARTICIPATION**

Participation will not attract any financial benefit. You will not be provided with any special incentive or travel allowance for you to take part in this research.

### **CONFIDENTIALITY**

Every effort will be made to ensure confidentiality of any identifying information provided by participants in this study. You will not be identified in any reports or publications resulting from the study.

### **SHARING THE RESULTS**

The knowledge that we obtain from doing this research will be shared with you if you so desire before it is made widely available to the public through journal publication. Confidential information will not be shared. We will publish the results in order that other interested people may learn from our research findings.

### **PARTICIPATION AND WITHDRAWAL**



You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. You may exercise the option of removing your data from the study. You may also refuse to answer any questions you don't want to answer and still remain in the study. The investigator may withdraw you from this research if circumstances arise that warrants doing so.

## **RIGHTS OF RESEARCH PARTICIPANTS**

You may withdraw your consent at any time and discontinue participation without penalty. You are not waiving any legal claims, rights or remedies because of your participation in this research study. I assure you that this study have been reviewed and approved by my supervisors and it had received ethics clearance through Health Research Ethics Committee of Kaduna State Ministry of Health and Human services. If you have any questions regarding your rights as a research participant, you can obtain further information about the research or voice your concerns to:

OYEWALE, Abdulwaheed Abdulazeez  
Department of Anatomy,  
Faculty of Medicine,  
Kaduna State University, Kaduna.  
Tel: 08065639317  
E-mail: [abdulwaheedoyewale@gmail.com](mailto:abdulwaheedoyewale@gmail.com)

or

Prof. B. Danbornon  
Department of Human Anatomy  
Faculty of Medicine,  
Ahmadu Bello University, Zaria.  
Tel: 08139429300  
E-mail: [sbdanbornon@yahoo.com](mailto:sbdanbornon@yahoo.com)

PART II: CERTIFICATE OF CONSENT

I have read the foregoing information provided for the study “Relationship between somatotypes and cardiovascular disease risk factors among Nigerian undergraduate students of Kaduna State University, Kaduna, Nigeria” as described herein. I understand that my participation is voluntary. I know enough about the purpose, methods, risks and benefits of the research study to judge that I want to take part in it. I understand that I may freely stop being part of this study at any time. I have received a copy of this consent form and additional sheet to keep for myself. I therefore consent voluntarily to participate as a participant in this research.

Name of Participant:

\_\_\_\_\_

Signature of participant:

\_\_\_\_\_

Date: \_\_\_\_\_

(Day/Month/Year)

**STATEMENT BY WITNESS**

I have witnessed the accurate explanation of the consent form to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Name of witness: \_\_\_\_\_

Signature of witness: \_\_\_\_\_

Date: \_\_\_\_\_

(Day/Month/Year)

**STATEMENT BY THE RESEARCHER/ PERSON TAKING CONSENT**

I have accurately explained the information sheet to the potential participant, and to the best of my ability made sure that the participant understands that the following will be done:

- i. Measurements of body composition
- ii. Measurements of blood pressure
- iii. Collection of blood sample for cholesterol analysis

I confirm that sufficient information, including risks and benefits, to make an informed decision have been fully explained to the participant. The participant was given opportunity to ask questions about the study, and all the questions asked by the participant have been answered correctly and to the best of my ability. I confirm that the individual has not been coerced into giving consent, and the consent has been given freely and voluntarily.

A copy of this Informed Consent Form has been provided to the participant.

Name of Researcher/Person taking the consent:

\_\_\_\_\_

Signature of Researcher/Person taking the consent:

\_\_\_\_\_

Date: \_\_\_\_\_

(Day/Month/Year)

### APPENDIX III

#### PROFORMA FOR THE STUDY/QUESTIONNAIRE

**TITLE: RELATIONSHIP BETWEEN SOMATOTYPES AND CARDIOVASCULAR DISEASE RISK FACTORS AMONG NIGERIAN UNDERGRADUATE STUDENTS OF KADUNA STATE UNIVERSITY, KADUNA, NIGERIA**

#### SECTIONA: SOCIO-DEMOGRAPHIC DATA

<b>i. Research ID</b>								
<b>ii. Date of birth</b>				<b>Age (yrs)</b>				
<b>iii. Sex</b>	1= Female 2=Male		<b>iv. Birth weight (kg)</b>			<b>v. Birth order</b>		
<b>vi. Ethnicity</b>		<b>vii. Marital status</b>			1=Single 2=Married 3=Divorced 4=Widow/Widower			
<b>viii. Department</b>		<b>ix. Level</b>		<b>x. Father's tribe</b>				
<b>xi. Father's occupation</b>			<b>xii. Father's level of education</b>		1= Primary 2= Secondary 3= Tertiary 4= None			
<b>xiii. Mother's tribe:</b>			<b>xiv. Mother's occupation</b>					
<b>xv. Mother's level of education</b>			1= Primary 2= Secondary 3= Tertiary 4= None					
<b>xvii. Do you smoke?</b>		1= present 2= Before 3 = Never			<b>xviii. Do you take alcohol?</b>		0=No 1=Yes	
<b>xix. Do you have Diabetic Mellitus?</b>			0 = No 1=Yes 2= Don't know					
<b>xx. Are you on any Anti-diabetic drug?</b>			0=No 1=Yes					
<b>xxi. Do you have hypertension?</b>			0 = No 1=Yes 2= Don't know					
<b>xxii. Are you on any Anti-hypertensive drug?</b>			0=No 1=Yes					



## **B: ANTHROPOMETRY/ SOMATOTYPING**

<b>i. Weight (kg)</b>		<b>ii. Height (cm)</b>		<b>iii. BMI (kg/m<sup>2</sup>)</b>	
<b>iv. Biepicondylar breadth of humerus (cm)</b>					
<b>v. Biepicondylar breadth of femur (cm)</b>					
<b>vi. Upper arm girth (cm)</b>				<b>vii. Calf girth (cm)</b>	
<b>viii. Triceps skinfold (cm)</b>				<b>ix. Subscapular skinfold (cm)</b>	
<b>x. Supraspinale skinfold (cm)</b>				<b>xi. Medial calf skinfold (cm)</b>	
<b>xii. Biceps skinfold (cm)</b>				<b>xiii. Abdominal skinfold (cm)</b>	
<b>xiv. Suprailiac skinfold (cm)</b>				<b>xv. Thigh skinfold (cm)</b>	
<b>xvi. Waist circumference (cm)</b>				<b>xvii. Hip circumference (cm)</b>	

## **C: PHYSIOLOGICAL AND BIOCHEMICAL PARAMETERS**

### **1. BLOOD PRESSURE**

	1st	2nd	3rd	Average reading
<b>i. Systolic blood pressure (mmHg)</b>				
<b>ii. Diastolic blood pressure (mmHg)</b>				

### **2. FASTING SERUM LIPIDS**

<b>i. High density lipoprotein cholesterol (HDL-C)</b>			
<b>ii. Low density lipoprotein cholesterol (LDL-C)</b>			
<b>iii. Very low density lipoprotein cholesterol (VLDL-C)</b>			
<b>iv. Triglycerides (TG)</b>		<b>v. Total cholesterol (TC)</b>	
<b>vi. Atherogenic index or Atherogenic risk ratio (TC: HDL-C)</b>			

