ASSOCIATION BETWEEN SOME NUTRITIONAL INDICES ANDBIOCHEMICAL PARAMETERS AMONG HYPERTENSIVE PATIENTS ATTENDING SELECTED HOSPITALS IN KANO METROPOLIS

BY

ABDULLAHI MUHAMMAD UMAR

(SPS/17/MBC/00062)

FEBRUARY, 2021

ASSOCIATION BETWEEN SOME NUTRITIONAL INDICES ANDBIOCHEMICAL PARAMETERS AMONG HYPERTENSIVE PATIENTS ATTENDING SELECTED HOSPITALS IN KANO METROPOLIS

BY

ABDULLAHI MUHAMMAD UMAR

(SPS/17/MBC/00062)

A DISSERTATION SUBMITTED TO THE DEPARTMENT OF BIOCHEMISTRY, FACULTY OF BASIC MEDICAL SCIENCES, BAYERO UNIVERSITY, KANO, IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF MASTER OF SCIENCE (NUTRITIONAL BIOCHEMISTRY)

FEBRUARY, 2021

DECLARATION

I hereby declare that this research work with titled "ASSOCIATION BETWEEN THE LEVEL OF ANGIOTENSIN 2 ANDNUTRITIONAL STATUS AMONG HYPERTENSIVE PATIENTS ATTENDING SOME SELECTED HOSPITALS IN KANO METROPOLIS" is the product of my own research efforts; undertaken under the supervision of Dr. A.M. Gadanya and has not been presented and will not be presented elsewhere for the award of degree of Master of science or certificate. All sources of literature have been duly acknowledged.

_____ _____

ABDULLAHI MUHAMMAD UMAR Date
(SPS/17/MBC/00062)

CERTIFICATION

This is to certify that the research work titled "ASSOCIATION BETWEEN THE LEVEL OF ANGIOTENSIN 2 ANDNUTRITIONAL STATUS AMONG HYPERTENSIVE PATIENTS ATTENDING SOME SELECTED HOSPITALS IN KANO METROPOLIS" for the project and the subsequent preparation of this report by Abdullahi Muhammad Umar (SPS/17/MBC/00062) was carried out under my supervision.

_____ _____

Dr. A.M. Gadanya Date

(Supervisor)

APPROVAL

This dissertation has been examined and approved for the award of Master of Science Degree in BIOCHEMISTRY (NUTRITION)

_____ _____

Date

(External Examiner)

_____ _____

Prof. M. S. Sule Date

(Internal Examiner)

_____ _____

Dr. A.M. Gadanya Date

(Supervisor)

Representative of the SPS

_____ _____

Dr. Y. Y. Muhammad Date

(Head of Department)

DEDICATION

This research work is dedicated to Almighty Allah and to my beloved parents, ALHAJI MUHAMMAD UMAR and HAJIYA AISHAT MUHAHAMMAD who have been my constant inspiration.

ACKNOWLEDGEMENTS

All praise is to Allah (S.W.A), the Lord of the world, for giving me the wisdom, courage and the ability to write this report, may the peace and blessing of Allah be upon our beloved prophet Muhammad (S.A.W), his family, companions and those who follow the right path until the day of judgment.

I am indebted to my project supervisor Dr. A.M. Gadanya who despite her enormous engagement guided me and gave useful suggestions and comments, prompt attention to my problems and for scrutinizing the work resulting in the improvement of the manuscript. I also wish to express my sincere thanks to Prof Muhammad Sani Sule, Dr. A.M. Abdulazeez, Dr. S.M. Abubakar and Dr. Lawan H. Adamu for their constant interest, tolerance, criticism, suggestion, purposeful encouragement and correction through the period of this work. May Allah (S.W.T) in His blessing to reward you and your family abundantly.

I am grateful to my lovely parents for their countless effort in proper upbringing, consistent advice, financial support and assurance of my success in life. May Allah (S.W.T) in His Blessing increase them in health, wealth, knowledge, long life and prosperity and make them among the dwellers of paradise.

My heartfelt gratitude goes to Alhaji Labaran Jafaru Dalhatu, his wife Hajiya Khadija Muhammad Umar and his family, Who gave me maximum support, prayers, and encouragement throughout my Msc programme. The prayer and support of my family members and friends cannot be over emphasized. May Allah (S.W.T) in His blessing to reward you abundantly .

In addition I would like to extend my regard and thanks to the courageous friends of mine whose support and useful advice to me can never be forgotten throughout the period of this research work Comrade Nasir Surajo (Class Rep), Comrade Abdullahi Musa (Speaker), Aisha Murtala (Murtizz), muhammad Ali Gambo deserved to be mentioned. I will not forget to appreciate the financial support given to me by brothers and sisters.

ABBREVIATIONS	
TG	TRIGLYCERIDE
TC	TOTAL CHOLESTEROL
LDL	LOW DENSITY LIPOPROTEIN
HDL	HIGH DENSITY LIPOPROTEIN
H	HEIGHT
W	WEIGHT
ABSI	A BODY SHAPE INDEX
CI	CONISITY INDEX
ACE	ANGIOTENSIN CONVERTING ENZYME
ANG II	ANGIOTENSIN II
WHR	WAIS HIP RATIO
WC	WAIST CIRCUMFERENCE
HC	HIP CIRCUMFERENCE
BMI	BODY MASS INDEX

TABLE OF CONTENTS

Declaration - - - - - - - - - - II

Certification - - - - - - - - - - III

Approval - - - - - - - - - - IV

Dedication - - - - - - - - - - V

Acknowledgement - - - - - - - - - VI

Abbreviations - - - - - - - - - VII

Abstract - - - - - - - - - - VIII

CHAPTER ONE - - - - - - - - - 1

INTRODUCTION - - - - - - - - - 1

1.0 BACKGROUND - - - - - - - - - 1

1.1 Statement of Research Problems - - - - - - - 3

1.2 Justification - - - - - - - - - 4

1.3 Aims and Objectives - - - - - - - - 5

CHAPTER TWO - - - - - - - - - 6

LITERATURE REVIEW - - - - - - - - 6

2.1 HYPERTENSION - - - - - - - - 6

2.1.1 Sign and Symptoms - - - - - - - - 10

2.1.2 Causes of Hypertension - - - - - - - - 11

2.1.2.1 Primary Hypertension - - - - - - - 11

2.1.2.2 Secondary Hypertension - - - - - - - 11

2.1.3 Diagnosis - - - - - - - - - 12

2.1.4 Prevention - - - - - - - - - 14

2.2 Obesity - - - - - - - - - - 14

2.2.1 Classification of Obesity - - - - - - - 14

2.2.2 Proportion of Disease Prevalence Attributable to Obesity - - - 17

2.2.5 Assessment of the Obese Patients - - - - - - 22

2.2.6 Global Causes of Obesity and Consequences - - - - - 23

2.2.7 Socioeconomic Status and obesity in Developing Countries - - - 25

2.2.8 Physical Activity Levels and its Impact on Obesity; Current Scenario in Developing Countries - - - - - - - - - - 27

2.3 Renin Angiotensin System - - - - - - - 28

2.3.1 Angiotensin II Peptides - - - - - - - - 31

2.3.2 Angiotensin II Receptors - - - - - - - 33

2.3.3 Angiotensin II Type I Receptor Gene - - - - - - 34

2.4 Serum Lipid Profile - - - - - - - 34

2.4.1 Component if Lipid Profile - - - - - - 35

2.4.2 Procedure and Indications - - - - - - - 35

2.4.3 Implication - - - - - - - - - 36

2.5 Dietary Assessment Methods - - - - - - - 40

2.5.1 Dietary Records - - - - - - - - - 40

2.5.2 Food Frequency Questionnaire (FFQ) - - - - - - 40

2.5.3 Twenty Four Hours Dietary Recall - - - - - - 41

2.3.4 Definition of Anthropometric and Biochemical Data - - - - 41

2.3.4.1Body Mass Index - - - - - - - - 41

CHAPTER THREE - - - - - - - - - 42

MATERIALS AND METHODS - - - - - - - 42

3.1 Materials - - - - - - - - - - 42

3.1.1 Target Population - - - - - - - - 42

3.1.2 Inclusion and Exclusion Criteria - - - - - - - - 42

3.1.3 Sample Size Determination - - - - - - - - 43

3.1.4 Ethical Approval - - - - - - - - - 43

3.1.5 Administration of Questionnaires - - - - - - - 44

3.1.6 Apparatus and Equipments- - - - - - - - - 44

3.1.7 Reagents - - - - - - - - - - 44

3.2 Methods - - - - - - - - - - 44

3.2.1 Specimen collection - - - - - - - - 44

3.2.2 Determination of Blood Pressure - - - - - - - - 45

3.2.3 Determination of ACE activity - - - - - - - - 45

3.2.4 Determination of ANG II Levels - - - - - - - 46

3.2.5 Lipid Profile - - - - - - - - - 47

3.2.6 Data Collection - - - - - - - - - 49

3.2.7 Dietary Assessment - - - - - - - - 50

3.2.8 Anthropometric Data Collection - - - - - - 50

3.2.9 Sample Procedure - - - - - - - - 50

3.3 Statistical Analysis - - - - - - - - 51

CHAPTER FOUR - - - - - - - - - 52

RESULTS AND DISCUSSION - - - - - - - 52

4.0 Results - - - - - - - - - - 52

4.1 Description of Anthropometry and Biochemical Parameters - - - 52

Discussion - - - - - - - - - - 74

CHAPTER FIVE - - - - - - - - - 78

SUMMARY, CONCLUSION AND RECOMMENDATION- - - - - 78

5.1Summary - - - - - - - - - 79

5.2 Conclusion - - - - - - - - - - 80

5.3 Recommendation - - - - - - - - - 80

5.4 Contribution to Knowledge - - - - - - - 81

References - - - - - - - - - - 82

ABSTRACT

The aim of this study is to evaluate the relationship between indices of nutritional status and some selected biochemical parameters among hypertensive patients attending some selected hospitals (Murtala Muhammad Specialist Hospital, Muhammad Abdullahi Wase Teaching Hospital, Sheik Muhammad Jiddah General Hospital, Sir Muhammad Sunusi General Hospital) in Kano metropolis, Kano State, Nigeria. Participants aged from 30-70 years were interviewed in a cross-sectional study conducted in 2019. Anthropometric indices for each participant were measured using standard methods. ELISA kit was used to measure the level of Angiotensin II. Strip was used for the lipid profile (Cholesterol, HDL, LDL and TAG) assay. Angiotensin converting enzyme activity was determined using the Cushman and Cheung method. Correlations and binary logistic regression models were used to determine the association between nutritional status, anthropometry with hypertension. There is lack of nutritional knowledge among the participants; in terms of attitude and practice most of the participants always practice consumption of foods rich in carbohydrates known for body building. Most of the participants do not plan and keep to the balanced food menu and taking of vitamins and minerals supplements. The results also showed a significance difference at $p < 0.05$ in most of the anthropometric indices between the hypertensive and control group (non-hypertensive). There was positive correlations with the ACE activity, ANG II and cholesterol levels with weight circumference, body mass index, visceral fats and weight. Weight also had the highest correlation with ANG II, cholesterol, HDL, LDL and BMI. Dietary diversity scores results shows that there is significant difference at $p < 0.05$ between the hypertensive (6.8 ± 1.94) and the control (5.5 ± 1.25). Cereals and vegetables were the most consumed food among the group. In conclusion, there is an association between the anthropometry indices, nutritional status and ACE activity with hypertension among the study populations.

CHAPTER ONE

INTRODUCTION

1.0 BACKGROUND

Hypertension is a rapidly emerging disease that contributes highly to morbidity and mortality worldwide. Risk factors of the disease not only include obesity, sedentary lifestyle, and age, but also genetics and environmental factors. Hypertension is usually asymptomatic as most patients only know they are hypertensive when they present to the hospital with complications. This happens mostly in developing countries where very minimal importance and resources are allocated to non-communicable diseases (Muhamedhussein *et.al.*, 2016). Abegunde *et al. (*2007) reported that 80% of deaths that result from non-communicable diseases occur in the developing world. Obesity and hypertension are the most prevalent cardiovascular risk factors and are associated with nutritional intakes and needs of individuals (Tibazarwa *et al.*, 2009). It is estimated that by 2030, 85% of the 23 million cardiovascular deaths will be in low and middle-income countries (Mathers and Loncal, 2006).

Nutritional status results from the relationship between nutrient intake and requirements; and the body's ability to digest, absorb and use these nutrients (FAO, 2007).Nutritional status is optimal when nutritional intake and needs are equal. A deviation from this results in malnutrition, a pathological condition caused by unbalanced or insufficient diet or defective assimilation and utilization of foods. Malnutrition refers to all deviations from adequate nutrition, which can be due to inadequacy of food (under nutrition) resulting in protein energy malnutrition, micronutrient deficiencies among others; or due to excess food intake (over nutrition) resulting in metabolic disorders such as obesity, diabetes and cardiovascular diseases. Thus, a poor nutrition reduces immunity, increases susceptibility to disease, impairs physical and mental

development, and reduces productivity (WHO, 2019). Inadequate diet and physical inactivity causes overweight and obesity, both of which are responsible for the significant increase in hypertension worldwide, especially in adolescents (Bibiloni *et al.,* 2013).Also, the restriction of vital nutrients during pregnancy leads to intrauterine growth restriction on organs and their functions, resulting in low birth weight and placental size, both of which contribute to the development of hypertension and other chronic diseases in adulthood (Reinhard *et al*., 2003; Perez-Escamilla, 2018). Hypertension, once considered a problem in high-income countries, is now on the rise in low- and middle-income countries, particularly in urban settings (WHO, 2019).

Obesity is associated with elevated leptin known to enhance platelet aggregation and arterial thrombosis, promote angiogenesis, and impair arterial distensibility, ultimately resulting in hypertension (Singhal *et al*., 2002). Several studies have suggested that renin angiotensin system (RAS) induces hypertension via adipose tissue and systemic circulation; and obesity is associated with the activation of both systems in humans (Jiang *et al*., 2016), most diet-induced obese rodents and genetic models (Yasue *et al.,* 2010). Obesity increases plasma levels of angiotensinogen as a result of adipocyte hypertrophy. Angiotensinogen is a precursor of angiotensin II (Ang II), produced by the action of renin and angiotensin converting enzyme (ACE) (Uckaya *et al*., 1999; Engeli *et al*., 2005). Ang II induces systematic vasoconstriction, direct sodium and water retention and increases aldosterone production. It also determines a high salt-sensitive blood pressure condition in obesity as it is produced at high rates and is not suppressed by volume expansion. It mediates its effects via cell surface receptors: angiotensin II type 1 receptor (ATR1) and angiotensin II type 2 receptor (ATR2); with ATR1 responsible for the vascular action of Ang II (Ingelfinger, 2009). The RAS is the most important blood pressure

control mechanism, and genes such as angiotensinogen (AGT), ACE, and ATR1, encoding components of this system have been strong candidates for investigating the genetic basis of hypertension (Hindorff*et al*., 2002). Some evidence of association between these genes have been reported (Kingue *et al*., 2015), though not always significant (Steyn *et al*., 2001).Variants of AGT gene have been shown to be associated with serum AGT in black and white children as well as in Chinese children (Cheng *et al*., 2012). Also, M235T and T174 M, both variants of AGT are significantly associated with hypertension in some populations (Mohana *et al*., 2012) but not in others (Rudnichi*et al*., 2004). Several studies and meta-analysis associate ATR1 with essential hypertension (Wang *et al*., 2010); however, conflicting results are also reported by few studies (Sugimoto *et al*., 2004). The A1166C polymorphism in the 3′ untranslated region of the ATR1 gene was detected in study by Bonnardeaux *et al*. (1994) and found to be associated with hypertension. Annette *et al.* (2013) using a mouse model also reported an increase in the activity of the AT1R in the adipose tissue, leading to an increase in the adiposity of the tissue, and subsequent increase in systolic pressure.

1.1 STATEMENT OF RESEARCH PROBLEM

Malnutrition affects every country in the world in various forms with differing reflections, critical problems and characteristics. While developing countries are concerned with having access to adequate food and micronutrients, for developed countries, adherence to a healthy and proper diet is paramount (Barrilla Center for Food and Nutrition, 2011). These nutrition disparities are due to the prevalence of undernutrition and overnutrition, or both, in inequitable social conditions (Perez-Escamilla, 2018). The coexistence of under- and overnutrition in low and middle-income countries (LMICs), which include Nigeria, creates a double burden of malnutrition (DBM) (Perez-Escamilla, 2018) and predisposes the population to chronic

diseases.According to World Health Organization (2018), the prevalence of overweight in adults (>18) rose from 35.7% in 2010 to 38.9% in 2016 and obesity rose from 11.2% to 13.1% in the same years. Overall, there were 2.01 billion overweight adults of which 678 million were obese. Obesity, a consequence of overnutrition, and one of the major causes of chronic diseases, such as diabetes, cancer, hypertension and other cardiovascular diseases, is globally on the increase(GNR, 2017). Hypertension affects about 1.1 billion people worldwide and its prevalence is expected to increase by 60% by the year 2025. It is estimated that while LMICs bear 78% of all deaths from non-communicable diseases, they are also hard-hit with consequences of undernutrion (WHO, 2018). In Nigeria, there are over 17.92 million hypertensive individuals (11.2% of the population) (Akinkugbe, 1997; Ulasi *et al*., 2011).

1.2 JUSTIFICATION

Cardiovascular diseases, the leading cause of mortality worldwide, particularly hypertension and diabetes, are the main illnesses associated with obesity. Obesity is highly influenced by genetics, and alteration of ReninAngiotensin System components have been associated with hypertension, nevertheless, the mechanisms underlying obesity-associated hypertension remains to be not adequately investigated (Tibazarwa *et al.*, 2009). Also, there is no consensus on the effects of some RAS parameters on overweight and obesity, probably due to study designs, sampling methods and specific populations. There is an increasing middle class in Nigeria, and with it is the increasing prevalence of obesity, which predisposes to chronic diseases, with hypertension being the most common chronic medical problem promoting visits to hospital(Kingue *et al*., 2015). However, there is limited information on RAS parameters in relation to obesity and hypertension status in Kano State, Nigeria.

1.3 AIM AND OBJECTIVES

The aim of this study was to evaluate the relationship between indices of nutritional status, and some selected biochemical parameters among hypertensive patients attending some selected hospitals (Murtala Muhammad Specialist Hospital, Muhammad Abdullahi Wase Teaching Hospital, Sheik Muhammad Jiddah General Hospital,Sir Muhammad Sunusi General Hospital) in kano metropolis, Kano State, Nigeria.

OBJECTIVES

1. To determine the nutritional status, anthropometric indices (Height, Weight, Hip and Waist circumference, Conicity index, Body fat and muscle composition and Visceral fat) and dietary diversity of (140) hypertensive patients attending some selected hospitals (MMSH), (MAWSH), (SMJGH),(MSGH) in Kano metropolis, Kano State, Nigeria.
2. To determine the serum lipid profile (Cholesterol, HDL,LDL and TAG) in Hypertensive patients and apparently healthy non-hypertensive Individuals attending some selected hospitals (MMSH), (MAWSH), (SMJGH), (MSGH) in Kano metropolis, Kano State, Nigeria.
3. ACE activity and ANG II levels in Hypertensive patients andapparently healthy non-hypertensive Individuals attending some selected hospitals (MMSH), (MAWSH), (SMJGH), (MSGH) in Kano metropolis, Kano State, Nigeria.

CHAPTER TWO

LITERATURE REVIEW

2.1 HYPERTENSION

Hypertension (HTN or HT), otherwise called high blood pressure or blood vessel hypertension, is an incessant disease where blood pressure in the arteries is perseveringly raised. Blood pressure can be determined by two estimations, the systolic and diastolic pressures, which are the high and low pressures, respectively in the circulatory system. The systolic pressure happens when the left ventricle is most contracted; the diastolic pressure happens when the left ventricle is most loose prior to the second contraction. Typical blood pressure at rest is within the scope of 100–140 millimeters mercury (mmHg) systolic and 60–90 mmHg diastolic. Hypertension is present if the resting blood pressure is perseveringly at or over 140/90 mmHg for most grown-ups; different indices apply to children(James*et al*., 2013).

Hypertension more often than not does not cause side effects at first, however sustained hypertension after some time is a major risk factor for hypertensive coronary disease, coronary artery disease (Lewington *et al*., 2002), stroke, aortic aneurysm, peripheral artery disease, and chronic kidney disease. Hypertension is categorized as either essential (basic) hypertension or secondary hypertension. Around 90–95% of cases are classified as essential hypertension, characterized as hypertension with no underlying fundamental cause (Carretero and Oparil, 2000). The other 5-10% of cases are categorized as secondary hypertension, characterized as hypertension because of an identifiable cause, for example, chronic kidney disesase, narrowing of the aorta or kidney arteries, or an endocrine disorders, for example, overabundance aldosterone, cortisol, or catecholamines.

Dietary and changes in lifestyle can lower blood pressure and the danger of health complications would decrease, despite the fact that treatment with drug is still frequently vital in individuals for whom lifestyle changes are insufficient or not effective. The treatment of moderately high blood pressure (characterized as >160/100 mmHg) with drugs is related with an improved future life expectancy (Musini *et al*., 2009). The advantages of treatment of blood pressure that is between 140/90 mmHg and 160/100 mmHg are less clear, with some certain findings, discovering absence of a proven benefits and others discovering advantage (Sundtrom *et al.*, 2015; Xie *et al*., 2015).

Hypertension have been a major public health issue around the world, prevalence of hypertension is estimated about 1 billion individuals, It causes about 7.1 million deaths each year and 4.5% of the burden of the disease, which is estimated as 64 disability adjusted life years (DALYs). The connection between hypertension and risk of cardiovascular diseases occasions is continuous, steady, and free of other risk factors. The higher the BP, the more prominent is the opportunity of heart attack, stroke, heart failure and kidney diseases (Chobanian *et al*., 2003). The burden of non-communicable diseases (NCDs), including hypertension, is fast rising globally, and reports from the 2013 World Health Day global brief on hypertension shows that Africa, in particular, is worst hit (WHO, 2013a).WHO public health experts and stakeholders have declared NCDs a global priority, as documented in the 2011 United Nations (UN) high-level meeting, with a target towards reducing this growing burden in Africa and other low and middle-income countries (LMICs) (Beaglehole *et al.*, 2011), where an existing burden from many infectious diseases has contributed to a double burden of disease (Aikins *et al*., 2010). Hypertension is estimated to affect about one billion people worldwide and is a major risk factor for many cardiovascular diseases (WHO, 2013b). Cardiovascular diseases are responsible

for about 17 million deaths globally, with complications from high blood pressure resulting in about 7.5 million deaths and 57 million disability-adjusted life years (DALYs) worldwide, both accounting for about 12.8 and 3.7% of global deaths and DALYs, respectively (WHO, 2013b;Kearney *et al.*, 2005). Nigeria, currently with a population of over 160 million, is the most populous African country (World Bank, Nigeria, 2013), and the prevalence of hypertension in the country hugely contributes to the overall burden in Africa (WHO AFRO, 2005). In 2008, the WHO estimated hypertension prevalence of 42.8% in Nigeria (WHO, 2011). This is believed to be due to an increasing adult population, rapid urbanization and uptake of western lifestyles, including high consumption of processed foods (with high salts and fats), tobacco and alcohol products (Bello, 2013; Mezue, 2013).

The 1997 Nigerian national NCDs survey Committee reported a hypertension prevalence of 11.2% in both sexes, which was then about 4.33 million hypertension cases in people aged above 15 years (Akinkugbe, 1997). The survey has underestimated the prevalence of hypertension in Nigeria, as the diagnosis was based on the definition of hypertension (Bello, 2013; Ogah, 2006). From the 2003 national NCDs survey conducted mainly in the south-west region, which was based on SBP at least 140mmHg and/or DBP at least 90 mmHg, the overall prevalence of hypertension was 28.9% (Onyemelukwe *et al*., 2003). In addition, recent surveys in various parts of Nigeria based on at least 140/90mmHg have also shown a higher prevalence of hypertension, ranging from 25.0 to 36.6% (Mezue, 2013).

Reducing the burden of hypertension would lower mortality and disability in moderately aged and older peoples and lead to an increase in personal life satisfaction. Decrease of hypertension predominance could be accomplished through risk factor prevention programs just as utilizing

low-cost management. In any case, in most nations of the African region, implementation of these programmes and approaches is hampered by shortage of information on the prevalence and control levels of hypertension. Scarcity of information is sometimes defined as non-existence of the problem (WHO, 2003). There is scarcity of hypertension predominance in many population of Nigeria. Accordingly, burden of hypertension in these populations may be underestimated and might leave the disease undiagnosed and untreated. Uncontrolled hypertension obviously puts a substantial strain on health care delivery system. Assessing the predominance of hypertension in populations of Nigeria would be helpful in efforts to control hypertension and different NCDs.

The worldwide prevalence of hypertension is on the expansion in 2000, 972 million individuals had hypertension with a prevalence rate of 26.4%. These are anticipated to increase to 1.54 billion people and prevalent of 29.4% in 2025 (Kearney *et al*., 2005).Hypertension was believed to be uncommon in rural areas of Africa (Shaper *et al*., 1969; Pobee *et al.*, 1977). It is presently winding up progressively common as urbanization increases and this has been appeared in amany findings in Africa (Cooper *et. al.,* 1998). A recent community based study semi-urban and rural population conducted in Enugu, Nigeria put the prevalence of hypertension in Nigeria at 32.8% (Adeloye *et al,* 2015). Uncontrolled hypertension is related with extreme end-organ damage, for example, coronary disease, stroke, renal disease and visual impairment (Cressman and Gifford, 1983; Post *et al*., 2003; Khakurel *et al.*, 2009) .These serious complications can be averted by sufficient blood pressure control (Cuspidi *etal*., 2000; Neal *et al*., 2000).

The anticipation of prevention and control of hypertension has not received due consideration in many developing countries in spite of the fact that it is one of the most modifiable risk factors for cardiovascular diseases. Awareness, treatment and control of hypertension are very low in

these nations as health care facilities are overpowered by different priorities, which include HIV/AIDS, tuberculosis and malaria. Reliable epidemiologic information is helpful for the design and implementation of effective strategies for the prevention and control of hypertension. Limited information on the patterns of prevalence propose that it has increased in economically developing nations in recent years while it remained steady or decreased in developed nations (Kearney *et al*., 2005). Because of changes in patterns of prevalence and epidemiological study of hypertension, the study should be often revisited on its prevalence to generate recent information in the developing countries specifically sub-Saharan Africa. In such manner, efforts should be made to generate comparable information to yield valuable data needed to fabricate the experimental proof base that ought to be amassed so as to trigger the vital arrangement reaction.

2.1.1 Sign and Symptoms

Hypertension is infrequently accompanied by many side effect, and its identification is normally through screening, or when looking for health care services for an unrelated issue. Some with hypertension report headaches (especially at the back of the head and toward the beginning of the day), just as light headedness, vertigo, tinnitus (humming or murmuring in the ears), adjusted vision or fainting episodes. These manifestations, however, may be identified with related anxiety rather than the hypertension itself (Marshall *et al.,* 2012). On physical assessment, hypertension might be related with the presence of changes in the optic fundus seen by ophthalmoscopy. The seriousness of the progressions typical of hypertensive retinopathy is evaluated from I-IV; grades I and II might be difficult to separate. The severity of the retinopathy corresponds generally with the duration as well as the severity of the hypertension (Fisher and Williams, 2005).

2.1.2 Causes of Hypertension

2.1.2.1 Primary Hypertension

Hypertension results from complicated interactions of genes and environmental factors. Various genetic variants that are common with little impacts on blood pressure have been recognized (Ehret *etal*., 2011) just as some genetic variants that are rare with enormous effects on blood pressure, however the genetic basis of hypertension is still not clearly understood. Blood pressure tends to rise with aging and the risk of getting to be hypertensive in later life is impressive. Many environmental factors have an influence on blood pressure. Taking high amount of salt raises blood pressure in individuals that are sensitive to salt. Also lack of physical activity such as exercise, stress, obesity and depression may play a role in individual cases. The possible role of different factors, for example, vitamin D deficiency and caffeine consumption are less. Resistance to insulin, which is normal in obesity and is a major segment of disorder X (or the syndrome), is additionally thought to contribute to the rise in blood pressure. Occasions in early life, for example, low birth weight, maternal smoking, and lack of breast milk for feeding the child might be the risk factors for adult essential hypertension, despite the fact that the mechanisms linking these exposures to adult hypertension remain indistinct (Lawlor and Smith, 2005).

2.1.2.2 Secondary Hypertension

Secondary hypertension is established from an identifiable cause. Kidney disease or kidney failure is one of the most common causes of secondary hypertension. It can also be caused as a result of endocrine conditions, for example, Cushing's disorder, hyperthyroidism, acromegaly,

hypothyroidism, Conn's disorder or hyperaldosteronism, pheochromocytoma and hyperparathyroidism (O'brien *et al*., 2007). Other causes of secondary hypertension include sleep apnea, obesity, coarctation of the aorta, pregnancy, excessive consumption of alcohol and certain recommended medicine, natural cures and illicit drugs (O'brien *et. al*., 2007; Grossman and Messerli, 2012). Exposure to Arsenic through drinking water has been shown to correspond with rise in blood pressure (Abhyankar *et al*., 2012; Jieying *etal*., 2015)

2.1.3 Diagnosis

Hypertension is diagnosed based on persistently raised blood pressure. Generally, the National Institute of Clinical Excellence suggests three separate sphygmomanometer measurements at month to month interval. The American Heart Association proposed that at least three measurements on at least two separate visits to the health care center. Ambulatory monitoring of blood pressure over 12 to 24 hours is the most precise strategy to confirm the diagnosis (Siu, 2015). A special case to this is those with exceptional hypertension readings particularly when there is poor organ function. Assessment of the hypertensive individuals should include a complete and comprehensive history and physical assessment. With the accessibility of 24-hour ambulatory blood pressure monitors and home blood pressure machine, the significance of not wrongly diagnosing the individuals who have white coat hypertension has prompted change in protocols. In the United Kingdom, current best practice is to follow up a single raised clinic reading of blood pressure with ambulatory measurement, or less preferably with home blood pressure monitoring throughout 7 days. The United States Preventative Services Task Force additionally suggests getting measurement outside the Healthcare centers (Siu, 2015). Pseudo hypertension in the older or non-compressibility artery disorder may likewise require

consideration. This condition is believed to be as a result of calcification of the arteries in abnormal high blood pressure reading with a blood pressure cuff while intra-arterial measurements of blood pressure are normal (Franklain *et al*., 2012). When blood pressure increases upon standing, it is called orthostatic hypertension (Kario, 2009). When the determination of hypertension has been made, healthcare providers should endeavor to recognize the basic causes on the risk factors and other different symptoms, if present.

Secondary hypertension is increasing in preadolescent children, with most cases caused by kidney disease. Essential or primary hypertension is increasing in adolescents and has multiple risk factors, including obesity and a family history of hypertension (Luma and Spiotta, 2006). Laboratory tests can also be performed to identify possible causes of secondary hypertension, and to determine whether hypertension has caused damage to the heart, eyes, and kidneys. Additional tests for diabetes and high cholesterol levels are usually performed because these conditions are additional risk factors for the development of heart disease and may require treatment (Carretero and Oparil, 2000). Serum creatinine is measured to assess for the presence of kidney disease, which can be either the cause or the result of hypertension. Serum creatinine alone may overestimate glomerular filtration rate and recent guidelines advocate the use of predictive equations such as the Modification of Diet in Renal Disease (MDRD) formula to estimate glomerular filtration rate (eGFR) (Chobanian *et al.*, 2003). eGFR can also provide a baseline measurement of kidney function that can be used to monitor for side effects of certain antihypertensive drugs on kidney function. Additionally, testing of urine samples for protein is used as a secondary indicator of kidney disease. Electrocardiogram (EKG/ECG) testing is done to check for evidence that the heart is under strain from high blood pressure. It may also show whether there is thickening of the heart muscle (left ventricular hypertrophy) or whether the heart

has experienced a prior minor disturbance such as a silent heart attack. A chest X-ray or an echocardiogram may also be performed to look for signs of heart enlargement or damage to the heart (O'Brien *et al.,* 2007).

2.1.4 Prevention

Much of the disease burden of high blood pressure is experienced by people who are not labeled as hypertensive. Consequently, population strategies are required to reduce the consequences of high blood pressure and reduce the need for antihypertensive drug therapy. Lifestyle changes are recommended to lower blood pressure, before starting drug therapy. The 2004 British Hypertension Society guidelines (Williams *et al.,* 2004) proposed the following lifestyle changes consistent with those outlined by the US National High BP Education Program in 2002 for the primary prevention of hypertension: maintain normal body weight for adults (e.g. body mass index 20–25 kg/m2),reduce dietary sodium intake to <100 mmol/ day (<6 g of sodium chloride or <2.4 g of sodium per day),engage in regular aerobic physical activity such as brisk walking (≥30 min per day, most days of the week),limit alcohol consumption to no more than 3 units/day in men and no more than 2 units/day in women, consume a diet rich in fruits and vegetables (e.g. at least five portions per day). Effective lifestyle modification may lower blood pressure as much as an individual anti-hypertensive drug. Combinations of two or more lifestyle modifications can achieve even better results (Williams *et al.,* 2004).

2.2 OBESITY

According to the World Health Organization Media Centre (WHO, 2013a), in 2008, the worldwide prevalence has more than doubled since 1980. A number of studies have reported that in each surge in weight, there is an increase in the risks for coronary heart disease, type 2

diabetes, cancers (endometrial, breast, and colon), hypertension, dyslipidemia, stroke, sleep apnea, respiratory problems, osteoarthritis, and gynecological problem (CDC, 2013). Demographic, economic development, environmental, and cultural changes have been impressive for last 30 years, particularly from 1970 to 1999, in developing regions (Momteiro *et al.,* 2002). During this period, a continuous reduction in underweight with a simultaneous increase in obesity has been reported (Momteiro *et. al.,* 2002). If current trends in obesity prevail, total healthcare costs attributable to obesity could reach up to the range of $861 to $957 billion by 2030 in the US (Go *et al*., 2013).

Changes in diet for the past 30 years have been significant in terms of more fat, more meat, added sugars and bigger portion sizes. "Nutrition transition," termed as a combination of improved access to food, decreased physical activity level (PAL) has been identified to be the prime risk factor for the increasing prevalence of overweight and chronic metabolic diseases in the developing countries (Hoffman, 2004). Such dietary patterns' alterations are often the manifestations of societal and environmental changes that emerge as a result of a lack of supportive policies in health, agricultural, transport, urban planning, food processing, distribution, marketing, and educational sectors (WHO, 2013b).

Initially, such dietary shifts and the emergence of obesity were primarily related to the higher socioeconomic (SE) strata of the populations among developing countries (Caballero, 2007). More recent trends demonstrate a shift in the prevalence from the higher to the lower socioeconomic level (Caballero, 2007). For instance, to date, in Mauritius (middle-income country), recent studies indicate clearly that obesity is on the rise on several target populations, namely, among middle aged (Dunneram and Jeewon, 2013) and postmenopausal women (Bhurosy and Jeewon, 2013) and adolescents of low SES (Fokeena and Jeewon, 2012). Notably,

low level of education and moderate PAL, cost per calorie, and weight of food items are important mediators identified in the SES BMI relationship (Fokeena and Jeewon, 2012). Various number of studies conducted in developing countries to assess the obesity-socioeconomic status (SES) relationship is minimal (Xiao *et al*., 2013). Moreover, discrepancies in studies can also be attributed to the use of a single or a combination of SE indicators as each SE variable has its own strengths and drawbacks when linked to BMI (Mclaren, 2007). Thus, understanding the concomitant outcomes of various SES indicators could yield important etiological insights into the SES-obesity relationship among developing countries (Dahly *et al*., 2010).

Obesity has resulted in many serious health issues that are potentially life threatening, including hypertension, increased risk for coronary disease, type II diabetes mellitus, increased unexplained heart failure, infertility, hyperlipidemia, higher prevalence of prostate, colon, endometrial, and breast cancer (Bloomgarden, 2006). Though the relationship between obesity and hypertension is well established in children and adults (Hall *et al.*, 2002), but the mechanism in which obesity directly lead hypertension is under investigation (Kotsis *et al.*, 2005). The amount of intra-abdominal and intra-vascular fat, sodium retention leading to increase angiotensin system, are the factors that were considered to play important functions in the pathogenesis of obesity-related hypertension,

2.2.1 Classification of Obesity

The initial step in evaluation of obesity is calculation of BMI. To measure BMI, one begins by weighing the patient in underclothes and no shoes. Height is measured without shoes. BMI is calculated by dividing weight (in kilograms) by square height (in meters). When measuring

weight in pounds and height in inches, the weight is divided by the square height and the quotient is multiplied by 703, as BMI is always reported and interpreted in kilograms per square meter. Most clinicians have an available BMI table that easily allows the clinician to correlate weight with BMI for a given height and shows a range of healthy weights for that height. BMI has replaced percentage ideal body weight as a criterion for assessing obesity for several reasons. BMI correlates significantly with body fat, morbidity, and mortality, and it can be calculated quickly and easily in a busy clinical setting. Furthermore, recommendations for treatment of obesity are based on BMI. A BMI of 25 kg/m^2 is the generally accepted threshold for identifying a patient at higher risk for obesity-related diseases, most notably type 2 diabetes, hypertension, and cardiovascular disease (Lyznicki *et al.*, 2001). Risk of death begins to increase at a BMI of 23 kg/m^2 when compared with the lowest risk group (BMI, 19.0 to 21.9 kg/m^2) (Aronne, 2001). Medical risk rises progressively with increasing degrees of obesity beginning with overweight, defined by BMI between 25.0 and 29.9 kg/m^2, through class I obesity (BMI, 30.0 to 34.9 kg/m^2), class II obesity (BMI, 35.0 to 39.9 kg/m^2), and class III or extreme obesity (BMI 40 kg/m^2) (Hirsch *et. al.*, 2001). More than 80% of deaths estimated to be caused by comorbidities associated with obesity occur in patients with a BMI of at least 30 kg/m2 (Aronne, 2001).

2.2.2 Proportion of Disease Prevalence Attributable to Obesity

The prevalence of diseases that attributed to Obesity was presented in Table 1. In which type 2 diabeties have high percentage of attribution to obesity having 61%.

Table 1. **Proportion of Disease Prevalence Attributable to Obesity**

Disease	Prevalence (%)
Type 2 diabetes	**61**
Uterine cancer	**34**
Gallbladder disease	**30**
Osteoarthritis	**24**
Hypertension	**17**
Coronary heart disease	**17**
Breast cancer	**11**
Colon cancer	**11**

This classification system of obesity by BMI was developed by the World Health Organization Obesity Task Force and has been adopted by the Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults, a group assembled by the National Heart, Lung, and Blood Institute of the National Institutes of Health (World Health Organization, 1997; NIH, NHLBI 1998).

Waist circumference is an important measure of obesity risk. Waist circumference is measured at the level of the top of the right iliac crest. The measuring tape should be snug but not compressing the skin and held parallel to the floor. The measurement is made at normal

respiration (NIH and NHLBI, 1998). A high-risk waist circumference is accepted to be 35 inches or greater for women and 40 inches or greater for men. Waist circumference is a practical indicator of visceral abdominal fat. Evidence suggests that abdominal fat carries a higher health risk than peripheral fat, and that the visceral fat component correlates the most strongly with increased risk (NIH and NHLBI, 1998). Some epidemiological studies have found the waist-to-hip ratio to correlate with increased risk for diabetes, coronary heart disease (CHD), and hypertension (NHLBI, 1998); however, this measure is not established as an independent risk factor. Waist circumference also has been found to be a superior indicator of abdominal fat distribution (NIH and NHLBI, 1998). The truncal fat distribution indicated by an increased waist circumference correlates with the hypertrophic form of obesity. Hypercellular obesity, which is characterized by an increased total number of fat cells, typically affects patients with a BMI 40 kg/m^2 but may be a lower risk form of disease.

In hypertrophic obesity, existing fat cells enlarge and produce proteins and metabolites involved in the pathophysiology of obesity (Bray and Ryan, 2000). These proteins include lipoprotein lipase, which contributes to hydrolysis of the triglycerides of very-low-density lipoproteins (VLDL) and chylomicrons, and cytokines (tumor necrotizing factor and interleukin-6), as well as angiotensinogen (Bray, 1998). The hypertrophied fat cell also produces leptin, a hormone involved in animal models of obesity. Hypertrophic obesity correlates with metabolic complications of obesity, including impaired glucose tolerance, adverse lipid profile, hypertension, and CHD (Bray and Ryan, 2000). Because waist circumference is an independent risk factor for increased risk of complications from obesity, treatment guidelines include this measurement as a parameter in algorithms designed to determine appropriate obesity treatment. Waist circumference may have additional value in the elderly, in whom decreased muscle mass

contributes to underestimation of obesity-related risk by BMI alone, and in some ethnic groups genetically predisposed to unfavorable distribution of fat despite normal body weight (NIH and NHLBI, 1998). Once patients begin treatment for obesity, waist circumference can show an improvement in body-fat distribution, implying a lower health risk even when BMI does not change. In comparison with measurement of the BMI and waist circumference, history and physical examination constitute the more time-consuming component of an obesity evaluation. In an initial and potentially time-limited assessment of obesity, the goal of which is to identify patients who should be treated for obesity, clinicians should ascertain the smoking history and family history of coronary artery disease. These are the two historical factors included in the Practical Guide's algorithm for medical risk assessment that will enable clinicians to identify a patient at increased medical risk; however, a comprehensive history and physical examination are essential. The history should address trends in the patient's weight over his or her lifetime; risk factors for obesity, such as family history; and diet and exercise habits. As appropriate, the patient should be screened for medical conditions established to contribute to obesity, with confirmatory laboratory studies sent if indicated. The current medication list should be reviewed. The patient also should be evaluated for complications of obesity. It may be helpful to review alcohol consumption habits, because alcoholism causes hypercortisolism and a central obesity syndrome similar to Cushing's syndrome (Fitzgerald, 2000). The pattern of weight gain and loss since puberty is important to ascertain when developing a patient's treatment plan. Evidence does not support an association between persistent metabolic abnormalities and a history of weight cycling, nor should weight cycling be considered a contraindication to obesity treatment (Weinsier and Kushner, 1995; Hirsch *et al.*, 2001). Patients with binge-eating disorder, characterized by eating large amounts of food over a short time, should be identified, because a

disproportionate percentage of these patients have psychiatric disorders that may respond to treatment (NIH and NHLBI, 1998). In such patients, and in others with suspected eating disorders, normalization of eating patterns should take precedence over weight loss (Collazo-Clavell, 1999). Because obese patients are rarely able to attain a weight lower than their minimum adult weight, this value also should be determined and used to guide treatment goals. Finally, diet history can identify patients who, despite a normal weight, have either recently gained or currently are gaining weight. Weight gain itself carries an increased risk of morbidity and mortality from obesity related diseases. For example, one study found a doubling of risk of developing type 2 diabetes with weight gain of 5 to 8 kg and a near-quadrupling of risk with a 22-kg weight gain (hazard ratio, 2.11 and 3.85, respectively) (Ford *et al.*, 1997).

Other studies have found increased risks of CHD with weight gain. Weight gain in early adulthood may predict an increased risk of CHD occurring much later in life. For example, among a cohort of Japanese-American men, weight gain of 5.1 to 10 kg after age 25 carried a relative risk of nonfatal myocardial infarction of 1.60 when compared with men who gained 2.5 kg or less. A gain of more than 10 kg was associated with a relative risk of 1.75 (Galanis *et al.*, 1998). For women as well, weight gain at any time in adulthood has been found to be a strong predictor of CHD later in life. Among a large cohort of women, CHD risk was studied in women with weight gain after age 18 compared with those with stable weight (5 kg). Relative risk was determined to be 1.64 for weight gain of 8 to 10.9 kg, 1.92 for weight gain of 11 to 19 kg, and 2.65 for weight gain of 20 kg or more (Wille *et al.*, 1995).

2.2.3 Assessment of the Obese Patients

One of the goals of assessment in an obese patient is to decide whom to treat. Three main issues must be considered:

1) Whether treatment is indicated,

2) Whether treatment is safe for the patient, and

3) Whether the patient is ready and motivated to lose weight.

This algorithm is a simplified version of the algorithm presented in the Practical Guide. The algorithm takes into account the BMI; waist circumference; and a finite group of risk factors, including cigarette smoking, hypertension, elevated LDL cholesterol, low levels of HDL-cholesterol, impaired fasting glucose, family history of coronary heart disease, and age. Of note, patients with a BMI of 30 kg/m^2 or greater should be considered for treatment regardless of waist circumference or risk factors. Treatment may be indicated for patients of normal weight if they have a waist circumference 35 inches for women or 40 inches for men, as well as two or more of the listed risk factors. The recommendations should not be implemented without first considering their applicability to an individual patient. In muscular patients and in edematous patients, for example, BMI can overstate medical risk by overestimating body fat. Because muscle mass declines with age, BMI can understate risk in the elderly. The relationship between BMI and body fat can vary with ethnicity and gender (NIH, NHLBI and NAASO, 2000), although including waist circumference as a parameter in risk assessment may help compensate for associated differences in fat distribution. Weight-loss therapy is contraindicated in some patients (NHLBI, 1998). In most obese pregnant or lactating women, weight maintenance, but not weight loss, may be recommended. Treatment of active psychiatric disorders, including most eating

disorders and forms of substance abuse, takes precedence over weight loss. Weight loss should not be recommended for patients with acute illnesses, or in the terminal stages of illness, such as cancer or in serious medical conditions, which might be exacerbated by caloric restriction (NIH and NHLBI 1998; Aronne, 2002).

Some studies have found health benefits associated with low-intensity exercise even when cardiovascular fitness has not increased. These benefits include an increase in HDL and a decrease in all-cause mortality (Blair *et al.*, 1989). Patients at low to moderate risk who are not ready to lose weight should be urged to maintain their current weight. A healthy lifestyle should be encouraged, and any complications of obesity should be managed appropriately. Patients at high risk should be educated about the benefits of weight loss, with the goal of motivating them while maintaining a therapeutic alliance. For patients who are ready to lose weight, reasonable goals for diet and physical activity should be set. For most patients, weight-loss goals should initially be 5% to 10% of current weight, because this degree of weight loss is reasonable and results in health benefits. Planned diets should provide 1000 to 1200 kcal/d. With very-low-calorie diets, muscle mass is lost as well as fat and body water. The clinician should draw on obesity-related resources, such as the Practical Guide and the LEARN Program, in evaluating and treating patients. Other professionals, such as dietitians and mental health practitioners, may need to be involved to help the patient lose weight safely and successfully. Finally a supportive empathetic approach should be taken to maintain an optimal therapeutic alliance.

2.2.4 Global Causes of Obesity and Consequences.

In all the cases of obesity worldwide, there is no single cause which can be used to explain it. Environmental factors that have association with obesity include socioeconomic status, region of

residence, ethnicity, season, and urbanization (Portela *et al*., 2015). Although, obesity ultimately results from an imbalance between energy intake and energy expenditure (Rosembaum and Leibel, 1998), genetic predisposition can be a determinant for weight gain. Furthermore, several findings have shown that genetic predisposition does not automatically lead to the development of obesity, because eating habits and physical activity patterns may play a significant role in weight gained (Racette *et al*., 2005). Additionally, a sedentary lifestyle as well as psychological factors such as depression, low esteem, or absence of night sleep can also largely contribute to weight gain (Mirowsky and Rose, 2003). Although the exact cause of weight gain remains to be clarified and likely arises from a complex combination of factors such as genetic factors that greatly affect the manner in which the body regulates the appetite, and the rate of metabolism (Guyenet and Schwart, 2012), excessive weight is clearly gained by consuming an excess of calories as compared to those utilized by the body, with the excess of calories being stored as fat tissue (Rosembaum and Leibel, 1998).

The quantity of fat in a person's diet may have greater impact on weight than the number of calories it contains. The majority of fat calories are immediately stored in fat cells, which add to the body's weight and girth as they expand and multiply (Life, 2014) while carbohydrates such as cereals, breads, fruits, and vegetable and proteins are converted to fuel almost immediately following consumption (Gaman and Sherrington, 2013). Additionally, fat regimens result in excessive and abnormally higher levels of cholesterol in the blood (hypercholesterolemia) (Kwok *et al.,* 2010). Depending on the balance between the fractions of saturated and unsaturated fatty acids, fat contained in the blood circulation can immediately affect certain organs such as liver and kidney, concomitantly with devastating local actions inside of the vessels through the formation of atherosclerosis (Ledwozyw *et al.,* 1986), an infringement of

medium and large arteries due to a buildup of fat inside of the arterial wall, termed visceral fat. The latter is mainly involved in metabolic syndrome (Matsuzawa, 2005). Another detrimental effect of high caloric intake is the increase of norepinephrine turnover in peripheral tissues, raising the resting plasma norepinephrine concentration, which is an indirect measurement of Systematic nervous system activity, and amplifying the increase of plasma norepinephrine in response to stimuli such as upright posture (Landsberg and Krieger, 1989). Thus, high dietary content in fat and carbohydrate has been suggested to acutely stimulate peripheral α1 and β-adrenergic receptors thereby leading to the elevation of sympathetic activity and hypertension (Rocchini *et al.,* 2004). Up-regulated hypothalamic tyrosine hydroxylase and hypothalamic adrenoceptor gene expression of the α2B receptor have been identified, in obese hypertensive rat (Coatmellec-Taglioni and Ribière, 2003). Similarly, in human, combined α and β adrenergic blockade significantly reduced blood pressure in obese relative to lean patients with essential hypertension (Wofford *et al.,* 1998), although elevated heart rate seems to be the effect of decreased parasympathetic activity (Hall and Louis, 1994). Elevated levels and abnormally distributed free fatty acids were reported in obese hypertensive in which they enhanced vascular α-adrenergic sensitivity and consequently the increase of α-adrenergic tone (Stepniakwoski,*et al.,* 1995). Although the mechanisms were endogenous, the effect exerted by free-fattyacid remains to be investigated. At present, it has been documented that free fatty acids inhibit Sodium(Na^+), Potassium (K^+) ATPase and the sodium pump raising vascular smooth muscle tone and resistance (Oishi *et al*., 1990; Bhurosy and Jeewon, 2014).

2.2.5 Socioeconomic Status and Obesity in Developing Countries

Since the early 1980s, economic globalization in developing countries has driven changes in dietary patterns and food choices. Since food choice is mainly dictated by its price in the

developing world, eliciting the influences of socioeconomic variables on food choice may be useful in explaining food behavior. Rapidly growing, developing, or transitional economies face the globalization of food markets, fast food chains, and the increasing availability of street vendors, who offer products at very competitive value due to economical acquisition of inputs such as raw and processed foods (Witkowski, 2007). Differences in diet quality arise due to more frequent consumption of fresh and better quality produce such as fresh fruits, vegetables, and fish among higher socioeconomic status (SES) individuals since fresh produce items are charged higher in grocery and convenience stores (Dunn *et al*., 2011). In particular, the poorer segments are often left to opt for energy-dense diets, rich in cheap vegetable oils, and trans-fats (Mclaren, 2007). For example, in countries of the Middle-East, Asia, and Africa, edible oil consumption has risen very rapidly (Dunn *et al*., 2011). In addition, the price per weight of food items is an important determinant of food choice. Low fat protein sources, for example, poultry and pulses, which cost less per weight, are the preferred choices of low SES participants (Xiao *et al*., 2013).

In every country worldwide, whether transitional economies or developed ones, non-communicable chronic conditions like obesity are either on the rise or have already reached alarming levels (Prentice, 2006). While low socioeconomic status (SES) has been associated with a higher prevalence of obesity and chronic diseases in developed countries, previous studies, in developing nations, have shown a positive SES-obesity relationship (Kumanyika *et. al*., 2002). The SES-obesity relationship in developing countries has been reported to bear similarities to that in developed ones. Delavari *et al.*(2013) predicted that the obesity pandemic will be unabated in the near future and low-income and lower-income countries will face the current trends of obesity observed in the upper-middle and high income countries in the coming years. In fact, education is the socioeconomic indicator which has been reported to be the most

significant predictor of diet quality (Delavari *et al.,* 2013). In Mauritius, a study conducted among young and middle-aged women (Bhurosy and Jeewon, 2013) demonstrated that educational level was the only factor significantly associated with diet quality. Educational disparities reflect educational differences pertaining to dietary knowledge, food purchasing behavior, and perceptions of healthy food items.

2.2.6 Physical Activity Level and Its Impact on Obesity: Current Scenario in Developing Countries

Low physical activity level (PAL) accounts for 6% of deaths worldwide and inadequate PAL, especially, concerns populations of low SES (Monsivais and Drewnowski, 2009). Reductions in PAL, over the past years, are linked to several factors: less energy expenditure activities such as farming and forestry, a rise in sporadic activities such as sitting in front of a computer terminal, and patterns of low activity during leisure hours (Bergier *et al.,* 2012). As such, concurrent marked reductions in PAL have been reported within every occupation (Dunn *et al*., 2011). In African regions, for example the epidemic of obesity, at least, can in part be explained by decreased levels of physical activity as in the late 1980s; roads were tarred with taxis and buses becoming the most common transport means and, in addition, there was an ongoing trend away from manual labor to less physically strenuous jobs and the shift to less nutrient-dense diets (Samuel and Atinmo, 2008). Use of screen time has also been associated with other equally unhealthy behaviors such as eating palatable fatty foods (Temple *et al*., 2001). Watching television is linked to high cholesterol levels and unhealthy diets and which is also influenced by unhealthy nutrition messages in commercials (Banks *et. al*., 2011). In addition, low PAL is amplified by inadequate community designs and infrastructure characteristics such as lack of safe walkways, bicycle paths, and playgrounds (Turconi*et al.*,,2008). Though low socioeconomic

status and low educational level in developing countries are associated to low PAL due to, primarily, a worse access to sports facilities, PAL is twice as much among rural residents than urban ones due to higher household activities which compensate for low PAL during free time (Monsivais *et al*., 2009). Since data on PAL is scarce and fragmented and is mostly based upon self-reports, information gathered on PAL in developing nations may be subjected to bias and the use of pedometers and other monitoring technologies is not yet widespread, even in developed countries (Raj and Kumar, 2010).

2.3 RENIN ANGIOTENSIN SYSTEM (RAS)

The renin-angiotensin system is important for the regulation of vascular smooth muscle tone, fluid and electrolyte balance, and the growth of cardiac and vascular smooth muscle. A normally functioning renin-angiotensin system contributes to the routine control of arterial blood pressure (Mizuiri and Ohashi, 2015). A variety of basic and clinical investigations have resulted in a broader understanding of the role of the RAS in the cardiovascular pathophysiology of hypertension, congestive heart failure, and more recently, atherosclerosis. Whether or not abnormal activity of the RAS contributes to the primary etiology of these diseases, pharmacological inhibition of the RAS has proved to be a valuable therapeutic strategy in the treatment of hypertension and congestive heart failure (Ferrario, 2006). The classical RAS comprises a series of biochemical steps leading to the production of a family of structurally related peptides (e.g. angiotensin II, angiotensin III and other smaller peptides with bio activity). Sites for pharmacological intervention in this system include the enzymatic steps catalyzed by renin, angiotensin-converting enzyme (ACE), and angiotensin receptors that mediate a particular physiological response (Mizuiri and Ohashi, 2015).

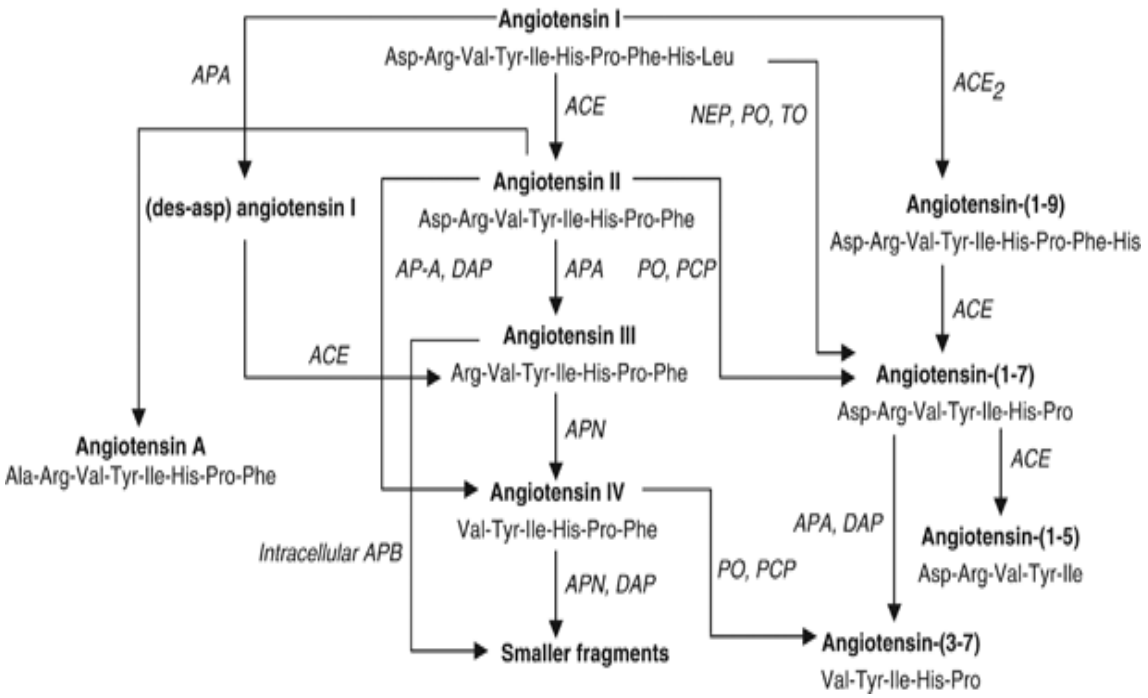


Figure 2: Synthesis and structure of Renin-Angiotensin system (RAS) components

The renin-angiotensin (ANG) system (RAS) is important in blood pressure control, and various findings from experimental animals and humans suggest activation of the RAS with obesity and hypertension(Pinterova *et al*., 2000). Several evidence supports the existence of tissue RAS, important in the local production of ANG II (Engeli *et al*., 2000). It was previously demonstrated that rat brown and white adipose tissues exhibit a high level mRNA expression of angiotensinogen, the only known precursor to ANG II (Cassis *et al.*, 1988). Further studies demonstrated that rat and human adipose tissue possess all of the components necessary for production of ANG II, including angiotensinogen (Engeli *et al*., 1999), activity renin-like (Shenoy and Cassis 1997), ANG-converting enzyme (ACE) (Pinterova *et al*., 2000), and the ANG type 1 (AT1) receptor (Engeli *et al*., 2000). Importantly, nutritional regulation of the angiotensinogen gene in adipose tissue has been shown, with reductions with starvation and elevations in obesity (Touyz and schiffrin, 1999). Previous studies demonstrate that over-

expression of the angiotensinogen gene selectively in adipose tissue of mice results in elevated plasma angiotensinogen and modest hypertension (Massiera *et al.*, 2001). Thus alterations in adipose derived angiotensinogen have the ability to impact the systemic RAS and influence blood pressure.

An important new finding showed that circulating concentrations of ANG peptides were markedly increased in rats with diet-induced obesity. Moreover, plasma concentrations of ANG II correlated positively to Mean Arterial Pressure, demonstrating a strong link between the RAS and obesity-induced hypertension. In addition, elevations in angiotensinogen mRNA expression were observed in selective adipose depots, but not in the liver, of obesity-prone rats. Adipose angiotensinogen mRNA expression was mirrored by a similar-magnitude increase in the circulating angiotensinogen concentration. In contrast, other components of the RAS were not altered in obesity-prone rats compared with control rats. These observations support a role for the RAS in hypertension from obesity and suggest that adipose tissue contributes to heightened levels of the systemic RAS. The obesity aspect of the diet-induced obesity model initially created by Levin *et al*. (2004) using a diet containing a moderate increase in fat (32% kcal as fat), similar to the Western diet (Sclafani, 1989), The obesity development in obesity-prone rats was characterized by monitoring food intake and body weight throughout the duration on the diet (Sclafani, 1989). As originally described by Levin *et al.* (2004), rats fed the moderately high fat diet segregate into obesity-prone and obesity-resistant rats with considerable differences in body weight gain and adiposity. Similar to other findings in this model, energy intake was increased in Obesity-Prone rats (Dobrian *et al*., 2001). However, the efficiency of weight gain (total energy intake/ body weight gain) was increased in obesity-prone rats, suggesting that elevations in food intake are not the sole mechanism for increased body weights in obesity-prone rats (Levin *et al*.,

1997). Therefore these results concur with the hypothesis of preexisting metabolic differences between obesity-prone and obesity-resistant Sprague-Dawley rats (Levin *et al.*, 2004). Angiotensinogen mRNA expression was increased selectively in the retroperitoneal adipose tissue of obesity-prone rats. It is unlikely that increased angiotensinogen mRNA expression in adipose tissue results directly from the high-fat diet, because the study obesity-resistant rats exhibiting normal adiposity in response to the moderately high fat diet did not exhibit these changes (Rahmouni *et al.,* 2005). Rather, the results suggest that elevations in angiotensinogen mRNA expression are associated with obesity.

2.3.1 Angiotensin II Peptides

Ang II is an octapeptide produced from the substrate angiotensinogen through sequential enzymatic cleavages by renin and angiotensin converting enzyme (ACE). Specifically, renin cleaves angiotensinogen, forming Ang I that in turn is converted to Ang II by ACE. The angiotensinogen substrate is produced in the liver, while renin is produced in the kidney and Ang II in the vascular tissue (Timmermans *et al.,* 1993). ACE is a circulating enzyme that also degrades bradykinin to inactive fragments, reducing the serum levels of endogenous vasodilators (Fleming*et al.,*2013). The genetic analysis of ACE has revealed an important insertion (I)/deletion (D) polymorphism, a 287 bp DNA sequence in the intron 16 of chromosome 17 in the ACE gene, a major locus that accounts for approximately 50% of the total phenotypic variance of circulating and tissue ACE (Rigat *et al.,* 1990). The ACE I/D polymorphism is a reliable tool to identify patients at risk of renal disease progression and to elect those who may benefit the most from treatment with Ang II blockers (Ruggenenti *et al.,*2008). It is conceivable that the same applies to patients with cardiovascular disease or progressive deterioration of brain

function including senile dementia and Alzheimer, but studies of this kind are not available at the moment.

ACE2 is another carboxypeptidase that cleaves one amino acid from Ang II leading to the production of the heptapeptide vasodilatory Ang 1–7 (Crackower *et al.*, 2002; Ferrario and Chappell, 2004) and the balance between ACE and ACE2 is crucial for controlling Ang II levels (Danilczyk and Penninger, 2006). Ang II levels can also be regulated by chymase, an enzyme expressed in the heart by mast cells, endothelial and mesenchymal interstitial cells (Urata *et al.*, 1993) and in the kidney by mesangial and vascular smooth muscle cells (Huang *et al.*,2003). Chymase-mediated Ang II production has emerged as an alternative pathway to ACE in cardiac, vascular and renal tissue, particularly in disease conditions (Huang *et al.*, 2003; Bacani and Frishman, 2006; Miyazaki and Takai, 2006). Finally, Ang II can also be cleaved in the circulation by other amino-peptidases generating Ang-(2–8) (Ang III) and Ang-(3–8) (Ang IV). Ang III has similar effects to Ang II, although with lower potency, of enhancing blood pressure and vasopressin release (Cesari *et al.*, 2002; Reaux *et al.*, 2001) and stimulating the expression of pro-inflammatory mediators in cultured renal cells (Ruiz-Ortega *et al.*, 2000). Ang IV exerts a protective role by increasing blood flow in the kidney (Hamilton *et al.*, 2001) and brain (Kramar *et al.*, 1997). Circulating Ang II contributes to increased blood pressure and influences renal tubuli to retain sodium and water (Brewster and Perazella, 2004; Kim *et al.*,2003). Ang II promotes reactive oxygen species (ROS) production, cell growth, apoptosis, cell migration and differentiation, extracellular matrix re-modeling, regulates gene expression and can activate multiple intracellular signaling pathways leading to tissue injury (Ruster and Wolf, 2006). In tissues such as kidney, heart and vasculature, Ang II induces an inflammatory response by fostering the expression of pro-inflammatory chemokines, responsible for tissue accumulation of

immunocompetent cells (Suzuki *et al.*,2003). In hypertension, a noxious amplification mechanism occurs in the kidney, in which Ang II induces renal angiotensinogen expression and thereby its own synthesis (Kobori *et al.*, 2001).

2.3.2 Angiotensin II Receptors

Angiotensin II acts through two pharmacologically distinct G protein-coupled receptors, angiotensin type 1 and the type 2 (AT_1 and AT_2) receptors (Hunyady and Catt, 2006; Porrello *et al.*, 2009). Human cells express a single AT_1 receptor, while two isoforms, AT_{1A} and AT_{1B} with 95% of amino acid sequence identity, can be found in rat and mouse. The AT_{1A} receptor, the closest murine homologue to the human AT_1 receptor, is expressed in the kidney, heart, brain adrenal gland, vascular smooth muscle, liver and several other tissues (Burson *et al.*, 1994). AT_{1B} on the other hand is predominantly expressed in anterior pituitary gland and adrenal zona glomerulosa (Oliverio and Coffman, 2000). AT_{1A} confers most of classical actions of Ang II such as blood pressure increase (Ito *et al.*, 1995), aldosterone release from the adrenal zona glomerulosa (Aguilera, 1992), salt retention in proximal tubular cells (Thekkumkara *et al.*, 1998) and stimulation of the sympathetic nervous system via receptors in the brain (Davisson *et al.*,2000). AT_{1B} regulates blood pressure when AT_{1A} receptor is absent (Oliverio and Coffman, 2000). Angiotensin type II receptor is ubiquitously expressed in developing foetal tissues; it decreases after birth remaining low in various adult tissues including adrenal medulla, uterus, ovary, vascular endothelium and distinct brain areas (Steckelings *et al.*, 2005). AT_1 and AT_2 receptor have counter-regulatory actions in the cardiovascular and renal system [reviewed by Schulman and Raij, (2008)]. Ang II binding to the AT_2 receptor induces vasodilation in resistance and conduit arteries and improves artery remodeling in humans and mice. The AT_2 receptor is up-regulated in conditions associated with cardiovascular injury and it exerts a cardio-

protective role against ischemia-reperfusion injury and acute myocardial infarction (Schulman and Raij, 2008).

2.3.3 Angiotensin II Type 1 Receptor Gene

The ATR1 gene, localized on chromosome 3q21-q25, encodes type 1 angiotensin II receptor, a dominant variant of the receptor in the cardiovascular system. The A1166C SNP, localized within the haplotype block in the 3-untranslated region of AGTR1 gene, may be involved in posttranscriptional modification and angiotensin II receptor-mediated cell signaling, which is critical for regulating tissue-specific receptor functions (Sounira *et al*., 2012). Several reports suggest the existence of a functional renin angiotensin system in WAT. A previous study by Laurent *et al*. (2005) speculate that Ang II acts locally to control adipose tissue mass., and it has been demonstrated (Laurent *et al*., 2005) that transgenic mice with over expression of AGT in the adipose tissue exhibited an increased fat mass due to adipocyte hypertrophy. However, In-vitro studies indicate a stimulatory effect of Ang II on lipogenesis in 3T3-L1 and human adipocytes (Agachan *et al*., 2003). Several studies using specific antagonists suggestthat the major vascular functions of Ang II are primarily mediated by the AT1*R* in adipose cells. Recent In-vitro studies have implicated AT2*R* in the lipogenic effect of Ang II (Agachan *et al*., 2003). Inoue*et al*.(1997), based on their In-vitro evidence, suggest that Ang II could have a trophic role in WAT, favoring both hypertrophy and hyperplasia of adipocytes.

2.4SERUM LIPID

Lipid profile or lipid panel is a panel of blood tests that serves as an initial screening tool for abnormalities in lipids, such as cholesterol and triglycerides. The results of this test can identify certain genetic diseases and can determine approximate risks for cardiovascular disease, certain forms of pancreatitis, and other diseases. Lipid panels are ordered as part of a physical

examination, along with other panels such as the complete blood count (CBC) and basic metabolic panel (BMP)

2.4.1 Components of Lipid Profile

The lipid profile typically includes: low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides and total cholesterol. Using these values, a laboratory may also calculate: Very low-density lipoprotein (VLDL), Cholesterol: HDL ratio

The lipid profile tests are of 7 types: Total lipids, Serum total cholesterol, serum HDL cholesterol, total cholesterol/HDL cholesterol ratio, serum triglycerides, serum phospholipids,

electrophoretic fractionation to determine the percentage of Chylomicrons, LDL, VLDL, HDL.

2.4.2Procedure and Indications

Current recommendations for cholesterol testing come from the Adult Treatment Panel (ATP) III guidelines, and are based on many large clinical studies, such as the Framingham Heart Study. For healthy adults with no cardiovascular risk factors, the ATP III guidelines recommend screening once every five years. (NCEP 2002) A lipid profile may also be ordered at regular intervals to evaluate the success of lipid-lowering drugs such as statins.

In the pediatric and adolescent population, lipid testing is not routinely performed. However, the American Academy of Pediatrics and NHLBI now recommend that children aged 9–11 be screened once for severe cholesterol abnormalities. (NHLBI and NIH 2012) This screening can be valuable to detect genetic diseases such as familial hypercholesterolemia that can be lethal if not treated early.

Traditionally, most laboratories have required patients to fast for 9–12 hours before screening. However, studies have questioned the utility of fasting before lipid panels, and some diagnostic labs now routinely accept non-fasting samples. (Sidhu and Naugler, 2012). Typically the laboratory measures only three quantities: total cholesterol; HDL; Triglycerides. From these three data LDL may be calculated. According to Friedewald's equation: (Friedewald *et al.,* 1972)

LDL = Total cholesterol - HDL - Triglycerides/5

Other calculations of LDL from those same three data have been proposed which yield some significantly different results. (Friedewald *et al.,* 1972)

VLDL may be defined as the total cholesterol that is neither HDL nor LDL. Then Friedewald's equation mentioned above yields:

VLDL = Triglycerides/5

The alternative calculations mentioned above may yield significantly different values for VLDL.

2.4.3 Implications

This test is used to identify dyslipidemia (various disturbances of cholesterol and triglyceride levels), many forms of which are recognized risk factors for cardiovascular disease and rarely pancreatitis. A total cholesterol reading can be used to assess an individual's risk for heart disease; however, it should not be relied upon as the only indicator. The individual components that make up total cholesterol reading—LDL, HDL, and VLDL—are also important in measuring risk. (Friedewald *et al.,* 1972)

For instance, someone's total cholesterol may be high, but this may be due to very high HDL ("good cholesterol") cholesterol levels,—which can actually help prevent heart disease (the test is mainly concerned with high LDL, or "bad cholesterol" levels). So, while a high total cholesterol level may help give an indication that there is a problem with cholesterol levels, the components that make up total cholesterol should also be measured. (Friedewald *et al.*, 1972)

Lipids and lipoproteins are risk factors for CHD. It has been demonstrated that high levels of serum total cholesterol (TC), triglycerides (TG), LDL cholesterol, very-low-density lipoprotein (VLDL), low concentration of HDL cholesterol, and increased body mass index (BMI) are significantly associated with CHD (George and Ludvik, 2000). Dyslipidemia is one of the top five major risk factors leading to cardiovascular disorders. It is characterized by elevated LDL cholesterol and TG and decreased HDL cholesterol. Although there are differences in defining dyslipidemia, however, European guideline on CVD prevention in clinical practice recommends TC below 190 mg/dL (5.0 mmol/L) and an LDL cholesterol below 115 mg/dL (3.0 mmol/L) for the general population. The goals are even lower: i.e., <175 mg/dL (4.5 mmol/L) for TC and <100 mg/dL (2.6 mmol/L) for LDL cholesterol in the case of multiple disorders like CHD, other diseases of CVD, or DM (De Backer *et al.* 2003).

Lipid abnormalities significantly contribute to the increased risk of cardiovascular disease and other morbidity in diabetics. VLDL and chylomicrons (CM) are major sources of fatty acid supply to the heart, but little is known about their metabolism in diabetic myocardium. Males and females appear to be equally susceptible to the effects of risk factors such as hypertension, increased plasma LDL cholesterol, and low levels of plasma HDL cholesterol. Estrogens have a favorable effect on lipid profile. It has been observed that they lower LDL cholesterol and

elevate HDL cholesterol. Estrogens are thought to increase HDL cholesterol by reducing hepatic triglycerides' lipase activity that catabolizes HDL cholesterol. Global studies of either gender have demonstrated that the risk of atherosclerosis is inversely related to blood levels of HDL cholesterol: the higher the level of HDL cholesterol, the lower will be the risk. It is indicated that for every 1 mg/dL rise in HDL cholesterol, the risk for developing cardiovascular disease decreases by 2–3 % (Toth 2005).

HDL cholesterol helps to extract excess cholesterol deposited in blood vessel walls and deliver it back to the liver for elimination through the gastrointestinal tract. HDL cholesterol helps to keep blood vessels dilated, thereby promoting better blood flow. It also reduces blood vessel injury through its antioxidant and anti-inflammatory functions, among other effects. HDL cholesterol carries "old" cholesterol that has been discarded by cells back to the liver for recycling or excretion. The main function of LDL cholesterol is to transport cholesterol from the liver to the tissues that incorporate it into the cell membranes. The oxidation of LDL cholesterol is believed to have a central role in atherogenesis. Oxidized LDL cholesterol may be involved in atherogenesis by inducing smooth muscle cell proliferation. Acute MI is the most important consequence of coronary artery disease. Some studies have defined TG also as an independent risk factor for Myocardial Infarction (Haffner *et al.* 1998).

High TG value could result from the elevation of several lipoproteins such as chylomicrons, different subclasses of VLDL, or intermediate-density lipoproteins (IDL cholesterol). The risk of MI in patients with DM without a history of myocardial infarction is as high as that in patients without MI who have had a myocardial infarction. Mortality after first MI is higher in both males and females with DM. Lipid abnormalities significantly contribute to the increased risk of CVD

in diabetes mellitus. Diabetes affects virtually all lipids and lipoproteins. Persons with DM typically have increased plasma concentrations of TG, low plasma concentrations of HDL cholesterol, and slightly raised plasma concentrations of LDL cholesterol. DM is also considered as an independent risk factor for cardiovascular disease (up to fivefold), and as many as 80 % of patients with type II diabetes die from cardiovascular complications (Johnson *et al.,* 2004). Persons with high blood cholesterol levels have a higher prevalence of hypertension, and those with high blood pressure have a higher prevalence of hypercholesterolemia (O'Brien *et al.,* 2003). Abnormalities in plasma lipoprotein metabolism play a central role in the pathogenesis of atherosclerosis, and arterial hypertension with elevated systolic or diastolic blood pressure is positively and independently associated with CHD. The risk of developing CVD associated with the presence of both hypertension and Dyslipidemia has been shown to be greater as compared to hypertension or Dyslipidemia alone (Johnson *et al.* 2004).

Moreover, patients with these two conditions found to have three to four times higher prevalence of MI (Wald and Law, 2003). Dyslipidemia causes endothelial damage and consequent loss of physiological vasomotor activity, which may be manifested as increased blood pressure. Asians experience the largest proportion of the worldwide burden of CVD. Further, Asians include several distinct ethnic subpopulations (South Asians, Chinese, etc.), who may differ in their lipid profiles. These differences may be the result of both genetic and environmental factors such as high-cholesterol carbohydrate diets, reduced physical activity, and obesity (Radhika *et al.,* 2009).

2.5 DIETARY ASSESSMENT METHODS

Dietary assessment methods are developed to acquire suitable data on dietary intakes, needed to promote health (Castell *et al*., 2015). Dietary assessment includes food production, supply, and purchases at households and food intakes by individuals (Coulston *et al*., 2013). The 24-hour dietary recall, dietary records and food frequency questionnaire are some usual subjective tools for dietary assessment used to collect data on food intakes of individuals (Shim *et al*., 2014).

2.5.1 Dietary Record

The individual provides written records of food and beverages, and portion sizes of each consumed food for one or more days during time of eating. The quantity of food eaten is measured with food scales or food models. Dietary record provides list on food name, cooking methods, recipes for food preparation and portion sizes. As such, dietary record provides data on food consumed during a specific period (Thompson and Subar, 2001).

2.5.2 Food Frequency Questionnaire (FFQ)

This assesses habitual food intake of selected list of foods, in terms of frequency and quantity of consumption in a specified period. The FFQ mostly focuses on dietary intake of defined nutrients which is associated with specific diseases. Also, FFQ establishes how dietary patterns relate to low intake of specified nutrients among a population (Rodrigo *et al*., 2015). Food frequency questionnaire has an advantage of providing usual dietary intake over a long period (Yanagisawa *et al*., 2016). Conversely, FFQ does not provide detailed, and accurate reflection on habitual diets of respondents, although, frequency and quantity of foods are measured (Rodrigo *et al*., 2015).

2.5.3 Twenty-Four-Hour Dietary Recall

This provides retrospective assessment of food and beverages consumed in the past day, beginning from morning to evening. The 24-hour food recall is characterized by providing information on precise description, portion sizes, food preparation methods, and where food is eaten. This dietary assessment tool provides both precise and valid data on energy and nutrients of individuals. However, it is limited by the over-reliance on memory of respondent during recalling and also has tendency to under or overestimating nutrients intake (Castell *et al*., 2015).

2.5.4 Definition of Anthropometric and Biochemical Data

2.3.4.1 Body Mass Index (BMI)

Body mass index is the ratio of weight (kg) to height squared (m^2). This is usually adopted to categorize people as underweight, normal, overweight and obesity in adults. BMI values are same for both sexes (NHMRC, 2013). BMI can help predict future health outcome and functional status of individuals (Grzegorzewska *et al*., 2016).

However, the use of BMI is limited to factors such as distribution of lean mass and body fat (Zhu *et al*., 2014), age variation, sex and ethnicity of individuals (Grzegorzewska *et al*., 2016). Globally, being obese is a predisposing factor to increased possibility of type 2 diabetes and cardiovascular diseases (Heiss and Goldberg, 2016).

CHAPTER THREE

MATERIALS AND METHODS

3.1 MATERIALS

3.1.1 Target Population

The study was an observational, cross-sectional study. Hypertensive subjects were selected by simple random probability sampling method from patients (males and females) attending the hypertensive clinic of some selected hospitals in Kano metropolis (Murtala Muhammad Specialist Hospital, Sheik Muhammad Jidda, Sir Muhammad Sunusi General Hospital,Muhammad Abdullahi Wase Specialist Hospital), Kano State, Nigeria. The study subjects comprised of hypertensive patients and apparently healthy subjects between the ages of 20 and 70 years, whose weight fall into normal, obese or overweight groups.

3.1.2 Inclusion and Exclusion Criteria

Patients studied werethose diagnosedas hypertensive. The hypertensive status of the patients was defined as described by the United States Seventh Joint National Committee on Detection, Evaluation and Treatment of Hypertension (JNC-VII), that is, Systolic Blood Pressure (SBP) greater than or equal to 140 mmHg and Diastolic Blood Pressure (DBP) greater than or equal to 90 mmHg. Women receiving oral contraceptives or hormone replacement therapy, pregnant women and lactating mothers, as well as those that refused to give their consent were excluded from the study.

3.1.3 Sample Size Determination

Sample size was determined in accordance with Fisher's formula, (Babandi*et al.*, 2017), with the following resource equation

$n = Z^2pq/d^2$

Where;

n = minimum sample size required in a population

Z = standard normal deviation usually at 1.96 which corresponds to 95% confidence interval

P =Hypertension prevalence rate in a previous study 32.8% (Adeloye*et al.*, 2015).

q = proportion of failure = (1- p)

d = degree of accuracy which is chosen as 5 % (0.05)

$$n = \frac{Z^2 pq}{d^2} \qquad \frac{(1.96)^2 \times 0.328 \times (1-0.328)}{0.05 \times 0.05} = 154$$ will be the minimum sample

Therefore, 200 participants were recruited for the whole study.

3.1.4 Ethical Approval

Ethical approval was obtained from Kano State Hospitals Management Board (HMB) (MOH/Off/797/T.I/1649). Participants were duly informed about the research and gave informed consent.

3.1.5 Administration of Questionnaires

A structured questionnaire was used to collect data on subjects socioeconomic and demographic characteristics, including nutritional status, food frequency questionnaire, knowledge, attitude and practices.

3.1.6Equipmentand Apparatus

Body composition monitor (karada scan omron BF 511, Zhengzhou, China),sphygmomanometer (ZP-800SI, Bojin, Shijianzhuang, China),Centrifuge (Biobase 16800RPM, Shandong, China), Refrigerator (Haier thermocool, HRF-250E, Qingdao, China), High Speed Refrigerated Centrifuge (PM-180R, Shandong, China), Incubator (Multi block heater-Lab line, Bioevopeak, Jinan city, China),Microplate reader (EZ Read 2000, Biochrom, Winooski,USA), Measuring Tape (LCR01, HaB, Warwickshire, UK).Beaker, measuring cylinder, syringe and needle, cotton wool, sample containers, test-tubes, tube racks, Micropipettes, Pipette tips, cuvettes, Questionnaire (KAP, FFQ), Hand Gloves and are products of reputable companies.

3.1.7 Chemicals and Reagents

Methylated Spirit, Lipid Profile Test Strip, ANG II Elisa kit, HHL, Washing buffer, Purification buffer (all were obtained from Bioneer Corporation, United States and were of analytical grade).

3.2 METHODS

3.2.1 Specimen Collection

Venous blood (5ml) was aseptically collected from patients and controls, with the use of syringe after sterilizing the site with methylated spirit. The samples were properly labeled and kept in arefrigerator at 4^0C for biochemical analysis.

3.2.2 Determination of Blood Pressure

Blood Pressure (BP) was measured by trained doctors and nurses using mercury sphygmomanometer in a seated position.

3.2.3 Determination of ACE Activity

The assay for ACE activity was determined using the Cushman and Cheung(1971) method with some modifications. Serum (50µL) was added to 50µL deionized water and the reaction was started by adding 0.2 ml of 5-mmol/L hippuric-histidyl-leucine (HHL). This was incubated at 37°C for 15 minutes and the reaction terminated by adding 0.25 ml of 1.0 N hydrochloric acid. Ethyl acetate (2.0 ml) was then added to extract the hippuric acid formed by the action of ACE. This was centrifuged at 3600xg for 2mins, and 1ml of upper layer was transferred into a microcentrifuge tube and heated by dry bath at 100°C for 15 minutes to remove ethyl acetate by evaporation. The resulting hippuric acid was dissolved in 3.0 ml distilled water, and the absorbance was read at 228 nm.

$$\text{Units/ml enzyme} = \frac{(A_{228nm}\ \text{Test} - A\ A_{228nm}\ \text{Blank})\ (2)\ (3)}{(a)(b)(c)(d)}$$

Where; 2 = Conversion factor since the hippuric acid detected was 1/2 of the total amount produced in the assay: 2mL of ethyl acetate was added and 1mL of the organic layer containing the product, hippuric acid, was removed.

3 = Total volume of hippuric acid solution

a = Millimolar extinction coefficient of hippuric acid at 228 nm (9.8)

b = Time (in minutes) of the assay as per the unit definition (15 minutes)

c = Extraction efficiency of ethyl acetate (0.91), d = Volume (in milliliter) of enzyme used (0.05)

Serum enzyme activity was expressed in units, which corresponded to l mol of hippuric acid released by hydrolysis of HHL per minute per milliliter serum/supernatant.

3.2.4 Determination of ANG II level

Principle

The ELISA kit involves Competitive-ELISA method. The micro titer plate in the kit has been pre-coated with Ang-II. During the reaction, Ang-II in the sample or standard competes with a fixed amount of Ang-II on the solid phase supporter for sites on the biotinylated detection Ab specific to Ang-II. Excess conjugate and unbound sample or standard are washed from the plate, and avidin conjugated to horseradish peroxidase (HRP) is added to each microplate well and incubated. Then a TMB substrate solution is added to each well. The enzyme-substrate reaction is terminated by the addition of a sulphuric acid solution and the color change is measured spectrophotometrically at a wavelength of 450 nm ± 2 nm. The concentration of Ang-II in the

samples is then determined by comparing the OD of the samples to the standard curve.

Procedure

All reagents and samples were maintained at room temperature before the commencement of the experiment. The samples were centrifuged again after thawing before the assay. All the reagents were mixed thoroughly by gentle swirling before pipetting.

Standard (50µl) were added in each well. The blank well was added with reference standard and sample diluent. Immediately biotinylated detection Ab working solution (50 µl) were added to each well and covered with a plate sealer and incubated for 45minutes at 37°C. Solutions were added to the bottom of micro ELISA plate well, to avoid inside wall touching and foaming as possible. Each well was aspirated and washed three times by filling each well with wash buffer (approximately 300µl). After the last wash, remaining wash buffer was removed by decantation. The plate and pat were inverted against thick clean absorbent paper. HRP Conjugate solutions (100µl) were added to each well, covered with a new plate sealer and incubated for 30 minutes at 37°C. The aspiration/wash processes were repeated for five times as conducted in above. Substrate Solution (90µl) were added to each well, covered with a new plate sealer and Incubated for about 15 minutes at 37°C. The reaction time was extended according to the actual color change for 5 minutes. When apparent gradient appeared in standard wells, the reaction can be terminated. Stop solution (50µl) was added to each well. Color turned to yellow immediately. The optical densities (OD value) of each well weredetermined at once, using a microplate reader set at 450nm.

3.2.5 Lipid profile

Lipid panel test system was used to measure lipid parameters. This test system use lipid panel test strips to measure cholesterol, HDL and triglyceride in whole blood/plasma/serum. The LDL cholesterol can then be calculated from the result obtained for other parameters using the formula, LDL = TC – HDL– [Trig/5]. CardioChek Plus professional analyzer provides the quantitative result. A MEMo Chip was provided with each package of test strips which must be properly inserted into the analyzer before any test can be run.

Principle

When blood is applied to a test strip, the blood reacts to produce color that is read by the analyzer using reflectance photometry. The amount of the color produced is proportional to concentration. The enzymatic reactions that occur are listed below (Tiet, 1990).

Cholesterol

Cholesterol esters + H_2O $\xrightarrow{\text{cholesterol esterase}}$ Cholesterol + fatty acid

Cholesterol + H_2O + O_2 $\xrightarrow{\text{cholesterol oxidase}}$ Cholestryl-4-ene-3-one + H_2O_2

$2H_2O_2$ + 4-AAP + Distributed Aniline $\xrightarrow{\text{peroxidase}}$ Quinoneimine dye + 4 H_2O_2

HDL Cholesterol

Principle:

The very low density (VLDL) and low density (LDL) lipoproteins from serum or plasma are precipitated by phosphotungstate in the presence of magnesium ions. After centrifugation the supernatant contain high density lipoprotein (HDL). The HDL cholesterol fraction is determined using the total cholesterol enzymatic reagent(Burstein *et al.*, 1970).

VLDL, LDL, HDL plasma $\longrightarrow$ VLDL, LDL, depleted plasma

Cholesterol ester + H_2O $\xrightarrow{\text{cholesterol esterase}}$ Cholesterol + fatty acid

Cholesterol + H_2O + O_2 $\xrightarrow{\text{cholesterol oxidase}}$ Cholestryl-4-ene-3-one + H_2O_2

$2H_2O_2$ + 4-AAP + Distributed Aniline $\xrightarrow{\text{peroxidase}}$ Quinoneimine dye + 4 H_2O_2

Triglycerides

Triglyceride + $3H_2O_2$ $\xrightarrow{\text{lipoprotein lipase}}$ Glycerol + 3 fatty acid

Glycerol + ATP $\xrightarrow{\text{glycerol kinase}}$ Glycerol-3-PO_4 + ADP

Glycerol-3-PO_4 + O_2 $\xrightarrow{\text{glycero phosphate oxidase}}$ Dihydroxyacetone-PO_4 + H_2O_2

$2H_2O_2$ + 4-AAP+N,N-distributed aniline $\xrightarrow{\text{peroxidase}}$ Quinoneimine dye + H_2O

Procedure

A MEMo chip that matches the lot number on the test strip vial was inserted into the analyzer and the analyzer was turned on. The test strip was held by the end marked PTS and the opposite end of the strip was inserted in to the analyzer. The strip was pushed in to as far as it could go. The command "APPLY SAMPLE" displayed on the screen of the analyzer. A capillary blood collector was used to apply 35-40 µL of serum to the test strip blood application window. When enough sample went into the application window the response "TESTING" displayed on the screen of the analyzer. In as little as 90 seconds the result appeared on the display automatically on the analyzer.

3.2.6 Data Collection

Structured questionnaires (Food Frequency Questionnaire and 24 hours dietary recall) were used to gather data on patients' demographic characteristics, food habits, life style, physical activities and food choices.

3.2.7 Dietary Assessment

A food frequency questionnaire (FFQ) containing a list of 36 common foods was used to assess dietary patterns of adolescents' intake of study participants over one week. The FFQ was designed in such a way to reflect dietary intake of the participants for the whole one week. A 24-hour duplicate recall for three days was used to assess current and usual dietary intakes of study participants (This was done because majority of participants reported consuming same meals daily). Handy measures of food items were used to allow participants to quantify amount of food eaten.

3.2.8 Anthropometric Data Collection

The weight, BMI, Visceral fat, fat composition, muscle composition, Resting metabolism of study participants was measured with Body Composition Monitor.Height without shoes was measured in centimeters, and weight in light clothing was measured in kilograms. Weight circumference (WC) was measured at the level midway between the lower rib margin and the iliac crest with participants in standing position without heavy outer garments and with emptied out pockets, breathing out gently. Hip circumference was recorded as the maximum circumference over the buttocks. BMI was calculated as weight divided by height squared (kg/m2). BMI was classified based on WHO guidelines as follows: underweight was a BMI < 18.50; normal weight was a BMI of 18.5–24.9;overweight was a BMI of 25.0–29.9; obese was a BMI ≥ 30.0.

3.3 STATISTICAL ANALYSIS

Data was analyzed using IBM SPSS advanced statistics version 24.0 (SPSS Inc., Chicago, IL). Numerical data was expressed as mean and standard deviation or median and range as appropriate. Qualitative data was expressed as frequency and percentage. Chi-square test, confidential interval and odd ratio were used to examine the association between qualitative variables at $P < 0.05$.

CHAPTER FOUR

RESULTS AND DISCUSSION

4.0 RESULTS

4.1 Description of the anthropometry and biochemical parameters

Table 2 shows the general description of the anthropometric and biochemical parameters which include the mean and standard deviation, minimum and maximum range.

Table 3 indicates the nutritional attitude of the hypertensive and control individuals attending some selected hospitals in Kano metropolis. Lack of nutritional knowledge among the participants was observed.

Table 4 shows the result of attitude and practice among the participant in which the larger percentage of the population were found to practice consumption of foods rich in carbohydrate (78%). Most of them do not practice to plan and keep to a balance food menu (64%). Almost all the participants practice rarely eating foods rich in fats and oils.

Table 5 shows the socio-demographic data of the study subjects. Among the study population 128 (64%) were female and 72 (36%) were male, almost (80%) of the participants were hypertensive. Majority of the participants (80%) were within the age of 45-70 years in which (80%) were hypertensive. Generally, the majority of the participants had the following characteristics: female (64%), Married (92%), secondary education (58%), civil servant (38%), income less than #18000 per month (40%) and number of children 9- above (38%).

Table 2: Description of the Anthropometric Indices and Biochemical Parameters of the Hypertensive Individuals

VARIABLES ***n = 200***	**Mean ± SD**	**Minimum**	**Maximum**
Age	52.02±11.15	31.00	70.00
Height(cm)	161.68±15.26	89.00	188.00
Weight (kg)	79.22 ± 15.49	53.50	131.50
Hip Index	97.22 ± 21.60	40.09	187.97
ABSI	0.79 ± 0.16	0.33	1.60
CONICITY I	18.22 ± 4.01	8.97	38.35
BMI	29.91 ± 5.24	20.00	42.90
Body Fat (%)	40.40 ± 10.30	14.90	59.30
Muscle (%)	27.09 ± 6.74	16.70	43.20
Visceral Fat	11.68 ± 4.24	4.00	24.00
RM (Kcal)	1589± 184.48	1315.00	1933.00
Waist	96.60 ± 21.80	35.00	185.00
HC	97.74 ± 23.64	34.00	180.00
WHR	0.99 ± 0.76	0.67	1.11
Cholesterol (mg/dl)	143.18±43.76	66.00	217.00
HDL (mg/dl)	56.42± 56.42	23.00	82.00
TAG (mg/dl)	113.40±55.83	50.00	241.00
LDL (mg/dl)	70.418±35.09	21.30	153.00
TC/HDL	29.43±5.02	1.70	7.60
ACE activity (µmol/min/ml)	0.36±0.13	0.18	0.68
ANG II Level (ng/ml)	20.44±4.99	11.48	32.20

Table 3: Nutritional Knowledge among the Hypertensive and Control Individuals Attending Selected Hospitals in Kano Metropolis

S/N	QUESTIONS	CHOICE	HYP(%)	CONT(%)	*P*
			n=160	n=40	
1	Awareness on significance of Nutrition	YES	77.8	80.5	0.713
		NO	22.2	19.5	
2	Awareness on balance diet	YES	75.0	81.0	0.440
		NO	25.0	19.0	
3	Awareness on the main food nutrient	YES	71.4	83.3	0.059
		NO	28.6	16.7	
4	Awareness on the risk of overweight	YES	66.7	85.7	0.002
		NO	33.3	14.3	
5	Awareness on the knowledge of food for growth and development	YES	69.2	83.8	0.024
		NO	30.8	16.2	
6	Awareness on the risk of overfeeding	YES	66.7	85.7	0.002
		NO	33.3	14.3	
7	Awareness of the harmful foods	YES	73.3	82.9	0.123
		NO	26.7	17.1	
8	Awareness on the best source of nutrition information is hospitals	YES	76.2	82.8	0.252
		NO	23.8	17.2	
9	Having problems with appetite	YES	78.6	87.5	0.247
		NO	21.4	12.5	
10	Losing weight using pills, laxatives, not eating	YES	82.9	73.3	0.123
		NO	17.1	26.7	

HYP (Hypertensive), CONT (non-hypertensive)

Table 4: Attitude and Practice among Hypertensive and Control Individual Attending Selected Hospitals in Kano Metropolis

S/N	QUESTIONS	CHOICE	HYP (%) *n = 160*	CONT (%) *n = 40*	*P*
1	How often do you eat food substance rich in carbohydrate	PA	76.9	90.9	0.041
		DP	0.0	0.0	
		PR	23.1	9.1	
2	How often do you eat body building foods like egg, beans and milk	PA	77.8	81.2	0.556
		DP	0.0	0.0	
		PR	22.2	18.8	
3	How often do you eat fats and oils food substances	PA	50.0	81.2	0.030
		DP	0.0	0.0	
		PR	50.0	18.8	
4	How often do you eat food that are rich in vitamins like fruits,vegetable etc.	PA	51.3	20.0	0.039
		DP	27.3	68.9	
		PR	21.4	11.1	
5	How often do you drink eight and more glasses of water daily	PA	87.0	74.1	0.023
		DP	0.0	0.0	
		PR	13.0	25.9	
6	How often do you plan and keep to a balance food menu	PA	10.2	0.0	0.000
		DP	66.7	75.0	
		PR	23.1	25.0	
7	How often do you eat foods rich in fibre/roughages like skin of fruits, wheat and grains	PA	43.4	26.6	0.264
		DP	40.0	43.0	
		PR	22.0	30.4	
8	How often do you eat fried foods and other fattening foods	PA	17.7	44.5	0.006
		DP	40.0	30.1	
		PR	42.3	25.4	
9	How often did you exercise	PA	38.8	33.4	0.353
		DP	27.5	45.8	
		PR	33.7	20.8	
10	How often do you take vitamins and minerals supplements	PA	19.6	50.0	0.000
		DP	60.4	29.6	
		PR	20.0	20.4	

PA= practice Always DP= Do not practice PR= Practice rarely

Table 5: Socio-Demographic Characteristics between Hypertensive and Control Individuals Attending Selected Hospitals in Kano Metropolis

Characteristics	Total (n=200)n(%)	Hypertensive (n =160) n(%)	Control (n=40) n (%)	Chi^2	P
SEX					
Male	72(36)	56 (35)	16 (40)	0.347	0.556
Female	128(64)	104 (65)	24 (60)		
AGE					
25-34	16(8)	8 (5)	8 (20)	15	0.001
35-44	24(12)	24 (15)	0 (0)		
45-70	160(80)	128 (80)	32 (80)		
MARITAL STATUS					
Married	184(92)	152 (95)	32 (80)	9.783	0.002
Divorced	16(8)	8 (5)	8 (20)		
EDUCATIONAL LEVEL					
No formal Education	44(22)	32 (20)	12 (30)	3.989	0.136
Secondary	116(58)	92 (57.5)	24 (60)		
Tertiary	40(20)	36 (22.5)	4 (10)		
EMPLOYMENT STATUS					
Self employed	64(32)	48 (30)	16 (40)	1.842	0.398
Civil Servant	76(38)	64 (40)	12 (30)		
Not Employed	60(30)	48 (30)	12 (30)		
MONTHLY INCOME (PM)					
<#18,000	80(40)	68 (42.5)	12 (30)	6.042	0.11
#18 –#50,000	24(12)	16 (10)	8 (20)		
#51 – #100,000	64(32)	48 (30)	16 (40)		
>#100,000	32(16)	28 (17.5)	4 (10)		
NUMBER OF CHILDREN					
None	63(31.5)	63 (39.4)	0 (0)	31.245	0.001
1 – 4	17(8.5)	9 (5.6)	8 (20)		
5 – 8	44(22)	28 (17.5)	16 (40)		
9 – above	76(38)	60 (37.5)	16 (40)		

Table 6 indicates the distribution of anthropometric variables in hypertensive and control individuals as follows: comparing the status of the individuals, there were no statistically significance differences at $p<0.05$ in the anthropometric indices except; hip, fat composition and muscle composition which showed statistical difference at $p<0.05$.

Tables 7 and 8 indicate the distribution of anthropometric variables in males and females which were shown as follows: comparing individuals with and without hypertension there were statistically significant difference at $p<0.05$ in anthropometric indices except; BMI in males, and except ABSI, conicity index, muscle composition and visceral fat in females.

Table 9 shows the descriptive statistics with mean, SD, t- value and p- value of biochemical parameters among hypertensive and non hypertensive patients. It was found that there was no statistical difference $p > 0.05$ among hypertensive and Control individuals. But when stratified into male and female, LDL shows significant difference between male hypertensive and Control and in female only TAG shows significant difference at $p <0.05$.

Table 6: Comparison of Anthropometric Parameters between Hypertensive and Control Individuals Attending selected Hospitals in Kano Metropolis

Parameters	Total *(n= 200) n (%)*	Hypertensive *(n= 160) n (%)*	Control *(n= 40) n (%)*	Chi^2	*P*
Height (m)					
Q1	52 (26)	44(27.5)	8(20)	7.986	0.069
Q2	44 (22)	40(25)	4(10)		
Q3	44 (22)	32(20)	12(30)		
Q4	60 (30)	44(27.5)	16(40)		
Weight(Kg)					
Q1	52 (26)	40(25)	12(30)	5.449	0.143
Q2	48 (24)	44(27.5)	4(10)		
Q3	52 (26)	40(25)	12(30)		
Q4	48 (24)	36(22.5)	12(30)		
Hip C. (cm)					
Q1	52 (26)	36(22.5)	16(40)	18.11	0.000
Q2	48 (24)	44(27.5)	4(10)		
Q3	52 (26)	48(30)	4(10)		
Q4	48(24)	32(20)	16(40)		
BMI					
Normal	28(14)	24(15)	4(10)	3.473	0.324
Overweight	80(40)	64(40)	16(40)		
Obese I	68(34)	56(35)	12(30)		
Obese II	24(12)	16(10)	8(20)		
Body shape Index					
Q1	52 (26)	40(25)	12(6)	2.083	0.555
Q2	48 (24)	40(25)	8(4)		
Q3	52 (26)	44(27.5)	8(4)		
Q4	48 (24)	36(22.5)	12(6)		
Conicity Index					
Q1	52 (26)	36(22.5)	16(40)	8.494	0.037
Q2	48 (24)	40(25)	8(20)		
Q3	52 (26)	40(25)	12(30)		
Q4	48 (24)	44(27.5)	4(10)		
Fat comp.(%)					
Q1	52 (26)	36(22.5)	16(40)	21. 795	0.000
Q2	48 (24)	48(30)	0(0)		
Q3	52 (26)	44(27.5)	8(20)		
Q4	48 (24)	32(20)	16(40)		
Musclecomp. (%)					
Q1	52 (26)	40(25)	12(30)	23.397	0.000
Q2	48 (24)	44(27.5)	4(10)		
Q3	52 (26)	48(30)	4(10)		
Q4	48 (24)	28(17.5)	20(50)		
Visceral fat					
Q1	60 (30)	44(27.5)	16(40)	8.135	0.043
Q2	44 (22)	36(22.5)	8(20)		
Q3	44(22)	32(20)	12(30)		
Q4	52 (26)	48 (30)	4(10)		
Waist (cm)					
≥ 94	60(30)	48(30)	12(30)	17.56	0.000
≥80	140(70)	112(70)	28(70)		
W/H Ratio					
>0.9	4(2)	4(2.5)	0(0)	1.020	0.312
>0.85	196(98)	156(97.5)	40(100)		

Table 7: Distribution of Anthropometric Variables in Male Individuals Attending Selected Hospitals in Kano Metropolis

Parameters	Total *(n=72) n (%)*	Hypertensive *(n= 56) n (%)*	Control *(n= 16) n (%)*	Chi^2	*P*
Height (m)					
Q1	8(11.1)	4(7.1)	4(25)	10.987	0.012
Q2	12(16.7)	12(21.4)	0(0)		
Q3	8(11.1)	4(7.1)	4(25)		
Q4	44(61.1)	36(64.4)	8(50)		
Weight (Kg)					
Q1	20(27.8)	16(28.6)	4(25)	14.143	0.003
Q2	8(11.1)	4(7.1)	4(25)		
Q3	20(27.8)	12(21.4)	8(50)		
Q4	24(33.3)	24(42.9)	0 (0)		
Hip (cm)					
Q1	18(25.0)	12(21.4)	6(37.5)	16.457	0.000
Q2	22(30.6)	18(32.1)	4(25)		
Q3	12(16.7)	8(14.3)	4(25)		
Q4	26(36.1)	18(32.1)	3(18.75)		
BMI					
Normal	12(16.7)	8(14.3)	4(25)	2.057	0..561
Overweight	36(50.0)	28(50)	8(50)		
Obese I	20(27.8)	16(28.6)	4(25)		
Obese II	4(5.50)	4(7.1)	0(0)		
Body shape Index					
Q1	32(44.4)	20(35.7)	12(75)	17.036	0.001
Q2	20(28.6)	20(35.7)	0(0)		
Q3	12(16.7)	12(21.4)	0(0)		
Q4	8(11.1)	4(7.1)	4(25)		
Conicity index					
Q1	32(44.4)	20(35.7)	12(75)	9.321	0.025
Q2	24(33.3)	20(35.7)	4(25)		
Q3	8(11.1)	8(14.3)	0(0)		
Q4	8(11.1)	8(14.3)	0(0)		
Fat comp. (%)					
Q1	48(66.7)	32(57.1)	16(100)	10.386	0.016
Q2	16(22.2)	16(28.6)	0(0)		
Q3	4(5.50)	4(7.1)	0(0)		
Q4	4(5.50)	4(7.1)	0(0)		
Muscle composition (%)				25.714	0.000
Q1	0(0)	0(0)	0(0)		
Q2	4(5.50)	4(7.1)	0(0)		
Q3	36(50.0)	36(64.3)	0(0)		
Q4	32(44.4)	16(28.6)	16(100)		
Visceral fat				13.370	0.004
Q1	20(27.8)	12(21.4)	8(50)		
Q2	4(5.50)	4(7.1)	0(0)		
Q3	24(33.3)	16(28.6)	8(50)		
Q4	24(33.3)	24(42.9)	0(0)		
Waist (cm)					
≥ 94	43(60)	31(55.4)	12(16)	1.996	0.158
≥80	29(40)	25(44.6)	4(6)		
W/H Ratio					
>0.9	8(11)	5(8.9)	3(18.8)	2.022	0.089
>0.85	64(89)	51(91.1)	13(81.2)		

Table 8: Distribution of Anthropometric Variables in Female Individuals Attending Selected Hospitals in Kano Metropolis

Parameters	Total *(n= 128) n (%)*	Hypertension *(n= 104) n (%)*	Control *(n= 24) n (%)*	Chi^2	*P*
Height (cm)				14.056	0.003
Q1	44(34.4)	40(38.5)	4(16.7)		
Q2	32(25)	28(26.9)	4(16.7)		
Q3	36(28.1)	28(26.9)	8(33.3)		
Q4	16(12.5)	8(7.6)	8(33.3)		
Weight (Kg)				26.256	0.000
Q1	32(25)	24(23.1)	8(33.3)		
Q2	40(31.3)	40(38.5)	0(0)		
Q3	32(25)	28(26.9)	4(16.7)		
Q4	24(18.7)	12(11.5)	12(50)		
Hip C. (cm)				11.847	0.008
Q1	12(9.4)	12(11.5)	0(0)		
Q2	28(21.9)	24(23.9)	4(33.3)		
Q3	40(31.3)	36(34.6)	4(33.3)		
Q4	48(37.4)	32(30.8)	16(66.7)		
BMI				9.78	0.021
Normal	16(12.5)	16(15.4)	0(0)		
Overweight	44(34.4)	36(34.6)	8(33.3)		
Obese I	48(37.4)	40(38.5)	8(33.3)		
Obese II	20(15.6)	12(11.5)	8(33.3)		
Body shape Index				6.470	0.091
Q1	20(15.6)	20(19.2)	0(0)		
Q2	28(21.9)	20(19.2)	8(33.3)		
Q3	40(31.3)	32(30.8)	8(33.3)		
Q4	40(31.3)	32(30.8)	8(33.3)		
Conicity index				4.197	0.241
Q1	20(15.6)	16(15.4)	4(16.7)		
Q2	24(18.8)	20(19.2)	4(16.7)		
Q3	44(34.4)	32(30.8)	12(50)		
Q4	40(31.3)	36(30.8)	4(16.7)		
Fat composition(%)				17.405	0.001
Q1	4(3.1)	4(3.8)	0(0)		
Q2	32(25)	32(30.8)	0(0)		
Q3	48(37.5)	40(38.5)	8(33.3)		
Q4	44(34.4)	28(26.9)	16(66.7)		
Muscle comp. (%)				4.154	0.245
Q1	52(40.6)	40(38.5)	12(50)		
Q2	44(34.4)	40(38.5)	4(16.7)		
Q3	16(12.5)	12(11.5)	4(16.7)		
Q4	16(12.5)	12(11.5)	4(16.7)		
Visceral fat				0.469	0.926
Q1	40(31.3)	32(30.8)	8(33.3)		
Q2	40(31.3)	32(30.8)	8(33.3)		
Q3	20(15.6)	16(15.4)	4(16.7)		
Q4	28(21.8)	24(23.1)	4(16.7)		
Waist (cm)					
≥ 94	17(13.3)	17(16.3)	0(0)	4.524	0.033
≥80	111(86.7)	87(83.7)	24(100)		
W/H Ratio					
>0.9	4(3.1)	4(3.8)	0(0)	1.953	0.061
>0.85	124(96.9)	100(96.2)	24(100)		

Table 9: Lipid Profile Parameters for the Hypertensive and Control Individuals Attending Selected Hospitals in Kano Metropolis

Sex	Parameters (mg/dl)	Hypertension *n =(80)*	Control *n =(20)*	T	*P*
All	**Cholesterol**	143.83 ± 44.75	140.60 ± 40.56	0.29	0.770
	HDL	55.58 ± 14.45	59.80 ± 14.76	-1.16	0.247
	TAG	115.98 ± 55.69	103.10 ± 56.63	0.92	0.359
	LDL	71.93 ± 35.10	64.37 ± 35.10	0.86	0.391
	Tcl/HDL	3.08 ± 1.18	2.77 ± 0.75	-0.35	0.728
Male	**Cholesterol**	134.71± 40.81	117.75 ± 23.29	-1.117	0.272
	HDL	54.07 ± 7.30	58.50± 6.52	1.549	0.130
	TAG	118.86 ± 61.72	139.5 ± 76.63	0.79	0.430
	LDL	69.42 ± 34.39	41.93 ± 25.50	-2.09	0.040[a]
	Tcl/HDL	2.91 ± 0.90	2.89 ± 0.90	-0.05	0.960
Female	**Cholesterol**	148.73 ± 46.37	155.83 ± 43.16	0.48	0.630
	HDL	56.38 ± 17.13	60.67 ± 18.63	0.77	0.450
	TAG	114.42 ± 52.72	60.67 ± 18.63	-2.88	0.020[b]
	LDL	73.33 ± 35.75	79.33 ± 33.22	0.53	0.590
	Tcl/HDL	3.17 ± 1.31	2.68 ± 0.67	-1.25	0.220

Values are expressed as mean± SD, mean values having different superscript in the same row are significantly different at (p<0.05)

Table 10 shows the ANG II level and ACE activity among hypertensive and Control individuals respectively. There is significantly difference at $p<0.05$ in the ACE activity and ANG II level between the hypertensive and non hypertensive individuals.

Table 11 presents the results of the correlation between the anthropometric indices with the biochemical parameters in hypertensive and control individuals. All anthropometric indices correlated significantly with cholesterol, body fat, hip and BMI. BMI had the highest correlation coefficient with body fat, visceral fat with ACE, ANG II, cholesterol and LDL, Weight also has the highest correlation with ANG II, cholesterol, HDL, LDL and BMI, while CI had the lowest.

Table 12 shows the correlation between biochemical parameters and anthropometric indices in hypertensive male which indicates as follows: There is high correlation between weight with ACE activity, ANG II and cholesterol, hip index with ACE activity and ANG II, BMI with ACE, body fat composition (%) with ACE, ANG II and cholesterol, Visceral fat with ACE and ANG II, RM with ACE, ANG II, cholesterol and LDL, waist and Hip with ACE and ANG II.

Table 13 shows the correlation between biochemical parameters and anthropometric indices in hypertensive female which indicates as follows: There is high correlation between BMI, weight with ACE activity, ANG II level, cholesterol and LDL. There is also high correlation between weight, BMI, visceral fat, body fat and RM with TC/HDL ratio.

Table 10 **ANG II Level and ACE activity for the Hypertensive and Control Individuals Attending Selected Hospitals in Kano Metropolis**

		HYP	**CONT**
		n = 80	*n =20*
ANG II LEVELS			
(ng/ml)	FEMALE	22.15 ± 5.25[a]	16.62 ± 0.66[a]
	MALE	20.30 ± 4.51[a]	15.65 ± 0.39[a]
ACE ACTIVITY			
(µmol/min/ml)	FEMALE	17.02 ±0.11[b]	2.48 ±0.01[b]
	MALE	10.34 ±0.09[c]	1.56 ±0.01[b]

All values are means ± standard deviation (SD). Mean values having different superscripts letter in the same column are significantly different at ($p<0.05$)

Table 11: Correlation between Biochemical Parameters and Anthropometric Indices in Hypertensive and Control Individuals Attending Selected Hospitals in Kano Metropolis

VARIABLES	ACE activity (r)	ANG II level (r)	Cholestrol (r)	HDL (r)	TAG (r)	LDL (r)	TC/HDL
Height (cm)	-0.061	-0.095	0.032	0.191	0.184	-0.050	-0.049
Weight (kg)	0.231*	0.379**	0.580**	0.367**	-0.099	0.462**	-0.109
Hip index	0.185	0.250*	0.239*	0.163	-0.032	0.137	-0.140
ABSI	-0.010	0.047	0.080	0.118	-0.001	0.011	-0.150
Conicity I	0.041	0.012	-0.094	0.083	0.064	-0.161	-0.134
BMI	0.535**	0.679**	0.585**	0.252*	-0.244*	0.517**	-0.042
Bodyfat(%)	0.510**	0.541**	0.502**	0.182	-0.174	0.418**	0.071
Muscle (%)	-0.455**	-0.343**	-0.364**	-0.347**	0.137	-0.200*	0.129
Visceral fat	0.418**	0.545**	0.414**	0.129	-0.085	0.360**	0.026
RM (kcal)	0.043	0.143	0.216*	-0.074	0.087	0.188	0.198*
WC (cm)	0.253*	0.368**	0.359**	0.253*	-0.083	0.250*	-0.136
Hip (cm)	0.239*	0.355**	0.398**	0.232*	-0.079	0.285**	-0.132
WHR	-0.016	-0.029	-0.158	-0.012	0.074	-0.109	0.075
AGE	0.043	-0.024	-.206*	-0.111	-0.053	-0.238*	-0.003

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

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

n = 100

Table 12: Correlation between Biochemical Parameters and Anthropometric Indices in Hypertensive Male Individuals Attending Selected Hospitals in Kano Metropolis

VARIABLES	ACE activity (r)	ANG II level (r)	Cholesterol (r)	HDL (r)	TAG (r)	LDL (r)
Height (cm)	-0.233	-0.178	0.407*	0.407*	0.313	0.045
Weight (kg)	0.698**	0.808**	0.808**	0.053	0.436*	0.331
Hip index	0.490**	0.597**	0.048	-0.201	-0.114	0.260
ABSI	0.292	0.396*	-0.054	-0.137	-0.006	0.350
Conicity I	-0.116	-0.028	-0.413*	-0.052	-0.273	0.131
BMI	0.842**	0.938**	0.545**	-0.188	0.186	0.314
Body fat (%)	0.689**	0.728**	0.508**	-0.146	0.250	0.406*
Muscle (%)	-0.445*	-0.392*	-0.196	0.197	-0.018	0.118
Visceral fat	0.652**	0.830**	0.422*	-0.169	0.059	0.322
RM (kcal)	0.758**	0.766**	0.776**	-0.155	0.544**	0.326
WC (cm)	0.695**	0.830**	0.399*	-0.142	0.175	0.436*
Hip (cm)	0.706**	0.817**	0.325	-0.205	0.076	0.356
WHR	-0.033	0.043	0.231	0.189	0.345	0.278
AGE	-0.546**	-0.503**	-0.022	0.295	-0.183	-0.359

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

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

n = 50

Table 13: Correlation between Biochemical Parameters and Anthropometric Indices in Hypertensive Female Individuals Attending Selected Hospitals in Kano Metropolis

VARIABLES	ACE activity (r)	ANG II level (r)	Cholesterol (r)	HDL (r)	TAG (r)	LDL (r)	TC/HDL
Height (cm)	-0.238	-0.227	0.066	0.279*	0.316*	-0.162	-0.066
Weight (kg)	0.731**	0.736**	0.558**	0.302*	-0.132	0.554**	-0.155
Hip index	0.058	0.140	0.132	0.170	0.085	0.028	-0.288*
ABSI	-0.050	0.176	-0.0004	0.214	0.097	-0.145	-0.300*
Conicity I	-0.202	-0.162	-0.116	0.167	0.125	-0.267	-0.277*
BMI	0.928**	0.940**	0.540**	0.133	-0.334*	0.681**	-0.075
Body fat (%)	0.806**	0.722**	0.527**	0.282*	-0.248	0.571**	-0.164
Muscle (%)	-0.397**	-0.254	-0.339*	-0.495**	-0.097	-0.159	0.243
Visceral fat	0.691**	0.716**	0.429**	0.077	-0.195	0.560**	0.009
RM (kcal)	0.090	0.126	-0.008	-0.231	0.243	0.078	0.423**
WC (cm)	0.296*	0.352*	0.203	0.245	-0.017	0.113	-0.285*
Hip (cm)	0.262	0.346*	0.262	0.203	0.036	0.182	-0.278*
WHR	0.116	0.054	-0.109	0.048	-0.061	-0.112	0.087
AGE	0.127	0.048	-0.134	-0.118	0.037	-0.118	0.173

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

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

n = 50

The logistic regression analysis for hypertension was conducted. Anthropometry indices that have statistical significance were chosen from hypertensive and Controlin both males and females. In the first model height, HC, conicity index, RM and BMI were statistically significant. In Model 2 height, HC, weight, fat composition, RM and BMI were statistically significant at $p<0.05$ with hypertension in the female category. In male category height, conicity, and muscle composition showed no significant difference at $p<0.05$(Table 14).

Table 14 shows the dietary diversity score of the hypertensive and control individuals, average of individual dietary diversity score for the food groups consumed the day before was 6.8 ± 1.94 for hypertensive against 5.6 ± 1.25 for the control. Hypertensive patients in the study seemed to have a better diversity in their diet with 8% of them who had a high score against control. In the both groups, 20 % of the individuals had low DDS.

Table14: The Relationship Between Hypertension And Anthropometric Variables In Both Males And Females Individuals Attending Selected Hospitals in Kano Metropolis

MODEL 1 BETWEEN HYPERTENSIVE AND NORMOTENSIVE						
CHARACTERISTICS	MODEL 1	*P*	MODEL 2	*P*		*P*
n = 200	GENERAL		MALE		FEMALE	
Height						
Q2	**2.00(0.78,5.15)**	**0.151**	0.25(0.04,1.46)	**0.1241**	**0.81(0.54,9.82)**	**0.0306**
Q3	**3.64(1.12,11.79)**	**0.031**	0.38(0.09,1.54)	0.1741	2.33(0.62,8.72)	0.2078
Q4	0.97(0.40,2.33)	0.945	0.71(00,00)	0.9981	**0.33(0.11,1.03)**	**0.0571**
Constant	2.75(0.22,1.34)	0.001	4.01(00,00)	0.0131	3(00,00)	0.0071
Hip		0.001268		1.0000		0.0571
Q2	4.89(1.50,15.92)	0.008439	0.98(00,00)	0.9986	0.42(1.31,12.40)	0.9986
Q3	5.33(1.64,17.32)	0.005342	0.99(00,00)	1.0000	**3.01(0.89,10.12)**	**0.0768**
Q4	0.89(0.38,2.06)	0.783652	00(00,00)	0.9985	**2.10(1.02,11.21)**	**0.0236**
Constant	2.25(1.93,16.87)	0.006956			4.50(1.36,14.86)	0.0136
Weight						
Q2	3.30(0.98,11.07)	0.0531	**0.22(0.05,1.08)**	**0.0627**	**9.99(2.41,41.37)**	**0.0015**
Q3	**0.98(0.40,2.49)**	**0.0074**	3.52(0.71,1.98)	0.9986	**6.99(1.67,29.38)**	**0.0078**
Q4	0.91(0.36,2.25)	0.8220	**0.22(0.05,1.08)**	**0.0627**	**3.5(0.97,12.29)**	**0.0506**
Constant	3.33(0.14,01.97)	0.0002	4.5(00,00)	0.0001	0.99(00,00)	1.0000
Conicity						
Q2	**2.22(0.85,5.81)**	**0.1034**	3.0(0.83,10.90)	0.0951	1.25(0.27,5.80)	0.7756
Q3	**1.48(0.62,3.55)**	**0.058**	00(00,00)	0.9988	0.67(0.19,2.40)	0.5350
Q4	**4.89(1.50,15.92)**	**0.008**	00(00,00)	0.9988	**2.25(0.50,10.14)**	**0.1225**
Constant	2.25(00,00)	0.007	1.67(00,00)	0.1618	4(00,00)	0.0131
Muscle						
Q2	0	0.997195	00(00,00)	0.9984	1.87(00,00)	1.0000
Q3	**2.44(0.94,6.36)**	**0.066932**	00(00,00)	0.9992	3.10(00,00)	0.9992
Q4	0.89(0.38,2.06)	0.783652	00(00,00)	0.9992	1.08(00,00)	0.9992
Constant	2.25(00,00)	0.006956	2.0(00,00)	0.0236	1.61(00,00)	0.9992
Fat						0.2774
Q2	**3.30(0.98,11.02)**	**0.053101**	0.99(00,00)	1.0000	**2.99(0.89,10.09)**	**0.0760**
Q3	**3.60(1.07,12.03)**	**0.037504**	**0.6(0.24,4.50)**	**0.028**	0.9(0.24,3.31)	0.8740
Q4	**0.42(0.18,0.99)**	**0.048917**	1,6(00,00)	0.9992	**3.33(0.22,9.73)**	**0.0003**
Constant	3.33(00,00)	0.000254			0.90.24,3.31)	0.8740
RMK						
Q2	**0.30(0.09,1.02)**	**0.053101**	00(00,00)	0.9992	**0.25(0.07,0.93)**	**0.0388**
Q3	0.45(0.13,1.3)	0.22525	00(00,00)	0.9992	0.11(00,00)	0.9979
Q4	0.20(0.06,0.67)	**0.008439**	00(00,00)	0.9992	**0.10(0.03,0.37)**	**0.0005**
Constant	10.99(00,00)	4.40E-06	00(00,00)	0.9992	10(00,00)	0.0000
BMI						
Normal	**0.67(0.20,2.20)**	**0.05049**	1.75(0.42,7.35)	0.4445	1.87(0.42,1.70)	0.1984
Overweight	**1.78(0.23,2.66)**	**0.04845**	2.11(0.39,10.16)	0.4032	**3.10(1.0,2.52)**	**0.0941**
Obese	**2.33(0.085,1.29)**	**0.01124**	**3.1(0.56,12.67)**	**0.0287**	1.08(1.6,2.902)	0.1283
Constant	5.99(00,00)	0.000908	2	0.0007	2.25(1.93,16.87)	0.0083

Table 15 Dietary Diversity Score between Hypertensive Individuals Attending Selected Hospitals in Kano Metropolis

Dietary Diversity Score	Hypertensive patients n =160 (%) Medium +/- SD = 6.8+/- 1.94	Control n = 40(%) Medium +/- SD = 5.5+/- 1.25
1	0.8	0.6
2	0.8	0.6
3	7	4
4	16	12
5	28	21
6	29	27
7	16	17
8	12	10
9	4	5
10	3	1
11	4	2
12	2	1
13	1	3
Dietary Diversity Terciles		
Low (1-4)	24.5	22.5
Medium (5-9)	70	81
High (10-14)	9	1.5

DISCUSSION

This study was conducted on patients attending some selected hospitals (Murtala Muhammad Specialist Hospital, Muhammad Abdullahi Wase Teaching Hospital, Sheik Muhammad Jiddah General Hospital, Sir Muhammad Sunusi General Hospital) in Kano metropolis. Most of the participants were women and married with the highest percentage having primary education. There is lack of nutritional knowledge among the participants; in terms of attitude and practice most of the participants always practice consumption of foods rich in carbohydrates and body building foods. Most of the participant do not practice to plan and keep to the balance food menu and taking vitamins and minerals supplements. The finding could be related with that of a similar study, which states that sound educational attainment is directly proportional to the nutritional knowledge, attitude and practice (Chimberengawa *et al*., 2019). Some societies that are vulnerable to malnutrition due to socio-economic factors, poverty, ignorance and poor educational background. These factors contributed to poor nutritional knowledge, attitude and practices (Chimberengawa *et al*., 2019).

In hypertensive and normotensive there is a difference in most of the anthropometric indices between hypertensive patients and normotensive individual including both overall and abdominal obesity indicators. These results suggested that hypertension is linked to obesity in both male and female, which is consistent with previous studies that both overall and abdominal obesity are significantly associated with hypertension (Nguyen *et al*., 2008; He *et al*., 2009). The mechanisms of this association have been suggested to include structural arterial abnormalities, leptin, and the activation of the renin–angiotensin–aldosterone axis (Nguyen and Lau, 2012). The adverse effects of BMI and waist circumference on the risk of hypertension have also been

shown in the multitude of longitudinal studies including Framingham Heart study (Wang*et al*., 2018).The NIH (US) has indicated that there is a graded increase in the health risk with increase in BMI, in the normotensive category increase in WC is also associated with high risk (NIH 1998). Furthermore, other studies showed that it may also be related to the variant of gene (Baudrand *et al*., 2015; Skrypnik *et al.*, 2017). Fortunately, obesity being a modifiable factor, Nutritional knowledge, attitude and practice, lifestyle modifications including increased physical activity, endurance and endurance-strength exercise, and dietary modifications can decrease the incidence of hypertension (Skrypnik *et al.*,2015 and 2016;Szulinska *et al.*, 2016).

In the present study, it was found that there were gender differences in the relationship between anthropometric indices and the prevalence of hypertension in patients attending some selected Hospitals (Murtala Muhammad Specialist Hospital, Muhammad Abdullahi Wase Teaching Hospital, Sheik Muhammad Jiddah General Hospital, Sir Muhammad Sunusi General Hospital) in Kano metropolis. In both genders, association was stronger between BMI and hypertension without considering the impact of age. These results are supported by numerous studies that the increase of BMI contributes to blood pressure increase (Wilsgaard *et al*., 2000; Droyvold *et al*., 2005). However, the association of HI and hypertension in females was stronger with increased age. Also, there are still some studies that reveal that high HC is a risk factor for the multi-metabolic disorders (Liu *et al.*, 2008). This result may be due to the following two reasons. First, HC carries some information on both overall obesity and abdominal obesity, since HC is positively correlated with BMI and WC (Wang *et al*., 2010). Second, studies have indicated that more gravidity was associated with a consistent increase in the risk of metabolic syndrome in women (Xu *et al*., 2014). In view of these results, it should be a priority to encourage those males with high BMI and females with high HC to pay more attention to guidelines of

hypertension. Besides, health care facilities should strengthen the screening and monitoring for those at-risk subgroups to early identify and treat the elderly with hypertension.

Another results of the current study indicated that, overall, obesity was associated with hypertension in males, and overweight was associated with hypertension in females. This may be concluded that the amount of body fat mass in males and the distribution of body fat in females is closely related to hypertension (Cho *et al*., 2009). Body fat composition as a measure of total fat in the body shows to have association with reduce insulin sensitivity, glucose tolerance, adverse lipid profiles and other metabolic abnormalities which are risk factors of hypertension and some other chronic diseases (Huxley *et al.,* 2010). In previous studies, general obesity and partial obesity usually showed the similar relationship with hypertension in different gender. For example, some studies indicated that general obesity has a stronger association with hypertension in both males and females (Khashayar*et al*., 2017). Others found that the association of partial obesity with hypertension was stronger in both genders (Lee *et al*., 2008). Results of this study indicated that the role of overall obesity and overweight in hypertension in separate gender may be different. Further studies are necessary to confirm this assumption.

In the presence study, among the anthropometric variables: WC, BMI, Visceral fat and weight were best correlated with ACE activity, ANG II level and cholesterol (positively) and negatively with TAG. There is no statistical difference in hypertensive and normotensive in males and females, but when stratified it shows statistical difference in HDL and LDL (male) hypertensive and normotensive , also TAG and TAG/HDL ratio female hypertensive and non hypertensive. The result is contrary to the previous studies which indicate that there is no significance

difference in the lipid profile parameters on gender among hypertensive and normotensive individuals (Wang*et al.,* 2018).

In the present study ACE activity and ANGII level show significant difference between hypertensive and normotensive, they also show significant correlation (positive) with WC, BMI, Visceral fat and weight. Therefore the present findings indicate that there is an increase in the ACE activity and the level of ANG II in obese hypertensive individuals. The finding was supported by the previous study which show the elevation in plasma ANG II concentrations accompanying obesity in hypertensives (Carine *et al.,* 2004). The mark elevation in ANG II in obese hypertensive individuals could mediate the increase in blood pressure through many mechanisms. As suggested by Carine (2004), ANG II may increase blood pressure in experimental animals by stimulating the symphatic nervous systems with release of norepinephrine in the brain and periphery. It may likely be due to the blockage in the arteries as a result of fat deposit on the wall of the arteries which stimulate the increase in the level of ANG II in the system. Researchers have also showed the AGT expression profile related to obesity; Umemura*et al*. (1997) reported a correlation between increased plasma AGT levels with BMI and blood pressure in obese individuals. Similarly, Giacchetti *et al.*(2000)investigated AGT mRNA expression in adipose tissue of obese individuals and reported a significant association between AGT expression and BMI in visceral adipose tissue.

Dietary diversity score consists of the total number of foods or food groups that contribute to the overall diet of an individual over a reference period(FAO, 2007). Dietary diversity assessed in this study consisted of simple count of food groups that individuals consumed over a 24-hoursreference period. One of the methods employed in defining cutoff points for assessing

varying levels of dietary diversity in populations is to create terciles and sometimes quintiles(Ruel, 2003). Terciles of DDS based on 14 food groups were adopted in this study to determine the proportion of subjects scoring low, average and high DDS. In previous studies, obesity was found to be related to dietary diversity in which authors observed a high prevalence of overweight or obesity in respectively 38% and 36% (Arimond *et al*., 2010). The mean DDS obtained was 6.8 diets groups for hypertensives and 5.5 diets groups for control. Other vegetables (72%) (onion, tomato and pepper), cereals (92%), fish (67%) and oils and fats (53%) were the most consumed food groups by the study individuals. The explanation of the same is that these items all consumed every day. In contrast, other fruits (23%), nuts and seeds (10%) were the least consumed food groups. Feeding behavior of the participant was found to be related to the financial status. Therefore, it is important to teach the healing properties of good alimentation and in particular fruits and vegetables consumption to the population. It will be appropriate to prescribe a diet for patients after each consultation. The finding could also be related with that of a similar study by Sanusi *et al*. (2010) which assessed dietary diversity in six Nigerian states and reported a prevalence of 92.1% for cereals, 99% for fats and 59.7% for tubercles and food rich in vitamin A. Although dietary diversity of populations has been reported to range from 3 to 6 (Savy *et al.,* 2005), different numbers of food groups and scoring systems have been employed in different countries to assess dietary diversity (Savy *et al.,*2008; Drescher, 2007).

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATION

5.1 SUMMARY

There are gender differences in the association between anthropometric indices of obesity and hypertension in obese individuals. More over BMI and HC were found to have a significant association with hypertension in both male and female categories.

The findings indicate a need to develop gender-specific strategies for the male and female in the primary and secondary prevention of hypertension. In practice, these anthropometric measures are surrogate measures of body fat and are cost free, practical and easy to interpret for healthcare providers and lay people. In context of developing countries, indices of obesity (both general and abdominal) could be used simultaneously but independently to predicts risk for both conditions, since they both performed well and possibly define different mechanisms of the association of obesity with hypertension and other cardiovascular disorders.

It showed that there is an increase in the activity of ACE and the level of ANG II in obese hypertensive individuals.

There is also poor KAP and below average of diet diversification. The findings also indicate that there is significant positive high correlation between lipid profile parameters (TAG, HDL, LDL Cholesterol) and anthropometric indices (Weight, Hip Circumference, BMI, Visceral fat, weight circumference, Body fat composition and resting Metabolism).

5.2 CONCLUSION

In conclusion, there is a poor nutritional knowledge, attitude and practices among the study individuals, also there is an average dietary diversity scores. Significant high correlation between some anthropometric indices and biochemical parameters was observed. Angiotensin converting enzymes activity and angiotensin II levels was significantly higher in the hypertensive individuals.

5.3 RECOMMENDATION

Base on the findings of this study, the following recommendations are made as follows;

1. These findings indicate a need to develop gender-specific strategies for the male and female in the primary and secondary prevention of hypertension.
2. Further research need to be carry on the expression on the angiotensinogen gene in obese hypertensive individuals
3. Further research need to be carried out on the expression of the two receptors of the ANG II i.e ATIR gene and AT2R gene.
4. Additional studies with larger populations should be performed to elucidate this combined relationship.

5.4 CONTRIBUTION TO KNOWLEDGE

1. Based on the finding of this study, Nutrition Education is highly needed for the prevention and Management of hypertension.

2. This findings indicate the need to develop gender-specific strategies for male and female in the prevention of hypertension.
3. It also indicates the need to adopt the use of new anthropometric indices such as Hip Index, Conocity Index and a body shape index, because these measures proved to be complementary to BMI and other risk factors of Hypertension.
4. Based on this study it shows that the individuals have average dietary diversity scores. Food diversification is highly need for the prevention and management of hypertension due to the fact that plant family has a unique combination of phyto-nutrients that may bind to specific proteins in the body.

REFERENCE

Abegunde, D. O., Mathers, C. D., Adam, T., Ortegon, M., and Strong, K., (2007). The burden and costs of chronic diseases in low-income and middle-income countries. The Lancet, 370 (9603): 1929–1938.

Abhyankar, L. N., Jones, M. R., Guallar, E., Navas, A. (2012). "Arsenic exposure and hypertension: a systematic review.".*Environmental Health Perspectives*. 120 (4): 494–500.

Adeloye, D., Basquill, C., Aderemi, A. V., Thompson, J. Y. and Obi, F. A. (2015). An estimate of the prevalence of hypertension in Nigeria: a systematic review and meta-analysis.

Agachan, B., Isbir, T., Yilmaz, H., Akoglu, E. (2003). Angiotensin converting enzyme I/D, angiotensinogen T174M–M235T and angiotensin II type 1 receptor A1166C gene polymorphisms inTurkish hypertensive patients. *Exp Mol Med*, 35:545–549.

Aguilera, G. (1992). Role of angiotensin II receptor subtypes on the regulation of aldosterone secretion in the adrenal glomerulosa zone in the rat. *Mol Cell Endocrinol*, 90:53–60.

Aikins, G. A., Unwin, N., Agyemang, C., Allotey, P., Campbell, C., Arhinful, D. (2010). Tackling Africa's chronic disease burden: from the local to the global. Global Health 6:5.

Akinkugbe, O. (1997). Noncommunicable diseases in Nigeria: final report of a national survey. Lagos: Federal Ministry of Health – National Expert Committee on Non-Communicable Diseases.

Annette, D. K., Dipanwita, P.,Lei, W.,Helmut, H.,Colin, S., Charles, J., James , P., Stephen, C.and Eric, G. K. (2013). Angiotensin Type 1a Receptors in the ParaventricularNucleus of the Hypothalamus Protect against Diet-InducedObesity.*The Journal of Neuroscience*, 33(11):4825– 4833.

Arimond M, Wiesmann D, Becquey E, Carriquiry A, Daniels M. (2010) Simple food group diversityindicators predict micronutrient adequacy of women's diets in 5 diverse, resource-poor settings. *Journal of Nutrition*, 140:2059-2069.

Aronne, L,J. (2001). Epidemiology, morbidity and treatment of overweight and obesity. *J Clin Psychiatry*, 62 (23):13–22.

Babandi, A.,Muhammed, I.Y., Murtala, Y.,Ibrahim, A.A., Madugu, A. U., Babagana, K.,Jobbi,Y., Mashi, J.A., Yakasai, H.M., Shehu, D. and Abubakar, S.M. (2017). Oxidative Stress Indices And CalciumLevel Among Hypertensive Patients In Kano-Nigeria *BJMLS*. 2(1): 576-581.

Bacani, C. and Frishman, W.H. (2006). Chymase: a new pharmacologic target in cardiovascular disease. *Cardiol Rev*. 14:187–193.

Banks, E., Lim, L., Seubsman, S.A., Bain, C. and Sleigh, A. (2011). Relationship of obesity to physical activity, domestic activities, and sedentary behaviours: cross-sectional findings from a national cohort of over 70,000Thai adults," *BMC Public Health*, 11(762): 14.

Barilla Centre for Food and Nutrition. (2011). Double Pyramid 2011- Healty Food for People, Sustainable for Planet. BCFN. Obesity and Overweight. Available online: http://www.Who.Int/mediacentre/factsheets/fs311/en/(accessed on 20 February 2018).

Baudrand, R., Goodarzi, M.O., Vaidya, A., Underwood, P.C., Williams, J.S., Jeunemaitre, X., Hopkins, P.N., Brown, N., Raby, B.A. and Lasky-Su, J., (2015). A prevalent caveolin-1 gene variant is associated with the metabolic syndrome in Caucasians and Hispanics. *Metabolism*, 64:1674–1681.

Beaglehole R, Bonita R, Alleyne G, Horton R, Li L, Lincoln P, et al. UN high-level meeting on non-communicable diseases: addressing four questions. Lancet 2011; 378:449–455.

Bello, M. (2013). Nigerians wake up to high blood pressure. Bull World Health Organ; 91:242–243.

Bergier, J., Kapka-Skrzypczak, L., Bili´nski, P., Paprzycki, P. and Wojtyła, A. (2012). Physical activity of Polish adolescents and young adults according to IPAQ: a population based study. *Annals of Agricultural and Environmental Medicine*, 19(1):109– 115.

Bhurosy, T. and Jeewon, R. (2013). "Food habits, socioeconomic status and body mass index among premenopausal and postmenopausal women in Mauritius." *Journal of Human Nutritionand Dietetics*, 26 (1):114–122.

Bhurosy, T. and Jeewon, R. (2014). "Overweight and Obesity Epidemic in Developing Countries: AProblem with Diet, Physical Activity, or Socioeconomic Status?" *The Scientific World Journal*, 2014(964236); 7.

Bibiloni, M.M., Pich, J. and Pons, A. (2013). Body image and eating patterns among adolescents. *BMC public Health*, 13 (1104).

Blair, S.N., Kohn, H.W. and Paffenbarger, R.S. (1989). Physical fitness and all-cause mortality: a prospective study of healthy men and women. *JAMA*, 262:2395–401.

Bloomgarden, Z.T. (2006). Third Annual World Congress on the Insulin Resistance Syndrome: Associated conditions. *Diabetes Care*, 29: 2165-2174.

Bonnardeaux, A., Davies, E. and Jeunemaitre, X. (1994). Angiotensin II type 1 receptor gene polymorphisms in human essential hypertension. *Hypertension*, 24 (1): 63–69.

Bray, G.A. and Ryan, D. H. (2000).Clinical evaluation of the overweight patient. *Endocrine.* 13:167–86.

Brewster, U.C., Perazella, M.A. (2004). The renin-angiotensin-aldosterone system and the kidney: effects on kidney disease. *Am J Med*, 116:263–272.

Burson, J.M., Aguilera, G., Gross, K.W., Sigmund, C.D. (1994). Differential expression of angiotensin receptor 1A and 1B in mouse. *Am J Physiol*, 267:E260–E267.

Caballero, B. (2007). The global epidemic of obesity: an overview, *Epidemiologic Reviews*, 29 (1): 1–5.

Carine, M., Boustany, A., Kalyani B., Alan, D., David, R., Brown, X., David, C., Randall, V., Lisa. A. Cassis, A. (2004). Activation of the systemic and adipose renin-angiotensin system in rats with diet-induced obesity and hypertension. *Am J Physiol Regul Integr Comp Physiol,* 287: R943–R949.

Carretero, O.A. and Oparil, S. (2000). Essential hypertension. *Part I: definition and etiology.* Circulation 101 (**3**): 329–35.

Cassis, L.A., Saye, J.A., Peach, M.J. (1988). Location and regulation of rat angiotensinogen messenger RNA. *Hypertension* 11:591–596.

Castell, G. S., Serra-Majem, L. and Ribas-Barba, L. (2015). What and how much do we eat? 24-hour dietary recall method. *Nutrition Hospital*, 31 (3): 46-48.

Centers for Disease Control and Prevention, (2013). "Overweight and obesity 2012," 2013, http://www.cdc.gov/obesity/adult/causes/ index.html.

Cesari, M., Rossi, G.P., Pessina, A.C. (2002). Biological properties of the angiotensin peptides other than angiotensin II: implications for hypertension and cardiovascular diseases. *J Hypertens*, 20:793–799.

Cheng, J.L., Wang, A. L. and Wan J. (2012). Association between the M235T polymorphism of the AGT gene and cytokines in patients with hypertension," *Experimental and Therapeutic Medicine*, 3 (3): 509–512.

Chimberengwa, P. T., Mergan, N. (2019). Knowledge, attitudes and practices related tohypertension among residents of a disadvantaged rural community in southernZimbabwe.*PLoS ONE*14(6): e0215500. https://doi.org/10.1371/journal.pone.0215500.

Cho, Y.G., Song, H.J., Kim, J.M., Park, K.H., Paek, Y.J., Cho, J.J., Caterson, I., Kang, J.G. (2009,). The estimation of cardiovascular risk factors by body mass index and body fat percentage in Korean male adults. *Metabolism* 58:765–771.

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., Jr-Jones, D. W., Materson, B. J., Oparil, S., Wright, J. T., Roccella, E. J. (Dec 2003). Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. National Heart, Lung, and Blood, Institute; National High Blood Pressure Education Program Coordinating, Committee."Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure". *Hypertension*. 42 (6): 1206–52.

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., Jr-Jones, D. W., Materson, B. J., Oparil, S., Wright, J. T., Roccella, E. J. (Dec 2003). Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. National Heart, Lung, and Blood, Institute; National High Blood Pressure Education Program Coordinating, Committee."Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure". *Hypertension*. 42 (6): 1206–52.

Coatmellec-Taglioni, G., and Ribière, C. (2003). Factors that influence the risk of hypertension in obese individuals. *Current Opinion Nephrology Hypertension* 12: 305-308.

Collazo-Clavell, M.L. (1999).Safe and effective management of the obese patient. *Mayo Clin Proc.*, 74:1255–9.

Cooper, R.S., Rotimi, C.N., Karfman, J.S. (1998). Hypertension treatment and control in sub-saharan Africa*: the epidemiological basis for policy.* BMJ. 316: 614 – 617.

Coulston, A. M., Boushey, C. J. and Ferruzzi, M. G. (2013). Nutrition in the prevention and treatment of disease, *Third edition, Elsevier Incorporation*, San Diego, USA, 5

Crackower, M.A., Sarao, R., Oudit. G.Y., Yagil, C., Kozieradzki, I., Scanga, S.E., Oliveira-dos-Santos, A.J., da Costa, J., Zhang, L. and Pei, Y. (2002). Angiotensin-converting enzyme 2 is an essential regulator of heart function. *Nature*, 417: 822–828. (doi:10.1038/nature00786)

Cressman, M.D., Gifford, R.W. (1983). Hypertension and stroke. *Journal of the American College of Cardiolog,*. 1: 521 -527.

Cushman, D.W., Cheung, H.S.(1971). Spectrophotometric assay and properties of the angiotensin-converting enzymes of rabbit lungs. *Biochem pharmacol 20, 1637-1648.*

Cuspidi, C., Lonati, L., Sampieri, L., Michev, L., MaccaG, Rocanova, J. I., Salerno, M., Fusi, V., Leonetti, G. and Zanchetti, A. (2000). Prevalence of target organ damage in treated hypertensive patients: different impact of clinic and ambulatory blood pressure control. *Journal of Hypertension*. 18:803-809.

Dahly, D. L., Gordon, L. P., Popkin, B. M., Kaufman, J. S. and Adair, L. S.(2010). "Associations between multiple indicators of socioeconomic status and obesity in young adult Filipinos vary by gender, urbanicity, and indicator used," *Journal of Nutrition,* 140 (2): 366–370.

Danilczyk, U., Penninger, J. M. (2006). Angiotensin-converting enzyme II in the heart and the kidney. *Circ. Res*, 98: 463–471.

Davisson, R.L., Oliverio, M.I., Coffman, T.M., Sigmund, C.D. (2000). Divergent functions of angiotensin II receptor isoforms in the brain. *J Clin Invest*, 106:103–106.

De Backer, G., Ambrosioni, E., Borch-Johnsen, K., Brotons, C., Cifkova, R., Dallongeville, J., Ebrahim, S., Faergeman, O., Graham, I., Mancia, G. (2003). European guidelines on cardiovascular disease prevention in clinical practice: third joint task force of European and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of eight societies and by invited experts). *Eur J Cardiovasc Prev Rehabil*, 10(4):S1–S10

Delavari,M., Sønderlund, A. L., Swinburn, B., Mellor,D. andRenzaho, A. (2013). "Acculturation and obesity among migrant populations in high income countries—a systematic review," *BMC Public Health*, 13(1): 11.

Development Initiatives, 2017. Global Nutrition Reports: Nourishing the SDGs. Bristol, UK: Development Initiatives.

Dobrian, A.D., Davies, M.J., Schriver, S.D., Lauterio, T.J. and Prewitt, R.L. (2001).Oxidative stress in a rat model of obesity-induced hypertension. *Hypertension*,37: 554–560.

Drescher, L. S., Thiele, S., Mensink, G. B. (2007). A New Index to Measure Healthy Food Diversity Better Reflects a Healthy Diet than Traditional Measures. *Journal of Nutrition,* 137:647-651.

Droyvold, W.B., Midthjell, K., Nilsen, T.I., Holmen, J. (2005). Change in body mass index and its impact on blood pressure: A prospective population study. *International Journal of Obesity* 29:650–655.

Dunn, R. A., Sharkey, J. R., Lotade-Manje, J., Bouhlal, Y. and Nayga, R. M. (2011). "Socio-economic status, racial composition and the affordability of fresh fruits and vegetables in neighborhoods of a large rural region in Texas." *Nutrition Journal,* 10 (1): article 6.

Dunneram, Y. and Jeewon, R. (. 2013). "A scientific assessment of sociodemographic factors, physical activity level, and nutritional knowledge as determinants of dietary quality among indomauritian women," *Journal of Nutrition and Metabolism,* 2013 (9): Article ID 572132.

Ehret, G.B., Munroe, P.B., Rice, K.M. (2011). Genetic variants in novel pathways influence blood pressure and cardiovascular disease risk. *Nature* 478 (7367): 103–9.

Engeli, S., Bohnke, J., Gorzelniak, K., Janke, J., Schling, P. and Bader, M. (2005). Weight loss and the renin-angiotensin-aldosterone system. *Hypertension*; 45: 356–362.

Engeli, S., Gorzelniak, K., Kreutz, R., Runkel, N., Distler, A. and Sharma, A.M. (1999).Co-expression of renin-angiotensin system genes in human adiposetissue. *J Hypertens,* 17: 555–560.

Engeli, S., Negrel , R., Sharma, A.M. (2000). Physiology and pathophysiology of the adipose tissue renin-angiotensin system. *Hypertension* 35:1270–1277.

FAO. (2007) Guidelines for Measuring Household and Individual dietary diversity. FAO Nutrition and consumer Protection Division, with Support from the EC/FAO Food Security Information for Action Programme and the Food and Nutrition Technical Assistance (FANTA) Project.

Ferrario, C.M., Chappell, M.C. (2004). Novel angiotensin peptides. *Cell Mol Life Sci*. 61:2720–2727.

Ferrario C. M. (2006). Angiotensin-converting enzyme 2 and angiotensin-(1-7): an evolving story incardiovascular regulation. *Hypertension*, 47;(515–521).

Fisher, N.D., Williams, G.H. (2005). "Hypertensive vascular disease". In Kasper DL, Braunwald E, Fauci AS. Harrison's Principles of Internal Medicine (16th ed.). New York, NY: McGraw-Hill, 1463–81. ISBN 0-07-139140-1.

Fitzgerald, P.A. (2000).Endocrinology. In: Tierney LM, McPhee SJ, Papadakis MA, eds. *Current Medical Diagnosis & Treatment.* New York: Lange Medical Books, 1079–151.

Fleming, M. T., Robinson, M., Thomson, B., Graetz, N. and Margono, C. (2013). Global, regional, and national prevalence of overweight and obesity in children and adults during 1980±2013: a systematic analysis for the Global Burden of Disease Study. *Lancet*, 384(9945):766±781..

Fokeena, W. B. and Jeewon, R. (2012). “Is there an association between socioeconomic status and body mass index among adolescents in mauritius?”.*The ScientificWorld Journal*, 2012 (9): Article ID 750659.

Ford, E.S., Williamson, D.F., Liu, S. (1997).Weight change and diabetes incidence: findings from a national cohort of US adults. *Am JEpidemiol.* 146:214–22.

Franklin, S.S., Wilkinson, I.B., and McEniery, C.M.. (2012). Unusual hypertensive phenotypes.*Journal of Hypertension* 59 (2): 173–8.

Friedewald WT, Levy RI, Fredrickson DS (1972). "Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge". Clin. Chem. 18: 499–502. (Cited in: Clin. Chem. 1990; 36:15-19).

Galanis, D.J, Harris, T., Sharp, D.S., Petrovich, H. (1998).Relative weight, weight change, and risk of coronary heart disease in the Honolulu Heart Program. *Am J Epidemiol.* 147:379–86.

Gaman, P.M. and Sherrington, K.B. (2013). The science of food: an introduction to food science, nutrition and microbiology. *Elsevier*.

George, P., Ludvik, B. (2000). Lipids and diabetes. *J Clin Basic Cardiol*, 3:159–162.

Giacchetti, G., Faloia, E., Sardu, C., Camilloni, M.A., Mariniello, B., Gatti, C. (2000). Gene expression of angiotensinogen in adipose tissue of obese patients. *Int J Obes Relat Metab Disord,* 24(2):142-3.

Go, A. S., Mozaffarian, D. And Roger V. L. (2013) "Heart disease and stroke statistics-2013 update: a report from the American Heart Association," *Circulation*, 127: e6–e245,.

Grossman, E. and Messerli, F. H., (2012). "Drug-induced Hypertension: An Unappreciated Cause of Secondary Hypertension". *America. Journal of Medicine*, 125 (1):14–22.

Grzegorzewska, A., Wolejko, K., Kowalkowska, A., Kowalczyk, G. and Jaroch, A. (2016). Proper BMI ranges for the elderly in the context of morbidity, mortality and functional status. *Gerontologia Polska*, 24: 114-118.

Guyenet, S.J. and Schwartz, M.W. (2012), Clinical review: Regulation of food intake, energy balance, and body fat mass: implications for the pathogenesis and treatment of obesity. *J Clin Endocrinol Metab*, 97: 745-755.

Haffner, S.M., Lehto, S., Ronnemaa, T., Pyorala, K., Laakso, M. (1998). Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. *N Engl J Med,* 339:229–234

Hall, J.E., Crook, E.D., Jones, D.W., Wofford, M.R. and Dubbert, P.M. (2002). Mechanisms of obesity-associated cardiovascular and renal disease. *Am J Med Sci* , 324: 127-137.

Hall, J.E. and Louis, K. (1994). Dahl Memorial Lecture. Renal and cardiovascular mechanisms of hypertension in obesity. *Hypertension* 23: 381-394.

Hamilton, T.A., Handa, R.K., Harding, J.W., Wright, J.W. (2001). A role for angiotensin IV/AT4 system in mediating natiuresis in the rat. *Peptides*. 22:935–944.

He, X., Zhu, D.L., Chu, S.L., Jin, L. and Xiong. M.M. (2001). Alpha-Adducin gene and essential hypertension in China. *ClinExpHypertens*. 23:579–589.

Heiss, C. J., and Goldberg, L. R. (2016). Associations among visceral, obesity, type 2 diabetes and dementia. *Journal of Obesity and Eating Disorders*, 2 (2): 27-39.

Hindorff, L. A., Heckbert, S. R. and Tracy R. (2002). "Angiotensin II type 1 receptor polymorphisms in the cardiovascular health study: relation to blood pressure, ethnicity, and cardiovascular events," *The American Journal of Hypertension*, 15 (12): 1050–1056.

Hirsch J, Salans LB, Aronne LJ. Obesity. In: Becker KL, ed. Principles and Practice of Endocrinology and Metabolism, 3rd ed. Philadelphia: Lippincott, Williams, and Wilkins; 2001, pp. 1239–46.

Hoffman, D. J. (2004). Upper limits in developing countries: warning against too much in lands of too little," *Journal of the American College of Nutrition*, 23 (6): 610–615.

Huang, X.R., Chen, W.Y., Truong, L.D., Lan, H.Y. (2003). Chymase is upregulated in diabetic nephropathy: implications for an alternative pathway of angiotensin II-mediated diabetic renal and vascular disease. *J Am Soc Nephrol*, 14:1738–1747.

Hunyady, L., Catt, K.J. (2006) Pleiotropic AT1 receptor signaling pathways mediating physiological and pathogenic actions of angiotensin II. *Mol Endocrinol*, 20:953–970.

Huxley, R., Mendis, S., Zheleznyakov, E.(2010). Body mass index, waist circumference and waist:hip ratio as predictors of cardiovascular risk—a review of the literature. *Eur J Clin Nutr*. 64:16–22.

Ingelfinger J. R. (2009). Angiotensin-converting enzyme 2: implications for blood pressure and kidney disease. *Current Opinion in Nephrology and Hypertension*, 18 (1): 79–84,.

Inoue, I., Nakajima, T., Williams, C,S., Quackenbush, J., Puryear, R., Powers, M., Cheng, T., Ludwig, E.H., Sharma, A. M., Hata, A., Jeunemaitre, X., Lalouel, J.M. (1997). A nucleotide substitution in the promoter of human angiotensinogen is associated with essential hypertension and affects basal transcription in vitro. *J Clin Invest*, 99:1786–1797.

Ito, N., Ohishi, M., Yamamoto, K., Tatara, Y., Shiota, A., Hayashi, N., Komai, N., Yanagitani, Y., Rakugi, H., Ogihara, T. (1995). Renin-angiotensin inhibition reverses advanced

cardiac remodeling in aging spontaneously hypertensive rats. *Am J Hypertens*, 20:792–799.

James, P.A., Oparil, S., Carter, B.L., Cushman, W.C., Dennison-Himmelfarb, C., Handler, J., Lackland, D.T. and Lefevre, M.L. (2013). 2014 Evidence-Based Guideline for the Management of High Blood Pressure in Adults: Report from the Panel Members Appointed to the Eighth Joint National Committee (JNC 8). J*AMA* 311 **(5**): 507–20.

Jiang, S. Z., Lu, W., Zong, X. F., Ruan, H. Y. and Liu, Y. (2016). Obesity and hypertension (Review).*Experimental and Therapeutic Medicine* 12: 2395-2399,.

Jieying, J., Mengling, L.,, Faruque, P. (2015). "Association between Arsenic Exposure from Drinking Water and Longitudinal Change in Blood Pressure among HEALS Cohort Participants". *Environmental Health Perspectives*. 123(8).

Johnson, M.L., Pietz, K., Battleman, D.S., Beyth, R. J. (2004). Prevalence of comorbid hypertension and dyslipidemia and associated cardiovascular disease. *Am J Manag Care,* 10:926–932

Kario, K. (2009). Psychological and physical stress-induced cardiovascular resctivity and diurnal blood pressure variation in women with different work shifts. *Hypertens Res*. 25: 543-551.

Kearney, P.M., Whelton, M., Reynolds, K., Muntner, P., Whelton, P.K. (2005*).* Global burden of hypertension: *Analysis of world wide data. Lancet.* 365 (9455): 217 -223.

Khakurel, S., Agrawal R. K. and Hada R. (2009). Pattern of end stage renal disease in a tertiary care center. *Journal of the Nepal Medical Association*. 48:126 – 130.

Khashayar, P., Aghaei Meybodi, H., Rezaei Hemami, M., Larijani, B. (2017). Role of obesity variables in detecting hypertension in an iranian population. High Blood Press. *Cardiovasc. Prev*, 24:305–312.

Kim, H. M., Shin, D. R., Yoo, O. J., Lee, H. and Lee, J. O. (2003). Crystal structure of *Drosophila* angiotensin I-converting enzyme bound to captopril and lisinopril. *FEBS Lett*. 538: 65–70.

Kingue, S., Ngoe, C.N .and Menanga, A.P. (2015). ‘’ Prevalance and risk factors of hypertension in Urban areas of Cameroon: A National Population-Based Cross Sectional Study,’’ *Journal of Clinical Hypertension*, 17 (10): 819-824,.

Kobori, H., Harrison-Bernard, L.M., Navar, L.G. (2001). Enhancement of angiotensinogen expression in angiotensin II-dependent hypertension. *Hypertension*, 37:1329–1335.

Kotsis, V, Stabouli, S., Bouldin, M., Low, A., Toumanidis, S. and Zakopoulos, N. (2005). Impact of obesity on 24-hour ambulatory blood pressure and hypertension. *Hypertension* 45: 602-607.

Kramar EA, Harding JW, Wright JW. Angiotensin II- and IV-induced changes in cerebral blood flow. Roles of AT1 and AT2, and AT4 receptor subtypes. Regul Pept. 1997;68:131–138.

Kumanyika, S., Jeffery, R. W., Morabia, V., Ritenbaugh, C. and Antipatis, V. J. (2002). "Obesity prevention: the case for action." *InternationalJournal of Obesity*, 26: (3), 425–436.

Kwok, C.Y. Wong, C.N., Yau, M.Y., Yu, P.H., Au, A.L., Poon, C.C., Seto, S.W., Lam, T.Y., Kwan, Y.W. and Chan, S.W.(2010). Consumption of dried fruit of Crataegus pinnatifida (hawthorn) suppresses high-cholesterol diet-induced hypercholesterolemia in rats. *J Funct Foods* 2: 179-186.

Landsberg, L. and Krieger, D.R. (1989). Obesity, metabolism, and the sympathetic nervous system. *Am J Hypertens,* 2: 125S-132S.

Laurent, Y., Patrick, E., May, B., Miche` le, G., Naima, M., Pascal, F. and Annie, Q. (2005). Deletion of the Angiotensin Type 2 Receptor (AT2*R*) Reduces Adipose Cell Size and Protects From Diet-Induced Obesity and Insulin Resistance *Diabetes,* 54: 991–999.

Lawlor, A. and Smith, D. (2005). *Current Opinion in Nephrology and Hypertension* 14(3):259-64

Ledwozyw, A., Michalak, J., Stepień, A. and Kadziołka, A. (1986). The relationship between plasma triglycerides, cholesterol, total lipids and lipid peroxidation products during human atherosclerosis. *Clin Chim Acta,* 155: 275-283.

Lee, C.M. Huxley, R.R., Wildman, R.P.. Woodward, M.(2008). Indices of abdominal obesity are better discriminators of cardiovascular risk factors than BMI: A meta-analysis. *Journal of Clinical Epidemiology*., 61 :646–653.

Levin, B.E., Dunn-Meynell, A.A., Balkan, B. and Keesey, R.E. (1997). Selective breeding for diet-induced obesity and resistance in Sprague-Dawley rats. *Am J Physiol Regul Integr Comp Physiol* 273: R725–R730,

Levin, B.E., Dunn-Meynell, A.A. and Banks, W.A. (2004). Obesity-prone rats have normal blood-brain barrier transport but defective central leptin signaling before obesity onset. *Am J Physiol Regul Integr Comp Physiol*, 286: 143–150.

Lewington, S., Clarke, R., Qizilbash, N., Peto, R. and Collins, R. ((2002). Prospective Studies, Collaboration. Age-specific relevance of usual blood pressure to vascular mortality: *A meta analysis of individual data for one million adults in 61 prospective studies. Lancet 360* (9349): 1903–13.

Life, J.S. (2014). The Life Plan Diet: How Losing Belly Fat is the Key to Gaining a Stronger, Sexier, Healthier Body. Simon and Schuster, New York, NY,

Liu, Y., Zhuoma, C., Shan, G. (2008). A1166C polymorphism of the angiotensin II type 1 receptor gene and essential hypertension in Han, Tibetan and Yi populations," *Hypertension Research*, 25(4):515–521.

Luma, G.B. and Spiotta, R.T. (2006). Hypertension in children and adolescents. *Am Fam Physician* 73 **(**9): 1558–68.

Lyznicki, J.M., Young, D.C., Riggs, J.A., Davis, R,M. (2001). Obesity: assessment and management in primary care. *Am Fam Pract Physician*, 63:2185–2189.

Marshall, I.J., Wolfe, C.D., McKevitt, C. (2012). Lay perspectives on hypertension and drug adherence: *systematic review of qualitative research*. *BMJ* (*Clinical research ed.)* 345: e3953.

Massiera, F., Bloch-Faure, M., Ceiler, D., Murakami, K., Fukamizu, A., Gasc, J.M., Quignard-Boulange, A., Negrel, R., Ailhaud, G., Seydoux, J., Meneton, P. and Teboul, M. (2001). Adipose angiotensinogen is involved in adipose tissue growth and blood pressure regulation. *FASEB J*, 15: 2727–2729.

Mathers, C.D. and Loncar, D. (2006). Projections of global mortality and burden of disease from 2002 to 2030. *PLoS Medicine*, 3 (11): article e442.

Matsuzawa, Y. (2005). Metabolic syndrome - definition and diagnostic criteria in Japan. *J Atheroscler Thromb*, 12: 301-301.

McLaren, L. (2007). "Socioeconomic status and obesity," *Epidemiologic Reviews*, 29 (1):29–48.

Mezue, K. (2013). The increasing burden of hypertesnsion in Nigeria: can dietary salt reduction strategy change the trend?.*Perspect Public Health*.

Mirowsky, J. and Ross, C.E. (2003). Social causes of psychological distress. Transaction Publishers, New Jersey.

Miyazaki, M., Takai, S. (2006). Tissue angiotensin II generating system by angiotensin-converting enzyme and chymase. *J Pharmacol Sci*. 100:391–397.

Mizuiri, S., and Ohashi, Y. (2015). ACE and ACE2 in kidney disease. *World J. Nephrol*, 4:74–82. doi: 10.5527/wjn.v4.i1.74.

Mohana, V. U., Swapna, N., Surender, R. S., Vishnupriya S. and Padma T. (2012). "Gender-related association of AGT gene variants (M235T and T174M) with essential

hypertension: a case-control study," *Clinical and Experimental Hypertension*, 34 (1): 38–44.

Momteiro, C. A., Conde, W. L. And Popkin, B. M. (2002). Is obesity replacing or adding to undernutrition? Evidence from different social classes in Brazil," *Public Health Nutrition A*, 5 (1): 105–112.

Monsivais, P. and Drewnowski, A. (2009). "Lower-energy-density diets are associated with higher monetary costs per kilocalorie and are consumed by women of higher socioeconomic status." *Journal of the American Dietetic Association*, 109(5):814–822.

Muhamedhussein, M. S., Nagri, Z.I. and Manji, K.P. (2016). Prevalence, Risk factors, Awareness, and Treatment and control of Hypertension in Mafia Island Tanzania. *International Journal of Hypertension*. 5. Article ID 1281384.

Musini, V.M., Tejani, A.M., Bassett, K., Wright, J.M. (2009*).* Pharmacotherapy for hypertension in the elderly.*The Cochrane database of systematic reviews* (4): CD000028.

National Health and Medical Research Council (2013). Clinical practice for the management of overweight and obesity in adults, adolescents and children in Australia. Melbourne: National Health and Medical Research Council.

National Heart, Lung and Blood Institute Obesity Education Initiative and National Institutes of Health. (2012). "Pediatric Cardiovascular Risk Reduction Guidelines ". *Archived from the original* on 2012-11-16.

National Institutes of Health (NIH), National Heart, Lung, and Blood Institute (NHLBI). (1998).Clinical guidelines on theidentification, evaluation and treatment of overweight andobesity in adults: The evidence report. *Obes Res.*, 6(2):51S–209S.

National Institutes of Health (NIH); National Heart, Lung, and Blood Institute (NHLBI); North American Association for the Study of Obesity (NAASO). (2000). The Practical Guide: Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. NIH Publication Number :4000 – 4084. Rockville, MD: National Institutes of Health;

Neal, B., MacMahon, S., Chapman, N. (2000). Effects of ACE inhibitors, calcium antagonists and other blood pressure lowering drugs*:* results of prospectively designed overviews of randomised controlled trials*. Lancet*. 356:1955-1964.

Nguyen, N.T., Magno, C.P., Lane, K.T., Hinojosa, M.W., Lane, J.S. (2008). Association of hypertension, diabetes, dyslipidemia, and metabolic syndrome with obesity: Findings from the national health and nutrition examination survey, 1999 to 2004. *Journal of American College of Surgeon*., 207:928–934.

Nguyen, T., Lau, D.C. (2012). The obesity epidemic and its impact on hypertension. *Canada Journal of Cardiology.*, 28:326–333.

NIH/NHLBI (National Institute of Health/ National Heart, Lung and Blood Institute) (1998). Clinical guidelines on the identification, evaluation and treatment of overweight and obesity in adults: the evidence report. *Obes Res*, 6(2): 51S.

O'Brien, E., Asmar, R., Beilin, L., Imai, Y., Mallion, J.M., Mancia, G. (2007). European Society of Hypertension recommendations for conventional, ambulatory and home blood pressure measurement. *J Hypertens*, 21:821–848.

O'Brien, C., Henrich, P.P., Passi, N., Fidock, D.A. (2011*). Recent clinical and molecular insights into emerging artemisinin resistance in Plasmodium falciparum. Current Opinion in Infectious Diseases 24* (6): 570–7. doi:10.1097/QCO.0b013e32834cd3ed.

Ogah, O.S. (2006). Hypertension in Sub-Saharan African populations: the burden of hypertension in Nigeria [letter]. *Ethn Dis,* 16:765.

Oishi K, Zheng B and Kuo JF (1990): Inhibition of Na,K-ATPase and sodium pump by protein kinase C regulators sphingosine, lysophosphatidylcholine, and oleic acid. *Journal Biology and Chemistry* 265: 70-75.

Oliverio, M.I., Coffman, T.M. (2000). Angiotensin II receptor physiology using gene targeting. *News Physiol Sci*, 15:171–175.

Perez-Escamilla, R., (2018). '' Nutrition disparities and global burden of malnutrition'' Science and politics of Nutrition. *BMJ.*

Pinterova, L., Krizanova, O. and Zorad, S. (2000). Rat epididymal fat tissue express all components of the renin-angiotensin system. *Gen PhysiolBiophys*, 19: 329–334.

Pobee, J.O., Larbi, E.B., Belcher, D.W., Wurapa, F.K., Dodu, S.R. (1977). Blood pressure distribution in a rural Ghanaian population. *Trans R Soc Trop Med Hyg.* 71: 66 – 72.

Porrello, E.R., Delbridge, L.M., Thomas, W.G. (2009). The angiotensin II type 2 (AT2) receptor: an enigmatic seven transmembrane receptor. Front BioSci, 14:958–972.

Portela, D.S., Vieira, T.O., Matos, S.M., Oliveira, N.F. and Vieira, G.O. (2015). Maternal obesity, environmental factors, cesarean delivery and breastfeeding as determinants of overweight and obesity in children: Results from a cohort. *BMC* Pregnancy Childbirth 15: 94,

Post, W. S., Hill, M. N., Dennison, J. L. (2003). High prevalence of target organ damage in young, African American Inner city men with hypertension. *Journal of clinical hypertension*. 5:2 30.

Prentice, A. M. (2006). The emerging epidemic of obesity in developing countries. *International Journal of Epidemiology,* 35 (1): 93–99.

Racette, S.B., Deusinger, S.S., Strube, M.J., Highstein, G.R. and Deusinger, R.H. (2005). Weight changes, exercise, and dietary patterns during freshman and sophomore years of college. *J Am Coll Health* 53: 245-251.

Radhika, G., Ganesan, A., Sathya, R.M., Sudha, V., Mohan, V. (2009). Dietary carbohydrates, glycemic load and serum high-density lipoprotein cholesterol concentrations among South Indian adults. *Eur J Clin Nutr*, 63(3):413–420.

Rahmouni, K., Mark, A.L., Haynes, W.G. and Sigmund, C.D. (2005). Adipose depot-specific modulation of angiotensinogen gene expression in dietinduced obesity. *Am J Physiol Endocrinol Metab* 286: E891–E895.

Raj, M. and Kumar, R. K. (2010).“Obesity in children & adolescents,” *Indian Journal of Medical Research*, 132(11):598–607.

Reaux, A., Fournie-Zaluski, M.C., Llorens-Cortes, C. (2001). Angiotensin III: a central regulator of vasopressin release and blood pressure. *Trends Endocrinol Metab*, 12:157–162.

Reinhard, T., Böhringer, D., Enczmann, J., Kögler, G., Mayweg, S., Wernet, P. and Sundmacher, R. (2003). HLA class I and II matching improves prognosis in penetrating normal-risk keratoplasty. *Dev Ophthalmol*. 36:42-9. doi: 10.1159/000067654. PMID: 12494679

Rigat, B., Hubert, C., Alhenc-Gelas, F., Cambien, F., Corvol, P., Soubrier, F. (1990) An insertion deletion polymorphism in the angiotensin I-converting enzyme gene accounting for half the variance of serum enzyme levels. *J Clin Invest* 86:1343–1346.

Rocchini, A.P., Yang, J.Q. and Gokee, A. (2004). Hypertension and insulin resistance are not directly related in obese dogs. *Hypertension* 43: 1011-1016.

Rodrigo, C. P., Aranceta, J., Salvador, G., and Varela-Moreiras, G. (2015). Food Frequency Questionnaires. *Nutrition Hospital*, 31 (3):49-56.

Rosenbaum, M. and Leibel, R.L. (1998). The physiology of body weight regulation: Relevance to the etiology of obesity in children. Pediatrics 101: 525-539.

Rudnichi, A., Safar, M. E., Lajemi, M. and Benetos, A. (2004). Gene polymorphisms of the renin-angiotensin system and age-related changes in systolic and diastolic blood pressure in subjects with hypertension. *American Journal of Hypertension*, 17 (4):321–327.

Ruel, M.T. (2003). OperationalizingmDietary Diversity: A Review of Measurement Issues and Research Priorities. *Journal of Nutrition 133:3911-3926.*

Ruggenenti, P., Bettinaglio, P., Pinares, F., Remuzzi, G. (2008). Angiotensin converting enzyme insertion/deletion polymorphism and renoprotection in diabetic and nondiabetic nephropathies. *Clin J Am Soc Nephrol*, 3:1511–1525.

Ruiz-Ortega, M., Lorenzo, O., Egido. J. (2000). Angiotensin III increases MCP-1 and activates NF-kappaB and AP-1 in cultured mesangial and mononuclear cells. *Kidney Int*, 57:2285–2298.

Ruster C. and Wolf, G. (2006). Renin-angiotensin-aldosterone system and progression of renal disease. *J. Am. Soc. Nephrol*.**17**(10): 2985–2991.

Samuel, F. and Atinmo, L. (2008). Obesity and cardiovascular diseases: the risk factor in African diets. *Forumon Public Policy*, 1–15.

Sanusi, R. A. (2010). An assessment of dietary diversity in six Nigerian states. *African Journal of Biomedical* Res, 13(3):161-167.

Savy, M., Martin-Prevel, Y., Daniel, P., Traissac, P., Dabire, H. (2008). Are Dietary Diversity Scores relatedto the socio-economic and anthropometric status of women living in an urban area in BurkunaFaso?.*Public Health Nutrition,* 11(2):132-41.

Savy, M., Martin-Prével, Y., Sawadogo, P., Kameli, Y., Delpeuch, F. (2005). Use of variety/diversity scoresfor diet quality measurement: relation with nutritional status of Women in a rural area in BurkinaFaso. *Eur J Clin Nutri*, 59(5):703-16.

Schulman, I.H., Raij, L. (2008). The angiotensin II type 2 receptor: what is its clinical significance.*Curr Hypertens Rep*, 10:188–193.

Sclafani, A. (1989). Dietary-induced overeating. *Ann NY Acad Sci* 575 (290): 281–289.

Shaper, A.G., Wright, D.H., Kyobe, J. (1969). Blood pressure and body built in three nomadic tribes of northern Kenya. *East Afr Med J.* 46*: 273 –281*

Shenoy, U. and Cassis, L. (1997).Characterization of renin activity in brown adipose tissue. *Am J Physiol Cell Physiol*, 272: C989–C999.

Shim, J., Oh, K. and Kim, H. C. (2014). Dietary assessment methods in epidemiologic studies. *Epidemiology and Health*, 36: e2014009.

Sidhu, D., Naugler, C. (2012). "Fasting Time and Lipid Levels in a Community-Based Population: A Cross-sectional Study / Fasting Time and Lipid Levels". *Archives of Internal Medicine*. 172(22):1–4. doi:10.1001/archinternmed.2012.3708.

Singhal, A., Sadaf Farooqi, I., Tim, J., Cole, Stephen, O., Mary, F., Mia, K., Alan, L. and John D. (2002).Influence of Leptin on Arterial Distensibility: A Novel Link Between Obesity and Cardiovascular Disease?.*American Heart Association.* Online ISSN: 1524-4539.

Siu, AL. (2015). Screening for High Blood Pressure in Adults: U.S. Preventive Services Task Force Recommendation Statement. *Annals of internal medicine*, 163: 778–86.

Skrypnik, D., Bogdanski, P., Madry, E., Karolkiewicz, J., Ratajczak, M., Krysciak, J., Pupek-Musialik, D., Walkowiak, J. (2015). Effects of endurance and endurance strength training on body composition and physical capacity in women with abdominal obesity. *Obes. Facts*, 8:175–187.

Skrypnik, D., Ratajczak, M., Karolkiewicz, J., Madry, E., Pupek-Musialik, D., Hansdorfer-Korzon, R., Walkowiak, J., Jakubowski, H., Bogdanski, P. (2016). Effects of endurance and endurance-strength exercise on biochemical parameters of liver function in women with abdominal obesity. *Biomed. Pharmacother*, 80:1–7.

Skrypnik, K., Suliburska, J., Skrypnik, D., Pilarski, L., Regula, J., Bogdanski, P. (2017). The genetic basis of obesity complications. *Acta Sci. Pol. Technol. Aliment.* 16:83–91.

Sounira, M., Sinda, M., Sonia, H., Amira, Z., Ameur, F., Fathi, B., Rachid M., Mohamed H. (2012). Renin-angiotensin system polymorphisms in relation to hypertension status and obesity in a Tunisian population. *Mol Biol Rep* 39:4059–4065

Steckelings, U.M., Kaschina, E., Unger, T. (2005). The AT2 receptor—a matter of love and hate. Peptides, 26:1401–1409.

Stepniakowski, K.T., Goodfriend, T.L. and Egan, B.M. (1995). Fatty acids enhance vascular α-adrenergic sensitivity. *Hypertension* 25: 774-778.

Steyn, K., Gaziano, T. A., Bradshaw, D.R., Laubscher, and Fourie J. (2001) "Hypertension in South African adults: results from the Demographic and Health survey," *Journal of Hypertension*, 19 (10): 1717–1725.

Sugimoto, K., Katsuya, T.and Ohkubo T. (2004) "Association between angiotensin II type 1 receptor gene polymorphism and essential hypertension: the Ohasama study." *Hypertension Research*, 27 (8): 551–556.

Sundström, J., Arima, H., Jackson, R., Turnbull, F., Rahimi, K., Chalmers, J., Woodward, M., Neal, B. (2015). Effects of Blood Pressure Reduction in Mild Hypertension. *Annals of Internal Medicine*, 162: 184–91.

Suzuki, Y., Ruiz-Ortega, M., Lorenzo, O., Ruperez, M., Esteban, V., Egido, J. (2003). Inflammation and angiotensin II. *Int J Biochem Cell Biol*, 35:881–900.

Szulinska, M., Skrypnik, D., Ratajczak, M., Karolkiewicz, J., Madry, E., Musialik, K., Walkowiak, J., Jakubowski, H. Bogdanski, P. (2016). Effects of endurance and endurance-strength exercise on renal function I abdominally obese women with renal hyperfiltration: A prospective randomized trial. *Biomed. Environ. Sci.*, 29: 706–712.

Temple, N. J., Steyn, K., Hoffman, M., Levitt, N. S. and Lombard, C. J. (2001). "The epidemic of obesity in South Africa: a study in a disadvantaged community," *Ethnicity and Disease*, 11(3): 431–437.

Thekkumkara, T.J., Cookson, R., Linas, S.L. (1998). Angiotensin (AT1A) receptor-mediated increases in transcellular sodium transport in proximal tubule cells. *Am J Physiol*, 274:F897–F905.

Thompson, F. E. and Subar, A. F. (2001). Dietary assessment methodology, In: Coulston, A.M., Rock, C.L. and Monsen, E.R. (Eds). Nutrition in the Prevention and Treatment of Disease. *Academic Press, San Diego,* USA. 5-9.

Tibarzawa, K., Ntyintyane, L. And Silwa, K. (2009) ‘’ A time bomb of cardiovascular risk factors in South Africa: results from the Heart of Soweto study ‘Heart Awareness Day’,’*’ International Journal of Cardiology*, 132 (2): 233-239.

Tiez, N.W. (1990). Serum triglyceride determination. In: chemical guide to laboratory test, second edition, W.B., sauders co., Philadelphia, USA; 554-556.

Timmermans, P.B., Wong, P.C., Chiu, A.T. and Herblin, W.F. (1993). Angiotensin II receptors and angiotensin II receptor antagonists. *Pharmacol. Rev*, 45: 205-251.

Toth, P.P. (2005). The “good cholesterol” high-density lipoprotein. *Circulation,* 111:89–91.

Touyz, R.M. and Schiffrin, E.L. (1999). Ang II-stimulated superoxide production is mediated via phospholipase D in human vascular smooth muscle cells. *Hypertension* 34: 976–982.

Turconi, G., Guarcello, M. and Maccarini, L. (2008). “Eating habits and behaviors, physical activity, nutritional and food safety knowledge and beliefs in an adolescent Italian population,” *Journal of the American College of Nutrition*, 27(1):31–43.

Uckaya, G., Ozata, M., Sonmez, A., Kinalp, C., Eyileten, T. and Bingol, N. (1999). Plasma leptin levels strongly correlate with plasma rennin activity in patients with essential hypertension. *Horm Metab Res*,31: 435–438.

Ulasi, I.I., Ijoma, C.K, Onwubere, B.J., Arodiwe, E., Onodugo. O. and Okafor, C. (2011). High prevalence and low awareness of hypertension in a market population in Enugu, Nigeria. *Int J Hypertens.*, 27 (869675):869675.

Umemura, S., Nyui, N., Tamura, K., Hibi, K., Yamaguchi, S., Nakamaru, M. (1997). Plasma angiotensinogen concentrations in obese patients. Am J Hypertens, 10(6):629-33.

Urata, H., Boehm, K.D., Philip, A., Kinoshita, A., Gabrovsek, J., Bumpus, F.M., Husain, A. (1993). Cellular localization and regional distribution of an angiotensin II-forming chymase in the heart. *J Clin Invest*, 91:1269–1281.

W.H.O. (2003). Factsheet: Obesity and overweight; http://www.who.int/hpr/NPH/docs/gs_obesity.pdf.

WHO World Health Organization (2018). Obesity and overweight. Geneve: WHO

Wald, N.J., Law, M.R. (2003). A strategy to reduce cardiovascular disease by more than 80%. *BMJ* 326:1419–1423.

Wang, J.L., Xu, L., Hao, P. P., Xu, F., Chen, Y.G. and Zhang Y.(2010) "Angiotensin II type 1 receptor gene A1166C polymorphism and essential hypertension in Chinese: a meta-analysis," *Journal of the Renin-Angiotensin-Aldosterone System*, 11 (2):127–135.

Wang, Q., Xu, L., Li, J., Sun, L., Qin, W., Ding, G., Zhu, J., Zhang, J., Yu, Z. and Xie, Su. (2018). Association of Anthropometric Indices of Obesity with Hypertension in Chinese Elderly: An Analysis of Age and Gender Differences. *Int. J. Environ. Res. Public Health*, 15:801.

Weinsier, R.L., Kushner, R.F. (1995).Clinical assessment of obese patients. In: Brownell KD, Fairburn CG, eds. *Eating Disordersand Obesity: A Comprehensive Handbook*. New York: The Guilford Press; 512–71.

WHO AFRO, (2005). Editor. Report of regional director: cardiovascular diseases in the African region: current situation and perspectives. Regional committee for Africa; Maputo, Mozambique: The WHO Regional Office for Africa.

Wille, W.C., Manson, J.E. and Stampfer, M.J. (1995).Weight, weight change, and coronary heart disease in women. Risk within the "normal" weight range. *JAMA.*;273:461–5.

Williams, B., Poulter, N.R., Brown, M.J., Davis, M., McInnes, G.T., Potter, J.F., Sever, P.S., and McGThom, S. (2004). British Hypertension, Society Guidelines for management of hypertension: report of the fourth working party of the British Hypertension Society, 2004-BHS IV. *Journal of Human Hypertension 18* (3): 139–85.

Wilsgaard, T., Schirmer, H., Arnesen, E. (2000). Impact of body weight on blood pressure with a focus on sex differences: The tromso study, 1986-1995. *Arch. Intern. Med*. 160:2847–2853.

Witkowski, T. H. (2007). "Food marketing and obesity in developing countries: analysis, ethics, and public policy." *Journal of Macromarketing*, 27 (2):126–137.

Wofford, M.R., Adair, C., Anderson, D.C., Hall, J.E., Miller, M.M. and Jones, D.W. (1998). Alpha and beta adrenergic blockade in obese and lean hypertensive subjects. *Hypertension*, 32: 595.

World Bank. Nigeria. (2013b). http://data.worldbank.org/country/nigeria. [22 June 2013]

World Health Organization (2019). Obesity and overweight. Geneve: WHO: (2019).

World Health Organization. (1997). Obesity: preventing and managing the global epidemic of obesity. Report of the WHO Consultation of Obesity. Geneva, Switzerland.

World Health Organization. (2011). Global status report on noncommunicable diseases. Geneva: WHO.

World Health Organization. (2013a). a global brief on hypertension: silent killer, global public health crises (World Health Day 2013). Geneva: WHO; 2013. http://apps.who.int/iris/bitstream/10665/79059/1/WHO_DCO_WHD_2013.2_eng.pdf.

Xiao, Y., Zhao, N. and Wang, H. (2013). "Association between socioeconomic status and obesity in a Chinese adult population," *BMCPublic Health*, 13 (10): article 355.

Xie, X., Atkins, E., Lv, J., Bennett, A., Neal, B., Ninomiya, T., Woodward, M., MacMahon, S., Turnbull, F., Hillis, G.S., Chalmers, J., Mant, J., Salam, A., Rahimi, K., Perkovic, V., Rodgers, A. (2015). Effects of intensive blood pressure lowering on cardiovascular and renal outcomes*: Updated systematic review and meta-analysis. Lance*t (London, England).

Xu, F.H., Huo, Y.N., Wu, M.B., Liu, J.D., Zhang, Y.L. (2014). Relationship of parity and other factors with metabolic syndrome in middle-age and elderly women in Nanchang community. *China Journal of Endocrinology*. *Metab*. 30:4.

Yanagisawa, A., Sudo, N., Amitani, Y., Caballero, Y., Sekiyama, M., Mukamugema, C., Matsuoka, T., Imanishi, H. and Sasaki, T. (2016). Development and Validation of a Data-based Food Frequency Questionnaire for Adults in Eastern Rural area of Rwanda. *Nutrition and Metabolic Insight*, 9:31-42.

Yasue, S., Masuzaki, H., Okada, S., Ishii, T., Kozuka, C. and Tanaka, T. (2010). Adipose tissue-specific regulation of angiotensinogen in obese humans and mice: impact of nutritional status and adipocyte hypertrophy. *Am J Hypertens*, 23: 425–431.

Zhu, Q., Shen, F., Ye, T., Zhou, Q., Deng, H. and Gu, X. (2014). Waist-to-weight ratio is an appropriate index for identifying cardiometabolic risk in Chinese individuals with normal body mass index and waist circumference. *Journal of Diabetes*, 6: 527-534.

APPENDICES

APPENDIX 1: NUTRITONAL KNOWLEDGE, ATTITUDE AND PRACTICE QUESTIONNAIRE

QUESTIONNAIRE

Name: _____

Height _____ Weight _____ BP_____

BMI _____ RM kcal_____ Sex: Male ☐ female ☐

Body fat%_____ Muscle% _____ Visceral fat _____

Waist (cm)_____ Hip(cm)_____

SECTION A

Socio-demographic Characteristics of Respondents

1. Please indicate your age in appropriate ranges

☐ 15-24 years ☐ 25-34 years ☐ 35-44 years ☐ 44 and above

2. Marital Status

☐ Married ☐ Divorced ☐ Widowed

4. Which of the following is your highest level of formal education?

☐ Primary ☐ Secondary ☐ Tertiary education ☐ No formal education

5. Which of the following is the respondent's husband highest level of formal education?

Primary ☐ Secondary ☐ Tertiary education ☐ No formal education ☐

5. Occupational Status

Civil servant ☐ Self employed (farmer, Businessman etc) ☐ Not employed ☐ Student ☐

6. Monthly income

< 50 000 ☐ 50-100,000 ☐ 101-150,000 ☐ >150,000 ☐

7. How many children do you have?

None ☐ 1-4 ☐ 5-8 ☐ 9 and above ☐

SECTION B

Nutritional Knowledge

Tick to the answer that best suit you

1. Are you aware about the importance of Nutrition?

Yes ☐ No ☐

2. Are you aware of consumption of balanced diet?

Yes ☐ No ☐

3. Are you aware of the main food nutrients that you should consume daily (balanced diet); i.e. proteins, carbohydrates, fats, minerals and vitamins?

Yes ☐ No ☐

4. Are you aware of the sources of main food groups that you should eat 3 main meals a day?

☐ Yes ☐ No

5. Are you aware about the risk of overfeeding?

☐ Yes ☐ No

6. Are you aware of potentially harmful foods?

☐ Yes ☐ No

7. Are you aware of the risk of overweight?

☐ Yes ☐ No

8. Are you aware of the foods for growth and development?

☐ Yes ☐ No

9. Are you aware that the best source of information for nutrition is in the hospital?

☐ Yes ☐ No

10. Do you have Any Problems with your Appetite, like not feeling hungry or feeling hungry all the time? Yes ☐ No ☐

11. Have you tried to lose weight or control your weight by vomiting, taking diet, pills or laxatives, or not eating? Yes ☐ No ☐

SECTION C

Nutritional Attitude and Practice

Please tick ☐ to the answer that best suits you.

Practice always = PA

Do not practice = DP

Practice rarely = PR

1. How often do you eat food substances that contain carbohydrate like starchy foods?

☐ PA ☐ DP ☐ PR

2. How often do you eat bodybuilding foods like milk, egg, beans etc?

☐ PA ☐ DP ☐ PR

3. How often do you eat fats and oil food substance from plant source?

☐ PA ☐ DP ☐ PR

4. How often do you eat food substances that are rich in vitamins like fruits, vegetables, fortified milk, fortified margarine, eggs, liver and fish?

☐ PA ☐ DP ☐ PR

5. How often do you drink eight and more glasses of fluids daily?

☐ PA ☐ DP ☐ PR

6. How often do you plan and keep to a balance food menu?

☐ PA ☐ DP ☐ PR

7. How often do you eat food substances rich in fiber/roughages like skin of fruits, wheat and grain?

☐ PA ☐ DP ☐ PR

8. How often do you eat fried foods and other fattening foods or high calorie foods some of which are animal fats, butter and starchy foods?

☐ PA ☐ DP ☐ PR

9. How often do you exercise?

☐ PA ☐ DP ☐ PR

10. How often do you take vitamins and mineral supplements?

☐ PA ☐ DP ☐ PR

APPENDIX 2: INFORMED CONSENT FORM

Informed Consent Form

I am Abdullahi Muhammad Umar, a final year student of M.Sc Nutritional Biochemistry, Department of Biochemistry, Faculty of Basic Medical Sciences, Bayero University Kano. I am conducting a research on‘‘Association of Angiotensin II type 1 receptor gene polymorphism with obesity among hypertensive patients attending hospitals in Kano metropolis”. This project will involve recruiting volunteers/participants from specialty clinics of Murtala Muhammad Specialist Hospital (MMSH), Sheik Muhammad Jidda Specialist Hospital and Sir Muhammad Sunusi specialist Hospital.

The aim of this study is to see how your response can help in determining ‘the relationship between Nutritional status and polymorphism of AT1R gene using questionnaire, anthropometric measurements, blood samples collection and analysis.

It is your right to either accept or refuse to participate in this study. Your participation in this study will not cause you any harm.

All your information will be strictly confidential. If you have any question or enquiry, you can contact me via my phone number +2348164846220 and/or via my email address: amumagashi@gmail.com.

CONSENT: Now that the study has been explained to me, I fully understand the consent of the study process. I will be willing to take part in the study.

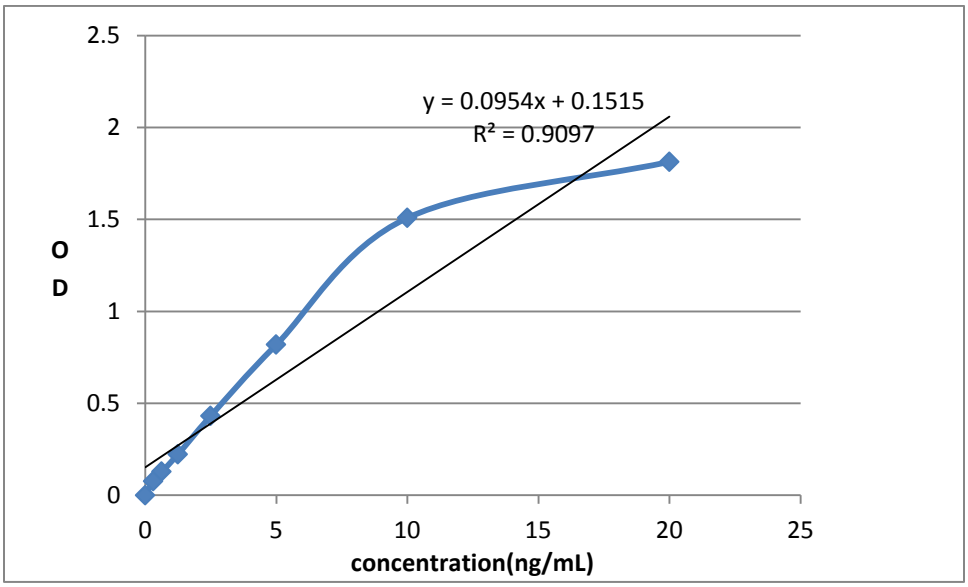
Participant Name _____

Participant signature/date _____

APPENDIX III: 24 HOURS DIETERY RECALL

Question number	Food groups	Examples	Yes=1 No= 0
1	Cereals	maize, rice, wheat, sorghum, millet	
2	White roots and tubers	Irish potato, yam, sweet potato	
3	Vitamin A rich vegetables & tubers	pumpkin, carrot, pepper	
4	Dark green leafy vegetables	Spinach	
5	Other vegetables	tomato, onion, red pepper	
6	Vitamin A rich fruits	mango, papaya, etc & their fruit juice	
7	Other fruits	wild fruits and their juice	
8	Organ meat	liver, kidney, heart	
9	Flesh meats	beef, lamb, goat, chicken,	
10	Eggs	eggs from chicken or any other egg	
11	Fish and seafood	fresh or dried fish	
12	Legumes, nuts and seeds	beans, peas, lentils, nuts, seeds	
13	Milk and milk products	milk, cheese, yoghurt, or other milk products	
14	Oils and fats	Oil added to food or used for cooking	
15	Sweets	sugar, honey, sweetened juice drinks, cookies, cakes, chocolate	
16	Spices, condiments & beverages	black pepper, salt, coffee, tea,	

APPENDIX IV


y = 0.0954x + 0.1515
R² = 0.9097
OD
concentration(ng/mL)
2.5
2
1.5
1
0.5
0
0
5
10
15
20
25

APPENDIX V

DETERMINATION OF SERUM ANGIOTENSIN II LEVEL

Principle

This ELISA kit uses Competitive-ELISA as the method. The micro titer plate provided in this kit has been pre-coated with Ang-II. During the reaction, Ang-II in the sample or standard competes with a fixed amount of Ang-II on the solid phase supporter for sites on the Biotinylated Detection Ab specific to Ang-II. Excess conjugate and unbound sample or standard are washed from the plate, and Avidin conjugated to Horseradish Peroxidase (HRP) is added to each microplate well and incubated. Then a TMB substrate solution is added to each well. The enzyme-substrate reaction is terminated by the addition of a sulphuric acid solution and the color change is measured spectrophotometrically at a wavelength of 450 nm ± 2 nm. The concentration of Ang-II in the samples is then determined by comparing the OD of the samples to the standard curve.

PROCEDURE

All reagents and samples were maintained at room temperature before the commencement of the experiment. The sampleswere centrifuged again after thawing before the assay. All the reagents were mixed thoroughly by gently swirling before pipetting.

50µl of Standard were added in each well. The blank well is added with Reference Standard & Sample Diluent. Immediately 50 µl of Biotinylated Detection Ab working solution were to each well and cover with a Plate sealer. And Incubated for 45minutes at 37°C. (Solutions are added to

the bottom of micro ELISA plate well, to avoid inside wall touching and foaming as possible.) Each well were aspirated and wash three times by filling each well with Wash Buffer (approximately 350µl) using automated washer. After the last wash, remaining Wash Buffer was removed by decantation. The plate and pat were inverted against thick clean absorbent paper. 100µl of HRP Conjugate solution were added to each well, covered with a new Plate sealer and Incubated for 30 minutes at 37°C.The aspiration/wash process were repeated for five times as conducted in step 2. 90µl of Substrate Solution were added to each well, covered with a new Plate sealer and Incubated for about 15 minutes at 37°C. The reaction time can be shortened or extended according to the actual color change, but not more than 30minutes. When apparent gradient appeared in standard wells, the reaction can be terminated. 50µl of Stop Solution were added to each well. Color turn to yellow immediately. The adding order of stop solution should be as the same as the substrate solution. The optical density (OD value) of each well were determine at once, using a microplate reader set at 450 nm. You should open the microplate reader ahead, preheat the instrument, and set the testing parameters. After experiment, the unused reagents were taken back to the refrigerator according to the specified storage temperature respectively.

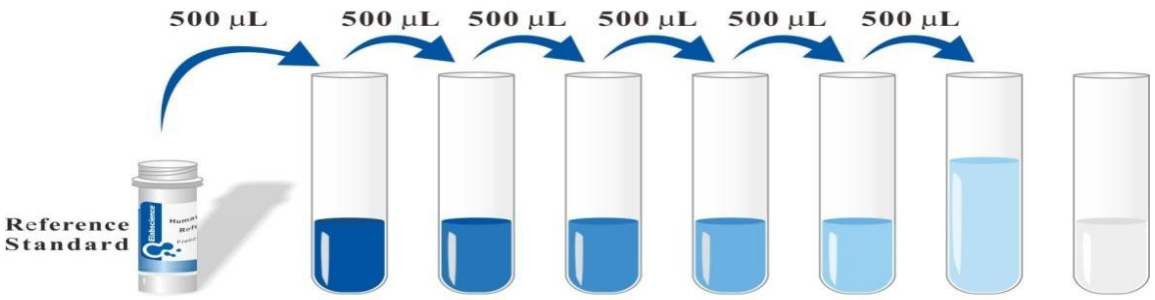
APPENDIX VI

Reagent Preparation

Bring all reagents to room temperature (18-25^{0}C) before use.

Wash Buffer - Dilute 30mL of Concentrated wash Buffer into 750mL of wash with deionized or distilled water. Put unused solution back at 4^{0}C. If crystals have formed in the concentrate, you can warm it with 40^{0}C water bath (Heating temperature should not exceed 50^{0}C) and mix it gently until the crystals have completely dissolved. The solution should be cooled to room temperature before use.

Standard – prepare standard within 15 minutes before use. Centrifuge at 10,000xg for 1 minute, and reconstitute the standard with 1.0mL of Reference standard & sample diluent. Tighten the lid, let it stand for 10 minutes and turn it upside down for several times. After it dissolve fully, mix it thoroughly with a pipette. This reconstitute produces a stock solution of 1000pg/mL. Then make serial dilutions as needed (making serial dilution in the wells directly is not permitted). The recommended concentrations are as follows: 1000, 500, 250, 125, 62.5, 31.25, 15.63, 0 pg/mL. if you want to make standard solution at the concentration of 500pg/mL, you should take o.5mL standard at 1000pg/mL, add it to an EP tube with 0.5mL Reference standard & sample diluent , and mix it. Procedures to prepare the remained concentrations are all the same. The undiluted standard serves as the highest standard (1000pg/mL). The Reference standard &Sample diluent serves as the zero (0pg/mL).



1	2	3	4	5	6	7	8	tube
1000	500	250	125	62.5	31.25	15.63	0	pg/mL

Biotinylated Detection Ab – Calculate the required amount before experiment (50µL/well). In actual preparation, you should prepare 100~200µL more. Centrifuge the stock tube before use, dilute the concentrated Biotinylated Detection Ab to the working concentration using Biotinylated Detection Ab Diluent (1:100).

Concentrated HRP Conjugate – Calculate the required amount before experiment (100µL/well). In actual preparation, you should prepare 100~200µL more. Dilute the Concentrated HRP Conjugate to the working concentration using HRP Conjugate Diluent (1:100).

Substrate Reagent: As it is sensitive to light and contaminants, so you shouldn't open the vial until you need it! The needed dosage of the reagent can be aspirated with sterilized tips and the unused residual reagent shouldn't be dumped back into the vial again.

Washing Procedure:

1. **Automated Washer:** Add 350μL wash buffer into each well, the interval between injection and suction should be set about 60s.

2. **Manual wash:** Add 350μLWash Buffer into each well, soak it for 1~2minutes. After the last wash, decant any remaining Wash Buffer by inverting the plate and blotting it dry by rapping it firmly against clean and toweling absorbent paper on a hard surface.