A PROJECT

ON

EVALUATION OF BLOOD PRESSURE AND GLUCOSE LEVELS AS RISK FACTORS OF METABOLIC SYNDROME AMONG PHYSIOLOGY STUDENTS IN UNIVERSITY OF BENIN

BY

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DEDICATION

I dedicates this work to God almighty for keeping and guiding me through my academic path

ABSTRACT

The objective of this experiment was to assess blood pressure and glucose levels as risk factors for metabolic syndrome (MetS) among physiology students at the University of Benin (UNIBEN), Edo, Nigeria. The study involved 50 students, comprising 20 males and 30 females, aged 16 to 26 years. All participants voluntarily completed questionnaires and provided written informed consent before the examination. They were required to fast for at least 12 hours before the tests. Blood pressure was measured using an automated sphygmomanometer, and fasting blood glucose levels were assessed with a glucometer, following standard protocols. Data analysis was performed using GraphPad Prism statistical package version 8.1, with descriptive statistics presented as percentages. A significance level of $P<0.05$ was applied. The average age of the participants was 21.5 years.Results showed that 86% of the students had normal glucose levels, while 14% fell within the MetS range. For systolic blood pressure, 90% were within the normal range, and 10% were within the MetS range. Regarding diastolic blood pressure, 96% were in the normal range, with 4% in the MetS range. The correlation between blood pressure levels and a family history of diabetes suggested a 1.047 risk of MetS, but this was not statistically significant ($P>0.05$). Other family histories, such as hypertension and MetS, also showed no significant association ($P>0.05$), and there was no increased risk of developing metabolic syndrome.In conclusion, this study indicated that some physiology students at UNIBEN are at risk of developing MetS based on their blood pressure, glucose levels, and family history. Therefore, these students should take proactive steps to maintain good health.

TABLE OF CONTENT

LIST OF FIGURES

LIST OF TABLES

CHAPTER ONE

1.0 INTRODUCTION

Metabolic syndrome refers to a group of risk factors for cardiovascular disease and type 2 diabetes that tend to occur together more frequently than by chance. In epidemiology, a risk factor is a variable linked to an increased likelihood of disease or infection, but these are correlational rather than causal, as correlation doesn't imply causation. The risk factors include high blood pressure, dyslipidemia (elevated triglycerides and lowered HDL cholesterol), elevated fasting glucose, and central obesity (Alberti *et al.,* 2009). Metabolic syndrome encompasses a set of metabolic abnormalities that significantly increase the risk of cardiovascular disease, type 2 diabetes, and other health issues. It's characterized by central obesity, insulin resistance, dyslipidemia, and hypertension. The global prevalence of this syndrome is on the rise, placing a heavy strain on healthcare systems (Grundy, 2016). It is most commonly seen in obese individuals, with central obesity, marked by excessive abdominal fat, being a key factor. Visceral fat accumulation contributes to insulin resistance, where cells don't respond well to insulin, leading to poor glucose regulation, hyperglycemia, and eventually diabetes. Dyslipidemia, featuring elevated triglycerides, reduced HDL cholesterol, and increased LDL cholesterol, also plays a major role in metabolic syndrome, creating an inflammatory environment that promotes atherosclerosis and cardiovascular

disease. Hypertension, often found alongside these metabolic disturbances, is driven by insulin resistance and dyslipidemia, which contribute to arterial stiffness and high blood pressure, raising the risk of cardiovascular incidents. Early detection and comprehensive management of metabolic syndrome are crucial due to its significant impact on health and quality of life. Lifestyle changes, such as improved diet, regular exercise, and weight control, are central to treatment, though medications targeting specific aspects of the syndrome may also be necessary to reduce cardiovascular risk and enhance metabolic health. While various diagnostic criteria have been proposed over the years, no single set of criteria is universally required, though waist circumference remains a valuable initial screening tool.

1.1. JUSTIFICATION OF STUDY

The metabolic syndrome is a major global public health burden that raises healthcare expenditures, morbidity, and mortality. Assessing blood pressure and blood glucose as risk variables is critical to comprehending the underlying mechanisms and developing successful preventative measures.

Researchers can determine crucial windows for intervention and clarify causal pathways by monitoring changes over time. The study's conclusions may have ramifications for future research initiatives, public health policies, and clinical

practice. They can help in the identification and successful management of those at high risk of metabolic syndrome by providing information for the creation of focused therapies, screening protocols, and risk prediction models.

1.2. AIMS OF STUDY

The aim of this study is to evaluate blood pressure and glucose levels as risk factors of metabolic syndrome physiology students in the university of Benin.

1.3. RESEARCH QUESTIONS

- What percentage of physiology students have blood glucose and blood pressure levels in the range for risk of metabolic syndrome?
- Is there any association between blood glucose levels and family history of hypertension, diabetes and metabolic syndrome?
- Is there any association between blood pressure levels and family history of hypertension, diabetes and metabolic syndrome?

1.4. SPECIFIC OBJECTIVES OF STUDY

- Determine the prevalence of blood glucose and high blood pressure within the range of metabolic syndrome in the study population.

- Explore potential effects of family history of hypertension, diabetes and metabolic syndrome on blood glucose levels.
- Explore potential effects of family history of hypertension, diabetes and metabolic syndrome on blood pressure levels.

CHAPTER TWO

2.0. LITERATURE REVIEW

2.0.1. Definition of Metabolic Syndrome

A syndrome, according to the British Medical Association Illustrated Medical Dictionary (2002), is a group of related medical indications and symptoms that are frequently connected to a certain illness or condition. There are situations when the term "syndrome" and the pathophysiological mechanism of a disease may be so closely related that it becomes challenging to distinguish between the two. A collection of metabolic variables have been combined to form the metabolic syndrome (MetS).

The metabolic syndrome is defined as a clustering of metabolic abnormalities that include central obesity, insulin resistance, hypertriglyceridemia, hypercholesterolemia, hypertension, and reduced high-density lipoprotein (HDL)-cholesterol concentrations (Dommermuth and Ewing, 2018). The presence of three abnormal findings out of five components qualifies a person for metabolic syndrome. The metabolic syndrome predicts cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM). A person with the metabolic syndrome will always be afflicted by its components, which is the reason that management must be sustained over a very long time. The risk factors usually included raised blood pressure, dyslipidemia (raised triglycerides and low HDL cholesterol), raised

fasting plasma glucose, raised fasting serum insulin and obesity (central obesity defined as a large waist circumference or waist–hip ratio or body-mass index) (Peter *et al.,* 2019*).* Although it is unclear whether there is a unifying pathogenic mechanism that could decipher the pathophysiology of the metabolic syndrome, it is highly likely that abdominal obesity and insulin resistance could play a central role in promoting the development of the metabolic syndrome (Genser *et al.,* 2016).

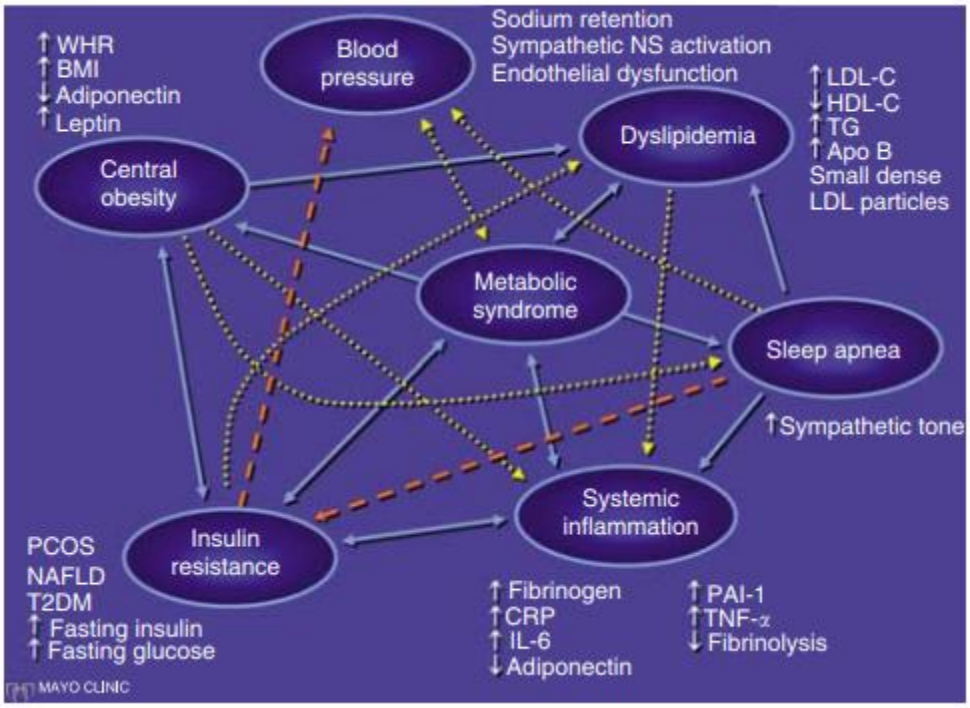

Figure 1. Metabolic Syndrome and each component likely to have an impact on its prevalence and pathophysiology (Batsis *et al.,* 2007). Apo, apolipoprotein; BMI, body mass index; CRP, C-reactive protein; HDL-C, high-density lipoprotein cholesterol; IL, interleukin; LDL-C, low-density lipoprotein cholesterol; NAFLD, non-alcoholic fatty liver disease; NS, nervous system; PAI-1, plasminogen-activating inhibitor-1; PCOS, polycystic ovarian syndrome; T2DM, type 2 diabetes mellitus; TG, triglyceride; TNF, tumor necrosis factor; WHR, waist–hip ratio.

2.1. PREVALENCE OF METABOLIC SYNDROME

Despite considerable research and clinical acceptance of the metabolic syndrome, the various cutoffs for its diagnostic components have varied. The etiology of the syndrome is multifactorial, involving both genetic and environmental factors that interact together (Corella and Ordovas, 2004). The frequency of the syndrome is lower in black than in white men, which could be attributed to less frequent atherogenic dyslipidemia observed in black men, as well as to the lower prevalence of obesity (23%) compared with Mexican (31%) or white (31%) American men, the prevalence of metabolic syndrome increased significantly over the past decade among both adults and adolescents (Kolovou *et al*., 2007). Fortunately, the National Health and Nutrition Examination Survey (NHANES) released recent data demonstrating declining numbers of the disease with 24% in men and 22% in women (Swarup *et al*., 2020).

2.2. COMPONENT OF METABOLIC SYNDROME

2.2.1. Blood Glucose level

The quantity of glucose (sugar) in your blood is known as your blood glucose level. Less than 100 mg/deciliter (5.55 mmol/liter) was recently established as the typical fasting plasma glucose level. It is unknown if greater fasting plasma glucose levels within this range in young adults independently predict type 2 diabetes. An impaired fasting plasma glucose level is now considered to include the range of 100 to 109 mg per deciliter (5.55 to 6.05 mmol per liter) (Genuth *et al., 2*003). The concept that persons with fasting plasma glucose levels of 100 to 109 mg per deciliter are at increased risk for the development of type 2 diabetes, as compared with those with fasting plasma glucose levels of less than 100 mg per deciliter, is substantiated by data (Shyong *et al.,* 2004). An impaired fasting plasma glucose level is a known risk factor for diabetes, along with other traditional risk factors such as a family history, sedentary lifestyle, central adiposity, dyslipidemia, and hypertension (Stern *et al.,* 2002). The discovery that a high-normal fasting plasma glucose level is a risk factor for type 2 diabetes could be useful in identifying young, healthy males who should be the target of preventative measures. In reality, a number of tactics, such as changing one's lifestyle and using drugs like orlistat, thiazolidinediones, metformin, and acarbose, have been shown to be effective

therapies that can postpone the onset of diabetes in certain populations that have traditional risk factors for the condition.

2.2.2. Blood Pressure

Blood pressure (BP) refers to the force exerted by your blood against the walls of your arteries as it circulates through your body. High blood pressure, also known as hypertension, is a long-term medical condition in which the blood pressure in the arteries is persistently elevated (Naish and Court, 2014). Red blood cells (RBCs), white blood cells (leukocytes), platelets (fragments of cells), and plasma (a liquid portion of the blood containing various components) make up around 7% of an individual's body weight. There are two primary components to blood pressure: a stable component linked to systemic vascular resistance and mostly associated with mean and diastolic blood pressure (DBP), and a pulsatile component connected to arterial stiffness and predominantly associated with pulse pressure or systolic blood pressure (SBP). In patients with impaired left ventricular ejection fraction (LVEF), both SBP and pulse pressure are primarily dependent on left ventricular stroke volume, while mean BP and DBP are highly dependent on total blood volume and the degree of peripheral vasodilatation (Tartiere *et al.,* 2006). High blood pressure (BP) is among the most important modifiable risk factors for cardiovascular disease and death (Forouzanfar *et al.,* 2017). Systolic blood pressure (SBP) of at least 110 mmHg has been related to multiple

cardiovascular and renal outcomes, including ischemic heart disease, cerebrovascular disease, and chronic kidney disease (Wright *et al.,* 2002). The burden of SBP of at least 110 mm Hg remains high despite the availability of preventive interventions and low-cost, effective antihypertensive medications (Kearney *et al.,* 2004). One of the main risk factors for heart disease, stroke, and mortality is hypertension. A significant segment of the global populace is impacted by it, and it continues to receive insufficient diagnosis and treatment. Prolonged hypertension induces diastolic dysfunction and left ventricular hypertrophy, which increases myocardial rigidity and makes the heart less responsive to changes in sympathetic tone, preload, and afterload.

2.2.3. Obesity

Obesity is defined as an excess amount of body fat. A common way to estimate body fat is by using body mass index (BMI), which is calculated by dividing weight in kilograms by height in meters squared. Clinically, a BMI between 25 and 29 kg/m² is considered overweight, while a BMI of 30 kg/m² or higher is classified as obesity. In terms of body fat percentage, obesity is defined as 25% or more in men and 35% or more in women. Measuring waist circumference is the most practical way to assess obesity in clinical settings, as excess abdominal fat is closely linked to metabolic risk factors (Grundy, 2004). Abdominal obesity is identified when the waist circumference is 102 cm or more in men and 88 cm or

more in women. The rising prevalence of obesity is largely due to an abundance of food and increasingly sedentary work environments, both of which are influenced by technological advancements. Obesity also reflects the nature of a free society, where there is a wide range of food options and job opportunities. A public health strategy aimed at combating obesity that restricts personal choices would likely be unacceptable in such a society. The primary physical consequence of obesity is atherosclerotic cardiovascular disease (ASCVD) (Grundy *et al*., 2004). A collection of risk factors that contribute to ASCVD is referred to as metabolic syndrome. Obesity is a major risk factor for both ASCVD and type 2 diabetes. Recent studies suggest that having metabolic syndrome significantly raises the risk of developing ASCVD and type 2 diabetes. Individuals with metabolic syndrome have at least twice the risk of ASCVD compared to those without it, and their risk of developing type 2 diabetes increases about fivefold for both men and women (Grundy et al., 2004). The risk is particularly high in those with impaired fasting glucose. The connection between metabolic risk factors and ASCVD development is complex and not fully understood. Central fat accumulation is linked to insulin resistance, while fat distributed peripherally is less metabolically significant (Engin, 2017).

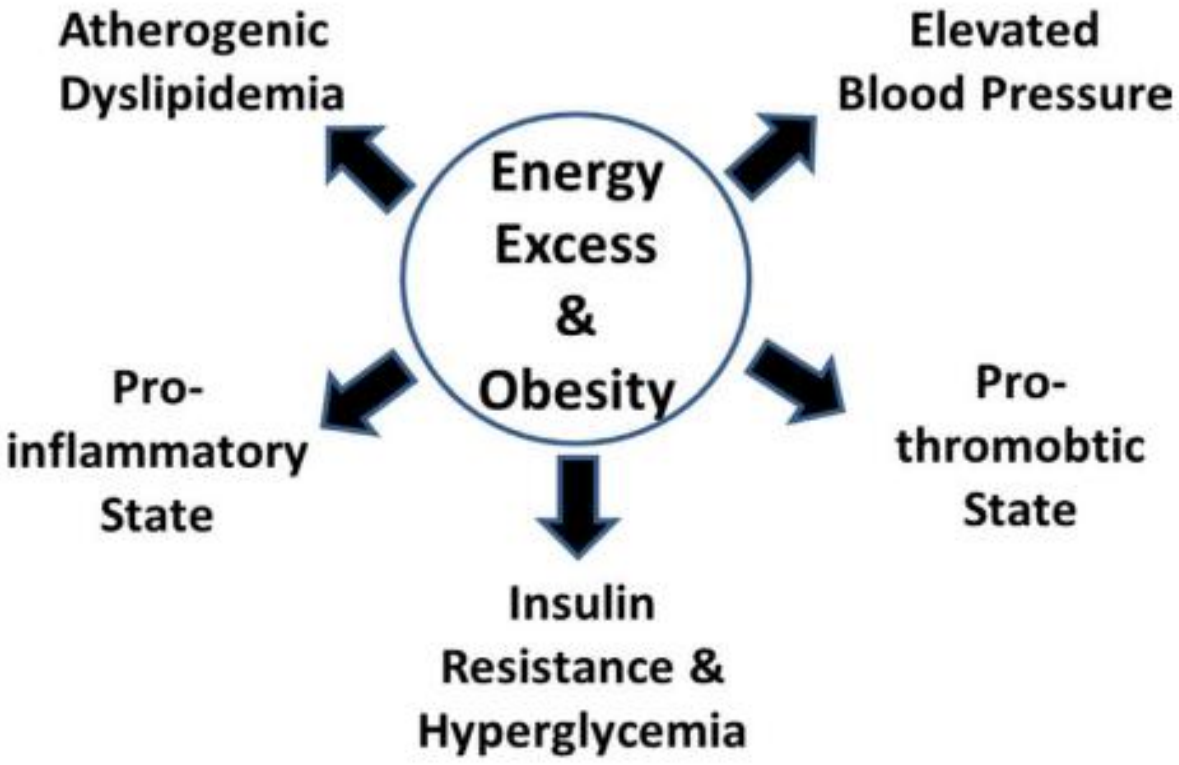

Figure 2. Relationships between energy excess/obesity and risk factors of the metabolic syndrome. Available evidence indicates that excess energy intake and concomitant obesity are major causes of all the metabolic risk factors (Grundy, 2016).

2.2.4. Dyslipidemia

All all, dyslipidaemias are among the chronic illnesses that are most frequently identified and treated. According to Berberich and Hegele (2022) they are traditionally defined by abnormal serum levels of triglycerides, cholesterol, or both. They may also involve aberrant levels of associated lipoprotein species. The imbalance of lipids, including cholesterol, triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein (HDL), is referred to as dyslipidaemia, also known as hypercholesterolaemia. This disorder can be inherited, brought on by food, tobacco use, or both. It can result in significant problems from cardiovascular disease (Pappan and Rehman, 2024). An additional cause of dyslipidaemia may be inherited diseases. The majority of cases of familial hypercholesterolaemia in LDL receptors, which raises LDL-C levels, are caused by autosomal dominant mutations (Defesche *et al*., 2017). The frequency of dyslipidaemia rises with advancing years. The main instrument used to assess dyslipidaemia is a lipid panel measured during fasting that includes total cholesterol, LDL, HDL, and triglycerides.

Table 1. Biochemical levels for dyslipidemia in adults >18 years of age (Mach *et al*., 2020).

	LDL-C	**TG**	**HDL-C**
Mild-to-moderate deviation			
Levels	3.4-4.9 mmol/L	2-9.9 mmol/L	0.7-0.9 mmol/L
	130-194 mg/dL	175-885 mg/dL	25-35 mg/dL
Etiology	Polygenic predisposition		
	plus, secondary factors		
Severe deviation			
Levels	≥ 5.0 mmol/L	≥ 10 mmol/L	< 0.7 mmol/L
	≥ 194 mg/dL	≥ 885 mg/dL	< 25 mg/dL
Etiology	Monogenic disorders and/or		
	marked polygenic predisposition plus		
	secondary factors		

Elevated LDL-C is a causal risk factor for CVD (Ference *et al*., 2017) and lowering LDL-C concentrations is the primary target for treatment and prevention of CVD. Elevated TG, and especially TRL, are also treatment targets recommended in guidelines for the management of dyslipidemia (Mach *et al*., 2020).

2.3. DIAGNOSTIC CRITERIA

Table 2. The 'harmonized' metabolic syndrome: criteria for clinical diagnosis (Borges *et al*., 2010).

Measure	**Categorical cut-points**
Elevated waist circumference	Population- and country-specific definitions
Elevated triglycerides	≥150 mg/dL (1.7 mmol/L)
Drug treatment for elevated triglycerides is an alternative indicator	
Reduced HDL cholesterol	Males: <40 mg/dL (1.0 mmol/L)
Drug treatment for reduced HDL-C is an alternative indicator	Females: <50 mg/dL (1.3 mmol/L)
Elevated blood pressure	Systolic≥130 and/or
Antihypertensive drug treatment in patients with a history of hypertension is an alternative indicator	Diastolic≥85 mmHg
Elevated fasting plasma glucose	≥100 mg/dL (≥5.6 mmol/L)
Drug treatment of elevated glucose is an alternative criterion	

2.4. PATHOPHYSIOLOGY

In addition to genetic and epigenetic factors (Fathi, 2018), some lifestyle and environmental factors such as overeating and lack of physical activity have been identified as major contributors to the development of MetS (Fahed *et al*., 2022).

2.4.1. Insulin Resistance and Hyperinsulinemia

The primary factors driving the development of metabolic syndrome are insulin resistance and hyperinsulinemia (Reaven, 2008). Over 30 years ago, hyperinsulinemia and insulin resistance were identified as key contributors to high blood pressure and elevated blood glucose levels seen in obesity and metabolic syndrome. Short-term studies in animals and humans have further shown that hyperinsulinemia may increase sympathetic nervous system (SNS) activity and renal sodium retention, which, if prolonged, can lead to higher blood pressure. The metabolic effects of insulin resistance, such as hyperglycemia and dyslipidemia, can work together with elevated blood pressure to damage blood vessels and kidneys, worsening hypertension and related cardiovascular and kidney injuries (Alexandre *et al*., 2020). Although serum insulin wasn't measured in this study, it is well established that hyperinsulinemia, along with inflammation in insulin-sensitive tissues, and insulin resistance (IR), are the main metabolic issues in obese patients (Maffeis and Morandi, 2018). Importantly, hyperinsulinemia and IR are more related to the distribution of body fat rather than its amount. In healthy

individuals, hyperinsulinemia temporarily increases sympathetic activity, but this is balanced by reduced peripheral vascular resistance. However, in cases of chronic hyperinsulinemia, this reduction in resistance may not occur to the same extent. Studies in both animals and humans have demonstrated that chronic hyperinsulinemia and inflammation lead to increased sympathetic activity, promoting insulin resistance and raising peripheral vascular resistance (Greenfield *et al*., 2009).

2.4.2. Adipose Tissue Dysfunction

Aside from being a thermoregulator and lipid storage facility, the recently discovered endocrine function of the adipose tissue provides additional mechanistic understandings to the development of MetS (Mohamed-Ali *et al*., 1998). The various adipokines released include hormones (e.g., leptin, adiponectin), peptides (e.g., angiotensinogen, apelin, resistin, and plasminogen activator inhibitor (PAI)-1), and inflammatory cytokines (e.g., interleukin (IL)-6, tumor necrosis factor α (TNFα), visfatin, omentin, and chemerin), all of which play a major role in the pathophysiology of insulin resistance and MetS (Trayhurn and Wood, 2004). Leptin is directly proportional to obesity. When body energy stores are adequate, leptin suppresses food intake and stimulates energy expenditure while also controlling glucose homeostasis and insulin sensitivity (Berglund *et al*., 2012). the adipose tissue produces the peptide angiotensin II (Ang II) after

activation of angiotensin-converting enzyme. Plasma Ang II levels were shown to be increased in obesity and insulin resistance (Saiki *et al*., 2009). The peptide exerts its pathogenic effects through activation of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase, which increases the production of reactive oxygen species (ROS) (Lassegue *et al*., 2001). ROS has multiple pleiotropic effects, including endothelial injury, platelet aggregation, oxidation of LDL and expression of lipoprotein receptor-1 (LOX-1) on vascular smooth muscle cells (VSMCs) and endothelium. Together, RAS, LOX-1, and ROS form a positive feedback loop and induce a vicious cycle of endothelial dysfunction, inflammation, and fibroblast proliferation, leading to the progression of dyslipidemia, T2DM, hypertension, vasculopathies, and CVDs (Mehta and Griendling, 2017).

2.4.3. Inflammation Pathway

The various pathogenic pathways contributing to the development of MetS culminate in a pro-inflammatory state that explains the elevation in various inflammatory markers such as interleukin-6 (IL-6), C-reactive protein (CRP), and tumor necrosis factor- α (TNFα) seen in individuals with MetS (Kopp *et al.,* 2003). Insulin resistance and obesity-induced systemic oxidant stress activates downstream inflammatory cascades, leading to tissue fibrosis, atherogenesis, and subsequently CVDs (Hotamisligil, 2006).

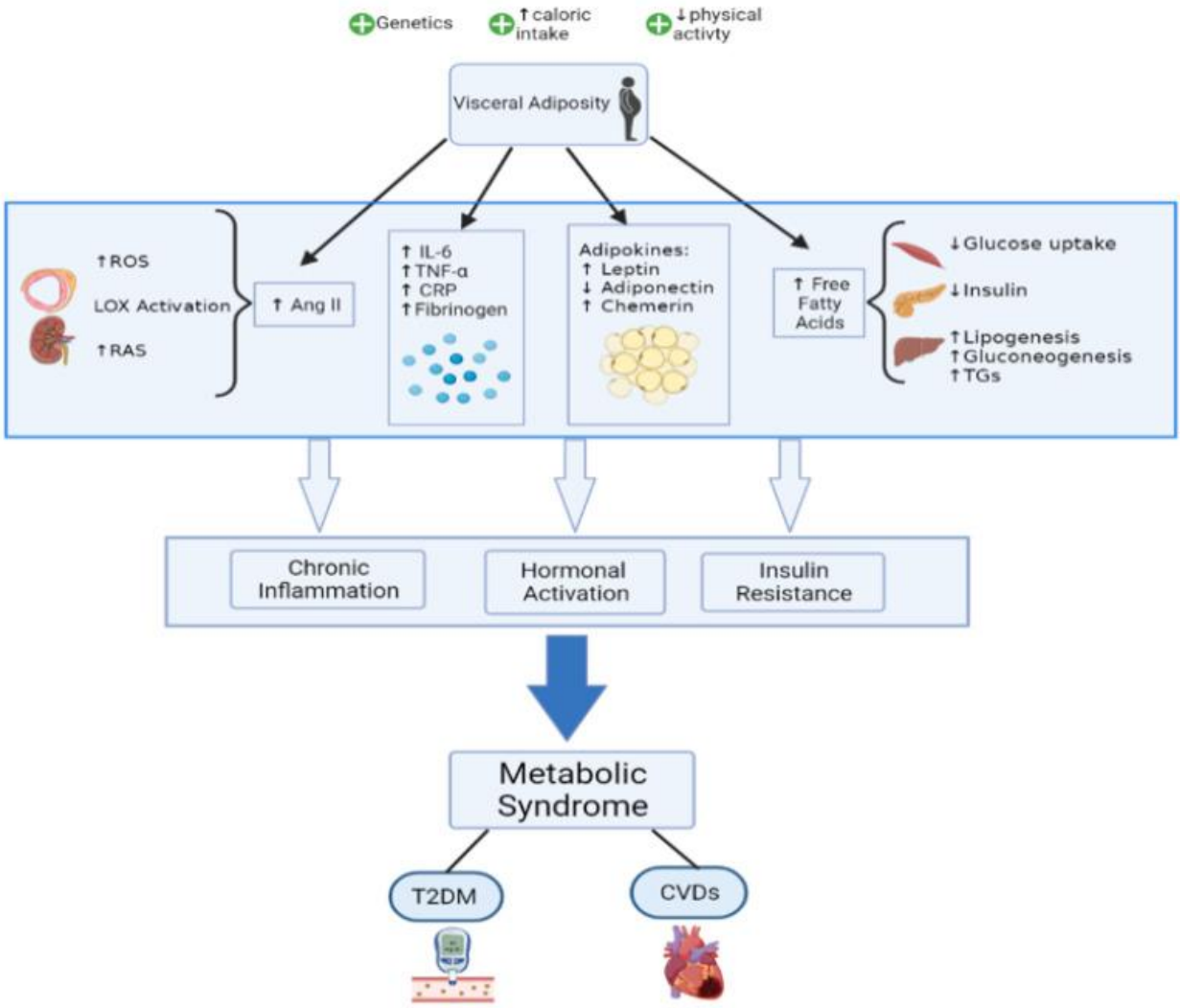

Figure 3. Mechanisms highlighting MetS pathophysiology (Fahed *et al.,* 2022).

2.5. MANAGEMENT AND TREATMENT

The treatment of MetS requires targeting the most prevalent cause of it, namely a sedentary lifestyle along with an inappropriate diet. The treatment also requires medical management of dyslipidemia, hypertension, and obesity (Batsis *et al*., 2007). Thus, treatment regimens include lifestyle modification, including exercise and dietary modifications in efforts to lose weight, pharmacological and surgical interventions, and managing risk factors in attempts to reduce the risk of developing type 2 diabetes mellites or cardiovascular disease (Batsis *et al*., 2007).

2.5.1. Lifestyle modifications

1. Exercise:

Physical activity has clearly been shown to improve glucose tolerance, improve insulin sensitivity, and reduce the risk of CV disease (Wannamethee and Shaper, 2001). Reductions in adipose tissue, particularly visceral fat, may mediate insulin sensitivity following weight loss. Regular exercise is known to improve other metabolic variables such as insulin and leptin levels in patients who may be overweight or obese (Frank *et al.,* 2005). Both exercise-induced weight loss and exercise without weight loss have been proven to reduce abdominal fat, itself a predictor of insulin resistance and dyslipidemia (Batsis *et al*., 2007).

2. Diet:

Caloric restriction in MetS patients who are obese or overweight should be a cardinal focus of dietary therapy. Dietary modification is a key component to any weight loss program. The influence of diet on CV function has been well delineated (Lichtenstein *et al.,* 2006). There is increasing evidence to show that specific composition of macro nutrients may positively affect metabolic profile. For this, preliminary data demonstrated that the Mediterranean diet may be superior to the standard low-fat or very low-fat diets (Kris-Etherton *et al.,* 2001) Characteristics of the diet include the consumption of a large amount of olive oil, wheat, grapes, and their derivatives.

Other treatment therapy includes pharmacological management like insulin sensitizers, biguanides, Orlistat, Sibutramine among others (Batsis *et al*., 2007).

2.6. CONCLUSION

In conclusion, the evaluation of blood glucose and blood pressure as risk factors of metabolic syndrome underscores their pivotal roles in assessing and managing this complex condition. Monitoring these parameters is crucial for early detection, intervention, and prevention of metabolic syndrome, thus highlighting the importance of comprehensive healthcare strategies aimed at addressing these key factors.

CHAPTER THREE

3.0. MATERIALS AND METHODS

3.1. MATERIALS

- Latex gloves
- Cotton wool
- Methylated spirit
- Glucometer
- Glucometer strip
- Lancets
- Automated Sphygmomanometer
- Questionnaire
- Consent form

3.2. STUDY AREA

The study was carried out at physiology laboratory in the University of Benin located at Ovia Northeast local government area Benin city, Edo state.

3.3. INCLUSION CRITERIA

Physiology student currently enrolled at the University of Benin.

Willingness to participate in the study.

3.4. EXCLUSION CRITERIA

Students who are unable to understand the study requirements or provide informed consent due to impairments or language barrier.

- The presence of chronic diseases or illness' known to alter heart rhythm.
- Students currently on regular medication, especially any that alters heart rhythm.
- Female students who are pregnant or breastfeeding.

3.5. STUDY DESIGN

The study enrolled 50 students (20 males and 30 females). All participants are physiology students at University of Benin, Benin city, Edo. The selection criteria were; interest to participate in the study, age of 16–26 years. Sociodemographic characteristics, past medical history and clinical examination were collected guided by a questionnaire.

3.6. MEASUREMENT OF PARAMETERS

1. Fasting Blood Glucose Testing

The individual must fast for at least 12 hours before the test. A blood sample is taken from a fingertip using a small prick. Blood can also be drawn from alternative sites such as the earlobe, heel, forearm, or palm. These alternative sites provide results comparable to those from a finger prick, especially when fasting or two hours after a meal. Testing from alternate sites may be less painful but may require a deeper lancet. It’s important to consult the glucometer manufacturer to ensure the device supports alternate site testing. The tools needed for capillary blood glucose testing include a lancet to puncture the skin, a glucometer, and test strips. Modern glucometers, often "smart" devices, require a very small blood sample (between 0.3 and 1 microL).

Advantages: Less unpleasant than venipuncture, small blood sample, quick testing period, variety of alternative testing locations, clear display on glucometer.

Venipuncture is used to get venous blood, and the sample is then processed in a laboratory of commercial calibre that has undergone the necessary complex quality control inspections.

Disadvantages: Painful procedure, risk of local tissue damage, unsuitable for frequent specimen collection

Steps undertaken during blood glucose test with a glucometer according to Matthew *et al*. (2023) are outlined below;

1. Cleaned and dried the testing location. To reduce damage to the underlying bone, the side of the distal fingertips is the suggested testing site on the palm. It is not advisable to use the fifth finger because the tissue may not be deep enough to stop the injury. Additionally, since the thumb and first finger are more sensitive than the other fingers, they should be avoided.
2. Ready-made apparatus.
3. To reduce the possibility of causing bone damage, primed the lancet to no more than 2.0 mm.
4. Without coming into contact with the sensor tip, take the glucose testing strip out of its container. The glucometer then turned on by itself when I placed the glucose testing strip inside of it.
5. Firmly positioned the lancet at the location where the sample will be collected, then pulled the trigger to puncture the skin.
6. To help the blood flow, I gently applied downward pressure at the puncture site and wiped off the first drop of blood.

7. Touched the tip of the glucose testing strip to collect the second drop of blood when it develops.
8. Set the glucometer in place and wrapped clean cotton wool over the skin puncture site. To halt more bleeding from the puncture site, apply pressure.
9. Unless there were issues with the sample collection—such as an inadequate sample, a low battery, an incorrect code, or a machine timeout—the machine produced a result at this point. In the event that the glucometer reports an error, troubleshoot as necessary.
10. I changed the equipment in the storage bag container and cleaned my hands.

2. Blood Pressure Testing Procedures

Steps undertaken during blood pressure test with an automated sphygmomanometer according to Murakami and Rakotz, (2015) are outlined below.

1. Placed the cuff's air tube inside the automated sphygmomanometer.

2. Encircled the naked arm with the cuff, making that the inner side of the arm's bladder centre was positioned two to three centimetres above the elbow joint.

3. Secured the cuff using the fabric fastener.

4. Holding the cuff at about heart level, place the arm with the palm facing up.

5. Started the sphygmomanometer that was automated.

6. Hit the begin button. When a goal value was attained, the machine expelled air from the cuff and the display started to count down.

7. The heart symbol flashed on the display as the gadget sensed the pulse.

8. The device showed the heart rate, systolic and diastolic blood pressures, and the cuff pressure as it dropped.

9. The heart symbol on the display indicated the final blood pressure and pulse measurements once all the air had been released.

10. The device has a memory feature that saves every outcome and removes the prior one automatically. Holding down the memory button will cause the last reading to appear.

3.7. ETHICAL CONSIDERATION

Experimental protocols were carried out in accordance with the recommendations from the ethics and research committee of the University of Benin, Edo State, Nigeria.

3.8. STATISTICAL ANALYSIS

Data were subjected to statistical analysis using the Graph-pad statistic software and relevant statistical values were obtained.

Graph pad prism statistical package version 8.1 was used, and descriptive statistics was carried out and data were presented in percentage. Values of $P<0.05$ were considered significant.

CHAPTER FOUR

4.0 RESULT

Table 4.1: showing the percentage distribution of blood pressure parameters and glucose in metabolic syndrome

PARAMETERS	NORMAL VALUE (<100)	MetS VALUE (≥100)
Glucose level (mg/dL)	43 (86.00%)	7 (14.00%)
	NORMAL VALUE (<130)	MetS VALUE (≥130)
SBP (mmHg)	45 (90.00%)	5 (10.00%)
	NORMAL VALUE (<85)	MetS VALUE (≥85)
DBP (mmHg)	48 (96.00%)	2 (4.0%)

Table 4.2: Relating distribution of blood pressure level with *different family health histories of diabetes.*

SBP	Family history of diabetes	No family history of diabetes	Total	Statistical indices
130 mmHg below	9 (90.00%)	34 (89.47%)	43	$X^2 = 0.00235$, df =1
Above 130mmHg	1 (10.00%)	4 (10.53%)	5	P-value = 0.9613
Total	10	38		Risk = 1.047

+ Significant p value, * chi square test.

Table 4.3: Relating distribution of blood pressure level with *different family health histories of hypertension.*

SBP	Family history of hypertension	No family history of hypertension	Total	Statistical indices
130 mmHg below	18 (85.71%)	26 (96.30%)	44	$X^2 = 2.980$, df =1
Above 130mmHg	3 (14.29%)	1 (3.70%)	4	P-value = 0.0843
Total	21	27		Risk = 0.4942

+ Significant p value, * chi square test.

Table 4.4: Relating distribution of blood pressure level with *different health histories of high blood pressure.*

SBP	High blood pressure	No history of High blood pressure	Total	Statistical indices
130 mmHg below	4 (80.0%)	39 (90.70%)	43	$X^2 = 0.5493$, df =1
Above 130mmHg	1 (20.00%)	4 (9.30%)	5	P-value = 0.4586
Total	5	43		Risk = 0.4651

+ Significant p value, * chi square test.

Table 4.5: Relating distribution of blood pressure level with *different family health histories of metabolic syndrome.*

SBP	Family history of metabolic syndrome	No family history of metabolic syndrome	Total	Statistical indices
130 mmHg below	3 (75.0%)	39 (88.64%)	42	$X^2 = 0.6234$, df =1
Above 130mmHg	1 (25.00%)	5 (11.36%)	6	P-value = 0.4298
Total	4	44		Risk = 0.4286

+ Significant p value, * chi square test.

Table 4.6: Relating distribution of blood glucose level with *different family health histories of diabetes.*

Blood glucose level	Family history of diabetes	No family history of diabetes	Total	Statistical indices
100 mg/dl below	8 (80.00%)	32 (84.21%)	40	$X^2 = 0.1011$, df =1
Above 100 mg/dl	2 (20.00%)	6 (15.79%)	8	P-value = 0.7506
Total	10	38		Risk = 0.8000

+ Significant p value, * chi square test.

Table 4.7: Relating distribution of blood glucose level with *different family health histories of hypertension.*

Blood glucose level	Family history of hypertension	No family history of hypertension	Total	Statistical indices
100 mg/dl below	16 (76.19%)	24 (88.89%)	40	$X^2 = 1.371$, df =1
Above 100 mg/dl	5 (23.81%)	3 (11.11%)	8	P-value = 0.2416
Total	21	27		Risk = 0.6400

+ Significant p value, * chi square test.

Table 4.8: Relating distribution of blood glucose level with *different health histories of high blood pressure.*

Blood glucose level	High blood pressure	No history of High blood pressure	Total	Statistical indices
100 mg/dl below	2 (40.0%)	38 (88.37%)	40	$X^2 = 7.546$, df =1
Above 100 mg/dl	3 (60.00%)	5 (11.63%)	8	P-value = 0.0060
Total	5	43		Risk = 0.1333

Table 4.9: Relating distribution of blood glucose level with *different family health histories of metabolic syndrome.*

Blood glucose level	Family history of metabolic syndrome	No family history of metabolic syndrome	Total	Statistical indices
100 mg/dl below	4 (100.0%)	36 (81.82%)	40	$X^2 = 0.6234$, df =1
Above 100 mg/dl	0 (0.00%)	8 (18.18%)	8	P-value = 0.4298
Total	4	44		Risk = 0.4286

CHAPTER FIVE

5.0. DICUSSION

In this study, we evaluated blood pressure and blood glucose levels as risk factors of metabolic syndrome and assessed the impact of family history in physiology students. Results showed 43 (86%) of the students were within normal range and 7 (14.00%) were within MetS range after evaluation of glucose as risk factor of MetS, 45 (90%) of the students were within normal range and 5 (10.00%) were within MetS range after evaluation of systolic blood pressure as a risk factor, 48 (96%) of the students were within normal range and 2 (4.00%) were within MetS range after evaluation of diastolic blood pressure as a risk factor (Table 4.2). The prevalence of high blood pressure (within MetS range) increased in students with family history of diabetes with an increased risk of 1.047 and it is in accordance with the work of Moon *et al.* (2017) that concluded that in Korean population those with family history of diabetes were at increased risk of metabolic disorder (like high blood pressure) in young adults. Students with family history of hypertension have 0.4942 (49.42%) risk probability of developing high blood pressure within MetS range (Table 4.3) though there was no significant association of this with the different family history ($P>0.05$) which contradicts the work of Ranasinghe *et al*. (2015) that showed significant association between family history of hypertension and high blood pressure within range of metabolic syndrome in Sri Lankan adults

this can be due to very large difference in sample size as well as difference in diagnostic criteria for hypertension. Students with family history of metabolic syndrome had 0.4286 (42.86%) risk probability of developing high blood pressure within MetS range (Table 4.5). Although there is an 80% (R = 0.8) risk of falling within the Mets range of blood glucose level in those with family history of diabetes there is no significant correlation ($P<0.05$) (Table 4.6). There is a 64% (R = 0.64) risk of falling within the Mets range of blood glucose level in those with family history of hypertension (Table 4.7). There was no significant association between blood glucose and family history of metabolic syndrome (Table 4.9) which contradicts the work of Lipińska *et al*. (2014) who showed a positive correlation between fasting glucose levels in the family history of MetS. It may be due to certain limitations of this study, including a small sample size because of rigorous inclusion and exclusion criteria intended to obtain homogenous groups. There is grossly limited knowledge of family history among participants as well as no proper means of confirming family history information. The lifestyle of participants was not taken into consideration.

5.1 CONCLUSION

In conclusion, this study showed that some physiology students of UNIBEN are at risk of developing MetS due to their blood pressure and blood glucose measurements and family history and as such, should take steps towards maintaining good health.

References

1998 Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults—the Evidence Report. *National Institutes of Health. Obes Res*. **6**(2): 51 –209.

Aird, W. C. (2011). Discovery of the cardiovascular system: from Galen to William Harvey. *J. Thromb. Haemost.* **9**(1): 118 –129.

Alberti, K. G., Eckel, R. H., Grundy, S. M., Zimmet, P. Z., Cleeman, J. I., Donato, K. A. and Smith, S. C. (2009). Harmonizing the Metabolic Syndrome: A Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation. **120**(16): 1640 –1645.

Alexandre, A. S., Jussara M. C., Xuan, L., Zhen, W., Alan, J. M. and John, E. H. (2020). Role of Hyperinsulinemia and Insulin Resistance in Hypertension: Metabolic Syndrome Revisited. *Canadian Journal of Cardiology*. **36**(5): 671 -682.

Batsis, J. A., Nieto-Martinez, R. E. and Lopez-Jimenez, F. (2007). Metabolic Syndrome: From Global Epidemiology to Individualized Medicine. *Clinical Pharmacology & Therapeutics*. **82**(5): 509 –524.

Berberich, A. J. and Hegele, R. A. (2021). A Modern Approach to Dyslipidemia, *Endocrine Reviews.* **43**(4): 611 –653.

Berglund, E. D., Vianna, C. R., Donato, J. Jr., Kim, M. H., Chuang, J. C., Lee, C. E., Lauzon, D. A., Lin, P., Brule, L. J. and Scott, M. M. (2012). Direct

leptin action on POMC neurons regulates glucose homeostasis and hepatic insulin sensitivity in mice. *J. Clin. Investig.* **122**: 1000 –1009.

Borges, R. L., Ribeiro, A. B. and Zanella, M. T. (2010). Uric Acid as a Factor in the Metabolic Syndrome. *Curr Hypertens Rep.* **12:** 113 –119.

Corella, D. and Ordovas, J. M. (2004). The metabolic syndrome: a crossroad for genotype-phenotype associations in atherosclerosis. *Curr Atheroscler Rep*. **6**: 186 –96.

Defesche, J. C., Gidding, S. S., Harada-Shiba, M., Hegele, R. A., Santos, R. D. and Wierzbicki, A. S. (2017). Familial hypercholesterolaemia. *Nat Rev Dis Primers*.**3**: 17093.

Dommermuth, R. and Ewing, K. (2018). Metabolic syndrome: systems thinking in heart disease. *Prim Care.* **45**: 109 –129.

Duncker, D. J., Gardien, M., Vranckx, P., Versluis, S., Hasan, D. and Slager, C. J. (2002). The clinical significance of whole blood viscosity in (cardio)vascular medicine. *Neth Heart J*. **10**: 512-516.

Engin, A. (2017). The Definition and Prevalence of Obesity and Metabolic Syndrome. In: Engin, A., Engin, A. (eds) Obesity and Lipotoxicity. *Advances in Experimental Medicine and Biology*. Vol 960.

Fahed, G., Aoun, L., Bou-Zerdan, M., Allam, S., Bou Zerdan, M., Bouferraa, Y. and Assi, H. I. (2022). Metabolic Syndrome: Updates on Pathophysiology and Management in 2021. *International journal of molecular sciences.* **23**(2): 786.

Fathi D. B. (2018). The investigations of genetic determinants of the metabolic syndrome. *Diabetes Metab. Syndr*. **12**: 783 –789.

Ference, B. A., Ginsberg, H. N., Graham, I., Ray, K. K., Packard, C. J., Bruckert, E., Hegele, R. A., Krauss, R. M., Raal, F. J. and Schunkert, H. (2017). Low density lipoproteins cause atherosclerotic cardiovascular disease. Evidence from genetic, epidemiologic, and clinical studies. A consensus statement from the European Atherosclerosis Society Consensus Panel. *Eur. Heart J.* **38**: 2459 –2472.

Ford, E. S. (2005). Metabolic Syndrome/Insulin Resistance Syndrome/Pre-Diabetes: risks for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome: a summary of the evidence. *Diabetes Care.* **28**: 1769 –1778.

Forouzanfar, M. H., Liu, P. and Roth, G. A. (2017). Global burden of hypertension and systolic blood pressure of at least 110 to 115 mmHg. *JAMA*. **317**(2): 165 -182.

Frank, L. L., Sorensen, B. E., Yasui, Y., Tworoger, S. S., Schwartz, R. S., Ulrich, C. M., Irwin, M. L., Rudolph, R. E., Rajan, K. B., Stanczyk, F., Bowen, D., Weigle, D. S., Potter, J. D. and McTiernan, A. (2005). Effects of exercise on metabolic risk variables in overweight postmenopausal women: a randomized clinical trial. *Obesity research.* **13**(3): 615 –625.

Genser, L. C., Mariolo, J. R., Castagneto-Gissey, L., Panagiotopoulos, S. and Rubino, F. (2016). Obesity, type 2 diabetes, and the metabolic syndrome: pathophysiologic relationships and guidelines for surgical intervention. *Surg Clin North Am.* **96**: 681 –701.

Genuth, S., Alberti, K. G., Bennett, P., Buse, J., Defronzo, R., Kahn, R., Kitzmiller, J., Knowler, W. C., Lebovitz, H., Lernmark, A., Nathan, D., Palmer, J., Rizza, R., Saudek, C., Shaw, J., Steffes, M., Stern, M., Tuomilehto, J., Zimmet, P. and Expert Committee on the Diagnosis and Classification of Diabetes Mellitus

(2003). Follow-up report on the diagnosis of diabetes mellitus. Diabetes care. **26**(11): 3160 –3167.

Greenfield, J. R., Miller, J. W., Keogh, J. M., Henning, E., Satterwhite, J. H., Cameron, G. S., Astruc, B., Mayer, J. P., Brage, S., See, T. C., Lomas, D. J., O'Rahilly, S. and Farooqi, I. S. (2009). Modulation of blood pressure by central melanocortinergic pathways. *N Engl J Med.* **360**: 44 –52.

Grundy, S. M. (2004). Obesity, Metabolic Syndrome, and Cardiovascular Disease, *The Journal of Clinical Endocrinology & Metabolism.* 89(6): 2595 –2600.

Grundy, S. M. (2016). Metabolic syndrome update. *Trends in cardiovascular medicine*. **26**(4): 364–373.

Grundy, S. M., Hansen, B., Smith Jr, S. C., Cleeman, J. I., Kahn, R. A., American Heart Association; National Heart, Lung, and Blood Institute; and American Diabetes Association (2004). Clinical management of metabolic syndrome: report of the American Heart Association/National Heart, Lung, and Blood Institute/American Diabetes Association conference on scientific issues related to management. *Circulation.* **109**: 551 –556.

Hotamisligil, G. S. (2006). Inflammation and metabolic disorders. *Nature Res*. **444**: 860 –867.

Jackson, C. A. (1992). Rapid renal potassium adaptation in rats. *Am J Physiol*. **263**(6): F1098-104.

Kearney, P. M., Whelton, M., Reynolds, K., Whelton, P. K. and He, J. (2004). Worldwide prevalence of hypertension. *J Hypertens*. **22**(1): 11 -19.

Klonoff, D. C., Lias, C., Beck, S., Parkes, J. L., Kovatchev, B., Vigersky, R. A., Arreaza-Rubin, G., Burk, R. D., Kowalski, A., Little, R., Nichols, J., Petersen,

M., Rawlings, K., Sacks, D. B., Sampson, E., Scott, S., Seley, J. J., Slingerland, R. and Vesper, H. W. (2016). Development of the Diabetes Technology Society Blood Glucose Monitor System Surveillance Protocol. *J Diabetes Sci Technol.* **10**(3): 697-707.

Kolovou, G. D., Anagnostopoulou, K. K., Salpea, K. D. and Mikhailidis, D. P. (2007). The Prevalence of Metabolic Syndrome in Various Populations. *The American Journal of the Medical Sciences.* **333**(6): 362 –371.

Kopp, H. P., Kopp, C. W., Festa, A., Krzyzanowska, K., Kriwanek, S., Minar, E., Roka, R. and Schernthaner, G. (2003). Impact of weight loss on inflammatory proteins and their association with the insulin resistance syndrome in morbidly obese patients. *Arter. Thromb. Vasc. Biol.* **23**: 1042 –1047.

Kris-Etherton, P., Eckel, R. H., Howard, B. V., St Jeor, S. and Bazzarre, T. L. (2001). AHA Science Advisory: Lyon Diet Heart Study. Benefits of a Mediterranean-style, National Cholesterol Education Program/ American Heart Association Step I Dietary Pattern on Cardiovascular Disease. *Circulation*. **103**: 1823 –1825.

Lassegue, B., Sorescu, D., Szocs, K., Yin, Q., Akers, M., Zhang, Y., Grant, S. L., Lambeth, J. D. and Griendling, K. K. (2001). Novel gp91(phox) homologues in vascular smooth muscle cells: Nox1 mediates angiotensin II-induced superoxide formation and redox-sensitive signaling pathways. *Circ. Res*. **88**: 888 –894.

Lichtenstein, A. H., Appel, L. J., Brands, M., Carnethon, M., Daniels, S., Franch, H. A., Franklin, B., Kris-Etherton, P., Harris, W. S., Howard, B., Karanja, N., Lefevre, M., Rudel, L., Sacks, F., Van Horn, L., Winston, M. and Wylie-Rosett, J. (2006). Diet and lifestyle recommendations revision 2006: a scientific statement

from the American Heart Association Nutrition Committee. *Circulation*. **114**(1): 82 –96.

Lipińska, A., Koczaj-Bremer, M., Jankowski, K. Kaźmierczak, A., Ciurzyński, M., Ou-Pokrzewińska, A., Mikocka, E., Lewandowski, Z., Demkow, U. and Pruszczyk, P. (2014). Does family history of metabolic syndrome affect the metabolic profile phenotype in young healthy individuals?. *Diabetol Metab Syndr*. **6**(75).

Mach, F., Baigent, C., Catapano, A. L., Koskinas, K. C., Casula, M., Badimon, L., Chapman, M. J., De Backer, G. G., Delgado, V. and Ference, B. A. (2020). ESC Scientific Document Group. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: Lipid modification to reduce cardiovascular risk. *Eur. Heart J.* **41**: 111 –188.

Maffeis, C. and Morandi, A. (2018). Body composition and insulin resistance in children. *Eur J Clin Nutr.* **72**: 1239 –1245.

Mehta, P. K. and Griendling, K. K. (2007). Angiotensin II cell signaling: Physiological and pathological effects in the cardiovascular system. *Am. J. Physiol. Cell Physiol*. **292**: C82–C97

Mohamed-Ali, V., Pinkney, J. H. and Coppack, S. W. (1998). Adipose tissue as an endocrine and paracrine organ. *Int. J. Obes. Relat. Metab. Disord.* **22**: 1145 – 1158.

Moon, J. H., Roh, E., Oh, T. J., Kim, K. M., Moon, J. H., Lim, S., Jang, H. C. and Choi, S. H. (2017). Increased risk of metabolic disorders in healthy young adults with family history of diabetes: from the Korea National Health and Nutrition Survey. *Diabetol Metab Syndr*. **9**(16).

Murakami, L. and Rakotz, M. (2015). Self-measured blood pressure monitoring program: Engaging patients in self-measurement. 1st ed. *American Medical Association and the Johns Hopkins University School of Medicine*.

Naish, J. and Court, D. S. (2014). Medical sciences E-book. *Elsevier Health Sciences*. pp 562.

Pappan, N. and Rehman, A. (2024). Dyslipidemia. In: StatPearls; Treasure Island (FL), USA: *StatPearls Publishing*.

Peter, M. N., Jaakko, T. and Lars, R. (2019). The metabolic syndrome – What is it and how should it be managed? *European Journal of Preventive Cardiology.* **26**(2): 33 –46.

Pop, G., De Boer, M-J., Stalenhoef, A. F. H. (2016). Reducing cardiac after-load by lowering blood viscosity in patients with familial hypercholesterolemia - a pilot study. Possible mechanism for occurrence of anemia in chronic heart failure patients? *Cor et Vasa*. **58**: 374 -378.

Ranasinghe, P., Cooray, D. N., Jayawardena, R. and Katulanda, P. (2015). The influence of family history of Hypertension on disease prevalence and associated metabolic risk factors among Sri Lankan adults. *BMC Public Health.* **15**(1).

Reaven, G. (2008). Why a cluster is truly a cluster: Insulin resistance and cardiovascular disease. *Clin. Chem.* **54**: 785 –787.

Saiki, A., Ohira, M., Endo, K., Koide, N., Oyama, T., Murano, T., Watanabe, H., Miyashita, Y. and Shirai, K. (2009). Circulating angiotensin II is associated

with body fat accumulation and insulin resistance in obese subjects with type 2 diabetes mellitus. *Metabolism.* **58**: 708 –713.

Simmonds, M., Meiselman, H., and Baskurt, Oguz. (2013). Blood rheology and aging. *Journal of geriatric cardiology*. **10**: 291 -301.

Shyong, T., Su, Y. G., Jeannette, J. M. L., Moh-Sim, W., Derrick, H., Kenneth, H., Suok, K. C., Jeffery, C., William, C., Ken, G., Kee, S. C. and Chee, E. T. (2004). Lowering the Criterion for Impaired Fasting Glucose: Impact on disease prevalence and associated risk of diabetes and ischemic heart disease. *Diabetes Care.* **27**(7): 1728 –1734.

Sloop, G. D., Weidman, J. J. and St Cyr, J. A. (2018). Perspective: interesterified triglycerides, the recent increase in deaths from heart disease, and elevated blood viscosity. *Ther Adv Cardiovasc Dis*. **12**: 23 -28.

Stern, M. P., Williams, K. and Haffner, S. M. (2002). Identification of persons at high risk for type 2 diabetes mellitus: do we need the oral glucose tolerance test? *Ann Intern Med*. **136**: 575 -581.

Swarup, S., Goyal, A., Grigorova, Y. and Zeltser R. (2020). Metabolic Syndrome. StatPearls; Treasure Island, FL, USA: *StatPearls Publishing.*

Tartiere, J. M., Logeart, D., Safar, M. E. and Cohen-Solal, A. (2006). Interaction between pulse wave velocity, augmentation index, pulse pressure and left ventricular function in chronic heart failure. *J Hum Hypertens.* **20**: 213 –219.

The British Medical Association Illustrated Medical Dictionary (2002). London. *Dorling Kindersley.* Pp. 536.

Trayhurn, P. and Wood, I. S. (2004). Adipokines: Inflammation and the pleiotropic role of white adipose tissue. *Br. J. Nutr.* **92**: 347 –355.

Wannamethee, S. G. and Shaper, A. G. (2001). Physical activity in the prevention of cardiovascular disease: an epidemiological perspective. *Sports Med*. **31**: 101 –114.

Wright, J. T. Jr., Bakris, G., Greene, T., Agodoa, L. Y., Appel, L. J., Charleston, J., Cheek, D., Douglas-Baltimore, J. G., Gassman, J., Glassock, R., Hebert, L., Jamerson, K., Lewis, J., Phillips, R. A., Toto, R. D., Middleton, J. P., Rostand, S. G. and African American Study of Kidney Disease and Hypertension Study Group (2002). Effect of blood pressure lowering and antihypertensive drug class on progression of hypertensive kidney disease: results from the AASK trial. *JAMA*. **288**(19): 2421 –2431.