University of Kerbala

College of Science

Department of Chemistry

Biochemical Monitoring Of Obese Individuals Under a Specific Dietary System.

A Thesis

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Chemistry Science

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بِسْمِ اللَّهِ الرَّحْمَٰنِ الرَّحِيمِ

(وَكُلُوا وَاشْرَبُوا وَلَا تُسْرِفُوا إِنَّهُ لَا يُحِبُّ الْمُسْرِفِينَ)

صَدق الله العلي العظيم

سورة الاعراف الآية 31

DEDICATION

To the experiences we never expected, and the paths that were redirected. To myself who faced all this. To the family and friends we found along the way.

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Abstract

The dietary system plays a crucial role in influencing obesity, which is a growing global health concern. This study aimed to evaluate the biochemical changes associated with weight loss in individuals with obesity who underwent a specific dietary intervention. The research was conducted in eth Department of Chemistry, College of Science, University of Kerbala, Karbala, Iraq. A total of 116 samples were collected from December 2023 to February 2025. Of these, 56 participants were classified as obese, while 60 healthy individuals served as the control group.

Participants were categorized into three groups according to their Body Mass Index (BMI): healthy, overweight, and obese. A series of laboratory tests were conducted to measure blood glucose, lipid profiles, liver and kidney enzyme levels, insulin, and growth hormone levels. These tests were repeated after four months of following the dietary regimen.

The findings revealed significant improvements in the health status of obese individuals, including reductions in weight and Body Mass Index (BMI), both of which showed statistically significant changes ($p = 0.001$). Additionally, there was a notable improvement in random blood sugar (RBS) and insulin levels, with p-values of 0.001 and

0.006, respectively. These results indicate a decrease in insulin resistance and a reduced risk of developing type 2 diabetes.

Moreover, the lipid profile showed significant changes, with levels of triglycerides (TG), total cholesterol (TC), low-density lipoprotein (LDL), and lipase decreasing, while high-density lipoprotein (HDL) increased. These improvements are significant for enhancing liver, coronary, and arterial functions, which in turn positively affect kidney function.

These exciting results highlight the remarkable impact of the proposed diet on combating obesity and significantly improving relevant health indicators for a healthier future.

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Table of Abbreviations and Symbol

LIST OF ABBREVIATIONS AND SYMBOLS	
Abbreviation and symbol	The meaning
ADP	Adenosine diphosphate
AF	atrial fibrillation
ATP	Adenosine triphosphate
BMI	Body Mass Index
˚C	Degrees centigrade
Cm	Centimeter
CV	Cardiovascular
CVD	Cardiovascular disease
CAD	Coronary Artery Disease
CR	Calorie restriction
ERV	expiratory reserve volume
FRC	functional residual capacity
FFAs	free fatty acids
GFR	glomerular filtration rate
GH	Growth hormone
G-6-P	Glucose-6-phosphate
HbA1C	Glycated haemoglobin

HC	Hexokinase
HCC	hepatocellular carcinoma
HTN	Hypertension
HTN	Hypertension
HF	Heart Failure
HDL	High Density Lipoprotein Cholesterol
HFrEF	heart failure with reduced ejection fraction (HFrEF
IL-6	interleukin-6
ICMR	INDIAN COUNCIL OF MEDICAL RESEARCH
IF	Intermittent fasting
KD	ketogenic diets
Kg	Kilogram
LVH	Left ventricular hypertrophy
LCAT	lecithin-cholesterol -acyltransferase
LDL	Low Density Lipoprotein Cholesterol
LDH	Lactate dehydrogenase
Mmol	Millimole
MetS	metabolic syndrome
MI	myocardial infarction
MS	Metabolic syndrome
mg/dl	Milligram/ deciliter
NASH	nonalcoholic steatohepatitis
NSTEMI	non-ST segment elevation myocardial infarction

NAFLD	non-alcoholic fatty liver disease
NSTEMI	Non-ST-Elevation Myocardial Infarction
NCDs	Non communicable disease
NAFLD	non-alcoholic fatty liver disease
NADPH	nicotinamide adenine dinucleotide phosphate hydrogen
ORG	obesity-related glomerulopathy
pH	potential Hydrogen
RAAS	renin-angiotensin-aldosterone system
SS	simple steatosis
STEMI	ST-elevation myocardial infarction
SSGT	Serum separation gel tubes
TG	Triglyceride
TC	Total cholesterol
TLC	total lung capacity
T2DM	diabetes mellitus type 2
TNF-α	tumor necrosis factor-alpha
Mkat	Micro catalytic activity
μmol/L	Micromole/ liter
VD3	VitaminD3
VLCKDs	Very-low calorie ketogenic diet
WHO	World Health Organization
WC	waist circumference

CHAPTER ONE

INTRODUCTION

and literature review

1.1 Introduction

Diet and physical activity are essential for achieving and maintaining good health, but their imbalance has significantly contributed to the global rise in obesity. Obesity is not just about carrying excess weight; it is a serious health condition. The leading cause of obesity is a long-term energy imbalance, where individuals consistently consume more energy from food than they expend through daily activities and exercise.(1) Dietary patterns have changed significantly in recent decades, resulting in a decrease in the consumption of fruits, vegetables, and whole grains while simultaneously seeing a rise in highly processed meals high in calories, fats, and sugars.(2).

Modern lifestyles promote sedentary behavior by limiting exercise opportunities, reducing physical labour, and increasing screen time. The combination of poor diet and lack of physical activity has made it increasingly difficult for many people to maintain a healthy weight. Obesity is a complex public health challenge, rather than just an individual issue, because social, economic, and environmental factors further complicate efforts to adopt healthier behaviors.

Understanding the interconnected roles of diet and exercise in the development of obesity is essential for designing effective interventions to combat this growing epidemic. According to a 2021 report from the World Health Organization, the prevalence of obesity has nearly tripled worldwide since 1975, underscoring the urgent need for preventive strategies that promote healthier diets and more active lifestyles. Addressing obesity requires a comprehensive approach that considers not only individual behaviors but also broader systemic factors such as food marketing, urban planning, and healthcare policies.(3)

1.2 Overview of Overweight and Obesity

Overweight and obesity are conditions characterized by excessive body fat that can harm health. These issues are typically measured using the Body Mass Index (BMI), which compares weight and height to categorize individuals as underweight, normal weight, overweight, or obese.

Overweight refers to having a BMI between 25 and 29.9, while obesity is defined as having a BMI of 30 or higher. Both conditions increase the risk of various health problems, including heart disease, diabetes, and certain types of cancer.

Addressing overweight and obesity involves a combination of lifestyle changes, such as improved nutrition and increased physical activity, along with support from healthcare professionals. Early intervention and awareness are key to preventing these conditions and promoting overall health.

Obesity is recognized as a significant global health issue of the 21st century, contributing notably to the increasing rates of illness and premature death among populations worldwide.(4-6)

The World Health Organization (WHO) classifies individuals as overweight if they have a Body Mass Index (BMI) between 25.0 and 29.9 kg/m². At the same time, obesity is defined as a BMI of 30.0 kg/m² or higher. This classification is based on body weight rather than body fat percentage, as directly measuring body fat remains a clinical challenge. (5)

Obesity is caused by a variety of factors, including biological, psychosocial, environmental, and socioeconomic influences..(6) Additionally, the way obesity causes adverse health outcomes differs greatly among individuals, adding to the complexity of its clinical management.(7)

Obesity is strongly associated with an increased risk of various chronic health conditions, including hypertension, cardiovascular diseases, insulin resistance, and hyperinsulinemia. It can also lead to sleep disorders, lipid imbalances, and several types of cancer. (10) Additionally, obesity raises the risk of developing non-alcoholic fatty liver disease and is often linked to elevated levels of triglycerides, cholesterol, and glucose. (8, 9)

Obesity significantly increases the risk of developing type 2 diabetes. Both obesity and type 2 diabetes are among the most pressing global public health challenges, as noted in recent WHO reports from 2020 and 2021. Furthermore, obesity has become the second most common preventable cause of cancer and is expected to overtake tobacco use in this regard in the near future.(10)

Recent studies have shown a strong link between obesity and worse outcomes in COVID-19 patients, including higher rates of hospitalization, reliance on mechanical ventilation, and increased mortality.(13-15)

Genetic factors, urban environments, poor dietary choices, and a decline in physical activity influence the increasing prevalence of obesity. (12) Moreover, research has shown links between obesity-related traits—such as BMI, waist circumference, and fat distribution—and genetic variants, epigenetic changes (e.g., DNA methylation, non-coding RNA), gut microbiota composition, and socio-demographic factors. (13) Socioeconomic disparities significantly impact health outcomes. People from low-income communities often have limited access to nutritious foods and safe places to exercise, which increases the risk of obesity. (14)

Social stigma is a significant issue for individuals who are overweight or obese, as they often experience bias and discrimination. This stigma can adversely

affect their self-esteem and relationships, and it may even influence the quality of healthcare they receive. When obesity begins in childhood, these negative perceptions can harm academic performance, interpersonal skills, and future employment opportunities.

Obesity affects the physical health, emotional well-being, and quality of life of individuals, their families, and the communities in which they live. (15)

1.3 Body Mass Index (BMI)

Body Mass Index (BMI) is a commonly used metric that assesses body weight in relation to height. It is calculated by dividing a person's weight in kilograms by the square of their height in meters. While BMI serves as a quick and cost-effective screening tool, recent research has pointed out its limitations in accurately reflecting an individual's metabolic health. It is the most commonly used simple measure of body fat; however, it has some limitations. BMI calculates presumed excess weight based on height rather than measuring actual body fat. Additionally, it does not provide information about how fat is distributed in the body. In adults, central adiposity, or fat accumulation around the abdomen, is more closely linked to health risks than general adiposity or overall body fat.(16)

One possible explanation for this issue is that Body Mass Index (BMI) is not a reliable tool for assessing body composition. It does not differentiate between the various factors that contribute to body weight, such as muscle mass, fat distribution (including visceral, subcutaneous, and intramuscular fat), and other components like water, bone, and internal organs. Consequently, relying on BMI can result in misclassifying an individual's fat levels. For example, individuals with a "normal" BMI may still have excess body fat. In contrast, those with a BMI of 25 kg/m^2 or

higher may not necessarily have fat levels that significantly increase their risk of mortality. (17)

1.4 Obesity and related diseases.

1.4.1 The relationship between obesity and liver disorders.

Obesity plays a significant role in the development of metabolic syndrome (MetS) and its associated comorbidities, such as nonalcoholic fatty liver disease (NAFLD). There is a strong correlation between the increasing prevalence and severity of NAFLD and the global rise in obesity rates.

Nonalcoholic fatty liver disease (NAFLD) includes a range of liver conditions, from simple steatosis (SS) to nonalcoholic steatohepatitis (NASH), which can progress to severe liver disorders such as cirrhosis and hepatocellular carcinoma (HCC).(18) Currently, NAFLD is one of the leading causes of chronic liver disease in industrialized countries, with a global prevalence estimated at 25–30%, and up to 90% among individuals classified as morbidly obese.(19)

As the early stages of NAFLD are often asymptomatic, the increasing prevalence of obesity-related NAFLD and its complications is expected to present a significant public health challenge in the next decade. (23,(20) Meanwhile, mortality related to NAFLD continues to rise, in contrast to the declining mortality rates associated with viral hepatitis. (21) Deaths linked to NAFLD arise from hepatic issues like cirrhosis, its complications, and hepatocellular carcinoma (HCC), as well as extrahepatic conditions such as chronic kidney disease, cardiovascular disease (CVD), and various cancers. (22) Despite its increasing prevalence, there is a significant unmet need for both noninvasive diagnostic tools and effective, targeted therapies for NAFLD.(23)

The primary source of intrahepatic triglycerides in non-alcoholic fatty liver disease (NAFLD) comes from free fatty acids (FFAs). Approximately 60% of these FFAs are derived from lipolysis in adipose tissue, 15% come from dietary intake, and around 25% originate from de novo lipogenesis in hepatocytes, which typically results from carbohydrate consumption. This metabolic imbalance leads to the accumulation of fat in ectopic sites within the liver, rather than in its usual locations. The development of simple steatosis (SS) and its progression to nonalcoholic steatohepatitis (NASH) are primarily influenced by two key processes: lipotoxicity and glucotoxicity. These processes occur when hepatocytes are exposed to excessive levels of lipids and carbohydrates, respectively. Both SS and NASH are closely associated with diets high in fats and carbohydrates, which is commonly observed in obesity. Additionally, endoplasmic reticulum stress, oxidative stress, and mitochondrial dysfunction are pathophysiological processes that connect lipotoxicity and glucotoxicity to the development of SS and NASH. (23-27)

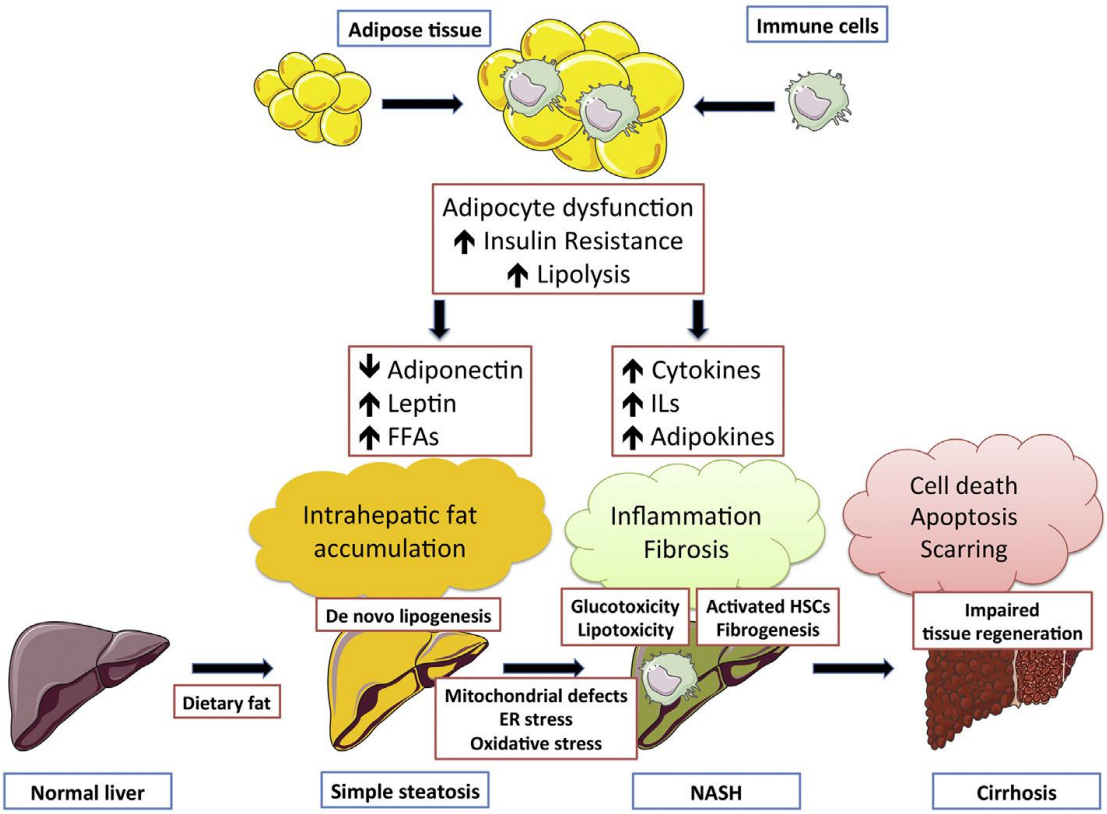


Figure (1-1): Obesity-driven pathophysiology of NAFLD(24).

1.4.2 Obesity and Disorders of the Respiratory System

Obesity is a significant and growing cause of breathing problems. One of the first aspects affected is the expiratory reserve capacity. People with obesity often experience reduced airflow, increased airway hyper-responsiveness, and a higher risk of developing pulmonary conditions such as hypertension, pulmonary embolism, respiratory infections, obstructive sleep apnea, and obesity hypoventilation syndrome. Over time, these physiological changes caused by excess fat can lead to hypoxic or hypercapnic respiratory failure. The underlying mechanisms for these changes include a state of systemic inflammation and the physical strain that excess fat places on the respiratory system. Losing weight can

significantly improve airway and respiratory function in individuals with obesity. (25)

Obesity primarily affects thoracic compliance, which in turn impacts lung capacity. The buildup of adipose tissue in the abdomen and chest wall creates physical limitations on chest expansion and diaphragm movement, resulting in reduced thoracic compliance in obese individuals. (26)(29) Obesity decreases lung compliance primarily by increasing pulmonary blood flow, but it also leads to peripheral airway closure, resulting in persistent microatelectasis.(28)

Lung compliance may further decrease due to increased alveolar surface tension from a reduction in functional residual capacity (FRC). (29) However, thoracic compliance appears to be more significant than the impact of decreased lung compliance. (30)

The earliest and most commonly reported effect of obesity on static lung volumes is a reduction in expiratory reserve volume (ERV), typically with residual volume remaining intact, ultimately resulting in a decrease in functional residual capacity (FRC). (31),(32) The severity of obesity is inversely related to various static lung volumes and capacities, demonstrating an exponential relationship with the decline in expiratory reserve volume (ERV).(31) A similar relationship is seen with the end-expiratory lung volume. (33). In extremely obese individuals, total lung capacity (TLC) may reduce by 20%, leading to a restrictive defect; however, TLC typically remains normal in other cases. (36)

Obesity alone is unlikely to fully explain the significant respiratory impairment observed in individuals. Therefore, a reduction in total lung capacity (TLC) in those with a BMI of up to 40 kg/m² should prompt an investigation into other potential respiratory conditions (32). The impact on static lung volumes is

more commonly seen in individuals with an abdominal (android) pattern of obesity, as this pattern more directly affects diaphragm function compared to those with a gynecoid pattern.

1.4.3. Obesity and cardiovascular diseases

Obesity significantly increases the risk of developing various cardiovascular disorders, including atrial fibrillation, dyslipidemia, diabetes, and coronary artery disease. (40-43) Additionally, it poses a significant risk for conditions such as hypertension, heart failure, and atherosclerotic cardiovascular disease, affecting their pathophysiological mechanisms, diagnostic processes, and clinical manifestations.(34)(35) (36). Early identification of cardiovascular (CV) risk is crucial for reducing mortality rates and maintaining quality of life, as cardiovascular disease (CVD) is a leading cause of death and disability, particularly among individuals who are overweight or obese. An estimated 17.9 million deaths each year are attributed to CVD, many of which could be prevented through optimal treatment, community initiatives, and clinical interventions aimed at addressing risk factors associated with overweight and obesity.(37).

1.4.3.1. Relation between Obesity and Coronary Artery Disease

Obesity and coronary atherosclerosis are closely related conditions. A study conducted on adolescents found early signs of cardiovascular disease associated with atherosclerosis. Individuals with higher BMI values tend to have more frequent and advanced atherosclerotic vascular lesions compared to those with normal body weight. (38) Obesity is closely linked to the development of coronary artery disease (CAD). CAD occurs when the arteries that supply blood to

the heart become narrowed or blocked due to plaque buildup caused by atherosclerosis. This condition can result in heart attacks and angina (chest pain). Longitudinal research suggests that obesity lasting at least 20 years may be an independent risk factor for coronary artery disease. (39)

After smoking, being overweight is considered the largest risk factor for non-ST-elevation myocardial infarction (NSTEMI) in young adults. The incidence of NSTEMI rises with body mass index (BMI). A similar correlation is observed in patients with ST-elevation myocardial infarction (STEMI). (40) Obesity is a significant risk factor for STEMI, as it may contribute to various vascular events beyond just age.(41)

1.4.3.2 Relation between Obesity and Hypertension (High Blood Pressure)

Excess body weight increases the demand for blood to supply oxygen and nutrients to tissues, which puts extra strain on the arterial walls. This added pressure may eventually lead to hypertension (HTN), further increasing the risk of heart disease and stroke. Both obesity and hypertension are commonly observed in patients with cardiovascular disease (CVD) and are linked to metabolic syndrome. They contribute to the development of heart failure (HF), which can occur with either preserved or reduced ejection fraction; the latter often arises after a myocardial infarction (MI). (42) Both conditions are significant risk factors for heart failure (HF), and their widespread prevalence highlights their clinical importance. Specifically, hypertension is marked by chronic low-grade inflammation that contributes to detrimental cardiac remodeling. Both conditions are significant risk factors for heart failure (HF), and their widespread prevalence highlights their clinical importance. Specifically, hypertension is marked by chronic low-grade inflammation that contributes to detrimental cardiac remodeling.

(42) Excess body fat due to obesity significantly contributes to vascular complications that can result in end-organ damage from hypertension. Notably, even when considering other risk factors associated with atherosclerosis, obesity is still strongly linked to early signs of cardiovascular problems. These signs include coronary artery calcification, thicker intima-media layers in the carotid arteries, and an enlarged left ventricular mass. Understanding these connections can help us tackle obesity and enhance heart health. Central obesity is crucially linked to higher risks of microvascular disease and arterial stiffness, which significantly contribute to the high prevalence of hypertension in this population. (41)

1.4.3.3 Obesity and Atrial Fibrillation

Obesity is linked to a higher risk of atrial fibrillation (AF), a type of irregular heartbeat that can lead to serious complications such as blood clots, stroke, and heart failure. AF is characterized by rapid activation of the atria, which causes disorganized contractions in the atria and irregular rhythms in the ventricles.

Atrial fibrillation (AF) can sometimes occur without any clear structural or electrical abnormalities. However, epidemiological studies have shown that it is increasingly associated with various comorbidities. These conditions can lead to structural and pathological changes in the atria, resulting in an arrhythmogenic environment known as atrial cardiomyopathy. (43)

There are several possible explanations for how obesity affects the onset and progression of atrial fibrillation.

1. Inflammatory Response: Excess body fat triggers a systemic inflammatory response, producing various pro-inflammatory chemicals. These chemicals can

damage the structure and electrical function of the heart, thereby increasing the risk of developing atrial fibrillation.

2. Structural Changes: Obesity can cause the left atrium of the heart to enlarge, altering its anatomy. This change makes it easier for irregular heartbeats to initiate and persist.

3. Hormonal Factors: Adipose tissue serves as a source of hormones that can also influence heart function.

These factors collectively contribute to the increased risk of atrial fibrillation in individuals with obesity. (43)

1.4.3.4. Fat Mass Disease: Direct and Indirect Effects on Cardiovascular Risk

For many years, it was believed that obesity and cardiovascular disease (CVD) were indirectly related. However, in recent decades, a significant amount of data has emerged demonstrating that obesity and CVD are causally related.(44) (45) (46). A vicious cycle of weight gain increases cardiovascular risk when an excessive rise in body weight limits energy expenditure, hinders mobility and physical activity, or exacerbates musculoskeletal comorbidities such as osteoarthritis. (47) Individuals who are overweight or obese experience increased cardiac output and total blood volume, which can lead to both anatomical and functional changes in the heart and vascular system. (48, 49, 55, 58)

Left ventricular hypertrophy (LVH) is a significant risk factor for increased left ventricular filling pressure and is frequently associated with left ventricular diastolic dysfunction, as well as a greater likelihood of heart failure. LVH is caused by factors such as increased intravascular volume and neuro-

hormonal pathways. Recent Mendelian randomization studies provide support for these causal associations. Obesity-related heart failure with reduced ejection fraction (HFrEF) can develop and may be worsened by kidney compression resulting from obesity. Additionally, this condition can be linked to the upregulation of the sympathetic nervous system and the renin-angiotensin-aldosterone system (RAAS), both of which heighten the risk of hypertension and stroke. (49)(50)

Addressing obesity and its related health issues has become one of the most significant challenges worldwide. While numerous studies indicate that weight loss is associated with a decreased risk of health problems, many overweight and obese individuals struggle to lose a substantial amount of weight or maintain that weight loss over the long term, despite efforts involving lifestyle changes, medications, and bariatric surgery. However, existing anti-obesity medications come with several drawbacks, including the need for long-term use, high costs, and the potential for limited effectiveness and side effects. (61-64)

Bariatric surgery has been shown to provide the most significant weight loss and high remission rates for metabolic syndrome and type 2 diabetes. However, it carries risks and side effects. Due to its high cost and limited eligibility, this option is not available to all individuals struggling with obesity. To address obesity and its associated health issues, three main dietary approaches have been established: Calorie Restriction (CR), Low-Carbohydrate Diets, and Intermittent Fasting. (65)

1.4.4 Obesity and Dyslipidemia

Obesity and dyslipidemia are interconnected health conditions that can significantly impact overall health. Obesity refers to excessive body fat that increases the risk of various diseases, while dyslipidemia involves abnormal levels of lipids, such as cholesterol and triglycerides, in the bloodstream. Both conditions can lead to serious health issues, including cardiovascular diseases, diabetes, and metabolic syndrome. Addressing these conditions through lifestyle changes, such as a balanced diet and regular exercise, is essential for improving health outcomes.

Obesity significantly affects lipid metabolism, leading to notable changes in the blood lipid profile. This disruption often results in dyslipidemia, which is characterized by imbalances in cholesterol and other blood lipids. Specifically, individuals with obesity typically show lower levels of high-density lipoprotein (HDL) cholesterol, often called "good" cholesterol, along with elevated levels of triglycerides and low-density lipoprotein (LDL) cholesterol, known as "bad" cholesterol. These lipid abnormalities are well-established risk factors for cardiovascular disease (CVD). Additionally, obesity increases the risk of CVD through several mechanisms, including elevated blood pressure, low HDL cholesterol, increased fasting triglycerides, and higher levels of insulin and blood glucose. It is also associated with elevated levels of triglycerides (TG) and free fatty acids (FFA), a normal or mildly increased level of LDL cholesterol, an increase in small dense LDL particles, and both reduced and functionally impaired HDL cholesterol. (51)

In obesity, elevated triglyceride levels often result from increased fat breakdown in adipose tissue and insulin resistance, both of which disrupt normal lipid processing. Consequently, LDL cholesterol, commonly referred to as "bad

cholesterol," becomes more abundant and smaller in size, which increases the likelihood of contributing to plaque formation in the arteries. The liver and intestines play a significant role in reversing cholesterol transfer by producing HDL particles. HDL cholesterol promotes the return of cholesterol from perivascular tissues, such as the artery wall, back to the liver. HDL particles absorb newly generated free cholesterol from surrounding tissues. This HDL cholesterol is then converted into cholesterol esters by the enzyme lecithin-cholesterol acyltransferase (LCAT), which is linked to HDL. (52)

1.4.5. Obesity and Diabetes

Pre-diabetes is a condition characterized by elevated blood glucose levels that are not high enough to be classified as diabetes. Individuals who are obese are significantly more likely to develop type 2 diabetes, and their risk of cardiovascular problems is even greater. Both obesity and diabetes are linked to high rates of preventable death and are complex health conditions. Additionally, both stroke and cardiovascular disease increase these risks. According to the American Heart Association, optimal cardiovascular health requires a body mass index (BMI) of less than 25 kg/m^2 and a fasting plasma glucose level below 100 mg/dL. (70)

The development of type 2 diabetes can be influenced by various risk factors. Some of these warning signals can be modified, while others cannot. Modifiable risk factors include high blood pressure, obesity (defined as a BMI greater than 30 kg/m^2), elevated cholesterol levels, and physical inactivity. On the other hand, unchangeable risk factors consist of age, race, ethnicity, family history, genetics, gestational diabetes, and a history of hyperglycemia or diabetes precursors. Type 2 diabetes mellitus (T2DM) has long-term effects, as the onset of

hyperglycemia is gradual and may go untreated for years. As time passes, the body begins to experience negative effects, increasing the risk of additional health problems, such as microvascular complications. In people with type 2 diabetes, insulin levels may be elevated or normal. However, if the islet cells in the pancreas are functioning properly, consistently high blood glucose levels are likely to cause an even greater rise in insulin production. (53)

1.4.6 Obesity and Insulin Resistance

One important factor contributing to the development of insulin resistance is obesity, particularly the accumulation of visceral fat. In individuals with obesity, expanding adipose tissue releases higher levels of pro-inflammatory cytokines (such as TNF-α and IL-6), adipokines (like leptin and resistin), and free fatty acids (FFAs). These substances can inhibit insulin signaling pathways, further exacerbating insulin resistance.(54) Chronic low-grade inflammation linked to obesity disrupts insulin receptor function and glucose uptake in peripheral tissues, especially in muscle and liver. (55) Excessive buildup of lipids in muscle cells, known as lipotoxicity, disrupts metabolic regulation by impairing the function of insulin. Additionally, insulin sensitivity is further diminished by oxidative stress and mitochondrial dysfunction, which are more prevalent in individuals with obesity. Furthermore, the obesity-related disruption of the insulin-sensitizing hormone adiponectin leads to increased insulin resistance. (56)

These pathophysiological processes collectively illustrate how obesity significantly contributes to insulin resistance and the onset of conditions such as type 2 diabetes and metabolic syndrome.

1.4.7. Obesity and Kidney Disease

Obesity is not only a risk factor for metabolic diseases but is also known to contribute to the development and progression of kidney disease. Excess body weight can lead to several anatomical and functional changes, including glomerular hyperfiltration, increased intraglomerular pressure, and kidney enlargement. (57)

Initially, adaptations were intended to offset increased metabolic demands; however, these early changes can ultimately lead to glomerulosclerosis, proteinuria, and a progressive decline in kidney function. Even before the onset of metabolic syndrome or diabetes, renal impairment can be present in individuals with obesity. Common indicators of decreased glomerular filtration rate (GFR) include elevated levels of blood creatinine and urea. A specific type of kidney damage associated with obesity, known as obesity-related glomerulopathy (ORG), is characterized by segmental sclerosis, glomerular hypertrophy, and podocyte destruction. Importantly, research has shown that weight loss, whether achieved through bariatric surgery or lifestyle modifications, improves kidney health by reducing albuminuria and hyperfiltration. Thus, addressing obesity is a crucial strategy for maintaining renal health and preventing cardiovascular diseases. (78-79)

The impact of kidney disease linked to obesity is anticipated to grow as global obesity rates continue to rise, emphasizing the need for early intervention and comprehensive treatment. (58)

1.4.8 Obesity and Vitamin D3

Vitamin D3 is an essential vitamin that helps maintain strong bones, supports the immune system, and regulates how our body utilizes calcium. Research also indicates that vitamin D plays a role in body weight and fat storage. (59)

Low blood levels of vitamin D are commonly found in obese individuals, and this can happen for several reasons. One key reason is that vitamin D is a fat-soluble vitamin, meaning that it gets stored in body fat. In obese individuals, more vitamin D is stored in adipose tissue, which reduces the amount available for the body to use. Additionally, obese people may spend less time outdoors, leading to decreased exposure to sunlight, which is essential for the skin's production of vitamin D. Furthermore, obesity-related liver abnormalities and inflammation can negatively affect the body's ability to activate vitamin D. (60)

Low levels of vitamin D3 can complicate weight loss due to its role in regulating appetite and fat metabolism. When vitamin D3 levels are insufficient, fat cells tend to grow larger and store more fat rather than converting it into energy. As a result, the body may find it easier to retain excess fat. (81)

Vitamin D_3 influences hormones such as leptin, which signals the brain that we are full. A deficiency in vitamin D_3 can lead to leptin malfunction, resulting in increased appetite and a higher likelihood of overeating. Additionally, vitamin D_3 stimulates enzymes involved in fat breakdown, aiding the body in burning fat. When vitamin D_3 levels are low, the body becomes less effective at using fat for energy, which may hinder weight loss efforts. (61)

Finally, the persistent low-grade inflammation associated with obesity worsens when vitamin D3 levels are low. This increased inflammation can disrupt normal

metabolism, making it even more challenging to lose fat. Overall, these factors suggest that addressing vitamin D3 deficiency may help individuals with obesity achieve weight loss. (84)

1.4.9 Obesity and Growth Hormone

Growth hormone (GH) is an important hormone produced by the pituitary gland that helps regulate body composition, muscle and bone growth, metabolism, and fat distribution. In individuals with obesity, secretion of growth hormone is often reduced. This condition is commonly referred to as "functional growth hormone deficiency," which means that while the pituitary gland may still produce GH, less of it is released than normal. One reason for this decrease is that excess body fat, particularly visceral fat, disrupts the normal signals that stimulate growth hormone production. Additionally, high insulin levels, which are common in obesity, can also suppress GH production. Moreover, free fatty acids and inflammatory cytokines released from adipose tissue further inhibit the generation of growth hormone. (85,86)

Low growth hormone levels in obese individuals contribute to increased fat storage, especially around the abdomen. Growth hormone (GH) typically aids in the development of lean muscle and promotes fat breakdown (lipolysis). Consequently, when GH levels are low, muscle mass declines, and fat burning slows down. This situation heightens the risk of weight gain and makes weight loss more challenging. Interestingly, research has shown that losing body weight can help stimulate the release of growth hormone. As individuals shed body fat, particularly visceral fat, GH levels often rise, which in turn encourages fat breakdown and supports a healthy metabolism. (62)

In conclusion, it is important to recognize that obesity and low growth hormone (GH) levels create a harmful cycle. Increased fat leads to a decrease in GH, and lower GH levels promote further fat storage. This emphasizes the importance of maintaining a healthy body composition for overall appearance, hormonal balance, and metabolic health.

1.5. Dietary system

Individual responses to nutrients and dietary components can vary significantly, and this biological diversity is a major source of variation in nutritional research. As modern tools and technologies have advanced, there has been increased focus on understanding these individual differences and how they influence disease risk. (63)(64). A primary contributor to obesity is the imbalance between energy intake and energy expenditure. Energy is obtained from the consumption of macronutrients, proteins, fats, and carbohydrates. The quantity and type of these nutrients influence both body fat (adiposity) and the maintenance of energy balance.(65).

The combination of sedentary lifestyles and poor dietary habits plays a significant role in the development of obesity. It is essential to address these issues within the broader context of the food system, especially since unhealthy environments disproportionately affect individuals from lower socioeconomic backgrounds. In these groups, there is a higher tendency to consume inexpensive foods that are high in saturated fats and refined carbohydrates, which increases the risk of obesity and related health problems. (66) To promote healthy eating habits, it is effective to implement strategies that focus on diets rich in whole grains, legumes, nuts, fruits, vegetables, and unsaturated fats. Additionally, it is strongly recommended to

reduce the intake of processed meats, saturated fats, refined carbohydrates, and added sugars. (67)

Research indicates that carbohydrate intake, rather than fat, has a stronger correlation with both short-term and long-term weight gain when overall caloric intake remains constant. Additionally, a meta-analysis comparing low-carbohydrate and low-fat diets provides further evidence that reducing carbohydrate consumption can improve indicators of metabolic syndrome, such as obesity, irrespective of total calorie intake or weight loss. (68).

1.5.1. Calorie restriction (CR):

A nutritional intervention involving reduced overall calorie intake while ensuring adequate nutrition focuses on lowering average daily caloric intake by approximately 20–40% without depriving the body of essential nutrients. This approach is not about starvation; instead, it emphasizes consuming fewer calories while maintaining a balanced and nutritious diet. Calorie restriction (CR) is closely related to obesity in several ways:

1. Prevention and Reduction of Obesity: By lowering caloric intake, CR naturally reduces body fat and body weight, which can help prevent obesity or assist in its treatment.

2. Improvement of Metabolic Health: Obesity is associated with metabolic dysfunctions, such as insulin resistance, high blood pressure, and inflammation. CR has been shown to enhance insulin sensitivity, decrease inflammation, and positively affect lipid profiles.

3. Impact on Obesity-Related Diseases: Chronic conditions including type 2 diabetes, cardiovascular disease, and certain types of cancer are linked to obesity.

CR may reduce the risk or severity of these diseases by promoting a healthier metabolic profile. (69)

1.5.1.1 Balanced Diet

Maintaining a healthy body relies significantly on the consumption of food. The assortment of foods that individuals eat is known as their diet, while the elements within those foods that the body digests, absorbs, and uses for various functions are called nutrients. A balanced diet consists of a diverse range of foods in the right amounts and proportions to sufficiently meet the body's needs for calories, minerals, vitamins, proteins, and other essential nutrients.

Nutrients provide the body with energy as well as the components needed for growth and repair. It is widely recognized that a balanced diet is crucial for preventing illness and promoting health. Everyone needs to consume a variety of foods to maintain good health. (98)

A balanced diet consists of a variety of foods consumed in adequate amounts and in the correct proportions to meet our daily requirements for essential nutrients. These nutrients include micronutrients, such as vitamins and minerals, as well as macronutrients like carbohydrates, proteins, and fats.

Maintaining a balanced diet helps protect us from lifestyle-related health issues and promotes overall well-being. It also provides a nutritional reserve of essential nutrients, allowing the body to cope with short periods of dietary deficiency.

Since no single food can supply all the essential nutrients our bodies require for proper functioning, a balanced diet is important. It involves obtaining the right amounts of nutrients from a diverse range of foods to support our overall health, as illustrated in figure (1-2). (99) A balanced diet contains all the essential nutrients in

the appropriate amounts and proportions. Creating a balanced diet is straightforward by including all five of the basic food groups recommended by the ICMR, as shown in table 1-1. Factors such as age, sex, physiological condition, and physical activity all affect the amount of food needed to meet daily nutrient requirements.

Table (1-1): ICMR Five Food Group

Food Group	Main Nutrients
I. Cereals, Grains and Products : Rice, Wheat, Ragi, Bajra, Maize, Jowar, Barley, Rice flakes, Wheat Flour.	Energy, protein, Invisible fat, Vitamin B1, Vitamin B2, Folic Acid, Iron, Fiber.
II. Pulses and Legumes : Bengal gram, Black gram, Green gram, Red gram, Lentil (whole as well as dhals), Cowpea, Peas, Rajmah, Soyabeans, Beans	Energy, Protein, Invisible fat, Vitamin B1, Vitamin B2, Folic Acid, Calcium, Iron, Fiber.
III. Milk and Meat Products : **Milk:** Milk, Curd, Skimmed milk, Cheese **Meat :** Chicken, Liver, Fish, Egg, Meat.	Protein, Fat, Vitamin B12, Calcium. Protein, Fat, Vitamin B2
IV. Fruits and Vegetables : **Fruits :** Mango, Guava, Tomato Ripe, Papaya,	Carotenoids, Vitamin –C, Fibre.

Orange. Sweet Lime, Watermelon.	
Vegetables (Green Leafy) : Amaranth, Spinach, Drumstick leaves, Coriander leaves, Mustard leaves, fenugreek leaves	Invisible Fats, Carotenoids, Vitamin – B2, Folic Acid, Calcium, Iron, Fibre.
Other Vegetables : Carrots, Brinjal, Ladies fingers, Capsicum, Beans, Onion, Drumstick, Cauliflower.	Carotenoids, Folic Acid, Calcium, Fibre
V. Fats and Sugars Fats Butter, Ghee, Hydrogenated oils, Cooking oils like Groundnut, Mustard, Coconut.	Energy, Fat, Essential Fatty Acids
Sugars: Sugar, Jaggery	Energy

Figure (1-2): Show the Five Food Group Systems

The recommended guidelines for designing a balanced diet assist in creating nutritious and healthy recipes that are tailored to specific regions and cultures. The composition of a balanced diet varies based on factors such as age,

sex, physiological condition, and physical activity level. All of these elements influence the quantity of food needed to meet daily nutrient requirements.

This study proposes a balanced diet that includes both macronutrients and micronutrients. Macronutrients, such as carbohydrates, fats, and proteins, are needed in larger amounts (measured in grams), while micronutrients, including vitamins and minerals, are required in smaller quantities (measured in milligrams and micrograms). A proper balanced diet should consist of 50-60% of total calories from carbohydrates, primarily from complex carbohydrates. Additionally, 10-15% of calories should come from proteins, and 20-30% should be derived from both visible and invisible fats. (100)

The body needs essential nutrients in the right amounts to maintain health and prevent various diseases. A well-balanced diet should provide not only macronutrients but also important micronutrients and functional foods. These include phytochemicals, prebiotics, antioxidants, and dietary fiber, all of which play a crucial role in overall health.

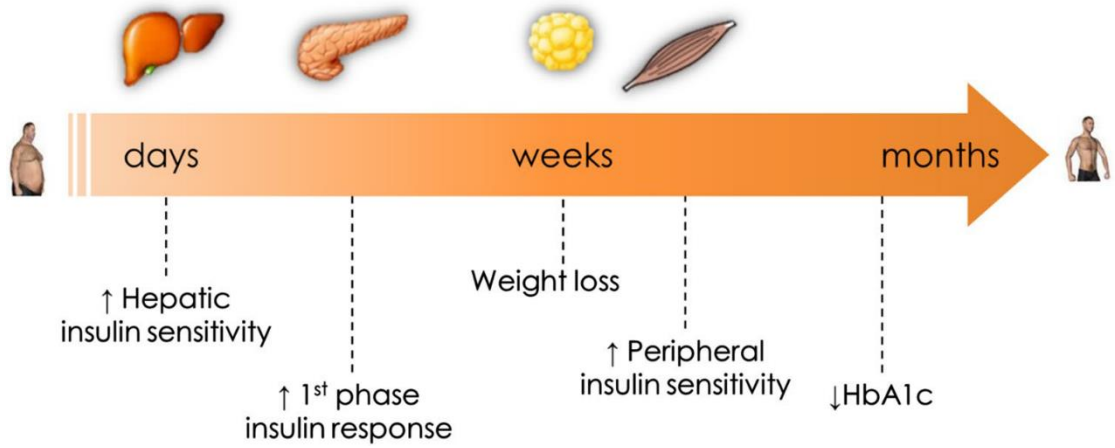
Antioxidants, such as vitamins C and E, beta-carotene, riboflavin, and selenium, help protect the body from oxidative stress caused by free radicals. To promote overall health, it's important to include beneficial foods in your diet while also limiting or avoiding unhealthy components, such as refined grains, alcohol, processed foods, trans fats, and refined sugars. The benefits of a balanced diet include providing essential nutrients for growth, physical development, optimal physiological functions, energy production, and overall well-being. Nutritious food is crucial for sustaining life and supporting daily activities.

Nutritional imbalances can occur due to either overconsumption (overnutrition) or insufficient intake (undernutrition). These imbalances may lead

to deficiency disorders or chronic conditions such as obesity, hypertension, diabetes, and other lifestyle-related diseases. It is essential to maintain a balanced and nutrient-dense diet throughout life. Poor nutrition can weaken the immune system, making the body more vulnerable to infections and illnesses. In contrast, a balanced diet helps prevent malnutrition in all its forms and reduces the risk of noncommunicable diseases (NCDs) and associated health complications. (70)

1.5.2. Low-Carbohydrate Diets

Low-carbohydrate diets, such as the ketogenic (KD) and Atkins diets, are characterized by low carbohydrate intake, high fat content, and adequate protein levels. These diets are commonly used for weight loss. The very low-calorie ketogenic diet (VLCKD) has been proposed as a therapeutic approach for various medical conditions, including severe obesity, obesity with comorbidities, non-alcoholic fatty liver disease (NAFLD), drug-resistant epilepsy, and as a supportive measure for weight loss prior to bariatric surgery. Additionally, VLCKD may provide potential benefits in improving blood pressure, serum glucose levels, and lipid profiles. (71)



Figure(1-4): Effects of VLCKD on glucose homeostasis and metabolic parameters in obese subjects with or without type 2 diabetes (72)**.**

1.5.3. Intermittent Fasting (IF)

An eating pattern that alternates between fasting and eating periods. It is currently very popular in the health and fitness community. Here are the most commonly used techniques for intermittent fasting:

1- The 16/8 Method: Also known as the Lean gains regimen, this method involves a 16-hour fast followed by an 8-hour eating window. Some individuals prefer to skip breakfast to achieve this, while others may choose to eat early in the day or skip dinner.

2- Eat-Stop-Eat: This involves fasting for 24 hours once or twice a week. It's advisable to try this method only if you are comfortable with fasting, as it can be a bit more challenging.

3- The 5:2 Diet: In this approach, you consume only 500–600 calories on two nonconsecutive days of the week while eating normally on the other five days. This diet can lead to various beneficial cellular and molecular changes in your body.

Insulin levels decrease while human growth hormone (HGH) levels increase. Additionally, cells in the body change gene expression and initiate important cellular repair processes. The most common reason people try intermittent fasting (IF) is to lose weight. This is because IF boosts the release of the fat-burning hormone norepinephrine (noradrenaline), which helps temporarily elevate the metabolic rate. (104,105)

1.6 The Relationship between Obesity and Dietary Patterns

Although it is well recognized that calorie intake has a significant impact on weight growth, it is crucial to emphasize that eating a healthy diet is essential to avoiding becoming overweight or obese. Consuming significant quantities of processed foods rich in starch, such as refined grains, fats, and sweets, on a regular basis has been linked to weight gain, whereas eating fruits, vegetables, whole grains, nuts, and yogurt has been linked to weight loss (73).

Diets tailored to address overweight or obesity can be structured based on specific food groups or their nutritional components, such as fat or dietary fiber. However, the intricate relationships, such as the strong correlations between the intake of various nutrients or food items, as well as biochemical interactions between nutrients, make it difficult to fully capture the complexity of the human diet in relation to overweight and obesity (74).

1.7 Dietary system and physical activity

A healthy lifestyle includes maintaining a balanced diet and engaging in regular exercise. Both factors significantly influence the risk of developing metabolic syndrome. Generally, individuals who are physically active are more

likely to have healthier eating habits compared to those who are sedentary, often associating their diet with their exercise routine. (108, 109)

At every stage of human growth and development, consuming a balanced diet is crucial for maintaining an active metabolism. Nutrition and physical activity significantly contribute to the development of various body tissues, including adipose (fat) tissue, skeletal muscles, and bones, particularly during childhood and adolescence. Importantly, even when physical activity levels decrease, the processes of development and maturation usually continue to progress. Regular physical activity is essential for helping individuals lose weight or maintain a healthy weight. (110-112)

Physical activity is significantly influenced by environmental factors. Both nutrition and physical exercise are essential for survival, but cultural shifts in various parts of the world may impact on people's daily spontaneous activities. (113) According to the highlights of the WHO global strategy, maintaining optimal health throughout life and preventing both acute and chronic illnesses depend on following a balanced diet and getting sufficient exercise. The WHO recommends that individuals engage in moderate to vigorous physical activity for at least 150 minutes per week to promote and maintain overall health. (114) Diets rich in fruits and fiber, along with lower consumption of salt, refined sugars, saturated fats, and alcohol, can help prevent or alleviate the symptoms of metabolic syndrome. (75)

In the context of obesity, it is essential to understand the importance of physical activity and its interaction with nutrition in relation to energy balance, as these factors contribute to maintaining a stable weight. For example, when evaluating a person's food consumption, it's important to consider their energy expenditure. Any increase in physical activity should be matched by an equivalent

increase in calorie intake. Energy flux, defined as the rate at which dietary energy is used for metabolic functions or stored in body tissues, plays a vital role in regulating the body's dynamic fuel homeostasis. Both diet and physical activity influence energy balance and are key components in managing body weight effectively. (76)

1.8. Aim and Objectives of the Study

The aim of this study is to evaluate the biochemical changes associated with weight loss in obese individuals following a structured dietary regimen and to assess the effectiveness of nutritional interventions in improving metabolic health outcomes.

1. To compares key biochemical parameters such as fasting blood glucose, HbA1c, lipid profile, and insulin levels between obese individuals and healthy controls.

2. To assess the impact of a low-calorie dietary regimen on metabolic indicators in obese participants.

3. To investigate the potential role of dietary modifications in reducing the risk of chronic diseases associated with obesity, including diabetes, cardiovascular disease, and metabolic syndrome.

4. To establish a correlation between anthropometric measurements (e.g., BMI and waist circumference) and the biochemical changes that result from dietary interventions.

CHAPTER TWO

Materials and Method

Chapter 2: Materials and Methods

2.1 Study Design

This longitudinal study was conducted from December 2023 to February 2025. It took place in the postgraduate laboratory of the Department of Chemistry at the College of Science, University of Kerbala, in collaboration with private medical laboratories, specifically Al-Ream Laboratory in the Al-Abbas neighborhood and Dr. Noor Alhuda Sabah Laboratory on Alaskan Street in Kerbala, Iraq. The study included 56 participants with obesity and 60 healthy volunteers who served as the control group. All participants were fully informed about the study, and verbal consent was obtained for their participation.

2.1.1Collection of Samples

A total of 56 Obese participants (224samples around four months), aged 13 to 65 years, were examined using random cluster sampling methods in Kerbala, Iraq.

Venous blood samples were collected from participants with obesity and healthy volunteers. Blood was drawn from a vein using sterilized syringes, with a volume of 5 milliliters. Each sample was placed into a labeled tube. We divided the blood into two parts: one part was placed in a gel tube, and the other part was placed in an EDTA tube. The blood in the gel tube was left at room temperature for 10 to 15 minutes to allow for clotting, and then it was centrifuged at 4000g for 10 minutes to separate the serum, which was subsequently placed in a designated examination tube.

For the second portion of blood in the EDTA tube, the tube was gently shaken several times to prevent clotting, after which the sample was placed in the designated examination

device. The following tests were conducted at private biochemistry laboratories: random blood glucose (RBG) and lipid profile (total cholesterol, triglycerides, LDL, and HDL),renal function test (B.urea and cratinine),liver function test (ALP,AST,ALT),Insulin, lipase, lactate dehydrogenase, growth hormone and vitamin D3. Plasma and serum samples were stored at -20°C until they were used for further testing.

2.1.2 Inclusion Criteria:

The participants diagnosed with overweight or obesity, having a body mass index (BMI) greater than 25 and without chronic diseases, were included in this study.

Table (2-1) number of participant in this study

Sex	Number
Women	13
men	10
Girls	17
Boys	15

2.1.3 Exclusion criteria:

During the investigation, we collected blood samples along with other essential information. The exclusion criteria were as follows: any missing data samples, the presence of diabetes in patients, prior drug use that could impair β-cell functions or lower blood lipid levels, and/or the presence of liver or digestive diseases that could affect the absorption, transport, and breakdown of free fatty acids (FFAs).

2.1.4 Demographic Data:

Demographic data were collected, including gender (male or female), age, smoking status, alcohol consumption, duration of obesity, duration of diabetes, hereditary obesity, hypertension, angina, stroke, and other diseases. Additionally, information regarding medication and the family history of cardiovascular diseases was gathered.

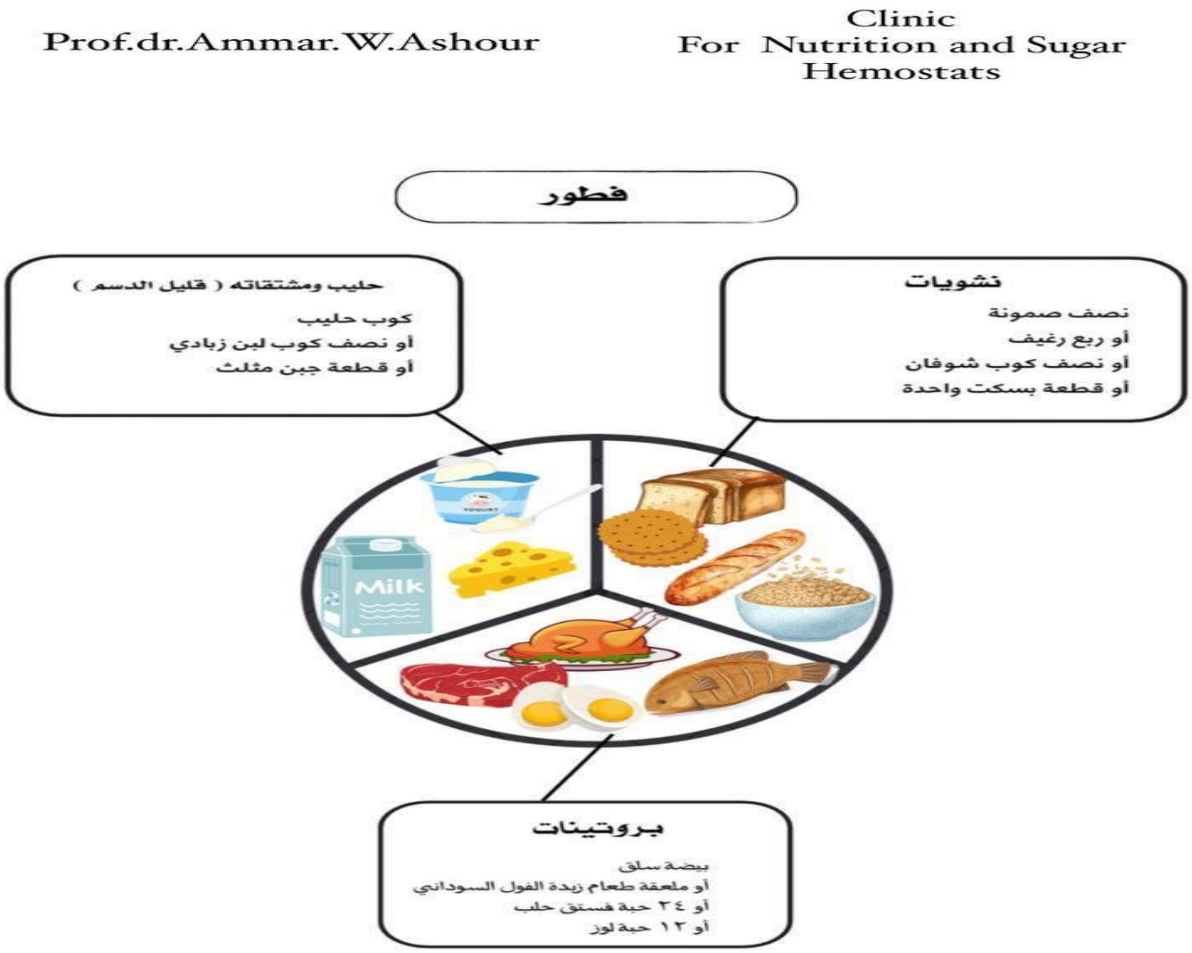
2.1.5 Anthropometric Measurements:

Measurements of height, weight, and waist circumference were recorded while subjects wore light clothing and no shoes. Body mass index (BMI, measured in kg/m^2) was recorded for all participants.

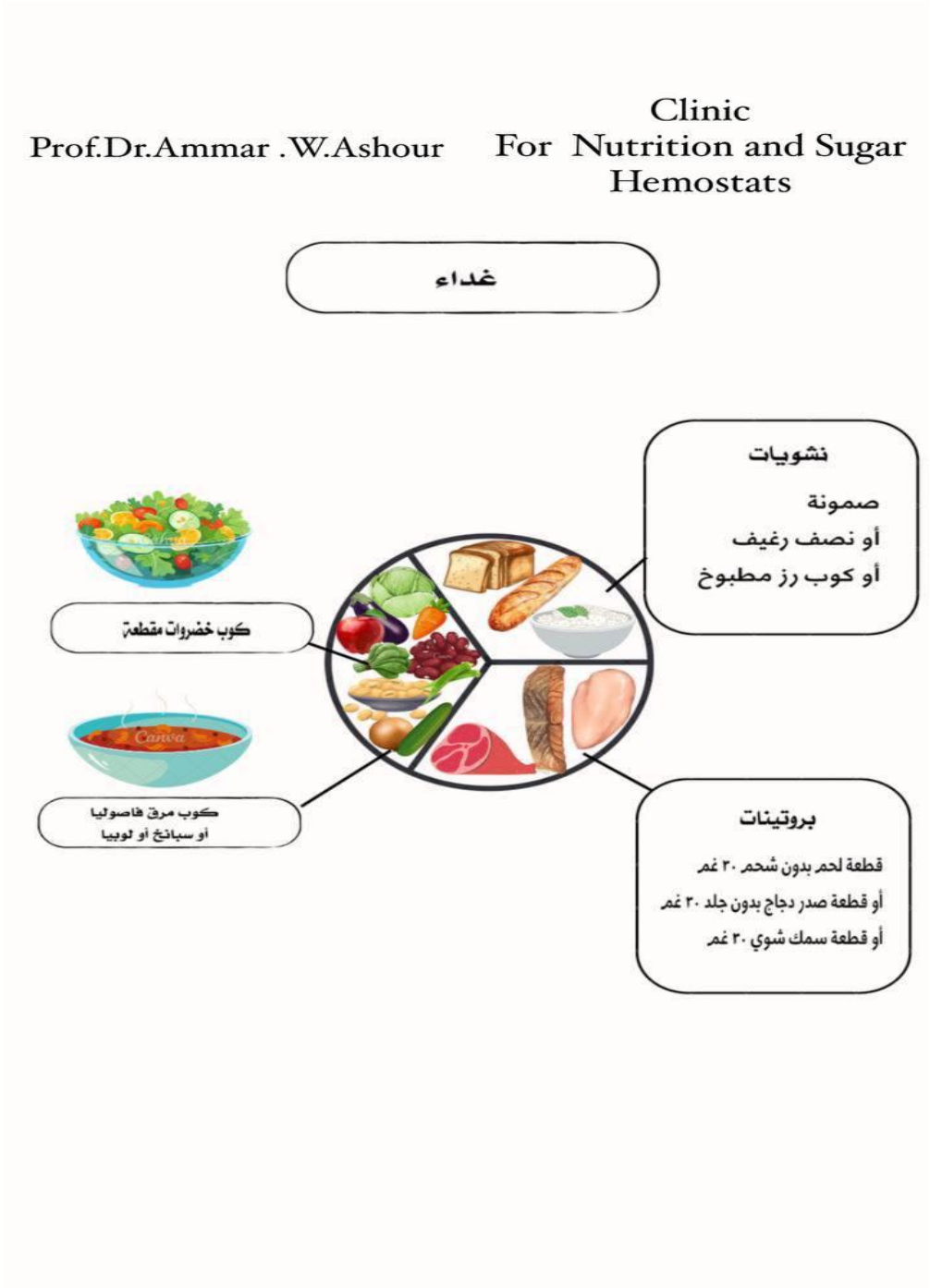
2.1.6 Dietary system:

the dietary system used in study was department from following:

1) Breakfast which consist from carbohydrate, protein and milk and diery products.



2)the lunch:


Clinic
Prof.Dr.Ammar .W.Ashour
For Nutrition and Sugar Hemostats
غداء
نشويات
صمونة
أو نصف رغيف
أو كوب رز مطبوخ
كوب خضروات مقطعة
كوب مرق فاصوليا
أو سبانخ أو لوبيا
بروتينات
قطعة لحم بدون شحم ٣٠ غم
أو قطعة صدر دجاج بدون جلد ٣٠ غم
أو قطعة سمك شوي ٣٠ غم

3)snack

Prof.Dr.Ammar .W.Ashour

Clinic
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Hemostats

وجبة خفيفة

فاكهة

كوب فاكهة مقطعة (رقي أو بطيخ)
أو تفاحة أو برتقالة أو موزة صغيرة
أو ٣٠ حبة عنب
أو خوخة أو كمثرى

4)the dinner:

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2.2 Materials and Instrumentation

2.2.1Chemicals and Materials

Table (2-2) below lists the chemicals along with their respective suppliers.

No	Chemicals	Supplier
1	Cobas-GLUC3 kit	Roche/Germany
2	Cobas -A1C kit	Roche/Germany
3	Cobas-TRIGL	Roche/Germany
4	Cobas-CHOL2	Roche/Germany
5	Cobas-HDLC3	Roche/Germany
6	Cobas-LDLC3	Roche/Germany
7	Cobas-UREAL	Roche/Germany
8	Cobas-CREATININE	Roche/Germany
9	Cobas-LDH	Roche/Germany
10	Cobas-insuline	Roche/Germany
11	Cobas-lipase	Roche/Germany

2.2.2. Instrumentation

The essential instruments are detailed in Table 2-2, highlighting their critical role in our analysis.

Table (2-3) The main instruments

No	The instrument	Company	Origin
1	Cobas Integra 400 plus	Roche –diagnostic	Germany
2	Cobas e 4 11	Roche –diagnostic	Germany
3	DRI-CHEM NX500	Fujifilm	Japan

4	Blood pressure instrument	Omron healthcare Co.	Japan
5	Centrifuge	Heraeus- lab200	Germany
6	Micro pipette	Dragon lab	China
7	Weight measuring device	Beurer GmbH	Germany
8	Refrigerator (-2-8c°)	Dia-lab	Denmark

2.3 Clinical Biochemistry Tests

2.3.1 Blood Glucose

The glucose concentration was precisely measured using the innovative Cobas e 411 system, ensuring accurate and reliable results.

2.3.1.1 1. Testing Principle

The exciting transformation of glucose to glucose-6-phosphate, catalyzed by hexokinase and powered by ATP, can be easily monitored using an ultraviolet spectrophotometer, highlighting the wonders of enzymatic reactions. (77)

$$\text{Glucose} + \text{ATP} \xrightarrow{\text{HC}} \text{G-6-P} + \text{ADP}$$

Glucose-6-phosphate transforms into gluconate-6-phosphate through the action of glucose-6-phosphate dehydrogenase, using NADP, all while keeping other carbohydrates safe from oxidation.

$$\text{G-6-P} + \text{NADP}^{+} \xrightarrow{\text{G-6-PDH}} \text{Gluconate-6-p} + \text{NADPH} + \text{H}^{+}$$

In this exciting reaction, more glucose means more NADPH formation, which it can easily observe using photometric techniques.

2.3.1.2 Assay

The glucose was measured using the following procedure: After collecting the blood in an SSGT tube, it was allowed to coagulate for 15 minutes. The serum was then separated by centrifugation at 4000g for 10 minutes and transferred to the device for analysis.

2.3.1.3 Reagents and Working Solutions

In this test, the working solutions included the following reagents:

R1 consists of:

- MES (2-(N-morpholino)ethane sulfonic acid) buffer: 5.0 mmol/L, pH 6.0

- Mg^{2+}: 24 mmol/L

- ATP: ≥ 4.5 mmol/L

- NADP: ≥ 7.0 mmol/L

- Preservative

R3 consists of:

- HEPES (4-(2-hydroxyethyl)-1-piperazineethanesulfonic acid) buffer: 200 mmol/L, pH 8.0

- Mg^{2+}: 4 mmol/L

- HK (Hexokinase from yeast): ≥ 300 μkat/L

- G-6-PDH (from E. coli): ≥ 300 μkat/L

- Preservative

2.3.1.4 Calculations

The glucose levels of each sample are automatically recorded by Roche/Hitachi Cobas c systems. Here are the classification categories for glucose levels:

- Normal value: 110–140 mg/dL

- Prediabetes: 140–200 mg/dL

- Diabetes: ≥ 200 mg/dL

The following conversion factors can be used for glucose level units:

- To convert from mmol/L to mg/dL: multiply by 18.02

- To convert from mmol/L to g/L: multiply by 0.1802

- To convert from mg/dL to mmol/L: multiply by 0.0555

2.3.2 Triglycerides TG

Triglycerides were accurately measured using the advanced Cobas Integra 400 Plus system, ensuring reliable and precise results.

2.3.2.1 Testing Principle

Triglycerides can be analyzed through enzymatic cleavage by lipoprotein lipase. The indicator is quinonimine, which is produced from 4-aminoantipyrine and 4-chlorophenol via hydrogen peroxide, under the catalytic action of peroxidase.

Triglycerides $\xrightarrow{LPL}$ Glycerol + fatty acid

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

Glycerol-3-phosphate + O_2 $\xrightarrow{GPO}$ Dihydroxyacetone phosphate + H_2O_2

2 H_2O_2 + Amino-antipyrine + 4-Chlorophenol $\xrightarrow{POD}$ Quinonimine + HCl + 4 H_2O

Where LPL is lipase, GK is glycerokinase, and GPO is glycerophosphate oxidase.

2.3.2.2 Assay

After collecting the blood in the SSGT tube, it was allowed to coagulate for 15 minutes. The serum was then separated by centrifugation at 4000g for 10 minutes before being placed in the device.

2.3.2.3 Reagents and Working Solutions

R1 consists of the following components:

- PIPES buffer: 50 mmol/L, pH 6.8
- Mg^{2+}: 40 mmol/L
- Sodium cholate: 0.20 mmol/L
- ATP: ≥ 1.4 mmol/L
- 4-Aminophenazone: ≥ 0.13 mmol/L
- 4-Chlorophenol: 4.7 mmol/L
- Lipoprotein lipase (from Pseudomonas species): ≥ 83 μkat/L
- Glycerokinase (from Bacillus stearothermophilus): ≥ 3 μkat/L
- Glycerol phosphate oxidase (from E. coli): ≥ 41 μkat/L
- Peroxidase (from horseradish): ≥ 1.6 μkat/L
- Preservatives and stabilizers

This formulation ensures optimal conditions for enzyme activity and stability.

2.3.2.4 Calculations

The serum triglyceride levels were monitored using the Cobas Integra 400 Plus system from Roche/Hitachi, which automatically calculates the concentration of triglycerides for each sample, displaying the results in either mg/dL or mmol/L.

According to Cleeman (2001), the normal value ranges for serum triglycerides are as follows:

- < 150 mg/dL: Normal
- 150-199 mg/dL: Borderline High
- 200-499 mg/dL: High

- ≥ 500 mg/dL: Very High

Unit Conversion: To convert mg/dL to mmol/L, use the following relationship:

mg/dL x 0.01126 = mmol/L.

2.3.3 Total Cholesterol (TC)

Each participant's total cholesterol level was assessed with the advanced Cobas-Integra 400 plus system, ensuring precision and reliability in our measurements.

2.3.3.1 Testing Principle

This method for measuring total cholesterol in serum utilizes three enzymes: cholesterol esterase (CE), cholesterol oxidase (CO), and peroxidase (POD). Cholesterol esterase breaks down cholesterol esters, while cholesterol oxidase converts cholesterol into cholest-4-en-3-one and hydrogen peroxide. In the presence of hydrogen peroxide, phenol and 4-aminoantipyrine (4-AA) react to form a quinoneimine dye. The intensity of this dye is proportional to the concentration of cholesterol in the serum sample.

$$\text{Cholesteryl esters} \xrightarrow{CE} \text{Cholesterol + Fatty acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow{CO} \text{Cholestenone} + H_2O_2$$

$$4\ \text{Aminoantipyrine} + \text{Phenol} + H_2O_2 \xrightarrow{POD} \text{Quinoneimine} + 4H_2O$$

2.3.3.2 Assay

After collecting blood in the SSGT tube, it was allowed to clot for 15 minutes. The serum was then separated by centrifugation at 4000g for 10 minutes before being placed in the device.

2.3.3.3 Reagents and Working Solutions

R1 consists is a dynamic formulation featuring a variety of essential components designed for optimal performance! It includes:

- PIPES (1,4-Piperazinediethanesulfonic acid) buffer: 225 mmol/L at a pH of 6.8
- Mg^{2+}: 10 mmol/L
- Sodium cholate: 0.6 mmol/L

- 4-Aminophenazone: ≥ 0.45 mmol/L
- Phenol: ≥ 12.6 mmol/L
- Fatty alcohol polyglycol ether: 3%
- Cholesterol esterase (Pseudomonas species): ≥ 25 μkat/L (≥ 1.5 U/mL)
- Cholesterol oxidase (*E. coli*): ≥ 7.5 μkat/L (≥ 0.45 U/mL)
- Peroxidase (horseradish): ≥ 12.5 μkat/L (≥ 0.75 U/mL)
- Stabilizers
- Preservative

2.3.3.4 Calculations

The cholesterol levels in serum were measured using the Cobas Integra 400 plus system from Roche/Hitachi, which automatically calculates the concentration of each sample's analyte, reported either in mg/dL or mmol/L.

The normal values for serum cholesterol are as follows:

- Less than 200 mg/dL: Desirable
- 200-239 mg/dL: Borderline
- 240 mg/dL and above: High

2.3.4. High Density Lipoprotein Cholesterol (HDL-C)

We conducted a precise and thorough analysis of HDL levels in serum, leveraging the cutting-edge technology of the Cobas-integra 400 plus system to ensure the highest accuracy and reliability in our measurements.

2.3.4.1 Testing Principle

The basic principle of monitoring HDL (high-density lipoprotein) levels is as follows: In the presence of magnesium ions, dextran sulfate selectively forms water-soluble complexes that include lipoproteins. This process is combined with polyethylene glycol (PEG) and modified enzymes such as cholesterol oxidase and cholesteryl esterase.

The cholesterol level in HDL is determined enzymatically using cholesterol esterase and cholesterol oxidase, along with approximately 40 percent PEG. Cholesterol esters are quantitatively broken down by cholesterol esterase into free cholesterol and fatty acids.

2.3.4.2 Assay

After collecting blood in the SSGT tube, it was allowed to clot for 15 minutes. The serum was then separated by centrifugation at 4000g for 10 minutes before being placed in the device.

2.3.5.3 Reagents and Working Solutions

R1 consists of the following components:

- HEPES (4-(2-hydroxyethyl)-1-piperazineethanesulfonic acid) buffer: 10.07 mmol/L
- CHES (N-Cyclohexyl-2-aminoethanesulfonic acid): 96.95 mmol/L, pH 7.4
- Dextran sulfate: 1.5 g/L
- Magnesium nitrate hexahydrate: > 11.7 mmol/L
- HSDA (sodium N-(2-hydroxy-3-sulfopropyl)-3,5-dimethoxyaniline): 0.96 mmol/L
- Ascorbate oxidase (Eupenicillium sp., recombinant): > 50 μkat/L
- Peroxidase (horseradish): > 16.7 μkat/L
- Preservative

R2 contains:

- HEPES buffer: 10.07 mmol/L, pH 7.0
- PEG-cholesterol esterase (Pseudomonas species): > 3.33 μkat/L
- PEG-cholesterol oxidase (Streptomyces sp., recombinant): > 127 μkat/L
- Peroxidase (horseradish): > 333 μkat/L
- 4-aminoantipyrine: 2.46 mmol/L
- Preservative

2.3.4.4 Calculations

The HDL (High-Density Lipoprotein) levels were measured using the Cobas-integra 400 plus system from Roche/Hitachi, with the concentration of HDL automatically calculated for each sample.

The normal values for HDL in serum are as follows:

- Less than 40 mg/dL: Major Risk Factor for Heart Disease
- 60 mg/dL or higher: Negative Risk Factor for Heart Disease

Conversion factors are as follows:

- To convert from mmol/L to mg/dL, multiply by 38.66
- To convert from mmol/L to g/L, multiply by 0.3866
- To convert from mg/dL to mmol/L, multiply by 0.0259

2.3.5. Low Density Lipoprotein LDL Cholesterol(LDL-C)

We expertly assessed LDL-cholesterol levels with the cutting-edge Cobas-Integra 400 plus system, guaranteeing unparalleled precision and confidence in the results.

2.3.5.1 Testing Principle

The LDL test is an exciting and innovative method for measuring cholesterol levels. By using a homogeneous enzymatic colorimetric assay, this method specifically targets cholesterol esters and free cholesterol found in low-density lipoprotein (LDL). It utilizes the powerful enzymes cholesterol esterase and cholesterol oxidase, along with special surfactants that gently isolate LDL particles, ensuring accuracy. Meanwhile, other lipoproteins, such as HDL, VLDL, and chylomicrons, are not included in this test, allowing for a focused analysis. During testing, cholesterol esters are expertly transformed into free cholesterol and fatty acids thanks to cholesterol esterase, while the reactions are thoughtfully inhibited by surfactants and a sugar compound, providing reliable results.

1) LDL-cholesterol esters + H_2O $\xrightarrow{\text{cholesterol esterase}}$ cholesterol + free fatty acid

In the presence of oxygen, cholesterol is oxidized by cholesterol oxidase to 4-cholestenone and hydrogen peroxide.

2) LDL-Cholesterol +O_2 $\xrightarrow{\text{cholesterol oxidase}}$ 4-cholestenone +H_2O_2

When peroxidase is present, hydrogen peroxide interacts with 4-aminoantipyrine and EMSE to create a vibrant red-purple dye, as illustrated in equation (3). The intensity of this beautiful dye is directly linked to cholesterol concentration, allowing us to measure it photometrically. This exciting process not only highlights the importance of chemistry but also provides a precise method for assessing cholesterol levels.

$2H_2O_2$ +4-aminoantipyrine + (EMSE) + H_2O +H^+ $\xrightarrow{\text{Peroxidase}}$ red purple pigment +5 H_2O

2.3.5.2 Assay

We collected the blood in the SSGT tube and allowed it to clot for 15 minutes. Then, we separated the serum by centrifuging it at 4000g for 10 minutes, ready to be placed in the device for further analysis.

2.3.5.3 Reagents and Working Solutions

R1: Bis(tris(2-hydroxyethyl)amino)tris(hydroxymethyl)methane buffer

- Concentration: 20.1 mmol/L
- pH: 7.0
- 4-aminoantipyrine: 0.98 mmol/L
- Ascorbic oxidase (AOD, Acremonium species): ≥ 66.7 μkat/L
- Peroxidase (recombinant from Basidiomycetes): ≥ 166.7 μkat/L
- Bovine serum albumin (BSA): 4.0 g/L
- Preservative: included

R2: MOPS (3-morpholinopropane-1-sulfonic acid) buffer

- Concentration: 20.1 mmol/L
- pH: 7.0
- EMSE: 2.16 mmol/L
- Cholesterol esterase (Pseudomonas species): ≥ 33.3 μkat/L
- Cholesterol oxidase (recombinant from E. coli): ≥ 31.7 μkat/L

- Peroxidase (recombinant from Basidiomycetes): ≥ 333.3 μkat/L
- Bovine serum albumin (BSA): 4.0 g/L
- Detergents: included
- Preservative: included

2.3.5.4 Calculations

The LDL levels for each sample were automatically recorded using the Cobas-INTEGRA 400 PLUS system from Roche/Hitachi.

Normal Value: Up to 160 mg/dL.

Conversion factors: mmol/L × 38.66 = mg/dL and mmol/L × 0.3866 = g/L.

2.3.6 Determination of blood Urea Level

2.3.6.1 Principle:

The urea nitrogen assay is an exciting evolution of a fully enzymatic procedure pioneered by Talke and Schubert back in 1965. This dynamic test operates as a kinetic assay, maintaining a linear reaction rate for a specific time, allowing us to capture accurate results.

In this fascinating process, urea in the sample is skillfully hydrolyzed by urease, generating ammonia and carbon dioxide. Then, glutamate dehydrogenase (GLD) steps in, transforming ammonia and α-ketoglutarate into glutamate and water while oxidizing reduced nicotinamide adenine dinucleotide (NADH) to nicotinamide adenine dinucleotide (NAD).

It is truly remarkable that for every mole of urea present, two moles of NADH are oxidized. It can easily determine the urea concentration in the sample by monitoring the initial rate of decrease in absorbance at 340 nm.

$$\text{Urea} + 2H2O \xrightarrow{\text{urease}} 2NH4^{+} + CO3^{-2}$$

$$NH4{+}1 + 2\text{-oxoglutarate} + NADH \xrightarrow{GLDH} \text{L-glutamate} + NAD{+}{+} + H_2O$$

The reduction in NADH levels is closely linked to the concentration of urea present in the specimen; as urea levels rise, we see a corresponding decrease in NADH. This relationship highlights the intricate connection between these two compounds in biological processes.

2.3.6.2 Assay

After collecting the blood in the SSGT tube, it was allowed to clot for 15 minutes. Following this, the serum was separated by centrifugation at 4000g for 10 minutes before being placed in the device.

2.3.6.3 Reagents and Working Solutions

Table (2.4) Reactive ingredients of serum urea level

Concentration	Reactive ingredients
2.95mmol/L	R1 NADH
99.8 mmol/L	R2 α-ketoglutaric acid
23.5KU/L	urease (jack bean)
63.5 KU/L	GLD (beef liver)
7.6 mmol/L	Adenosine diphosphate

2.3.6.4 Calculations

The blood urea level for each sample was recorded automatically using the Cobas INTEGRA 400 PLUS system from Roche/Hitachi.

Normal range: male (8-24 mg/dL), female (6-21 mg/dL).

2.3.7 Determination of serum Creatinine Level

2.3.7.1 Principle:

At an alkaline pH, creatinine in the sample reacts with picrate to form a creatinine-picrate complex. The rate of increase in absorbance at 500 nm, resulting from the

formation of this complex, is directly proportional to the concentration of creatinine in the sample. Creatinine is the end product of the metabolism of creatine and creatine phosphate. Creatine, a nitrogenous organic acid, is primarily produced in the kidneys and liver, with some production also occurring in the pancreas, using three amino acids: glycine, arginine, and methionine.

Creatinine + picric acid $\xrightarrow{\text{Alkaline ph}}$ yellow-orange complex

2.3.7.2 Assay

After collecting blood in the SSGT tube, it was allowed to clot for 15 minutes. Subsequently, the serum was separated by centrifugation at 4000g for 10 minutes before being placed in the device.

2.3.7.3 Reagents and Working Solutions

Table (2-5) reactive ingredients of serum creatinine level

Reactive ingredients	Concentration
R1 sodium hydroxide	0.8 mol/L
R2 picric acid	24 mmol/L

2.3.7.4 Calculations

The creatinine level for each sample was automatically recorded using the Cobas-INTEGRA 400 PLUS system from Roche/Hitachi.

Normal ranges are as follows: male (0.74-1.35 mg/dl), female (0.59-1.1 mg/dl).

2.3.8 Determination of serum Alanine Aminotransferase(ALT)

2.3.8.1 Principle:

Alanine aminotransferase (ALT) catalyzes the reversible transfer of an amino group from L-alanine to 2-oxoglutarate, resulting in the formation of pyruvate and L-glutamate.

The pyruvate produced is subsequently reduced to L-lactate by lactate dehydrogenase (LDH), which simultaneously oxidizes NADH to NAD^+.

The decrease in absorbance caused by the consumption of NADH is measured photometrically at 340 nm and is directly proportional to the ALT activity in the sample.

L-alanine + 2-oxoglutarate → (ALT) → pyruvate + L-glutamate

Pyruvate + NADH + H^+ → (LDH) → L-lactate + NAD^+

2.3.8.2 Assay

After collecting the blood in the SSGT tube, it was allowed to clot for 15 minutes. The serum was then separated by centrifugation at 4000g for 10 minutes before being placed in the device.

2.3.8.3 Reagents and Working Solutions

A dry chemistry analyzer is a process where a liquid test sample is applied directly to a dry reagent strip, which is tailored for each specific project. In this method, the moisture from the sample serves as a solvent, initiating a specific chemical reaction that facilitates the chemical analysis.

2.3.8.4 Calculations

The ALT levels for each sample were semi-automatically recorded using the FUJIFILM NX 500 instrument.

Normal values are: male: 10-40 U/L; female: 7-35 U/L.

2.3.9 Determination of serum Aspartate Aminotransferase (AST)

2.3.9.1 Principle:

Aspartate aminotransferase (AST) facilitates the transfer of an amino group from L-aspartate to 2-oxoglutarate, resulting in the formation of oxaloacetate and L-glutamate. Subsequently, oxaloacetate is converted into L-malate by malate dehydrogenase (MDH), during which NADH is oxidized to NAD^+.

The decrease in absorbance at 340 nm, caused by the oxidation of NADH, is directly proportional to the activity of AST.

L-aspartate + 2-oxoglutarate → (AST) → oxaloacetate + L-glutamate

Oxaloacetate + NADH + H^+ → (MDH) → L-malate + NAD^+

2.3.9.2 Assay

After collecting blood in the SSGT tube, it was left to clot for 15 minutes. The serum was then separated by centrifugation at 4000g for 10 minutes before being placed in the device.

2.3.9.3 Reagents and Working Solutions

A dry chemistry analyzer is a process in which a liquid test sample is added directly to a dry reagent strip manufactured specifically for each project. The dry chemistry analyzer uses the moisture of the sample being tested as a solvent to cause a specific chemical reaction that leads to a chemical analysis.

2.3.9.4 Calculations

The (AST) level for each sample was semi-automatically recorded using FUJIFILM NX 500 instrument.

Normal value:

male: 10-40 U/L

female: 9-32 U/L

2.3.10 Determination of serum Alkaline Phosphatase (ALP)

2.3.10.1 Principle:

Alkaline phosphatase (ALP) catalyzes the hydrolysis of p-nitrophenyl phosphate (pNPP) under alkaline conditions, producing p-nitrophenol, a yellow-colored compound.

The formation of p-nitrophenol is measured spectrophotometrically at 405 nm. The increase in absorbance is directly proportional to ALP activity.

p-Nitrophenyl phosphate + H_2O → (ALP) → p-Nitrophenol + phosphate

2.3.10.2Assay

After collecting the blood in the SSGT tube, it was left to clot for 15 minutes, after that the serum was separated by centrifugation at 4000g for 10 minutes to be placed in the device.

2.3.10.3Reagents and Working Solutions

A dry chemistry analyzer is a process in which a liquid test sample is added directly to a dry reagent strip manufactured specifically for each project. The dry chemistry analyzer uses the moisture of the sample being tested as a solvent to cause a specific chemical reaction that leads to a chemical analysis.

2.3.10.4 Calculations

The (AST) level for each sample was semi-automatically recorded using FUJIFILM NX 500 instrument.

Normal value:

male: 40-130 U/L

female: 35-105 U/L

teenagers : 100-300U/L

2.3.11 LIPASE

Enzymatic Lipase was directly measured in serum using Cobas e 411 system.

2.3.11.1 Test Principle

Enzymatic colorimetric assay with 1,2-O-dilauryl-rac-glycero-3-glutaric-acid-(6-methylresorufin) ester as substrate.

The chromogenic lipase substrate 1,2-O-dilauryl-rac-glycero-3-glutaric-acid-(6-methylresorufin) ester is cleaved by the catalytic action of alkaline lipase solution to form 1,2-O-dilauryl-rac-glycerol and an unstable intermediate, glutaric acid-(6-methylresorufin) ester.

This decomposes spontaneously in alkaline solution to form glutaric acid and methylresorufin. Addition of detergent and colipase increases the specificity of the assay for pancreatic lipase.

1,2-O-dilauryl-rac-glycero-3-glutaric acid-(6-methylresorufin) ester $\xrightarrow{\text{lipase}}$ 1,2-O-dilauryl-rac-glycerol + glutaric acid-(6-methylresorufin) ester

glutaric acid-(6-methylresorufin) ester $\xrightarrow{\text{spontaneous decomposition}}$ glutaric acid +methylresorufin

The color intensity of the red dye formed is directly proportional to the lipase activity and can be determined photometrically.

2.3.11.2 Assay

After collecting the blood in the SSGT tube, it was left to clot for 15 minutes, after that the serum was separated by centrifugation at 4000g for 10 minutes to be placed in the device**.**

2.3.11.3 Reagents and Working Solutions

R1 BICINa) buffer: 50 mmol/L, pH 8.0; colipase (porcine pancreas):
≥ 0.9 mg/L; Na-deoxycholate: 1.6 mmol/L; calcium chloride:
10 mmol/L; detergent; preservative

R2 Tartrate buffer: 10 mmol/L, pH 4.16;

1,2-O-dilauryl-rac-glycero-3-glutaric acid-(6-methylresorufin) ester:

0.27 mmol/L; taurodeoxycholate: 8.8 mmol/L; detergent**;**Preservative

a) BICIN = N,N-bis(2-hydroxyethyl)glycine

2.3.11.4 Calculations

Cobas e 411 systems automatically calculate the analyte activity of each sample.

Normal range:

- 10-140 U/L adult younger than 60
- 24-151 U/L adult age 60 and older

2.3.12 Lactate Dehydrogenase (LDH)

Enzymatic Lactate Dehydrogenase was directly measured in serum using Cobas e 411 system.

2.3.12.1 Test Principle

Lactate dehydrogenase catalyzes the conversion of L-lactate to pyruvate; NAD is reduced to NADH in the process.

L-Lactate + NAD+ $\xrightarrow{\text{LDH}}$ Pyruvate + NADH + H+

The initial rate of the NADH formation is directly proportional to the catalytic LDH activity. It is determined by photometrically measuring the increase in absorbance**.**

2.3.12.2Assay

After collecting the blood in the SSGT tube, it was left to clot for 15 minutes, after that the serum was separated by centrifugation at 4000g for 10 minutes to be placed in the device**.**

2.3.12.3 Reagents and Working Solutions:

R1 N-methylglucamine: 400 mmol/L, pH 9.4 (37 °C); lithium lactate:

62 mmol/L; stabilizers

R2 NAD: 62 mmol/L; stabilizers; preservatives

R1 is in position B and R2 is in position C**.**

2.3.12.4 Calculations

Cobas e 411 systems automatically calculate the analyte activity of each sample.

Normal range:

- Males: 135-225 U/L
- Females: 135-214 U/L

2.3.13 Insulin

Enzymatic Insulin was directly measured in serum using Cobas e 411 system**.**

2.3.13.1 Test Principle

Insulin is a 51-residue peptide hormone with a molecular weight of 5808 Da. It is secreted by the β-cells of the islets of Langerhans in the pancreas, and passes into circulation via the portal vein and the liver.
Insulin is generally released in pulses.

The biologically active insulin molecule is monomeric and consists of two polypeptide chains, the 21 amino acid α-chain and the 30 amino acid β-chain joined by disulphide bridges. Insulin is the biosynthetic product of the single-chain precursor preproinsulin, which is subsequently cleaved to give proinsulin.2,3,4,5 Specific proteases further cleave proinsulin to produce insulin and the connecting (C)-peptide which pass into the bloodstream simultaneously in equimolar concentrations. Circulating insulin has a half-life of 3-5 minutes and is preferentially retained and degraded in the liver.

Therefore only about half of the insulin reaches the systemic circulation. Inactivation or excretion of pro insulin and C-peptide mainly takes place in the kidney and virtually none of the C-peptide is retained in the liver. As a result, C-peptide has a higher plasma concentration than insulin.

The amino acid sequence of insulin is extremely well conserved, with the result that prior to the development of genetically engineered human insulin it was possible to successfully use porcine or bovine insulin in the therapy of diabetes mellitus.

The action of insulin is mediated by specific receptors and primarily consists of facilitation of glucose uptake by the cells of the liver, fatty tissue and musculature; this is the basis of its hypoglycemic action.

2.3.13.2 Assay

After collecting the blood in the SSGT tube, it was left to clot for 15 minutes, after that the serum was separated by centrifugation at 4000g for 10 minutes to be placed in the device

2.3.13.3 Reagents and Working Solutions:

M Streptavidin-coated microparticles (transparent cap), 1 bottle, 6.5 mL:
Streptavidin-coated microparticles 0.72 mg/mL; preservative.

R1 Anti-insulin-Ab~biotin (gray cap), 1 bottle, 10 mL:
Biotinylated monoclonal anti-insulin antibody (mouse) 1 mg/L; MES[1])
buffer 50 mmol/L, pH 6.0; preservative.

R2 Anti-insulin-Ab~Ru(bpy) (black cap), 1 bottle, 10 mL:
Monoclonal anti-insulin antibody (mouse) labeled with ruthenium
complex 1.75 mg/L; MES buffer 50 mmol/L, pH 6.0; preservative**.**

1) MES = 2-morpholino-ethane sulfonic acid

2.3.13.4 Calculation

The analyzer automatically calculates the analyte concentration of each sample (either in µU/mL or pmol/L).

Normal range:

- 2-20 µU/mL for fasting
- 60-180 µU/mL after meal

2.3.14 Determination of Vitamin D3 Level

2.3.14.1 Principle:

Vitamin D total III assay is based on a competitive electrochemiluminescence immunoassay (ECLIA).

It quantitatively determines 25-hydroxyvitamin D [$25(OH)D_2$ and $25(OH)D_3$] levels in serum or plasma.

Reaction Steps:

1. Pretreatment Phase:

• 25(OH)D is released from vitamin D binding protein (VDBP) in the serum using a reagent buffer.

2. First Incubation:

• The released 25(OH)D binds with a ruthenium-labeled VDBP (vitamin D binding protein).

3. Second Incubation:

• A biotinylated 25(OH)D analog and streptavidin-coated microparticles are added.

• Unbound ruthenium-labeled VDBP binds to the biotinylated analog, forming immune complexes.

• These complexes attach to microparticles through biotin–streptavidin interaction.

4. Measurement (Detection Phase):

• The immune complexes are magnetically captured on an electrode.

• Application of voltage induces electrochemiluminescence from the ruthenium complex.

• The emitted light is measured:

Signal intensity is inversely proportional to the concentration of 25(OH)D in the patient sample.

2.3.14.2 Assay

After collecting the blood in the SSGT tube, it was left to clot for 15 minutes, after that the serum was separated by centrifugation at 4000g for 10 minutes to be placed in the device

2.3.14.3 Reagents and Working Solutions

• R1 (Reagent 1):

• Composition: Ruthenium-labeled vitamin D binding protein (150 µg/L); bis-tris propane buffer (200 mmol/L); human albumin (25 g/L); pH 7.5; preservative

• Volume: 18.8 mL

• Function: Binds to 25(OH)D in the sample, forming a complex detectable via electrochemiluminescence .

• R2 (Reagent 2):

• Composition: Biotinylated 25-hydroxyvitamin D (20 µg/L); bis-tris propane buffer (200 mmol/L); pH 8.6; preservative

• Volume: 15.8 mL

• Function: Competes with sample 25(OH)D for binding sites, facilitating the competitive assay mechanism .

• M (Microparticles):

• Composition: Streptavidin-coated microparticles (0.72 mg/mL); preservative

• Volume: 12.4 mL

• Function: Captures biotinylated complexes, allowing magnetic separation and detection .

2.3.14.4 Calculations

Cobas e 411 systems automatically calculate the analyte activity of each sample.

Normal value: <20ng/mL Deficient

20-30 ng/mL insufficient

30-50 ng/mL sufficient

>100 ng/mL potential toxicity

2.3.15 Determination of Growth Hormone Level

2.3.15.1 Principle:

1. Sample Incubation:

• The patient's serum or plasma sample is incubated with a biotinylated monoclonal anti-GH antibody and a ruthenium-labeled monoclonal anti-GH antibody.

• These two antibodies bind to different epitopes on the GH molecule, forming a sandwich complex.

2. Magnetic Particle Addition:

• After the complex forms, streptavidin-coated magnetic microparticles are added.

• These particles bind to the biotin-labeled antibody, enabling the entire sandwich complex to be captured magnetically.

3. Magnetic Separation:

• The mixture is transferred into the measurement cell.

• A magnetic field holds the microparticles on the electrode surface, while unbound substances are washed away.

4. Electrochemiluminescence Measurement:

• A voltage is applied, triggering a light-emitting reaction from the ruthenium complex.

• The light signal is directly proportional to the concentration of GH in the sample.

2.3.15.2 Assay:

After collecting the blood in the SSGT tube, it was left to clot for 15 minutes, after that the serum was separated by centrifugation at 4000g for 10 minutes to be placed in the device.

2.3.15.3 Reagents and Working Solutions

1. Reagent R1 (Antigen-Antibody Reaction Components):

• Ruthenium-labeled Anti-hGH Antibody: Monoclonal antibody specific to hGH, labeled with a ruthenium complex (Tris(2,2'-bipyridyl)ruthenium(II)).

• Buffer Solution: Maintains optimal pH and ionic strength for antigen-antibody interactions.

2. Reagent R2 (Microparticle Components):

• Biotinylated Anti-hGH Antibody: Monoclonal antibody specific to a different epitope of hGH, labeled with biotin.

• Streptavidin-coated Microparticles: Facilitate the capture of the biotinylated antibody-hGH complex onto the solid phase.

• Buffer Solution: Ensures stability and proper reaction conditions.

2.3.15.4 Calculations:

Cobas e 411 systems automatically calculate the analyte activity of each sample**.**

Normal value:

Male: 0.01- 2.47 ng/mL

Female: 0.01- 10 ng/mL

Teenagers: 10-50 ng/mL

2.4. Statistical Analysis

The data for this study were analyzed and presented as mean ± standard deviation. The analysis was conducted using the Statistical Package for the Social Sciences (SPSS) software version 26, provided by International Business Machines Corporation (IBM), New York, United States. Mean differences between subjects with obesity and healthy volunteers with normal distributions were assessed using Student's t-test for independent samples. Additionally, the analysis of monitoring biomarkers was performed using analysis of variance (ANOVA). Pearson's correlation test was used to examine various correlations. Two-tailed P-values were calculated, and statistical significance was considered at $P < 0.05$.

CHAPTER THREE

Results and Discussion

Chapter Three: Results and Discussion

3.1 Demographic Data of Studied Participants

The study comprised 56 patients (male/female ratio = 25/31) and 60 healthy adults (male/female ratio = 30/30), as shown in Table (3-1)

Table (3-1) Details of collected samples.

Samples	Number of samples	percent
Obese (female)	13	24.2%
Obese (male)	10	18.8%
Obese (girls)	17	30.3%
Obese (boys)	15	26.7%

All participants in this study had their age and gender recorded, along with other relevant information, using a standard questionnaire (see Appendix 1). The observed data indicated that there were no significant differences in mean age between the participants with obesity (mean age = 32.4 ± 11.34) and the healthy volunteers (mean age = 29.53 ± 9.6). The median age of the participants was 30 years, with an age range extending from 13 to 53 years.

3.2 Results of Body Composition

The body composition of all participants in this study was assessed through measurements of height, weight, waist circumference, and body mass index.

3.2.1Height and Weight

The statistical analysis of changes in anthropometric and biochemical measurements in response to dietary treatment is presented in Table 2. After three months of dietary intervention, all variables demonstrated significant improvements, regardless of diet type or sex. This indicates that the diets were effective in reducing anthropometric and biochemical parameters for both women and men.

The analysis of the data regarding height and weight revealed the following findings: the mean height of obese participants was 165.58 ± 10.62 cm, while the mean height of healthy volunteers was 161.84 ± 9.18 cm. Additionally, the mean weight of obese participants was 105.62 ± 23.79 kg, compared to a mean weight of 59.85 ± 6.04 kg for healthy volunteers.

The findings revealed that while height didn't vary significantly between obese participants and healthy volunteers, there was a notable difference in weight. This highlights the unique factors influencing body composition.

Table (3. 2) General subjects characteristics

Variable	Obesity	Mean	Std. Deviation	P. Value
Age	Healthy	29.53	9.96	0.13
	Obese	32.4	11.34	
weight	Healthy	59.85	6.04	0.000
	Obese	105.62	23.79	
height	Healthy	161.84	9.18	0.453

	Obese	165.58	10.62	
BMI	Healthy	20.91	2.13	0.000
	Obese	34.23	5.37	
Weight circumference	Healthy	80.89	5.47	0.132
	Obese	91.42	4.86	

3.2.2. Waist Circumference (WC)and Body Mass Index (BMI)

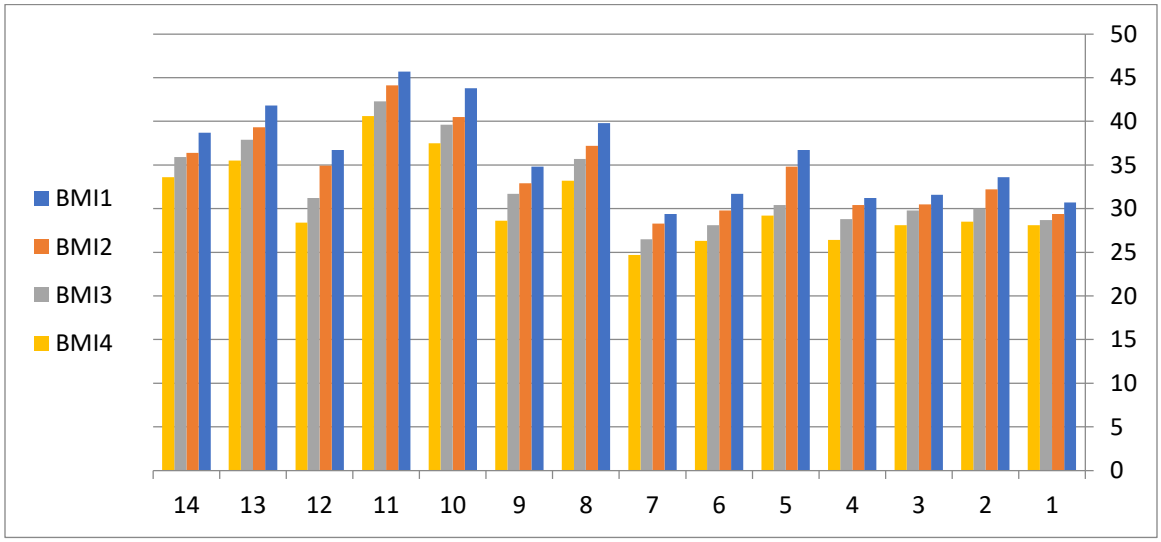
There is a trend indicating no significant difference in waist circumference (WC) between obese participants (91.42 ± 4.86 cm) and healthy volunteers (80.89 ± 5.4 cm), with a P value of 0.132. However, there is a significant difference in body mass index (BMI) between obese participants (34.23 ± 5.37 kg/m²) and healthy volunteers (20.91 ± 2.13 kg/m²).

Differences in BMI were also observed based on sex. In women, the analysis of changes after dietary intervention revealed a significant difference in BMI for adult women (P value = 0.026) and for girls (P value = 0.0009). In contrast, there was no significant difference in BMI for men (P value = 0.32), although boys did show a significant difference (P value = 0.001).

The overall analysis indicates a significantly greater decrease in BMI among both women and girls following the dietary intervention, while men showed a lesser decrease compared to the other groups.

BMI showed a strong association with age, which aligns with findings from prior studies (118). In the teenage group (both boys and girls) aged 13 to 17 years, there

was a notable decrease in BMI compared to the adult group, as illustrated in the figures below.



Figure(3-1) the difference in BMI among 4 months in women.

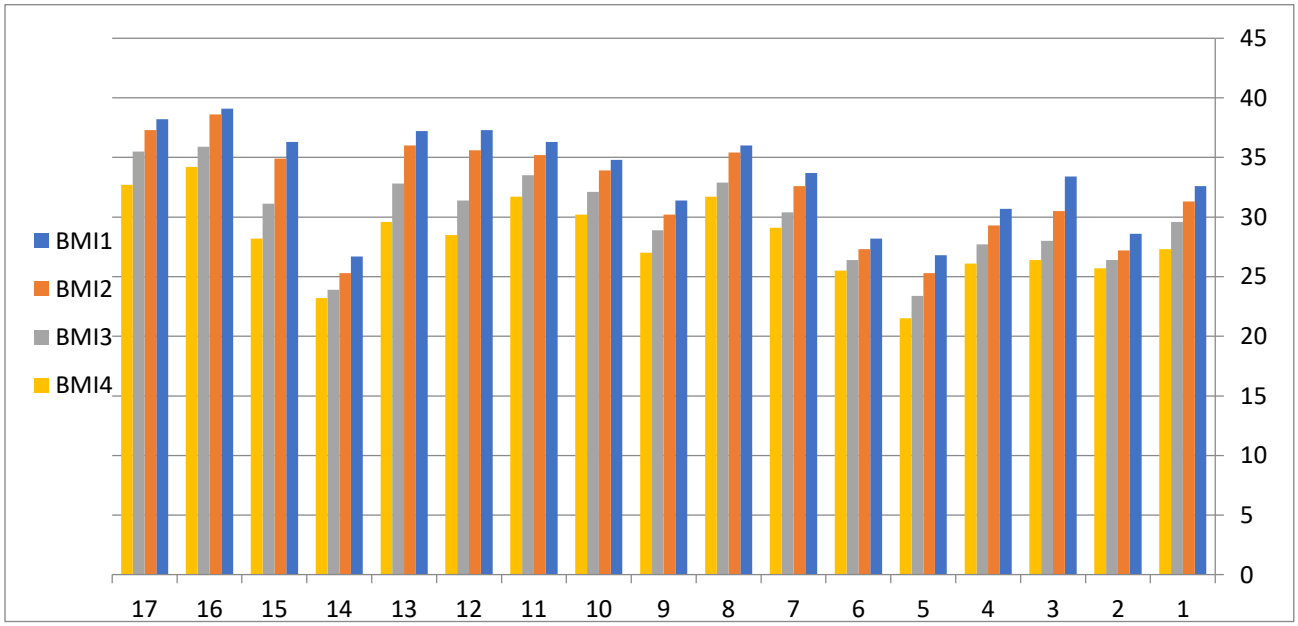


Figure (3-2) the difference in BMI among 4 months in girls.

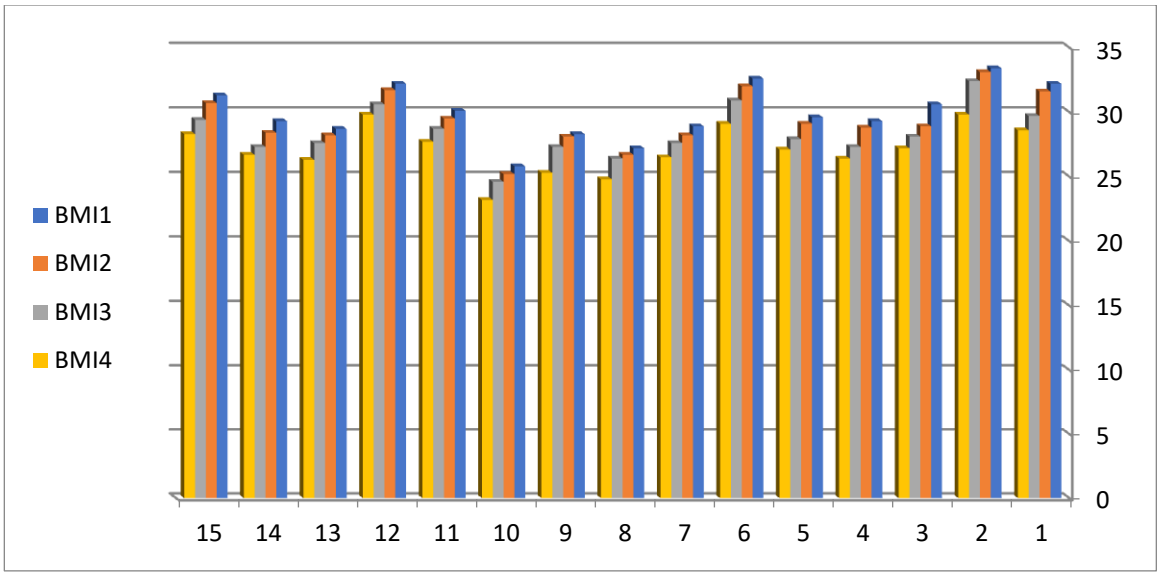


Figure (3-3) shows the difference in BMI among 4 months in boys.

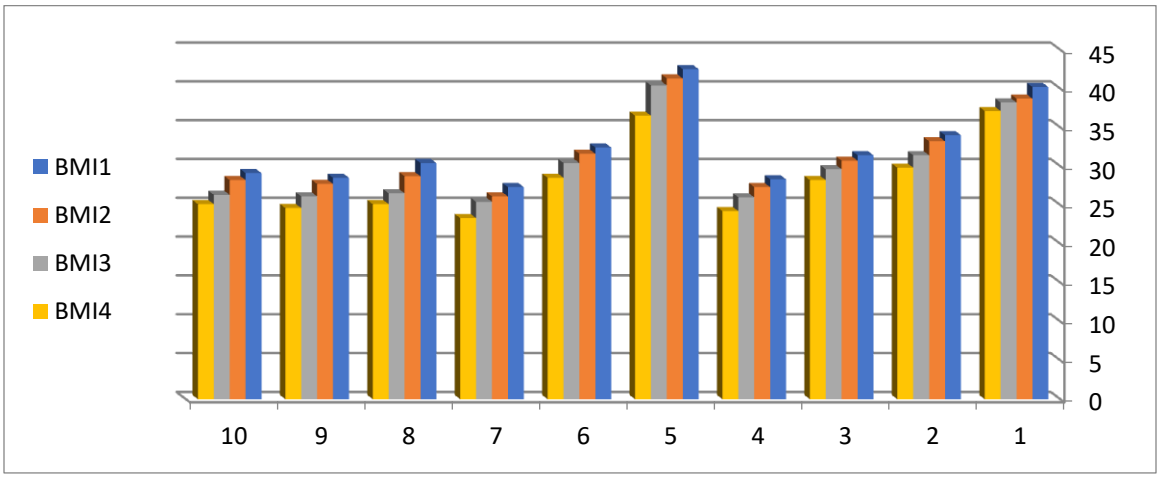


Figure (3-4)) the difference in BMI among 4 months in men.

3.3 The impact of dietary systems on reducing diabetes

The increasing prevalence of type 2 diabetes, a chronic condition marked by insufficient insulin production or ineffective insulin utilization, is closely linked to obesity. One of the main symptoms of this disease is elevated blood glucose

levels. Key characteristics of type 2 diabetes include peripheral insulin resistance and inadequate insulin release from beta cells, resulting in elevated fatty acid levels. This situation results in increased fat breakdown, heightened glucose production by the liver, and reduced glucose uptake by muscle cells.(78)

In the obese group, several parameters related to glycometabolism were elevated. This includes random blood sugar (RBS), which showed a significant correlation with body mass index (BMI) in different demographics: adult women (P = 0.001), girls (P = 0.05), and boys (P = 0.006). However, there was no significant correlation found in the adult male group (P value = 0.7), as illustrated below.

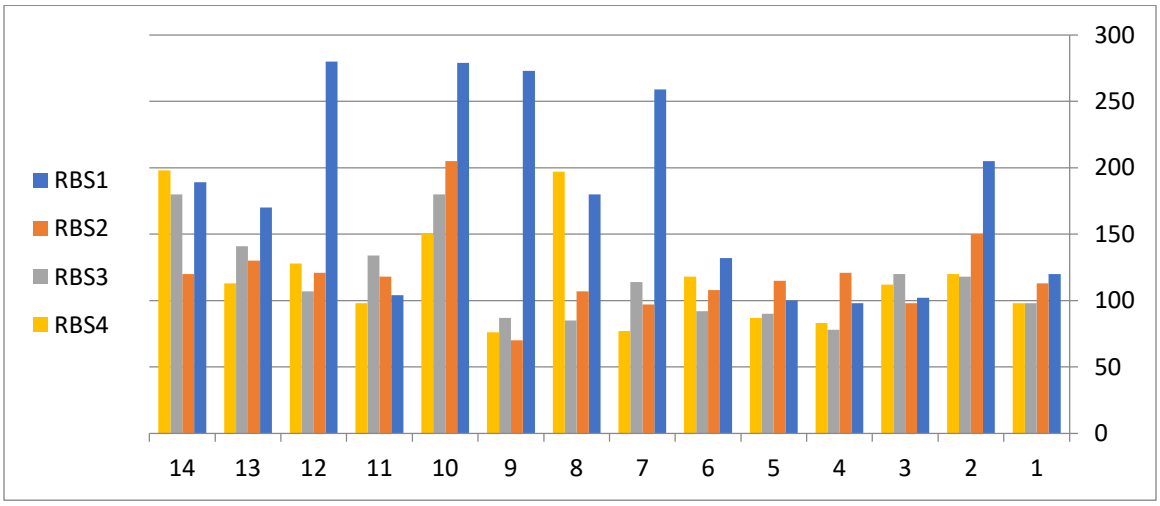


Figure (3-5) RBS among 4 months for women.

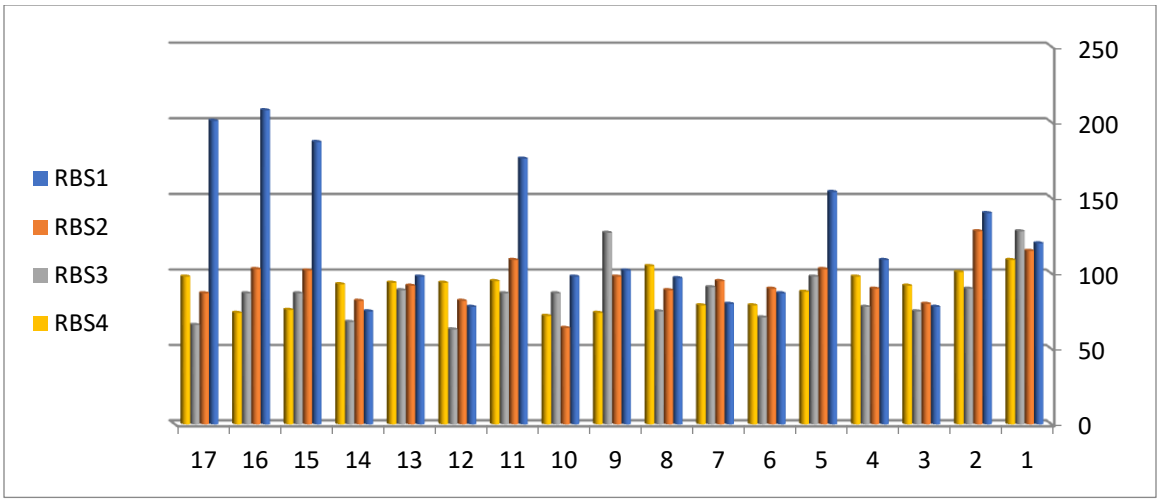


Figure (3-6) RBS among 4 months for Girls.

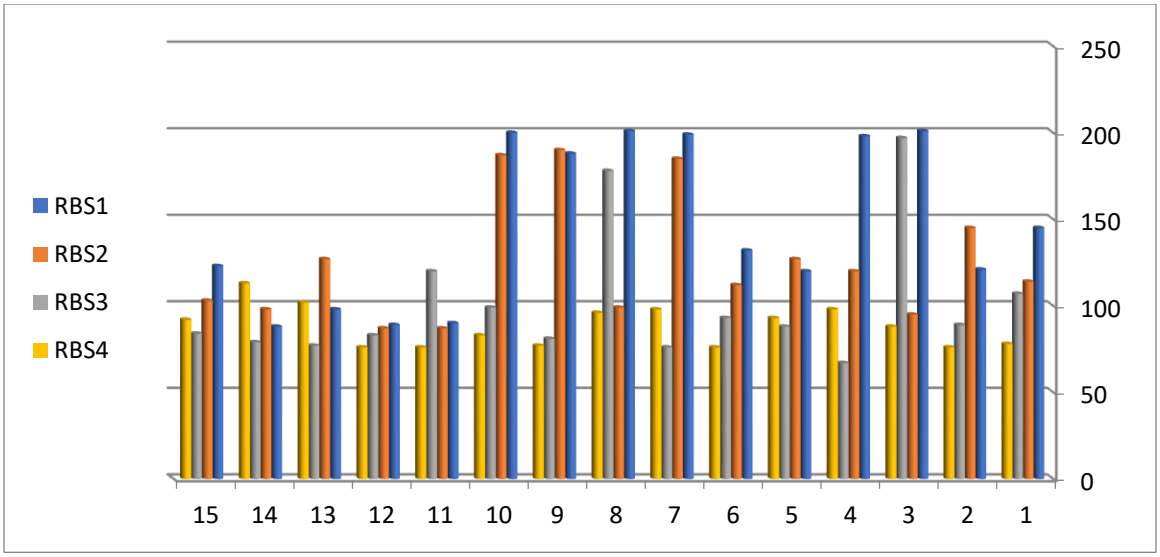


Figure (3-7) RBS among 4 months for boys.

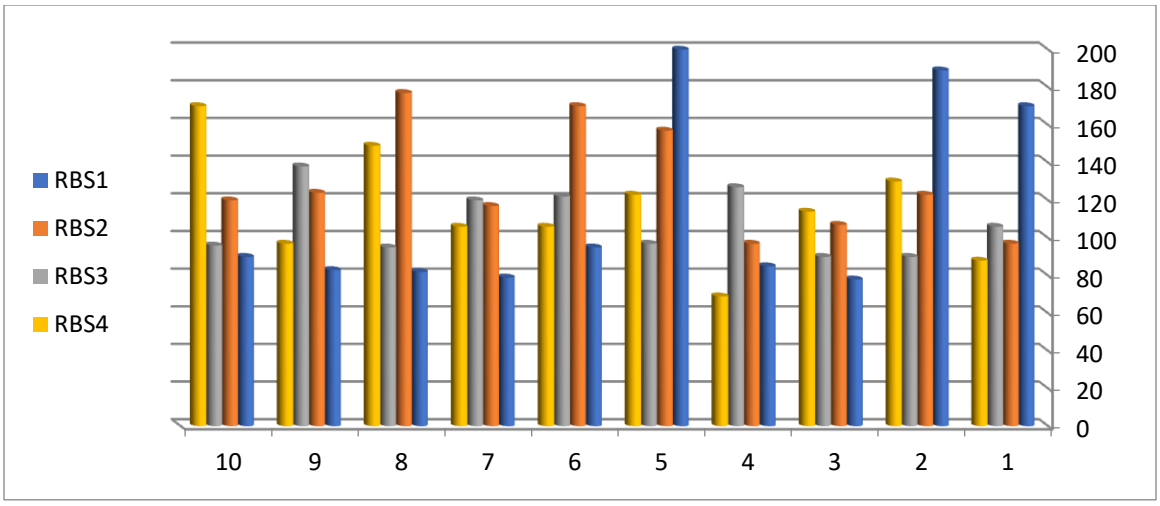


Figure (3-8) RBS among 4 months for men.

In this study of obesity and diabetes among individuals with a BMI greater than 25 kg/m², insulin levels play a notable role, particularly in teenagers. Excitingly, it found significant results: for girls, the p-value is 0.05, and for boys, it's 0.006.

However, in adults, the differences seem less pronounced, with women at a p-value of 1.1 and men at 0.7. It's fascinating to see how age interacts with these metabolic factors.

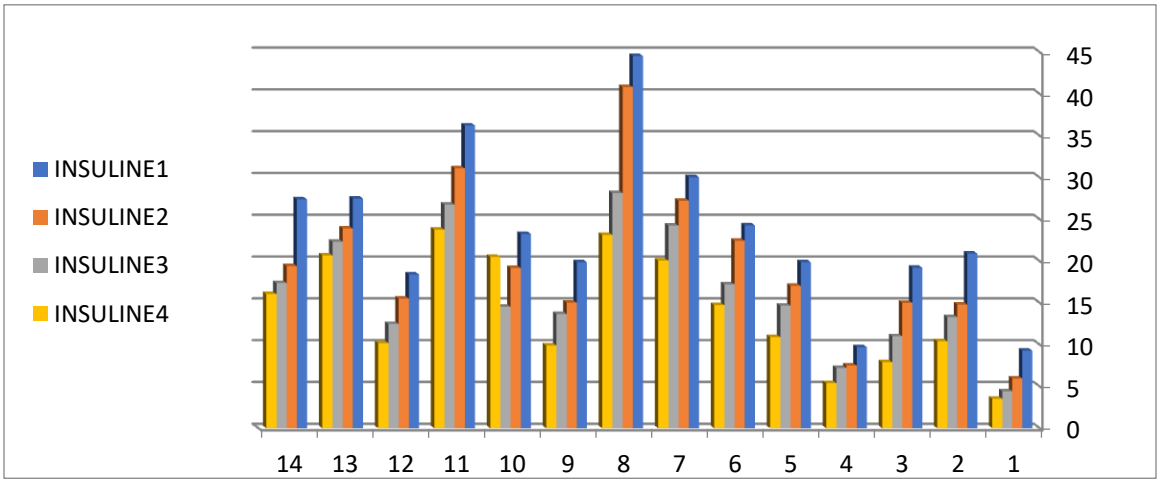


Figure (3-9) Insulin among 4 months for women.

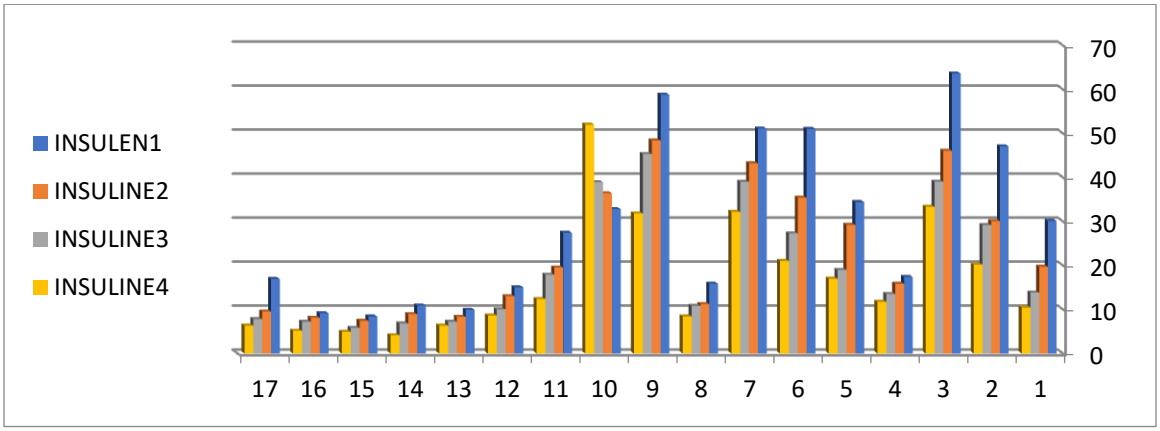


Figure (3-10) Insulin among 4 months for Girls.

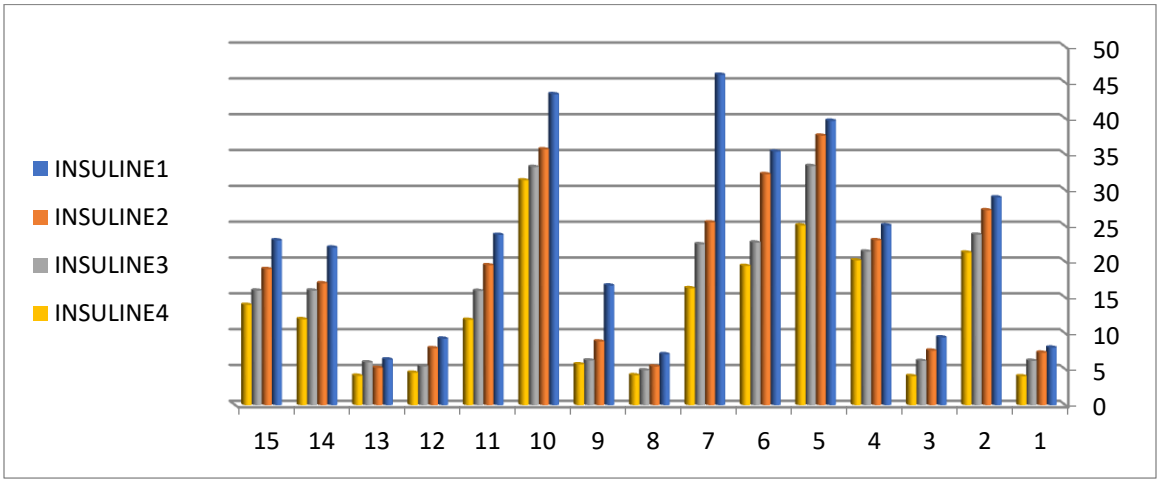


Figure (3-11) Insulin among 4 months for boys.

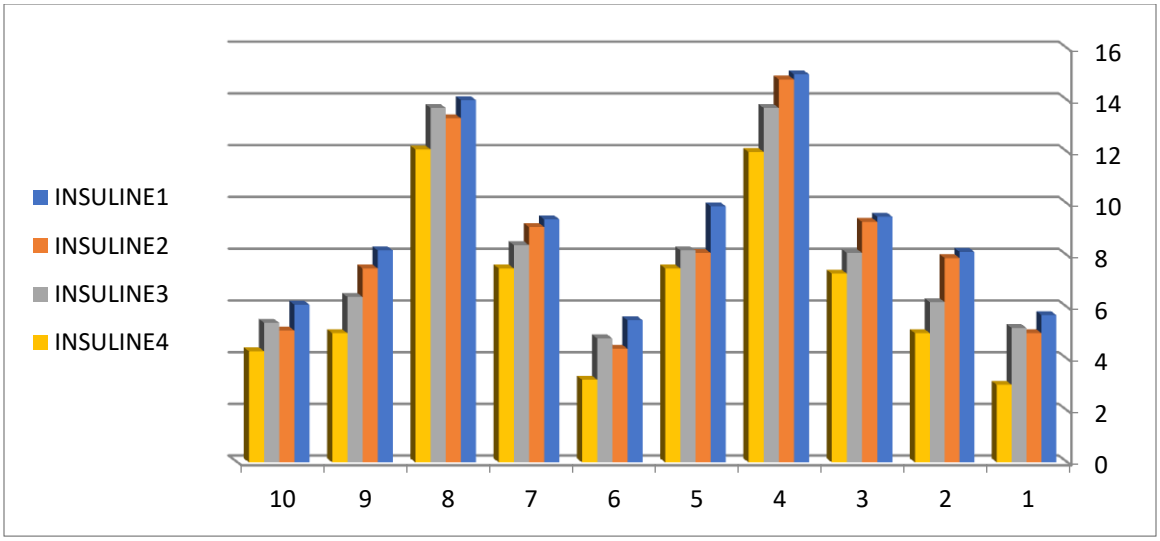


Figure (3-12) Insulin among 4 months for men.

Studies show a strong link between diabetes and BMI. Since insulin resistance often correlates with obesity, it's clear that maintaining a healthy weight can play a vital role in managing diabetes and related concerns. (79)(80)

Diabetes is a multifaceted condition that involves consistently high blood sugar levels due to either inadequate insulin production or its ineffective use. Understanding, it helps to manage it better and improve overall health. The recent increase in type 2 mellitus cases is largely due to rising obesity rates, an important risk factor it can change. By addressing this issue, it can have a wonderful opportunity to improve health outcomes and combat the progression of the disease together. (78)

3.4. The impact of dietary changes on reducing cardiovascular disease

Long-term consumption of excess calories leads to significant fat accumulation that surpasses the storage capacity of adipose tissue. This results in elevated levels of free fatty acids in the bloodstream and abnormal fatty acid storage in vital organs such as the liver, pancreas, and skeletal muscle, which are

essential for overall metabolic control. The presence of these free fatty acids causes lipotoxicity, leading to oxidative stress, inflammation, and metabolic dysregulation in these critical organs.

Obesity can lead to pro-inflammatory states and abnormal immune responses, which contribute to conditions such as insulin resistance, hypertension, atherosclerosis, kidney disease, and other chronic health issues. Additionally, the presence of excess adipose tissue around the heart, commonly found in individuals who are overweight or obese, further increases the risk of cardiovascular disease (CVD).(39)(81)

The biostatistical analysis of the lipid profile (triglycerides (TG), total cholesterol (TC), HDL, and LDL) in obese participants shows a strong correlation with BMI.

In women, the lipid profile parameters exhibited direct correlations with triglycerides (p-value = 0.009), total cholesterol (p-value = 0.0001), and LDL cholesterol (p-value = 0.0005), while HDL-C showed no significant correlation.

In men, the data analysis revealed direct correlations with triglycerides (p-value = 0.02), total cholesterol (p-value = 0.002), and HDL-C (p-value = 0.003), but no significant correlation with LDL-C (p-value = 0.5).

In girls, there are significant correlations with triglycerides (p-value = 0.0004) and total cholesterol (p-value = 0.02); however, there are no significant correlations with HDL-C (p-value = 0.5) or LDL-C (p-value = 0.6).

In boys, there are significant correlations with all parameters: triglycerides (p-value = 0.0001), total cholesterol (p-value = 0.001), HDL-C (p-value = 0.0005), and LDL-C (p-value = 0.001).

After following a specific dietary regimen that restricted calorie intake, there was a noticeable impact on TG, TC, and LDL levels, alongside improvements in HDL results, as shown below.

A healthy diet supports good health. Beyond its essential role in providing nutrients, food can also serve as a powerful tool in preventing and treating diseases, including obesity(82).

a) TG in women

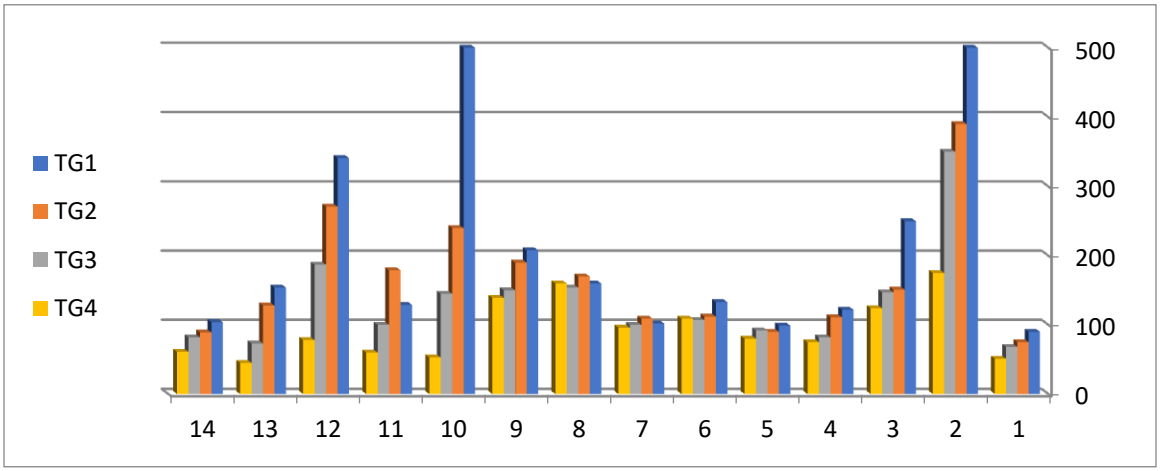


Figure (3-13)(a) the improvement in TG level after following dietary system in 4 months for women.

b) TG in girls

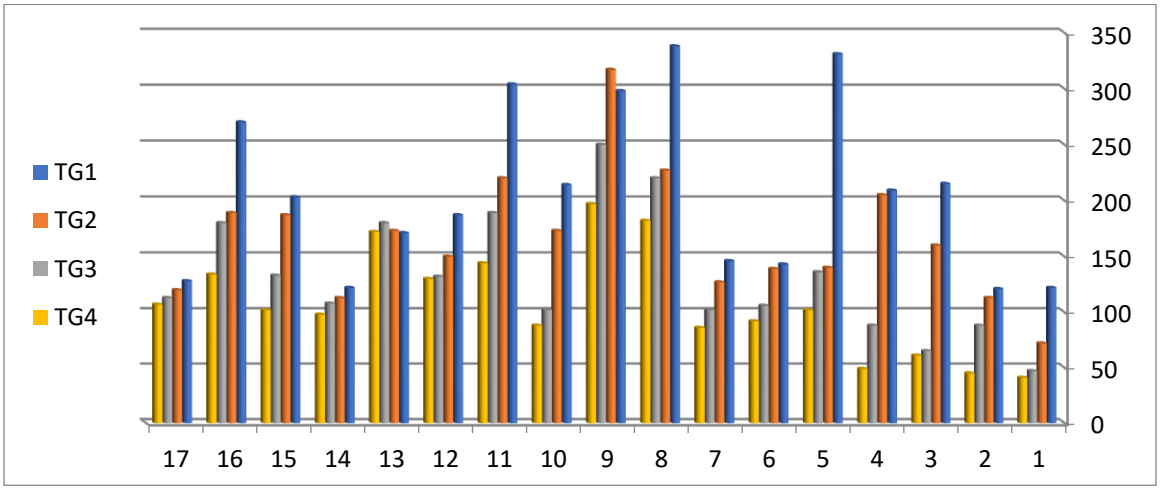


Figure (3-13)(B) the improvement in TG level after following dietary system in 4 months for girls.

c) TG in boys

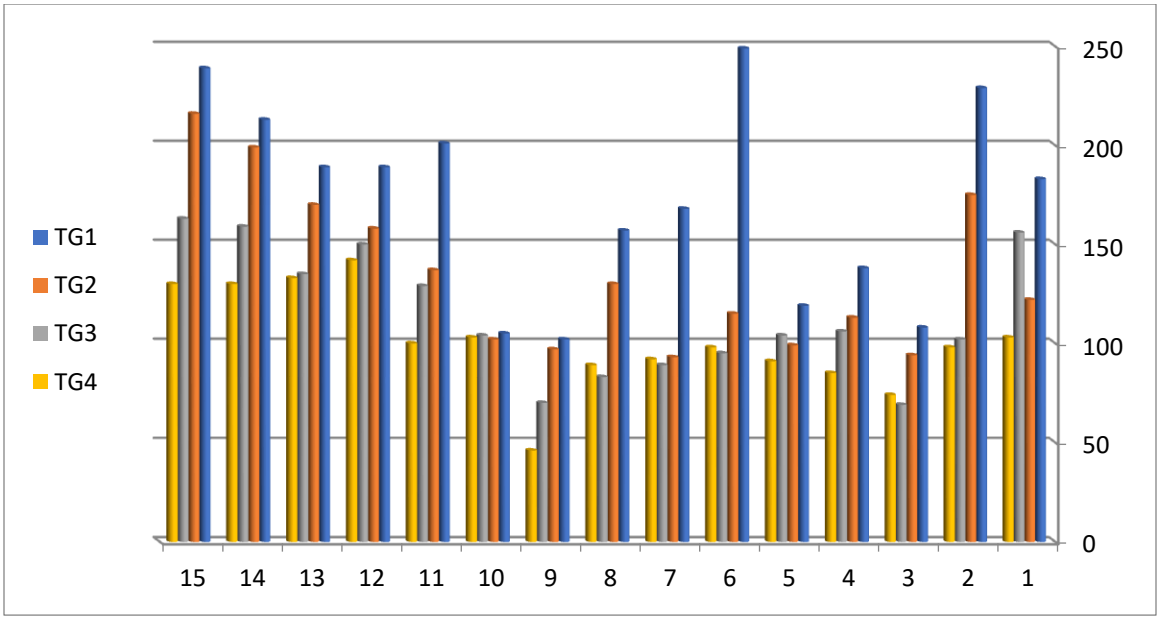


Figure (3-13)(c) the improvement in TG level after following dietary system in 4 months for boys.

d) TG in men

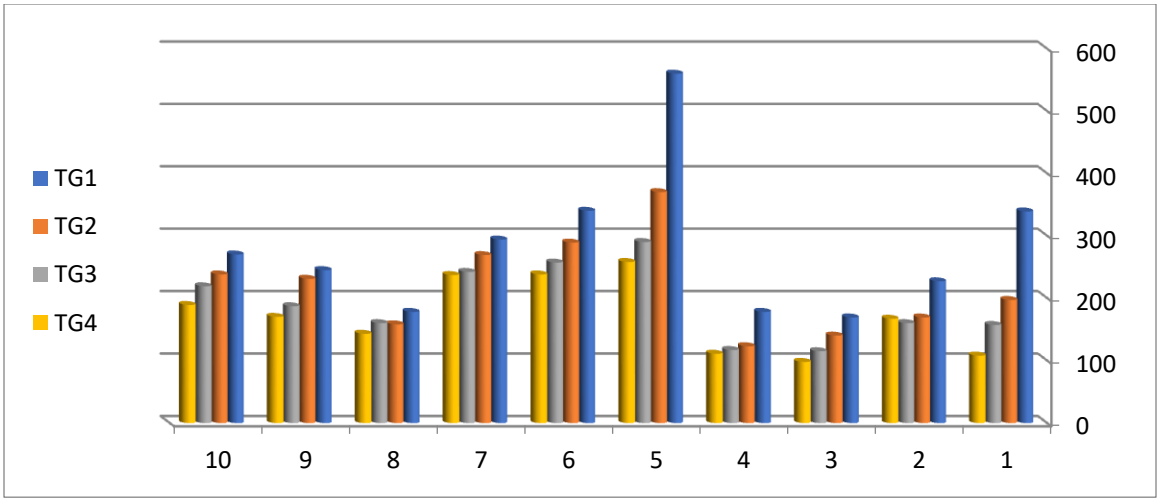


Figure (3-13)(d) the improvement in TG level after following dietary system in 4 months for men.

a) TC in women

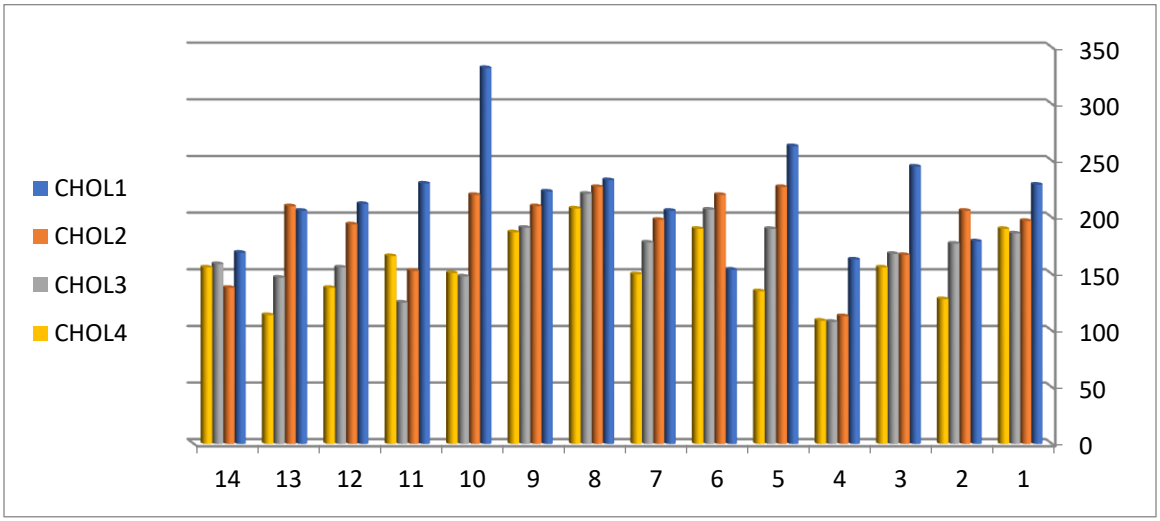


Figure (3-14)(a) the improvement in TC level after in 4 months following dietary system for women.

b) TC in girls

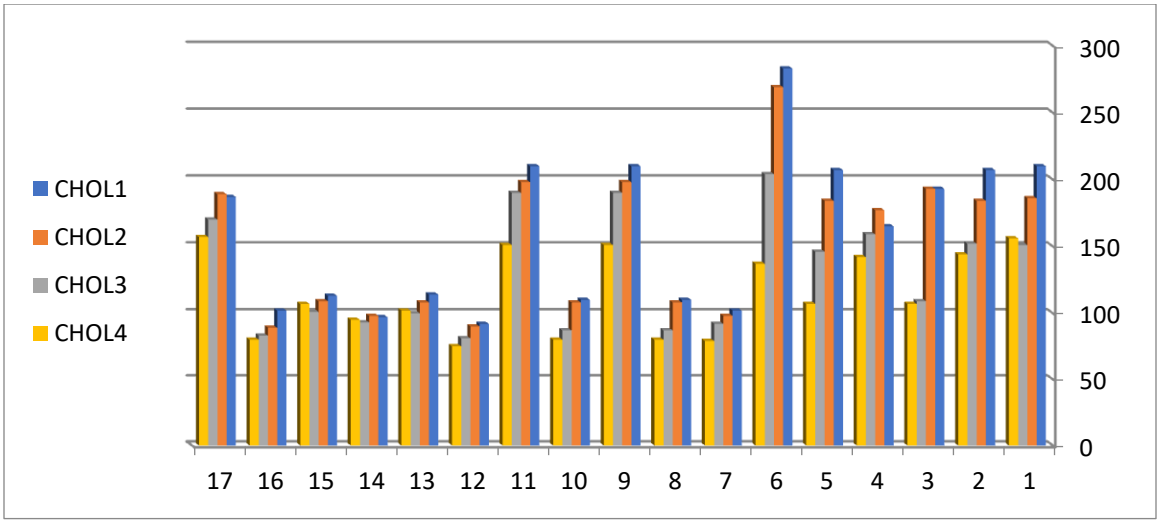


Figure (3-14)(b) the improvement in TC level after in 4 months following dietary system for girls.

c) TC in boys

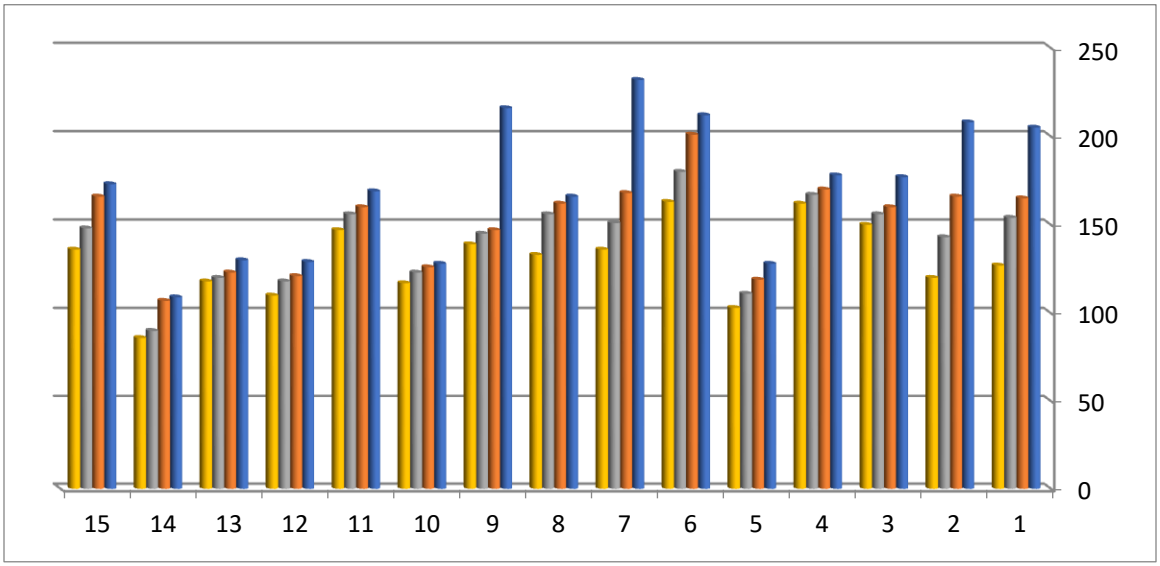


Figure (3-14)(c) the improvement in TC level after in 4 months following dietary system for boys.

d) TC in man

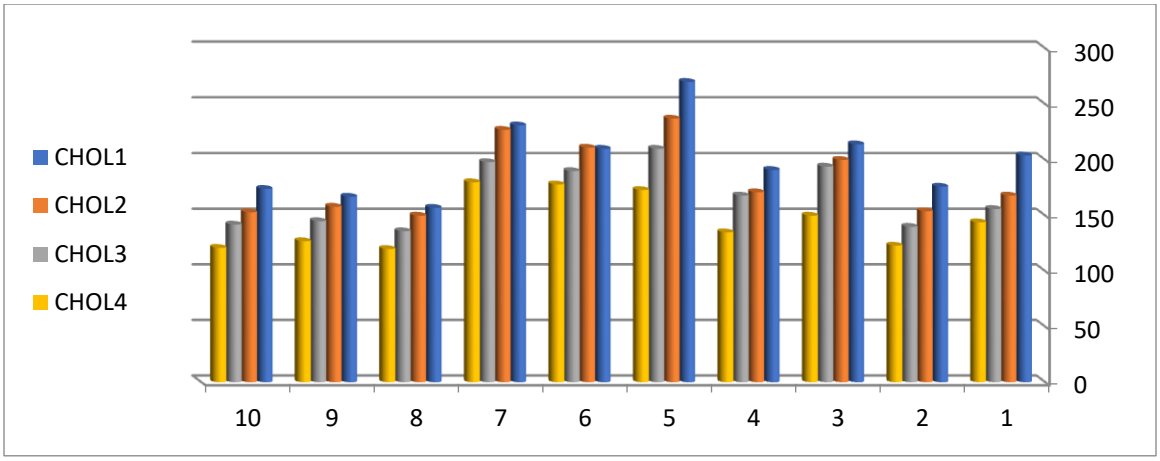


Figure (3-14)(d) the improvement in TC level after in 4 months following dietary system for men.

a) HDL-C in women

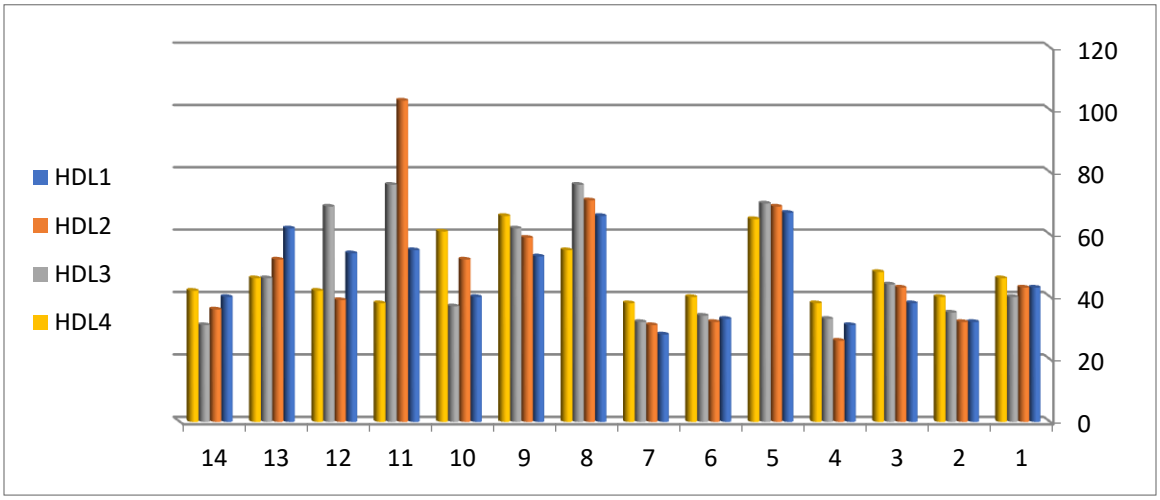


Figure (3-15)(a) the improvement in HDL-C level after in 4 months following dietary system in women.

b) HDL in girls

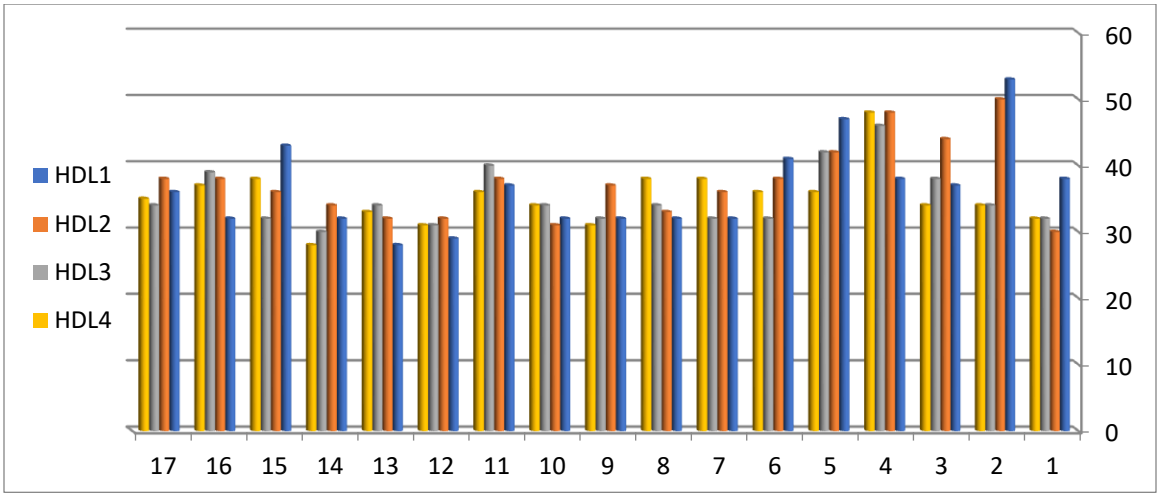


Figure (3-15)(b) the improvement in HDL-C level after in 4 months following dietary system in girls.

C. HDL-C in boys

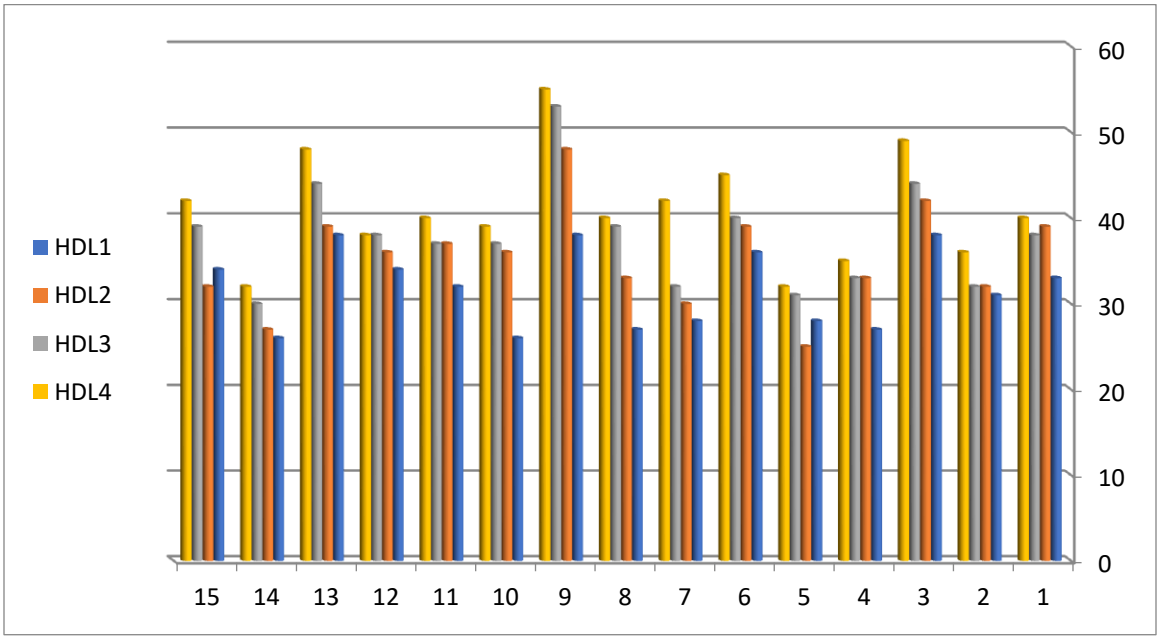


Figure (3-15)(c) the improvement in HDL-C level after in 4 months following dietary system in boys.

D. HDL in men

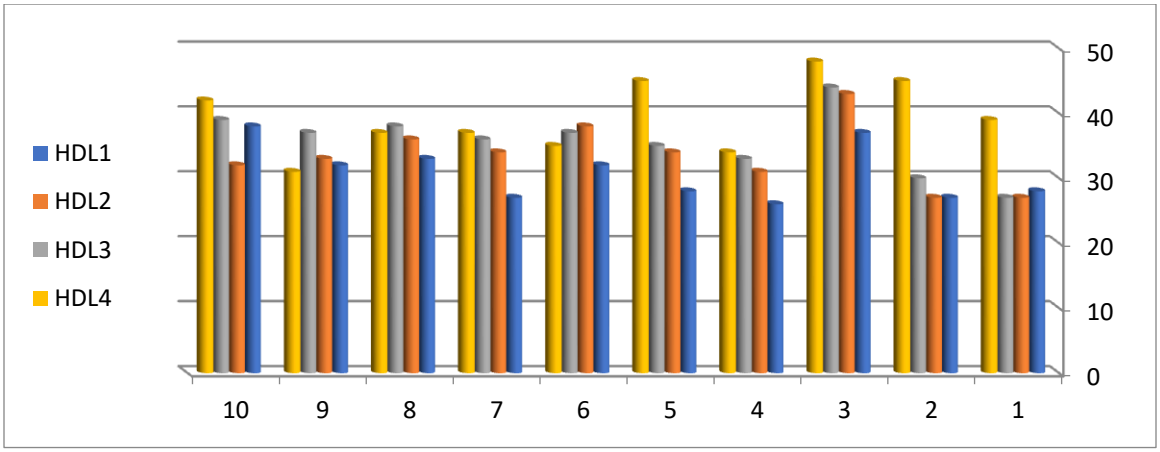


Figure (3-15)(d) the improvement in HDL-C level after in 4 months following dietary system for men.

a) LDL-C in women

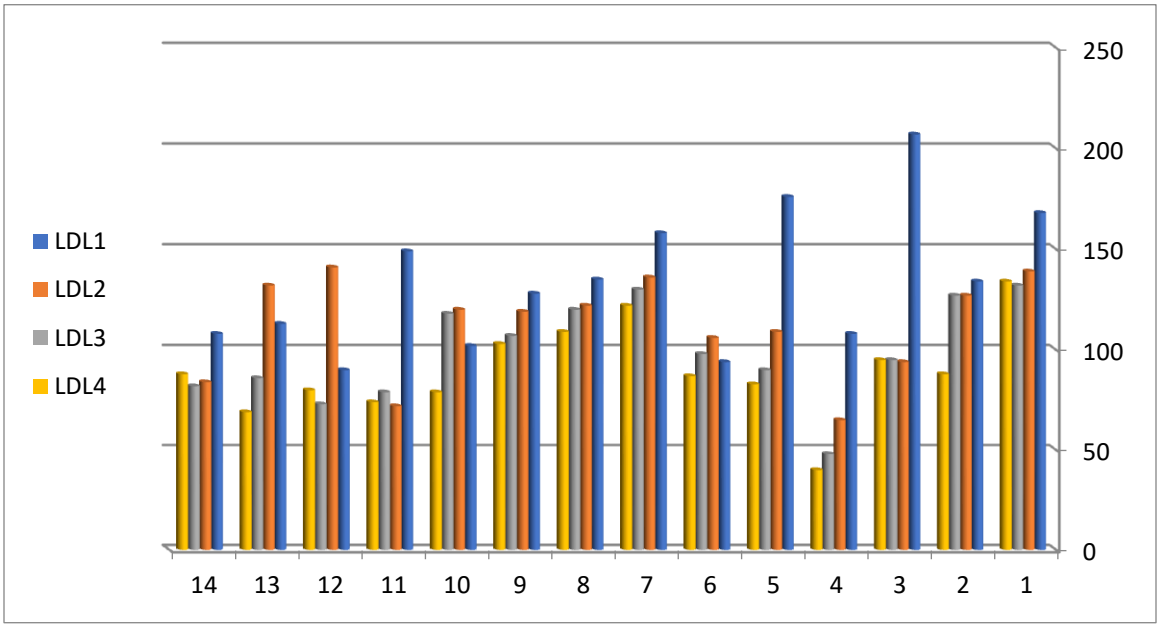


Figure (3-16)(a) the improvement in LDL-C level after in 4 months following dietary system in women.

b) LDL-C in girls

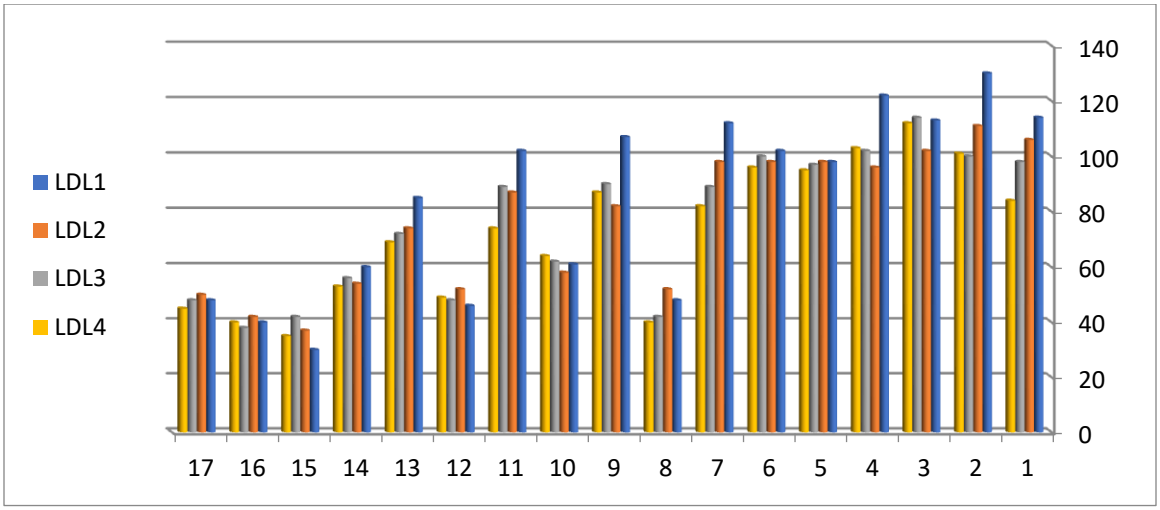


Figure (3-16)(b) the improvement in LDL-C level after in 4 months following dietary system in girls.

c) LDL-C in boys

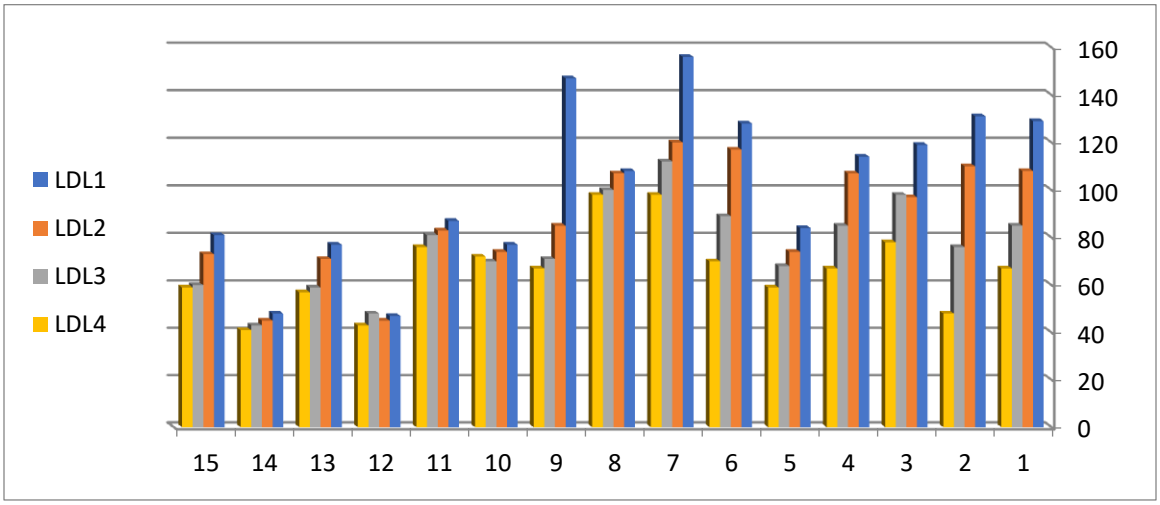


Figure (3-16)(c) the improvement in LDL-C level after in 4 months following dietary system in boys.

d) LDL-C in men

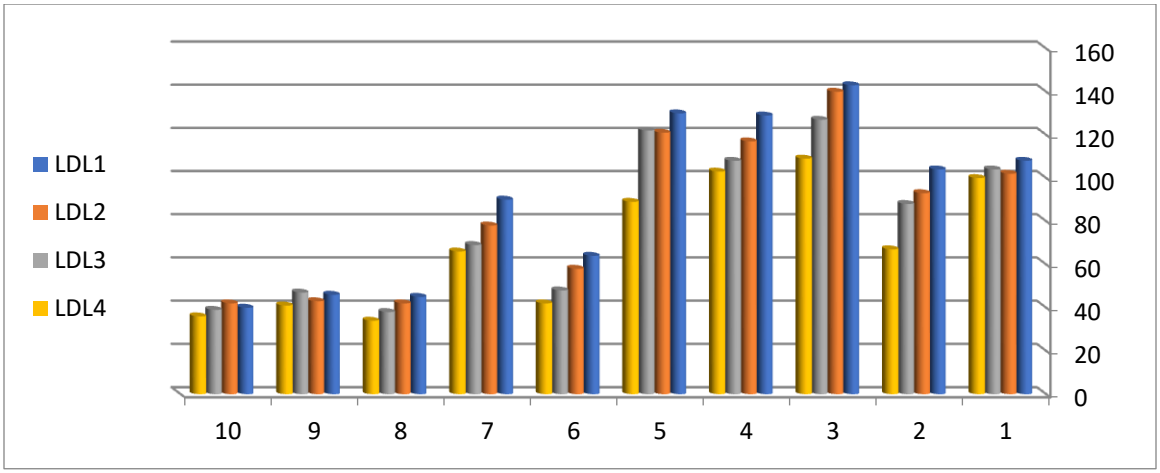


Figure (3-16)(d) the improvement in LDL-C level after in 4 months following dietary system in men.

Table (3.3) the difference variable of biomarker before and after the diet system for women.

Biomarker	Obesity	Mean	Std.deviation	P. value
BMI	Before diet	36.1571429	5.19374095	0.001
	After diet	30.6214286	4.69060904	
RBS	Before diet	177.928571	71.4277088	0.001
	After diet	103.714286	20.5105173	
TG	Before diet	206.571429	142.043176	0.008
	After diet	93.2857143	42.1105296	
TC	Before diet	217.428571	46.0997485	0.001
	After diet	155.571429	29.9453715	

HDL	Before diet	45.8571429	13.4442275	0.6
	After diet	47.5	10.1356188	
B.UREA	Before diet	24.4214286	6.00809436	0.7
	After diet	23.9571429	5.02083571	
S.CREATININE	Before diet	0.70571429	0.19310078	0.6
	After diet	0.681428571	0.141522335	
ALT	Before diet	23.85714286	15.89318743	0.1
	After diet	20.42857143	12.0875924	
AST	Before diet	26.42857143	8.234208997	0.001
	After diet	19.21428571	7.777468161	
ALP	Before diet	116.7857143	26.64922736	0.001
	After diet	83.14285714	15.64440004	
LIPASE	Before diet	31.9285714	10.41052933	0.7
	After diet	31	8.584422385	
LDH	Before diet	27.07142857	11.15943399	0.4
	After diet	25.64285714	8.57193222	
GH	Before diet	1.672428571	1.888627125	0.001
	After diet	6.27	4.764100997	

INSULIN	Before diet	23.59785714	9.400452672	0.001
	After diet	14.08	6.687306	
VD3	Before diet	13.18142857	5.215158169	0.001
	After diet	33.22142857	19.0813404	

Table (3.4) the difference variable of biomarker before and after the diet system for GIRLS.

Biomarker	Obesity	Mean	Std.deviation	P. value
BMI	Before diet	33.37058824	4.040848411	0.001
	After diet	28.15294118	3.362498633	
RBS	Before diet	122.8235294	45.78104861	0.01
	After diet	89.47058824	11.66789209	
TG	Before diet	207.1764706	75.79927052	0.001
	After diet	107.6470588	47.04245579	
TC	Before diet	159.5294118	57.84258557	0.001
	After diet	114.7058824	31.04586266	
HDL	Before diet	36.41176471	6.614934084	0.5
	After diet	35.23529412	4.323329327	

LDL	Before diet	83.41176471	33.0209381	0.002
	After diet	72.29411765	25.10917339	
B.UREA	Before diet	27.28235294	8.048154703	0.1
	After diet	24.62941176	6.203604265	
S.CREATININE	Before diet	0.842352941	0.167911041	0.008
	After diet	0.705882353	0.164128716	
ALT	Before diet	31.29411765	9.745285436	0.001
	After diet	21.64705882	8.521598856	
AST	Before diet	28.82352941	9.850350845	0.001
	After diet	21.05882353	7.445389414	
ALP	Before diet	127.7058824	44.64409914	0.01
	After diet	108.7647059	46.52221165	
LIPASE	Before diet	25.94117647	10.4011453	0.001
	After diet	38.35294118	13.55978049	
LDH	Before diet	73.11764706	28.70078909	0.1
	After diet	65.64705882	22.19499149	
GH	Before diet	0.846470588	0.578863231	0.001
	After diet	1.519176471	0.994486251	

INSULIN	Before diet	29.39647059	18.69495552	0.001
	After diet	16.80117647	13.4291897	
VD3	Before diet	17.89529412	11.44592893	0.001
	After diet	29.78470588	7.689457164	

Table (3.5) the difference variable of biomarker before and after the diet system for BOYS.

Biomarker	Obesity	Mean	Std.deviation	P. value
BMI	Before diet	29.96666667	2.112096679	0.001
	After diet	27.12	1.852103052	
RBS	Before diet	146.2	46.53907728	0.001
	After diet	88.13333333	11.79507565	
TG	Before diet	172.6	49.69449524	0.001
	After diet	100.9333333	24.99847614	
TC	Before diet	170.6666667	38.78082491	0.001
	After diet	129.8	21.46492155	
HDL	Before diet	31.73333333	4.527166674	0.001
	After diet	40.86666667	6.356848575	

LDL	Before diet	102.2	33.63289206	0.001
	After diet	66.66666667	16.89744979	
B.UREA	Before diet	25.59333333	4.492130685	0.001
	After diet	30.52666667	4.0622771	
S.CREATININE	Before diet	0.705333333	0.188750276	0.006
	After diet	0.845333333	0.14126301	
ALT	Before diet	24.06666667	7.205817755	0.001
	After diet	17.06666667	6.496885701	
AST	Before diet	24.73333333	5.675343497	0.001
	After diet	17.66666667	4.099941928	
ALP	Before diet	283.2	84.27268664	0.001
	After diet	240.4	70.29712451	
LIPASE	Before diet	23.8	11.10469656	0.001
	After diet	37.4	12.3218737	
LDH	Before diet	59	9.848857802	0.006
	After diet	112.2666667	65.07958498	
GH	Before diet	0.677142857	1.320512596	0.02
	After diet	2.275571429	3.340323193	

INSULIN	Before diet	22.96	13.6025171	0.001
	After diet	13.21333333	8.872617319	
VD3	Before diet	9.018666667	3.759547592	0.001
	After diet	24.678	9.599730056	

Table (3.6) the difference variable of biomarker before and after the diet system for MEN.

Biomarker	Obesity	Mean	Std.deviation	P. value
BMI	Before diet	32.41	5.156753932	0.001
	After diet	28.24	4.976879879	
RBS	Before diet	115.1	49.92093749	0.5
	After diet	104.4	21.52104913	
TG	Before diet	280	116.8760027	0.003
	After diet	171.9	58.1462715	
TC	Before diet	199.4	34.05942519	0.001
	After diet	145.1	24.11292876	
HDL	Before diet	30.8	4.289522118	0.001
	After diet	39.3	5.518655231	

LDL	Before diet	89.9	38.85428619	0.001
	After diet	68.7	29.73232435	
B.UREA	Before diet	32.78	6.690590407	0.02
	After diet	28.28	5.304044578	
S.CREATININE	Before diet	0.912	0.220645518	0.2
	After diet	0.818	0.1386282	
ALT	Before diet	32.8	7.554248253	0.001
	After diet	25.9	6.773313648	
AST	Before diet	34.18181818	9.907756374	0.001
	After diet	24.63636364	6.407382106	
ALP	Before diet	88.5	25.859664	0.001
	After diet	70.4	22.35670419	
LIPASE	Before diet	59	8.485281374	0.005
	After diet	73.3	16.87898101	
LDH	Before diet	73.06	24.99494171	0.001
	After diet	83.5	26.16719745	
GH	Before diet	0.997	0.74858459	0.01
	After diet	1.285	0.851681344	

INSULIN	Before diet	9.144	3.247943623	0.001
	After diet	6.69	3.272257121	
VD3	Before diet	16.23	9.360205363	0.01
	After diet	29.58	11.737291	

3.5 Correlation of several biochemical markers over four months in obese individuals undergoing dietary intervention.

The data illustrates the correlation relationships between various biochemical markers observed over four months in obese individuals undergoing dietary intervention. By interpreting these results, we gain insights into the metabolic adaptations that take place during weight loss. Correlation coefficients (r) indicate the strength and direction of these associations, with values closer to +1 or -1 signifying stronger relationships. In general, values between -0.3 and +0.3 are considered weak and may not hold clinical significance.

3.5.1 Correlation between lipase and lipid profile

Delving into the relationship between lipase levels and lipid profiles can reveal valuable insights into how each biomarker reflects the activity of the lipase.

The biostatistical analysis seems to be conducted thoroughly.

a) for women:

In Month 1, lipase exhibited the strongest correlation with triglycerides (TG), with a correlation coefficient of r = 0.406, indicating a weak positive relationship, as

shown in Table 3-3(a). Other correlations appeared negligible. Elevated levels of TG and lipase may occur together in cases of pancreatic inflammation or metabolic dysregulation, such as hyperlipidemia.(83).

In Month 2, we observed a striking positive correlation with triglycerides (TG), underscoring a significant link to pancreatitis due to hypertriglyceridemia. This finding suggests a critical relationship that warrants further investigation.(84).

Month 3: Lipase demonstrated a remarkable correlation with LDL ($r = 0.79$), highlighting the important connections in the lipid-pancreatic axis, which may be shaped by diet or metabolic conditions. (85)

Month 4: it was observed that there was a noticeable decline in all correlations, suggesting that their clinical significance has diminished to minimal or even nonexistent levels.

b) for girls:

Over a period of four consecutive months, lipase consistently showed weak negative correlations with the lipid profile markers (triglycerides, cholesterol, HDL, and LDL) as illustrated in Table 3-3(b). However, none of these correlations were statistically significant, with r values ranging between -0.48 and 0.05. This suggests that there is no meaningful linear relationship between serum lipase activity and lipid levels in this female cohort.

Lipase, an enzyme primarily involved in fat digestion, is not typically used as a marker for interpreting lipid profiles. Previous studies have demonstrated that serum lipase is not a strong predictor of dyslipidemia in non-pancreatic disorders, further supporting these findings. (127)

c) for boys

Over the course of these four months, it can be observed some intriguing trends regarding lipase and its relationship with triglycerides (TG), cholesterol (CHOL), high-density lipoprotein (HDL), and low-density lipoprotein (LDL), as detailed in Table 3-3(c). The most noteworthy correlation was with LDL during the first month ($r = -0.41$), hinting at a mild inverse relationship. This suggests that lipase plays a role in fat metabolism, but the overall weak correlations indicate that its impact on circulating lipid levels isn't consistent over time. It's a fascinating area for further exploration, especially regarding how diet-induced weight loss might influence these dynamics.

d)For men:

Over the course of four months, the correlation between lipase levels and lipid parameters remained weak and statistically insignificant, as shown in Table 3-3 (d). Lipase, an enzyme involved in lipid metabolism, demonstrated minimal associations with triglycerides (TG), cholesterol (CHOL), high-density lipoprotein (HDL), and low-density lipoprotein (LDL). This suggests that variations in lipase levels did not have a significant impact on or reflect changes in lipid components. The lack of a clear relationship may be due to unaccounted dietary or hormonal factors that were not controlled for in the analysis

Table(3.7) (a) Exploring the relationship between lipase levels and various lipids: triglycerides (TG), cholesterol (CHOL), HDL, and LDL, in women.

Month 1					
	LIPASE1	TG1	CHOL1	HDL1	LDL1
LIPASE1	1	0.406	-0.055	0.148	0.096

Month 2					
	LIPASE2	**TG2**	**CHOL2**	**HDL2**	**LDL2**
LIPASE2	1	0.65	-0.31	-0.09	-0.18
Month 3					
	LIPASE3	**TG3**	**CHOL3**	**HDL3**	**LDL3**
LIPASE3	1	0.19	0.50	0.02	0.79
Month 4					
	LIPASE4	**TG4**	**CHOL4**	**HDL4**	**LDL4**
LIPASE4	1	0.13	0.28	-0.30	-0.005

Table(3.7)(b) Exploring the relationship between lipase levels and various lipid types: triglycerides (TG), cholesterol (CHOL), HDL, and LDL, in girls.

Month 1					
	LIPASE1	**TG1**	**CHOL1**	**HDL1**	**LDL1**
LIPASE1	1	-0.23	-0.45	-0.36	-0.35
Month 2					
	LIPASE2	**TG2**	**CHOL2**	**HDL2**	**LDL2**
LIPASE2	1	-0.31	-0.23	-0.14	-0.32
Month 3					
	LIPASE3	**TG3**	**CHOL3**	**HDL3**	**LDL3**

LIPASE3	1	0.05	-0.19	-0.21	-0.48
Month 4					
	LIPASE4	TG4	CHOL4	HDL4	LDL4
LIPASE4	1	0.03	0.02	-0.08	-0.44

Table(3.7) (c) Exploring the relationship between lipase levels and various lipid types: triglycerides (TG), cholesterol (CHOL), HDL, and LDL, in boys

Month 1					
	LIPASE1	TG1	CHOL1	HDL1	LDL1
LIPASE1	1	0.24	-0.40	0.18	-0.41
Month 2					
	LIPASE2	TG2	CHOL2	HDL2	LDL2
LIPASE2	1	0.44	-0.16	-0.21	-0.28
Month 3					
	LIPASE3	TG3	CHOL3	HDL3	LDL3
LIPASE3	1	0.28	-0.24	-0.20	-0.36
Month 4					
	LIPASE4	TG4	CHOL4	HDL4	LDL4
LIPASE4	1	0.40	-0.10	-020	-0.36

Table(3.7) (d) Exploring the relationship between lipase levels and various lipid types: triglycerides (TG), cholesterol (CHOL), HDL, and LDL, in men.

Month 1					
	LIPASE1	TG1	CHOL1	HDL1	LDL1
LIPASE1	1	-0.37	-0.09	-0.15	-0.04
Month 2					
	LIPASE2	TG2	CHOL2	HDL2	LDL2
LIPASE2	1	-0.27	-0.08	0.10	-0.19
Month 3					
	LIPASE3	TG3	CHOL3	HDL3	LDL3
LIPASE3	1	-0.04	0.04	0.30	-0.25
Month 4					
	LIPASE4	TG4	CHOL4	HDL4	LDL4
LIPASE4	1	-0.01	-0.07	-0.25	-0.17

3.5.2. Correlations LDH with CHOL, Vitamin D3 (VD3), and TG

Exploring the relationships between LDH and key biomarkers, such as CHOL, Vitamin D3, and TG, offers exciting insights into their interconnections and potential health implications.

a) for women:

Month 1-2: Lactate dehydrogenase (LDH) showed a moderate correlation with cholesterol levels (r = 0.54 and 0.46), suggesting potential tissue damage related to lipid metabolism.

Month 3: There was a negative correlation with vitamin D (VD3) (r = -0.53), which aligns with existing literature indicating that low vitamin D levels may lead to increased oxidative stress and higher LDH activity(86).

Month 4: Weak associations were observed across all variables, indicating minimal diagnostic utility, as shown in Tables 3-4.(a).

b) for girls:

Over the course of all months, lactate dehydrogenase (LDH) demonstrated weak and statistically non-significant correlations with cholesterol (CHOL), vitamin D (VD3), and triglycerides (TG), with correlation coefficients ranging from -0.33 to 0.22, as presented in Table 3-4(b). This suggests that there is no meaningful relationship between LDH activity and these biochemical markers.

LDH is an intracellular enzyme that reflects tissue damage rather than metabolic balance. In a cohort study, the levels of LDH did not show significant correlations with vitamin D or lipid levels, except in cases of tissue necrosis or cancer(87).

c) for boys:

In the early months, LDH displayed a moderate negative correlation with cholesterol (r = -0.52) and a positive correlation with vitamin D3 (r = 0.54), as shown in Table 3-4(c). These results point to an interesting relationship between LDH activity and metabolic stress or inflammation, commonly seen with

dyslipidemia and vitamin D deficiency in those who are obese. Excitingly, recent studies suggest that elevated LDH levels may signal subclinical tissue damage occurring in oxidative environments, particularly in cases of vitamin D deficiency. (88)

d)for men:

LDH (lactate dehydrogenase) presented weak and non-significant correlations with cholesterol, VD3, and TG throughout the study period. Although LDH is known to rise in metabolic stress or tissue injury, the absence of a meaningful correlation here may suggest that LDH variations were not primarily driven by lipid alterations or vitamin D3 levels in this cohort.

Table (3-8) (a) Correlation LDH with CHOL, VD_3, and TG in women

Month 1				
	LDH1	CHOL1	$VD_3$1	TG1
LDH1	1	0.54	0.08	0.06
Month 2				
	LDH2	CHOL2	$VD_3$2	TG2
LDH2	1	0.46	0.01	-0.15
Month 3				
	LDH3	CHOL3	$VD_3$3	TG3
LDH3	1	0.23	-0.53	0.07
Month 4				
	LDH4	CHOL4	$VD_3$4	TG4

LDH4	1	0.32	0.35	-0.06

Table (3-8) (b) Correlation LDH with CHOL, VD_3, TG in girls.

Month 1				
	LDH1	**CHOL1**	**$VD_3$1**	**TG1**
LDH1	1	-0.09	0.11	0.22
Month 2				
	LDH2	**CHOL2**	**$VD_3$2**	**TG2**
LDH2	1	-0.21	-0.10	0.11
Month 3				
	LDH3	**CHOL3**	**$VD_3$3**	**TG3**
LDH3	1	0.09	-0.33	0.13
Month 4				
	LDH4	**CHOL4**	**$VD_3$4**	**TG4**
LDH4	1	0.06	0.02	0.09

Table (3-8) (c) Correlation LDH with CHOL, $VD_{3,}$ TG in boys.

Month 1				
	LDH1	CHOL1	$VD_3$1	TG1
LDH1	1	-0.43	0.54	-0.04
Month 2				
	LDH2	CHOL2	$VD_3$2	TG2
LDH2	1	-0.52	0.21	-0.02
Month 3				
	LDH3	CHOL3	$VD_3$3	TG3
LDH3	1	-0.38	0.30	-0.03
Month 4				
	LDH4	CHOL4	$VD_3$4	TG4
LDH4	1	-0.34	-0.15	-0.01

Table (3-8) (d) Correlationwith LDH and CHOL, $VD_{3,}$ TG in men.

Month 1				
	LDH1	CHOL1	$VD_3$1	TG1
LDH1	1	-0.01	0.29	0.16
Month 2				

	LDH2	CHOL2	$VD_3$2	TG2
LDH2	1	-0.16	0.50	-0.06
Month 3				
	LDH3	CHOL3	$VD_3$3	TG3
LDH3	1	-0.25	0.49	-0.14
Month 4				
	LDH4	CHOL4	$VD_3$4	TG4
LDH4	1	-0.29	0.32	-0.10

3.5.3 Correlation Between TG and Liver Enzymes (ALT, AST, ALP)

Investigating the relationship between triglycerides (TG) and liver enzymes (ALT, AST, ALP) can provide valuable insights into how TG levels affect liver function.

a) for women:

Month 1: TG was moderately correlated with AST (r = 0.54) as shown in table(3-5)(a), suggesting hepatic stress, possibly due to non-alcoholic fatty liver disease (NAFLD)(89).

Months 2–4: Weak and insignificant correlations imply fluctuating liver enzyme levels unrelated to triglycerides.

b) for girls:

In months 1 through 4, TG showed variable correlations with ALT, AST, and ALP, with moderate positive correlations with ALT (e.g., r = 0.54 in month 4) and ALP

(e.g., r = 0.50 in month 1) as shown in table (3-5) (b), suggesting some potential linkage between triglyceride levels and liver function.

Hypertriglyceridemia is associated with liver enzyme elevation in non-alcoholic fatty liver disease (NAFLD). A study confirmed that ALT and TG are often positively correlated in hepatic steatosis(90).

c) for boys:

Correlations between TG and liver enzymes (ALT, AST, ALP) were weak and mostly non-significant, except for a slight negative association with ALP in month three (r = –0.45) as shown in table (3-5) (c). This may suggest that triglyceride reduction due to dietary intervention has limited short-term influence on liver enzyme levels, or that hepatic adaptations take longer to manifest in obese individuals.

d)for men:

A progressive increase in correlation was observed between TG and ALP over the four months, becoming moderately strong in months 3 and 4 (r=0.74 and r=0.82 respectively) as shown in table(3-5) (d), while correlations with ALT and AST remained weak. This suggests a potential association between triglyceride levels and cholestatic or hepatic enzyme responses. Elevated ALP in obesity has been linked to liver steatosis and biliary pressure(91).

Table (3-9) (a) Correlation between TG and ALT, AST, ALP for women.

Month 1				
	TG1	ALT1	AST1	ALP1
TG1	1	0.07	0.54	-0.02

Month 2				
	TG2	**ALT2**	**AST2**	**ALP2**
TG2	1	0.01	0.24	0.16
Month 3				
	TG3	**ALT3**	**AST3**	**ALP3**
TG3	1	0.10	-0.01	0.19
Month 4				
	TG4	**ALT4**	**AST4**	**ALP4**
TG4	1	0.26	-0.33	-0.11

Table (3-9) (b) Correlation between TG and ALT, AST, ALP for girls.

Month 1				
	TG1	**ALT1**	**AST1**	**ALP1**
TG1	1	0.03	0.002	0.50
Month 2				
	TG2	**ALT2**	**AST2**	**ALP2**
TG2	1	0.47	0.23	0.26
Month 3				
	TG3	**ALT3**	**AST3**	**ALP3**

TG3	1	0.48	0.02	0.38
Month 4				
	TG4	ALT4	AST4	ALP4
TG4	1	0.54	0.21	0.41

Table (3-9) (c) Correlation between TG and ALT, AST, ALP for boys.

Month 1				
	TG1	ALT1	AST1	ALP1
TG1	1	0.09	-0.01	0.09
Month 2				
	TG2	ALT2	AST2	ALP2
TG2	1	0.27	0.06	-0.08
Month 3				
	TG3	ALT3	AST3	ALP3
TG3	1	-0.02	-0.25	-0.45
Month 4				
	TG4	ALT4	AST4	ALP4
TG4	1	0.12	-0.12	-0.21

Table (3-9) (d) Correlation between TG and ALT, AST, ALP for men.

Month 1				
	TG1	ALT1	AST1	ALP1
TG1	1	0.02	-0.10	0.37
Month 2				
	TG2	ALT2	AST2	ALP2
TG2	1	0.002	0.02	0.63
Month 3				
	TG3	ALT3	AST3	ALP3
TG3	1	0.13	0.11	0.74
Month 4				
	TG4	ALT4	AST4	ALP4
TG4	1	0.24	0.11	0.82

3.5.4 Correlation CHOL with Liver Enzymes (ALT, AST, ALP)

Examining the relationship between cholesterol levels (CHOL) and liver enzymes (ALT, AST, ALP) provides valuable insights into how cholesterol levels impact liver function.

a) for women:

Month 1: A strong correlation with AST (r = 0.63) supports hepatic involvement in cholesterol metabolism.

Month 3: ALP correlation increased (r = 0.50), which could reflect biliary pathology or metabolic bone disease.

Month 4: Moderate ALT correlation (r = 0.51) supports the above observation.

b) for girls:

Total cholesterol (CHOL) had weak and insignificant correlations with ALT, AST, and ALP in all months (r range: -0.27 to 0.03), showing no consistent trend in table (3-6) (b).

In general populations, cholesterol levels do not directly reflect liver injury markers unless significant liver dysfunction is present(92).

c) for boys:

Cholesterol levels showed only weak correlations with ALT, AST, and ALP throughout all months as shown in table (3-6) (c). This lack of association is consistent with modern research showing that total cholesterol may not directly reflect hepatic enzyme fluctuations unless liver damage or metabolic syndrome is advanced.

d)for men:

The correlation between CHOL and liver enzymes was consistently weak across all months. Although cholesterol metabolism is partly regulated by liver function, the lack of significant associations may imply that the changes in cholesterol levels were not substantial enough to impact hepatic markers in this population(93).

Table(3-10) (a) Correlation between CHOL and ALT, AST, ALP for women.

Month 1				
	CHOL1	ALT1	AST1	ALP1
CHOL1	1	0.20	0.63	0.18
Month 2				
	CHOL2	ALT2	AST2	ALP2
CHOL2	1	0.43	0.29	0.41
Month 3				
	CHOL3	ALT3	AST3	ALP3
CHOL3	1	0.35	-0.22	0.50
Month 4				
	CHOL4	ALT4	AST4	ALP4
CHOL4	1	0.51	0.07	0.33

Table(3-10) (b) Correlation between CHOL and ALT, AST, ALP for girls.

Month 1				
	CHOL1	ALT1	AST1	ALP1
CHOL1	1	0.03	-0.20	-0.21
Month 2				
	CHOL2	ALT2	AST2	ALP2

CHOL2	1	-0.04	-0.16	-0.13
Month 3				
	CHOL3	ALT3	AST3	ALP3
CHOL3	1	0.02	-0.24	0.02
Month 4				
	CHOL4	ALT4	AST4	ALP4
CHOL4	1	0.005	-0.27	-0.04

Table(3-10) (c) Correlation between CHOL and ALT, AST, ALP for boys.

Month 1				
	CHOL1	ALT1	AST1	ALP1
CHOL1	1	0.03	0.01	0.09
Month 2				
	CHOL2	ALT2	AST2	ALP2
CHOL2	1	0.16	0.13	-0.17
Month 3				
	CHOL3	ALT3	AST3	ALP3
CHOL3	1	0.17	0.13	-0.11
Month 4				

	CHOL4	ALT4	AST4	ALP4
CHOL4	1	0.18	0.12	0.007

Table(3-10) (d) Correlation between CHOL and ALT, AST, ALP for men.

Month 1				
	CHOL1	ALT1	AST1	ALP1
CHOL1	1	021	0.08	0.17
Month 2				
	CHOL2	ALT2	AST2	ALP2
CHOL2	1	0.31	0.26	0.25
Month 3				
	CHOL3	ALT3	AST3	ALP3
CHOL3	1	0.27	0.20	0.11
Month 4				
	CHOL4	ALT4	AST4	ALP4
CHOL4	1	0.27	0.25	0.34

3.5.5 Correlation Between HDL and Liver Enzymes (ALT, AST, ALP)

a) for women:

Correlations were consistently weak (ranging 0.07–0.59), except for a moderate ALT correlation in Month 4 (r = 0.59) as shown in table (3-7) (a), potentially reflecting improved liver metabolic response (94).

b) for girls:

HDL showed negative and weak correlations with ALT and ALP across all months, with values generally below r = ±0.3.

Low HDL levels are associated with liver dysfunction in metabolic syndromes, but the correlation strength is often modest. An observational study concluded that HDL has limited utility as a liver biomarker unless used in conjunction with other indices(95).

c) for boys:

The most notable relationship here is between HDL and ALT (r = 0.65 in month one) as shown in table (3-7)(c), with moderate correlations persisting across other months. This might reflect improved hepatic function with elevated HDL levels, which are known to exert anti-inflammatory and protective effects on liver metabolism. This supports findings in recent studies that link HDL elevation with reduced hepatic steatosis and inflammation.

d)for men:

HDL had modest and non-significant associations with ALT, AST, and ALP as shown in table (3-7) (d), despite its function in reverse cholesterol transport and

liver health. This might be a result of other variables that affect HDL without affecting hepatic enzyme activity, such food, exercise, or insulin sensitivity(96).

Table (3-11) (a) Correlation Between HDL and Liver Enzymes (ALT, AST, ALP) in women.

Month 1				
	HDL1	ALT1	AST1	ALP1
HDL1	1	0.22	0.07	0.22
Month 2				
	HDL2	ALT2	AST2	ALP2
HDL2	1	0.21	0.20	0.13
Month 3				
	HDL3	ALT3	AST3	ALP3
HDL3	1	0.29	-0.03	0.02
Month 4				
	HDL4	ALT4	AST4	ALP4
HDL4	1	0.59	0.24	0.36

Table (3-11) (b) Correlation Between HDL and Liver Enzymes (ALT, AST, ALP) in girls.

Month 1				
	HDL1	ALT1	AST1	ALP1
HDL1	1	-0.20	0.06	-0.21
Month 2				
	HDL2	ALT2	AST2	ALP2
HDL2	1	-0.08	-0.06	-0.34
Month 3				
	HDL3	ALT3	AST3	ALP3
HDL3	1	-0.22	-0.01	-0.06
Month 4				
	HDL4	ALT4	AST4	ALP4
HDL4	1	-0.33	0.24	-0.22

Table (3-11) (c) Correlation Between HDL and Liver Enzymes (ALT, AST, ALP) in boys.

Month 1				
	HDL1	ALT1	AST1	ALP1
HDL1	1	0.65	0.01	0.19

Month 2				
	HDL2	ALT2	AST2	ALP2
HDL2	1	0.35	-0.12	0.16
Month 3				
	HDL3	ALT3	AST3	ALP3
HDL3	1	0.50	0.06	0.18
Month 4				
	HDL4	ALT4	AST4	ALP4
HDL4	1	0.42	0.20	0.22

Table (3-11) (d) Correlation Between HDL and Liver Enzymes (ALT, AST, ALP) in men.

Month 1				
	HDL1	ALT1	AST1	ALP1
HDL1	1	-0.61	-0.49	0.15
Month 2				
	HDL2	ALT2	AST2	ALP2
HDL2	1	0.06	0.003	0.10
Month 3				
	HDL3	ALT3	AST3	ALP3
HDL3	1	-0.09	-0.10	0.18

Month 4				
	HDL4			
HDL4	1	-0.10	-0.10	-0.08

3.5.6 Correlation between G.H and lipid profile

a) for women:

Negative correlations with HDL and CHOL in early months (r up to -0.52) align with GH's known lipolytic and anti-lipogenic effects.

Month 4 showed weak positive associations as shown in table (3-8) (a), possibly due to hormonal regulation variability over time.

b) for girls:

Growth hormone showed weak and non-significant correlations with TG, CHOL, HDL, and LDL in all months (r range: -0.49 to 0.08)as shown in table(3-8) (b).

GH can affect lipid metabolism, but serum GH levels vary and are not directly predictive of lipid levels in healthy individuals. Recent data suggest GH affects lipid metabolism indirectly via insulin-like growth factor-1 (IGF-1) rather than directly (97).

c) for boys:

Growth hormone (GH) was negatively correlated with LDL (r = –0.55, –0.53) and CHOL as shown in table (3-8) (c), especially in the first two months. GH has known lipolytic effects and improves lipid clearance, especially LDL and total cholesterol, in obese patients. These results align with recent findings that GH

therapy or natural increases in GH are associated with improved lipid metabolism and reductions in LDL (98,99)

d)for men:

The correlation between GH and lipid parameters remained weak throughout. Although GH is known to influence lipid metabolism, particularly by promoting lipolysis, this study suggests the relationship was either blunted or obscured—possibly due to age-related decline in GH secretion or insulin resistance associated with obesity.

Table (3-12) (a) Correlation between Growth Hormone (GH) and Lipids (TG, CHOL, HDL, LDL) for women.

Month 1					
	G.H1	TG1	CHOL1	HDL1	LDL1
G.H1	1	-0.02	-0.38	-0.52	0.11
Month 2					
	G.H2	TG2	CHOL2	HDL2	LDL2
G.H2	1	-0.14	-0.19	-0.49	-0.22
Month 3					
	G.H3	TG3	CHOL3	HDL3	LDL3
G.H3	1	-0.13	0.06	-0.43	-0.04
Month 4					
	G.H4	TG4	CHOL4	HDL4	LDL4

G.H4	1	0.21	0.30	-0.16	0.22

Table (3-12) (b) Correlation between Growth Hormone (GH) and Lipids (TG, CHOL, HDL, LDL) for girls.

Month 1					
	G.H1	TG1	CHOL1	HDL1	LDL1
G.H1	1	0.02	-0.05	-0.34	-0.32
Month 2					
	G.H2	TG2	CHOL2	HDL2	LDL2
G.H2	1	-0.14	-0.19	-0.49	-0.22
Month 3					
	G.H3	TG3	CHOL3	HDL3	LDL3
G.H3	1	-0.01	0.05	-0.10	0.03
Month 4					
	G.H4	TG4	CHOL4	HDL4	LDL4
G.H4	1	0.08	-0.33	0.21	0.02

Table (3-12) (c) Correlation between Growth Hormone (GH) and Lipids (TG, CHOL, HDL, LDL) for boys.

Month 1					
	G.H1	TG1	CHOL1	HDL1	LDL1
G.H1	1	0.13	-0.41	0.10	-0.55
Month 2					
	G.H2	TG2	CHOL2	HDL2	LDL2
G.H2	1	0.19	-0.40	-0.01	-0.53
Month 3					
	G.H3	TG3	CHOL3	HDL3	LDL3
G.H3	1	0.23	-0.28	-0.03	-0.28
Month 4					
	G.H4	TG4	CHOL4	HDL4	LDL4
G.H4	1	0.41	-0.30	-0.14	-0.09

Table (3-12) (d) Correlation between Growth Hormone (GH) and Lipids (TG, CHOL, HDL, LDL) for men.

Month 1					
	G.H1	TG1	CHOL1	HDL1	LDL1
G.H1	1	0.12	0.32	-0.36	-0.06

Month 2					
	G.H2	TG2	CHOL2	HDL2	LDL2
G.H2	1	0.26	0.43	-0.04	-0.11
Month 3					
	G.H3	TG3	CHOL3	HDL3	LDL3
G.H3	1	0.19	0.42	0.02	-0.01
Month 4					
	G.H4	TG4	CHOL4	HDL4	LDL4
G.H4	1	0.24	0.44	-0.29	0.15

3.5.7 Correlation between Insulin and Lipid Parameters

a) for women:

Insulin showed mostly weak and insignificant effects across all months.

Exception in Month 3: moderate to strong correlation with HDL (r = 0.61) as shown in Table 3-9 (a), which may suggest insulin sensitivity improvements(100).

b) for girls:

Insulin showed modest correlations in some months (e.g., r = 0.34 with TG in month 2) as shown in Table 3-9 (b), but most were weak and statistically insignificant.

Insulin resistance is a well-established contributor to dyslipidemia, especially in metabolic syndrome. Still, single-time point insulin measurements may not correlate tightly with lipids(101).

c) for boys:

Insulin showed moderate inverse correlation with HDL (e.g., r = –0.67 in month two) as shown in Table 3-9 (c), indicating that insulin resistance may impair HDL metabolism. These results align with modern studies that link insulin resistance to reduced HDL levels and impaired lipid handling in obesity.

d)for men:

The analysis revealed a consistently negative but weak correlation between insulin and lipid variables, as shown in Table 3-9 (d), particularly for triglycerides (TG). This supports the existing literature, which highlights the role of insulin resistance in hypertriglyceridemia. However, the weak magnitude of correlation may reflect complex feedback loops in metabolic regulation or short study duration (102).

Table (3-13) (a) Correlation between Insulin and Lipid Parameters (TG, CHOL, HDL) in women.

Month 1				
	Insulin1	TG1	CHOL1	HDL1
Insulin1	1	0.13	0.01	0.18
Month 2				
	Insulin2	TG2	CHOL2	HDL2
Insulin2	1	0.08	0.23	0.25

Month 3				
	Insulin3	TG3	CHOL3	HDL3
Insulin3	1	0.001	0.11	0.61
Month 4				
	Insulin4	TG4	CHOL4	HDL4
Insulin4	1	0.12	0.18	-0.08

Table (3-13) (b) Correlation between Insulin and Lipid Parameters (TG, CHOL, HDL) in girls.

Month 1				
	Insulin1	TG1	CHOL1	HDL1
Insulin1	1	0.11	0.21	-0.07
Month 2				
	Insulin2	TG2	CHOL2	HDL2
Insulin2	1	0.34	0.28	0.09
Month 3				
	Insulin3	TG3	CHOL3	HDL3
Insulin3	1	-0.05	0.21	-0.01
Month 4				
	Insulin4	TG4	CHOL4	HDL4
Insulin4	1	-0.28	0.09	-0.13

Table (3-13) (c) Correlation between Insulin and Lipid Parameters (TG, CHOL, HDL) in boys.

Month 1				
	Insulin1	TG1	CHOL1	HDL1
Insulin1	1	-0.02	-0.04	-0.47
Month 2				
	Insulin2	TG2	CHOL2	HDL2
Insulin2	1	0.04	-0.01	-0.67
Month 3				
	Insulin3	TG3	CHOL3	HDL3
Insulin3	1	-0.11	-0.01	-0.59
Month 4				
	Insulin4	TG4	CHOL4	HDL4
Insulin4	1	-0.17	0.03	-0.32

Table (3-13) (d) Correlation between Insulin and Lipid Parameters (TG, CHOL, HDL) in men.

Month 1				
	Insulin1	TG1	CHOL1	HDL1
Insulin1	1	-0.35	-0.15	-0.21
Month 2				
	Insulin2	TG2	CHOL2	HDL2

Insulin2	1	-0.43	-0.10	0.13
Month 3				
	Insulin3	TG3	CHOL3	HDL3
Insulin3	1	-0.48	-0.07	0.12
Month 4				
	Insulin4	TG4	CHOL4	HDL4
Insulin4	1	-0.37	-0.28	-0.18

3.5.8 Correlation between Insulin and LDH

a) for women:

Month 3: moderate correlation (r = 0.44) as shown in table (3-10)(a), reflecting potential mitochondrial dysfunction or oxidative stress response in insulin resistance(103).

b) for girls;

Insulin and LDH correlations ranged from (-0.12 to 0.25) as shown in Table 3-10 (b), indicating no significant relationship.

Insulin has no direct known effect on LDH, and LDH elevation generally signals tissue breakdown, not insulin sensitivity(104).

c) for boys:

The correlation between insulin and LDH was moderate in the first month (r = 0.60) but diminished over time, as shown in Table 3-10(c). This could indicate

metabolic stress or early subclinical inflammation, which is often associated with hyperinsulinemia in obesity. Recent work highlights LDH as a marker for early metabolic strain in individuals with insulin resistance.

d)for men:

The correlation between insulin and LDH was weakly positive in months 2–4. Given LDH role in anaerobic metabolism, this could indicate a mild link between hyperinsulinemia and subclinical tissue stress or hypoxia in obese individuals, yet the relationship lacked strength for clinical significance.

Table (3-14) (a) Correlation between Insulin and LDH in women.

Month 1		
	Insulin1	LDH 1
Insulin1	1	0.16
Month 2		
	Insulin2	LDH2
Insulin2	1	0.15
Month 3		
	Insulin3	LDH3
Insulin3	1	0.44
Month 4		
	Insulin4	LDH4
Insulin4	1	0.34

Table (3-14) (b) Correlation between Insulin and LDH in girls.

Month 1		
	Insulin1	LDH 1
Insulin1	1	0.25
Month 2		
	Insulin2	LDH2
Insulin2	1	0.06
Month 3		
	Insulin3	LDH3
Insulin3	1	-0.08
Month 4		
	Insulin4	LDH4
Insulin4	1	-0.12

Table (3-14) (c) Correlation between Insulin and LDH in boys.

Month 1		
	Insulin1	LDH 1
Insulin1	1	0.60
Month 2		
	Insulin2	LDH2

Insulin2	1	0.06
Month 3		
	Insulin3	LDH3
Insulin3	1	0.33
Month 4		
	Insulin4	LDH4
Insulin4	1	0.25

Table (3-14) (d) Correlation between Insulin and LDH in men.

Month 1		
	Insulin1	LDH 1
Insulin1	1	-0.32
Month 2		
	Insulin2	LDH2
Insulin2	1	0.29
Month 3		
	Insulin3	LDH3
Insulin3	1	0.37
Month 4		
	Insulin4	LDH4

Insulin4	1	0.36

3.5.9 Correlation between Insulin and RBS (Random Blood Sugar)

a) for women:

All months showed weak correlation; only Month 4 reached (r = 0.43) as shown in table (3-11) (a), suggesting delayed glycemic response to insulin levels(105).

b) for girls:

The insulin-RBS correlation was variable across months (r from -0.33 to +0.35) as shown in table (3-11) (b), with no consistent pattern. Insulin and glucose levels are expected to correlate under controlled testing, but random glucose measurements are inherently variabl(106).

c) for boys:

In all months, no strong correlations were observed between insulin and RBS. However, a mild negative trend appeared in month three (r = –0.39) as shown in table (3-11) (c), suggesting possible early glycemic control improvement with dietary regulation. This is consistent with longitudinal findings that dietary changes may improve insulin sensitivity before a notable reduction in RBS is observed.

d)for men:

Insulin-RBS correlations were weak and inconsistent as shown in table (3-11) (d), suggesting possible insulin resistance or variable glycemic control among subjects. This aligns with the pathophysiology of metabolic syndrome, where elevated insulin levels may not effectively lower glucose(107).

Table (3-15) (a) Correlation between Insulin and RBS (Random Blood Sugar) in women.

Month 1		
	Insulin1	RBS1
Insulin1	1	-0.07
Month 2		
	Insulin2	RBS2
Insulin2	1	-0.15
Month 3		
	Insulin3	RBS3
Insulin3	1	0.01
Month 4		
	Insulin4	RBS4
Insulin4	1	0.43

Table (3-15) (b) Correlation between Insulin and RBS (Random Blood Sugar) in girls.

Month 1		
	Insulin1	RBS1
Insulin1	1	-0.32

Month 2		
	Insulin2	**RBS2**
Insulin2	1	-0.18
Month 3		
	Insulin3	**RBS3**
Insulin3	1	0.35
Month 4		
	Insulin4	**RBS4**
Insulin4	1	-0.33

Table (3-15) (c) Correlation between Insulin and RBS (Random Blood Sugar) in boys.

Month 1		
	Insulin1	**RBS1**
Insulin1	1	-0.04
Month 2		
	Insulin2	**RBS2**
Insulin2	1	0.20
Month 3		
	Insulin3	**RBS3**

Insulin3	1	-0.39
Month 4		
	Insulin4	RBS4
Insulin4	1	-0.007

Table (3-15) (d) Correlation between Insulin and RBS (Random Blood Sugar) in men.

Month 1		
	Insulin1	RBS1
Insulin1	1	-0.32
Month 2		
	Insulin2	RBS2
Insulin2	1	0.05
Month 3		
	Insulin3	RBS3
Insulin3	1	0.04
Month 4		
	Insulin4	RBS4
Insulin4	1	-0.16

3.5.10 Correlation between Insulin and Lipase

a) for women:

All correlations are weak and insignificant, as shown in Table 3-12 (a), indicating limited pancreatic enzyme feedback via insulin pathways under normal physiology.

b) for girls:

Insulin had a strong negative correlation with lipase in months 2, 3, and 4 (r values of -0.55 to -0.65) as shown in Table 3-12 (b), indicating a potential inverse relationship.

Emerging evidence suggests that insulin may suppress pancreatic enzyme secretion, including lipase, in metabolic stress states(108).

c) for boys:

Correlations between insulin and lipase were consistently weak as shown in table (3-12) (c), suggesting minimal direct association. This is expected, as lipase operates more directly in fat digestion rather than in insulin-regulated lipid storage or glucose metabolism.

d)for men:

The analysis found weak and declining correlation between insulin and lipase across the months as shown in table (3-12) (d), transitioning from mildly positive to negative. This could indicate that lipase activity is not substantially modulated by circulating insulin levels in the short term or in the absence of overt pancreatic dysfunction(109).

Table (3-16) (a) Correlation between Insulin and Lipase in women.

Month 1		
	Insulin1	Lipase1
Insulin1	1	0.17
Month 2		
	Insulin2	Lipase2
Insulin2	1	0.10
Month 3		
	Insulin3	Lipase3
Insulin3	1	-0.13
Month 4		
	Insulin4	Lipase4
Insulin4	1	0.06

Table (3-16) (b) Correlation between Insulin and Lipase in girls.

Month 1		
	Insulin1	Lipase1
Insulin1	1	0.31
Month 2		
	Insulin2	Lipase2

Insulin2	1	-0.59
Month 3		
	Insulin3	Lipase3
Insulin3	1	-0.65
Month 4		
	Insulin4	Lipase4
Insulin4	1	-0.55

Table (3-16) (c) Correlation between Insulin and Lipase in boys.

Month 1		
	Insulin1	Lipase1
Insulin1	1	0.02
Month 2		
	Insulin2	Lipase2
Insulin2	1	0.10
Month 3		
	Insulin3	Lipase3
Insulin3	1	-0.01
Month 4		

	Insulin4	Lipase4
Insulin4	1	-0.24

Table (3-16) (d) Correlation between Insulin and Lipase in men.

Month 1		
	Insulin1	Lipase1
Insulin1	1	0.34
Month 2		
	Insulin2	Lipase2
Insulin2	1	0.21
Month 3		
	Insulin3	Lipase3
Insulin3	1	0.06
Month 4		
	Insulin4	Lipase4
Insulin4	1	-0.08

3.6. Sensitivity and Specificity Analysis

Sensitivity and specificity are crucial statistics that help to understand how well clinical tests diagnose diseases. Sensitivity, or the true positive rate, highlights a test's strength in identifying individuals with a condition. On the other hand, specificity, or the true negative rate, showcases its effectiveness in recognizing those who are healthy.

In this analysis, we will explore five biomarkers: Vitamin D3 (VD3), Growth Hormone (GH), Insulin, Lipase, and Lactate Dehydrogenase (LDH). We will examine their diagnostic performance among various demographic groups, including women, girls, boys, and men, providing valuable insights into their health.

3.6.1 Sensitivity and Specificity of biomarkers in women

In women, VD3 demonstrated exceptional diagnostic performance, achieving a sensitivity and specificity of 100% at a cut-off value of 20.45 ng/mL, as indicated in Table 3-13. This indicates complete accuracy in identifying both diseased and healthy individuals. In contrast, GH and insulin showed 0% sensitivity and 100% specificity, meaning they failed to identify any true positive cases (i.e., all actual diseased individuals were misclassified as healthy), even though they accurately identified healthy individuals.

Lipase and LDH could not be evaluated due to uniformity in sample results (i.e., all values were either positive or negative), making it impossible to calculate sensitivity, specificity, or establish a diagnostic threshold. These patterns suggest that these biomarkers may lack significant variability, or their diagnostic relevance needs further validation in a larger and more diverse cohort.

Table (3-17) Sensitivity and Specificity for women.

Tests	Positive	Negative	Total	Cut-off	Sensitivity	Specificity
VD3	**33**	**23**	**56**	**20.45**	**1**	**0**
G.H	**17**	**39**	**56**	**-0.91**	**0**	**0**
Insulin	**10**	**46**	**56**	**2.28**	**0**	**0**
Lipase	**0**	**56**	**56**	**null**	**null**	**null**
LDH	**56**	**0**	**56**	**null**	**null**	**null**

3.6.2 Sensitivity and Specificity of biomarkers in girls:

Similar to the findings in the adult female cohort, Vitamin D3 (VD3) demonstrated excellent diagnostic utility in girls, with both sensitivity and specificity reaching 100%. This was established using a cut-off point of 20.40 ng/mL, as shown in Table 3-14. In contrast, insulin failed to detect any positive cases, indicating a bias toward false negatives; however, it accurately classified all healthy individuals.

The diagnostic parameters for growth hormone (GH), lipase, and lactate dehydrogenase (LDH) could not be computed due to the lack of both positive and negative cases, reflecting the limitations seen in the adult female cohort. This highlights the need for stratified sampling and the potential requirement for alternative thresholds tailored specifically to pediatric populations.

Table (3-18) Sensitivity and Specificity for girls

Tests	Positive	Negative	Total	Cut-off	Sensitivity	Specificity
VD3	**29**	**39**	**68**	**20.40**	**1**	**0**
G.H	**0**	**568**	**68**	**null**	**null**	**null**
Insulin	**23**	**45**	**68**	**4.84**	**0**	**0**
Lipase	**0**	**68**	**68**	**null**	**null**	**null**
LDH	**68**	**0**	**68**	**null**	**null**	**null**

3.6.3. Sensitivity and Specificity of biomarkers in boys:

In the group of male adolescents, Vitamin D3, growth hormone (GH), and insulin demonstrated excellent classification performance, achieving 100% sensitivity and specificity with cutoff values of 20.1, 8.10, and 24.32 units, respectively, as shown in Table 3-15. This indicates that these biomarkers have strong potential to serve as reliable diagnostic indicators for boys.

In contrast, lactate dehydrogenase (LDH) revealed a sensitivity of 0%, meaning that all individuals with the condition were misclassified, although its specificity was 100%. Furthermore, the data for lipase were statistically uninformative due to a uniform distribution within the test population.These results underscore the variability in biomarker effectiveness across genders and highlight the importance of contextual validation.

Table (3-19) Sensitivity and Specificity for Boys.

Tests	Positive	Negative	Total	Cut-off	Sensitivity	Specificity
VD3	**49**	**11**	**60**	**20.1**	**1**	**0**
G.H	**3**	**57**	**60**	**8.10**	**1**	**0**
Insulin	**13**	**47**	**60**	**24.32**	**1**	**0**
Lipase	**0**	**60**	**60**	**null**	**null**	**null**
LDH	**57**	**3**	**60**	**292**	**0**	**0**

3.6.4. Sensitivity and Specificity of biomarkers in men

In adult males, VD3 demonstrated perfect diagnostic accuracy, achieving 100% sensitivity and specificity at a cutoff of 20 ng/mL, as shown in Table 3-16. The other biomarkers (GH, Insulin, Lipase, and LDH) did not provide diagnostic value in this cohort due to consistent test results, making further analysis impossible.

These limitations may arise from factors such as a small sample size, a narrow range of biomarker concentrations, or the need for demographic-specific thresholds. Therefore, future studies should aim to diversify and expand cohort sizes to encompass a broader range of clinical spectra.

Table (3-20) Sensitivity and Specificity for men.

Tests	Positive	Negative	Total	Cut-off	Sensitivity	Specificity
VD3	24	16	40	20	1	0
G.H	0	40	40	null	null	null
Insulin	0	40	40	null	null	null
Lipase	0	40	40	null	null	null
LDH	40	0	40	null	null	null

Generally, VD3 has demonstrated impressive diagnostic capabilities across various demographic groups, highlighting its promise as a primary biomarker for disease screening and monitoring, particularly in metabolic and nutritional evaluations. The challenges faced by GH and Insulin in identifying positive cases in specific cohorts highlight their limited standalone diagnostic value, indicating that they may be best used as supplementary markers. Furthermore, the difficulty in assessing Lipase and LDH across multiple groups highlights the need for more robust statistical approaches in analyzing homogeneous data. By adopting more inclusive and stratified study designs, we can ensure that all cases, both positive and negative, are effectively represented, leading to even better diagnostics.

3.7. Conclusions and Recommendations

3.7.1. Conclusions

This study offers valuable insights into the biochemical and metabolic impacts of obesity, highlighting significant areas of concern:

1. Participants with obesity showed notable impairments in glucose metabolism, marked by elevated fasting glucose and HbA1c levels. This signals challenges in glycemic control and suggests a heightened risk for developing type 2 diabetes, which we can address through proactive measures.

2. We observed a concerning prevalence of dyslipidemia, characterized by increased total cholesterol, triglycerides, and LDL cholesterol, coupled with lower HDL cholesterol levels. This combination can raise cardiovascular risk, but the right lifestyle changes can help improve these values.

3. The study clearly established a connection between obesity and insulin resistance, indicated by higher serum insulin levels and increased HOMA-IR scores. Recognizing this relationship is crucial for developing effective strategies to enhance insulin sensitivity.

4. Interestingly, serum urea and creatinine levels were moderately elevated among those with obesity, potentially signaling early stages of renal dysfunction, even without evident kidney disease. This highlights the importance of ongoing monitoring.

5. Additionally, increased activity of lipase and lactate dehydrogenase (LDH) enzymes points to systemic metabolic stress and possibly subclinical inflammation, which we can work to alleviate.

6. Strong correlations exist among BMI, waist circumference, and various biochemical parameters, further underscoring the connection between body composition and overall metabolic health.

In summary, the effects of obesity extend across the metabolic, cardiovascular, and renal systems. These findings underscore the vital importance of early prevention and intervention to foster better health outcomes for the future.

3.7.2. Recommendations

Building on the compelling insights uncovered in this study, it can strongly encourage the researchers to embrace the following transformative recommendations:

1. Early Biochemical Screening: Implement regular biochemical assessments for individuals with elevated BMI to identify metabolic risks at an early stage.

2. Lifestyle Modification Programs: Create integrated strategies that combine balanced diets, calorie reduction, and increased physical activity to promote weight management and metabolic stability.

3. Public Awareness Campaigns: Increase awareness of the health risks associated with obesity and the importance of preventive behaviors through community-based education initiatives.

4. Policy Development: Advocate for public health policies that enhance access to healthy foods, restrict the availability of processed foods, and foster environments that encourage physical activity.

5. Further Research: Support longitudinal studies to evaluate the long-term effects of dietary interventions and lifestyle changes on metabolic health in the obese population

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Appendixes

Kerbala University
College of Science
Department of Chemistry

Questionnaire for the Participant with Obesity & the Controls

1. Name: No. The date:
2. Gender: Male Female
3. Age:
4. Does the participant have any disease? Yes No

   If yes, explain:
5. Status of cigarette-smoking? Yes No
6. Status of alcoholic? Yes No
7. Time duration of the obesity:
8. Dose any family member have obesity? Yes No

   If yes, how many and what the related with the participant:
9. Other infections around the body associated with obesity:
10. Other notes (other diseases):
11. Excluded cases (patients with): Diabetes, Autoimmune diseases, Pregnant women, and aged people.

Appendix (1): Questionnaire for the Participant with Obesity and the Controls

الخلاصة

يلعب النظام الغذائي دورًا حاسمًا في التأثير على السمنة، وهي مصدر قلق صحي عالمي متزايد. هدفت هذه الدراسة إلى تقييم التغيرات الكيميائية الحيوية المرتبطة بفقدان الوزن لدى الأفراد الذين يعانون من السمنة بعد الخضوع لنظام غذائي محدد. وقد أُجريت الدراسة في قسم الكيمياء كلية العلوم جامعة كربلاء، كربلاء، العراق. وجُمعت 116 عينة في الفترة من ديسمبر 2023 إلى فبراير 2025. وقد صُنف 56 مشاركًا على أنهم يعانون من السمنة، بينما شكل 60 فردًا سليمًا.

صُنف المشاركون إلى ثلاث مجموعات وفقًا لمؤشر كتلة الجسم :(BMI) سليم، زائد الوزن، وبدين. أُجريت سلسلة من الاختبارات المختبرية لقياس مستوى السكر في الدم، ومستويات الدهون، ومستويات إنزيمات الكبد والكلى، والأنسولين، وهرمون النمو. أُعيدت هذه الاختبارات بعد شهر و استمرت المتابعة فترة أربعة أشهر من اتباع النظام الغذائي.

كشفت النتائج عن تحسن كبير في الحالة الصحية للأفراد الذين يعانون من السمنة، بما في ذلك انخفاض في الوزن ومؤشر كتلة الجسم(BMI) ، وكلاهما أظهر تغيرات ذات دلالة إحصائية .($p = 0.0001$) بالإضافة إلى ذلك، سُجِّل تحسن ملحوظ في مستويات سكر الدم العشوائي (RBS) والأنسولين، حيث بلغت قيم الاحتمالية 0.001 و0.006 على التوالي. تشير هذه النتائج إلى انخفاض في مقاومة الأنسولين وانخفاض خطر الإصابة بمرض السكري من النوع الثاني.

علاوة على ذلك، أظهر ملف الدهون تغيرات ملحوظة، حيث انخفضت مستويات الدهون الثلاثية(TG) ، والكوليسترول الكلي(TC) ، والبروتين الدهني منخفض الكثافة(LDL) ، والليباز، بينما ارتفعت مستويات البروتين الدهني عالي الكثافة .(HDL) تُعد هذه التحسينات مهمة بشكل خاص لتحسين وظائف الكبد والشريان التاجي والشرايين، مما يؤثر إيجابًا على وظائف الكلى.

تُبرز هذه النتائج المثيرة التأثير الملحوظ للنظام الغذائي المقترح في الدراسة في مكافحة السمنة، وتعزيز المؤشرات الصحية ذات الصلة بشكل كبير لمستقبل أكثر صحة.

جامعة كربلاء

كلية العلوم

قسم الكيمياء

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رسالة مقدمة الى

مجلس كلية العلوم – جامعة كربلاء

من قبل

ريام عباس مطشر

بكالوريوس علوم في كيمياء (2019) / جامعة كربلاء

بأشراف

أ.م.د. ثائر مهدي مدلول

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