

**A THESIS SUBMITTED TO  
THE GRADUATE SCHOOL OF NATURAL AND APPLIED SCIENCES  
OF ÇANKIRI KARATEKİN UNIVERSITY**

**EXPLORATION OF THE RELATIONSHIP BETWEEN ASPROSIN  
WITH OXIDATIVE STRESS INDEX IN OBESE IRAQI PATIENTS**

**IN PARTIAL FULFILLMENT OF THE REQUIREMENTS  
FOR  
THE DEGREE OF MASTER OF SCIENCE  
IN  
CHEMISTRY**

**BY**

**ALI THAER MOHAMMED ALSAJRI**

**ÇANKIRI**

**2022**

EXPLORATION OF THE RELATIONSHIP BETWEEN ASPROSIN WITH  
OXIDATIVE STRESS INDEX IN OBESE IRAQI PATIENTS

By Ali Thaer Mohammed ALSAJRI

May 2022

We certify that we have read this thesis and that in our opinion it is fully adequate, in scope and in quality, as a thesis for the degree of Master of Science

**Advisor** : Assoc. Prof. Dr. Şevki ADEM

**Co-Advisor** : Asst. Prof. Dr. Mustafa Taha MOHAMMED

**Examining Committee Members:**

**Chairman** : Prof. Dr. Volkan EYÜPOĞLU  
Chemistry  
Çankırı Karatekin University

**Member** : Asst. Prof. Dr. Beytullah EREN  
Environmental Engineering  
Sakarya University

**Member** : Assoc. Prof. Dr. Şevki ADEM  
Chemistry  
Çankırı Karatekin University

**Approved for the Graduate School of Natural and Applied Sciences**

**Prof. Dr. İbrahim ÇİFTÇİ**  
**Director of Graduate School**

**I hereby declare that all information in this document has been obtained and presented in accordance with academic rules and ethical conduct. I also declare that, as required by these rules and conduct, I have fully cited and referenced all material and results that are not original to this work.**

**Ali Thaer Mohammed ALSAJRI**

## ABSTRACT

### EXPLORATION OF THE RELATIONSHIP BETWEEN ASPROSIN WITH OXIDATIVE STRESS INDEX IN OBESE IRAQI PATIENTS

Ali Thaer Mohammed ALSAJRI

Master of Science in Chemistry

Advisor: Assoc. Prof. Dr. Şevki ADEM

Co-Advisor: Asst. Prof. Dr. Mustafa Taha MOHAMMED

May 2022

The present study is a case control that included 120 obese and also its included 60 healthy controls. The mean age of obese was  $(34.2 \pm 9.37)$  years. whereas that of controls was  $(29.22 \pm 12.83)$  p value 0.038,. On the other hand, the results showed that the weight of controls  $(69.15.1 \pm 6.5)$  is a significant different with patients cases  $(81.6 \pm 11.96)$  p value = 0.011 but the height thers no significant may be aafect mean and SD of control  $161.88 \pm 4.5$  as compared with patient group  $(161.54 \pm 7.49)$ , p value  $>0.05$ . The results of BMI showed that controls  $(22.13 \pm 3.8)$  are significantly different from patients group cases  $(29.9 \pm 4.0)$  p-value 0.01. The level of total oxidant status has elevated significantly ( $P < 0.01$ ) in obese patients cases  $(4.12 \pm 0.51)$  a compared to healthy control  $(1.19 \pm 0.22)$ . A significant higher level of TOS has observed obese patients compared control p-value  $< 0.01$ . This result shown a highly significant decrease of vitamin C. Relationship between patients and healthy control group in order to Asporin. This finding appeared a significant difference between patients and control groups in comparison between mean values of asporin for control,  $(25.879 \pm 1.4271, 13.516 \pm 1.8931)$  respectively (p-value  $< 0.01$ ), also lipid profile have been asignificant correlation with obese patients as compared with control.

**2022, 33 pages**

**Keywords:** Obesity, BMI, Asprosin, Oxidative stress, Total antioxidant capacity.

## ÖZET

# OBEZ IRAKLI HASTALARDA ASPROSİN İLE OKSİDATİF STRES ENDEKSİ ARASINDAKİ İLİŞKİNİN İNCELENMESİ

Ali Thaer Mohammed ALSAJRI

Kimya, Yüksek Lisans

Tez Danışmanı: Doç. Dr. Şevki ADEM

Eş Danışman: Dr. Öğr. Üyesi Mustafa Taha MOHAMMED

Mayıs 2022

Bu çalışma 120 obez ve 60 sağlıklı kontrolü içeren bir vaka kontrolüdür. Obezlerin ortalama yaşı ( $34,2 \pm 9,37$ ) yılıdır. kontrollerinki ise ( $29,22 \pm 12,83$ ) p değeri 0,038 idi. Öte yandan, sonuçlar, kontrollerin ağırlığının ( $69,15.1 \pm 6.5$ ) hasta vakaları ( $81,6 \pm 11,96$ ) p değeri = 0,011 ile anlamlı bir şekilde farklı olduğunu gösterdi, ancak bunların yüksekliği, kontrolün ortalama ve SD'sini etkilemeyebilir. Hasta grubu ( $161,54 \pm 7,49$ ) ile karşılaştırıldığında ( $161,88 \pm 4,5$ ), p değeri  $>0.05$ . BMI sonuçları, kontrollerin ( $22,13 \pm 3,8$ ) hasta grubu vakalarından ( $29,9 \pm 4,0$ ) önemli ölçüde farklı olduğunu gösterdi p-değeri 0,01. Toplam oksidan durum seviyesi, obez hasta vakalarında ( $4,12 \pm 0,51$ ) sağlıklı kontrole ( $1,19 \pm 0,22$ ) kıyasla önemli ölçüde yükselmiştir ( $P < 0,01$ ). Kontrol p-değeri  $<0,01$  ile karşılaştırıldığında obez hastalarda anlamlı derecede daha yüksek bir TOS seviyesi gözlemlenmiştir. Bu sonuç, Asporin için hastalar ve sağlıklı kontrol grubu arasındaki ilişkide C vitamini düzeyinde oldukça anlamlı bir düşüş olduğunu göstermiştir. Bu bulgu, kontrol için sırasıyla ( $25,879 \pm 1,4271$ ,  $13,516 \pm 1,8931$ ) asporin ortalama değerleri arasında (p-değeri  $<0,01$ ) karşılaştırıldığında hastalar ve kontrol grupları arasında anlamlı bir fark ortaya çıktı, ayrıca lipid profili de obez hastalar ile aşağıdaki gibi anlamlı bir korelasyon gösterdi. kontrol ile karşılaştırılır.

**2022, 33 sayfa**

**Anahtar Kelimeler:** Obezite, BMI, Asprosin, Oksidatif stres, Toplam antioksidan kapasite

## **PREFACE AND ACKNOWLEDGEMENTS**

Dear Father: Whom God has endowed me with dignity and whose name I bear with pride, it is he who has brought me to this stage.

My beloved mother: The one who made the Lord - Glory be to Him - the earth under her feet, and honored her in writing.

To my brothers and sisters who have had a beautiful impact on my academic career.

I would like to thank my thesis advisor, Assoc. Prof. Dr. Şevki ADEM and his assistant. Mustafa Taha Muhammad on his patience. I would also like to thank my parents once again for their endless support during my studies, guidance and understanding.

**Ali Thaer Mohammed ALSAJRI**

**Çankırı-2022**

## CONTENTS

<b>ABSTRACT</b> .....	<b>i</b>
<b>ÖZET</b> .....	<b>ii</b>
<b>PREFACE AND ACKNOWLEDGEMENTS</b> .....	<b>iii</b>
<b>CONTENTS</b> .....	<b>iv</b>
<b>LIST OF SYMBOLS</b> .....	<b>vi</b>
<b>LIST OF ABBREVIATIONS</b> .....	<b>vii</b>
<b>LIST OF FIGURES</b> .....	<b>viii</b>
<b>LIST OF TABLES</b> .....	<b>ix</b>
<b>1. INTRODUCTION</b> .....	<b>1</b>
<b>1.1 Objectives of the Study</b> .....	<b>2</b>
<b>1.1.1 Specific Objectives</b> .....	<b>2</b>
<b>2. LITERATURE REVIEW</b> .....	<b>3</b>
<b>2.1 Asprine Definetion</b> .....	<b>3</b>
<b>2.1.1 Asprosin structure</b> .....	<b>3</b>
<b>2.1.2 Action of asprosin</b> .....	<b>4</b>
<b>2.1.3 Asporin and obesity</b> .....	<b>5</b>
<b>2.2 Clinical and Biochemical Analyses of Asprosin in Metabolic Diseases</b> .....	<b>7</b>
<b>2.3 Antioxidants and Asporin</b> .....	<b>8</b>
<b>2.3.1 Oxygen-reacting species</b> .....	<b>10</b>
<b>2.3.2 Mechanisms of antioxidant defense</b> .....	<b>10</b>
<b>2.4 Vitamin C</b> .....	<b>11</b>
<b>3. MATERIALS AND METHODS</b> .....	<b>12</b>
<b>3.1 Study of Population</b> .....	<b>12</b>
<b>3.2 Samples Collection</b> .....	<b>12</b>
<b>3.3 Data Collection</b> .....	<b>12</b>
<b>3.4 Blood Sampling and Processing</b> .....	<b>13</b>
<b>3.5 Resources and Equipment</b> .....	<b>13</b>
<b>3.6 Kits</b> .....	<b>13</b>
<b>3.7 Methods</b> .....	<b>14</b>
<b>3.7.1 Determination of total oxidant status</b> .....	<b>14</b>
<b>3.8 Biochemical Parameters</b> .....	<b>14</b>

3.8.1 Determination of serum asporin hormone .....	15
3.8.2 Determination of vitamin C .....	15
3.8.3 Detection of cholestrol by linear kit .....	16
3.8.4 Detection of TG by linear kit .....	17
3.8.5 Detection of LDL by linear kit.....	17
3.8.6 Detection of HDL by linear kit .....	17
3.8.7 VLDL– cholesterol estimation .....	18
3.9 Statistical Analysis.....	18
<b>4. RESULTS AND DISCUSSION .....</b>	<b>19</b>
4.1 Clinical Characteristics in Present Study.....	19
4.2 Oxidative Stress .....	22
4.2.1 Total oxidant status .....	23
4.2.2 Relation ship between vitamin C as as antioxidant levels with obsity .....	25
4.3 Relation Ship Between Asporin with Obsity.....	26
4.4 Levels of Lipid Profile and Obsity and Control Groups.....	27
4.5 Personal Correlation Between Asporin with Another Biochemical Marker .....	29
<b>5 CONCLUSIONS AND RECOMMENDATION.....</b>	<b>31</b>
5.2 Conclusions .....	31
5.3 Recommendations .....	31
<b>REFERENCES.....</b>	<b>32</b>
<b>CURRICULUM VITAE.....</b>	<b>36</b>

## LIST OF SYMBOLS

%	Percent
±	Plus-minus
°C	Degrees Celsius
cm	Centimeter
dL	Deciliter
g	Gram
kg	Kilogram
L	Liter
m <sup>2</sup>	Square meters
mg	Milligram
mL	Milliliters
mmol	Millimoles
ng	Nanogram
nm	Nanometer

## LIST OF ABBREVIATIONS

A	Absorbance
ADMA	Asymmetric dimethylarginine
ANOVA	Analysis of variance
CBC	Complete blood counts
CDC	Centers for disease control
EIZA	Enzyme immunoassay
ERK	Extracellular signal-regulated kinase



## LIST OF FIGURES

Figure 2.1 Asprosin action in meatobolic process (Ramnanan 2021) .....	5
Figure 4.1 Mean of age, for patient and control groups.....	20
Figure 4.2 Mean of weight, heghit and BMI for patient and control groups .....	20
Figure 4.3 Frequency % of smoking in stedied groups.....	21
Figure 4.4 Mean of TOS for patients cases with control .....	24
Figure 4.5 Mean of vitamin C for patients cases with control.....	26
Figure 4.6 Mean for asporin levels in stedied groups .....	27
Figure 4.7 Mean of lipid profile in patients and control groups .....	28



## LIST OF TABLES

Table 3.1 Tools and instruments which were used in the study and their company of manufacture .....	13
Table 3.2 Kits used in the study and their companies.....	13
Table 3.3 Erel method used.....	14
Table 4.1 General characteristics of present study .....	19
Table 4.2 Mean and SD of TOS panel for patients cases with control .....	23
Table 4.3 Mean and SD of vitamin C in patient and control groups .....	25
Table 4.4 Mean and SD for asporin in studied groups .....	26
Table 4.5 Mean and SD of lipid profile in present studied .....	28
Table 4.6 Personal correlation between hepcidin hormone and iron panel in present study population .....	30

## 1. INTRODUCTION

Asprosin's 30-kilogram C-terminus is made up of 140 amino acids (fibrillin1). When you don't eat, the WAT secretes a hormone that aids the liver in the production of glucose. Fasting has been associated to insulin resistance and polycystic ovarian syndrome (PCOS), as well as type 2 diabetes in certain persons (Elnagar et al. 2018).

People used to believe that adipose tissue was a passive source of energy storage, but this is no longer the case. Adipose tissue is the body's most active endocrine organ. It regulates adipokine synthesis and glucolipid breakdown through endocrine, autocrine, and paracrine functions (Kojta et al. 2020).

Asprosin is a novel adipokine discovered in 2016 after the genomes of newborns with FBN1 mutations were sequenced and physically examined. Asprosin is a substance produced by white adipose tissue that increases blood sugar levels. According to recent research, both people and mice have much too much asprosin in their systems. Asprosin reduces insulin sensitivity by causing muscle and islet cells to undergo apoptosis or necrosis (Romere et al. 2016).

Because asprosin makes it simpler for OLFR734 hepatic receptors to bind, mice with gene deletion and siRNA interference have increased glycogenolysis. Asprosin is not an anorectic medication. It is, instead, a hormone. Asprosin must first bind to OLFR734 receptors on the surface of the cells before AgRP neurons can be activated. Overweight, diabetic, and PCOS patients have higher levels of the asprosin protein in their systems (Ke et al. 2020).

## **1.1 Objectives of the Study**

Evaluation of asprosin level in sera of obese patients as well as the levels of total oxidant status (TOS), in iraqi population.

### **1.1.1 Specific Objectives**

1. Examine the link between asporine and antioxidant status such as vitamin C.
2. In order to determine whether asporin is associated with any of the following factors: (serum Glucose,Triglycerides,Cholesterol,HDL-cholesterol,Hydrogen peroxide, Vitamin C, O-dianisidine, Xylenol orange,Ferrous ammonium sulfate, DTNB).
3. -the relation ship between asporin level within BMI and aging distribution.



## **2. LITERATURE REVIEW**

### **2.1 Asprosin Definition**

Asprosin is a protein produced by your fat cells during a fast. Carbamazepine, a corticosteroid, enhances the G protein-cAMP-PKA pathway's performance (Carbamazepine). To satisfy your want for eating, the hypothalamus activates the AgRP (adenosine receptor polypeptide). In those afflicted by obesity, asprosin levels are especially high, according to research. On the other hand, anti-asprosin antibodies were shown to help obese mice shed pounds by increasing asprosin levels and decreasing food intake. Recent studies found a link between asprosin and male fertility, notably in men, particularly in males. Asprosin may aid in the treatment of a reproductive problem caused by aging or obesity. Asprosin levels in the blood may be affected in a number of ways by exercise. Asprosin levels increase during anaerobic exercise but reduce during aerobic activity. Asprosin's role in the body and its potential as a drug require more study. (Wang *et al.* 2017).

#### **2.1.1 Asprosin structure**

Scientists discovered and called asprosin, a fasting-induced glucogenic hormone, near the end of the twentieth century (Romere et al. 2016). Three N-glycosylation sites are conceivable for the 30 kDa protein with 140 amino acids (aa) (the bacterial recombinant form of this protein is nonglycosylated and 17 kDa in size). The sperm protein profibrillin is encoded by two exons of the FBN1 gene. This protein is derived from asprosin, which is produced when fibrillin-1 protein degrades (Mishra et al. 2021).

Obesity and diabetes induced by diet are less likely to occur in animals with the Fbn1NPS/+ phenotype, according to studies using Fbn1NPS/+ murine and rabbit models (DM). Mishra et al. 2021).

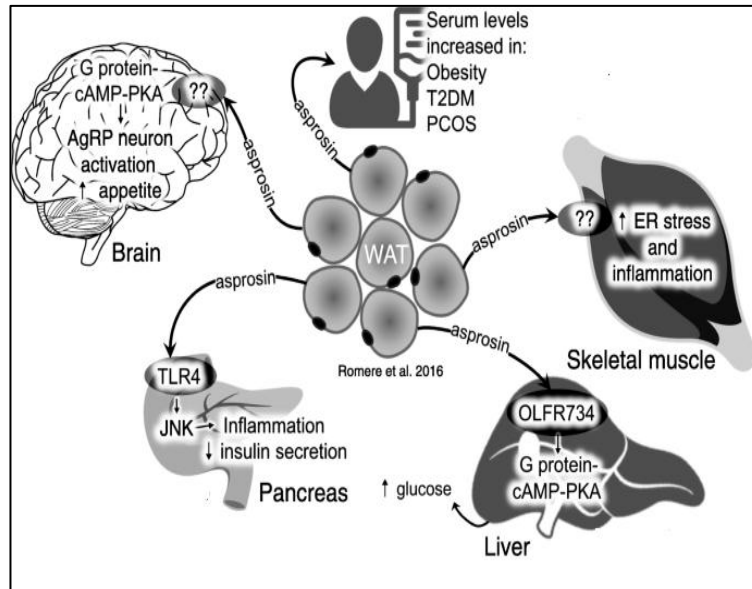
People with acromegaly, which is caused by insulin resistance and diabetes, and adipose tissue abnormalities have reduced asprosin levels (fat loss). Mishra et al. 2021).

Asprosin levels in fasting adipocytes derived from adipose tissue range from 5.94 to 3.04 nmol/L in healthy males and from 4.02 to 0.49 nmol/L in healthy women. Asprosin only functions for 20–145 minutes in its bacterial His-Tag form. It only acts for 145–145 minutes in its glycosylated form. Mishra et al. 2021).

### **2.1.2 Action of asprosin**

An increase in glucose production and release may occur as a result of asprosin's ability to activate the Olfr734 receptor in the liver. Asprosin stimulates glucose production in the liver, which benefits both persons who are underweight and those who are overweight. Both humans and mice possess similar OR4M1 receptors in their bodies. While the body is in a state of fasting, the liver causes certain proteins, such as G proteins, cAMP messengers, and protein kinase A, to be stimulated. This causes the hepatocytes to release glucose more quickly. (Li et al. 2019). Li and his fellow employees (2019)

Asprosin has no effect on glucagon or catecholamines, and as a result, it has no impact on the production of glucose (Figure 2.1). As can be seen in Figure 2.1, when insulin levels are high, asprosin is unable to carry out its job, which in turn prevents PKA and glucose release from occurring. Insulin and asprosin are said to compete with one another in terms of their functions, according to this study. As it does during periods of fasting, the synthesis of asprosin increases when glucose levels are low and drops when glucose levels are high (feeding condition). After a night of abstaining from food, the asprosin levels in the blood should increase and then decline in step with the circadian cycle. The levels of insulin and glucose in the blood immediately begin to increase after being given recombinant asprosin. (Ramnanan 2021).



**Figure 2.1** Asprosin action in metabolic process (Ramnanan 2021)

### 2.1.3 Asporin and obesity

Recent studies have shown that asprosin may play an important part in obesity; yet, these studies also seem to contradict one another. There have been a number of research that have shown a connection between obesity and higher levels of the chemical asprosin in both people and animals. By using antibodies that are specific for asprosin, researchers were able to demonstrate that obese mice had a lower body weight and a reduced intake of food, which indicates that their blood asprosin levels are unusually high (Mishra et al. 2021)

There is a correlation between asprosin levels and both cholesterol levels and the circumference of the waist (TG) According to the findings of another piece of research, the salivary glands in the human body have the capability of manufacturing asprosin. The levels of asprosin and low-density lipoprotein cholesterol (LDL-C) in the patients' saliva increased in proportion to the growth of their body mass index (BMI) (Ugur et al. 2019).

Asprosin levels were significantly higher in the obese participants in a study that also included 57 healthy adults as controls. The obese participants had a BMI that was more

than 35 kg/m<sup>2</sup>, while the healthy participants had a BMI that was less than 35 kg/m<sup>2</sup>. In addition, the research discovered that those who had bariatric surgery and had greater blood levels of asprosin lost more weight six months following the procedure. In the first six months after surgery, patients who dropped more than 55 percent of their body weight had considerably greater pre-surgical asprosin levels than patients who did not lose any weight at all (those who lost less than 35 percent of their body weight within 6 months after surgery). It is possible to predict effective responders with a sensitivity of 76% and a specificity of 75% by evaluating asprosin levels (Wang et al. 2017)

The findings obtained from a (Sasaki et al. 2018). AM6545 is a neutral antagonist of peripheral cannabinoid receptors, and research has shown that it may prevent the weight gain that is caused by monosodium glutamate (MSG). The ability of AM6545 to reduce levels of asprosin in the blood may have contributed to a reduction in adiposity and insulin resistance in MSG mice.

Despite the failure of observational studies to show a causal association between the two, further study is required to confirm the link between obesity and the asprosin biomarker. This is the case even though the correlation cannot be established as being caused by obesity. According to Jung et al research, 's the results of earlier studies have been all over the place, but recombinant asprosin does not, contrary to what was previously believed, cause mice to either put on or lose weight. [Citation needed] (Jung et al. 2019).

As far as researchers are aware, there is no link between asprosin and obesity. Plasma asprosin levels were found to be lower, hunger was reduced, and excessive lethargy was seen in study participants as well as mice with an NPS-associated mutation. A chemical known as asprosin has the potential to cause changes in eating habits. According to a new research, serum asprosin may be able to pass the blood-brain barrier, which may lead to increased appetite as well as obesity. This occurs because it activates AgRP neurons via a cAMP-dependent mechanism. (Mishra *et al.* 2021)

## 2.2 Clinical and Biochemical Analyses of Asprosin in Metabolic Diseases

Clinical studies have shown that elevated levels of asprosin are associated with both obesity and diabetes. As a result of having higher amounts of asprosin in their blood, patients with type 2 diabetes have been demonstrated to have had four different types of studies. FGF-1 and asprosin that is produced from FGF-1 have an effect on adipocyte activity as well as metabolic illness (Wang et al. 2018).

Patients diagnosed with type 2 diabetes mellitus exhibited greater plasma asprosin concentrations than those who did not have the condition, but these patients also had lower blood glucose levels, insulin levels, obesity markers, and lipid profiles (Wang et al. 2018)

Patients with type 2 diabetes had a decreased asprosin response to variations in glucose levels compared to the control group, which suggests that this difference may be a contributing factor. In addition, previous research has shown a correlation between levels of asprosin in saliva and plasma and indicators of obesity. It was discovered that the plasma levels of asprosin in 87 obese children and adolescents were lower than those seen in a healthy control group.

The amount of asprosin that can be detected in men's blood as opposed to the amount that can be found in women's blood reveals a sexual dimorphism. In a recent research that included 119 Chinese youngsters, levels of asprosin were shown to be connected to insulin resistance in the participants. It has been shown that elevated levels of asprosin are associated to the following metabolic disorders: According to the findings of a recent study, women who have Polycystic Ovarian Syndrome, a disease that is known to increase a person's risk of acquiring type 2 diabetes, had higher plasma levels of the hormone asprosin (Alan et al. 2019).

The levels of asprosin were significantly increased in pregnant women who were diagnosed with gestational diabetes and preeclampsia. The severity of unstable angina

pectoris is connected to the amount of asprosin that patients with the illness have in their blood. Asprosin levels have been shown to be connected to weight loss in patients who have had bariatric surgery, which lends credence to the idea that asprosin might be a valuable marker. Additional research and clinical tests are necessary before these results can be considered to have any kind of practical significance. The use of asprosin as a biomarker may be of assistance to diabetics and others suffering from other metabolic diseases. ELISA kits were used to conduct the asprosin concentration tests. On the other hand, it is not always possible to tell to what extent these strategies are effective. It is necessary to demonstrate and standardize the sensitivity and specificity of asprosin testing over a wide range of products in order to assess the validity and consistency of these tests. (Acara *et al.* 2018).

### **2.3 Antioxidants and Asporin**

In the context of Halliwell, any agent that significantly slows down or inhibits the oxidation process, even when present in relatively low quantities in comparison to an oxidizable substrate. Any therapy that lessens, stops, or gets rid of oxidative damage is referred to as "oxidative damage," and the phrase is used interchangeably. A major source of support may be found in one's network of friends and coworkers (Halliwell 2014)

The degradation of substrates like proteins, lipids, and DNA is what happens during the proteolytic oxidation process. Antioxidative capabilities have been shown by a broad variety of medications *in vitro*; however, this does not always translate to advantages at the cellular level. It's possible that the antioxidative properties of the chemical won't work on people. When determining the efficacy of a chemical substance, it is necessary to take into account a number of different aspects, including an antioxidant mechanism, a target biomolecule, and an exterior or internal site of action. Antioxidants prevent oxidative stress from causing harm to cells by eliminating free radicals from the body. This allows the body to function more normally (Willcox *et al.* 2004).

Metal-binding proteins like transferrin and albumin are the first line of defense in the body's antioxidative system (ROS). Antioxidants are included in the treatment plan's second stage of implementation. In the process of antioxidants stopping, spreading, and continuing the oxidation cycle, a stable byproduct is formed. This byproduct may then be used. Version 2.3.1 is now being presented by B. Halliwell and the rest of the development team. Vitamin C is one of the most effective anti-oxidants that are commercially accessible (ascorbic acid). Those who are unhappy with the way things are currently run (Willcox et al. 2004).

Free radicals are produced as a byproduct of cellular metabolism. For instance, the presence of an electron that is unpaired in a free radical makes it possible for the radical to exist in an unrestricted state. Because of the very large number of electrons that they contain, free radicals are inherently unstable, fleeting, and highly reactive. Due to the high reactivity of these compounds, it is possible that they will steal electrons from other molecules. Free radicals are produced whenever a molecule breaks apart and one of its electrons is released into the surrounding space. The process of cell death begins as soon as the cascade reaction has been initiated (Valko et al. 2007).

Free radicals are produced by the human body on a daily basis in order to fight inflammation, eliminate infections, and maintain control of the smooth muscles that are responsible for the regulation of internal organs and blood vessels. It is considered that a very tiny number of these helpful free radicals make their way into each and every cell that makes up the human body. When it comes to the development of many different illnesses, free radicals play an important role in the pathophysiology. Cancer, rheumatic pain, Alzheimer's disease, diabetes, Parkinson's disease, and Alzheimer's disease are only some of the illnesses that have been related to dangerous amounts of these compounds. Other disorders include Parkinson's disease and diabetes. (Phaniendra *et al.* 2015).

### **2.3.1 Oxygen-reacting species**

The most significant radicals found in biological systems are known as reactive oxygen species. These radicals are produced when oxygen is broken down (ROS). Due to the fact that they include oxygen, reactive oxygen species (also known as ROS) are also referred to as oxygen-containing reactive species. One example of an oxygen-reactive species is peroxyradical hydroperoxides. Another example is hyperchlorous acid. The hydroxyl radical, the singlet oxygen ( $^1O_2$ ), the peroxy radicals ( $LOO\cdot$  and  $LO\cdot$ ), and hypochlorous acid are all examples of reactive oxygen species (ROS). In this category include compounds such as nitric oxide (NO), peroxy nitrite ( $NO_2$ ), nitrogen dioxide ( $NO_2$ ), and other nitrogen oxides. (Foster and Miller 2012).

### **2.3.2 Mechanisms of antioxidant defense**

Antioxidant defense has the potential to prevent oxidative damage caused by free radicals, reactive oxygen species (ROS), and other kinds of oxidative stress. An "antioxidant" is a molecule that, when present in low concentrations, slows down or prevents the oxidation of an oxidizable substrate. Antioxidants may also be used to prevent cellular damage. As a result, the following is a definition of oxidative stress: In this circumstance, oxidants are more prevalent than antioxidants (Birben et al. 2012).

Antioxidants may reduce the number of unpaired electrons held by free radicals in one of two ways: by either consuming or donating electrons. There are three distinct classes of antioxidants.

The following are examples of endogenous antioxidants that fall within the first category:

Enzymes such as superoxide dismutase and catalase are responsible for activating some antioxidants.

The plasma's metal-binding proteins are responsible for two of the most significant sources of antioxidant activity in the body. Albumin, metallothioneine, and ferritin are some of the proteins that fall under this category. Also included are lactoferrin and lactoferrin. Antioxidants, which are these proteins, may be found in the body and bind to metals. (Birben et al (2012).

The human body has been showing signs of a decline in the levels of biochemical antioxidants such as uric acid. (Birben et al (2012).

Foods that contain antioxidants: Because of their potential use in medicine, the pharmaceutical industry has a strong interest in the total quantity of antioxidants that are found in nature, particularly those that are found in plants. Polyphenolic compounds include things like proanthocyanidins and flavan-3-ol oligomers, in addition to the vitamins A, C, and E. (Birben et al (2012).

## **2.4 Vitamin C**

There is a large range of foods that are rich in vitamin C, which is an important nutrient that acts as an antioxidant. When it comes to antioxidants, vitamin C (also known as ascorbic acid) and vitamin E (also known as tocopherol) stand out for their different abilities to act in the aqueous phase. By consuming vitamin C, it is possible to rid the body of reactive oxygen species (ROS), such as superoxide, hydrogen peroxide, and singlet oxygen. Vitamin C has the potential to inhibit the oxidation of tocopherols. Once glutathione has been oxidized, it begins the process of converting dehydroascorbate to ascorbic acid. It should come as no surprise that the more ascorbic acid you consume, the more of it your body will produce. Plasma has lower quantities than the adrenal glands, brain, and leukocytes, all of which have larger concentrations. There are many different foods that are rich in vitamin C, some of which include black currants, peppers, Brussels sprouts, and kiwifruit. The recommended daily intake for Sweden is 75 milligrams. The primary sources of vitamin C are fruits (93 mg), berries (80 mg), vegetables (60 mg), and beverages (60 mg) (80 mg) (Akbari *et al.* 2016).

### **3. MATERIALS AND METHODS**

#### **3.1 Study of Population**

Prior to the research, half of the participants had been diagnosed with hypertension, while the other half were healthy obese individuals. All participants were between the ages of 18 and 60, with a standard deviation of 34.2. The investigation was carried out by a team from Al-Kadhmia Teaching Hospital. From December 2021 to February 2022, an assessment of 30 healthy people in Baghdad discovered that the average age of the control group was 29.22 years old, with a standard deviation of 0.08. ( $29.22 \pm 12.83$ ). Exclusion criteria: obese with any associated disease other than hypertension were excluded.

#### **3.2 Samples Collection**

The serum was extracted by centrifugation at room temperature after the blood had been transferred to a gel tube. Ten minutes of centrifugation at 2500 g yielded the desired results. Afterwards, the serum was divided into three Eppendorf tubes and stored in a deep freezer (-20 C) until the time of analysis.

#### **3.3 Data Collection**

The kilos per square meter of body weight were used to determine BMI. Before getting their weight and height measured, patients were told to take off any bulky clothing or shoes. The weight was measured using a medical balance. BMI of 18.5-24.9 kg/m, 25.0-29.9 kg/m, and 30.0 kg/m<sup>2</sup> were considered normal weight, overweight, and obesity, respectively.

### 3.4 Blood Sampling and Processing

TOS, glucose, and lipid profile in serum can be determined using spectrophotometric methods and ELISA kits, whereas asporsin is tested using ELISA kits. As a control group, the study includes 60 healthy individuals between the ages of 18 and 60 who are not overweight or obese.

### 3.5 Resources and Equipment

The tools used in the study, as well as instruments are listed in Table 3.1, along with their provenance of manufacturing

**Table 3.1** Tools and instruments which were used in the study and their company of manufacture

NO.	TOOLS AND INSTRUMENTS	COMPANY / COUNTRY
1	Centrifuge	Beckman model TJ-6 /Germany
2	Deep freezer	Royalrahmany / China
3	Disposable tips	Eppendorf / Germany
4	Disposable syringe	Afco / Jordan
5	ELISA reader,	ELX800 BioTech / USA
6	Gel tubes	Afco / Jordan
7	Test tubes	Afco / Jordan
8	Spectrophotometer UV_VIS computer	Cecil, CE10N / England
9	Water bath	Memmert / Germany
10	Balance	Sartorius BL 210S

### 3.6 Kits

All kits used in the study are listed in Table 3.2 with their manufacturing companies

**Table 3.2** Kits used in the study and their companies

NO.	KITS	COMPANY / COUNTRY
1	Asporsin ELISA kit	HUMAN ELIZA /GERMENY

2	Glucose Kit	LINEAR /SPAIN
3	Triglycerides kit	LINEAR /SPAIN
4	Cholesterol kit	LINEAR /SPAIN
5	HDL-cholesterol kit	LINEAR /SPAIN
6	Vitamin C	BIOLABO / France

### 3.7 Methods

#### 3.7.1 Determination of total oxidant status

Erel method was used to determine total oxidant status value in the samples, as shown in Table 3.3.

**Table 3.3** Erel method used

SOLUTION (A)	SOLUTION (B)	SOLUTION (C)
(150 µM xylenol orange) (140 mM NaCl) (1.35M glycerol, pH 1.75)	(5 mM ferrous ammonium sulfate) (10 mM Odianisidine)	Standard Solution H <sub>2</sub> O <sub>2</sub> (200 µM)
After dissolving 114 mg of xylenol orange and 8.18 grams of sodium chloride in 900 mL of a solution of 25 mM H <sub>2</sub> SO <sub>4</sub> , the glycerol solution was added. This reagent can be stored in a refrigerator at 4°C for at least six months.	1g ferrous ammonium sulfate and 2g O-dianisidine were dispersed in 1000ml of 25M H <sub>2</sub> SO <sub>4</sub> solution. For at least six months, this reagent can be stored in a refrigerator at 4°C. Working with odianisidine necessitates the use of protective gear such as gloves and a mask because to the drug's potential for causing cancer.	Hydrogen peroxide (485104 M) was diluted (13 L) with distilled water to produce this solution (1 L).

### 3.8 Biochemical Parameters

The levels of Aspirin, serum lipid profile, Vitamin C, and glucose was determined for the complete research population.

### **3.8.1 Determination of serum asprosin hormone**

The quantitative serum asprosin was determined by ELISA (DRG diagnosis – Germany. cat.no E4095hu).

An anti-huma asprosin antibody was employed to cover the ELISA plate in this experiment. Asprosin is added to the sample, which binds to antibodies on the well. Because to the addition of biotinylated anti-asprosin antibody, it is able to detect the protein. The biotinylated is then bound by streptavidin-HRP, which is recognized by streptavidin.

### **3.8.2 Determination of vitamin C**

Utilizing the Vitamin C Assay Kit, one is able to determine the amount of vitamin C present in blood, plasma, food, drinks, and other types of biological materials. The K-ASSAY® Vitamin C Assay Reagent and Calibrator manufactured by the Kamiya Biomedical Company is now available for purchase at Cobo Scientific.

Introduction: Vitamin C, also known as L-ascorbic acid, is a coenzyme that is water-soluble and has a high reduction capacity. Collagen production in the human body cannot occur without vitamin C's presence. The two different chemical forms of vitamin C are referred to as ascorbic acid (AsA) and dehydroascorbic acid (DHAsA). An assay for vitamin C is a test that determines how much vitamin C is present in the body (AsA and DHAsA). The livers of humans do not create a type of vitamin C called a six-carbon lactone from glucose, as the livers of other animal species do. As a direct consequence of this, the human body is incapable of operating properly in the absence of ascorbate. Ascorbate functions as an electron donor in eight different enzymes that are found in humans. Because of its antioxidant properties, it may lower your risk of developing cancer, cardiovascular disease, and cataracts.

To determine how much vitamin C is present in a sample of blood or plasma, the K-ASSAY® Vitamin C Assay employs an enzymatic method. L-Ascorbate is oxidized by the enzyme ascorbate oxidase, which is a catalytic enzyme. This process results in the formation of hydrogen peroxide (vitamin C). Hydrogen peroxide, when combined with the appropriate chromogen, may be used to produce a blue dye with a high level of sensitivity. Whatever it is that vitamin C does, it doesn't seem to have any impact on this reagent at all. The K-Test® Vitamin C test may be performed on a clinical chemistry analyzer that uses two different reagents. The K-ASSAY® Vitamin C Calibrator and saline solution are used throughout the calibration process. Using this calibration, one is able to determine the amounts of vitamin C in both the serum and the plasma. Calibration Kit and Reagent for Accuracy (KT-75000) (KT-75000) range: 0.02 to 20 nanomoles for each test tube

### **3.8.3 Detection of cholesterol by linear kit**

The code for the product is MAK043. Cholesterol Quantitation Kit REACH was created by Sigma, which is the kit's producer. In mammalian cells, membrane cholesterol functions as a route for intracellular transport, cell signaling, and fluidity. Membrane cholesterol is a lipid. There is a possibility of free acid and cholesterol esters being present in the blood. In the treatment and prevention of atherosclerosis and cardiovascular disease, one of the most significant therapeutic targets is the reduction of cholesterol levels. As more information becomes available, there is a growing consensus that chronic inflammation is associated with an abnormal cholesterol homeostasis.

The Cholesterol Quantification Kit, available from Principle, may be used to ascertain the amount of free cholesterol and cholesteryl esters that are present in one's blood (total). The quantity of cholesterol in a sample is exactly proportional to the amount of enzyme that is present in the sample when using an enzyme test with a colorimetric or fluorometric product.

### **3.8.4 Detection of TG by linear kit**

Triglycerides MR Enzymatic colorimetric method Endpoint REF 1155005

Principle: The enzyme lipoprotein lipase in the blood breaks down the triglycerides into glycerol and free fatty acids (FFA) (LPL). It is possible to generate ADP in the presence of glycerolkinase (GK) due to the fact that ATP generates glycerol-3-phosphate (G-3-P) in the presence of G-3-P. (ADP). Both dihydroxyacetone phosphate (DHAP) and hydrogen peroxide are byproducts of the enzyme glycerophosphate oxidase (GPO). The color of the chromogen changes when the number of triglycerides in the sample increases because peroxidase may convert 4-Aminoantipyrine (4-AA), phenol, and H<sub>2</sub>O<sub>2</sub> into 4-Aminoantipyrine (4-AA).

### **3.8.5 Detection of LDL by linear kit**

LDL-Cholesterol differential precipitation Enzymatic colorimetric test Endpoint REF 1133105.

Principle: Using polyvinyl sulfate, LDL cholesterol is precipitated in whole serum and the remaining lipoproteins (VLDL+ HDL) in the clear supernatant are analyzed for traces of the disease-causing lipid. When total cholesterol is subtracted from the supernatant cholesterol, LDL-cholesterol is produced.

### **3.8.6 Detection of HDL by linear kit**

HDL-Cholesterol differential precipitation enzymatic colorimetric test Endpoint REF 1133010

Principle: Precipitation with phosphotungstic acid/MgCl<sub>2</sub> is followed by centrifugation and enzymatic examination of the clear supernatant as high-density lipoproteins (HDLs) in order to identify specific apolipoprotein B-containing lipoproteins (HDL).

### **3.8.7 VLDL– cholesterol estimation**

According to the hypothesis that VLDL exists in blood at a level equal to 0.2 of the Triglyceride content, the level of serum of LDL-C was calculated.

VLDL-C is equal to 0.2 glycerides (TG\5)

### **3.9 Statistical Analysis**

It was determined that the data had been normalized, comparable, and distributed normally. The likelihood was also assessed using IBM SPSS version 25.0 and a student independent T-test with median standard deviation. When the probability was less than 0.05, it was considered statistically significant.

## 4. RESULTS AND DISCUSSION

The present study is a case control included 120 obese and also its included 60 healthy controls. The mean age of obese was (34.2 ± 9.37) years. whereas that of controls was (29.22 ± 12.83) p value 0.038 as shown in Table 4.1.

### 4.1 Clinical Characteristics in Present Study

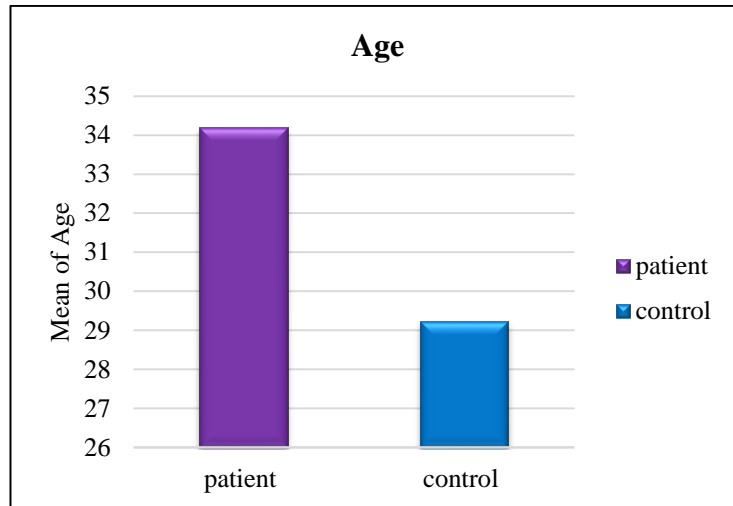
Table 4.1 illustrating the general characteristics of the study population according to age, weight and BMI. with obese cases and control groups.

In present study also showed that there is no significant difference in age between controls (29.22±12.83) and patients cases (34.2±9.37) p value >0.05, as shown in Figure 4.1. On the other hand, the results showed that the weight of controls (69.15.1±6.5) is a significant different with patients cases (81.6±11.96) p value = 0.011 but the height there no significant may be affect mean and SD of control 161.88±4.5 as compared with patient group (161.54± 7.49), p value >0.05, as shown in Figure 4.2, 4.3.

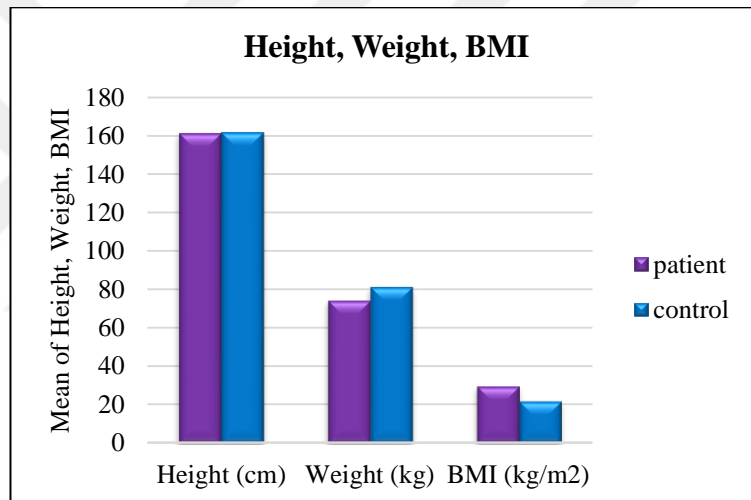
The results of BMI showed that controls (22.13±3.8) are significantly different from patients group cases (29.9±4.0) p value 0.01. Regarding smokers among family, 17 (34%) of controls and 33 (66%) of cases reported that they have a smoker among family and this difference between cases and controls was statistically significant p<0.05.

**Table 4.1** General characteristics of present study

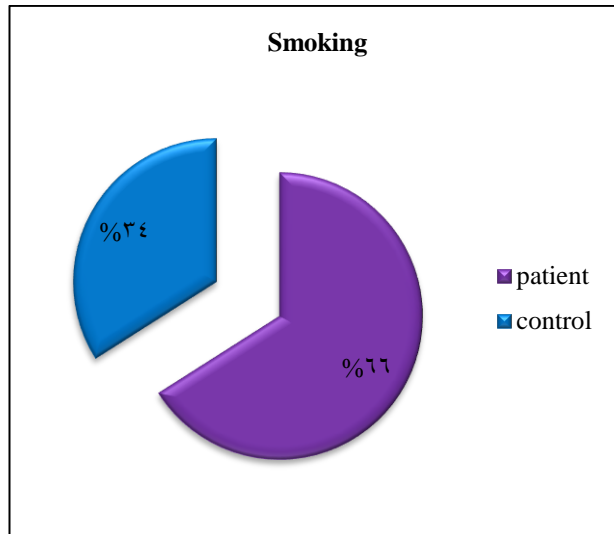
CLINICAL CHARACTERSTIC	MEAN ± SD		SIGNIFICANT VALUE
	Patients	Control	p-value
Age	34.2±9.37	29.22±12.83	0.38
Height (cm)	161.54± 7.49	161.88±4.5	0.70
Weight (kg)	74.6±12.46	(81.6±11.96)	0.01 *
BMI (kg/m <sup>2</sup> )	29.9±4.0	22.13±3.8	0.033*
Smoking	33 (66%)	17 (34%)	-
Gender	Male	Female	-
	44 %	56 %	-



**Figure 4.1** Mean of age, for patient and control groups



**Figure 4.2** Mean of weight, heghit and BMI for patient and control groups



**Figure 4.3** Frequency % of smoking in studied groups

According to the findings of the research, being obese is associated with having a higher BMI (Iseki 2006). Patients who are overweight or obese have an increased risk of developing metabolic syndrome and a high body mass index (BMI). Even in cases when obesity and metabolic syndrome are successfully managed, it is still possible to develop renal failure.

The findings of a research carried out in 2017 by Herrington and colleagues suggest that obesity is linked to OCD. As a direct result of their research, it has come to their attention that persons of all different types of health statuses, including those who already have diabetes, hypertension, or cardiovascular disease, are at a greater risk of having more advanced illnesses owing to their obesity (Herrington et al. 2017).

An issue that affects the whole world is obesity. In most nations, the likelihood of a man or woman getting overweight or obese is essentially identical to the likelihood of either gender becoming overweight or obese. When compared to women in more developed nations, women in less developed countries, particularly those in the Middle East and North Africa, are more likely to be overweight. Although men in affluent nations have a greater risk of becoming overweight, this is not always the case in other parts of the world. It's possible that socioeconomic variables are to blame for the gender gap in the prevalence of obesity among men and women. When a woman claims that she is trying

to eat healthier, it does not always indicate that she is increasing the amount of sugar that she consumes. As a consequence of a wide range of social and cultural variables, both men and women experience similar patterns of weight increase. Changes in the types of food available in many developing countries have had an impact on the amount of physical activity that women engage in, which has led to an increase in body mass index for both men and women. In many different cultures, pregnant women, healthy women, and wealthy women are more likely to be of a bigger frame. By analyzing the disparities in weight that exist between the sexes, we may be able to get a deeper comprehension of the pandemic of obesity that is sweeping the globe (Nissen 2017).

Both the usage of tobacco products and the prevalence of obesity are on the increase around the globe; both trends have a detrimental effect on people's health. In industrialized nations, smoking is a major contributor to the development of cancer, as well as other respiratory and cardiovascular diseases, including heart attack and stroke. Due to the elevated mortality rates linked with diabetes and IHD, obesity is now the fifth leading cause of death in the world. According to the findings of the Framingham Study, obese smokers have a life expectancy advantage of 13 years when compared to those who have never smoked (Li et al. 2020)

Less than one-third of those who smoke are successful in quitting the habit. Seventy-five to eighty percent of those who attempt to stop smoking are successful during the first six months of their efforts. One of the primary reasons why individuals are unable to break their smoking habit is addiction. One of the primary factors that contributes to smokers' resistance to giving up the habit is their concern about putting on weight. Many adolescents are very motivated to start smoking because they are under the impression that doing so would assist them in reducing their body fat percentage. (Fidler *et al.* 2011).

## **4.2 Oxidative Stress**

In the twenty-first century, obesity affects individuals of all nationalities, cultures, and socioeconomic backgrounds, regardless of the fact that they are living in the twenty-first

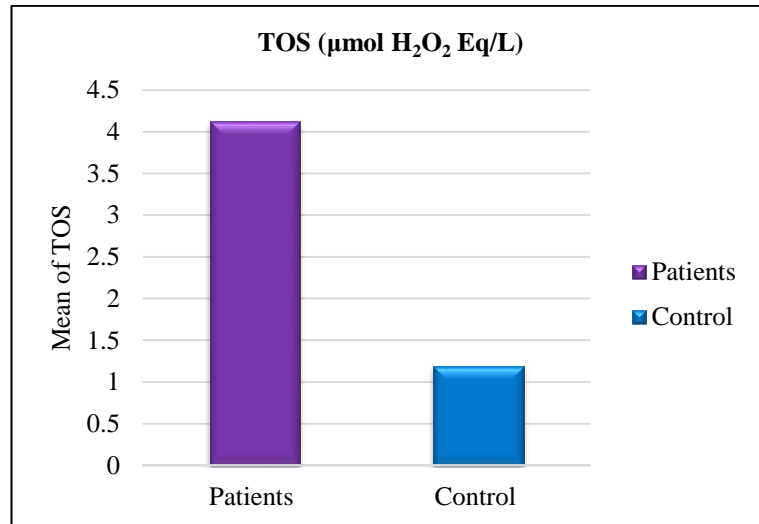
century. 1 Being fat not only cuts down on a person's expected lifetime but also increases the likelihood that they may die prematurely. In addition, this drives up the expense of receiving medical treatment. 2 Obesity is linked to a wide range of additional issues, in addition to the myriad of health issues it already causes. A wide variety of risk factors, including but not limited to asthma, sleeplessness, renal disease, hepatic dysfunction, and infertility, may all play a role in the development of type 2 diabetes. According to research that indicate an increase in preadipocyte proliferation, development, and mature adipocyte size in animal and cell cultures, there is some evidence that white adipose tissue (WAT) may be connected with obesity. This evidence comes in the form of a hypothesis. (Pollin *et al.* 2008).

#### 4.2.1 Total oxidant status

The level of total oxidant status has elevated significantly ( $P < 0.01$ ) in obese patients cases ( $4.12 \pm 0.51 \mu\text{mol H}_2\text{O}_2 \text{ Eq. /L}$ ) a compared to healthy control ( $1.19 \pm 0.22 \mu\text{mol H}_2\text{O}_2 \text{ Eq. /L}$ ). A significant higher level of TOS has observed obese patients compared control p value  $< 0.01$  (Table 4.2, Figure 4.4).

**Table 4.2** Mean and SD of TOS panale for patients cases with control

GROUPS	MEAN $\pm$ SD	P-VALUE
	TOS ( $\mu\text{mol H}_2\text{O}_2 \text{ Eq. /L}$ )	
Patients	$4.12 \pm 0.51$	0.001**
Control	$1.19 \pm 0.22$	
Significant comments	** : significant at $P \leq 0.01$	



**Figure 4.4** Mean of TOS for patients cases with control

Oxidative stress has been linked to the co-morbidities that are associated with obesity. According to current research, an increase in oxidative stress in humans is associated with obesity as a contributing factor. There are a number of factors that may contribute to the oxidative stress that is associated with obesity, including hyperleptinemia (a high amount of leptin in the blood), insulin resistance, increased muscular activity to promote weight gain, decreased endothelial function, and a poor diet. These are only some of the many variables that might contribute to the oxidative stress that is associated with obesity. MDA, 8-iso Prostaglandin F<sub>2</sub> (8-isoPGF<sub>2</sub>), and protein carbonylation are a few of the oxidative stress markers that may be detected in blood, serum, and urine. Other signs include hydroxyl radicals and lipid peroxidation. It has been shown that there is a connection between markers of oxidative stress and body mass index (BMI).

(Manna and colleagues) found that total antioxidant status (TAS) and ferric reducing antioxidant power (FRAP) were two of the most often used antioxidant neutralization capacity tests in plasma in 2015. (FRAP). According to the findings of a number of studies, people who are fat have decreased plasma levels of FRAP and TAS compared to controls who are not obese. There were also Vicente's coworkers in the crowd with them.

The oxidative stress that is brought on by obesity has been related to a number of different health problems, including cardiovascular disease, sleep difficulties, asthma, cancer, reproductive complications, and rheumatological concerns. In this research, we will investigate a wide range of variables that have a role in the oxidative stress that is associated with obesity. Oxidative stress, which will be researched as part of this study, is suspected of having a connection to obesity-related health problems. Attendees at Manna's birthday celebration included both his buddies and the people he works with. (Manna and Jain 2015).

#### 4.2.2 Relation ship between vitamin C as as antioxidant levels with obsity

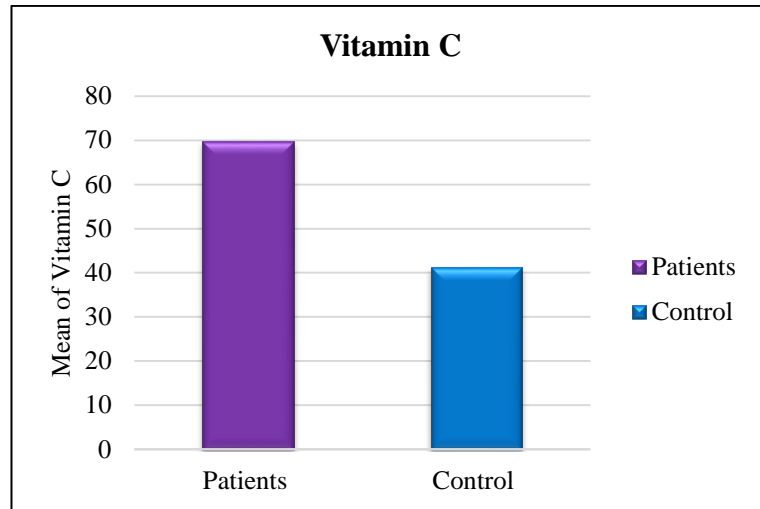
Vitamin C is a cofactor for a wide spectrum of biosynthetic and regulatory metalloenzymes, including enzymes that drive gene transcription and epigenetic changes.

Appropriate in vivo vitamin C concentrations are required for optimum kinetics of these vitamin C-dependent enzymes. When vitamin C levels are low, overweight or obese people are more susceptible to illness (Garcia 2017).

Tis result shown highly significant decrease of vitamin C as shown in Table 4.3 and Figure 4.5. Meas and SD for patients ( $69.85 \pm 11.691$ ) and control ( $41.30 \pm 4.88$ ) p-value  $< 0.01$ .

**Table 4.3** Mean and SD of vitamin C in patient and control groups

GROUPS	MEAN $\pm$ SD	P-VALUE
	Vitamin C	
Patients	$69.85 \pm 11.691$	0.0011**
Control	$41.30 \pm 4.88$	
Significant comments	**: significant at $P \leq 0.01$	



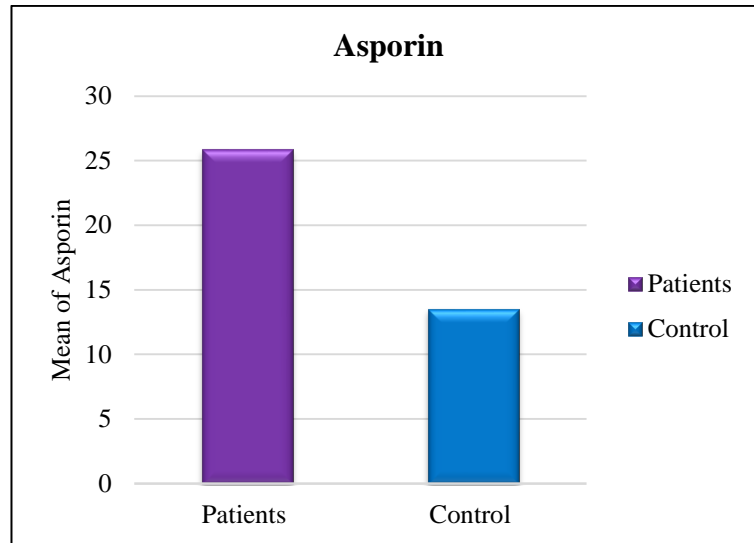
**Figure 4.5** Mean of vitamin C for patients cases with control

### 4.3 Relation Ship Between Asprosin With Obsity.

Table 4.4 and Figure 4.6, illustrate the relationship between patients and healthy control group in order to Asporin. This finding appered a significant diffrencer between patients and control groups in comparison between mean values of asporin for control, ( $25.879 \pm 1.4271$ ,  $13.516 \pm 1.8931$ ) respectively (p value  $< 0.01$ )

**Table 4.4** Mean and SD for asporin in stodied groups

GROUPS	MEAN $\pm$ SD	P-VALUE
	Asprosin	
Patients	$25.87 \pm 1.42$	0.0001
Control	$13.51 \pm 1.89$	
Difference	-8.363	
Pooled Standard Deviation	1.67637	
Standard error	0.30606	



**Figure 4.6** Mean for asporin levels in studied groups

There is an inverse correlation between insulin resistance and asporin levels, but not between insulin resistance and BMI or waist circumference (adiposity). Even after taking HOMA-IR into account, there was a statistically significant gap between the levels of asporin found in patients and those found in the controls. Despite the popular idea that insulin resistance plays a role in the development of NAFLD, it is possible that NAFLD is unaffected by IR. As opposed to what was expected, it was not able to establish a connection between asporin concentrations and fibrosis or other histological abnormalities in order to differentiate between SS and NASH. More clinical trials and mechanistic research are required in order to validate the results of this study and get a deeper comprehension of the role that asporin plays in NAFLD and IR. We shouldn't rush to any conclusions just now since this is the very first time that asporin has been connected to NAFLD. (Tahara *et al.* 2012).

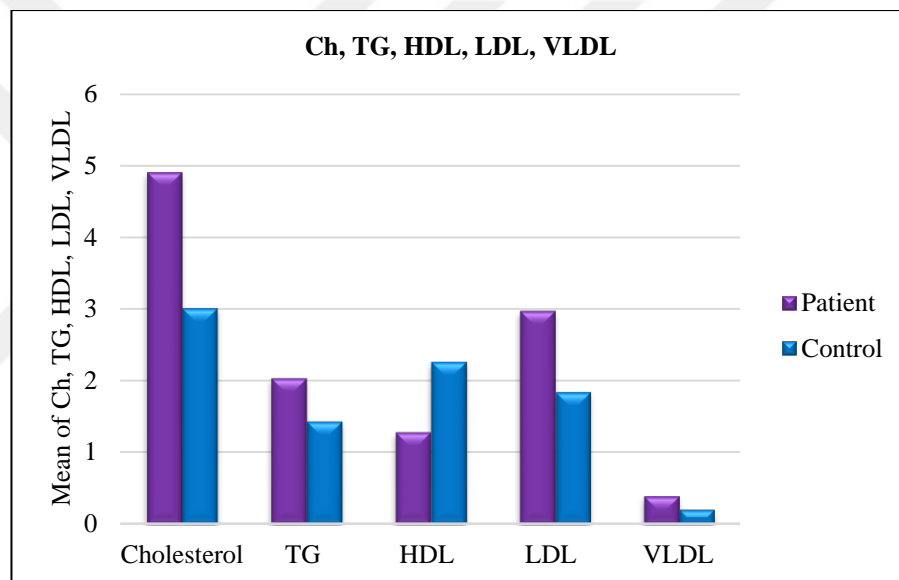
#### **4.4 Levels of Lipid Profile and Obesity and Control Groups.**

Table 4.5 and Figure 4.7, dyslipidemia, stroke, coronary heart disease, and other conditions can all be connected to obesity. A lipid metabolic abnormality known as dyslipidemia is typically present in patients with Type 2 Diabetes Mellitus (T2DM). Triglycerides and LDL were observed to grow in the lipid profile, HDL was demonstrated to diminish compared to the prior study's (HDL) (HDL). T2DM patients

in Indonesia have increased in recent decades, which has resulted in a twofold increase in death rates. In present study shown as insignificant correlation with lipid profile

**Table 4.5** Mean and SD of lipid profile in present studied

PARAMETERS	PATIENTS	CONTROL	P-VALUE
Cholesterol	4.910±0.791	3.0±0.47	0.01**
TG mmol/L	2.047±0.64	1.433±0.22	0.033*
HDL mmol/L	1.292± 0.27	2.264±0.19	0.01**
LDL mmol/L	2.982±0.53	1.845±0.88	0.01**
VLDL	0.407±0.13	0.21±0.099	0.01**



**Figure 4.7** Mean of lipid profile in patients and control groups

A correlational study was carried out to investigate the connection that exists between the lipid profiles of persons who suffer from type 2 diabetes and the degree to which they are obese. Levels of glucose and the risk of obesity (total cholesterol, HDL, LDL, and triglycerides) It has been shown that obesity is linked to increased levels of both total cholesterol and low density lipoprotein. [Citation needed] (LDL). Despite their little stature, they developed a strong friendship. There was a connection between HDL and triglyceride levels, although a tenuous one ( $r = -0.072$ ,  $p = 0.798$ ). This association was statistically significant. P-values that have a significance threshold of 0.05 or below are regarded as having some amount of relevance. There is no correlation between an

increase in triglyceride levels and a drop in HDL cholesterol and the existence of type 2 diabetes, despite the widespread perception to the contrary (Alaaraji et al. 2020).

The National Health and Nutrition Examination Survey (NHANES) was conducted between 1999 and 2006, during which time it was shown that there is a significant correlation between obesity and type 2 diabetes. According to the findings of this research, the BMI of an individual has a direct bearing on their levels of fasting blood glucose. Patients who have a higher body mass index (BMI) have a greater risk of becoming obese (BMI). There are a number of lifestyle factors that have been associated to the development of type 2 diabetes mellitus. These factors include being overweight or obese, overeating, not getting enough exercise, and adopting a more Westernized way of life (Kern et al. 2001). The digestive system is responsible for the breakdown of food into its component parts. Carbohydrates are responsible for the production of three different types of sugars: glucose, amino acids, and fatty acids. The nutrients that are broken down and absorbed in the intestines are used as a source of fuel by a variety of organs throughout the body. After digestion, the cells of food can only be turned into usable energy. Because it enables glucose to enter cells and be utilized as a source of fuel, insulin is an essential hormone for the metabolic process (Alobaidi et al. 2020). When there is an enough amount of insulin bound to the insulin receptors on the cell membrane, sugar may enter the cell and be utilized as fuel. Under more typical conditions, one would not anticipate this happening. As a consequence of this, sugar levels have returned to normal. Because of the high quantities of both mRNA and protein that it produces, LITAF (LIPOP-induced TNF-factor) has been connected to metabolic diseases such as obesity. (Al-Khafajy *et al.* 2021).

#### **4.5 Personal Correlation Between Asporin with Another Biochemical Marker**

Table 4.6 illustrated the personal correlation when compared Asporin and another lipid profile in present studeied groups. according to this result thers significant correlation between Asporin with cholestrole and LDL and HDL but no significany between TG and VLDL with Asporin level in obese patients.

**Table 4.6** Personal correlation between hepcidin hormone and iron panel in present study population

PARAMETERS	ASPORIN	
	Correlation	Significant value
Cholesterol	0.424	0.01*
TG mmol/L	0.2	0.10
HDL mmol/L	0.39	0.0197*
LDL mmol/L	0.56	0.006**
VLDL	0.25	0.0856

## **5 CONCLUSIONS AND RECOMMENDATION**

### **5.2 Conclusions**

1. In patients with asprosin level have a significant correlation in obese group as compared with control.
2. -When compared to the total oxidative status, this result shown a shgnificant increase of TOS in patients group and also have correlation with Asprosin level.
3. Vitamin C in present study have a significant correlation with obese group because its decrease with increasing of TOS.
4. . Asporin have positive correlation with cholestrol LDL and negative correlation with HDL concentrations.
5. Asporin which have been no correlation when compared with TG and VLDL in obese patients
6. Thers no significant appear when compared the obese cases with advanced age.
7. BMI have significant with increasing of obese chance
8. Smoking status have a significant study accompiend with obese patients

### **5.3 Recommendations**

1. Because a highly significant ration between Asprosin and another marker with obese, therefore i recomedbed usage a gentic study fo Asprosin.
2. Taken a specific marker suh assome immunological marker such as C3 C4

## REFERENCES

- Acara, A. Ç., Bolatkale, M., Kızıloğlu, İ., İbişoğlu, E. and Can, Ç. 2018. A novel biochemical marker for predicting the severity of ACS with unstable angina pectoris: asprosin.
- Akbari, E., Asemi, Z., Daneshvar Kakhaki, R., Bahmani, F., Kouchaki, E., Tamtaji, O. R. and Salami, M. 2016. Effect of probiotic supplementation on cognitive function and metabolic status in Alzheimer's disease: a randomized, double-blind and controlled trial. *Frontiers in aging neuroscience.*, 8: 256.
- Alaaraji, S. F., Awad, M. M. and Ismail, M. A. 2020. Study the Association of Asprosin and Dickkopf-3 with KIM-1, NTpro-BNP, GDF-15 and CPP among Male Iraqi with Chronic Kidney Disease. *Systematic Reviews in Pharmacy.*, 11: 10-17.
- Alan, M., Gurlek, B., Yilmaz, A., Aksit, M., Aslanipour, B., Gulhan, I. and Taner, C. E. 2019. Asprosin: a novel peptide hormone related to insulin resistance in women with polycystic ovary syndrome. *Gynecological Endocrinology.*, 35: 220-223.
- Al-Khafajy, D. A., Majeed, M. J., Al-Azzawi, O. F. and Khaleel, A. I. 2021. Asprosin Role for Obese Male Patients with Diabetic Mellitus Type II. *Prof.(Dr) RK Sharma.*, 21: 1345.
- Alobaidi, M. B. A. and Al-Samarrai, R. R. H. 2020. Correlation between Serum Asprosin Level And Oxidative Stress in Iraqi Patients with Type Ii Diabetes Mellitus. *Systematic Reviews in Pharmacy.*, 11: 1729-1733.
- Birben, E., Sahiner, U. M., Sackesen, C., Erzurum, S. and Kalayci, O. 2012. Oxidative stress and antioxidant defense. *World allergy organization journal.*, 5: 9-19.
- Elnagar, A., El-Belbasi, H. I., Rehan, I. F. and El-Dawy, K. 2018. Asprosin: a novel biomarker of type 2 diabetes mellitus. *Veterinary medicine in-between health & economy.*, 55: 333-347.
- Fidler, J. A. and West, R. 2011. Enjoyment of smoking and urges to smoke as predictors of attempts and success of attempts to stop smoking: a longitudinal study. *Drug and alcohol dependence.*, 115: 30-34.
- Foster, J. and Miller, R. S. 2012. A priori analysis of subgrid mass diffusion vectors in high pressure turbulent hydrogen/oxygen reacting shear layer flames. *Physics of Fluids.*, 24: 075114.

- Garcia, L. F. 2017. improving industrial performance of *chlamydomonas reinhardtii* through genetic engineering: a focus on stress tolerance and iron transport. *PloS*.
- Halliwell, B. 2014. Cell culture, oxidative stress, and antioxidants: avoiding pitfalls. *Biomedical journal.*, 37: 99.
- Herrington, W. G., Smith, M., Bankhead, C., Matsushita, K., Stevens, S., Holt, T. and Woodward, M. 2017. Body-mass index and risk of advanced chronic kidney disease: Prospective analyses from a primary care cohort of 1.4 million adults in England. *PloS one.*, 12: e0173515.
- Iseki, K. 2006. Body mass index and the risk of chronic renal failure: the Asian experience. In *Obesity and the Kidney*. Karger Publishers., 151: 42-56.
- Jung, E. H., Jeon, N. J., Park, E. Y., Moon, C. S., Shin, T. J., Yang, T. Y. and Seo, J. 2019. Efficient, stable and scalable perovskite solar cells using poly (3-hexylthiophene). *Nature.*, 567: 511-515.
- Ke, X., Duan, L., Gong, F., Zhang, Y., Deng, K., Yao, Y. and Zhu, H. 2020. Serum levels of asprosin, a novel adipokine, are significantly lowered in patients with acromegaly. *International Journal of Endocrinology.*, 2020.
- Kern, P. A., Ranganathan, S., Li, C., Wood, L. and Ranganathan, G. 2001. Adipose tissue tumor necrosis factor and interleukin-6 expression in human obesity and insulin resistance. *American journal of physiology-endocrinology and metabolism.*
- Kojta, I., Chacińska, M. and Błachnio-Zabielska, A. 2020. Obesity, bioactive lipids, and adipose tissue inflammation in insulin resistance. *Nutrients.*, 12: 1305.
- Li, H., Zhang, Z., Chen, L., Sun, X., Zhao, Y., Guo, Q. and Zhang, S. 2019. Cytoplasmic Asporin promotes cell migration by regulating TGF- $\beta$ /Smad2/3 pathway and indicates a poor prognosis in colorectal cancer. *Cell death and disease.*, 10: 1-14.
- Li, Y., Schoufour, J., Wang, D. D., Dhana, K., Pan, A., Liu, X. and Hu, F. B. 2020. Healthy lifestyle and life expectancy free of cancer, cardiovascular disease, and type 2 diabetes: prospective cohort study. *Bmj.*, 368.
- Manna, P. and Jain, S. K. 2015. Obesity, oxidative stress, adipose tissue dysfunction, and the associated health risks: causes and therapeutic strategies. *Metabolic syndrome and related disorders.*, 13: 423-444.

- Mishra, I., Duerrschmid, C., Ku, Z., He, Y., Xie, W., Silva, E. S. and Chopra, A. R. 2021. Asprosin-neutralizing antibodies as a treatment for metabolic syndrome. *Elife.*, 10: e63784.
- Nissen, T., Wayant, C., Wahlstrom, A., Sinnett, P., Fugate, C., Herrington, J. and Vassar, M. 2017. Methodological quality, completeness of reporting and use of systematic reviews as evidence in clinical practice guidelines for paediatric overweight and obesity. *Clinical obesity.*, 7: 34-45.
- Phaniendra, A., Jestadi, D. B. and Periyasamy, L. 2015. Free radicals: properties, sources, targets, and their implication in various diseases. *Indian journal of clinical biochemistry.*, 30: 11-26.
- Pollin, I. S., Kral, B. G., Shattuck, T., Sadler, M. D., Boyle, J. R., McKillop, L. and Michos, E. D. 2008. High prevalence of cardiometabolic risk factors in women considered low risk by traditional risk assessment. *Journal of Women's Health.*, 17: 947-953.
- Ramnanan, S. A. 2021. *The Power of a Sound Mind: Exploring Meditation and Sound Therapies for Treating the Emotional Impact of Tinnitus.*
- Romere, C., Duerrschmid, C., Bournat, J., Constable, P., Jain, M., Xia, F. and Chopra, A. R. 2016. Asprosin, a fasting-induced glucogenic protein hormone. *Cell.*, 165: 566-579.
- Sasaki, A., Nagatake, T., Egami, R., Gu, G., Takigawa, I., Ikeda, W. and Fujita, Y. 2018. Obesity suppresses cell-competition-mediated apical elimination of RasV12-transformed cells from epithelial tissues. *Cell reports.*, 23: 974-982.
- Tahara, N., Yamagishi, S. I., Matsui, T., Takeuchi, M., Nitta, Y., Kodama, N. and Imaizumi, T. 2012. Serum levels of advanced glycation end products (AGEs) are independent correlates of insulin resistance in nondiabetic subjects. *Cardiovascular therapeutics.*, 30: 42-48.
- Ugur, K. and Aydin, S. 2019. Saliva and blood asprosin hormone concentration associated with obesity. *International journal of endocrinology.*, 2019.
- Valko, M., Leibfritz, D., Moncol, J., Cronin, M. T., Mazur, M. and Telser, J. 2007. Free radicals and antioxidants in normal physiological functions and human disease. *The international journal of biochemistry and cell biology.*, 39: 44-84.

- Wang, G., Wei, Y., Qiao, S., Lin, P. and Chen, Y. 2018. Generalized inverses: theory and computations. Singapore. Springer., 53.
- Wang, S., Liu, C., Sun, Z., Yan, P., Liang, H., Huang, K. and Tian, J. 2017. IL-1 $\beta$  increases asporin expression via the NF- $\kappa$ B p65 pathway in nucleus pulposus cells during intervertebral disc degeneration. Scientific reports., 7: 1-13.
- Willcox, J. K., Ash, S. L. and Catignani, G. L. 2004. Antioxidants and prevention of chronic disease. Critical reviews in food science and nutrition., 44: 275-295.



## **CURRICULUM VITAE**

### **Personal Information**

Name and Surname : Ali Thaer Mohammed ALSAJRI

### **Education**

MSc Çankırı Karatekin University  
Graduate School of Natural and Applied Sciences 2020-2022  
Department of Chemistry

Undergraduate University of Tikrit  
College of Education for Pure Sciences 2015-2019  
Department of Chemistry