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ارتباط هرمون أسبروسين وبعض العناصر المعدنية في المرضى الذين يعانون من السمنة في مدينة بغداد

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خلاصة :

في السنوات الأخيرة ، أصبحت السمنة قضية متزايدة باستمرار يتعين على أنظمة الرعاية الصحية التعامل معها ، بسبب الآثار الجسدية الضارة المرتبطة بالسمنة مثل ارتفاع ضغط الدم ، وأمراض التمثيل الغذائي ، وأمراض القلب ، ومرض السكري من النوع 2 ، بالإضافة إلى زيادة خطر فيروس كورونا ، بالإضافة إلى ذلك ، يُعتقد أن السمنة عاملة مساهمة في كل من الخلل الجنسي والسرطان. تشير التقديرات إلى أن أكثر من ملياري شخص في جميع أنحاء العالم يعانون من السمنة أو زيادة الوزن ، مما يجعلها واحدة من الأسباب الرئيسية للوفاة في العالم ، حيث تمثل أكثر من 3.4 مليون حالة وفاة سنويًا. تعد المسارات الأساسية غير المعروفة العقبة الأساسية أمام تقدم أبحاث السمنة ، مما يحد من فهمنا لأصل المرض وإنشاء علاجات فعالة. بعض الظروف الأيضية التي تسببها السمنة لها تأثير على الخلايا والأنسجة المختلفة ، والأسبوسين هو هرمون تم تحديده حديثًا الناجم عن الصيام على الرغم من وعده كهدف حيوي أو هدف علاجي لأمراض التمثيل الغذائي ، لا تزال هناك تحديات تتعلق باكتشاف مستوياتها في البيئات السريرية والبحث المستمر في هذا الهرمون الجديد ، قد يفتح آفاق جديدة للعلاج الفعال لمتلازمات التمثيل الغذائي. في هذه الدراسة ، تم تقدير مستوى هرمون أسبروسين وبعض العناصر المعدنية (الزنك ، السيلينيوم ، المغنيسيوم) في مصل الدم للمرضى الذين يعانون من السمنة المفرطة. . كشفت النتائج أن مستويات المرضى من هرمون أسبروسين كانت أعلى بكثير من تلك الموجودة في المجموعة الضابطة ، وأن تركيز العناصر المعدنية كان أقل بكثير في المرضى الذين يعانون من السمنة المفرطة. أظهرت نتائج ارتباط هرمون أسبروسين مع الزنك ارتباطًا إيجابيًا ، في حين أن النتائج لم تظهر أي ارتباط بين أسبروسين والسيلينيوم وبين أسبروسين والمغنيسيوم على مستوى الاحتمال 0. 05> P.

الكلمات المفتاحية: أسبروسين ، الزنك ، السيلينيوم ، المغنيسيوم

Correlation Of Asprosin Hormone And Some Mineral Elements In Patients With Obesity In Baghdad City

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Abstract :

In recent years, obesity has become an ever-increasing issue that healthcare systems have to deal with, due to the adverse physical effects associated with obesity such as high blood pressure, metabolic diseases, heart disease, and type 2 diabetes, in addition to the increased risk of coronavirus, Additionally, obesity is thought to be a contributing factor to both sexual dysfunction and cancer. It is

estimated that over 2 billion people worldwide suffer from obesity or overweight, making it one of the world's leading causes of death, accounting for over 3.4 million deaths annually. Unknown underlying pathways are the primary obstacle to obesity research advancement, limiting our comprehension of the disease's origin and the creation of effective treatments. Certain metabolic conditions brought on by obesity have an impact on various cells and tissues, and Asprosin is a newly identified fasting-induced hormone Despite its promise as a biomarker or therapeutic target for metabolic diseases, there are still challenges related to the detection of its levels in clinical settings and continued research into this novel hormone may open new horizons for the effective treatment of metabolic syndromes. In the present study, the level of Asprosin hormone and some mineral elements (zinc, selenium, magnesium) were estimated in the blood serum of obese patients. . The findings revealed that patients' levels of the hormone asprosin were much higher than those of the control group, and that the concentration of mineral elements was significantly lower in obese patients. The results of the correlations of Asprosin hormone with zinc showed a positive correlation, while the results didn't show any correlation between Asprosin and selenium and between Asprosin and magnesium at the probability level P<0. 05.

Keywords: asprosin , zinc , selenium ,magnesium

1.Introduction:

Obesity is one of the most prevalent health issues affecting children, adolescents and the elderly and is a complex disease caused by genetic and environmental factors. Obesity develops from a combination of low energy intake and excessive calorie intake [1] In recent years, obesity has become an ever-increasing issue that healthcare systems have to deal with. The World Health Organisation (WHO) has stated that obesity is now an epidemic and therefore a threat to individual health for a large population in many continents.[2] The adverse physical effects associated with obesity include hypertension, metabolic diseases, cardiovascular diseases, kidney diseases, strokes and type 2 diabetes mellitus [3]. The adverse physical effects associated with obesity include hypertension, metabolic diseases, cardiovascular diseases, kidney diseases, strokes, and type 2 diabetes,[3] as well as an increased risk of COVID-19 infection in obese populations,[4] and obesity is associated with infectious diseases, as some infections may lead to an increase in obesity, such as human adenovirus 36, H1N1/ influenza, HIV, and Helicobacter pylori [5], Obesity is linked to cancer, the second greatest cause of mortality globally after cardiovascular disease, and is also a known risk factor for sexual dysfunction in both sexes. With an annual incidence of over 3.4 million deaths, obesity and overweight are among the main causes of mortality globally, affecting an estimated 2 billion people.[6]. 4 million deaths [6]. Since obesity creates distinct

biochemical environments that impact various cells and tissues, one of the biggest obstacles to the advancement of obesity research is the lack of knowledge about the underlying pathways that restrict our comprehension of the disease's genesis and the creation of treatments [7]. Obese people typically have higher levels of white adipose tissue (WAT), and research has shown that this tissue not only stores triacylglycerol but also functions as an endocrine system, releasing a variety of chemical messengers that affect and communicate with other tissues. Adipocyte-derived peptide is linked to numerous regulatory actions on energy balance[8], as well as the regulation of inflammatory processes [9].

Asprosin is a newly identified fasting-induced hormone that has received considerable attention in the field of scientific research since its discovery and first described by Romer et al. in 2016. Despite its promise as a biomarker or therapeutic target for metabolic diseases, there are still challenges related to the detection of its levels in clinical settings and continued research into this novel hormone may open new avenues for effective treatment of metabolic syndromes [10]. Asprosin is a 140-amino acid glucogenic protein that is synthesised mainly in white adipose tissue during fasting, although it can also be produced by other tissues such as the lungs, heart and pancreatic beta cells under certain conditions, and plays a crucial role in the regulation of metabolism. It plays a critical role in the regulation of metabolism and energy and plays a vital role in the regulation of appetite, and has emerged as a key factor in the body's response during periods of fasting. Aspartate aminotransferase is polarized by the liver after entering the circulation, where it adheres to the surface of liver cells and increases blood sugar levels—a critical component of brain survival and function[11]. As a recently identified adipokine hormone, asprosin seems promising for further investigation. Through a progressive rise in appetite, this fasting-induced hormone controls food intake and energy supply. Its level is correlated with the amount of adipose tissue, which serves as its primary source [12]. A recent addition to the new subclass of protein hormones known as codamines is adipokine aspirin. In contrast to conventional protein hormones and peptides, this subclass is synthesized by the protease Furin, which cuts the C-terminus of the parent protein, proviprilin-1, to produce aspirin. This cut resulted in the 320 kDa glycoprotein fibrillin-1 and the novel peptide hormone aspirin. Aspirin is a protein around 30 kDa long in mammals [13]. White adipose tissue has the greatest mRNA expression of FBN1, indicating that it is the primary source of asprosin. Numerous research studies have indicated that physical exercise may influence the amount of asprosin, a protein that plays a regulatory role in the browning of primary adipocytes.[14] Zinc is the second most prevalent mineral in living things after iron, found throughout the body, particularly in skeletal muscle (~60%) and bone (~30%).

After iron, zinc is the second most prevalent mineral in living things. Although zinc is present throughout the body, it is particularly abundant in bone (~30%) and skeletal muscle (~60%). Zinc is essential for brain signal transmission, cell autolysis, differentiation, and proliferation. Since many proteins need zinc to function, it is a structural element of thousands of protein domains and a catalytic cofactor for 300 enzymes of all types [15].Numerous studies on both humans and animals demonstrate the critical functions that selenium plays in preventing chronic metabolic disorders including obesity and type 2 diabetes. Inhibiting the inflammatory response, boosting immunity, and reducing oxidative stress are the major ways that selenium has positive benefits. Probiotics that are high in selenium may be a good option for treating type 2 diabetes and obesity [16]. As a cofactor in enzymatic pathways, magnesium is essential for many metabolic processes. Hypomagnesemia has been observed in approximately 30% of diabetic patients, and studies have demonstrated that increased magnesium intake enhances insulin secretion and sensitivity. For these reasons, magnesium supplementation may be a clinical strategy to improve type 2 diabetes[17].

2.Experimental part:

In the present study, 60 blood samples were collected from obese subjects and 60 blood samples were collected from healthy subjects who were considered as a control group from Baghdad governorate. 5mL of venous blood was drawn and placed in gel tubes and the samples were left at room temperature and then centrifuged at 3000r.p.m to obtain serum which was stored in Eppendorf tubes at -20oC until the studied chemistry measurements were performed.

2.1 Assay of Asprosin hormone level:

The Sandwich-ELISA principle was used to an ELISA kit. An antibody designed specifically for the human ASPN test was applied to the ELISA plate beforehand. The ELISA plate's pits are filled with samples (or standard solutions), which react with the antibody. Each well of the plate is filled with a horseradish peroxidase (HRP)-conjugated antibody and a human ASPN-specific detection antibody, which are then placed in the incubator. Clean the components that have been unbound. Fill each pit with the substrate solution. Only those pits containing the human ASPN detection antibody, biotinylated detection antibody and Avidin-HRP conjugate will appear blue When the stop solution is added, the enzyme-ligand reaction is stopped, and yellow becomes the new color. Optical density (OD) is measured using spectrophotometry at a wavelength of 450 nm. The OD value corresponds to the human ASPN level. The quantity of human ASPN in the samples may be ascertained by comparing their optical density to the reference curve.

2.2 Estimation of zinc concentration:

The kit was used for the determination of zinc based on the reaction of zinc in the sample with 5-Br-PADAP to produce a colour complex, the depth of colour being directly proportional to the zinc ion concentration. The zinc ion concentration is measured at a wavelength of 560 nm.

2.3Determination of selenium concentration:

An off-the-shelf kit based on the determination of selenium in a biological sample by its ability to catalyse the oxidation of phenylhydrazine to azo ion by potassium chlorate was used, where the colour intensity measured at 520 nm is directly proportional to the concentration of selenium in the sample.

2.4 Determination of magnesium concentration:

A ready-made kit based on the reaction of magnesium in the sample with the complex reagent (Calmagite) was used to form a calmagite-Mg complex whose absorbance is measured at 540nm and the concentration of magnesium is directly proportional to the absorbance of the complex.

3. Results and discussion:

3.1 Asprosin hormone level

The results of the current study showed that there was a significant increase in the level of asprosin hormone in obese patients at a probability level of P<0.0001 compared to the control group, as the level of asprosin hormone in patients reached 4.45±0.82ng/mL while the control group reached 2.63±0.47ng/mL as shown in Figure (1).

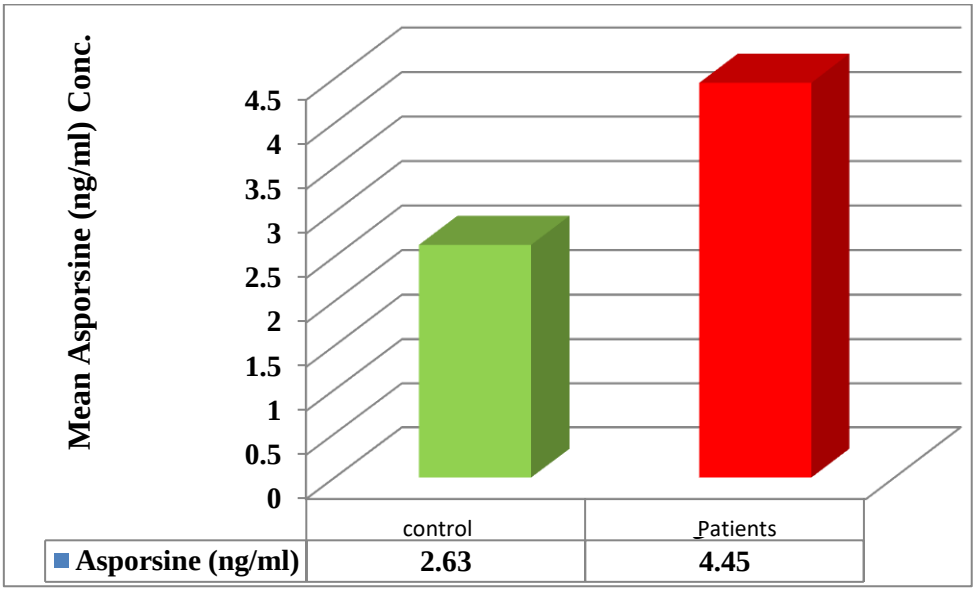

Figure (1) The level of asprosin hormone in obese patients compared to the control group

Some researchers found higher concentration of asprosin in obese subjects compared to normal weight control group which is consistent with the results obtained in our current study. Studies have also reported a positive correlation between asprosin and various obesity-related markers, including BMI and insulin resistance and asprosin has been proposed as a biomarker of obesity and metabolic disorders and a therapeutic target for obesity [18, 19].

Asprosin is secreted from adipose tissue and can modulate appetite. Asprosin secretion is influenced by a variety of conditions such as BMI, glucose, insulin and high-density lipoprotein (HDL), all of which are associated with obesity [19]. Aspirin has also shown a negative correlation with ALT which may be due to hyperphagia and increased gluconeogenesis in the liver suggesting that the liver may be a vital organ in the regulation of aspirin [20]. Plasma asprosin levels are also increased in insulin resistant humans as individuals with insulin resistance feel hungry quickly and experience delayed satiety [21]. Asprosin enhances food intake because it stimulates agRP peptide neurons through cAMP, which in turn promotes appetite [22]. The main source of asprosin's nanomolar levels in the bloodstream is white adipose tissue, which rises with obesity. Asprosin can pass through the blood-brain barrier and activate AgRP neurons via cAMP, increasing appetite, food intake, and absorption. As obesity increases, energy is transformed into fat mass.[23]. This mechanism is supported by the extremely high asprosin levels in our present investigation. Many related disorders, such as cardiovascular disease, may develop in this manner depending on the metabolic consequences of obesity. Aspirin may be the source of the increased hepatic glucose production since asprosin promotes appetite and induces the liver to release glucose [24]

3.2 Zinc level in obese patients and control group

The results of the current study showed a significant decrease in zinc concentration in obese patients compared to the control group at a probability level of $P<0.0001$, where the zinc concentration in obese patients was 0.32±0.06m.mol/L, while in the control group it was 0.54±0.06m.mol/L as shown in Figure (2).

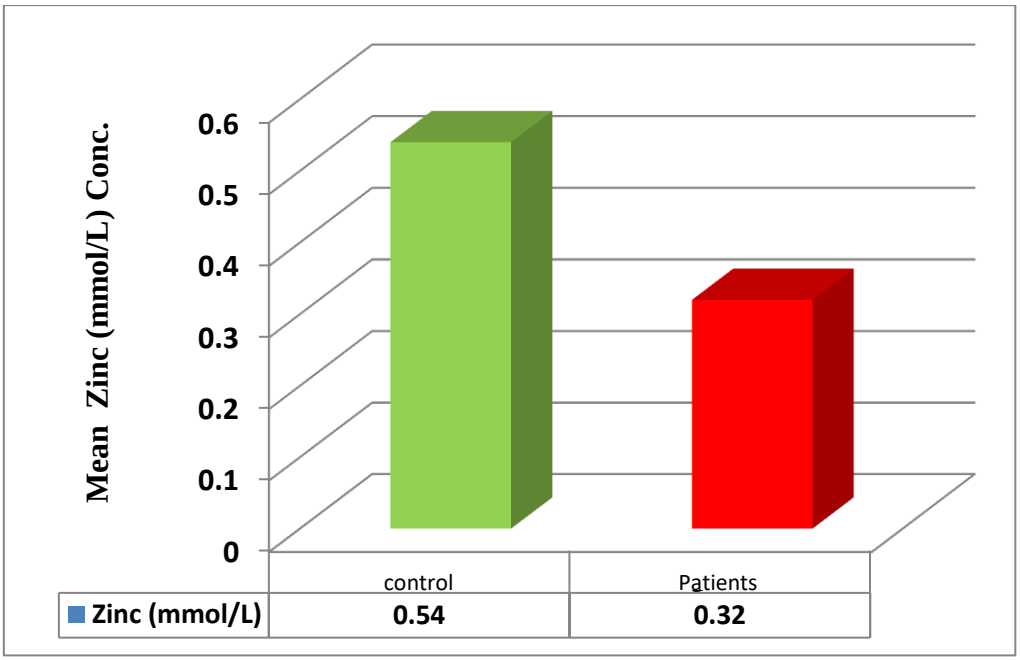

Figure (2) Zinc concentration in obese subjects compared to the control group

The present study's findings are in line with earlier research that discovered that obese adults [26] and children and adolescents [25] had lower zinc concentrations than the control group. Since zinc and TNF have a strong negative correlation, the effect of the inflammatory process on zinc metabolism may be the cause of the decrease in zinc concentration in obese patients. A prior study found a negative correlation between average serum zinc concentration and waist circumference and BMI [27]. These results are linked to the buildup of adipose tissue and the increase in the production of adipocytokines and cortisol, which causes chronic inflammation, which in turn encourages the liver's zinc buildup. The negative relationship between blood zinc levels and BMI in obese people may have been caused by zinc buildup in the liver and adipocytes[28], While Pelletat et al. [30] discovered that taking zinc supplements for four weeks had no positive impact on lipid levels in obese women with normal glucose tolerance. However, another study discovered that zinc supplements improved lipid levels in patients with diabetes and metabolic syndrome by raising HDL cholesterol and lowering glucose in patients with type 2 diabetes. On the other hand, a different study discovered that taking regular mg20 of zinc every day for eight weeks dramatically decreased mean fasting blood glucose and insulin without affecting LDL cholesterol, BMI, or waist circumference[31]

3.3 Selenium level in obese patients and control group

The results of the current study showed a significant decrease in the level of selenium in obese patients compared to healthy people at a probability level of $P<0.0001$, where the average selenium concentration in patients was 8.80mM±67.45, while the selenium level in the control group was 6.74mM±109.8 as shown in Figure (3).

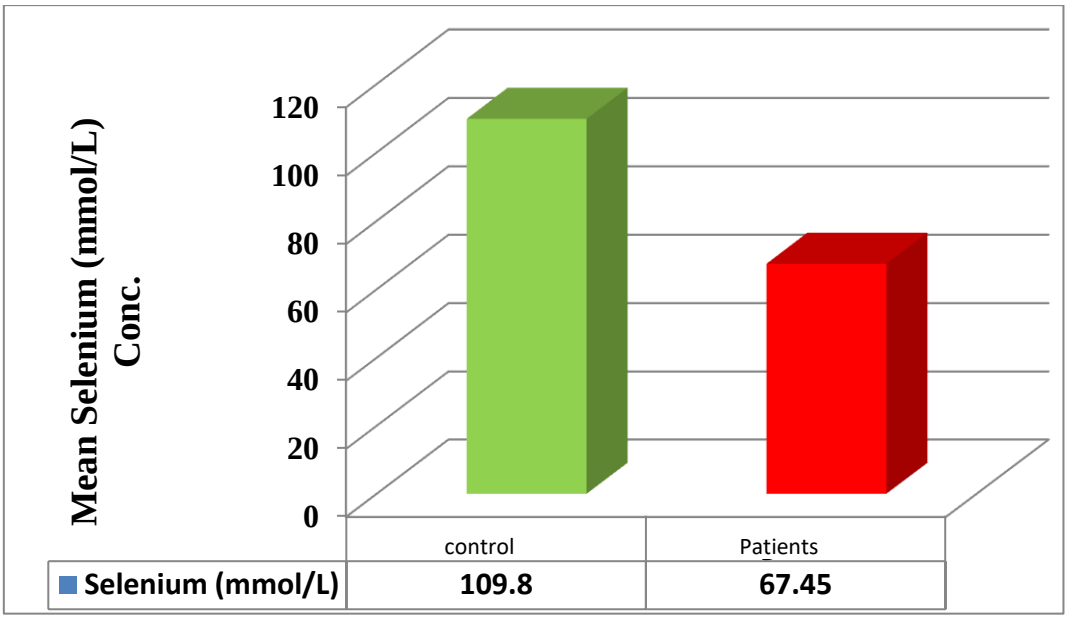

Figure 3: Selenium concentration in obese patients compared to the control group

Studies have shown that obese people have lower serum selenium levels than normal weight people, due to the increased oxidative stress associated with obesity, leading to greater consumption of antioxidants such as selenium, as obesity is associated with increased oxidative stress and chronic inflammation [32, 33], and several studies have confirmed a negative relationship between serum Se concentration and body mass index in obese patients, with fat mass being higher than the control group. [34] that Se supplementation has a positive effect on body mass formation, which is indicated by the association between dietary Se intake and lower leptin levels in patients treated with Se. Physiological leptin signalling is known to be essential for the maintenance of body weight [35] and Wang et al. found that when mice received high doses of Se, their body weight was significantly decreased, and the ratio of adipose tissue to body weight decreased as well[36]. It is necessary for the growth and function of adipose tissue to have selenium and selenoproteins, namely selenoproteins. Given the unique roles of selenoproteins, the mechanisms underlying their effects on adipose tissue may primarily involve the modulation of redox balance and endoplasmic reticulum stress. Selenium also controls the proliferation, differentiation, and maturation of adipocyte precursors as well as cellular functions (lipid accumulation and lipolysis). Defects in selenoprotein synthesis may result in adipocyte malfunction, which can cause a number of illnesses, including the emergence of obesity, because these pathways are connected to the hypothalamic action of selenium[37].

3.4 Magnesium level in obese patients and control group

The results showed a significant decrease in magnesium concentration in obese patients at the probability level P<0.0001, where the magnesium concentration in patients was 0.37±0.08m.mol/L, while the magnesium concentration in healthy people was 0.75±0.17m.mol/L as shown in Figure (4).

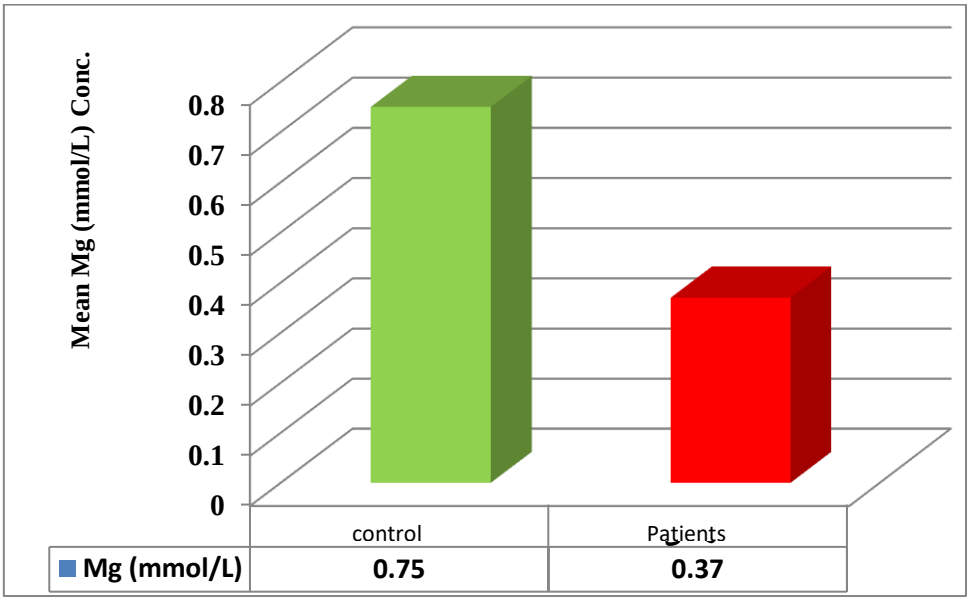

Figure (4) Magnesium concentration in the serum of obese patients compared to the control group

The current study's findings are in line with those of other research that found a negative correlation between magnesium levels and obesity [38, 39]. Other research revealed that obese subjects had lower serum magnesium levels than controls, and that there was a significant inverse relationship between magnesium and C-reactive protein levels. Similarly, lower serum magnesium was linked to higher levels of TNF-alpha protein in obese subjects. Other research has also demonstrated that serum magnesium protects against metabolic diseases, including obesity [40–42]. According to one research, those with normal serum magnesium had a considerably lower BMI than those with hypomagnesaemia [43]. In diabetic individuals, Babiker et al. [44] likewise found a significant inverse relationship between serum magnesium and BMI. Given the strong correlation between metabolic diseases and obesity, the substantial correlation between serum magnesium levels and obesity raises the possibility that serum magnesium regulates metabolism [45]. Additionally, Zachuk et al. found a significant negative correlation between serum magnesium levels and the severity of childhood obesity: serum magnesium levels declined significantly as obesity increased [46]. severity of childhood obesity [46], in another study a negative correlation was found between magnesium intake and BMI so studies have confirmed that higher serum magnesium levels have a significant positive effect on reducing obesity [47], Additionally, it has been shown that there is a negative correlation between blood CRP and magnesium in

obese individuals; this means that a significant portion of the impact of magnesium on obesity is due to its effects on Consequently, serum magnesium's preventive function against obesity stems in part from its anti-inflammatory properties, and it has been demonstrated that CRP, a recognized indicator of inflammation, is substantially linked to obesity [48]. By preventing the generation of reactive oxygen species, studies have also demonstrated the antioxidant and anti-inflammatory properties of magnesium. As a result, serum magnesium may be crucial for preserving insulin function and preventing the onset and progression of obesity [49]. Numerous studies have shown that magnesium ions, which act as cofactors for enzymes like tyrosine kinase and glucokinase, are essential for regulating insulin secretion and function as well as glycolysis. Therefore, in order to avoid the development of obesity, increased serum magnesium levels may have an effect on the control of energy metabolism, namely glucose metabolism[50].

3.5 Correlation between Asprosin and the studied chemistry variables

The results of our current study as shown in Table 1 showed a positive correlation between asprosin and zinc (r=0.356) Figure (5), which is consistent with a previous study that showed a positive correlation between asprosin and zinc [51]. Previous studies do not provide conclusive evidence of the direct effect of zinc supplementation on asprosin levels, but it is known that zinc plays vital roles in insulin signalling and glucose metabolism, which may indirectly affect asprosin. Thus, changes in zinc levels may have downstream effects on asprosin. While the study did not find any correlation of Asprosin with magnesium and selenium

Table (1) Correlation between Asprosin and the studied chemistry variables

Test	r	P-value
Selenium	-0.015	.9090
Mg	.0100	.9390
Zinc	.3560	.0050

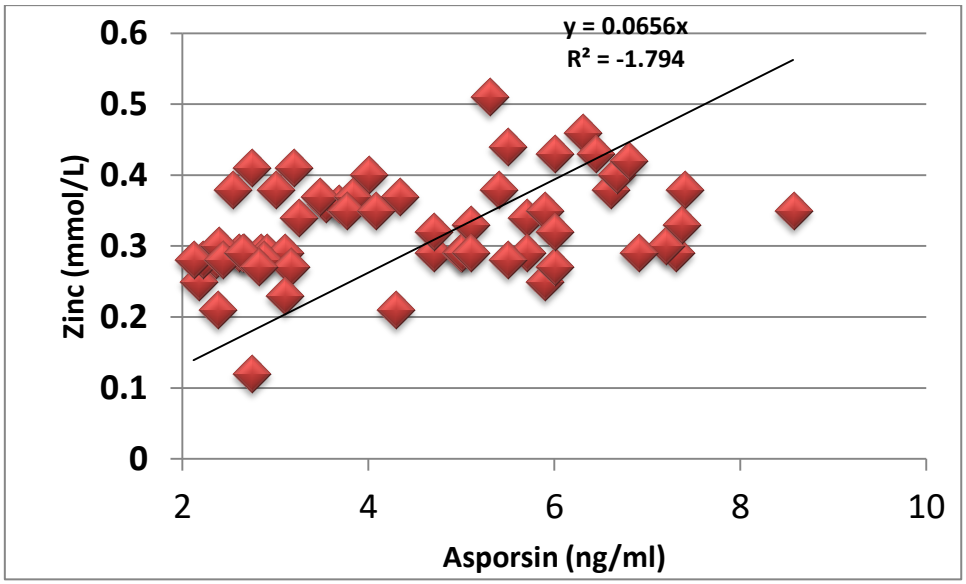

Figure (5) Correlation between asprosin and zinc

4.Conclusion:

Due to the adverse physical effects associated with obesity such as metabolic diseases, the main challenge to progress in obesity research is the unknown underlying pathways that limit our understanding of the etiology of the disease and the development of therapies. In the present study, the level of the newly identified hormone asprosin was estimated, which holds promise as a therapeutic target for metabolic diseases and continued research into this new hormone may open new avenues for the effective treatment of metabolic syndromes, in addition to the estimation of some mineral elements (zinc, selenium, magnesium) in the serum of patients with obesity. In addition to the estimation of some mineral elements (zinc, selenium, magnesium) in the blood serum of obese patients, the results showed a significant increase in the level of asprosin in patients compared to the control group, as asprosin is secreted at nanomolar levels in the blood circulation mainly by white adipose tissue, which increases with obesity, which leads to the activation of aspartate aminotransferase. This leads to the activation of AgRP neurons through cAMP to increase appetite, increasing food intake, which leads to increased absorption, converting energy into fat mass as obesity continues to increase in this cycle. The findings also revealed a notable drop in the estimated concentration of mineral elements in obese patients; the impact of the inflammatory process on the metabolism of zinc in obese patients may be the cause of the decline in zinc concentration in obese patients. Furthermore, compared to people of normal weight, obese individuals have lower blood levels of selenium because of the increased oxidative stress that comes with being obese. This causes them to consume more antioxidants, including selenium, because obesity is linked to

increased oxidative stress and chronic inflammation. Additionally, because obesity is closely linked to metabolic diseases, high blood levels of magnesium may be crucial for controlling metabolism. suggests that serum magnesium may play an important role in the regulation of metabolism. Furthermore, the results of the correlations of asprosin with zinc showed a positive correlation, as zinc plays vital roles in insulin signalling and glucose metabolism, which may indirectly affect asprosin, while the results did not show any correlation between asprosin and selenium and between asprosin and magnesium, while the results showed no correlation between asprosin and selenium.

5.References:

1. Oussaada, S.M., et al., *The pathogenesis of obesity.* Metabolism, 2019. **92**: p. 26-36.
2. Vries, J.d., *The association of obesity and quality of life in the Dutch province Flevoland in 2016*. 2021.
3. Abd Al-Subaihawi, R.M., S.A. Al-Qaysi, and A.J. Mahrath, *A Biochemical Study of Angiotensin II, Electrolytes and Vitamin D in Obese Patients.* International journal of health sciences. **6**(S6): p. 4471-4480.
4. Petrakis, D., et al., *Obesity-a risk factor for increased COVID-19 prevalence, severity and lethality.* Molecular medicine reports, 2020. **22**(1): p. 9-19.
5. Ahmed, H.O. and A. Muhialdeen, *Link of Helicobacter Pylori (H. pylori) to Overweight Patients and Patients with Obesity in two Obesity Centers-Sulaimani Governorate-Kurdistan-Iraq, Prospective Case Study Series.* Technium BioChemMed, 2021. **2**(3): p. 25-34.
6. Mohajan, D. and H.K. Mohajan, *Obesity and its related diseases: a new escalating alarming in global health.* Journal of Innovations in Medical Research, 2023. **2**(3): p. 12-23.
7. Lustig, R.H., et al., *Obesity I: Overview and molecular and biochemical mechanisms.* Biochemical pharmacology, 2022. **199**: p. 115012.
8. Richard, A.J., et al., *Adipose tissue: physiology to metabolic dysfunction.* Endotext [Internet], 2020.
9. ACHCHI, I. and S. DEROUICHE, *STUDY OF THE EFFECT OF OBESITY ON IMMUNE CELLS ACTIVITY AND CARDIOVASCULAR DISEASE RISK IN EL-OUED (ALGERIA) WOMEN.* Innovare Journal of Medical Sciences, 2021: p. 28-30.
10. Feng, B., P. Xu, and Y. He, *Novel targets in glucose homeostasis and obesity—lesson from rare mutations.* Current Diabetes Reports, 2020. **20**: p. 1-10.
11. Shabir, K., et al., *Asprosin, a novel pleiotropic adipokine implicated in fasting and obesity-related cardio-metabolic disease: Comprehensive review of preclinical and clinical evidence.* Cytokine & Growth Factor Reviews, 2021. **60**: p. 120-132.

12. Mazur-Bialy, A.I., *Asprosin—a fasting-induced, glucogenic, and orexigenic adipokine as a new promising player. Will it be a new factor in the treatment of obesity, diabetes, or infertility? A review of the literature.* Nutrients, 2021. **13**(2): p. 620.
13. Ovali, M.A. and I. Bozgeyik, *Asprosin, a C-terminal cleavage product of fibrillin 1 encoded by the FBN1 gene, in health and disease.* Molecular Syndromology, 2022. **13**(3): p. 175-183.
14. Wachowska, D., et al. *Asprosin, a novel adipokine, in the mouse hypothalamus: expression and in vitro effect on GT1-7 cells proliferation and GnRH secretion. Preliminary data*. in *Endocrine Abstracts*. 2023. Bioscientifica.
15. Hernández-Camacho, J.D., et al., *Zinc at the crossroads of exercise and proteostasis.* Redox biology, 2020. **35**: p. 101529.
16. Barchielli, G., A. Capperucci, and D. Tanini, *The role of selenium in pathologies: an updated review.* Antioxidants, 2022. **11**(2): p. 251.
17. Xu, L., et al., *Effects of magnesium supplementation on improving hyperglycemia, hypercholesterolemia, and hypertension in type 2 diabetes: A pooled analysis of 24 randomized controlled trials.* Frontiers in Nutrition, 2023. **9**: p. 1020327.
18. Zhang, L., et al., *Circulating asprosin concentrations are increased in type 2 diabetes mellitus and independently associated with fasting glucose and triglyceride.* Clinica chimica acta, 2019. **489**: p. 183-188.
19. Beutler, L.R. and Z.A. Knight, *A spotlight on appetite.* Neuron, 2018. **97**(4): p. 739-741.
20. Long, W., et al., *Decreased circulating levels of asprosin in obese children.* Hormone research in paediatrics, 2019. **91**(4): p. 271-277.
21. Wang, Y., et al., *Plasma asprosin concentrations are increased in individuals with glucose dysregulation and correlated with insulin resistance and first-phase insulin secretion.* Mediators of inflammation, 2018. **2018**(1): p. 9471583.
22. Ma, H., et al., *Peripheral CB1 receptor neutral antagonist, AM6545, ameliorates hypometabolic obesity and improves adipokine secretion in monosodium glutamate induced obese mice.* Frontiers in pharmacology, 2018. **9**: p. 156.
23. Coelho, M., T. Oliveira, and R. Fernandes, *State of the art paper Biochemistry of adipose tissue: an endocrine organ.* Archives of medical science, 2013. **9**(2): p. 191-200.
24. Ugur, K. and S. Aydin, *Saliva and blood asprosin hormone concentration associated with obesity.* International journal of endocrinology, 2019. **2019**(1): p. 2521096.
25. Marreiro, D.d.N., M. Fisberg, and S.M.F. Cozzolino, *Zinc nutritional status and its relationships with hyperinsulinemia in obese children and adolescents.* Biological trace element research, 2004. **100**: p. 137-149.

26. Ozata, M., et al., *Increased oxidative stress and hypozincemia in male obesity.* Clinical biochemistry, 2002. **35**(8): p. 627-631.
27. Feitosa, M.C.P., et al., *Plasma concentration of IL-6 and TNF-α and its relationship with zincemia in obese women.* Revista da Associação Médica Brasileira, 2013. **59**: p. 429-434.
28. Ferro, F.E.D., et al., *Biomarkers of metabolic syndrome and its relationship with the zinc nutritional status in obese women.* Nutricion hospitalaria, 2011. **26**(3): p. 650-654.
29. Kadhim, H.M., et al., *Effects of melatonin and zinc on lipid profile and renal function in type 2 diabetic patients poorly controlled with metformin.* Journal of pineal research, 2006. **41**(2): p. 189-193.
30. Beletate, V., R. El Dib, and Á.N. Atallah, *Zinc supplementation for the prevention of type 2 diabetes mellitus.* Cochrane Database of Systematic Reviews, 2007(1).
31. Zaky, D.S., et al., *Zinc level and obesity.* The Egyptian Journal of Internal Medicine, 2013. **25**: p. 209-212.
32. Ortega, R.M., et al., *Young children with excess of weight show an impaired selenium status.* International journal for vitamin and nutrition research, 2012. **82**(2): p. 121.
33. Stranges, S., et al., *A prospective study of dietary selenium intake and risk of type 2 diabetes.* BMC public health, 2010. **10**: p. 1-8.
34. Watanabe, L.M., et al., *The influence of serum selenium in differential epigenetic and transcriptional regulation of CPT1B gene in women with obesity.* Journal of Trace Elements in Medicine and Biology, 2024. **83**: p. 127376.
35. Cavedon, E., et al., *Selenium supplementation, body mass composition, and leptin levels in patients with obesity on a balanced mildly hypocaloric diet: A pilot study.* International journal of endocrinology, 2020. **2020**(1): p. 4802739.
36. Wang, Y., et al., *Significant beneficial association of high dietary selenium intake with reduced body fat in the CODING study.* Nutrients, 2016. **8**(1): p. 24.
37. Tinkov, A.A., et al., *Selenium and selenoproteins in adipose tissue physiology and obesity.* Biomolecules, 2020. **10**(4): p. 658.
38. Xu, M.-R., et al., *Serum Magnesium Levels Are Negatively Associated with Obesity and Abdominal Obesity in Type 2 Diabetes Mellitus: A Real-World Study.* Diabetes & Metabolism Journal, 2024.
39. Pei, X., et al., *Relationship between serum magnesium concentration and obesity in Chinese adults.* Wei Sheng yan jiu= Journal of Hygiene Research, 2018. **47**(6): p. 1002-1007.
40. ul Hassan, S.A., et al., *Comparison of serum magnesium levels in overweight and obese children and normal weight children.* Cureus, 2017. **9**(8).

41. Shi, Z. and A.B. Abou-Samra, *Association of low serum magnesium with diabetes and hypertension: Findings from Qatar Biobank study.* Diabetes research and clinical practice, 2019. **158**: p. 107903.
42. Li, Q., et al., *Associations of serum magnesium levels and calcium–magnesium ratios with mortality in patients with coronary artery disease.* Diabetes & Metabolism, 2020. **46**(5): p. 384-391.
43. Ozcaliskan Ilkay, H., et al., *Association between magnesium status, dietary magnesium intake, and metabolic control in patients with type 2 diabetes mellitus.* Journal of the American College of Nutrition, 2019. **38**(1): p. 31-39.
44. Babikr, W.G., et al., *Impact of body mass index on hypomagnesemia and hypocalcemia in type 2 diabetic patients.* Biomedical and Pharmacology Journal, 2016. **9**(3): p. 933-940.
45. Alberti, K.G.M., P. Zimmet, and J. Shaw, *The metabolic syndrome—a new worldwide definition.* The Lancet, 2005. **366**(9491): p. 1059-1062.
46. Zaakouk, A.M., M.A. Hassan, and O.A. Tolba, *Serum magnesium status among obese children and adolescents.* Egyptian Pediatric Association Gazette, 2016. **64**(1): p. 32-37.
47. Lu, L., et al., *Magnesium intake is inversely associated with risk of obesity in a 30-year prospective follow-up study among American young adults.* European journal of nutrition, 2020. **59**: p. 3745-3753.
48. Choi, J., L. Joseph, and L. Pilote, *Obesity and C-reactive protein in various populations: a systematic review and meta-analysis.* Obesity reviews, 2013. **14**(3): p. 232-244.
49. Barbagallo, M. and L. Dominguez, *Magnesium and aging.* Current pharmaceutical design, 2010. **16**(7): p. 832-839.
50. Kostov, K., *Effects of magnesium deficiency on mechanisms of insulin resistance in type 2 diabetes: focusing on the processes of insulin secretion and signaling.* International journal of molecular sciences, 2019. **20**(6): p. 1351.
51. Senyigit, A., et al., *The Associations between Asprosine, Clusterin, Zinc Alpha-2-Glycoprotein, Nuclear Factor Kappa B, and Peroxisome Proliferator-Activated Receptor Gamma in the Development of Complications in Type 2 Diabetes Mellitus.* Journal of Clinical Medicine, 2024. **13**(20): p. 6126.