History of Medicine, 2023, 9(2): 514–518
DOI: 10.17720/2409-5834.v9.2.2023.068

Estimation of Asprosin hormone in obese Iraqi patients with hypothyroidism

Lamia H. A. Al- Sultan[1], Yasser A. H. Al-Issa[2]

[1]*College of Science, Chemistry Department, University of Baghdad, Iraq*
Email: lamchemist20@gmail.com
[2]*College of Science, Chemistry Department, University of Baghdad, Iraq*
Email: yasser.a@sc.uobaghdad.edu.iq

Received: 11 May 2023 **Accepted:** 10 June 2023

Citation: Al- Sultan LHA, Al-Issa YAH (2023) Estimation of Asprosin hormone in obese Iraqi patients with hypothyroidism. History of Medicine 9(2): 514–518. https://doi.org/10.17720/2409-5834.v9.2.2023.068

Abstract

The hormone asprosin is secreted by adipose tissue. It performs a variety of functions in the brain and other bodily systems, including hunger, cellular demise, insulin resistance (IR), and glucose metabolism. A pathological relationship exists between IR and high asprosin levels. patients with obesity are more likely to experience thyroid issues. The present study aimed to compare asprosin levels between the control, obesity and hypothyroidism with obese. This study included 170 patients, fifty of them had hypothyroidism with obesity, and sixty obese patients compared with sixty healthy controls.

The results show that the concentrations of asprosin, HbA1c, BMI, TSH, T4, T3, cholesterol, triglycerides, VLDL, and LDL all considerably increased in hypothyroidism and obesity groups, but decreased HDL level compared to the healthy group, However, there was no statistically significant changes in lipid profile levels of obesity compared to the hypothyroidism with obese group. Low serum asprosin level was seen in hypothyroidism with obesity patients but higher asprosin level was detected in obesity which is associated with inadequate control patients. In summary, this paper argued that is a close relationship of asprosin for thyroid and obesity disorder.

Key Words

Asprosin, Hypothyroidism, Thyroid dysfunction, Obesity ,Lipid profile

The second most frequent endocrine system glandular disorder is thyroid dysfunction[1, 2].Hypothyroidism and hyperthyroidism are the two most prevalent thyroid conditions or diseases [3, 4].The primary factor behind thyroid issues, including hypothyroidism Notw it hstanding the fact that the most common cause of primary hypothyroidism in regions with abundant iodine supplies is autoimmune thyroiditis (Hashimoto's disease), there is an environmental iodine deficit [5, 6].The majority of professional medical guidelines recommend low T4 monotherapy "as the preparation of choice" for thyroid hormone replacement due to the uncertainty surrounding long-term efficacy and potential safety concerns, and advise against the "routine use" of combination therapy with T4 and low T3 [7].

Low thyroid hormone levels cause hypothyroidism, which has a wide range of causes and symptoms[8]. Hypothyroidism left untreated raises mortality and morbidity [9]. Thyroid hormones and body mass index BMI have a more complex relationship than first meets the eye. Obesity is a complicated condition that can come from altered eating patterns, endocrine, genetic, or hypothalamic issues [10, 11].

Adipocyte hypertrophy, visceral adiposity, ectopic fat deposition, hormone release, and the production of proinflammatory proteins like the abundance of cytokines all contribute to the maintenance of adiposopathy, which may result in metabolic disease [12]. Because adiposopathy leads to the deregulation of metabolic pathways, it can be therefore classifying

obesity as a primary illness. The metabolic conditions most closely linked to primary obesity are atherosclerosis, hypertension, dyslipidemia, type II diabetes, hyperandrogenism in women, and hypo-/hyperandrogenism in men [13].

According to recent research, obesity can result from a thyroid hormone deficit (hypothyroidism), whereas a thyroid hormone surplus (hyperthyroidism) results in weight loss [14]. Adipocyte hypertrophy, visceral adiposity, ectopic fat deposition, hormone release, and the production of proinflammatory proteins like the abundance of cytokines all contribute to the maintenance of adiposopathy, which may result in metabolic disease[15].

The combined effects of hypothyroidism and hypometabolism include weight gain, increased cholesterol levels, reduced lipolysis, and decreased gluconeogenesis [16].One of the signs of hypothyroidism is typically weight gain [17, 18]. Obesity and weight growth are caused by increased adipose tissue energy storage [19, 20]. A novel hormone called asprosin is released by white adipocytes while they are fasting and acts as a glucogenic [21].

Recent researches suggested that asprosin may significantly contribute to obesity, yet these studies also seem to conflict with one another. Obesity and higher asprosin levels in both people and animals have been related in numerous studies [22, 23]. Researchers in these studies were able to show that fat mice had lower body weights and a decreased appetite thanks to asprosin-specific antibodies, which suggests that their blood asprosin levels are particularly high [22-24].

Asprosin concentrations are correlated with both cholesterol levels and waist circumference triglyceride (TG). Another study's findings support the notion that asprosin can be produced by the salivary glands in humans[25]. According to the patients' rising body mass index (BMI), asprosin and low-density lipoprotein cholesterol (LDL-C) levels rose in their saliva [26, 27]. Weight gain, higher cholesterol levels, impaired lipolysis, and reduced gluconeogenesis are symptoms of hypothyroidism and hypometabolism [28].

Materials and procedure

Each Iraqi participant in the study conducted at the National Center for Educational Labs gave verbal agreement after being informed of the study's goals between December 2022 and March 2023. Patients between the ages of 35 and 65 were chosen for this study. In the same period, sixty obese patients were compared with healthy controls, and fifty patients were diagnosed with hypothyroidism along with obesity. Individuals who underwent a thyroidectomy, had thyroid cancer, hyperthyroidism, diabetes, or were pregnant were excluded in this study. The function of thyroid hormones was evaluated utilizing an enzyme-linked immunoassay sandwich method (DRG). Moreover, an enzyme-linked immunoassay was used with the manufacturer's kit to determine the presence of asprosin (MyBioSource). Also, using an enzymatic colorimetric method, the kit made available by (Linear, Spain) was utilized to determine (total cholesterol, triglyceride, HDL, LDL, and VLDL). This study aimed to compare asprosin levels between the Control, Obesity and hypothyroidism with obese.

Statistics examination

The statistical analysis is Prism 9.5.0 was used to carry out the needed data analysis. The independent sample t-test was used to compare parameter means between groups; we used the more general descriptive statistic to give a high-level summary of our results. Statistics were deemed significant when the p-value was less than 0.05.

Results and Discussion

In (hypothyroidism with obesity), thyroid hormones, BMI, and lipid profiles have been evaluated and compared to the obesity and control groups, The results show that there is a significant increase differences in asprosin, HbA1c, BMI, T3, total cholesterol, triglyceride , LDL and VLDL ($p<0.001$) when the comparison between the control group with obesity group, while a significant decrease in thyroid stimulating hormone (TSH), triiodothyronine (T4) and high-density lipoprotein (HDL) as shown in table 1.

Table 1: Table (1): Comparisons between the studied parameters between obesity group and healthy subject group

Parameters	Groups	N	Mean ±SD	P-value
ASPROSIN (ng /mL)	Control	48	27.28±5.754	0.001***
	Obesity	48	34.86±9.921	
TSH (uUI / L	Control	60	2.571±1.267	0.001***
	Obesity	60	1.360±0.1382	
T3 (nmol/L)	Control	60	1.989±0.4608	0.001***
	Obesity	60	2.346±0.07897	
T4 (nmol/L)	Control	60	97.05±22.25	0.001***
	Obesity	60	64.73±3.312	
BMI(Kg/m²)	Control	60	25.79 ± 2.452	0.001***
	Obesity	60	35.03±2.392	
HbA1c	Control	60	4.413±0.3510	0.001***
	Obesity	60	5.457±0.3886	
total cholesterol (mg/dl)	control	60	146.5±25.28	0.001***
	obesity	60	276.1±39.18	
triglyceride (mg/dl)	control	60	91.82±6.808	0.001***
	obesity	60	227.1±48.86	
HDL (mg/dl)	control	60	53.33±4.803	0.001***
	obesity	60	33.02±1.778	0.001***
LDL (mg/dl)	control	60	74.81±26.30	0.001***
	obesity	60	206.4±38.68	
VLDL (mg/dl)	control	60	18.36± 1.362	0.001***
	obesity	60	45.43±9.772	

(Mean + SD) is the result given.

P-values below 0.05 are regarded as significant

P-values over 0.05 are regarded as being no significant

Obesity is a complicated ailment that can come from altered eating patterns, endocrine, genetic, or hypothalamic issues [29, 30].

The underlying cause of obesity is a condition known as adiposopathy, which is also known as "sick fat." It is described as "pathologic adipose tissue anatomic/functional disturbances promoted by positive caloric balance in genetically and environmentally susceptible individuals that result in adverse endocrine and immune responses that may cause or worsen metabolic disease." [31]. Dyslipidemia, a disorder that affects lipoprotein metabolism, usually occurs in obese patients. Low-density lipoprotein (LDL) and total cholesterol levels all rise, but the levels of high-density lipoprotein (HDL) are extremely low [32]. Dyslipidemia, or excessively high levels of total cholesterol, LDL, and triglycerides in the blood, is another condition associated with hypothyroidism. The production, elimination, and modification of cholesterol are all impacted by thyroid hormone. However, recent research show that TSH performs lipid metabolism independently of thyroid hormone[33].

On the other hand, the results in table 2 show that there is a significant decrease differences in asprosin, BMI, T3 ($p<0.001$) when comparing the group of people with obesity and hypothyroidism to the obese group, while significant increase in thyroid stimulating hormone (TSH), (T4) and HbA1c. But The results shown that there is no significant in lipid profile between the group obesity and hypothyroidism with obese group.

Table (2): Comparisons between the studied parameters between hypothyroidism with obesity group and obesity group.

Parameters	Groups	N	Mean ±SD	P-value
Asprosin (ng /mL)	obesity	48	34.86±9.921	0.001***
	Hypothyroidism with obese	48	21.31 ±6.925	
TSH (uUI / L	obesity	60	1.360±0.1382	0.001***
	Hypothyroidism with obese	60	19.38±16.81	
T3 (nmol/L)	obesity	60	2.346 ±0.07897	0.001***
	Hypothyroidism with obese	60	1.036 ± 0.9095	
T4 (nmol/L)	obesity	60	64.73± 3.312	0.001***
	Hypothyroidism with obese	60	50.48 ± 7.093	
BMI(Kg/m²)	obesity	60	35.03 ± 2.392	0.001***
	Hypothyroidism with obese	60	32.48 ± 1.923	
HbA1c	Obesity	60	5.457 ±0.3886	0.001***
	Hypothyroidism with obese	60	6.521 ± 0.360	
Total cholesterol (mg/dl)	obesity	60	276.1 ± 39.18	0.2032
	Hypothyroidism with obese	60	267.2 ± 32.57	
Triglyceride (mg/dl	Obesity	60	227.1 ± 48.86	0.4420
	Hypothyroidism with obese	60	220.1±46.40	
HDL (mg/dl)	Obesity	60	33.02± 1.778	0.1070
	Hypothyroidism with obese	60	34.44±1.387	
LDL (mg/dl)	Obesity	60	206.4 ± 38. 68	0.1075
	Hypothyroidism with obese	60	193.8 ± 42.84	
VLDL (mg/dl)	obesity	60	45.43 ± 9.772	.442
	Hypothyroidism with obese	60	44.02 ± 9.281	

(Mean + SD) is the result given.

P-values below 0.05 are regarded as significant

P-values over 0.05 are regarded as being no significant

This study investigates potential associations between asprosin hormone and the condition in people with hypothyroidism and obesity.

As part of the study, obese patients were diagnosed with hypothyroidism by a skilled medical professional, and obese control groups were compared clinically and biochemically. Patient with obesity and hypothyroidism exhibited increased lipid levels compared to control.

The data in the previous table show that when TSH levels are high, T4 levels are noticeably lower in individuals with (hypothyroidism with obesity). The T3 level revealed nothing particularly notable. Poor BMI control is more common in obese individuals.

T4 levels are low when obesity is uncontrolled[34]. It has been associated with peripheral T4 to T3 conversion suppression, which frequently returns to normal control [35, 36]. As shown by Table 1 and Table 2 the obesity with hypothyroidism and obese populations had considerably higher asprosin hormone levels than the control group. Weight gain and alterations in lipid profiles are results of hypothyroidism. These modifications impact fat tissue, glucose, and insulin[23]. Thyroid profile abnormalities might affect asprosin secretion, which can cause further problems[18].

According to the results of this study, those patients with obesity-related hypothyroidism have BMIs that are noticeably greater than those in the control group. In contrast to lower TSH receptor expression, which results in less negative feedback and higher levels of TSH and T4 in the bloodstream, increased adipose mass in obesity promotes TSH release, which raises T4 through the hypothalamic-pituitary-thyroid (HPT) axis. These occurrences result in thyroid malfunction and decreased energy expenditure, which enhance fat storage [37].

Thyroid dysfunction and obesity are two conditions that have a close connection [38]. All blood lipoproteins, including total cholesterol, triglyceride, LDL, and VLDL, were significantly higher in the hypothyroidism with obesity group in this study compared to the healthy group, with the exception of serum HDL levels, which were lower in patients compared to the control group. In a recent investigation, individuals with obesity and hypothyroidism had significantly higher levels of all lipid markers except HDL [39]. On the other hand, Asprosin can also be secreted by cells under hyperlipidemic conditions [28]. Strong correlations between asprosin and triglyceride , HDL, HbA1C, and BMI have been discovered [40]. These findings demonstrate that individuals with obesity and hypothyroidism have metabolic problems that cause considerable fat deposition in the liver and adipose tissue, especially white adipose tissue, which is the main source of the asprosin hormone and lowers asprosin levels [41].

Conclusion

According to the study's results, patients with obesity and hypothyroidism had close ties between poor fat, BMI, and serum asprosin levels in comparison with the control group. Asprosin in control group showed decrease level when compared to obesity, hypothyroidism with obesity. As a result, this investigation explores the probable connection between hypothyroidism and asprosin hormone to identify risk factors for thyroid dysfunction and obesity.

References

1.Rashead, Q.A. and D.M. Hamid, The effect of thyroid hormone on some biochemical factors of kidney. International Journal of Advanced Research, 2015. 3(7): p. 290-297.

2.Weheeb, M.A., K.M. Sultan, and M.W.A. Obaidy, Spiro metric Tests and Thyroid Hormone Concentrations in sample of Iraqi Patients with Chronic Obstructive Pulmonary Disease. Journal of the Faculty of Medicine Baghdad, 2014. 56(3): p. 278-282.

3.Kanase, N.V., S.A. Pangoankar, and A.R. Panat. A Robust Approach of Estimating Voice Disorder Due to Thyroid Disease. in Advances in Signal and Data Processing: Select Proceedings of ICSDP 2019. 2021. Springer.

4.Wahab, T.A.A., Effect of Iodine Overdose on Thyroid Hyperplasia. Iraqi National Journal of Nursing Specialties, 2009. 22(2).

5.Chaker, L., et al., Hypothyroidism and hypertension: fact or myth?–Authors' reply. The Lancet, 2018. 391(10115): p. 30.

6.Aqeeli, M.J.S.A. and H.S. Hassan, Effectiveness of an Instructional Program on Hypothyroidism Patients' Knowledge toward Self Care in Baghdad Teaching Hospitals.

7.Fawzi, S.M., I.A. Abdul-hassan, and B.M. Mahdi, Correlation between thyroid hormones and anti-TSHR Ab in graves' disease. Iraqi journal of biotechnology, 2018. 17(1).

8.Hassan, A.F. and S.A. Ahmed, Effectiveness of an Educational Program on Nurses' Knowledge concerning Early Detection of Hypothyroidism/Hyperthyroidism in Baghdad Teaching Hospitals. Pakistan Journal of Medical and Health Sciences, 2021. 15(3): p. 782-784.

9.Duarte Ariza, Ó.J. and C. Arteaga, PentaHealth. 2022, Universidad del Rosario.
10.Saleh, B.O., M.J. Majeed, and G.M. Oreaby, Irisin peptide is myokine, anti-obesity and anti-lipidemic factor. Am J Res Comput, 2015. **3**(1): p. 20-30.
11.Ali, L.K., The Effect of Chronic Renal Failure on Thyroid Hormones. Iraqi J pharm Sci, 2010. **19**(1).
12.De Lorenzo, A., et al., Why primary obesity is a disease? Journal of translational medicine, 2019. **17**(1): p. 1-13.
13.Obesity, P.C. and B. High, Overweight & Obesity. CDC. Available online: https://www. cdc. gov/obesity/childhood/defining. html (accessed on 14 June 2019), 2019.
14.Pearce, E.N., Thyroid hormone and obesity. Current Opinion in Endocrinology, Diabetes and Obesity, 2012. **19**(5): p. 408-413.
15.Volke, L. and K. Krause, Effect of thyroid hormones on adipose tissue flexibility. European Thyroid Journal, 2021. **10**(1): p. 1-9.
16.Groener, J.B., et al., Asprosin response in hypoglycemia is not related to hypoglycemia unawareness but rather to insulin resistance in type 1 diabetes. PLoS One, 2019. **14**(9): p. e0222771.
17.Muñoz-Ortiz, J., et al., Prevalence of hyperthyroidism, hypothyroidism, and euthyroidism in thyroid eye disease: a systematic review of the literature. Systematic reviews, 2020. **9**(1): p. 1-12.
18.Kantorowicz, M., et al., Nordic walking at maximal fat oxidation intensity decreases circulating asprosin and visceral obesity in women with metabolic disorders. Frontiers in Physiology, 2021. **12**: p. 726783.
19.Phillips, C.L. and B.E. Grayson, The immune remodel: weight loss-mediated inflammatory changes to obesity. Experimental Biology and Medicine, 2020. **245**(2): p. 109-121.
20.Mohammed, D.H., B.K. Abed, and F.A.K. Khazaal, Prevalence of Anxiety in Adults Obese in Baghdad (Obesity Center) According to Standard Scoring System (Beck Anxiety Inventory). Prevalence, 2016. **6**(4).
21.Ameen, M.R., D.M. Sulaiman, and K.H. Muho, Serum Asprosin levels in women with polycystic ovary syndrome in Duhok city, Kurdistan Region of Iraq. Journal of Life and Bio Sciences Research, 2021. **2**(2): p. 36-41.
22.Wang, M., et al., Serum asprosin concentrations are increased and associated with insulin resistance in children with obesity. Annals of nutrition and metabolism, 2019. **75**(4): p. 205-212.
23.Cantay, H., et al., Investigation of serum and adipose tissue levels of asprosin in patients with severe obesity undergoing sleeve gastrectomy. Obesity, 2022. **30**(8): p. 1639-1646.
24.Sünnetçi Silistre, E. and H.U. Hatipoğl, Increased serum circulating asprosin levels in children with obesity. Pediatrics International, 2020. **62**(4): p. 467-476.
25.Morcos, Y.A., et al., Sensitive asprosin detection in clinical samples reveals serum/saliva correlation and indicates cartilage as source for serum asprosin. Scientific Reports, 2022. **12**(1): p. 1340.
26.Ugur, K. and S. Aydin, Saliva and blood asprosin hormone concentration associated with obesity. International journal of endocrinology, 2019. **2019**.
27.Al-Hadidi, E.E.K. and W.M.L. Al-Obaidi, Assessment of asprosin level and some of physiological variables in patients with cardiovascular diseases in Kirkuk city, Iraq. Biomedicine, 2022. **42**(5): p. 973-977.
28.Gopalakrishnan, M., T. Kamalakshi, and P. Geetha, Insulin resistance and serum lipid profile in hypo-and hyper-thyroidism and their relationship with serum thyroid-stimulating hormone levels. National Journal of Physiology, Pharmacy and Pharmacology, 2020. **10**(3): p. 247-252.
29.Bays, H.E., Adiposopathy: is "sick fat" a cardiovascular disease? Journal of the American College of Cardiology, 2011. **57**(25): p. 2461-2473.
30.Hayyawi, A.H., K.R. Hasan, and R.K. Lafta, Impact of nutrition clinic on obesity in Baghdad, Iraq: first year outcome. Saudi Journal of Obesity, 2016. **4**(2): p. 80.
31.Neeland, I.J., P. Poirier, and J.-P. Després, Cardiovascular and metabolic heterogeneity of obesity: clinical challenges and implications for management. Circulation, 2018. **137**(13): p. 1391-1406.
32.Vekic, J., et al., Obesity and dyslipidemia. Metabolism, 2019. **92**: p. 71-81.
33.Liu, H. and D. Peng, Update on dyslipidemia in hypothyroidism: the mechanism of dyslipidemia in hypothyroidism. Endocrine Connections, 2022. **11**(2).
34.Marmouch, H., et al., Eating behaviour, alexithymia, and BMI in obesity. Metabolism-Clinical and Experimental, 2021. **116**.
35.Luongo, C., M. Dentice, and D. Salvatore, Deiodinases and their intricate role in thyroid hormone homeostasis. Nature Reviews Endocrinology, 2019. **15**(8): p. 479-488.
36.Aruna, V., Correlation of T3, T4 and TSH with lipid indices in obesity. Int J Res Med Sci, 2021. **9**(2078): p. 10.18203.
37.Xiao, Y., et al., Chronic stress, epigenetics, and adipose tissue metabolism in the obese state. Nutrition & Metabolism, 2020. **17**(1): p. 1-16.
38.Mahdavi, M., et al., Investigating the prevalence of primary thyroid dysfunction in obese and overweight individuals: Tehran thyroid study. BMC Endocrine Disorders, 2021. **21**(1): p. 1-12.
39.Li, Y., et al., Evaluation of the thyroid characteristics and correlated factors in hospitalized patients with newly diagnosed type 2 diabetes. Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy, 2022: p. 873-884.
40.Hong, T., et al., High serum asprosin levels are associated with presence of metabolic syndrome. International journal of endocrinology, 2021. **2021**.
41.Muthu, M.L. and D.P. Reinhardt, Fibrillin-1 and fibrillin-1-derived asprosin in adipose tissue function and metabolic disorders. Journal of Cell Communication and Signaling, 2020. **14**(2): p. 159-173.