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The Association Between Leptin and Asprosin Levels in Men Patients with Type II Diabetes Mellitus

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RESEARCH ARTICLE

The Association Between Leptin and Asprosin Levels in Men Patients with Type II Diabetes Mellitus

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ABSTRACT

Leptin and Asprosin are adipokines secreted by Adipose tissue. The leptin and asprosin molecules have a number of functions in the central nervous system and other body systems: appetite, glucose metabolism, insulin resistance, and cellular death. The point of this study is to know the potential relationship between leptin and asprosin hormone in men patients with type II diabetes mellitus. The present study was designed to conduct a comparative analysis of several essential biomarkers found in the sera of individuals with diabetes via estimating Leptin, Asprosin, Fasting blood Glucose, HbA1c, Body Mass Index, lipid profile as well as liver function testes with type II diabetes mellitus. The study groups consist of 60 sample that were separated into two groups: group I consisted of 30 diabetic men, and group II included 30 healthy men. Biochemical parameters of every participant were ascertained. The quantification of leptin and asprosin in serum was conducted using an enzyme-linked immunosorbent assay. According to the results of this investigation, levels of leptin, asprosin, Fasting blood Glucose and HbA1c were markedly high for type II diabetes mellitus group compared with control group [(5.72 ± 1.05), (10.47 ± 2.14), (209.97 ± 49.71), and (9.40 ± 1.31)] respectively, with a significant difference ($P = 0.001$). Elevated concentrations of leptin and asprosin in the serum of patients were associated with inadequate glycemic control. A positive association appears to exist between leptin and asprosin in patients. The study concluded that leptin and asprosin are diagnostic factor for type II diabetes mellitus.

Keywords: Adipose tissue, Asprosin, Fasting blood glucose, Leptin, Type II diabetes mellitus

Introduction

In recent years, the prevalence of diabetes mellitus (DM), which is among the most significant public health issues, has increased.[1] 90% of all instances of diabetes are attributed to DMII.[2] Diabetes mellitus is a subset from metabolic disorders known as hyperglycemia caused by the secretion of pancreatic insulin, or insulin development, or both.[3] Dyslipidemia, hyperglycemia, and insulin resistance (IR) are all conditions associated with DMII.[4] Adipokines are involved in a multitude of physiological processes, encompassing fullness of stomach and appetite regulation, insulin sensitivity, adipogenesis, energy metabolism in insulin-sensitive tissues, endothelial function, maintenance of blood pressure, energy expenditure activity, hemostasis, and fat distribution within pancreatic cells.[5,6] Recent research has devoted a substantial amount of attention to examining the functions of adipokines, particularly asprosin and leptin, in the regulation of metabolism, given their emergence as noteworthy variables of interest.[7] Leptin, a peptide that is produced primarily in white adipose tissue; its secretion is proportional to fat mass.[8] Leptin is particularly influential in the regulation of energy expenditure and ingestion by transmitting signals to the hypothalamic feeding centers.[9] Individuals experiencing a leptin deficiency

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acquire weight, consuming more food, and accumulation of more adipose tissue. Leptin is also capable of regulating insulin function and glucose homeostasis.[10] Several target tissues, including the central nervous system, liver, pancreas, and skeletal muscle, are likely to be involved in the direct and/or indirect mediation of leptin's effects to enhance glucose homeostasis. Inconsistent results have been observed concerning leptin levels in individuals with diabetes; some researches indicated that diabetic subjects have lower leptin levels than healthy controls, while others find no difference and still others reported higher leptin levels. In addition, numerous variables influence levels of leptin, including anti-diabetes medication, age, gender, body mass index, central adiposity, insulin levels, and insulin sensitivity.[11] Asprosin, a novel hormone that is secreted by white adipocytes in response to fasting, stimulates the G-protein AMP-PKA pathway and increases glucose release from hepatocytes. In addition to its participation in a multitude of processes occurring within the central nervous system (CNS), the asprosin molecule is also present in numerous organs and tissues. It participates in cellular apoptosis, glucose metabolism, appetite, IR., and additional processes.[12] According to clinical investigations, individuals with metabolic syndrome and DMII have a higher concentration of serum asprosin than healthy controls.[13] Understanding the intricate interplay between leptin and asprosin and the pathophysiology of DMII is essential for developing targeted therapeutic interventions. The aim of this study is finding the relation between leptin and asprosin in men patients with DMII and healthy men. Ultimately, we are trying to ascertain whether leptin and asprosin can serve as indicators for DMII. Expected findings may contribute to the development of innovative strategies to combat DMII and improve the lives of individuals affected by this prevalent metabolic disorder.

Materials and methods

Patients and control

The expressed verbally informed consent was obtained from every member of the groups in the study performed in General Hospital General Hospital (Baghdad). This study received approval from Committee of Scientific Research of the College of Science for Women at the University of Baghdad. 60 men between the ages of 30 and 60 were chosen. Following a consultation with an experienced medical practitioner, 30 men patients were diagnosed with DMII, while 30 healthy volunteers were chosen to serve as healthy controls at the same time. Data regarding the manner of life and demographic information of the participants were collected through in-person interviews. The questionnaire for this purpose had been developed in advance. The blood specimen was acquired from the subject following a ten to twelve-hour fasting. Samples of 10 ml of venous blood were withdrawn from each patient and the control group using a disposable syringe. After placing the blood into a gel tube and allowing it to separate, serum was obtained through centrifugation of the tube at 3000 revolutions per minute for ten minutes. Serum of 1 ml was analyzed for lipid profiles (Cholesterol, Triglycerides, HDL: high-density lipoprotein, LDL: low-density lipoprotein) and blood glucose levels was measured manually using kit (human, Germany), while the liver enzymes were determined from a (1.5 ml) sample of each individual (patient and healthy) serum: AST (Alanine aminotransferase), ALT (Alanine aminotransferase), and ALP (Alkaline phosphatase) was measured manually using kit (Linear, Spain). In order to quantify leptin and asprosin by using an enzyme-linked immunoassay sandwich by (Cloud-Clone Corp, USA), the residual serum was frozen at –20 ˚C.

Statistical analysis

Data were introduced into SPSS V26 statistical software program Tables and graphs were used to display descriptive statistics. Chapiro walk test was used to test normality of distribution of numerical scale data. Independent sample t-test was used to find out significance of differences between mean and Std. P value less than 0.05 was considered a point for distinguishing significance, correlation analysis was used to test the linear relationship between variables.

Including and excluding

1. Diabetes medical history, have already been diagnosed with the type II diabetes mellitus was made according to the World Health Organization Expert Committee on diabetes mellitus.
2. Patients ranging from 30–60 years.
3. Exclusion criteria: - obese patients.

Results and discussion

The clinical characteristics of the study subjects are shown in Tables 1 to 3. The results in Table 1. showed the mean ± SD values of leptin, asprosin and Body Mass Index (BMI) for patients with DMII and control groups. A significant difference ($p = 0.0001$) was noticed in leptin and asprosin levels between patient and control groups [(5.72 ± 1.05), (1.38 ± 0.42), and

Table 1. Serum levels of leptin and asprosin hormone in men patients with DMII and controls.

Parameters	(DMII) (N = 30)	Control Group (N = 30)	P-value
Leptin (ng/ml)	5.72 ± 1.05	1.38 ± 0.42	0.001
Asprosin (ng/ml)	10.47 ± 2.14	1.73 ± 0.35	0.001
BMI (Kg/m^2)	28.35 ± 2.37	27.81 ± 2.71	0.624

Table 2. Serum levels of FBG, HbA1C and lipid profile of study groups.

Parameters	(DMII) Group (N = 30)	Control Group (N = 30)	P-value
FBG (mg/dl)	209.97 ± 49.71	90.10 ± 9.05	0.001
HbA1C %	9.40 ± 1.31	5.01 ± 0.52	0.001
Cholesterol (mg/dl)	215.03 ± 26.01	174.90 ± 24.92	0.869
Triglycerides (mg/dl)	188.47 ± 44.52	116.13 ± 26.08	0.062
HDL (mg/dl)	34.50 ± 8.92	45.20 ± 8.97	0.632
LDL (mg/dl)	125.83 ± 25.93	111.73 ± 19.24	0.303

Table 3. Serum levels of liver enzymes in DMII and control groups.

Parameters	(DMII) Group (N = 30)	Control Group (N = 30)	P-value
AST (U/L)	19.23 ± 5.51	18.83 ± 5.92	0.321
ALT (U/L)	15.01 ± 4.86	18.33 ± 5.69	0.440
ALP (U/L)	76.97 ± 15.38	78.37 ± 12.05	0.219

(10.47 ± 2.14), (1.73 ± 0.35)] respectively, but there was no significant difference (p = 0.624) in body mass index (BMI) between patient and control groups.

As for parameter values showed in Table 2, fasting blood glucose (FBG) levels and glycosylated hemoglobin A1c (HbA1C) were significantly difference in patient group (209.97 ± 49.71), (9.40 ± 1.31) respectively compared to control group (90.10 ± 9.05), (5.01 ± 0.52) respectively at (p = 0.001), while the levels of Cholesterol, Triglycerides, HDL and LDL showed no significant difference (P ≥0.05) between patient group and control group, as shown in Table 2.

The mean ± SD values of serum AST (19.23 ± 5.51), serum ALT, (15.01 ± 4.86), and serum ALP (76.97 ± 15.38) of men with (type II diabetes mellitus (DMII)) showed no significant difference (P ≥0.05) from that of healthy men, as shown in Table 3.

There were significantly negative correlation between leptin and asprosin in control group (r = –0.340, p value = 0.066), while there were significantly positive correlation between leptin and asprosin in men patients with DMII group (r = 0.456, p value = 0.011) by using Pearson correlation at (p≤0.05), this is due to the body's attempt to obtain energy by transmitting signals to the hypothalamic feeding centers which positively affects the secretion of high levels of leptin and asprosin in DMII group.

DM is frequently regarded as a syndrome characterized by hyperglycemia, or abnormally elevated blood glucose levels, which indicates a metabolic disorder. Thirty healthy individuals and thirty diabetic patients participated in this investigation. A lack of statistically significant variation was observed in the age and BMI of the participants across both categories. The patients' diabetic condition was verified through the documentation of their comprehensive medical history. Ultimately, confirmation of DM was established by estimating the FBG concentration to be greater than 126 mg/dL on two separate occasions. In the present study, the serum of leptin and asprosin hormone were markedly high for DMII group compared with control group with significant difference (P < 0.001) as shown in Table 1. The result was agreement with other research, it was shown that serum leptin levels are higher in DMII comparing with control.[7,10,14] On the other hand, the result disagreed with other research, it showed no significant associations between plasma leptin levels and diabetes.[11] According to this study and the study by Peng X, et al., the high levels of leptin in the present study population could be due to its appealing characteristic of reducing glucose levels. Leptin has the ability to decrease blood glucose levels autonomously, specifically in hyperglycemic models of insulin deficiency.[10] The regulatory functions of leptin in relation to energy expenditure and food intake via central signaling pathways are well-defined. Leptin transmits signals to the hypothalamus and hindbrain, which are central, to induce a reduction in food intake and an increase in energy expenditure.[7,9,14] Furthermore, central signaling pathways may be utilized by leptin to exert significant glucoregulatory and insulin-sensitizing effects.[9,10] The direct influence of leptin on neurons that express proopiomelanocortin (POMC) has been shown to contribute to the glucoregulatory effects of leptin signaling, thereby expanding upon these findings. Leptin receptor selective re-expression in POMC-expressing central neurons reduces circulating glucagon concentrations and ameliorates dyslipidemia, hepatic insulin resistance, and blood glucose. Moreover, by influencing lipid metabolism, leptin may potentially contribute to enhancements in insulin sensitivity.[9] The result was in agreement with other research, it showed that in patients with DMII, asprosin serum concentrations were considerably elevated in comparison to those of the healthy controls.[15,16] Research has demonstrated that asprosin inhibits β-cell autophagy via the adenosine monophosphate-activated protein kinase (AMPK) and mammalian target of rapamycin (mTOR) signaling pathways, thereby inducing β-cell apoptosis.[17–19] Other research, found

that asprosin may lead to β-cell dysfunction and impaired glucose tolerance in patients with DMII.[17,19] The study conducted by Romere et al., revealed that asprosin elevates the concentration of glucose in the circulation through the promoting hepatic gluconeogenesis,[20,21] which supports the conclusion in this study that asprosin is associated with FBG and HbA1C. BMI showed no significant ($P > 0.05$) differences in DMII men in this study because of we excluded the patients who had obesity. However, the Median FBG were significantly ($P < 0.001$) higher in men with diabetes subjects than their normal healthy controls as shown in Table 2. Elevated blood glucose levels are a sign of DMII, which is results from impaired pancreatic-cell activity, which is necessary for secretion of insulin. Table 2, shows the serum concentrations of Cholesterol, T.G, HDL, and, LDL in DMII patients' group and control group. In our study, the Cholesterol, T.G, HDL, and, LDL levels no significant difference appeared when compared between patient group and control group with ($P \geq 0.05$). Liver enzymes (ALT, AST, and ALP) levels also were no significant difference appeared when compared between patient group and control group with ($P \geq 0.05$) as shown in Table 3. These results indicate that patients with DMII have metabolic abnormalities that cause raising leptin and asprosin levels, this could be due to the body's attempt to obtain energy by transmitting signals to the hypothalamic feeding centers which positively affects the secretion of high levels of leptin and asprosin in DMII group.

Conclusion

Levels of leptin and asprosin were significantly higher in patients' with DMII that although BMI was similar between the two groups. Thus, a positive association appears to exist between leptin and asprosin in individuals diagnosed with diabetes mellitus. On the basis of these results and those of previous research, leptin and asprosin may influence glucose levels; thus, they may play a significant role in the therapeutic or diagnostic objectives for diabetes.

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Authors' declaration

- Conflicts of Interest: None.
- We hereby confirm that all Tables in the manuscript are ours.
- No animal studies are present in the manuscript.
- Author(s) signed on ethical consideration's approval.
- Ethical Clearance: The project was approved by the local ethical committee at University of Baghdad.

Authors' contribution statement

D.Q.B. and A.Y.A. contributed to the design and implementation of the research, to the analysis of the results and to the writing of the manuscript.

References

1. Hekim MG, Kelestemur MM, Bulmus FG, Bilgin B, Bulut F, Gokdere E, *et al.* Asprosin, a novel glucogenic adipokine: A potential therapeutic implication in diabetes mellitus. Arch Physiol Biochem. 2023;129(5):1038–1044. https://doi.org/10.1080/13813455.2021.1894178
2. Wang LK, Wang H, Wu XL, Shi L, Yang RM, Wang YC. Relationships among resistin, adiponectin, and leptin and microvascular complications in patients with type 2 diabetes mellitus. J Int Med Res. 2020;48(4):117–407. https://doi.org/10.1177/0300060519870407
3. Antar SA, Ashour NA, Sharaky M, Khattab M, Ashour NA, Zaid RT, *et al.* Diabetes mellitus: Classification, mediators, and complications; a gate to identify potential targets for the development of new effective treatments. Biomed Pharmacother. 2023;168:115–134. https://doi.org/10.1016/j.biopha.2023.115734
4. Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, *et al.* Pathophysiology of type 2 diabetes mellitus. Int J Mol Sci. 2020;21(17):62–75. https://doi.org/10.3390/ijms21176275
5. Bakr DQ, Abed AY. The association between leptin and asprosin levels in female patients with type II diabetes mellitus. J Fac Med Baghdad. 2024;66(3):368–73. https://doi.org/10.32007/jfacmedbaghdad.6632346
6. Ali SI, Hasan QI, Ali LI, Khaleel FM, Ali FI. Correlation between asprosin and Irisin in Iraqi patients with type 2 diabetes or high blood pressure. Baghdad Sci. J. 2025;22(7):2246–53. https://doi.org/10.21123/2411-7986.4991
7. Hameed MS, AL-Khakani MF. The dual role of adiponectin and leptin in type 2 diabetes. J Popul Ther Clin Pharmacol. 2023;30(13):200–214. https://doi.org/10.47750/jptcp.2023.30.13.021
8. Liu H, Du T, Li C, Yang G. STAT3 phosphorylation in central leptin resistance. Nutr Metab. 2021;18:1–3. https://doi.org/10.1186/s12986-021-00569-w
9. Cummings BP. Leptin therapy in type 2 diabetes. Diabetes Obes Metab. 2013;15(7):607–612. https://doi.org/10.1111/dom.12048
10. Peng X, Huang J, Zou H, Peng B, Xia S, Dong K, *et al.* Roles of plasma leptin and resistin in novel subgroups of type 2 diabetes driven by cluster analysis. Lipids Health

Dis. 2022;21(1):7–10. https://doi.org/10.1186/s12944-022-01623-z
11. Onyemelukwe OU, Ogoina D, Onyemelukwe GC. Leptin concentrations in type 2 diabetes and non-diabetes Nigerian-Africans. Am J Cardiovasc Dis. 2020;10(4):444–561.
12. Lefta NA, Abed AY, Abed BA. Estimation of asprosin levels in female Iraqi patients with type 2 diabetes and hypothyroidism. J Med Chem Sci. 2023;6(2):433–457. https://doi.org/10.26655/JMCHEMSCI.2023.2.23
13. Zhang X, Jiang H, Ma X, Wu H. Increased serum level and impaired response to glucose fluctuation of asprosin is associated with type 2 diabetes mellitus. J Diabetes Investig. 2020;11(2):349–355. https://doi.org/10.1111/jdi.13148
14. Katsiki N, Mikhailidis DP, Banach M. Leptin, cardiovascular diseases and type 2 diabetes mellitus. Acta Pharmacol Sin. 2018;39(7):1176–1188. https://doi.org/10.1038/aps.2018.40
15. Farrag M, Ait Eldjoudi D, González-Rodríguez M, Cordero-Barreal A, Ruiz-Fernández C, Capuozzo M, *et al.* Asprosin in health and disease, a new glucose sensor with central and peripheral metabolic effects. Front Endocrinol. 2023;13:110–191. https://doi.org/10.3389/fendo.2022.1101091
16. Naiemian S, Naeemipour M, Zarei M, Lari Najafi M, Gohari A, Behroozikhah MR, *et al.* Serum concentration of asprosin in new-onset type 2 diabetes. Diabetol Metab Syndr. 2020;12:1–8. https://doi.org/10.1186/s13098-020-00564-w
17. Wang R, Hu W. Asprosin promotes β-cell apoptosis by inhibiting the autophagy of β-cell via AMPK-mTOR pathway. J Cell Physiol. 2021;236(1):215–221. https://doi.org/10.1002/jcp.29835
18. Ali SE, Khaleel FM. Assessing the activity of renin and GST in the serum of ladies suffering from polycystic ovary syndrome and COVID-19 to predict the danger of cardiac disease. Baghdad Sci J. 2023;20(3):0986–0989. https://dx.doi.org/10.21123/bsj.2023.7879
19. Romere C, Duerrschmid C, Bournat J, Constable P, Jain M, Xia F, *et al.* Asprosin, a fasting-induced glucogenic protein hormone. Cell. 2016;165(3):566–579. https://doi.org/10.1016/j.cell.2016.02.063
20. Zhang Y, Yang P, Zhang X, Liu S, Lou K. Asprosin: Its function as a novel endocrine factor in metabolic-related diseases. J Endocrinol Invest. 2024;3:1–12. https://doi.org/10.1007/s40618-024-02360-z
21. Samawi KA, Ali SI, Salman TA, Alshekhly BA. Exploring the capabilities of metal-doped phthalocyanine (MPC, M = Co, Cu, Fe, Ni, Zn) for adsorption of CO2: A DFT study. J. Mol. Graph. Model. 2025;1(40):109–088. https://doi.org/10.1016/j.jmgm.2025.109088

العلاقة بين مستويات اللبتين والاسبروسين لدى المرضى الذكور المصابين بداء السكري من النوع الثاني

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الخلاصة

اللبتين والأسبروسين عبارة عن اديبوكينات تفرزها الانسجة الدهنية . تلعب جزيئات اللبتين والأسبروسين العديد من الأدوار في الجهاز العصبي المركزي وأجهزة الجسم الأخرى : الجوع واستقلاب الجلوكوز ومقاومة الأنسولين وموت الخلايا .الهدف من هذه الدراسة هو معرفة العلاقة المحتملة بين هرمون اللبتين وهرمون الأسبروسين لدى المرضى الرجال المصابين بداء السكري من النوع الثاني. صممت الدراسة الحالية لمقارنة بعض المؤشرات الحيوية في امصال مرضى السكري من خلال تقدير هرمون اللبتين والأسبروسين ونسبة الجلوكوز في الدم الصائم والسكر التراكمي HbA1c ومؤشر كتلة الجسم وملف الدهون بالإضافة الى اختبارات وظائف الكبد لدى الذكور المصابين بداء السكري من النوع الثاني. تكونت مجموعة الدراسة من 60 عينة تم تقسيمها الى مجموعتين: المجموعة الأولى (30 ذكراً مصاباً بالسكري) والمجموعة الثانية (30 شخصاً من الاصحاء). تم تحديد المتغيرات الكيميائية الحيوية في جميع المشاركين. تم قياس تراكيز مصل اللبتين والأسبروسين باستخدام مقياس الأمتزازالمناعي المرتبط بالإنزيم. وفقا لنتائج هذه الدراسة ان مستويات اللبتين والأسبروسين والجلوكوز في الدم الصائم والسكر التراكمي HbA1c كانت مرتفعة بشكل ملحوظ لدى مجموعة المرضى مقارنة بالمجموعة الضابطة [(5.72± 1.05), (10.47± 2.14), (209.97± 49.71), and (9.40± 1.31)] على التوالي مع وجود فرق معنوي (P = 0.001). ارتبطت التراكيز المرتفعة من اللبتين والاسبروسين في مصل المرضى مع عدم كفاية التحكم في نسبة السكر في الدم. اظهر المرضى علاقة إيجابية بين هرمون اللبتين والأسبروسين. وخلصت الدراسة إلى ان اللبتين والأسبروسين هما من العوامل التشخيصية لمرض السكري النوع الثاني.

الكلمات المفتاحية: الأنسجة الدهنية، الأسبروسين، جلوكوز الدم الصائم، اللبتين، داء السكري من النوع الثاني.