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RESEARCH ARTICLE

Correlation between Asprosin and Irisin in Iraqi Patients with Type 2 Diabetes or High Blood Pressure

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ABSTRACT

Diabetics are twice as likely to experience high blood pressure as non-diabetics. Asprosin a protein hormone has 140 amino-acids, is secreted by white adipose-tissue in response to fasting. When the cAMP, protein kinase-A pathway is activated, it causes a fasting-induced increase in hepatic glucose release into the bloodstream. Irisin, a peptide consisting of 112 amino-acids, is produced by cleaving a protein in the plasma membrane to release an adipocytokine. Enhanced lipid and glucose metabolism, along with anti-inflammatory and antioxidant properties, are among the advantageous impacts of irisin. The purpose of this study is to determine the function of Asprosin and it's linked with irisin in individuals suffering from hypertension and type 2 diabetes. The study included 60 samples, ages 35–65, divided into two groups according to the diagnosis: T2DM patients and patients with hypertension, obtained from the Al-Yarmouk Hospital/Baghdad/Iraq, between June-February/2023. Some biochemical factors were measured for all study groups, which include: Fasting-glucose and lipid-profile were measured by the enzymatic-oxidation method. Using an enzymatic process, Asprosin, insulin, and irisin were measured with an enzyme-linked-immunosorbent-assay. Finally, Homeostasis-Model-Assessment-Insulin-Resistance was calculated according to the specific formula. The result showed a significance in BMI, WHR, irisin, FBS, HOMA-IR. Also, a positive correlation between irisin and Asprosin with BMI, FBS, and insulin. Finally, based on the ROC curve, Asprosin and irisin levels have a high diagnostic value for T2DM and hypertension respectively. From these findings, it is possible to assess the degree of hypertension and T2DM using the Asprosin or irisin level as a biomarker.

Keywords: Asprosin, Diabetes mellitus, Hypertension, Insulin resistance, Irisin, T2DM

Introduction

Type 2 Diabetes Mellitus (T2DM) is a metabolic illness that affects people for the rest of their lives.[1] Accurate blood level regulation is compromised by diabetes mellitus. Type I and Type II DM are the two main varieties. Type I diabetes is identified by malfunctioning or damaged pancreatic cells, exogenous insulin should be administered through injection or insulin pump to patients with Type I DM in order to maintain life. Type II DM usually develops later because of obesity and other comorbidities that induce cells to become resistant to insulin's hormonal activity[2] and characterized by insufficient insulin activity (insulin resistance) and varying degrees of pancreatic beta-cell failure. Unsustainable levels of

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insulin synthesis (increasing need at the onset of the disease) put a lot of pressure on the β-cell secretory machinery, leading to endoplasmic reticulum (ER) stress, and "exhaustion" owing to sustained excessive insulin production.[3] The pancreas is a long, soft, flat, lobulated and yellow gland that sits transversely on the posterior abdominal wall. It is divided into two compartments: pancreatic ducts and acini comprise the extensive exocrine compartment, accounting for 98–99% of the pancreatic mass, while the islets of Langerhans comprise the smaller endocrine compartment, which makes up 1–2 percent of the pancreatic mass. During embryonic development, both endocrine and exocrine cells arise from a single progenitor. The gland's digestive duties are carried out by ductal and acinar cells, which synthesize and release enzymes into the duodenum.[4] The pancreas' endocrine compartment has five distinct hormone-secreting. There are various types of cells, including β-cells that secrete insulin, α-cells that secrete glucagon, δ-cells that release somatostatin, PP-cells that secrete pancreatic polypeptide, and α-cells that produce basal hormone. All of these hormones play role in maintaining glucose homeostasis and nutrition metabolism. The islets of Langerhans are made up of endocrine cells that are mixed with blood arteries, neurons, and a mesodermal derived stromal component. Hormone release is regulated by the close interplay of endocrine and vascular cells, resulting in a perfectly tuned glucose homeostasis in the body.[5] Hypertension is characterized by an increase in blood pressure for an unexplained reason, which raises the risk of heart, brain, and kidney disorders. Ninety percent of people in developed countries will at some point in their lives develop hypertension (blood pressure greater than 140/90 mm Hg). Typically, hypertension coexists with additional cardiovascular risk factors like aging, obesity, HOMA IR, DM, and dyslipidemia.[6] Initial stages of hypertensive cardiovascular disease are marked by subtle damage to target organs including cognitive impairment, microalbuminuria, and left ventricular hypertrophy. Still, long-term uncontrolled hypertension is typically associated with serious consequences such as dementia, heart attack, stroke, and renal failure. The greatest indicator of lowered cardiovascular risk is the drop in blood pressure that all antihypertensive medications cause.[7] Most hypertensive patients need two or more medicines to maintain decreasing blood pressure while taking statins at the same time reduces the risk factor. The bulk of people with hypertension still have uncontrolled hypertension and associated risk factors, despite the availability of numerous safe and effective antihypertensive medications.[8] A unique glucogenic adipokine called asprosin isprimarily produced and released by white adipose tissue during fasting. It has a complicated function in the organs, peripheral tissues, and central nervous system (CNS).[9] A thermogenic hormone called irisin increases brown adipose tissue (BAT), which in turn leads to an increase in energy expenditure. Irisin protein is this protein hormone that makes white adipose tissue (WAT) become brown adipose tissue (BAT). During conversion, this results in energy consumption. Triglycerides and fatty acids are stored in WAT, which also has very few mitochondria. Additionally, they contribute to the emergence of insulin resistance (IR). Triglycerides and fatty acids are stored by WAT, an organelle with a very low mitochondrial content that aids in the formation of IR.[10] Function of asprosin and it's linked with irisin in individuals suffering from hypertension and type 2 diabetes.

Materials and methods

The study included 60 samples from individuals with ages 35–65, divided into two groups according to the diagnosis: patients with T2DM and patients with hypertension, collected from Al-Yarmouk Hospital in Baghdad, Iraq, from June-February-2023. For each study group, biological markers were measured, including: Fasting glucose, cholesterol, triglycerides, LDL, and HDL were measured by the enzymatic oxidation method by use kit (Human, Germany)/enzymatic process. Asprosin, insulin and irisin were measured with an enzyme-linked immunosorbent assay by ELISA kits (Abcam, USA/Catalog No. ab275108), (My BioSource, USA/Catalog No. MBS706887). Finally, HOMA IR was computed using the prescribed formula.

Five milliliters of blood were collected from all patients' participants. The test tubes containing blood were kept at room temperature (25 ˚C) for half an hour. Then, the blood samples were centrifuged at 2000–3000 Rpm for ten minutes to obtain the serum. The separated serum was applied to determine studied parameters.

Inclusion criteria

Patient with hypertension or with type 2 diabetes mellites.

Exclusion criteria

Patients with type 1 diabetes mellites or patients with renal or liver disease, and gestational diabetes.

Table 1. BMI levels between study groups.

Group Parameters	Type 2 diabetes mellites group	Hypertension group	*p*-value
BMI (kg/m^2)	25.64 ± 2.20	35.40 ± 1.38	<0.05
WHR	1.23 ± 0.10	0.94 ± 0.15	<0.05

Table 2. Lipid profile and glucose levels between study groups.

Groups Parameters	Type 2 diabetes mellites group	Hypertension group	*p*-value
FBS (mg/dL)	90.88 ± 4.50	85.47 ± 4.57	<0.05
Cholesterol (mg/dL)	186.78 ± 10.55	190.22 ± 10.51	NS
Triglycerides (mg/dL)	150.41 ± 35.91	155.24 ± 33.18	NS
HDL (mg/dL)	22.41 ± 3.72	20.4 ± 4.12	NS
LDL (mg/dL)	121.31 ± 30.41	130.45 ± 22.7	<0.05

Statistical

Statistically analyzed by SPSS v. 27. The variables were reported as means ± SD, T-test statistical, ROC curve analysis the statistically significant at $p \leq 0.05$.

Results and discussion

The mean ± SD are seen, respectively, in Table 1, which shows a significant difference ($p < 0.05$) in BMI and WHR when comparing the hypertension group and the T2DM group.

Asia is not an exception to the global increase in the prevalence of obesity. It is thought to be a major risk factor for numerous chronic conditions, including dyslipidemia, hypertension, and type 2 diabetes.[9] The result of this study is similar with other research,[11] which reported that WHR is associated with T2DM, as well as, it was noted that among Asians, these markers had a lower correlation with T2DM and hypertension. However, another studies,[12] revealed that in individuals who appeared to be in good health, there was a stronger correlation between BMI and hypertension. It is believed that the most significant risk factor for type 2 diabetes is BMI. Abdominal fat accumulation may be more accurately reflected by WHR, a straightforward indicator of abdominal obesity.[12] However, other research[13] indicates that compared to people in other populations with the same BMI, those with a higher body fat percentage are more likely to develop type 2 diabetes, hypertension, and heart disease. According to our data, WC was more strongly linked to dyslipidemia and T2DM. This is not unexpected given that T2DM and insulin resistance can result from visceral fat, which is the primary source of free fatty acids and inflammatory cytokines in abdominal obesity. For this reason, even in cases where the BMI is not as high, WHR should be measured precisely in order to prevent and diagnose dyslipidemia and type 2 diabetes. Furthermore, weight seems to be more strongly correlated with hypertension than abdominal obesity because a higher body mass index (BMI) is associated with increased peripheral resistance (such as altered cell membranes, hyperinsulinemia), body fluid volume, and hyperactivity of the renin-angiotensin system, all of which contribute to cardiac output, structural hypertrophy, and functional constriction. High visceral fat, which raises leptin levels and induces inflammatory cytokines, lipid disorders, and insulin resistance, may be the cause of the positive correlation between WHR and hypertension.

The results listed in Table 2. explain FBS and lipid levels in Type 2 diabetes mellites, and hypertension groups. A significant difference at ($p < 0.05$) in FBS and LDL levels. While There were no significant differences ($p > 0.05$) in cholesterol, triglyceride, and HDL among each of Type 2 diabetes mellites, hypertension when compared to each other.

The result agree with other researches.[14] A decline in glucose homeostasis and the emergence of hyperglycemia are dependent on progressive b-cell failure, which may account for the significant correlation found between the baseline plasma glucose level and the development of type 2 diabetes. There was a highly significant elevation in irisin in hypertension patients compared with patients with T2DM ($p = 0.05$), as presented in Table 2, so according to this, an increase in the measurement of irisin had a good prognosis.

A paired sample T-test was applied to compare the insulin, HOMA IR, SBP, and DBP between hypertension and type 2 diabetes mellitus groups. Measurements and interaction with patient groups as presented in Table 3. Accordingly, there was a significant difference between the insulin, HOMA IR, and SBP measurements in hypertension and type 2 diabetes mellitus and no significant difference in DBP between patient groups.

The result showed that serum asprosin increase significantly in patient with hypertension more than

Table 3. Irisin and other parameters levels between study groups.

Groups Parameters	Type 2 diabetes mellites group	Hypertension group	*p*-value
Asprosin (ng/mL)	7.25 ± 1.58	9.35 ± 1.30	<0.05
Irisin (ng/mL)	355.78 ± 15.47	205.18 ± 30.40	<0.05
Insulin (mmol/L)	10.58 ± 1.57	5.55 ± 0.47	<0.05
HOMA IR	4.88 ± 0.69	2.73 ± 0.89	<0.05
SBP (mmHg)	136.54 ± 6.78	150.27 ± 5.47	<0.05
DBP (mmHg)	75.45 ± 7.41	80.17 ± 8.44	NS

T2DM patients, while some research has looked into the connection between asprosin and type 2 diabetes, more proof is required to fully understand how asprosin contributes to the development of T2DM for two main reasons. First, changes in ethnicity typically impact adipokine levels. The findings of research done in various populations indicate that when adipokines are investigated in various populations, mixed results may be found. Furthermore, there have been discrepancies in the findings of earlier research concerning the relationship between asprosin and fat metabolism.[15] Many physiologically active substances, including adipokines, are secreted by adipose tissue. According to earlier research, the severity of acute coronary syndrome and unstable angina is linked to asprosin, a novel form of adipokine released by white adipose tissue.[16]

The result is agreement with other research[17] shown that aspersin levels in the blood are noticeably higher in CVD patients for the first time. The severity of the patients' disease was found to be significantly correlated with their asprosin levels by researchers with other research.[18] On the other hand, in a five-year follow-up cohort study, with other research[9] found that elevated asprosin levels protected patients with dilated cardiomyopathy against adverse cardiac events. Individuals with type 2 diabetes were more likely to have adverse clinical outcomes if their asprosin levels were lower. The authors postulated that asprosin directly protects cardiomyocytes by increasing mitochondrial respiration in response to hypoxia.[19] Excessive energy consumption is converted to fat by adipocytes, leading to obesity, aberrant adipokine levels, insulin resistance, dyslipidemia, and a higher chance of adverse cardiovascular events.[20] An increase in obesity results in higher levels of asprosin release, which increases glucose synthesis.[21] Conversely, asprosin levels in overweight/obese people were successfully lowered by aerobic exercise training and bariatric surgery.[22] Also result found elevated in asprosin in T2DM which agreement with another research[23] that found individuals with type 2 diabetes mellitus may have dysregulated asprosin secretion by white adipose tissue, which causes asprosin concentrations in these patients to be pathologically elevated.

Irisin is the myokine that has the ability to lower body weight, boost energy expenditure, and ultimately enhance insulin sensitivity. Currently, mice are the subjects of a large number of irisin studies, with relatively few human studies being done.[24] Our findings are in agreement with another research,[25] which found that the levels of serum irisin in hypertension were significantly higher than those in the T2DM patients. It was hypothesized that irisin elevation in hypertensive patients could be a feedback mechanism to preserve homeostasis in reaction to oxidative stress and inflammation associated with hypertension. Further research is necessary to uncover unknown are the fundamental mechanisms behind this function. Furthermore, high blood pressure can lead to clinical findings related to hypertension, including stroke, peripheral arterial disease, chronic kidney disease, and coronary heart disease. The mechanism behind these results for irisin is still unknown. Nonetheless, some studies indicate that the onset of insulin resistance and type 2 diabetes may be associated with a drop in serum irisin levels.[26]

A positive significant correlation ($P < 0.05$) was found in Table 4. between Irisin and Asprosin with BMI, FBS, TG, insulin, and HOMA IR where no significant correlation ($P > 0.05$) was observed with other parameters in Type 2 diabetes mellites. Also, a positive significant association was exhibited between Irisin and Asprosin with BMI, cholesterol, triglyceride, insulin in hypertension group as shown in Table 4.

The findings also support the positive correlations between irisin and HDL cholesterol and BMI, IR, and FBS that have been reported in earlier studies.[26] Based on the correlation between irisin levels and body mass index (BMI), we developed suitable models to demonstrate that irisin remains linked to insulin resistance even after adjusting for BMI. In addition to being linked to other, as of yet undiscovered mechanisms, such as less efficient metabolism and/or direct effects on MetS risk factors, this suggests that irisin is associated with increased BMI or adiposity, which would attenuate effect estimates.[27] The findings in humans may indicate that irisin is secreted at a higher baseline because of the increased levels of muscle and adipose tissue in obesity, or they may indicate that

Table 4. Correlation between study parameters and Irisin and Asprosin.

	Irisin				Asprosin			
	T2DM		Hypertension		T2DM		Hypertension	
	r value	*P* value	*r* value	*P* value	*r* value	*P* value	*r* value	*P* value
BMI (kg/m^2)	0.393**	0.001	0.299**	0.001	0.326**	0.001	0.370**	0.001
FBS (mg/dL)	0.396*	0.011	0.457	0.218	0.857**	0.001	0.220**	0.001
Cholesterol (mmol/L)	0.049	0.859	0.388**	0.013	0.243	0.210	0.325*	0.033
Triglycerides (mmol/L)	0.061	0.596	0.389**	0.001	0.331*	0.038	0.319*	0.031
HDL (mmol/L)	0.025	0.949	0.155	0.471	0.124	0.721	0.176	0.421
LDL (mmol/L)	0.023	0.696	0.027	0.920	0.547	0.150	0.034	0.724
Insulin (μ IU/mL)	0.315**	0.025	0.016*	0.031	0.150	0.451	0.195	0.680
HOMA-IR	0.256	0.947	0.02	0.578	0.258*	0.021	0.232*	0.001

Table 5. Area under the curve for parameters Asprosin and Irisin.

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Lower Bound	Confidence Interval Upper Bound
Asprosin	.828	.095	.016	.641	1.000
Irisin	.948	.049	.000	.853	1.000

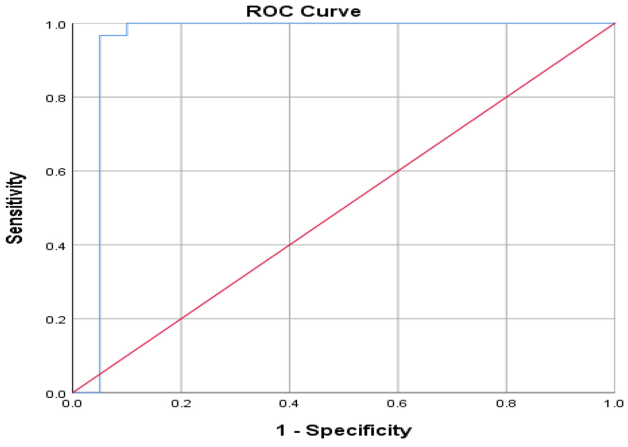

Fig. 1. ROC curve of Irisin of the study participants.

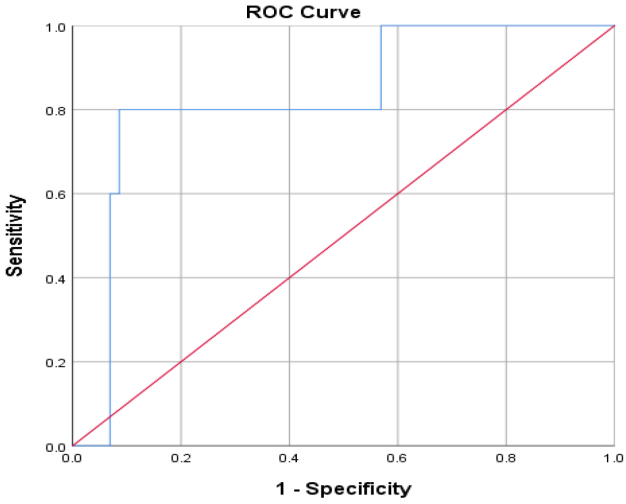

Fig. 2. ROC curve of Asprosin of the study participants.

irisin levels are increased compensatory to reverse the effects of obesity, the MetS, and/or irisin resistance, similar to the well-established relationship between leptin resistance and depression, depending on how irisin promotes metabolism. [28] Asprosin level and BMI were found to be positively correlated in the current study, which is consistent with findings from earlier research by with other research. [29] Nonetheless, there is a strong contradiction in the link between asprosin and obesity. In patients with type 2 diabetes and hypertension, asprosin showed a significant positive correlation with FBS, HOMA-IR, and TG. Similar results were reported by with other research. [30]

Receiver operator characteristics (ROC)

Receiver operator characteristics (ROC) curve used to assess the optimal diagnosis irisin and Asprosin level for in hypertension and T2DM as shown in Fig. 1, Fig. 2 and Table 5.

Conclusion

Finally, these results suggest that asprosin may have a role in the pathophysiology of both hypertension and T2DM, and that treating insulin resistance and dyslipidemia in these patients may be worthwhile. Additionally, considering asprosin as a potential biomarker, as indicated by the ROC curve analysis, highlights its diagnostic potential. To delve deeper into the subject, further investigation into the underlying mechanisms by which asprosin influences these conditions is essential. Understanding these mechanisms could provide insights into novel therapeutic targets or diagnostic strategies.

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Authors' declaration

- Conflicts of Interest: None.
- We hereby confirm that all the figures and tables in the manuscript are ours. Furthermore, figures and images, that are not ours, have been included with the necessary permission for re-publication, which is attached to the manuscript.
- No animal studies are present in the manuscript.
- Authors signed on ethical consideration's approval.
- Ethical Clearance: The project was approved by the local ethical committee at AL- Nahrain University, Baghdad, Iraq.

Authors' contribution statement

S. I. A. performed the acquisition of data, analysis, interpretation, drafting the manuscript. While F. M. K., Q. I. H., F. I. A. and L. I. A. conducted the analysis, design interpretation, revision and proofreading of the manuscript. While F. M. A. conducted the diagnosis of patients.

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العلاقة بين الاسبروسين والايريسين في المرضى العراقيين المصابين بالسكري النوع الثاني و ارتفاع ضغط الدم

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الخلاصة

يعتبر مرضى السكري أكثر عرضة للإصابة بارتفاع ضغط الدم بمرتين من غير المصابين بالسكري. الأسبروسين هو هرمون بروتيني يحتوي على 140 حمض أميني، تفرزه الأنسجة الدهنية البيضاء استجابة للصيام. عندما يتم تنشيط مسار أدينوسين الأدينوزين الحلقي G، مسار بروتين كيناز-A، فإنه يسبب زيادة مستحثة بالصيام في إطلاق الجلوكوز الكبدي في مجرى الدم. يتم إنتاج الإيريسين، وهو ببتيد يتكون من 112 حمضًا أمينيًا، عن طريق شق بروتين في غشاء البلازما لإطلاق مادة شحمية. يعد تعزيز استقلاب الدهون والجلوكوز، إلى جانب الخصائص المضادة للالتهابات ومضادات الأكسدة، من بين التأثيرات المفيدة للإيريسين. الغرض من هذا البحث هو تحديد وظيفة الأسبروزين وارتباطه بالإيريسين لدى الأفراد الذين يعانون من ارتفاع ضغط الدم ومرض السكري من النوع الثاني. شملت الدراسة 60 عينة، تتراوح أعمارهم بين 35-65 عاماً، مقسمة إلى مجموعتين حسب التشخيص: مرضى السكري من النوع الثاني ومرضى ارتفاع ضغط الدم، تم جمعها من مستشفى اليرموك/بغداد/العراق، للفترة من حزيران-شباط/2023. تم قياس بعض العوامل الكيموحيوية لجميع مجموعات الدراسة والتي تشمل: قياس نسبة الجلوكوز في الصيام والدهون بطريقة الأكسدة الأنزيمية. باستخدام عملية إنزيمية، تم قياس الأسبروسين والأنسولين والإيريسين باستخدام مقايسة الامتصاص المناعي المرتبط بالإنزيم. أخيرًا، تم حساب التوازن-تقييم النموذج-مقاومة الأنسولين وفقًا للصيغة المحددة. تظهر النتيجة ارتفاعًا ملحوظًا في مؤشر كتلة الجسم وWHR وإيريسين وFBS وHOMA-IR. كما أن هناك علاقة إيجابية بين الإيريسين والأبروسين مع مؤشر كتلة الجسم، FBS، والأنسولين. أخيرًا، كان لمستويات الأسبروسين والإيريسين قيمة تشخيصية عالية لكل من ارتفاع ضغط الدم والسكري من النوع الثاني، وفقًا لمنحنى ROC. من هذه النتائج، من الممكن تقييم درجة ارتفاع ضغط الدم ومرض السكري من النوع الثاني باستخدام مستوى الأسبروزين أو الإيريسين كعوامل تشخيصية.

الكلمات المفتاحية: اسبروسين، مرض السكري، ارتفاع ضغط الدم، مقاومة الأنسولين، إيريسين، السكري النوع الثاني.