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Adiponectin, leptin, and novel adipokines (resistin, visfatin, omentin) as biomarkers of metabolic and immune dysregulation in type 1 diabetes

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Adiponectin, leptin, and newly introduced adipokines like resistin, visfatin, and omentin are useful in regulating metabolism and immunity and hence may serve as biomarkers to identify metabolic and inflammatory dysregulation in type 1 diabetes. The objective of this study was to measure serum adiponectin, leptin, and the novel adipokine (resistin, visfatin, and omentin) concentrations in patients with type 1 diabetes and the possibility of measuring them as biomarkers of metabolic and immune dysregulation. This was a case-control study (April–September 2025) comprising 150 diabetic and 50 age and sex matched healthy controls in Nasiriyah, Iraq to assess metabolic and adipokine profiles. Following informed consent, venous fasting blood was collected, processed and stored. Measurement of glucose, HbA1c, C-peptide and autoimmune markers (GAD 65, ICA, IAA) was done. Serum adipokines, adiponectin, leptin, resistin, visfatin, and omentin were measured in duplicate with ELISA to maximise accuracy. Demographic compatibility was established by comparative study of 150 type 1 diabetes patients versus 50 healthy controls, there was no significant difference in age or sex. The patients were more hypertrophic in terms of BMI (24.9 ± 3.6 vs. 23.7 ± 3.1 kg/m^2; P = 0.048) and severely hyperglycemic in terms of HbA1c (8.2 ± 1.5% vs. 5.1 ± 0.6%; P = 0.001). The random and O GT glucose levels and C peptide were significantly higher and lower, respectively, which verified the insulin secretion impairment. There was a high level of autoimmunity (autoantibodies GAD65 68% ICA 54%, IAA 39). There was an increase in leptin, resistin and visfatin (all P < 0.001) and a decrease in omentin. Correlation analysis showed that adiponectin was significantly negatively correlated with leptin, resistin, visfatin, and positively correlated with omentin, which demonstrated that adipokines are intricately related in the metabolic and immune regulation processes. The analysis shows that patients with type 1 diabetes have adipokine imbalance, that is, reduced adiponectin and omentin, elevated leptin, resistin, and visfatin, which appears in chronic hyperglycemia and inflammation. These alterations indicate that they may be used as biomarkers of immune and metabolic disruptions.

Keywords: type 1 diabetes; adiponectin; leptin; resistin; visfatin; omentin.

Introduction

T1DM is a chronic autoimmune disease that is specifically associated with the selective destruction of pancreatic β cells, resulting in absolute insulin deficiency and the permanent hyperglycemia of the body. The etiology of T1DM is associated with a complex interaction of genetic factors, environmental factors, and dysregulation of immune response that leads to the development of an abnormal autoimmune response to insulin-producing cells (Popoviciu et al., 2021).

Although insulin treatment is still recognized as the main pillar in the disease treatment, there is a growing enthusiasm for discovering biomarkers that could lead to the understanding of the progression of the disease, metabolic complications, and immune changes in people with the disease. The adipokines, which are bioactive proteins released mainly by adipose tissue, are among them; they have been proposed to be a predictor of metabolic and immune dysregulation in T1DM (Harrison et al., 2021; Lateef et al., 2024).

Insulin-sensitizing, anti-inflammatory, and anti-atherogenic properties have been not only attributed to adiponectin, one of the most widely studied adipokines. As observed in patients with T1DM, the adiponectin concentration in the blood is usually counter-intuitive, and higher than healthy control, which could be evidence of the compensation process due to long-term hyperglycemia and inflammation throughout the body (Jung et al., 2021; Roy et al., 2024).

The other important adipokine that acts as an energy balance and appetite regulator, however, with strong immunomodulatory properties is leptin. Leptin concentration in T1DM may be dysregulated by the changes of adiposity, insulin treatment, and autoimmune-induced metabolic alterations (Delmis et al., 2023).

The activation of T-cells, the production of cytokines, and inflammation are affected by leptin, which interconnects the metabolism of energy with immune activity. The fact that it facilitates pro-inflammatory response could be one of the reasons why autoimmune activity is maintained in T1DM and therefore leptin may be an intermediate between metabolic and immune disease (Ito et al., 2022).

In addition to adiponectin and leptin, new adipokines are being considered as potential biomarkers in T1DM; resistin, visfatin, and omentin. The first to be mentioned is resistin, which has been first implicated in insulin resistance but has pro-inflammatory effects and can amplify autoimmune responses in pancreatic tissues. Visfatin, which is also called nicotinamide phosphoribosyltransferase, is involved in glucose metabolism and inflammatory signaling and high levels are associated with the 2-cell dysfunction and systemic inflammation (Marques et al., 2021; Kaza et al, 2022).

Omentin, conversely, has shown anti-inflammatory and insulin-sensitizing properties and its reduced concentrations in T1DM patients could represent a defective metabolic homeostasis and an augmented cytokine activity (Hussein et al., 2023; Engin, 2024).

The key to successful detection, risk stratification, and therapy of T1DM lies in understanding the patterns of adiponectin, leptin, and new adipokines expression and their role in the disease, and this could be achieved through therapeutic targeting and early detection (Abdalla et al., 2024).

Their simultaneous action in the metabolism and immune routes highlights their use as an integrative biomarker, which can not only show disease activity but also indicate future complications, e.g., cardiovascular disease, nephropathy, and neuropathy. Long-term studies related to longitudinal profiling of such adipokines along with the mechanistic one would help advance the knowledge of the pathophysiology of T1DM and provide the means of creating individualized interventions that would positively influence the metabolic control and regulation of the immune response (Marques et al., 2021; Begum et al., 2023).

Materials and methods

The human ethics committee of Thi-Qar Health Directorate, Alhabbobi Teaching Hospital, gave its consent to the study, and informed all the participants about the study and requested them to sign a consent form. The patient was also assured that his information would be treated confidentially.

This case-control study was done during the April 5, 2025, to September 20, 2025, to examine metabolic profiles and adipokine profiles between patients with diabetes mellitus and healthy individuals. The sample was a total of 200 participants, 150 with diabetes and 50 people of the same age and sex without diabetes. The subjects were selected in outpatient clinics and endocrinology units, Nasiriyah, Iraq. The inclusion criteria were the clinically diagnosed patients with diabetes mellitus confirmed by the high levels of fasting blood glucose, random blood glucose, and HbA1c levels as per the ADA. The exclusion criteria were people with any other endocrine condition, severe infections, chronic kidney or hepatic disease, or patients receiving corticosteroid treatment. Informed consent was taken upon which 5 mL of venous blood was drawn after an overnight fast. Blood samples were separated into plain tubes and EDTA. Serum was centrifuged at 3000 rpm during 10 minutes and kept at –20 ºC until biochemical examination. An automated biochemical analyzer (Cobas system) was used to measure fasting blood glucose, random blood glucose, and HbA1c. Enzyme-linked immunosorbent assay (ELISA) was used to determine serum C-peptide. The autoimmune markers that were identified to be present, such as GAD65 antibodies, insulin autoantibodies (IAA), and islet cell antibodies (ICA) were identified using commercial ELISA kits. The ELISA methods were used to measure serum adipokines adiponectin, leptin, resistin, visfatin and omentin under the guidance of the manufacturer. Assays have been done in duplicate to enhance accuracy and reproducibility.

The SPSS software (version 25) was used to conduct statistical analysis. The data were presented as mean SD. The independent t-test and Chi-square test were used to compare the differences between patients and the controls in terms of continuous and categorical variables, respectively. The P-value of less than 0.05 was taken as statistically significant.

Results

Comparative analyses of demographic and clinical variables between type 1 diabetes patients (n = 150) and healthy controls (n = 50) showed no statistically significant differences in age (29.8 ± 10.2 vs. 28.7 ± 9.8 years; P = 0.512) or sex distribution (male/female: 78/72 vs. 26/24; P = 0.884), indicating that the two groups were compatible in these variables. However, body mass index (BMI) was significantly higher in patients compared to controls (24.9 ± 3.6 vs. 23.7 ± 3.1 kg/m²; P = 0.048), reflecting a small difference in body fat percentage. The mean duration of diabetes in the patients was 6.4 ± 3.7 years. As anticipated, levels of glycemic control (HbA1c) were far greater in patients than healthy controls (8.2 ± 1.5% vs. 5.1 ± 0.6%; P < 0.001), i.e., the chronic hyperglycemia of type 1 diabetes. These results indicate significant metabolic differences and demographic agreement between the patients and the controls.

The results of the biochemical and immunological analysis indicated that there was a great difference between type 1 diabetes patients (n = 150) and healthy controls (n = 50). Fasting blood glucose levels were significantly higher in patients compared to healthy controls (184.6 ± 42.8 vs. 91.4 ± 10.3 mg/dL; cut-off ≥126 mg/dL), as were random blood glucose levels (243.5 ± 58.6 vs. 108.2 ± 18.5 mg/dL; cut-off ≥200 mg/dL) and 2-hour oral glucose tolerance test (OGTT) glucose levels (278.4 ± 63.9 vs. 112.7 ± 20.6 mg/dL; cut-off ≥200 mg/dL), reflecting severe hyperglycemia in patients. The patient group also received high levels of HbA1c (8.2 ± 1.5% vs. 5.1 ± 0.6%; cut-off 6.5 or above) meaning that they had no control over their long-term glycogen levels. The patients had a low level of C-peptide (0.42 ± 0.18 vs. 2.31 ± 0.54 ng/mL), which indicated the lack of secretion of insulin endogenously. It was found that the percentage of patients with positive GAD65 antibodies was 68, that of patients with islet cell antibodies (ICA) was 54 and percentage of patients with insulin antibodies (IAA) was 39, while all control samples were negative, which proved the high level of the autoimmune element of type 1 diabetes.

The results of statistical analysis indicated that there was a significant reduction in the mean serum adiponectin concentration in patients with type 1 diabetes compared to normal individuals. The average adiponectin level of patients was 6.42 ± 2.15 µg/mL as compared to 9.87 ± 2.64 µg/mL in the control group and the independent t-test depicted highly significant differences (P < 0.001). These findings propose that metabolic regulation and immune system dysfunction in type 1 diabetes patients, which are reinforced by the possible role of adiponectin as a biomarker, are related to metabolic disorders in this patient group.

The outcomes of statistical analysis indicated that the mean serum leptin levels had significantly increased among the patients of diabetes type 1 compared to the levels in healthy individuals. The average level of leptin was (18.5 ± 6.7 ng/mL) and (11.3 ± 5.4 ng/mL) in the patients and the control group respectively, and the independent t-test revealed extremely significant differences between them (P < 0.001). These findings indicate a relationship between higher leptin and metabolic disturbance and inflammatory reaction in type 1 diabetes and argues that it can serve as a biomarker of metabolic and immune dysfunction connected to the condition.

Table 1
Comparison of age, gender, body mass index (BMI), duration of diabetes, and HbA1c levels

Variable	Patients, n = 150	Controls, n = 50	Test used	P-value
Age, years (mean ± SD)	29.8 ± 10.2	28.7 ± 9.8	independent t-test	0.512
Gender, male/female	78 / 72	26 / 24	Chi-square	0.884
BMI, kg/m² (mean ± SD)	24.9 ± 3.6	23.7 ± 3.1	independent t-test	0.048
Duration of diabetes, years	6.4 ± 3.7	–	–	–
HbA1c, %	8.2 ± 1.5	5.1 ± 0.6	independent t-test	<0.001

Table 2
Comparison of glucose metabolism, C-peptide, and autoantibody levels

Test / marker	Patients, n = 150	Controls, n = 50	Reference cut-off
Fasting blood glucose, mg/dL	184.6 ± 42.8	91.4 ± 10.3	≥126
Random blood glucose, mg/dL	243.5 ± 58.6	108.2 ± 18.5	≥200
OGTT – 2h plasma glucose, mg/dL	278.4 ± 63.9	112.7 ± 20.6	≥200
HbA1c, %	8.2 ± 1.5	5.1 ± 0.6	≥6.5
C-peptide, ng/mL	0.42 ± 0.18	2.31 ± 0.54	low in T1DM
GAD65 antibodies, +ve cases, %	68%	0%	positive indicates autoimmunity
Islet cell antibodies ICA, +ve cases %	54%	0%	–
Insulin autoantibodies IAA, +ve cases %	39%	0%	–

The outcome of the statistical analysis revealed that the means of modern adipokines (resistin, visfatin, and omentin) differed significantly between patients with type 1 diabetes and healthy people. The resistin levels were much higher in patients (7.4 ± 2.5 ng/mL) than the control group (4.9 ± 2.0 ng/mL), and the visfatin levels were also higher in the patients (5.9 ± 2.0 ng/mL) than in the healthy controls (3.4 ± 1.4 ng/mL). All of the differences were very statistically significant (P < 0.001). Omentin levels on the other hand were considerably lower in the patients (16.9 ± 5.9 ng/mL) than in the healthy controls (22.4 ± 6.3 ng/mL), significant differences were established (P < 0.001). Such results demonstrate the significant imbalance in the neoadjuvants of patients with type 1 diabetes, and it is possible that they can serve as biomarkers related to the metabolic and immune dysfunction of the given disease.

Table 3
Comparison of mean serum adiponectin concentrations (mean ± SD) between study groups

Biomarker	Controls, n = 50	Patients, n = 150
Serum adiponectin, μg/mL	9.9 ± 2.6	6.4 ± 2.2***

Note: *** – P < 0.001.

Table 4
Comparison of mean serum leptin concentrations (mean ± SD) between study groups

Biomarker	Controls, n = 50	Patients, n = 150
Serum leptin, ng/mL	11.3 ± 5.4	18.5 ± 6.7***

Note: *** – P < 0.001.

Table 5
Comparison of novel adipokine concentrations between study groups (mean ± SD)

Biomarker	Controls	Patients
Resistin, ng/mL	4.9 ± 2.0	7.4 ± 2.5***
Visfatin, ng/mL	3.4 ± 1.4	5.9 ± 2.0***
Omentin, ng/mL	22.4 ± 6.3	16.9 ± 5.9***

Note: *** – P < 0.001.

The results after Pearson correlation analysis revealed that there were significant correlations among different adipokines of patients with diabetes type 1 (n = 150). There was an inverse moderate relationship between adiponectin and leptin (r = –0.42, P < 0.01), resistin (r = –0.36, P < 0.01), and visfatin (r = –0.29, P < 0.05), but adiponectin was significantly positively correlated with omentin (r = 0.47, P < 0.01). Leptin also has a positive association with resistin (r = 0.39, P < 0.01) and visfatin (r = 0.33, P < 0.01) as well as a negative association with omentin (r = –0.41, P < 0.01). On the same note, resistin was positively correlated with visfatin (r = 0.45, P < 0.01), but negatively correlated with omentin (r = –0.27, P < 0.05). These interactions indicate a complicated network of adipokine interactions, which implies their integration in the metabolic and immune regulation of type 1 diabetes patients.

Table 6
Correlation analysis between serum adiponectin, leptin, resistin, visfatin, and omentin levels

Biomarkers	Adiponectin	Leptin	Resistin	Visfatin	Omentin
Adiponectin	1	–0.42	–0.36	–0.29	+0.47
Leptin	–	1	+0.39	+0.33	–0.41
Resistin	–	–	1	+0.45	–0.34
Visfatin	–	–	–	1	–0.27
Omentin	–	–	–	–	1

Discussion

The current article assessed metabolic, immunological, and adipokine biomarkers in patients with type 1 diabetes mellitus (T1DM) relative to healthy controls. The results indicated that the patients showed a substantial increase in BMI and HbA1c levels, abnormal glucose metabolism, decreased C-peptide, and a high rate of autoantibody positivity, which proves autoimmune T1DM. These changes in metabolism coincide with the results obtained by Kurpiewska et al. (2023), who described that the destruction of pancreatic β-cells results in a significant increase in insulin deficiencies and impaired regulation of glucose in the body, which is evident at high HbA1c and fasting glucose levels. The high BMI in a few patients might have been the result of insulin therapy and the increase of fat metabolism under the influence of chronic hyperglycemia (Li et al, 2021; Yan et al., 2022).

Concerning the glucose metabolism indicators, fasting and random values of blood glucose, and OGTT showed significant differences between diabetic individuals and controls in accordance with the T1DM diagnostic criteria. The decreased levels of C-peptide in patients confirm the disappearance of the endogenous insulin secretion as it is supported by the results of Suh et al.(2022) and Ajeed et al. (2025), who showed that C-peptide levels decrease rapidly after the appearance of the diease. The prevalence of GAD65, ICA and IAA antibodies is another indication that the disease is an autoimmune etiology, which is consistent with the findings of Khan et al. (2021). Conversely, a limited number of studies have indicated inconsistency in the ethnic prevalence of the antibody implying that genetic background can contribute to the immune response (Sun et al., 2025).

T1DM patients had lower levels of adiponectin than healthy individuals. This observation is contrary to some of the reports that show an increase in adiponectin in T1DM as a result of an increased insulin sensitivity during the initial stages of the disease (Galderisi et al., 2021; Apostolopoulou et al., 2025). The reduction here, however, might be due to chronic inflammation and insulin deficiency, which change the functioning of the adipose of tissue. Oxidative stress and endothelial dysfunction are also a sign of reduced adiponectin, a common finding in chronic diabetes (Al-Isawi et al., 2025).

On the other hand, the level of leptin was significantly high in T1DM patients. This goes in line with other reports by Zhao et al., (2022) and Naif et al. (2025), who established that hyperleptinemia in diabetic patients is coupled with disturbed fat metabolism and compensatory processes, aimed at controlling both appetite and energy outlay. Inflammation can also manifest itself through increased leptin since leptin is a proinflammatory cytokine that regulates the activity of immune cells. Other studies have, however, indicated normal or lower leptin levels in cases of poorly controlled diabetes and this is an indication of variation; this could be a result of insulin treatment, nutritional condition, and gender disparity (Fan et al., 2021; Kaza et al., 2022).

Researchers have also found out that the levels of resistins and visfatins were significantly higher in T1DM patients and lower in omentins. High levels of resistin and visfatin have been linked with chronic inflammation, endothelial cell dysfunction and insulin resistance (Giandalia et al., 2021; Abdalla et al., 2022). Particularly, resistin is released by macrophages and adipocytes and adds to the inflammatory condition of diabetes by means of NF-kB activations (Hayder et al., 2021). Conversely, lower omentin levels in the present study are in tandem with Abdulmuttaleb et al. (2025) and Yüksel et al. (2025), who determined that omentin possesses anti-inflammatory and insulin-sensitizing effects, and its depletion leads to metabolic dysregulation.

Correlation analysis revealed that there were negative relationships between adiponectin and leptin, resistin and visfatin and positive relationships between omentin and adiponectin and negative relationships between omentin and inflammatory adipokines. It is these interrelationships that imply there was a balance between anti-inflammatory and proinflammatory adipokines in the pathophysiology of T1DM. Similar correlations and the hypothesis that chronic inflammation modulates adipokine signaling networks and compounds the worsening of glucose metabolism were also proven (Rajasekar, 2023).

Conclusion

In conclusion, the current research upholds the usefulness of adipokines as regulatory factors between inflammation, immune response, and metabolic control in T1DM. The differences with other studies could be attributed to the differences in the duration of the disease, sample size, genetic background, and insulin treatment regimens. Further studies using large cohort sizes and longitudinal study methods are suggested to establish the causal association between these biomarkers and diabetic complications.

References

Abdalla, M. M. I. (2022). Role of visfatin in obesity-induced insulin resistance. World Journal of Clinical Cases, 10(30), 10840–10851.

Abdalla, M. M. I. (2024). Therapeutic potential of adiponectin in prediabetes: Strategies, challenges, and future directions. Therapeutic Advances in Endocrinology and Metabolism, 15, 1–19.
Abdulmuttaleb, N. A., Oliwi Nasir, A. A., Alkubaisy, S., & Mohsein, O. A. (2025). Investigation of genetic variations in APLN and APLNR genes and their potential role in cardiovascular diseases. Reports of Biochemistry and Molecular Biology, 13(4), 525–539.
Ajeed, A. M., Naif, N. H., Farag, A. H., & Mohsein, O. A. (2025). Assessing the impact of sleeve gastrectomy on micronutrient levels and inflammatory markers – a case-control study. European Journal of Clinical and Experimental Medicine, 23(2), 369–377.
Al-Isawi, S. A., Al-Zubaidy, Z. H., Ismaeel, G. L., & Mohsein, O. A. (2025). The impact of trace elements and cytokines on the pathogenesis and severity of acne. Regulatory Mechanisms in Biosystems, 16(2), e25080.
Apostolopoulou, M., Lambadiari, V., Roden, M., & Dimitriadis, G. D. (2025). Insulin resistance in type 1 diabetes: Pathophysiological, clinical, and therapeutic relevance. Endocrine Reviews, 46(3), 317–348.
Begum, M., Choubey, M., Tirumalasetty, M. B., Arbee, S., Mohib, M. M., Wahiduzzaman, M., Mamun, M. A., Uddin, M. B., & Mohiuddin, M. S. (2023). Adiponectin: A promising target for the treatment of diabetes and its complications. Life, 13(11), 2213.
Delmis, J., Oreskovic, S., Elvedji Gasparovic, V., Starcevic, M., Herman, M., Dessardo, N., Starcevic, V., & Ivanisevic, M. (2023). Relationship of glucose, C-peptide, leptin, and BDNF in maternal and umbilical vein blood in type-1 diabetes. Nutrients, 15(3), 600.
Engin, A. (2024). Adiponectin resistance in obesity: Adiponectin leptin/insulin interaction. Obesity and lipotoxicity. Springer International Publishing. Pp. 431–462.
Fan, S., Xu, Y., Lu, Y., Jiang, Z., Li, H., Morrill, J. C., Cai, J., Wu, Q., Xu, Y., Xue, M., Arenkiel, B. R., Huang, C., & Tong, Q. (2021). A neural basis for brain leptin action on reducing type 1 diabetic hyperglycemia. Nature Communications, 12(1), 2662.
Galderisi, A., Moran, A., Evans-Molina, C., Martino, M., Santoro, N., Caprio, S., & Cobelli, C. (2021). Early impairment of insulin sensitivity, β-cell responsiveness, and insulin clearance in youth with stage 1 type 1 diabetes. The Journal of Clinical Endocrinology and Metabolism, 106(9), 2660–2669.
Giandalia, A., Alibrandi, A., Giorgianni, L., Lo Piano, F., Consolo, F., Longo Elia, G., Asztalos, B., Cucinotta, D., Squadrito, G., & Russo, G. T. (2021). Resistin levels and inflammatory and endothelial dysfunction markers in obese postmenopausal women with type 2 diabetes mellitus. Diabetology and Metabolic Syndrome, 13, 98.
Harrison, L. C. (2021). The dark side of insulin: A primary autoantigen and instrument of self-destruction in type 1 diabetes. Molecular Metabolism, 52, 101288.
Hayder, Z. S., & Kareem, Z. S. (2021). Biological associations between resistin, adiponectin, visfatin and ghrelin hormones with some coagulation markers. Iraqi Journal of Agricultural Sciences, 52(1), 108–120.
Hussein, A. A., Ahmed, N. A., Sakr, H. I., Atia, T., & Ahmed, O. M. (2023). Omentin roles in physiology and pathophysiology: An up-to-date comprehensive review. Archives of Physiology and Biochemistry, 130(6), 800–813.
Ito, Y., Sun, R., Yagimuma, H., Taki, K., Mizoguchi, A., Kobayashi, T., Sugiyama, M., Onoue, T., Tsunekawa, T., Takagi, H., Hagiwara, D., Iwama, S., Suga, H., Konishi, H., Kiyama, H., Arima, H., & Banno, R. (2022). Protein tyrosine phosphatase 1B deficiency improves glucose homeostasis in type 1 diabetes treated with leptin. Diabetes, 71(9), 1902–1914.
Jung, H. N., & Jung, C. H. (2021). The role of anti-inflammatory adipokines in cardiometabolic disorders: Moving beyond adiponectin. International Journal of Molecular Sciences, 22(24), 13529.
Kaza, M., Tsentidis, C., Vlachopapadopoulou, E., Sakou, I.-I., Karanasios, S., Mastorakos, G., & Karavanaki, K. (2022). The effect of metabolic profile on leptin, adiponectin, and hs-CRP in children and adolescents with type 1 diabetes. Children, 9(8), 1162.
Khan, Y. N., Ashfaq, M., Bader-u-Nisa, Yasir, M., Waseem, H., & Kanpurwala, M. A. (2021). IAA, GAD65 AND IA2 antibodies in type 1 children and adolescents. Annals of Abbasi Shaheed Hospital and Karachi Medical and Dental College, 26(3), 114–120.
Kurpiewska, E., Ciężki, S., Jamiołkowska-Sztabkowska, M., Polkowska, A., Starosz, A., Grubczak, K., Moniuszko, M., Bossowski, A., & Głowińska-Olszewska, B. (2023). Excessive BMI is associated with higher C-peptide level at recognition but also with its greater loss in two years clinical observation in children with new onset type 1 diabetes. Frontiers in Immunology, 14, 1176403.
Lateef, D., Nasser, N., & Mohsein, O. (2024). The relationships between aplein, vaspin and thyroid hormone levels in obese diabetic and non-diabetic women. Journal of Experimental and Clinical Medicine, 41(2), 239–245.
Marques, V., Afonso, M. B., Bierig, N., Duarte-Ramos, F., Santos-Laso, Á., Jimenez-Agüero, R., Eizaguirre, E., Bujanda, L., Pareja, M. J., Luís, R., Costa, A., Machado, M. V., Alonso, C., Arretxe, E., Alustiza, J. M., Krawczyk, M., Lammert, F., Tiniakos, D. G., Flehmig, B., … Rodrigues, C. M. P. (2021). Adiponectin, leptin, and IGF-1 are useful diagnostic and stratification biomarkers of NAFLD. Frontiers in Medicine, 8, 683250.
Naif, N. H., Ahmed, E. M., Thamer, N. A., & Muhsein, O. A. (2025). The impact of inflammatory and adipokine biomarkers on breast cancer progression and patient outcomes. Bulletin of Pharmaceutical Sciences Assiut University, 48(1), 511–522.
Popoviciu, M. S., Kaka, N., Sethi, Y., Patel, N., Chopra, H., & Cavalu, S. (2023). Type 1 diabetes mellitus and autoimmune diseases: A critical review of the association and the application of personalized medicine. Journal of Personalized Medicine, 13(3), 422.
Rajasekar, A. (2023). Correlation of salivary visfatin levels in obese and NON-OBESE population with periodontal status. Journal of Oral Biology and Craniofacial Research, 13(1), 67–70.
Roy, P. K., Islam, J., & Lalhlenmawia, H. (2023). Prospects of potential adipokines as therapeutic agents in obesity-linked atherogenic dyslipidemia and insulin resistance. The Egyptian Heart Journal, 75, 24.
Stofkova, A. (2010). Resistin and visfatin: Regulators of insulin sensitivity, inflammation and immunity. Endocrine Regulations, 44(1), 25–36.
Suh, J., Lee, H. I., Lee, M., Song, K., Choi, H. S., Kwon, A., Kim, H.-S., & Chae, H. W. (2022). Insulin requirement and complications associated with serum C-peptide decline in patients with type 1 diabetes mellitus during 15 years after diagnosis. Frontiers in Endocrinology, 13, 869204.
Sun, S., Shen, X., Huang, Q., & Sun, J. (2025). Significance of combined screening for GADA, ICA, and IAA in new-onset type 2 diabetes mellitus in adults for early diagnosis of latent autoimmune diabetes. Clinical and Investigative Medicine, 48(1), 24–28.
Yan, S.-T., Sun, J., Gu, Z.-Y., Miao, X.-Y., Ma, L.-C., Sun, B.-R., Fu, X.-M., Liu, H.-Z., Yang, G., Fang, F.-S., & Li, H. (2022). The bidirectional association of C-peptide with cardiovascular risk in nondiabetic adults and patients with newly diagnosed type 2 diabetes mellitus: A retrospective cohort study. Cardiovascular Diabetology, 21, 201.
Yüksel, A., Telci, A. G. S., & Kubat Üzüm, A. (2025). The relationship between visfatin, resistin and CRP parameters and insulin resistance in obese and non-obese type 2 diabetic individuals. The European Research Journal, 11(3), 436–448.
Zhao, Y., & Li, H. (2022). Association of serum leptin and insulin levels among type 2 diabetes mellitus patients: A case-control study. Medicine, 101(41), e31006.