**Open Access** **Baghdad Science Journal** **P-ISSN: 2078-8665**
**Published Online First: May, 2023** **2024, 21(1): 117-123** **E-ISSN: 2411-7986**

DOI: https://doi.org/10.21123/bsj.2023.8119

# Relationship between serum Nesfatin-1, Adiponectin, Resistin Concentration, and Obesity with Type 2 Diabetes Mellitus

***Baydaa Ahmed Abed*[1]** ***Layla Othman Farhan*[* 2]** ***Ashgan Slman Dawood*[2]**

[1] National Diabetes Center, Mustansiriyah University, Baghdad, Iraq.
[2]Department of Chemistry, College of Science for Women, University of Baghdad, Baghdad Iraq.
[*]Corresponding author: laylaof_chem@csw.uobaghdad.edu.iq
E-mail addresses: baydaaahmed@yahoo.com, ashgnsdchem@csw.uobaghdad.edu.iq

Received 16/11/2022, Revised 28/1/2023, Accepted 29/1/2023, Published Online First 20/5/2023, Published 01/1/2024



## Abstract:

Diabetes mellitus caused by insulin resistance is prompted by obesity. Neuropeptide Nesfatin-1 was identified in several organs, including the central nervous system and pancreatic islet cells. Nesfatin-1 peptide appears to be involved in hypothalamic circuits that energy homeostasis and control food intake. Adiponectin is a plasma collagen-like protein produced by adipocytes that have been linked to the development of insulin resistance (IR), diabetes mellitus type 2 (DMT2), and cardiovascular disease (CVD). Resistin was first identified as an adipose tissue–specific hormone that was linked to obesity and diabetes. The aim of this study was to estimate the relationship between human serum nesfatin-1, adiponectin, resistin concentration, and obesity with T2DM. The results show a significant increase in serum neisfatin-1 and resistin levels in the obese diabetic group compared to the non-obese diabetic group. Adiponectin levels had a highly significant decrease in the obese diabetic group compared to the non-obese group and obese control group. Serum Nesfatin-1 and some variables, there was a significant positive correlation with (BMI, insulin, HOMA-IR), and serum resistin had a significant positive correlation with (BMI, WC, TG, insulin, HOMA-IR).On the other hand, there was a negative correlation between serum adiponectin and (BMI, TG, and HOMA-IR). The present results suggest that nesftin-1 may have a role in controlling food intake as well as the development of IR in obese patients.

**Keywords**: Adiponctin, HOMA-IR, Nesfatin-1, Resistin, Type 2 diabetes mellitus T2DM.

## Introduction:

Diabetes is characterized as a condition that happens on account of the absence of insulin or nearness of factors contradicting the activity of insulin, bringing about an expansion (hyperglycemia) in blood glucose level [1]. The purpose behind the profound benefit of fat tissue-derived hormones lies in the increasing incidence of human population over the World. It is believed that the existence of obesity generously builds the danger of related comorbidities, for example, dyslipidemia, diabetes, insulin resistance, hypertension, and others [2]. The close connection between obesity and its related complications has been entrenched, however, the exact instrument straightforwardly connecting one to the other is not clear, as yet [3]. Obesity is generally related to hyperinsulinemia and IR [4], it's a noteworthy risk factor for the improvement of DMT2 and CVD [5]. Whilst free fatty acids discharged to adipose tissue have for some time been involved in the advancement of this obesity-related complications[6].

Nesfatin-1 is an 82-amino-acid peptide generated from a hypothalamus protein precursor. It regulates appetite stimulation, body weight, food intake, and the energy balance in the body[7]. Nesfatin-1controls intracellular glucose metabolism and increase insulin sensitivity [8]. Adiponectin is a specific cytokine in adipose tissue that has an important effect against anti-inflammatory and insulin resistance and found to protect against metabolic diseases [9]. Adiponectin controls glycogenesis, glucose uptake, and fatty acid oxidation all of which are linked to diabetes etiology[10]. Resistin is a hormone produced by adipose tissue that inhibits glucose homeostasis in rats by resisting insulin action. That, in turn, causes

to develop (DMT2) [11]. Resistin, an individual from the newfound group of cysteine rich secretory proteins, has been portrayed as a result of fat tissue, taking part in the inflammation in mice, adipogenesis and pathogenesis of insulin resistance [12]. The aim of this study was to high light the relationship between human serum nesfatin-1, adiponctin, resistin concentration and obesity with DMT2.

## Materials and Methods:

### Subjects

The study was designed cross sectional study. The study included 90 subjects, divide into two groups, patients and healthy control. Sixteen patients (31 males, 29 females) and 30 control (17 males, 13 females). In this study were collected during the period from (Septemper2021 to December 2021 at National Diabetic Center/ Mustansiriyah University.

### Specimen Collection and Analysis:

After twelve hours of fasting, a blood sample was taken from venous to measure fasting blood glucose (FBG) , lipid profile (TG ,TC, HDL,LDL,VLDL),insulin, nesfatin-1,resistin and adiponectin levels .The tallness and weight had been measured. Overweight and corpulent Individuals those with BMI, individually, Z25 or30 kg/m2 and Z30 kg/m2. The IR has been calculated from the HOMA-IR, as indicated by distributed algorithm [13]. The biochemistry measurement was done by using Kenza( 240TX) (Biolabo) instrument and (Biolabo) kit (FBG, TG ,TC, HDL) . The HbA1c was measured by using the Tosoh analyzer (HLC-723GX). The insulin, adiponctin, resistin, nesfatin-1 concentration were determined by using ELISA kit (Al-Shkairate Establishments, Jordan).

To determine if variables had a normal distribution, the Anderson-Darling test was performed. If they did, then the mean and standard deviation were applied. When there are differences between more than two groups (and if there are no major outliers and the distribution is normal), one method of analyzing these differences is with an ANOVA. The significance level was set at a $P \leq 0.05$. Explanation of the regression analysis using a scatter plot, r (correlation coefficient or standardized beta is a representative of magnitude and direction of the relationship). Positive signs reflect direct relationships, whereas negative signs suggest the opposite.

### Results:

The results of this study demonstrateda high significant difference in (FBG, HbA1c) ($p<0.001$), and (TC, TG) ($p<0.05$) in diabetic patients group is comparison with thecontrol group. While high significant difference in Nesfatin-1 and resistin levels (4.96±1.03), (14.89 ± 5.4) in diabetic patients compared to that of control group (1.71±0.80) (9.6 ± 3.39) $p<0.05$. There was a significant decrease in adiponectin levels in diabetic group when compared to that of control group (9.79 ± 1.97) vs. (15.63 ± 2.57, $p< 0.01$), as presented in Table 1.

**Table 1. Comparison between patients and control.**

| Characteristics | Control No: 30 (mean± SD) | Diabetic patients: No 60(mean± SD) | P value |
|---|---|---|---|
| Age, years | 48.41 ± 8.45 | 48.31± 8.42 | N.S |
| BMI (kg/m$^2$) | 32.12 ± 2.8 | 33.15 ± 3.02 | N.S |
| WC (cm) | 110.76 ± 8.9 | 113.98 ± 12.79 | N.S |
| FBG (mg/dl) | 100.7 ± 41.23 | 187.24 ± 43.13** | <0.01 |
| HbA1c % | 5.51 ± 0.56 | 9.78 ± 1.56** | <0.01 |
| TC (mg/dl) | 191.45 ± 32.47 | 240.73 ± 55.8* | <0.05 |
| TG (mg/dl) | 131.88 ± 33.98 | 159.55 ± 41.3* | <0.05 |
| HDL-C (mg/dl) | 43.06 ± 2.58 | 37.39 ± 3.35* | <0.05 |
| LDL-C (mg/dl) | 126.18 ± 23.75 | 130.52 ± 31.09 | N.S |
| Insulin (µIU/ml) | 9.01 ± 2.48 | 14.43 ± 3.23* | <0.05 |
| HOMA-IR | 4.92 ± 1.9 | 7.53 ± 2.9** | <0.01 |
| Nesfatin-1(ng/ml) | 1.71±0.80 | 4.96 ± 1.03* | <0.05 |
| Resistin (ng/ml) | 9.6 ± 3.39 | 14.89 ± 5.4* | <0.05 |
| Adiponectin (ng/ml) | 15.63 ± 2.57 | 9.79 ± 1.97** | <0.01 |

The data was evaluated using either the mean (mean) , and the standard deviation (± SD ).

Table. 2, showed a significant increase in serum nesfatin-1and resistinlevel in the obese diabetic group compared to non-obese diabetic group (5.96 ± 2.51) (16.87 ± 3.4) vs. (3.89 ± 2.01) (11.6 ± 2.4, $p< 0.05$) and a higher significant increase compared to obese control group (5.96 ± 2.51) (16.87 ± 3.4) vs. (1.89±0.84) (10.04 ± 2.14, $p<0.01$). There was a highly significant decrease in

adiponectin levels in obese diabetic group compared to no obese group and obese control group (7.98 ± 1.81) vs. (12.63 ± 2.17, p<0.01; (7.98 ± 1.81) vs. (11.7 ± 2.01) respectively.

Moreover, there was a significant increase in serum insulin, cholesterol, triglycerides, and HOMA-IR in obese diabetic group compared to non- obese diabetic group, and obese control group (16.43 ± 3.91 vs. 12.01 ± 3.48, p<0.05; 16.43 ± 3.91 vs. 11.64 ± 3.46, p<0.05), (266.33 ± 37.78 vs. 198.54 ± 30.76, p<0.05; 266.33 ± 37.78 vs. 199.14 ± 40.54, p<0.05), (186.95 ± 42.3 vs. 128.68 ± 33.65, p<0.01; 186.95 ± 42.3 vs. 152.52 ± 32.85, 0.05), (8.53 ± 2.01 vs. 6.12 ± 1.33, p<0.05; 8.53 ± 2.01 vs. 5.23 ± 1.93, p<0.01) respectively. On the other hand, there was a significant decrease in serum HDL- cholesterol in obese diabetic group compared to non-obese (35.67 ± 3.24 vs. 39.90 ± 2.37, p<0.05), and the data are presented in Table 2**.**

**Table 2. Comparison between obese, non-obese patients, and obese control group.**

| Characteristics | Obese –control No.17 (mean± SD**)** | Non-obese DM No:15 | Obese DM No:15 (mean± SD**)** |
|---|---|---|---|
| Age, years | 48.05 ± 7.32 | 48.76 ± 8.84 | 48.24 ± 8.53 |
| BMI (kg/m$^2$) | 33.98 ± 3.95 | 26.02 ± 2.9 | 36.05 ± 3.12$^{**}$ |
| WC (cm) | 114.04 ± 10.68 | 89.76 ± 7.9 | 117.06 ± 13.79$^{**}$ |
| FBG (mg/dl) | 99.39 ± 34.51 | 182.7 ± 58.23 | 198.24 ± 43.73$^{\#\#}$ |
| HbA1c % | 5.8 ± 1.3 | 8.91 ± 1.78 | 9.89 ± 1.03$^{\#\#}$ |
| TC (mg/dl) | 199.14 ± 40.54 | 198.54 ± 30.76 | 266.33 ± 37.78$^{*\#}$ |
| TG (mg/dl) | 152.52 ± 32.85 | 128.68 ± 33.65 | 186.95 ± 42.3$^{**\#}$ |
| HDL-C (mg/dl) | 36.01 ± 2.58 | 39.90 ± 2.37 | 35.67 ± 3.24$^{*}$ |
| LDL-C (mg/dl) | 127.85 ± 30.46 | 129.68 ± 21.97 | 131.52 ± 31.56 |
| Insulin (µIU/ml) | 11.64 ± 3.46 | 12.01 ± 3.48 | 16.43 ± 3.91$^{\#*}$ |
| HOMA-IR | 5.23 ± 1.93 | 6.12 ± 1.33 | 8.53 ± 2.01$^{*\#\#}$ |
| Nesfatin-1 (ng/ml) | 1.89 ± 0.84 | 3.89 ± 1.31 | 5.96 ± 2.51$^{*\#\#}$ |
| Adiponectin (ng/ml) | 11.7 ± 2.01 | 12.63 ± 2.17 | 7.98 ± 1.81$^{\#\#**}$ |

*P<0.05 is considered significant,**P<0.01 is a highly significant when compared to obese non-obese patients.
$^{\#}$ Significant at p < 0.05, and $^{\#\#}$ significant at p < 0.001 as compared DM to obese control group.

Table 3 showed the Pearson Chi-square correlation coefficient among serum nesfatin-1 and some variables, there was a significant positive correlation with (BMI, insulin, HOMA-IR), and significant negative correlation with age. Serum resistin had a significant positive correlation with (BMI, WC, TG, insulin, HOMA-IR), and significant negative correlation with HDL. On the other hand there was negative correlation between adiponectin and (BMI, TG and HOMA-IR), and a positive correlation between adiponectin and HDL in obese patients.

**Table 3. Correlation analysis between Nesfatin-1, Resistin, Adiponectin, and variables in obese patients.**

| Characteristics | Nesfatin-1 R | Resisting R | Adiponectin R |
|---|---|---|---|
| **Age** | -0.089 | -0.098 | +0.190 |
| **BMI** | 0.299$^{*}$ | 0.393$^{*}$ | -0.312$^{*}$ |
| **WC** | 0.199 | 0.298$^{*}$ | -0.185 |
| **FBG** | 0.206 | 0.130 | -0.198 |
| **HbA1$_c$** | 0.171 | 0.182 | -0.121 |
| **TC** | 0.163 | 0.184 | -0.096 |
| **TG** | 0.405 | 0.308$^{*}$ | -0.289$^{*}$ |
| **HDL-C** | 0.206 | -0.485$^{**}$ | 0.431$^{**}$ |
| **LDL-C** | 0.103 | 0.094 | -0.028 |
| **Insulin** | 0.425$^{*}$ | 0.398$^{*}$ | -0.150 |
| **HOMA-IR** | 0.627** | 0.592$^{**}$ | -0.401$^{**}$ |

*Correlation is significant at the 0.05
**Correlation is a highly significant at the 0.01

## Results:

The results of this study shows that a significant higher resistin levels in diabetic patients compared to control group (14.89 ± 7.4 vs 9.6 ± 3.39, p<0.05). On the other hand, there was a significant decrease in adiponectin levels in diabetic group compared to control group (9.79 ± 1.97 vs 15.63 ± 2.57 , p< 0.01 ), as shown in table 1.

Table2 show a significant increase in serum resistin level in obese diabetic group compared to

non-obese diabetic group (16.87 ± 3.4 vs 11.6 ± 2.4, p< 0.05) and a higher significant increase compared to obese control group (16.87 ± 3.4 vs 10.04 ± 2.14, p<0.01). On the other hand, there was a highly significant decrease in adiponectin levels in obese diabetic group compared to non- obese group and obese control group (7.98 ± 1.81 vs 12.63 ± 2.17, p<0.01; 7.98 ± 1.81 vs , 0.01,
respectively).

Moreover, there was a significant increase in serum insulin, cholesterol, triglycerides, and HOMA-IR in obese diabetic group compared to non-obese diabetic group, and obese controlgroup (16.43 ± 3.91 vs 12.01 ± 3.48, p<0.05; 16.43 ± 3.91 vs 11.64 ± 3.46, p<0.05), (266.33 ± 37.78 vs 198.54 ± 30.76, p<0.05; 266.33 ± 37.78 vs 199.14 ± 40.54, p<0.05), (186.95 ± 42.3 vs1 28.68 ± 33.65, p<0.01; 186.95 ± 42.3 vs 152.52 ± 32.85, 0.05), (8.53 ± 2.01 vs 6.12 ± 1.33, p<0.05; 8.53 ± 2.01 vs 5.23 ± 1.93, p<0.01) respectively. On the other hand, there was a significant decrease in serum HDL-cholesterol in obese diabetic group compared to non-obese (35.67 ± 3.24 vs 39.90 ± 2.37, p<0.05), as shown in table 2.

Table 3 show the pearson Chi-square correlation coefficient between serum resistin and some variables and metabolic syndrome components, there was a significant positive correlation with (BMI, WC, TG, insulin, HOMA-IR), and significant negative correlation with HDL. On the other hand there was negative correlation between serum adiponectin and BMI, TG, and HOMA-IR. And there was positive correlation between adiponectin and HDL in obese patient

## Discussion:

Obesity is a serious cause of death and illness across the world [14]. Under situations of poor glucose metabolism, nesfatin-1 is said to have an anti-hyperglycemic action[15]. In reaction to hyperglycemia, it may also operate in the brain to up regulate insulin sensitivity and enhance insulin release in beta cells. Food intake was also discovered to be inhibited by Nesfatin-1 in the central nervous system[16]. Nesfatin-1 has also been proven to increase lipid metabolism and have anti-inflammatory properties in several studies [17]. Obesity is a common cause of T2DM inflammation, eating disorder, and insulin action dysfunction [18]. Anther studies have discovered higher circulatinesfatin-1 levels in DMT2 as a result of lowering blood glucose, improving lipid metabolism, restricting food consumption, and decreasing inflammation [19].The results in this study is also agreed with the results done by Zhang Z, ET *al*.[20], In individuals with Type 2 diabetes, nesfatin-1 levels were shown to be higher. In diabetic groups with severely impaired glucose metabolism, HOMA-IR was shown to be substantially higher than in control groups [6]. The results in this study is also agreed with the results done by Anwar GM *et al*. [21], who found that obese people had considerably greater nesfatin-1 levels than healthy people. Prepronesfatin was shown to be localized in the islets of both rats and mi in a recent research, suggesting that prepones fat in-derived peptides may play a role in insulin production and glucose metabolic. The Result in Table 3, demonstrated a direct significant relationship between nesfatin-1 and BMI .which agreed with Başar Ö et al [22].

Resistin is a tiny secreted protein generated mostly by mononuclear cells in the peripheral blood, macrophages in humans, and bone marrow cells in humans. Resistin pro-inflammatory function has been demonstrated in a variety of illnesses, including DMT2, CVD, kidney disease, and rheumatoid arthritis[23]. In the present work serum resistin levels are higher significant in diabetic and obese diabetic patient when compared to normal subjects. Serum resistin, delivered by adipose tissue, in Individuals have higher fat content the resistin available at greater circulating levels. Notwithstanding, the greater generation of resistin by the non-fat fraction of adipose tissue [24], May be because of absence of organization found in our overweight patients, in whom the adipocyte part of the fat tissue may be more noteworthy concerning the stromo vascular fraction [25]. The commonness of obesity has increased significantly lately and is related with a few chronic illnesses, for example, coronary artery disease, hypertension, metabolic disorder, and, specifically T2DM. One of the features of these diseases is IR [26]. Adipose tissue saving body fat reserves, privileged insights a few peptide hormones and cytokines that assume essential parts in vitality homeostasis and are captured in several chronic diseases [27]. Adiponectin is a fat tissue specific cytokine has a protective role against IR and anti-inflammatory activity and protect against metabolic diseases [28]. We additionally found in this study two important discovering. first, its agree with anther studies that the low plasma adiponectin concentration is related with DMT2 and obesity and through different ethnic groups with marked differences in propensity for obesity, [29] and T2DM indicated for hypoadiponectinemia. Second, the results showed that the fasting insulinemia and insulin sensitivity plasma are more closely related to adiponectin concentration than to glycemia and adiposity, in patients with fatness and DMT2 in large part attributable to IR and hyperinsulinemia which

propose that the hypo adioponectinemia. Past studies agreement with our study demonstrated that is negatively correlation between plasma adiponectin concentration with BMI, triglyceride and HOMA-IR [30].

Adiponectin, an anti-inflammatory adipocyte, is copiously emitted by fat tissue and straightforwardly sharpens body tissues to insulin. A diminishing in levels of adiponectin because of hereditary or hormonal factors has been emphatically embroiled in the improvement of IR, DMT2, metabolic disorder, and other chronic diseases that are related with corpulence [6]. Although anther studies have exhibited a significant relationship among IR in fatness DMT2 Individuals and patients with other fatness related diseases with adiponectin levels [31]. This study, affirming a portion of the distributed discoveries, recognized a significant opposite relationship between fasting adiponectin levels and IR in obese DMT2 patients. Accordingly, steady with the discoveries of past reports, the aftereffects of this investigation demonstrated that adiponectin level can be utilized as a prescient list for IR in fat patients with DMT2[24].

Although adipose tissue was principle wellspring of adiponectin, inquire about discoveries recommend that the blood level of adiponectin is lessened in obese or T2DM who have huge stores of fat tissue [32-36]. Lessening of adiponectin levels has been proposed to assume a focal part in the expanded rate of T2DM and IR disorder [37-40]. In obese animal models the Adiponectin levels have reduced, which decreases insulin sensitivity and induce DMT2 start [32, 41-42].

Moreover, in this study a significant opposite relationship was observed between fasting glucose levels and adiponectin. That refer to an increase in fasting glucose level has connected with a decrease in adiponectin level, which is the main determinant of T2DM in patients with obesity.

## Conclusion:

Our findings suggest that nesfatin-1 may have a role in the control of food intake as well as the development of IR in obese patients. Serum resistin raised in response to hyperglycemia and further in response to hyperinsulinemia. These study demonstrates that fatness and T2DM have connected with low plasma adiponectin and demonstrate that the level of hypoadiponectinemia is all the more firmly identified with the level of IR and hyperinsulinemia than the level of glucose and adiposity. As a biomarker for DMT2 related IR and obesity, adiponectin can be used.

## Acknowledgement:

The authors express are sincere appreciation to the Department of Chemistry, College of Science for Women, University of Baghdad, Jadwiga, and Baghdad, Iraq for supporting this study.

## Authors' Declaration:

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are mine ours. Besides, the Figures and images, which are not mine ours, have been given the permission for re-publication attached with the manuscript.
- Authors sign on ethical consideration's approvalyes
- Ethical Clearance: The project was approved by the local ethical committee in University of Baghdad.

## Authors' Contribution Statement:

B. A.A.'s role in this research was collecting samples and, analyzing the results. L.O.F. designed, analyzed, proof read, and presented ideas of the research A. S. D.'s role in this research was to do analytics and proof editing.

## References:

1. Zafar M, Irum S, Khan L, Sadia H, Roshan S, Rahman TU, et al. Prevalence of Diabetes Mellitus in Hepatitis C Patients in Wazirabad Tehsil of Gujranwala District of Pakistan: Hepatitis C in Diabetic patients. Baghdad Sci J. 2020; 17(4): 1154-1159. https://doi.org/10.21123/bsj.2020.17.4.1154
2. Ormazabal V, Nair S, Elfeky O, Aguayo C, Salomon C, Zuñiga FA. Association between insulin resistance and the development of cardiovascular disease. Cardiovasc. Diabetol. 2018; 17(1): 1–14. https://doi.org/10.1186/s12933-018-0762-4
3. Schwartz MW, Seeley RJ, Zeltser LM, Drewnowski A, Ravussin E, Redman LM, et al. Obesity pathogenesis: An Endocrine Society Scientific Statement. Endocr Rev. 2017; 38(4): 267–296. https://doi.org/10.1210/er.2017-00111
4. Lomteva S V, Shkurat TP, Bugrimova ES, Zolotykh OS, Alexandrova AA, Karantysh G V. Violation of the Hormonal Spectrum in Polycystic Ovaries in Combination with Insulin Resistance. What is the Trigger: Insulin Resistance or Polycystic Ovary. Baghdad Sci J. 2022;19 (5):990-998. https://doi.org/10.21123/bsj.2022.6317
5. Cercato C, Fonseca FA. Cardiovascular risk and obesity. Diabetol Metab Syndr. 2019; 11(74): 1-15. https://doi.org/10.1186/s13098-019-0468-0.
6. Abozaid YJ, Zhang X, Mens MM, Ahmadizar F, Limpens M, Ikram MA, Rivadeneira F. Plasma circulating microRNAs associated with obesity, body fat distribution, and fat mass: the Rotterdam Study Int

J Obes. 2022; 46(1): 2137–2144. https://doi.org/10.1038/s41366-022-01227-8 .
7. Alotibi MN, Alnoury AM, Alhozali AM. Serum nesfatin-1 and galanin concentrations in the adult with metabolic syndrome: relationships to insulin resistance and obesity. Saudi Med J. 2019; 40(1):19-25. https://doi.org/10.15537/smj.2019.1.22825
8. Mogharnasi M, Taheri Chadorneshin H, Papoli-Baravati SA, Teymuri A. Effects of upper-body resistance exercise training on serum nesfatin-1 level, insulin resistance, and body composition in obese paraplegic men. Disabil Health J. 2019; 12(1):29–34. https://doi.org/10.1016/j.dhjo.2018.07.003
9. Choi HM, Doss HM, Kim KS. Multifaceted physiological roles of adiponectin in inflammation and diseases. Int J Mol Sci. 2020; 21(4): 1219. https://doi.org/10.3390/ijms21041219
10. Dong Y, Huang G, Wang X, Chu Z, Miao J, Zhou H. Meta-analysis of the association between adiponectin SNP 45, SNP 276, and type 2 diabetes mellitus. PLoS One. 2020; 15(10): 2-23. https://doi.org/10.1371/journal.pone.0241078
11. Tripathi D, Kant S, Pandey S, Ehtesham NZ. Resistin in metabolism, inflammation, and disease. FEBS J. 2020; 287(15): 3141-3149. https://doi.org/10.1111/febs.15322
12. Lin Q, Johns RA. Resistin family proteins in pulmonary diseases. Am J Physiol Cell Mol Physiol. 2020; 319(3): 422–434. https://doi.org/10.1152/ajplung.00040.2020
13. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: Insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985; 28(7):412-419. https://doi.org/10.1007/BF00280883
14. Varagic J, Desvigne-Nickens P, Gamble-George J, Hollier L, Maric-Bilkan C, Mitchell M, et al. Maternal morbidity and mortality: Are we getting to the "heart" of the matter? J Women's Heal. 2021; 30(2): 178–86. https://doi.org/10.1089/jwh.2020.8852
15. Su Y, Zhang J, Tang Y, Bi F, Liu J-N. The novel function of nesfatin-1: anti-hyperglycemia. Biochem Biophys Res Commun. 2010; 391(1): 1039–1042. https://doi.org/10.1016/j.bbrc.2009.12.014
16. Tekin T, Cicek B, Konyaligil N. Regulatory peptide nesfatin-1 and its relationship with metabolic syndrome. Eurasian J Med. 2019; 51(3): 280-284. https://doi.org/10.5152/eurasianjmed.2019.18420
17. Dong J, Xu H, Xu H, Wang P, Cai G, Song H, et al. Nesfatin-1 stimulates fatty-acid oxidation by activating AMP-activated protein kinase in STZ-induced type 2 diabetic mice. PLoS One. 2013; 8(12):1-8: https://doi.org/10.1371/journal.pone.0083397
18. Farhan LO, Taha EM, Farhan AM. A Case control study to determine Macrophage migration inhibitor, and N-telopeptides of type I bone collagen Levels in the sera of osteoporosis patients. Baghdad Sci J. 2022, 19(4): 848-854. https://doi.org/10.21123/bsj.2022.19.4.0848.
19. Algül S, Dinçer E. The essential role of nesfatin-1 as a biological signal on the body systems. Kastamonu Med J. 2021;1(4):113–8. https://doi.org/10.51271/KMJ-0028
20. Zhang Z, Li L, Yang M, Liu H, Boden G, Yang G. Increased plasma levels of nesfatin-1 in patients with newly diagnosed type 2 diabetes mellitus. Exp Clin Endocrinol diabetes. 2012; 120(02): 91–5. https://doi.org/10.1055/s-0031-1286339
21. Anwar GM, Yamamah G, Ibrahim A, El-Lebedy D, Farid TM, Mahmoud R. Nesfatin-1 in childhood and adolescent obesity and its association with food intake, body composition and insulin resistance. Regul Pept. 2014; 188(10): 21–4. https://doi.org/10.1016/j.regpep.2013.12.001
22. Başar Ö, Akbal E, Köklü S, Koçak E, Tuna Y, Ekiz F, et al. A novel appetite peptide, nesfatin-1 in patients with non-alcoholic fatty liver disease. Scand J Clin Lab Invest. 2012; 72(6): 479–83. https://doi.org/10.3109/00365513.2012.699097
23. Huang J, Peng X, Dong K, Tao J, Yang Y. The association between insulin resistance, leptin, and Resistin and diabetic nephropathy in type 2 diabetes mellitus patients with different body mass indexes. Diabetes, Metab Syndr Obes Targets Ther. 2021; 24(14) 2357–65. https://doi.org/10.2147/DMSO.S305054.
24. Liu W, Zhou X, Li Y, Zhang S, Cai X, Zhang R, et al. Serum leptin, resistin, and adiponectin levels in obese and non-obese patients with newly diagnosed type 2 diabetes mellitus: a population-based study. Medicine. 2020; 99(6):1-7. https://doi.org/10.1097/MD.0000000000019052.
25. Palumbo Piccionello A, Riccio V, Senesi L, Volta A, Pennasilico L, Botto R, et al. Adipose micro-grafts enhance tendinopathy healing in ovine model: An in vivo experimental perspective study. Stem Cells Transl Med. 2021; 10(11): 1544–60. https://doi.org/10.1002/sctm.20-0496
26. Abass EAA, Abed BA, Mohsin SN. Study Of Lysyl Oxidase-1 And Kidney Function In Sera Of Iraqi Patients With Diabetic Nephropathy. Biochem. Cell. Arch. 2021; 21(1) :1129-1132. https://connectjournals.com/03896.2021.21.1129
27. Zhang S, Zhao J, Xie F, He H, Johnston LJ, Dai X, et al. Dietary fiber-derived short-chain fatty acids: A potential therapeutic target to alleviate obesity-related nonalcoholic fatty liver disease. Obes Rev. 2021; 22(11): e13316. https://doi.org/10.1111/obr.13316
28. Guo Q, Cao S, Wang X. Adiponectin Intervention to Regulate Betatrophin Expression, Attenuate Insulin Resistance and Enhance Glucose Metabolism in Mice and Its Response to Exercise. Int J Mol Sci. 2022; 23(18): 1-12. https://doi.org/10.3390/ijms231810630
29. Ameen EM, Mohammed HY. Correlation between Tumor Necrosis Factor–Alfa and Anti-tyrosine Phosphatase with Obesity and Diabetes Type 2. Iraqi J Sci. 2022; 63(8): 3322–3331. https://doi.org/10.24996/ijs.2022.63.8.7 .
30. Weyer C, Funahashi T, Tanaka S, Hotta K, Matsuzawa Y, Pratley RE, et al. Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and

hyperinsulinemia. J Clin Endocrinol Metab, 2001; 86(5): 1930–1935. https://doi.org/10.1210/jcem.86.5.7463
31. Hotta K, Funahashi T, Arita Y, Takahashi M, Matsuda M, Okamoto Y, et al. Plasma concentrations of a novel, adipose-specific protein, adiponectin, in type 2 diabetic patients. Arterioscler Thromb Vasc Biol. 2000; 20(6): 1595–1599. https://doi.org/10.1161/01.atv.20.6.1595
32. Hotta K, Funahashi T, Bodkin NL, Ortmeyer HK, Arita Y, Hansen BC, et al. Circulating concentrations of the adipocyte protein adiponectin are decreased in parallel with reduced insulin sensitivity during the progression to type 2 diabetes in rhesus monkeys. Diabetes. 2001; 50(5): 1126–1133. https://doi.org/10.2337/diabetes.50.5.1126 .
33. Zhang Y, Chen G. The Link of Nutrient Fluxes to Hepatic Insulin Resistance at Gene Expression. Arch Gastroenterol Res. 2020; 1(3): 52–60. https://doi.org/10.33696/Gastroenterology.1.011
34. Farhan LO. Determanation of Several Biochemical Parameters in Sera of Iraqi Patients with type 2 Diabetes. Baghdad Sci J. 2015; 12(2): 362-370. https://doi.org/10.21123/bsj.2015.12.2.362-370.
35. Farhan LO, Mustafa SA, Mubder NS. Effect of Pregnancy on Selenium, Cupper, Zinc and Others Biochemical Feacture. Baghdad Sci J. 2013; 10(4): 1182-1189. https://doi.org/10.21123/bsj.2013.10(4):1182-1189
36. Hamid GS, Allawi AA, Ghudhaib KK. Correlation of Pentosidine with Kidney Diseases in Iraqi Patients with Diabetic Nephropathy. Iraqi J Sci. 2021; 62(10):3436–42. https://doi.org/10.24996/ijs.2021.62.10.2.
37. Palit SP, Patel R, Jadeja SD, Rathwa N, Mahajan A, Ramachandran A V, et al. A genetic analysis identifies a haplotype at adiponectin locus: association with obesity and type 2 diabetes. Sci Rep. 2020; 10(1): 1–10. https://doi.org/10.1038/s41598-020-59845-z .
38. Ghaib ZJ, Ghudhaib KK, Mohsen FY. Assessment of Neuron Specific Enolase Level and some Related Biochemical Factors in Patients with Diabetic Peripheral Nerve Disorders. Indian J Forensic Med Toxicol. 2021; 15(3): 1494-1500 https://doi.org/10.24996/ijs.2021.62.10.2 .
39. Abed BA, Al-AAraji SB, Salman IN. Estimation Of Galanin Hormone In Patients With Newly Thyroid Dysfunction In Type 2 Diabetes Mellitus. Biochem. Cell. Arch. 2021; 21 (1): 1317-1321.
40. Khaleel FM, N-Oda N, A Abed B. Disturbance of Arginase Activity and Nitric Oxide Levels in Iraqi Type 2 Diabetes Mellitus. Baghdad Sci J. 2018;15(2):189–191. DOI: http://dx.doi.org/10.21123/bsj.2018.15.2.0189
41. Abed BA, Hamid GS. Evaluation of Lipocalin-2 and Vaspin Levels in In Iraqi Women with Type 2 Diabetes Mellitus. Iraqi J Sci. 2022;63(11):650–658. DOI: https://doi.org/10.24996/ijs.2022.63.11.3.
42. Al-Saad NNO, Khaleel FM, Al-Jumaili EFA. Purification and Characterization of Arginase from Serum of Healthy and Type II Diabetic Iraqi Patients. Indian J Forensic Med Toxicol. 2021; 15(3): 1194-1203. https://doi.org/10.37506/ijfmt.v15i3.15475.

# العلاقة بين مصل Nesfatin-1 و Adiponctin وتركيز Resistin والسمنة مع مرض السكري من النوع 2

بيداء احمد عبد [1] ليلى عثمان فرحان[2] اشكان سلمان داود [2]

[1] المركز الوطني لعلاج وبحوث السكري، الجامعة المستنصرية، بغداد، العراق.
[2] قسم الكيمياء، كلية العلوم للبنات، جامعة بغداد، بغداد، العراق.

**الخلاصة:**

داء السكري الناجم عن مقاومة الأنسولين هو الدافع وراء السمنة. تم تحديد بين نسفاتين -1 ، أديبونكتين في العديد من الأعضاء ، بما في ذلك الجهاز العصبي المركزي وخلايا جزرالبنكرياس . Nesfatin-1 ، له دور في دوائر التوازن التي تعمل على الطاقة الاستتبابية والتحكم في تناول الطعام .أديبونكتين هو بروتين شبيه بالكولاجين في البلازما تنتجه الخلايا الدهنية ويرتبط بتطور مقاومة الأنسولين (IR) ومرض السكري من النوع 2 (DMT2) وأمراض القلب والأوعية الدموية (CVD) . تم تحديد Resisting لأول مرة على أنه هرمون خاص بالأنسجة الدهنية مرتبط بالسمنة ومرض السكري. كان الهدف من هذه الدراسة هو تقدير العلاقة بين نسفاتين -1 ، أديبونكتين ، تركيز رسستين والسمنة معDMT2 . أظهرت النتائج زيادة معنوية في مستوى نسفاتين 1 ومستوى الريسيستين في مجموعة مرضى السكري البدينين مقارنة بمجموعة السكري غير البدينين. اظهرت مستويات الأديبونكتين انخفاضًا معنويًا للغاية في مجموعة مرضى السمنة مقارنة بالمجموعة غير البدناء ومجموعة التحكم في السمنة. نسفاتين 1 وبعض المتغيرات كانت له علاقة ارتباط موجبة مع ( BMI ، insulin، HOMA-IR ) كما وجد ارتباط معنوي موجب لمصلي المقاومة مع (BMI، WC، TG، insulin HOMA-IR ). بينما كان هناك ارتباط سلبي بين اديبونيكتين المصل مع (BMI, TG and HOMA-IR) .تشير نتائج الدراسة الحالية إلى أن nesfatin-1 قد يكون له دور في التحكم في تناول الطعام وكذلك تطوير مقاومة الانسولين في المرضى الذين يعانون من السمنة المفرطة.

**الكلمات المفتاحية:** ادبينوستين, مقاومة الانسولين ,نسفاتين 1,ريزيستين ,داء السكري النوع الثاني .