# Assessment of Insulin Resistance According to Degrees of Obesity Among Iraqis with Type 2 Diabetes

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## Abstract

Diabetes mellitus [DM] is a syndrome characterized by impaired carbohydrate metabolism. Obesity is a significant risk factor for type 2 diabetes mellitus [T2DM]. Obesity is defined as an excess of body fat. In obese individuals, adipose tissue releases more significant levels of non - esterified fatty acids, glycerol, hormones, pro -inflammatory cytokines, and other substances involved in establishing insulin resistance [IR]. The link between body mass index [BMI] and diabetes and IR is substantial. This study assessed the effect of obesity and insulin resistance in Iraqi diabetic patients. The objective is to assess the impact of IR on the degree of obesity in Iraqis with T2DM. We conducted a case-control study on 180 participants [ 99 female /81males] between the ages of 30 and 60, from December 1, 2021 , to February 28 , 2022. In addition, the subjects [control and Cases] were categorized based on their weight:average weight, overweight, obesity, and marked obesity.

All documented subjects provided informed consent to WHO guidelines. We examined the homeostatic model assessment of insulin resistance [HOMA-IR], HbA1c, fasting blood glucose [FBG], lipid profile, and BMI. The case group had significantly higher levels of FBG, HbA1c, cholesterol [CHO.], triglyceride [TG], low-density lipoprotein [LDL], and IR than the control group [P = 0.001], but there was no significant difference between the two groups in high- density lipoprotein [HDL] levels. Recent research shows that a higher BMI makes insulin less sensitive and worsens IR, leading to T2DM.

***Keywords**: obesity, BMI, insulin resistance, type 2 diabetes mellitus.*

## تقييم مقاومة الأنسولين حسب درجات السمنة بين العراقيين المصابين بداء السكري من النوع الثاني

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**الخلاصة**

يُعرَّف داء السكري بأنه متلازمة إكلينيكية تتميز باستقلاب غير طبيعي للكربوهيدرات. السمنة عامل خطر كبير لتطور داء السكري من النوع 2. لقياس السمنة ، التي تُعرَّف على أنها زيادة في دهون الجسم ، في الأفراد الذين يعانون من السمنة المفرطة ، تطلق



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الأنسجة الدهنية كميات متزايدة من الأحماض الدهنية غير المؤسترة ، والجليسرول ، والهرمونات ، والسيتوكينات المسببة للالتهابات ، وعوامل أخرى تشارك في تطوير مقاومة الأنسولين. مؤشر كتلة الجسم له علاقة قوية بمرض السكري ومقاومة الأنسولين. أجريت هذه الدراسة لتقييم تأثير السمنة ومقاومة الأنسولين لدى مرضى السكري العراقيين .الهدف هو تحديد تأثير مقاومة الأنسولين حسب درجات السمنة بين العراقيين المصابين بداء السكري من النوع 2. أجرينا دراسة حالة وضبط على 180 شخصًا [ 99 إناثًا / 81 ذكورًا] تتراوح أعمارهم بين 30-60 عامًا ، بما في ذلك المرضى الذين يزورون مختبر الدكتور علي منيب الربيعي / بغداد / العراق من الأول من كانون الأول [ ديسمبر] 2021 إلى نهاية شباط [ فبراير] 2022. كما تم تقسيم المفحوصين [ الضابطة والحالات] حسب وزنهم إلى متوسط الوزن وزيادة الوزن والسمنة والسمنة الملحوظة. تم الحصول على الموافقة المستنيرة من جميع الموضوعات الموثقة وفقًا لمعايير منظمة الصحة العالمية. تم قياس HOMA-IR و FBG وملف الدهون ومؤشر كتلة الجسم. كان هناك مستوى أعلى بشكل ملحوظ من FBS، HbA1c، .CHO، TG، LDL ، و IR في مجموعة الحالة مقارنة بمجموعة التحكم [ قيمة P <0.001] ، بينما لم يتم الحصول على فرق كبير بين مجموعات الدراسة فيما يتعلق HDL. تظهر النتائج الحديثة أن ارتفاع مؤشر كتلة الجسم يؤدي إلى انخفاض حساسية الأنسولين ودرجة أعلى من مقاومة الأنسولين ويساهم في تطور مرض السكري من النوع الثاني.

***الكلمات المفتاحية:*** *السمنة ، مؤشر كتلة الجسم ، مقاومة الأنسولين ، السكري من النوع الثاني .*

## Introduction

T2DM is a disease with hyperglycemia characteristic features resulting from the abolished response of insulin receptors to circulating insulin, customarily known as (IR) [1]. Insulin resistance (IR) means that ordinary quantities of insulin are not giving the normal response i.e., does not the use of insulin by cells is not enough to decrease triglyceride and glucose concentration. IR is a mixture of lifestyle factors [ reduced physical activity, diet ] and genetics [2] . The BMI is a reliable indicator of body fatness in humans and can be viewed as an alternative to direct measurements of body fat. BMI calculation is one of the most accurate approaches for assessing overweight and obesity in a population. It is inexpensive and straightforward for physicians and the general public to calculate because just height and weight are required [3]. This study intends to examine the link between lipid profile and BMI in individuals with type 2 diabetes mellitus in Iraq. Insulin's essential functions include inhibiting lipolysis in adipose tissue and enhancing lipoprotein lipase enzyme activity.

Obesity- related expansion of adipose tissue mobilizes free fatty acids [FFA] in circulation via the cAMP-dependent enzyme hormone-sensitive lipase. In addition, FFA is released in the tissues by the action of lipoprotein lipase from lipolysis of TG- rich lipoproteins [4]. The prevalence of obesity, metabolic syndrome, and its components is influenced by genetic susceptibility, physical activity, age, gender, amount and kind of food consumed, and body habitus [5]. HOMA-IR is a method for assessing a- cell function and insulin resistance [IR] [ Tung et al., 2020].



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**Materials and Methods**

A total of 180 subjects were collected (90) patients with type 2 diabetes mellitus (48 females, 42 male) and (90) healthy control group subgroup (39 males and 51 females), The age range of both groups was (30- 65 years). Also, the subjects (Control and Cases) were divided according to their weight to average weight, overweight, obesity, and marked obesity. The FBS, HbA1c, CH, TG, HDL, LDL, and VLDL levels were determined using the COBAS INTEGRA® 400 plus automatic biochemistry analyzer [ Roche/ Hetachi Diagnostics Ltd Company, Japan]. The COBAS INTEGRA® 400 plus automated biochemistry analyzer can perform colorimetric, immunoturbidimetric, and ion-selective tests.IR was determined by Homeostasis model assessment [HOMA] via equation as follows:

HOMA= { glucose [in mole/ L] × insulin [in micro U/mL]}/ 22.5

**Statistical Analysis**

The data were analyzed using statistical software for the accessible SPSS-24 [Statistical Packages for the Social Sciences - version 24]. The data were displayed using frequency, percentage, mean, and standard deviation.

**Results**

There were a total of 180 participants in this study. Table 1 displays the baseline characteristics of the subjects investigated. The persons with diabetes had a mean age of 90, while the controls had a mean age of 90.

There was a notable increase. Significantly higher levels of FBS, Hba1c, CHO., TG, LDL, a nd IR were seen in the case group compared to the control group [P 0.001]. However, there was no significant difference between the two groups' HDL values. As evidenced in the table (1).

**Table (1)**: The distribution of the study markers according to the study groups.

| Varibles | Groups | | P – value |
|---|---|---|---|
| | Control group | Case group | |
| | Mean ( ±SD) | Mean (±SD) | |
| **Fasting blood sugar (mmol / L)** | 5.4 (±0.4) | 9.8 (± 2.9) | < 0.001 |
| **HbAlc** | 4.3 (±0.6) | 8.3 (±2.5) | < 0.001 |
| **Cholesterol (mg/dl)** | 163.2(±21.1) | 208.5 (±54.0) | < 0.001 |



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| | | | |
|---|---|---|---|
| **Triglycerides (mg/dl)** | 78.1 (±19.7) | 216.5 (±85.9) | < 0.001 |
| **LDL (mg/d)** | 100.8 (±21.3) | 132.4 (±33.2) | < 0.001 |
| **HDL(mg/dl)** | 48.1 (±6.6) | 50.5 ( ±28.6) | < 0.001 |
| **VLDL(mg/dl)** | 18.1 ( ±8.3) | 36.9 (±20.7) | < 0.001 |
| **Fasting Insulin (mIU/L)** | 9.0 ( ±0.8) | 10.7(±4.4) | < 0.001 |
| **Insulin resistance** | 2.1 (±0.3) | 4.5 (±1.5) | < 0.001 |

The distributions of body mass index were drastically different between the two groups. While only 23.3% of individuals in the control group were obese, and 6.7% were severely obese, 38.9% of patients in the case group were obese, and 30% were severely obese table (2).

**Table (2):** Distribution of the irisin and adropin according to the study groups.

| **BMI** | **Groups** | | **Total** | **P – value** |
|---|---|---|---|---|
| | **Control group** | **Case group** | | |
| | **N [ % ]** | **N [ % ]** | | |
| **Normal** | 42 [46.7] | 12 [13.3] | 54 [30.0] | < 0.001 |
| **Overweight** | 21 [23.3] | 16 [17.8] | 37 [20.6] | < 0.001 |
| **Obesity** | 21 [ 23.3] | 35 [38.9] | 56 [ 31.1] | < 0.001 |
| **Markedobesity** | 6 [ 6.7] | 27[30/0] | 33 [ 18.3] | < 0.001 |

Normal-weight individuals showed a highly significant difference from the normal- weight individuals in the control group regarding fasting blood sugar, hemoglobin A1c, and insulin resistance, but there was no difference between the groups in terms of insulin. For obesity, fasting blood sugar, hemoglobin A1c, and insulin differed significantly between the case and control groups, while insulin resistance [IR] did not. Obese subjects in the case group were found to have significantly different FBS, HbA1c, insulin, and insulin resistance levels compared to the control group. There was a highly-significant difference regarding FBS, HbA1c, and insulin resistance between participants with marked obesity in the case group and those in the control group; except with insulin, there is a non- significant difference between the study groups. As demonstrated in table [3].



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**Table (3):** Distribution of FBS, HbA1c, insulin, and IR according to the BMI grade.

| Body mass index ( kg/m$^{2)}$ | Study groups | Investigations | | | |
|---|---|---|---|---|---|
| | | **FBS (mmol/L) Mean (±SD)** | **HbAlc Mean (±SD)** | **Insulin (mIU/L) Mean (±SD)** | **HOMA-IR Mean (±SD)** |
| **Normal weight** | **Case N=12** | 10.6 (3.1) | 8.1 (1.9) | 8.8 (1.4) | 4.1 (1.0) |
| | Control N=42 | 5.4 (0.3) | 4.2 (0.2) | 9.0 (0.6) | 2.2 (0.2) |
| **P – value** | | **0.001** | **0.001** | 0.424 | **0.001** |
| **Overweight** | Case N=16 | 0.8 (2.3) | 7.7 (2.4) | 11.6 (6.4) | 4.4 (2.2) |
| | Control N=21 | 5.4 (0.5) | 4.1 (0.1) | 8.9 (0.9) | 2.1 (0.3) |
| **P-value** | | **0.001** | **0.001** | **0.001** | 0.66 |
| **Obesity** | Case N=35 | 10.1 (3.4) | 8.3 (2.0) | 11.4 (5.3) | 4.8 (1.7) |
| | Control N=21 | 5.2 (0.4) | 4.8 (1.0) | 8.8 (0.4) | 2.0 (0.1) |
| **P-value** | | **0.001** | **0.001** | **0.034** | **0.001** |
| **Markid obesity** | Case N=27 | 9.8 (2.5) | 8.9 (3.2) | 10.0 (1.1) | 4.3 (0.8) |
| | Control N=6 | 5.4 (0.2) | 4.1 (0.8) | 10.2 (1.9) | 2.4 (0.5) |
| **P-value** | | **0.001** | **0.001** | 0.814 | **0.001** |

There was a highly significant difference regarding Cholesterol, Triglyceride, LDL, and VLDL between participants with normal weight within the case study and individuals in the control group, except with HDL that there is a non-significant difference between the study groups. There was a highly significant difference regarding CHO, TG, LDL, and VLDL between participants with



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overweight in the case study and individuals in the control group, except with HDL that there is a non-significant difference between the study groups. There was a highly significant difference regarding CHO, TG, LDL, and VLDL between participants with obesity in the case study and individuals in the control group, except with HDL that there is a non-significant difference between the study groups. There was a highly significant difference between TG, and VLDL participants with marked obesity in the case study and individuals in the control group, except with CHO, LDL, and HDL; there is a non-significant difference between the study groups. As shown in table (4).

**Table (4):** Mean distribution of diabetic patients' Lipid profile parameters among BMI groups.

| Body mass index (kg/m$^2$) | Study groups | Investigations | | | | |
|---|---|---|---|---|---|---|
| | | Cholesterol (mg/dl) Mean (±SD) | Triglyceride (mg/dl) Mean (±SD) | LDL (mg/dl) Mean (±SD) | VLDL (mg/dl) Mean (±SD) | HDL (mg/dl) Mean (±SD) |
| **Normal Weight** | Case N=12 | 230.4 (51.2) | 166.1 (62.3) | 144.8 (60.4) | 31.7 (13.8) | 54.0 (27.3) |
| | Control N=42 | 170.2 (20.5) | 75.6 (12.8) | 106.8 (20.5) | 17.7 (6.9) | 48.2 (8.7) |
| **P-value** | | 0.001. | 0.001 | 0.001 | 0.001 | 0.237 |
| **Overweight** | Case | 200.7 (50.7) | 210.3 (137.3) | 126.9 (47.6) | 36.3 (30.5) | 47.6 (13.7) |
| | Control | 150.2 (19.3) | 72.8 (16.9) | 88.9 (18.5) | 20.0 (12.9) | 46.8 (4.6) |
| **P-value** | | 0.001 | 0.001 | 0.002 | 0.034 | 0.796 |
| **Obesity** | Case N=35 | 218.8 (58.4) | 225.4 (78.3) | 139.0 (46.7) | 38.1 (20.3) | 50.2 (39.9) |
| | Control N=21 | 165.7 (20.5) | 85.0 (27.1) | 99.6 (23.9) | 16.9 (5.2) | 49.1 (2.7) |
| **P-value** | | 0.001 | 0.001 | 0.001 | 0.001 | 0.899 |
| **Marked obesity** | Case N=27 | 189.9 (46.8) | 231.0 (56.1) | 100.6 (38.9) | 38.1 (17.1) | 51.0 (17.3) |
| | Control N=6 | 151.5 (1.6) | 90.5 (30.1) | 84.9 (10.4) | 18.3 (5.8) | 48.5 (6.0) |
| **P-value** | | 0.056 | 0.001 | 0.340 | 0.009 | 0.723 |

## Discussion

This investigation revealed that the concentration of glycated hemoglobin in the blood of pat ients was substantially greater [P 0.001] than that of healthy individuals. This indicates poor glycemic control during that period. These results are compatible with Atari et al., who reported that the HbA1c of type 2 diabetes patients higher than the control group [7]. Glycated hemoglobin



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[HbA1c] production is a normal part of the physiological activity process. HbA1c is the most accurate measurement of chronic glycemia and is strongly correlated with the risk of long - term consequences of diabetes; hence, it is commonly regarded as the test of choice for managing and monitoring chronic diabetes [Ahmed et al., 2018]. HbA1c blood testing shows the average blood glucose levels during the previous two to three months, which is the expected half - life of red blood cells [ RBCs]. In T2DM patients, HbA1c variability was related to macrovascular disease, although short- term glucose fluctuation was not. It was linked to macrovascular and microvascular conditions [8]. The resistance of body cells to produce insulin is the cause of the rise in fasting blood glucose levels in diabetic patients[ Prasad et al., 2022 ]. Insulin resistance is a frequent sign of type 2 diabetes, which is essential to the progression of the disease [10]. TG concentration showed a significant increase in a patient with T2D compared to control.

This result agrees with [ Finch, Shamsa, and Lee 2014]. Our results go along with a study by Shahid et al. [11]. Patients with T2DM are not an exception to the rule that individuals with diabetes have abnormal lipid levels. Insulin resistance has been linked to the aberrant lipid profile of T2DM because it increases fatty acid release, lowers insulin - dependent muscle - free fatty acid absorption, and increases hepatic fatty acid synthesis [12]. Diabetes is characterized by increased LDL, triacylglycerol, and low HDL values. Researchers have shown that those with diabetes have a higher lipid profile [Alzahrani et al ., 2019 ]. Wexler et al. observed that HbA1c and lipid profiles [ TC and LDL] have a highly positive and statistically significant connection [14] ; Other researchers have shown a relationship between HbA1c and these lipid profiles in T2DM patients [13]. The relationship between obesity and T2D is very significant. Obesity is the leading cause of insulin resistance, which manifests early in the disease and is compensated predominantly by hyperinsulin emia[15].

In addition, the current study demonstrates that T2DM patients with insulin resistance had considera bly greater triglycerides and lower HDL cholesterol than those with normal insulin sensitivity. The effect of increased BMI referred to the presence of obesity in type 2 DM. Patients agreed with the study[16],which determined that being overweight or obese is a risk factor for acquiring type 2 diab etes. With changes to modern lifestyles in recent years, the prevalence of type 2 diabetes has increased in Iraq. In Iraq and other nations, an increased BMI contributes to the development of type 2 diabetes. [Abid, Alaaraji, and Alrawi, 2019]. [ Al - Bachir and Bakir, 2017 ] Obesity is a risk factor for type 2 diabetes mellitus, and the relative risks for diabetes mellitus in patients with a BMI of > 27 kg/m2 were considerably higher than in those with a BMI of 23 kg / m2. [19]. Demonstrated that BMI was negatively correlated with glucose disposal and positively associated with glucose production in type



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2 diabetes mellitus. [20]. Even in nonobese patients with type 2 diabetes, BMI was the most critical indicator of insulin resistance, according to a study. In this study, a greater BMI was related to lower insulin sensitivity, supporting the positive association between BMI and insulin resistance in type 2 diabetes. [Gonzalez- Cantero et al., 2018] . TG level increment is ascribed to insulin lack, causing hyperglycemia and activation of unsaturated fats from fat tissue, the unsaturated fats from fat tissue are assembled for vitality reasons, and an abundance of unsaturated fats is collected in the liver. They are changed over to triglycerides, and the VLDL– C increment of patients with T2DM perhaps results from hyperinsulinemia and resultant increment in triglycerides, LDL – C, and VLDL - C. It is realized that insulin and development hormones advance the generation of VLDL– C [ Zhang et al., 2022 ]. Activating lipolysis in the fat tissues and triglycerides in the liver, patients with T2DM exhibited overall increased plasma LDL - C cholesterol levels, which may be ascribed to the fact that insulin increases the number of LDL - C receptors; therefore, persistent insulin deficiency, as observed in T2DM, may be associated with a diminished size of LDL- C receptors and a resultant rise in plasma LDL- C cholesterol levels [23].

All lipid profile parameters were lower in lean diabetics compared to all other groups. This agrees with Ahmed [ Shatwan, Ahmed, and Badkook 2013] but does not agree with [ Shavana, Khan, and Anandan 2017], who showed no significant differences in lipid profile in lean diabetes irrespectively of glycemic status. DM type 2 is often associated with overweight, which nearly agreed with [24], who observed that DM type 2 was often associated with obesity.



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**References**


1. Nascè A, Gariani K, Jornayvaz FR, Szanto I. NADPH Oxidases Connecting Fatty Liver Disease, Insulin Resistance and Type 2 Diabetes: Current Knowledge and Therapeutic Outlook. Antioxidants. 2022;11(6):1131.
2. Rasmussen L, Knorr S, Antoniussen CS, Bruun JM, Ovesen PG, Fuglsang J, et al. The Impact of Lifestyle, Diet and Physical Activity on Epigenetic Changes in the Offspring—A Systematic Review. Nutrients. 2021;13(8):2821.
3. Kullo IJ, Lewis CM, Inouye M, Martin AR, Ripatti S, Chatterjee N. Polygenic scores in biomedical research. Nat Rev Genet. 2022;1–9.
4. Li Y, Li Z, Ngandiri DA, Perez ML, Wolf A, Wang Y. The molecular brakes of adipose tissue lipolysis. Front Physiol. 2022;13.
5. Harada MD. The Role of Health and Diet in the Development of Metabolic Syndrome Stratified by Race, Sex, and Age. Walden University; 2022.
6. Tung Y-T, Pan C-H, Chien Y-W, Huang H-Y. Edible mushrooms: Novel medicinal agents to combat metabolic syndrome and associated diseases. Curr Pharm Des. 2020;26(39):4970–81.
7. Atari-Hajipirloo S, Valizadeh N, Khadem-Ansari M-H, Rasmi Y, Kheradmand F. Altered concentrations of copper, zinc, and iron are associated with increased levels of glycated hemoglobin in patients with type 2 diabetes mellitus and their first-degree relatives. Int J Endocrinol Metab. 2016;14(2).
8. Mulani MR, Gawali PS. Effect of Iron deficiency on glycation ofhemoglobin in non–diabetics. ACCLM. 2016;2(2):4–7.
9. Prasad M, Jayaraman S, Eladl MA, El-sherbiny M. A Comprehensive Review on Therapeutic Perspectives of Phytosterols in Insulin Resistance : A Mechanistic Approach. 2022;1–17.
10. Hashim HA, Al-Shammaa NMJ. Study of Irisin Level Hormone and Some Biochemical Parameters in Iraqi Diabetic Type 2 without Hypothyroidism. Pakistan J Med Heal Sci. 2022;16(06):403.
11. Shahid SU, Sarwar S. The abnormal lipid profile in obesity and coronary heart disease (CHD) in Pakistani subjects. Lipids Health Dis. 2020;19(1):1–7.
12. Meex RCR, Blaak EE, van Loon LJC. Lipotoxicity plays a key role in the development of both insulin resistance and muscle atrophy in patients with type 2 diabetes. Obes Rev. 2019;20(9):1205–17.
13. Alzahrani SH, Baig M, Aashi MM, Al-Shaibi FK, Alqarni DA, Bakhamees WH. Association between glycated hemoglobin (HbA1c) and the lipid profile in patients with type 2 diabetes mellitus at a tertiary care hospital: a retrospective study. Diabetes, Metab Syndr Obes targets Ther. 2019;12:1639.
14. Wexler DJ, Grant RW, Meigs JB, Nathan DM, Cagliero E. Sex disparities in treatment of cardiac risk factors in patients with type 2 diabetes. Diabetes Care. 2005;28(3):514–20.
15. Deeb A. Insulin Resis-tance. Impair Fasting, Glucose Intol-erance Type II Diabetes Mellit Overweight Obese Child Abu Dhabi. 2017;1–8.




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E-ISSN:2664-0554

**16.** Barb D, Repetto EM, Stokes ME, Shankar SS, Cusi K. Type 2 diabetes mellitus increases the risk of hepatic fibrosis in individuals with obesity and nonalcoholic fatty liver disease. Obesity. 2021;29(11):1950–60.

**17.** Abid SM, Alaaraji SF, Alrawi KF. Study of Interleukins 13, 18, 27, 32 and 38 Levels in Iraqi Type 2 diabetes with Insulin Resistance. J Educ Sci Stud. 2019;4:14.

**18.** Al-Bachir M, Bakir MA. Predictive value of body mass index to metabolic syndrome risk factors in Syrian adolescents. J Med Case Rep. 2017;11(1):1–7.

**19.** Al-Fartosy AJM, Awad NA, Abdalemam DJ. Biochemical study of the effect of insulin resistance on adiponectin, lipid profile and some antioxidants elements with relation to obesity in type 2 diabetic patients/Basrah-Iraq. Am J Biochem. 2017;7(4):73–82.

**20.** Taniguchi A, Nakai Y, Sakai M, Yoshii S, Hamanaka D, Hatae Y, et al. Relationship of regional adiposity to insulin resistance and serum triglyceride levels in nonobese Japanese type 2 diabetic patients. Metab Exp. 2002;51(5):544–8.

**21.** Gonzalez-Cantero J, Martin-Rodriguez JL, Gonzalez-Cantero A, Arrebola JP, Gonzalez-Calvin JL. Insulin resistance in lean and overweight non-diabetic Caucasian adults: Study of its relationship with liver triglyceride content, waist circumference and BMI. PLoS One. 2018;13(2):e0192663.

**22**. Zhang J, Yu H, Zhong H, Wang Q, Tang J, Shen F, et al. Dietary emulsifier glycerol monodecanoate affects gut microbiota contributing to regulating lipid metabolism, insulin sensitivity and inflammation. Food Funct. 2022;

**23.** Majid A, Sayer SA, Farhood HB. Study of some biochemical parameters for patients with Type II Diabetes Mellitus in Thi-Qar Governorate, Iraq. J Pharm Sci Res. 2018;10(11):2938–41.

**24**. Klein S, Gastaldelli A, Yki-Järvinen H, Scherer PE. Why does obesity cause diabetes? Cell Metab. 2022;34(1):11–20.



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