Tikrit Journal of Pure Science Vol. 31 (4) 2026

DOI:https://doi.org/10.25130/tjps.v31i4.2061

IRAQI Academic Scientific Journals

Tikrit Journal of Pure Science

ISSN: 1813 – 1662 (Print) --- E-ISSN: 2415 – 1726 (Online)

Journal Homepage: https://tjpsj.org/

Evaluation of Malondialdehyde (MDA) Levels and Some Inflammatory Cytokines as Early Biomarkers for the Detection of Gestational Diabetes in Kirkuk City

Marwa Jassim Mohammed, Wedad Mahmood L Al-obaidi

Department of Biology, College of Science, University of Kirkuk, Kirkuk, Iraq

Received: 2 Jun. 2025 **Received in revised forum:** 15 Jul. 2025 **Accepted:** 19 Jul. 2025

Final Proofreading: 23 Jul. 2025 **Available online:** 25 Aug. 2026

ABSTRACT

Gestational diabetes is a glucose intolerance disorder that occurs during pregnancy because hormonal and immunological changes increase insulin resistance, including elevated inflammatory cytokines such as Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL-6), which impair cells' response to insulin. This study aimed to evaluate physiological and immune variables associated with gestational diabetes, including malondialdehyde (MDA), Glutathione (GSH), IL-6, and TNF-α. (60) blood samples were collected from pregnant women in the (10-12) weeks of pregnancy with a body mass index (BMI) ≥30 (A). After follow-up in the second trimester (20-24 weeks), they were divided into two groups (B and C) based on blood glucose levels. The results showed a significant increase($P<0.001$) in both fasting blood sugar (FBS) and Hemoglobin A1c (HbA1c), As for oxidative stress markers such as glutathione (GSH) there were no significant differences ($P>0.05$), but MDA record high significant differences($P<0.001$), likewise for inflammatory markers interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) in obese pregnant women with Gestational diabetes mellitus (GDM) compared to those without GDM in the first trimester. In conclusion, elevated levels of FBS, HbA1c, and insulin resistance, along with increased oxidative stress and inflammation markers, were evident in pregnant women diagnosed with GDM relative to non-GDM pregnant women.

Keywords: GMD, GSH, IL-6, Lipid profile, MDA, Oxidative stress, TNF-α.

Name: **Marwa Jassim Mohammed** **E-mail:** Scbd24017@uokirkuk.edu.iq

Tikrit Journal of Pure Science Vol. 31 (4) 2026
DOI:https://doi.org/10.25130/tjps.v31i4.2061

تقييم مستويات المالون ثنائي الديهايد MDA وبعض السيتوكينات الالتهابية كمؤشرات مبكرة للكشف عن مرض السكري الحملي في مدينة كركوك.

مروة جاسم محمد، وداد محمود العبيدي

قسم علوم الحياة، كلية العلوم، جامعة كركوك، كركوك، العراق

الملخص

سكري الحمل هو اضطراب عدم تحمل الجلوكوز يحدث أثناء الحمل نتيجة زيادة مقاومة الأنسولين بسبب التغيرات الهرمونية والمناعية، بما في ذلك ارتفاع مستويات السيتوكينات الالتهابية مثل عامل النخر الورمي – الفا (TNF-α) و الأنترلوكين–6 (IL-6)، مما يضعف استجابة الخلايا للأنسولين. هدفت الدراسة الحالية إلى تقييم بعض المتغيرات الفسيولوجية والمناعية المرتبطة بسكري الحمل، مثل المالون ثنائي الديهايد (MDA) و الجلوتاثيون (GSH) و IL-6 و TNF-α . تم جمع (60) عينة دم من النساء الحوامل في الأسبوع (10–12) من الحمل بمؤشر كتلة الجسم ≥ 30 (BMI) (A)، بعد متابعة الحالات في الثلث الثاني من الأسبوع (20–24) من الحمل، تم تقسيمهن إلى مجموعتين (B و C) وفقًا لمستوى سكر الدم. أظهرت النتائج زيادة كبيرة ($P < 0.001$) في كل من سكر الدم الصائم (FBS) و الهيموغلوبين السكري (HbA1c)، أما بالنسبة لعلامات الإجهاد التأكسدي مثل الجلوتاثيون (GSH) فلم تكن هناك فروق ($P>0.05$) ، ولكن سجل MDA فروقًا كبيرة($P < 0.001$) ، وبالمثل بالنسبة لعلامات الالتهاب IL-6 و TNF-α في النساء الحوامل البدينات المصابات بسكري الحمل مقارنة بأولئك غير المصابات بسكري الحمل في الأشهر الثلاثة الأولى. وفي الختام، كانت المستويات المرتفعة من FBS و HbA1c ومقاومة الأنسولين، إلى جانب زيادة علامات الإجهاد التأكسدي والالتهاب، واضحة لدى النساء الحوامل المصابات بسكري الحمل مقارنة بالنساء الحوامل غير المصابات بسكري الحمل.

INTRODUCTION

Diabetes is a type of metabolic disease characterized by chronically elevated blood sugar levels [1]. Diabetes is divided into four types: Type 1 diabetes (insulin-dependent), Type 2 diabetes (non-insulin-dependent), gestational diabetes, and special types of diabetes. Gestational diabetes mellitus (GDM) is a condition in which the mother's pancreatic beta cells are unable to function properly, resulting in a deficiency in the amount of insulin produced by the cells, and thus poor regulation of glucose levels throughout pregnancy [2], If risks are detected and addressed as soon as possible, there is a chance they can be reduced [3]. It affects about 9-25% of all pregnancies worldwide, however this percentage may vary according to demographic and diagnostic criteria [4, 5]. Gestational diabetes affects about 13% of pregnancies worldwide and is associated with an increased risk of delivering a large-for-gestational-age (LGA) baby. Gestational diabetes can lead to birth complications and babies are at increased risk of developing cardiovascular disease later in life. The prevalence of gestational diabetes remains high even when blood sugar is well controlled. Studies have shown that continuous glucose monitoring in women with gestational diabetes who give birth to large-for-gestational-age babies has periods of mild hyperglycemia. It is not clear how this causes gestational diabetes, but gestational diabetes is associated with altered placental growth and function [6]. Insulin resistance contributes to the development of various diseases, including type 2 diabetes mellitus (T2DM) and gestational diabetes mellitus (GDM) [7], Insulin resistance occurs when the body does not respond well to insulin, as glucose has less ability to activate the cells of any organ in the tissues that depend on insulin in normal conditions, causing a disturbance in food metabolism [8]. Oxidative stress is defined as an

imbalance between oxidants and antioxidants, favoring the latter, and is recognized as a major mechanism that impairs molecular signaling pathways and enzyme activities, leading to tissue damage [9]. Oxidative stress impairs cell function through a number of metabolic processes. It hinders the entry of proinsulin vesicles into the plasma membrane and causes a marked reduction in insulin production. Furthermore, in reaction to variations in blood glucose levels, it decreases cell secretion. Furthermore, it may trigger apoptotic processes in pancreatic cells, which result in beta cell loss and death. Numerous proapoptotic compounds can induce apoptosis in pancreatic cells by strongly interacting with oxidative stress [10]. Pregnancy is linked to increased susceptibility to oxidative stress , preeclampsia, recurrent pregnancy loss, placental dysfunction, fetal developmental defects, intrauterine growth restriction, and, in severe cases, fetal death are all possible consequences of increased oxidative stress during pregnancy [11], There is also evidence that children of mothers with GDM are more likely to develop childhood obesity and type 2 diabetes. Risk factors for GDM are not well understood, but women of advanced gestational age, obesity or overweight, and a family history of diabetes or a previous history of GDM are high-risk groups. Therefore, the incidence rate of GDM has increased with the increase in females. Obesity rates and mild cases can be resolved by modifying diet and exercising, but in severe cases medical interventions are required, such as insulin injections [12]). Pregnancy causes inflammation, and women with gestational diabetes mellitus have poorly controlled immunological responses during pregnancy. A common symptom of hyperglycemic diseases, such as GDM, is oxidative stress, which exacerbates insulin resistance and triggers an inflammatory response. Oxidative stress also damages cellular components and affects cytokine release, activation, and proliferation [13, 14]. Post-insulin receptors and inflammatory cytokines work together to prevent the insulin receptor substrate's normal tyrosine phosphorylation, which reduces the substrate's capacity to bind the insulin receptor and affects the regulation of glucose metabolism [15]. Obesity remains a major health problem in many countries. Physical inactivity and unhealthy eating habits are obesity-related behaviors that contribute to many problems, highlighting the need for early prevention [16]. The World Health Organization (WHO) defines obesity as excessive or aberrant fat buildup that presents a risk to one's health. With over 4 million fatalities linked to overweight or obesity, obesity has reached epidemic proportions. One common pathway linking obesity and its consequences is oxidative stress. An imbalance between the production of free radicals and antioxidant defenses is known as oxidative stress. Additional pathways linked to tissue damage, including inflammation, excess formation of extracellular matrix, activation of endoplasmic reticulum stress, and disruption of autophagic flow, may be triggered by this oxidative environment [17]. This study aimed to evaluate the effectiveness of selected biomarkers and clinical indicators in the early detection of gestational diabetes during the end of the first trimester of pregnancy, with the aim of enabling early intervention and reducing the progression of maternal and fetal health complications. The current study also sought to identify factors associated with an increased risk of developing the disease, which would contribute to the development of effective preventive strategies and improve the care of pregnant women at risk.

MATERIALS AND METHODS

Study population

Samples were collected in Kirkuk city for the period from March 2024 to February 2025. In addition to private clinics, the patients were those who visited the Obstetrics and Gynecology Department of Al-Nasr Maternity Hospital. Each participant received a questionnaire to complete and sign, along with a consent form. Every patient received a questionnaire with demographic data like age, body mass index (BMI), cycle pattern, number of births, and other pertinent information, as well as a medical examination and an interview with a gynecologist

Inclusion criteria

Two groups of pregnant women in the first trimester who are obese with a BMI ≥ 30. The groups are followed up and divided according to the incidence of gestational diabetes into 2 subgroups in the second trimester of pregnancy.

Exclusion criteria

Pregnant women who had type 1 or type 2 diabetes, women who used medications that raise blood sugar levels such as cortisol, women with frequent miscarriages and those with chronic high blood pressure were excluded.

Study procedure

Pregnant women attending Al-Nasr Hospital for Obstetrics and Gynecology were screened according to the above-mentioned inclusion and exclusion criteria and were in their first trimester of pregnancy. After a full explanation of the procedure and the purpose of the study, they were enrolled in the study after obtaining written informed consent. According to WHO guidelines, they were followed until they entered the second trimester of pregnancy and after a 12-hour fast. Three 75-gram oral glucose tolerance (OGTT) tests were performed on pregnant women in their second trimester. Based on the OGTT and HbA1c results, study subjects were divided into GDM and normal glucose tolerance (NGT) controls. Thus, 100 GDM patients and 100 non-GDM pregnant women were enrolled in the study.

Biochemical analysis

Following a 12-hour fast, each study participant had a venipuncture to obtain 5 ml of blood. For the HbA1c test, one milliliter of fresh venous blood was separated into an EDTA tube, and the other four milliliters were transferred into sterile gel tubes. After a short period, the blood began to clot and then it was centrifuged at 3000 rpm for 15 minutes. The serum was drawn using a mechanical pipette and transferred to an Eppendorf tube, and each tube was coded. with the patient's code and then store it in the freezer (Deep freeze at -20°C) to evaluate subsequent bioassays, to examine glucose, lipid profile, insulin, GSH, MDA, IL-6 and TNF-α, and these tests are preceded by an OGTT examination. Then, after loading 75 g of glucose, two samples were taken again at 1-hour intervals. Plasma glucose analysis was performed on the day of collection. The serum was stored in a refrigerator at -20°C until further analysis for insulin, liped profile, and the rest of the tests in one test tube. The concentration of glucose in blood serum was measured automatically using the GLUC3 (Glucose HK Gen.3 Roche/Hitachi Cobas c systems), and the insulin level was assessed using an automated autoanalyzer for chemiluminescence (Elecsys, Cobas e411, Roche Diagnostic GmbH, Mannhein, Germany). Also, a ready-made kit from SunLong Biotech Co., LTD was used, where the ELISA-type technique was used. (Sandwich - ELIZA) As a way to measure GSH, MDA,IL-6, TNF-α concentration [18]. The insulin resistance index was estimated by multiplying the fasting blood sugar (FBS) mg concentration by the fasting insulin level and dividing by 22.5 which will provide the Homa IR index [19].

- (Homeostasis model evaluation insulin resistance index)

HOMA-IR = FBS (mmol/L) ×FINS(μU/ml)/22.5

- (Steady-state model assessment islet beta cells secrete index)

HOMA-β = 20× FINS(μU/ml)/ FBS (mmol/L) -3.5

The ratio indices were also calculated according to the following equations [20]:

Triacylglycerol glucose index = Ln [TG*FBS/2]

TG/HDL-c ratio is calculated as = TG/HDL-c.

TC/HDL-c ratio is calculated as = TC/HDL-c.

Statistical analysis

The data were analyzed using one-way anova and the difference between means was tested according to Duncan's multiple range test using SPSS version 27.

RESULTS

Characteristics of study participants

(60) blood samples were selected for pregnant women in the (10-12) week of pregnancy with a body mass index ≥30 BMI, which is (A). After following up on the cases in the second half (20-24)

weeks of pregnancy, a number of samples that did not meet the conditions were excluded, specifically those taking sugar-lowering medications such as metformin, with cases of miscarriage or other cases. It was noted that (43) of the pregnant women suffered from a slight increase in a number of variables, but no cases of gestational diabetes were recorded, which is (B). While (17) of the total number of obese pregnant women who entered pregnancy with excess weight and were suffering from obesity suffered in the second half of pregnancy from physiological and hormonal changes in most of the studied variables that have a close relationship with the blood sugar level and all related analyses, which were diagnosed as cases of gestational diabetes and were considered cases (C). The results of the current study showed that group C recorded a significant increase at the probability level (P<0.001) in the concentration of FBS and in the concentration of the insulin hormone, the binding peptide C-pep. and HbA1c. Also, significant differences were recorded in the value of the HOMA-IR index and Homa-β, as in Table (1).

Table 1: Diabetes parameters in different trimesters of pregnancy in mothers with and without gestational diabetes.

variables	Groups			P value
	A Mean ±SD	B Mean ±SD	C Mean ±SD	
FBS mg/dl	94.63±14.64^{c}	108.6±8.32^{b}	148.58±15.01^{a}	P<0.001
HbA1c %	5.4±0.30^{b}	5.75±0.32^{b}	7±0.51^{a}	P<0.001
Insulin µu/ml	11.55±4.71^{b}	25.33±5.94^{a}	26.29±8.71^{a}	P<0.001
C-pep ng/ml	1.36±0.63^{c}	3.1±0.88^{b}	5.28±1.06^{a}	P<0.001
Homa-IR	2.71±1.24^{c}	6.77±1.67^{b}	8.81±2.64^{a}	P<0.001
Homa-β	148.84±83.01^{b}	208.63±66.93^{a}	119.85±55.60^{c}	P<0.001
BMI	30.07±1.03^{b}	31.45±1.03^{ab}	32±1.19^{a}	P<0.01

Different letters indicate significant differences between groups.

For the lipid profile, the results showed significant differences in cholesterol concentration at the probability level (P<0.01). The results showed significant differences in LDL concentration at the probability level (P<0.01) and a significant decrease at the probability level (P<0.05) in HDL concentration. As for VLDL and Triglyceride, the results showed significant differences at the probability level (P<0.001), as shown in Table (2).

Table 2: Association of Patterns of Change in lipid profiles and lipid ratios with GDM risk in early to middle pregnancy.

Groups Lipid profile	A Mean ±SD	B Mean ±SD	C Mean ±SD	P value
Cholesterol mg/dl	185.77±40.72^{b}	222.46±34.65^{a}	234.11±60.59^{a}	P<0.01
Triglyceride mg/dl	187.3±74.36^{b}	262.4±61.59^{a}	268.11±59.75^{a}	P<0.001
HDL mg/dl	44±9.81^{a}	37.13±5.62^{b}	38.47±6.76^{b}	P<0.05
LDL mg /dl	106.15±63.49^{b}	132.78±51.99^{a}	142.02±46.09^{a}	P<0.01
VLDL mg/dl	37.15±19.18^{b}	52.48±12.31^{a}	53.62±11.95^{a}	P<0.001
Tyg index	5.27±2.64^{a}	5.13±0.13^{a}	5.26±0.09^{a}	P>0.05
Ch/HDL index	4.47±1.78^{b}	6.08±1.22^{a}	6.28±1.99^{a}	P<0.001
TG/HDL index	4.55±2.29^{b}	7.57±2.26^{a}	7.06±1.55^{a}	P<0.001

Different letters indicate significant differences between groups.

The study also showed highly significant differences (P<0.001) for MDA, IL-6 and TNF-, while there were no differences (P>0.05) in the level of GSH, as shown in Table (3).

Table 3: Some indicators of oxidative stress and inflammation in pregnant women

Groups variables	A Mean ±SD	B Mean ±SD	C Mean ±SD	P value
MDA ng/ml	37.2±9.08[c]	57.72±16.25[b]	75.06±34.84[a]	P<0.001
GSH ng/ml	2.19±1.02[a]	1.95±0.84[a]	2.10±0.76[a]	P>0.05
TNF-α ng/ml	45.7±10.68[c]	51.01±20.50[c]	63.97±28.89[a]	P<0.001
IL-6 ng/ml	9.99±5.29[c]	16.72±7.01[b]	20.35±9.37[a]	P<0.001

Different letters indicate significant differences between groups.

DISCUSSION

Metabolic syndrome is intricately associated with diabetes and obesity. Elevated obesity fosters inflammation and oxidative stress, which are precursors to several problems, including elements of metabolic disorders such as insulin resistance and hyper lipidaemia [21]. A metabolic disorder that results in glucose intolerance during pregnancy, gestational diabetes (GD) significantly affects the health of both the mother and the fetus. Risk factors for insulin resistance and cellular and molecular abnormalities in glucose regulation include ethnicity, Obesity, a family history of diabetes, and advanced maternal age. About 14% of people worldwide have it. GD puts the mother and the baby at risk for both immediate and long-term effects. GD increases the risk of metabolic syndrome, cardiovascular disease, and type 2 diabetes in women. Pregnancy-related GD exposure raises the risk of obesity, glucose intolerance, and metabolic issues in later life. [22]. The results of the current study as in Table (1) showed that women who have a higher level of glycated hemoglobin (HbA1c) in the first trimester of pregnancy have a higher risk of developing gestational diabetes. These results are consistent with what was reported by [23]. They showed that the high level of glycated hemoglobin A1c (HbA1c), resulting from the spontaneous binding of glucose to the beta chain of hemoglobin, in different types of diabetes in the bloodstream, indicates an increase in glucose concentration during the previous two to three months. A study by [24] also evaluated the relationship between glycated hemoglobin (HbA1c) levels and adverse pregnancy outcomes in 2,048 women with gestational diabetes. The results showed a strong association between HbA1c levels and these adverse pregnancy outcomes at the time of gestational diabetes diagnosis (24–28 weeks). Pregnant women with an HbA1c level above 5.5% were at greater risk of adverse outcomes compared to pregnant women with an HbA1c level between 5.1 and 5.4%, particularly below 5.0%. Based on these findings, it is suggested that HbA1c may be a biomarker of adverse pregnancy outcomes in women with gestational diabetes. Gestational diabetes mellitus (GDM) can have adverse effects on pregnancy. Gestational diabetes mellitus (GDM) is one of the most prevalent metabolic diseases in pregnant women [25]. Changes in the lipid profiles of pregnant women as in Table (2) are linked to GDM. TG, LDL-c, triglyceride glycaemic index, TG/HDL-c, and TC/HDL-c were all positively associated with the risk of GDM in the early stages of pregnancy, but HDL-c was negatively associated, which is in line with previous findings [20, 26]. However, in a Chinese study, women with GDM had statistically significant differences in TG concentrations and HDL-c levels when compared to controls; however, other TC, such as LDL-c, did not reveal statistically significant variations between the two groups [27], Furthermore, a meta-analysis revealed that, in comparison to women with healthy pregnancies, women with GDM had lower HDL-c and higher TG in the early and mid-stages of their pregnancies. Our findings are biologically plausible, even if the mechanisms underlying the association between lipid profiles, lipid ratios, and GDM are still unknown. Insulin resistance and elevated estrogen levels during pregnancy promote the liver's ability to produce fat [28]. The physiological changes that occur in a woman's body

during pregnancy prioritize lipid metabolism over glucose metabolism, as the mother uses lipids as an energy source to promote fetal growth and development while saving glucose. Lipids increase during the first trimester of pregnancy, raising blood levels of free fatty acids. This can affect insulin sensitivity and create a vicious cycle between high lipid levels and insulin resistance, which can result in decreased glucose tolerance and the onset of diabetes mellitus [29]. Moreover. Because low HDL-C levels decrease AMP-activated protein kinase activity, insulin secretion, and insulin sensitivity, they can have an impact on glucose homeostasis [30]. The current study is also consistent with what [31] found regarding an increase in the level of harmful cholesterol and LDL levels with weight gain (obesity). During pregnancy, oxidative stress indicators such as MDA increase, as well as pro-inflammatory cytokines such as IL-6, as shown in Table (3). Gestational diabetes mellitus (GDM) causes oxidative stress in both mothers and infants and is strongly linked to unfavorable pregnancy outcomes[32]. The two primary components of the metabolic syndrome linked to oxidative stress are obesity and insulin resistance, indicating a close connection between oxidative stress and excessive fat accumulation [33]. Similarly, another study found that reducing body weight in obese patients with moderate calorie restriction for eight weeks increased antioxidant defense and decreased oxidative stress indicators. Alongside these modifications, there was also an improvement in glucose tolerance, which led to a reduction in insulin resistance [34]. According to a recent paper, mitochondrial dysfunction in GDM affects the insulin signaling pathway and raises oxidative stress, both of which raise the risk of hyperglycemia [22]. It has long been known that obesity-related inflammation contributes to oxidative stress [17]. Interleukin 6 (IL-6) is an interleukin that works as a proinflammatory cytokine and as an anti-inflammatory myokine. The production of IL-6 in humans is under genetic control [35].

CONCLUSIONS

Gestational diabetes exacerbates oxidative stress during pregnancy, threatening maternal and fetal health. Understanding the pathogenesis of this disease is critical for developing targeted interventions. Cytokines play a pivotal role in the development of obesity and insulin resistance resulting from inflammation. The increased rate of abnormal production of these small proteins capable of influencing gene expression triggers a signaling cascade that contributes to the diabetic phenotype. Conclusions: - Increased levels of FBS, HbA1c, and insulin resistance were observed in pregnant women with gestational diabetes compared to pregnant women without gestational diabetes, as well as increased indicators of oxidative stress and inflammatory indicators. This suggests a close association between increased oxidative stress markers, pro-inflammatory cytokines, insulin resistance, and consequently gestational diabetes.

Acknowledgments

The authors would like to thank the University of Kirkuk and the Al-Nasr Hospital for Gynecology and Pediatrics, affiliated with the Iraqi Ministry of Health, for their facilities during the experimental work, as well as all the pregnant women who participated in this study.

Conflict of interests: The authors declared no conflicting interests.

Sources of funding: Self – funding.

Author contribution: Authors contributed equally in the study.

REFERENCES

1. Sulaiman AH, Ghassan ZI, Omar TN. Biochemical Evaluation of Carbonic Anhydrase and Some Antioxidant Markers in Patients with Diabetes Complications. Arch Razi Inst. 2022;77(1):169-78. https://doi.org/10.22092/ARI.2021.356308.1817

2. Diyaa D, AlKatib S, Aziz N. Exploring Interleukin 6 as a Promising Marker for The Diagnosis of Gestational Diabetes Mellitus. Karbala Journal of Pharmaceutical Sciences. 2024;14(23):106-15. https://doi.org/10.62472/kjps.v14.i23.106-115

3. Rasool J, Sadegh A. An Intelligent Gestational Diabetes Mellitus Recognition System Using Machine Learning Algorithms. Tikrit Journal of Pure Science. 2023;28(1):82-8. https://doi.org/10.25130/tjps.v28i1.1269

4. Shareef L, Abdulrahman C, Salih S. Relation of Vitamin D With Macrosomia in Pregnant Women Attending Kirkuk General Hospital. The Medical Journal of Tikrit University. 2019;25(2):212-7. https://mjtu.tu.edu.iq/index.php/mjtu/article/view/41

5. Choudhury AA, Devi Rajeswari V. Gestational diabetes mellitus - A metabolic and reproductive disorder. Biomedicine & Pharmacotherapy. 2021;143:112183. https://doi.org/10.1016/j.biopha.2021.112183

6. Byford AR. The impact of maternal glucose fluctuations in gestational diabetes on placental development: University of Leeds; 2023.

7. Szlapinski SK, Hill DJ. In vivo models of gestational and type 2 diabetes mellitus characterized by endocrine pancreas cell impairments. Journal of Endocrinology. 2024;260(3):e230317. https://doi.org/10.1530/JOE-23-0317

8. Lee SH, Park SY, Choi CS. Insulin Resistance: From Mechanisms to Therapeutic Strategies. Diabetes Metab J. 2022;46(1):15-37. https://doi.org/10.4093/dmj.2021.0280

9. Sies H, Berndt C, Jones DP. Oxidative Stress. Annu Rev Biochem. 2017;86:715-48. https://doi.org/10.1146/annurev-biochem-061516-045037

10.Bhatti JS, Sehrawat A, Mishra J, Sidhu IS, Navik U, Khullar N, et al. Oxidative stress in the pathophysiology of type 2 diabetes and related complications: Current therapeutics strategies and future perspectives. Free Radical Biology and Medicine. 2022;184:114-34. https://doi.org/10.1016/j.freeradbiomed.2022.03.019

11.Grzeszczak K, Lanocha-Arendarczyk N, Malinowski W, Ziętek P, Kosik-Bogacka D. Oxidative Stress in Pregnancy. Biomolecules. 2023;13:1768. https://www.mdpi.com/2218-273X/13/12/1768#

12.Ersahin SS, Yurci A. Cord blood and maternal serum preptin and irisin concentrations are regulated independently in GDM. Eur Rev Med Pharmacol Sci. 2021;25(4):1954-8.

13.Basu J, Datta C, Chowdhury S, Mandal D, Mondal NK, Ghosh A. Gestational Diabetes Mellitus in a Tertiary Care Hospital of Kolkata, India: Prevalence, Pathogenesis and Potential Disease Biomarkers. Exp Clin Endocrinol Diabetes. 2020;128(04):216-23. . https://doi.org/10.1055/a-0794-6057

14.Djelti F, Merzouk H, Merzouk SA, Narce M. In vitro effects of oil's fatty acids on T cell function in gestational diabetic pregnant women and their newborns. J Diabetes. 2015;7(4):512-22. https://doi.org/10.1111/1753-0407.12210

15.Wang J, Zhu Q-W, Cheng X-Y, Sha C-X, Cui Y-B. Clinical significance of neutrophil–lymphocyte ratio and monocyte–lymphocyte ratio in women with hyperglycemia. Postgraduate Medicine. 2020;132(8):702-8. https://doi.org/10.1080/00325481.2020.1764235

16.Shakir N. From Calories to Nutrients: The Role of Lifestyle Changes for Addressing Obesity. Tikrit Journal of Pure Science. 2024;29(6):10-22. https://doi.org/10.25130/tjps.v29i6.1675

17.Martinez-Martinez E, Cachofeiro V. Oxidative Stress in Obesity. Antioxidants. 2022;11:639. https://doi.org/10.3390/antiox11040639

18.Ibrahim NAK. Assessment of the Insulin Resistance, Inflammatory Markers and Gene Polymorphism in Polycystic Ovarian Syndrome patients. Kirkuk Journal of Science. 2025;20(1):1-10. https://doi.org/10.32894/kujss.2025.155788.1187

19.Ma N, Bai L, Lu Q. First-Trimester Triglyceride-Glucose Index and Triglyceride/High-Density Lipoprotein Cholesterol are Predictors of Gestational Diabetes Mellitus Among the Four Surrogate Biomarkers of Insulin Resistance. Diabetes, Metabolic Syndrome and Obesity.

2024;17:1575-83. https://doi.org/10.2147/DMSO.S454826

20.Xu X, Luo S, Lin J, Zhou J, Zheng L, Yang L, et al. Association between maternal lipid profiles and lipid ratios in early to middle pregnancy as well as their dynamic changes and gestational diabetes mellitus. BMC Pregnancy and Childbirth. 2024;24(1):510. https://doi.org/10.1186/s12884-024-06692-9

21.Abd AM, Khaleel FM. Assessing the Role of GSK3 β and Arrestin Beta1 in Regulating Oxidative Stress that Causes Metabolic Disorders. Kirkuk Journal of Science. 2025;20(1):43-53. https://doi.org/10.32894/kujss.2025.157067.1193

22.Torres-Torres J, Monroy-Muñoz IE, Perez-Duran J, Solis-Paredes JM, Camacho-Martinez ZA, Baca D, et al. Cellular and Molecular Pathophysiology of Gestational Diabetes. Int J Mol Sci. 2024;25(21). https://doi.org/10.3390/ijms252111641

23.Gu ZJ, Song QJ, Gu WQ, Zhang GP, Su Y, Tang Y, et al. New approaches in the diagnosis and prognosis of gestational diabetes mellitus. Eur Rev Med Pharmacol Sci. 2023;27(21):10583-94.

24.Muhuza MPU, Zhang L, Wu Q, Qi L, Chen D, Liang Z. The association between maternal HbA1c and adverse outcomes in gestational diabetes. Front Endocrinol (Lausanne). 2023;14:1105899. https://doi.org/10.3389/fendo.2023.1105899

25.Zhang Y, Ye Y, Jia X, Wang P, Xiong Z, Zhu H. The protective effects of Salusin-α against oxidative stress and inflammatory response in mice with gestational diabetes mellitus (GDM). Archives of Physiology and Biochemistry. 2025;131(3):493-502. https://doi.org/10.1080/13813455.2025.2456876

26.Zeng Y, Yin L, Yin X, Zhao D. Association of triglyceride-glucose index levels with gestational diabetes mellitus in the US pregnant women: a cross-sectional study. Front Endocrinol (Lausanne). 2023;14:1241372. https://doi.org/10.3389/fendo.2023.1241372

27.Li Y, Wang X, Jiang F, Chen W, Li J, Chen X. Serum lipid levels in relation to clinical outcomes in pregnant women with gestational diabetes mellitus: an observational cohort study. Lipids Health Dis. 2021;20(1):125. https://doi.org/10.3389/fendo.2023.1241372

28.Rahnemaei FA, Pakzad R, Amirian A, Pakzad I, Abdi F. Effect of gestational diabetes mellitus on lipid profile: A systematic review and meta-analysis. Open Med (Wars). 2022;17(1):70-86. https://doi.org/10.1515/med-2021-0408

29.Manell H, Kristinsson H, Kullberg J, Ubhayasekera SJK, Mörwald K, Staaf J, et al. Hyperglucagonemia in youth is associated with high plasma free fatty acids, visceral adiposity, and impaired glucose tolerance. Pediatr Diabetes. 2019;20(7):880-91. https://doi.org/10.1111/pedi.12890

30.Di Bartolo BA, Cartland SP, Genner S, Manuneedhi Cholan P, Vellozzi M, Rye KA, et al. HDL Improves Cholesterol and Glucose Homeostasis and Reduces Atherosclerosis in Diabetes-Associated Atherosclerosis. J Diabetes Res. 2021;2021:6668506. https://doi.org/10.1155/2021/6668506

31.Abbood JJ, Al-Obaidi WML. Evaluation of Fat Levels in Obese Women with Diabetes in Kirkuk City. Central Asian Journal of Medical and Natural Science. 2024;5(4):735-41.

32.Fang L, Lu S, Fang L, Yu J, Kakongma N, Hu W. Metformin ameliorates gestational diabetes mellitus via inhibiting ferroptosis of trophoblasts through the Nrf2/HO-1 signaling pathway. Free Radical Research. 2025;59(2):190-203. https://doi.org/10.1080/10715762.2025.2468737

33.Jakubiak GK, Osadnik K, Lejawa M, Osadnik T, Goławski M, Lewandowski P, et al. "Obesity and Insulin Resistance" Is the Component of the Metabolic Syndrome Most Strongly Associated with Oxidative Stress. Antioxidants (Basel). 2021;11(1). https://www.mdpi.com/2076-3921/11/1/79#

34.Kanikowska D, Kanikowska A, Swora-Cwynar E, Grzymisławski M, Sato M, Bręborowicz A, et al. Moderate Caloric Restriction Partially Improved

Oxidative Stress Markers in Obese Humans. Antioxidants (Basel). 2021;10(7). https://doi.org/10.3390/antiox10071018

35.Ali A. The Role of IL-6 in Inflammatory Reaction during Coronavirus-19 Infection: A Review. Journal of Communicable Diseases. 2022:210-4. https://doi.org/10.24321/0019.5138.202233