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RESEARCH ARTICLE

ASSESSMENT OF INFLAMMATORY AND ANTI-INFLAMMATORY CYTOKINES IN IRAQI PATIENTS WITH TYPE 2 DIABETES

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Abstract

Chronic low-grade inflammation. is increasingly recognized as a central feature in the pathogenesis of type 2 diabetes mellitus (T2DM). Pro- and anti-inflammatory cytokines play critical roles in mediating immune-metabolic interactions that influence insulin resistance and disease progression. This study aims to evaluate the serum levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α) and interleukin-10 (IL-10) in patients with T2DM compared to healthy individuals also to assess their relationship with clinical parameters such as age and body mass index (BMI). This case-control study included 100 participants: 70 patients diagnosed with T2DM and 30 healthy age- and sex- mostly matched controls. Serum cytokine levels were measured using enzyme-linked immunosorbent assay (ELISA). Clinical data including age and BMI were recorded. Statistical analysis was performed to compare cytokine levels between groups and assess correlations with age and BMI. Diabetic patients exhibited significantly higher levels of IL-6 (8.2 ± 1.7 pg/mL) and TNF-α (13.3 ± 7.2 pg/mL) compared to controls (3.5 ± 1.2 pg/mL and 5.5 ± 3.1 pg/mL, respectively; p-value < 0.05). Conversely, IL-10 levels were markedly lower in the diabetic group (4.6 ± 2.9 pg/mL) than in controls (7.3 ± 3.3 pg/mL; p = 0.011). While age was similar between groups (p = 0.21), BMI was significantly higher in diabetic patients (p-value < 0.001), and positively correlated with IL-6 and TNF-α. Patients with T2DM exhibit an imbalanced cytokine profile marked by elevated pro-inflammatory and reduced anti-inflammatory markers which consistent with a state of chronic inflammation. These alterations appear to be associated more with obesity than with age. Monitoring cytokine levels may offer valuable insight into the immunometabolic status of diabetic patients and could serve as a target for therapeutic intervention.

Keywords: Type 2 diabetes mellitus, IL-6, TNF-α, IL-10, Cytokines, Inflammation, BMI, Insulin resistance.

Introduction

Diabetes mellitus (DM) is a complex and multifactorial metabolic disorder characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both [1]. Globally, the prevalence of diabetes has risen dramatically over the past few decades, reaching epidemic proportions. According to “the International Diabetes Federation (IDF)”, over 537 million adults were living with diabetes in 2021, and this number is expected to elevate significantly in the coming decades [2]. While the metabolic derangements associated with diabetes are well recognized, a growing body of evidence has underscored the critical role of the immune system in both the pathogenesis and progression of the disease [3].

There are two main forms of diabetes mellitus: type 1 diabetes mellitus (T1DM), which is an autoimmune disorder leading to the destruction of pancreatic β-cells, and type 2 diabetes mellitus (T2DM) which is characterized by insulin resistance, relative insulin deficiency, and chronic low-grade inflammation [4]. While T1DM clearly involves immune-mediated mechanisms, emerging research has shown that T2DM also involves significant immune system dysregulation, contributing to disease onset, progression, and complications [5]. In fact, the pathophysiological link

between chronic inflammation and insulin resistance has redefined T2DM not only as a metabolic disorder but also as an immune-metabolic condition [6].

The immune system, comprising innate and adaptive components, plays a central role in maintaining homeostasis and responding to pathogens [7]. However, in diabetes, immune activation becomes persistent and dysregulated. In T2DM, adipose tissue, liver and skeletal muscle exhibit a pro-inflammatory milieu marked by the recruitment of immune cells such as macrophages, T-lymphocytes and B-cells [8]. These immune cells release various cytokines and chemokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α) and interferon-gamma (IFN-γ)—which interfere with insulin signaling and promote insulin resistance [9]. This chronic inflammatory state contributes to pancreatic β-cell stress, apoptosis, and impaired insulin secretion.

Moreover, regulatory immune mechanisms are often impaired in diabetes [10]. Anti-inflammatory cytokines like interleukin-10 (IL-10) and transforming growth factor-beta (TGF-β), which usually help resolve inflammation and maintain immune tolerance, are often reduced or dysfunctional in diabetic patients [11]. This imbalance between pro-inflammatory and anti-inflammatory signals contributes to the sustained activation of immune pathways, leading to tissue damage and systemic complications [12]. The role of IL-39 and its impact in type 2 diabetic Iraqi patients was investigated recently [13]. The researchers found an elevation in the level of IL-39 in type 2 diabetic patients, displaying its role in the pathogenesis of the disease; however, it lacks its association with demographic, clinical and cardiovascular complications.

The relationship between immune dysfunction and diabetes is bidirectional. On one hand, hyperglycemia itself can induce immune cell activation and impair their function [14]. On the other hand, chronic inflammation can worsen insulin resistance and contribute to glycemic instability [15]. Importantly, this immune-metabolic crosstalk plays a critical role in the development of diabetes-related complications, including cardiovascular disease, nephropathy, neuropathy, and retinopathy [16]. For example, increased levels of inflammatory markers such as C-reactive protein (CRP), IL-6 and TNF-α have been associated with endothelial dysfunction, atherosclerosis and increased cardiovascular risk in diabetic patients [17].

Despite the growing recognition of the immune system's involvement in diabetes, routine clinical assessments do not typically include immune biomarkers. There is an urgent need to better characterize the immunological landscape of diabetes to identify novel diagnostic markers and therapeutic targets. Immune profiling may help stratify patients based on their inflammatory status, allowing for more personalized treatment strategies [18]. Furthermore, immunomodulatory therapies such as anti-cytokine agents, immune cell inhibitors, and regulatory T cell therapies hold promise for the treatment of diabetes and prevention of its complications [19]. This study aims to evaluate the immunological profile of patients with diabetes mellitus by assessing circulating cytokine levels, inflammatory markers and lymphocyte subsets. We hypothesize that diabetic patients exhibit distinct immunological alterations compared to healthy individuals, and these changes may provide insights into the underlying mechanisms of disease progression. Understanding these immunological shifts may facilitate the development of immune-based diagnostic tools and therapeutic interventions in the management of diabetes.

Materials and Methods

Study Design and Participants

This cross-sectional observational study was conducted at General Al Hussein hospital in Samawah province, Iraq in period from May 2023 to April 2025. The aim was to assess the circulating levels of specific cytokines including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-10 (IL-10) in patients with type 2 diabetes mellitus (T2DM) compared to healthy controls.

Many factors were considered during selection and calculation of the sample size of this study including; type1 error (α) set at 0.05, power (1-β) typically at 0.80 or 0.90; the effective size and the difference in mean biomarker levels, and proportions; and ratio of cases to controls. In the current study, a total of 100 participants were enrolled and divided into two groups, with the first group as Diabetic Group (n = 70) including Individuals diagnosed with T2DM based on the American Diabetes Association (ADA) criteria. The second group was Control Group (n = 30), making 2.3:1 case-to-control ratio.

The selection criteria for participants, especially matching controls for age and sex, should be detailed comprehensively.

The matching between control and patient groups was done for age and sex to reduce the confounding variables. Moreover, the matching of the control subjects to diabetic patients was based on age (± 3-5 years) and sex. Inclusion Criteria include age in 30–70 years, confirmed diagnosis of T2DM for at least one year whereas For controls, the normal fasting blood glucose (<100 mg/dL) and HbA1c (<5.7%).

Exclusion Criteria including type one diabetes mellitus, gestational diabetes, acute or chronic infections, autoimmune or malignant diseases, recent surgery or trauma or use of corticosteroids, immunosuppressants, or anti-inflammatory medications within the past 3 months

Blood Sample Collection

This study was conducted according to the ethical principles as recorded by the Helsinki Declaration. All patients informed verbally about the aims of the study and and signed a consent form approved by the research and ethical committee/ college of dentistry/ Al-Iraqia university. After overnight fasting (8–10 hours), 5 mL of venous blood was collected from each participant into plain (clot activator) tubes. The blood samples were allowed to clot at room temperature and then centrifuged at 3000 rpm for 10 minutes. The serum was separated and stored at −80°C until further analysis [20], [21].

Cytokine analysis

The method of cytokine measurement via ELISA is described, but specifics such as assay sensitivity, intra- and inter-assay variability, and calibration procedures are missing.

The concentration of serum IL-6, TNF-α, and IL-10 levels were measured using a commercially available enzyme-linked immunosorbent assay (ELISA) kits (Rochi, Cobas e411, Roche Diagnostics, Germany), according to the manufacturer's instructions. The used ELISA kits were with high specificity and sensitivity for human cytokines [22].

Each sample was tested in duplicate to ensure reliability. Briefly, the ELISA procedures involved the incubation with cytokine-specific antibodies, then enzymatic detection using a TMB (tetramethylbenzidine) substrate. Eventually, the absorbance was measured at 450 nm using a microplate reader. The standard curves were produced using 7-points serial dilutions of concentrations that provided in the kit standards. The calibration range was as follows: IL6: 3.12-300 pg/ml, TNF-α:5-500 pg/ml, and IL-10:2.5-200 pg/ml. The standard curve was used to determine the sample concentrations by interpolating in the curve using 4-parameter logistic regression model. Additionally, the assay sensitivity/ the lower limit of detection (LOD) for each cytokine was < 2pg/ml, < 3pg/ml, < 2 pg/ ml for IL-6, TNF-α, and IL-10, respectively. All data were analyzed using SPSS software (version 20) and GraphPad Prism (Version 9). The intra-assay coefficient of variation (CV), displaying the variability within a single assay run was <8%, < 7%, <10% for IL-6, TNF-α, and IL-10, respectively. Continuous variables were presented as mean ± standard deviation (SD). Comparisons between diabetic patients and controls were performed using the independent samples t-test with p-value ≤ 0.05 was considered statistically significant [23].

Results and Discussion

The demographic analysis of the study participants showed that the mean ages of the diabetic and control groups were comparable, with no statistically significant difference observed (54.3 ± 8.7 years vs. 52.1 ± 7.9 years, p = 0.21) as show in Table 1 and Figure 1.

Table 1: Comparison of Age, BMI, and Serum Cytokine Concentrations between T2DM Patients and Controls.

Parameter	Diabetic Group (n = 70)	Control Group (n = 30)	p-value
Age (years)	54.3 ± 8.7	52.1 ± 7.9	0.21
BMI (kg/m²)	26.8 ± 3.4	24.4 ± 2.7	< 0.05
IL-6 (pg/mL)	8.2 ± 1.7	3.5 ± 1.2	< 0.05
TNF-α (pg/mL)	13.3 ± 7.2	5.5 ± 3.1	< 0.05
IL-10 (pg/mL)	4.6 ± 2.9	7.3 ± 3.3	0.011

This similarity in age distribution between the groups strengthens the validity of subsequent comparisons of immunological parameters, as it minimizes the potential confounding effect of age on inflammatory markers [24]. Age is a known factor that can influence immune function and cytokine levels, with older individuals often exhibiting increased systemic inflammation; thus, matching or having no significant difference in age between groups helps ensure that observed differences in cytokines are more likely attributable to diabetes status rather than age-related immune changes [25].

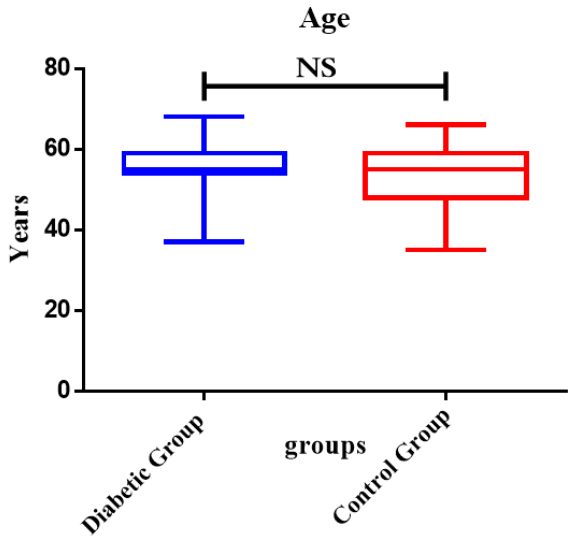

Fig. 1: Comparison of age distribution among study groups, P value ≤0.05

In contrast, body mass index (BMI) was significantly higher in the diabetic group (26.8 ± 3.4 kg/m²) compared to healthy controls (24.4 ± 2.7 kg/m²) (p < 0.05) as in Figure 2. This results aligns with the well-established relationship between obesity and type 2 diabetes mellitus [26]. Excess adiposity contributes to the pathogenesis of T2DM by promoting insulin resistance through several mechanisms, including the secretion of pro-

inflammatory cytokines by adipose tissue [27]. The higher BMI in diabetic patients likely reflects the role of obesity as a major risk factor and driver of metabolic dysregulation in this population [28]. Moreover, increased adiposity is associated with chronic low-grade inflammation, which can further exacerbate insulin resistance and contribute to the observed elevations in pro-inflammatory cytokines such as IL-6 and TNF-α in diabetic patients [29]. Consequently, the elevated BMI observed here not only corroborates the metabolic status of the diabetic cohort but also provides context for interpreting their altered immunological profiles [30].

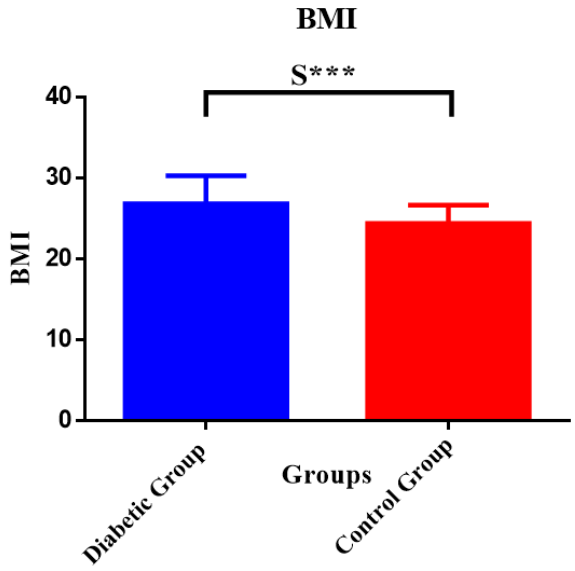

Fig. 2: Comparison of BMI between type 2 diabetic patients and healthy controls

The serum concentrations of IL-6, TNF-α, and IL-10 are presented in Table 1 and Figure 1. Mean IL-6 levels were significantly elevated in the diabetic group (8.2 ± 1.7 pg/mL) compared to the control group (3.5 ± 1.2 pg/mL), with a p-value < 0.05 as in Figure 3. Similarly, TNF-α levels were higher in diabetic patients (13.3 ± 7.2 pg/mL) than in controls (5.5 ± 3.1 pg/mL), showing a highly significant difference (p < 0.05) as shown in Figure 4. IL-10 levels were significantly lower in the diabetic group (4.6 ± 2.9 pg/mL) compared to the control group (7.3 ± 3.3 pg/mL), indicating a reduced anti-inflammatory response in T2DM patients (p: 0.011) as shown in Figure 5.

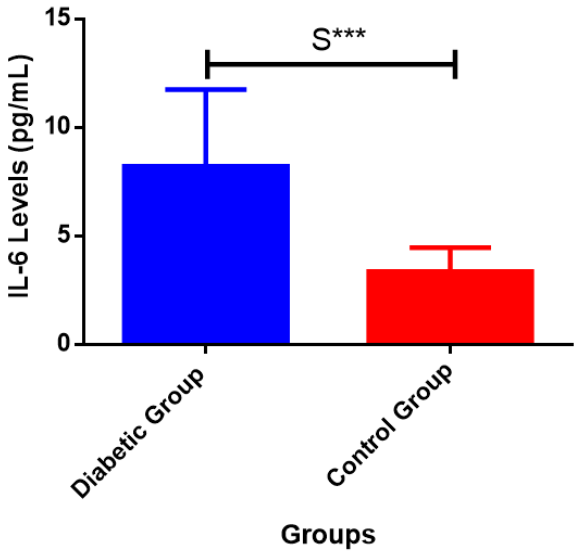

Fig. 3: IL-6 Levels in type 2 diabetic patients compared to healthy controls

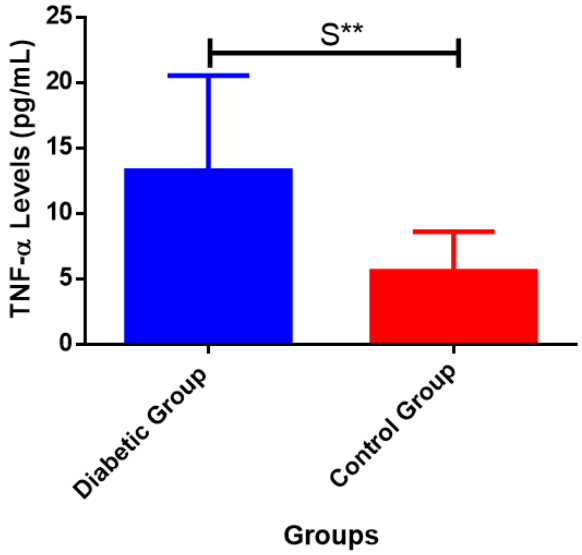

Fig. 4: TNF- α Level in type 2 diabetic patients compared to healthy controls

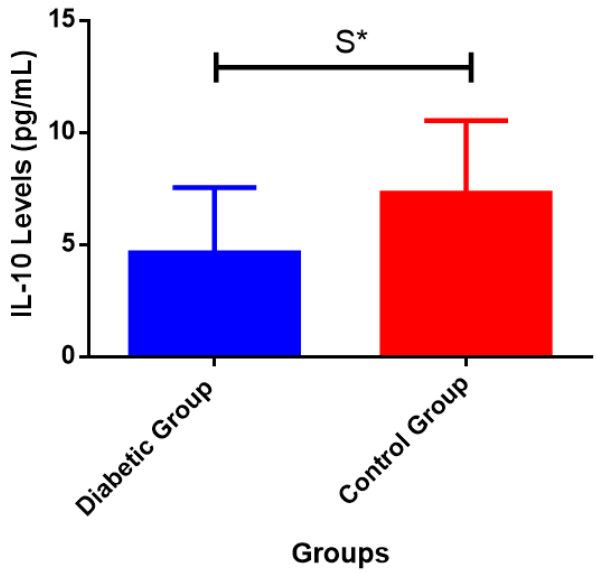

Fig. 5: IL-10 Level in type 2 diabetic patients compared to healthy controls

These results clearly indicate that individuals with T2DM exhibit a significant pro-inflammatory shift in their cytokine profile. Elevated levels of IL-6 and TNF-α suggest an active inflammatory process, which is a hallmark of insulin resistance and endothelial dysfunction in T2DM. Both cytokines are known to interfere with insulin receptor signaling pathways [31]. IL-6 promotes hepatic glucose output and inhibits insulin action, while TNF-α reduces insulin sensitivity through serine phosphorylation of insulin receptor substrate (IRS) proteins [32].

In contrast, IL-10 which is an important anti-inflammatory mediator and was found to be significantly reduced in diabetic patients. IL-10 plays a crucial role in limiting immune responses and preventing chronic inflammation [33]. The observed decrease in IL-10 levels suggests an impaired regulatory mechanism in T2DM, allowing pro-inflammatory processes to persist unchecked.

This cytokine imbalance (high IL-6 and TNF-α, low IL-10) not only reflects the inflammatory nature of diabetes but may also contribute to its chronic complications. Chronic inflammation accelerates the development of atherosclerosis, nephropathy, and other end-organ damage in diabetic individuals [34]. The results of the current study are consistent with previous research highlighting the role of inflammation in diabetes and support the concept of T2DM as an immunometabolic disorder [35]. This understanding opens avenues for using cytokines not just as biomarkers for diagnosis or disease monitoring but also as therapeutic targets. For example, drugs targeting IL-6 or TNF-α are being explored in clinical trials for metabolic and cardiovascular conditions associated with diabetes.

The cytokine profile observed in this study mirrors the immune dysregulation that underlies not only insulin resistance but also the micro-vascular and macro-vascular complications commonly observed in diabetes. Chronic inflammation contributes to endothelial dysfunction, atherosclerosis, and organ damage, emphasizing the clinical relevance of monitoring immune markers in diabetic populations [36].

However, several limitations should be noted. The cross-sectional nature of this study prevents establishing causality. The sample size, though sufficient to detect significant differences, may not capture the full variability of immune responses in larger or more diverse populations. Additionally, we did not examine correlations between cytokine levels and clinical parameters such as HbA1c, BMI, or duration of diabetes, which could provide further insights.

Conclusion

A distinct immunological profile in patients with T2DM were approved in the current study, accompanied with elevated IL-6 and TNF-α and reduced IL-10. These changes reflect a pro-inflammatory state and impaired immune regulation, contributing to the pathophysiology and complications of diabetes. The authors recommend more future studies that focus on monitoring cytokine levels to enhance clinical management and provide new therapeutic targets in the future.

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Conflict of Interest

Authors declare there is no conflict of interest

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مقالة بحثية

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المُلخّص

تزايد التعرف على الالتهاب المزمن واطئ الدرجة بشكل كبير كصفة مركزية في التسبب في داء السكري النوع الثاني. تلعب السيتوكينات المؤيدة والمضادة للالتهابات أدوارًا حاسمة في التوسط في التفاعلات المناعية الأيضية التي تؤثر على مقاومة الأنسولين وتطور المرض. تهدف هذه الدراسة إلى تقييم مستويات المصل من إنترلوكين-6، عامل نخر الورم ألفا-α وإنترلوكين-10 في المرضى الذين يعانون من داء السكري النوع الثاني مقارنة مع الأفراد الأصحاء أيضا لتقييم علاقتها مع العلامات السريرية مثل العمر ومؤشر كتلة الجسم. شملت هذه الدراسة المقارنة 100 مشارك: 70 مريضًا مُشخَّصًا بمرض السكري من النوع الثاني، و30 شخصًا سليمًا، معظمهم من مجموعة السيطرة، متطابقين في العمر والجنس. قُيّست مستويات السيتوكينات في المصل باستخدام مقايسة الممتز المناعي المرتبط بالإنزيم (الاليزا). وسجلت البيانات السريرية، بما في ذلك العمر ومؤشر كتلة الجسم. واجري التحليل الاحصائي لمقارنة مستويات السيتوكينات بين المجموعات، وتقيم ارتباطها بالعمر ومؤشر كتلة الجسم. أظهر مرضى السكري مستويات أعلى بكثير من إلانترلوكين - 6 (8.2 ± 1.7 بيكوغرام/مل) وعامل نخر الورم ألفا-α (13.3 7 7.2 بيكوغرام/مل) مقارنة مع السيطرة (3.5 1 1.2 بيكوغرام/مل و5.5 3 3.1 بيكوغرام/مل، على التوالي؛ وكانت قيمة -اف < 0.05). على العكس من ذلك، كانت مستويات الانترلوكين -10 أقل بشكل ملحوظ في مجموعة السكري (4.6 2 2.9 بيكوغرام/مل) مما كانت عليه في السيطرة (7.3 3 3.3 بيكوغرام/مل؛ ع = 0.011). بينما كان العمر متشابها بين المجموعات (ع = 0.21)، كان مؤشر كتلة الجسم أعلى بكثير في مرضى السكري (قيمة -اف < 0.001)، وارتبط بشكل إيجابي مع الانترلوكين-6 وعامل نخر الورم ألفا-α. اظهروا مرضى داء السكري النوع الثاني صورة خلوية غير متوازنة تتميز بارتفاع العوامل المؤيدة للالتهاب وقلة في المستقبلات الالتهابية والتي تتوافق مع حالة الالتهاب المزمن. يبدو أن هذه التغييرات مرتبطة بالسمنة أكثر من العمر. قد توفر مراقبة مستويات السيتوكين نظرة ثاقبة حول حالة التمثيل الغذائي المناعي لمرضى السكري ويمكن أن تكون بمثابة هدف للتدخل العلاجي.

الكلمات المفتاحية: داء السكري النوع الثاني، الانترلوكين -6، عامل نخر الورم ألفا-α، إنترلوكين-10، السايتوكينات الالتهابية، مؤشر كتلة الجسم، مقاومة الانسولين.

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