

# Exploring the Link between Iron Status and Catalase Activity in Type 2 Diabetes Mellitus

Esraa H. Oleiwi<sup>✉</sup> , Saba Z. Hussein\*<sup>✉</sup> 



© 2024 The Author(s). Published by College of Medicine, University of Baghdad. This open-access article is distributed under the terms of the Creative Commons Attribution 4.0 International License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

## Abstract:

**Background:** Catalase is an antioxidant enzyme found in all living organisms that is responsible for the degradation of hydrogen peroxide, a type of harmful compounds known as reactive oxygen species (ROS), into harmless oxygen and water. It is necessary for the cell protection from ROS-induced oxidative damage in type 2 diabetes mellitus (T2DM) individuals. T2DM is rapidly rising in prevalence worldwide, emerging as a significant public health challenge. It can disrupt iron regulation in the bloodstream, leading to the production of ROS, which can damage the cells.

**Objective:** To explore the correlation between iron status and catalase activity in T2DM patients, and the possibility of using it as a predictor for the onset and severity of diabetes.

**Patients and Methods:** One hundred and fifty participants were included in the study, comprising 50 healthy volunteers who served as the control group (C) and 100 cases diagnosed with T2DM who were divided into three groups based on the duration of their disease: Group A1 (n=38; < 5 years), A2 (n=37; 5-10 years) and A3 (n=25; >10 years). The participants were recruited from the Al-Kindi Teaching Hospital, Baghdad, during the period from October 2022 to the end of January 2023. The study assessed various blood markers in all participants, including: Fasting blood glucose (FBG), glycosylated hemoglobin A1c (HbA1c), Iron, total iron binding capacity (TIBC), unsaturated iron binding capacity (UIBC), ferritin, transferrin, saturation of transferrin (S.Trans) and catalase activity (CAT).

**Results:** Compared to the control group, patients with T2DM showed significantly higher levels of FBG, HbA1c, and ferritin, notably lower levels of iron, TIBC, UIBC, and S.Trans. Interestingly, only the A2 group had significantly lower transferrin levels compared to control. There was a significant decrease in catalase activity across all patient groups. Additionally, a positive correlation was observed between iron levels and catalase activity in all patient groups.

**Conclusion:** Increases in ferritin level might be a risk factor for developing T2DM. The observed association between lower iron levels and reduced catalase activity in T2DM is intriguing, and can serve as a future predictor for the onset and severity of diabetes, warranting further investigation.

**Keywords:** Catalase activity; Ferritin; Iron; Transferrin; Type 2 Diabetes.

Received: Feb. 2024  
Revised: June. 2024  
Accepted: July. 2024  
Published: Oct. 2024

## Introduction:

Iron (Fe) is an essential element that is necessary for health and appropriate physiological function for almost all living organisms. Iron's significant functions are oxygen transport and hematopoiesis. It can remarkably act as an electron donor and acceptor (1). Iron may also damage cells by catalyzing the production of free radicals and oxidative stress (2). Plasma transferrin has long been recognized to play an essential role in these activities, primarily by transporting iron in a soluble, non-toxic form across human tissues and organs (3). Transferrin is the body's iron-binding protein; its levels rise as iron demand increases. However,

serum iron is difficult to detect and analyze in isolation due to the diversity and consistency of variances in all body iron (4). On the other hand, many studies realize that serum iron affects glucose metabolism even when there is no appreciable iron overload or deficit (5). Serum ferritin levels are frequently used as an indicator of body iron storage, and in epidemiological research, greater ferritin levels and dietary iron consumption predict the possibility of type two diabetes mellitus (T2DM) (6,7). As an iron carrier, ferritin, a cytosolic protein, is released into the serum at trace levels (8). Moreover, ferritin is correlated with blood glucose levels and insulin resistance (9). Type 2 diabetes mellitus, a serious and chronic metabolic disorder marked by insufficient insulin production by the body or does not effectively respond to the insulin it produces, is becoming increasingly common and poses a serious threat to

\*Corresponding author: Dept. of Chemistry/ College of Science / University of Baghdad, Baghdad-Iraq  
[sa78ba2016@sc.uobaghdad.edu.iq](mailto:sa78ba2016@sc.uobaghdad.edu.iq)  
[esraahasanolaiwi@gmail.com](mailto:esraahasanolaiwi@gmail.com)

public health worldwide (10,11). Chronic hyperglycemia of T2DM is associated with the long-term harm, malfunction, and failure of several organs, including the eyes, kidneys, heart, nerves, and blood vessels (12,13). In Iraq, diabetes is the most common public health issue, where it is among the highest prevalence rates in the Middle East (14,15). The adult diabetes prevalence in Iraq is 10.4% which means that more than three million people have diabetes (16). Numerous human experimental research studies have provided evidence of a strong correlation between T2DM and changes in the metabolism of several trace elements. Low levels of trace elements, including iron, magnesium, chromium, and others, have been associated with insulin resistance, decreased insulin release, and glucose intolerance in T2DM. These trace elements are essential for optimal insulin synthesis and function (4, 17). Experts believe systemic iron excess to be a contributing factor to impaired glucose metabolism. T2DM is produced by oxidative stress on pancreatic  $\beta$ -cells, resulting in insulin insufficiency, cell death, and reduced insulin output. Alternatively, it can be caused by iron excess and hepatic dysfunction, leading to insulin resistance (18-20).

T2DM is a polygenic condition caused by insulin receptor defects and is characterized by oxidative stress, which involves the generation of reactive oxygen species (ROS). ROS can react with DNA, proteins, and lipids, causing these molecules to be destroyed (21). Iron and oxidative stress are inextricably related. Increased oxidative stress is frequently linked with T2DM patients, which can lower serum iron levels (22). The Fenton reaction produces highly hazardous free radicals such as hydroxide and the superoxide anion, which triggers lipid peroxidation. To operate as a pro-oxidant agent, iron must be accessible. Iron is liberated from ferritin by reducing agents, which convert  $\text{Fe}^{3+}$  to  $\text{Fe}^{2+}$  (23). Interestingly, iron is a potent oxidant that can accelerate the generation of a lot of reactive oxygen radicals, which in turn influence the secretion of insulin and disrupt the process of glucose metabolism by controlling the signal transduction of islet  $\beta$ -cells. Iron is also crucial for the mitochondria, which can influence insulin secretion levels and promote adenosine triphosphate (ATP) synthesis, both of which can result in disorders related to glucose metabolism (7,18). Moreover, iron influences insulin action reciprocally. It interferes with insulin action by inhibiting liver production of glucose. The insulin metabolism and hepatic extraction is reduced which leads to peripheral hyperinsulinemia, when iron stores increase (24).

The human body has many defense mechanisms that work together to prevent harm from ROS. These mechanisms are referred to as antioxidants. Included in these defense mechanisms are enzymatic antioxidants like glutathione peroxidase (GPx) and catalase (CAT), which both detoxify hydrogen peroxide ( $\text{H}_2\text{O}_2$ ), and superoxide dismutase (SOD), which breaks down superoxide anion ( $\text{O}_2^-$ ) (25). The

Se, Cu–Zn, and Fe metals are cofactors for the GPx, cytoplasmic SOD, and CAT enzymes, respectively (23,26). Catalase (EC 1.11.1.6; CAT) is a crucial component of the body's antioxidant defense, essential to protect cells from the detrimental effects of ROS. This enzyme is present in practically all living species. It protects against oxidative stress, which has been linked to a variety of clinical disorders such as diabetes, atherosclerosis, cataracts, cancer, ischemia/ reperfusion damage, nutritional deficiencies, and aging (27,28). Iron is a constituent of several metalloenzymes, including catalase (EC 1.11.1.6) and peroxidase (EC 1.11.1.7) (29). So, both enzymes could be used as biochemical indicators of iron availability. Thus, understanding the relationship between iron status and catalase activity in T2DM patients may provide valuable insights into the underlying mechanisms of the disease and potentially open possibilities for novel therapeutic interventions.

#### Cases and Methods:

According to the standard American Association criteria, a total of 100 patients with T2DM, between 20-70 years of age were divided into three groups based on the duration of diabetes:

Group1: A1 consists of (38) patients who had diabetes (<5 years)

Group2: A2 consists of (37) patients who had diabetes (5-10 years)

Group 3: A3 consists of (25) patients who had diabetes (>10 years)

Fifty age-matched healthy volunteers were enrolled as the control (C) group. The patients and controls were recruited from Al-Kindi Teaching Hospital, Baghdad, during the period from October 2022 to the end of January 2023 and were diagnosed by the physician specialist to be free of diabetic complications for diabetes patients and free of diabetes for controls.

**Exclusion criteria:** T1DM, alcohol drinking, smoking, and other complications of the disease, such as anemia, hepatitis, retinopathy, neuropathy, nephropathy, and cardiovascular diseases (CVD) have been excluded, as they may be confounders of the variables addressed in this study.

Blood samples (7 ml) were obtained from the patients and controls after fasting overnight for (8-12) hours. The blood samples were separated into aliquots (2 and 3 ml). The first aliquot was placed in a tube containing EDTA to calculate HbA1c. The second aliquot was placed in a plain gel tube and allowed to stand at room temperature for 10 minutes before being centrifuged at 3000 rpm for 10 minutes to collect serum, which was transferred to Eppendorf tubes and stored at  $-20^\circ\text{C}$  until tested.

Fasting blood glucose (FBS) was quantified using an enzymatic colorimetric technique using a commercially available kit (Spinrecat, Spain). The HbA1c was measured by immunoturbidimetric assay with an automatic analyzer (Spinrecat, Spin 240) for the directed kit (PZ Cormay, Poland). Iron and total iron-binding capacity (TIBC) concentrations in

serum were determined using kits (Human, Germany). The UIBC was determined using the following equation:

$$\text{UIBC } (\mu\text{g/dl}) = \text{TIBC } (\mu\text{g/dl}) - \text{Iron conc. } (\mu\text{g/dl})$$

The transferrin can be determined indirectly by using the following equation (30):

$$\text{Transferrin } (\mu\text{g/dl}) = 0.7 \times \text{TIBC } (\mu\text{g/dl})$$

The saturation of transferrin with Iron was determined using the following equation:

$$\text{Saturation of transferrin } (\%) = (\text{Iron conc.} / \text{TIBC}) \times 100$$

Using an available Cobas kit, ferritin serum levels were analyzed using an immunoturbidimetric by an automatic platform (Cobas C311- Germany). The CAT activity in human serum was measured using a spectrophotometric method dependent on ammonium molybdate (31).

**Statistical analysis:** The data were analyzed using SPSS (version 22) and presented in mean  $\pm$  standard deviation ( $\pm$ SD). A one-way analysis of variance (ANOVA) was used to compare the groups. The difference between groups was statistically highly significant if the  $p < 0.01$ , significant if the  $p < 0.05$ , and non-significant if the  $p > 0.05$ . Pearson correlation coefficient was used to determine the correlations between variables.

**Results:**

Table 1 shows the demographic characteristics of all studied groups. The controls were 50% males and 50% females; while the patients were distributed as follows: Males and females were 58% and 42% in A1, 43% and 57% in A2, 48% and 52% in A3.

**Table 1: The demographic characteristics of the study groups**

| Characteristics    | Groups        |                  |                 |                  |                  |
|--------------------|---------------|------------------|-----------------|------------------|------------------|
|                    | C             | A1               | A2              | A3               |                  |
| Number             | n=50          | n=38             | n=37            | n=25             |                  |
| Age (year)         | Mean $\pm$ SD | 43.1 $\pm$ 11.05 | 42.8 $\pm$ 9.89 | 54.3 $\pm$ 10.47 | 57.2 $\pm$ 11.24 |
| Sex                | Male (%)      | 25 (50)          | 22 (58)         | 16 (43)          | 12 (48)          |
|                    | Female (%)    | 25 (50)          | 16 (42)         | 21 (57)          | 13 (52)          |
| DM Duration (year) | Mean $\pm$ SD | -                | 2.0 $\pm$ 1.12  | 7.9 $\pm$ 2.24   | 17.4 $\pm$ 5.66  |

Table 2 illustrates the mean FBG and HbA1c in patients and control groups. The results showed significantly higher FBG levels in A1, A2, and A3 groups compared to the controls. In addition, they were significantly higher in A2 and A3 groups than

in A1. The HbA1c levels in groups A1, A2, and A3 were significantly higher than those of the control group, and A3 showed a significantly higher level compared to A1.

**Table 2: The mean $\pm$ SD FBG and HbA1c levels of all study groups**

| Parameters      | Groups          |                                  |                                   |                                   |
|-----------------|-----------------|----------------------------------|-----------------------------------|-----------------------------------|
|                 | C (n=50)        | A1 (n=38)                        | A2 (n=37)                         | A3 (n=25)                         |
| (Mean $\pm$ SD) |                 |                                  |                                   |                                   |
| FBG (mg/dl)     | 92.2 $\pm$ 8.58 | 186.6 $\pm$ 78.34 <sup>a**</sup> | 228.7 $\pm$ 72.09 <sup>ab**</sup> | 237.4 $\pm$ 89.02 <sup>ab**</sup> |
| HbA1c %         | 4.4 $\pm$ 0.79  | 7.7 $\pm$ 2.05 <sup>a**</sup>    | 7.9 $\pm$ 1.93 <sup>a**</sup>     | 8.7 $\pm$ 1.32 <sup>a**b*</sup>   |

\* $p < 0.05$ , \*\* $p < 0.01$

a: significant difference between C with A1, A2 and A3

b: significant difference between A1 with A2 and A3

The Iron status parameters, including iron, TIBC, UIBC, transferrin, saturation transferrin (S.Trans), and ferritin in patients and control groups, are presented in Table 3. A highly significantly lower serum iron in all study groups (A1, A2 and A3) was detected compared to the controls. Compared to groups C and A1, a substantially lower TIBC level was found in groups A2 and A3. Non-significant differences were found between UBIC levels for all patient groups compared to the controls, while groups

A2 and A3 showed a considerably lower level compared to the A1.

In A2, a highly significantly lower transferrin level was found compared to the controls. Compared to group C, a highly significantly lower S.Trans level was found in A1 and A2 groups. Moreover, ferritin levels were significantly higher in A1 and A2 groups compared to the controls, table 3.

**Table 3:- The Iron status parameters of all study groups**

| Parameters<br>(Mean±SD) | Groups        |                              |                                |                               |
|-------------------------|---------------|------------------------------|--------------------------------|-------------------------------|
|                         | C (n=50)      | A1 (n=38)                    | A2 (n=37)                      | A3 (n=25)                     |
| Iron (µg/dl)            | 86.6±27.52    | 66.8 ± 25.45 <sup>a**</sup>  | 64.6 ± 23.31 <sup>a**</sup>    | 73.4 ± 30.28 <sup>a*</sup>    |
| TIBC (µg/dl)            | 314.0 ± 40.33 | 314.7 ± 61.51                | 283.0 ± 58.18 <sup>a**b*</sup> | 290.5 ± 54.38 <sup>a*b*</sup> |
| UIBC (µg/dl)            | 226.9 ± 49.71 | 246.2 ± 70.55                | 217.7 ± 59.38 <sup>b*</sup>    | 217.1 ± 64.96 <sup>b*</sup>   |
| Transferrin (µg/dl)     | 220.7 ± 29.41 | 215.4 ± 50.60                | 197.7 ± 40.90 <sup>a**</sup>   | 202.99 ± 40.31                |
| S.Trans (%)             | 28.1 ± 9.55   | 21.9 ± 8.65 <sup>a**</sup>   | 23.4 ± 9.86 <sup>a*</sup>      | 26.5 ± 12.44                  |
| Ferritin (ng/ml)        | 111.2 ± 33.43 | 150.3 ± 53.51 <sup>a**</sup> | 132.4 ± 50.45 <sup>a*</sup>    | 128.1 ± 35.32                 |

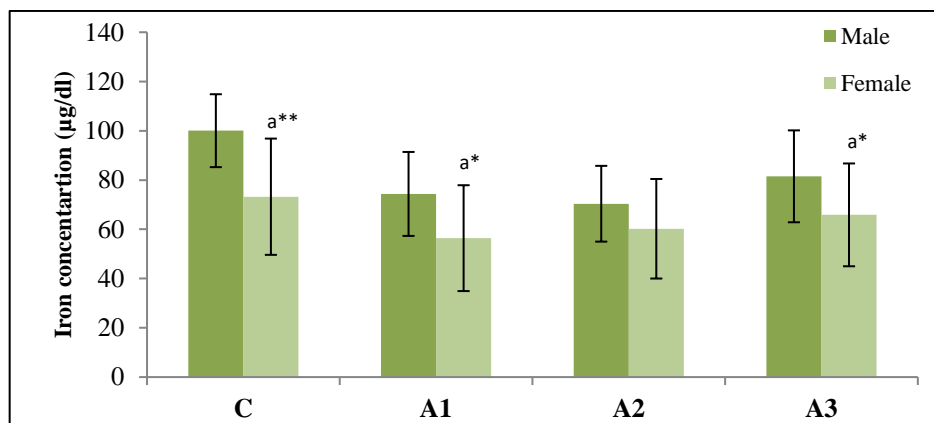
\* $p < 0.05$ , \*\* $p < 0.01$

<sup>a</sup> significant difference between C with A1, A2 and A3

<sup>b</sup> significant difference between A1 with A2 and A3

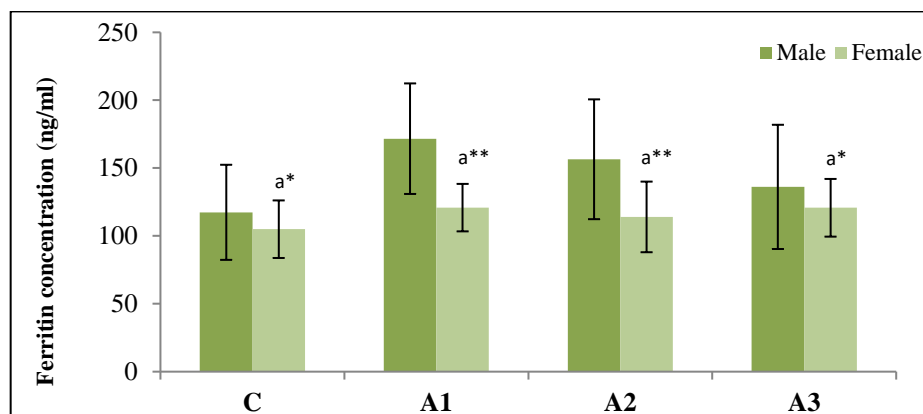
The differences between males and females regarding iron and ferritin levels are presented in figures 1 and 2, respectively. The result indicated a statistically significant difference in iron and ferritin levels

between males and females in all groups (C, A1, A2 and A3), showing that iron and ferritin levels in females were lower than in males.



**Figure 1:** Serum iron level in males and females in patient and control groups

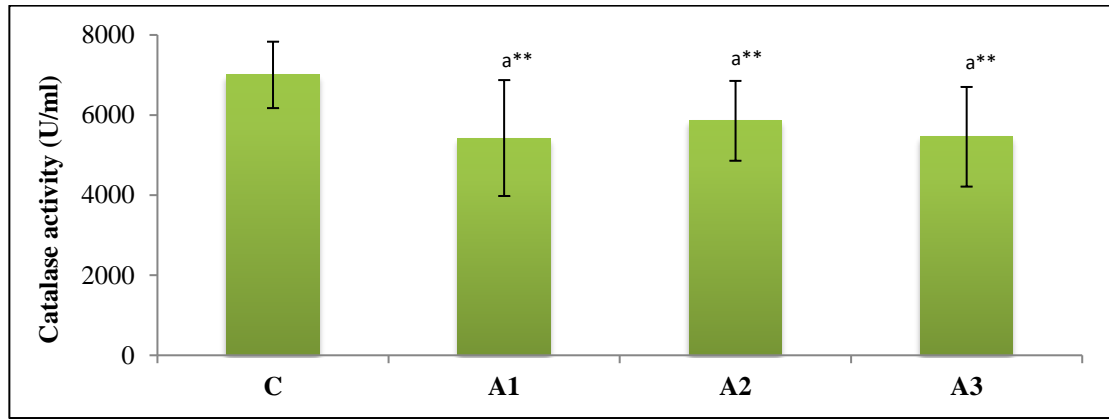
\* $p < 0.05$ , \*\* $p < 0.01$ ; <sup>a</sup> significant difference between males and females in the same group



**Figure 2:** Serum ferritin level in males and females in patient and control groups

\* $p < 0.05$ , \*\* $p < 0.01$ ; <sup>a</sup> significant difference between males and females in the same group

The activity of CAT in the A1, A2, and A3 groups was significantly lower than in the control group, as shown in figure 3.



**Figure 3:** Serum catalase activity (U/ml) in all study groups

\* $p < 0.05$ , \*\* $p < 0.01$ ; <sup>a</sup> significant difference between C with A1, A2 and A3

The correlation between all variables in the patient groups A1, A2, and A3 are illustrated in Tables 4, 5, and 6, respectively. In the A1 group, a highly significant positive correlation was found between (FBG with HbA1c), (iron with S.Trans), (TIBC with UIBC), (UIBC with transferrin) and (TIBC and transferrin). Also, a highly significant negative correlation was observed between S.Trans with TIBC, UIBC and transferrin. A significant negative correlation was reported between iron and UIBC, while a significant positive correlation was found between iron and CAT activity.

**Table 4:- Pearson correlation coefficients between the biochemical parameters in the A1 group**

| Parameters  | HbA1c   | Iron  | TIBC   | UIBC    | Transferrin | S.Trans  | Ferritin | CAT    |
|-------------|---------|-------|--------|---------|-------------|----------|----------|--------|
| FBG         | 0.701** | 0.273 | 0.127  | 0.007   | 0.124       | 0.154    | -0.023   | -0.056 |
| HbA1c       |         | 0.086 | -0.074 | -0.093  | -0.019      | 0.068    | -0.007   | -0.008 |
| Iron        |         |       | 0.046  | -0.375* | -0.040      | 0.818**  | 0.084    | 0.359* |
| TIBC        |         |       |        | 0.872** | 0.883**     | -0.415** | -0.200   | -0.044 |
| UIBC        |         |       |        |         | 0.796**     | -0.706** | -0.202   | -0.127 |
| Transferrin |         |       |        |         |             | -0.440** | -0.140   | -0.014 |
| S.trans     |         |       |        |         |             |          | 0.226    | 0.120  |
| Ferritin    |         |       |        |         |             |          |          | 0.141  |

\* $p < 0.05$ ; \*\*  $p < 0.01$

Table 5, shows clearly that there is a highly significant positive correlation between (FBG and HbA1c), (iron and S.Trans), (TIBC and UIBC), (TIBC and transferrin) and (UIBC and transferrin). A

highly significant negative correlation was observed between UIBC and S.Trans, while a significant positive correlation was noticed between iron and CAT activity in the A2 group

**Table 5: Pearson correlation coefficients between the biochemical parameters in the A2 group**

| Parameters  | HbA1c   | Iron   | TIBC   | UIBC    | Transferrin | S.Trans  | Ferritin | CAT    |
|-------------|---------|--------|--------|---------|-------------|----------|----------|--------|
| FBG         | 0.490** | -0.015 | -0.055 | -0.048  | -0.068      | -0.020   | -0.241   | 0.170  |
| HbA1c       |         | 0.059  | -0.002 | -0.024  | -0.031      | 0.049    | -0.041   | -0.023 |
| Iron        |         |        | 0.241  | -0.238  | 0.240       | 0.880**  | 0.168    | 0.330* |
| TIBC        |         |        |        | 0.883** | 0.999**     | -0.201   | -0.262   | 0.125  |
| UIBC        |         |        |        |         | 0.882**     | -0.624** | -0.320   | -0.038 |
| Transferrin |         |        |        |         |             | -0.202   | -0.262   | 0.125  |
| S.trans     |         |        |        |         |             |          | 0.309    | 0.245  |
| Ferritin    |         |        |        |         |             |          |          | 0.053  |

\* $p < 0.05$ ; \*\*  $p < 0.01$

A highly significant positive correlation was found between (iron with S.Trans), (TIBC with UIBC), (TIBC with transferrin) and (UIBC with transferrin). A highly significant negative correlation was found

between (iron with UIBC) and S.Trans with TIBC, UIBC and transferrin. A significant positive correlation was noticed between iron and CAT activity in the A3 group.

**Table 6:** Pearson correlation coefficients between the biochemical parameters in the A3 group

| Parameters  | HbA1c | Iron   | TIBC   | UIBC     | Transferrin | S.Trans  | Ferritin | CAT    |
|-------------|-------|--------|--------|----------|-------------|----------|----------|--------|
| FBG         | 0.118 | 0.063  | -0.269 | -0.254   | -0.204      | 0.189    | -0.410   | -0.117 |
| HbA1c       |       | -0.072 | 0.296  | 0.282    | 0.329       | -0.169   | -0.085   | -0.036 |
| Iron        |       |        | -0.105 | -0.554** | -0.131      | 0.864**  | 0.178    | 0.301* |
| TIBC        |       |        |        | 0.886**  | 0.980**     | -0.567** | 0.041    | -0.335 |
| UIBC        |       |        |        |          | 0.882**     | -0.878** | -0.049   | -0.187 |
| Transferrin |       |        |        |          |             | -0.575** | -0.055   | -0.317 |
| S.trans     |       |        |        |          |             |          | 0.125    | -0.004 |
| Ferritin    |       |        |        |          |             |          |          | -0.144 |

\* $p < 0.05$ ; \*\*  $p < 0.01$

### Discussion:

The most common non-communicable disease in the world that may lead to death is DM. Reports indicate that over 50% of DM cases worldwide are undiagnosed (32). The global prevalence of diabetes in 2019 is estimated to be 9.3% (463 million people). By 2030, it will increase to 10.2% (578 million), and by 2045, it will reach 10.9% (700 million) cases (33). The IDF reports that T2DM was the most common type of diabetes among people in 2011 (5), accounting for 90% of all diabetes cases worldwide (34). Iraq has an extremely high prevalence of diabetes, with one in ten persons suffering from T2DM (35). Thus, diabetes is a serious public health problem for Iraqis due to its high prevalence rate, growing incidence rate, and financial burden.

The FBG level for T2DM patients in the current study was higher than the control; consistence with other studies (36,37). One of the most effective indicators of the complications risk, including kidney and cardiovascular diseases, is the duration of diabetes. It was found that the incidence of severe hyperglycemia rises with age and disease duration (38). The blood glucose level and the red blood cells' lifetime affect the level of HbA1c in the blood (39). Based on HbA1c and FBG concentrations, our study revealed substantial changes in glycemic control in the three diabetic study groups. There is a chance that the illness will worsen as it becomes more chronic, consistent with earlier studies (40). The current study showed that T2DM patients had significantly higher HbA1c levels than healthy controls, with an exceptionally high level in those with the longest duration of DM. This is consistent with a prior study by Ito et al., who reported that HbA1c values rise with longer disease duration, both in older and younger diabetics (41). The higher FBG levels were linked to the increase in HbA1c levels in A1 and A2 groups, in the current study. Zhu et al. (42) also found a positive correlation between FBG and HbA1c. The American Diabetes Association (ADA) recommended HbA1c

levels of 5.7-6.4% for prediabetes diagnosis and 6.5% or higher for diabetes progression (43).

According to earlier studies, T2DM and iron metabolism are intricately related. Iron levels have been shown to influence glucose metabolism, and glucose metabolism influences several iron metabolic processes. Iron and the metabolism of glucose have a reciprocal interaction. Further factors that affect and amplify these interrelated processes are oxidative stress and inflammatory cytokines (44). One of the vital trace elements for the human body is Iron. Three to five grams of Iron are found in the body, and the body mainly controls iron levels through absorption. Body dysfunction may result from either an excess or a deficiency of Iron (45). The results of the present study showed that healthy controls had significantly higher iron levels than T2DM patients in all three groups, but it remains within the normal range. These findings are in agreement with those of previous studies (46,47). The decrease in serum iron levels in people who have T2DM can be caused by several factors, including the metabolic disruptions in T2DM which may result in the "iron resistance" phenotype, which can cause signals that regulate iron homeostasis to become dysregulated (48). T2DM patients may encounter iron deficiency anemia, which can lead to lower serum iron levels (49). These results contrast other studies that showed higher iron levels in T2DM cases than in healthy controls (50-52). The current study pointed to the possible role of iron in the etiology of T2DM and the numerous difficulties induced by free iron in T2DM patients.

Ferritin is the main intracellular iron storage protein and has been identified as a biomarker of inflammation and body iron storage (53). The current study showed that serum ferritin in T2DM patients is significantly higher than in controls in consistence with previous studies (19,54,55). However, its level has no correlation with blood sugar or HbA1c in diabetic patients. These findings agreed with the results of Thilipkumar et al. (56). Ferritin as an acute-

phase reactant which may indicate inflammation. Delayed clearance of glycosylated ferritin in diabetics may result in elevated ferritin levels, which may represent elevated iron stores; which are a few possible explanations for elevated ferritin levels in T2DM patients (57,58). The ferritin level was exceptionally high in the A1 group in the current study in agreement with an earlier study that reported a substantial correlation between serum ferritin level and newly diagnosed diabetes (59). Memon et al. (60) reported that T2DM patients with poor glycemic control more frequently had elevated serum ferritin levels compared to patients with good glycemic control. This observation can explain the findings that the long duration of the disease had lower ferritin levels compared to the short disease duration, which may be due to the patient's good glycemic control. Serum ferritin levels are frequently correlated with insulin resistance indicators, such as higher blood glucose and insulin levels. On the other hand, some studies are increasingly recognizing that serum iron affects glucose metabolism, even without severe iron overload or lack of iron (61). Therefore, high iron stores can predict diabetes development, according to previous epidemiological studies. Iron converts reactive free radicals into highly reactive ones. As the serum ferritin level increases, it affects insulin synthesis and secretion by the pancreas and interferes with the insulin-extraction capacity of the liver. Deposition of iron in muscles leads to muscle damage and decreases glucose uptake (62). Also, the glycation of transferrin stimulates ferritin synthesis by decreasing its capacity to bind ferrous iron and increasing the quantity of free iron (24).

The most significant molecule for delivering iron into cells is serum transferrin, a glycoprotein with two iron-binding domains. Transferrin is primarily produced in the liver and performs various tasks, including intracellular iron transport, iron transport across the intestinal mucosa, and non-specific defiance against microbes through the chelation of free iron (30,63). Our study showed that T2DM patients had a significantly lower serum transferrin concentration than healthy controls in the A2 group only. Transferrin is highly correlated with TIBC in all duration groups. T2DM incidence and a low soluble transferrin receptor-to-ferritin ratio level were observed by Arija et al. (64), but there was no correlation with the soluble transferrin receptor. The direct relationship between serum transferrin and T2DM has been the subject of very few investigations. Serum transferrin is prone to leaking from glomeruli due to its molecular weight and negative charges. Consequently, tracking urine transferrin levels may help assess the development of diabetic complications like diabetic nephropathy early on (45). Total iron-binding capacity (TIBC) is a total quantity of iron measurement that blood proteins are capable of binding, which aids in assessing the body capacity to bind and move iron through the blood. It equals UIBC plus the serum iron level. While low levels may signal iron overload, high

TIBC levels may indicate iron deficiency (48). In iron-deficient conditions, the relative transferrin content compared to iron content increases, and thus, the TIBC values are high. For this reason, the increase in TIBC levels in this study may be due to the decrease in iron levels in T2DM patients.

The concentration of iron and ferritin levels in male T2DM is higher than in females. Manikandan et al. (24) reported that the differences between sexes may be because most females are anemic due to physiological processes like pregnancy and menstruation. This result is consistent with Han et al. (65) and Al Akl et al. (66) studies, which found a statistically significant positive connection between blood ferritin levels and diabetes, metabolic syndrome, and obesity in male patients rather than female patients.

It has been demonstrated that  $H_2O_2$  acts as an oxidant and damages the  $\beta$ -cell interrupting the signaling pathway of insulin production. A previous study reported that a four-fold increase in the  $H_2O_2$  concentration was observed in T2DM patients than in healthy individuals, and the observation was corroborated with observations of low CAT activity in the  $\beta$ -cells of hyperglycemic mice models. Thus, the lack of CAT, which is responsible for degrading the  $H_2O_2$  to water and oxygen, can contribute to the development of DM (24). The present study found that CAT activity was lower in all T2DM study groups than in healthy controls. Prior studies reported lower plasma iron concentrations and CAT activity in T2DM, consistent with our findings (8,67). Oxidation plays an important role in different T2DM complications. Because of the CAT low levels or activity, cells may have higher concentrations of hydrogen peroxide which leads to oxidative stress conditions leading to the progression of different complication types (24).

Our results indicated a significant positive correlation between CAT activity and iron concentration in all patient groups. One of the T2DM risk factors is increasing oxidative stress with a reduction in total antioxidant capacity (TAC) reported by Mahmood (17), in contrast to increased iron levels. One of the reasons for decreased CAT activity in T2DM is genetic factors; reduced blood CAT activity brought on by the many CAT mutations found in diabetic individuals may result in higher blood and tissue amounts of hydrogen peroxide. The oxidation-sensitive pancreatic  $\beta$ -cells may be harmed by these elevated hydrogen peroxide levels, which would reduce insulin synthesis (67). Since CAT needs iron for its catalytic activity, the decrease in CAT activity in our research might be caused by a reduction in iron levels. According to these findings, CAT deficiency may be a risk factor for T2DM. However, applying the findings to all T2DM patients is not suitable. To better understand the association between iron levels and CAT activity in T2DM patients and the applicability of using this correlation as a predictor for both the onset and severity of diabetes, additional

research with a larger sample size and collecting samples from different regions of Iraq is required.

**Conclusions:**

Iron plays a significant role in the pathogenesis and complications of T2DM patients. Increases in ferritin levels might be a risk factor for developing T2DM. The observed association between lower iron levels and reduced catalase activity in T2DM is intriguing and can serve as a future predictor for the onset and severity of diabetes, warranting further investigation.

**Authors' declaration:**

We confirm that all the Figures and Tables in the manuscript are ours. Besides, the figures and images, which are not ours, have been given permission for re-publication attached to the manuscript. Authors sign on ethical consideration's approval-Ethical Clearance: The project was approved by the local ethical committee in (College of Science, University of Baghdad) according to the code number (Ref. CSEC/0123/0012, Date: 27.01.2023).

**Conflicts of Interest: None**

**Authors' Contribution:**

Study conception & design: (Saba Z. Hussein & Esraa H. Oleiwi). Literature search: (Esraa H. Oleiwi). Data acquisition: (Esraa H. Oleiwi). Data analysis & interpretation: (Esraa H. Oleiwi). Manuscript preparation: (Saba Z. Hussein & Esraa H. Oleiwi). Manuscript editing & review: (Saba Z. Hussein & Esraa H. Oleiwi). Funding acquisition: (Esraa H. Oleiwi).

**References:**

1. Kim JD, Lim DM, Park KY, Park SE, Rhee EJ, Park CY, et al. Serum Transferrin Predicts New-Onset Type 2 Diabetes in Koreans: A 4-Year Retrospective Longitudinal Study. *Endocrinology and Metabolism*. 2020;35:610-617. <https://doi.org/10.3803/EnM.2020.721>.
2. Galaris D, Barbouti A, Pantopoulos K. Iron homeostasis and oxidative stress: An intimate relationship. *Biochimica et Biophysica Acta (BBA) - Molecular Cell Research*. 2019;1866(12):118535. <https://doi.org/10.1016/j.bbamcr.2019.118535>.
3. Elsayed ME, Sharif MU, Stack AG. Transferrin Saturation: A Body Iron Biomarker. *Advances in Clinical Chemistry*. 2016;75:71-97. <https://doi.org/10.1016/bs.acc.2016.03.002>.
4. Saed HHM, Ali ZA. Correlation and comparison of some trace elements and their related proteins in Type 2 Diabetes mellitus in Kalars city/Kurdistan-Iraq. *Al-Qadisiyah Journal of Pure Science*. 2022;27(1):17-31. <https://doi.org/10.29350/jops.2022.27.2.1493>.
5. Fillebeen C, Lam NH, Chow S, Botta A, Sweeney G, Pantopoulos K. Regulatory Connections between

*Iron and Glucose Metabolism. Int J Mol Sci*. 2020;21(20):1-18.

<https://doi.org/10.3390/ijms21207773>.

6. Arumugam S, Suyambulingam A. Association between Serum Ferritin and the Duration of Type 2 Diabetes Mellitus in a Tertiary Care Hospital in Chennai. *Cureus*.2024;16(1): e53117.

<https://doi.org/10.7759/cureus.53117>.

7. Liu J, Li Q, Yang Y, Ma L. Iron metabolism and type 2 diabetes mellitus: A meta-analysis and systematic review. *Journal of Diabetes Investigation*. 2020;11: 946–955.

<https://doi.org/10.1111/jdi.13216>.

8. Kuba RH, Saheb EJ, Mosa IS. Detection of Iron and Ferritin in Diabetes Mellitus Type 2 Patients. *Malaysian Journal of Medicine and Health Sciences*. 2022;18(Supp 4):7-10.

[https://medic.upm.edu.my/upload/dokumen/202203151721452\\_0149.pdf](https://medic.upm.edu.my/upload/dokumen/202203151721452_0149.pdf).

9. Hamed AME, El Hadidy KE, Salem MN. Ferritin as a marker of Insulin Resistance in Diabetic Patients. *Egyptian Journal of Medical Research (EJMR)*. 2022;3(2):110-120.

<https://doi.org/10.21608/EJMR.2022.237765>.

10. Hasankhani MB, Mirzaei H, Karamoozian A. Global trend analysis of diabetes mellitus incidence, mortality, and mortality-to-incidence ratio from 1990 to 2019. *Scientific Reports*. 2023; 13: 1-8.

<https://doi.org/10.1038/s41598-023-49249-0>.

11. Dilworth L, Facey A, Omoruyi F. Diabetes Mellitus and Its Metabolic Complications: The Role of Adipose Tissues. *International Journal Molecular Sciences*. 2021;22(14):1-18.

<https://doi.org/10.3390/ijms22147644>.

12. AL-Mohammad HMK, Jawad MM, Ali HA, Hassan F. Role Iron in Diabetes mellitus type 2 patients in province Diwaniya. *Al-Kindy College Medical Journal*. 2017;13(1):63-65.

<https://doi.org/10.47723/kcmj.v13i1.125>.

13. Demir S, Nawroth PP, Herzig S, Ekim Üstünel B. Emerging Targets in Type 2 Diabetes and Diabetic Complications. *Adv Sci (Weinh)*. 2021;8(18):1-23.

<https://doi.org/10.1002/advs.202100275>.

14. Abusaib M, Ahmed M, Nwayyir HA, Alidrisi HA, Al-Abbood M, Al-Bayati A, et al. Iraqi Experts Consensus on the Management of Type 2 Diabetes/Prediabetes in Adults. *Clin Med Insights Endocrinol Diabetes*. 2020;13:1-11.

<https://doi.org/10.1177/1179551420942232>.

15. Abuyassin B, Laher I. Diabetes epidemic sweeping the Arab world. *World Journal of Diabetes*. 2016; 7(8):165.

<https://doi.org/10.4239/wjd.v7.i8.165>.



16. Al-Attaby AKT, Al-Lami MQD. Role of calcium-regulating hormones, adipocytokines and renal function test in progressing type 2 diabetes mellitus in a sample of Iraqi patients. *Iraqi Journal of Agricultural Sciences*. 2019; 50(1): 343-351. <https://doi.org/10.36103/ijas.v50i1.300>.
17. Mahmood AR. Estimation of oxidative stress and some trace elements in Iraqi men patients with type 2 diabetes mellitus. *Iraqi Journal of Pharmaceutical Sciences*. 2016;25(1):17-22. <https://doi.org/10.31351/vol25iss1pp17-22>.
18. Blesia V, Patel VB, Al-Obaidi H, Renshaw D, Zariwala MG. Excessive Iron Induces Oxidative Stress Promoting Cellular Perturbations and Insulin Secretory Dysfunction in MIN6 Beta Cells. *Cells*. 2021;10(5):1-20. <https://doi.org/10.3390/cells10051141>.
19. Miao R, Fang X, Zhang Y, Wei J, Zhang Y, Tian J. Iron metabolism and ferroptosis in type 2 diabetes mellitus and complications: mechanisms and therapeutic opportunities. *Cell Death & Disease*. 2023;14(3):1-9. <https://doi.org/10.1038/s41419-023-05708-0>.
20. Harrison AV, Lorenzo FR, McClain DA. Iron and the Pathophysiology of Diabetes. *Annual Review of Physiology*. 2023; 85:339-362. <https://doi.org/10.1146/annurev-physiol-022522-102832>.
21. Malghooth HD, Tahmasebi P, Hamzah NA. Catalase gene Polymorphisms of type 2 Diabetes Patients in Al-Diwaniyah Province, Iraq. *Biochemical and Cellular Archives*. 2021; 21(2):3429-3434. <https://doi.org/connectjournals.com/03896.2021.21.3429>.
22. Angelovski M, Spirovska M, Nikodinovski A, Stamoski A, Atanasov D, Mladenov M. et al. Serum Redox Markers in Uncomplicated type 2 Diabetes Mellitus Accompanies with Abnormal Iron Levels. *Central European Journal for Public Health*. 2023;31(2): 133-139. <https://doi.org/10.21101/cejph.a7399>.
23. Imam MU, Zhang S, Ma J, Wang H, Wang F. Antioxidants Mediate both Iron Homeostasis and Oxidative Stress. *Nutrients*. 2017;9(7):671. <https://doi.org/10.3390/nu9070671>.
24. Manikandan A, Ganesh M, Silambanan S. Study of Iron Status in Type 2 Diabetes Mellitus. *International Journal of Clinical Biochemistry and Research*. 2015;2(2):77-82. <https://api.semanticscholar.org/CorpusID:76494397>
25. Eddaikra A, Eddaikra N. Endogenous Enzymatic Antioxidant Defense and Pathologies. *Antioxidants - Benefits, Sources, Mechanisms of Action*. IntechOpen. 2021. <http://dx.doi.org/10.5772/intechopen.95504>.
26. Junsi M, Takahashi Yupanqui C, Usawakesmanee W, Slusarenko A, Siripongvutikorn S. *Thunbergia laurifolia* Leaf Extract Increased Levels of Antioxidant Enzymes and Protected Human Cell-Lines In Vitro Against Cadmium. *Antioxidants (Basel)*. 2020;9(1):1-17. <https://doi.org/10.3390/antiox9010047>
27. Krishna H, Avinash K, Shivakumar A, Al-Tayar NGS, Shrestha AK. A quantitative method for detecting and validating catalase activity at physiological concentration in human serum, plasma and erythrocytes. *Spectrochim Acta A Mol Biomol Spectrosc*. 2021;251:119358. <https://doi.org/10.1016/j.saa.2020.119358>.
28. Abd Al-Hameed NM, Al-Ani AW. Assessment of systemic oxidative stress and antioxidants in Iraqi women with newly diagnosed and tamoxifen-treated breast cancer. *Eurasian Chemical Communication*. 2023; 5:204-215. <https://doi.org/10.22034/ecc.2023.365482.1538>.
29. Lu Z, Lightcap IV, Tennyson AG. An organometallic catalase mimic with exceptional activity, H<sub>2</sub>O<sub>2</sub> stability, and catalase/peroxidase selectivity. *Dalton Trans*. 2021; 50:15493-15501. <https://doi.org/10.1039/D1DT02002A>.
30. Hasan HR, Mahmoud HQ. Iron and its related parameters in serum and saliva of Iraqi patients with non-alcoholic and alcoholic liver disease. *MOJ Addiction Medicine & Therapy*. 2018;5(6):249-253. <https://doi.org/10.15406/mojamt.2018.05.00133>.
31. Yakoviichuk A, Krivova Z, Maltseva S, Kochubey A, Kulikovskiy M, Yevhen Maltsev. Antioxidant Status and Biotechnological Potential of New *Vischeria vischeri* (Eustigmatophyceae) Soil Strains in Enrichment Cultures. *Antioxidants*. 2023;12(3):654. <https://doi.org/10.3390/antiox12030654>.
32. Tummalacharla SC, Pavuluri P, Maram SR, Vadakedath S, Kondu D, Karpay S, et al. Serum Activities of Ferritin Among Controlled and Uncontrolled Type 2 Diabetes Mellitus Patients. *Cureus*. 2022;14(5):e25155. <https://doi.org/10.7759/cureus.25155>.
33. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, et al. IDF Diabetes Atlas Committee. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. *Diabetes Research and Clinical Practice*. 2019;157:107843. <https://doi.org/10.1016/j.diabres.2019.107843>.

34. Diwan AG, Vora A, AVaidya N, Padwal MK, Momin AA. Hyperferritinemia: An Early Predictor of Insulin Resistance and Other Complications of Type 2 Diabetes Mellitus. *Bharati Vidyapeeth Medical Journal*. 2022; 2(1):20-28. [https://doi.org/10.56136/BVMJ/2022\\_00031](https://doi.org/10.56136/BVMJ/2022_00031).
35. Al-Musawi HS, Al-Lami MQ, Al-Saadi AH. Assessment of Glycemic Control, Renal Function, and Oxidative Stress Parameters in Type 2 Diabetes Mellitus Patients. *Iraqi Journal of Science*. 2021;62(12):4628-4638. <https://doi.org/10.24996/ij.s.2021.62.12.4>.
36. Hussein MK, Saifalla PH. Estimating insulin resistance and creatine kinase among Iraqi patients with type 2 diabetes mellitus. *Eurasian Chemical Communication*. 2022; 4:1193-1200. <https://doi.org/10.22034/ecc.2022.343053.1470>.
37. Alwan MH, Hussein SZ. Evaluation of Oxidative Stress in Iraqi Male Patients with Myocardial Infarction and Type 2 Diabetes Mellitus. *Iraqi Journal of Science*. 2023;64(10): 4918-4929. <https://doi.org/10.24996/ij.s.2023.64.10.3>.
38. Al-Attaby AKT, Al-Lami MQD. Effects of Duration and Complications of Type 2 Diabetes Mellitus on Diabetic Related Parameters, Adipocytokines and Calcium Regulating Hormones. *Iraqi Journal of Science*. 2019;60(11),2335-2361. <https://doi.org/10.24996/ij.s.2019.60.11.5>.
39. Wang J, Zhang L, Bai Y, Wang X, Wang W, Li J, et al. The influence of shorter red blood cell lifespan on the rate of HbA1c target achieved in type 2 diabetes patients with an HbA1c detection value lower than 7. *Journal of Diabetes*. 2023;15(1):7-14. <https://doi.org/10.1111/1753-0407.13345>.
40. Bharti R. Long duration of diabetes is associated with inadequate glycemic control and lipid profile. *Journal of Research in Applied and Basic Medical Sciences*. 2023;9(4):210-214. <https://doi.org/10.61186/rabms.9.4.210>.
41. Ito H, Omoto T, Abe M, Matsumoto S, Shinozaki M, Nishio S, et al. Relationships between the duration of illness and the current status of diabetes in elderly patients with type 2 diabetes mellitus. *Geriatrics & Gerontology International*. 2017;17(1):24-30. <https://doi.org/10.1111/ggi.12654>.
42. Zhu HT, Yu M, Hu H, He QF, Pan J, Hu RY. Factors associated with glycemic control in community-dwelling elderly individuals with type 2 diabetes mellitus in Zhejiang, China: a cross-sectional study. *BMC Endocr Disord*. 2019; 57:1-11. <https://doi.org/10.1186/s12902-019-0384-1>.
43. Davidson MB. Historical review of prediabetes/intermediate hyperglycemia diagnosis: Case for the international criteria. *Diabetes Research and Clinical Practice*. 2022;185:109219. <https://doi.org/10.1016/j.diabres.2022.109219>.
44. Hizomi AR, Fakhri F, Naeimi TM, Talebi F, Talebi Z, Rashidi N, et al. Prevalence of anaemia and its associated factors among patients with type 2 diabetes mellitus in a referral diabetic clinic in the north of Iran. *BMC Endocrine Disorders*. 2023;23(1):1-8. <https://doi.org/10.1186/s12902-023-01306-5>.
45. Liu J, Li Q, Yang Y, Ma L. Iron metabolism and type 2 diabetes mellitus: A meta-analysis and systematic review. *Journal of Diabetes Investigation*. 2020;11(4):946-955. <https://doi.org/10.1111/jdi.13216>.
46. Ahmed SYA, Albayaty NK. A Study of Hcpidin Levels and Other Biochemical Parameters in Women with Osteoporosis with Type 2 Diabetes Mellitus. *Ibn AL-Haitham Journal for Pure and Applied Sciences*. 2022;35(4):183–193. <https://doi.org/10.30526/35.4.2867>.
47. Hernández C, Lecube A, Carrera A, Simó R. Soluble transferrin receptors and ferritin in Type 2 diabetic patients. *Diabetic Medicine*. 2005;22(1):97-101. <https://doi.org/10.1111/j.1464-5491.2004.01331.x>.
48. Mohammed KJ, Rubat SK, Imran AF. Association of Iron Profile with Type 2 Diabetes Mellitus: Review. *International Journal of Pharmaceutical and Bio-Medical Science*. 2024;4(3):199-205. <https://doi.org/10.47191/ijpbms/v4-i3-13>.
49. Taderegew MM, Gebremariam T, Tareke AA, Woldeamanuel GG. Anemia and Its Associated Factors among Type 2 Diabetes Mellitus Patients Attending Debre Berhan Referral Hospital, North-East Ethiopia: A Cross-Sectional Study. *Journal of Blood Medicine*. 2020;11:47-58. <https://doi.org/10.2147/JBM.S243234>.
50. Shobha MV, Jagadamba A, Prabhakar K, Shashidhar KN. Association of Serum Iron Indices with Insulin Resistance Index in Euglycaemic Offspring's of Diabetic and Non Diabetic Parents: A Case-control Study. *Journal of Clinical and Diagnostic Research*. 2022; 16(7): CC05-CC09. <https://doi.org/10.7860/JCDR/2022/55678.16593>.
51. Zerín N, Sultana S, Afroz F, Rahman M, Chowdhury SA. Iron Status in Type 2 Diabetes Mellitus Patients: A Tertiary Care Hospital Study. *Acta Scientific Medical Sciences*. 2023;7(9):120-129. <https://doi.org/10.31080/ASMS.2023.07.1664>.
52. Saha S, Sarker A, Afrin S, Al-Aharama, Begum IA, Sarker CR, et al. Serum Iron Profile in Type 2 Diabetes Mellitus. *Journal of Rangpur Medical College*. 2023;8(2):40-43. <https://doi.org/10.3329/jrpmc.v8i2.69372>.

53. Valenti L, Corradini E, Adams LA, Aigner E, Alqahtani S, Arrese M, et al. Consensus Statement on the definition and classification of metabolic hyperferritinaemia. *Nature Reviews Endocrinology*. 2023; 19: 299-310. <https://doi.org/10.1038/s41574-023-00807-6>.
54. Chaudhari RK, Niraula A, Gelal B, Baranwal JK, Sarraf DP, Maskey R. Increased Serum Ferritin Levels in Type 2 Diabetes Mellitus Patients: A Hospital Cross-Sectional Study. *Journal of Universal College of Medical Sciences*. 2021;09(02):56-60. <https://doi.org/10.3126/jucms.v9i02.42009>.
55. Moirangthem MM, Rajkumari VD. Study of Serum Ferritin and HbA1c Levels in Type 2 Diabetes Mellitus. *Journal of Evidence-Based Medicine and Healthcare*. 2020; 7(27):1282-1285. <https://doi.org/10.18410/jebmh/2020/272>.
56. Thilipkumar G, Saravanan A, Ramachandran C, Ashok JN. Mean blood glucose level and glycated haemoglobin level in patients of non-insulin dependent diabetes mellitus and its correlation with serum ferritin level. *International Journal of Medical Sciences*. 2011;4(1&2):13-17. [http://researchjournal.co.in/upload/assignments/4\\_1\\_3-17.pdf](http://researchjournal.co.in/upload/assignments/4_1_3-17.pdf).
57. Saha S, Sarker A, Afrin S, Al- Aharama, Begum IA, Sarker CR, et al. Serum Iron Profile in Type 2 Diabetes Mellitus. *Journal of Rangamati Medical College*. 2023;8(2):40-43. <https://doi.org/10.3329/jrpmc.v8i2.69372>.
58. Mamatha BV, Leela P, Samalad VMS, Rakshitha MN. Effects of Iron on blood glucose levels in type 2 diabetes mellitus – A study in tertiary care centre. *Journal of Cardiovascular Disease Research*. 2022;13(04):6-11. <https://jcdronline.org/admin/Uploads/Files/62577fb435b147.72470334.pdf>.
59. AL-Adhami IAA, Al-Shamahy HA, Al-Meeril AM. Plasma Ferritin and Hepcidin Levels in Patients with Type 2 Diabetes Mellitus. *Universal Journal of Pharmaceutical Research*. 2019;4(1):1-6. <https://doi.org/10.22270/ujpr.v4i1.231>.
60. Memon S, Das B, Nisa N, Anjum S, Rafique S, Memon R. Influence of serum ferritin on glycemic control in patients with type 2 diabetes mellitus. *The Professional Medical Journal*. 2024;31(2):183-188. <https://doi.org/10.29309/TPMJ/2024.31.02.7869>.
61. Fillebeen C, Lam NH, Chow S, Botta A, Sweeney G, Pantopoulos K. Regulatory Connections between Iron and Glucose Metabolism. *International Journal of Molecular Sciences*. 2020;21(20):7773. <https://doi.org/10.3390/ijms21207773>.
62. Szklarz M, Gontarz-Nowak K, Matuszewski W, Bandurska-Stankiewicz E. Ferritinology-Iron Is an Important Factor Involved in Gluco- and Lipocrinology. *Nutrients*. 2022;14(21):1-22. <https://doi.org/10.3390/nu14214693>.
63. Zhao L, Zou Y, Zhang J, Zhang R, Ren H, Li L, et al. Serum transferrin predicts end-stage Renal Disease in Type 2 Diabetes Mellitus patients. *International Journal of Medical Sciences*. 2020;17(14):2113-2124. <https://doi.org/10.7150/ijms.46259>.
64. Arija V, Fernández-Cao JC, Basora J, Bulló M, Aranda N, Estruch R, et al. Excess body iron and the risk of type 2 diabetes mellitus: a nested case-control in the PREDIMED (Prevention with Mediterranean Diet) study. *The British Journal of Nutrition*. 2014;112(11):1896-904. <https://doi.org/10.1017/S0007114514002852>.
65. Han LL, Wang YX, Li J, Zhang XL, Bian C, Wang H, et al. Gender differences in associations of serum ferritin and diabetes, metabolic syndrome, and obesity in the China Health and Nutrition Survey. *Molecular Nutrition & Food Research*. 2014;58(11):2189-2195. <https://doi.org/10.1002/mnfr.201400088>.
66. Al Akl NS, Khalifa O, Errafii K, Arredouani A. Association of dyslipidemia, diabetes and metabolic syndrome with serum ferritin levels: a middle eastern population-based cross-sectional study. *Sci Rep*. 2021; 1:1-9. <https://doi.org/10.1038/s41598-021-03534-y>.
67. Góth L, Rass P, Páv A. *Catalase enzyme*

How to Cite this Article:

Oleiwi EH, Hussein SZ. Study the Iron Status and its Correlation with Catalase Activity in Iraqi Patients with Type Two Diabetes Mellitus. *J Fac Med Baghdad [Internet]*.;66(3). Available from: <https://iqjmc.uobaghdad.edu.iq/index.php/19JF/acMedBaghdad36/article/view/2302>

استكشاف العلاقة بين حالة الحديد ونشاط الكاتالاز لدى المرضى المصابين بداء السكري من النوع الثاني

اسراء حسن عليوي  
م.د. صبا زهير حسين  
ماجستير كيمياء حياتية  
كيمياء حياتية

#### الخلاصة:

**خلفية البحث:** الكاتالاز هو إنزيم مضاد للاكسدة موجود في جميع الكائنات الحية، وهو مسؤول عن تحليل بيروكسيد الهيدروجين، وهو نوع من أنواع المركبات الضارة المعروفة باسم أنواع الأوكسجين التفاعلية (ROS)، إلى أوكسجين وماء غير الضارين. يعد هذا الإنزيم ضرورياً لحماية الخلايا من الأضرار التأكسدية التي يسببها ROS لدى الأفراد المصابين بداء السكري من النوع الثاني T2DM. يتزايد انتشار مرض السكري من النوع الثاني (T2DM) بسرعة، ويشكل تحدياً كبيراً للصحة العامة في جميع أنحاء العالم. يمكن أن يعطل T2DM تنظيم الحديد في مجرى الدم، مما يؤدي إلى إنتاج أنواع الأوكسجين التفاعلية والتي يمكن أن تلحق الضرر بالخلايا.

**الهدف:** تبحث الدراسة الحالية في العلاقة بين حالة الحديد ونشاط الكاتالاز لدى المرضى الذين يعانون من T2DM، وإمكانية استخدامه كمتنبئ لبداية وشدة المرض.

**المرضى وطرق العمل:** تضمنت هذه الدراسة 150 مشاركاً، 50 من الأفراد الأصحاء كانوا بمثابة مجموعة ضابطة (C) و100 شخص تم تشخيص إصابتهم بداء السكري من النوع الثاني T2DM وتم تقسيمهم إلى ثلاث مجموعات بناءً على مدة مرضهم: المجموعة A1 (ن = 38؛ أقل من 5 سنوات)، A2 (ن = 37؛ 5-10 سنوات) وA3 (ن = 25؛ أكثر من 10 سنوات). تم أخذ العينات من مستشفى الكندي التعليمي في بغداد خلال الفترة من تشرين الأول 2022 إلى نهاية كانون الثاني 2023. قامت الدراسة بتقييم علامات الدم المختلفة لدى جميع المشاركين، بما في ذلك: نسبة الجلوكوز في الدم الصائم (FBG)، خضاب الدم السكري A1c (HbA1c)، الحديد، إجمالي سعة ربط الحديد (TIBC)، سعة ربط الحديد غير المشبعة (UIBC)، الفيريتين، الترانسفيرين، وتشبع الدم بالترانسفيرين (S.Trans) ونشاط الكاتالاز (CAT).

**النتائج:** بالمقارنة مع المجموعة الضابطة، أظهر المرضى الذين يعانون من T2DM مستويات أعلى بشكل ملحوظ من FBG، وHbA1c، وFeritin، ولا سيما مستويات أقل من الحديد، وTIBC، وUIBC، وS.Trans. ومن المثير للإهتمام أن المجموعة A2 فقط كانت لديها مستويات أقل بكثير من الترانسفيرين مقارنة بالمجموعة الضابطة. لوحظ انخفاض كبير في نشاط الكاتالاز في جميع فئات المرضى. بالإضافة إلى ذلك، لوحظ وجود علاقة إيجابية بين مستويات الحديد ونشاط الكاتالاز في جميع مجموعات المرضى.

**الاستنتاج:** إن ارتفاع مستويات الفيريتين في الدم قد يكون عاملاً خطراً لحدوث النوع الثاني من داء السكري. بالإضافة إلى ذلك، فإن العلاقة الملحوظة بين انخفاض مستويات الحديد وانخفاض نشاط الكاتالاز لدى المرضى الذين يعانون من النوع الثاني من داء السكري أمر مثير للاهتمام. من المحتمل أن يكون هذا الارتباط بمثابة مؤشر مستقبلي لبداية مرض السكري وشدته، مما يستدعي إجراء مزيد من البحث.

**الكلمات المفتاحية:** داء السكري من النوع الثاني، الحديد، الفيريتين، الترانسفيرين، نشاط الكاتالاز