

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

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## 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.

**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, a 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 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.

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

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## 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 public health worldwide (10,11). Chronic hyperglycemia of T2DM is

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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  $Fe^{3+}$  to  $Fe^{2+}$  (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 ( $H_2O_2$ ), and superoxide dismutase (SOD), which breaks down superoxide anion ( $O_2^-$ ) (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  $-20^\circ C$  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.

**Ethical Approval:** The study protocol was approved by the Ethics Committee of the College of Science, University of Baghdad; Ref. CSEC/0123/0012, Date: 27.01.2023.

**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 (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

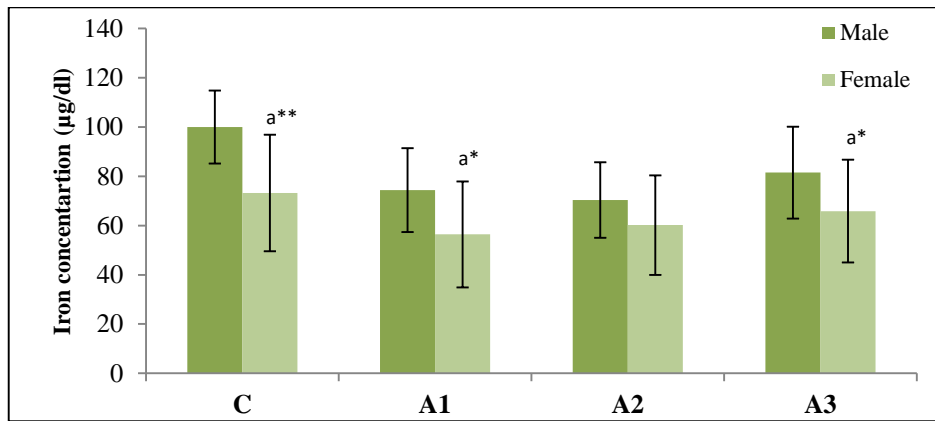
\* $p < 0.05$ , \*\* $p < 0.01$

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

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

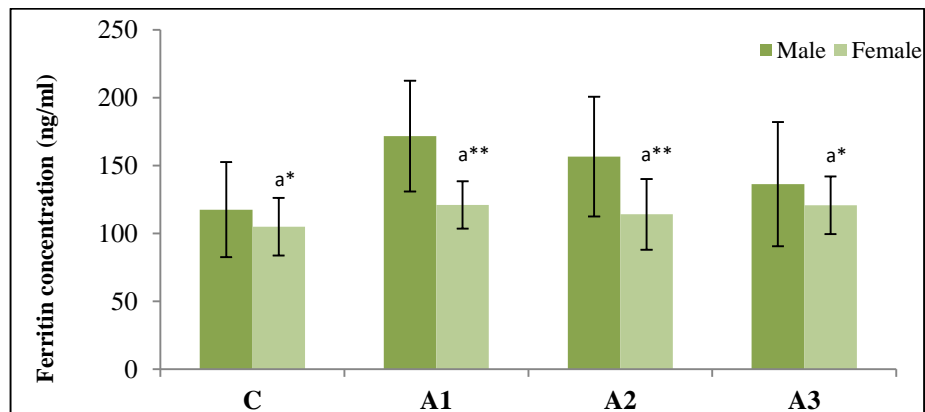
The differences between males and females regarding iron and ferritin levels are presented in figures 1 and 2, respectively. The result indicated a statistically significant difference in iron and ferritin levels

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

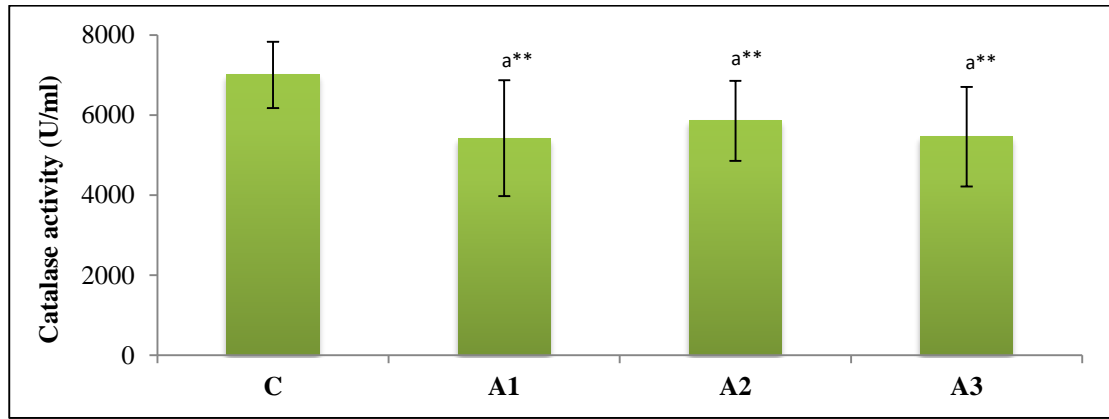
\* $p < 0.05$ , \*\* $p < 0.01$ ; <sup>a</sup> significant difference between males and females in the same group



**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; consistency 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 consistency 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's 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 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

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## Authors' Contribution

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

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### 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 والمنهجية:** 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 بناء على مدة مرضهم: المجموعة A1 (n = 38؛ أقل من 5 سنوات)، A2 (n = 37؛ 5-10 سنوات) وA3 (n = 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 HbA1c، FBG، وFeritin، ولا سيما مستويات أقل من الحديد، وTIBC، وUIBC، وS. Trans. ومن المثير للاهتمام 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 عامل خطر 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.