

ISSN Print: 2664-6781
ISSN Online: 2664-679X
NAAS Rating (2026): 4.77
IJACR 2026; 8(8): 102-106
www.chemistryjournals.net
Received: 13-05-2026
Accepted: 14-06-2026

Bahaulddin Jafar Seger
Pharmacy College, University
of Al-Qadisiyah, Al-
Diwaniyah, Al-Qadisiyah
Governorate, Iraq

Study of serum copper levels and their correlation in obese patients with type 2 diabetes mellitus according to disease duration in the Iraq

Bahaulddin Jafar Seger

DOI: <https://www.doi.org/10.33545/26646781.2026.v8.i8b.736>

Abstract

Purpose: This research examined blood and dietary Copper levels in obese T2DM patients and healthy controls. An analysis also determined which characteristics best predict metabolic regulation and serum Copper levels.

Materials and Methods: Al-Diwaniyah General Hospital examined 60 diabetic patients from February to June 2026. Clinical examination diagnoses. 30 healthy people were controls.

Results: Copper symbols reduced its involvement in type 2 diabetics. Table (2) Fig. (2) displays the mean $\pm$ SD serum Copper levels in mg/dl for normal control and diabetes patients type 2, along with biostatistic calculations and student t-tests. Significantly lower blood Copper levels in diabetes patients type 2 compared to the control group ($p < 0.0001$).

Conclusion: A pioneer study in AL-Diwaniyah tested serum glucose forms in diabetes (type 2) patients, including 60 females and 30 males aged 40-65 and 30 normal healthy controls aged 45-65. Measurements included these parameters:

The glucose and copper (Cu) tests in this investigation revealed.

A considerable serum glucose rise compared to healthy controls. A considerable drop in serum Cu compared to healthy controls, and no difference in copper levels between male and female diabetics.

Keywords: Copper (Cu), diabetes mellitus type 2, copper deficiency, serum Copper

Introduction

Due to its increased frequency, long-term morbidity, and silent long-term effects, diabetes mellitus type 2 (DM2) is a severe public health ^[1]. Over 529 million people had diabetes in 2021, and 1.31 billion by 2050 ^[2]. Type 2 diabetes mellitus (T2DM) is common and characterised by beta-cell dysfunction and insulin resistance. Central nervous system pathways link obesity to type 2 diabetes. These pathways synthesise peripheral organ and environmental data to control food and energy intake ^[3]. Obesity increases insulin resistance, raising blood glucose and HbA1c ^[4]. Micronutrients including selenium (Se), zinc (Zn), copper (Cu), manganese (Mn), iodine (I), and iron (Fe) are essential for enzymatic reactions, signalling pathways, immunological responses, and metabolic activities. Zn transport into β -cells is essential for insulin production, storage, and release. Changes in Zn levels may lead to reduced glucose-induced insulin response ^[5]. Copper (Cu) was demonstrated to play a role in the etiology of diabetes via prooxidant processes ^[6]. Copper is an essential trace element in redox reactions. Cellular defence against superoxide radicals requires enzymes like Cu/Zinc superoxide dismutase. Copper deficiencies may cause cardiac hypertrophy and vascular issues ^[7].

Copper not only directly activates insulin signaling but also inhibits insulin-degrading enzymes (IDE), hence reducing the breakdown of extracellular insulin and boosting insulin levels ^[8]. Copper, a vital redox-active metal, is necessary for mitochondrial electron transport and many oxidase functions; yet, unregulated copper levels can exacerbate oxidative stress and disrupt insulin signaling ^[9]. Copper, a pro-oxidant, generates free radicals that may affect oxidative stress ^[10].

Corresponding Author:
Bahaulddin Jafar Seger
Pharmacy College, University
of Al-Qadisiyah, Al-
Diwaniyah, Al-Qadisiyah
Governorate, Iraq

However, greater whole blood Cu levels raise the risk of obesity-related waist circumference. Catherine *et al.* found a serum Cu-abdominal obesity correlation. Rising Cu tertiles reduced the incidence of increased TGs ^[10].

Studies indicate a correlation between abnormalities of copper regulation and the onset of diabetes-related cardiomyopathy. An excess or lack of copper results in oxidative-reductive imbalances, stimulates inflammatory responses, and worsens diabetes-related cardiomyopathy ^[11]. Investigations on the association between copper and diabetes are few and yield equivocal findings, with existing studies presenting contradictory outcomes. This study seeks to elucidate existing studies on the relationship between copper and diabetes, so offering clinicians insights into the significance of copper supplementation for the improvement and/or prevention of diabetes resulting from copper deficiency.

This study will assist researchers in obtaining proof regarding this association and promote additional inquiry in this domain.

Clinical manifestations of copper

Cellular transport, mitochondrial oxidative metabolism, neurotransmitter synthesis, and neurological and haematological system structure and function need copper. Many foods contain it, which the stomach and proximal duodenum absorb. Copper deficiency, often known as “swayback” or “enzootic ataxia,” has been prevalent in animals, particularly ruminants. Recently discovered, acquired copper insufficiency causes myelopathy and neuropathy in humans. Clinically, copper insufficiency resembles mild combined degeneration from cobalamin deficiency. Balance impairment, numbness, leg paraesthesia, and bilateral lower extremity weakness are common ambulation issues ^[12]. Elevated Cu concentration was detected at the RPE/choroid and RIS/OLM within the retinal layer. Cu was previously shown to participate in neurotransmission inside the plexiform layer. Copper deficiency is documented to induce neuropathy, as well as diminished eyesight and participation of the optic nerve ^[13].

Patients and Methods

This cross-sectional research examined outpatient diabetes patients at AL-Diwaniyah teaching hospital and diabetic center lab from February to June 2026. The research addressed 60 cases (60 patients and 30 controls). Each patient gave a brief history before submitting samples. We divided our patients into two groups for serum blood sugar and Copper measurements based on age, gender, weight,

height, and diabetes mellitus. 60 diabetic cases 30 male, 30 female, and 30 control patients.

Data collection

Fasting for almost 10 hours yielded 8 mL of venous blood under aseptic conditions. Six millilitres of blood went into EDTA tubes and two into gel separator tubes. An automated approach determined (F.B.S) from the EDTA tube sample. Spin the gel separator tubes at 3000 rpm for 5 minutes to obtain the serum. Calculating serum Copper levels using Cobas Pure.

A technician verified the findings. Each patient's profile comprised serum Copper levels below 0.011 to 0.005 mmol/L and infection duration diabetes. Created and saved worksheet with complete data.

Management of Outliers

We utilized the prevalent Three Standard Deviation Rule (mean $\pm$ 3SD) to detect probable outliers in serum copper content, an approach appropriate for our approximately normally distributed data.

Results

Table shows the relevance of glucose indicators in type 2 diabetes. (1) and Fig. (1) show the mean $\pm$ SD blood glucose concentrations in mmol/L for normal controls and type 2 diabetes patients, together with biostatistical analysis and t-test.

There is a notable elevation in serum glucose levels among type 2 diabetic patients relative to the control group ($p < 0.0001$).

Copper symbols were less important in type 2 diabetics. Table (2) and Figure (2) showcase blood copper concentrations ($\mu\text{g/dl}$) for normal controls and type 2 diabetes patients, together with biostatistical analysis and Student's t-test.

A reduction in serum copper concentration is seen in type 2 diabetic patients relative to the control group ($p < 0.0001$).

There is a no significant correlation between serum glucose levels in diabetic patients and the duration of the disease between patients (duration < 10) & (duration > 10) years as shown in Table (3) and, Figure (3) Biostatistical calculation and study t-test for copper level mg/dl in serum of diabetic patient and Normal healthy (controls).

Statistical Analysis

All data is in an Excel sheet. Analysing all data will need statistical analysis.

Table 1: Biostatistical analysis and t-test for serum glucose levels in mmol/l among diabetic patients and healthy controls.

"Glucose mmol/l	Normal healthy controls	Patient
Sample size	30	60
Mean $\pm$ SD	5.2622 $\pm$ 0.654632	11.92671 $\pm$ 5.201118
Range	4.0 – 6.9	4.9 – 22.1
Standard error of mean	0.1035181	0.6713032
T – test	8.068	
probability	<0.0001 Highly significant"	

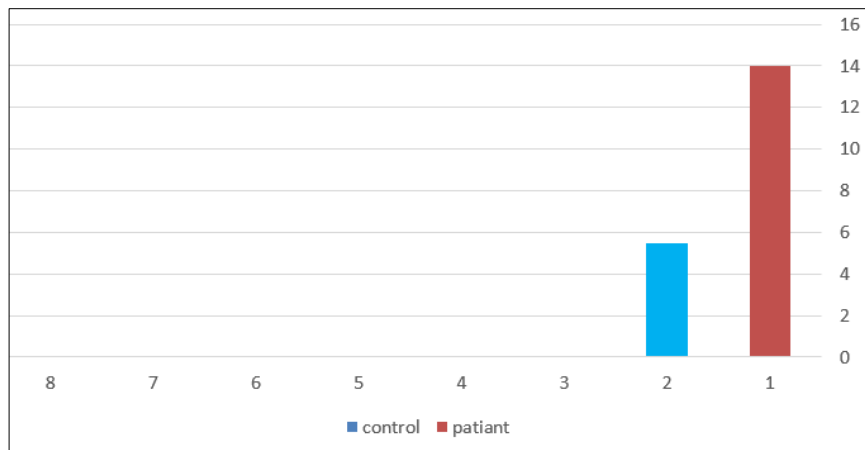


Fig 1: "Glucose test of diabetic patient and control".

Table 2: "Biostatistical analysis and t-test examination for serum copper levels in $\mu\text{g/dl}$ among diabetic patients and healthy controls".

Cu $\mu\text{g/dl}$	Normal healthy controls	Patient
Sample size	30	60
Mean $\pm$ SD	204.8789 $\pm$ 73.60299	142.565 $\pm$ 64.36792
Range	96.5 - 289.8	35.8 - 29692.3
Standard error of mean	11.4801	8.439022
T – test	4.472	
probability	<0.0001 Highly significant	

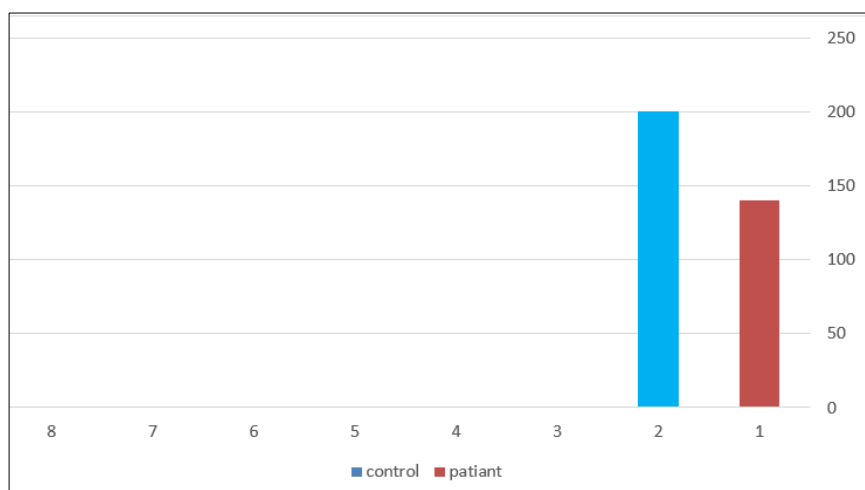


Fig 2: "copper test of diabetic patient and control".

Table 3: Biostatistical calculation and study t-test for copper level mmol/l in serum of diabetic patient and Normal healthy (controls) According to Disease Duration (duration <10) & (duration > 10) years.

Cu ($\mu\text{g/dl}$)	Patient (by duration)	
Group	< 10 years	>10 years
Sample size	30	30
Mean $\pm$ SD	144.4592 $\pm$ 70.05322	138.493 $\pm$ 57.41321
Range	38.3 - 296.5	34.8 - 231.4
Standard error of mean	11.68111	11.76312
T – test	0.290	
probability	0.0776 NS	

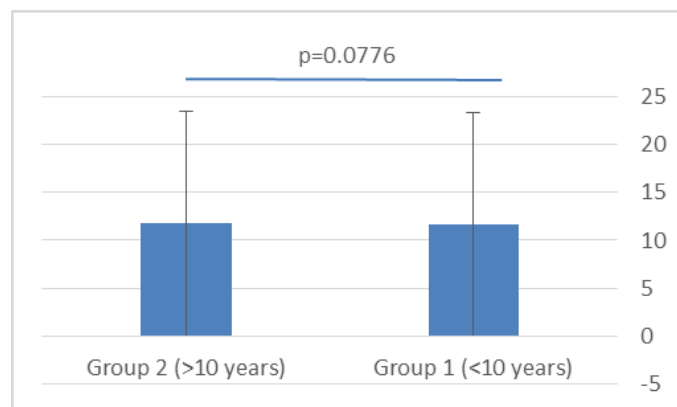


Fig 3: "Biostatistical calculation and study t-test for copper level mg/dl in serum of diabetic patient and Normal healthy (controls)".

Discussion

Type 2 Diabetes Mellitus (T2DM) and obesity are two of the worst public health challenges of the 21st century, affecting 537 million and 890 million people, respectively [14]. Other metabolic processes including insulin secretion depend on trace minerals. Essential trace elements, crucial for life, and non-essential minerals. Trace elements, important micronutrients, are essential to metabolic function, but persistent imbalances either excess or deficiency can cause health problems including type 2 diabetes [15]. Copper is a bimetal in enzymes involved in oxidative phosphorylation, iron transport, and oxidative stress, which may affect insulin metabolism and cause chronic diseases [16]. Metabolic stress may cause concurrent diseases like diabetes. Copper's role in glutamic acid decarboxylase (GAD), a beta cell antioxidant affected by reactive oxygen species (ROS), may also explain the link between copper and hyperglycemia [17].

This research examined the relationship between copper levels and diabetes development and control over time.

The findings of this paper demonstrate a markedly significant elevation in serum glucose levels in diabetic patients compared to normal healthy controls. ($p < 0.0001$) as illustrated in Table (1) Figure (1).

No significant difference in copper levels has been identified in individuals with type 2 diabetes, regardless of gender (male or female), as illustrated in Table (2) and Figure (2).

The findings of this study demonstrate a statistically significant reduction in serum copper levels in patients with type 2 diabetes compared to the normal control group according to Disease Duration (duration < 10) & (duration > 10) years ($p < 0.0001$), as illustrated in Table (3) and the accompanying figure. (3) These findings have been consistent.

Conclusion

The concentration of serum Cu^{2+} is positively related to the development rate of Type 2 diabetes.

Our recent investigations on patients from AL-Diwaniyah City indicate that copper (Cu^{2+}) levels are not correlated with gender, age, weight, or height. The fasting blood sugar test yields precise results and serves as an effective instrument for diagnosing diabetes; copper (Cu^{2+}) deficiency is prevalent among patients with T2DM. Individuals infected for ten years or more exhibit a pronounced insufficiency in copper (Cu^{2+}) levels compared to those infected for less than ten years. Utilizing

supplements as prescribed by physicians to lessen the worsening effects of copper (Cu^{2+}) deficiency in certain patients especially in cases of kidney disease and malnourishment, or in cases of absorption issues or alterations in copper-transporting proteins like ceruloplasmin while considering factors that may raise copper levels in patients with type 2 diabetes in cases of oxidative stress and inflammation, as well as improving insulin sensitivity. Copper deficiency in the blood is linked to poor blood sugar regulation, which results in kidney problems and malabsorption, according to this study of type 2 diabetes patients.

This study indicates a notable prevalence of Hypocupremia among individuals with diabetes, suggesting that the assessment of copper (Cu^{2+}) levels should be incorporated into routine screening, especially for those with a history of Hypocupremia, older age, prolonged diabetes duration, and compromised glycemic control.

To conclude, the physiological efficacy of copper is determined by its concentration. It reveals a dual effect, operating as an antioxidant within optimum quantities and as an oxidizing agent when concentrations are heightened. The study's outcomes related to the link between copper ingestion and the potential for diabetes are perplexing. Yet, in crafting dietary directives for copper, it is important to take into consideration variances in individual consumption patterns. To avoid deficiency or toxicity, individuals need to ensure they consume an adequate amount of dietary copper, meeting the suggested daily intake of 900 $\mu\text{g}/\text{day}$. It is important to stress the peril of copper toxicity, mostly caused by dietary supplements. For patients experiencing a serious copper shortage and unable to secure enough dietary intake, supplementation may be warranted. Supplementary research, especially randomized controlled trials, is urgently needed to enable investigators to clarify this association and establish clear outcomes.

References

1. Abusalah MAH, *et al.* Prevalence of type 2 diabetes mellitus in the general population of Saudi Arabia, 2000-2020: a systematic review and meta-analysis of observational studies. *Saudi J Med Med Sci.* 2023;11(1):1-10.
2. Qiu Y, Yang Y, Liu W, Wu Z. Anisotropic photonic crystal hydrogel array for naked-eye visual pH and glucose sensing. *Microchem J.* 2026;118423.
3. Młynarska E, *et al.* Type 2 diabetes mellitus: new pathogenetic mechanisms, treatment and the most important complications. *Int J Mol Sci.* 2025;26(3):1094.
4. Deng L, Jia L, Wu XL, Cheng M. Association between body mass index and glycemic control in type 2 diabetes mellitus: a cross-sectional study. *Diabetes Metab Syndr Obes.* 2025;555-563.
5. Eroglu B, *et al.* Trace elements and risk of diabetes-related vascular complications: results from the EPIC-Potsdam cohort study. *Cardiovasc Diabetol.* 2025;24(1):302.
6. Samadi A, *et al.* Zinc, copper, and oxysterol levels in patients with type 1 and type 2 diabetes mellitus. *Clin Nutr.* 2020;39(6):1849-1856.
7. Tan PY, Roy MS. Dietary copper and selenium are associated with insulin resistance in overweight and obese Malaysian adults. *Nutr Res.* 2021;93:38-47.
8. Sun Z, *et al.* The link between trace metal elements and glucose metabolism: evidence from zinc, copper, iron,

- and manganese-mediated metabolic regulation. *Metabolites*. 2023;13(10):1048.
9. Sarıkaya U, Selek Ş, Sarıkaya A. Serum zinc, copper, and the Zn/Cu ratio across the glycemic spectrum: evidence from an automated clinical chemistry platform. *Clin Biochem*. 2026;111097.
 10. Escobedo-Monge MF, Barrado E, Parodi-Román J, Escobedo-Monge MA, Torres-Hinojal MC, Marugán-Miguelsanz JM. Copper/zinc ratio in childhood and adolescence: a review. *Metabolites*. 2023;13(1):82.
 11. Jia D, Liu L, Liu W, Li J, Jiang X, Xin Y. Copper metabolism and its role in diabetic complications: a review. *Pharmacol Res*. 2024;206:107264.
 12. Chami HA, Hall MAK, Chami H. Copper deficiency and polyneuropathy: a case report. *Cureus*. 2022;14(8).
 13. Dascalu AM, *et al*. Serum levels of copper and zinc in diabetic retinopathy: potential new therapeutic targets. *Exp Ther Med*. 2022;23(5):324.
 14. Podder D, *et al*. Incretin-based multi-agonist therapies for type 2 diabetes mellitus and obesity: mechanisms, clinical efficacy, and future directions. *Diabetology*. 2026;7(3):46.
 15. Amer HM, Mohamed SA, Elshafeey F, Elhalawany SH. Health implications of prediabetes and the role of trace elements in insulin resistance. *Future J Pharm Sci*. 2025;11(1):59.
 16. Arefhosseini S, Tutunchi H, Arefhosseini SR, Ghavami SZ, Ebrahimi-Mameghani M. Copper status and its relation to abdominal obesity indices and liver function in non-alcoholic fatty liver disease: a case-control study. *BMC Res Notes*. 2024;17(1):370.
 17. Eljazzar S, *et al*. The role of copper intake in the development and management of type 2 diabetes: a systematic review. *Nutrients*. 2023;15(7):1655.