



## **Glycaemic Regulation with Zinc Combination in Type 2 Diabetes Mellitus**

**Gangaram Bhadarge<sup>1</sup>, Pratibha Dawande<sup>2</sup>, Nandkishor Bankar<sup>3\*</sup>  
and Raunak Kotecha<sup>4</sup>**

<sup>1</sup>Department of Biochemistry Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences Sawangi (Meghe) Wardha-442001, India.

<sup>2</sup>Department of Pathology Datta Meghe Medical College, Shalinitai Meghe Hospital and Research Centre (Datta Meghe Institute of Medical Sciences), India.

<sup>3</sup>Department of Microbiology Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi (Meghe), Wardha, India.

<sup>4</sup>Datta Meghe Medical College, Shalinitai Meghe Hospital and Research Centre (Datta Meghe Institute of Medical Sciences), India.

### **Authors' contributions**

*This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.*

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### **ABSTRACT**

**Introduction:** Zn supplementation improved glutathione peroxidase enzyme activity and decreased malondialdehyde and nitric oxide levels in diabetic rats, revealing Zn's defensive effect against oxidative stress in type 2 diabetes. The investigators have discovered that consuming Zn increased liver function and protected pancreatic tissue from damage caused by diabetes. Since Zn also prevents chronic hyperglycemia, it helps to minimize oxidative stress caused by type 2 diabetes. Diabetes mellitus (DM) is a global health problem that affects more than 3 million people worldwide (16% of population). Chronic hyperglycemia causes oxidative stress in diabetic patients by the development of free radicals (oxidants) and lowering the antioxidant protection mechanism.

**Aim:** Glycaemic Regulation with Zinc Combination in Type 2 Diabetes Mellitus.

**Materials and Methods:** Faculty of Medicine and Diabetic Opd, Datta Meghe Medical College and Shalinitai Meghe Hospital and Research Center, Nagpur in collaboration with Datta Meghe Institute of Medical Sciences Deemed to be University, Sawangi, Wardha, Maharashtra.

**Results:** The mean Zn level was  $12.213 \pm 2.342$  in all participants and  $9.121 \pm 1.782$  in the control group, whereas it was significantly low ( $9.121 \pm 1.782$ ) in the diabetic group, and there was statistically significant difference in Zn levels between the controls and the diabetic group ( $P < 0.001$ ). FBS, HbA1C, serum Zinc mean effects between control and patients showed statistically significant differences in type 2 diabetes mellitus ( $P < 0.0001$ ).

**Conclusion:** Our findings show that people with diabetes have lower levels of Zn than healthy people. The cause and effect of the association between very low levels of Zn and the progression of diabetes, or diabetes that causes Zn deficiency, is still unknown. Low levels of Zn are associated with poor glycemic control, and poor glycemic control is a good indication of Zn deficiency, as there was a negative association between serum Zn and FBS and HbA1C. If diabetic patients have low glycemic regulation, a long history of diabetes, obesity, or are over the age of 50, we look to assess their levels in Zn so that Zn alternative treatment can begin to release oxidative stress in this high-risk group.

**Keywords:** Zinc; DM; insulin; hyperglycemia; pancreas.

## 1. INTRODUCTION

Diabetes mellitus (DM) is a severe form of hyperglycaemia caused by insulin resistance or a related insulin deficiency. The International Diabetes Federation estimates that 463 million people worldwide have diabetes by 2019. In addition, the number of diabetic patients is growing worldwide, with 578 million predicted by 2030 [1].

Diabetes mellitus (DM) is a metabolic disease caused by insulin deficiency or resistance. The exocrine half of the pancreas makes up 84 % of its volume, while the endocrine half makes up just 2%. Since the nature and operation of these two areas of the planet are so closely linked, a disruption of one will have an impact on the other [2].

Diabetes mellitus (DM) is a global health problem that affects more than 3 million people worldwide (16 % of population). Chronic hyperglycemia causes oxidative stress in diabetic patients by the development of free radicals (oxidants) and lowering the antioxidant protection mechanism. This causes oxidative cellular damage, which contributes to cellular dysfunction [3].

Diabetes is on the increase, and by 2030, the number of people suffering from depression will have doubled, from 171 million in 2000 to 366 million, with India leading the way. In India, DM is expected to affect up to 79.4 million people by 2030. India is now the world's largest DM outbreak. Type 1 and type 2 diabetes are caused

by reduced or absent insulin secretion and insulin resistance, respectively. Insulin tolerance is described as a reduction in a target organ's response to the effects of chemical insulin. Insulin is formed in pancreatic islets by cells. Exocrinal acinar cells include clusters of endocrine islet cells, which secrete both endocrine and exocrine hormones [4].

Between Zn deficiency and DM, there is no clear cause and effect association. By increasing excretion and decreasing absorption from the intestines or by excretion from the kidneys, DM may cause a decrease in Zn levels in the body. The suggested mechanism of action for Zn is that it acts as a cofactor in the synthesis, transportation, and, most likely, secretion of insulin from the pancreas. Insulin tolerance is a major cause in type 2 diabetes, and can be exacerbated by zinc deficiency. Zn plays an essential part in muscle and fat cell glucose utilization. It serves as a cofactor for enzymes involved in protein, lipid, and glucose metabolism within the cell. In addition to insulin receptor synthesis, zinc can play a role in the insulin receptor-initiated signal transduction pathway. Zn supplementation improved glutathione peroxidase enzyme activity and decreased malondialdehyde and nitric oxide levels in diabetic rats, revealing Zn's defensive effect against oxidative stress in type 2 diabetes. The investigators have discovered that consuming Zn increased liver function and protected pancreatic tissue from damage caused by diabetes. Since Zn also prevents chronic hyperglycemia, it helps to minimize oxidative stress caused by type 2 diabetes. As Zn is a portable cofactor of

carboxypeptidase H, the enzyme that causes the conversion of proinsulin into insulin, plays a role in insulin secretion, secretion and action. By transferring glucose to cells, Zn also aids insulin phosphorylation. Patients with high serum Zn concentrations increased their insulin sensitivity, indicating the importance of Zn in reducing chronic hyperglycemia in type 2 diabetes [5,6].

While there is still some controversy over its applicability for diagnosis, HbA1c is now officially endorsed in many countries as a diagnostic and control tool for (type 2) diabetes. HbA1c is a measure of glycaemia over time rather than a test of glycaemia at a particular point in time. It provides an interconnected index of glycaemia over the red blood cell's entire 120-day lifetime, but during that time frame, recent glycaemia has the greatest impact on the HbA1c rating. 50% of HbA1c was produced in the month prior to sampling, and 25% in the month prior to that. As a result, it seems plausible that a test like this will be useful in diagnosing a disorder marked by chronic hyperglycemia and a slow progression to complications [7].

## 2. AIM

Glycaemic Regulation with Zinc Combination in Type 2 Diabetes Mellitus.

## 3. MATERIALS AND METHODS

The Department of Biochemistry conducted this cross-sectional analysis. The study group consisted of 40 type 2 diabetic patients of both sexes, ranging in age from 35 to 60 years. Faculty of Medicine and Diabetic opd, Datta Meghe Medical College and Shalinitai Meghe Hospital and Research Center, Nagpur in collaboration with ABVRH and JNMC (Datta Meghe Institute of Medical Sciences Deemed to be University), Sawangi, Wardha, Maharashtra.

### 3.1 Sample Collection

7ml of blood samples are collected from each patient and are clearly distributed, sodium

fluoride and EDTA as 3ml, 2ml and 2ml respectively. A serum sample from a plain vial was used to measure Sr. Zinc and Lipid profile while EDTA samples were used to measure HbA1c, sodium fluoride samples were used to measure FBS.

### 3.2 Biochemical Analysis

Serum Zinc , HbA1C, FBS were estimated on AU480 Analyser.

## 4. RESULTS

Table 1 shows the comparison of laboratory data between the control group and the study group. FBS was significantly elevated (215.0±73.8) in type 2 patients compared with healthy subjects (93.2 ±14.1). HbA1C was significantly elevated (7.60±1.42) in type 2 patients compared with healthy subjects (4.7±0.90).

The mean Zn level was 12.213±2.342 in all participants and 9.121±1.782 in the control group, whereas it was significantly low (9.121±1.782) in the diabetic group, and there was statistically significant difference in Zn levels between the controls and the diabetic group (P < 0.001). FBS, HbA1C, serum Zinc mean effects between control and patients showed statistically significant differences in type 2 diabetes mellitus (P < 0.0001).

## 5. DISCUSSION

Zn levels were significantly lower in the group of poor glycemic-controlled diabetic participants compared with those with glycemic-controlled diabetic participants (P < 0.001). This is in line with a study conducted by Bandeira.8 that showed that plasma Zn levels were significantly different from glycosylated hemoglobin in type 2 diabetes patients (r = 0.318, P = 0.004). Similar results are shown in a local study in Saudi Arabia that showed that high HbA1C is associated with lower levels of Zn [8].

**Table 1. comparison healthy individuals with type 2 DM subjects**

Parameters	Controls (n=20)	T2 DM patients(n=20)	P-value
FBS	93.2±14.1	215.0±73.8	P < 0.0001
HbA1c	4.7±0.90	7.60±1.42	P < 0.0001
Zinc	12.213±2.342	9.121±1.782	P = 0.0001

This Zn deficiency can be compensated for with Zn substitution, as evidence has shown that Zn has health advantages for healthier people as well as a beneficial effect for diabetic patients. Zhu et al. [9] found that Zn supplementation improved superoxide dismutase function and decreased malondialdehyde concentrations in both serum and pancreas in diabetic mice.

This is in line with the findings of McNair et al. [10], who found definite hypozincemia in 39.7% of the patients. In that analysis, serum zinc was found to be inversely proportional to the cases' glycemic status, with a P value of less than 0.005.

Hypozincemia was also reported in DM by Garget et al., [11]. Williams et al. [12] found DM to be the most commonly correlated disorder of hypozincemia, with a 17 % drop in controls (P< 0.0001).

In view of the above, it is reasonable to assume that patients with type 2 diabetes have a significant decrease in serum zinc, although it is unclear what the priorities are: The impact of diabetes and hyperglycemia on zinc metabolism, as well as the effects of changes in zinc homeostasis on carbohydrate metabolism. Hyperglycemia appears to interfere with the successful transport of Zn back to renal tubular cells, leading to hyperzincuria. In addition, Zn improves insulin sensitivity by increasing the ability of insulin to bind to its receptors. As a result, a combination of different results, combined [13].

Saharia and Goswami et al. [14]. The serum Zn concentration appears to be decreasing in this sample population of upper Assam, and it would be important to see how successful any intervention is in this population in the country's easternmost region in future studies [15-18].

## 6. CONCLUSION

Our findings show that people with diabetes have lower levels of Zn than healthy people. The cause and effect of the association between very low levels of Zn and the progression of diabetes, or diabetes that causes Zn deficiency, is still unknown. Low levels of Zn are associated with poor glycemic control, and poor glycemic control is a good indication of Zn deficiency, as there was a negative association between serum Zn and FBS and HBA1C. If diabetic patients have low glycemic regulation, a long history of

diabetes, obesity, or are over the age of 50, we look to assess their levels in Zn so that Zn alternative treatment can begin to release oxidative stress in this high-risk group.

## CONSENT AND ETHICAL APPROVAL

As per international standard or university standard guideline patients consent and ethical approval has been collected and preserved by the authors.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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