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Impact of Minerals and Trace Elements on Insulin Resistance and Diabetes

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Abstract

Essential micronutrients needed for the body's normal operation include minerals and trace elements. These components are especially advantageous for physiological processes. Several biochemical processes require the presence of minerals and trace elements, which also serve as cofactors for numerous enzymes and as stabilising elements in proteins and enzymes. Certain trace elements control important biological processes by attaching to the cell membrane's receptor site or by altering the receptor's structure to block the entry of specific molecules. Micronutrients perform a dual function: they keep cellular structures stable at ideal levels, but when levels are low, alternate pathways are opened up and illnesses may result. These crucial micronutrients reveal clear correlations with diabetes and have significant physiological effects.

Keywords: Micronutrients • Physiological • Biochemical

Introduction

The estimation of important micronutrient deficiency overload comes from reputable sources like scientific findings and clinical data from diabetes research. However, it is challenging for doctors to give nutritional advice for diabetics because of the numerous conflicting research [1]. The life expectancy of diabetes people has increased due to advancements in interventions and research, which has coincided with a growth in the elderly population as a whole. Diabetes affects the antioxidant enzymes that are related with trace elements. Numerous cohort studies have demonstrated that diabetes mellitus can change the homeostasis of trace elements. The disruption of insulin metabolism may be significantly influenced by early abnormalities in particular components. The bulk of cohort studies either concentrate on a single element or a small number of element combinations.

Description

Micronutrients are recognised as essential nutrients that are needed in very small amounts for the maintenance of homeostasis and the proper functioning of enzymes. The four main categories of micronutrients are macro elements, vitamins, trace elements, and organic acids. Chloride, calcium, phosphorous, magnesium, sodium, potassium, and iron are the major macroelements. On the other hand, certain trace elements, including as cobalt, boron, chromium, copper, sulphur, iodine, zinc, and molybdenum, improve the action of insulin by activating insulin receptor sites. The mode of action of certain macro and trace elements is altered in type 2 diabetes mellitus, and these trace elements play distinct roles in the development and progression various randomised controlled trials, cohort and case-controlled studies, observational studies [2].

An illustration of how changing trace element and mineral levels contribute to oxidative stress's amplified effects on the development of insulin resistance and diabetes. Boron, a vital but frequently underutilised trace micronutrient present in several meals, has a variety of vital functions in metabolism. Borax's effects on human health are especially significant for bone growth and regeneration, wound healing, sex hormone production, vitamin D metabolism, calcium absorption, and usage [3]. magnesium is a mineral. Dietary boron influences plasma insulin concentrations, according to studies. According to research by Bakken et al., boron-deficient rats had considerably higher plasma insulin concentrations than boron-supplemented rats. Boron deficiency has not been linked to changes in plasma glucose levels and is not reliant on magnesium intake or vitamin D status from food. By binding to NAD+ and/or cyclic ADP ribose and blocking the release of Ca2+ in response to ryanodine receptor agonist, boric acid also influences the release of insulin and brain function. According to studies conducted on animals, boron has an impact on triglyceride levels and may function as an enzyme system metabolic regulator. The maternal status of boron in both healthy and diabetic pregnancies, according to a study, is not the same [4].

linked with levels of lipids and boron. There was no discernible difference in the amounts of boron in the serum lipids between the 15 non-gestational diabetic and the 19 gestational diabetic women. In a cell model, adipogenesis was shown to be inhibited by boric acid and sodium pentaborate pentahydrate, according to a different study [5]. By controlling important growth factors, catenin, AKT, and extracellular signal-regulated kinase signalling pathways, the treatment with boric acid suppressed the expression of genes and proteins associated with adipogenesis. In diabetic rats, boron therapy also showed a reduction in oxidative stress, demonstrating an antioxidant impact and pancreatic beta-cell preservation. Insulin resistance and secretion are significantly influenced by calcium homeostasis. Diabetes affects calcium homeostasis, which results in poor cell control in erythrocytes, cardiac muscles, and other tissues [6].

skeletal muscles and platelets. The compromised homeostasis is alarming since it may play a substantial role in regulating normal insulin secretion and action, as well as influencing different vascular problems on its own.Changes in calcium and vitamin D levels were demonstrated to play a role in the emergence of T2DM. The study found a weakly consistent correlation between the prevalence of T2DM or metabolic syndrome and low vitamin D status and calcium or dairy intake. Analysis of serum 25-OHD levels and the prevalence of metabolic syndrome and T2DM revealed inverse relationships between incidence of T2DM or metabolic syndrome for those with higher combined vitamin D and calcium intakes compared to those with lower intakes [7]. Having high blood sugar had a negative impact on vitamin D and while supplementing with these two nutrients demonstrated positive effects on glucose metabolism, calcium deprivation did not.

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There was a difference in the amounts of serum calcium reported in two small group trials. A research in Baghdad with participants aged to years found that serum calcium levels increased while parathyroid levels significantly decreased. Another study conducted in India found that diabetic individuals had considerably lower serum calcium levels than non-diabetic controls. Serum calcium levels and elevated plasma blood glucose levels were inversely associated. The outcomes revealed revealed as compared to the control group with normal levels of HbA1c, the diabetic group with higher Hb1Ac saw a significant drop in serum calcium levels. When compared to control individuals, diabetic patients with uncontrolled hyperglycemia are at greater risk of hypocalcemia, according to this negative link between serum calcium levels and HbA1c in these patients [8].

There aren't any cohort studies looking at the role of increased serum calcium levels as indicators of faulty glucose metabolism. One of these studies showed a higher risk of diabetes in people with higher serum calcium values. According to the study's findings, serum calcium levels increased generally in 77 T2DM cases over the course of follow-up. These outcomes are consistent with earlier cross-sectional investigations where patients with diabetes displayed elevated serum calcium levels than non-diabetic people, which remained significant after calcium supplement users and people with calcium levels outside of the normal range were eliminated. This finding showed an increased risk of T2DM with higher serum calcium levels. Another study found that 1329 middle-aged and elderly Korean adults had higher serum calcium levels and were more likely to have metabolic syndrome and diabetes. Age, sex, body mass, serum creatinine, phosphorus, parathyroid hormone, levels, smoking, alcohol consumption, exercise, total energy intake, and calcium and sodium intake were not associated with this connection [9]. There is a complicated relationship oxidative stress, as well. High cytosolic calcium levels may be related to insulin resistance, according to research conducted in cell culture. Previous dose dependent meta-analyses of cohort studies have demonstrated that calcium intake in the diet delays the onset of T2D.

According to a number of studies, the average serum cobalt level is less than through its glucose-lowering cobalt chloride was shown by to reduce gluconeogenesis in diabetic rats. In the visceral organs of diabetic rats, cobalt alone or in combination with ascorbate inhibits lipid peroxidation [10]. In a rat model of type 2 diabetes, cobalt therapy improved nephropathy and heart function although serum levels of cobalt decreased in compared to non-diabetic counterparts. Between calcium levels and the development of diabetes, according to studies.

Conclusion

Reduced-cell activity was linked to aberrant calcium regulation, which may also be related to a change in glucose homeostasis. In contrast to other research done on streptozotocin treated diabetic rats, they reported a greater mean concentration of cobalt in diabetic patients on multi-element serum analysis. In comparison to healthy people, Flores et al. found that diabetic patients had significantly greater serum concentrations of Al, Cd, Cu, Mn, Hg, and Ni, and significantly lower serum concentrations. For, the amounts of trace elements in the urine and serum of diabetic patients and healthy individuals were examined in the range. According to the study, diabetes patients had greater urine levels of Cr, As, Cu, and Zn and lower levels of Cd, Co, Pb, Mn, Mo, Ni, and Se than healthy people. Only the variations in Cd and Zn, though, were statistically significant.

Acknowledgement

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Conflict of Interest

None.

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