Role of selenium in type-2 diabetes and insulin resistance

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Description

Selenium is an essential trace element that acts physiologically through selenium proteins. Among other effects, the endogenous antioxidant selenoprotein glutathione peroxidase and the circulating selenium transporter selenoprotein P play important roles in type-2 diabetes and insulin resistance by attenuating the insulin signaling cascade through various mechanisms.

Adequate bioavailability of selenium is important for many aspects of human biology, including the endocrine system, muscle function, and the cardiovascular system. In nature, inorganic Se occurs in four different oxidation states selenite, selenite, elemental Se, and selenide. Biological systems can convert this inorganic selenium into more bioavailable organic forms, such as selenocysteine and selenomethionine, selenium-containing amino acids that become part of proteins called selenoproteins. In the human proteome, selenoproteins consist of a total of 25 proteins with diverse functions, including protection against oxidative stress, Se storage and transport, and redox signaling. Their important role in the oxidative stress system derives from their ability to neutralize reactive oxygen and nitrogen species. These selenoproteins can be distinguished by enzymatic activity or lack of such activity like the selenoprotein K family.

Unlike other micronutrients, selenium intake varies widely around the world, ranging from deficiency to toxic levels that can lead to nail loss, hair loss, poor dental health, and even nervous system and skin disorders be connected. Numerous studies have shown beneficial effects of Se in various medical conditions such as cancer, the immune system, and hyperlipidemia. Selenium supplementation in patients with hashimoto's thyroiditis is associated with decreased thyroid autoantibody and thyroid-stimulating hormone levels due to its antioxidant capacity and hyperregulation of regulatory T cells.

A study of leukocyte DNA integrity found that selenium supplementation improved its interaction with dietary micronutrients. Selenium supplementation proved beneficial, especially when folic acid was low and methionine intake was high, due to increased homeostatic apoptosis. Among all the beneficial and detrimental properties of Se, the association between high Se levels and the risk of developing type-2 diabetes is relatively new. An analysis of non-experimental studies based on dietary and blood selenium levels revealed a non-linear dose-response relationship with type-2 diabetes risk. It has also been suggested that the association with Se, obesity and T2DM may be due to abnormal adipocyte metabolism due to excessive release of fatty acids and/or hormones. Additionally, high selenium intake in people without a history of diabetes is associated with a higher risk of hospitalization for type-2 diabetes. Finally, in a randomized study of patients with type-2 diabetes, Se supplementation adversely affected blood glucose homeostasis in Se-deficient patients, even when Se concentrations reached optimal levels of antioxidant activity. The pancreas is essential for controlling metabolism and energy expenditure. It consists of exocrine pancreas and endocrine pancreas, which are morphologically and functionally distinct.

The exocrine pancreas contains ductal and acinar cells that are involved in the production and release of digestive enzymes into the small intestine for the digestion of fats, carbohydrates, and proteins for absorption. At high concentrations, Se also acts as insulin mimetic, as it promotes glucose uptake in adipocytes by facilitating translocation of glucose transporters to the cell membrane and activating serine/threonine kinases.

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Conflict of Interest

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