Zinc, Insulin and Diabetes

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The relationship between diabetes, insulin and zinc (Zn) is complex with no clear cause and effect relationships. In Type 1 diabetes there is a lack of insulin production, in Type 2 diabetes resistance to the effects of insulin are predominant. Both Type 1 and Type 2 have the same long-term complications. Diabetes effects zinc homeostasis in many ways, although it is most probably the hyperglycemia, rather than any primary lesion related to diabetes, which is responsible for the increased urinary loss and decreases in total body zinc. The role of Zn deficiency, which could, at least potentially, exacerbate the cytokine-induced damage in the autoimmune attack which destroys the islet cell in Type 1 diabetes, is unclear. Since Zn plays a clear role in the synthesis, storage and secretion of insulin as well as conformational integrity of insulin in the hexameric form, the decreased Zn, which affects the ability of the islet cell to produce and secrete insulin, might then compound the problem, particularly in Type 2 diabetes. Several of the complications of diabetes may be related to increased intracellular oxidants and free radicals associated with decreases in intracellular Zn and in Zn dependent antioxidant enzymes. There appears to be a complex interrelationship between Zn and both Type 1 and Type 2 diabetes. The role of Zn in the clinical management of diabetes, its complications, or in its prevention is, at best, unclear.

Key teaching points:

- Zinc plays a key role in the synthesis and action of insulin, both physiologically and in the pathologic state of diabetes.
- Hyperglycemia from either Type 1 or Type 2 diabetes causes physiologically important losses of Zn from the body. These losses may worsen the underlying diabetes but are probably not responsible as the causal agent.
- The complications of diabetes may be mediated, at least in part, through oxidative stress and Zn plays a key role in the cellular antioxidative defense. If there is insufficient Zn, oxidative stress may damage the cell irreversibly, producing or exacerbating some of the classic complications of diabetes.