

Abstract

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Zinc and diabetes--clinical links and molecular mechanisms.

Jansen J, Karges W, Rink L.

Institute of Immunology, Medical Faculty, RWTH Aachen University, 52074 Aachen, Germany.

BACKGROUND: Zinc is an essential trace element crucial for the function of more than 300 enzymes and it is important for cellular processes like cell division and apoptosis. Hence, the concentration of zinc in the human body is tightly regulated and disturbances of zinc homeostasis have been associated with several diseases including diabetes mellitus, a disease characterized by high blood glucose concentrations as a consequence of decreased secretion or action of insulin.

DISCUSSION: Zinc supplementation of animals and humans has been shown to ameliorate glycemic control in type 1 and 2 diabetes, the two major forms of diabetes mellitus, but the underlying molecular mechanisms have only slowly been elucidated. Zinc seems to exert insulin-like effects by supporting the signal transduction of insulin and by reducing the production of cytokines, which lead to beta-cell death during the inflammatory process in the pancreas in the course of the disease. Furthermore, zinc might play a role in the development of diabetes, since genetic polymorphisms in the gene of zinc transporter 8 and in metallothionein (MT)-encoding genes could be demonstrated to be associated with type 2 diabetes mellitus. The fact that antibodies against this zinc transporter have been detected in type 1 diabetic patients offers new diagnostic possibilities.

SUMMARY: This article reviews the influence of zinc on the diabetic state including the molecular mechanisms, the role of the zinc transporter 8 and MT for diabetes development and the resulting diagnostic and therapeutic options.

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