

Abstract

J Nutr. 2008 Sep;138(9):1664-70.

Zinc deficiency induces membrane barrier damage and increases neutrophil transmigration in caco-2 cells.

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OBJECTIVE: Zinc may contribute to the host defense by maintaining the membrane barrier. In this study, we questioned whether zinc deficiency affects the membrane function and junctional structure of intestinal epithelial cells, causing increased neutrophil migration.

METHODS: We used the Caco-2 cell line grown in control (C), zinc-deficient, or zinc-replete medium until differentiation.

RESULTS: Zinc deprivation induced a decrease of transepithelial electrical resistance and alterations to tight and adherens junctions, with delocalization of zonula occludens (ZO-1), occludin, beta-catenin, and E-cadherin. Disorganization of F-actin and beta-tubulin was also found in zinc deficiency. These changes were associated with a loss of the amounts of ZO-1, occluding, and beta-tubulin. In addition, zinc deficiency caused a dephosphorylation of occludin and hyperphosphorylation of beta-catenin and ZO-1. Disruption of membrane barrier integrity led to increased migration of neutrophils. In addition, zinc deficiency induced an increase in the secretion of interleukin-8, epithelial neutrophil activating peptide-78, and growth-regulated oncogene-alpha, alterations that were not found when culture medium was replete with zinc.

CONCLUSIONS: These results provide new information on the critical role played by dietary zinc in the maintenance of membrane barrier integrity and in controlling inflammatory cell infiltration.

PMID: 18716167

