

Abstract

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Selenium deficiency in celiac disease: risk of autoimmune thyroid diseases

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BACKGROUND: The essential trace mineral selenium is of fundamental importance to human health. It is incorporated in the proteome in the forms of the genetically encoded amino acids selenocysteine and selenomethionine, which are the characteristic components of selenoproteins (SeP) such as glutathione peroxidases (GPx), thioredoxin reductases and iodothyronine deiodinase families.

DISCUSSION AND FINDINGS: Thyroid is especially sensitive to selenium deficiency, because SeP can modify thyrocytes function by acting as antioxidants and modifying redox status and thyroid hormone metabolism. SeP are also involved in apoptosis, cell growth and modification of the action of cell signalling systems and transcription factors. Some intestinal GPx modulate apoptosis by removing the cells affected by oxidative damage preserving tissue integrity. The malfunctioning of the GPx antioxidant system in intestinal mucosa can trigger a continuous cycle of reactive oxygen species and inflammation. Selenium deficiency is a risk factor, due to the malabsorption, in celiac disease (CD) because the inflammatory damage affects the small intestine; this deficiency can modulate SeP genes expression, with consequent reiteration of inflammation and increase of mucosal damage. In active CD, overexpression of interleukin-15 (IL-15) may increase activation of effector mechanisms of epithelial damage by stimulating T helper 1 cytokine proliferation and production and intraepithelial lymphocytes cytotoxicity by protecting these lymphocytes from apoptosis. Blocking IL-15 has the potential to provide new therapeutic tools to prevent both tissue damage and complication of CD such as autoimmune thyroid diseases (AITD) where IL-15 expression is also increases.

CONCLUSIONS: In view of the role played by SeP in apoptosis inhibition, the presence of environmental factors such as selenium deficiency can be considered an important direct factor of thyroidal damage in development of AITD.

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