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Myo-inositol in autoimmune thyroiditis, and hypothyroidism

Poupak Fallahi [1], Silvia Martina Ferrari [2], Giusy Elia [2], Francesca Ragusa [2], Sabrina Rosaria Paparo [2], Claudia Caruso [2], Giovanni Guglielmi [3], Alessandro Antonelli [4]

Affiliations expand

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Abstract

Myo-inositol (Myo-Ins) plays an important role in thyroid function and autoimmunity. Myo-Ins is the precursor for the synthesis of phosphoinositides, which takes part in the phosphatidylinositol (PtdIns) signal transduction pathway, and plays a decisive role in several cellular processes. In the thyroid cells, PtdIns is involved in the intracellular thyroid-stimulating hormone (TSH) signaling, via Phosphatidylinositol (3,4,5)-trisphosphate (PtdIns(3,4,5)P3) (PIP-3). Moreover, the phosphatidyl inositol 3 kinases (PI3K) family of lipid kinases regulates diverse aspects of T, B, and Tregs lymphocyte behaviour. Different mouse models deficient for the molecules involved in the PIP3 pathway suggest that impairment of PIP3 signaling leads to dysregulation of immune responses and, sometimes, autoimmunity. Studies have shown that cytokines modulate Myo-Ins in thyroid cells. Moreover, clinical studies have shown that after treatment with Myo-inositol plus seleniomethionine (Myo-Ins + Se), TSH levels significantly declined in patients with subclinical hypothyroidism due to autoimmune thyroiditis. The treatment was accompanied by a decline of antithyroid autoantibodies. After treatment serum CXCL10 levels declined, confirming the immune-modulatory effect of Myo-Ins. Additional research is necessary in larger population to evaluate the effect on the quality of life, and to study the mechanism of the effect on chemokines.

Keywords: Autoimmune thyroid diseases; Autoimmune thyroiditis; CXCL10; Hypothyroidism; Myo-inositol; Seleniomethionine.

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