Exp Clin Endocrinol Diabetes. 2003 Sep;111(6):325-9. doi: 10.1055/s-2003-42721.

Influence of iodide and iodolactones on thyroid apoptosis. Evidence that apoptosis induced by iodide is mediated by iodolactones in intact porcine thyroid follicles

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PMID: 14520597 DOI: 10.1055/s-2003-42721

Abstract

Iodine induced thyroid involution is caused by apoptosis rather than necrosis. This effect of iodide on apoptosis of thyroid epithelial cells may be not a direct one but mediated by iodinated derivatives i.e. of polyunsaturated fatty acids, especially of iodolactones, which have previously shown to inhibit thyroid cell proliferation. We studied the influence on apoptosis of iodide (2 microM and 20 microM) and iodolactone (0.05 microM and 0.5 microM), with and without TSH (1 mU/ml), using a well characterized ex vivo- culture system of intact porcine thyroid follicles in three-dimensional culture. Apoptosis and necrosis was evaluated by electron-microscopy. Stimulation with 2 and 20 microM iodide rapidly induced a rate of apoptosis (4 - 6 %) comparable to about 40-fold lower doses of delta-iodolactone (0.05 microM and 0.5 microM). Addition of TSH (1 mU/ml) caused a slight but not significant further increase of the incidence of apoptotic cells. The rate of necrotic thyroid epithelial cells (1 - 2 %) was similar in all experiments. As delta-iodolactone in very low concentrations--comparable to iodide in higher concentrations--not only inhibits growth but also induces apoptosis, it has to be supposed that the effect of iodide is mediated by this iodinated compound. However, further experiments are necessary to confirm this hypothesis. In addition it could be demonstrated, that apoptosis is a very rapid and limited process in intact follicles. This also may explain, why iodine supplementation even in high doses does not lead to thyroid atrophy but only normalisation of thyroid size. These results confirm that apoptosis is an important regulated and limited mechanism in goiter involution.

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