Câncer de tireóide. O iodo inorgânico induz apoptose no câncer de tireóide.

Iodine induces apoptosis via regulating MAPKs-related p53, p21, and Bcl-xL in thyroid cancer cells.

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Abstract

Thyroid cancer is the most common endocrine malignancy and exhibits the full range of malignant behaviors from the relatively indolent occult differentiated thyroid cancer to uniformly aggressive and lethal anaplastic thyroid cancer. Iodine is a well known key element in thyroid normal function maintenance and thyroid cancer development. However, the effects induced by iodine and the molecular mechanisms involved remain poorly understood in thyroid cancer. We investigated the apoptotic effect of iodine on three different subtypes of thyroid cancer cells. We observed that apoptosis induced by iodine was mitochondrial-mediated. Iodine treatment decreased the level of mutant p53 including the R273H mutant that possesses anti-apoptotic features, but increased the p21 level. Surprisingly, high doses of iodine promoted instead of suppressed the expression of anti-apoptotic protein Bcl-xL expression. Moreover, iodine transiently activated the subfamily members of mitogen activated protein kinases (MAPKs) (ERK1/2, p38 and JNK1/2) which contribute to modulate p53, p21 and Bcl-xL expression. The further results showed the three subfamily members of MAPKs all worked as anti-apoptotic factors. Collectively, iodine-induced apoptotic pathway is involved in the activation of MAPKs-related p21, Bcl-xL and mutant p53 regulation. The findings provide solid molecular evidence to explain the potential pathway for iodine to influence thyroid cancer development. It may also reveal some novel molecular targets for the treatment of thyroid cancer.

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