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Induction of expression of pd-l1 by thyroid hormone interferes with resveratrol-induced anti-proliferation in cancer cells

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The checkpoint programmed cell death protein 1 (PD-1)/programmed death-ligand 1 (PD-L1) plays an important role in cancer proliferation. Thyroid hormone, L-thyroxine (T₄), induces the expression of PD-L1 and promotes cell proliferation in cancer cells. Resveratrol induces anti-proliferation in various types

of cancer cells via binding to integrin $\alpha\beta 3$ to increase nuclear inducible cyclooxygenase (COX)-2 accumulation, complex with p53, and induce p53-dependent anti-proliferation. We investigated the mechanism by which L-thyroxine impairs resveratrol-induced anti-proliferation in human cancer cells. L-thyroxine increased expression and cytoplasmic accumulation of PD-L1. The increased PD-L1 retained resveratrol-induced COX-2 in cytoplasm and prevented COX-2 nuclear accumulation. By inhibiting activated PI-3K-STAT3 signal transduction axis by thyroid hormone, nanodiamino-tetrac (NDAT) was able to inhibit thyroid hormone-induced PD-L1 and relieved the inhibitory effect of L-thyroxine on resveratrol-

induced nuclear accumulation of COX-2. NDAT is a thyroid hormone derivative. The NDAT-inhibited consequences were reestablishing COX-2/p53-dependent gene expression and anti-proliferation. These findings provide new insights into the antagonizing effect of NDAT on L-thyroxine-induced interference with resveratrol-induced anticancer properties.

Biography

Lin received his PhD degree at SUNY Buffalo in 1991. Dr. Lin has served at the VA Hospital and Ordway Research Institute in Albany, NY as a senior scientist. Later, he worked at the Pharmaceutical research Institute (PRI) at the Albany College of Pharmacy and Health Sciences, Albany, NY. Dr. Lin joined Taipei Medical University as full professor in 2012. For the past three years, his research has focused on mechanisms involved in thyroid hormone-induced pathogenesis in colorectal cancer and other types of cancers.

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