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3,5,3′-triiodothyronine (T3) is a survival factor for pancreatic β-cells undergoing apoptosis

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Abstract

3,5,3′-triiodothyronine (T3) is essential for the growth and the regulation of metabolic functions, moreover, the growth-stimulatory effect of T3 has largely been demonstrated and the pathways via which T3 promotes cell growth have been recently investigated. Type 1 diabetes (T1D) is due to the destruction of β-cells, which occurs even through apoptosis. Aim of our study was to analyze whether T3 could have an antiapoptotic effect on cultured β-cells undergoing apoptosis. We have demonstrated that T3 promotes cell proliferation in islet β-cell lines (rRINm5F and hCM) provoking an increment in cell number (up to 55%: rRINm5F and 45%: hCM), cell viability, and BrdU incorporation, and regulating the cell cycle-related molecules (cyc A, D1, E, and p27kip1). T3 inhibited the apoptotic process induced by streptozocin, S-Nitroso-N-Acetylpenicylamine (SNAP), and H2O2 via regulation of the pro- and anti-apoptotic factors Bcl-2, Bcl-XL, Bad, Bax, and Caspase 3. The T3 protective effect was PI-3 K-, but not MAPK- or PKA-mediated, involving pAktThr308. Thus, T3 could be considered a survival factor protecting islet β-cells from apoptosis. J. Cell. Physiol. 206: 309–321, 2006. © 2005 Wiley-Liss, Inc.

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Apoptosis is the process of programmed cell death (PCD) that may occur in multicellular organisms. Biochemical events lead to characteristic cell changes (morphology) and death. These changes include blebbing, cell shrinkage, nuclear fragmentation, chromatin condensation, and chromosomal DNA fragmentation.