

Białka z rodziny Bcl-2 a przekaznictwo sygnału wapniowego w zarodkach ssaków uzyskiwanych techniką klonowania somatycznego

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The Bcl-2 family proteins and calcium signaling in mammalian embryos generated by somatic cell cloning

Summary

The efficiency of somatic cell nuclear transfer (SCNT) technology in mammalian species remains unsatisfactory. One of the main causes of low developmental capability of pre- and peri-implanted somatic cell cloned embryos is the high occurrence of apoptotic cell death, which is prompted by incorrect calcium signaling. The latter is accompanied by upregulation of the members from the Bcl-2 protein family in the blastomeres of SCNT embryos derived from the reconstructed oocytes exposed to artificial activating factors that induce the phenomenon of Ca^{2+} ion excitotoxicity. Overexpression of antiapoptotic proteins from the Bcl-2 family plays a fundamental role in suppression of different pathways involving intracellular transduction of programmed cell death signal in the somatic cell cloned embryos. Enhancement of Bcl-2 synthesis in the cytoplasm as well as on the outer/cytoplasmic surface of cisterns and tubules of granular endoplasmic reticulum (ER_g) and thereby increase in its concentration and activity in the membranes of ER and mitochondria prevents the redistribution of free calcium cations from ER to mitochondria. The purpose of this article is to provide an overview of the current knowledge on molecular aspects of controlling calcium intracellular homeostasis in mammalian SCNT embryos, in which apoptotic cell death was stimulated by an improper activation of reconstituted oocytes.

Key words:

SCNT embryo, artificial activation, programmed cell death, Bcl-2, endoplasmic reticulum, Ca^{2+} ion, calcium-dependent ATPase, phospholamban, ion channel, mitochondrial Ca^{2+} uniporter.

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