Role of caspase-3 in regulating neurotrophic and NMDA-dependent PCD in the mammalian CNS in vivo

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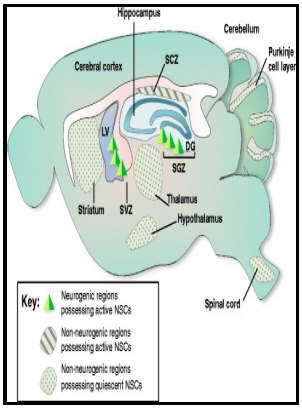

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Notes: Thesis (M.Sc.) -- University of Toronto, 2001.
This edition was published in 2001

Filesize: 68.28 MB

Tags: #Regulation #of #caspase

CELL DEATH

Unraveling the interplay between these pathways will be important for understanding the control of motoneuron survival.

Programmed Cell Death of Embryonic Motoneurons Triggered through the FAS Death Receptor

However, shortened telomeres impair immune function that might also increase cancer susceptibility.

Pathological apoptosis in the developing brain

As to TCD, we reasoned that successful Casp3 RNAi should match to improved cell survival, the constitutive activity of the protease being nullified by post-transcriptional gene silencing. These findings recapitulate that of glucocorticoids on synaptic plasticity, indicating that common or converging pathways control synaptic and mitochondrial functions.

CELL DEATH

Vesicles may be specialized for various purposes.

Regulation of caspase

Efferocytosis also induces the secretion of VEGF by both epithelial cells and macrophages, which enhances the proliferation of pulmonary microvascular endothelial cells, and protects both endothelial and epithelial cells against ultraviolet-induced apoptosis. The steps involved in apoptosis have been immensely studied in recent years and seem very regular. Astrocyte-neuron lactate transport is required for long-term memory formation Cell 2011 144 5 810 823 10.

Elevated expression levels of proBDNF and p75 NTR in CR-proBDNF cortical neurons. Thus, besides to RNAi and Ac-DEVD-CMK experiments, a further proof for the pSCAT3-DEVD specific recognition of Casp3 came from subcellular distribution imaging, and led to safely conclude that pSCAT3-DEVD was specifically detecting Casp3 and not Casp7 activity in organotypically cultured CGCs.

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