Anatoly B Uzdensky et al., Brain Disord Ther 2017, 6:2(Suppl)
http://dx.doi.org/10.4172/2168-975X-C1-014

Involvement of calcium-dependent signaling in the axotomy-induced death of satellite glial cells

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Neurons and satellite glia mutually support survival of each other, but signaling processes that control their life and death after axotomy (AT) are insufficiently explored. We used a simple model object- axotomized crayfish mechanoreceptor consisting of single sensory neuron surrounded by glial envelope. Control: Undamaged mechanoreceptor that saved connection with abdominal ganglion. Necrosis and apoptosis of glial cells increased at 8 hours after AT when death of neurons was not detected yet. 3-fold increase in $[Ca^{2+}]o$ induced glial apoptosis in axotomized but not control samples. Unexpectedly, glial necrosis decreased in $3[Ca^{2+}]o$, but increased in $(1/3)[Ca^{2+}]o$. Inhibition of endoplasmic reticulum Ca-ATPase (SERCA) by thapsigargin induced glial apoptosis after AT but not in control. Calcium ionophore ionomycin induced glial apoptosis both after AT and in control samples. Fluphenazine, inhibitor of calmodulin did not influence glial apoptosis, but induced glial necrosis. Dantrolene and ochratoxin A that release Ca2+ from endoplasmic reticulum stimulated AT-induced glial apoptosis but not necrosis. The blockage of mitochondrial permeability pores with cyclosporine A, which prevented calcium release from mitochondria, reduced cell death. Thus, AT combination with high $[Ca^{2+}]o$ or with activation of pathways increased $[Ca^{2+}]i$ induced apoptosis of glial cells. Decrease in $[Ca^{2+}]o$, inhibition of SERCA, calmodulin, or Ca^{2+} ionophore promoted glial necrosis. Blockage of mitochondrial permeability pores protected glia from axotomy-induced death. So, axotomy induces necrosis and apoptosis of satellite glial cells and Ca^{2+} is involved in the detrimental effect of AT.

Biography

Anatoly B Uzdensky is a Head of the Laboratory of Molecular Neurobiology at Southern Federal University, Russia. He completed his PhD and Doctor of Science Degree in Physiology and Biophysics in 1980 and 2005, respectively. He is the Author of about 120 journal papers and four books. His current research interests include Neuroscience, Neuro-degeneration and Neuroprotection, Stroke, Cell-signaling and Proteomics.

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