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Cellular stress, apoptosis and autophagy
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Stress induced and physiological apoptosis during early development of sea urchin *Paracentrotus lividus*

M. Agnello[1], S. Filosto[1], F. Trinchella[2], *M.C. Roccheri*[1]
[1]Dip. Biologia Cellulare e dello Sviluppo "A. Monroy", Univ. Palermo (Palermo); [2]Univ. Federico II, Dip.di Scienze Biologiche, Sezione di Biologia Evolutiva e Comparata (Naples)

We investigate the toxic effect of cadmium on *P. lividus* embryo development. We already ascertained that 1mM and 100µM $CdCl_2$ induce apoptosis and found a competition between Cd and Ca uptake during early development. Using calcein-AM we show that intracellular amount of Ca in Cd-treated embryos is effectively lower than controls. We find cadmium to induce apoptosis through caspase-3 activation, while caspase-7 is the main effector of physiological apoptosis. Caspase-10 results active only in physiological apoptosis, since caspase-8 is predominantly involved in stress-induced apoptosis. Another stress inducer, the TPA, causes activation of all examined caspases. Immunocytochemical experiments with anti-cleaved-caspase-9 antibody, immunoblotting with anti- cytochrome-C antibody and fluorescent assays with JC-1 fluorochrome fail to find a mitochondrial involvement in apoptosis. Moreover, we show a detectable generation of reactive oxygen species, following exposure to Cd. We studied also caspases expressions during early physiological development and demonstrate that caspase-3 and caspase-10 are well-represented only after mesenchyme blastula stage.