

# ABCD

## ASSOCIAZIONE DI BIOLOGIA CELLULARE E DEL DIFFERENZIAMENTO

CON IL PATROCINIO DI



UNIVERSITÀ DEGLI  
STUDI DI PARMA



Comune di Parma



*Associazione Chiara Tassoni*  
per la ricerca sulla leucemia e  
sul cancro-ONLUS-PARMA

### XX Convegno Annuale ABCD sul Tema

## STRESS CELLULARE: SOPRAVVIVENZA ED APOPTOSI



17-18 Aprile 2009  
Starhotels Du Parc, Parma



Presidente: Pier Giorgio Petronini  
Organizzatori: Roberta Alfieri, Lina Ghibelli, Andrea Cavazzoni

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### ABSTRACTS

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## Sea urchin embryos: development and stress toxicity

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Heavy metals like Cadmium (Cd) are toxic as trace elements, others, like Manganese (Mn) play roles as nutrients but act as toxicants at elevated concentrations. In echinoderms, stress response is stage specific event, embryos are sensitive to pollutants, and often show alternative developmental phenotypes. Here, we investigated distinct and simultaneous effects of exposure to Cd and Mn in sea urchin (*P. lividus*) embryos and the capacity of these to withstand to each single insult, activating different strategy for protection. CdCl<sub>2</sub> exposure causes delay and morphological aberrations accompanied by the synthesis of specific stress proteins. Atomic Absorption Spectrometry (AAS) analysis pointed out the progressive accumulation of the metal in embryos, causing apoptosis. Here we demonstrate that ROS increase significantly during Cd insult. Moreover, we show the interference between Cd and Ca uptake during development, suggesting the competition for using the same ionic channels. Likewise, we investigated on effects of Mn on embryos cultured in the presence of the MnCl<sub>2</sub> from fertilization. Mn produce specific malformation in a time- and dose-dependent manner: we found an increase in the pluteus abnormality showing retarded, arms lacking, abnormal apex and skeleton defects. The highest concentration completely inhibits elongation of spicules in about 80% of embryos. By AAS analysis we found that: Mn is accumulated into the embryos 24 hrs after fertilization, while Ca concentration is reduced. The inhibition of spicule formation and perturbation to the skeleton-producing cells (PMC) migration into the blastocoel could be linked to the competition between Mn and Ca for membrane transport sites or other specific molecular targets. 2DE proteins were analyzed in control and exposed embryos, highlighting protein expression differences in response to Mn exposure. We explored if Mn would produce a stress response in embryos, by the up-regulation of synthesis of hsp's or by apoptosis. We found Mn exposure does not induce Hsp's neo-synthesis, neither triggers apoptosis nor starts ROS production, but cause an increase in some Hsc levels. Phosphorylation events mediated by protein kinases (ERK and p38 MAPK) were investigated of Mn-treated embryos. Both kinases showed a modify activation-times in Mn-treated embryos. The effects of Cd/Mn co-treatment were studied on embryos treated with a Mn concentration 10-fold molar excess with respect to Cd concentration. We found several abnormalities, developmental delay and apoptosis as results of summary of the single metal treatment.