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Polychlorinated biphenyls (PCBs) are organochlorine compounds with persistent and bioaccumulative properties that can accumulate in soil, plants and animals so entering in the food chain[1]. They are also classified as "Endocrine Disruptor Chemicals" (EDCs) because they can interact with several functions of endocrine system. PCB congeners are similar in thyroid hormone (TH) structure and are able to interact with TH receptors (TR) leading to destruction of the normal thyroid homeostasis[2,3]. Our study was designed to evaluate the effects of PCBs in lizards living on PCBs-contaminated soil. The soil for the terraria was taken from three areas with different concentrations of PCBs from the Bagnoli brownfield area situated into the western part of the city of Naples (Campania region), Southern Italy. The concentrations of ∑PCB in the soil were 2.55 mg/kg (low-dose: Group A), 4.31 mg/kg (medium-dose: Group B) and 7.60 mg/kg (high-dose: Group C). After 120 days, blood samples were collected to perform hormonal dosages and thyroid gland was removed and weighed for histopathological analysis. We have demonstrated that PCB-polluted soil induced a dose- and time-dependent reduction of lizard weight and a 40% of mortality in group C. Moreover, PCB pollution induced a strong increase of TRH plasma levels but a dose-dependent decrease of TSH, T3 and T4 plasma levels. Hormonal reduction was also confirmed by histological feature showing a strong decrease of thyroid epithelium height. Altogether, our results suggest that PCB pollution in the soil was able to negative affect functionality of thyroid gland with a persistent inhibition pituitary-thyroid gland.

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VANADIUM INDUCES CALCIUM DEPLETION AND CELL SELECTIVE APOPTOSIS DURING DEVELOPMENT OF SEA URCHIN EMBRYOS

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Vanadium (V) is a metal widely distributed in soil, water and air. It has recently received growing interest because its compounds are often used in different applications, from industry to medicine.[1] Here, using atomic absorption spectrometry, we demonstrate the predisposition of V to accumulate directly into embryonic cells, interfering with Ca uptake. At the morphological level, we observed dose- and time-dependent effects on phenotypes and on skeletal malformations. At the molecular level, V-exposed embryos showed the activation of the cellular stress response, inducing Hsp 60 and Hsp 70 synthesis and the activation of autophagy and apoptosis. The Hsps-mediated stress response to V appeared to counteract the damage induced by low (50 nM and 100 nM) and intermediate (500 nM and 1 µM) concentrations, while high cytotoxic doses (500 µM and 1 mM) induced more marked cell death mechanisms starting at 24 h of development, when the control embryos reached the gastrula stage.[2] Only few cells showed nuclei with apoptotic DNA fragmentation, particularly in the ectodermal layer. Mesodermal and endodermal cells did not appear to be involved in this process of selective apoptosis.[3] Microscopic fluorescence inspections indicated that primary mesenchyme cells (PMCs) were not involved in apoptotic processes; therefore, their inability to carry on the skeletogenesis could be due to the Ca depletion. These results allow us to elect the sea urchin embryo as a suitable experimental model for studying the metal-correlated cellular/molecular responses.[4,5]

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VANADIUM PERTURBS THE FERTILIZATION OUTCOME AND THE METALLOPROTEINASE ACTIVITY IN SEA URCHIN EMBRYOS

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Metal toxicology represents a current major topic due to the dispersion of these elements in the environment. Metals are released from both natural sources and industrial activities. Some of them have also a clinical interest due to their application as metallodrugs (*i.e.*, Pt, Cu, Au, Ru, and Y) or in medical diagnosis (Gd).[1,2] Recently, V derivatives are considered as potential therapeutic factors in some diseases (*e.g.*, obesity, diabetes, cancer, neurodegenerative and heart disorders). As a consequence, pharmaceutical residues could represent emerging pollutants of aquatic environments, as wastewater treatment plants do not sufficiently remove these compounds[3]. Embryonic models represent an adequate system for testing metal toxicity as they are sensitive to these elements. Here, we analysed the effects of different V concentrations, from very cytotoxic (1mM) to environmentally relevant doses (50nM), using two approaches: the fertilization test (FT) and the metalloproteinase (MMPs) activity.[4] We observed that V affected, in a dose-dependent manner, the percentage of fertilization and increased abnormalities regarding the egg and/or the fertilization membrane morphology. MMPs could represent another marker of V toxicity since it generates a cellular imbalance of metal ions. This would disturb the catalytic mechanism of these enzymes as they require ions as cofactors. Therefore, their dysfunction could represent a biomarker of metal-induced damage. We observed a total of 9 MMPs. Those with high molecular weight (from 309 to 59kDa) seemed to be mainly induced by elevated V concentrations (1mM, 500µM and 100µM). Conversely, low molecular weight MMPs (from 34 to 22kDa) appeared to be completely inhibited by these high V doses. On the other hand, lower V concentrations seemed to be more tolerated as there were no significant differences compared to control. In conclusion, FT and MMP activity could represent a reliable method to test V toxicity, using the sea urchin as a sensitive model system.

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