ble for toxicity (i.e., molecular mode of action). Cholinesterase (ChE) enzymes have a relevant role among enzymatic biomarkers used to assess sub-lethal effects of widespread contaminants. Heavy metals, one of the most pervasive classes of contaminants, have potentially adverse effects on both the environment and human health. Although not conventionally asserted as anti-ChE compounds, their interference with ChE activity has been described, though few studies have addressed the molecular mechanisms underlying this potential deleterious effect. The main purpose of the present study was to elucidate the inhibition mechanisms of various ChEs by inorganic mercury. The results of our studies on the impact of mercury toxicity on ChEs suggested that the type and effectiveness of ChE inhibition by mercury strongly depends on the biological origin of the ChE. The aim of this presentation is to discuss the use of ChE activity as a general biomarker of exposure to anti-ChE agents (including metals) using a mechanistic-based approach.

Biotransformation and role of arsenic in the polychaete, *Sabella spallanzanii*

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Abstract

The Mediterranean polychaete, Sabella spallanzanii, is characterized by elevated basal levels of arsenic in branchial crowns (>1000 ppm) and an unusual prevalence of relatively toxic dimethylarsinic acid (DMA). The aim of this work was to further investigate the capability of this polychaete to accumulate this arsenic compound and/or to generate it from precursor molecules via biotransformation reactions.. Organisms were exposed to arsenic in different chemical forms, i.e., arsenate (As^V), DMA, trimethylarsine (TMA) and arsenobetaine (AsB). Comparison of bioaccumulation and chemical speciation of arsenic revealed significant differences among tissues and experimental design. In branchial crowns, the highest increase of arsenic content was caused by arsenate exposure, which enters the cells through the phosphate carrier system; lower arsenic levels were measured following exposures to DMA and TMA. While not significant, changes of total As also occurred after treatment with AsB. In body tissues, exposure to As^V, DMA, TMA confirmed a progressively lower accumulation of total arsenic, while a marked increase was caused by AsB. DMA was the most accumulated molecule in all treatments, suggesting that S. spallanzanii can mediate methylation and de-methylation reactions of inorganic and tri-methylated arsenicals, thus explaining the elevated basal levels of DMA typical of this species. Arsenobetaine was not converted into DMA, confirming a microbial degradation of this molecule, particularly important in body tissues containing bacteria associated with digestive tracts. Overall, this study supports the biological role of arsenic in S. spallanzanii as an adaptive mechanism in more vulnerable tissues, providing a biological interpretation for their limited palatability.

Cadmium accumulation and metallothionein induction in *Solea senegalensis*

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Abstract

It is well known that metallothioneins (MTs) are induced by metals and provide a protective role by binding them to cysteine residues. Among metals, cadmium has a high capacity to induce these proteins. To examine the usefulness of this biomarker for cadmium pollution, the flatfish, Solea senegalensis, was injected intraperitoneally (2.5 mg Cd kg⁻¹ wet weight). Metal accumulation and metallothionein levels were measured in liver, gill, kidney and whole digestive system at days 0, 3 and 6 after dosing. The hepatic MT levels were significantly induced by cadmium after 3 days compared to controls, then decreased at day 6, as did the Cd concentration, indicating their role in detoxification and elimination of metals in this organ. Concentrations of MTs and Cd measured in digestive system were elevated until day 3 then the metal concentration remained constant, probably due to its lower capacity of elimination. A similar tendency, increasing MT concentrations after 3 days, was observed for gills and kidneys. These results are inadequate to confirm whether MTs are suitable biomarkers of exposure to cadmium pollution. MT induction was detected at the initial phase of exposure, when other biochemical processes may not play a significant role in metal storage or excretion. Moreover, the route of exposure is also an important factor that may affect the function of MTs.

Cloning and regulation of the peroxisomal β-oxidation genes (acyl-CoA oxidase, multifunctional protein, thiolase) in thicklip grey mullets and mussels after treatment with organic xenobiotics

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Abstract

Exposure to some organic compounds may cause peroxisome proliferation in different animal species. Notably, exposure to peroxisome proliferators causes an increase of the peroxisomal β-oxidation activity that in rodents is due to a transcriptional up-regulation of the genes coding for the three enzymes in the pathway. These genes have been found to be duplicated in most of the genomes studied to date. In the present study, palmitoyl-CoA oxidase (AOX), multifunctional protein (MFP) and 3-ketoacyl-CoA thiolase (THIO) were cloned in the thicklip grev mullet (Chelon labrosus) and mussel (Mytilus galloprovincialis) using degenerate primers. Their expression was measured semiguantitatively after exposure to a typical mammalian peroxisome proliferator, perfluorooctanosulfonate (PFOS), and after injection with benzo(a)pyrene. Since AOX is the rate limiting enzyme in the pathway, the whole ORF was cloned by RACE-PCR revealing similarities with functionally characterized vertebrate AOX genes. The typical FAD binding motif and the conserved PTS1 peroxisomal targeting sequence were detected. The inducible form of MFP (MFP1) was cloned in mullets and the non-inducible MFP2 in mussels. Exposure to PFOS resulted in a significant overexpression of AOX in both organisms, while benzo(a)pyrene injection significantly induced AOX, MFP1 and THIO expression in mullets. In conclusion, peroxisomal β-oxidation genes are transcriptionally inducible in selected aquatic organisms under treatment with PFOS and benzo(a)pyrene. Future studies