

**CADMIUM TOXIC EFFECTS ON HEART VENTRICLE
OF HALOBATRACHUS DIDACTYLUS–
CHRONIC EXPOSURE STUDY**

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EXTENDED ABSTRACT ONLY – DO NOT CITE

Introduction

Due to its potential as a toxic substance, cadmium is one of the metals to which a special statute in questions of environmental and health is given. As no biological function has been attributed to cadmium, this metal is toxic for the cell, even in low concentrations. Various studies connect cadmium with oxidative stress, since this metal can alter the antioxidant defence system in several tissues of several animals, causing a depletion in the levels of reduced glutathione, as well as an alteration in the activity of antioxidant enzymes, and a change in the structure of the cellular membrane through a process of lipid peroxidation (Jamall et al., 1989; Palace et al., 1993; Sarkar et al., 1995; Zikic et al., 1996). Cadmium has a high potential of toxicity, mainly in the liver and kidney. However, its interaction with the cardiac muscle cell is not well understood. The objective of this work was to analyse antioxidant defence system responses induced by a chronic exposure to a sub-lethal cadmium concentration (1 mg/kg), on heart ventricle of a teleost fish, *Halobatrachus didactylus* (toadfish).

Materials and Methods

H. didactylus individuals were collected from Ria Formosa (South Coast of Portugal) and divided into two groups: Control group (CTRL), injected intraperitoneally (i.p.) at day 0 with 0.9% NaCl, and sacrificed after 6 weeks; Cadmium exposure group (Cd), injected i.p. at day 0 with 1 mg/kg of Cd as CdCl₂ in NaCl 0.9%, and sacrificed after 6 weeks of exposure.

Heart ventricle from each individual was collected after sacrifice and cytosolic and mitochondrial fractions were prepared for determination of superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (total GPx and Se-GPx) activities. Lipid peroxidation products were also analysed using TBA method. Cadmium concentrations were determined in total, cytosolic and mitochondrial fractions using flame/furnace atomic absorption spectroscopy.

All parameters studied are presented as averages of measurements taken from 5 individuals in each group. The Mann-Whitney test, the non-parametric equivalent of the analysis of variance between two treatments, was applied to test differences between groups, on all the parameters analysed. The significant level used was 5% ($\alpha=0.05$).

Results and Discussion

The results indicate alterations of the antioxidant defence systems (Table I): SOD seems to be inhibited both in mitochondrial and cytosolic fraction; CAT and GPx activities are not affected significantly after 6 weeks exposure. Subcellular Cd distribution is presented in Table II.

In the experimental conditions described, there was a significant increase in lipid degradation products levels. These results, together with those of the antioxidant enzymes activity, indicate an ineffective response of the cellular defence mechanisms in protecting the cell against the oxidative stress caused by this metal. Lipid peroxidation can be one of the consequences of oxidative stress, a situation that usually occurs when the production of reactive oxygen species (ROS) exceeds that of the antioxidant defence systems. The ROS can be inactivated through the action of antioxidant enzymes or other unspecific antioxidants.

Prior studies in this specie, with the same Cd concentration but in 1 day and 1 week of exposure (Correia et al., 1998), indicate a strong and effective response of the antioxidant enzymes and no significant increase in lipid peroxidation. These results together seem to show that, although antioxidant mechanisms protect the cell during an acute Cd exposure, this protection is not sufficient in longer periods and cellular injury may result.

The process of lipid peroxidation determines the alteration in the structure of cell membrane (Cheeseman, 1993). Future studies are needed to clarify functional consequences of the membrane injury in ventricle tissue.

Table I – Percentual variation of antioxidant enzymes activity and lipid degradation products after 6 weeks Cadmium exposure.

Parameter	Fraction	% variation
		6 weeks
SOD	cytosolic	-22,4*
	mitochondrial	-23,7*
CAT	cytosolic	-10,9
	mitochondrial	8,4
GPx	total	4,6
	Se-GPx	-5,3
Lipid peroxidation	total	36,1*

* significant differences with CTRL groups.

Table II – Subcellular Cadmium distribution ($\mu\text{g/g}$) in the heart ventricle of *Halobatrachus didactylus*.

Fractions	0days	6weeks	
	CTRL	CTRL	Cd
Total	0,341	0,615	5,879
Mitochondrial	0,000	0,235	1,817
Cytosol	0,000	0,044	0,632

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