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Editorial:

METAL TOXICITY

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Metal Toxicity is one of the cutting-edge topics of our partner journal Archives of Toxicology. Unlike many other compounds metals are not degraded facilitating the accumulation of toxic concentrations (Beyersmann and Hartwig, 2008). Mechanisms of metal toxicity include oxidative stress induction, DNA repair inhibition, modification of cell proliferation and cadherin interference. Due to the multitude of mechanisms involved and the target structures, metal toxicity represents an extremely complex field of research. To give our readers an overview of recent publications on metal toxicity we have summarised their key messages in Table 1.

Table 1: Recent results in research on **metal toxicity**

Key message	Reference
Potassium dichromate inhibits brush border membrane enzymes and causes oxidative stress in rat intestine.	Arivarasu et al., 2008
This article reviews the toxic mechanisms of aluminium and lead in brain: membrane biophysics, altered cell signalling and the impairment of neurotransmission are highlighted.	Verstraeten et al., 2008
The paper describes mechanisms on how methyl-mercury suppresses arginase I activity: covalent modification of MeHg and leakage of Mn ions from the active site.	Kanda et al., 2008a
Cadmium, cobalt and lead cause characteristic alterations of gene expression patterns in cultivated human bronchial epithelial cells.	Glahn et al., 2008
No interaction between in vivo relevant concentrations of arsenic and malathion was observed in a rat in vivo study.	Naraharisetti et al., 2008
This is a comprehensive review about the molecular mechanisms of carcinogenic metals.	Beyersmann and Hartwig, 2008
A meta-analysis shows that lead may reduce nerve conduction velocity. The lowest lead blood concentration at which a relationship could be detected was 33.0 microg/dl.	Krieg et al., 2008
Cadmium alters transferrin and hepcidin expression in fish.	Chen et al., 2008
Copper gluconate may act as a rat liver carcinogen as evidenced by a medium-term liver carcinogenicity protocol.	Abe et al., 2008
Diphenylarsinic acid is a product of degradation of arsenic-containing chemical weapons and was detected in well water in Japan. This is a pharmacokinetic study in cynomolgus monkeys describing distribution and excretion of arsenic after repeated administration of diphenylarsenic acid.	Kobayashi et al., 2008
Lead acetate administered into the yolk sac causes developmental neurotoxicity in chicks.	Müller et al., 2008

Key message	Reference
Lead induced foetal developmental toxicity in mice is enhanced by meso-2,3-dimercaptosuccinic acid.	Yu et al., 2008
Methyl mercury and mercuric sulfide modify pentobarbital induced hypnotic tolerance.	Chuu et al., 2008
Cisplatin-induced nephrotoxicity in rats is attenuated by the phytoalexin resveratrol.	Do Amaral et al., 2008
Arsenic-induced oxidative myocardial injury in mice is ameliorated by arjunolic acid.	Manna et al., 2008
Inorganic mercury accumulates in the proximal tubules of the kidney and causes apoptosis. Apoptosis induction in this context is antagonized by overexpression of arginase II.	Kanda et al., 2008b
An optimum combination of micronutrients (calcium and ascorbic acid) with 2,3-dimercaptosuccinic acid for the treatment of lead-intoxicated mice was established.	Liao et al., 2008
Vitamin D receptor gene variants are associated with circulating concentrations of lead in exposed humans.	Rezende et al., 2008

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