EXCLI Journal 2011;10:133-134 – ISSN 1611-2156
Received: August 17, 2011, published: August 18, 2011

Editorial:

METAL TOXICITY

Joanna D. Stewart

Leibniz Research Centre for Working Environment and Human Factors (***IfADo***), Ardeystrasse 67, 44139 Dortmund, Germany

E-mail: stewart@ifado.de, Telephone: +49 231-1084-406, Fax: +49 231-1084-403

Metals remain a major focus of current toxicological research because of their continued occupational and environmental occurrence and relevance (Beyersmann and Hartwig, 2008; Hengstler et al., 2003; Glahn et al., 2008). The mechanisms underlying how metals damage the mitochondrial membrane, thus inducing oxidative stress, have been summarized in the review of Kumar and Gill (2009); a "must-read" for anyone interested in mitochondrial toxicity. The table summarizes the take home messages of recent publications on metal toxicity.

Table 1: Research on metal toxicity

Key message	Reference
This review focuses on the mechanisms by which aluminium induces mitochondrial toxicity and oxidative stress as possible causes of neurobehavioural changes.	Kumar and Gill, 2009
Sub-chronic, low level exposure to methylmercury causes hypertension in rats by nitric oxide depletion and generation of reactive oxygen species.	Grotto et al., 2009
Lead acetate compromises mitochondrial membrane potential in rat proximal tubular cells and causes apoptotic cell death.	Wang et al., 2009
Inorganic arsenite in drinking water (100 mg/l) may affect learning and memory functions in offspring rats.	Xi et al., 2009
This study analyzes the time course of arsenic species in the brain and liver of mice and presents evidence for the existence of a mechanism that actively clears dimethyl arsenic acid from the brain.	Juárez-Reyes et al., 2009
Bicyclol pre-administration can prevent the nephrotoxicity induced by cisplatin in mice.	Yu and Chen, 2009
Lead nitrate binds to histone proteins and causes condensation of DNA.	Rabbani-Chadegani et al., 2009
The use of Fluoro-Jade for histological staining of neurons after exposure to neurotoxic chemicals was evaluated.	Schmuck and Kahl, 2009
^{137}Caesium impairs vitamin D metabolism in the offspring of rats following maternal exposure.	Tissandie et al., 2009
Selenium protects against methylmercury induced DNA damage.	Grotto et al., 2009
Arsenic induces suicidal erythrocyte death.	Mahmud et al., 2009
Long term exposure to depleted uranium causes renal dysfunction in rats.	Zhu et al., 2009
The expression of organic anion transporters Oat1 and Oat3 were decreased in renal bosolateral membranes after the exposure of rats to a nephrotoxic dose of $HgCL_2$. Oat1 and 3 are involved in $HgCL_2$ uptake, suggesting that their down regulation is a protective mechanism.	Di Giusto et al., 2009

REFERENCES

Beyersmann D, Hartwig A. Carcinogenic metal compounds: recent insight into molecular and cellular mechanisms. Arch Toxicol 2008;82:493-512.

Di Giusto G, Anzai N, Ruiz ML, Endou H, Torres AM. Expression and function of Oat1 and Oat3 in rat kidney exposed to mercuric chloride. Arch Toxicol 2009;83: 887-97.

Glahn F, Schmidt-Heck W, Zellmer S, Guthke R, Wiese J, Golka K et al. Cadmium, cobalt and lead cause stress response, cell cycle deregulation and increased steroid as well as xenobiotic metabolism in primary normal human bronchial epithelial cells which is coordinated by at least nine transcription factors. Arch Toxicol 2008;82:513-24.

Grotto D, de Castro MM, Barcelos GR, Garcia SC, Barbosa F Jr. Low level and sub-chronic exposure to methylmercury induces hypertension in rats: nitric oxide depletion and oxidative damage as possible mechanisms. Arch Toxicol 2009;83:653-62.

Hengstler JG, Bolm-Audorff U, Faldum A, Janssen K, Reifenrath M, Götte W et al. Occupational exposure to heavy metals: DNA damage induction and DNA repair inhibition prove co-exposures to cadmium, cobalt and lead as more dangerous than hitherto expected. Carcinogenesis 2003;24: 63-73.

Juárez-Reyes A, Jiménez-Capdeville ME, Delgado JM, Ortiz-Pérez D.Time course of arsenic species in the brain and liver of mice after oral administration of arsenate. Arch Toxicol 2009;83:557-63.

Kumar V, Gill KD. Aluminium neurotoxicity: neurobehavioural and oxidative aspects. Arch Toxicol 2009;83:965-78.

Mahmud H, Föller M, Lang F. Arsenic-induced suicidal erythrocyte death. Arch Toxicol 2009;83:107-13.

Rabbani-Chadegani A, Abdosamadi S, Fani N, Mohammadian S. A comparison of the effect of lead nitrate on rat liver chromatin, DNA and histone proteins in solution. Arch Toxicol 2009;83:565-70.

Schmuck G, Kahl R. The use of Fluoro-Jade in primary neuronal cell cultures. Arch Toxicol 2009;83:397-403.

Tissandie E, Guéguen Y, Lobaccaro JM, Grandcolas L, Grison S, Aigueperse J et al. Vitamin D metabolism impairment in the rat's offspring following maternal exposure to 137cesium. Arch Toxicol 2009;83:357-62.

Wang L, Wang H, Hu M, Cao J, Chen D, Liu Z. Oxidative stress and apoptotic changes in primary cultures of rat proximal tubular cells exposed to lead. Arch Toxicol 2009;83:417-27.

Xi S, Sun W, Wang F, Jin Y, Sun G. Transplacental and early life exposure to inorganic arsenic affected development and behavior in offspring rats. Arch Toxicol 2009; 83:549-56.

Yu YN, Chen H, Li Y. Protect effect of bicyclol on cisplatin-induced nephrotoxicity in mice. Arch Toxicol 2009;83:381-7.

Zhu G, Xiang X, Chen X, Wang L, Hu H, Weng S. Renal dysfunction induced by long-term exposure to depleted uranium in rats. Arch Toxicol 2009;83:37-46.