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Heavy Metal Toxicity and Therapeutics

Honglian Shi*

Pharmacology and Toxicology, University of Kansas, USA

It has long been known that heavy metal ions can be toxic and carcinogenic. Many metals such as arsenic and chromium induce cellular damages and carcinogenesis by increasing free radical generation and lowering cellular antioxidative defense capacity [1]. Targets of redox signaling induced by metals have been suggested to be potential therapeutic interventions in metal-induced toxicity and carcinogenesis. Aluminum is a known risk factor for neurodegeneration such as those in Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis. In the issue of volume 4 issue 4, Thirunavukkarasu et al. [2] reported that aluminum and its salts extensively damaged nervous system and impaired learning and memory of rats. More interestingly, they demonstrated that *Manasamitra vatakam* (*MMV*), a berbo-mineral formulation, provided protection against aluminum-induced toxicity in the rats. Furthermore, MMV markedly increased synthesis and release of neurotransmitter 5-hydroxytryptamine (5-HT) and decreased the expression of apoptotic genes such as *Bcl-2, Bcl-xL* and *caspas-3*.

Yet, it is not clear how MMV improves the level of 5-HT and apoptotic proteins in the rats exposed to aluminum. In addition, aluminum is known to cause oxidative stress. Does MMV effect on the antioxidative defense system? If so, how? Further investigation is needed to provide more insight to the mechanisms of MMV-mediated brain function improvement in neurodegeneration.

References

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***Corresponding author:** Honglian Shi, Associate professor, Pharmacology and Toxicology, University of Kansas, USA, Tel: 785-864-6192; Fax: 785-864-5219; E-mail: hshi@ku.edu

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