

## The Potential Role of Some Nutrients and Drugs in Providing Protection from Neurotoxicity Induced by Aluminium in Rats

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**Abstract :** Background: Aluminium (Al) represents an environmental risk factor. Exposure to high levels of Al causes neurotoxic effects and different diseases. Vinpocetine is widely used to improve cognitive functions, it possesses memory-protective and memory-enhancing properties and has the ability to increase cerebral blood flow and glucose uptake. Cocoa bean represents a rich source of iron as well as a potent antioxidant. It can protect from the impact of free radicals, reduces stress as well as depression and promotes better memory and concentration. Wheatgrass is primarily used as a concentrated source of nutrients. It contains vitamins, minerals, carbohydrates, amino acids and possesses antioxidant and anti-inflammatory activities. Coenzyme Q10 (CoQ10) is an intracellular antioxidant and mitochondrial membrane stabilizer. It is effective in improving cognitive disorders and has been used as anti-aging. Zinc is a structural element of many proteins and signaling messenger that is released by neural activity at many central excitatory synapses. Objective: To study the role of some nutrients and drugs as Vinpocetine, Cocoa, Wheatgrass, CoQ10 and Zinc against neurotoxicity induced by Al in rats as well as to compare between their potency in providing protection. Methods: Seven groups of rats were used and received daily for three weeks AlCl<sub>3</sub> (70 mg/kg, IP) for Al-toxicity model groups except for the control group which received saline. All groups of Al-toxicity model except one group (non-treated) were co-administered orally together with AlCl<sub>3</sub> the following treatments; Vinpocetine (20mg/kg), Cocoa powder (24mg/kg), Wheat grass (100mg/kg), CoQ10 (200mg/kg) or Zinc (32mg/kg). Biochemical changes in the rat brain as acetyl cholinesterase (ACHE), A $\beta$ , brain derived neurotrophic factor (BDNF), inflammatory mediators (TNF- $\alpha$ , IL-1 $\beta$ ), oxidative parameters (MDA, SOD, TAC) were estimated for all groups besides histopathological examinations in different brain regions. Results: Neurotoxicity and neurodegenerations in the rat brain after three weeks of Al exposure were indicated by the significant increase in A $\beta$ , ACHE, MDA, TNF- $\alpha$ , IL-1 $\beta$ , DNA fragmentation together with the significant decrease in SOD, TAC, BDNF and confirmed by the histopathological changes in the brain. On the other hand, co-administration of each of Vinpocetine, Cocoa, Wheatgrass, CoQ10 or Zinc together with AlCl<sub>3</sub> provided protection against hazards of neurotoxicity and neurodegenerations induced by Al, their protection were indicated by the decrease in A $\beta$ , ACHE, MDA, TNF- $\alpha$ , IL-1 $\beta$ , DNA fragmentation together with the increase in SOD, TAC, BDNF and confirmed by the histopathological examinations of different brain regions. Vinpocetine and Cocoa showed the most pronounced protection while Zinc provided the least protective effects than the other used nutrients and drugs. Conclusion: Different degrees of protection from neurotoxicity and neuronal degenerations induced by Al could be achieved through the co-administration of some nutrients and drugs during its exposure. Vinpocetine and Cocoa provided the most protection than Wheat grass, CoQ10 or Zinc which showed the least protective effects.

**Keywords :** aluminum, neurotoxicity, vinpocetine, cocoa, wheat grass, coenzyme Q10, Zinc, rats

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