Abstract Information

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Address :	EDEN UNIVERSITY LUSAKA ZAMBIA
Participation :	symposium
Title of the Symposium :	The Brain Environment and Nutraceuticals: Combating Neurodegeneration through
	Neuroprotection
Category :	Academic/Researcher
Thematic Area :	Neuropharmacology, and Ethnopharmacology
Title :	Apoptotic Inducement of Neuronal Cells by Aluminium Chloride and The Neuroprotective
	Effects of Eugenol in Wistar rats.
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Aluminium is known to accelerate oxidative stress, amyloid beta (A?) deposition, and plaque Abstract : formation in the brain of rats. The present study is aimed at studying the neuroprotective effects of eugenol following aluminium-induced neurotoxicity on caspase-3, apoptotic proteins (Bcl-2 and Bax), and oxidative stress markers in Wistar rats such as superoxide dismutase (SOD), glutathione peroxidase (GPx), nitric oxide (NO), and assay oxidative stress to mitochondrial DNA (mtDNA) by measuring the levels of 8-hydroxy-2-deoxyguanosine (8-OHdG). Materials and methods. Twenty (20) adult Wistar rats were randomly divided into four (4) groups with five animals in each group. Route of administration was oral throughout the duration of this study and this study lasted for 21 days. Rats were sacrificed 24 hours after administration of the last dose (i.e., day 22) with 0.8?mg/kg ketamine as an anaesthetic agent. Results. Exposure to AICI3 resulted in a significant (p<0.01) elevation in the levels of nitric oxide and 8-hydroxy-2-deoxyguanosine (8-OHdG), enhanced the activity of caspase-3, increased the level of proapoptotic protein Bax and reduced the levels of antiapoptotic protein Bcl-2, and significantly (p<0.01) reduced the levels of SOD and GPx. However, treatment with eugenol resulted in a significant reduction (p<0.01) in the levels of nitric oxide (NO) and 8-hydroxy-2-deoxyguanosine (8-OHdG) levels, inhibited the activity of caspase-3, increased

levels of Bcl-2 and significantly (p<0.05) reduced levels of Bax protein, respectively, and also significantly (p<0.05) increased the levels of SOD and GPx. Conclusion. Our results would hereby suggest that eugenol would provide a therapeutic value against aluminium-induced oxidative stress as related to antioxidant and antiapoptotic activities.

Keywords: Aluminium Chloride, Neurotoxicity, Antipapoptotic Protein, Antioxidant, 8-hydroxy-2-deoxyguanosine.