World Academy of Science, Engineering and Technology
International Journal of Energy and Environmental Engineering
Vol:11, No:01, 2017

The Possible Interaction between Bisphenol A, Caffeine and Epigallocatechin-3-Gallate on Neurotoxicity Induced by Manganese in Rats

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Abstract : Background: Manganese (Mn) is a naturally occurring element. Exposure to high levels of Mn causes neurotoxic effects and represents an environmental risk factor. Mn neurotoxicity is poorly understood but changing of AChE activity, monoamines and oxidative stress has been established. Bisphenol A (BPA) is a synthetic compound widely used in the production of polycarbonate plastics. There is considerable debate about whether its exposure represents an environmental risk. Caffeine is one of the major contributors to the dietary antioxidants which prevent oxidative damage and may reduce the risk of chronic neurodegenerative diseases. Epigallocatechin-3-gallate is another major component of green tea and has known interactions with caffeine. It also has health-promoting effects in CNS. Objective: To evaluate the potential protective effects of Caffeine and/or EGCG against Mn-induced neurotoxicity either alone or in the presence of BPA in rats. Methods: Seven groups of rats were used and received daily for 5 weeks MnCl2.4H2O (10 mg/kg, IP) except the control group which received saline, corn oil and distilled H2O. Mn was injected either alone or in combination with each of the following: BPA (50 mg/kg, PO), caffeine (10 mg/kg, PO), EGCG (5 mg/kg, IP), caffeine + EGCG and BPA +caffeine +EGCG. All rats were examined in five behavioral tests (grid, bar, swimming, open field and Y- maze tests). Biochemical changes in monoamines, caspase-3, PGE2, GSK-3B, glutamate, acetyl cholinesterase and oxidative parameters, as well as histopathological changes in the brain, were also evaluated for all groups. Results: Mn significantly increased MDA and nitrite content as well as caspase-3, GSK-3B, PGE2 and glutamate levels while significantly decreased TAC and SOD as well as cholinesterase in the striatum. It also decreased DA, NE and 5-HT levels in the striatum and frontal cortex. BPA together with Mn enhanced oxidative stress generation induced by Mn while increased monoamine content that was decreased by Mn in rat striatum. BPA abolished neuronal degeneration induced by Mn in the hippocampus but not in the substantia nigra, striatum and cerebral cortex. Behavioral examinations showed that caffeine and EGCG co-administration had more pronounced protective effect against Mn-induced neurotoxicity than each one alone. EGCG alone or in combination with caffeine prevented neuronal degeneration in the substantia nigra, striatum, hippocampus and cerebral cortex induced by Mn while caffeine alone prevented neuronal degeneration in the substantia nigra and striatum but still showed some nuclear pyknosis in cerebral cortex and hippocampus. The marked protection of caffeine and EGCG co-administration also confirmed by the significant increase in TAC, SOD, ACHE, DA, NE and 5-HT as well as the decrease in MDA, nitrite, caspase-3, PGE2, GSK-3B, the glutamic acid in the striatum. Conclusion: Neuronal degeneration induced by Mn showed some inhibition with BPA exposure despite the enhancement in oxidative stress generation. Co-administration of EGCG and caffeine can protect against neuronal degeneration induced by Mn and improve behavioral deficits associated with its neurotoxicity. The protective effect of EGCG was more pronounced than that of caffeine even with BPA co-exposure.

Keywords : manganese, bisphenol a, caffeine, epigallocatechin-3-gallate, neurotoxicity, behavioral tests, rats
Conference Title : ICEPPC 2017 : International Conference on Environmental Pollution and Pollution Control
Conference Location : London, United Kingdom
Conference Dates : January 19-20, 2017