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Influence of Protein Malnutrition and Different Stressful Conditions on Aluminum-Induced Neurotoxicity in Rats: Focus on the Possible Protection Using Epigallocatechin-3-Gallate

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Abstract : Background: Aluminium (Al) is known as a neurotoxin environmental pollutant that can cause certain diseases as Dementia, Alzheimer's disease, and Parkinsonism. It is widely used in antacid drugs as well as in food additives and toothpaste. Stresses have been linked to cognitive impairment; Social isolation (SI) may exacerbate memory deficits while protein malnutrition (PM) increases oxidative damage in cortex, hippocampus and cerebellum. The risk of cognitive decline may be lower by maintaining social connections. Epigallocatechin-3-gallate (EGCG) is the most abundant catechin in green tea and has antioxidant, anti-inflammatory and anti-atherogenic effects as well as health-promoting effects in CNS. Objective: To study the influence of different stressful conditions as social isolation, electric shock (EC) and inadequate Nutritional condition as PM on neurotoxicity induced by Al in rats as well as to investigate the possible protective effect of EGCG in these stressful and PM conditions. Methods: Rats were divided into two major groups; protected group which was daily treated during three weeks of the experiment by EGCG (10 mg/kg, IP) or non-treated. Protected and non-protected groups included five subgroups as following: One normal control received saline and four Al toxicity groups injected daily for three weeks by ALCl3 (70 mg/kg, IP). One of them served as Al toxicity model, two groups subjected to different stresses either by isolation as mild stressful condition (SI-associated Al toxicity model) or by electric shock as high stressful condition (EC- associated Al toxicity model). The last was maintained on 10% casein diet (PM -associated Al toxicity model). Isolated rats were housed individually in cages covered with black plastic. Biochemical changes in the brain as acetyl cholinesterase (ACHE), Aβ, brain derived neurotrophic factor (BDNF), inflammatory mediators (TNF-α, IL-1β), oxidative parameters (MDA, SOD, TAC) were estimated for all groups. Histopathological changes in different brain regions were also evaluated. Results: Rats exposed to Al for three weeks showed brain neurotoxicity and neuronal degenerations. Both mild (SI) and high (EC) stressful conditions as well as inadequate nutrition (PM) enhanced Al-induced neurotoxicity and brain neuronal degenerations; the enhancement induced by stresses especially in its higher conditions (ES) was more pronounced than that of inadequate nutritional conditions (PM) as indicated by the significant increase in Aβ, ACHE, MDA, TNF-α, IL-1β together with the significant decrease in SOD, TAC, BDNF. On the other hand, EGCG showed more pronounced protection against hazards of Al in both stressful conditions (SI and EC) rather than in PM .The protective effects of EGCG were indicated by the significant decrease in Aβ, ACHE, MDA, TNF-α, IL-1β together with the increase in SOD, TAC, BDNF and confirmed by brain histopathological examinations. Conclusion: Neurotoxicity and brain neuronal degenerations induced by Al were more severe with stresses than with PM. EGCG can protect against Al-induced brain neuronal degenerations in all conditions. Consequently, administration of EGCG together with socialization as well as adequate protein nutrition is advised especially on excessive Al-exposure to avoid the severity of its neuronal toxicity.

Keywords : environmental pollution, aluminum, social isolation, protein malnutrition, neuronal degeneration, epigallocatechin-3-gallate, rats
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