Neuroprotective potential of Aqueous Cinnamon Extract on cerebellar Amyloid-β plaques in Alzheimer’s Disease.

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Background Aluminum (AL) is a neurotoxic substance that plays an important role in the etiology, pathogenesis and development of Amyloid-β plaques. This study was carried out to evaluate neuroprotective effect of Aqueous Cinnamon Extract against aluminum chloride (ALCl₃)-induced Alzheimer’s disease.

Materials and methods 40 adult male albino rats, randomly divided into four equal groups: Group I: control; Group 2 were administered 200 mg/kg b. w./day aqueous cinnamon extract (ACE) via oral tube; Group 3: received daily intraperitoneal (i.p.) injection of ALCl₃ (100 mg/kg body weight) for 60 days to induce neurotoxicity; Group 4 received a combination of ALCl₃ and ACE in the same dose and route as group 2 & 3. Results AL administration significantly enhanced the memory impairment, Aβ burden and the cerebellum exhibited significant reduction in the number of Purkinje cells, marked decrease in the density of dendritic arborization and prominent perineuronal spaces in the molecular layer around basket and stellate cells, loss of dendritic spines, neurofibrillary degeneration and appearance of neuritic plaques. Concomitant administration of ACE with AL displayed an observable protection against these changes with progressive improvement in memory and in intellectual performance. It has also been suggested that the loss of astrocyte functions may precede neurodegeneration and that aluminum could be a contributing factor [1]. Astrocytes are the principal target of the toxic action of aluminum [2] and it can cause astrocyte death through apoptosis [3]. Conclusion: ACE may be considered an efficacious therapeutic strategy to alleviate Amyloid-β plaques. It is recommended that AL not be used in cooking utensils, water tubes, and to control occupational exposure.

![Fig (3A)](image1)
![Fig (3B)](image2)
![Fig (3C)](image3)
![Fig (3D)](image4)

References