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Preventive role of Melatonin against Aluminum Chloride Induced rat model of Alzheimer's disease by attenuating Amyloid β accumulation and Endoplasmic Reticulum stress in the rat brain

Alzheimer's disease (AD) is a progressive neurodegenerative disease characterized clinically by cognitive decline and memory loss. Aluminium (Al) has been proposed to be one of the environmental factors responsible to cause AD. This experimental study investigated the neuroprotective effect of melatonin in aluminium chloride (AlCl3) induced rat model of AD. Twelve week old male Wistar rats were used as expeimental subjects and divided into four groups on the basis of specific dose supplementation for eight weeks: group one orally received 175 mg/kg AlCl3, group two orally received 175 mg/kg AlCl3 plus 5 mg/kg melatonin intraperitoneally, group three intraperitoneally received 5 mg/kg melatonin and group four (control) orally received saline solution. A series of behavioral tests including Morris water maze and Novel object recognition test, blood biochemical tests and expression of AD associated proteins in brain were determined in all treatments. Results of the behavioral tests revealed that AlCl3 treatment did not induced memory and cognition impairment. However, melatonin treatment attenuated amyloid beta (Aβ) (1-42) level by decreasing β-secretase, augmented low-density lipoprotein receptor-related protein 1 and neprilysin protein expression. Moreover, aluminum-induced elevation in endoplasmic reticulum (ER) stress and oxidative stress was decreased by melatonin. These results suggested that melatonin protected against Aβ peptide accumulation, ER stress, and oxidative stress in AlCl3-treated rats. Hence, melatonin supplement might be an alternative way to alleviate development of AD.

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