ORIGINAL ARTICLE

AMELIORATIVE EFFECT OF VITAMIN C AND UV-B RAYS ON NIGROSTRIATAL AND CORTICOSTRIATAL DEGENERATION IN HALOPERIDOL INDUCED PARKINSONISM IN WISTAR RATS

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ABSTRACT

Prolonged inhibition of dopamine-2 receptor (D2R) is shown to cause degeneration of dopamine neurons leading to parkinsonism. Previously we have shown that vitamin D3 receptor stimulation improved motor–cognitive functions in dopamine-2 receptor (D₂R) parkinsonian mice model. Presently, we examined the ameliorative effect of vitamin C and UV-B rays on nigrostriatal and corticostratal degeneration in drug induced parkinsonism in Wistar rats. Twenty male Wistar rats with average weight of 120 g were distributed into four groups (NS, -D2, -D2+UV-B and –D2+Vit.C). Parkinsonism was induced by administering 10 mg/kg b.wt. of haloperidol for 14 days (intraperitoneally) without and with treatment of 125 mg/kg b.wt. of vitamin C or 2 hours of exposure to morning sunlight between 8-10 am. The animals were subjected to cylinder, pole and stepping test for motor functions. Motor cortex (M1), substantia nigra pars compacta (SNc) and striatum (CPu) were processed and stained using haematoxylin and eosin and Cresyl violet stains. Cell count was done using ImageJ software (version 5). Data were presented as mean ± standard error of mean; analysed using one-way analysis of variance and Tukey's multiple comparison test, and significant level was determined at 0.05 (p < 0.05*).

Haloperidol induced parkinsonism caused significant bradykinesia (*p < 0.05), rigidity (** p < 0.01), neuron lost (**p < 0.01) and expressions of degeneration hallmarks in SNc and M1. UV-B or Vit. C treatment showed ameliorative potentials in reducing motor deficit experience in parkinsonism, but not regenerating the already lost neurons.

Key words: Parkinsonism, Vitamin C, UV-B rays, Dopamine-2 receptor blocker, Substantia nigra (SN), Striatum (CPu), Motor cortex (M1)

INTRODUCTION

Several studies have shown that prolonged inhibition of dopamine-2 receptor (-D2R) causes loss of dopamine neurons, resulting in to parkinsonism (Shirayama et al. 2000; Iderberg et al. 2015). These dopamine neurons project from substantia nigra to striatum (nigrostriatal tract), and are also found in the cortex (corticostratal tract) (Singh 2009). It is known that haloperidol has the ability to centrally block dopamine-2 receptors (Seeman and Tallerico 2003;
REFERENCES


