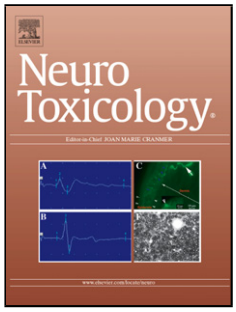

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Authors: Harikesh Dubey, Kavita Gulati, Arunabha Ray

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Amelioration by Nitric oxide (NO) mimetics on neurobehavioral and biochemical changes in experimental model of Alzheimer`s disease in rats

Harikesh Dubey, Kavita Gulati and Arunabha Ray*

Department of Pharmacology, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi-110 007, India

Corresponding author:
Prof. Arunabha Ray, Department of Pharmacology, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi-110 007, India
(E-mail: arunabha14@yahoo.co.in)

Abstract

The present study evaluated the effects of s-nitrosoglutathione (GSNO), a nitrosothiol and sustained NO releaser, on experimental model of sporadic Alzheimer`s disease (sAD) in rats. Levels of Aβ40, Aβ42 and BDNF were assessed in brain hippocampal homogenates for correlative purposes. Intracerebroventricular-Streptozotocin (icv-STZ) induced increased escape latencies (acquisition) and reduced time in target quadrant (probe trial) in Morris Water Maze (MWM) test at 3 months post icv-STZ administration. These behavioural changes were associated with increased Aβ depositions and lowered BDNF levels in brain hippocampal homogenates. Pre-treatment with GSNO (50 µg/kg/day), reduced the icv-STZ induced cognitive deficits in acquisition and probe trials in the MWM. The icv-STZ induced elevations in Aβ40 and Aβ42 and reduced levels of BDNF in hippocampal homogenates were also attenuated after GSNO treatment in these rats. The NO-precursor, L-arginine (100 mg/kg) induced similar effects on behavioural and biochemical parameters tested but was marginally less consistent as compared to those seen with GSNO. The results suggest that GSNO ameliorates the cognitive deficits and associated brain biochemical changes in this experimental model of sporadic AD, and NO-BDNF interactions could play crucial role in these effects.