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Partial Scholarship Award Winner

# Glial activation in nitrous oxide toxicity is related to oxidative stress and glutamate excitotoxicity

Sandeep Kumar Singh, U.K. Misra and J. Kalita

Department of Neurology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, INDIA

Presenting author: [sandeepcjb@gmail.com](mailto:sandeepcjb@gmail.com)

## Abstract

Myelin disorders can be due to diverse mechanisms such as autoimmune, parainfectious, metabolic or toxic. The prototype of immune mediated demyelination is multiple sclerosis. To understand the underlying mechanism of cell damage in vitamin b12 deficiency, a number of animal models have been used which include total gastrectomy (TGX), cobalamine deficient diet and N<sub>2</sub>O exposure (Tredici G, et al., 1998; Scalabrino G, 2001). Six adult wistar male rats were exposed to N<sub>2</sub>O oxygen mixture in 1:1 ratio at a rate of 2 L/min for 120 min for 60 days. The control rats received only oxygen and room air. At the end of exposure, spontaneous locomotor activity (total distance travelled, time resting, time moving, number of rearing, stereotypic count) and grip strength. Plasma glutathione (GSH), total antioxidant capacity (TAC) and serum malondialdehyde (MDA) and serum homocysteine (Hcy) were measured by spectrophotometer. Glutamate in the cerebral cortex and cerebellum was measured by colorimetry. Immunohistochemistry for GFAP expression in brain and spinal cord was done and quantified using image J software. The N<sub>2</sub>O exposed rats had significant reduction in total distance travelled, time moving, number of rearing and increased time resting compared to the controls. Hcy, glutamate and MDA levels were increased, and GSH and TAC decreased in N<sub>2</sub>O exposed group compared to the controls. GFAP was more expressed in N<sub>2</sub>O exposed group, and its expression was higher in spinal cord compared to brain. The GFAP expression correlated with neurobehavioral changes, oxidative stress and glutamate level. N<sub>2</sub>O toxicity results in GFAP expression suggesting astrocytic reaction, which is mediated by oxidative stress and excitotoxicity.

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