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Nitric Oxide Modulatory Mechanism in the Protective Effect of Retigabine Against Spinal Nerve Ligation Induced Neuropathic Pain

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Introduction: Decreasing the hyper excitability of neurons through opening of voltage-gated potassium (Kv7, also termed as KCNQ) channels has been suggested as one of the protective mechanisms in the effective management of neuropathic pain. Reactive oxygen/nitrogen species and inflammatory pathways are well implicated in the pathophysiology of neuropathic pain. Further, M current generated by opening of KCNQ channels has been modulated by reactive oxygen/nitrogen species.

Aim & Objectives: The present study has been designed to elucidate the nitric oxide modulatory mechanism in the protective effect of retigabine against spinal nerve ligation induced neuropathic pain in rats.

Methods: Ligation of L5 and L6 spinal nerves resulted alterations in various behavioral (as evident from marked increase in thermal and mechanical hyperalgesia and allodynia), biochemical (raised lipid peroxidation, nitrite, & depletion of GSH, SOD, catalase) and inflammatory parameters (raised TNF-alpha) as compared to naive treatment.

Results: Administration of retigabine (10 mg/kg) for 28 days attenuated these behavioral, biochemical and inflammatory cascades as compared to control. Further, L-arginine (100 mg/kg) pretreatment with retigabine (5 mg/kg) significantly reversed the protective effect of retigabine in spinal nerve ligated rats. However, L-NAME (10 mg/kg) pretreatment with retigabine (5 mg/kg) significantly potentiated their protective effects which were significant as compared to their effect per se respectively.

Conclusions: Present study highlights the involvement of nitric oxide mechanism in the protective effect of retigabine against L5/L6 spinal nerve ligation induced behavioral and biochemical alterations in rats.

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