Turkish Journal of Medical Sciences

Turkish Journal

of

Medical Sciences

Nimodipine Aggravates Systemic Kainic Acid Toxicity in Retinal Ganglion Cells of Intact Mice

Akçahan GEPDIREMEN[1]
Murat KAYA[2]
Ibrahim KOÇER[2]
Cemal GÜNDOĞDU[3]
Departments of [1]Pharmacology, [2]Ophthalmology and [3]Pathology, Faculty of Medicine, Atatürk University, Erzurum-Turkey

Keywords
Authors

medsci@tubitak.gov.tr

Scientific Journals Home Page

Abstract: The aim of the present study was to investigate the role of the L-type voltage dependent Ca^{2+} channel blocker, nimodipine, in kainate induced toxicity in retinal ganglion cells of mice. Kainate in 10mg/kg was administered intraperitoneally following the vehicle or nimodipine. Nimodipine, 45 minutes prior to kainate in 10, 15, 25, 50, 100 and 250 mg/kg doses was administered intraperitoneally. Surprisingly, nimodipine potentiated the ganglional cell death induced by kainate, with respect to the control and kainate+vehicle treated group. In accordance with our results, blockage of L-type calcium channels by nimodipine may worsen the prognosis and survival of retinal neurons in kainate receptor stimulated toxicity. Blocking of calcium channels may lead to the rebound activation of sodium channels or stimulate the release of intracellular calcium by activating ryanodine sensitive calcium channels.

Key Words: Kainate, retina, nimodipine, neurotoxicity, mice.

Turk J Med Sci 2000; **30**(3): 213-218.
Full text: pdf
Other articles published in the same issue: Turk J Med Sci,vol.30,iss.3.