Neurosci Lett. 2000 Aug 25;290(2):105-8.

Early-onset tolerance in rat global cerebral ischemia induced by a mitochondrial inhibitor.

Nakase H, Heimann A, Uranishi R, Riepe MW, Kempski O.

Institute for Neurosurgical Pathophysiology, Johannes Gutenberg-University Mainz, Langenbeckstrasse 1, 55101, Mainz, Germany.

was studied whether a subtoxic dose of the mitochondrial neurotoxin, 3-nitropropionic acid (3-NPA), can initiate early-onset tolerance induction for subsequent ischemic injury. Wistar rats were pretreated for 3 h by intraperitoneal 3-NPA (20 mg/kg body weight; n=13) or solvent (n=12). Fifteen minutes global cerebral ischemia was induced by bilateral carotid artery occlusion and hypobaric hypotension. rCBF and tissue hemoglobin oxygen saturation were measured by laser Doppler scanning and a microspectrophotometric method. Ischemic insult and brain temperature were identical in both groups. Body weight and neurological scores recovered in the pretreated group but further deteriorated in the non-treated group ($P<0.05$). Quantitative histology demonstrated a better neuronal density in neocortex and hippocampal CA2, CA3, and CA4 of pretreated animals ($P<0.05$).