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The effects of glycyrrhetinic acid derivatives on picrotoxin-induced epileptiform activity

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The common features of all types of epilepsy are synchronized and uncontrolled discharges of nerve cell ensembles. The reason underlying the pathologically synchronized discharges of the neurons is not precisely known yet. Recent studies asserted that gap junctional intercellular communication has a critical role in epileptic neuronal events. The aim of present study is to investigate the effects of glycyrrhetinic acid derivatives, gap junctional blocker, on picrotoxin-induced epileptiform activity. In the present study, 4-month-old male Sprague Dawley rats were used for this purpose. Permanent screw electrodes allowing EEG monitoring from conscious animals and guide cannula providing the infusion of the substance to the brain ventricle were placed in to the cranium of rats under anesthesia. At the end of the postoperative recovery period, epileptiform activity was induced by injecting picrotoxin (1 μ g) through the ventricular guide cannula. Epileptiform activity monitored from data acquisition system, when it reached its peak level, 200 nmol dose of 18 α -glycyrrhetinic acid, 18 β -glycyrrhetinic acid, 18 β -glycyrrhetinic acid and carbenoxolone decreased mean spike frequency to 49%, 49% and 59%, respectively. At the 60 min of recording, spike amplitudes were 32%, 29% and 30% less than control values, respectively. Eventually, 18 α -glycyrrhetinic acid, 18 β -glycyrrhetinic acid and carbenoxolone attenuated epileptiform activity by decreasing spike frequency and amplitude of epileptiform activity.

Biography

M Ömer Bostanci has completed his PhD from Ondokuz Mayis University and Post-doctoral studies from Ondokuz Mayis University School of Medicine. He is the Head of Department of Physiology and Deputy Dean of Medical Faculty of Hitit University. He has published more than 20 papers in reputed journals.

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