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Research Article

Myoinositol Attenuates the Cell Loss and Biochemical Changes Induced by Kainic Acid Status Epilepticus

Lia Tsverava,[1] Tamar Lordkipanidze,[1,2] Eka Lepsveridze,[1] Maia Nozadze,[1,2] Marina Kikvidze,[1] and Revaz Solomonia[1,2]

[1]*Institute of Chemical Biology, Ilia State University, 3/5 K. Cholokashvili Avenue, 0162 Tbilisi, Georgia*
[2]*I. Beritashvili Center of Experimental Biomedicine, 14 L. Gotua Street, 0160 Tbilisi, Georgia*

Correspondence should be addressed to Revaz Solomonia; revaz_solomonia@iliauni.edu.ge

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Identification of compounds preventing or modifying the biochemical changes that underlie the epileptogenesis process and understanding the mechanism of their action are of great importance. We have previously shown that myoinositol (MI) daily treatment for 28 days prevents certain biochemical changes that are triggered by kainic acid (KA) induced status epilepticus (SE). However in these studies we have not detected any effects of MI on the first day after SE. In the present study we broadened our research and focused on other molecular and morphological changes at the early stages of SE induced by KA and effects of MI treatment on these changes. The increase in the amount of voltage-dependent anionic channel-1 (VDAC-1), cofilin, and caspase-3 activity was observed in the hippocampus of KA treated rats. Administration of MI 4 hours later after KA treatment abolishes these changes, whereas diazepam treatment by the same time schedule has no significant influence. The number of neuronal cells in CA1 and CA3 subfields of hippocampus is decreased after KA induced SE and MI posttreatment significantly attenuates this reduction. No significant changes are observed in the neocortex. Obtained results indicate that MI posttreatment after KA induced SE could successfully target the biochemical processes involved in apoptosis, reduces cell loss, and can be successfully used in the future for translational research.

1. Introduction

Epilepsy is a heterogeneous syndrome characterized by recurrent and spontaneous seizures. Approximately 1% of the population in the world suffers from epilepsy. However, 20%–30% of the patients are refractory to therapies using currently available antiepileptic drugs (AEDs) [1]. Current epilepsy therapy is symptomatic using AEDs. This therapy suppresses seizures but does not prevent or cure epilepsy. Thus, treatment strategies that could interfere with the process leading to epilepsy (epileptogenesis) would have significant benefits over the current approach [1–3] and will be of great importance for epilepsy treatment. Unfortunately, at present, there is no drug which could fulfill these demands and effectively prevent the process of epileptogenesis in humans. The alternative goal for epileptogenesis treatment would be disease modification, which means that although a treatment may not prevent the occurrence of a disease, it may nevertheless modify the natural course of the disease [1]. Disease modification after epileptogenic brain insults may affect the development of spontaneous seizures in that the seizures, if not prevented, are less frequent and less severe [1].

Some native plants of the Ranunculaceae family (to which plant *Aquilegia vulgaris* belongs) are widely used in Chinese and Tibetan folk medicine as antiepileptic and soporific medicaments [4]. In our early studies we discovered that water extract of *Aquilegia vulgaris* contains compounds which act on γ-aminobutyric acid- (GABA-) A receptors; namely, it completely inhibits ^{3}H-muscimol (a GABA-A receptor agonist) binding to rat brain membranes and also increases ^{3}H-flunitrazepam (a specific ligand for the GABA-A receptor benzodiazepine site) binding by approximately a factor of two [4]. γ-Aminobutyric acid (GABA) is the main inhibitory neurotransmitter of the mammalian central