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Evaluation of the neuroprotective effect of taurine and green tea extract against oxidative stress induced by pilocarpine during status epilepticus

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Abstract Status epilepticus (SE) has functional and structural consequences resulting in brain damage. The present study aims to investigate the role of taurine and green tea extract in the neuroprotection against oxidative stress and changes in acetylcholinesterase (AChE) and Na^+,K^+-ATPase activities during SE induced by pilocarpine in the hippocampus of adult male rats. Animals received an oral administration of either taurine (100 mg/kg) or green tea extract containing 100 mg/kg epigallocatechin gallate for 3 days before the induction of SE with pilocarpine (380 mg/kg, i.p.) and were sacrificed 1 h after pilocarpine injection. Data indicated that a state of oxidative stress has evolved during SE as evident from the significant increase in lipid peroxidation level and significant decrease in reduced glutathione (GSH) level. Significant decreases in AChE and Na^+,K^+-ATPase activities were also recorded. Pretreatment of rats with taurine exaggerated the increase in lipid peroxidation and failed to prevent the decrease in Na^+,K^+-ATPase activity resulting from pilocarpine. However, taurine pretreatment prevented the reduced activity of hippocampal

Abbreviations: ACh, acetylcholine; AChE, acetylcholinesterase; EC, (−)-epicatechin; ECG, (−)-epicatechin gallate; EGC, (−)-epigallocatechin; EGCG, (−)-epigallocatechin gallate; GSH, reduced glutathione; GST, glutathione-S-transferase; LP, lipid peroxidation; MDA, malondialdehyde; NO, nitric oxide; ROS, reactive oxygen species; SE, status epilepticus; SOD, superoxide dismutase; SRS, spontaneous recurrent seizures; TLE, temporal lobe epilepsy

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AChE induced by pilocarpine during SE. Pretreatment of rats with green tea extract prevented the increase in lipid peroxidation occurring during SE. However, it failed to inhibit the decrease in Na^+,K^+-ATPase activity. In conclusion, taurine pretreatment failed to reduce the oxidative stress induced during SE. In contrast, pretreatment of rats with green tea extract ameliorated the oxidative stress induced by pilocarpine and this may assist in reducing the insults of hyperexcitability and excitotoxicity that occur during SE and thereby reduce neuronal damage.

Introduction

Status epilepticus (SE) is a severe clinical manifestation of epilepsy and has functional and structural consequences resulting in brain damage (Matzen et al., 2008). In this condition, a single seizure or a series of seizures lasts for 30 min or more without restoration of consciousness, and is associated with a significant morbidity and mortality, including neuronal damage and dysfunction (Fountain, 2000). SE is the first phase that occurs during the evolution of temporal lobe epilepsy (TLE). SE is followed by a silent phase and spontaneous recurrent seizures (SRS) (Cavalheiro et al., 1994).

The hippocampus has been a focus of interest in temporal lobe epilepsy research because it contains several well-described neuronal circuits linked to seizure onset and because it develops, in the time course of the disease, a severe loss of pyramidal cells in CA1, CA3 and the dentate gyrus (Pitkanen and Lukasiuk, 2009).

It is well known that when reactive oxygen species (ROS) production is excessive, the intrinsic antioxidant scavenging capacity is overwhelmed resulting in the development of oxidative stress which can induce tissue injury and may activate apoptosis processes (Todorova et al., 2004). The hippocampus is particularly susceptible to lipid peroxidation due to the simultaneous presence of high levels of polyunsaturated fatty acids and iron (Halliwell and Gutteridge, 1989). In addition, the hippocampus presents a low number of antioxidant systems (de Freitas, 2010).

Oxidative stress can dramatically alter neuronal function and has been associated with neurochemical changes observed during SE and SRS induced by pilocarpine (Barros et al., 2007). Moreover, oxidative stress and mitochondrial dysfunction could be acute consequences of SE that are related to the mechanism of seizure-induced neuronal cell death and subsequent epileptogenesis (Patel, 2004). Therefore, antioxidant compounds have been of great interest as potential therapies for treatment of epilepsy (Golden and Patel, 2009).

Taurine (2-aminoethansulfonic acid) is present at high concentrations in the mammalian brain (Guidotti et al., 1972). Several functions of taurine have been reported, including neuroprotection (Chen et al., 2001), neuromodulation (Kuiyama, 1980) and neurotransmission (Taber et al., 1986).

Animal studies have shown that taurine has anticonvulsant action (Huxtable and Nakagawa, 1985). In some animal studies, a significant reduction in seizure frequency was observed (König et al., 1977; Takahashi and Nakane, 1978). The intracerebroventricular injection of taurine restored the normal-like electroencephalographic features during the interictal period in pilocarpine-treated rats during spontaneous recurrent seizures (Radwan, 2001) whereas no benefit was seen in other studies (Mantovani and DeVivo, 1979). Moreover, taurine has been shown to hyperpolarize neurons in the hippocampus (del Olmo et al., 2000) by opening chloride channels through its interaction with $GABA_A$ receptors (Bureau and Olsen, 1991), glycine receptors (Hussy et al., 1997), or taurine receptors (Wu et al., 1992). Moreover, taurine has antioxidant properties (Chen et al., 2012), due to its ability to improve mitochondrial function by stabilizing the electron transport chain and inhibiting the generation of reactive oxygen species (Jong et al., 2012).

Green tea is one of the plant products that have significant effects on human health. It contains many polyphenolic compounds, including (−)-epigallocatechin gallate (EGCG), (−)-epigallocatechin (EGC), (−)-epicatechin gallate, and (−)-epicatechin. Of these, EGCG is the most abundant and most effective antioxidant having one diphenolic and two triphenolic groups (Kondo et al., 1999). Green tea polyphenols can penetrate the blood–brain barrier and remain in the brain for more than 24 h (Suganuma et al., 1999). Green tea, its extract and its isolated constituents were found to be effective in preventing oxidative stress (Babu et al., 2006) and brain problems (Unno et al., 2007). Several studies suggested that green tea polyphenols might protect against Parkinson's and Alzheimer's diseases and other neurodegenerative diseases (Pan et al., 2003; Weinreb et al., 2004). Previously, in an electroencephalographic study, Yokoi et al. (1989) examined the effects of EGC or EGCG on the occurrence of epileptic discharges in EEGs induced by the injection of iron ions into the sensorimotor cortex of rats. The authors found that pretreatment with EGC or EGCG prevented the occurrence of epileptic discharges in some rats and slowed them in other rats. They suggested that EGC or EGCG scavenged active oxygen free radicals to prevent the formation of an epileptic focus. A recent study of Kang et al. (2010) showed that EGCG has a strong protective effect against hippocampal neuronal oxidative stress and cell death both *in vitro* and *in vivo*.

The main objective of the present study was to gain further insight into the role of taurine and green tea in the neuroprotection against oxidative stress and changes in acetylcholinesterase (AChE) and Na^+,K^+-ATPase enzyme activities resulting from pilocarpine-induced SE in adult male rats.