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Oral Presentation

Reactive Oxygen Species and Epilepsy

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Abstract

Seizure activity has been proposed to result in the generation of reactive oxygen species (ROS), which then contribute to seizure-induced neuronal damage and eventually cell death. Although the mechanisms of seizure-induced ROS generation are unclear, mitochondria and cellular calcium overload have been proposed to have a crucial role. We aim to determine the sources of seizure-induced ROS and their contribution to seizure-induced cell death. Live cell imaging techniques in glio-neuronal cultures and in ex vivo epileptic brain tissue. We show that prolonged seizure-like activity increases ROS production in an NMDA receptor-dependent manner. Unexpectedly, however, mitochondria did not contribute to ROS production during seizure-like activity. ROS were generated primarily by NADPH oxidase and later by xanthine oxidase (XO) activity in a calcium-independent manner. Inhibition of NADPH or XO markedly reduced seizure-like activity-induced neuronal apoptosis. In addition, ROS were upregulated in chronic epilepsy in ex vivo brain slices. Inhibition of ROS production in vivo by AEBSF, a NADPH oxidase inhibitor, markedly reduced seizure-induced cell death. These findings demonstrate a critical role for ROS, generated by NADPH oxidase, contributing to seizure-induced cell death. These findings point to NADPH oxidase inhibition as a novel treatment strategy to prevent brain injury in seizures, status epilepticus and chronic epilepsy.

Keywords: Cell Death, Epilepsy, Brain Injury

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