Microglia might lessen seizure severity in epilepsy

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New research in mice highlights the potential protective effect of microglia—a type of non-neuronal cell in the brain—against overactivation of the central nervous system during acute epileptic
seizures. The study is published in the *American Journal of Physiology-Cell Physiology*.

Epilepsy is a brain disorder in which the electrical activity of the nerve cells (neurons) in the brain becomes disrupted. Overactivation—also called overexcitation—of neurons causes a seizure. Previous studies suggest that astrocytes, another type of non-*neuronal cell* in the brain, play an important role in maintaining balance of these electrical signaling patterns. Microglia are also *immune cells*, and become chronically activated when they sense inflammation or *nerve damage* in the central nervous system. However, the role microglia play in acute epilepsy is less clear.

Researchers studied a mouse model of epilepsy in mice with depleted stores of microglia and a *control group* with normal levels of microglia. The research team used electroencephalography testing to measure electrical activity in the animals' brains during a seizure. Compared to the controls, the mice without microglia had more severe seizures and more extensive neuronal degeneration (excitotoxicity). Excitotoxicity is damage or death of nerve cells that occurs when they are overstimulated, such as during a seizure.

"Our study indicates that microglia are innately ready to quench acute seizures and protect neurons from excitotoxicity-induced degeneration," the researchers wrote. These results pave the way for additional studies that may "reveal in the future how *microglia* can immediately respond to neuronal overexcitation and take a protective role against seizures and neuronal excitotoxicity," they added.

The study, titled "Microglia depletion exacerbates acute seizures and hippocampal neuronal degeneration in mouse models of epilepsy," is published in the *American Journal of Physiology-Cell Physiology*. 

Provided by American Physiological Society

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