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Microglia-neuron communication in Epilepsy

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Epilepsy represents a neurological disorder that can manifest in uncontrolled seizures in patients. Microglia are exquisitely sensitive to disruptions in the central nervous system. Since epilepsy is characterized by neuronal hyperactivity rooted in excessive glutamate release and ionic imbalance, it is conceivable that microglia responds to and regulates neuronal activities during the pathology. Here, we found an increased number of microglial primary processes in the hippocampus during kainic acid-induced seizure activity. Consistently, global glutamate induced robust Microglial Process Extension (MPE) towards neurons making increased contact with neurons in both brain slices and in the intact brain in vivo. The mechanism of the glutamate-induced MPE involves the activation of neuronal NMDA receptors, calcium influx, subsequent ATP release, and microglial response through P2Y12 receptors. In addition, we serendipitously found that extracellular $Ca2^{+}$ reduction induced microglial processes to converge at distinct sites, a phenomena we termed Microglial Process Convergence (MPC). Our studies revealed that MPC occurs independent of astrocytic functions and are not directed towards astrocytes but target neuronal dendrites. Similar to glutamate-induced MPE, extracellular $Ca2^{+}$-dependent MPC is also mediated by ATP and microglial P2Y12 receptor. Finally, we found that P2Y12 knockout mice exhibited reduced seizure-induced increases in microglial process numbers and worsened kainic acid-induced seizure behaviors. These studies are the first to investigate the microglial dynamics and discovered MPE and MPC during acute epilepsy. Our results elucidate the molecular mechanisms underlying microglia-neuron communication that may be potentially neuroprotective in the epileptic brain. Studying microglia-neuron communication in epilepsy informs the development of novel therapies targeting microglia in the treatment of epilepsy.

Biography

Long Jun Wu has completed his PhD from University of Science and Technology of China in 2004. After his PhD study, he was trained as a Post-Doctoral Fellow at University of Toronto (2004-2008) and Harvard Medical School (2008-2010), and was promoted to Instructor at Harvard Medical School (2011-2012). Since September 2012, he has been an Assistant Professor in the Department of Cell Biology and Neuroscience at Rutgers University. His lab at Rutgers mainly focuses on microglia-neuron communication in normal and diseased brain. He has published more than 72 peer-review research papers, including those in *Nature Neuroscience, Neuron, PNAS, Science Translational Medicine, Journal of Neuroscience,* Glia, etc.

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