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Glia and mast cells as targets for palmitoylethanolamide, an anti-inflammatory and neuroprotective lipid mediator.

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Abstract

Glia are key players in a number of nervous system disorders. Besides releasing glial and neuronal signaling molecules directed to cellular homeostasis, glia respond also to pro-inflammatory signals released from immune-related cells, with the mast cell being of particular interest. A proposed mast cell-glia communication may open new perspectives for designing therapies to target neuroinflammation by differentially modulating activation of non-neuronal cells normally controlling neuronal sensitization-both peripherally and centrally. Mast cells and glia possess endogenous homeostatic mechanisms/molecules that can be upregulated as a result of tissue damage or stimulation of inflammatory responses. Such molecules include the N-acylethanolamines, whose principal family members are the endocannabinoid N-arachidonoylethanolamine (anandamide), and its congeners N-stearoylethanolamine, N-oleoylethanolamine, and N-palmitoylethanolamine (PEA). A key role of PEA may be to maintain cellular homeostasis when faced with external stressors provoking, for example, inflammation: PEA is produced and hydrolyzed by microglia, it downmodulates mast cell activation, it increases in glutamate-treated neocortical neurons ex vivo and in injured cortex, and PEA levels increase in the spinal cord of mice with chronic relapsing experimental allergic encephalomyelitis. Applied exogenously, PEA has proven efficacious in mast cell-mediated experimental models of acute and neurogenic inflammation. This fatty acid amide possesses also neuroprotective effects, for example, in a model of spinal cord trauma, in a delayed post-glutamate paradigm of excitotoxic death, and against amyloid β-peptide-induced learning and memory impairment in mice. These actions may be mediated by PEA acting through "receptor pleiotropism," i.e., both direct and indirect interactions of PEA with different receptor targets, e.g., cannabinoid CB2 and peroxisome proliferator-activated receptor-alpha.

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