Glial modulation by N-acylethanolamides in brain injury and neurodegeneration

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Neuroinflammation involves the activation of glial cells and represents a key element in normal aging and pathophysiology of brain damage. N-acylethanolamides (NAEs), naturally occurring amides, are known for their pro-homeostatic effects. An increase in NAEs has been reported in vivo and in vitro in the aging brain and in brain injury. Treatment with NAEs may promote neuroprotection and exert anti-inflammatory actions via PPAR? activation and/or by counteracting gliosis. This review aims to provide an overview of endogenous and exogenous properties of NAEs in neuroinflammation and to discuss their interaction with glial cells. © 2016 Herrera, Kölliker-Frers, Barreto, Blanco and Capani.

Gliosis

N-acylethanolamides

Neuroinflammation

Neuroprotection

PPAR?

amide

anandamide

n acylethanolamide derivative

n oleoylethanolamine

palmidrol

peroxisome proliferator activated receptor

unclassified drug

aging

Alzheimer disease

antiinflammatory activity

astrocyte

astrocytosis

brain injury

brain ischemia

cell activation

cell interaction

cerebrovascular accident

degenerative disease

drug efficacy

drug structure

glia cell

gliosis

human

learning disorder

memory disorder

microglia

nerve degeneration

nervous system inflammation

neuromodulation

neuroprotection

nonhuman

Parkinson disease

pathophysiology

perinatal asphyxia

peripheral neuropathy

pleiotropy

receptor upregulation

Short Survey

signal transduction

spinal cord injury

traumatic brain injury