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Neuroscience Center of Excellence

School of Medicine

Chancellor's Award Lecture

in Neuroscience

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8th Floor
Neuroscience Center
of Excellence
Conference Room
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Aspects of inflammation in Alzheimer's disease – resolution and effects of systemic events

Alzheimer's disease (AD) is a neurodegenerative disease characterized by extra- and intracellular deposits of aggregated proteins in the brain as in the amyloid plaques and neurofibrillary tangles. In addition, there is an ongoing inflammatory process in the brain, evidenced by glial activation and increase in inflammatory mediators. The immune response of the organism is a mechanism to take care of injurious stimuli such as pathogens and mechanical injury and restore homeostasis, but inflammation can also lead to cell and tissue damage, especially if it becomes chronic, and our data suggest that chronic peripheral inflammation may induce AD-like pathology in mice. Some inflammatory factors also serve as messengers in neuronal functions. Inflammation normally ends by the resolution process that includes removal of inflammatory stimuli as well as tissue repair and restoration. Endogenously produced, so called specialized pro-resolving mediators (SPMs), derived from polyunsaturated fatty acids, are involved in the resolution. Evidence indicates a disturbed resolution process in the AD brain including alterations in proteins involved in SPM synthesis and signaling. In vitro studies indicate direct effects of SPMs on microglia and neurons, supporting the idea that stimulation of pro-resolving activities may be of potential benefit in AD and other neurodegenerative disorders.