Neuroinflammation

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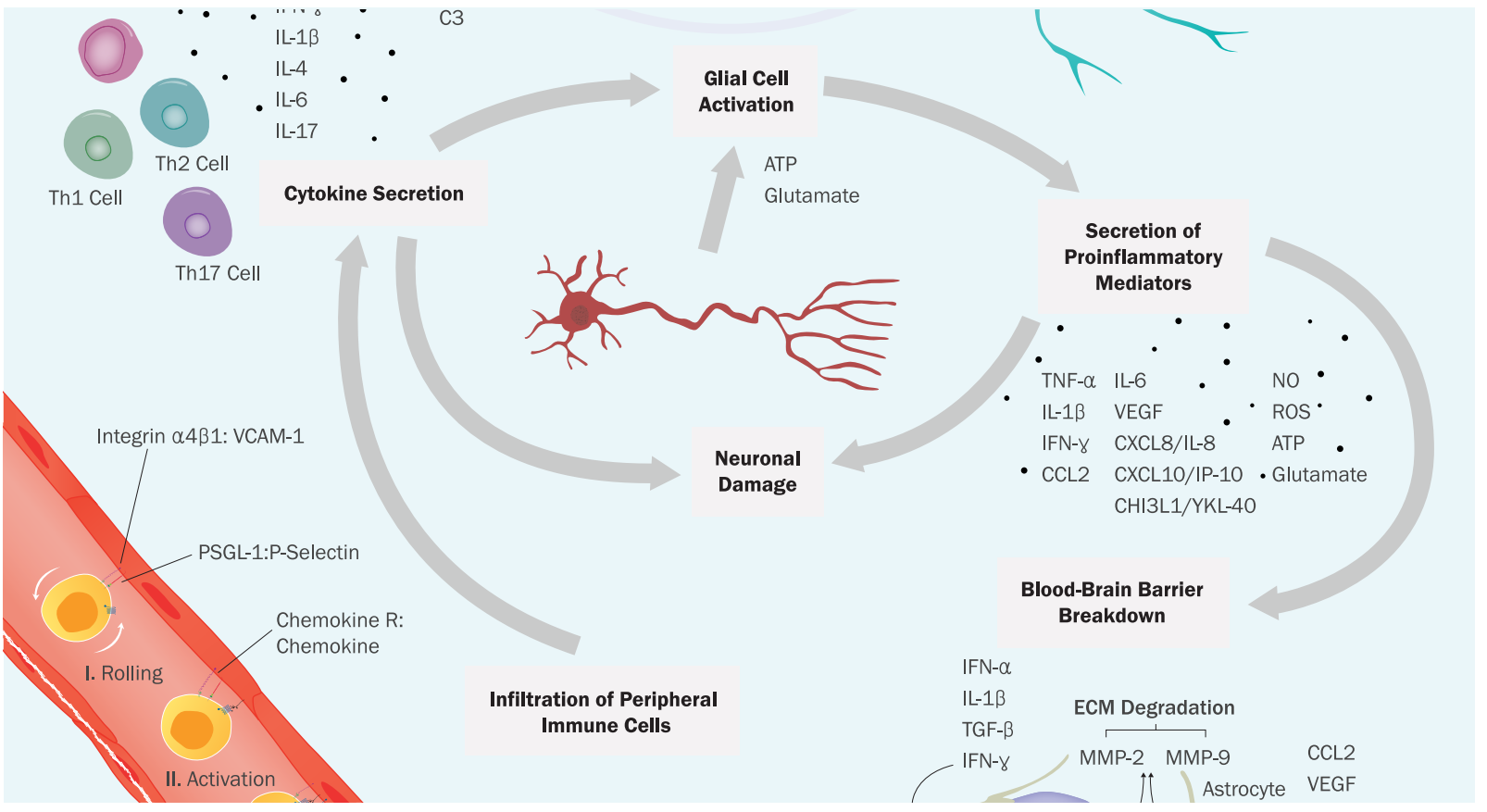

Neuroinflammation이란?

여러가지의 Response에 의하여 Glial cell의 활성화, 면역 매개체들의 분비, ROS의 생성 등 많은 상황이 펼쳐지는 일종의 Mechanism입니다.
Neuroinflammation의 원인은 다양하게 존재 하며 Neurodegeneration으로 발전 할 수 있는 경우도 존재합니다.
그림은 Neuroinflammation의 간략한 모식도 이며 자세한 그림은 브로셔 또는 그림을 클릭해 보세요.

PLOS one

Prolonged Neuroinflammation after Lipopolysaccharide Exposure in Aged Rats Click HERE…

PLOS | ONE

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LPS-treated rats showed up-regulated mRNA expression and protein levels of pro-inflammatory cytokines in the hippocampus. These changes associated with astrogliosis in the hippocampus dentate gyrus (DG), IL-1b immunoreactivity and elevated NF-kB p65 expression up to day 30 post LPS exposure. Overall, these data demonstrate that LPS induces prolonged neuroinflammation and astrocyte activation in the hippocampus of aged rats. Hippocampal NF-kB p65 and excessive astrocytes-derived IL-1b release may play a pivotal role in regulating longlasting neuroinflammation.

ELSEVIER - Journal of Autoimmunity

Myeloid sphingosine-1-phosphate receptor 1 is important for CNS autoimmunity and neuroinflammation

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reported that C57BL/6J mice harboring phosphorylation defective S1P receptor 1 (S1P1) (with mutated serines in the carboxyl terminus, leading to impaired receptor internalization) [S1P1(S5A)] developed severe, TH17-dominant experimental autoimmune encephalomyelitis. In this study, we demonstrate that S1P1-mediated TH17 polarization is not an intrinsic T cell effect, but dependent on sustained S1P1 signaling in myeloid cells. First, utilizing the S1P1(S5A) mice in the EAE model, we observed that S1P1 activated and enhanced antigen presentation function in myeloid cells. Second, sequential phosphorylation of STAT3 occurred in dendritic cells, monocytes, and macrophages/microglia during neuroinflammation. Third, we show that pro-inflammatory (CD45hiCD11b+Ly6Chi) monocytes contribute to TH17 differentiation and neuroinflammation by regulating IL-6 expression.

Journal of Neuroscience

EP3R-Expressing Glutamatergic Preoptic Neurons Mediate Inflammatory Fever.

JNeurosci
THE JOURNAL OF NEUROSCIENCE

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Fever is a common phenomenon during infection or inflammatory conditions. This stereotypic rise in body temperature (Tb) in response to inflammatory stimuli is a result of autonomic responses triggered by prostaglandin E2 action on EP3 receptors expressed by neurons in the median preoptic nucleus (MnPOEP3R neurons). To investigate the identity of MnPOEP3R neurons, we first used in situ hybridization to show coexpression of EP3R and the VGluT2 transporter in MnPO neurons. Retrograde tracing showed extensive direct projections from MnPOVGluT2 but few from MnPOVgat neurons to a key site for fever production, the raphe pallidus. Ablation of MnPOVGluT2 but not MnPOVgat neurons abolished fever responses but not changes in Tb induced by behavioral stress or thermal challenges.