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Neurodegenerative Disease

R&D Systems 제품과 함께한 Current Reference를 확인해보세요.

IL-17A exacerbates neuroinflammation and neurodegeneration by activating microglia in rodent models of Parkinson's disease

Have shown that helper T (Th)17 cells facilitate dopaminergic neuronal loss in vitro. Herein, we demonstrated that interleukin (IL)-17A, a proinflammatory cytokine produced mainly by Th17 cells, contributed to PD pathogenesis depending on microglia. Mouse and rat models for PD were prepared by intraperitoneal injection of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) or striatal injection of 1-methyl-4-phenylpyridinium (MPP+), respectively. Both in MPTP-treated mice and MPP+-treated rats, blood–brain barrier (BBB) was disrupted and IL-17A level increased in the SN but not in cortex. Effector T (Teff) cells that were adoptively transferred via tail veins infiltrated into the brain of PD mice but not into that of normal mice. The Teff cell transfer aggravated nigrostriatal dopaminergic neurodegeneration, microglial activation and motor impairment. Contrarily, IL-17A deficiency alleviated BBB disruption, dopaminergic neurodegeneration, microglial activation and motor impairment. Anti-IL-17A-neutralizing antibody that was injected into lateral cerebral ventricle in PD rats ameliorated the manifestations mentioned above. IL-17A activated microglia but did not directly affect dopaminergic neuronal survival in vitro. IL-17A exacerbated dopaminergic neuronal loss only in the presence of microglia, and silencing IL-17A receptor gene in microglia abolished the IL-17A effect. IL-17A-treated microglial medium that contained higher concentration of tumor necrosis factor (TNF)-α facilitated dopaminergic neuronal death.

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사용제품 : Recombinant IL-17A

사용용도 : Primary microglial culture & treatment

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Traumatic brain injury induced matrix metalloproteinase2 cleaves CXCL12α (stromal cell derived factor 1α) and causes neurodegeneration

MMP2 has a significant role in neuroinflammation and neurodegeneration by modulating the chemokine CXCL12α (stromal cell derived factor SDF-1α) signaling pathway and the induction of apoptosis. SDF-1α is responsible for cell proliferation and differentiation throughout the nervous system and is also implicated in various neurodegenerative illnesses. We hypothesized that TBI leads to MMP2 activation and cleavage of the N-terminal 4 amino acid residues of CXCL12α with generation of the highly neurotoxic fragment SDF-1(5–67). Using an in vitro stretch-injury model of rat neuronal cultures and the in vivo fluid percussion injury (FPI) model in rats, we found that oxidative stress has a significant role in the activation of MMP2. This is initiated by the induction of free radical generating enzyme NADPH oxidase 1 (NOX1). Induction of NOX1 correlated well with the signatures of oxidative stress marker, 4HNE in the injured neuronal cultures and cerebral cortex of rats. Further, using MMP2 siRNA and pharmacological MMP2 inhibitor, ARP100, we established the neurodegenerative role of MMP2 in cleaving SDF-1α to a neurotoxic fragment SDF-1(5–67). By immunofluorescence, western blotting and TUNEL experiments, we show the cleaved form of SDF leads to apoptotic cell death in neurons. This work identifies a new potential therapeutic target to reduce the complications of brain damage in TBI.

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사용제품 : Rabbit anti-cleaved caspase-3

사용용도 : Immunofluorescence and microscopy, Western blot

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R&D Systems Recombinant Protein

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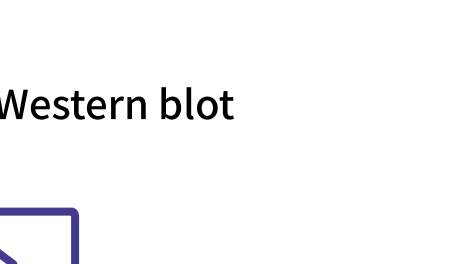

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