Poster | Sensory System

[2P]Somatosensation

Thu. Jul 30, 2020 1:30 PM - 3:30 PM Poster Session

*Videos are available throughout the meeting period.

[2P-085]Microglial depletion under thalamic hemorrhage ameliorates pain-like behavior and suppresses aberrant axonal sprouting

*Shinichiro Hiraga[1,2,3,4,5], Shin-Ichiro Hiraga[1], Takahide Itokazu[1,2,3], Maki Hoshiko[1,2], Hironobu Takaya[1], Mariko Nishibe[1,4], Toshihide Yamashita[1,2,3,5] (1.Dept. of Molecular Neuroscience, Grad. Sch. of Medicine, Osaka Univ., Osaka, Japan, 2.WPI Immunology Frontier Research Center, Osaka Univ., Osaka, Japan, 3.Department of Neuro-Medical Science, Graduate School of Medicine, Osaka Univ., Osaka, Japan, 4.Office of Strategic Innovative Dentistry, Graduate School of Dentistry, Osaka Univ., Osaka, Japan, 5.Graduate School of Frontier Biosciences, Osaka Univ., Osaka, Japan)

Central post-stroke pain (CPSP) often occurs following a hemorrhagic stroke that damages the somatosensory nucleus of the thalamus. Previously, activation of microglia at damaged brain tissues has been implicated to be associated with CPSP in response to the stroke, however, the underlying neuronal mechanism of CPSP is not clear. Here, we examined the microglial function that is specifically towards neuronal network. By employing a focal thalamic hemorrhage model in mice, we observed local microglial activation increased not only at the damaged thalamus but also within the somatosensory cortex. By utilizing pharmacological method to ablate microglia (i.c., the oral administration of selective CSF1 receptor inhibitor PLX3397), we found that the ablation of microglia completely prevented the development of CPSP. To elucidate the neuronal changes, we examined the thalamo-cortical projection by using retrograde neuronal tracer and revealed the occurrence of axonal projections in the somatosensory cortex after stroke. Specifically, compared to intact mice, the stroke group showed a significant increase in the connectivity from the thalamus to the cortical layer IV. We then observed with oral administration of PLX3397 the connectivity from the thalamus and cortex layer IV diminished, suggesting the absence of microglia may have inhibited the stroke-induced projections from the thalamus to the cortex. We are currently investing how such microglial activation directly influences the neuronal changes. The present finding suggests the profound influence of the microglia on the neurons particularly after stroke, in particular in the aspect of neuronal projections.