BDR SEMINAR (Kobe/online hybrid)

Kei Hashimoto

Academic Production, Ochanomizu University

Thursday, April 14, 2022

12:30-13:30

1F Auditorium, DB Building C, Kobe / Broadcast online via Zoom

Zoom meeting URL will be announced on the event day by e-mail.

※This seminar is open only to BDR members.

Glial toxicity in neurodegenerative diseases

Summary

Since brain function is established by the communication between neurons and glial cells, autoimmune dysfunction is a critical cause of pathogenesis in neurodegenerative diseases, however the interaction manner between reactive glial cells and neuronal damage remains unclear. *Grn*$^{-/-}$ mice have provided important insights into disease onset as a mouse model of frontotemporal dementia (FTD). One critical phenotype in *Grn*$^{-/-}$ mice is the age-dependent increase of reactive microglia, supporting a key role of microglia in neurodegenerative diseases. To reveal how *Grn*$^{-/-}$ microglia contributes to the pathogenesis in FTD, multi-omic approaches, including single-nucleus RNA-sequencing, proteomics, and lipidomics were performed using aging cohort of *Grn*$^{+/+}$ and *Grn*$^{-/-}$ brains. In this seminar, I will present the mechanisms of aged microglia-mediated neurotoxicity, which is caused by the excessive secretions of complement and proinflammatory lipids, and promotes the TDP-43 proteinopathy and neuronal cell death in *Grn*$^{-/-}$ brains. Also, I would like to discuss the potential of new insight into the contribution of autoimmune dysfunctions into the dementia onset.

Host: Katsuyuki Shiroguchi

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