The 465 th IMEG Seminar

【Date & Time】 Thu, 6 July, 15:00 - 16:00

【Title】 Primary neurons obtained from rodents and human neurons derived from iPSCs (The basis of neurotransmission and analysis of lysosomal storage diseases)

【Venue】 Conference Room, IMEG and Online (Hybrid)

【Speaker】 Tadahiro Numakawa, PhD.

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【Summary】

As increased blood concentration of glucocorticoid (stress hormone, regulated via the hypothalamic-pituitary-adrenal axis) or downregulation of brain-derived neurotrophic factor (BDNF, essential neurotrophic factor for neuronal survival) have been suggested to be associated with the pathophysiology of mental disorders including depression. Primary neurons prepared from rodent central nervous system (CNS) is powerful tool to investigate the basis of neurotransmission because they grow up so fast, therefore, we investigated negative impact of glucocorticoid exposure on synaptic enhancement by BDNF in cultured CNS neurons (***PNAS***, 2009), although responses of human neurons should be clarified.

We recently reported presynaptic dysfunction in human neurons derived from lysosomal storage disease iPSCs, including GM1 gangliosidosis, Tay-Sachs disease, and Sialidosis (***Neuroscience***, 2019; ***Stem Cell Reports***. 2020; ***Neurobiol Dis***. 2021). Human iPSCs have great potential to elucidate the molecular pathogenesis of brain diseases including neurodegenerative disease such as Alzheimer's disease (AD), in addition to lysosomal storage diseases. Here, we introduce a possible mechanism underlying depression (primary neurons) and deficits in synaptic function in both lysosomal storage disease and AD neurons (iPSC-derived human neurons).

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