

REGULATION OF PHOSPHATIDYLINOSITOL-3-KINASES (PI 3-KINASES) IN ACTIVATED RODENT MICROGLIA

FRIDAY
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3:00PM – 3:30PM

ANATOMY SEMINAR ROOM,
L2, MD10, DEPARTMENT OF
ANATOMY, NUS.

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Abstract:

Microglia are the resident macrophages of the central nervous system (CNS). While their role in neuroinflammation and phagocytosis have been extensively studied, knowledge of their involvement in the formation of memory is still unclear. Recent studies are making it more apparent that microglia do not just have passive housekeeping roles in the brain but actively interact with neuronal synapses and bring about changes that ultimately influence memory. Phosphatidylinositol 3-kinase (PI3K) has been shown to play a significant role in synaptic plasticity in neurons. Its expression in microglia has long been attributed to the microglial inflammatory response however its role in microglia-mediated information processing has yet to be studied. Downstream effectors of PI3K such as Akt, CREB and BDNF have also been found to influence synapses. We hypothesise that microglial PI3K contributes to memory formation and that its function is epigenetically regulated by histone modifications and SUMOylation. Elucidating the exact mechanisms by which PI3K affects memory may broaden our understanding of how microglia are involved in synaptic plasticity. Subsequently, we plan to look at the role of PI3K in models of neurological disorders such as Alzheimer's disease and autism spectrum disorder (ASD).