manitoba neuroscience network

How to disrupt excitotoxic cascades in Alzheimer's disease

SEMINAR & VISITING SPEAKER SERIES

DATE
Friday, March 25, 2022
9:00AM

ZOOM LINK
https://us02web.zoom.us/j/83594461654?pwd=cGR6OW96WTVGSkFXRDE5MGRKaDNXZz09

MEETING ID
835 9446 1654

PASSCODE
771772

SPEAKER
Michael F. Jackson, PhD

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Department of Pharmacology & Therapeutics
Principal Investigator, Kleysen Institute for Advanced Medicine

BIO

Michael Jackson joined the Department of Pharmacology & Therapeutics at the University of Manitoba in 2013. His laboratory is located in the Neurosciences research laboratories in the Kleysen Institute for Advanced Medicine (KIAM). Dr. Jackson obtained his PhD from McGill University in the Department of Pharmacology. Following postdoctoral training at the University of Toronto in the Department of Physiology, he moved to the Robarts Research Institute at Western University in 2008 as a Research Scientist and Adjunct Professor in the Department of Physiology and Pharmacology.

RESEARCH

Accumulation of soluble amyloid beta oligomers (AβOs) is closely linked to cognitive decline in Alzheimer's disease (AD). Our research program is focused on identifying molecular mechanisms through which AβOs disrupt the function and plasticity of glutamate excitatory synapses and thus precipitate cognitive decline. AβOs are known to provoke accumulation of glutamate, associated with excitotoxic stimulation of Ca2+ permeable NMDA-type glutamate receptors (NMDARs). Notably, partial block of NMDARs is responsible for the beneficial (though modest) response in patients treated with memantine/Ebixa, a "weak" NMDAR blocker. As more potent NMDAR blockers are associated with intolerable side effects, our research has sought to identify downstream effectors of NMDAR-initiated excitotoxic cascades. The research presented will highlight our recent findings which links the stimulation of NMDARs, expressed in microglia, to synaptotoxic inflammatory responses triggered by AβOs. Thus, our findings provide a unifying mechanism linking elevated glutamate in AD to chronic neuroinflammation.

For more information:

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