Amyloid-beta regulation of hippocampal synaptic plasticity

https://neurodegenerationresearch.eu/survey/amyloid-beta-regulation-of-hippocampal-synaptic-plasticity/ Principal Investigators

Zhang, Xia

Institution

University of Ottawa

Contact information of lead PI Country

Canada

Title of project or programme

Amyloid-beta regulation of hippocampal synaptic plasticity

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CIHR

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5

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Research Abstract

Deposition of a protein called amyloid beta (Abeta) in the brain has long been believed to be a central pathological feature of Alzheimer's disease (AD). However, a large number of recent studies have clearly shown a link between early changes of AD-related memory impairment and soluble Abeta-elicited impairment of long-term neural transmission. There are two major types of brain cells: neurons and glial cells. All the scientific evidence accumulated during the past century has shown key roles of neurons in brain functioning with glial cells providing nutrition, support, etc. Our recent article accepted for publication in 2012 March 3rd issue of the top-elite journal Cell provides the first evidence that astrocyte type of glial cells regulates neuronal transmission and subsequently working memory – a supporting actor becomes the leading

actor! As a natural extension of this pioneer research, we have conducted a series of pilot experiments, which, together with other indirect lines of evidence, lead us to hypothesize that soluble Abeta stimulates brain cells to produce endocannabinoids (chemicals existing in our brain that act as cannabis or marijuana), which activate astroglial cells to regulate neuronal transmission, resulting in impairment of working memory. We will employ a variety of cutting-edge technology to critically examine this hypothesis in rats and mice.

Further information available at:

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