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大学院セミナー開催通知

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講　座　名 (責任者名)(内線)	生命医科学講座・分子創薬科学講座 形態制御解析学分野（第一解剖）・ゲノム創薬学分野 責任者名(森望／岩田修永)　内線（7019 / 2435)
演　　題	Neuron-glia crosstalk under pathological condition of Alzheimer's disease
講　師　等	**Prof. Inhee Mook-Jung** Department of Biochemistry and Biomedical Sciences, Seoul National University College of Medicine, Seoul, Korea
ｾﾐﾅｰの概要	Alzheimer's disease (AD) is the most devastating neurodegenerative disease in modern society because of the insurmountable difficulties in early diagnosis and lack of therapeutic treatments. Pathological hallmarks of AD are extracellularly deposited amyloid plaques and intracellular neurofibrillary tangles in the brain. Even though amyloid beta (Aβ) has been regarded as one of the main causative substances and intensive research has been focused on Aβ in AD, there are still difficulties to explain the complexities of AD pathogenesis. Activated microglia and reactive astrocytes are commonly found in and around the senile plaques. Astrocytes respond to neuronal activity through the release of gliotransmitters such as glutamate, ATP and other proteins. How gliotransmitters regulate neuronal activity, however, is not well defined and even controversial. Also, astrocytes secrete several proteins to the synapse, which modulate synaptic function directly or indirectly to the neurons. In the present study, we examined the effect of ATP, thrombospondin-1 (TSP-1) and insulin degrading enzyme (IDE), those are secreted from astrocytes by Aβ. We found that exogenous ATP protects against Aβ42-mediated reduction in synaptic molecules, such as NMDA receptor 2A, PSD-9ATP5 and synaptophysin, through purinergic receptor P2X in primary hippocampal neurons. As astrocyte-secreted proteins, thrombospondin-1 (TSP-1) and IDE were examined *in vitro*, in AD animal model and human AD samples. The release of TSP-1 from astrocytes was decreased by Aβ42 *in vitro*, and the reduced level of TSP-1 was observed in brains of AD animal models. In addition, IDE levels were reduced in the cerebrospinal fluid (CSF) of patients with AD and in AD model mice. We found IDE is secreted from astrocytes through an autophagy-based unconventional secretory pathway in AD conditions. Taken together, neuron-released Aβ stimulates astrocytes and astrocyte-releasing factors including ATP, TSP-1 and IDE can affect neuronal functions in AD condition. These neuron-glia crosstalk and their releasing mechanisms will be discussed more in the seminar.
開 催 日 時	平成２７年１０月８日　（木） １７：３０～１８：３０
場　　所	薬学部　第一講義室（１階）
備　　考	先端医療科学特論（基礎編）の単位認定に該当

■先端医療科学特論(基礎編)　□先端医療科学特論(臨床編)
□先端新興感染症病態制御学特論　□先端放射線医療科学特論