

A cytokine derived material to recover Alzheimer's disease memory impairment via microglia activation: electrophysiology and behavioral approaches

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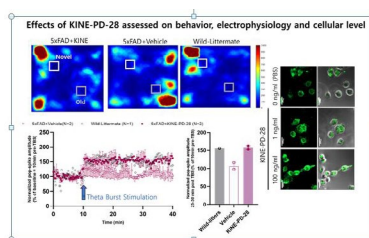
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Alzheimer's disease (AD) is a progressive neurodegenerating disease caused by abnormal aggregation of amyloid beta ($A\beta$) that results in synaptic damage and leading to cognitive impairment. Recently, several drugs reducing the accumulation of $A\beta$ had been developed and been approved for clinical use. Here, we developed a novel cytokine derived small peptide-based substance, KINE-PD-28, that remove $A\beta$ and simultaneously relieve AD symptom.

To test the effect of the drug on cognitive deficit, we performed behavioral experiments. A human APP overexpressing mouse, 5xFAD, or their wild-type littermate was injected with saline or KINE-PD-28 and tested with modified Y-maze, object location recognition test and passive avoidance (PA). After behavioral experiments their hippocampal synaptic network was assessed with in-vivo LTP electrophysiology method. To determine the mode of action, we analyzed the change of $A\beta$ after treating KINE-PD-28 on bv2 cells, a microglia-derived cell line.

Our experiments showed that Kine-101 injected mice performed better compared with saline injected group. Time spent in novel arm or exploring time on novel object was significantly higher in KINE-PD-28 injected mice. Similarly in PA test, KINE-PD-28 injected mice showed better retention of memory. In electrophysiology, 25-30min after LTP induction, while 5xFAD+saline group showed low LTP response, KINE-PD-28 injected mice recovered the LTP similar with the control group. We observed reduction of $A\beta$ in the hippocampus and the cortex through histological analysis. Furthermore, we detected dose dependent internalization of $A\beta$ in the microglia after treating KINE-PD-28.

Altogether, behavioral and electrophysiological studies suggest that KINE-PD-28 relieves the symptoms of AD and recovers memory impairment. Molecular experiments suggest that the recovery is due to the enhancement of microglial $A\beta$ phagocytosis.



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Biography

Jun Il Kang has expertise in neuroscience electrophysiology. Using his knowledge and experience he studied the circuitry of learning and memory combined with advanced technique such as optogenetics or applying mathematical analysis methods. He has also great passion on understanding the network of brain cognition. Now he is working in a pharmaceutical company R&D team to develop cures against brain disease such as Alzheimer's or fronto-temporal dementia. He focuses on remedy that not only relieve the symptoms but also delay the pathology. Kine science, the company that he is currently working for, develops ultra-small peptide innovative medicines based on functional immunomics against multiple chronic diseases.

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