IL-33 ameliorates Alzheimer's-like pathology and cognitive decline

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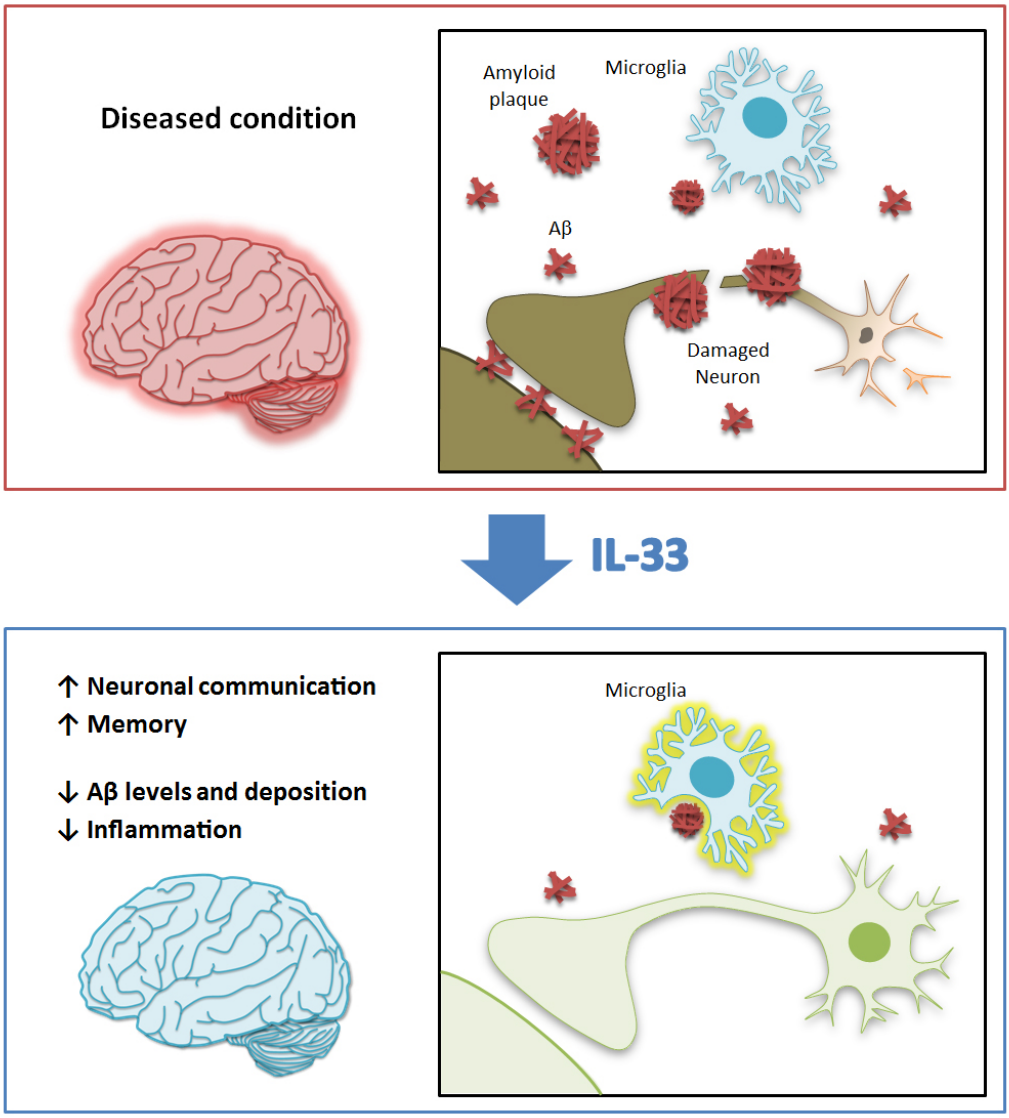

IL-33 in Alzheimer's disease. Credit: Division of Life Science, HKUST

Alzheimer's disease (AD) is a devastating condition with no known effective treatment. The disease is characterized by memory loss as well as impaired locomotor ability, reasoning, and judgment. Emerging evidence suggests that the innate immune response plays a major role in the pathogenesis of AD.

While the mechanisms underlying the onset and progression of AD remain unclear, scientists from the Hong Kong University of Science and Technology (HKUST) recently conducted a study on the potential therapeutic role of interleukin-33 (IL-33) in AD, where they injected the protein into transgenic mouse models of AD. The injection of IL-33 rescues contextual memory deficits and reduces the deposition of β-amyloid peptide (Aβ) in the transgenic mouse model, suggesting that IL-33 can be developed as a new therapeutic intervention for AD.

The findings were published in the journal *PNAS*.

"There is no effective therapy for AD, in part because of our limited knowledge of its underlying pathophysiological mechanisms," said Prof Nancy Ip, Dean of Science, Director of the State Key Laboratory of Molecular Neuroscience and The Morningside Professor of Life Science at HKUST, who directed the research effort. "Nonetheless, targeting the innate immune system has been considered a promising strategy for developing effective therapeutics for AD. The present study demonstrates that peripheral IL-33 injection in AD mouse models alleviates AD-like pathology by enhancing microglial phagocytosis and degradation of Aβ."

"We believe that IL-33 is a critical factor in maintaining a healthy

brain," Prof Ip said. "Disturbances in this signal mechanism, owing to genetic disposition or environmental influence, may contribute to the onset of AD. The next step will be to translate the findings from the mouse study into clinical treatments for humans."

The research was the result of a collaborative effort among scientists from HKUST, the University of Glasgow, and Zhejiang University.

More information: Amy K. Y. Fu et al, IL-33 ameliorates Alzheimer's disease-like pathology and cognitive decline, *Proceedings of the National Academy of Sciences* (2016). DOI: 10.1073/pnas.1604032113

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