Deletion of Caspase-6 using CRISPR improves the memory function in models of Alzheimer disease (Ramot)

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THE NEED

Currently, there is no cure for Alzheimer'. Although caspase-6 was demonstrate to play a role in the pathology there is no clinical available caspase-6 inhibitors.
Caspases, a family of cysteine proteases, are major mediators of apoptosis and inflammation. Increasing evidence has shown that caspase-6 is highly involved in axon degeneration and neurodegenerative diseases, such as Huntington's disease (HD) and Alzheimer's disease (AD). Therefore, caspase-6 is considered to be an up-stream modulator of AD pathogenesis, and as a result, a viable therapeutic target for the treatment of AD.

TECHNOLOGY

by injecting AAV viruses containing Caspase 6 CRISPR into the brain hippocampus of mouse model of AD we were able to cause a reduction in the levels of caspase-6 gene in this area . Moreover, caspase-6 inhibition improved memory function in mouse models of AD and reduced the amyloid aggregations.

POTENTIAL APPLICATION

Our novel approach to reduce caspase-6 activity in the brain, by targeted caspase 6 deletion might be used as a treatment for AD and other neuronal diseases were aggregation of proteins and toxic protein fragments play a major role. Caspase-6 inhibition show improvement also in Huntington disease and inflammatory pain.

ADVANTAGES

Caspase-6 is express in the brain only in aged people or in patients. Therefore, inhibition of caspase-6 should to be safe. By removing the caspase 6 gene this would allow a one time intervention

PATENTS

Provisional patent filed in 2017

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