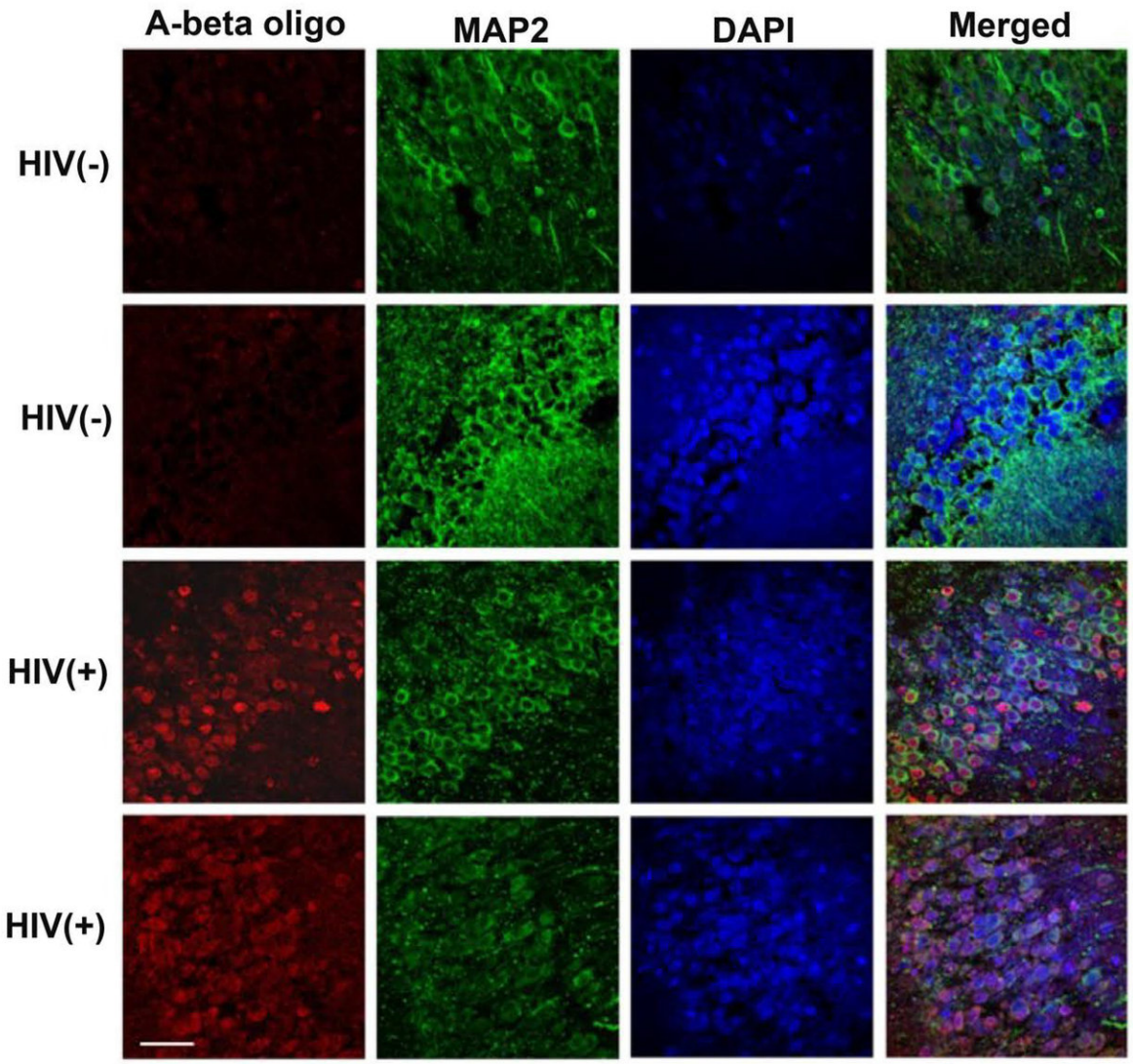
Overlapping mechanisms in HIV cognitive disorders and Alzheimer's disease

April 9 2018



Aβ oligomers are elevated in the brains of HIV(+) cases. Paraffin-embedded

tissue sections from hippocampus of HIV(-) and HIV(+) individuals were prepared for immunofluorescent analysis and visualized by laser confocal microscopy. Representative images are shown from hippocampal sections triple-labeled for Aβ oligomers (red), MAP2 (green), and nuclei (blue). Red and green colocalization appears yellow. Credit: Stern et al., *JNeurosci* (2018)

A protein involved in Alzheimer's disease (AD) may be a promising target for treating neurological disorders in human immunodeficiency virus (HIV) patients, suggests a study published in *JNeurosci* of rat neurons and brain tissue from deceased humans. The research shows that the two conditions may damage neurons in similar ways.

Although HIV-associated neurological disorders (HAND) and AD have symptoms in common, whether they also share underlying mechanisms of disease progression is controversial because HAND patients do not exhibit the amyloid plaques that are characteristic of AD. To address this question, Kelly Jordan-Sciutto and colleagues investigated the role of a well-known AD protein—β-site amyloid precursor protein cleaving enzyme 1 (BACE1)—in HAND.

The researchers found elevated levels of BACE1 and Aβ oligomers—the compound thought to be responsible for neuronal damage in AD—in postmortem brain tissue of HIV-positive humans. Treating rat neurons with HIV-infected white blood cells from healthy humans revealed similar mechanisms of neurotoxicity.

More information: BACE1 mediates HIV-associated and excitotoxic neuronal damage through an APP-dependent mechanism, *JNeurosci* (2018). DOI: 10.1523/JNEUROSCI.1280-17.2018

Provided by Society for Neuroscience