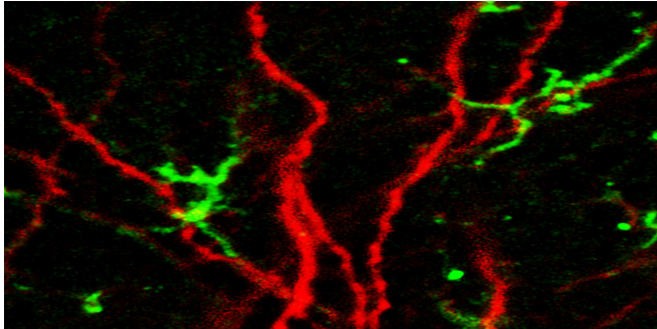# Alzheimer's risk gene impairs development of new neurons in mice

30 July 2018

ApoE-expressing astrocytes (green) interacting with tdTomato-expressing dendrites (red) from adult newborn hippocampal neurons. Credit: Tensaouti et al., *eNeuro* (2018)

Scientists have taken a step closer to understanding how the strongest known genetic risk factor for Alzheimer's disease (AD) contributes to memory impairment. Reporting their findings in *eNeuro*, the researchers demonstrate a critical role of the risk gene in the proper development of adult-born neurons in the hippocampus.

Apolipoprotein E (ApoE) is among the genes that regulate ongoing generation of neurons in the dentate gyrus of the [hippocampus](link). A variant of this gene called ApoE4—present in 10 to 20 percent of the human population—is also associated with the development of late-onset AD.

Investigating the effects of ApoE on adult neurogenesis, Tzong-Shiue Yu, Steven Kernie, and colleagues found reduced complexity of the dendrites of adult-born neurons in mice with genetically silenced ApoE compared to unaltered mice, as well as in those expressing ApoE4 compared to ApoE3-expressing mice—ApoE3, the most common variant found in humans, is not associated with disease risk.

These findings suggest a potential explanation for the increased risk of neuropsychiatric diseases involving the hippocampus, including AD, among ApoE4 carriers.

  More information: ApoE regulates the development of adult newborn hippocampal neurons, *eNeuro*, [DOI: 10.1523/ENEURO.0155-18.2018](link)

Provided by Society for Neuroscience

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