The 5× familial Alzheimer's disease (5×FAD) mice show alleviated mechanical allodynia which can be regained by genetic activation of anterior cingulate cortex (ACC) excitatory neurons. Furthermore, the lower peak neuronal excitation, delayed response initiation, as well as the dendritic spine reduction of ACC pyramidal neurons in 5×FAD mice, can be mimicked by Rac1 or actin polymerization inhibitor in Wild-type (WT) mice. These findings indicate that abnormal pain sensitivity in Alzheimer's disease modeling mice is closely related to the variation of neuronal activity and dendritic spine loss in ACC pyramidal neurons, suggesting the crucial role of dendritic spine density in pain processing [1)]

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1)

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