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Dendritic Impulse Propagation is Altered in Neocortical Pyramidal Neurons of Tg2576 Mice, an Animal Model of Alzheimer's Disease

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Alzheimer's disease (AD) is one of the most frequent neurological degenerative disorders. During the course of the illness accumulations of amyloid-beta peptide appear in the brain tissue as insoluble extracellular plaques, and neurons and synapses show signs of degeneration. We investigated the dendritic impulse propagation in layer II/III pyramidal neurons of the somatosensory cortex in human amyloid precursor protein over expressing Tg2576 transgenic mice and compared the signal propagation to that of the control, healthy neurons from wild type mice.
Earlier studies detected morphological changes in these pyramidal neurons of Tg2576 mice. However, physiological measurements with somatic electrodes could not differentiate between the mutant and control principal neurons based on their passive membrane properties and action potential generation.
We used morphologically detailed passive segmental cable models of these cells (n=58) within the NEURON (Duke University, USA) simulation environment. Current was injected to various dendritic points of mutant and healthy neurons to simulate local activity of synapses and current and voltage transfers to the soma were investigated as a function of path distance of injection site from the soma. Spatial distributions of the dendritic surface area and dendritic length were also studied.
First in literature, we investigated the dendritic impulse propagation systematically in pyramidal neurons from Tg2576 mice. We found that current transfer is more effective in the apical dendrites of mutant neurons and concluded that this higher effectivity may contribute to the hyperexcitability of mutant neurons, a phenomenon also found in humans with AD.

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