Amyloid Beta Leads to Synaptic Depression

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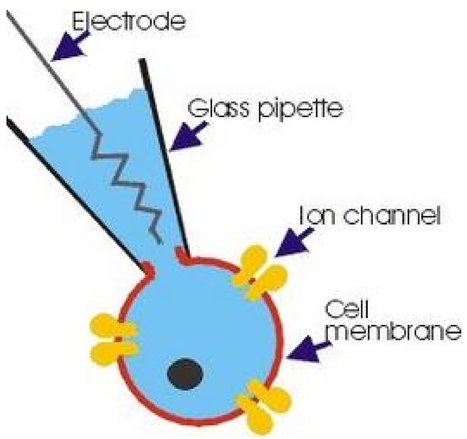
Abstract

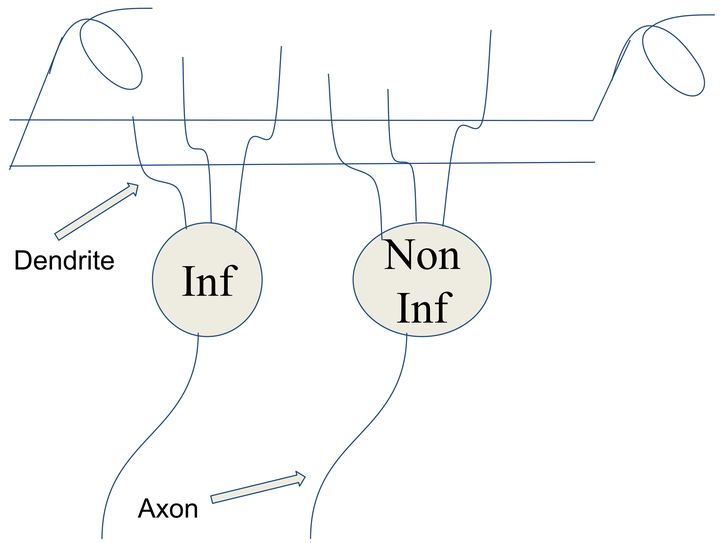
Alzheimer's disease (AD) is one of the most common neurodegenerative diseases in people ages 65 and older. The toxic brain peptide, amyloid-beta (Aß), is believed to play a central role in the development of AD. Aß is thought to weaken the synapse, a structure that that is responsible for learning, memory by allowing neurons to pass an electrical signal to one another. The impact of Aß on the synapse is not fully understood. In this report we used patch clamping, a method used to measure electrical currents in a cell, to show that Aß overexpression causes a weakening of synaptic transmission. Our hypothesis is that Aß causes a decrease in the expression of receptors at the surface and thus decreases electrical currents. We will do patch clamp recordings from a control cell and a cell over expressing Aß to test this idea.

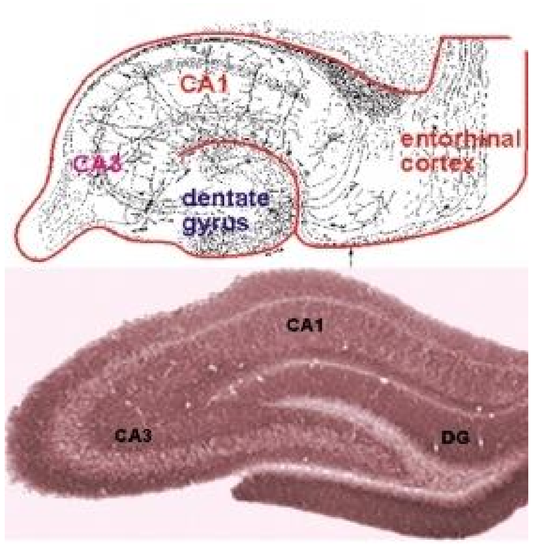
The Patch Clamp Method

In a patch-clamp recording, a glass pipette containing an electrolyte solution forms a tight seal onto the cell membrane and thus isolates a membrane patch electrically. Currents fluxing through the channels in this patch tend to flow into the pipette and can be recorded by an electrode that is connected to an amplifier. The current measured is proportional to the summation of the conductance of each individual channel.

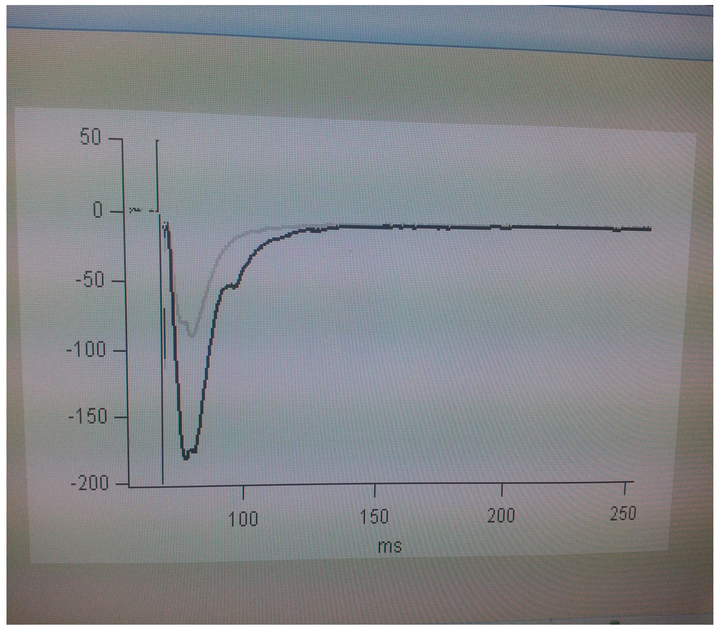


Whole Cell Recording





Data



Conclusions

We found that the electrical synaptic currents were decreased in the presence of amyloid-beta due to a decrease of receptors on the surface.

Future Research

Researchers are now focusing on immunization strategies and production blockers of amyloid beta. Current studiesare aiming to find ways to block plaque formation or to reduce Aß load.

References

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- LaFerla, Frank M., KiN. Green, and Salvatore Oddo. "Intracellular Amyloid-bold Beta in Alzheimer's Disease." *Www.nature.com*. Nature Reviews Neuroscience, 1 July 2007. Web. 16 July 2015.
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