Emergence of cognitive impairment in an Alzheimer’s Disease mouse model: the role of NMDA receptor currents.

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Research Abstract

Current evidence suggests that memory loss in early Alzheimer’s disease (AD) starts when amyloid-beta (Ab) and tau proteins interfere with the capacity of neurons to modify the strength of their connections (synaptic plasticity), an indispensable brain function for storing new memories. However, the events that set off the disease process are not well understood and available treatments are only modeslty effective. To understand the earliest mechanisms of AD,

we will use a mouse model in which overproduction of A? can be switched on and off. The student will use a combination of behavioural and physiological experiments to link adult-onset memory impairment with synaptic function. The focus will be on glutamate receptors of the NMDA subtype (NRs), which are essential for many forms of learning and memory and known targets of A?. Furthermore, the student will investigate whether (early and late) NR changes and memory impairment are reversible, by switching off A? production. The results of this work will highlight the timing and relevance of NR modifications to the onset of cognitive impairment. A better understanding of their role in memory loss will allow us to target them more effectively in the search for drugs that modify AD symptoms or progression.

Further information available at:

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