

# Non-amyloid-related hippocampal network dysfunction as an early biomarker of Alzheimer's disease

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**Name of Fellow**

**Institution**

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**Contact information of fellow**

**Country**

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Non-amyloid-related hippocampal network dysfunction as an early biomarker of Alzheimer's disease

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**Research Abstract**

Alzheimer's disease (AD) is the most common form of dementia. Generally, it is diagnosed in

people over 65 years of age, although the less prevalent early-onset Alzheimer's can occur much earlier. As of September 2010, this number is reported to be 35.6 million worldwide. AD develops for an indeterminate period of time before becoming fully apparent, and it can progress undiagnosed for years. Currently used treatments offer a small symptomatic benefit; no treatments to delay or halt the progression of the disease are as yet available. Therefore, an early marker of AD might be a useful new tool to improve life quality of patients.

It is well documented that AD patients and animals models of AD exhibit massive reorganization of hippocampal and cortical networks, initiated by an early imbalance between excitation and inhibition. This alteration of hippocampal networks might lead to early perturbations of hippocampal oscillatory activity. Brain oscillations in the theta (3-12Hz) and gamma frequency bands (30-250Hz) are crucial for supporting normal cognitive and executive functioning.

Accordingly, increasing evidence suggests that oscillatory activity in theta and gamma range is altered in AD. Despite the genetic and cell biological evidence that supports the amyloid hypothesis, it is becoming clear that AD etiology is complex and that A $\beta$  alone is unable to account for all aspects of AD, i.e. amyloid deposits might be present in cognitively normal individuals, whereas some AD patients show no amyloid plaques. These results strongly suggest that non-amyloid factors may also be responsible for hippocampal networks dysfunction. The present proposal opens an original and innovative avenue of research for understanding the cellular mechanisms responsible for early cognitive abnormalities in AD by focusing on non-amyloid related alterations in hippocampal oscillations. This research program will serve to develop electrophysiological tools for the early diagnostic of AD.

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