

Detecting early dementia in mice: The contribution of amyloid vs tau

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Country

United Kingdom

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Research Abstract

Alzheimer's disease (AD) leads to a progressive cognitive decline; endstage neuropathological hallmarks comprise severe brain atrophy amidst accumulation of beta-amyloid and tau species. However, early degenerative events are extremely subtle and difficult to detect, thus hampering diagnosis and corresponding therapeutic success.

Our recently developed innovative knock-in mouse lines (PLB) express subtle levels of human amyloid precursor protein (APP), and/or tau and/or presenilin transgenes. The genetic design is such that mono-, bi- and tri-genic AD lines are genetically directly comparable, devoid of genetic artefacts and disease progression is slow and traceable. In comparing these lines, we seek to

reveal the earliest onset of failing or altered cognitive performance and determine the underlying electrophysiological and pathological mechanisms. This is achievable via quantitative electroencephalogram (qEEG) utilising a novel wireless EEG recording device (Nat) and combined with interactive data analyses tools from which we predict improved sensitivity and reliability.

Both, behavioural task selection and EEG electrode placement is tailored to reveal the function of hippocampus and prefrontal/cingulate cortex, for which pathological changes in PLB mice have been confirmed. Tasks comprise spatial/reversal learning in the Barnes maze as an episodic-like task and social transmission of food preference probing semantic memory. Behaviour in identified learning strategies will be linked to biochemical changes and physiological EEG correlates analysed for spectral power density and phase synchrony, shifts in autoregressive spectra and Granger-based connectivity readouts. This approach will not only detect early physiological and cognitive changes, but also provide mechanistic insights regarding underlying malfunctions and pathological damage.

Further information available at:

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