SLEEP MICROSTRUCTURE AND EARLY ALZHEIMER'S DISEASE NEUROPATHOLOGY

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Text: Alterations in sleep are hallmarks of the ageing process and emerge as risk factors for Alzheimer’s disease (AD). While the fine-tuned coalescence of sleep microstructure elements may influence age-related cognitive trajectories, its association with AD-related processes is not fully established. Here, we investigated whether key elements of sleep microstructure are associated with early amyloid-beta (Aβ) brain burden, hallmark of AD neuropathology, and cognition in 100 late-midlife healthy individuals (50 - 70y; 68 women).

We first found that spontaneous arousal during sleep are heterogenous and differently associated with Aβ and cognition. While the density of arousals associated with changes in sleep stages were associated with more early deposit of Aβ, the density arousals not interrupting sleep continuity were associated with less Aβ burden and better cognitive performance to attentional tasks and with change in memory performance at 2 years. We further found the young-like co-occurrence of spindles and slow-depolarisation slow waves was associated to lower early burden of Aβ and was predictive of memory decline at 2-year follow-up. In contrast the density of spindles and slow waves as well as other more macroscopic metrics of sleep were not associated with early deposit of Aβ.

These findings unravel early links between sleep, AD-related processes and cognition and support that the spontaneous arousals and the altered coupling of sleep microstructure elements that are key to its mnesic functions contributes to poorer brain and cognitive trajectories in ageing.

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