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Science

The world's leading cause of dementia, Alzheimer's disease (AD) is a progressive, neurodegenerative disorder affecting an increasing aged population. Apart from progressive cognitive decline, sleep and circadian rhythm disturbances frequently occur in AD, severely impacting the quality of life of AD patients and their caregivers, thus forming a major cause of institutionalisation. The underlying mechanisms of sleep disturbance in AD are not elucidated, and consequently there are no therapies directed specifically towards ameliorating AD-mediated perturbations of sleep/wake control mechanisms. Growing evidence supports the interrelationship between sleep-wake cycle disruption, AD-related cognitive deterioration and the associated neuropathological progression of the disease.

Sleep disturbances could exacerbate a fundamental process leading to neurodegeneration, and optimization of sleep time could potentially inhibit aggregation of toxic proteins and slow the progression of AD. Thus, pointing out the need for further research, addressing the effective management and efficient treatment of circadian rhythm disturbances in AD.

Sleep studies in demented patients are difficult, and large scale screening of potential medications for sleep disturbances in AD is not evident with human subjects. Therefore we aim to address this AD-related behaviour in preclinical settings.

The well-validated transgenic APP23 mouse model of AD carrying the Swedish double mutation displays a disturbed activity pattern, resembling sundowning behaviour in AD. Our EEG/EMG/activity unit allows us to provide an elaborate circadian phenotyping of sleep and activity rhythms by simultaneous recording of cage activity and the electrophysiological aspects of sleep.

This approach allows continuous monitoring of the development and progression of circadian rhythm and sleep disturbances in adult APP23 mice over time under quasi-natural and undisturbed conditions and may provide a useful preclinical bioassay which we will use to evaluate the temporal dynamics and relative efficacies of currently used and novel pharmacological and non-pharmacological therapeutics. This might open new insights to future treatment choices concerning circadian rhythm alterations and attribute to a better management of AD.