# Free Communications: Abstract 3

## Sleep architecture changes in the APP23 mouse model manifest at the onset of cognitive deficits

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**Objectives**. Alzheimer's disease (AD) patients typically experience increased sleep fragmentation, excessive daytime sleepiness and nighttime insomnia. Here, we sought to investigate the link between sleep architecture, cognition and amyloid pathology in the APP23 amyloidosis mouse model for AD.

**Methods**. By means of polysomnographic recordings, the sleep-wake cycle of freely-moving APP23 and wild-type (WT) littermates of 3, 6 and 12 months of age was examined. In addition, ambulatory cage activity was assessed by interruption of infrared beams surrounding the home cage. To assess visuo-spatial learning and memory a hidden-platform Morris-type Water Maze (MWM) experiment was performed.

**Results**. We found that sleep architecture is only slightly altered at early stages of pathology, but significantly deteriorates from 12 months of age, when amyloid plaques become diffusely present. APP23 mice of 12 months old had quantitative reductions of NREM and REM sleep during the dark phase compared to WT littermates, which was confirmed by increased ambulatory cage activity. No quantitative differences in sleep parameters were observed during the light phase. However, during this light phase, the sleep pattern of APP23 mice was more fragmented from 6 months of age, the point at which also cognitive abilities started to be affected in the MWM. Sleep time positively correlated with MWM performance.

**Conclusions**. To conclude, our results indicate that sleep architectural changes arise around the time the first amyloid plaques start to form and cognitive deterioration becomes apparent. These changes start subtle, but gradually worsen with age, adequately mimicking the clinical condition.

Sleep, Cognition, EEG, APP23, Morris Water Maze