quantify the level of i) intrusions and ii) instability. Finally, we used these measures as predictors of sleep group and sleep quality metrics.

Results: INS and SSM showed significantly higher intrusions than GS. INS was characterized by wake intrusions during sleep and SSM by the opposite. Wakefulness was significantly more unstable for SSM than for the rest. Sleep intrusions positively correlated with power in lower frequencies (<8 Hz), while wake intrusions with power in higher frequencies (>10 Hz). Finally, we obtained a classification accuracy of 0.8, overcoming any previous attempts of detecting SSM using PSG data.

Conclusion: As recently proposed, SSM could be more related to a mismeasurement of sleep quality rather than to a misperception. In this line, we developed a method tailored to detect intrusions in sleep dynamics which unveiled the specific signatures of SSM. The implications of this work extend from clinical biomarkers to a deeper comprehension of sleep quality.

Conflict of Interest: No.

O59

Oral Session 10: Sleep Quality and Neurological Health: Biomarkers and Clinical Implications

Age-related changes in the association between REM sleep and the polygenic risk for Parkinson's disease

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Introduction: REM sleep behaviour disorder (RBD) almost unescapably leads to the diagnosis of Parkinson's disease (PD) within about 10 y, such that PD is one of the rare diseases for which sleep alteration is a true marker of disease outcome. How the association between sleep and PD emerges over the healthy lifetime trajectory is not known. To address this issue, we examined association of REM sleep metrics with the polygenic risk score (PRS) for PD in healthy young and older individuals.

Method: In this prospective observational study, in-lab EEG recordings of habitual sleep were conducted on 346 young (18–31 y; 22.1 y ± 2.7) and 83 older individuals (50–70 y; 59.4 y ± 5.3). Automatic procedures were used to score sleep stages, detect artefacts and arousals and extra EEG REM theta energy (4–8 Hz overnight cumulated power). We also extracted DNA from saliva/blood for PRS determination. Summary statistics of large PD GWAS was used to compute PRS using SBayesR approach implemented through GCTB software. Generalized Additive Model for Location, Scale and Shape (GAMLSS) was used for statistical analyses seeking associations between sleep parameters and PRS, while controlling for age, sex, BMI and total sleep time.

Results: In the younger cohort, the analysis revealed positive association of REM theta energy ($p = 0.019$; β: 0.01) and REM percentage ($p = 0.002$; β:0.004) with PD PRS. Negative association was observed between REM latency and PRS for PD ($p = 0.024$; β:−0.01). In contrast the analysis of the older cohort, revealed negative association of REM theta energy ($p = 0.034$; β: −0.02) with PRS.

Conclusion: Our findings show that REM sleep is associated with the polygenic risk for developing PD in healthy individuals aged <31 y. A higher risk for PD is associated with higher REM sleep intensity during early adulthood while it is associated with lower REM sleep intensity after 50 y. This reveals a switch in the association between younger, presumably free of alpha-synuclein inclusions, and older healthy individuals in which low levels of these inclusions may be present. These findings may contribute to unravelling the core association between PD and sleep and to the identification of novel intervention targets to prevent or delay PD.

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Oral Session 10: Sleep Quality and Neurological Health: Biomarkers and Clinical Implications

Slow wave/spindle coupling better predicts amyloid-beta dynamics than slow wave activity

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Introduction: Slow wave activity (SWA) facilitates amyloid-beta (Aβ) clearance from the brain, suggesting a neuroprotective role against the cascade of neurodegeneration. In the aging brain, the disruption of slow wave (SW)/spindle coupling has been linked to memory decline and medial frontal atrophy. Whether SW/spindle coupling is associated with Aβ dynamics remains uncertain, as studies in healthy older adults have yielded mixed results, and clinical populations have not been investigated yet.

Method: Here, 47 sleep-pathology-free older adults (mean age: 70.47 (0.67), range: 59–80) on a spectrum of cognitive functioning (Montreal Cognitive assessment Score (MoCA) range: 20–30) spent an adaptation night as well as an experimental night in the sleep laboratory. Blood samples collected post-experimental night were analyzed for abundance of Aβ peptides 1–42 and 1–40, and the 1–42/1–40-ratio, a key biomarker for neurodegeneration. We used both forward and backward stepwise multiple regression analysis and considered SWA, spindle power, SW/spindle-coupling strength and -hierarchy, age and MoCA Score as predictors for plasma Aβ levels.

Results: We found that a higher 1–42/1–40-ratio (indicative of lower Aβ-burden) is best explained by increased SW/spindle coupling strength as well as a SW/spindle hierarchy resembling a younger