Slow Wave Sleep in Alcohol Use Disorder and the Brain Correlates

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Disruptions in slow wave sleep (SWS) are commonly observed in patients with alcohol use disorder (AUD) and associated with higher risk for relapse and poor executive function. Cross-sectional evidence suggests that SWS reduction in AUD persists long after abstinence. However, the brain mechanism underlying disrupted SWS in AUD is still unclear. Furthermore, individual variations in SWS recovery which can only be examined with longitudinal design haven't been closely investigated. Here, we sought to 1) reveal brain mechanisms account for SWS impairment in AUD compared to healthy controls (HC); 2) determine brain mechanisms associated with individual differences in SWS during recent detoxification as well as with SWS recovery after 3-week inpatient detoxification with longitudinal data.

Thirty-six subjects who had moderate or severe AUD and a minimum of 5-year heavy drinking history were included in the study. Among them, thirty patients completed a 3-week inpatient treatment as well as all sleep and MRI measures on both week 1 and week 3. SWS was measured with ambulatory sleep EEG. For brain correlates, grey matter (GM) structure and resting state functional connectivity (RSFC) were examined.

AUD patients showed reduced SWS compared to HC and total lifetime drinking was negatively associated with SWS in AUD. GM not only mediated the effect of AUD but also the effect of total lifetime drinking on SWS in patients. Although at group level there was no significant recovery of SWS after 3-week detoxification, great variations were observed among patients. Patients who showed greater SWS increases after detoxification had enhanced midline default mode network (DMN) RSFC indicating greater brain recovery as lower DMN RSFC is commonly seen in patients with substance use disorder and is associated with impaired self-awareness. In contrast, GM was associated with SWS both on week 1 and week 3, but not with SWS changes suggesting that GM greatly accounts for individual differences but not within-subject changes in SWS.

Our findings suggest that GM atrophy induced by chronic alcohol use greatly accounts for SWS reduction in AUD patients. At individual level, SWS increases are associated with improved brain function in AUD reflected by enhanced midline DMN RSFC. Therefore, SWS might be a good biomarker for monitoring AUD recovery. Enhancing SWS might promote brain recovery among patients who showed limited SWS improvement.

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