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HIPPOCAMPAL SLEEP SPINDLE DYNAMICS DURING REM SLEEP AND THEIR DISTINCT UNDERLYING PARVALBUMIN AND SYNAPTIC PROTEINS EXPRESSION IN THE RETICULO-THALAMIC NUCLEUS OF THE PARKINSONIAN RATS

POSTER SESSION 02 - SECTION: PARKINSON'S DISEASE AND MOVEMENT DISORDERS

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We investigated the alterations of reticulo-thalamic (RT) GABAergic parvalbumin (PV+) interneurons and synaptic re-organization underlying the altered hippocampal high voltage sleep spindle (HVS) dynamics during REM sleep in the rat models of Parkinson's disease (PD). Adult male Wistar rats were implanted for 6h sleep recording during light phase in four experimental groups: control (implanted controls), PD cholinopathy (bilateral lesion of *the nucleus pedunculopontinus tegmentalis*−PPT), hemiparkinsonism (unilateral lesion of *the nucleus substantiae nigrae pars compacta*−SNpc) and hemiparkinsonism with PD cholinopathy (unilateral SNpc/bilateral PPT lesion). Following 14 days of the surgical procedure we differentiated the Wake/NREM/REM 10s epochs, and the HVSs detection and extraction was done automatically (4.1–10 Hz band pass filter, 1s minimum duration) and visually validated. Hippocampal HVS dynamics were analyzed during 1h of NREM/REM sleep. Alterations of the PV+ interneurons and synaptic re-organization within the RT were determined by the parvalbumin, MAP2 and PSD-95 immunostaining. REM sleep is a predisposing state for the HVSs induction in all experimental models of PD neuropathology. Whereas the PD cholinopathy induced the prolongation and higher density of hippocampal HVSs, the hemiparkinsonism with PD cholinopathy increased the hippocampal HVSs intrinsic frequency during REM sleep. In contrast to the unaltered PV+ interneurons/partially enhanced MAP2/suppressed PSD-95 expression during PD cholinopathy, we evidenced the PV+ interneurons reduction/enhanced MAP2/no change of PSD-95 expression in the RT during hemiparkinsonism with PD cholinopathy. Distinct PV+ interneurons alteration and inhibition/excitation balance in the RT could be the underlying mechanisms of HVS generation/alteration during REM sleep in the parkinsonian rats.

Pubmed:

34445628: Radovanovic L, Petrovic J, Saponjic J
Hippocampal and Reticulo-Thalamic Parvalbumin Interneurons and Synaptic Re-Organization during Sleep Disorders in the Rat Models of Parkinson's Disease Neuropathology.
We investigated the alterations of hippocampal and reticulo-thalamic (RT) GABAergic parvalbumin (PV) interneurons and their synaptic re-organizations underlying the prodromal local sleep disorders in the distinct rat models of Parkinson's disease (PD). We demonstrated for the first time that REM sleep is a predisposing state for the high-voltage sleep spindles (HVS) induction in all experimental models of PD, particularly during hippocampal REM sleep in the hemiparkinsonian models. There were the opposite underlying alterations of the hippocampal and RT GABAergic PV+ interneurons along with the distinct MAP2 and PSD-95 expressions. Whereas the PD cholinopathy enhanced the number of PV+ interneurons and suppressed the MAP2/PSD-95 expression, the hemiparkinsonism with PD cholinopathy reduced the number of PV+ interneurons and enhanced the MAP2/PSD-95 expression in the hippocampus. Whereas the PD cholinopathy did not alter PV+ interneurons but partially enhanced MAP2 and suppressed PSD-95 expression remotely in the RT, the hemiparkinsonism with PD cholinopathy reduced the PV+ interneurons, enhanced MAP2, and did not change PSD-95 expression remotely in the RT. Our study demonstrates for the first time an important regulatory role of the hippocampal and RT GABAergic PV+ interneurons and the synaptic protein dynamic alterations in the distinct rat models of PD neuropathology.
Int J Mol Sci, 2021; 22
33038348: Petrovic J, Radovanovic L, Saponjic J
Prodromal local sleep disorders in a rat model of Parkinson's disease cholinopathy, hemiparkinsonism and hemiparkinsonism with cholinopathy.
We investigated the prodromal alterations of local sleep, particularly the motor cortical and hippocampal sleep, along with spontaneous locomotor activity in the rat models of Parkinson's disease (PD). We performed our experiments in adult, male