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Werner Felix-Martin

Euro Academy Pößneck, Germany

Is there a prophylactic medication in REM sleep disturbances, which hint at synucleinopathies?

Introduction: On the DGSM congress 2014, rapid eye movement sleep (REM) behavioural disorders were reported, while the SPECT examination showed an asymmetric description of the dopamine transporter systems. These sleep disturbances predict synucleinopathies such as Parkinson's disease. The present study aims at the question, whether there is a prophylactic mediation for this neurodegenerative disease, will be answered by a neural network in the extrapyramidal system.

Material & Methods: The neural network can be described as follows: D1 and D2 dopaminergic neurons in the substantia nigra activate dopaminergic neurons in the caudate nucleus. D1 dopaminergic neurons weakly activate dynorphin neurons, which weakly inhibit via kappa receptors substance P neurons. The latter neurons activate weakly via NK1 receptors GABAergic neurons in the globus pallidus internus. In the caudate nucleus D2 dopaminergic neurons weakly activate GABAergic neurons in the globus pallidus externus, which inhibit glutaminergic neurons in the subthalamic nucleus. The latter neurons strongly inhibit via NMDA receptors D2 dopaminergic neurons in the substantia nigra and GABAergic neurons in the globus pallidus internus. In these nucleus, GABAergic neurons weakly inhibit glutaminergic neurons, which activate other glutaminergic neurons in the cortex. These neurons can activate D1 and D2 dopaminergic neuros in the caudate nucleus. In the globus pallidus internus GABAergic neurons weakly inhibit M4 muscarinic cholinergic, 5-HT2A serotonergic and NTS1 neurotensin neurons in the putamen. The latter neurons transmit a strong postsynaptic excitatory impulse to glutaminergic neurons, which inhibit via NMDA receptors D2 dopaminergic neurons in the putamen. The D2 dopaminergic neurons in the putamen are connected to other dopaminergic neurons in the caudate nucleus.

Results: Since in Parkinson's disease, apart from dopamine and actylcholine alterations, a GABA deficiency and a glutamate surplus can be found, it might be possible to weaken the neurotransmitter imbalance by a drug, which has at the same time a GABAA agonistic and an NMDA antagonistic effect. Through the GABAA agonistic effect the acetylcholine, serotonin and neurotensin surplus could be reduced. The NMDA antagonistic effect could increase the dopamine levels through a reduced presynaptic inhibition.

Conclusion: It is of important to observe patients with REM sleep behavioural disorders and to minimize the risk for synucleinpathies with an appropriate medication.

Biography

Werner Felix-Martin studied Human Medicine at the University of Bonn. He has been working as a Medical Teacher for geriatric nurses, occupational therapists and assistants of the medical doctor at the Euro Academy in Pößneck since 1999. He has been doing scientific work at the Institute of Neurosciences of Castilla and León in Salamanca in Spain since 2002. With Prof. Rafael Coveñas, he assisted at over 30 national and six international congresses of neurology and published over 20 reviews about neural networks in neurological and psychiatric diseases. Since 2014, he belonged to the Editorial Board of the Journal of Cytology & Histology.

felixm-werner@versanet.de