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Symptoms and Therapeutic Options of the Anti-NMDA Receptor Encephalitis According To a Neural Network

Felix-Martin Werner[1,2*] **and Rafael Covenas**[2]

[1]*Higher Vocational School of Elderly Care and Occupational Therapy, Euro Academy, Pobneck, Thuringia, 07381, Germany*

[2]*Laboratory of Neuroanatomy of the Peptidergic Systems (Lab. 14), Institute of Neurosciences of Castilla y León (INCYL), University of Salamanca, Salamanca, Castilla-León, 37007, Spain*

***Corresponding author:** Felix-Martin Werner, University of Salamanca, Instituto de Neurociencias de Castilla y León (INCYL), Laboratorio de Neuroanatomía de los Sistemas Peptidérgicos (Lab. 14), c/ Pintor Fernando Gallego, 137007-Salamanca, Spain, Tel: +34923294400, E-mail: felixm-werner@versanet.de

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Introduction

The anti-NMDA receptor encephalitis concerns above all young women and girls and can occur as the paraneoplastic syndrome of a teratome or without a primary cause. A neural network is developed in order to explain the symptoms of the disease and to derive the possible therapies.

Material and Methods

In the midbrain glutaminergic neurons weakly inhibit serotonergic neurons via NMDA receptors, so that serotonergic neurons have a high activity through 5-HT1A receptors. Activated by the serotonergic neurons, GABAergic neurons strongly inhibit via GABAA receptors noradrenergic neurons, which transmit a weak impulse to glutaminergic neurons via alpha1 receptors. These alterations hint the changes in awaremess.

In the mesolimbic system glutaminergic neurons strongly inhibit via NMDA receptors serotonergic neurons, which have a high activity via 5-HT2A receptors. Since dopaminergic and serotonergic neurons activate each other through D2 and 5-HT1A receptors in the A10 cell group, dopamine hyperactivity via D2 receptors occurs as well. This explains the psychotic symptoms and also mania which happens in some cases [1].

In the extrapyramidal system glutaminergic neurons in the putamen weakly inhibit via NMDA receptors dopaminergic neurons weakly activate muscarinic cholinergic neurons. The dopaminergic neurons in the caudate nucleus enhance via D2 receptors the GABAergic inhibition in the external globus pallidus of glutaminergic neurons in the nucleus subthalamicus. Since the glutaminergic neurons weakly inhibit dopaminergic neurons in the substantia nigra via NMDA receptors, dopamine hyperactivity is enhanced. The glutaminergic neurons in the nucleus subthalamicus transmit a weak activating impulse to GABAergic neurons in the internal globus palidus, which inhibit muscarinic cholinergic neurons in the putamen. These alterations lead to catatonic movement disturbances [1].

In the hippocampus a blockade of the NMDA receptors leads to dopamine hyperactivity and serotonin and GABA hypoactivity. This neurotransmitter alteration can cause epileptic seizures and a status epilepticus (Figure 1) [2].

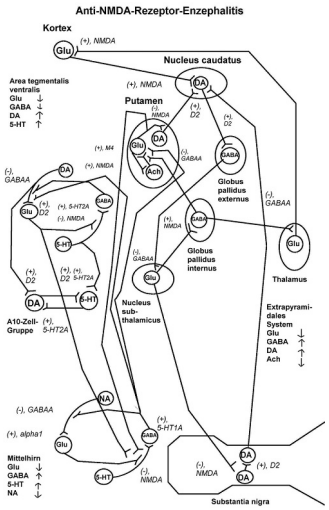

Figure 1: Neuronal pathways, classical neurotransmitters and neuropeptides involved in Parkinson's disease in the extrapyramidal system. 5-HT: serotonin; Ach: acetylcholine; DA: dopamine; GABA: gamma-aminobutyric acid; Glu: glutamate; NA: noradrenaline. The following subreceptors are indicated: alpha1: alpha1 receptor, a subreceptor of the noradrenergic receptor; GABAA: GABAA receptor, a subreceptor of the GABA receptor; 5-HT1A: 5-HT1A receptor, a subreceptor of the serotonergic receptor; 5-HT2A: 5-HT2A receptor, a subreceptor of the serotoninergic receptor; D2: D2 receptor, a subreceptor of the dopaminergic receptor; kappa: kappa receptor: a subreceptor of the opioid receptor; M4: M4 receptor: a subreceptor of the muscarinic cholinergic receptor; NMDA: NMDA (N-methyl-D-aspartate) receptor, a subreceptor of the ionotropic glutaminergic receptor; A plus mark indicates a postsynaptic excitatory impulse; a minus mark indicates a presynaptic inhibitory impulse.

Results

The following therapies can be performed in this disease:

- Plasmapheresis in order to normalize the neurotransmitter alterations.
- Immunotherapy. Werner [2] made experiments about immunotherapy. According to these results, a strong immunological

reaction is possible, if the antigen/antibody ratio is equivalent. After an administration of antibodies, anti-antibodies are formed. It remains to be investigated in experiments when after the first administration of antibodies and in which concentration this ratio between the primary antibody and the secondary antibody is achieved so that a strong immunological reaction occurs.

- Benzodiazepines and antipsychotic drugs in order to reduce dopamine and serotonin hyperactivity. Among the antipsychotic drugs quetiapine and clozapine with a strong 5-HT2A antagonistic effect should be preferred, because a blockade of 5-HT2A receptors counteracts glutamate deficiency [1].

Conclusion

This neural network, which should be examined in depth, enables the explanation of the symptoms and the finding of therapies of the disease.

References

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