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Risk genes in schizophrenia and their application in choosing the approriate antipsychotic drug

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Schizophrenia is a chronic mental illness with a genetic etiology in 80% of the cases. In the last years, about 260 risk genes in schizophrenia have been discovered and correlations between risk genes and the therapeutic efficacy of an antipsychotic treatment/pharmacotherapy resistance have been reported. In schizophrenia, important risk genes, such as catechol-O-methyl transferase (COMT), monoamine oxidase (MAO A/B), glutamic acid decarboxylase 67 (GAD 67), dysbindin-1 and neuregulin-1 will be mentioned. To describe the function of these risk genes, neural networks in the ventral tegmental area, hippocampus and prefrontal cortex will also be developed. An association between the SNPs of some risk genes and the efficacy of a specific antipsychotic treatment is reported: SNPs such as rs165599 (COMT gene), rs1801028 (D2 receptor gene) and rsSer9 Gly (D3 receptor gene) are associated with a better antipsychotic treatment efficacy (e.g., treatment of negative schizophrenic symptoms with risperidone). The rs4680 SNP (COMT and D2 receptor genes) is associated with pharmacotherapy resistance. The function of risk genes is described: COMT and MAO A/B genes, with reduced activity in the corresponding enzymes, are associated with a decreased dopamine degradation and hence dopamine hyperactivity occurred via D2 receptors. The GAD 67 risk gene is linked with GABAergic dysfunction and consequently GABAergic neurons weakly presynaptically inhibit D2 dopaminergic neurons. The D-amino acid oxidase activator (DAOA) risk gene is connected with glutamatergic dysfunction via NMDA receptors. Glutamatergic neurons might exert a weak presynaptic inhibition upon 5- HT2A serotonergic neurons located in the ventral tegmental area and hippocampus. Neural networks in the latter two regions and in the prefrontal cortex are updated. It is important to examine the SNPs of the risk genes involved in schizophrenia to establish a correlation between these SNPs and the efficacy of a determined antipsychotic drug. Thus, schizophrenic patients with a good response to a determined antipsychotic treatment and patients with resistance to this treatment might be well differentiated.

Biography

Felix-Martin Werner studied human medicine at the university of Bonn. He has been working as a medical teacher in the formation of geriatric and general nurses, occupational therapists and assistents 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 (INCYL) in Salamanca (Spain) since 2002. With Prof. Rafael Coveñas, he assisted at over 30 national and 12 international congresses of neurology and published over 60 reviews about neural networks in neurological and psychiatric diseases. In 2017, they published the e-book: Classical neurotransmitters and neuropeptides involved in chizoaffective disorder: focus on prophylactic medication. Since 2014, Dr. Werner has belonged to the editorial board of the Journal of Cytology & Histology.

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