Dopamine Dysfunction in Schizophrenia and its Significance

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About the Study

Current Antipsychotic Drugs (APDs) operate on D2 receptors, and preclinical studies show that D2 antagonist injection consistently downregulates spontaneously activated DA neurons by causing overexcitation-induced inactivation of firing (depolarization block). Animal models of schizophrenia based on Moderate Acute Malnutrition injection during pregnancy yield offspring with adult characteristics compatible with schizophrenia, such as ventral hippocampus hyperactivity and DA (dopamine) neuron overactivity. The MAM model demonstrates that APDs behave differently in a hyperdopamineregic system than in a normal one, including rapid onset of depolarization block in response to acute D2 antagonist administration and downregulation of DA neuron population activity in response to acute and repeated D2 partial agonist administration, which are not observed in normal rats.

On the basis of the hypothesis that glutamatergic dysfunction is key to schizophrenia pathophysiology, several target drugs have been designed. Despite showing promise in preclinical studies, none of the innovative medications made it through clinical trials. However, preclinical research is often conducted in normal, drug-free rats, whereas models with disease-relevant pathology and Dep

might mirror schizophrenia's developmental history. The model demonstrated that a vHPC lesion in the early postnatal period causes the adult emergence of behavioural impairments and DA dysfunction associated with schizophrenia. Developed by the NVHL model's findings and research establishing the role of immune activation during pregnancy as a risk factor for schizophrenia, another significant neurodevelopmental model of schizophrenia, the Methylazoxymethanol Acetate (MAM) model, was established.