TÍTULO
SCHIZOPHRENIA: DOPAMINERGIC THEORY, ANTIPSYCHOTICS AND COGNITIVE DEFICIT

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RESUMO
Introduction: Schizophrenia is a psychosis considered multifactorial, involving genetic and external factors. Dopamine receptors (D1-D5) are central to the understanding of dopaminergic theory. The distribution of these receptors in various brain areas and their excitatory or inhibitory action on the synthesis of cAMP correlates brain areas where there is underactive and dopaminergic hyperactivity associated with schizophrenia. D1 receptors are excitatory and its reduction in the prefrontal cortex is possibly associated with deficit cognitive. No agreement on the pattern of cognitive impairment, but the precocity of these changes and their long-term stabilization is well documented. Objectives: address some gaps dopaminergic theory: the unsatisfactory results of antipsychotics on negative symptoms and antipsychotics; the relationship dopamine-receptors in the frontal lobes and changes in cognition.

Method: Researching data in Medline/PubMed and Lilacs utilizing the terms: schizophrenia, dopamine theory, cognition schizophrenia, antipsychotics, dopamine receptors, cognition deficit in schizophrenia.

Discussion: the dopaminergic theory has been the most widely accepted, pointing to the brain of the neurotransmitter dopamine dysregulation and its receptors. Uma hypofunction dopamine in the prefrontal cortex would be responsible for negative symptoms, deficit cognitive and a primary event in schizophrenia, leading to a hyperfunction dopaminergic secondary in the striatum, which would be the cause of positive symptoms. As cognitive changes have high prevalence, are present even in the premorbid. Studies “post mortem” phase are facing considerable reduction of D1 receptors in the prefrontal cortex of schizophrenic patients cognitive deficit. Conclusion: It is believed that atypical antipsychotics do not cause extrapyramidal symptoms because they have a connection to the D2 receptor less strongly than the D2-dopamine, so there is no shortage in the extrapyramidal system which would be the cause of Parkinsonian syndrome with the use of typical antipsychotics. A hypothesis for the unsatisfactory results of antipsychotics in negative symptoms and cognitive deficit is the excitatory action of D1 receptors in the prefrontal cortex. A dopaminergic hypofunction cortex in this area has been correlated with negative symptoms and cognitive deficit. An ideal antipsychotic should, in addition, inhibit the mesolimbic pathway, leverage the mesocortical pathway, which supplies the prefrontal cortex, this may be possible through differências between D1 and D2 receptors.

References


