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Glutamate and other neurotransmitters in control of motor behaviour

Introduction

It is almost generally accepted that a hyperactivity of the corticostriatal glutamatergic projection – together with a hypofunction of the striatal dopamine system – play an important role in movement disorders, particularly in Parkinson's disease.

The aim of this section is to present (1) new vistas on direct or indirect interactions of different neurotransmitters with glutamatergic and dopaminergic systems in the control of motor behaviour and (2) to find out some possible novel targets for drugs which could be useful in the future in the treatment of Parkinson's disease.

According to this, the symposium has been divided into two partly overlaping parts: (1) a group of papers mainly presenting new vistas on physiology and pharmacology of the dopaminergic-glutamatergic equilibrium in the brain and (2) a group of papers dealing mainly with interactions of different neurotransmitters with the glutamate system.

The first paper gives – as a kind of introduction to the section – a brief description of the phylogenetic development, physiology, function and pharmacology of the whole system within which glutamate mediated cortical signals mix their effects in the basal ganglia with the reinforcing/rewarding impulses of dopamine neurons resulting finally in information conveyed to the thalamus via two (direct loop) or three (indirect loop) GABAergic neurons. Particular attention is paid to the reinforcement of favourable behaviour by dopamine and the role of NMDA receptors in the phenomenon of sensitization.

In the following paper the role of drugs acting on different binding sites of the NMDA receptors in models of Parkinson's disease (mainly in models of parkinsonian muscle rigidity) are examined. An interesting analysis of functional differences between direct and indirect strionigral loops for the pathophysiology and therapy of Parkinson's disease is given. At the end of this part of the section, the role of glutamate receptors in degeneration of dopamine neurons which is at the origin of Parkinson s disease is analysed. Interesting similarities as well as differences between neuronal degenerations seen in Parkinson's disease and Alzheimer's disease are discussed.

The second group of papers is devoted to the examination of interactions between cholecystokinin and glutamate, adenosine and glutamate as well as of the histamine and glutamate receptors. These studies are particularly interesting, as they seem to offer new possibilities for the future therapy of Parkinson's disease.

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