Abstract:

Basal ganglia disorders such as Parkinson's disease, dystonia, and Huntington's disease are characterized by a dysregulation of the basal ganglia neuromodulators (dopamine, acetylcholine, and others), which impacts cortico-striatal transmission. Basal ganglia disorders are often associated with an imbalance between the midbrain dopaminergic and striatal cholinergic systems. In contrast to the extensive research and literature on the consequences of a malfunction of midbrain dopaminergic signaling on the plasticity of the cortico-striatal synapse, very little is known about the role of striatal cholinergic interneurons in normal and pathological control of cortico-striatal transmission. In this review, we address the functional role of striatal cholinergic interneurons, also known as tonically active neurons and attempt to understand how the alteration of their functional properties in basal ganglia disorders leads to abnormal cortico-striatal synaptic plasticity. Specifically, we suggest that striatal cholinergic interneurons provide a permissive signal, which enables long-term changes in the efficacy of the cortico-striatal synapse. We further discuss how modifications in the striatal cholinergic activity pattern alter or prohibit plastic changes of the cortico-striatal synapse. Long-term cortico-striatal synaptic plasticity is the cellular substrate of procedural learning and adaptive control behavior. Hence, abnormal changes in this plasticity may underlie the inability of patients with basal ganglia disorders to adjust their behavior to situational demands. Normalization of the cholinergic modulation of cortico-striatal synaptic plasticity may be considered as a critical feature in future treatments of basal ganglia disorders.

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