

Determining the role of striatal cell types, regions and compartments in the generation of Parkinson's akinesia and dyskinesia.

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Research Abstract

The striatum is attributed a crucial role in generating parkinsonian and dyskinetic motor features. Parkinson's disease (PD) has been related to an overactivity of the striatal spiny projection neurons of the "indirect pathway" (iSPN) over the "direct pathway" spiny projections

neurons (dSPN). On the contrary, the phenomenology of L-DOPA-induced dyskinesia (LID) has been extensively associated to a predominant role of dSPN over iSPN. However, this notion has not been possible to prove experimentally and the spatiotemporal interplay and specific contributions of both subpopulations in the generation of PD and LID have not been unraveled to date. Moreover, the role of SPNs subtypes, regions and compartments to the generation of these movement disorders is presently unknown. Using new recording/imaging technologies combined with chemogenetic approaches we aim to identify patterns of striatal SPN activities that code for akinetic and dyskinetic movement disorders. We hypothesize that the following disturbances in information processing underlie akinetic and dyskinetic movement disorders: (i) an altered pattern and degraded topographical selectivity as well as an abnormal temporal interplay in the ensemble activity of d/iSPN; (ii) a different SPN cell-type contribution to akinetic and dyskinetic movement disorders; (iii) a striatal regional and compartmental specificity of d/iSPN activity in PD and LID.

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