Redundant dopaminergic activity may enable compensatory axonal sprouting in Parkinson disease.

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Abstract:

Neurodegenerative diseases become clinically apparent only after a substantial population of neurons is lost. This raises the possibility of compensatory mechanisms in the early phase of these diseases. The importance of understanding these mechanisms cannot be underestimated because it may guide future disease-modifying strategies. Because the anatomy and physiology of the nigrostriatal dopaminergic pathways have been well described, the study of Parkinson disease can offer insight into these early compensatory mechanisms. Collateral axonal sprouting of dopaminergic terminals into the denervated striatum is the most studied compensatory mechanism in animal (almost exclusively rodent) models of Parkinson disease and is correlated with behavioral recovery after partial lesions. This sprouting, however, does not respect the normal anatomy of the original nigrostriatal pathways and leads to aberrant neuronal networks. We suggest here that the unique physiologic property of the dopaminergic innervation of the striatum, namely redundancy of information encoding, is crucial to the efficacy of compensatory axonal sprouting in the presence of aberrant anatomical connections. Redundant information encoding results from the similarity of representation of salient and rewarding events by many dopaminergic neurons, from the wide axonal field of a single dopaminergic neuron in the striatum, and from the nonspecific spatial effect of dopamine on striatal neurons (volume conductance). Finally, we discuss the relevance of these findings in animal models to human patients with Parkinson disease.

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