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We use this collection and it's working

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Abstract

In Parkinson's disease (PD), motor dysfunctions only become apparent after extensive loss of DA innervation. This resilience has been hypothesized to be due to the ability of many motor behaviors to be sustained through a diffuse basal tone of DA; but experimental evidence for this is limited. Here we show that conditional deletion of the calcium sensor synaptotagmin-1 (Syt1) in DA neurons (Syt1 cKO^{DA} mice) abrogates most activity-dependent axonal DA release in the striatum and mesencephalon, leaving somatodendritic (STD) DA release intact. Strikingly, Syt1 cKO^{DA} mice showed intact performance in multiple unconditioned DA-dependent motor tasks and even in a task evaluating conditioned motivation for food. Considering that basal extracellular DA levels in the striatum were unchanged, our findings suggest that activity-dependent DA release is dispensable for such tasks and that they can be sustained by a basal tone of extracellular DA. Taken together, our findings reveal the striking resilience of DA-dependent motor functions in the context of a near-abolition of phasic DA release, shedding new light on why extensive loss of DA innervation is required to reveal motor dysfunctions in PD.

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Protocol



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Primary Culture of Mouse Mesencephalic Neurons

VERSION 1

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