DYSTONIA

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Editorial: Dystonia and cerebellum

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Editorial on the Special Issue
Dystonia and cerebellum

This special issue examines the growing evidence suggesting that dystonia arises from dysfunction distributed across interconnected motor networks, including the cerebellum, thalamus, basal ganglia, and cerebral cortex. Contributions span genetic models, cellular physiology, systems-level imaging, human electrophysiology, and emerging therapeutic concepts. Together, these provide a broad and integrated view of the importance and complexity of cerebellar involvement in dystonia. Cerebellar abnormalities may contribute directly to pathological movement in some contexts, reflect broader network dysfunction in others, and influence motor and non-motor features of the disorder, highlighting potential entry points for therapeutic development.

Although a substantial part of this Special issue focuses on dissecting mechanistic cerebellar contributions to dystonia using genetic mouse models, the human studies provide critical insights into patient symptoms that can guide future questions in animal studies. Adury et al. compared global *Dyt1* mutations with mutations restricted to cortical and hippocampal neurons, showing that the global mutation produces more widespread reductions in functional connectivity across sensorimotor networks, while the regionally restricted mutation generates a distinct pattern of cerebellar, striatal, and brainstem connectivity changes. These findings reinforce the view of dystonia as a network disorder whose expression depends on the distribution of molecular dysfunction. Complementing their study, Xing et al. showed that Purkinje cell-specific *Dyt1* mutations do not produce significant changes in spontaneous firing and they only produce subtle alterations in intrinsic physiology, suggesting that the more pronounced Purkinje cell abnormalities observed in global models may not be strictly cell-autonomous. In another study, Xing et al. identified sex-dependent alterations in Purkinje cell firing in *Sgce* knockout mice, a model of DYT11 myoclonus-dystonia. The more pronounced electrophysiological abnormalities in female mice paralleled an approximately fourfold increase in myoclonus compared with male knockout mice, although the causal relationship between these phenotypes remains to be established. Liu et al. further examined cerebellar torsinA dysfunction using an acute, adult-onset, Cre-mediated manipulation. Reduced torsinA expression in the cerebellum decreased Purkinje cell spontaneous firing and intrinsic excitability and produced overt dystonia in the majority of animals. The

emergence of severe spinning in a subset of mice raises important questions about broader network involvement.

Several contributions extend these mechanistic observations toward systems-level and clinical questions. Jackson et al. synthesized evidence for Purkinje cell and cerebellar nuclei dysfunction across dystonia subtypes and highlighted impaired calcium regulation as a potential point of convergence for future investigation. Mulcahey et al. demonstrated that motor priming alters low-frequency cerebellar resting-state dynamics differently in individuals with writer's cramp and healthy participants, raising the possibility that the timing and behavioral context of neuromodulation could influence the engagement of disease-relevant networks. Seyyed Mousavi et al. provided rare direct electrophysiological recordings from a pediatric patient with secondary dystonia. Task-aligned activity across basal ganglia and thalamic sites, including the ventral intermediate nucleus (VIM) and centrolateral nucleus (CL), supports the involvement of distributed cerebellothalamic and basal ganglia circuits in movement timing and may help guide future studies of physiology-informed DBS. Kim et al. broadened the clinical framework by reviewing cerebellar contributions to motor and non-motor features of dystonia, particularly sleep and cognition, and emphasized the need for therapies that address overall quality of life rather than motor symptoms alone. Finally, Iacono offered a unique perspective, linking developmental movements, adaptation to gravity, and cerebellar–basal ganglia interactions, ultimately establishing new hypotheses about how environmental demands shape motor network organization.

Collectively, the studies in this special issue position the cerebellum as an important component of the distributed networks underlying dystonia, while also emphasizing that its contribution is unlikely to be uniform across disease subtypes. Findings from genetic models show that Purkinje cell abnormalities may be direct, circuit-dependent, or modified by sex and developmental timing. Human imaging and electrophysiology further demonstrate that cerebellar function must be understood in relation to interconnected basal ganglia, thalamic, and cortical systems. Defining when cerebellar dysfunction initiates dystonia, when it amplifies pathology elsewhere in the network, and when it can be therapeutically engaged will be essential for translating these insights into more precise and effective interventions.

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