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Commentary: Cerebellar atrophy and its contribution to cognition in frontotemporal dementias

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A Commentary on

Cerebellar atrophy and its contribution to cognition in frontotemporal dementias
by Chen, Y., Kumfor, F., Landin-Romero, R., Irish, M., Hodges, J. R., and Piguet, O. (2018). Ann. Neurol. 84, 98–109. doi: 10.1002/ana.25271

THE EMBODIED LITTLE BRAIN: FROM NEUROCOGNITION TO NEURODEGENERATION

Chen et al. (2018) provide unprecedented evidence of syndrome-specific changes in cerebellar gray matter integrity (mainly in lobules VI, Crus I and Crus II) across three frontotemporal dementia (FTD) subtypes, alongside specific associations with attentional, visuospatial, mnesic, and language-motor deficits. Moreover, results survived covariation with each group's distinctive atrophy pattern. These outcomes illuminate the critical role of the cerebellum in non-motor processes, while highlighting the relevance of distributed network approaches to cognitive (dys)function.

Although the cerebellum has been implicated in higher-order domains (Roca et al., 2013; García et al., 2017; Sokolov et al., 2017), including executive functions, language, interoception, and social cognition, these results may prove surprising to many clinical neuroscientists. Indeed, the cerebellum remains notably underexplored within neurocognitive assessments of dementia, where it is still largely conceived as a specifically motoric region and is thus often excluded from imaging analyses seeking to map brain-behavior associations. Similarly, except for research on ataxia, systematic assessments of regional, and network-level alterations involving the cerebellum are wanting in the field. This counterproductive neglect, we believe, stems from a dissociation between dementia studies and current neurocognitive theories (Ibáñez and García, 2018).

Fertile ground could be gained by anchoring neurodegeneration research on the embodied cognition approach, which has revealed multidimensional links between action-related circuits and higher-order functions. The cerebellum, as a core hub in these cortical-subcortical networks, would play an important, enactive role in several cognitive processes. While lesion and agenesia studies suggest that this role may not be causal, cerebellar circuits have been directly implicated in embodied domains (Koziol et al., 2012; Birba et al., 2017; García et al., 2017; Cervetto et al., 2018). Beyond the field's traditional focus on canonical atrophy patterns and selected cognitive skills, emerging evidence suggests that diffuse neurocognitive dysfunctions are partially overlapped across dementias. The profuse interconnectedness, functional richness, and transdiagnostic vulnerability

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of the cerebellum render it a key target to examine embodied cognitive deficits in FTD and other conditions.

Accordingly, embodied theories of the cerebellum could become critical tools to foster relevant translational developments. First, they underscore the need to systematically report cerebellar involvement in diverse neurocognitive deficits. Also, they provide a profitable platform to track intercognitive phenomena—enactive synergies among varied psychobiological (dys)functions—from a network-based perspective (Koziol et al., 2012; Ibanez et al., 2014, 2018; García and Ibáñez, 2016; Birba et al., 2017; García et al., 2017; Cervetto et al., 2018; Ibáñez and García, 2018). Moreover, they promote a reinterpretation of symptoms from an action-grounded neurocognitive rationale (Krakauer et al., 2017). These milestones could have direct clinical implications, as the lack of proper theoretical frameworks can lead to neglecting, downplaying, or delaying the report of cerebellar disturbances across pathologies. We call for novel studies integrating embodied, intercognitive, network-based conceptualizations of the cerebellum to foster translational breakthroughs in dementia research.

AUTHOR CONTRIBUTIONS

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