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Spike-time-dependent plasticity induction reveals dissociable ventral and supplementary premotor-motor pathways to automatic imitation.

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Introduction: Humans tend to spontaneously imitate others'behavior, even when detrimental to the task at hand. The Action Observation Network (AON) is consistently recruited during imitative tasks. However, whether automatic imitation is mediated by cortico-cortical projections from AON regions to the primary motor cortex (M1) remains speculative. Similarly, the potentially dissociable role of AON-to-M1 pathways involving the ventral premotor cortex (PMv) or supplementary motor area (SMA) in automatic imitation is unclear.
Methods: Here, we used cortico-cortical paired associative stimulation (ccPAS) to enhance or hinder effective connectivity in PMv-to-M1 and SMA-to-M1 pathways via Hebbian spike-time-dependent plasticity (STDP) to test their functional relevance to automatic and voluntary motor imitation.
Results: ccPAS affected behavior under competition between task rules and prepotent visuomotor associations underpinning automatic imitation. Critically, we found dissociable effects of manipulating the strength of the two pathways. While strengthening PMv-to-M1 projections enhanced automatic imitation, weakening them hindered it. On the other hand, strengthening SMA-to-M1 projections reduced automatic imitation but also reduced interference from task-irrelevant cues during voluntary imitation.
Discussion: Our study demonstrates that driving Hebbian STDP in AON-to-M1 projections induces opposite effects on automatic imitation that depend on the targeted pathway. Our results provide unprecedented causal evidence of the functional role of PMv-to-M1 projections for automatic imitation, seemingly involved in spontaneously mirroring observed actions and facilitating the tendency to imitate them. Moreover, our findings support the notion that SMA exerts an opposite gating function, controlling M1 to prevent overt motor behavior when inadequate to the context.

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No

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