The Neural Basis of Fatigability: Too Little Inhibition is Detrimental for Motor Performance

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Central mechanisms of motor fatigability are not completely understood. Here we investigated repetitive low-force movements which cause a gradual reduction in movement speed (or 'motor slowing'), likely reflecting a central component of fatigability in healthy adults. We show that motor slowing is associated with a gradual increase of net excitability in the motor network and, specifically, in primary motor cortex (M1), which results from overall disinhibition. Importantly, we link performance decrements to a breakdown of surround inhibition in M1, which is associated with high coactivation of antagonistic muscle groups. We further show that motor slowing can be overcome in the presence of reward, but this effect seems to be caused by invigoration of the motor activity rather than by reducing muscular coactivation. We propose that the release of inhibition in M1 is an important mechanism underpinning motor fatigability which can, however, be counteracted by increasing motor effort. An imbalance of these mechanisms might contribute to pathological fatigue as frequently observed in patients with brain disorders.