Computational modeling of basal ganglia impairments in developmental stuttering

A. Daliri, L. Max, & F. Towhidkhah

Recent data from psychophysical, neuroimaging, and pharmacological studies suggest intriguing new hypotheses regarding the neuromotor mechanisms underlying persistent developmental stuttering. Specifically, stuttering individuals differ from nonstuttering individuals in several aspects of sensorimotor learning and control, and some differences are not limited to speech production but are also observed in unrelated sensorimotor systems. These results are consistent with functional brain imaging data revealing atypical activation across cortical and subcortical neural circuits—including the basal ganglia—in stuttering individuals. The latter finding is especially noteworthy in light of pharmacological work showing that D2 receptor antagonist medications decrease stuttering severity. Thus, independent lines of research provide compelling evidence suggesting that the basal ganglia dopaminergic system may play an important role in stuttering. Here, we propose a computational model of the mechanisms through which hyperactivity in the dopaminergic pathways may cause speech dysfluencies. Our computational approach models the direct and indirect basal ganglia pathways within the cortico-thalamo-corticol loop, disinhibition versus inhibition of the thalamus by these respective pathways, and differential effects of dopamine on D1 and D2 receptors. Simulations demonstrate how dopaminergic hyperactivity can lead to sound/syllable repetitions and how the pharmacological modulation of neurotransmitter levels can reduce stuttering severity. [Funded, in part, by NIH/NIDCD grant R01DC007603]