



Distinguished Fellowship Award Brief

INVESTIGATING AND OPTIMIZING REINFORCEMENT LEARNING IN CEREBELLAR ATAXIA (2016-2018)

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Research Question: (1) Can we use reinforcement motor learning to improve specific symptoms of cerebellar ataxia? (2) Can we improve the efficiency of reinforcement learning following cerebellar damage with non-invasive brain stimulation?

Interdisciplinary Approach: This fellowship project integrates knowledge from the fields of computational neuroscience, machine learning, and non-invasive brain stimulation to approach a long-standing problem in rehabilitation therapy with a novel perspective.

Potential Implications of Research: If reinforcement learning and non-invasive brain stimulation can improve ataxic reaching movements, this raises the potential to develop novel rehabilitation techniques to ameliorate motor function and quality of life in patients with cerebellar damage.

Many people have experienced difficulties with balance, walking, speaking, and limb control that result from having too many drinks at a party. This condition of impaired coordination is called *ataxia* and results from disrupting the function of a brain structure called the cerebellum. While alcohol-induced ataxia is often temporary, many neurological conditions (e.g. stroke, M.S., genetic) permanently damage the cerebellum leaving the individuals affected with chronic ataxia. There are currently no medications to treat ataxia, so patients rely on physical therapy. However, physical therapy is problematic for ataxia because many programs exploit a type of motor learning (called error-based learning) that is impaired by cerebellar damage. This learning uses sensory information about movement errors to compare the intended action with what actually occurred. In the clinic, therapists encourage error-based learning by providing patients with a lot of sensory feedback (e.g. vision of errors in direction or extent), but people with ataxia are often unable to use this information to correct their movements. Fortunately, the brain has other learning mechanisms that are less cerebellum-dependent and harnessing them could improve ataxia rehabilitation.

Reinforcement learning is one mechanism that uses connectivity between 2 brain areas, the primary motor cortex (M1) and the basal ganglia, to bias movements toward actions that yield the most rewarding results (e.g. points). This type of learning uses binary information about movement outcome (e.g. hit or miss) instead of using precise sensory errors. We have recently shown that reinforcement learning is intact in individuals with ataxia and could be used to learn a new reaching movement. The same individuals could not learn this movement in an error-based learning task. These findings suggest that ataxia patients may learn better from less movement feedback. However, reinforcement learning did function less efficiently in patients due to their ataxia increasing movement variability that the brain could not estimate. Therefore, investigating whether we can improve the efficiency of reinforcement learning would benefit any new therapy, as it would reduce the amount of rehabilitation needed.

The funded project has two aims. The first aim is to take the next step and determine whether reinforcement learning mechanisms can be used to improve a symptom of ataxia that impairs patients' movements in daily life. Specifically, ataxia causes movements with highly irregular movement paths that exhibit a high magnitude of jerk (the derivative of acceleration). Learning to minimize jerk is thought to be integral for making smooth, accurate movements, which is the goal of rehabilitation training. We hypothesize that reinforcing reaching movements that minimize jerk may help ataxia patients improve the smoothness of their reaches. The second aim is to determine whether we can improve the efficiency of reinforcement learning in cerebellar ataxia using non-invasive brain stimulation. Transcranial direct current stimulation (tDCS) applies weak electrical current to the skull to alter the activity of neurons in the underlying brain region. Damage to the cerebellum decreases neural activity in M1. Increasing activity in M1 with anodal tDCS has been shown to reduce motor variability in the control of arm movements in ataxia patients. We hypothesize that increasing M1 activity with anodal tDCS may improve reinforcement learning in cerebellar patients by decreasing some of the motor variability caused by ataxia.