

# Abstract

Human sensorimotor control can achieve highly reliable movements under circumstances of noise, redundancy, uncertainty, and sensory delays. Our ability to achieve reliable and accurate movements is in the fact we have a nervous system that learns these limitations and continuously compensates for them. The purpose of the thesis is to understand brain mechanisms and computations underlying supervised motor learning, its interaction with reinforcement learning and study its relation to motor variability. To address these issues, we have investigated factors influencing supervised motor learning such as neurological disease condition, the role of the reinforcement signal, motor variability and motor redundancy.

Traditionally, supervised or error-based learning and reinforcement or reward based learning are thought to be occurring at anatomically different places and have functionally separate mechanisms. By leveraging the performance of human patients with Parkinson disease and cerebellar ataxia disease, we demonstrate how the presence and absence of dopamine medication and subthalamic deep brain stimulation (STN-DBS) influenced supervised learning. Furthermore, we also show that the presence and absence of reinforcement at the end of the trial profoundly affected learning such that the difference in learning as a consequence of medication reduced significantly. These results suggest that the basal ganglia modulate the gain of supervised learning in the cerebellum based on the reinforcement received at the end of the trial.

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Furthermore, we explored motor variability (thought to be an unwanted characteristic of the motor system) and investigated its significance and effect on supervised motor learning. We propose that some part of motor variability arises out of the redundancy in the joints in the human arm. We showed that greater uses of redundancy in the arm lead to faster learning across healthy subjects. We observed these both in dynamic perturbation learning and kinematic perturbation learning. Interestingly, we also found differences in the use of redundancy between the dominant hand and non-dominant hand, suggesting that the nervous system actively controls the redundancy. Furthermore, we also observed some directions in reaching are difficult to learn in comparison to others directions. To understand such behavior, we separated direction wise errors and constructed errors ellipses and found out that eccentricity of ellipse change with learning, which suggests brain while reducing errors in learning, is also trying to homogenize the distribution of errors caused by the perturbation. We also found interesting differences between redundancy and motor learning that was selectively impaired in PD patients but not cerebellar patients, possibly pointing to a role of the basal ganglia in processing of the use of redundancy in motor learning.

In summary, the results in the thesis provide experimental support for the hypothesis that the basal ganglia modulate the gain of supervised learning and exploration of redundancy aids in learning and that the redundancy component of the motor variability is not noise. In future, we hope that this relationship between basal ganglia, reinforcement, and redundancy in supervised motor learning can be leveraged to enhance motor rehabilitation and motor skills in patients with motor deficits.