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Key brain regions talk directly with each other, scientists say

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Researchers at the University of Pittsburgh have found new evidence that the basal ganglia and the cerebellum, two important areas in the central nervous system, are linked together to form an integrated functional network. The findings are available online this week in the *Proceedings of the National Academy of Sciences*.

"The basal ganglia and the cerebellum are two major subcortical structures that receive input from and send output to the cerebral cortex to influence movement and cognition," explained senior author Peter L. Strick, Ph.D., professor of neurobiology and co-director of the Center for the Neural Basis of Cognition, Pitt School of Medicine.

Each subcortical structure houses a unique learning mechanism. Basal ganglia circuits are thought to be involved in reward-driven learning and the gradual formation of habits. In contrast, cerebellar circuits are thought to contribute to more rapid and plastic learning in response to errors in performance.

"In the past, these two learning mechanisms were viewed as entirely separate, and we wondered how signals from the two were integrated," Dr. Strick said. "Using a unique method for revealing chains of synaptically linked neurons, we have demonstrated that the cerebellum and basal ganglia are actually interconnected and communicate with each other."

This result not only has important implications for the normal control of

movement and cognition, but it also helps to explain some puzzling findings from patients with basal ganglia disorders.

For example, Parkinson's disease is known to be caused by the degeneration of a specific set of neurons and their synapses in the basal ganglia. However, one of the treatments for the characteristic "resting" tremor of Parkinson's disease is to interrupt signals from the cerebellum to the cerebral cortex. Imaging studies of patients with Parkinson's disease and patients with dystonia, another disorder thought to be of basal ganglia origin, show abnormal increases in activity in the cerebellum.

"Our findings provide a neural basis for these findings," Dr. Strick said. "In essence, the pathways that we have discovered may enable abnormal signals from the basal ganglia to disrupt cerebellar function. The alterations in cerebellar function are likely to contribute to the disabling symptoms of basal ganglia disorders. Thus, a new approach for treating these symptoms might be to attempt to normalize cerebellar activity."

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