Rutgers researcher finds brain connections may reorganize in Parkinson’s disease

NEW BRUNSWICK, N.J.– Researchers at Rutgers, The State University of New Jersey, have discovered critical clues that may explain why parts of the brain damaged by Parkinson’s disease, specifically those that control sensory-guided movements, aren’t repaired by dopamine replacement therapy.

Parkinson’s is the second most common neurodegenerative disease affecting older people in the United States and is characterized by a loss of dopamine in the basal ganglia portion of the brain.

“Our research indicates that the loss of dopamine seems to cause connections in the brain to reorganize. This affects the brain’s ability to communicate effectively with body parts, for example, the ability to respond to stimuli from the body in order to control body movements,” explained Mark West, a behavioral neuroscientist and professor in the department of psychology at Rutgers’ Faculty of Arts and Sciences-New Brunswick.

His findings are presented in the study “Dopamine depletion causes fragmented clustering of neurons in the sensorimotor striatum: Evidence of lasting, altered responsiveness to corticostriatal input,” published in the (date) issue of the Journal of Comparative Neurology.

West and his research team studied rats and produced the first-ever microelectrode functional map of the sensorimotor portion of the basal ganglia in a Parkinsonian brain. They found that clusters of neurons in the basal ganglia that control specific movements became smaller in size when dopamine deprived.

“The neurons weren’t shrinking or moving closer together; they were still distributed about the same as a normal brain,” explained West. “But evidence showed that around the edges of the clusters some of the neurons changed their responsiveness and were thereby excluded from their original clusters. Some remained isolated and others became affiliated with nearby clusters.”

For example, according to West, a fore-limb cluster neuron could become affiliated with a hind-limb cluster and act just like a regular hind-limb neuron. The problem is that the neuron now receives hind-limb signals but maintains its original downstream communication with the fore-limb. The result is that the brain is unable to process these signals to control movement of the fore-limb.

“We saw this switching of connectivity happening throughout the basal ganglia, impacting many different body parts. In some cases these neurons would affiliate with more than one cluster, making accurate response to stimulation, such as touch, almost impossible,” stated West.

According to West, this connection switching appears permanent, with these changes still evident up to a year after dopamine loss. “These switched connections may explain why L-DOPA and other drugs are unable to restore complex sensorimotor behaviors such as maintaining manual contact with an unseen, moving object.”

Although conducted with animal models, West’s research is applicable to Parkinson’s disease in humans. “Human studies of the basal ganglia show overlapping areas that control leg and arm movements, organized similarly to rats, which means it is possible for connections to get switched in the human brain just as in the rat brain.”

West’s results are also consistent with previous studies that have shown that loss of dopamine in humans and animals causes the cortex to sprout new connections to the basal ganglia.

West hopes his findings, which show apparently permanent changes caused by the loss of dopamine to the brain, will encourage more research focused not only on treating, but also preventing Parkinson’s disease.

West has been studying the impact of dopamine deficiency and neuron activity in the brain related to Parkinson’s disease for more than a decade. He heads a behavioral neuroscience laboratory at Rutgers.