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Synchronous Neuronal Firing May Underlie Parkinson's Disease

Science Daily — In a finding that contradicts current theories behind Parkinson's disease, neuroscientists at Duke University Medical Center have discovered in mice that critical nerve cells fire all at the same time and thus overwhelm the brain's ability to control the body's movements.

Senior study investigator Miguel Nicolelis, M.D., Ph.D. (Image courtesy of Duke University Medical Center) Ads by Google Advertise on this site

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Previously, scientists had thought that the abnormal body movements characteristic of Parkinson's resulted from nerve cells in a specific brain region called the motor cortex firing at a decreased rate, though still in an ordered manner.

"Imagine an orchestra playing a beautiful symphony, with each instrument playing a different part, but in harmony. That is the way the brain normally works, with nerve cells sending different but coordinated signals throughout the brain," said senior study investigator Miguel Nicolelis, M.D., Ph.D., Anne W. Deane Professor of Neuroscience. "We found that in an animal model of Parkinson's, nerve cells seem to fire all at the same time, rather than in harmony. It's like having all instruments playing the same note over and over again at the same time during the symphony, rather than the different instruments playing at different times."

Although the researchers made their discoveries in genetically engineered mice, they believe the same processes may occur in humans.

The findings may help researchers to better understand Parkinson's disease and to develop new therapeutics for the debilitating disorder, said lead study investigator Rui Costa, D.V.M., Ph.D., chief of the section of in vivo neural function at the National Institutes of Health, who launched this study as a postdoctoral fellow in Nicolelis' laboratory.

"Therapeutic interventions that restore the normal synchrony of these neurons in the brain may potentially be beneficial in treating Parkinson's disease," Costa said.

The researchers published the findings in the Oct. 19, 2006, issue of the journal *Neuron*. The work was funded by the National Institutes of Health, the Hereditary Disease Foundation and the Anne W. Deane chair endowment to Nicolelis.

Parkinson's disease is the second most common neurodegenerative disorder in the United States, surpassed only by Alzheimer's disease. Approximately 1 million Americans have Parkinson's disease, and more than 50,000 new cases are diagnosed each year. The symptoms of Parkinson's disease include tremors or trembling, general slowness of movement, stiffness or rigidity of muscles, and difficulty maintaining balance and gait.

Parkinson's disease results from the loss of nerve cells, or neurons, that produce an important brain chemical called dopamine. Neurobiologists previously believed that the tremors and muscular rigidity of this disease were caused by decreases in the activity of neurons in the motor cortex.

Dopamine is a neurotransmitter, a chemical that neurons release to their neighbors to signal them to fire nerve impulses. Dopamine is known to control movement, balance, emotion, and the sense of pleasure.

Normally, when a signal needs to travel through the brain, neurons release dopamine to transport the signal across the gap, or synapse, between neurons. A kind of protein pump, called a transporter, recycles dopamine back to the signaling neurons to prepare for the next burst of signal.

In studies 10 years ago, Marc Caron, Ph.D., James B. Duke professor of cell biology at Duke and a co-investigator in the current study, used the techniques of genetic engineering to produce a strain of mice that lacked this protein transporter. Treatment of these mice with a chemical that completely stops the production of dopamine resulted in mice that quickly ran out of their supply of the neurotransmitter. The treated mice became rigid and immobile, displaying symptoms similar to those experienced by patients with Parkinson's disease.

In the current study, the researchers measured the electrical activity simultaneously in the motor cortex and the striatum, another critical area of the brain, in these dopamine-depleted mice. Communication of signals among the neurons in these regions controls movement, balance and walking, and it is this communication that is disabled in Parkinson's disease.

Using electrodes finer than a human hair implanted into individual neurons, the researchers could monitor signals passed among neurons in the treated mice. They

found the overall level of activity of the neurons in the motor cortex did not change. Instead, the neurons fired in unison, leaving the Parkinsonian mice unable to direct or control their movement in a normal manner.

In another part of the study, the researchers found evidence that may provide insight into the underlying biology of schizophrenia. In these tests, the researchers examined mice not treated with the dopamine-blocking chemical, as the excess dopamine that accumulates in the brains of such animals is known to make them exhibit the bizarre behaviors experienced by people with schizophrenia.

It turned out that the excessive dopamine caused the mice's neurons to fire in a less synchronous manner, just the opposite of what happened in the Parkinsonian mice, the researchers said.

Other researchers who participated in the study were Shih-Chieh Lin, Tatyana Sotnikova, Michael Cyr and Raul Gainetdinov.

Note: This story has been adapted from a news release issued by Duke University Medical Center.

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