UT SOUTHWESTERN MEDICAL CENTER NEWS

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UT Southwestern public forum speaker to discuss research into the proteins behind Alzheimer's

DALLAS – Oct. 25, 2010 – Dr. Lennart Mucke, professor of neurology and neuroscience at the University of California, San Francisco, will discuss his research on proteins involved in neurodegenerative conditions like Alzheimer's disease at a Fall Public Forum on Nov. 3 at UT Southwestern Medical Center.

The 7 p.m. event, presented by the Friends of the Alzheimer's Disease Center at UT Southwestern, is titled "What Will It Take to Defeat Alzheimer's Disease?" It will be held in the Simmons/Hamon Biomedical Research Buildings on the North Campus, 6000 Harry Hines Blvd. Complimentary valet parking is available.

The public forum is free, but because seating is limited attendance should be confirmed by calling the medical center's Office of Development at 214-648-2344.

The abnormal buildup of various proteins, both inside and outside of brain cells, is associated with Alzheimer's and Parkinson's diseases. Research by Dr. Mucke, director and senior investigator of the Gladstone Institute of Neurological Disease, focuses on how the proteins contribute to the malfunction of brain cells. He has found that proteins that are harmful on their own can interact with others to cause even greater neurologic damage.

"We're trying to find out what causes the cognitive impairment and discover ways to make the brain more resistant to these damaging proteins," said Dr. Mucke, who received this year's Potamkin Prize for Research in Pick's, Alzheimer's, and Related Diseases, awarded by the American Academy of Neurology.

"If one could remove the toxic proteins from the brain or block their detrimental actions, I can imagine that there might be a remarkable recovery of function."

Alzheimer's disease is associated with the abnormal accumulation of several proteins; beta-amyloid and tau are the most infamous. Beta-amyloid forms clusters called plaques outside the brain cells of people with the disease. Tau protein, which normally stabilizes the shape of brain cells, creates abnormal clumps within the cells during the disease. Parkinson's disease is associated with another protein, alpha-synuclein, which forms clumps in the brain called Lewy bodies.

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Dr. Mucke found that beta-amyloid can damage connections between nerve cells even without being deposited into visible plaques. "These plaques are like large garbage dumps of amyloid," he said. "We think they're relatively inert compared to these cruise missiles that are floating around and that impair the communications between brain cells."

Using mice genetically engineered to model both Alzheimer's and Parkinson's diseases, Dr. Mucke and his colleagues have found that beta-amyloid and alpha-synuclein can interact to cause even worse cognitive and motor problems than either one alone.

Dr. Mucke also discovered that in mouse models of Alzheimer's disease, brain activity alternates between an epilepsy-like abnormal excitation and abnormal inhibition. Using genetic engineering to reduce the amount of tau protein in mice prevented these alterations and halted cognitive deficits induced by beta-amyloid.

"If drugs that reduce tau could be found, this approach could complement strategies aimed at beta-amyloid itself, and might prove especially useful if the efficacy and long-term safety of anti-amyloid strategies turn out to be limited," he said.

In ongoing studies, Dr. Mucke and his colleagues are exploring how beta-amyloid affects nerve function and survival. They are also looking at how reducing tau protein protects against deficits induced by beta-amyloid, and whether there is a way to exploit this protective effect therapeutically.

The Friends of the Alzheimer's Disease Center was established in 1996 to provide financial support for Alzheimer's research at UT Southwestern. All of the group's contributions go directly to support Alzheimer's research at the medical center.

Since its founding, the group has raised more than $989,200 for grants to researchers. For information on joining the group, call the UT Southwestern development office at 214-648-2344.

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