New study in mice tracks cause of trigeminal neuralgia pain

Mice may help scientists gain insight into the loss of a protective nerve coating that is thought to cause the shocking facial pains of trigeminal neuralgia—the world's most painful medical problem.

In an effort to develop an animal model for this agonizing human disorder, neuroscientists at the University of Florida are launching studies in mice deficient in a gene that produces myelin, a fatty substance which surrounds nerves like an insulating sheath. The initial year-long study is under way at UF's McKnight Brain Institute, aided by a grant from TNA-The Facial Pain Association.

Lucia Notterpek, Ph.D., project director, said her team will track the biological events that destroy myelin to the point that nerve impulses cannot be conducted normally. Loss of myelin coating on the trigeminal nerve, close to where it enters the brain, is strongly suspected as a cause of trigeminal neuralgia. Myelin damage has been found in many, but not all, patients with TN pain.

"Through multiple repeated experiments, we will try to figure out the least amount of nerve damage required to initiate myelin damage in both healthy mice and in mice bred with a gene deficiency," Notterpek said. "We hope to determine whether mice deficient in the myelin gene (the PMP22 gene) are more susceptible than normal mice to the loss or erosion of myelin."

Notterpek said myelin damage will be induced in the mice by surgically tying a thread around a nerve (under anesthesia) in order to constrict the nerve. The intent is to reproduce the type of nerve compression found in most patients with trigeminal neuralgia. In textbook cases of the disease, the trigeminal (three-part) nerve, which energizes all areas of the face, is irritated and eroded by an aging or injured artery that sags and presses against the nerve.

The researchers will attempt to correlate myelin damage with behavioral changes, specifically how the mice respond to the application of heat or mild pressure on the area of skin innervated by the injured nerve.

The outcome of this study may lay the groundwork for testing various pain-relieving treatments, including nerve-protective medications or by "patching" missing segments of myelin with myelin-producing cells derived from mouse stem cells. Notterpek said the study also may aid understanding and treatment of muscular sclerosis, in which small areas of myelin are damaged.

Notterpek, professor and chair of the UF Department of Neuroscience, earned her Ph.D. in neuroscience from the University of California at Los Angeles and completed postdoctoral training in neurobiology at Stanford University.