

January 10, 2013

NIH Statement

In July 2012, the National Institutes of Health (NIH) initiated research including an examination of the brain of the former NFL player, Junior Seau, which had been donated by his family. NIH National Institute of Neurological Disorders and Stroke (NINDS) invited several nationally recognized neuropathologists to consult in the analysis of the brain tissue. At the request of the family, the NIH is today releasing the results of the analysis.

On initial examination the brain looked normal but under the microscope, with the use of special staining techniques, abnormalities were found that are consistent with a form of chronic traumatic encephalopathy (CTE). The neuropathologists each examined tissue samples from three different unidentified brains. The official, unanimous diagnosis of Mr. Seau's brain was a "multi-focal tauopathy consistent with a diagnosis of chronic traumatic encephalopathy." In addition there was a very small region in the left frontal lobe of the brain with evidence of scarring that is consistent with a small, old, traumatic brain injury.

Specifically, the neuropathologists found abnormal, small clusters called neurofibrillary tangles of a protein known as tau within multiple regions of Mr. Seau's brain. Tau is a normal brain protein that folds into tangled masses in the brain cells of patients with Alzheimer's disease and a number of other progressive neurological disorders. The regional brain distribution of the tau tangles observed in this case is unique to CTE and distinguishes it from other brain disorders.

The type of findings seen in Mr. Seau's brain have been recently reported in autopsies of individuals with exposure to repetitive head injury, including professional and amateur athletes who played contact sports, individuals with multiple concussions, and veterans exposed to blast injury and other trauma.

CTE was first described in studies of boxers who developed dementia and Parkinson's disease-like symptoms. The signature sign of this progressive degenerative brain disorder included tangles of tau inside brain cells as well as extensive cell death and shrinkage of the brain. More recently, some people with repetitive head injury have also been found to have a more limited, multifocal tauopathy without brain shrinkage, dementia or parkinson-like symptoms. In many cases friends and family described personality changes, depression, increased irritability, and trouble with attention. The relationship between the multifocal tauopathy form of CTE and the symptoms is poorly understood. Whether and how the multi-focal form of CTE progresses to the more extensive brain degeneration is still unclear.

CTE research is in a very early stage. Currently, physicians are unable to diagnose the multi-focal tauopathy form of CTE in a living person; CTE can only be confirmed by examining the brains from individuals upon autopsy. No data are available to indicate the frequency of CTE. Similarly, we do not understand which individuals with multiple impacts to the head or exposures to blast injury are at risk for CTE. Investigators at NIH are now attempting to correlate brain tissue pathology with detailed images taken with the NIH's high resolution 7 Tesla MRI scanner. Only research will reveal answers to the vexing problems that this condition presents.

A copy of the report is attached.

NIH is indebted to the family for the donation of Mr. Seau's brain for research on CTE. As per the family's wishes, further questions about the results should be directed to Russell Lonser, M.D., Chair of Neurological Surgery at Ohio State University and immediate past Chief of Surgical Neurology at NINDS. NIH will not discuss any further details of the analysis or the results.

Background:

The National Institutes of Health is the largest funder of medical research in the world. More than 80% of the NIH's budget goes to more than 300,000 research personnel at over 3,000 universities and research institutions. In addition, about 6,000 scientists work in NIH's own Intramural Research laboratories, most of which are on the NIH main campus in Bethesda, Maryland.

Questions about the science of CTE or research on CTE can be directed to the Office of Communications at NINDS (NINDSPressTeam@ninds.nih.gov). A report from a recent NINDS workshop on chronic traumatic encephalopathy is available at: www.ninds.nih.gov/2012CTEworkshop.