First former college football player diagnosed with CTE

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The Center for the Study of Traumatic Encephalopathy (CSTE) at Boston University School of Medicine (BUSM) announced today that a deceased former college football player who died at age 42 was already suffering from the degenerative brain disease, Chronic Traumatic Encephalopathy (CTE). This is the first time an advanced case of CTE has been discovered in a college football player that did not play professionally. It is also the first case diagnosed in a wide receiver. CTE has been diagnosed post-mortem in at least seven recently deceased former National Football League players, and early signs of the disease were recently found by CSTE researchers in an 18 year-old deceased football player.

CTE was diagnosed in Mike Borich, a Snow College and Western Illinois University player in the 1980s, by neuropathologist Ann McKee, MD, co-director of the CSTE. Borich went on to become an award-winning division I college football coach, and was named the Offensive Coordinator of the Year in 2001, while coaching at Brigham Young University under head coach Gary Crowton. Borich also coached for the NFL's Chicago Bears in 1999-2000. He left coaching in 2003 struggling with overwhelming drug and alcohol addictions, ultimately dying from a drug overdose in February 2009. Other CTE sufferers, such as Tom McHale of the Tampa Bay Buccaneers, died with similar late-onset drug and alcohol problems. Borich was known to have approximately 10 concussions during his college football career with no subsequent concussions or head injuries since that time.

Robert Cantu, MD, a leading sports concussion expert and BUSM CSTE co-director and clinical professor of neurosurgery at BUSM said, "CTE is the only fully preventable cause of dementia. It is our hope that this evidence helps draw the focus of the CTE discussion to amateur athletes, where it belongs. Young men and women are voluntarily exposing themselves to repetitive brain trauma without full knowledge of the potential consequences, and the rules of the games are designed without an appreciation for the risks carried by the players."

Joe Borich, Mike Borich's father, donated his son's brain tissue to the CSTE Brain Bank, a brain tissue repository for the study of CTE. By donating Mike's brain, he hoped to enable athletes to play sports more safely. He also anticipated that the analysis might provide a window into Mike's personality changes and increasingly self-destructive behavior. "Mike suffered greatly in his last few years. Through donating his brain to research I hope that his suffering will now have had meaning, and his legacy will be that in his death he helped to save others."

Cantu and McKee and the other co-directors of the BUSM CSTE, Robert Stern, PhD, and Chris Nowinski, a former division I football player, published a paper that reported all CTE findings in athletes in the July issue of the *Journal of Neuropathology and Experimental Neurology* (2009, vol.68¸ pp. 709-735). McKee also recently presented these findings to the NFL Mild Traumatic Brain Injury Committee and NFL Players Association.

Stern added, "The US House Judiciary Committee is holding a hearing on the NFL head injury crisis next week. This evidence should be part of the discussion. Brain trauma in sports is a public health problem, not just an NFL problem."

Borich was not part of the CSTE Brain Donation Registry when he died. The CSTE Brain Donation Registry has now enrolled over 175 athletes

in the C.O.N.T.A.C.T. research program (Consent to Offer Neural Tissue of Athletes with Concussive Trauma). These athletes will be interviewed annually by phone throughout their lives and, upon death, their brain tissue will be examined by the CSTE. This prospective approach with allow the researchers to examine the relationship between clinical symptoms and pathology for the first time.

CTE, first reported in 1928 and originally referred to as "dementia pugilistica" because it was believed to only affect boxers, is a progressive neurodegenerative disease caused by repetitive trauma to the brain. The use of the terms Traumatic Encephalopathy and CTE were first used in the 1960s.

The disease is characterized by the build-up of a toxic protein called tau in the form of neurofibrillary tangles (NFTs) and neuropil threads (NTs) throughout the brain. The abnormal protein initially impairs the normal functioning of the brain and eventually kills brain cells. Early on, CTE sufferers may display clinical symptoms such as memory impairment, emotional instability, erratic behavior, depression and problems with impulse control. However, CTE eventually progresses to full-blown dementia. Although similar to Alzheimer's disease, CTE is an entirely distinct disease.

Source: Boston University Medical Center