

Researchers report first case of chronic traumatic encephalopathy in an active college football player

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The Center for the Study of Traumatic Encephalopathy (CSTE) at Boston University School of Medicine (BUSM) announced today that University of Pennsylvania (Penn) football co-captain Owen Thomas was suffering from mild stages of the degenerative brain disease, Chronic Traumatic Encephalopathy (CTE), when he committed suicide in April at the age of 21, becoming the first case of CTE in an active college football player. It was the Thomas family's request that these findings be made public.

Thomas joins a growing list of active and retired football players and other contact sport athletes that have been diagnosed post-mortem with CTE, which is caused by repetitive concussions as well as sub-concussive brain injuries. The diagnosis was made by neuropathologist and CSTE co-director Ann McKee, MD, an associate professor of neurology and pathology at BUSM, as well as Director of Neuropathology for the Department of Veterans Affairs at the Bedford VA Medical Center. The CTE diagnosis was independently confirmed by another neuropathologist, Daniel Perl, MD, professor of pathology at Uniformed Services University of the Health Sciences. McKee has now diagnosed CTE in 12 of 13 former [professional football players](#), as well as amateur football players, professional boxers, and hockey players. According to Thomas's medical records and family, he had never been diagnosed with a concussion during 13 seasons of football, beginning at age 9. However, the positions he had played (e.g., lineman, linebacker) typically involve

multiple hits to the head during every game and practice, with estimates of approximately 1000 hits per season or more.

Thomas, who will be honored and remembered during a ceremony Saturday before Penn's opening game versus Lafayette, was finishing his junior year at Penn when he committed suicide at his off-campus apartment on April 26, 2010. He was named one of four co-captains of the 2010 Penn team after being named Second-Team All-Ivy after the 2009 season at defensive end. Referred to as "the most popular kid on our team," by Penn players and coaches, Thomas had no history of mental illness and did not show signs of depression. His parents, the Reverends Thomas Thomas and Katherine Brearley, reported Owen was "feeling stressed" after a difficult academic spring, saying, "I'm failing everything."

Symptoms of CTE include memory impairment, emotional instability, erratic behavior, depression, and problems with impulse control. The disease eventually progresses to dementia if the individual lives long enough. It is unclear if Thomas's feelings of stress in the spring were related to the pathology in his brain. In addition, it is impossible to definitively link his suicide to CTE, although other CTE researchers have noted a pattern of suicide and suicidal behavior in CTE victims, such as National Football League (NFL) stars Andre Waters, Justin Strzelczyk, and Terry Long.

"We will never truly know what led Owen to take his own life," said his mother, Rev. Brearley. "However, knowing that he had a brain disease that could have had an impact on his emotional state, cognitive functioning, and impulse control, brings us bittersweet solace," she said.

"It may have been out of his control," added his father, Rev. Thomas, who went on to say, "if he hadn't taken his life now, I'm concerned by how his life would have turned out given the progressive nature of CTE."

In January 2009, McKee reported finding the earliest stages of CTE in the youngest athlete to date, a high school multi-sport athlete who passed away at the age of 18, and whose name has been withheld at the request of his family. Thomas, at 21, is the second-youngest case of CTE in a football player, but his disease appeared more advanced than that of the 18 year-old.

CSTE co-director Chris Nowinski, who, like Thomas, was a Second-team All-Ivy defensive lineman when he played for Harvard University in 1999, said, “These findings should serve as another wake-up call that we need to continue to radically re-think how we play football in America, especially at the youth level. One in eight boys in America plays football, and yet we have never studied the effects of repetitive brain trauma at that age, even though scientists suspect that brain trauma is more damaging for the developing brain.”

Nowinski and CSTE co-director Robert Cantu, MD, founded the non-profit Sports Legacy Institute (SLI) in 2007 to “Solve the Sports Concussion Crisis” and partnered with Boston University to form the CSTE in 2008. To combat that lack of attention and resources in youth sports, yesterday SLI released Minimum Recommended Guidelines for Brain Protection in Youth Sports, which contains seven simple and free steps that every youth sports program in America should follow.

Dr. Cantu said, “We can, and we must, develop brain trauma guidelines similar to the ‘Pitch Count’ regulations now used in Little League baseball. We count the pitches of every baseball player to ensure a small number do not develop shoulder and elbow problems, and yet we don’t count how often children get hit in the head playing football, even though it can lead to early dementia, or possibly depression and suicide years later.” Cantu, a clinical professor of neurosurgery at BUSM, was recently appointed senior advisor to the NFL Head, Neck and Spine Committee.

In addition, SLI recently helped pass a law in Massachusetts that requires mandatory brain trauma education for all coaches, athletes, and parents in sports governed by the Massachusetts Interscholastic Athletic Association. The bill also calls for no return-to-play on the day of a suspected concussion and requires athletes be cleared by a medical professional after a concussion.

CTE 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. CTE is caused by repetitive brain trauma, including concussive or subconcussive blows to the head, but the symptoms of the disease may not be evident for years or decades after the trauma.

The CSTE maintains a registry of more than 350 living athletes who have agreed to donate their brain and spinal cord following death and to take part in annual telephone-based interviews and assessments during their lifetimes. Robert Stern, PhD, CSTE co-director and an associate professor of neurology at BUSM, oversees the CSTE registry as well as a study of retired NFL players which is about to begin and involves extensive neurological, cognitive, psychiatric, cerebrospinal fluid, and neuroimaging studies.

“It is only through extensive research that we will understand the genetic and other risk factors for CTE, as well as be able to diagnose the disease while someone is alive and eventually treat it,” said Stern, who also serves as director of the BU Alzheimer's Disease Center Clinical Core. “We know that repetitive blows to the brain cause CTE, but we don't

know why some individuals like Owen Thomas get it and others do not. Football and other contact sports have tremendous benefits to those who play them and to our society as a whole. The ultimate goal of our research is to make sure that these sports are played safely and that we reduce the potential for long-term consequences of repetitive [brain injury](#) in youth, college, and professional athletes,” added Stern. In order to help move this research forward, Owen Thomas’s father, Rev. Thomas, who himself played college football, recently became a participant in the BU CSTE research registry.

CTE, originally referred to as “dementia pugilistica” because it was believed to only affect boxers, is a progressive brain disease believed to be caused by repetitive trauma to the brain, including concussions or subconcussive blows. It is characterized by deposits of an abnormal protein called tau in the form of neurofibrillary tangles, glial tangles, and neuropil threads throughout the brain, and, in some cases, the presence of another protein - associated with motor neuron disease - known as TDP-43. These abnormal proteins are associated with the impaired functioning and eventual death of brain cells. Early on, CTE sufferers may display symptoms such as memory impairment, emotional instability, erratic behavior, depression, and problems with impulse control. CTE may eventually progress to full-blown dementia. Although similar to Alzheimer’s disease, CTE is pathologically distinct, and it is the only known preventable cause of dementia.

Provided by Boston University

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