

Suicide in Professional Athletes: is it related to the sport?

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If recent headlines about athletes dying by suicide have made you wonder whether progress in brain research can help shed light on the potential role in these suicides of head hits in sport, then you are right—and not just in the case of the athletes. Research on suicide across its spectrum has told us enough about the brain to greatly help in considering whether and how head trauma may have set the stage when an individual dies by suicide.



Suicide is the third leading cause of death in the United States in young people aged 10-24 years and the second leading cause of death among ages 25 to 34 years. These ages also span the period when most professional athletes are competing. We think of the causes and protective factors in suicide as a combination of stressors, or triggers, together with a predisposition for suicidal behavior. The two commonest triggers are an acute major depressive episode and a psychosocial crisis. The predisposition includes personal characteristics such as impulsive aggressive traits (reflecting a tendency to act on powerful emotions), pessimism (a tendency to see the cup as half empty and to experience intense suicidal thoughts when depressed), and cognitive rigidity or difficulties with problem solving (more likely to feel trapped with no solution or way out).

Most suicides, over 90 percent, occur in the context of a psychiatric illness that, in about four out of five cases, is untreated. In the United States, major depression is the psychiatric illness that accounts for about 60 percent of suicides. The highly publicized suicides in former professional football players illustrate the potential causal contribution of acquired brain disease due to the cumulative effects of repeated head hits damaging the parts of the brain that contribute to the risk for depression and suicide.

In May, 2012, *The Washington Post* reported: “Junior Seau, a linebacker who played in the NFL for 20 seasons and was among the most widely respected players of his generation, was found dead in his California home Wednesday... with a gunshot wound to the chest. There was no suicide note (and) police officials said a gun was found near Seau and his death appeared to be a suicide.” The paper also reported that “he had survived a 100-foot fall down a cliff in his car in October 2010, ... and police said it was believed he fell asleep at the wheel”. Police seem to have reached this conclusion based on Mr Seau stating he had fallen asleep at the wheel, but another potential explanation is that this had been a suicide attempt that he was fortunate to survive. The importance of this question is that a nonfatal suicide attempt is associated with a 20- to 50-fold greater risk for suicide compared with the general population.

Two other former NFL players also died by suicide recently. In April, 2012 former Atlanta Falcons safety Ray Easterling’s death at age 62 was ruled a suicide. And in 2011, former Chicago Bears safety Dave Duerson committed suicide. Easterling is reported to have suffered from depression and insomnia, and then dementia that progressively worsened. He and Duerson each died by a self-inflicted gunshot wound. Tellingly, Duerson shot himself in the chest and in his suicide note made it clear that he did so to preserve his brain so that it could be studied by researchers investigating brain damage in NFL players. Like Easterling, Duerson described a progressive deterioration in his memory and difficulty stringing words together. More than 1,500 former players are now suing the league, claiming that, for years, it ignored evidence that repeated blows to the head trigger chronic traumatic encephalopathy, or CTE, a progressive neurodegenerative disease caused by repetitive trauma to the brain which has been linked to dementia and depression.

As of Jan 2009, the Center for the Study of Traumatic Encephalopathy at Boston University School of Medicine had reported six former NFL players with CTE and even a case in an 18 yr. old high school footballer. CTE is characterized by a very high level of a protein called tau that aggregates into neurofibrillary tangles and is found in the brains of individuals engaged in contact sports that have died from other causes such as suicide. It is hypothesized that this abnormal protein, which preferentially accumulates in outer layers of the brain, impairs and eventually kills brain cells. Early on, CTE causes emotional instability, poor concentration, word-finding difficulties, depression, suicidal thoughts and problems with impulse control. However, CTE also involves memory problems that eventually progress to dementia. A key feature is that the illness continues to progress for years after the trauma and concussions. Football players are not alone: sports like [ice hockey, soccer and boxing are thought to increase the risk for CTE](#) and [soldiers exposed to bomb blasts have developed CTE](#). All of the first six NFL former players who were found to have CTE by the Boston group had died by age 50 and three were suicides.

Although it seems that dementia and suicide in football players brought this disease to public notice, the pathology is the same as that seen years earlier in boxers, called *dementia pugilistica* and a variant that causes Parkinson's disease. Muhammad Ali is the most famous example of this illness. Although the severity of the brain pathology in boxers is correlated with the number of rounds boxed over a lifetime, no such relationship has been identified with the number of concussive episodes in sport, likely because records of such episodes are highly inaccurate.

We do not understand the reason for the difference in brain pathology seen between acute severe trauma and repeated milder trauma, but the difference implies a different pathogenesis and potential different treatment. Unlike a single [severe head injury](#), since CTE is the result of repeated milder head trauma or hits, it can be prevented by the very simple measure of discontinuing the activity that results in the repeated head hits before the illness acquires a momentum that leads it to become progressive in the absence of ongoing trauma to the head. The medical research challenge is to quantify the number and severity of head hits, or their effect on the brain, so as to determine the point in time at which

this intervention becomes essential to prevent disease progression.

How big a problem is this? We are not sure. About 1.5 million Americans suffer minor head injuries annually without loss of consciousness and no need for hospitalization. Apparently it is the repetitive nature of such injuries, perhaps with yet to be identified vulnerabilities, that cause some individuals to develop CTE. We do not know the lower limit of severity of head hits that should be counted. A group of Dutch professional soccer players was found to have cognitive deficits in memory and planning in comparison with elite athletes from other sports, and the magnitude of the deficits was in proportion to the frequency of "heading the ball".

Why are suicides and depression so common in the early phase of CTE? We are not certain but many of these sports involve blows to the front of the head and that part of the brain includes areas such as the prefrontal cortex and anterior cingulate that are required for mood regulation and impulse control. Injuries or strokes in those brain regions can affect mood and impulsiveness. [Abnormal input of the neurotransmitter serotonin](#) to those parts of the brain is observed in suicides and in major depressive disorder. Therefore acquired damage to the same brain regions may favor the development of depression and increase the likelihood of acting on depressed and suicidal thoughts and attempting suicide. The same brain regions are also involved in regulating angry feelings and the probability of aggressive acts. A history of mild head injury resulting in loss of consciousness for a short period is associated with more aggressive behavior following the injury and a greater risk of suicidal behavior. The increase in aggressive behaviors is greater in those who were more aggressive prior to the injury. Therefore, pre-existing predisposition to aggression and perhaps depression and suicidal behavior, moderates the impact of head injury on future mental health.

What needs to be done next? We need better methods of measuring the effect on future health of contact sports and in the military of soldier exposure to the percussion injury of road-side bombs. Two main approaches involve measuring the number and severity of head hits by helmet monitors and by cognitive tests. Future tests measuring the accumulation of tau protein in the brain may add precision to the determination of the effect on the brain and in particular the cumulative effect. One way to quantify or track the progressive

effect of repeated head hits over time is to image the quantity of tau protein just as we can image the amount of amyloid protein that accumulates excessively in Alzheimer's disease and after severe acute head injury. As treatments are developed for CTE we can then use such methods to track the effectiveness of the treatments in lowering levels of tau in the brain.

Further Reading:

An [excellent summary](#) is provided by Steven T. DeKosky, M.D., Milos D. Ikonovic, M.D., and Sam Gandy, M.D., Ph.D. (N Engl J Med 2010; 363:1293-1296). Trauma to the head can involve a single major acute head injury can result in accumulation of AB (beta amyloid) protein and leave residual impairment including dementia if severe enough. Milder repeated head trauma such as due to boxing, wrestling, hockey, soccer, or military service may result in CTE and often with no excessive numbers of amyloid plaques but instead a striking excessive of tau protein and neurofibrillary tangles.

In a recent study in *Neurology* in 2012 by Derek Clark (<http://www.neurology.org/content/78/22/e140.full.pdf>), 214 Division I college footballers and hockey players had devices placed in their helmets to measure head hits, including those that did not result in concussion or symptoms. Players were tested before and after the season and also compared with 45 noncontact sport athletes like rowers and

runners. Cognitive test scores in the two groups were comparable before and after the season. Although there appeared to be a subgroup of contact sport athletes whose cognitive performance declined, there was no statistically significant effect in the group as whole despite averaging 470 minor head contacts over the season. This seems like good news but perhaps the severity of the hits is greater in professional sport and in soldiers exposed to road-side bombs. Alternatively, it is the cumulative effect of head hits through high school and college and then in professional sport that increases the risk. A study of college footballers found that students who had >3 head hits were twice as likely to be symptomatic (eg headaches) for > one week (<http://jama.jamanetwork.com/article.aspx?volume=290&issue=19&page=2549>). This provides a clinical indicator of a cumulative effect. But, as the number of reported hits in the NCAA study (defined as concussions) is far fewer than the Clark study where a device in the helmet was used to detect hits that included non-concussions, we lack clear quantitative data to allow us to compare two such studies.

An example of how the threshold for the lower limit of severity of the head contact may be less than generally thought is indicated by the findings of a [study](#) that reported that soccer players had cognitive deficits in proportion to the frequency with which they would head the ball.