



Chronic Traumatic Encephalopathy Fact Sheet

What is CTE?

1. CTE is a neurodegenerative disease characterized by a unique pattern of abnormal tau deposition in the brain.¹
2. CTE appears to be progressive in most cases, with tau continuing to accumulate and spread after head impacts stop.²

What causes CTE?

3. According to the [National Institutes of Health](#), CTE is a delayed neurodegenerative disorder that research to date suggests is caused in part by repeated traumatic brain injuries.³
4. Repeated traumatic brain injuries can be caused by both concussion and nonconcussive head impacts.⁴
5. Current research suggests that CTE risk is related to the number and severity of head impacts sustained over a lifetime.⁵

How do we diagnose CTE?

6. CTE can be definitively diagnosed only postmortem by examining the brain.⁶
7. The NINDS/NIBIB CTE Diagnostic Criteria define CTE as low- and high-stage. In low-stage CTE, pathology is primarily limited to the cortex. In high-stage CTE, pathology is widely distributed throughout the hippocampus, amygdala, entorhinal cortex, thalamus, and brainstem.⁷

How common is CTE?

8. CTE is rarely seen in the general population. All studies with $n > 100$ published to date have reported a range of 0% to 2.8%.^{8 9 10 11 12 13 14}
9. Because CTE cannot be diagnosed during life, it is unknown how many athletes have CTE. One study suggests that at least 10% of individuals who have played in the National Football League (NFL) die with CTE.¹⁵



Who is at risk for CTE?

10. CTE has been now diagnosed in contact and collision sports athletes who participated in football, ice hockey, soccer, boxing, rugby union, boxing, Australian Rules Football, mixed martial arts, lacrosse, amateur wrestling, some extreme sports, and bull riding/rodeo.^{16 17 18 19}
11. CTE has been diagnosed in non-athletes exposed to repetitive head impacts, including military veterans, victims of interpersonal violence, stuntpersons, individuals with uncontrolled epilepsy, and individuals with neurodevelopmental abnormalities who engaged in repetitive head banging behavior.²⁰
12. While there may be additional causes of CTE beyond RHI, evidence is limited. As of 2023, there are only 16 cases in the scientific literature claiming to have diagnosed CTE in the absence of RHI. In 6 of those cases (Bieniek 2020), families were not contacted for sports participation history, and for an additional 5 (Iverson 2019), sport participation was not properly queried, and the lead authors of the CTE diagnostic criteria have asserted ARTAG was misdiagnosed as CTE.^{21 22}
13. A single concussion in the absence of additional head impacts has not been consistently shown to be a risk factor for CTE.²³ Most individuals diagnosed with CTE were exposed to more than 1,000 head impacts over their lifetime.²⁴
14. The youngest athlete diagnosed with CTE was 17 years old when he died.²⁵

What are symptoms of CTE?

15. Severe CTE, in the absence of any other degenerative brain disease, is associated with a 4x increased odds of a clinical diagnosis of dementia.²⁶
16. Current evidence suggests that CTE can cause cognitive, functional, and certain neurobehavioral symptoms.²⁷
17. CTE has been associated with probable rapid eye movement (REM) sleep behavior disorder.²⁸
18. One out four individuals diagnosed with CTE developed Parkinsonism.²⁹
19. In younger individuals at risk for CTE, it is unclear if presenting symptoms are related to CTE, other types of brain damage from RHI, or unrelated.³⁰



How can CTE be prevented?

20. Research suggests a reduction in the number and strength of head impacts could reduce the odds of developing CTE.³¹
21. Most individuals diagnosed with CTE were exposed to repetitive head impacts through participation in contact and collision sports. Other primary exposures include military service, interpersonal violence, brain diseases that cause frequent falls, falls secondary to epilepsy, ritualistic head-banging behaviors, and participation in specific types of entertainment in which head impacts can occur.³²
22. In brain bank studies, the odds of having CTE have been shown to increase with each additional year of participation in football (30%), ice hockey (34%), and rugby (14%).^{33 34 35}
23. The number of diagnosed or reported concussions does not predict the presence of CTE.³⁶
24. While concussion management is important, there is no research that supports the hypothesis that improved concussion management might reduce the risk of developing CTE.

¹ Bieniek KF, Cairns NJ, Crary JF, Dickson DW, Folkerth RD, Keene CD, Litvan I, Perl DP, Stein TD, Vonsattel JP, Stewart W, Dams-O'Connor K, Gordon WA, Tripodis Y, Alvarez VE, Mez J, Alosco ML, McKee AC; TBI/CTE Research Group. The Second NINDS/NIBIB Consensus Meeting to Define Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy. *J Neuropathol Exp Neurol*. 2021 Feb 22;80(3):210-219. doi: 10.1093/jnen/nlab001. PMID: 33611507; PMCID: PMC7899277. <https://pubmed.ncbi.nlm.nih.gov/33611507/>

² Alosco ML, Cherry JD, Huber BR, Tripodis Y, Baucom Z, Kowall NW, Saltiel N, Goldstein LE, Katz DI, Dwyer B, Daneshvar DH, Palmisano JN, Martin B, Cantu RC, Stern RA, Alvarez VE, Mez J, Stein TD, McKee AC. Characterizing tau deposition in chronic traumatic encephalopathy (CTE): utility of the McKee CTE staging scheme. *Acta Neuropathol*. 2020 Oct;140(4):495-512. doi: 10.1007/s00401-020-02197-9. Epub 2020 Aug 11. PMID: 32778942; PMCID: PMC7914059. <https://pubmed.ncbi.nlm.nih.gov/32778942/>

³ <https://www.ninds.nih.gov/current-research/focus-disorders/focus-traumatic-brain-injury-research>

⁴ Nowinski CJ, Rhim HC, McKee AC, Zafonte RD, Dodick DW, Cantu RC, Daneshvar DH. 'Subconcussive' is a dangerous misnomer: hits of greater magnitude than concussive impacts may not cause symptoms. *Br J Sports Med*. 2024 Jul 1;58(14):754-756. doi: 10.1136/bjsports-2023-107413. PMID: 38719575; PMCID: PMC11228231. <https://pubmed.ncbi.nlm.nih.gov/38719575/>

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