

<https://www.nadtreatmentcenter.com/chronic-traumatic-encephalopathy>

Call: 844 623-7587

[Chronic traumatic encephalopathy](#) (CTE) is a progressive degenerative disease caused by repetitive injuries to the brain occurring over a period of time. The pathology of CTE is trademarked by the buildup of abnormal proteins, also known as “tau.” Many of the symptoms associated with CTE are also found in neurodegenerative disorders and aging, which include [cognitive factors](#) such as impaired concentration, memory and language.

[Neurobehavioral changes associated with CTE](#) include:

- Severe depression
- Substance abuse
- Emotional instability
- Aggressiveness
- Poor impulse control
- Irritability
- Dementia

CTE symptoms can present gradually over a long period of time, although biological changes can be seen within a single traumatic brain injury event. Anyone who receives repetitive impacts to the head is at risk for CTE, and most cases of CTE are found in athletes participating in contact sports. One [study](#) found symptoms of post-concussion syndrome in 19-28 percent of high school athletes who had no documented history of previous concussions. Unfortunately, many [veterans](#) returning from combat also display symptoms of CTE.

Comorbidities of Chronic Traumatic Encephalopathy

Clinical features of CTE are often correlated with [comorbidities and environmental factors](#), such as:

- Developmental environment
- Neurodevelopmental disorders
- Drug and alcohol abuse
- Surgeries and anesthesia
- Sleep disturbances

Current Treatment Options

According to the [Mayo Clinic](#), the focus around current treatments for CTE is around preventing head injury in the first place. Other prevention strategies include getting plenty of rest and limiting physical activity. The Mayo Clinic also recommends surrounding yourself with a calm and supportive environment.

Alternative Therapies for CTE

Research has recently shown [NAD+](#) intravenous therapy as a promising treatment for several different neurological conditions, including CTE. [Studies](#) have shown increasing NAD+ levels can promote neurogenesis, or the growth of new brain cells, even after trauma.

In CTE, there are holes in the brain, dead areas or areas where abnormal plaques develop. Also, in ALL aging brains, lack of NAD+ seems to promote neurons to go “off-line”. Even though the brain cells are present, they don’t conduct electrical impulses as efficiently and stop reaching out to other brain cells, thus resulting in impaired brain function.

NAD⁺ encourages new connections in the brain and the connections may bypass injured or non-functional areas of the brain; resulting in increased senses and clarity of thoughts.

During our earliest years in life, the nervous system matures by seeking new connections and cementing new pathways. Over time, these pathways are either reinforced or are no longer utilized due to injury and/or fluctuations in energy. When NAD⁺ is replenished, existing brain cells seem to come back online almost immediately.

NAD⁺ encourages new connections in the brain resulting in heightened senses and clarity of thought.

It is suggested that NAD⁺ has the ability to repair and regenerate neuronal cells through several mechanisms of action. NAD⁺ encourages the mitochondria (cellular power plants) to communicate with the cell nucleus. This causes the cell to kick back into function by increasing the energy and robustness, as well as increasing sirtuin production.

A class of enzymes, known as [sirtuins](#), have been associated with the growth and extension of [new neurons](#). Sirtuins are also known to protect neurons from [oxidative stress](#) and can [inhibit cell death](#). Improving brain energy metabolism through increasing available NAD⁺ has been shown to help degrade [dysfunctional proteins](#) associated with neurodegeneration.

A recent (March 23, 2017) [report](#) from the Harvard Medical School Genetics laboratory found that NAD⁺ can help restore youthful levels of DNA repair and even reverse the effects of radiation. NAD⁺ activates the enzyme responsible for DNA repair, PARP1, which is compromised by low levels of NAD⁺ associated with aging and disease.