

These are selected posts on potential medical uses for cannabis that I wrote for [the Fresh Toast](#). As of Aug. 2024, almost all the links still work.

Can Cannabis Treat Concussion and Save Football?

Sept. 19, 2016

Richard Faulk

Citing neuroprotective properties, retired NFL players and pharma entrepreneurs endorse medical cannabis on the gridiron, but are they just blowing smoke?

Chronic traumatic encephalopathy (CTE) is a name culled from the Greek that means exactly what it says: a disease of the brain caused by injuries sustained over a long period of time. The symptoms include memory loss, confusion, poor impulse control, emotional volatility, depression, suicidal thoughts, and dementia. On the upside, it can take years or even decades after injury for these to show.

CTE is extraordinarily rare for anyone who doesn't get their head repeatedly knocked around. In fact, for years it was assumed to be limited to boxers, and it was even called *dementia pugilistica*, "boxing madness." But that was before 2003, when an autopsy on deceased Pittsburgh Steeler center Mike Webster found unmistakable signs of CTE.

A subsequent wave of suicides among retired NFL players who had been displaying symptoms of CTE skyrocketed awareness of the disease. Strident denial by the NFL only stirred the fires of controversy. From the flames have emerged a *Frontline* [documentary](#) (later made into a book) and—the ultimate sign of public concern—a very serious [Will Smith movie](#).

Outrage over the hidden epidemic of sports concussions seems to have quelled, and we see fewer editorials like [this one](#) questioning the future of aggressive sports like football in high school. Nevertheless, the underlying problem is real: Last year, researchers at Boston University announced that they had found CTE in the brains of [96 percent of the NFL players](#) they have examined. When they included high school, college, and semi-pro players, that number only dipped to 79 percent.

If the US is prepared to accept CTE as the cost of playing sports, the question then shifts to: How can we best lessen its effect?

I bet you can't have guessed that, for some, the answer is cannabis.

The [Gridiron Cannabis Coalition](#), a coalition of retired athletes who made their reputation on the gridiron, plus Snoop Dogg (because, cannabis), is lobbying hard to get the NFL to drop its ban on marijuana. Their medical argument is not only that cannabis can treat the symptoms of CTE—in particular pain and emotional disturbances—but that it can actually protect the brain against traumatic injury.

The first point is a slam dunk. Er, touchdown. Not only is marijuana an effective (if mild) [pain reliever](#), it has the even better effect of helping [patients taper their use of opioids](#), which have far worse side effects than weed.

On that second point, however, Snoop and his doobie brothers are getting ahead of the science. While there is evidence that cannabis can provide relief for neurological conditions such as epilepsy, Parkinson's, even Alzheimer's, we're less sure that it can actually prevent damage when administered in advance, and there's no literature that specifically addresses CTE.

However, that work is underway. Researchers at Temple University are collaborating with a private pharmaceutical firm to see if a newly-formulated synthetic CBD, which they call “50 times more potent [and] 500-fold safer” than the real deal, can protect the brain against traumatic blows. Any results, though, will likely be years away.

Cannabis and Mad Cow Disease

Sept. 13, 2016

Richard Faulk

Good News: Cannabis may kill it. Bad News: Once you display symptoms, it's too late.

Today's flashback takes you to 1995, the year when the noontime luster of Cool Britannia (embodied in this classic Pulp track) was dimmed by the shadow of mad cow disease, which had claimed its first ever human victim, just outside of London.

Creutzfeldt-Jakob disease, which, as anyone in the mid-'90s could have told you, is the more correct name for the human variant of mad cow, is part of the family of transmissible spongiform encephalopathies (TSE), which attack the brain, rendering it into a soggy, tattered mess, like an old kitchen sponge. TSEs can take years to present symptoms, but once they do the poor victim will experience memory loss, hallucinations, tics and seizures, growing dementia, then death.

You catch TSEs from eating brains or nerves. Creutzfeldt-Jakob comes from cows. If you eat a person, you might catch a kuru, like New Zealand's Foré people did back in the early 20th century. (One more reason not to eat a person—even if it's consensual.)

There is no treatment for TSEs. Once they get going, there's no stopping them. The good news is that it is very difficult to catch them. Just don't eat people. (Did we already tell you that?) And don't eat cervelle de veau from an infected animal. (Steak tartare, however, is A-OK.)

We tend to think of diseases as invading organisms that want to feast on our insides or, at the least, make a home of them. But TSEs are caused by prions, which are simply proteins. Proteins with bad attitudes. To function properly, proteins have to be folded in very precise ways. But prions are slovenly, and their mere presence gives other proteins...*ideas*. Like that troupe of mean girls your daughter fell in with in junior high who introduced her to fashion that made you feel prudish and music that made you feel old, prions entice the proteins in your brain to twist themselves in strange and provocative ways.

A corkscrew will work whether it twists clockwise or the reverse. Not so our brains. When the proteins in our head all twist the wrong way, we don't suddenly develop Genghis Khan goatees and a taste for mayhem, like anti-world Spock does. No indeed. We look just the same; we just end up dead.

A growing body of literature shows that cannabis can protect the brain against degenerative diseases, such as Parkinson's and Alzheimer's. However, only one study so far has tested its effect on TSE. Researchers observed the effect of the cannabinoid CBD on both tissue samples and living mice that had been infected by a TSE called scrapie, that typically affects sheep. The results showed that CBD both slowed the migration of prions into the brain and extended life for mice already infected, in each case by about 6 percent. Six percent is hardly a figure for celebration, but considering that there is absolutely no current treatment, it's better than chopped liver.

However, there is one big problem: CBD is most effective against prions when it taken directly after infection—but TSEs can take years to manifest. In addition to that, there are myriad “little” problems, including the fact that scrapie is not Creutzfeldt-Jakob and mice are not people.

If you'd like to stroll into the scientific weeds of this subject, read the [initial study](#) and this [more accessible critique](#) by the Prion Alliance.

Cannabis and Sleep Apnea

Aug 24, 2016

Richard Faulk

Cannabinoids may counteract this common sleep disorder and help you get the z-z-z-s you need.

Sleep apnea is a commonish disorder that shatters restful sleep with interruptions in breathing that can last anywhere from seconds to even minutes. Severe cases might present up to thirty such disruptions per minute.

To observers, sleep apnea can look like a cartoonish snoring fit, complete with trumpet-like noises, sputtering, and a sudden eruption into semi-wakefulness amid mutterings of “wha-a, wha-a, what...” Appropriately, it affects primarily the male, the overweight, and the no-longer young. (Think Homer Simpson. Or, for that matter, your author.)

Throat surgery can be a treatment option. A less drastic but far more awkward option is continuous positive airway pressure (or CPAP), which is provided by a plastic ventilation tube attached to a face mask at one end and an air pump on the other. The whole is evocative less of a restful night's sleep than of Darth Vader.

But, take heart, apneacs, there may be hope.

A University of Illinois study from 2002 found that injections of THC (from cannabis) and oleamide (a cannabinoid produced by us animals) suppressed serotonin—a neurotransmitter known to exacerbate sleep apnea—and thus reduced sleep disruption in lab rats by up to 58 percent.

Good for rodents, you say. But what about us humans?

Good question!

A proof of concept trial from 2013, performed by some of the same U. Illinois researchers, showed that THC had the same apnea-quelling effects in humans and that it was well tolerated. “Proof of concept” means that the findings may not actually be valid, but they are strong enough to justify a full-scale clinical trial—which is currently in the works.

And there's more: Yet another study from U. Illinois, this time from 2014, zeroed in on how the cannabinoid treatment might be working. (This time they decided to use rats.) They injected THC into the nodose ganglia (whisper that softly to yourself—really, try it), a nerve in the back of the throat. The THC countered the serotonin, as they'd already seen, but it also “increase[d] phasic activation of the genioglossus.” That means, the THC tightened that muscle in the back of your mouth that helps you stick out your tongue. It is also the muscle that, when relaxed, causes you to snore.

So, does all this research translate to “Hello, bedtime toke; goodbye, sleep apnea”?

Well...not just yet: A 20015 research review concludes that cannabinoids “hold promise”—but CPAP “will probably remain the gold standard for the foreseeable future.”