

Retired National Football League Players are Not at Greater Risk for Suicide

Grant L. Iverson*

Department of Physical Medicine and Rehabilitation, Harvard Medical School; Spaulding Rehabilitation Hospital; MassGeneral Hospital for Children Sport Concussion Program; & Home Base, A Red Sox Foundation and Massachusetts General Hospital Program, Boston, MA 02129, USA

*Corresponding author at: Center for Health and Rehabilitation Research, Department of Physical Medicine & Rehabilitation, Harvard Medical School, 79/96 Thirteenth Street, Charlestown Navy Yard, Charlestown, MA, 02129, USA. Tel: +617-952-6194.

E-mail address: giverson@mgh.harvard.edu

Editorial Decision 25 April 2019; Accepted 29 April 2019

Abstract

Objective: Some researchers have claimed that former National Football League (NFL) players are at increased risk for suicide as a clinical feature of chronic traumatic encephalopathy (CTE). This review examines the literature on risk for suicide in former professional football players, and the association between suicide and CTE.

Method: A narrative review of the literature published between 1928 and 2018.

Results: Between 1928 and 2009, suicide was not considered to be a clinical feature of CTE in the literature. The best available evidence from epidemiological studies suggests that former NFL football players are at *lesser risk* for suicide, not greater risk, compared to men in the general population. However, surveys have revealed that a substantial minority of former NFL players have depression and other mental health problems, chronic pain and opioid use is relatively common, and those with depression and chronic pain also have greater life stress and financial difficulties. That minority would be at increased risk for suicidal thoughts and behaviors.

Conclusions: Researchers and clinicians are encouraged to be cautious and circumspect when considering the clinical presentation of former athletes, and to not assume that depression and suicidality are caused by specific types of neuropathology. This represents a reductionistic and Procrustean view. Some former football players have mental health problems, but it should not be assumed uncritically that the underlying cause is an inexorably progressive neurodegenerative disease. Providing evidence-informed and evidence-supported treatments for depression and suicidality might reduce suffering and improve their functioning.

Keywords: Athletes; suicidal ideation; suicide; concussion; chronic traumatic encephalopathy

Introduction

There appears to be a fundamental misunderstanding in the media and the published medical literature that former National Football League (NFL) players are at increased risk for suicide. This misunderstanding might have arisen, in part, because suicide has recently been claimed to be a clinical feature of chronic traumatic encephalopathy (CTE) (Baugh et al., 2012; Gavett, Stern, & McKee, 2011b; McKee et al., 2013; Omalu, 2014; Omalu, Bailes, Hammers, & Fitzsimmons, 2010a; Stern et al., 2011a, 2013). Between 1928 and 2009, however, suicide was *not* discussed as a cause of death or a clinical feature of CTE in the published medical literature (McKee et al., 2009; Roberts, 1969; Victoroff, 2013). Moreover, the best available evidence suggests that former NFL football players are at *lesser risk* for suicide, not greater risk, compared to men in the general population (Baron, Hein, Lehman, & Gersic, 2012; Lehman, Hein, & Gersic, 2016). This review examines the literature on risk for suicide in former professional football players, and the association between suicide and CTE.

The clinical features of CTE

During the 20th century, CTE was known by several names, including “punch drunk” (Critchley, 1949; Martland, 1928), traumatic encephalopathy of boxers (Grahmann & Ule, 1957), chronic progressive traumatic encephalopathy (Critchley, 1957), and dementia pugilistica (Grahmann & Ule, 1957; Millspaugh, 1937). CTE was considered to be a neurological disorder that affected a subgroup of current and former high exposure boxers (Martland, 1928; Roberts, 1969). Cumulative and chronic brain injury (Jordan, 2000), often manifesting in neurological signs and deficits while the boxer was still active and competing (Martland, 1928; Roberts, 1969), was believed to be the underlying neuropathology (Corsellis, Bruton, & Freeman-Browne, 1973). The syndrome was characterized by varying degrees of slurred or dysarthric speech, abnormalities in gait and coordination, Parkinsonism, cognitive deficits, and dementia (Jordan, 2000; Martland, 1928; Roberts, 1969). The brain damage documented in these boxers was often described as “pyramidal” (e.g., abnormal reflexes) and “extrapyramidal” (tremors and Parkinsonian gait disturbance), and both were noted to be present in some young boxers, in their 20 s and 30 s, before they retired from the sport (Corsellis et al., 1973; Critchley, 1957; Harvey & Davis, 1974; Jedlinski, Gatarski, & Szymusik, 1970; Johnson, 1969; Mawdsley & Ferguson, 1963; Parker, 1934; Roberts, 1969; Sercl & Jaros, 1962). It was also well recognized that boxers with chronic brain damage exhibited other psychiatric and neurological problems that co-occurred with the more core characteristic features (Johnson, 1969; Mawdsley & Ferguson, 1963; Payne, 1968; Roberts, 1969). Throughout the 20th century, it was not clear the extent to which CTE in boxers was static, progressive, or whether its course represented two distinct conditions (Carrol, 1936; Courville, 1962; Critchley, 1949; Grahmann & Ule, 1957; Johnson, 1969; Martland, 1928; Mendez, 1995; Parker, 1934; Roberts, 1969; Victoroff, 2013). In fact, researchers emphasized that the chronic brain damage in boxers did not present as a “single entity” (Courville, 1962), that boxers presented with “variable clinical features” (Parker, 1934), and there were several syndromes (Critchley, 1949, 1957; Johnson, 1969).

At present, there are no agreed upon *clinical* diagnostic criteria for CTE, although several sets of criteria have been proposed in the past few years (Jordan, 2013; Montenigro et al., 2014; Reams et al., 2016; Victoroff, 2013). CTE is a post-mortem neuropathological diagnosis (McKee et al., 2013, 2016). The defining neuropathological feature of CTE is the accumulation of hyperphosphorylated tau (p-tau), in a patchy distribution at the depths of the cortical sulci (McKee et al., 2013, 2016). P-tau accumulates in the brain in normal aging and in neurodegenerative diseases (Alonso et al., 2018; Braak, Thal, Ghebremedhin, & Del Tredici, 2011; Crary et al., 2014; Kovacs et al., 2016; Morsch, Simon, & Coleman, 1999; von Bergen, Barghorn, Biernat, Mandelkow, & Mandelkow, 2005), but it is not believed to accumulate in a *patchy distribution* in the depths of sulci around small vessels in association with aging or other diseases (McKee et al., 2013, 2016). It has been asserted that CTE pathology is only found in people (i.e., mostly boxers, football players, and contact sport athletes) who have been exposed to repetitive neurotrauma (Baugh et al., 2012; Baugh, Robbins, Stern, & McKee, 2014; McKee, Daneshvar, Alvarez, & Stein, 2014; Mez, Stern, & McKee, 2013; Stern et al., 2011b, 2013). There is evidence, however, that the neuropathology described as unique to CTE may not be completely unique because it has been identified in some people with no known exposure to repetitive neurotrauma, in association with (i) substance abuse (Noy, Krawitz, & Del Bigio, 2016), (ii) temporal lobe epilepsy (Puvanna et al., 2016), (iii) amyotrophic lateral sclerosis (Fournier, Gearing, Upadhyayula, Klein, & Glass, 2015; Gao et al., 2017), (iv) multiple system atrophy (Koga, Dickson, & Bieniek, 2016), and (v) other neurodegenerative diseases (Ling et al., 2015).

A modern theory of CTE, emphasized in the past few years, is that it is a *distinct* (Baugh et al., 2012, 2014; Gavett et al., 2011b; McKee et al., 2009; Mez et al., 2013) and *unique* (Baugh et al., 2014) neurodegenerative disease (Baugh et al., 2012, 2014; Gavett et al., 2011a; McKee et al., 2014; Mez et al., 2013), in the family of tauopathies (McKee et al., 2013), although the evidence to support this theory is both limited and mixed. In recent years, a major change in conceptualizing CTE has been to assert that a broad range of *psychiatric* and *psychosocial problems* are common clinical features, such as (i) depression and anxiety (Baugh et al., 2012; McKee et al., 2013; Omalu et al., 2011); (ii) substance abuse (Baugh et al., 2012; Montenigro et al., 2014; Omalu et al., 2011); (iii) suicidal thinking and completed suicide (Baugh et al., 2012; Gavett et al., 2011b; McKee et al., 2013; Omalu et al., 2010a; Omalu, 2014; Stern et al., 2011a, 2013); (iv) poor financial decisions and bankruptcy (Omalu et al., 2011), (v) gambling (Montenigro et al., 2014), and (vi) marital problems, separation, and divorce (Omalu, 2014). Some authors have even stated that generalized body aches and pain (Omalu et al., 2011) and insomnia (Omalu, 2014) are clinical features of CTE. Preliminary research criteria for “traumatic encephalopathy syndrome” (Montenigro et al., 2014) include three proposed phenotypic expressions of CTE, (i) “cognitive”, (ii) “behavioral” (i.e., anger dyscontrol), and (iii) “mood” (i.e., depression or hopelessness). Two supportive features must also be present [i.e., impulsivity, anxiety, apathy, paranoia, suicidality, headache, motor signs, a progressive clinical course, or a delayed onset of symptoms (for example after retirement from sport)]. The current emphasis on depression as a clinical phenotype, and suicidality as a “supportive feature,” represents a major recent change in the conceptualization of CTE.

Suicide and CTE

As noted above, between 1928 and 2009, suicidal ideation and suicide were *not* considered to be clinical features of CTE in the published medical literature (McKee et al., 2009; Roberts, 1969; Victoroff, 2013). For example, in 1969, Roberts published a book relating to brain damage and CTE based on clinical examinations of retired professional boxers (Roberts, 1969). He did not discuss suicidality or completed suicide as a clinical feature. Roberts began with a list of 16,781 people and obtained an age-stratified random sample of 250. Of those, 16 had died, and there were no confirmed cases of suicide, although there was one suspicious death associated with carbon monoxide poisoning (the details of this were not available). In the first review of all presumed cases of CTE, by McKee and colleagues, suicidality and suicide were not considered features of the syndrome (McKee et al., 2009). In that seminal review published in 2009, the clinical features in the previously published case studies were carefully documented—and suicidality was not mentioned.

Similarly, Victoroff (2013) carefully examined the clinical features of 97 cases published between 1928 and 2010 (92 were boxers), and he found that psychiatric problems were fairly common, but suicidal behavior was extremely uncommon. Victoroff identified a single case in the literature with “suicidal behavior” (noted in Table 3 of his article). The citation for this case was not provided. In my review of the literature of all possible cases of CTE from the 20th century, I also found only a single case. Case #9 in the seminal neuropathology paper on brain damage and traumatic encephalopathy in boxers, published in 1973 by Corsellis and colleagues, was noted to have attempted suicide at the age of 65, prior to be admitted to a psychiatric hospital. Ironically, it was recently discovered that this particular case did not actually have CTE, based on the modern consensus criteria for defining the neuropathology of CTE (McKee et al., 2016). Goldfinger and colleagues recently re-examined tissue from his brain and reported that he suffered from Lewy Body dementia and he also had aging-related tau astrogliopathy (Goldfinger et al., 2018; Kovacs et al., 2016).

How did suicide become an asserted clinical feature of CTE?

In 2010, suicidality and suicide were introduced as clinical features in the published literature by Omalu, Hamilton, Kambh, DeKosky, and Bailes (2010c) (although researchers discussed this in the media prior to 2010). This assertion appears to have arisen because two of the three autopsy case studies of former NFL players examined by Omalu had suicide as their cause of death. Omalu and colleagues did not cite any published studies or other scientific evidence to support this opinion. Omalu and colleagues had also published an autopsy case study of a former wrestler (Omalu, Fitzsimmons, Hammers, & Bailes, 2010b) who allegedly killed his wife and son, and then completed suicide by hanging, but this case was not presented in their 2010 article as evidence of suicide being a clinical feature of CTE. After 2010, researchers from Boston University and Omalu and colleagues repeatedly stated that suicidality and suicide are common clinical features of CTE as a *fait accompli* (Baugh et al., 2012; Gavett et al., 2011b; McKee et al., 2013; Omalu et al., 2010a; Omalu, 2014; Stern et al., 2011a, 2013). In general, however, they cited limited or no scientific evidence for that opinion. For example, in a 2011 review paper on CTE, Gavett et al. (2011a) stated that suicidality was common. They cited McKee et al. (2009) and Omalu et al. (2011) as evidence for that opinion. However, as previously noted, the McKee et al. (2009) review of the world literature on CTE did not conclude that suicidality or suicide were clinical features. Omalu et al. (2011) reported that suicidality and completed suicide was present in many of their post-mortem case series (e.g., 5 of 17 cases had suicide as their cause of death). In 2012, Baugh et al. (2012) cited the above-mentioned 2011 review by Gavett et al. (2011a) as evidence that suicidality was common in CTE. In the introduction to their large case series published in 2013 (McKee et al., 2013), McKee and colleagues reported that CTE was associated with “heightened suicidality” and cited their 2009 review paper (McKee et al., 2009) as the only source. Therefore, during this time period, 2010–2013, the cited evidence for suicidality and suicide being a clinical feature of CTE appears to be based on both proof by assertion and circular reasoning. The authors did not cite cross-sectional, prospective, or epidemiological studies showing an association between suicide, football, or CTE.

The case series by McKee and colleagues, published in 2013 (McKee et al., 2013), is sometimes cited as evidence that suicidality and suicide are clinical features of CTE (Montenigro et al., 2014). If one examines the causes of death in former athletes in that post-mortem case series, listed in a table in that article (McKee et al., 2013), those with CTE pathology were significantly *less likely* to have completed suicide than those with no neuropathology of CTE (Fig. 1). This also illustrates the ascertainment bias associated with highly selected autopsy case series, and the over-representation of suicide as a cause of death in these case series.

Montenigro and colleagues published research criteria for the clinical diagnosis of CTE (using the new term “traumatic encephalopathy syndrome”) in 2014 (Montenigro et al., 2014). They reported that the criteria were based on their review of 202 published cases of CTE, and they summarized the clinical features of CTE in Table 1 of that article. Five studies were

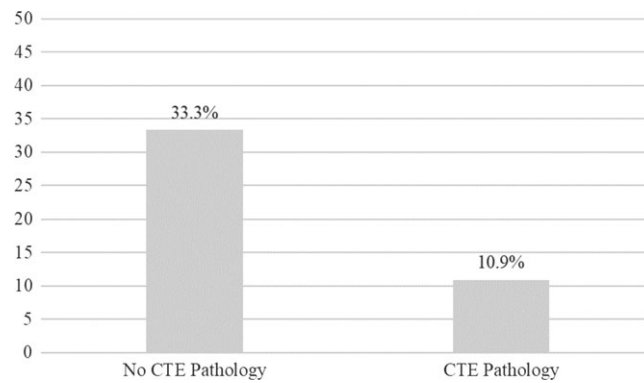


Fig. 1. Rate of suicide in those with and without CTE pathology in the [McKee and colleagues \(2013\)](#) post-mortem case series. *Note:* The rate of suicide as the manner of death in a large post-mortem case series ([McKee et al., 2013](#)) was significantly lower in those identified as having CTE pathology (7/64; 10.9%) compared to those with no CTE pathology [6/18, 33.3%; $\chi^2(1,82) = 5.28, p = 0.022$].

Table 1. Biopsychosocial risk factors for suicide in men

Risk factors

- Childhood adversities, such as physical abuse, sexual abuse, or family violence [Bruffaerts and colleagues \(2010\)](#)
- Interpersonal or family conflict [Foster \(2011\)](#)
- Depression [Hawton, Casanas, Haw, and Saunders \(2013\)](#)
- Substance abuse [Nock, Hwang, Sampson, and Kessler \(2010\)](#); [Wilcox, Conner, and Caine \(2004\)](#)
- Major interpersonal stressful life events in those with alcohol problems [Conner and colleagues \(2012\)](#)
- Financial difficulties [Richardson, Elliott, and Roberts \(2013\)](#)
- Physical illnesses [Scott and colleagues \(2010\)](#) and poor general health [Conwell and colleagues \(2010\)](#)
- Chronic pain [Hassett, Aquino, and Ilgen \(2014\)](#); [Hooley, Franklin, and Nock \(2014\)](#); [Tang and Crane \(2006\)](#)
- Traumatic brain injury [Bahraïni, Simpson, Brenner, Hoffberg, and Schneider \(2013\)](#); [Fralick and colleagues \(2018\)](#); [Madsen and colleagues \(2018\)](#)
- Personality disorders [Arsenault-Lapierre, Kim, and Turecki \(2004\)](#)
- Impulsivity and aggression [Conner, Conwell, Duberstein, and Eberly \(2004\)](#); [Gvion and Apter \(2011\)](#); [McGirr and Turecki \(2007\)](#)
- Limited social connectedness [Fassberg and colleagues \(2012\)](#)
- Hopelessness [Conner, Duberstein, Conwell, Seidnitz, and Caine \(2001\)](#)

High risk patterns of thinking or cognitive states associated with suicidal ideation and behavior (see [Brown and Green \(2014\)](#) for a review)

- Hopelessness [Beck, Brown, Berchick, Stewart, and Steer \(1990\)](#); [Beck, Steer, Kovacs, and Garrison \(1985\)](#)
- Problem-solving deficits, such as difficulty conceptualizing adaptive solutions to life problems [McAuliffe, Keeley, and Corcoran \(2002\)](#)
- Perceived burdensomeness and thwarted belongingness (a belief that one does not have meaningful interpersonal relationships) [Van Orden, Merrill, and Joiner \(2005\)](#)

Source: The information presented in Table 1 was adapted mostly from a review paper by [Iverson \(2016\)](#).

cited as providing support for suicidality as a clinical feature ([McKee et al., 2013](#); [Omalu et al., 2005, 2006, 2010b](#); [Payne, 1968](#)). Three of the five studies, however, clearly do not support this opinion ([Omalu et al., 2005, 2010b](#); [Payne, 1968](#)). For example, a study by Payne published in 1968 was cited; it reported the clinical features of six former boxers ([Payne, 1968](#)). Suicide and suicidality were not mentioned in that article as being present in any of the six former boxers. Similarly, the first case of CTE in a former NFL player was cited, published by Omalu and colleagues in 2005 ([Omalu et al., 2005](#)). Omalu and colleagues did not discuss suicide or suicidality as being a clinical feature in that article, however. In 2006, Omalu and colleagues presented a second case of CTE in a former NFL player, and he had an extensive history of suicidal ideation, attempts, and he died by suicide ([Omalu et al., 2006](#)). The 2010 article by Omalu and colleagues was also cited ([Omalu et al., 2010b](#)); it reported on a case of a former wrestler who allegedly killed his wife and son and then hung himself. However, Omalu and colleagues did not attribute his suicide to CTE in that article. Therefore, with the exception of the [McKee et al.](#) case series published in 2013 ([McKee et al., 2013](#)), and the single case published by Omalu and colleagues ([Omalu et al., 2006](#)), the other studies cited by [Montenegro and colleagues](#) as supportive of suicidality being a clinical feature were actually not supportive based on what was written by the original authors.

Suicide in former NFL players

In a study published in *JAMA* in 2017, it was reported that 99% of current or former NFL players who have died and undergone post-mortem examinations of their brains have small, medium, or large amounts of neuropathology suggestive of

CTE (Mez et al., 2017). If CTE is very common in former NFL players, and suicide is a clinical feature of CTE, then NFL players should be at increased risk for suicide—because CTE would confer risk separately from all the other known risk factors for suicide in men. However, according to an epidemiological study reporting causes of death in former NFL players, nine completed suicide over the course of 47 years (1960–07), a rate that is *less than half* of what is expected from men in the general population (Baron et al., 2012). In that cohort of former NFL players, 334 had died and nine had intentional self-harm listed as their cause of death (i.e., 2.7%). Therefore, based on that epidemiological study, before 2008, the only published evidence indicated that former NFL players were at substantially *lower risk* for suicide than men in the general population. Lehman and colleagues conducted an epidemiological study focused entirely on the rate of suicide in former NFL players from 1960 through 2013 (Lehman et al., 2016). They replicated and extended the previous finding indicating that the suicide rate was significantly less than would be expected in comparison with the United States population (standardized mortality ratio = 0.47; 95% CI, 0.24–0.82). In that expanded cohort of former NFL players, 537 had died and 12 had intentional self-harm listed as their cause of death (i.e., 2.2%).

Using an internet search strategy, Webner and Iverson identified 26 men who were current or former professional football players who died by suicide between 1920 and 2015 (Webner & Iverson, 2016). More than half of the deaths since 1920 occurred between 2000 and 2015 (58.7%), and a large percentage occurred between 2009 and 2015 (42.3%). Specifically, the number of suicides between 1920 and the end of 2008 was 15 (in 89 years), and the number between 2009 and the end of 2015 was 11 (Webner & Iverson, 2016). In 2012 alone, six deaths by suicide were identified—likely for diverse and multifactorial reasons. Between 2013 and 2016, authors of four review papers concluded that the scientific evidence to support the assertion that suicide is a clinical feature of CTE is lacking and inconclusive (Iverson, 2014, 2016; Maroon et al., 2015; Wortzel, Shura, & Brenner, 2013). Webner and Iverson raised the possibility that the extensive media coverage on this topic might have adverse psychological effects on some retired athletes (Webner & Iverson, 2016). It is possible that repeated exposure to news stories might elicit or reinforce suicidal ideation in some at-risk athletes, such as those with marital and family problems, financial problems, substance abuse difficulties, and depression. There has been a longstanding interest in the influence of the media on suicidal behavior (Sisask & Varnik, 2012; Yang et al., 2013) and in “contagion” (Cheng, Li, Silenzio, & Caine, 2014), and research is needed to examine whether the media coverage on contact sports, CTE, and suicide could be contributing to psychological distress in some former athletes. Research is also needed to determine if there is any association between media coverage of suicides in current and former professional athletes and suicides in the general population. A recent study reported an association between the famous actor Robin Williams’ death by suicide and an increase in suicides in the US general population, particularly in men, especially men between the ages of 30–44, in the months following his death (Fink, Santaella-Tenorio, & Keyes, 2018).

Are former NFL players at risk for suicidal ideation?

Some former NFL players likely have episodic or frequent suicidal ideation. Suicidal ideation is a cardinal diagnostic feature of major depressive disorder (American Psychiatric Association, 1994). Surveys have revealed that a substantial minority of former NFL players have depression and other mental health problems (Gonzalez, Tarraf, Whitfield, & Vega, 2010; Schwenk, Gorenflo, Dopp, & Hipple, 2007; Weir, Jackson, & Sonnega, 2009), chronic pain and opioid use is relatively common (Cottler et al., 2011), and those with depression and chronic pain also have greater life stress and financial difficulties (Schwenk et al., 2007). In non-athlete populations, people seeking treatment for chronic pain have high rates of comorbid depression (Banks & Kerns, 1996), and people with chronic pain are at increased risk for suicidal ideation (Ilgen, Zivin, McCammon, & Valenstein, 2008) and for suicide (Tang & Crane, 2006). Therefore, without question, there is a subgroup of former NFL players who suffer from depression who likely have suicidal thoughts associated with their mental health problems.

Recently, Mez and colleagues reported that suicidality was very common in former football players who had CTE pathology discovered in their brains after death (Mez et al., 2017). In former players who had mild CTE pathology in their brains (Stage I and Stage II), depressive symptoms were present in 67%, hopelessness in 69%, suicidality in 56%, and 27% died by suicide (Mez et al., 2017). However, it seems unlikely that the small amount of pathology seen in Stage I CTE drives complex changes in behavior underlying suicidality and suicide. Stage I CTE has been described in the literature as being pre-clinical and asymptomatic in some people (Baugh et al., 2012; Daneshvar, Goldstein, Kiernan, Stein, & McKee, 2015; Gavett et al., 2011b; McKee et al., 2014; Stern et al., 2013). Therefore, it is important to consider other causes of depression and suicidality in former football players.

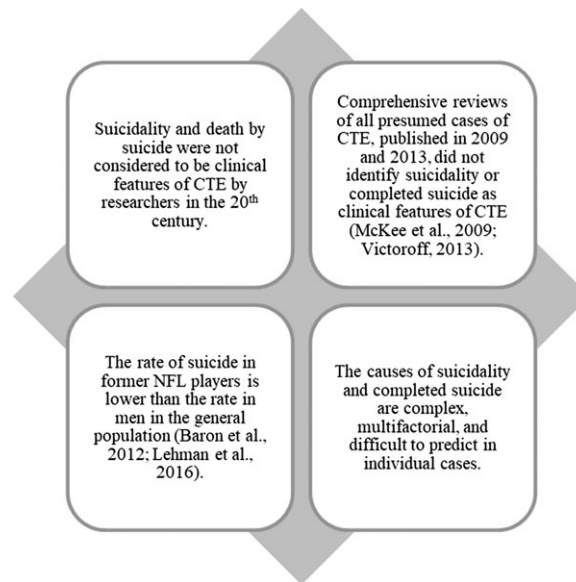


Fig. 2. Key points

Suicide in the general population

In the general population, the highest rates of suicide occur among those in the following age groups: 45–54, 55–64, and 75–84 (18.9, 15.9, and 17.0/100,000, respectively), and men are three times more likely to complete suicide than women (Karch, Logan, Patel, Centers for Disease, & Prevention, 2011). Suicide is a leading cause of death in young people. In 2008, the incidence of suicide in people between the ages of 15 and 19 was estimated to be 8.1/100,000, and for those between 20 and 24 it was 12.7/100,000 (Karch et al., 2011). There are many biopsychosocial risk factors for depression and suicidality, and the causes of completed suicide are usually multifactorial and difficult to predict in individual cases. According to a CDC report examining suicides in 2008, common precipitating events were depression, a life crisis, intimate partner problems, physical health problems, occupational or financial problems, and criminal or legal proceedings (Karch et al., 2011). There is an enormous published literature on suicidal ideation, behavior, and completed suicide. Some risk factors relevant to middle-aged men are presented in Table 1.

Conclusions and clinical implications

Prior to 2005, depression and suicidality were not considered to be core or defining clinical features of CTE (Fig. 2). During the 20th century, depression occasionally was documented in the case histories of some boxers who had chronic brain damage and presumed CTE (Critchley, 1949, 1957; Johnson, 1969; Mawdsley & Ferguson, 1963; Payne, 1968). When documented, it was reported to occur in association with an obvious neurological disorder, not as an isolated or primary disorder. In the past few years, however, there has been a much greater emphasis on mental health problems, such as depression and anxiety (Baugh et al., 2012; McKee et al., 2013; Montenigro et al., 2014; Omalu et al., 2011), being possible core clinical features—with suicidality believed to be a “supportive feature” (Montenigro et al., 2014). Some former NFL players who have died by suicide, and whose brains were donated for research, have been found to have the neuropathology characteristic of CTE (McKee et al., 2013; Mez et al., 2017). Overall, however, as a group, former NFL players are at lesser risk for suicide than men in the general population (Baron et al., 2012; Lehman et al., 2016). Researchers and clinicians are encouraged to be cautious and circumspect when considering the clinical presentation of former athletes, and to not assume uncritically that depression and suicidality are caused by specific CTE neuropathology. This represents a Procrustean and reductionist view.

Nonetheless, a subgroup of former football players experience depression (Gonzalez et al., 2010; Schwenk et al., 2007; Weir et al., 2009) and they likely have episodic or frequent suicidal thoughts. It is reasonable to assume that retired football players with chronic pain, depression, substance abuse problems, life stress, and financial difficulty will be at increased risk for suicidal thoughts and behaviors—regardless of whether they have small, medium, or large amounts of CTE pathology in their brains. It has not been established that CTE neuropathology represents the substrate of an inexorably progressive

neurodegenerative disease, or that depression is a core clinical phenotype of this neurodegenerative disease. Therefore, the clinical diagnosis of CTE in people with depression, given its dire prognosis, might have iatrogenic effects and adversely affect treatment. Those experiencing depression, distorted patterns of thinking, and substance abuse problems might be particularly vulnerable. Importantly, however, there are effective treatment options available for depression, suicidality, anxiety, headaches, chronic pain, sleep problems, substance abuse, gambling, and life stress. These are all problems that occur in some men in the general population, they likely occur in a minority of former football players, and they certainly are not unique to people who have some neuropathology of CTE. Providing evidence-informed and evidence-supported treatments might reduce suffering and improve functioning in former athletes with depression.

Conflict of interest

None declared.

Disclosures

Grant Iverson, Ph.D. has been reimbursed by the government, professional scientific bodies, and commercial organizations for discussing or presenting research relating to mild TBI and sport-related concussion at meetings, scientific conferences, and symposiums. He has a clinical and consulting practice in forensic neuropsychology involving individuals who have sustained mild TBIs (including athletes). He serves as a scientific advisor for BioDirection, Inc., Sway Operations, LLC, and Highmark, Inc. He has received research funding from several test publishing companies, including ImpACT Applications, Inc., CNS Vital Signs, and Psychological Assessment Resources (PAR, Inc.). He has received research funding as a principal investigator from the National Football League, and salary support as a collaborator from the Harvard Integrated Program to Protect and Improve the Health of National Football League Players Association Members. He acknowledges unrestricted philanthropic support from ImpACT Applications, Inc., the Mooney-Reed Charitable Foundation, and the Spaulding Research Institute.

References

- Alonso, A. D., Cohen, L. S., Corbo, C., Morozova, V., Elldrissi, A., Phillips, G., et al. (2018). Hyperphosphorylation of tau associates with changes in its function beyond microtubule stability. *Frontiers in Cellular Neuroscience*, *12*, 338. doi:10.3389/fncel.2018.00338.
- American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: Author.
- Arsenault-Lapierre, G., Kim, C., & Turecki, G. (2004). Psychiatric diagnoses in 3275 suicides: a meta-analysis. *BMC Psychiatry*, *4*, 37. doi:10.1186/1471-244X-4-37.
- Bahraini, N. H., Simpson, G. K., Brenner, L. A., Hoffberg, A. S., & Schneider, A. L. (2013). Suicidal ideation and behaviours after traumatic brain injury: a systematic review. *Brain Impairment*, *14*, 92–112.
- Banks, S. M., & Kerns, R. D. (1996). Explaining high rates of depression in chronic pain: a diathesis-stress framework. *Psychological Bulletin*, *119*, 95–110.
- Baron, S. L., Hein, M. J., Lehman, E., & Gersic, C. M. (2012). Body mass index, playing position, race, and the cardiovascular mortality of retired professional football players. *American Journal of Cardiology*, *109*, 889–896. doi:10.1016/j.amjcard.2011.10.050.
- Baugh, C. M., Robbins, C. A., Stern, R. A., & McKee, A. C. (2014). Current understanding of chronic traumatic encephalopathy. *Current Treatment Options in Neurology*, *16*, 306. doi:10.1007/s11940-014-0306-5.
- Baugh, C. M., Stamm, J. M., Riley, D. O., Gavett, B. E., Shenton, M. E., Lin, A., et al. (2012). Chronic traumatic encephalopathy: neurodegeneration following repetitive concussive and subconcussive brain trauma. *Brain Imaging and Behavior*, *6*, 244–254. doi:10.1007/s11682-012-9164-5.
- Beck, A. T., Brown, G., Berchick, R. J., Stewart, B. L., & Steer, R. A. (1990). Relationship between hopelessness and ultimate suicide: a replication with psychiatric outpatients. *American Journal of Psychiatry*, *147*, 190–195.
- Beck, A. T., Steer, R. A., Kovacs, M., & Garrison, B. (1985). Hopelessness and eventual suicide: a 10-year prospective study of patients hospitalized with suicidal ideation. *American Journal of Psychiatry*, *142*, 559–563.
- Braak, H., Thal, D. R., Ghebremedhin, E., & Del Tredici, K. (2011). Stages of the pathologic process in Alzheimer disease: age categories from 1 to 100 years. *Journal of Neuropathology and Experimental Neurology*, *70*, 960–969. doi:10.1097/NEN.0b013e318232a379.
- Brown, G. K., & Green, K. L. (2014). High-risk cognitive states. In Koslow S. H., Ruiz P., & Nemeroff C. B. (Eds.), *A Concise Guide to Understanding Suicide: Epidemiology, Pathophysiology and Prevention* (pp. 42–51). Cambridge, UK: Cambridge University Press.
- Bruffaerts, R., Demyttenaere, K., Borges, G., Haro, J. M., Chiu, W. T., Hwang, I., et al. (2010). Childhood adversities as risk factors for onset and persistence of suicidal behaviour. *British Journal of Psychiatry*, *197*, 20–27. doi:10.1192/bjp.bp.109.074716.
- Carroll, E. J. (1936). Punch-drunken. *American Journal of the Medical Sciences*, *191*, 706–712.
- Cheng, Q., Li, H., Silenzio, V., & Caine, E. D. (2014). Suicide contagion: a systematic review of definitions and research utility. *PLoS One*, *9*, e108724. doi:10.1371/journal.pone.0108724.
- Conner, K. R., Conwell, Y., Duberstein, P. R., & Eberly, S. (2004). Aggression in suicide among adults age 50 and over. *American Journal of Geriatric Psychiatry*, *12*, 37–42.

- Conner, K. R., Duberstein, P. R., Conwell, Y., Seidlitz, L., & Caine, E. D. (2001). Psychological vulnerability to completed suicide: a review of empirical studies. *Suicide & Life-Threatening Behavior, 31*, 367–385.
- Conner, K. R., Houston, R. J., Swogger, M. T., Conwell, Y., You, S., He, H., et al. (2012). Stressful life events and suicidal behavior in adults with alcohol use disorders: role of event severity, timing, and type. *Drug and Alcohol Dependence, 120*, 155–161. doi:10.1016/j.drugalcdep.2011.07.013.
- Conwell, Y., Duberstein, P. R., Hirsch, J. K., Conner, K. R., Eberly, S., & Caine, E. D. (2010). Health status and suicide in the second half of life. *International Journal of Geriatric Psychiatry, 25*, 371–379. doi:10.1002/gps.2348.
- Corsellis, J. A., Bruton, C. J., & Freeman-Browne, D. (1973). The aftermath of boxing. *Psychological Medicine, 3*, 270–303.
- Cottler, L. B., Ben Abdallah, A., Cummings, S. M., Barr, J., Banks, R., & Forchheimer, R. (2011). Injury, pain, and prescription opioid use among former National Football League (NFL) players. *Drug and Alcohol Dependence, 116*, 188–194. doi:10.1016/j.drugalcdep.2010.12.003.
- Courville, C. B. (1962). Punch drunk. Its pathogenesis and pathology on the basis of a verified case. *Bulletin of the Los Angeles Neurological Society, 27*, 160–168.
- Crary, J. F., Trojanowski, J. Q., Schneider, J. A., Abisambra, J. F., Abner, E. L., Alafuzoff, I., et al. (2014). Primary age-related tauopathy (PART): a common pathology associated with human aging. *Acta Neuropathologica, 128*, 755–766. doi:10.1007/s00401-014-1349-0.
- Critchley, M. (1949). Punch-drunk syndromes: the chronic traumatic encephalopathy of boxers. In Maloine (Ed.), *Hommage a Clovis Vincent* (pp. 131–145). Strasbourg: Imprimerie Alascienne.
- Critchley, M. (1957). Medical aspects of boxing, particularly from a neurological standpoint. *British Medical Journal, 1*, 357–362.
- Daneshvar, D. H., Goldstein, L. E., Kiernan, P. T., Stein, T. D., & McKee, A. C. (2015). Post-traumatic neurodegeneration and chronic traumatic encephalopathy. *Molecular and Cellular Neurosciences, 66*, 81–90. doi:10.1016/j.mcn.2015.03.007.
- Fassberg, M. M., van Orden, K. A., Duberstein, P., Erlangsen, A., Lapiere, S., Bodner, E., et al. (2012). A systematic review of social factors and suicidal behavior in older adulthood. *International Journal of Environmental Research and Public Health, 9*, 722–745. doi:10.3390/ijerph9030722.
- Fink, D. S., Santaella-Tenorio, J., & Keyes, K. M. (2018). Increase in suicides the months after the death of Robin Williams in the US. *PLoS One, 13*, e0191405. doi:10.1371/journal.pone.0191405.
- Foster, T. (2011). Adverse life events proximal to adult suicide: a synthesis of findings from psychological autopsy studies. *Archives of Suicide Research: Official Journal of the International Academy for Suicide Research, 15* (1), 1–15. doi:10.1080/13811118.2011.540213.
- Fournier, C. N., Gearing, M., Upadhyayula, S. R., Klein, M., & Glass, J. D. (2015). Head injury does not alter disease progression or neuropathologic outcomes in ALS. *Neurology, 84*, 1788–1795. doi:10.1212/WNL.0000000000001522.
- Fralick, M., Sy, E., Hassan, A., Burke, M. J., Mostofsky, E., & Karsies, T. (2018). Association of concussion with the risk of suicide: a systematic review and meta-analysis. *JAMA Neurology, doi:10.1001/jamaneurol.2018.3487*.
- Gao, A. F., Ramsay, D., Twose, R., Rogaeva, E., Tator, C., & Hazrati, L. N. (2017). Chronic traumatic encephalopathy-like neuropathological findings without a history of trauma. *International Journal of Pathology and Clinical Research, 3*, 050.
- Gavett, B. E., Cantu, R. C., Shenton, M., Lin, A. P., Nowinski, C. J., McKee, A. C., et al. (2011a). Clinical appraisal of chronic traumatic encephalopathy: current perspectives and future directions. *Current Opinion in Neurology, 24*, 525–531. doi:10.1097/WCO.0b013e32834cd477.
- Gavett, B. E., Stern, R. A., & McKee, A. C. (2011b). Chronic traumatic encephalopathy: a potential late effect of sport-related concussive and subconcussive head trauma. *Clinical Sports Medicine, 30*, 179–188. doi:S0278-5919(10)00086-4, xi.
- Goldfinger, M. H., Ling, H., Tilley, B. S., Liu, A. K. L., Davey, K., Holton, J. L., et al. (2018). The aftermath of boxing revisited: identifying chronic traumatic encephalopathy pathology in the original Corsellis boxer series. *Acta Neuropathologica, 136*, 973–974. doi:10.1007/s00401-018-1926-8.
- Gonzalez, H. M., Tarraf, W., Whitfield, K. E., & Vega, W. A. (2010). The epidemiology of major depression and ethnicity in the United States. *Journal of Psychiatric Research, 44*, 1043–1051. doi:10.1016/j.jpsychires.2010.03.017.
- Grahmann, H., & Ule, G. (1957). [Diagnosis of chronic cerebral symptoms in boxers (dementia pugilistica & traumatic encephalopathy of boxers)]. *Psychiatr Neurol (Basel), 134*, 261–283.
- Gvion, Y., & Apter, A. (2011). Aggression, impulsivity, and suicide behavior: a review of the literature. *Archives of Suicide Research: Official Journal of the International Academy for Suicide Research, 15*, 93–112. doi:10.1080/13811118.2011.565265.
- Harvey, P. K., & Davis, J. N. (1974). Traumatic encephalopathy in a young boxer. *Lancet, 2*, 928–929.
- Hassett, A. L., Aquino, J. K., & Ilgen, M. A. (2014). The risk of suicide mortality in chronic pain patients. *Current Pain and Headache Reports, 18*, 436. doi:10.1007/s11916-014-0436-1.
- Hawton, K., Casanas, I. C. C., Haw, C., & Saunders, K. (2013). Risk factors for suicide in individuals with depression: a systematic review. *Journal of Affective Disorders, 147*, 17–28. doi:10.1016/j.jad.2013.01.004.
- Hooley, J. M., Franklin, J. C., & Nock, M. K. (2014). Chronic pain and suicide: understanding the association. *Current Pain and Headache Reports, 18*, 435. doi:10.1007/s11916-014-0435-2.
- Ilgen, M. A., Zivin, K., McCammon, R. J., & Valenstein, M. (2008). Pain and suicidal thoughts, plans and attempts in the United States. *General Hospital Psychiatry, 30*, 521–527. doi:10.1016/j.genhosppsych.2008.09.003.
- Iverson, G. L. (2014). Chronic traumatic encephalopathy and risk of suicide in former athletes. *British Journal of Sports Medicine, 48*, 162–165.
- Iverson, G. L. (2016). Suicide and Chronic Traumatic Encephalopathy. *Journal of Neuropsychiatry and Clinical Neurosciences, 28*, 9–16. doi:10.1176/appi.neuropsych.15070172.
- Jedlinski, J., Gatarski, J., & Szymusik, A. (1970). Chronic posttraumatic changes in the central nervous system in pugilists. *Polish Medical Journal, 9*, 743–752.
- Johnson, J. (1969). Organic psychosyndromes due to boxing. *British Journal of Psychiatry, 115*, 45–53.
- Jordan, B. (2000). Chronic traumatic brain injury associated with boxing. *Seminars in Neurology, 20*, 179–185.
- Jordan, B. D. (2013). The clinical spectrum of sport-related traumatic brain injury. *Nature Reviews. Neurology, 9*, 222–230. doi:10.1038/nrneurol.2013.33.
- Karch, D. L., Logan, J., & Patel, N., Centers for Disease, C., & Prevention. (2011). Surveillance for violent deaths—National Violent Death Reporting System, 16 states, 2008. *Morbidity and Mortality Weekly Report. Surveillance Summaries, 60* (10), 1–49.
- Koga, S., Dickson, D. W., & Bieniek, K. F. (2016). Chronic Traumatic Encephalopathy Pathology in Multiple System Atrophy. *Journal of Neuropathology and Experimental Neurology, 75*, 963–970. doi:10.1093/jnen/nlw073.

- Kovacs, G. G., Ferrer, I., Grinberg, L. T., Alafuzoff, I., Attems, J., Budka, H., et al. (2016). Aging-related tau astroglial pathology (ARTAG): harmonized evaluation strategy. *Acta Neuropathologica*, *131*, 87–102. doi:10.1007/s00401-015-1509-x.
- Lehman, E. J., Hein, M. J., & Gersic, C. M. (2016). Suicide mortality among retired national football league players who played 5 or more seasons. *American Journal of Sports Medicine*, *44*, 2486–2491. doi:10.1177/0363546516645093.
- Ling, H., Holton, J. L., Shaw, K., Davey, K., Lashley, T., & Revesz, T. (2015). Histological evidence of chronic traumatic encephalopathy in a large series of neurodegenerative diseases. *Acta Neuropathologica*, *130*, 891–893. doi:10.1007/s00401-015-1496-y.
- Madsen, T., Erlangsen, A., Orlovskaya, S., Mofaddy, R., Nordentoft, M., & Benros, M. E. (2018). Association between traumatic brain injury and risk of suicide. *Journal of the American Medical Association*, *320*, 580–588. doi:10.1001/jama.2018.10211.
- Maroon, J. C., Winkelman, R., Bost, J., Amos, A., Mathyssek, C., & Miele, V. (2015). Chronic traumatic encephalopathy in contact sports: a systematic review of all reported pathological cases. *PLoS One*, *10*, e0117338. doi:10.1371/journal.pone.0117338.
- Martland, H. S. (1928). Punch drunk. *Journal of the American Medical Association*, *19*, 1103–1107.
- Mawdsley, C., & Ferguson, F. R. (1963). Neurological disease in boxers. *Lancet*, *2*, 795–801.
- McAuliffe, C., Keeley, H. S., & Corcoran, P. (2002). Problem solving and repetition of parasuicide. *Behavioural and Cognitive Psychotherapy*, *30*, 385–397.
- McGirr, A., & Turecki, G. (2007). The relationship of impulsive aggressiveness to suicidality and other depression-linked behaviors. *Current Psychiatry Reports*, *9*, 460–466.
- McKee, A. C., Cairns, N. J., Dickson, D. W., Folkerth, R. D., Keene, C. D., Litvan, I., et al. (2016). The first NINDS/NIBIB consensus meeting to define neuropathological criteria for the diagnosis of chronic traumatic encephalopathy. *Acta Neuropathologica*, *131*, 75–86. doi:10.1007/s00401-015-1515-z.
- McKee, A. C., Cantu, R. C., Nowinski, C. J., Hedley-Whyte, E. T., Gavett, B. E., Budson, A. E., et al. (2009). Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury. *Journal of Neuropathology and Experimental Neurology*, *68*, 709–735.
- McKee, A. C., Daneshvar, D. H., Alvarez, V. E., & Stein, T. D. (2014). The neuropathology of sport. *Acta Neuropathologica*, *127*, 29–51. doi:10.1007/s00401-013-1230-6.
- McKee, A. C., Stein, T. D., Nowinski, C. J., Stern, R. A., Daneshvar, D. H., Alvarez, V. E., et al. (2013). The spectrum of disease in chronic traumatic encephalopathy. *Brain: A Journal of Neurology*, *136*, 43–64.
- Mendez, M. F. (1995). The neuropsychiatric aspects of boxing. *International Journal of Psychiatry in Medicine*, *25*, 249–262.
- Mez, J., Daneshvar, D. H., Kiernan, P. T., Abdolmohammadi, B., Alvarez, V. E., Huber, B. R., et al. (2017). Clinicopathological evaluation of chronic traumatic encephalopathy in players of American Football. *Journal of the American Medical Association*, *318*, 360–370. doi:10.1001/jama.2017.8334.
- Mez, J., Stern, R. A., & McKee, A. C. (2013). Chronic traumatic encephalopathy: where are we and where are we going? *Current Neurology and Neuroscience Reports*, *13*, 407. doi:10.1007/s11910-013-0407-7.
- Millspaugh, J. A. (1937). Dementia pugilistica. *U.S. Naval Medicine Bulletin*, *35*, 297–303.
- Montenegro, P. H., Baugh, C. M., Daneshvar, D. H., Mez, J., Budson, A. E., Au, R., et al. (2014). Clinical subtypes of chronic traumatic encephalopathy: literature review and proposed research diagnostic criteria for traumatic encephalopathy syndrome. *Alzheimer's Research & Therapy*, *6*, 68. doi:10.1186/s13195-014-0068-z.
- Morsch, R., Simon, W., & Coleman, P. D. (1999). Neurons may live for decades with neurofibrillary tangles. *Journal of Neuropathology and Experimental Neurology*, *58*, 188–197.
- Nock, M. K., Hwang, I., Sampson, N. A., & Kessler, R. C. (2010). Mental disorders, comorbidity and suicidal behavior: results from the National Comorbidity Survey Replication. *Molecular Psychiatry*, *15*, 868–876. doi:10.1038/mp.2009.29.
- Noy, S., Krawitz, S., & Del Bigio, M. R. (2016). Chronic traumatic encephalopathy-like abnormalities in a routine neuropathology service. *Journal of Neuropathology and Experimental Neurology*. doi:10.1093/jnen/nlw092.
- Omalu, B. (2014). Chronic traumatic encephalopathy. *Progress in Neurological Surgery*, *28*, 38–49. doi:10.1159/000358761.
- Omalu, B., Bailes, J., Hamilton, R. L., Kamboh, M. I., Hammers, J., Case, M., et al. (2011). Emerging histomorphologic phenotypes of chronic traumatic encephalopathy in American athletes. *Neurosurgery*, *69*, 173–183. doi:10.1227/NEU.0b013e318212bc7b; discussion 183.
- Omalu, B. I., Bailes, J., Hammers, J. L., & Fitzsimmons, R. P. (2010a). Chronic traumatic encephalopathy, suicides and parasuicides in professional American athletes: the role of the forensic pathologist. *American Journal of Forensic Medicine and Pathology*, *31*, 130–132. doi:10.1097/PAF.0b013e3181ca7f35.
- Omalu, B. I., DeKosky, S. T., Hamilton, R. L., Minster, R. L., Kamboh, M. I., Shakir, A. M., et al. (2006). Chronic traumatic encephalopathy in a national football league player: part II. *Neurosurgery*, *59*, 1086–1092. ; discussion 1092-1083.
- Omalu, B. I., DeKosky, S. T., Minster, R. L., Kamboh, M. I., Hamilton, R. L., & Wecht, C. H. (2005). Chronic traumatic encephalopathy in a National Football League player. *Neurosurgery*, *57*, 128–134. ; discussion 128-134.
- Omalu, B. I., Fitzsimmons, R. P., Hammers, J., & Bailes, J. (2010b). Chronic traumatic encephalopathy in a professional American wrestler. *Journal of Forensic Nursing*, *6*, 130–136. doi:10.1111/j.1939-3938.2010.01078.x.
- Omalu, B. I., Hamilton, R. L., Kamboh, M. I., DeKosky, S. T., & Bailes, J. (2010c). Chronic traumatic encephalopathy (CTE) in a National Football League Player: Case report and emerging medicolegal practice questions. *Journal of Forensic Nursing*, *6*, 40–46. doi:10.1111/j.1939-3938.2009.01064.x.
- Parker, H. L. (1934). Traumatic encephalopathy ('punch drunk') of professional pugilists. *Journal of Neurology and Psychopathology*, *15*, 20–28.
- Payne, E. E. (1968). Brains of boxers. *Neurochirurgia (Stuttg)*, *11*, 173–188. doi:10.1055/s-0028-1095326.
- Puvanna, V., Engeler, M., Banjara, M., Brennan, C., Schreiber, P., Dadas, A., et al. (2016). Is phosphorylated tau unique to chronic traumatic encephalopathy? Phosphorylated tau in epileptic brain and chronic traumatic encephalopathy. *Brain Research*, *1630*, 225–240. doi:10.1016/j.brainres.2015.11.007.
- Reams, N., Eckner, J. T., Almeida, A. A., Aagesen, A. L., Giordani, B., Paulson, H., et al. (2016). A Clinical approach to the diagnosis of traumatic encephalopathy syndrome: a review. *JAMA Neurology*, *73*, 743–749. doi:10.1001/jamaneurol.2015.5015.
- Richardson, T., Elliott, P., & Roberts, R. (2013). The relationship between personal unsecured debt and mental and physical health: a systematic review and meta-analysis. *Clinical Psychology Review*, *33*, 1148–1162. doi:10.1016/j.cpr.2013.08.009.
- Roberts, A. (1969). *Brain damage in boxers: A study of prevalence of traumatic encephalopathy among ex-professional boxers*. London: Pitman Medical Scientific Publishing Co.
- Schwenk, T. L., Gorenflo, D. W., Dopp, R. R., & Hipple, E. (2007). Depression and pain in retired professional football players. *Medicine and Science in Sports and Exercise*, *39*, 599–605. doi:10.1249/mss.0b013e31802fa679.

- Scott, K. M., Hwang, I., Chiu, W. T., Kessler, R. C., Sampson, N. A., Angermeyer, M., et al. (2010). Chronic physical conditions and their association with first onset of suicidal behavior in the world mental health surveys. *Psychosomatic Medicine*, 72, 712–719. doi:10.1097/PSY.0b013e3181e3333d.
- Sercl, M., & Jaros, O. (1962). The mechanisms of cerebral concussion in boxing and their consequences. *World Neurology*, 3, 351–358.
- Sisask, M., & Varnik, A. (2012). Media roles in suicide prevention: a systematic review. *International Journal of Environmental Research and Public Health*, 9, 123–138. doi:10.3390/ijerph9010123.
- Stern, R. A., Daneshvar, D. H., Baugh, C. M., Seichepine, D. R., Montenigro, P. H., Riley, D. O., et al. (2013). Clinical presentation of chronic traumatic encephalopathy. *Neurology*, 81, 1122–1129. doi:10.1212/WNL.0b013e3182a5517f.
- Stern, R. A., Riley, D. O., Daneshvar, D. H., Nowinski, C. J., Cantu, R. C., & McKee, A. C. (2011a). Long-term consequences of repetitive brain trauma: chronic traumatic encephalopathy. *PM & R: The Journal of Injury, Function, and Rehabilitation*, 3, S460–S467. doi:10.1016/j.pmrj.2011.08.008.
- Stern, R. A., Riley, D. O., Daneshvar, D. H., Nowinski, C. J., Cantu, R. C., & McKee, A. C. (2011b). Long-term consequences of repetitive brain trauma: chronic traumatic encephalopathy. *PM & R: The Journal of Injury, Function, and Rehabilitation*, 3, S460–S467. doi:10.1016/j.pmrj.2011.08.008.
- Tang, N. K., & Crane, C. (2006). Suicidality in chronic pain: a review of the prevalence, risk factors and psychological links. *Psychological Medicine*, 36, 575–586. doi:10.1017/S0033291705006859.
- Van Orden, K. A., Merrill, K. A., & Joiner, T. E. (2005). Interpersonal-psychological precursors to suicidal behavior: A theory of attempted and completed suicide. *Current Psychiatry Reviews*, 1, 187–196.
- Victoroff, J. (2013). Traumatic encephalopathy: review and provisional research diagnostic criteria. *NeuroRehabilitation*, 32, 211–224. doi:10.3233/NRE-130839.
- von Bergen, M., Barghorn, S., Biernat, J., Mandelkow, E. M., & Mandelkow, E. (2005). Tau aggregation is driven by a transition from random coil to beta sheet structure. *Biochimica et Biophysica Acta*, 1739, 158–166. doi:10.1016/j.bbadis.2004.09.010.
- Webner, D., & Iverson, G. L. (2016). Suicide in professional American football player in the past 95 years. *Brain Injury*, 30, 1718–1721.
- Weir, D. R., Jackson, J. S., & Sonnega, A. (2009). *National Football League Player Care Foundation: Study of retired NFL players*. Ann Arbor, MI: University of Michigan, Institute for Social Research.
- Wilcox, H. C., Conner, K. R., & Caine, E. D. (2004). Association of alcohol and drug use disorders and completed suicide: an empirical review of cohort studies. *Drug and Alcohol Dependence*, 76, S11–S19. doi:10.1016/j.drugalcdep.2004.08.003.
- Wortzel, H. S., Shura, R. D., & Brenner, L. A. (2013). Chronic traumatic encephalopathy and suicide: a systematic review. *BioMed Research International*, 2013, 424280. doi:10.1155/2013/424280.
- Yang, A. C., Tsai, S. J., Yang, C. H., Shia, B. C., Fuh, J. L., Wang, S. J., et al. (2013). Suicide and media reporting: a longitudinal and spatial analysis. *Social Psychiatry and Psychiatric Epidemiology*, 48, 427–435. doi:10.1007/s00127-012-0562-1.