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The brain and beyond in the aftermath of head trauma - a systems view of development for contact sport athletes

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Abstract: The most recent attention raised regarding the developmental struggles some contact sport athletes face has turned to examining the effects of exposure to head trauma. Specifically, the presence of a singular neuropathology, Chronic Traumatic Encephalopathy (CTE), has been found in the brains of dozens of deceased former contact sport athletes who suffered a range of debilitating symptoms. Research into the etiology, course, and effects of CTE has revealed vital findings into understanding the diverse range of behavioral, somatic, cognitive, motor, and affective symptoms experienced by these athletes. However, scientists examining CTE have likewise acknowledged that many of these symptoms do not occur until several years (approximately 6–15) after contact sport athletes leave their careers and the field of play. Coincidentally, alongside the findings in CTE research, an examination of the psychosocial struggles athletes face upon career transition and retirement has revealed a variety of symptoms similar to the range revealed in patients who have been diagnosed with CTE. This paper will provide a selective literature review of the approximately 50 years of research conducted on these psychosocial struggles. The focus of this review will be specifically on how the loss of identity, the breakdown of social structures, and the reinforcement of violent tendencies can play a significant role in exacerbating a similar range of symptoms to CTE.

ABOUT THE AUTHORS

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PUBLIC INTEREST STATEMENT

Recent attention has been placed upon the effect that exposure to severe and recurrent levels of head trauma may cause for contact sport athletes. Chronic Traumatic Encephalopathy (CTE) has been found in the brains of dozens of former contact sport athletes, many of whom experienced difficult career transitions or even tragic ends to their lives. The range of symptoms found in former contact sport athletes who were diagnosed with CTE includes memory loss, lack of motivation, depression, mania, bipolar, suicidal thoughts, irritability, loss of emotional control, and rage. Nonetheless, athletic career transition research has been conducted for roughly 50 years and similar symptoms have been found in athletes who have experienced little or no head trauma during their careers. Ultimately, this paper offers valuable insight for anyone who plays contact sports, has played contact sports, coaches contact sports, or is involved in aiding the career transitions of these former athletes.

Subjects: Neuroscience; Development Studies, Environment, Social Work, Urban Studies; Sport and Exercise Science; Sports Psychology

Keywords: CTE; head trauma; contact sport athletes; athletic career transition; psychosocial struggles

1. Introduction

Recent attention has been raised regarding the developmental struggles that contact sport athletes must navigate upon careers exposing them to severe and recurrent levels of head trauma. Much of this attention has been sparked by a string of deaths, tragedies, suicides, arrests, and testimony from former athletes into the cognitive, behavioral, emotional, and social struggles faced in life after sport. The most recent attention in the scientific community has turned to examining the effects of head trauma, in particular the effect of a singular neuropathology: Chronic Traumatic Encephalopathy (CTE) (Omalu et al., 2005). Since that time, dozens of autopsies have overwhelmingly confirmed the prevalence of CTE in the brains of former and now deceased contact sport athletes. Many of these individuals suffered from a range of somatic, cognitive, behavioral, affective, psychological, and social symptoms prior to their death and, especially, upon their retirement from sports. The range of these symptoms includes, though is not limited to, memory loss, deteriorating motor skills, anhedonia, depression, mania, bipolar, suicidal ideation, irritability, loss of executive function, and rage (Stern et al., 2011). The two clinical classifications of CTE that have been determined thus far are early onset, which mostly generates mood and behavioral changes, and late onset, which involves more cognitive and motor deficits (Bailes, Turner, Lucke-Wold, Patel, & Lee, 2015). In consideration of the latter, multiple studies have noted a latency period of unknown origin between retirement from sport and CTE symptom occurrence which ranges approximately from six to fifteen years (Bailes et al., 2015; McKee, Alosco, & Huber, 2016). However, by focusing on the neurological dimension as the sole etiological root of these symptoms and behaviors, important attention is taken away from factors that may prove to be comparatively causal.

Alongside the justifiable attention that has been paid to the effects of head trauma contributing to these symptoms, there exists approximately 50 years of research focusing solely on psychological and social factors that contribute to a similar range of symptoms for athletes as a result of complications experienced in athletic career transition. Though the psychosocial research on athletic career transition has occurred almost wholly independent from the CTE and head trauma research, it investigates nuances of the experience that might be exacerbating some of the same struggles and a similar range of symptoms that the CTE and head trauma research have revealed. First, questions of identity and identity foreclosure are examined, in particular, both identity loss and the challenge of reshaping identity after sport (Brewer, Van Raalt, & Linder, 1993). Second, an examination of the reinforced behaviors and mindsets formed in contact sports, as well as the ability to reintegrate these behaviors (e.g. aggression, violence, rage, intimidation, etc.) into a professional career after sport (Kerr, 2005; Zillmann, Katcher, & Milavsky, 1972). Finally, there has been a close look at the role that interpersonal shifts and newly altered relational dynamics might play in affecting the former athlete's life after retirement (Aquilina, 2013; Stambulova, 2010).

There are two tremendous obstacles at this moment for individuals who have experienced head trauma and are living with symptoms associated with CTE. First, there is, to date, no living diagnosis for CTE. Every case of CTE has only been diagnosed postmortem and though there is reason to believe that the future will provide for a living diagnosis, an uncounted number of former athletes may be living with some range of the symptoms associated with CTE with no clear diagnostic assessment. Second, there exists a much greater and, to this date, unsolved problem for those who are living with CTE. To date, there is no consensus for a possible biological intervention that might alleviate or reduce the presence of CTE and/or the symptoms associated with it. Unlike the warranted optimism for an in-life diagnosis, there is currently a dearth of confidence regarding the possibility of a biological intervention. As a result, there is only one plausible step to prevent the progression of CTE in the brains of contact sport athletes: stop playing.

For many reasons, this response is far from tenable, especially for those who have already invested years of their career in contact sports. Ultimately, though biological research has provided significant insight into the etiology, progression, and activity of CTE as a unique neurodegenerative tauopathy, the hope for a biological resolution for this disease is at best many years on the horizon. Upon reviewing the full scope of the literature, one is left to ponder what the proper relationship should be between ascribing causation to head trauma and/or the general psychosocial difficulties many athletes face upon transitioning out of sport in light of this range of symptoms. It seems quite possible that both strains of research (head trauma and psychosocial investigations of difficulties in career transition) can contribute to understanding the etiology of this diverse range of symptoms.

Ultimately, it appears plausible that scientists, doctors, and psychologists who work with this population should take a broader systems approach in order to understand the difficulties athletes face after competing in a career that exposes them to head trauma. A systems approach argues that only a holistic conceptualization of these struggles and symptoms will fully account for their etiology and, consequently, any potential successful intervention for the athlete. A systems approach states that the individual, "develops and functions psychologically as an integrated organism. Maturational, experiential and cultural contributions are fused in this ontogeny. Single aspects do not develop and function in isolation, and they should not be divorced from totality in analysis" (Magnusson & Cairns, 1996, p. 12). By assessing the conclusions of the psychosocial research on athletic career transition to light with patients who have been exposed to head trauma and, thus, may be living with CTE, there exists at least the possibility that doctors, consultants, and the athletes themselves can be briefed on methods that may alleviate, curb, and/or reduce the some of the symptoms that have been associated with both CTE patients and athletes who struggle with retirement or deselection from sport.

This paper will make precisely such an argument—in short, that a broader approach to conceptualizing the symptoms associated with CTE must be explored further in order to aid athletes who may be suffering from the symptoms associated with CTE or a career in contact sports. The research presented will demonstrate how psychological and social factors can contribute to and/or exacerbate a similar range of symptoms that has been associated with CTE for former contact sport athletes. Likewise, it is plausible to at least hypothesize that the intervention techniques suggested by the psychosocial literature could work to stabilize or even alleviate some range of these symptoms. By providing a meta-synthesis of the literature in distinct categories—findings in the field of CTE research, athletic identity research, the reinforced tendencies of aggression and violence, and the breakdown of social support structures—we will form the foundation of a theory explication and commentary that will reassess the etiology of the symptoms, behaviors, and difficulties associated with CTE for former contact sport athletes, as well as possible intervention techniques for individuals living with this range of symptoms.

Ultimately, there exists an exigent value in providing a theory explication and commentary regarding the etiology and treatment of symptoms associated with CTE. As mentioned, to date, there is no form of biological intervention offered to individuals who may be living with CTE. No biological intervention—except for the suggestion of avoiding head trauma, an option that is not plausible for those already diagnosed—has been offered that can mitigate the symptoms or reverse CTE's neurodegeneration. A broad conceptualization of the symptoms associated with CTE that accounts for psychological and social factors in its etiology will promote the demand for intervention that involves social support, psychoeducation, and psychological coping for former athletes struggling with these symptoms or symptoms closely related to CTE. Furthermore, the intention of setting the foundation of a systems approach would be to spur further research that broadly examines the symptoms and struggles athletes experience after careers that expose them to repeated head trauma and abrupt career transitions.

2. Complicated case studies

To begin, it is vital to take note of several anecdotal examples from the dozens of known cases of CTE to illustrate the point that head trauma research would benefit from an intersectional dialog with psychosocial research on athletic career transition. Among the dozens of subjects who have been diagnosed with CTE, few of these individuals demonstrate unequivocal signs and symptoms of CTE *during* their careers in contact sports. One of these athletes is former Kansas City Chiefs linebacker Javon Belcher. In the tragic case of Mr Belcher, it was reported that he was displaying socially isolating behaviors, signs of depression, memory loss, irritability, limited executive function, and a diminished sense of stability in his relationships in the months before he murdered the mother of his 3 month old daughter and committed suicide in the Kansas City Chief's parking lot. However, in a majority of the dozens of CTE victims, the impact and presence of the symptoms related to CTE either were not reported during their athletic career or are seriously exacerbated only in the aftermath of retirement/deselection from the individual's career in athletics (Bailes et al., 2015; McKee et al., 2016).

The cases of both Mike Webster and Junior Seau provide for such a timeline and, thus, the need for a biopsychosocial conceptualization. In both of these cases, the timeline of symptomatic presence was consistent. It wasn't until *after* both men completed their NFL careers that the symptoms of CTE became prevalent and invasive in their lives. Azad, Li, Pendharkar, Veeravagu, and Grant (2016) provide a summarized analysis of the case history of Mr Seau:

> Seau had complained of headaches, episodes of dizziness, and insomnia since as early as the 1990s. Seau's ex-wife recalled to the media, "When he would come home from games, he would go straight to the room … [He'd] lower the blinds, the blackout blinds, and just say, 'Quiet, my head is, is burning.'" After Seau's retirement from the NFL in 2010, his family and friends reported surprising behavioral changes that included withdrawal, heavy alcohol consumption, reckless business and financial decisions, and gambling. Seau also became more aggressive and sometimes violent with his close friends and family, which was reported as uncharacteristic. He was arrested on 18 October 2010 for a domestic violence incident with his girlfriend. On 2 May 2012, Seau was found dead from apparent suicide at age 43. According to his publicly released autopsy report from the Office of the Medical Examiner, County of San Diego, California (Case Number 12-00960), Seau had no history of smoking or illicit drug use, and his alcohol history was that he "drank socially." His prescription medications found at the time of his death included 10 mg zolpidem, 500–125 mg amoxicillin, 500 mg naproxen, and 50 mg propylthiouracil. (Azad et al., 2016)

For Mr Seau, it was roughly 2 years after his career that he committed suicide. Though symptoms began to show for Mr Seau during his career, these are relatively mild as compared to the massive behavioral and mood changes that occurred upon his retirement. Seau was on an active NFL roster until the year 2009, leading into his 40th birthday and 19th season in the NFL. Throughout the course of his career, Seau was always viewed as an emotional individual and many can point to his first retirement press conference in 2006 as evidence of perhaps some emotional instability (e.g. Mr Seau tearfully announced his retirement in 2005 only to resign back with an NFL team 4 days later). However, beyond this, there are no documented accounts of emotional instability, depression, lack of executive function, and antisocial tendencies during his career. In fact, just the opposite has been overwhelmingly documented, with teammates, coaches, and friends attesting consistently to how passionate, devoted, gregarious, and affectionate a friend and teammate Mr Seau was throughout the duration of his career. As documented by his family and close friends, CTE symptoms occurred only *after* his leaving the game of football leading, ultimately, to Mr Seau's tragic suicide in 2012 (Kirk, Gilmore, & Wiser, 2013).

For Mike Webster, the range of symptoms associated with CTE became present only after retiring from the NFL and remained with him for roughly a decade until his abrupt death at the young age of 50. Mr Webster's family members, similar to Mr Seau's, documented the significant changes in social and emotional stability occurring only *after* he had completed his illustrious career with the Steelers.

Though Mr Webster did not commit suicide and, ultimately, had a longer post-career lifespan than Mr Seau, it is quite possible that the downturn for Mr Webster was even more stark. Addiction, emotional volatility, self-injurious tendencies, social isolation, and insomnia all riddled the thirteen years Mr Webster lived after his career. When he passed at age 50, doctors could only describe such an early death as his body shutting down (Kirk et al., 2013). What is clear is that, similar to Mr Seau, this range of behaviors, tendencies, and symptoms were documented for Mr Webster only *after* his hall of fame career in the NFL. It was when both men *left* the arena of competition that symptoms associated with CTE occurred and, as mentioned, this timeline is quite common with an overwhelming percentage of those individuals who have been diagnosed with CTE. None of this is to deny that Mr Seau or Mr Webster had CTE throughout the course of their careers or that head trauma played a significant role in affecting their emotional stability, depression, social withdrawal, and even their respective deaths. Instead, the hypothesis is that the presence of CTE led to an onset of symptoms that were quite likely exacerbated as a result of certain psychosocial difficulties associated with athletic career transition.

3. Neuropsychological conceptualizations

CTE has been linked to participation in contact sports such as boxing, hockey, soccer, lacrosse, and American football. CTE research has offered insight into the potential causal linkage between traumatic brain injury (TBI), minor traumatic brain injury (mTBI), and a range of symptoms, behaviors, and cognitive impairment that individuals who have competed in contact sports might exhibit during and after their careers. Among these, CTE has been linked to a progressive decline of memory and cognition, as well as a series of affective, social, and abnormal disorders such as depression, suicidal behavior, bipolar, apathy, poor impulse control, aggressiveness, parkinsonism, sleep apnea, and, eventually, dementia (Stern et al., 2011). The results show that athletes who suffer TBI or mTBI are at risk to develop this singular tauopathy resulting in the behavioral, emotional, somatic, and cognitive symptoms.

As mentioned in the introduction, sports psychologists have spent roughly a half-century attempting to conceptualize the psychosocial roots of a similar range of symptoms and behaviors in the lives of athletes. CTE research is groundbreaking because it potentially pinpoints the cause of affective abnormality, cognitive impairment, and behavioral deviance to buildups of tau protein and neurofibrillary tangles in localized regions of the brain. Ultimately, neuroscientific findings have clearly and empirically uncovered one of the factors contributing to this range of symptoms beyond the possible difficulties faced in career transitions for former contact sport athletes.

3.1. An overview of chronic traumatic encephalopathy

CTE is currently understood as a distinct tauopathy—a class of neurodegenerative diseases marked by the pathological aggregation of tau protein—that can be differentiated from more commonly known tauopathies such as Alzheimer's disease (AD) and Parkinson's disease. The epidemiology of CTE is actively being researched, but the prevalence, mechanism, and course of this disease is currently unknown. Physicians conduct postmortem examinations of brains suspected of having CTE, and are able to distinguish CTE from other known tauopathies through gross and microscopic pathology. Dennis Dickson, MD, Professor of Pathology, Mayo Clinic, Jacksonville notes that, "none of the individual pathologic features (such as tau pathology) are unique to CTE, but what confers uniqueness is their peculiar distribution within the brain" (National Institutes of Health, 2013). Recently, a UCLA study examined whether brain tau deposits can be detected in living brains by utilizing positive emission tomography (PET) scans with intravenous injections of a chemical compound known as FDDNP, which bind to such deposits (Small et al., 2013):

> Using a new imaging tool, researchers found a strikingly similar pattern of abnormal protein deposits in the brains of retired NFL players who suffered from concussions. The innovative imaging technique … was initially tested in five retired NFL players and described in an article published in 2013. Now, building on their previous work, UCLA researchers found the same characteristic pattern in a larger number of retired players who had sustained concussions.

> The latest study also shows that the brain imaging pattern of people who have suffered concussions is markedly different from the scans of healthy people and from those with Alzheimer's disease. Researchers say the findings could help lead to better identification of brain disorders in athletes and would allow doctors and scientists to test treatments that might help delay the progression of the disease before significant brain damage and symptoms emerge. (Champeau, 2015)

The results of this study give hope for a living diagnosis, but, to date, are not conclusive. As recently as 2015, the FDA has reprimanded the neuroscience lab at UCLA for premature publication and for unjustifiably touting the potency of the research. Inspired by the UCLA study, further advancements have been made in the attempt to provide an in life diagnosis of CTE (Cherry et al., 2016; Coughlin et al., 2015; Vile & Atkinson, 2017).

There is a long, though scattered and sporadic, history of scientific research on brain injury and its effects. In 1928, Martland, described a symptom spectrum in boxers, which he termed "punch drunk," that appeared to result from the repeated blows experienced in the sport. This symptom occurred particularly in slugging boxers who took significant head punishment as part of their fighting style. Millspaugh (1937) introduced the term dementia pugilistica to describe the syndrome characterized by motor deficits and mental confusion in boxers. In 1966, Miller conducted one of the first articles to examine head injuries encountered that are encountered in civilian life (e.g. industrial and traffic accidents) and the potentially psychological and mental effects of these injuries. By the 1970s, a neuropathologic report, by Corsellis and Freeman-Browne (1973), demonstrated dementia pugilistica in 15 boxers and concluded that, although similar to other neurodegenerative diseases, dementia pugilistica is a neuropathologically distinct disorder.

In order to gain a comprehensive understanding of CTE, the condition's etiology and progression must first be analyzed to provide a basis for examining causation and pathology of the neurological degeneration. From a causal perspective, the onset of CTE is typically attributable to repetitive concussive and sub-concussive blows to the head sustained over a period of time. The syndrome then manifests after a latency period of variable duration, though typically this occurs after retirement when analyzed in former athletes (McKee et al., 2016). Through the course of their research, Omalu et al. (2005) and McKee et al. (2016) have determined several features to be constitutive of CTE; some of these include tau (τ) neurofibrillary tangles and plaques in the neocortex; cerebral and cerebellar cortical atrophy; degeneration and depigmentation of the substantia nigra; reduced brain weight; thalamic and hypothalamic atrophy; and atrophy of the amygdala and hippocampus (McKee et al., 2016; Omalu et al., 2005). The aggregated presence of τ protein within neuronal and glial tangles is of particular prevalence as the excessive propagation of its improper formation is apparent in the pathologies of approximately twenty different neurodegenerative disorders (Koliatsos, Xu, Ryu, & Ziogas, 2017). One theory Koliatsos et al. (2017) offers for the excessive quantity of τ protein is the prion formation, which explains the spread of misfolded proteins through their seeding into stable conformational states that then corrupts the normal proteins and templates into β formation, the replication of which eventually disrupts function and causes neuronal death. The malformation of τ protein is particularly damaging as it, when functioning properly, stabilizes microtubules and facilitates the movement of organelles and molecules down microtubule trails in order to facilitate neuronal function. This is one potential explanation for the drastic increase of τ protein in brains with neurodegenerative diseases. Through an understanding of this neuropathology and etiology, the symptoms that present themselves in CTE patients can better be understood and analyzed.

With respect to etiology, it still remains unclear whether a small number of large impacts or a large number of small impacts is more likely to result in CTE. This is unlikely to be resolved until an *in vivo* test is developed to detect the syndrome and monitor its stage-wise progression. Currently these understandings are limited as diagnosis of CTE can only be made retroactively through the autopsy of a subject's brain; which is limited in and of itself as there is currently no exact determination of CTE's observable clinicopathological progression beyond its four discernable stages (Koliatsos

et al., 2017). Though much of the research on CTE and neurological psychopathology focuses on TBI, concussions, and major brain injuries in individuals and their consequent effects on psychological life, more recent research has focused on how mTBI can sometimes lead to persistent post concussion symptoms (PCS). Stern and Kayward (2006) write:

> One well accepted hypothesis claims that chronic PCS has a neural origin, and is related to neurobehavioral deficits. 11 out of 38 mTBI participants (29%) were found to suffer from PCS. This subgroup of mTBI patients performed poorly on neuropsychological test batteries. Thereby, a correlation was found between PCS symptom severity and test performance suggesting that participants with more pronounced PCS symptoms performed worse in cognitive tasks ... The results support the idea that mTBI can have sustained consequences, and that the subjectively experienced symptoms and difficulties in everyday situations are related to objectively measurable parameters in neurocognitive function. (Stern & Kayward, 2006, p. 5)

The major contribution of this study and others (Stern et al., 2011) serves to demonstrate how the prevention of concussions or malicious blows to the head is a necessary though not sufficient condition in controlling PCS and potentially CTE. The very nature of contact sports creates conditions where, throughout the course of any given match, a pervasive and ubiquitous level of subconcussive trauma to the head takes place. The accumulations of subconcussive and lower impact blows to the head are likely to bear some amount of the responsibility for the formation of PCS and CTE.

Finally, the symptoms of CTE can be broken into distinct categories of classification. From a behavioral perspective, individuals with CTE demonstrate increased propensity toward aggression, impulsivity, explosivity, violence, substance abuse, and impaired judgment. The expression of these behaviors typically correlates with early onset CTE, and (from the neuropathological perspective) relates to the damage in the midbrain and various neurotransmitters. Cognitively, people with CTE may suffer deficits in learning, memory, and concentration (Bailes et al., 2015; Omalu et al., 2005). These losses are likely attributable to the atrophy of key components of the limbic system, namely the hippocampus and the amygdala, which are largely responsible for learning and memory. The manifestation of CTE symptoms that pertain to mood and motor function are often attributed to the clinical classification of "older" onset and become apparent after a latency period of six to fifteen years. Regarding changes in mood, some shifts include an increased tendency toward depression, apathy, irritability, morbid jealousy, and paranoia—factors that ultimately culminate in an increased suicide risk (Bailes et al., 2015; McKee et al., 2016; Omalu et al., 2005). The final symptom classification, associated with the latent onset of the syndrome, relates to motor deficits that can manifest through tremor, dysarthria, decreased reaction time, lack of coordination, erratic eye motion, and movement disorders similar to Parkinson's disease (Bailes et al., 2015; Omalu et al., 2005).

3.2. Future inquiries into CTE

Despite this rather lengthy history of examining the effects of head trauma and concussions on contact sport athletes, questions still remain and research seems to still be in a nascent stage. Due to its efforts, empirical grounding, and relation to questions of social, affective, cognitive, motor, and developmental abnormalities, neuroscientific findings surrounding TBI, mTBI, and CTE have carved out an essential seat at the table for conceptualizing the difficulties individuals face after exposure to head trauma. Yet, the analysis provided by neuroscience has questions that are left unaddressed. For example, to date, no control group study or longitudinal assessment has been performed to account for the possibility of athletes who are living with CTE but not suffering the symptoms, or same level of symptoms, associated with the original case studies. While the symptoms of CTE can be expressed variably with respect to temporality and intensity, some subjects are found to be totally asymptomatic despite postmortem confirmations of CTE (McKee et al., 2016; Stern et al., 2013). In fact, a majority of contact sport athletes will never present CTE's external neuropathological symptoms, regardless of any neuropathological progression evinced by their brains postmortem (Bailes et al., 2015). As mentioned, the symptoms of CTE can be organized into two classifications (early and late onset) and four categories (behavioral, cognitive, mood, and motor). However, what leads to the

division into two clinical presentations of CTE (early and late onset) is not presently clear from a neuropathological perspective (Stern et al., 2013).

Several studies have already begun to push back against the notion that CTE certainly and definitively causes the symptoms associated with patients who have been diagnosed with CTE postmortem. Davis, Castellani, and McCrory (2015) write, "Sport–related CTE (can) occur in a retired athlete with or without a history of concussions … There are many confounding variables that can account for brain tau deposition, including genetic mutations, drugs, normal again, (and) environmental factors." Similar to Davis et al. (2015), Iverson, Gardner, McCrory, Zafonte, and Castellani (2015) argue that:

> The described clinical features in recent cases are very similar to how depression manifests in middle-aged men and with frontotemporal dementia as the disease progresses. It has not been established that the described tau pathology, especially in small amounts, can cause complex changes in behavior such as depression, substance abuse, suicidality, personality changes, or cognitive impairment. (Iverson et al., 2015)

In both critiques of the current CTE literature, the groundwork is being laid to argue for a larger conceptualization of these symptoms when they become present in the lives of retired contact sport athletes. Ban, Madden, Bailes, Hunt Batjer, and Lonser (2016) go a bit further, directly addressing the issues of sampling bias, hindsight testimony bias on the part of family members of the deceased players, and the lack of longitudinal evidence comparing and contrasting a control population to those who have been diagnosed with CTE.

Finally, Gaetz (2017) likewise suggests that scientists need to examine athletes who had CTE and examine other factors that affected them such as substance abuse, chronic pain, and stress associated with career transition. He writes:

> At present, there is little scientific evidence to suggest that all CTE symptomology is the product of CTE pathophysiology. It has been assumed that CTE pathophysiology causes CTE symptomology (Iverson et al., 2016; Meehan et al., 2015) but this link has never been scientifically validated … There is significant overlap between the case reports of athletes with post-mortem diagnoses of CTE, and symptom profiles of those with a history of substance use, chronic pain, and athlete career transition stress. The athlete post-career adjustment (AP-CA) model is intended to explain some of the symptoms that athletes experience at the end of their careers or during retirement. The AP-CA model consists of four elements: neurotrauma, chronic pain, substance use, and career transition stress. Based on the existing literature, it is clear that any one of the four elements of the AP-CA model can account for a significant number of CTE symptoms. In addition, depression can be a chronic lifelong co-morbid condition that may be present prior to an athletic career.

Ultimately, and similar to the point made in this paper, Gaetz asserts that while head trauma is a *necessary* factor in developing the disorder, it is *not sufficient* in and of itself and other factors must be present to render CTE symptomatic. Though the head researchers at Boston University's Legacy Institute for CTE research have rightfully acknowledged many of these criticisms (Stern et al., 2011, 2013), moving forward, CTE researchers will benefit from a broadened conceptual lens of the etiology, course, and treatment of the disorders and its related symptoms.

Adding more curiosity and complexity to the current state of this research is the study provided by Savica et al. (2012). In this study, researchers examined the differences in a community between long-term neurological issues and psychopathology with individuals who played high school football and those who did not. Ultimately, there was discovered to be no statistical difference between those who played high school football and those who did not. Both groups were assessed as elderly adults and there was no statistical difference regarding the effects of head trauma from contact sports accrued early in their life for those who played high school football. Of course, this is only one community study and these individuals only played high school football. However, there has been the detection of CTE in the brains of high school and early college aged contact sport athletes, simply

further adding to some of the questions left to be resolved despite the openings this research has made (BU, Eric Pelley Case).

Researchers should be applauded for identifying this unique neuropathology, and recognizing its strong association with TBI and mTBI. However, the retrospective analysis of highly selected research samples has made consensus about many aspects of CTE difficult. For example, there is still no consensus on how much trauma is needed to induce CTE; there is no consensus on how head trauma causes CTE, or whether trauma alone is *sufficient* to cause CTE; there is no advanced diagnostic criteria for CTE; currently, there exists no consensus on how CTE progresses, and there are inconsistent findings about how the symptoms associated with CTE correlate with the neuropathological findings (and whether these neuropathological findings are *sufficient* in explaining psychological changes in at-risk populations). In a summary of a recent CTE conference series, Gabrielle Stobel notes "Media attention has far outpaced the available science. In most areas of science, researchers make progress in relative obscurity before the general media develops an interest. Here the opposite has happened" (2011, p. 4).

Most importantly, the direction of current research seems to overlook the experiential factors that impact individuals who have engaged in a career exposing them to head trauma. The abrupt transition of ending an athletic career or returning to a life outside of sport has thus far been relatively excluded from the conversation about CTE research findings. Science can gain a better understanding about the progression of CTE's neuropathology and clinical presentation with a more integrative assessment of the lives of at-risk individuals. For example, how can we differentiate the variances in individual responses to TBI in terms of initial CTE development, and, once CTE begins, how do we account for different individual phenotype progressions in terms of neuropathology and clinical presentations? These questions are in no way intended to raise doubt about the impact neurodegeneration can have on psychological experience. Rather, the intent of this paper is to enlarge current CTE research considerations and to appreciate the relational inseparability, of neurogenic activation and development with experience. The identification and prevention of a neurodegenerative disease is a noble goal, but a more nuanced exploration of the risk factors that influence the clinical presentation of many retired athletes is surely needed.

These questions are in no way intended to cast doubt about the impact neurodegeneration can have on psychological experience. Rather, the intent of this paper is to enlarge current CTE research considerations and to appreciate the relational inseparability, of neurogenic activation and development with experience. A strict focus on head trauma and clinicopathological correlations will impoverish research findings and diminish the benefits of potential interventions. The identification and prevention of a neurodegenerative disease is a noble goal, but a more nuanced exploration of the risk factors that influence the clinical presentation of many retired athletes surely needed.

4. The brain & beyond—psychosocial conceptualizations

4.1. Athletic identity studies

Brewer et al. (1993) have provided a quantitative survey that is absolutely central to the psychology of career transition for athletes—The Athletic Identity Measurement Scale. This scale has participants answer a series of questions that would calculate the extent of athletic identity demonstrated at that moment in their lives, becoming a barometer to predict the quality of transition for the athlete. This has led many sports psychologists to argue that the key to career transition is a dissolving or diluting of athletic identity, preferably before the athlete's career has actually been completed (Brewer et al., 1993; Petitpas, 1978; Petitpas & France, 2010). In short, the more an individual still considers himself solely an athlete, the less likely a successful career transition out of the competitive arena will be. Inevitably, it is proposed that one's concept of athletic identity plays a central role in the experience of loss in sport.

Participants who demonstrate high levels of athletic identity generally experience identity foreclosure. Identity foreclosure, a term applied to the field of sports psychology by Petitpas but holding its roots in developmental psychology and psychoanalysis through the work of Marcia (1966), refers to an inability or unwillingness of the individual to exhibit exploratory behavior and, thus, commit to specific and narrow roles of identity. Athletes who experience identity foreclosure make an unwavering commitment to sport to avoid identity crises. Athletes experiencing psychological identity foreclosure view their sport successes as the only means of maintaining their coaches' or peers' approval. As a result, they are given the perceived means to defend against any threats to their ego identity by avoiding people or situations that might challenge the salience of athletic participation.

Ultimately, athletic identity and identity foreclosure research on athletic career transition provide insight into another possible etiological source for a range of symptoms associated with CTE. At minimum, depression, disorientation, frustration, social withdrawal, apathy, and a sense of distorted cognition could plausibly be traced back to the loss of identity an athlete might experience upon retirement (Brewer et al., 1993; Kerr & Dacyshyn, 2000; Miller, 2009; Stephan & Brewer, 2007).

4.2. Violent tendencies

A second psychological lens that will be explored in this section is the role that reinforced violence plays on the cognitive and behavioral tendencies of contact sport athletes upon career transition. This section will begin by synthesizing an already established body of research that examines the role that reinforced violence plays in affecting athletes. As far back as the 1970s, psychologists were examining the relationship between exercise, arousal, and aggression. Zillmann et al. (1972) performed a Milgram-esque experiment on the conditions that would make it likely for a participant to perform a high voltage electrical shock to a peer. Participants were put in different scenarios and then asked to administer the shocks. One of the two groups of participants who delivered the highest levels of electrical shock to their peers were those who were aroused by exercise prior to being asked to administer the shock. The theory that can be drawn from this experiment is that sports, in particular contact sports that involve a high level of arousal and anger, make an individual prone to increased levels of hostile aggression beyond the arena of sport. Ultimately, Zillman and his team concluded that the experience of competing arouses an individual in a way that makes them less averse to violence, hostility, and antisocial behavior, perhaps even luring them toward a propensity for these behaviors after competing or training.

Kerr (1999, 2002, 2005) offers research on the potential positive effects of violence, as well as a healthy attraction to violence in contact sport athletes. According to Kerr, the International Society for Sport Psychology (ISSP) incorrectly conflated all forms of violence in sport when they offered an unequivocal stand against all violence in sport in the 1990s (Tenenbaum, Sacks, Miller, Golden, & Doolin, 2000; Tenenbaum, Singer, Stewart, & Duda, 1997). Kerr uses reversal theory (Apter, 2001) to re-conceptualize the motivational processes, social experience, attractions, and pleasures that can realistically be derived from violence in sport in a healthy manner. Reversal theory offers a model of motivation and emotion that focuses on juxtaposing motivational states and individual experiences (e.g. regarding our motivations toward rules, we are driven by a desire to conform and rebel). In the case of sport, our relationships with others are driven by conflicting desires to uphold mastery/dominance and sympathy/care. The individual's ability to change states over time is based off of the ability to fulfill his or her motives within the previous state. In other words, athletes who are able to fulfill their motives around mastery and dominance of others in sport will then be offered space to shift toward an emotional and motivational lens of sympathy toward others (presumably, outside of the arena of sport). The ability to achieve competence in one state is what allows individuals to then reexamine and shift an emotional lens to juxtaposing motivational goals. Finally, a differentiation is made by Kerr between sanctioned and unsanctioned acts of violence, as well as the type of violence that contact sport athletes engage in versus spectator violence around the sport (e.g. hooliganism in soccer).

There is also a debate in the research over whether or not directed violence in sport can lead to a substantial reduction in ensuing aggressive acts. The notion here is that violence in sport can work as a form of assertion—and, consequently, catharsis—as opposed to aggression. In other words, physical behaviors in sport may be used to establish dominance on the field as opposed to harming the opponent and, consequently, used as a form of catharsis prior to the release of wanton violent behaviors (Thirer, 1994). Only little empirical support has been provided on this theory while a larger portion of experimental data has shown little decrease (and more likely an increase) in violent tendencies after engaging in or witnessing a violent event (Arms, Russell, & Sandilands, 1979; Bandura, Ross, & Ross, 1961; Berkowitz, 1989; Goldstein & Arms, 1971).

Finally, sociological research offers literature to help understand the relationship between sport participation and violence (Goldstein, 1983), as well as concepts in social psychology such as deindividuation that may aid in conceptualizing violence in sport (Festinger, Pepitone, & Newcomb, 1952; Zimbardo, 1969). Team sports in particular can provoke a loss of individuality in the arena and, consequently, a loss of responsibility for the behaviors that one pursues. Similar to a riot, the promotion of the team or some higher purpose over the individual might open a space that provokes this individual to engage in violent endeavors and be drawn to hostile behavior in a way that eschews any sense of personal accountability. Lastly, a large portion of experimental data in social psychology and sociology has shown an increase in violent tendencies to be more likely after engaging in or witnessing a violent event (Arms et al., 1979; Bandura et al., 1961; Berkowitz, 1989; Goldstein & Arms, 1971).

The range of symptoms associated with CTE includes a large range of variability. In some cases, depression, apathy, and social withdrawal are present while in others, aggression, hostility, loss of executive function, irritability, and high-risk behavior are manifest. It seems possible that these two categories of psychological examination might account on some level for the etiology of such a wide range of symptoms. Just as athletic identity and identity foreclosure might account for the symptoms more closely associated with depression, the reinforcement of violence in contact sports may contribute in some way to the symptoms more associated with mania.

4.3. Camaraderie studies

Research has investigated the psychosocial impact that the loss of camaraderie can have on former athletes as they transition from their athletic careers. Fuller's meta-synthesis (2014) of roughly thirty years of qualitative research demonstrates how participants expressed how the loss of camaraderie was difficult to manage for former elite-level athletes and contributed to difficulties in career transition. Fuller cites several qualitative studies (Ingebritsen, 1996; Lally, 2007; Wilson, 2007) that speak to the range of difficulties athletes experienced and anticipated in their career transitions out of sport. He writes:

> Some athletes saw their retirement from college athletics as posing a threat to the positive feelings they experienced as being a part of a team. They feared that camaraderie felt with their teammates would not be repeated in other aspects of their lives. (Fuller, 2014, p. 6)

Specifically, athletes feared the loss of a unique connection that they had formed to teammates and attempted to be proactive in curbing any foreseeable deficit of solidarity. After their careers, participants began to take explicit efforts to forge close bonds to a few teammates they did not want to lose contact with, acknowledging that the same level of connection to all teammates would prove too unwieldy.

Miller and Kerr's (2002) research demonstrates how student athletes often faced a triad of tension regarding three facets of their life: athletic demands, academic demands, and social demands. It was found that student athletes almost always first compromise social engagements for their athletic and academic requirements and that when a choice is to be made between athletic and academic demands, often athletic commitments will trump academic and social commitments. As a

result of upholding sport over academic and social demands, a series of unintended consequences regarding the psychosocial development of the student athlete can occur. Most important to this study are how the student athlete might found his or herself with delayed identity development and a lack of role experimentation as a result of sacrificing academic and, especially, social demands. It seems plausible to surmise that as a result of univocal commitment to sport, the athlete's social cognition will continue to shape the way the athlete engages in and perceives the quality of his or her relationships after leaving a career in sport.

Aquilina (2013) employs qualitative methods (life story analysis and a narrative approach) to examine the "decision-making processes that gave meaning to the athletes' lives and how these decisions then affected the way these athletes viewed their world." The conclusions drawn by Aquilina demonstrate how athletes who limit their engagement in facets of their lives outside of sport—social and academic endeavors, in particular—fail to strike a balance that is essential for both successful athletic performance and healthy career transitions out of sport. It is vital that athletes acknowledge that there is simply more to life than sport and that they are able to take responsibility for establishing a balanced lifestyle while competing. It is a false dilemma to suggest that athletes must choose between having social/academic interests and upholding their obligations in sport. Ultimately, the real detriment occurs when athletes uphold a univocal commitment to sport and sacrifice social and academic interests. Again, this detriment is toward both performance *and* career transition.

Stambulova et al. (2009) offer career-planning strategies in a cross-cultural meta-analysis of the literature on athletic career transition. First, it is clear that normative (predictable) transitions from sport offer significantly less tumult to the athlete. For these transitions to be predictable, the athlete must have a raised awareness of the immanent nature of the transition. This awareness is most effective when offered by a social support structure surrounding the athlete (e.g. parents, coaches, consultants, etc.). These supports offer space to plan for retirement from sport and facilitate the transition. Likewise, social support from the athlete's significant others can facilitate a more holistic, multidimensional view of the self, as well as general academic and vocational development for the athlete in transition. Furthermore, Stambulova (2010) offers a five-step career planning strategy (5-SCP) as a counseling framework to aid the transition of athletes from a career in sport. The 5-SCP was established partly to address that the lack of social support, among other resources, that athletes lack in the face of retirement or deselection from sport. The other major influence for the 5-SCP comes from Vygotsky's developmental perspective on lifelong learning and accessing zones of proximal development for future growth. Vygotsky's model for future development rests on the need for social support, dialog, and scaffolding strategies offered by peers. In the case of offering social support to enter zones of proximal development for athletes, Stambulova suggests that sports consultants offer this psychosocial support for athletes in discussing future goals and cultivating planning skills. In short, whether the social support structure is offered through coaches, parents, consultants, or peers, the research demonstrates clearly that these structures are imperative to aiding a career transition out of sport for athletes.

Park, Lavallee, and Tod (2011) provide a meta-analysis of career transition research. In this work, psychosocial factors represent a category related significantly to the health and well-being of the athlete in retirement. Out of 29 studies on athletic career transition, 27 revealed that holding support from close relationships with peers, loved-ones, etc., all positively contributed to a healthy career transition. Several studies revealed the role that forming a coherent account of one's career and communicating that account to peers and loved one's could play in supporting a career transition (Barnes, 2002; Lavallee, Gordon, & Grove, 1997). Most importantly, the meta-analysis revealed that six separate studies demonstrated how the loss of social connection and social networks after sports contributed to difficulties in career transition (Kane, 1991). On the other hand, athletes who were able to transition into careers and situations with a more solid social support network were able to navigate a less difficult transition (Schwendener-Holt, 1994).

These studies demonstrate how the perceived lack of quality in one's relationships, underdeveloped social cognition, the loss of solidarity with one's teammates, and a limited scope of personal identity could all contribute to the onset of symptoms similar to the range found in CTE case studies (e.g. social withdrawal, depression, irritability, high risk behavior, apathy, and even suicidal ideation, among others). For the purposes of a bio-psycho-social examination, a perceived sense of alienation might cause and/or exacerbate symptoms that are associated with CTE. It is possible that this is contributing to a maladaptive social cognition, relational ambiguity (e.g. are these people *really* my friends?), and a lack of social motivation.

5. Concluding remarks

Moving forward, researchers, doctors, therapists, and the athletes themselves would benefit from a biopsychosocial systems approach when it comes to conceptualizing both the etiology of these symptoms, as well as possible intervention techniques. Recent findings in the field of neuroscience have contributed incommensurably to our understanding of the difficulties athletes face as a result of exposure to head trauma. The meta-synthesis provided in this commentary paper demonstrates just how much similarity exists in the range of symptoms associated with CTE, as well as with psychosocial difficulties faced in career transition. Coupled with the timeline often experienced by patients with CTE (little to no symptomatic presence until *after* one leaves sport), the lack of an in-life diagnosis, and the vacuum regarding a biological intervention, a broader concept to these symptoms should be applied

Despite the substantive contributions of neuroscience in discovering CTE, a systems approach should be called upon to more broadly conceptualize the range of symptoms that can occur as a result of a career in contact sports. At this current time, it is unknown precisely how CTE affects the behavior, cognition, and social orientation of the individual. It is also unknown whether or not individuals are currently living with CTE but not experiencing the same deleterious range of symptoms. Neuroscientific findings would benefit deeply from a dynamic interface with career transition studies on contact sport athletes. Until more clarity is brought to this—in particular, in the form of longitudinal and control group studies—it would be appropriate for scientists to form a broad conception of the etiology of these symptoms, as well as treatment interventions for athletes who have completed careers exposing them to head trauma.

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