TRAUMATIC BRIAN INJURY (TBI): COMPREHENSIVE REVIEW

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ABSTRACT

Traumatic brain injury (TBI) is a major global concern and due to increased surveillance and recognition of signs and symptoms is now becoming an emerging area of research around the world. While TBI is often caused by a discrete event, such as a blow to the head or blast injury, it results in a broad spectrum injury with a pathology that is poorly understood. This essay seeks to draw out the differences in not only the types, and pathophysiology of TBI but also compare the different groups that are susceptible to TBI. Furthermore this essay will start with a review of animal models used in TBI research, pathophysiology, and public health relevance and differentiate military and civilian TBI. Unlike many diseases traumatic brain injury is something that will be persistent in the population regardless of medical intervention. The more research we initiated to better understand the pathological events of TBI, the more realistic will be our chances to develop an working therapeutic intervention against the deviating consequences of TBI.

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Introduction

Traumatic brain injury (TBI) is a term that encompasses many types of head or neck injuries resulting in both morbidity and mortality around the world of TBI. With a global estimated incidence of 10 million cases annually and 1.7 million emergency room visits in the U.S alone, there is precedence to understand long term consequences of these injuries [1]. The two groups of individuals with the most elevated risk of TBI's are contact sport players (boxing, American football, rugby, ice hockey *etc*.), and military personal. Until around 1975 there had not been a generally accepted TBI classification system. Today the most commonly accepted TBI classification system is known as the Glasgow Coma Scale (GCS) [2], which measures a patient's level of consciousness based on verbal, motor and eye-opening responses after an injury. Together, these parameters are used to define the severity of the injury. GCS scores range from 0-15 and are broken down as follows: 3-8 is considered to have sustained a severe TBI, 9- 12 moderate, and >12 mild [2-5].

Mild traumatic brain injury (mTBI) is the most common form of TBI and can often go undetected; therefore the incidence of TBI may well be largely under reported. Examples of mTBI include concussion, subconcussion, and explosive blasts exposure. This is the most common type of TBI injury effecting military personnel deployed to Afghanistan and Iraq [6]. The estimated prevalence of mTBI in returning military personal range from 15.2% - 22.8%, which equates to around 320,000 individuals [6, 7]. Much of the long-term effects of mTBI's and TBI's are unclear, but studies have shown that a single TBI can produce long term grey and white matter atrophy, resulting in an increased

risk of Alzheimer's disease (AD), Parkinson disease, motor neuron disease, and many other psychiatric diseases [8].

In the next 35 years it is projected that nearly 13 million Americans will be living with AD costing the U.S. health care system approximately 1.2 trillion dollars [9]. It is expected that the prevalence of dementia in veterans will rise as a result of increased incidences of TBI's thus creating a paradox never seen before where increased battlefield survival from these singular events may have long term neurodegenerative effects later in life [10]. In the civilian spectrum, multiple concussions or other repetitive head injuries is known as chronic traumatic encephalopathy (CTE) and is observed in contact sport players [11], victims of physical abuse, and poorly controlled epilepsy [12].

Due to the increased preponderance of TBI in the clinical setting there has been growing interest in TBI research and public policy. Understanding more about head trauma and its relation to neurodegenerative disorders later in life is an issue that should be considered by anyone in the public health field. This essay seeks to bring to light the long-term effects of TBI's both in military and civilian settings to foster a better understanding of the relationship of TBI's to psychiatric disorders.

Public Health Relevance

With no genetic or serious environmental factors predisposing someone to TBI, it is a disease that can affect anyone around the globe; making it relevant to public health. As can be seen in Figure 1, there are an average of 50,000 deaths per year in the United States due to TBI and an ever increasing pyramid of hospitalizations and emergency department visits that facilitate the deaths [13]. Currently in the United States, approximately 2% of the population (5.3 million) live with a long term or lifelong disability associated with TBI. Over the past ten years the incidence of TBI has gone up by 70% while the death rate has gone down by 7% marking a great advance in both the recognition and treatment of these events [14]. The cause for an increased incidence and a decreased death rate is due in part to the advances in brain imaging, non-imaging biomarkers, and neuropathology over the past decade that has required researchers, clinicians, and policy makers to revise their views about TBI.

Figure 1. Average annual number of traumatic brain injury related emergency department visits, hospitalizations, and deaths, U.S. 1995-2001

CDC (2004)

Men are nearly twice as likely to have a TBI as women and nearly three times as likely to die from such an event [15]. This may be in part due to the effect of estrogen acting as a potent antioxidant in the context of injury, while progesterone may in fact reduce neuronal hyper excitability post injury [16, 17]. Estrogen therapy has also been found to improve a patient's outcome following TBI in experimental animal models [17].

Since 2009 all 50 states have enacted some form of legislation with regards to increased safety and TBI [18]. Most of the laws are put in place around sports, and youth sports specifically. Others introduced legislation addressing TBI in veterans, providing funds to TBI prevention or treatment programs as well as to hospitals and health maintenance organizations to provide insurance coverage for survivors of TBI. California, Illinois, Indiana, Iowa, New Hampshire, North Dakota, and Virginia all have legislation requiring the mental health and rehabilitative services program within the Department of Veterans Affairs to cooperate with localities that establish special treatment procedures for veterans and active military service members [18].

Since 1985 when Mortimer et al. first set out to determine if there was a link between TBI and AD and dementia it has been generally accepted that there is [19]. In 15 case studies evidence was found that supported an association between a history of previous head injury and the risk of developing AD. With an odds ratio of 1.58 (95% CI 1.21 to 2.67) over-all and an odds ratio of 2.26 in men compared to 0.92 in women, it can be seen that the association between AD and TBI is present and more so in males [20]. A possible explanation for the gender difference in the risk of Alzheimer's disease following head injury is the role of the female hormones, estrogen and progesterone.

Review

In the United States a TBI occurs every 15 seconds, generating 1.7 million new head injuries each year. In a civilian setting, the four most common causes for TBI are falls (35%), motor-vehicle or traffic-related accidents (17%), struck by/against (16%)(mostly sports related), and assault (10%) (Figure 2) [21]. The incidence of sport-related concussions is estimated to be 130,000 per year among children 5–18 years of age [22]. Among active military personnel, blast injury is the most common cause of TBI [6].

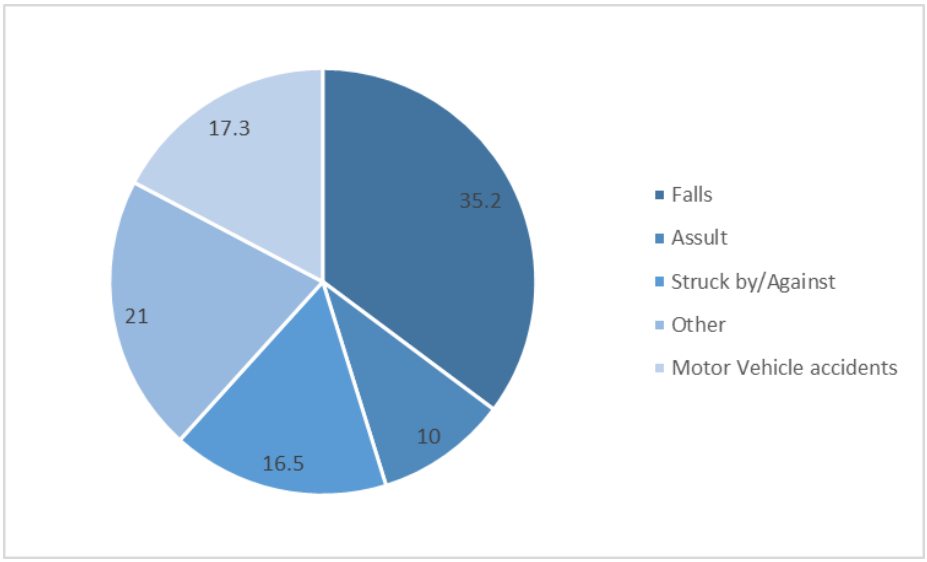

Figure 2. Estimated average percentage of annual TBI Emergency department visits, hospitalizations, and deaths in the United States. 2002-2006

One of the best ways to study TBI is through the use of animal models, and there are several experimental animal models that accurately replicate human pathophysiology. There are two main types of injuries that are studied through animal models, primary injury and secondary injury. Primary injury refers to the initial impact that causes the brain to be displaced within the skull. Secondary injuries gradually occur as a consequence of ongoing cellular events that cause further damage [23].

Animal Models

Multiple rodent models have been developed to replicate different types and severities of TBI for studying the pathological cascade of events and possible therapeutic approaches. As seen in Table 1 there are two basic types: closed head injuries (CHI) and open head injuries (OHI). Closed head injuries are the easiest and simplest models of TBI as there is no need for a craniotomy. There are three main types of CHIs, impaction, rotation, and blast models. Impaction is the most common because it is simple and effective at recreating the most common types of TBIs. One of the biggest downfalls of the impaction model is the inability to remain consistent in the exact impact site and severity between mice. The blast CHI is used primarily in military studies to help discern the effects of different types of explosions on the brain.

Table 1. Summary of the most commonly used animal models of TBI.

TBI model	Advantages	Disadvantages
Closed head injuries (CHI) [24]		
Impact/acceleration	Simple Inexpensive Immediate behavior observation No craniotomy	Rebound injury High variability Skull fracture
Head rotation	Mimics many neuropathological features of human TBI	No mouse model available
Blast [25]	Relevant to military TBI No craniotomy	Not yet widely disseminated
Open head injuries (OHI) [24]		
Fluid percussion	Graded levels of injury Good control of injury parameters	Craniotomy required Variable outcomes between technicians No immediate behavioral observation
Controlled cortical impact (CCI)	Graded levels of injury Acute and reliable control of many parameters No rebound injury	Craniotomy required No immediate behavioral observation

A blast TBI is fundamentally different than the other types. When a blast goes off, a wave of over-pressurization radiates outward followed by a wave of under-pressurization that causes objects to be displaced. The model to replicate this works by creating a similar wave in a sealed tube that a mouse is placed in where the blast can be controlled both in its wave amplitude as well as duration.

In a study by Goldstein et al. a wave was generated to be consistent with a blast from a 5.8 kg trinitrotoluene (TNT) bomb 5.5 meters away [25]. When conducting a blast experiment the mouse should have a full range of movement in the head and neck region, as it is often the sudden movement or jostling that causes some of the injury.

Open head injuries offer a much greater level of control, but at the cost of many other factors. Fluid percussion injuries are created by a pocket of pressurized water hitting the brain through craniotomy. A thin membrane is placed on the craniotomy and then connected to a tube of water that can be contacted on the opposing side to create waves of energy that travel through the tube and contact the brain. CCI injuries occur in a similar fashion with the main difference being that an air-driven or pneumatic piston is fired through the craniotomy.

There are many behavior experiments that can be run on animal models of TBI to better understand the behavior changes that follow the injury. Some of the most common approaches are: forced swim test, elevated plus maze, fear conditioning, and Barnes maze. In the forced swim test, for example, the rodents are placed in a tub of water with no platform to stand on and the time is recorded until the animal gives up. This is an indication of the willingness of the animal to survive and is a good indication of depression [26].

Fear conditioning is a paradigm that helps to understand how an animal learns in a new situation. This is performed by placing the rodent in a small chamber and playing an auditory cue followed by an electric shock. In a series of conditions and recordings, after a period of learning, the experiment determines how the animal responds to the auditory cue. It is the animals' natural reaction to freeze when placed in a situation they feel eminent danger and trapped so the total time of freezing after the auditory cue can be measured and used to determine novel learning [26].

One of the biggest disconnects between animal models and human clinical trials lies in the outcome after therapeutics. Many promising neuroprotective drugs have been identified in animals TBI models, but all have failed in phase II or III clinical trials [27]. The most common types of therapeutics seen now fall into three categories; neuroprotective, neurorestorative, and anti-inflammatory agents. Some neuroprotective and neurorestorative approaches that are currently being looked into are; calcium channel blockers, corticosteroids, free radical scavengers, satins, and environmental enrichment [27].

Pathophysiology

Better understanding the pathophysiology of a TBI is necessary for the development of new therapeutics and procedures for treating the condition. To better understand the pathophysiology in multiple compared to a single TBI exposure, Petraglia et al. used CCI model to induce multiple TBI and to determine its association to CTE (figure 3). The study compared single and multiple TBI across different time points from the impact to determine if there were any noticeable changes in astrocytes, microglia, and tau proteins.

The single TBI mice were impacted once and then sacrificed at intervals of 7 day, 1 month, and 6 month time periods. The multiple TBI mice were impacted 6 times a day for 7 days and the sacrificed in that same time frame as the single impact mice.

The results of this study showed that brains from a single and multiple TBI lacked macroscopic tissue damage in all time points. In the single impact animals there were persistent astrocytosis at 7 days post injury that resolved in the later time points, while the repetitive TBI mice demonstrated widespread astrocytosis through the 6 month time point. The same pattern of changes occurred with phospho-tau proteins and microglia activation, elevated by immunostaining. The single impact mice demonstrated immunoreactivity at 1 month and no detectable changes at the 6 month time point, while

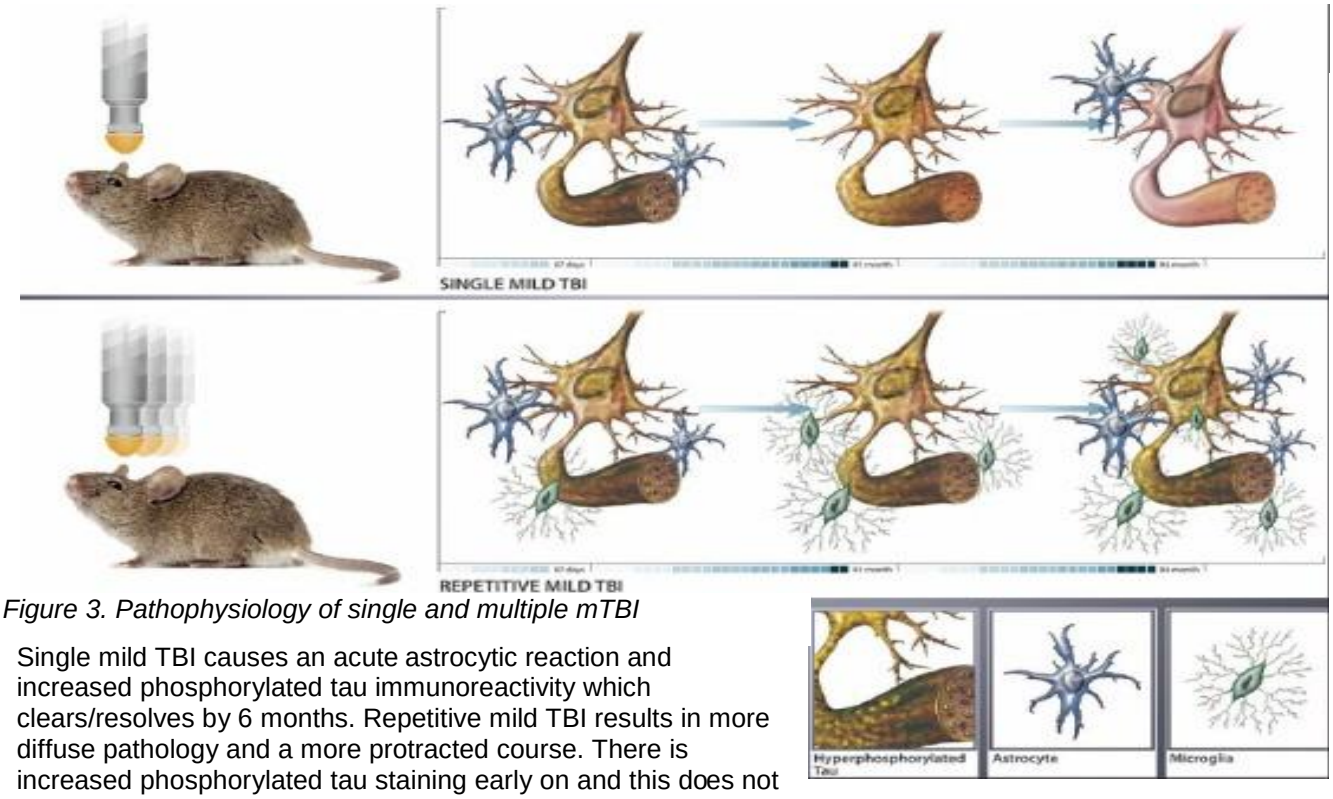

Figure 3. Pathophysiology of single and multiple mTBI

Single mild TBI causes an acute astrocytic reaction and increased phosphorylated tau immunoreactivity which clears/resolves by 6 months. Repetitive mild TBI results in more diffuse pathology and a more protracted course. There is increased phosphorylated tau staining early on and this does not clear by 6 months post-injury. Microglia are activated early on as well and progressively increase through 6 months.

Petraglia (2014)

the multiple impact mice had elevated levels of both, phosphor-tau and signs of activated microglia, through all time points [28].

Normal brain function is controlled by neuro transmitter-mediated activation of receptors and controlled ionic changes in the post synaptic membranes of the neurotransmitter releasing cells. These changes are closely regulated by sodium-potassium (NA+ / K+) ATPase pumps. A disruption in the concentration of either of these ions in the brain can have dramatic effects. The effects and concentration changes of some major chemical components can be seen in figure 4 [26].

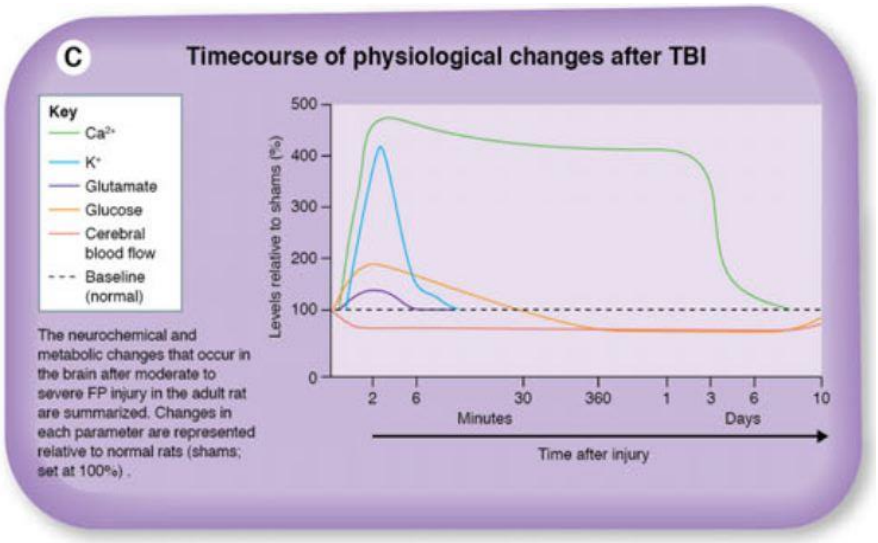

Figure 4. Time course of physiological changes after TBI.

Prins (2013)

Within the first hour after a TBI there is a substantial increase in the amount of calcium (Ca+), potassium (K+), glutamate, and glucose inside the cell body of an effected neuron [23]. The increase of glutamate from presynaptic terminals disrupt the equilibrium on the postsynaptic membranes, which is accompanied by an increase in K+. The amount of potassium that is released increases with the severity of the injury, thereby creating a larger differential in the K+ and NA+ equilibrium with more traumatic injuries. In addition

to an increased K+ there is a massive increase in the amount of Ca+ that can be observed after a TBI. Excess Ca+ induces mitochondrial Ca^{2+} uptake, and overloading of this pathway has been shown to induce oxidative stress and impair mitochondrial function [23].

A single TBI injury can lead to a constellation of events that include axonal injury, impaired cerebral blood flow, metabolic changes, edema, raised intracranial pressure (ICP), increased blood-brain barrier (BBB) permeability, calcium influx, elevated oxidative stress, free-radical-mediated damage, excitatory neurotransmitter release, inflammation and cell death. It is assumed, and been proven that multiple TBIs can compound the problem and make it harder to recover from these adverse events [24].

There is mounting evidence of a link between TBI and Alzheimer's disease (AD) developing later in life. A recent meta-analysis of case control studies found that individuals with a history of TBI were 60% more likely to develop AD compared to others [29]. Within a population of AD patients it has also been reported that TBI accelerates the onset of AD [30, 31]. There is also the possibility that the effects of TBI in the context of developing AD, vary depending on the individuals genetic background. The most notable of these is the presence of allele e4 of Apolipoproetin (APOE) [32]. APOE is a plasma lipoprotein that is responsible for the transportation of lipids through the CNS, which plays a major role in the remodeling, repairing, and maintaining of neurons [33]. A study by Mayeux et al. reports that carriers of APOE e4 allele who have had TBI have a 10-fold increased risk of AD, while carriers of the same allele but no TBI history had no increased risk [33].

TBI is a complex dynamic process that begins a multitude of cascades of pathological cellular pathways, some of which are still unknown. The symptoms vary with each individual, injury type, injury severity, age and gender, making it a challenge to diagnose, treat and understand. Research efforts to understand the common underlying responses to TBI have the potential to provide further therapeutic options for early intervention [23].

Military / Civilian Setting

There are many key differences between the military and civilian setting. Along with the increased exposure to TBI generating settings there are a plethora of compounding factors present in a military setting not found in the civilian spectrum. In a combat setting, brain as well as other injuries frequently occur in drawn out settings or battles where there is no clear single event that may have caused the damage. In a civilian setting a TBI is nearly always a discrete event that can be easily tracked to its source, and used to help diagnose and treat the injury [1].

Civilian TBI cases often have access to medical care available to them in a short amount of time after the injury occurs. On the other hand military personnel are often on missions for long periods of time and do not have access to non-essential medical care until they return to base. Members of the armed forces that experience combat TBIs are also often operating under high levels of physical and emotional stress. This can impact the soldier's ability to recognize and self-diagnose possible brain related trauma that they have been exposed to, resulting in an interference with the ability to recall actual events leading to the injury when questioned later.

TBI in a military setting can come from multiple origins, but the main difference from the civilian TBI exposure is blast related. The primary injury from blast is a result of the overpressurization shockwave associated with high energy explosions. Secondary, and compounding, injuries can result from impact flying debris, or the soldiers' impact with a stationary object, Table 2. The shockwaves emanated by a blast can travel at the speed of sound and have the potential to be multiplied by reflection. The damage caused by this mechanism is thought to differ considerably from that of a traditional TBI [34].

Table 2. Immediate effects of blasts and explosions

Primary –	direct effects (overpressuration and underpressurizations) Rupture of tympanic membranes Pulmonary damage Rupture of hollow viscera
Secondary -	penetrating trauma Fragmentation injuries
Tertiary -	effects of structural collapse and of persons being thrown by the blast wind Crush injuries and blunt trauma Penetrating or blunt trauma Fractures and traumatic amputations Open or closed brain injuries
Quaternary -	burns, asphyxia, and exposure to toxic inhalants

Due to an increase in the association of TBI and military settings, the DOD has adopted a common definition of brain injury:

"Any traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force that is indicated by new onset or worsening of at least one of the following clinical signs, immediately following the event:

1. Any period of loss of or a decreased level of consciousness

2. Any loss of memory for events immediately before or after the injury

3. Any alteration in mental state at the time of the injury (e.g., confusion, disorientation, slowed thinking)

4. Neurological deficits (e.g., weakness, balance disturbance, praxis, paresis/paraplegia, change in vision, other sensory alterations, aphasia) that may or may not be transient

5. Intracranial lesion" [35]

A majority of military related TBI are associated with high energy explosions which affect the brain in novel ways that are rarely seen in the noncombatant environment. This along with the fact that military personnel are often deployed multiple times to combat zones [36, 37] which can expose them to multiple TBIs makes soldiers very susceptible to receiving one or more TBI while serving.

Analytical

In the last 50 years the topic of TBI has grown in interest in the eyes of the public but also in the scientific community. Figure 5 shows the number of published articles available in PubMed retrieved by the search term “traumatic brain injury”. Approximately every five years the number of articles published doubles as more and more people help to uncover more information about the effects of TBI.

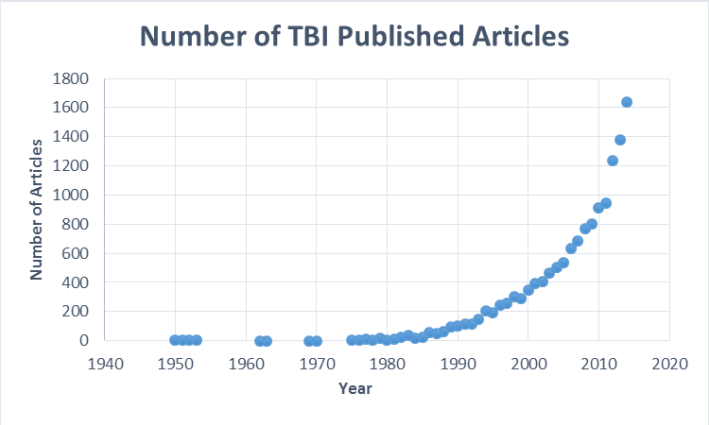

Figure 5. Number of TBI published articles

PubMed.gov (2015)

Military Setting

One of the largest neurological disorders present in the military is post-traumatic stress disorder (PTSD). While a full understanding of this disorder is not known, it is of great concern when coupled with TBI because the two may have compounding effects on each other [38].

The relationship between PTSD and military services has been well documented. However the relationship between TBI and PTSD is a much less understood area of PTSD. Mac Donald et al. looked into prospectively assessed clinical outcomes relating to

TBI of US military personal and found there was little relation to the severity TBI and the severity of PTSD. This was looked into as part of a larger goal of determining if blast related TBI had a different effect than impact related TBI on a soldier.

Figure 6 A shows documentation of individuals who either had a TBI or did not, as well as individuals that were exposed to a blast or not. The blast plus impact TBI was statically different from its nonblast counterpart. The scale used to determine the combat exposure intensity is called a combat exposure scale (CES), ranging from 0-41. The higher the number the worst the combat situation the soldier was exposed to. The blast plus impact TBI group has a much higher CES score than the other two groups showing the correlation between combat intensity and likelihood of TBI. In part B of Figure 6, a positive correlation was found between CES score and the Clinician-Administered PTSD Scale (CAPS) total score. This means a higher CES score directly relates to a higher CAPS score, or more intense combat equates to more intense PTSD. In part C the same analysis was done as in part B but on the TBI individuals. There was no relation between

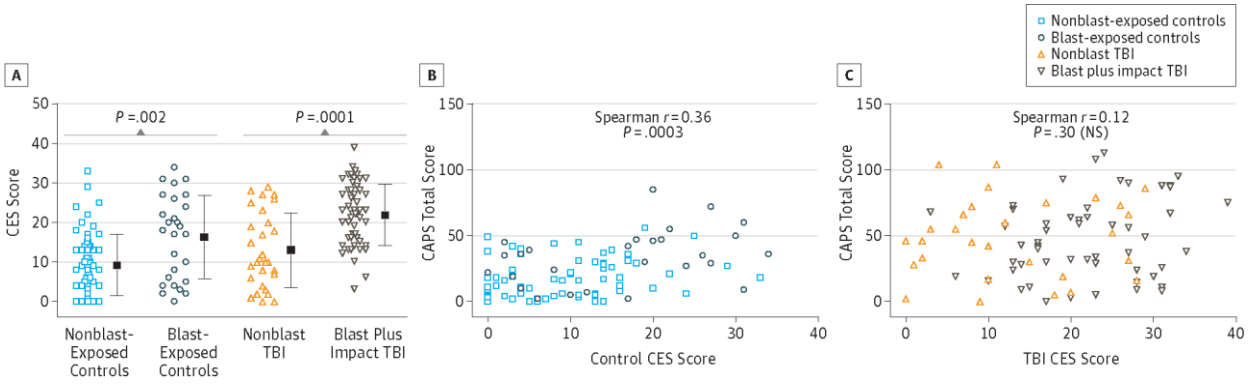

Figure 6. Blast vs. non-blast TBI

A, Combat exposure intensity was assessed by the Combat Exposures Scale (CES). Higher scores indicate greater self-reported combat exposure (maximum score, 41). *P* values were calculated using 1-tailed Mann-Whitney test and were reported if significant after correction for multiple comparisons at $P < .0125$. B, A positive correlation was found between the Clinician-Administered PTSD Scale for *DSM-IV*(CAPS) total score and the combat exposure intensity measured by the CES in control subjects. C, In contrast, no correlation was observed between the CAPS total score and the CES score in the traumatic brain injury (TBI) groups. NS indicates not significant.

Mac Donald (2014)

the CAPS total score and the CES score. This shows the TBI is a modifier of PTSD and while there is no clear trend from this data there is evidently some relationship between receiving a TBI and the severity of PTSD later on. By expanding this to the entire study about blast vs. nonblast TBI, they found that the blast plus impact TBI and nonblast TBI groups were essentially indistinguishable with regard to clinical outcomes at 6 to 12months after injury.

Outside of PTSD there are still many military specific aspects related to TBI, the most common of these being blast injury. Blast exposure is rare in civilian life, but is a commonality in today's military involvements. In two separate studies using rat models researchers looked at blast induced TBI via differing levels of blast pressure and respective cognition outcomes.

Ahlers et al. looked specifically at the overall vulnerability of brain to blasts and concluded that it was very much dependent on how the blast wave impacts the head. This encompasses the angle, force and duration of the overpressuration [39]. A second study by Gama Sosa et al. concluded the same thing, but neither of the studies were able to produce and tangible link differentiating between blast exposure and impact TBI [39]. Exposure to a blast inducing TBI seems to cause similar symptoms and long term effects as an impact TBI does. The disparity with blast TBI may come with the fact that they are often accompanied by an impact TBI which would in theory exacerbate symptoms which was something not looked at by either Gama Sosa or Ahlers.

The symptoms of brain injury can manifest themselves in many different ways and at many different times. Based on the veteran population Gama Sosa et al. looked at, the total number of reported TBI related issues that the patients were having, symptoms were

broken down into four clusters: affective, somatic, vestibular, and cognitive. Affective covering: low frustration tolerance, irritable, anxious/tense, depressed/sad, fall/stay asleep, and fatigue. Somatic covering: Light/noise sensitivity, vision problems, headache, nausea, numbness/tingling, taste/smell, and pain severity. Vestibular covering: Loss of balance, dizzy, and clumsy/poor coordination. Finally cognitive covering: Cannot get organized/finish things, poor concentration, forgetfulness, and difficulty making decisions [38].

Figure 7 shows the mean number of symptoms broken down into these clusters. The veterans with no history of TBI had an average of 9.4 total symptoms, while the veterans with TBI had 16.7. Affective and somatic were the two most reported symptom clusters in both populations [40]. This study shows that all across a variety of symptoms there is a connection in the military realm between TBI and greater mental strain later in life.

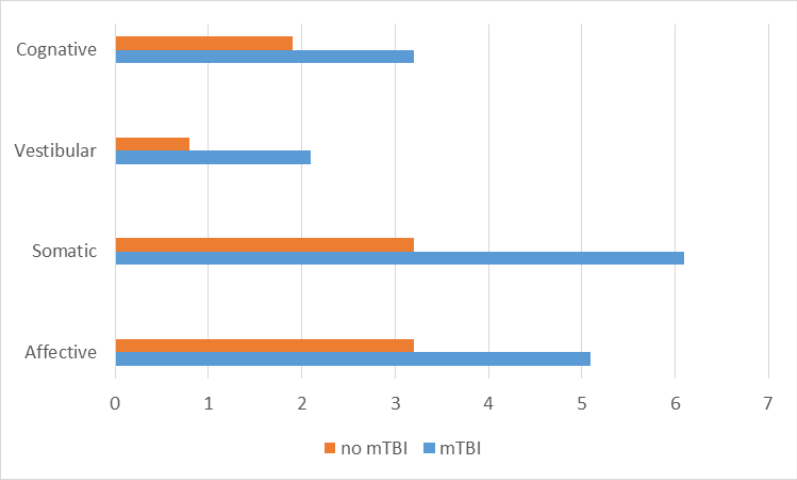

Figure 7. Mean number of symptoms: mild traumatic brain injury (mTBI) versus no mTBI.

Civilian Setting

In the United States alone each year, approximately 1.6 – 3.8 million sports related concussions occur; most of them suffered by football players [41]. This does not include the spectrum of neurological damage that is sub-concussive but may still may cause neuronal dysfunction. Though the immediate effects of head injuries are a concern, the lasting effects are of concern as well with the primary concern being the neurodegenerative disease known as chronic traumatic encephalopathy (CTE). CTE is marked by accumulation of hyperphosporylated tau protein, leading to neuronal destruction and the resulting symptoms. The clinical features of CTE can be shown in many ways but are most commonly associated with mood, behavioral, and cognitive impairments that often result in progressive dementia.

CTE was first described nearly 70 years ago but much about the disease is still unknown and there is not a current in vivo diagnosis possible. This makes research into the topic very challenging and consequently it has been an understudied disease in the past. Thankfully there is a distinctive neuropathological profile of the disorder lends promise for future research into its prevention, diagnosis, and treatment.

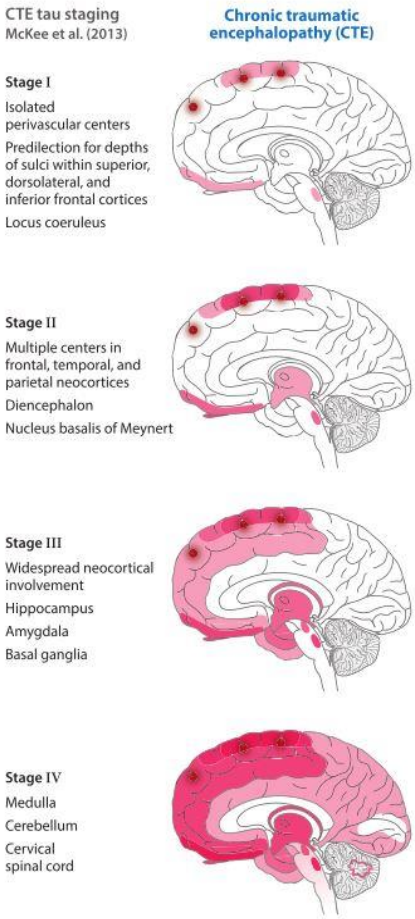

Figure 8. Multiple stages of CTE, and what areas of the brain they effect.

McKee (2014)

Figure 8 shows the multiple stages of tau expression in CTE. Over time a person suffering from CTE will progress from stage I to stage IV, though on a different time scale for every person. In stages I and II the tau protein expression is mostly localized to the frontal and temporal lobes. This can lead to symptoms reflective of frontotemporal dysfunction, including behavioral and cognitive deficits and a dysexecutive syndrome [42]. By the time a patient has reaches stage IV there is widespread cerebral involvement including the medulla, cerebellum, hippocampus, amygdala, and spinal cord. Once a person suffering from CTE reaches this stage there is little left in full control and the brain will slowly shut down.

Looking at the studies presented and the countless ones not mentioned here, it is clear that TBI is a growing area of concern for many people groups around the world. Not only is it being looked into more often, but the more it is researched the more intricate and elusive an answer to the question of safety before or after a TBI becomes.

Conclusion

In recent years the research on TBI has grown dramatically. This is a great step forward but there is still much unknown about the effects of TBI on the human brain and pathological conditions that follow TBI.

While we begin to understand the immediate effects of TBI, and limited therapeutic approaches help to combat some of the acute symptoms, the long term effects following a TBI are mostly unpredictable and incurable. The more we learn about molecular mechanisms of TBI, immediate and long term effects of brain injuries, the more opportunities we will have to design, develop and apply target therapies to prevent long term complications of TBI that most probably end up with severe dementia and a substantial risk for CTE and AD.

One area of concern is TBI in sports and in the military being unreported based on the idea that it would show weakness or remove them from any possible involvement to come. In both of these groups there is a well-established pretense that gives rise to the idea that if an injury occurs (regardless of how mild) it is best to handle it yourself if possible. If this dogma can be overcome and self-reporting increases there is a much better hope for accurate data collection and treatment at an early stage in possible prevent any long term symptoms.

As a former contact sport player, I have seen this first hand. It would take immense effort in order to uproot this idea, but I believe it can be done. If an intervention were to be implemented at an early age, and children made aware of the effects of TBI there is hope for a change. If young athletes share knowledge about potential negative effects of

TBI, than the collective team may be able to make better decisions than any single player alone.

In the military theater there are many of the same possible interventions to help reduce the long term effects of TBI as in the sports realm, but also a few novel ones. One of these unique protection methods would be a badge that is attached to every soldier's helmet that would indicate in some way that they had been through a blast that would be violent enough to cause damage. The only way to get a replacement badge on your helmet would be from a doctor that could quickly run though some cognitive tests to make sure no unseen damage has occurred. In this way all of the soldiers would all know if the badge gets set off they need to see a doctor when they get back to a base before they can be sent out again.

As always, increasing the protective equipment of people who are putting themselves at risk for a TBI is always a concern. For the sake of this paper it is assumed that protective measures like that will be put into practice regardless, and thus will not be discussed.

Unlike many diseases, traumatic brain injury is something that will be persistent in the population regardless of medical intervention. This is a topic that should be continually researched being that it is probably never going to be removed from the population. However it can be reduced, and managed much more effectively than it currently is thus allowing for a healthier population.

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