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FROM INJURY TO IMPRISONMENT: HOW TRAUMATIC BRAIN INJURY CAN LEAD TO VIOLENT CRIMINAL BEHAVIOR

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Introduction

According to accumulated national data, the United States sees an average of more than 1.4 million cases of traumatic brain injury (TBI) per year, resulting in more than 1.1 million emergency department visits (Frieden, Houry, & Baldwin, 2015). Of these cases, roughly 50,000 lead to death (Langlois, Rutland-Brown, & Wald, 2006). About 87% of those seen for brain injuries are treated and released, while another 11% are hospitalized and treated. Furthermore, there are an estimated 5.3 million people in the United States living with various types of persisting disabling conditions ranging in severity due to past brain injuries (Shiroma, Ferguson, & Pickelsimer, 2010). However, in reality these statistics are an underestimate of the true number of traumatic brain injuries in the United States, as many people don’t receive care following an injury for many reasons including lack of healthcare or poor access to the proper resources. These numbers also do not include various private institutions and military facilities which could raise the statistics dramatically (Langlois et al., 2006).

Traumatic brain injury is an important national public health problem whose severity often goes unrecognized in our country. A traumatic brain injury is defined as any injury that disrupts the normal functioning of the brain for a period of time. These injuries can be caused by jolts or blows to the head, which are considered closed head injuries. Open head injuries exist as well and can be caused by objects penetrating the skull. (Frieden et al., 2015).

It is important to note that head trauma does not affect all populations evenly. A disproportionate amount of treated TBIs is attributed to adults aged 75 and up, who are at a higher risk of TBI-related hospitalization as a result of falls or other accidents (Frieden et al., 2015). Further, it was seen that those discharged from the hospital following a brain injury are 7.5 times more likely to die within the first year after hospitalization than those without a TBI.
(Shiroma et al., 2010). Another affected population are male youths, who are twice as likely to experience a traumatic brain injury as females of the same age range, most likely caused by high-contact sports and other activities. These injuries have been known to be associated with higher levels of impulsivity and negative emotion ratings in male youths following the trauma (Vaughn, Salas-Wright, Delisi, & Perron, 2014; Emerson, 2008).

A brain injury can result in many related and lasting health effects depending on the gravity of the incident. A significant portion of brain injuries are followed by a period of unconsciousness, which is often helpful in indicating the extent of the trauma. However, the most significant brain damage does not always occur at the moment of impact but rather may emerge at a later point in life as the brain continues to adapt and develop. There is a wide spectrum of symptoms that may continue well past the time of the injury (Hume Adams, Graham, Scott, Parkert, & Doyle, 1980). In order to better locate the best times for treatment and rehabilitation to ideally reduce these symptoms, researchers have identified the most crucial stages surrounding the occurrence of a TBI which will be discussed further (Chesnut et al., 1999).

The most common symptom of traumatic brain injury is cognitive dysfunction, characterized by an impairment of attention, memory, or other executive function (Riggio & Wong, 2009). The impact an injury has on these forms of higher thinking is incredibly crucial to understand so the proper treatment can be administered and a victim’s health can be monitored. Brain injuries can further lead to personality changes, psychological deficits, and behavioral disorders which may cause a domino effect of other issues. The most notable change to be focused on here is a development of poor anger control and similar mood problems which lead to aggression and violence, specifically those which give rise to criminal behavior.
The estimated prevalence of traumatic brain injury in the general population is about 8.5% in the United States (Silver, Kramer, Greenwald, & Weissman, 2001). However, it can be inferred that prison populations across the country experience a higher rate of individuals with traumatic brain injury as a result of the loss of anger control that many TBI victims experience. The United States currently has over 2 million people residing in prisons and jails across the country (Bronson & Carson, 2019; Center for Disease Control and Prevention (CDC), 2009). The current literature review aims to study the prevalence of brain injuries across these imprisoned populations.

The results of this review can have serious implications in law, medicine, and rehabilitation services. A traumatic brain injury can have significant influence over criminal justice procedures including ability to stand trial and proper sentencing depending on the timing and intensity of the offense. In addition, TBI prison prevalence could be implicated with the future of medicine and treatment protocols for individuals who suffer head trauma. Finally, the discussion of head injuries in prison populations leads to the need for analysis of proper rehabilitation services for TBI victims and of protocols for societal reintegration of those released from prison with such injuries. By studying the prevalence of traumatic brain injury in imprisoned communities, one can gain a deeper understanding of the problems that are closely intertwined with such important health issues.

Aspects of Traumatic Brain Injury

As mentioned previously, a traumatic brain injury can take many different forms. Open and closed head injuries can cause different types of trauma to the brain and the resulting issues can range in severity. There are countless instances which can lead to a TBI including falls, assaults, car accidents, blasts, being struck by objects, and more. The percentages at which these
injury mechanisms occur varies with age groups. For example, falls are the leading cause of TBI in the youngest age bracket (children 0 to 4 years of age) and the oldest age bracket (adults aged 65 and older) according to researchers. In the two age groups which collectively span from 15 to 44 years of age, mechanisms including assaults, falls, and traffic accidents are nearly equal (Wood & Bhushan S, 2015).

The abrupt incident of head trauma is called the primary injury. This is the moment of impact that a victim suffers an immediate insult to the brain. In most cases, there is a specific primary injury that occurs, although there are cases where repeat minor injuries such as concussions confound to add up and create changes in the brain after a certain tipping point. Following the primary is the secondary injury, which is considered any physiological or biomolecular change made in the brain as a result of the primary injury. A secondary injury can occur at any time after the original incident. Brain injuries can also be classified as either focal or diffuse. Focal damage is confined to one spot in the brain, usually a particular structure. Diffuse damage is more complicated because the trauma can spread or affect other linked regions of the brain in addition to the immediate injury (Riggio & Wong, 2009). Because of this, victims of head trauma should monitor their health in the months following an incident in order to determine if the damage becomes more widespread over time.

Because brain injuries are such complex instances, many researchers use varying definitions of TBI to understand the scope of the trauma. It is important to differentiate between the levels of traumatic brain injury- mild, moderate, and severe. A mild injury usually consists of minor vomiting and dizziness, lethargy, some temporary memory loss, and brief (if any) loss of consciousness (LOC). A moderate injury becomes more serious including unconsciousness up to 24 hours following the impact, contusions or brain bleeding, and some signs of trauma when
neuroimaging tests are performed. A severe brain injury is generally defined as unconsciousness exceeding 24 hours (coma) and a loss of the sleep/wake cycle during LOC. Clear signs of injury are also present in neuroimaging scans, and a period of memory loss following a regain of consciousness is expected (Frieden et al., 2015).

Another way to define brain injuries is through use of the Glasgow Coma Scale. This measurement uses three parameters to define the severity of a head injury. The first standard is based on an eye-opening response, where different reactions to visual stimuli are graded from 1 to 4 on increasing reactivity. The second standard is based on a verbal response graded from 1 to 5 with increasing levels of oriented conversation. The final standard is centered on a motor response graded from 1 to 6 with increasing levels of obedience and purposeful movement in response to pain. Based on the scale, a mild head injury would have a cumulative score between 13 and 15. A moderate head injury would result in a score ranging from 9 to 12, and a severe head injury would be a score of 8 or lower* (Jones, 1979). For reference, a coma would provide a score lower than 8 with a definite lack of eye-opening response, no ability to follow commands, and a lack of word verbalization. An example of such grading usage can be found in a study published in 2010, showing that lower GCS scores indicate more damaged brain regions (Kasahara et al., 2010). These scores provide relative indications of the level of damage patients endure and can offer insight into what areas of the brain should be focused on for TBI treatment depending on the scoring of certain categories.

Due to the varying severities of head trauma, symptoms are not identical across all cases. Brain injuries can cause physical, emotional, cognitive, and behavioral impairments depending on the location of the incident in the head. Symptoms of these injuries are categorized into two types- somatic and neuropsychiatric. Somatic symptoms are physical representations of the

* A version of the Glasgow Coma Scale is attached in the appendix.
damage to the brain and can range in intensity. The most common somatic symptom is a resulting headache. Acute posttraumatic headaches can begin within two weeks of the injury and last up to two months, and chronic headaches can persist longer than 8 weeks following the incident. The second most common somatic symptom is dizziness or nausea caused by the damage, and fatigue closely follows these. Other important symptoms to monitor include sleep disturbances, visual disturbances, hearing loss, seizures, and sensitivity to light and sound (Riggio & Wong, 2009). More extreme somatic symptoms can include brain swelling as a response to injury, hypoxic damage if oxygen supply is interrupted, or a subdural hematoma with blood collection outside of the brain (Hume Adams et al., 1980).

Perhaps more concerning than somatic symptoms are neuropsychiatric symptoms which manifest in either cognitive deficits or behavioral disorders. Cognitive dysfunction, including impairment to attention, memory, or executive function, can vary among different types of injury. These deficits generally resolve themselves within a matter of days following the incident and rarely last more than three months in victims of a mild TBI. However, more serious cases of brain trauma can have lasting cognitive problems. Different periods of loss of consciousness and posttraumatic amnesia following brain damage are known to be decent indicators of future cognitive deficits (Riggio & Wong, 2009).

**Mental Disorders as a Result of Traumatic Brain Injury**

Behavioral disorders can appear as a result of preexisting psychiatric disorders or as a secondary issue to cognitive disorders following injury. For certain disorders, there are specific critical periods during which the development of a post-injury disorder is most likely. For instance, development of major depressive disorder generally occurs between 3 months and a year post-injury (Riggio & Wong, 2009). This occurrence is much more likely in cases of severe
injury; roughly 24.5% of TBI victims experience depression, more than twice the prevalence of the general population. Unfortunately, this rate also happens to be positively correlated to socioeconomic factors including minority status, unemployment, and low income (Rogers & Read, 2007). Further studies have found that suicidality is linked to TBI, with about 8.1% of participants suffering an attempt following an injury with a loss of consciousness compared to a 1.9% general population prevalence (Riggio & Wong, 2009).

In addition to depression, TBI victims are also at a higher risk of developing substance abuse disorders. About 22% of participants in a study reported cases of substance abuse, about 1.3 times more than the national population (Riggio & Wong, 2009). Researchers also discovered that in all ranges of severity, risk of substance abuse in patients without pre-existing psychiatric disorders was highest in the first 6 to 12 months following a TBI, but luckily decreases following the second and third years post-injury (Rogers & Read, 2007).

Other mental disorders are known to increase in prevalence following brain trauma, among them generalized anxiety disorder (GAD) and post-traumatic stress disorder (PTSD). TBI victims are at an elevated risk for GAD for years following incidence, but risk for PTSD declines steadily as time after injury continues (Riggio & Wong, 2009). However, TBI victims are still more likely to develop these disorders at a higher rate than the general population, 16.5% for PTSD and 9.1% for GAD (Rogers & Read, 2007). These disorders come with a variety of symptoms including delusions, hallucinations, irritability, irrational behavior, and mania. The biggest indicator of disorder development is most clearly personality changes, which seem to be present in almost all mood and mental disorders following traumatic brain injury.
Pre-existing Mental Disorders and TBI

Many disorders have a complicated relationship with traumatic brain injury. It is important to note that not all disorders arise after an injury, and many are actually present prior to head trauma. Some pre-existing disorders have a direct effect on how a brain injury will affect a person, and some correlate to instances which present greater opportunity for TBI risk. For example, aggression scores in TBI victims with intermittent explosive disorder were higher than those of non-disordered control participants. The same was true for those with antisocial personality disorder, and both these results were significant for those with mild to moderate cases of brain trauma (Ferguson & Coccaro, 2009). Increased aggression levels are a common trend following a traumatic brain injury, and the following studies presented will be discussed in the context of violent criminal offenders who experienced a TBI.

Antisocial personality disorder has been known to be paired with borderline personality and other traits including higher rates of alcoholism than the general population. Alcoholism is a disorder with a complicated relationship to both traumatic brain injuries and aggression. One study found that a sample of violent offenders had a drinking problem and scored twice as high as controls on the self-survey scoring list known as the Michigan Alcoholism Screening Test. Though this test is an introspective survey, the results allow researchers to better identify drinking behaviors which can then be correlated to related brain regions. These scores further reflected elevated feelings of alcohol-related mood dysphoria in the test subjects. Similarly, 44% of these offenders regularly used drugs and other tranquilizers, about three times as often as the control group. Many of the drugs mentioned are known to induce paranoia and psychoses, which often lead to violent outbursts and are a large concern when combined with a prior brain injury (Langevin, Ben-Aron, Wortzman, Dickey, & Handy, 1987). History of substance abuse like this
as well as intoxication at the time of a brain injury have both been found to lead to more complications and longer hospital stays following head trauma. In addition, greater intoxication led to poorer discharge status from the hospital, and history of substance abuse was associated with higher mortality rates and greater likelihood of repeat injuries (Corrigan, 1995).

Abuse is another complex situation in regard to traumatic brain injury. Physical and sexual abuse are common causes for neurologic abnormalities, and many cases of brain trauma result from domestic abuse scenarios. One study found a documented history of prolonged physical abuse in nearly 84% of individuals on trial for violent offenses (Blake, Pincus, & Buckner, 1995). Head injuries can also be significant predictors for partner-abuse. Marital aggression was related to previous head trauma in a study focused on men proven to be wife batterers. More than half of the men had a history of at least one TBI prior to their placement into a group treatment program, indicating that head trauma can lead to unwanted increases in aggression (Rosenbaum et al., 1994).

Women on the receiving end of abuse who suffer traumatic brain injuries also show neurologic abnormalities related to increased violence. A study done with a sample of female prison inmates found that 95% of them had some sort of neurologic history before their criminal offenses, and 42% of the subjects reported at least one TBI with a loss of consciousness, most as a result of physical abuse. Many of these injuries were found primarily in the frontal-temporal region of the brain, which was also correlated to lower cortisol levels in the violent female offenders. The study found an average of two TBIs with loss of consciousness per each offender (Brewer-Smyth, Burgess, & Shults, 2004). These high numbers in female prisoners, many of whom were victims of abuse, show how damage to the head can influence aggression levels and lead to further complications following trauma.
Overall, TBIs are generally paired with other organic diagnoses and neurological abnormalities. In a hospital catered toward mentally-disordered violent offenders, about a quarter of male patients had histories of damage to the head, and 45% of those victims reported a loss of consciousness. Many of these individuals reported their brain damage to be paired with disorders such as schizophrenia, dissociative disorder, and depression (Martell, 1992). These varying rates of traumatic brain injury across different populations of violent offenders and mentally disordered individuals proves that the problem with brain damage can become increasingly more complex when other variables such as substance abuse, battering, or other disorders are added into the mix.

Brain Regions Related to Violence

It is known that the brain has certain regions of functional specificity and that some specialized areas of the brain have particular responsibilities. Aggression is often cited to be a result of changes in only one or two areas, but realistically it can be influenced by many regions working together. One of the most popular brain structures to study in relation to violence is the amygdala, a small component of the limbic system found in the anterior portion of the temporal lobes.* The amygdala is generally referred to as the emotional regulator of the brain, and plays a role in behavioral control as well. It is one of the largest players during a fight-or-flight situation and relays messages which control the release of stress hormones that allow an individual to react to danger or fear. The most common example of violence as a result of amygdalar interference is the case of Charles Whitman, or the “Texas Tower Sniper” who killed multiple people including his wife and mother in 1966. An autopsy following his death revealed a tumor pressing against his amygdala, overstimulating it and severely impacting its function (Batts, 2009).

*See appendix for diagram of relevant brain regions
A study of violent male offenders also focused on brain abnormalities in violent criminals. Researchers found that the amygdalar volumes of the offenders were up to 30% greater than control subjects, which went against previous hypotheses of amygdala hypofunction in those showing psychopathic tendencies similar to the offenders being studied. This suggests that the amygdala may influence other structures within related functional circuits of the brain including the hypothalamus, serotonin pathways, and dopaminergic systems to increase changes like impulsive aggression and reduced sensitivity to stress (Boccardi et al., 2011).

Though the amygdala seems to play one of the largest roles in aggression control, other areas in the surrounding temporal lobes have been studied in regards to violence and criminality. Temporal abnormalities are relatively common in violent offenders. One study found 29% of violent criminals had a temporal irregularity, and all but one test subject had some form of dysfunction (Blake et al., 1995). Furthermore, other researchers found reduced gray matter volumes in the temporal lobes of violent criminal offenders which were linked to antisocial personality disorder and symptoms including limited emotional regulation and heightened levels of reactive aggression (Bertsch et al., 2013). This area is involved in the interpretation of other peoples’ motives, which could explain how miscommunication and increased aggression could lead to instances of criminal violence. The temporal lobe is part of the limbic system, which is responsible for regulating emotions in response to stimuli as well as reinforcing behavior. The temporal lobe within this system has been connected to other regions of the brain, especially in a recent study which mapped lesion networks throughout the brains of criminals (Darby, Horn, Cushman, & Fox, 2018). There was a significant amount of damage in the bilateral temporal areas throughout the sample of offenders, and these lesions seemed to be connected to other systems of the brain with similar functions. Overall, researchers determined that damage from
specific regions in the brain caused effects in other brain structures (a phenomenon called ‘diaschisis’) which were all part of a network in the mind known to manage moral decision-making. The criminal offenders all had head trauma which localized in a common network and shared a temporal relationship, causing the quality of their decision-making to decline (Darby et al., 2018).

In addition to the temporal regions, the lesion network mapping study found similar results localized to the orbitofrontal cortex. This is an area that is part of the frontal lobes and is involved with decision-making as well as cognitive control, connecting it to the same network as the affected temporal regions. Lesions within this structure are thought to inhibit proper cognitive and emotional control as well as rational thought (Darby et al., 2018).

Many past studies have reflected on the function of the orbitofrontal cortex (OFC) and how it communicates with other parts of the brain. For example, a study found offenders with antisocial personality disorder had tissue reductions in the ventral striatum as well as the hippocampus and orbitofrontal cortex. This means that certain regions within and connected to the OFC are significantly smaller in these offenders than the average person, and have since been linked to deficits in reversal learning which prevents the subject from inhibiting a reward-response to a negative behavior. The dopaminergic pathways of the brain are thus affected and synaptic plasticity is decreased in regions of reversal learning since the neurons are not being used (Boccardi et al., 2011).

In addition to reversal learning, the orbitofrontal cortex has been linked to lack of impulse control. In past studies, observation of patients with lesions within the OFC yielded an increase in impulsive, inappropriate, and uninhibited behaviors. Similar increases in lack of apathy and activity led to impulsive responses when patients were presented with problems
Pathways within the brain which are generally responsible for self-evaluation, consideration of consequences, and control of proper response elicitation are detrimentally affected when lesions arise in the OFC, and thus decision-making and irritability are impacted leading to outbursts and often violence. Many parts of the orbitofrontal cortex further project into the hypothalamus and communicate with the amygdala, making it an important center for aggression control.

Perhaps changes in irritability and decision-making can be attributed to personality changes as well. Oftentimes, personality changes come as a result of traumatic injury to the frontal lobe of the brain which is responsible for personality and other high-level executive functioning. This area is crucial to memory and self-control, but studies have cited injury in this region related to aggression and other emotional traits. One study done on violent murderers found 64.5% of them had frontal dysfunction due to lesions. Many of these subjects noted personality changes following their documented brain injuries including heightened feelings of aggression. This dysfunction was also paired with various personality disorders among the studied murderers (Blake et al., 1995).

Antisocial personality disorder is linked to the limbic prefrontal circuit and is commonly paired with borderline personality or similarly highly “psychopathic” traits. In one study comparing criminal offenders diagnosed with these disorders to controls, there were volumetric reductions as well as several lesions in the orbitofrontal and ventromedial prefrontal cortex regions (Bertsch et al., 2013). Antisocial personality disorder is usually found in higher numbers of criminal offenders than the national population, and these prefrontal differences often contribute to this explanation. The cited areas are known to influence emotion regulation in regard to self-referential information and recognizing the emotions of others. Furthermore, these
clusters within the PFC are linked to pathways controlling reactive aggression in individuals. Violence stemming from lesions in this area can greatly play a factor in personality changes and resulting effects.

In addition to self-reflection and personality, researchers have found correlations between voxels (3-dimensional pixels) in the frontal lobe and decision-making. As one of the highest forms of executive function, decision-making is impacted by a number of factors that have to do with cognitive control. The lesion network mapping study mentioned previously found lesions in the medial prefrontal cortex and the orbitofrontal cortex within the same neurobiological network in the brains of criminals (Darby et al., 2018). Similar to the last study, subjects suffered from lower ability to make moral decisions as well as lower cognitive control including increased reactive aggression. These localized parts of the brain contribute to a larger network dedicated to decision-making and goal-directed behavior which is impacted by lesions.

An important subset of traumatic brain injuries is caused by explosive blasts, most often suffered by veterans of war. Researchers in the late 1990s performed a survey investigation into the lives of veterans of the Vietnam War who had been victims of head trauma. All of the subjects had some form of dysfunction in their frontal lobe as a result of localized lesions. Based on family observation and self-report studies following EEG and CT testing, results showed that patients with frontal ventromedial lesions demonstrated Aggression/Violence Scale scores consistently higher than controls and those with lesions in other regions (Grafman et al., 1996). These results include regular verbal threats, physical confrontations, and aggressive reactions. Like other studies mentioned, correlations to decision-making, moral choices, and self-referential thinking are greatly affected by lesions in various spots in the frontal lobe.
A final brain structure to mention relevant to aggression is the hypothalamus. This portion of the brain dedicated to controlling parts of the autonomic nervous system is also partially responsible for communicating with the amygdala in fight-or-flight situations. The hypothalamus is known to work with several different neurotransmitters, and can also be electrically stimulated. One study in mice found that increasing the amount of acetylcholine in the hypothalamus had similar effects to electrical stimulation, and caused impulsive aggression in situations following the experiment. Related instances in humans suggest a cholinceptive mechanism within the hypothalamus which can influence violence, meaning irregular acetylcholine amounts can similarly influence aggression levels in a person if the related hypothalamic regions are damaged (Smith, King, & Hoebel, 1970). Escalated aggressive symptoms can result from many varying changes in the brain, and it is important to note that brain trauma can affect every brain differently.

**TBI in Imprisoned Populations**

As noted earlier, imprisoned populations across the country on average have a higher prevalence of traumatic brain injury incidents than the general population. Many studies have focused on the cases of brain trauma in criminal offenders, and the numbers seem to range across various populations. One group of researchers centered their study on offenders in a county jail using neuropsychological tests and self-report to understand the effects of past TBI occurrences suffered by the offenders. The results showed that 87% of the 69 subjects had endured a traumatic brain injury in their lifetime, and over 36% had a head injury within the past year. Furthermore, those who had the more recent brain damage had higher anger and aggression scores and also a higher prevalence for psychiatric issues as well as poorer cognitive test results (Slaughter, Fann, & Ehde, 2003). This percentage of brain injuries, roughly ten times larger than
the average population, highlights the need for both TBI prevention and increased attention to affected criminal offenders.

Some researchers have focused on broader instances of TBI in criminal populations. For example, one study concentrated on brain injury across multiple offender groups, and found that roughly 60% of criminals overall had experienced brain trauma. In addition to these findings, approximately 50% of TBI victims had also experienced a loss of consciousness following their injury (Shiroma et al., 2010). As mentioned previously, a loss of consciousness is generally used to determine how severe an injury was, and a longer LOC indicates a more serious injury. It can be inferred that since such a high number of offenders suffered a loss of consciousness, their injuries were considered relatively severe.

Studies have also been done to determine if certain population subsets suffer from a greater prevalence of head injuries. Some studies focus on male-specific offender populations to see if the rates differ from females. Researchers have found that roughly 60% of imprisoned men reported brain injuries, and correlated these injuries to major findings about behavioral trends in jails and prisons (Williams, Mewse, et al., 2010b). The men who experienced TBI prior to incarceration were known to enter the prison system at a younger age, around 16 years old on average, than those without head trauma, usually around 20. Additionally, offenders with a TBI had higher rates of re-offending, and generally spent greater amounts of time imprisoned in the last 5 years than control subjects without head injuries (Williams, Mewse, et al., 2010b). These trends noticed by researchers suggest that TBI has a larger influence on aggression and imprisonment than originally believed.

Some studies calculated the rate of TBI in incarcerated females to compare to the rates seen in males. One meta-study collected data across many female imprisoned populations, and
determined just under 70% of the female subjects claimed a head injury prior to their offense. Furthermore, roughly 55% of said female subjects included a loss of consciousness in their injury report (Shiroma et al., 2010). The rates in female offenders seem to be slightly higher, which may be attributed to instances of abuse and other mistreatment which are often precursors to criminal activity.

Younger populations have also been studied, including juvenile offenders who were victims of brain injuries. One sample of males in an institute for young offenders, ages 11 to 19, was surveyed regarding TBI, crime, and other factors. About 65% of the young males reported head injuries, and 46% reported head injuries associated with a loss of consciousness. About a third of these males indicated repeat injury with more than one loss of consciousness, and these cases correlated to more criminal convictions and offenses of greater violence than those with singular incidences of trauma. In addition, the offenders claiming head injury also had 2 more convictions on average than those without previous injury (Williams et al., 2018; Williams, Cordan, Mewse, Tonks, & Burgess, 2010a). These injuries at a young age can be attributed to multiple factors including abuse, traffic accidents, or high-contact activities during stages of development throughout the males’ youth. Traumatic brain injury seems to affect various populations differently, but it is clear that the problem is one affecting imprisoned populations at elevated rates. Regardless of the subset or category of offenders, head trauma is disproportionately seen in cases of criminality across the country.

Limitations and Implications

It is important to note that the results of these studies cannot simply dictate the outcome of traumatic brain injury. Many studies offer signs which may predict the development of impulsive aggression over time following a TBI, and several simply suggest brain regions which
may contribute to elevated violence when damaged. A brain injury is not the end-all be-all which is sure to lead to increased aggression and potentially crime, but merely creates an elevated risk and complications when examining the link between the two instances. Similarly, it is easy to link injury, behavior, and crime, but researchers must be wary of using reverse inference in correlating brain abnormalities to violence post-injury. In addition, studies using self-report survey methods must be considerate of the potential for falsities in responses from subjects. Only documented cases of TBI are helpful in providing backed-up evidence of brain trauma for each subject.

Though there is clearly a strong link between TBI and violence, it is not to say that outside influences have no effect. Genetics can play a large role in levels of aggression and susceptibility to brain changes, and upbringing, background, and other sociological factors can similarly contribute to the predisposition for criminal behavior (Greve et al., 2001). Because of these other confounding variables, it is in fact difficult to isolate instances of TBI and separate them from other influencing socioeconomic factors that may coexist. These influences may cause certain populations to be at a higher risk for brain injuries and also separately likely to end up in prison. Traumatic brain injury is purely another factor which can unfortunately aid in the link between aggression and crime.

Despite the need for vigilance when using brain trauma as a grounds for violence, there are countless implications in this research. Because of the correlation between TBI and crime, there are many ways these results can be applied to legal processes. The most obvious use is the relevance of brain trauma to mental state defenses when offenders stand trial for their crimes. A well-documented case of head injury can help with law mitigation to argue for acquittal from a psychological stance citing confusion, judgment, or reasoning disabilities (Wood & Bhushan S,
There have been many cases of harsh prosecutions given to individuals with head injury, and recent injury can further create difficulty for an individual to participate in the preparation of their case (Sarapata, Herrmann, Johnson, & Aycock, 1998). The complications of TBI in prosecution provide an added layer of uncertainty when determining the outcome of a criminal case.

Though TBI can be used to the benefit of an offender in court, there are many warnings against using speculative results to avoid admission of guilt. For example, brain imaging scans can offer a deeper look at brain abnormalities, but these should not be used to diagnose mental impairment as a complete defense (Kulynych, 1996; Mills & Raine, 1994). This includes considering conceptual issues such as structure versus function and structural specificity before using imaging as evidence for actual brain dysfunction. Furthermore, a criminal defense requires reliability and validity of a testimonial in addition to brains scans to ensure self-reported health screenings are accurate. Without confirmed, documented medical records, TBI as a defense strategy is useless (Center for Disease Control and Prevention (CDC), 2009; Moriarty, Langleben, & Provenzale, 2013). The risk of factual inconsistencies to assist counsel in trial must be avoided to ensure that the proper justification can be reached through the outcome of the case.

In addition to law, traumatic brain injury clearly has implications relating to areas of medicine as well. Various neuroimaging technologies can be tested and used to determine specificity of brain regions when comparing states before and after head trauma (Kulynych, 1996). This can shed light on the importance of certain areas of the brain and further provide results for anatomical and physiological studies regarding the functionality of different regions.

A comparison of prior cognitive functioning levels to post-injury levels done by
professionals can be incredibly relevant to treatment and rehabilitation options, especially for young people who experience high rates of injury or delinquent behavior during periods of growth and development (León-Carrión & Ramos, 2003). Furthermore, a compilation of these results can offer data to use for the creation of protocols relating to TBI. This can allow professionals at hospitals and primary care centers to identify the neuropsychological consequences of various levels of head injury and respond accordingly.

Past studies have shown that neuropsychological conditions can be managed with medication, and the same can be true for aggression. There have been suggestions for use of Tegretol and beta-blocking propranolol as aggression reducers secondary to brain impairment (Martell, 1992). Similarly, treatment of ADHD-like symptoms through medication was found to reduce rates of criminality in patients, and trials for comparable treatment to disorders stemming from impulsivity/aggression have been recommended. Researchers have suggested that mechanisms for different medications should be assessed to treat certain types of injury and injury locations that are common among victims of TBI (Lichtenstein et al., 2012; Miller, 1994). Studies of TBI would be a fantastic source for pharmacological treatment for aggressive behavior disorders to prevent future complications.

A final application of the current research is regarding rehabilitation and societal reintegration of criminal offenders who experienced brain injury. Studies have found that incarcerated individuals who reported a head injury were involved in disciplinary infractions in prison twice as often as non-injured residents (Merbitz, 1995). This goes to show that prisoners with a history of TBI are automatically at more of a disadvantage than others due to their state of being. It has been suggested that this is due to memory deficits, increased irritability, and delayed response causing miscommunication and impressions of deliberate defiance leading to problems
with correctional officers (Centers for Disease Control and Prevention, 2010). Because of these difficulties, incarcerated people with a history of head trauma are likely to have more trouble getting out of the prison system.

To combat these complications, there have been studies done on the effectiveness of early intervention and compensatory cognitive rehabilitation. Researchers compared TBI victims placed into formalized rehabilitation at an acute care hospital to those receiving “nonformalized” care or no rehabilitation at all following brain damage. The results showed that those with formalized treatment had roughly 1/3 the duration of coma and rehabilitation stays as well as better motor, sensory, and cognitive parameter outcomes in comparison to the other group (Chesnut et al., 1999). These findings suggest that an increase in formalized treatment of all patients seeking care for TBI can significantly reduce the negative symptoms that occur after an incident. Application of such results would likely decrease symptoms related to aggression and can thus prevent the onset of new violent behaviors. The positive effects of these findings can be related to incarcerated individuals and could be applied to their rehabilitation following injury to prevent additional violence and allow their reintegration into society following imprisonment to be more comfortable and straightforward.

**Future Suggestions**

There are many directions to take the results of the present research in order to improve the lives of those suffering from TBI. The first is greater access to healthcare as well as mental health and protective services. The opportunity to be seen by a health professional or protective service worker could prevent persisting instances of injury and protect those susceptible to higher risk. Prevention of serious risk factors would be effective in ensuring that TBI rates are decreased and that the symptoms of injury can be reduced over time.
In addition to prevention factors, neuro-rehabilitative dimensions installed in areas of high crime—especially those involving adolescents—would be incredibly helpful in crime reduction and potential for further injury. These would include specific types of rehabilitative therapy and similar programs implemented in order to improve brain function, reduce symptoms, and re-train the brain to restore its cognitive abilities following damage. After head trauma, immediate screening for symptom intervention is necessary to prevent as much future damage as possible. These screenings could also be useful in determining the proper placement into the custodial system for criminal offenders. Additionally, observation of behavior patterns and routine screenings for prisoners with a history of TBI can help identify difficulties which may lead to unnecessary disciplinary infractions during incarceration as a result of brain damage.

The future of research regarding TBI is also crucial, as new findings can clearly improve the lives of millions affected by head trauma. Many suggestions have been made, including a study which focused on brain damage treatment using non-invasive infrared light wavelengths to modulate mitochondria activity which may otherwise block oxygen from reaching specific cells in the brain (Sanderson et al., 2017). In addition, studies are currently being done to identify biomarkers in both the blood and cerebrospinal fluid which can be measured in real time to monitor the progression of a TBI (Korley & Burns, 2018). These tactics, as well as other recent medicinal suggestions, may be helpful to highlight the injured regions and promptly treat symptoms to improve the lives of patients. The future of neuroscience and medicine will be able to properly identify and eventually cure many side effects before they can cause further damage to the brain.

A final suggestion for future change is the addition of community re-entry and transition staff who are trained and equipped with resources for TBI treatment and neuro-rehabilitative
care. By providing this type of help to criminal offenders finishing their sentence, instances of reoffending can be reduced and avoided if the proper help is given to those released individuals who suffer unique symptoms to others. Paired with case management services and community treatment programs, transition resources for released offenders can aid in a smooth reintegration into society following a period of incarceration with brain injury.

**Conclusion**

Traumatic brain injury is a complex phenomenon, and understanding the epidemiology, results, and rehabilitation opportunities available are crucial to improving the quality of life of those suffering from the effects of TBI. Although prevention is ideal, cases of brain damage are inevitable and it is imperative for health professionals and researchers to learn and apply the most effective strategies in order to mitigate the detrimental effects of head injuries to prevent further complications like aggression and violence. Because of the diverse range of consequences caused by TBI, more research is recommended to identify the best course of strategy for future treatment. By better understanding the relationship between head trauma, aggression, and crime, researchers can greatly improve the lives of those who have fallen victim to traumatic brain injury.
References


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Appendix

Glasgow Coma Scale

Eye Opening Response
- Spontaneous—open with blinking at baseline 4 points
- To verbal stimuli, command, speech 3 points
- To pain only (not applied to face) 2 points
- No response 1 point

Verbal Response
- Oriented 5 points
- Confused conversation, but able to answer questions 4 points
- Inappropriate words 3 points
- Incomprehensible speech 2 points
- No response 1 point

Motor Response
- Obeys commands for movement 6 points
- Purposeful movement to painful stimulus 5 points
- Withdraws in response to pain 4 points
- Flexion in response to pain (decorticate posturing) 3 points
- Extension response in response to pain (decerbrate posturing) 2 points
- No response 1 point

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Categorization:
Coma: No eye opening, no ability to follow commands, no word verbalizations (3-8)

Head Injury Classification:
Severe Head Injury—GCS score of 8 or less
Moderate Head Injury—GCS score of 9 to 12
Mild Head Injury—GCS score of 13 to 15
(Adapted from: Advanced Trauma Life Support: Course for Physicians, American College of Surgeons, 1993).

A. A version of the Glasgow Coma Scale used to rate the severity of a brain injury.
B. A diagram of the brain with highlighted regions relevant to violence.

C. A diagram highlighting the location of the hypothalamus.