Head Injury, Post-Concussion Symptoms and Crime

DOCTORATE IN CLINICAL PSYCHOLOGY

Literature Review

Head Injury, Post-Concussive Symptoms, and Aggression in Young Offenders.

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Declaration:

"I certify that all material in this assignment/assessment which is not my own work has been identified properly and attributed. I have conducted the work in line with the BPS DCP Professional Guidelines."

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Head Injury (HI) is the leading cause of death and disability in individuals under 45 years of age in western societies (Bruns & Hauser, 2003; Fleminger & Ponsford, 2005). HI can leave deficits in individuals’ cognitive, behavioural and physical abilities, which can result in profound and lasting effects on sufferers, in mild cases some of these on-going problems can be referred to as post-concussion symptoms (PCS). Teasdale and Engberg (2005) found reduced employment, restricted social and family relations, and quality of life in those who had experienced a HI.  

Risk factors for sustaining a HI are often reported to be age (under 5 years, adolescence and older age), male gender, and lower socio-economic level (Yates et al., 2006). In addition, prevalence rates of self-reported HI (of any severity) in prison populations, including young offenders (YO), have been found to be between 65% and 87% (Williams et al., 2010; Schofield et al., 2006 & Slaughter, Fann & Ehde, 2003). Which is considerably greater than the estimated rates of HI in the general population; these vary from 5-25% (Silver, Kramer, Greenwald, & Weissman, 2001; Farrer & Hedges, 2011). This difference could suggest a relationship between HI and offending behaviour. However, HI in the prison population and specifically the YO population, has until recently, been a relatively neglected area in research and practice. It has been rare for criminal justice professionals to screen offenders for HIs, or

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1 Other difficulties have been identified, such as increased levels of depression, loneliness and family burden (Hoofien et al., 2001 and Martin et al., 2001).
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for neuro-rehabilitation services to be offered when needed. Consequently, given the potential for negative outcomes post HI, it is common for associated health and mental health needs of YOs to go unrecognised. Research exploring the relationship between HI, PCS and YOs is still very much in its early stages and there is a limited understanding of the relationship between these factors.

This review will begin with an overview of the search strategies utilised. It will then provide a summary of the literature base on HI, PCS, offending behaviour and aggression in YOs. Thereafter, research exploring additional influences, such as substance misuse, will be discussed.

**Search Strategy**

Specific terms were used to search a number of online bibliographic databases (see table 1).

**Table 1**

*Search Terms*

<table>
<thead>
<tr>
<th>Concept</th>
<th>Search terms</th>
</tr>
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<tbody>
<tr>
<td>Head injury</td>
<td>(head injury OR traumatic head injury OR head trauma OR mild head injury)</td>
</tr>
<tr>
<td>Crime</td>
<td>(crime OR offending OR youth offending)</td>
</tr>
<tr>
<td>Aggression</td>
<td>(aggression OR violence)</td>
</tr>
<tr>
<td>Post-concussion symptoms</td>
<td>(post-concussion OR post-concussion symptoms)</td>
</tr>
</tbody>
</table>
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Titles and abstracts from the initial search results were then examined. Searches were restricted to papers and book chapters written in English and where only the full article could be accessed (see table 2). Researchers who have already conducted studies in this area were contacted to identify other relevant published or unpublished articles; Google scholar was searched and the reference lists of all the articles retrieved were scanned for additional studies. See chart 1 for a summary of the literature search.

Chart 1

*Literature Search Results Flow Chart*

- (n=420) References Identified from Databases
  - (n=28) Duplicates
  - (n=392) Titles/Abstracts screened
    - (n=336) Excluded
    - (n=56) Full Text Retrieval (where possible)
      - (n=20) Other Sources (e.g. identified from reference lists, authors, recommendations etc).
        - (n=76) Full Text Screened for Inclusion Criteria
          - (n=4) Excluded
            - (n=72) Meeting Study Selection Criteria and Included in Review
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Table 2

Number of Relevant Papers Found During Literature Search

<table>
<thead>
<tr>
<th>Database</th>
<th>Total no. of relevant papers</th>
</tr>
</thead>
<tbody>
<tr>
<td>PsychINFO</td>
<td>29</td>
</tr>
<tr>
<td>Pubmed</td>
<td>14</td>
</tr>
<tr>
<td>Science Direct</td>
<td>9</td>
</tr>
<tr>
<td>PsychArticles</td>
<td>0</td>
</tr>
</tbody>
</table>

Theoretical Background

There are a number of influential theories exploring the neurological and psycho-social development of adolescents that can further our understanding of the relationship between HI, and aggressive or offending behaviour. Moffit’s (1993) dual pathway theory of criminal behaviour defines two criminal trajectories adolescents may take: one consists of early onset offenders who become lifetime offenders and the other consists of late onset offenders who limit offending to the adolescent life stages. Late onset is theorised to result from the frustration prompted by the adolescent-adulthood maturity gap and deviant peer influence. Early onset offending is proposed as a consequence of temperamental and neurological antecedents present early in life, which give rise to deviant behaviour especially when combined with environmental factors like poor parenting. The addition of HI into this mix of risk factors and its association with earlier age of first offence gives cause for concern.

Adolescents are recognised to engage in more risky behaviours than children and adults (Steinberg, 2008). A dual systems model proposes that risky behaviour in adolescents is the product of the lack of synchrony in the development of two of the critical brain systems that
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enable fully adaptive behaviour. The “socio-emotional” system, which is localized in limbic and paralimbic areas of the brain; and a “cognitive control” system, which is mainly composed of the lateral prefrontal and parietal cortices and those parts of the anterior cingulate cortex to which they are interconnected. According to this dual systems model, adolescent risk-taking is hypothesized to be stimulated by a rapid increase in dopaminergic activity within the socio-emotional system around the time of puberty, which is presumed to lead to increases in reward seeking. This system develops rapidly compared to the frontal system that is supposed to regulate it, and the cognitive control system that will, in time, moderate it. The teenage brain, therefore, has an adult-like ability to reason, but with a heightened need for basic reward, and a lowered capacity to buffer immediate influences and potential short-term rewards for greater, longer-term gains. This creates a period of heightened vulnerability to risk-taking during middle adolescence (Steinberg, 2008).

A further influential theory for understanding the relationship between cognition and emotion was developed by Damasio (1994, 1999). According to this theory, cognitive representations interact with internal representations of relevant emotional state. Emotional states, termed ‘somatic markers’, serve the purpose of allocating and maintaining the limited resources of attention in working memory (Damasio, 1994). Following pre-frontal cortex injury, a person may fail to recognise emotional significance in the actions of others, or take account of such significance in planning their responses. Damasio (1999) suggests that deficits in decision making and planning relative to social knowledge, which are commonly observed in pre-frontal injury, are caused by an inability to respond emotionally to thoughts. Of course, a deficit in social behaviour might not be due to inability to integrate a somatic marker per se but in organising one’s decision making effectively. Turkstra (2003) proposed that those who have suffered a HI may misperceive elements of a situation (such as not reading the emotions
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of others effectively and perceiving threat where there was none), make poor social
judgements which can lead to behaving inappropriately and lack communication skills to
negotiate conflict.

**Head Injury**

HI\(^2\) can be referred to as acquired brain injury, traumatic brain injury and head trauma in
publication. HI is often graded as mild, moderate, or severe defined by the level of
consciousness using the Glasgow Coma Scale score (GCS- Teasdale & Jennett, 1974)\(^3\). Studies have identified a number of possible risk factors for acquiring a HI. Specifically, HI
is more common in adolescents, where risky behaviours are seen more frequently
(Newacheck et al., 2003). A retrospective epidemiological study of an Emergency
Departments' data base of HI attendances (Yates et al., 2006), found adolescent males were at
increased risk of injury compared to females.

HI can have profound and lasting effects that negatively impact upon a person's functioning.

Previous studies have resulted in inconsistent prevalence rates of effects post injury.

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\(^2\) This review will refer to HI as "nondegenerative, noncongenital, insult to the brain from an external
mechanical force, possibly leading to permanent or temporary impairments of cognitive, physical and
psychosocial functions with an associated diminished or altered state of consciousness" (Dawodu, 2007, p.1).

\(^3\) Mild HI (GCS 14–15) in most cases is a concussion, reported as resulting in full neurological recovery,
although many of these patients report short-term memory and concentration difficulties. In moderate HI (GCS
9–13) the patient is lethargic, and in severe injury (GCS 3–8) the patient is comatose, unable to open his or her
eyes or follow commands.
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especially when looking at mild HIs (mHIs), making it difficult to draw firm conclusions
around the on-going effects of HI (Binder et al., 1997 & Frencham et al., 2005). Pertab,
James and Bigler (2009) explored some of the contradictory research in this area and found a
considerable degree of variation within the qualitative and statistical methodology used to
calculate the clinical effect sizes, which may restrict the internal and external validity of the
findings. Some of the additional differences found were: the effect sizes of neurological
measures used within the post injury phase; the criteria employed to define mHI; and the
populations from which the mHI samples were selected. Outcomes of more moderate HI are
less favourable with some authors indicating that an estimated 50% of these individuals
suffer long-term injury related disabilities (Kraus et al., 2005). Thus highlighting the
relationship between increasing severity of injury and worse prognosis for long term
functioning.

Furthermore, evidence suggests that in comparison to single concussions; repeat concussions
appear to have increased negative effects upon cognitive and behavioural functioning (Wall
et al., 2006). Around 80% of all HIs are classified as mild (Fleminger & Ponsford, 2005).
mHIs are not usually associated with long term problems, however, when these injuries are
complicated or accumulative, there can be neuropsychological sequelae, particularly
involving attention and executive systems (Williams, Potter & Ryland, 2010). It is possible
that those who have experienced mild repeated concussions may not be expected to suffer
significant long term effects if their injuries are considered as isolated events and there are no
apparent complications detected at the time of injury. The possible accumulative effect of
repeated injuries may get overlooked and therefore assessment and interventions are not
readily offered. Moderate to severe HIs are typically associated with more
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neuropsychological deficits such as behavioural problems and poor social outcomes (Stamrook et al., 1990).

A recent review undertaken by Bigler (2013) considered the neuropathological perspective of such severities of HI and concluded that HI severity could be viewed on a continuum and describes how neuronal damage can occur through mechanical deformation from stretching, twisting and shearing actions brought on by head impact and acceleration/deceleration of the brain during the event that caused the concussion. As summarized by Graham and Lantos (2002) shear and tensile strains at the axonal level are the “most important single factors contributing to the severity of brain damage in any patient who sustains a blunt HI because it occurs at the moment of injury” (p.867). Thus, suggesting even though individuals who suffer mHIs are expected to make a full recovery, it is possible that some subtle neurological damage may have occurred.

Head Injury and Post-Concussive Symptoms

Given the above, it appears important to consider the impact of mHI as well as more severe injuries. The difficulty arises with how to identify such injuries when immediate impact is not as obvious as that of moderate/severe HIs? PCS can offer a potential solution. Young people with mHI have been reported to display a variety of cognitive (problems with memory, attention and concentration, the performance of daily tasks, and decision making), somatic (headaches, sleep disturbance, dizziness, sensitivity to noise or light, visual problems and
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nausea), and affective symptoms (depression, irritability, anxiety, poor frustration tolerance and loss of motivation or apathy) (Axlerod et al., 1996, Bohnen et al., 1995, Piland et al., 2006). Collectively these problems have been referred to as PCS. They are more frequent and severe than those reported by children with injuries not involving the head (Ponsford et al., 1999, Yeates et al., 1999)\(^4\).

There has been an ongoing debate as to whether PCS are due to biological factors from neural damage or a psychological response to the HI (Mittendberg et al., 1992, Mulhern & McMillan, 2006). Research has provided evidence to suggest that PCS symptom report is influenced by depression (Suhr & Gunstad, 2002), chronic pain (Radanov et al., 1992), post traumatic stress (Kennedy et al., 2010), anxiety (Moore et al., 2006), fatigue (Johansson et al., 2009) and involvement in litigation (Less-Haley et al., 2001). However, Moran et al. (2011) propose elevated PCS can be attributed, in part, to actual changes in the brain structure or function.\(^5\) This indicates brain dysfunction could be a significant factor in PCS (Yeates & Taylor, 2005).

Findings also suggest that HI severity correlates with PCS (Yeates et al., 1999, Wilde et al., 2008). Ponsford et al. (2000) suggested, in the first few weeks following injury, somatic symptoms are best at discriminating individuals with mHI from controls. Following mHI symptoms can be relatively subtle making it difficult for others to recognise the association with previous HIs. As a result, they may not serve as an adequate warning sign to trigger

\(^4\) Although PCS typically resolve within several weeks (Carroll et al., 2004 & Light et al., 1998), they can persist for months and sometimes years following injury (Mittenberg et al., 1997, & Yeates et al., 2009).

\(^5\) Evidence exists highlighting that people with PCS have deficits on standardised tests of cognitive function (Ryan & Warden, 2003) and score lower than controls on neuropsychological tests measuring attention, verbal learning, reasoning and information processing.
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appropriate interventions. It appears that the assessment of ongoing PCS may reflect severity of HI, particularly the subtle signs of mHI.

However, Moran et al. (2011) provided some caution around using PCS to discriminate HI severity among children. They found PCS could not consistently discriminate between children with high and low severity mHI at different times post injury. However, post-hoc analysis revealed that only a subset of PCS had discriminatory power in determining severity, two symptoms consistently emerged as strong discriminators, headaches and dizziness (somatic symptoms). Furthermore, Ayr et al. (2009) looked at parent and child ratings of PCS following mHI which resulted in consistent factors reflecting cognitive and somatic symptom dimensions. Dimensions of emotional and behavioural symptoms were less robust across time and raters and therefore the authors suggested these were not likely to represent a consistent part of the constellation of PCS in children with mHI.

Moderate and severe HIs can account for some of the more severe on-going problems, but prevalence rates demonstrate that mHIs represent the vast majorities of HIs. However, most previous studies of mHI have assessed outcomes by using standardised tests of cognitive abilities or broad-based ratings of behavioural adjustment. Furthermore, there are identified shortcomings within the vague and inconsistent definitions of mHI often used in research concerning mHI in children (Williams et al., 1990). Assessment of PCS after HI may help define injury severity and become an integral part of decision making regarding further assessment and intervention for young people.
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**Head Injury and Offenders**

There is evidence to suggest a history of HI is more common in the offender population compared with the general population (Williams et al., 2010). Prevalence of HI in prison populations has been explored in a number of studies; the use of self-reports has produced rates that vary greatly. Between 25-87% of offenders reported experiencing a HI (Schofield et al., 2006 & Morrell et al., 1998) as compared to an estimated 8% in a community population (Slaughter et al., 2003).

As already suggested, adolescence is a risk factor for acquiring HI, it is also a risk factor for exhibiting offending behaviour (Forest et al., 2000, & Mobbs, 2008). Hux et al. (1998) reported a 50% prevalence rate of YOs who had experienced a HI, which was defined by asking whether the participants had ever received a blow to the head. One third of this sample were described by their parents as suffering adverse, long term HI related effects such as diminished ability to regulate behaviour and affect, difficulties with attention, interpersonal skills and school performance. This high rate of HI found in this population could be a result of the broad definition used within the study to identify HI.

The reliability of the methodological approaches typically used in prevalence research can be questionable due to its reliance upon self-reports to obtain HI information; which poses the

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6 A more precise definition of HI appears to lead to a reduced prevalence rate, which as a result may capture only the most severe forms of HI. For example, Brian et al. (2008) identified HI with a large sample of YOs within the USA by asking participants if they had ever had a HI that caused them to black out for more than 20 minutes. Results found that nearly one-in-five (18.3%) of the youths reported a HI. Interestingly, associations were also found between higher rates of psychiatric disorders, and earlier onset of criminal and substance use behaviour in HI sufferers, thus highlighting a number of possible co-morbid factors.
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potential risk of report biases and inaccurate recollection. The inclusion of other sources of information such as medical records may provide further accuracy. However, there is a body of evidence to support the usefulness of self-report: Schofield, Butler, Hollis and D'Este (2011) found self-report of HI was similar in accuracy to hospital records in a group of prisoners. Furthermore, Jolliffe et al. (2003) demonstrated that the validity of self-reports of offending was high when they undertook a prospective longitudinal survey of 808 youths comparing annual court referral data to self-reported data.

A common limitation of research in this field is the lack of a control group (non-offender sample). However, a meta-analysis by Farrer and Hedges (2011), which looked at results from studies employing a control group; found the likelihood of HI remained elevated in YO compared to non-offenders.

Head Injury, Mechanisms of Influence and Offending

Research into this field is in its infancy, but various studies have offered some insight into the possible mechanisms by which HI influences offending behaviour. Diaz (1995) claimed that the most likely features that lead to future violence are severe HI with frontal lobe abnormalities, prolonged unconsciousness and temporal lobe epilepsy. This is supported by work by Raine et al., which has found damage to the frontal lobes of murderers (Raine, 2001; Raine et al., 2000). Identifying influences of HI in offending behaviour in YO is still inconclusive.
Head Injury, Post-Concussion Symptoms and Crime

The possible neurological abnormalities in the brain function of offenders and the interaction between biological, neurological and social factors that may be associated with violent offending has been explored. Pagani and Pinard, (2001) found there was a positive correlation between frontal lobe damage and frequency of violence. Leon-Carrion and Ramos (2003) found violent offenders in their sample reported experiencing significantly more blows to the head than non-violent offenders. This highlights the possible accumulative effect multiple HIs can have on functioning. Education, learning difficulties, childhood illness and psychological/psychiatric treatment did not differ between the two groups. These results suggest that a history of discrete neurological damage as a consequence of injury to the head is more indicative of violent behaviour than intellectual problems or medical or family background. In addition, the pattern of responses among the group suggests that when the various above factors are combined, the risk of violent offending increases.

Regarding YOs in particular, those with a reported history of HI have been found to report an earlier onset of offending (Perron & Howard, 2008). Looking at the influence of multiple HI, Williams et al (2010) found YO who had more than three HI (compared to those with less than three or no HI) reported a greater number of convictions and increased violent convictions.

Timonen et al. (2002) studied a general population birth cohort in Finland involving over 12,000 subjects followed until the age of 31; additional data was retrieved from the hospital case notes. Results indicated that sustaining a HI during childhood and adolescence is linked with fourfold increased risk of developing later psychiatric disorders with coexisting offending in males. The use of a large sample and the rigorous measurement of HI adopted
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in this study strengthens its external validity. However, retrieving data from hospital records limits the applicability of the findings to only those who were hospital treated and therefore, with the most severe outcomes.

A study by Fazel et al. (2011) examined health care and criminal records in the Swedish general population. They found that 9% of those found to have a hospitalised HI were three times more likely to have committed a violent crime in comparison to the non-injured general population at 3%. Importantly, they also looked at the siblings of HI victims and found they too went on to offend at a higher rate than the general population at 4.5%. This highlighted the influence of social and familial factors on offending.

The prevalence rates and above research findings suggests a relationship between HI and worse offending profiles. However, caution is required when attempting to establish the direction of causality between HI and offending given the relatively sparse research in this area. Furthermore, consideration should be given to the possible impact of multiple co-morbid factors. Turkstra, Jones and Toler (2003), suggested a number of risk factors related to both HI and offending, including; increased rates of substance abuse, aggression and dysfunctional family background. This suggests a possible epiphenomenon occurring within the original highlighted phenomenon between HI and offending.

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7 Socio-economic status (SES) is another such risk factor. Anderson et al., (2004) found children of lower SES were at increased risk of long-term neurobehavioral impairments post HI. Yates et al. (2006) also provided evidence that those from lower SES will report longer lasting difficulties from HI and also higher frequency of HI. Thoughts around this increased risk have suggested that those within lower SES families may lack the means to seek appropriate rehabilitation support post HI (Yates et al., 2006). Crime of varying types has also been repeatedly recorded as more prevalent in areas of increased deprivation (Kawachi, Kennedy & Wilkinson, 1999; Howarth, Kenway).
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*Head Injury, Offending and Substance Use*

As highlighted, the role of drug and alcohol use is particularly relevant to the relationship between HI and offending behaviour. The prevalence of substance use is significantly higher in offenders than the general population (Fazel, Bains & Doll, 2006). Furthermore, substance use and HI are highly co-morbid: Schofield et al. (2006) found in a group of adult offenders illicit drug use was associated with in increased likelihood of HI. Williams et al. (2010) found more frequent cannabis use in a sample of YOs with HI than YO without HI. Peron and Howard (2008) provided evidence to suggest earlier onset of substance misuse was associated with HI.

Kenny et al. (2007) studied a sample of 242 YOs in Australia of which 85 had a history of HI and were 2.37 times more likely to have committed a serious violent crime. This risk was increased when hazardous alcohol consumption was also reported. The authors suggested HIs impairs inhibition of aggressive impulses, especially in the presence of alcohol misuse further raising the risk of offending: however the lack of a control group within this study limits the conclusions that can be made. This study identifies alcohol as an independent risk factor for severe violent offending, viewing HI as a contributory factor rather than causal\(^8\). Research looking at the relationship between substance misuse, offending and HI in YO is required to further understand the interaction.

*Post-Concussive Symptoms and Offending*

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\(^8\) Lubman, Yucci and Hall (2007) suggested HI and substance misuse may reduce inhibition of inappropriate responses leading to an increase in aggressive offences.
Head Injury, Post-Concussion Symptoms and Crime

Research findings on the long term effects of HI in the general population are being echoed in research using offenders. Schofield et al. (2006) found 52% of participants reported ongoing sequelae as a result of HI. Likewise, Kenny and Lennings (2007) found over half of a YO sample reported continuing PCS after HI. Furthermore, this study demonstrated a significant positive relationship between the number of unconscious episodes and the persistence of symptoms within participants, which suggests a 'dose-response' effect, whereby those with greater frequency of HI experienced greater symptoms.

With numerous risk factors for HI and offending behaviour, it is difficult to determine the nature of the relationship and causality: investigating the link between HI, PCS and offending behaviour, may highlight PCS to be a more reliable indicator of offending behaviour than self-reported HI. Furthermore, if recognition and treatment of HIs within the youth justice system is currently considered as lacking, then there is a need to further explore the prevalence and potential effects of PCS in the YO population.

Head Injury, Offending and Aggression

As already suggested, there is good evidence that those with HI have an increased risk of aggression\(^9\) and agitation. For example, when compared with patients with multiple traumas without HI, three times as many HI patients showed significant aggression during the first 6 months post injury (Tateno, 2003). This aggressive behaviour could be viewed, therefore, as an on-going problem related to a history of HI. For example a quarter of patients from an in-

\(^9\) The definition of aggression encompasses both verbal and physical aggression against self, objects and other people (Yudofsky, 1986).
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patient rehabilitation unit displayed aggressive behaviour at follow-up 6, 24 and 60 months after discharge (Baguley, 2006).

Studies of adults have shown that damage to the frontal area results in recurrent impulsive, aggressive and antisocial behaviour, immature moral reasoning and a poor appreciation for the subjective experience of others (Raine et al., 1994, Brower and Price, 2001). This evidence highlights accumulative injuries can impact on the frontal limbic system, which can result in individuals having significant difficulties in managing their behaviour appropriately, suggesting a possible explanation for the manifestation of aggression in those with HI: which could lead to significant problems such as offending behaviour (Jolliffe and Farrington, 2004).

A distinction between proactive and reactive aggression can offer an interesting insight into the varying origins of aggression and subsequent offending. Proactive aggression has been characterised as instrumental, organised, and “cold-blooded”, with little evidence of autonomic arousal (Dodge, 1991, Mirsky & Siegel, 1994). In contrast, reactive aggression has been conceptualised as a fear-induced, irritable and hostile affect-laden defensive response to provocation (Dodge, 1991 & Volavka, 1995). Furthermore, links to a lack of inhibitory functions, reduced self-control and increased impulsivity have been found (Raine et al., 1998).

10 Proactive aggression has its roots in Social Learning Theory (Bandura, 1973, as cited in, Hubbard et al, 2002) whereby aggression is characterised as a learned behaviour, instrumental, planned and goal oriented in nature, controlled by positive reinforcement (Dodge, 19992, as cited in Hubbard et al, 2002). Reactive aggression is derived from the frustration-aggression hypothesis (Berkowitz, 1993, as cited in, Hubbard et al, 2002) whereby high emotional arousal (threat, vulnerability for example) is experienced as a result of perceived frustration or provocation, culminating in an aggressive response.
Raine et al (2006) undertook a study reporting on the development of the Reactive-Proactive Aggression Questionnaire (RPQ) and the differential correlates of these two forms of aggression. Both forms of aggression were associated with excessive fighting at age 7. Reactive total scores were 2.55 times higher than proactive scores, suggesting that reactive aggression may be more adaptive and quasi-normative, whereas proactive was characterised as being more pathological. Dooley et al. (2008) reported a study of aggression in an adolescent HI group. They found the HI group, compared to non-injured controls, was significantly more likely to engage in reactive aggression behaviours, characterised by emotional lability, inability to tolerate frustration and anger. Aggression was typically in response to provocation, but not purposive, that is, not to display interpersonal dominance or object acquisition. It would appear therefore that injuries to frontal systems required for executive functions, may alter an individual’s self-regulatory capacity, which could be relevant to the origins of offending.

Summary

The literature has demonstrated some of the on-going sequelae of HI as well as the high prevalence rate of HI within the prison population and more specifically YOs. This review has also highlighted that increased consideration should be given to the co-occurrence of other variables within this population such as substance use which can offer additional risk factors for offending behaviour and HI. Research into PCS has shown that HI should be viewed as a chronic health condition which can lead to changes in the brain associated with
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on-going symptoms. These symptoms can include cognitive, somatic and affective
difficulties, which may highlight the specific impairments impacting on an individual’s
ability to function effectively in their environment. However, these symptoms may go
unrecognised and thus intervention is not considered. Furthermore, assessment of PCS after
HI may be related to injury severity and therefore provide an interesting measure to inform
clinical decision-making regarding this group of individuals.

Future Research

There is a need to further understand the relationship between HI and offending behaviour
and the research within this area is scarce within the YO population. Fazel and Danesh
(2002) highlighted the need for better management of mental and physical health needs of
prison populations, with the potential to reduce the number of incarcerated individuals.
Adolescence is a key period for prevention and rehabilitation of HI which in turn could lead
to a reduction in offending and improve public protection. It has been suggested many
offences might not occur if people with HI were given prompt and comprehensive treatment
after the injury (Sarapata et al., 1998). Consequently, this study will explore the relationship
between self-reported HI, PCS, aggression, offending and substance use in YO.
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