Timing of Traumatic Brain Injury in Childhood and Intellectual Outcome

Louise M. Crowe,1,2 PhD, Cathy Catroppa,1,2 PhD, Franz E. Babl,1,4 MD, MPH, FRACP, FAAP, FACEP, Jeffrey V. Rosenfeld,5,6 MBBS, MD, MS, FRACS, FRCS(Ed), FACS, FRCS (Glasg) HON., FACTM, MRACMA, and Vicki Anderson,1,2,3 PhD

1Critical Care and Neuroscience, Murdoch Children’s Research Institute, 2Psychological Science, University of Melbourne, 3Psychology Department, Royal Children’s Hospital, 4Emergency Department, Royal Children’s Hospital, 5Department of Neurosurgery, Alfred Hospital, and 6Department of Surgery, Monash University, Melbourne, Australia

All correspondence concerning this article should be addressed to Dr. Louise M. Crowe, Department of Child Neuropsychology, Critical Care and Neuroscience, Murdoch Children’s Research Institute, Royal Children’s Hospital, Parkville, Victoria, 3052, Australia. E-mail: louise.crowe@mcri.edu.au

Received October 2, 2011; revisions received April 23, 2012; accepted April 25, 2012

Objective Typically, studies on outcomes after traumatic brain injury (TBI) have investigated whether a younger age at injury is associated with poorer recovery by comparing 2 age groups rather than participants injured across childhood. This study extended previous research by examining whether the influence of age on recovery fits an early vulnerability or critical developmental periods model. Methods Children with a TBI (n = 181) were categorized into 4 age-at-injury groups—infant, preschool, middle childhood, and late childhood—and were evaluated at least 2-years post-TBI on IQ. Results Overall, the middle childhood group had lower IQ scores across all domains. Infant and preschool groups performed below the late childhood group on nonverbal and processing speed domains. Conclusions Contrary to expectations, children injured in middle childhood demonstrated the poorest outcomes; this age potentially coincides with a critical period of brain and cognitive development.

Key words children; IQ; outcome; plasticity; traumatic brain injury.

Introduction

Traumatic brain injury (TBI) is a major cause of disability in children (Kraus, Rock, & Hemyari, 1990). An extensive body of research has demonstrated that TBI results in impaired cognition, with recovery influenced by individual characteristics, injury, and environmental factors (Anderson et al., 2006; Catroppa, Anderson, Morse, Haritou, & Rosenfeld, 2008). Understanding the influence of these factors is of great interest to both clinicians and researchers. In terms of injury factors, increased injury severity is a well-established predictor of cognitive sequelae (Anderson, Morse, Catroppa, Haritou, & Rosenfeld, 2004; Anderson, Catroppa, Morse, Haritou & Rosenfeld, 2005; Ewing-Cobbs, Fletcher, Levin, Iovino, & Miner, 1998; Ewing-Cobbs et al., 2006). Environmental factors, including low socioeconomic status (SES), dysfunctional family environments, and reduced access to resources, are also associated with poorer cognitive recovery (Taylor et al., 2001). It appears that environment interacts with injury severity to influence outcomes. This theory is referred to as the “double-hazard theory”, which states that while severe TBI is associated with the poorest outcomes, severe TBI coupled with a family of social disadvantage or dysfunction has the worst functional outcomes (Escalona, 1982). In contrast, the presence of only one of these factors, either severe TBI or environmental risk
factors (social disadvantage/family dysfunction) is linked to significantly better recovery (Anderson et al., 2005; Escalona, 1982; Taylor & Alden, 1997).

Individual characteristics, such as gender, age, and pre-injury function, also influence outcome. Relevant to the present study, the impact of age at injury has been a focus of several research studies, with varied results. Those arguing for the importance of age at injury propose that poor outcomes are explained by the incomplete development of the brain in early life, with a TBI derailing ongoing maturational processes and leading to neurobehavioral impairment (Anderson et al., 2005; Ewing-Cobbs, Miner, Fletcher, & Levin, 1989). Childhood is indeed a time of rapid brain development. In the first two years of life processes, such as dendritization and synaptogenesis are highly active. Further, from birth and during critical periods through childhood, the brain undergoes extensive myelination, particularly in the anterior and subcortical regions (Spencer-Smith & Anderson, 2009; Stiles, 2000). Early in childhood, while these processes are ongoing, the brain is less functional committed than an adult brain, with researchers arguing that this greater plasticity leads to an increased ability to recover after insult, referred to as early plasticity theory (Kennard, 1936; Teuber, 1971). While this view is supported by focal lesion and hemispherectomy studies (Ballantyne, Spilkin, Hesselink, & Trauner, 2008; Dennis & Whitaker, 1976; Hertz-Pannier et al., 2002), when damage is diffuse with little healthy tissue available for compensation (such as in TBI), early plasticity theories are insufficient to explain recovery patterns.

In response to these limitations, the early vulnerability theory was suggested, proposing that the young brain is more susceptible to damage and associated cognitive impairment, particularly in the context of diffuse injury such as TBI (Anderson & Moore, 1995; Donders & Warschausky, 2007; Hebb, 1942; Kriel, Krach, & Panser, 1989). Young children and infants are particularly vulnerable to damage after TBI for physiological and neurological reasons, for example, a thin and pliable skull, a disproportionately large and heavy head with weak neck muscles increasing susceptibility to rotational and shearing forces and increased elasticity of blood vessels (Case, 2008; Hahn, Chyung, Barthel, Bailes, Flannery & McLone, 1988; Margulies & Thibault, 2000).

Despite this growing literature, as yet there is no consensus concerning the specific age range that characterizes this early vulnerability. For example, one study comparing children injured <1 year of age to children injured between 1 and 2 years found no difference in outcomes (Keenan, Runyan, & Nocera, 2006). Similarly, Ewing-Cobbs et al. (1997) compared children injured at a between 4 and 41 months to those injured at 42–72 months and also reported no age-at-injury effect. In contrast, our team has repeatedly demonstrated poorer outcomes for children aged <7 years compared to older children (Anderson & Moore, 1995; Anderson et al., 2005). While methodological differences may underpin these contradictory results, it may be that the assumption of a linear relationship between age at injury and outcome is not appropriate. This possibility is consistent with the principles of developmental biology that characterize early brain development as a stepwise process consisting of peaks and plateaus, often termed critical periods, during which neural maturation is rapid and neural networks are consolidated (Huttenlocher & Pribram, 1990; Kolb, Pellis, & Robinson, 2004). Interruption of these processes via injury has been reported to have a dramatic impact, although the details of these processes remain far from precise. Recent studies examining the functional consequences of these developmental processes has provided limited support for a linear age at injury effect but has highlighted the significant vulnerability of the brain prior to 2 years of age (Anderson et al., 2009), which is a period of increased dendritization and synaptogenesis (Huttenlocher, 1979; Huttenlocher & Dabholkar, 1997) and when many cognitive skills begin to develop.

This study extended previous research by examining the impact of age at insult in a sample aged two months to 12 years at injury, divided across four developmental stages, in order to explore whether recovery from TBI is best represented by an early vulnerability model with earlier injury associated with poorest outcome or a critical period model with recovery dependent on neurological and cognitive development at time of injury. This follow-up time was chosen to ensure that early recovery processes had stabilized and so that even the youngest children could be tested using the Wechsler Scales. Drawing from past research, the following hypotheses were developed. First, we expected that children with severe TBI would record the lowest IQ scores. Second, consistent with the majority of literature available, we expected that very early injuries (<2 years) would be associated with the greatest IQ deficits, and conversely, late child injury (10–12 years) would be associated with fewer IQ impairments. There is little empirical research to guide hypotheses for preschool and middle childhood insults (3–9 years), but we speculated that, due to growth spurts from 6 to 9 years (Huttenlocher, 1979; Huttenlocher & Dabholkar, 1997; Kolb et al., 2004), this age group would also be vulnerable to poor outcome. Third, we predicted that findings would support the “double-hazard” theory: that is, severe TBI and social disadvantage or family dysfunction would be
associated with the poorest performance. The role of injury and environmental factors, such as SES and family function, were also examined to explore their contribution to outcome.

**Methods**

**Participants**

Children presenting with an accidental TBI were recruited through attendance at the Emergency Department or admission to the Intensive Care Unit of the Royal Children’s Hospital, Melbourne, Australia, between 1997 and 2010. For this study, only accidental TBI were included, as children with inflicted TBI present with specific pathological, neurological, and environmental characteristics, and post-injury social experiences (e.g., removal from home and difficulty locating), which are likely to confound effects of TBI (Ennis & Henry, 2004; Ewing-Cobbs, Kramer, Prasad et al., 1998; Keenan, Runyan, Marshall, Nocera, & Merten, 2004).

Inclusion criteria were: (i) age at injury 0–13 years, (ii) documented evidence of TBI including a period of altered consciousness, and (iii) ability to complete cognitive assessment. The following exclusion criteria were applied: English as a second language, prior TBI, and pre-existing neurological or developmental disorder. Children were followed up between 24 and 45 months after injury (M = 30.06 months). Two hundred and fifty-six families were approached to participate in the study with 186 families agreeing to participate (participation rate = 72.5%). There was no response from 70 families, and due to ethics restrictions, we are unable to clarify why they did not respond, which could be a number of reasons (e.g., moved residence, lack of interest in study, etc.). The data from five children were excluded from analysis because of excessive missing data; therefore, the final sample consisted of 181 children. The study was conducted according to the requirements of the Human Research Ethics Committee, Royal Children’s Hospital, Melbourne, Australia.

Children (n = 181) were divided according to TBI severity using the following criteria: (i) mild TBI (n = 57): lowest Glasgow Coma Score (GCS; Teasdale and Jennett, 1974) of 13–15, modified if children under 3 years, loss of consciousness (LOC) <1 h, post-traumatic amnesia (PTA) of <24 h, and no evidence of mass lesion on CT or MRI scan if completed; (ii) moderate TBI (n = 77) lowest GCS 9–12 and mass lesion and/or evidence of specific injury on CT/MRI scan; and (iii) severe TBI (n = 45): GCS 3–8 and mass lesion and/or evidence of pathologic condition on CT/MRI scan. The GCS modified for children takes into account the language abilities of children <3 years. Specifically, the verbal items are replaced by questions about crying and interactions with parents (Reilly, Simpson, Sprod, & Thomas, 1988).

Children were categorized into groups based on the age at TBI. Groupings were based on timing of cerebral growth spurts (van Praag, Kempermann, & Gage, 2000; Kolb et al., 2004; Giza & Prins, 2006) and previously utilized by Anderson et al. (2009). The sample was divided into four groups: infant (n = 48), 2 months to 2 years at injury; preschool (n = 43), 3–6 years at injury; middle childhood (n = 45), 7–9 years at injury; and late childhood (n = 43), 10–12 years at injury.

**Measures**

**Injury, Demographic, and Family Variables**

Information on children’s medical and developmental histories and parental occupation were collected. Socioeconomic status (SES) was calculated on parent occupation using the Scale of Occupational Prestige (Daniel, 1983). This scale rates parent occupation on a 7-point scale, with higher scores representing lower status and provides a standardized measure of distal environmental factors. The higher status of both parent occupations was used.

**Family Functioning**

The Family Functioning Questionnaire (FFQ; Noller, 1988) was used to assess family function. It contains 30 items and uses a 6-point rating scale (1 = totally agree to 6 = totally disagree). Three factors were derived: conflict (60 points), intimacy (72 points), and parenting style (30 points). This questionnaire has been used in other studies investigating outcomes after TBI (Anderson, Morse, Catroppa, Haritou, & Rosenfeld, 2004). The conflict score was used as a measure of family dysfunction for this study with a higher score indicating less conflict.

**Intellectual Functioning**

To measure global intellectual function, the Wechsler Preschool and Primary School Intelligence Scale (WPPSI-R/WPPSI-III; Wechsler, 1989, 2002) and the Wechsler Intelligence Scale for Children (WISC-III; Wechsler, 1991) were administered dependent on the age of the child (<6.5 years the WPPSI-R/WPPSI-III and ≥6.5 years the WISC-III) with the intelligence quotient (IQ) calculated. The verbal IQ (VIQ), performance IQ (PIQ), processing speed index (PSI), and full-scale IQ scores (FSIQ) were calculated each with a mean (SD) of 100 (15). Children in the infant group completed the WPPSI-R (n = 20) and the WPPSI-III (n = 29). Children in the preschool group completed the WPPSI-R (n = 10).
and the WISC-R (n = 33). All children in the middle childhood and late childhood group completed the WISC-R. Of note, we have previously analyzed and reported on potential test-specific effects associated with the use of different versions of the Wechsler scales in this sample (Anderson et al., 1997, 2005), with results suggesting no evidence of a systematic score bias.

**Statistical Analysis**

The severity groups and age groups were compared for demographic and injury-related variables to identify any group differences that might influence outcome. A two-way ANOVA (age x severity) was conducted to analyze IQ scores, and effect sizes (d) were calculated. As injury severity proportions showed some inconsistency across groups, we re-analyzed these data using brain injury severity (GCS) as a covariate. As this did not alter the findings, these analyses are not reported. Post hoc analysis was used (Tukey HSD). Holmes adjustment was also used to reduce chance of Type I error. The proportion of scores in the impaired range, defined as one standardized score below the mean (≤ 85) for each age group, was analyzed using chi-square and standardized residuals.

Two two-way ANOVAs: 1-severity x SES (high/low) and 2-severity x FFQ conflict (high/low) were conducted to test for a double-hazard model. SES was classified into high/low based on previous psychometric data (Noller, Seth-Smith, Bouma & Schweitzer, 1992).

Predictors of outcome were identified using hierarchical regression analysis with SES entered in step one and lowest GCS, age at injury, and FFQ conflict entered at step two.

**Results**

**Demographics**

Table I lists the demographic details of the sample. There were no significant differences for the age groups or severity groups for gender, time since injury, or SES. There was no difference for age at assessment for the severity groups. Table II displays the injury and neurological characteristics of the groups. Of note, similar rates of motor vehicle accidents, falls, and other causes were recorded across the four age-at-injury groups, with the exception that more children in the infant group were injured in falls.

As expected, there were group differences on all medical variables with the severe TBI group presenting with significantly more complications than the other severity groups. There was no significant difference for injury...
severity across the age at injury groups [$\chi^2 (3, 181) = 8.16$, $p = .227$].

**Injury Severity**

IQ scores across the four age groups (infant, preschool, middle childhood, and late childhood) and severity levels (mild, moderate, and severe) are presented in Table III. Visual inspection indicates that children with mild and moderate TBI had IQ scores in the average range, while the severe TBI group had scores mostly in the low average range (80–90). There was a significant group difference for VIQ, PIQ, PSI, and FSIQ measures, with post hoc testing indicating that the severe group performed below the mild ($p < .001, d = .85–1.13$) and moderate groups ($p = .000–.02, d = .49–.94$) for each of these domains.

**Age at Injury**

For age groups, significant differences were found across the groups for each IQ variable. For VIQ, the middle childhood group had lower scores than all other groups (infant: $p = .003, d = .79$; preschool: $p = .03, d = .57$; and late childhood: $p = .04, d = .55$). For PIQ, the infant ($p = .011, d = .64$), preschool ($p = .03, d = .55$), and middle childhood ($p < .001, d = .89$) groups recorded significantly lower scores than the late childhood group. For PSI, the middle childhood ($p = .04, d = .33$) and preschool ($p = .001, d = .93$) groups had lower scores than the late childhood group. For overall intellectual ability (FSIQ), the middle childhood scored lower than the late childhood group ($p = .003, d = .73$).

**Rates of Cognitive Impairment**

The proportion of children with IQ scores in the impaired range (IQ ≤ 85) was investigated across age at injury groups. For VIQ, $\chi^2 (3, 181) = 10.85$, $p = .01$, a larger proportion of the middle childhood group (SR = 2.3) fell in the impaired range than expected. For PSI, $\chi^2 (3, 146) = 13.03$, $p = .005$, a larger proportion of the preschool group (SR = 2.8) fell in the impaired range than expected. No differences were found for PIQ or FSIQ.

**Predictors of Outcomes**

To assess if the results fitted with the “double-hazard” model, the interaction of severity and SES for IQ outcomes (VIQ, PIQ, and FSIQ) were analyzed. No interaction effect was found for VIQ and FSIQ. An interaction effect was found for severity and SES for PIQ. $F(3, 150) = 2.34$, $p = .04$, with lower SES and greater severity contributing to poorer score. The interaction between severity and family dysfunction (FFQ conflict) for IQ outcomes (VIQ, PIQ, and FSIQ) were analyzed. No interaction effect was found for VIQ, PIQ, and FSIQ. The contribution of SES, lowest GCS, and FFQ conflict to outcome (VIQ, PIQ, PSI, and FSIQ) were investigated using hierarchical regression analysis, with SES entered at step one, and GCS, family conflict, and age at injury at step two. The results show that SES and the other predictors accounted for a significant amount of the variance (Table IV).

For Verbal IQ, SES alone accounted for 12% of the variance, but in combination with the other predictors accounted for 21% of the variance. For PIQ, SES accounted

---

**Table II. Injury Characteristics of Age Groups**

<table>
<thead>
<tr>
<th></th>
<th>Infancy (0–2 years)</th>
<th>Preschool (3–6 years)</th>
<th>Middle childhood (7–9 years)</th>
<th>Late childhood (≥10 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>50</td>
<td>43</td>
<td>45</td>
<td>43</td>
</tr>
<tr>
<td>Injury cause</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Falls, n (%)</td>
<td>37 (74)</td>
<td>21 (49)</td>
<td>21 (47)</td>
<td>19 (44%)</td>
</tr>
<tr>
<td>MVA, n (%)</td>
<td>9 (18%)</td>
<td>19 (44)</td>
<td>18 (40)</td>
<td>17 (40%)</td>
</tr>
<tr>
<td>Others, n (%)</td>
<td>4 (8%)</td>
<td>3 (7)</td>
<td>6 (13)</td>
<td>7 (16%)</td>
</tr>
</tbody>
</table>

Note. MVA = Motor vehicle accident.

---

**Table III. Wechsler IQ Scores Across Injury Severity and Age-at-Injury Groups**

<table>
<thead>
<tr>
<th></th>
<th>Infant Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Preschool Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Middle Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Late Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>20</td>
<td>23</td>
<td>7</td>
<td>11</td>
<td>19</td>
<td>13</td>
<td>10</td>
<td>21</td>
<td>14</td>
<td>17</td>
<td>15</td>
<td>11</td>
</tr>
<tr>
<td>VIQ, M (SD)</td>
<td>98.8</td>
<td>94.4</td>
<td>93.1</td>
<td>97.6</td>
<td>97.7</td>
<td>86.0</td>
<td>90.5</td>
<td>88.7</td>
<td>82.5</td>
<td>98.4</td>
<td>92.3</td>
<td>89.7</td>
</tr>
<tr>
<td></td>
<td>(10.7)</td>
<td>(10.2)</td>
<td>(10.2)</td>
<td>(12.8)</td>
<td>(11.0)</td>
<td>(13.7)</td>
<td>(8.0)</td>
<td>(11.5)</td>
<td>(12.3)</td>
<td>(9.9)</td>
<td>(16.5)</td>
<td>(11.4)</td>
</tr>
<tr>
<td>PIQ, M (SD)</td>
<td>104.3</td>
<td>99.5</td>
<td>89.7</td>
<td>110.4</td>
<td>103.1</td>
<td>90.3</td>
<td>96.6</td>
<td>100.0</td>
<td>89.7</td>
<td>116.5</td>
<td>108.0</td>
<td>101.6</td>
</tr>
<tr>
<td></td>
<td>(11.7)</td>
<td>(15.8)</td>
<td>(14.1)</td>
<td>(14.5)</td>
<td>(12.6)</td>
<td>(16.6)</td>
<td>(12.8)</td>
<td>(14.3)</td>
<td>(14.3)</td>
<td>(11.1)</td>
<td>(13.4)</td>
<td>(13.3)</td>
</tr>
<tr>
<td>PSQ, M (SD)</td>
<td>103.0</td>
<td>97.5</td>
<td>–</td>
<td>99.7</td>
<td>96.8</td>
<td>82.8</td>
<td>98.5</td>
<td>102.7</td>
<td>85.5</td>
<td>109.3</td>
<td>103.4</td>
<td>97.4</td>
</tr>
<tr>
<td></td>
<td>(12.1)</td>
<td>(14.4)</td>
<td>–</td>
<td>(12.8)</td>
<td>(9.3)</td>
<td>(12.0)</td>
<td>(12.5)</td>
<td>(11.4)</td>
<td>(11.7)</td>
<td>(12.6)</td>
<td>(11.8)</td>
<td>(12.5)</td>
</tr>
<tr>
<td>FSIQ, M (SD)</td>
<td>99.9</td>
<td>98.0</td>
<td>90.7</td>
<td>103.8</td>
<td>100.1</td>
<td>85.8</td>
<td>93.9</td>
<td>93.5</td>
<td>86.1</td>
<td>107.3</td>
<td>99.5</td>
<td>94.7</td>
</tr>
<tr>
<td></td>
<td>(10.2)</td>
<td>(14.2)</td>
<td>(15.6)</td>
<td>(13.8)</td>
<td>(9.9)</td>
<td>(11.5)</td>
<td>(10.7)</td>
<td>(12.3)</td>
<td>(14.7)</td>
<td>(8.3)</td>
<td>(15.9)</td>
<td>(12.7)</td>
</tr>
</tbody>
</table>
Discussion

Using a large sample size of children with mild, moderate, and severe TBI sustained from infancy to late childhood, this study investigated TBI severity, age at injury, and environmental factors to intellectual function (IQ). Our first expectation that children with severe TBI would have the lowest IQ scores was supported (Catroppa et al., 2008; Ewing-Cobbs et al., 1997). Our second expectation that very early TBI (<2 years) would be associated with the greatest IQ deficits and that late childhood injury would be associated with the least impairment was somewhat supported. Findings suggest that recovery is influenced by age at injury as well as stage of brain and cognitive maturation. The third expectation that a “double-hazard” model would explain recovery was only found for nonverbal abilities, with lowered SES and increased severity contributing to the greatest IQ deficits. Time since injury has been identified as a factor needing more consideration in research (Taylor & Alden, 1997), and it was controlled for in this study with a 21-month range, beginning after recovery processes had stabilized.

This study examined the impact of age at injury on cognitive outcomes and, in particular, whether there is a linear relationship present, in order to contribute to the early plasticity/vulnerability debate. While this has been investigated for different types of insults, this is the first time that the critical period’s perspective has been applied to child TBI. Compared to children injured in late childhood, children injured in infancy and preschool years had lower nonverbal abilities. Children with preschool injuries (3–6 years) also demonstrated lower processing speed, with other performances not significantly below the other groups. Surprisingly, children injured in middle childhood (7–9 years) displayed poorer performance on verbal IQ, nonverbal IQ, processing speed, and overall IQ than children injured in late childhood.

The finding that the younger children (those injured in infancy and preschool years) performed below those injured in older childhood has previously been reported (Anderson & Moore, 1995; Kriel et al., 1989), but the finding that the middle childhood group is at risk for cognitive outcomes is novel. The children with mild TBI in this age group appeared to show particular difficulties, performing at similar levels to children with moderate TBI. In this time period, there may be significant developmental changes occurring, for example, recent diffusion tensor imaging studies have shown that middle childhood is characterized by a peak in myelination (Lebel, Walker, Leemans, Phillips, & Beaulieu, 2008). Further, from a cognitive perspective, this period is characterized by rapid development of more “dynamic” abilities, such as selective attention and executive skills particularly inhibition and self-monitoring (Klenberg, Korkman, & Lahti-Nuutila, 2001; Levin et al., 1991; McKay, Halperin, Schwartz, & Sharma, 1994; Rebok et al., 1997). It is also a key time for language skill development (Paul, 2006). It may be hypothesized that an insult during this developmental stage can result in impairments in these skills, which then impacts overall cognitive performance. Of note, each of these skills are critical for “down-the-line” abilities such as learning, working memory, task planning, time management, reasoning ability, and impulsivity.

It is possible that a child injured in this developmental period (7–9 years) may miss important educational input due to deficits in these “dynamic” abilities underpinning skill acquisition and knowledge. While research comparing middle childhood to other age groups is limited, there is some research that supports the view that an insult in middle childhood is associated with particularly poor outcomes. Anderson et al. (2009) reported that children who acquired an insult at this age had higher levels of speech delay, need for extra school tuition and classroom aides, and were more likely to be attending a school for disabled children than children injured in preschool or
late childhood. Other studies have reported a lack of linearity after brain insult with children injured between 2 and 6 years, displaying poorer social skills than those injured in the prenatal and infancy period (Greenham, Spencer-Smith, Anderson, Coleman & Anderson, 2010).

A double-hazard model of outcome was supported, with injury severity and lowered SES contributing to non-verbal IQ. This is supported by previous research (Escalona, 1982). Overall, SES provided the largest contribution to outcome, a finding reported previously (Catroppa et al., 2008; Donders & Nesbit-Greene, 2004). A combination of SES, injury severity, and family dysfunction further increased the explanatory power for intellectual outcomes. While injury severity has consistently been associated with outcome, the contribution of the environmental factors, both distal (SES) and proximal (family conflict), is particularly interesting (Gerring & Wade, 2012). It appears that outcomes after TBI have a particular “environmental vulnerability” with children from families of greater social disadvantage and family dysfunction at greatest risk of poor outcomes.

**Limitations**

There are limitations to this study. First, although this is one of the largest studies investigating IQ and age at injury in children, cell sizes for specific age/severity groups were small and results may have been skewed by relatively small numbers in various age/severity subgroups. For example, there was a small sample for infants with severe TBI (n = 7), which possibly reflects the exclusion of inflicted TBI, which is the main cause of severe TBI in this age group (Kraus et al., 1990). However, inclusion of this group may have led to confounds with respect to both injury mechanism and pre- and postinjury environment (Ennis & Henry, 2004; Ewing-Cobbs, et al., 1998). It is likely that the performance of the infant group would be poorer if inflicted injuries were included, with research indicating poorer outcomes for inflicted versus accidental TBI (Ewing-Cobbs, Kramer et al., 1998). In addition, the injury mechanism varied across the age groups, with more children in the younger group sustaining TBI from falls, and the possible impact of this could not be assessed. A number of families (70 out of 256 approached) contacted did not participate, and we are unable to clarify why this was because of ethics restrictions. It is possible that there is a bias in those who did not participate, and this may have impacted results.

Second, using such a wide age range presents significant challenges. An example common to many longitudinal developmental studies is the problem of using multiple test measures. In the present study, two separate measures of intellectual ability were used, and while correlations across these measures were robust (Yule et al., 1982), it is difficult to determine the subtle impact of these variations in methodology, although our previous studies have not identified any systematic bias (Anderson et al., 1997). Further, clarifying injury severity is complicated in longitudinal developmental studies, with post-traumatic amnesia unable to be accurately measured in young children, and imaging rarely conducted in this age group.

**Future Directions**

These findings represent an initial attempt to explore the relationship between brain maturation factors and functional recovery. Replication is needed to confirm our results. In particular, reduced intellectual ability after TBI is likely to reflect deficits in more specific cognitive areas such as executive function and language skills, which impede future learning, but cannot be adequately characterized using IQ tests. Future studies that examine a wider range of skills would better clarify the range of impairment after TBI. The use of magnetic resonance imaging would also be helpful in clarifying whether the type of insult sustained and resultant pathology across the age groups is equivalent.

**Clinical Implications**

Clinically, predicting outcome from TBI is complicated. Factors demonstrated by this study to influence outcome include age at injury, environmental factors, and injury severity. In the present context, neither age at injury nor injury severity can be modified, but the influence of the family environment provides an opportunity for intervention to maximize recovery. These results suggest that particular child characteristics may be usefully targeted for treatment, and that greater social disadvantage are particularly vulnerable to poor outcomes and likely to require ongoing clinical services. With respect to age at injury, only older children appear to escape their injury relatively unscathed. Children with preschool insults need ongoing surveillance. Similarly, children injured in middle childhood also show poor outcomes, whether due to injury factors, developmental factors or context (that is, early school experience) remains unclear.

**Conclusions**

The results suggest that not all TBI are “created equal,” and that at certain stages of cerebral and cognitive development, children are particularly vulnerable to residual
cognitive impairments. Surprisingly, children injured in middle childhood appear to be particularly vulnerable. In contrast, sustaining a TBI in late childhood displayed best outcomes. Preschool age and infancy were also times of vulnerability for specific cognitive domains. Further, environment has a significant influence on outcome and, along with injury severity, should be carefully considered when predicting TBI outcome and determining need for clinical follow-up and intervention.

Funding

The National Health and Medical Research Council of Australia, Foundation for Children and the Victorian Government’s Operational Infrastructure Support Program.

Conflicts of interest: None declared.

References


from eight to thirteen years. Child Neuropsychology, 3, 28–46.