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Predictors of longitudinal outcome and recovery of pragmatic language and its relation to externalizing behaviour after pediatric traumatic brain injury

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ABSTRACT

The purpose of the present investigation was to evaluate the contribution of age-at-insult and brain pathology on longitudinal outcome and recovery of pragmatic language in a sample of children and adolescents with traumatic brain injury (TBI). Children and adolescents with mild to severe TBI ($n = 112$) were categorized according to timing of brain insult: (i) Middle Childhood (5–9 years; $n = 41$); (ii) Late Childhood (10–11 years; $n = 39$); and (iii) Adolescence (12–15 years; $n = 32$) and group-matched for age, gender and socio-economic status (SES) to a typically developing (TD) control group ($n = 43$). Participants underwent magnetic resonance imaging (MRI) including a susceptibility weighted imaging (SWI) sequence 2–8 weeks after injury and were assessed on measures of pragmatic language and behavioural functioning at 6- and 24-months after injury. Children and adolescents with TBI of all severity levels demonstrated impairments in these domains at 6-months injury before returning to age-expected levels at 2-years post-TBI. However, while adolescent TBI was associated with post-acute disruption to skills that preceded recovery to age-expected levels by 2-years post injury, the middle childhood TBI group demonstrated impairments at 6-months post-injury that were maintained at 2-year follow up. Reduced pragmatic communication was associated with frontal, temporal and corpus callosum lesions, as well as more frequent externalizing behaviour at 24-months post injury. Findings show that persisting pragmatic language impairment after pediatric TBI is related to younger age at brain insult, as well as microhemorrhagic pathology in brain regions that contribute to the anatomically distributed social brain network. Relationships between reduced pragmatic communication and more frequent externalizing behavior underscore the need for context-sensitive rehabilitation programs that aim to increase interpersonal effectiveness and reduce risk for maladaptive behavior trajectories into the long-term post injury.

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1. Introduction

Childhood TBI is a common cause of childhood disability, and is associated with elevated risk for cognitive, social and behavioural impairment (Beauchamp & Anderson, 2010; Rosema, Crowe, & Anderson, 2012; Yeates et al., 2007). While evidence suggests that some children with TBI experience difficulties with basic aspects of expressive and receptive language (Chapman, Levin, Wanek, Weyrauch, & Kufera, 1998; Ewing-Cobbs et al., 1997; Sullivan & Riccio, 2010), such injuries can also be associated with long-term impairments in higher order aspects of pragmatic communication (Didus, Anderson, & Catroppa, 1999; Ryan et al., 2013; Sullivan & Riccio, 2010), defined as the ability to use and comprehend language in context (Watts & Douglas, 2006). Preliminary reports show that these impairments have profound consequences for social and behavioural functioning among individuals with TBI (Ryan et al., 2013; Yeates et al., 2004), but factors contributing to variability in outcome and recovery of pragmatic communication across individual children remain poorly understood.

Pragmatic communication emerges rapidly during middle childhood (Dennis & Barnes, 1990; Gerrard-Morris et al., 2010), mediated by a distributed network of brain regions implicated in a range of specific executive and social cognitive processes, including the anterior temporal lobes, orbitofrontal cortex, lateral frontopolar cortex, anterior prefrontal cortex and the inferior and superior parietal lobe (Barbey, Colom, & Grafman, 2013). Pragmatic communication impairments suggest that brain regions involved in these anatomically distributed neural networks may be vulnerable to the effects of TBI (Chapman et al., 2004; Didus et al., 1999; Ryan et al., 2013), however the challenge remains to identify factors that confer elevated risk for poor long-term outcome and recovery of these skills.

The Heuristic Model of Social Competence (Yeates et al., 2007) provides a framework for conceptualizing how injury-related, child, and environmental factors may explain variability in social outcome across individual survivors of childhood TBI. More specifically, the model postulates that various injury and non-injury-related risk and resilience factors may independently or interactively contribute to social outcome after childhood TBI. Injury factors, including injury severity, lesion location and timing of cerebral insult, are conceptualized as risk factors that increase the likelihood of impaired social information processing and communication. Environmental factors such as interventions and better family functioning represent sources of resilience that may buffer against the neurological consequences of injury. Moreover, in keeping with diathesis-stress perspectives (Yeates et al., 2007), the model accounts for the possibility that impairments in one or more aspects of neurocognitive functioning (social cognitive, cognitive–executive) may influence social and behavioural functioning either directly or indirectly via their influence on social interaction. For example, it may be that social communicative dysfunction associated with injury leads to rejection by peers and subsequent distress, reflected in externalizing behaviour symptoms (e.g. aggression, rule breaking, conduct problems) that persist or even worsen with time since injury (Alderman, 2003; Cattelani, Lombardi, Brianti, & Mazzucchi, 1998; Li & Liu, 2013; Ylvisaker, Turkstra, & Coelho, 2005).

While there is preliminary evidence for a dose–response relationship between injury severity and social outcomes (Catroppa & Anderson, 2004; Rosema et al., 2012; Ryan, Anderson, et al., 2014), damage to one or multiple components of anatomically distributed social cognitive neural networks is likely to disrupt acquisition of high-level social functions, including pragmatic communication (Kennedy & Adolphs, 2012). Based on lesion studies that link impairments in social functioning to damage to particular areas of the frontal and temporal cortices (Geraci, Surian, Ferraro, & Cantagallo, 2010; Muller et al., 2010), it may be that focal lesions to these brain regions contribute to impaired pragmatic language. Another approach may be to target the corpus callosum (CC) as an index of white matter disruption, and a common site of injury in pediatric TBI (Beauchamp, Ditchfield, Catroppa, et al., 2011; Levin et al., 2000). Since the CC grows and establishes white matter connecting pathways between high level association cortices implicated in a range of specific social cognitive and executive processes (Ewing-Cobbs et al., 2012), it is plausible that damage to this structure in childhood may disrupt structural connectivity and thus interfere with the acquisition and establishment of pragmatic language skills.

Age at brain insult may influence outcome and recovery of neurobehavioural skills (Anderson, Spencer-Smith, & Wood, 2011; Anderson et al., 2009; Jacobs, Harvey, & Anderson, 2007), although this is likely not a linear association, but rather influenced by critical periods during development, such that outcomes are dependent on neurological and cognitive development at the time of insult (Anderson et al., 2009, 2011; Crowe, Catroppa, Babl, Rosenfeld, & Anderson, 2012; Dennis, 1988; Dennis et al., 2014; Kolb, Pellis, & Robinson, 2004). It has been argued that neurocognitive skills emerging or developing at the time of insult are at heightened risk for persisting disruption, while established skills may experience transient disruption before recovering to levels that approximate pre-injury baseline (Dennis, 1988; Dennis et al., 2014). Since pragmatic communication skills undergo protracted development through the early school years and into adolescence (Didus et al., 1999; Dumontheil, Apperly, & Blakemore, 2010; Gerrard-Morris et al., 2010), a critical period model would predict that these skills are at heightened risk for disruption during middle childhood, when they are undergoing rapid development and refinement. Although there is preliminary evidence that poorer pragmatic language is associated with younger age at brain insult (Chapman et al., 1998; Didus et al., 1999), the limited size and age range of previous samples underscores the need for further research to evaluate the contribution of timing of cerebral insult to longitudinal outcome and recovery of these skills.

The purpose of the present prospective longitudinal study was to investigate (1) the contribution of age-at-insult to outcome and recovery of pragmatic communication after TBI sustained in middle childhood (5–9 years), late childhood (10–11 years) and early adolescence (12–15 years); (2) examine relations between pragmatic communication, injury severity and brain pathology; and (3) evaluate relationships between pragmatic communication and externalizing symptoms at 6- and 24-months post-TBI.

In keeping with critical period perspectives (Anderson et al., 2009, 2011; Crowe et al., 2012; Dennis, 1988; Dennis et al., 2014; Kolb et al., 2004), we expected that relative to typically developing (TD) controls, children sustaining TBI in middle childhood would show impaired pragmatic language at 6- and 24-months post injury. Conversely, TBI in late childhood and adolescence would be associated with significantly reduced performance at 6-months post-injury, but comparable performance to TD controls at 24 months, reflecting recovery of pre-injury function. We also predicted that poorer pragmatic communication would be associated with (i) greater injury severity, (ii) frontal and temporal brain pathology, and (iii) white matter pathology (i.e. corpus callosum lesions). Consistent with diathesis-stress perspectives (Yeates et al., 2007), we predicted that poorer pragmatic communication would be associated with more frequent externalizing symptoms, as measured by aggression, conduct problems, and rule breaking at 6- and 24-months post injury.