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CRITICAL REVIEW

Impairments in Social Cognition Following Severe Traumatic Brain Injury

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Abstract

Severe traumatic brain injury (TBI) leads to physical, neuropsychological, and emotional deficits that interfere with the individual's capacity to return to his or her former lifestyle. This review focuses on social cognition, that is, the capacity to attend to, recognize and interpret interpersonal cues that guide social behavior. Social cognition entails "hot" processes, that is, emotion perception and emotional empathy and "cold" processes, that is, the ability to infer the beliefs, feelings, and intentions of others (theory of mind: ToM) to see their point of view (cognitive empathy) and what they mean when communicating (pragmatic inference). This review critically examines research attesting to deficits in each of these domains and also examines evidence for theorized mechanisms including specific neural networks, the role of simulation, and non-social cognition. Current research is hampered by small, heterogeneous samples and the inherent complexity of TBI pathology. Nevertheless, there is evidence that facets of social cognition are impaired in this population. New assessment tools to measure social cognition following TBI are required that predict everyday social functioning. In addition, research into remediation needs to be guided by the growing empirical base for understanding social cognition that may yet reveal how deficits dissociate following TBI. (*JINS*, 2013, *19*, 231–246)

KeyWords: Emotion perception, Theory of mind, Pragmatics, Brain injury, Empathy

INTRODUCTION

Traumatic brain injury (TBI) arises from motor vehicle accidents, warfare, assaults, and accidents. Severe injuries[1] lead to protracted coma and/or altered consciousness acutely and to chronic physical, neuropsychological and emotional deficits that interfere with the resumption of former lifestyles. According to their relatives, changes in behavior and personality, for example, childishness, self-centeredness, disinterest or dislike of others, quarrelsome, unreasonable or socially inappropriate behavior, unhappiness, and excitation are frequent and chronic (Brooks, Campsie, Symington, Beattie, & McKinlay, 1986; Brooks & McKinlay, 1983; Kinsella, Packer, & Olver, 1991; McDonald & Saunders, 2005; McKinlay, Brooks, Bond, Martinage, & Marshall, 1981; Thomsen, 1984). Such changes predict relative stress (Brooks et al., 1986; Brooks & McKinlay, 1983; Schönberger, Ponsford, Olver, & Ponsford, 2010) and poor social adjustment and participation (Cattran, Oddy, Wood, & Moir, 2011).

In 1978, Lezak described impaired capacity for social perceptiveness as a key feature of the characterological changes seen post injury (Lezak, 1978). Thirty-five years later research into the mechanisms underpinning poor social perceptiveness is only just commencing, fuelled by the growing field of social neuroscience. A central construct is social cognition, that is, the ability to understand other people (Lieberman, 2007). Social cognition enables us to predict the behavior of others, share experiences and communicate effectively. As the human species relies upon cooperation and competition within groups to survive, social cognition is argued to be an evolutionary imperative, resulting in its modular development independent of non-social information processing skills (Adolphs, 2003). Behaviorally, there is evidence for dissociations between non-social and social cognition. Individuals with discrete frontal lesions from trauma or other

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[1] Definitions of severity of TBI have changed over the past decades. Originally a severe TBI was defined as that incurring a period of altered consciousness of 1 day or greater and very severe as 7 days or more (Russell & Smith, 1961) and much or the literature has used this definition. Classifications have since moved toward defining a period of altered consciousness of 1-7 days as reflecting a moderate injury with severe injuries associated with a period of confusion of longer than 7 days (Williamson, Scott, & Adams, 1996). The research cited in this study is based upon the original definition of "severe" with the understanding that this will sometimes include those with moderate injuries as defined by other standards.

pathology often present with social functioning that is disproportionately impaired relative to intellect (e.g., Blair & Cipolotti, 2000; Cicerone & Tanenbaum, 1997; Eslinger & Damasio, 1985; Tranel, Bechara, & Denburg, 2002).

At base, social cognition entails the ability to construct representations of the mental states of others, that is, their beliefs, feelings, experiences, and intentions, in relation to ourselves and to use these flexibly to guide social behavior (Adolphs, 2001; Amodio & Frith, 2006). These are matters that cannot directly be observed but must be inferred from both incoming stimuli and our knowledge of the social world. Conceptually, a distinction is drawn between "hot" social cognition, that is, emotion processing including identifying and empathizing with another's emotional state and "cold" social cognition that is, thinking about things from another's point of view, including Theory of Mind (ToM) abilities. The discovery of "mirror" neuron systems in the premotor cortex that are activated when observing the actions of others (Rizzolatti & Sinigaglia, 2010), along with physiological evidence of mimicry (discussed further below), has spurred theorizing that social cognition encompasses simulation, that is, the representation of the minds and experience of others in oneself as a means to understand them. To this end, it is critical to be self-aware, knowing one's own mind to represent others and also to differentiate between self and other. Effortful control ensures that emotional responses are regulated, the perspective between self and other is maintained, we are able to put social information in context and we can flexibly accommodate changing social input. These aspects of social cognition are summarized in Figure 1.

The extent to which social cognition is modular is hotly debated. In part this arises because there are different levels of social cognitive processing, not all of which are specialized. Perception of social stimuli entails both conscious explicit processing (e.g., *via* visual cortex) and also rapid coarse processing *via* the superior colliculi. Perception is specialized for different inputs (facial expressions, prosody, biological movement) (Adolphs, 2010). Evaluation and interpretation of social information also appears to be mediated by a specialized system of interconnected networks involving the orbital and ventromedial frontal cortex, cingulate cortex and striatum, insula, and amygdala. These structures orchestrate the automatic, often implicit, appraisal of emotionally salient information and mental states (Adolphs, 2009; Lieberman, 2007; Phillips, Drevets, Rauch, & Lane, 2003). Finally, effortful regulation of responses and contextualization is mediated by dorsal regions of the lateral and medial prefrontal cortex in concert with the hippocampus and temporo-parietal zones (Lieberman, 2007; Phillips et al., 2003). Unlike the former stages, these cognitive and memory processes are probably generic and not specific to social cognition.

Structures underlying social cognition are vulnerable to severe traumatic brain injury. Although TBI produces variable multifocal and diffuse neuropathology, typical patterns arise due to acceleration-deceleration forces that scrape the soft brain tissue across the bony floor of the anterior and middle fossa of the skull (Bigler, 2007). Medial frontal surfaces are compressed against the dorsal bone and collide with the cerebral falx (Bigler, 2007). Immediate contusions and wallerian

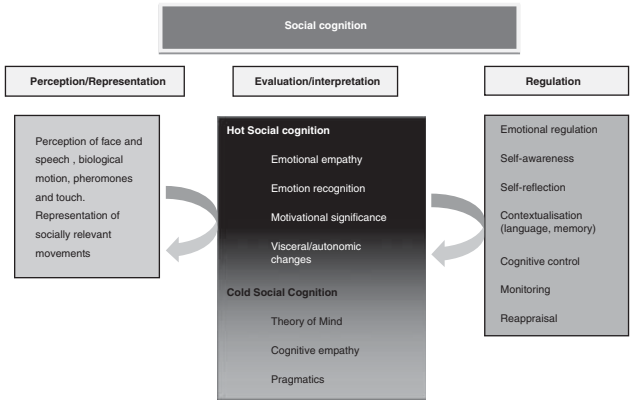

Fig. 1. Processes in social cognition, adapted from Adolphs (2010).

degeneration causes disruption to medial regions and their connections. Thus, pathology is often concentrated in the ventrolateral, medial and orbital frontal lobes and the ventromedial temporal lobes (Bigler, 2007; Bigler & Maxwell, 2011; Courville, 1945; Gentry, Godersky, & Thompson, 1988; Hadley et al., 1988). There is also diffuse axonal injury to the brainstem, corpus callosum, and the gray-white matter junctions of the cerebral cortex (Adams et al., 1989; Meythaler, Peduzzi, Eleftheriou, & Novack, 2001; Viano et al., 2005) further disrupting connections between subcortical and frontal systems (Kennedy et al., 2009) and possibly somatosensory and motor cortex (Green, Turner, & Thompson, 2004). As the ventromedial and orbital frontal lobes are highly vulnerable in TBI, many psychosocial problems reported may usefully be examined within the rubric of disorders of social cognition. This is not, however, a simple task. The neuropathology of TBI is complex and highly variable. No one individual with TBI has identical deficits to another. Furthermore, the information processing requirements of social cognition are only beginning to be understood. At this time, it is clear they represent a complex interplay between specifically social facets of processing and generic cognitive, memory and executive functions. This hampers conclusions as to the extent to which specific deficit in social cognition arise following TBI and also the identification of subtypes. The goal of this review is to critically evaluate the evidence for disorders in "hot" social cognition, that is, affective empathy, emotion perception and emotional resonance and "cold" cognition, that is, ToM, cognitive empathy and pragmatics following severe TBI. A further aim is to consider whether some of the hypothesized mechanisms underpinning social cognition, developed from the normal literature, functional neuroimaging, and focal lesion research, are relevant to explaining social cognition deficits post TBI.

Disorders of "Hot" Social Cognition

Affective empathy

Affective empathy refers to the ability to emotionally resonate with others' feelings while understanding that they are distinct from one's own (Baron-Cohen & Wheelwright, 2004).

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Using self-report measures such as the Balanced Emotional Empathy Scale (Mehrabian, 2000), 60–70% of adults with severe TBI self-report little to no emotional empathy compared to 30% of matched control participants (de Sousa, McDonald, & Rushby, 2012; de Sousa et al., 2010, 2011; Williams & Wood, 2010; Wood & Williams, 2008). The use of self-report measures has been criticized in TBI research as these are vulnerable to loss of insight, attentional bias and cognitive impairments affecting complex language processing, attention and flexibility. Despite this, evidence suggests they can be a valid measure of emotional changes following even severe TBI (Kinsella, Moran, Ford, & Ponsford, 1988). Furthermore, the relatively similar incidence rates using self-report across studies gives validity to the claim that empathy is reduced after TBI. Self-reported empathy is unrelated to injury severity (length of post-traumatic amnesia), time since injury or co-existing cognitive deficits (Williams & Wood, 2010; Wood & Williams, 2008) raising questions as to the cause of this self-perceived deficit. Emotional empathy is a complex construct entailing emotion perception, emotional resonance, self-awareness, and regulation. Consequently an examination of components may be more revealing as discussed below.

Emotion Perception

Facial emotion

Empirical research into emotion perception deficits following TBI commenced in the 1980s (Braun, Baribeau, Ethier, Daigneault, & Proulx, 1989; Jackson & Moffatt, 1987; Prigatano & Pribam, 1982) and since then, a plethora of studies have reported deficits in the recognition of photographs of facial expressions in adults with both acute and chronic severe TBI (Borgaro, Prigatano, Kwasnica, Alcott, & Cutter, 2004; Croker & McDonald, 2005; Green et al., 2004; Ietswaart, Milders, Crawford, Currie, & Scott, 2008; Knox & Douglas, 2009; McDonald & Saunders, 2005; Milders, Fuchs, & Crawford, 2003; Milders, Ietswaart, Crawford, & Currie, 2008; Spell & Frank, 2000). While samples sizes in this area are typically small, a meta-analysis of 296 adults with moderate-severe TBI from 13 studies (Babbage et al., 2011) indicated a relatively large effect size (1.1 *SD*) differentiating people with TBI from matched controls. Overall, it was estimated that up to 39% of people with severe TBI experience deficits in recognizing emotions from static presentations of facial expressions.

Static photographic stimuli bear little resemblance to naturally occurring facial expressions which are dynamic, evolve rapidly from one emotion to another and provide additional cues *via* facial movement (Bassili, 1978). Dissociations between recognition of static and dynamic expressions have been reported in patients with non-traumatic brain lesions (Adolphs, Tranel, & Damasio, 2003; Humphrey, Donnelly, & Riddoch, 1993) which suggests separable neural systems; ventral fronto-temporal systems mediating static images and dorsal fronto-parietal zones mediating facial movement (Adolphs et al., 2003). As the ventral fronto-temporal lobes are especially vulnerable to TBI due to their position within the anterior and middle fossa (Bigler & Maxwell, 2011) disorders recognizing static expressions as a result of focal pathology may be expected to occur more frequently than disorders recognizing dynamic images. In one study that directly compared the two this was found to be the case, that is, 8/34 participants *versus* 1/34, respectively (McDonald & Saunders, 2005).

Brain-behavior relationships are difficult to establish in TBI, in part because of the heterogeneity of the TBI population. Where subgroups have been compared, differences in emotion perception scores between those with frontal *versus* other pathology have been marginal or insignificant (Ietswaart et al., 2008; McDonald & Flanagan, 2004). Other confounds complicate the picture. Slowed processing speed and poor cognitive flexibility interfere with emotion perception tasks, both static (Ietswaart et al., 2008) and dynamic (McDonald & Saunders, 2005) and, indeed, in one study (Ietswaart et al., 2008) these entirely accounted for between group differences. Injury severity, indexing extent of cognitive impairment, also partially predicts poor performance (Ietswaart et al., 2008; McDonald & Saunders, 2005). Arguably, complex, dynamic display of emotions tax cognitive abilities more than static (Knox & Douglas, 2009) and certainly additional skills have been found to contribute to dynamic emotion recognition including premorbid intellectual ability, working memory, reasoning and new learning (McDonald et al., 2006). While convergent evidence from various sources (as will be discussed below) suggests that impairment in facial emotion recognition is a real problem for many people with TBI, the correlation between indices of severity, various neuropsychological measures and behavioral responses to emotion identification does suggest that incidence figures are likely to be inflated.

Vocal Emotion

Recognition of emotional expression in voice is also impaired following TBI (Dimoska, McDonald, Pell, Tate, & James, 2010; Hornak, Rolls, & Wade, 1996; McDonald & Pearce, 1996; McDonald & Saunders, 2005; Milders et al., 2003, 2008; Spell & Frank, 2000). Emotional prosody engages brain systems (especially right hemisphere) which overlap but do not entirely coincide with those engaged in facial expressions (Adolphs, Damasio, & Tranel, 2002). Consequently, dissociations on the basis of neuropathology might also be expected in TBI and there is evidence for this both in terms of individual patients having problems in face not voice or vice versa (Hornak et al., 1996) and also in a lack of correlation between face *versus* voice discrimination (McDonald & Saunders, 2005).

Confounding this issue, however, is the fact that tasks of prosody and face recognition are often not well equated (Ietswaart et al., 2008). In one study where effort was made to equate them, differences emerged to suggest more participants experienced significant impairment with (static) facial emotion than vocal emotion but the group, as a whole, experienced a loss of efficiency with prosody (McDonald & Saunders, 2005). This highlights an inherent problem with

this field of research, that is, face and voice discrimination have different cognitive demands that, in general, might facilitate facial processing. First, facial processing provides greater scope for additional strategies (e.g., the use of verbalization) (Hornak et al., 2003). Second, emotions in voice are conveyed by two sources: speech content and quality, making dual processing and working memory demands (Dimoska et al., 2010). Thus, impaired recognition of prosody may reflect a loss of efficiency that is not specific to vocal emotion. Nevertheless, more general cognitive impairment cannot fully account for deficits in prosodic perception. For example this does not explain differential impairment across categories of emotion (Dimoska et al., 2010; Spell & Frank, 2000). In addition, when semantic content is experimentally reduced, problems with prosody are amplified, suggesting a difficulty processing the tonal quality *per se* (Dimoska et al., 2010).

Potential Mechanisms Underpinning Impaired Emotion Processes

Research into both normal adults and those with focal lesions has provided a more detailed account of emotion processing. This has motivated studies in TBI that focus upon specific impairment in the processing of negatively valenced stimuli, as well as the role of simulation and self-awareness.

Differential impairment in processing negatively valenced materials post TBI

The ventromedial frontal regions, amygdala and insula appear to be preferentially geared to rapidly orientate to and process threat related emotions (Adolphs, 2002; Adolphs, Russell, & Tranel, 1999; Adolphs & Tranel, 2004; Graham, Devinsky, & LaBar, 2007; Harmer, Thilo, Rothwell, & Goodwin, 2001; Phillips et al., 1997; Sato et al., 2002). Differential impairment in the perception of negative expressions (fear, disgust, sadness, and anger) relative to positive is found in TBI studies (e.g., Braun et al., 1989; Callahan, Ueda, Sakata, Plamondon, & Murai, 2011; Croker & McDonald, 2005; Dimoska et al., 2010; Hopkins, Dywan, & Segalowitz, 2002; Jackson & Moffat, 1987; McDonald, Flanagan, Rollins, & Kinch, 2003; Prigatano & Pribam, 1982). This could be construed as evidence for deficits to the ventromedial system although the pattern is not always seen (Ietswaart et al., 2008; McDonald & Saunders, 2005) so in these cases there must be more pervasive impairment or the contribution of other factors. Another consideration for both TBI research and more generally, is the uneven representation of positive [happy and sometimes (pleasant) surprise] and negative (angry, sad, fearful, disgust) emotions skewed further by the almost universal recognition of happy expressions. Thus, differential impairment in the recognition of negative expressions may reflect the nature of the materials rather than difficulties with particular categories of emotion *per se*. Evenso, throughout the psychological literature, there does appear to be a pattern whereby negative events are afforded preferential treatment over positive (Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001). Furthermore, other evidence (below) reinforces the view that processing of negative emotions is especially vulnerable to TBI.

Impaired physiological responsivity

Another feature of the ventromedial system is that it mediates autonomic responses to emotional stimuli even before conscious awareness (Phillips et al., 2003). This may also be compromised as a result of severe TBI. A minority self-report that their emotional experiences are dulled (Croker & McDonald, 2005; Hornak et al., 1996) and many studies have reported reduced physiological reactivity to unpleasant stimuli, e.g., reduced modulation of the startle reflex (Sanchez-Navarro, Martınez-Selva, & Roma′n, 2005; Saunders, McDonald, & Richardson, 2006), dampened skin conductance changes (arousal) and reduced facial reactivity when viewing affectively valenced pictures and films (de Sousa et al., 2012, 2010; Soussignan, Ehrle, Henry, Schaal, & Bakchine, 2005). This has been reported for both positive and negative stimuli (de Sousa et al., 2012; Sanchez-Navarro et al., 2005; Soussignan et al., 2005) but also specifically to negative (Angrilli, Palomba, Cantagallo, Maietti, & Stegagno, 1999; de Sousa et al., 2010; Saunders et al., 2006). In some reports, changes in physiological responses to negative images corresponded to subjective reports that they did not find the stimuli arousing (de Sousa et al., 2012, 2010; Saunders et al., 2006) although a dissociation between physiological changes and subjective report has also been reported (Sanchez-Navarro et al., 2005; Soussignan et al., 2005).

Simulation following TBI

Simulation appears to be intrinsic to emotion perception. Adults typically demonstrate facial mimicry (Dimberg & Lundquist, 1990; Dimberg & Petterson, 2000; Dimberg & Thunberg, 1998), changes in skin conductance (Merckelbach, van Hout, van den Hout, & Mersch, 1989; Vrana & Gross, 2004) and subjective experience (Hess & Blairy, 2001; Wild, Erb, & Bartels, 2001) when viewing facial expressions. In turn, facial movements alter emotional experience (Adelman & Zajonc, 1989; Levenson et al., 1990) and the emotional state of the observer influences recognition of emotional states in others (Neidenthal, Brauer, Halberstadt, & Innes-Ker, 2001). At a neural level, the mirror neuron system in the premotor cortex is activated when viewing facial expressions (Carr, Iacoboni, Dubeau, Maxzziotta, & Lenzi, 2003; Kilts, Egan, Gideon, Ely, & Hoffman, 2003). Activation of the somatosensory cortex also occurs, thought to provide the viewer with sensory cues "as if" the expression were their own (Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000) while autonomic changes reflect ventromedial activation.

In TBI, specific impairment in the early automatic mimicry of angry expressions relative to happy has been reported (McDonald, Li, et al., 2011) along with reduced skin conductance changes (Blair & Cipolotti, 2000; de la Plata et al., 2011; Hopkins et al., 2002). Impairment is specific to angry expressions providing further evidence for differential

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impairment with negatively valenced emotions. It also discounts explanations based upon motor paralysis or deficits in mirroring, both of which should affect all emotions. It is consistent with an impairment of processing mediated by the ventromedial system.

Relation between simulation and emotion processes

Simulation is argued to be an implicit component of emotion recognition, providing cues that aid recognition (Goldman & Sripada, 2005; Neidenthal et al., 2001). However, evidence for the simulation theory in TBI studies is weak. On the one hand, subjective reports of altered emotional experience after TBI do correlate with emotion perception accuracy (Croker & McDonald, 2005) and poor emotion perception and impaired emotional responses can co-occur in individual patients (Blair & Cipolotti, 2000). But, in general, correlations between mimicry and/or skin conductance and emotion perception have, to date, been insignificant (McDonald, Li, et al., 2011; McDonald, Rushby, et al., 2011). This lack of concordance is also seen in normal populations (Blairy, Herrera, & Hess, 1999; Hess & Blairy, 2001) and casts doubt on the role of simulation in emotion perception. Simulation does, however, have a clearer role in empathy, providing both a vicarious empathic reaction (McIntosh, 1996) and a communicative role, conveying an understanding of the situation. For example, mimicry has been found to vary systematically with the extent to which the participant knows s/he is being observed (Bavelas, Black, Chovil, Lemery, & Mullett, 1988; Bavelas, Black, Lemery, & Mullett, 1986). There is evidence that impaired automatic mimicry is related to low emotional empathy in both people with TBI (de Sousa et al., 2011) and normal adults (Sonnby-Borgström, Jönsson, & Svensson, 2003). Deficits in motor mimicry also extend to the capacity to make emotional expressions whether spontaneous or posed. This is not a motor impairment *per se* as happy expressions are normal (Dethier, Blairy, Rosenberg, & McDonald, 2012).

Impairments in self-awareness and self-regulation

Self-awareness and self-regulation are necessary to emotional empathy so as to recognize one's own emotional experience, to see it as separate from the other and to control it effectively (Decety & Meyer, 2008). Self-awareness appears to be impaired in severe TBI although empirical evidence comes from a scattered, relatively small literature. People with severe TBI reportedly have impaired sensitivity to internal somatic states, specifically their own heartbeat (Hynes, Stone, & Kelso, 2011). They also report less congruent mood changes when adopting a body posture consistent with an angry or sad emotional state compared to happy (Dethier, Blairy, Rosenberg, & McDonald, in press).

Poor self-awareness is also documented in studies of alexithymia., that is, difficulties identifying and describing one's emotions and physiological reactions. Using the Toronto Alexithymia Scale (Bagby, Parker, & Taylor, 1994), between 32 and 58% of convenience samples of people with TBI self-report alexithymia (Allerdings & Alfano, 2001; Henry, Phillips, Crawford, Theodorou, & Summers, 2006; Koponen et al., 2005; McDonald, Rosenfeld, et al., 2011; Williams et al., 2001; Wood & Williams, 2007) compared to the much lower incidence in the general population (7–15%) (Koponen et al., 2005; Pasini, Chiale, & Serpia, 1992). Furthermore, alexithymia post TBI is reportedly associated with empathy (Williams & Wood, 2010). Although this is consistent with expectations based on theory, caution regarding the validity of the construct of alexithymia (literally "without words for emotions") in the TBI population is required. Given its strong association with poor verbal and working memory capabilities (Wood & Williams, 2007) it is not entirely clear what alexithymia represents in cognitively impaired people with TBI.

Loss of self-awareness is intrinsically related to self-regulation. Indeed, alexithymia and poor self-regulation are linked in many clinical populations (Connelly & Denney, 2007; Taylor, Bagby, & Parker, 1997). Disorders of emotion regulation are common following TBI, manifest as apathy (disorder of drive) or poor frustration tolerance and disinhibition (disorders of control) (Kinsella et al., 1991; Tate, 1999) and these too, have been linked to both alexithymia (Koponen et al., 2005) and empathy (de Sousa et al., 2012) although, once again, the research is scant and preliminary.

Mood disorders

A major consideration for emotion processing in severe TBI is the prevalence of depression and anxiety (Bombardier et al., 2010). In non-brain injured populations, depression impairs emotion perception (Langenecker et al., 2005; Leppänen, Milders, Bell, Terriere, & Hietanen, 2004) and empathy (Cusi, MacQueen, Spreng, & McKinnon, 2011) and has similarly altered brain circuits to those discussed in relation to TBI (Cusi, Nazarov, Holshausen, MacQueen, & McKinnon, 2012). Many TBI studies have addressed this confound by either matching groups for depression and anxiety or examining the contribution statistically. These found that mood disorders were not the major contributor to impairment in emotion perception (Ietswaart et al., 2008; Milders et al., 2008) or empathy (de Sousa et al., 2010; Wood & Williams, 2008) although they were co-morbid with alexithymia (Wood & Williams, 2007).

Overall, it is reasonable to conclude that deficits in emotion perception and empathy are a consequence of TBI. However, these are unlikely to be uniform, representing a complex admixture of impairment arising from structural lesions underpinning emotion processes, mood disorders, and cognitive impairments, overlaid upon pre-existing personality attributes. Future research may identify subtypes of emotion processing disorders in TBI but at this time, the evidence is too exploratory and the numbers too few.

"Cold" Social Cognition

Cold social cognition entails the ability to explain one's own and others' behavior on the basis of thoughts, intentions and

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beliefs, that is, to have a theory of Mind (ToM) (Castelli, Frith, Happé, & Frith, 2002, p. 1839). It also refers to the ability to use ToM to appreciate another's point of view in addition to one's own, that is, to have cognitive empathy (Rogers, Dziobek, Hassenstab, Wolf, & Convit, 2007, p. 709) and to use ToM to understand pragmatic inference, that is, intended meanings in communication. Impairment of these inter-related domains following severe TBI is suggested by several indicators. Relatives report their person with TBI to be self-centered (Kinsella et al., 1991), insensitive (Brooks & McKinlay, 1983) and disinterested and childish (Elsass & Kinsella, 1987; Thomsen, 1984). Experimental tasks demonstrate that adults with TBI have difficulty identifying the source of interpersonal conflict or the meaning of social behavior in stories or videoed interactions (Channon & Crawford, 2010; Hynes et al., 2011; Kendall, Shum, Halson, Bunning, & Teh, 1997; Turkstra, 2008), interpreting non-verbal interpersonal interactions (Bara, Cutica, & Tirassa, 2001; Cicerone & Tanenbaum, 1997) and filling out a personality questionnaire as though they were someone else (Spiers, Pouk, & Santoro, 1994).

Cognitive empathy

Cognitive empathy is normally assessed using self-report scales such as the Interpersonal Reactivity Scale (Davis, 1983), the Empathy Scale (Hogan, 1969) and the Brock Adaptive Functioning Questionnaire (Hopkins et al., 2002). Using these, individuals with TBI self-report lower cognitive empathy than do matched controls (de Sousa et al., 2010; Grattan & Eslinger, 1989; Wells, Dywan, & Dumas, 2005). In these convenience samples (the study by Grattan & Eslinger had a mixed neurological group, including TBI), the incidence of impaired cognitive empathy was around 50% (de Sousa et al., 2010; Grattan & Eslinger, 1989) and was associated with high distress in care-givers (Wells et al., 2005).

ToM tasks

ToM is conventionally measured using laboratory tasks, typically relying upon comprehension of stories, cartoons, photos and videos. Adults and children with severe TBI often fare poorly on these. In a recent meta-analysis based upon 173–354 adults with acquired brain injury, roughly 50% of whom had TBI, effect sizes for ToM tasks were moderate to large (0.5–0.7) and this was true for the TBI group alone (Martin-Rodriguez & Leon-Carrion, 2010). Tasks include comprehension of complex stories that require knowing that one of the protagonists is operating on a false belief or has committed a faux pas (Bibby & McDonald, 2005; Geraci, Surian, Ferraro, & Cantagallo, 2010; Milders et al., 2003; Milders, Ietswaart, Crawford, & Currie, 2006; Milders et al., 2008; Spikman, Timmerman, Milders, Veenstra, & van der Naalt, 2012; Stone, Baron-Cohen, & Knight, 1998; Turkstra, Williams, Tonks, & Frampton, 2008), appreciating jokes based upon understanding the character's thoughts (Bibby & McDonald, 2005; Milders et al., 2006, 2008; Spikman et al., 2012), and predicting the intentions of characters in cartoon sequences (Havet-Thomassin, Allain, Etcharry-Bouyx, & Le Gall, 2006; Muller et al., 2010). People with TBI also have difficulty making judgments about mental states based upon the eye region of the face (Geraci et al., 2010; Havet-Thomassin et al., 2006; Henry, Phillips, Crawford, Ietswaart, & Summers, 2006; Turkstra et al., 2008) or deducing thoughts and intentions of speakers in video vignettes (McDonald & Flanagan, 2004; Turkstra, Dixon, & Baker, 2004).

Pragmatics

ToM plays a critical role in *pragmatics*, that is, language use. For example, when giving instructions, the speaker needs to think about the task from the listener's point of view and structure the explanation accordingly. When asking a favor they need to anticipate potential objections when framing their request. On occasion they may need to avoid offence by hinting what is on their mind. The incidence of aphasia post TBI is generally low (2–30% (Heilman, Safran, & Geschwind, 1971; Sarno, 1980, 1984, 1988; Sarno & Levita, 1986), yet 43% of mothers, when surveyed, reported language impairment (Kinsella et al., 1991). This suggests there were additional difficulties *using* language effectively. In the absence of aphasia, difficulties with language use have also been documented on tasks that require tailoring responses to the listener's needs (McDonald, 1993; McDonald & Pearce, 1995, 1998; McDonald & Van Sommers, 1993; Turkstra, McDonald, & Kaufmann, 1996).

ToM is pivotal to comprehension of pragmatic inference. Speakers often allude to what they mean indirectly, or politely lie when diplomacy is required. Alternatively, they may assert the opposite to what they mean to ridicule or scorn (i.e., be sarcastic). To comprehend pragmatic inference, listeners need to impute what the speaker intends by their remarks from facial and body cues and also an understanding of the context. Children, adolescents and adults with severe TBI are reportedly poor at comprehending pragmatic inference in text and in videoed vignettes (Channon & Crawford, 2010; Channon, Pellijeff, & Rule, 2005; Dennis, Purvis, Barnes, Wilkinson, & Winner, 2001; McDonald & Flanagan, 2004; McDonald et al., 2003; McDonald & Pearce, 1996; Shamay-Tsoory, Tomer, & Aharon-Peretz, 2005; Turkstra et al., 2004; Turkstra, McDonald, & DePompei, 2001). In comparison to other ToM tasks, performances on pragmatic inference tasks yield the largest effect size (0.87) (Martin-Rodriguez & Leon-Carrion, 2010). Furthermore, there is a significant relation between impairments in ToM, cognitive empathy and comprehension of sarcasm (Channon et al., 2005; McDonald & Flanagan, 2004; Shamay, Tomer, & Aharon-Peretz, 2002). Other types of pragmatic communication have also been reported to be impaired, such as understanding inferred meanings in real-world ambiguous advertisements which rely upon a play on words (Pearce, McDonald, & Coltheart, 1998), or making judgments about the social skills of conversational partners (such as whether they are able to share the conversation equally) (Turkstra et al., 2004). It is inevitable

that language problems *per se* will compound difficulties with pragmatic inference. Most experimental tasks have control tasks with similar language demands but still reveal problems specific to pragmatic inference. Potentially more difficult to partial out, is the reliance of such tasks on working memory and information processing speed (McDonald et al., 2006).

Potential Mechanisms Underpinning ToM

As with social cognition more broadly, it has been speculated that ToM is a specialized, modular, indeed unique, feature of human cognition (Havet-Thomassin et al., 2006; Rowe, Bullock, Polkey, & Morris, 2001) independent of generic cognitive skills. However, modularity has been difficult to demonstrate empirically, especially within the heterogeneous TBI population. Two approaches have been used, behavioral tasks and neuroimaging.

Relation between ToM and non-social reasoning

One approach to establishing modularity is to examine the association between ToM performance and standard neuropsychological tests. An inherent confound is that different ToM tasks (e.g., stories *vs*. photographs) rely differentially upon visual attention, language, etc. They also vary in complexity making disparate demands upon flexibility, working memory, learning and abstract reasoning, abilities that are often compromised as a result of TBI. Research samples are often small, making it even more difficult to find general patterns across studies. Unsurprisingly, evidence for the independence of ToM is mixed.

Several research studies have reported a lack of association between measures of cognitive processes, especially executive function and ToM (Havet-Thomassin et al., 2006; Muller et al., 2010; Spikman et al., 2012). However, this is not a universal finding and, indeed, individual measures of working memory, processing speed, inhibition and flexibility have been correlated with ToM performance (Bibby & McDonald, 2005; Channon & Crawford, 2010; Dennis, Agostino, Roncadin, & Levin, 2009; Havet-Thomassin et al., 2006; Henry, Phillips, Crawford, Ietswaart, et al., 2006; Milders et al., 2006; Turkstra, 2008) and cognitive empathy (Grattan & Eslinger, 1989; Shamay-Tsoory, Tomer, Goldsher, Berger, & Aharon-Peretz, 2004). The strength of associations that are reported also varies. In Dennis et al. (2009), a study of school aged children with TBI, it was concluded that poor ToM was entirely accounted for by cognitive inhibition and working memory deficits, that is, the ToM requirements were non-domain specific. On the other hand, Bibby and McDonald (2005) examining adults with severe TBI found that while simple first order ToM (i.e., understanding what a person thinks) was not reliant upon working memory and general inferencing capacity, more complex, second order ToM tasks (i.e., understanding what one person thinks about another person's thoughts) were, suggesting that the former may be tapping into a particular ToM impairment.

An alternative approach is to compare performance on a mental inference task with a similar task that requires non-mental inferences. Here too, results are equivocal, that is, both verbal and pictorial non-mental inference analogue tasks are frequently impaired (Bibby & McDonald, 2005; Martin & McDonald, 2005; Milders et al., 2006; Muller et al., 2010) although not always (Channon & Crawford, 2010; Milders et al., 2003; Muller et al., 2010). Failure on non-mental inferences does not preclude additional requirements in the ToM version of the task. The clearest way to reveal specific ToM deficits would be to statistically control for performance on non-mental inferencing when examining ToM performance. This is rarely reported but when it has been, it seems that much of the deficits in ToM tasks can be explained by similar deficits on other inference making tasks although, again, not for simple ToM (Bibby & McDonald, 2005). In general, it would appear that there are common processes required for social and non-social tasks, depending upon the medium and response requirements (spoken, written, etc.), but there are unique requirements called into play when making ToM judgments.

Neural accounts

A recent approach to ToM is to use functional neuroimaging during simple tasks that require thinking about mental states. In healthy adults this paints a complex picture of composite processes (Frith & Frith, 2010, 2003; Schmitz, Rowley, Kawahara, & Johnson, 2006). The temporo-parietal junction is activated (Castelli, Frith, Happé, & Frith, 2002) when viewing animated movement (such as when viewing light points attached to actors filmed in the dark; Heberlein, Adolphs, Tranel, & Damasio, 2004) and people readily infer intention from such cues, even when viewing inanimate objects programmed to move and interact (Heider & Simmel, 1944). Greater activation occurs in the right temporo-parietal junction when oneself is agent (Decety & Meyer, 2008). The medial prefrontal cortex is consistently implicated in any task requiring the participant to think about themselves, regardless of its medium (verbal, visual, emotional, spatial) (Northoff et al., 2006) and also when thinking about others who are similar to self (Mitchell, Banaji, & Macrae, 2005b) raising the specter of simulation, that is, self-reference, when understanding the mental state of others.

Posterior dorsal regions of the (especially left) medial prefrontal cortex, attributed to action monitoring and updating (Amodio & Frith, 2006) are engaged when considering psychological attributes (Mitchell, Banaji, & Macrae, 2005a, 2005b), especially from the viewpoint of a third person (D'Argembeau et al., 2007). Inferior dorsolateral and orbitofrontal regions, known to play a role in inhibition of inappropriate responses (Collette et al., 2001; Nigg, 2001) are also activated when considering the perspective of another and may reflect the need to inhibit one's own perspective to do so (D'Argembeau et al., 2007; Ruby & Decety, 2004). Finally, temporal pole activation (especially left) is common (D'Argembeau et al., 2007; Frith & Frith, 2003) possibly reflecting the role of semantic processing, autobiographical recall, etc., to place information in context. In all, neuroimaging

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research suggests that ToM engages numerous processes for attributing mental states, perspective taking and contextualization mediated by a neural network including ventromedial, dorsolateral, and orbital frontal lobes, the temporo-parietal junction and the temporal poles.

It might be assumed that a similar functional imaging approach would advance understanding of ToM abilities in TBI. Such studies have been undertaken (Newsome et al., 2010; Schmitz et al., 2006; Schroeter, Ettrich, Menz, & Zysset, 2010) but their validity is questionable. They reveal a complex picture of impaired processes and compensatory activation that is difficult to unravel. Structural imaging in TBI, arguably, provides a clearer picture of brain-behavior relations. Shamay-Tsoory and colleagues have conducted several such studies, excluding patients with diffuse axonal injuries. They reported that, consistent with normal imaging, ToM deficits are especially severe following ventromedial lesions, although are also seen with extensive dorsolateral frontal pathology (Shamay-Tsoory & Aharon-Peretz, 2007; Shamay-Tsoory, Aharon-Peretz, & Perry, 2009; Shamay-Tsoory et al., 2005). The extent to which these findings generalize, however, is limited by the exclusion of diffuse pathology, given its prevalence in severe TBI.

Finally, an interesting issue from both a conceptual and clinical perspective, is the extent to which deficits in cognitive *versus* emotional empathy occur independently. Dissociations have been reported after TBI (de Sousa et al., 2010; Eslinger, Satish, & Grattan, 1996) and neuroanatomically, there is argument for both overlap and potential dissociation. Both cognitive and affective empathy appear to recruit ventromedial frontal systems (Shamay-Tsoory et al., 2004). Within the medial prefrontal cortex, cognitive processing of self and others appears to differentially engage ventral and dorsal regions while emotional resonance and empathy seems to rely upon the anterior cingulate and insula (Shamay-Tsoory, 2011) in combination with the amygdala (Carr et al., 2003; Phillips, 2003) and the mirror neuron system in the inferior frontal gyrus (Nummenmaa, Hirvonen, Parkkola, & Hietanen, 2008; Shamay-Tsoory et al., 2009).

CONCLUSION

Current theorizing suggests that specialized, overlapping neural systems mediate core emotional processes and ToM judgments, sharing reciprocal functionality with both perceptual and regulatory mechanisms. The propensity for neuropathology following TBI to compromise the ventromedial frontal lobes highlights the likelihood of problems in one or more aspects of social cognition in this population. Diffuse axonal injury, also prevalent in TBI, will further disrupt critical connections in circuits underpinning social cognition. Characterization of ensuing deficits in social cognition is, however, far from simple. Most tasks designed to tax social cognition engage perceptual, language, memory and executive abilities. The challenge for researchers in social cognition in TBI is to ensure that all tasks adequately control for these more general impairments. This review has focused upon those with severe injuries. Little is known about the impact of mild–moderate injuries on social cognition, nor about the pattern of recovery post-injury. Two studies that have examined recovery over 12 months point to stable deficits in both emotion and ToM (Ietswaart et al., 2008; Milders et al., 2008) in the context of increasing behavioral problems suggesting a complex relationship. Another issue is the increasing salience of blast injuries that lead to differing patterns of neuropathology (Nakagawa et al., 2011) increasing the heterogeneity within this population and calling into question the generalizability of research that has focused primarily upon those with acceleration–deceleration injuries.

With these caveats in mind, the rapidly growing field of social neuroscience provides a fruitful avenue for researching other facets of social cognition following TBI. For, example, neuroimaging studies suggest that metacognition and self-awareness are related to the capacity to make ToM judgments. Loss of insight regarding cognitive abilities is common following TBI as is impaired ToM. The relation between the two is yet to be explored.

There are also other phenomena within the umbrella of social cognition that are yet to be examined in detail. Stereotypical social knowledge (regarding gender, race, attractiveness, etc.) is thought to arise from gradual implicit learning of relationships that have emotional significance, that subsequently guide social intuition and social behavior (Lieberman, 2000). Automatic social cognitions are mediated by the same frontal-amygdala systems as already discussed. For example, judgments concerning physical attractiveness (Kampe, Frith, Dolan, & Frith, 2001; O'Doherty et al., 2003) and sexual orientation (Ishai, 2007) activate the medial prefrontal cortex while "trustworthiness" based on facial characteristics (Adolphs, Tranel, & Damasio, 1998; Winston, Strange, O'Doherty, & Dolan, 2002) is mediated by the amygdala. These automatic stereotypes provide the basis for initial, habitual responses to social phenomena that are regulated by more effortful executive control (Satpute & Lieberman, 2006). TBI may disrupt the influence of social stereotypes by either loss of access (Milne & Grafman, 2001) or dysregulation (Barker, Andrade, & Romanowski, 2004; Gozzi, Raymont, Solomon, Koenigs, & Grafman, 2009; McDonald, Saad, & James, 2011) but there is a need for further research in this field.

Moral reasoning, or the ability to follow ethical and accepted rules and norms (Blair & Cipolotti, 2000) is another area of relevance to TBI. Failures of moral reasoning occur in people with fronto-temporal dementia (Mendez, Anderson, & Shapira, 2005) and focal ventromedial damage (Koenigs et al., 2007) possibly due to deficiencies in emotional responsiveness when confronted with moral dilemmas. For example, most people balk at deciding to push a stranger off a footbridge in front of an oncoming trolley to save five people on the main track whereas those with frontal damage are less reluctant. Research into this area with people with TBI is yet to be developed.

From a clinical perspective, standard neuropsychological assessment is unlikely to provide a clear overview of

Table 1. Some of the more common measures of social cognition used to examine impairment following TBI and examples of studies that have cited these measures

	Name of instrument	Studies that report data on TBI using this instrument
Emotion perception	Ekman & Friesen Faces including Facial Expression of Emotion: Stimuli and Tests (Young, Perret, Calder, Sprengelmeyer, & Ekman, 2002)	(Croker & McDonald, 2005; Henry, Phillips, Crawford, Ietswaart, et al., 2006; Ietswaart et al., 2008; Milders et al., 2003, 2008).
	Florida Affect Battery (Bowers, Bauer, & Heilman, 1993; Bowers, Blonder, & Heilman, 1991)	(Ietswaart et al., 2008; Milders et al., 2003; Milders et al., 2008)
	The Awareness of Social Inference Test (McDonald, Flanagan, et al., 2011)	(Knox & Douglas, 2009; McDonald & Flanagan, 2004; McDonald et al., 2004; McDonald et al., 2003; McDonald & Saunders, 2005)
Theory of Mind	Reading the Mind in the Eyes – Revised (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001)	(Havet-Thomassin et al., 2006; Henry, Phillips, Crawford, Ietswaart, et al., 2006; Milders et al., 2003; Muller et al., 2010)
	Faux Pas Recognition Test (Stone, Baron-Cohen, & Knight, 1998)	(Milders et al., 2003, 2006, 2008, 2010)
	Cartoon task (Happè, Brownell, & Winner, 1999)	(Bibby & McDonald, 2005; Milders et al., 2006, 2008)
	Character Intentions Test (Sarfati, Hardy-Bayle, Becsche, & Widlocher, 1997)	(Havet-Thomassin, et al., 2006; Muller et al., 2010)
	The Awareness of Social Inference Test (McDonald, Flanagan, et al., 2011)	(McDonald & Flanagan, 2004; McDonald et al., 2004, 2003)
Empathy	Balanced Emotional Empathy Scale (Mehrabian, 2000)	(de Sousa, et al., 2012, 2010, 2011; Williams & Wood, 2010; Wood & Williams, 2008)
	Interpersonal Reactivity Index (Davis, 1980, 1983)	(de Sousa et al., 2010; Muller et al., 2010)

difficulties in social perception. Whether problems arise from modular deficits in social reasoning or as a result of more generic cognitive impairments is not strictly relevant. What is important is that tests used are able to predict interpersonal problems. Furthermore, it will be important for TBI research that there is a standardization to the assessment of social cognition, as has been recommended for neuropsychological testing more broadly (Wilde et al., 2010). The realm of social cognition is very recent. Although several tests have been used repeatedly with this population (see Table 1) this cannot be taken to suggest they are the most sensitive or predictive of functional deficits.

Text based stories encompassing the need to understand sarcasm (Channon & Crawford, 2010) or Faux Pas (Stone et al., 1998) have proven sensitive to TBI but, to date, have not been found to predict behavioral problems according to relatives (Milders et al., 2003, 2008). On the other hand, emotion perception based on photos does predict those who are likely to misinterpret the mood of others (Hornak et al., 1996) and those who relatives rate as having poor pragmatic communication (Milders et al., 2008; Watts & Douglas, 2006) and low social integration (Knox & Douglas, 2009).

Video vignettes with follow-up probes such as The Awareness of Social Inference Test (TASIT) (McDonald, Flanagan, & Rollins, 2011), the Video Social Inference Test (VSIT) (Turkstra, 2008) and the Assessment of Social Context (ASC) (Hynes et al., 2011) provide a better approximation of real life encounters although only TASIT has substantial norms. TASIT and ASC are predictive of everyday social behavior as rated by relatives (Hynes et al., 2011; McDonald et al., Submitted) and independent observers (McDonald, Flanagan, Martin, & Saunders, 2004). In general, further test development is required before social cognition assessment is locked into particular assessment frameworks.

The delineation of social cognition deficits following TBI highlights not only the need for specific assessments in this area but also remediation. While remediation research after TBI is a large and growing literature, there is a relative dearth of research into remediation for social cognition. On PsycBITE (www.PsycBITE.com), the comprehensive database of treatment studies, as of May 2012 there were 906 treatment studies listed that provide evidence for treatment of psychologically based disorders after TBI. Of these, only 14 bear any clear relation to treatments for social cognition or social communication. Selecting the few randomized control trials from this group, treatment of disorders of emotion perception has yielded modest benefits (Bornhofen & McDonald, 2007, 2008), as has broader treatment approaches focused upon interpersonal communication and social skills (Dahlberg et al., 2007; Helffenstein & Wechsler, 1982; McDonald et al., 2008).

The heterogeneity of TBI and its variable impact upon social cognition is a major stumbling block for group treatment studies. Single case experimental studies broaden the scope for assessing treatment effects in unusual or rare conditions. This along with growing sophistication of social cognition research should provide new avenues for designing treatment. For example, if emotional processing and responsivity are mediated by a relatively automatic, ventral system that is regulated by a dorsolateral frontal system (Phillips et al., 2003) a deficit in the automatic system (for example,

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a loss of arousal to emotionally salient material) may be ameliorated by strategies that engage the dorsal regulatory system. Preliminary research suggests this may be the case. Low physiological arousal to negative faces seems to normalise if people with TBI are given explicit instructions to attend to the images (McDonald, Rushby, et al., 2011). Deliberate mimicry of emotional expressions to improve engagement and recognition of emotions is another ploy that has theoretical potential, although in the one study to date to examine this (McDonald, Bornhofen, & Hunt, 2009), the results were not promising. More recent research suggests that the subjective and physiological effects of mimicry itself may be impaired in TBI (Dethier, et al., in press). By examining these effects in detail, further insights may come to light as to how best remediate and/or manage deficits in social cognition following TBI, so as to tackle one of the core areas of impairment and disability in this population.

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