

THE UNIVERSITY OF HULL

The Relationship between Social Cognition and  
Behavioural Difficulties in Acquired Brain Injury

being a Thesis submitted for the Degree of Doctorate in  
Clinical Psychology in the University of Hull

by

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This portfolio is dedicated to the loving memory of George Wood a grandfather like no other.

# Overview

The portfolio has three parts:

Part one is a systematic literature review, in which the theoretical, conceptual and empirical literature relating to experiencing emotional empathy after acquired brain injury is explored. Studies investigating the ability to experience emotional empathy following acquired brain injury, using either self-report or physiological measures, are reviewed and critically evaluated.

Part two is an empirical paper, which explores the impact of acquired brain injury (ABI) on social cognition, specifically empathy and theory of mind (ToM), and behavioural difficulties, specifically aggression. The study aimed to determine whether deficits in empathy and/or ToM components are able to explain heightened levels of aggression post-ABI. To do so, an ABI group displaying low levels of aggression ( $N=16$ ) were compared against an ABI group with high levels of aggression ( $N=19$ ) on measures of social cognition. Comparative analysis of the results revealed no significant difference between the groups on measures of the components of ToM and empathy. It is therefore concluded that a deficit in the components of ToM and/or empathy are unable to explain aggressive behaviour post-ABI.

Part three comprises the appendices.

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## **Part One**

# **Experiencing Emotional Empathy after Acquired Brain Injury: A Systematic Literature Review into the Nature and Extent of Deficits**

This paper is written in the format ready for submission to the journal 'Health Psychology Review'. Please see Appendix B for the author guidelines.

Word count: 12,802

## **Abstract**

A reduction in appropriate social functioning has been commonly reported following Acquired brain injury (ABI). A post-ABI empathy deficit has been suggested as a possible cause of this; specifically the ability to experience emotional empathy which has been defined as vicariously feeling what someone else is feeling. This review sought to investigate the nature and extent of emotional empathy deficits post-ABI. A systematic search of four databases yielded 10 articles that met inclusion criteria. Specific data was extracted from each article and a methodological quality score was awarded in accordance with a quality checklist. The review revealed that studies used either self-report or physiological readings as measures of experienced emotional empathy. The overarching finding was that experienced emotional empathy deficits are common post-ABI, specifically the ability to experience emotional empathy from negative emotional expressions. The measures being used to assess the experience of emotional empathy were critically appraised and their limitations used to critically assess the studies results. The strengths and limitations of literature reviewed, measures used, neurological findings and the review itself are critically analysed and possible future research discussed.

Key words: 'EMOTIONAL EMPATHY', 'EMOTIONAL RESPONSIVITY', 'BRAIN INJURY', 'REVIEW'



## **1. Introduction**

The term ‘acquired brain injury’ (ABI) refers to a non-developmental brain injury. ABI’s can be traumatic, infectious, haemorrhagic, anoxic and vascular. Due to advances in life saving technologies (Lux, 2007), cases of individuals living with an ABI have risen over the last decade (British Society of Rehabilitation Medicine, 2003; Neurological Alliance, 2003; Yates, Williams, Harris, Round & Jenkins, 2006). ABI is associated with physical, cognitive, behavioural and psychosocial problems (Fleminger, 2005). It is estimated that 135,000 individuals in the UK are experiencing long term problems as a result of an ABI (Neurological Alliance, 2003).

Over the last decade research demonstrating social cognitive deficits in individuals with ABI has emerged (Bibby & McDonald, 2005; Henry, Phillips, Crawford, Ietwaart & Summers, 2006; Stone, Baron-Cohen, Calder, Keane & Young, 2003; Wood & Williams, 2008; Williams & Wood, 2010). Manchester, Hodgkinson and Casey (1997) identified sexual disinhibition, aggression, agitation, lethargy and apathy as just some of the problematic behaviours associated with ABI. It has been postulated that deficits in social cognition result in behavioural disorders (Lezak, 1995; Stuss & Levine, 2002; Lee, Farrow, Spence & Woodruff, 2004; Bach & David, 2006).

Empathy is a social cognitive process that allows humans to share and understand feelings (Schulte-Ruther, Markowitsch, Fink and Piefke, 2007), and plays a fundamental role in social functioning (Decety, 2010; Rankin, Kramer & Miller, 2005; Dimberg Andreasson & Thunberg, 2011). Empathy is defined as having an emotional and cognitive component (Shamay-Tsoory, 2011; Kaukiainen et al, 1999; Shamay-Tsoory, Tomer, Goldsher, Berger & Aharon-Peretz, 2004; Davis, 1994; Beven, O’Brien-Malone & Hall, 2004) alongside a compassionate component (Wood & Williams, 2008) encompassed by emotional empathy (Baron-Cohen & Wheelwright,

2004). Emotional empathy, the process of ‘feeling what another person is feeling’ (Wood & Williams, 2008), has been reported to be impaired in ABI populations (Wood & Williams, 2008, Shamay-Tsoory, et al, 2004, de Sousa, McDonald, Rushby, Li, Dimoska & James, 2010a), however research is seemingly scarce. It has been proposed that a deficit of emotional empathy results in behavioural disturbances such as aggression (Wood & Williams, 2008; Kaukiainen et al 1999; Beven, O’Brien-Malone & Hall, 2004; Price, Gardner & Erickson, 2004; Blair, 2001).

To articulate whether behavioural deficits post-ABI are a consequence of emotional empathy is beyond the scope of this review. Through a systematic methodology the present review aims to investigate and critically appraise the literature assessing emotional empathy deficits post-ABI. This will assess the nature and extent of an experienced emotional empathy deficit post-ABI, the neuro-anatomical structures indicated in said deficit, the means of measuring emotional empathy and a comparison of the findings with a theoretical perspective of the emotional empathy process.

### 1.1 The differentiation between cognitive and emotional empathy.

Empathy is defined as having an emotional and cognitive component (Shamay-Tsoory, 2011; Kaukiainen et al, 1999; Shamay-Tsoory, et al 2004; Davis, 1994; Beven, O’Brien-Malone & Hall, 2004). Whereas cognitive empathy is the process of ‘understanding what someone is feeling’, emotional empathy is the process of ‘feeling what someone is feeling’ (Wood & Williams, 2008). Cognitive empathy shares commonality with theory of mind processes, allowing us to understand what others may think, feel, or intend based on social context (Stone et al, 2003; Schulte-Ruther, Markowitsch, Fink & Pieflke, 2007; Singer, 2006). Conversely, emotional empathy can be described as the experience of a feeling produced from the observation of social and

emotionally salient stimuli (this term will be used throughout the review and refers to stimuli that socially communicate the emotional state of others i.e. a facial expression). Emotional empathy is the sum of emotional contagion and emotional recognition, whereas cognitive empathy involves perspective taking and mentalising (Shamay-Tsoory, Aharon-Peretz & Perry, 2009). Emotional and cognitive empathy have been found to depend upon two dissociated neural pathways (Shamay-Tsoory et al, 2009; Shamay-Tsoory, 2011), supporting the separation of the two processes.

## 1.2 The process of emotional empathy

Emotional empathy is the ability to perceive and experience emotions produced by social stimuli (Dimberg et al, 2011). It allows humans to experience and therefore react to the emotional expressions of others (de Sousa et al 2010a).

The phenomenon of experiencing emotion has developed from a primitive reflex system, motivating creatures to move towards desired appetitive stimuli and withdraw from dangerous or painful stimuli (Bradley & Lang, 2000; Nummenmaa, Hirvonen, Parkkola & Hietanen, 2008). In humans, this motive system has developed into a complex mantra of affective experiences; identifiable through behaviour, emotional language and physiological reactions (Bradley & Lang, 2000).

Phillips, Drevets, Rauch and Lane (2003) propose a three part process of emotion perception that occurs when presented with an emotionally salient stimulus (this term will be used throughout the review and refers to a ‘stimulus that would motivate us through emotion’ i.e. a snake). The first phase is the appraisal of the affective salience of the stimuli. The second phase is the production of an affective mental state. This occurs through the activation of specific autonomic, neuroendocrine and somatomotor responses as well as conscious emotional ‘feeling’. The final phase of the process is the

regulation of the induced affective state and the conscious decision to act on or suppress it. This model is similar to Levanthal's (1984) perceptual motor model of emotion and Ohman's (1993) theory on levels of processing emotional information (for a review see Sonnyby-Borgstrom, Jonssoon & Svensson, 2003) in that it suggests the experience of a physiological arousal state provides the base for establishment of a conscious affective state. It is also similar to Preston and de Waal's (2002) perception-action hypothesis in that it states humans must experience an emotion in order to consciously recognise it in another.

Similar to the experience of emotional perception, individuals high in emotional empathy experience the emotion being presented by another. Of course, it is not always beneficial to respond by mirroring the emotion presented to us (Decety, 2010). This can be accounted for through the third phase of the Phillip et al (2003) emotion perception model in which a conscious decision can be made regarding the feeling. For example when seeing an individual in distress, this conscious action may override or suppress the emotion experienced in phase two (i.e. comforting the distressed individual). A conscious realisation may also alter the initial appraisal and encourage the suppression of the experienced emotion (i.e. the individual in distress is a child who does not want to continue shopping). Both of these scenarios would result in the alteration of our emotional state, but they would occur after the initial contagion of the emotion. Therefore the third phase in this model of emotional perception may reflect the cognitive elements of empathy and the way in which they shape our emotions dependent on social context. However, this perspective does not account for the necessity to understand that the experienced feelings belong to another.

Differentiation between self and other's emotional state is not necessary for emotional perception but is a key component of social cognitive processes such as

empathy (Schulte-Ruther, et al, 2007). Processes of theory of mind mediate the differentiation between self and other mental states. Furthermore, neural areas attributed with higher cognitive function are necessary for differentiation of self and other perspectives (Schulte-Ruther et al, 2007; Zaki, Bolger & Ochsner, 2009). Considering the perspective taking and mentalising role of cognitive empathy (Shamay-Tsoory, 2011), it is realistic to assume that differentiation between self and others emotional states is a modicum of cognitive empathy.

Taking these perspectives into consideration it is possible to predict how the separate processes of emotional and cognitive empathy may interact to produce empathy (see fig. 1).

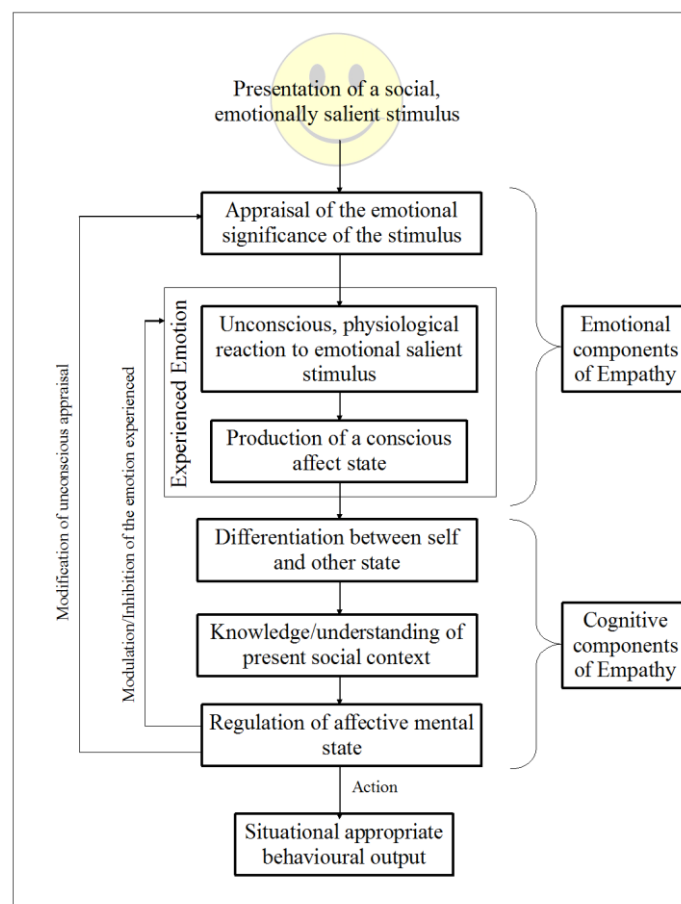


Fig. 1 A diagrammatic model of emotional and cognitive empathy

This psychological model indicates that experiencing the emotional state of another aids our ability to understand and react to it. This would represent the collaborative

divide of emotional empathy and cognitive empathy, the former preceding and being revised (halted/altered) by the latter. Phylogenetically this makes sense as emotional empathy is an earlier system that is present in rodents and birds, than cognitive empathy which presents in chimpanzees (Shamay-Tsoory et al, 2009; de Waal, 2008; Shamay-Tsoory, 2011). The dissociation between these systems suggests that it would also be possible to reach an understanding of another's affective state through an in-depth understanding of social situations without experiencing the observed emotion. Evidence for this is provided in the psychopathy literature, a population known for their intact cognitive empathy and lack of emotional empathy (Kiehl, 2006; Tangney & Stuewig, 2004). Such individuals are able to understand but not experience the affective state of others. The collaboration of both emotional and cognitive empathy would allow a rapid and accurate response to social, emotionally salient stimuli, which would be difficult for either component to achieve individually.

This model therefore represents a bottom-up and top-down process, accounting for the perspectives of simulation theory; a view that proposes humans internally simulate the mind set of another to understand them (Davies & Stone, 1995), and 'theory' theory which is a perspective suggesting humans understand the behaviour of others through sets of schematic laws and rules, equating to a framework of explanatory concepts (Churchland, 1990; Churchland, 1991).

### 1.3 Emotion appraisal post-ABI

As outlined in figure 1, emotional empathy is made up of two parts, namely the experience of the emotion preceded by the appraisal of the stimulus. Post-ABI, individuals are suggested to display a deficit in the ability to accurately recognise emotions in; faces (Kucharska-Pietura, Phillips, Gernand & David, 2003; Adolphs &

Tranel, 2003; Knox & Douglas, 2009; Adolphs, Tranel & Damasio 2001; Yip, Leung, Li & Lee, 2004; Braun, Traue, Frisch, Deighton & Kessler, 2005; Green, Turner & Thompson, 2004); music (Gosselin et al, 2005); and prosody (Pell, 1998; Pell, 2006; Charbonneau, Scherzer, Aspirot & Cohen, 2003). Although this seems to suggest a deficit in the appraisal stage of emotional empathy, studies have commonly relied upon participants selecting an emotional state or verbally stating the emotion observed. This process of labeling the observed emotion is a function of cognitive labelling (Tyson, 1998). Therefore, the inability to accurately label the emotion observed may not suggest an emotional empathy deficit but rather a cognitive deficit in the ability to label emotions. It is possible that an inability to accurately appraise emotions would lead to an inability to experience the observed emotion through emotional empathy, although this cannot be determined from the current literature. The ability to accurately appraise emotions post-ABI is outside the scope of this review and therefore literature investigating the appraisal of emotion will not be included.

#### 1.4 Experienced emotional empathy: Neural anatomy and mirror neurons.

In a recent review, Shamay-Tsoory (2011) outlined the neural basis of emotional empathy. This consists of the inferior frontal gyrus (IFG), inferior parietal lobule (IPL), anterior cingulate cortex (ACC) and the anterior insula (AI) (For a review of the neural basis of emotional and cognitive empathy, see Shamay-Tsoory, 2011). The former two regions are associated with emotional contagion whilst the latter two are associated with shared pain. These interlinked processes produce experienced emotion from social stimuli (depicted in fig. 1) through the activation of neural systems pertaining to the experience of the emotion observed (Carr, Liacoboni, Dubeau, Mazziotta & Lenzi, 2003; Jackson, Meltzoff, Decety, 2005), i.e. the presentation of sad/happy faces

evoking the feeling in the viewer (Wild, Erb, Bartels, 2001). Additionally, the right thalamus, extrastriate body area and fusiform gyrus have also been implicated in emotional empathy (Nummenmaa et al, 2008).

Emotional contagion and pain sharing are processes by which we are able to experience what another is feeling, and therefore vital in the process of emotional empathy (Nummenmaa, et al 2008; Shamay-Tsoory, 2011; Shamay-Tsoory et al 2009; Sonnby-Borgstrom et al, 2003). The rapid processing between observing the stimuli and experiencing the emotion (within 500ms) (Lishner, Cooter & Zald, 2008), indicate the importance of rapid social processing in human survival (Singer, Seymour, O'Doherty, Kaube, Dolan & Frith, 2004). Sonnby-Borgstrom (2002) suggests that facial mimicry is an automatic response presenting as an early component of emotional empathy, as those high in emotional empathy display fast, accurate mimicry when compared to low emotional empathisers. Those high in emotional empathy displayed greater sensitivity to emotional content and more intense experienced emotions resulting from emotional contagion (Dimberg et al, 2011).

The mirror neuron system (MNS) consists of neurons that fire when observing an action and performing the same action. This MNS system has been suggested to be the cornerstone of emotional contagion (Schulte-Ruther et al, 2007; Frith & Singer, 2008; Nummenmaa, et al, 2008). Sonnby-Borgstrom et al (2003) reported the observation of expressed emotion produced facial muscle imitation and evoked the expressed emotion in the viewer. The activation of premotor areas during observation of emotional expressions further supports this (Carr et al, 2003). Studies have demonstrated that the observation of pain in others activates neural regions involved in self-pain processing, suggesting the involvement of mirror neurons (Jackson et al, 2005; Singer et al, 2004). Furthermore, individuals scoring higher in emotional empathy display stronger



activation in the ACC and left AI when they perceive their partner as being in pain (Singer et al, 2004). Emotional empathy produced increased activity in areas responsible for emotional processing, perceiving faces and bodies and simulating others actions (Nummenmaa et al, 2008). All of this suggests the important role of emotional contagion and mirror neurons in the experience of emotional empathy. However, though there are strong theoretical links for the role of the MNS in empathy processing, there is currently insufficient evidence to clarify its role (for a review see Decety, 2011).

Therefore, damage to the MNS or other areas associated with emotional empathy may result in social and behavioural disorders, as individuals are unable to experience the emotions displayed by others and therefore unable to relate to them.

### 1.5 Measuring emotional empathy

Researchers measuring emotional empathy have predominantly relied upon self-report questionnaires (Wood & Williams, 2008; Jolliffe & Farrington, 2006; Mehrabian, 2000; Singer et al 2004; Macaskill, Maltby & Day, 2002; Shamay-Tsoory, et al 2004). Commonly used questionnaires include, the Balanced Emotional Empathy Scale (BEES, Mehrabian, 2000), the Questionnaire Measure of Emotional Empathy (QMEE, Mehrabian & Epstein, 1972) and the Interpersonal Reactivity Index (IRI, Davis, 1980). All of these measures have individual and shared flaws regarding their ability to measure emotional empathy. Jolliffe and Farrington (2006) suggest the QMEE and IRI rely on questions that have greater relation with sympathetic reactions rather than emotional empathy. Sympathy is considered separate from empathy, referring to the ability to reflect on how one feels about another's emotional state (Jolliffe & Farrington, 2006). Therefore if these measures are assessing sympathy, they can be

considered to be assessing the emotion experienced after cognitive regulation (see fig 1) and not before (emotional empathy). The BEES appears to do this less, with more questions focusing on the shared emotional experience, although this cannot be said for all the questions. e.g. “I get a strong urge to help when I see someone in distress”. A shared limitation of these measures is their dependence on self-report. Empathy is an abstract modality that cannot be easily assessed ‘on-line’. However the prosocial nature of empathy may lead people to over-report their empathic nature. This proves a greater problem within ABI populations who are often reported to lack insight (Bach & David, 2006). Whilst a reliable measure of emotional empathy should assess the second part of experienced emotion (production of a mirrored affective state (fig, 1.)) it is possible that measures may assess experienced emotion after cognitive regulation or receive inaccurate responses from participants.

An alternative way of measuring experienced emotion is through measures assessing the first part of experienced emotion, physiological emotional responsivity (de Sousa, McDonald, Rushby, Li, Dimoska & James, 2010b). Evidence suggests individuals displaying high levels of emotional empathy display a greater mimicking response to emotional faces suggesting a greater sensitivity to emotional content, a.k.a. the ability to experience emotions (Dimberg et al, 2011). Whether this represents a motor response which in turn produces the emotional state in the observer, or is the result of feeling the expressed emotion, it is suggested that it is an indication of socially induced experienced emotion (Lishner et al, 2008). Further evidence for this process extends from research into the human startle response. The human startle response is dependent on the affective state of the recipient; a pleasant states reduces startle response, whilst negative states increase startle response (Bradley Cuthbert & Lang, 1990). Individuals with temporal lobe damage have been found to display a lack of startle response despite

viewing stimuli designed to induce an affective state of fear or disgust (Buchanan, Tranel & Adolphs, 2004).

Although facial mimicry studies (Hess & Blair, 2000, Dimberg & Thunberg, 1998) and startle response studies are common (Bradley, Cuthbert & Lang, 1990), there are multiple physiological responses that precede conscious affective states e.g. electrodermal skin conductance (Andersson & Finset, 1998), heart rate (Sanchez-Navarro, Martinez-Selva & Roman, 2005), etc. However, whilst these measures are able to ascertain a physiological reaction, the ability to which they are able to identify the experience of a shared emotion is less obvious.

In order to measure experienced emotional empathy, researchers rely on measures of somatic responsivity (assessing physiological reactions) and verbal self-reports (assessing induced conscious affective mental states). Both have strengths and weaknesses in their ability to accurately assess the emotional empathy experienced by participants.

### 1.6 The present review

The present review aims to identify whether individual's post-ABI are able to experience emotional empathy to the same degree as non-brain injured individuals. The measures used, and their accuracy in assessing this phenomenon of emotional empathy will be critically reviewed. The neuro-anatomical areas implicated within the literature will be extracted and compared against those currently highlighted as important in the experience of emotional empathy. The quality of each article reviewed will be assessed and all data presented in a data synthesis table. The review focuses on experienced emotional empathy and therefore research investigating recognition of emotion will not be included.

## 2. Method

Using the model outlined in figure 1, included studies were required to have assessed the ability to experience emotional empathy, either the affective mental state or the physiological, emotional responsivity in a brain injured population. For the latter, only studies using social, emotionally salient stimuli (‘stimuli that socially communicate the emotional state of others’) not just emotionally salient stimuli (‘stimulus that would motivate us through emotion’) were used. The core difference between the two is that the former, whilst pertaining to the same rules as the latter, is produced from another person, i.e a social source.

### 2.1 Data Sources and Search Strategy

A systematic search of four computerised data bases, CINAHL, PsychInfo, Science Direct, MEDLINE, was undertaken. Due to the high hit ratio of unrelated articles the Science Direct search was limited to keywords, abstracts and titles only.

The following keywords were used to obtain studies investigating experienced emotional empathy:

- emotion\* reactivity
- social cogniti\*
- compassion\*
- sympathy
- emotion\* percept\*
- emotion\* recogni\*
- emotion\* experience\*
- emotiona\*contagion
- empath\*

The following keywords were used to obtain studies using brain injured participants:

- head injur\*
- brain injur\*
- brain damage\*
- stroke
- hypoxi\*
- anoxi\*
- tumo#r
- lesion\*

Using a Boolean search method all keywords encapsulating emotional empathy and acquired brain injury were combined via 'AND'. An additional search was conducted replacing the emotional empathy words that contained the word 'emotion' with the word 'affect' and the word 'affective'. This was completed using an 'OR' command e.g. affect responsivity OR affective responsivity. To avoid hit repetition the exclusion criteria of "NOT emotion\*" was added to each of these searches.

Titles and abstracts were reviewed online and full texts of potentially eligible articles were obtained. The reference sections of eligible articles were searched for potentially relevant articles and the titles, abstracts and full texts (when appropriate) of these articles were reviewed. Also, authors with an interest in the field were contacted to establish whether they had any relevant articles 'in press'. These correspondences can be found in appendix D.

## 2.2 Study selection criteria

Included studies were required to meet the following criteria:

- Study participants must include individuals who have sustained an acquired brain injury. Studies investigating neurological degenerative disorders such as dementia and Huntington’s disease will not be included.
- If using self reports the studies must be measuring the ability to experience emotional empathy. Studies assessing the ability of individuals to experience emotion generally will not be included.
- If using physiological recordings the studies must use social emotionally salient stimuli. Studies using emotionally salient stimuli will not be considered for this review.
- The study must use participants over 16 years of age.
- The study must have been published or ‘in press’ in a peer reviewed journal. Dissertations, posters and unpublished articles will not be considered.
- The study must have been published within the last 10 years (post 2000) to ensure the use of current measures of experienced emotional empathy.
- The study must be available in English.

### 2.3 Study quality assessment

A systematic qualitative approach was undertaken to assess the quality of each study. This was deemed important to control for inadequacies (i.e. study design, conduct or analysis) which may result in biases (Centre for Reviews and Dissemination, CRD 2009). Checklists that had been previously used to review the quality of articles were examined. Khan, ter Riet, Popay, Nixon and Kleijnen (2001) recommend the use of a study design hierarchy (table 1) to establish the effectiveness of studies. To ensure the accuracy of results, only studies meeting the criteria for level one and two were included.

Table 1: Study Design Hierarchy (Khan et al, 2001)

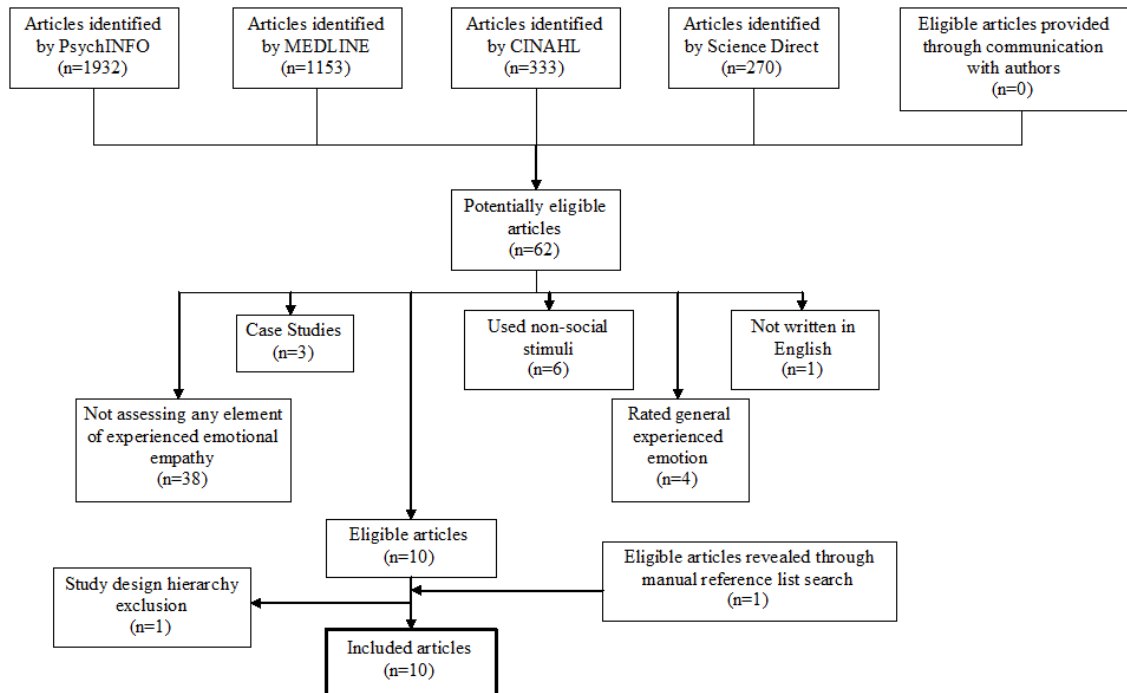
<b>Level</b>	<b>Description</b>
1.	Experimental studies (e.g. RCT with concealed allocation)
2.	Quasi-experimental studies (e.g. experimental study without randomisation)
3a.	Controlled observational studies - Cohort studies
3b.	Controlled observational studies - Case control studies
4.	Observational studies without control groups
5.	Expert opinion based on pathophysiology, bench research or consensus.

In addition, the theoretical and methodological orientations of each study were assessed using a checklist designed against the specifications of CRD (2009). The checklist was developed through the review of the Downs and Black (1998) quality assurance checklist and the Consolidation Standards of Reporting Trials (CONSORT, Schulz, Altman & Moher, 2010) statement. Neither was suitable for the current review as the former was designed to assess the quality of interventions studies and the latter randomised control trial studies. Appropriate questions from each were removed and edited to develop the 22 item, quality assurance checklist used in this study (See appendix F).

Each paper was awarded a score, the maximum being 23. Scores can be found in appendix G. All papers were peer reviewed using the same quality checklist. Inter-rater agreement was measured using a Cohen's Kappa. It was found to be 0.62 which according to Landis and Koch (1997) suggests 'substantial agreement'.

## 2.5 Data extraction

Figure 2 A flowchart depicting the process of article selection.



The online review of 3,688 titles and abstracts revealed 62 potentially eligible articles (accounting for the removal of duplicates). Contact with authors interested in this area of study revealed one eligible article that had already been identified through the literature search. No other eligible articles were revealed. The full text of these 62 articles was assessed against the inclusion criteria. 38 articles were excluded as they did not assess any element of experienced emotional empathy. A further 3 articles were removed as they were case studies. 6 articles were excluded as the stimuli used did not pertain to social interaction. 4 studies were removed as they asked participants to rate experienced emotion unrelated to social stimulation. 1 article was excluded as it was not in English. Finally, 1 article was excluded as it fell below the quality criteria set out by the study design hierarchy (Khan, et al 2001), due to it lacking a comparison group. The reference section of the 9 remaining articles was reviewed for relevant articles. Of these 7 were identified. On review of the full text only 1 was relevant.



Included articles were reviewed using a data extraction sheet (appendix E) developed with reference to examples provided by the CRD (Khan, ter Riet, Glanville, Sowden & Kleijnen, 2001). The form was edited to make it appropriate for the current review.

## 2.6 Data Synthesis

Quantitative analysis of the articles was not possible due to the methodological variation between studies concerning measures used, sample size, etc. Because of this a qualitative analysis using data synthesis was undertaken.

## **3. Results**

### 3.1 Critical Appraisal

A systematic literature review of four electronic databases revealed 10 articles matching the study criteria. The methodological quality of each study ranged from 11-20 with a mean of 17.2 (see appendix G). The scale was out of 23, suggesting each had flaws.

The information extracted from the included studies can be found in the data synthesis tables below. Furthermore, descriptions of all the measures used can be found in appendix H and appendix I.

Table 2: Studies Investigating the Ability to Experience Emotional Empathy Post-ABI

Reference and country	Characteristic ABI group	Characteristic control group	Exclusion criteria	Aim of Study	Study design and quality	Measures used	Neurological areas implicated by this study	Methodological procedures	Main Findings in relation to Experienced Emotional Empathy	Other Findings
de Sousa et al 2010(a) (Australia)	<ul style="list-style-type: none"> <li>• N =21</li> <li>• ABI Type = TBI</li> <li>• Mean age (S.D.) =48.4(8.8)</li> <li>• Male:Female 17:4</li> <li>• Mean PTA (S.D.) =80.1(70.9)</li> <li>• Mean GCS (S.D.) =N/A</li> <li>• Years Post Injury (S.D.) =11.9(7.8)</li> <li>• Age at Injury (S.D.) =N/A</li> <li>• Years in Education (S.D.) = 12.9(3.8)</li> <li>• Recruitment Setting = Brain Injury Unit's (Sydney).</li> </ul>	<ul style="list-style-type: none"> <li>• N =22</li> <li>• Mean age (S.D.) =36.1(12.6)</li> <li>• Male:Female 14:8</li> <li>• Years in Education (S.D.) = 14(3.4)</li> <li>• Matched as closely to the demographics of the TBI group as possible on gender, and education.</li> <li>• However control group significantly younger.</li> </ul> <p>(N.B. same control used in de Sousa et al 2010b)</p>	<p><u>ABI Group Only</u></p> <ul style="list-style-type: none"> <li>• Brain injury other than TBI</li> <li>• PTA&lt;1 day</li> <li>• Years since injury&lt;8 months</li> <li>• Agnosia</li> <li>• Aphasia</li> <li>• Unable to comprehend and/or adhere to instructions</li> <li>• Pre-TBI neurological impairments</li> </ul> <p><u>Control Group Only</u></p> <ul style="list-style-type: none"> <li>• A neurological disorder or brain injury.</li> </ul>	To confirm the lack of emotional empathy post-TBI. Examine the relationship between emotional empathy and emotional responsivity	Quasi-experimental Quality = 19	<p><u>Experienced Emotional Empathy</u></p> <ul style="list-style-type: none"> <li>• BEES</li> <li>• facial EMG</li> <li>• SCR</li> </ul> <p><u>Other Measures</u></p> <ul style="list-style-type: none"> <li>• DASS-21</li> </ul>	N/A	<ul style="list-style-type: none"> <li>• Viewed social, emotionally salient images (Facial expressions from Ekman &amp; Friesen, 1976)</li> <li>• Face's were either happy or angry</li> <li>• Stimuli presented for 6000ms each with a 1500ms break</li> <li>• Facial EMG and skin conductance was recorded</li> <li>• The BEES was administered after physiological testing.</li> </ul>	<p>The TBI group displayed significantly lower emotional empathy scores on the BEES.</p> <p>The TBI group displayed less reactivity in their skin conductance levels when angry faces were displayed</p> <p>High emotional empathy correlated with high responsivity.</p> <p>TBI participants showed an impaired mimicry response to angry faces compared against controls, but not happy faces. This suggests a deficit in the ability to experience the negative emotional states of others.</p> <p>Impaired responsivity to angry faces is only established amongst TBI participants low in emotional empathy</p>	

Reference and country	Characteristic ABI group	Characteristic control group	Exclusion criteria	Aim of Study	Study design and quality	Measures used	Neurological areas implicated by this study	Methodological procedures	Main Findings in relation to Experienced Emotional Empathy	Other Findings
Wood & Williams 2008 (UK)	<ul style="list-style-type: none"> <li>• N =89</li> <li>• ABI Type = TBI</li> <li>• Mean age (S.D.) =42.3(11.8)</li> <li>• Male:Female 59:30</li> <li>• Mean PTA (S.D.) =13.99(29)</li> <li>• Mean GCS (S.D.) =10.28(4.44)</li> <li>• Years Post Injury (S.D.) =3.72(3.81)</li> <li>• Age at Injury (S.D.) =38.7(12.05)</li> <li>• Years in Education (S.D.) = 11.72(2)</li> <li>• Recruitment Setting = Head injury Clinic (Swansea)</li> <li>• Premorbid IQ (S.D.) =96.6(13.5)</li> </ul>	<ul style="list-style-type: none"> <li>• N =84</li> <li>• Mean age (S.D.) =40.29(11.92)</li> <li>• Male:Female 51:33</li> <li>• Years in Education (S.D.) = N/A</li> <li>• Premorbid IQ (S.D.) =99.6(8.9)</li> <li>• Matched to TBI group on gender, age, socio-economic status and estimated intellectual level.</li> </ul>	<p><u>Both Groups</u></p> <ul style="list-style-type: none"> <li>• A history of psychiatric input, personality disorders, learning disabilities.</li> </ul> <p><u>ABI Group Only</u></p> <ul style="list-style-type: none"> <li>• Brain injury other than TBI</li> <li>• Age&lt;22</li> <li>• Dysphasia</li> <li>• Capacity to participate in the study</li> <li>• Pre-TBI neurological impairments</li> <li>• Neurological impairments that would prevent study completion</li> </ul> <p><u>Control Group Only</u></p> <ul style="list-style-type: none"> <li>• A neurological disorder or brain injury.</li> </ul>	To investigate the frequency of low emotional empathy in a TBI population.	Quasi-experimental  Quality = 20	<p><u>Experienced Emotional Empathy</u></p> <ul style="list-style-type: none"> <li>• BEES</li> </ul> <p><u>Other Measures</u></p> <ul style="list-style-type: none"> <li>• NART-2</li> <li>• WAIS-III (Vocabulary, Similarities, Comprehension, Block Design, Matrix, Letter-Number Sequencing, Picture Arrangement)</li> <li>• BDI</li> <li>• BAI</li> <li>• BADS (Zoo Map)</li> <li>• Hayling and Brixton</li> </ul>	N/A	<ul style="list-style-type: none"> <li>• Cognitive testing was administered as part of a routine neuropsychology battery.</li> <li>• Mood measures and the BEES administered after neuropsychological battery.</li> <li>• Using z-scores from the BEES, both groups were separated into high, average and low aggression categories</li> </ul>	<p>The TBI group displayed significantly lower emotional empathy scores on the BEES.</p> <p>Males displayed lower emotional empathy scores in both groups.</p>	<p>No cognitive difference across emotional empathy groups, suggesting verbal and cognitive flexibility are not a factor in emotional empathy.</p> <p>Severity of injury did not correlate with emotional empathy.</p> <p>No relationship between emotional empathy and anxiety or depression.</p>

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Muller et al 2010 (France)	<ul style="list-style-type: none"> <li>• N =15</li> <li>• ABI Type = TBI (CHI)</li> <li>• Mean age (S.D.) =37.2(12.3)</li> <li>• Male:Female 13:2</li> <li>• Mean PTA (S.D.) =N/A</li> <li>• Mean GCS (S.D.) =4.8(1.7)</li> <li>• Months Post Injury (S.D.) =102.9(121.2)</li> <li>• Age at Injury (S.D.) =N/A</li> <li>• Years in Education (S.D.) = 10.4(2)</li> <li>• Recruitment Setting = Neuro rehabilitation Sites, Nursing homes and Hospital wards.</li> </ul>	<ul style="list-style-type: none"> <li>• N =15</li> <li>• Mean age (S.D.) =37(12.5)</li> <li>• Male:Female 13:2</li> <li>• Years in Education (S.D.) = 11(1.7)</li> <li>• Matched to TBI group on gender, age, education.</li> </ul>	<p><u>Both Groups</u></p> <ul style="list-style-type: none"> <li>• A history of psychiatric input.</li> </ul> <p><u>ABI Group Only</u></p> <ul style="list-style-type: none"> <li>• Brain injury other than TBI</li> <li>• TBI other than closed head injury.</li> <li>• Pre-TBI neurological impairments</li> </ul> <p><u>Control Group Only</u></p> <ul style="list-style-type: none"> <li>• A neurological disorder or brain injury.</li> </ul>	Ability of TBI to infer the mental states of others via ToM. To assess the correlation of ToM and empathy.	Quasi-experimental  Quality = 15	<p><u>Experienced Emotional Empathy</u></p> <ul style="list-style-type: none"> <li>• IRI – PD, EC</li> </ul> <p><u>Other Measures</u></p> <ul style="list-style-type: none"> <li>• IRI – PT, FS</li> <li>• WAIS-R</li> <li>• Stroop colour word test</li> <li>• Trail making task</li> <li>• Semantic and formal lexical evocation adapted</li> <li>• California verbal learning test</li> <li>• Interpretation of indirect speech</li> <li>• Faux pas test</li> <li>• First-order and second-order false belief task</li> <li>• Character intention task</li> <li>• Reading the mind in the eyes</li> </ul>	N/A	<ul style="list-style-type: none"> <li>• All measures were administered in random order.</li> </ul>	No difference in emotional empathy between groups. No correlation between EE and ToM	<p>ToM deficit in the TBI population apart from first-order false belief.</p> <p>Indirect speech was significantly lower than controls.</p> <p>No difference between groups in Cognitive empathy</p> <p>No correlation between cognitive empathy and ToM</p>

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McDonald et al 2011(a) (Australia)	<ul style="list-style-type: none"> <li>• N =14</li> <li>• ABI Type = TBI</li> <li>• Mean age =49</li> <li>• Male:Female 11:3</li> <li>• Mean PTA (S.D.) =84.3(49.1)</li> <li>• Mean GCS (S.D.) =N/A</li> <li>• Years Post Injury (S.D.) =13(7)</li> <li>• Age at Injury (S.D.) =N/A</li> <li>• Years in Education (S.D.) = 13.4(3.7)</li> <li>• Recruitment Setting = Brain injury units (Sydney)</li> </ul> <p>Participants also used in a related study by de Sousa et al 2010a)</p>	<ul style="list-style-type: none"> <li>• N =18</li> <li>• Mean age (S.D.) =N/A</li> <li>• Male:Female 12:6</li> <li>• Years in Education (S.D.) = 13.9(3)</li> <li>• Matched to TBI group on gender and education.</li> </ul>	<p><u>Both Groups</u></p> <ul style="list-style-type: none"> <li>• A history of developmental disorder, communication deficit or psychiatric input.</li> <li>• Severe anxiety or depression</li> </ul> <p><u>ABI Group Only</u></p> <ul style="list-style-type: none"> <li>• Brain injury other than TBI</li> <li>• Agnosia</li> <li>• Aphasia</li> <li>• Psychosis</li> <li>• Unable to comprehend and/or adhere to instructions</li> <li>• Pre-TBI neurological impairments</li> </ul> <p><u>Control Group Only</u></p> <ul style="list-style-type: none"> <li>• A neurological disorder or brain injury.</li> </ul>	To replicate and extend previous work examining physiological responses in people with TBI when viewing repeated emotional expressions	Quasi-experimental  Quality = 19	<p><u>Experienced Emotional Empathy</u></p> <ul style="list-style-type: none"> <li>• SCR</li> <li>• SCL</li> <li>• ECD</li> </ul> <p><u>Other Measures</u></p> <ul style="list-style-type: none"> <li>• DASS-21</li> </ul>	N/A	<p><u>Passive Paradigm</u></p> <ul style="list-style-type: none"> <li>• Viewed social, emotionally salient images (Facial expressions from Ekman &amp; Friesen, 1976)</li> <li>• Face's were either happy or angry</li> <li>• Stimuli presented for 6000ms each with a 1500ms break</li> <li>• ECD and skin conductance was recorded</li> </ul> <p><u>Active Paradigm</u></p> <ul style="list-style-type: none"> <li>• Same as the passive paradigm</li> <li>• But participants to select the emotion expressed from a list of 7 emotions.</li> </ul>	<p>Severe TBI differentially impaired in physiological response.</p> <p>No group difference on skin conductance levels, regardless of emotion or paradigm.</p> <p>No group difference in evoked cardiac response regardless of emotion or paradigm, when groups were adjusted for age differences</p> <p>The TBI group showed lower longstanding arousal (SCL) to angry faces. in the passive paradigm.</p>	<p>TBI group displayed poorer recognition.</p> <p>The TBI group had a lower baseline arousal</p> <p>Emotional responsivity does not predict emotional recognition</p>

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Shamay-Tsoory et al 2004 (Israel)	<ul style="list-style-type: none"> <li>• N=10</li> <li>• Area Injured = Prefrontal cortex</li> <li>• Hemisphere injured(n) =Left(10); Right(9); Bilateral(17).</li> <li>• ABI Type(n) = TBI(26), Meningioma removal (6), CVA(4).</li> <li>• Mean age =35.44(13)</li> <li>• Male:Female 30:6</li> <li>• Mean PTA (S.D.) =N/A</li> <li>• Mean GCS (S.D.) =N/A</li> <li>• Years Post Injury (S.D.) =N/A</li> <li>• Age at Injury (S.D.) =N/A</li> <li>• Years in Education (S.D.) = 12.52(1.7)</li> <li>• Recruitment Setting = Cognitive Neurology Unit</li> </ul>	<ul style="list-style-type: none"> <li>• N=10</li> <li>• Area Injured = Parietal cortex</li> <li>• Hemisphere injured(n) =Left(8); Right(7); Bilateral(15).</li> <li>• ABI Type(n) = TBI(3); Meningioma removal (5); CVA(7).</li> <li>• Mean age =41.6(16.07)</li> <li>• Male:Female 10:5</li> <li>• Mean PTA (S.D.) =N/A</li> <li>• Mean GCS (S.D.) =N/A</li> <li>• Years Post Injury (S.D.) =N/A</li> <li>• Age at Injury (S.D.) =N/A</li> <li>• Years in Education (S.D.) =13.47(2.3)</li> <li>• Recruitment Setting = Cognitive Neurology Unit</li> </ul>	<ul style="list-style-type: none"> <li>• N=19</li> <li>• Mean age (S.D.) =34.05(15.81)</li> <li>• Male:Female 15:4</li> <li>• Years in Education (S.D.) = 13.78(3.1)</li> <li>• Matched to TBI group on age</li> </ul>	<p><u>Both Groups</u></p> <ul style="list-style-type: none"> <li>• Not speak fluent Hebrew</li> <li>• A history of psychiatric illness, developmental disorders, drug or alcohol abuse.</li> </ul> <p><u>ABI Group Only</u></p> <ul style="list-style-type: none"> <li>• Diffuse axonal injuries</li> <li>• Time post trauma/surgery &lt;6 months</li> <li>• Verbal, visual or motor deficits that would prevent completion of testing</li> <li>• Pre-injury/tumour history of head trauma with loss of consciousness or other neurological deficits</li> </ul> <p><u>Control Group Only</u></p> <ul style="list-style-type: none"> <li>• A neurological disorder or brain injury.</li> </ul>	<p>To examine the effect of localised lesions on various aspects of empathy</p> <p>To examine the impact of right and left hemisphere lesions on empathy processes.</p> <p>To assess whether empathy related to cognitive flexibility or emotional recognition.</p>	<p>Quasi-experimental</p> <p>Quality = 11</p>	<p><u>Experienced Emotional Empathy</u></p> <ul style="list-style-type: none"> <li>• QMEE</li> </ul> <p><u>Other Measures</u></p> <ul style="list-style-type: none"> <li>• IRI - PT, FS.</li> <li>• Raven's Progressive Matrices</li> <li>• BDI</li> <li>• WCST</li> <li>• Verbal Fluency</li> <li>• Design Fluency</li> <li>• Alternative Uses Test</li> <li>• Torrance test of creative thinking (The circles subscale)</li> <li>• Recognition of Facial Expression</li> <li>• Recognition of affective prosody</li> </ul>	<ul style="list-style-type: none"> <li>• Right parietal cortex</li> <li>• right and left Prefrontal cortex</li> <li>• The orbito-prefrontal and medial-prefrontal cortex (regarding empathy as whole)</li> </ul>	N/A	<p>Prefrontal cortex damage impairs emotional empathy (as well as cognitive empathy).</p> <p>Prefrontal damage and right parietal cortex damage impairs emotional empathy.</p> <p>Emotional empathy does not correlate with any other measures</p> <p>Emotional empathy and cognitive empathy correlate.</p>	<p>Prefrontal cortex group impaired on cognitive flexibility and affective and facial expression recognition.</p> <p>Both ABI groups are impaired on affective prosody.</p> <p>Right hemisphere damage participants displayed greater deficit in emotional prosody and expression recognition compared to left</p>

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McDonald et al 2011(b) (Australia)	<ul style="list-style-type: none"> <li>• N =21</li> <li>• ABI Type = TBI</li> <li>• Mean age =48.4(8.8)</li> <li>• Male:Female 17:4</li> <li>• Mean PTA (S.D.) =80.1(70.9)</li> <li>• Mean GCS (S.D.) =N/A</li> <li>• Years Post Injury (S.D.) =11.9(7.8)</li> <li>• Age at Injury (S.D.) =N/A</li> <li>• Years in Education (S.D.) = 12.9(3.8)</li> <li>• Recruitment Setting = Brain injury units (Sydney)</li> </ul>	<ul style="list-style-type: none"> <li>• N =20</li> <li>• Mean age (S.D.) =36.2(13.2)</li> <li>• Male:Female 12:8</li> <li>• Years in Education (S.D.) = 14.6(3.7)</li> <li>• Matched to TBI group on gender and education.</li> </ul>	<p><u>Both Groups</u></p> <ul style="list-style-type: none"> <li>• Clinical levels of depression or anxiety, psychosis</li> <li>• inability to communicate</li> </ul> <p><u>ABI Group Only</u></p> <ul style="list-style-type: none"> <li>• Brain injury other than TBI</li> <li>• Not judged to be experiencing social problems</li> <li>• Agnosia</li> <li>• Aphasia</li> <li>• Psychiatric history</li> <li>• History of neurological disorder</li> <li>• Unable to comprehend and/or adhere to instructions</li> <li>• Pre-TBI neurological impairments</li> </ul> <p><u>Control Group Only</u></p> <ul style="list-style-type: none"> <li>• A neurological disorder or brain injury.</li> </ul>	Examine facial mimicry of happy and sad faces by people with TBI	Quasi-experimental  Quality = 18	<p><u>Experienced Emotional Empathy</u></p> <ul style="list-style-type: none"> <li>• Facial EMG</li> </ul> <p><u>Other Measures</u></p> <ul style="list-style-type: none"> <li>• DASS-21</li> <li>• Emotion Matching</li> </ul>	N/A	<p><u>Static Paradigm</u></p> <ul style="list-style-type: none"> <li>• Viewed social, emotionally salient images (Facial expressions from Ekman &amp; Friesen, 1976)</li> <li>• Face's were either happy or angry</li> <li>• Stimuli presented for 6000ms each with a 1500ms break</li> <li>• Facial EMG was recorded</li> </ul> <p><u>Dynamic Paradigm</u></p> <ul style="list-style-type: none"> <li>• Same as the passive paradigm</li> <li>• Stimuli created using 8 photos of the same actors used in the static images.</li> <li>• Faces morph from neutral to either happy or angry on a continuum.</li> </ul> <p><u>Emotion matching task</u></p> <ul style="list-style-type: none"> <li>• Match stimulus face with one of four expression pictures (using different actors of the same gender).</li> </ul>	<p>TBI individuals displayed limited corrugator supercilii response to angry, static faces, compared with controls. This was only present in the early (500ms-1000ms) stimulus exposure period.</p> <p>No difference was found between groups for happy faces. Although TBI did display a "muted" response, though not significant.</p> <p>No group difference was displayed between groups for dynamic images</p>	<p>The group differences are not simply a loss of motor simulation as only anger mimicry was impaired.</p> <p>Emotional recognition was impaired in TBI. However, control performed at ceiling.</p> <p>The TBI group were more impaired on the recognition of negative affective expressions.</p>

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Williams & Wood 2010 (UK)	<ul style="list-style-type: none"> <li>• N =64</li> <li>• ABI Type = TBI</li> <li>• Mean age =35.84(13.33)</li> <li>• Male:Female 53:11</li> <li>• Mean PTA (S.D.) =16.85(27.84)</li> <li>• Mean GCS (S.D.) =9.30(4.46)</li> <li>• Years Post Injury (S.D.) =3.19(2.58)</li> <li>• Age at Injury (S.D.) =32.77(13.32)</li> <li>• Years in Education (S.D.) =2.14(2.18)</li> <li>• Recruitment Setting = referred between 2007-2008 to Swansea University Brain Injury Clinic</li> </ul>	<ul style="list-style-type: none"> <li>• N =64</li> <li>• Mean age (S.D.) =36.09(14.24)</li> <li>• Male:Female 53:11</li> <li>• Years in Education (S.D.) = 12.98(2.775)</li> <li>• Matched to TBI group on gender, age, employment, education and marital status.</li> </ul>	<p><u>Both Groups</u></p> <ul style="list-style-type: none"> <li>• Age&lt;20</li> <li>• Lacking capacity to consent to participate</li> <li>• A history of psychiatric/ personality issues</li> <li>• Learning disability</li> <li>• Dysphasia</li> </ul> <p><u>ABI Group Only</u></p> <ul style="list-style-type: none"> <li>• Brain injury other than TBI</li> <li>• Pre-TBI neurological impairments</li> </ul> <p><u>Control Group Only</u></p> <ul style="list-style-type: none"> <li>• A neurological disorder or brain injury.</li> </ul>	<p>Confirm alexthymia and low emotional empathy present post TBI.</p> <p>To investigate the link between alexthymia and emotional empathy.</p>	<p>Quasi-experimental</p> <p>Quality = 18</p>	<p><u>Experienced Emotional Empathy</u></p> <ul style="list-style-type: none"> <li>• BEES</li> </ul> <p><u>Other Measures</u></p> <ul style="list-style-type: none"> <li>• TAS-20</li> <li>• WAIS-III (at least one subtest measuring each of the following: verbal ability; working memory; cognitive flexibility)</li> </ul>	N/A	<ul style="list-style-type: none"> <li>• All measures were administered as part of a standard neurological screen.</li> </ul>	<p>64.4% of the TBI group displayed low emotional empathy, significantly higher than the 34.4% of the control group with low emotional empathy.</p> <p>Cognitive abilities were unable to explain variance in groups for BEES scores.</p> <p>The TAS-20 was able to explain BEES variance within groups, suggesting a link between emotional empathy and alexithymia.</p> <p>Negative correlation between emotional empathy and alexithymia.</p>	<p>Severity of injury and time since were unable to explain difference in emotional empathy and alexithymia scores.</p> <p>A higher proportion of the TBI displayed higher alexthymia scores (60.9%) than the control group (10.9%)</p>



Reference and country	Characteristic ABI groups			Characteristic control group	Exclusion criteria	Aim of Study	Study design and quality	Measures used	Neurological areas implicated by this study	Methodological procedures	Main Findings in relation to Experienced Emotional Empathy	Other Findings
Shamay-Tsoory et al 2009 (Israel)	<ul style="list-style-type: none"> <li>• N=11</li> <li>• Area Injured = Ventromedial prefrontal (VM)</li> <li>• ABI Type(n) = TBI(8), Meningioma (2), Stroke(1).</li> <li>• Mean age =36.45(16.2)</li> <li>• Male:Female 9:2</li> <li>• Mean PTA (S.D.) =N/A</li> <li>• Mean GCS (S.D.) =N/A</li> <li>• Years Post Injury (S.D.) =9.36(11.85)</li> <li>• Age at Injury (S.D.) =N/A</li> <li>• Years in Education (S.D.) = 11.7(1.41)</li> <li>• Recruitment Setting = N/A</li> <li>• Matched to rest of ABI groups on lesion size</li> </ul>	<ul style="list-style-type: none"> <li>• N=8</li> <li>• Area Injured = Inferior Frontal Gyrus (IFG)</li> <li>• ABI Type(n) = TBI(6); Meningioma (2); Stroke(0).</li> <li>• Mean age =32.75(15.06)</li> <li>• Male:Female 8:0</li> <li>• Mean PTA (S.D.) =N/A</li> <li>• Mean GCS (S.D.) =N/A</li> <li>• Years Post Injury (S.D.) =7.25(6.94)</li> <li>• Age at Injury (S.D.) =N/A</li> <li>• Years in Education (S.D.) =14.12(2.58)</li> <li>• Recruitment Setting = N/A</li> <li>• Matched to rest of ABI groups on lesion size</li> </ul>	<ul style="list-style-type: none"> <li>• N=11</li> <li>• Area Injured = Posterior Cortex (PC)</li> <li>• ABI Type(n) = TBI(6); Meningioma (3); Stroke(2).</li> <li>• Mean age =38(14.49)</li> <li>• Male:Female 7:4</li> <li>• Mean PTA (S.D.) =N/A</li> <li>• Mean GCS (S.D.) =N/A</li> <li>• Years Post Injury (S.D.) =7.27(5.38)</li> <li>• Age at Injury (S.D.) =N/A</li> <li>• Years in Education (S.D.) =13.36(1.74)</li> <li>• Recruitment Setting = N/A</li> <li>• Matched to rest of ABI groups on lesion size</li> </ul>	<ul style="list-style-type: none"> <li>• N=34</li> <li>• Mean age (S.D.) =N/A</li> <li>• Male:Female N/A</li> <li>• Years in Education (S.D.) = N/A</li> <li>• Matched to ABI groups N/A</li> </ul>	<p><u>Both Groups</u> N/A</p> <p><u>ABI Group Only</u></p> <ul style="list-style-type: none"> <li>• Tumour participants &lt;1 year post surgery</li> <li>• TBI and stroke participants not in chronic phase of recovery (&lt;6 months post)</li> <li>• No post operative imaging or behavioural data</li> </ul>	To establish the neural substrates pertaining to emotional and cognitive empathy. To investigate the relationship between the two.	Quasi-experimental  Quality = 17	<p><u>Experienced Emotional Empathy</u></p> <ul style="list-style-type: none"> <li>• IRI – EC, PD</li> </ul> <p><u>Other Measures</u></p> <ul style="list-style-type: none"> <li>• IRI - PT, FS.</li> <li>• Raven’s Progressive Matrices</li> <li>• BDI</li> <li>• WCST</li> <li>• Verbal Fluency</li> <li>• WAIS-R (Similarities and Digit Span)</li> <li>• Second order false belief task</li> <li>• Emotional Recognition Task</li> </ul>	<ul style="list-style-type: none"> <li>• IFG</li> <li>• Superior Temporal Sulcus (STS)</li> <li>• Brodmanns Area (BA) 44</li> </ul>	<ul style="list-style-type: none"> <li>• Measures administered in random order</li> </ul>	<p>IFG group displayed greater deficit in emotional empathy than controls and PC group. The VM group was approaching significant difference (p=0.054)</p> <p>Emotional Empathy correlates with emotional recognition. However this was only on the PD sub-scale of the IRI.</p> <p>The implication BA44 in emotional empathy suggests the necessity of mirror neurons in the emotional empathy process</p>	<p>VM displayed impairment in cognitive empathy and ToM</p> <p>The IFG group were impaired in emotional recognition</p> <p>Cognitive empathy correlates with ToM</p> <p>STS damage displayed impaired ToM</p>

Reference and country	Characteristic ABI group	Characteristic control group	Exclusion criteria	Aim of Study	Study design and quality	Measures used	Neurological areas implicated by this study	Methodological procedures	Main Findings in relation to Experienced Emotional Empathy	Other Findings
Hopkins, Dywan & Segalowitz 2002 (Canada)	<ul style="list-style-type: none"> <li>• N =15</li> <li>• ABI Type = Closed head injury ((CHI) TBI)</li> <li>• Mean age =29.4(5.78)</li> <li>• Male:Female 12:3</li> <li>• Mean PTA (S.D.) =N/A</li> <li>• Mean days in coma =46(45)</li> <li>• Mean GCS (S.D.) =N/A</li> <li>• Years Post Injury (S.D.) =9.6(5.29)</li> <li>• Age at Injury (S.D.) =32.77(13.32)</li> <li>• Years in Education (S.D.) =13.4(3.07)</li> <li>• Recruitment Setting = supported independent settings</li> </ul>	<ul style="list-style-type: none"> <li>• N =15</li> <li>• Mean age (S.D.) =29.9(6.10)</li> <li>• Male:Female 12:3</li> <li>• Years in Education (S.D.) = 14.1(2.5)</li> <li>• Matched to TBI group on gender, education and age.</li> </ul>	<p><u>Both Groups</u></p> <ul style="list-style-type: none"> <li>• Any condition affecting central nervous system functioning</li> </ul> <p><u>ABI Group Only</u></p> <ul style="list-style-type: none"> <li>• Brain injury other than CHI TBI</li> <li>• Not reached plateau after lengthy rehab period</li> <li>• &lt;moderate injury severity.</li> </ul> <p><u>Control Group Only</u></p> <ul style="list-style-type: none"> <li>• No neurological disorder or brain injury.</li> </ul>	To investigate whether expression identification deficits and arousal abnormalities are present in diffuse CHI as is the case in orbital and medial regions of the prefrontal cortex (OMPFC)	Quasi-experimental  Quality = 16	<p><u>Experienced Emotional Empathy</u></p> <ul style="list-style-type: none"> <li>• non-dominant hand EDA</li> </ul> <p><u>Other Measures</u></p> <ul style="list-style-type: none"> <li>• WAIS-R (Vocabulary subtest)</li> <li>• Culture Fair Test of non-verbal problem solving</li> <li>• BAFQ</li> <li>• Expression Identification Test</li> <li>• BRFT</li> </ul>	N/A	<p><u>Passive Paradigm</u></p> <ul style="list-style-type: none"> <li>• Viewed social, emotionally salient images (Facial expressions from Ekman &amp; Friesen, 1976)</li> <li>• Face's were either negative (fear, disgust, anger), positive (happy) or neutral</li> <li>• Stimuli presented for 2sec.</li> <li>• There was a 15-20sec.break post stimuli (to return to baseline)</li> <li>• The largest voltage response from 1-7 sec. of stimulus onset was recorded</li> </ul> <p><u>Active Paradigm</u></p> <ul style="list-style-type: none"> <li>• Same as passive</li> <li>• Participants required to comment on the stimulus observed.</li> </ul> <p>The test battery was administered after the physiological measure.</p>	<p>There was greater response to stimuli in the active paradigm (29.99kΩ) than the passive (14.16 kΩ).</p> <p>CHI group responded with EDA equivalent to control participants to positive faces, but a substantially reduced response to negative stimuli.</p> <p>EDA did not correlate with the ability to recognise emotion or perception matching.</p>	<p>The BFAQ revealed no deficit in empathy, however it is unclear whether this is a reliable/valid measure of empathy, and specifically emotional empathy.</p> <p>The CHI group displayed a reduced awareness of their difficulties.</p> <p>Compared to controls the CHI group displayed lower, intellectual functioning, flexibility, arousal and memory</p> <p>Perception deficits could only account for the accurate identification of sadness.</p>

Reference and country	Characteristic ABI group	Characteristic control group	Exclusion criteria	Aim of Study	Study design and quality	Measures used	Neurological areas implicated by this study	Methodological procedures	Main Findings in relation to Experienced Emotional Empathy	Other Findings
de Sousa et al 2010(b) (Australia)	<ul style="list-style-type: none"> <li>• N =20</li> <li>• ABI Type = TBI</li> <li>• Mean age (S.D.) =47.4(10)</li> <li>• Male:Female 15:5</li> <li>• Mean PTA (S.D.) =80.9(71.5)</li> <li>• Mean GCS (S.D.) =N/A</li> <li>• Years Post Injury (S.D.) =13.4(6.9)</li> <li>• Age at Injury (S.D.) =N/A</li> <li>• Years in Education (S.D.) = 12.5(2.9)</li> <li>• Recruitment Setting = Brain Injury Unit's (Sydney).</li> </ul>	<ul style="list-style-type: none"> <li>• N =22</li> <li>• Mean age (S.D.) =36.1(12.6)</li> <li>• Male:Female 14:8</li> <li>• Years in Education (S.D.) = 14(3.4)</li> <li>• Matched as closely to the demographics of the TBI group as possible on gender, and education. However control group significantly younger.</li> </ul> <p>(N.B. same control used in de Sousa et al 2010a)</p>	<p><u>ABI Group Only</u></p> <ul style="list-style-type: none"> <li>• Brain injury other than TBI</li> <li>• PTA&lt;1 day</li> <li>• Years since injury&lt;1 year</li> <li>• Agnosia</li> <li>• Aphasia</li> <li>• Lacking Cognitive and Motor capacity to follow instructions</li> <li>• Severe depression or anxiety</li> <li>• Pre-TBI neurological impairments</li> </ul> <p><u>Control Group Only</u></p> <ul style="list-style-type: none"> <li>• A neurological disorder or brain injury.</li> </ul>	To verify group empathy deficits post-TBI and investigate the impact such deficits have on emotional responsivity. To assess the relationship between empathy and emotional responsivity.	Quasi-experimental  Quality = 19	<p><u>Experienced Emotional Empathy</u></p> <ul style="list-style-type: none"> <li>• IRI-EC</li> <li>• EQ-ER</li> <li>• BEES</li> </ul> <p><u>Other Measures</u></p> <ul style="list-style-type: none"> <li>• IRI-PT</li> <li>• EQ-CE</li> <li>• DASS-21</li> <li>• facial EMG</li> <li>• SCR</li> <li>• Valence scale</li> <li>• Arousal scale</li> </ul>	N/A	<ul style="list-style-type: none"> <li>• Viewed emotionally salient images (images from the International Affective Picture System (IAPS, Centre for the Study of Emotion and Attention, 1999)</li> <li>• Stimuli presented for 6000ms each with a 1500ms break</li> <li>• Facial EMG and skin conductance was recorded</li> <li>• After each stimuli participants rated their arousal and valence</li> <li>• Questionnaires were administered afterwards. No order is stated.</li> </ul>	<p>Lower levels of emotional empathy were displayed by the TBI group (70%) compared against the control group (31.8%).</p> <p>Higher empathy correlated with higher corrugator supercilii response to emotional salient stimuli in the control group.</p>	<p>Lower Cognitive empathy in TBI group</p> <p>Rating of arousal lower in the TBI group</p> <p>TBI group displayed a lower corrugator supercilii response to negative stimuli</p> <p>There was no difference between groups on zygomaticus response to pleasant stimuli</p> <p>There was no difference in ratings of valence</p>

### 3.1.1 Participant samples

#### 3.1.1.1 Size and demographic details

The ABI sample size used in each study varied greatly, the smallest being 14 (McDonald et al, 2011a) and the largest being 89 (Wood & Williams, 2008). Two studies divided their ABI sample depending on neural damage location (Shamay-Tsoory et al, 2004; Shamay-Tsoory et al, 2009), resulting in group numbers as low as 8. Generally the control group's size exceeded the experimental group by no more than four participants and no less than one participant. The only exception to this was the study by Wood and Williams (2008) which had 89 ABI participants and 84 controls.

The average age, years in education and the gender ratio for each group was reported by the majority of studies. Three studies (Wood & Williams, 2008; Shamay-Tsoory et al, 2009; McDonald et al, 2011a) did not supply this demographic information for their control group, in fact Shamay-Tsoory et al (2009) did not supply any information for their control group. The majority of the articles supplied further demographic information of the ABI group, time post injury (in years: Shamay-Tsoory, 2009; McDonald et al, 2011b; McDonald et al, 2011a; de Sousa et al, 2010b; de Sousa et al, 2010a; in months: Muller et al, 2010), or both age at injury and time post injury (Williams & Wood, 2010; Hopkins et al 2002; Wood & Williams, 2008).

Eight studies reported a predictor of injury severity, Post traumatic amnesia (PTA, Williams & Wood, 2010; McDonald et al, 2011b; McDonald et al, 2011a; de Sousa et al, 2010b; de Sousa et al, 2010a; Wood & Williams, 2008), Glasgow coma score (GCS, Muller et al 2010; Williams & Wood, 2010; Wood & Williams, 2008) and days in coma (Hopkins et al, 2002). The most thorough description of group demographics was given by Wood and Williams (2008), providing information on gender, PTA, GCS, years post injury, age at injury, years in education, and premorbid IQ. Whilst there is no

discernable relationship between quality rating and reported demographic detail, it should be acknowledged that Wood and Williams (2008) scored the highest in the rating of study quality.

Demographic tables, outlining the demographic details, injury type, etc were provided by five studies (McDonald et al, 2011b; de Sousa et al, 2010b; Shamay-Tsoory et al, 2004; Muller et al, 2010; Shamay-Tsoory et al, 2009).

#### 3.1.1.2 Recruitment and group matching

Only one article did not provide any information regarding the recruitment of ABI participants (Shamay-Tsoory et al, 2009). It was not always clear from the articles what setting participants had been recruited from (i.e. inpatient, community based, etc.) This level of detail is important, as the type of setting from which participants are recruited can provide information about the severity of injury and the level of functioning at time of testing.

The majority of studies attempted to match control and ABI participants as closely as possible on demographic details. Shamay-Tsoory et al (2004) matched ABI participants individually with healthy controls on age. Other studies matched participants on gender age and education (Muller et al 2010; Hopkins et al 2002). Four studies used advertisements to recruit controls matched as closely as possible on demographic details (McDonald et al 2011b; de Sousa et al, 2010b; de Sousa et al, 2010a; McDonald et al 2011a). Despite this, though groups displayed no significant differences on education or gender, the control group was significantly younger in all four studies. Of these four, two ran the same statistical analyses with age adjusted groups (control participants older than 23 and TBI (Traumatic Brain Injury) participants younger than 55, de Sousa et al, 2010b; de Sousa et al, 2010a) and one included age as a covariant in

all statistical analyses (McDonald et al 2011a) to ensure group age differences had no bearing on results. McDonald et al (2011b) did not control for the statistical difference between groups suggesting that facial mimicry is as robust in old age as in young adults. Two studies expanded on age, gender and education matching; one further matching groups on employment and marital status (Williams & Wood, 2010) and the other comparing the groups on socio-economic status and estimated intellectual ability (but not education, Wood & Williams, 2008). Shamay-Tsoory et al (2009) reported no significant difference between lesion groups on lesion size but provided no information on how participants were comparable to controls, or amongst lesion groups in any other domain. However, considering the rarity of focal lesions, conducting a study in which lesion groups were matched on demographic details would be near impossible.

There was some sharing of participant samples between studies. This is most noticeable between studies led by de Sousa and McDonald. De Sousa et al (2010a) and (2010b) used the same control group and McDonald et al (2011a) reports using the same experimental group (with additional participants) as de Sousa et al (2010a). Furthermore, de Sousa et al 2010a reports using the same experimental sample as McDonald (2010b). This use of the same experimental sample can reduce the generalisability of the findings (as it may be the sample that displays a significant difference to controls and not the population). Therefore when interpreting any overlapping findings (i.e. reduced experience of emotional empathy) it must be acknowledged that both are using the same sample. This sample sharing is likely the result of both studies investigating the same population type (TBI), from the same settings (inpatient, neurorehabilitation units, Sydney, Australia), and collaboration between authors affiliated with the same university. The collaboration between the two authors is further demonstrated by the methodological quality ratings of their work,

three of which are of an equivalent standard (19) with the other scoring just one point lower (McDonald et al, 2011b).

### 3.1.1.3 Exclusion criteria

Exclusion criteria on the most basic level for all ABI groups excluded those who would not be able to complete testing due to cognitive or motor deficits, had no psychiatric history and no neurological history prior to the ABI. This was also the basic exclusion criteria for the control group with the addition of no neurological history. Two studies controlled for social immaturity (social cognitive development) by excluding participants younger than 20 (Williams & Wood, 2010) and 22 (Wood & Williams, 2008). Three studies excluded participants with ‘severe’ anxiety or depression (McDonald et al, 2011b; McDonald et al, 2011a; de Sousa et al, 2010b) as a means of controlling for mood disorders. Three studies controlled for injury severity, two using PTA of less than one day as an exclusion criteria (de Sousa et al, 2010b; de Sousa et al, 2010a) and the other describes excluding participants below moderate severity but not how this was measured (Hopkins et al, 2002). Four studies controlled for time post injury; Shamay-Tsoory et al (2009) excluded tumour participants under one year post surgery and TBI and stroke participants less than six months post trauma; Shamay-Tsoory et al (2004) excluded participants less than 6 months post surgery/trauma; de Sousa et al (2010b) excluded participants less than one year post injury; and de Sousa et al (2010a) excluded participants less than 8 months post injury. Eight studies controlled for injury type by recruiting only TBI participants (Muller et al, 2010; Williams & Wood, 2010; Wood & Williams, 2008; McDonald et al 2011b; de Sousa et al, 2010b; de Sousa et al, 2010a; McDonald et al 2011a) or specifically CHI (Closed Head Injury) TBI (Hopkins et al, 2002). Studies that did not control for injury

type, controlled for areas injured through neural lesion mapping and ensured all were focal lesions by excluding participants with diffuse axonal damage (Shamay-Tsoory et al 2004; Shamay-Tsoory et al, 2009). This level of variation between studies for exclusion criteria makes comparison of their findings difficult.

#### 3.1.1.4 ABI type

There was much homogeneity between studies regarding ABI sample injury type. Eight studies included only certain types of ABI, all of which were TBI and one study only included closed head injured TBI patients (Hopkins et al, 2002). Studies assessing the role of specific neuro-anatomical structures implicated in the process of emotional empathy included a wider variety of ABI types, including TBI, haemorrhagic and anoxic brain injuries (Shamay-Tsoory et al 2004; Shamay-Tsoory et al, 2009).

#### 3.1.2 Aims of the studies

There was a large variation in the studies aims. Two studies aimed to investigate emotional empathy after ABI (Williams & Wood, 2010; Wood & Williams, 2008), three studies aimed to investigate physiological response to emotional stimuli post-ABI (Hopkins et al, 2002; McDonald et al, 2011b; McDonald et al, 2011a) and two aimed to investigate both, as well as the relationships between these processes (de Sousa et al, 2010b; de Sousa et al, 2010a). Furthermore, two studies sought to establish the neural structures associated with a deficit of empathy (Shamay-Tsoory et al 2004; Shamay-Tsoory et al, 2009). One study did not aim to investigate experienced emotional empathy post-ABI but did wish to establish whether ToM correlated with empathy (Muller et al, 2010).



### 3.1.3 Definitions of empathy and emotional empathy

Definitions of empathy and emotional empathy were present in six of the articles. Of these, two describe emotional empathy as a process of “feeling what another person is feeling” (Shamay-Tsoory, 2009; Wood & Williams, 2008). Although this is an adequate description of emotional empathy, clearer descriptions are provided in other articles “...the ability to vicariously experience the emotions of others” (Williams & Wood, 2010) and “...to experience affective reactions to the emotional displays of others” (de Sousa et al 2010a). Vague, inaccurate descriptions of emotional empathy are provided by two articles, “...involves the actual emotional reaction” (Muller et al, 2010) and “...the ability to share emotional experiences”. The latter could be argued to pertain to an ability to feel the same as someone else for the same reason, rather than because of them. It should also be noted that Muller et al (2010) were not investigating the individual components of empathy. Of the 4 articles that did not define empathy, 3 were investigating physiological phenomenon related to the observation of facial expressions and had not proposed they were assessing a process of emotional empathy. However, de Sousa (2010b) investigated the relationship between emotional responsivity and both cognitive and emotional empathy, as such it is improper that they do not provide a definition of these neuropsychological phenomena.

## 3.2 Findings of the Review

### 3.2.1 Measures used to assess experienced emotional empathy

One aim of this review was to investigate the measures being used to assess the ability of an ABI population to experience emotional empathy. Six of the sample studies used self-report measures to assess emotional empathy, three used physiological readings to monitor the emotional reactions to social stimuli and one study used both.

Of the self report measures used one study used the QMEE (Shamay-Tsoory et al, 2004), three studies used the IRI, Personal Distress (PD) and Empathic Concern (EC) scales (Muller et al, 2010; Shamay-Tsoory, 2009) or just the EC (de Sousa et al, 2010b), one study used the Empathy Quotient-Emotional Responsivity (EQ-ER; the first time this measure has been used in this way, de Sousa et al 2010a) and four studies used the BEES (Williams & Wood, 2010; de Sousa et al, 2010b; de Sousa et al, 2010a; Wood & Williams, 2008). Only de Sousa et al (2010b) used multiple self-report measures to assess emotional empathy.

A variety of physiological measures were used, Hopkins et al (2002) used Electrodermal response (EDA) readings from participants 'volar surfaces of the distal phalanges' of their non-dominant hand to establish their skin conductance response to the presented stimuli. Two studies took electromyography (EMG) readings from two facial muscles, the corrugator supercilii (associated with negative expressions) and the zygomaticus (associated with positive expressions) (McDonald et al 2011b; de Sousa et al 2010a). De Sousa et al (2010a) also monitored skin conductance response (SCR). McDonald et al (2011a) used multiple physiological measures including SCR, skin conductance levels (SCL) and evoked cardiac deceleration (ECD). EDA and SCR both refer to skin conductance measured as electrodermal response. SCL was differentiated from the former two by McDonald et al (2010b) suggesting the SCL represents the lasting level of electrodermal response. All stimuli used in conjunction with physiological measures were facial expressions taken from Ekman and Friesen (1976). Stimuli were presented as static frames in each study. McDonald et al (2011a) also used a dynamic paradigm in which the faces went from neutral to emotionally expressive. Furthermore, two studies adopted 'active' paradigms in which participants had to comment on the stimulus (Hopkins et al, 2002), or select the emotion being expressed

by the stimulus from a list of seven emotions (McDonald et al, 2011a). This was done to produce active attendance to the stimuli. The majority (3) of studies using physiological measures used happy and angry facial stimuli as well as neutral faces. The only study to use additional negative emotions (disgust and fear) was Hopkins et al (2002).

Although de Sousa et al (2010b) did investigate physiological responsivity to emotionally salient stimuli, the stimuli used were not socially salient, in the form of a social communication (i.e. a facial expression). Therefore, the physiological readings would be assessing an emotional experience, not the experience of emotional empathy.

### 3.2.2 Neural substrates associated with experiencing emotional empathy

Only two studies distinguished groups by focal lesions (Shamay-Tsoory et al, 2004; Shamay-Tsoory et al, 2009). Therefore, the amount of information regarding the damaged neural structures underlying experienced emotional empathy is limited. Shamay-Tsoory et al (2004) implicated the right and left prefrontal cortex and the right parietal lobe in processing emotional empathy. They also suggest the role of orbito/medial-prefrontal cortex as having a significant role in processing empathy as a whole. Shamay-Tsoory et al (2009), expanded on the findings of Shamay-Tsoory et al (2004), implicating the IFG primarily in processing emotional empathy. Those impaired in emotional empathy frequently displayed damage to BA 44. The STS was also implicated in both emotional and cognitive empathy (Shamay-Tsoory et al 2009). De Sousa et al (2010a) reported that their ABI sample displayed no amygdala damage suggesting that amygdala damage is not necessary for an impairment of emotional empathy.

Ventral frontal damage is common after TBI due to the rostral-caudal gradient and acceleration-deceleration phenomenon of TBI (Lux, 2007). Four studies suggest that their results implicate the ventromedial cortex (de Sousa et al, 2010a; McDonald et al, 2011a; McDonald et al, 2011b; Williams & Wood, 2010) and one implied the nearby orbital and medial prefrontal regions (Hopkins et al 2002) in the experience of emotional empathy. However this is merely hypothetical and although suggesting similar areas to studies using participants with focal lesions, the implications cannot be certain.

### 3.2.3 Experienced emotional empathy post-ABI

All studies included in the review were considered to be assessing the ability to experience emotional empathy post-ABI. Of the studies using self-report measures, six reported that ABI group(s) displayed a significantly lower emotional empathy score than controls (Shamay-Tsoory et al, 2004; Shamay-Tsoory et al, 2009; Williams & Wood, 2010; de Sousa et al, 2010b; de Sousa et al, 2010a; Wood & Williams, 2008) and one study did not (Muller et al, 2010). Also, Wood and Williams (2008) reported a gender bias, with females displaying higher levels of emotional empathy compared with males.

Of the studies using physiological measures all four reported a reduction in physiological responses to negative, social stimuli but a preserved response to positive, social stimuli (Hopkins et al, 2002; McDonald et al, 2011b; McDonald et al, 2011a; de Sousa et al, 2010a). Therefore, it can be suggested that post-ABI, individuals have difficulty experiencing the negative emotional states of others. McDonald et al (2011b) described a “muted” response in their TBI group to happy faces, but reported that this

failed to reach significance, perhaps suggesting that ability to experience positive emotional empathy post-ABI is slightly reduced.

McDonald et al (2011b) found the group difference in physiological response to negative facial expressions was only present in the early stage of stimulus presentation (500-1000ms), not in the later stage, suggesting that the experience of emotional empathy is rapid. This supports assertions that rapid social processing is essential for human survival (Singer, Seymour, O'Doherty, Kaube, Dolan & Frith, 2004).

Interestingly, McDonald et al (2011a) found a significant difference between groups on SCL scores but not on ECD or SCR. SCL is able to indicate sustained arousal whilst SCR pertains to rapid orientation. Therefore, this finding may suggest that ABI individuals fail to maintain an emotional empathy state. Other evidence suggests that the experience of emotional empathy is rapid and impaired in ABI, using SCR (de Sousa et al 2010a) and facial EMG (McDonald et al, 2011b). All of which suggests the ability to experience emotional empathy, rapidly, or maintain it, is impaired post-ABI.

Hopkins et al (2002) reported a greater EDA response to stimuli when participants were required to comment on stimuli than when they were merely observing. This suggests the more attentive an individual is to a phenomenon, the greater the experience of emotional empathy.

Higher emotional empathy predicted greater physiological responsivity, and low physiological responsivity to negative faces was only established in brain injured participants with low emotional empathy (de Sousa et al, 2010a). Also, higher corrugator supercilii response to emotionally salient stimuli was associated with higher emotional empathy (de Sousa et al, 2010b). All of which support the notion that physiological responses to emotional social salient stimuli are a part of the emotional empathy experience.

### 3.2.4 Related findings

The relationship between emotional empathy and emotional recognition was examined by four studies. Shamay-Tsoory et al (2009) found that the IFG group that were impaired in emotional empathy were also impaired in emotional recognition. McDonald et al (2011b) also found an impairment of emotional recognition in their TBI group but acknowledged that the control performed at ceiling. Furthermore, Williams and Wood (2010) found emotional empathy displayed a negative correlation with alexithymia, a condition characterised by, amongst other things, difficulty identifying and describing emotions (see Williams & Wood, 2010 for a detailed summary). However, McDonald et al (2011a) explained that emotional responsivity did not predict emotional recognition, suggesting the relationship is not as simple as a deficit in one predicts a deficit in the other.

## **4. Discussion and Interpretation of Findings**

The purpose of the review was to investigate the nature and extent of emotional empathy deficits post-ABI whilst also critically appraising the measures used to assess such deficits, and the neurological areas implicated in the experience of emotional empathy. Emotional empathy was defined as the ability to vicariously experience the emotional states of others. In this section the results of the review will be critically discussed, beginning with the nature and extent of an emotional empathy deficit, moving onto the critical appraisal of the measures used to establish the findings, and lastly exploring the neurological areas implicated in the experience of emotional empathy. The limitations of the review and proposals for future research will then be discussed.

#### 4.1 The nature and extent of experienced emotional empathy deficits post-ABI

The primary focus of this review was to establish the nature and extent to which individuals post-ABI could experience emotional empathy, as suggested by the current literature. Of the 10 included studies nine found a deficit of emotional empathy in a brain injured sample, suggesting that a deficit in the ability to experience emotional empathy is common post-ABI. Only one study did not find an emotional empathy deficit post-ABI (Muller et al, 2010). There are several factors that may explain Muller et al (2010)'s negative results. The study displayed the second lowest methodological quality rating of all the studies using self-report measures. The sample size was relatively small (15 per group) and its protocol was quite lengthy, with participants required to complete 11 measures. Finally, it used the IRI, which has been criticised as inappropriate in measuring emotional empathy, with its items pertaining to experienced sympathy rather than emotional empathy (Jolliffe & Farrington, 2006).

Studies utilising physiological measures provide more specific information regarding the physiological mechanisms affected by this experienced emotional empathy deficit. They suggest the presentation of positive emotional expressions (happiness) evokes a physiological emotional response in the ABI sample, similar to that of controls. However, when presented with a negative emotional expression (anger, fear, disgust), the ABI population displayed impaired physiological responsivity. This suggests that the ABI population are impaired in the ability to experience emotional empathy in response to the presentation of negative emotions, but not positive. This of course does not indicate that the ABI population are unable to experience negative emotion, just that they do not experience emotional empathy from the observation of these emotional states in others. This raises the question of why there is a deficit in the ability to physiologically respond to the emotional expressions of negative emotions but not

positive emotions? There are two potentially overlapping rationales that can address this question:

The first suggests that damage to a specific area, implicated in the processing of negative emotions, but not positive, would explain this deficit (de Sousa et al 2010a). The ventrolateral frontal cortex is associated with the processing of anger, whereas the processing of happiness, fear and sadness are associated with the amygdala (de Sousa et al 2010a). Angry faces were used as negative emotional stimuli in each of the studies utilising physiological measures. Furthermore, each of these studies only used participants with TBI, which has been linked with ventral system damage (de Sousa et al 2010s). Therefore, damage to the ventrolateral frontal cortex would explain the present finding of a deficit in the ability to experience an emotional empathy response from happy facial expressions, but not angry facial expressions. However, Hopkins et al (2002)'s found a deficit in the ability to experience emotional empathy to the presentations of 'negative faces' post-ABI, and this included fear and disgust as well as anger. Considering this, the neural area implicated would have to encompass all negative emotions and as fear processing has been linked with the amygdala, alongside happiness (de Sousa et al 2010a), it is unclear how this would occur. However, as the responsivity to specific emotions was not reported, merely discussing 'negative emotional facial expressions' it is not possible to determine the extent to which each negative emotion was impaired post-ABI. Therefore, it is equally plausible that their findings were produced because of the use of angry facial stimuli and not that of fear and disgust. Future research should aim to replicate Hopkins et al (2002)'s findings, differentiating between a variety of emotional expressions.

An alternative of this 'specific neural area' hypothesis is that the emotional empathy response to positive emotional faces is also impaired post-ABI, though not as prominent



as responsivity to negative faces due to evolutionary prioritising. Essentially this perspective would suggest that negative facial stimuli demand a higher level of attention and produce a greater arousal than positive facial stimuli, and are thus noticeable when compared against controls. Negative emotional expressions are a means of communicating danger (Bradley & Lang, 2000) and therefore associated with survival dependent heightened attentional awareness. Also, humans would require a higher level of arousal to negative faces to facilitate the urgency of the fight or flight response and increase chances of survival. It can therefore be postulated that a deficit in arousal to positive stimuli may go unnoticed on physiological recordings, as control group arousal to the stimuli is generally low compared to negative stimuli. This may explain why McDonald et al (2011b) and Hopkins et al (2002) displayed a ‘muted response’ to happy facial stimuli; not impaired, but lessened. Graphical data from the majority of studies using physiological measures suggests that higher arousal was achieved in the control group for unpleasant stimuli, emphasising the ‘normal’ arousal differentiation between positive and negative stimuli (de Sousa, et al, 2011b; McDonald et al, 2011a; de Sousa et al, 2010a; Hopkins et al, 2002). Therefore, it is possible to suggest that the ability to physiologically respond to all emotional faces is impaired post-ABI, but due to negative stimuli demanding higher levels of arousal, the impairment is more prominent.

Building upon the evolutionary hypothesis discussed above, McDonald et al (2011a) found that this ABI impairment to physiologically respond to negative emotional facial stimuli disappeared when individuals were required to actively attend to the stimulus. However, one other study (Hopkins et al 2002) used an ‘active paradigm’ and found no difference between their active and passive (just observing stimuli) paradigms. It is possible that this difference in findings results from the form of ‘active’ paradigm used.

McDonald et al (2010a) required participants to name the emotion being observed, whereas Hopkins et al (2002) merely required participants to comment on the picture. It can be postulated that by requiring participants to name the emotion, McDonald et al (2010a) were encouraging participants to bypass an emotional empathy impairment via cognitive processes. The need to cognitively understand the emotion being observed is likely the cause of the physiological response associated with that emotion. This supports the work of simulation theorists, who hypothesise that to recognise the emotional state of others it must be experienced (Preston & de Waal, 2002). This therefore suggests that the ability to automatically experience emotional empathy to the presentation of facial stimuli is impaired post-ABI and as a result the heightened arousal is unachievable unless bypassed via cognitive processes. It is unclear from this whether an impairment in the initial automatic appraisal of the stimuli (see fig. 1) or the automatic ability to experience emotional empathy from the stimulus (see fig. 1) is the cause of this observable deficit. Further investigation is required before it is possible to determine the extent of this emotional empathy deficit, be it in the ability to appraise the stimuli as being socially emotionally salient or the ability to experience the presented emotion from the stimulus.

A further finding regarding the nature of experienced emotional empathy deficits post-ABI refers to the duration of an observable group difference. When comparing the physiological recording of ABI group facial EMG to that of controls, McDonald et al (2011b) established an impairment in the ability of the ABI group to experience emotional empathy in the first 500-1000ms of stimulus presentation but not over the entire 6000ms of stimulus presentation. As stated above this was only present when angry facial expressions were used. Whilst this may seem to suggest that the ABI group are not impaired, just slowed, in their experience of emotional empathy,

graphical data suggests that the control group response to emotional facial stimuli decreases over the 6000ms each stimulus was presented. Therefore, the presence of an experienced emotional empathy deficit in the early stages of stimulus presentation is not the result of slowed processing speed, but rather the presence of a physiological regulatory action, performed by the control group. This supports the notion that the experience of emotional empathy occurs rapidly and then subsides over time (Singer, Seymour, O’Doherty, Kaube, Dolan & Frith, 2004). This reduction in arousal is possibly the mediation response of cognitive empathy mitigating the emotional experience i.e. “It’s just a picture of an angry face, therefore there is no need for concern”, and thereby reducing arousal. This supports the model presented in fig. 1 as well as authors who refer to emotional processing as having a regulatory component (Sonny-Borgstrom et al, 2003; Phillip et al, 2003). The concept that cognitive empathy acts as mitigating force in the experience of emotional empathy should be the focus of future research.

#### 4.2 A critical appraisal of experienced emotional empathy measures

This review also evaluated the current measures being used to assess experienced emotional empathy within the ABI population. It is important to consider the validity of the measures used, as this will clearly impact upon the validity of the results. As the validity of each measure is to be critically appraised, it is useful to define different types of validity. ‘Concurrent validity’ refers to the correlation between measures of the same phenomenon (Rust & Golombok, 1989). This of course is a weak criterion as measures may correlate but neither may measure the proposed phenomenon. This leads onto ‘construct validity’, which refers to the ability of a test to assess the phenomena predicted by the theory, a more robust form of validity and the primary mode of

validation of psychometric measures (Rust & Golombok, 1989). Two types of measure were commonly used, self-report and physiological readings. The merits and limitations of each are explored.

#### 4.2.1 Self-report measures

The use of self-report measures was more common than physiological readings and four different measures were used, the IRI-EC/PD, the QMEE, the BEES and the EQ-ER.

The IRI was used as a measure of emotional empathy in three studies (de Sousa et al 2010b; Shamay-Tsoory et al, 2009; Muller et al, 2010). The IRI is a measure of both cognitive and emotional empathy consisting of four 7-item scales. Two of these scales, the EC and PD, are designed to measure emotional empathy. However, the construct validity of the IRI-EC has been criticised, suggesting it is measuring concern for others and experienced sympathy rather than the ability to feel what another is feeling (emotional empathy, Jolliffe & Farrington, 2006). Furthermore, the IRI-PD has been suggested to lack construct validity making it less psychometrically valid (de Sousa, 2010b). Shamay-Tsoory et al (2009, pp.620) describe the IRI-EC as measuring ‘the respondents feelings of warmth, compassion and concern for others’ whilst the IRI-PD assesses the ‘self oriented feelings of anxiety and discomfort resulting from tense interpersonal settings’. Neither of these statements is in line with the Wood and Williams (2008) ‘feels what another is feeling’ definition of emotional empathy. Therefore, the results of the studies solely using the IRI to assess emotional empathy (Shamay-Tsoory et al, 2009; Muller et al, 2010) should be interpreted with caution.

The QMEE was used by Shamay-Tsoory et al (2004) to assess emotional empathy. Similarly to the IRI, the construct validity of the QMEE’s has been questioned and it

has been suggested to incorrectly equate sympathy with empathy (Jolliffe & Farrington, 2006). Despite the scales apt description of emotional empathy ‘vicarious emotional response to the perceived emotions of others’, it fails to adhere to this, with items relating to emotional concern and sympathy (Jolliffe & Farrington, 2006). Furthermore, Shamay-Tsoory et al (2004) found that a deficit in emotional empathy as reported by the QMEE coincided with the neuroanatomical region associated with sympathy, which may suggest that the QMEE to some degree measures sympathy rather than emotional empathy. The sensitivity of the QMEE was also queried by Shamay-Tsoory et al (2004) as it was less able to demonstrate significant results than measures of cognitive empathy, perhaps suggesting a weaker validity. Therefore, like the IRI, the results of Shamay-Tsoory et al (2004), should be considered with caution as the QMEE’s ability to assess emotional empathy is questionable.

The BEES was the most commonly used self-report measure in the sample, being utilised in four studies (de Sousa et al, 2010b; de Sousa et al, 2010a; Williams & Wood, 2010; Wood & Williams, 2008). The BEES was developed from the QMEE as a broad measure of emotional empathy (Harrison, Morgan & Critchley, 2010) and is considered a reliable and valid measure of emotional empathy (Mehrabian, 2000). The BEES has been suggested to be a more reliable and valid self-report measure of emotional empathy than the IRI-EC and EQ-ER with a higher number of items and higher reliability coefficient (de Sousa et al, 2010b). Furthermore, studies using the BEES produced quality scores higher than those using other self-report measures. However, the BEES displayed concurrent validity with the IRI-EC (de Sousa et al 2010b) and the QMEE (Mehrabian, 2000), suggesting that it may be victim to similar limitations. For example some of the BEES items can be suggested to assess sympathy rather than emotional empathy (e.g. ‘I cannot feel much sorrow for those who are responsible for

their own misery’). Further investigation is warranted to establish the ability of the BEES to assess emotional empathy. Again these concerns should be considered when interpreting the results of studies using the BEES.

De Sousa et al (2010a) produced the only study using the EQ-ER and to the authors knowledge is the first to divide and use the EQ in this way. As a result, little is known about the scale apart from that reported by de Sousa et al (2010a). The EQ-ER is suggested to measure emotional reactivity and displayed a concurrent validity with the IRI-EC and the BEES, suggesting they are measuring a similar construct (de Sousa et al, 2010a). Considering the uncertainty of whether these measures are assessing sympathy or emotional empathy, the EQ-ER should also be interpreted with caution.

The construct validity of all of the self report measures used to assess emotional empathy is highly questionable as all appear to use items associated with the process of sympathy rather than emotional empathy. Sympathy is defined as a construct separate from empathy (Joliffe & Farrington, 2006) concerned chiefly with the appraisal of how one feels about another’s emotional state. As outlined in figure 1, sympathy can be considered the self-generated emotional state produced following cognitive empathy, replacing the emotional state produced by emotional empathy and motivating us to action. Joliffe and Farrington (2006) appear to agree with this appraisal, stating that in emotional empathy the experienced emotion is the same as that displayed by the target other, but in sympathy the experienced emotion may differ due to the addition of cognitive appraisal. It can therefore be suggested that the self-report measures used to assess emotional empathy post-ABI are actually assessing sympathy. If this is the case, it is not possible from the self-report results to suggest that an emotional empathy deficit is present post-ABI but that a deficit in sympathy is. A deficit in emotional empathy may be the cause of the observed sympathy deficit, however future research

using emotional empathy measures with higher construct validity will be required before the emotional empathy deficits suggested post-ABI by these studies can be confirmed.

It must be acknowledged that the adequacy of each self-report measure as an accurate measure of emotional empathy (rather than sympathy) is not distinguishable within the limits of this review. Again, further research is required to confirm the orientation of these self-report measures to the constructs of either emotional empathy or sympathy.

An additional flaw of emotional empathy self-report measures is that they require a great deal of self reflection over an abstract concept. Therefore the results on such measures, within an ABI population, may be susceptible to a lack of insight (Hart, 2003; Parson, Carpenter-Hyland, Burright & Donovanick, 1995). It remains unclear to what extent participant's post-ABI may lack insight but it will be important for future research to control for potential insight and awareness deficits that may influence have influenced the current results.

#### 4.2.2 Physiological reading measures

Four studies used physiological measures to assess experienced emotional empathy. The most common physiological measures used were facial EMG (McDonald et al 2011b; de Sousa et al, 2010a) and skin conductance (Hopkins et al, 2002; McDonald et al, 2011a; de Sousa et al, 2010a). Heart deceleration (ECD) was also used in one study (McDonald et al, 2011a). All these measures have been suggested to assess physiological responsivity to the presentation of social communications of emotion, a social, emotionally salient stimulus (i.e. a facial expression of emotion). The physiological response recorded is considered the physiological experience of

emotional empathy, the physiological experience of a shared emotional state (Preston & de Waal's, 2002). This is supported by McDonald et al (2010b) suggesting that a motor mimicry response deficit cannot account for the present findings. Should motor mimicry be impaired post-ABI it would be present in both facial mimicry of negative and positive facial expressions. However, the preserved zygomaticus response to positive facial stimuli suggests that there is an emotional simulation deficit in which the ABI-samples are unable to experience the observed emotion through emotional empathy. In this way the physiological measures could be said to have high construct validity as the processes measured pertain to the theoretical definition of emotional empathy.

There are of course difficulties in measuring the experienced emotional empathy using physiological recordings. The main issue being that it is difficult to suggest that the physiological readings correspond to the exact emotion being presented. The benefit of using facial EMG is that unlike skin conductance and ECD, it is possible to assess whether the emotion being experienced is positive or negative, due to activation of the corrugator or zygomaticus. This can then be compared against the emotional orientation of the presented stimulus for confirmation of a shared emotional experience. Skin conductance indicates an arousal has occurred but not the emotional orientation. Therefore, of the two, the facial EMG would be the better method for measuring experienced emotional empathy. However, facial EMG, may be limited by the association between corrugator supercilii activation and thinking (McDonald et al, 2011b). Therefore, the combined use of facial EMG and skin conductance would be recommended for future research investigating the ability to experience emotional empathy post ABI.



In assessing emotional empathy using physiological readings, the selection of appropriate stimuli is as important as using an appropriate physiological measure. This is because the use of social, emotionally salient stimuli (i.e. a social communication of emotion such as facial expression) will assess experienced emotional empathy whilst stimuli that are just emotionally salient (an image that would evoke an emotion within us, not originally produced by someone else i.e. a snake) would not. This difference was demonstrated by de Sousa et al (2010b) who, using emotionally salient stimuli from the IAPS, found their ABI group's facial responsivity did not correlate with its emotional empathy. Conversely, de Sousa et al (2010a) established a negative correlation between ABI groups facial EMG and their emotional empathy when using stimuli that were both social and emotionally salient (emotional facial expressions). De Sousa et al (2010b) attribute this inconsistency to the differing stimuli, with facial expressions having a greater 'intrinsic biological significance' than the IAPS images which are less social in nature. Therefore, future research should ensure that stimuli used to assess the experience of emotional empathy are social and emotionally salient.

It can be suggested that the use of physiological measures alone will never provide substantial evidence to confirm an emotional empathy deficit as they only assess the physiological mechanisms that underlie the experience of emotional empathy. According to Rust and Golombok (1989), construct validity is never complete and due to its positivist, hypothetico-deductive origins, future research using alternative methodological standpoints to assess the observable mechanisms of experienced emotional empathy is required before the concept that ABI commonly produces a experienced emotional deficit can be accepted.

#### 4.2.3 Self-report measures versus physiological readings

De Sousa et al (2011a) were the only authors to utilise both physiological readings and self-report measures when assessing experienced emotional empathy. They found a moderate, positive correlational relationship between facial EMG responsivity and scores of emotional empathy from the BEES. Whilst it is not possible to suggest from this that both are measures of experienced emotional empathy, it lends support to previous literature suggesting emotional responsivity and emotional empathy are interlinked processes (Sonny-Borgstrom et al, 2003; Preston & de Waal, 2002; Phillip et al, 2003; Nummenmaa et al, 2008; Dimberg, et al, 2011). Whilst this finding seems to suggest concurrent validity, because the correlation was moderate it is more likely that both are measuring related constructs, but not the same specific phenomenon. Due to the overlapping nature of empathy components, sympathy and physiological arousal, it is difficult to separate subtle semantic constructs such as emotional empathy. It can be assumed that the BEES and facial EMG are measuring related processes but to suggest both are assessing emotional empathy would be premature. Given the evidence presented above and the 'moderate' correlation it is likely that both are assessing a feeling state, but not necessarily that both are assessing emotional empathy.

As established above, both types of measure have strengths and weaknesses. These should be taken into consideration when interpreting the primary finding of the review, that there is a deficit in the ability to experience emotional empathy post-ABI (particularly from angry faces), and when selecting measures of emotional empathy for future research. From the strengths and weaknesses presented above, it is suggestible that physiological measures are the most appropriate choice in assessing experienced emotional empathy. This is due largely to the self-report measures construct validity appearing to be quite poor. Physiological measures are of course not able to measure

emotional empathy directly but are able to assess a mechanism of emotional empathy within the theoretical framework of emotional empathy (the ability to vicariously experience the emotional state of another). Therefore, future research will be required, assessing other mechanisms that adhere to this definition of emotional empathy, to establish an all encompassing perspective of emotional empathy post-ABI. This could potentially be done using self-report measures of emotional empathy that do not merge into the assessment of sympathy.

The limitations of the measures may weaken the validity of the findings of each study, and therefore the findings of the review. It is beyond the limits of this review to say whether self report or physiological ratings would be most appropriate in the assessment of experienced emotional empathy; however, it is currently not possible to confirm a deficit in emotional empathy post-ABI, considering the weaknesses of the current self report measures being utilised. The physiological measures indicate a deficit in the physiological responsivity associated with emotional empathy specifically responding to negative emotions via emotional empathy, but further research is required to confirm this, especially considering the different levels of arousal demanded by positive and negative stimuli.

#### 4.2.4 Neurological areas involved in the experience of emotional empathy

In exploring the nature of an experienced emotional empathy deficit post-ABI it was important to assess the neuroanatomical structures underlying such a deficit. The review revealed only two studies (Shamay-Tsoory et al, 2004; Shamay-Tsoory et al, 2009) investigating emotional empathy in focal lesion groups. As a result, what can be inferred is limited. The right parietal lobe and prefrontal cortex, and more specifically, the IFG were implicated in the processing of emotional empathy. Particularly,

individuals with damage to Brodmann's area 44 of the IFG (an area associated with the MNS) scored significantly lower on measures of emotional empathy (Shamay-Tsoory et al, 2009). Due to the implicated BA 44, Shamay-Tsoory et al (2009) argued the MNS as essential in the experience of emotional empathy. This claim supports the suggestions of authors who would argue that the ability to experience emotional empathy is dependent on the ability to simulate the emotion being presented by another in oneself (Davies & Stone, 1995; Preston & de Waal, 2002; Phillip et al, 2003; Nummenmaa et al, 2008; Dimberg, et al, 2011; Sonny-Borgstrom et al, 2003). However, as suggested by Decety (2011), further, more robust investigation into the impact of the MNS on emotional empathy is required before the role of the MNS in emotional empathy can be accepted.

The IFG and BA 44 have been implicated as key components in the process of emotional empathy in a recent review by Shamay-Tsoory (2011). Therefore, the present review was unable to expand upon the neural areas currently believed responsible for the process of emotional empathy, due to the lack of focal lesion studies investigating the ability to experience emotional empathy. Despite this the review was able to critically assess the evidence upon which the implicated neural structures was based. Both studies used self-report measures of emotional empathy, the QMEE (Shamay-Tsoory et al, 2004) and the IRI-EC/PD (Shamay-Tsoory, 2009), the construct validity of which have been questioned (see 4.2.1). Considering this, it is possible that emotional empathy may not have been assessed and therefore neuroanatomical findings may be inaccurate. Therefore, the suggestions of Shamay-Tsoory et al (2004) and Shamay-Tsoory et al (2009), that an emotional empathy deficit is observable following right parietal, prefrontal cortex, and/or IFG damage, should be considered with care.

Future research should aim to replicate the work of Shamay-Tsoory et al (2004) and Shamay-Tsoory et al (2009), using measures of emotional empathy with higher validity. At the current time the BEES appears to be the most accurate self-report measure in assessing emotional empathy. However, given the possibility that this is in parts assessing sympathy, the most valid measure to use would be facial EMG. It would be possible to use facial EMG alongside social, emotionally salient stimuli (i.e. emotional faces) to assess the ability of specific lesion groups to experience emotional empathy physiologically. Until self-report measures with higher construct validity are developed, research in this area is dependent upon physiological measures, which are of course not without their flaws (see 4.2.2).

Furthermore, as the current review only found two studies investigating emotional empathy with lesion samples, it is evident that a great deal of further research with focal lesion patients is required before any confirmation of the neuro anatomy related to an emotional empathy deficit can be made.

#### 4.2.5 Limitations

The present review is subject to several limitations. Firstly, the development of a hypothetical model (fig. 1) of emotional and cognitive empathy was not the aim of the review. Despite this, the current literature regarding the process of empathy (Preston & de Waal, 2002) and emotional perception (Phillips et al, 2003; Levanthal, 1984; Ohman, 1993) was felt to be lacking in its ability to explain the emotional empathy experience. It was therefore necessary to provide a framework within which this could be understood. The development of such a model would likely be the focus of an entire review utilising a greater amount of literature. Therefore, the presented model is not

intended as an all encompassing model for empathy, merely a framework on which future research and reviews can build upon.

Although the review aimed to investigate the impact of ABI on the experience of emotional empathy, a second limitation is that all studies reviewed used a TBI sample and only two included other types of ABI. Therefore, the review is less able to generalise the present findings to the ABI population. This trend could not be foreseen and emphasises the necessity of studies investigating the effects of other types of brain injuries on the experience of emotional empathy.

Thirdly, the inability of the study to expand on the present consensus of damaged neural structures producing emotional empathy deficits is disappointing. However, the review did highlight the lack of literature examining emotional empathy in focal lesion participants.

The review is only able to report the lack of a deficit in experiencing emotional empathy from negative expressions within a brain injured population. It is not able to establish whether an earlier stage in the emotional empathy process is the cause of the deficit, i.e. the appraisal of the negative emotional expressions as socially important/something that should be reacted to. Whilst research assessing emotional recognition post-ABI appears to be assessing the appraisal of a socially emotionally charged stimulus (fig. 1) they commonly rely upon participants choosing an emotional state. This decision making process could be described as cognitive in nature, as a function of cognitive labelling (Tyson, 1998). Therefore, the ability to produce an accurate account of the emotion presented in the stimulus would represent a deficit at any point in the process of empathy, not just in the stimulus appraisal stage.

The review is also limited by the limitations of the sum of its parts, i.e. the studies. For example, all studies using physiological measures employed Ekman and Friesen's

(1976) faces as stimuli. These greyscale faces are criticised for their extreme presentation of emotional expressions and may lack ecological validity for this reason (McDonald, et al 2011b). Furthermore, the use of self report measures on a group with ‘poor insight’ makes the findings questionable (de Sousa et al, 2010a).

#### 4.2.6 Future Research

Considering that the present literature review only yielded 10 articles, it is evident that more research investigating the ability to experience emotional empathy post ABI is needed in order to form a robust understanding of the process and methods of rehabilitation. A focus should be paid to brain injury type’s alternative to TBI. Research should also attempt to produce focal lesion studies utilising various measures of experienced emotional empathy, to provide a greater understanding to the neural anatomy implicated in clinical presentations of emotional empathy deficits.

The suggestions of Hopkins et al (2002) require further investigation. It will be useful to establish whether a deficit in the ability to experience emotional empathy in response to negative emotional expressions, or just expressions of anger, is present post-ABI.

Future studies should also aim to establish the construct validity of the presented measures as measures of experienced emotional empathy and develop alternative measures that assess mechanisms within the theoretical frame of emotional empathy, possibly utilising auditory stimuli, startle probe modulation and construct valid self-report measures.

Also, future researchers should expand on the model presented in the current review. The model should be seen as a framework incorporating empathy processing and emotional perception ideology. A literature review to identify further relevant literature

would be ideal to develop the model further. A theoretical model of empathy that incorporates its cognitive and emotional underpinnings and the underpinnings of both of these constructs is essential to aid in the progressive understanding of human social interaction.

#### 4.2.7 Conclusion and Implications

The purpose of this review was to investigate the extent to which individuals, post-ABI, could experience emotional empathy and the nature of such a deficit. The findings of the review suggest that the experience of emotional empathy is frequently impaired following ABI. Furthermore, physiological measures suggest this is related to the inability to experience the emotions produced by negative facial stimuli. The ability to experience emotional empathy from positive affective states (happiness) appears to be preserved. The IFG and BA 44 were also implicated by the review and support the perspective that the MNS is crucial for emotional empathy.

However, both self-report and physiological measures have limitations that may impact the findings of the current review. This is especially relevant regarding self-report measures, the construct validity of which is highly questionable. Therefore, the findings of the review should be considered critically. Despite this it still remains (largely through the findings of studies utilising physiological measures) that there is an abnormality in the ability to experience emotional empathy post-ABI pertaining to negative facial stimuli. Furthermore, this deficit is negligible when ABI sufferers are required to attend to the presented stimuli. Therefore, the experienced emotional empathy deficits may pertain to an inability to automatically appraise a stimulus as being socially, emotionally salient. Further investigation is required to confirm an



emotional empathy deficit post-ABI and establish whether it is the result of a specific deficit in automatic stimulus appraisal.

Further research should seek to expand on the current measures of emotional empathy ensuring they are constructed within the theoretical perspective of emotional empathy. Also future research should aim to recreate the neurological findings highlighted by the review, using more valid measures of emotional empathy. Future research should also seek to develop the field of emotional empathy post-ABI by utilising ABI types other than TBI, establishing which specific emotional expressions are associated with experienced emotional empathy and expanding on the model presented in this review.

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## **Part Two**

# **Social Cognition and Behavioural Difficulties: The Role of Empathy and Theory of Mind in Post-ABI Aggression**

This paper is written in the format ready for submission to the journal 'Brain and Cognition'. Please see Appendix C for the author guidelines.

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## Abstract

Behavioural problems are common after acquired brain injury (ABI). Of these, aggression and agitation are reported as the most problematic with 70% of ABI sufferers displaying aggressive tendencies at some point in their recovery. It has been suggested that deficits in social cognition may contribute to behavioural problems post-ABI. The current study aimed to investigate whether the social cognitive processes of empathy and theory of mind (ToM) varied within brain injured populations with and without aggression. It was hypothesised specifically that a deficit in one component of empathy, emotional empathy, would lead to heightened aggression. Therefore, the components of ToM and empathy were assessed individually. 35 participants with a diagnosis of ABI were recruited from neurobehavioural sites across the UK. Participants were divided into high aggression ( $n=19$ ) and low aggression ( $n=15$ ) groups determined by requirement for specialist intervention specifically for aggressive behaviour. Participants completed a battery of measures assessing components of ToM and empathy, as well as measures of mood and intellectual functioning. There was no difference found between the high and low aggression groups on any of the measures. It was therefore concluded that social cognitive deficits are not sufficient to explain aggression post-ABI. The implications of these results and recommendations for future research are discussed.

Key Words: 'THEORY OF MIND', 'EMPATHY', 'COMPONENTS', 'AGGRESSION, 'BRAIN INJURY'

## **1. Introduction**

### 1.1 Incidence of brain injury in the UK and common problems

Acquired brain injury (ABI) is an umbrella term incorporating traumatic (TBI), haemorrhagic, vascular, anoxic (and metabolic), and infective brain injuries. Advancements in life saving medical procedures and technologies have resulted in an increase in survival rates following ABI (Lux, 2007). It is estimated that the annual UK prevalence lies between 40 and 600 per 100 000 (British Society of Rehabilitation Medicine (BSRM), 2003; Kay & Teasdale, 2001; Neurological Alliance, 2003; Thornhill, Teasdale, Murray, McEwen, Roy & Penny, 2000; Yates, Williams, Harris, Round & Jenkins, 2006).

Approximately 135,000 individuals in the UK live with long term problems following brain injury (Neurological Alliance, 2003) with ABI sufferers commonly experiencing physical, cognitive, emotional, behavioural and psychosocial difficulties (Fleminger, 2005). Although 65-85 % of ABI patients make a good physical recovery, other disabilities can remain (BSRM, 2003). Whilst there is an inverse correlation between injury severity and a “good” recovery, disability is common at one year follow up regardless of brain injury severity (Thornhill, et al 2000).

### 1.2 Aggression following ABI

Behavioural difficulties following ABI are frequently reported by families as the most difficult to manage (Kinsella, Packer & Olver, 1991). Problematic behaviours post-ABI can be both positive (sexual disinhibition, aggression, agitation) and negative (lethargy, apathy) (Manchester, Hodgkinson, & Casey, 1997).

Aggression and agitation are reported as the most problematic (Fleminger, Greenwood and Oliver, 2003), with 70% of ABI sufferers likely to display aggressive

tendencies at some point in their recovery (McKinlay, Brooks, Bond, Martinage & Marshall, 1981). Relatives report aggression as a problem to a greater extent than ABI sufferers (Hart et al, 2003), suggesting incomplete insight into behaviour.

This lack of self-awareness can lead to frustration in ABI sufferers who do not understand why they are labelled as aggressive by their partner and family. Due to the burden placed on partners and families it is common to see relationships breakdown following ABI (Parsons, Carpenter-Hyland, Burright & Donovanick, 1995).

Research into the mediators of aggression following ABI has been hindered by the use of global measures of psychopathology, and measures of behaviours considered aggressive (i.e. stubbornness) producing inconsistent results (Dooley, Anderson, Hemphill & Ohan, 2008). Research applying measures based on theoretical frameworks of aggression have revealed a tendency for higher levels of anger, verbal aggression and maladaptive behaviours in ABI individuals when compared with non-brain injured comparison groups (Dyer, Bell, McCain & Rauch, 2006; Andrews, Rose & Johnson, 1998; Baguley, Cooper & Flemingham, 2006).

Aggression post-ABI has been postulated to result from an inability to inhibit aggressive impulses, as part of a dysexecutive syndrome (Golden, Jackson, Peterson-Rohne, & Gontkovsky, 1996; Grafman, Schwab, Warden, Pridgen, Brown & Salazar, 1996; Greve, Love, Sherwin, Stanford, Mathias & Houston, 2002; Hawkins & Trobst, 2000). Executive functioning is the process by which humans are able to plan, initiate, sequence, inhibit and switch between behaviours pertaining to a goal (Burges & Alderman, 2004; Wood, 2001). However, measures of cognitive functioning fail to reliably correlate with levels of behavioural deficits and/or self-awareness deficits (often attributed to be a factor in behavioural difficulties following ABI) (Blair & Cipolotti, 2000; Lanham, Weissenburger, Schwab & Rosner, 2005; McKinlay &

Brooks, 1984; Prigatano & Altman, 1990). Recent research has suggested that executive function problems are insufficient to explain behavioural problems such as aggression post ABI, and that the role of metacognitive processes must be considered (Bach & David, 2006; Lee, Farrow, Spence & Woodruff, 2004; Lezak, 1995; Stuss and Levine, 2002).

### 1.3 The rationale for Social Cognition as a mediator of aggression

Flavell (1976) defines metacognition as “one’s knowledge concerning one’s own cognitive processes or anything related to them” (p.232). The social cognitive domain is said to comprise of multiple metacognitive and cognitive processes (Bjorkqvist & Osterman, 2000), which enable us to understand and relate to the thoughts, feelings, beliefs and behaviours of other people (Beer & Ochsner, 2006; Bjorkqvist, & Osterman, 2000; Singer, 2006).

Bjorkqvist, Osterman and Kaukiainen, (1992), suggest that as humans develop we utilise aggression in an increasing socially intellectual manner. Physical aggression pre-exists language skills. As language and social skills develop, we come to rely more on verbal and indirect aggression. Indirect aggression puts great demand on learned social intelligence, enabling humans to socially manipulate individuals and inflict psychological harm with reduced likelihood of direct repercussions. Bjorkqvist and Osterman (2000), imply that a deficit in social cognition would diminish indirect aggressive strategies, forcing the recipient to rely on verbal and physical aggression which, due to their overt and socially unacceptable nature, often results in greater consequences.

Two social cognitive processes which have been associated with increased levels of aggression are Theory of Mind (Harvey, Fletcher & French, 2001; Renouf et al, 2010) and empathy (Bjorkqvist & Osterman, 2000; Kaukiainen et al, 1999).

#### 1.4 The complexity of Theory of Mind and its role in aggression post-ABI

Social cognition research in brain-injured populations has largely focused on Theory of Mind (ToM) (Muller et al, 2010). Baron-Cohen (2000) defines ToM as the ability to reflect on and understand the thoughts of oneself and another, enabling navigation through social situations. ToM is considered to be a key process of social cognition and poor TOM has been shown to result in an inability to effectively navigate the social world (Martin-Rodriguez & Leon-Carrion, 2010). Social cognitive deficits following an ABI may underlie self-awareness deficits (Beer & Ochsner, 2006) which may, in turn, play a role in aggression (Bach & David, 2006; Bjorkqvist, & Osterman, 2000; Hart et al, 2003). Bibby and McDonald (2005), emphasise that many of the sequelae of brain injury such as poor insight, inappropriate affect and poor social judgement overlap considerably with ToM deficits, explaining behavioural problems and suggesting that ToM problems may be a common occurrence after brain injury.

Research investigating ToM deficits post-ABI has produced inconsistent results (Martin-Rodriguez & Leon-Carrion, 2010). Bibby and McDonald, (2005) found that control participants performed better than traumatic brain injured participants on a range of story and cartoon measures of ToM. Henry, Phillips, Crawford, Ietwaart and Summers (2006), assessed the ability to infer affective mental states using the reading the mind in the eyes task (Baron-Cohen, Jolliffe, Mortimore & Robertson, 1997), which found a deficit in ToM ability following TBI. They also found the TBI group displayed a greater deficit in emotional recognition.

Bach, Happe, Fleminger and Powell (2000) were unable to explain social dysfunction post-ABI using a ToM deficit as a rationale. Milders, Ietswaart, Crawford and Currie (2008), found no significant association between measures of ToM and ratings of behaviour, indicating an intact ToM. Bach and David (2006) also found intact ToM despite participants displaying behavioural difficulties. However, they suggested that the ToM measures may not have been sensitive enough, with most participants performing at ceiling.

ToM has frequently been studied as a unitary construct however ToM is a metacognitive process which utilises many separate cognitive domains (memory, attention, executive functioning, etc.). Given ToM comprises of a variety of complex processes, this somewhat reductionist view of ToM has the potential to effect research findings. Stone, Baron-Cohen, Calder, Keane & Young (2003) distinguish ToM as being the combination of three components; *Attribution of epistemic mental states* (those referring to something in the world, i.e. knowledge, attention or belief); *Attribution of intention* (understanding whether an act was intentional or accidental); *Attribution of affective mental states* (e.g. desire, fear/anger, etc). The Bach and David (2006) paper previously mentioned for example appeared only to investigate the ability to infer epistemic mental states

Muller et al (2010) explored all three components of Stone et al (2003) finding a deficit of intention and possibly a deficit of affective mental state inference, with intact ability to infer epistemic mental state (Stone et al, 2003). However, it was not the intention of Muller et al (2010) to investigate these specific components proposed by Stone et al (2003), thus what we can infer from their results is limited.

### 1.5 Empathy as a mitigating factor in aggression and empathy deficits post-ABI

Empathy, like ToM, is a complex metacognitive process, able to be considered as the collection of component processes rather than a unitary phenomenon. Wood and Williams (2008), identify three components pertaining to the phenomenon of empathy. These are; *cognitive empathy*, the ability to understand what another is feeling; *compassionate empathy*, the ability to respond compassionately to another's distress; and *emotional empathy*, the ability to feel what another person is feeling (Wood & Williams, 2008).

Although considered part of emotional empathy (de Sousa, McDonald, Rushby, Li, Dimoska & James, 2010a; Baron-Cohen & Wheelwright, 2004), compassionate empathy can be distinguished as responding to another's emotional state rather than emotional empathy, the production of feelings resulting from another's emotional state (de Sousa; McDonald; Rushby; Li; Dimoska & James, 2010b; Shamay-Tsoory, Aharon-Peretz & Perry, 2009). Difficulty separating out these two processes is potentially the reason research into compassionate empathy is scarce. Furthermore, there appears to be no identifiable research investigating compassionate empathy following an ABI. Due to the lack of a concise definition and appropriate measures, the current study will focus on the remaining two components of empathy.

Research investigating empathy following ABI is scarce, although those that have suggest a deficit. Whilst cognitive empathy is the recipient of the majority of study (Grattan, Bloomer, Archambault & Eslinger, 1994; Knox & Douglas, 2009; Wells, Dywan & Dumas, 2005), more recent studies attempt to explore deficits of emotional empathy (Williams & Wood, 2010; Wood & Williams, 2008). Despite the expected dissociation between the neural pathways of each process (Shamay-Tsoory et al, 2009;

Shamay-Tsoory, 2011), deficits have been found in both emotional and cognitive empathy post-ABI (de Sousa et al, 2010a).

Levels of empathy in ABI sufferers have been suggested to have an adverse impact on carers' ratings of life satisfaction (Wells, et al 2005). Despite this, research focusing on identification of the neuroanatomical locality of the empathy process has become the norm (Nummenmaa, Hirvonen, Parkkola & Hietanen, 2008; Shamay-Tsoory, Tomer, Berger & Aharon-Peretz, 2003; Shamay-Tsoory, Tomer, Goldsher, Berger & Aharon-Peretz, 2004; Shamay-Tsoory et al 2009), with little research investigating the links between empathy and socio-behavioural deficits following ABI.

Rumble, Van Lange and Parks (2010), suggest that empathy may sustain cooperation in social dilemmas, whilst Kaukiainen et al (1999), propose that empathy mitigates aggression. One could therefore assume that aggression following ABI is the result of an empathy deficit (Bjorkqvist, & Osterman, 2000). It remains unclear whether behavioural changes are a result of an inability to accurately recognise emotional stimuli occurring naturally in the environment (facial expressions, etc.) or whether an inability to accurately recognise emotional states in another (i.e. anger/disgust), leads to an inability to respond within the expectations of others (Shamay-Tsoory et al, 2003). Furthermore, it is likely that an empathy deficit may prevent accurate interpretation of one's own emotional state (Beer & Ochsner, 2006).

Knox and Douglas (2009), suggest the social inappropriateness and breakdown in relationships, both common following ABI, result from an inability to accurately recognise facial expressions, an important facet of empathy (Cheung, Lee, Yip, King & Li, 2006; Shamay-Tsoory et al, 2009). Shamay-Tsoory et al (2004), discovered a deficit of emotional and cognitive empathy in individuals who had suffered a brain injury to the prefrontal cortex, specifically the orbital and medial regions, suggesting that they



mediate empathy. Damage to these areas has also been associated with altered social interaction and ‘acquired sociopathy’ (Blair & Cipolotti, 2000). Blair and Cipolotti (2000), report on the case of J.S. who displayed severe behavioural problems following an ABI. His ToM remained intact however his ability to empathise was severely inhibited. It can therefore be suggested empathy has an important role in mediating social interaction and mitigating aggression.

It is probable that a deficit to each of the components of empathy would result in different behavioural sequelae (Wood & Williams, 2008). Emotional empathy mitigates aggression through the production of emotions that will negatively reinforce future aggressive behaviour, i.e. through the sharing of distress from victim to aggressor (Beven, O’Brien-Malone & Hall, 2004). Therefore, an emotional empathy deficit would present itself in an egocentric way and thus a higher inclination to take action without being sensitive to the emotional impact on others would result. Cognitive empathy is a process of understanding, and can provide insight into what another is feeling, but not produce an affective reinforcer, the ability to know but not care (Kaukiainen et al, 1999). Therefore, a cognitive empathy deficit is likely to present as insensitivity to the emotional needs of others, and inability to adhere to the subtleties of social discretion. Considering these descriptions, it is likely that individuals displaying aggressive tendencies are more likely suffering from an emotional empathy deficit, whilst individuals suffering a cognitive empathy deficit are more likely to be considered rude but less aggressive as they will experience behavioural reinforcers through emotional empathy.

De Sousa et al (2010a; 2010b), demonstrated that impaired emotional responsivity is related to damage to two distinct, dissociated (cognitive and emotional) empathy pathways (Shamay-Tsoory et al 2009), with the latter emphasising a deficit to the

emotional empathy pathways. Furthermore, Mehrabian (1997) found a negative correlation between emotional empathy and levels of aggression and hostility. It is therefore possible that cognitive and emotional empathy deficits may play a role in the aggression post-ABI, however, evidence appears to indicate emotional empathy as the predominant factor.

### 1.7 The Present Study

The present study investigated the link between aggression and social cognition, specifically ToM and empathy. The vast majority of former research has specifically investigated deficits in ABI samples compared with normal/non-brain injured controls. The majority postulate that demonstrated deficits could explain problematic behaviours post-ABI. However, as the majority of studies compare an ABI sample to a “normal” population, they fail to consider the differences within an ABI population in terms of aggressive behaviour.

The current study compares an ABI sample which displays highly aggressive behaviour, requiring specialist input, with an ABI sample that does not show levels of aggression requiring specific clinical input. It is expected that participants would display a deficit in ToM and empathy comparatively against a “normal” population. However, this study is primarily concerned with the differences within the ABI populations.

The following hypotheses were tested:

First, consistent with the notion that empathy would serve to mitigate aggressive behaviour (Bjorkqvist, & Osterman, 2000) and the position that an emotional empathy deficit would be more likely to lead to aggression (Wood and Williams, 2008) the

“high aggression group” would display a greater deficit in emotional empathy than the “low aggression group”.

Second, considering the findings of Bach and David (2006) and Muller et al (2010), there would be no significant difference between the high and low aggression groups ability to attribute epistemic mental state.

Third, as suggested with Milders et al (2008), there would be no significant difference between the groups’ ability to attribute intention or attribute affective mental states.

Finally, the cognitive empathy levels for both groups will yield no significant difference, as suggested by Milders et al (2008) and the notion that cognitive empathy would cause someone to lack social discretion rather than display aggression (Wood & Williams, 2008).

## **2. Method**

The following study was approved by the Leeds (West) NHS Research Ethics Committee (REC) and ethics committee of each participating rehabilitation unit.

### 2.1 Participants

All individuals participating in the study had a brain injury severe enough to warrant specialist neurorehabilitation on an inpatient unit. It was deemed necessary to perform a power calculation to establish the required sample size for the present study. A power calculation using the effect sizes from the New Test of Theory of Mind (Martin, 2008), suggested that by using a significance level of 0.0083 the study would have an 80% power to detect the Martin (2008) effect sizes with a sample size of 14 in each group. As the effect sizes for other measures were not available it was not possible to more accurately determine a required sample size. Through reviewing previous literature

investigating social cognition post-ABI and using the results of this power analysis, it was felt that a sample of 15 in each group would be an appropriate sample size for use in the present study. This included the assumption that statistical methods used to reduce type I error would also be implemented (i.e. Bonferroni Correction).

### 2.1.1 Group Allocation

Two groups of individuals with ABI were required for the present study, one consisting of individuals who display high levels of aggression following ABI and one who display low. Self report and other rater measures were considered too subjective to act as an accurate group allocation device. Aggression and agitation behavioural data (recorded by neurorehabilitation sites) was also dismissed for two reasons. Firstly, measures were not consistent across neurorehabilitation organisations preventing accurate comparison. Secondly, the behavioural data was considered to provide information into the aggression and agitation levels of ABI individuals as they resided on the neurobehavioural unit. As these units are highly controlled and designed to limit episodes of aggression it was believed that the behavioural data would not provide a 'true' picture of participant aggression, instead demonstrating aggressive tendencies affected by the contrived clinical environment of the unit.

There is a clear divide between neurorehabilitation sites, those that are able to admit individuals who display overtly challenging behaviours and those that cannot. As aggression can be considered an overtly challenging behaviour, it was deemed appropriate to divide groups by type of neurorehabilitation site. Therefore the high aggression group consisted of participants residing on neurorehabilitation sites specialising in the rehabilitation of individuals who show challenging behaviours post-ABI and the low aggression group consisted of individuals residing on neurorehabilitation sites that are not equipped to rehabilitate individuals who display

high levels of overtly challenging behaviours post-ABI. Furthermore, it was important to establish that participants being selected for the high aggression group displayed high levels of aggression rather than other forms of overtly challenging behaviour, i.e. sexual disinhibition, etc. Therefore the clinical neuropsychology lead of the high aggression unit was consulted to ensure the selection of ABI individuals displaying high levels of aggression. Table 1 depicts the participant group allocation criteria.

**Table 1.**  
Participant Group Allocation Criteria

	<b>High Aggression Group</b>	<b>Low Aggression Group</b>
<b>Neurorehabilitation site type</b>	Specialising in rehabilitation for individuals displaying overtly challenging behaviours post-ABI	Not specialising in rehabilitation for individuals displaying overtly challenging behaviours post-ABI
<b>Consultation from clinical neuropsychology lead</b>	Selection of participants displaying high levels of aggression	

### 2.1.2 High aggression group

19 individuals who showed high levels of aggressive behaviour post-ABI, (13 male, 6 female) were recruited from two neurorehabilitation sites in the UK, York House (Brain Injury Rehabilitation Trust (BIRT)) and the Kemsley Unit (St Andrews Healthcare). Both sites specialise in the rehabilitation of individuals who show challenging behaviour following brain injury. All participants met the following criteria: (a) all were aged between 16 and 65, (b) all had suffered a brain injury, (c) each displayed or was known to have displayed a clinically significant level of aggressive behaviour requiring specialist input at a behavioural disorder unit, suggested by the clinical neuropsychology lead, (d) all were residing on an inpatient unit for the treatment of behavioural problems, (e) deemed to have capacity to consent to participation in accordance with the Mental Capacity Act (2005), (f) all had no severe physical disabilities that would prohibit the completion of the measures, e.g. blindness, tetraplegia, (g) all were able to adequately understand verbal explanations or written

information given in English and had no special communication needs that would prohibit administration of measures, (h) none had a history of aggression at the level of criminal offence prior to injury.

All participants gave informed consent to partake in the study. At the time of testing, the participants were aged between 19 and 64 (M age = 39.33 years SD = 12.57). Insufficient post traumatic amnesia data was available to report. Cohort years post-injury ranged from 4 to 23 years (M years = 13.94 years SD = 6.015). Participants age at injury ranged from 8 to 53 years (M age=25.53 years SD=15.36). Brain injuries were sustained by the cohort as result of trauma (n=10), anoxia (n=3), infection (n=2) and haemorrhage (n=1); information regarding the type of injury was unavailable for 3 members of the cohort. The education level of the cohort varied to include postgraduate (n=1), undergraduate (n=1), A-level/college (or equivalent) (n=2), GSCE/O-Levels (n=2) and no formal qualifications (n=11). Information regarding education level was unavailable for 2 participants.

### 2.1.3 Low aggression group

16 individuals with ABI displaying clinically insignificant levels of aggression (8 male, 8 female) were recruited from three neurobehavioural sites across the UK, Thomas Edwards Milton House (BIRT), Goole Neuro Rehabilitation Centre (BIRT and NHS) and Daniel Yorath House (BIRT). All sites specialised in rehabilitation following ABI and would not accept clients who displayed overtly aggressive tendencies. All participants met the following criteria: (a) all were aged between 16 and 65, (b) all suffered a brain injury (c) none displayed a clinically significant level of aggressive behaviour requiring specialist input at a behavioural disorder unit, (d) deemed to have capacity to consent to participation in accordance with the Mental Capacity Act (2005),

(e) all had no severe physical disabilities that would prohibit the completion of the measures, e.g. blindness, tetraplegia, (f) all were able to adequately understand verbal explanations or written information given in English and required no special communication needs that would prohibit administration of measures, (g) all had no past history of aggression at the level of criminal offence.

All participants gave informed consent to partake in the study. The participants were aged between 21 and 61 (M age = 36.88 years SD = 14.315). Insufficient post traumatic amnesia data was available to report. Cohort years post injury ranged from 0 to 18 years (M years = 2.75 years SD = 4.655). Participants injury age ranged from 8 to 60 years (M age=34.12 years SD=15.73). Brain injuries were sustained by the cohort as result of trauma (n=8), anoxia (n=2), infection (n=2) and haemorrhage (n=4). The education level of the cohort varied to include undergraduate (n=4), A-level/college (or equivalent) (n=5), GCSE/O-Levels (n=4) and no formal qualifications (n=1). Information regarding education level was unavailable for 2 participants.

#### 2.1.4 Group comparisons

Pearson's Chi Square analysis indicated that there were no significant differences between the groups for gender distribution ( $\chi^2 = 1.23, p = .317$ ) or injury type ( $\chi^2 = 2.22, p = .634$ ). There was a significant difference between groups for education level ( $\chi^2 = 12.06, p = .009$ ), with the low aggression group displaying a higher level of education. This difference is likely the result of 8 participants in the high aggression group sustaining their injury during childhood (pre-16 years old) therefore effecting their attainment of academic qualification. There was only one childhood brain injured participant in the low aggression group. A post-hoc Pearson's Chi Square was

performed to confirm a significant difference ( $\chi^2 = 5.48, p = .039$ ) between groups in childhood brain injuries.

T-test analysis revealed that the high aggression group displayed a larger number of years post injury ( $M=13.94, SD=6.02$ ) than the low aggression group ( $M=2.75, SD=4.66$ ) to a significant degree ( $t(31) = 5.95, p = .000$ ). This difference is the result of individuals displaying high levels of aggression requiring longer term rehabilitation than those displaying little to no aggression. The T-test analysis revealed no significant differences in age at testing ( $t(32) = 0.53, p = .598$ ) or age at injury ( $t(31) = -1.59, p = .122$ ). The lack of significant difference between groups at age of injury is interesting considering the 8:1 ratio between groups of participants who suffered a childhood ABI. This is possibly due to a large variation within the groups for age of injury, with the high aggression group ranging between age 8-53 and the low aggression group age at injury ranging between ages 8-60.

## 2.2 Questionnaires and Measures

All measures were completed by participants in private on the rehabilitation units under the supervision of the chief investigator. The total test battery took approximately 90-120 minutes per participant.

### 2.2.1 Psychometric measure of intelligence

Wechsler Abbreviated Scale of Intelligence (WASI, Wechsler, 1999): A measure of intelligence was necessary in the current study to ensure that IQ was not a confounding variable, affecting performance on other measures. The WASI is a brief intelligence measure developed from subtests of the Wechsler Adult Intelligence Scale III (WAIS-III, Wechsler, 1997). The selected subtests demonstrate high loading on general



intelligence and have exceptionally high reliability (Wechsler, 1999). Equivalency data between the WASI and the WAIS-III, endorse the validity of the WASI as a measure of general intelligence.

### 2.2.2 Measure of anxiety and depression

Hospital anxiety and depression scale (HADS, Zigmond & Snaith, 1983): The HADS is a screening device for measuring the severity of anxiety and depression separately. It is appropriately normed to be used in hospital settings and was originally applied to stroke patients. A measure of anxiety and depression was necessary to ensure mood factors did not confound results.

### 2.2.3 Questionnaires measuring aggression

Whilst assignment to group was based on the rehabilitation setting participants resided in a measure of subjective levels of aggression displayed by each participant and an 'other' who had substantial interaction with a participant were also taken.

Aggression Questionnaire (AQ, Buss & Perry, 1992): The AQ is a 29 item questionnaire. It required participants to rate how characteristic each statement was of them generally. It measures total aggression and in four subcategories: affective (anger), cognitive (hostility) and behavioural (physical and verbal). The AQ displays internal consistency between all four subscales and total score (coefficient  $\alpha = 0.72-0.89$ , Buss & Perry, 1992). Furthermore, test-retest data suggests the reliability of the measure over time (Buss & Perry, 1992). The AQ also displays adequate construct validity established from comparison of the completed AQ's with ratings from knowledgeable informants (Buss & Perry, 1992). The AQ has been used to assess aggression levels in previous studies involving brain injury participants (Dyer, et al, 2006; Greve, et al 2002). A paper by Dooley et al (2008) merits the AQ for its

theoretical groundings acknowledging that global measures of psychopathology do not permit detailed analysis of specific behaviours such as aggression.

Aggression Questionnaire – Partner Version (AQ-P, O’Conner; Archer, Fredrick & Wu, 2001): The AQ-P, developed from the AQ (Buss & Perry, 1992) is a 29 item ‘other-rater’ questionnaire, designed as a peer rating measure comparable with the AQ. O’Conner et al (2001) report the intercorrelations of the four subcategories to be within acceptable (moderate-high) boundaries, suggesting the reliability of the AQ-P as an ‘other-rater’. Furthermore, the congruent validity can be derived from the moderately high correlation of the AQ scales and AQ-P scales (O’Conner et al 2001). Vanderploeg, Belanger, Duchnick and Curtiss (2007), found the self rater reports of individuals with brain injury to be less reliable than those of professionals or peers. Therefore it was considered necessary to gain an ‘other-rating’ of participants’ aggression. As participants are inpatients it was felt that key workers would be in the best position to comment on participants levels of aggression. All participants and their key workers agreed to the completion of the AQ-P before it was administered.

#### 2.2.4 Video measure of ToM

The new test of ToM (NTTM, Martin, 2008): The New Test of ToM was developed due to dissatisfaction with available measures of ToM in use with ABI patients. It adopts the definition of ToM proposed by Stone et al (2003), discussed in the introduction). It assesses each of these three ToM components, providing content validity. Inter-rater reliability was established through administration of the test to a volunteer and two neuropsychologists. Its convergent validity has been established through its correlation (coefficient  $\alpha = 0.59$ ) with the “Faux Pas Task” (Stone, Baron-Cohen & Knight, 1998) a widely used measure of ToM which is suggested to measure

multiple components (attribution of intention and affective mental state). Furthermore, this video measure maintains ecological validity and does not rely on verbal ability. Although construct validity cannot be established due to a lack of empirical use, considering it is the only ToM measure known to the authors normed on an ABI population and assessing the three components outlined by Stone et al (2003), the other forms of validity and reliability would be sufficient.

#### 2.2.5 Measures assessing Empathy

Empathy Quotient (EQ, Baron-Cohen & Wheelwright, 2004): The EQ is a 60 item scale consisting of 40 empathy items and 20 filler items. According to Baron-Cohen and Wheelwright (2004), the EQ measures both cognitive and emotional empathy, as well as social skills. However, further investigation has suggested that the 40 item EQ can be reduced into 28 items measuring three factors, cognitive empathy, emotional reactivity and social skills (Lawrence, Shaw, Baker, Baron-Cohen & David, 2004). Therefore, this study will not be using the EQ as a measure of emotional empathy as it appears to assess processes involved with emotional empathy and not emotional empathy as a whole. Furthermore, the cognitive empathy subscales of the EQ (EQ-CE) has been shown to demonstrate adequate validity (Cronbach's  $\alpha = 0.84$ ) (Muncer & Ling, 2006). De Sousa et al (2010a) have used the EQ in this way when investigating empathy deficits in TBI patients. They reported no correlation between the EQ-CE and the 'perspective taking' subscale of the Interpersonal Reactivity Index (IRI-PC) (Davis 1980), although both are suggested to assess cognitive empathy. They attribute this to the IRI-PC tapping into attributions of epistemic mental state rather than cognitive empathy and affective ToM. This further suggests that the EQ-CE is appropriate to

assess cognitive empathy. In this current study the entire EQ was administered. EQ-CE scores were calculated from this.

Balanced Emotional Empathy Scale (BEES, Mehrabian, 2000): The BEES is a 30 item scale designed to reduce “acquiescence bias” (i.e. the tendency of some people to agree with most statements put to them and the tendency of others to generally disagree with any statement). It allow for the assessment of an individuals ability to vicariously experience another persons experience through shared emotion (Mehrabian, 2000). The BEES is internally consistent (coefficient  $\alpha = .87$ , Mehrabian, 1997) and displays satisfactory test-retest reliability (coefficient  $\alpha = .79$ ). The BEES high positive correlation with the Emotional Empathy Tendency Scale (EETS, Mehrabian & Epstein, 1972) suggest its strong convergent validity. Furthermore, the BEES has displayed a high construct validity, being used successfully to investigate emotional empathy in the ‘normal’ population (Shaprio, Morrison & Boker, 2004; Singer, Seymour, O’Doherty, Kaube, Dolan, and Frith, 2004; Van Hasselt, Baker, Romano, Sellers, Nosner & Smith, 2005) and an ABI population (de Sousa et al, 2010a; Wood & Williams, 2008). Perhaps most importantly, the BEES has negatively correlated with aggression and violence at a significant level ( $p < 0.01$ , Mehrabian, 1997) indicating its appropriateness as a measure of emotional empathy.

### 2.3 Procedure

At each site, potential participants were identified and initially approached by a member of the clinical team and consent to meet with a researcher to discuss the project was obtained. The chief investigator met with potential participants, in private on the unit to discuss the project and answer questions. Participants had 24 hours to consider participation (in line with NHS REC protocol), after which they were contacted by chief

investigator in person or via telephone to confirm their participation. An appointment was arranged to complete the test battery at the neurorehabilitation site. Prior to the commencing of testing, participants were given chance to answer any further questions and complete the consent forms. The testing was completed in private on site and lasted approximately 90-120 minutes. The tests were completed in the following order with each participant: HADS, BEES, AQ, NTTM, EQ, WASI. For the HADS, BEES, AQ, & EQ, the participants were given the measure, the instructions (on the top of each measure) were read to them and participants filled in the scale. The NTTM required participants to watch a series of scenes on a laptop computer and answer questions about the scene after each. The questions were read aloud by the examiner but were also presented on the screen. The WASI was administered as to adhere with standardisation (Weschler, 1999). If the participant was unable to read or write the questions were read to them, and verbal answers written down by the researcher.

The HADS was scored whilst the participant completes the New Test of Theory of Mind. Any scores above 10 (above borderline) for anxiety or depression were discussed with the client at the end of testing with the prospect of feeding the HADS information to the clinical team if it was what the client desired or an issue of risk arose. The participants were then thanked for their participation and given opportunity to ask any questions.

Once an appointment was arranged with participants it was possible to approach their key workers. The information sheet was discussed with the key worker and they were given chance to ask any questions. They were then given 24 hours to consider their participation after which they were contacted and an appointment was arranged to complete the AQ-P. The key worker filled in the AQ-P. This was done in private and lasted approximately 5 minutes. To respect the key workers high work load, the

researcher was flexible in allowing them to take the AQ-P away to fill in when they had time. They were informed that should they have any further questions they should locate the researcher before proceeding. Key workers were then debriefed and thanked for their participation.

## 2.4 Data analysis

A Pearson's Chi Square and Independent T-tests were used to compare the demographic details of participants and establish any significant differences between groups.

All results were analysed using SPSS for Windows, version 18.0 (SPSS Inc). All measures of social cognition in the test battery were subject to a univariate analysis using independent t-tests or Mann-Whitney U's to compare the performance of both groups on each subscale.

A correlation analysis comparing the measures of social cognition with measures of aggression was used to assess the relationship between the two constructs.

The self and other aggression rater scores for each group underwent an intraclass correlation analysis to establish the level of agreement between participants and their key workers. Further, graphical analysis was performed to establish any trends in participants over or under rating their aggressive behaviour compared to their key workers.

## **3. Results**

### 3.1. Missing Data

Participant disengagement and unavailable information resulted in incomplete data sets in the high aggression group (n=4) and low aggression group (n=2).

### 3.2. Data Screening

Participants were required to complete the HADS and WASI to determine any group differences in mood or intellectual functioning that may affect results. The mean scores are summarised in table 1. A test of normality was conducted to determine the use of a parametric measure or nonparametric measures.

#### 3.2.1. Mood

The Kolmogorov-Smirnov test of Normality indicated significant within group distribution in the low aggression group depression scores (K-S = .23,  $p=.023$ ) and total HADS scores (K-S = .24  $p=.014$ ), implying the requirement for nonparametric analysis for these measures. To allow comparison, nonparametric measures were used for all mood subtests. A Mann-Whitney U nonparametric measure revealed no significant differences between groups for anxiety ( $U=118.5$ ,  $N1=19$   $N2=16$ ,  $p=.265$ ), depression ( $U=144$ ,  $N1=19$   $N2=16$ ,  $p=.790$ ) and total HADS score ( $U=139$ ,  $N1=19$   $N2=16$ ,  $p=.666$ ). As the alpha scores do not fall within the statistically significant range there is no indication that anxiety or depression are confounding variables.

#### 3.2.2. Intellectual Functioning

The group IQ scores are compared in Fig.1. The Kolmogorov-Smirnov test of Normality indicated no significant distribution within groups, therefore indicating the appropriate use of parametric measures. An independent t-test was performed. The Levene's test of Equality was significant for performance IQ ( $W=7.6$ ,  $p=.010$ ) indicating that the variance within the performance IQ sample was not equal. Therefore, the equality of variance was not assumed for PIQ. The independent t-test revealed no significant differences between groups for verbal IQ ( $t(30) = -2$ ,  $p=.057$ ), performance IQ ( $t(21.32) = -1.3$ ,  $p=.209$ ) or full scale IQ ( $t(30) = -1.94$ ,  $p=.062$ ). Therefore there was no reason to believe that intellectual functioning confounded group differences.

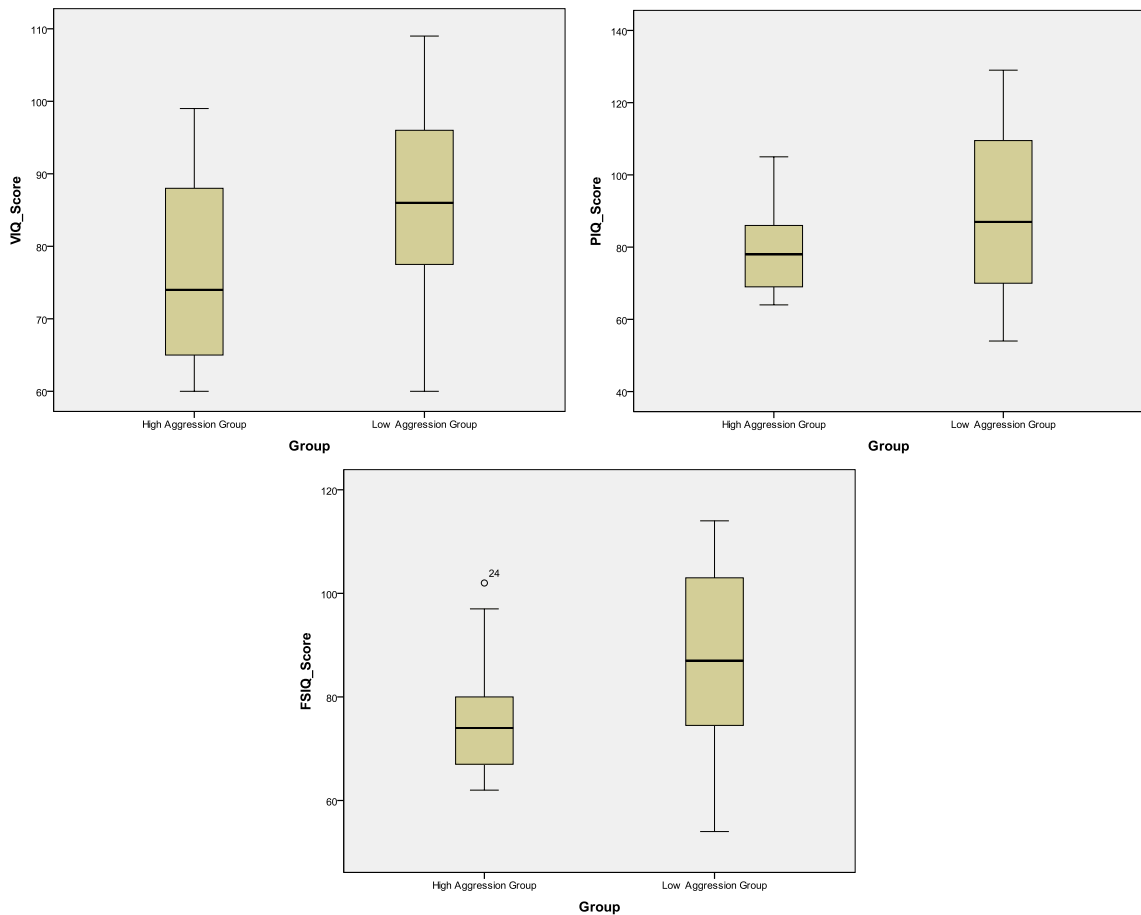
**Table 2**

The mean and standard deviation scores for high aggression, low aggression groups on measures of intellectual functioning and mood.

	Mood			Intelligence (WTAR)		
	Anxiety	Depression	Total HADS	Verbal IQ	Performance IQ	Full Scale IQ
High Aggression Group						
<i>n</i>	19	19	19	17	17	17
Mean	6.89	6.26	13.16	76.71	79.88	76.35
S.D.	3.57	4.03	5.167	11.94	13.64	11.37
Low Aggression Group						
<i>n</i>	16	16	16	15	15	15
Mean	6.25	6.56	12.81	85.87	89.13	86.47
S.D.	6.03	5.6	10.55	14.24	24.5	17.76

**Fig.1**

Boxplot representation of verbal IQ (VIQ), performance IQ (PIQ) and full scale IQ (FSIQ) scores for both high aggression and low aggression groups.





### 3.3. Aggression grouping

The mean group self and other rated aggression scores can be found in table 2. A Levene's test of equality was significant for other ratings of anger ( $W=10.15$ ,  $p=.003$ ) and total aggression ( $W=4.82$ ,  $p=.036$ ), therefore the equality of their variance is not assumed. Independent T-tests revealed no significant group difference for self ratings of hostility ( $t(33) = 1.11$ ,  $p = .277$ ) and verbal aggression ( $t(33) = 1.27$ ,  $p = .213$ ) as well as other ratings of hostility ( $t(31) = .87$ ,  $p = .391$ ), verbal aggression ( $t(31) = 1.10$ ,  $p = .281$ ) and total aggression ( $t(23.2) = 2.04$ ,  $p = .053$ ), though the latter is approaching significance. Independent T-tests revealed significant differences between other ratings of physical aggression ( $t(31) = 3.09$ ,  $p = .004$ ) and anger ( $t(20.63) = 2.09$ ,  $p = .049$ ) as well as self ratings of anger ( $t(33) = 2.89$ ,  $p = .007$ ), physical aggression ( $t(33) = 2.61$ ,  $p = .014$ ) and total aggression ( $t(33) = 2.88$ ,  $p = .007$ ). These results suggest that the high aggression group displays a higher level of overall aggression, attributable to higher levels of physical aggression and anger.

**Table 3**

The mean and standard deviation scores for high aggression, low aggression groups on self and other ratings of aggression.

	Self rated Aggression					Other rated Aggression				
	Physical	Verbal	Hostility	Anger	Total	Physical	Verbal	Hostility	Anger	Total
High Aggression Group										
n	19	19	19	19	19	18	18	18	18	18
Mean	27.26	17.74	25.26	22.89	93.16	26.05	16.28	20.22	22	84.89
S.D	7.82	5.02	8.03	5.07	17.93	7.07	4.23	7.98	4.6	19.07
Low Aggression Group										
n	16	16	16	16	16	15	15	15	15	15
Mean	19.88	15.5	22.13	16.94	73.19	17.93	14.33	17.8	16.87	66.93
S.D	8.97	5.38	8.76	7.11	23.13	8.87	5.92	7.96	8.52	29.39

### 3.4. Social Cognition

Mean BEES, EQ-CE and NTTM (epistemic, affective and intention) scores were calculated for both groups. These scores are summarised in table 3. Normality tests were completed to determine the use of parametric or nonparametric tests. The Kolmogorov-Smirnov reported no significance difference within the groups on the BEES, the NTTM or the EQ-CE. Therefore, parametric analysis was appropriate for all social cognition measures.

#### 3.4.1. Cognitive and Emotional Empathy

Two independent t-test revealed no significant difference between the groups in levels of cognitive empathy (EQ-EC:  $t(32) = -.29$ ,  $p = .772$ ) and emotional empathy (BEES:  $t(33) = .538$ ,  $p = .594$ ), suggesting that groups did not differ in cognitive or emotional empathy levels.

#### 3.4.2 Epistemic, Intention, and Affective ToM

Two independent t-tests demonstrated no significant difference between groups for levels of affective ToM (NTTM affective:  $t(32) = -1.30$ ,  $p = .202$ ) or epistemic ToM (NTTM epistemic:  $t(32) = -.41$ ,  $p = .681$ ). The Levene's test of Equality was significant for intention ToM ( $W = 4.73$ ,  $p = .037$ ) indicating that there was not equal variance within the intention ToM sample. A Bonferroni Correction was performed to adjust for multiple concepts being compared (epistemic ToM, intention ToM, affective ToM). This reduced the value at which a difference would be considered statistically significant from 0.05 to 0.017, meaning only p values less than 0.017 would be deemed statistically significant. An independent t-test, not assuming equal variance, revealed no significant difference between groups on scores of intention ToM (NTTM intention:  $t(27.61) = -2.27$ ,  $p = .031$ ). This indicates a lack of difference between groups on all components of ToM.

**Table 4**

Mean and standard deviation scores for high aggression, low aggression groups on measures of social cognition, incorporating normative values.

	Empathy		Theory of mind (NTTM)		
	BEES	EQ-CE	Epistemic	Intention	Affective
High Aggression Group					
<i>n</i>	19	18	18	18	18
Mean	32.26	12.61	33.56 <sup>b</sup>	30.19 <sup>a, b</sup>	85.51 <sup>b</sup>
S.D	35.59	5.7	11.06	15.17	36.54
Low Aggression Group					
<i>n</i>	16	16	16	16	16
Mean	26.81 <sup>b</sup>	13.13	34.97 <sup>b</sup>	39.69 <sup>a, b</sup>	99.52 <sup>b</sup>
S.D	21.05	4.37	8.46	8.69	23.96
Normative data					
Mean	45	13.18	43.44	50.11	129.89
S.D	24	4.02	4.9	8.52	19.7

Note: Normative data obtained from: BEES (Mehrabian, 2000); EQ-CE (De Sousa et al, 2010a (control group)); NTTM (Martin, 2008 (control group))

<sup>a</sup> Significant ( $p < 0.05$ ) difference between high aggression and low aggression groups. However, not significant at the Bonferonni corrected ( $p < 0.017$ ).

<sup>b</sup> Significant ( $p < 0.01$ ) difference between group and normative sample.

### 3.5 Comparison of Sample to Normative Data

Comparison of the current sample and normative data derived from a 'normal' population were analysed using one sample t-tests. In the absence of available norm data, control group data was used to establish a normative comparison and analysed with independent t-tests. There was a significant difference between the normative sample and the current sample for epistemic ToM (high aggression group  $t(25) = 2.54$ ,  $p = .001$ ; low aggression group  $t(23) = 2.74$ ,  $p = .001$ ; total ABI sample  $t(41) = 2.72$ ,  $p = .000$ ), affective ToM (high aggression group  $t(25) = 3.38$ ,  $p = .000$ ; low aggression group  $t(23) = 3.23$ ,  $p = .000$ ; total ABI sample  $t(41) = 3.40$ ,  $p = .000$ ), intention ToM (high aggression group  $t(25) = 3.64$ ,  $p = .000$ ; low aggression group  $t(23) = 2.90$ ,  $p = .000$ ; total

ABI sample  $t(41) = 3.30, p = .000$ ). This suggests that both groups (assessed separately and together) displayed deficit in all components of theory of mind processing.

There was no significant difference between the normative sample and the current sample for cognitive empathy (high aggression group  $t(38) = -.37, p = .713$ ; low aggression group  $t(36) = -.04, p = .971$ ; total ABI sample  $t(54) = -.26, p = .797$ ), indicating that neither group displayed a cognitive empathy deficit.

A significant difference in emotional empathy between the current sample and normative sample was present in the low aggression group ( $t(15) = -3.46, p = .004$ ) and total ABI sample ( $t(34) = -3.05, p = .004$ ) but not in the high aggression group ( $t(19) = -1.56, p = .136$ ), suggesting the low aggression group and total sample displayed a deficit in emotional empathy whilst the high aggression displayed intact emotional empathy.

### 3.6. Aggression, Self vs Other Ratings

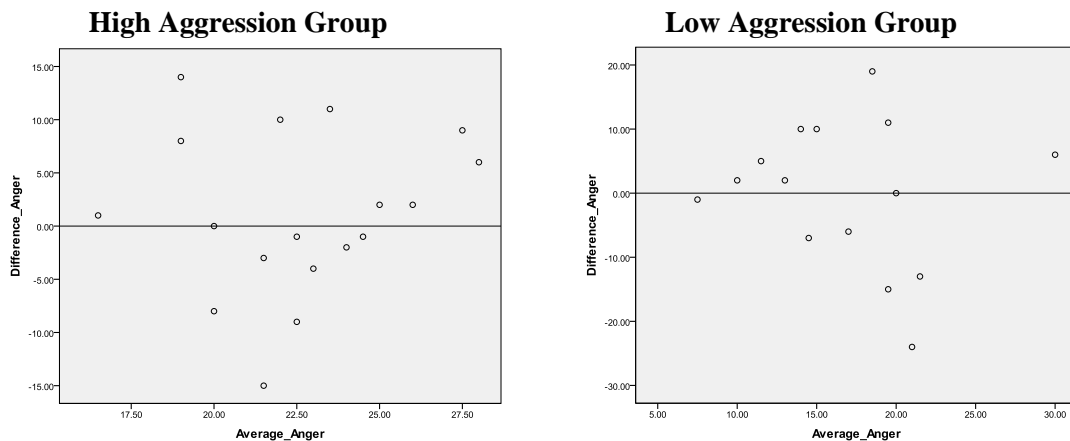
To establish aggression rating levels of agreement between participants and key workers for each group, 10 intraclass correlation coefficients were carried out. There were no strong levels of agreement between participant (AQ) and key worker (AQ-P) ratings of anger (high aggression group:  $ICC = -.19, p = .804$ ; low aggression group:  $ICC = .01, p = .477$ ); physical aggression (high aggression group:  $ICC = .39, p = .044$ ; low aggression group:  $ICC = .34, p = .095$ ); verbal aggression (high aggression group:  $ICC = -.11, p = .670$ ; low aggression group:  $ICC = -.08, p = .610$ ); hostility (high aggression group:  $ICC = -.21, p = .655$ ; low aggression group:  $ICC = .05, p = .424$ ) and total aggression (high aggression group:  $ICC = -.003, p = .504$ ; low aggression group:  $ICC = .13, p = .314$ ). This data suggests that participants and key workers disagreed on interpretations of participant aggression across most types of aggression for both

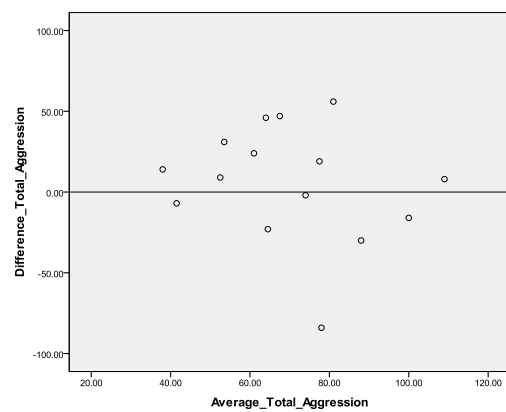
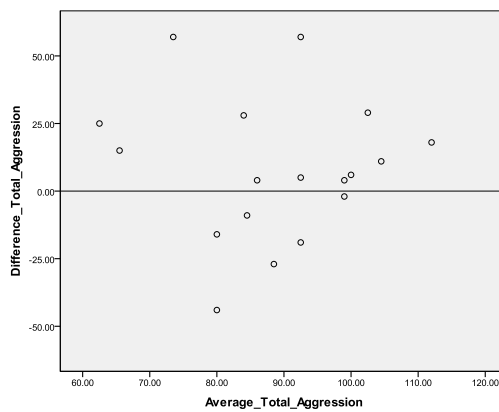
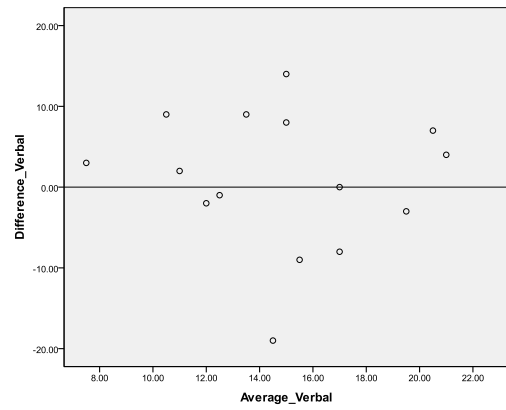
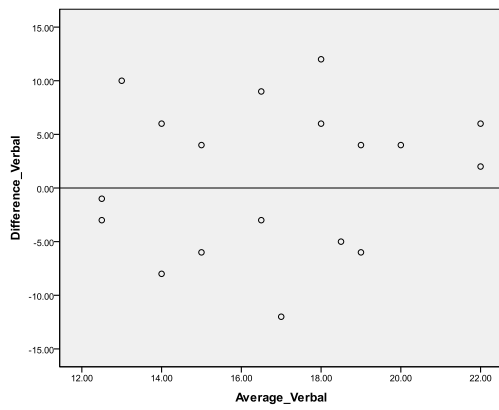
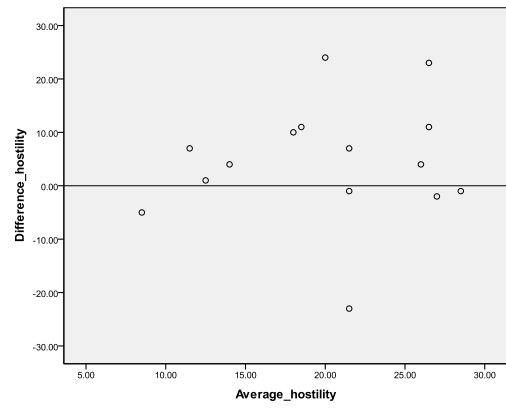
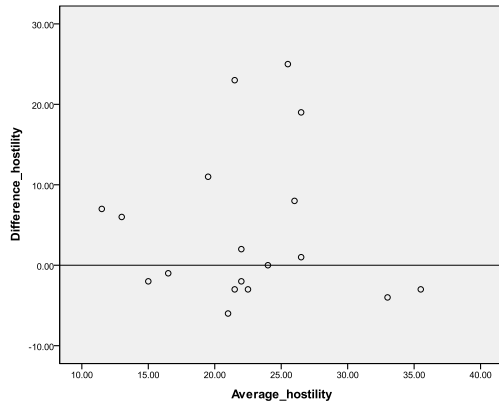
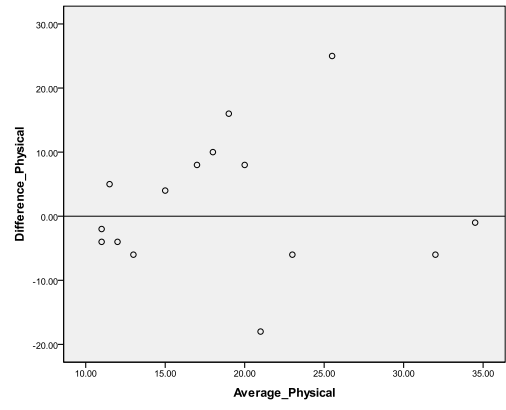
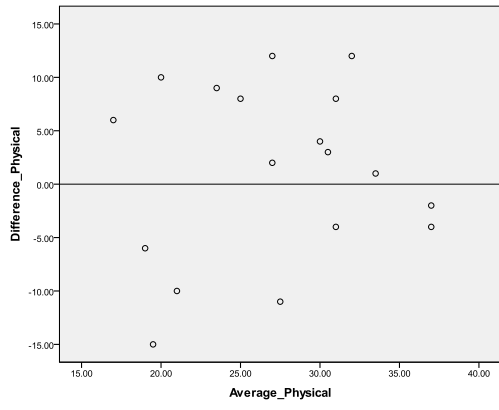
groups. The only exception being the low aggression groups ratings of physical aggression.

The differences between participant and key worker ratings and the average of the two were compared for each type of aggression and the total score. These comparisons are represented as scatterplots (fig. 2) and were used to identify any trends in key worker reports of aggression, i.e. under reported or over reported aggression, when compared with participant ratings. Review of the scatterplots suggests that participants in the high aggression group generally (12:6) rated their total levels of aggression higher than their key worker. Furthermore, in the low aggression group, the majority of participants (10:5) rate their hostility levels higher than their key workers rate their hostility. No other obvious trends of rating differences were present.

**Fig. 2**

Scatter Plots depicting the difference between the participant and key worker rating of different types of aggression (e.g. Self rated anger – Other rated anger) plotted against the average for the two ratings (e.g. (Self rater anger + Other rater anger)/2). The horizontal line represents agreement. Scores above the line indicate a higher participant rating and below indicate a lower participant rating.





### 3.7. Correlation of Social Cognition Measures

A Pearson's correlation analysis (Table 4) revealed a significant moderate correlation between the measures of empathy (EQ-CE and BEES:  $r(32) = .62, p = .000$ ). A significant moderate correlation was also present between epistemic ToM and intention ToM (NTTM epistemic and NTTM intention:  $r(32) = .46, p = .007$ ) and intention ToM and affective ToM (NTTM intention and NTTM affective:  $r(32) = .54, p = .001$ ). Measures of affective ToM did not correlate significantly with epistemic ToM ( $r(32) = .296, p = .090$ ). No significant correlations between the empathy and ToM measures were revealed. This suggests a moderate relationship between emotional and cognitive empathy, affective and intentional ToM and intentional and epistemic ToM.

**Table 5**

A table depicting the results of a Pearson's correlation coefficient analysing the correlation between measures of social cognition.

		<b>Correlations</b>				
		EQ-CE	BEES Raw	NTTM Epistemic	NTTM Intention	NTTM Affective
EQ-CE	Pearson Correlation	1	.618**	.067	.158	.035
	Sig. (2-tailed)		.000	.707	.372	.846
	N	34	34	34	34	34
BEES Raw	Pearson Correlation	.618**	1	.098	.129	.285
	Sig. (2-tailed)	.000		.582	.469	.103
	N	34	35	34	34	34
NTTM Epistemic	Pearson Correlation	.067	.098	1	.457**	.296
	Sig. (2-tailed)	.707	.582		.007	.090
	N	34	34	34	34	34
NTTM Intention	Pearson Correlation	.158	.129	.457**	1	.544**
	Sig. (2-tailed)	.372	.469	.007		.001
	N	34	34	34	34	34
NTTM Affective	Pearson Correlation	.035	.285	.296	.544**	1
	Sig. (2-tailed)	.846	.103	.090	.001	
	N	34	34	34	34	34

\*\* . Correlation is significant at the 0.01 level (2-tailed).

### 3.8. Correlation of Social Cognition Measures and Aggression Measures

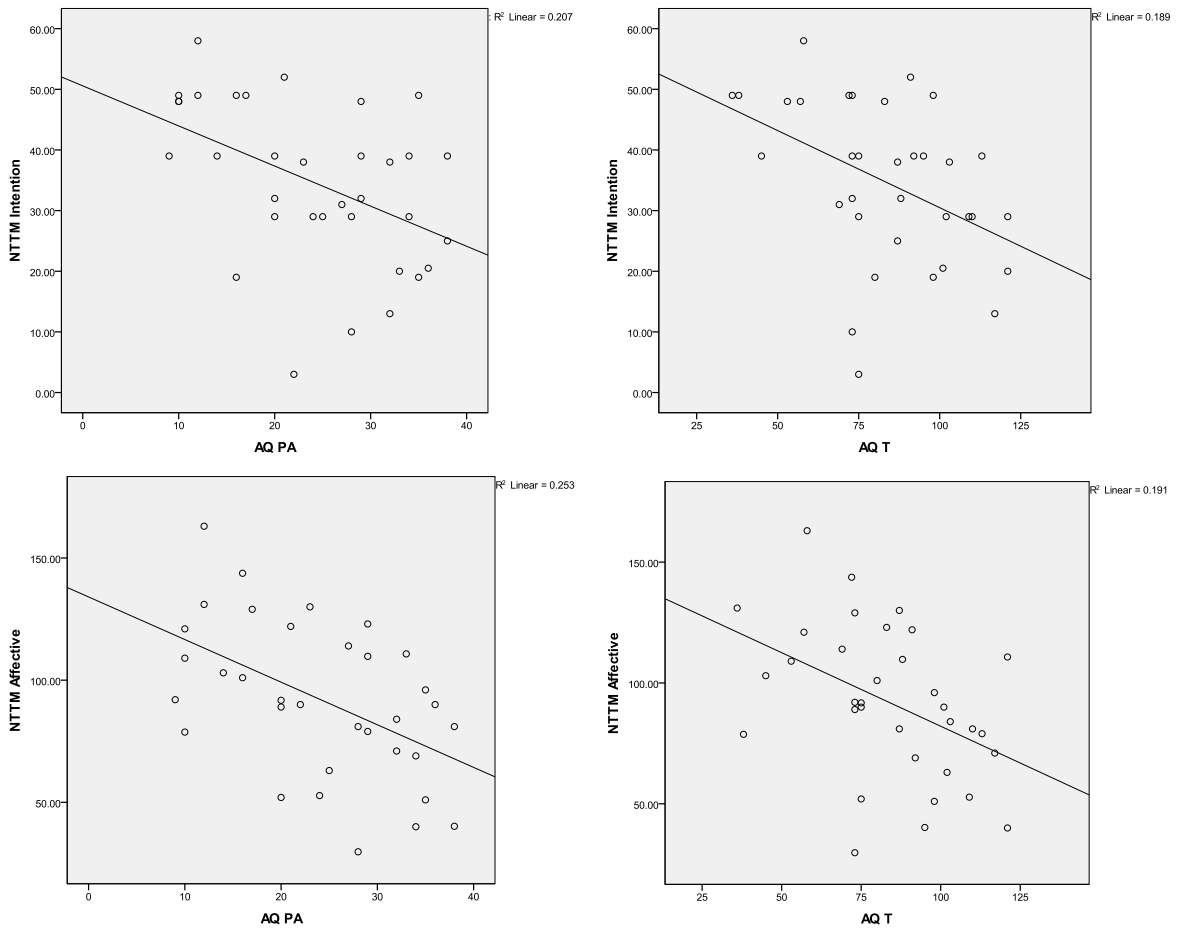
A Pearson's correlation was performed to identify whether the measures of aggression (self and other) correlated with the measures of social cognition (BEES, EC, NTTM Intention, NTTM epistemic and NTTM affective). Of these there was no significant relationship between any of the 'other rated' types of aggression and any of the measures of social cognition. This indicates a lack of relationship between aggression as rated by key workers and social cognition. Furthermore, there was no significant difference between any type of 'self rated' aggression and the BEES, EQ-EC or NTTM epistemic, suggesting a lack of relationship between self rated aggression and emotional empathy, cognitive empathy and epistemic ToM. Following a Bonferroni Correction reducing the level of statistical significance to  $p < .017$  (accounting for the NTTM, AQ and Pearson's correlation), there was a significant, negative, moderate, correlation between NTTM intention and self rate physical aggression ( $r(34) = -.45, p = .007$ ), and total aggression ( $r(34) = -.44, p = .010$ ). Self rated hostility was approaching significance ( $r(34) = -.38, p = .023$ ). Furthermore, using the same Bonferroni Correction, there was a significant, negative, moderate correlation between NTTM affective and self rated physical aggression ( $r(34) = -.5, p = .002$ ) and total aggression ( $r(34) = -.44, p = .010$ ). Self rated verbal aggression was approaching significance ( $r(34) = -.38, p = .027$ ). This indicates that intention ToM and affective ToM both moderately diverge from self rated physical and total aggression. This relationship is depicted in scatterplots in figure 3.



**Fig. 3**

Scatter Plots depicting the divergent relationship between self ratings of physical and total aggression and intentional ToM and affective ToM. Each scatterplot contains a linear line of best fit.

*Note:* AQ PA = physical aggression; AQ T = total aggression; NTTM affective = affective ToM; NTTM intention = intention ToM.



#### 4. Discussion

The current study was designed to investigate the hypothesised link between aggression post-ABI and social cognitive processes, specifically the components of ToM and empathy. It was hoped that the establishment of such a link would inform rehabilitation units specialising in brain injured individuals displaying aggressive behaviour. This was actualised through the administration of measures of both empathy and ToM to two brain injured populations, one displaying high levels of aggression and one displaying low levels.

As expected there were no statistically significant differences between high and low aggression groups on the three components of ToM; attribution of epistemic mental state, attribution of affective mental state and attribution of intention. This finding suggests that ToM deficits are not a reliable factor influencing differences in aggression within the ABI population. This supports previous research assessing ToM in a behaviourally disordered brain injured population (Bach & David, 2006; Bach et al 2000; Milders et al, 2008). However, it should be acknowledged that Intention ToM was approaching significance.

As hypothesised, there was no significant difference between the high and low aggression group's cognitive empathy scores. Cognitive empathy therefore does not explain differences in levels of aggression post-ABI, supporting notions broached in previous studies (Milders et al 2008; Wood & Williams, 2008).

In contrast to the hypothesis, there was no difference in levels of emotional empathy between the high aggression and low aggression group, suggesting that levels of emotional empathy are not a substantial explanation of the differences in levels of aggression displayed within an ABI population. This finding contests the proposal of Bjorkqvist and Osterman (2000) that empathy would mitigate aggressive behaviour and the suggestion that a lack of emotional empathy would produce aggressive tendencies (Wood & Williams, 2008). The primary finding of this study is that social cognitive processes of ToM and empathy are unable to account for differences in aggressive behaviours within the ABI population. If indeed there is a unifying deficit that explains an increment in aggression post-ABI it appears to be something other than ToM or empathy. Therefore, the present study would dissuade the introduction of social cognition rehabilitation programs into neurorehabilitation settings as a means to reduce aggressive behaviour.

The use of such stringent significance level ( $p < .017$ ) for analysis of the NTTM may have produced a type II error, specifically regarding the NTTM intention, which prior to the Bonferroni correction was significant. Bonferroni corrections are used within statistical analysis to avoid type I error. Whilst this level of experimental rigor is important to ensure results are accurate there is the concern that being overly rigorous may produce type II error. The use of a Bonferroni correction is not an exact science, with different schools of thought favouring the use of different criteria, i.e. dividing the significance level by the number of measures used, dividing the significance level by the number of concepts measured, etc. As such there is no clear definition of how rigorous one should be in using a Bonferroni correction

A particularly rigorous stance was taken on the statistical analysis of the NTTM, dividing the significance level by the number of components being assessed. The NTTM is an unpublished measure, developed at the level of doctoral dissertation. Due to its lack of empirical use its construct validity has not been established and it was therefore necessary to impose strict significance criteria (a Bonferroni Correction) in line with responsible statistical analysis.

Furthermore, the power analysis used to determine the number of participants required for the present study relied upon the NTTM. The results of this analysis suggested that the use of 14 participants in each group provided an 80% power to detect a significant effect, providing the level at which a p value would be statistically significant was set below 0.0083. Therefore, the use of stringent significance levels is in keeping with the requirements of using the NTTM with a sample of this size. It will be important for future research to investigate the role of intention ToM in aggression to provide clarity to this issue. The current paper however is unable to suggest a deficit in intention ToM produces aggressive behaviour post-ABI.

Comparison of the current ABI sample to normative data revealed a deficit in all components of ToM within each group and total ABI sample. This expected finding lends support to those who would propose a ToM deficit post-ABI (Bibby and McDonald, 2005; Henry et al, 2006). The current sample (high aggression, low aggression and total ABI sample) displayed preserved cognitive empathy in comparison with the normative group. This contests previous findings of cognitive empathy deficits in an ABI population (de Sousa et al 2010a, Shamay-Tsoory et al, 2004). Furthermore, when compared with the normative sample the low aggression group displayed a deficit in emotional empathy inconsistent with the high aggression group. This result is unexpected and emphasises the null hypothesis that emotional empathy deficits fail to explain aggressive behaviour post-ABI. Despite this inconsistency, the total ABI sample displayed a deficit in emotional empathy when compared with the normative group, supporting previous research (de Sousa et al 2010a; Williams & Wood, 2009; Wood & Williams, 2008).

The high aggression group displayed significantly higher scores than the low aggression group on self ratings of anger, physical and total aggression, whilst also significantly differing on other ratings of anger and physical aggression, with total aggression approaching significance ( $p=0.053$ ). In an ABI population heightened anger is likely to lead to physical outbursts and produce a great difficulty in management of behaviours. This will most likely require specialist input at a neurorehabilitation site specialising in the management of such overt aggressive behaviour. Therefore, it is logical that the groups displayed the differences they did. The fact that hostility and verbal aggression did not produce group difference is understandable considering the management of verbal aggression, though unpleasant, is not as difficult to manage as physical outbursts and therefore both groups are equally likely to display it. Hostility is

considered to be a form of indirect aggression requiring a social cognition and reducing risks (Bjorkqvist et al, 1992). As no group difference were found for social cognition measures, it is understandable that no group differences should be displayed in hostility, supporting the notions of Bjorkqvist et al (1992).

There was a moderate correlation between empathy and ToM measures suggesting the neuropsychological processes being measured are similar, but not the same. This lends support to the concept of emotional and cognitive empathy as different processes unified under the construct of empathy (Shamay-Tsoory et al 2009; Shamay-Tsoory, 2011; Wood & Williams, 2008). The study also revealed a moderate correlation between epistemic ToM and intention ToM, and between intention ToM and affective ToM. This suggests a connection between these components of ToM, but emphasises their value as separate constructs, supporting the assertions of Stone et al (2003). Interestingly epistemic ToM and affective ToM did not correlate. Attribution of an epistemic mental state and attribution of affective mental states are quite polarised concepts, the prior pertaining to something in the world and the latter representing an abstract phenomenon (Stone et al, 2003). It is conceivable that the failure of the two components to correlate is an indication of their separateness on a processing level; it is not necessary to understand what someone is thinking to understand what they are feeling. Furthermore, correlation of each with an ability to infer intent accounts for a plausible mediation between the two, a process which relies on attribution of affective and/or epistemic mental states to infer intentions. Therefore, this finding lends support to Stone et al (2003) and builds on their component model of ToM, expressing the unifying factor between them as the ability to infer intention. Furthermore, these findings emphasise the importance of assessing components of ToM and empathy rather than considering them to be unitary constructs.

The comparison of participants ratings of aggression and ratings provided by their key worker suggest a disagreement across all forms of aggression (verbal, physical, hostility and anger), regardless of group. There was no indication that participants rated their aggressive behaviour consistently higher or lower than their key workers rated it. Previous literature (Hart, 2003; Parson et al, 1995) has suggested that following an ABI individuals have a reduced insight into their behaviour indicated by lower scores on 'self' ratings of behaviour compared against 'other' ratings. The current study does not share this finding, instead suggesting that brain injured individuals in most cases rated their aggression 'differently' to their keyworker rather than lower. Where trends did occur, participants rated their aggression higher than rated by their key workers, not lower. This is possibly due to the use of key workers rather than family members as in previous studies. The key workers professional role in a neurorehabilitation setting results in their experience of multiple individual's post-ABI, differing in levels of aggression. As a result staff may become desensitised to aggression and therefore underreport it. Furthermore, family members have a premorbid experience of the individual and often an emotional investment in the situation, both of which may skew their ratings of aggression. Finally, the participant may rate their aggression levels as high if they have retained enough insight to compare themselves to their premorbid self. It is clear that multiple factors may influence the comparison of aggression from self and other ratings, therefore interpretations should be made with caution.

Comparison of social cognitive scores against self and other ratings of aggression post-ABI indicated a divergent relationship between intention and affective ToM with self rated, physical and total aggression. This suggests that as affective and intentional ToM decline ABI sufferers are likely to rate themselves as more physically aggressive, likely producing higher total aggression scores. It is possible that an inability to

accurately understand the intentions of others may lead to aggressive outbursts, e.g. not understanding that staff cannot let you smoke at this time as it is session time. As the correlation is moderate further research is required to understand the implications of this relationship, although explanations must be considered. Furthermore, an inability to understand the affective mental state of another could also lead to aggressive behaviour, e.g. not understanding that someone is ignoring you because they are upset. As demonstrated these ToM components correlate and it is likely that they compensate for one another i.e. being able to understand the intentions of another would perhaps allow for an inability to understand what they are feeling and vice versa.

Interestingly, the 'other' ratings of aggression did not correlate with any social cognitive constructs. One could suggest that participants lacked insight (Hart, 2003; Parson et al, 1995) and therefore their ratings are inaccurate, however, it is equally plausible that key worker ratings of participant aggression are inaccurate, considering their opinion is subjective and no doubt skewed by the context within which they work. Key workers working in a high aggression setting are likely to have a higher threshold for what they consider aggressive, whilst those in low aggression settings are likely to have a lower threshold. This further highlights the difficulty of using self and other ratings to account for awareness deficits. For these reasons the groups were distinguished by rehabilitation setting and reason for referral. Neurorehabilitation units offering specialist behavioural input, are generally more expensive than units that do not. Therefore when patients no longer need this input they are moved elsewhere. Therefore, individuals with ABI in the specialist behavioural units can be suggested to be actively displaying high levels of aggression, supporting the group selection criteria used in this study.

#### 4.1 Limitations of the Study

Firstly, the use of self report measures to assess levels of emotional empathy, aggression and cognitive empathy may have led to participants reporting a less than accurate, subjective view of themselves. Considering the societal weight placed on caring behaviour as ‘good’, participant awareness that agreeing/disagreeing with certain statements may depict an unfavourable view of them may have produced inaccurate responses. Furthermore, the differing levels of insight often displayed post-ABI suggest the possibility that a proportion of the sample was unable to accurately reflect on complex abstract concepts such as empathy. In addition, use of self report measures assessing emotional and cognitive empathy in ABI populations is commonplace throughout the literature (de Sousa et al 2010a; Williams & Wood, 2010; Wood & Williams, 2008).

Secondly, considering the noncompliance associated with highly aggressive ABI patients, it is worth considering whether the ABI sample was a true representation of individuals who are highly aggressive post-ABI. Between the right to opt out of participation and the lack of capacity to consent exclusion criteria, it is possible that the high aggression group used in this sample did not incorporate those individuals displaying the highest levels of aggression. This was controlled for by separating groups according to neurorehabilitation site and reason for referral. However, it is worth considering that the ABI clients displaying the highest levels of aggression were possibly unable to participate. It could be considered that an objective measure (i.e. participant’s unit aggression records) would be better able to provide group differentiation. However, this too has limitations as different organisations use different scales and participants may display with lower/higher levels of aggression due to the structured context of the unit, and not because they have low/high social cognition.



Furthermore, due to the diverse injury type and lack of neurological data, it was not possible to implicate any neural structures in the current findings. This information would be beneficial allowing indication of specific neurological structures involved with social cognition and aggression.

Additionally, although a rationale for the statistical group differences of ‘years post injury’ and ‘years of education’ was established (see section 2.1.3), these differences were not controlled for within the statistical analysis of the data. Whilst it can be deemed unlikely that the result were affected by these differences, without statistical analysis controlling for these difference it is not possible to be sure. Therefore, a limitation of the study is its failure to account for these differences using a statistical analysis such as a regression analysis or an analysis of covariance.

Finally, due to population type it was not possible to control for medication or the effects this may have on awareness and social cognitive processes. The vast majority of participants were on some form of medication, the side effects of which possibly varied greatly and idiosyncratically. The only form of control available was that any side effects did not prevented participants from completing the test battery.

#### 4.2 Recommendations for future research

The findings of the current study suggest researchers should shift their focus from social cognition onto other explanations of aggressive behaviour post ABI. This would involve considering the variety of factors contributing to aggressive behaviour and investigating the possibility of a multiple factor explanation. Research should investigate the moderate divergent relationship between physical aggression and both intention and affective ToM to establish whether they play a role in mitigating physical aggression.

Future research assessing social cognitive processes should endeavour to assess the components of ToM and empathy instead of their unitary construct. This will enable researchers to establish which components attribute to specific deficits and provide a better understanding of the social cognition. Also research should strive to use more objective measures of aggression, controlling as best as possible for subjective limitations.

Although the use of self-report and verbal measures as a means of assessing high order cognitive processes has been a standard, it is possible that the use of such measures is not appropriate with this population (and perhaps any population) to obtain an accurate and reliable report of empathy. Recent research (de Sousa et al, 2010a; de Sousa et al, 2010b; McDonald et al, 2011 in press) investigating empathy processes in an ABI population has utilised physiological measures (skin conductance levels, facial electromyography) as means of measuring emotional empathy responses. Future research should consider the use of these alternative methodologies alongside, or as opposed to, self report measures.

Over the last decade there has been an abundance of research attempting to understand the neuropsychological processes that, if damaged, would result in a poor socio-behavioural outcome. Despite this, the vast majority of research relies on comparisons of a brain injured population against a 'normal' sample. Rarely do studies investigate these processes within an ABI population. Whilst comparison with a 'normal' population is vital to our understanding of the consequences of ABI, it does little to illuminate the reasons behind socio-behavioural differences commonly displayed within the brain injury population itself. In order to gain a greater understanding of social and behavioural problems post-ABI, future research should focus on comparing behavioural and social differences within the ABI population,

regardless of the proposed causality (neurological, neuropsychological, premorbid personality, genetic, systemic, etc). It is possible that there are multiple causes of aggression post-ABI, however they will not be established through comparison of ABI and 'normal' populations alone.

#### 4.3 Conclusion

Although deficits in social processes may be commonplace post-ABI, their causal role in aggressive behaviour is at present unsupported. Therefore, research should begin to additionally investigate other, possibly multi-factorial, explanations of aggression post-ABI. Research looking to form links between socio-behavioural difficulties post-ABI and neuropsychological concepts is invaluable. It is necessary to inform neurorehabilitation settings of community integration strategies for this complex idiosyncratic client group. Such information will provide a better socio-behavioural outcome in the form of a reduced strain on family systems and a lower re-referral rate.

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## **Part Three**

## **Appendices**

## Appendix A – Reflective statement

The following is a reflective account of my experience of the thesis production process. This statement will evaluate the lessons learnt along the way and those learnt from the process as a whole. I have attempted to keep the statement concise by having a continual narrative of ‘lessons’ running throughout. However, I am aware that my reflection style can be somewhat jumbled and as such I apologise in advance if my reflections appear unclear at points.

There were a few stumbling blocks on the road to completion of my thesis. Compared to other trainee’s in my year group I had no problems that I would consider major (i.e. Research Ethic Committee rejection, etc) and thus I use the term stumbling blocks. However, these stumbling blocks were troublesome and some were larger than others but all have provided lessons that should be reflected upon.

In hindsight (the powerful tool that it is) I feel I should have spent a greater proportion of time establishing the effectiveness of the measures I used. The measures were selected according to their use in previous studies and their apparent ability to assess the social cognitive components being measured. However, as discussed in part one and two, although the face validity of a measure appears strong, the construct validity may not be as strong. I do not think it is fair to suggest that I was careless with my selection of measures as a great deal of thought led to the inclusion of these specific measures. However, I do feel that a great influence behind the selection of my measures was that the uniqueness of the concepts I intended to measure, which left few available tools. This made the selection of measures difficult, especially regarding the use of the new test of ToM. Though unpublished it was the only measure available that clearly defined and assessed each component of ToM and was therefore the most appropriate

measure available. Furthermore, I feel that I may have been overly accepting that because another researcher had used a measure to assess 'x' that I was able to use it to assess 'x' as well. On reflection perhaps a more critical assessment of the measures was required. In fact, I would step beyond this and suggest that it is important to be critical of our ability as researchers to measure such abstract processes such as ToM and empathy accurately.

This brings me onto my next stumbling block, the acquisition of a social constructionist perspective. Over the course of my training I came across the social constructionist perspective and became quite interested in it. This perspective can be said to boast a critical stance on 'taken for granted knowledge', disputing that what we know of the world is gained from its unbiased, objective observation. In this way it opposes empiricism and positivism. Gaining this perspective half way through the completion of my thesis was quite the conundrum. It caused me to question, not only whether we are able to measure neuropsychological processes, but whether they exist in the first place. Where my thesis was concerned, this was (as you can imagine) quite an undermining consideration. It suggested that all I can be said to be investigating is a socially constructed concept, relevant within this culture at this point in time and not a neuropsychological process underlying human nature, as I had originally believed. I recall initially finding it difficult to accept that the project was actually investigating anything and admittedly lost some faith in it for a time. However, on reflection I would have been lucky to have this perspective at the beginning of my thesis journey. What originally appeared to be a second-guessing burden was actually a critical perspective that has allowed me to break down my project on a level above that of the pros and cons of the research but rather on the pros and cons of how we understand and accept processes like empathy, aggression, theory of mind, brain injury. Had I had this



perspective at the early stages of the project it could have allowed me to be more critical of that which I was investigating. Whilst I feel this perspective has been evident within my thesis to quite a low extent, it has allowed me to critically view the work I am producing and how it may add to 'taken for granted knowledge'.

Moving onto a less abstract stumbling block, the process of recruiting was possibly the most difficult stage of the project. This was not so much regarding individual participants but rather arranging recruitment with the brain injury rehabilitation sites. On reflection now and at the time, I recognise that these are very busy work environments and the clinical neuropsychologists whom I hounded, were in high demand and had more important things to focus on than the project of some clinical psychology trainee. Despite this, I recall feeling a deep seated frustration at being unable to move forward with the project because my emails were not being replied to. At the time, I believed this frustration was because my project progress had been hindered, and this was possibly true to an extent. However on reflection, I feel that this frustration may have been because these sites were putting me in a situation that brought up a personal discomfort for me. Whilst reflecting on what was frustrating about this situation I realised that part of it regarded having to email them asking if they had received my previous emails. I came to the conclusion that I did not feel comfortable troubling people who were clearly very busy or were uninterested. Whether this concerned their seniority or an underlying desire on my part not to burden them is unclear and possibly a combination of the two. What was clear was that the idea of badgering the clinical head of a neurorehabilitation unit felt very uncomfortable indeed. I shared this with my supervisor who encouraged me to be firmer in my pursuit of replies to my emails, and I was. On reflection, I feel that this consideration of the appropriateness of my requests combined with this firm, persistent attitude resulted in

the seemingly good relationship I feel was established with all sites. This experience not only gave me an insight into myself, but taught me that to establish an effective research relationship with recruitment sites one must be not only considerately patient but also persistently firm.

Throughout this process lessons have been learnt, not only from the stumbling blocks but also from the areas that have enhanced my project. Of the positive experiences I have encountered during the completion of my thesis, the ones I am most grateful for and feel most reflective about are the people I have met. By investigating the 'ABI' population I feel that I may have, erroneously grouped people into one category and thus, lost the uniqueness of their individuality, brain injury and experience. This notion has built over the course of meeting individuals who have had similar injuries and are yet very different from one another in presentation. Through reflection, I wonder whether the quantitative interpretation of these individuals has possibly led to an inaccurate representation of individuals' post-ABI and I wonder whether qualitative research would be in a better position to address the experiences of people after a brain injury. Despite being on placement in a neurorehabilitation unit during my data collection period, my experience of this field was substantially broadened by visiting these different sites and meeting these individuals. I was moved by the stories of each participant and felt privileged to have had the opportunity to gain a broader understanding of what life is like for individuals following a brain injury. I will undoubtedly carry this experience into my future work, be it research or clinical.

Though deviating from the previous topic, I would like to reflect for a moment on the rationale behind my selection of the journals I plan to submit to. As I am investigating the effect damaged neuropsychological processes may have on behaviour, my first choice of journal for the empirical paper was 'Neuropsychologia'. However, I

was concerned that because of my paper did not investigate the neurological structures underlying hypothesised social cognitive deficit I would not meet their criteria, a concern that was later confirmed by the journal's editor. 'Brain and Cognition' displayed very similar interests to 'Neuropsychologia', but did not chiefly, concern itself with neurological investigation and therefore it felt like an appropriate choice. 'Health Psychology Review' was selected for the systematic literature review, largely because of its interest in review articles, but also for its ability to reach clinicians who work within health settings. I felt it important to publish to a clinical audience as they would be the people working with individuals' post-ABI, and inevitably be influencing service strategy and delivery. These are the first papers I have written for publication, as such I expect my understanding of the journal selection process will develop as I gain a greater experience of submitting to different journals.

Throughout this statement I have reflected on my experience of going through the process of producing a thesis project. I now would like to reflect on what I have learnt about the process itself. An obvious statement though it may be, I feel that research should not be taken lightly and that this is an easy and common mistake. I recall, during my undergraduate, wondering how the limitation sections of articles were so long, and feeling that they could and should have avoided these limitations. The idea that you can avoid all limitations is of course absurd and leads me to the perspective that research will always have flaws, regardless of how much one controls for variable, inevitably it will stumble at points. An important lesson I feel I have taken away regarding the process of research is the importance of getting each stage underway as quickly as possible. I am aware that at times I idly let time pass by, considering I had plenty. However, unexpected time consumers (chasing sites, gaining ethical approval, scoring) delayed my research project and ultimately led to panic. I feel that these unexpected

time consumers are a natural part of the research process and thus staying ahead is important. On reflection, the main thing that I will take away about the production of research is that it is doable. In the beginning the thesis seemed like a gargantuan behemoth, one which was insurmountable. However, by breaking it down into sections and a series of manageable deadlines the beast can be overcome.

Finally, I would like to reflect upon how this process has altered/confirmed my approach to research. I feel that my approach to research has shifted from a purely quantitative perspective to one which is beginning to see the merits of a qualitative approach. I cannot be certain whether this is due to this research process alone or whether external contributors have influenced this shift. It is not that I don't see the merit of quantitative research but rather I see its limitations more clearly. I felt a discomfort towards my project towards the end and this was perhaps the result of my loss of confidence in the ability of quantitative measures to display a true picture. I am however aware that I have much still to learn about both types of research and I have no doubt that my future research endeavours will lead me to utilise both.

The production of this thesis has confirmed the importance of, and encouraged me to, pursue future research projects. Research findings are the core driver of health service development. As researchers we are seen as experts and as such our findings can often be considered fact. It is therefore crucial that accurate, accountable research is being produced. It will be important therefore, to critically appraise the findings of research that influence the development of service strategy. I feel that I would very much like to be part of this process, both the development and critique of future research and service development.

# Appendix B – Author Guidelines for Health Psychology Review

## Instructions for Authors

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There is no formal word limit for manuscripts. Submissions should, however, be as long as necessary and authors of submissions of excessive length which do not convey ideas and points succinctly and concisely will be asked to truncate their manuscript. We also draw authors' attention to the Health Psychology Review online repository of supplemental materials that provides a permanently accessible resource of materials that are too long for the print version of the journal (e.g., oversized tables, intervention protocols, questionnaires etc.) but to which authors of articles appearing in Health Psychology Review wish readers to have access.

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**Tables** should be numbered consecutively with Arabic numbers in order of appearance in the text. Type each table double-spaced on a separate page, with a short descriptive title typed directly above and with essential footnotes below.

### **Competing interests**

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All authors are expected to have made substantive intellectual contributions to, and to have been involved in drafting or revising the manuscript. Each author should have participated sufficiently in the work to take public responsibility for appropriate portions of the content. Acquisition of funding, collection of data, or general supervision of the research group, alone, does not justify authorship. With the submission of a manuscript, it is assumed that all authors have read and approved the final manuscript.

### **Acknowledgements**

All contributors who do not meet the above criteria for authorship, should be listed in an acknowledgements section in accordance with the APA guidelines. The acknowledgements should be contained on the title page of the manuscript as making acknowledgements available to reviewers will compromise the masked peer-review process. Examples of those who might be acknowledged include those who provided general, technical, or writing assistance. Acknowledgement of funding/grants are also included in this section.

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# Appendix C – Author Guidelines for Brain and Cognition

## GUIDE FOR AUTHORS

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It is important that the file be saved in the native format of the wordprocessor used. The text should be in single-column format. Keep the layout of the text as simple as possible. Most formatting codes will be removed and replaced on processing the article. In particular, do not use the wordprocessor's options to justify text or to hyphenate words. However, do use bold face, italics, subscripts, superscripts etc. When preparing tables, if you are using a table grid, use only one grid for each individual table and not a grid for each row. If no grid is used, use tabs, not spaces, to align columns. The electronic text should be prepared in a way very similar to that of conventional manuscripts (see also the Guide to Publishing with Elsevier: <http://www.elsevier.com/guidepublication>). Note that source files of figures, tables and text graphics will be required whether or not you embed your figures in the text. See also the section on Electronic illustrations.

To avoid unnecessary errors you are strongly advised to use the "spell-check" and "grammar-check" functions of your wordprocessor.

### ***Article structure***

*Subdivision - numbered sections*

Divide your article into clearly defined and numbered sections. Subsections should be numbered 1.1 (then 1.1.1, 1.1.2, ...), 1.2, etc. (the abstract is not included in section numbering). Use this numbering also for internal cross-referencing: do not just refer to "the text". Any subsection may be given a brief heading. Each heading should appear on its own separate line.

*Introduction*

State the objectives of the work and provide an adequate background, avoiding a detailed literature survey or a summary of the results.

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Provide sufficient detail to allow the work to be reproduced. Methods already published should be indicated by a reference: only relevant modifications should be described.

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A Theory section should extend, not repeat, the background to the article already dealt with in the Introduction and lay the foundation for further work. In contrast, a Calculation section represents a practical development from a theoretical basis.

*Results*

Results should be clear and concise.

*Discussion*

This should explore the significance of the results of the work, not repeat them. A combined Results and Discussion section is often appropriate. Avoid extensive citations and discussion of published literature.

*Conclusions*

The main conclusions of the study may be presented in a short Conclusions section, which may stand alone or form a subsection of a Discussion or Results and Discussion section.

*Appendices*

If there is more than one appendix, they should be identified as A, B, etc. Formulae and equations in appendices should be given separate numbering: Eq. (A.1), Eq. (A.2), etc.; in a subsequent appendix, Eq. (B.1) and so on. Similarly for tables and figures: Table A.1; Fig. A.1, etc.

#### **Essential title page information**

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A concise and factual abstract is required. The abstract should state briefly the purpose of the research, the principal results and major conclusions. An abstract is often presented separately from the article, so it must be able to stand alone. For this reason, References should be avoided, but if essential, then cite the author(s) and year(s). Also, non-standard or uncommon abbreviations should be avoided, but if essential they must be defined at their first mention in the abstract itself.

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Immediately after the abstract, provide a maximum of 6 keywords, using American spelling and avoiding general and plural terms and multiple concepts (avoid, for example, "and", "of"). Be sparing with abbreviations: only abbreviations firmly established in the field may be eligible. These keywords will be used for indexing purposes.

#### **Abbreviations**

Define abbreviations that are not standard in this field in a footnote to be placed on the first page of the article. Such abbreviations that are unavoidable in the abstract must be defined at their first mention there, as well as in the footnote. Ensure consistency of abbreviations throughout the article.

#### **Acknowledgements**

Collate acknowledgements in a separate section at the end of the article before the references and do not, therefore, include them on the title page, as a footnote to the title or otherwise. List here those individuals who provided help during the research (e.g., providing language help, writing assistance or proof reading the article, etc.).

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### *Reference style*

*Text:* All citations in the text should refer to:

1. *Single author:* the author's name (without initials, unless there is ambiguity) and the year of publication;
2. *Two authors:* both authors' names and the year of publication;
3. *Three or more authors:* first author's name followed by "et al." and the year of publication.

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*List:* References should be arranged first alphabetically and then further sorted chronologically if necessary. More than one reference from the same author(s) in the same year must be identified by the letters "a", "b", "c", etc., placed after the year of publication.

### *Examples:*

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Reference to a book:

Strunk Jr., W., White, E.B., 1979. *The Elements of Style*, third ed. Macmillan, New York.

Reference to a chapter in an edited book:

Mettam, G.R., Adams, L.B., 1999. How to prepare an electronic version of your article, in: Jones, B.S., Smith, R.Z. (Eds.), *Introduction to the Electronic Age*. E-Publishing Inc., New York, pp. 281–304.

### *Journal abbreviations source*

Journal names should be abbreviated according to

Index Medicus journal abbreviations: <http://www.nlm.nih.gov/tsd/serials/lji.html>;



List of title word abbreviations: <http://www.issn.org/2-22661-LTWA-online.php>;  
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  - Telephone and fax numbers
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- Keywords
- All figure captions
- All tables (including title, description, footnotes)

Further considerations

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# **Appendix D – Emails Sent to Researchers to Identify Articles for Review**

## **Email conversation with Prof McDonald**

Dear Prof McDonald,

My name is Paul Walton, I'm a Trainee Clinical Psychologist in my final year of study at the University of Hull. As part of my doctoral thesis, I am currently conducting a systematic literature review exploring the effects of brain injury on experienced emotional empathy. I understand that you have done a substantial amount of research into physiological arousal and emotional mimicry in adults with traumatic brain injury. I am emailing you to ask whether you have any 'in press' articles, or any documents that are not in the public domain that you might be willing to share, for the purpose of my review?

Thank you very much for your time and help.

Yours sincerely,

Paul Walton,

Trainee Clinical Psychologist

Dear Paul,

Thankyou for your email. I am not sure which articles you have but two recent ones are attached

Best wishes

Skye

## **Email conversation with Prof Wood**

Dear Prof Wood,

My name is Paul Walton, I'm a Trainee Clinical Psychologist in my final year of study at the University of Hull. As part of my doctoral thesis, I am currently conducting a systematic literature review exploring the effects of brain injury on experienced emotional empathy. I

understand that you have done a substantial amount of research into emotional empathy deficits in adults with traumatic brain injury. I am emailing you to ask whether you have any 'in press' articles, or any documents that are not in the public domain that you might be willing to share, for the purpose of my review?

Thank you very much for your time and help.

Yours sincerely,

Paul Walton,

Trainee Clinical Psychologist

Paul

Here are some papers which might help. I will ask Claire Williams to send you a pdf of the JCEP paper, which I don't have.

Good luck

Rodger Ll. Wood

## **Email conversation with Dr Shamay-Tsoory**

Dear Dr Shamay-Tsoory,

My name is Paul Walton, I'm a Trainee Clinical Psychologist in my final year of study at the University of Hull. As part of my doctoral thesis, I am currently conducting a systematic literature review exploring the effects of brain injury on experienced emotional empathy. I understand that you have done a substantial amount of research into the neurological basis of cognitive and emotional empathy. I am emailing you to ask whether you have any 'in press' articles, or any documents that are not in the public domain that you might be willing to share, for the purpose of my review?

Thank you very much for your time and help.

Yours sincerely,

Paul Walton,

Trainee Clinical Psychologist

Dear Paul,

Thank you for your interest in my work. I don't have anything in press right now.

Good luck with the review.

Best

simone

### **Other emails that did not receive a reply:**

Dear Arielle de Sousa,

My name is Paul Walton, I'm a Trainee Clinical Psychologist in my final year of study at the University of Hull. As part of my doctoral thesis, I am currently conducting a systematic literature review exploring the effects of brain injury on experienced emotional empathy. I understand that you have done a substantial amount of research into empathy deficits in adults with traumatic brain injury. I am emailing you to ask whether you have any 'in press' articles, or any documents that are not in the public domain that you might be willing to share, for the purpose of my review?

Thank you very much for your time and help.

Yours sincerely,

Paul Walton,

Trainee Clinical Psychologist

# Appendix E – Data Extraction Sheet

**Date of data extraction:**

**Identifying features of the study:**

- Author:
- Article Titles:
- Source (Journal/Year/Vol/Pages/Country of Origin):
- Institutional Affiliation (first Author) and/or contact address:

**Identification of the reviewer:** Paul Walton

**Epistemological Quality**

Definition of Empathy/Emotional Empathy:

Aim of study:

**Group characteristics:**

**Experimental**

**Control**

1. Target population (ABI type)
2. Inclusion Criteria
3. Exclusion Criteria
4. Recruitment procedure
5. Number in group
6. Mean participant information
  - a. Age
  - b. Ethnicity
  - c. PTA
  - d. GCS
  - e. Years since injury
  - f. Age at injury
  - g. gender
  - h. Employment history
  - i. Education history
  - j. medication
  - k. setting (care/rehab/community)
  - l. geographical region
  - m. Neurological damage data provided?
7. Dropout rate:

8. Were ABI group and control group comparable?

**Methodological quality:**

- Design of study (Khan et al 2001):
- Methodological procedures (e.g. quali./qunati. , opportunity sample):
- Test setting:
- Control for medication?:
- Confounding variables:
- Brief procedural description:

**Measures:**

- Measure(s) of experienced emotional empathy (Physiological(P) or Self rater(S)):
- Measures not assessing experienced emotional empathy:
- Stimulus used to evoke emotion (If applicable):
- Examples of questions for self rater:
- Justification for measures used:
- Description of measures used (Y/N):

**Statistical Analysis:**

- Statistical techniques used:
- Does this adjust for confounding? (i.e. Multiple measures):
- Missing data stated?:
- Length of time until follow up:

**Results:**

EEE 1

EEE2

EEE3

EEE4

- Control group mean (S.D.)
- ABI group mean (S.D.)
- Other group 1 mean (S.D.)
- Other group 2 mean (S.D.)

**Findings:**

- Experienced emotional empathy related (Stat Sig?):
  
- Other findings:

**Neurological data:**

- Areas Implicated in experienced emotional empathy from results:
  
- Methods of neurological differentiation between samples (i.e. amygdala damage):
  
- Range of damaged brain regions in ABI group:

**Limitations:**

Highlighted limitations:

Observed limitations:

**Notes:**

## Appendix F – Quality Checklist: Items and Guidance

Question number	Question	Further Info
1	<i>Is the hypothesis/aim/objective of the study clearly described?</i>	
2	<i>Is there a structured summary of experimental design, methods, results and conclusions in the abstract?</i>	
3	<i>Is there a scientific background and explanation of the rationale?</i>	
4	<i>Are the main outcomes to be measured clearly described in the Introduction or Methods section?</i>	If the main outcomes are first mentioned in the Results section, the question should be answered no.
5	<i>Are the characteristics of the patients included in the study clearly described?</i>	In cohort studies and trials, inclusion and/or exclusion criteria should be given. In case-control studies, a case-definition and the source for controls should be given.
6	<i>Is the design and procedure clearly explained to a degree that would allow its repetition?</i>	
7	<i>Are the independent and dependent variables of interest clearly described?</i>	Treatments and placebo (where relevant) that are to be compared should be clearly described.
8	<i>Are the distributions of confounding variables in each group of subjects to be compared clearly described?</i>	A list of principal confounders is provided.
9	<i>Are the main findings of the study clearly described?</i>	Simple outcome data (including denominators and numerators) should be reported for all major findings so that the reader can check the major analyses and conclusions. (This question does not cover statistical tests which are considered below).
10	<i>Does the study provide estimates of the random variability in the data for the main outcomes?</i>	In non normally distributed data the inter-quartile range of results should be reported. In normally distributed data the standard error, standard deviation or confidence intervals should be reported. If the distribution of the data is not described, it must be assumed that the estimates used were appropriate and the question should be answered yes.
11	<i>Have actual probability values been reported ( e.g. 0.035 rather than &lt;0.05) for the main outcomes except where the probability value is less than 0.001?</i>	
12	<i>Were those subjects who participated in the study representative of the entire population from which they were recruited?</i>	The study must identify the source population for patients and describe how the patients were selected. Patients would be representative if they comprised the entire source population, an unselected sample of consecutive patients, or a random sample. Random sampling is only feasible where a list of all members of the relevant population exists. Where a study does not report the proportion of the source population from which the patients are derived, the question should be answered as unable to determine.
13	<i>Were inclusion and exclusion criteria for all participants clearly defined and</i>	



	<i>adhered too?</i>	
14	<i>Did the study report the setting in which data was collected?</i>	
15	<i>Are the limitations of the study critically discussed?</i>	
16	<i>Were the measures used ecologically valid?</i>	Did the measures administered require participants to perform tasks they would come across in day to day life?
17	<i>Was an attempt made to blind study subjects to the purpose of the study?</i>	For studies where the patients would have no way of knowing which intervention they received, this should be answered yes.
18	<i>If any of the results of the study were based on "data dredging", was this made clear?</i>	Any analyses that had not been planned at the outset of the study should be clearly indicated. If no retrospective unplanned subgroup analyses were reported, then answer yes.
19	<i>Were the statistical tests used to assess the main outcomes appropriate?</i>	The statistical techniques used must be appropriate to the data. For example nonparametric methods should be used for small sample sizes. Where little statistical analysis has been undertaken but where there is no evidence of bias, the question should be answered yes. If the distribution of the data (normal or not) is not described it must be assumed that the estimates used were appropriate and the question should be answered yes.
20	<i>Were the main outcome measures used accurate (valid and reliable)?</i>	For studies where the outcome measures are clearly described, the question should be answered yes. For studies which refer to other work or that demonstrates the outcome measures are accurate, the question should be answered as yes.
21	<i>Was there adequate adjustment for confounding variables in the analyses from which the main findings were drawn?</i>	This question should be answered no for trials if: the distribution of known confounders in the different treatment groups was not described; or the distribution of known confounders differed between the treatment groups but was not taken into account in the analyses. In nonrandomised studies if the effect of the main confounders was not investigated or confounding was demonstrated but no adjustment was made in the final analyses the question should be answered as no.
22	<i>Did the study recruit enough participants to have sufficient power to detect a statistically important effect?</i>	Is the number of participants required reported and if so is it matched or exceeded?

# **Appendix G – Quality Checklist Scores**

## Appendix G – Quality Checklist Scores

	Quality Checklist Item Number																						Total
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	
de Sousa et al, 2010a	1	1	1	1	1	1	1	2	1	1	1	0	0	1	1	0	1	1	1	1	1	0	19
Shamay-Tsoory et al, 2009	1	1	1	1	1	0	1	1	1	1	1	1	0	0	1	0	1	1	1	1	1	0	17
Williams & Wood, 2010	1	1	1	1	1	1	1	2	1	1	0	1	1	1	1	0	0	1	1	0	1	0	18
Shamay-Tsoory et al, 2004	0	0	1	1	0	0	1	1	1	1	0	1	1	0	0	0	0	1	1	1	0	0	11
McDonald et al, 2011b	1	1	1	1	1	0	0	2	1	1	1	1	1	0	1	0	1	1	1	1	1	0	18
McDonald et al, 2011a	1	1	1	1	1	1	1	2	1	1	0	1	1	1	1	0	0	1	1	1	1	0	19
Muller et al, 2009	1	1	1	1	1	1	1	1	1	1	0	1	0	0	1	0	0	1	1	0	1	0	15
Wood & Williams, 2008	1	1	1	1	1	1	1	2	1	1	0	1	1	1	1	0	0	1	1	1	1	1	20
Hopkins et al, 2002	1	1	1	1	0	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	0	16
de Sousa et al, 2010b	1	1	1	1	1	1	1	2	1	1	1	1	1	0	0	0	1	1	1	1	1	0	19

## Appendix H – Measures of experienced emotional empathy (from part one)

The descriptions of the measures have been obtained from the article that featured them in the review. References are linked to part one reference section

Balanced Emotional Empathy Scale (BEES, Mehrabian, 2000)	A measure of emotional empathy, the ability to experience the feelings of another. It consists of 15 negatively worded questions and 15 positively worded questions and utilises a 9 point liker scale, ranging from -4 to +4.
Ekman & Friesen (1976) emotional facial expression stimuli	Greyscale pictures of faces depicting the expression of various emotions. Pictures use male and female actors and emotions are expressed to varying degrees
Electrodermal response (EDA) and Skin Conductance Response (SCR)	A physiological measure of electrical skin conductance often recording the largest voltage produced over a specified time period. Used in conjunction with presentation of stimuli.
Empathy Quotient (EQ, Baron-Cohen & Wheelwright, 2004)	<i>Emotional Reactivity Subscale</i> – A 22 item measure of emotional empathy adapted from the EQ.
Evoked Cardiac Deceleration (ECD)	A physiological measure assessing the reduction in heart rate associated with the end of heightened sensory intake, post evoked cardiac response. Used in conjunction with presentation of stimuli.
Facial Electromyography (EMG)	A physiological measure used to record changes in facial muscle activity. Used in conjunction with presentation of stimuli.
Interpersonal Reactivity Scale (IRI, Davis, 1980)	<i>Empathic Concern</i> – A 7 item scale assessing the how frequently an individual feels empathy for others. <i>Personal Distress</i> – A 7 item scale assessing the feelings of discomfort resulting from tense interpersonal settings.
Questionnaire Measure of Emotional Empathy (QMEE, Mehrabian & Epstein, 1972)	The QMEE was designed to assess the chronic tendency to react emotionally to emotional to the emotional experiences of others. Scores range from -132 to +132.
Skin Conductance Level (SCL)	A physiological measure of longer lasting levels of electrodermal reactivity associated with tonic levels of arousal. Used in conjunction with presentation of stimuli.

## Appendix I – Other measures (from part one)

The descriptions of the measures have been obtained from the article that featured them in the review. References are linked to part one reference section

Alternate Uses Test (Lezak, 1995)	A measure of cognitive flexibility
The Arousal Scale (de Sousa et al 2010b)	A self rating index of arousal from calm (1) to very excited (9)
The Beck Depression Inventory (BDI, Beck, 1987)	A measure of depression
The Beck Anxiety Inventory (BAI; Beck, Epstein, Brown & Steer, 1988)	A measure of anxiety
Benton Face Recognition Test (BFRT; Benton, Hamsher & Varney, 1983)	A measure of the ability to match faces based on their features
The Brixton Test (Burgess & Shallice, 1997)	A measure of rule detection
Brock Adaptive Functioning Questionnaire (BAFQ, Dywan, Roden & Murphy, 1995; Dywan & Segalowitz, 1996)	A measure consisting of 12 scale assessing: Planning, Initiation, Flexibility, Compulsiveness, Attention, Memory, Arousal, Emotionality, Impulse Control, Aggressiveness, Social Monitoring and Empathy. It is also able to measure self-awareness.
California Verbal Learning Task (CVLT; Delis, Freeland, Kramer & Kaplan, 1988)	A measure of verbal memory
Character Intention Task (Brunet, Sarfati, Hardy-Bayle & Decety, 2000).	A ToM task assessing the ability to understand intention of others
The Culture Fair Test (CFT; Cattell & Cattell, 1960).	Used to assess general non-verbal problem solving abilities
Depression, Anxiety and Stress Scale (DASS (Lovibond & Lovibond, 1995)	A measure of depression, anxiety and stress
Design Fluency (Jones-Gotman & Milner, 1977)	A measure of cognitive flexibility
Emotional Matching Task (used faces from Ekman & Friesen, 1976)	A measure of the ability to identify emotional expressions presented by face from four other emotionally expressive faces
Emotional Recognition Task (used content from Ekman & Friesen, 1976; Baron-Cohen, Wheelwright, Hill, Raste & Plumb, 2001)	A task to assess the ability to recognise emotional expressions in a series of faces.
Expression Identification Test (used faces from Ekman & Friesen, 1976)	A measure of the ability to identify emotional expressions presented by face.

	The maximum score was 60.
Faux Pas Test (8 items from the Adult version of the Faux Pas Recognition Test (Stone et al, 1998))	A ToM measure assessing the ability to recognise a faux pas.
First-order and Second-order False Belief Task (Frith & Corcoran, 1996; Bach, Happe, Fleming & Powell, 1998; Rowe, Bullock, Polkey & Morris, 2001)	A ToM measure designed to assess the ability to understand the first and second order beliefs of others
The Hayling Test (Burgess & Shallice, 1997)	A measure of response initiation speed and response suppression
The International Affective Picture System (IAPS; Centre for the Study of Emotion and Attention, 1999)	A catalogue of pleasant, unpleasant and neutral visual images demonstrated to evoke physiological arousal.
Interpersonal Reactivity Scale (IRI, Davis, 1980)	<i>Perspective Taking</i> – A 7 item subscale measuring the ability to adopt the psychological view point of another. The score ranges from <i>Fantasy Scale</i> – A 7 item subscale measures the ability to transpose oneself into fictional situations using ones imagination. The score ranges from
Interpretation of Indirect Speech Act Task (Joanette, Ska & Cote, 2004)	Part of the Montreal Evaluation of Communication Protocol that assesses how people identify indirect speech.
National Adult Reading Test (NART-2; No author is provided by Wood & Williams, 2008)	A measure of premorbid intellectual functioning
Ravens Progressive Matrices (Beaumont & Davidoff, 1992)	Assess reasoning and provides an estimate of general intellectual functioning
Reading the Mind in the Eyes (Baron-Cohen et al, 2001)	A ToM task used to assess the ability to someone's mental state from their eyes
Recognition of Affective Prosody - Hebrew Version (Lapidot, Most, Pik & Schnider, 1998)	A measure of emotion identification adapted from original (Ross, Thompson & Yenkosky, 1997) into Hebrew. Participants listen to a recorded sentence and are to identify the emotion present in the voice
Recognition of Facial Expression (used Ekman & Friesen, 1976)	A measure of emotion identification consisting of 35 pictures depicting 1 of 7 emotional states (anger, neural, sadness, disgust, happiness, surprise, and fear)
Semantic and Formal Lexical Evocation (Adapted from Cardebat, Doyon, Puel, Goulet & Joanette, 1990)	A measure of verbal fluency
Second-order ToM Task (No Author provided by Shamay-Tsoory et al 2009)	A ToM task assessing the ability to understand what someone else thinks about what someone else thinks

Stroop Color Word Test (Stroop, 1935)	A measure of response inhibition/interference
Toronto Alexthymia Scale (TAS-20; Bagby Parker & Taylor, 1994)	A 20 item measure used in the diagnosis of alexthymia
Torrance Test of Creative Thinking (The circles sub-scale) (Torrance, 1974)	A measure of cognitive flexibility
Trail Making Test A and B (Reitan & Wolfson, 1993)	A measure of mental flexibility
The Valence Scale (de Sousa et al 2010b)	A self rating index of mood from negative (1) to positive (9)
Verbal Fluency (Author not provided by Shamay-Tsoory et al 2004)	A measure of cognitive flexibility
Wechsler Adult Intelligence Scale – Revised (WAIS-R, Wechsler, 1987)	A measure of intelligence and working memory. <i>Vocabulary Subtest</i> – An assessment of general verbal intelligence <i>Digit Span Subtest</i> – An assessment of attention span <i>Similarities Subtest</i> – An assessment of verbal reasoning
Wechsler Adult Intelligence Scale Third Edition (WAIS-III; Wechsler, 1997)	<i>Vocabulary, Similarities and Comprehension</i> – Subtests assessing verbal ability <i>Digit Span, Letter-Number Sequencing and Spatial Span</i> – Subtests assessing working memory <i>Block Design, Matrix Reasoning, Letter-Number Sequencing and Picture Arrangement</i> – Subtests measuring cognitive flexibility
The Wisconsin Card Sorting Test (WCST; Heaton, Chelune & Talley, 1993)	A measure of cognitive flexibility
The Zoo Map Test (Wilson, Alderman, Burgess, Emslie & Evans, 1996)	A measure of planning ability from the behavioural assessment of dysexecutive syndrome.

# Appendix J – Research Ethics Committee Approval

## Leeds (West) Research Ethics Committee

First Floor  
Millside  
Mill Pond Lane  
Leeds  
LS6 4EP

Telephone: 0113 3050122  
Facsimile:

17 August 2010

Mr Paul Walton  
Trainee Clinical Psychologist  
Department of Clinical Psychology and Psychological Therapies  
Hertford Building  
The University of Hull  
Cottingham Road  
Hull  
HU6 7RX

Dear Mr Walton

**Study Title:** Social Cognition in Brain Injury: The Role of Theory of Mind and Empathy in Behavioural Disorders  
**REC reference number:** 10/H1307/88

Thank you for your letter of 15 July 2010, responding to the Committee's request for further information on the above research [and submitting revised documentation](#).

The further information has been considered on behalf of the Committee by the [Chair](#).

### Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation [as revised](#), subject to the conditions specified below.

### Ethical review of research sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

### Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start



of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

For NHS research sites only, management permission for research (“R&D approval”) should be obtained from the relevant care organisation(s) in accordance with NHS research governance arrangements. Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at <http://www.rdforum.nhs.uk>.

*Where the only involvement of the NHS organisation is as a Participant Identification Centre (PIC), management permission for research is not required but the R&D office should be notified of the study and agree to the organisation’s involvement. Guidance on procedures for PICs is available in IRAS. Further advice should be sought from the R&D office where necessary.*

*Sponsors are not required to notify the Committee of approvals from host organisations.*

**It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).**

### **Approved documents**

The final list of documents reviewed and approved by the Committee is as follows:

<i>Document</i>	<i>Version</i>	<i>Date</i>	
Investigator CV		29 April 2010	
Protocol	3	18 December 2009	
Protocol	4	15 July 2010	
REC application		07 May 2010	
Covering Letter		05 May 2010	
Participant Information Sheet: Key worker	1	15 July 2010	
Response to Request for Further Information		15 July 2010	
Participant Information Sheet	3	15 July 2010	
Participant Consent Form: Key worker	1	15 July 2010	
Participant Consent Form	2	05 May 2010	
CV - Catherine Derbyshire			
Referees or other scientific critique report		08 January 2010	

### **Statement of compliance**

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

### **After ethical review**

Now that you have completed the application process please visit the National Research Ethics Service website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document "*After ethical review – guidance for researchers*" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email [referencegroup@nres.npsa.nhs.uk](mailto:referencegroup@nres.npsa.nhs.uk).

<b>10/H1307/88</b>	<b>Please quote this number on all correspondence</b>
--------------------	---

Yours sincerely

**Dr Rhona Bratt  
Chair**

Email: [Elaine.hazell@leedsth.nhs.uk](mailto:Elaine.hazell@leedsth.nhs.uk)

*Enclosures:* "After ethical review – guidance for researchers"

*Copy to:* *Mr Stephen Walker*

# Appendix K – Brain Injury Rehabilitation Trust Ethics Approval



Founded in 1979  
Patron:  
Her Grace  
The Duchess of  
Northumberland

Life President:  
Stephen E. Love MA

Life Vice-Presidents:  
Norman C Thody

Barbara Besant-Hutchins  
Graham Anderson

## STRICTLY PRIVATE AND CONFIDENTIAL

Mr Paul Walton  
Department of Clinical Psychology and  
Psychological Therapies  
The Hertford Building  
The University of Hull  
Cottingham Road  
Hull  
HU6 7RX

29<sup>th</sup> November 2010

Dear Mr Walton,

### **Research Proposal: Social Cognition in Brain Injury: The Role of Theory of Mind and Empathy in Behavioural Disorders**

Thank you for providing detail of the above proposal for scrutiny by a panel of our  
Ethics Committee.

I am pleased to confirm that the panel supports your proposal, and wishes you  
every success with your research.

Yours sincerely,

Iain Mackrory-Jamieson  
Company Secretary

Direct Tel: 01444 237297  
E-mail: [Iain.Mackrory-Jamieson@TheDTGroup.org](mailto:Iain.Mackrory-Jamieson@TheDTGroup.org)

cc Prof Mike Oddy




INCORPORATING:  
The Brain Injury  
Rehabilitation Trust  
Hamilton Lodge Trust

The Disabilities Trust is a company limited by guarantee incorporated in England and Wales under  
registration number 2334589 and registered as a charity in England and Wales under registration number  
800797 and in Scotland under registration number SC038972.  
Registered office as shown.

The Disabilities Trust  
32 Market Place  
Burgess Hill, West Sussex RH15 9NP  
Telephone: 01444 239123  
Fax: 01444 244978  
Email: [info@thetdgroup.org](mailto:info@thetdgroup.org)  
Internet: [www.thetdgroup.org](http://www.thetdgroup.org)

# Appendix L – Northern Lincolnshire and Goole Hospitals R&D Ethics Approval



Northern Lincolnshire and Goole Hospitals   
NHS Foundation Trust

Scunthorpe General Hospital  
Cliff Gardens  
Scunthorpe  
North Lincolnshire  
DN15 7BH

Research & Development Department  
Tel: 01724 290410  
Fax: 01724 387869

Tel: 01724 282282  
www.nlg.nhs.uk

12<sup>th</sup> October 2010

Mr Paul Walton  
Trainee Clinical Psychologist  
Department of Clinical Psychology and Psychological Therapies  
Hertford Building  
University of Hull  
Cottingham  
Hull  
HU6 7RX

Dear Mr Walton

**Re: Social Cognition in Brain Injury: The Role of Theory of Mind and Empathy in Behavioural Disorders**

Thank you for submitting the protocol for the above study to the Northern Lincolnshire & Goole Hospitals NHS Foundation Trust Research & Development Department, for Research Governance compliance check.

The Trust Research & Development Department grant approval for your study to commence and a 'Letter of Access' has been sent to you. You are required to inform the Trust Research & Development Department in advance of any significant changes to the original protocol or issues of safety. In addition the Northern Lincolnshire & Goole Hospitals NHS Foundation Trust Research & Development Department will require an end of study notification.

Should you require any further assistance regarding this study, please do not hesitate to contact me.

Wishing you every success with your study.

Kind regards

Mr Jim Bold  
Assistant to the Medical Director  
Northern Lincolnshire & Goole  
Hospitals NHS Foundation Trust

Family Services Division - Trust wide



INVESTOR IN PEOPLE



# Appendix M – St Andrew’s Healthcare Honorary Contract

## ST ANDREW’S HEALTHCARE Honorary

### CONTRACT & PRINCIPAL STATEMENT OF EMPLOYMENT PARTICULARS

<b>NAME (in full):</b>	Paul Walton
<b>APPOINTMENT:</b>	Post: Researcher
<b>NAME OF APPOINTING ORGANISATION:</b>	St Andrew’s Healthcare (“St Andrew’s”)
<b>FUNDING BODY / PRIMARY EMPLOYER:</b>	University of Hull
<b>PLACE OF WORK:</b>	Your normal place of work is at St Andrew’s Hospital, Northampton or such other premises of St Andrew’s which St Andrew’s may reasonably require.
<b>HEAD OFFICE:</b>	The Head Office is presently sited at St. Andrew’s Hospital, Billing Road, Northampton, NN1 5DG
<b>STARTING DATE:</b>	<b>1<sup>st</sup> November 2010</b>
<b>TERMINATION DATE:</b>	<b>31<sup>st</sup> October 2011</b>
<b>PROBATIONARY PERIOD:</b>	This post is subject to a 6 month probationary period. You are required to sign a Probationary Declaration that forms part of the terms and conditions of your contract of employment. This Declaration confirms that during your probationary period St Andrew’s will not be bound by the terms of the Disciplinary Policy.
<b>PAY:</b>	You are not an employee of St Andrew’s and therefore this post is not remunerated.
<b>HOURS OF DUTY:</b>	Your normal hours of duty will be agreed by the Registered Manager.
<b>ANNUAL LEAVE:</b>	<p>This contract carries no entitlement to paid holidays or bank holidays. If, however, you wish to take time off, please speak with the nominated Consultant supervisor.</p> <p>You will take annual leave according to your main employment contract elsewhere, you are required to advise your nominated Consultant Supervisor your leave dates with as much notice as possible.</p>



<b>ACCOUNTABILITY:</b>	You are accountable to the Registered Manager, via a nominated Consultant supervisor.
<b>SICKNESS:</b>	Your Consultant supervisor, as Line Manager, must be informed as early in the day as possible on the first day of absence. You do not receive any sick pay.
<b>TERMS AND CONDITIONS OF EMPLOYMENT:</b>	The terms and conditions of this appointment are contained within this Contract (although may from time to time be amended by agreement between yourself and the Registered Manager). This Contract takes precedence over any other document provided to you. Other non-contractual terms are contained within the relevant policies or agreements. For the avoidance of doubt, you are not an employee of St Andrew's.
<b>WORK PERMITS:</b>	Individuals required to have a Work Permit must also obtain Leave to Remain in line with their Work Permit before commencement. The appropriate documentation must be sent to the Recruitment Department.
<b>LEGAL RIGHT TO WORK IN THE UK:</b>	All staff will be asked to produce documents to verify their legal right to work in the UK. All renewals / extensions of stay must be brought to the HR / Recruitment Department to record. All documents submitted in accordance with the Home Office Regulations will be copied and retained on file and may be required to be submitted to Home Office Officials for verification or confirmation of your legal right to work in the UK.
<b>WORKING OUTSIDE THE UK:</b>	There are no requirements for working abroad.
<b>TERMINATION OF CONTRACT:</b>	At any time prior to the date of termination referred to above, this appointment can be terminated by mutual agreement or by either party giving a minimum of 1 month's notice or, in the event of any breach of this Contract by you, at any time without notice.
<b>CONFIDENTIALITY:</b>	It is a condition of your contract that you shall not, either during the continuance of your appointment or after your contract has ended, without the prior consent in writing of St Andrew's, divulge to any person, firm, company, hospital or other organisation or use or exploit, except for the benefit of St Andrew's (and any part of it), business affairs or confidential information concerning patients and staff. Any breaches of confidentiality may result in this appointment being terminated. Any exception to this must be in line with the Public Interest Disclosure ("Whistleblowing") Policy. St Andrew's follows General Medical Council and Caldicott guidelines on clinical confidentiality.
<b>OWNERSHIP OF DOCUMENTS:</b>	When requested, and in any event, upon the termination of this appointment, howsoever occurring, you will be required to deliver up immediately and promptly to the Registered Manager, all lists of



clients or patients, correspondence and all other documents, papers and records, together with any other property of St Andrew's which may have been prepared by you or have come into your possession in the course of your contract with St Andrew's. You will not be entitled to and shall not retain any copies thereof. Any exception to this must be in line with the Public Interest Disclosure ("Whistleblowing") Policy.

**CRIMINAL RECORDS BUREAU:**

You are required to immediately notify your Line Manager, the Medical Director and the Director of Human Resources of any Police convictions, charges, cautions, reprimands or warnings that you receive during the currency of your contract with St Andrew's. If you fail to disclose these immediately, any subsequent discovery of such offences may result in this contract being terminated.

**PROFESSIONAL REGISTRATION:**

Qualified clinicians are required to maintain their registration with respective Professional Bodies and to notify the Medical Director and Director of Human Resources of any changes. Failure to do so may result in this contract being terminated. Evidence of periodic registration in the form of an annual certificate must be given to your Registered Manager.

**PROFESSIONAL DEFENCE ORGANISATION:**

You are indemnified by St Andrew's for clinical activities during the course of this appointment in relation to acts or omissions carried out in the course of your duties on behalf of St Andrew's. Nevertheless you are advised to consider being a member of a recognised professional defence organisation to cover "good Samaritan" incidents, and other contingencies.

**EQUAL OPPORTUNITIES:**

St Andrew's Healthcare is committed to the promotion of Equality of Opportunity and by its Equal Opportunities Policies aims to ensure that no-one associated with St Andrew's receives less favourable treatment on the grounds of gender, colour, age, family responsibilities, gender reassignment, national extraction, social origin, pregnancy, sex, sexual orientation, transsexualism, religion, political conviction, on the grounds that they are a member or non-member of a trade union, race, marital or civil partnership status, disability or any other unjustifiable criteria through the recruitment process, in training and promotion. You will be expected to share this commitment.

**ST ANDREW'S KEYS:**

St Andrew's cares for some severely ill patients. Strict key control is essential and it is a condition of this appointment that you take the greatest care not to lose or otherwise compromise keys issued to you for use at St Andrew's. The loss of any St Andrew's key is to be reported to the Duty Security Officer as soon as the loss is discovered. Any failure to do so may result in this contract being terminated. To have a St Andrew's key copied other than via the Head of Security may result in this contract being terminated.

**PERSONAL**

Those who are issued with a PIT are personally responsible for its



**INFRARED TRANSMITTER (PIT):**

safe keeping at all times. Security and safe keeping of a PIT is essential and it is a condition of employment that staff take the greatest care not to lose or otherwise compromise PITs issued to them for use at St Andrew's Healthcare. The loss of a PIT is to be reported to your Consultant Supervisor immediately. Failure to do so may result in this contract being terminated. In the event of loss deemed to be through carelessness, you may be required to reimburse St Andrew's for the full replacement cost.

**USE OF ST ANDREW'S PROPERTY:**

Under no circumstances should St Andrew's property be removed from the premises without seeking prior permission from the appropriate Registered Manager or Executive Director.

**USE OF COMPUTERS:**

Under no circumstances should external computer disks be used on any St Andrew's computer system nor should any downloading of data from the Internet take place without the express permission of the Systems Department in accordance with their security system. Viewing, storing or distributing inappropriate files or data from the Internet is viewed as gross misconduct.

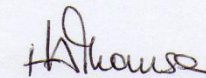
**LOSS OF PERSONAL BELONGINGS:**

St Andrew's cannot accept liability for the loss of, or damage to, personal belongings (including bicycles and motor vehicles) on St Andrew's premises, whether by fire or theft, with the exception of money or other valuables that have been handed over to a duly authorised officer of St Andrew's for safe custody, and for which a receipt has been given. You are advised to cover yourself by insurance so far as you think appropriate.

**DATA PROTECTION:**

You consent to the holding and processing by St Andrew's of personal data (including, where appropriate, sensitive personal data) relating to you for the purposes of personnel or pensions administration, employee management or compliance with any laws or regulations applicable to St Andrew's or its business.

Signed by the Registered Manager on behalf of St Andrew's Healthcare:




Date:

7/10/10

I acknowledge receipt of my Honorary Contract and accept the appointment on the terms and conditions set out. I am aware that any false or misleading information given by me in my application, may affect this contract.

Signature:



Date:

19/11/10



# Appendix N – Participant Information Sheet (Version 3)

## Participant Information Sheet

### Study Title

The impact of brain injury on social cognition and aggression

We would like to invite you to take part in our research study. Before you decide we would like you to understand why the research is being done and what it would involve for you. Please take time to read the following information carefully. Talk to other people about the study if you wish. One of our team will also go through the information sheet with you and answer any questions you have.

Part 1 tells you the purpose of this study and what will happen to you if you take part.

Part 2 gives you more detailed information about the conduct of the study.

Ask us if there is anything that is not clear, or if you have any questions. Please take your time in deciding if you would like to take part.

### PART 1

#### What is the Purpose of the Study?

The purpose of the research is to investigate the effect that brain injury can have on social thinking, and whether difficulties with social thinking can lead to aggression. By ‘social thinking’, or ‘social cognition’ we mean the ways people can understand someone else’s point of view and how the other person reached it. Some people who have experienced a brain injury find it difficult to control aggressive outbursts. We are interested in investigating suspected links between this and social cognition.

It is hoped that by doing this research we may gain greater understanding into the consequences of brain injury, its impact on social cognition and whether it can cause aggressive behaviour.

#### Why have I been invited?

You have been invited to take part in the study as we are looking at how people like yourself, who have suffered a brain injury, are at social thinking. We will be involving people who experience difficulties regarding aggression and those who do not. This will allow us to determine any links between social cognition and aggression. The care manager of the home was contacted by us and we asked him/her whether there was anyone in their care who might be appropriate to invite. He/She told us that you might be appropriate and that we could invite you.

### Do I have to take part?

It is up to you to decide to join the study. We will describe the study and go through this information sheet. If you agree to take part, we will then ask you to sign a consent form. You are free to withdraw at any time, without giving a reason. This would not affect the standard of care you receive.

### What will happen to me if I take part?

If you agree to take part you will be asked to complete 7 questionnaires which together will take about 1 ½ hours. The questionnaires will look at social thinking, mood and problem solving and would be done in private. The questionnaires are designed to look at people's strengths and weaknesses, so you may find some parts easy and some parts more difficult. We would only meet once and complete all the questionnaires during this visit. Breaks would be available during this visit whenever you feel they were needed. At the end of the questionnaires you will be given an opportunity to ask any questions and discuss any concerns you may have.

As well as this we would need to look in your case file to find out the type of brain injury you suffered and learn which areas of your brain were injured.

Finally I'll ask your key worker to participate in the study and fill in one questionnaire similar to those that you have been doing.

Here is a brief outline of the questionnaires you will be asked to complete:

- Hospital Anxiety and Depression Scale (HADS)  
The HADS is a 14 item questionnaire for measuring levels of anxiety and depression. It takes roughly 2-5 minutes to complete. Each question will have 4 answers to choose from. You should pick the one that feels most relevant to you.
- Aggression Questionnaire (AQ)  
This 29 items questionnaire will require you to rate each statement depending on how characteristic it is of you.
- Wechsler Abbreviated Scale of Intelligence (WASI)  
The WASI is a brief measure of intelligence taking approximately 30 minutes to complete. It will require you to perform some problem solving tasks, some of which will be timed.
- The new test of Theory of Mind  
This task will require you to watch a video of several situations and tell me if someone has said or done something they shouldn't have
- Empathy Quotient (EQ)  
This empathy questionnaire consists of 60 statements and will ask you to say how much you agree with each one
- Balanced Emotional Empathy Scale (BEES)  
The BEES is an emotional empathy questionnaire consisting of 30 statements. You will be required to say how much you agree with each.

What are the disadvantages of taking part?

Some people may find it difficult to concentrate for long periods of time. We are able to take breaks to help if you find this is the case. It is also important to remember that everyone will find some parts easier than others, and the researcher will be with you throughout to answer any questions.

We will allow some time at the end to talk through any concerns you have and to answer any questions. We will also give you contact information in case you have any other questions later on.

What are the possible benefits of taking part?

We cannot promise the study will help you but the information we get from this study will help improve the understanding of brain injury and therefore improve treatment of people who have suffered a brain injury.

What happens when the research study stops?

Once all the participants have been seen and the research has been completed we will write to you with a summary of the results.

What if there is a problem?

Any complaint about the way you have been dealt with during the study or any possible harm you might suffer will be addressed. The detailed information on this is given in Part 2.

Will my taking part in the study be kept confidential?

Yes. We will follow ethical and legal practice and all information about you will be handled in confidence. The details are included in Part 2.

If the information in Part 1 has interested you and you are considering participation, please read the additional information in Part 2 before making any decision.

PART 2

What will happen if I don't want to carry out the study?

You are free to withdraw from the study at any point. This will not affect the care you receive in any way. If you withdraw from the study we will ask you whether you would like the information collected up till that point to be included in the study, or if you would like it to be destroyed.

What if there is a problem?

If you have a concern about any aspect of this study, you should ask to speak to the researcher who will do their best to answer your questions [see contact details for Paul Walton]. You can also contact the supervisor of the study (Paul Walton) or talk to

a member of your care team. If you remain unhappy you can complain formally, the researcher is obliged to provide these details when requested.

Will my taking part in this study be kept confidential?

All information which is collected during the course of the research will be kept strictly confidential. Your information will be seen by the researcher only. All documentation will be kept in a locked filing cabinet. The information you provide will be identified by number so everything will remain anonymous and completely confidential. Your data will be held for up to 5 years after the study has ended and then destroyed. All data will be managed in line with the Data Protection Act.

There are occasions when confidentiality must be broken. If you disclose information indicating that you or others are at risk of harm it will be necessary to inform the appropriate authorities.

Data will be collated in a computer database using codes to identify individuals. All files will be password protected.

What will happen to the results of the study?

We intend to publish the results of this study. You will not be identified in any publication.

Once all the participants have been seen and the research has been completed we will write to you with a summary of the results.

Who is organising and funding the research?

This study is being organised by The University of Hull and funded by the Humber NHS Foundation Trust.

Who has reviewed the study?

The research is being supervised and monitored by the Dept of Clinical Psychology and Psychological Therapies at the University of Hull. In addition all research in healthcare settings are looked at by an independent group of people, called a Research Ethics Committee, to protect your interests. This study has been reviewed and given favourable opinion by Leeds (West) Research Ethics Committee.

Further information

I am able to provide further information should you require it. I can be contacted via the following:

Address: Mr Paul Walton  
Department of Clinical Psychology and Psychological Therapies  
Hertford Building  
University of Hull  
Cottingham Road  
Hull  
HU6 7RX  
Telephone: 01482 464 106

# Appendix O – Participant Information Sheet (Key Worker, Version 1)

## Participant Information Sheet (Key Worker)

### Study Title

The impact of brain injury on social cognition and aggression

We would like to invite you to take part in our research study. Before you decide we would like you to understand why the research is being done and what it would involve for you. Please take time to read the following information carefully. Talk to other people about the study if you wish. One of our team will also go through the information sheet with you and answer any questions you have.

Part 1 tells you the purpose of this study and what will happen to you if you take part.

Part 2 gives you more detailed information about the conduct of the study.

Ask us if there is anything that is not clear, or if you have any questions. Please take your time in deciding if you would like to take part.

### PART 1

#### What is the Purpose of the Study?

The purpose of the research is to investigate the effect that brain injury can have on social thinking, and whether difficulties with social thinking can lead to aggression. By 'social thinking', or 'social cognition' we mean the ways people can understand someone else's point of view and how the other person reached it. Some people who have experienced a brain injury find it difficult to control aggressive outbursts. We are interested in investigating suspected links between this and social cognition.

It is hoped that by doing this research we may gain greater understanding into the consequences of brain injury, its impact on social cognition and whether it can cause aggression.

#### Why have I been invited?

You have been invited to take part in the study as we are looking at how people, who have suffered a brain injury, are at social thinking. A client who you are the key worker for has agreed to participate in this study. As part of this study we require information regarding their behaviour from you, their key worker. We will be involving people who experience difficulties regarding aggression and those who do not. Therefore your client may or may not display aggressive behaviour. Your client has permitted us to contact you to request your participation in the study and consented to you revealing information about them.

### Do I have to take part?

It is up to you to decide to join the study. We will describe the study and go through this information sheet. If you agree to take part, we will then ask you to sign a consent form. You are free to withdraw at any time, without giving a reason. This would not affect your status as a professional.

### What will happen to me if I take part?

If you agree to take part you will be asked to complete one questionnaire which will take about 5 minutes. The questionnaire will be done in private. It will provide statements (e.g. Sometimes my client flies off the handle for no good reason) and ask you to rate how applicable each statement is to client. We would only meet once to complete the questionnaire. At the end of the questionnaire you will be given an opportunity to ask any questions and discuss any concerns you may have. Here is a brief outline of the questionnaire you will be asked to complete:

- Partner Aggression Questionnaire (AQ-P)  
This 29 items questionnaire will require you to rate each statement depending on how characteristic it is of your client. The term “partner” will be substituted with the term “client” throughout.

### What are the disadvantages of taking part?

It can sometimes be difficult to reveal information about a client if you fear it will portray them in what could be considered a negative way. The researcher will be available whilst you are completing the questionnaire to answer any questions and discuss any concerns you have about the information you are providing.

We will also provide you our contact information in case you have any further questions.

### What are the possible benefits of taking part?

We cannot promise the study will help you or your client but the information we get from this study will help improve the understanding of brain injury and therefore improve treatment of people who have suffered a brain injury.

### What happens when the research study stops?

Once the all the participants have been seen and the research has been completed we will write to you with a summary of the results.

### What if there is a problem?

Any complaint about the way you have been dealt with during the study or any possible harm you might suffer will be addressed. The detailed information on this is given in Part 2.

### Will my taking part in the study be kept confidential?

Yes. We will follow ethical and legal practice and all information about you and your client will be handled in confidence. The details are included in Part 2.

If the information in Part 1 has interested you and you are considering participation, please read the additional information in Part 2 before making any decision.

## PART 2

### What will happen if I don't want to carry out the study?

You are free to withdraw from the study at any point. This will not affect your professional status in any way. If you withdraw from the study we will ask you whether you would like the information collected up till that point to be included in the study, or if you would like it to be destroyed.

### What if there is a problem?

If you have a concern about any aspect of this study, you should ask to speak to the researcher who will do their best to answer your questions [see contact details for Paul Walton]. You can also contact the supervisor of the study (Paul Walton) or talk to your site manager. If you remain unhappy you can complain formally, the researcher is obliged to provide these details when requested.

### Will my taking part in this study be kept confidential?

All information which is collected during the course of the research will be kept strictly confidential. Your information will be seen by the researcher only. All documentation will be kept in a locked filing cabinet. The information you provide will be identified by number so everything will remain anonymous and completely confidential. Your data will be held for up to 5 years after the study has ended and then destroyed. All data will be managed in line with the Data Protection Act.

There are occasions when confidentiality must be broken. If you disclose information indicating that you or others are at risk of harm it will be necessary to inform the appropriate authorities.

Data will be collated in a computer database using codes to identify individuals. All files will be password protected.

### What will happen to the results of the study?

We intend to publish the results of this study. You will not be identified in any publication.

Once all the participants have been seen and the research has been completed we will write to you with a summary of the results.

### Who is organising and funding the research?

This study is being organised by The University of Hull and funded by the Humber NHS Foundation Trust.

### Who has reviewed the study?

The research is being supervised and monitored by the Dept of Clinical Psychology at the University of Hull. In addition all research in healthcare settings are looked at by an independent group of people, called a Research Ethics Committee, to protect your interests. This study has been reviewed and given favourable opinion by Leeds (West) Research Ethics Committee.

Further information

I am able to provide further information should you require it. I can be contacted via the following:

Address: Mr Paul Walton  
Department of Clinical Psychology and Psychological Therapies  
Hertford Building  
University of Hull  
Cottingham Road  
Hull  
HU6 7RX

Telephone: 01482 464 106



# Appendix P – Participant Consent Form (Version 1)

Centre Number:

Study Number:

Participant Identification Number:

## CONSENT FORM

Title of Project:

Name of Researcher:

Please initial  
box

1. I confirm that I have read and understand the information sheet dated.....  
(version.....) for the above study. I have had the opportunity to consider the  
information, ask questions and have had these answered satisfactorily.
2. I understand that my participation is voluntary and that I am free to withdraw at  
any time without giving any reason, without my health care or legal rights being  
affected.
3. I understand that relevant sections of my case notes need to be accessed by  
researchers as part of my participation in this study and hereby authorise their  
access to such material.
4. I agree to take part in the above study.

\_\_\_\_\_  
Name of Participant

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Name of Person Taking Consent

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature

# Appendix Q – Key Worker Consent Form (Version 1)

Centre Number:

Study Number:

Participant Identification Number:

## CONSENT FORM (Key Worker)

Title of Project:

Name of Researcher:

Please initial  
box

1. I confirm that I have read and understand the information sheet dated.....  
(version.....) for the above study. I have had the opportunity to consider the  
information, ask questions and have had these answered satisfactorily.
2. I understand that my participation is voluntary and that I am free to withdraw at any  
time without giving any reason, without my health care or legal rights being affected.
3. I agree to take part in the above study.

\_\_\_\_\_  
Name of Participant

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Name of Person Taking Consent

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature

# **Appendix R – Portfolio Thesis Word Count**

Part One Word Count – 12,802 (excludes abstract, tables, figures, references, appendices and main heading)

Part Two Word Count – 10,398 (excludes abstract, tables, figures, references, appendices and main heading)

Appendix A (Reflective Statement) Word Count – 2,010

Total Portfolio Thesis Word Count – 32,585 (excludes: tables, figures, references and Appendices B, C, G, K, L and M)