

Shields, Jennifer (2010) *Theory of mind & executive function in Asperger's Syndrome and traumatic brain injury: a comparative study and clinical research portfolio*. D Clin Psy thesis.

<http://theses.gla.ac.uk/2206/>

Copyright and moral rights for this thesis are retained by the author

A copy can be downloaded for personal non-commercial research or study, without prior permission or charge

This thesis cannot be reproduced or quoted extensively from without first obtaining permission in writing from the Author

The content must not be changed in any way or sold commercially in any format or medium without the formal permission of the Author

When referring to this work, full bibliographic details including the author, title, awarding institution and date of the thesis must be given

***Theory of Mind & Executive Function  
in Asperger's Syndrome and Traumatic Brain Injury:  
A Comparative Study  
and  
Clinical Research Portfolio***

**Volume I  
(Volume II bound separately)**

***Jennifer Shields***

**University of Glasgow  
Department of Psychological Medicine  
July 2010**

*Submitted in part fulfilment of the requirements for the  
degree of Doctorate in Clinical Psychology*

## Faculty of Medicine Graduate School

### “Declaration of Originality Form”

You have a responsibility to the University, the Faculty, your classmates, and most of all to yourself, to act with integrity in your academic work. In particular, the work that you submit for assessment, other than for team exercises, *must be your own*. Just as cheating in examinations is a serious offence, so any form of collusion or plagiarism in assessed exercises is dishonest and unacceptable to the University.

**The following is an extract from the University’s Statement on Plagiarism. Please read it carefully and sign the declaration below.**

Plagiarism is defined as the submission or presentation of work, in any form, which is not one's own, without acknowledgement of the sources. Plagiarism can also arise from one student copying another student's work or from inappropriate collaboration. Allowing someone else to copy your work is just as bad as copying someone else's work yourself. It is vital that you do not allow anyone else to copy your work. Take care when discarding work and do not leave copies of your own files on a hard disk where others can access them. If you have any doubt as to what level of discussion is acceptable, you should consult your lecturer or the Course Director.

**The incorporation of material without formal and proper acknowledgement (even with no deliberate intent to cheat) can constitute plagiarism.**

With regard to essays, reports and dissertations, the rule is: if information or ideas are obtained from any source, that source must be acknowledged according to the appropriate convention in that discipline; and any direct quotation must be placed in quotation marks and the source cited. Any failure to acknowledge adequately or to properly cite sources of information in submitted work constitutes an act of plagiarism. Plagiarism is considered to be an act of fraudulence and an offence against University discipline. Alleged plagiarism will be investigated and dealt with appropriately by the University.

The University Plagiarism statement is available from:

**<http://senate.gla.ac.uk/academic/plagiarism.html>** Please complete the information below in BLOCK CAPITALS.

Name Miss Jennifer Shields

Matriculation Number 0702124s .....

Course Name..Doctorate in Clinical Psychology (D.Clin.Psy) .....

Assignment Number/Name. Major Research Project & Clinical Portfolio .....

#### **DECLARATION:**

I am aware of the University’s policy on plagiarism and certify that this assignment is my own work.

Signed..... Date.....

## **Acknowledgements**

I would first of all like to thank Professor Tom McMillan for his continued guidance and support over the duration of my research training.

At the heart of my acknowledgements are the individuals who participated in this study. This research would not have been possible without you and I give you thanks for your willingness to give your time and for sharing your thoughts. I am particularly grateful to all the individuals and organisations that assisted me with recruitment and the practical issues I faced.

I would also like to acknowledge my clinical supervisors who provided me with such a memorable learning experience. A huge thank you also goes to all my peers for all their support and for being part of this unbelievable journey. To my mentor, I would like to extend special thanks. It has been a privilege to have had your guidance and support over the past five years. You are an inspiration.

Lastly, I would like to thank those who are near and dear to me. Mum, thank you for always believing in me. You gave me the confidence to pursue my goals and I'm eternally grateful to you and my step-dad for all your support. Heartfelt thanks also go to a very special friend, for always being there when I needed you and beyond. Finally, I would like to thank my partner for all the encouragement and understanding I needed during the writing of this thesis.

*~ For KW ~*

## Table of Contents

	Page Number
<b>Chapter 1: Systematic Literature Review</b>	05 – 38
<i>Associations between Theory of Mind and Executive Function in Schizophrenia.</i>	
<b>Chapter 2: Major Research Project</b>	39 - 79
<i>Theory of Mind in Asperger's Syndrome and Traumatic Brain Injury: A Comparative Study.</i>	
<b>Chapter 3: Advanced Clinical Practice I (Abstract only)</b>	80 - 81
<i>A new model of thinking about thinking: a reflective critical account.</i>	
<b>Chapter 4: Advanced Clinical Practice II (Abstract only)</b>	82 - 83
<i>Multidisciplinary to Transdisciplinary team working and the role of the Clinical Psychologist: a reflective critical account.</i>	

---

## Appendices

<b>Appendices Contents Page</b>	84
<b>Appendix 1: Systematic Literature Review</b>	85 - 92
<b>Appendix 2: Major Research Project</b>	93 - 123

**Chapter 1**  
**Systematic Literature Review**

***Associations between  
Theory of Mind and Executive Function  
in Schizophrenia***

**Jennifer Shields**

Written According to guidelines for submission to the Journal:

*Schizophrenia Bulletin*

(Author's Instructions – see Appendix 1.1)

Address for correspondence:

Ms Jennifer Shields

Division of Community Based Sciences

Section of Psychological Medicine

University of Glasgow

Gartnavel Royal Hospital

1055 Great Western Road

Glasgow

G12 0XH

## ABSTRACT

**Background:** There is considerable evidence to suggest theory of mind is impaired in individuals with schizophrenia. Schizophrenia is also associated with deficits in executive function, although the extent to which executive function and theory of mind are distinct is not well understood. **Objectives:** To undertake a systematic review of moderate to high quality studies whose primary aim is to explore associations between theory of mind and executive function in schizophrenia. The aim is to provide a review of the evidence and key findings in this area. **Search Strategy:** Electronic searches of articles ranging from January 2000 - January 2010 were undertaken within databases contained in EBSCO-Host and OVID interfaces. Hand searches of key journals and reference lists were also undertaken. **Selection Criteria:** All English language studies that included variables of interest in adults with a diagnosis of schizophrenia/psychosis /delusion disorder were included. Unpublished studies or dissertations were excluded as were studies which did not incorporate a control group. **Data Collection & Analysis:** Data was collected from included studies and measured by a quality rating tool. This tool was compiled in accordance with the CONSORT Guidelines and the Cochrane Library guidelines. Inter-rater reliability was calculated. **Main Results:** Eleven moderate to high quality studies were included in the review. Findings show executive function and theory of mind difficulties are prevalent in schizophrenia. However, results from most papers do not demonstrate clear associations between EF and ToM. Methodological approaches are discussed and suggestions for future research provided. **Conclusions:** There is currently limited evidence to support specific associations between deficits in executive function and theory of mind in schizophrenia.

## INTRODUCTION

Theory of mind (ToM) is the ability to represent other's mental states, intentions, beliefs and knowledge (Baron-Cohen, 1999). ToM is also described as mentalising, and is considered the means by which we participate in successful social interaction (Happe, 1994). Impairments in ToM are common in schizophrenic disorders (Bora, 2009). Individuals with schizophrenia tend to have poor functional outcomes related to difficulties with social cognition, particularly mental state decoding (Bora, 2006; Concoran, 2003). These problems have been compared to the social difficulties experienced by other clinical groups such as autism and brain injury (Kleinhans et al. 2005; Milders et al. 2008; Milders, Fuchs & Crawford, 2003; Sprong et al. 2007; Pickup, 2008; Bora, 2009). The cognitive modalities and neurological basis of these difficulties are currently unclear (Bora, 2009; Pickup, 2008).

Studies have found ToM to be a multifaceted concept. Emerging themes from the literature suggest ToM to span both social-cognitive and socio-perceptual domains (Tager-Flusberg & Sullivan, 2000; Sabbagh, 2004). Social cognitive ToM is the ability to infer mental states based on observation of behaviour while social perceptual ToM is suggested to be independent of other cognitive abilities but dependent upon emotion recognition skills (Bora, 2009). The interplay of cognitive abilities in ToM, particularly executive function (EF) has been explored in number of studies (Pickup 2008; Brune, 2005). Given the complex and multifaceted nature of ToM and EF in schizophrenia, it is an area which requires regular review and synthesis of key findings.



Historically, studies of ToM in schizophrenia explored whether deficits were trait or state dependent. Unlike autism or brain injury, symptoms of schizophrenia can fluctuate between illness and recovery. Frith (1992), hypothesised deficits in ToM stemmed from a person's mental state and symptomatic profile. Patients whose symptoms were in remission were thought to exhibit normal levels of mentalising abilities (Frith & Concoran, 1996). This was in contrast to those who were paranoid, disorganised or actively delusional, who were expected to display higher levels of ToM impairment. Although this view has found some support (Pousa et al. 2008), meta-analyses have upheld the opposing view that ToM deficits are stable traits and not a state dependent phenomenon (Sprong, 2007). This review found that patients with either active or remitting symptoms had impaired ToM; one standard deviation less than controls (Sprong, 2007).

Alongside ToM deficits, individuals with schizophrenia also have concurrent difficulties with EF. EF denotes higher cognitive abilities such as inhibition, abstract reasoning, attention and cognitive flexibility (Perner & Lang, 1999). Deficits in EF and ToM have also been evidenced to co-occur in Dementia (Gregory, 2002), Multiple Sclerosis (Henry et al. 2009) and Traumatic Brain Injury (Shaw et al. 2004; Milders, 2003). This has provoked debate regarding the relationship between executive dysfunction and ToM impairments across clinical groups. Despite the high frequency of co-occurrence, however, a minority of studies have found executive function to be directly associated with ToM (Rowe, 2001; Stone, 1998; Stuss et al. 2001). This is in contrast to a plethora of papers which seemed to find no relationships (Bora, 2009; Pickup

2008; Havet Thomassin, 2006; Bach, Happe, Fleming & Powell, 2000). These conclusions, however, seem contrary to evidence from functional imaging studies which suggest frontal lobes as integral for ToM tasks (Gallagher & Frith 2004). The extent to which other brain regions are involved is still up for debate.

Subsequent research has therefore attempted to deconstruct ToM and EF into specific areas hypothesised to interact during mentalisation. Langdon (2001), hypothesised that an inability to elicit inhibitory reactions could hinder ToM. If unable to disengage from dynamic, salient factors, a person may not be mindful of the subtle cues required to deduce another's belief. Deficits in abstract reasoning were also hypothesised to limit use of representational thought. Although Langdon did find strong associations between EF and ToM difficulties, further analysis using regression models found ToM to have greater predictability for schizophrenia than executive function. Investigations to date, therefore render executive function as a necessary, but insufficient resource for successful ToM. These findings have led other theorists to propose a specific cognitive module to underpin ToM abilities (Fodor 1983). This specific area of brain function, however, exists as a theoretical construct only.

The prevalence of social difficulties across a number of clinical groups has fuelled theorists to continue investigating these variables. What remains unclear, however, is whether conceptually, EF and ToM are areas which are simply too broad to analyse. For ToM, a recurring yet promising delineation in the literature is the constructs of social perceptual versus social cognitive

elements of ToM. Bora (2006), found that mental state decoding could better predict social functioning than mental state reasoning in schizophrenia. Although these concepts appear similar, the important distinction is the demands each element puts upon other aspects of cognition such as working memory and language (Bibby & MacDonald, 2005). Social perceptual or mental state decoding, for example, require relatively less computations as eliciting faux pas. These are vital considerations in the appraisals of results of past studies and the validity of their conclusions.

As ToM is a sub-component of social cognition with perceptual, cognitive and affective elements, deficits can be measured in a variety of ways. These include emotion identification/decoding/inference, intention inference or false belief tasks, tests of pragmatic language (metaphor and irony) in verbal, auditory and visual formats. Stimuli presentations range from computer based tasks to photographs and cartoons, and some tests are more validated than others. Likewise, EF encompasses a plethora of highly complex and inter-related cognitive functions. This would suggest multiple measures of each construct would be required to produce reasonable levels of construct and convergent validity. Selecting measures of EF which can isolate cognitive functions would allow particular facets of EF to be tested. Furthermore, if studies focused on a particular component of ToM (social-cognitive versus social-perceptual), it may be possible to further explore these constructs and accurately test correlates to specific higher executive functions.

The consensus of the literature to date appears to suggest EF and ToM to be independent of each other. To explore EF and ToM in the context of schizophrenia, this paper will systematically review a selection of studies and methodological approaches which have directly contributed to this conclusion. Papers of interest are studies which have aimed to explore ToM and EF in schizophrenia. The evidence and key findings will be discussed. A related aim is to critically review the paradigms and measures employed to explore these associations.

## **METHOD**

A systematic review of the literature was undertaken to explore associations between theory of mind, executive function and schizophrenia. Searches were designed using key search terms identified from past review papers. Checks for alternative keywords were undertaken using a subject heading search. Specificity of searches was checked by exploring the Embase Thesaurus. Additional subject headings included did not produce further relevant results. Key terms (see Table 1) were agreed and utilised to search the following overarching electronic databases: OVID and EBSCO-Host. These facilitated searches of MEDLINE, EMBASE, EBM reviews, including the Cochrane database for Systematic Reviews, CINAHL, PSYCH-Info, Psych-Articles, Social Work Abstracts, the NASW Clinical Register, Health Management Information Consortium and the British Nursing Index. Limitation criteria specified studies published in the English language between January 2000 and January 2010.

**Table 1**

<i>Keywords</i>				
<i>[Theory of Mind] or** [Mentalis*ing] or [Mindblind*ness] or [Social Cognit*ion] or [Social Percept*ion] or [Perspective Taking]</i>	<i>and**</i>	<i>Executive or Cogniti*on</i>	<i>and**</i>	<i>[Schizophreni*a] or [psychos*] or [psychot*ic] or [delusion*al]</i>

\* - symbol denotes use of database operator {\$} which includes truncations of the term to be included within the search.

\*\* - searches were combined using Boolean Operators 'AND' and 'OR'

Titles of articles were used to identify which abstracts should be read in order to identify variables of interest (ToM and EF in Schizophrenia Disorders). Additional methods of gathering articles included hand searches and a manual examination of reference lists obtained from key papers. These included previous systematic reviews extracted from the databases searches. All abstracts obtained were read to ascertain if the full article was relevant to the purpose of the review. Inclusion and exclusion criteria (see below) were applied to all articles with variables of interest.

### ***Inclusion criteria***

1. Studies included participants aged 18 years and over with a diagnosis of schizophrenia, schizoaffective disorder or psychosis.
2. Studies were articles printed in the English language between January 2000 and January 2010.
3. Studies included all variables of interest i.e. theory of mind and executive function in schizophrenia spectrum disorders.
4. Studies used at least one validated measures for each of theory of mind and executive function.

5. Studies included a control group for comparison.

### ***Exclusion Criteria***

1. Studies which took the form of single case reports or unpublished dissertation articles.
2. Previous literature reviews, systematic reviews and meta-analyses were not included.

### ***Assessment of methodological quality***

Final papers were assessed by the author and a fellow trainee clinical psychologist for methodological quality: clarity of objectives, design & methodology, sample characteristics, assessment and outcome, statistical analyses and conclusions. This was carried using a Quality Rating Scale designed by the author (see Appendix 1.2). This scale comprised 30 items based upon CONSORT guidelines of methodological quality for non RCT research studies (CONSORT, 2001). Additional items were incorporated or augmented to ensure relevant aspects of quality were being measured. Studies were then categorised as high, moderate or low quality using an arbitrary grading system whereby > 70 % = High Quality, 40 – 70 % = Moderate Quality < 39 % = Low Quality. Studies deemed to be of low quality were not included in the review. Discrepancies during the rating process were discussed until joint agreements upon subsection scores were agreed.

### ***Data extraction & Synthesis***

This is a qualitative systematic review. Quantitative analysis was not undertaken. Appraisal of quality and characteristics of included studies were carried out by the author and co-rated for increased reliability. Final quality rating scores and significant features are outlined in Table 2. Data extracted from individual papers included: primary aim, number of participants, relevant diagnoses, sampling methods, design, measures utilised, statistical analyses, main findings, effect size (if reported) and results of ToM and EF correlations.

## **RESULTS**

### ***Study inclusion and characteristics***

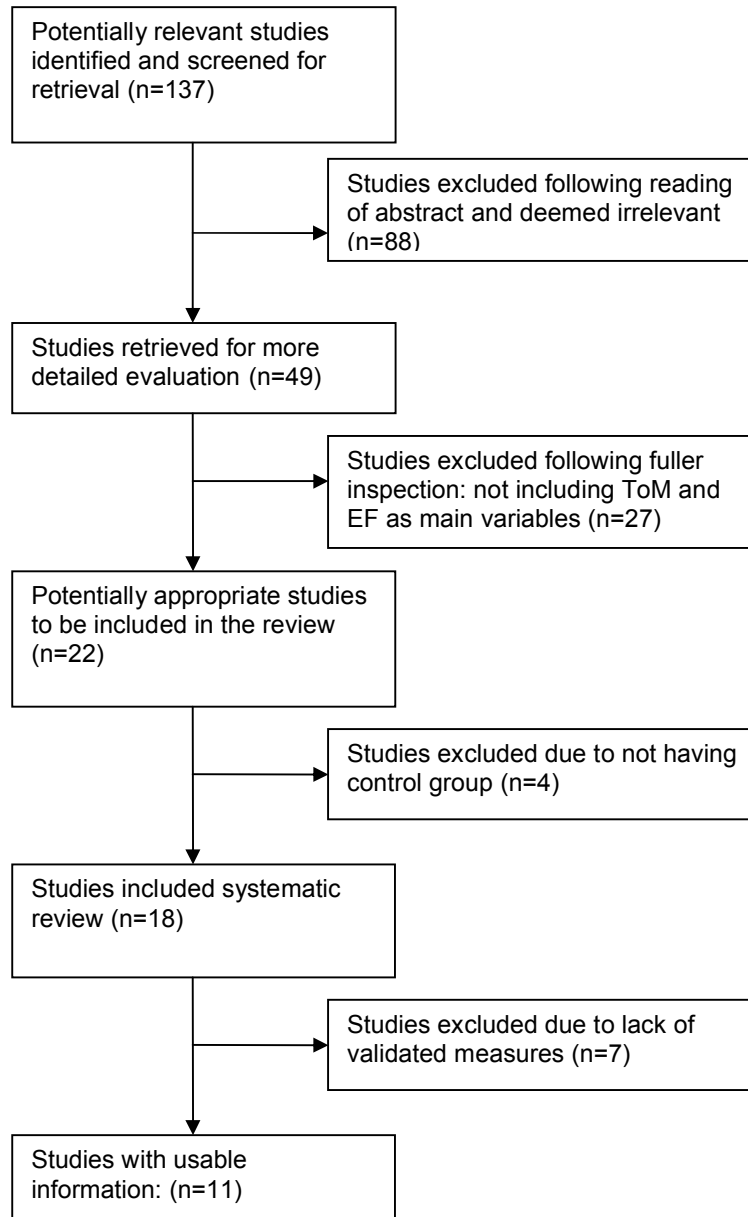
Database searches revealed 2510 papers. Of these 2361 were excluded on the basis of title alone. Following inspection of abstracts, 88 of 137 papers were excluded. The remaining 49 papers were retrieved in full.

On initial consideration of full papers, 27 articles were excluded on the grounds of not investigating ToM and EF as the main variables of interest. A further 11 studies were excluded on rigorous application of the inclusion/exclusion criteria. 4 did not include a control group and 7 did not include validated measures. A flowchart of article inspection is depicted below in Figure 1. The remaining 11 studies which met all criteria were reviewed in full.

The 11 studies were subject to quality rating. Analysis using the Kappa statistic was performed to determine inter-rater reliability of the Quality Rating Scale. On the basis of scores from a random sample of 9 of the 11 studies, the inter-

rater reliability quotient was found to be high ( $Kappa = 0.8$ ;  $p < 0.05$ ), (Cohen 1988). Discrepancies were resolved by discussion. 9 out of 11 studies were rated as 'high quality' and 2 were of 'moderate' levels of quality. Key features of the included papers are summarised in Table 2.

Figure 1: Systematic Review Flow Chart (amended from Consort 2010)





### ***General characteristics***

Studies investigated theory of mind and executive function in individuals with schizophrenia spectrum disorders. Additional variables included symptoms, generic social cognition, pragmatic language, social function, emotion recognition and expressivity. Studies therefore varied in terms of hypotheses and objectives but recurrent themes were evident. All papers attempted to establish if executive function could account for deficits in ToM. Studies mainly featured univariate and correlation analyses comparing groups of patients with schizophrenic spectrum conditions with controls on various measures relating to ToM, EF, social functioning and general cognition. Unfortunately few studies reported effect sizes. Shortages of consistent descriptive data hindered attempts to calculate these for the purposes of this review.

The participant sample sizes in the reviewed papers ranged from 32 to 182 participants. Studies attempted to match participants to controls in 6 of the 11 papers. One study compared three groups; the third being parents of patients with schizophrenia. A further study compared individuals with psychosis to those with familial or psychometric risk in addition to controls. All studies included individuals with schizophrenia; however some papers also included other schizophrenia spectrum conditions such as schizoaffective disorder. Of the 11 studies, 7 included participants with schizophrenia only. All 11 studies reported clinical and demographic characteristics. Some studies attempted to match educational history, but this remained a common source of difference between groups. Recruitment of clinical populations was predominantly from convenience samples, such as outpatient clinics or inpatient wards. Hence,

results cannot be generalised to the wider populations of people with schizophrenia.

**Table 2: Characteristics of Included Studies**

Study	Diagnosis & number Participants	ToM Measures	Exec Fn Measures	Other measures e.g. neurocognitive/ clinical/screening	Main Findings	ToM / executive function correlations / associations	Effect Size (d) Reported ToM/EF	Quality Rating - Score/30
Anselmetti et al 2009	47 participants with Schizophrenia and their parents 47 healthy controls and their parents	Picture Sequencing Task	WCST Symbol Coding Task	NART PANSS SCID-I	Participants with schizophrenia performed poorly on both neurocognitive & ToM measures.	Regression analyses show neuropsychological tests were able to predict ToM ability in patients. Results show ToM impairment not correlated to other aspects of cognitive fn.	No	High (23/30)
Ba' et al (2008)	16 outpatients with schizophrenia spectrum disorders 16 controls.	False Belief Short Stories (1 <sup>st</sup> & 2 <sup>nd</sup> order)	WCST ROCF copy D2 (attention) encumbrance test Categorical verbal fluency.	Rey AVLT Rey OCF AIPSS QFS ERA BPRS-24 Standard Progressive Matrices	Neurocognitive deficits in memory, attention and executive function in patient group. (no differences on some exec measures: visual spatial planning and problem solving. ToM deficits on 2 <sup>nd</sup> order tests in patient group.	Findings suggest no direct relation between neurocognitive impairments and social dysfunction. To answer question regarding neurocognition, social function and ToM the number of px should be increased and groups homogenous. Each dimension should be measured using wider range of tools aid predictive value.	No	Moderate (17/30)

*Abbreviations: AIPSS: Assessment of Interpersonal Problem Solving Skills; BADS: Behavioural Assessment of Dysexecutive Syndrome; BLERT: Bell-Lysaker Emotion Recognition Task; BPRS-24: Brief Psychiatric Rating Scale-24; CANTAB: Cambridge Neuropsychological Test Automated Battery; CAPE: Community Assessment of Psychiatric Experience; D2: Test of Attention; ERA – Echelle de relation avec les autres; FEDT: Face Emotion Discrimination Task; FEIT: Face Emotion Identification Task; IPSAQ: Internal, Personal and Situational Attributions Questionnaire; NCCES: Aphasia Test ; OCCPI: Operational Checklist for Psychotic Disorder; QFS: ; RBANS: Repeatable Battery Assessment of Neuropsychological Status; ROCF: Rey Osterith Complex Figure ; Rey AVLT: Rey Auditory Verbal Learning Test; SAPS: Scale for Assessment of Postive Symptoms; SANS: Scale for Assessment of Negative Symptoms; SCID-I: Structured Clinical Interview for DSM disorders; SCST: Schema Comprehension Sequencing Task; NART: National Adult Reading Test; WAIS: Wechsler Adult Intelligence Scale; WMS: Wechsler Memory Scale ; WCST: Wisconsin Card Sorting Test; WRAT-III: Wide Range Achievement Test; 1= partial eta squared.*

<i>Study</i>	<i>Diagnosis &amp; number Participants</i>	<i>ToM Measures</i>	<i>Exec Fn Measures</i>	<i>Other measures e.g. neurocognitive/ clinical/screening</i>	<i>Main Findings</i>	<i>ToM / executive function correlations / associations</i>	<i>Effect Size (d) Reported ToM/EF</i>	<i>Quality Rating - Score/30</i>
Bora (2006)	Schizophrenia n=50 split into two groups of 25.	Eyes Test Hinting Task	Stroop Interference	Social Functioning Scale Auditory consonant trigrams Trail Making Test (for psychomotor speed) WAIS Information	Patients with poor functional outcome poorer on Eyes Test. Effect size large. No significant differences on hinting task or neurocognitive tasks.	Mental state decoding tasks (Eyes) correlated highly with good functional outcome. No correlation found between Eyes test of ToM and other clinical variables. Author concludes more specific deficits may impinge on neurocognition and ToM.	0.26 <sup>1</sup> /0.10 <sup>1</sup>	High (26/30)
Bora (2008)	91 outpatients 55 controls.	Hinting Task Eyes Test	Verbal Fluency Stroop Interference	WAIS Information Auditory consonant trigrams	Patients were poorer on executive function (Stroop) and ToM tests. Differences disappeared when corrected for working memory deficit and age. ToM deficits more pronounced in +ve & -ve symptoms. No difference between symptomatic & nonsymptomatic patients.	ToM deficits in non-symptomatic schizophrenic patients are secondary to other cognitive deficits. Findings compatible with domain model of ToM. Domain of general ability & working memory seems necessary. No mention of executive function within this domain.	0.80/ 0.48	High (28/30)
Brune et al (2005)	Schizophrenia n=23 Controls n=18	Facial Affect Test Cartoon Picture Stories	WCST BADs – Key Search & Zoo Map.	PANSS Social Behaviour Scale (SBS)	Patients were significantly impaired in comparison to controls on all tasks, both executive and ToM.	Impaired executive function only partially accounts for deficits in social perception and social cognition.	No	High (22/30)

Study	Diagnosis & number Participants	ToM Measures	Exec Fn Measures	Other measures e.g. neurocognitive/ clinical/screening	Main Findings	ToM / executive function correlations / associations	Effect Size (d) Reported ToM/EF	Quality Rating - Score/30
Brune (2009)	Schizophrenia n=50 Controls n = 30	False belief cartoons	Zoo Map WCST	Social Functioning Scale Auditory consonant trigrams Trail Making Test (for psychomotor speed) WAIS Information	Non verbal expressivity reduced in patients, lack of prosocial non verbal signals associated with poor social competence and partially with impaired understanding of others minds but not social cognition or medication.	Patients' social competence was associated with levels of executive functioning and mentalising abilities. Largest –ve correlation between cognitive disorganisation and nonverbal expressivity.	No	High (25/30)
Champagne – Lavau & Stip (2010)	Schizophrenia n= 20 Controls n = 20	False Belief Stories. Metaphor comprehension	WCST Stroop Hayling Trail Making Verbal Fluency	NCCES (Aphasia)	Schizophrenic patients exhibit significant pragmatic impairments which co-occur with executive dysfunction such as lack of flexibility and ToM.	Analyses of covariance suggested that ToM could play a role in pragmatic understanding while flexibility did not. Therefore this study partially supported involvement of prefrontal cortex.	No	High (24/30)
Langdon (2001)	Schizophrenia n=30 Schizoaffective Disorder n = 2 Controls n = 24.	Picture Sequencing	Tower of London	WMS SAPS SANS	ToM & executive function deficits found in patients. Logistic regression analyses showed false belief scores to be a predictor of patient status after all other task variables had been fitted.	Study supports modular hypothesis of ToM; that a cognitive module exists which is dedicated to the inferring and representation of mental states.	No	High (25/30) 0)

Study	Diagnosis & number Participants	ToM Measures	Exec Fn Measures	Other measures e.g. neurocognitive/ clinical/screening	Main Findings	ToM / executive function correlations / associations	Effect Size (d) Reported ToM/EF	Quality Rating - Score/30
Pinkham & Penn (2008)	Schizophrenia n=49 Controls n = 44	ToM Vignettes Hinting Task Conversation probe role play FEIT FEDT	RBANS Trail Making	SCST BLERT WRAT-III WMS	Impaired performance across several domains of neuro-cognition, social function and interpersonal skills.	Social competence contributed unique variance to interpersonal skill beyond neuro-cognition.	No	High (23/30)
Shur, Shamay Tsoory & Levkovitz (2008)	Schizophrenia n= 26 Controls n = 35	Faux Pas Eyes Test	CANTAB	WAIS Similarities PANSS SCID-D	Schizophrenic participants were impaired in affective and cognitive ToM integration. Faux pas task was affected by performance on orbitofrontal tasks not dorsolateral.	Selective impairment in affective ToM rather than general ToM. Orbitofrontal functions (where cognition and effect intersect) Results suggest a 50 variance in faux pas is explained by symptomatology rather than cognition.	0.90/ 0.95	High (24/30)
Van Hooren Et al (2008)	Psychotic Disorder = 44 Familial risk=47 Psychometric risk = 41 Controls = 54	Hinting Task Beads Task Action Recognition Test Speech Attribution Task IPSAQ	The Stroop Colour Word Test Trail Making Test .	CAPE OCCPI	Neurocognitive and social cognition were loaded on different EFA factors. Lack of overlap among social cognition measures suggests term social cognition encompasses various cognitive mechanisms.	Neurocognition and social cognition cannot be explained by a single underlying factor.	No	Moderate (20/30)

### ***Executive Function and Theory of Mind in Schizophrenia***

All 11 papers consistently found EF and ToM to be impaired in patients with schizophrenia (Anselmetti et al. 2009; Ba et al. 2008; Bora, 2006; Bora, 2008; Brune et al. 2005; Brune et al. 2009; Champagne – Lavau & Stip, 2010; Langdon, 2001; Pinkham & Penn, 2008; Shur & Shamay –Tsoory, 2008; Van Hooren et al. 2008). Nine of the eleven papers were rated as high quality whilst two were of moderate quality. Study characteristics, aspects of methodology, key findings and ToM and EF associations are discussed for each paper beginning with studies of high quality.

Anselmetti et al. (2009), compared ToM in individuals with schizophrenia, their parents and controls. The association between specific cognitive impairments and ToM were explored comparing patients and their parents. This study also measured cognitive flexibility and attention using perseverative errors on the Wisconsin Card Sorting Task (WCST) and the Symbol Coding Task (SCT) from the Brief Assessment of Cognition in Schizophrenia (BACS). ToM was assessed using the Picture Sequencing Task (Brune, 2005) and an additional ToM questionnaire relating to the cartoon stories. More than one measure in each domain (EF and ToM) was employed. Multiple regression analyses explored whether cognitive functioning could account for impairments in ToM. ToM was found to be predicted by pre-morbid IQ, the SCT and WCST (perseverative errors). Anselmetti et al. (2009), concluded that ToM relates to a specialised social cognitive network in the brain including the medial prefrontal cortex.

Bora et al. (2006), explored theory of mind, social function and neurocognition (including EF) in outpatients with schizophrenia. Two measures of ToM (Eyes and Hinting Task), and one of EF (Stroop Task) were used alongside additional measures of general cognition. ANOVA's, ANCOVA and multiple regression analyses were applied with corrections for multiple comparisons. Regression analysis was used to model if ToM performance could be predicted by cognitive functioning. EF and sustained attention were included as covariates. The symbol coding task, Wisconsin Card Sorting Task (WCST) and pre-morbid IQ were significant predictors of ToM performance. These predictions were specific to patients only, and not found in parents of patients with schizophrenia.

Bora et al. (2008), investigated relationships between two aspects of ToM, cognitive deficits and residual symptoms in schizophrenia. Two measures of ToM were used to measure both perceptual and conceptual aspects alongside two measures of EF. The ToM tasks utilised pictures of Eyes and ability to infer intentions behind indirect speech utterances. Analysis comprised univariate tests and included covariance for working memory and age. Bonferroni corrections were used for multiple comparisons. Significant differences in ToM were found between controls and patients. In non-symptomatic patients, ToM differences disappeared after controlling for working memory and age. No correlational analyses were performed. This study supports the view that ToM deficits are secondary to other cognitive deficits and thus supports domain general models of ToM.



Brune et al. (2005) examined emotion recognition, theory of mind and social behaviour in schizophrenia versus controls. Using perceptual and conceptual tests of ToM, (facial affect test and cartoon stories) and two measures of EF, Brune found significant between group differences using non parametric tests followed by stepwise regression analysis. Correlation matrices found strong associations of ToM performance and executive functioning. ToM scores were the strongest predictors of patient versus control group membership.

Brune et al. (2009) examined the correlation between non verbal expressivity, impoverished social competence and neurocognition. Two measures of EF and one measure of ToM were used. The ToM test assessed conceptual elements using false belief cartoons. Non parametric analyses found patients' social competence to be associated with levels of executive function and mentalising abilities. Despite data deviating from normality, ANCOVA's were utilised to explore power of covariates. Perseverative errors on the WCST were correlated with the ToM measure.

Champagne-Lavau & Stip (2010), explored whether pragmatic deficits exist with ToM or EF impairments in schizophrenia. To assess ability to process non literal speech, a standardised language assessment protocol was employed which had been validated with norms for age and education levels. A range of EF tasks were administered including the WCST for which perseveration errors were measured. A verbal format false belief test was also administered. T Tests and ANOVA were performed on data and alpha level adjustments set to  $p < 0.01$ . Spearman correlations were also performed. Performance on

pragmatic tasks correlated significantly with performance on the WCST, particularly perseverative errors and the Trails test of EF. False belief tasks also correlated positively with the EF tests. Inflexibility was not significant after covariance adjustment. This study concluded that pragmatic deficits cannot be completely explained by executive dysfunction.

Langdon, (2001) evaluated the argument against the non modular account that attributes poor mentalising to generalised difficulties in hypothesising state of affairs and inhibiting salient misleading contextual information. One ToM and EF test were employed. Deficits in both EF and ToM were found in schizophrenic patients. Logistic regression analyses showed false belief scores predicted group membership after all other task variables had been accounted for. The findings of this study support an independent mentalising module which is dedicated to inferring and representing mental states.

Pinkham & Penn, (2008) explored the relationships between neurocognition, social cognition and interpersonal skills. A range of ToM and EF measures demonstrated impaired performance across several domains of neurocognition, social function and interpersonal skills in schizophrenic patients. Univariate and multivariate levels of analyses were used in addition to correlation and regression models of analyses. Overall, performance on social cognitive tasks predicted almost twice the variance in interpersonal skill then neurocognitive factors.

Shur et al. (2008) examined integrative affective ToM and neurocognitive correlates of impairments. Using two ToM tests and the CANTAB battery to assess EF, this study found faux pas tasks were affected by performance on orbitofrontal tasks but not dorsolateral ones. Results also suggested that 50% of the variance in faux pas is explained by symptoms rather than cognition. T-Tests and multiple regression models were employed to examine relationships. Although a broad measure of cognitive function was used, this study helpfully conceptualised difficulties into orbitofrontal dysfunctions rather than dorsolateral dysfunctions.

Ba' et al. (2008) explored neurocognition, social functioning and ToM in patients and controls alongside relationships to psychiatric symptoms. This study used a range of EF measures and one ToM measure in a verbal format: namely False Belief Short Stories. T-tests and correlations (corrected for multiple comparisons), were followed by regression analyses with neurocognitive and ToM variables considered as predictor variables. This study concluded that no direct association exists between neurocognitive impairments and social dysfunction. Study limitations included modest participant numbers (16 in each group) and heterogeneous samples.

## **DISCUSSION**

This paper synthesises key findings from studies whose primary aim was to explore associations between theory of mind and executive function in schizophrenia. Understanding possible relationships between executive function and theory of mind is an area of ongoing investigation across a range

of clinical groups. In schizophrenia, however, there are additional facets of complexity. These include the spectrum of schizophrenic disorders, symptomatic variations and oscillations on a continuum between illness and recovery. EF and ToM in schizophrenia is therefore multifaceted and complex thus requiring regular review.

Studies identified in the review were all of moderate to high quality. Papers frequently found statistically significant relationships between tests of EF and ToM. These associations were often obtained from correlation models of analysis or analyses of covariance. These effects were frequently lost, however upon controlling for between group variables such as pre-morbid IQ, symptoms (Brune, 2005; Shur, Shamay-Tsoory & Levkovitz, 2008), verbal IQ (Bora et al. 2006), working memory and age (Bora et al. 2008). Subsequently, the trend suggested by these studies is that EF does not completely account for deficits in ToM. Some studies concluded independence of ToM and EF in schizophrenia. In light of the heterogeneity of findings and methodological issues, however, further consideration may be required before arriving at this conclusion.

Additional difficulties synthesising clear outcomes stem from the diverse use of concepts and constructs to define aspects of social cognition. Various descriptors are used interchangeably with ToM despite being comparatively broader terms denoting a wider range of abilities. Papers which attempted to distil ToM into theoretically related components such as perceptual versus conceptual ToM appeared to offer a valuable means by which elements of ToM

can be understood. In contrast, studies seemed to concur on the aspects of EF being measured. These normally included attention, flexibility of thinking and inhibition. This seemed helpful in providing a consistent basis from which to focus comparisons. Orbitofrontal and dorsolateral distinctions were also offered as a means of understanding cognitive processes. Despite a wealth of research exploring associations between ToM and EF, relatively few focused solely upon this as a primary aim.

This is in contrast to the large number of papers investigating the effects of symptoms on ToM and EF. Two studies in this review also compared ToM performance in relation to symptoms and cognition. Although evidence strongly supports the view that EF and ToM difficulties are stable traits in schizophrenia (Sprong, 2007), one paper concluded that up to fifty per cent of the variance in ToM could be explained by symptoms rather than cognition (Shur, 2008). A further study concluded ToM deficits in non-symptomatic patients to be secondary to cognitive deficits and domain specific (Bora, 2008). Hence, this appears to be an area of continued debate. Although these are interesting findings, papers infrequently provided detailed hypotheses about the possible mechanisms that may be interacting between symptoms and ToM. When EF was explored as a mediating factor, findings were rarely verified through predictive forms of analyses. In addition, none of the reviewed studies reported power calculations to verify sample planning and support models of analysis. Difficulties were also experienced in attempting to calculate overall effect sizes. None of the 11 studies reported confidence intervals.

Given the heterogeneity in schizophrenic symptoms, few studies were able to examine EF and ToM associations within homogenous samples where diagnoses or symptoms were similar. Convenience samples were frequently utilised which may not represent the population and limits generalisability. Although samples were mainly recruited from health services, it was difficult to ascertain homogeneity in sampling, as only 54% of the papers reviewed included validated clinical screening measures (e.g. PANSS) (Anselmetti et al. 2009; Brune et al. 2005; Ba' et al. 2008, Shur et al. 2008, and Pinkham & Penn, 2008). Therefore it is unclear which clinical populations on the schizophrenia spectrum were being represented within the samples. Thus extracting reliable and consistent findings is problematic in this area.

A wide variety of measures were also evident in the 11 studies. Although all studies included measures of ToM and executive function, some studies also measured additional aspects of general cognition. This was particularly helpful as variables such as working memory were controlled for (Bora et al. 2008). With regards to ToM measures, a total of twelve different measures were used across the 11 studies. These again varied widely. Information pertaining to the reliability and validity of these tests were not regularly included. Tests ranged from measures of emotion recognition (using photographs of faces or eyes), to intention inferences (faux pas tests, hinting test, false belief) whilst encompassing verbal, auditory, pictorial, photographic and cartoon media. The demands of tests also varied widely, as did the methods by which they were presented. The heterogeneity of measures appeared to reflect the conceptual myriad within the currently investigated term 'theory of mind'. This observation

suggests value in defining set elements within ToM for the purposes of ongoing research.

Measures of EF also varied widely. A total of thirteen tests/subtests of executive function were used in the eleven papers. This number does not include ancillary cognitive measures of memory, language functions, psychomotor functions and visual spatial skills. The most commonly administered measure was the Wisconsin Card Sorting Test (WCST), used in 40% of studies. The majority of cognitive measures were standardised and well recognised assessment tools. A large proportion of the subtests utilised are widely considered to be informal, thus not validated, stand-alone tests. An example is the Zoo Map from the Behavioural Assessment of Dysexecutive Syndrome (BADS) which was used in two papers. As the validity and reliability of the test battery may be reduced if using single subtests, these findings should be interpreted with caution. Validated measures of pre-morbid intelligence or verbal comprehension were found in one study only.

Given the methodological issues highlighted in this review, a number of key areas may require further consideration. ToM and EF are complex and multifaceted areas which appear to require more discrete levels of analysis. Studies would benefit from theoretically viable levels of investigation such as distinguishing between perceptual or conceptual elements of ToM. This could provide a basis for the development of reliable and validated measures for each component. In addition, it may be useful to provide composite scores (where EF scores are standardised and combined) and/or contrast measures (where

overlapping cognitive functions are isolated and partitioned out of analysis). This would perhaps increase levels of construct and convergent validity. Confounds in working memory and general verbal ability could also be controlled for as standard. These would be valuable methodological steps towards the development of more robust paradigms in order to accurately explore possible associations between EF and ToM.

When investigating ToM and EF in schizophrenia, there would be a clear benefit if participant groups comprised homogenous groups of schizophrenic presentations. It would also be advantageous if use of medication was clearly stated and if possible, controlled for. This would be particularly relevant for medications which could affect cognitive functioning. Studies could also incorporate functional brain imaging scans within discrete populations to differentiate cognitive modalities being accessed. Subsequent studies could be extremely powerful by including imaging and neuropsychological paradigms. As research has identified orbitofrontal versus dorsolateral regions of activity (where cognitive and affective abilities are proposed to interact) specific ToM tasks could be developed to further explore ToM facets and corresponding brain activity. This paradigm may be particularly robust if combined with ToM tests in which correlations to executive function have been frequently found (for example the WCST and Symbol Search). Additionally, given the dynamic nature of schizophrenia as an illness, it may also be useful to conduct repeat studies on a longitudinal basis. This could perhaps add to the state/trait debate.



In conclusion, there are substantial gaps in the understanding of the intricate relationships which may exist between EF and ToM in schizophrenia. From a review of evidence to date, there appears to be an array of methodological issues which require further consideration before accepting the current view that theory of mind and executive function are independent in schizophrenia.

## REFERENCES

Anselmetti, S., Bechi, M., Bosia, M., Quarticelli, C., Ermoli, E., Smeraldi, E., et al. (2009). 'Theory' of mind impairment in patients affected by schizophrenia and in their parents. *Schizophrenia research*, 115(2-3), pp.278-285.

Bâ, M., Zanello, A., Varnier, M., Koellner, V., & Merlo, M. C. (2008). Deficits in neurocognition, theory of mind, and social functioning in patients with schizophrenic disorders: Are they related? *Journal of Nervous & Mental Disease*, 196(2), pp.153-156.

Bach L., Happé F., Fleminger S., Powell J. (2000) Theory of mind: independence of executive function and the role of the frontal cortex in acquired brain injury. *Cognitive Neuropsychiatry*, 5: pp.175–92.

Baron-Cohen, S. et al (1999) *Social Intelligence in the normal and autistic brain: an fMRI study. European Journal of Neuroscience*, Vol. 11, pp. 1891-1898.

Baron-Cohen, S. (2001) *Special Issue of the International Review of Mental Retardation*; pp. 23-169.

Bibby, H. & McDonald, S. (2005) Theory of Mind after traumatic brain injury *Neuropsychologia*, 43, pp.99-114

Bora, E., Eryavuz, A., Kayahan, B., Sungu, G., & Veznedaroglu, B. (2006) Social functioning, theory of mind and neurocognition in outpatients with

schizophrenia; mental state decoding may be a better predictor of social functioning than mental state reasoning. *Psychiatry research*, 145(2-3), pp.95-103.

Bora, E., Sehitoglu, G., Aslier, M., Atabay, I., & Veznedaroglu, B. (2007). Theory of mind and unawareness of illness in schizophrenia. *European Archives of Psychiatry & Clinical Neuroscience*, 257(2), 104-111.

Bora, E., Gökçen, S., Kayahan, B., & Veznedaroglu, B. (2008). Deficits of social-cognitive and social-perceptual aspects of theory of mind in remitted patients with schizophrenia: Effect of residual symptoms. *The Journal of nervous and mental disease*, 196 (2), pp.95-99.

Bora, E. (2009) Theory of mind in schizophrenia spectrum disorders. *Türk Psikiyatri Dergisi*, 20(3), pp.269-281.

Brüne, M. (2005). Emotion recognition, 'theory of mind,' and social behavior in schizophrenia. *Psychiatry research*, 133(2-3), pp.135-147.

Brune, M., Abdel-Hamid, M., Sonntag, C., Lehmkamper, C., & Langdon, R. (2009). Linking social cognition with social interaction: Non-verbal expressivity, social competence and "mentalising" in patients with schizophrenia spectrum disorders. *Behavioral and Brain Functions*, 5, 6, pp. 1-10.

Champagne-Lavau, M., & Stip, E. (2010). Pragmatic and executive dysfunction in schizophrenia. *Journal of Neurolinguistics*, 23(3), pp.285-296.

Corcoran, R. (2003) Inductive reasoning and the understanding of intention in schizophrenia. *Cognitive Neuropsychiatry*, 8, pp. 223 -235

Consort (2010) Flow chart of Included Studies: Available from:  
<http://www.consort-statement.org/consort-statement/overview0/>

Fodor, J.A. (1983) *The Modularity of Mind*. Cambridge, MA: MIT Press.

Frith CD (1992) *The Cognitive Neuropsychology of Schizophrenia*. Hillsdale, Erlbaum Associates Ltd.

Frith, C.D., & Concoran, R. (1996) Exploring theory of mind in people with schizophrenia. *Psychological Medicine*, 31, pp. 207-220.

Gallagher H.L. & Frith C.D. (2004) Dissociable neural pathways for the perception and recognition of expressive and instrumental gestures. *Neuropsychologia*, 42, (13), pp. 1725-1736.

Gregory, C., Lough, S., Stone, V., Erzinclioglu, S., Martin, L., Baron-Cohen, S., & Hodges, J. (2002). Theory of mind in patients with frontal variant frontotemporal dementia and Alzheimer's disease: Theoretical and practical implication. *Brain*, 125, pp. 752-764.

Happe, F. (1994). An advanced test of theory of mind: Understanding of story characters' thoughts and feelings by able autistic, mentally handicapped, and normal children and adults, *Journal of Autism and Development Disorders*, 24, pp.129-154.

Havet-Thomassin, V., et al (2006) What about theory of mind after severe brain injury? *Brain Injury*, Vol. 20, 1, pp. 83-91.

Henry, J.D. et al., (2009) Evidence for deficits in facial affect recognition and theory of mind in multiple sclerosis. *Journal of the International Neuropsychological Society*, 15, pp. 277-285.

Kleinhans, N., Akshoomoff, N. Delis, D.C. (2005) Executive Functions in Autism and Asperger's Disorder: Flexibility, Fluency and Inhibition. *Developmental Neuropsychology*, Vol. 27, 3, pp. 379-401.

Langdon, R., Coltheart, M., Ward, P. B., & Catts, S. V. (2001). Visual and cognitive perspective-taking impairments in schizophrenia: A failure of allocentric simulation? *Cognitive Neuropsychiatry*, 6(4), pp. 241-269.

Milders, M., Fuchs, S. & Crawford, J.R. (2003). Neuropsychological impairments and changes in emotional and social behaviour following severe traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 25, pp.157-172.

Milders, M. et al. (2008) Neuropsychological Impairments and Changes in Emotional and Social Behaviour Following Severe Traumatic Brain Injury; *Journal of Clinical and Experimental Neuropsychology*. Vol. 25, No 2, pp. 157-172.

Perner, J. & Lang, B. (1999) Development of theory of mind and executive control. *Trends in Cognitive Sciences*, 3, 337-344.

Pickup, G. J. (2008). Relationship between theory of mind and executive function in schizophrenia: A systematic review. *Psychopathology*, 41(4), pp.206-213.

Pinkham, A. E., & Penn, D. L. Neurocognitive and social cognitive predictors of interpersonal skill in schizophrenia. *Psychiatry research*, 143(2), pp.167-178.

Pousa, E., Duñó, R., Brébion, G., David, A. S., Ruiz, A. I., & Obiols, J. E. (2008). Theory of mind deficits in chronic schizophrenia: Evidence for state dependence. *Psychiatry research*, 158(1), pp.1-10.

Rowe AD, Bullock PR, Polkey CE, Morris RG: 'Theory of mind' impairments and their relationship to executive functioning following frontal lobe excisions. *Brain* 2001, 124, pp.600–616.

Sabbagh, M.A. (2004) Understanding orbitofrontal contributions to theory of mind reasoning: Implications for Autism. *Brain & Cognition*, 55 pp. 209-219.

Shaw et al (2004) The Impact of early and late damage to the human amygdala on 'theory of mind' reasoning. *Brain*, 127, pp.1535-1548.

Shur, S., Shamay-Tsoory, S., & Levkovitz, Y. (2008). Integration of emotional and cognitive aspects of theory of mind in schizophrenia and its relation to prefrontal neurocognitive performance. *Cognitive Neuropsychiatry*, 13(6), pp.472-490.

Sprong, M., Schothorst, P., Vos, E., Hox, J., & van Engeland, H. (2007). Theory of mind in schizophrenia: Meta-analysis. *British Journal of Psychiatry*, 191, pp.5-13.

Stone, V., Baron-Cohen, S., & Knight, R.(1998). Frontal lobe contributions to theory of mind. *Journal of Cognitive Neuroscience*, 10, pp.640-656.

Stuss, D.T. et al. (2001) The frontal lobes are necessary for theory of mind. *Journal of cognitive neuroscience*, 15, 324-337.

Tager-Flusberg, H., Sullivan, K. (2000) A componential view of theory of mind: evidence from Williams Syndrome. *Cognition*, 76, 59-89.

van Hooren, S., Versmissen, D., Janssen, I., Myin-Germeys, I., à Campo, J., Mengelers, R., et al. (2008). Social cognition and neurocognition as independent domains in psychosis. *Schizophrenia research*, 103, (1-3), pp.257-265.

## **Chapter 2**

### **Major Research Project**

#### ***Theory of Mind & Executive Function in Asperger's Syndrome and Traumatic Brain Injury: A Comparative Study***

**Jennifer Shields**

Written According to guidelines for submission to the Journal:

*Journal of Neurology, Neurosurgery & Psychiatry*

(Notes for Contributors - see Appendix 2.1)

Address for correspondence:

Ms Jennifer Shields

Division of Community Based Sciences

Section of Psychological Medicine

University of Glasgow

Gartnavel Royal Hospital

1055 Great Western Road

Glasgow

G12 0XH



## ABSTRACT

**Background:** Deficits in inferring mental states, (theory of mind: ToM) and executive function (EF) are frequently found in Asperger's Syndrome (AS) and are common after traumatic brain Injury (TBI). Although there are commonalities between ToM and EF, there are few studies examining them by comparing these two clinical groups. **Aim:** To compare ToM and EF in individuals with a diagnosis of Asperger's Syndrome (AS) to those with TBI relative to a control group. **Methods:** A between groups design compared three groups of 15 participants with TBI or AS and healthy controls. Measures included the Faces Test, Eyes Test and Cartoon Test, Hayling Sentence Completion and Delis Kaplan Executive Function Subtests. Control measures for mood and general intellect were also administered. **Results:** The TBI and AS groups performed more poorly on ToM measures than controls  $F(2, 40) = 16.39, p = 0.001$ . The TBI group performed more poorly than the AS group on ToM measures  $F(1, 28) = 8.17, p = 0.01$ ; however this effect became non significant upon covarying for anxiety. The TBI group performed significantly more poorly than controls on all measures of EF. No significant differences in EF were found between TBI and AS groups. Scores on EF and ToM measures were correlated in the AS group but not the TBI group. **Conclusions:** Although individuals with TBI and AS have similar levels of impairment in EF and ToM, findings suggest that different mechanisms may underpin between group differences. **Applications:** Existing interventions for ToM deficits in AS may have clinical utility with individuals with TBI, however further research is needed in this area.

## INTRODUCTION

The ability to infer mental states is a fundamental skill in social interaction (Wellman, 1990). Conceptualised as 'Theory of Mind' (ToM), it encapsulates the capacity to interpret behaviour, mood states and plan responses (Baron-Cohen, 2001). The term 'Theory of Mind', is used interchangeably with 'mind-reading' and 'mentalising' (Happe, 1994). It has incorporated a number of components, namely perceptual and conceptual dimensions. The former is the ability to integrate visual information such as social cues. In contrast, conceptual ToM requires inference based upon contextual information thus contains a heuristic component (Concoran, 2000). It can also span affective and cognitive forms of perspective taking (Shamay-Tsoory et al. 2007). Therefore as a construct, ToM seems multifaceted and complex. Reflecting this conceptual myriad is a plethora of measures and assessments.

Despite its complexity, this aspect of social intelligence is regarded as distinct from general intelligence (Baron-Cohen et al. 1999). Abilities are thought to advance rapidly from three to four years of age (Baron-Cohen, 1999), leading theorists to propose existence of a 'Theory of Mind module' which is activated around this time (Baron-Cohen, 1995). Although this implies a degree of neural independence, (Fodor, 1983), there has yet to be agreement upon cognitive modalities or neuroanatomical locations of ToM (Geraci et al. 2010). Its relationship with executive function is also an area of continued debate (Bibby & McDonald 2005; Henry, 2006).

The study of ToM impairments has emerged predominantly from research of autistic spectrum disorders (ASD). Difficulties with social interaction are particularly evident in this population. Individuals with Asperger's Syndrome (AS) display marked social impairments despite high levels of general intelligence and exceptional verbal skills (Baron-Cohen, 1999). They are particularly challenged interpreting facial expressions, deciphering pragmatic language (Mazza, 2008) and gauging appropriate responses. Such impairments manifest as socially inappropriate behaviours, difficulties with social reciprocity and an unawareness of salient non verbal cues (Baron-Cohen 1999).

Similar presentations of ToM difficulties are evident in a range of clinical populations. These include individuals with Schizophrenia (Corrigan, 1997), Dementia (Gregory, 2002), Multiple Sclerosis (Henry et al. 2009) and Traumatic Brain Injury (Shaw et al. 2004, Milders, 2003). It is a prominent feature of the sequelae of frontal lobe damage, particularly in the right hemisphere (Milders, Ietswaart, Crawford & Currie, 2006; Havet-Thomassin, 2006; Happe, 1999). Neuroimaging paradigms investigating these clinical groups have not led to a consensus on the neural networks thought to underpin ToM. This perhaps reflects the complex nature of ToM and the array of functions which may contribute to this ability.

A broad overview of the literature identifies numerous brain regions as possible contributors to ToM. The medial pre-frontal cortex, anterior paracingulate cortex, superior temporal sulci and temporal poles bilaterally have been

consistently implicated (Gallagher and Frith, 2004). Functional magnetic resonance imaging (fMRI) suggests frontal areas such as the orbitofrontal cortex, superior temporal gyrus and the amygdala to be required (Baron-Cohen, 1999; Bibby & McDonald, 2005). Other theorists propose activations of posterior regions to form representations alongside an executive component of action initiation served by prefrontal regions (Abu-Akel, 2004). Dorsal and ventral streams of visual processing have also been explored due to their influence upon pattern recognition and integration of information (Frith & Frith, 2006). The findings show a range of possible brain areas.

A recurrent debate in the literature concerns the influence of frontal lobe and/or executive functions (EF) upon impairments in ToM. The overlap between EF and ToM impairments is evidenced in Schizophrenia, Autism (Delis et al. 2003), TBI (Milders, Fuchs & Crawford, 2003), MS (Henry, 2009), Dementias and Stroke (Gregory, 2002). Although there is a wealth of evidence to suggest that frontal lobes are necessary for ToM (Rowe 2001; Stone 1998, Stuss et al. 2001), no clear relationship between ToM and EF has been established (Bora, 2009, Pickup 2008, Havet Thomassin, 2006; Bach, Happe, Fleminger & Powell 2000). Recent systematic reviews and meta-analyses of ToM and executive function (EF) in Schizophrenia have concluded that ToM and EF exist independently of each other (Pickup, 2008). This conclusion seems anomalous in the face of functional imaging studies which appear to suggest the contrary (Gallagher & Frith, 2004).

Although intuitively it seems likely that higher level cognitive functions are required for theory of mind (Snowden et al. 2003), a reason for this anomaly may be that “conceptually, executive function may be too broad a level of analysis” (Baron-Cohen, 1997: pp.16). Within ASD literature, attempts have been made to construct a more detailed profile of the cognitive abilities required for ToM. Analyses using discriminant functional analysis suggest involvement of complex language, memory, reasoning and concept formation (Minschew, Meyer & Gold, 2002; Ozonoff, 1994; Russel 1997). This approach has promoted an ‘unpicking’ of the multifaceted and multifactorial construct of executive function.

Subsequently, this study will comprise a design which uses specific tests of executive function that are often thought to be impaired after TBI. By deconstructing and comparing specific aspects of EF, it may be possible to explore possible differences or overlap between populations. By deconstructing ToM and measuring ‘perceptual’ versus ‘conceptual’ components, it is hoped that this will reduce the possibility of type one error due to lowered reliability within ToM measures. In using visual measures of ToM it is hoped that deficits in working memory and verbal abilities will not confound results. Additionally, by using composite scores of both sets of measures, it is hoped that this will provide a focused and reliable paradigm by which to compare performance on both EF and ToM variables.

Although the ToM difficulties of individuals with Asperger’s Syndrome (AS) and Traumatic Brain Injury (TBI) can present similarly, (Martin & MacDonald, 2003)

there are no studies which contrast ToM and EF directly between different clinical groups. Subsequently, there is no evidence to indicate that the pragmatic social difficulties found in these populations stem from the same aspects of ToM or EF. If there are similarities, this could strengthen the argument for ToM and EF being somewhat related. If there are differences this could also implicate alternative clusters of executive functions or an independent module as mediating ToM abilities within different populations.

The rationale for this study was to explore two clinical groups with similar social functioning and ToM and EF impairments in an attempt to deconstruct of ToM and EF in order to investigate possible relationships. Stand-alone tests of aspects of EF hypothesised to be pertinent to social function will be administered. These will assess cognitive flexibility, attention and inhibition. Performance upon these measures will be compared across groups to performance on perceptual and conceptual measures of ToM. By deconstructing ToM and EF whilst comparing the clinical groups, it may be possible to establish overlaps in impairments between groups or exclude EF as underpinning this ability. By contrasting individuals with TBI to a population where Theory of Mind difficulties are widely evidenced, it is hoped that this may offer further insights into how these problems can be conceptualised and intervened with.

## **Aim**

This study will compare theory of mind abilities in individuals with Traumatic Brain Injury (TBI), Asperger's Syndrome (AS) and healthy controls. A related

aim is to compare groups on aspects of executive functioning speculated to coexist with theory of mind deficits: namely attention, inhibition, and cognitive flexibility. This study will also seek to explore possible relationships between ToM and EF in AS and TBI.

## **Hypotheses**

- 1) Theory of mind deficits will be impaired in both AS and TBI groups in comparison to controls.
- 2) The profile of ToM deficits will differ between clinical groups: TBI participants will perform better than AS participants on all Theory of Mind tests.
- 3) TBI and ASD groups will differ with respect to executive function: The TBI group will score more poorly than the AS group on executive function tests of attention, inhibition and cognitive flexibility.

## **METHOD**

### ***Participants***

A total of forty five adults were recruited to form a sample comprising three groups of fifteen. Groups included individuals with a diagnosis of Asperger's Syndrome (11 males, 4 females), Traumatic Brain Injury (13 males, 2 females) and a control group (11 males, 4 females). Clinical group participants were recruited from a range of voluntary organisations and community based services in Greater Glasgow. These included Headway, The National Autistic Society, The Autism Resource Centre and the West Dunbartonshire Acquired Brain Injury Service. Controls comprised a convenience sample of individuals

known to the author. The mean age was 38.1 years (S.D. = 10.6). The ratio of male to female participants broadly reflects the frequency by which males present with AS and TBI (Ehlers & Gillberg, 2006; Yates, Williams & Harris et al. 2006). All head injured participants reported having suffered a severe head injury resulting in post-traumatic amnesia (PTA) > 1 day (Russell & Nathan, 1946). Demographic information including education history, employment status, Scottish Index of Multiple Deprivation (SIMD, 2006) decile, nature of injury and time post injury are detailed in Table 1.

*Table 1 - Demographic Information*

<b>Continuous Variable (mean (S.D.))</b>	<b>Control Participants (number/mean (S.D.))</b>	<b>AS Participants (number/mean (S.D.))</b>	<b>TBI Participants (number/mean/range (S.D.))</b>
<b>N (in each group)</b>	15	15	15
<b>Mean Age (years)</b>	39.7 (11.5)	32.6 (9.0)	42.0 (8.5)
<b>Gender</b>	11M, 4F	11M, 4F	13M, 2F
<b>Mean yrs Education</b>	13 (2.7)	13.8 (2.3)	12 (2.1)
<b>% In Employment</b>	80	53	13
<b>Mean SIMD (Deciles):</b>	4	4	3
<b>Mean Time Since Injury (years)</b>	n/a	n/a	11.9 (9.6)
<b>PTA (range/days)</b>	n/a	n/a	2 – 67
<b>Nature of Injury %</b>			
- Assault	n/a	n/a	33
- RTA			27
- Fall			20
- Bleed			20



### ***Inclusion criteria***

1. Participants within the AS group have a diagnosis of Asperger's Syndrome as outlined in the criteria within the DSM-IV.
2. Participants within the TBI group have a diagnosis of severe/very severe Traumatic Brain Injury as classified by Post Traumatic Amnesia lasting 1day or more (Russell and Nathan 1946).
3. Participants aged 16 – 64 years old.
4. Participants whose first language is English.
5. Participants functioning above the learning disability range (IQ > 70) and able to consent to take part in the study.

### ***Exclusion Criteria***

GP records were used to exclude participants if they met any of the following criteria. Where GP records were not available, recruitment sources utilised their own databases to ensure potential participants were suitable for inclusion.

1. Participants with a dual diagnosis of TBI & AS.
2. Participants with any current chronic psychiatric condition or had symptoms of trauma.
3. Any participants with significant levels of risk to self or others were not included in the study.

### **Measures**

#### ***Theory of Mind Measures***

Three types of Theory of Mind tasks were administered to form a composite score of ToM. The Faces Test (Baron-Cohen et al. 1997) assessed ability to

recognise mental state from facial expressions. This test is comprised of 20 photographs of a person expressing basic to complex emotional states. Each photograph offered a choice of two possible answers. The Reading the Mind in the Eyes Test (Baron-Cohen, 2001) measures the ability to infer mental states from looking at expressions from the eye region only. Comprising 37 photographs of eyes, each photo is presented with a choice of four emotions to assist participants infer the displayed emotional state. Both the Faces Test and Eyes Test have been used in a number of studies with patients with Autistic Spectrum Disorders (Baron-Cohen, 2001), Traumatic Brain Injuries (Henry, 2006), Schizophrenia (Pickup 2008) and Dementia (Gregory, 2002)

The Cartoon Task consists of twelve cartoons from previously published studies (Happe, 1999; Milders et al. 2006). They assess capacity to make inferences and/or attribute false beliefs in a non verbal format. Six cartoons require inferences of physical states or situations whilst six require inference of mental states such as false belief. Participants were asked “what is funny” for each cartoon and verbatim responses recorded.

The Social Skills Group Questionnaire (Goldstein & Pollock, 1998) consists of a list of twenty three skills for social competence upon which participants can rate themselves. This was administered in two forms; one for participants to complete and one for a relative or carer (see Appendix 2.2).

### ***Executive Function Measures***

Measures of executive function (EF) were obtained using subtests of the Delis-Kaplan Executive Function System: The Trail Making Test (Condition 4), Verbal Fluency Test (FAS) & Tower Test (D-KEFS; Delis, Kaplan, & Kramer, 2001).

These are stand alone tests which allow for the extraction of contrast scores to ensure discrete measurement of specific areas of function, for example cognitive flexibility, attention and inhibition. The Hayling Sentence Completion Test of Dysexecutive Syndrome (Burgess & Shallice, 1997) was also administered as an additional measure of response initiation and response suppression. Subsequently measures formed an overall composite score of EF. The Dysexecutive Questionnaire (DEX) from the Behavioural Assessment of Dysexecutive Syndrome (Wilson et al. 1996) was also administered in two forms, one designed to be completed by the participant and one for completion by a relative or carer. This is a 20 item questionnaire which covers four broad areas: emotional lability and personality, motivation, behaviour and cognition.

### **Control Measures**

Control measures included a test of intellectual ability and mood. The Wechsler Test of Adult Reading (WTAR, 2001) was administered to establish pre-morbid IQ. The Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983) was used to control for cognitive interference due to the presence of a mood disorder.

## **Design**

This is a between groups design comprising individuals with TBI, AS and a healthy control group. Participation was controlled to ensure no significant differences on variables known to influence cognition such as years in education and IQ.

## **Justification of Sample Size**

As there are no previous papers directly comparing TBI and AS groups, studies exploring similar variables formed the basis of sample size estimations. Henry et al (2006) conducted a study of TBI versus a control group ( $n = 16$  &  $17$  respectively). This study compared verbal fluency executive function tests and mental state attribution tests to examine 'affective versus cognitive' aspects of ToM using the Mind in the Eyes Test. For the ToM dependent measure the control group mean score ( $25.9$ ,  $SD\ 4.06$ ), was compared to a TBI group mean score ( $22.4$ ,  $SD\ 6.49$ ) to give an overall effect size of  $0.66$ . Power calculations for the present study based on this data indicate that a minimum total of  $60$  participants would be necessary to achieve a power of  $0.8$  with  $\alpha = 0.05$ .

## **Research Procedures**

Research procedures were approved by the West of Scotland Ethics Committee prior to sample recruitment (see Appendix 2.3). All participants gave informed consent prior to inclusion. Participants were invited to attend a single ninety minute assessment session. Sessions took place within local community resources within a quiet meeting room setting. Participants were offered a brief outline of the session content and given an opportunity to ask questions. Given

the length of testing, participants were encouraged to ask for breaks when required.

Participants were first asked to complete the Hospital Anxiety and Depression Scale (HADS). This was immediately scored in order to exclude participants with a current mood disorder. This was followed by the Dysexecutive Questionnaire (DEX) from the Behavioural Assessment of Dysexecutive Syndrome (BADs) and the Social Skills Group Questionnaire (SSGQ). The researcher offered support to participants if required. Additional copies of the DEX and SSGQ were then given in a stamped addressed envelope for completion by a relative or carer.

The following sequence of test administration was standard for each participant. The Faces Test was administered first of all given its relative simplicity and external focus. Participants were then presented with the Wechsler Test of Adult Reading (WTAR) and asked to read words aloud. Accuracy of responses was recorded on the WTAR record form. The Eyes test followed, consisting of 36 items. Participants were offered a five minute break after item 17. The Verbal Fluency and Trails subtests from the Delis Kaplan were administered followed by the Hayling Test of Sentence Completion. A further break was offered at this point. The Cartoons subtest was then administered followed by the Tower subtest from the Delis Kaplan. Finally, the Wechsler Vocabulary subtest completed the session. Participants were offered regular breaks. If seeming fatigued or frustrated, participants were reminded of their choice to terminate their session and continue at a later date.

## **Data Analysis**

Exploration of the data was undertaken using The Statistics Package for Social Sciences (SPSS) Version 18. Checks for normality, homogeneity of variance, linearity and multicollinearity were undertaken in advance of parametric inferential statistics. Outputs in the form of histograms and stem and leaf plots suggested that five data sets deviated from normality [Eyes Test, Tower Test, Trails Test, HADS (Depression Index), Cartoons (Mental Inference Items)]. Results of Shapiro-Wilks Tests confirmed this finding. The source of the abnormality originated mainly from scores in the higher performing control group. Data transformation procedures (square root, logarithms and inverse) were unable to normalise distributions with the exception of one measure (HADS – Depression Index). As parametric tests are proven to be robust where assumptions of normality are not met, a series of one way Analyses of Variance (ANOVAS) were chosen for analysis with paired samples comparisons using Tukey's HSD Test. ANCOVA's were also utilised to control for extraneous between group variables. Parametric analyses which violated assumptions of normality were also ratified using a non parametric equivalent. Test scores of ToM and EF were then standardised by converting to z scores. Composite scores for the ToM and EF created from the z scores were used to carry out between groups' analyses of TBI and AS groups using ANCOVA. The composite scores were computed from scores from the AS and TBI groups only. By excluding the control group from these analyses, assumptions of ANCOVA were met. Sidak post hoc corrections were then administered for multiple comparisons (Sidak 1967). The less conservative Sidak correction was chosen over the Bonferroni adjustment in order to maintain power within

analyses (Field, 2007). Means and standard deviations were used to calculate the value of Cohen's  $d$  to indicate effect size. Confidence intervals were calculated to the 95% level.

## **RESULTS**

### **Demographic Information**

A total of 45 participants took part, with 15 in each of the Traumatic Brain Injury (TBI), Asperger's Syndrome (AS) and control groups. Demographics are described in Table 1. A Kruskal-Wallis chi-squared test indicated no significant differences in gender between groups ( $X^2(2) = 1.006, p = .605$ ). The mean participant age was 38.1 years (S.D = 10.6). A one-way ANOVA followed by Tukey's HSD Test found significant differences in age between the AS and TBI group, ( $F(2, 42) = 3.78, p = 0.03$ ). This suggested a need to covary for age in inferential analyses. There were no significant differences between groups for years of education  $F(2, 42) = 17.5, p = 0.14$  or socio-economic status  $X^2(2) = 5.11, p = 0.08$ . Within the TBI group, time since injury ranged from 3 to 32 years ( $M = 11.9$  S.D = 9.6). Retrospective estimates of PTA ranged from 2-67 days, (median = 7). 70% of the sample reported PTA ranging from 2-28 days suggesting this proportion had sustained a severe head injury. The remaining 30% reported PTA lasting between 1-3 months which is indicative of very severe head injury (Russell 1971).

### **Control Measures**

Mann Whitney U tests found significant between group differences on both indices of the HADS (see Table 2). The AS group were significantly more anxious than controls, ( $U = 17, z = -3.98, p = 0.01$ ) and the TBI group, ( $U = 24,$

$z = -3.66$ ,  $p = 0.01$ ). Anxiety in the TBI and Control groups did not differ significantly, ( $U = 98$ ,  $z = -6.04$ ,  $p = 0.58$ ). The AS mean score for anxiety, (11.3, *S.D.* 3.3) is in the 'moderate' range but is typical of the mood profile of individuals with Asperger's Syndrome (Tantam, 2000). Anxiety was covaried for in inferential analyses.

Table 2 – Control Measures

<b><i>Measures</i></b>	<b><i>Control Participants (mean (S.D.))</i></b>	<b><i>AS Participants (mean (S.D.))</i></b>	<b><i>TBI Participants (mean (S.D.))</i></b>	<b><i>P Value</i></b>
HADS – Dep	3.3 (3.4)	5.0 (2.7)	5.3 (2.9)	0.03*
HADS - Anx	5.5 (2.8)	11.3 (3.3)	4.9 (3.7)	0.01**
WTAR	109.4 (9.2)	105.5 (13.7)	101.5 (12.7)	0.21

\* - Denotes significance at 0.05 level \*\* - Denotes significance at 0.01 level

For depression, there were significant group differences between controls and the AS group ( $U = 59$ ,  $z = -2.24$ ,  $p = 0.03$ ), and the TBI group ( $U = 63.4$ ,  $z = -2.05$ ,  $p = 0.04$ ). The TBI and AS groups were not significantly different on depression scores, ( $U = 106$ ,  $z = 0.27$ ,  $p = 0.79$ ). As scores for depression were not clinically significant, depression was not controlled for within inferential analyses. A one way ANOVA found no significant differences between groups for intelligence, ( $F(2, 42) = 1.64$ ,  $p = 0.21$ ).

### **Dependent Variable Measures**

Scores on theory of mind and executive function measures were compared between AS, TBI and control groups. Descriptive and inferential values for overall between group effects are outlined in Table 3. Higher scores indicate



better performance. As the Trails score represented completion time (lower score = better performance), scores were subtracted from the maximum time allowed (240 seconds) to give a 'time remaining score' (higher value = better performance in line with other scores). Inferential analyses are described with and without covarying for age and anxiety.

*Table 3: Dependent Variable Measures: Descriptive and Inferential statistics*

<b>Measures</b>	<b>Control Group (mean (S.D.))</b>	<b>AS Group (mean (S.D.))</b>	<b>TBI Group (mean(S.D.))</b>	<b>P Value</b>	<b>P Value# (non.parametric)</b>
ToM Eyes	28.9 (4.0)	23.7 (4.6)	18.9 (4.46)	0.01**	0.01**
Faces	18.1 (1.7)	18.3 (1.0)	16.3 (2.4)	0.03*	
Cartoons (Mental State)	15.2 (2.3)	10 (5.0)	8.1 (5.4)	0.01**	0.01**
Cartoons (Physical)	13.1 (3.3)	10.4 (4.69)	8.2 (5.2)	0.02*	
Soc Skills QA	94.5 (15.2)	72.1 (16.2)	80.6 (10.0)	0.01**	
EF Hayling (Scaled Score)	17.7 (2.8)	14.1 (4.0)	12.2 (5.7)	0.01**	0.01**
Trails	171.4 (25.6)	140.6 (50.7)	97.9 (63.1)	0.01**	0.01**
Tower	17.3 (2.3)	15.5 (4.5)	12.3 (4.5)	0.01**	
Verbal Fluency	39.7 (12.9)	36.2 (13.7)	21.5 (11.7)	0.01**	
DEX	12.7 (7.5)	34.7 (12.8)	23.3 (12.1)	0.01**	
ToM Composite Score (all groups)	1.9 (1.4)	0.15 (1.9)	-2.05 (2.3)	0.01**	
EF Composite Score (all groups)	2.3 (1.9)	0.31 (2.9)	-2.57 (3.5)	0.01**	

# - p value for non parametric equivalents is provided where data did not meet assumptions of parametric statistics.

\*- Denotes significance at 0.05 level    \*\*-Denotes significance at 0.01 level

Data from ToM and EF measures were transformed to Z scores from which a composite score was derived (see Table 3). Results from Pearson intercorrelation matrices for measures (see Table 4 and 5) found EF measures to correlate significantly with each other ( $p < 0.01$ ) and ToM measures to correlate significantly with each other ( $p < 0.01$ ). Pearson coefficient values ranged from medium to large strength correlations.

Table 4 – Intercorrelations (Pearson Coefficients) for ToM Measures

<b><i>Measures</i></b>	<b><i>Eyes</i></b>	<b><i>Faces</i></b>	<b><i>Cartoon-M</i></b>
Eyes	1	0.53**	0.67**
Faces	0.53**	1	0.36**
Cartoon-M	0.67**	0.36**	1

\*\* - Denotes significance at  $p < 0.01$  level

Table 5 – Intercorrelations (Pearson Coefficients) for EF Measures

<b><i>Measures</i></b>	<b><i>Hayling Total Scaled Score</i></b>	<b><i>Tower</i></b>	<b><i>Trails</i></b>	<b><i>Verbal Fluency</i></b>
Hayling Total Scaled Score	1	0.57**	0.49**	0.63**
Tower	0.57**	1	0.74**	0.64**
Trails	0.49**	0.74**	1	0.73**
Verbal Fluency	0.63**	0.64**	0.73**	1

\*\* - Denotes significance at  $p < 0.01$  level

## **Hypothesis 1**

*Theory of mind will be impaired in both AS and TBI groups in comparison to controls.*

A one way ANCOVA on the composite score for ToM (combining z scores for the Eyes Test, Faces Test and Cartoons Test: Mental Inference items) while covarying for age and anxiety, found a between groups difference ( $F(2, 40) = 16.39, p = 0.01$ ). Paired comparisons using Tukey's HSD Test indicate a poorer performance in both the AS ( $p = 0.02$ ; 95% Confidence Interval (CI) 0.28, 4.89,  $d = 1.1$ ) and the TBI ( $p = 0.01$ ; CI 2.14, 5.61,  $d = 1.7$ ) groups relative to controls after corrections for multiple comparisons.

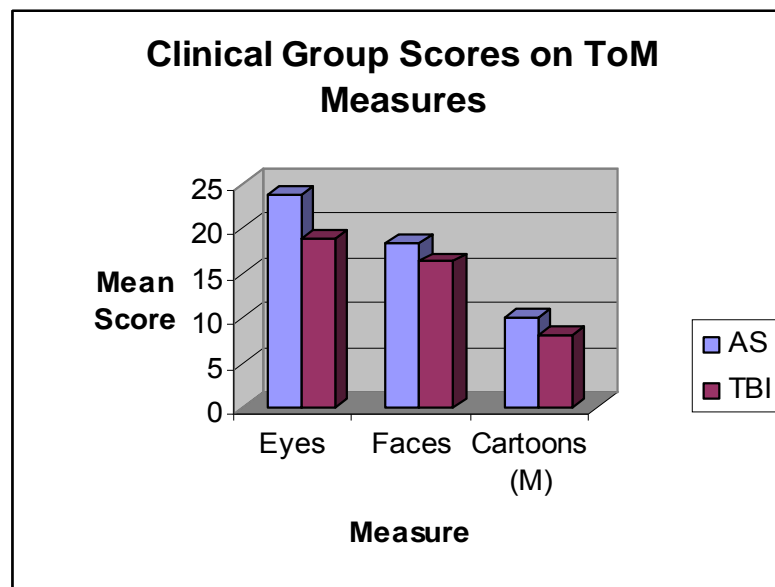
On the Eyes Test, a one way ANCOVA found significant between group differences ( $F(2, 40) = 20.3, p = 0.01$ ). Paired comparisons indicate that the AS group ( $p = 0.01, d = 1.21, CI 1.48, 12.02$ ) and the TBI group ( $p = 0.01, d = 2.36, CI 5.86, 13.79$ ) scored more poorly than controls. For the Faces Test, a one way ANCOVA found between group differences ( $F(2, 40) = 4.00, p = 0.03$ ). Paired comparisons found that the TBI group scored more poorly than controls ( $p = 0.03, d = 0.87, CI 0.173, 3.50$ ) whilst the AS group did not differ from controls ( $p = 0.99, CI -2.10, 2.32$ ). For the Cartoon Test (Mental Inference items), both clinical groups scored significantly less than controls ( $F(2, 40) = 11.63, p = 0.001$ ). Paired samples comparisons found the AS group ( $p = 0.004, d = 1.30, CI 2.01, 12.74$ ) and the TBI group scored more poorly than controls ( $p = 0.001, d = 1.71, CI 2.78, 10.85$ ).

## Hypothesis 2

*The profile of ToM deficits will differ between the clinical groups: TBI participants will perform better than AS participants on all Theory of Mind Tests.*

Mean scores for the two clinical groups on individual measures of Theory of Mind are illustrated in *Figure 1*.

*Figure 1: Clinical Group Mean Scores for ToM*



A one way ANOVA comparing ToM composite scores for the AS and TBI groups (see Table 6), found the TBI group to perform significantly worse than the AS group on ToM tasks ( $F(1, 28) = 8.17, p = 0.01, d = 1.0$ ). However this lost significance upon covarying for age and anxiety ( $F(1, 27) = 0.55, p = 0.55$ ). Anxiety was a significant covariant, ( $F(1, 27) = 4.44, p = 0.05$ ).

*Table 6: Composite Scores for Clinical Groups, Descriptive & Inferential Statistics.*

<b>Measures</b>	<b>TBI Group (z score, (S.D.))</b>	<b>AS Group (z score, (S.D.))</b>	<b>F</b>	<b>Df</b>	<b>P Value</b>
<b>ToM Composite Scores</b>	-1.14 (2.39)	1.14 (1.98)	8.17	1, 28	0.01**
<b>EF Composite Scores</b>	-1.39 (3.34)	1.39 (2.75)	6.23	1, 28	0.05*

\*- Denotes significance at 0.05 level

\*\* -Denotes significance at 0.01 level

Paired comparisons from one way ANOVA's comparing clinical groups on individual ToM measures, found the TBI group to be significantly more impaired than the AS group on the Eyes Test; ( $F(2, 42) = 19.25$ ;  $p = 0.01$ ,  $d = 1.05$ ) and the Faces Test; ( $F(2, 42) = 6.20$ ;  $p = 0.05$ ,  $d = 1.09$ ). From comparisons using ANCOVA (covarying for age and anxiety), levels of significance were lost on both the Eyes Test; ( $F(2, 42) = 19.25$ ;  $p = 0.45$ ) and the Faces Test ( $F(2, 42) = 6.20$ ;  $p = 0.22$ ). No difference was found between AS and TBI groups on the Cartoon Test (Mental Inference items) ( $F(2, 42) = 10.18$ ;  $p = 0.56$ ) in either form of analysis.

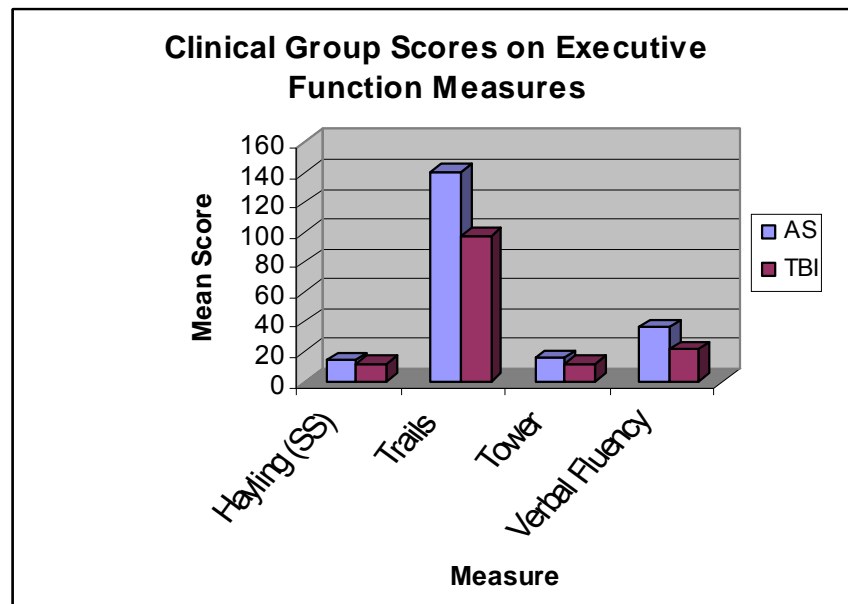
For the Social skills self report questionnaire, both clinical groups rated themselves as less socially skilled than controls ( $F(2, 42) = 9.72$ ,  $p = 0.02$ ). Paired comparisons found that both TBI and AS participants rated their social skills similarly; ( $p = 0.23$ , 95% CI -21.33, 4.26). Postal returns for carer/family reports of social skills were insufficient to perform analyses of self versus carer report discrepancies.

### **Hypothesis 3**

*TBI and AS groups will differ with respect to executive function: the TBI group will be deficient in comparison to the AS group on executive functions subtests of attention, inhibition and cognitive flexibility.*

Clinical group mean scores for individual measures of Executive Function are illustrated in Figure 2.

Figure 2: Clinical Group Mean Scores for EF



Data from EF measures (Hayling Total Scaled Score, Trails, Tower, Verbal Fluency raw scores) were transformed to Z scores. A one way ANOVA comparing EF composite scores found significant differences between AS and TBI groups ( $F(1, 28) = 6.23, p = 0.05, d = 0.90$ ). A one way ANCOVA comparing EF composite scores between TBI and AS groups whilst covarying for age and anxiety was found to be non significant; ( $F(2, 27) = 0.05, p = 0.82$ ).

One way ANCOVA's compared groups on individual EF measures. Significant between group differences were found on the Hayling Test ( $F(2, 41) = 6.17, p = 0.01, d = 0.39$ ). No significant differences were found between the AS group and controls ( $p = 0.12, CI = -1.38, 9.46$ ). The TBI group and controls did differ ( $p = 0.01, d = 1.13, CI = 1.41, 9.56$ .) The AS and TBI group, however, performed similarly on the Hayling with no significance from paired samples comparisons ( $p = 0.91, CI = -4.37, 7.27$ ). Significance was not affected upon controlling for anxiety ( $F(1, 40) = 0.13, p = 0.72$ ).

For the Tower Test, a one way ANCOVA controlling for age and anxiety, found between group differences ( $F(2, 41) = 5.89, p = 0.01, d = 0.71$ ). Paired samples comparisons found that the TBI group differed significantly from controls ( $p = 0.01, d = 1.40, CI = 1.13, 8.14$ ). The AS group did not differ from controls ( $p = 0.09, CI = -0.425, -8.90$ ). The AS and TBI groups performed similarly on the Tower Test ( $p = 1.00, CI = -4.6, 5.4$ ). Levels of significance remained after controlling for anxiety, ( $F(1, 40) = 3.67, p = 0.06$ ).

For the Trails Test, a one way ANCOVA controlling for age and anxiety, found between group differences ( $F(2, 41) = 8.31, p = 0.01, d = 0.75$ ). Paired comparisons found that the TBI group differed significantly from controls ( $p = 0.001, d = 1.53, CI = 26.0, 116.04$ ) however the AS group did not ( $p = 0.11, CI = -8.67, 111.01$ ). The AS and TBI group performed similarly on the Trails Test ( $p = 0.83, CI = -44.41, 84.11$ ). Again, values were not affected upon controlling for anxiety, ( $F(1, 40) = 2.10, p = 0.16$ ).

On the Verbal Fluency Test, a one way ANCOVA controlling for age and anxiety, found significant between group differences ( $F(2, 41) = 9.56, p = 0.01, d = 1.15$ ). Paired comparisons found the TBI group to score significantly poorer than controls ( $p = 0.001, d = 1.48, CI = 6.56, 29.28$ ) whilst the AS group did not ( $p = 0.44, CI = -6.70, 23.5$ ). The AS group performed similarly to the TBI group on the VF Test ( $p = 0.44, CI = -23.5, 6.70$ ). Anxiety was again non significant, ( $F(1, 40) = 3.17, p = 0.08$ ).

Self reported executive abilities, as measured by the Dysexecutive Questionnaire (BADS) suggested both clinical groups to score significantly less than controls ( $F(2, 42) = 14.94, p = 0.01, d = 0.91$ ). Paired samples comparisons found the TBI group to score themselves significantly higher than the AS group for executive functions ( $p = 0.01, CI = 1.45, 21.48$ ).

### **Additional Analyses**

Exploration of the relationship between executive function and ToM tests was undertaken on a within groups basis. Bivariate Pearson correlations were carried out to explore associations between EF and ToM tests within both TBI and AS groups (see Figures 3 and 4).



Figure 3: Relationships between scores on a composite measure of executive function tests in the Asperger's group.

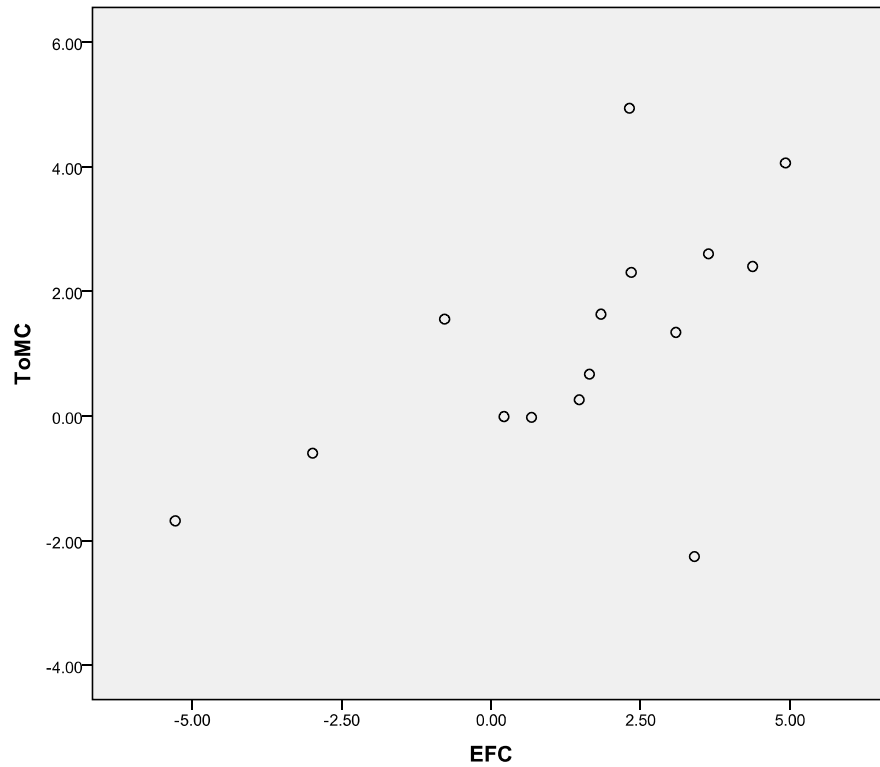
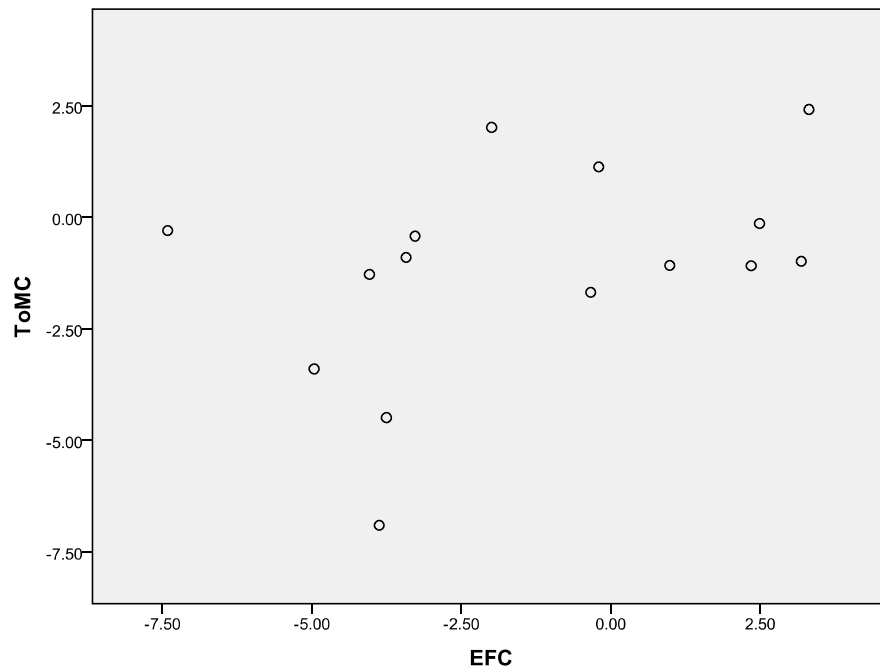


Figure 4: Relationships between scores on a composite measure of executive function tests in the Traumatic Brain Injury group



A statistically significant, positive relationship was found between EF and ToM from z scores derived from the Asperger's Syndrome Group; ( $r = .569$ ,  $p$  (two-tailed)  $p = 0.03$ ). Significance remained upon controlling for anxiety, ( $r = .549$ ,  $p$  (two-tailed)  $p = 0.05$ ). Thus the unique variance in ToM explained by EF is 32%. No significant relationship was found between EF and ToM in the TBI group, ( $r = .409$ ,  $p$  (two-tailed)  $p = 0.13$ ).

## **DISCUSSION**

### **Key Findings**

This study compares theory of mind and executive function abilities in individuals with Traumatic Brain Injury (TBI), Asperger's Syndrome (AS) and healthy controls. Ultimately this study sought to compare profiles of apparently similar difficulties between two diverse clinical groups. In accordance with the first hypothesis, ToM was impaired in both the AS and TBI groups in comparison to controls. In contrast to the greater ToM difficulties hypothesised in the AS group (hypothesis 2), the TBI group was found to be more impaired. This effect, however, became non-significant after controlling for group differences in anxiety (higher anxiety in AS). Given that anxiety is a characteristic of AS (Tantam 2000), a conservative interpretation would be that both TBI and AS groups perform poorly on measures of ToM.

The third hypothesis predicted that the TBI group would be more impaired on measures of executive function than the AS and control groups. Consistent with this, the TBI group was more impaired on both composite scores and

individual measures of EF. Interestingly, the AS group did not differ from the TBI group or controls on individual or composite measures of EF. These results imply that scoring in the AS group fell between those of the controls and TBI group. Results therefore support the null hypothesis that TBI and AS groups would perform similarly on EF. Although this could suggest that AS and TBI groups have similar profiles of EF and ToM, only the TBI group were deficient in EF and ToM compared to controls. It could therefore be postulated that larger sample sizes may be required to accurately discriminate clinical groups. Within group correlations were significant for ToM and EF in the AS group and not in the TBI group. ToM scoring in the TBI group was therefore not associated with executive function. Although correlation does not inform causality, this finding suggests that the profiles of EF and ToM deficits seen in AS and TBI may not be underpinned by the same causal mechanisms.

An additional divergent feature between groups was the degree to which test performance was related to anxiety. Anxiety was a prominent feature in the AS group. The relationship between EF, ToM deficits and anxiety was highly significant in the AS group. Past studies propose that anxiety may reduce EF performance or conversely difficulties with EF can cause anxiety (Frith 2004). The current study does not throw light on the cause/effect debate but is consistent with others in finding a relationship between anxiety and EF in AS.

If simply considering the clinical presentation and not controlling for anxiety, the TBI group were poorer than the AS group in the Eyes Test, as found in previous studies (Havet-Thomassin et al, Henry 2006, Milders, Fuchs & Crawford 2003)

This suggests survivors of TBI may have particular difficulties with the perceptual component of ToM. A recent study using The Awareness of Social Inference Test (TASIT) found emotion recognition and mental inference to be markedly impaired in TBI. Interpreting the expression of 'disgust' was particularly difficult (McDonald & Flanagan 2004). These difficulties could stem from a variety of causes such as impairment in visual perception, processing or semantic organisation. Thus, disentangling what elements affect each group is a complex process.

Self report social skill measures suggest that individuals with TBI have a degree of insight into social difficulties. Both AS and TBI groups rated themselves as significantly less socially skilled than controls. From results of the Dysexecutive Questionnaire, however, individuals with TBI do not attribute difficulties to their own behaviours to the same degree as the AS group. It is unclear if this demonstrates differences in degrees of insight or a valid attribution of difficulties. If impaired insight was an additional problem in the TBI group, this would have clinical implications for the development and implementation of social skills strategies.

### **Clinical Implications**

Further clinical implications for both groups can be drawn from these findings. For the AS group, social difficulties could be conceptualised as stemming from both anxiety and executive function deficits. EF and difficulties with information processing are long established features of the disorder (Hill & Bird 2000). Possible interactions between EF and ToM in the context of anxiety may

therefore benefit from further investigation. For the TBI group, it would appear that social difficulties could stem from a more complex myriad of cognitive difficulties which may or may not be influenced by impairments in a specific cognitive module. Given that injuries from TBI are diffuse, it may be that a host of deficits (disinhibition, lack of flexibility, visual attention and processing deficits) could compound difficulties in ToM abilities. As TBI participants were more impaired on measures of cognitive flexibility, inhibition and attention than the AS group, it could also be hypothesised that the TBI group are less able to compensate for difficulties in ToM. Therefore, the AS group may be more able to employ strategies for inferring mental states and inhibiting first responses whereby the TBI group are not. This view echoes past studies by Channon & Crawford (2000) and Snowden (2003).

Although complex, ToM and EF are clearly two areas of impairment which have a profound effect on individuals with AS and TBI. Conclusions drawn from findings suggest that individuals with TBI may require similar levels of support in social interaction as individuals with social communication disorders such as AS. Before interventions currently evidenced in AS could be reliably generalised for use in TBI, further considerations are required. These primarily concern levels of executive dysfunction which could impinge upon the learning and implementation of social skills strategies. Although the profiles of ToM and EF difficulties exhibited similarities between groups, results of correlation analyses suggest that different underlying pathways could lead to this presentation. The extent to which ToM is underpinned or exacerbated by limited skills in other areas of cognition is therefore still a matter for debate.

## **Strengths & Limitations**

This study compared two clinical groups where difficulties with social skills are common. By employing measures which focused on discrete aspects of ToM and EF, this study attempted to minimise demands on verbal and working memory abilities known to hinder performance (Bibby & McDonald 2005). The study also increased construct validity by using composite scores of related measures. Results of intercorrelation matrices found medium to large correlations between both ToM and EF measures. The highly significant correlations suggest that the measures are related and support the contention that they are measuring the same constructs.

Although these are methodological strengths, limitations of this study surround small sample sizes and difficulties comparing homogenous groups. The AS group differed particularly on measures of anxiety. This difference appeared to have a profound effect on results of parametric statistics. Although anxiety is known to affect test performance, particularly in EF when measured by verbal fluency (Airaksinen, Larsson & Forsell, 2005), it is a predominant feature in Asperger's (Tantam, 2000). It may therefore be beneficial to control for this by comparing AS groups to a moderately anxious control group. Furthermore, given that assessments of ToM are primarily designed for individuals with Autism Spectrum Disorders (Bibby & MacDonald 2005) it is also proposed that the cross referencing of parallels and differences between clinical populations may be hindered by a lack of specificity in the assessment tools. This study is also substantially underpowered to undertake meaningful multivariate forms of analysis to explore direct relationships between EF and ToM.

## **Future Research Considerations**

McDonald and Martin (2003), highlight a shortage of studies which look concurrently at different groups who present with similar pragmatic social and/or language difficulties. This has subsequently led to a lack of theoretical accounts which are applicable to other populations who display theory of mind difficulties. With its inherent complexity, it may be useful for future research to focus upon paradigms which incorporate neuropsychological measures with functional imaging measures. Despite the multifaceted nature of EF and ToM, an increasing number of studies have proposed separate neural frameworks which have been implicated for social processing (Stone, Baron-Cohen & Knight 1998). The ventral stream which links the orbitofrontal areas to areas near the amygdale has been argued to process different aspects compared to the dorsal stream which links the medial prefrontal cortex, anterior cingulate and superior temporal sulcus. By using a social cognitive neuroscience framework, it could be possible to compare discrete cognitive and executive functions with both components of ToM (perceptual and conceptual). By comparing between clinical groups, this would also assist understanding of the wider phenomena.

As ToM and EF are both complex and multifaceted, studies may benefit from adopting standardised approaches to investigation, for example examining perceptual versus conceptual elements of ToM. The development of reliable and validated measures for components of ToM would further increase methodological rigour and the power of subsequent analyses. Studies could also incorporate functional brain imaging scans of patients with discrete lesions

to locate and differentiate possible cognitive modalities. As studies have identified orbitofrontal versus dorsolateral regions of activity (where cognitive and affective abilities are proposed to interact) it would be interesting to explore these areas further. An additional area of further exploration relates to the consistent interaction of anxiety and ToM in the AS group. It would be interesting to explore the effects of anxiety reduction and ToM performance. A proposed paradigm would be to measure Theory of Mind abilities in an AS group versus controls before and after receipt of an evidence based anxiety intervention.

## **CONCLUSIONS**

Although this study did not evidence associations between EF and ToM in TBI, it may be important to advocate for more specific levels of enquiry before final conclusions are made about these two complex areas. Careful consideration of constructs, measures and models of analysis would ideally precede designs incorporating neuroscience paradigms. By speculating existence of intricate and specialised mechanisms, it would follow that the research methods employed to investigate these may require some development. It may also be invaluable to consult with client groups via qualitative means in order to formulate how individuals with TBI or AS attempt to make sense of their social world.



## REFERENCES

Airaksinen, E., Larsson, M., Forsell, Y. (2005) Neuropsychological functions in anxiety disorders in population based samples: evidence for episodic memory dysfunction. *Journal of Psychiatric Research*, 39 (2), pp. 207-214.

Abu-Akel , A. (2003) *A Neurobiological Mapping of Theory of Mind*, *Brain Research Reviews* 43, pp.29–40

Bach L, Happé F, Fleminger S, Powell J.(2000) Theory of mind: independence of executive function and the role of the frontal cortex in acquired brain injury. *Cognitive Neuropsychiatry*, 5, pp.175–92

Baron-Cohen, S. Tager-Flusberg, H., & Cohen, D. (Eds.) (1993). *Understanding other minds: perspectives from autism*, Oxford University Press.

Baron-Cohen, S. (1995) *Mindblindness: an essay on autism and theory of mind*. Boston: MIT Press/Bradford Books

Baron-Cohen, S. (1997) Theory of Mind in Autism: its relationship to executive function and central coherence. In D. Cohen & F. Volkmar (Eds.) *Handbook of Autism and pervasive developmental disorders*. 2<sup>nd</sup> Edition, John Wiley & Sons.

Baron-Cohen, S. et al (1999) Social Intelligence in the normal and autistic brain: an fMRI study. *European Journal of Neuroscience*, Vol. 11. pp. 1891-1898.

Baron-Cohen S., et al. (2001) The 'Reading the Mind in the Eyes' test revised version: a study with normal adults, and adults with Asperger syndrome or high-functioning autism. *J Child Psychology & Psychiatry*, 42, pp.241–251.

Baron-Cohen (2001) *Special Issue of the International Review of Mental Retardation*, 23, pp.169.

Bird C. M., Castelli F., Malik O., Frith U., Husain M. (2004). The impact of extensive medial frontal lobe damage on 'Theory of Mind' and cognition. *Brain*, 127(Pt 4): pp.914-28

Bora, E., Yucel, M., & Pantelis, C. (2009). Theory of mind impairment in schizophrenia: Meta-analysis. *Schizophrenia research*, 109(1-3), pp. 1-9.

Burgess, P, & Shallice, T. (1997). *Hayling and Brixton Tests*. London: Harcourt Assessment.

Channon, S., & Crawford, S. (2000). The effects of anterior lesions on performance on a story comprehension test : left anterior impairment on a theory of mind type task. *Neuropsychologia*, 38, pp.1006- 1017.

Concoran, R. (2000) Theory of Mind in other clinical conditions: Is selective 'theory of mind' deficit exclusive to autism? In Baron Cohen et al (Eds.) *Understanding others' minds*, 2<sup>nd</sup> ed. Oxford. UK. Oxford University Press.

Corrigan, P. (1997). *The social perceptual deficits of schizophrenia*. *Psychiatry*, 60, 309–326.

Delis, D.C., Kaplan, E., & Kramer, J.H. (2001) *Delis Kaplan Executive Function System*. San Antonio, Texas: The Psychological Corporation.

Ehlers, S., & Gillberg, C. (2006) The epidemiology of Asperger's Syndrome. A total population study. *Journal of Child Adolescent Psychopharmacology*, 16, pp.737-756.

Fodor, J.A. (1983) *The Modularity of Mind*. Cambridge, MA: MIT Press.

Frith, C.D., Frith, U. (2006). *How we predict what other people are going to do; Brain Research* 1079 (1), pp.36-46.

Gallagher H.L. & Frith C.D. (2004) Dissociable neural pathways for the perception and recognition of expressive and instrumental gestures, *Neuropsychologia*, 42, (13), pp. 1725-1736.

Geraci, A. et al. (2010) Theory of Mind in patients with ventromedial or dorsolateral prefrontal lesions following traumatic brain injury. *Brain Injury*, Vol. 24, No.7-8, pp. 978-987.

Goldstein, S. (1988) *Social Skills Assessment Questionnaire*. Neurology Learning Behaviour centre, Salt Lake City, UT.

Gregory, C., Lough, S., Stone, V., Erzinclioglu, S., Martin, L., Baron-Cohen, S., & Hodges, J. (2002). Theory of mind in patients with frontal variant frontotemporal dementia and Alzheimer's disease: Theoretical and practical implication. *Brain*, 125, pp.752-764.

Happe, F. (1994). An advanced test of theory of mind: Understanding of story characters' thoughts and feelings by able autistic, mentally handicapped, and normal children and adults; *Journal of Autism and Development Disorders*, 24, pp.129-154.

Happe, F., Frith, U. (2006). The weak coherence account: detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 35,(1), pp.5-25.

Happé, F., Brownell, H., & Winner, E. (1999). Acquired 'theory of mind' impairments following stroke. *Cognition*, 70, pp.211-240.

Havet-Thomassin, V., et al (2006) What about theory of mind after severe brain injury? *Brain Injury*, Vol. 20, 1, pp. 83-91.

Henry, J.D. (2006) Theory of mind following traumatic brain injury: the role of emotion recognition and executive dysfunction. *Neuropsychologia*, 44 (10); pp.1623-8

Henry, J.D. et al (2009) Evidence for deficits in facial affect recognition and theory of mind in multiple sclerosis. *Journal of the International Neuropsychological Society*, (2), 277-285.

Hill, E.L., Bird, C.M. (2006) Executive processes in Asperger Syndrome: Patterns of performance in a multiple case series. *Neuropsychologia*, Vol. 44, 14, pp.2822-2835.

Kleinmans, N., Akshoomoff, N. Delis, D.C. (2005) Executive Functions in Autism and Asperger's Disorder: Flexibility, Fluency and Inhibition. *Developmental Neuropsychology*, Vol. 27, 3, pp. 379-401.

Mazza, M., Di Michele, V., Pollice, R., Casacchia, M., & Roncone, R. (2008). Pragmatic language and theory of mind deficits in people with schizophrenia and their relatives. *Psychopathology*, 41(4), 254-263.

McDonald, S., & Flanagan, S. (2004). Social perception deficits after traumatic brain injury: interaction between emotion recognition, mentalizing ability and socialcommunication. *Neuropsychology*, 18, 572-579.

McDonald, S. & Martin, I. (2003) Weak coherence, no theory of mind, or executive dysfunction? Solving the puzzle of pragmatic language disorders. *Brain & Language* (85) pp.451-466.

Minschew, N. J., Meyer, J., & Goldstein, G (2002) Abstract reasoning in autism: A dissociation between concept formation and concept identification. *Neuropsychology*, 16, pp.327-334.

Milders, M., Fuchs, S. & Crawford, J.R. (2003). Neuropsychological impairments and changes in emotional and social behaviour following severe traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 25, 157-172.

Milders, M., Ietswaart, M., Crawford, J.R., & Currie, D. (2006) Impairments in 'theory of mind' shortly after traumatic brain injury and at one-year follow-up.

*Neuropsychology*, 20, pp.400-408.

Ozonoff, S., et al.(1994) Executive function abilities in autism and Tourette Syndrome: an information processing approach. *Journal of Child Psychology and Psychiatry*, 35, pp.1015-1032.

Pickup, G. J. (2008). Relationship between theory of mind and executive function in schizophrenia: A systematic review. *Psychopathology*, 41(4), pp.206-213

Rowe A.D., Bullock P.R., Polkey C.E., Morris R.G., (2001);Theory of mind' impairments and their relationship to executive functioning following frontal lobe excisions. *Brain*, 124, pp.600–616.

Russell, W.R., Nathan, P.W. (1946) Amnesia following head injuries. *Lancet*, 2, pp. 280-300.

Russell, W.R. (1971) *The Traumatic Amnesias*. Oxford, Oxford University Press.

Russell, J. (Ed.). (1997) *Autism as an executive disorder*. Oxford: Oxford University Press.

Scottish Index of Multiple Deprivation (2006) Available from <http://www.sns.gov.uk>.

Šidák, Z. (1967) Rectangular confidence region for the means of multivariate normal distributions. *Journal of the American Statistical Association*, 62, pp.626–633.

Shamay-Tsoory, S., et al. (2007) Dissociation of cognitive from affective components of theory of mind in schizophrenia. *Psychiatry research*, 149(1-3), pp. 11-23.

Shaw et al (2004) The Impact of early and late damage to the human amygdala on 'theory of mind' reasoning. *Brain*; 127, pp 1535-1548.

Snowden JS, Gibbons ZC, Blackshaw A, Doubleday E, Thompson J, Craufurd D, et al.(2003) Social cognition in fronto-temporal dementia and Huntington's disease. *Neuropsychologia*, 41, pp.688–701

Stone, V.E., Baron-Cohen, R.T., Knight, R.T. (1998) Frontal lobe contributions to theory of mind. *Journal of Cognitive Neuroscience*, Vol. 10, (5), pp.640-656.

Stuss, D.T. et al. (2001) The frontal lobes are necessary for theory of mind. *Journal of cognitive neuroscience*, 15, 324-337.

Tantam, D (2000) Psychological Disorder in Adolescents and Adults with Aspergers Syndrome. *Autism*, Vol. 4, pp.47-62.

Wellman, H. M. (1990) *The child's theory of mind*. Cambridge, MA: MIT Press.

Wechsler, D. (2001) *Wechsler Test of Adult Reading*. NFER Nelson, London.

Wilson, B.A., Alderman, N., Burgess, P.W., Emslie, H.E., & Evans, J.J. (1996) *Behavioural Assessment of Dysexecutive Syndrome*. Bury St Edmunds, UK: Thames Valley Test Company.

Yates, P.J. et al (2006) An epidemiological study of head injuries in a UK population attending an emergency department. *Journal of Neurology, Neurosurgery & Psychiatry*, 77, (5), pp.699-701.

Zigmond, A.S., & Snaith, R.P. (1983) The Hospital Anxiety and Depression Scale, *Acta Psychiatrica Scandania*, Vol. 67, pp. 361-370.



**Chapter 3**  
**Advanced Clinical Practice 1**

**Reflective Account**

*(Abstract only)*

***A new model of thinking about thinking:  
A reflective account.***

**Jennifer Shields**

July 2010

Submitted in part fulfilment of the requirements  
for the degree of Doctorate in Clinical Psychology

Address for correspondence:

Ms Jennifer Shields

Division of Community Based Sciences

Section of Psychological Medicine

University of Glasgow

Gartnavel Royal Hospital

1055 Great Western Road

Glasgow

G12 0XH

## **ABSTRACT**

The ability to operate as a reflective practitioner is fundamental to training within Clinical Psychology doctorate courses. To be increasingly aware of and reflect upon one's actions is a necessary skill which in itself is an area of lifelong development. This reflective account is written with a view of demonstrating one or more key roles in which a Clinical Psychologist should be competent (National Occupation Standards; 2002). These include the ability to implement professional and ethical standards and the application of psychological theory, methods and models which are empirically founded and evidenced based. This particular account focuses upon my reflections of learning a new model of thinking about other people thinking, namely using psychodynamic and attachment based approaches. Using Johns' Model of Reflection (1994) this account seeks to explore this ongoing learning process which I plan to continue pursuit of as a clinician. This account also takes stock of professional implications of my reflections.

**Chapter 4**  
**Advanced Clinical Practice 2**  
**Reflective Account**

*(Abstract only)*

***Multidisciplinary to Transdisciplinary Team Working  
and the Role of the Clinical Psychologist: A Reflective Account***

**Jennifer Shields**

July 2010

Submitted in part fulfilment of the requirements  
for the degree of Doctorate in Clinical Psychology

Address for correspondence:

Ms Jennifer Shields  
Division of Community Based Sciences  
Section of Psychological Medicine  
University of Glasgow  
Gartnavel Royal Hospital  
1055 Great Western Road  
Glasgow  
G12 0XH

## **ABSTRACT**

Multidisciplinary team working is an integral part of the role of the clinical psychologist. Both the Department of Health (2000) and British Psychological Society Guidelines (2007) advocate for integrated and joined up working practices in the care of patients. As a trainee clinical psychologist, working psychologically in teams has been an important area of my development. As a reflective practitioner, I have been mindful of my perspectives on team working and my reactions to individual experiences whilst working within teams and across disciplines. I have become increasingly aware of the inherent complexities which can accompany the benefits of integrated working practices. These reflections have broadened significantly within my final year advanced clinical practice placement. In addition to learning about multidisciplinary models of team functioning, I have become aware of emerging models of transdisciplinary processes within multidisciplinary teams. The following reflective account will seek to outline my experiences of team working in my final stages of training alongside developments in my thinking about the role of the clinical psychologist within such teams. Using the Rolfe et al (2001) framework for reflective practice, growth of key skills in this area will be discussed alongside my plans for future professional development. This aspect of skill acquisition will be discussed in line with national occupation standards (NOS 2001), which outline requirements of the psychologist in relation to team functioning and management.

## APPENDICES

### ***Contents***

<u>Appendix 1 – Systematic Review</u>	<i>Page Number</i>
1.1. Schizophrenia Bulletin - Information for Authors	85-88
1.2. Quality Rating Scale	89-92
<u>Appendix 2- Major Research Project</u>	<i>Page Number</i>
2.1. Journal of Neurology, Neurosurgery and Psychiatry Author's Instructions	93-96
2.2. Social Skills Group Questionnaire (Goldstein & Pollock 1988)	97-98
2.3. Research Approval Letter	99-100
2.4. Major Research Proposal (Submitted 4 <sup>th</sup> Sep 2009)	101-123

## **APPENDIX 1.1: Schizophrenia Bulletin - Information for Authors**



# Information for Authors

## MANUSCRIPT PREPARATION

All manuscripts are submitted and reviewed via the journal's web-based manuscript submission system accessible at <http://mc.manuscriptcentral.com/szbltn>. New authors should create an account prior to submitting a manuscript for consideration.

Manuscripts submitted to Schizophrenia Bulletin should be prepared following the American Medical Association Manual of Style, 10th edition. The manuscript text (including tables) should be prepared using a word processing program and saved as an .rtf or .doc file. Other file formats will not be accepted. Figures must be saved as individual .tif files and should be numbered consecutively (i.e., Figure 1.tif, Figure 2.tif, etc.). The text must be double-spaced throughout and should consist of the sections described below.

### Title Page

This page should consist of (i) the complete title of the manuscript, (ii) a running title not to exceed 50 characters including spaces, (iii) the full name of each author and the authors' institutional affiliations, (iv) name, complete address, telephone, fax, and e-mail address of the corresponding author, and (v) separate word counts of the abstract and text body.

### Manuscript Length

Manuscripts should be concisely worded and should not exceed 6,000 words for invited articles for theme issues, 4,500 words for regular articles, or 2,500 words for invited special features. The word count should include the abstract, text body, figure legends, and acknowledgments and must appear together with the abstract word count on the title page of the manuscript. Supplementary data, including additional methods, results, tables, or figures will be published online.

### Abstract

Provide a summary of no more than 250 words describing why and how the study, analysis, or review was done, a summary of the essential results, and what the authors have concluded from the data. The abstract should not contain unexplained abbreviations. Up to six key words that do not appear as part of the title should be provided at the end of the abstract.

### Main Text

Unsolicited original manuscripts reporting novel experimental findings should be comprised of these sections, in this order: Abstract, Introduction, Methods, Results, Discussion, Acknowledgments, References, and Figure Legends. Review articles must contain an abstract;

however, the body of the text can be organized in a less structured format. Authors of review articles are encouraged to use section headers to improve the readability of their manuscript.

Number pages consecutively beginning with the title page. Spelling should conform to that used in Merriam-Webster's Collegiate Dictionary, eleventh edition. Clinical laboratory data may be expressed in conventional rather than Système International (SI) units.

## Acknowledgments

These should be as brief as possible but include the names of sources of logistical support.

## References

Authors are encouraged to be circumspect in compiling the reference section of their manuscripts and to adhere to the following guidelines: Invited article for a theme: up to 50 references; Regular article: up to 40 references; Theme introduction and Special features: up to 25 references. Authors who anticipate submitting a manuscript with additional citations are encouraged to contact the editorial office before proceeding.

Each reference should be cited in consecutive numerical order using superscript arabic numerals, and reference style should follow the recommendations in the American Medical Association Manual of Style, 10th edition, with one exception: in the reference list, the name of all authors should be given unless there are more than 6, in which case the names of the first 3 authors are used, followed by "et al."

- Book: Talairach J, Tournoux P. Co-planar stereotaxic atlas of the human brain. New York, NY: Thieme Medical Publishers; 1998.
- Book chapter: Goldberg TE, David A, Gold JM. Neurocognitive deficits in schizophrenia. In: Hirsch SR, Weinberger DR, eds. Schizophrenia. Oxford, England: Blackwell Science; 2003:168-184.
- Journal article: Thaker GK, Carpenter WT. Advances in schizophrenia. Nat Med 2001;7:667-671.
- Journal article with more than 6 authors: Egan MF, Straub RE, Goldberg TE, et al. Variation in GRM3 affects cognition, prefrontal glutamate, and risk for schizophrenia. Proc Natl Acad Sci USA 2004;101:12604-12609.
- Article published on Advance Access only: Gilad, Y. and Lancet, D. March 5, 2003. Population Differences in the Human Functional Olfactory Repertoire. Mol Biol Evol doi: 10.1093/molbev/msg013.
- Article first published on Advance Access: Gilad, Y. and Lancet, D. 2003. Population Differences in the Human Functional Olfactory Repertoire Mol Biol Evol 2003;20:307-314. First published on March 5, 2003, doi: 10.1093/molbev/msg013.

Journal names should be abbreviated in accordance with Index Medicus ([www.nlm.nih.gov/tsd/serials/lji.html](http://www.nlm.nih.gov/tsd/serials/lji.html)).

Manuscripts in which the references do not follow this format will be returned for retyping. References to meeting abstracts, material not yet accepted for publication, or personal communications are not acceptable as listed references and instead should be listed parenthetically in the text. It is the authors' responsibility for obtaining the necessary permissions from colleagues to include their work as a personal communication.

Note: In the online version of Schizophrenia Bulletin there are automatic links from the reference section of each article to cited articles in Medline. This is a useful feature for readers, but is only possible if the references are accurate. It is the responsibility of the author to ensure the accuracy



of the references in the submitted article. Downloading references directly from Medline is highly recommended.

## Figures and Tables

Full length manuscripts including regular and invited theme articles should contain no more than a combined total of 5 tables and figures. Theme introductions and special features are limited to 2 tables or figures (total). Figures and tables must be referred to using arabic numbers in order of their appearance in the text (e.g., Figure 1, Figure 2, Table 1, Table 2, etc.).

Tables should be created with the table function of a word processing program; spreadsheets are not acceptable. Include only essential data, and format the table in a manner in which it should appear in the text. Each table must fit on a single manuscript page and have a short title that is self-explanatory without reference to the text. Footnotes can be used to explain any symbols or abbreviations appearing in the table. Do not duplicate data in tables and figures.

Please be aware that the figure requirements for initial online submission (peer review) and for reproduction in the journal are different. Initially, it is preferred to embed your figures within the word processing file or upload them separately as low-resolution images (.jpg, .tif, or .gif files). However, upon submission of a revised manuscript, you will be required to supply high-resolution .tif files for reproduction in the journal (1200 d.p.i. for line drawings and 300 d.p.i. for color and half-tone artwork). It is advisable to create high-resolution images first as these can be easily converted into low-resolution images for online submission. Figure legends should be typed separately from the figures in the main text document. Additional information on preparing your figures for publication can be located at <http://cpc.cadmus.com/da>.

Wherever possible figures should be submitted in their desired final size, to fit the width of a single (88 mm) or at most a double (180 mm) column width. All letters and numerals appearing in a particular figure should be of the same size and in proportion to the overall dimensions of the drawing. Letter labels used in figures should be in upper case in both the figure and the legend. The journal reserves the right to reduce the size of illustrative material.

## **APPENDIX 1.2: Quality Rating Scale for Systematic Review**

# A systematic review of the associations between theory of mind, executive function and schizophrenia

Quality rating scale for included studies

Article Number
Author & Year
Title
Reviewer
Total Score

Study Design:

Measures used:

Client Population(s):

What was the study question/aim? :

Study Findings:

<b>1. <u>Title and Abstract</u></b>	<b>Max Score = 2</b>
1.1 Does abstract include relevant diagnoses of participant group(s)?	Yes = 1 No = 0
1.2 Does abstract include adequate description of experimental design?	Yes = 1 No = 0
	Score <input style="width: 40px; border: 1px solid black;" type="text"/>
<b>2. <u>Introduction &amp; Objectives</u></b>	<b>Max Score = 2</b>
2.1 Does the introduction clearly outline background information and link this to a rationale for the study?	Yes = 1 No = 0
2.2 Are the hypotheses/aims/objectives of the study clearly described?	Yes = 1 No = 0
	Score <input style="width: 40px; border: 1px solid black;" type="text"/>
<b>3. <u>Design</u></b>	<b>Max Score = 2</b>
3.1 Is the study design appropriate to test the hypotheses?	Yes = 1 No = 0
3.2. Were settings/locations of data collection stated?	Yes = 1 No = 0
	Score <input style="width: 40px; border: 1px solid black;" type="text"/>

**4. Participants****Max Score = 9**

- 4.1 *Is the population, and how it was identified/recruited clearly stated?* Yes = 1 No = 0
- 4.2 *Did recruitment avoid convenience sample/bias wherever possible?* Yes = 1 No = 0
- 4.3 *Are participants' demographic and baseline clinical characteristics clearly described to allow adequate comparisons to be made?* Yes = 1 No = 0
- 4.4 *Were attempts made to minimise variation between groups (e.g. characteristics & baseline clinical characteristics included in the study demographically matched?)* Yes = 1 No = 0
- 4.5 *Is the population homogenous with respect to diagnosis?* Yes = 1 No = 0
- 4.6 *Are the inclusion/exclusion criteria clearly specified?* Yes = 1 No = 0
- 4.7 *Are participants matched to an appropriate control / comparison group?* Yes = 1 No = 0
- 4.8 *Was an accepted diagnostic criteria used to confirm diagnosis (e.g. DSM – IV)?* Yes = 1 No = 0
- 4.9 *Were potential co-morbid psychological disorders/symptoms measured / screened for using a reliable and valid tool (e.g. BDI-II)?* Yes = 1 No = 0

Score **5. Assessment Measures****Max Score = 5**

- 5.1 *Does the study use measures of theory of mind and executive function as a primary outcome measure?* Yes = 1 No = 0
- 5.2 *Were measurement tools valid, reliable and sensitive to change?* Yes = 1 No = 0
- 5.3 *Was more than one standardised measure of theory of mind utilised?* Yes = 1 No = 0
- 5.4 *Was more than one standardised measure of executive function utilised?* Yes = 1 No = 0
- 5.5 *Have measurement tools been used at appropriate time points in relation to the design and focus of the study?* Yes = 1 No = 0

Score **6. Results****Max Score = 6**

- 6.1 *Is there adequate reporting of descriptive statistics (i.e. means, standard deviations)?* Yes = 1 No = 0
- 6.2 *Were the statistical analyses used to assess the main outcomes appropriate ( e.g. multivariate with ancillary analyses if appropriate) and clearly related to the study aims, questions and hypotheses?* Yes = 1 No = 0
- 6.3 *Have power calculations been carried out to assess the*

<i>required cohort size?</i>	Yes = 1 No = 0
6.4. <i>Were effect sizes calculated?</i>	Yes = 1 No = 0
6.5. <i>Were effect sizes medium or large?</i>	Yes = 1 No = 0
6.6. <i>Did the results relate to the initial hypothesis?</i>	Yes = 1 No = 0
<b>Score</b>	<input type="text"/>

---

### **7.0 Conclusions**

**Max Score = 4**

7.1 <i>Does the study relate the results directly to the original aim(s), research question (s) and hypothesis(es)?</i>	Yes = 1 No = 0
7.2 <i>Do the conclusions drawn directly link to the results achieved?</i>	Yes = 1 No = 0
7.3 <i>Are recommendations for clinical practice or future research discussed in relation to the findings?</i>	Yes = 1 No = 0
7.4 <i>Are the limitations of the study clearly expressed?</i>	Yes = 1 No = 0

**Score**

---

**Total Score:**

**Overall Quality Rating:**

A – High Quality (>70%+)  
 B – Moderate Quality (40-70%)  
 C – Low Quality (0 - 39%)

**APPENDIX 2.1: Journal of Neurology, Neurosurgery and Psychiatry  
Author's Instructions**



## Manuscript format

The manuscript format must be presented in the following order:

1. Title page
2. Abstract (or summary for case reports)
3. Main text (tables should be in the same format as your article and embedded into the document where the table should be cited; images must be uploaded as separate files)
4. Acknowledgments, Competing interests, Funding
5. Copyright licence statement
6. References
7. Appendices

Do not use the automatic formatting features of your word processor such as endnotes, footnotes, headers, footers, boxes etc.

Provide appropriate headings and subheadings as in the journal. We use the following hierarchy: **BOLD CAPS**, **bold lower case**, Plain Text, *Italics*.

Cite illustrations in numerical order (fig 1, fig 2 etc) as they are first mentioned in the text.

Tables should be in the same format as your article and embedded into the document where the table should be cited.

Images **must not** be embedded in the text file but submitted as individual files (view further details in File Formats.)

## Statistics

Statistical analyses must explain the methods used.

[Guidelines on presenting statistics.](#)

[Guidelines on RCTs: CONSORT, QUORUM, MOOSE, STARD, and Economic submissions.](#)

## Style

Abbreviations and symbols must be standard and SI units used throughout except for blood pressure values which are reported in mm Hg.

Whenever possible, drugs should be given their approved generic name. Where a proprietary (brand) name is used, it should begin with a capital letter.

Acronyms should be used sparingly and fully explained when first used.

[View more detailed style guidelines.](#)

## Figures/illustrations

**Black and white** images should be saved and supplied as **GIF, TIFF, EPS or JPEG** files, at a **minimum resolution of 300 dpi** and an image size of 9 cm across for single column format and 18.5 cm for double column format.

**Colour** images should be saved and supplied as **GIF, TIFF, EPS or JPEG** files, to a **minimum resolution of 600 dpi** at an image size of 9 cm across for single column format and 18.5 cm for double column format.

Images should be mentioned in the text and figure legends should be listed at the end of the manuscript.

During submission, when you upload the figure files please label them as Figure 1, Figure 2, etc. The file label will not appear in the pdf but the order in which the figures uploaded should be sufficient to link them to the correct figure legend for identification.

We can accept multi-page Powerpoint files. Alternatively, Powerpoint files can be saved as JPEG files and submitted as a standard image file.

Histograms should be presented in a simple, two-dimensional format, with no background grid.

### Unacceptable file formats

Any file using OLE (Object Linking and Embedding) technology to display information or embed files, Bitmap (.bmp), PICT (.pict), Photoshop (.psd), Canvas (.cnv), CorelDRAW (.cdr); Excel (.xls); and locked or encrypted PDFs are not acceptable.



## Tables

Tables should be submitted in the same format as your article and embedded into the document where the table should be cited. Please note: Bench>Press **cannot** accept Excel files. If your table(s) are in Excel, copy and paste them into the manuscript file. In extreme circumstances, Excel files can be uploaded as supplementary files; however, we advise against this as they will not be acceptable if your article is accepted for publication.

Tables should be self-explanatory and the data they contain must not be duplicated in the text or figures.

## References

Authors are responsible for the accuracy of references cited: these should be checked against the original documents before the paper is submitted. It is vital that the references are styled correctly so that they may be hyperlinked.

Punctuation of references must follow the [slightly modified] Vancouver style:  
12 Surname AB, Surname CD. Article title. Journal abbreviation. Year;Vol:Start page-End page.

Use one space only between words up to the year and then no spaces. The journal title should be in italic and abbreviated according to the style of Medline. If the journal is not listed in Medline then it should be written out in full.

[Check journal abbreviations using PubMed.](#)

**APPENDIX 2.2: Social Skills Group Questionnaire (SSGQ)**  
**(Goldstein & Pollock, 1988)**

## SOCIAL SKILLS GROUP ASSESSMENT QUESTIONNAIRE

(Goldstein and Pollock 1988)

**Directions:** Listed below are lists of social skills which are important for social competence. Please read the description of each skill and circle the answer which best describes your opinion of \_\_\_\_\_ social skills. Please use the space at the end of the questionnaire if you feel there are additional social related problems which should be noted.

SOCIAL SKILL	is very poor at this skill	exhibits this skill as well as others	exhibits this skill better than others
Meeting new people	1 or 2	3 or 4	5 or 6
Beginning a conversation	1 or 2	3 or 4	5 or 6
Listening during a conversation	1 or 2	3 or 4	5 or 6
Ending a conversation	1 or 2	3 or 4	5 or 6
Joining an ongoing activity with others	1 or 2	3 or 4	5 or 6
Asking questions appropriately	1 or 2	3 or 4	5 or 6
Asking for a favour appropriately	1 or 2	3 or 4	5 or 6
Seeking help from peers appropriately	1 or 2	3 or 4	5 or 6
Seeking help appropriately	1 or 2	3 or 4	5 or 6
Sharing	1 or 2	3 or 4	5 or 6
Interpreting body language	1 or 2	3 or 4	5 or 6
Playing a game successfully	1 or 2	3 or 4	5 or 6
Suggesting an activity to others	1 or 2	3 or 4	5 or 6
Working cooperatively	1 or 2	3 or 4	5 or 6
Offering help to others	1 or 2	3 or 4	5 or 6
Saying thank you	1 or 2	3 or 4	5 or 6
Giving a compliment	1 or 2	3 or 4	5 or 6
Accepting a compliment	1 or 2	3 or 4	5 or 6
Apologising	1 or 2	3 or 4	5 or 6
Understanding the impact his or her behaviour has upon others	1 or 2	3 or 4	5 or 6
Demonstrating the ability to understand others' behaviour	1 or 2	3 or 4	5 or 6
Rewards self	1 or 2	3 or 4	5 or 6
Follows directions	1 or 2	3 or 4	5 or 6
<b>COMMENTS</b>			

**APPENDIX 2.3: Final Ethics Procedure Management Approval Letter  
(Greater Glasgow & Clyde R&D / West of Scotland Ethics Committee, 2009)**



**Greater Glasgow  
and Clyde**  
R&D Management Office  
Western Infirmary  
Tennent Institute  
1st Floor, 38 Church Street  
Glasgow, G11 6NT

Coordinator/administrator: Darren Gibson/Elaine O'Donnell  
Telephone Number: 0141 211 6208  
Fax Number: 0141 211 2811  
E-Mail: [Darren.Gibson@ggc.scot.nhs.uk](mailto:Darren.Gibson@ggc.scot.nhs.uk)

23 February 2010

Miss Jennifer Shields  
Trainee Clinical Psychologist  
Gartnavel Royal Hospital  
1055 Great Western Road  
Glasgow G12 0XH

### **R&D Management Approval**

Dear Miss Shields

**Project Title:** Theory of mind & executive function in autism & acquired brain injury: A comparative study.  
**Chief Investigator:** Miss Jennifer Shields  
**R&D Reference:** GN09CP544  
**Protocol:** Version 1, 17 Sept 09

I am pleased to confirm that Greater Glasgow & Clyde Health Board is now able to grant **Management Approval** for the above study.

As a condition of this approval the following information is required during the lifespan of the project:

1. SAES/SUSARS – If the study is a **Clinical Trial** as defined by the Medicines for Human Use Clinical Trial Regulations, 2004 (CTIMP only)
2. Recruitment Numbers on a quarterly basis (not required for commercial trials)
3. Any change of Staff working on the project named on the ethics form
4. Change of CI
5. Amendments – Protocol/CRF etc
6. Notification of when the Trial / study has ended
7. Final Report
8. Copies of Publications & Abstracts

**Please add this approval to your study file as this letter may be subject to audit and monitoring.**

Yours sincerely

Dr Darren Gibson  
Research Co-ordinator

***Delivering better health***

[www.nhsggc.org.uk](http://www.nhsggc.org.uk)

## **APPENDIX 2.4: Major Research Proposal (Submitted 4<sup>th</sup> Sep 2009)**

# **Major Research Proposal**

Submission Date: 4<sup>th</sup> September 2009

## ***Theory of Mind & Executive Function in Autism and Traumatic Brain Injury A Comparative Study***

*Jennifer Shields*  
Trainee Clinical Psychologist

Research Supervisors  
*Professor Tom McMillan*  
*Professor Andrew Jahoda*

Word Count – 3659

## Abstract

**Background:** Difficulties inferring others' mental states, broadly conceptualised as deficits of theory of mind (ToM), is an area widely researched within Autistic Spectrum Disorders (ASD). Relative to this vast literature base is a smaller yet increasing number of studies investigating the theory of mind profiles and deficits within other clinical populations. Consequently, there is limited evidence to demonstrate that these deficits originate from the same causal mechanisms. The generalisability therefore, of interventions for theory of mind deficits designed for ASD populations to client groups such as individuals with Traumatic Brain Injury (TBI) is at present unsubstantiated. **Aims:** The primary aim of this study is to compare the theory of mind profiles of individuals with ASD, TBI versus a control group. It is hypothesised that theory of mind deficits will be impaired in both ASD and TBI groups in comparison to controls. It is also hypothesised that the profile of ToM deficits will differ between clinical groups. A secondary aim is to compare groups on aspects of executive functioning which are speculated to underlie theory of mind deficits, namely attention, inhibition, and cognitive flexibility. It is hypothesised that TBI and ASD groups will differ with respect to these associated profiles of executive function. **Methods:** This study will comprise a between groups design of 17 participants with TBI, 17 participants with ASD and a control group of 17 participants without ASD or TBI. Measures will comprise the Mind in the Eyes Test, non verbal false belief tests, emotion recognition tests and assessments of executive function. Control measures of mood and general intellectual ability will also be administered. **Applications:** It is hoped that findings will contribute to the growing literature on ToM deficits in TBI whilst allowing comparisons to



ASD populations where these difficulties are well evidenced. This may inform the possible benefits of augmenting existing interventions for ToM deficits currently used with individuals with ASD for use with other clinical populations.

## **Introduction**

Theory of Mind, conceptualised as the ability to infer the mental states of others or mentalise is widely regarded as the means by which human beings interact successfully within social situations (Wellman, 1990). The capacity to interpret behaviour, mood states and to plan responses is generally viewed as a form of social intelligence which is regarded as distinct from general intelligence. The process of cognitive development from egocentrism to the ability to mentalise is generally thought to develop slowly from around the age of 5 (Piaget, 1956). Recent research, however using false belief tasks which assess ability to distinguish between appearance and reality indicate a rapid advancement in this during the third year of age. This has led psychologists to propose that the underlying cognitive structure responsible for Theory of Mind is an innate module which is activated around this time (Baron-Cohen, 1995). Although this would imply a degree of neural independence, the identification of the cognitive modalities or neuroanatomical locations underpinning this capacity is an area of continued debate.

The regions of the brain hypothesised to engage in Theory of Mind ability vary widely. Recent functional magnetic resonance imaging studies have postulated frontal areas such as the orbitofrontal cortex and the amygdala (Baron-Cohen, 1999; Bibby & McDonald, 2005). Functional imaging has consistently implied

the involvement of three particular areas; these are the anterior paracingulate cortex, the superior temporal sulci and the temporal poles bilaterally (Gallagher and Frith 2004). Other theorists propose activations of posterior regions as a means of forming representations, with an executive component of action initiation served by prefrontal regions (Abu-Akel, 2004). Recent research has also focused upon dorsal and ventral streams of visual processing and their influence upon pattern recognition and integration of information (Frith & Frith, 2006). Although the literature frequently refers to the involvement of executive abilities, some case studies can be found depicting lesions in frontal regions whilst reporting theory of mind skills as remaining intact (Bird et al. 2004). Despite some findings suggesting independence from executive function, there is growing evidence of theory of mind difficulties in clinical groups who are known to have frontal lobe and executive impairments. These include individuals with schizophrenia (Corrigan, 1997), dementia, Multiple Sclerosis (Henry et al. 2009) and traumatic brain injury (Shaw et al. 2004; Milders et al. 2003, Henry et al. 2006).

The study of deficits in Theory of Mind has emerged predominantly from studies of individuals with autistic spectrum disorder (ASD) who frequently present with difficulties with theory of mind (ToM). Without the flexibility to think beyond prototypical representations of information or to consider complex conceptual themes and patterns of interaction, individuals with ASD are viewed as intuitively disadvantaged socially (Baron-Cohen, 1999). At present, the construct of 'Theory of Mind' is used in ASD literature interchangeably with terms such as 'mind-blindedness' and 'mind-reading' (Baron-Cohen, 2001).

These concepts serve to capture the broad range of difficulties individuals on the autistic spectrum have with ToM. Although the same concepts are used to describe similar difficulties in other populations, there is limited evidence to surmise that the deficits stem from the same source or affect the same range of aspects of ToM.

Traumatic brain injury in particular has been shown to precipitate a range of social skills deficits which are evidenced to stem from difficulties with theory of mind (Milders et al. 2003; Milders, Fuchs & Crawford, 2003). Despite parallels to difficulties found in ASD populations, McDonald and Martin (2003), highlight a shortage of studies which look concurrently at different populations who present with similar pragmatic social and/or language difficulties (Astington, 1994; Astington, Harris & Olson, 1988; Baron-Cohen, 1995; Baron-Cohen, Tager-Flusberg & Cohen, 1993). This has subsequently led to a lack of theoretical accounts which are applicable to other populations who display theory of mind difficulties (McDonald & Martin 2003).

In contrast, there is a plethora of theories which attempt to explain theory of mind difficulties in ASD populations. These include Social Inference Theory (Brownell & Martino 1998), Weak Coherence Theory (Happe & Frith, 2006) and Executive Dysfunction Theory (Happe, 1996; Russell 1997) to name but a few. Despite a strong effort to establish theoretical underpinnings, cognitive modalities or sub-systems contributing to 'theory of mind', there is still much debate in terms of what functions are necessary and sufficient for skills of theory of mind (Baron Cohen, 1995). Within ASD literature, some advances in

research area have postulated a profile of deficits using discriminant functional analysis. This has outlined deficits in complex language, complex memory, reasoning and concept formation (Minshew, Meyer & Gold, 2002). Although these higher/executive functions have been postulated to provide the mechanisms required for theory of mind (Snowden et al. 2003), there is also evidence to suggest that “conceptually executive function may be too broad a level of analysis” (Baron-Cohen, 1997; p.16). Conversely, as some evidence indicates that ToM skills may be independent of executive function (Bach et al. 2000), it appears necessary to investigate ToM profiles using a more detailed level of analysis.

Given the assessments broadly used in studies of ToM are primarily designed for individuals with ASD (Bibby & MacDonald, 2005) it is proposed that the cross referencing of parallels and differences between clinical populations may be hindered by a lack of specificity in the assessment tools. Despite the similarities evidenced by individuals with ASD and TBI at an observable pragmatic social level (see Martin & MacDonald 2003 for a review) there are no known studies which contrast theory of mind and/or executive abilities directly between populations using specific measures within a focused approach. This may have contributed to the difficulties generalising theories from ASD populations or to propose alternative models.

In summary, there is no evidence to indicate that the pragmatic social difficulties these populations experience stem from the same aspects of theory of mind; to the same degree, or are related to deficits in the same underlying mechanisms.

Given that executive dysfunction does not lead to autism, it can be suggested that the theory of mind deficits found in individuals with ASD are not fully generalisable to those found in individuals with TBI. The rationale for this particular research study, therefore, is to explore, using specific measures, differences between clinical populations with respect to theory of mind deficits and executive function. By contrasting individuals with TBI to a population where Theory of Mind difficulties are widely evidenced, this may offer further insights into how these problems can be conceptualised and intervened with.

## **Aim & Hypotheses**

### **Aim**

It is the primary aim of this study; therefore to compare the theory of mind profiles of individuals with traumatic brain injury (TBI) with participants with Aspergers Disorder and a control group. A secondary aim is to compare groups on aspects of executive functioning which are speculated to underlie theory of mind deficits, namely attention, inhibition, and cognitive flexibility.

### **Hypotheses**

- 1) Theory of mind deficits will be impaired in both ASD and TBI groups in comparison to controls.
- 2) The profile of ToM deficits will differ between clinical groups: TBI participants will perform better than ASD participants on all Theory of Mind Tests namely Reading the Mind in the Eyes, Facial Affect Recognition and false belief tests.

3) TBI and ASD groups will differ with respect to executive function: The TBI group will be deficient in comparison to the ASD group on executive functions subtests of attention and inhibition. This will be illustrated with the Hayling subtest of Dysexecutive Syndrome, and on selected subtests sub tests of the Delis Kaplan Executive Function Test.

## **Plan of Investigation**

### **Participants**

Participants will comprise 17 adult participants who have a diagnosis of Aspergers Disorder, 17 who have Traumatic Brain Injury (TBI) and a control group of 17 participants who have neither ASD nor TBI. These individuals will ideally be siblings or family members of participants with ASD/TBI.

ASD and TBI Services will be approached within the Greater Glasgow and Ayrshire Health Board areas. This will include Greater Glasgow Social Services, West Dunbartonshire Social Services, Headway and the Scottish Society for Autism. Initial contact will be made via emails to heads of service prior to ethics submission to ascertain feasibility of recruitment with respect to the required numbers above.

### **Inclusion/Exclusion Criteria**

#### *Inclusion criteria*

1. Participants within the ASD group will have a diagnosis of Aspergers Disorder as outlined in the criteria within the DSM-IV.

2. Participants within the TBI group will have been diagnosed with a severe Traumatic Brain Injury as classified by the Glasgow Coma Scale. A severe injury will necessitate a period of Post Traumatic Amnesia of > 1 day (Russell and Nathan, 1946).
3. Participants will be adults aged 16 – 64 years old.
4. Participants first language is English.
5. Participants are functioning above the learning disability range (IQ > 70) and are able to consent to taking part in the study.

#### *Exclusion criteria*

1. Individuals who have been deemed to lack capacity under the Adults with Incapacity Act and are under the supervision of a guardianship order.
3. Any participant who has displayed significant levels of risk to self or others or symptoms of trauma will also not be included in this study.
3. Participants will be excluded if they have a current chronic psychiatric condition.
4. Individuals with TBI will be excluded if they also have a diagnosis of ASD.
5. Individuals with ASD will be excluded if they have also acquired TBI.

#### **Recruitment Procedures**

Participants will be recruited from local TBI and ASD Services between Ayrshire and Glasgow NHS Trusts and associated partnership organisations. Siblings of suitable participants will also be approached to comprise an appropriate control group. All potential participants will be sent an information sheet outlining the aims of the study and a letter of invitation with an attached consent form. If

returned, this consent form would indicate interest in participating in the study and consent for their diagnosis of TBI/ASD (where appropriate) to be verified and their suitability for inclusion assessed. If included in the study participants will then be sent a letter inviting them to meet the main researcher at a local clinical setting. This letter will be followed up by a telephone call to confirm attendance and to check for any special requirements and to check if the participant has any questions which will inform their choice to participate.

## **Measures**

The following measures will be administered during one ninety minute assessment session.

### Dependent Variable Measures

#### *Tests of Theory of Mind*

- Reading the Mind in the Eyes Test – *a measure of adult mentalising capability.*
- Faces Test - *measure of affect recognition.*
- 1<sup>st</sup> and 2<sup>nd</sup> order False Belief Tasks (Cartoon Format.) – *These measure a person's social cognitive ability to attribute false beliefs to others in a non verbal format.*

#### *Tests of Executive Function*

- Hayling Test of Dysexecutive Syndrome - *a measure of response initiation and response suppression.*



- Dysexecutive Questionnaire (DEX) from the Behavioural Assessment of Dysexecutive Syndrome. - A 20 item 'Dysexecutive questionnaire' (DEX). *Covers four broad areas of likely change: emotional or personality changes, motivational changes, behavioural changes, and cognitive changes. The DEX comes in two forms, one designed to be completed by the patient and one by a relative or carer.*
- Subtests of Delis-Kaplan Executive Function Test: *Trail Making, Verbal Fluency Test & Tower Test.*

#### *Control Measures*

- Hospital Anxiety and Depression Scale – *to control for cognitive interference due to the presence of a mood disorder.*
- Wechsler Test of Adult Reading (WTAR) – *to establish pre-morbid IQ.*

#### **Design**

This is a between groups design comprising individuals with TBI, ASD and a control group. Participants will ideally be matched upon variables of IQ and age.

#### **Research Procedures**

Following the application of inclusion and exclusion criteria, selected participants will be invited by letter to attend a clinic for a two hour assessment session. This letter will also enclose two Dysexecutive Questionnaires from the Behavioural Assessment of Dysexecutive Syndrome (DEX) & the Hospital Anxiety and Depression Scale (HADS) for completion prior to attendance. The

former is designed for completion by a family member/carer or close acquaintance. An alternative format is for the individual as a means of self reporting difficulties. Participants will be asked to return these by reply via a Stamped Addressed Envelope or in person upon arrival at the clinic. Participants can also opt to complete the HADS and the DEX with support in clinic.

Upon presenting for assessment, participants will be given an additional verbal explanation of the study and reminded of their right to opt out of the study at any time or to shorten the session length depending on their levels of comfort. If participants are happy to proceed, the main researcher will administer the aforementioned tests and control measures. If participants are observed to be struggling to attend to tasks or seem fatigued, the main researcher will discuss this with the participant and offer further breaks/session if this will facilitate the participant to complete the study. Participants will be reminded of their choice to terminate the session at any time.

### **Justification of Sample Size**

Studies of Theory of Mind Deficits in Traumatic Brain Injury formed the basis for power calculations using G-Power from the UCLA website. As there are no previous studies comparing TBI and ASD groups, the following studies were chosen due to their use of similar measures and subsequent methods of analysis.

Henry et al. (2006), conducted a study of TBI versus a control group ( $n = 16$  &  $17$  respectively). This study correlated verbal fluency executive function tests and mental state attribution tests examining 'affective versus cognitive' aspects of Theory of Mind using the Mind in the Eyes Test. For the theory of mind dependent measure the Control group mean score ( $25.9$ ,  $SD\ 4.06$ ), was compared to a TBI group mean score ( $22.4$ ,  $SD\ 6.49$ ) to give an overall effect size of  $0.66$ . Power calculations for the present study based on this data indicates that a minimum of  $60$  participants in total would be necessary for a power of  $0.8$  and an alpha of  $0.05$ .

Milders, Fuchs & Crawford, (2003) also used the Mind in the Eyes Test. Again this involved a TBI group ( $n = 16$ ) and a control group ( $n = 17$ ). This study had an effect size of  $0.68$  for inferring mental states from the eyes and for face recognition  $d = 1.17$ . (It should be noted that no corrections were implemented for multiple comparisons to correct for Type 1 error). Power calculations for this present study, however, indicate that  $56$  participants in total would be necessary to achieve a power of  $0.8$  and an alpha of  $0.05$ .

With respect to executive function, again there have been no previous studies directly comparing ASD and TBI. Looking at the performance of these groups separately, the measures used have comprised broad measures of both executive function and intellectual ability. One paper illustrating this is by Kleinmans, Akshoomoff & Delis, (2005) that look at Autistic populations. Calculations from this paper indicate medium effect sizes within subtests of the Delis Kaplan which would suggest the need for large samples.. As this study is

focusing upon specific aspects of executive function, namely inhibition, attention and cognitive flexibility in relation to theory of mind, it is expected that this more focal approach using more homogenous groups (Aspergers versus TBI) will allow for smaller participant groups.

As this study is looking to explore differences between TBI & ASD groups using specific measures of pre-identified aspects of executive function, , it seems appropriate to initially plan recruitment of similar numbers of participants as the aforementioned studies. Considering issues of feasibility in relation to time, it seems viable that a minimum of 17 participants recruited to each of the control, TBI and ASD groups respectively will be sufficient to achieve a power of 0.8 and an alpha of 0.05.

### **Settings and Equipment**

All assessments will be undertaken within an NHS/Local Authority setting which is familiar with the client if possible. Domiciliary visits will not be undertaken and the researcher/participant will not be in an isolated environment. Equipment will consist of the aforementioned tests and associated materials.

### **Data Analysis**

Data will be analysed using SPSS statistical software. In order to investigate whether the predicted differences exist between the TBI, ASD and control groups, inferential statistical analyses will be carried out to look at variance between groups in terms of differences in performance on dependent variable measures. Parametric inferential statistics will ideally include ANOVA with

investigations of interaction effects between group and test type. It may also be necessary to convert scores to composite scores for the purpose of analysis. Post hoc corrections will also be administered as required for multiple comparisons. Non parametric tests of statistical significance will only be chosen when assumptions of normal distribution and homogeneity of variance are violated.

### **Health and Safety**

#### **Researcher Safety Issues**

Levels of risk to both participants and researcher will be assessed on an ongoing basis. The researcher will meet participants within a pre-arranged clinical setting which has been deemed acceptable for purpose.

#### **Participant Safety Issues**

Participants who, at the time of participation, are exhibiting marked aggression, self harm, marked disinhibition or any other behaviour deemed inappropriate or posing risk to self or others will be excluded from the study. All clinical interviews or assessments will take place within a clinical setting. Participants will be reminded of their choice to leave the session at any time and to request breaks or adjournment at their leisure. Measures will be taken at all times to ensure client confidentiality and safety.

#### **Ethical Issues**

Potential issues will surround aspects relating to capacity and risk. Capacity and levels of risk will be established prior to attending assessment. Given the

length of time required by each participant in terms of assessment, it will also be necessary to monitor participants for levels of fatigue or discomfort. Although the length of assessment is shorter than standard neuropsychological assessments, the researcher will be a Clinical Psychologist in final year of training who is skilled to monitor and appropriately manage clients who display fatigue or marked difficulties related to their condition. Regular breaks will be offered and the assessment paced to support the client to complete assessment comfortably. Any issues presented by clients who cause concern will be referred to an appropriate source with supervision from Professor Tom McMillan.

### **Ethical Approval and Management Submissions**

Application to the Greater Glasgow Local Research Governance and Ethics department will follow approval of this Proposal by the Department of Psychological Medicine. This will also involve both NHS Greater Glasgow & Clyde and NHS Ayrshire & Arran Departments of Research & Development via the Integrated Research Application System (IRAS).

### **Financial Issues**

Assessments of executive function, IQ and mood will be sourced from the Psychological Departmental resources. Assessments of Theory of Mind will be resourced from the Autism Research Centre where they will be downloaded for research purposes at no charge. Additional funding will be requested to cover costs of stationary, postage, travel expenses and standardised assessment recording sheets (see attached costing sheet).

**Timetable**

Ethics approval will be sought in September/October 2009. Study set up will follow in late October and recruitment ideally in early November 2009. Assessment sessions will run November 2009-April 2010 and analyses undertaken by May 2010. Write up of the project will continue from May to July 2010 with amendments being made in August 2010 for final submission.

**Practical Applications**

It is hoped that this study will provide evidence to inform the clinical utility of interventions for theory of mind deficits which are currently offered within ASD client groups for individuals with Acquired Brain Injury. It is hoped that gaining a fuller understanding of the apparent difficulties which underpin theory of mind may inform and refine future interventions

## References

- Abu-Akel, A.(2003) A Neurobiological Mapping of Theory of Mind. *Brain Research Reviews*, 43, pp. 29–40.
- Astington, J. (1994) *The Child's Discovery of the Mind*. Cambridge, Harvard University Press.
- Astington, J., Harris, P., & Olson, D. (1988). *Developing theories of mind*. New York: Cambridge University Press.
- Bach L., Happé F., Fleminger S., Powell J. (2000) Theory of mind: independence of executive function and the role of the frontal cortex in acquired brain injury. *Cognitive Neuropsychiatry*, 5: pp.175–92.
- Baron-Cohen, S. (1995) *Mindblindness: an essay on autism and theory of mind*. Boston, MIT Press / Bradford Books.
- Baron-Cohen, S. Wheelwright, S. & Jolliffe, T. (1997) Is there a language of the eyes? Evidence from normal adults and adults with autism or Asperger syndrome. *Visual Cognition*, 4, pp. 311-331.
- Baron-Cohen, S. Tager-Flusberg, H., & Cohen, D. (Eds.) (1993). *Understanding other minds: perspectives from autism*; Oxford University Press.



Baron-Cohen, S. et al (1999) *Social Intelligence in the normal and autistic brain: an fMRI study. European Journal of Neuroscience*, Vol. 11, pp. 1891-1898.

Baron-Cohen, S. (2001) *Special Issue of the International Review of Mental Retardation*; pp. 23-169.

Bibby, H. & McDonald, S. (2005) Theory of Mind after traumatic brain injury. *Neuropsychologia*, 43, pp. 99-114.

Bird C. M., Castelli F., Malik O., Frith U., Husain M. (2004) The impact of extensive medial frontal lobe damage on 'Theory of Mind' and cognition. *Brain*, 127(Pt 4), pp. 914-28.

Brownell, H. & Martino, G. (1998) Deficits in inference and social cognition: The effects of right hemisphere brain damage on discourse. In M. Beeman, & C. Chiarello (Eds.) *Right hemisphere language comprehension: Perspectives from cognitive neuroscience*, pp. 309-328) Lawrence Erlbaum Associates.

Corrigan, P. (1997) The social perceptual deficits of schizophrenia. *Psychiatry*, 60, pp. 309–326.

Delis, D.C., Kaplan, E., & Kramer, J.H. (2001) *Delis Kaplan Executive Function System*. San Antonio, Texas: The Psychological Corporation.

Frith, U. (1989) *Autism: explaining the enigma*. Oxford, Basil Blackwell.

Frith, C.D., Frith, U. (2006) How we predict what other people are going to do. *Brain Research*, 1079, (1), pp.36-46.

Gallagher H.L. & Frith C.D. (2004) Dissociable neural pathways for the perception and recognition of expressive and instrumental gestures. *Neuropsychologia*, 42, (13), pp. 1725-1736.

Happe, F., Frith, U. (2006) The weak coherence account: detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 35, (1), pp.5-25.

Happe, F. (1996) Studying weak central coherence at low levels: children with autism do not succumb to visual illusions: A research note. *Journal of Child Psychology and Psychiatry*, 37, pp. 873-877..

Henry, J.D. et al. (2006) Theory of mind following traumatic brain injury: the role of emotion recognition and executive dysfunction, *Neuropsychologia*, 44, (10), pp. 1623-8.

Henry, J.D. et al. (2009) Evidence for deficits in facial affect recognition and theory of mind in multiple sclerosis. *Journal of the International Neuropsychological Society*, (2), pp 277-85.

Kleinmans, N., Akshoomoff, N. Delis, D.C. (2005) Executive Functions in Autism and Asperger's Disorder: Flexibility, Fluency and Inhibition. *Developmental Neuropsychology*, Vol. 27, 3, pp. 379-401.

McDonald, S. & Martin, I. (2003) Weak coherence, no theory of mind, or executive dysfunction? Solving the puzzle of pragmatic language disorders. *Brain & Language*, (85) pp. 451-466.

Milders, M. et al (2003) Neuropsychological Impairments and Changes in Emotional and Social Behaviour Following Severe Traumatic Brain Injury. *Journal of Clinical and Experimental Neuropsychology*, Vol. 25, No 2, pp. 157-172.

Minshew, N. J., Meyer, J., & Goldstein, G (2002) Abstract reasoning in autism: A dissociation between concept formation and concept identification. *Neuropsychology*, 16, pp. 327-334.

Piaget, J. (1955) *The Child's Construction of Reality*. London: Routledge

Shaw et al. (2004) The Impact of early and late damage to the human amygdala on 'theory of mind' reasoning. *Brain*, 127, pp 1535-1548.

Snowden J.S., Gibbons Z.C., Blackshaw A., Doubleday E., Thompson J., Crawford D, et al.(2003) Social cognition in fronto-temporal dementia and Huntington's disease. *Neuropsychologia*, 41, pp 688–701.

Tager-Flusberg, H. (1993). What language reveals about the understanding of minds in children with autism. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding other minds: perspectives from autism*, Oxford University Press.

Russell, W.R., Nathan, P.W. (1946) Amnesia following head injuries. *Lancet*, 2, pp. 280-300

Russell, J. (Ed.). (1997) *Autism as an executive disorder*. Oxford: Oxford University Press.

Wellman, H. M. (1990) *The child's theory of mind*. Cambridge, MA: MIT Press.

Wechsler, D. (2001) *Wechsler Test of Adult Reading*. NFER Nelson, London.

Wilson, B.A., Alderman, N., Burgess, P.W., Emslie, H.E., & Evans, J.J. (1996) *Behavioural Assessment of Dysexecutive Syndrome*. Bury St Edmunds, UK: Thames Valley Test Company.

Zigmond, A.S., & Snaith, R.P. (1983) The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, Vol. 67, pp. 361-370.