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**Asperger's Syndrome and Traumatic Brain Injury: The role of  
Anxiety in Executive Function and Theory of Mind Deficits  
and  
Clinical Research Portfolio**

**VOLUME I**

(Volume II bound separately)

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Institute of Health and Wellbeing  
University of Glasgow  
June 2014

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

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## Chapter 1

### Systematic Literature Review

#### **Prevalence of anxiety in autism spectrum disorders in clinical populations: A systematic review**

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Prepared according to instructions for *Research in Autism Spectrum Disorders* (see Appendix 1.1)

## Abstract

**Purpose:** Current literature in autism spectrum disorders (ASD) suggests that individuals suffer from anxiety disorder at a higher rate than in the general population and several factors specific to ASD may contribute to this association. However, no studies to date have examined the prevalence of anxiety disorder in a population based ASD sample. In the absence of such research, this systematic review considers the evidence addressing the question of prevalence of anxiety disorder in adults with ASD presenting in clinical settings. It also examines evidence relating to potential factors mediating the relationship between anxiety disorders and ASD.

**Methodology:** An electronic and manual search identified 12 articles which fulfilled inclusion and exclusion criteria. Methodological quality was assessed based on a combination of published guidelines for systematic reviews.

**Results:** This review highlights the dearth of adequate population-based prevalence studies of anxiety in ASD. Findings of prevalence in clinical settings appear to be dependent on recruitment source, participant variables, study design and measurement of anxiety. The prevalence rate among a clinical sample of adults with ASD could not be established due to the heterogeneity of methods used. Confounding factors that impact on the relationships between ASD and anxiety are explored.

**Conclusions:** The scarcity of good quality studies investigating the role of anxiety in adults with ASD suggest we have yet to determine phenomenology and aetiology. Causal relationships need to be established before we can develop treatments for a condition that is not yet understood.

## **1. Introduction**

The recent publication of the DSM-V with changes to the categorisation of autism spectrum disorders (ASD) indicate no separate diagnosis for autistic disorder, Asperger syndrome, childhood disintegrative disorder, or the catch-all diagnosis of pervasive developmental disorder not otherwise specified. Instead, a more dimensional model of autism is suggested with varying degrees of difficulty in social interaction, communication and rigid behaviours or interests (American Psychiatric Association, 2013). With current estimates of the prevalence of ASD in the general population at 9.7 per 1000 (Brugha, et al., 2011), increasing recognition is being given to the difficulties faced by those with ASD.

There is also growing evidence for the association between ASD and anxiety disorder. Tantam (2003) has argued that a growing self-awareness and increasingly complex and ambiguous social situations enhance the risk of people with ASD developing mood and anxiety disorders. In particular, problems with psychosocial functioning emerge when environmental demands exceed personal capacity and the individual is aware of this inadequacy. Similarly, obsessions, generalised worries and generalised anxiety disorder have been found to be more common in higher as opposed to lower functioning ASD in adolescents (Kerns & Kendall, 2012). However, Kanner (1943) suggested symptoms of ASD are anxiety driven, with anxiety as a core feature of behaviour governed by an obsessive desire for sameness. Structural and functional neurological anomalies (Amaral, Bauman, & Schumann, 2003) and genetic vulnerabilities (Piven & Palmer, 1999) have been implicated in the relationship between ASD and anxiety. Theory of mind and executive function deficits have also been widely established in adults with ASD

(Swettenham, 1996; Kana, Keller, Cherkassky, Minshew, & Just, 2009), although the relationship between psychosocial functioning, theory of mind and executive function remains unclear.

In a systematic review of anxiety disorders within the general population, Somers, Goldner, Waraich and Hsu (2006) found lifetime prevalence rates of 16.6%. Anxiety disorders were approximately twice as prevalent among women, with overall stability across the lifespan. The need for investigations among special populations was highlighted, as well as the dearth of information about risk and protective factors in anxiety.

In the child literature, a number of investigations have found significantly higher rates of anxiety in ASD, compared with age-matched peers (Wood & Gadow, 2010; Kuusikko et al, 2008). Ozsivadjian, Knott and Magiati (2012) argue that the triggers and presentation of anxiety in ASD is distinguishable from that in typically developing youths. It has also been suggested that ASD presentations in childhood may differ clinically from adult ASD. For example, King, Ollendick and Mattis (1994) found specific phobias and panic decreasing from childhood into adolescence in ASD and White, Oswald, Ollendick and Scahill (2009) suggesting specific types of anxiety disorder exist at discrete stages of development.

Kerns and Kendall's (2012) reviewed the literature on anxiety in ASD. The included articles were predominantly from child data, with only one study investigating anxiety in adults over the age of 16 years (Hofvander et al, 2009). Similar reviews have been

conducted with a focus on childhood ASD and anxiety (Skokauskas & Gallagher, 2010; MacNeil, Lopes, & Minnes, 2009).

Despite the fact that ASD is a life-long disorder, there is a paucity of research investigating anxiety in adults with ASD (Howlin, 2000). Perhaps this is a result of the complexity of dual diagnosis and the difficulty in disentangling ASD and psychiatric comorbidity. Moreover, adults with such difficulties may require complex treatment and a lack of appropriate services may inhibit systematic identification of prevalence and need within the population. Howlin (2000) conducted a review of studies investigating outcome in adult life for individuals with ASD. She found no prevalence studies of psychiatric morbidity and estimates suggested anywhere between 7% and 80%.

In evaluating the literature on ASD and psychiatric comorbidity, Tantam (2003) suggested comorbid learning disability may play a role in the relationship between anxiety and ASD. Specifically, he identified the challenge of distinguishing ASD from learning disability. A further difficulty is described by Antonacci and Attiah (2008) who suggest that with increasing severity of learning disability, diagnosing psychiatric disorder becomes more difficult. Informant reports are relied upon, but for difficulties such as depression and anxiety, many symptoms are internal to the individual.

Symptom overlap between anxiety and other psychiatric disorders have been well documented within the general population (Davidson, 2002). Comorbidity with depression, obsessive compulsive disorder and psychosis has been related to

severity of mental illness. Other psychiatric disorders may also play a similar role, with well documented associations between ASD and bipolar disorder (DeLong & Nohria, 1994).

Stewart, Barnard, Pearson, Hasan, and O'Brien (2006) conducted a review of depression in ASD and found that several factors may play a role in its identification. In particular, they suggest that individuals with ASD may have difficulty expressing and communicating emotion because of insufficient language skills and problems with theory of mind. They found that repetitive and obsessional behaviours decrease when individuals with ASD become depressed. However, this may be perceived as an improvement in symptoms by carers and clinicians, implying informant ratings of depression in ASD may be inappropriate. Thus, methodological issues in assessing anxiety may play a significant role in findings of prevalence.

Similarly, Williams et al (2006) indicate that diagnostic criteria impacted on their findings about the prevalence of ASD. Some studies use DSM-IV (for example, autistic disorder, Asperger syndrome), while others use 'autism spectrum disorder' as a diagnostic label for a heterogeneous population. They also noted that studies in Japan had significantly higher estimates of ASD than those in North American studies, suggesting differences in culture and geography, particularly as the Japanese studies recruited from more urban areas.

Thus, several factors have been identified that may influence reported rates of anxiety in the ASD population. Given the nature of the current understanding of anxiety disorder in adults with ASD, the present review aims to synthesise the

available evidence to ascertain prevalence rates of anxiety in adults with ASD. Since there are currently no prevalence studies that have been conducted with population based samples, the review will focus on the prevalence rate of anxiety in individuals with ASD presenting in clinical settings.

The primary research question for this review is: what is the prevalence of anxiety disorder in adults with ASD in clinical settings? As highlighted above, a number of factors may play a role in the prevalence of anxiety in ASD. Thus the following factors will be considered:

- Age
- Gender
- Intellectual function
- Type of ASD
- Comorbidity (for example, psychiatric disorder)

## **2. Methods**

### *2.1. Search Strategy*

All searches were completed on 27<sup>th</sup> May 2013 using the following terms:

1. (Anxiety OR anxious OR anxiety disorder)
2. (Autism OR autism spectrum disorder OR autism spectrum condition OR autistic OR Asperger)
3. 1. AND 2.

Searches were limited to those published in English and involving human adults. No limits were placed on publication date as no reviews were found related to the current aims.

The electronic search included the following databases: Cochrane Library; CINHAL; MEDLINE; PsychINFO; Psychology and Behavioural Sciences Collection. To increase the sensitivity of the search, reference lists of relevant articles were also checked to identify further papers. Key journals in this area were manually searched (*Autism and Research in Autism Spectrum Disorders*).

## 2.2. *Inclusion and Exclusion Criteria*

Relevant articles that indicated an investigation of anxiety in ASD were screened against the inclusion and exclusion criteria below.

### 2.2.1. *Inclusion Criteria*

- Human participants aged 16 and over
- Published in English
- Published in a peer reviewed journal
- Assessment of anxiety symptoms
- Sample consists of diagnosable ASD

### 2.2.2. *Exclusion Criteria*

- Participants aged 15 and below
- Non-clinical samples/ general population without diagnosis of ASD
- Qualitative data

- Single case studies
- Unpublished studies
- Book chapters, dissertations, conference extracts
- Studies without a measure of anxiety symptoms
- Intervention trials.

### *2.3. Assessment of Methodological Quality & Data Extraction*

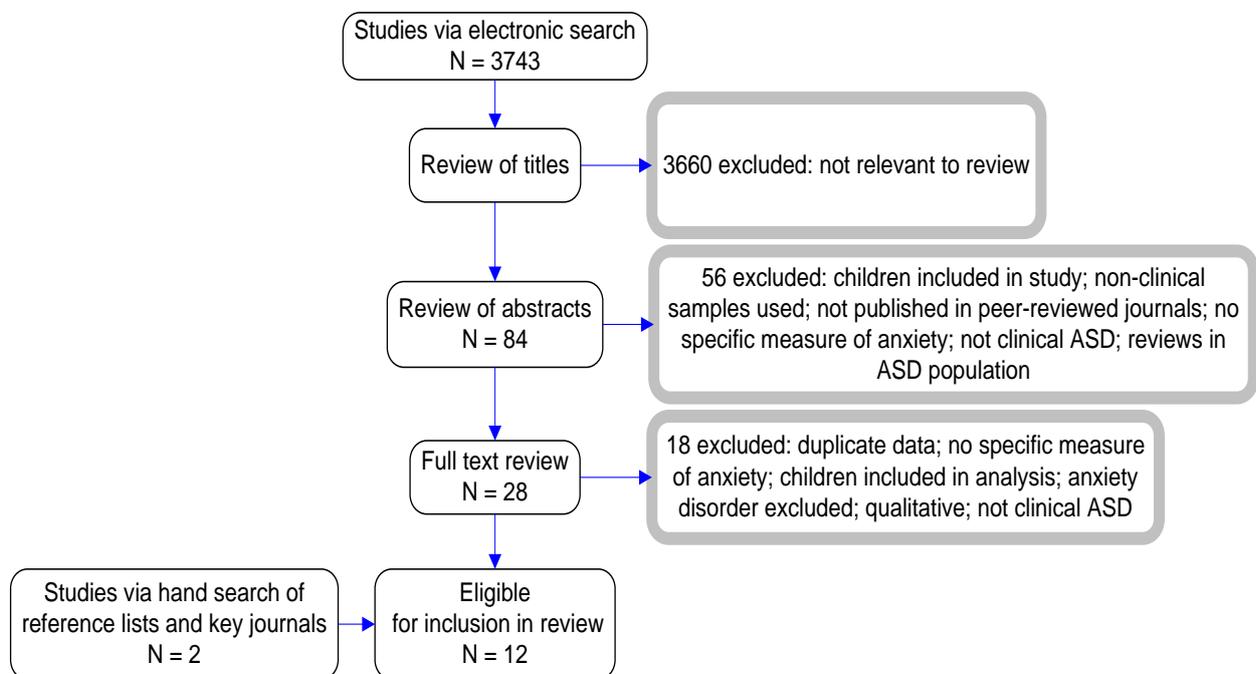
A methodological quality rating scale was developed by the author (see Appendix 1.2) based on a combination of STROBE (Vandenbrouke, et al., 2007) and SIGN 50 guidelines (Scottish Intercollegiate Guidelines Network, 2011), as well as Giannakopoulos, Rammelsberg, Eberhard and Schmitter's (2012) guidelines for evaluating prevalence studies. The rating points were adapted to focus on anxiety disorder prevalence in ASD and to the review aims. Specific items for anxiety disorder were adapted from Somers et al's (2006) review within the general population. The quality rating checklist comprised 22 items with a maximum total score of 27. Scores were converted into percentages to provide an overall quality rating, with higher percentages indicating superior quality. Scores were arbitrarily categorised as High ( $\geq 75\%$ ), Moderate (50-74%), Low (25-49%) or Very Low ( $\leq 24\%$ ). The quality rating was conducted by the author and an independent reviewer. Agreement on each of the individual item scores between the two raters was 92%. Disagreement was resolved and 100% agreement reached by discussion.

## **3. Results**

### *3.1. Search Selection*

Articles generated in the electronic search were pooled (n = 3743) and those with no relevance for the review topic following review of title (n = 3660) were removed. Abstract review of the remaining articles (n = 84) indicated removal of 56 more papers based on exclusion criteria. Full texts were obtained and reviewed for 28 articles. Two studies used duplicate data so only one was included. Further eligibility examination indicated seven papers investigated children under 16 years; one used qualitative methodology, one excluded participants with anxiety disorder; two did not include clinical levels of ASD and six did not assess severity of anxiety.

Reference lists of the ten remaining articles revealed another two studies that met eligibility criteria for the review. Hand searches of *Autism* and *Research in Autism Spectrum Disorders* did not reveal further articles. Figure 1 below depicts the strategy and results of the search.



**Figure 1. Flow chart of search strategy and results**

The 12 articles were then evaluated for the methodological quality as described above. Data were extracted to allow comparisons across studies.

Table 1 summarises key information from all 12 articles. This includes sample size; mean age; country of study; recruitment sites; retrospective or prospective design; primary assessments for anxiety and ASD; main finding of prevalence; and quality evaluation, based on the rating scale developed.

**Table 1. Summary of included studies**

First author	Country	ASD sample size	Age Range (Mean, SD) <sup>j</sup>	Recruitment Site	Design	Primary assessment of ASD	Primary assessment of anxiety	Prevalence of anxiety	Quality
Ghaziuddin	USA	28	18-57 (26.5, 11.3)	Asperger syndrome clinic	P <sup>l</sup>	School records, clinical interview	Clinical interview	21.4 %	67% moderate
Lugnegard	Sweden	54	(27, 3.9)	ASD outpatient clinics	P	DISCO-11 <sup>n</sup>	SCID-1	56%	59% moderate
Ryden	Sweden	84	(30, 10)	Developmental disabilities clinic	P	ASDI	Screening version of SCID	35.7%	59% moderate
Tani	Japan	99	18-63 (30.7, 8.41)	Outpatient service for ASD	P	AQ	Clinical interview	28%	56% moderate
Hofvander	France & Sweden	122	16-60 (29, 11)	Developmental disabilities clinic	P	87% (N = 106) received ASDI <sup>d</sup>	SCID-1 <sup>k</sup>	72%	52% moderate
Tsakanikos	UK	137	(28.4)	Mental health services	R	Clinical interview	Clinical interview	4.4%	48% low
Charlot	USA	13	(39, 13.8)	Developmental disability & psychiatric service	R <sup>m</sup>	Clinical judgement	Semi-structured interview	62%	44% low
La Malfa	Italy	90	(37.4)	Residential & day care centres	P	PDD-MRS <sup>e</sup>	DASH-II <sup>f</sup>	No conclusions	44% low
Cath	Netherland	12	(34.5, 10.5)	Psychiatric outpatient clinics	P	AQ <sup>a</sup>	BAI <sup>b</sup>	100%	41% low
LoVullo	USA	42	(48.5, 11.6)	Developmental disabilities service	P	Clinical interview	ASD-CA <sup>g</sup>	3.1%	41% low
Gilliot	UK	34	18-56 (37, 7.4)	Local clinicians & day centres for ASD	P	Diagnosis assumed	SCASD	73.1%	37% low
Prichard	USA	40	16-56 (28.4, 10.4)	Dual diagnosis service (inpatient & outpatients)	R	ADOS <sup>l</sup>	Clinical interview	40%	27% low

SD<sup>j</sup> = Standard Deviation; DISCO-11<sup>n</sup> = Diagnostic Interview for Social and Communication Disorders; SCID-1<sup>k</sup> = Structured Clinical Interview for DSM-IV; AQ<sup>a</sup> = Autism Questionnaire; BAI<sup>b</sup> = Beck Anxiety Inventory; ASDI<sup>d</sup> = Asperger Syndrome Diagnostic Interview; PDD-MRS<sup>e</sup> = Scale of Pervasive Developmental Disorders in Mental Retarded Persons; DASH-II<sup>f</sup> = Diagnostic Assessment for the Severely Handicapped-Revised; ASD-CA<sup>g</sup> = Autism Spectrum Disorders-Comorbidity for Adults; SCASD = Spence Children's Anxiety Scale-Parent; P<sup>l</sup> = Prospective; R<sup>m</sup> = Retrospective

### 3.2. *Prevalence of Anxiety in ASD*

Table 2 summarises the quality ratings for each of the included articles. All of the studies included in the review were of moderate or low quality and are discussed below.

Ghaziuddin and Zafar (2008) report anxiety disorder in 6 out of 28 participants, a prevalence of 21.4%. They found that the most common psychiatric comorbidity was depression ( $n = 14$ ; 50%). In terms of quality, they stated how the ASD and anxiety disorder diagnoses were given according to DSM-IV criteria, as well as obtaining multiple sources of information (school records, neuropsychological assessment, medical records). However, participants were recruited from a specialist diagnostic clinic for those seeking an ASD assessment. This would suggest that those in the population who had already been diagnosed, perhaps in childhood, were not included, thus impacting on the representativeness of the sample. No control group was used, so prevalence of anxiety cannot be compared with the general population. Also, apart from the percentage of individuals with specific diagnoses, no inferential statistics were reported. As indicated in Table 2 below, Ghaziuddin and Zaffar (2008) had a quality rating of 63%.

Similarly, Lugnegard, Hallerback, & Gillberg (2011) recruited from ASD clinics covering a specific geographical area (pop. 280 000), and stated that these clinics were the most suitable for covering the defined area. They received a quality rating of 59% as they adequately described efforts to address sources of bias in the sample recruited and this is the only study to provide analysis of

differences between the sample recruited and potential participants who did not take part. Autism diagnosis was confirmed by methods that would have been used in the ASD population; inclusion and exclusion criteria were clearly described and appropriate; standardised criteria and assessment methods were used for anxiety; and demographic and other confounding factors were reported and included in analyses. However, this study did not receive a higher rating because of limitations in sampling; low response rate; lack of a control group; and lack of inferential statistics. Thirty out of the 54 participants reported anxiety disorder (56%), with generalised anxiety disorder (n = 12; 22%) and social anxiety disorder (n = 12; 22%) most common.

Ryden and Bejerot (2008) conducted a cross-sectional controlled study to investigate ASD within a psychiatric population. In the ASD group, 12 people had obsessive compulsive disorder (23%); 9 had social phobia (17%); 5 had panic disorder (9.4%); three had generalised anxiety disorder (5.7%) and one had post traumatic stress disorder (1.9%), with an overall prevalence for anxiety disorder of 35.7%. Comparison of prevalence with a control group suggested no significant differences in rate anxiety disorder. Although this study did measure and report anxiety adequately, it was not a representative sample of the ASD population and no efforts were reported to address sources of bias. The control group consisted of individuals who were experiencing social or communication difficulties and sought an ASD assessment. Thus, for the purposes of this review, unable to shed light on the prevalence of anxiety in ASD compared with the general population. They received 59% for quality, with itemised ratings in Table 2 below.

Tani et al (2012) used clinical interview, questionnaires, and school and medical records to assess ASD and anxiety in adults referred for ASD assessment, receiving 56% for methodological quality . In total 668 individuals were assessed for ASD and 14.8% were diagnosed with Asperger Syndrome, all of whom were included for further investigation. They found anxiety to be significantly higher in the ASD group (n = 28; 28%) compared with a control group (n = 6; 10%;  $\chi^2 = 8.17$ ,  $p = 0.004$ ). Higher levels of confusion in new environments, insomnia and depressive mood were also found in the ASD group. In terms of quality of the study, it was limited by a lack of standardised pre-specified criteria for anxiety, inclusion and exclusion criteria were inappropriately defined and a low reported response rate.

Hofvander et al (2009) recruited one of the largest samples of ASD participants (n = 122), obtained from developmental disability diagnostic clinics. Prevalence estimate for anxiety disorder in the sample was 72%. They found obsessive compulsive disorder was most common (n = 29), followed by generalised anxiety disorder (n = 18) and social phobia (n = 16). ASD diagnosis was confirmed using measures found in clinical practice and anxiety was assessed using objective measures against standardised criteria. Demographic variables (such as gender, educational level, employment and marital status) as well as possible confounders (such as psychiatric comorbidity, type of ASD, personality disorder) were considered in the analysis. Nevertheless, this study failed to apply standardised data collection methods across all participants, not all participants who took part in the study

were included in the final analysis and no control group was used. It received 52% for quality.

According to the methodological criteria set in this review, Tsakanikos et al (2007) rated 48% (low quality). This was a retrospective review of medical notes between 1983 and 2000 in mental health services within a specific geographical area. The sample size is the largest of all included studies (n = 137), but the prevalence of anxiety is one of the lowest (4.4%). Although Tsakanikos et al (2007) did report that standardised criteria was used to define and assess anxiety, only clinical interview was used to diagnose both anxiety and ASD. No inclusion or exclusion criteria were reported for the study, or why not all medical records between the time points chosen were not examined.

Charlot et al (2008) and La Malfa et al (2007) also rated low for methodological quality (44%). Both failed to gain samples that were representative of the ASD population due to the recruitment sites used; inclusion and exclusion criteria were not clearly defined or not appropriate; ASD was not diagnosed adequately and response rate was low. Charlot et al (2008) found a prevalence rate of anxiety in ASD of 62% and La Malfa et al (2007) did not draw conclusions on the prevalence of anxiety in their study. Both studies investigated individuals who had learning disability and no individuals with an IQ above 70 were included.

Again, LoVullo and Matson (2009) investigated individuals with ASD and learning disability, with those of normal IQ excluded as a result of the

recruitment source. Cath et al (2007) did not exclude on the basis of IQ, but as their study was designed to investigate the similarities between ASD and social anxiety and obsessive compulsive disorder, those in the ASD group that did not have an anxiety disorder were excluded from analysis. Thus, both studies received 41% for methodological quality. Cath et al (2007) report the highest anxiety at 100% since they only included adults with ASD who had an anxiety disorder, while LoVullo and Matson (2009) report the lowest of all the studies at 3.1%.

Gilliot and Standen (2007) rated 37% for quality. Compared with a learning disability control group, they found significantly higher panic, agoraphobia, separation anxiety and obsessive compulsive disorder in ASD. Effect size indicated 73.1% of the ASD group had scores outside the range of the control group. They used a modified version of the informant-based SCAS. Prichard et al (2010) rated the lowest at 27%. Both studies did not confirm the ASD diagnosis and assumed this had been assessed prior to the study. No inclusion or exclusion criteria were reported.

**Table 2. Quality Ratings for included studies**

	Ghazziuddin	Lugnegard	Ryden	Tani	Hofvander	LoVullo	La Malfa	Tsakanikos	Charlot	Cath	Prichard	Gilliot
<b>Ethical approval</b>	1	1	1	1	1	1	0	0	0	1	0	1
<b>Diagnosis confirmed</b>	1	1	1	1	1	1	1	1	0	1	0	0
<b>ASD assessment</b>	1	1	1	1	1	0	0	0	0	1	0	0
<b>Probability sampling</b>	0	0	0	0	0	0	0	0	0	0	0	0
<b>Representative sample</b>	1	0	0	1	1	0	0	0	0	0	0	1
<b>Inclusion/exclusion criteria</b>	1	1	0	0	0	0	0	0	0	0	0	0
<b>Response rate</b>	0	0	0	0	0	0	0	0	0	0	0	0
<b>Time points referenced</b>	1	1	0	1	0	0	0	1	0	0	1	0
<b>Bias addressed</b>	0	1	0	0	0	0	0	0	0	0	0	1
<b>Control group</b>	0	0	1	1	0	1	0	0	1	1	0	1
<b>Anxiety defined</b>	1	1	1	0	1	1	1	1	1	1	0	1
<b>Data collection</b>	1	0	1	1	0	1	1	1	1	1	0	1
<b>Instrument used</b>	1	1	1	1	1	1	1	1	1	0	0	0
<b>Anxiety threshold</b>	1	1	1	0	1	0	1	1	0	0	0	0

	Ghazziuddin	Lugnegard	Ryden	Tani	Hofvander	LoVullo	La Malfa	Tsakanikos	Charlot	Cath	Prichard	Gilliot
<b>Reliability/ validity</b>	0	0	0	0	0	2	0	0	1	0	0	0
<b>Number of participants</b>	1	1	1	0	0	0	1	0	0	0	1	0
<b>All included in analysis</b>	1	1	1	1	1	1	1	0	1	1	1	1
<b>Demographic factors</b>	2	2	2	2	2	1	2	2	2	1	2	1
<b>Confounding factors</b>	2	2	2	2	2	1	2	2	2	2	2	0
<b>Confidence intervals</b>	0	0	0	0	0	0	0	0	0	0	0	0
<b>Planned analysis</b>	1	1	1	1	1	1	1	1	1	1	1	1
<b>Appropriate design</b>	0	0	1	1	1	1	1	1	1	0	0	1
<b>Total score (maximum = 27)</b>	<b>17</b>	<b>16</b>	<b>16</b>	<b>15</b>	<b>14</b>	<b>13</b>	<b>13</b>	<b>12</b>	<b>12</b>	<b>10</b>	<b>10</b>	<b>9</b>
<b>Score (%)</b>	<b>63%</b>	<b>59%</b>	<b>59%</b>	<b>56%</b>	<b>52%</b>	<b>48%</b>	<b>48%</b>	<b>44%</b>	<b>44%</b>	<b>37%</b>	<b>37%</b>	<b>33%</b>
<b>Quality rating*</b>	<b>M</b>	<b>M</b>	<b>M</b>	<b>M</b>	<b>M</b>	<b>L</b>						

\*Key to quality rating: M = Moderate; P = Poor

Table 2 above allows evaluation across the included articles and indicates that no study considered response rates, used probability sampling, or reported confidence intervals. Inclusion and exclusion criteria were only cited by two articles (Ghaziuddin & Zafar, 2008; Lugnegard et al, 2011), as were attempts to address bias (Gilliot & Standen, 2007; Lugnegard et al, 2011). Only LoVullo and Matson (2009) and Charlot, et al. (2008) reported reliability and validity for their assessment measures.

### *3.3. Confounding factors impacting on anxiety prevalence*

Possible confounding factors in the relationship between anxiety and ASD will be discussed below, according to the review aims. Although confounding factors were reported to some degree in all the studies, very few considered them specifically in those with anxiety. Only information available in the context of the relationship with anxiety will be considered here.

#### *3.3.1. Age*

Although all the included studies reported mean or median age ranges, no consideration was given to the impact of age on reported anxiety. None of the included studies investigated the suggestion that anxiety disorders decrease in frequency with increasing age (King et al, 1994). No trend was observed across the articles, although the lowest prevalence of anxiety reported in LoVullo and Matson (2009) also reported the highest mean age at 48.5 years.

#### *3.3.2. Gender*

Lugnegard et al (2011) found that of the 26 males, 14 had an anxiety disorder (54%) and of the 28 females, 16 had an anxiety disorder (57%). Thus no significant difference was found in gender. Similarly, Hofvander et al (2009) found that 53 out of 82 males had an anxiety disorder (64.6%) and 35 out of 40 females had an anxiety disorder (87.5%).

### 3.3.3. *Intellectual function*

No specific study investigated degree of learning disability and its impact on anxiety prevalence. However, the lowest rates of anxiety were found in LoVullo and Matson (2009) who only included those with a learning disability. Two studies reported moderate/severe learning disability in the ASD group and also used matched controls (LoVullo & Matson, 2009; Gilliot & Standen, 2007).

### 3.3.4. *Type of ASD*

Hofvander et al (2009) found the highest rate of anxiety disorder in the Asperger Syndrome group, with 48 out of 67 individuals diagnosed (71.6%). Forty of the 50 diagnosed with 'pervasive developmental disorder not otherwise specified' also had comorbid anxiety (80%). None of the five individuals diagnosed with Autistic Disorder had a diagnosable anxiety disorder.

### 3.3.5. *Comorbidity and other variables*

In Lugnegard et al's (2011) study, out of the 30 individuals who had an anxiety disorder, 11 fulfilled criteria for two or more anxiety disorder diagnoses. Gilliot

and Standen (2007) found positive correlations between anxiety and 'change', 'anticipation', 'unpleasant events', and 'sensory/personal contact' in their ASD group.

Lugnegard et al (2011) reported no significant differences were found in those who were in employment for anxiety. Prichard et al (2010) found that of those with ASD, 30% of the outpatient group and 10% of the inpatient group were diagnosed with anxiety disorder.

#### *3.4. Synthesis of effect sizes*

Six articles included control groups which were used to compare the rate of anxiety (Cath et al, 2007; Charlot, et al, 2008; Gilliot & Standen, 2007; LoVullo & Matson, 2009; Ryden & Bejerot, 2008; Tani, et al, 2012). However, Charlot et al (2008), Gilliot and Standen (2007) and LoVullo and Matson (2009) used individuals with varying degrees of learning disability in the control group, rather than a general population control. Tani et al's (2012) convenience sample control group were all referred for ASD assessment, and although they did not receive a diagnosis, all had social and/or communication difficulties. A similar sampling method was used for Ryden and Bejerot (2008). Although Cath et al (2007) did recruit a control group from the general population, they specifically recruited ASD individuals who had an anxiety disorder and therefore cannot provide data on prevalence within a general ASD population. Given these disparate methods, synthesis of effect sizes across studies could not be conducted.

## **4. Discussion**

### *4.1. Primary Research Question*

The studies identified in the current review as moderate quality indicate a wide range in the prevalence of anxiety in adults with ASD presenting in clinical settings, ranging between 21.4 and 72%. These are tentative estimates given the lack of high quality studies and several methodological issues that are addressed in detail below, but include recruitment rates, recruitment source, confounding of participant variables, study design and the measures used to assess ASD and anxiety.

Somers et al's (2006) systematic review found anxiety prevalence rates of 16.6% in the general population. As others have suggested, the prevalence appears higher in those with ASD (Kerns & Kendall, 2012). In child studies, the rates of anxiety in ASD have been suggested as 11 to 84% (White et al, 2009). This is largely in line with the current review. Overall, estimates would suggest higher prevalence of anxiety in ASD, compared with the general population. This finding may reflect qualitative accounts indicating there are more environmental sources (e.g. physical environment; unexpected or sudden change; sensory sensitivity) for anxiety in adults with ASD (Trembath, Germano, Johanson, & Dissanayake, 2012). Nevertheless, this assertion needs to be considered within the context of the current research base exclusively focused on clinical settings. There is no definitive evidence to indicate higher rates of anxiety in ASD in a representative sample. Indeed, the higher rates of anxiety indicated by the current review may be reflective of

those presenting in clinical settings more generally, regardless of whether or not they have ASD.

#### *4.2. Secondary Research Question*

The present review also sought to determine the potential impact confounding factors may be having on the rate of anxiety reported in the ASD literature.

##### *4.2.1. Age*

Unfortunately, no data could be obtained to determine the role of age in anxiety in ASD. Berney (2004) argues that ASD traits diminish with age, but also suggests secondary problems, related to increasing social and functional demands, are exacerbated. Howlin (2000) found that the average age of diagnosis for children with Asperger syndrome was over 11 years (compared to five years for autism). Many were reported to have been diagnosed into adulthood. Failure to provide necessary support from an early age may lead individuals to entrenched difficulties that are treatment-resistant in adulthood.

Also, Kuusikko et al (2008) found that symptoms of social anxiety, such as fear of negative evaluation and social avoidance, increased in severity from childhood into adolescence in ASD, which is an inverse of the trend observed in the general population.

##### *4.2.2. Gender*

In contrast with findings from general population studies (Somers et al, 2006), no significant differences were found between female and male anxiety rates

in the studies that considered gender. Synthesising findings from Lugnegard et al (2011) and Hofvander et al (2009) indicates 67 out of 108 males were diagnosed with anxiety (62%) and 51 out of 68 females were diagnosed (75%). This would indicate a higher prevalence of anxiety in females with ASD. All the included articles had a higher number of male participants, a phenomenon that is reflective of the ASD population. Speculating on this preliminary finding suggests there may be specific risk factors associated with anxiety in ASD, which are compounded upon the existing risks found in the general population, whether they be biological, psychological or social.

#### 4.2.3. *Intellectual function*

A surprising finding of this review was that no study has quantitatively investigated the role of intellectual functioning in the relationship between anxiety and ASD. Tantam (2003) suggests that adults with normal IQ on the autistic spectrum have insight into their social difficulties. Problems with psychosocial functioning emerge when environmental demands exceed personal capacity and the individual is aware of this inadequacy. Conversely, those with learning disability experience less anxiety. Similarly, obsessions, generalised worries and generalised anxiety disorder have been found to be more common in higher as opposed to lower functioning ASD in adolescents (Kerns & Kendall, 2012). The role of insight into one's own difficulties was not examined in any of the studies reviewed here.

However, Gilliot and Standen (2007) found anxiety was consistently higher in an ASD group, compared with matched controls. Both groups had mean IQs

indicative of moderate learning disability. Similarly, LoVullo and Matson (2009) found significantly higher levels of reported anxiety in an ASD group who also had moderate to severe learning disability, compared with matched controls. Therefore, intellectual function may not impact rates of anxiety, as suggested previously (Antonacci & Attiah, 2008; Tantam, 2003). Future investigations in ASD will need to address this issue in order to gain an understanding of the impact of intellectual function and insight.

Stewart et al's (2006) review of depression in ASD also highlighted problems with identifying affective disorders. The majority of the assessment methods in the included articles rely on an individual's ability to attribute mental states to self and others (theory of mind), and to have sufficient language skills to express these mental states. This may explain some of the difference found in prevalence of anxiety in studies adopting generic clinical interviews. The interview protocols may not have been adapted for the ASD population. Thus, ASD specific assessments are required.

#### 4.2.4. *Type of ASD*

Again, there is a lack of consideration in the literature given to the nature of ASD and the wide spectrum of symptomatology covered by the diagnosis. Although studies did report numbers of participants in each category of ASD, only Hofvander et al (2009) considered the spread of anxiety across the diagnoses. The Asperger syndrome group had a prevalence of 71.6%; the pervasive developmental disorder had 80% and 0% prevalence in the autistic disorder group. The lack of anxiety in the autistic group may have been due to

the relatively smaller sample size. The Asperger syndrome and pervasive developmental disorder group had higher rates of anxiety, which is reflective of findings from studies in childhood ASD (MacNeil et al, 2009). Individuals with pervasive developmental disorder avoid social situations because of a lack of interest, according to DSM-IV (American Psychiatric Association, 1994). Those with social phobia have an interest but often lack the necessary skills to interact effectively. However, Attwood (1998) argues many with ASD do have an interest and often seek social interaction, but the associated anxiety with these interpersonal encounters interferes with their ability to engage with others.

#### *4.2.5. Comorbidity and Other variables*

Comorbidity remains a controversial issue in ASD. Kerns and Kendall's (2012) review of anxiety in youths with ASD raised many implications that can be generalised to adults. They question the reliability and validity of comorbid diagnoses and argue diagnostic categories are often artefacts of sampling bias, symptom overlap and diversity in overt symptomatology. Similarly, Wood and Gadow (2010) suggest true comorbidity requires a comorbid disorder to be identical in aetiology and phenotype to its monomorbid form. They propose unique ASD versions of anxiety. For example they argue that accurate dual diagnosis of social anxiety disorder in ASD is impossible, given the social communication deficits that define ASD.

In the current review, obsessive compulsive disorder has been included in the prevalence rates reported. However, the distinction between ASD and

obsessive compulsive disorder remains indefinite. Only LoVullo and Matson (2009) utilised a specific measure to differentially diagnose psychiatric comorbidity in ASD.

Themes related to anxiety severity identified by Ozsivadjian et al (2012), may be applicable across the lifespan. A higher rate of specific fears related to sensory stimuli have been reported, particularly loud noises, closed spaces and the dark, further supporting Wood and Gadow's (2010) assertion of specific ASD presentations of anxiety.

Piven and Palmer (1999) suggest further research to identify biological markers of anxiety in ASD are required. These may yield pertinent information about comorbidity and shed light on the continued debate.

#### *4.3. Methodological Quality of Research in ASD*

One of the most concerning weaknesses in the current evidence base is the lack of consensus on the assessment of anxiety in ASD, arising from the phenomenological issues described above. In this review, methods ranged from self report questionnaires, semi-structured interviews, medical note reviews, to informant interviews and semi-structured diagnostic interviews. As suggested earlier, comorbidity and diagnostic overlap are common in psychiatric disorders (Davidson, 2002), and these difficulties may be further compounded by diverse methodology. Similarly, those who used DSM-IV criteria to define anxiety or used the SCID for assessment rated better overall for methodological quality. Perhaps this indicates DSM provides clear and

structured means to assess anxiety objectively, as opposed to a clinical interview with general indicators. The studies that did not use clinical interview tended to find higher rates of anxiety (Lugnegard et al, 2011; Ryden & Bejerot, 2008; Hofvander, et al, 2009; Cath et al, 2007; Gilliot & Standen, 2007). However, the lack of ASD-specific assessments also raises the question of whether symptoms of ASD are misconstrued as anxiety (leading to inflated prevalence) or if true anxiety symptoms are dismissed as ASD (diagnostic overshadowing).

MacNeil et al (2009) suggest a comprehensive assessment of anxiety in ASD should take account of biological and psychological symptoms. Similar to the current review, they found the majority of studies in their review used solely rating scales of anxiety, with no studies reporting norms in the measures used for the ASD population. MacNeil et al (2009) suggest a range of methods including clinical interview, rating scales, direct observation and physiological measures need to be utilised, with multiple informants.

Also, recruitment source played an important role in the quality of the studies. Clinic-based samples are often required to gain adequate sample sizes for statistical power. However, these participants may not provide generalisable findings, particularly in relation to the severity of difficulties, functioning, previous treatment exposure and insight. No studies in this review utilised a population-based cohort study. No probability sampling was used to identify potential participants. Those who recruited from psychiatric populations received lower quality ratings overall, perhaps because of the lack of

specialised assessment methods for ASD. In terms of study design, the retrospective reviews identified in the current evaluation (Charlot et al, 2008; Prichard et al, 2010; Tsakanikos et al, 2007), all received low ratings for quality.

Similar methodological issues have been raised here to Somers et al's (2006) review. They indicated the considerable heterogeneity across studies may be attributable to criteria used for anxiety, country studied, response rate and sample size. Further research is clearly needed in ascertaining anxiety in ASD. For example, future epidemiological studies could utilise community-based surveys using probability sampling techniques in order to establish a true prevalence of anxiety in ASD. Research in other areas, such as in the learning disability population (Cooper, Smiley, Morrison, Williamson, & Allan, 2007), has overcome these limitations to a certain extent by considering more robust sampling and assessment techniques.

However, in the ASD population, studies have been unable to use a standard methodological approach to prevalence for several reasons. In particular, case ascertainment is difficult because of the large proportion of those with ASD living independently with little or no support. Those who do have contact with health and social services may not receive a diagnosis due to diagnostic overshadowing (for example, learning disability) or mis-diagnosis (for example, obsessive compulsive disorder). Perhaps as more vigorous techniques are used to establish the prevalence of ASD in the general

population, investigation of the prevalence of anxiety within these ASD populations may follow.

As discussed above, the diagnosis of ASD has changed with the recent publication of DSM-V and the varying diagnostic protocols used to diagnose ASD complicate any interpretations from the current literature. Williams et al (2006) found that the covariate most strongly associated with their findings of prevalence of ASD was the diagnostic criteria used. Mattila et al (2011) found that the DSM-V criteria for ASD were less sensitive for Asperger Syndrome and high functioning autism. Thus, sensitivity to symptoms is reduced at the milder level of autism symptomatology, particularly those who would previously have been diagnosed with Asperger Syndrome. If this is the case, those experiencing relatively high levels of anxiety may be excluded from diagnosis as they are also more likely to be more highly functioning.

The new DSM-V (APA, 2013) framework has also been criticised by the British Psychological Society (2011) as being largely based on social norms, with little regard to confirmed evidence of biological markers. They argue that many of the ASD diagnoses are merely descriptors, and have little regard to context. For example, an individual with ASD may experience anxiety very differently to someone in the general population, and therefore having the same anxiety disorder diagnosis may prove unhelpful, if indeed they meet the same criteria. Although the subjective nature of the DSM-V criteria and its reliance on clinical judgement raises concerns over its reliability and validity, much research has nevertheless relied on it as a classification system.

In response to the criticisms of DSM-V, the National Institute of Mental Health launched the Research Domain Criteria (RDoC) which it stated they will attempt to incorporate genetics, imaging, neuropsychology and other research in order to develop a new classification system (NIMH, 2008). Although this framework will initially be used for research purposes and is likely to take several years to develop and evaluate it may lead to changes in identification, assessment and management in clinical practice.

#### *4.4. Clinical Implications & Future Research*

No study has established a true prevalence rate among a representative sample of adults with ASD. There is also a need to identify the incidence rate of anxiety in ASD. The review suggests that there are specific risk factors for developing anxiety disorder in the ASD population, but again these findings are tentative. Overall, the literature suggests anxiety rates are higher in ASD than in the general population; rates for adults with ASD are largely in line with childhood ASD; gender differences in anxiety are reflective of the general population (higher anxiety in females); higher functioning ASD is associated with an increased rate of anxiety.

As discussed in Chapter Two, the experience of heightened anxiety is known to have an adverse impact on functioning, particularly theory of mind and executive function (Kana et al, 2009; Swettenham, 1996). What remains unclear is the causal relationship between anxiety and functioning. Wood and Gadow (2010) suggest speech fluency and coherence deficits often seen in high-functioning ASD may be primarily triggered, or at least exacerbated by

social anxiety. Other observable symptoms, such as insistence on sameness, hostility and tantrums, could also be triggered by anxiety. The benefits associated with the repetitive behaviours suggest they are used as a coping strategy or distraction from current mood state. Similarly, anxiety associated with changes in routine do not easily fit into any particular diagnostic criteria for anxiety. Atypical ways in which anxiety presents will need to be considered in future.

The current systematic review suggests that the existing studies recruiting from clinical settings in order to determine anxiety in ASD have several methodological and conceptual limitations. These will need to be addressed before a prevalence rate can be ascertained. Among studies of moderate methodological quality, estimates vary between 21.4 and 72% for prevalence of anxiety. The rate appears to be dependent on a number of factors, primarily the recruitment source, confounding participant variables, study design and the measures used to assess anxiety.

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## Chapter 2

### Major Research Project

#### **Asperger's Syndrome and Traumatic Brain Injury: The role of Anxiety in Executive Function and Theory of Mind Deficits**

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Prepared according to instructions to authors for the *Journal of Neuropsychology* (See Appendix 2.1)

## Abstract

**Background:** Theory of mind and executive function deficits have been implicated in a wide range of neurological conditions, including traumatic brain injury (TBI) and autism spectrum disorder (ASD). Uncertainty remains about the degree of overlap of these functions, as well as the mechanisms that may be involved in specific disorders. The role of other processes, such as anxiety, has also not been considered. **Aims:** To investigate the role of anxiety in individuals with ASD and TBI in their executive function and theory of mind abilities. **Methods:** A prospective between groups design was used to compare ASD and TBI groups. Relaxation training was implemented and individuals were compared pre- and post- relaxation on measures of executive function and theory of mind. **Results:** Pre-relaxation scores indicated the ASD group had significantly higher anxiety than the TBI group, but with relaxation improving anxiety in both groups. Significant interactions of executive function, theory of mind and group were found, with the ASD group scoring higher after relaxation. A significant correlation was also found between executive function and theory of mind, but only in the ASD group. **Conclusions:** The present results suggest anxiety may play a significant role in the executive function and theory of mind deficits observed in ASD. TBI may involve different mechanisms which impact on functioning. **Applications:** The findings raise important implications for intervention and the potential to develop executive function and theory of mind skills in ASD.

## **Introduction**

The ability to attribute mental states (for example, knowledge, beliefs, desires) to both oneself and others, and to understand that others can have thoughts, beliefs and knowledge that are different from our own, is a fundamental skill in social interaction (Rasmussen, Talwar, & Wyper, 2009). Theory of Mind (ToM) is understood as a developmentally advancing ability, with acquisition beginning in childhood. Stone, Baron-Cohen and Knight (1998) argue that impairment in ToM ability is more pronounced in tasks requiring abilities that develop later. For example, they suggest the Reading the Mind in the Eyes Test represents the most developmentally advanced tasks and is a good measure of subtle ToM deficit.

The development of ToM appears to be strongly related to the development of Executive Function (EF), a set of processes that monitor, control, and supervise both thought and action, including self-regulation, inhibitory control, planning, attentional flexibility, error correction and working memory (Carlson, Moses, & Breton, 2002; Hughes, 1998). However, the nature of the link between ToM and EF is an area of continued debate. Some suggest ToM is processed by domain-general cognitive functions, namely executive function (Frye, Zelazo, Brooks, & Samuels, 1996) and ToM therefore is merely one aspect of a general ability. Perner and Lang (1990) suggest that planning, inhibitory control and set-shifting are required to represent mental states of others. There is some evidence suggesting an overlap in the brain regions involved in ToM and EF tasks, particularly the prefrontal cortex (PFC; Channon & Crawford, 2000; Sabbagh & Taylor, 2000). In contrast, others

have found intact EF independent of ToM deficit (Fine, Lumsden, & Blair, 2001; Bach, Happe, Fleminger, & David, 2006).

### **Autism Spectrum Disorder**

Theory of Mind (ToM) and Executive Function (EF) deficits have been implicated in autism spectrum disorder (ASD; Klinhans, Akshoomoff, & Delis, 2005). The recent publication of the DSM-V and the changes to categorisation of autism spectrum disorders (ASD), have renewed debate about the nature of ASD and the core features that define the disorder. The new criteria indicate no separate diagnosis for autistic disorder, Asperger Syndrome, childhood disintegrative disorder, or the catch-all diagnosis of pervasive developmental disorder not otherwise specified (American Psychiatric Association, 2013).

Nevertheless, Liss, Saulnier, Fein, & Kinsbourne (2006) suggest many of the difficulties individuals with ASD experience are not recognised by diagnostic criteria, and instead are impairments in sensory modulation and integration. Specifically, individuals with ASD may exhibit over-selective attention, with focus on a single element in a complex array. Over-arousal and exaggerated focus on elementary features of objects may amplify the experience of auditory, visual, tactile and olfactory stimuli. Those with ASD may thus concentrate on small details of the environment for an inordinate amount of time, filtering out peripheral stimuli. In a sample of 222 participants, they found that those with features of over-selective attention were the highest functioning, but also the most socially impaired. The lowest functioning

individuals were under-reactive to sensory stimuli. Over-focusing, therefore, may be associated with social behaviour, and not necessarily with intellectual functioning.

Similarly, Ornitz (1988) suggested this difficulty in over-selective attention may cause difficulties in shifting focus, leading to perseverative behaviour. Ability to shift from one problem/situation and emotional control ability was strongly associated with ASD symptomatology (Christ, Kanne, & Reiersen, 2010). Kinsbourne (1980) also suggested similarities between these features and the 'displacement' behaviour that is observed in animals when in over-aroused states of frustration. The behaviours appear to serve the function of moderating arousal levels. In relation to ASD, over-selective attention may be serving to regulate arousal levels resulting from an inability to cope with the vast amounts of stimuli from the social environment.

Hussman (2001) postulated the 'noisy' firing of cortical neurons may offer a neurobiological explanation of the over-focusing displayed in ASD. Disinhibition due to release of gabanergic inhibition in ASD, results in hyper-excitation of target neurons and in difficulty distinguishing target stimuli from competing 'noise' in the environment. Over-focus and perseverative behaviours would compensate for this difficulty, serving to reduce sensory input to a narrow, filtered and therefore more manageable scope. Casanova, Buxhoeveden, Switala and Roy (2002) lent support to this theory in an investigation of nine individuals with ASD at post-mortem. Microcellular columns in the brain were smaller, more numerous and dispersed in their

formation, than those of four matched controls. They suggested this pattern would lead to over-connected but insufficiently inhibited neuronal networks, leading to impaired selective attention.

Thus, ASD may predispose an individual to difficulties in social situations. Over-arousal may arise in the context of a changeable and unpredictable social situation, with too many stimuli and not enough resources to filter irrelevant information. What remains unclear is the way in which this over-arousal manifests in high functioning individuals and the subsequent psychosocial implications. This will be discussed later.

Both EF and ToM require the ability to integrate information efficiently. Orienting attention, sensory modulation and filtering are prerequisites to EF and ToM abilities. Therefore, the deficits in EF and ToM observed in ASD may be more related to these underlying cognitive abilities which then impact on higher-order cognitive function. Thus the DSM-V symptomatology that defines ASD may in fact not be the core features of the disorder, but manifestations of underlying impairments in over-arousal and difficulty filtering information from the environment.

Despite the neurobiological underpinnings of ToM and EF, several studies have shown improvements in these abilities in ASD through cognitive training (Fisher & Happe, 2005); neurofeedback (Kouijzer, de Moor, Gerrits, Congedo, & van Schie, 2009); or slowing down the presentation of visual stimuli (Tardif, Laine, Rodriguez, & Gepner, 2007).

## **Traumatic Brain Injury**

Deficits in EF and ToM have also been observed after Traumatic Brain Injury (TBI; Henry, Phillips, Crawford, Ietswaart & Summers, 2006; Bach et al, 2000; Martin-Rodriguez & Leon-Carrion, 2010). Milders, Fuchs, and Crawford (2003) found that those with TBI scored significantly lower than controls on measures of ToM (tests of naming facial expressions and recognising mental states from pictures of eyes).

In contrast with the above assertion that ASD involves over-selective attention, and the implication that attentional capacity is intact, those with severe TBI show slowing of information processing, thought to be an effect of diffuse axonal injury (Gale, Johnson, Bigler, & Blatter, 1995). Similarly, it is argued that disinhibition may play a role in social skill deficits after TBI. Impaired ability to suppress literal, irrelevant and multiple meanings in certain circumstances can make it difficult for individuals to choose correct non-literal interpretations (Champagne-Lavau & Joannette, 2009). For example, evidence suggests individuals with ASD are unimpaired on the Stroop task compared with controls (Ozonoff & Jensen, 1999), whereas those with TBI are impaired (Ben-David, Nguyen, & Lieshout, 2011).

Also, Henry et al (2006) compared 16 individuals with TBI and healthy controls on measures of EF (verbal fluency) and ToM (Reading the Mind in the Eyes Test and Faces Test). There was a significant correlation between ToM and verbal fluency in the TBI group, but not in the control group.

However, using the same tests, Shields and McMillan (2010) found significant correlations between EF and ToM in a group with Asperger Syndrome but not in a TBI group. Thus, the relationship between ToM and EF remains unclear.

### **Anxiety**

Tantam (2000) suggests that anxiety is almost universally comorbid with Asperger Syndrome and that high trait anxiety is a common feature of individuals across the autistic spectrum. He suggests that a growing self-awareness and increasingly complex and ambiguous social situations enhance the risk of people with Asperger Syndrome developing mood and anxiety disorders. Recent evidence suggests prevalence rates of anxiety in ASD to be between 21 and 74% (Ghazziudin & Zafar, 2008; Gilliot & Standen, 2007).

In ASD, the inconclusive explanations may be related to the influence of anxiety on information processing, EF and ToM. In a non-clinical sample, Basso, Schefft, Ris and Dember (1996) reported that positive mood and optimism were associated with a global processing bias, integrating information more readily and inversely related to local bias, while individuals with trait anxiety displayed a tendency for a local processing bias. Those with high trait anxiety were not able to efficiently integrate information and tended to focus on specific aspects of the environment. Bellini (2004) reported higher levels of social skill deficits in socially anxious adolescents with ASD. Structural and functional abnormalities in the amygdala, hippocampus and related limbic system structures associated with emotion processing in ASD

are also implicated in disorders involving fear and anxiety (LaBar & LeDoux, 2006).

In TBI, Godfrey and Shum (2000) suggest EF and ToM deficits are in conscious awareness, particularly because situations in which they coped with before the injury are now more challenging. An awareness of the mismatch between the demands placed on the individual and their ability inevitably leads to anxiety in this context. They argue that social anxiety is relatively common in TBI. A recent longitudinal study indicated the incidence of anxiety (increased in the first 12 months post-injury with poor resolution rates (Gould, Ponsford, Johnston, & Schonberger, 2011). However, the role of anxiety in EF and ToM deficits remains unclear in this population.

Abbreviated Progressive Muscle Relaxation Training (APRT) has been established as an efficacious treatment for eliciting relaxation (Carlson & Hoyle, 1993), with Powlow and Jones (2002) finding APRT reduced cognitive (self-report) and autonomic (salivary cortisol) anxiety.

Unfortunately there is currently a paucity of research investigating the impact of anxious arousal in ASD and TBI on performance on neuropsychological tests and ToM measures. This is surprising given the observations of increased anxiety in these populations, as well as the known impact of anxiety on functioning described above.

Nevertheless, Shields and McMillan (2010) investigated the role of EF and ToM in individuals with ASD and TBI, relative to controls. They found that EF and ToM was impaired in both ASD and TBI, compared with controls. On covarying for anxiety, ToM differences between the ASD group and controls became non-significant, thus suggesting differing mechanisms underlying the TBI and ASD deficits. An exploratory analysis also suggested the TBI and ASD groups differed in the relationship between EF and ToM. In the ASD group, EF and ToM were positively correlated but there was no significant correlation in the TBI group. The present study investigated this further by utilising an anxiety intervention and investigating the impact on ToM and EF in both TBI and ASD.

The following hypotheses are explored:

1. EF and ToM scores improve to a greater degree in the ASD group, compared with the TBI group, after adjusting for pre-relaxation anxiety.
2. EF and ToM are positively and significantly correlated in the AS group and not significantly correlated in the TBI group, at pre- and post-relaxation.

## **Methods**

### ***Sample***

Participants were recruited from voluntary organisations covering Glasgow and Ayrshire, Scotland. These included Headway, National Autistic Society, the Autism Resource Centre and the Durrans Community Brain Injury Rehabilitation Centre. Eligible participants were identified by site management

and provided with a participant information sheet (Appendix 2.2), invitation letter (Appendix 2.3) and consent form (Appendix 2.4). If they agreed to participate, they were asked to complete the consent form and return in a prepaid envelope, or contact the researcher via telephone or email. Inclusion and exclusion criteria were applied based on information from site staff and self report. Inclusion criteria included: adults aged 18-64 years old; within the ASD group, participants had a diagnosis of Asperger Syndrome or high functioning autism; within the TBI group, participants had a diagnosis of severe Traumatic Brain Injury identified by self report; first language was English; participants functioning above the learning disability range (IQ > 70) and able to consent to participate in the study. Exclusion criteria included: dual diagnosis of TBI and ASD; those with any current chronic psychiatric condition or presenting with symptoms of trauma; anyone identified with significant levels of risk to self or others; lack of capacity under the Adults with Incapacity Act.

Demographic information, including Scottish Multiple Index of Deprivation (SIMD) derived from participant postcodes

([www.scotland.gov.uk/Topics/Statistics/SIMD/SIMDPostcodeLookup](http://www.scotland.gov.uk/Topics/Statistics/SIMD/SIMDPostcodeLookup)) is

summarised in Table 1.

### ***Estimation of sample size***

Sample size was estimated using G\*Power 3 (Faul, Erdfelder, Land & Buchner, 2007). No studies to date have compared two clinical samples on EF and ToM before and after relaxation training. The State Trait Anxiety Inventory was used by Powlow and Jones (2002) to examine the effects of

relaxation training on an experimental versus control group, both consisting of non-clinical samples. They found significantly lower anxiety in their experimental group post-intervention. Based on post-intervention data comparing the experimental group ( $M = 25.8$ ,  $SD = 5.6$ ) and the control group ( $M = 34.3$ ,  $SD = 7.5$ ), power calculation indicated 22 total participants, 11 in each group, would be required for a power of 0.8 and alpha of 0.05. For EF, Shields and McMillan's (2010) data compared ASD and TBI on the Hayling sentence completion test. The ASD group had 140.6 ( $SD = 50.7$ ) seconds remaining to complete the task on average, and the TBI group had 97.9 ( $SD = 63.1$ ); with power of 0.8 and alpha of 0.05 a total of 30 participants would be required (15 per group). Thus, the conservative estimate of a total sample size of 30 participants was used, with 15 in each ASD and TBI group sufficient to detect any significant difference between the groups.

### ***Design***

A prospective between groups design comprised individuals with ASD and TBI. In both groups, participants completed EF, ToM and anxiety measures before and after receiving relaxation training. These are described below.

### ***Measures***

The order of the measures was randomised across participants using a randomisation plan generator programme (Dallal, 2008).

### ***Executive Function***

Symbol Search from the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph, 1998) was used which is primarily a measure of focused attention and response speed, which are both sensitive to executive function impairments. Participants are asked to rapidly process novel symbol-number pairs and identify correct symbols corresponding to numbers. The more symbol-number pairs identified within 90 seconds, the higher the score. A and B versions were used pre- and post-relaxation training.

Two versions of verbal fluency were utilised, with F, A and S (Benton & Hamsher, 1989) in one version and B, D and T (Borkowski, et al, 1967) in the other. Ruff, Light and Parker (1996) found the correlation between these two versions was high ( $R = 0.74$ ,  $p < 0.001$ ). Participants are asked to list as many words as possible beginning with each of the three letters in turn. One minute is allowed per letter. Scores for the total number of correct responses provides indication of executive function.

Working memory was assessed using reverse digit span, with the pre- and post- versions utilising RBANS A and B modifications (Randolph, 1988). This is primarily a measure of attention and working memory, both of which contribute to executive function abilities (Miller, 1956). Participants were asked to repeat progressively longer strings of digits in reverse order.

The Hayling Sentence Completion Test of Dysexecutive Syndrome (Burgess & Shallice, 1997) was also used. The test was divided for the purposes of the

study, into two parts, A and B. This test measures the ability to inhibit a prepotent response, an important aspect of executive function. Errors were also recorded.

### *Theory of Mind*

The Reading the Mind in the Eyes Test was again divided into A and B, for the purposes of the study (Baron-Cohen, Wheelwright, Hill, Raste & Plumb, 2011). This is a measure of adult theory of mind. Participants are asked to decide what emotion (from a choice of four) is conveyed via pictorial presentations of human eyes.

The Faces Test (Baron-Cohen, Wheelwright & Jolliffe, 1997) was also utilised, again divided into pre- and post- relaxation. Participants are asked to decide what emotion is conveyed in pictures of human faces, each with differing configurations of facial features.

### *Anxiety*

The State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983) was used as a measure of anxiety. It has been shown to have high test-retest reliability ( $r = 0.88$ ; Barnes, Harp, & Jung, 2002). The Trait anxiety measure was administered at the beginning of the session and the State anxiety measure was administered pre- and post- relaxation. Granacher (2003) suggests this is a useful tool for evaluating anxiety throughout rehabilitation and treatment for TBI.

### ***Intervention***

Abbreviated Progressive Relaxation Training (APRT) was utilised, which has an established clinical efficacy (Powlow & Jones, 2002). This technique takes approximately twenty minutes to complete and is a shortened version of Jacobsen's (1939) original Progressive Muscle Relaxation approach, involving tensing and relaxing 16 different muscle groups. A standard script was employed following Bernstein and Borkovec's (1973) standard procedure (See Appendix 2.5).

### ***Procedure***

Research procedures were approved by the University of Glasgow College of Medical and Veterinary Sciences Ethics Committee (See Appendix 2.6). All participants gave informed consent prior to inclusion and what this entails was discussed at initial contact. They were invited to attend a single two-hour appointment within a local clinical setting. Participants were provided with information about the study, given an opportunity to ask questions and reminded of informed consent and right to withdraw. All participants were asked about age, gender, education history and postcode.

The Trait anxiety measure was completed followed by the first versions of the above measures, as indicated by the randomisation plan. Participants were offered a five minute break prior to starting relaxation training. The above relaxation procedure was then administered. The second versions of the measures were then completed, again randomised.

### ***Statistical Analysis***

All analyses were undertaken using the Statistics Package for Social Sciences (SPSS) Version 19. To investigate the predicted differences between the ASD and TBI groups inferential analyses were conducted to explore the variance between the groups on the dependent variable measures. Before conducting parametric inferential statistics, checks for normality, homogeneity of variance, linearity and multicollinearity were undertaken. If violated, non parametric tests were used. Bonferroni corrections were administered for multiple comparisons, with the adjusted p value at 0.0065 in Tables 2 and 3 below. Where appropriate, means and standard deviations were used to calculate the value of Cohen's d to indicate effect size, which were reported and described according to Cohen's (1988) recommendations of small ( $r = 0.1$ ), medium ( $r = 0.3$ ) and large ( $r = 0.5$ ).

## **Results**

### ***Sample characteristics***

A total of 28 individuals consented to take part in the study and met inclusion criteria. Demographic information from the 28 participants is summarised in Table 1. There were no significant differences in gender ( $\chi^2 (1) = 1.26, p = 0.262$ ) and SIMD quintile ( $\chi^2 (3) = 3.39, p = 0.335$ ) between the groups. The TBI group was older ( $t (26) = -4.93, p < 0.001$ ), and had fewer years in education ( $t (26) = 2.12, p = 0.044$ ).

Table 1. Demographic information for the ASD and TBI groups

	<b>ASD</b>	<b>TBI</b>
<b>N</b>	13	15
<b>Mean age in years (SD)</b>	26.38 (8.16)	45.93 (12.09)
<b>Gender</b>	9 male, 4 female	13 male, 2 female
<b>Mean years education (SD)</b>	15.23 (3.56)	12.80 (2.48)
<b>Median SIMD (IQR)</b>	1.00 (1.50)	1.00 (1.00)

All TBI participants reported having suffered a severe head injury resulting in post-traumatic amnesia (PTA) of more than one day (Russell & Nathan, 1946). In the TBI group, two participants reported greater trait anxiety than the rest of the group, and were treated as outliers. A sensitivity analysis was conducted and this indicated these outliers did not impact on the significance of the results below. They were therefore included in the analysis to maximise power.

### ***Dependent variable measures***

Shapiro-Wilk's tests ( $p > 0.05$ ) and a visual inspection of histograms, normal Q-Q plots and box plots showed that the scores were approximately distributed for both groups, apart from pre- and post- Hayling completion scores for both groups. The post- administration of the Faces test was also not normally distributed, but only for the ASD group. Data transformation procedures were unable to normalise distributions. Thus, non-parametric equivalents for this data are used below.

Scores on ToM, EF and anxiety measures were compared between the two groups pre- and post- relaxation training. Descriptive and inferential values for group effects are given in Table 2. Higher scores on EF and ToM tests indicate better performance. The Hayling score represented completion time (i.e. lower score = better performance); completion times were subtracted from 200 seconds to give a score in line with the other tests (higher score = better performance). Errors on the Hayling were also converted so that higher scores indicated better performance by subtracting the number of errors from the maximum number of errors possible.

Table 2. Within group differences on EF and ToM tests pre and post relaxation (mean and SD); paired samples t-tests or Wilcoxon signed rank test values

	ASD			TBI		
	Mean (SD)/ Median (IQR)			Mean (SD)/ Median (IQR)		
	Pre	Post	p value	Pre	Post	p value
<b>Eyes</b>	14.20 (3.23)	15.10 (2.51)	0.134	9.47 (3.98)	9.20 (3.17)	0.617
<b>Faces</b>	8.00 (8)	9.00 (6)	0.089 <sup>a</sup>	8.00 (5)	8.00 (6)	0.685 <sup>a</sup>
<b>Verbal Fluency</b>	34.50 (17.56)	40.20 (19.53)	0.008	26.33 (13.68)	25.07 (12.47)	0.115
<b>Digit Span</b>	6.50 (1.65)	8.30 (3.13)	0.024	6.47 (2.69)	6.33 (2.80)	0.744
<b>Symbol Search</b>	40.90 (8.65)	44.70 (11.19)	0.048	22.33 (11.59)	23.07 (12.13)	0.577
<b>Hayling time</b>	164.00 (184.00)	161.00 (174.00)	0.812 <sup>a</sup>	151.00 (131.00)	151.00 (121.00)	0.233 <sup>a</sup>
<b>Hayling errors</b>	4.70 (1.77)	5.30 (1.70)	0.313	4.00 (1.89)	3.87 (1.96)	0.769
<b>State Anxiety</b>	35.60 (11.61)	29.80 (8.32)	0.005*	41.40 (14.58)	32.00 (8.32)	0.007

<sup>a</sup> p value for non parametric equivalents is provided where data did not meet assumptions of parametric statistics

\* significant at adjusted p value of 0.00625

State anxiety was significantly reduced in both groups after relaxation training (Table 2). A repeated measures analysis of variance (ANOVA) found a significant effect of state anxiety in the ASD and TBI groups ( $F(1) = 15.29$ ,  $p = 0.001$ ) and the effect size was small ( $\eta_p^2 = 0.37$ ). The interaction of anxiety and group was not significant ( $F(1) = 0.25$ ,  $p = 0.624$ ; Figure 1 below). The ASD group also ( $M = 49.23$ ,  $SD = 17.39$ ) had higher pre-relaxation trait anxiety than the TBI group ( $M = 36.54$ ,  $SD = 8.02$ ;  $t(16.88) = 2.39$ ,  $p = 0.029$ ).

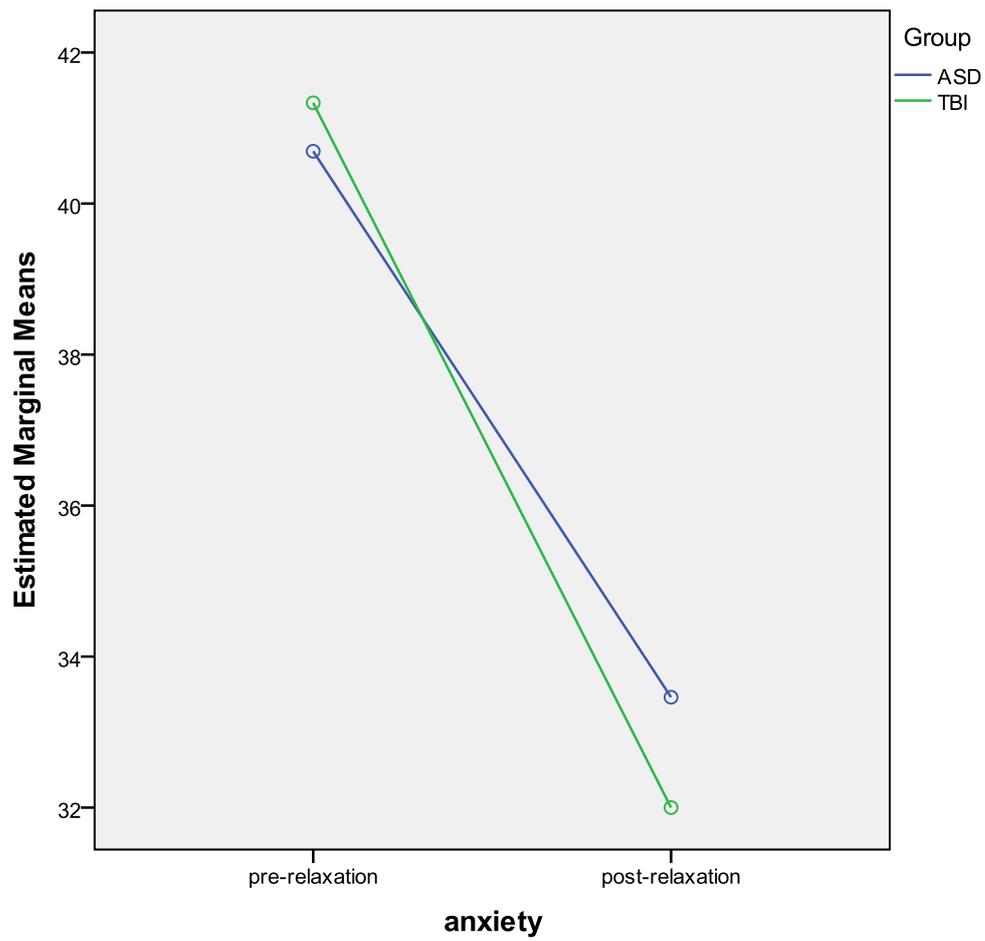


Figure 1. Interaction of group and state anxiety at pre- and post- relaxation

Table 3. Between group differences on EF and ToM tests pre or post relaxation (mean and SD); independent samples t-tests and Mann Whitney U tests

	Pre scores			Post scores		
	Mean (SD)/ Median (IQR)			Mean (SD)/ Median (IQR)		
	ASD	TBI	p value	ASD	TBI	p value
<b>Eyes</b>	14.20 (3.23)	9.47 (3.98)	0.005*	15.10 (2.51)	9.20 (3.17)	<0.00*
<b>Faces</b>	8.40 (1.27)	7.53 (1.73)	0.187	9.00 (6)	8.00 (6)	0.021 <sup>a</sup>
<b>Verbal Fluency</b>	34.50 (17.56)	26.33 (13.68)	0.205	40.20 (19.53)	25.07 (12.47)	0.26
<b>Digit Span</b>	6.50 (1.65)	6.47 (2.70)	0.972	0.89 (0.17)	0.76 (0.19)	0.100
<b>Symbol Search</b>	40.90 (8.65)	22.33 (11.59)	<0.00*	43.60 (11.29)	23.07*(12.13)	<0.00*
<b>Hayling</b>	164.00 (184.00)	151.00 (131.00)	0.063 <sup>a</sup>	161.00 (174.00)	151.00 (121.00)	0.052 <sup>a</sup>
<b>Hayling errors</b>	2.30 (1.77)	3.00 (1.89)	0.362	1.70 (1.70)	3.13 (1.96)	0.72
<b>State Anxiety</b>	35.60 (11.61)	41.40 (14.58)	0.303	29.80 (8.32)	32.00 (8.32)	0.524

<sup>a</sup> p value for non parametric equivalents is provided where data did not meet assumptions of parametric statistics

\* significant at adjusted p value of 0.00625

Data from EF measures were transformed to z scores from which a composite score was derived. Tables 4 and 5 show the correlation matrices for the pre- and post- EF measures. On pre-relaxation symbol search and digit span, the correlation was nearing significance at  $p = 0.068$ . Hayling scores were correlated using Spearman's correlation. As can be seen from the Tables,

Hayling error scores did not correlate significantly with any of the other EF measures, and they were therefore not included in the composite scores derived for EF.

Table 4. Correlation coefficients for pre-relaxation scores on EF measures

	<b>Verbal fluency</b>	<b>Digit span</b>	<b>Symbol search</b>	<b>Hayling time</b>	<b>Hayling error</b>
<b>Verbal fluency</b>	1	0.65**	0.65**	0.72**	-0.19
<b>Digit span</b>	-	1	0.37	0.49*	-0.23
<b>Symbol search</b>	-	-	1	0.59**	0.07
<b>Hayling time</b>	-	-	-	1	-0.050

\*\*denotes significance at  $p < 0.01$

\* denotes significance at  $p < 0.05$

Table 5. Correlation coefficients for post-relaxation scores on EF measures

	<b>Verbal fluency</b>	<b>Log Digit span</b>	<b>Log Symbol search</b>	<b>Hayling time</b>	<b>Hayling errors</b>
<b>Verbal fluency</b>	1	0.69**	0.77**	0.71**	0.19
<b>Log Digit span</b>	-	1	0.61**	0.57*	-0.00
<b>Log Symbol search</b>	-	-	1	0.66**	0.44*
<b>Hayling time</b>	-	-	-	1	-0.86

\*\*denotes significance at  $p < 0.01$

\* denotes significance at  $p < 0.05$

A similar process was deployed for the ToM composite score. Spearman's correlation matrices for the pre-relaxation Reading the Mind in the Eyes test and the Faces test indicated the two tests were significantly correlated ( $r = 0.59$ ,  $n = 28$ ,  $p = 0.002$ ).

Shapiro-Wilk tests, visual examination of normal Q-Q plots and box plots indicated the EF and ToM composite measures were approximately normally distributed across both ASD and TBI groups. Table 5 below provides means and standard deviations for pre- and post- composite scores in the two groups.

Table 5. Independent sample and paired sample t-tests for composite scores

	EF		P value	ToM		P value
	Mean (SD)			Mean (SD)		
	Pre	Post		Pre	Post	
<b>ASD</b>	0.14 (0.85)	0.29 (0.86)	0.029	0.16 (1.09)	0.36 (0.98)	0.087
<b>TBI</b>	-0.12 (0.75)	-0.25 (0.67)	0.011	-0.14 (0.74)	-0.31 (0.74)	0.083
<b>P value</b>	0.410	0.072		0.392	0.051	

\*No significant differences observed at adjusted p value of 0.00625

### ***Hypothesis 1***

*EF and ToM scores improve to a greater degree in the ASD group, compared with the TBI group, after adjusting for pre-relaxation anxiety.*

A repeated measures analysis of covariance (ANCOVA) on the composite scores for EF and ToM was conducted, with age, years in education and trait anxiety as covariates. No significant two-way interaction between group and age ( $p = 0.128$ ) or group and years in education ( $p = 0.773$ ) was found and these variables were excluded from further analyses to retain maximum power.

A repeated measures ANCOVA on the composite scores for EF, covarying for trait anxiety was conducted. Preliminary analysis evaluating the homogeneity of regression assumption indicated that the relationship between trait anxiety and post-relaxation EF did not differ significantly as a function of group ( $F(1) = 1.18, p = 0.288$ ). The main effect of EF was not significant ( $F(1) = 0.86, p = 0.362$ ). The interaction between EF and group was significant ( $F(1) = 12.32, p = 0.002$ ) and the effect size was small ( $\eta_p^2 = 0.33$ ; see Figure 2). Adjusted means are presented in Appendix 2.7 ( $p = 0.800$ ).

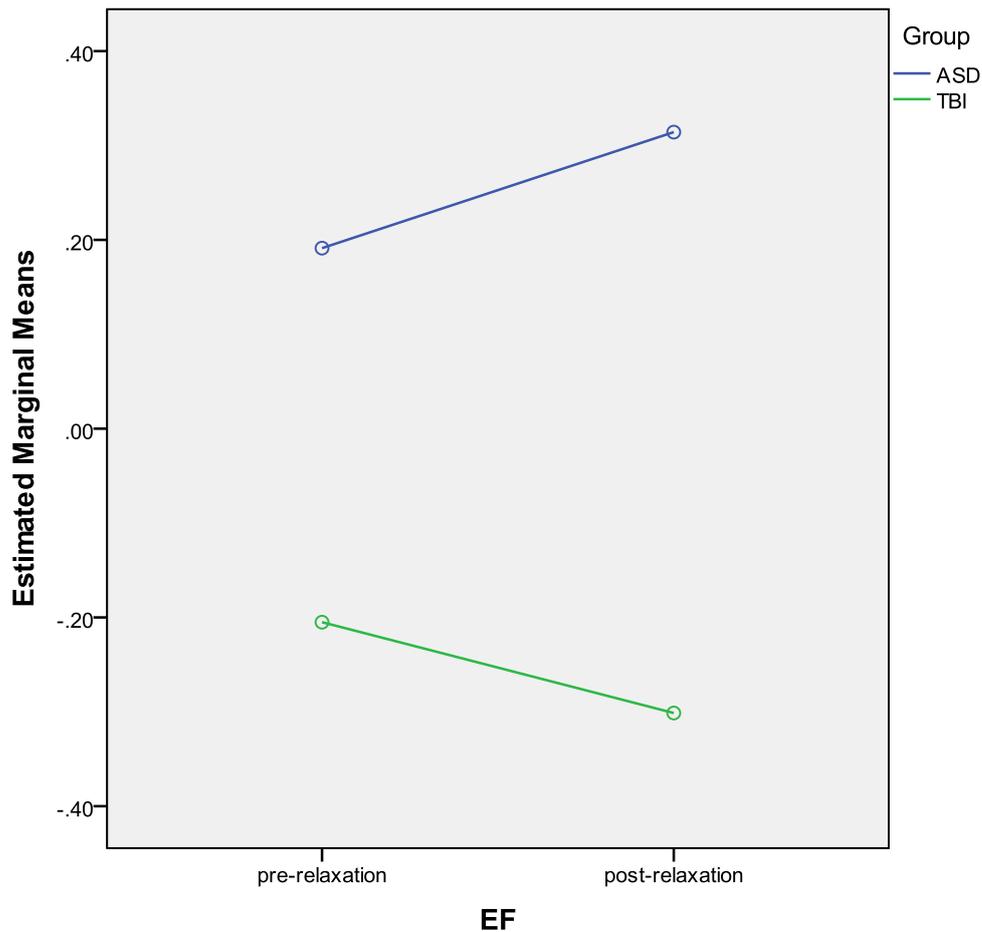


Figure 2. Interaction of group and EF composite scores pre- and post-relaxation

ANCOVA on the composite scores for ToM, covarying for trait anxiety, revealed no significant main effect of ToM ( $F(1) = 0.68, p = 0.418$ ). A significant interaction was found between ToM and group ( $F(1) = 7.51, p = 0.011$ ), and the effect size was small ( $\eta_p^2 = 0.23$ ; see Figure 3). No significant interaction between ToM and the covariate trait anxiety ( $F(1) = 0.65, p = 0.427$ ) was found. The adjusted means are reported in Appendix 2.7 ( $p = 0.843$ ).

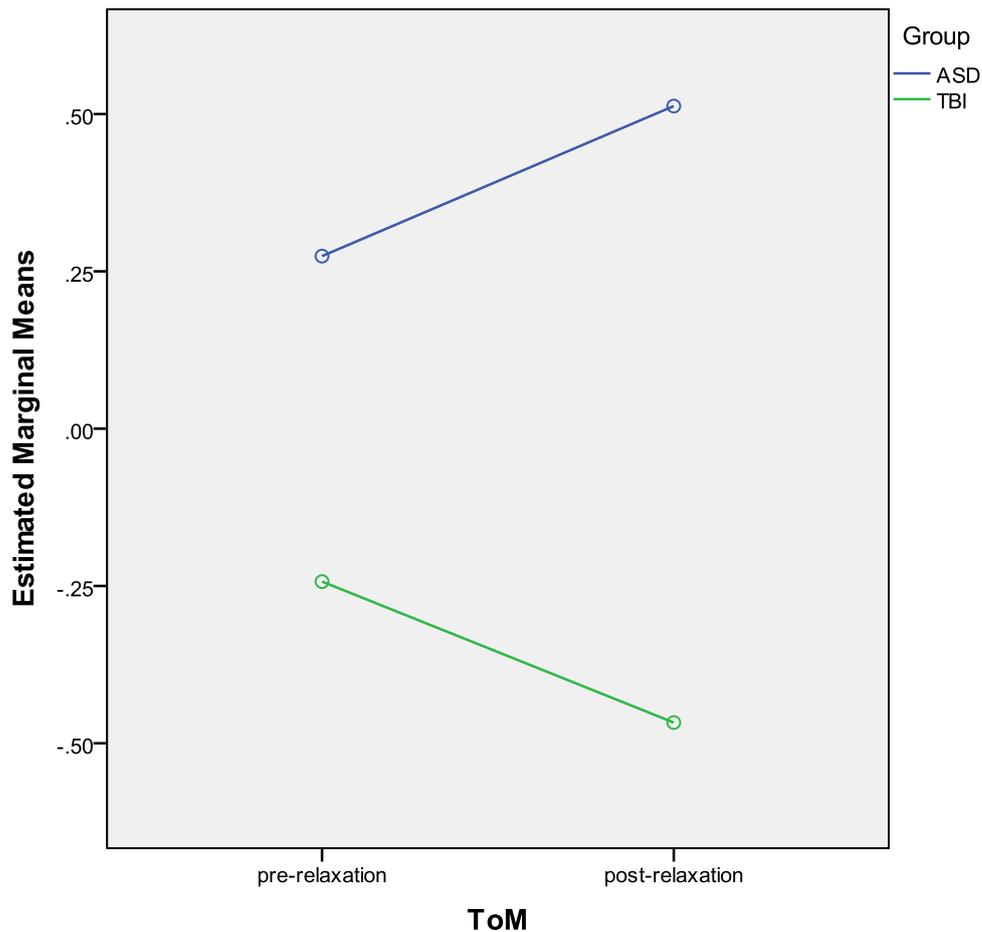


Figure 3. Interaction between group and ToM composite scores pre- and post-relaxation

To examine the relationship between changes in anxiety, EF and ToM between the two time points, change scores were constructed by subtracting post-relaxation values from pre-relaxation values. Pearson's correlations between changes in anxiety and changes in EF (ASD  $r = -0.46$ ,  $p = 0.117$ ; TBI  $r = -0.17$ ;  $p = 0.544$ ) and ToM (ASD  $r = 0.04$ ,  $p = 0.906$ ; TBI  $r = 0.19$ ;  $p = 0.507$ ) were not significant.

**Hypothesis 2**

*EF and ToM are positively and significantly correlated in the AS group and not significantly correlated in the TBI group.*

A Pearson correlation indicated a statistically significant positive relationship between EF and ToM composite scores in the ASD group pre-relaxation ( $r = 0.87, p < 0.001$ ) and post-relaxation ( $r = 0.82, p = 0.001$ ). Figure 3 below illustrates this correlation.

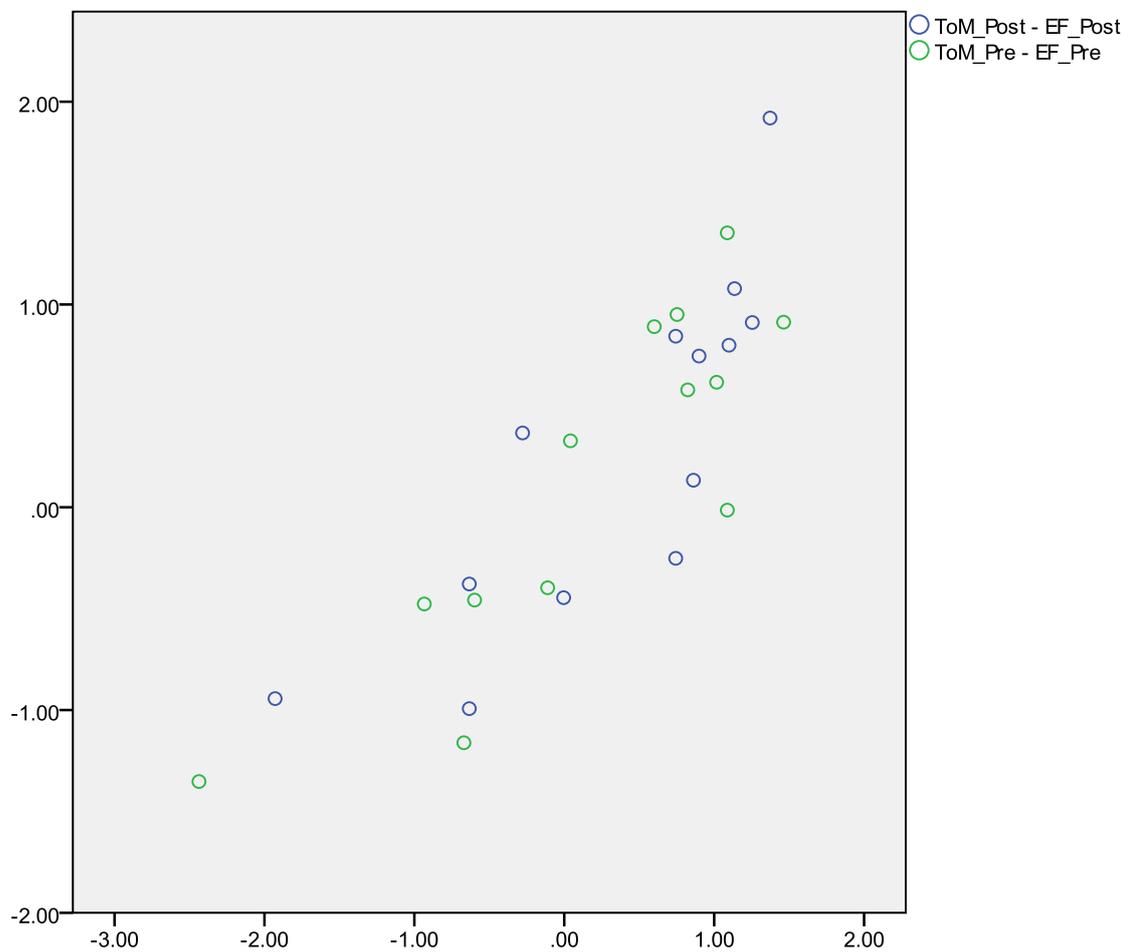


Figure 3. ASD composite score correlations across pre- and post- relaxation

No significant relationships were found between EF and ToM in the TBI group pre-relaxation ( $r = 0.19$ ,  $p = 0.501$ ) or post-relaxation scores ( $r = 0.14$ ,  $p = 0.619$ ).

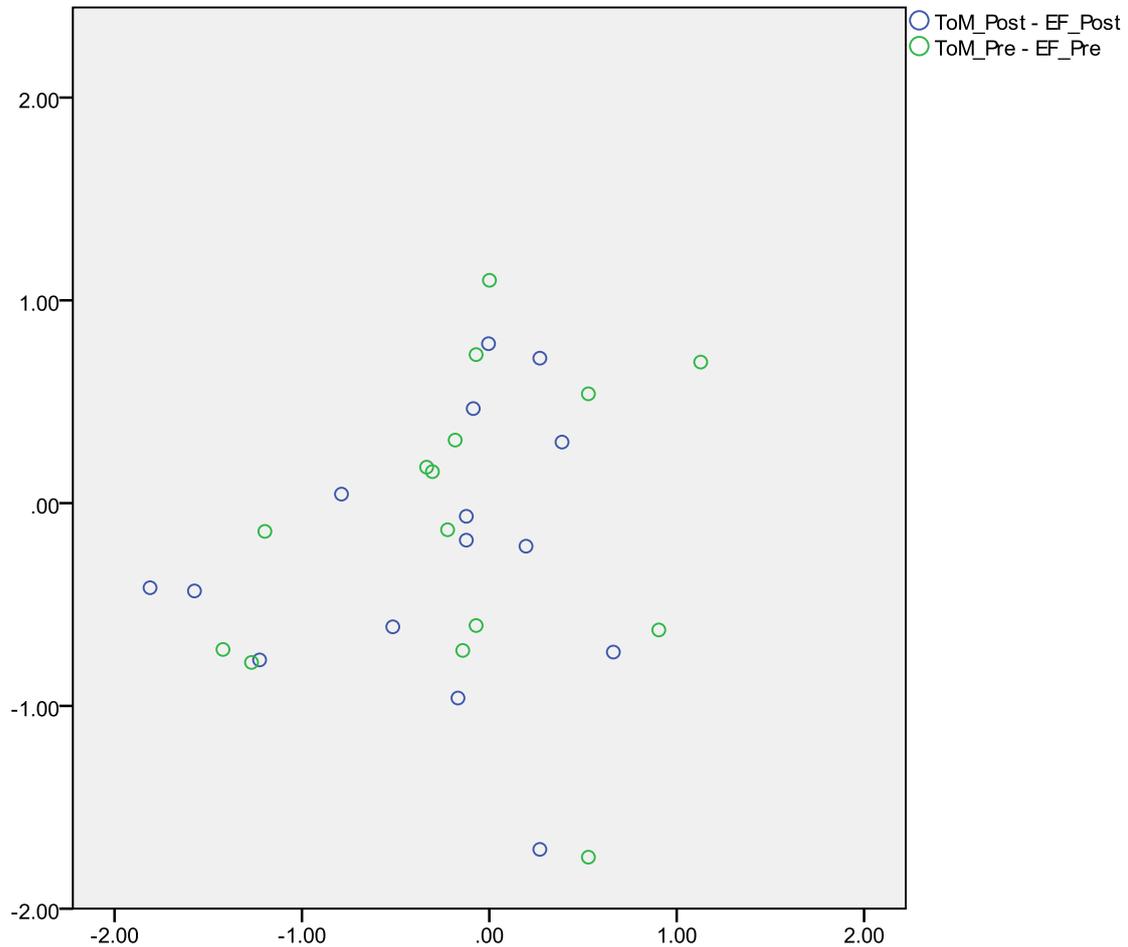


Figure 4. TBI composite score correlations across pre- and post- relaxation

## Discussion

### *Key Findings*

The present study compared EF and ToM in individuals with ASD and TBI, taking into account the effect of reducing anxiety on EF and ToM. The ASD group had significantly higher trait anxiety than the TBI group pre-relaxation, as others have found when comparing ASD with other clinical groups (Limoges, Mottron, Bolduc, Berthiaume, & Godbout, 2005). The relaxation

intervention significantly reduced state anxiety in both groups, with the TBI group experiencing a greater reduction in state anxiety. A significant interaction between group and EF and group and ToM was found, with the ASD group scoring higher after relaxation, thus rejecting the null hypothesis. Similarly, the second hypothesis that there is a significant correlation between EF and ToM in ASD but not TBI is supported, further adding weight to the assertion that ASD and TBI deficits involve distinct processes.

Shackman et al (2006) suggest competition between task-relevant and anxiety-relevant goals for limited attention resources, which is consistent with other findings that anxiety disrupts the efficient allocation of attention (Basso et al, 1996; Kouijzer et al, 2009). In ASD, the over-selective attention hypothesis would explain the current findings. When individuals are anxious, they tend to focus on specific aspects of the environment in order to alleviate arousal. This local processing bias interferes with task-relevant demands, thus leading to observed low performance. This may only be pertinent for tasks requiring integration of information and function, such as EF and ToM. As in-session anxiety is alleviated, processing becomes more global as attentional resources are more effectively allocated. This also reflects findings from non-clinical samples (Basso et al, 1996).

Other studies report mixed results in relation to the relationship between EF and ToM. Henry et al (2006) found that in a sample of TBI, performance on the Reading the Mind in the Eyes task was significantly correlated with the Controlled Oral Word Association Test (which is sensitive to EF impairment),

relative to controls ( $r = 0.74$ ,  $p = 0.04$ ). Conversely, others have found ToM impairment in TBI may be independent of EF function (Bach, et al, 2006; Shields & McMillan, 2010). The findings here support the latter assertion.

Taken together, the findings suggest the impairments in EF and ToM observed in ASD and TBI involve different processes. Anxiety is suggested to play a role in the impairments observed in ASD, with possible mechanisms involving over-selective attention. This may be supported by the finding of a significant difference between the two groups in the Symbol Search task. Slowed processing, assessed via Symbol Search, often underlies attentional deficits and this has been associated with TBI (Spikman, Kiers, Deelman, & van Zomeren, 2000). The findings suggest attention is not as impaired in ASD, possibly indicating that they performed relatively better in a task that requires visual scanning and focused attention, with little need to integrate a range of sources of information. Nevertheless both group scores were below normative data pre- and post-relaxation (Lezak, Howieson, Loring, Hannay, & Fischer, 2004).

### ***Strengths and Limitations***

This study increased construct validity by using composite scores for EF and ToM. Results of intercorrelation matrices found medium to large significant correlations between EF and ToM measures at both time points, suggesting the tests used measured similar constructs.

A limitation of the study was in test-retest reliability. The period between pre-relaxation testing and post-relaxation testing was too short to avoid practice effects that may mimic improvement in EF and ToM. Although, the TBI group did not appear to benefit from practice effects as EF and ToM did not improve across the time points, there may be several reasons for this. In particular, the TBI group may have experienced fatigue post-relaxation. The level of cognitive impairment may have been severe enough to inhibit practice effects and any learning between the two time points. Therefore, the ASD and TBI group profiles across the two time points may be reflective of a lack of practice effect and fatigue in the TBI group, with the ASD group benefiting from repeated testing. Further investigations may be able to account for this by increasing the time period between testing. Future research may also test the hypotheses in a more robust manner by using a different model with experimental and control arms within the ASD and TBI groups. This would eliminate the need for a pre- and post- assessment, thus addressing the problem of practice effects and fatigue.

The sample in this study was recruited from voluntary organisations to which people with TBI and ASD were registered, and this was deemed the most effective way of reaching a representative sample of the two populations. However, the ASD sample was primarily recruited from an initiative that involved supporting individuals back to employment. This is reflected in the difference in age and years in education between the two groups. The ASD group were significantly younger, so they may have had greater opportunities for further education following their diagnosis (Fein, et al., 2013), and these

two factors may be related. As the TBI group were older, and previous studies suggest anxiety rates decrease with increasing age (Jorm, 2000), this is an important limitation of the study. Some of the above EF measures have been shown to be sensitive to years in education, such as verbal fluency (Lezak, et al, 2004). Another consequence was that the ASD group had lower levels of anxiety compared with previous studies because they may have developed compensatory strategies for managing anxiety in certain situations (Shields & McMillan, 2010). Future studies may be able to take advantage of more rigorous approaches to the measurement of anxiety as well as control for these factors.

### ***Clinical Implications***

The findings suggest that for those with ASD, anxiety may play a significant role in the deficits traditionally thought to characterise the disorder. By reducing anxiety, even temporarily, significant improvement in EF and ToM can be observed. Clinically, this has implications for perceived level of functioning, the role of anxiety interventions, and the validity of neuropsychological assessment in this population.

Although this study found similar levels of EF and ToM functioning in both groups, different levels of support may be required. Shields and McMillan (2010) found similar deficits, with TBI participants scoring lower on measures of cognitive flexibility, attention and inhibition than the ASD group. This would suggest individuals with ASD may be able to employ more adaptive strategies to compensate for difficulties (Chanon & Crawford, 2000).

### ***Future Research Considerations***

Further work is required to first replicate these findings and then to extend them to other neuropsychological functions, as well as clinical populations. Both EF and ToM are multifaceted dimensions that may benefit from investigation of the mechanisms involved in each. For example, ToM includes perceptual and conceptual elements (Fisher & Happe, 2005). Separating the components may allow for examination of interaction between the two constructs.

Within the ASD literature, previous findings suggest impairments in sensory modulation and integration. Given that higher functioning ASD is associated with greater impairment in social behaviour, the present study may contribute to our understanding of the mechanisms involved in impairments in sensory modulation and social behaviour. Specifically, the role of anxiety has largely been neglected and may mediate the relationship between sensory modulation and social behaviour. Future research will need to investigate this further by determining causality.

### **Conclusions**

This study contributes to the growing understanding of the role anxiety has in cognitive functioning. Specifically in the clinical populations investigated, it is suggested different mechanisms are involved in apparently very similar deficits. The implications are that anxiety interventions may not just improve

psychosocial function in adults with ASD, but also lead to significant improvement in cognitive function.

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## **Chapter 3**

### **Advanced Clinical Practice 1: Reflective Critical Account**

**(abstract only)**

**The constraints of the NHS: Are they necessary for development as a  
trainee?**

**Aisha Tariq**

## **Abstract**

The recent Scottish Government targets for reducing waiting times have had considerable impact on all aspects of mental health in the National Health Service. Over the course of my current placement, these demands have played an increasingly more prominent role. In this Account, gaining experience of various service models and ways of working will be utilised to understand how managerial and political demands impact on ethical practice. Throughout key learning experiences during training, the tension between managerial demands and clinician ideals have been pertinent in shaping practice as well as underpinning knowledge and skill acquisition. In order to develop and maintain professional standards, it has been essential to use the process of reflection to critically evaluate where we, as clinicians, fit in this constant struggle. In highlighting these experiences in the Account, I endeavour to analyse the multiple factors that lead to new insights and how skills and competence have developed over time. Thus, reflections focus on the impact of experiences of service demands and how this may influence clinical practice.

## **Chapter 4**

### **Advanced Clinical Practice 2: Reflective Critical Account**

#### **Group Analysis in the Context of Professional Development: A Reflective Account**

**Aisha Tariq**

## **Abstract**

On my final placement as part of clinical training, I have had the opportunity of receiving supervision from a group analyst. As a result of this focus on psychodynamic principles, learning experiences that have shaped my development have included consideration of group influences. As a trainee clinical psychologist, I have been affiliated with a number of groups that have shaped my practice. Indeed, key learning experiences have often occurred at critical time points in relation to the life of a group. These group memberships will be reflected on and analysed in relation to the complex learning environment and how competencies have developed over time. Learning experiences vary from individual therapeutic contacts which have allowed development of clinical skills, to awareness of how resources and services are managed within the constraints of the NHS. I will also reflect how I intend to use these experiences in future learning and development.

## Appendix 1.1. Guidelines for submission to Research in Autism Spectrum Disorders



### RESEARCH IN AUTISM SPECTRUM DISORDERS GUIDE FOR AUTHORS

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

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

#### Article structure

##### Subdivision - numbered sections

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

#### Introduction

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

#### Material and methods

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

#### Theory/calculation

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

#### Results

Results should be clear and concise.

#### Discussion

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

## Conclusions

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

## Appendices

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

## Essential title page information

- Title. Concise and informative. Titles are often used in information-retrieval systems. Avoid abbreviations and formulae where possible.
- Author names and affiliations. Where the family name may be ambiguous (e.g., a double name), please indicate this clearly. Present the authors' affiliation addresses (where the actual work was done) below the names. Indicate all affiliations with a lower-case superscript letter immediately after the author's name and in front of the appropriate address. Provide the full postal address of each affiliation, including the country name and, if available, the e-mail address of each author.
- Corresponding author. Clearly indicate who will handle correspondence at all stages of refereeing and publication, also post-publication. Ensure that phone numbers (with country and area code) are provided in addition to the e-mail address and the complete postal address. Contact details must be kept up to date by the corresponding author.
- Present/permanent address. If an author has moved since the work described in the article was done, or was visiting at the time, a 'Present address' (or 'Permanent address') may be indicated as a footnote to that author's name. The address at which the author actually did the work must be retained as the main, affiliation address. Superscript Arabic numerals are used for such footnotes.

## Abstract

An abstract should be submitted that does not exceed 200 words in length. The abstract should be brief, concise, and complete in itself without reference to the body of the paper. Include purpose, methodology, results, and conclusions where applicable.

## Highlights

Highlights are mandatory for this journal. They consist of a short collection of bullet points that convey the core findings of the article and should be submitted in a separate file in the online submission system. Please use 'Highlights' in the file name and include 3 to 5 bullet points (maximum 85 characters, including spaces, per bullet point). See <http://www.elsevier.com/highlights> for examples.

## Keywords

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

## Abbreviations

Define abbreviations that are not standard in this field in a footnote to be placed on the first page of the article. Such abbreviations that are unavoidable in the abstract must be defined

at their first mention there, as well as in the footnote. Ensure consistency of abbreviations throughout the article.

#### Acknowledgements

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

#### Math formulae

Present simple formulae in the line of normal text where possible and use the solidus (/) instead of a horizontal line for small fractional terms, e.g., X/Y. In principle, variables are to be presented in italics. Powers of e are often more conveniently denoted by exp. Number consecutively any equations that have to be displayed separately from the text (if referred to explicitly in the text).

#### Electronic artwork

##### General points

- Make sure you use uniform lettering and sizing of your original artwork.
- Embed the used fonts if the application provides that option.
- Aim to use the following fonts in your illustrations: Arial, Courier, Times New Roman, Symbol, or use fonts that look similar.
- Number the illustrations according to their sequence in the text.
- Use a logical naming convention for your artwork files.
- Provide captions to illustrations separately.
- Size the illustrations close to the desired dimensions of the printed version.
- Submit each illustration as a separate file.

A detailed guide on electronic artwork is available on our website:

<http://www.elsevier.com/artworkinstructions>

You are urged to visit this site; some excerpts from the detailed information are given here.

##### Formats

If your electronic artwork is created in a Microsoft Office application (Word, PowerPoint, Excel) then please supply 'as is' in the native document format.

Regardless of the application used other than Microsoft Office, when your electronic artwork is finalized, please 'Save as' or convert the images to one of the following formats (note the resolution requirements for line drawings, halftones, and line/halftone combinations given below):

EPS (or PDF): Vector drawings, embed all used fonts.

TIFF (or JPEG): Color or grayscale photographs (halftones), keep to a minimum of 300 dpi.

TIFF (or JPEG): Bitmapped (pure black & white pixels) line drawings, keep to a minimum of 1000 dpi.

TIFF (or JPEG): Combinations bitmapped line/half-tone (color or grayscale), keep to a minimum of 500 dpi.

Please do not:

- Supply files that are optimized for screen use (e.g., GIF, BMP, PICT, WPG); these typically have a low number of pixels and limited set of colors;
- Supply files that are too low in resolution;
- Submit graphics that are disproportionately large for the content.

#### Figure captions

Ensure that each illustration has a caption. Supply captions separately, not attached to the figure. A caption should comprise a brief title (not on the figure itself) and a description of the illustration. Keep text in the illustrations themselves to a minimum but explain all symbols and abbreviations used.

#### Tables

Number tables consecutively in accordance with their appearance in the text. Place footnotes to tables below the table body and indicate them with superscript lowercase letters. Avoid vertical rules. Be sparing in the use of tables and ensure that the data presented in tables do not duplicate results described elsewhere in the article.

#### References

##### Citation in text

Please ensure that every reference cited in the text is also present in the reference list (and vice versa). Any references cited in the abstract must be given in full. Unpublished results and personal communications are not recommended in the reference list, but may be mentioned in the text. If these references are included in the reference list they should follow the standard reference style of the journal and should include a substitution of the publication date with either 'Unpublished results' or 'Personal communication'. Citation of a reference as 'in press' implies that the item has been accepted for publication.

##### Web references

As a minimum, the full URL should be given and the date when the reference was last accessed. Any further information, if known (DOI, author names, dates, reference to a source publication, etc.), should also be given. Web references can be listed separately (e.g., after the reference list) under a different heading if desired, or can be included in the reference list.

##### Reference management software

This journal has standard templates available in key reference management packages EndNote (<http://www.endnote.com/support/enstyles.asp>) and Reference Manager (<http://refman.com/support/rmstyles.asp>). Using plug-ins to wordprocessing packages, authors only need to select the appropriate journal template when preparing their article and the list of references and citations to these will be formatted according to the journal style which is described below.

##### Reference style

**Text:** Citations in the text should follow the referencing style used by the American Psychological Association. You are referred to the Publication Manual of the American Psychological Association, Sixth Edition.

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

**Examples:**

**Reference to a journal publication:**

Van der Geer, J., Hanraads, J. A. J., & Lupton, R. A. (2010). The art of writing a scientific article. *Journal of Scientific Communications*, 163, 51–59.

**Reference to a book:**

Strunk, W., Jr., & White, E. B. (2000). *The elements of style*. (4th ed.). New York: Longman, (Chapter 4).

**Reference to a chapter in an edited book:**

Mettam, G. R., & Adams, L. B. (2009). How to prepare an electronic version of your article. In B. S. Jones, & R. Z. Smith (Eds.), *Introduction to the electronic age* (pp. 281–304). New York: E-Publishing Inc.

## Appendix 1.2. Quality Rating Scale for Systematic Review

### Assessment of Methodological Quality

**Study identification:**

**Reviewer:**

**Date:**

#### Sampling/Recruitment

Criteria	Score	Comments
Ethical approval reported	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	
Autism diagnosis confirmed	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	
Autism, as defined by the measure used, matches that found in the target population	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	
Probability sampling used to identify potential respondents	Complicated <input type="checkbox"/> 2 Simple <input type="checkbox"/> 1 No <input type="checkbox"/> 0	
The sample was representative of the Autism population	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	
Inclusion & exclusion criteria clearly defined and appropriate	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	
Adequate response rate (equal to or greater than 80%)	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	
Reference made to time points data was collected (e.g. on admission)	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	
Described efforts to address sources of bias	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	
Control group used	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	

#### Measurement

Criteria	Score	Comments
How is anxiety defined?	Standardised criteria <input type="checkbox"/> 1 Subjective report <input type="checkbox"/> 0	
Data collection methods standardised across all participants	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	
Type of instrument used to assess anxiety	Non-standardised (e.g. self-report) <input type="checkbox"/> 0 Standardised subjective (e.g. clinical interview, questionnaire) <input type="checkbox"/> 1 OR objective (clinician diagnostic interview) <input type="checkbox"/> 2 Standardised subjective AND objective	
Pre-specified threshold for anxiety	Yes <input type="checkbox"/> 1 No <input type="checkbox"/> 0	

Reliability and/or validity reported for anxiety measure	No	<input type="checkbox"/>	0
	Reliability only OR Validity only	<input type="checkbox"/>	1
	Reliability AND validity reported	<input type="checkbox"/>	2

### Analysis

Criteria	Score	Comments
Numbers of individuals at each stage of study reported (e.g. potentially eligible, screened, confirmed eligibility)	Yes	<input type="checkbox"/> 1
	No	<input type="checkbox"/> 0
All participants who took part were included in the analysis	Yes	<input type="checkbox"/> 1
	No	<input type="checkbox"/> 0
Consideration of demographic factors (e.g. age, IQ, socio-economic status)	Reported AND included in statistical analysis	<input type="checkbox"/> 2
	Reported	<input type="checkbox"/> 1
	No	<input type="checkbox"/> 0
Consideration of other confounding factors (e.g. comorbidity, medication)	Reported AND included in statistical analysis	<input type="checkbox"/> 2
	Reported	<input type="checkbox"/> 1
	No	<input type="checkbox"/> 0
Satisfactory confidence intervals ( $\geq 90\%$ )	Yes	<input type="checkbox"/> 1
	No	<input type="checkbox"/> 0
All analyses were planned	Yes	<input type="checkbox"/> 1
	No	<input type="checkbox"/> 0
The analysis was appropriate to design and measures	Yes	<input type="checkbox"/> 1
	No	<input type="checkbox"/> 0

<b>Additional comments:</b>	
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**SCORE:**

27

- Quality Rating:**
- High ( $\geq 75\%$ )
  - Moderate (50-74%)
  - Low (25-49%)
  - Very Low ( $\leq 24\%$ )

## Appendix 2.1. Guidelines for submission to the Journal of Neuropsychology



Journal of Neuropsychology

Author Guidelines

The Journal of Neuropsychology publishes theory-driven patient studies. The central brief is to learn more from patients with brain dysfunctions to gain a better understanding of brain-behaviour relationships and to help future patients. Important developments in neuropsychology will follow from a multidisciplinary approach embracing neighbouring fields such as developmental psychology, neurology, psychiatry, physiology, endocrinology, pharmacology and imaging science. The journal publishes group and case studies addressing fundamental issues concerning the cognitive architecture of the brain. In addition, the journal includes theory-driven studies regarding the epidemiology of specific deficits, new assessment tools, and the evaluation of treatment regimes.

### Manuscript requirements

- Contributions must be typed in double spacing with wide margins. All sheets must be numbered.
- Manuscripts should be preceded by a title page which includes a full list of authors and their affiliations, as well as the corresponding author's contact details. A template can be downloaded [here](#).
- Tables should be typed in double spacing, each on a separate page with a self-explanatory title. Tables should be comprehensible without reference to the text. They should be placed at the end of the manuscript with their approximate locations indicated in the text.
- Figures can be included at the end of the document or attached as separate files, carefully labelled in initial capital/lower case lettering with symbols in a form consistent with text use. Unnecessary background patterns, lines and shading should be avoided. Captions should be listed on a separate sheet. The resolution of digital images must be at least 300 dpi.
- All articles should be preceded by an Abstract (see point 3 for guidelines), giving a concise statement of the intention, results or conclusions of the article.
- For reference citations, please use APA style. Particular care should be taken to ensure that references are accurate and complete. Give all journal titles in full and provide DOI numbers where possible for journal articles.

- SI units must be used for all measurements, rounded off to practical values if appropriate, with the imperial equivalent in parentheses.
- In normal circumstances, effect size should be incorporated.
- Authors are requested to avoid the use of sexist language.
- Authors are responsible for acquiring written permission to publish lengthy quotations, illustrations, etc. for which they do not own copyright.

For guidelines on editorial style, please consult the [APA Publication Manual](#) published by the American Psychological Association.

### Supporting Information

JNP is happy to accept articles with supporting information supplied for online only publication. This may include appendices, supplementary figures, sound files, videoclips etc. These will be posted on Wiley Online Library with the article. The print version will have a note indicating that extra material is available online. Please indicate clearly on submission which material is for online only publication. Please note that extra online only material is published as supplied by the author in the same file format and is not copyedited or typeset. Further information about this service can be found at <http://authorservices.wiley.com/bauthor/suppmat.asp>.

### Copyright and licenses

If your paper is accepted, the author identified as the formal corresponding author for the paper will receive an email prompting them to login into Author Services, where via the Wiley Author Licensing Service (WALS) they will be able to complete the license agreement on behalf of all authors on the paper.

### **For authors signing the copyright transfer agreement**

If the OnlineOpen option is not selected the corresponding author will be presented with the copyright transfer agreement (CTA) to sign. The terms and conditions of the CTA can be previewed in the samples associated with the Copyright FAQs below:

CTA Terms and Conditions [http://authorservices.wiley.com/bauthor/faqs\\_copyright.asp](http://authorservices.wiley.com/bauthor/faqs_copyright.asp)

### Colour illustrations

At the editors' discretion, colour figures can be provided for use in the journal. Good quality photographs will be considered for inclusion where they add substantially to the argument, to a maximum of three per article. These can be supplied electronically as TIF files scanned to at least 300dpi. If they are not printed in colour, then they can be reproduced in colour online and black and white in print.

### Pre-submission English-language editing

Authors for whom English is a second language may choose to have their manuscript professionally edited before submission to improve the English. A list of independent suppliers of editing services can be found at [http://authorservices.wiley.com/bauthor/english\\_language.asp](http://authorservices.wiley.com/bauthor/english_language.asp). All services are paid for and arranged by the author, and use of one of these services does not guarantee acceptance or preference for publication.

## Appendix 2.2. Participant Information Sheet



My name is Aisha Tariq and I am a trainee clinical psychologist at the University of Glasgow. I would like to invite you to take part in a study looking at the role anxiety plays in our ability to think clearly and focus on mental tasks. The information gathered is for research purposes only. You will not be identifiable in the results.

Before you decide whether or not to take part, it is important for you to understand why the research is being carried out and what it will involve. Please take your time to read the following information carefully. If there is anything that is unclear or if you would like more information, please let me know. Take your time to decide whether or not you wish to take part.

### **What is the title of this project?**

Asperger's Syndrome and Traumatic Brain Injury: The role of Anxiety in Executive Function and Theory of Mind Deficits

### **Why is this study important?**

Although previous research has shown that people with Asperger's Syndrome and those that have suffered a traumatic brain injury are more likely to experience difficulties in doing mental tasks, there may be many reasons for this. The current study will look at the role anxiety has to play in these difficulties and whether there is a difference between the two groups. The current study aims to reduce levels of anxiety by using relaxation training and to see if this is affected by either Asperger's Syndrome or brain injury.

### **Who can take part in this study?**

Anyone, aged between 18 and 65, with either a diagnosis of Asperger's Syndrome or severe Traumatic Brain Injury can take part.

### **Do I have to take part?**

Participation in this study is entirely voluntary. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to come out of the study at any time without giving a reason.

### **What does participation involve?**

If you decide to take part, you will be sent a consent form to be completed and sent back by pre-paid post. You will be asked to meet with me at a local setting that you are familiar with for a two hour appointment, at a time that is convenient to you. At the beginning of this meeting, you will be given the chance to ask any questions you may have about the study. You will then be asked to complete a number of questionnaires and puzzles, designed to look at the different ways people problem-solve. You will then participate in the relaxation training, which will be with myself and will involve practising some

relaxation exercises whilst sitting. The relaxation will be approximately 20 minutes. Following this, we will complete some more questionnaires and puzzles to look at if the relaxation training had an impact on how you solve the tasks.

### **What are the possible risks of taking part?**

There are no direct risks of taking part although it is possible that the questionnaires may make you think about any mood or anxiety difficulties you are experiencing. If we are worried about your wellbeing, we would ask you if you wanted us to let your GP know and make suggestions about further support that can be offered.

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

Regular use of the relaxation training may help with anxiety in your daily life, although we cannot guarantee that it will do so.

### **What if there is a problem?**

If you wish to complain, or have any concerns about any aspect of the way you have been approached or treated during the course of this study, you can contact my supervisor, Professor Tom McMillan at the University of Glasgow (Tel: 0141 211 0694), who will be able to advise you on the appropriate complaints procedure, should you wish to use this.

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

Yes. We will follow ethical and legal practice and all information about you will be handled in confidence. Your GP will only be contacted after you have given consent to let them know you are taking part.

All of the information collected about you during the research study will be kept strictly confidential. Personal details (such as your name and address) will not be stored. I will be offering presentations of the results of the study to the centres that request it following completion of the study. The results will also be submitted for publication to a peer-reviewed journal, although no identifiable information will be used.

### **Who is organising and funding the research?**

This study is being funded and organised through the University of Glasgow.

### **Who has reviewed the study?**

All research at the University of Glasgow looked at by an independent group of people, called a Research Ethics Committee, to protect your interests. This study has been approved and given a favourable opinion by the Medical, Veterinary and Life Sciences College Ethics Committee.

### **Further information and contact details**

If you would like further details about the study, you can either contact me by phone or email. Alternatively you can write down any questions you have and we can discuss them at our first meeting together.

If you decide that you would like to take part in the study you can either contact me on 07833095371 or a.tariq.1@research.gla.ac.uk and we will arrange an appointment. If you decide you don't want to, there is no need to reply.

You are under no obligation to take part; participation in this study is completely voluntary. You do not have to give a reason for not wanting to take part in this study.

I would like to take this opportunity to thank you for your time and consideration.

## Appendix 2.3. Participant Invitation Letter



Institute of Health and Wellbeing  
Academic Centre, Gartnavel Royal Hospital  
1055 Great Western Road  
Glasgow, G12 0XH

Dear Sir/Madam

### **Title of study: Asperger's Syndrome and Traumatic Brain Injury: The role of Anxiety in Executive Function and Theory of Mind Deficits**

We are writing to ask whether you would be willing to take part in some research which may help us understand the role anxiety plays in our ability to think and understand the world.

The study is to be conducted by Aisha Tariq (Trainee Clinical Psychologist) and will be supervised by Professor Tom McMillan at the University of Glasgow.

We have enclosed an information sheet to give you an idea of what is involved in the study. There is also an opt-in form provided. If you are interested in becoming involved in this study then please complete the opt-in form and return it to me using the enclosed freepost envelope. Alternatively you can contact Aisha Tariq on **07833095371** to let her know you are interested in becoming involved.

If you have any questions, please contact Aisha Tariq on the number above or Tom McMillan on 0141 211 3920.

Yours faithfully,

Aisha Tariq  
Trainee Clinical Psychologist

Tom McMillan  
Professor in Clinical Neuropsychology

## Appendix 2.4. Participant Consent Form



Centre Number:

Participant Identification Number:

### CONSENT FORM

Title of Project: Asperger's Syndrome and Traumatic Brain Injury: The role of Anxiety in Executive Function and Theory of Mind Deficits

Name of Researcher: Aisha Tariq

Please  
initial box

I confirm that I have read and understand the information sheet for the above study. I understand I have the opportunity to consider the information, ask questions and have these answered satisfactorily.

I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical care or legal rights being affected.

I agree to my GP being informed of my participation in the study.

I agree to take part in the above study.

Contact telephone number: \_\_\_\_\_

\_\_\_\_\_  
Name of participant

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Date

\_\_\_\_\_  
Name of person taking consent

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Date

## **Appendix 2.5. Relaxation Script**

Before I begin I would like you to take a few seconds to relax as much as you can totally on your own..... Settle back as comfortably as you can. Let yourself relax to the very best of your ability....Now, as you relax like that, clench both fists, just clench your fists tighter and tighter, and study the tension as you do so. Keep them clenched and feel the tension in your fists, hands, forearms....and now relax. Let the fingers of your hands become loose, and observe the contrast in your feelings...now let yourself go and try to become more relaxed all over...

Once more, clench your fists really tight...hold them, and notice the tension again....Now let go, relax; your fingers straighten out, and you notice the difference once more....Each time, pay close attention to your feelings when you tense up and when you relax. Now straighten your arms, straighten them so that you feel the most tension in the triceps muscles along the back of your arms, stretch your arms and feel that tension...and now relax. Get your arms back into a comfortable position. Let the relaxation proceed on its own. Your arms should feel comfortably heavy as you allow them to relax...Straighten your arms once more so that you feel the tension in the triceps muscles; straighten them. Feel the tension...and relax. Get your arms comfortable and let them relax further and further. Continue relaxing your arms even further. Even when your arms seem fully relaxed, try to go that extra bit further; try to achieve deeper and deeper levels of relaxation.

Let all of your muscles go loose and heavy. Just settle back quietly and comfortably. Wrinkle up your forehead now; wrinkle it tighter...and now stop wrinkling your forehead, relax and smooth it out. Picture your entire forehead and scalp becoming smoother as the relaxation increases... Now, close your eyes tighter and tighter...place all of your tension in your eyes....and relax your eyes. Keep your eyes closed, gently, comfortably, and notice the relaxation....Now clench your jaws, bite your teeth together; study the tension throughout the jaws....relax your jaws now....learn to appreciate the relaxation....Now press your lips together tighter and tighter....relax the lips. Note the contrast between tension and relaxation. Feel the relaxation all over your face. The relaxation progresses further and further....

Now attend to your neck muscles. Press your head back as far as it can go and feel the tension in the neck; roll it to the right and feel the tension shift; now roll it to the left. Straighten your head and return to a comfortable position, study the relaxation, let the relaxation really develop....Now shrug your shoulders, pull them up and try to touch your ears. Hold the tension....drop your shoulders and feel the relaxation. Neck and shoulders are relaxed. Shrug your shoulders again and move them around. Bring your shoulders up, forward, and back. Feel the tension in your shoulders and in your upper back...drop your shoulders once more and relax. Let the relaxation spread deep into the shoulders, right into your back muscles; relax your neck and throat, and your jaws and other facial areas as the pure relaxation takes over and grows deeper....deeper....ever deeper.

Relax your entire body to the best of your ability....feel that comfortable heaviness that accompanies relaxation.... breathe easily...freely in and freely out. Notice how the relaxation increases as you exhale. As you breathe out just feel that relaxation. Now breathe right in, fill your lungs, inhale deeply and hold your breath....study the tension...now exhale, let the walls of your chest grow loose and push the air out automatically...continue relaxing and breathe freely and gently... feel the relaxation and enjoy it ... with the rest of your body as relaxed as possible, fill your lungs again, breathe in deeply and hold it again. That's fine, breathe out and feel the relief ... just breathe normally. Continue relaxing your chest and let the relaxation spread to your back, shoulders, neck and arms. Merely let go and enjoy the relaxation.

Now pay attention to your stomach muscles, make your stomach hard ... notice the tension ... and relax. Let the muscles loosen and notice the contrast. Feel the general well being that comes when you relax your stomach. Now draw your stomach in, pull the muscles right in and feel the tension this way, now push out and feel the tension ... once more pull in and feel the tension ... now relax your stomach fully ...Let the tensions dissolve as the relaxation grows deeper, deeper, ever deeper. Each time you breathe out notice the rhythmic relaxation both in your lungs and in your stomach ... notice thereby how your chest and stomach relax more and more.

Try and let go of all contractions anywhere in your body ... all parts relaxing further and further, ever deeper. Now relaxed as you are, I would like you to tense all the muscles in your right leg. Hold it ... and now relax. Let the muscles loosen as the relaxation takes over. Once more tighten the muscles of your right leg while the rest of your body stays as relaxed as possible ... and now relax. Relaxing more and more, ever deeper. Now push your right foot downwards away from your face. Feel the tension in your arch, ankle ... now return your foot to a comfortable and relaxed position. Now while the rest of your body stays as relaxed as possible, tense the muscles of your left leg ... hold it ...and now relax. Simply let the relaxation develop. Once more tense the muscles of your left leg ... and now relax ... relaxing more and more. Now, push your left foot downwards away from your face. Feel the tension in your arch, ankle ... and now relax your foot.

Keep relaxing like that for a while ... feel how heavy your muscles have become. In a state of perfect relaxation, you would feel unwilling to move a single muscle. Think about the effort that would be required to raise your right arm ... as you think about raising your arm, notice if tensions may have crept in. If so let them go and relax your arm more. Now you can become twice as relaxed as you are right now merely by taking two very deep breaths ... and slowly ... very slowly exhaling ... (wait 10 seconds). Now relaxed as you are, I would like you to imagine your own neutral scene, something for you that is comfortable and very relaxing.

## Appendix 2.6. Research Ethics Committee Approval Letter



25 June 2013

Dear Professor Mcmillan

### **MVLS College Ethics Committee**

**Project Title:** Asperger's Syndrome and Traumatic Brain Injury: The role of anxiety in executive function and theory of mind deficits

**Project No:** 200120054

The College Ethics Committee has reviewed your application and has agreed that there is no objection on ethical grounds to the proposed study. They are happy therefore to approve the project, subject to the following conditions:

- The research should be carried out only on the sites, and/or with the groups defined in the application.
- Any proposed changes in the protocol should be submitted for reassessment, except when it is necessary to change the protocol to eliminate hazard to the subjects or where the change involves only the administrative aspects of the project. The Ethics Committee should be informed of any such changes.
- If the study does not start within three years of the date of this letter, the project should be resubmitted.
- You should submit a short end of study report to the Ethics Committee within 3 months of completion.

Yours sincerely



Dr Dorothy McKeegan  
College Ethics Officer

Dr Dorothy McKeegan

Senior Lecturer

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**Appendix 2.7 Adjusted means for ASD and TBI at both time points, CI 95%**

	EF		ToM	
	Pre	Post	Pre	Post
<b>ASD</b>	-0.325, 0.618	-0.159, 0.743	-0.329, 0.738	-0.075, 0.895
<b>TBI</b>	-0.565, 0.311	-0.672, 0.166	-0.673, 0.318	-0.806, 0.095

## **Appendix 2.8. Major Research Project Proposal**

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### **Major Research Project Proposal**

Version No            11

Date                    17th June 2013

Word Count            3644

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Asperger's Syndrome and Traumatic Brain Injury: The role of Anxiety in  
Executive Function and Theory of Mind Deficits

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**Aisha Tariq**

**0905859**

**Research Supervisor: Professor Tom McMillan**

**Field Supervisor: Dr Jennifer Shields**

## **1. ABSTRACT**

### **Background**

Executive function (EF) and theory of mind (ToM) have been implicated as deficits in both Asperger's Syndrome (AS) and Traumatic Brain Injury (TBI). Research in both groups has also indicated prevalence rates of anxiety are consistently higher than in the general population. The relationship between anxiety and impaired performance on EF and ToM tasks has also been established, but with no investigation of how anxiety impacts on EF and ToM abilities in these populations. It has also been suggested that although AS and TBI groups have deficits in EF and ToM, the underlying mechanisms may differ. The present study aims to investigate these issues further by utilising an anxiety intervention and investigating the impact on ToM and EF in both TBI and AS.

### **Research Objectives**

The primary objective is to reduce anxiety in AS and TBI and evaluate the impact of this reduction in ToM and EF scores. It is hypothesised that reduced anxiety in AS will significantly improve ToM and EF scores but will have no impact in TBI, given the differing mechanisms suggested in AS. The secondary hypothesis is that EF and ToM will be significantly correlated in the AS group and not in the TBI group.

### **Methods**

A prospective between groups design will be utilised, in which those with AS and TBI will complete measures of ToM, EF and anxiety before and after receiving relaxation training.

### **Applications**

By establishing the role anxiety has to play in the deficits observed in these population groups, future interventions can be tailored to better meet their needs.

## **2. BACKGROUND AND RATIONALE**

Asperger Syndrome (AS) is characterised as a pervasive developmental disorder with significant impairment in social interaction and stereotyped patterns of behaviour with no significant developmental delay (American Psychiatric Association, 2000). Hofvander et al (2009) described clinical characteristics of a sample of adults with Autism Spectrum Disorders. Fifty per cent of those with AS met DSM-IV criteria for anxiety disorder. Generalised anxiety disorder and obsessive compulsive disorder was most common, followed by social phobia and panic disorder. Gorden et al (2001) suggest that this high rate of anxiety in AS may be due to a vulnerability to stress resulting from restricted coping mechanisms. This may stem from the deficits that define the disorder. Conversely, they suggest many of the behaviours that are typically described as originating from the disorder may be functionally related to a lack of coping skills, such as decreased social interaction as a result of being unable to cope with uncertain situations.

Tantam (2003) suggests that a growing self-awareness and increasingly complex and ambiguous social situations enhance the risk of people with AS developing mood and anxiety disorders. More recently, this developing insight has been recognised as a possible marker for interventions in AS, particularly cognitive behavioural therapy (CBT; Cardaciotto & Herbert, 2004; Spek, van Ham & Nyklicek, 2013; Gaus, 2011).

Another clinical population with similar characteristics is adults with severe Traumatic Brain Injury (TBI). In terms of anxiety, Hibbard, Uysal, Kepler, Bogdany and Silver (1998) found rates of 19% for post-traumatic stress disorder (PTSD), 15% for

obsessive compulsive disorder (OCD) and 14% for panic disorder after TBI. Although not as prevalent, anxiety has nevertheless been a focus of intervention in TBI (Hsieh et al, 2012)

Theory of Mind (ToM) and Executive Function (EF) deficits have been widely established in both adults with Asperger's Syndrome (AS; Swettenham, 1996; Kana, Keller, Cherkassky, Minshew, & Just, 2009; Kleinhans, Akshoomoff, & Delis, 2005) and those with Traumatic Brain Injury (TBI; Henry, Phillips, Crawford, Ietswaart & Summers, 2006; Bach, Happe, Fleming & Powell, 2000; Martin-Rodriguez & Leon-Carrion, 2010).

ToM is the ability to attribute mental states (for example, knowledge, beliefs, desires) to both oneself and others, and to understand that others can have thoughts, beliefs and knowledge that are different from our own (Rasmussen, Talwar & Wyper, 2009). The development of ToM appears to be strongly related to the development of EF, which is a set of processes that monitor, control, and supervise both thought and action, including self-regulation, inhibitory control, planning, attentional flexibility, error correction and working memory (Carlson, Moses & Breton, 2002; Hughes, 1998). However, the nature of the link between ToM and EF is an area of continued debate (Bibby & McDonald, 2005; Perner & Lang, 1990). There is some evidence suggesting an overlap in the brain regions involved in ToM and EF tasks, namely the prefrontal cortex (PFC; Rothbart & Posner, 1985; Channon & Crawford, 2000; Sabbagh and Taylor, 2000).

The relationship between anxiety and impaired performance on EF tasks has long been noted (Darke, 1988; Hopko, Crittendon, Grant & Wilson, 2005). Similar to studies in the AS population, individuals experiencing high levels of anxiety have decreased functional connectivity, particularly between distal regions of the brain involving the medial PFC (Ding et al, 2011). Kim et al (2011) suggest that stronger functional connectivity between the amygdala and the ventral medial PFC predicted lower levels of anxiety, suggesting that a less coherent amygdala-ventral medial PFC connection predicts higher levels of anxiety. Thus, it is hypothesised in the current study that the mechanism underlying the deficits in AS are more similar to anxiety disorder than to TBI.

Unfortunately there is currently a paucity of research investigating the impact of anxiety in AS on performance on neuropsychological tests and ToM measures. This is surprising given the observations of increased anxiety in this population, as well as the known impact of anxiety on EF and ToM described above.

Nevertheless, Shields and McMillan (2010) investigated the role of EF and ToM in individuals with AS and TBI, relative to controls. They found that EF and ToM was impaired in both AS and TBI, compared with controls. In the AS group, EF and ToM were correlated, but not in the TBI group. However, on covarying for anxiety, ToM differences between the AS group and controls became non-significant, thus suggesting differing mechanisms underlying the TBI and AS deficits. The present study aims to investigate this further by utilising an anxiety intervention and investigating the impact on ToM and EF in both TBI and AS.

### **3. STUDY OBJECTIVES**

#### 3.1 Primary Research Objective

Reducing anxiety will significantly improve ToM and EF scores in the AS group but reducing anxiety levels in TBI will have no impact on performance.

#### 3.2 Secondary Research Objective

EF and ToM will be significantly correlated in the AS group and not in the TBI group.

### **4. STUDY PLAN AND PROCEDURES**

#### 4.1 Overall study design

This study will utilise a prospective between groups design comprising individuals with AS and TBI. In both groups, participants will complete EF, ToM and anxiety measures before and after receiving relaxation training.

Application of inclusion and exclusion criteria to those consenting to take part in the study will allow selected participants to be invited to attend a two hour appointment. A telephone number will be provided if questions or concerns arise prior to appointment.

Upon presentation, 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 lengths depending on their levels of comfort. The anxiety measure will be completed and scored in-session. If participants are happy to proceed, the EF and ToM tests described below will be administered, followed by a scripted APRT

session. The script will be consistent for all participants. This will be followed by the second EF and ToM battery of tests below.

If participants are observed to be struggling to attend to tasks or seem fatigued, the researcher will discuss this with the participant and offer further breaks if this will facilitate completion of the study. Participants will be reminded of their choice to terminate the sessions at any time.

## 4.2 Selection of study population

Participants will be individuals who have a diagnosis of Asperger's Syndrome for the AS group and individuals who have experienced a severe traumatic brain injury for the TBI group.

### 4.2.1 Inclusion criteria

- Adults aged 18-65 years old.
- Within the AS group, participants will have a diagnosis of Asperger's Syndrome as defined in the DSM-IV-TR (American Psychiatric Association, 2000).
- Within the TBI group, participants will have a diagnosis of severe Traumatic Brain Injury defined as a Glasgow Coma Scale score of less than nine (Jennett and Teasdale, 1974) or Post Traumatic Amnesia lasting one day or more (Russell & Nathan, 1946).
- First language is English.
- Participants functioning above the learning disability range (IQ > 70) and able to consent to participate in the study.

#### 4.2.2 Exclusion criteria

- Dual diagnosis of TBI and AS.
- Those with any current chronic psychiatric condition or presenting with symptoms of trauma.
- Anyone identified with significant levels of risk to self or others.
- Lack of capacity under the Adults with Incapacity Act.

#### 4.2.3 Subject information and consent

See appendices for information sheet and consent form. Potential participants from all accessible organisations will be sent an information sheet outlining the aims of the study and a letter of invitation with an attached consent form. These organisations will include:

- The National Autistic Society
- The Autism Resource Centre
- University of Glasgow Learning Disability Service
- Headway Glasgow
- Headway Ayrshire
- Dirrans Centre (Ayrshire)

If returned, their suitability for inclusion will be checked. 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. Any questions participants have at this point which will inform their choice to participate will be addressed.

#### 4.2.4 Discontinuation/ Withdrawal of participants from study

Consent may be withdrawn at any point during the study. All participant data would be erased.

## **5. STUDY INTERVENTION/TREATMENT**

### 5.1 Description of Study Intervention/Treatment

Abbreviated Progressive Relaxation Training (APRT) will be utilised, which has an established clinical efficacy (Powlow & Jones, 2002). This technique takes about twenty minutes and is a shortened version of Jacobsen's (1939) original Progressive Muscle Relaxation approach, involving tensing and relaxing 16 different muscle groups. A standard script will be employed following Bernstein and Borkovec's (1973) standard procedure (See Appendix).

## **6. STUDY MEASUREMENTS AND ENDPOINTS**

### 6.1 Primary and Secondary Outcome measures

Theory of Mind and Executive Function are the primary measures. Anxiety pre- and post- intervention will allow impact of intervention to be assessed.

### 6.2 Methods of assessment

Tests of ToM:

Pre-

- Half of the Reading the Mind in the Eyes Test (Baron-Cohen, Wheelwright, Hill, Raste & Plumb, 2001).
- Half of the Faces Test (Baron-Cohen, Wheelwright & Jolliffe, 1997).

Post-

- Part Two of Reading the Mind in the Eyes Test.
- Part Two of the Faces Test.

Tests of EF:

Pre-

- Symbol Search from the Repeatable Battery for the Assessment of Neuropsychological Status (A; RBANS; Randolph, 1998)
- Verbal fluency using BDT
- Reverse Digit Span from RBANS (A)
- Half of the Hayling Sentence Completion Test of Dysexecutive Syndrome (Burgess & Shallice, 1997).

Post-

- Second half of the Hayling Test
- Symbol Search from RBANS (B)
- Verbal fluency using FAS
- Reverse Digit Span from RBANS (B)

Measure of anxiety:

- State-Trait Anxiety Inventory (Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983). The Trait anxiety measure will be administered at the beginning of the session and the State anxiety will be pre- and post-intervention. Granacher (2003) suggests this is a useful tool for evaluating anxiety throughout rehabilitation and treatment for TBI.

## **7. DATA MANAGEMENT**

NHS and University of Glasgow Codes of Confidentiality will be followed at all times. Initially, data gathered will be in paper form. All data will be transferred onto an encrypted laptop issued by the University. Data will be anonymised before entering on computer; each participant will be allocated a number and this will be used to store all information on the database, e.g. scores on the above tests. Only the researcher and supervisor will have access to the raw data.

## **8. STATISTICAL METHODS AND DETERMINATION OF SAMPLE SIZE**

### 8.1 Determination of sample size

Power calculations were conducted using G\*Power 3 (Faul, Erdfelder, Land & Buchner, 2007). To date, there are no published studies comparing AS and TBI populations. The studies below were therefore chosen due to their use of similar measures and subsequent methods of analysis.

Power was calculated for the main hypothesis using Pawlow and Jones' (2002) data since a similar experimental design will be used. The State Trait Anxiety Inventory was used which indicated significantly lower anxiety in the experimental group post-intervention. G\*Power calculation based on post-intervention data comparing the experimental group ( $M = 25.8$ ,  $SD = 5.6$ ) and the control group ( $M = 34.3$ ,  $SD = 7.5$ ) indicated 22 total participants, 11 in each group, would be required for a power of 0.8 and alpha of 0.05.

In relation to ToM, Spek, Scholte and van Berckelaer-Onnes (2010) utilised the Reading the Eyes in the Mind task in an AS group ( $M = 11.86$ ,  $SD = 3.88$ ). Geraci,

Surian, Ferraro and Cantagallo (2010) used the same task in TBI ( $M = 21.2$ ,  $SD = 4.2$ ). If comparing data across these two studies,  $G$  the effect size is  $2.31$ ; with power of  $0.8$  and alpha  $0.05$  a total of ten participants (five per group) are needed.

For EF, Shields et al's (2010) data compared AS and TBI on the Trail Making test. The AS group had  $140.6$  ( $SD = 50.7$ ) seconds remaining to complete the task on average, and the TBI group had  $97.9$  ( $SD = 63.1$ ); with power of  $0.8$  and alpha of  $0.05$  total of 30 participants would be required (15 per group).

Power was calculated for the secondary hypothesis again based on the data from Shields et al (2010). A positive correlation was found between composite scores for EF and ToM in the AS group ( $r = 0.57$ ). G\*Power indicates nine participants in the AS group would have power of  $0.8$  to detect alpha at  $0.05$ .

Thus, a total of sample size of 30 participants, with 15 in each AS and TBI group would be sufficient to meet the research hypotheses.

## 8.2 Statistical analysis

Data will be analysed using the Statistics Package for Social Sciences (SPSS) Version 18 as described below.

- ANCOVA investigating effects pre-intervention and post-intervention.
- Correlational analyses investigating relationship between EF and ToM in AS and TBI.

Checks for normality, homogeneity of variance will be undertaken prior to parametric tests.

## **9. SAFETY AND RISK ASSESSMENT**

Each participant will be interviewed individually with levels of risk to both participant and researcher being assessed on an ongoing basis. The sessions will take place within a pre-arranged clinical setting.

## **10. STUDY MANAGEMENT**

### 10.1 Ethics

#### 10.1.1 Ethical conduct of the study

Potential issues may include capacity and risk, which will be established prior to the sessions. Given the temporal length of the sessions, it will also be necessary to monitor participants' levels of fatigue and discomfort. The main researcher will monitor and appropriately manage participants. Regular breaks will be offered and the sessions will be paced to support the participants in completing the study comfortably. Any issues presented by participants who cause concern will be referred to their General Practitioner, if not already engaged in specialist services. Supervision will be from Professor Tom McMillan. Domiciliary visits will not be undertaken and the researcher and participant will not be in an isolated environment.

#### 10.1.2. Ethics Review

Ethical approval will be sought from the University of Glasgow after approval of the Protocol by the Department of Health and Wellbeing at University of Glasgow.

### 10.2 R&D Management Approval

Since no NHS sites or NHS staff are involved in the study, NHS R&D approval is not required.

### 10.3 Finance and Indemnity

The tests of EF, IQ and mood will be sourced from the Department of Health and Wellbeing, University of Glasgow. Assessments of ToM will be resourced from the Autism Research Centre where they will be downloaded for research purposes at no charge. Additional funding has been approved from the University to cover costs of stationary, postage and standardised assessment recording sheets.

### 10.4 Study timetable

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<b>March 2013</b>	<b>Submission of research proposal</b>
<b>March – April 2013</b>	Seek ethical approval
<b>May – Sept 2013</b>	Recruitment
<b>August – Sept 2013</b>	Data collection
<b>October 2013</b>	Conclude recruitment and complete data collection
<b>October – Nov 2013</b>	Data analysis and write up
<b>November 2013</b>	Draft submission to supervisors
<b>December 2013</b>	Final submission

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### 10.5 Dissemination

Submission to a peer-reviewed journal and internal report to local Health Boards. Presentations to participating sites will be offered.

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