INVESTIGATING THE EXISTENCE, COGNITIVE ATTRIBUTES AND POTENTIAL PATHOLOGICAL CONSEQUENCES OF THE EXTREME FEMALE BRAIN

Sarah Louise JONES

Submitted for the degree of Doctor of Philosophy

Division of Psychology
Faculty of Social Sciences
University of Bradford

2016

Abstract

INVESTIGATING THE EXISTENCE, COGNITIVE ATTRIBUTES AND POTENTIAL PATHOLOGICAL CONSEQUENCES OF THE EXTREME FEMALE BRAIN

Sarah Louise Jones

Key words: extreme female brain, empathising, systemising, sex differences, autism, schizophrenia, memory, cognition, gene imprinting, paranoid ideation

The 'extreme female brain' (EFB) is derived from the empathising – systemising theory (E-S) which hypothesises that sex differences in cognition exist on a continuum, based on abilities in 'empathising' and 'systemising' (Baron-Cohen, 2003). The EFB profile; extreme empathising alongside deficient systemising, has received little attention in social cognitive neuroscience research, compared to the extreme male brain, which has advanced the knowledge of sex differences in the expression of autism.

Currently, there is no solid evidence of a clinical pathology relating to the EFB nor a marker of cognition associated with a person's 'place' on the E-S continuum. Here, an episodic memory paradigm with social and non-social conditions was given to participants along with measures of empathising and systemising. Scores on the social condition predicted where a person lies on the E-S continuum. The thesis then investigated the hypothesis that schizophrenia is expressed in the feminised profile (Badcock & Crepsi, 2006)

and the presumption that empathising and systemising demonstrate a trade-off.

Elements of paranoia were associated with an empathising bias. However, a bias in systemising ability was associated with schizotypy along with a significant overlap in the expression of autistic traits and schizotypy. Therefore, schizophrenia as a whole is unlikely to be the pathology seen in the EFB, rather, the *positive symptoms of schizophrenia*. A trade-off between empathising and systemising was seen but *only* in participants over 36 years. These results have significant implications for assessment and treatment of neuropsychological disorders and provide more specific details on the potential EFB pathology.

Acknowledgments

I dedicate this thesis to my Dad, who has supported me throughout my whole PhD journey. Thank you for all your encouragement and support.

First and foremost, a massive thank you to my super amazing primary supervisor Dr Valerie Lesk for all her inspiration, endless encouragement, help and guidance throughout the project.

I would also like to thank my 2nd supervisor Dr Gill Waters for her support throughout this project, and Rebecca Durrans for her help with recruitment of participants and statistical analysis support. Also, thank you to everyone who has come and gone from 'E5' for your encouragement and most importantly, friendship.

I would like to say a huge, special thank you to Laura; you have supported me in every way throughout this PhD and I couldn't be more grateful.

To my whole family, thank you for all your support.

Acknowledgements and thanks are owed to the Autism Research Centre at Cambridge University for making their cognitive tests and inventories available for academic use.

And finally, a huge thank you to each and every one of the participants who gave their time to contribute to the project.

Table of Contents

List of Abbreviations

AM	associative memory
ANOVA	analysis of variance
AQ	autism spectrum quotient
ASD	autism spectrum disorder
BPD	borderline personality disorder
CAM-MR	Cambridge mind-reading face-voice battery
CogDis	O-LIFE cognitive disorganisation
D/R-M	Deese/Roediger-McDermott paradigm
EAT-26	eating attitudes scale
ED	eating disorders
EFB	extreme female brain
EFT	embedded figures task
EMB	extreme male brain
EQ	empathising quotient
EQ-S	empathising quotient (short form)
E-S	empathising-systemising
E = S	balanced brain
E > S	greater empathising than systemising
E >> S	greater empathising than systemising (extreme)
FB	folded box test
FNE	fear of negative evaluation
fT	fetal testosterone
GAD-7	GAD-7 Anxiety

GLM	general linear model
IBT	imprinted brain theory
ImpNon	O-LIFE impulsive nonconformity
InAn	O-LIFE introvertive anhedonia
IPT	intuitive physics test
JTC	jumping to conclusions
M	mean
MANOVA	multivariate analysis of variance
MNS	mirror neuron system
MROT	mental rotation task
MTL	medial temporal lobes
n	number
ns	non-significant
OAIB	over-attribution image battery
O-LIFE	Oxford and Liverpool Inventory of feelings and experiences
PCC	paranoia checklist (conviction)
PCD	paranoia checklist (distress)
PCF	paranoia checklist (frequency)
PFC	prefrontal cortex
PI	paranoid ideation
PPQ	physical predictions questionnaire
PPT	physical predictions test
PT	pairing task
RMTF	reading the mind in the films

RTME	reading the mind in the eyes
±SD	standard deviation
S > E	greater systemising over empathising
S >> E	greater systemising over empathising (extreme)
SJT	sweet jar task
SM	source memory
SME	source monitoring error
SPSS	statistic package for the social sciences
SQ	systemising quotient
SQ-S	systemising quotient (short form)
schizophrenia	schizophrenic spectrum disorders
ToM	theory of mind
UnEx	O-LIFE unusual experiences
VIF	variance inflation factor
WR	word recall

Chapter 1

Thesis overview

1.1 Introduction and aims

The 'extreme female brain' (EFB) profile (Baron-Cohen, 2003) has captured the attention of psychologists and cognitive neuroscientists in recent years (Brosnan et al., 2010; Russell-Smith et al., 2010; Smit, 2010, Jones & Lesk, 2013), mostly because of the simplistic, yet intriguing account of the significant influence sex differences may have on human cognition and the manifestation of psychological disorder.

The EFB profile is characterised by a high level of social intelligence (empathising) alongside a severe deficit in mechanical style thinking (systemising) (Baron-Cohen, 2003). An example of empathising is the intuitive ability to understand another person's emotional state. Examples of systemising would be *intuitive* understanding of mathematical equations, physics, or map reading and spatial awareness (Baron-Cohen et al., 2003).

The EFB concept is derived from the empathising – systemising theory (E-S) (Baron-Cohen, 2003); a model which hypothesises that every person can be placed on a continuum of cognitive profiles based solely upon measurement of their individual ability in both empathising and systemising intelligence (See Figure 1).

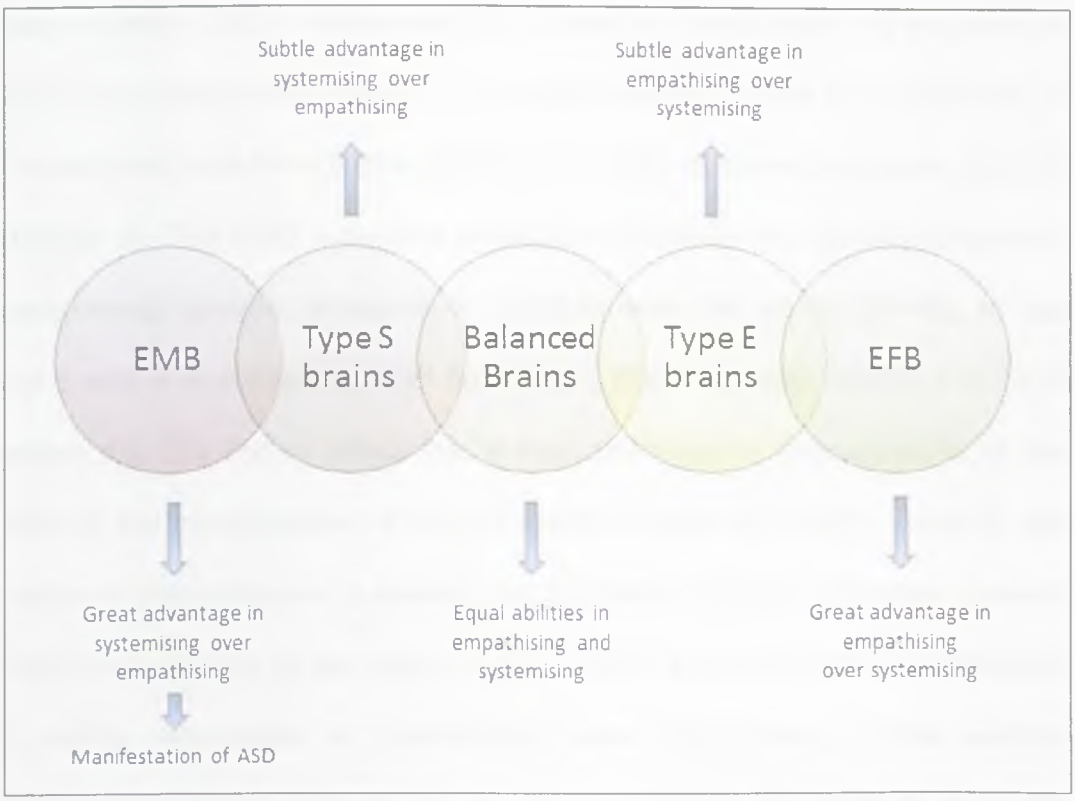


Fig. 1: The E-S model

The EFB is located at one end of the E-S continuum, where Baron-Cohen (2003) claims that this brain type is void of any pathological consequence, because no known pathological disorder is fitting with a cognitive profile that presents with an extreme deficit in systemising alongside an advanced ability in empathising.

The E-S theory is based on the notion that sex differences in the brain have become hard-wired through a process of evolutionary adaptation to historical social gender roles (Baron-Cohen, 2003). Baron-Cohen (2003) offers that the main instigator for our preference for empathising and systemising styles of thinking (and therefore our place on the continuum) is ***not*** due to our biological sex, but instead the level of fetal testosterone (fT) we have been exposed to during neurodevelopment.

Baron-Cohen (2003) introduces the concept of 'brain types' to the field of social cognitive neuroscience. At one extreme end of the E-S continuum is the extreme male brain profile (EMB) (or sometimes termed 'extreme type S') (Figure 1). The EMB individual presents cognitively with greatly advanced systemising abilities, alongside a deficit in empathic ability (S>>E), to the point where empathising is all but absent (Baron-Cohen, 2003). It is here where the E-S theory infers that a neuropathological consequence of the EMB is the manifestation of autism spectrum disorder (ASD). Towards the centre of the continuum is moving into the territory of the male brain (known from here onwards as the 'type S brain'), (S>E) a profile which demonstrates a subtle advantage in systemising over empathising; some autistic tendencies may be evident, but do not disrupt daily life so much as to be considered clinically relevant. In the middle of the continuum is the balanced brain (E=S), where systemising and empathising abilities are equal, this is the brain type that the majority of the population will be categorised into, simply because most people will demonstrate neither an advantage or disadvantage in empathising or systemising (Baron-Cohen, 2003). Further along the continuum will be the female brain (E>S) (known as 'type E' profile from this point onwards); this person will demonstrate a subtle advantage in empathic ability alongside slightly less ability in systemising. At the opposite end of the continuum to the EMB profile is the EFB; which in Baron-Cohen's own words (2003, p.170) is 'unknown terrain' and void of any known pathological consequence, unlike the EMB.

At present, the E-S theory claims that no pathological consequence occurs as a result of a feminised brain profile (Baron-Cohen, 2003). However, a recent theory has in-directly suggested that the EFB may be implicated in schizophrenia (Badcock & Crespi, 2008). This theory has not received great amounts of empirical investigation. This thesis explores the relationship between the feminised (type E) brain profile and schizotypy in non-clinical populations, by investigating individual phenotypes of schizotypy in relation to the E-S theory. This investigation was based on the notion that ASD and schizophrenia exist as diametric opposites on a continuum (Badcock & Crespi, 2008). This postulation belongs to the imprinted brain theory (IBT); a model that suggests that if an extreme bias in gene imprinting occurs during neurodevelopment, either towards paternal imprinting (ASD) or maternal imprinting (schizophrenia), the consequence is the manifestation of psychological disorder (detailed discussion of the IBT is found in Chapter 5).

The reporting of investigations in sex differences in cognition can sometimes be met with claims of 'neurosexism' (Fine, 2010). In that some academics believe sex difference research is driven by a political agenda (Fine, 2008). However, this thesis takes the position that there is ample evidence for sex differences in cognition and brain anatomy (Chapters 2); therefore, it is important to continue with the study of sex differences in the brain to allow us to gather an understanding of how they affect behaviour, and how these *innate* differences might influence the expression of psychological disorder. Ultimately, a comprehensive understanding of sex differences may be useful for research into therapeutic treatments (sex-specific or otherwise).

The experimental research presented throughout this thesis investigates whether there is a potential pathological consequence of the feminised profile. It takes into consideration that in an attempt to link the EFB profile with schizophrenia, previous works have failed to recognise that schizophrenia consists of *numerous* individual components, that may share specific relationships with empathising and systemising. Therefore a more accurate investigation into the IBT hypothesis may be derived from investigation into the individual phenotypes of schizotypy, rather than quantifying schizophrenia as a whole.

In addition to this primary investigation, this thesis aimed to advance the knowledge pertaining to the links between the E-S continuum and other cognitive networks, such as memory ability. In an attempt to discover if cognitive abilities that have not previously been considered, are implicated in the E-S model. Furthermore, this thesis analyses the validity of the E-S theory and the IBT in their account of sex differences in the brain, by testing their fundamental assumption that empathising and systemising cognition share a 'trade-off' relationship. In that, as one ability advances (empathising), the other diminishes (systemising) and vice versa. This was rationalised to be an important secondary aim of this thesis for three main reasons; firstly, the majority of previous work pertaining to the E-S concept had neglected to test this important relationship, and in those that did, results were inconsistent. Secondly, as this thesis progressed, it was observed that results were clearly demonstrating a lack of support for this *assumed* trade-off between empathising and systemising. Thirdly, the theoretical concept of the EFB

profile (the primary investigation in this thesis) is dependent upon the fundamental notion of a trade-off between empathising and systemising, therefore it became pertinent to address this inconsistency.

1.2 Introducing the main concepts

The main concepts that will be referred to throughout this work are detailed below.

Sexual dimorphism – the term sexual dimorphism refers to phenomena that exists in 'two forms', so that there is no, or very little overlap between them (McCarthy & Konkle, 2005). This is a term often used incorrectly in the neuroscientific research of sex differences, as the structure of the male and female brains *overlap greatly,* even though they show structural and anatomical differences.

Sex differences – sex differences are the result of determining effects of sex hormones that subsequently influence sex specific cognitive traits and behaviours (McCarthy & Konkle, 2005).

Trait level – here in this thesis, the term 'trait' refers to measureable individual differences of phenotypes of particular neuropsychological disorder. These traits are non-clinical, they are apparent in healthy individuals and do not cause disruption to daily living.

Gender differences – this term refers to differences in behaviour that exist between men and women that are rooted in biology but have been primarily influenced by social norms and expectations.

Gene imprinting – this term refers to the parent of origin effect of expressed genes, where one of the two genes inherited from our parents is silenced and the other is expressed (Morison et al., 2001). A concept that the IBT is built upon (discussed further in Chapter 5).

Cognition – refers to a range of processes that involve comprehension of higher order thinking, including 'attention, decision-making, self-regulation, problem solving, language, and memory' ability (Etkin et al., 2013, p.419).

Theory of mind – a subdomain of social cognition, theory of mind is the ability to attribute another person's mental state, wishes, beliefs and intentions to oneself (Gooding & Pflum, 2011).

Empathising – the term empathising can be *broadly* used interchangeable with theory of mind. It refers to two factors (i) the ability to recognise another person's emotional state (theory of mind) and (ii) the ability to appropriately respond to that state of mind; termed 'affective empathy' (Baron-Cohen, 2009).

Systemising – is a term specifically associated with the E-S theory. It refers to the intuitive ability to understand without effort the mechanical workings of

a 'system'. It is the drive to analyse the components in said system (Baron-Cohen et al., 2003; Larson et al., 2015), and construct systems that are based on rules and have predictable regularity (Baron- Cohen 2009).

Empathising - Systemising theory – brings together empathising and systemising abilities to predict that everyone can be placed on a continuum, based on a 'trade-off' between the two processes. Most people will be placed in the centre of the continuum, as they will most likely demonstrate equal ability in empathising and systemising (e.g., balanced brains). However, some people will place towards the ends of the continuum with a greater ability in empathising alongside a subtle deficit in systemising (type E), or vice versa (type S). Biological sex is a predictor of empathising and systemising abilities, with males more likely to present as systemisers and females as empathisers (Baron-Cohen, 2003), however, biological sex is simply a predictor of empathising and systemising ability, *not* a determinant.

Extreme male brain – is the observation that ASD presents as an extreme of male typical sex differences (Andrew et al., 2008). The EMB profile is one which is highly advanced in systemising ability, whilst having a severe deficit in empathising abilities.

Extreme female brain – is a theoretical cognitive profile that demonstrates highly advanced empathising alongside a severe deficit in systemising (Baron-Cohen, 2003).

Imprinted Brain Theory – a model based in genetics that hypothesises that ASD and schizophrenia exist on a continuum of cognitive profiles as diametric opposites (Badcock & Crespi, 2006). The IBT predicts that a bias towards maternal gene imprinting during neurodevelopment results in the manifestation of schizophrenia, whereas a bias towards paternal imprinting results in the manifestation of ASD.

1.3 Overview of thesis structure

The thesis consists of 11 chapters. Chapters 1 to 5 are introductory chapters where the necessary background information is presented. The following five chapters are experimental (Chapters 6–10), where empirical findings obtained throughout the data collection period of this research project are communicated. The final chapter (Chapter 11) comprises of a conclusive discussion that debates and interprets the empirical findings, along with reasoning for further research. The following provides an overview of each chapter:

Chapter 2 begins this thesis with a general overview of the current knowledge surrounding sex differences in cognition. The male/female advantages and disadvantages on particular neuropsychological tasks are discussed, along with a detailed discussion of neuroanatomical and neurochemical differences. Alternative factors that may influence sex-specific behaviours are offered and the chapter concludes with a discussion of the controversies surrounding sex difference research in neuroscience.

Chapter 3 introduces the E-S theory – the primary theory on which this thesis concentrates. This chapter offers a comprehensive discussion of the aetiological presumptions and evolutionary basis of the manifestation of empathising and systemising cognition. It continues onward with discussion of the previous research that has offered both behavioural and neuroanatomical support for the principles of the E-S model and concludes with relevant critique and issues surrounding the E-S theory concept.

Chapter 4 moves on to discussion of the pathological consequences of the E-S theory by concentrating on the EMB theory of ASD. Firstly, a detailed, general discussion of ASD is presented, followed by discussion of Baron-Cohen's (2002) hypothesis that ASD is manifest due to the consequence of male sex differences at an extreme level. This chapter concludes with a brief discussion of how a feminised profile at an extreme level may be implicated in schizophrenic pathology.

Chapter 5 introduces the IBT hypothesis that ASD and schizophrenia exist on a continuum of diametric opposites based on social brain intelligence (Badcock & Crespi, 2006) as a potential theory to explain pathological consequence of the feminised profile. This is followed by a general introduction of schizophrenia; including discussion of different schizophrenic conditions and the notion that schizophrenia can be dimensional in relation to the expression of particular symptoms. The chapter concludes with the rationale for the argument that positive schizophrenia (phenotypes that

include paranoia, hallucination and delusion [Mason & Claridge, 2005]) may be an accurate characterisation of the EFB.

Chapter 6 is the first of the experimental studies conveyed in this thesis. As far as the researcher is aware, a relationship between other cognitive processes and how they are implicated in the E-S model has not yet been investigated. This is important, as this investigation furthers the knowledge of how far reaching the E-S continuum could be in terms of explaining how sex differences are implicated in other networks. A two-part study is reported which employed a novel episodic memory task paradigm, which was administered in a student population sample to explore the hypothesis that type E brains would demonstrate greater ability in episodic memory recall, alongside greater susceptibility to source monitoring failure. Results demonstrated that memory ability was a significant cognitive marker on the E-S continuum, specifically when information involved a social context. This demonstrated that other cognitive networks are significantly implicated in the E-S theory, a notion that had not previously been considered.

Chapter 7 explores the hypothesis that type E profiles have the potential to be associated with schizophrenia (Badcock & Crespi, 2008) eating disorder (ED), and anxiety disorder (AD) traits (Bremser & Gallup, 2012) by employing a method not previously used in the literature. Using a variety of self-report inventories, results revealed that type S brains scored significantly greater than both balanced and type E brains on scores of schizophrenic traits. This is a somewhat surprising and significant finding, which calls into questions

the accuracy of the IBT's prediction of a relationship between the type *E* brain profile and positive schizotypy.

Chapter 8 considers the curious findings reported in the previous chapter. Chapter 8 explored the relationship between empathising-systemising bias (E-S bias) and *specific* components of schizotypy. This was for the purpose of considering the *individual* components of schizotypy in relation to empathising and systemising, rather than schizotypy as a whole; a concept that has not previously been explored in the psychology literature and could potentially be causing some discrepancies in research findings. A novel task paradigm measuring paranoid ideation (PI) and jumping to conclusions bias (JTC) (a concept which refers to the amount of information a person requires before making a rational decision) was administered in a non-clinical population. Results demonstrated that performance on the JTC task (but not PI) was significantly affected by E-S bias, with greater empathisers and lesser systemisers showing greater tendency to jump to conclusions. Supporting the notion that a feminised profile can be associated with *specific components* of schizotypy.

Chapter 9 assesses the *exact* nature of the relationship between ASD and schizophrenic traits, in an adult neurotypical sample using a validated self-report measure of dimensional schizotypy. Results demonstrate that ASD and schizophrenia traits (overall) were positively correlated, suggesting an *overlap* of diagnostic phenotypes rather than the *diametric* one – significantly

rejecting the IBT hypothesis and offering implications for the ASD – schizophrenia continuum model.

Chapter 10 reports the last experimental study in this thesis. A two-part study which focused on the fundamental trade-off presumption between empathising and systemising. This study administered and interpreted data from validated psychological tasks and inventories to explore social and spatial cognition in a student sample (Study 1) and general population sample (Study 2). Results demonstrated a general dependency between empathising and systemising, unless age-related effects are considered. A trade-off relationship **is** evident in adults over the age of 36 years, suggesting that the trade-off relationship between empathising and systemising is much more complex than previously understood and importantly, is changeable over the lifespan. This again has significant implications not only for the E-S model, which as far as the researcher is aware has never been investigated with respect to age, but also in treatment and understanding of certain neuropsychological conditions that may be associated with empathising and systemising processes.

Chapter 11 brings together the main findings from the experimental studies presented in this thesis. It discusses the implications that these findings have for the assessment, diagnosis and treatment of neuropsychological disorders. It also discusses how this research has significantly contributed to the knowledge surrounding the EFB concept and social cognitive

neuroscience literature in general. Concluding remarks are offered along with a discussion surrounding areas for future directions.

Chapter 2

Sex differences in cognition

2.1 Introduction

We are fascinated by sex differences (Eliot, 2011; Eagly et al., 2012; Joel, 2012; McCarthy et al., 2012; Mendrek, 2015). The mention of sex differences in the brain is often met with a gasp of apprehension about what pseudoscience is going to be reported next. Why is it that we find the topic of sex differences so controversial? Is it because ultimately, we want to know if either sex has superiority? Do we want scientific justification for social norms and pressures in terms of gender equality or oppression? Some maintain that the differences between the sexes are vast (Kimura, 1996; Goldstein et al., 1998; Moffat et al., 1998; Baxter et al., 2003; Sommer et al., 2004; Hamilton, 2008; Cook & Saucier, 2010; Chou et al., 2011; Ingalhalikar, et al., 2013), whilst others say that anyone who purports such statements are simply sexist or endorsing a political agenda (Fine, 2010, 2012).

There is support that our brains are *innately* gendered (Connellan et al., 2001), however at the same time, it is difficult, structurally and anatomically speaking, to find distinguishing signs of whether or not a brain is male or female (Joel et al., 2015). Thus, making the study of sex differences one of the most controversial and complex areas of research in psychology and neuroscience (Cahill, 2006; Jordan-Young & Rumiati, 2011).

Sex differences are of interest because ultimately, they may help us understand gender specific human behaviour (Wizemann & Pardue, 2001). This notion seems like it would be a strong driving force for research;

however, sex differences remain a curious and hesitant subject for neuroscientists, due to the political and sociological implications the findings can have (Cahill, 2014).

It was not until the 1990s when neuroscientists began to look closely at sexual dimorphism and sex differences in the brain in order to gather a more comprehensive understanding of the brain basis of observable sex differences in cognition and behaviour (Cosgrove et al., 2007). Whilst the majority of research was in favour of a clear evidence base for the manifestation of sex differences, astonishingly, it was only in 1993 that the NIH (National Institutes of Health [USA]) made it mandatory for female subjects to be included in clinical traits (Beery & Zucker, 2011). This apparent oversight provides an indication as to how the possible confounding factor of sex difference and sexual dimorphism has been discounted in considering the interpretation of neuroscientific studies. Sacher et al. (2011) suggest that because researchers do not consistently report the biological sex of participants in experimental research, we do not yet have a comprehensive picture of how biological sex truly influences cognition. However, on the other side, Beery and Zucker (2011) offer that it is not yet mandatory to include both sexes in animal studies, suggesting that the implications of sex differences are ignored as it is *assumed* that the results are applicable to both males and females. This is an important consideration, as results are almost always inferred to be generalizable to the whole population regardless of gender. Further to this, the fact that psychological disorders often show sex related differences, and can effect prevalence rates (e.g., ASD, schizophrenia, depression, AD, substance abuse, [Eaton et al., 2012]

Angelman syndrome and Prader-Willi syndrome [Knoll et al., 1989; Kirkilionis et al., 1991; Oliver et al., 2007; Buiting, 2010]), makes the study of sexual dimorphism and sex difference *vital* in order to gain a progressive understanding of how the influence of sex hormones may affect psychological disorder, and provide clues as to effective treatment strategies (Cosgrove et al., 2007).

2.2 Sex difference in cognitive ability

Baron-Cohen and Hammer (1997) argue that there is a great amount of evidence to suggest that cognitive biases are evident in males and females (Cook & Saucier, 2010). For instance, findings show that *on average* females' exhibit better performance on emotional memory, social sensitivity, emotion recognition, verbal fluency, verbal memory, language comprehension and executive function tests, whereas males perform better on tasks which involve visuospatial processing, such as imaginative rotation, spatial perception, motor ability, navigational and mathematical tests (Kimura, 1996; Goldstein et al., 1998; Moffat et al., 1998; Baxter et al., 2003; Sommer et al., 2004; Hamilton, 2008; Wallentin, 2009; Cook & Saucier, 2010; Chou et al., 2011; Ingalhalikar, et al., 2013; Jones & Lesk, 2013; Miller & Halpern, 2014). Whilst Miller and Halpern (2014) point out that it must be remembered that these sex differences are *dependent* on the characteristics of the tasks employed to measure these apparent differences; collectively, Ingalhalikar et al. (2013) suggest that these findings support the notion that the male brain is *structured* to expedite connectivity between perception and co-ordinated actions. Whereas the female brain is better at assisting

communication between reasoned and intuitive processing (Ingalhalikar et al., 2013). However, whilst there is vast support for these cognitive differences, it seems equally important to point out that *realistically*, differences in cognitive abilities seldom amount to more than half a standard deviation (Weiss et al., 2003). It is suggested that differences in cognitive abilities differ more within genders as they do between genders (Hyde, 2007; Neave & O'Connor 2008), however whilst sex differences are very subtle, statistically, they do exist.

2.3 Neuroanatomy of sex differences

As well as a clear difference between the sexes on cognitive tasks, neuroanatomical differences between males and females are widely acknowledged (Sowell et al., 2007), despite being controversial (Cahill, 2014). Relatively recent advances in neuroimaging have allowed for a non-invasive way of exploring neuroanatomical differences pertaining to biological sex, instead of relying on post-mortem and animal studies to uncover the structural and anatomical brain differences between males and females (Gong et al., 2009; Sacher et al., 2011).

According to Bao and Swaab (2010), sex differences in the neuroanatomy of the brain are 'countless' (see Figure 2). For instance, males have a larger cranium then females, regardless of relative body size (Allen et al., 2003;

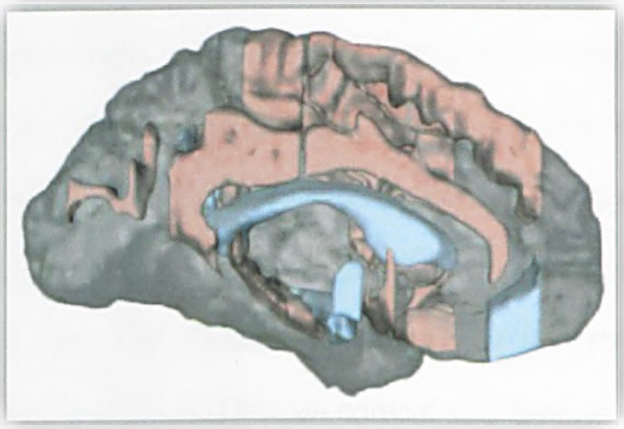


Fig. 2. The pink areas are reflective of structures of the brain thought to be larger in females; the blue areas are representative of the areas larger in males.

Image credit to Cahill (2006)

Chen et al., 2007; Gong et al., 2009). During childhood, male brains are on average 9% larger than females (Giedd et al., 1997). Whereas women have shown to have more grey matter than males (Leonard et al., 2008; Ingalhalikar et al., 2013), however, this has been disputed, with some studies finding decreased grey matter in females or no sex differences when overall brain size is controlled for [Courchesne et al., 2000; Good et al., 2001; Ge et al., 2002; Sullivan et al., 2004]). Furthermore, male brains are more 'asymmetrically organised' than females, with no apparent age-related effects (Cahill, 2014).

According to Sacher et al. (2011), the areas in the brain that tend to show greater volume in males include the left inferior temporal gyrus, the right occipital lingual gyrus, the right medial temporal gyrus and both cerebellar hemispheres. On the other hand, women consistently show greater volume of grey matter in the dorsal anterior, ventral cingulated cortices and the right inferior parietal lobule (Sacher et al., 2011). Sex differences in the corpus callosum are also evident, in that the structure is larger in females compared to males (Allen et al., 1991; Leonard et al., 2008), whereas the hypothalamus

is greater in males compared to females (Bao & Swaab, 2010). The hippocampus and amygdala also show differences during the childhood years; the amygdala increases in size for males compared to females, whereas the hippocampus increases in size greater for females compared to males (Giedd et al., 1996, 1997). On account of this, both Sowell et al. (2007) and Bao and Swaab (2010) suggest that these apparent sex differences between male and female cortical structures should take into account the age of the cohort employed in the study. As age can significantly influence anatomical structure, therefore can be considered a confounding variable that is not always controlled for.

Levels of intelligence do not differ between the sexes, however males and females use different areas of the brain to achieve the same IQ scores (Cosgrove et al., 2007). For instances males IQ scores 'correlate with volume of gray matter in the frontal and parietal lobes' whereas in females IQ score correlates with, 'gray matter volume in the frontal lobes and Broca's area' (Cosgrove et al., 2007, p. 4). Generally, females tend to demonstrate more efficient overall cortical connectivity than males (Gong et al., 2009). Yet at the same time, sex differences are associated with different patterns of neuronal activation. For instance, males show better neural activation to emotional faces in the limbic and prefrontal areas, with females demonstrating greater activity in the right subcallosal gyrus for the same stimuli (Fusar-Poli et al., 2009; Sacher et al., 2011). Different patterns of activation according to gender have also been reported in the processing of sad faces (Lee et al., 2002) and visuospatial processing activates different areas of the brain in males and females (Gura et al., 2000; Sacher et al., 2011). Interestingly, in

relation to visuospatial processes, Moffat et al. (1998) tells us that the male advantage in spatial ability is the most reliable and consistent finding which pertains to sex differences in cognition. However, much more research is required to fully understand exactly how sex *specifically* influences these processes (Sacher, et al., 2011).

The study of neuroanatomical differences between male and female brains is often contradictory. Whereas anatomical differences are widely reported (as above), Jordan-Young and Rumiati (2011) state that the only 'structural difference that has been independently replicated is in INAH-3, a tiny cell group in the hypothalamus that is larger in men than women' (p. 4). Suggesting that sex differences in neuroanatomy are not as obvious as the literature proclaims. Joel et al. (2015) offer an interesting study that examined the magnetic resonance imaging (MRI) of over 1400 human brains. The study revealed widespread overlap between structures in male and female brains and concluded that to come across a brain that is *obviously* male or female is *extremely rare*. Therefore, whilst they acknowledge sex differences are evident, they do not support the categorisation of brains into the strict male and female brain classifications (Joel et al., 2015).

2.4 Biology, neurochemistry and sex hormones

There is evidence to suggest biology plays an important role in sex differences (Collaer & Hines, 1995; Ngun et al., 2012). However as previously alluded to, the notion that sex differences are solely the result of innate organising effects is widely debated (Fine, 2010). In an attempt to discover if sex differences have a biological or social determinant,

Connellan's et al. (2000) study looked for sex differences in new-born babies at age one hour old. Their findings were suggestive of an innate biological determinism of sex differences, in that, new-born girls were more inclined to focus for longer on faces. Whereas boys focused longer on mechanical objects when presented with visual stimuli placed above their cots. This suggests that from the very beginning of life, a preference for emotional stimuli (empathising) or mechanical stimuli (systemising) was evident for boys and girls respectively (Connellan et al., 2001).

An influential animal study that claimed to observe that preference for particular stimuli is evident at an early age (and void of social gender role influence) is that of Alexander and Hines (2002). They reported that girls (green vervet monkeys) preferred to play with stimuli that spoke to their empathic intuition (e.g., dolls), whereas boys preferred to play with toy cars and balls, hinting that these cognitive 'preferences' are biologically innate.

We can say with some certainty that cognitive differences are due to the organisational effects of sex hormones (Baron-Cohen, 2003). Particularly the hormone testosterone, an androgen that influences sexual dimorphism in that, surges of testosterone drive masculinisation and the absence of testosterone determines feminisation (Neave & O'Conner, 2008; Bao & Swaab, 2010; Baron-Cohen et al 2011). Sex hormones have an organising influence on the brain during neurodevelopment and throughout life (Hampson, 1990). There are two particularly influential periods of testosterone surge during mid-pregnancy and within the first three months after birth (Bao & Swaab, 2010).

It has been proposed that higher levels of fetal testosterone (fT) influence the growth of the right hemisphere (Manning, 2001), and it is suggested that this may be a reason spatial abilities are *on average* better in males (Burkitt et al., 2007; Auyeung et al., 2008; Barbeau et al., 2009; Auyeung et al., 2011). In studies exploring levels of testosterone (plasma) in relation to cognition, higher fT levels have been shown to positively correlate with enhanced systemising abilities (Connellan et al., 2001; Baron-Cohen et al., 2004; Hines, 2010; Whitehouse et al., 2010). Further, evidence linking fT levels to sex differences in cognition is offered by Lutchmaya et al. (2002) who found that the amount of time a one-year-old maintains eye contact and the quality of social relationships, is inversely correlated with higher levels of testosterone. This suggests that lower levels of fT are associated with better *social* aspects of development. In addition, Whitehouse et al. (2010) found females who showed higher levels of fT presented with poorer language ability, which fits with the notion that there is a male disadvantage in language processing. Further, the Cambridge fT project (Baron-Cohen et al., 2004; Auyeung et al., 2006), is a longitudinal study of psychological variables of normally developing children whose fT levels were sampled during amniocentesis. The study found fT levels were inversely correlated with eye contact at one year of age, language ability and quality of relationships at four years and empathic traits at eight years. The levels of fT were found to positively relate to systemising traits at three years and better performance on the embedded figures test (EFT) (see section 6.3.2.2 for description of this task) at eight years (Baron-Cohen et al., 2011). This indicates that levels

of fT *significantly* contribute to the cognitive biases that are evident between males and females.

2.5 Sex differences and disorder

As referred to previously, at extreme levels, sex differences have a significant influence on the manifestation of neuropsychological disorder (Romano et al., 2016). Here in section 2.5, a brief summary of some of the disorders that are affected by sex differences are offered. A *detailed* discussion of ASD and schizophrenia in relation to sex differences is offered in Chapter 4 and Chapter 5 respectively.

We have evidence that sex differences are related to psychological disorder from the prevalence figures that show higher rates of incidence for one particular sex; for instance, Bao and Swabb (2010) claim that in cases of Rett syndrome, lympocytic hypophtsitis, anorexia and bulimia and hypnic headache syndrome, 75% of the cases are female. On the other hand, we see that, males are reported to present with dyslexia, attention deficit disorder, ASD, sleep apnea, Tourette's syndrome, Kallmann syndrome and Kleine-Levin syndrome in over 75% of cases (see Bao & Swaab, 2010 for further details on prevalence figures). A particularly relevant disorder that is *greatly* influence by sex difference is schizophrenia (Badcock & Crespi, 2006). Greater numbers of males present with the negative symptoms such as flattened affect and social withdrawal, whereas females show a greater level of positive symptoms such as hallucinations and delusions, suggesting sex differences are a significant modulating factor in the expression of the

disorder (Badcock & Crespi, 2006). However, Bao and Swaab (2010) argue that the notion that psychological disorder *arises* from sex differences is not established and that it may be the case that sex difference affects the already established disorder.

2.6 Considerations

As previously specified in section 2.3, neuroanatomically speaking, it is accepted that sex differences exist in the brain (Goldstein et al., 1999), however caution is suggested when interpreting results found by neuroimaging, as there is a tendency to 'infer mental states' (Fine, 2010) from such data. The 'sheer complexity of the brain, together with our assumptions about gender, lend themselves beautifully to over interpretation' (Sutton 2010, p.900) and some academics make the suggestion that neuroscience seemingly ignores the rationale that the brain is *soft wired* and is simply reflective of the environment. They suggest that neuropsychological tests are not evidence enough to offer staunch support for these so-called 'hard-wired' differences, which are gendering the brain (Hyde, 2007; Fine, 2008, 2010). Fine (2010) suggests that the irresponsible reporting of sex differences in anatomy, chemistry and cognition have resulted in a wave of 'neurosexism'.

The sociologist would argue that the reason behind these observed sex differences is constructionism, in that people conform to social roles and therefore become practiced in that gender role rather than having an innate preference towards empathising and systemising behaviours (Banjani & Hardin, 1996).

Whilst it is the position of this thesis that there are clear sex differences in anatomy, brain chemistry and cognition, influenced by organising effects of sex hormones, rational thinking tells us that the similarities that exist between the sexes heavily outweigh the differences. This does not mean however, that sex differences should be ignored; they are an important factor in the interpretation of scientific studies and should be considered. However, it is acknowledged that there is often a media culture of inaccurately reporting novel sex differences for the purpose of creating interesting headlines for the general public (Eliot, 2011). This often results in an air of pseudoscience around the study of sex differences in that findings are often embellished and subsequently misinterpreted (Maney, 2016). Equally, some take the position that the reporting of sex differences can be a bargaining tool for political or misogynistic agendas (Halpern, 2012), with Halpern (2012) offering a warning that 'one of the most distressing outcomes of modern neuroscience is the way the findings are being misused to advance political agendas'. Thus, neuroscientists have a responsibility to communicate results pertaining to sex differences mindfully and responsibly.

> *"Biological and behavioural differences between the sexes range from obvious, to subtle or non-existent. Neuroanatomical differences are particularly controversial, perhaps due to the implication that they might account for behavioural differences"* (Leonard et al., 2008, p.2920).

Another area of consideration that needs to be addressed here is the structure to function issue as referred to by Leonard et al. (2008) in the quote above. Do these observed anatomical differences equate to behavioural and cognitive differences between the sexes? *It is likely*, but not proven that

behavioural and cognitive sex differences are due to differences in neuroanatomy (Gur et al., 1999). The structure - function relationship between the brain and behaviour is informed by research that *presumes* a relationship between brain and behaviour based on volume of brain areas relative to task performance. For instance, Baron-Cohen (2009) offers that areas involved in language abilities such as Wernicke and Broca's area (Harasty et al., 1997), the dorsolateral prefrontal cortex and the superior temporal gyrus (Schlaepfer et al., 1995), are larger in girls, and girls tend to have better aptitude for language use compared to boys (Baron-Cohen, 2009). Therefore, there seems to be evidence for a tentative link between structure and function. However, as Halpern (2012) offers, it is a long leap from neurons to actual behaviour (see Roskies, 2009 for detailed debate).

Whilst these controversies are important to consider, this thesis takes the position that there is a clear basis for the notion of sex differences in the brain (Cahill, 2014). Neuroscience still has a way to go in determining the exact nature of the implications biological sex has on brain structure and function. However, there *is* ample evidence to suggest sex differences are an important consideration and research should continue to further the current knowledge regardless of the controversies.

2.7 Chapter summary

This chapter has presented the case that sex differences are quantifiable in males and females. It has considered the validity of the evidence for differences in cognitive aptitude between males and females that are *consistently* reported in the literature. In addition, this chapter has considered

the anatomical and structure differences in the brain between the sexes, at the same time acknowledging the debate that differences are likely to vary *within* the genders as they do between them. This thesis aims to contribute to the debate of sex differences in cognition by focusing on two of the current and most prominent models of sexual differentiation- the E-S theory and the IBT, to investigate their ability to accurately link sex differences to pathology at trait symptom level. The E-S theory is introduced in detail in the following chapter.

Chapter 3

The empathising – systemising theory

3.1 Introduction

Rumiati and Humphreys (2015) review the recent progress neuroscience has made in relation of the knowledge of the neural basis of social behaviour, which provides a link between the brain basis of social behaviours and other disciplines of psychology such as social psychology. The investigation into E-S theory is an apt example of this progression, as concepts such as empathising and systemising could be argued to refer to learnt behaviour through a process of social development. However, Baron-Cohen's (2003) E-S theory is rooted within developmental cognitive neuroscience, which aims to define empathising and systemising as abilities that are *innate*, determined by strict biological organising effects.

The E-S theory can be defined as a model that is aiming to explain sex differences in human cognition, by suggesting that the difference can be explained by quantifying empathising and systemising abilities; two distinctions which Baron-Cohen (2003) suggests 'gender' the brain. The model aims to quantify the whole population on a continuum of cognitive profiles, born out of the observation that ASD presents as the *extreme* of male typical behaviours such as higher levels of testosterone, increased systemising and reduced empathising ability (Baron-Cohen, 2002) (*Note:* the EMB concept is discussed in greater detail in Chapter 4). This led Baron-

Cohen (2003) to question whether the observation of extreme sex differences in ASD could inform the field of psychology about the role of sex typical behaviours in the neurotypical population.

Given the success the EMB has had in progressing the knowledge in relation to ASD, in that it is a well-accepted characterisation of ASD pathology (Baron-Cohen, 2002). Investigation into the *EFB* and the feminised profile has the potential to discover psychopathological phenotypes that may be affected or determined by a hyper-feminised – hypo-masculinised brain, which is the primary aim of this thesis.

According to the E-S theory, the whole population exists on a continuum of cognitive profiles (Figure 1, p.2) which are defined by ability to empathise and systemise. Empathising, according to Baron-Cohen (2008), consists of two factors. Firstly, the ability to recognise another person's emotional state, which is termed cognitive empathy. Secondly, an ability to *appropriately* respond to said state, termed, affective empathy (Barn-Cohen, 2003). Ultimately, empathising is the tool we use to make sense of the social world around us (Muncer & Ling, 2006). Systemising on the other hand, is the ability to understand or construct systems; it is an ability to understand phenomena that work according to 'laws', examples of systemising would be map reading, spatial awareness, or effortlessly understanding the laws of physics (Baron-Cohen, 2009). Systemising allows us to develop rules to assist our understanding of how *things* work (Muncer & Ling, 2006). Empathising and systemising abilities could be loosely referred to as 'people people' and 'things people' respectively (Badcock & Crespi, 2008, p.1055).

In terms of how empathising and systemising are quantified in the lab; usually, cognitive task paradigms that involve theory of mind (ToM) or false belief tasks are used as proxies to measure empathising abilities (Baron-Cohen et al., 2001). Whereas tasks involving spatial awareness, mental rotation, folk physics and global versus local processing (such as the EFT) are often employed to measure systemising ability (Vandenberg & Kuse, 1978; Linn & Petersen, 1985; Parsons et al., 2004).

3.2 The evolution of empathising and systemising

The E-S theory posits that females are *hardwired* to empathise and males are *hardwired* to systemise; that our biology distinguishes between male and female. This drive subsequently produces differences between the sexes in brain anatomy and structure, as well as sex specific cognitive and behavioural characteristics (Baron-Cohen et al., 2001, 2003; Baron-Cohen, 2003).

However, interestingly, in terms of the underpinning of these apparent innate sex differences, the E-S theory hypothesises that sex differences have ***become*** hardwired through a process of brain plasticity, as a responsive adaptation to historical social gender roles (Baron-Cohen, 2003). Recent research suggests that there is now ample evidence to suggest that brain plasticity differs between the sexes, and that specifically, males are 'more susceptible...to perturbations in genes involved in synaptic plasticity' which indicates involvement of brain plasticity in male bias disorders such as ASD (Mottron et al., 2015, p.1). This suggests that evolution has the ability to reflect social norms and gender roles, and as a response has advanced the

higher order mental processes associated with these roles. Thus, females have *evolved* to be more empathic based on their historical social gender role as the caregiver, whereas males have *evolved* to have an advanced understanding of systemising phenomena based on the hunter-gatherer gender role (Baron-Cohen, 2003).

3.3. Support for the E-S theory

Studies using the EQ (Baron-Cohen & Wheelwright, 2004) and SQ (Wakabayashi et al., 2006) (appendix 1 & 2), (self-report inventories developed specifically to measure these traits), have shown support for the principles of the E-S theory (Goldenfeld et al., 2005; Baron-Cohen, 2008; Barbeau et al., 2009). Males tend to score higher on the self-report systemising quotient (SQ) (Baron-Cohen et al., 2003) and lower on the empathising quotient (EQ) (Baron-Cohen & Wheelwright, 2004) than females, and likewise, females score higher on the EQ and lower on the SQ than males (Baron-Cohen et al., 2004). Generally, empathising and systemising abilities have been found to negatively (though very weakly) correlate, suggesting that they are independent, yet related mechanisms, implying a trade-off relationship between the two processes (Baron-Cohen et al., 2003; Baron-Cohen & Wheelwright, 2004; Golan et al., 2006; Wheelwright et al., 2006; Focquaert et al., 2007; Bronsan et al., 2010; Von Horn et al., 2010). *Significantly, the notion of this trade-off is the most important concept of the E-S theory. It is the fundamental presumption that the theory is built upon. Interestingly, very little research has investigated this assumption (Chapter 10 of this thesis investigates this).*

In addition, studies employing psychometric tests to directly measure these abilities also support the principles of the E-S theory, (Goldstein et al., 1998; Cook & Saucier, 2010; Chou et al., 2011). Suggesting (as previously referred to in Chapter 2), that females tend to demonstrate a subtle advantage in emotional memory, ToM, verbal fluency and executive functions (Voyer et al., 1995; Baron-Cohen et al., 1997; Goldstein et al., 1998; Cook & Saucier, 2010; Chou et al., 2011). Whereas males perform better on spatial tests, such as the mental rotation task (MROT) and navigational or mathematical tests (Voyer et al., 1995; Baron-Cohen et al., 1997; Goldstein et al., 1998; Cook & Saucier, 2010; Chou et al., 2011). However, it is important to point out that whilst there is a wealth of evidence for sex differences in cognitive ability, as offered previously, these cognitive biases differ more *within* genders as they do *between* genders (Hyde, 2007; Neave & O'Connor, 2008).

3.4 Brain types

Central to the concept of the E-S theory and previously introduced in section 1.1 of this thesis (also see Figure 1 in Chapter 1), is the idea of 'brain types'. To briefly recap; at one extreme end of the continuum, is the EMB profile, where individuals present cognitively with greatly advanced systemising abilities, alongside severe deficit in empathic ability (S>>E), to the point where empathic ability is all but absent. It is here where the E-S theory infers that a neuropathological consequence of these extreme biases towards the 'male' cognition will be the manifestation of ASD (Baron-Cohen, 2002). Continuing back towards the centre of the continuum is the type S brain,

which demonstrates a subtle advantage in systemising over empathising (S>E), some autistic traits may be evident, but not clinically relevant. In the middle of the continuum is the balanced brain (E=S), where systemising and empathising abilities are equal; the brain type which the majority of the population will fall into (Baron-Cohen, 2003). Towards the opposing end of the continuum is the type E brain (E>S), which demonstrates subtle advantage in empathic ability alongside slightly less ability in systemising. Further along the continuum is the EFB (E>>S), a profile which presents with far advanced empathic ability alongside minimal to absent systemising aptitude (Baron-Cohen, 2011). There is no known pathological consequence which occurs here.

3.5 Biological determinants of the E-S theory

As discussed in section 2.4 of Chapter 2, level of exposure to fT during neurodevelopment has an organising effect on the brain (Auyeung et al., 2009, 2011; Baron-Cohen et al., 2005). The amount of fT is associated with the expression of male typical behaviours (Neave & O'Conner, 2008) and has been shown to positively correlate with enhanced systemising abilities in general populations (Connellan et al., 2001; Lutchmaya et al., 2002; Baron-Cohen et al., 2004; Hines, 2010; Whitehouse et al., 2010). According to the E-S theory, level of fT expression is the determining factor in the manifestation of 'brain types', ***not*** biological sex (Baron-Cohen, 2003). Pertinent to the discussion here is the condition Congential Adrenal Hyperplasia (CAH). CAH occurs when a female foetus is exposed to higher levels of prenatal testosterone. Because of this, masculinisation of the body

occurs and 'male' typical behaviours are observed (Neave & O'Conner, 2008). Therefore, we can deduce that fT is the driving force in the manifestation of sex differences, regardless of biological gender. CAH shows a lower (masculinised) digit ratio (2D:4D method) (Brown et al., 2002; Putz et al., 2004; Galis et al., 2010), demonstrating a direct effect of sex hormone dysregulation on neurodevelopment, irrespective of biological sex (Servin et al., 2003; van der Beek et al., 2004). Returning to the digit ratio as mentioned above, this is a popular (Voracek & Dressler, 2007) yet contentious (Voracek, 2009) method which is used as a proxy to measure the levels of exposure to fT, which is thought to be a probable biomarker for the levels of prenatal testosterone in the brain (Manning, 2002). Apparently, the level of prenatal testosterone influences the growth of the forth digit (ring finger) during early development, more specifically, between the 8^{th} and 14^{th} week of gestation (Gooding et al., 2010), whilst prenatal oestrogen influences the growth of the second digit (index finger), for reasons not yet understood (von Horn et al., 2010). In the lab, measurements are taken for the difference between the second and fourth digit and this figure is representative of the 2D:4D measurement. Research suggests that higher levels of exposure to fT results in a low or masculinised digit ratio and lower exposure, results in a higher or feminised digit ratio (Fink et al., 2006; Lutchmaya et al., 2004). Males generally have a low (masculinised) and females a higher (feminised) digit ratio (Lutchmaya et al 2004; Putz et al 2004; Voracek 2008; Barbeau et al 2009). Correlations between the 2D:4D method and E-S theory have been found, in that lower empathising has been associated with higher digit ratio (Skuse, 2009; Baron-Cohen, 2009; Jones & Lesk, 2013) and systemising,

with lower digit ratio, in the general population (Baron-Cohen et al., 2004; Auyeung et al., 2006; Baron-Cohen, 2009; Manning et al., 2010; Wakabayashi & Nakazawa, 2010), following the predictions of the E-S theory. However, this pattern is not *always* supported and other research offers inconsistent results (see Manning, et al., 2010; von Horn et al., 2010; Voracek & Dressler, 2006; Falter et al., 2008). Although, whilst the E-S theory can boast support from these findings, there is suggestion that the 2D:4D method is far too in-direct and its' aetiology is too poorly understood to be a validated measure of fT levels (Baron-Cohen et al., 2009), therefore studies involving direct measures of testosterone are preferred.

3.6 Neuroanatomical support

Generally, the majority of support for the principles of the E-S theory has been derived from behavioural data (Baron-Cohen et al., 2001; Baron-Cohen et al., 2003; Baron-Cohen & Wheelwright, 2004). However, a few recent studies have furthered the knowledge pertaining to the brain basis of both empathising and systemising. Whilst much more research is required, interesting findings have been yielded. For instance, in a study investigating cortical activation in relation to systemising, Billington et al, (2008) concluded that increased activation in the parietal and extrastriate visual cortices and the lateral prefrontal cortex, was correlated with higher SQ score and enhanced performance on the Navon task, (a task which measures systemising ability). From this, it can be inferred that enhanced systemising ability is related to enhanced activation in these brain areas. Furthermore, neuroimaging studies have begun to shed light on the structure - function

relationship between the E-S model and areas of the brain. Takeuchi, et al. (2013) employed voxel-based morphometry and reported that empathising ability was *positivity* correlated with regional white matter volume (rWMV) and systemising was *negatively* correlated with rWMV in the right inferior parietal lobule and temporoparietal junction. Takeuchi, et al. (2013) also found support for greater mirror neurone (MN) activity associated with empathising. They also claim that their results demonstrate the first imaging support for the trade-off relationship between empathising and systemising – a concept that is as yet unresolved (Andrew et al., 2006). In another study, Jack et al. (2013) reports an fMRI study, which suggests that there is a significant relationship between the two brain networks that control spatial and social cognition. In that, whilst completing psychological tasks associated with social and spatial cognition, a reciprocal suppression of brain networks was apparent. For instance, when participants were completing problem-solving tasks, the introduction of a task involving social cognition *deactivated* the network used for completion of the non-social spatial task, and vice versa. Jack et al. (2013) concluded that this provides evidence that it is difficult for these two networks to work together simultaneously.

In terms of the brain basis of empathising, several important brain regions have been implicated, specifically, the orbito and medial frontal cortex, superior temporal sulcus and the amygdala (Baron-Cohen & Ring, 1994; Frith & Frith, 1999; Baron-Cohen et al., 1999, 2000). Interestingly, the brain basis of systemising remains to be studied in-depth (Baron-Cohen et al., 2003). Therefore, this area remains severely underdeveloped (Dinsdale et al., 2016). However, Baron-Cohen (2009) suggests that if the brain basis of

both empathising and systemising were fully established, the E-S theory of sex differences would be difficult to discount.

3.6.1 Mirror neuron system

Here, it is important to consider the potential role of the human mirror neuron system (hMNS) in relation to the E-S theory. Frith and Frith (2010) suggest that the hMNS is the brain basis of empathic understanding. Originally observed in macaque monkeys (Gallese et al., 1996; Rizzolatti et al., 1996; Rizzolatti & Craighero, 2004), mirror neurons (MN) are a system of neurons which respond when an action is carried out and also when a similar goal orientated action is performed by another person (Williams et al., 2001). This system has provided an interesting basis for understanding social behaviours such as imitation and empathy (Iacobona & Dapretto 2006) (see Hamiliton et al. [2007] for review of hMNS in relation to empathic processing). In relation to the E-S theory, it would be hypothesised that the hMNS would show greater activity in the type E brain. Cheug et al. (2009) offers in-direct support for this hypothesis, using voxel-based morphometry analysis. Cheug et al. (2009) studied the underlying neuroanatomical mechanisms of MNs, synchronised with self-reports of empathy dispositions. Findings were suggestive of more activation in the female hMNS in relation to empathy (Cheug et al., 2009) providing support for this association. In addition, a study by Schulte-Ruther et al. (2008) also indirectly supports the E-S theory. During a functional magnetic resonance imaging (fMRI) study of males and females involved in an empathy task, involving emotion (expressed through facial expressions), activation of the regions involving the hMNS (in particular

the inferior frontal gyrus and Brodman's areas 44/45) was seen in both sexes, yet *stronger* activation was found in females (Schulte- Ruther et al., 2008), supportive of the involvement of the hMNS in empathic processes.

3.7 Considerations

Baron-Cohen (2003) attributes the process of innate sex differences to evolutionary brain plasticity which has responded to historical socialisation roles, a notion that is *reasonable* to theorise yet unresolved. Whilst Baron-Cohen presents a persuasive argument for gendered brains, the basis on which this theory was derived is in need of scrutiny. For instance, Baron-Cohen suggests that there is *'evidence beyond reasonable doubt'* that males and females are predisposed to sex differences, a discovery that (he suggests) is found in an influential study by Connellan et al (2000). Connellan et al. (2000) report that new-born male babies focus for longer on physical-mechanical objects rather than social objects (e.g., faces), compared to new-born females. This led the researchers to conclude that male and female brains are innately gendered as this method eliminated any form of socialisation. Whilst these results are interesting, it seems reasonable to consider that these findings *alone* do not provide enough evidence of innately gendered brains. For instance, some academics have highlighted that Connellan's et al. (2000) study is fraught with methodological issues. For instance, Nash and Grossi (2007) suggest that it is unclear as to whether or not new-borns are able to pay attention for sustained periods. Also, the social stimuli used in the study was the researcher's face in real time, whereas the mechanical stimuli were placed on a mobile over the child's cot. It seems

logical to suggest that attention paid to the appearance of a real life person is not comparable to attention paid to inanimate objects. Further to this, the babies were tested in different settings; in some instances, the babies were tested whilst laying in their cots, in other instances they were placed on their parents' laps (Nash & Grossi, 2007). Considering these factors, it is reasonable to suggests the authors may have overstated the significance of these findings, especially since these results have not been replicated in humans (although there is some evidence to support the notion of innately gendered brains without socialisation from research with primates [Hassett et al., 2008; Simpson et al., 2016]) and seemingly lack the principles of controlled scientific enquiry. It seems at times that Baron-Cohen's theories may slip into the dangerous territory of offering inflated statements. Further research is required to strengthen the fundamental principles of the E-S theory.

However, considering the principles of the E-S theory, that females are hardwired to empathise, whereas males are hardwired to systemise (Baron-Cohen, 2003); what is important to note here is that, when referring to these 'innate' abilities, what is being referred to are *biological components*. Whilst these biological components are determinants, it *must* be recognised they are also susceptible to environmental influence, they are not unchangeable, but instead develop *within*, and change according to the environment; a plasticity which remains into old age (Halpern et al., 2007). Baron-Cohen (2009) is often accused of been a biological determinist, however he offers a rebuttal; instead he insists that these cognitive profiles have a neurobiological determinism, which are not determined by sex *per se*, but the level of

exposure to androgen levels in utero (Auyeung et al., 2009). Therefore, although a person's biological sex is not a determinant of their cognitive profile, instead, it is simply a *good indicator* when dealing in statistical averages. As it happens, statistically, women show greater empathising tendencies and men show greater systemising abilities *on average* (Baron-Cohen, 2003). All the E-S theory is really claiming, is that cognition can be grouped by two main styles - empathising and systemising, irrespective of biological sex and based on level of exposure to fT (Baron-Cohen, 2003). This point is illustrated by the condition CAH (described in section 3.5) which occurs when a female foetus is exposed to very high levels of prenatal testosterone and subsequently presents with masculinisation and defeminisation, regardless of biological sex.

Whilst this thesis accepts the support offered in terms of the principles of the E-S theory, it takes the position that it is equally important to remember when considering the E-S theory, that sex differences seen in the healthy population amount seldom to more than a half a standard deviation (Weiss et al., 2003). Realistically, differences in cognitive abilities differ more *within* genders as they do *between* genders (Hyde 2007; Leonard et al., 2008). Yet, at the same time, statistically sex differences *do* exist, albeit very subtly. However, the pertinent question remains; could sex differences be important in helping explain certain neurodevelopmental disorders, specifically those that may relate to the EFB profile.

3.8 Chapter summary

This chapter has introduced the E-S theory (Baron-Cohen, 2003) - one of the primary theories which this thesis investigates. It has discussed the principles of the theory in relation to the idea that our brains can be gendered. This 'gendering' is based on our innate aptitudes for empathising and systemising abilities, which are manifest as a result of the level of fT the brain has been exposure to. This chapter also considered the support derived for the E-S theory in terms of behavioural data and neuroimaging research. The chapter concluded with the premise that the E-S theory may be able to explain how sex differences are implicated in neuropsychological disorder. A gap in the knowledge when considering the *feminised* profile (and the primary aim of this thesis), yet the highly masculinised brain profile has been widely researched; therefore, the following chapter moves on to introduce the EMB theory of ASD (Baron-Cohen, 2002).

Chapter 4

The extreme male brain theory of autism spectrum disorder

4.1 Introduction

The E-S theory (Chapter 3) was derived from the observation that ASD presents cognitively as the extreme of male typical behaviours (Baron-Cohen, 2002). The EMB has come a long way in advancing the knowledge pertaining to the role of sex differences in psychopathology (Dinsdale et al., 2016). In that, autistic individuals tend to demonstrate exemplar ability in systemising, alongside substantial deficits in empathising cognition – an extreme presentation of male typical behaviours (Baron-Cohen, 2002). This observation allowed Baron-Cohen (2002) to infer that there is *clearly* a link between biological sex and ASD. For instance, the prevalence of ASD according to biological sex is approximately 4.3:1 (Fombonne, 2007) and this, along with the extreme expression of masculinisation in ASD according to Baron-Cohen (2003), is nature's way of providing neuroscience with its biggest clue as to the elusive aetiology of ASD (Baron-Cohen, 2003).

4.2 Autism spectrum disorder

Often diagnosed in childhood, ASD (which includes Asperger's syndrome [Crespi et al., 2016]) is categorised as a neurodevelopmental disorder (Romano et al., 2016), *mainly* characterised by a tendency to partake in restricted repetitive behaviour, alongside difficultly with social intelligence and

communication (Rivet & Matson, 2011). There is no understood neuropathology of ASD, but it is recognised as a complex, genetically heterogeneous condition, which has a strong male bias in prevalence with males four times more likely to be diagnosed (Stogell et al., 2001; Constantino & Todd, 2003). However, interestingly, females are most severely affected (Crespi et al., 2016) (this observation is discussed in detail in the following chapter). Wing (1981) reports a more detailed investigation into sex and the prevalence of ASD and concludes that, in ASD with learning difficulties there is a significant but lower 2:1 male to female ratio, however, in higher functioning ASD including Asperger's syndrome, males are *15 times* more likely to have a diagnosis compared to females.

The autistic individual finds social communication difficult and often finds social situations *impossible* to comprehend (Larson et al., 2015). ToM deficit is the most recognisable difficulty in ASD, but delays in language development are also often present (Baird et al., 2006) (although this is not always the case in Asperger's syndrome), along with empathising and imagination difficulties, often resulting in social withdrawal (Baron-Cohen, 2002; Crespi et al., 2016). In addition, the autistic individual shows unusually narrow interests, obsessive repetitive behaviour and a preference for (and sometimes savant skills[1]) in systemising ability (Del Giudice et al., 2010). Del Guidice et al. (2010) offer an overview of ASD from the perspective of the **advantages** ASD involves. For instance, they highlight that advanced

[1] The autistic savant is an individual who presents with low-level intelligence, but shows genius level intelligence in specific subjects such as memory ability or mathematics (Fitzgerald, 2010).

abilities that are usually observed in ASD, such as systemising behaviours, can greatly help the ASD individual to be highly successful in pursuit of their interests, ultimately leading to the development of extremely useful novel skills, despite difficulties in other areas of intelligence. There is no readily accepted treatment of ASD, however the usual support that a ASD individual may receive is educational support, alongside occupational therapy and speech and language therapy (NHS, 2016b).

ASD exists on spectra of severity, including Asperger's syndrome (which presents with above average intelligence and normal language development however the autistic difficulty in social intelligence is usually observable [Geschwind, 2011]) The spectrum nature of ASD infers that ASD 'traits' are evident in non-clinical populations. These traits are subtle and not clinically relevant as they require no medical intervention, but nevertheless are quantifiable in the healthy populations (Constantino & Todd, 2003; Posserud et al., 2006; Baron-Cohen, 2011).

4.3 The extreme male brain

The EMB theory of ASD (Baron-Cohen, 2002) has been well received and has strong empirical support (Baron-Cohen et al., 2014). Its strength is in offering a more comprehensive explanation of autistic behaviour than previous accounts, such as the weak central coherence theory (Frith, 1989) and the 'mindblindness' theory (Baron-Cohen et al., 1985; Baron-Cohen, 1995) (Baron-Cohen et al., 2003). This is because it can explain reduced

ToM abilities *whilst* accounting for observable above average systemising abilities (Klin, 2009).

At a psychological level, the EMB theory infers that autistic individuals will score low on the empathising and high on the systemising ability (Baron-Cohen, 2009) – and research supports this postulation (Baron-Cohen et al., 2003; Baron-Cohen & Wheelwright, 2004). On tests of social cognition, people with ASD *consistently* perform worse than controls (Baron-Cohen et al., 1997; Baron-Cohen et al., 2001), but perform significantly better on tasks of spatial ability (Jolliffe & Baron-Cohen, 1997; Baron-Cohen et al., 1999), supportive of the notion that ASD presents as a masculinised profile.

Neurologically, there are interesting, supportive findings. The regions of the brain, which are on average smaller in males than females namely, the anterior cingulate, superior temporal gyrus, prefontal cortex and thalamus, are even smaller in autistics individuals, regardless of biological sex (Bauman & Kemper, 2005). In addition, regions that present as larger in males, namely, the amygdala, cerebellum, and the overall cortex, are larger still in ASD individuals (Baron-Cohen et al., 2005). In addition, ASD shows greatly reduced MN activation (Williams et al., 2001; Oberman et al., 2005; Theoret et al., 2005; Dapretto et al., 2006; Bernier et al., 2007) and higher levels of fT (Baron-Cohen, 2002; Lutchmaya et al., 2002; Auyueng et al., 2009; Barbeau et al., 2009; Auyueng et al., 2010; Gooding et al., 2010). These findings are not only suggestive of support for Baron-Cohen's (2003) claim that biological sex is not a *determinant* of brain gender or pathology, only a *predictor*, they

also suggest that sex differences in the brain can account for the manifestation of pathology in the masculinised profile.

4.3.1 The extreme female brain

The notion that sex differences at extreme levels can influence the manifestation of pathology (as in the case of the EMB theory of ASD), begs the question of the greatly under researched potential pathological consequence of a feminised profile.

Baron-Cohen (2003) posits that the opposing end of the E-S continuum, the EFB, will most likely be void of pathology, for two major reasons. Firstly, there is no diagnosable pathology that is *fitting* with a highly advanced empathising ability alongside a severe deficit in systemising ability (Baron-Cohen, 2003). Secondly, it would be much easier for the EFB individual to compensate for reduced systemising abilities in modern society with advanced technological aids that make the intuitive ability to systemise somewhat redundant (Baron-Cohen, 2003; Larson et al., 2015). Therefore, perhaps these profiles are less easy to observe. Furthermore, advanced empathising would likely present as a *great advantage*, affecting daily living in a positive way, rather than contributing to any observable deficit.

However, a novel hypothesis from the field of genetic research has prompted the question of whether schizophrenia would be a fitting pathology for the EFB profile; in that, they suggest ASD and schizophrenia are *one* disorder that presents diametrically in relation to social brain dysfunction (Badcock &

Crespi, 2006). This thesis now moves on to discuss this hypothesis in detail in the final introductory chapter, before moving on to report a series of experimental studies that aimed to explore this hypothesis.

4.4 Chapter summary

This chapter has introduced the concept of the EMB, a brain type on the E-S theory continuum, which presents cognitively with a severe *deficit* in empathising ability alongside a greatly *advanced* preference for systemising. It has discussed the research that has supported the notion that ASD can be *accurately* characterised by a masculinised profile at an extreme level. The chapter concludes with discussion of the *EFB* concept, and how the observation of pathology because of a masculinised brain profile, begs the question of possible pathology of the extreme feminised profile. The next chapter introduces the IBT (Badcock & Crespi, 2006), a model which may provide an accurate characterisation of the EFB.

Chapter 5

The imprinted brain theory

5.1 Introduction

The IBT (Badcock & Crespi, 2006) considers genetics, neurology and psychology to propose a model of sex differences in cognition that claim to explain the manifestation of both ASD and schizophrenia, determined by the organising effects of gene imprinting bias (Palkhivala, 2009).

An in-direct attempt to address the characteristics of the EFB (the main question of this thesis) can be inferred from Badcock & Crespi (2006,2008). They suggest that ASD and schizophrenia exist as related components at the extreme ends of a continuum (see Figure 3), with clinical diagnosis of ASD and schizophrenia at the extremes, along with the healthy population falling at different points along the continuum.

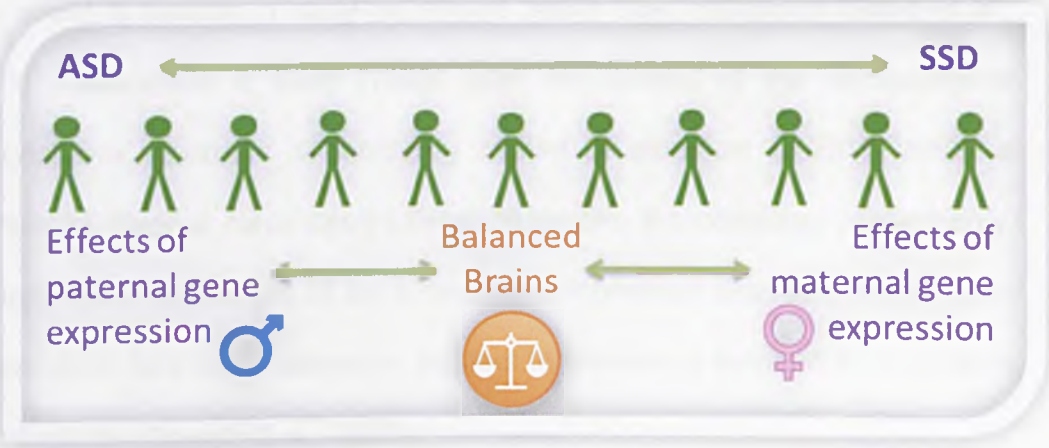


Fig. 3: the IBT model

Attributing their hypothesis largely to a complex 'tug of war' (Smit, 2010) in genomic imprinting, Badcock & Crespi (2008) hypothesise that a bias toward

either maternal or paternal gene expression during brain development results in a gendered brain. They conjecture that the *hyper*-development of social cognition as seen in the positive symptoms of schizophrenia (such as over attribution of the intentions of others, hallucinations or delusional episodes [Mason & Claridge, 2005; Lindenfors et al., 2007; Abu-Akel, 2008; Thakkar et al., 2008; Crespi, 2010]), may be the diametric opposite of the *hypo*-developed social brain (the inability to attribute intentions of others, lack of ToM [Baron-Cohen, 2002; Frith, 2004]) which is observable in ASD (Badcock & Crespi, 2008).

5.2 Overview of schizophrenia

Like ASD, schizophrenia is a heterogeneous condition (Lindsberg et al., 2009) where symptoms and behaviours exist on a continuum of severity (Binbay et al., 2011). However, it is different from ASD in one major way, in that onset is usually in early adulthood rather than childhood (Saha et al., 2005). Cadenhead & Braff (1999) offer that central to the schizophrenia concept are 'disorders of inhibitory control of attention' (p.231). Whilst a number of theories have been offered to explain the condition, increasingly, schizophrenia is thought of as a neurodevelopmental disorder, where brain organisation has been altered or subject to disruption *before* the expression of symptoms (Sowell et al., 2000).

Whilst schizophrenia exists at the extreme end of the ASD – schizophrenia continuum, schizotypy traits taper back along the continuum to exist in the general population (Cochrane et al., 2010; Ettinger et al., 2011). The current diagnostic thinking on schizophrenia, including at trait level, is that it is

multidimensional, in that a person may present with a certain group of symptoms which can be broadly categorised as positive, negative or disorganised schizophrenia (Fonseca- Pedrero et al., 2008).

Positive symptoms refer to the episodic experience of paranoid ideation, delusions and hallucinations (Steel et al., 2007). Negative schizophrenia refers to the tendency to be socially isolated, experience high anxiety and lack emotional expression (Del Giudice et al., 2010). Whereas disorganised (sometimes termed hebephrenia) can refer to a plethora of symptoms (see Shorter et al., 2010) including incoherent speech (Stanford et al., 2011), eccentric behaviours (Daly et al., 2012), impairments on tasks of attention, working memory and verbal fluency, alongside lack of emotional expression (Minzenberg et al., 2003).

Interestingly, in terms of sex differences, some studies report no difference in prevalence rates between the genders (Hambrecht et al., 1994; Saha et al., 2005). However, others report that males are more likely than females to be diagnosed with schizophrenia (McGarth et al., 2004; McGarth, 2005). Interestingly, whilst gender prevalence in *dimensional* schizophrenia is less researched (Ochoa et al., 2012), males have been found to experience negative symptoms of schizophrenia more severely than females and score greater on levels of negative symptoms compared to females (Shtasel et al., 1992; Cowell et a., 1996; Galderisi et al., 2012). On the other hand, females have been found to show higher prevalence of positive symptoms than males (Andia et al., 1995; Morgan et al., 2008). The last notion is supportive of the

IBT conception that it is likely the *positive* expressions of schizophrenia are linked with a feminised profile.

5.3 Support for the imprinted brain theory

Support for the notion of the ASD - schizophrenia continuum is derived from studies which have shown that, by manipulating the expression of imprinted genes to a maternal or paternal bias in the mouse brain, structures of the limbic system and the neo-cortex develop differently (Keverne et al., 1996). Badcock and Crespi (2006) offer a comprehensive review of the factors which they believe support their hypothesis of the IBT; the main observation is that schizophrenia is associated with underdevelopment in childhood in terms of a smaller cortex and brain size. Whereas in ASD, the exact opposite trend is evident; heavier in birth weight and larger cortex and brain size, supporting *diametric* observations.

The IBT hypothesises that ASD is a male dominated disorder (Badcock & Crespi, 2008). Therefore, if the IBT is accurate, then schizophrenia should theoretically be female dominant. However, it is not, in fact as referred to previously, it is male biased (Aleman et al., 2003). To address this Badcock & Crespi (2008) propose it is specifically the *positive* symptoms of schizophrenia which present as the diametric opposite of ASD and interestingly, in support of this statement, a female bias is consistently found in positive schizophrenic samples (Leung & Chue, 2000; Smit, 2010).

At a psychological level, ToM deficits are evident in both ASD and schizophrenia (de Achával et al., 2010; Fett et al., 2010; Lysaker et al., 2010; Mehl et al., 2010). However, interestingly, these ToM deficits are most

evident in *negative*, not *positive* schizophrenia (Roncone et al., 2000; Mazza et al., 2001; Langdon et al., 2002; Frith, 2004; Greig et al., 2004; Brune, 2005; Kim et al., 2011), fitting with the IBT hypothesis of the diametric expression. Studies of MN activity in psychosis have concluded that the MN system in ASD is *underdeveloped*, however, the MN system in schizophrenia has been found to be dysregulated and in some cases *overactive* compared to controls, indicating a different type (maybe, diametric) of ToM abnormality (Aleman & Kahn, 2005; Quintana et al., 2005; van Rijn et al., 2005).

Importantly, Badcock & Crespi's (2006) hypothesis predicts that the healthy schizotypy (trait level schizophrenia) will demonstrate enhanced *accurate* ToM abilities. It is when schizotypy becomes clinical that ToM will be so hyper-developed that it becomes an *inaccurate* over-interpretation of the mental states of others (e.g. paranoia). As discussed in Chapter 4, ASD is most severe in females (Roman et al., 2016), and fascinatingly, schizophrenia is most severe in males (Badcock & Crespi, 2006). Badcock & Crespi's (2006) explanation for this is because of the complex *conflict* which occurs when the direction of gene expression opposes the influences of biological sex, resulting in a severe manifestation of either disorder (a notion that requires further investigation).

Support also comes from studies which have found that schizophrenia in parents has shown to be a significant predictor of ASD in offspring (Larsson et al., 2005; Daniels et al., 2008; King & Lord, 2011), suggestive of *some type* of link between the two phenomena. Regardless of these intriguing indicators of ASD and schizophrenia as diametric opposites, the strongest

support for the IBT continuum is derived from research which suggests that, in conditions where genomic imprinting is a factor, ASD and schizophrenia tendencies are more prominent (Badcock & Crespi, 2006; Cohen et al., 2005). For instance, children with Angelman syndrome[2] demonstrate ASD traits and sometimes (in a small number of cases [NHS, 2016]) the disorder is the result of paternal bias in expressed genes on chromosome 15 (Badcock & Crespi, 2006). Angelman syndrome is implicated by alterations to the maternally expressed gene UBE3A (in 70% of cases [NHS, 2016]) which is also believed to be a risk factor for ASD (Badcock & Crespi, 2006). Similarly, support comes from Prader-Willi syndrome[3] (PWS), a disorder which is caused by maternally biased expression on chromosome 15q11-q13 (Dykens et al., 2011), interestingly, PWS has found to be a strong predictor of later development of psychosis (Verhoeven et al., 2003).

A small number of studies have investigated the claims of the IBT at a behavioural level and found contradictory results. For instance, Brosnan, et al. (2010) found that in a sample of 70 female undergraduates, higher levels of self-report schizotypy traits were associated with self-report profiles of hyper-empathising and hypo-systemising. Further, Russell-Smith et al.

[2] Angelman syndrome is a disorder based in genetics (where the gene UBE3A is absent or malfunctions – in 70% of angelman syndrome cases, the maternal gene is absent) which affects physical and intellectual abilities. Usually diagnosable around 6-18 months of life, symptoms include abnormal levels of smiling or laughing and excitability, short attention span, problems with sleep and an unusual fascination with water. Problems with ataxia are also present along with strabismus and scoliosis, and seizures can be commonplace (NHS, 2016a).

[3] Prader-Willi syndrome, a rare genetic disorder, characterised by hypotonia, hypogonadism, obesity and endocrine glad dysfunction, causes problems with social and emotional development (PWSA, 2016). In addition, a permanent feeling of hunger is present, alongside restricted growth patterns and general learning difficulties (NHS, 2014).

(2013) investigated the E-S theory in relation to ASD and positive psychosis in a student sample, using self-report and behavioural measures and found no support for the notion that ASD and psychosis are diametrically opposed in empathising and systemising. However, Russell-Smith, et al. (2010) found that people reporting higher levels of psychosis traits were slower at identifying embedded figures on the embedded figures task (EFT) (see section 6.3.2.2), supporting the IBT notion that psychosis traits are associated with the lesser ability to systemise. These are the only studies as far as the research is aware which have directly investigated the EFB and its relationship with psychosis and they are limited in their findings. For instance, Brosnan et al. (2010) only employed self-report measures of empathising and systemising, therefore it could be argued that all that was measured was a person's opinion of their ability rather than their actual ability.

5.4 Characterising the EFB with schizophrenia

To recap, the EFB profile is one which is highly attune in empathic ability, alongside a significant deficit in systemising understanding (Baron-Cohen, 2003). There has been a dearth of research pertaining to the pathological consequence of the EFB; however, the IBT *may* offer an indirect characterisation.

The E-S theory and IBT significantly differ in two main ways; firstly, the E-S theory attributes the manifestation of sex differences to the level of exposure to fT, where greater levels of fT influence masculinisation and lower levels, feminisation (Baron-Cohen, 2003). Whereas the IBT theorises that imbalance

in genetic imprinting is the driving cause of sex differences and subsequent pathology at extreme levels (Badcock & Crespi, 2006). The second differentiation between the theories is in their explanation of the result of a highly feminised profile. The IBT suggests that schizophrenic traits would be fitting with what might occur in a cognition that has been subject to an extreme level of maternal bias (Badcock & Crespi, 2006). On the other hand, the E-S theory postulates that the EFB cannot possibly exist as a pathological manifestation of any known neurodevelopmental disorder, because no known disorder is fitting with the characterisation of the EFB (Baron-Cohen, 2003). Critics of the E-S theory suggest the lack of attention to the EFB is disappointing; suggesting it is a logical, plausible brain type, and questions why the E-S continuum does not attribute the same level of neuropathological consequence to the EFB as it attributes to the EMB (Ellis, 2005). Instead, it supposes the EFB will be, 'hyper sensitive' to empathising, but showing no clinical symptomology.

Baron-Cohen's (2002) partial reasoning for suggesting a clinical condition cannot be characterised as the EFB, is that, to fall within the predictions of the entire E-S continuum, hyper-empathising would have to be accompanied by hypo-systemising or 'systemblindness'- **directly opposite to the principles of the EMB**. Baron-Cohen (2003) argues that whilst systemising ability would be non-existent for the EFB, the EFB profile would *correctly* apply ToM ability to a highly accurate degree and no disorder (that is currently known) presents this way. The IBT responds with the postulation that because empathising (or to use the IBT's terminology; 'mentalising') is of such great intuitive ability, mentalising others emotions becomes so *over-*

inferred that it becomes inaccurate (Badcock & Crespi, 2006), which *is* fitting with *positive symptoms* of schizophrenia.

5.5 Chapter summary

Significantly, this chapter has presented the argument that sex differences that establish a feminised brain profile are likely to be implicated in pathological consequence, namely schizophrenia. There are clear links between the E-S theory and the IBT. For instance, both accounts, regardless of their different aetiological presumptions, argue that the whole population can be placed on a continuum of cognitive profiles with clinically relevant ASD at one extreme. Therefore, it is reasonable to suggest an association between schizophrenia and the EFB profile.

This thesis, through a series of experimental studies, investigates this important gap in the literature, using a variety of experimental paradigms. The following five chapters present studies that have investigated this possible link. Chapter 6 is presented next; it aimed to discover if cognitive markers, other than empathising and systemising can predict a person's place on the E-S continuum. This hypothesis is based on the speculation that episodic memory ability will be reduced in higher empathisers due to the link between schizophrenia and source monitoring error.

Chapter 6

Associative memory and source monitoring in relation to empathising and systemising: a two-part study[4]

6.1 Introduction

This first experimental chapter reports a study that aimed to investigate whether or not performance on episodic memory tasks can be a significant cognitive marker on the E-S continuum (Baron-Cohen, 2003). This exploration not only aimed to widen the knowledge pertaining to the overall E-S continuum and be applicable to the broader topic of sex differences that may affect cognition, but also link the IBT hypothesis to the elusive EFB profile (Baron-Cohen, 2003), which is the primary aim of this PhD thesis.

6.1.1 Background

The E-S theory (Baron-Cohen, 2003) suggests that cognitive profiles of the general population exist on a continuum, which can be identified by empathising and systemising traits. (see Chapter 3 for detailed discussion of the E-S theory). Similarly, IBT (Badcock & Crespi, 2008) can be cautiously linked with the E-S theory in that both incorporate continuum models that are based on the theory that innate sex differences influence aptitude for mentalising and mechanical styles of thinking. The IBT posits that clinically relevant consequences occur at *both* end of the continuum in that ASD and

[4] Some of the information presented in this chapter is subject of manuscripts in preparation titled 'Episodic memory and the empathising – systemising theory' and 'Source monitoring in higher empathisers and lower systemisers' by S.L. Jones and V. Lesk.

schizophrenia are linked, existing as diametric opposites at the extremes of the continuum influenced by an extreme bias towards either paternal or maternal imprinting (see Chapter 5).

6.1.1.1 Objective of this study

This study aims to bring together the principles of the E-S theory and the IBT hypothesis by exploring cognitive markers that should be able to predict a person's place on the E-S continuum, based on the hypothesis of the IBT.

6.1.2 Memory ability, empathising and systemising

Can cognitive traits or abilities other than empathising and systemising predict a person's place on the E-S continuum? Direct investigation into the potential relationship between the E-S continuum and memory processes is novel. There are no studies, to the knowledge of the researcher, that have so far reported any results. Badcock (2009) *in-directly* suggests that research into the potential relationship would be valuable, as there is evidence to suggest that ASD and schizophrenia may demonstrate diametric strengths and weaknesses in episodic and semantic (factual) memory (Badcock, 2009). According to Badcock's (2009) theory, deficits in episodic memory exhibited in ASD and schizophrenia individuals would show a diametric relationship. In that, episodic memory in ASD would be a deficit because the individual fails to place themselves in an episodic memory due to the lack of ToM, whereas schizophrenic individuals show deficits because they may not be able to 'include anyone but themselves' due to a preoccupation with self-perception (p. 102).

It is widely acknowledged that ASD often shows a *superior ability* in semantic[5] or factual memory (O'Shea et al., 2005; Toichi et al., 2002; Williams & Minshew, 2007). This is most likely due to the superior ability ASD shows in systemising and the sometimes occurring savant skills in recalling patterned information, which may facilitate semantic memory. On the other hand, ASD tends to present with mild to severe *deficits* in episodic memory[6] (Lind & Bowler, 2009; Bright-Paul et al., 2008) which may be explained by the social context of episodic memory and the autistic difficultly with ToM and social brain skills.

The knowledge surrounding how memory is affected in schizophrenia is slightly less clear. Collectively, findings in relation to episodic memory dysfunction in schizophrenia can be contradictory (Mitchell & Johnson, 2009a; Saunders et al., 2012; Schmidt-Hansen & Honey, 2009). The collective opinion is that the majority of studies find evidence of significant memory deficits in schizophrenia (Aleman et al., 1999; Manoach et al., 2000; Achim & Lepage, 2005; Danion et al., 2007; Ragland et al., 2009), yet others fail to support this notion, finding that episodic memory remains intact in schizophrenic samples (Rushe et al., 1999).

---

[5] Semantic memory is 'declarative knowledge acquired about the world (Binder & Desai, 2011, p.527). It is the knowledge of facts that are not personal in nature, including knowledge of objects, history or factual information learned throughout our lifetime (Binder & Desai, 2011).

[6] Episodic memory is a sub-section of declarative memory, which broadly refers to the memory for events, and requires the conscious remembering of events of the past (Tulving, 1972). Episodic memory is the remembering of *context dependant* events that are encoded and recalled with specific information in relation to places, times, and people (Steinvorth et al., 2005).

6.1.3 The neural basis of memory

The hippocampus and medial temporal lobes (MTL) are the primary regions that are associated with the remembering of events and the spatial context of that remembering (Burgess et al., 2002; Eichenbaum, 2004; Ross & Slotnick, 2008; Dickenson & Eichenbaum, 2010) and the encoding and retrieval of associations (Cohen et al., 1999; Eldridge et al., 2000; Davachi & Wagner, 2002; Giovanello et al., 2003). Neuropsychology studies have revealed that damage to the MTL severely implicates episodic memory ability (Bayley et al., 2006; Rosenbaum et al., 2008), which is the memory system we are interested in here.

The role that the hippocampus plays in memory acquisition and formation is known to be lateralised, in that the parahippocampus specifically involves the processing of the spatial context (Ekstrom & Bookheimer, 2007). Whereas memory of location is implicated in the right hippocampus and verbal memory and social context such as autobiographical remembering (Burgess et al., 2002) and facial working memory (Phillips et al., 1998) is associated with the left hippocampus.

Interestingly, in relation to the evolutionary psychology context of both the E-S theory and IBT, all non-declarative and *some* declarative memory abilities are thought to be 'evolutionary primitive' in both humans and animals (Dickerson & Eichenbaum, 2010). However, neuroscientists believe that episodic memory is a recent evolutionary addition to the memory network and whether or not episodic memory is evident in animals is debatable (see Dickerson & Eichenbaum, 2010). This suggests that the evolutionary development of empathising and systemising due to a process of adaptive

brain plasticity and the recent evolution of episodic memory processes may have similar aetiologies.

6.1.4 Episodic memory and the development of theory of mind

Another important way of looking at the possible relationship between episodic memory ability and the E-S continuum might be to consider the close relationship between memory ability and ToM development. Neuroscientists tend to agree that in order to be able to appreciate another person's state of mind, we must rely (in part) on our ability to recall past personal experiences (autobiographical memory) (Tulving, 2002).

Investigations into ToM and memory have largely progressed independently of each other (Gaesser, 2013). Yet recent neuroimaging studies allow us to recognise that the areas of the brain that are implicated in ToM overlap greatly with areas that are involved in episodic memory (frontal and medial temporal systems) (Buckner & Carroll, 2007; Shamay-Tsoory, 2011; Ciaramelli et al. 2013), which, in turn, allows us to infer that the two are connected in some way.

Furthermore, in order to experience emotion, people must have adequate ToM and neuroscientists agree that there is enough evidence to suggest that episodic memory and ToM develop at the same time and are likely to be *dependently* connected (see Ciaramelli et al., 2013). In that, the connection between the two systems is likely strengthened as we develop our ToM ability by recalling past memories and applying them to the mental states of others, therefore understanding how others might feel based on our own personal experiences (Rabin & Rosenbaum, 2012). It is also recognised that

emotional memories are recalled with greater accuracy and clarity than memories without social context (Buchanan, 2007). This has been attributed to the interaction of the amygdala, hippocampus and prefrontal cortex due to the high activity of the amygdala during emotional states, which subsequently influences the enhanced encoding of memories (Buchanan, 2007). Empirical evidence has shown that the development of episodic memory ability is closely related to the development of ToM ability in healthy children (Perner et al., 2007; Bright-Paul et al., 2008). To further support the association; where ToM is impaired in ASD children, episodic memory is also impaired when compared to matched controls (Ben Shalom, 2003; O'Shea et al., 2005; Lind et al., 2009; Adler et al., 2010). Yet significantly, semantic memory is intact (Bowler et al., 2000a). O'Shea et al. (2005) propose that the reason for this observable episodic memory deficit in ASD individuals might be explained not by a general deficit in attaching context to the memory, but instead involves the inability to attach *social context* to memories due to the interdependence of ToM and episodic memory. In support of this dependence between the two cognitive processes, studies have shown that other clinical populations (namely, dementia patients), tend to show deficits in empathic ability (Cuerva et al., 2001; Gregory et al., 2002; Snowden et al., 2003; Lough et al., 2006; Zaitchik et al., 2006; Torralva et al., 2007; Fernandez-Duque et al., 2010; Laisney et al., 2013), which further confirms the apparent dependency between ToM and episodic memory systems.

This link between ToM and episodic memory processes is a major rationale for investigating this type of memory process in relation to the E-S continuum. It presumes a need to investigate episodic memory within both a

social context and a non-social context to investigate if any differences in memory ability are present dependant on this factor. Interestingly, a recent study by Wang & Fu (2011) reviewed a collection of research studies which investigated the role of emotion on memory recall in a neurotypical sample and concluded mixed results. For instance, some studies found that enhanced recall was evident when the stimulus involved an emotional context (Doerksen & Shimamura, 2001; Anderson & Shimamura, 2005; D'Argembea & Van der Linden, 2005), whereas other studies found that an emotional context *inhibited* recall (Cook et al., 2007; Maddock & Frein, 2009). Why this inconsistency is evident is unclear, however, it has been suggested that emotional stimuli may 'arouse' greater attention or that people process memories differently, according to their mood at the time (see Levine & Pizarro (2004) for a detailed debate). This informs the reasoning for employing both social and non-social conditions in the memory tasks employed in this study.

Shedding some light on this discrepancy, Rosenbaum et al. (2007) investigated the dependency between episodic memory and ToM by comparing healthy controls to patients with traumatic brain injury who had lost the ability to recall personal memories on false belief tasks. Interestingly, results showed that there was no difference in ToM ability between the two groups, which led Rosenbaum et al. (2007) to conclude that it is not necessary to have the ability to remember past events in order to have a fully working ToM. This suggests memory and ToM are *not* as dependent on each other as previously thought. Although Rosenbaum et al. (2007) makes clear that this study does not provide any information on the necessity of

autonoetic consciousness for the development of ToM, instead, only suggests that a severe lack of *current* episodic memory ability does not affect ToM performance. In addition, studies with amnesic patients suggest that the two can sufficiently work independent of each other, in that amnesic patients often have sufficiently working ToM despite a severe memory deficit (Rabin & Rosenbaum, 2012). A possible explanation for this is that ToM type thinking can be executed through semantic knowledge of how a person may feel in a specific situation, so people may demonstrate ToM, but not from instinctual knowledge of another person's state of mind, but instead through knowledge of previously learned social etiquette (see Rabin & Rosenbaum, 2012 for further discussion).

6.1.5 Source and associative memory

Source memory (SM) (or sometimes termed source monitoring) and associative memory (AM) are concepts under the wider umbrella of episodic memory (Davidson et al., 2008) and are the focus of this study. SM is the recollection of the contextual knowledge of how we came to know or experience something (Meiser & Bröder, 2002). AM on the other hand, refers to the encoding of associations between obscure stimuli, people, actions, places or events (Eichenbaum, 2004). AM is closely related to SM but can be modulated by one particular distinction; AM involves processing an association between two items (inter-item associations), which is different from SM, in that SM involves processing item/s *within* a social or spatial context (item-context associations) (Park et al., 2012).

AM is most often tested in the lab using pairs of items which are presented together, AM ability is assessed by the number of correct pairs of items recalled after an interval of time using free and/or cued recall (Guez et al., 2013; Cohn & Moscovitch, 2007). SM on the other hand, is most often measured by source monitoring error (SME) experiments; where participants are presented with items within a particular context (e.g., a list of words visually presented on differing backgrounds, or audio of different people speaking) and are measured by the ability to correctly recall the source of the memory (e.g., "which word was associated with a red background?" or, "who spoke the following sentence...?"), using free and cued recall after a period of time (Davidson et al. 2008). Interestingly, studies have found that in some cases, using words which elicit emotions assist the associative binding process, in that these words are recalled at a higher rate than neutral words (Murray & Kensinger, 2012). In some cases, emotional context can hinder the associative binding process in that the rate of recall *falls* compared to neural words (Mackey & Ahmetzanov, 2005; Easterbrook, 1959).

6.1.6 Considerations

Here, it is important to consider the areas of contention that are apparent in relation to the theorising surrounding the E-S and IBT continuums and possible diametric differences in memory ability. Especially due to the novelty of the investigation. As previously mentioned, studies investigating memory deficits in terms of schizophrenia are often contradictory and unclear, which means there is no readily accepted doctrine as to how it is implicated. Yet,

there are some findings that would theoretically contradict what the IBT continuum would predict in relation to the patterns of episodic memory.

For instance, schizophrenia participants have often shown episodic memory deficits similar to those found in ASD (Doré et al., 2007; Mitchell & Johnson, 2009). This is intriguing, as if we are to recognise that episodic memory is related to ToM ability (Naito, 2003; Bright-Paul et al., 2008), we might presume that episodic memory may be an *overlapping* trait in ASD and schizophrenia rather than a diametric aspect of cognition, as the IBT would predict (Badcock & Crespi, 2006). However, we might also consider Badcock & Crespi's (2008) postulation that ToM in ASD presents as a deficit due to an *underdeveloped* mirror neuron system (MNS) whereas ToM in schizophrenia presents as a deficit not because of an absence of the MNS, but an *overactive* or deregulated MNS which causes ToM to be so sensitive it becomes dysfunctional.

6.2 Aims of this study

It was the aim of this study to explore episodic memory ability in relation to the E-S continuum in healthy population participants.

The rationale for employing neurotypical participants is twofold. Firstly, despite the E-S and IBT models incorporating clinical populations, empathising and systemising individual differences should be sensitive enough to be measurable in healthy population samples. Secondly, if we are to incorporate the elements of the IBT into the E-S continuum (the ASD - schizophrenia continuum), the manifestation of ASD and schizophrenia exist at trait level, where neurotypical populations can demonstrate measurable

indicators of both conditions, as per the principles of the continuum model (Baron-Cohen, 2003; Mason & Claridge, 2005). This allows researchers to investigate the continuum in healthy populations (i) without the possible interference of medication therapy effects that may be present in clinical populations, and have the potential to significantly interrupt the cognitive processes that we are aiming to measure and (ii) issues pertaining to diversity of diagnosis may be a confounding variable (e.g., the effects nonspecific psychopathology [see Cash, 1973] or ethical considerations concerning decision making capacities [Wilson & Stanley, 2006]).

The overall rationale for this two-part study was to contribute to the dearth in knowledge surrounding specific cognitive markers that can predict a person's position on the E-S continuum and subsequently advance the knowledge of the link between memory ability and the feminised profile. The hypothesis in relation to memory capacity was derived from the IBT which postulates that advanced episodic memory ability would be observable in higher empathisers.

6.3 Study 1

6.3.1 Overview

The overall purpose of study one was to investigate the hypothesised relationship between episodic memory ability and the E-S continuum.

It was hypothesised here, based on theorising by the IBT (Chapter 5) that those participants who presented with a greater capacity in empathising and

lower aptitude for systemising would show greater episodic memory ability (measured by SM and AM), both within a social and non-social context.

6.3.2 Methods

6.3.2.1 Participants

A total of 43 student participants from the University of Bradford were recruited to take part in this study. The sample consisted of 31 females (72%) and 12 males (28%). No age data is available; however, all participants were over the age of 18 years old. Inclusion criteria specified that participants must have (i) no clinical diagnosis of mental illness (ii) normal or corrected to normal vision (iii) a fluent understanding of English language (iv) no dyslexia (v) not taken part in previous studies conducted by the researcher due to over exposure effects. Ethical approval was obtained from the Humanities, Social & Health Science Research Ethics committee at the University of Bradford. All participants gave their informed written consent.

6.3.2.2 Materials

The Empathising Quotient (EQ) - The short form version of the EQ (EQ-S) (Wakabayshi et al., 2006b), measuring self-report cognitive and affective empathy was administered. The EQ scale has previously shown validity and reliability (Lawrence et al. 2004). Consisting of 22 statements, an example statement from the EQ-S would be; *'I can easily tell if someone else wants to enter a conversation'* or *'I can pick up quickly if someone says one thing but means another'*. Participants respond to the statement using a forced choice scale of (i) strongly agree, (ii) slightly agree, (iii) slightly disagree or (iv)

strongly disagree. Participants receive a score between 0 and 44. Higher scores indicate higher levels of empathising. Full inventory can be found in appendix 1.

The Systemising Quotient (SQ) **-** The short form version of the SQ was employed (SQ-S), which measures self-reported 'everyday' systemising ability (Wakabayshi et al., 2006b). Consisting of 25 statements, an example statement from the SQ-S would be *'In math, I am intrigued by the rules and patterns governing numbers'* or *'I find it easy to grasp exactly how odds work in betting'*. Participants respond using a forced choice scale of (i) strongly agree, (ii) slightly agree, (iii) slightly disagree or (iv) strongly disagree. Participants receive a score between 0 and 50. Higher scores indicate higher levels of systemising. Full inventory can be found in appendix 2.

The Cambridge mind-reading face-voice battery (CAM-MR) **-** The CAM-MR is a test of understanding and recognition of subtle complex emotions through visual and auditory channels. The CAM-MR was developed to provide a test of complex emotion recognition, which aims to be closer ecologically to real life as it uses moving pictures and voice clips rather than still pictures. The task comprises of 20 emotions which are expressed through video clips and voice clips of one actor, acting out a particular emotion (see Figure 4). There are 50 trials for the video clips each lasting three to five seconds and 50 trials for voice clips, each lasting 3-5 seconds. The participant is presented with 4 words after each trial. The aim is to choose the adjective which best describes the emotional context of the trial. ***Note-*** only the visual (video clips) condition of the CAM-MR was employed in this study.

Fig. 4: Example of the CAM-MR task (image taken from video clip)

The mental rotation task (MROT) **-** An adapted computerised version of the Vanderberg and Kuse (1978) mental rotation task (MROT) was administered. The task comprises of 20 trials, each consisting of a target three dimensional image made up of 10 three-dimensional cubes and four comparison images (see Figure 5). Two of the comparison images are the target image rotated to different degrees of clockwise rotation, the remaining two images are different images. The task aims to measure visual-spatial ability. It requires participants to mentally retain the image whilst rotating it in space in order to match it to the comparison images. One point is given per trial if both of the correct images are selected. No points are given per trial if only one correct image is selected.

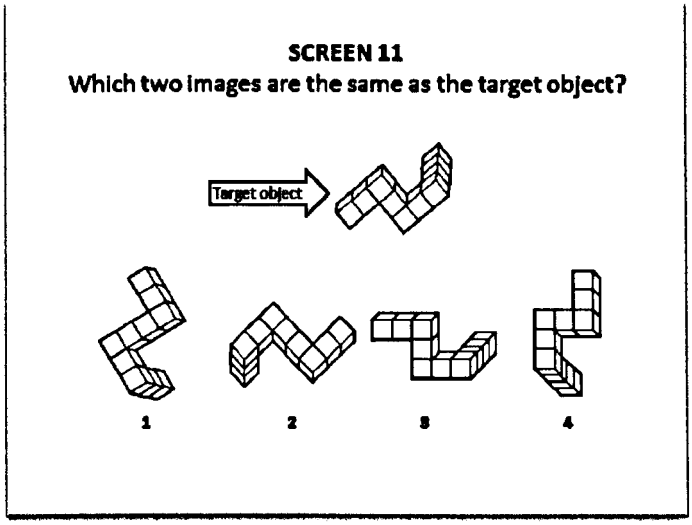


Fig. 5: A trial on the MROT, adapted from Vanderberg & Kuse (1978). Correct answers are images 3 and 4.

The embedded figures task (EFT) **-** A pencil and paper variant of the EFT was administered. Each trial has a target image and two other images, one of which contained the embedded figure. The EFT measures attention to detail with a focus on global versus local processing ability (Auyeung et al., 2011). The aim of the task is to locate the embedded figure within one of the two images and outline it with the pen provided. The target figure is only present in one of the two images. Scores are based on correct answers within the five-minute limit time frame.

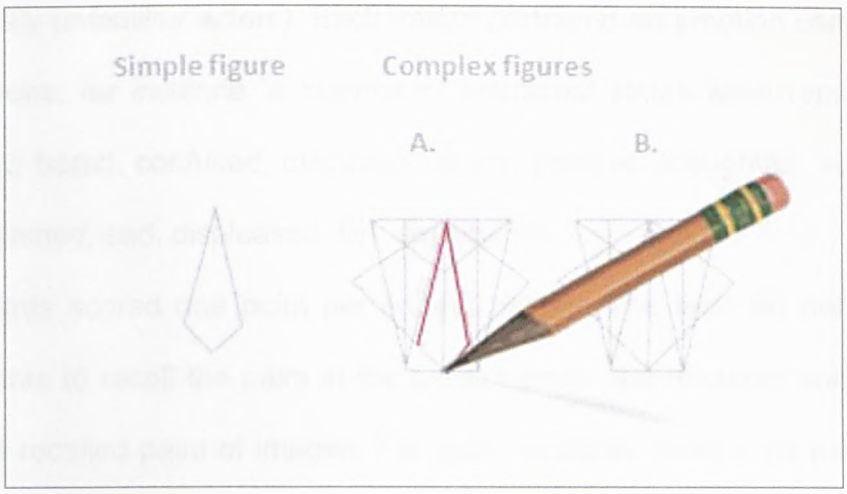


Fig. 6: Example of the EFT.

Pairing task – the pairing task aimed to measure AM. The task comprised of two conditions (i) inanimate (abstract shapes) (Figure 7, a.) and (ii) social (facial expressions) (Figure 7, b.) which were measured separately. In each condition, participants were presented with six pairs of images, which were not connected to each other either semantically, auditory or visually, therefore were randomly paired, presented on a 15.6" computer screen. Each pairing was shown for five seconds and was then followed by a fixation cross. Participants were then presented with exact replica 'cut outs' of the 12 individual images that made up the six pairs, placed randomly in front of them on the desk. Each cut out image was matched for exact size and colour quality as was seen on the computer screen. Participants were then asked to match the images into their correct pairings as recalled from the computer presentation sequence.

For the inanimate condition, images consisted of both two-dimensional and three-dimensional solid colour shapes. The social condition consisted of facial expressions demonstrated by one male and one female actor

(previously unfamiliar actors). Each image portrayed an emotion using facial expressions, for instance, a number of emotional states were represented including; bored, confused, disgusted, angry, passive, thoughtful, surprised, happy, pained, sad, displeased, flirt, shy and silly.

Participants scored one point per correct pairing. The task did not require participants to recall the pairs in the correct order; the measure was simply correctly recalled pairs of images. For each condition, three x six pairs were employed. Overall scores were combined for each condition, therefore the maximum score for each condition was 18 points. Participants completed a practice trial for both the inanimate and social conditions before the commencement of the task.

a. inanimate condition

b. social condition

Fig. 7: Examples of trials on the pairing task.

Word recall – The word recall task aimed to measure SM. The task required participants to observe a series of words (ten words per trial x two), with each word presented on a computer screen for five seconds (see Figure 8) followed by a fixation cross, followed by the next word. Words were randomly placed in either one of four boxes on the screen (either top left, top right, bottom left or bottom right), and were presented randomly in either red or blue font (first trial) and green or orange font (second trial). Participants observed the first ten words (table, fruit, music, train, monkey, ball, paper, glass, coat, pencil). The researcher then stated each word (one at a time) in a random order and asked the participant to recall the location (by pointing to the section of the grid on the computer screen) and verbalise the colour of that particular word. Participants scored one point per correct response (two points for both correct location and colour recall); therefore, the maximum score *per trial* was 20 points. The same procedure was carried out for the second ten words (font, smell, train, fruit, paint, swim, tree, sky, goose, bus). Scores were combined, determining a possible maximum score of 40 points.

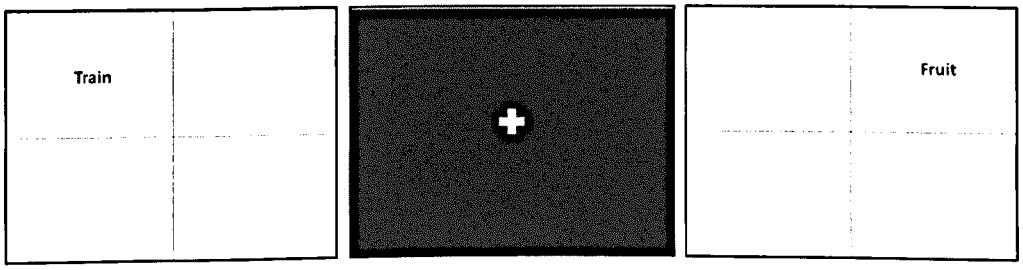


Fig. 8: Examples of trials on the word recall task.

6.3.2.3 Design

A cross-sectional, primarily correlational design was employed, concerned with exploring the relationships between empathising, systemising and episodic memory ability using direct performance and self-report measures. This study employed standardised neuropsychological tasks developed for non-clinical populations using standardised scoring. Familiarity with the theoretical bases of these tasks allowed for interpretation of cognitive styles and abilities. The researcher administered the battery of tests in random order for the purpose of controlling for order effects (except for the self-report measures which were completed online prior to the lab session).

6.3.2.4 Procedure

Participants were recruited via email advertisement to all faculties at the University of Bradford. All participants completed the experiment in two sessions. The first session was online and included the self-report measures (EQ-S and SQ-S) and the second session was carried out at the psychology labs in the Division of Psychology, University of Bradford where the battery of direct performance measures was administered. On receipt of interest in taking part in the study, participants were sent more information (via email) about the study and were required to read the online information sheet pertaining to the study and if happy to proceed, provide written consent (electronic signature was accepted). Sessions in the lab lasted approximately 40 minutes and no participants requested a break during the sessions.

6.4 Results

The aim of this study was to investigate if episodic memory performance could significantly predict empathising and systemising ability, thus allowing us to explore whether episodic memory performance can accurately predict a person's place on the E-S continuum. A non-clinical population was administered both self-report (EQ-S and SQ-S) and direct performance measures (the embedded figures task, the mental rotation task, the Cambridge mindreading task [proxies for measuring empathising and systemising performance], the social and inanimate pairing task [associative memory] and the word recall task [source memory]).

Means and standard deviations for all variables can be found in table 1.

Table 1: Means and standard deviations for scores on all variables

Variable	*n*	M	±SD	Min-Max possible score
EQ-S	40	28.38	8.70	0-44
SQ-S	40	17.80	7.45	0-40
EFT	40	4.18	0.78	0-05
MROT	43	30.00	6.13	0-40
CAM-MR	42	40.19	4.82	0-50
PT social	43	10.81	4.18	0-18
PT shapes	43	11.51	4.44	0-18
WR	42	30.48	6.99	0-40

Note: EQ-S= empathising quotient (short form); SQ-S= systemising quotient (short form); EFT= embedded figures task; MROT= mental rotation task; CAM-MR= Cambridge mindreading face-voice battery; PT social= pairing task social condition; PT shapes= pairing task shapes condition; WR= word recall

6.4.1 Main effects of gender

The relationship between empathising, systemising and biological sex is an important factor in this research area (Baron-Cohen, 2002), therefore it was important to discover the effect (if any) gender had on the measures administered in this study. As we would expect, MANOVA statistic (where the dependent variables were test scores) revealed there was a significant main effect of gender on EQ-S score (F (1, 30) = 11.74, p<0.01), with independent samples t-test revealing that males scored significantly lower than females on the EQ-S (t (38) = -2.50, p<0.05). However, interestingly, gender had no significant effect on any other variable, suggesting, as Baron-Cohen (2003)

posits, whilst gender can be a predictor of empathising and systemising performance, it is not necessarily significantly important.

6.4.2 Regression analysis

In all regression analysis, scatterplots showed an even disperse of data points around zero, demonstrating the assumptions of linearity and homoscedasticity were met. In addition, no issues of multicollinearity were present. It assumed that findings could therefore be generalised to the population.

6.4.3 Source memory (word recall) and empathising

A linear regression was calculated to see if EQ-S score could be predicted based on word recall performance. The regression model (word recall) significantly predicted EQ-S score (F (1, 37) = 5.98, p<0.02). There was a small (R =.37) relationship between word recall score and EQ-S and the model could explain 14% of the variance (R^2 = .14, Adjusted R^2 =.12) (β = .37, t (38) = 2.45, p<0.02: 95% *CI* .078 - .833). Participants' predicted EQ-S score was equal to 14.49 + .46 (word recall) when word recall is measured in 1 point per correct response. EQ-S score increased .46 for each correct response on the word recall task, meaning that, as SM score increases, so does EQ-S score.

Table 2: The unstandardized and standardised regression coefficients for the predictor variable word recall

	B	SE B	B
Constant	14.49	5.82	
Word recall	.46	.19	.37*

*p<0.05, **p<0.01, R^2 .14

A linear regression was also calculated to predict CAM-MR score based on word recall score. The regression model (word recall) significantly predicted CAM-MR score (F (1, 40) = 10.41, p<0.01), the relationship between word recall and CAM-MR was moderate (R =.45) and the model could explain 21% of the variance (R^2 = .21, Adjusted R^2 =.19) (β = .45, t (41) = 3.23, p<0.01: 95% *CI* .126 - .548). Participants' predicted CAM-MR score is equal to 29.73 + .34 (word recall) when word recall is measured in 1 point per correct response. CAM-MR score increased .34 for each correct response on the word recall task. Meaning that, as SM ability increased, so too did CAM-MR score.

Table 3: The unstandardized and standardised regression coefficients for the predictor variable word recall

	B	SE B	B
Constant	29.73	3.26	
Word recall	.34	.10	.20**

*p<0.05, **p<0.01, R^2 .21

These results confirm that as predicted, **source memory score was a significant predictor of empathising**, both self-report and actual performance. Suggesting that as empathising score raised, so did source memory capacity.

6.4.4 Source memory (word recall) and systemising

Linear regression analysis was calculated to predict SQ-S, EFT and MROT score based on source memory score. The regression models were non-significant (*ns*).

6.4.5 Associative memory (pairing task) and empathising

A linear multiple regression analysis was calculated to predict EQ-S score based on the two AM conditions (PT social and PT shapes). The regression model (F (2, 37) = 5.45, p<0.01), determined that PT shapes was *not* a predictor of EQ-S score (*ns*). However, the relationship between EQ-S and the PT social was moderate (R =.47). The model could explain a moderate 23% of the variance (R^2 = .23, Adjusted R^2 =.19) (β = .49, t (39) = 3.23, p<0.01: 95% *CI* .377 - .1.64). Participants' predicted EQ-S score was equal to 1.010 + .313 (PT social) when performance was measured in 1 point per correct response. EQ-S score increased .31 for each correct response on the PT social task. Meaning that more accurate performance on the social condition of the pairing task was associated with greater EQ-S score.

Table 4: The unstandardized and standardised regression coefficients for the predictor variables PT social and PT shapes

	B	SE B	B
Constant	18.68	4.22	
PT social	1.01	.31	.49**
PT shapes	-.12	.31	-.06

*p<0.05, **p<0.01, R^2 .23

Further, a linear regression line was calculated to predict CAM-MR score based on performance on the pairing task. The regression model (PT social) but not PT shapes (*ns*) significantly predicted CAM-MR score (F (1, 41) = 5.66, p<0.05), the relationship between PT social and CAM-MR was small (R =.35) and the model could explain 12% of the variance (R^2 = .12, Adjusted R^2 =.10) (β = .35, t (42) = 2.38, p<0.05: 95% *CI* .065 - .798). Participants' predicted CAM-MR score was equal to 35.22 + .43 (PT social) when PT social score was measured in 1 point per correct response. CAM-MR score increased .43 for each correct response on the PT social task. Meaning that as performance on the social condition of the pairing task increased, so too did CAM-MR score.

Table 5: The unstandardized and standardised regression coefficients for the predictor variable AM social

	B	SE B	B
Constant	35.22	2.10	
AM social	.43	.18	.35*

*p<0.05, **p<0.01, R^2 .12

These results suggest that as predicted, **those who scored greater on empathising (both self-report and direct measures) also demonstrated greater associative memory performance** for the social condition of the pairing task.

6.4.6 Associative memory (pairing task) and systemising

A linear regression was calculated to predict SQ-S score based on pairing task score. The regression model (PT social) (but not PT shapes [*ns*]) significantly predicted SQ-S score (F (1, 38) = 4.65, p<0.05), the negative relationship between PT social and SQ-S was small (R =.33) and the model could explain 11% of the variance (R^2 = .11, Adjusted R^2 =.09) (β = -.33, t (39) = -2.16, p<0.05: 95% *CI* -1.125 - -.035). Participants' predicted SQ-S score was equal to 24.14 + -.60 (PT social) when PT social score was measured in 1 point per correct response. SQ-S score decreases .60 for each correct response on the PT social task. Meaning that as SQ-S score increased, ability on the social condition of the pairing task decreased.

Table 6: The unstandardized and standardised regression coefficients for the predictor variable PT social

	B	SE B	B
Constant	24.14	3.15	
PT social	-.60	.27	-.33*

*p<0.05, **p<0.01, R^2 .11

A linear regression was calculated to predict EFT score based on pairing task score. The regression model (PT shapes) revealed that PT shapes (but not PT social [*ns*]) significantly predicted EFT score (F (2, 37) = 7.009, p<0.01). There was a moderate (R =.52) relationship between EFT score and PT shapes score and the model could explain 28% of the variance (R^2 = .28, Adjusted R^2 =.24) (β = .51, t (38) = 3.510, p<0.01: 95% *CI* .038 - .143). Participants predicted EFT score was equal to 3.057 + .091 (PT shapes) when PT shapes is measured in 1 point per correct response. EFT score increased .091 for each correct pairing on the PT shapes task. Meaning that as EFT score increased, so too did memory performance on the inanimate condition of the pairing task.

Table 7: The unstandardized and standardised regression coefficients for the predictor variable AM shapes

	B	SE B	B
Constant	3.057	.378	
EFT	.091	.026	.37**

*p<0.05, **p<0.01, R^2 .24

A linear regression was calculated to predict MROT score based on the pairing task. The regression model (PT shapes) found that PT shapes, but not PT social (*ns*) significantly predicted MROT score (F (2, 40) = 5.062, p<0.01). There was a moderate (R =.45) relationship between PT shapes score and MROT and the model could explain 20% of the variance (R^2 = .20, Adjusted R^2 =.16) (β = .45, t (41) = 3.013, p<0.01: 95% *CI* .204 – 1.036)[7]. Participants' predicted MROT score was equal to 27.42 + .62 (PT shapes) when MROT is measured in 1 point per correct response. MROT score increased .62 for each correct pairing on the PT shapes task. This means, as we saw for the EFT results, similarly, as scores increased on the MROT, so too did scores on the inanimate condition of the pairing task.

[7] Interestingly, the regression model revealed that there was a strong trend (but not significant at the 0.05 alpha level) for a negative relationship between MROT score and AM social score (r = -.14) (β = -.287, t (41) = -1.929, p = **0.06**: 95% *CI* -.863 – .020).

Table 8: The unstandardized and standardised regression coefficients for the predictor variable MROT

	B	SE B	B
Constant	27.42	2.89	
MROT	.62	.21	.45**

*p<0.05, **p<0.01, R^2 .20

These results suggest that, **those who scored greater on systemising (self-report) also demonstrated lesser ability in social associative memory performance,** and are **supportive of the trend expected if associative memory can be a significant cognitive marker on the E-S continuum.**

Interestingly, results also highlighted that systemising (direct measures) score can be predicted by associative memory that is spatial in nature, which suggests that memory performance is improved for those scoring greater on systemising when the context of the memory is spatial.

6.5 Main findings - study one

Results from study one supported the hypothesis that elements of episodic memory could be a significant cognitive marker on the E-S continuum.

The rationale for this hypothesis was informed by the tentative link between the E-S theory and the ASD-schizophrenia continuum, and the overall focus of this PhD thesis to investigate further the accurate characterisation of the elusive EFB profile.

Schizophrenic individuals often present with hallucinations and delusional episodes, which are thought to be due to overly active ToM systems (Badcock & Crespi, 2006). In relation to the E-S continuum and the theoretical EFB profile, this suggests that (based on the apparent link between ToM and episodic memory development [see section 6.1.4]), we would expect that those who show higher levels of empathising (located towards the type E end of the continuum) would also demonstrate greater episodic memory ability. Put together within the principles of the E-S theory, we would also expect to see systemising show the opposite pattern of results, with greater systemisers showing lesser ability in episodic memory.

This study is the first of the author's knowledge to investigate this postulation, and therefore the findings here are novel. **This study provides support for the notion that elements of episodic memory capacity can be a significant cognitive marker on the E-S continuum**. Strongest support came from the finding that AM aptitude was able to significantly predict greater empathising and lesser systemising. This leads us to conclude that

AM ability has the potential to be a linked with the telling cognitive markers of the E-S continuum, specifically, AM that is *social* in nature, most likely due to its close relationship with ToM processes (Perner, et al., 2007; Bright-Paul et al., 2008).

Further to this, partial support was found for a link between the E-S continuum and SM capacity. As hypothesised, greater empathising ability was found to be significantly associated with greater ability in SM in this study, as measured by the word recall task. *However*, remembering that we are only interested here in the whole E-S continuum, to suggest that SM may be a cognitive marker on the E-S continuum, we would require a relationship between SM and systemising to be apparent; a trend that was not evident in this study. This is a significant finding, as it highlights the potential complexity of the relationship between the E-S continuum and episodic memory ability, in that these results demonstrate that only particular elements of episodic memory, namely *AM* are able to significantly predict a person's placement on the E-S theory continuum.

It is suggested here that further investigation into the relationship between empathising, systemising and AM is warranted, to (i) confirm these results and (ii) to investigate the association using different stimuli.

6.6 Study 2

6.6.1 Rationale

The purpose of study two was to explore AM (and also features of SM) that consisted of an interactive element involving human communication. The stimuli employed here was novel; participants were exposed to real life people, looking and speaking directly to them (through the computer screen). The purpose of this was to ascertain if the results found in study one (that AM was a significant predictor of empathising and systemising ability) was also evident during SM and AM ability which involved real life people, and was therefore *closer* to real life SM and AM instances than the previously employed stimuli.

Study two also sought to proceed further with the investigation between the E-S continuum and memory capacity by taking a closer look at SME (source monitoring error) (or sometimes termed 'false memory'). The motivation for this enquiry centred again around the link between the E-S continuum and the IBT continuum (Brosnan et al., 2010; Jones & Lesk, 2013) as discussed in section 6.2.2 in that individuals with schizophrenia have a tendency to exhibit SMEs (Lenzenweger & Gold, 2000; Brébion et al., 2002; Brébion et al., 2013; González-Ortega et al., 2013). Therefore, if the IBT can be accurately linked to the E-S continuum, can false memory also be an indicator of a person's place on the E-S continuum?

6.6.2 Background - source monitoring error

Our memories very rarely provide us with an *exact* account of what we have previously experienced, and it is usually only when an experience is 'highly novel' that our memories encoded and recalled with great accuracy (Lo et al., 2016). Often, memories can be encoded that are confused with prior similar episodes, or our imagination might 'fill in' gaps to create memories, which, as a consequence create false memory retrieval, where our *believed* recollection of events is inaccurate (Johnson & Raye, 1998). The neurological basis of false memory has been informed by patients with damage to the basal forebrain, ventromedial and orbitomedial frontal structures (bilaterally), who very often report high levels of SME and memory confabulation[8], confirming the involvement of these networks in false memory encoding and recall (Johnson & Raye, 1998; Karanian & Slotnick, 2014).

In terms of the E-S theory, there is no available data on the relationship between SME and empathising - systemising processes. However, considering the effect of gender on both empathising and systemising (that biological sex is a predictor, but not a determinant of empathising and systemising preferences [Baron-Cohen, 2003]) gender differences in source monitoring should be considered in the rationale for this research question.

Interestingly, whilst some have reported that women show significantly more associative memory illusions using the Deese/Roediger-McDermott paradigm

[8] Memory confabulation refers to the total belief in what one is saying, or knows to be true, despite the fact that such knowledge is based upon false memories (Kopelman et al., 1995).

(D/R-M)[9] (Stadler et al., 1999) in false memory recall on negative valenced word lists (Dewhurst et al., 2012), gender differences in false memory are not often evident (Seamon et al., 2002; Bauste and Ferraro, 2004; Kreiner et al., 2004; Smeets et al., 2006), which leads to the suggestion that gender is not a particularly significant factor in SME.

Also, in the context of the IBT continuum, false memory is a particularly curious notion, as schizophrenic patients, most specifically those with hallucinations and delusional symptoms (symptoms which involve a difficulty in separating lived experiences from phenomena they have experienced mentally [Keefe et al. 2002]) have been found to experience greater difficulty with SM when compared to controls and other clinical populations (Lenzenweger & Gold, 2000; Brébion et al., 2002; Brébion et al., 2013; González-Ortega et al., 2013). Furthermore, the areas of the brain which are known to be implicated in hallucinations and delusions in schizophrenia patients have also been shown to be active in SM recall in healthy brains (Simons et al., 2006), which is suggestive of a significant link between the two phenomena. Intriguingly, Bentall et al. (1991) puts forward that SME may actually be the *underlying* cause of the positive symptoms of schizophrenia rather than a consequence of the disorder. One particularly interesting study conducted by Brébion et al. (2002) found that SMEs were associated with

[9] The D/R-M paradigm is a test commonly used to induce false memory in humans. The test consists of exposure to a list of semantically related words (e.g., spoon, fork, plate, salt, table, napkin, cup, glass etc.), and on prompting, the participant is required to recall the word list. Participants often recall words that were not presented but are semantically related (e.g., knife, dish, pepper etc.), or that are related to a 'lure' word that is verbally presented to them during the recall and recognition part of the task but did not appear on the list (in this example the lure word could be 'dinner'), thus resulting in the inducing of false memories (Cann et al., 2011).

positive symptoms of schizophrenia (hallucinations, delusional and paranoid ideation) yet *negatively* associated with the negative symptoms such as flattened affect and introversion, which are often present in ASD and therefore a pertinent indication that false memory may be a significant marker on the E-S / IBT continuums.

It has been theorised that delusions in schizophrenia are formed because of the susceptibility to the encoding and retrieval of false memories, thus the source monitoring failure results in hallucinations and delusion (Jeannerod & Pacherie, 2004; Laws & Bhatt, 2005; Moritz & Woodward, 2006: Hodgetts et al., 2015). Indeed, greater false memory errors have been widely reported in schizophrenic patients and schizotypy prone samples compared to controls (Stirling et al., 1997; Vinogradov et al., 1997; Brebion et al., 1999; Bhatt et al., 2010; Saunders et al., 2012). However, we must also consider that other studies have reported that schizophrenia patients (and schizotypy prone samples) do not show greater levels of false memory errors when compared to control groups (Elvevag et al., 2004; Moritz et al., 2004: Peters et al., 2004; Dagnall & Parker, 2009). In terms of these contradictory findings, it is unclear as to whether or not confounding variables such as pharmacological interventions might have an effect (Mitropoulou et al., 2005). This is an important point to consider, as there is evidence to suggest that the relationship between SM and executive function may be modulated by neurotransmitters such as dopamine and acetylcholine (Johnson and Raye, 1998). The antipsychotic medications often prescribed in schizophrenia conditions are therefore likely to affect episodic memory processes, therefore

this may be a factor in explaining these inconsistencies (Johnson and Raye, 1998).

Considering the opposite end of the IBT and E-S continuums (the type S end of the continuum [Baron-Cohen, 2003; Badcock & Crespi, 2008]) and consistent with what we would expect in terms of the diametric trend, individuals with ASD have shown *better* ability in discriminating between false and true memories compared to controls using word lists tasks (Beversdorf et al., 2000). Although, again, conflictingly, this trend is not *always* followed as ASD individuals have also demonstrated no evidence of an advantage in discriminating between false and true memories when compared to matched controls using the D/R-M paradigm (Bowler et al., 2000b; Hillier et al., 2007) and other source monitoring tasks (Farrant et al., 1998). However, ASD individuals have been found to produce less false memory in visual paradigms that involved geometric figures (Hillier et al., 2007). The inconsistency in these findings are suggested to be due to differences in IQ and language abilities in the ASD samples employed in different studies (Hillier et al., 2007). However, it is suggested here that a much simpler explanation is possible. Is it simply the case that any remembering that involves a *systemising process* in the encoding and recalling of information (especially stimulus that involves patterned information) provide ASD individuals with an advantage on particular memory tasks? Of course, this is just conjecture, however, by investigating SME in relation to social stimuli in relation to empathising and systemising, may allow us to further explore

possible cognitive markers on the E-S continuum and infer possible explanations for inconsistency in findings.

It was hypothesised that the results of study one (that AM that is social in context is able to predict a person's place on the E-S continuum) would be replicated *and* extended to include AM that included an 'interactive' social component. It was also hypothesised that due to the (debateable) indication that false memory is reduced in ASD individuals and increased in schizophrenia individuals, *and* based on the understanding that the E-S theory can be linked to the predictions of the IBT in terms of the ASD – schizophrenia model; that SMEs would be associated with greater levels of empathising, alongside lower levels of systemising.

6.6.3 Methods

6.6.3.1 Participants

A total of 56 participants took part. All participants were over 18 years of age with a mean age of 26.67 years (±SD= 8.24, Range= 32). Participants were recruited via several channels, including by email invitation to all faculties at the University of Bradford, posters, mailing lists and social media associated with the psychology department at University of Bradford. Exclusion criteria specified participants must have (i) no clinical diagnosis of mental illness or neurological disorder (ii) no uncorrected visual impairment (iii) and a fluent understanding of English language (iv) not participated in part one of this study. Ethical approval was obtained from the Humanities, Social & Health Science Research Ethics committee at the University of Bradford. All participants gave their informed written consent.

6.6.3.2 Materials

The Embedded Figures Task (EFT) **-** A pencil and paper variant of the EFT was administered as per section 6.3.2.2.

Reading the mind in the eyes (RTME) **-** This task comprised of 36 items which aimed to measure advanced ToM or 'mentalising' ability by employing black and white photographs of the eye regions (from the bridge of the nose to middle forehead) of males and females (see Figure 9), who were displaying subtle facial expressions. Participants were required to consider the photograph, infer the mental state of the person in the photograph and choose one of four adjectives (surrounding the image) that they thought best represented the emotion displayed. Participants had access to a glossary to which they could freely refer, in order to confirm the correct definition of any words they were not familiar with. One point was scored for each correct interpretation of the emotional state; therefore, maximum score was 36 points (Baron-Cohen et al., 2001)

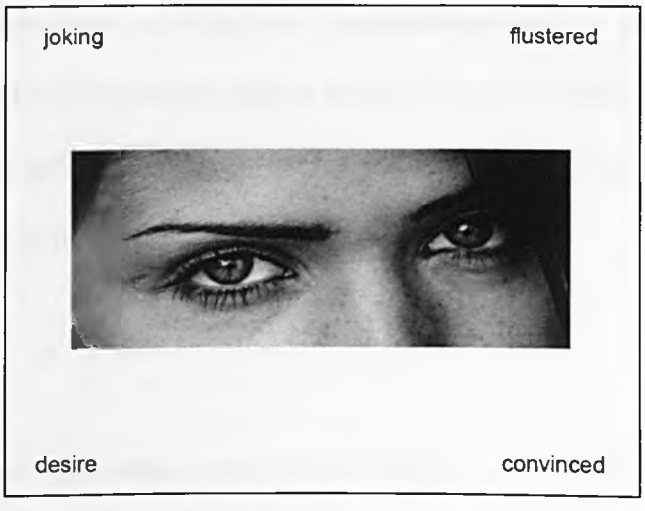


Fig. 9: Examples trials on the RTME task. The correct response is 'desire'.

'Say what?' **–** An assessment of source and associative memory, with the ability to pick up on source monitoring errors developed by the researcher was piloted in this study. Participants were presented with ten video clips, with a fixation cross displayed between each clip. Each trial involved an actor (different each time) verbalising one sentence (an example sentence would be; "*It would take 63 hours to walk from Leeds to Edinburgh*"). The chosen lines of text were factual random statements (see appendix 3 for complete list of questions and example images). The actors in the clips looked directly into the camera and verbalised the statement, lasting approximately three seconds. After the presentation, associative memory ability was measured by presenting the participant with 15 lines of text and a screen shot of each actor that appeared in the clips. The list included five lines of text that were not included in the test paradigm. This aspect aimed to measure source monitoring errors by false-recognition (the mistaken claim that novel stimuli has previously been encountered [Garoff-Eaton et al., 2006]). The participant was instructed to pair the line of text to the actor they believed to have verbalised the sentence, scoring one 'associative memory' point per correct pairing. Participants scored one 'false memory' point for each false memory e.g. pointing out **with conviction** that a particular actor voiced a statement that did not appear in the video clips (see appendix 3).

6.6.3.3 Design

A cross-sectional, primarily correlational design was employed; concerned with exploring the relationships between empathising, systemising and source monitoring using direct performance measures. The rationale for

measuring empathising and systemising ability by direct measures is derived from issues pertaining to the apparent discrepancies that can occur between self-report and direct measure scores due to self-perception issues (Dunning et al., 2004). Therefore to eliminate any issues of self-report inaccuracy, only direct performance measures were employed in study two. Tasks administered were developed for non-clinical populations using standardised scoring. Familiarity with the theoretical bases of these tasks allowed for interpretation of cognitive styles and abilities. The researcher administered the battery of tests in random order for the purpose of controlling for order effects.

6.6.3.4 Procedure

Participants were recruited via email advertisement to all faculties at the University of Bradford. All participants completed the experiment in one session that was carried out at the psychology labs in the Division of Psychology, University of Bradford. Before beginning the battery of measures, participants were required to read the information sheet pertaining to the study and if happy to proceed, provide their written consent. Sessions in the lab lasted approximately 30 minutes and no participants requested a break during the sessions.

6.7 Results

Study two aimed to replicate the results obtained in study one, that AM was able to significantly predict a person's place on the E-S continuum. Also, we advanced study two to include an 'interactive' AM element. Furthermore,

study two aimed to investigate the relationship between SME (false memory) in relation to empathising and systemising.

Table 9: Means and standard deviations of scores for all measures

Variable	*n*	M	±SD	Min-Max possible score
EFT	52	4.12	0.81	0-5
RTME	56	24.39	3.74	0-36
SW	57	5.49	2.08	0-10
FM	57	1.23	1.70	0-10

Note: EFT= embedded figures task; RTME= reading the mind in the eyes task; SW= say what?; FM= false memory

6.7.1 Regression analysis

6.7.1.1 Associative memory, empathising and systemising

A linear regression was calculated to predict RTME (empathising) score based on Say What (AM) performance. The regression model (say what) significantly predicted RTME score (F (1, 54) = 4.485, p<0.05). There was a small (R =.28) relationship between Say What score and RTME and the model could explain 8% of the variance (R^2 = .08, Adjusted R^2 =.06) (β = .28, t (54) = 2.11, p<0.05: 95% *CI* .027 - .988). Participants' predicted RTME score was equal to 21.64 + .51 (say what) when say what is measured in 1 point per correct response. RTME score increased .51 for each correct response on the say what task.

Table 10: The unstandardized and standardised regression coefficients for the predictor variable RTME

	B	SE B	B
Constant	21.64	1.39	
Say what	.51	.24	.28*

*p<0.05, **p<0.01, R^2 .08

Regression modelling revealed that there was no significantly relevant relationship between EFT performance and the say what task (*ns*).

This suggests that, as hypothesised, like the findings in study one, **AM shares a significant relationship with empathising**.

6.7.1.2 Source monitoring error, empathising and systemising

A linear regression was calculated to predict RTME score based on SME. The regression model (SME) significantly predicted RTME score (F (1, 54) = 4.10, p<0.05). There was a small (R =.27) relationship between SME and RTME score, and the model could explain 7% of the variance (R^2 = .07, Adjusted R^2 =.05) (β = -.58, t (54) = -2.02, p<0.05: 95% *CI* -1.15 - -.01). Participants predicted RTME score was equal to 25.10 + -.58 (source monitoring errors) when source monitoring is measured in 1 point per correct response. RTME score decreased .58 for each source monitoring error.

Table 11: The unstandardized and standardised regression coefficients for the predictor variable RTME

	B	SE B	B
Constant	25.10	0.60	
Source monitoring	-.58	.29	-.27*

*p<0.05, **p<0.01, R^2 .07

This suggests that although empathising shares a positive relationship with AM ability, **greater empathising was also associated with greater source monitoring memory errors.**

6.8 Main findings - study two

The results for study two provided support for the results of study one, confirming an association between social AM and empathising abilities using direct performance measures (RTME task) which extended to include stimuli that involved human interaction. Therefore testing different episodic memory brain systems and increasing the implications for social cognitive neuroscience. However, interestingly results here did not support the previous finding that systemising shared a negative relationship with AM ability. This is a significant finding as it questions the strength of the association between AM and the E-S continuum as a whole. However, intriguingly, the *same trend* was evident in false memory performance, in that SME was associated with empathising, but *not* systemising. This result could perhaps be explained by the particular task employed to measure

systemising ability in study two. The EFT measures global versus local processing (attention to detail), which is arguably just one small element of systemising; therefore, it would be interesting to include other systemising tasks to see if similar results are obtained, a notion which is worthy of future investigation[10].

The investigation into SME in relation to the E-S continuum is the first of its kind (to the knowledge of the author). The judgement that false memory was in fact a significant predictor of empathising ability is perfectly fitting with the view that *if* it is an accurate postulation that the ASD-schizophrenia continuum can overlap with the E-S continuum (Brosnan et al., 2010: Jones & Lesk, 2013), we would *expect* higher empathisers to (i) perform better on tasks of episodic memory and (ii) be more susceptible to false memories due to the *theorised* link between higher empathisers and schizophrenia (Chapter 5). This is a significant finding, both in furthering the knowledge of specific cognitive abilities in relation to the E-S continuum, and also, in providing support for the notion of a linkage between the E-S and the ASD-schizophrenia (IBT) continuums. Thus, the results obtained here offer the literature, novel characterisation of the EFB profile. In that, there is now evidence to suggest that the feminised profile will not only be characterised by greater empathic understanding alongside a deficit in systemising, but

[10] *Note:* Alternative explanation for this particular trend may lay in the lack of support for the fundamental 'trade-off' relationship between empathising and systemising, an investigation that is the primary focus of Chapter 10 of this thesis. Chapter 10 questions the validity of the fundamental principles of the E-S theory as an accurate model of cognitive profiling, and this argument is also a repeated trend in Chapters 7 and 8.

also, show greater ability in episodic memory alongside a subtle susceptibility to source monitoring failure.

6.9 General discussion

The primary aim of this two-part study was to explore additional aspects of cognition that could possibly predict a person's place on the E-S continuum (other than empathising and systemising) and furthermore, contribute to the knowledge of cognitive processes which may possibly be implicated in the investigation into the cognitive uniqueness of the EFB.

By exploring aspects of episodic memory such as SM, AM and SME (Study 2) in relation to the E-S continuum, results here put forward that **episodic memory and SME can indeed be significant cognitive markers on the E-S continuum, specifically when information involves a social context.** Meaning that as a person's place on the continuum places further towards the 'female' end of the E-S continuum, their aptitude for episodic memory increases. It also means that although capacity for episodic memory is increased, proneness to SME is also increased in the feminised profile. This finding is the first to the researcher's knowledge that is supportive of the trend in episodic memory predicted by the IBT (Badcock, 2011). For instance, the IBT predicts that episodic memory ability would be *advanced* towards the 'female' end if the continuum, which is supported in this study. Yet, a *deficit* in episodic memory would be apparent towards the EFB end of the continuum due to the manifestation of schizophrenia (Badcock, 2011). We can infer from these results that as a person plots further towards the female end of the continuum, their episodic memory ability is advanced,

however they are also susceptible to greater SME, seemingly due to the association between SME and positive schizophrenia (Lenzenweger & Gold, 2000; Brébion et al., 2002; Brébion et al., 2013; González-Ortega et al., 2013), and therefore supportive of the prediction of the IBT (Badcock, 2011). This study therefore furthers the knowledge on the cognitive profiles that exist on the E-S continuum and can boast a *new* cognitive measure that is implicated in the E-S theory model.

6.9.1 Interpretations

Firstly, considering the prominent finding that AM was a significant cognitive marker on the E-S continuum, [and if we take for granted that empathising and systemising abilities are gendered, which, collectively findings tend to support (see Baron-Cohen, 2003)], it could be argued that our results are in line with that of previous studies which found a female advantage during episodic memory tasks (Herlitz et al., 1997,1999; Herlitz & Yonker, 2002; Yonker et al., 2003; Herlitz & Rehnman, 2008), (specifically tasks which involve the remembering of faces [Lewin & Herlitz, 2002] as was employed in both Study 1 and 2). A male advantage in episodic memory is usually evident for stimuli that implicates visuospatial processes (Herlitz et al., 1997,1999; Lewin et al., 2001). This links with the results of study one which found that performance on the MROT and the EFT (systemising tasks) was significantly predicted by greater levels of correct pairings on the inanimate (shapes) AM task, but interestingly, not social (facial expressions) AM. Neurologically speaking, the finding that **social** AM was a significant predictor of empathising and systemising may be linked with the notion that

patterns of amygdala activation have been found to differ between males and females on emotional memory processes (Cahill et al., 2001, 2004). For instance, enhanced memory ability is associated with activation of the right amygdala for males on viewing emotional stimuli; however, in females left amygdala activation is associated with memory for emotional stimuli (Cahill et al., 2001, 2002). These findings led Cahill et al. (2001) to conclude that neurobiology *must* consider the important role of gender in emotional memory processes. This line of thinking is advocated here, as it is offered that cognitive processes that are influenced by gender, such as empathising and systemising ability (Baron-Cohen, 2003) can be significantly associated with memory networks, which suggested that gender/sex differences plays a significant role in this relationship.

Is it the case that empathisers encode emotional stimuli with greater detail than systemisers do? For instance, is it feature binding of social stimuli that is more pronounced in empathisers (Johnson & Raye, 1998), or perhaps enhanced activation of memory processes due to a preference for the stimuli? Further research that incorporates neuroimaging such as event related potentials (ERPs) for instance would be useful to further comprehend these findings.

Secondly, it was hypothesised (based on the knowledge that schizophrenia participants often demonstrate SMEs [Lenzenweger & Gold, 2000; Brébion et al., 2002; Brébion et al., 2013; González-Ortega et al., 2013]), that higher empathisers would demonstrate greater susceptibility to SME, based on the notion that the IBT has the potential to accurately characterise the EFB. Not only has this study found evidence of a link between episodic memory

capacity and the E-S continuum (Study 1); it has also provided support for the link between higher empathising and greater susceptibility to SMEs (Study 2). In turn, these results support the hypothesis that the E-S theory and the IBT can accurately fit together to explain the unknown pathology of the EFB profile. This is a significant finding and the first to the author's knowledge to link the two theories together using a stringent and controlled cognitive paradigm which goes beyond questionnaires measuring empathising, systemising and self-report ASD and schizotypy traits.

Thirdly, considering the apparent links between memory ability and ToM (Perner, et al. 2007; Bright-Paul et al. 2008), an interesting and important area for future research would be the investigation of the hMNS (Chapter 3, section 3.6.1) in relation to the E-S continuum. For instance, Badcock and Crespi (2008) postulate that ToM processes in ASD and schizophrenia are diametrically opposed. They advise that ToM in ASD individuals presents as a deficit due to an *underdeveloped* hMNS whereas ToM in schizophrenia presents as a deficit because of an *overactive* or deregulated hMNS which causes ToM to be so sensitive it becomes dysfunctional. Greater MU rhythm suppression has been found in schizophrenia patients compared to psychosis and control samples (McCormick et al., 2012). However, how this compares to ASD samples has not been determined. We would expect that ASD and schizophrenia would show contrasting patterns of activation in imaging studies when investigating social brain processes (Badcock & Crespi, 2008). It would be interesting to further explore the notion of a diametric relationship in MNS activity in relation to the extreme ends of the E-

S and IBT continuums which would allow the knowledge pertaining to this field of social cognitive neuroscience to progress further.

6.9.2 Conclusions

This study employed a novel episodic memory paradigm which explored SM, AM and SME in relation to the E-S continuum using both still pictures and social interactive measures in order to quantifying memory capacity. Results revealed that AM and SME can indeed be significant cognitive markers on the E-S continuum, specifically when the information participants were processing involved a social context. These findings confirm that there is evidence to suggest that the EFB profile will not only be characterised by greater empathic understanding, alongside a deficit in systemising, but also show greater ability in episodic memory, alongside a subtle susceptibility to source monitoring failure. Significantly, this deduces that memory ability can be a telling indication of where a person may place on the E-S continuum. This study not only furthers the knowledge pertaining to the cognitive correlates of the E-S model, but it has also advanced the debate on the compatibility between the E-S theory and the IBT continuum and provided novel implications for the field of social cognitive neuroscience.

6.10 Chapter summary of key points

- This chapter focused on the identification of cognitive functioning that had not previously been considered in relation to the E-S model.
- The rationale for the investigation into memory ability and the E-S theory was derived from the hypothesis of the IBT that posits episodic memory ability will be advanced in higher empathisers. Thus, allowing exploration into the links between the E-S theory and the IBT.
- The two-part study administered novel source monitoring and AM task paradigms in university student samples.
- Results revealed that both **AM ability** and the **frequency of SMEs** are a **predictive cognitive marker** of a person's place on the E-S continuum, specifically when the memory involved a social context.
- This is the first study to the knowledge of the researcher that has investigated memory in relation to the E-S theory and the IBT. Not only does this establish new knowledge in relation to the other cognitive networks that are implicated in the E-S model, it also provides the first study to find empirical support for the IBT's postulation that higher empathisers are likely to experience source monitoring failure, based on the theorised link between diminished episodic memory capacity and schizophrenia.
- The following chapter directly investigates E-S theory 'brain types' in relation to schizotypy.

Chapter 7

Empathising – systemising 'brain types' and their relationship with schizotypy, eating disorder and anxiety[11]

7.1 Introduction

It has been proposed that a bias in innate sex differences at extreme levels can significantly contribute to the development of particular neurodevelopmental disorders (Baron-Cohen, 2002; Badcock & Crespi, 2008). The aim of this experiment was to explore the specific characteristics of the 'brain types' (see Chapter 3, section 3.4) proposed by E-S (Baron-Cohen, 2003) in relation to proposed pathological consequences of these innate sex differences. This chapter investigates if it is plausible that schizophrenia, eating disorder (ED) or anxiety disorder (AD) could be considered an accurate consequence of a type E brain profile.

7.1.1 Background

The observation that ASD can convincingly be linked with sex differences in terms of behaviour and cognitive preferences (e.g. systemising and empathising) led to the development of the E-S theory (Baron-Cohen, 2003). See Chapters 3 and 4 for details. To briefly recap, the E-S theory proposes that sex differences in cognition have become hard-wired due to a process of brain plasticity adapting to reflect historical social gender roles played by

[11] Some of the information presented in this chapter is subject to a manuscript in preparation entitled 'Empathising – systemising brain types in relation to schizotypy' by S. L. Jones and V. Lesk.

males (systemisers) and females (empathisers) (Baron-Cohen, 2003). Empathising is our intuitive ability to understand social surroundings, to communicate with others, and appreciate that other people have a state of mind that might be different from our own. It also encompasses our ability to respond appropriately to other people's behaviour, even when we cannot predict what it might be (Baron-Cohen, 2004). Systemising on the other hand, refers to our spontaneous aptitude to analyse systems, to have a preference for things that can be understood by cause and effect or input-output logical, rather than unpredictable mechanisms and do so with relative ease (Baron-Cohen, 2004).

The E-S theory (Baron-Cohen, 2003) ascertains that by quantifying levels of empathising and systemising ability, it is possible to plot people on a continuum of cognitive profiles according to their 'brain type' (see Figure 10).

BRAIN TYPE	COGNITIVE PROFILE
EXTREME TYPE S (S>>E) (EXTREME MALE BRAIN)	Significantly advanced systemising ability alongside a major deficit in empathising ability
TYPE S (S>E)	Subtle advanced systemising ability alongside a subtle deficit in empathising
TYPE E = S (BALANCED BRAIN)	Balanced brains are those which present with no advantage or deficit in either empathising or systemising
TYPE E (E>S)	Subtle advanced empathising ability alongside a subtle deficit in systemising
EXTREME TYPE E (E>>S) (EXTREME FEMALE BRAIN)	Significantly advanced empathising ability alongside a major deficit in systemising ability

Fig. 10: The E-S theory brain types (Baron-Cohen, 2003)

The extreme type E profile, also known as the EFB (Baron-Cohen, 2003), is characterised by superior cognitive ability in ToM processing and social intelligence (empathising) alongside a severe deficit in systemising intellect. The EFB (as yet), has not been convincingly linked to any known pathological consequence (Baron-Cohen, 2003). However, it is suggested that it is *probable* that sex differences at extreme levels like those theorised in the EFB profile would realistically result in clinically relevant pathology that disrupts daily living significantly enough to require diagnosis and intervention. The IBT (Badcock & Crespi, 2008) offers us (indirectly) a plausible candidate for a pathological consequence of the EFB. It posits that ASD and schizophrenia exist as on a continuum as diametric opposites as disorders of the social brain.

The proposal of the ASD – schizophrenia continuum has led some researchers to investigate the compatibility between the E-S theory and the IBT, to establish if schizophrenia is in fact a plausible candidate for the pathological consequence of the EFB profile. So far, preliminary support for a positive association between empathising and schizotypy has been reported (Brosnan et al., 2010; Jones & Lesk, 2013), however a *consistent* relationship between schizotypy and systemising ability has not been apparent (Russell-Smith et al., 2010).

In addition, Chapter 6 of this thesis supports the association between a feminised profile and indicators of SME, which is predicted by the IBT to be evident in schizotypy, thus providing preliminary support for the compatibility between the feminised and schizophrenic phenotypes. This chapter suggests that whilst an association between empathising ability and schizotypy can

boast some *limited* support, overall, the link between the EFB profile and schizophrenia as a pathological consequence is not well supported. This is because we would expect to observe a consistent *negative* association between systemising ability and schizotypy, and this relationship has yet to be established. Unless schizotypy and schizophrenia can be linked to both empathising and systemising processes in the pattern that would be expected by the EFB profile, the IBT does not offer us a pathological consequence to the allusive EFB pathology.

7.1.2 The EFB and disordered eating

Whilst the major investigation of this thesis is in relating schizophrenic phenotypes to the feminised profile, other researchers have taken a different approach in investigating possible pathological candidates for the EFB profile. For instance, Bremser and Gallup (2012) hypothesised an association between the EFB and disordered eating.

ED can exist both at clinical and general population levels, ranging from a preoccupation with controlling calorie intake, to severe life-threatening mental illness (MIND, 2013). Signals of ED can include obsessions with the amount of food consumed, constant worry about body weight and complete avoidance of particular food groups (MIND, 2013). Interestingly, disordered eating can often be the result of more than just a fear of becoming overweight; it often involves a way for the individual to feel in control, or can be an expression of painful feelings and can manifest as a coping mechanism to deal with difficulties in other areas of life (MIND, 2013). The

most common ED is anorexia nervosa (referred to as 'anorexia' going forth), where the individual is under the illusion that they are overweight, even if they are perfectly healthy or even underweight (NIMH, 2016). To control this, individuals with anorexia excessively limit the amount of food they consume, resulting in becoming dangerously underweight (NIMH, 2016). Another common eating disorder is bulimia nervosa (bulimia). Bulimia is characterised by out of control intermittent overindulging on copious amounts of food followed by forced vomiting as a way of controlling calorie intake (Russell, 1979). According to the NIMH (2016), any type of disordered eating is most likely to occur during adolescence and the early 20s and affects both genders. In addition, it is most likely influenced by genetics as the condition has a tendency to run in families. All types of disordered eating are related to an obsession with food and calorie intake, whether it been under or over consumption of food. The manifestation of ED ultimately leads to range of harmful psychological and physical consequences that usually require professional medical intervention (NIMH, 2016).

Bremser and Gallup (2012) hypothesise an association between ED and the EFB profile for a number of reasons. Firstly, they state that EDs exist on a continuum much like ASD (the pathology of the extreme masculinized cognitive profile). Secondly, females are 10 times more likely than males to be diagnosed with an eating disorder. Therefore, Bremser and Gallup (2012) propose that there are some obvious similarities between the principles of the E-S theory and ED. Further to this, Bremser and Gallup (2012) offer that there is also evidence that androgens and estrogens play an important role in the development of ED, specifically anorexia and bulimia, much like the role

of hormones in ASD. Interestingly in terms of the possible links with the type E profile, lower levels of fT (measured by the 2D:4D proxy) have been found to be associated with higher prevalence of EDs (Quinton et al., 2011). It is also considered by Bremser and Gallup (2012) that the manifestation of EDs are likely linked to 'fear of negative evaluation' anxiety (FNE), an anxiety experience that results in the avoidance of any social situation that involves anticipated negative evaluation by others (Waston & Friend, 1969). FNE is likely to be appropriate here, in that weight gain can be associated with the fear of the judgement by others in social situations (Bremser & Gallup, 2012). In support of this, other researchers have found that anxiety is often a common co-morbidity of ED (Garfinkel et al., 1995; Rowe et al., 2002; Keel et al., 2005; Swinbourne & Touyz, 2007). Including conditions such as panic disorder, agoraphobia, specific and social phobia, generalized anxiety disorder and post-traumatic stress disorder (Hudson et al., 2007).

In their four-part study Bremser and Gallup (2012) found that (in experiment one) those who demonstrated a bias for empathising over systemising scored significantly greater on scores of disordered eating *and* FNE anxiety in a healthy female sample, suggesting support for a possible association between the EFB, eating disorder and fear of negative evaluation. Furthermore, they found that self-report empathising was consistently positively correlated with greater levels of ED and FNE traits (this result was replicated in the further three experiments), yet on closer inspection of these studies, throughout, systemising *failed* to show a consistent association with either ED or FNE. The only significant finding pertaining to systemising cognition was a negative correlation between performance on the mental

rotation task and self-report ED in the fourth experiment. However, consistent associations between systemising and ED and FNE were evident.

Bremser and Gallup (2012) are the only researchers to the researcher's knowledge to have investigated the links between the EFB profile and ED traits. However, Hambrook and colleagues (2008) were interested in the relationship between ASD traits, empathising, systemising and in-patient anorexia. They reported no significant differences on scores of empathising or systemising in anorexia patients when compared to healthy controls (female only sample). Yet, they did find that anorexia patients scored significantly higher on ASD traits than matched controls, which implies a link between ASD traits and anorexia, going against the trend expected if ED is a likely EFB candidate.

Despite the findings by Bremser and Gallup (2012), this chapter argues that there is substantially more support for a probable link between a *type S* (S>E) profile rather than a *type E* profile, and ED. For instance, cohorts with anorexia have been found to have elevated levels of ASD traits (Baron-Cohen et al., 2013; Tchantura et al., 2013) and Wentz et al. (2005) advises that 23% of people with a diagnosis of anorexia meet the cut off for diagnosis of ASD. Tchantura et al. (2013) propose there is support for a significant *overlap* between anorexia and ASD in terms of cognitive and behavioural features, rather than a discrepancy. For instance, impaired recognition of facial emotion and poorer performance on ToM tasks, similar to those reported in ASD, have been observed in both clinically relevant disordered eating (Kucharska-Pietura et al 2004; Tchanturia et al., 2004; Pollatos et al., 2008; Jansch et al., 2009; Russell et al., 2009; Harrison et al., 2010a,b) and

non-clinical but 'at risk' cohorts (Jones et al., 2009; Ridout et al., 2010,2012 ;Sharpe et al., 2016). These findings suggest a deficit rather than an advantage in empathising is associated with ED behaviours. Further to this, a systemising profile similar to that of ASD individuals has been observed in cohorts with ED, for instance, superior ability on the EFT has been found to be associated with ED (Lopez et al., 2008). Additionally, Oldershaw et al. (2011a) employed the 'reading the mind in the films and voices' task (Golan et al., 2006) to measure cognitive empathy and the Wisconsin card sorting task and the EFT to measure systemising in an in-patient ED cohort. They concluded that ED individuals present with cognitive profiles that are similar to individuals with ASD, where ToM was disadvantaged and systemising ability was advanced.

Moreover, there is evidence of shared endophenotypes in both ASD and anorexia (Zucker et al., 2007; Treasure, 2007; Oldershaw et al., 2011b; Treasure, 2013), along with atypical brain structure in ED similar to that which is usual in ASD (Zucker et al., 2007). Also, children with ASD have found to have significantly more disordered eating traits than controls (Schreck et al., 2004), again, supportive of a stronger link between a type S profile and ED, rather than a probable EFB candidate. However, this chapter suggests that it is unclear if these behaviours involve the manifestation of ED, or are they the result of obsessive and compulsive behaviours that are associated with ASD itself?

Bremser and Gallup's (2012) findings are interesting, however, as previously alluded to; there is no strong evidence of a consistent relationship between systemising and ED or FNE reported in their studies. This suggests that the

declaration of a link between the EFB and ED is relatively weak. These findings require replication and scrutiny especially in terms of the consideration of systemising abilities, in order for any real conclusions to be made.

7.1.3 The EFB and anxiety

Pertaining to sex differences in the prevalence of ADs, anxiety and stress related disorders are consistently more prevalent in females than males (Turk & Heimberg, 1998; Weinstock, 1999; Armstrong & Nigar, 2002; Vesga-López et al., 2008; McLean & Anderson, 2009; Bangasser et al., 2010; Mclean et al., 2011). Because this trend is seen across cultures (Altemus, 2006), there is an argument that females are more than likely genetically predisposed to ADs compared to males (see McLean & Anderson, 2009 for a detailed review). It is also suggested that it is more than likely that reproductive hormones play an important role in the manifestation of feelings of anxiety (Altemus, 2006). For instance, lower levels of estrogen and progesterone are thought to contribute to the experience of greater levels of anxiety and stress responses reported in premenstrual females (Seeman, 1997) and levels of estrogen significantly affect the course of preexisting anxiety disorders (Pigott, 1999). Furthermore, social anxiety is a usual co-occurrence (Bremser & Gallup 2012) and a *pre*-occurrence of the manifestation of an ED (Kaye et al. 2004; Steinhausen 2002), suggesting that it is possible that the manifestation of disordered eating may sometimes be the result of underlying AD.

Bremser and Gallup (2012) employed 122 participants from a student sample and found EQ scores were positively associated with higher self-report anxiety levels. Moreover, other investigators have hypothesised that individuals with social anxiety will likely show greater empathising abilities. This is due to a heightened concern pertaining to the perceptions other people might have about them, resulting in better than average social perception abilities (Tibi-Elhanany & Shamay-Tsoory, 2011). However adversely, it is also widely reported that ASD often presents with co-morbidity of AD (Ghaziuddin et al., 1998; Munis & Steerneman, 1998; Kim et al., 2000; Tatum, 2000; Kessler et al., 2005; Russell & Sofronoff, 2005; White et al., 2009; Davis et al., 2011). In fact, the prevalence of AD is approximately 50% in ASD (White et al., 2009) compared to just between 2% and 5% in the general population (Airaksinen et al., 2004), with higher-functioning ASD experiencing even greater levels of anxiety (Weisbrot et al., 2005). However, we make the argument here that it is *probable* that it is more likely the case that feelings of anxiety manifest in ASD *as a result* of the complexities of living with ASD, rather than a shared aetiology between ASD and AD. Supportive of this line of thinking, the presence of the experience of anxiety in ASD typically worsens during adolescence, possibly due to the increasing complexity of the social environment at this time. This could be due to people becoming more aware of their own difficulties with social communication, hence causing enhanced levels of anxiety (White et al., 2009).

Is there an argument to be made here that it is specifically the *anxiety* component of the ED hypothesis that is a more plausible match for the cognitive profile of the EFB, when considering the apparent female bias in

prevalence of the disorder? This chapter suggests that this is a reasonable theory and is worthy of investigation.

7.2 The present study: research aims

The novelty of the present study was that, considering the issues surrounding the inconsistencies in terms of the relationship between schizotypy, ED, anxiety and systemising, the aim here was to quantify E-S theory 'brain types' in order to *comprehensively* explore the suggested links between these disorders in relation to the potential pathology of the EFB. The justification for this method is that Bremser and Gallup (2012) conclude evidence to suggest a link between ED, fNE and the EFB profile without fully considering that such an association *must* incorporate systemising as much as empathising, to be in line with the theoretical characteristics of the EFB. The overall rationale here is that by quantifying the sample into brain types according to self-report empathising and systemising scores, this method will provide a more accurate investigation into the possible pathological characterisation of the EFB as it incorporates empathising and systemising equally.

7.3 Methods

7.3.1 Participants

A total of 158 participants took part in the study. Respondents were recruited from the University of Bradford and the general population via various social media channels. Gender was not recorded, as analysis was carried out using 'brain types' (Carroll & Yung, 2006); the postulation that gender is not a

determinant of brain type, that level of exposure to fT masculinises and feminises the brain. The mean age recorded by participants was 34 years (±SD= 14.01, Range= 56). Inclusion criteria specified that participants must have (i) no clinical diagnosis of mental illness or neurological disorder (ii) a fluent understanding of English language (iii) must not have taken part in previous studies conducted by the researcher in the past (due to over exposure effects). Ethical approval was obtained from the Humanities, Social & Health Science Research Ethics committee at the University of Bradford.

7.3.2 Design

An independent groups design was employed in which the sample was categorised (post data collection) into 'brain types' using the method by Carroll and Yung (2006).

7.3.3 Materials

Self-report measures were administered which aimed to measure empathising and systemising, schizotypy, ED, fNE and generalized AD.

The Empathising Quotient (EQ) - The short form (EQ-S) version of the EQ (Wakabayshi et al., 2006b) measuring self-report cognitive and affective empathy was administered. (See section 6.3.3.2 and appendix 1).

The Systemising Quotient (SQ) - The short form version (SQ-S) of the SQ was employed, which measures self-reported 'everyday' systemising ability (Wakabayshi et al., 2006b). (See section 6.3.3.2 and appendix 2).

The Oxford and Liverpool Inventory of Feelings and Experiences (O-LIFE)- The O-LIFE (Mason, et al., 2005) is designed to measure schizotypal personality; it is specifically employed to measure schizotypy in the healthy population. It consists of 43 statement questions which participants respond with a yes/no option. It measures four dimensions of schizotypy; unusual experiences (UnEx), cognitive disorganisation (CogDis), impulsive non-conformity (ImpNon) and introvertive anhedonia (InAn). A higher score is representative of higher trait levels of psychosis. The four dimensions can be scored collectively for a 'schizotypy' score or scored individually in order to look at the dimensions separately. Full inventory can be found in appendix 4.

Eating attitudes scale (EAT-26) - The EAT-26 (Garner, et al., 1982) is a specifically designed standardised measure of ED and the severity of ED traits. Comprising of 26 items, the short form version with six response options was employed. The scale measures three factors of ED (i) focusing on dieting (ii) bulimia and (iii) food preoccupation. It is the most widely used and reliable measure of EDs (Bremser & Gallup 2012). Full inventory can be found in appendix 5.

GAD-7 Anxiety (GAD-7) - The GAD-7 (Spitzer, et al., 2006) is a self-report measure used to determine level of generalised AD. It consists of seven statement items and a four point Likert scale response format. Internal consistency and good re-test reliability have been reported (Lowe et al.,

2008; Spitzer et al., 2006). A higher score indicates a higher level of generalised AD traits. Full inventory can be found in appendix 6.

Fear of negative evaluation scale (FNE) - The FNE (Leary, 1983) is a self-report measure of social anxiety and social avoidance. It is designed to measure *specifically* anxiety surrounding possible negative evaluations by other people. The brief version (bFNE) was administered in this study, which includes 12 statement items with Likert scale response format. A higher score indicates higher levels of social anxiety traits. Full inventory can be found in appendix 7.

7.3.4 Procedure

All participants completed the battery of measures online using a specifically designed survey website. Before beginning the battery, respondents were required to read the information sheet pertaining to the study and if happy to proceed, completed the online consent process. The battery of measures was administered in the same order to all participants. Respondents took approximately 40 minutes to complete all measures. Responses from participants who failed to complete the whole battery of measures were automatically excluded from the data set by the survey software.

7.4 Results

The aim of this study was to explore schizotypy, ED and AD traits in relation to the E-S theory 'brain types' in a neurotypical population sample (n= 158). Analyses were carried out using the Statistic Package for the Social Sciences

(SPSS) version 20.0 using score data. Any missing variables were excluded pairwise.

The sample was categorised into groups of 'brain types' using the method offered by Carroll and Jung (2006). EQ-S and SQ-S scores were firstly converted to *z*-scores and the difference between the scores was calculated. A score of >1.00 was suggestive of a bias for a systemising profile or a 'type S' brain. A score of <-1.00 suggests a bias for an empathising profile or a 'type E' brain and scores between -1.00 and 1.00 represents a 'balanced brain' profile, which is neither a bias in empathising or systemising. Ranges, means and standard deviations by 'brain type' can be found in Table 12.

Table 12: Means and standard deviations for scores on all variables

BRAIN TYPE	SCALE	*n*	MEAN	±SD	MIN-MAX POSSIBLE SCORE
Type S (n = 23)	O-LIFE	18	20.28	8.19	0-43
	UnEx	21	5.29	2.51	0-12
	CogDis	22	6.09	3.52	0-11
	InAn	21	3.95	2.62	0-10
	ImpNon	21	4.19	2.38	0-10
	EAT-26	21	8.48	7.59	26-156
	GAD-7	22	5.05	5.06	0-21
	bFNE	22	32.05	9.93	12-60
Balanced brains (n = 85)	O-LIFE	77	15.19	6.74	0-43
	UnEx	83	4.90	2.79	0-12
	CogDis	84	4.76	2.94	0-11
	InAn	82	2.43	2.20	0-10
	ImpNon	81	3.11	1.86	0-10
	EAT-26	71	11.52	9.50	26-156
	GAD-7	85	6.34	5.33	0-21
	bFNE	80	39.89	9.93	12-60
Type E (n = 24)	O-LIFE	19	13.21	7.15	0-43
	UnEx	22	3.50	2.94	0-12
	CogDis	23	4.22	2.78	0-11
	InAn	20	2.25	1.86	0-10
	ImpNon	24	2.75	1.98	0-10
	EAT-26	19	7.68	6.34	26-156
	GAD-7	24	4.83	4.40	0-21
	bFNE	24	34.63	11.89	12-60

Note: O-LIFE= Oxford and Liverpool inventory of feelings and experiences (total score); UnEx= O-LIFE unusual experiences; CogDis= O-LIFE Cognitive disorganisation; InAn= O-LIFE introvertive anhedonia; ImpNon= O-LIFE impulsive nonconformity; EAT-26= Eating attitudes test; GAD-7= GAD-7 anxiety; bFNE= Fear of negative evaluation (brief)

7.4.1 Schizotypy

One-way analysis of variance (ANOVA) was conducted to explore the impact of brain type on levels of schizotypy as measured by the O-LIFE scale. There was a significant difference between the three groups (F (2,111) = 5.20, $p<0.01$), $\eta^2 = 0.09$. Post-hoc comparisons indicated that type S brains (M= 20.28, ±SD= 8.19) scored greater than type E brains (M= 13.21, ±SD= 7.15), and balanced brains (M= 15.17, ±SD= 6.74), but there was no significant difference in schizotypy scores between balanced and type E brains.

The O-LIFE scale comprises of four dimensions; ANOVA analysis revealed that brain types scored differently on the InAn dimension (F (2,120) = 4.33, $p<0.05$, $\eta^2 = 0.07$) and the ImpNon dimension (F (2,123) = 3.32, $p<0.05$, $\eta^2 = 0.5$). No differences on the other scales were evident although trends were apparent [UnEx (p = 0.06), CogDis (p = 0.09)]. Post-hoc tests revealed the difference in score on the InAn scale was between the type S (M= 3.95, ±SD= 2.62) and type E (M= 2.25, ±SD= 1.86) brains, type S and balanced (M= 2.43, ±SD= 2.20) brains, but not balanced and type E brains. The difference between scores on the ImpNon scale lay between type S (M= 4.19, ±SD= 2.38) and type E brains (M= 2.75, ±SD= 1.98), but not balanced (M= 3.11, ±SD= 1.86) and type S brains or balanced and type E brains.

7.4.2 Eating disorder

ANOVA analysis offered that there was no difference between brain types on scores on the EAT-26 (*ns*).

7.4.3 Anxiety

ANOVA analysis offered that there was no difference between brain types on scores on the GAD-7 scale (*ns*).

7.4.4 Fear of negative evaluation

ANOVA analysis offered that there was no difference between brain types on scores on the bFNE scale (*ns*).

7.5 Discussion

This study aimed to investigate E-S theory 'brain types' in relation to dimensional schizotypy, disordered eating and anxiety traits. The purpose of this study was to contribute to the wider question of whether or not there is a fitting pathological consequence of the EFB.

Results here offer no support for previous findings that supported an association between a hyper-empathising profile, disorder eating, and fNE anxiety (Bremser & Gallup, 2012). It was argued in the introduction that in order to conclude any convincing evidence of an association between any personality trait *or* pathological condition and the EFB, the model **must** incorporate the role of systemising as well as with empathising. These results, which employed a model to incorporate systemising ability by quantifying E-S brain types, showed *no* difference between type E, type S and balanced brains on measures of disordered eating or FNE, therefore we do not advocate ED or FNE to be an accurate candidate for the allusive EFB profile and reject previous findings. Bremser and Gallup (2012) failed to find a *consistent* relationship between systemising, ED or FNE, therefore we

argue that, considering this important aspect, there is not sufficient evidence to argue that ED or FNE are plausible candidates for the pathological consequence of the EFB as Bremser and Gallup (2012) have previously suggested.

Results here also fail to support an association between anxiety and the type E brain, as type E, S and balanced brains did not score significantly different on levels of social or generalised anxiety.

However, our results offer important and interesting findings in terms of the relationship between E-S theory brain types and schizotypy. It would be expected by the IBT (and results in Chapter 6) that a profile with greater empathising ability and lesser systemising would present with greater levels of schizotypy. Overall, our results demonstrated that **type S brains scored greater on scores of schizotypy than both balanced brains and type E brains**. This is a major finding and suggests that cognitive profiles that have a subtle advantage in systemising over empathising, experience greater schizotypy, going *against* the notion of the IBT.

The dimensional nature of schizophrenia signifies the importance of exploring the different expressions of schizotypy when quantifying these traits (Mason & Claridge, 2005). On closer inspection of the O-LIFE results, the difference in scores between the brain groups was evident on **negative**[12] and **impulsive nonconformity**[13] traits. Brain types showed no differences on

[12] Negative symptoms of schizophrenia refer to reduced affect, the avoidance of intimacy and the lack of enjoyment from social situations (Mason & Claridge, 2005).

[13] Impulsive nonconformity refers to impulsive behaviours, a lack of self-control and anti-social behaviours (Mason & Claridge, 2005).

levels of positive[14] or disorganised[15] phenotypes, although directional trends were evident in way of greater scores for type S brains on both positive (p = 0.06) and thought disorder (p = 0.09) dimensions, with type E brains scoring lowest on positive and thought disorder traits of the three profiles. This goes directly against the predictions that would be expected if schizotypy were to be associated with the EFB, showing little support for the ASD – schizophrenia continuum. Interestingly, these results provide confirmation of similar results reported in Chapter 9 of this thesis, which found that higher levels of ASD traits were associated with both negative and impulsive nonconformity dimensions of schizotypy.

In ways, we would expect type S brains to score greater on negative affect symptoms [flattened affect, poverty of speech and language (Andereasen, 1982)] as these traits are common in ASD and as an extension, type S brains, due to the established links between the two cognitive profiles (Baron-Cohen, 2002). As for the unexpected relationship between greater levels of impulsive nonconformity and the type S brain, further investigation is warranted. We can think of impulsive nonconformity as a positive dimension of schizophrenia, therefore, to find that levels of impulsive nonconformity are greater in type S brains is perplexing for the idea that positive dimensions of schizophrenia would be associated with type E brains (Badcock & Crespi, 2006). Impulsive nonconformity has been found to be a significant predictor of manic episodes of schizophrenia (Blechert & Meyer, 2005) and associated

[14] Positive symptoms refer to magical thinking, hallucinations and paranoid ideation (Mason & Claridge, 2005).

[15] Thought disorder refers to poor attention, social anxiety and poor decision making abilities (Mason & Claridge, 2005).

with greater levels of creativity (Nettle, 2006; Batey & Furnham, 2008; Claridge & Blakley, 2009; Nelson & Rawlings, 2010), strengthening its relationship with positive symptomology. Troublingly, creativity is often diminished in ASD (Craig & Baron-Cohen, 1999). Further supporting the link between the experience of psychosis and creativity, similar functional brain activity has been found in psychosis and creative processing (Fink et al., 2013). In fact, creativity has a long standing association with psychosis (Eysenck, 1999), which suggests our findings do not support the notion of the ASD - schizophrenia continuum, at least in healthy population samples, as theoretically we would expect greater levels of impulsive nonconformity in type E brains.

As for the higher level (trend only, $p = 0.06$) of thought disorder[16] in type S brains compared to balanced and type E brains, further investigation is also required. It has been suggested by other researchers that thought disorder in ASD is probably not related to schizophrenia symptoms, but instead related to pragmatic language abnormalities that are present in individuals with ASD (Solomon et al., 2008). Nevertheless, thought disorder has been previously reported in ASD samples (Dykens et al., 1991; Volden & Lord, 1991; Baltaxe & D'Angiola, 1992; Ghaziuddin et al., 1995; Van der Gagg et al., 2005). Therefore, the fact that results in this study showed that type S brains were associated with greater thought disorder follows suit with the characteristics we would expect to observe on the E-S continuum.

[16] A disruption to the flow of thoughts (Eussen et al., 2014).

7.5.1 Concluding remarks

Baron-Cohen (2003) stated that as yet, there is no known pathology of the EFB, as we are yet to observe a pathology that is highly attune to empathising whilst encompassing a severe deficit in systemising. This study must conclude support for that statement. This is the first study, to the knowledge of the researcher, that has investigated 'brain types', in relation to schizotypy, ED and AD traits. It is concluded that no support for the notion of the IBT as an accurate characterisation of the type E brain has been found. If the principles of the IBT were accurate, relationship between psychosis and the type E brain profile should be evident in trait populations.

7.6 Chapter summary of key points

- This chapter explored the hypothesis that schizotypy is associated with a type E brain profile (Badcock & Crespi, 2008) by quantifying the sample into 'brain types' using the method by Carroll & Yung (2006), in relation to dimensional schizotypy.
- Results revealed that contrary to the hypothesis of the IBT, **type S brains** (those who scored *lesser* on empathising and *greater* on systemising abilities) scored ***greater*** on scores of **schizotypy** (overall) than both balanced brains and type E brains.
- This important finding ***does not*** support the IBT's prediction that schizotypy is associated with the feminised profile. Instead, results demonstrated that a *masculinised* profile scores *significantly* higher on schizotypy traits, leading to the conclusion that schizotypy is

associated with a systemising bias, *not* an empathising bias – in direct opposition to the IBT.

- This however is *not* surprising when considering the difficulties with social intelligence experienced in ASD, which may lead to the expression of symptoms which are qualitatively similar to negative symptoms of schizotypy such as flattened affect, avoidance of social intimacy and situations.
- This study highlighted the importance of further investigating the link between schizotypy and the type E profile by exploring the *individual phenotypes* of schizotypy for a more accurate investigation into the potential pathology of the feminised profile.

Chapter 8

Is the 'female brain' more likely to be associated with jumping to conclusions bias and paranoid ideation?[17]

8.1 Introduction and Background

Paranoid ideation is a major component of positive schizophrenic symptomology (Badcock & Crespi, 2006). The IBT speculates that positive symptoms of schizophrenia exist at the extreme end of a continuum of cognitive profiles, as the diametric opposite of ASD. It is plausible to consider that the IBT's hypothesis may accurately characterise the cognitive profile of the E-S theory's allusive EFB profile (Baron-Cohen, 2003). The E-S theory incorporates the well-established model of the EMB, in that ASD presents with a preference for a 'systemising' cognitive style (i.e. a tendency for structured, logical, cause and effect thinking) alongside a *deficit* in empathising ability (Baron-Cohen, 2002).

Previous studies have devised experimental research to assess the possibility that the EFB can be accurately characterised by the IBT, but so far have reported equivocal results. For instance, Brosnan et al. (2010) found that self-reported positive psychosis traits were positively correlated with self-reported empathic traits in a population of 70 healthy females. Further to this, Jones & Lesk (2013) also found that self-reported positive schizotypy traits shared a positive relationship with both self-reported and direct performance

[17] Some of the information presented in this chapter is subject to a manuscript in preparation entitled 'Is the 'female brain' more likely to be associated with jumping to conclusions bias and paranoid ideation?' by S. L. Jones and V. Lesk.

measures of empathising (measured by ToM tasks), in a neurotypical sample of 21 females and 10 males. Interestingly, Jones & Lesk (2013) also found a smaller *negative* relationship between self-report positive schizotypy traits and *some* measures of systemising ability. This lends support at a healthy population level to the concept of the diametric ASD-schizophrenia continuum. Additionally, Russell-Smith et al. (2010) considered local versus global processing (to investigate systemising cognition) in relation to the predictions of the IBT. They concluded that in two groups of 20, one defined by low autistic traits alongside high schizotypy and the other defined by high autistic traits alongside low schizotypy, the group identified as low autism - high schizotypy were significantly slower in identifying embedded figures compared to the high autism - low schizotypy group. This too offers support for the predictions of the IBT continuum. Yet, in a large sample of 1001 students (two studies), Russell-Smith et al. (2011) investigated the relationship between self-report measures of schizotypy and autistic traits and concluded little support for the *diametric* nature of the continuum. They instead found evidence for an *overlap* of autistic and schizotypy, in that those who reported greater autistic tendencies also reported greater schizotypy, offering little support for the IBT. Similarly, Chapter 9 of this thesis reports a comorbidity of self-reported schizotypy and autistic traits in a neurotypical sample of 157 participants, but only in negative symptoms[18] and thought disorder[19] phenotypes, whereas positive symptoms of schizotypy (as the IBT

[18] Negative symptoms of schizophrenia include flattened affect, avoidance of intimacy and lack of enjoyment from social situations (Mason & Claridge, 2005).

[19] Thought disorder in schizophrenia pertains to poor attention, decision making and symptoms relating to social anxiety (Mason & Claridge, 2005).

would predict) did not show any association with the manifestation of ASD traits. Also, in a separate study (n = 57) reported in Chapter 7 type S brains were found to experience *greater* levels of schizotypy than type E brains. Again, these results fail to support the expected trend of the IBT. Furthermore, Dinsdale et al. (2013) examined the ASD - schizophrenia continuum in a large non-clinical student population of 380 females and 225 males. They concluded limited support for the diametric relationship (evident only when involving social skills and visual spatial ability), yet also found evidence to suggest an overlap in certain traits (those which related to interest in and ability to understand social communication). However, these correlations were most strongly evident when involving negative symptoms of schizotypy and autistic traits. Collectively, results offer a complex and confusing experimental insight to the theoretical predictions of the IBT, and further investigation is warranted.

This reflection highlights the importance of further exploring the many questions surrounding the evidently complex relationship between ASD and schizophrenia.

Is the postulation of the diametric nature between the broad combined phenotypes of ASD - schizophrenia too simplistic? It seems significant to consider the notion that specific cognitive processes or behavioural or personality traits may share *specific* types of relationships with each other. Theoretically, the diametric nature of the two pathologies is plausible, if not convincing (see introductory chapters for detail). However, considering the shortage of experimental support for IBT, it seems necessary to explore with greater focus on the individual components of ASD and schizophrenia traits.

To therefore investigate if it would be more accurate to suggest specific cognitive processes share specific types of relationships. By focusing on individual processes and abilities in isolation, we may be able to offer a more comprehensive investigation into the seemingly complex relationship between ASD and schizophrenia traits and ultimately how this relationship might relate to the E-S theory and answering the question of whether or not the IBT has in-directly suggested an accurate pathology for the elusive EFB.

The theoretical justification for the present study derives from the close affiliation between ToM and the experience of paranoia (Badcock & Crespi, 2008; Thakkar et al., 2008). Also, the notion that the IBT is primarily a model of social brain dysfunction [i.e. the IBT suggests that positive schizophrenia occurs as a consequence of ToM processes which are so deficiently sensitive that they become dysfunctional (Badcock, 2009)]. The IBT proposes that people with positive schizophrenia symptoms 'over-infer' the intentions of others, which results in paranoid thoughts, as opposed to the significant lack of ToM demonstrated in ASD profiles, where one cannot attribute others' mental states due to a severe deficit in ToM (Badcock & Crespi, 2006). Interestingly, and pertinent to this investigation, a review of the literature exploring the relationship with ToM and paranoia led Chan and Chen (2011) to conclude that whilst a deficit in ToM is widely reported in schizophrenia, the specific relationship between the *separate components* of schizophrenia (e.g., paranoid delusion) is a gap in the knowledge, in that so far, the relationship is little more than theoretical.

8.1.1 Paranoia

Our ability to make judgements as to whether or not we can trust other people is a central part of our social intelligence (Freeman et al., 2008). It is thought that paranoid thoughts may arise from evolutionary adaptations, which have been helpful to our survival and have developed for the purpose of protecting us from threat (Ellett & Wildschut, 2012). Ellett and Wildschut (2012 p. 328) describe the main component of paranoia as 'a perception of malevolence from another person or group'. It is a belief that others' hold an intention to harm you in some way, characterised by 'hypervigilance, emotional arousal and selective attention for threat' (Isnanda et al., 2013, p.95).

It has been established that paranoia exists on a continuum much like empathising and systemising (sometimes referred to as the 'paranoia spectrum'), where paranoid thoughts are frequently reported in the healthy population, yet not to the extent that the presence of these paranoid thoughts have any real effect on us or interfere with normal daily living (Freeman et al., 2011). Numerous studies have reported the presence of paranoid thought in neurotypical samples (Fenigstein & Vanable, 1992; Ellett et al., 2003; Combs et al., 2007; Freeman et al., 2010; Ellett & Wildschut, 2012; Isnanda et al., 2013; Moritz & Van Quaquebeke, 2014), so much so that it is thought that paranoia is frequently prominent in around one third of the healthy adult population (Freeman, et al., 2011). In a study using healthy university students, Ellett, et al. (2003) report 153 out of the 324 participants experienced paranoid thoughts directly related to the notion that other people

had intentions to harm them. This led to the presumption that paranoid thinking should be considered a 'common human experience' (p. 425).

Paranoia at a non-clinical level varies in degree (Combs et al., 2007), and the presence of paranoia may include experiences of 'self-centred thought, suspiciousness, assumptions of ill will or hostility' (Fenigstein & Vanable, 1992, p. 130). However, at a clinical level, paranoid thoughts often lead to diagnosis of schizophrenic conditions and are usually resistant to treatment intervention (Bucca, 2012). When paranoid thoughts are present in clinical samples, patients are often out of touch with reality; fiction and reality have become blurred and the person is unable to distinguish between the two (Isnanda et al., 2013).

The present study takes the novel approach of investigating the positive symptoms of schizotypy individually, with a specific focus on paranoid ideation (PI) in relation to the E-S and IBT theories. The purpose of this investigation is to determine if a more accurate characteristic of the EFB could possibly be associated with PI, rather than the broader notion of collective positive schizophrenia, which has limited support (Crespi, 2010; Russell-Smith et al., 2011; Dinsdale et al., 2013). The three qualitative components of paranoia that will define the way the present study investigates PI are as follows, (i) that it is interpersonal i.e. that the thoughts are usually involving others in relation to oneself (ii) that the perception of threat is from external sources i.e. that the threat comes directly from another person/object (iii) that thoughts are ambiguous in relation to others actions, for instance uncertainty or suspiciousness about the intentions of others (Ellett et al., 2013). Paranoia is defined by Freeman, et al. (2008, p. 258) as

an '*unfounded* mistrust'. Therefore, it is important that any amount of paranoia measured has not been subject to any form of induced manipulation, i.e., experimental tasks involving the researchers purposefully manipulating a situation where you would reasonably expect a person to experience paranoid thoughts (Freeman et al., 2008; Ellett & Wildschut, 2012; Ellett et al., 2013).

8.1.2 Jumping to Conclusions

A linear relationship exists between paranoia and jumping to conclusions (JTC) bias (Freeman, 2007). Freeman (2007) and Freeman et al. (2002) discuss the well-replicated finding that persons who experience persecutory delusions also demonstrate poor logical reasoning bias, in that they are quick to make decisions based on very limited information.

To investigate this, previous studies have used the 'beads task' (Huq et al., 1988). The 'beads task' measures probabilistic logical reasoning where participants are presented with two transparent jars of beads, the beads are organised into the jars using a ratio formula, usually 60:40 (difficult) or 85:15 (easy) (for example using the 60:40 ratio, one jar contains 60 blue beads and 40 red beads and vice versa in the other jar). Beads are then drawn (by the researcher) out of sight of the participant, one at a time from *one* of the jars and shown to the participant. The participant has to decide which jar the beads have been drawn from, using the beads as evidence and requesting as many beads as they feel necessary in order to make their decision. Whilst the participant is under the impression that the aim is to guess correctly, the real measure of the task is to see how many beads the participant requests

before making the decision. The usual measurement suggests that participants who offer a decision about which jar the beads are coming from after having only seen two (or fewer) beads, are deemed to have a JTC bias (Jänsch & Hare, 2014). This is because an accurate *informed* decision cannot be made using only one or two beads; a reasonable decision would be made after seeing more than two beads from the jar. Using this task, it has been found that patients with psychosis make decisions based on only one or two beads 50-60% of the time, compared to 20-30% in control samples (Lincoln et al., 2010). Interestingly it has also been identified that the JTC bias is also present in healthy 'psychosis prone' samples (using self-report measures). This links in with the notion of the paranoia continuum, in that paranoid ideation exists in the healthy populations as well as at a clinical level (McKay et al., 2006; Freeman et al., 2008; Lincoln et al., 2009).

The significance of employing tasks such as the beads task to investigate PI comes from the premise that those who demonstrate JTC bias tend to form *unjustified* assumptions on the basis of inadequate evidence, and this thought process is thought to play a key role in the construction of paranoid thinking (McKay et al., 2006).

In relation to the E-S theory, adolescents with ASD have been found to require more information before decision making (measured using the beads task) compared to controls (Brosnan, et al., 2013) suggesting as expected, that those with a tendency to show a preference for systemising would require more evidence based on mathematical probability on JTC tasks. Furthermore, in a healthy population sample, Brosnan et al., (2014) also

found that those who showed a JTC bias also showed greater levels of empathising and less systemising on self-report measures.

8.2 The present study: research aims

The aim of this study is to explore the relationships between E-S bias and paranoid thinking, in a neurotypical sample, using both direct performance and self-report measures. It was hypothesised that participants with a bias towards empathising would (i) score higher on tasks measuring paranoid thinking and (ii) would show greater JTC bias, in line with the theoretical position of the ASD - schizophrenia continuum.

8.3 Methods

8.3.1 Participants

A total of 57 participants from the University of Bradford were recruited to take part in the study. The sample consisted of 45 females (78%) and 12 males (21.1%). All participants where over the age of 18 years and the mean age of the sample was 26 years (±SD = 8.24, range = 32). Inclusion criteria specified that participants must have (i) no clinical diagnosis of mental illness or neurological disorder (ii) normal or corrected to normal vision (iii) a fluent understanding of English language. Furthermore, because of the nature of the study, those who reported using cannabis on a regular basis were excluded. This is due to the interference effects of cannabis on heightened PI (Skosnik et al. 2001; Fridberg et al. 2011; Freeman et al. 2011). Ethical approval was obtained from the Humanities, Social & Health Science Research Ethics committee at the University of Bradford. All participants gave their informed written consent.

8.3.2 Design

A cross-sectional, primarily correlational design was employed, concerned with exploring the relationships between the E-S theory and paranoid thoughts at a healthy population level. Post data collection, the sample was also categorised according to E-S bias using *z* scores, which created two groups- higher EQ/lower SQ (E bias) and lower EQ/higher SQ (S bias). This made for analysis in way of an independent groups design. The researcher administered the battery of tests in random order for the purpose of controlling for order effects.

8.3.3 Materials

The Empathising Quotient (EQ) - The short form (EQ-S) version of the EQ (Wakabayshi et al., 2006b) measuring self-report cognitive and affective empathy was administered. See section 6.3.3.2 and appendix 1.

The Systemising Quotient (SQ) - The short form version (SQ-S) of the SQ was employed, which measures self-reported 'everyday' systemising ability (Wakabayshi et al., 2006b). See section 6.3.2.2 and appendix 2.

Paranoia Checklist - PI was assessed using the Paranoia Checklist (Freeman et al., 2005). An example statement would be *'people deliberately try to irritate me'*. Each of the 18 items are rated by three dimensions of paranoid ideation: (i) frequency *(How often have you had the thought?)*, (ii) conviction *(How strongly do you believe it?)* and (iii) distress *(How upsetting is it for you?)* on a five point Likert scale. Lincoln et al. (2009) reports excellent internal consistency of the scale (Cornbach $\alpha > .90$) and the scale has shown to be sensitive to PI in healthy population samples (Freeman et

al., 2005). The score can be determined by the summing of individual scores on each scale with a maximum score of 90. Higher scores indicate higher levels of PI. Full version is available in appendix 8.

Over-attribution image battery (OAIB) - The OAIB was developed by the researcher as a measure of unfounded 'uneasiness'. The OAIB aimed to measure subtle paranoid thoughts in neutral conditions. Carried out using a custom designed online survey program (www.onlinesurveys.ac.uk), participants were presented with 10 non-threatening still colour images (13 x 17cm) on a computer screen (See Figure 11). Participants were instructed to *'consider this image and place yourself within this picture'* and to rate the level of uneasiness that they perceive within the context of the image, using a scale response format of (1) not uneasy at all, (2) slightly uneasy, (3) somewhat uneasy, (4) very uneasy, (5) extremely uneasy'. Higher scores indicated a higher level of unfounded unease, maximum score was 40.

All images originated from the researcher's personal collection of photographs (with permission from any persons appearing in the image granted). It was proposed that the benefit of choosing images from this collection was that the researcher was witness to the 'real life' context of these photographic scenes, therefore providing a sense ecological validity when measuring the level of unease the participant infers from the context of the image. All images were presented to a judging panel of three people before being included in the battery of images for the purposes of assessing suitability. The judging panel was asked if they felt that there was any obvious suggestions of induced uneasiness; none were reported on the selected images.

Fig. 11: Example image from the OAIB (not to scale)

The Sweet Jar Task (SJT) – The SJT is a 'jumping to conclusions' reasoning task. The original task is known as the 'beads task' (Dudley et al., 1997). A modified computer version created by the researcher was employed in this study, as the original task was financially unviable. Participants were exposed to two transparent jars of sweets, each containing two different types of sweets (e.g., red and blue bonbons) in an opposite but equal ratio system of 60:40 (Dudley et al. 1997) (e.g., 60% red bonbons and 40% blue bonbons and the reverse in the other jar- see Figure 12). The participant was informed that the aim of the task is for them to make a decision as to which jar (jar one *or* jar two) the sweets were been drawn from. They were advised that they could request to see as many sweets as they desired from the jar in order to make a decision and that each sweet they saw was returned to the jar after each withdrawal. A memory aid was present, in the form of a laminated sheet placed on the desk, which served to remind the participant which ratio of red and blue sweets was assigned to each jar, also the

computer was set up to allow the participant to keep track of the previous draws of sweets by displaying them in the top corner of the screen (this was for the purpose of eliminating working memory pressures). Sweets were then drawn one at a time, from one jar only (out of sight of the participant) and then presented to the participant. The variable here was the number of pieces of information (sweets) requested before a decision was made, regardless of a correct or incorrect guess. The JTC bias was determined to be evident if only two or fewer sweets were requested before a decision was made. Three trials (of a maximum offering of 20 sweets per trial) were completed by the participant.

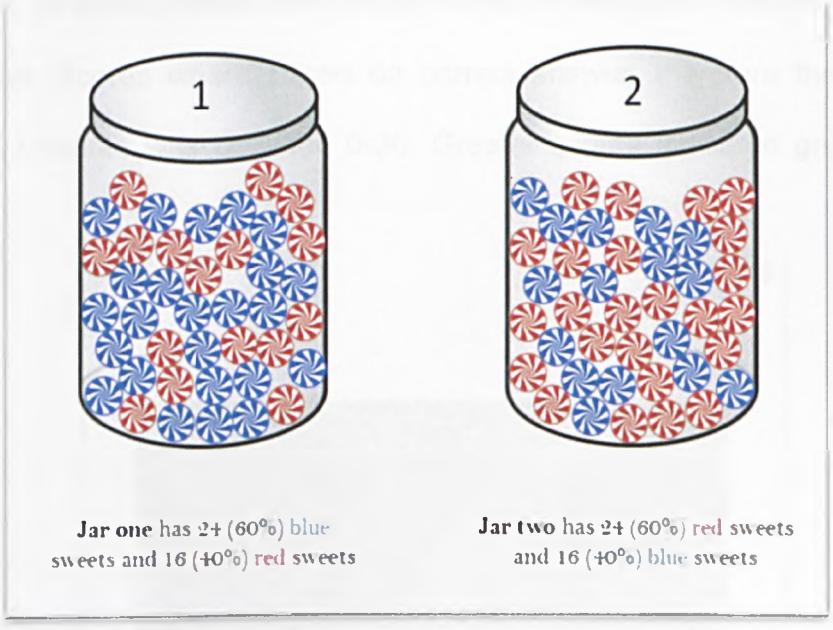


Fig. 12: The sweet jar task

The Revised Reading the Mind in the Eyes Task (RMTE) – The pencil and paper version of the RMTE was administered as a measure of subtle differences in social intelligence for healthy adults with normal intelligence. It is described as an advanced test of ToM or an understanding of the 'language of the eyes' (Baron-Cohen et al., 2001). The task comprised of 36 trials, each trial comprised of a 16 x 12cm black and white photograph of either male or female eye region (from mid-nose to eyebrow). Surrounding the photograph are four emotion adjectives (see Figure 13 for an example). Participants were instructed to choose one of the four words they thought best represented the mental state of the person in the picture. Participants read aloud their answer to the researcher. They were also provided with a glossary of words, which they could consult if they were unsure of a word definition. Scores where based on correct answer, therefore the possible range of scores was between 0-36. Greater scores indicated greater ToM ability.

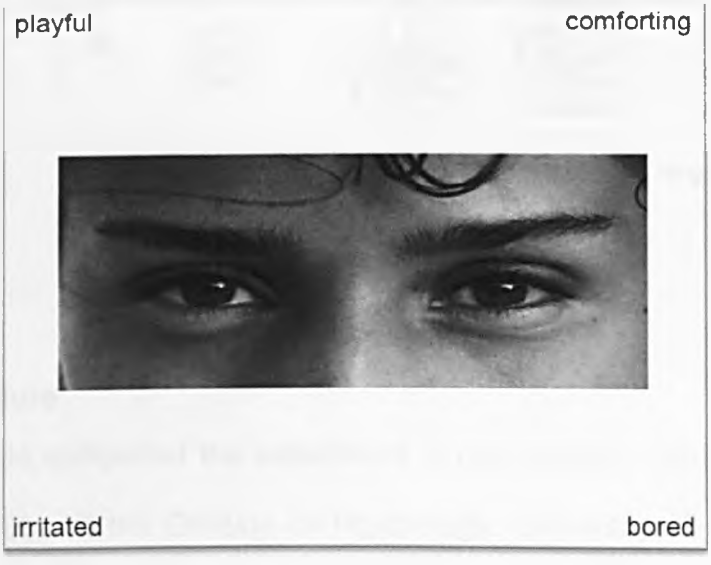


Fig. 13: An example trail from the RTME task. Correct response is 'playful'.

Embedded figures task (EFT) - A pencil and paper variant of the embedded figures task was administered, consisting of five trials (www.brainwaves.com/Puzzles_Tests.html). Each trial had a target image (e.g., A. in Figure 14) and two other images (1. and 2. in Figure 14), one of which contained the embedded figure. The EFT measures attention to detail with a focus on global versus local (visual) processing ability, theoretically relevant for the present study as higher systemisers tend to show greater local processing ability (Auyeung et al., 2012). Greater local processing ability would aid faster location of the embedded figure as a preference for focusing on detail is involved. The aim of the task was to locate the embedded figure within one of the two images and outline it with a pen provided. The target figure was only present in one of the two images. Scores were based on the number of correct answers within a 5 minute time limit.

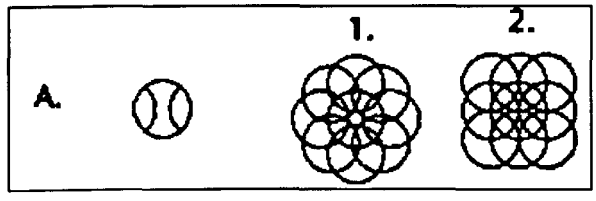


Fig. 14: A trial from the embedded figures task. The target figure (image A) is located at the centre of image 1.

8.3.4 Procedure

All participants completed the experiment in one session, carried out at the psychology labs at the Division of Psychology, University of Bradford. On arrival participants were required to read the information sheet pertaining to the study and if happy to proceed, then signed the consent form. The battery of measures was then administered in random order. Sessions lasted

between 30 and 40 minutes and no participants requested a break during the sessions.

8.4 Results

The aim of this study was to explore the relationship between empathising, systemising, paranoid ideation and JTC bias in a neurotypical sample (*n*= 57). Analyses were carried out using the Statistic Package for the Social Sciences (SPSS) version 20.0 using score data. Ranges, means and standard deviations for all measures can be found in Table 13.

Table 13: Means and standard deviations for scores on all variables

Measure	*n*	Mean	±SD	Min-Max possible score
EQ	54	26.19	8.20	0-44
SQ	55	20.90	8.92	0-50
PCF	57	22.37	5.86	0-90
PCC	56	24.02	6.53	0-90
PCD	57	32.91	14.57	0-90
SJT	50	12.54	6.44	1-60
OAIB	54	6.07	3.72	0-40
RTME	56	24.39	3.74	0-36
EFT	52	4.16	0.81	0-5

Note: EQ= empathising quotient; SQ= systemising quotient; PCF= Paranoia checklist (frequency); PCC= Paranoia checklist (conviction); PCD= Paranoia checklist (distress); SJT= Sweet jar task; OAIB= Over-attribution image battery; RTME: Reading the mind in the eyes; EFT= Embedded figures task

The results were submitted to a general linear model (GLM). Multivariate analysis revealed that there was no significant main effects of gender on any of the variables (p>0.05), although, a trend was evident for EQ scores (F (1, 42) = 3.12, p = 0.08).

8.4.1 Main effects of E-S bias

The sample was categorised into groups of empathising (E bias) (E>>S) and systemising (S bias) (S>>E). To assess the tendency for participants to show bias towards an empathising or systemising cognitive profile, EQ and SQ scores were firstly converted to z-scores and the difference between the scores were calculated. A score of >0.00 specified a bias for a systemising profile. A score of <0.00 suggests a bias for an empathising profile. Table 14 shows the distribution of participants into the two categories.

Table 14: Distribution of participants into E-S bias by biological sex

Biological Sex	Brain Type	Total	%
Male n = 11	Empathising bias (E>>S) Systemising bias (S>>E)	3 8	27.3 72.7
Female n = 43	Empathising bias (E>>S) Systemising bias (S>>E)	24 19	55.8 44.2

Note: as expected, a higher number of males showed a pattern of bias towards systemising. The pattern was less obvious in females, where only slightly over half showed a pattern towards empathising.

MANOVA revealed that there was a significant main effect of E-S bias on performance on the SJT where the group with a cognitive bias towards empathising (E>>S) took significantly less turns on the SJT (M=10.89, ±SD=5.22) than the group with a bias towards systemising (S>>E) (M=15.00,

±SD=6.98) (F (1, 47) = 5.79, p<0.05). There was no significant main effect of E-S bias on the RTME, EFT, PCF, PCC, PCD or the OAIB (*ns*).

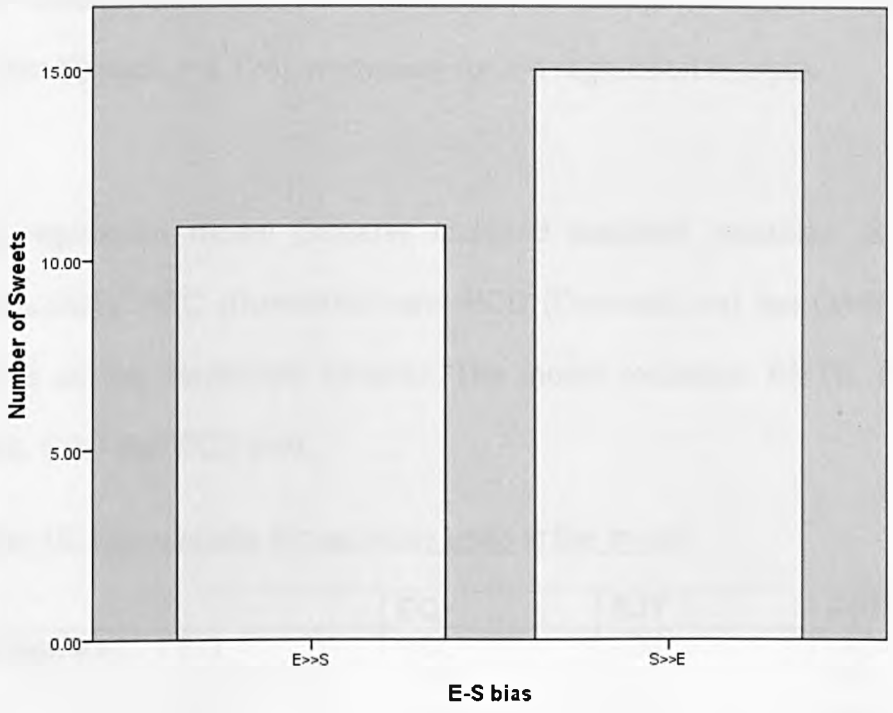


Fig. 15: Bar graph demonstrating that those who showed greater bias for empathising (E>>S) took significantly less turns on the SJT than those who showed a bias for systemising (S>>E)

8.4.2 Regression analysis exploring empathising and systemising as separate entities

8.4.2.1 Empathising

As a fundamental assumption of the E-S theory, regardless of the hypothesis being explored in the present study, empathising and systemising should theoretically share a trade-off relationship, however no significant association was evident (r = .19, p>0.05). Therefore, in order to explore the relationship between paranoid ideation and empathising and systemising as separate

entities, regression analysis was carried out. Data was examined for tenable assumptions, all were met including generalisation assumptions [variance inflation factor (VIF) = 1.002], scatterplots demonstrated an even disperse of dots around zero and the assumption of independent errors were met (Durbin-Watson = 2.126), necessary for the regression analysis.

The regression model primarily included predictor variables: SJT, PCF (Frequency), PCC (Conviction) and PCD (Distress) and the OAIB with EQ scores as the dependent variable. The model excluded: RMTE, EFT, SQ, OAIB, PCC and PCD (*ns*).

Table 15: Correlations for variables used in the model

		EQ	**SJT**	**PCF**
Pearson's r	EQ	1.00	-.34*	-.40*
	Sweet Jar	-.34*	1.00	.05
	PCF	-.40*	.05	1.00
N	EQ	49	49	49
	Sweet Jar	49	49	49
	PCF	49	49	49

*p<0.01 (one-tailed)

Note: table 15 demonstrates a modest correlation between the EQ and the Sweet Jar task, also the EQ and the Paranoia Checklist (Frequency). No correlation was found between the Sweet Jar Task and the Paranoia Checklist (Frequency), satisfying the assumptions of multicollinearity.

Table 16: The unstandardized and standardised regression coefficients for the predictor variables

	B	SE B	β
Constant	43.11	4.36	
Sweet Jar Task	-0.41	0.16	-.32*
PCF	-0.52	0.17	-.39**

Dependent variable: EQ *p<0.05, **p<0.01

The enter method was used in the analysis.

Overall, the regression model was able to significantly predict EQ score (F (2, 46) = 8.30, p<0.001). The SJT significantly predicted EQ score (t (46)= -2.53, p<0.01, 95% *CI* -.73 - -.08), explaining 12% of the score variance (R^2 =.12, Adjusted R^2 = .10). Adding the Paranoia Checklist (Frequency) (t (46)= -.3.07, p<0.001 95% *CI* -.87 - -.18) to the model then explained 27% of the variance (R^2 =.27, Adjusted R^2 = .23). Results demonstrated that as EQ score increased by 1 point, scores on the PCF decreased by 0.52 points (β = -0.52), also, as EQ scores increased by 1 point, turns on the SJT reduced by 0.41 turns (β = -0.41).

Note: The three dimensional measurements of the Paranoia Checklist were positively correlated; PCF (Frequency) score was positively correlated with PCC (Conviction) score (r = .60, p<0.001) and PCD (Distress) score (r = .29, p<0.05), PCC (Conviction) score was also positively correlated with PCD (Distress) score (r = .28, p<0.05). These results suggest, interestingly, that although the different dimensions of the Paranoia Checklist share a positive relationship, it was only PCF (Frequency) scores that were able to predict EQ

scores. No relationships between the RTME task and Paranoid ideation were evident (*ns*).

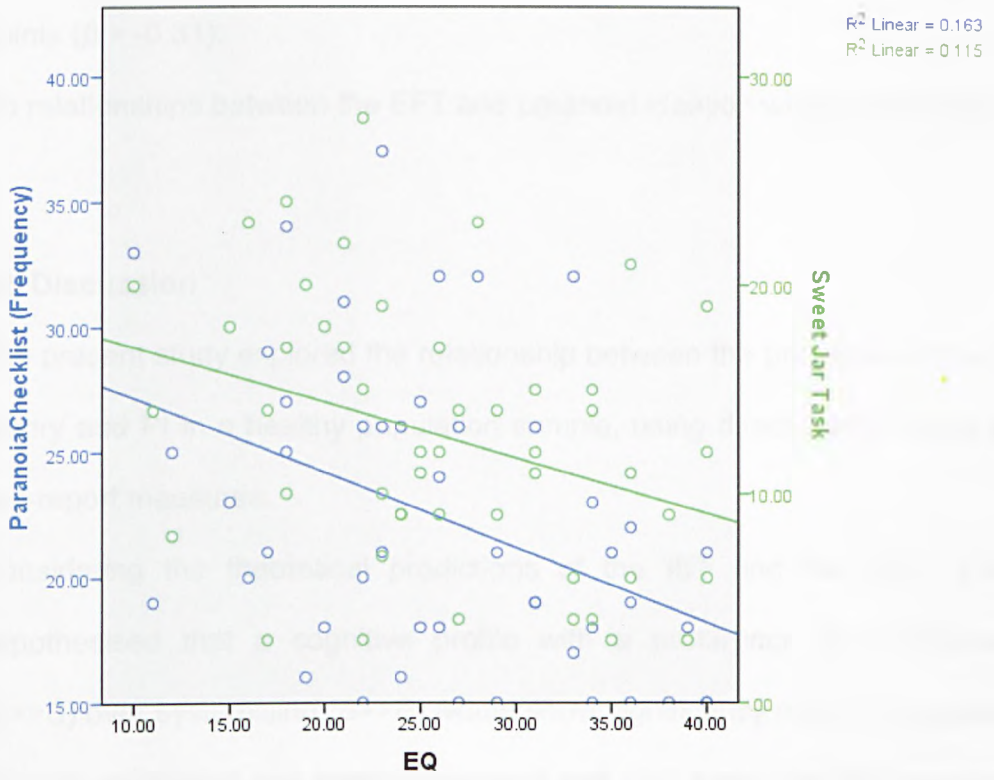


Fig. 16: Scatterplot showing PCF scores decreasing as EQ scores increased and SJT scores increasing as EQ scores decreased

8.4.2.2 Systemising

The regression model primarily included predictor variables: SJT, PCF (Frequency), PCC (Conviction) and PCD (Distress) and the OAIB with SQ scores as the dependent variable. The model excluded all variables except the OAIB (*ns*).

Pearson's correlation coefficient revealed a negative association between SQ score and feeling of unease on the OAIB task (r = -.31, p<0.05). The model

was significant (F (1, 44) = 4.51, p<0.05). OAIB was able to significantly predict SQ score (t (44) = -2.12, p<0.05, 95% CI -1.25 - -.03) explaining 9% of the score variance (R^2 =.09, Adjusted R^2 =.07). Results demonstrate that as SQ score increased by 1 point, scores on the OIAB decreased by 0.31 points (β = -0.31).

No relationships between the EFT and paranoid ideation were evident (*ns*).

8.5 Discussion

The present study explored the relationship between the principles of the E-S theory and PI in a healthy population sample, using direct performance and self-report measures.

Considering the theoretical predictions of the IBT and the EFB, it was hypothesised that a cognitive profile with a preference for empathising (E>>S) over systemising (S>>E) would show significantly more PI (measured by both self-report and direct measures) and JTC bias. The null hypothesis was retained in terms of paranoia; no relationship between E-S bias and paranoid ideation was evident. However, performance on the JTC task was significantly affected by E-S bias, with greater empathisers and lesser systemisers showing greater tendency to JTC. This demonstrated that those who presented with an empathising bias required less evidential information before making a decision, than those who presented with systemising bias, supporting the expected trend theorised by the IBT and supportive of previous results found by Brosnan et al. (2011). These findings offer support for the notion of an association between JTC bias and the 'female brain'.

8.5.1 The issue of the presumed 'trade-off'

It is important to be reminded at this point that research questions surrounding the E-S theory rely on the principle assumption that empathising and systemising cognitive processes share a trade-off relationship i.e. that those who demonstrate a greater ability in either empathising or systemising will show a deficit (of the same extent) in either empathising or systemising respectively.

Although not the main investigation in the present study, the results obtained here add to the growing number of research studies that have failed to find any evidence of this theoretical trade-off presumption, and support the notion that these two mechanisms work independently of each other (Baron-Cohen et al 2003; Carroll & Yung 2006; Russell- Smith et al., 2013; Jones & Lesk, 2013). Further, a trend in the way of a small positive relationship was found in the present study between EQ and SQ scores (r = .19, p = 0.08, n = 57). This is problematic for the fundamental presumptions of the E-S theory. The 'trade-off' relationship between E and S is discussed in detail in Chapter 10. Additionally, direct measures of empathising and systemising failed to be associated with self-report measures of empathising and systemising. Therefore, it could be argued here that this creates a fundamental problem with exploring PI, or indeed any other cognitive variable in relation to the E-S theory, in that there is very limited evidence of an underlying relationship between empathising and systemising networks.

8.5.2 Empathising and paranoia

Due to the seemingly complex nature of relationship between empathising and systemising (Chapter 10), it is important to explore the two cognitive dimensions as separate entities, under the presumption that they work independently of each other, in line with the results of this study.

Results demonstrated that both the frequency that a person experiences paranoid thoughts and JTC bias could significantly predict empathising ability. Collectively they were able to explain over a quarter of the score variance. What is most interesting here is that these results propose that *greater* levels of empathising were predicted by *lesser* frequency of paranoid thoughts, indicating that as someone reports greater empathising ability, they also report experiencing paranoid thoughts less frequently. This goes directly against what would be expected by the ASD - schizophrenia model. It is also interesting to consider that the results demonstrated that although the three dimensions of PI (frequency, conviction and distress) positively correlated with each other, it was only the frequency of experience of paranoid thoughts that significantly predicted the variance in empathising score. We can interpret from this, that whilst higher empathisers are experiencing significantly lesser levels of paranoid thoughts, their belief in the conviction of these thoughts, and the distress caused by these thoughts is not a factor in the relationship between paranoid ideation and empathising.

8.5.3 Empathising and jumping to conclusions

Even more curious, empathising was also predicted by *lesser* requests for sweets on the JTC task, meaning that those who reported greater levels of empathising ability were quicker to 'jump to conclusions'. This finding is in

line with the predictions of the ASD - schizophrenia continuum and offers support for the notion of an association between JTC bias and the 'female brain'.

8.5.4 Paranoid Ideation, jumping to conclusions bias and systemising

Neither PI nor JTC bias shared a relationship with systemising. This leads us to conclude that these findings do not lend themselves fully to the search for an accurate characterisation of the EFB. If this were the case, we would expect to see systemising show the opposite relationship that empathising shares with PI or JTC bias. It is suggested here that in a broader sense this result adds more weight to the growing number of studies that call into question the fundamental assumptions and indeed the validity of the E-S theory.

However, what is interesting, is that results demonstrated that levels of systemising could be predicted by the level of 'unease' that was experienced on the OAIB task, in that, as levels of systemising decreased, levels of the unease experienced by the respondent increased, this finding has the potential to be supportive of the trend expected by the IBT, yet no relationship was evident between empathising and the OAIB. From this, we are unable to offer any evidence that solidly supports the IBT continuum. However, further in-depth investigation into paranoia measured in this format may be able to shed further light on this potential relationship with systemising.

8.5.5 Considerations

Considering the findings of the present study as a whole, there are a number questions that should be reflected upon. Primarily, let us consider that JTC bias and the manifestation of paranoid thoughts share a positive relationship, (Freeman et al., 2002; Freeman et al., 2005; Freeman 2007; Moritz et al., 2012), yet we observed no relationship between the two phenomena in this sample. Nonetheless, both were able to significantly predict empathising score. So, what does this mean for the hypothesised relationship between the IBT, E-S theory and paranoia? It is important to reflect on the sample; was this pattern evident because we tested the neurotypical population and would not expect a high frequency of paranoid thoughts but expect to find JTC bias? However, a problem with this consideration is two-fold; firstly as previously alluded to, PI *was* evident in the sample and was significantly associated with *lesser* empathising. Secondly, PI is widely accepted to be experienced at a healthy population level (Garety et al., 2005; Buchy et al., 2007; McKay et al., 2006; Freeman et al., 2008; Lincoln et al., 2009) and due to the *continuum* nature of paranoia and indeed the E-S and IBT models, these patterns of cognitive markers should be evident in the neurotypical population (albeit to a lesser extent), in the *same direction* as they would be at the extreme ends of the continuum.

The finding that greater empathising was predicted by lesser frequency of paranoid thoughts (along with the finding that E-S bias groups did not differ in their score of paranoid thoughts) does not support the EFB hypothesis of the present study. Or in a more broader sense, the theoretical predictions of the IBT and instead adds weight to the growing body of experimental evidence

which indicates components of positive schizotypy are not associated with a E>>S cognition as is theorised by the IBT (Russell-Smith et al., 2011). However, in terms of the E-S theory, it could be argued that this finding reasonably supports Baron-Cohen's (2003) argument that there is unlikely to be any pathological consequence of the 'female brain' or EFB, however this argument would be a stronger in its support for Baron-Cohen (2003) had empathising and systemising demonstrated a negative relationship.

We can consider in light of this pattern of results that what was found here was support for research that suggests autistic traits and schizotypy overlap, or frequently occur together (Petty et al., 1984; Starling & Dossetor, 2009; Skoukaskas & Gallagher, 2010; Russell-Smith et al., 2011; Abu-Akel et al., 2015). ASD tendencies present with a lack of, or a deficiency in ToM ability (Baron-Cohen, 2010), and due to this, are paranoid thoughts more frequent owing to a deficiency of social intuition, in that an ASD cognition has greater difficultly in reading the intentions of other people, therefore experiences a heightened level of paranoid thoughts?

Indeed, our results are somewhat in line with Spain et al. (2016) who conducted a systematic review looking at paranoia in relation to ASD and found individuals with ASD had consistently greater levels of paranoia than non-clinical controls *and* psychosis. Similarly, Blackshaw et al. (2001) who found that PI was significantly greater in Asperger's syndrome when compared to controls. Blackshaw et al. (2001) go on to suggest that although this pattern was unexpected, perhaps paranoia associated with ASD has a different aetiology than that of schizophrenia. Our results here provide support for this line of thinking.

It is suggested here that future studies should look at these patterns of cognitive markers in clinical samples. Would the same pattern be evident supporting the continuum model, or would we find non-linear results in the healthy population and clinical samples, which may add further convincing evidence to suggest the continuum model of the IBT requires revision.

Our results also pertain to the broader question of the reliability of self-report data; whilst we see novel, thought-provoking results in relation to E-S bias, we must keep in mind that the E-S bias was measured solely by self-report measures, and importantly, no relationships were found between self-report empathising, systemising and direct performance measures of these cognitive dimensions. Future research should aim to clarify the reliability of the EQ and SQ self-report measures as they relate to direct measures in order to offer more robust conclusions.

8.5.6 Limitations

Limitations of the present study should be considered. Firstly, the sample was heavily bias towards females, and whilst gender was found to have no main effect on any variables measured, a more balanced number of males and females would have been preferable due to the close links between 'brain type' and biological sex (Baron-Cohen, 2003). Secondly, results reported here in relation to the OAIB should be interpreted as pilot study findings, the measure, whilst tested for validity using a judging panel to assess the images used in the task, has not been previously employed in any studies and no reliability data is available.

8.5.7 Overall conclusion

The present study failed to find any support for the association of the 'female brain' profile and self-report paranoia. However, results indicated an association between greater empathising alongside lesser systemising and a JTC tendency, indicating a potential relationship between the EFB and JTC bias. More fundamentally, the results fail to provide support for the E-S theory's presumption that empathising and systemising share a trade-off relationship (discussed in more detail in Chapter 10), therefore empathising and systemising where examined in isolation from each other. Results suggested interestingly, that *higher* levels of empathising were associated with lesser frequency of paranoid thoughts which directly contradicts the theoretical concepts of the IBT and therefore fails to provide any evidence of an accurate characterisation of the EFB, but suggests a relationship between cognitive empathy and jumping to conclusions on logical reasoning.

8.6 Chapter summary of key points

- There is a lack of experimental support for the IBT's ASD – schizophrenia continuum, could a more accurate characterisation of the type E brain involve paranoia phenotypes, rather than a collective set of positive symptoms of schizotypy?
- It seemed rational to consider, based on the results of the previous chapter, that it is likely that *specific* phenotypes may share *specific* types of relationships.

- E-S bias was investigated in relation to paranoid ideation and jumping to conclusions bias using a novel task paradigm in a non-clinical sample.
- Results demonstrated that performance on the JTC task was significantly affected by E-S bias, with **greater empathisers and lesser systemisers showing greater tendency to jump to conclusions**.
- These findings established that, those who presented with an empathising bias required less evidential information before making a rational decision, compared to those who presented with systemising bias, supporting the expected trend theorised by the IBT (Badcock & Crespi, 2006).
- It was concluded that there is evidence of a link between positive schizotypy and the 'female brain', but only when considering ***individual phenotypes*** of schizotypy, as clearly, all components of positive schizotypy cannot be accurately associated with the female brain.

Chapter 9

How does the AQ relate to dimensional schizotypy in a neurotypical sample?

9.1 Introduction

The study sought to assess the specific nature of the relationship between autistic traits and schizotypy in an adult neurotypical sample. The primary aim of the study was to examine the theorised diametrical model of ASD and schizophrenia proposed by the IBT (Badcock & Crespi, 2008), by assessing the relationship between self-reported autistic traits and dimensional schizotypy.

To recap: the IBT hypothesises that ASD and schizophrenia exist on a diametric continuum as one connected disorder, rather than two distinct conditions (Badcock & Crespi, 2006,2008). This diametric model, Badcock and Crespi (2008) suggest, is due to a 'tug of war' in gene imprinting which results in either a balance or a significant bias in either maternal or paternal gene imprinting and the subsequent gene expression during neurodevelopment. This process is speculated to have considerable organising influence on the "social brain", resulting in the manifestation of a cognitive profile which they term; *mechanistic*, *balanced* or *mentalistic* brain types (Badcock, 2011) (see Figure 17).

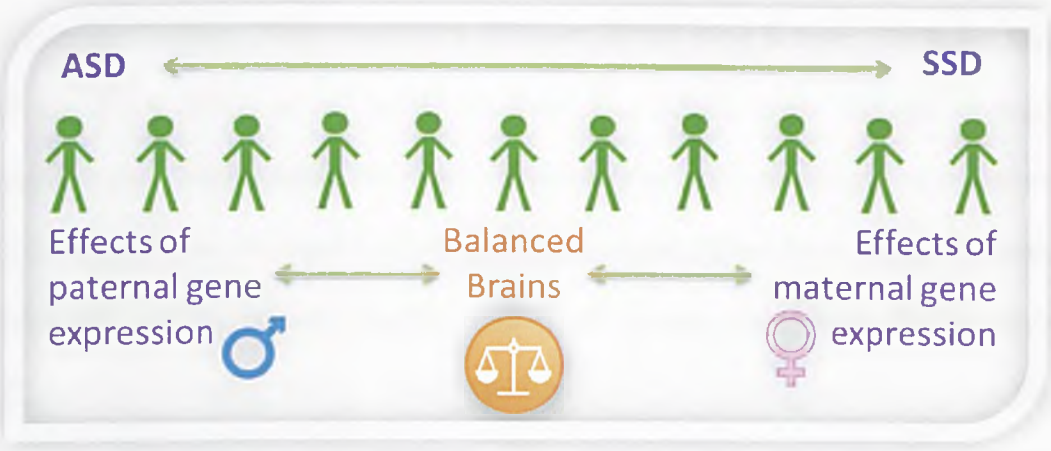


Fig. 17: The IBT continuum model ©individual images from clipart

The notion that ASD and schizophrenia are closely related disorders is not a new philosophy. Over 100 years ago, the works of psychiatrist Eugen Bleuler (1857-1939) and later Leo Kammer (1984-1981) discuss autism as a sub-set of schizophrenia in the childhood years. Later autism was re-defined by Kammer as a separate disorder (Crespi, 2010), and the distinction between the two has remained the same going forth into modern psychiatry. Nowadays, if a clinical patient is thought to be presenting with positive symptoms of psychosis and ASD at the same time, a diagnosis of co-morbidity of ASD and schizophrenia is recorded, maintaining the distinction between the two phenomena (Keller et al., 2016).

Rutter (1996) resolved that ASD and schizophrenia are separate and unconnected both genetically and biologically, and Tantum (1991) comes to the same conclusions from a behavioural perspective. However, more recent research unconnected from the IBT investigation has suggested that there is evidence to indicate that the two conditions do share biological, genetic and

psychological similarities (Adbi & Sharam, 2004; Sporn et al. 2004; Burbach & van der Zwaag, 2009; Craddock & Owen, 2010; King & Lord, 2011; Mealey et al., 2014; Moos et al., 2016; Pediaditakis, 2016). Even though findings have been inconsistent in their conclusions, the preferred connection between the two disorders is one which 'overlaps' rather than a diametrically opposed link, as collectively the majority of research supports this position (Craddock & Owen, 2010). Spek and Wouters (2010) suggest that given the genetic overlap between ASD and schizophrenia, it is probable that the two disorders will naturally present with similar symptomology.

The major difference between the two, is that ASD is usually diagnosed in early childhood, usually around the age of three years, whereas schizophrenia is diagnosed after the age of 18, when personality has fully developed (Fitzgerald, 2011). Because it has been established that many symptoms and cognitive deficits can overlap, including, ToM (Pilowsky et al., 2000), central coherence (Happé & Frith, 2006; Uhlass & Silverstein, 2005), executive function (Kerns et al., 2008), and social skills deficits (Russell-Smith et al., 2011), Hurst et al. (2006) points out that clarification of the affiliation between ASD and schizophrenia is vital and should aim to make the diagnostic process more informed and much less confused (Luciano et al., 2014).

Research by Hurst et al. (2006) has shown that the obvious difference between ASD and schizophrenia involves the presence of positive symptoms of psychosis [hallucinations, delusions, magical thinking (Mason et al., 2005)]. In that, the negative symptoms of psychosis [lack of affect, social withdrawal, avoidance of intimacy [Mason et al., 2005)] are often present in

ASD, whereas the manifestation of positive symptoms of schizophrenia are not usually reported (Petty et al., 1984; Dyken et al., 1991; Konstantareas and Hewitt, 2001; Sheitman et al., 2004; Spek & Wouters, 2010). It is this notion that is the focus of the IBT; it suggests that whilst an overlap of negative traits is apparent, it is plausible that a diametric relationship exists between positive psychosis and ASD (Badcock & Crespi, 2008). The main reasoning for this postulation comes from the observation that whilst ASD and schizophrenia share diagnostic criteria involving deficits in social intelligence, the IBT observes that they are deficits of extreme difference. In so much that, ASD involves an *absence* of ToM whereas schizophrenia involves an *overly active* ToM to the point of dysfunction[20]. Signifying that the autist simply lacks the manifestation of ToM processes and is therefore unable to comprehend social and emotional phenomena; whereas the positive schizophrenic experiences ToM processes so acutely attune that they distort reality (Badcock & Crespi, 2006).

Badcock and Crespi (2006) endorse that because the theory is a *continuum* of cognitive profiles, the logical starting point in terms of exploratory research should be at a general population level. Reasoning for this is two-fold; firstly, because everyone can be placed on the continuum, and that both ASD and schizophrenia exist on a spectrum of severity from trait to clinical levels, patterns in sub-clinical populations are detectable (Abu-Akel et al., 2015). Secondly, non-clinical population level research omits the effects of drug intervention which is likely to be administered as treatment in clinical

[20] ToM is thought to be overly active in only the positive expressions of schizophrenia, namely paranoid ideation, hallucinations and delusion (Badcock & Crespi, 2006).

populations (pertaining more to schizophrenia) which therefore controls for pharmacological influence.

Research into the IBT is in its infancy. Crespi (2010) offers that recent tests of data from seven copy number variant loci has offered statistical support for the diametric link between the two disorders (see Craddock et al., 2009). At a behavioural level, only a few studies have explored this theory directly; Brosnan et al. (2010) found that mentalising profiles score greater on positive schizotypy in a female non-clinical sample and Dinsdale et al. (2013) found support for both overlap and a diametric relationship when using principle component analysis to look at different dimensions of ASD and schizophrenia in a large non-clinical sample. Considerable overlap in features that relate to social skills and communication was evident, alongside an inverse association between the core ASD traits and positive schizotypy phenotypes, providing support for the IBT prediction. However, Rawlings and Locarnini (2008) report a correlation between autistic traits and negative schizotypy, but found no suggestion of a relationship between ASD and positive schizotypy. However, other studies report a positive correlation between autistic traits and negative *and* positive schizotypy, which offers no support to the diametric model proposed by the IBT (Hurst et al., 2006; Russell-Smith et al., 2011; Mealey et al., 2014). The importance of establishing an accurate account of the relationship between ASD and schizophrenia lays not only in questioning the validity of Badcock and Crespi's (2006) model, but in the importance of using informed criteria for an accurate diagnosis and appropriate intervention for both disorders (Spek & Wouters, 2010).

9.2 The present study: research aims

The present study aimed to contribute to the knowledge of how ASD and schizophrenia are related by quantifying autistic traits and dimensional schizotypy in a neurotypical adult sample and exploring the relationship between the phenotypes. Specifically, it is hypothesised, based on the IBT model that positive schizotypy will share a significant inverse relationship with autistic traits. It is expected that negative schizotypy will share a positive relationship with autistic traits, based on evidence from previous findings (Petty et al., 1984; Dyken et al., 1991; Konstantareas and Hewitt, 2001; Sheitman et al., 2004; Spek & Wouters, 2010). The present study will employ a validated self-report measure of schizotypy which measures not only positive and negative symptomology but will also quantify and consider other components of schizotypy, namely, thought disorder and impulsive behaviours (Mason et al., 2005).

9.3 Methods

9.3.1 Participants

A total of 158 participants took part in the present study. Respondents were recruited from the University of Bradford via email advertisement, and the general population via various social media channels. The mean age recorded by participants was 34 years (±SD= 14.01). Gender was not recorded. Inclusion criteria specified that participants must have (i) no clinical diagnosis of mental illness or neurological disorder (ii) a fluent understanding of English language (iii) must not have taken part in previous studies

conducted by the researcher (due to over exposure effects). Ethical approval was obtained from the Humanities, Social & Health Science Research Ethics committee at the University of Bradford.

9.3.2 Design

A cross-sectional, correlational design was employed, using score data from two self-report inventories measuring ASD and schizophrenia at trait level in healthy population samples.

9.3.3 Materials

The autism spectrum quotient (AQ) – The AQ (Baron-Cohen et al., 2001) is a self-report 50 item forced choice questionnaire designed to measure individual differences in autistic traits in populations of normal intelligence. It measures five major phenotypes of the autism spectrum; poor social skills, poor attention switching, exceptional attentional to detail, poor communication skills, and poor imagination skills (Barnevald et al., 2011). Each item has four possible responses: definitely agree, slightly agree, slightly disagree and definitely disagree. The maximum possible score is 50. A greater score indicates a greater degree of autistic traits. Baron-Cohen et al. (2001) suggests 80% of clinically diagnosed autistic individuals will score over 32, whereas only 2% of controls would score greater than 32, which is used as the cut off figure for clinically relevant ASD. The AQ has previously shown reliability and validity as a diagnostic screening tool (Hurst et al., 2006). The full version of the AQ can be found in appendix 9.

The Oxford and Liverpool Inventory of Feelings and Experiences (O-LIFE) - The O-LIFE (Mason et al., 2005) is designed to measure schizotypal personality; it is specifically employed to measure schizotypy in the healthy populations of normal intelligence. The short form which was employed in this study. See section 7.3.3 and appendix 4.

9.3.4 Procedure

All participants completed the battery of measures online using a specifically designed survey website. Before beginning the battery, respondents were required to read the information sheet pertaining to the study and if happy to proceed, completed the online consent process. The battery of measures was administered in the same order for all participants. Respondents took approximately 40 minutes to complete. Responses from participants who failed to access the entire battery of measures were automatically excluded from the data set.

9.4 Results

The aim of this study was to explore the relationship between ASD and schizophrenia traits by employing self-report inventories in a healthy population sample. Outliers greater than 3.29 were removed and the assumption of normality was tenable. Missing cases were excluded pairwise. Ranges, means and standard deviations for all measures can be found in Table 17.

Table 17: Means and standard deviations for scores on all variables

Variable	n	M	±SD	Min-Max possible score
AQ	129	18.22	6.68	0-50
O-LIFE	149	15.56	7.29	0-43
UnEx	149	4.78	2.77	0-12
CogDis	153	4.88	3.03	0-11
InAn	145	2.57	2.28	0-10
NonCon	150	3.21	2.12	0-10

Note: AQ= autism quotient; O-LIFE= Oxford and Liverpool inventory of feelings and experiences (total score); UnEx= O-LIFE sub group unusual experiences; CogDis= O-LIFE sub group cognitive disorganisation; InAn= O-LIFE sub group introvertive anhedonia;:ImpNon= O-LIFE sub group impulsive nonconformity

Four participants (3.1%) scored greater than the cut off (>32) point on the AQ for clinically relevant autistic traits.

9.4.1 Regression analysis of the AQ and O-LIFE

Pearson's correlation coefficient revealed a positive moderate correlation between overall scores on the AQ and O-LIFE (r = .52, p<0.001). A simple linear regression was calculated to predict AQ scores based on O-LIFE scores (n = 114). The model was significant (F (1, 112) = 40.99, p<0.001). O-LIFE score was able to significantly predict AQ score (t (113) = 6.40, p<0.001, 95% *CI* .33 - .62), explaining 26% of the score variance (R^2 =.29,

Adjusted R^2 = .26). Results demonstrated that as O-LIFE score increased by 1 point, scores on the AQ also increased by 0.47 points (β = -0.47).

The study also aimed to assess the independent dimensions of schizotypy in relation to AQ score, therefore scores on the separate dimensions of schizotypy were used as predictor variables with AQ score as the dependent variable. The model was significant (F (4, 117) = 25.04, p<0.001). AQ score was significantly predicted by CogDis (t (127)= 3.92, p<0.001, 95% *CI* .35 – 1.08) and InAn score (t (123)= 6.68, p<0.001, 95% *CI* 1.03 - .1.90), explaining 44% of the score variance (R^2 =.46, Adjusted R^2 = .44) (see Figure 18). Results demonstrated that as AQ score increased by 1 point, scores on the CogDis increased by 0.71 points (β = 0.71) and InAn score increased by 1.46 points (β = 1.46). The model excluded UnEx and ImpNon scores (*ns*).

9.4.2 Partial correlation

Third order partial correlation was conducted to explore the unique contribution between the AQ and the dimensions of the O-LIFE whilst controlling for the influence of the other dimensions. Results showed a positive moderate correlation between the AQ and CogDis (pr = 35, p<0.001) when controlling for UnEx, InAn and ImpNon. A positive moderate correlation was also evident between the AQ and InAn (pr = 47, p<0.001) whilst controlling for UnEx, ImpNon and Cog Dis. No associations were evident for ImpNon and UnEx and the AQ (*ns*).

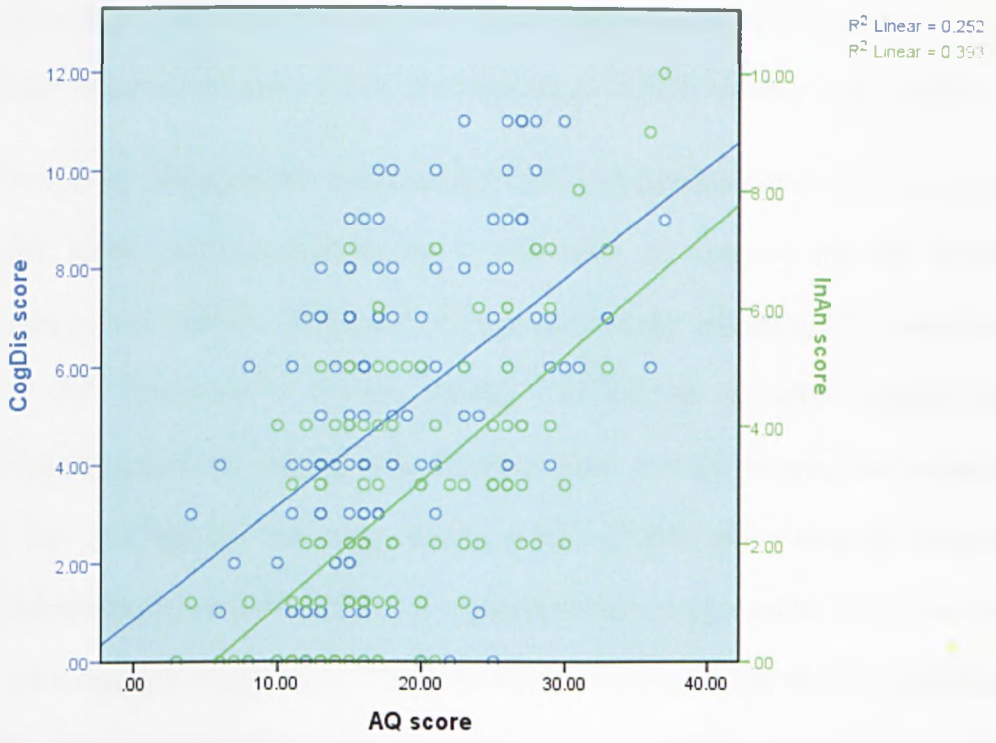


Fig.18: Linear relationship between AQ, disorganised (CogDis) and negative schizotypy (InAn) subgroups.

9.5 Discussion

The purpose of the present study was to assess the relationship between ASD and schizotypy in a neuro-typical adult sample. Consistent with previous findings (Spek & Wouters, 2010; Hurst et al., 2006), these results demonstrated that ASD and schizophrenia traits overall were positively correlated (r = .52), suggesting an overlap of diagnostic phenotypes rather than the diametric one. However, on closer inspection of the results, it was *negative* and *thought disorder* phenotypes of schizotypy that were positively correlated with autistic traits, adding support for the numerous other studies that have also found an overlap of ASD and negative schizophrenia at both a

clinical and trait level (Petty et al., 1984; Dyken et al., 1991; Sheitman et al., 2004; Spek & Wouters, 2010; Dinsdale et al., 2013; Mealey et al., 2014).

No inverse associations between positive or impulsive schizotypy and autistic traits were yielded, leading us to conclude no support for the diametric relationship between ASD and positive schizotypy, rejecting the prediction by the IBT (Badcock & Crespi, 2008). Our results do offer support for a dimension specific relationship between ASD and schizotypy and replication of the findings by Rawlings and Locarnini (2008) who failed to observe a relationship between autistic and positive schizotypy traits using the same measures employed here. These findings are also in line with Konstantareas and Hewitt (2001) who compared symptoms of ASD and schizophrenia in two clinical groups of adult males, one group with high functioning autism and the other group with schizophrenia, the group with high-functioning autism showed symptom overlap with the schizophrenic group on negative symptoms but not positive symptoms of schizophrenia. However, Gadow (2013) and Russell-Smith et al. (2011) both recorded a positive association between AQ scores and all dimensions of schizotypy, including positive elements, adding greater weight to the notion that the IBT is in need of revision.

9.5.1 Implications of results

These findings add to the argument surrounding the validity of the ASD-schizophrenia continuum. No support has been found for the diametric relationship between positive schizotypy and autism as the IBT postulates. Based on our findings, we conclude here that positive schizotypy and autistic

phenotypes exist on separate spectra. Evidentially the overlapping behavioural traits that ASD and schizophrenia share are the negative symptoms of schizophrenia [social and emotional withdrawal, blunted affect, diminished emotional range, poverty of speech (Blanchard & Cohen, 2006; Kirkpatrick et al., 2006)]. Hurst et al. (2006) suggests that because ASD and schizophrenia can present with very similar behavioural phenotypes, there is a great danger of inaccurately diagnosing either condition when measuring behavioural traits only, as they can be difficult to differentiate.

The results here demonstrated no relationship between the positive phenotypes of schizophrenia and ASD and provide an experiment of the replicability of previous findings (Hurst et al., 2006; Rawlings & Locarnini, 2008; Russell-Smith et al. 2011) that together add to the growing body of evidence to suggest the conjecture of the IBT is in need of reconsideration. However, an interesting study by Abu-Akel et al. (2015) investigated how a diagnosis of both ASD and psychosis may influence cognition and behaviour. Whilst they reported a small but significant positive correlation between the AQ and self-report schizotypy, they are also the first study to observe that those who show co-occurring autistic and psychosis traits may experience a 'normalising' effect on social-cognitive deficits. Suggesting a diametric nature of ASD and psychosis traits that may in fact create a 'buffer' against cognitive deficits, due to the balance of autistic and psychotic phenotypes.

Also to be considered, is research by Russell-Smith et al. (2011) that found whilst they were unable to conclude support for Badcock and Crespi's (2008) theory, due to strong positive associations between autistic traits and all dimensions of schizotypy, by breaking down the elements of the AQ, they

noted that imagination scores on the AQ negatively correlated with positive schizotypy. Whilst our findings cannot claim to support the IBT's diametric model, they do highlight the importance of future research *must* break down the positive elements of schizophrenia into their individual features (such as hallucinations, delusion, paranoid ideation) and looks at these elements in relation to the relationship with ASD individually (as was carried out in Chapter 7 of this thesis).

These results fail to support the notion of characterising positive schizophrenia phenotypes as the pathological consequence of the EFB.

9.5.2 Limitations

Gender data was unavailable in this sample, analysis which looked at the patterns of results in males and females separately may have been insightful due to the gender bias is both ASD (Baron-Cohen, 2003) and schizophrenia (McGrath et al., 2004).

9.6 Chapter summary of key points

- This study explored the *exact* nature of the relationship between ASD and schizophrenia traits in an adult neurotypical sample.
- The rationale for this investigation was derived from the lack of experimental support for the predictions of the IBT model in the previous chapters, and to therefore apply clarity to the results.
- Administering self-report inventories, results in this study demonstrated that **ASD and schizophrenia** traits (overall) were

positively correlated (r = .52), therefore signifying an ***overlap* of diagnostic phenotypes rather than the diametric one.**

- It was concluded that *positive* schizotypy and autistic phenotypes exist on separate spectra, with phenotypes relating to negative symptomology overlapping; *significantly* rejecting the prediction by the IBT at a non-clinical population level and providing clarity to the previous studies (Chapters 7 & 8).
- Again, this study highlights the need to explore individual phenotypes of schizophrenic conditions in relation to the EFB rather than schizophrenia as a whole.

Chapter 10

Testing the continuum; does empathising and systemising cognition trade-off? A two-part study considering age effects[21]

10.1 Introduction

The overall objective of this thesis was to explore the cognitive correlates of the EFB (Baron-Cohen, 2003). In doing so, it became apparent that it was pertinent to explore the underlying basis of the concept of the EFB, namely the E-S theory continuum.

The aim of this study was to investigate the specific nature of the relationship between social and spatial cognition. It is widely assumed these two cognitive processes work independently of each other (Wakabayashi et al., 2012). Whilst gender dis/advantages are *statistically* well founded in terms of social and spatial cognitive abilities, the nature of the *exact* relationship between mentalising and mechanical cognitive processes is not clear. The question of whether empathising and systemising correlate or share a trade-off relationship is *predicted* (Crespi & Badcock, 2008; Baron-Cohen, 2009), but is yet to be resolved. The model plots empathising and systemising as two dimensional coordinates which create brain types (see Figure 19 for illustration of the E-S model). Baron-Cohen et al. (2003) and Baron-Cohen (2009) suggest a trade-off relationship between the two intellects is probable,

[21] Some of the information presented in this chapter is subject to a manuscript in preparation entitled 'Does empathising and systemising cognition trade-off? A two-part study considering age effects' by S. L. Jones and V. Lesk.

but it is not *necessarily* present, and that they may work independently but share a 'special relationship'. However, inferring from Baron-Cohen's many works, the suggestion that testosterone increases systemising ability whilst decreasing empathising ability, guides that a negative relationship between empathising and systemising *should* be apparent if hypotheses of the E-S theory are accurate (Andrew et al., 2008; Carroll & Yung, 2006; Grove et al., 2013).

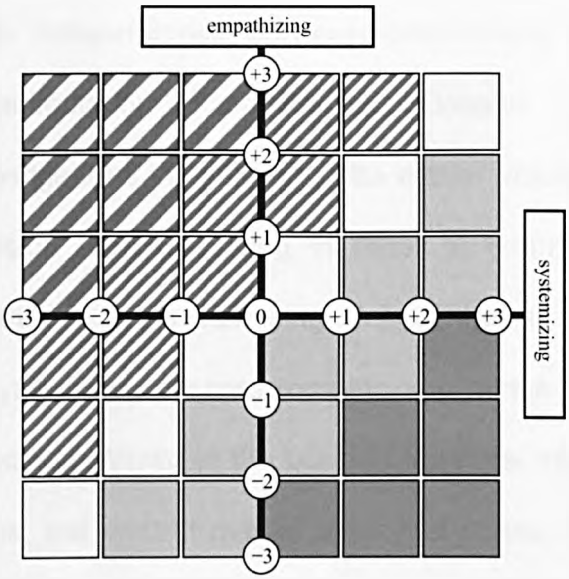


Fig. 19: The E-S theory model. Type B (E = S): unshaded; type E (E > S): narrow diagonal stripes; type S (E < S): grey shading; extreme type E: wide diagonal stripes; extreme type S: dark grey shading. Axes show SD from the mean. Image and footnote information belongs to Baron-Cohen et al. (2003)

So far, there is conflicting evidence in terms of support for the assumption of a trade-off relationship between empathising and systemising, which indicates that the relationship may be more complex than prophesied by the E-S theory and IBT. Some studies which have employed the EQ and SQ

have found a negative correlation suggesting an empathising and systemising trade-off, which is supportive of the E-S and IBT predictions that the two are separate yet related constructs (Baron-Cohen et al., 2003; Baron-Cohen & Wheelwright, 2004; Goldenfeld et al., 2005; Wheelwright et al., 2006; Nettle, 2007; Brosnan et al., 2010; Grove et al., 2013; Groen et al., 2015). Yet other studies fail to find any statistically significant relationship between the two (Lawson et al., 2004; Carroll & Yung 2006; Ling et al 2009; Russell- Smith, et al., 2012; Jones & Lesk, 2013; Di Ceglie et al., 2014), which suggests an independence between empathising and systemising rather than the manifestation of a trade-off relationship. Further, by using voxel-by-voxel investigations of regional white matter volume and fractional anisotropy of diffusion tensor imaging in order to examine white matter structures of empathising and systemising, Takeuchi et al. (2013) concluded empathising and systemising *d* score (empathising minus systemising) was inversely associated with rWMV in the bilateral temporal lobe, near the right inferior frontal gyrus, the ventral medial prefrontal cortex and the posterior cingulate cortex. Whereas a positive association was evident in the superior longitudinal fasciculus, suggesting support for the trade-off relationship at a neurological level, but interestingly, not at a behavioural level as no correlation was evident between the EQ and SQ measures (Takeuchi et al., 2013).

It is thought that whilst biological sex can be a good predictor of 'brain gender', people often show an atypical cognitive profile for their biological sex. Therefore, some suggest that the broader study of sex differences in cognition should be now focused on the investigation of 'cognitive style'

rather than the influence of biological sex on cognition and what this can offer in terms of the implications at a clinical level (Lai et al., 2012). Investigation into these proposed cognitive styles has already proposed implications for clinical psychology, for instance, as well as offering an explanation of subtle differences in normally developing cognition; these theories extend to attempt to explain the aetiologies of psychological disorders of the social brain. For example, as discussed in Chapter 4, the concept of the E-S theory was originally developed from the observation that ASD presents as the extreme of male typical behaviours, in that a typical cognitive profile in ASD demonstrates hyper-developed systemising ability alongside a hypo-developed empathising ability; the EMB profile (Baron-Cohen, 2002).

The IBT takes this line of thinking further, by suggesting that whilst ASD presents as the extremes of male sexual dimorphism, schizophrenia, bipolar disorder and depression seem to present as the opposite; the extreme of female sexual dimorphism (hyper-developed empathising alongside hypo-developed systemising) (Badcock & Crespi, 2008). Therefore, the IBT goes as far as to suggest ASD and schizophrenia exist as one disorder, connected diametrically at opposite ends on a continuum of cognitive profiles which characterises the entire population (Badcock & Crespi, 2008) (see Figure 20).

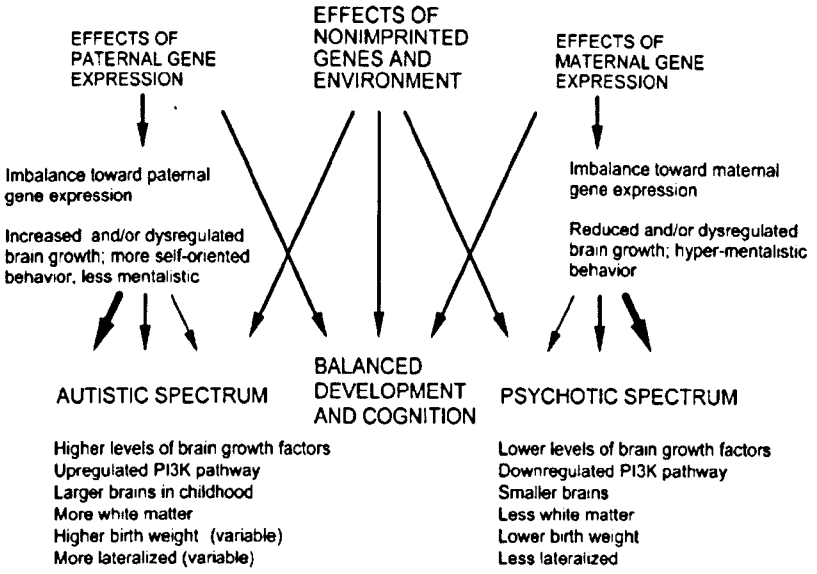


Fig. 20: The Imprinted Brain theory model of autism and psychosis. Image belongs to Crespi and Badcock (2008)

Investigation into the extremes of the continuum is required (see Chapter 5). It seems pertinent that the questions surrounding the unclear nature of the relationship between empathising and systemising is somewhat of a prerequisite. Indeed, the works produced by both Baron-Cohen (2002, 2003, 2008, 2009), Crespi and Badcock (2006), Badcock and Crespi (2008) and Badcock (2010) offer stimulating and intriguing possibilities in providing accurate models in explaining psychological disorders of the social brain, but the exact relationship between empathising and systemising (on which these explanations are based) requires further investigation.

10.2 Research aims

It is suggested here, that investigation into the central and fundamental suggestion of the IBT and the E-S theory (that there should exist a trade-off

between mentalistic and mechanicalistic cognitive ability) should be investigated in isolation from investigation into the relationship between the ASD and schizophrenia diametric relationship, This may offer us (i) further knowledge about the nature of the relationship between two axis of cognition and how they relate to each other in healthy cognition and (ii) support the fundamental assumptions of the ASD-schizophrenia continuum. For the proposed continuum to be *accurate* it is hypothesised that the healthy population should demonstrate a subtle trade-off [in terms of a negative correlation (Baron-Cohen et al., 2002)] between systemising/mechanistic cognition and empathic/mentalistic cognition.

By administering and interpreting data from psychological tasks and inventories, which explore both social and spatial cognition at the general population level, study one aimed to investigate the proposed trade-off between the two as predicted by the IBT and the E-S theory. The novelty of this study is that it takes investigation beyond self-report measures and investigates the trade-off relationship with direct performance measures. The argument could be made that the self-report measures used in previous research may fail to capture actual ability in relation to empathising and systemising. For instance, Carroll & Yung (2006) suggest that asking someone if they are *'fascinated by how machines work'* (an example of an item on the SQ), simply measures someone's preference for systemising- not their ability at it.

As per the logic inferred by predictions of the E-S theory and the IBT, it is hypothesised that (i) tasks of spatial ability will negatively correlate with tasks of social cognition, in that as a person's ability in systemising increases, their

empathising will decrease, and vice versa (ii) likewise, that self-report empathising will share an inverse relationship with self-report systemising.

Study two was conducted with an aim to replicate the findings of study one by employing self-report measures of empathising and systemising in a community based sample determined by G* Power priori analysis (Faul et al., 2009). This method controls statistical power before the study is conducted. The sample size was calculated in consideration of the alpha level, the beta level and effect size (based on previous findings) that was necessary for the null hypothesis to be rejected.

10.3 Study 1

10.3.1 Methods

10.3.1.1 Participants

A total of 35 student participants from the University of Bradford were recruited to take part in this study. The sample consisted of 23 females (66%) and 12 males (34%). No age data is available, however all participants were over the age of 18 years. Inclusion criteria specified that participants must have (i) no clinical diagnosis of mental illness (ii) have normal or corrected to normal vision (iii) and a fluent understanding of English language. Ethical approval was obtained from the Humanities, Social & Health Science Research Ethics committee at the University of Bradford. All participants gave their informed written consent.

10.3.1.2 Materials

Six neuropsychological tasks were employed as proxies to measure social and spatial cognition alongside two questionnaires which measured self-report empathising and systemising ability. See below for descriptions;

The Empathising Quotient (EQ) - The short form (EQ-S) version of the EQ (Wakabayshi et al., 2006b) measuring self-report cognitive and affective empathy was administered. See section 6.3.3.2 and appendix 1.

The Systemising Quotient (SQ) - The short form version (SQ-S) of the SQ was employed, which measures self-reported 'everyday' systemising ability (Wakabayshi et al., 2006b). See section 6.3.3.2 and appendix 2.

Together the EQ and SQ provide a score of empathising and systemising bias, and have previously demonstrated internal reliability and re-test reliability (Lawrence et al., 2004).

The Intuitive Physics Test (IPT) - The IPT (Baron-Cohen et al., 2001) is a direct test of systemising which measures what Baron-Cohen et al. (2001) terms 'folk physics', which is the natural, untaught ability to understand how mechanisms operate. The IPT comprises of 20 trials which each require the participant to work out the movement of an object which is caused by an initial movement. Each trial was presented as a diagram and required the participant to use spatial ability to hold the image in space whilst following the rules of movement to understand the cause and effect movement (see figure 21 for example). The correct answer is chosen from 4 possible answers. Scores are based on number of correct trials; the range of score is 0-20. Participants were given a maximum of 20 minutes to complete the test.

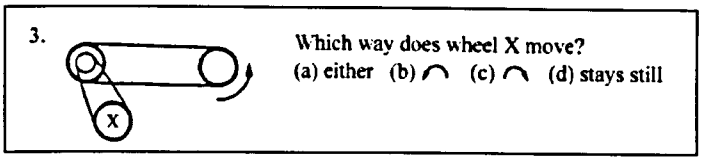


Fig. 21: Trial from the IPT. The correct response is (b).

The Folded Box Test (FB) - The FB task (adapted from Newton & Bristoll, n.d) is a measure of spatial ability. It aims to measure a person's ability to accurately identify which one of the four flat boxes is the layout of the assembled 3D box. The task has four trials (see figure 4 for example trial), each with one target assembled box, and four possible correct answers Participants were given a maximum of 4 minutes to complete the test.

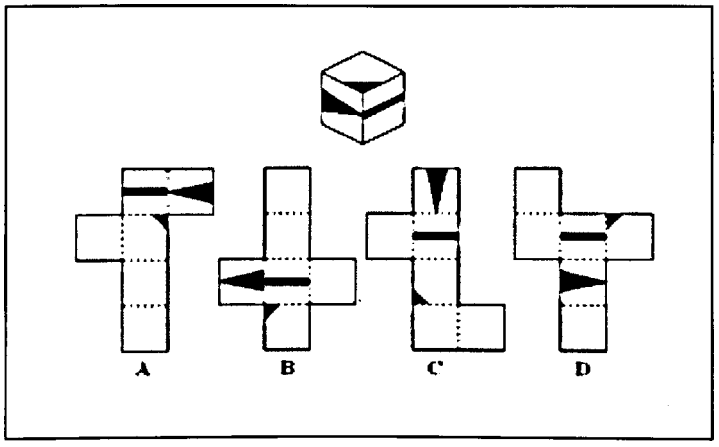


Fig. 22: example of the folded box task

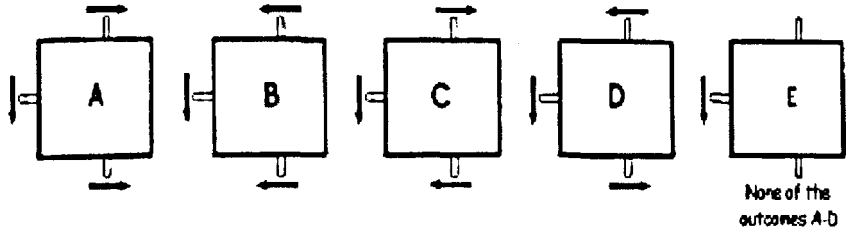
The Physical Predictions Test (PPT) – The PPT measures understanding of systemising. It is designed to be difficult enough to reveal individual differences in non-clinical populations (Lawson et al., 2004). The PPT is made up of 40 trials for which the respondent is required to "study

mechanical diagrams and predict the movement of two levers or bobs in response to the movement of a connected lever" (Lawson et al., 2004 p. 304). The participant was required to identify one correct answer out of five possible correct answers (see Figure 23 for example page from the test booklet). Scores are based on correct answers and the range of possible scores is 0-40. No timing data was recorded. Participants were given a maximum of 20 minutes to complete the test.

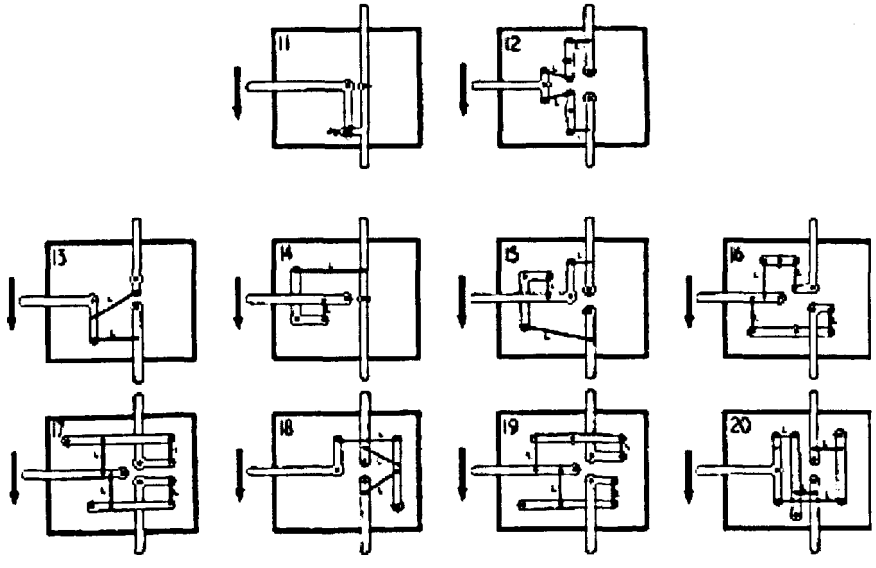
SECTION 2 continued: (Remember that option E indicates neither A,B,C or D)

Predict the movement of the top and bottom levers in response to the side lever being moved downwards.

Outcomes.



Diagrams of open boxes (P – pulley wheel: PS – pin and slot: L– solid link)



Answer box

Diagram	A	B	C	D	E	Diagram	A	B	C	D	E
11						16					
12						17					
13						18					
14						19					
15						20					

Fig. 23: The psychical predictions test

The Cambridge mind-reading face-voice battery (CAM-MR) **-** The CAM-MR (Golan et al., 2006a) is a measure of a person's understanding and recognition of subtle complex emotions through both visual and auditory channels. The CAM-MR was developed to provide a test of complex emotion recognition which aims to be closer ecologically to real life, as it uses moving pictures and voice clips rather than still pictures. The task comprised of 20 different emotions which were expressed through video clips and voice clips, of a professional actor acting out a particular emotion. There were 50 video clips trials and 50 voice clips trials, each lasting three to five seconds. The participant was presented with four words after each trial. The aim was to choose the adjective which best describes the emotional context of the trial (see figure 24 for example of a video clip). Scores were based on the number of correctly identified emotions, therefore scores range between 0-100.

Fig. 24: Example of the CAM-MR faces battery (still image taken from video)

The reading the mind in the films (RMTF) - The RMTF (Golan et al., 2006b) task is another task of complex emotion recognition. It is different to other ToM tasks in that it aims to grasp ecological validity by presenting the participant with film clips demonstrating social interaction between two to four people in social situations. The participant is required to observe 22 short clips from feature films and respond to a question about the emotional context of the clip after each video. For instance, a clip is played and a question relating to the clip is asked, such as *"how is the old lady in the clip feeling?"*. A choice of four adjectives are available for the participant to choose from in an attempt to correctly identify the correct emotion (see figure 25). Scores are based on correctly identifying the emotion, therefore scores range from 0-22. *Note:* Participants are presented with the list of feature films that the clips in this task have been extracted from, before taking part in this task. If the participant is familiar with, or has seen any one of the films listed, their data is removed from the collection to control for over exposure effects.

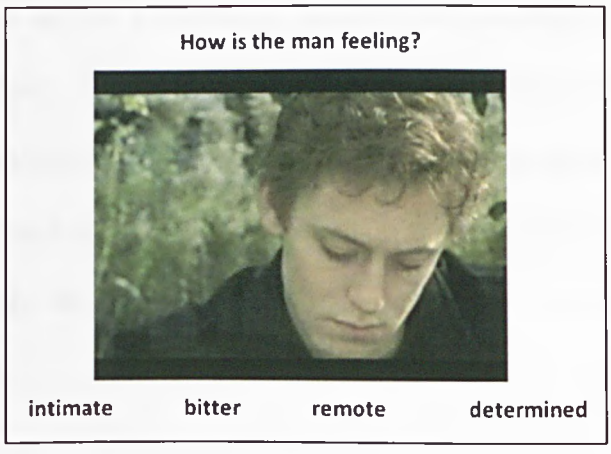


Fig. 25: Example of the CAM films battery
(still image taken from clip)

10.3.1.3 Design

A cross-sectional, primarily correlational design was employed; concerned with exploring the relationships between empathising and systemising using direct performance and self-report measures. Post data collection, the sample was also categorised into 'brain types' according to the method proposed by Carroll and Yung (2006). This made for analysis in way of an independent groups design. This study employed standardised neuropsychological tasks developed for non-clinical populations using standardised scoring. Familiarity with the theoretical bases of these tasks allowed for interpretation of cognitive styles and abilities. The researcher administered the battery of tests in random order for the purpose of controlling for order effects.

10.3.1.4 Procedure

Participants were recruited via email advertisement to all faculties at the University of Bradford. All participants completed the experiment in one session, carried out at the psychology labs in the Division of Psychology, University of Bradford. On arrival, participants were required to read the information sheet pertaining to the study and if happy to proceed, then signed the consent form. The battery of measures was then administered. Sessions lasted approximately 60 minutes and no participants requested a break during the sessions.

10.4 Results

The aim of this study was to investigate the proposed trade-off relationship between empathising and systemising, predicted by the E-S theory and the IBT by employing both self-report and direct measures.

Regression analysis was deemed inappropriate due to a relatively small sample size in relation to the number of variables. A correlational design was employed followed by an independent groups design in which the sample was categorised by Baron-Cohen's (2003) 'brain type' (post data collection) using the method offered by Carroll & Young (2006).

One participant was excluded from brain type analysis due to incomplete data.

Outliers greater than 3.29 were removed and the assumption of normality was tenable across the majority of variables; therefore parametric tests were employed at an alpha level of 0.05. Ranges, means and standard deviations for all measures can be found in Table 18.

Table 18: Means, standard deviations and range of scores for all measures

Variable	n	M	±SD	Min-Max possible score
EQ-S	34	27.71	5.11	0-44
SQ-S	35	22.20	9.22	0-50
FB	35	3.06	1.03	0-5
PPQ	35	8.69	5.27	0-40
IPT	35	11	3.73	0-2
CAM-MR	34	76074	7.25	0-100
RTMF	35	13.03	2.76	0-17

Note: EQ-S= empathising quotient (short form); SQ-S= systemising quotient (short form); FB= folded boxes; PPQ= physical predictions questionnaire; IPT= intuitive physics task; CAM-MR= Cambridge mindreading face-voice battery; RTMF= Reading the mind in the films

10.4.1 Main effect of brain type

In order to investigate the claims of the E-S theory, the sample was categorised into groups of 'brain types' (Carroll & Jung, 2006). EQ-S and SQ-S scores were firstly converted to *z*-scores, and the difference between the scores were calculated. A score of >1.00 was suggestive of a bias for a systemising profile or a type S brain. A score of <-1.00 suggests a bias for an empathising profile or a type E brain and scores between -1.00 and 1.00 represents a 'balanced brain' profile, which is neither a bias in empathising or

systemising. Table 19 shows the distribution of participants into the three categories[22] and table 20 shows the distribution of brain type by gender.

Table 19: Distribution of brain types across the sample

Brain type	Frequency	Percentage of sample
Type S brain	8	23.5
Balanced brain	22	64.7
Type E brain	4	11.8

Table 20: Distribution of brain types by gender

Gender	Brain type	Frequency	Percentage of sample
Male	Type S brain	6	50
	Balanced brain	6	50
	Type E brain	0	0
Female	Type S brain	2	9.1
	Balanced brain	16	72.7
	Type E brain	4	18.2

Note: table 3 shows that whereas half the male participants scored as type S brains, the majority of female participants scored as balanced brains, and where no males scored as type E brains, two female participants scored as type S brains.

[22] Means and standard deviations for all variables by 'brain type' as supplementary analysis in appendix 10

Univariate analysis of variance (GLM) revealed that gender had a significant main effect on determining brain type [F (1,32) = 5.48, p<0.05].

ANOVA revealed that type S brains (M = 14.13, ±SD = 3.09) scored significantly greater than balanced brains (M = 10.41, ±SD = 3.53) and type E brains (M = 8.25, ±SD = 2.99) on the IPT [F (2,31) = 5.07, p<0.05]. No differences were shown between balanced and type E brains (*ns*). Also, ANOVA revealed that type S brains (M = 71, ±SD = 5.07) scored significantly worse than balanced brains (M = 78.52, ±SD = 6.73) but not type E brains (M = 77.75, ±SD = 9.91) on the CAM-MR task [F (2,30) = 3.63, p<0.05]. No differences were evident between type E and balanced brains were evident (*ns*). No significant differences on performance on the FB task, the PPQ or RTMF task were established (*ns*).

10.4.2 Correlational analysis

Trade-off - Pearson's correlation coefficient revealed a *positive* relationship between scores on the EQ-S and the SQ-S (r =.01, p<0.05), not supportive of the hypothesised trade-off relationship or the independent hypothesis (see Figure 26). Correlational analysis did not establish any consistent trade-off relationships between the direct performance measures employed in this study (*ns*). The only evident relationship existed between the RTMF task and the FB task, demonstrating a significant negative relationship (r = -.33, p<.05), however, because no other variables demonstrated this same direction, it is possible that this result may be due to type I error.

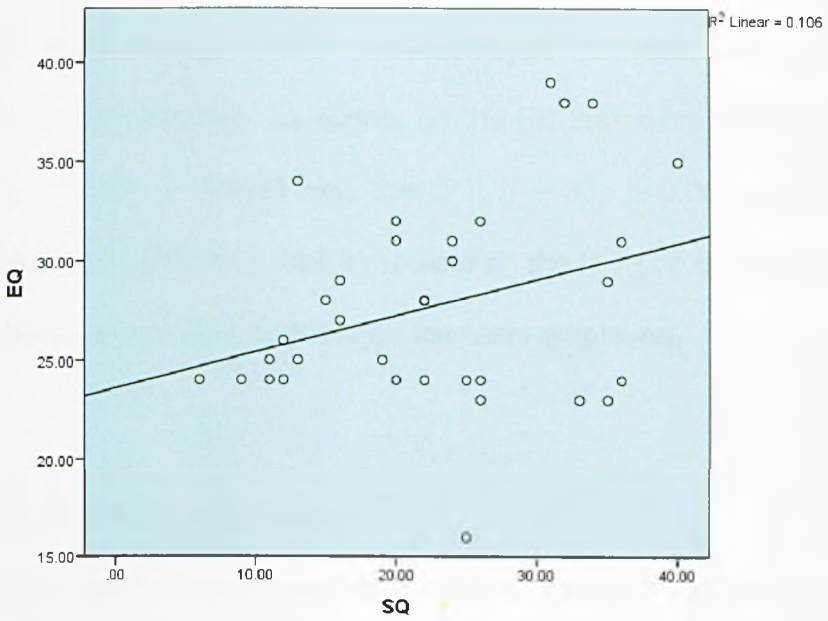


Fig. 26: Relationship between EQ-S and SQ-S (Study 1)

Associations between self-report and direct performance measures – In order to consider the relationship between self-report measures and performance measures, the study looked at correlational analysis between the EQ-S and the performance measures employed. No relationship between the EQ-S and the direct measures of empathising [CAM-MR, RTMF] were evident (*ns*), questioning the validity of the EQ-S. A positive relationship between the SQ-S and the PPQ was found (r = .46, p<0.005), suggesting that as a person's self-reported systemising ability increased, so too did their measured performance ability. However, relationships between the SQ-S and the FB task and the IPT were not apparent (*ns*).

Associations between direct performance measures - Pearson's correlation coefficient revealed that tasks measuring direct performance of cognitive empathy (CAM-MR and RMTF) shared a moderate positive

relationship (r =.43, p<.01). Correlational analysis revealed that there was also moderate to high positive relationships between tasks measuring systemising. For instance, as scores on the FB task increased they also on the PPQ (r =.49, p<0.005) and the IPT (r =.33, p<0.05) and as scores increased on the PPQ they also increased on the IPT (r =.67, p<0.001) which demonstrates a consistency between the tests employed.

10.5 Main findings - study one

Study one aimed to investigate the predicted trade-off relationship between empathising and systemising proposed by the IBT (Crespi & Badcock, 2008) and the E-S theory (Baron-Cohen, 2003). By employing self-report and direct performance measures of empathising and systemising in a student sample (n=35) it was expected that an inverse relationship between empathising and systemising would be apparent.

Firstly, biological sex is thought to be a predictor of 'brain type', and results showed that gender was a significant predictor of brain type which was an expected trend and was supportive of previous research (Baron-Cohen, 2003; Jones & Lesk, 2012). Secondly, and most prominent; results failed to provide any support for the theoretical trade-off relationship for both the self-report and direct performance measures. Instead, these results suggested a small dependency between the two dimensions, highlighted by the positive relationship between self-report empathising and systemising. This is a significant finding, as it calls into question the validity of the fundamental assumptions of the IBT and E-S theory on which the theories of neuropsychological consequences of extreme sex differences are built. Our

study is not the first to report a positive association between empathising and systemising measured by the EQ and SQ (Wright & Skagerberg, 2012); however, this is the first study to our knowledge to employ this particular battery of behavioural measures (CAM-MR, RTMF, FB, IPT and PPQ) to explore the theoretical trade-off relationship. Taking inference from correlational and 'brain type' analysis, our results failed to establish a consistent trade-off between empathising and systemising behaviours, which ultimately adds weight to the growing number of experimental studies which have guided the notion that the inverse relationship is unfounded.

However, our results did establish a small negative correlation between scores on the RTMF task and the folded boxes task, which does support the trade-off prediction. Yet, whilst that relationship was significant, this chapter would argue that the failure to identify any other significant relationships between any other variables does not assert a prominent finding and may be due to a type I error. Or, alternatively, perhaps highlights the need to consider the individual components of empathising and systemising that these particular tasks measured and ask the question of whether or not specific components of empathising and systemising share a trade-off relationship? Further research to explore this consideration is required.

10.6 Study 2

Study two aimed to replicate the unexpected findings of study one (the positive relationship between EQ-S and SQ-S), using a larger, more diverse sample by G*Power priori analysis.

10.6.1 Methods

10.6.1.1 Participants

A total of 158 participants took part, with a mean age of 34 years (±SD= 14.01, Range= 56). Only participants who responded to all questions were included, therefore 27 participants were excluded due to missing data. Participants were recruited via several channels, including by email invitation, posters, mailing lists and social media. Exclusion criteria specified no clinical diagnosis of mental illness. Ethical approval was obtained from the Humanities, Social & Health Science Research Ethics committee at the University of Bradford. All participants gave their informed written consent.

10.6.1.2 Materials

The EQ-S and SQ-S were employed. See section 6.3.3.2 and appendix 1 and 2.

10.6.1.3 Design

A cross-sectional, correlational design was employed, concerned with exploring the relationship between self-report empathising and systemising using a sample number determined by G* Power calculation. Participants completed the self-report measures online. Post data collection the data set was categorised into younger (18-35 years) and older (36+ years) age groups.

10.6.1.4 Procedure

All participants carried out the survey online via a custom designed survey hosted by Bristol Online Surveys. Firstly, participants were asked to read the information sheet carefully and were informed that by taking part they were declaring that they met the exclusion criteria to the best of their knowledge and that they were happy to participate in the study. When carrying out the survey, participants were not aware of the inventory the questions belonged to. Once respondents had accessed each question, the session ended. The software automatically excluded data from respondents who did not provide a response to all questions.

10.7 Results

The aim of this study was to investigate the trade-off relationship between empathising and systemising by employing self-report measures of empathising and systemising in a large community based sample.

In order to investigate the hypothesised trade-off relationship, G*Power calculation determined a minimum sample size of 115 subjects was required for a small to medium correlation (bivariate normal model) (based on results from Study 1) at an error probability of 0.05. Outliers greater than 3.29 were removed and the assumption of normality was tenable.

10.7.1 Correlational analysis

Pearson's correlation coefficient demonstrated a small positive association between scores on the EQ-S (M= 28.93, ±SD= 7.27) and scores on the SQ-S

(M= 18.14, ±SD= 10.37) (r =.16, p<0.05, n =131) (see Figure 27). Regression analysis revealed an R^2 of .02 (R^2 Adj=.02), and the model was just above the significance cut off at p = 0.07 [F (1,129) = 3.20].

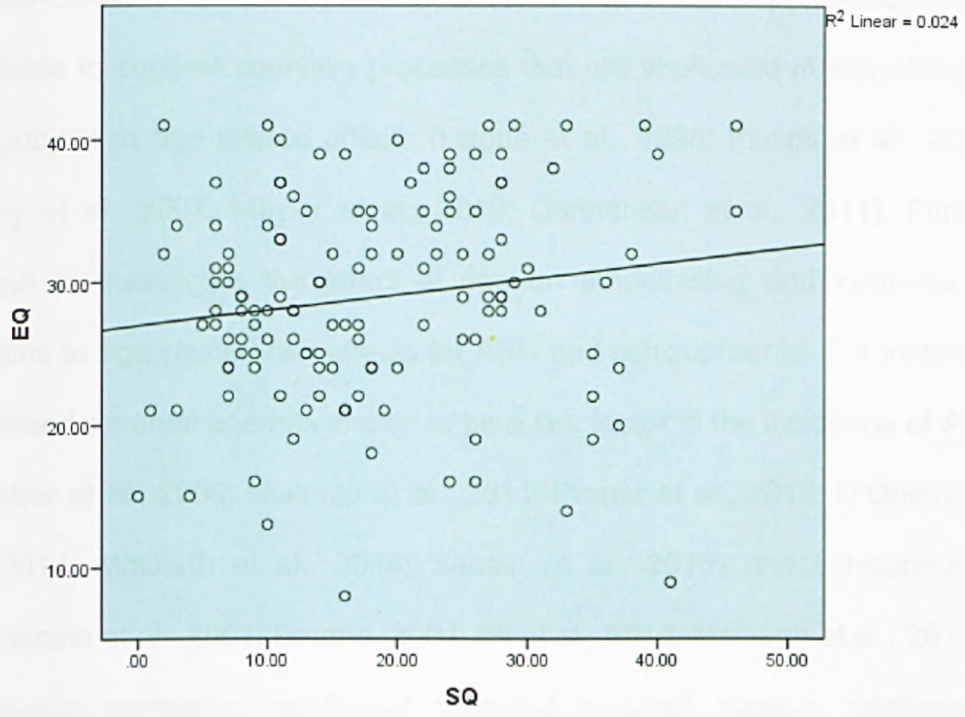


Fig. 27: Relationship between EQ-S and SQ-S (Study 2)

10.7.2 Age effects

One variable that has not previously been considered in the literature, as far as the researcher is aware, is the effect of a person's age on empathising and systemising cognition. It is not unreasonable to assume that because of factors that change over a person's lifetime that empathising and systemising may change also. For instance, changes occur in the brain as a result of natural aging processes (Gilsky, 2007). There is evidence of age related

cognitive changes in the hippocampus (Driscoll et al., 2003; Rosenzweig & Barnes, 2003 Lister & Barnes, 2009), a structure that is thought to be closely associated with systemising cognition (Magiure et al., 2000). Age related differences are also evident on tasks of spatial cognition (Grqady et al., 1994; Reuter-Lorenz et al., 2000; Moffat et al., 2006). Additionally, there is also evidence to suggest cognitive processes that are implicated in empathising, are subject to age related affects (Happé et al., 1998; Philips et al., 2002; Bailey et al., 2007; Maylor et al., 2010; Carstensen et al., 2011). Further reason to investigate the effect of age on empathising and systemising, pertains to age related risk effects for ASD and schizophrenia. For instance, increased parental age has shown to be a risk factor in the incidence of ASD (Grether et al., 2009; Hultman et al., 2011; Parner et al., 2012; D'Onofrio et al., 2014; McGarth et al., 2014; Sanain et al., 2016) and schizophrenia (Malasoina et al., 2001; Bourne, 2004; Ek et al., 2014; McGarth et al., 2014). Pearson's correlation coefficient revealed a small positive relationship between scores on the EQ-S and SQ-S for 18 to 35 year olds (M= 27.90 years ±SD= 8.42) (r= .32, p<0.05, n=94). The regression model was significant [F (1,92) = 9.76, p<0.05], with SQ-S scores explaining 10% of score variance [R^2=.10 (R^2 *Adj* =.09)]. The opposite was found for those between the ages of 36-74 years (M= 53.72 years ±SD= 7.70)]; a small moderate inverse correlation was evident (r= -.30, p=0.055, n= 28), the regression model was *ns*. See Figure 28.

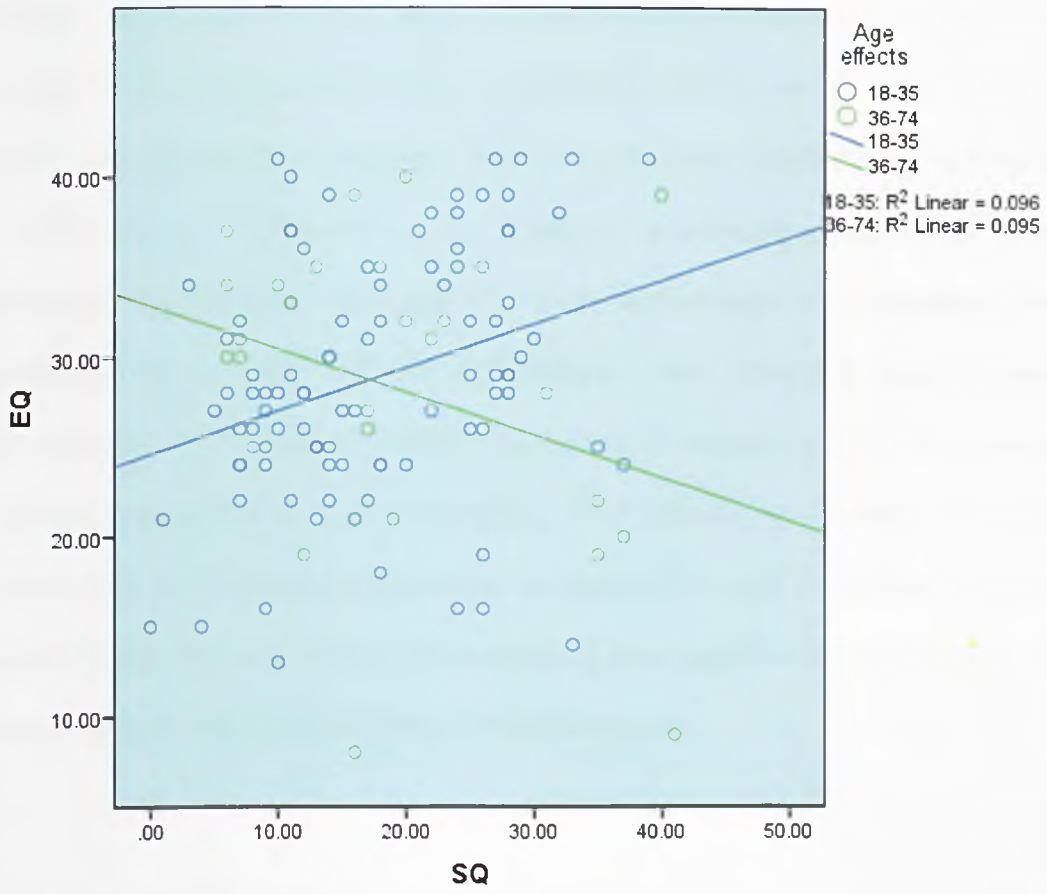


Fig. 28: Age effects on the relationship between EQ-S and SQ-S

10.8 Main findings - study two

Study two was conducted to provide replication of the positive relationship between empathising and systemising found in study one in a larger community based sample (determined by G* Power analysis). Results supported the previous findings; a small positive relationship was evident between empathising and systemising (EQ-S & SQ-S). This suggests that as empathising increased, so too did systemising, which again, points to a dependency between the two cognitions rather than the predicted trade-off. However, an interesting pattern of results emerged when the sample was subsequently examined in relation to age effects. The study found that in the

younger age group (18-35 years) a positive (larger) association between empathising and systemising was maintained, however in the group of 36+ years, we found the opposite trend; an inverse relationship between empathising and systemising was apparent, **providing support for the trade-off relationship between the two dimensions of cognition, as predicted by the IBT and the E-S theory**. This is the first study to the researcher's knowledge to explore age related effects on the relationship between empathising and systemising. This discovery provides the first insight into the potential importance of considering age in relation to how empathising and systemising work together and signifies the importance of exploring this observation further in future research.

10.9 General discussion

In relation to study one, the notion of a trade-off between mentalistic and mechanistic cognition fails to be substantiated by a robust evidence base, and it is argued here that there is likely to be some error in the presumption of a *consistent* trade-off relationship between the two dimensions.

Further to this, not only does this study not support a trade-off, we also fail to support independence between the two dimensions which Baron-Cohen (2003) suggests is also a possibility. It instead supports the notion of a small dependency between empathising and systemising. In terms of the implications for the assumptions of the IBT and the E-S theory at a behavioural level, our results are not in line with the trends between these two cognitions that would be expected. However, interestingly, when considering findings from study two, we can offer support for the predicted

trade-off relationship, but only in person's over the age of 36 years. Study two found that the dependency between empathising and systemising was maintained in the younger adults, yet an inverse relationship between empathising and systemising was found in the older group. Why would this be the case? This is the first study to our knowledge that has investigated age related effects on empathising and systemising and replication is required before robust conclusions can be drawn, however, this chapter suggests a few possible explanations.

Firstly, let us consider the influence of biological and psychological changes within the brain that occur with the natural aging process (Gilsky, 2007). Unique patterns of loss occur in different structures of the brain (Driscall et al., 2009); how far can we infer that these changes explain the difference in the relationship between empathising and systemising cognition according to age? There is some evidence to suggest that systemising abilities are implicated in the hippocampus (Maguire et al., 2000; Driscoll et al., 2003), and it has been found that (for women especially) the hippocampus is particularly vulnerable to age related loss (Crivello et al., 2014), which may begin to decline in early adulthood (20/30 years of age) (Salthouse, 2009). This would be particularly relevant here, as our *older* age group comprised of person's over the age of 36 years. Further to this, cognitive functions associated with empathising have found to be maintained in age related cognitive decline (Carstensen et al., 2011). Could it be that empathising is maintained and systemising is subject to greater loss in older adults, therefore we observe the trade-off relationship between empathising and systemising in older ages? Obviously, the structure function relationship is

complex and subject to much inference, in that neuroanatomical changes may *not* lead to cognitive performance changes (Raz & Rodrigue, 2006). Therefore, we offer this as postulation only, however further research is warranted.

Secondly, it is suggested that the structure of the measures employed, namely the SQ, does not take into account, and adapt to, the increasing use of smart technology in everyday life, especially in younger adults. Take for instance this statement; '*I rarely read articles or web pages about new technology*'; before the popularity of smartphones and portable computer devices, to respond positively to this statement, the respondent would have to be actively seeking out articles about technology. However, in today's world, we are exposed to vast amounts of new information at a rapid pace, which is easily accessible. News and media outlets on our smart devices offer this information to us without any effort on our part to pursue it, which may mean that we are exposed to this information without seeking it out. Another example is the SQ statement '*I find it difficult to learn my way around a new city*'. Again, our access to smart technology means that systemising can be done for us, respondents may factor in that they use their smart devices to guide them around new places without the need to use signage, paper maps or even spatial awareness. Therefore, some respondents may respond positively to this statement because they are aided by smart technology in this task. Does this frequent use of and easy access to smart technology allow respondent's to answer differently to what they might do if we did not have technology readily available to systemise *for* us? This is particularly relevant when considering the concept of age effects on

empathising and systemising in relation to smart technology, as research has shown that when taking into account education and affluence, older adults who are considered less affluent are 'largely disconnected' to smart technology (Smith, 2014). Further, Wakefield (2015) suggests that there is a dearth in the of use of smart technology in older adults of today, due to a fundamental lack of understanding of basic technology which leads older adults to not appreciate the usefulness of modern technology aids. *However*, whilst this may be a useful postulation in terms of elderly adults, the older adults in our study comprised a range of ages between 36 and 74 years with a mean age of 53 years, therefore it is difficult to apply this research to such a broad range of ages and *unlikely* to apply to people in mid-life. However, it is proposed here that revision to the SQ items to control for statements which have the potential to refer to technology that systemises for us rather than measures interest in, or ability in systemising behaviours is warranted. Whilst considering these explanations, our results strongly support that age has a significant effect on the presence of the assumed trade-off; finding that older adults demonstrate the trade-off relationship between empathising and systemising. Whereas, young adults demonstrate a positive correlation between the two cognitive processes. These findings represent the first results to our knowledge that illustrate this trend and provide a new insight into the relationship between empathising and systemising.

In terms of how these results contribute to the ongoing debate in the relationship between mentalistic and mechanistic cognition, they add to the studies that fail to support the notion of a *consistent* trade-off (Russell-Smith et al., 2012). Yet, as previously discussed, some studies have found a trade-

off between the EQ and SQ (Baron-Cohen et al., 2003; Baron-Cohen & Wheelwright, 2004; Goldenfeld et al., 2005; Wheelwright et al., 2006; Brosnan et al., 2010; Grove et al., 2013; Groen et al., 2015), which when considered collectively, signify the importance of teasing out the complexities of this relationship. However the evidence presented here of a positive relationship between empathising and systemising in younger adults and an **inverse** relationship between empathising and systemising in older adults, is a thought-provoking novel finding that requires further investigation.

10.9.1 Considerations

Our analysis highlights the concern of incongruity between self-report and direct performance measures. Russell-Smith et al. (2012) also found a significant lack of correlation between self-report measures and direct performance measures of empathising and systemising when conducting a similar study. The inconsistency found in this study drives the need for future research, which is aimed at teasing out the reasons for this incongruity. Andrew et al., (2008) argue that most of the investigation into the E-S theory is limited, in that it employs the EQ and SQ exclusively. The EQ and SQ are ultimately self-report measures, which are weighted with the criticism that they are problematic due to measuring a person's opinion of how empathic or systematic they may be, instead of directly assessing skilfulness in said abilities. It is notable that much of the work surrounding the E-S theory and the relationships between empathising and systemising has employed these measures, which according to Andrew, et al. (2008) are not well validated (although both the EQ and SQ have been found to be well validated, see

Lawrence et al. [2004]). This study contributes to the argument surrounding the reliability of self-report measures as this study found no correlations between self-report empathising and direct measures of empathising in study one; questioning whether self-reported ability is reliable enough to draw conclusions from.

In relation to study one, only cognitive empathy was measured and measures of affective empathy may show different results as the two components of empathy (cognitive and affective) have been found to measure as two distinct abilities in ASD samples (Henry et al., 2007; Russell-Smith et al., 2012). Future studies should employ direct measures of affective empathy as well as cognitive empathy for a comprehensive measure of both empathy components.

If empathising and systemising fail to show an association at a general population level, then it leads us to conclude that the nature of the E-S theory may not be a *continuum* at all. It would be difficult to link the relationship between empathising and systemising in the general population to the relationship between empathising and systemising in an ASD sample- where the negative relationship is observably apparent (Carroll & Yung, 2006). It may be more accurate to propose that the EMB theory (Baron-Cohen, 2002) and the E-S theory can be understood as separate models. A question proposed by the findings in this study lead us to consider; should investigation into the wider research area of the ASD - schizophrenia continuum go forth separately relative to the investigation into the typical sex differences in the general population?

10.9.2 Limitations

It is acknowledge that there are some limitations in this study which must be considered before inferring any solid conclusions. The tasks employed in this study may be insufficient in their assessment of empathising and systemising in that the tasks employed may measure only a narrow part of the broader empathising and systemising concept (as referred to above). For instance, whilst the EQ measures both cognitive and affective empathy, the empathising tasks are a measure of cognitive empathy but *not* affective empathy. Also, the tasks employed cannot claim to comprehensively measure systemising, but instead only the specific parts of spatial or logical reasoning ability. Future studies should investigate the narrow branches of empathising and systemising in relation to each other and analyse them separately in order to investigate whether only certain aspects of both empathising and systemising share the predicted trade-off of the E-S theory and IBT concepts. Lastly, Russell-Smith et al. (2012) advises that given the close links between systemising and IQ, controlling for IQ in studies that investigate empathising and systemising is advantageous; this should be a consideration in future studies.

10.10 Conclusions

These results provide substantial evidence that supports the need to reconsider the fundamental assumptions about the relationship between empathising and systemising made the IBT and the E-S theory. Results also provide novel findings that emphasise how exploring the age effects has a

significant contribution to the relationship between empathising and systemising.

It is only logical that a negative relationship between the two *should* exist. The fact that this study and others (Baron-Cohen et al., 2003; Carroll & Yung, 2006; Ling et al., 2009; Auyeung et al., 2012; Jones & Lesk, 2013; Meng-Chuan et al., 2012; Russell- Smith et al., 2012; Di Ceglie et al., 2014; Takeuchi et al., 2014) fail to find this is problematic, especially since this study adds novelty to the literature in that it employed a particular battery of standardised direct measures of empathising and systemising, *not solely* self-report scales. Further, both Andrew, et al. (2008) and Jarrod, et al. (2000) argue that the very small correlation (usually around $r = -.17$) found in some supportive studies does not provide robust support for the trade-off predicted by Baron-Cohen. Jarrod, et al. (2000) signifies, and this paper can only conclude a similar opinion that, the E-S theory should have a substantial evidence base for a negative relationship between empathising and systemising and if this is not manifest in experimental research, then the theory needs revision or at least some clarity on what exactly its fundamental assumptions are. However, the notion of a trade-off between empathising and systemising is apparent in older adults alongside a linear dependency between empathising and systemising in younger adults, which provides novel findings that add to the complexity of the relationship between the two processes.

It is important that the IBT or E-S continuum have a body of supportive research that demonstrates that its theory is *grounded* within an *accurate* model to explaining sex differences. Because the whole E-S continuum relies

on these fundamental assumptions between empathising and systemising (as does the predictions of the IBT), it must be addressed that if experimental evidence is not supporting such a postulation then the theory needs some revision. We believe the findings from this study offer new insights into the seemingly complex relationship between empathising and systemising which highlights the potential importance of age effects determining the relationship between empathising and systemising. Further research is warranted.

10.11 Chapter summary of key points

- The concept of the EFB sits within the wider concept of the E-S continuum.
- Experimental support for the E-S in terms of the predicted trade-off between empathising and systemising is limited and contradictory.
- Study one employed both self-report and direct performance measures of empathising and systemising in a student sample (n=35); hypothesising that a trade-off between the two types of cognition would be apparent.
- Results indicated a positive association between empathising and systemising, suggesting a dependency between the two.
- Study two was conducted to replicate the previous result in a larger community based sample, and also explored possible confounding factors such as age, to determine if these cognitive abilities remain constant throughout the lifespan.

- Results demonstrated that there is a significant dependency between empathising and systemising in younger adults, however, a **trade-off between empathising and systemising is apparent in adults older than 36 years of age.**
- This is the first study to the researcher's knowledge to explore age related effects in reference to the relationship between empathising and systemising. This provides the first insight into the importance of considering how age *significantly determines* the relationship between empathising and systemising.
- Considering the changes that occur in social relationships throughout the lifespan and the affects that life events such as pregnancy and menopause can have on hormone levels, this chapter argues that age must be taken into account in studies that aim to measure cognitive functioning that is involved in emotional processing. Further, investigation into the social changes that occur throughout the lifespan should be investigated – for instance, social psychology concepts could be useful here, for future research to explore the social changes in the lifespan, and how they may contribute to the relationship between empathising and systemising cognition.

Chapter 11

General conclusion

11.1 Thesis objectives and rationale

The E-S theory is based on the premise that sex differences can be categorised into two main concepts; empathising ability, the drive to understand others' mental states, and systemising ability, the intuitive ability to understand phenomena that uses a system (Baron-Cohen, 2003). By quantifying these cognitive abilities, the E-S theory is able to categorise the population into 'brain types' based on a trade-off between empathising and systemising. When this trade-off is at an extreme bias towards systemising, research shows that this profile – the extreme masculinised profile – results in the expression of ASD (Baron-Cohen, 2002).

The primary aim of this thesis was to address an important gap in the literature – can a feminised brain profile result in pathology in the same way that the masculinisation of the brain can result in neuropsychological disorder? This thesis explored this question based on the hypothesis of the IBT; that ASD and schizophrenia exist on a continuum as diametric disorders of the social brain, determined by a bias towards either maternal or paternal gene imprinting (Badcock & Crespi, 2006). The question of a possible link between a feminised brain profile and pathology is important to explore due to the dearth of research into pathology of the feminised brain profile. Especially when considering the progress that has been made in furthering

the knowledge of how extreme sex differences can be implicated in disorders such as ASD (Baron-Cohen, 2002), Angelman syndrome and Prader-Willi syndrome (Knoll et al., 1989; Kirkilionis et al., 1991; Oliver et al., 2007; Buiting, 2010) (Chapters 2, 4 & 5). This thesis investigated the feminised brain profile in relation to schizotypy traits in the healthy population. It took into consideration that, in an attempt to link the EFB profile with schizophrenia, previous works have failed to recognise that schizophrenic conditions consists of *numerous* individual components that may share specific relationships with empathising and systemising. Therefore, it was argued that previous work has been too general, and a more accurate investigation into this IBT hypothesis was derived from investigation into the individual phenotypes of schizotypy, rather than quantifying schizotypy as a whole. It seemed rational to consider the notion that *specific* cognitive processes, behavioural, or personality traits may share *specific* types of relationships with each other, thus aiming to collate a *clearer* theory of the relationship between the feminised profile and schizotypy in this thesis compared to previous findings in the EFB literature.

Secondly, cognitive networks which have not previously been explored in relation to the E-S model of sex differences were examined. This thesis investigated the predictive power of episodic memory ability in relation to accurately placing a person on the E-S continuum. As far as the researcher is aware, this was the first investigation that considered a link between the E-S model and memory ability. This was an important study that demonstrated that other networks are implicated in empathising and systemising which

identifies that the E-S model is capable of incorporating other cognitive processes, other than just networks involved in spatial and social cognition.

Thirdly, an additional *unavoidable* objective became apparent as this thesis progressed. It became pertinent to address an important grey area in the literature - the question of the accuracy of the E-S theory in terms of the fundamental *assumption* that empathising and systemising share a 'trade-off' relationship. This specific question was important in three ways. Firstly, any investigation relating to psychological and neuropsychological disorder that is thought to be expressed due to these patterns of extreme sex differences (as per the primary aim of this thesis), need a solid evidence base on which to test hypothesises – and this presumed trade-off was *not* apparent *consistently* throughout this thesis. Secondly, the E-S theory has been subject to vast amounts of research in an attempt to explain sex differences in non-clinical populations (Chapter 3). However, on closer inspection of these works, very few studies have raised the question of the presence of the presumed trade-off relationship that *should* be apparent if the model is accurate. Therefore, not only was thorough investigation into this central association between empathising and systemising required to clarify the results in this thesis, it also questioned the findings presented in previous research that seemingly ignores the questions surrounding the trade-off relationship between empathising and systemising. Thirdly, the definition of the EFB concept is *based* on the E-S model; therefore, if the E-S model is in need of revision to its fundamental assumptions in terms of the relationship between empathising and systemising, this significantly effects investigation into the EFB.

11.1.1 Experimental design

These questions were investigated using a variety of cognitive task paradigms and self-report inventories. In order to explore the notion of gendered brains (i.e., the E-S theory), this thesis employed three different experimental methods; the grouping variable of 'brain type' (Carroll & Yung, 2006), the grouping variable of E-S bias using z-scores and the notion of empathising and systemising as independent variables using continuous data in regression analysis. This procedure was to ensure that a comprehensive investigation into the principles of the E-S theory was carried out and the method employed to categorise the data was suitable for the specific research question in each experimental paradigm. All of the experiments reported in this thesis employed non-clinical samples; however, due to the continuum nature of this research area, the findings reported here have implications for clinical populations of ASD and schizophrenia cohorts.

The subsequent section provides a summary of the experimental chapters presented throughout this work. Following this, a discussion surrounding the implications, possible methodological considerations and issues is presented. Finally, suggestions for possible future studies that are appropriate to further advance these discoveries are offered, along with concluding remarks.

11.2 Summaries of experimental chapters

Chapter 6 focused on the identification of cognitive functioning that had not previously been considered in relation to the E-S model. The hypothesis that memory ability may be implicated in the E-S model was based on the notion

the IBT posits that advanced episodic memory should be evident in feminised profiles. This is due to the theoretical association between the feminised profile and schizophrenia, which is based on the belief that episodic memory failure is thought to be the underlying cause of schizophrenia symptoms such as hallucinations and delusions (Badcock, 2011). This study employed a novel episodic memory paradigm which explored source memory (SM), associative memory (AM) and source monitoring error (SME) to test the hypothesis that participants who scored greater on empathising, and lesser on systemising ability (i.e., type E brains) would demonstrate greater ability in episodic memory recall. Results revealed that both **AM ability** and the **frequency of SMEs** could indeed be a **predictive cognitive marker** of a person's place on the E-S continuum, specifically when the memory involved, a **social context.** Not only does this establish new knowledge in relation to the other cognitive networks that are implicated in the E-S model, it also provides the first (to the researcher's knowledge) study to find empirical support for the IBT's postulation that higher empathisers are likely to experience source monitoring failure based on the link between diminished episodic memory capacity and schizophrenia (Badcock, 2011). Further research should investigate more cognitive functions that could be associated with empathising and systemising such as attention, semantics, perception and other areas of memory processing such as semantic memory, all with a social and non-social condition.

Having identified novel cognitive markers on the E-S continuum in Chapter 6, **Chapter 7** moved on to explore *specifically* the hypothesis that schizophrenia

is associated with a type E brain profile (Badcock & Crespi, 2008). The study investigated E-S theory 'brain types' (see section 3.4 for a detailed description of this concept) in relation to dimensional schizotypy (Chapter 9). Results revealed that, interestingly, and contrary to the hypothesis of the IBT, **type S brains** (those who scored *lesser* on empathising and *greater* on systemising abilities) scored ***greater*** on scores of **schizotypy** (overall) than both balanced brains and type E brains. This is an important finding which ***does not*** support the IBT's prediction that schizophrenia is associated with the feminised profile. Instead, results demonstrated that a *masculinised* profile scores significantly higher on schizotypy, leading to the conclusion that schizotypy is associated with a systemising bias, *not* an empathising bias – in direct opposition to the IBT hypothesis. This however is *not* surprising when considering the difficulties with social intelligence experienced in ASD, which may lead to the expression of negative symptoms of schizotypy such as flattened affect, avoidance of social intimacy and situations.

Considering the unanticipated findings reported in Chapter 7, more *in-depth* investigation was warranted into the theoretical link between positive schizotypy and the type E brain profile based on the predictions of the IBT. The study reported in **Chapter 8,** asked the question of whether the postulation of the diametric relationship between the broad *combined* phenotypes of ASD and schizophrenia is too simplistic. It seemed rational to consider, based on the results of the previous chapter, that the notion that *specific* phenotypes may share *specific* types of relationships is likely. This

study explored the relationship between E-S bias (section 8.3.2 for details) using a novel task paradigm measuring paranoid ideation (PI) and jumping to conclusions bias (JTC) (section 8.1.2 for detailed overview of these concepts). The theoretical justification for this study derived from the close affiliation between ToM (empathising) and the experience of paranoia (Thakkar et al., 2008). Results demonstrated that performance on the JTC task was significantly affected by E-S bias, with **greater empathisers and lesser systemisers showing greater tendency to jump to conclusions**. These findings established that, those who presented with an empathising bias required less evidential information before making a rational decision, compared to those who presented with systemising bias. This supports the expected trend theorised by the IBT (Badcock & Crespi, 2006). *However*, interestingly, **greater empathising** was also associated with **lesser experience of paranoid thoughts**, once again going directly *against* the IBT hypothesis. This study, therefore offered both support *and* conflicting findings for the IBT model. Here, it was concluded that there is evidence of a link between positive schizotypy and the 'female brain', but only when considering ***individual phenotypes*** of schizotypy – as clearly, not all components of schizophrenia can be considered an accurate characterisation of the EFB.

Chapter 9 aimed to assess the *exact* nature of the relationship between ASD and schizophrenia traits in an adult neurotypical sample. The rationale for this investigation was derived from the lack of experimental support for the predictions of the IBT model reported in Chapters 7 and 8. Therefore, it was

important to directly address the relationship between ASD and schizophrenia traits in order to apply clarity to previous findings. Badcock and Crespi's (2006) diametric model creates a continuum of cognitive profiles that incorporate ASD and schizophrenia at the extreme ends, with trait populations in between. Administering self-report inventories, results in this study demonstrated that **ASD and schizophrenia** traits (overall) were positively correlated (r = .52), therefore signifying an ***overlap* of diagnostic phenotypes rather than the diametric one**. Based on this finding, it was concluded that *positive* schizophrenia and autistic phenotypes exist on separate spectra, with phenotypes relating to negative symptomology (diminished affect, avoidance of social intimacy and lack of enjoyment from social situations [Mason & Claridge, 2005]) overlapping; *significantly* rejecting the prediction by the IBT at a non-clinical population level and providing clarity to the previous studies (Chapters 7 & 8). Again, this study highlights the need to explore individual phenotypes of schizophrenic conditions in relation to the EFB rather than schizophrenia as a whole.

Chapter 10 was the final experimental study reported in this thesis. It reported a two-part study which focused on clarifying the fundamental trade-off *presumption* between empathising and systemising cognition. When exploring the data sets collected in previous studies, it became apparent that the presumed trade-off was *consistently* not evident using correlation analysis of tasks measuring empathising and systemising. This study interpreted data from psychological tasks and inventories which explored both social and spatial cognition in a student sample (Study 1) and a general

population sample (Study 2). Results from study one demonstrated a small **dependency between empathising and systemising cognition**, significantly calling into question the validity of the fundamental assumptions of the IBT and E-S theory. Study two was conducted to provide replication of the positive relationship between empathising and systemising found in study one, in a larger community based sample (determined by G* Power analysis). This also explored possible confounding factors such as age-related effects to determine if these cognitive abilities remain constant throughout the lifespan. Results demonstrated that there is a significant dependency between empathising and systemising in younger adults, however, a **trade-off between empathising and systemising is apparent in adults older than 36 years of age**. This is the first study to the researcher's knowledge to explore age related effects on the relationship between empathising and systemising and provides the first insight into the importance of considering how age significantly determines the relationship between empathising and systemising. This is not a surprising finding, given that changes in social relationships over the lifespan occur and life events such as pregnancy and menopause affect hormone levels, therefore, age is a factor that must be taken into account in studies such as this.

11.2.1 Summary of findings

To summarise, the results presented here suggest that memory ability is significantly associated with empathising and systemising ability; capacity for **social associative memory can significantly predict a person's place on the E-S continuum**. This was an original finding that had not previously

been investigated, therefore adding significant, new knowledge to the E-S theory literature. Also, **higher empathisers are also more susceptible to SMEs**. This provided the first (to the researcher's knowledge) empirical evidence supporting the prediction of the IBT that episodic memory capacity is likely to be reduced in feminised profiles. At the same time, further support for the IBT model came from the finding that **JTC bias was associated with empathising bias**, suggesting a possible link between the female end of the E-S continuum and *individual phenotypes* of schizophrenia. In direct conflict of the IBT presumption, **type S brains were found to have greater levels of schizophrenia traits**. We would expect, if the schizophrenia can accurately characterise the EFB profile that female brains would present with *greater* positive schizophrenia traits, therefore it was concluded that schizotypy is most likely to be associated with the masculinised profile. This finding was supported by the next experimental study that found **ASD and schizophrenic traits positively correlated** in a large general population sample, again, providing support for the previous findings *and* confirming a lack of experimental support for the predictions of the IBT model of diametric opposites. Results of the final study demonstrated that in **adults aged over 36 years, a trade-off relationship between empathising and systemising is evident**, supporting the E-S theory's fundamental assumption yet suggesting that the 'trade-off' is a concept *much more* complex than previously anticipated.

11.3 Implications of this thesis

This thesis adds to the debate on whether sex differences should be considered a modulating factor in all neuroscientific enquiry. It questions the notion that data may not be generalisable; perhaps results should be considered sex specific when only one gender is employed in a sample? This thesis significantly contributes to the debate by highlighting the notion that this area is indeed a complex and important area of research. Most importantly, whilst widening the knowledge pertaining to both the E-S and IBT, at the same time, this thesis offers new knowledge to the research area and argues that some of the major claims of the most popular theories are in need of serious revision.

The implications for the field of social cognitive neuroscience are vast, especially when considering the impact that human sex differences have on psychopathology and neuropsychological disorder (Baron-Cohen, 2002; Zahn-Waxler et al., 2008). This thesis argues that there is a clear need for the further development of accurate models of sex differences in relation to brain structure, anatomy, and behaviour. This would allow the field to establish useful treatment formulas and explore possible strategies for intervention before pathology is manifest (for example, the notion that testosterone could have a protective effect of the pathology of the EFB due to the theoretical deficiency of testosterone in this profile [Chapter 4]). In addition, the observation that age significantly affects how cognitive functions work together with each other (the E-S trade-off) suggests that age should be a consideration in studies such as the kind reported in this thesis.

Specifically in relation to the implications for the EFB concept, this thesis provides a much clearer picture of the comparability between the E-S theory and the IBT than previous accounts. This thesis establishes a link between the 'female brain' profile and JTC bias (Chapter 8), which is a non-pathological concept within the broader positive schizophrenia symptomology. However, contrary to the initial hypothesis, these works significantly rejected the notion of a potential relationship between the EFB profile and schizophrenia. Specifically, when considering that this thesis found that (i) type S brains are significantly associated with schizotypy and (ii) ASD traits and schizotypy were found to share significant overlap of phenotypes. It is concluded here that the IBT at a behavioural level requires revision.

Considering this, in terms of the implications for the field of study more generally, this particular finding signifies the importance of investigating the *individual components* of larger phenotypes in relation to the EFB (and indeed other cognitive profiles). Whilst this thesis did not employ clinical samples, this notion can have implications for the concept of 'diagnostic semantics'. For instance, schizophrenia, borderline personality disorder, depression and ED amongst others, all share 'trans-diagnostic' causes and symptoms, and are often associated with schizophrenia through high rates of co-morbidity (Dinsdale et al., 2016). Therefore, Dinsdale et al. (2016) suggest that in the context of this specific research area 'it is important to bear in mind that psychiatric diagnoses represent artificial constructs rather than etiologically-defined diseases per se' (p. 12). Considering this, together with the significant finding that *individual concepts* of positive schizophrenia were

found to share a relationship with the 'female brain' profile. It is advised here, that research should aim to explore individual symptoms of disorder, rather than disorders as a whole, in order to (i) gain more in-depth, accurate research (ii) help unravel the complex issue of diagnostic issues in relation to symptom overlap and (iii) to provide more reliable normative data in which to compare (for example taking age into account).

Further to this, let us consider that even if ASD and schizophrenia share similar phenotypes (which is apparent in Chapter 9), they may not share the same underlying aetiology. Perhaps a more accurate way of modelling the E-S theory and the IBT, would be to discount the continuum model and instead consider that it is plausible that phenotypes are connected in a non-linear fashion. It seems reasonable, based on this thesis, that the EMB and the EFB profiles should be thought of as phenotypically separate from the E-S theory.

Additionally, it was revealed that the majority of previous research into the EFB concept has failed to fully take into account the role of systemising. In that, experimental paradigms often only involve measures of empathising, or disregard the results pertaining to systemising. As a consequence of not including, or removing systemising in the experimental paradigms, investigators are treating empathising and systemising as two separate entities, when the hypothesis of the E-S theory is that they are connected on a continuum. This thesis strongly recommends that academics aiming to investigate 'brain types' such as the EFB, *must* consider the role of systemising to the same extent as empathising in order to accurately investigate the EFB profile. Therefore, it is argued that the data presented in

this thesis provides the field of research with a more defined picture of the type E profile, by including methods that measure both empathising and systemising equally.

Shortly after the experimental data collection period of this thesis was concluded, Bernard Crespi and colleagues (Dinsdale et al., 2016) reported a new study that claimed a more accurate EFB characterisation may be that of borderline personality disorder (BPD). Their results (meta-analysis) communicate that performance on the RMTE task is enhanced in sub-clinical depression and preserved in BPD, suggesting that together with the high female to male ratio, BPD is a more accurate fit with the EFB pathological consequence. However, once again, Dinsdale et al. (2016) failed to adequately take systemising into account, highlighting the obvious neglect in the literature for this imperative consideration. In the same paper, Dinsdale et al. (2016) acknowledge that empathising and systemising *should* trade-off based on the mediating effect of fT, yet, failed to investigate the role of systemising in their investigation. Instead, they concentrated solely on hyper-empathising in an aim to characterise the EFB. Whilst it is suggested here that Dinsdale's et al. (2016) findings are useful and promising results that are worthy of further investigation, it is the position of this thesis that, as yet, the authors can only claim to have established an association between BPD and hyper-empathising – not an association between BPD the EFB profile. It is argued here that this thesis is one of the very few works that *comprehensively* investigates systemising (as well as empathising) in relation to psychopathological traits in an attempt to accurately explore the potential pathology of the EFB profile.

Since the conclusion of the data collection period of this thesis, Baron-Cohen and colleges (Larson et al., 2015) directly responded to the hypothesis that the IBT provides a fitting model equipped to characterise the EFB profile. Larson et al. (2015) investigated the relationship between the EFB and psychosis in a clinical sample (the first study to do so), they compared empathising bias (see section 8.3.2) in two groups; one group was diagnosed with ASD without psychosis and the other was diagnosed with ASD with co-morbid psychosis. Results showed that the co-morbid psychosis group had a greater empathising bias than the ASD without psychosis group, suggestive of a link between empathising bias and psychosis. However, the authors note that on greater examination of the results, the 'driver' for this pattern was higher than average I.Q (Larson et al., 2015). In addition, it was recognised that the sample as a whole had a preference for systemising over empathising, as would be expected in an ASD sample. Therefore, at best, those with a bias for empathising could only be considered as 'balanced brains', not 'female brains'. Larson et al. (2015) concluded that the IBT model is much too simplistic in its current form - even if there is a relationship between empathising and psychosis. They offer that a plausible explanation for the lack of empirical evidence for the ASD – schizophrenia continuum, (as was evident in this thesis), is that rather than a bias in empathising and systemising playing a causal role in the manifestation of psychosis, it may be more accurate to consider that the manifestation of psychosis subsequently effects empathising and systemising ability. A notion that is worthy of further research.

Furthermore, this work has demonstrated that the E-S theory is capable of incorporating additional markers of cognitive function (memory capacity) that can predict where a person places on the continuum, thus significantly extending the knowledge pertaining to the E-S theory. This finding demonstrated that memory for social stimuli was greater in those who scored as higher empathisers and lower systemisers. Does this finding support the notion that social cognitive neuroscience should consider the role of biological sex, or empathising and systemising ability in emotional processing? In support of this, Cahill et al. (2007) suggests that neurobiology must take into account the importance of gender/sex differences in emotional processing. Is it the case that those with greater empathising skills are able to encode emotional or social memory with greater detail? Does feature binding work better in empathisers due to a better underlying ability to understand and identify social context? Further research is required here, as these novel findings not only extend the knowledge of additional cognitive markers on the E-S continuum, but also offer the first support (to the author's knowledge) for the theoretical predictions of the IBT, in that people who present as empathisers will show significantly better episodic memory alongside a greater susceptibility to source monitoring error, *presumably* due to the links between psychosis and the inability to distinguish true from false memories (Badcock, 2011).

The results of thesis contribute to the discussion surrounding the importance of developing robust diagnostic processes in relation to the disorders that have been discussed throughout this thesis. For instance, Van Schalkwyk et al. (2015) offer that there is great potential for misdiagnosis in ASD and

schizophrenia, due to the relative overlap of symptoms (a conception that was evident in this thesis in trait samples only). In a series of case studies, they concluded that often, psychotic episodes that are observed in ASD would be better understood as part of ASD symptomology rather than a separate diagnosis of schizophrenia, which in turn, causes difficultly with putting into place correct treatment plans. This, along with the implications discussed above, signifies the importance of continuing to explore the *exact* nature of the relationship between ASD and schizophrenia phenotypes.

In conclusion, the implications for the field of social cognitive neuroscience in terms of the accuracy of the IBT in characterising the EFB, are informative and offer an opportunity to advance the knowledge pertaining to the feminisation of the brain both at a healthy and extreme level. However, one *key* finding was that there was no apparent trade-off between empathising and systemising, on either self-report or direct performance measures. As this thesis progressed it became apparent that the lack of support for the trade-off relationship between empathising and systemising (Chapter 10 [and consistently replicated throughout the thesis]) was disconcerting and has highlighted how much of a consideration this discovery *must* be, in any further investigation into the E-S theory model. If this notion is ignored, it is argued here that investigations into the EFB profile may be compromised. The finding that age-related effects are significantly implicated in the trade-off relationship between empathising and systemising was an imperative finding of this work and one that has not been considered in previous studies (as far as the researcher is aware). This has demonstrated the importance of considering confounding variables such as age in terms of the trade-off

relationship between empathising and systemising. The field must therefore consider that the presumed trade-off is not consistent throughout the lifespan. Ultimately, the trade-off between empathising and systemising is the concept on which the EFB profile is built.

11.4 Considerations and future directions

In today's political climate, it is important to touch upon the important concept of neurosexism in scientific research (Fine, 2010b; Fine et al., 2013). Baron-Cohen (2007) addresses these issues by emphasising that what the E-S theory refers to is 'averages' and that these averages have 'very little to do with individuals' (p. 159). He suggests that the political climate of the 1960/70's made an open-minded discussion of sex differences impossible for science, but that in modern times, thankfully, the debate surrounding biology and how it influence sex differences is met with more impartiality. Baron-Cohen (2007) states that his own personal view is that science should be sensitive to gender inequalities and not over-state any conclusions. He goes on to suggest that the fact that his work focuses on statistical averages and says nothing about the individual meets this requirement. He stresses that the E-S theory suggests that individuals can be atypical for their sex, and this demonstrates that the E-S theory requires you to look at individuals on their own merit and does not endorse a strict gender dependent cognitive profiles. Baron-Cohen (2007) goes on to recommend that scientific enquiry into sex differences needs to remain separate from social policy discussion, that the E-S theory is looking at cognitive abilities, not pre-determined abilities in accordance with biological sex. However, it could be argued here that any

theory that uses the terms 'male and female brains' is of course relevant to the debate on how biological sex differences are applicable in a social context. This is potentially dangerous territory that requires sensitivity to the consequences of endorsing political power structures (Nash, 2014). Whilst rigorous scientific enquiry is vital to further our understanding of sex differences, researchers in the field have a moral duty to appreciate the historical landscape of political agenda and power play, and interpret findings with caution and sensitivity.

In terms of the experimental paradigm employed here, this thesis administered direct tests of cognitive empathy, but *not* affective empathy. The reason for this methodological choice was that a developed and validated test of affective empathy was not available and it was deemed important to continue the trend in the literature by employing the most popular measures of empathising in order to be comparable with previous studies. This is an important consideration for future studies as there is evidence to suggest a dissociation between cognitive and affective empathy processes (Shamay-Tsoory et al., 2007; Kalbe et al., 2010), thus this issue should be a considered in relation to the findings here, and addressed in further study. However, there is also evidence to suggest that cognitive and affective empathy are significantly connected (Wlodarski, 2015) which infers results employing affective empathy tasks are likely to be very similar to the ones reported in this thesis.

In terms of how to continue with the investigation into the EFB, E-S and IBT concepts, the field of study is in need of research that specifically identifies the neuronal mechanisms that drive empathising and systemising (Baron Cohen, 2009; Dinsdale et al., 2016). Whilst the neurological basis of empathy is progressing (Baron-Cohen, 2009), the knowledge in relation to the brain basis of systemising is vague (Chapter 3). Future research is required in order to explore the neurological basis of empathising and systemising, both separately and as a possible connected network as this area of debate also remains unresolved. It is also important to take this research beyond behavioural data by measuring the electroencephalographic (EEG) mu rhythm as a proxy for measuring mirror neuron (MN) activity for instance. MN dysfunction has not previously been investigated in terms of the E-S theory, and is the next logical step (see Chapter 2). Previous research has demonstrated that MN activity is associated with empathic ability in normal cognition (Zaki et al 2009; Hooker et al 2010) and dysfunction of this system is apparent in ASD (Iacoboni and Dapretto, 2006; Rizzolatti and Craighero, 2005). Interestingly, MN activity, in relation to schizophrenia conditions is unclear; studies report contrasting results. For instance, Enticott et al (2008) found MN activity is reduced in schizophrenic patients and shows a similar pattern to that found in autistic patients, on the other hand Quintana et al (2001) and McCormack et al (2012) concluded that MN activation is higher in schizophrenia than it is in controls, along with a correlate of severity. Whilst the notion of the IBT struggles with the results presented here in this thesis, exploration of the IBT at a neuronal level may simplify the seemingly complex relationship between ASD and schizophrenia (plus sub-types), and

empathising and systemising. It would be expected if the ASD – schizophrenia continuum was evident at a neuronal level that MN activity would be reduced in the male brain and heightened in the female brain.

Additionally, an interesting future study, which would have implications at the clinical level would be an exploration of the E-S model in relation to hormones and the neuroendocrine system in general. The menstrual cycle for example, is the major biological distinction between males and females; it controls the variation in sex hormones across the monthly cycle and significantly influences sexual differentiation in brain structure and neurochemistry (Cosgrove et al., 2007). The role of oestrogen is an important area of research pertaining to the EFB profile. It has already been established that oestrogen regulation affects learning and memory processes (Gillies & McArthur, 2010). Therefore, future studies should aim to further the knowledge related to the role of oestrogen in the determination of E-S brain profiles. Gillies and McArthur (2010) suggest if the field of neurobiology can unravel the exact process of oestrogen effects, there is a promising possibility of developing oestrogen based therapies that may be useful for the treatment of brain disorder.

In relation to future studies concerning behavioural data, there are still areas for investigation. For instance, even though, this thesis must reject the postulation of the characterisation of the EFB profile with complete schizophrenia pathology, in order to *comprehensively* investigate Badcock & Crespi's (2008) theory of the ASD – schizophrenia continuum, further research employing clinical populations is warranted. This thesis can only claim to apply its' findings to non-clinical populations and trait behaviours.

Lastly, in light of the new findings relating to age-related effects in the relationship between empathising and systemising, longitudinal research would be useful to investigate any apparent changes over time and any specific influence of these changes, such as pregnancy or menopause in order to extend the knowledge of this new observation.

11.5 Thesis conclusion

This thesis investigated the potential pathology and cognitive attributes of the EFB. The EFB profile is derived from the E-S theory, which hypothesises that sex differences in cognition exist on a continuum, based on abilities in empathising and systemising (Baron-Cohen, 2003). The EFB profile is defined as advanced empathising alongside deficient systemising (Baron-Cohen, 2003) and compared to the EMB profile has received little attention in social cognitive neuroscience research to date. This thesis revealed that episodic memory ability for social situations was able to significantly predict where a person lies on the E-S continuum, suggesting support for a new cognitive dimension that is implicated in the E-S model. Regarding potential pathology, results from this thesis showed that individual elements associated with paranoia (such as 'jumping to conclusions') were associated with an empathising bias. However, a bias in systemising ability was also associated with schizotypy, along with a significant overlap between the expression of autistic traits and negative expressions of schizotypy. Therefore, considering the results of this thesis collectively, it is argued that whilst the specific phenotypes of positive schizotypy have demonstrated associative links with the type E profile, which offers some degree of support

for the hypothesis of the IBT, the notion that *all* elements of positive schizotypy *combined* are associated with the EFB is too general.

Furthermore, it is essential to look at age in any investigation of a trade-off between empathising and systemising. These findings are novel, and will allow the field of social cognitive neuroscience, biological psychology, clinical psychology, neuropsychiatry, evolutionary psychology and cognitive psychology to progress. These results have significant implications for the assessment, diagnosis and treatment of neuropsychological disorders.

Future research should look at investigating the neural basis of empathising and systemising by employing a wide range of neuroimaging methods to further advance the knowledge of how these two cognitive processes are represented in the brain. The next logical step would be to investigate the mu rhythm in relation to MNs and their role in empathising and systemising and 'brain types'. This should be combined with investigations of other cognitive functions such as attention, perception, and language to get a fully informed picture of brain types, cognitive processes and neural function.

References

Abdi, Z. and Sharma, T. (2004) 'Social cognition and its neural correlates in schizophrenia and autism'. *CNS Spectrums*, 9, pp.335-343

Abu-Akel, A. M., Wood, S. J., Hansen, P. C. and Apperly, I. A. (2015) 'Perspective-taking abilities in the balance between autism tendencies and psychosis proneness'. *Proceedings of the Royal Society B: Biological Sciences*, 282, 20150563, dx.doi.org/10.1098/rspb.2015.0563

Abu-Akel, A. (2008) 'Theory of mind in autism, schizophrenia and in-between'. *Behavioural and Brain Sciences,* 31:3, pp.261-262

Achim, A. M., Ouellet, R., Roy, M. A. and Jackson, P. L. (2010) 'Assessment of empathy in first-episode psychosis and meta analytic comparison with previous studies in schizophrenia'. *Psychiatry Research,* 190:1, pp.03-08

Airaksinen, E., Larsson, M. and Forsell, Y. (2004) 'Neuropsychological functions in anxiety disorders in population-based samples: evidence of episodic memory dysfunction'. *Journal of Psychiatric Research*, 39, pp.207-214

Aleman, A. and Kahn, R. S. (2005) 'Strange feelings: do amygdala abnormalities dysregulate the emotional brain in schizophrenia?'. *Progress in Neurobiology*, 77, pp.283–298

Aleman, A., Hijman, A. R., de Haan, E. H. F. and Kahn, R. S. (1999) 'Memory impairment in schizophrenia a meta-analysis'. *American Journal of Psychiatry*, 156:9, pp.1358-1366

Allen, J. S., Damasio, H., Grabowski, T. J. Bruss, J. and Zhang, J. (2003) 'Sexual dimorphism and asymmetries in the grey–white composition of the human cerebrum'. *NeuroImage*, 18:4, pp.880–894

Allen, L. S., Richey, M. F., Chai, Y. M. and Gorski, R. A. (1991) 'Sex differences in the corpus callosum of the living human being'. *The Journal of Neuroscience*, 11:4, pp.933-942

Alexander G. M., Hines M. (2002) 'Sex differences in response to children's toys in nonhuman primates (Cercopithecus aethiops sabaeus)'. *Evolution and Human Behaviour*, 23:4, pp.67–79

Altemus, S. M. (2006) 'Sex differences in depression and anxiety disorders: potential biological determinants'. *Hormones and Behaviour*, 50, pp.534-538

Anderson, L. and Shimamura, A. P. (2005) 'Influences of emotion on context memory while viewing film clips'. *American Journal of Psychology*, 118, pp.323-337

Andia, A. M., Zisook, S., Heaton R. K., Hesselink, J., Jernigan, T., Kuck, J., Moranville, J. and Braff, D. L. (1995) 'Gender differences in schizophrenia.' *Journal of Nervous and Mental Disease*, 183:8, pp.522–528

Andreasen, N. C. (1982) 'Negative symptoms in schizophrenia: definition and reliability'. *Archives of General Psychiatry*, 39:7, doi:10.1001/archpsyc. 1982.04290070020005

Andrew, J., Cooke, M. and Muncer, S. J. (2008) 'The relationship between empathy and machiavellianism: an alternative to empathizing- systemizing theory'. *Personality and Individual Differences*, 44, pp.1203-1211

Andrews, S. C., Enticott, P. G., Hoy, K. E., Thomson, R. H. and Fitzgerald, P. B. (2015) 'No evidence for mirror system dysfunction in schizophrenia from a multimodal TMS/EEG study'. Psychiatry Research, 228:3, pp.421-440

Armstrong, K. A. and Nigar, G. (2002) 'Gender differences in anxiety: an investigation of the symptoms, cognitions, and sensitivity towards anxiety in a non-clinical population'. *Behavioural and Cognitive Psychotherapy*, 30, pp.227-231

Auyeung, B., Allison, C., Wheelwright, S. and Baron-Cohen, S. (2012) 'Brief report: development of the adolescent empathy and systemising quotients'. *Journal of Autism and Developmental Disorders*, 42, pp.2225-2235

Auyeung, B., Knickmeyer, R., Ashwin, E., Taylor, K., Hackett, G. and Baron-Cohen, S. (2011) 'Effects of fetal testosterone on visuospatial ability'. *Archives of Sexual Behavior*, 41:3, pp.571-581

Auyeung, B., Taylor, K., Hackett, G. and Baron-Cohen, S. (2010) 'Foetal testosterone and autistic traits in 18 to 24-month-old children'. *Molecular Autism*, 1, pp.1-11

Auyeung, B., Baron-Cohen, S., Ashwin, E., Knickmeyer, R., Taylor, K. and Hackett, G. (2009) 'Fetal Testosterone and Autistic Traits'. *The British Journal of Psychology*, 100, pp.01-22

Badcock, C. (2009) *The imprinted brain: how genes set the balance between autism and psychosis*. London: Jessica Kingsley Publishers

Badcock, C. and Crespi, B. (2008) 'Battle of the sexes may be set in the brain'. *Nature*, 454:7205, pp. 1054-1055

Badcock, C. and Crespi, B. (2006) 'Imbalanced genomic imprinting in brain development: an evolutionary basis for the aetiology of autism'. *Journal of Evolutionary Biology*, 19:4, pp.1007-1032

Bailey, P. E., Henry, J. D. and Von Hippel, W. (2007) 'Empathy and social functioning in late adulthood'. *Aging and Mental Health*, 12:4, pp.499-503

Baird, G., Simonoff, E., Pickles, A., Chandler, S. and Loucas, T. (2006) 'Prevalence of disorders of the autism spectrum in a population cohort of children in South Thames: the Special Needs and Autism Project (SNAP)'. *Lancet,* 368, pp.210–215

Baltaxe, C. A. and D'Angiola, N. (1992) 'Cohesion in the discourse interaction of autistic, specifically language-impaired, and normal children'. *Journal of Autism and other Developmental Disorders*, 22:1, pp.1-21

Banaji, M. R. and Hardin, C. D. (1996) 'Automatic Stereotyping'. *Psychological Science,* 7:3, pp.136-143

Bangasser, D. A., Curtis, A., Reyes, B. A. S., Bethea, I. P., Ischiropoulos, H., Van Bockstaele, E. J. and Valentino, R. J. (2010) 'Sex differences in corticotropin-releasing factor receptor signalling and trafficking: potential role in female vulnerability to stress-related psychopathology'. *Molecular Psychiatry*, 15:9, pp.877-904

Bao, A-M. and Swaab, D. F. (2010) 'Sex differences in the brain, behaviour, and neuropsychiatric disorders'. *The Neuroscientist*, 16:5, pp.550-565

Bardeau, E. B., Mendrek, A. and Moltron, L. (2009) 'Are autistic traits autistic?'. *British Journal of Psychology,* 100:1, pp.23-29

Barneveld, P. S., Pieterse, J., de Sonneville, L., van Rijn, S., Lahuis, B., van Engeland, H. and Swaab, H. (2011) 'Overlap of autistic and schizotypal traits in adolescents with autism spectrum disorders'. *Schizophrenia Research*, 126, pp.231-236

Baron-Cohen, S., Cassidy, S., Auyeung, B., Allison, C., Achoukhi, M., Robertson, S., Pohl, A. and Meng-Chuan, L. (2014) 'Attenuation of typical sex differences in 800 adults with autism vs. 3,900 controls'. *PLOS one*, 9:7, doi:10.1371/journal.pone.0102251

Baron-Cohen, S., Jaffa, T., Davies, S., Auyeung, B., Allison, C. and Wheelwright, S. (2013) 'Do girls with anorexia nervosa have elevated autistic traits?'. *Molecular Autism*, 4:24, doi:10.1186/2040-2392-4-24

Baron-Cohen, S., Lomdardo, M. V., Auyeung, B., Ashwin, E., Chakrabarti, B. and Knickmeyer, R. (2011) 'Why are autistic spectrum conditions more prevalent in males?'. *PLoS Biology,* 9:6, pp.1-10

Baron-Cohen, S. (2010) Empathizing, systemizing, and the extreme male brain theory of autism. *In*: Savic, I., ed. *Progress in Brain Research; Sex differences in the human brain, their underpinnings and implications*. 186. Academic Press, pp. 167- 175

Baron-Cohen, S. (2009) 'Autism: The empathising- systemising (E-S) theory'. *The Year in Cognitive Neuroscience 2009*, 1156, pp.68-80

Baron-Cohen, S. (2008) 'Autism, hyper systemising and truth'. *The Quarterly Journal of Experimental Psychology*, 61:1, pp.64-75

Baron-Cohen, S. (2007) Sex differences in mind: keeping science distinct from social policy. *In:* Ceci, S. J. and Williams, W. M., eds. Why aren't more women in science? Top researchers debate the evidence. Washington D.C: American Psychological Association, pp.159-172

Baron-Cohen, S. and Belmonte, M. K. (2005) 'Autism: a window onto the development of the social and analytic brain'. *Annual Review Neuroscience*, 28, pp.109-126

Baron-Cohen, S., Knickmeyer, R. C. and Belmonte, M. K. (2005) 'Sex differences in the brain: implications of explaining autism'. *Science*, 310, pp.819-823

Baron-Cohen, S. (2004) 'The cognitive neuroscience of autism'. *Journal of Neurology, Neurosurgery and Psychiatry*, 75, pp.945-948

Baron-Cohen, S. and Wheelwright, S. (2004) 'The empathy quotient: an investigation of adults with Asperger's syndrome or high functioning autism and normal sex differences'. *Journal of Autism and other Developmental Disorders*, 34(2), pp.163-178

Baron-Cohen, S. (2003) *The essential difference*. London: Penguin Books

Baron-Cohen, S., Richler, J., Bisarya, D., Gurunathan, N. and Wheelwright, S. (2003) 'The systemising quotient (SQ): an investigation of adults with Asperger's syndrome or high-functioning autism and normal sex differences'. *Philosophical Transactions of the Royal Society*, 358, pp.361-374

Baron-Cohen, S. (2002) 'The extreme male brain theory of autism'. *Trends in Cognitive Sciences*, 6:6, pp.248-254

Baron-Cohen, S., Wheelwright, S., Lawson, J., Griffin, R. and Hill, J. (2002) The exact mind: empathizing and systemizing in autism spectrum conditions. *In:* Goswani, U., ed. *Blackwell Handbook of Childhood Cognitive Development.* Malden, USA: Blackwell Publishers Ltd. pp.491-508

Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y. and Plumb, I. (2001) 'The "reading the mind in the eyes" test revised version: a study with normal adults, and adults with Asperger syndrome or high-functioning autism'. *Journal of Child Psychology and Psychiatry*, 42:2, pp.241-251

Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J. and Clubley, E. (2001) 'The autism-spectrum quotient (AQ): evidence from Asperger syndrome/high-functioning autism, males and females, scientists and

mathematicians'. *Journal of Autism and Developmental Disorder,* 31:1, pp.5-17

Baron-Cohen, S., Wheelwright, S., Spong, A., Scahill, V. and Lawson, J. (2001) 'Are intuitive physics and intuitive psychology independent? A test with children with Asperger's syndrome'. *Journal of Developmental and Learning Disorders*, 5, pp.47-78

Baron-Cohen, S., Wheelwright, S., Stone, V. and Rutherford, M. (1999) 'A mathematician, a physicist and a computer scientist with Asperger Syndrome: performance on a folk psychology and folk physics test'. *Neurocase*, 5, pp.475-483

Baron-Cohen, S., Ring, H., Wheelwright, S., Bullmore, E. T. and Brammer, M. J. (1999) 'Social intelligence in the normal and autistic brain: an fMRI study'. *European Journal of Neuroscience*, 11, pp.1891–1898

Baron-Cohen, S. (1997) *Mindblindness: an essay on autism and theory of mind.* USA: MIT Press

Baron-Cohen, S. and Hammer, J. (1997) 'Is autism an extreme form of the 'male brain?'. *Advances in Infancy Research,* 11, pp.193-217

Baron-Cohen, S., Jolliffe, T., Mortimore, C. and Robertson, M. (1997) 'Another advanced test of theory of mind: evidence from very high functioning adults with autism or Asperger syndrome'. *Journal of Child Psychology and Psychiatry*, 38:7, pp.813-822

Baron-Cohen, S., Leslie, A. M. and Frith, U. (1985) 'Does the autistic child have a theory of mind?'. *Cognition*, 21:1, pp.37-46

Barrantes-Vidal, N., Lewandowski, K. E. and Kwapil, T. R. (2010) 'Psychopathology, social adjustment and personality correlates of schizotypy clusters in a large nonclinical sample'. *Schizophrenia Research,* 1:3, pp.219-125

Batey, M. and Furnham, A. (2008) 'The relationship between measures of creativity and schizotypy'. *Personality and Individual Differences*, 45, pp.816-821

Bauman, M. L. and Kemper, T. L. (2005) 'Neuroanatomic observations of the brain in autism: a review and future direction'. *International Journal of Developmental Neuroscience, 23*: pp.183–187

Bauste, G. and Ferraro, R. (2004) 'Gender differences in false memory production'. *Current Psychology*, 23:3, pp.238-244

Baxter, L. C., Saykin, A. J., Flashman, L. A., Johnson, S. C., Guerin, S. J., Babcock, D. R. and Wishart, H. A. (2003) 'Sex differences in semantic language processing: a functional MRI study'. *Brain and Language*, 84:2, pp.264-272

Beery, A. K. and Zucker, I. (2011) 'Sex bias in neuroscience and biomedical research'. *Neuroscience & Biobehavioural Reviews*, 35:3, pp.565-572

Benbow, C. P. (1988) 'Sex differences in mathematical reasoning ability in intellectually talented preadolescents: their nature, effects, and possible causes'. *Behavioral Brain Sciences*, 11, pp.169–232

Bentall, R. P., Baker, G. A. and Havers, S. (1991) 'Reality monitoring and psychotic hallucinations', British Journal of Clinical Psychology 30:3, pp.213–222

Benton, D. and Young, H. A. (2015) 'Do small differences in hydration status affect mood and mental performance?' *Nutrition Reviews*, 73:2, pp.86-96

Benton, D. (2011) 'Dehydration influences mood and cognition: a plausible hypothesis?' *Nutrients*, 3:5, pp.555-573

Ben Shalom, D. (2003) 'Memory in autism: review and synthesis'. *Cortex*, 39, pp.1129-1138

Beversdorf, D. Q., Smith, B. W., Crucian, G. P., Anderson, J. M., Keillor, J. M., Barrett, A. M., Hughes, J. D., Felopulos, G. J., Bauman, M. L., Nadeau, S. E. and Heilman, K. M. (2000) 'Increased discrimination of 'false memories' in autism spectrum disorder'. *PNAS*, 97:15, doi:10.1073/pnas.97.15.8734

Bhatt, R., Laws, K. P. and McKenna, P. J. (2010) 'False memory in schizophrenia patients with and without delusions'. *Psychiatry Research*, 178:2, pp.260-265

Billington, J., Baron-Cohen, S. and Bor, D. (2008) 'Systemizing influences attentional processes during the Navon task: an fMRI study'. *Neuropsychologia,* 46, pp.511-520

Billington, J., Baron-Cohen, S. and Wheelwright, S. (2007) 'Cognitive style predicts entry into physical sciences and humanities: questionnaire and performance tests of empathy and systemising'. *Learning and Individual Differences*, 17:3, pp.260-268

Binbay, T., Drukkler, M., Elbi, H., Tank, F.A., Ozkinay, F., Onay, H., Zagh, N., van Os, J. and Alptekin, K. (2011) 'Testing the psychosis continuum: differential impact of genetic and nongenetic risk factors and comorbid psychopathology across the entire spectrum of psychosis'. *Schizophrenia Bulletin*, 38:5, pp.992-1002

Blackshaw, A. J., Kinderman, P., Hare, D. J. and Hatton, C. (2001) 'Theory of mind, casual attribution and paranoia in Asperger syndrome'. *Autism*, 5:2, pp.147-163

Blanchard, J. J and Cohen, A. S. (2006) 'The structure of negative symptoms within schizophrenia: implications for assessment'. *Schizophrenia Bulletin*, 32:2, pp. 238-245

Blechert, J. and Meyer, T. D. (2005) 'Are measures of hypomanic personality, impulsive nonconformity and rigidity predictors of bipolar symptoms?'. *British Journal of Psychology*, 44:1, pp.15-27

Bourne, H. (2004) 'Parental age difference and schizophrenia'. *British Journal of Psychiatry*, 184, pp.540-541

Bowler, D. M., Gardiner, J. M., Grice, S. and Saavalainen, P. (2000) 'Memory illusions: False recall and recognition in adults with Asperger's syndrome'. *Journal of Abnormal Psychology*, 109, pp.663–672

Brébion, G., Bressan, R. A., Ohlsen, R. I. and David, A. S. (2013) 'A model of memory impairment in schizophrenia: cognitive and clinical factors associated with memory efficiency and memory errors'. *Schizophrenia Research*, 151:1-3, pp.70-77

Brébion, G., Gorman, J. M., Amador, X., Malaspina, D. and Sharif, Z. (2002) 'Source monitoring impairments in schizophrenia: characterisation and associations with positive and negative symptomatology'. *Psychiatry Research*, 112:1, pp.27–39

Brébion, G., Amador, X., Smith, M. J., Malaspina, D., Sharif, Z. and Gorman, J. M. (1999) 'Opposite links of positive and negative symptomology with memory errors in schizophrenia'. *Psychiatry Research*, 88:1, pp.15-24

Bright-Paul, A., Jarrold, C. and Wright, D.B. (2008) 'Theory-of-mind development influences suggestibility and source monitoring'. *Developmental psychology*, 44:4, pp.1055–68.

Brosnan, M., Hollinworth, M., Antoniadou, K. and Lewton, M. (2014) 'Is empathizing intuitive and systemizing deliberative?'. *Personality and Individual Differences*, 66, pp.39-43

Brosnan, M., Ashwin, C. and Gamble, T. (2011) 'Greater empathizing and reduced systemizing in people who show jumping to conclusions bias in the general populations: implications for psychosis'. *Psychosis, Psychological, Social and Integrative Approaches*, 5:1, pp.71-81

Brosnan, M., Ashwin, C., Walker, I. and Donaghue, J. (2010) 'Can and extreme female brain be characterised in terms of psychosis?'. *Personality and Individual Differences*, 49, pp.738-742

Brown, W. M., Hines, M., Fane, B. A. and Breedlove, S. (2002) 'Masculinized finger length patterns in human males and females with congenital adrenal hyperplasia'. *Hormones and Behaviour*, 42, pp.380–383

Bucca, A. (2012) 'The shared ideation of the paranoic delusion. Implications of empathy, theory of mind and language'. *Journal of Psychopathology*, 18, pp.383-388

Buchy, L., Woodward, T. S. and Liotto, M. (2007) 'A cognitive bias against disconfirmatory evidence (BADE) is associated with schizotypy'. *Schizophrenia Research*, 90:1-3, pp.334-337

Buckner, R. L. and Carroll, D. C. (2007) 'Self-projection and the brain'. *Trends in Cognitive Sciences,* 11:2, pp.49-57

Burbach, J. P. H. and van der Zwaa, B. (2009) 'Contact in genetics of autism and schizophrenia'. *Trends in Neurosciences*, 32, pp.62-72

Burkitt, J., Widman, D. and Saucier, D. M. (2007) 'Evidence for the influence of testosterone in the performance of spatial navigation in a virtual water maze in women but not men'. *Hormones and Behaviour,* 51, pp.649-654

Burne, T. H. J., Eyles, D. W. and McGrath, J. J. (2008) 'Animal models may help fractionate shared and discrete pathways underpinning Schizophrenia and Autism'. *Brain and Behavioural Sciences,* 33:1, pp.24-25

Cadenhead, K. S. and Braff, D. L. (1999) Schizophrenia spectrum disorders. *In:* Dawson, M. E., Schell, A. M. and Böhmelt, A. H., eds. *Startle modification: implications of neuroscience, cognitive science, and clinical science*. USA: Cambridge University Press

Carroll, J. M. and Yung, C. K. (2006) 'Sex discipline differences in empathising, systemising and autistic symptomatology: evidence from a student population'. *Journal of Autism and Developmental Disorders*, 36:7, pp.949-957

Cahill, L. (2014) 'Equal the same: sex differences in the human brain'. Cerebrum, The DANA Foundation [online] Available at: <http://www.dana.org/Cerebrum/2014/Equal_%E2%89%A0_The_Same__Sex_ Differences_in_the_Human_Brain/> [Accessed January 2016].

Cahill, L. (2014) 'Fundamental sex difference in human brain architecture'. *PNAS*, 111:2, pp.577-578

Cahill, L. (2006) 'Why sex matters for neuroscience'. *Nature Reviews Neuroscience,* 7:6, pp.477-484

Cahill, L., Haier, R. J., White, N. S., Fallon, J., Kilpatrick, L., Lawrence, C. and Alkire, M. T. (2001) 'Sex-related differences in amygdala activity during emotionally influenced memory storage'. *Neurobiology of Learning and Memory*, 75, pp.1–9

Carr, L., Iacoboni, M., Dubeau, M. C., Mazziotta, J. C. and Lenzi, G. L. (2003) 'Neural mechanisms of empathy in humans: A relay from neural systems for imitation to limbic areas'. *Proceedings of the National Academy Science,* 100, pp.5497–5502

Carstensen, L. L., Turan, B., Scheibe, S., Ram, N., Ersner-Hershfield, H., Samanez-Larkin, G. R., Brooks, K. P. and Nasselroade, J. R. (2011) 'Emotional experience improves with age: evidence based on over 10 years of experience sampling'. *Psychology and Aging*, 26:1, pp.21-33

Cash, T. F. (1973) 'Methodological problems and progress in schizophrenia research: a survey' [Abstract]. *Journal of Consulting and Clinical Psychology*, 40:2, pp.278-286

Chan, K. K. S. and Chen, E. Y. H. (2011) 'Theory of mind and paranoia in schizophrenia: a game theoretical investigation framework'. *Cognitive Neuropsychiatry*, 16:6, pp.505-29

Chen, X., Sachdev, P. S., Wen, W. and Anstey, K. J. (2007) 'Sex differences in regional grey matter in healthy individuals aged 44–48 years: a voxel-based morphometric study'. *NeuroImage*, 36:3, pp. 691–699

Cheug, Y., Chou, K. H., Decety, J., Chen, I. Y., Hung, D., Tzeng, O. J. L. and Lin, C. P. (2009) 'Sex differences in the neuroanatomy of human mirror neuron system: a voxel-based morphometric investigation'. [Abstract] *Neuroscience,* 158:2, pp.713-720

Chou, K. H., Cheug, Y., Chen, I. Y., Lin, C. P. & Chu, W. C. (2011) 'Sex linked white matter microstructure of the social and analytic brain'. *NeuroImage*, 54, pp.725-733

Ciaramelli, E., Bernardi, F. and Moscovitch, M. (2013) 'Individualized theory of mind (iToM): when memory modulates empathy. *Frontiers in Psychology*, Available at: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3561727& tool=pmcentrez&rendertype=abstract.

Claridge, G. and Blakley, S. (2009) 'Schizotypy and affective temperament: relationships with divergent thinking and creativity styles'. *Personality and Individual Differences*, 46, pp.820-826

Cochrane, M., Petch, I. and Pickering, A. D. (2010) 'Do measures of schizotypal personality provide non-clinical analogues of schizophrenic symptomology? *Psychiatry Research*, 176, pp.150-154

Collaer, M. L., and Hines, M. (1995) 'Human behavioural sex difference: a role for gonadal hormones during early developments' [Abstract]. *Psychological Bulletin*, 118:1, pp.55-107

Combs, D. R, Penn, D. L., Chadwick, P., Trower, P., Michael, C. O. and Basso, M. R. (2007) 'Subtypes of paranoia in a nonclinical sample'. *Cognitive Neuropsychiatry*, 12:6, pp.537-553

Connellan, J., Baron-Cohen, S., Wheelwright, S., Ba'tki, A. and Ahluwalia, J. (2001) 'Sex differences in human neonatal social perception'. *Infant Behavior and Development*, 23, pp.113-118

Constantino, J. and Todd, R. D. (2003) 'Autistic traits in the general population'. *Archives Genetic Psychiatry,* 60, pp.524-531

Cook, C. M. and Saucier, D. M. (2010) 'Mental rotation, targeting ability and Baron-Cohen's Empathising-Systemising theory of sex differences'. *Personality and Individual Differences*, 49, pp.712-716

Cook, G. I., Hicks, J. L., & Marsh, R. L. (2007). 'Source monitoring is not always enhanced for valenced material'. *Memory and Cognition*, 35, 222–230

Cosgrove, K. P., Mazure, C. M. and Staley, J. K. (2007) 'Evolving knowledge of sex differences in brain structure, function, and chemistry'. *Biological psychiatry*, 62:8, pp.847–55

Courchesne, E., Chisum, H. J., Townsend, J., Cowles, A., Covington, J., Egaas, B., Harwood, M., Hinds, S. and Press, G. A. (2000) 'Normal brain development and aging: quantitative analysis at in vivo MR imaging in healthy volunteers'. *Radiology*, 216, pp.672–682

Cowell, P. E., Kostianovsky, D. J., Gur, R. C., Turetsky, B. I. and Gur, R. E. (1996) 'Sex differences in neuroanatomical and clinical correlations in schizophrenia'. *American Journal of Psychiatry*, 153:6, pp.799–805,

Craddock, N. and Owen, M. J. (2010) 'Revisiting Bleuler: relationship between autism and schizophrenia' [author's reply]. *The British Journal of Psychiatry*, 196, pp.495-497

Craddock, N., Kendler, K., Neale, M., Numberger, J., Purcell, S., Rietshel, M., Perlis, R., Santangel, S. L., Schulze, T., Smoller, J. W. and Thapar, A. (2009) 'Dissecting the phenotype in genome-wide association studies of psychiatric illness'. *The British Journal of Psychiatry*, 195:2, pp.97-99

Craig, J. and Baron-Cohen, S. (1999) 'Creativity and imagination in autism and Asperger syndrome'. *Journal of Autism and Developmental Disorders*, 29:4, pp.319-326

Crespi, B., Leach, E., Dinsdale, N., Mokkonen, M. and Hurd, P. (2016) 'Imagination in human social cognition, autism, and psychotic-affective conditions'. *Cognition*, 150, pp.181-199

Crespi, B. and Croft, H. (2012) 'Association testing of copy number variants in schizophrenia and autism spectrum disorders'. *Journal of Neurodevelopmental Disorders*, 4:15, doi:10.1186/1866-1955-4-15

Crespi, B. J. and Thiselton, D. L. (2011) 'Comparative immunogenetics of autism and schizophrenia'. *Genes, Brain and Behaviour*, 10, pp.689-701

Crespi, B. J. (2010) 'Revisiting Bleuler: relationship between autism and schizophrenia'. *The British Journal of Psychiatry*, 196, pp.495-497

Crespi, B. and Badcock, C. (2008) 'Psychosis and autism as diametrical disorders of the social brain'. *Brain and Behavioural Sciences*, 31:1, pp.241-261

Crivello, F., Tzourio-Mazoyer, N., Tzaurio, C. and Mazoyer, B. (2014) 'Longitudinal assessment of global and regional rate of grey matter atrophy in 1172 healthy older adults: modulation by sex and age'. *PLoS One*, 9:12, doi:10.1371/journal.pone.0114478

D'Argembeau, A. and Van Der Linden, M. (2005) 'Influence of emotion on memory for temporal information'. *Emotion*, 5:4, pp.503-507

D'Onofrio, B. M., Rickert, M. E., Frans, E., Kuja-Halkola, R., Almqvist, C., Sjölander, A., Larsson, H. and Lichtenstein, P. (2014) 'Paternal age at childbearing and offsring psychiatric and academic morbidity'. *JAMA Psychiatry*, 71:4 doi:10.1001/jamapsychiatry.2013.4525

Dagnall, N. and Parker, A. (2009) 'Schizotypy and false memory'. *Journal of Behavioural Therapy and Experimental Psychiatry*, 40, pp.179-188

Daly, M. P., Afroz, S. and Walder, D.J. (2012) 'Schizotypal traits and neurocognitive functioning among nonclinical young adults'. *Psychiatry Research*, 200:2-3, pp.635-640

Daniels, J. L., Forssen, U., Hultman, C. M., Cnattingius, S., Savitz, D. A., Feychting, M. and Sparen, P. (2008) 'Parental psychiatric disorders associated with autism spectrum disorders in the offspring'. *Pediatrics,* 121, pp.1357–1362

Danion, J-M., Huron, C., Vidailhet, P. and Berna, F. (2007) 'Functional mechanisms of episodic memory impairment in schizophrenia'. *The Canadian Journal of Psychiatry*, 52:11, pp.693-701

Dapretto, M., Davies, M. S., Pfeifer, J. H., Scott, A. A., Sigman, M., Bookheimer, S. Y. and Iacoboni, M. (2006) 'Understanding emotions in others: mirror neuron dysfunction in children with autism spectrum disorders'. *Nature*, 9, pp.28–30

Davis, T. E., Moree, B. N., Dempsey, T., Reuther, E. T., Fodstad, J. C., Hess, J. A., Jenkins, W. S. and Matson, J. L. (2010) 'The relationship between autism spectrum disorders and anxiety: the moderating effect of communication'. *Research in Autism Spectrum Disorders*, 5, pp.324-329

Del Giudice, M., Angeleri, R., Brizio, A. and Elena, M. R. (2010) 'The evolution of autistic-like and schizotypal traits: a sexual selection hypothesis'. *Frontiers in Psychology*, 1:41, pp.01-41

Dewhurst, S. A., Anderson, R. J. and Knott, L. M. (2012) 'A gender difference in the false recall of negative words: women DRM more than men'. *Cognition & Emotion*, 26:1, pp.65-74

Di Ceglie, D., Skagerberg, E., Baron-Cohen, S. and Auyeung, B. (2014) 'Empathising and systemising in adolescents with gender dysphoria', *Opticon*, 16:6, pp.01-08

Dinsdale, N., Mökkönen, M. I. and Crespi, B. (2016) 'The 'extreme female brain': increased cognitive empathy as a dimension of psychopathology'. *Evolution and Human Behavior*, doi:10.1016/j.evolhumbehav.2016.02.003

Dinsdale, N. L., Hurd, P. L., Wakabayashi, A., Elliot, M. and Crespi, B. J. (2013) 'How are autism and schizotypy related? Evidence from a non-clinical population'. *PLoS ONE*, 8:5, e63316. doi:10.1371/journal.pone.0063316

Doerksen, S. and Shimamura, A. P. (2001) 'Source memory enhancement for emotional words'. *Emotion*, 1:1, pp.5-11

Doré, M. C., Caza, N., Gingras, N. and Rouleau, N. (2007) 'Deficient relational binding processes in adolescents with psychosis: evidence from impaired memory for source and temporal context'. *Cognitive Neuropsychiatry*, 12:6, pp.511–36

Driscoll, I., Davatzikos, C., An, Y., Wu, X., Shen, D., Kraut, M. and Resnick, S. M. (2009) 'Longitudinal pattern of regional brain volume change differentiates normal aging from MCI'. *Neurology*, 72:22, pp.1906-1913

Driscoll, I., Hamilton, D. A., Petropoulos, H., Yeo, R. A., Brooks, W. M., Baumgartner, R. N. and Sutherland, R. J. (2003) 'The aging hippocampus: cognitive, biochemical and structural findings'. *Cerebral Cortex*, 12, pp.1344-1351

Dudley, R. E. J., John, C. H., Young, A. W. and Over, D. E. (1997) 'Normal and abnormal reasoning in people with delusions'. *British Journal of Clinical Psychology*, 36, pp.243-258

Dykens, E. M., Lee, E., Roof, E. (2011) 'Prader-Willi syndrome and autistic spectrum disorders an evolving story'. *Journal Neurodevelopmental Disorders,* 3, pp.225-237

Dykens, E., Volkmar, F. and Glick, M. (1991) 'Thought disorder in high-functioning autistic adults'. *Journal of Autism and Developmental Disorders*, 21:3, pp.291-301

Eagly, A. H., Eaton, A., Rose, S. M., Riger, S. and McHugh, M. C. (2012) 'Feminism and psychology: analysis of a half-century of research on women and gender'. *The American Psychologist*, 67:3, pp.211-230

Eaton, N. R., Keyes, K. M., Krueger, R. F., Balsis, S., Skodal, A. E., Markon, K. E., Grant, B. F. and Hasin, D. S. (2012) 'An invariant dimensional liability model of gender differences in mental disorder prevalence: evidence from a national sample'. *Journal of Abnormal Psychology*, 121:1, pp.282-288

Ek, M., Wicks, S., Svensson, A. C., Idring, S. and Dalman, C. (2014) 'Advancing paternal age and schizophrenia: the impact of delayed fatherhood'. *Schizophrenia Bulletin*, 41, pp.708-714

Eliot, L. (2011) 'The trouble with sex differences'. *Neuron*, 72:6, pp.895-898

Ellett, L., Allen-Crooks, R., Stevens, A., Wildschut, T. and Chadwick, P. (2013) 'A paradigm for the study of paranoia in the general population: the prisoner's dilemma game'. *Cognition and Emotion*, 27:1, pp.53-62

Ellett, L. and Wildschut, T. (2012) 'Are we all paranoid?'. *The Psychologist*, 27:5, pp.328-330

Ellett, L., Lopes, B. and Chadwick, P. (2003) 'Paranoia in a non-clinical population of college students'. *Journal of Nervous and Mental Disease*, 191, pp.425-430

Ellis, H. D. (2005) 'Book review: Simon Baron Cohen: The Essential Difference'. *Cognitive Neuropsychiatry,* 10:1, pp.73-75

Elvevåg, J. E., Fisher, J. E., Weickert, T. W., Weinberger, D. R. and Goldberg, T. E. (2004) 'Lack of false recognition in schizophrenia: a consequence of poor memory?'. *Neuropsychology*, 42:4, pp.546-554

Enticott, P. G., Hoy, K. E., Herring, S. E., Johnston, P. J., Daskalakis, Z. J. and Fitzgerald, P. B. (2008) 'Reduced motor facilitation during action

observation in schizophrenia: a mirror neuron deficit?'. *Schizophrenia Research*, 102, pp.116–121

Etkin, A., Gyurak, A. and O'Hara, R. (2013) 'A neurobiological approach to the cognitive deficits of psychiatric disorders'. *Dialogues in Clinical Neuroscience*, 15:4, pp.419-429

Ettinger, U., Williams, S. C., Meisenzahl, E. M., Moller, H. J., Kumar, V. M. and Koutsouleris, N. (2011) 'Association between brain structure and psychometric schizotypy in healthy individuals'. *World Journal of Biological Psychiatry*, 13:7, pp.544-549

Eussen, M. L. J., de Bruin, E. I., Van Gool, A. R., Louwerse, A., van der Ende, J., Verheij, F., Verhulst, F. C. and Greaves-Lord, K. (2014) 'Formal thought disorder in autism spectrum disorder predicts future symptom severity, but not psychosis prodrome'. *European Journal of Child and Adolescent Psychiatry*, doi:10.1007/s00787-014-0552-9

Eysenck, H. J. (1995) Creativity as a product of intelligence and personality. *In:* D. H. Saklofske and M. Zeidner, eds. *International handbook of personality and intelligence*. New York: Plenum Press, pp. 231–247

Falter, C. M., Plaisted, K. C. and Davis, G. (2008) 'Visuo-spatial processing in autism – Testing the predictions of extreme male brain theory'. *Journal of Autism and Developmental Disorders*, 38, pp.507–515

Farrant, A., Blades, M. and Boucher, J. (1998) 'Source monitoring by children with autism'. *Journal of Autism and Developmental Disorder*, 28:1, pp.43-50

Faul, F., Erdfelder, E., Bucher, A. and Lang, A. G. (2009) 'Statistical power analysis using G*Power 3.1: tests for correlation and regression analysis'. *Behavior Research Methods*, 41:4, pp.1149-1160

Fenigstein, A. and Vanable, P. (1992) 'Persecutory ideation and self-consciousness'. *Journal of Personality and Social Psychology*, 62, pp.129-138

Fett, A. K., Viechtbauer, W., Dominguez, M. D., Penn, D. L., van Os, J. and Krabbendam, L. (2010) 'The relationship between neurocognition and social

cognition with functional outcomes in schizophrenia: a meta-analysis'. *Neuroscince and Biobehavioural* Reviews, 35:3, pp.573-588

Fine, C., Jordan-Young, R., Kaiser, A. and Rippon, G. (2013) 'Plasticity, plasticity, plasticity...and the rigid problem of sex'. *Trends in Cognitive Sciences,* 17:11, pp.550-551

Fine. C. (2012) 'Is there neurosexism in functional neuroimaging investigations of sex differences? *Neuroethics,* doi: 10.1007/s12152-012-9169-1

Fine, C. (2010a) 'From scanner to sound bite: issues in interpreting and reporting sex differences in the brain'. *Current Directions in Psychological Science,* 19:5, pp.280-283

Fine, C. (2010b) *The real science behind sex differences: the delusions of gender*, Icon Books: London

Fine, C. (2008) 'Will working mother's brain's explode? The popular new genre of neurosexism'. *Neuroethics,* 1, pp.69-72

Fink, A., Weber, B., Koschutnig, K., Benedek, M., Reishofer, G., Ebner, F., Papousek, I. and Weiss, E. M. (2013) 'Creativity and schizotypy from the neuroscience perspective'. *Cognitive, Affective and Behavioural Neuroscience*, 14:1, pp.378-387

Fink, B., Manning, J. T. and Neave, N. (2006) 'The 2nd- 4th finger digit ratio (2D:4D) and neck circumference: implications for risk factors in coronary heart disease'. *International Journal of Obesity*, 30, pp.711-714

Fitzgerald, M. (2011) Creativity psychosis autism and the social brain. *In*: Mohammadi, M. R., ed. A comprehensive book on autism spectrum disorders. InTech. pp.214-224. [online] Available at: <http://www.intechopen.com/books/a-comprehensive-book-on-autism-spectrum-disorders/creativity-psychosis-autism-and-the-social-brain> [Accessed 16 March 2016]

Focquaert, F., Steven, M. S., Wolford, G. L., Colden, A. and Gazzaniga, M. S. (2007) 'Empathising and systemising cognitive traits in the sciences and humanities'. *Personality & Individual Differences,* 43, pp.619-625

Fombonne, E. (2007) Epidemiological surveys of pervasive developmental disorders. *In:* Volkmar, F. R., ed. *Autism and pervasive developmental disorders.* New York: Cambridge University Press, pp.33-68

Fonseca-Pedrero, E., Paino, M., Lemos-Giraldez, S., Garcia-Cuero, E., Campillo-Alvarez, A., Villazon-Garcia, U. and Muniz, J. (2008) 'Schizotypy assessment: state of the art and future prospects'. *International Journal of Clinical and Health Psychology*, 8:2, pp.577-593

Freeman, D. (2007) 'Suspicious minds: the psychology of persecutory delusions'. *Clinical Psychology Review*, 27:4, pp.425-457

Freeman, D., McManus, S., Brugha, T., Meltzer, H., Jenkins, R. and Bebbington, P. (2011) 'Concomitants of paranoia in the general population'. *Psychological Medicine*, 41, pp.923-936

Freeman, D., Pugh, K., Vorontsova, N. and Antley, A. (2010) 'Testing the continuum of delusional beliefs: an experimental study using virtual reality'. *Journal of Abnormal Psychology*, 119:1, pp.83-92

Freeman, D., Pugh, K., Antley, A., Slater, M., Bebbington, P., Gittins, M., Dunn, G., Kuipers, E., Fowler, D. and Garety, P. (2008) 'Virtual reality study of paranoid thinking in the general population'. *The British Journal of Psychiatry*, 192, pp.258-263

Freeman, D., Pugh, K. and Garety, P. (2008) 'Jumping to conclusions and paranoid ideation in the general population'. *Schizophrenia Research*, 102:1-3, pp.254-260

Freeman, D., Garety, P. A., Bebbington, P. E., Smith, B., Rollinson, R., Fowler, D., Kuipers, E., Ray, K. and Dunn, G. (2005) 'Psychological investigation of the structure of paranoia in a non-clinical population'. *British Journal of Psychiatry*, 186, pp.427-435

Freeman, D., Garety, P. A., Kuiper, E., Fowler, D. and Bebbington, P. E. (2002) 'A cognitive model of persecutory delusions'. *British Journal of Clinical Psychology*, 41, pp.331-347

Freeman, D. and Garety, P. A. (2000) 'Comments on the contents of persecutory delusions: does the definition need clarification?'. *British Journal of Clinical Psychology*, 39, pp.302-308

Fridberg, D. J., Vollmer, J. M., O'Donnell, B. F. and Skosnik, P. D. (2011) 'Cannabis users differ from non-users on measures of personality and schizotypy'. *Psychiatry Research*, 186, pp.46-52

Frith, C. D. (2004) 'Schizophrenia and theory of mind'. *Psychological medicine*, 34, pp.385–389

Frith, U. (1989) *Autism: explaining the enigma*. U.K: Wiley Blackwell

Frith, U. and Frith, C. (2010) 'The Social Brain: allowing humans to boldly go where no other species has been'. *Philosophical Transactions of The Royal Society,* 365, pp.165-176

Fusar-Poli, P., Placentino, A., Carletti, F., Landi, P., Allen, P., Surguladze, S., Benedetti, F., Abbamonte, M., Gasparotti, R., Barale, F., Perez, J., McGuire, P. and Politi, P. (2009) 'Functional atlas of emotional faces processing: a voxel-based meta-analysis of 105 functional magnetic resonance imaging studies'. *Journal of Psychiatry and Neuroscience*, 34:6, pp.418-432

Gadow, K. D. (2013) 'Association of schizophrenia spectrum and autism spectrum disorder (ASD) symptoms in children with ASD and clinic controls'. *Research in Developmental Disabilities*, 34, pp.1289-1299

Galderisi, S., Bucci, P., Ücok, A. and Peuskens, J. (2012) 'No gender differneces in social outcome in patients suffering from schizophrenia'. *European Psychiatry: the Journal of the Association of European psychiatrists*, 27:6, pp.406-408

Gallese, V., Fadiga, L., Fogassi, L. and Rizzolatti, G. (1996) 'Action recognition in the premotor cortex'. *Brain*, 119, pp.593-609

Gallup, G. G. and Bremser, J. A. (2012) 'From one extreme to the other: negative evaluation anxiety and disordered eating as candidates for the extreme female brain'. *Evolutionary Psychology*, 10:3, pp.457-486

Garety, P. A., Freeman, D., Jolley, S., Dunn, G., Bebbington, P. E., Fowler, D. G., Kuipers, E. and Dudley, R. (2005) 'Reasoning, emotions and delusional conviction in psychosis'. *Journal of Abnormal Psychology*, 114:3, pp. 373-384

Garfinkel, P. E., Lin, E., Goering, P., Spegg, C., Goldbloom, D. S., Kennedy, S., Kaplan, A. S. and Woodside, D. B. (1995) 'Bulimia nervosa in a Canadian community sample: prevalence and comparison of subgroups'. *The American Journal of Psychiatry*, 152:7, pp.1052-1058

Garner, D. M., Olmsted, M. P., Bohr, Y. and Garfinkel, P. E. (1982) 'The eating attitudes test: psychometric features and clinical correlates'. *Psychological Medicine*, 12, pp.871-878

Geary, D. (1996) 'Sexual selection and sex differences in mathematical abilities'. *Behavioral Brain Sciences*, 19, pp.229–284

Geschwind, D. H. (2011) 'Genetics of autistic spectrum disorders'. *Trends in Cognitive Sciences,* 15:9, pp.409-417

Ghaziuddin, M., Weidmer, M. and Ghaziuddin, N. (1998) 'Comorbidity of Asperger syndrome: a preliminary report'. *Journal of Intellectual Disability*, 42, pp.279-283

Ghaziuddin, M., Leininger, L. and Tsai, L. (1995) 'Brief report: thought disorder in Asperger syndrome: comparison with high-functioning autism'. *Journal of Autism and Developmental Disorders*, 25:3, pp.311-317

Giedd, J. N., Vaituzis, A. C., Hamburger, S. D., Lange, N., Rajapakse, J. C. and Rapoport, J. L. (1996) 'Quantitative MRI of the temporal lobe, amygdala, and the hippocampus in normal human development: ages 4-18 years'. *The Journal of Comparative Neurology*, 366:2, pp.223-230

Giedd, J. N., Castellanos, F. X., Rajapakse, J. C., Vaituzis, A. C. and Rapoport, J. L. (1997) 'Sexual dimorphism of the developing human brain'.

Progress in Neuro-Psychopharmacology and Biological Psychiatry, 21:8, pp.1185-1201

Gillies, G. E. and McArthur, S. (2010) 'Estrogen actions in the brain and the basis for differential action in men and women: a case for sex-specific medicines'. *Pharmalogical Review*, 62:2, pp.155-198

Gilsky, E, L. (2007) *Cognitive function in human aging*. Boca Raton: Taylor and Francis

Golan, O., Baron-Cohen, S. and Hill, J. (2006a) 'The Cambridge mindreading (CAM) face-voice battery: testing complex emotion recognition in adults with and without Asperger syndrome' *Journal of autism and other developmental disorders*, 36:2, pp.169-184

Golan, O., Baron-Cohen, S., Hill, J. and Golan, Y. (2006b) 'The reading the mind in films task: complex emotion recognition in adults with and without autism spectrum disorders' *Social Neuroscience*, 1:2, pp.111-123

Goldenfeld, N., Baron-Cohen, S., and Wheelwright, S. (2005) 'Empathizing and systemizing in males, females and autism'. *Clinical Neuropsychiatry*, 2:1, pp.1–8

Goldstein, J. M., Seidman, L. J., O'Brien, L. M., Horton, N. J., Kennedy, D. N., Makris, N., Caviness, V. S., Faraone, S. V. and Tsuang, M. T. (2002) 'Impact of Normal Sexual Dimorphisms on sex differences in structural brain abnormalities in schizophrenia assessed by magnetic resonance imaging'. *Archives of Genetic Psychiatry,* 59, pp.154-164

Goldstein, J. M., Seidman, L. J., Goodman, J. M., Koren, D., Lee, H., Weintraub, S. and Tsuang, M.T. (1998) 'Are there sex differences in neuropsychological functions among patients with schizophrenia'. *American Journal of Psychiatry,* 155, pp.1358-1364

Good, C. D., Johnsrude, I. S., Ashburner, J., Henson, R. N., Friston, K. J. and Frackowiak, R. S. (2001) 'A voxel-based morphometric study of ageing in 465 normal adult human brains'. *NeuroImage*, 14, pp.21–36

Gooding, D. C. and Pflum, M. J. (2011) 'Theory of mind and psychometric schizotypy'. *Psychiatry Research*, 188, pp.217-223

Gooding, D. C., Johnson, M. and Peterman, J. S. (2010) 'Schizotypy and altered digit rations: a second look'. *Psychiatry Research,* 178, pp.73-78

Gong, G., Rosa-Neto, P., Carbonell, F., Chen, Z. J., He, Y. and Evans, A. C. (2009) 'Age and gender related differences in the cortical anatomical network'. *The Journal of Neuroscience*, 29:50, pp.15684-15693

Gonzalez-Ortega, L., de los Mozos, V., Echeburúa, E., Mezo, M., Besga, A., Ruiz de Azúa, S., Gonzàlez-Pinto, A., Gutierrez, M., Zorrilla, L. and Gonzalez-Pinto, A. (2013) 'Working memory as a predictor of negative symptoms and functional outcome in forst episode psychosis'. *Psychiatry Research*, 206, pp.8-16

Grady, C. L., Maisog, J. M., Horwitz, B., Ungerleider, L. G., Mentis, M. J., Salerno, J. A., Pietrini, P., Wagner, E. and Haxby, J. V. (1994) 'Age-related changes in cortical blood flow activation during visual processing of faces and location'. *The Journal of Neuroscience*, 14, pp.1450-1462

Green, C. E., Freeman, D., Kuipers, E., Bebbington, P., Fowler, D., Dunn, G. and Garety, P. A. (2008) 'Measuring ideas of persecution and social reference: the Green et al. paranoid thought scales (GPTS)'. *Psychological medicine*, 38:1, pp.101–11

Grether, J. K., Anderson, M. C., Croen, L. A., Smith, D. and Windham, G. C. (2009) 'Risk of autism and increasing maternal and paternal age in a large north American population'. *American Journal of Epidemiology*, 170, pp.1118-1126

Groen, Y., Fuermaier, A. B. M., Den Heijer, A. E., Tucha, O. and Althaus, M. (2015) 'The empathising and systemising quotient: the psychometric properties of the Dutch version and a review of the cross-cultural stability'. *Journal of Autism and Developmental Disorders*, 45, pp.2848-2864

Grove, R., Bailie, A., Allison, C., Baron-Cohen, S., Hoekstra, R. A. (2013) 'Empathizing, systemizing, and autistic traits: latent structure in individuals

with autism, their parent, and general population controls'. *Journal of Abnormal Psychology*, 122:2, pp.600-609

Guara, R. C., Alsopb, D., Glahna, D., Pettya, R., Swansona, C. L., Maldjianb, J. A., Turetskya, B. I., Detrec, J. A., Geea, J. and Gura, R. E. (2000) 'An fMRI Study of Sex Differences in Regional Activation to a Verbal and a Spatial Task'. *Brain and Language*, 74:2, pp.157-170

Gur, R. C., Gunning-Dixon, F., Bilker, W. B. and Gur, R. E. (2002) 'Sex differences in temporo-limbic and frontal brain volumes of healthy adults'. *Cerebral Cortex*, 12, pp.998–1003

Gur, R. C., Turetsky, B. I., Matsui, M., Yan, M., Bilker, W., Hughett, P. and Gur, R. E. (1999) 'Sex differences in brain gray and white matter in healthy young adults: correlations with cognitive performance'. *Journal of Neuroscience*, 19, pp.4065–4072

Hall, J. A. (1978) 'Gender effects in decoding nonverbal cues'. *Psychological Bulletin,* 85, pp.845–858

Halpern, D. F. (2012) *Sex differences in cognitive abilities* (4thed). UK: Psychology Press

Halpern, D. F., Benbow, C. P., Geary, D. C., Gur, R. C., Hyde, J. S. and Gernsbacher, M. A. (2007) 'The science of sex differences in science and mathematics'. *Psychological Science in the Public Interest,* 8:1, pp.01-53

Hambrecht, M., Riecher-Rössler, A., Fätkenheuer, B., Louzã, M. R. and Häfner, H. (1994) 'Higher morbidity risk for schizophrenia in males: fact or fiction?' *Comprehensive Psychiatry*, 35:1, pp.39-49

Hambrook, D., Schmidt, U., Russell, T., Treasure, J. and Tchanturia, K. (2008) 'Empathy, systemizing, and autistic traits in anorexia nervosa: a pilot study'. *British Journal of Clinical Psychology*, 47, pp.335-339

Hamilton, A., Brindley, R. and Frith, U. (2007) 'Imitation and action understanding in autistic spectrum disorder: How valid is the hypothesis of a deficit in the mirror neuron system?' *Neuropsychologia,* 45, pp.1859-1868

Hamilton, C. (2008) *Cognition and sex differences*. Basingstoke: Palgrave Macmillan

Hampson, E. (1990) 'Variations in sex-related cognitive abilities across the menstrual cycle'. *Brain and Cognition*, 14, pp.26-43

Happé, F. and Frith, U. (2006) 'The weak central coherence account: detail-focused cognitive style in autism spectrum disorders'. *Journal of Autism and Developmental Disorders*, 36, pp.5-25

Happé, F. G. E., Winner, E. and Brownell, H. V. (1998) 'The getting of wisdom: theory of mind in old age'. *Developmental Psychology*, 34, pp.358-362

Harasty, J., Double, K. L., Halliday, G. M., Kril, J. J. and McRitchie, D. A. (1997) 'Language-associated cortical regions are proportionally larger in the female brain'. *Archives of Neurology*, 54:2, pp.171-176

Harrison, A., Sullivan, S., Tchanturia, K. and Treasure, J. (2010a) 'Emotional functioning in eating disorders: attentional bias, emotion recognition and emotion regulation'. *Psychological Medicine*, 40:11, pp.1887-1897

Harrison, A., Tchanturia, K. and Treasure, J. (2010b) 'Attentional bias, emotion recognition, and emotion regulation in anorexia: state or trait?'. *Biological Psychiatry*, 68:8, pp.755-761

Hassett, J.M., Siebert, E. R. and Wallen, K. (2008) 'Sex differences in rhesus monkey toy preferences parallel those of children'. *Hormones and Behavior*, 54:3, pp.359-364

Henry, J. D., Bailey, P. E. and Rendell, P. G. (2008) 'Empathy, social functioning and schizotypy'. *Psychiatry Research*, 160, pp.15-22

Herlitz, A. and Rehnman, J. (2008) 'Sex differences in episodic memory'. *Current Directions in Psychological Sciences*, 17:1, pp.52-56

Herlitz, A. and Yonker, J. E. (2002) 'Sex differences in episodic memory: the influence of intelligence'. *Journal of Clinical and Experimental Neuropsychology*, 24, pp.107–114

Herlitz, A., Airaksinen, E. and Nordström, E. (1999) 'Sex differences in episodic memory: the impact of verbal and visuospatial ability'. *Neuropsychology*, 13:4, pp.590-597

Herlitz, A., Nilsson, L-G. and Bäckman, L. (1997) 'Gender differences in episodic memory' [Abstract]. *Memory & Cognition*, 25:6, pp.801-811

Hillier, A., Campbell, H., Keillor, J., Phillips, N. and Beversdorf, D. Q. (2007) 'Decreased false memory for visually presented shapes and symbols among adults on the autism spectrum'. *Journal of Clinical and Experimental Neuropsychology*, 29:6, pp.610-616

Hines, M. (2010) 'Sex- related variation in human behaviour and the brain'. *Trends in Cognitive Science,* 14:10, pp.448-457

Hodgetts, S., Hausmann, M. and Weis, S. (2015) 'High estradiol levels improves false memory rates and meta-memory in highly schizotypal women'. *Psychiatry Research*, 229:3, pp.708-714

Honekopp, J. (2012) 'Digit ratio 2D:4D in relation to autism spectrum disorders, empathising and systemising: a quantitative review'. *Autsim Research,* 5(4), pp.221-230

Hooker, C. I., Verosky, S. C., Germine, L. T., Knight, R. T. and D'Esposito, M. (2010) 'Neural activity during social signal perception correlates with self-reported empathy'. *Brain Research,* 1308, pp.100–113

Horan, W. P., Blanchard, J. J., Clark, L. A. and Green, M. F. (2008) 'Affective traits in schizophrenia and schizotypy'. *Schizophrenia Bulletin*, 34, pp.856–874

Hudson, J. I., Hiripi, E., Pope, H. G. and Kessler, R. C. (2007) 'The prevalence and correlates of eating disorders in the national comorbidity survey replication'. *Biological Psychiatry*, 61:3, pp.348-358

Hugdhl, K., Thomsen, T. and Ersland, L. (2006) 'Sex differences in visuo-spatial processing: an fMRI study of mental rotation'. *Neuropsychologia,* 44:9, pp.1575-1583

Hultman, C. M., Sandin, S., Levine, S. Z., Lichtenstein, P. and Reichenberg, A. (2011) 'Advancing paternal age and risk of autism: new evidence from a population-based study and a meta-analysis of epidemiological studies'. *Molecular Psychiatry*, 16, pp.1203-1212

Huq, S. F., Garety, P. and Hemsley, D. R. (1988) 'Probabilistic judgements in deluded and non-deluded subjects'. *The Quarterly Journal of Experimental Psychology Section A*, 40:4, pp.801-812

Hurst, R., Nelson-Gray, R. O., Mitchell, J. and Kwapil, T. R. (2006). 'The relationship of Asperger's characteristics and schizotypal personality traits in a non-clinical adult sample'. *Journal of Autism and Developmental Disorders*, 37, pp.1711-1720

Hyde, J. S. (2007) 'New directions in the study of gender similarities and differences'. *Current Directions in Psychological Science*, 16:5, pp.259-263

Iacobona, M. and Dapretto, M. (2006) 'The mirror neuron system and the consequences of its dysfunction'. *Nature,* 7, pp.942-953

Ingalhalikar, M., Smith, A., Parker, D., Satterwaite, T. D., Elliott, M. A., Ruparel, K., Hakonarson, H., Gur, R. E., Gur, R. C. and Verma, R. (2013) 'Sex differences in the structural connectome of the human brain'. *PNAS*, doi:10.1073/pnas.1316909110

Isnanda, R. G., Brinkman, W. P., Veling, W., van der Gaag, M. and Neerinex, M. A. (2013) 'Priming to induce paranoid thought in a non-clinical population'. *Studies in Health Technology and Informatics*, 191, pp.95-99

Jack, A. I., Dawson, A. J., Begany, K. L., Leckie, R. L., Barry, K. P., Ciccia, A. H. and Synder, A. Z. (2013) 'fMRI reveal reciprocal inhibition between social and physical cognitive domains'. *NeuroImage*, 66, pp.385-401

Jänsch, C. and Hare, D. J. (2014) 'An investigation of the 'jumping to conclusions' data-gathering bias and paranoid thoughts in Asperger syndrome'. *Journal of Autism and Developmental Disorders*, 44:1, pp.111-119

Jänsch, C., Harmer, C. and Cooper, M. J. (2009) 'Emotional processing in women with anorexia nervosa and in healthy volunteers'. *Eating Behaviours*, 10:3, pp.184-191

Jarrod, C., Butler, D. W., Cottington, E. M. and Jimenez, F. (2000) 'Linking theory of mind and central coherence bias in autism and the general population' *Developmental Psychology*, 36:1, pp.126-138

Jeannerod, M. and Pacherie, E. (2004) 'Agency simulation and self-identification'. *Mind and Language*, 19:2, pp.113-146

Joel, D (2012) 'Genetic-gonadal-genitals sex (3G-sex) and the misconception of brain and gender, or , why 3G-males and 3G-females have intersex brain and intersex gender'. *Biology of Sex Differences*, 17:3, doi:10.1186/2042-6410-3-27

Joel, D., Berman, Z., Tavor, I., Wexler, N., Gaber, O., Stein, Y., Shefi, N., Pool, J., Urchs, S., Margulies, D. S., Liem, F., Hänggi, J., Jäncke, L. and Assaf, Y. (2015) 'Sex beyond the genitalia: the human brain mosaic'. *PNAS*, 112:50, pp.15468-15473

Johnson, W. and Bouchard, T. J. (2007) 'Sex differences in mental abilities: g masks the dimensions on which they lie'. *Intelligence*, 35, pp.23-39

Jollife, T. and Baron-Cohen, S. (1997) 'Are people with Autism and Asperger's Syndrome faster than normal on the embedded figures test? *Journal of Clinical Psychology and Psychiatry*, 38:5, pp.527-534

Jones, C. M., Braithwaite, V. A. and Healy, S. D. (2003) 'The evolution of sex differences in spatial ability'. *Behavioural Neuroscience*, 117, pp.403–411

Jones, L., Harmer, C., Cowen, P. and Cooper, M. (2009) 'Emotional face processing in women with high and low levels of eating disorder related symptoms', *Eating Behaviour*, 9, pp.389-397

Jones, S. L. and Lesk, V. (2013) 'Exploring the relationship between the extreme female brain and schizophrenic spectrum disorders' [Presented at the British Neuropsychological Society Spring Conference, March 2013, London, by S. L. Jones]

Jordan-Young, R. and Rumiati, R. I. (2011) 'Hardwired for sexism? Approaches to sex/gender in neuroscience'. Neuroethics, 5, doi:10.1007s12152-011-9134-4

Kalbe, E., Schegel, M., Sack, A. T., Nowark, D. A., Dafotakis, M., Bangard, C., Brand, M., Shamay-Tsoory, S., Onur, O. A. and Kessler, J. (2010) 'Dissociating cognitive from affective theory of mind: a TMS study'. *Cortex*, 46:6, pp.769-780

Kanazawa, S. and Vandermassen, G. (2005) 'Engineers have more sons, nurses have more daughters: an evolutionary psychological extension of Baron- Cohen's extreme male brain theory of autism'. *Journal of Theoretical Biology,* 233, pp.589-599

Keefe, R. S. E., Arnold, M. C., Bayen, U. J., McEvoy, J. P. and Wilson, W. H. (2002) 'Source-monitoring deficits for self-generated stimuli in schizophrenia: multinomial modelling of data from these sources'. *Schizophrenia Research*, 57:1, pp. 51-67

Keel, P. K., Klump, K. L., Miller, K. B., McGue, M. and Iacono, W. G. (2005) 'Shared transmission of eating disorders and anxiety disorders'. *International Journal of Eating Disorders*, 38:2, pp.99-105

Keller, R., Piedimonte, A., Bianco, F., Bari, S. and Cauda, F. (2016) 'Diagnostic characteristics of psychosis and autism spectrum disorder in adolescence and adulthood. A case series'. *Autism Open Access*, 6:159. doi:10.4172/2165-7890.1000159

Kerns, J. G., Nuechterlein, K. H., Braver, T. S. and Barch, D. M. (2008) 'Executive functioning component mechanisms and schizophrenia'. *Biological Psychiatry*, 64:1, pp. 26-33

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R. and Walters, E. E. (2005) 'Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication'. *Archives of General Psychiatry*, 62, pp.593-602

Keverne, E. B., Fundere, R., Narasimha, M., Barton, S. C. and Surani, M. A. (1996) 'Genomic imprinting and the differential roles of parental genomes in brain development'. *Developmental Brain Research*, 92:1, pp.91-100

Kim, J., Szatmari, P., Bryson, S., Streiner, D. and Wislon, F. (2000) 'The prevalence of anxiety and mood problems among children with autism and Asperger syndrome'. *Autism*, 4, pp.117-132

Kim, S. H., Shin, N. Y., Jang, J. H., Kim, E., Shim, G., Park, H. Y., Hong, K. S. and Kwon, J. S. (2011) 'Social cognition and neurocognition as predictors of conversion to psychosis in individuals at ultra-high risk'. *Schizophrenia Research,* 130, pp.170-175

Kimura, D. (1996) 'Sex, sexual orientation and sex hormones influence human cognitive function'. *Current Opinion in Neurobiology*, 6:2, pp.259-263pelp

King, B. H. and Lord, C. (2011) 'Is schizophrenia on the autism spectrum?'. *Brain Research,* 1380, pp.34-41

Kirkpatrick, B., Fenton, W. S., Carpenter, W. T. and Marder, S. R. (2006) 'The NIMH-MATRICS consensus statement on negative symptoms'. *Schizophrenia Bulletin*, 32:2, pp.214-219

Klin, A. (2009) 'Embracing the challenge of bold theories of autism'. *British Journal of Psychology*, 100, pp.29-32

Konstantaeas, M and Hewitt, T. (2001) 'Autistic disorder and schizophrenia: diagnostic overlaps'. *Journal of Autism and Developmental Disorders*, 31, pp.19-28

Kreiner, D. S., Price, R. Z., Gross, A. M. and Appleby, K. L. (2004) 'False recall does not increase when words are presented in a gender-congruent voice'. *Journal of Articles in support of the Null Hypothesis*, 3, pp.1-18

Kucharska-Pietura, K., Nikolaou, V., Masiak, M. and Treasure, J. (2004) 'The recognition of emotion in the faces and voice of anorexia nervosa'. *International Journal of Eating Disorders*, 35:1, pp.42-47

Lai, M-C., Lombardo, M. V., Auyeung, B., Chakrabarti, B. and Baron-Cohen, S. (2015) 'Sex/gender differences and autism: setting the scene for future research'. *Journal of the American Academy of Child and Adolescent Psychiatry*, 54:1, pp.11-24

Lai, M., Lombardo, M., Ruigrok, A., Chakrabarti, B., Wheelwright, S., Auyeung, B., Alison, C. and Baron-Cohen, S. (2012) 'Cognition in males, females with autism: similarities and differences' *PLoS One*, 7:1, doi:10.1371/journal.pone.0047198

Larson, F. V., Lai, M-C., Wagner, A. P., MRC AIMS Consortium, Baron-Cohen, S. and Holland, A. J. (2015) 'Testing the 'extreme female brain' theory of psychosis in adults with autism spectrum disorder with or without co-morbid psychosis'. *PLoS ONE* 10:6, e0128102. doi:10.137/journal. pone.0128102

Larsson, H. J., Eaton, W. W., Madsen, K. M., Vestergaard, M., Olesen, A. V., Agerbo, E., Schendel, D., Thorsen, P. & Mortensen, P. B. (2005) 'Risk factors for autism: perinatal factors, parental psychiatric history, and socioeconomic status'. *Am. J. Epidemiol*, 161, pp.916–925

Lawence, E. J., Shaw, P., Baker, D., Baron-Cohen, S. and David, A. S. (2004) 'Measuring empathy: reliability and validity of the empathy quotient'. *Psychological Medicine*, 34, pp.911-924

Laws, K. R. and Bhatt, R. (2005) 'False memories and delusional ideation in normal healthy subjects'. *Personality of Individual Differences*, 39:4, pp.775-781

Lawson, J., Baron-Cohen, S., and Wheelwright, S. (2004) 'Empathising and systemising in adults with and without Asperger syndrome'. *Journal of Autism and Developmental Disorders*, 34:4, pp.301–310

Leary, M. R. (1983) 'A brief version of the fear of negative evaluation scale'. *Personality and Social Psychology Bulletin*, 9, pp.371-376

Lee, T. M., Lui, H. L., Hoosain, R., Liao, W. T., Wu, C. T., Yuen, K.S., Chan, C. C., Fox, P.T. and Gao, J. H. (2002) 'Gender differences in neural correlates of recognition of happy and sad faces in humans assessed by

functional magnetic resonance imaging'. *Neuroscience Letters*, 333:1, pp.13-16

Lenzenweger, M. F. and Gold, J. M. (2000) 'Auditory working memory and verbal recall memory in schizotypy'. *Schizophrenia Research*, 42, pp.101-110

Leonard, C. M., Towler, S., Welcome, S., Halderman, L. K., Otto, R., Eckert, M. A. and Chiarello, C. (2008) 'Size matters: cerebral volume influences sex differences in neuroanatomy'. *Cerebral Cortex*,18:2, pp.2920 –2931

Leung, A. and Chue, P. (2000) 'Sex differences in Schizophrenia: a review of the literature'. *Acta Psychiatrica Scandinavica,* 101, pp.03-38

Levine, L. J. and Pizarro, D. A. (2004) 'Emotion and memory research: a grumpy overview'. *Social Cognition*, 22:5, pp.530-554

Lewin, C. and Herlitz, A. (2002) 'Sex differences in face recognition- women's faces make the difference'. *Brain and Cognition*, 50:1, pp.121-128

Lewin, C., Wolgers, G. and Herlitz, A. (2001) 'Sex differences favouring women in verbal but not visuospatial episodic memory' [Abstract]. *Neuropsychology*, 15:2, pp.165-173

Lincoln, T. M., Lange, J., Burau, J., Exner, C. and Moritz, S. (2010) 'The effect of state anxiety on paranoid ideation and jumping to conclusions. An experimental investigation'. *Schizophrenia Bulletin*, 36:6, pp.1140-1148

Lind, S. E. and Bowler, D. M. (2009) 'Recognition memory, self-other source memory, and theory-of-mind in children with autism spectrum disorder'. *Journal of Autism and Developmental Disorders*, 39:9, pp.1231–9.

Lindsberg, J., Poutiainen, E. and Kalska, H. (2009) 'Clarifying the diversity of first-episode psychosis: neuropsychological correlates of clinical symptoms'. *Nordic Journal of Psychiatry*, 63:6, pp.493-502

Ling, J., Burton, T.C., Salt, J.L. and Muncer, S.J. (2009) 'Psychometric analysis of the systemizing quotient (SQ) scale'. *British Journal of Psychology*, 100, pp.539-552

Linn, M. C. and Petersen, A. C. (1985) 'Emergence and characterisation of gender differences in spatial abilities: a meta-analysis'. *Child Development*, 56, pp.1479-1498

Lister, J. P. and Barnes, C. A. (2009) 'Neurobiological changes in the hippocampus during normative aging'. *Archives of Neurology*, 66:7, pp.829-833

Lo, J. C., Chong, P. L. H., Ganesan, S., Leong, R. L. F. and Chee, M. W. L. (2016) 'Sleep deprivation increases formation of false memory'. *Journal of Sleep Research*, 25:6, pp.673-682

Lopez, C. A., Tchanturia, K., Stahl, D. and Treasure, J. (2008) 'Central coherence in women with bulimia nervosa'. *International Journal of Eating Disorders*, 41:4, pp.340-347

Lowe, B., Decker, O., Muller, S., Brahler, E., Schellberg, D., Herzog, W. and Herzberg, P. Y. (2008) 'Validation and standardization of the generalized anxiety disorder screener (GAD-7) in the general population'. *Medical Care*, 46:3, pp.266-274

Luciano, C. C., Keller, R., Politi, P., Aguglia, E., Magnano, F., Burti, L., Muraro, F., Aresi, A., Damiani, S. and Berardi, D. (2014) 'Misdiagnosis of high function autism spectrum disorder: an Italian case series'. *Autism*, 4:2. doi:10.4172/2165-7890.1000131

Lutchmaya, S., Baron-Cohen, S., Raggatt, P., Knickmeyer, R. and Manning, J. T. (2004) '2nd to 4th digit ratios, fetal testosterone and estradiol'. *Early Human Development*, 77:1-2, pp.23-28

Lutchmaya, S., Baron-Cohen, S., and Raggatt, P. (2002) 'Foetal testosterone and eye contact in 12-month-old Infants'. *Infant Behaviour and Development*, 25, pp.327–335

Maddock, R. J., & Frein, S. T. (2009). 'Reduced memory for the spatial and temporal context of unpleasant words'. *Cognition & Emotion*, 23, 96–117

Maguire, E. A., Gadian, D. G., Johnsrude, I. S., Good, C. D., Ashburner, J., Frackowiak, R. S. J. and Frith, C. D. (2000) 'Navigation-related structural change in the hippocampi of taxi drivers'. *PNAS*, 97:8, pp.4398-4403

Malaspina, D., Harlap, S., Fennig, S., Heiman, D., Nahon, D., Feldman, D. and Susser, M. D. (2001) 'Advancing paternal age and the risk of schizophrenia'. *Archives of General psychiatry*, 58:4, pp.361-367

Maney, D. L. (2016) 'Perils and pitfalls of reporting sex differences'. *Philosophical Transactions of the Royal Society*. doi:10.1098/rstb.2015.0119.

Mann, V. A., Sasanuma, S., Sakuma, N. and Masaki, S. (1990) 'Sex differences in cognitive abilities: a cross cultural perspective'. *Neuropsychologia*, 28:10, pp.1063-1077

Manning, J. T., Reimers, S., Baron-Cohen, S., Wheelwright, S. and Fink, B. (2010) 'Sexually dimorphic traits (digit ratio, body height, empathizing & systemizing scores) and gender segregation between occupations: evidence from the BBC internet study'. *Personality and Individual Differences*, 49, pp.511-515

Manning, J. T. (2002) *Digit ratio: a pointer to fertility, behaviour, and health*. Rutgers University Press: New York

Manoach, D. S., Gollub, R. L., Benson, E. S., Searl, M. M., Goff, D. C., Halpern, E., Saper, C. B. and Rach, S. L. (2000) 'Schizophrenic subjects show aberrant fMRI activation of dorsolateral prefrontal cortex and basal ganglia during working memory performance'. *Biological Psychiatry*, 48, pp.99-109

Manson, C. and Winterbottom, M. (2012) 'Examining the association between empathising, systemising, degree subject and gender'. *Educational Studies*, 38:1, pp.73-88

Masento, N. A., Golightly, M., Field, D. T., Butler, L. T. and van Reekum, C. M. (2014) 'Effects of hydration status on cognitive performance and mood'. *The British Journal of Nutrition*, 111:10, pp.1841-1852

Mason, O., Linney, Y. and Claridge, G. (2005) 'Short scales for measuring schizotypy'. *Schizophrenia Research*. 78, pp.293–296

Maylor, E. A., Moulson, J. M., Muncer, A-M. and Taylor, L. A. (2002) 'Does performance on theory of mind tasks decline in old age?'. *British Journal of Psychology*, 93:4, pp.465-485

Mazza, M., DeRisio, A. and Surian, L. (2001) 'Selective impairments of theory of mind in people with schizophrenia'. *Schizophrenia Research*, 47, pp.299–308

McCabe, K., Houser, D., Ryan, L., Smith, V. and Trouard T. A. (2001) 'Functional imaging study of cooperation in two-person reciprocal exchange'. *Proceedings from the National Acadamy of Science*, 98:11, pp.832–835

McCarthy, M. M., Arnold, A. P., Ball, G. F., Blaustein, J. D. and De Vries, G. (2012) 'Sex differences in the brain: the not so inconvenient truth'. *The Journal of Neuroscience*, 32:7, pp.2241-2247

McCarthy, M. M. and Konkle, A. T. M. (2005) 'When is a sex difference not a sex difference?'. *Frontiers in Neuroendocrinology*, 26, pp.85-102

McCormick, L. M., Brumm, M. C., Beadle, J. N., Paradiso, S., Yamada, T. and Andreason, N. (2012) 'Mirror Neuron Function, Psychosis, and Empathy in Schizophrenia'. *Psychiatry Research: Neuroimaging,* 201, pp. 233-239

McGarth, J. J. (2005) 'Myths and plain truths about schizophrenia epidemiology- the NAPE lecture 2004'. *Acta Psychiatrica Scandinavica*, 111:1, pp.4-11

McGarth, J. J., Petersen, L., Agerbo, E., Mors, O., Mortensen, P. B. and Pedersen, C. B. (2014) 'A comprehensive assessment of parental age and psychiatric disorders'. *JAMA Psychiatry*, 71, pp.301-309

McGarth, J., Saha, S., Welham, J., El Saadi, O., MacCauley, C. and Chant, D. (2004) 'A systematic review of the incidence of schizophrenia: the distribution of rates and the influence of sex, urbanicity, migrant status and methodology'. *BMC Medicine*, 2:13. doi:10.1186/1741-7015-2-13

McKay, R., Langdon, R. and Colteart, M. (2006) 'Need for closure, jumping to conclusions, and decisiveness in delusion-prone individuals'. *The Journal of Nervous and Mental Disease*, 194:6, pp.422-426

McLean, C. P., Asnaani, A., Litz, B. T. and Hofmann, S. G. (2011) 'Gender differences in anxiety disorders: prevalence, course of illness, comorbidity and burden of illness'. *Journal of Psychiatric Research*, 45, pp.1027-1035

McLean, C. P. and Anderson, E. R. (2009) 'Brave men and timid women? A review of the gender differences in fear and anxiety'. *Clinical Psychology Review*, 29, pp.496-505

Mealey, A., Abbott, G., Byrne, L. K. and McGillivray, J. (2014) 'Overlap between autistic and schizotypal personality traits is not accounted for by anxiety and depression'. *Psychiatry Research*, 219:2, pp.380-385

Mendrek, A. (2015) 'Is it important to consider sex and gender in neurocognitive studies?' *Frontiers in Psychiatry*, 6:83, doi: 3389/fpsyt.2015.00083

Meng-Chuan, L., Lombardo, M. V., Chakrabarti, B., Ecker, C., Sadek, S. A., Wheelwright, S., Murphy, D. G. M., Suckling, J., Bullmore, E. T. and Baron-Cohen, S. (2012) 'Individual differences in brain structure underpin empathising- systemising cognitive styles in male adults'. *NeuroImage*, 61:4, pp.1347-1357

Menta, U. M., Thirthalli, J., Aneelraj, D., Jadhav, P., Gangadhar, B. N. and Keshavan, M. S. (2014) 'Mirror neuron dysfunction in schizophrenia and its functional implications: a systematic review'. Schizophrenia Review, 160:1-3, pp.9-19

Miller, D. I. and Halpern, D. F. (2014) 'The new science of cognitive sex differences'. *Trends in Cognitive Sciences*, 18:1, pp.37-45

MIND (2013) *Eating problems*. [online] Available at: <http://www.mind.org.uk/information-support/types-of-mental-health-problems/eating-problems/#.V2EV0_krLIU> [Accessed June 2016]

Minzenberg, M. J., Poole, J. H. and Vingradov, S. (2003) 'Slowed lexical access is uniquely associated with positive and disorganised symptoms in schizophrenia'. *Cognitive Neuropsychiatry*, 8:2, pp.107-127

Mitchell, K. J. and Johnson, M. K. (2009) 'Source monitoring 15 years later: what have we learned from fMRI about the neural mechanisms of source memory?', *Psychological Bulletin*, 135:4, pp.638–77

Mitropoulou, V., Harvey, P. D., Zegarelli, G., New, A. S., Silverman, J. M. and Siever, L. J. (2005) 'Neuropsychological performance in schizotypal personality disorder: importance of working memory'. *American Journal of Psychiatry*, 162, pp.1896-1903

Moffat, S., Elkins, W. and Resnick, S. (2006) 'Age differences in the neural systems supporting human allocentric spatial navigation'. *Neurobiology of Aging*, 27:7, pp.965-972

Moffat, S. D., Hampson, E. and Hatzipantelis, M. (1998) 'Navigation in a "virtual" maze: sex differences and correlation with psychometric measures of spatial ability in humans'. *Evolution and Human Behavior*, 19, pp.73-87

Mohring, N., Shen, C., Hahn, E., Ta, T. M., Dettling, M. and Neuhaus, A. H. (2015) 'Mirror neuron deficit in schizophrenia: evidence from repitition suppression'. *Schizophrenia Research*, 168, 1-2, pp.174-179

Montag, C., Heinz, A., Kunz, D. and Gallinat, J. (2007) 'Self-reported empathic abilities in schizophrenia'. *Schizophrenia Research,* 92, pp.85-89

Moos, W. H., Maneta, E., Pinkert, C. A., Irwin, M. H., Hoffman, M. E., Faller, D. V. and Steliou, K. (2016) 'Epigenetic treatment of neuropsychiatric disorders: autism and schizophrenia'. *Drug Development Research*, doi:10.1002/ddr.21295

Morison, I. M., Paton, C. J. and Cleverley, S. D. (2001) 'The imprinted gene and parent-of-origin effect database'. *Nucleic Acids Research*, 29:1, pp.275-276

Moritz, S. and Van Quaquebeke, N. (2014) 'Are you sure? Delusion conviction moderaes the behavioural and emotional consequences of paranoid ideas'. *Cognitive Neuropsychiatry*, 19, pp.164-180

Moritz, S., Van Quaquebeke, N. and Lincoln, T. M. (2012) 'Jumping to conclusions is associated with paranoia but not general suspiciousness. A comparison of two versions of the probabilistic reasoning paradigm'. *Schizophrenia Research and Treatment*, e384039. doi:10.1155/2012/384039

Moritz, S. and Woodward, T. S. (2006) 'Metacognitive control over false memories: a key determinant of delusional thinking'. *Current Psychiatry Reports*, 8, pp.184-190

Moritz, S., Woodward, T. S., Cuttler, C., Whitman, J. C. and Watson, J. M. (2004) 'False memories in schizophrenia'. *Neuropsychology*, 18:2, pp.276-283

Mottron, L., Duret, P., Mueller, S., Moore, R. D., Forgeot d'Arc, B., Jacuemont, S. and Xiong, L. (2015) 'Sex differences in brain plasticity: a new hypothesis for sex ratio bias in autism'. *Molecular Autism*, 6:33, doi: 10.1186/s13229-015-0024-1

Muncer, S. J. and Ling, J. (2016) 'Psychometric analysis of the empathy quotient (EQ) scale'. *Personality and Individual Differences*, 40:6, pp.1111-1119

Muris, P. and Steerneman, P. (1998) 'Comorbid anxiety symptoms in children with pervasive developmental disorders'. *Journal of Anxiety Disorders*, 12, pp.387-393

Naito, M. (2003) 'The relationship between theory of mind and episodic memory: Evidence for the development of autonoetic consciousness'. *Journal of Experimental Child Psychology*, 85:4, pp.312–336

Nash, A. (2014) 'Challenging dangerous ideas: a multi-disciplinary critique of evolutionary psychology'. *Dialectical Anthropology*, 38:38, pp.281-285

Nash, A. and Grossi, G. (2007) 'Picking Barbie's brain: inherent sex differences in scientific ability'. *Journal of Interdisciplinary Feminist Thought*, 2:1, pp. 1-23

Neave, N. and O'Connor, D. B. (2008) 'Testosterone and male behaviours'. *The Psychologist,* 22:1, pp.28-32

Nelson, B and Rawlings, D. (2010) 'Relating schizotypy and personality to the phenomenology of creativity'. *Schizophrenia Bulletin*, 32:6, pp.388-399

Nettle, D. (2007) 'Empathizing and systemizing: what are they, and what do they contribute to our understanding of psychological sex differences?'. *British Journal of Psychology*, 98, pp. 237-255

Nettle, D. (2006) 'Schizotypy and mental health amongst poets, visual artists, and mathematicians'. *Journal of Personality Research*, 40, pp.876-890

Newton, P. and Bristoll, H. (n.d) Psychometric Success, Spatial Ability, Practice Test 1. www.psychometric-sucess.com

Ngun, T. C., Ghahramani, N., Sanchez, F. J., Bocklandt, S. and Villan, E. (2012) 'The genetics of sex differences in brain and behaviour' *Frontier in Neuroendocrinology,* 32:2, pp.227-246

NHS (2016a) *Angelman syndrome* [online] Available at: <http://www .nhs.uk/conditions/angelman-syndrome/Pages/Introduction.aspx> [Accessed January 2016]

NHS (2016b) *Autism spectrum disorder (ASD)* [online] Available at: <http://www.nhs.uk/conditions/autistic-spectrum-disorder/pages/introduction. aspx> [Accessed March 2016]

NHS (2014) *Prader-Willis syndrome* [online] Available at: <http://www.nhs.uk /Conditions/prader-willi-syndrome/Pages/introduction.aspx> [accessed January 2016]

NIMH (The National Institute of Mental Health) (2016) *Eating Disorders* [online] Available at: <http://www.nimh.nih.gov/health/ topics/ eating-disorders/index.shtml> [Accessed June 2016]

Norton, P. J., Sexton, K. A., Walker, J. R. and Norton, R. G. (2005) 'Hierarchical model of vulnerabilities for anxiety: replication and extension with a clinical sample'. *Cognitive Behaviour Therapy*, 34:1, pp.50-63

Nosh, A. and Grossi, G. (2007) 'Picking Barbie's brain: inherent sex differences in scientific ability?'. *Journal of Interdisciplinary Feminist Thought,* 1:2, pp.1-23

O'Shea, A. G., Fein, D. A., Cillessen, A. H., Klin, A. and Schultz, R. T. (2005) 'Source memory in children with autism spectrum disorders'. *Developmental Neuropsychology*, 27:3, pp.337–60

Oberman, L.. M., Hubbard, E. M., McCleery, J. P., Altschuler, E. L., Ramachandran, V. S. and Pineda, J. A. (2005) 'EEG evidence for mirror neuron dysfunction in autism spectrum disorders'. *Cognitive Brain Research*, 24, pp.190–198

Oldershaw, A., Treasure, J., Hambrook, D., Tchanturia, K. and Schmidt, U. (2011a) 'Is anorexia nervosa a version of autism spectrum disorders?'. *European Eating Disorders Review*, 19:6, pp.462-474

Oldershaw, A., Hambrook, D., Stahl, D., Tchanturia, K., Treasure, J. and Schmidt, U. (2011b) 'The socio-emotional processing stream in anorexia nervosa'. *Neuroscience and Biobehavioural Review*, 35:3, pp.970-988

Palkhivala, A. (2009) 'Are autism and psychosis polar opposites?'. *Bulletin on Early Childhood Development,* 8:1, p.10

Parner, E. T., Baron-Cohen, S., Lauritsen, M. B., Jørgensen, M., Schieve, L. A., Yeargin-Allsopp, M. and Obel, C. (2012) 'Parental age and autism spectrum disorder'. *Annals of Epidemiology*, 22:3, pp.143-150

Parsons, T. D., Larson, P., Krat, K., Thiebaux, M., Bluestein, B., Buckwalter, G. J. and Rizzo, A. A. (2004) 'Sex differences in mental rotation and spatial rotation in a virtual environment'. *Neuropsychologia*, 42, pp.555-562

Peachey, M. (2015) *BBC NEWS: How the UK's new rules on paternity leave work* [online] Available at: <http://www.bbc.co.uk/news/business-32130481> [Accessed June 2016]

Pelphrey, K. A., Shultz, S., Hudac, C. M., Wyk, V. and Brent, C. (2011) 'Research review: constraining heterogeneity: the social brain and its development in Autism spectrum disorder'. [Abstract] *Journal of Child Psychology and Psychiatry,* 52:6, pp.631-644

Perner, J., Kloo, D. and Gornik, E. (2007) 'Episodic memory development: theory of mind is part of re-experiencing experienced events'. *Infant & Child Development*, 16, pp.471–490

Peters, E., Joesph, S., Day, S. and Garety, P. (2004) 'Measuring delusional ideation: the 21-item Peters et al. delusions inventory (PDI)'. *Schizophrenia Bulletin*, 30:4, pp.1005-1022

Peters, M., Jancke, L., Staiger, J. F., Schlaug, G., Huang, Y. and Steinmetz, H. (1998) 'Unsolved problems in comparing brain sizes in homo sapiens'. *Brain and Cognition*, 37, pp.254–285

Petty, L. K., Ornitz, E. M., Michelman, J. D. and Zimmerman, E. G. (1984) 'Autistic children who later become schizophrenic'. *Archives of General Psychiatry*, 41, pp.129-135

Phillips, L. H., MacLean, R. D. J. and Allen, R. (2002) 'Age and the understanding of emotions: neuropsychological and sociocognitive perspectives'. *Journal of Gerontology: Psychological Sciences*, 57, pp.526-530

Pigott, T. A. (1999) 'Gender differences in the epidemiology and treatment of anxiety disorders'. *Journal of Clinical Psychiatry*, 60:18, pp.4-15

Pilowsky, T., Yirmia, N., Arbelle, S. and Mozes, T. (2000) 'Theory of mind abilities of children with schizophrenia, children with autism and normally developing children'. *Schizophrenia Research*, 42, pp.145-155

Pollatos, O., Herbert, B. M., Schandry, R. and Gramann, K. (2008) 'Impaired central processing of emotional faces in anorexia nervosa'. *Psychosomatic Medicine*, 70, pp.701-708

Posserud, M. B., Lundervold, A. J. and Gillberg, C. (2006) 'Autistic features in a total population of 7-9 year old children assessed by the ASSQ (autism

spectrum screening questionnaire)'. *Journal of Child Psychology and Psychiatry and Allied Discipline*, 47, pp.167-175

Putz, D. A., Gaulin, S. J. C., Sporter, R. J. and McBurney, D. H. (2004) 'Sex hormones and finger length: What does 2D:4D indicate?'. *Evolution and Human Behaviour,* 25, pp.182–199

PWSA (2016) *About PWS* [online] Available at: <https://www.pwsa.co.uk/diagnosis/about-pws-new.html> [Accessed January 2016]

Quintana, J., Davidson, T., Kovalik, E., Marder, S. R. and Mazziotta, J. C. (2001) 'A compensatory mirror cortical mechanism for facial affect processing in schizophrenia'. *Neuropsychopharmacology,* 25, pp.915–924

Quinton, S. J., Smith, A. R., and Joiner, T. (2011) 'The 2nd to 4th digit ratio (2D:4D) and eating disorder diagnosis in women'. *Personality and Individual Differences*, 51:4, pp.402-405

Ragland, J. D., Laird, A. R., Ranganath, C., Blumenfeld, R. S., Gonzales, S. M. and Glahn, D. C. (2009) 'Prefrontal activation deficits during episodic memory in schizophrenia'. *American Journal of Psychiatry*, 166, pp.863-874

Ragsdale, G. and Foley, R. A. (2012) 'Testing the imprinted brain: parent of origin effects on empathy and systemising' *Evolution and Human Behaviour*, 33, pp.402-410

Rawlings, D. and Locarnini, A. (2008) 'Dimensional schizotypy, autism, and unusual word associations in artists and scientists'. *Journal of Research in Personality*, 42, pp.465-471

Raz, N. and Rodrique, K. M. (2006) 'Differential aging of the brain patterns, cognitive correlates and modifiers'. *Neuroscience and Biobehavioural Reviews*, 30:6, pp.730-748

Reuter-Lorenz, P., Jonides, J., Smith, E., Hartley, A., Miller, A., Marshuetz, C. and Koeppe, R. (2000) 'Age differences in the frontal lateralization of verbal and spatial working memory revealed by PET'. *Journal of Cognitive Neuroscience*, 12:1, pp.174-187

Rilling, J., Gutman, D., Zeh, T., Pagnoni, G., Berns, G. and Kilts, C. (2002) 'A neural basis for social cooperation'. *Neuron,* 35, pp.395–405

Ridout, N., Wallis, D. J., Autwal, Y. and Sellis, J. (2012) 'The influence of emotional intensity on facial emotion recognition in sub-clinical disordered eating'. *Appetite*, 59, pp.181-186

Ridout, N., Thom, C. and Wallis, D. (2010) 'Emotion recognition and alexithymia in females with non-clinical disordered eating'. *Eating Behaviours*, 11, pp.1-5

Rivet, T. T. and Matson, J. L. (2011) 'Review of gender differences in core symptomology in autism spectrum disorders'. *Research in Autism Spectrum Disorders*, 5:3, pp.957-976

Rizzolatti, G. and Craighero, L. (2005) 'Mirror neuron: a neurological approach to empathy'. *Neurobiology of Human Values* pp.107–123

Rizzolatti, G. and Craighero, L. (2004) 'The mirror-neuron system'. *Annual Review of Neuroscience,* 27, pp.169–192

Rizzolatti, G., Fadiga, L., Gallese, V. and Fogassi, L. (1996) 'Premotor cortex and the recognition of motor actions'. *Cognitive Brain Research*, 3, pp.131-141

Romano, E., Cosentino, L., Laviola, G. and De Filippis, B. (2016) 'Genes and sex hormones interaction in neurodevelopmental disorders'. *Neuroscience and Biobehavioural* Reviews, doi:http://dx.doi.org/ doi:10.1016/j.neubiorev .2016.02.019 NBR 2364

Roncone, R., Falloon, I. R. H., Mazza, M., De Risio, A., Pollice, R., Necozione, S., Morosini, P. L. and Casacchia, M. (2000) 'Is theory of mind in schizophrenia more strongly associated with clinical and social functioning than with neurocognitive deficits?' *Psychopathology*, 35, pp.280–288

Rosenzweig, E. S. and Barnes, C. A. (2003) 'Impact of aging on hippocampal function: plasticity, network dynamics, and cognition'. *Progress in Neurobiology*, 69:3, pp.143-179

Roskies, A. L. (2009) 'Brain-mind and structure-function relationships: a methodological response to Coltheart'. *Philosophy of Science*, 76:5, pp.927-939

Rowe, R., Pickles, A., Simonoff, E., Bulik, C. M. and Silberg, J. L. (2002) 'Bulimic symptoms in the Virginia twin study of adolescent behavioural development: correlates, comorbidity, and genetics'. *Biological Psychiatry*, 51:2, pp.172-182

Rumiati, R. and Humphreys, G. W. (2015) 'Cognitive neuroscience goes social'. *Cortex*, 70, pp.1-4

Rushe, T. M., Woodruff, P. W. R., Murray, R. M. and Morris, R. G. (1999) 'Episodic memory and learning in patients with chronic schizophrenia'. *Schizophrenia Research*, 35:1, pp.85-96

Russell, E. and Sofronoff, K. (2005) 'Anxiety and social worries in children with Asperger syndrome'. *Australian and New Zealand Journal of Psychiatry*, 39, pp.633-638

Russell, G. (1979) 'Bulimia nervosa: an ominous variant of anorexia nervosa'. *Psychological Medicine*, 9:3, pp.429-448

Russell, T., Schmidt, U. and Tchanturia, K. (2009) 'Aspects of social cognition in anorexia nervosa: affective and cognitive theory of mind'. *Psychiatry Research*, 168, pp.181-185

Russell-Smith, S. N., Bayliss, D. M., Mayberry, M. T. and Tomkinson, R. L. (2012) 'Are autism and positive schizotypy spectra diametrically opposed in empathizing and systemizing?'. *Journal of Autism and Developmental Disorders*, 43:4, pp.695-706

Russell-Smith, S. N., Marybery, M. T. and Bayliss, D. M. (2011) 'Relationships between autistic-like and schizotypy traits: an analysis using the autism spectrum quotient and Oxford-Liverpool inventory of feelings and experiences'. *Personality and Individual Differences*, 51:2, pp.128-132

Russell-Smith, S. N., Mayberry, M. T. and Bayliss, D. M. (2011) 'Relationship between autistic-like and schizotypy traits: an analysis using the autism

spectrum quotient and Oxford-Liverpool inventory of feelings and experiences'. *Personality and Individual Differences*, 51, pp.128-132

Russell-Smith, S. N., Maybery, M. T. and Bayliss, D. M. (2010) 'Are the autism and positive schizotypy spectra diametrically opposed in local versus global processing?'. *Journal of Autism and Developmental Disorders*, 40:8, pp.968-977

Rutter, M. (1996) 'Autism research: prospects and priorities'. Journal of Autism and Developmental Disorders, 26, pp.257-275

Sacher, J., Neumann, J., Okon-Singer, H., Gotowiec, S. and Villringer, A. (2011) 'Sexual dimorphism in the human brain: evidence from neuroimaging'. *Magnetic Resonance Imaging*, 31, pp.366-375

Sachse, M., Schlitt, S., Hainz, D., Ciaramidaro, A., Walter, H., Poustka, F., Bölte, S. and Freitag, C. M. (2014) 'Facial emotion recognition in paranoid schizophrenia and autism spectrum disorder'. *Schizophrenia Research*, 159, pp. 509-514

Saha, S., Chant, D., Welham, J. and McGarth, J. (2005) 'A systematic review of the prevalence of schizophrenia'. *PLoS Medicine*, doi: 10.1371/journal. pmed .0020141

Sanain, S., Schendel, D., Magnusson, P., Hultman, C., Surén, P., Susser, E., Grønborg, T., Gissler, M., Gunnes, N., Gross, R., Henning, M., Carter, K., Francis, R., Parner, E., Leonard, H., Rosanoff, M., Stoltenberg, C. and Rechenberg, A. (2016) 'Autism risk associated with parental age and with increased difference in age between the parents'. *Molecular Psychiatry*, 21, pp.693-700

Saunders, J., Randell, J. and Reed, P. (2012) 'Recall of false memories in individuals scoring high in schizotypy: memory distortions are scale specific'. *Journal of Behavior Therapy and Experimental Psychiatry*, 43:2, pp.711–5.

Schlaepfer, T. E., Harris, G. J., Tien, A. Y., Peng, L., Lee, S. and Pearlson, G. D. (1995) 'Structural differences in the cerebral cortex of healthy female

and male subjects: a magnetic resonance imaging study'. *Psychiatry Research: Neuroimaging*, 61:3, pp.129-135

Schmidt-Hansen, M. and Honey, R. C. (2009) 'Working memory and multidimensional schizotypy: dissociable influences of the different dimensions'. *Cognitive Neuropsychology*, 26:7, pp.655–70.

Schreck, K. A., Williams, K. and Smith, A. F. (2004) 'A comparison of eating behaviours between children with and without autism'. *Journal of Autism and Developmental Disorders*, 34:4, pp. 433-438

Schutle-Ruther, M., Markowitsch, H. J., Shah, N. J., Fink, G. R. and Piefe, M. (2008) 'Gender differences in brain networks supporting empathy'. *Neuroimage,* 42, pp.393-403

Seamon, J. P. Guerry, J. D., Marsh, G. P. and Tracy, M. C. (2002) 'Accurate and false recall in the Deese/Roediger and McDermott procedure: a methodological note on sex of participant'. *Psychological Reports*, 91, pp.423-427

Servin, A., Nordenstro¨m, A., Larsson, A. and Bohlin, G. (2003) 'Prenatal androgens and gender-typed behavior: A study of girls with mild and severe forms of congenital adrenal hyperplasia'. *Developmental Psychology*, 39, pp.440–450

Shamay-Tsoory, S. G., Shur, S., Barcai-oodman, L., Medlovich, S., Harari, H. and Levkovitz, Y. (2007) 'Dissociation of cognitive from affective componants of theory of mind in schizophrenia'. Psychiatry Research, 149:1-3, pp.11-23

Sharpe, E., Wallis, D. J. and Ridout, N. (2016) 'The influence of variations in eating disorder-related symptoms on processing of emotional faces in a non-clinical female sample: an eye tracking study'. *Psychiatry Research*, 240, pp.321-327

Sheitman, B., Bodfish, J. W. and Carmel, H. (2004) 'Are the negative symptoms of schizophrenia consistent with an autistic spectrum illness?' *Schizophrenia Research*, 69, pp.119-120

Shorter, E. V., Vaidya, E. and Fink, N. A. (2010) 'The failure of the schizophrenia concept and the argument for its replacement by hebephrenia: applying the medical model for disease recognition'. *Acta Psychiatrica Scandinavica*, 122:3, pp.173-183

Shtasel, D. L., Gur, R. E., Gallacher, F., Heimberg, C. and Gur, R. C. (1992) 'Gender differences in the clinical expression of schizophrenia'. *Schizophrenia Research*, 7:3, pp.225-231

Simons, J. S., Davis, S. W., Gilbert, S. J., Frith, C. D. and Burgess, P. W. (2006) 'Discriminating imagined from perceived information engages brain areas implicated in schizophrenia'. *NeuroImage*, 32, pp.696-703

Simpson, E. A., Nicolini, Y., Shetler, M., Suomi, S. J., Ferrari, P. F. and Paukner, A. (2016) 'Experience-independent sex differences in newborn macaques: females are more social than males'. *Nature*, 6:19669, doi:10.1038/srep19669

Skosnik, P. D., Spatz-Glenn, L. and Park, S. (2001) 'Cannabis use is associated with schizotypy and attentional disinhibition'. *Schizophrenia Research*, 48, pp.83-92

Skoukaskas, N. and Gallagher, L. 'Psychosis, affective disorders, and anxiety in autistic spectrum disorder: prevalence and nosological considerations'. *Psychopathology*, 43, pp.08-16

Skuse, D.H. (2009) 'Is autism really a coherent syndrome in boys and girls?'. *British Journal of Psychology,* 100, pp.33-37

Smeets, T., Jelicic, M. and Merckelbach, H. (2006) 'Stress-induced cortisol responses sex differences, and false recollections in the DRM paradigm'. *Biological Psychology*, 72, pp.164-172

Smit, H. (2010) 'A conceptual contribution to battles in the brain'. *Biological Philosophy,* 25, pp.803-821

Smith, A. (2014) *Older adults and technology use, Pew Research Center.* [online] Available from: <http://www.pewinternet.org/2014/04/03/older-adults-and-technology-use/> [Accessed April 2016]

Solomon, M., Ozonoff, S., Carter, C. and Caplan, R. (2008) 'Formal thought disorder and the autism spectrum: relationship with symptoms, executive control, and anxiety'. *Journal of Autism and Developmental Disorders*, 38:8, pp.1474-1484

Sommer, I. E., Aleman, A., Bouma, A. and Kahn, R. S. (2004) 'Do women really have more bilateral language representation than men? a meta-analysis of functional imaging studies'. Brain, 127, pp.1845–1852

Sowell, E. R., Levitt, J., Thompson, P. M., Holmes, C. J., Blanton, R. E., Kornsand, D. S., Caplan, R., McCracken, J., Asarnow, R. and Toga, A. W. (2000) 'Brain abnormalities in early-onset schizophrenia spectrum disorder observed with statistical parametric mapping of structural magnetic resonance images'. American Journal of Psychiatry, 157, pp.1475-1484

Sowell, E. R., Peterson, B. S, Kan, E., Woods, R. P., Yoshii, J., Bansal, R., Xu, D., Zhu, H., Thompson, P. M. and Toga, A. W. (2007) 'Sex differences in cortical thickness mapped in 176 healthy individuals between 7 and 87 years of age'. Cerebral Cortex, 17, pp.1550 –1560

Spain, D., Sin, J. and Freeman, D. (2016) 'Conceptualising paranoia in ASD: a systematic review and development of a theoretical framework'. Research in Autism Spectrum Disorders, 25, pp.97-111

Spek, A. A. and Wouters, S. G. M. (2010) 'Autism and schizophrenia in high-functioning adults: behavioral differences and overlap'. *Research in Autism Spectrum Disorders*, 4, pp. 709-717

Spitzer, R. L., Kroenke, K., Williams, J. B., W. and Lowe, B. (2006) 'A brief measure for assessing generalized anxiety disorder: the GAD-7'. *Archives of Internal Medicine*, 166:10, pp.1092-1097

Sporn, A., Addington, A., Gogray, N., Ordonez, A., Gornick, M., Clasen, L., Greenstein, D., Tossell, J. W., Gochman, P., Lenane, M., Sharp, W. S., Straub, R. E. and Rapoport, J. L. (2004) 'Pervasive developmental disorder and childhood onset schizophrenia: comorbid disorder or a phenotypic

variant of a very early onset illness?'. *Biological Psychiatry*, 55:10, pp.989-994

Stadler, M. A., Roediger, H. L. and McDermott, K. B. (1999) 'Norms for word lists that create false memories'. *Memory & Cognition*, 27, pp.494-500

Stanford, A. D., Messinger, J., Malaspina, D. and Corcoran, C. M. (2011) 'Theory of mind in patients at clinical high risk for psychosis'. *Schizophrenia Research*, 131, pp.11-17

Starling, J. and Dossetor, D. (2010) 'Pervasive developmental disorders and psychosis'. *Current Psychiatry Reports*, 11:3, pp. 190-196

Startup, H., Freeman, D. and Garety, P. (2008) 'Jumping to conclusions and persecutory delusions'. *European Psychiatry*, 23:6, pp.457-459

Steel, C., Garety, P. A., Freeman, D., Craig, E., Kuipers, E., Bebbington, P., Fowler, D. and Dunn, G. (2007) 'The multidimensional measurement of the positive symptoms of psychosis'. *International Journal of Methods in Psychiatric Research*, 16:2, pp.88-96

Stirling, J. D., Hellewell, J. S. E. and Hewitt, J. (1997) 'Verbal memory impairment in schizophrenia: no sparing of short-term recall'. *Schizophrenia Research*, 25:2, pp.85-95

Stodgell, C. J., Ingram, J. I. and Hyman, S. L. (2001) 'The role of candidate genes in unravelling the genetics of autism'. *International Review Research in Mental Retardation,* 23, pp.57–81

Stone, V. E., Baron-Cohen, S., Knight, R. T. (1999) 'Frontal lobe contributions to the theory of mind'. *Journal of Cognitive Neuroscience*, 10, pp.640-656

Stumpf, H. and Jackson, D. N. (1994) 'Gender related differences in cognitive abilities: evidence from a medical school admissions testing program'. *Personality and Individual Differences*, 17:3, pp.335-344

Sullivan, E. V., Rosenbloom, M., Serventi, K. L. and Pfefferbaum, A. (2004) 'Effects of age and sex on volumes of the thalamus, pons, and cortex'. *Neurobiology of Aging*, 25, pp.185–192

Sutton, J. (2010) 'Interview: The battle of the sexes, Jon Sutton interviews Cordelia Fine about neurosexism and more'. *The Psychologist,* 23:1, pp.900-904

Swinbourne, J. M. and Touyz, S. W. (2007) 'The co-morbidity of eating disorders and anxiety disorders: a review'. *European Eating Disorders Review*, 15:4, pp.253-274

Takagishi, H., Takahashi, T., Inukai, K., Shinada, M., Tanida, S., Takahashi, C., Mifune, N., Horita, Y., Hashimoto, H. Yang, L., Yokota, K., Kameda, T. and Yamagishi, T. (2010) 'Salivary testosterone levels and autism spectrum quoitent in adults;. *Neuroendocrinology Letters*, 31: 6, pp.101-106

Takeuchi, H., Taki, Y., Nouchi, R., Sekiguchi, A., Hashizume, H., Sassa, Y., Kotozaki, Y., Miyauchi, C. M., Yokoyama, R., Iizuka, K., Nakagawa, S., Nagase, T., Kuntitoki, K. and Kawashima, R. (2014). 'Association between resting state functional connectivity and empathising systemising'. *NeuroImage*, 99, pp.312-322.

Takeuchi, H., Taki, Y., Thyreau, B., Sassa, Y., Hashizume, H., Sekiguchi, A., Nagase, T., Nouchi, R., Fukushima, A. and Kawashima, R. (2013) 'White matter structures associated with empathizising and systemizing in young adults'. *NeuroImage*, 77, pp.222-236

Tantum, D. (2000) 'Psychological disorder in adolescents and adults with Asperger syndrome'. *Autism*, 4, pp.47-62

Tantum, D. (1991) Asperger syndrome in adulthood. In: Frith, U., ed. *Autism and Asperger Syndrome*. Cambridge: University Press. pp. 147-183

Tchanturia, K., Smith, E., Weineck, F., Fidanboylu, E., Kern, N., Treasure, J. and Baron-Cohen, S. (2013) 'Exploring autistic traits in anorexia: a clinical study'. *Molecular Autism*, 4:44, doi:10.1186/2040-2392-4-44

Tchanturia, K., Happe, F., Treasure, J. L. (2004) 'Theory of mind in anorexia nervosa'. *European Eating Disorders Review*, 12, pp.361-366

Thakkar, K. N., Matthews, N. and Park, S. (2008) 'A complete theory of psychosis and autism as diametric disorders of social brain must consider full

range of clinical syndromes'. *Behavioural and Brain Sciences*, 31:3, pp.277-278

Theoret, H., Halligan, E., Kobayashi, M., Fregni, F., Tager-Flusberg, H. and Pascual-Leone, A. (2005) 'Impaired motor facilitation during action observation in individuals with autism spectrum Disorder'. *Current Biology*, 15, pp.84–85

Tibi-Elhanany, Y. and Shamay-Tsoory, S. G. (2011) 'Social cognition in social anxiety: first evidence for increased empathic abilities'. *The Israel Journal of Psychiatry and Related Sciences*, 48:2, pp.98-106

Toichi, M., Kamio, Y., Okada, T., Sakihama, M., Youngstrom, E. A., Finding, R. L. and Yamamoto, K. (2002) 'A lack of self-consciousness in autism'. *The American Journal of Psychiatry*, 159:8, pp.1422–4

Treasure, J. L. (2007) 'Getting beneath the phenotype of anorexia nervosa: the search for viable endophenotypes and genotypes'. *Canadian Journal of Psychiatry*, 52, pp.212-219

Uhlhaas, P. L. and Silverstein, S. M. (2005) 'Perceptual organization in schizophrenic spectrum disorderss: empirical research and theoretical implications'. *Psychological Bulletin*, 131, pp.618-632

van de Beek, C., Thijssen, J. H. H., Cohen-Kettenis, P. T., van Goozen, S. H. M. and Buitelaar, J. K. (2004) 'Relationships between sex hormones assessed in amniotic fluid, and maternal and umbilical cord serum: what is the best source of information to investigate the effects of fetal hormonal exposure?'. *Hormone Behaviour*, 46, pp.663–669

Vandenberg, S. G. and Kuse, A. R. (1978) 'Mental rotations, a group test of three-dimensional spatial visualisation'. *Perceptual and Motor Skills*, 47, pp.599-604

Van der Gaag, R., Caplan, R., van Engeland, H., Loman, F. and Buitelaar, J. K. (2005) 'A controlled study of formal thought disorder in children with autism and multiple complex developmental disorders'. *Journal of Child and Adolescent Psychopharmacology*, 15:3, pp.465-476

van Rijn, S., Aleman, A., Swaab, H. and Kahn, R. S. (2005) 'Neurobiology of emotion and high risk for schizophrenia: role of the amygdala and the X-chromosome'. *Neuroscience and Bio-behavioural Reviews,* 29, pp.385–97

Van Schalkwyk, G. I., Peluso, F., Qayyum, Z., McPartland, J. C. and Volkmar, F. R. (2015) 'Varieties of misdiagnosis in ASD: an illustrative case series'. *Journal of Autism and Developmental Disorders*, 45, pp. 911-918

Verhoeven, W. M., Tuinier, S. and Curfs, L. M. (2003) 'Prader-Willi syndrome: The psychopathological phenotype in uniparental disomy'. *Journal of Medical Genetics*, 40, pp.112-116

Vesga-López, O., Schneier, F. R., Wang, S., Heimberg, R. G., Liu, S-M., Hasin, D. S. and Blanco, C. (2008) 'Gender differences in generalized anxiety disorder: results from the national epidemiologic survey on alcohol and related conditions (NESARC)'. *The Journal of Clinical Psychiatry*, 69:10, pp.1606-1616

Vinogradov, S., Willis-Shore, J., Poole, J. H., Marten, E., Ober, B. A. and Shenaut, G. K. (1997) 'Clinical and neurocognitive aspects of source monitoring errors in schizophrenia'. *American Journal of Psychiatry*, 154, pp.1530-1537

Volden, J. and Lord, C. (1991) 'Neologisms and idiosyncratic language in autistic speakers'. *Journal of Autism and other Developmental Disorders*, 21:2, pp.109-130

von Horn, A., Backman, L., Davidsson, T. and Hansen, S. (2010) 'Empathizing, systemizing and finger length ratio in a Swedish sample'. *Personality and Social Sciences*, 51, pp.31-37

Voracek, M. and Dressler, S. G. (2006) 'Lack of correlation between digit ratio (2D:4D) and Baron-Cohen's "Reading the Mind in the Eyes" test, empathy, systemizing, and autism-spectrum quotients in a general population sample'. *Personality and Individual Differences*, 41, pp.1481–1491

Voracek, M. (2008) 'Digit ratio as a marker for mental disorders: low (masculinised) 2D:4D in autism spectrum disorders, high (feminised) 2D:4D

in schizophrenic spectrum disorderss'. *Brain and Behavioural Sciences,* 31:3, pp.283-284

Wakabayashi, A., Sasaki, J. and Ogawa, Y. (2012) 'Sex differences in two fundamental cognitive domains' *Journal of Individual Differences*, 33:1, pp.24-34

Wakabayashi, A. and Nakazawa, Y. (2010) 'On relationships between digit ratio (2D:4D) and two fundamental cognitive styles, empathizing and systemizing, in Japanese sample'. *Personality and Individual Differences*, 49, pp.928-931

Wakabayashi, A., Baron-Cohen, S., Uchiyama, T., Yoshida, Y., Kuroda, M. and Wheelwright, S. (2006) 'Empathising and systemsing in adults with and without autism spectrum conditions cross cultural stability'. *Journal of Autism and Developmental Disorders*, 37, pp.1823-1832

Wakabayashi, A., Baron-Cohen, S., Uchiyama, T., Yoshida, Y., Kuroda, M. and Wheelwright, S. (2006) 'Empathising and systemising in adults with and without autism spectrum conditions cross cultural stability'. *Journal of Autism and Developmental Disorders,* 37, pp.1823-1832

Wakabayashi, A., Baron-Cohen, S., Wheelwright, S., Goldenfeld, N., Delaney, J., Fine, D., Smith, R. and Weil, L. (2006) 'Development of short forms of the empathy quotient (EQShort) and the systemizing quotient (SQ-Short)'. *Personality and Individual Differences*, 41:5, pp.929–940

Wallentin, M. (2009) 'Putative sex differences in verbal abilities and language cortex: a critical review'. *Brain and Language*, 108:3, pp.175-183

Wakefield, J. (2015) 'The generation that tech forgot' BBC NEWS. Available at: http://www.bbc.co.uk/news/technology-32511489

Wang, B. and Fu, X. (2011) 'Time course of effects of emotion on item memory and source memory for Chinese words'. *Neurobiology of Learning and Memory*, 95:4, pp.415–24

Watson, D. and Friend, R. (1969) 'Measurement of social-evaluative anxiety'. *Journal of Consulting and Clinical Psychology*, 33, pp.448-457

Weinstock, L. S. (1999) 'Gender differences in the presentation and management of social anxiety disorder'. *Journal of Clinical Psychiatry*, 60, pp.9-13

Weisbrot, D. M., Gadow, K. D., DeVincent, C. J. and Pomeroy, J. (2005) 'The presentation of anxiety in children with pervasive developmental disorders'. *Journal of Child and Adolescent Psychophamacology*, 15, pp.477-496

Weiss, E. M., Kemmler, G., Deisenhammer, E. A., Fleischhacker, W. W. and Delazer, M. (2003) 'Sex differences in cognitive functions'. *Personality and Individual Differences*, 35, pp.863-875

Wentz, E., Lacey, J. H., Waller, G., Rastam, M., Turk, J. and Gillberg, C. (2005) 'Childhood onset neuropsychiatric disorders in adult eating disorder patients: a pilot study'. *European Child and Adolescent Psychiatry*, 14, pp.431-437

Werling, D. M. and Geschwind, D. H. (2013) 'Sex differences in autism spectrum disorders'. *Current Opinion in Neurology*, 26:2, pp.146-153

Wheelwright, S. Baron-Cohen, S., Goldenfeld, N., Delaney, J., Fine, D., Smith, R., Weil, L. and Wakabayashi, A. (2006) 'Predicting Autism spectrum quotient (ASQ) from the Systemising quotient- revised (SQ-R) and empathising quotient (EQ)'. *Brain Research*, 1079:1, pp.47-56

White, S. W., Oswald, D., Ollendick, T. and Scahill, L. (2009) 'Anxiety in children and adolescents with autism spectrum disorders'. *Clinical Psychology Review*, 29, pp.216-229

Whitehouse, A., Maybery, M., Hart, R., Mattes, E., Newham, J., Sloboda, D., Stanley, F. and Hickey, M. (2010) 'Fetal androgen exposure and pragmatic language ability of girls in middle childhood: Implications for the extreme male brain theory of autism'. *Psychoneuroendocrinology,* 35, pp.1259-1264

Williams, D. L. and Minshew, N. J. (2007) 'The profile of memory function in children with Autism'. *Neuropsychology*, 20:1, pp.21–29

Williams, J. H. G., Whiten, A., Suddendorf, T. and Perrett, D. I. (2001) 'Imitation, mirror neurons and autism' *Neuroscience and Biobehavioral Reviews*, 25, pp.287–295

Wilson, S. T. and Stanley, B. (2006) 'Ethical concerns in schizophrenia research: looking back and moving forward'. *Schizophrenia Bulletin*, 32:1, pp.30-36

Wing, L. (1981) 'Sex ratios in early childhood autism and related conditions'. *Psychiatry Research*, 5:2, pp.129-137

Wizemann, T. M. and Pardue, M-L. eds. (2001) *Exploring the biological contributions to human health: does sex matter?* Washington D.C: National Academic Press

Wlodarski, R. (2015) 'The relationship between cognitive and affective empathy and human mating strategies'. Evolutionary Psychological Science, 1:4, pp.232-240

Wright, D. B and Skagerberg, E. M. (2012) 'Measuring empathizing and systemizing with a large US sample'. *PLoS One*, 7:2, e31661. doi:10.1371/journal.pone.0031661

Yamasue, H., Kuwabara, H., Kawakubo, Y. and Kasai, K. (2009) 'Oxytocin, sexually dimorphic features of the social brain and Autism'. *Psychiatry and Clinical Neurosciences,* 63, pp.129-143

Yorker, J. E., Eriksson, E., Nilsson, L-G. and Herlitz, A. (2003) 'Sex differences in episodic memory: minimal influence of estradiol'. *Brain and Cognition*, 52:2, pp.231-238

Zahn-Waxler, C., Shirtclif, E. A. and Marceau, K. (2008) 'Disorders of childhood and adolescence: gender and psychopathology'. *Annual Review of Clinical Psychology*, 4, pp.275-303

Zaki, J., Weber, J., Bolger, N. and Ochsner, K., (2009) 'The neural bases of empathic accuracy'. *Proceedings of the National Academy of Sciences of the United States of America*, 106, pp.11382–11387

Zucker, N. L., Losh, M., Bulik, C. M., LaBar, K. S., Piven, J. and Pelphrey, K. A. (2007) 'Anorexia nervosa and autism spectrum disorders: guided investigation of social cognitive endophenotypes'. *Psychological Bulletin*, 133:6, pp.976-1006

Appendix 1: The EQ

How to Fill Out the Questionnaire

Below is a list of statements. Please read each statement carefully and rate how strongly you agree or disagree with it by ticking the corresponding box. There are no right or wrong answers, or trick questions.

IN ORDER FOR THE SCALE TO BE VALID, YOU MUST ANSWER EVERY QUESTION.

	Strongly Agree	Slightly Agree	Slightly Disagree	Strongly Disagree
I can easily tell if someone else wants to enter a conversation				
I really enjoy caring for other people				
I find it hard to know what to do in a social situation. a				
I often find it difficult to judge if something is rude or polite. a				
In a conversation, I tend to focus on my own thoughts rather than on what my listener might be thinking. a				
I can pick up quickly if someone says one thing but means another.				
It is hard for me to see why some things upset people so much.a				
I find it easy to put myself in somebody else's shoes.				
I am good at predicting how someone will feel.				
I am quick to spot when someone in a group is feeling awkward or uncomfortable.				
I can't always see why someone should have felt offended by a remark.a				
I don't tend to find social situations confusing.				
Other people tell me I am good at understanding how they are feeling and what they are thinking				
I can easily tell if someone else is interested or bored with what I am saying.				
Friends usually talk to me about their problems as they say that I am very understanding.				
I can sense if I am intruding, even if the other person doesn't tell me.				
Other people often say that I am insensitive, though I don't always see why.a				
I can tune into how someone else feels rapidly and intuitively.				
I can easily work out what another person might want to talk about.				
I can tell if someone is masking their true emotion.				
I am good at predicting what someone will do.				
I tend to get emotionally involved with a friend's problems.				

Appendix 2: The SQ

How to Fill Out the Questionnaire

Below is a list of statements. Please read each statement carefully and rate how strongly you agree or disagree with it by ticking the corresponding box. There are no right or wrong answers, or trick questions.

IN ORDER FOR THE SCALE TO BE VALID, YOU MUST ANSWER EVERY QUESTION.

	Strongly Agree	Slightly Agree	Slightly Disagree	Strongly Disagree
If I were buying a car, I would want to obtain specific information about its engine capacity.				
If there was a problem with the electrical wiring in my home, I'd be able to fix it myself.				
I rarely read articles or web pages about new technology.a				
I do not enjoy games that involve a high degree of strategy.a				
I am fascinated by how machines work.				
In math, I am intrigued by the rules and patterns governing numbers.				
I find it difficult to understand instruction manuals for putting appliances together.a				
If I were buying a computer, I would want to know exact details about its hard disk capacity and processor speed.				
I find it difficult to read and understand maps.a				
When I look at a piece of furniture, I do not notice the details of how it was constructed.a				
I find it difficult to learn my way around a new city.a				
I do not tend to watch science documentaries on television or read articles about science and nature. a				
If I were buying a stereo, I would want to know about its precise technical features.				
I find it easy to grasp exactly how odds work in betting.				
I am not very meticulous when I carry out D.I.Y.a				
When I look at a building, I am curious about the precise way it was constructed.				
I find it difficult to understand information the bank sends me on different investment and saving systems. a				
When travelling by train, I often wonder exactly how the rail networks are coordinated.				
If I were buying a camera, I would not look carefully into the quality of the lens.a				
When I hear the weather forecast, I am not very interested in the meteorological patterns.a				
When I look at a mountain, I think about how precisely it was formed.				
I can easily visualize how the motorways in my region link up.				
When I'm in a plane, I do not think about the aerodynamics.a				
I am interested in knowing the path a river takes from its source to the sea.				
I am not interested in understanding how wireless communication works.a				

Appendix 3: The Say What? Task

Statements

- It would take 63 hours to walk from Leeds to Edinburgh **PRESENT**
- The M62 is the second busiest motorway in the UK **PRESENT**
- According to the telegraph, people in the UK are happiest on a Sunday- **FALSE**
- More people watch TV than read novels- **FALSE**
- Thursday is the day before Friday- **PRESENT**
- The UK imports 30,000 tonnes of apples every month **PRESENT**
- The scientific name for a rose is rosa- **FALSE**
- Elephants are the largest land animals on earth **PRESENT**
- According to the energy saving trust, you will pay £30 extra a year powering appliances on stand-by- **PRESENT**
- In 2013 183 million books were sold in the UK **PRESENT**
- On average an apple has 52 calories- **FALSE**
- When spring arrives, so do the daffodils **PRESENT**
- Asia is the world's largest continent- **PRESENT**
- The lifespan of an emperor penguin is 20 years – **FALSE**

The following page shows screengrabs of the Say What? Task. The first screenshot shows participant instructions. The second screenshot shows the clip. The third screenshot shows the source recall response screen.

In this task, you will watch 10 short video clips.

Try your best to pay attention to these clips.

Afterwards, you will be asked some questions about them.

Press space bar to begin the first clip.

Who said: *'It would take 63 hours to walk from Leeds to Edinburgh'?*

Appendix 4: The O-LIFE

The Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE) Short Version

Unusual Experiences (12 items)

1. When in the dark do you often see shapes and forms even though there is nothing there?
2. Are your thoughts sometimes so strong that you can almost hear them?
3. Have you ever thought that you had special, almost magical powers?
4. Have you sometimes sensed an evil presence around you, even though you could not see it?
5. Do you think that you could learn to read other's minds if you wanted to?
6. When you look in the mirror does your face sometimes seem quite different from usual?
7. Do ideas and insights sometimes come to you so fast that you cannot express them all?
8. Can some people make you aware of them just by thinking about you?
9. Does a passing thought ever seem so real it frightens you?
10. Do you feel that your accidents are caused by mysterious forces?
11. Do you ever have a sense of vague danger or sudden dread for reasons that you do not understand?
12. Does your sense of smell sometimes become unusually strong?

Cognitive Disorganisation (11 items)

13. Are you easily confused if too much happens at the same time?
14. Do you frequently have difficulty in starting to do things?
15. Are you a person whose mood goes up and down easily?
16. Do you dread going into a room by yourself where other people have already gathered and are talking?
17. Do you find it difficult to keep interested in the same thing for a long time?
18. Do you often have difficulties in controlling your thoughts?
19. Are you easily distracted from work by daydreams?
20. Do you ever feel that your speech is difficult to understand because the words are all mixed up and don't make sense?
21. Are you easily distracted when you read or talk to someone?
22. Is it hard for you to make decisions?
23. When in a crowded room, do you often have difficulty in following a conversation?

Introvertive Anhedonia (10 items)

24. Are there very few things that you have ever enjoyed doing?
25. Are you much too independent to get involved with other people?
26. Do you love having your back massaged?a
27. Do you find the bright lights of a city exciting to look at?a
28. Do you feel very close to your friends?a
29. Has dancing or the idea of it always seemed dull to you?
30. Do you like mixing with people?a
31. Is trying new foods something you have always enjoyed?a
32. Have you often felt uncomfortable when your friends touch you?
33. Do you prefer watching television to going out with people?

Impulsive Nonconformity (10 items)

34. Do you consider yourself to be pretty much an average sort of person?a
35. Would you like other people to be afraid of you?
36. Do you often feel the impulse to spend money which you know you can't afford?
37. Are you usually in an average kind of mood, not too high and not too low?a
38. Do you at times have an urge to do something harmful or shocking?
39. Do you stop to think things over before doing anything?a
40. Do you often overindulge in alcohol or food?
41. Do you ever have the urge to break or smash things?
42. Have you ever felt the urge to injure yourself?
43. Do you often feel like doing the opposite of what other people suggest even though you know they are right?

Key: a Score 1 for no, 0 for yes.

Appendix 5: EAT-26

Eating Attitudes Test (EAT-26)©

Instructions: This is a screening measure to help you determine whether you might have an eating disorder that needs professional attention. This screening measure is not designed to make a diagnosis of an eating disorder or take the place of a professional consultation. Please fill out the below form as accurately, honestly and completely as possible. There are no right or wrong answers. All of your responses are confidential.

Part A: Complete the following questions:

1) Birth Date Month:	Day:	Year:	2) Gender: Male ☐ Female ☐
3) Height Feet :	Inches:		
4) Current Weight (lbs.):	5) Highest Weight (excluding pregnancy):		
6) Lowest Adult Weight:	7: Ideal Weight:		

Part B: Check a response for each of the following statements:		Always	Usually	Often	Some times	Rarely	Never
1.	Am terrified about being overweight.	☐	☐	☐	☐	☐	☐
2.	Avoid eating when I am hungry.	☐	☐	☐	☐	☐	☐
3.	Find myself preoccupied with food.	☐	☐	☐	☐	☐	☐
4.	Have gone on eating binges where I feel that I may not be able to stop.	☐	☐	☐	☐	☐	☐
5.	Cut my food into small pieces.	☐	☐	☐	☐	☐	☐
6.	Aware of the calorie content of foods that I eat.	☐	☐	☐	☐	☐	☐
7.	Particularly avoid food with a high carbohydrate content (i.e. bread, rice, potatoes, etc.)	☐	☐	☐	☐	☐	☐
8.	Feel that others would prefer if I ate more.	☐	☐	☐	☐	☐	☐
9.	Vomit after I have eaten.	☐	☐	☐	☐	☐	☐
10.	Feel extremely guilty after eating.	☐	☐	☐	☐	☐	☐
11.	Am preoccupied with a desire to be thinner.	☐	☐	☐	☐	☐	☐
12.	Think about burning up calories when I exercise.	☐	☐	☐	☐	☐	☐
13.	Other people think that I am too thin.	☐	☐	☐	☐	☐	☐
14.	Am preoccupied with the thought of having fat on my body.	☐	☐	☐	☐	☐	☐
15.	Take longer than others to eat my meals.	☐	☐	☐	☐	☐	☐
16.	Avoid foods with sugar in them.	☐	☐	☐	☐	☐	☐
17.	Eat diet foods.	☐	☐	☐	☐	☐	☐
18.	Feel that food controls my life.	☐	☐	☐	☐	☐	☐
19.	Display self-control around food.	☐	☐	☐	☐	☐	☐
20.	Feel that others pressure me to eat.	☐	☐	☐	☐	☐	☐
21.	Give too much time and thought to food.	☐	☐	☐	☐	☐	☐
22.	Feel uncomfortable after eating sweets.	☐	☐	☐	☐	☐	☐
23.	Engage in dieting behavior.	☐	☐	☐	☐	☐	☐
24.	Like my stomach to be empty.	☐	☐	☐	☐	☐	☐
25.	Have the impulse to vomit after meals.	☐	☐	☐	☐	☐	☐
26.	Enjoy trying new rich foods.	☐	☐	☐	☐	☐	☐

Part C: Behavioral Questions: In the past 6 months have you:		Never	Once a month or less	2-3 times a month	Once a week	2-6 times a week	Once a day or more
A	Gone on eating binges where you feel that you may not be able to stop? *	☐	☐	☐	☐	☐	☐
B	Ever made yourself sick (vomited) to control your weight or shape?	☐	☐	☐	☐	☐	☐
C	Ever used laxatives, diet pills or diuretics (water pills) to control your weight or shape?	☐	☐	☐	☐	☐	☐
D	Exercised more than 60 minutes a day to lose or to control your weight?	☐	☐	☐	☐	☐	☐
E	Lost 20 pounds or more in the past 6 months	Yes ☐		No ☐			

* Defined as eating much more than most people would under the same circumstances and feeling that eating is out of control

Appendix 6: GAD-7

GAD-7 Anxiety

Over the last two weeks, how often have you been bothered by the following problems?	Not at all	Several days	More than half the days	Nearly every day
1. Feeling nervous, anxious, or on edge	0	1	2	3
2. Not being able to sleep or control worrying	0	1	2	3
3. Worrying too much about different things	0	1	2	3
4. Trouble relaxing	0	1	2	3
5. Being so restless that it is hard to sit still	0	1	2	3
6. Becoming easily annoyed or irritable	0	1	2	3
7. Feeling afraid, as if something awful might happen	0	1	2	3

Column totals _____ + _____ + _____ + _____ =

Total score _____

If you checked any problems, how difficult have they made it for you to do your work, take care of things at home, or get along with other people?

Not difficult at all	Somewhat difficult	Very difficult	Extremely difficult
☐	☐	☐	☐

Source: Primary Care Evaluation of Mental Disorders Patient Health Questionnaire (PRIME-MD-PHQ). The PHQ was developed by Drs. Robert L. Spitzer, Janet B.W. Williams, Kurt Kroenke, and colleagues. For research information, contact Dr. Spitzer at rls8@columbia.edu. PRIME-MD® is a trademark of Pfizer Inc. Copyright© 1999 Pfizer Inc. All rights reserved. Reproduced with permission

Scoring GAD-7 Anxiety Severity

This is calculated by assigning scores of 0, 1, 2, and 3 to the response categories, respectively, of "not at all," "several days," "more than half the days," and "nearly every day."
GAD-7 total score for the seven items ranges from 0 to 21.

0–4: minimal anxiety

5–9: mild anxiety

10–14: moderate anxiety

15–21: severe anxiety

Appendix 7: FNE (brief)

Brief Fear of Negative Evaluation Scale
Leary (1983)

Read each of the following statements carefully and indicate how characteristic it is of you according to the following scale:

1 = Not at all characteristic of me
2 = Slightly characteristic of me
3 = Moderately characteristic of me
4 = Very characteristic of me
5 = Extremely characteristic of me

_____ 1. I worry about what other people will think of me even when I know it doesn't make any difference.

_____ 2. I am unconcerned even if I know people are forming an unfavorable impression of me.

_____ 3. I am frequently afraid of other people noticing my shortcomings.

_____ 4. I rarely worry about what kind of impression I am making on someone.

_____ 5. I am afraid others will not approve of me.

_____ 6. I am afraid that people will find fault with me.

_____ 7. Other people's opinions of me do not bother me.

_____ 8. When I am talking to someone, I worry about what they may be thinking about me.

_____ 9. I am usually worried about what kind of impression I make.

_____ 10. If I know someone is judging me, it has little effect on me.

_____ 11. Sometimes I think I am too concerned with what other people think of me.

_____ 12. I often worry that I will say or do the wrong things.

From: Leary, M. R. (1983). A brief version of the Fear of Negative Evaluation Scale. *Personality and Social Psychology Bulletin, 9*, 371-376.

Appendix 8: Paranoia checklist

How often have you had any of these thoughts?

	Rarely	Once a month	Once a week	Several times a week	At least once a day
I need to be on my guard against others					
There might be negative comments being circulated about me					
People deliberately try to irritate me					
I might be being observed or followed					
People are trying to make me upset					
People communicate about me in subtle ways					
Strangers and friends look at me critically					
People might be hostile towards me					
Bad things are being said about me behind my back					
Someone I know has bad intentions towards me					
There is a possibility of a conspiracy against me					
People are laughing at me					
I am under threat from others					
I can detect coded messages about me in the press/TV/radio					
My actions and thoughts might be controlled by others					

How strongly do you believe these thoughts?

	I do not believe it	Believe it a little	Believe it somewhat	Believe it a lot	Absolutely believe it
I need to be on my guard against others					
There might be negative comments being circulated about me					
People deliberately try to irritate me					
I might be being observed or followed					
People are trying to make me upset					
People communicate about me in subtle ways					
Strangers and friends look at me critically					
People might be hostile towards me					
Bad things are being said about me behind my back					
Someone I know has bad intentions towards me					
There is a possibility of a conspiracy against me					
People are laughing at me					
I am under threat from others					
I can detect coded messages about me in the press/TV/radio					
My actions and thoughts might be controlled by others					

How upsetting are these thoughts for you?

	Not distressing	A little distressing	Somewhat distressing	Moderately distressing	Very distressing
I need to be on my guard against others					
There might be negative comments being circulated about me					
People deliberately try to irritate me					
I might be being observed or followed					
People are trying to make me upset					
People communicate about me in subtle ways					
Strangers and friends look at me critically					
People might be hostile towards me					
Bad things are being said about me behind my back					
Someone I know has bad intentions towards me					
There is a possibility of a conspiracy against me					
People are laughing at me					
I am under threat from others					
I can detect coded messages about me in the press/TV/radio					
My actions and thoughts might be controlled by others					

Appendix 9: The AQ

How to fill out the questionnaire

Below are a list of statements. Please read each statement very carefully and rate how strongly you agree or disagree with it by circling your answer.

DO NOT MISS ANY STATEMENT OUT.

Examples

E1. I am willing to take risks.	definitely agree	slightly agree	slightly disagree	definitely disagree
E2. I like playing board games.	definitely agree	slightly agree	slightly disagree	definitely disagree
E3. I find learning to play musical instruments easy.	definitely agree	slightly agree	slightly disagree	definitely disagree
E4. I am fascinated by other cultures.	definitely agree	slightly agree	slightly disagree	definitely disagree

1. I prefer to do things with others rather than on my own.	definitely agree	slightly agree	slightly disagree	definitely disagree
2. I prefer to do things the same way over and over again.	definitely agree	slightly agree	slightly disagree	definitely disagree
3. If I try to imagine something, I find it very easy to create a picture in my mind.	definitely agree	slightly agree	slightly disagree	definitely disagree
4. I frequently get so strongly absorbed in one thing that I lose sight of other things.	definitely agree	slightly agree	slightly disagree	definitely disagree
5. I often notice small sounds when others do not.	definitely agree	slightly agree	slightly disagree	definitely disagree
6. I usually notice car number plates or similar strings of information.	definitely agree	slightly agree	slightly disagree	definitely disagree
7. Other people frequently tell me that what I've said is impolite, even though I think it is polite.	definitely agree	slightly agree	slightly disagree	definitely disagree
8. When I'm reading a story, I can easily imagine what the characters might look like.	definitely agree	slightly agree	slightly disagree	definitely disagree
9. I am fascinated by dates.	definitely agree	slightly agree	slightly disagree	definitely disagree
10. In a social group, I can easily keep track of several different people's conversations.	definitely agree	slightly agree	slightly disagree	definitely disagree
11. I find social situations easy.	definitely agree	slightly agree	slightly disagree	definitely disagree
12. I tend to notice details that others do not.	definitely agree	slightly agree	slightly disagree	definitely disagree

13. I would rather go to a library than a party.	definitely agree	slightly agree	slightly disagree	definitely disagree
14. I find making up stories easy.	definitely agree	slightly agree	slightly disagree	definitely disagree
15. I find myself drawn more strongly to people than to things.	definitely agree	slightly agree	slightly disagree	definitely disagree
16. I tend to have very strong interests which I get upset about if I can't pursue.	definitely agree	slightly agree	slightly disagree	definitely disagree
17. I enjoy social chit-chat.	definitely agree	slightly agree	slightly disagree	definitely disagree
18. When I talk, it isn't always easy for others to get a word in edgeways.	definitely agree	slightly agree	slightly disagree	definitely disagree
19. I am fascinated by numbers.	definitely agree	slightly agree	slightly disagree	definitely disagree
20. When I'm reading a story, I find it difficult to work out the characters' intentions.	definitely agree	slightly agree	slightly disagree	definitely disagree
21. I don't particularly enjoy reading fiction.	definitely agree	slightly agree	slightly disagree	definitely disagree
22. I find it hard to make new friends.	definitely agree	slightly agree	slightly disagree	definitely disagree
23. I notice patterns in things all the time.	definitely agree	slightly agree	slightly disagree	definitely disagree
24. I would rather go to the theatre than a museum.	definitely agree	slightly agree	slightly disagree	definitely disagree

25. It does not upset me if my daily routine is disturbed.	definitely agree slightly agree slightly disagree definitely disagree
26. I frequently find that I don't know how to keep a conversation going.	definitely agree slightly agree slightly disagree definitely disagree
27. I find it easy to "read between the lines" when someone is talking to me.	definitely agree slightly agree slightly disagree definitely disagree
28. I usually concentrate more on the whole picture, rather than the small details.	definitely agree slightly agree slightly disagree definitely disagree
29. I am not very good at remembering phone numbers.	definitely agree slightly agree slightly disagree definitely disagree
30. I don't usually notice small changes in a situation, or a person's appearance.	definitely agree slightly agree slightly disagree definitely disagree
31. I know how to tell if someone listening to me is getting bored.	definitely agree slightly agree slightly disagree definitely disagree
32. I find it easy to do more than one thing at once.	definitely agree slightly agree slightly disagree definitely disagree
33. When I talk on the phone, I'm not sure when it's my turn to speak.	definitely agree slightly agree slightly disagree definitely disagree
34. I enjoy doing things spontaneously.	definitely agree slightly agree slightly disagree definitely disagree
35. I am often the last to understand the point of a joke.	definitely agree slightly agree slightly disagree definitely disagree
36. I find it easy to work out what someone is thinking or feeling just by looking at their face.	definitely agree slightly agree slightly disagree definitely disagree

37. If there is an interruption, I can switch back to what I was doing very quickly.	definitely agree	slightly agree	slightly disagree	definitely disagree
38. I am good at social chit-chat.	definitely agree	slightly agree	slightly disagree	definitely disagree
39. People often tell me that I keep going on and on about the same thing.	definitely agree	slightly agree	slightly disagree	definitely disagree
40. When I was young, I used to enjoy playing games involving pretending with other children.	definitely agree	slightly agree	slightly disagree	definitely disagree
41. I like to collect information about categories of things (e.g. types of car, types of bird, types of train, types of plant, etc.).	definitely agree	slightly agree	slightly disagree	definitely disagree
42. I find it difficult to imagine what it would be like to be someone else.	definitely agree	slightly agree	slightly disagree	definitely disagree
43. I like to plan any activities I participate in carefully.	definitely agree	slightly agree	slightly disagree	definitely disagree
44. I enjoy social occasions.	definitely agree	slightly agree	slightly disagree	definitely disagree
45. I find it difficult to work out people's intentions.	definitely agree	slightly agree	slightly disagree	definitely disagree
46. New situations make me anxious.	definitely agree	slightly agree	slightly disagree	definitely disagree
47. I enjoy meeting new people.	definitely agree	slightly agree	slightly disagree	definitely disagree
48. I am a good diplomat.	definitely agree	slightly agree	slightly disagree	definitely disagree

49. I am not very good at remembering people's date of birth.	definitely agree	slightly agree	slightly disagree	definitely disagree
50. I find it very easy to play games with children that involve pretending.	definitely agree	slightly agree	slightly disagree	definitely disagree

Developed by: The Autism Research Centre, University of Cambridge

Appendix 10: Chapter 9 supplementary data

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
						Lower Bound	Upper Bound		
Folded boxes	Male	8	3.5000	.92582	.32733	2.7260	4.2740	2.00	4.00
	Balanced	22	3.0000	1.06904	.22792	2.5260	3.4740	1.00	4.00
	Female	4	2.7500	.95743	.47871	1.2265	4.2735	2.00	4.00
	Total	34	3.0882	1.02596	.17595	2.7303	3.4462	1.00	4.00
Physical predictions questionnaire	Male	8	12.5000	6.74007	2.38296	6.8652	18.1348	.00	19.00
	Balanced	22	7.6818	4.35766	.92906	5.7497	9.6139	.00	17.00
	Female	4	6.7500	4.99166	2.49583	-1.1928	14.6928	.00	11.00
	Total	34	8.7059	5.35146	.91777	6.8387	10.5731	.00	19.00
	Male	8	14.1250	3.09269	1.09329	11.5410	16.7090	10.00	18.00
	Balanced	22	10.4091	3.52757	.75208	8.8451	11.9731	4.00	17.00
Intuitive physics task	Female	4	8.2500	2.98608	1.49304	3.4965	13.0035	5.00	12.00
	Total	34	11.0294	3.77781	.64789	9.7113	12.3476	4.00	18.00
	Male	8	12.8750	2.64237	.93422	10.6659	15.0841	10.00	18.00
	Balanced	22	13.0000	3.00793	.64129	11.6664	14.3336	9.00	19.00
CAM (films)	Female	4	14.2500	1.25831	.62915	12.2478	16.2522	13.00	16.00
	Total	34	13.1176	2.74962	.47159	12.1582	14.0771	9.00	19.00
	Male	8	71.0000	5.07093	1.79334	66.7606	75.2394	63.00	77.00
	Balanced	21	78.5238	6.72770	1.46810	75.4614	81.5862	61.00	91.00
CAM (faces and voices)	Female	4	77.7500	9.91211	4.95606	61.9776	93.5224	69.00	92.00
	Total	33	76.5061	7.31635	1.27357	74.0111	79.2010	61.00	92.00