

Autistic traits and cognition in individuals with obsessive
compulsive disorder.

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Thesis declaration form

I confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Signature:

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Date:

Overview

This thesis focuses on exploring similarities between obsessive compulsive disorder (OCD) and autism spectrum disorders (ASD). Part 1 reviews research literature examining the overlap of symptomatology and traits across the disorders. The reviewed studies provide evidence for elevated levels of ASD traits in some individuals with OCD and vice versa with variable results as to which specific traits this applies. None of the reviewed studies provides sufficient evidence to support or refute explanations for the nature of this apparent overlap in traits across disorders.

Part 2 reports an investigation into autistic cognition in a population of adults with OCD in relation to their self-reported autistic traits. Although the study provides some tentative evidence for some individuals with OCD having neurodevelopmental aetiology (e.g. atypical neurocognitive performances), group and multiple single case series analysis failed to identify relationships between autistic cognition and autistic traits at group and individual levels respectively. Whether the apparent elevation of self-reported autistic traits identified in this OCD population represents genuine ASD symptomatology is unclear and explanations for these ambiguous results are proposed together with directions for future research. This investigation formed part of a joint study with Josselyn Hellriegel (trainee clinical psychologist, UCL) (Hellriegel, 2014).

Part 3 discusses some of the practical, methodological and ethical complexities inherent in conducting research with a clinical population with significant mental health difficulties such as OCD, including challenges in recruitment, risk management and neurocognitive assessment. The importance of flexibility both in research design and analysis is emphasised. Benefits of employing multiple single case series analysis in heterogeneous populations such as OCD are highlighted.

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Part 1: Literature Review

The overlap of the symptomatology and traits of obsessive compulsive disorder and autism spectrum disorder: A review of the literature.

Abstract

Aim

To review current evidence for the overlap in symptomatology and traits of obsessive compulsive disorder (OCD) and autism spectrum disorder (ASD).

Method

A systematic search of online databases, PubMed and PsycINFO, and searching reference lists and citations of all relevant articles identified 16 studies meeting quality and relevance criteria for review.

Results

Research was categorised into four sections according to the key research questions and participants included: (1) OCD symptomatology in ASD populations; (2) ASD symptomatology in OCD populations; (3) OCD and ASD symptomatology within families; and (4) OCD and ASD symptomatology in non-clinical populations. The studies provide consistent evidence for the apparent existence of at least sub-clinical levels of ASD symptomatology in some individuals with OCD and vice versa, with variable results as to which specific symptoms this applies.

Conclusions

The review considers and finds inadequate evidence to support or refute three possible explanatory models for the identified elevation of traits across disorders; comorbidity, genuine symptom overlap and superficial symptom overlap. Measures of symptomatology employed struggled to discriminate adequately between specific diagnostic constructs. The studies highlight the need for clinicians to be mindful that repetitive behaviours in ASD may not be ego-syntonic and may cause as much distress as those similar repetitive behaviours seen in OCD.

Introduction

Obsessive compulsive disorder (OCD) is defined as a disorder where the repeated occurrence of obsessions and/or compulsions is of sufficient severity that they are time-consuming (> 1 hour per day) or cause marked distress or discomfort (American Psychiatric Association (APA), 2013). It has an estimated lifetime prevalence of between 1% and 2% of the general population (Clark, 2004), occurring slightly more in women than in men (Andrews, Henderson & Hall, 2001). It tends to take a chronic course with spontaneous remission being rare (Skoog & Skoog, 1999) and impacts negatively on daily living and personal attainment.

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterised by impaired communication and social interaction, repetitive behaviours and restricted interests (APA, 2013; World Health Organisation, 1992). Thus similarly to OCD, core symptoms of ASD are repetitive behaviour and compulsivity. It is a lifelong disorder with an estimated prevalence of 1% of the population and affects three times more males than females (Baird et al. 2006; Chakrabarti, & Fombonne, 2001).

The DSM-IV classified OCD as an anxiety disorder (APA, 2000) and as a unitary diagnostic entity. However, under DSM-5 OCD is classified not as an anxiety disorder but under ‘Obsessive Compulsive and Related Disorders’, with related diagnoses including body dysmorphic disorder, hoarding disorder, trichotillomania and skin picking disorder (APA, 2013). This change of classification followed increasing evidence to support the heterogeneity of symptoms which fall under an OCD diagnosis and the growing number of clinical researchers challenging the prevailing view of OCD as an anxiety disorder (Clark, 2004), a key argument being

that obsessions and compulsions, rather than anxiety, are the fundamental features of the disorder (Mataix-Cols, Pertusa & Leckman, 2007). It has been posited that possible differences in the biochemistry between OCD and other anxiety disorders and greater functional impairment in OCD exist (Enright, 1996). In addition, Hollander, Kim, Khanna and Pallanti (2007) noted that the neurocircuitry of OCD and anxiety disorders differ in that OCD demonstrates dysfunction in the frontal-striatal circuitry whilst anxiety disorders involve the amygdala and a fear response.

This literature review forms part of an ongoing debate as to whether OCD is an anxiety disorder, whether it is better defined as a neurodevelopmental disorder or whether within OCD both a neurodevelopmental and an anxiety subgroup exist. This debate is fueled by evidence suggesting that, despite significant progress in the efficacy of interventions for OCD, approximately 50% of patients remain clinically unwell following a drop out from or a limited response to recommended intervention (Abramowitz, 2006). Some suggest that the focus of research into OCD as an anxiety disorder characterised by harm avoidance (Calamari et al. 2006) at the expense of research into OCD motivated by feelings of incompleteness, may explain a lack of effective treatment for a large percentage of those with an OCD diagnosis (Ecker & Gonner, 2008).

Personality disorders and traits have also been emphasised as important in the treatment outcome of OCD (Bejerot, Nylander & Lindstrom, 2001). Specifically there is evidence that the presence of cluster A personality disorder (odd and eccentric), obsessive compulsive personality disorder, or total number of personality disorders are predictive of a poorer outcome in OCD (Baer et al. 1992; Cavadini, Erzegovesi, Ronchi, & Bellodi, 1997; MiniChiello, Baer & Jenike, 1987). Bejerot et al. (2001) suggested that a proportion of those classified with comorbid personality

disorder may in fact be individuals with high functioning ASD and noted that the negative predictors of treatment outcome of OCD are strikingly similar to characteristics common in ASD. For example, males living alone (Buchanan, Meng & Marks, 1996), difficulties with interpersonal relations (Fals-Stewart & Lucente, 1993), hoarding (Black et al. 1998), abnormal personality, social impairment, and childlessness (de Silva, Rachman, & Seligman, 1977) have all been found to be negative predictors in treatment outcome of OCD.

Consistent with Bejerot et al.'s (2001) theoretical proposition is the phenomenological overlap of some OCD symptoms with ASD. Many OCD patients are characterised by repetitive behaviours (similar to those observed in ASD). In OCD, many patients have ordering and symmetry compulsions, as well as repetition compulsions and a desire to achieve a "just right" feeling (Rapoport, 1989; Rasmussen & Eisen, 1992), which is often thought of as a particular OCD symptom dimension, i.e. "Symmetry and Ordering" (Baer, 1994). The overlap of symptom presentation at a phenomenological level appears striking. There is however a common assumption that these repetitive behaviours differ between disorders in that the repetitive behaviours of OCD are ego-dystonic (in conflict with the needs and goals of the ego or preferred identify of the individual) and as such cause distress, whereas the repetitive behaviours of ASD are ego-syntonic and as such do not cause the individual significant distress (Paula-Perez, 2013).

One study of children and adolescents with OCD found that half had low levels of activity and sociability and high levels of shyness (Ivarsson & WingeWestholm, 2004) and hypothesised that some of these individuals might have ASD traits. In the same study, the other half showed normal levels of activity and sociability, high levels of emotionality and low levels of shyness, perhaps more akin

to an anxiety disorder. ASD and OCD have also been found to be comorbid at a higher level than in the normal population (Williams, Higgins & Brayne, 2006). Thus, within the larger debate as to whether OCD is better defined as an anxiety or neurodevelopmental disorder, there is a question as to the relationship between OCD and ASD.

In light of the recent focus on and changes in conceptualisation of OCD, the similarities between OCD and ASD have fostered curiosity about the possibility of overlap. Numerous studies have investigated rates of comorbidity between the two disorders, a recent meta-analysis summarising these studies found 17.4% of individuals with ASD had comorbid OCD (van Steensel, Bögels & Perrin, 2011). However, these prevalence studies do not provide details regarding the quality of the symptoms measured; the possibility that elevated rates of comorbidity in fact reflect measurement error or symptom overlap requires a deeper analysis. Indeed, discriminating between superficial or genuine symptom overlap is a challenge in symptom focussed research and dependent on the specificity of measures of symptomatology employed. This challenge is exacerbated by the possible comorbidity of the two disorders.

There has been emerging research into whether OCD and ASD are interrelated and the nature of this relationship at a symptom level. Specifically some studies have focussed on OCD symptomatology in ASD, some on ASD symptomatology in OCD and others on shared symptomatology in the general population, or relatives of probands with one of the disorders, using symptom measures with proven psychometric value. If there is an overlap of symptomatology or, as Bejerot et al. (2001) suggest, a subgroup of individuals with a diagnosis of OCD who actually have high functioning ASD, it will be important to understand

further the nature of the relationship between the two disorders and how available measurement tools can support this understanding in order to inform better treatment packages for the individuals.

The current review

The purpose of this review is to summarise and critically evaluate studies that have investigated empirically the overlap in the symptomatology or traits of OCD and ASD in order to address two key aims:

1. To investigate whether there is an overlap between ASD and OCD, by considering whether OCD symptoms are more common than would be expected by chance amongst people with ASD and vice versa.
2. To provide insight into whether any observed symptom overlap reflects comorbidity, aetiological factors shared between OCD and ASD, or measurement error whereby distinct symptoms of one disorder bear a superficial similarity to those of another.

No previous reviews published in this area have systematically reviewed the overlap between the full range of ASD and OCD symptoms. Previous work in this area has tended to be non-systematic (Paula-Perez, 2013), or to focus only on an incomplete range of OCD / ASD symptoms (Chasson et al. 2011; Paula-Perez, 2013) or has failed to include and systematically review research into ASD symptoms in OCD (Fischer-Terworth & Probst, 2009). In addition, there have been a number of papers published in recent years which would not have been included in previous reviews.

Method

Search strategy

Articles were retrieved through (a) searching PubMed and PsycINFO electronic databases and (b) searching reference lists and citations of all relevant articles. No year limits were placed. Searches were restricted to English language articles with human subjects.

Search terms

The search focused on two domains in combination:

- 1) Obsessive compulsive disorder and 2) Autistic spectrum disorders.

An initial scope of the literature was conducted in order to identify relevant search terms. Based on this, the search terms presented below in table 1 were used.

Table 1: *Literature review search terms*

Any of following terms for obsessive compulsive disorder	Combined with "AND"	Any of following terms for autistic spectrum disorders
Obsessive Compulsive Disorder* or Obsess* or Compuls* or OCD or "Obsessive compulsive symptoms" or "Obsession* and Compulsion*" or "obsessive-compulsive"		ASD or Autism Spectrum Disorder* or Autis* or Asperg* or HFA or "High Functioning Autism" or "High-Functioning-Autism" or "Autism-spectrum" or Autistic

* Indicates that terms were truncated to allow for multiple word endings. Phrases in quotation marks indicate that word streams were taken as a whole to search for specific phrases.

The search was designed to identify only those studies with at least one search term from each of the two domains in the title, abstract or as a keyword.

Searches of PsycINFO and PubMed yielded 665 and 406 results respectively, with much overlap in these results.

Selection strategy

The titles and abstracts of all articles identified by electronic searches were screened by the author to exclude duplicates and to evaluate the studies against the following inclusion criteria:

Inclusion criteria

The following criteria were used to determine whether a study should be included in the review:

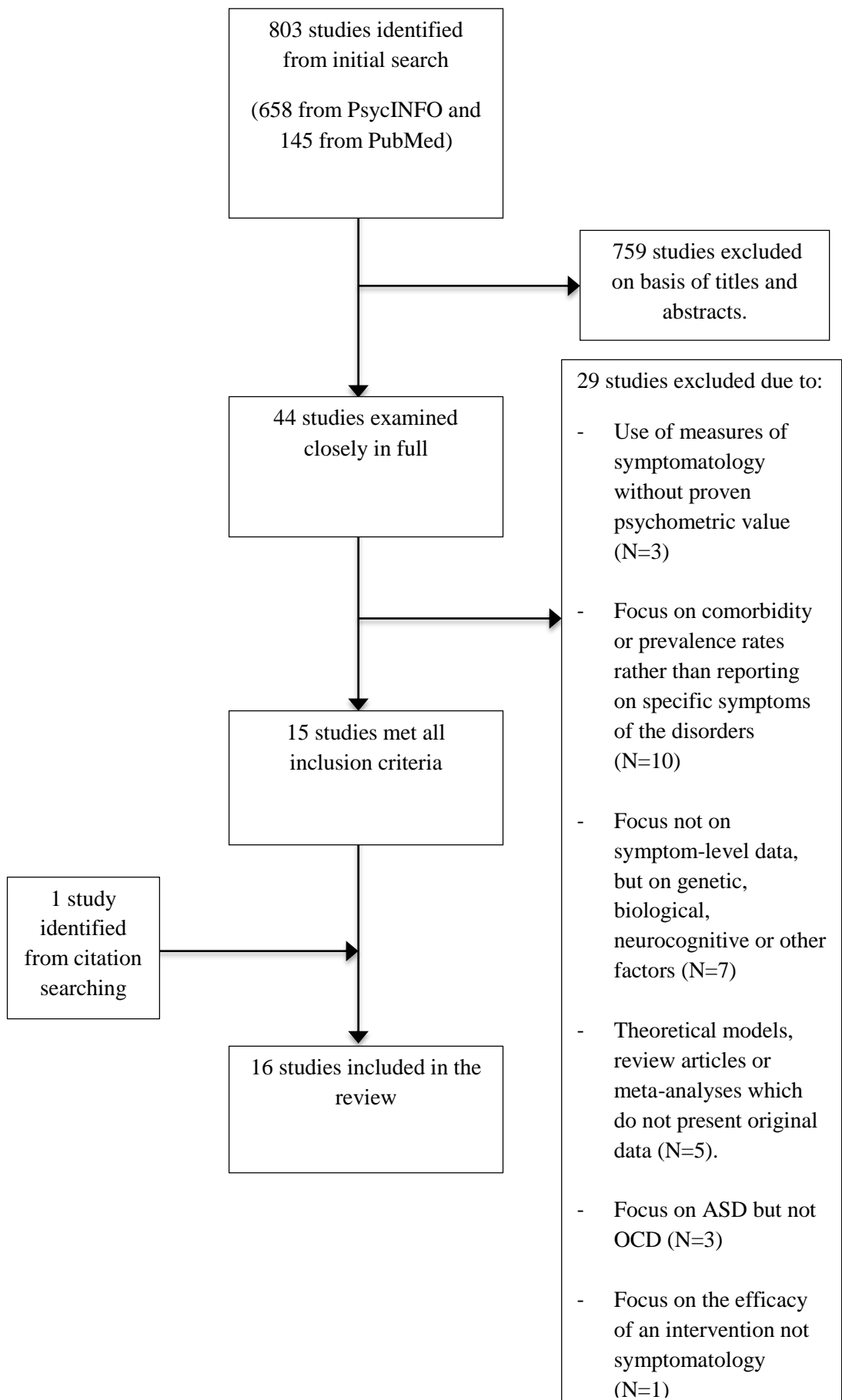
- The article related to humans and was published in English, in a peer-reviewed journal to control for quality;
- The study measured OCD symptoms in a population with a diagnosis of autism or autism probands using at least one OCD measure with proven psychometric value, that is measures which have published information evidencing satisfactory reliability and validity, OR
- The study measured ASD symptoms in a population with a diagnosis of OCD or OCD probands using at least one ASD measure with proven psychometric value, OR
- The study measured symptomatology of both ASD and OCD in a non-clinical population using at least one measure of ASD with proven psychometric value and one measure of OCD with proven psychometric value.

Study selection process

Figure 1 shows the study selection process. The electronic search returned a total of 803 studies. Studies were first screened by titles and abstracts resulting in a list of 44 potentially eligible studies, the manuscripts of which were examined in full. Many articles were excluded at the stage of scanning abstracts because they clearly did not meet one or more of the inclusion criteria e.g. studies which focused on OCD but not ASD, ASD but not OCD or neither disorder (N=363); single case studies (N=70); studies focussing on the efficacy of an intervention on the reduction of symptoms rather than on gathering symptom specific information (N=57); articles presenting theoretical models, review articles or meta-analyses which did not present original data (N=169); studies detailing comorbidity or prevalence rates rather than reporting on specific symptoms of the disorders (N=22); studies exploring ASD and OCD which did not present symptom-level data but rather focused on genetic, neurocognitive, biological or other factors (N=72); and studies using measures of symptoms of OCD or ASD without proven psychometric value (N=6).

Fifteen of the 44 potentially eligible studies met all the inclusion criteria. The 29 studies excluded at this stage either used measures of symptoms of OCD or ASD without proven psychometric value (N=3); focussed on comorbidity or prevalence rates rather than reporting on specific symptoms of the disorders (N=10); focussed not on symptom-level data, but on genetic, biological, neurocognitive or other factors (N=7); presented theoretical models, were review articles or meta-analyses which did not present original data (N=5); focussed on ASD but not OCD (N=3); or focussed on the efficacy of an intervention not symptomatology (N=1). An additional study was identified through citation searching. In total 16 studies were included in the review.

Figure 1: *Study selection flowchart*



Results:

A summary of the 16 studies included is presented in Table 2. The studies are categorised into four sections according to the key research questions and participants included: (1) OCD symptomatology in ASD populations which includes ten studies; (2) ASD symptomatology in OCD populations which includes 5 studies; (3) OCD and ASD symptomatology within families which includes 2 studies; and (4) OCD and ASD symptomatology in non-clinical populations which includes 1 study. Two studies explored both ASD symptomatology in OCD populations and OCD symptomatology in ASD populations and as such are included in both sections 1 and 2. Table 3 describes the measures employed by research included in this review.

Table 2: *Summary table of reviewed studies*

Author	Populations (N)	Diagnostic instrument (delivered by)	Group matching / Controls	Measures of symptomatology (delivered by)	Conclusion
Obsessive Compulsive symptoms and traits in populations with ASD					
McDougle et al. 1995	1. Adults with ASD-(50) 2. Adults with OCD-(50)	Groups recruited from specialist clinics – Diagnosis not independently verified.	<ul style="list-style-type: none"> • Groups matched for age and gender. • OCD group excluded those with mental retardation or borderline intellectual functioning. • Excluded if history of other significant neurological or medical illnesses. • All participants- medication free at time of study. 	OCD symptomatology: <ul style="list-style-type: none"> • The YBOCS-(not specified) 	Types of repetitive thoughts and behaviour in adults with OCD vs. ASD are significantly different.
Russell Mataix-Cols, Anson & Murphy, 2005	1. Adults with HFA-(40) 2. Adults with OCD-(45)	Groups recruited from specialist clinics 1. Psychiatric interview (Psychiatrist)+ 58% ADI. 2. Psychiatric interview (Psychiatrist).	<ul style="list-style-type: none"> • Matched for gender • Excluded if IQ<70, comorbid psychosis and/or substance misuse. 	OCD symptomatology: <ul style="list-style-type: none"> • YBOCS-(experienced clinician). 	<ul style="list-style-type: none"> • Obsessions and compulsions, which are distressing and time-consuming, are common in adults with HFA. • Types of obsessions and compulsions are similar.

Author	Populations (N)	Diagnostic instrument (delivered by)	Group matching / Controls	Measures of symptomatology (delivered by)	Conclusion
*Zandt, Prior, & Kyrios, 2007	<ol style="list-style-type: none"> 1. Children with autism-(19) 2. Children with OCD-(17) 3. TD children-(18) 	<ul style="list-style-type: none"> • Clinical interview with parents and children including language and cognitive assessments–(experienced professionals). 	<ul style="list-style-type: none"> • Excluded if comorbid neurological disorder, intellectual disability, language disorder and/or Axis 1 disorder. • Verbal and performance IQ measured (WISC-III). 	<p>ASD symptomatology:</p> <ul style="list-style-type: none"> • RBQ (parental-report) <p>OCD symptomatology:</p> <ul style="list-style-type: none"> • CYBOCS – (Clinical Psychologist) 	<ul style="list-style-type: none"> • Similar levels of sameness behaviours and repetitive movements in two clinical groups. • Suggests types of behaviours differ between the groups; obsessions and compulsions being less sophisticated in ASD group.
*Cath, Ran, Smit, van-Balkom & Comjris, 2008	<ol style="list-style-type: none"> 1. Adults with ASD-(12) – (a) with comorbid OCD-(6) (b) with comorbid SAD-(6) 2. Adults with OCD-(12) 3. Healthy adults-(12) 	<ol style="list-style-type: none"> 1. ASD assessed using a clinically structured interview in line with DSM-IV–(Independent clinicians). 2. OCD only diagnosis confirmed using the SCID-I (not specified). 3. The SCID-I used to screen control group (not specified). 	<ul style="list-style-type: none"> • Groups matched for age, sex and educational level. • Excluded if comorbid severe depression, psychosis, mental deficiency or inability to read/speak Dutch. 	<p>ASD symptomatology:</p> <ul style="list-style-type: none"> • AQ (self-report). <p>OCD symptomatology:</p> <ul style="list-style-type: none"> • YBOCS (not specified) • Four questions measuring ego-dystonia of repetitive symptoms (not specified). 	<p>There is phenomenological overlap of autistic-like traits in comorbid ASD and pure OCD - shared difficulties in social skills and attention to detail may reflect symptom and aetiological overlap.</p>

Author	Populations (N)	Diagnostic instrument (delivered by)	Group matching / Controls	Measures of symptomatology (delivered by)	Conclusion
Ruta, Mugno, D'Arrigo, Vitiello, & Mazzone, 2010	<ol style="list-style-type: none"> 1. Children with ASD-(18) 2. Children with OCD-(20) 3. TD children-(22) 	<ol style="list-style-type: none"> 1. ASD diagnosis confirmed using ADI-R, ADOS and ASD-I (not specified). 2. OCD diagnosis confirmed using the K-SADS-PL (not specified). 3. The K-SADS-PL used to screen control group (not specified). 	<ul style="list-style-type: none"> • Exclusion of individuals with mental retardation, neurological diseases, ADHD, tic disorder. • No participants receiving psychotropic medication or psychological therapy at time of study. • Analysis revealed no significant between group differences in age, gender, IQ. 	<p>OCD symptomatology:</p> <ul style="list-style-type: none"> • CY-BOCS-(Principal investigator) 	<ul style="list-style-type: none"> • Children with OCD and ASD report more obsessions and compulsions than TD children. • Types of OC symptoms endorsed by the ASD vs. OCD group differed significantly
Mack et al. 2010	<ol style="list-style-type: none"> 1. Children with both OCD and ASD-(12) 2. Children with OCD only-(12) 	<p>Groups recruited from specialist clinic.</p> <ol style="list-style-type: none"> 1. Diagnosis of ASD in accordance with ICD-10 criteria (clinical team). Consensus of 1 experienced clinician obtained. 	<ul style="list-style-type: none"> • Matched for gender and age. • IQ information not collected for all participants. • Children with other comorbidities excluded. 	<p>OCD symptomatology::</p> <ul style="list-style-type: none"> • CYBOCS • ChOCI (experienced clinician). <p>Broader difficulties:</p> <ul style="list-style-type: none"> • SDQ-child, parent, teacher versions-(Self-report). 	<ul style="list-style-type: none"> • Children with ASD may experience OC symptoms that are as impairing and distressing as those in OCD. • OC symptom type and frequency does not differ between groups significantly.

Author	Populations (N)	Diagnostic instrument (delivered by)	Group matching / Controls	Measures of symptomatology (delivered by)	Conclusion
Dewrang & Dahlgren Sandberg, 2011	<ol style="list-style-type: none"> Adolescents/young adults with Asperger's disorder-(27) TD adolescents and young adults-(14) 	<ol style="list-style-type: none"> Previous diagnosis of ASD (experienced clinician). Not verified. The absence of developmental disorder/mental health difficulty not verified. 	<ul style="list-style-type: none"> IQ of all participants in ASD group reported to be in 'normal range'. (No evidence of verification) Groups matched for age. 	<p>OCD symptomatology:</p> <ul style="list-style-type: none"> The COIS-(Parents and young person - self-report). The CYBOCS-(Researcher). 	<ul style="list-style-type: none"> No evidence of OCD symptomatology as described in DSM-IV in Asperger's disorder group. Some evidence of greater difficulties with OC behaviours and social interaction in ASD group from pre-school and throughout school.
Lewin Wood, Gunderson, Murphy & Storch, 2011	<ol style="list-style-type: none"> Children with OCD only -(35) Children with both OCD and ASD-(35) 	<ol style="list-style-type: none"> 1&2 – OCD Diagnosis (and non-ASD comorbid diagnoses) made using the ADIS-IV-C/P and confirmed by review of clinical records and unstructured clinical interview (Senior Clinician). ASD diagnoses confirmed using ADI-R and ADOS (trained rater), an unstructured clinical interview, observation of child and review of records (Child psychologist and/or psychiatrist) 	<ul style="list-style-type: none"> Inclusion criteria – OCD primary and most impairing diagnosis. Excluded if bipolar disorder, psychotic disorder, current suicidality and/or IQ <70. Matched for age and gender. Other demographic variables equivalent between groups 	<p>OCD Symptomatology:</p> <ul style="list-style-type: none"> CYBOCS-(trained rater) 	<ul style="list-style-type: none"> OC like repetitive actions/ behaviours are no more common in individuals with OCD+ASD vs. those with pure OCD There may be a phenotypical alteration of OCD in ASD.

Author	Populations (N)	Diagnostic instrument (delivered by)	Group matching / Controls	Measures of symptomatology (delivered by)	Conclusion
Anagnostou et al. 2011	Children and young adults with ASD (181)	ASD diagnosis confirmed using ADI-R (not specified).	<ul style="list-style-type: none"> Excluded if individual from family with a member known to have a medical condition associated with autism (e.g., fragile X, PKU). 	<p>OCD symptomatology:</p> <ul style="list-style-type: none"> YBOCS – (not specified) 	<ul style="list-style-type: none"> Factor analysis of YBOCS scores revealed 4 factor structure which differs from results of factor analyses of YBOCS scores in OCD populations
Spiker, Lin, Van Dyke & Wood, 2012	Children with HFA and a comorbid anxiety disorder - separation anxiety disorder (SAD), social phobia, generalised anxiety, or obsessive compulsive disorder (OCD) (68-specific group numbers not specified)	<ol style="list-style-type: none"> ASD diagnosis confirmed using ADI-R, ADOS-module 3, parent report and review of previous assessments (not specified) Anxiety disorder confirmed using semi structured interview ADIS-C/P for 84% of children. (Not specified). 	<ul style="list-style-type: none"> All children had verbal abilities >70 on standardised cognitive assessment. Children either not taking medication or on stable dose of medication (i.e. \geq one month same dosage prior to assessment). 	<p>Restrictive interests (RI):</p> <ul style="list-style-type: none"> YSIS–(parent report) <p>OCD symptomatology:</p> <ul style="list-style-type: none"> CYBOCS – (trained clinicians) 	<ul style="list-style-type: none"> Children with symbolically enacted RIs exhibit significantly more obsessions and compulsions than those without these RIs and are related to obsessive hoarding, aggressions and miscellaneous obsessions. Symbolically enacted RI may operate as a maladaptive coping strategy similar to OCD compulsions or these ASD-RI behaviours and OCD symptoms may be confused due to measurement error.

Author	Populations (N)	Diagnostic instrument (delivered by)	Group matching / Controls	Measures of symptomatology (delivered by)	Conclusion
Autism spectrum disorder symptoms and traits in populations with OCD					
*Zandt, Prior, & Kyrios, 2007	<ol style="list-style-type: none"> 4. Children with autism-(19) 5. Children with OCD-(17) 6. TD children-(18) 	<ul style="list-style-type: none"> • Clinical interview with parents and children including language and cognitive assessments–(experienced professionals). 	<ul style="list-style-type: none"> • Excluded if comorbid neurological disorder, intellectual disability, language disorder and/or Axis 1 disorder. • Verbal and performance IQ measured (WISC-III). 	<p>ASD symptomatology:</p> <ul style="list-style-type: none"> • RBQ (parental-report) <p>OCD symptomatology:</p> <ul style="list-style-type: none"> • CYBOCS – (Clinical Psychologist) 	<ul style="list-style-type: none"> • Similar levels of sameness behaviours and repetitive movements in two clinical groups. • Suggests types of behaviours differ between the groups; obsessions and compulsions being less sophisticated in ASD group.
*Cath, Ran, Smit, van-Balkom & Comjris, 2008	<ol style="list-style-type: none"> 4. Adults with ASD-(12) – (a) with comorbid OCD-(6) (b) with comorbid SAD-(6) 5. Adults with OCD-(12) 6. Healthy adults-(12) 	<ol style="list-style-type: none"> 4. ASD assessed using a clinically structured interview in line with DSM-IV–(Independent clinicians). 5. OCD only diagnosis confirmed using the SCID-I (not specified). 6. The SCID-I used to screen control group (not specified). 	<ul style="list-style-type: none"> • Groups matched for age, sex and educational level. • Excluded if comorbid severe depression, psychosis, mental deficiency or inability to read/speak Dutch. 	<p>ASD symptomatology:</p> <ul style="list-style-type: none"> • AQ (self-report). <p>OCD symptomatology:</p> <ul style="list-style-type: none"> • YBOCS (not specified) • Four questions measuring ego-dystonia of repetitive symptoms (not specified). 	<p>There is phenomenological overlap of autistic-like traits in comorbid ASD and pure OCD - shared difficulties in social skills and attention to detail may reflect symptom and aetiological overlap.</p>

Author	Populations (N)	Diagnostic instrument (delivered by)	Group matching / Controls	Measures of symptomatology (delivered by)	Conclusion
Ivarsson & Melin (2008)	Children with OCD-(109).	OCD diagnosis confirmed by CYBOCS-(not specified)	Excluded if previous primary diagnosis of mental retardation, psychotic disorders, anorexia nervosa or autism.	<p>ASD symptomatology:</p> <ul style="list-style-type: none"> • ASSQ parental rating scale-(self-report). <p>Psychiatric disorders:</p> <ul style="list-style-type: none"> • KSADS-PL interview-(child psychiatrists). 	<ul style="list-style-type: none"> • ASD traits are common in paediatric patients with OCD. • 60% of variance of ASD traits in OCD not explained by comorbidities-suggests OCD itself is associated with some lower level ASD traits.
Anholt et al. (2010)	<ol style="list-style-type: none"> 1. Adults with OCD-(109). 2. Healthy adults-(87) 	<ol style="list-style-type: none"> 1. OCD diagnosis confirmed using the SCID-I (not specified). 2. The SCID-I used to screen control group (not specified). 	<ul style="list-style-type: none"> • Preliminary analysis revealed group discrepancy in education level-subsequently treated as a covariate. • Patients with OCD excluded if comorbid psychosis, substance dependence, 'mental deficiency' or unable to speak/read Dutch. 	<p>ASD symptomatology:</p> <ul style="list-style-type: none"> • AQ Danish version -(self-report) <p>OCD symptomatology:</p> <ul style="list-style-type: none"> • YBOCS-(self-report) 	Substantial overlap in disorder symptomatology exists perhaps indicating overlapping aetiologies or shared executive dysfunction between disorders.

Author	Populations (N)	Diagnostic instrument (delivered by)	Group matching / Controls	Measures of symptomatology (delivered by)	Conclusion
Weidle, Melin, Drotz, Jozefiak & Ivarsson (2012)	<ol style="list-style-type: none"> 1. Children with OCD-(105) 2. TD children-(108) 	<ol style="list-style-type: none"> 1. OCD diagnosis confirmed using the KSADS-PL, CYBOCS and clinical interviews (child psychiatrists) and the CBCL (Parent self-report). 2. ASD screened for using the SCQ and other psychiatric diagnoses screened for using CBCL-(Parent self-report) 	<ul style="list-style-type: none"> • Group SES equivalent. • Matched for age and gender • Excluded from control group if neuropsychiatric disorder or sibling already taking part in the study. 	<p>ASD symptomatology:</p> <ul style="list-style-type: none"> • SCQ-(Parent self-report) <p>Emotional and behavioural problems:</p> <ul style="list-style-type: none"> • CBCL-(Parent self-report) 	<ul style="list-style-type: none"> • ASD symptoms and traits are more common in OCD population than in normal population. • ASD and OCD may co occur in a subgroup of OCD population. • Communication difficulties and (less frequently) social difficulties are autistic symptoms that OCD paediatric patients may endorse.
Autistic traits and obsessive compulsive traits within families.					
Abramson et al. 2005	<ol style="list-style-type: none"> 1. Probands with autism-(45) 2. Parents of probands – (69) 	<ul style="list-style-type: none"> • ASD diagnosis confirmed using clinical evaluation, the ADI-R and review of medical records (lead clinicians). • Screening for neuropsychiatric disorders in parents not reported. 	<ul style="list-style-type: none"> • Probands excluded if developmental level < 18 months or IQ < 35, comorbid Tuberous Sclerosis complex, Fragile-x syndrome, Smith-Lemli-Opitz syndrome, structural brain abnormality and/or significant prenatal or perinatal events. 	<p>ASD symptomatology:</p> <ul style="list-style-type: none"> • ADI-R (lead clinician). <p>OCD symptomatology:</p> <ul style="list-style-type: none"> • Y-BOCS (self-report version). 	<p>There is convergence of OCD symptoms and insistence on sameness autistic repetitive behaviours in families.</p>

Author	Populations (N)	Diagnostic instrument (delivered by)	Group matching / Controls	Measures of symptomatology (delivered by)	Conclusion
Kloosterman Summerfeldt, Parker & Holden, 2013	<ol style="list-style-type: none"> 1. Stage 1 - Unaffected parents (957), (683 mothers; 274 fathers) from families with one or more children with a principal DSM-IV diagnosis of an ASD. 2. Stage 2 - Unaffected parents (458), (298 mothers; 160 fathers) from families with one or more children with a principal DSM-IV diagnosis of an ASD. 	Parents in stage 2 completed ADI-R to confirm diagnosis of child's ASD - (Trained interviewer).	<ul style="list-style-type: none"> • Comparison groups for stage 1 - (families with 1 child with ASD vs. >1 child with ASD) matched for gender and age. 	<p>OCD symptomatology:</p> <ul style="list-style-type: none"> • OC-TCDQ – (Parent report). <p>ASD symptomatology (stage 2 only):</p> <ul style="list-style-type: none"> • ADI-R –(Parent report). 	<ul style="list-style-type: none"> • Resistance to change in children with ASD unique predictor of incompleteness in parents • Incompleteness higher in parents with > 1 child with ASD implying heritability. • Suggests incompleteness may be an endophenotype and underlying trait for both ASD and OCD.
Autistic traits and obsessive compulsive traits within a healthy non-clinical population					
Wakabayashi, Baron-Cohen & Ashwin, 2012	<ol style="list-style-type: none"> 1. Undergraduate psychology students- (347- males=189:females=158) 	Screening of diagnostic status not reported.	Order of presentation of measures controlled for.	<p>Autism symptomatology:</p> <ul style="list-style-type: none"> • Japanese version of AQ (self-report) <p>OCD symptomatology:</p> <ul style="list-style-type: none"> • The PI (self-report) 	<ul style="list-style-type: none"> • A small overlap of autism spectrum and obsessive compulsive spectrum may exist. • Tendency towards shared executive dysfunction in two disorders.

* Studies which explored both ASD symptomatology in OCD populations and OCD symptomatology in ASD populations are included in both sections 1 and 2.

Table 3: *Description of measures used in research included in the review*

Instrument	Version	Author	Measures	Population	Characteristics	Research evidencing Reliability and Validity
<i>ASD:</i>						
The high functioning Autism Spectrum Screening Questionnaire-(ASSQ)		Ehlers, Gillberg & Wing (1999)	Symptoms characteristic of Asperger syndrome and other high-functioning ASD.	Children (ages: 6-17)	Parent/ Teacher / Lay-informant report	Ehlers et al. (1999)
The Autism Quotient-(AQ)	Original (English version)	Baron-Cohen, Wheelwright, Skinner, Martin & Clubley, (2001)	ASD traits–50 item questionnaire. A score of 32+ indicates likely clinically significant levels of autistic traits. Domains covered: social skills, communication skills, imagination, attention to detail and attention switching.	Adults	Self-Report	Baron-Cohen et al. (2001) Hoekstra, Bartels, Cath, & Boomsma (2008) Wakabayashi, Tojo, Baron-Cohen & Wheelwright, (2004)
	Dutch version					
	Japanese version					
Social Communication Questionnaire-(SCQ)		Rutter, Bailey & Lord (2003)	ASD symptomatology. Measure of symptom severity for both preschool and current period. Domains covered: social interaction, communication and repetitive/stereotyped behaviour.	Children (ages:4+)	Parent-report	Naglieri & Chambers (2009)

Instrument	Version	Author	Measures	Population	Characteristics	Research evidencing Reliability and Validity
Autism Diagnostic Interview-Revised-(ADI-R)		Lord, Rutter & LeCouteur, (1994)	ASD symptomatology. Sub-sections: social interaction, communication/language and restricted and repetitive behaviours.	Children (2+) to Adult.	Semi-Structured interview-Clinician administered	Naglieri & Chambers (2009)
The Repetitive Behaviour Questionnaire-(RBQ)		Turner, (1995)	ASD symptomatology. Severity, nature, frequency of repetitive behaviours. Scores calculated for repetitive language, sameness behaviour, repetitive movements plus total repetitive behaviour.	Children	Parent/teacher report	Leekam et al. (2007)
Yale Special Interest Survey-(YSIS)		Klin, Danovitch, Merz & Volkmar, (2007)	Restricted Interests (RI); modality in which they are expressed and measurement of the amount of time spent engaged in RI in different social domains.	Children	Parent report	Klin et al. (2007)

Instrument	Version	Author	Measures	Population	Characteristics	Research evidencing Reliability and Validity
<i>OCD:</i>						
Yale-Brown Obsessive Compulsive Scale-(YBOCS)		Goodman Price, Rasmussen & Mazure (1989);	OCD symptom severity and presence	Adults	Clinician Administered	Goodman et al. (1989)
		Steketee, Frost & Bogart (1996)			Self-report	Steketee et al. (1996)
Children's Yale-Brown Obsessive Compulsive Scale-(CYBOCS)		Scahill et al (1997)		Children	Clinician Administered	Scahill et al. (1997)
The Children's Obsessive Compulsive Inventory-(ChOCI)		Shafran et al. (2003)	Obsessive-compulsive symptoms and associated impairment.	Children and young people	Self-report	Shafran et al. (2003)
Child Obsessive Compulsive Impact Scale-(COIS)		Piacentini & Jaffer (1999)	Obsessive-compulsive disorder related impairment across settings e.g. school, home	Children	Self-report	Piacentini, Bergman, Keller, & McCracken, (2003)

Instrument	Version	Author	Measures	Population	Characteristics	Research evidencing Reliability and Validity
Obsessive-compulsive trait core dimension questionnaire-(OC-TCDQ)		Summerfeldt, Kloosterman, Parker, Antony, & Swinson, 2001	Obsessive-compulsive traits-two dimensions; harm avoidance and incompleteness.	Adult	Self-report	Summerfeldt, Kloosterman, Parker, Antony, & Swinson, 2001
Padua Inventory-(PI)	Japanese version	Sanavio, 1988	Degree of obsessive-compulsive traits in four main areas: 'contamination fears', 'checking', 'impaired control over mental activities', and 'impulsiveness'	Adults	Self-report	Wakabayashi & Aobayashi (2007)
<i>OTHER:</i>						
Strengths and Difficulties Questionnaire (SDQ)		Goodman (1997)	Behavioural screening questionnaire: emotional and behavioural difficulties	Children (ages: 3-16)	Self/parent/ teacher report	Goodman (2001)
Kiddie Schedule for Affective disorders and Schizophrenia – Present and Lifetime version (KSADS-PL)		Kaufman et al. (1997)	Current and past episodes of psychopathology	Children	Semi-structured diagnostic interview– Clinician administered	Kaufman et al. (1997)
Child Behaviour Checklist-(CBCL)	Swedish version	Achenbach (1991); Larsson & Frisk(1999)	Presence and extent of emotional and behavioural problems	Children	Parent self-report	Larsson & Frisk (1999)

Obsessive compulsive symptoms and traits in populations with autism spectrum disorders

Obsessive compulsive (OC) symptoms and traits in individuals with ASD were measured in ten of the sixteen studies (Anagnostou et al. 2011; Cath, Ran, Smit, van Balkom & Comijs, 2008; Dewrang & Dahlgren Sandberg, 2011; Lewin, Wood, Gunderson, Murphy & Storch, 2011; Mack et al. 2010; McDougle et al. 1995; Russell, Mataix-Cols, Anson & Murphy, 2005; Ruta, Mugno, D'Arrigo, Vitiello & Mazzone, 2010; Spiker, Lin, Van Dyke & Wood, 2012; Zandt, Prior & Kyrios, 2007). All ten studies used a clinician-administered version of the Yale-Brown Obsessive Compulsive Scale (YBOCS) (in adult populations) or CYBOCS (in child populations) to measure the presence of OC symptoms, the severity of these symptoms and the type of these symptoms endorsed.

Four of these studies (McDougle et al. 1995; Russell et al. 2005; Ruta et al. 2010; Zandt et al. 2007) compared symptomatology in ASD and OCD clinical populations. One study completed a factor analysis of YBOCS scores in an ASD population (Anagnostou et al. 2011). One study (Dewrang & Dahlgren Sandberg, 2011) compared symptomatology in those with Asperger's disorder with healthy controls. One study explored the relationship between OCD traits and autistic traits in a population with comorbid ASD and an anxiety disorder (including OCD) (Spiker et al. 2012), whilst three studies (Cath et al. 2008; Lewin et al. 2011; Mack et al. 2010) compared the symptomatology of those with OCD only with those with comorbid OCD and ASD (OCD+ASD).

The earliest of these studies, completed by McDougle et al. (1995), compared OC symptomatology of adults with ASD with that of adults with OCD revealing significant differences in the types of obsessions and compulsions endorsed.

Specifically the ASD group were found to be less likely to experience aggressive, contamination, sexual, religious, symmetry or somatic obsessions and cleaning, checking or counting compulsions but more likely to experience repetitive ordering, hoarding, touching and self-damaging compulsions. The authors concluded that the symptomatology of adults with OCD and adults with ASD in terms of the types of repetitive behaviours and thoughts experienced are different. Groups were matched for age and gender and the sample size of each group was sufficient to afford appropriate power to detect medium group differences. However, the validity of results is questionable because it is likely that the IQ of the ASD group was significantly lower than the OCD group, (the mean IQ in the ASD group was 69.7 placing it in a 'mild retardation' range; in the OCD group, individuals with mental retardation or borderline intellectual functioning were excluded). Low IQ may affect an individual's ability to perceive and communicate their experiences of obsessions and compulsions meaning that the YBOCS may not be able to capture their true symptomatology. This may explain some of the significant differences in symptom type found.

This methodological limitation was addressed in Russell et al.'s (2005) study which again recruited adults with ASD and adults with OCD for comparison. IQ was explicitly measured in the ASD group to ensure exclusion of those individuals with an IQ < 70. Researchers also ensured each participant's understanding of 'obsessions' and 'compulsions' (defined as causing some degree of discomfort or interfering with individual's daily life), as distinct from repetitive behaviours characteristic of ASD, before administering the YBOCS. It should be noted that making this distinction based on the ego-dystonia of symptoms is debatable as discussed later in the review.

Findings showed that obsessions and compulsions were common (50% of ASD group) and as distressing and time-consuming as those found in the OCD population. Types of obsessions and compulsions endorsed by the two groups were similar with the exception that somatic obsessions and checking and repeating compulsions were more common in the OCD group. There are some limitations to this study. Comorbidities were reported to be high in the ASD group and analysis of the impact of this on symptomatology was not explicitly reported. The OCD group was significantly older than the ASD group and the authors recognised the impact that this discrepancy may have on the validity of the results. Despite these limitations the study raises the possibility that a significant group of individuals with ASD may suffer from OCD symptomatology; whether this represents comorbidity between disorders or an overlap of core symptomatology is not clear.

Zandt et al.'s (2007) study, which compared symptomatology in children with ASD, with OCD and typically developing (TD) children, measured the IQ of all participants and found no significant difference between groups, thus controlling for the key limitation of McDougle et al.'s (1995) study. Comparison of OCD symptomatology revealed significantly more obsessions and compulsions in the OCD group than in the ASD group who in turn reported significantly more than the TD group. Differences in the types of obsessions and compulsions reported by each group were also identified with the OCD group being more likely to endorse all obsessions except those of a religious theme (comparable endorsement in the ASD group) and miscellaneous obsessions (unspecified). The OCD group was also more likely to endorse compulsions of all types except those involving another person and ordering compulsions where group endorsement was comparable. Unfortunately these group differences are documented only as raw percentages and statistical

analyses have not been reported so that their significance is unclear. The authors concluded that the OC behaviour differs between the two groups in that it is less frequent and less sophisticated in the ASD group. This conclusion is consistent with the results of McDougle et al.'s (1995) study. However, there were significant gender differences between groups with more boys in the ASD group; this may have impacted on the expression and type of symptomatology reported by each group and as such on the validity of results. Also the existence of comorbidities in the groups could have been better screened for using formal diagnostic tools.

The Ruta et al.'s (2010) study, which also compared OCD symptomatology in children with ASD, children with OCD and TD children, addressed the key methodological weaknesses of the three studies discussed. Specifically, groups were equivalent in age, gender and IQ and both confirmation of diagnosis and screening for comorbidities were completed using formal diagnostic tools.

Consistent with Zandt et al.'s (2007) results, the authors found that the severity of OCD symptoms in children with OCD was significantly higher than in children with ASD, which in turn was significantly higher than in the TD group. Differences in types of obsessions and compulsions were noted. The OCD group had higher levels of contamination and aggressive obsessions and checking compulsions. The authors noted that there were no significant differences between the groups' insight into the excessiveness/senselessness of OC beliefs. They suggest that symptom ego-dystonia may not discriminate between the two groups.

The authors concluded that, rather than representing comorbidity, the subclinical levels of OCD symptomatology in the ASD group may support the theory that there is a continuum of symptoms across the two disorders and an overlap

between them. This study is potentially the most methodologically robust of the four discussed but the validity and generalisability of the results are impacted by the small sample size, which may have impacted the power of the analyses to detect differences present.

Anagnostou et al.'s (2011) factor analysis of YBOCS scores in young people with ASD aimed to identify categories of obsessive and compulsive behaviours in autism. The four-factor model of behaviours identified within the ASD group differed from models that had been derived from groups of individuals with OCD in previous research. Specifically, the ASD model consisted of a pure obsession factor. This differed from the majority of OCD models where obsessions and compulsions were disaggregated. The implication is that the pattern and frequency of OC behaviours are substantially different between ASD and OCD groups. This is largely in accordance with the results of the studies discussed above. However, these results are unreliable as this study did not use a well-matched OCD comparison group but relied on the results of previous research meaning that differences in models of OC behaviours could be explained by factors other than disorder, such as IQ or age.

The findings of Dewrang and Dahlgren Sandberg's (2011) study contradict the findings of previous studies discussed. Unlike the other studies, the comparison of OC symptoms using the CYBOCS revealed no elevated OC symptomatology in the ASD group when compared to a TD group. The results of the Child Obsessive Compulsive Impact Scale (COIS) demonstrated that parental and self-report ratings of psychosocial impairment due to OC features were significantly higher in the ASD group across settings. The authors concluded that OC symptomatology is not experienced at a clinically significant level in the ASD group but that there may be sub-clinical levels of OC behaviours and related psychosocial impairment in this

group, which the CYBOCS was unable to identify. However, the conclusions of this study are unreliable as the small sample size of the TD group (N=14) will have impacted the ability of the study to identify significant differences present and is likely to explain the inconsistent CYBOCS results. The study does not report that the TD group were screened for clinical presentations and thus the absence of neurodevelopmental or psychiatric difficulties in this group is unconfirmed. Given the small sample size of this group, the impact of even a small number having a neuro-psychiatric condition could affect the validity of the results.

Two studies compared OCD symptomatology in children with comorbid OCD and ASD with that in those with OCD only (Lewin et al. 2011; Mack et al. 2010). Mack et al. (2010) found no significant difference in the frequency and types of obsessions and compulsions between groups, except for a non-significant trend towards fewer somatic obsessions in the OCD+ASD group. The authors argued that the similarity in OC symptomatology between the two groups may point towards a phenomenological overlap between distressing OCD compulsions and repetitive behaviours characteristic of autism and that this may represent an area of genuine shared symptomatology. Unfortunately IQ was not measured in all participants and the very small sample sizes may have resulted in the analyses being underpowered which could have caused the lack of difference in OCD symptoms between the two groups.

Lewin et al. (2011) used well-matched groups of adequate size to power analyses in their study and included clinician administered measures to confirm diagnoses and explore symptomatology. Similarly to Mack et al. (2010), no significant difference in OC symptom severity was found between the pure OCD group and OCD+ASD group. However, on exploration of OC type, findings showed

that the OCD+ASD group were significantly less likely to experience sexual obsessions and/or checking, washing, cleaning or repeating compulsions than the pure OCD group. The authors noted that the OC behaviours in young people with OCD+ASD do not exclusively resemble autism-like repetitive behaviours and occur with equal frequency to those with OCD only. They suggested that the reduced likelihood of experiencing some of the more classic OC symptoms in those with comorbid ASD might be explained by a phenotypical alteration of OCD in ASD, characterised by fewer fear-evoking obsessions.

Cath et al. (2008) also compared the symptomatology of those with a diagnosis of OCD and those with a diagnosis of OCD+ASD in their study but in an adult population. Consistent with Mack et al. (2010) but in contrast to Lewin et al. (2011), differences in symptom type between the clinical groups were not reported. However, in accordance with both studies, OC symptom severity in the pure OCD group and the OCD+ASD group was equivalent. In contrast to Lewin et al.'s (2011) study, sample sizes in Cath et al.'s (2008) study were small and it is likely that analyses were not adequately powered to detect group differences present, which may explain the inconsistent results.

Cath et al. (2008) also focussed on ego-dystonia of symptoms finding no between group differences. This compliments the findings of Ivarsson and Melin (2008) (see below) and Ruta et al. (2010) who proposed, following similar results, that ego-dystonia may not be an appropriate means of accurately discriminating between ASD and OCD symptomatology. Cath et al. (2008) argued that previous research, suggesting that repetitive behaviours in ASD are more ego-syntonic, results from the low cognitive ability of participants and thus their inability to report adequately distress experienced in relation to these behaviours.

A fourth study (Spiker et al. 2012) investigated the relationship between autistic like repetitive interests (RI) (as measured by the Yale Special Interest Survey (YSIS)) and OCD symptoms in children with HFA and a comorbid anxiety disorder (which for an unspecified number was OCD). Symbolically enacted RIs (that is the enactment or emulation of characters or objects related to an RI) were found to be significantly related to more obsessions and compulsions. The authors concluded that either symbolically enacted RIs are a coping mechanism within ASD for OCs underpinned by anxiety, or that behavioural manifestations of symbolically enacted RIs and OCD symptoms are so alike that they are misinterpreted as each other due to measurement error. The lack of specificity regarding comorbid diagnosis renders these interpretations in relation to overlap of OC and ASD symptoms unreliable as the impact of specific comorbidity other than OCD on symptom presentation has not been accounted for.

The conclusions of these four studies in relation to shared symptomatology of ASD and OCD are limited by the exclusion of a pure ASD group for comparison and so a lack of control for the influence of comorbidity on symptom expression in the two disorders. At best it is possible that these studies identify whether the presentation of OCD when comorbid with ASD differs significantly from the presentation of OCD without comorbidity.

In summary, the results of the ten studies are variable but overall support the apparent existence of elevated symptoms/traits of OCD in ASD. Four studies (Cath et al, 2008; Lewin et al. 2011; Mack et al. 2010; Russell et al. 2005) concluded that obsessive and compulsive symptoms are as common in a population with ASD and as impairing and distressing as in a population with OCD. However, three of these four studies (Cath et al, 2008; Lewin et al. 2011; Mack et al. 2010) involved

comparison of OCD with comorbid OCD+ASD and as such the finding that OC symptom severity is comparable between groups is unsurprising. Two studies (Ruta et al. 2010; Zandt et al. 2007) argued that obsessive and compulsive symptoms are elevated in ASD when compared to healthy controls but not as common or severe as those found in OCD. However, one study (Dewrang & Dahlgren Sandberg, 2011) concluded that there was no evidence that OCD symptomatology, as described in the DSM-IV, was present in a population with ASD. Those studies reporting on the differences in types of OC symptom endorsed by the two clinical groups without comorbidity overwhelmingly support the existence of some differences, although reports of where these differences lie are less consistent. Two of the three studies comparing OC symptomatology in OCD with that in comorbid OCD+ASD found no difference in types of symptom endorsed, although both studies were underpowered which could explain the null results.

Autistic symptoms and traits in populations with obsessive compulsive disorder

Autistic symptoms and traits in individuals with OCD were investigated in five of the sixteen studies (Anholt et al. 2010; Cath et al. 2008; Ivarsson & Melin, 2008; Weidle, Melin, Drotz, Jozefiak & Ivarsson, 2012; Zandt et al. 2007). The five studies each used different measures of ASD symptomatology and traits. All five studies identified that ASD symptomatology and traits were more common in OCD populations than would be expected in the general population. Two studies explored ASD traits and symptomatology in adults with a diagnosis of OCD (Anholt et al. 2010; Cath et al. 2008) using the Autism Quotient (AQ).

Cath et al.'s (2008) study, which compared both ASD and OCD traits in adults with OCD (N=12) and adults with OCD+ASD (N=6), included only very

small sample sizes in each group meaning that null results need to be interpreted cautiously with the impact of limited power in mind. Analysis of the results of the AQ revealed that, although the comorbid group scored significantly higher than the OCD group on the AQ subscale ‘attention shifting’, the OCD group in turn scored significantly higher than the control group on this domain. No significant differences were found between the OCD group and comorbid ASD group on AQ subscales ‘attention to detail’ and ‘social skills’. The authors suggested these results support the notion of genuine symptom overlap between the two disorders noting that, in addition to the repetitive behaviours characteristic of both disorders, OCD and ASD may also share difficulties in social skills. In addition the authors noted that deficits in attention to detail reported in both groups may be underpinned by similar deficits in executive function.

Correlational analyses exploring whole study group results (N=36) to determine any relationship between the measures used found positive correlations between AQ total scores and all AQ subscales (except social skills and imagination subscales) with the YBOCS severity scores. The authors noted that the validity of these correlations, implying real relationships between separate diagnostic constructs, is compromised by possible measurement error; that is, where the measures lack specificity for their intended diagnostic constructs.

Anholt et al. (2010) compared ASD symptomatology in adults with OCD with that in healthy adults. This study included large sample sizes and groups matched for demographic factors. The results showed that the OCD group scored significantly higher on the AQ than did the healthy adults, indicating elevated levels of ASD traits. In correlational analyses numerous relationships were found between ASD traits and OC symptomatology. There was an overall positive correlation

between AQ total scores and YBOCS severity scores. Specifically, the AQ subscales, attention shifting and communication, were significant predictors of OCD symptom severity whereas attention to detail demonstrated low correlation with OCD symptoms and severity. In the prediction of specific OC symptoms the AQ subscales, attention switching and communication, were the most important predictors of OC ‘aggression and checking’, ‘symmetry and ordering’ and ‘contamination and washing’ symptoms. AQ scores did not predict OC hoarding symptoms. Similarly to Cath et al. (2008), the authors interpreted these results as indicative of the substantial overlap between ASD and OCD symptomatology, which they suggested, may be explained by overlapping aetiologies. They also proposed that the importance of the AQ subscale ‘attention switching’ in predicting OC symptomatology may indicate shared executive dysfunction between the disorders.

The remaining three studies in this section focussed on child populations. Ivarsson and Melin (2008) explored autistic traits in a paediatric population with OCD using the high functioning Autism Spectrum Screening Questionnaire (ASSQ). Individuals with comorbid anxiety, depression, ADHD, tic disorder or ASD were included in order to explore the impact of these comorbidities on the expression of ASD traits in OCD. The findings of this study identified significant relationships between ASD traits and the presence of a comorbid tic disorder, ADHD or any ASD. These variables were identified as having the capacity to explain approximately 40% of the variance in ASD symptomatology in those with OCD. The authors purported that this may support the notion that a large proportion (60%) of variance in the expression of ASD traits in OCD cannot be explained by comorbidities and as such they suggested that OCD itself may be associated with some lower level ASD traits.

In contrast to the findings of Anholt et al. (2010), no positive relationship was found between ASD traits and OCD severity. In addition, no relationship was found between level of insight into rationality of OCD symptoms and ASD symptomatology. This brings into question the possibility that discrimination of OCD and ASD symptomatology can be based on symptom ego-dystonia. The authors concluded that the relationship between ASD traits and OCD may be independent of comorbidity, adding weight to the possibility that there may be a subsample of individuals with a diagnosis of OCD with genuine ASD traits.

Weidle et al. (2012) explored differences in ASD traits between children with OCD and TD children using the Social Communication Questionnaire (SCQ), finding that total scores, preschool and current symptom scores were significantly higher in the OCD group indicating higher rates of autistic symptomatology. However the authors noted that not all children with OCD demonstrated ASD traits and therefore suggested that OCD and ASD may co-occur in only a subgroup of the OCD population.

Items of the SCQ relating to preschool symptoms that were found to be significantly more endorsed in the OCD group included failure to use gestures and poor quality of social overtures. The authors suggested that for some children with OCD, communication difficulties and (less frequently) social difficulties are the ASD symptoms most likely to be endorsed. The validity of these reported preschool symptoms was dependent on the accurate memory of parents. The authors noted that to increase the validity of results in future research a longitudinal research design should be employed.

In terms of current symptomatology significantly higher endorsement of verbal rituals, compulsions, hand and finger mannerisms and unusual sensory interests were reported in the OCD group when compared to TD children. The authors acknowledged that not all SCQ items are specific to ASD and that many of these significant results could reflect the measures' inability to discriminate between ASD specific and OCD specific symptomatology making it more difficult to draw conclusions.

Comparison of group levels of emotional and behavioural problems using the Child Behaviour Checklist (CBCL) revealed significantly more difficulties in the OCD group and significantly lower total social competence scores (e.g. more likely to have fewer friends).

The study also explored whether ASD traits identified in OCD can be explained independently of other psychiatric disorders, finding a significant relationship, independent of CBCL score, between group membership and SCQ score. However, the OCD group included a high number of participants with comorbid neurodevelopmental disorders (Tourette's-16.2% and ADHD-19.2%) which are not necessarily accounted for by controlling for the CBCL score and may impact the conclusions that can be drawn in determining the independent relationship between ASD traits and OCD. Other weaknesses which impact the validity of the findings include a lack of control for IQ differences between groups.

In the four studies noted above it would have been beneficial to have included a comparison group of individuals with a diagnosis of autism such that the symptomatology of ASD in OCD and in ASD could have been directly compared.

Zandt et al. (2007) is the only study included in this section that directly compared ASD symptomatology in children with ASD, children with OCD and TD children. The study employed the Repetitive Behaviour Questionnaire (RBQ) to explore ASD type repetitive behaviours in each group, finding no significant differences between clinical groups in total repetitive behaviour, sameness behaviour or repetitive movements, although both groups demonstrated significantly higher scores in each of these domains than did the TD group. Repetitive language featured comparably at a very low level in both clinical groups but did not feature in the TD group. The authors tentatively concluded that there are some similarities in symptomatology between the two groups in terms of levels of repetitive behaviours. The conclusion is rightly tentative given the small sample sizes included in this study, which would have impacted the ability of the analyses to detect significant differences between group symptomatology, if present.

It seems all these studies imply that there are heightened levels of ASD traits in at least some individuals with OCD which is beyond that which can be explained by comorbidity alone. Whether or not this represents genuine or superficial overlap in symptomatology is difficult to unpick given the apparent inability of measures used to identify clearly diagnostic specific constructs.

Autistic traits and OC traits within families

ASD and OCD symptomatology and traits within families were investigated in two of the sixteen studies (Abramson et al., 2005; Kloosterman et al. 2013). Abramson et al. (2005) explored OCD and ASD symptomatology in 45 families with one child with autism. OC symptomatology in parents was measured using the YBOCS finding that 33% of the parents included had clinically significant scores.

Autistic repetitive behaviours were measured in the children with ASD using the restrictive and repetitive behaviour domain of the Autism Diagnostic Interview-Revised (ADI-R) and principal component analysis revealed two main factors within this domain, insistence on sameness and repetitive motor and sensory phenomena. Correlational analysis between parental YBOCS scores and children's autistic repetitive behaviours identified positive relationships between child 'insistence on sameness scores' and parental YBOCS scores. No relationships were identified between the children's overall score on the restrictive and repetitive behaviour domain or on the 'repetitive motor and sensory phenomena' subsection scores and the parental YBOCS scores. The authors proposed that the results indicating convergence of OCD symptoms and insistence on sameness autistic repetitive behaviours in families might be explained by an overlap of OCD and ASD phenomenology, in that there may be a continuum of repetitive behaviours in autism which includes OC features. Kloosterman et al. (2013), who investigated relationships between the OCD trait of incompleteness (using the obsessive-compulsive trait core dimension questionnaire (OC-TCDQ)) in parents of children with ASD and their children's autistic repetitive behaviours, using the ADI-R, revealed similar results. Specifically, resistance to change in children was found to be a unique predictor of sense of incompleteness in their parents. In addition, repetitive sensory motor actions in the children were significantly associated with parental levels of incompleteness. Sense of incompleteness was also higher in parents with more than one child with ASD which the authors argued implies heritability and suggested incompleteness may be an endophenotype and underlying trait for both ASD and OCD. Unfortunately limitations in both studies limit the reliability of these results. Neuropsychiatric disorders in the parent groups were not

screened for and so the potential impact of additional comorbidities on YBOCS/OC-TCDQ scores cannot be ruled out. In addition, the potential influence of parental modelling in determining the nature of the repetitive behaviours seen in their children with autism was not accounted for in either study and may offer an alternative explanation for the positive correlation identified between autistic restrictive and repetitive behaviours and YBOCS/ OC-TCDQ scores.

Autistic traits and OC traits within a healthy non-clinical population

One study investigated autistic and OC traits in a healthy non-clinical population (Wakabayashi et al. 2012). Wakabayashi et al. (2012) examined whether traits of OCD and ASD overlapped in a non-clinical population based on the analogue assumption that these clinical disorders represent the extreme end of a normal distribution whereby clinical symptoms differ from those found in the typical population only in their severity and frequency. A large number of undergraduate students (N=347) were recruited resulting in appropriate power for analyses. Participants completed two self-report measures, the AQ (Japanese version) to measure ASD traits and the Padua Inventory (PI) to measure OCD traits. Moderate positive correlations were found between total PI and total AQ scores. Additionally PI subsection 'impaired control of mental activities' was positively correlated with total AQ score whilst the other PI subsections showed weak correlations with total AQ score. 16% of the variance in AQ score could be explained by two PI factors, impaired control of mental activities and impulsiveness. The authors compared individuals who scored above the AQ cut off for probable ASD diagnosis with total group scores on the PI, finding that the high scoring AQ group scored significantly higher than the total group on total PI score and on the impaired control of mental activities and impulsiveness subsections. They concluded that a relationship exists

between traits of OCD and ASD but that this relationship is only partial, proposing that a partial symptom overlap may be explained by a tendency towards an executive dysfunction characterised by impaired control of mental activity. They suggested that the differences between disorders' symptomatology may be in part evidenced by the finding that repeating/checking rituals were no more common in those with high AQ scores, perhaps implying that these OC symptoms are not shared by or part of the symptomatology of those with ASD. Although these results may add weight to the research in clinical populations that found some symptom overlap and argue for a broader phenomenological description of the disorders (e.g. Cath et al. 2008), the use of a non-clinical population will impact the generalisability of the results and its applicability and comparability to the symptomatology of those populations with clinically significant OCD or ASD.

Discussion

Main findings

The 16 studies in this review employed a range of measures with proven psychometric value to explore the overlap in the symptomatology of OCD and ASD. Strengths of the studies as a whole included the consistent use of measures of symptomatology with proven psychometric value and generally high standards of reporting. Overall the studies provide consistent evidence for the apparent existence of at least sub-clinical levels of ASD symptomatology and traits in a proportion of those with OCD and vice versa, with variable results as to which traits and symptoms this applies.

In summary, exploration of similarities and differences in OC symptom types endorsed by the two disorders revealed some tentative evidence that the expression

of OC symptoms and traits in an ASD population is largely different from that expressed in an OCD population. The different studies consider where these differences lie, the most consistent finding being that those with OCD were more likely to experience checking compulsions and somatic obsessions. Fewer studies in this review explored ASD symptoms in an OCD population so conclusions are harder to draw. However, there is some preliminary evidence for deficits in social skills and communication in OCD populations as well as evidence for the existence of autistic-like repetitive behaviours. None of the studies found evidence for difficulties with language or imagination in an OCD population.

With regard to this apparent overlap of symptomatology or traits in ASD and OCD, three broad explanatory models could be applied. The first explanatory model is that the identified apparent elevated traits of one disorder in the other represent genuine symptom overlap with shared aetiology such as a common genetic vulnerability or neurocognitive deficit. This potential model and the possibility that ASD and OCD share genetic vulnerabilities is supported by research which indicates that the occurrence of OCD within families can predict a genetic vulnerability for autism (Fischer-Terworth & Probst, 2009). For example, molecular genetic studies indicate a possible genetic link between OCD and ASD (e.g. Hollander et al, 1999) and Hollander, King, Delaney, Smith and Silverman (2003) found that repetitive behaviours in children with autism are frequently positively correlated with obsessive compulsive behaviours in parents. Additional family studies have implicated mutations of the serotonin transporter genes in both disorders (Fischer-Terworth & Probst, 2009).

In addition, research exploring neurocognitive similarities between disorders has revealed possible shared executive deficits, which may underpin symptoms of

the two disorders. For example, Delorme et al. (2007) found the existence of executive dysfunction in the unaffected first-degree relatives of probands with OCD, which was similar to that observed in the relatives of patients with autism.

The studies included in the current review do not provide sufficient evidence to support or refute this first explanatory model. However, those studies which investigated relationships between specific traits of the two disorders provide some emerging evidence for similarities, especially regarding those traits, which might be explained by executive dysfunction. Specifically, measures of autistic-like inattention were consistently found to be positively correlated with OCD symptomatology. The inclusion of studies investigating commonalities in neurocognitive profiles of the two disorders was beyond the scope of this review but these results indicate that this might be a key area for future research and could potentially identify more accurately any overlap in symptomatology.

In order to support the possibility of genuine symptom overlap it would also be interesting to investigate the similarities and differences in symptomatology of the two disorders over time, employing longitudinal designs, particularly as ASD is considered a life-long neurodevelopmental disorder where symptomatology is present from a young age, which is in contrast to the current conceptualisation of OCD and its symptomatology.

A second potential explanatory model is that the symptoms of ASD and OCD are fundamentally distinct, but that they are sometimes observed in the same individual due to comorbidity, where two or more discrete disorders present simultaneously in an individual. Within this framework, the fact that ASD and OCD co-occur more often than would be expected by chance could arise because ASD is a

risk factor for the development of OCD and as such explains elevated traits of OCD in an ASD population. It should be noted that this explanation is much less convincing when applied to OCD as a risk factor for the development of ASD given that ASD is a neurodevelopmental disorder which by definition arises in the first years of life. Alternatively it could be that both disorders share a common risk factor making their co-occurrence more likely.

If comorbidity were responsible for the apparent existence of elevated symptoms of OCD in ASD and vice versa then it would be reasonable to expect that this comorbidity would be identifiable by the existence of the complete range of symptoms of both disorders co-occurring. That is, the presentation of OCD comorbid to ASD should not differ substantially from OCD without comorbidity (and vice versa). Two of the three studies reviewed, that compared OCD symptomatology in OCD+ASD with OCD without comorbidity, found no significant differences in symptomatology between the two groups and as such support the comorbidity argument. However, both these studies were underpowered which undermines the validity of their null findings. In addition, the third study within this category, which was adequately powered, did find significant differences in symptom types between the two groups.

The comorbidity argument is also insufficient when considering those studies that compared ASD and OCD (without comorbidity) which consistently found the existence of some but not all traits/symptoms of one disorder in the other and vice versa. Such findings could be understood in light of the theory of ASD symptom fractionation (Happé, Ronald & Plomin, 2006). This suggests that the core symptoms of ASD, repetitive and restrictive behaviours and social and communication deficits, cannot be explained by a single cause at genetic, neural or

cognitive level but instead that the various deficits may be explained by separate or ‘fractioned’ causal factors (Brunsdon & Happé, 2014). The implication of this theory is that some elements (but not necessarily all) of ASD symptomatology may have a shared aetiology with symptoms seen in other disorders such as OCD.

In addition, Ivarsson and Melin’s (2008) finding that 60% of variance in ASD symptomatology in those with OCD could not be explained by comorbidities (including ASD) undermines the adequacy of this comorbidity argument. Comorbidity as an explanatory model for the existence of elevated traits across disorders is therefore not adequately supported by the studies included in this review.

The third explanatory model is that the disorders may not be truly related and the apparent overlap of symptomatology identified in the reviewed studies is in fact superficial and a consequence of epiphenomenon and/or measurement error. Epiphenomenon is where symptom(s) develop incidentally during the course of a disorder but are unconnected to that disorder; for example, it could be hypothesised that social impairments identified in some individuals with OCD are a consequence of core symptoms of the disorder (e.g. compulsions) rather than representing a core OCD symptom and an area of real symptom overlap with ASD. Measurement error occurs when the measures of symptomatology employed are incorrectly identifying the symptom in one disorder as the same as another symptom in the other, rather than picking up diagnostic specific constructs. For example, repetitive behaviours are reported as present in both OCD and ASD in most of the studies but convincing discrimination between that which is OCD-specific rather than ASD-specific has proven notoriously difficult (Paula-Perez, 2013). The key difficulty with making discriminations of symptomatology in these two clinical groups is this possibility of measurement error (Cath et al. 2008; Spiker et al. 2012).

The ability of the studies included in this review to reach convincing conclusions in relation to the nature of the relationship between ASD and OCD symptomatology is also impacted by the lack of evidence that attempts were made to support participants' and researchers' understanding and ability to discriminate between OC behaviours and repetitive behaviours characteristic of autism. Where such discrimination has been attempted (e.g. Russell et al. 2005) it has been based on the premise of symptom ego-dystonia.

Ego-dystonia of symptoms is purported by many as a means of discriminating between similar ASD and OCD symptomatology (Mack et al. 2010; Paula-Perez, 2013) and in particular in relation to discriminating similar repetitive and compulsive behaviours, with the assumption that these symptoms would be more ego-dystonic and thus distressing in those with OCD. This potentially helpful basis for discrimination has been addressed in three of the studies included in this review which specifically investigated the ego-dystonia of symptoms in the two groups, revealing no significant differences. This counters the commonly held view that repetitive behaviours often seen in ASD are less distressing than similar repetitive behaviours seen in OCD and the quality and experience of these behaviours may be more similar than previously thought. The finding that the quality of repetitive behaviours in the two disorders may be more similar than previously assumed may support the first explanatory model that the identified apparent elevated traits of one disorder in the other represent genuine symptom overlap.

Methodological issues and research implications

The inconclusive findings of the studies included in this review highlight that further investigation into the overlap of symptomatology in OCD and ASD is

warranted to clarify the nature of the similarity of symptomatology in OCD and ASD and address some of the common methodological difficulties encountered by the research to date, allowing for more robust conclusions to be formed.

In particular, it may be helpful for future research to include more clinician rated measures of symptomatology, which are based on observation of behaviours, as well as participant reports of symptom experience. This methodology may be better able to measure objectively and pick up more subtle qualitative aspects of symptomatology, avoid measurement error and aid ASD and OCD symptom discrimination. This is particularly relevant in relation to research investigating ASD symptomatology in OCD. Whilst the studies investigating OC symptomatology in ASD included in this review consistently employed the clinician administered YBOCS, all five studies investigating ASD symptomatology in OCD relied on self or parent report measures. It would be advisable to employ measures which are not reliant on self-report, such as the ADOS (Lord et al. 2000), in future research aiming to determine the presence and validity of autistic traits in OCD. In addition, further research that employs neurocognitive and genetic approaches that seek to identify common underpinnings for OCD and ASD symptomatology may provide greater insight into symptom overlap.

Additional methodological difficulties encountered by studies included in this review were insufficient sample size for statistical analyses (Cath, et al. 2008; Dewrang & Dahlgren Sandberg, 2011; Mack et al. 2010; McDougle et al. 1995; Ruta, et al. 2010; Zandt et al. 2007) and/or a lack of appropriate matching of comparison groups (Anagnostou et al. 2011; McDougle et al. 1995; Russell et al. 2005; Weidle et al. 2012; Zandt et al. 2007). This inevitably will have impacted on the validity of the results of the affected studies as referenced throughout the body of

the review. Clearly future research would benefit from including comparison groups, carefully matched for age, gender and IQ as well as adequate sample sizes to enable analyses to have sufficient power to identify differences or relationships of interest.

If the evidence-base expands in support of genuine symptom overlap between OCD and ASD it might be helpful to introduce research into the efficacy of intervention packages adapted for those individuals who experience this more shared presentation in symptomatology.

Clinical implications

The findings suggest that discrimination between the symptomatology of ASD and OCD is a complex task and as such more careful consideration is needed when making diagnoses and administering appropriate intervention packages for these populations. This is particularly important given the apparent difficulty of many of the measures commonly employed clinically in discriminating between the two disorders' symptoms and traits. Whether such discrimination is possible is open to debate with previously considered distinguishing features such as symptom ego-dystonia proving to be an unreliable dissimilarity.

The mixed results raise the possibility that the apparent overlap of symptomatology may apply to a subgroup of individuals rather than to all those with an OCD or ASD diagnosis. It may be that certain features (e.g. social skill deficits in OCD) highlight those to whom this overlap is likely to be applicable and identification of these features might support clinicians in the development of more appropriate treatment. Clearly this is an area that warrants more research before conclusions can be drawn but clinician awareness of the possibility of overlap is important.

This review highlights the frequency that symptoms of repetitive behaviours cause distress in individuals with ASD. This has been particularly apparent in the studies which included individuals with a diagnosis of ASD without intellectual disability. These individuals were able to understand questions in relation to and communicate their feelings of distress. The implication is that clinicians should take time to support adequately individuals with ASD, with and without intellectual disability, to express distress experienced in relation to their repetitive behaviour and should not assume symptoms are ego-syntonic. It may be that interventions employed to support individuals with OCD can be adapted to support those individuals with ASD who experience distress in relation to repetitive behaviours.

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Part 2: Empirical Paper

Autistic traits and cognition in individuals with obsessive compulsive disorder.

Abstract

Aims

This study investigated whether neurocognitive performances characteristic of ASD co-varied with higher levels of self-reported autistic traits in adults with OCD in order to determine the validity of autistic traits identified.

Method

Twenty adults with OCD completed a measure of autistic traits (the Autism Quotient (AQ)) and a battery of neurocognitive assessments specifically selected to identify cognition associated with ASD. Both group and multiple single case series designs were employed to investigate relationships between AQ scores and neurocognitive profiles.

Results

In accordance with results of previous research, adults with OCD demonstrated elevated levels of autistic traits on all elements of the AQ apart from the subscale attention to detail. However, no clear neurocognitive profile was elucidated in relation to autistic traits and multiple single case series analysis did not clearly reveal any individuals with both autistic traits and cognition who might indicate the presence of an ASD subgroup within OCD.

Conclusions

At the group level, the results present some tenuous evidence in support of atypical neurodevelopment within OCD, characterised by a detail-focused processing style. However, the validity of the autistic traits identified within this group is not supported by the results of cognitive assessments. The exploratory multiple single case series analysis suggests the value of this approach in heterogeneous groups, such as OCD populations, in future subgrouping research.

Introduction

Obsessive compulsive disorder (OCD) is a common and disabling psychiatric disorder characterised by obsessions (which cause marked anxiety or distress) and/or compulsions (which serve to neutralise distress) (American Psychiatric Association (APA), 2013). The dominant conceptualisation of OCD has recently changed. Formerly considered a unitary nosological entity (APA, 2000) and an anxiety disorder, OCD is now viewed as a heterogeneous diagnostic entity where individuals with OCD present with disparate, non-overlapping symptom patterns. As such, OCD has been reclassified under ‘Obsessive Compulsive and Related Disorders’ in the DSM-5 (APA, 2013) rather than as an anxiety disorder.

Numerous ways of understanding the heterogeneous symptom presentation in OCD and of informing interventions have been suggested (for example, symptom categorisation (Rasmussen & Eissen, 1998)). It has been proposed, for example, that the identification of specific abnormalities in brain anatomy, chemistry, and function might represent different etiologic or genetic forms of the illness and lead to the development of new diagnostic and treatment approaches (Rosenberg & Hanna, 2000).

Some OCD symptomatology bears a striking resemblance to that of the neurodevelopmental disorder, autistic spectrum disorder (ASD) (Bejerot, Nylander, & Lindstrom 2001). ASD is a lifelong disorder with an estimated prevalence of 1% of the population (Baird et al. 2006), characterised by impaired communication and social interaction, repetitive behaviours and restricted interests (APA, 2013; WHO, 1992). Similarly many individuals with OCD are characterised by repetitive behaviours, ordering and symmetry compulsions (Rapoport, 1989; Rasmussen & Eisen, 1992). In addition, Ivarsson and WingeWestholm (2004) investigated the

temperamental features of children and adolescents with OCD and found that about half the sample had low levels of activity and sociability and high levels of shyness perhaps indicating impaired social interaction in some individuals similar to that seen in ASD.

Bejerot et al. (2001) noted that the negative predictors of treatment outcome of OCD, males living alone (Buchanan, Meng & Marks, 1996), difficulties with interpersonal relations (Fals-Stewart & Lucente, 1993), hoarding (Black et al. 1998), abnormal personality, social impairment, and childlessness (de Silva, Rachman & Seligman, 1977), are all characteristics common in ASD. Indeed, research indicates that whilst for some, OCD is episodic and remitting, for a substantial group of patients their illness follows a more chronic course (Venkatasubramanian, Rao & Behere, 2009). This group is characterised by individuals, more often male, with severe symptoms and early onset OCD (Venkatasubramanian et al. 2009). In addition, elevated neurological soft sign abnormalities have been identified in individuals with OCD (Jaafari et al. 2013) and children with OCD are more likely to exhibit neurological signs than adults (Geller, Biederman, Griffin, Jones & Lefkowitz, 1996). Familial OCD has also been found to be more prevalent in those with early onset OCD implicating genetic factors in this hypothesised subgroup (Pauls et al. 1995). Parallels between this OCD group and ASD have been drawn leading to the hypothesis that, at least for some individuals with OCD, a neurodevelopmental deviation rather than an acquired degenerative process contributes to the pathogenesis of the disorder (Rosenberg & Keshavan, 1998) and that this group may reflect those with ASD (Bejerot et al. 2001). Furthermore, elevated levels of self-reported ASD traits have been identified in OCD (Anholt et al. 2010) and levels of comorbidity between ASD and OCD are higher than would be

expected (25%) compared with disorder prevalence rates in the normal population (Russell, Mataix-Cols, Anson, & Murphy, 2005).

The phenomenological similarities between OCD and ASD have fostered curiosity regarding the relationship between these disorders and some research has investigated similarities in symptomatology (e.g. McDougle et al. 1995; Russell et al. 2005), biological features and genetic markers (e.g. see Jacob, Landeros-Weisenberger and Leckman (2009) for a review). However, this research has not provided clear conclusions in relation to disorder overlap. It is suggested that investigation of the cognitive profiles of people with OCD and comparison of the findings with the cognitive profile commonly found in ASD might elucidate more clearly any shared aetiology or overlap between them.

Cognitive neuropsychology of OCD

Cognitive processing deficits are frequently reported and generally accepted to exist in OCD (Tallis, 1997), although reports of where these deficits lie are highly variable (Chamberlain, Blackwell, Fineberg, Robbins & Sahakian, 2005). Theory-based studies of neurocognitive function have yet to reveal a reliable cognitive profile and interpretation has often been confounded by the influence of co-morbidities not controlled for (Chamberlain et al. 2005).

Accordingly, the relationship between neuropsychological findings and their underlying brain abnormality in OCD has not been clearly elucidated (Aycicegi, Dinn, Harris & Erkmén, 2003; Kuelz, Hohagen, & Voderholzer, 2004; Lacerda et al. 2003) but some neuroimaging studies have suggested that specific neural correlates may be associated with different symptom dimensions (Rauch & Baxter, 1998).

However, the finding that there is an association between OCD and underperformance in tasks that assess response inhibition is robust (Aycicegi et al. 2003; Chamberlain et al. 2005; Penades et al. 2007). Chamberlain et al. (2005) suggest that the perseverative thoughts and behaviours that are symptomatic of the disorder may reflect a loss of normal inhibitory processes.

Numerous studies investigating the neurocognitive profiles of individuals with OCD have identified set-shifting as a deficit (Purcell, Maruff, Kyrios & Pantelis, 1998; Veale, Sahakian, Owen & Marks, 1996). Chamberlain, Robbins and Sahakian (2007) suggest that difficulties shifting attentional focus may result in cognitive inflexibility and contribute to the generation of compulsive symptoms. Interestingly one study found that, although individuals with OCD had poorer set-shifting abilities than controls, those with symmetry/ordering symptoms demonstrated a significantly greater deficit (Lawrence et al. 2006).

There are inconsistent results as to whether individuals with OCD demonstrate impairments in memory, planning and decision making abilities (Chamberlain et al. 2005). For example, there is a debate as to whether identified memory impairment in OCD represents deficits in recall or in the employment of appropriate organisational strategies supporting recall (Chamberlain et al. 2005). Similarly, there is an argument that where deficits in planning are identified, it results from abnormal psychomotor slowing rather than a pure planning deficit (Chamberlain et al. 2005). These inconsistencies have led researchers to argue that deficits in this area may only apply to a subgroup of individuals with OCD (Chamberlain et al. 2005). In addition, mixed findings in relation to deficits in decision-making abilities have led researchers to hypothesise that such deficits may

apply only to certain forms of the disorder (e.g. treatment resistant OCD) (Chamberlain et al. 2005).

Although literature does not identify social cognition impairments in OCD, some adults with OCD have been characterised with a behaviourally inhibited temperamental style, defined as a characteristic propensity to react to both social and nonsocial novelty with inhibition (Van Ameringen, Mancini & Oakman, 1998). This temperamental style has also been found to be a childhood predictor of OCD symptoms (Muris, Meesters & Spinder, 2003).

The lack of consistency in neuropsychological findings does not afford any real certainty with regard to cognition in OCD and may reflect the heterogeneity of OCD and represent the existence of subtypes with distinct neurocognitive profiles within the disorder (Nedeljkovic et al. 2009).

Cognitive neuropsychology of ASD

There have been many group based studies researching cognition in individuals with autism, the key findings of which have implicated a number of common specific neurocognitive deficits and provided support for a possible shared pattern of cognitive strengths and weaknesses, an autistic cognitive profile (Mandy, Murin & Skuse, 2014).

One of the most consistently reported findings in studies looking at cognitive deficits in ASD is executive dysfunction (Bennetto, Pennington & Rogers, 1996; Ozonoff & Jensen, 1999; Pennington & Ozonoff, 1996; Prior & Hoffman, 1990; Rumsey & Hamburger, 1988; Russell, 1997). Executive dysfunction refers to deficits in those skills required to prepare for and execute complex behaviour (Ozonoff et al. 2004). Deficits and differences in executive function in those with autism have been

implicated particularly in the non-social aspects of ASD such as repetitive and stereotyped behaviours (Pennington & Ozonoff, 1996).

Within the construct of executive function is a relatively robust finding that set-shifting is impaired relative to age and IQ matched typically developing controls (Ozonoff et al. 2004; Pennington & Ozonoff, 1996). In addition, in ASD the repetition of certain behavioural patterns may result from an inability to generate novel behavioural patterns. Generativity is a process which requires respondents to generate spontaneously appropriate novel responses. Impaired generativity is thought to mediate deficits in pretend play commonly identified in children with ASD (Turner, 1997). Inhibition, when strictly defined (that is, teased apart from tasks demanding cognitive flexibility), appears to be intact among persons with autism with developmental levels greater than 6 years (Russo et al. 2007).

In addition to the executive dysfunction theory in autism are two other prominent theories which are proposed to explain differences in cognition and which might underpin behavioural aspects of ASD.

Impairments in theory of mind (TOM), that is the ability to mentalise or to identify and attribute mental states to others (Leslie, 1987), have been consistently demonstrated in those with autism and are theorised to underpin the difficulties in social functioning and communication which are defining features of autism (APA, 2000; Baron-Cohen & Wheelwright, 2003; Orsmond, Krauss & Seltzer, 2004).

Weak central coherence in autism is also frequently observed, that is individuals with ASD have a more detailed focussed style of processing and are thus less likely to integrate local information in the search for global meaning (Frith & Happé, 1994). Weak central coherence has been implicated in commonly identified

ASD characteristics such as insistence for sameness/routine and attention to parts of objects (Booth & Happé, 2010).

The theories and research outlined above provide information to define a possible disorder-specific cognitive profile for autism which is characterised by impairments in set-shifting, generativity and theory of mind but strengths in detail-focussed processing and inhibition (Mandy, Murin & Skuse, 2014).

Comparison of cognitive profiles of OCD and ASD

There is currently a paucity of research into the similarities and differences in the cognitive profiles of ASD and OCD. To our knowledge two studies (Delorme et al. 2007; Zandt, Prior & Kyrios, 2009) have attempted to compare the neurocognitive profiles of the two groups but clear conclusions have not been reached. Zandt et al. (2009) found that areas of cognitive impairment differed between the two disorders; specifically children with ASD showed deficits in generativity whilst those with OCD demonstrated deficits in inhibition. Zandt et al. (2009) concluded that similarities in symptoms of the disorders (specifically repetitive behaviours) might be superficial and derived from different cognitive processes. In contrast, Delorme et al. (2007) proposed that shared cognitive deficits, specifically in planning and spatial working memory, do exist, potentially representing a shared cognitive phenotype, which explains similar symptomatology of the two disorders. However the potential impact of anxiety on cognitive processes is highlighted as a confounding factor which may affect the reliability of these results.

To our knowledge no study has investigated the neurocognitive profiles within a population of adults with OCD in relation to their levels of autistic traits. If high levels of autistic traits are found to co-occur with an autistic cognitive

performance it would support the validity of autistic traits identified within an OCD population.

In addition, to date, the majority of studies investigating cognitive abilities in OCD have been designed to accommodate statistical comparison of group means. There is a risk that relying solely on this group methodology may preclude analysis of the heterogeneity evident in the cognitive abilities of individuals with OCD and obscure identification of the existence of groups of individuals within OCD with distinct patterns of cognitive deficits (Towgood, Meuwese, Gilbert, Turner & Burgess, 2009). As such, in addition to group analyses, a statistical approach (novel in its application to OCD), multiple single case series methodology, will be applied in this exploratory study using a broad range of neuropsychological assessments to explore the patterns of cognitive strengths and difficulties of individuals with OCD.

Aims and hypotheses of study

The study has three key aims:

1. To replicate previous findings of elevated levels of self-reported autistic traits in a population of individuals with OCD.
2. To investigate whether levels of autistic traits co-vary with elements of cognitive performance that are associated with ASD. Specifically, it is hypothesised that impairment in cognitive deficits common to ASD, set-shifting, theory of mind, and generativity and weak central coherence, will be positively correlated to levels of self-reported autistic traits whilst no such relationship is predicted between inhibition and self-reported autistic traits.
3. To investigate, using a multiple single case series approach, whether there are individuals with OCD who demonstrate both elevated self-reported autistic

traits and cognitive profiles associated with ASD. Specifically, we wanted to explore the possibility that these autistic traits and cognition are limited to a subgroup of individuals with OCD.

Method

Setting

The study took place at an NHS clinic specialising in OCD services.

Participants

Sample size

Intended power

A power analysis for this study was carried out using G-Power (Faul, Erdfelder, Lang & Buchner, 2007) to estimate an appropriate sample size. No comparable single samples group comparison studies or correlational studies were identified and, as such, necessary sample size to achieve adequate power for these analyses was based on an assumption of a medium effect size of $d=0.5$ (for the group comparisons) and $r=0.3$ (for the correlational analyses). Therefore, for both single samples group comparisons and correlational analyses, sample size was calculated based on an effect size of $d=0.5$ or $r=0.3$ respectively, an alpha setting at 0.05 and a power of 0.8. These calculations determined that sample sizes of $N=34$ and $N=82$ were necessary to achieve adequate power for the single samples group comparisons and correlational analyses respectively. Unfortunately, difficulties with recruitment in the current study (see participant recruitment) meant that reaching the desired sample size to achieve adequate power for group comparisons and correlational

analyses was not possible and caution will therefore be exercised in interpreting results.

Achieved power

A power analysis was also completed for this study using G-Power (Faul, et al. 2007) to determine the effect size that the actual sample was powered to detect. Based on actual sample size achieved (N=20) with alpha setting at 0.05 and a power of 0.8, the sample provided sufficient power to detect large effect size, greater or equal to $r=0.55$, for the correlational analyses and large effect size, greater or equal to $d=0.66$, for the group comparisons.

Demographics

Participants were 20 outpatients with a current diagnosis of OCD at the NHS OCD clinic where the research was being conducted. Participants were 14 females and 6 males with a mean age of 46.35 years (SD = 11.26). They were recruited following their participation in a pilot study taking place at the OCD services to determine levels of self-reported autistic traits (see procedure for detail). Participants were eligible if they were over 18 and spoke fluent English and excluded if they had current or history of head trauma or neurological impairment or learning disability. All participants who had completed participation in the pilot study and provided consent to be contacted regarding future research met inclusion criteria for the current study. Table 1 summarises the participant characteristics.

Table 1: *Demographic characteristics of study sample*

Participant characteristic	Number	Percent ^a	Mean	SD	Range
Gender:					
Male	6	30			
Female	14	70			
Age			46.35	11.26	26-62
Marital Status:					
Single	9	45			
Married	8	40			
Divorced	3	15			
Widowed	0	0			
Employment status:					
Employed	7	36.84			
Unemployed	9	47.37			
On sick leave	1	5.26			
Retired	2	10.53			
IQ (WASI FSIQ)			89	9.22	75-111
Comorbidity					
Affective disorder	10	50			
Psychotic disorder	1	5			
Neurotic disorder	0	0			
Addictions	1	5			
Personality disorder	3	15			
Other psychological disorder	5	25			
Physical disorder	6	30			

Note. a: The percentage values given are calculated on the basis of the number of respondents who provided information on the respective demographic variable

Abbreviations = WASI FSIQ, Wechsler Abbreviated Scale of Intelligence ®, Full Scale IQ (Wechsler, 1999).

A one samples t-test revealed that the mean IQ of the current population was significantly lower than the mean normative score (M=100, SD=15) published for the WASI –II (Wechsler, 1999); $t(19)=-5.34, p<0.001$. As such the impact of current sample IQ on analyses completed will be considered as appropriate.

Procedure

This study formed part of an on-going larger investigation currently being conducted at the NHS OCD clinic where this research was being completed. Adults with a primary diagnosis of OCD were invited to enrol in a cross-sectional study to investigate the prevalence of autistic disorder and traits in the OCD population. Participants recruited were screened for the presence of autistic symptoms using the Autism Quotient (AQ) (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001).

In addition all individuals referred to this service are assessed for OCD severity using the Yale-Brown Obsessive Compulsive Scale (YBOCS) (Goodman, Price, Rasmussen & Mazure, 1989) administered by a clinician. The current diagnosis of OCD in participants recruited to the study was confirmed where possible through repetition of a YBOCS administered by a clinician.

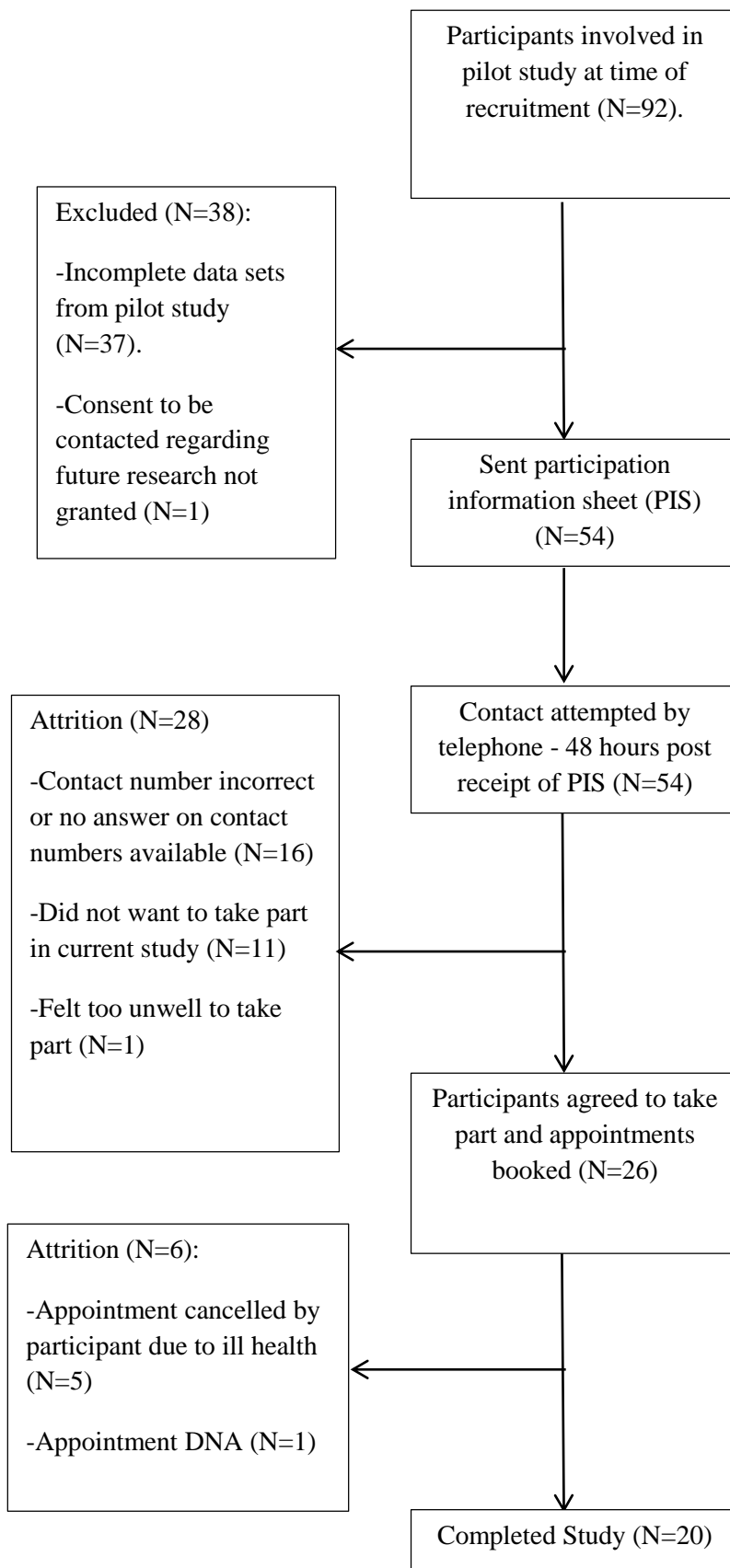
All participants taking part in this larger investigation, who consented to be contacted regarding further research, were identified for inclusion in the current study. Following identification of suitability, these individuals were contacted to determine interest in participation and where appropriate recruited to the study. Participants were recruited jointly with Josselyn Hellriegel (trainee clinical psychologist), with whom I jointly tested the participants for our studies (Hellriegel, 2014). For the purposes of this paper and for clarity, I will only explain the methods for my study but please see Appendix 2 for details of joint work. Each testing session involved the completion of mood screens and a series of neuropsychological tests (see measures), lasted approximately 3 hours and took place at an NHS hospital in a standard clinical room. Breaks were provided when appropriate and required. At the

end of the testing session participants were reimbursed travel expenses (up to a maximum of £10), debriefed and any questions were answered.

Participant recruitment

At the time of recruitment for the current study 92 participants had been or were due to be enrolled in the pilot study. Of these, 37 had not completed the pilot study and were therefore ineligible for the current study and one participant had not provided consent to be contacted regarding future research. The remaining 54 participants were sent a participation information sheet (PIS) (see Appendix 1) in the post by the researchers which outlined the purpose of the study and what would happen should they choose to become involved. Allowing for 48 hours post-receipt of the PIS, we attempted to contact all 54 participants by phone to discuss their participation in the current study. We were unable to reach 16 participants by phone; either the phone number available was incorrect or the individual did not pick up, despite numerous attempts and messages left with contact numbers for returning calls. Eleven participants did not want to take part in more research and one participant was interested in the research but felt too unwell to partake at that time. Twenty-six participants agreed to take part in the study and were booked into appointments, of whom five cancelled their appointments due to ill health and one did not attend their appointment, leaving 20 participants who completed the study. A diagram of the recruitment process can be seen in Figure 1.

Figure 1: *Participant recruitment*



Ethical approval

Ethical approval was obtained from the Harrow NHS ethics committee (See Appendix 3). All participants were provided with a detailed information sheet emphasising that their non-participation would not affect the care they receive. Written consent was attained from all participants (See Appendix 4). All information collected remained confidential and was held anonymously.

Measures

During the testing session participants completed a range of valid and reliable neuropsychological tests to assess a wide range of cognitive abilities (outlined below; see Appendix 5 for detailed descriptions of tests). These tests are routinely used in UK clinical neuropsychological practice and were administered according to the procedures outlined in the appropriate testing manuals or published papers. A fixed order of testing was used for all participants.

Intelligence. Intelligence was measured using the 2 subtest version of the Wechsler Abbreviated Scale of Intelligence ® (WASI – II) (Wechsler, 1999).

Overall executive function. Overall executive function was measured using the modified six elements subtest of the Behavioural Assessment of the Dysexecutive Syndrome (Wilson, Alderman, Burgess, Emslie & Evans, 1996).

Set-shifting. Set-shifting (cognitive flexibility) was measured using the intra-extra dimensional (IED) shift task from the Cambridge Automated Neuropsychological Test Battery (CANTAB) (Cambridge Cognition, 2006).

The CANTAB is a computerised neuropsychological touch-screen test battery which incorporates a number of executive and memory tasks examining a range of neurocognitive functions which tap the frontal lobes and their sub cortical connections (Patel et al. 2010).

Response inhibition. Response inhibition was measured using the Stop Signal task from the CANTAB (Cambridge Cognition, 2006).

Generativity. Generativity was measured using the design fluency subtest from the Delis-Kaplan Executive Function System (D-KEFS) (Delis, Kaplan & Kramer, 2001).

Theory of mind (TOM). Theory of mind was measured using the Revised Eyes test (Baron-Cohen, Wheelwright, Hill, Raste & Plumb, 2001).

Central coherence. Central coherence was measured using the Rey-Osterrieth Complex Figure Test (RCFT) (Osterrieth, 1944).

In addition levels of participant anxiety and depression were assessed using the following measures (See Appendix 5 for details of these measures):

Anxiety. The State-Trait Anxiety Inventory (STAI) (Spielberger, Gorsuch & Lushene, 1970).

Depression. The Montgomery-Asberg Depression Rating Scale (MADRS) (Montgomery & Asberg, 1979).

Before participation in the current study participants completed the following measures of clinical symptomatology as part of the larger investigation being conducted at the NHS clinic (as described above):

Autistic traits. The Autism Quotient (AQ) (Baron-Cohen et al. 2001) was used to screen for autistic traits. The AQ is a 50 item questionnaire designed to quantify autistic traits in individuals with normal intelligence. Each question demands a forced choice response on a four point Likert scale which allows the participant to indicate the extent to which they agree or disagree with the item. The questions are equally divided to cover five different domains associated with ASD: social skills; communication skills; imagination; attention to detail; and attention switching.

The total AQ score (which can range from 0-50) has been used to screen for individuals with likely ASD. A score of 32+ on the AQ has been proposed as a useful cut off for distinguishing those who have clinically significant levels of autistic traits (Baron-Cohen et al. 2001), and correctly identifies 76% of patients (sensitivity 0.77, specificity 0.74) when the AQ is used in a referred clinical sample (Ruzich et al. 2015). Alternatively a score of 26+ on the AQ has been proposed as a more useful cut off threshold (sensitivity is 0.95, specificity 0.52, positive predictive value 0.84, and negative predictive value 0.78) as it correctly classifies a greater number of individuals, 83% (Woodbury-Smith, Robinson, Wheelwright & Baron-Cohen, 2005). The AQ has demonstrated good psychometric properties (Baron-Cohen et al. 2001); the total AQ score and its five subscale scores are normally distributed, have demonstrated good test-retest reliability and good internal consistency (Ruzich et al. 2015).

OCD symptom severity. OCD symptom severity was measured using the Yale-Brown Obsessive Compulsive Scale (YBOCS) administered by a clinician. The YBOCS is widely acknowledged as the gold standard measure of OCD symptom severity and presence; it is a clinician-administered instrument with good psychometric properties (Goodman, Price, Rasmussen & Mazure, 1989).

Design

Both group and multiple single case series designs were employed to investigate relationships between the AQ scores of individuals with OCD and their neurocognitive profiles.

Statistical analysis

Data was analysed using SPSS 22.0 for windows. All data was explored for assumptions of normality. Where assumptions were not met, an appropriate non-parametric test was used as appropriate. There were no outliers or data excluded. All statistical tests used a 0.05 significance level.

One sample T-tests were completed to compare sample population mean scores on neurocognitive tasks to normative means for each task.

Within group correlational analyses between AQ scores and scores on neurocognitive tasks were completed.

Multiple single case series methodology was employed to analyse individual participant cognitive profiles. In multiple single case series design, differences within, rather than between individuals are the basis of investigation and each individual acts as their own control (Towgood et al. 2009). This approach is considered particularly useful when the heterogeneity of a condition, such as OCD,

may lead to group means, which may not reflect the behaviour of any individual within that group (Shallice, Burgess & Frith, 1991).

Results

Sample characteristics

Mental health of participants

Table 2a and 2b show the clinical characteristics of the study sample.

Table 2a: *Characteristics of OCD within study sample*

Participant characteristic	Number	Percent	Mean	SD	Range
OCD severity*					
YBOCS compulsions	18	90	10.53	4.33	2-18
YBOCS obsessions	18	90	10.24	3.51	3-17
YBOCS Total	18	90	19.94	7.77	6-35
OCD – age of onset			11.70	5.66	5-25
OCD – Treatment stage**					
Stage 1	0	0			
Stage 2	2	10			
Stage 3	4	20			
Stage 4	3	15			
Stage 5	2	10			
Stage 6	6	30			
Stage 7	3	15			

Note.

*Of the total sample (N=20), YBOCS data was missing for 10% (N=2).

**See Appendix 6 for definition of treatment stages.

Of the total sample, 90% (N=18) had completed the YBOCS at the start of the study. Of these, 16 participants had clinical levels of OCD symptoms (considered to be indicated by a score >7 (Goodman et al. 1989)); 11.11% (N=2) had subclinical levels of OCD symptoms (scoring between 0-7); 16.67% (N=3) had mild symptoms of OCD (scoring between 8-15); 33.33% (N=6) had moderate symptoms of OCD (scoring between 16-23); 33.33% (N=6) had severe symptoms of OCD (scoring between 24-31); and 5.56% (N=1) had extreme symptoms of OCD (scoring between 32-40).

Table 2b: *Levels of depression and anxiety within study sample*

Participant characteristic	Sample Mean (SD)	Range
MADRS score (Depression)	17.80 (8.03)	7-32
STAI–Trait Score (Trait anxiety)	59.25 (6.77)	44-67
STAI–State Score (State anxiety)	43.10 (13.55)	21-80

Symptoms of depression:

100% of the participants had elevated symptoms of depression (considered to be indicated by a score >6 on the MADRS (Herrmann, Black, Lawrence, Szekely & Szalai, 1998); 70% (N=14) had mild symptoms of depression (scoring between 7-19) and 30% (N=6) had moderate symptoms of depression (scoring between 20-34).

Self-reported Symptoms of anxiety:

100% of the sample (N=20) completed the STAI. Of these, 50% (N=10) had clinically significant levels of anxiety (considered to be indicated by a score above 39-40 on the STAI-State inventory (Addolorato et al. 1999; Knight, Waal-Manning & Spears, 1983).

In addition normative means for the scores on both the STAI Trait and State in adults divided according to gender are, for men, a mean STAI-State score of 35.72 (SD= 10.40) and mean STAI-Trait score of 34.89 (SD=9.19); for women, a mean STAI-State score of 35.20 (SD= 10.61) and a mean STAI-Trait score of 34.79 (SD= 9.22) (Spielberger et al. 1970). Calculating comparative normative means for the current sample according to gender ratio yielded a STAI-State mean score of 34.82 (SD=9.21) and a STAI-Trait mean score of 35.36 (SDS =10.55). Using these proposed normative means for the STAI inventory in one samples t-tests revealed significantly higher STAI State and Trait scores in the current sample compared to

the normative sample ($t(19) = 2.73, p = 0.013$; $t(19) = 15.77, p < 0.001$ respectively).

This suggests that the current sample expressed significantly more symptoms of trait and state anxiety than the normative population.

Medication:

All participants (N=20) were taking at least one form of medication for mental health difficulties at the time of the study. 90% (N=18) were taking a selective serotonin re-uptake inhibitor (SSRI), 5% (N=1) were taking tricyclic antidepressants (TCAs), 50% (N=10) were taking an antipsychotic medication and 15% (N=3) were taking an anxiolytic medication.

Levels of self-reported autistic traits in the study sample

Table 3: *Levels of autistic traits within study sample vs. normative sample: AQ total and subscale scores*

Measure of Autistic Traits	Mean (SD) current sample (N=20)	Range	Mean (SD) <i>Baron-Cohen et al. (2001)</i> norms (N=174)
AQ Total	25 (6.93)	10-36	16.40 (6.30)
AQ Subscales:			
Social Skills	4.60 (2.39)	1-9	2.60 (2.30)
Attention Switching	7.50 (1.82)	3-10	3.90 (1.90)
Attention to detail	5.15 (2.23)	1-9	5.30 (5.20)
Communication	4.05 (1.85)	0-7	2.40 (1.90)
Imagination	3.70 (2.58)	0-8	2.30 (1.70)

All participants included in this study (N=20) completed the AQ, of which 15% (N=3) scored 32 or above and 40% (N=8) scored 26 or above, both potentially indicating clinically significant levels of autistic traits. Baron-Cohen et al. (2001) suggest that only 8% of the general population would score 26+ on the AQ; comparative analysis indicates that the occurrence of clinically significant levels of self-reported autistic traits within the current population (40%) is significantly greater than that which would be anticipated in the general population ($p < 0.001$).

A one-sample t-test revealed that the mean total AQ score in the current sample was significantly higher than the mean total AQ score of the normative sample in Baron-Cohen and colleague's (2001) study; $t(19) = 5.55, p < 0.001$.

In addition, one sample t-tests revealed significantly elevated scores (at $p < 0.05$) in the current population on all AQ subscales when compared to the normative sample, apart from the AQ subscale attention to detail where there was no significant difference between the normative and current sample mean scores.

Relationships between measures of clinical symptomatology

Correlational analyses between scores on the AQ, MADRS and STAI revealed a number of significant relationships. The total score on the AQ was found to be significantly positively correlated to scores on both the MADRS and STAI-Trait Inventory ($r(18) = 0.62, p = 0.004$ and $r(18) = 0.51, p = 0.023$ respectively). The impact of mood will therefore be considered in the analysis of any relationship between AQ score and performance on neurocognitive task.

There were also significant positive correlations between the scores on both the Trait and State Inventories on the STAI and the scores on the MADRS ($r(18) = 0.58, p = 0.007$ and $r(18) = 0.48, p = 0.032$ respectively). The common co-occurrence of anxiety and depression is widely acknowledged (Lamers et al. 2011) and as such this relationship is not unexpected.

No significant relationships were found between current OCD symptomatology as measured by the YBOCS and levels of autistic traits measured by the AQ indicating that levels of autistic traits are independent of the current level of OCD symptomatology and vice versa.

Neurocognitive performance of participants

In order to explore the performance of the current clinical sample on each neurocognitive task, their scores were compared, where available, to widely published normative scores using one samples t-tests. As data obtained from the stop signal task was significantly skewed and as such not normally distributed, a non-parametric equivalent, the one sample Wilcoxon signed rank test, was used to compare the normative and sample performance on the inhibition task.

Table 4: *Mean scores attained on each neurocognitive measure: study sample vs. normative sample.*

Neuro-cognitive domain	Neurocognitive task	Clinical sample Mean, (SD), range	^a Normative Mean, (SD)	Comparison of sample means and normative means
Central Coherence	ROCF- Immediate recall	37.75, (15.99), 20-69	50 (10)	t(19)=-3.43, p=0.003**
	ROCF- Delayed recall	37.45, (17.08), 20-70	50 (10)	t(19)=-3.29, p=0.004**
Generativity	DKEFS- Total Correct	9.10, (2.67), 5-15	10 (3)	t(19)=-1.51, p=0.15
	DKEFS- Composite score	9.15, (2.46), 6-15	10 (3)	t(19)=-1.55, p=0.14
	DKEFS- Contrast score	9.25, (2.83), 3-15	10 (3)	t(19)=-1.19, p=0.25
Inhibition	CANTAB – SST SSRT	173.62, (74.19), 87.23-407.35	186.50 (41.14)	Z = 67, p=0.16
Set-Shifting	CANTAB – IED Total errors (adj)	32.25, (24.21), 7-75	24.15(26.82)	t(19)=1.50, p=0.15
	CANTAB – IED EDS errors	13.85, (11.81), 2-30	7.52 (8.27)	t(19)=2.40, p=0.03*
	CANTAB – IED Stages completed	8.30, (0.98), 7-9	8.62 (1.07)	t(19)=-1.47, p=0.16
Theory of Mind	Mind in the Eyes-Revised	25.95, (4.07), 20-33	26.20 (3.6)	t(19)=-0.28, p=0.79
General Executive Function	BADS-Six Elements Task	3.65, (0.49), 3-4	3.52 (0.8)	t(19)=1.19, p=0.25

Note. *Indicates p<0.05. **Indicates p<0.01.

^a See Appendix 7 for source of normative data.

Abbreviations = BADS: Behavioural Assessment of Dysexecutive syndrome (Wilson et al. 1996). DKEFS: Delis-Kaplan Executive Function System (Delis, et al. 2001). ROCF: Rey-Osterrieth Complex Figure Test (Meyers & Meyers, 1995). IED: Intra-Extra Dimensional Task, EDS: Extra-Dimensional Shift (Cambridge Cognition, 2006). SST: Stop Signal Task, SSRT: Stop Signal Reaction Time (Cambridge Cognition, 2006).

Compared to the normative population, one sample t-tests revealed that the current population displayed significantly greater detailed-focussed processing in tasks of central coherence ($p < 0.01$) and possible impairment in set-shifting at the point of extra dimensional shift (represented by the EDS error scores) ($p < 0.05$). Significant impairments were not identified in generativity, TOM, inhibition or overall executive function compared to the normative populations. It should be noted that performance on the BADS modified six elements task, which measures general executive function, seemed to be at ceiling for the current clinical population and similar ceiling effects for this task have been noted within the normative population (Strauss, Sherman & Spreen, 2006)

Relationship between AQ scores and neurocognitive performance

It was hypothesised that neurocognitive performance would be associated with autistic traits; specifically it was hypothesised that a higher AQ score, indicating greater levels of autistic traits, would be significantly associated with greater impairments in overall executive function, set-shifting, TOM and generativity and weaker central coherence. In addition it was hypothesised that AQ score and greater levels of autistic traits would be negatively correlated to impairments in inhibition.

Control strategy

In order to ensure the reliability and validity of any relationships found between the variables of interest in this study, it is necessary to control for the presence of any other variable (or confound) that may, through association with the variables of interest, distort the outcome and lead to inaccurate results.

Given the low power of the study, in order to determine whether to control for IQ as a third variable, not only significant relationships but also trends (referring

to those associations with an alpha value, $p < 0.1$) were considered sufficient to justify inclusion as a control variable (see correlation matrices in Appendix 8 and 9). There were no significant relationships identified between IQ and performance on any of the neurocognitive tasks apart from significant positive correlations between IQ and performance on the tasks of central coherence (ROCF-delayed and immediate recall) and the total number of correct responses achieved on the generativity task (DKEFS); $r(18) = 0.51$, $p = 0.02$, $r(18) = 0.56$, $p = 0.01$ and $r(18) = 0.45$, $p = 0.05$ respectively). Trends ($p < 0.1$) were also identified between IQ and performance on the composite score in the generativity task (DKEFS), the set-shifting outcome measures, IED total errors adjusted and stages completed, and performance on the task of TOM (Mind in Eyes). A significant relationship was also identified between IQ and the AQ subscale attention shifting ($r(18) = -0.46$, $p = 0.04$). As such, when investigating the relationship between the autistic trait 'attention shifting' and performance on these seven neurocognitive tasks, IQ was entered into the analysis to control for its effect on performance.

In addition, given the significant relationships between AQ score and STAI-Trait and MADRS scores, a preliminary correlational analysis was also performed to determine whether relationships between scores on the STAI-Trait and MADRS and neurocognitive performance might exist independently of AQ. Given the low power of the study, in order to determine whether to control for mood as a third variable, not only significant relationships but also trends (referring to those associations with an alpha value, $p < 0.1$) were considered sufficient to justify inclusion as a control variable. (see correlation matrices in Appendix 8 and 9). Statistically significant relationships and trends were found between the AQ total and all AQ subscales scores (apart from 'attention to detail') and MADRS scores. No statistically

significant relationships were found between these mood measures and neurocognitive performance. However trends ($p < 0.1$) were identified between performance on the set-shifting tasks (IED total errors (adjusted) and Stages completed) and levels of depression. Thus, in investigating the relationship between autistic traits in all domains (apart from attention to detail) and performance on these neurocognitive tasks, the MADRS scores were entered into the analysis as a confounding factor to control for its effect on performance. Neither significant relationships nor trends were identified between STAI- Trait scores and any of the neurocognitive scores and no significant relationships or trends were identified between STAI-State and neurocognitive scores. Therefore, it is assumed that levels of anxiety are not confounding factors in the relationship between autistic traits and neurocognitive performance.

Correlational analyses – AQ scores and neurocognitive performance

Pearson's correlational analyses were performed to assess the strength of relationship between AQ score and performance on neurocognitive tasks (see Table 5). As data obtained from the stop signal task was significantly skewed and as such not normally distributed, a non-parametric equivalent, Spearman's Rho, was used to assess the relationship between impairment in inhibition and autistic traits.

Table 5: Correlations between scores on AQ (total and subscale scores) and scores on neurocognitive tasks

	EF BADs		Generativity DKEFS		Central Coherence ROCF		Inhibition CANTAB	Set-shifting CANTAB		TOM	
	6 elements	Total correct	Composite	Contrast	Immediate recall	Delayed recall	SST Stop Signal Reaction Time	IED EDS Errors	IED Total Errors (adj)	IED Stages Complete	Mind in Eyes - revised
AQ Total	r=-.22 p=.36	r=-.36 p=.13	r=-.26 p=.27	r=-.48* p=.03	r=.16 p=.51	r=-.17 p=.48	r=.18 p=.46	r=.21 p=.38	r=-.00 p=.99	r=-.01 p=.97	r=-.05 p=.83
AQ Social Skills	r=.10 p=.68	r=-.10 p=0.69	r=-.13 p=.58	r=-.20 p=.39	r=.20 p=.40	r=.24 p=.30	r=.39 p=.09	r=.08 p=.73	r=-.18 p=.47	r=.11 p=.65	r=.05 p=.85
AQ Attention Switching	r=-.33 p=.16	r=-.37 p=.12	r=-.24 p=.33	r=-.24 p=.31	r=.11 p=.65	r=-.03 p=.90	r=.33 p=.16	r=.06 p=.82	r=.08 p=.76	r=-.10 p=.69	r=.04 p=.90
AQ Attention to detail	r=-.09 p=.69	r=-.00 p=.10	r=.33 p=.15	r=-.61** p=.01	r=-.20 p=.39	r=-.31 p=.18	r=-.42 p=.06	r=-.19 p=.43	r=-.16 p=.51	r=.15 p=.53	r=.17 p=.48
AQ Communication	r=-.15 p=.52	r=-.37 p=.12	r=-.34 p=.15	r=-.24 p=.30	r=-.10 p=.68	r=-.10 p=.66	r=.33 p=.16	r=.33 p=.16	r=.18 p=.47	r=-.20 p=.42	r=-.20 p=.39
AQ Imagination	r=-.26 p=.28	r=-.36 p=.13	r=-.35 p=.13	r=-.23 p=.33	r=-.24 p=.32	r=-.15 p=.53	r=.09 p=.70	r=.26 p=.26	r=.13 p=.59	r=-.07 p=.77	r=-.09 p=.72

Note. *Indicates $p < 0.05$. **Indicates $p < 0.01$.

Abbreviations = AQ: Autism Quotient (Baron-Cohen et al. 2001). EF: Executive Function. BADs: Behavioural Assessment of Dysexecutive syndrome (Wilson et al. 1996). DKEFS: Delis-Kaplan Executive Function System (Delis et al. 2001). ROCF: Rey-Osterrieth Complex Figure Test (Meyers & Meyers, 1995). IED: Intra-Extra Dimensional Task, EDS: Extra-Dimensional Shift (Cambridge Cognition, 2006). SST: Stop Signal Task (Cambridge Cognition, 2006). TOM: Theory of Mind.

In contrast to predictions, no significant relationships were identified between AQ scores and cognitive abilities in set-shifting (as determined by the IED shift task), inhibition, theory of mind, central coherence or TOM. There were also no

significant relationships between AQ and the BADS six elements general measure of executive function.

However, in accordance with predictions, significant negative correlations were identified between the contrast scores on the generativity task and the AQ total score and AQ subscale attention to detail score ($r(18)=-0.48$, $p=0.03$ and $r(18)=-0.61$, $p=0.01$ respectively). These findings should however be treated very cautiously given the number of correlations completed in the analysis and thus the increased risk of Type I errors.

Multiple single case series analysis:

The individual cognitive profiles of participants

Individual cognitive profiles were investigated to determine if any of the individuals within the current sample have a ‘classic autistic profile’ as defined in the hypotheses; deficits in generativity, set-shifting, TOM and general executive function, a more detailed focussed processing style and unimpaired inhibition. In order to determine these cognitive profiles, individual strengths and weaknesses were calculated based on comparison of individual performance (scores) on each neurocognitive task with the normative mean for that task. Strengths and weaknesses are defined as individual scores which deviate from the normative mean for that task by more than one standard deviation (based on the standard deviation of the normative sample) (see Table 4 for normative means and standard deviation for each neurocognitive task). The direction of the deviation determines if it is a weakness or strength. When the deviation was within one standard deviation of the normative mean then the individual’s score was considered to be within the ‘normal’ range for that task.

Performance on the general executive function task (BADS six elements task) seemed to be at ceiling for the current clinical population; all participants fell within the ‘normal’ range for this task. Therefore, the result of this task for each individual in determining the match with the classic autistic cognitive profile has been excluded.

Table 6: Analysis of the individual cognitive profiles of participants; areas of strength, weakness and normal performance.

.p Number	Central Coherence		Generativity			EF	Inhibition	Set-shifting			TOM
	ROCF Immediate recall	ROCF Delayed Recall	DKEFS-TOTAL CORRECT	DKEFS – Composite Score	DKEFS - Contrast Score	BADS – 6 elements	SST - Stop Signal Reaction Time	IED-Total errors adjusted	IED-EDS errors	IED-Stages completed	Mind in eyes
1	S	S	S	S	N	N	S	N	N	N	N
2	W	W	N	N	N	N	S	N	N	N	N
3	W	W	N	N	N	N	N	N	W	N	S
4	W	W	W	W	N	N	S	N	W	W	W
5	W	W	N	N	N	N	W	N	N	N	W
6	W	W	N	N	N	N	W	W	W	W	W
7	N	N	N	N	W	N	S	N	N	N	S
8	W	W	N	N	N	N	N	W	W	W	S
9	W	W	N	N	N	N	S	N	N	N	N
10	N	N	N	N	N	N	W	N	N	N	N
11	S	S	N	N	S	N	N	N	N	N	N
12	S	S	S	N	S	N	N	N	N	N	N
13	W	W	W	W	N	N	S	W	W	W	N
14	W	W	W	W	N	N	N	W	W	W	W
15	N	W	N	N	N	N	N	N	W	W	N
16	N	N	N	N	N	N	N	N	N	N	N
17	W	W	N	N	W	N	S	N	N	N	S
18	W	N	N	N	N	N	N	W	W	W	N
19	N	W	N	N	N	N	W	N	N	N	N
20	W	W	N	N	W	N	S	N	N	N	N

Abbreviations = S=Strength. W=Weakness. N=Normal. EF: Executive Function. BADS: Behavioural Assessment of Dysexecutive syndrome (Wilson et al. 1996). DKEFS: Delis-Kaplan Executive Function System (Delis, Kaplan & Kramer, 2001). ROCF: Rey-Osterrieth Complex Figure Test (Meyers & Meyers, 1995). IED: Intra-Extra Dimensional Task (Cambridge Cognition, 2006). SST: Stop Signal Task (Cambridge Cognition, 2006).

Two individuals (10% of the sample; participant numbers 4 and 14 in Table 6) clearly demonstrated the classic autistic cognitive profile with deficits in each cognitive domain apart from inhibition. One participant (participant number 13 in Table 6) demonstrated the classic autistic cognitive profile apart from demonstrating a normal performance in TOM. However, it should be noted that this ‘unimpaired’ performance in TOM was only 0.4 points from being considered a weakness.

In addition, three participants (15%, participant numbers 2, 9 and 20 in Table 6) demonstrated weak or normal performances in all cognitive domains apart from inhibition in which they demonstrated a cognitive strength. 10% (N=2, participant numbers 7 and 17 in Table 6) demonstrated weak or normal performances in all cognitive domains apart from inhibition and TOM in which they demonstrated cognitive strengths.

The AQ scores of these 8 individuals identified as having cognitive profiles similar to the classic autistic cognitive profile are detailed in Table 7.

Table 7: AQ scores of participants with cognitive profiles similar to the classic autistic profile

Participant number	4	14	13	2	9	20	7	17
AQ Total:	22	25	25	10	22	34	35	31
AQ subscales:								
Social Skills	1	4	4	1	1	6	7	7
Attention Switching	8	6	8	3	9	10	8	6
Attention to detail	7	2	4	6	9	9	8	5
Communication	3	6	5	0	2	4	6	5
Imagination	3	7	4	0	1	5	6	8

Note: Minimum and maximum scores attainable on each AQ subscale are 0 and 10 respectively. For each subscale, a higher score indicates a higher level of autistic traits.

This multiple single case series methodology is exploratory and has been employed in place of traditional group-level statistics in order to provide an in-depth examination of individual patterns of cognition. In contrast to predictions, there was no evidence for a relationship between individual AQ score and similarity in individual neurocognitive profile to the ‘classic autistic profile’. Instead the participants highlighted, who have cognitive profiles which are more aligned with this autistic profile, have total AQ scores ranging from 10 to 35. However, seven of these eight participants had relatively high AQ scores, scoring above the average AQ score (16.4) for the normative population (Baron-Cohen et al. 2001).

In addition, two of the three participants with AQ scores above the proposed cut off for clinically significant levels of autistic traits (32+) have profiles which are

similar to the ‘classic autistic profile’, although neither of these match this cognitive profile exactly.

Results of an independent samples t-test comparing the mean AQ scores of those individuals with ($M = 25.50$, $SD = 8.09$, $N=8$) and without autistic cognition ($M = 24.67$, $SD = 6.40$, $N=12$) show that there is no significant difference, or any trend towards significance, ($t = -0.26$, $df = 18$, $p = 0.80$) between group scores. However, clearly the numbers included in this analysis do not provide sufficient power to identify relationships of interests and as such these findings should be considered extremely cautiously.

Discussion

In accordance with predictions and the results of previous research (Anholt et al. 2010), elevated levels of autistic traits were identified on the AQ in the current sample of individuals with OCD. However, results relating to associations between self-reported autistic traits and autistic cognition were ambiguous and multiple single case series analysis did not suggest that, in individuals with OCD, there is a subset with both elevated autistic traits and cognition. Whether these elevated AQ scores reflect genuine autistic traits, and whether some individuals with OCD have neurodevelopmental aetiology akin to ASD, has therefore not been verified.

Evidence of elevated traits of ASD in OCD

The finding that eight participants (40%) within the study demonstrated likely clinically significant levels of autistic traits is consistent with previous research indicating an elevated rate of autism within OCD (25%) (Russell et al. 2005) compared to normative populations, adding weight to the possibility that there may be individuals with OCD who have more of a neurodevelopmental aetiology akin to

ASD. Interestingly, participants demonstrated elevated scores on the AQ across all domains associated with ASD apart from attention to detail. This is consistent with previous research demonstrating that this subscale was the only AQ domain which did not predict OCD symptoms or severity (Anholt et al. 2010). Attention to detail is thought to be a measure of an individual's tendency towards detail-oriented attention and repetitive behaviours (Baron-Cohen et al. 2001). It has been suggested that this may be a dimension of autism which is relatively independent of its other features (Hoekstra, Bartels, Cath & Boomsma, 2008; Hurst, Mitchell, Kimbrel, Kwapil & Nelson-Gray 2007). The higher AQ scores for individuals with OCD on all elements of the AQ apart from attention to detail imply that overall scores are not simply being inflated by constructs related to repetitive OCD behaviours but that other characteristics are contributing to the outcome. The possibility that individuals with OCD may share some but not all traits associated with ASD is consistent with the fractionation theory of ASD which suggests that social and non-social symptoms of ASD have distinct causes at the genetic, neural, cognitive and behavioural levels (Happé & Ronald, 2008; Happé, Ronald & Plomin, 2006).

However, it is unclear whether this apparent elevation of self-reported autistic traits represents genuine ASD symptomatology, as opposed to non-autistic difficulties with socialising and communication. This is particularly pertinent given the significant correlations identified between measures of trait anxiety and depression and scores on the AQ, which brings into question the specificity of the AQ. Similar relationships between these measures of clinical symptomatology have been identified in previous research leading to questions around whether the AQ is specifically measuring autistic traits or merely incorrectly identifying depressive or anxious symptoms as autistic traits (Liew, Thevaraja, Hong & Magiati, 2015).

Although there is published evidence of the psychometric value of the AQ (Baron-Cohen et al. 2001), there is some emerging research criticising its comparative value in accurately identifying autistic traits. For example, in a study comparing the psychometric properties of self-report measures of the broad autism phenotype, the AQ was found to have the weakest performance (Ingersoll, Hopwood, Wainer & Donnellan, 2011). Additionally, the validity of results of self-report measures such as the AQ will, by their nature, be impacted by the introspective ability and level of understanding of the individuals answering them, which may be particularly relevant in populations with high levels of ASD traits/symptomatology (Bishop & Seltzer, 2012).

If the AQ is not actually measuring autistic traits but is in fact more a reflection of mood, it is not appropriate to assume that higher scores on the AQ represent genuine ASD symptomatology in this OCD group. However, it should be noted that individuals with ASD are thought to be particularly vulnerable to mental health difficulties such as depression and anxiety (Tantam & Prestwood, 1998); thus it might be that these positive relationships between autistic traits and symptoms of anxiety and depression reflect this increasing vulnerability.

The analysis of neurocognitive profiles within the current sample and their relationship to the self-reported autistic traits was designed to address the question of the validity of any self-reported autistic traits identified.

Do these elevated AQ scores represent genuine autistic symptomatology in OCD?

Results demonstrated that individuals with OCD, compared to normative populations, had lower IQ, impairments in some set-shifting tasks and a more detail-

focussed style of processing. Taken broadly, this provides some tenuous support for individuals with OCD having neurodevelopmental aetiology since an impaired/atypical cognitive performance and lower than average IQ would be anticipated for individuals with neurodevelopmental disorders like ASD that stem from atypical development of the brain (Tager-Flusberg, 1999).

However, high levels of depression and anxiety identified within the current population may explain the lower IQ; research suggests that these mood disorders can affect cognition by biasing attention, perception and memory and interfering with executive function (Chepenik, Cornew & Farah, 2007).

It is of note that, consistent with previous research (Chamberlain et al. 2007), there may be significant impairment in set-shifting within an OCD population since this is a well established area of impairment in ASD. Perhaps set-shifting in OCD warrants further exploration in relation to other indications of atypical neurodevelopment, for example, neurological signs and age of OCD onset.

Central coherence is an area of cognition hitherto largely neglected in OCD research. These findings suggest that further investigation is warranted to determine if a more detailed style of processing is, at least for some with OCD, a stable characteristic and as such an endophenotype as has been proposed for individuals with ASD (Frith & Happé, 1994). All other areas of cognition were unimpaired compared to population norms, including inhibition, which contrasts with previous research into OCD cognition (Chamberlain et al. 2005).

Contrary to predictions, no clear neurocognitive profile was elucidated in relation to autistic traits and, given the limited power of the study, the results indicate that if these relationships do exist they are unlikely to be large in effect. The

hypothesis that individuals with greater levels of autistic traits would demonstrate greater impairments in certain areas of cognition (generativity, set-shifting and TOM) alongside weak central coherence and greater inhibitory control is unsubstantiated.

However, negative correlations were identified between the contrast scores on the generativity task and scores on the AQ total and attention to detail subscale. Although this task is designed mainly to measure generativity, the contrast scores also indicate ability to shift attention and abilities in non-verbal fluency. It may be that within an OCD population there are individuals with higher levels of ASD traits, particularly in the autistic domain attention to detail, which are underpinned by difficulties with generativity and set-shifting. The significance of these findings is, however, undermined by the number of correlations completed and thus the elevated risk of Type I error. However, the possibility that assessing performances in generativity and set-shifting in an OCD population may provide one means of identifying individuals more likely to have neurodevelopmental aetiology like ASD, warrants further investigation in a study with greater power.

The results also present some interesting possible trends (defined as those relationships with an alpha value, $p < 0.1$) between specific cognitive domains and autistic traits, which certainly cannot be assumed to represent true relationships within the data, but may highlight areas of interest for further research with larger samples. For example, in accordance with predictions, a trend was identified between better inhibitory control and greater levels of autistic traits in attention to detail.

It has not been verified by the results of the group analyses correlating cognitive profiles and self-reported autistic traits that these elevated AQ scores represent genuine ASD symptomatology. However, the results highlight interesting areas for future exploratory research and, taken together with group comparisons of neurocognitive performance with normative populations, indicate there may be some neurodevelopmental aetiology in OCD populations.

Is there an ASD subgroup?

The exploratory multiple single case series analysis provides some tenuous descriptive support for the theory that some individuals with OCD have atypical neurodevelopment, similar to that seen in ASD; three participants (15%) presented with the 'classic autistic profile'. However, although each of these participants scored above the mean AQ for a normative population as determined by Baron-Cohen et al. (2001) ($M=16.4$), none scored above the proposed cut offs (32+ or 26+) for a likely diagnosis of autism. Broadening the definition of the 'classic autistic profile' to include those with marked strength in inhibition (which research indicates would be unexpected in an individual with OCD but not necessarily in an individual with a neurocognitive profile akin to ASD) and normal or weak performances in all or all but one of the other cognitive domains assessed resulted in identification of eight individuals (40%) who might be considered to have this broader autistic cognitive profile. Identifying participants in this way captured two of the three individuals who attained AQ scores above the higher cut off (32+) for a likely diagnosis of autism. This study clearly did not have sufficient numbers and therefore power to determine if this group of eight individuals might be meaningfully distinct (in terms of their self-reported autistic traits) from the twelve individuals without this autistic cognition. However, it is of interest that comparison of mean AQ scores

between those with and without this autistic cognition, provides no indication whatsoever of difference.

The results provide insufficient evidence to support the hypothesised relationship between this ‘classic autistic cognitive profile’ and higher autistic traits in OCD. It remains unclear whether there is a subgroup of individuals with OCD who reflect those with ASD.

Research limitations and strengths

The findings described above should be considered cautiously in light of the limitations of the AQ described above and the following methodological limitations.

The small sample size included in this study, resulting from substantial recruitment difficulties (see critical appraisal), limits the power to detect meaningful differences and important associations may have been missed. On the other hand, the number of correlations and analyses completed will have raised the risk of Type I errors and it could be that the associations found are a product of this error rather than representing real relationships of interest. The small sample size leaves the results of group analyses vulnerable to being skewed by non-representative participants or anomalous results. Thus, results are potentially unrepresentative of the wider OCD population meaning that their generalisability is impacted.

The lack of a control group and reliance on normative means to complete group comparisons represents a key weakness in the reliability of these findings and results of these comparisons should be considered cautiously, as estimates.

The specificity of the neuropsychological measures needs to be considered as these are rarely specific to a single cognitive domain and often demand numerous

underlying cognitive processes for completion (Brunsdon & Happé, 2013). Although care was taken to select neuropsychological tasks, which were supported by research to tap into the relevant cognitive domains, in some instances this specificity was unobtainable. For example, research suggests that poorer performance on the Rey Complex Figure Task reflects weaker central coherence (Lezak, Howieson, Loring, Hannay & Fischer, 2004; Spreen & Strauss, 1998) but that this also reflects weaker visuoconstructional ability and visual memory (Webber, Riccio & Cohen, 2012). Attempting to determine the prevalence of a specific cognitive profile using these neurocognitive measures is therefore potentially limited by their lack of specificity.

One strength of the study is that it screened for mood disorders and thus controlled for the possibility that neurocognitive deficits might be mediated by mood rather than neurodevelopmental processes, a factor which has limited previous research in this area (Delorme et al. 2011; Zandt et al. 2007). In addition, this study is the first to explore an overall neurocognitive profile for OCD rather than describe performance on a narrow range of neurocognitive tests.

Conclusions

The results of the current study provide some preliminary evidence for the theory that some individuals with OCD demonstrate cognitive profiles suggestive of atypical neurodevelopment, including lower overall IQ, impaired performances on some neurocognitive tasks and heterogeneous cognitive profiles. However, the results have not provided clear evidence to support the theory that some individuals within this population really do have traits of high functioning autism. It is likely that the apparent elevated autistic traits identified by the AQ in this OCD population

do not represent genuine ASD symptomatology. Nevertheless, the apparent disconnect between AQ scores and neurocognitive profiles may not necessarily reflect the absence of an autistic subgroup within the OCD population. The following explanations for these results should also be considered.

The first explanation relates to the fractionation theory of autism (Happé et al. 2006). It may be that rather than a subgroup with ASD existing within the OCD population, as theorised by Bejerot et al. (2001), there are individuals with OCD who present with some specific autistic traits underpinned by certain cognitive deficits but not with the complete cognitive profile or set of symptoms associated with ASD. Fractionation of the cognitive ASD profile within the OCD population might explain the varied group results in relation to overall associations between autistic traits and cognitive deficits within this population.

Another possible explanation is that a clear ‘autistic cognitive profile’ does not exist and the ambiguous results reflect an attempt erroneously to match traits of autism in an OCD population to a pattern of cognitive strengths and weaknesses that is not reliably associated with ASD. This would be consistent with research which suggests that relationships between cognitive test performance and real-life behaviour in ASD is inconclusive (Brunsdon & Happé, 2013) and the proposal that heterogeneity of cognitive profile within and between individuals with ASD is a defining feature of the disorder (Towgood et al. 2009). Thus, the lack of support for a link between the ‘classic autistic profile’ and AQ scores in the current study may simply reflect an erroneous means of identifying an autistic subgroup within the OCD population.

It may be that a subgroup with ASD traits does exist within an OCD population but it was not clearly identifiable in this study as a result of measurement error and the small sample. The AQ may not have captured traits of autism accurately and specifically (due to potential limitations described above), thereby obscuring clear results pertaining to a relationship between ASD traits and a ‘classic cognitive autistic profile’. This would support the finding that there was a mismatch between individuals within the current sample who demonstrated a ‘classic cognitive autistic profile’ and those who attained clinically significant AQ scores.

Future directions for research

The heterogeneity of cognitive profiles within this small population of individuals with OCD was notable. This heterogeneity suggests that individuals with OCD may be characterised by highly variable cognitive profiles or that numerous subgroups with distinct neurocognitive patterns exist within this population as previously suggested by Nedeljkovic et al. (2009). The multiple single case series analysis, piloted with this population in the current study, has provided some interesting findings with regard to identifying individuals with similar cognitive profiles in a heterogeneous group. This research has highlighted the value of multiple single case series analysis in an OCD population and suggests further research is warranted using this approach (not based on group design and averages which may preclude identification of subgroups) but with greater numbers and therefore power to discriminate subgroups.

Repetition of the study with greater numbers of participants might elucidate more clearly relationships between autistic traits and cognitive strengths and weaknesses in OCD and support clearer identification of specific subgroups within

OCD such as one well described according to ASD traits. It would also be advisable to employ a more objective measure of ASD symptomatology and traits, such as the ADOS (Lord et al. 2001), which is clinician administered and uses behavioural observations to determine presence of autistic traits, meaning it is less likely to be subject to measurement error than the self-report AQ.

Given the preliminary evidence suggesting some atypical neurodevelopment within the current sample of individuals with OCD it would be interesting to explore the nature of this hypothesised neurodevelopment in OCD in more detail. As neurodevelopmental disorders emerge in childhood it would be interesting to investigate changing cognitive processes and autistic traits in children with early onset OCD in a longitudinal designed study.

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Part 3: Critical Appraisal

Introduction

This appraisal will reflect on some of the practical and conceptual issues encountered in the process of conducting this research. The impact of some of the methodological weaknesses, such as difficulties with recruitment and data collection and limitations of statistical analyses employed, will be discussed, as will ethical and practical considerations of carrying out research with a population with complex mental health difficulties. The benefits of conducting joint research will be highlighted and recommendations made for future research.

Origins of the study and motivation for the research

I was motivated to undertake a project related to autism spectrum disorders (ASD) following three years' experience (pre-clinical psychology training) of working with this clinical group. Having observed the diversity both in symptom expression and severity, which was apparent in the individuals with ASD with whom I had worked, I was interested in exploring the theoretical conceptualisation and perception of some disorders as spectrum disorders and the potential for symptom fractionation in autism. The fractionation theory of autism suggests that social and non-social symptoms of ASD have distinct causes at the genetic, neural, cognitive and behavioural levels (Happé, Ronald, & Plomin, 2006; Happé & Ronald, 2008), which opens up the possibility that certain dimensions of autism or autistic traits (in the absence of others) could be shared by other disorders.

The publication of the DSM-5 (American Psychiatric Association, 2013) saw obsessive compulsive disorder (OCD) reconceptualised under the Obsessive Compulsive Spectrum Disorders category, acknowledging a shift in perception of this disorder to a heterogeneous diagnostic entity. During my work in autism

services I had encountered a number of individuals with comorbid diagnoses of OCD and ASD and had noted confusion regarding symptom overlap and as a consequence the interventions offered. The profound impact of both disorders on quality of life for individuals and their families and/or care systems if inadequate or inappropriate support is provided is well documented (Renty & Roeyers, 2006; Subramaniam, Soh, Vaingankar, Picco & Chong, 2013). This motivated my research into the relationship between these disorders with the objective that findings might elucidate areas of overlap and inform more appropriate treatment packages.

Methodological considerations of the Empirical Study

Ethical considerations

Directly recruiting from a single NHS outpatient service and testing at this site was considered to be ethically appropriate given the complex and chronic nature of the mental health presentations of many of the individuals being cared for by the service. Specifically it was thought that familiarity with this service and structure would support both the comfort of the participants during their participation in research and the management of risk as each individual was well known to and had been recently seen by clinicians in the service. Being embedded in the service as a researcher with access to clinicians who were clinically responsible for participants, afforded participants quick access to appropriate professional support should any difficulties arise during the research process. However, in retrospect, it may be that our placement within the clinical team, together with our title of Trainee Clinical Psychologist, could have presented some confusion to participants about our role as researchers rather than clinicians. Although our role as researchers was clearly defined and explained to participants at both the recruitment and testing phases of the

research and outlined within the Participant Information Sheet, participants frequently let us know the degree of distress they were experiencing with a minority expressing suicidal ideation. This was easily managed during the testing sessions which were located within the service where their clinical team was based so that immediate support for them from appropriate clinicians could be arranged. However, expressions of extreme distress and suicidal ideation were also encountered on a few occasions over the phone during the process of recruitment. In these cases it seemed that potential participants were regarding us as part of their clinical care team. Although protocol was in place for the management of these situations (to develop a safety plan over the phone with the individual and immediately contact the clinical team and/or emergency services as appropriate), it raised questions as to the participants' understanding of the limits of our roles and whether this information would have been shared with us had this understanding been in place. Practically we were limited as clinicians as we had restricted background clinical information for each potential participant so that we could remain blind to their likely presentation during the testing phase. This presented an ethical question as to whether it was possible and/or appropriate to manage risk as a clinical researcher whilst also remaining blind to the complexities of a participant's presentation. Although risk was managed adequately, perhaps in retrospect, given the severity of the mental health difficulties of a number of potential participants, a more thorough screening process should have been implemented so that we were informed by the service clinicians of individuals likely to be in particular distress before we made contact about their participation in our research. This might have protected potential participants from divulging information to us that they might

otherwise not have done and allowed for clearer delineation of our roles as researchers rather than clinicians.

Recruitment of clinical participants

Directly and solely recruiting from a pool of individuals who had already completed some related research and had given consent to being contacted for future related research was considered to be an efficient recruitment strategy at the start of the research process. Indeed, before starting the research, we had been assured by the service in which the study was taking place that we would be able to achieve sufficient numbers for our project with ease. However, as the research progressed it became clear that this dependency on a single service and participants from previous related research created some unforeseen limitations to progressing recruitment. In retrospect, recruitment of participants presented the most significant practical challenge of the research process and difficulties encountered necessitated a study redesign. Specifically, the original design of the study had been to divide participants, according to their scores on the AQ, into those with higher and lower autistic traits and compare cognitive profiles between these two groups. An original power analysis was calculated based on a large effect size ($d=0.87$) found in a comparable study which discriminated individuals with ASD from typical controls according to executive function using the CANTAB (Ozonoff et al. 2004). It was determined that an appropriate sample size would be 18 in each group to detect significant group differences.

Unfortunately, as the research project on which our recruitment relied was progressing alongside our research project, the clinicians and researchers were very involved in their own recruitment and struggled to find time to provide us with

information regarding those participants who had provided consent to be contacted regarding future research. We were instead provided with approximately five names and contact numbers every few weeks which was considerably fewer than the anticipated number of participants promised at the outset and from which we expected to recruit. This significantly impacted the progress we could make in recruitment. Service instability and pressures contributed to this disappointing recruitment process. Specifically, there was an imminent threat of service relocation, which had left the service, its systems and its employees in a state of flux, making it difficult for individuals working within this service to keep our research and requirements in mind. Numerous strategies were implemented to speed up the recruitment process including supporting the clinicians with the administrative burden of identifying potential participants and attending clinic days to introduce ourselves and the project to possible participants where appropriate before asking for any commitment to the research. This however was not a straightforward process as disorganised filing systems made identification of potential participants extremely difficult and clinic days seemed to change without warning leading to much time wasting. Ultimately the related research project on which we relied was only able to deliver a pool of 54 potential participants for us to recruit from. It therefore became apparent before the end of our recruitment phase that the possibility of attaining sufficient sample numbers to complete group comparisons as initially planned was unrealistic. The design of the study was necessarily reconceptualised to accommodate the smaller sample size attainable; hence the use of correlational and multiple single case series analysis.

Statistical analyses

The small sample size in the current study presented some challenges in design as the study lacked sufficient power to use group analyses such as the t-test. Fortunately the breadth and amount of data collected for each participant was considerable and enabled the use of both correlational analyses and a novel and interesting analytic approach within the OCD population, multiple single case series analysis. This methodology has been employed successfully in furthering understanding of cognitive deficits both in populations with neurodevelopmental disorders such as ASD (Towgood, Meuwese, Gilbert, Turner & Burgess, 2009) and in other populations such as in schizophrenia (Shallice, Burgess & Frith, 1991) and allows for exploration of heterogeneity within populations which might support identification of sub-groups. As such, in relation to the current research question, this methodology was extremely apt. The interesting findings that were elucidated from the analyses of the current study however are limited by the small sample size in terms of their generalisability to the wider OCD population and research employing this methodology alongside group analyses with greater numbers of participants would be of interest.

In addition, due to the exploratory nature of this study, multiple statistical tests were used which may have increased the risk of type-I error, (making a false positive result). In contrast the low power of the study will have increased the risk of type-II error (making false negative results). Both these possibilities bring into question the reliability and meaningfulness of results attained in the current study; thus it would be recommended that these findings are used for the purposes of identifying areas of interest for future research which would benefit from the use of a larger sample to allow for more robust statistical analyses.

Selection of neurocognitive measures and mood screens

Selection of the neuropsychological measures and mood screens was made with awareness of the cumulative time required for completion to ensure that testing would not be over burdensome and tiring for participants. This was to ensure that participants were able to give their best efforts. A calculation was made that the testing phase should take approximately 2 hours for those without mental health or cognitive difficulties. Taking into consideration the impact that OCD might have on an individual's ability to progress through the tasks at a typical pace, testing sessions of 3 hours were scheduled on the assumption that this would allow for frequent breaks when necessary and longer time for completion. However, this research process has highlighted how difficult completing a comprehensive battery of testing with participants with OCD can be, when their levels of disability and distress are profound and significantly impact their ability to progress through the research tasks.

For example, although for most participants 3 hours was sufficient, for some it became clear that testing within this time frame was neither practical nor ethical as they struggled to progress from one item to the next. In these instances judgements were made to extend testing time allowing for extended breaks to reduce any pressure that might be felt by participants to complete within a given time frame. Completion of questionnaires presented one of the more significant stumbling blocks for many participants with OCD who were often crippled by uncertainty about providing 'just the right' answer. Indecisiveness, intolerance of ambiguity and the need for reassurance are characteristics commonly associated with OCD, which have been identified as potentially interfering with assessment processes (Swinson, Antony, Rachman & Richter, 1998). Numerous strategies were therefore introduced to support completion including gentle encouragement, allowing

participants to find a quiet place where they could answer questions alone and/or providing large visual aids which displayed the options for answers more clearly. Preferences for delivery of the questionnaires were also explored with participants, as some found auditory processing more accessible. In these instances we would read out the items on each questionnaire to support understanding. A dictionary was also made available so that, should the participant wish, they could confirm their understanding of statements or questions. These supportive measures were introduced to balance the provision of sufficient and appropriate time per item with the awareness that it was important to prevent the individual from becoming stuck and caught up in the uncertainty of providing a 'correct' answer, which might cause undue distress. Without exception participants expressed a sense of accomplishment and achievement once they had progressed successfully through the research process and therefore ensuring their success in completion was a priority. However, without this flexibility in time to accommodate differences in abilities, success may not have been supported which might have had a negative impact on participants' sense of well-being. Future research, which aims to complete a comprehensive battery of testing with participants with OCD, should be designed to accommodate these variances in ability and to anticipate challenges so that individuals can, as above, be supported appropriately to succeed in completing the research.

Other considerations

Impact of comorbidity and medication on cognitive performance

Comorbidity and use of medication within the current sample population was high; 70% of participants had at least one physical or mental health disorder comorbid with OCD and 100% of participants were taking at least one form of

medication for mental health difficulties at the time of the study. It was not possible to exclude participants on the basis of comorbidity or medication use given the prevalence of these factors within the population from which we were recruiting. In addition it was thought that this level of comorbidity and medication use might be more representative of the wider population of individuals with OCD (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005) potentially making the results of this study more generalisable. Criticisms of previous research into cognitive deficits within an OCD population have highlighted the failures of studies to screen for or consider the contribution of comorbidity to cognitive profiles (Kuelz, Hohagen & Voderholzer, 2004). Specifically, ignoring the impact that primary mood disorders, such as depression or anxiety, might have on neurocognitive performance has been highlighted as a particular flaw in previous research (Chamberlain et al. 2005). The current study attempted to manage these potentially confounding factors by screening for levels of depression and anxiety using psychometrically sound assessment tools (the MADRS (Montgomery & Asberg, 1979) and the STAI (Spielberger, Gorsuch, & Lushene, 1970) respectively) and controlling for the impact of these on cognitive performance in statistical analyses. It was not possible or appropriate, however, given the time constraints and number of analyses already being completed, to screen independently and control for the impact of each comorbid condition or medication on cognitive performance. However, the impact of medication, or of comorbid conditions, common in OCD, on cognitive performance may be interesting areas to explore in future research with greater resources, time and sample sizes. This may elucidate further the complex relationship between OCD and cognitive deficits.

Joint work

This research was completed jointly with Josselyn Hellreigel (see Appendix 2 for details). There were various benefits to sharing the research process; principally it allowed us to share the burden of recruitment and testing participants, both time consuming processes, and supported the recruitment of more participants than would have been possible independently. Had this process not been shared we would have been trying to recruit from the same small pool of potential participants which might have created unnecessary competition between the projects to recruit the same participants. Working together meant that we were able to pool our financial resources which allowed us to contribute to the cost of participants' travel. This was key in securing as many participants' engagement in the research as possible, as many participants lived some distance from the national OCD service where the testing took place and the cost of their travel was often cited as a significant barrier to their participation.

As we were embedded within a clinical team who were completing associated research there were significant opportunities for sharing knowledge and resources. However, the benefits of working as a research team in this way went beyond providing a forum for mutual learning and development as it also created an environment which promoted motivation and provided support when obstacles to the research process presented themselves.

Conclusions

The issues considered above highlight the methodological and ethical complexities inherent in conducting research with a clinical population with significant mental health difficulties such as OCD. Although adjustments were

required both in the research design and the empirical methods employed to ensure successful completion of the research, this study has demonstrated that, even with small sample sizes, when the appropriate level of flexibility is afforded, meaningful research can be completed with people with complex OCD.

This research has provided some interesting results regarding the likely prevalence of atypical neurodevelopment within OCD which may, at least in part, be associated with autism at a neurocognitive level. The generalisability of these findings is impacted by methodological weaknesses such as the small sample size. However, the research has identified interesting possible associations between evidence of atypical development and autistic traits which can be used to guide future research. In addition the research has successfully piloted a novel statistical approach, multiple single case series analysis, within the OCD population in exploring neurocognitive profiles and highlighted the strengths of combining group and individual analyses in heterogeneous populations.

On reflection, conducting this exploratory research has been a challenging but useful learning process, which has required flexibility and unanticipated adaptations to the research design supporting my development as a scientist-practitioner. The research process has added to my clinical understanding of OCD and reinforced my interest in spectrum disorders such as autism. My initial aim to create a piece of research with ‘perfect’ clear results has been challenged and replaced by an understanding that there is value in exploratory research with clear strengths and limitations, which can be used as a platform to encourage future research in an area which has been relatively neglected.

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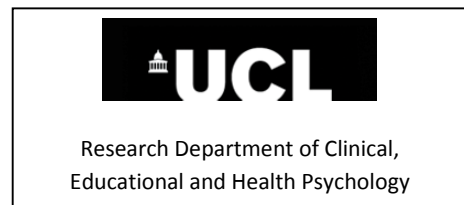
Subramaniam, M., Soh, P., Vaingankar, J.A., Picco, L. & Chong, S.A. (2013). Quality of life in obsessive-compulsive disorder: impact of the disorder and of treatment. *CNS Drugs*, 27, 367-383.

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APPENDICES

Appendix 1 – Participant Information Sheet



PARTICIPANT INFORMATION SHEET

Social style, motivations and reasoning ability of people with OCD

We would like to invite you to take part in our research study. Before you decide whether you would like to participate we would like you to understand why the research is being done and what it would involve for you. **One of our team will go through the information sheet with you and answer any questions you may have.** We suggest this should take about 15 minutes. Please ask us if anything is not clear.

PART 1

What is the purpose of this study?

In this study we are interested in further exploring the underlying mechanism of the obsessions and compulsions seen in OCD to improve treatment outcome. We are interested in the variation in people's social style. Some people find it more difficult to navigate through social situations. These people may have mild symptoms of autism. We are interested if some of the obsessions and compulsions might be related to people's social communication style.

If you are interested in taking part we will ask you to complete questionnaires and complete puzzles and reasoning problems in order to explore people's social style and reasoning abilities. Our hope is that the results will help inform better treatment packages for individuals with a diagnosis of OCD, in particular, better treatment for those who have not benefitted very much from the treatment received so far.

Why have I been invited?

We are inviting participation from patients who have attended or are attending Xxxxx Hospital OCD services and who have taken part in the initial

study being completed by our colleagues in the OCD specialist clinic at XXXXX.

You have been invited because we understand that you have indicated that you would be happy to be contacted regarding participation in further related studies.

We are also inviting participation from patients at the XXXXX OCD services who, although may have missed the opportunity to take part in the initial study, are interested in taking part in this current study.

Our aim is to recruit two groups of equal number of participants to the study; one group who represent those who may have mild autistic traits and one group who do not appear have these traits based on the questionnaires you have previously completed with your clinical care team at XXXXX. All participants will have a diagnosis of OCD. The group results will be compared.

Do I have to take part?

No. Participating in this study is entirely voluntary. It is up to you to decide whether to join the study. We will describe the study and go through this information sheet. If you agree to take part, we will then ask you to sign a consent form. You are free to withdraw or decline to participate at any time, without giving a reason. This would not affect your medical care or legal rights.

What will I have to do if I take part?

If you agree to take part we will invite you to Xxxxx Hospital where we will ask you to complete three questionnaires about your compulsions, sensory experiences (e.g. to noise) and attitudes.

In addition, we will ask you to complete a selection of eight tasks and puzzles which will help us get an understanding of your cognitive strengths and the things you find more difficult e.g. response inhibition. Some of these tasks and puzzles will be paper based (e.g. joining dots in a drawing) and some will be computer based (e.g. pressing a button in response to a picture).

We may ask you to complete up to 4 additional questionnaires about your mood and current OCD symptoms. We will only ask you to complete these additional questionnaires if you have not already done so recently in a clinical appointment at your OCD service.

The questionnaires and tasks should take no longer than 3 hours. You will be able to take breaks during the assessment and can reschedule the assessment or parts of the assessment for another time if you so wish.

We will make a contribution up to a maximum of £10 to any travel expenses with presentation of a travel receipt.

Please refer to the enclosed map for directions.

If I agree to take part what happens to my results?

All the information collected is confidential. Your questionnaires and response booklets will be anonymised and be kept locked in an office. Only the researchers involved in this study will have access to this information.

Reporting the study findings

We will write a report which states group results. We will not include your name or any other information about you that can identify you. Nobody else will know that you took part in the study. In other words, we can guarantee that information about you will be anonymous because we will talk about groups not individuals.

Are there any risks to taking part in the study?

As we will not be giving you any additional treatment for the purpose of the study, there are no specific risks or side effects of taking part in this study. It is unlikely that new difficulties would emerge during the participation in this study that you were not previously aware of and that have not previously been identified by your care team at XXXXX. However, should we identify new symptoms such as those indicating ASD traits, low mood, heightened anxiety, pronounced difficulties in planning and organisation skills we would inform your clinical care team who will consider suitable routes of support if necessary.

We do not think that you will feel distressed as a result of participating in this study. If, however, you do become uncomfortable you will be provided with the opportunity to debrief with a clinician following the task should you wish. As you are part of the OCD clinic professional ongoing support can also be provided if necessary.

Are there any benefits to taking part in the study?

We can not promise that the study will help you but we hope that the information that we get from this study can be used to help other individuals with a diagnosis of OCD. Specifically we hope that the results will help to inform better treatment packages for individuals with OCD and in particular will be beneficial to individuals who have not had successful outcomes from treatment for OCD provided so far.

What happens when the research stops?

Throughout the duration of the research, your care at the XXXXX OCD service will continue as usual. After completion of the research these arrangements will not change and you will still have access to the care support of the OCD clinic

What if there is a problem?

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

Will my taking part in the study be kept confidential?

Yes. We will follow ethical and legal practice and all information about you will be handled in confidence as detailed in part 2.

PART 2:

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

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

You are free to withdraw from the study at any time. If you do withdraw from the study we may still use the data collected up to your withdrawal.

What if there is a problem and how do I make complaints?

If you have any concerns or questions about any aspect of this study, you should contact [REDACTED] who is managing the study.

[REDACTED]

If you wish to complain, or have any concerns about any aspect of the way you have been approached or treated by members of staff you may have experienced due to your participation in the research, National Health Service or UCL complaints mechanisms are available to you.

The Patient Advice and Liaison Service (PALS) can be contacted at:

[REDACTED]

Please ask your research doctor if you would like more information on this. In the unlikely event that you are harmed by taking part in this study, compensation may be available.

If you suspect that the harm is the result of the Sponsor's (University College London) or the hospital's negligence then you may be able to claim compensation. After discussing with your research doctor, please make the claim in writing to the [REDACTED], who is the Chief Investigator for the research and is based at University College London. The Chief Investigator will then pass the claim to the Sponsor's Insurers, via the Sponsor's office. You may have to bear the costs of the legal action, and you should consult a lawyer about this.

Will my taking part in the study be kept confidential?

If you join the study, some relevant parts of your medical records and data collected for the study will be looked at by authorised persons from the

sponsor which includes the researchers from UCL and the care staff at the OCD services at XXXXX. They may also be looked at by authorised people to check that the study is being carried out correctly. All have a duty of confidentiality to you as a research participant and we will do our best to meet this duty. In addition any information, collected during the course of the research, which leaves the hospital will have your name and address removed so that you cannot be recognised.

What will happen to the results of the research study?

The broad scientific results of this study will be presented in peer reviewed journals and conferences. All data presented is based on a group analysis and is anonymised. No individual participant will be identified in any report publication.

Who is organising and funding the research?

This study is being sponsored by the University College London

Who has reviewed the study?

All research in the NHS is looked at by an independent group of people called a Research Ethics Committee to protect your interests. This study has been reviewed and given favourable opinion by London - Harrow Research Ethics Committee- REC number: 13/LO/0595

Appendix 2 – Details of Joint Work

This study was conducted jointly with Josselyn Hellriegel (trainee clinical psychologist).

We both required participants from the same OCD service who had taken part in an initial pilot study which determined their levels of autistic traits by completion of the Autism Quotient (AQ) and as such we jointly recruited and tested participants. This meant that some of the data collection for each of our studies was undertaken by the other trainee. For example, if Ms. Hellriegel met a participant to complete some questionnaires regarding motivational processes in OCD, she would be responsible for completing the neurocognitive measures for my study. Likewise, if I met a participant to complete the neurocognitive tasks for my study I would be required to complete the measures of motivational processes with them for the purposes of Ms. Hellriegel's study. In addition certain data collected, such as the mood questionnaires, were required by both separate research projects and jointly testing participants prevented the duplication of measure administration.

If the task of data collection had not been shared, recruitment of participants to both studies would have been unwieldy as it would have required each participant, who clinically were often quite unwell, to make two rather than one research commitment. Sharing the task of recruitment allowed us to individually obtain more participants, as the number of people willing and able to attend one rather than two recruitment appointments was likely significant. Furthermore, it also allowed us to pool our financial resources so that we were able to give participants compensation for travel to the clinic. Given that the OCD clinic from which we completed our research represented a national OCD service and as such participants often lived some distance away from the service, this financial flexibility may have prevented

the cost of travel acting as a deterrent to participation and enabled recruitment of greater numbers of participants.

Appendix 3 – Ethics approval documentation



Health Research Authority

NRES Committee London - Harrow

Bristol Research Ethics Committee Centre
Level 3, Block B
Whitefriars
Lewins Mead
Bristol
BS1 2NT
Telephone: 01173 421383
Facsimile: 01173 420455

28 June 2013

Dr William Mandy
Lecturer, Research Dept. of Clinical, Educational and Health Psychology, UCL
University College London
Research Dept of Clinical, Educational and Health Psychology
University College London
Gower Street, London
WC1E 6BT

Dear Dr Mandy

Study title: Autistic Traits in Obsessive Compulsive Disorder:
Exploring Underlying Motivational Processes and
Neurocognitive Profiles
REC reference: 13/LO/0595
Protocol number: 1
IRAS project ID: 121916

Thank you for your letter of 28th June 2013. I can confirm the REC has received the documents listed below and that these comply with the approval conditions detailed in our letter dated 26 June 2013

Documents received

The documents received were as follows:

<i>Document</i>	<i>Version</i>	<i>Date</i>
Participant Information Sheet	3	28 June 2013

Approved documents

The final list of approved documentation for the study is therefore as follows:

<i>Document</i>	<i>Version</i>	<i>Date</i>
Covering Letter		21 March 2013
Evidence of insurance or indemnity		30 July 2012
Investigator CV	W.Mandy	06 February 2013
Other: CV- J Hellriegel		20 March 2013

Other: CV- Caroline Barber		06 February 2013
Other: CV- Professor Fineberg		
Other: CV- Maheshi		01 January 2013
Other: CV- Skuse		
Other: CV- Dr Ayman Guirguis		
Other: CV- Sonia		
Other: CV- Sangi		
Other: Neuropsychology Test- Bads 6 elements test		
Other: Neuropsychology Test- Design Fluency test		
Other: Neuropsychology Test- Haylings sentence completion test		
Other: Neuropsychology Test- Revised Eyes test		
Other: Neuropsychology Test- Rey Osterrieth Figures Instructions and Test		
Other: Neuropsychology Test- WASI pages 1-5		
Other: Neuropsychology Test- Subtitle: CANTAB EDID		
Other: Neuropsychology Test- Subtitle: CANTAB ERT		
Other: Letter from Academic Supervisor		08 February 2013
Other: Insurance Confirmation Letter		05 November 2012
Participant Consent Form	2	12 June 2013
Participant Information Sheet	3	28 June 2013
Protocol	1	06 February 2013
Questionnaire: Clinical Global Impression (CGI)		
Questionnaire: Montgomery and Ashberg Depression Rating Scale (MADRS)		
Questionnaire: Yale-Brown Obsessive Compulsive Scale		
Questionnaire: Self-Evaluation Questionnaire - STAI		
Questionnaire: Clinical Global Impression (CGI)		
Questionnaire: Responsibility Attitude Scale (RAS)		
Questionnaire: Sensory Profile		
REC application		27 March 2013
Referees or other scientific critique report	1	
Referees or other scientific critique report		16 October 2012
Response to Request for Further Information		14 June 2013
Summary/Synopsis	1	06 February 2013

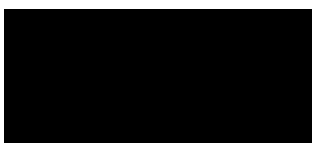
You should ensure that the sponsor has a copy of the final documentation for the study. It is the sponsor's responsibility to ensure that the documentation is made available to R&D offices at all participating sites.

13/LO/0595	Please quote this number on all correspondence
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Yours sincerely



Health Research Authority



Assistant Committee Co-ordinator

E-mail: nrescommittee.london-harrow@nhs.net

Copy to: *Dr Clara Kalu,
Professor Tim Gale, Hertfordshire Partnership NHS Foundation Trust*

NRES Committee London - Harrow

Bristol Research Ethics Committee Centre
Level 3, Block B
Whitefriars
Lewins Mead
Bristol
BS1 2NT
Tel: 01173 421383
Fax: 01173 420455

06 September 2013

Dr William Mandy
Lecturer, Research Dept. of Clinical, Educational and Health Psychology, UCL
University College London
Research Dept of Clinical, Educational and Health Psychology
University College London
Gower Street, London
WC1E 6BT

Dear Dr Mandy

Study title: Autistic Traits in Obsessive Compulsive Disorder:
Exploring Underlying Motivational Processes and
Neurocognitive Profiles
REC reference: 13/LO/0595
Protocol number: 1
Amendment number: Minor Amendment- To replace Hayling Inhibition Task
with Stop-Signal Task
Amendment date: 28 August 2013
IRAS project ID: 121916

Thank you for your letter of 28 August 2013, notifying the Committee of the above amendment.

The Committee does not consider this to be a "substantial amendment" as defined in the Standard Operating Procedures for Research Ethics Committees. The amendment does not therefore require an ethical opinion from the Committee and may be implemented immediately, provided that it does not affect the approval for the research given by the R&D office for the relevant NHS care organisation.

Documents received

The documents received were as follows:

Document	Version	Date
Participant Information Sheet: Quantitative Study	4	28 August 2013
Notification of a Minor Amendment	Minor Amendment- to replace Hayling Inhibition Task with Stop-Signal Task	28 August 2013
Protocol	2	28 August 2013

Statement of compliance

A Research Ethics Committee established by the Health Research Authority

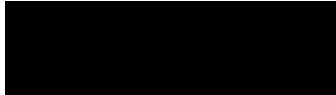


Health Research Authority

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

13/LO/0595:	Please quote this number on all correspondence
--------------------	---

Yours sincerely



REC Assistant

E-mail: nrescommittee.london-harrow@nhs.net

Appendix 4 – Consent form

Centre Number:

Study Number:

Patient Identification Number for this study:



Research Department of Clinical,
Educational and Health Psychology

CONSENT FORM

Title of Project: Social style, motivations and reasoning ability of people with OCD

Name of Researcher(s): **Caroline Barber and Josselyn Hellriegel**

Please initial all
boxes

1. I confirm that I have read and understand the information sheet dated 28 August 2013 (Version 4) for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

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

3. I understand that relevant sections of my medical notes and data collected during the study may be looked at by the research members from University College London, the sponsor, Regulatory Authorities or from the NHS Trust, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records.

4. I agree for my care team to be informed of any additional difficulties arising from the research assessments.

5. I understand that by completing and returning this form, I am giving consent that the personal information I provide will only be used for the purposes of this project and not transferred to an organisation outside of UCL. The information will be treated as strictly confidential and handled in accordance with the provisions of the Data Protection Act 1998.

6. I agree to take part in the above study.



_____	_____	_____
Name of Participant	Date	Signature
_____	_____	_____
Name of Person taking consent	Date	Signature

Appendix 5 – Description of neurocognitive tests employed

Psychometric Tool	Construct measured	Description of test
Wechsler Abbreviated Scale of Intelligence ® -2 subtest version (WASI – II) (Wechsler, 1999).	Intelligence	This brief assessment consists of two measures; vocabulary (measures word knowledge, verbal concept formation, and fund of knowledge) and matrix reasoning (measures visual information processing and abstract reasoning skills) from which a reliable estimate of general intellectual ability can be obtained (full scale IQ (FSIQ)).
The modified six elements subtest of the Behavioural Assessment of the Dysexecutive Syndrome (Wilson et al. 1996);	Overall executive function	An ecologically valid measure of executive function (Norris & Tate, 2000). In this test participants must plan and organise their time (10 minutes) to complete some of each of six separate sub-tasks whilst following predefined rules (Towgood et al. 2009).
The intra-extra dimensional (IED) shift task from the Cambridge Automated Neuropsychological Test Battery (CANTAB) (Cambridge Cognition, 2006).	Set-shifting	<p>The IED task involves the presentation of a series of screens on a tablet computer which demand a touch screen response from the participant. The task consists of nine different stages of increasing difficulty where participants learn a series of nine two-alternative, forced-choice discriminations using feedback provided automatically by the computer. Participants must achieve 6 consecutive correct responses at each stage to progress to the next stage. Initially participants are presented with two simple visual stimuli (coloured shapes) and must learn through trial and error the rule indicating which shape is “correct”. Once the rule is achieved on six consecutive occasions a new rule is introduced which the participant must learn based on the feedback from the computer. In later trials, a second shape is transposed onto each shape, so that the participant must take another dimension into account when determining which shape is correct. Two critical shifts occur during the test, one at the sixth rule change when subjects must shift to new exemplars of the most recent dimension (an intra-dimensional shift) and a second at the eighth rule change, where subjects must shift to a second dimension (an extra-dimensional shift) (Edgin et al. 2010).</p> <p>Three outcome measures were selected to capture set-shifting difficulties within the sample population:</p> <p><i>EDS errors</i>; the number of errors made at the stage where the extra dimensional shift occurs, thought to be a good measure of attentional set-shifting</p> <p><i>IED Total Errors (adjusted)</i>; the number of errors made across the whole task, thought to be a good measure of performance efficiency, adjusted to account for each stage not completed due to failure.</p> <p><i>Stages completed</i>; the number of stages completed out of a total of 9.</p>

Psychometric Tool	Construct measured	Description of test
The Stop Signal task from the CANTAB (Cambridge Cognition, 2006).	Response Inhibition	<p>The Stop Signal task involves the presentation on a tablet computer of a series of screens with a white ring, displayed to alert the subject. Following a fixed 500ms delay, a visual stimulus is displayed within the ring consisting of an arrow pointing to the left or to the right.</p> <p>There are two parts to the test; the first part is a practice round consisting of 16 trials. In this practice round the subject is introduced to the press pad and told to press the left hand button when they see an arrow pointing to the left and the right hand button when they see an arrow pointing to the right. The second part consists of five assessed blocks each of 64 trials. The subject is told to continue pressing the buttons on the press pad when they see the arrows as before, but, if they hear an auditory signal (a beep), to withhold response and not press the button. The test gives a measure of the individual's ability to inhibit a response.</p> <p>One outcome measure was selected to capture impairments in inhibition (specifically prepotent inhibition) within the current sample:</p> <p><i>Stop Signal Reaction Time (SSRT)</i>; an estimate of the length of time between the go stimulus and the stop stimulus at which the subject is able to successfully inhibit their response on 50% of trials. It is therefore a measure of the internal time required to stop the already-triggered motor response.</p>
The Revised Eyes test (Baron-Cohen et al. 2001).	Theory of Mind (TOM)	<p>The Eyes Test is suitable for the detection of mild ToM impairments in individuals with HFA and Asperger syndrome. Scores on the Eyes Test have been found to be negatively correlated to AQ scores (Baron-Cohen et al. 2001). The test consists of 36 black-and-white photographs of the eye region which are presented separately and in a specified order. In each trial, participants are instructed to choose a descriptor from four choices which they believe is the best match to describe what the person in the photograph was thinking or feeling. This procedure requires participants to make decisions with regard to the mental states of others.</p>
The Rey-Osterrieth Complex Figure Test (RCFT) (Osterrieth, 1944);	Central Coherence	<p>A pen and paper test in which participants are asked to copy a complex figure from a piece of paper and then asked to recall the figure without previous warning after an interval of approximately 3 minutes and then again after an interval of 30 minutes. Lower rates of recall often suggest a more detail-focused style (Spreeen & Strauss, 1998; Lezak et al. 2004).</p>

Psychometric Tool	Construct measured	Description of test
The design fluency subtest from the Delis-Kaplan Executive Function System (D-KEFS) (Delis, Kaplan & Kramer, 2001).	Generativity	<p>This test is a nonverbal variant of the verbal fluency test. It is based on earlier versions of the task, such as Design Fluency (Jones-Gotman & Milner, 1977). This is a pen and paper task which requires the production of as many different line-drawing designs by connecting a series of dots according to predefined rules within a delineated time period. There are three separate conditions with differing predefined rules; conditions 1 and 2 involve connecting either filled or empty dots whereas condition 3 involves switching between empty and filled dots which increases cognitive load.</p> <p>Three outcome measures (Delis, Kaplan & Kramer, 2001) were selected to capture impairments in generativity in the current sample:</p> <p><i>Total correct designs completed</i>; representing the total number of correct designs completed across all three conditions.</p> <p><i>Composite score</i>; representing the total number of correct designs generated in conditions 1 and 2. This captures the participant's non-verbal generativity without having to engage in simultaneous cognitive switching.</p> <p><i>Contrast score</i>; representing the total number of correct designs generated in condition 3 as compared with the composite score. As such this score represents the participants' ability to generate designs with set-shifting relative to their ability to generate designs without set-shifting. The contrast score therefore provides a measure of the degree to which a participant may exhibit an impairment in set-shifting above and beyond any deficits in non-verbal generativity.</p>
The State-Trait Anxiety Inventory (STAI) (Spielberger et al. 1970).	Anxiety	The STAI is a widely used measure of state and trait anxiety which has both State and Trait scales. Each scale consists of 20 self-report items that are rated on a 4-point Likert scale. The STAI has demonstrated good psychometric properties (Barnes, Harp, & Jung, 2002); a higher score on each scale represents higher levels of anxiety.
The Montgomery-Asberg Depression Rating Scale (MADRS) (Montgomery & Asberg, 1979).	Depression	The MADRS is a clinician rated 10-item scale designed to measure symptoms of depression. Each item is rated on a 7 point scale (0 to 6) and higher total scores indicate higher levels of depressive symptoms. The MADRS has demonstrated good psychometric properties (Montgomery & Asberg, 1979).

Appendix 6 – Description of OCD treatment stage:

Treatment stage	Description
1	Treatment Naïve.
2	Inadequate course of evidence based treatment.
3	Responded to course of treatment.
4	Failed one course of CBT or evidence based pharmacological treatment with good adherence.
5	Failed 2 courses of CBT or 2 courses evidence based pharmacological treatment with good adherence.
6	Failed 2 courses of CBT including home based therapy or evidence based Pharmacological therapy augmented with antipsychotic or high dose SSRI.
7	Failed 2 courses of CBT including inpatient CBT or augmentation of evidenced based pharmacological therapy.

Note.

Treatment stage defined and assessed by clinicians based within the OCD clinic in which the research took place.

Appendix 7 – Source of normative data for neurocognitive tasks:

- Central Coherence – normative data taken from the Rey Complex Figure Test and Recognition Trial: Professional Manual (Meyers & Meyers, 1995)
- Generativity – normative data taken from the DKEFS Examiner’s Manual (Delis, Kaplan & Kramer, 2001)
- Inhibition – Official normative data for the Stop Signal Task is not yet available from Cambridge Cognition (Cambridge Cognition, 2006) and as such the normative mean score for the SSRT outcome measure was derived from results of previous literature published in a peer reviewed journal (Chamberlain et al. 2007b) which reported mean SSRT score for 20 healthy adult subjects with no history of psychiatric or neurological illness.
- Set-shifting - normative data for the CANTAB IED task were retrieved from Cambridge Cognition. This data had been obtained from over 2000 studies with normal subjects aged 4 to 90 years who participated in several studies conducted primarily in the United Kingdom (Cambridge Cognition, 2006). The normative data was stratified by age and as such for the purposes of the analysis above a single comparative normative mean was calculated for each IED outcome measure according the age ratio in the current clinical sample.
- Theory of Mind - Normative data for the Mind in the Eyes task was taken from Baron-Cohen, wheelwright, Hill, Raste & Plumb’s (2001) study which determined mean normative scores based on the results of 122 healthy adult control participants.
- General executive function – Normative data for the Modified Six Elements task of the BADS was taken from research published by the authors of the test who normed it on a group of 216 healthy adults (Wilson et al. 1996).

Appendix 8 – Correlation matrix – Mood, IQ and neurocognitive performance.

	General Executive Function - BADS	Generativity – DKEFS		Central Coherence – ROCF		Inhibition – CANTAB	Set-shifting – CANTAB		TOM		
	6 elements	Total correct	Composite	Contrast	Immediate recall	Delayed recall	SST Stop Signal Reaction Time	IED EDS Errors	IED Total Errors (adj)	IED Stages Complete	Mind in Eyes - revised
MADRS:	r=-.18 p=.45	r=-.21 p=.39	r=-.19 p=.43	r=-.18 p=.44	r=-.16 p=.49	r=-.18 p=.46	r=.19 p=.43	r=.27 p=.25	r=.38 ^a p=.10	r=-.39 ^a p=.09	r=.00 p=.99
STAI-State:	r=-.08 p=.73	r=.14 p=.56	r=.19 p=.43	r=-.23 p=.34	r=.25 p=.29	r=.19 p=.42	r=.18 p=.44	r=-.08 p=.75	r=.02 p=.94	r=.01 p=.98	r=.34 p=.14
STAI-Trait:	r=-.08 p=.73	r=-.15 p=.52	r=-.07 p=.78	r=-.23 p=.34	r=.19 p=.41	r=.09 p=.72	r=-.10 p=.69	r=.05 p=.85	r=.14 p=.57	r=-.13 p=.58	r=.22 p=.35
WASI IQ:	r=.27 p=.25	r=.45* p=.05	r=.41 ^a p=.07	r=-.01 p=.98	r=.56* p=.01	r=.51* p=.02	r=-.14 p=.56	r=-.38 ^a p=.10	r=-.38 ^a p=.10	r=.35 p=.13	r=.38 ^a p=.10

Note. *Indicates p<0.05. **Indicates p<0.01. ^aIndicates a possible trend p<0.1

Appendix 9 – Correlation matrix – Mood, IQ and autistic traits (AQ scores).

	AQ Total Score	AQ-Social skills	AQ-Attention Switching	AQ-Attention to detail	AQ-Communication	AQ-Imagination
MADRS:	r=-.62** p=.00	r=-.54* p=.01	r=-.48* p=.03	r=-.01 p=.96	r=-.62** p=.00	r=-.39 ^a p=.09
STAI-State:	r=.28 p=.24	r=.42 ^a p=.07	r=.25 p=.30	r=.14 p=.55	r=.31 p=.18	r=-.16 p=.49
STAI-Trait:	r=.51* p=.02	r=.37 p=.11	r=.29 p=.21	r=.24 p=.31	r=.39 ^a p=.09	r=.32 p=.17
WASI IQ:	r=-.29 p=.20	r=-.05 p=.84	r=-.46* p=.04	r=.10 p=.69	r=-.26 p=.26	r=-.32 p=.16

Note. *Indicates p<0.05. **Indicates p<0.01. ^aIndicates a possible trend p<0.1