Special Issue Article

autism

Autism
2014, Vol 18(1) 17–30

DOI: 10.1177/1362361313499456
aut.sagepub.com
SAGE

Exploring the 'fractionation' of autism at the cognitive level

Victoria EA Brunsdon and Francesca Happé

Abstract

Autism spectrum disorders are defined by difficulties across a range of areas: social and communication difficulties and restricted and repetitive behaviours and interests. It has been suggested that this triad of symptoms cannot be explained by a single cause at the genetic, neural or cognitive level. This article reviews the evidence for a 'fractionable' autism triad at the cognitive level, highlighting questions for future research.

Keywords

autism spectrum disorder, central coherence, cognitive theories, executive function, fractionable triad, Theory of Mind

Introduction

Autism has for many years been diagnosed on the basis of the characteristic 'triad' of impairments: social deficits, communicative impairments and restricted and repetitive behaviours and interests (RRBIs) (World Health Organization, 1992). Although the latest edition of *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*) (American Psychiatric Association (APA), 2013) collapses social and communication symptoms into one domain (further discussed below), deficits across the three areas of the triad are still required for a diagnosis of 'autism spectrum disorder (ASD)'. Wing and Gould (1979) introduced the concept of the triad of impairments after finding that children with social impairments often exhibited communication deficits and impoverished imaginative play, with repetitive stereotyped behaviour.

Based on Wing and Gould's epidemiological data, it has long been assumed that the behavioural symptoms of ASD have common causes at the genetic, cognitive and neural levels. However, Wing and Gould (1979) themselves noted that some children presented with only certain aspects of the triad. More recently, it has been found that 10% of children in the general population present with just one impairment (defined as scoring in the most impaired 5%) without co-occurring deficits in other parts of the triad (Ronald et al., 2006a), and modest-to-low phenotypic correlations between triad features have been reported in individuals with ASD (Dworzynski et al., 2009) and trait-wise in general population samples (Ronald et al., 2006b). These findings have been taken to suggest that the triad of impairments is separable at the behavioural level, although this has been a matter of some debate. The work by Constantino et al. (2004), for example, has suggested that a single factor is sufficient to explain variation on the Social Responsiveness Scale. However, more recent work by this group has supported a two-factor solution, distinguishing social and communicative symptoms from rigid and repetitive behaviours (e.g. Frazier et al., 2012). In addition, twin studies have uncovered the relatively independent heritability of each of the three impairments of the triad (Robinson et al., 2012; Ronald et al., 2006a, 2006b, 2011), suggesting that largely non-overlapping genes influence each part of the triad. These observations have led to the proposal of the 'fractionable' autism triad, a theory in which the social and non-social symptoms of ASD are suggested to have distinct causes at the genetic, neural, cognitive and behavioural levels (Happé et al., 2006; Happé and Ronald, 2008). The purpose of this article is to examine the proposal that autism is 'fractionable' at the cognitive level.

King's College London, UK

Corresponding author:
Victoria EA Brunsdon, MRC Social, Genetic & Developmental Psychiatry Centre, Institute of Psychiatry (PO 80), King's College London, Denmark Hill, London SE5 8AF, UK.
Email: victoria.brunsdon@kcl.ac.uk

A range of cognitive accounts have been proposed to explain the symptoms of ASD. These theories posit a primary deficit either in the social domain (e.g. Theory of Mind (ToM), emotion processing and social motivation/reward) or in the non-social domain (e.g. executive dysfunction, weak central coherence (CC) and reduced top-down modulation). However, it is questionable whether any of these theories can account for the full triad of diagnostic features of ASD, let alone the associated features such as raised incidence of talents and uneven cognitive profile. For example, the ToM deficit hypothesis provides a good explanation for the social and communication impairments in ASD, but struggles to explain the non-social domain of ASD, such as RRBIs, motor problems, sensory abnormalities and savant skills. Conversely, non-social cognitive accounts of ASD provide a good explanation for the non-social aspects of ASD. For example, executive dysfunction in ASD may underlie RRBIs due to a failure to generate new behaviours or shift set. In addition, a detail-focused cognitive style may account for 'insistence on sameness', narrow special interests and high rates of talent in ASD. Neither account, however, explains the specific pattern of intact and impaired social cognition (for review, see Frith and Frith, 2010). Consequently, Happé et al. (2006; Happé and Ronald, 2008) proposed that multiple cognitive accounts may apply, each explaining different parts of the ASD triad. This proposal makes a number of predictions (e.g. no one cognitive characteristic of ASD need be specific to ASD), but here we will focus on just two: (1) that performance on social and non-social cognitive tasks should be relatively unrelated and (2) that specific cognitive tests should relate differentially to distinct aspects of the triad of symptoms in ASD.

This article will review the evidence that cognitive functions are fractioned in ASD. First, the relative independence of cognitive functions will be explored. Second, published studies addressing the relation between cognitive tasks and symptoms in ASD will be summarised. Finally, a multiple cognitive deficit account of ASD, incorporating several cognitive functions, will be suggested to provide a better explanation for the complete profile of ASD.

Prediction (1): relations among putative cognitive characteristics of ASD

While by no means the only cognitive theories of ASD, the 'Theory of Mind' (for review, see Frith et al., 1991), 'Executive dysfunction' (Hill, 2004) and 'weak coherence' (Happé and Booth, 2008; Happé and Frith, 2006) accounts are of sufficiently long-standing to have been examined empirically in relation to one another. The fractionated triad account proposed that these three cognitive deficits/styles may be relatively independent and underlie different impairments in ASD (Happé and Ronald, 2008). What is the state of the empirical evidence to date?

ToM and executive function (EF)

In contrast to the prediction that cognitive deficits are independent, a link between ToM and EF in ASD has been reported. Studies with children with ASD have reported positive correlations between false-belief tasks testing ToM and tasks measuring various aspects of EF, including the Luria Hand Game (Bigham, 2010), the Windows task (Russell et al., 1991), the NEPSY Knock-Tap task (no correlations with four other EF tasks; Joseph and Tager-Flusberg, 2004), the Dimensional Change Card Sort task (Colvert et al., 2002; Zelazo et al., 2002), the Wisconsin Card Sort Task and the Tower of Hanoi (Ozonoff et al., 1991). Ozonoff et al. (1991) found that performance on tasks measuring ToM and EF was related in ASD when controlling for IQ, although this correlation was not found in the control group. However, the ASD group exhibited a universal deficit in EF that was not apparent for ToM. Ozonoff et al.'s (1991) conclusion was that executive dysfunction is primary in ASD and is dissociable from ToM deficits, as the two deficits did not always co-occur. In contrast, Harris et al. (2008) reported that individuals with ASD who performed poorly on ToM performed poorly on EF tasks, and vice versa. In addition, Pellicano (2007) reported a significant correlation in an ASD group between a ToM composite and several components of EF (planning, set-shifting and inhibition), independent of age and IQ. Furthermore, and contrary to Ozonoff et al.'s (1991) original finding, EF and ToM were dissociable in one direction only: impaired ToM with intact EF.

Pellicano's (2007) findings offer insight into a possible developmental relation between ToM and EF in ASD. Russell (1996, 1997) suggested that EF is crucial for the development of ToM and that deficits in EF may lead to a failure to develop mental state understanding in ASD. This hypothesis is supported by Pellicano's (2007) results showing that competent EF could be seen without ToM understanding. Examining the same cohort 3 years later, Pellicano (2010b) found that EF was longitudinally predictive of children's ToM test performance. A relation in the opposite direction was not found. Pellicano's work suggests that EF may be a prerequisite for ToM development and may also be critical in determining the developmental trajectory of children's ToM.

These findings do not support the fractionated theory of ASD, which predicts that the distinct cognitive impairments should be independent from each other. However, a number of points should be noted. First, correlational data do not speak directly to causation (Rutter, 2007), and two measures may show a relation due to, for example, general maturational factors at key developmental stages without any direct causal link. Second, cognitive tests are rarely 'process pure', and there is an important distinction to be made between correlations due to shared task demands and correlations due to related underlying processes. For example, some ToM tasks (notably standard false-belief test) require *inhibition* of response based on own belief and may

therefore tap some aspects of EF and mental state attribution. Some EF tasks may also involve social elements; the Luria Hand Game (cited by Pellicano, 2007 as tapping inhibitory control) may also tap the participant's ability to infer the experimenter's intentions so that the participant can produce the opposite action to the experimenter. Ozonoff (1995) showed that performance on a computerised version of the Wisconsin Card Sort Task showed less impairment in ASD than the traditional experimenter-presented version, again suggesting a possible social element to at least some standard EF tests. More recently however, Williams and Jarrold (2013), using a more closely controlled experimental design, failed to find poorer performance on experimenter-administered planning and set-shifting tasks compared to computer versions of the same tasks in an ASD group.

White (2013) has recently proposed, in place of executive dysfunction accounts of ASD, a 'Triple I impairment': impairment in 'Inferring Implicit Information'. White suggests that impairments on EF tasks are not in fact due to core executive dysfunction but instead secondary to mentalising difficulties, that is, those with ASD have difficulties forming an explicit understanding of the experimenter's expectations of the task, resulting in irregular behaviour and performance on only those EF (and other) tasks where inferring this information is essential. It may also be hypothesised that problems in reflecting on *own* mental states (part of the ToM impairment in ASD; Williams and Happé, 2009) may have secondary consequences for EF: for example, difficulties in imaginatively rehearsing possible future activities may lead to impaired planning. While Williams and Jarrold's (in press) study disconfirmed one prediction made by the Triple I hypothesis (better ASD performance on EF tasks when computer- versus experimenter-administered), the authors maintain that ToM and EF may be indirectly linked via developmental effects of ToM on communication and subsequent inner speech.

CC and ToM

The relation between CC and cognitive deficits in ASD has been less widely studied. Some studies have found no links between tasks measuring CC and ToM (Happé, 1997; Pellicano et al., 2006). A local processing bias and poor global processing have been observed in children with ASD, regardless of whether they pass or fail ToM tasks (Happé, 1994, 1997). Burnette et al. (2005) found a link between verbal measures of CC and ToM ability, but this was no longer significant once IQ was taken into account. A similar pattern of results was noted by Pellicano et al. (2006) who found that correlations between performance in ToM and weak CC measures disappeared once age, verbal ability and non-verbal ability were accounted for. Only one study has described a relation between individual differences in ToM and weak CC task performance in ASD (Jarrold et al., 2000). These authors concluded that a ToM deficit may be the result of an inability to take a global view of social situations and a weak drive to integrate social information. It should, perhaps, be noted that Happé and Booth (2008) have suggested that weak CC may itself reflect two separable components that are often confounded in tests: increased local processing and decreased global processing. This raises the possibility that, for example, superior eye for detail is unrelated to ToM, but that reduced integration of information in context may have a detrimental impact on understanding social situations and accurately attributing mental states.

There are a number of other theoretical accounts related to weak coherence, which posit only superior local processing, including Mottron et al.'s (2006) 'enhanced perceptual functioning' theory and Baron-Cohen's 'empathising-systemising' hypothesis. The latter is relevant to the present discussion because systemising (the drive to discover and understand regular systems) is set in contrast to 'empathising' (understanding of social and emotional signals). In discussion of his model, Baron-Cohen typically portrays these social and non-social traits as orthogonal and independent; however, work from his laboratory on the effects of foetal testosterone suggests inverse effects on social-communicative functioning and visuo-spatial and repetitive ASD traits (Auyeung et al., 2010). However, the correlation between performance on tests of empathising (e.g. Reading the Mind in the Eyes) and systemising (e.g. folk physics) has not been widely assessed in an ASD sample; Baron-Cohen et al. (2001) did report a significant negative correlation in a small sample of boys with Asperger syndrome.

CC and EF

Finally, executive dysfunction and weak coherence appear to be dissociable (Booth et al., 2003; Pellicano, 2010b; Pellicano et al., 2006). Pellicano et al. (2006) found that good performance on CC measures was related to better performance on EF tasks in an ASD group, but that correlations were not significant once age and ability were co-varied, perhaps in part because the CC measures used (e.g. Pattern-Construction Task) tapped visuo-spatial ability along with style. In addition, Booth et al. (2003) compared boys with ASD and those with attention deficit/hyperactivity disorder (ADHD) on a drawing task examining both cognitive processing style and planning ability. Only boys with ASD were more detail-focused than controls, but both ASD and ADHD groups showed planning impairments. Furthermore, poor planning ability did not predict a detail-focused cognitive style. Booth and Happé (2010) also report results from a verbal test of coherence in the same ASD and ADHD groups. Here again, only ASD boys were characterised by detail-focus (making more local sentence completions), while both ASD and ADHD groups showed response selection deficits on a Go/No-Go task, and performance on the two tests was not significantly correlated. Research to date

therefore suggests that weak coherence is independent of executive dysfunction, in line with the proposals of the fractionable triad account of ASD.

Finally, Pellicano (2010a, 2010b) conducted the first prospective study to investigate the development of multiple cognitive atypicalities in ASD over a 3-year period. Group differences were reported; children with ASD showed difficulties in false-belief understanding, higher order planning and cognitive flexibility at ages 4–7 and 7–10 relative to typically developing controls. Principal components analysis at Time 1 yielded four factors, with ToM, CC and EF measures falling on separate factors – perhaps supporting in part a fractionable triad view. At Time 2, however, only two factors emerged, with the ToM and EF tasks loading together and only the CC measures remaining distinct. Examining predictors of change over time, Pellicano found that change in ToM showed independent influence from EF and CC performance, while change in EF was not predicted by ToM or CC (over and above Time 1 EF and general ability measures), nor was change in CC performance significantly predicted by ToM or EF measures. Thus, the pattern of interrelations was partly supportive of and partly counter to a fractionable triad view: while EF and CC emerged as relatively distinct, ToM and EF showed a significant concurrent (at Time 2) and developmental relation. The relation between ToM and EF has also been much discussed and researched in the literature on typical development (e.g. Hughes and Ensor, 2007) and acquired neurological damage (e.g. Aboulafia-Brakha et al., 2011), with evidence of strong associations between task performance in the two domains. However, given our specific focus on ASD, further discussion of this work is beyond the scope of the current review.

Prediction (2): relations between cognitive accounts and behavioural symptoms

The fractionated triad theory of autism suggests that different cognitive functions may underlie the distinct symptom domains of ASD (Happé and Ronald, 2008). This predicts that performance on, for example, ToM tests should relate most strongly to social-communicative symptoms, while EF tests may correlate best with non-social repetitive behaviour, and CC measures may relate specifically to uneven cognitive profile, talents and narrow interests. However, surprisingly few studies have investigated whether different cognitive functions are differentially related to distinct parts of the ASD triad of impairments. Of course, the prediction of differential cognition-behaviour links rests on an assumption that significant correlations can be found between any cognitive tasks and everyday behaviours, symptoms or traits. Studies examining these links, and specifically those relevant to the differential links prediction of the fractionable triad hypothesis, are summarised in Table 1 and briefly reviewed below.

ToM and ASD symptoms

Deficits in social cognition, specifically impaired ToM, are hypothesised to underlie the social and communicative symptoms that define ASD (see Tager-Flusberg, 1999). A number of studies have reported a relation between performance on ToM tasks and everyday social abilities in ASD. An early study by Frith et al. (1994) found significantly better real-life social insight (e.g. ability to keep secrets, understand lies) in children with ASD who passed ToM tasks compared to those who failed. Four very recent studies have supported and extended this finding. Lerner et al. (2011) found that ToM ability was negatively correlated with ASD symptoms and social impairments and that fewer ASD symptoms significantly predicted higher ToM scores. Ames and White (2011) investigated the relation between ADHD-related behaviours in a sample of children with ASD and behavioural and cognitive impairments. Poorer performance on ToM measures was significantly related to social difficulties but not to ADHD-related behaviours. Shimoni et al. (2012) found that performance on tasks measuring various aspects of ToM was related to social and communication impairments in ASD, as measured by the Autism Diagnostic Interview–Revised (ADI-R; Lord et al., 1994). Finally, Bennett et al. (2013) reported a significant association between ToM ability in late childhood with later communication skills in adolescence (when controlling for language ability in childhood).

However, not all studies have found a significant relation between performance on ToM measures and everyday social ability in ASD. For example, Loth et al. (2010) found no significant relation between symptoms of ASD and ToM ability in a group of boys with ASD. In addition, Bennett et al. (2013) found no significant associations between ToM ability in late childhood with later social skills in adolescence. Overall, previous findings favour a link between ToM and social skills in ASD, but further studies are necessary to understand the somewhat mixed findings.

Executive functions and ASD symptoms

Executive dysfunction has been hypothesised to explain the RRBIs observed in individuals with ASD. Difficulties in inhibiting inappropriate behaviour, shifting set and generating appropriate new behaviours have been hypothesised to underlie RRBIs (Turner, 1997). Several previous studies have investigated RRBIs in ASD in relation to specific executive processes. Turner (1995) found that RRBIs were most strongly linked to generativity deficits (e.g. verbal fluency) in a sample of young people with ASD. Mosconi et al. (2009) reported that impaired inhibition of prepotent responses was related to increased severity of higher order

Table 1. Published studies assessing cognitive domains in relation to symptom type and severity in autism spectrum disorder (ASD).

Reference	Participants (group means)	ASD diagnosis	Control variables	Cognitive tasks	Symptom measures	Relevant findings (with effect sizes)
ToM						
Ames and White (2011)	55 ASD (9 autism, 30 AS, 16 ASD/PDD-NOS); 48 M, 7 F, CA 10, VIQ 105, PIQ 94	Prior clinician diagnosis	–	ToM Battery, Hayling Sentence Completion Test (to test inhibitory control)	3Di	Low ASD-social impairment group > high ASD-social impairment group on inhibitory control and ToM tasks (d = 0.55). ToM predicts social interaction in ASD (R^2 = 0.08), but not in ADHD group
Bennett et al. (2013)	Time 1: 68 HFA; CA 7, IQ > 68, PIQ 86 Time 2: 39 HFA; CA 15 Time 3: 35 HFA; 31 M, 4 F, CA 17	*DSM-III-R* criteria	Time 1: language	Time 2: Reading the Mind in the Eyes test	VABS	Time 2: ToM predicts Time 3 VABS Communication, controlling for language, but not Socialisation
Frith et al. (1994)	24 autism; 17 M, 7 F, CA 15, MA 7, VIQ 52 15 TD; 5 M, 10 F, CA 4, MA 4, VIQ 93 11 MLD; 7 M, 4 F, CA 9, MA 5, VIQ 60	*DSM-III-R* criteria	–	2 FB tasks (groups divided into passers and failures)	VABS	ASD ToM-passers > ToM-failures on VABS Communication (d = 0.63) and Socialisation (d = 0.54) domain scores.
Lerner et al. (2011)	30 ASD; 24 M, 6 F, CA 14	Prior clinician diagnosis plus SCQ and SRS	–	ToM Inventory	SCQ, Social Skills Rating System–Parent, SRS	ToM scores × parent-reported social skills correlated (r = 0.61), ToM scores × autism-related social impairment negatively correlated (SCQ r = −0.55, SRS r = −0.75). Higher social skills and fewer autistic symptoms predicts ToM scores (R^2 = 0.66).
Loth et al. (2010)	20 ASD (HFA or AS); all M, CA 12, FIQ 108, VIQ 107, PIQ 107 18 TD; all M, CA 11, FIQ 109, VIQ 109, PIQ 112	Prior clinical diagnosis	Groups matched on CA, VIQ, PIQ	ToM: FB task, Strange Stories CC: EFT, Block Design, Sentence Completion Task	Childhood AS Test	No correlation between ToM or CC tasks and ASD symptoms (rs < 0.27)
Shimoni et al. (2012)	25 ASD child; CA 13 25 ASD child's mother; CA 42 25 ASD child's father; CA 47 28 TD child; CA 14 28 TD child's mother; CA 43 28 TD child's father; CA 46	ADOS-G	Age, education and income of parents	ToM: Social Attribution Task	Children: ADOS-G, ADI-R, VABS; Parents: ADI-R, VABS–Expanded Edition	Pertinence index positively correlated with ADI-R social interaction (r = 0.27) and communication deficits (r = 0.39) Salience index negatively correlated with ADI-R (r = −0.35) and communication deficits (r = 0.34). ToM Affective and Salience indices correlated with ADOS-G (Stereotypic and Limited interest items) (r = 0.51)
White et al. (2009)	45 ASD (8 autism, 25 AS, 12 ASD); 41 M, 4 F, CA 9, VIQ 111, PIQ 98 27 TD; 21 M, 6 F, CA 9, VIQ 115, PIQ 103	Prior diagnosis, confirmed with 3Di	Matched gender, CA, VIQ, PIQ	Standard ToM battery; 11 FB tasks and Penny-Hiding Task, Strange Stories	3Di	ASD children with poor ToM had more severe social (d = 0.83) and communication (d = 0.69) symptoms, but not repetitive behaviours
EF						
Akbar et al. (2013)	62 ASD; 47 M, 15 F, CA 9, FIQ 80	ADI-R, ADOS-G	–	Delis–Kaplan EF System, Developmental Neuropsychological Assessment, BRIEF	ADI-R, ADOS-G	Symptom severity does not predict inhibition, working memory or organisation score. Both PIQ and severity of ASD symptoms predicts EF shift score (R^2 = 0.33)
Bishop and Norbury (2005)	14 HFA; PIQ 107 17 SLI; PIQ 99 25 PLI; PIQ 105 18 TD; PIQ 111	SCQ and ADOS-G	PIQ	Two subtests from Test of Everyday Attention for Children (to measure inhibition)	ADOS, SCQ, Children's Communication Checklist	No correlation between inhibition and symptom measures of ASD (rs < 0.14)
Boyd et al. (2009)	61 HFA (31 autism, 22 AS, 5 PDD-NOS); CA 10, IQ 100 64 TD; CA 12, IQ 111	*DSM-IV* criteria, ADI-R, SCQ	–	BRIEF	RBS-R, Sensory Questionnaire	Behaviour regulation correlated with repetitive behaviour (r = 0.43), not sensory impairments (r = 0.03) A diagnosis of ASD, lower age, higher scores on Sensory Questionnaire and BRIEF Behaviour Regulation predicts RBS-R total score (R^2 = 0.86)

(Continued)

Table 1. (Continued)

Reference	Participants (group means)	ASD diagnosis	Control variables	Cognitive tasks	Symptom measures	Relevant findings (with effect sizes)
D'Cruz et al. (2013)	41 ASD (22 autism, 12 PDD-NOS, 7 AS); 34 M, 7 F, CA 15, FIQ 104, VIQ 102, PIQ 105 37 TD; 30 M, 7 F, CA 18, FIQ 109, VIQ 109, PIQ 107	*DSM-IV-TR*, ADOS, ADI-R	CA, gender, IQ	Probabilistic Reversal Learning Task	ADI-R, RBS-R	Positive correlation between poor flexible behaviour and RBS-R total score ($r = 0.34$), ADI RRBI score ($r = 0.37$) and stereotyped, repetitive or idiosyncratic behaviour (ADI-R B3 subscale; $r = 0.38$).
Dichter et al. (2009)	50 ASD; CA 10 IQ 102 42 TD; CA 11 IQ 112	*DSM-IV* criteria, ADI-R, SRS	CA	Generativity tasks: Animals Fluency Task, The Use of Objects Task	ADI-R, SCQ, SRS, RBS-R, Children's Communication Checklist	Only correlations between communication impairments and animal fluency task scores ($r = 0.42$–0.46) No correlation between generativity and repetitive behaviours ($r < 0.30$)
Gilotty et al. (2002)	35 ASD (HFA or autism); 30 M, 5 F, CA 10, IQ 104	*DSM-IV* criteria	–	BRIEF	VABS	BRIEF Initiate and Working Memory subscales negatively correlated with Communication ($r = -0.48$; $r = -0.52$) and Socialisation domains of VABS ($r = -0.64$; $r = -0.57$).
Kenworthy et al. (2009)	89 ASD (34 autism, 32 AS, 23 PDD-NOS); CA 10, VA 10	*DSM-IV* criteria, ADI-R, ADOS	–	BRIEF, Test of Everyday Attention for Children, Tower of London, Semantic Fluency	ADI, ADOS	EF tasks predict communication symptoms (semantic fluency: $\beta = -0.63$; BRIEF: $\beta = 0.30$), social interaction symptoms (divided attention and working memory: $\beta = -0.44$; semantic fluency: $\beta = -0.60$) and RRBIs (BRIEF: $\beta = 0.38$), after accounting for VA and age
LeMonda et al. (2012)	22 ASD; 5 F, 17 M, CA 8, PIQ 98 22 DLD; 5 F, 17 M, CA 8, PIQ 99	*DSM-III-R* criteria, Wing Autistic Disorder Interview Checklist	CA, gender, PIQ, parent education	Wisconsin Card Sorting Task, Mazes subtest of Wechsler Intelligence Scale for Children–Revised Edition, Stanford–Binet Matrices subtest–Fourth Edition	Stereotypies measure: 30-min coded video of semi-structured play	EF tasks predict motor stereotypies ($R^2 = 0.33$). Lower EF scores predict higher frequencies and longer duration of stereotypies in ASD group only ($\beta \geq -0.48 \leq -0.26$)
Lopez et al. (2005)	17 ASD; CA 29 17 TD; CA 29	ADI-R, ADOS-G, Gilliam Autism Rating Scale	CA	Delis–Kaplan Executive Function Scale, Wisconsin Card Sorting Task	ADOS, ADI-R, Gilliam Autism Rating Scale, Aberrant Behavior Checklist-Community	Cognitive flexibility, working memory and response inhibition correlated with RRBIs ($r = 0.63$, $r = -0.56$, $r = 0.58$, respectively). Planning and fluency not correlated with RRBIs ($r = -0.09$, $r = -0.45$, respectively). Together, cognitive flexibility, working memory and response inhibition accounted for a significant proportion of variance in RRBIs ($R^2 = 0.52$)
McEvoy et al. (1993)	17 autism; 10 M, 7 F, CA 5, NVA 12, VA 14 13 DD; CA 4, NVA 12 16 TD; 10 M, 6 F, CA 3, VA 20	*DSM-III-R* criteria, Childhood Autism Rating Scale	Autism and DD: NVA, CA Autism and TD: VA	Piagetian A-not-B Error Task, Delayed Response Task, Spatial Reversal Task, Alternation Task	Early Social Communication Scales	Social interaction and EF correlated ($r = -0.44$), in part due to social interaction and joint attention being correlated ($r = 0.42$).
Mosconi et al. (2009)	18 ASD (13 autism, 5 AS); 14 M, 4 F, CA 18, VIQ 110, PIQ 107, FIQ 109 15 TD; 11 M, 4 F, CA 20, VIQ 110, PIQ 107, FIQ 110	*DSM-IV* criteria, ADI-R, ADOS-G	CA, IQ	Visually guided Saccade Task	ADI-R	Impairments in inhibitory control and higher order RRBIs positively correlated ($r = 0.65$), after controlling for age (partial $r = 0.73$)
Reed et al. (2013)	15 ASD; CA 8, PIQ 71, MA 6 15 TD; CA 7	*DSM-IV*, plus Gilliam Autism Rating Scale	Matched ASD MA to TD CA	Card Sort Task	Gilliam Autism Rating Scale	Perseverative errors correlated to stereotyped behaviours ($r = 0.69$). Perseverative errors not correlated to communication difficulties or social interactions ($rs < 0.26$)
Teunisse et al. (2001)	35 HFA; 26 M, 9 F, CA 19, VIQ 91	*DSM-IV* criteria	–	Card Sorting Tests, CANTAB ID/ED, Switch-in-series	Checklist of 12 *DSM-IV* diagnostic criteria, Wechsler Adult Intelligence Scale–Picture Arrangement, VABS Socialisation Domain	No correlations between EF tasks and social measures (social IQ $r = -0.31$, social competence $r = -0.03$, ASD symptoms $r = 0.22$)
Yerys et al. (2009)	42 ASD (35 HFA, 7 PDD-NOS); 33 M, 9 F, CA 10, FIQ 112 84 TD; 65 M, 19 F, CA 10, FIQ 113	*DSM-IV-TR* criteria ADOS-G, ADI-R	–	ID/ED	ADI-R	ID/ED and RRBI domain of ADI-R correlated ($r = 0.43$, FIQ partialled out). ID/ED and social or communication domain scores not correlated (social symptoms $r = 0.19$; communication symptoms $r = 0.20$)

Table 1. (Continued)

Reference	Participants (group means)	ASD diagnosis	Control variables	Cognitive tasks	Symptom measures	Relevant findings (with effect sizes)
Zandt et al. (2009)	19 ASD (15 AS, 2 autism, 1 PDD-NOS; 16 M, 3 F, CA 11, VIQ 96, PIQ 95 17 OCD; 8 M, 9 F, CA 12, VIQ 94, PIQ 96 18 TD; 6 M, 12 F, CA 12, VIQ 95, PIQ 103	*DSM-IV-TR*	CA, VIQ, PIQ	Verbal Fluency Task, Concept Generation Task–Child Version, Rey Figure, Walk Don't Walk Task, BRIEF	Repetitive Behaviour Questionnaire, Children's Yale–Brown Obsessive Compulsive Scale	Impairment in EF related to higher rates of RRBIs ($r = -0.54$)
CC						
Burnette et al. (2005)	Time 1: 31 HFA; 26 M, 5 F, CA 11 16 TD; CA 11 17 LD Time 2 (15–19 months): 23 HFA; 19 M, 4 F, VIQ 110, PIQ 110 20 TD (12 TD + 6 LD +2 new LD); 15 M, 5 F, CA 11, VIQ 117, PIQ 117	*DSM-IV*	VIQ, PIQ	Block Design, Differential Ability Scales–Pattern Construction, EFT, Homograph Task	HFA Spectrum Screening Questionnaire, Australian Scale for AS	No correlations between CC tasks and ASD symptoms (no statistics stated)
Chen et al. (2009)	29 ASD; 26 M, 3 F, CA 12, IQ 114	Clinical diagnosis, ADOS, SCQ	–	EFT	Childhood Routines Inventory, Short Sensory Profile	Degree of RRBIs ($r = -0.39$), but not sensory abnormalities ($r = -0.02$), predict completion time on the EFT
Drake et al. (2010)	27 TD; 9 M, 18 F, CA 10	–	–	Block Design, EFT, Copying Task	Childhood AS Test	Performance on CC tasks not related to RRBIs ($rs < 0.27$).
Loth et al. (2010)	20 ASD (HFA or AS); all M, CA 12, FIQ 108, VIQ 107, PIQ 107 18 TD; all M, CA 11, FIQ 109, VIQ 109, PIQ 112	Prior clinical diagnosis	CA, VIQ, PIQ	EFT, Block Design, Sentence Completion Task	Childhood AS Test	No correlation between CC tasks and ASD symptoms ($rs < 0.27$).
Morgan et al. (2003)	21 ASD (19 autism, 2 PDD-NOS); 19 M, 2 F, CA 5, MA 3, PIQ 95, VIQ 77 21 TD; 16 M, 5 F, CA 5, MA 5, PIQ 105, VIQ 101	*DSM-IV* criteria	Gender, CA, PIQ	Preschool EFT, Differential Ability Scales – Pattern Construction	ADI-R, ADOS	CC predicts ASD group membership ($d = 1.86$).
Russell-Smith et al. (2012)	80 TD; CA 19, 56 F	–	Age and gender	EFT (group divided by AQ 'social skills' and 'details/patterns' (either high or low)	Autism Spectrum Quotient	EFT scores: high social difficulty > low social difficulty ($d = 0.59$). High details/patterns = low details/patterns ($d = 0.17$)
South et al. (2007)	19 ASD; 14 M, 5 F, CA 15, VIQ 115, PIQ 111, FIQ 114 18 TD; 11 M, 7 F, VIQ 112, PIQ 113, FIQ 112	*DSM-IV* criteria, ADI-R, ADOS-G	CA, VIQ, PIQ, FIQ	EFT, Gestalt closure test	ADOS, ADI-R, Repetitive Behaviour Interview, Yale Special Interests Interview	No correlations between CC tasks and RRBI measures ($rs < 0.30$)
Teunisse et al. (2001)	35 HFA; 26 M, 9 F, CA 19, VIQ 91	*DSM-IV* criteria		Children's EFT, EFT, California Verbal Learning Test-Semantic and Serial Gradient, Visual Object and Space Perception Test–Object Recognition Tasks, Search-for-Difference Task	Checklist of 12 *DSM-IV* diagnostic criteria, Wechsler Adult Intelligence Scale–Picture Arrangement, VABS Socialisation Domain	No correlations between CC tasks and social measures (social IQ $r = 0.00$, social competence $r = 0.16$, autistic symptoms $r = 0.11$)
White and Saldana (2011)	45 ASD; 41 M, 4 F, CA 9, VIQ 111, PIQ 98 27 TD; 21 M, 6 F, CA 9, VIQ 115, PIQ 103	Prior diagnosis ICD-10 criteria, confirmed with 3Di	Gender, CA, VIQ, PIQ	Children's EFT	3Di	No correlations between EFT and ASD symptomatology ($r < 0.22$)

(Continued)

Table 1. (Continued)

Reference	Participants (group means)	ASD diagnosis	Control variables	Cognitive tasks	Symptom measures	Relevant findings (with effect sizes)
Multiple cognitive deficits						
Joseph and Tager-Flusberg (2004)	31 ASD (27 autism, 4 ASD); CA 9, VIQ 20, PIQ 23	*DSM-IV* criteria, ADI-R, ADOS	–	5 EF tasks; Word Span, Block Span, Day–Night, Knock-Tap, Tower	ADOS	ToM correlated with communication ($r = -0.64$), but not social symptoms or RRBIs when non-verbal MA and language controlled for ($rs < 0.34$). Language ($R^2 = 0.33$), ToM ability ($R^2 = 0.28$) and Tower score ($R^2 = 0.05$) predict communication symptoms. Neither ToM nor EF accounted for additional variance in social interaction or repetitive behaviour symptoms
Pellicano et al. (2006)	40 ASD (30 autism, 10 PDD-NOS); 35 M, 5 F, CA 5.6, VIQ 101, PIQ 114 40 TD; 31 M, 9 F, CA 5.4, VIQ 103, PIQ 113	*DSM-IV* criteria, confirmed with ADI-R; TD screened with SCQ	CA, VIQ, PIQ, gender	CC: EFT, Pattern-Construction task, Figure-Ground task, Developmental Test of Visual–Motor Integration ToM: 6 first-order FB tasks, 2 second-order FB tasks EF: Luria Hand Game, Mazes task, Tower of London, Set-shifting task	ADI-R	All cognitive tasks failed to correlate with either ADI-R total or domain scores. EFT and social domain (at 4–5 years) negatively correlated ($r = 0.41$)
Pellicano (2013)	Time 1: 45 ASD; 40 M, 5 F, CA 5.6, VIQ 97, PIQ 113 45 TD; 37 M, 8 F, CA 5.4, VIQ 101, PIQ 116 Time 2: (3 years) 37 ASD; 33 M, 4 F, CA 8.4, VIQ 94, PIQ 104 31 TD; 25 M, 6 F, CA 8.2, VIQ 100, PIQ 107	*DSM-IV* criteria, Time 1 with ADI-R, Time 2 with ADOS-G	Age, VIQ, PIQ (at Time 1)	Time 1: ToM: 2 first-order FB tasks, 1 second-order FB task EF: Teddy-bear set-shifting task, Luria Hand Game, Mazes task CC: EFT, Pattern-Construction task, Figure-Ground task	Time 2: ADOS-G, Repetitive Behaviour Questionnaire	ToM negatively correlated with social communication ($r = -0.42$). EF negatively correlated with social communication and RRBIs (both $r = 0.42$). CC not correlated ($r < 0.21$) EF predicts symptom severity (ADOS; $R^2 = 0.16$) and repetitive behaviours ($R^2 = 0.15$)

ADI-R: Autism Diagnostic Interview–Revised; ADOS-G: Autism Diagnostic Observation Schedule–Generic; AS: Asperger syndrome; β: standardised regression coefficient; BRIEF: Behaviour Rating Inventory of Executive Functioning; CA: chronological age in years; CANTAB: Cambridge Automated Neuropsychological Assessment Battery; CC: central coherence; *d*: Cohen's *d*; DD: developmental delay; 3Di: Developmental, Dimensional and Diagnostic Interview; DLD: developmental language disorder; *DSM: Diagnostic and Statistical Manual of Mental Disorders*; EF: executive function; EFT: Embedded Figures test; F: females; FB: false-belief; FIQ: full-scale intelligence quotient; HFA: high-functioning autism; ICD-10: International Classification of Diseases–Tenth Revision; ID/ED: Intradimensional/Extradimensional shift; LD: learning disability; M: males; MA: mental age in years, MLD: moderate learning difficulties; NVA: non-verbal ability; OCD: obsessive compulsive disorder; PDD-NOS: pervasive developmental disorder–not otherwise specified; PIQ: performance intelligence quotient; PLI: pragmatic language impairment; *r*: correlation coefficient; R^2: coefficient of determination; RBS-R: Repetitive Behaviour Scale–Revised; RRBIs: restricted and repetitive behaviours and interests; SCQ: Social Communication Questionnaire; SLI: specific language impairment; SRS: Social Responsiveness Scale; TD: typically developing, ToM: Theory of Mind; VA: verbal ability; VABS: Vineland Adaptive Behaviour Scales; VIQ: verbal intelligence quotient.

Cohen's *d*, Pearson's correlation coefficient *r* and R^2 are reported to convey effect sizes. Small, medium and large effects for *d* are considered as 0.2, 0.5 and 0.8, respectively, and for *r*, the effects are considered as 0.1, 0.3 and 0.5, respectively (Cohen, 1969). Small, medium and large effects for R^2 are considered as 0.01, 0.09 and 0.25, respectively (Cohen, 1988). In the 'Participants' column, participant characteristics for ASD groups are reported first, followed by participant characteristics for any comparison groups. To group studies by cognitive domain, some articles appear more than once.

repetitive behaviours (e.g. compulsions) in ASD. Furthermore, inhibitory control was unrelated to social and communication symptoms, or sensorimotor behaviours. The same pattern was found for the EF domain of set-shifting; Yerys et al. (2009) reported a significant correlation between set-shifting difficulties and repetitive behaviour (but not social or communicative symptoms) in ASD. South et al. (2007) also found support for a link between cognitive flexibility and repetitive behaviours in children with ASD. In addition, behavioural flexibility has been recently reported to be related to RRBI behaviours but not to social or communication symptoms, in both high- and low-functioning ASD (D'Cruz et al., 2013; Reed et al., 2013). Taking a more comprehensive view of EF, Lopez et al. (2005) noted that some specific executive processes (cognitive flexibility, working memory and response inhibition) were highly related to RRBIs, whereas other executive processes (planning and fluency) were not significantly correlated with RRBIs in adults with ASD.

Just as 'EF' is an umbrella term covering many dissociable components, the RRBI domain of ASD is a varied set. For example, Szatmari et al. (2006) found that RRBIs, as measured by the ADI-R, loaded onto two factors: insistence on sameness versus repetitive sensory and motor behaviours. It may be important to distinguish which aspects of RRBIs are correlated with distinct domains of EF. LeMonda et al. (2012) measured various aspects of EF in children with ASD and developmental language disorders. Lower EF scores predicted higher incidences and longer durations of motor stereotypies (e.g. hand flapping, rocking) in ASD only, when controlling for age, gender and parental education. On the contrary, Boyd et al. (2009) found that EF correlated with RRBIs but not with sensory abnormalities.

Not all studies have documented a significant relation between EF and RRBIs. Zandt et al. (2009) assessed several executive processes and RRBIs in individuals with obsessive compulsive disorder and ASD. The only significant relation uncovered was between generativity and obsessions in the ASD group. Dichter et al. (2009) also found no relation between generativity ability and severity of RRBIs, nor with subscales of higher or lower order repetitive behaviours. In contrast, they found that impaired generativity was related to communication impairments. In a different domain of EF, Bishop and Norbury (2005) did not find an association between inhibition and any of the three symptom domains of ASD. Failure to find a significant relation between executive processes and specific symptoms of ASD may in some cases reflect limited sample size and hence statistical power (e.g. Teunisse et al., 2001). In addition, there is currently no single task or battery of tasks to cover comprehensively all aspects of EF, and different findings may reflect different task or domain selection (see, for example, White's division of EF tasks according to implicit ToM demands, discussed above).

While executive dysfunction has been hypothesised to explain RRBIs in ASD, it may also be relevant to everyday social interaction. Social interactions likely tax many aspects of EF, such as *initiation* of social approach, *flexibility* in social response, *attention* to social cues such as facial expressions, *inhibition* of socially inappropriate behaviour and keeping social networks or different individuals' mental states in *working memory*. In support of this, a link between EF and social-communication skills has been described in young children with ASD (McEvoy et al., 1993). A more comprehensive study was undertaken by Kenworthy et al. (2009) to investigate the link between EF and the three symptom domains of ASD. A composite of scores from the ADI-R and the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000) was used to characterise the three symptom domains, and performance in multiple aspects of EF was examined. Correlation and regression analyses indicated that semantic fluency and divided attention were related to social symptoms, semantic fluency was related to communication symptoms and cognitive flexibility was related to RRBIs, after accounting for verbal ability and age. This study shows the potential for the executive dysfunction account to expand beyond explaining RRBIs to include social and communication symptoms. The applicability of these results to everyday adaptive behaviour has been explored by Gilotty et al. (2002); initiation of behaviour and working memory were found to be related to impairments in social interaction and communication. Thus, some specific elements of EF may have a special relation with social and communication impairments in ASD.

CC and ASD symptoms

The weak CC theory of ASD, describing detail-focus and difficulty integrating information in context for meaning (Frith, 1989), was proposed to explain 'insistence on sameness', narrow interests, uneven cognitive profile and perhaps sensory abnormalities and savant skills (Happé and Vital, 2009). However, as detailed below, studies that have investigated the association between a detailed-processing style and the symptoms of ASD have produced mixed results.

Chen et al. (2009) found a link between a detail-focused processing style in the visual domain and degree of repetitive behaviour in children with ASD. However, there was no relation between detail-focused processing and sensory processing abnormalities. They concluded that sensory processing is a lower level process and so cannot be directly compared to performance on higher level CC tasks. Loth et al. (2008) used sensitivity to context-appropriateness in a change blindness paradigm to tap CC and found a moderate but only marginally significant relation ($r = -0.49$) between ADOS RRBI scores and differences in change detection as a function of context in an ASD sample. Other studies have

found no relation between several measures of repetitive behaviours and CC measures in both children with ASD (South et al., 2007) and typically developing children (Drake et al., 2010). In general, there is a surprising paucity of studies, considering the theoretical appeal of the weak CC account in explaining restricted and repetitive behaviours in ASD – perhaps reflecting the relative lack of research on non-social (compared to social/communicative) aspects of ASD.

Happé and Frith (2006) have specifically limited the explanatory scope of the weak CC account to the non-social features of ASD. However, detail-focus may also have interesting implications for social and communicative functioning in ASD (e.g. Noens and van Berckelaer-Onnes, 2005, 2008). Social interactions involve the integration of discrete cues in context to understand social situations. For example, face-processing and (context-dependent) communication may involve the integration of local details (e.g. facial features) in context. An association between detailed-processing bias and social impairments in 'neurotypical' undergraduates has been reported (Russell-Smith et al., 2012). However, weak coherence has been reported to be unrelated to several measures of social symptoms in ASD samples (Burnette et al., 2005; Teunisse et al., 2001). For example, Morgan et al. (2003) found no relation between measures of CC and social or communicative skills (e.g. joint attention and pretend play) in children with ASD aged 3–5 years.

ToM, EF and CC in relation to ASD symptoms

Only a handful of studies have considered multiple cognitive deficits in relation to the behavioural symptoms of ASD. For example, among a sample of pupils receiving extra support with learning, Best et al. (2008) found that ToM, weak CC and EF all contributed significantly and independently to the prediction of behavioural indicators of ASD (measured by the Social Communication Questionnaire). Only three studies have specifically investigated the relation between test performance in all these cognitive tasks and the symptom domains in individuals with ASD. In Joseph and Tager-Flusberg's (2004) study, ADOS was used to measure symptom severity in children with ASD, and ToM and EF tests were administered. Limited relations were found between the two cognitive tasks and symptom severity in ASD, and relations could be largely accounted for by language ability. However, ToM ability and higher level EF were significantly related to the severity of communication symptoms in ASD, while reciprocal social interaction and RRBIs were relatively independent. In addition, in Pellicano et al.'s (2006) study, parents with children of ASD completed the ADI-R as a measure of symptom severity and were administered a similar battery of tasks to measure CC, ToM and EF. Contrary to Joseph and Tager-Flusberg's (2004) findings and their own predictions, the three cognitive profiles failed to correlate with any of the three symptom domains of ASD.

Pellicano (2013) examined whether early cognitive skills could predict later behavioural symptoms of ASD as measured by the ADOS and Repetitive Behaviour Questionnaire at a 3-year follow-up. ToM was negatively associated with social-communication skills and EF was strongly associated with both social-communication skills and repetitive behaviours. Specifically, early EF, not ToM ability, predicted the degree of social-communication impairment and repetitive behaviours, thus elucidating the important role of EF in influencing the behavioural symptoms of ASD. This very recent study conflicts with the fractionable triad approach as specific cognitive functions were not found to be uniquely associated with distinct ASD symptoms. Instead, Pellicano has suggested that there is unlikely to be one-to-one mapping between cognition and behaviour and that different environmental interactions may affect the way in which cognition influences behaviour, and vice versa.

Towards a multifaceted cognitive account of ASD: questions and future directions

Single cognitive deficit models of ASD have attempted to reduce the varied behavioural symptoms of the condition to a single underlying cognitive deficit. These single deficit models predict strong intercorrelation between performance on tests of ToM, EF and CC. The present review of the existing evidence suggests significant relations between ToM and EF, with some evidence of independence of CC from these abilities. The evidence on relations between cognitive test performance and real-life behaviour or symptoms is patchier, and it is interesting to speculate why test-symptom correlations are often non-significant. Clearly, one of the factors that interposes between individuals' underlying cognitive deficits or style and their behaviour or symptoms is their background of compensatory skills. The pattern of symptoms will reflect both the degree of impairment or cognitive style atypicality and the alternative resources and abilities that the individual can bring to bear in order to compensate for, circumvent or alleviate those difficulties. While these will include commonly measured factors such as IQ and language abilities, they may also reflect differences in environment, intervention, memory or attention. Johnson (2012) has proposed differences in EF as particularly important in compensatory skills. This might provide one explanation for the association found between ToM and EF in the work reviewed above. Work is needed to disentangle the effects of compensation, perhaps by contrasting implicit (e.g. 'anticipatory gaze', see Senju et al., 2009) and explicit ToM task performance in relation to EF abilities in ASD.

Among other areas requiring further research is the examination of developmental effects (e.g. Pellicano, 2013). What might we hypothesise about the relative fractionation of the triad across development? On the one hand, even primarily distinct abilities or traits might be hypothesised to become more intercorrelated with age, due to downstream effects. For example, even if reduced global integrative processing and ToM have independent origins, a child's tendency to interpret stimuli in a context-independent fashion might have developmental effects on their social skills; interaction might be sensitive to mental states but not to different contexts. Similarly, a child with poor inhibitory skills might be poorly tolerated by peers, have reduced social learning opportunities and develop less accurate social insight. On this view, studies with younger age groups will show clearer fractionation of triad domains than studies with older groups.

However, the opposite hypothesis might also be proposed. Neuro-constructivist theories, and accounts of brain development postulating 'interactive specialisation', might suggest greater definition ('modularisation'; D'Souza and Karmiloff-Smith, 2011) of many cognitive abilities with age. Patterns of brain activation during some cognitive tasks become more specialised and focal with age, and one might therefore predict greater differentiation of skills and cognitive functions with increasing age. Further longitudinal studies are needed to test which of these two predictions is correct.

Previous studies have used correlational analyses to assess the degree to which cognitive deficits and behavioural symptoms are associated (e.g. Joseph and Tager-Flusberg, 2004; Pellicano, 2013; Pellicano et al., 2006). However, these types of analyses cannot provide evidence of a direction of causality. Confirmatory factor analysis may be useful in assessing the underlying structure of the behavioural symptoms. Path analysis could be implemented to assess the degree of relation between cognitive processes and behaviour. More complex statistical methods could also be implemented to provide a more parsimonious approach, such as latent class analysis and factor-mixture modelling. These statistical techniques have the potential to provide additional information about cognitive and behavioural subtypes of ASD. For example, Georgiades et al. (2013) used factor-mixture modelling to suggest that the two ASD symptom domains of social-communicative impairments and RRBIs may be independent. The differing symptom profiles of severity suggested support for the existence of three homogeneous subgroups of ASD. Hypothetically, differing cognitive deficits may underlie the symptom profiles of these three subgroups of ASD. Additional analyses, such as latent growth modelling, could also be used to explore cognitive functioning across development and its altering relations with ASD symptoms using a longitudinal framework. These analyses could help test the multiple cognitive deficit model or fractionated triad theory of ASD.

The present review has been concerned with studies of ASD, but clearly of relevance to the fractionated triad account is the existence of other clinical groups in which deficits in just ToM, EF or CC can be documented (see Happé and Ronald, 2008 for discussion). The new *DSM-5* includes a new category of Social (Pragmatic) Communication Disorder, aimed in part at capturing those individuals who may have social and communication problems without RRBIs. It will be interesting and important to see how this influences research and to discover whether ToM, EF and/or CC are affected in such individuals.

Future studies using, for example, intervention approaches as a window into causal relations will be valuable. A pilot study by Fisher and Happé (2005) suggested that training studies may be informative; these authors found that training set-shifting improved ToM skills but not vice versa – although the latter comparison was limited by non-significant gains in generalised set-shifting ability.

If, as the fractionated triad account suggests, ASD is caused by different genes, neural patterns and cognitive components that influence distinct behavioural symptoms, then it is possible that intervention can target particular aspects of ASD while leaving other aspects valued by ASD self-advocates untouched. Understanding the fractionable or monolithic cognitive underpinnings of the autism phenotype has the potential to be both theoretically and practically informative.

Acknowledgements

We are extremely grateful to Harriet Tulip for her help assembling the table.

Declaration of conflicting interests

None.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

References

Aboulafia-Brakha T, Christe B, Martory M-D, et al. (2011) Theory of Mind tasks and executive functions: a systematic review of group studies in neurology. *Journal of Neuropsychology* 5: 39–55.

Akbar M, Loomis R and Paul R (2013) The interplay of language on executive functions in children with ASD. *Research in Autism Spectrum Disorders* 7(3): 494–501.

American Psychiatric Association (APA) (2013) *Diagnostic and Statistical Manual of Mental Disorder (DSM-5)*. 5th ed. Washington, DC: American Psychiatric Association.

Ames CS and White SJ (2011) Brief report: are ADHD traits dissociable from autistic profile? Links between cognition and behaviour. *Journal of Autism and Developmental Disorders* 41(3): 357–363.

Auyeung B, Taylor K, Hackett KG, et al. (2010) Foetal testosterone and autistic traits in 18 to 24-month-old children. *Molecular Autism* 1: 11.

Baron-Cohen S, Wheelwright S, Spong A, et al. (2001) Are intuitive physics and intuitive psychology independent? A test with children with Asperger syndrome. *Journal of Developmental and Learning Disorders* 5: 47–78.

Bennett TA, Szatmari P, Bryson S, et al. (2013) Theory of Mind, language and adaptive functioning in ASD: a neuroconstructivist perspective. *Journal of the Canadian Academy of Child and Adolescent Psychiatry* 22(1): 13–19.

Best CS, Moffat VJ, Power MJ, et al. (2008) The boundaries of the cognitive phenotype of autism: Theory of Mind, central coherence and ambiguous figure perception in young people with autistic traits. *Journal of Autism and Developmental Disorders* 38(5): 840–847.

Bigham S (2010) Impaired competence for pretense in children with autism: exploring potential cognitive predictors. *Journal of Autism and Developmental Disorders* 40(1): 30–38.

Bishop DVM and Norbury CF (2005) Executive functions in children with communication impairments, in relation to autistic symptomatology. 2: response inhibition. *Autism* 9(1): 29–43.

Booth R and Happé F (2010) 'Hunting with a knife and fork': examining central coherence in autism, attention deficit/hyperactivity disorder, and typical development with a linguistic task. *Journal of Experimental Child Psychology* 107(4–5): 377–393.

Booth R, Charlton R, Hughes C, et al. (2003) Disentangling weak coherence and executive dysfunction: planning drawing in autism and attention-deficit/hyperactivity disorder. *Philosophical Transactions of the Royal Society of London: Series B – Biological Sciences* 358(1430): 387–392.

Boyd BA, McBee M, Holtzclaw T, et al. (2009) Relationships among repetitive behaviours, sensory features, and executive functions in high functioning autism. *Research in Autism Spectrum Disorders* 3(4): 959–966.

Burnette CP, Mundy PC, Meyer JA, et al. (2005) Weak central coherence and its relation to Theory of Mind and anxiety in autism. *Journal of Autism and Developmental Disorders* 35(1): 63–73.

Chen YH, Rodgers J and McConachie H (2009) Restricted and repetitive behaviours, sensory processing and cognitive style in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders* 39(4): 635–642.

Cohen J (1969) *Statistical Analysis for the Behavioral Sciences*. NY: Academic Press.

Cohen J (1988) *Statistical Power Analysis for the Behavioral Sciences (2nd ed.)*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Colvert E, Custance D and Swettenham J (2002) Rule-based reasoning and Theory of Mind in autism: a commentary on the work of Zelazo, Jacques, Burack and Frye. *Infant and Child Development* 11(2): 197–200.

Constantino JN, Gruber CP, Davis S, et al. (2004) The factor structure of autistic traits. *Journal of Child Psychology and Psychiatry and Allied Disciplines* 45(4): 719–726.

D'Cruz AM, Ragozzino ME, Mosconi MW, et al. (2013) Reduced behavioural flexibility in autism spectrum disorders. *Neuropsychology* 27(2): 152–160.

Dichter GS, Lam KSL, Turner-Brown LM, et al. (2009) Generativity abilities predict communication deficits but not repetitive behaviors in autism spectrum disorders. *Journal of Autism and Developmental Disorders* 39(9): 1298–1304.

Drake JE, Redash A, Coleman K, et al. (2010) 'Autistic' local processing bias also found in children gifted in realistic drawing. *Journal of Autism and Developmental Disorders* 40(6): 762–773.

D'Souza D and Karmiloff-Smith A (2011) When modularization fails to occur: a developmental perspective. *Cognitive Neuropsychology* 28(3–4): 276–287.

Dworzynski K, Happé F, Bolton P, et al. (2009) Relationship between symptom domains in autism spectrum disorders: a population based twin study. *Journal of Autism and Developmental Disorders* 39(8): 1197–1210.

Fisher N and Happé F (2005) A training study of Theory of Mind and executive function in children with autistic spectrum disorders. *Journal of Autism and Developmental Disorders* 35(6): 757–771.

Frazier TW, Thomas W, Youngstrom EA, et al. (2012) Validation of proposed *DSM-5* criteria for autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry* 51(1): 28–40.

Frith U (1989) *Explaining the Enigma*. Oxford: Blackwell.

Frith U and Frith CD (2010) The social brain: allowing humans to boldly go where no other species has been. *Philosophical Transactions of the Royal Society of London: Series B – Biological Sciences* 365(1537): 165–176.

Frith U, Happé F and Siddons F (1994) Autism and Theory of Mind in everyday life. *Social Development* 3(2): 108–124.

Frith U, Morton J and Leslie AM (1991) The cognitive basis of a biological disorder: autism. *Trends in Neurosciences* 14(10): 433–438.

Georgiades S, Szatmari P, Boyle M, et al. (2013) Investigating phenotypic heterogeneity in children with autism spectrum disorder: a factor mixture modeling approach. *Journal of Child Psychology and Psychiatry and Allied Disciplines* 54(2): 206–215.

Gilotty L, Kenworth L, Sirian L, et al. (2002) Adaptive skills and executive function in autism spectrum disorder. *Child Neuropsychology* 8(4): 241–248.

Happé F (1994) An advanced test of Theory of Mind: understanding of story characters thoughts and feelings by able autistic, mentally handicapped, and normal children and adults. *Journal of Autism and Developmental Disorders* 24(2): 129–154.

Happé F (1997) Central coherence and Theory of Mind in autism: reading homographs in context. *British Journal of Developmental Psychology* 15(1): 1–12.

Happé F and Booth RDL (2008) The power of the positive: revisiting weak coherence in autism spectrum disorders. *Quarterly Journal of Experimental Psychology* 61(1): 50–63.

Happé F and Frith U (2006) The weak coherence account: detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders* 36(1): 5–25.

Happé F and Ronald A (2008) The 'fractionable autism triad': a review of evidence from behavioural, genetic, cognitive and neural research. *Neuropsychology Review* 18(4): 287–304.

Happé F and Vital P (2009) What aspects of autism predispose to talent? *Philosophical Transactions of the Royal Society of London – Series B: Biological Sciences* 364(1522): 1369–1375.

Happé F, Ronald A and Plomin R (2006) Time to give up on a single explanation of autism. *Nature Neuroscience* 9(10): 1218–1220.

Harris JM, Best CS, Moffat VJ, et al. (2008) Autistic traits and cognitive performance in young people with mild intellectual impairment. *Journal of Autism and Developmental Disorders* 38(7): 1241–1249.

Hill E (2004) Evaluating the theory of executive dysfunction in autism. *Developmental Review* 24(2): 189–233.

Hughes C and Ensor R (2007) Executive function and Theory of Mind: predictive relations from ages 2 to 4. *Developmental Psychology* 43(6): 1447–1459.

Jarrold C, Butler DW, Cottington EM, et al. (2000) Linking Theory of Mind and central coherence bias in autism and in the general population. *Developmental Psychology* 36(1): 126–138.

Johnson MH (2012) Executive function and developmental disorders: the flip side of the coin. *Trends in Cognitive Sciences* 16(9): 454–457.

Joseph RM and Tager-Flusberg H (2004) The relationship of Theory of Mind and executive functions to symptom type and severity in children with autism. *Development and Psychopathology* 16(1): 137–155.

Kenworthy L, Black DO, Harrison B, et al. (2009) Are executive control functions related to autism symptoms in high-functioning children? *Child Neuropsychology* 15(5): 425–440.

LeMonda BC, Holtzer R and Goldman S (2012) Relationship between executive functions and motor stereotypies in children with autistic disorder. *Research in Autism Spectrum Disorders* 6(3): 1099–1106.

Lerner MD, Hutchins TL and Prelock PA (2011) Brief report: preliminary evaluation of the Theory of Mind Inventory and its relationship to measures of social skills. *Journal of Autism and Developmental Disorders* 41(4): 512–517.

Lopez BR, Lincoln AJ, Ozonoff S, et al. (2005) Examining the relationship between executive functions and restricted, repetitive symptoms of autistic disorder. *Journal of Autism and Developmental Disorders* 35(4): 445–460.

Lord C, Risi S, Lambrecht L, et al. (2000) The Autism Diagnostic Observation Schedule-Generic: a standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders* 30(3): 205–223.

Lord C, Rutter M and Le Couteur A (1994) Autism Diagnostic Interview-Revised: a revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders* 24(5): 659–685.

Loth E, Gómez JC and Happé F (2008) Detecting changes in naturalistic scenes: contextual inconsistency does not influence spontaneous attention in high-functioning people with autism spectrum disorder. *Autism Research* 1(3): 179–188.

Loth E, Happé F and Gómez JC (2010) Variety is not the spice of life for people with autism spectrum disorders: frequency ratings of central, variable and inappropriate aspects of common real-life events. *Journal of Autism and Developmental Disorders* 40(6): 730–742.

McEvoy RE, Rogers SJ and Pennington BF (1993) Executive function and social communication deficits in young autistic children. *Journal of Child Psychology and Psychiatry and Allied Disciplines* 34(4): 563–578.

Morgan B, Maybery M and Durkin K (2003) Weak central coherence, poor joint attention, and low verbal ability: independent deficits in early autism. *Developmental Psychology* 39(4): 646–656.

Mosconi MW, Kay M, D'Cruz AM, et al. (2009) Impaired inhibitory control is associated with higher-order repetitive behaviors in autism spectrum disorders. *Psychological Medicine* 39(9): 1559–1566.

Mottron L, Dawson M, Soulières, et al. (2006) Enhanced perceptual functioning in autism: an updated model, and eight principles autistic perception. *Journal of Autism and Developmental Disorders* 36(1): 27–43.

Noens I and van Berckelaer-Onnes I (2005) Captured by details: sense-making, language and communication in autism. *Journal of Communication Disorders* 38(2): 123–141.

Noens I and van Berckelaer-Onnes I (2008) The central coherence account of autism revisited: evidence from the ComFor study. *Research in Autism Spectrum Disorders* 2(2): 209–222.

Ozonoff S (1995) Reliability and validity of the Wisconsin Card Sorting Test in studies of autism. *Neuropsychology* 9(4): 491–500.

Ozonoff S, Pennington BF and Rogers SJ (1991) Executive function deficits in high-functioning autistic individuals: relationship to Theory of Mind. *Journal of Child Psychology and Psychiatry and Allied Disciplines* 32(7): 1081–1105.

Pellicano E (2007) Links between Theory of Mind and executive function in young children with autism: clues to developmental primacy. *Developmental Psychology* 43(4): 974–990.

Pellicano E (2010a) The development of core cognitive skills in autism: a 3-year prospective study. *Child Development* 81(5): 1400–1416.

Pellicano E (2010b) Individual differences in executive function and central coherence predict developmental changes in Theory of Mind in autism. *Developmental Psychology* 46(2): 530–544.

Pellicano E (2013) Testing the predictive power of cognitive atypicalities in autistic children: evidence from a 3-year follow-up study. *Autism Research*. Epub ahead of print 14 March 2013. DOI: 10.1002/aur.1286.

Pellicano E, Maybery M, Durkin K, et al. (2006) Multiple cognitive capabilities/deficits in children with an autism spectrum disorder: 'Weak' central coherence and its relationship to Theory of Mind and executive control. *Development and Psychopathology* 18(1): 77–98.

Reed P, Watts H and Truzoli R (2013) Flexibility in young people with autism spectrum disorders on a card sort task. *Autism* 17(2): 162–171.

Robinson EB, Koenen KC, McCormick MC, et al. (2012) A multivariate twin study of autistic traits in 12-year-olds: testing the fractionable autism triad hypothesis. *Behavior Genetics* 42(2): 245–255.

Ronald A, Happé F, Bolton P, et al. (2006a) Genetic heterogeneity between the three components of the autism spectrum: a twin study. *Journal of the American Academy of Child and Adolescent Psychiatry* 45(6): 691–699.

Ronald A, Happé F, Price TS, et al. (2006b) Phenotypic and genetic overlap between autistic traits at the extremes of the general population. *Journal of American Academy of Child and Adolescent Psychiatry* 45(10): 1206–1214.

Ronald A, Larsson H, Anckarsater H, et al. (2011) A twin study of autism symptoms in Sweden. *Molecular Psychiatry* 16(10): 1039–1047.

Russell J (1996) *Agency: Its Role in Mental Development*. Hove: Erlbaum.

Russell J (1997) How executive disorders can bring about an inadequate Theory of Mind. In: Russell J (ed.) *Autism as an Executive Disorder*. Oxford: Oxford University Press, pp. 256–304.

Russell J, Mauthner N, Sharpe S, et al. (1991) The windows task as a measure of strategic deception in pre-schoolers and autistic subjects. *British Journal of Developmental Psychology* 9(2): 331–349.

Russell-Smith SN, Maybery MT, Bayliss DM, et al. (2012) Support for a link between the local processing bias and social deficits in autism: an investigation of embedded figures test performance in non-clinical individuals. *Journal of Autism and Developmental Disorders* 42(11): 2420–2430.

Rutter M (2007) Proceeding from observed correlation to causal inference: the use of natural experiments. *Perspectives on Psychological Science* 2(4): 377–395.

Senju A, Southgate V, White S, et al. (2009) Mindblind eyes: an absence of spontaneous Theory of Mind in Asperger syndrome. *Science* 325(5942): 883–885.

Shimoni HN, Weizman A, Yoran RH, et al. (2012) Theory of Mind, severity of autistic symptoms and parental correlates in children and adolescents with Asperger syndrome. *Psychiatry Research* 197(1): 85–89.

South M, Ozonoff S and William M (2007) The relationship between executive functioning, central coherence, and repetitive behaviors in the high-functioning autism spectrum. *Autism* 11(5): 437–451.

Szatmari P, Georgiades S, Bryson S, et al. (2006) Investigating the structure of the restricted, repetitive behaviours and interest domain of autism. *Journal of Child Psychology and Psychiatry and Allied Disciplines* 47(6): 582–590.

Tager-Flusberg H (1999) A psychological approach to understanding the social and language impairments in autism. *Review in Psychiatry* 11(4): 325–334.

Teunisse JP, Cools AR, van Spaendonck KPM, et al. (2001) Cognitive styles in high-functioning adolescents with autistic disorder. *Journal of Autism and Developmental Disorders* 31(1): 55–66.

Turner M (1995) *Repetitive behaviour and cognitive functioning in autism*. Unpublished PhD thesis, University of Cambridge, UK.

Turner M (1997) Towards an executive dysfunction account of repetitive behaviour in autism. In: Russell J (ed.) *Autism as an Executive Disorder*. Oxford, NY: Oxford University Press, pp. 57–100.

White SJ (2013) The triple I hypothesis: taking another('s) perspective on executive dysfunction in autism. *Journal of Autism and Developmental Disorders* 43(1): 114–121.

White SJ and Saldana D (2011) Performance of children with autism on the embedded figures test: a closer look at a popular task. *Journal of Autism and Developmental Disorders* 41(11): 1565–1572.

White SJ, Hill E, Happé F, et al. (2009) Revisiting the strange stories: revealing mentalizing impairments in autism. *Child Development* 80(4): 1097–1117.

Williams DM and Happé F (2009) What did I say? Versus what did I think? Attributing false beliefs to self amongst children with and without autism. *Journal of Autism and Developmental Disorders* 39(6): 865–873.

Williams DM and Jarrold C (2013) Assessing planning and set-shifting abilities in autism: are experimenter-administered and computerised versions of the tasks equivalent? *Autism Research*. Epub ahead of print 26 July 2013. DOI: 10.1002/aur.1311.

Wing L and Gould J (1979) Severe impairments of social interaction and associated abnormalities in children: epidemiology and classification. *Journal of Autism and Developmental Disorders* 9(1): 11–29.

World Health Organization (1992) *The ICD-10 Classifications of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines*. Geneva: World Health Organization.

Yerys BE, Wallace GL, Harrison B, et al. (2009) Set-shifting in children with autism spectrum disorders: reversal shifting deficits on the Intradimensional/Extradimensional Shift Test correlate with repetitive behaviors. *Autism* 13(5): 523–538.

Zandt F, Prior M and Kyrios M (2009) Similarities and differences between children and adolescents with autism spectrum disorder and those with obsessive compulsive disorder: executive functioning and repetitive behaviour. *Autism* 13(1): 43–57.

Zelazo PD, Jacques S, Burack JA, et al. (2002) The relation between Theory of Mind and rule use: evidence from persons with autism-spectrum disorders. *Infant and Child Development* 11(2): 171–195.