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.

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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). Concentrating more on the non-social aspects of ASD, 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 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...
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<td>ToM</td>
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<tr>
<td>Ames and White (2011)</td>
<td>55 ASD (9 autism, 30 AS, 16 ASD-PDD-NOS); 48 M, 7 F, CA 10, VIQ 105, PIQ 94</td>
<td>Prior clinician diagnosis</td>
<td>–</td>
<td>ToM Battery, Hylineing Sentence Completion Test (to test inhibitory control)</td>
<td>3Di</td>
<td>Low ASD-social impairment group &gt; high ASD-social impairment group on inhibitory control and ToM tasks (d = 0.55). ToM predicts social interaction in ASD (R² = 0.08), but not in ADHD group.</td>
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<td>Frith et al. (1994)</td>
<td>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</td>
<td>DSM-III-R criteria</td>
<td>–</td>
<td>2 FB tasks (groups divided into passers and failures)</td>
<td>VABS</td>
<td>ASD ToM-passers &gt; ToM-failures on VABS Communication (d = 0.63) and Socialisation (d = 0.54) domain scores.</td>
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<td>Lemer et al. (2011)</td>
<td>30 ASD; 24 M, 6 F, CA 14</td>
<td>Prior clinician diagnosis plus SCQ and SRS</td>
<td>–</td>
<td>ToM Inventory</td>
<td>SCQ, Social Skills Rating System–Parent, SRS</td>
<td>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 (R² = 0.66).</td>
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<td>Lother et al. (2010)</td>
<td>20 ASD (HFA or AS); all M, CA 12, PIQ 108, VIQ 107, PIQ 107 18 TD; all M, CA 11, PIQ 109, VIQ 109, PIQ 112</td>
<td>Prior clinical diagnosis</td>
<td>Groups matched on CA, VIQ, PIQ</td>
<td>ToM: FB task, Strange Stories CC: EFT, Block Design, Sentence Completion Task</td>
<td>Childhood AS Test</td>
<td>No correlation between ToM or CC tasks and ASD symptoms (rs &lt; 0.27).</td>
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<td>Shimoni et al. (2012)</td>
<td>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</td>
<td>ADOS-G</td>
<td>Age, education and income of parents</td>
<td>ToM: Social Attribution Task</td>
<td>Children: ADOS-G, ADI-R, VABS; Parents: ADI-R, VABS–Expanded Edition</td>
<td>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)</td>
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<td>White et al. (2009)</td>
<td>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</td>
<td>Prior diagnosis, confirmed with 3Di</td>
<td>Matched gender, CA, VIQ, PIQ</td>
<td>Standard ToM battery; 11 FB tasks and Penny-Hiding Task, Strange Stories</td>
<td>3Di</td>
<td>ASD children with poor ToM had more severe social (d = 0.83) and communication (d = 0.69) symptoms, but not repetitive behaviours.</td>
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<td>EF</td>
<td>Aldar et al. (2013)</td>
<td>ADI-R, ADOS-G</td>
<td>–</td>
<td>Delis–Kaplan EF System, Developmental Neuropsychological Assessment, BRIEF</td>
<td>ADI-R, ADOS-G</td>
<td>Symptom severity does not predict inhibition, working memory or organisation score. Both PIQ and severity of ASD symptoms predicts EF shift score (R² = 0.33)</td>
</tr>
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<td>Bishop and Norbury (2005)</td>
<td>14 HFA; PIQ 107 17 SLT; PIQ 99</td>
<td>SCQ and ADOS-G</td>
<td>PIQ</td>
<td>Two subtests from Test of Everyday Attention for Children (to measure inhibition)</td>
<td>ADOS, SCQ, Children’s Communication Checklist</td>
<td>No correlation between inhibition and symptom measures of ASD (rs &lt; 0.14)</td>
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<tr>
<td>Boyd et al. (2019)</td>
<td>61 HFA (31 autism, 22 AS, 5 PDD-NOS); CA 10, IQ 100 64 TD; CA 12, IQ 111</td>
<td>DSM-IV criteria, ADI-R, SCQ</td>
<td>–</td>
<td>BRIEF</td>
<td>RBS-R, Sensory Questionnaire</td>
<td>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 (R² = 0.86)</td>
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<td>D'Cruz et al. (2013)</td>
<td>41 ASD (22 autism, 12 PDD-NOS, 7 AS); 34 M, 7 F, CA 15, FIQ 104, PIQ 105</td>
<td>ADI-R, ADOS, ADI-R</td>
<td>CA, gender, IQ</td>
<td>Probabilistic Reversal Learning Task</td>
<td>ADI-R, RBS-R</td>
<td>Positive correlation between poor flexible behaviour and RBS-R total score ($r = 0.34$), ADI RRB score ($r = 0.37$) and stereotyped, repetitive or idiosyncratic behaviour (ADI-R B3 subscale: $r = 0.38$).</td>
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<td>Dichter et al. (2009)</td>
<td>50 ASD; CA 10 IQ 102</td>
<td>DSMIV criteria, ADI-R, SRS</td>
<td>CA</td>
<td>Generativity tasks; Animals Fluency Task, The Use of Objects Task</td>
<td>ADI-R, SCQ, SRS, RBS-R, Children's Communication Checklist</td>
<td>Only correlations between communication impairments and animal fluency task scores ($r = 0.42-0.46$). No correlation between generativity and repetitive behaviours ($r &lt; 0.30$).</td>
</tr>
<tr>
<td>Gilroy et al. (2002)</td>
<td>35 ASD (HPA or autism); 30 M, 5 F, CA 10, IQ 104</td>
<td>DSMIV criteria</td>
<td>–</td>
<td>BRIEF</td>
<td>VABS</td>
<td>BRIEF Initiate and Working Memory subscales negatively correlated with Communication ($r = −0.48, r = −0.52$) and Socialisation domains of VABS ($r = −0.64, r = −0.57$).</td>
</tr>
<tr>
<td>Kenworthy et al. (2009)</td>
<td>89 ASD (34 autism, 32 AS, 23 PDD-NOS); CA 10, VA 10</td>
<td>DSMIV criteria, ADI-R, ADOS</td>
<td>–</td>
<td>BRIEF, Test of Everyday Attention for Children, Tower of London, Semantic Fluency</td>
<td>ADI, ADOS</td>
<td>EF tasks predict communication symptoms (semantic fluency: $β = −0.63$; BRIEF: $β = 0.30$), social interaction symptoms (divided attention and working memory: $β = −0.44$; semantic fluency: $β = −0.60$ and RRBIs (BRIEF: $β = 0.38$), after accounting for VA and age.</td>
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<tr>
<td>LeMonda et al. (2012)</td>
<td>22 ASD; 5 F, 17 M, CA 8, PIQ 98</td>
<td>DSMIV criteria, Wing Autistic Disorder Interview Checklist</td>
<td>CA, gender, PIQ, parent education</td>
<td>Wisconsin Card Sorting Task, Mazes subtest of Wechsler Intelligence Scale for Children–Revised Edition, Stanford-Binet Matrices subtest–Fourth Edition</td>
<td>Stereotypes measure: 30-min coded video of semi-structured play</td>
<td>EF tasks predict motor stereotypes ($R = 0.33$). Lower EF scores predict higher frequencies and longer duration of stereotypes in ASD group only ($β ≥ −0.48 ≤ −0.26$).</td>
</tr>
<tr>
<td>Lopez et al. (2005)</td>
<td>17 ASD; CA 29</td>
<td>ADI-R, ADOS-G, Gilliam Autism Rating Scale</td>
<td>CA</td>
<td>Delis–Kaplan Executive Function Scale, Wisconsin Card Sorting Task</td>
<td>ADOS, ADI-R, Gilliam Autism Rating Scale, Aberrant Behavior Checklist-Community</td>
<td>Cognitive flexibility, working memory and response inhibition correlated with RRBs ($β = 0.63, r = −0.56$, $r = 0.58$, respectively). Planning and fluency not correlated with RRBs ($r = −0.09$, $r = −0.45$, respectively). Together, cognitive flexibility, working memory and response inhibition accounted for a significant proportion of variance in RRBs ($R^2 = 0.52$). Social interaction and EF correlated ($r = −0.44$), in part due to social interaction and joint attention being correlated ($r = 0.42$).</td>
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<tr>
<td>McEvoy et al. (1993)</td>
<td>17 autism; 10 M, 7 F, CA 5, NVA 12, VA 14</td>
<td>DSMIV criteria, Childhood Autism Rating Scale</td>
<td>Autism and DD: NVA, CA Autism and TD: VA</td>
<td>Piagetian Anot-B Error Task, Delayed Response Task, Spatial Reversal Task, Alternation Task</td>
<td>Early Social Communication Scales</td>
<td>Social interaction and EF correlated ($r = −0.44$), in part due to social interaction and joint attention being correlated ($r = 0.42$).</td>
</tr>
<tr>
<td>Mosconi et al. (2009)</td>
<td>18 ASD (13 autism, 5 AS); 14 M, 4 F, CA 18, IQ 110, PIQ 107, PIQ 105</td>
<td>DSMIV criteria, ADI-R, ADOS-G</td>
<td>CA, IQ</td>
<td>Visually guided Saccade Task</td>
<td>ADI-R</td>
<td>Impairments in inhibitory control and higher order RRBs positively correlated ($r = 0.65$), after controlling for age (partial $r = 0.73$).</td>
</tr>
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<td>Reed et al. (2013)</td>
<td>15 ASD; CA 8, PIQ 71, MA 6</td>
<td>DSMIV criteria, plus Gilliam Autism Rating Scale</td>
<td>Matched ASD MA to TD CA</td>
<td>Card Sort Task</td>
<td>Gilliam Autism Rating Scale</td>
<td>Perseverative errors correlated to stereotyped behaviours ($r = 0.69$). Perseverative errors not correlated to communication difficulties or social interactions ($r &lt; 0.26$). No correlations between EF tasks and social measures (social IQ $r = −0.31$, social competence $r = −0.03$, ASD symptoms $r = 0.22$).</td>
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<tr>
<td>Teunisse et al. (2001)</td>
<td>35 HFA; 26 M, 9 F, CA 19, PIQ 91</td>
<td>DSMIV criteria</td>
<td>–</td>
<td>Card Sorting Tests, CANTAB ID/ED, Switch-in-series</td>
<td>Checklist of 12 DSM-IV diagnostic criteria, Wechsler Adult Intelligence Scale–Picture Arrangement, VABS Socialisation Domain</td>
<td>ID/ED and RRB domain of ADI-R correlated ($r = 0.43$, PIQ partialed out). ID/ED and social or communication domain scores not correlated (social symptoms $r = 0.19$; communication symptoms $r = 0.20$).</td>
</tr>
<tr>
<td>Yerys et al. (2009)</td>
<td>42 ASD (35 HFA, 7 PDD-NOS); 33 M, 9 F, CA 10, PIQ 112</td>
<td>DSMIV-TR criteria, ADOS-G, ADI-R</td>
<td>–</td>
<td>ID/ED</td>
<td>ID/ED and RRB domain of ADI-R correlated ($r = 0.43$, PIQ partialed out). ID/ED and social or communication domain scores not correlated (social symptoms $r = 0.19$; communication symptoms $r = 0.20$).</td>
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<td>Zandt et al. (2009)</td>
<td>19 ASD (15 AS, 2 autism, 1 PDD-NOS); 16 M, 3 F, CA 11</td>
<td>DSM-IV-TR</td>
<td>CA, VIQ, PIQ</td>
<td>Verbal Fluency Task, Concept Generation Task–Child Version, Rey Figure, Walk Don’t Walk Task, BRIEF</td>
<td>Repetitive Behaviour Questionnaire, Children’s Yale-Brown Obsessive Compulsive Scale</td>
<td>Impairment in EF related to higher rates of RRBIs ($r = -0.54$)</td>
</tr>
<tr>
<td></td>
<td>17 OCD; 8 M, 9 F, CA 12, VIQ 94, PIQ 96 18 TD; 6 M, 12 F, CA 12, VIQ 95, PIQ 103</td>
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<tr>
<td>CC</td>
<td>Burnette et al. (2005)</td>
<td></td>
<td>DSM-IV</td>
<td>VIQ, PIQ</td>
<td>HFA Spectrum Screening Questionnaire, Australian Scale for AS</td>
<td>No correlations between CC tasks and ASD symptoms (no statistics stated)</td>
</tr>
<tr>
<td>Time 1:</td>
<td>31 HFA; 26 M, 5 F, CA 11</td>
<td></td>
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<tr>
<td>16 TD; CA 11</td>
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<tr>
<td>Time 2 (15–19 months)</td>
<td>23 HFA; 19 M, 4 F, VIQ</td>
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<tr>
<td></td>
<td>10 PIQ 110 20 TD (12 TD + 6 LD + 2 new LD); 15 M, 5 F, CA 11, VIQ 117, PIQ 117</td>
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<tr>
<td>Chen et al. (2009)</td>
<td>29 ASD; 26 M, 3 F, CA 12, IQ 114</td>
<td>Clinical diagnosis, ADOS, SCQ</td>
<td>–</td>
<td>EFT</td>
<td>Childhood Routines Inventory, Short Sensory Profile</td>
<td>Degree of RRBIs ($r = -0.39$), but not sensory abnormalities ($r = -0.02$), predict completion time on the EFT</td>
</tr>
<tr>
<td>Drai et al. (2010)</td>
<td>27 TD; 9 M, 18 F, CA 10</td>
<td>–</td>
<td>–</td>
<td>Block Design, EFT, Copying Task</td>
<td>Childhood AS Test</td>
<td>Performance on CC tasks not related to RRBIs ($r &lt; 0.27$)</td>
</tr>
<tr>
<td>Looth et al. (2010)</td>
<td>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</td>
<td>Prior clinical diagnosis</td>
<td>CA, VIQ, PIQ</td>
<td>EFT, Block Design, Sentence Comletion Task</td>
<td>Childhood AS Test</td>
<td>No correlation between CC tasks and ASD symptoms ($r &lt; 0.27$)</td>
</tr>
<tr>
<td>Morgan et al. (2003)</td>
<td>21 ASD (19 autism, 2 PDD-NOS); 19 M, 2 F, CA 3, MA 3, PIQ 95, VIQ 77 21 TD; 16 M, 5 F, CA 5, MA 5, PIQ 105, VIQ 101</td>
<td>DSM-IV criteria</td>
<td>Gender, CA, PIQ</td>
<td>Preschool EFT, Differential Ability Scales – Pattern Construction</td>
<td>ADI-R, ADOS</td>
<td>CC predicts ASD group membership ($d = 1.86$)</td>
</tr>
<tr>
<td>Russell-Smith et al. (2012)</td>
<td>80 TD; CA 19, 56 F</td>
<td>–</td>
<td>Age and gender</td>
<td>EFT (group divided by AQ ‘social skills’ and ‘details/ patterns’ (either high or low)</td>
<td>Autism Spectrum Quotient</td>
<td>EFT scores: high social difficulty &gt; low social difficulty ($d = 0.59$), High details/patterns = low details/patterns ($d = 0.17$)</td>
</tr>
<tr>
<td>South et al. (2007)</td>
<td>19 ASD; 14 M, 5 F, CA 15, FIQ 115, PIQ 111, FIQ 114 18 TD; 11 M, 7 F, VIQ 112, PIQ 113, RIQ 112</td>
<td>DSM-IV criteria, ADI-R, ADOS-G</td>
<td>CA, VIQ, PIQ, FIQ</td>
<td>EFT, Geriatric closure test ADOS, ADI-R, Repetitive Behaviour Interview, Yale Special Interests Interview</td>
<td>No correlations between CC tasks and RRBi measures ($r &lt; 0.30$)</td>
<td></td>
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<tr>
<td>Teunisse et al. (2001)</td>
<td>35 HFA; 26 M, 9 F, CA 19, VIQ 91</td>
<td>DSM-IV criteria</td>
<td>–</td>
<td>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</td>
<td>Checklist of 12 DSM-IV diagnostic criteria, Wechsler Adult Intelligence Scale–Picture Arrangement, VABS Socialisation Domain</td>
<td>No correlations between CC tasks and social measures (social IQ $r = 0.00$, social competence $r = 0.16$, autistic symptoms $r = 0.11$)</td>
</tr>
<tr>
<td>White and Saldana (2011)</td>
<td>45 ASD; 41 M, 4 F, CA 9, VIQ 111, PIQ 98 27 TD; 21 M, 6 F, CA 9, VIQ 115, PIQ 103</td>
<td>Prior diagnosis ICD-10 criteria, confirmed with 3Di</td>
<td>Gender, CA, VIQ, PIQ</td>
<td>Children’s EFT</td>
<td>3Di</td>
<td>No correlations between EFT and ASD symptomatology ($r &lt; 0.22$)</td>
</tr>
</tbody>
</table>
Table 1. (Continued)

<table>
<thead>
<tr>
<th>Reference</th>
<th>Participants (group means)</th>
<th>ASD diagnosis</th>
<th>Control variables</th>
<th>Cognitive tasks</th>
<th>Symptom measures</th>
<th>Relevant findings (with effect sizes)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Multiple cognitive deficits</strong></td>
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<tr>
<td>Joseph and Tager-Rusberg (2004)</td>
<td>31 ASD (27 autism, 4 ASD); CA 9, VIQ 20, PIQ 23</td>
<td>DSM-IV criteria, ADI-R, ADOS</td>
<td>–</td>
<td>5 EF tasks; Word Span, Block Span, Day–Night, Knock–Tap, Tower</td>
<td>ADOS</td>
<td>ToM correlated with communication ($r = -0.64$), but not social symptoms or RRBIs when non-verbal MA and language controlled for ($r &lt; 0.34$). Language ($R^2 = 0.33$). ToMability ($R^2 = 0.28$) 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.</td>
</tr>
<tr>
<td>Pellicano et al. (2006)</td>
<td>40 ASD (30 autism, 10 PDD-NOS); 35 M, 5 F, CA 5.6, VIQ 101, PIQ 114</td>
<td>DSM-IV criteria, confirmed with ADI-R; TD screened with SCQ</td>
<td>CA, VIQ, PIQ, gender</td>
<td>CC: EFT, Pattern-Construction task, Figure-Ground task, Developmental Test of Visual–Motor Integration</td>
<td>ADI-R</td>
<td>All cognitive tasks failed to correlate with either ADI-R total or domain scores. EFT and social domain (at 4–5 years) negatively correlated ($r = 0.41$).</td>
</tr>
<tr>
<td>Pellicano (2013)</td>
<td>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</td>
<td>DSM-IV criteria, Time 1 with ADI-R; TD</td>
<td>Age, VIQ, PIQ (at Time 1)</td>
<td>Time 1: ToM; 2 first-order FB tasks, 1 second-order FB task EF; Luria Hand Game, Mazes task, Teddy-bear set-shifting task, Luria Hand Game, Mazes task CC: EFT, Pattern-Construction task, Figure-Ground task</td>
<td>Time 2: ADOS-G, Repetitive Behaviour Questionnaire</td>
<td>ToM negatively correlated with social communication ($r = -0.42$). EF negatively correlated with social communication and RRBIs (both $r = 0.42$). CC not correlated ($r &lt; 0.21$). EF predicts symptom severity (ADOS; $R^2 = 0.16$) and repetitive behaviours ($R^2 = 0.15$).</td>
</tr>
</tbody>
</table>

**Note:** 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; Cohen's d: developmental delay; DDD: Developmental 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; ICID-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: obsessional compulsive disorder; PDD-NOS: pervasive developmental disorder–not otherwise specified; PIQ: performance intelligence quotient; PLE: 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 RRBIs 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 RRBIs 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 RRBIs 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.

**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.

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References


