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Published in:
Autism Research

DOI:
10.1002/aur.1630

Publication date:
2016

Document version
Accepted manuscript

Document license
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Citation for published version (APA):

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Download date: 02. Nov. 2019
Exploring ‘The Autisms’ at a Cognitive Level

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Grant information

This study was supported by Grant sponsor: The Psychiatric Research Foundation in the Region of Southern Denmark, Grant sponsor: The PhD grant in Region of Southern Denmark and Grant sponsor: Sofiefonden
Lay abstract (229/250 words)

The autism spectrum is full of variation both in terms of the range of symptoms and differences between individuals. Even so, it is possible that a single cause might be present underneath this diversity in all individuals with autism spectrum disorders (ASD). This study focussed on three well-known cognitive differences that are thought to affect the way people with ASD process information: Theory of Mind (ToM), Executive Function (EF) and a Local Processing Bias (LB). Thirty-one high-functioning children with ASD and thirty-seven children with neurotypical development of similar age, gender and intelligence completed several ToM, EF and LB tasks. Everyday behaviours were also assessed through parent and teacher questionnaires, parent interview and direct observation. We found that ToM and EF difficulties were common and performance on these tasks could be used to accurately divide most of the children into those with and without ASD. Performance on ToM tasks was related to performance on EF tasks but neither related to any of the everyday behaviours. Only a small group of individuals with ASD had a LB, which did not relate to the other measures; a LB may be the cause of symptoms that were not included in an ASD diagnosis. Future studies may reinforce the idea that there is a single cause of ASD in all affected individuals.
Scientific abstract

The autism spectrum is characterised by genetic and behavioural heterogeneity. However, it is still unknown whether there is a universal pattern of cognitive impairment in autism spectrum disorder (ASD) and whether multiple cognitive impairments are needed to explain the full range of behavioural symptoms. This study aimed to determine whether three widely acknowledged cognitive abnormalities (Theory of Mind (ToM) impairment, Executive Function (EF) impairment and the presence of a Local Processing Bias (LB)) are universal and fractionable in autism, and whether the relationship between cognition and behaviour is dependent on the method of behavioural assessment. Thirty-one high-functioning children with ASD and thirty-seven children with neurotypical development (NTD), comparable in age, gender and IQ, completed several tasks tapping into ToM, EF and LB, and autistic symptomatology was assessed through parental and teacher questionnaires, parental interview and direct observation. We found that ToM and EF deficits differentiated the groups and some ToM and EF tasks were related to each other. ToM and EF were together able to correctly classify more than three-quarters of the children into cases and controls, despite relating to none of the specific behavioural measures. Only a small subgroup of individuals displayed a LB, which was unrelated to ToM and EF, and did not aid diagnostic classification, most likely contributing to non-diagnostic symptoms in a subgroup. Despite the characteristic heterogeneity of the autism spectrum, it remains a possibility therefore that a single cognitive cause may underlie the range of diagnostic symptoms in all individuals with autism.

Keywords: Autism Spectrum Disorders, Cognition, Theory of Mind, Executive Function, Local Bias, Fractionation, Symptomatology, and Behaviour
Exploring ‘The Autisms’ at a Cognitive Level

Autism spectrum disorder (ASD) is now widely accepted as a neurodevelopmental disorder with a genetic basis resulting in atypical brain development, although relatively little is known about the exact genetic or biological abnormalities underlying the disorder (Betancur, 2011; Gliga, Jones, Bedford, Charman, & Johnson, 2014; Goldani, Downs, Widjaja, Lawton, & Hendren, 2014; Happé & Ronald, 2008; Lai, Lombardo, & Baron-Cohen, 2013). An ASD diagnosis therefore relies upon a defined set of behavioural criteria, encompassing social interaction and communication difficulties and the presence of repetitive behaviours (DSM-5, American Psychiatric Association, 2013). A behavioural diagnosis has the disadvantage of heterogeneity: there can be many different causes of the same behaviour, or equally, different behaviours in different individuals can result from the same underlying cause due to interaction with other factors (Rutter, 2000). Indeed, the shopping list-style diagnostic criteria (Morton, 2008) expect and furthermore embrace behavioural heterogeneity, leading the term ‘the autisms’ (Geschwind & Levitt, 2007) to enter the literature.

While it is acknowledged that different individuals vary wildly in their personal presentation of this shared diagnostic label (Geschwind, 2009; Munson, Faja, Meltzoff, Abbott, & Dawson, 2008; Ronald, Happé, Price, Baron-Cohen, & Plomin, 2006), this same heterogeneity is much harder to reconcile at a causal level of explanation. Still, heterogeneity is clearly evident at the genetic level (Abrahams & Geschwind, 2008; O’Roak et al., 2012), leading the field to pursue endophenotypic markers that can homogeneously draw subgroups of individuals together (Charman et al., 2007) and heterogeneity certainly appears to decrease as we travel down the causal chain from genetics to cognition. There is convergence from genetics to neurobiology (Geschwind & Levitt, 2007; Zoghbi, 2003) and similarly from cellular to systems neuroscience (Amaral, Schumann, & Nordahl, 2008). Whilst theories
abound at the cognitive level, Theory of Mind (ToM), executive function (EF) and a local processing bias (LB) remain the most prominent (Brunsdon & Happé, 2014; Frith, 2012; Rajendran & Mitchell, 2007) drawing together diverse biological and behavioural findings. The ToM account of ASD, a difficulty representing the mental states of others, was proposed by Baron-Cohen, Leslie, and Frith (1985) as a cognitive explanation for the socio-communicative impairment. The EF account originally aimed to explain the repetitive and stereotyped behaviours through a lack of higher order control processes such as planning, flexibility and inhibition (Ozonoff, Pennington, & Rogers, 1991), but has since also attempted to account for the socio-communicative symptoms (Bishop & Norbury, 2005a). The third account, LB, aims to explain why individuals with ASD have trouble integrating information but also show “islets of ability”, through a tendency to process local details at the expense of global meaning (Frith, 1989; Happé & Frith, 2006).

It has thus been suggested that there may not be a unitary underlying cause of autism at any level and that leaving the “single explanation” approach behind may be the key to identifying the genetic and neurocognitive origins of autism (Happé, Ronald, & Plomin, 2006); it seems likely that a number of different mechanisms are required to explain different aspects of autistic symptomatology (Happé & Ronald, 2008). While such fractionation is evident at the behavioural level, it is still unclear whether the different proposed cognitive impairments in autism are similarly separable (Brunsdon & Happé, 2014); very few studies have examined ToM, EF and LB simultaneously in ASD and these findings have been contradictory (Brunsdon et al., 2014; Lai et al., 2012; Lam, 2013; Pellicano, Maybery, Durkin, & Maley, 2006). Furthermore, only one of these studies (Pellicano et al., 2006) has examined the relationship between cognition and symptomatology, finding no associations, and hence it is unknown whether these cognitive impairments really can explain independent aspects of autistic behaviour; it is conceivable that the tasks used to tap into these cognitive
difficulties may instead measure constructs related to ASD but independent of the core symptoms, such as language or intelligence. One as yet unexplored possibility is that the detection of relationships between cognition and symptomatology may be dependent on the tool used to evaluate diagnostic symptoms, whether assessed through parent or teacher report, through interview or direct observation. The present study aims to provide further evidence to inform the fractionation debate at the cognitive and behavioural levels.

The idea of fractionating autism addresses the proposal that there is no mechanism at any level of causality that is sufficient to explain the totality of the autistic syndrome. Inter-individual heterogeneity addresses a subtly different issue: that there is no causal mechanism common to all individuals, a notion that is also widely presumed to be true but which has received much less empirical attention (Brunsdon & Happé, 2014). In fact, inter-individual heterogeneity has even been suggested to be a more distinct marker for autism than any one neuropathology (Towgood, Meuwese, Gilbert, Turner, & Burgess, 2009), attempting to explain why one study may find support for and the next find no evidence in favour of a particular underlying deficit. It remains an open question whether there is a universal pattern of impairment that can draw together all individuals with autism, or whether there is truth in the term ‘the autisms’ not just at a behavioural level but also at a causal level of explanation. The present study aims to address this issue.

This study therefore attempts to shed light on the following:

1) Are the cognitive impairments ToM, EF and LB fractionable in ASD?
2) Are any of these cognitive impairments common to all individuals with ASD?
3) Do these cognitive impairments predict autistic symptomatology?
4) Is the relationship between cognition and behaviour dependent on the method of behavioural assessment?

We approached these questions by examining all three cognitive domains (ToM, EF
and LB) in a group of high-functioning children with ASD as well as a group of children with neurotypical development (NTD), comparable in age, gender and IQ. We used several tasks within each domain to ensure the validity of the cognitive measures and, given the propensity in the literature for high-functioning individuals with ASD to pass such tests, we selected tasks that have previously been found to be sensitive. We planned to study group differences to identify impairments relevant to autism, as well as individual performance by identifying outliers in each cognitive domain to tackle the issue of universality. Calculating correlations between these variables would allow us to address the question of fractionation, as would patterns of impairment in each individual. In addition, the children were comprehensively assessed with widely used instruments of autistic symptomatology, including parental and teacher questionnaires, parental interview and direct observation, enabling us to investigate whether cognitive task performance could predict the behavioural symptoms of ASD and whether this was dependent on the method of behavioural assessment.

**Method**

The study was approved by the Ethical Committee, Region of Southern Denmark (S-20090071).

**Participants**

The ASD group was recruited from two Child and Adolescent Mental Health Services in the Region of Southern Denmark by searching the Patient Administrative System for date of birth (8-12 year olds) and ICD-10 diagnosis of Pervasive Developmental Disorder (F84.0-84.9). The diagnostic files were reviewed and children who did not fulfil an ASD-diagnosis or had a full-scale IQ (FSIQ) below 70 were excluded. A total of 82 clinically diagnosed children with ASD were invited to participate in the study, of which 54 families responded, from which 37 children agreed to participate. Three additional cases were recruited from special education schools for children with ASD.
Participants in the ASD group were only included if they also met DSM-IV-TR criteria for an ASD at the time of the assessment. An individual clinical conference of trained clinicians included all previous diagnostic information in conjunction with current scores on the ADOS and ADI-R (see below), and formed the basis for confirmation of diagnosis. At this stage, 11 children met the criteria for Autistic Disorder, 7 for Asperger’s Syndrome and 17 for Pervasive Developmental Disorder – Not Otherwise Specified (PDD-NOS). As the study aims were relevant to the whole autism spectrum, all of these children were included. The remaining 5 children no longer fulfilled diagnostic criteria for an ASD and were excluded. Four further children were excluded due to current FSIQ below 70 (estimated from three verbal and three performance subtests from the Danish translation of the Wechsler Intelligence Scale of Children, WISC-III, Wechsler, 2003) leaving a total of 31 for analyses.

The NTD children (N=37) were recruited from four mainstream schools using similar inclusion criteria (FSIQ>70, age: 8-12 years). None of the NTD participants had elevated scores on the SRS or SCQ questionnaires (see below) or were reported to have any developmental disorder or family history of such difficulties.

The groups did not significantly differ on gender ($\chi^2(1)=0.97$), age ($t(63)=0.32$), performance IQ ($t(66)=0.60$), verbal IQ ($t(64)=1.48$) or full-scale IQ ($t(66)=1.48$). The majority of children were Caucasian and from middle-class families, and we found no significant difference in their parents’ educational level ($\chi^2(1)=.29$), defined by the highest-ranking parent’s education (more/less than 13 years education; see table 1).

**Behavioural assessment of symptoms**

For the ASD group, symptomatology was measured using the Autism Diagnostic Observation Schedule-Generic (ADOS, Lord et al., 2000) and Autism Diagnostic Interview, Revised (ADI-R, Lord, Rutter, & Le Couteur, 1994) to observe the child’s behaviour in a clinical setting and assess parents’ perception of their child’s disabilities (see table 1). All
children reached the cut-off in ADOS social interaction, but seven fell below cut-off in communication, and six did not reach the total cut-off.

Symptomatology scores were recorded for both groups from parents and teachers using two questionnaires: the Social Responsiveness Scale (SRS, Constantino et al., 2003) and the Social Communication Questionnaire (SCQ, formerly the Autism Screening Questionnaire, Berument, Rutter, Lord, Pickles, & Bailey, 1999). Significant group differences were found on both questionnaires for parents (SRS, t(35)=11.734, p<0.001; SCQ, t(36)=9.926, p<0.001) and teachers (SRS, t(42)=9.570, p<0.001; SCQ, t(43)=4.710, p<0.001). Interestingly, parents in the ASD group tended to rate their children as having significantly more symptoms than the teachers did (SRS, t(27)=2.671, p=0.013; SCQ, t(26)=2.076, p=0.048), whereas the opposite was true for the NTD group on the SCQ (t(22)=3.976, p=0.001; no difference on SRS, t(25)=0.609).

Table 1 about here

**Instruments**

We assessed the cognitive domains, ToM, EF and LB, with a battery containing multiple cognitive tasks within each domain to ensure the validity of the cognitive measures. These tasks, the outcome measures and references to the procedures used are shown in table 2.

For the ToM tasks, excellent agreement was reached (intraclass correlation coefficients: Strange Stories, 0.989; Frith-Happé Animations, 0.946) between the first author and a corater blind to group (20% of responses randomly selected).

Table 2 about here

**Results**

As IQ and age varied greatly in these samples and both variables correlated to the majority of task measures, we chose to calculate individual performance levels for each task
independent of IQ and age. We entered data from the NTD group as the dependent variable in a regression with FSIQ and age as the predictor variables, the resulting regression equation was applied to the ASD group, and residuals were collected for both groups. These were converted to z-scores in relation to the NTD group’s mean and standard deviation and used in all further analyses. To detect individuals in the ASD group with deviant performance on each measure, any NTD group outliers performing more than 1.65 standard deviations (SDs) below the NTD group mean were first removed in order to obtain a better estimate of normal performance, regardless of NTD children who might have performed abnormally on any one task. Deviant performance was defined as below the 5th centile (1.65 SD) of this corrected NTD group performance (White et al., 2006).

**Theory of Mind (ToM)**

The ASD group performed significantly worse than the NTD group on the Strange Stories assessing mental state inferences (\(t(44)=3.10, p=0.003\)). We did not find a significant difference between the groups on any other story type (\(ps>0.08\)). For the Frith-Happé Animations, we found significant group differences in both the appropriateness and intentionality of answers in the ToM condition (\(t(50.7)=4.63\) and \(t(51.3)=4.28, ps<0.001\)). The ASD group’s answers were also less appropriate in the GD condition (\(t(66)=3.39, p=0.001\)).

We found positive correlations between the mental state Strange Stories and both of the ToM scores from the Frith-Happé Animations (\(r=0.326, p=0.007\) & \(r=0.272, p=0.025\)), although these did not hold in the groups separately. Nevertheless, given both tasks were designed to tap into the same theoretical construct, we combined each individual’s z-scores on these ToM measures to create a total ToM score, first averaging the two Frith-Happé Animation ToM scores before averaging them with the Strange Stories mental state score. This ToM total score also revealed a significant group difference (\(t(43.2)=5.253, p<0.001\))
with the ASD group performing on average 2SDs below the NTD group mean (see figure 1). Scores can be seen to span the whole range of the NTD group performance but with an elongated tail of individuals performing particularly poorly; indeed, 58% of children in the ASD group fell below the 5th centile cut-off.

Figure 1 about here

**Executive function (EF)**

For generativity we found a significant group difference on Verbal Fluency in both conditions (letter, \(t(65.7) = 2.935, p = 0.005\); category, \(t(54.7) = 2.358, p = 0.022\)), and on Pattern Meanings in the total number of correct responses (\(t(63.3) = 4.855, p < 0.001\)), with the ASD group performing more poorly. On the CANTAB tests, we found no significant group difference on any of the sub-scores in the SOC task (min. moves, \(t(66) = 1.107, p = 0.272\); initial thinking, \(t(66) = 1.118, p = 0.268\); subsequent thinking, \(t(66) = 0.060, p = 0.952\)), in the SSP task (\(t(66) = 0.252, p = 0.802\)), in the SWM task (number of errors in each condition, \(t(66) < 0.983, ps > 0.33\); strategy used, \(t(66) = 0.691, p = 0.492\)) or in the IED task (extra dimensional shift, \(t(66) = 1.712, p = 0.092\)).

We found significant correlations between the letter condition in Verbal Fluency and the Pattern Meanings task (\(r = 0.428, p < 0.001\)), which held in the NTD (\(r = 0.338, p = 0.041\)) but not the ASD group (\(r = 0.287, p = 0.117\)). A total generativity score was calculated as the mean of Verbal fluency (mean of both conditions) and Pattern Meanings. Within the CANTAB, the SSP and the SWM tasks were correlated (\(r = 0.293, p = 0.015\)), which held in the ASD (\(r = 0.517, p = 0.003\)) but not the NTD group (\(r = 0.088, p = 0.606\)). A total mean CANTAB score was calculated, where each task was weighted equally. The total CANTAB and total generativity scores were then averaged to provide an EF total score, given EF is widely acknowledged to encompass a disparate range of functions with the common purpose of higher-order control (e.g. Castellanos, Sonuga-Barke, Milham, & Tannock, 2006).
The ASD group performed on average just less than 1SD below the NTD group on this EF total score \( t(66)=3.856, p<0.001 \); see figure 1). The overall distribution was quite striking; there was significant overlap in the range of scores in the two groups. While 29% of children fell below the 5th percentile cut-off, the majority (52%) fell between this cut-off and the NTD group mean.

**Local Bias (LB)**

There was a non-significant tendency for children in the ASD group to perform slightly better than the NTD group on the EFT \( t=1.924, p=0.059 \). In the HVOT, we did not find a significant group difference on the time taken to complete each correct trial \( t=1.585, p=0.118 \) or on the number of correct answers \( t=1.567, p=0.122 \). Although there were no correlations between the two LB tasks, they were designed to tap into different aspects of the same construct and so were combined to give a LB total score, which did not differ significantly between the groups \( t=0.094, p=0.925 \). Low z-scores indicated a local bias (high EFT and low HVOT). Only 13% of children in the ASD group showed a profile indicative of a local processing style, falling below the 5th percentile cut-off. Taking the EFT or HVOT separately gave an even smaller number of outliers (EFT: 6%, HVOT: 6%).

**Relationships between cognitive domains**

We found no correlations between the composite scores of the three cognitive domains (correlations across the whole sample unless otherwise stated). Given the lack of association between many of the elements of the composite scores, we also explored correlations between these elements across the different domains. Within ToM, performance on the Frith-Happé Animations was correlated to the EF composite \( r=0.343, p=0.004 \) and, within EF, generativity was correlated to the ToM total score \( r=0.299, p=0.013 \). These correlations seemed to be driven by an association specifically between the generativity composite and performance on the Frith-Happé Animations \( r=0.446, p<0.001 \). Specifically, analyses
showed that the Frith-Happé Animations correlated to Verbal Fluency (letter: $r=0.281$, $p=0.020$) and Pattern Meanings ($r=0.487$, $p<0.001$); this latter correlation also held in the ASD group alone $r=0.355$, $p=0.050$). Likewise, correlations were found between the generativity composite and both the intentionality score ($r=0.391$, $p=0.001$) and the appropriateness score ($r=0.450$, $p=0.001$) in the Frith-Happé animations.

Patterns of impairment in each individual were studied. While the majority of children had a ToM impairment only, Figure 2 shows that individuals exist with most possible combinations of impairment. Figure 3 shows specific examples of such combinations, portraying individual profiles across these three domains. This reveals that, although rare, the less frequent combinations of impairment are not an artefact of the cut-off methodology used; children exist with significant impairment in the affected domains whilst having retained performance in the remaining domains. Figure 2 further reveals a proportion of children with ASD who appear to have no significant cognitive impairments on the tests used here; 16% of children fell into this category although none had positive z-scores across all three cognitive domains.

Figures 2 & 3 about here

**Relationships between cognition and symptomatology**

Relationships between cognitive and behavioural variables were explored in the ASD group alone as ADOS and ADI scores were not available for the NTD group. The ToM and EF total scores were unrelated to scores on the ADI but the LB total score was negatively correlated to a subscale of the ADI communication domain (delay in spoken language without attempts to compensate through gestures, $r=0.368$, $p=0.042$), indicating that the presence of autism-related communication symptoms was associated with a local processing bias (this would not withstand correction for multiple comparisons however). Likewise, ToM and EF total scores were unrelated to ADOS scores but the LB total score was correlated with
a subscale of the ADOS repetitive behaviour domain (stereotypical behaviours, \( r=0.398, p=0.027 \)), indicating that the absence of autism-related stereotypical behaviour was associated with a local processing bias (would not withstand correction for multiple comparisons). We found no correlations between any cognitive domain and SRS scores or subscales; likewise for the SCQ.

**Predicting diagnostic group from cognitive test performance**

Wilks’ discriminant function analysis was used to investigate which cognitive factors (ToM, LB or EF) were best able to predict group membership. Variables were entered and removed in a step-wise manner. ToM was the best discriminator, correctly classifying 74% of children, and EF was found to significantly increase this discrimination to 79% \( (\chi^2(2)=36.90, \text{Wilks’ lambda}=0.57, p<0.001; \text{see Table 3}; \text{this increased to 81% when substituting the ToM Animations score and Generativity composite}). Misclassification occurred equally in both groups, indicative of false negatives and false positives. When entered alone, EF classified 67% correctly. LB was not found to significantly aid in discriminating the groups.

**Discussion**

This case control study aimed to determine whether the cognitive impairments ToM, EF and LB are fractionable, and whether any are common to all individuals with autism. Furthermore, we aimed to investigate whether these cognitive impairments predict autistic symptomatology, and whether the relationship between cognition and behaviour is dependent on the method of behavioural assessment.

We found that ToM and EF deficits differentiated children with ASD from those with neurotypical development at a group level and a proportion of individuals were characterised by each difficulty. Together, these abilities were able to correctly classify more than three-quarters of children into cases and controls. Furthermore, these cognitive impairments
appeared to be related to each other, suggesting a lack of fractionation between these domains. On the other hand, we found little support for the presence of a LB in autism, with only a small subgroup of individuals affected. Performance in this domain was unrelated to ToM and EF, indicating fractionation and that this processing style may explain specific aspects of autistic symptomatology that are present only in a small subgroup. We found no evidence that performance on ToM and EF tasks predicted autistic symptomatology regardless of the method of behavioural assessment.

**Cognitive universality**

Our ToM results at a group level are consistent with other studies of autism finding a group difference on the ToM-condition in the Frith-Happé Animations (Abell, Happé, & Frith, 2000; Castelli, Frith, Happé, & Frith, 2002; Salter, Seigal, Claxton, Lawrence, & Skuse, 2008) and in the Strange Stories vignettes that assessed mental state inference (Kaland, Callesen, Moller-Nielsen, Mortensen, & Smith, 2008; Spek, Scholte, & Van Berckelaer-Onnes, 2010; Velloso Rde, Duarte, & Schwartzman, 2013; White, Hill, Happé, & Frith, 2009), as well as with those studies assessing performance across multiple cognitive domains (Brunsdon et al., 2014; Lai et al., 2012; Lam, 2013; Pellicano et al., 2006). Despite great diversity in the tasks and methods used, ToM is consistently found to be impaired. Further, we found here that 58% of children with ASD performed within the bottom 5\textsuperscript{th} centile of neurotypical performance, and that ToM alone was able to correctly predict diagnostic status for 74% of children. This indicates that the ToM impairment may be the most frequently-occurring, well-specified and robust impairment, as well as being clinically relevant. With more sensitive tests (e.g. Senju, Southgate, White, & Frith, 2009), a ToM impairment may well be present in an even larger proportion of children with ASD. Whether this holds true across the full span of development is a matter for future investigation.
Likewise, EF impairment seems to be quite reliably identified across studies (for reviews, see: Hill, 2004; Russo et al., 2007), including in 4 out of 5 cross-domain studies (present study included). There appears to be an attenuating effect across EF tasks however: only a proportion of tasks produce group differences in each study and there is variability in which tests give rise to group differences across studies (Russo et al., 2007). Here, our significant group difference in the EF domain was largely driven by the poor performance of the ASD group on the generativity tasks (which support previous findings on generativity, Ambery, Russell, Perry, Morris, & Murphy, 2006; Bishop & Norbury, 2005b; Turner, 1999). Indeed, no group differences were seen on any CANTAB test, a finding that is not unusual (Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Goldberg et al., 2005). One possible explanation lies in the computerised administration of the CANTAB; a participant’s ability to infer the experimenter’s intentions may affect test performance in an experimenter-administered situation (see Kenworthy, Yerys, Anthony, & Wallace, 2008; White, 2013), although Williams and Jarrold (2013) have recently published evidence to the contrary. Another possibility is that the verbal nature of the generativity tasks, rather than the EF properties, posed a problem for the children with ASD. Despite this, the EF composite placed 29% of children with ASD in the bottom 5th centile of neurotypical performance, and significantly strengthened the group classification algorithm. Furthermore, the EF composite alone correctly classified 67% of children into their diagnostic groups, indicating that our 5th centile cut-off technique may have been overly conservative. An EF impairment certainly appears to be present in a substantial subgroup of individuals with autism.

The presence of a LB in autism has the weakest support both from the general autism literature (Happé & Frith, 2006) as well as from cross-domain studies (2 out of 5 studies, present study included); even when significant effects are identified, it remains unclear whether these are driven by an enhancement in local processing or a deficit in global
processing. Our finding of a non-significant trend for enhanced local processing is certainly within the range of previous results. The lack of effect on our global processing task could be due to the choice of task and instructions given; it is possible that participants were able to identify the object by looking at a single piece rather than attempting to combine the pieces. This may have been avoided by using the modified version of the task (Jolliffe & Baron-Cohen, 2001) where the combined picture cannot be interpreted from the fragments. Together, our tasks classified only 13% of children as displaying a LB. If a LB is present in only a small subsample of the autistic population, this could explain the lack of group differences often reported in this domain.

One possible limitation to the study’s ability to address the issue of universality is the inclusion criteria. With the advent of more conservative diagnostic criteria, it is acknowledged that some of the children may not have met criteria for an ASD as defined by DSM-5 (2013). This is likely to have increased heterogeneity, decreasing the probability of finding a universal cognitive impairment. We were unfortunately unable to reassess each case against DSM-5 criteria; we may well have found ToM and EF impairments and a LB in a larger proportion of children if we had been able to exclude children who did not meet DSM-5 criteria. If this proves true, assessment of cognitive atypicalities could provide an objective means of identifying ASD as an aid to current clinical practice.

**Cognitive fractionation**

Across the cognitive functions, measures were generally unrelated, most possible combinations of impairment were found in different children with ASD, and individual profiles revealed double dissociations between cognitive domains. While this paints a picture of cognitive fractionation in autism, we did find a strong relationship between generativity and ToM performance on the Frith-Happé animations; ToM and EF also classified very similar sets of children into their diagnostic groups. It seems likely that these specific ToM
and EF tests were related here either due to overlapping task demands (cf. verbal tasks and tasks requiring inference of the experimenter’s intentions) or because these two cognitive processes fundamentally rely on a common neurocognitive mechanism (e.g. predictive coding, Lawson, Rees, & Friston, 2014; Pellicano & Burr, 2012). Despite a lack of clarity as to the roots of this association, the past literature in ASD certainly supports the existence of such a relationship (Joseph & Tager-Flusberg, 2004; Ozonoff et al., 1991; Pellicano, 2007; Zelazo, 2002). Similar to our results, Pellicano (2007) found a correlation between ToM and EF and argued that EF was crucial for the development of ToM in children with ASD because impaired ToM was seen with intact EF but not vice versa. While this pattern has been interpreted as indicating developmental primacy of EF, an alternative possibility is that EF difficulties are simply less common and/or less severe and therefore have less explanatory power. Further, we found EF impairments alone in four children in the present study. Later, Pellicano (2010) supported her hypothesis by finding that EF and LB predicted change in ToM longitudinally; further such work using a variety of methodologies is desperately needed to explore this relationship in greater depth. Whatever draws these domains of ToM and EF together may prove to be a key cognitive component impaired in autism.

On the other hand, the presence of a LB certainly appears to be fractionated from the ToM and EF impairments. However, the importance of this atypical processing style appears limited given its very low prevalence.

Cognition to behaviour

To the best of our knowledge, only one study (Pellicano et al., 2006) has previously assessed correlations between multiple cognitive domains and symptomatology, and the few associations they found failed to survive correction for multiple comparisons. Even though we included multiple measures that probed behavioural symptomatology in different ways, associations between cognitive performance and behavioural symptomatology in the present
study were similarly sporadic and weak. Surprisingly, LB was the only domain that was correlated with symptomatology, being associated with less stereotypical behaviours on the ADOS and delay in spoken language without attempts to compensate through gestures on the ADI-R. These correlations were unexpected and did not survive Bonferroni correction.

This lack of association across multiple methodologies and across two studies now, raises the question of construct validity of the behavioural and cognitive test measures. While both clearly differentiate the autistic from the neurotypical group, it is possible that tests at either or both levels of representation are tapping into variance in some factor orthogonal to the one intended. Behavioural measures are intrinsically liable to the subjective opinion of the parent, teacher or experimenter, and the behaviours of interest are susceptible to being overshadowed by individual differences in intelligence, language, personality, education etc. This could be tested by including items that explicitly ask about ToM, EF and LB abilities, in order to assess whether parent and teacher reports, and indeed experimenter judgements, are a reliable proxy for cognitive test performance. Cognitive measures are likewise rarely pure measures of a single cognitive process. Recent work looking at more implicit measures (Schuwerk, Vuori, & Sodian, 2015; Senju, 2012; Sodian & Thoermer, 2008) holds promise for tapping more directly into the cognitive impairments underlying autism.

In summary, our multi-domain study of cognition in autism indicated that difficulties on ToM and EF tasks characterise the majority of cases, raising the possibility that one or both may after all prove to be universal in autism given more sensitive cognitive measures. If so, this would counter the idea of ‘the autisms’ at least at the cognitive level and indicate that the spectrum should be approached as a unitary disorder. Our results also contribute to understanding fractionation in autism: ToM and EF were related, although the exact nature of this association has yet to be determined. ToM and EF were also the only variables to display
clinical relevance, together distinguishing the vast majority of cases from controls, despite a lack of specific associations between cognitive and behavioural measures. While a LB appears to be fractionated from ToM and EF, it was detected in only a few children and did not improve diagnostic classification, most likely contributing to non-diagnostic symptoms in a subgroup. Despite the characteristic heterogeneity of the autism spectrum, it remains a possibility that a single cognitive cause may underlie the range of diagnostic symptoms in all individuals with autism.
Acknowledgements

The authors wish to thank the participating children and families.

We also wish to thank Uta Frith for her insight and helpful feedback.

We are grateful to the foundations that have supported this project: The Psychiatric Research Foundation in the Region of Southern Denmark; The PhD grant in Region of Southern Denmark, and; Sofiefonden.
References


EXPLORING "THE AUTISMS" AT A COGNITIVE LEVEL

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Table 1: Participants characteristics; means with standard deviations (SD) in parentheses and range below.

<table>
<thead>
<tr>
<th>Measure</th>
<th>ASD group</th>
<th>NTD group</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (Male:Female)</td>
<td>31 (25:6)</td>
<td>37 (26:11)</td>
</tr>
<tr>
<td>Age</td>
<td>10.98 (1.37) (8.1-12.8)</td>
<td>10.87 (1.33) (8.3-12.8)</td>
</tr>
<tr>
<td>Performance IQ</td>
<td>102.06 (18.10) (76-137)</td>
<td>108.59 (18.22) (64-134)</td>
</tr>
<tr>
<td>Verbal IQ</td>
<td>104.77 (13.94) (64-132)</td>
<td>102.57 (16.30) (80-143)</td>
</tr>
<tr>
<td>Full-scale IQ</td>
<td>104.45 (16.04) (75-139)</td>
<td>107.46 (18.27) (75-145)</td>
</tr>
<tr>
<td>Parents’ educational level</td>
<td>19:12</td>
<td>25:12</td>
</tr>
<tr>
<td>(higher:lower)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCQ(^a), parents***</td>
<td>16.74 (7.30) (2-30)</td>
<td>3.09 (2.39) (0-9)</td>
</tr>
<tr>
<td>SCQ(^b), teachers***</td>
<td>12.81 (5.54) (3-24)</td>
<td>6.83 (3.31) (2-14)</td>
</tr>
<tr>
<td>SRS(^c), parents***</td>
<td>91.41 (30.99) (38-140)</td>
<td>19.51 (12.09) (5-54)</td>
</tr>
<tr>
<td>SRS(^d), teachers***</td>
<td>71.90 (28.22) (32-155)</td>
<td>16.84 (13.04) (1-56)</td>
</tr>
<tr>
<td>ADOS total</td>
<td>9.90 (3.67) (4-22)</td>
<td>-</td>
</tr>
<tr>
<td>ADI total</td>
<td>30.39 (14.26)</td>
<td>-</td>
</tr>
<tr>
<td>-----------</td>
<td>---------------</td>
<td>---</td>
</tr>
<tr>
<td></td>
<td>(9-79)</td>
<td></td>
</tr>
</tbody>
</table>

\[ a \] N=31:33

\[ b \] N=27:23

\[ c \] N=29:33

\[ d \] N=20:26

*** p<0.001
Table 2: Cognitive tasks (sorted by domain) with reference to previous studies describing the task procedure.