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He said, she said: Autism spectrum diagnosis and gender differentially affect relationships between executive functions and social communication

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Abstract

Autism spectrum disorder is characterized by difficulties with social communication, with a preponderance in males. Evidence supports a relationship between metacognitive executive functions (e.g. planning, working memory) and social communication in autism spectrum disorder, yet relationships with specific metacognitive executive functions and how gender alters the expression of these relationships require further study. We used multiple regression to examine relationships between informant-based measures of metacognitive executive function and social communication in intellectually able (IQ $\geqslant$ 85) female (n = 111; mean age = 10.2 $\pm$ 2.8; 31 autism spectrum disorder) and male youth (n = 310; mean age = 10.5 $\pm$ 1.9; 146 autism spectrum disorder) with and without autism spectrum disorder from the Autism Brain Imaging Data Exchange-II database. Executive function–social communication relationships were different in females and males with autism spectrum disorder. Relationships between the entire metacognitive index and social communication were stronger in males with autism spectrum disorder than without; this pattern was also observed for metacognitive sub-indices 'monitor' and 'working memory'. These patterns were not observed in females. Relationships between executive function and social communication appear different for female and male youth with an autism spectrum disorder diagnosis. To better understand the nature of metacognitive contributions to social communication in autism spectrum disorder, future work should investigate the co-development of monitoring, working memory and social communication, while taking gender into account.

Keywords

autism, executive functions, metacognition, social communication, working memory

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by social and non-social difficulties. Although diagnostic criteria are the same for males and females, far more males are diagnosed. The overall ratio is ~3–3.5:1 (Loomes et al., 2017), but in intellectually able individuals it is closer to 10:1, while in individuals with moderate-to-severe intellectual disability, it is ~2:1 (Fombonne, 2009). One reason for the varying ratios is that intellectually able females with ASD may compensate better for their social impairment (Dworzynski et al., 2012), through increased effort to appear socially 'typical' (Lai et al., 2011). A second, related reason is the growing body of research suggesting that intellectually able females with ASD are under or mis-diagnosed because their symptoms do not resemble those typically seen in autistic males (Kirkovski et al., 2013). This is often referred to as a 'camouflaging' of the female's autistic symptoms (Dean et al., 2017; Ratto et al., 2018). Although the gender differential poses a practical challenge, research into gender differences in the ASD phenotype is needed.

The Executive Dysfunction Theory of ASD (see Hill, 2004) proposes that differences in executive functioning are central to ASD symptomatology. Executive functions (EF) allow a person to adapt flexibly to their environment by integrating information across systems in support of goal-directed behaviour (Brocki and Bohlin, 2004). Lack of flexibility is a hallmark of ASD (e.g. Ozonoff et al., 2004), and integration of information processing is also compromised (Chouinard et al., 2017; Just et al., 2004).

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Despite its face validity, studies on the Executive Dysfunction Theory of ASD are mixed. Although impairments in some abilities, such as shifting (Maes et al., 2011; Miller et al., 2015; Yerys et al., 2009) and emotion regulation (Goldsmith and Kelley, 2018; Mazefsky et al., 2013; Samson et al., 2012, 2015) are reliably reported, impairments in abilities such as inhibition (for a review, see Geurts et al., 2014) and working memory (Wang et al., 2017) are less consistently reported. One potential reason for these inconsistencies is the contrast between EF contributions to behaviour in everyday-life and lab-based measures of EF. For example, lab-based measures of working memory often fractionate the skill into memory and storage components, which is at variance with how working memory operates in everyday life (Baddeley, 1996). Ecological validity may be particularly important in ASD; a gap has been found between how well individuals with ASD score on lab-based tests of cognitive flexibility and their behavioural inflexibility in everyday life (Geurts et al., 2009).

An ecologically valid tool for assessing EF in clinical populations is the Behaviour Rating Inventory of Executive Functions (BRIEF; Gioia et al., 2000b). The BRIEF is a questionnaire completed by an informant (e.g. parent, caregiver) who is familiar with the participant. Informants rate real-life situations such as 'Gets out of control more than friends' and 'Becomes upset with new situations', where higher scores represent greater impairment. Scores are summarized in a Behaviour Regulation Index (BRI), which includes inhibition, shifting and emotional control, and a Metacognition Index (MCI), which includes initiation, monitoring, organization, planning and working memory. Theoretically, BRI functions are responsible for coordinating cognition and emotion, while MCI functions confer mental flexibility and the ability to manage interference, engage in goal-directed behaviours and anticipate consequences (Ardila, 2008).

Several recent studies using the BRIEF provide converging evidence of a specific relationship between metacognitive EF and social communication in ASD. Kenworthy et al. (2009) evaluated relationships among MCI and BRI scores and autistic symptomatology in 89 children (74 males) with ASD, Asperger syndrome and Pervasive Developmental Delay–Not Otherwise Specified (PDD-NOS). Three autism symptomatology indices, 'communication', 'reciprocal social interaction' and 'RRBIs', were derived as composite measures from relevant scores of the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000) and Autism Diagnostic Interview–Revised (ADI-R; Lord et al., 1994). They found that both behavioural regulation (BRI) and metacognitive EF (MCI) were correlated with a 'reciprocal social interaction' score (Kenworthy et al., 2009). The small sample size precluded an examination of the influence of gender.

A follow-up study (Leung et al., 2016) used a specific measure of social communication abilities, the Social Responsiveness Scale (SRS; Constantino and Gruber, 2005). The SRS comprises five subscales: four measuring social communication (Social Awareness, Social Cognition, Social Communication and Social Motivation) and one measuring non-social traits (Restricted Interests and Repetitive Behaviours). An overall SRS-Total score collates these subscales. Higher scores represent greater impairment, with $\geqslant 60$ indicating clinically significant symptomatology. Leung et al. examined EF–SRS relationships in youth aged 6–15 years with ($n = 70$; 61 males) and without ($n = 71$; 54 males) ASD. Multiple regression analyses revealed that while BRI was related to SRS-Total in both groups, a relationship between MCI and SRS-Total was specific to the ASD group. Again, the number of females was too small to examine the impact of gender.

A third study (Torske et al., 2018) evaluated 86 youth (23 females; 63 males) aged 6–18 years with ASD, Asperger syndrome or PDD-NOS and expanded on the preceding work by evaluating relationships between the MCI and BRI and each of the five SRS subscales to determine whether relationships were specific to a certain component of social functioning. Torske et al. (2018) found that both the MCI and BRI were associated with four of the five SRS subscales, while the Social Motivation subscale was associated with the MCI only (Torske et al., 2018). Although the number of females included in their study was typically small, Torske et al. (2018) found correlations between the MCI and SRS-Total in both females and males, and in the younger (6–12 years) but not in the older (13–18 years) age cohorts. Furthermore, they noted that females in their sample had greater impairment on the SRS-Total and SRS-Social Cognition subscale, compared to males.

This set of findings suggests the following hypotheses: (1) metacognitive EF has explanatory value for social functioning in ASD beyond the contribution of behaviour regulation EF, (2) relationships between metacognitive EF and social functioning are specific to the ASD group and (3) gender will influence the impact of EF impairments in ASD, particularly when those impairments are interrogated using everyday measures of EF. Here, we evaluated these hypotheses using data obtained from the Autism Brain Imaging Data Exchange (ABIDE-II). The BRIEF measured 'everyday' EF and the SRS measured social communication. Use of the ABIDE-II data provided a sample large enough to investigate a number of predictor variables (five MCI sub-indices, age and IQ) and to evaluate the role of gender on EF–social communication relationships in ASD.

Methods

Participants

Participants from the ABIDE-II database (http://fcon_1000.projects.nitrc.org/indi/abide/abide_II.html, accessed 8 May 2017) were included if they were aged 5–18 years, had

Table 1. Within-gender comparisons between individuals with and without ASD.

	Females			Males		
	ASD *M* (SD)	No ASD *M* (SD)	*p*	ASD *M* (SD)	No ASD *M* (SD)	*p*
	n = 31	*n* = 80		*n* = 146	*n* = 164	
Demographics						
Age	10.27 (2.8)	10.15 (1.4)	0.839	10.16 (2.8)	10.61 (2.1)	0.068
FIQ	107.6 (13.5)	116.4 (12.4)	0.003	111.0 (14.8)	114.9 (12.7)	0.005
Clinical tests						
BRIEF						
BRI	68.32 (12.3)	43.71 (6.5)	<0.0005	65.17 (11.8)	43.15 (6.9)	<0.0005
MCI	68.35 (11.7)	46.09 (8.2)	<0.0005	65.61 (10.5)	44.41 (8.9)	<0.0005
GEC	69.55 (11.9)	45.01 (7.2)	<0.0005	66.77 (10.7)	43.35 (7.9)	<0.0005
SRS						
SRS-Social	74.00 (10.5)	43.20 (4.6)	<0.0005	71.73 (10.8)	43.76 (4.9)	<0.0005
SRS-Total	84.00 (14.0)	43.96 (5.3)	<0.0005	76.28 (13.2)	42.80 (5.1)	<0.0005

ASD: autism spectrum disorder; SD: standard deviation; FIQ: full-scale IQ; BRIEF: Behaviour Rating Inventory of Executive Function; BRI: Behaviour Regulation Index; MCI: Metacognitive Index; GEC: Global Executive Composite; SRS: Social Responsiveness Scale.
All tests Mann–Whitney *U*. Bonferroni corrected *p*-value is 0.007.

Full-Scale IQ (FIQ) ⩾85 and possessed informant scores on both the SRS and BRIEF. Outliers with SRS-Total or BRIEF global executive composite scores ⩾3 standard deviations from their diagnostic group mean were removed (two males and one female without ASD were removed due to excessively high SRS-Social scores). The final sample comprised 421 participants from five ABIDE-II sites;[1] 177 with ASD (146 males, 31 females) and 244 without (no-ASD; 164 males, 80 females).

A majority of the demographic and clinical variables were non-normally distributed in either the ASD or no-ASD group or both. Transformation attempts (log 10, square root, squared, reciprocal, reciprocal squared) failed to normalize the data, so for demographic group comparisons, the non-parametric Mann–Whitney *U* was used, unless otherwise noted. Within each gender, individuals with ASD differed from those without on all variables ($p \leqslant 0.005$) except age (Table 1). Within each diagnostic-group, females and males with ASD differed on SRS-Total scores (females exhibiting greater impairment than males, $p = 0.004$), while females and males without ASD did not differ on any of the variables ($ps \geqslant 0.08$; Table 2).

ABIDE-II data were collected with strict ethical and confidentiality procedures,[2] and this study was approved by the School of Psychology Research Ethics Committee at Trinity College Dublin.

Materials

BRIEF-Informant (Gioia et al., 2000a). In line with previous research, our analyses examined the behaviour regulation index (BRI; inhibition, shifting and emotional control), and the MCI (initiation, working memory, planning, organization and monitoring).

SRS-Informant (Constantino and Gruber, 2005). To specifically examine relationships between EF and social communication, rather than using the SRS-Total score, our analyses focused on a 'social communication index' score that collated the four SRS social communication subscales, denoted in this study as 'SRS-Social'. Within our ASD sample, females and males differed on SRS-Total scores, but not SRS-Social scores. Accordingly, we eliminated a potential confounding variable by focusing on SRS-Social scores, as planned, in our analyses. This focus is supported by a previous factor analysis of the SRS-2, which found definite groupings of SRS-2 items separating social communication and RRBIs (Frazier et al., 2014), as well as research that indicates that females have restricted interests that appear more socially appropriate than those displayed by males, thus masking their clinical symptoms (Kirkovski et al., 2013) and potentially leading to an underestimation of RRBIs in females when current standardized assessments, such as the SRS-2, are used. Finally, SRS-2 exhibits high internal consistency (Constantino and Gruber, 2012) and our SRS-Social and SRS-Total scores were highly correlated in individuals with (Spearman's rho = 0.962, $p < 0.0005$) and without ASD (Pearson $r = 0.960$, $p < 0.0005$).

Procedure

Using SPSS24, we ran hierarchical multiple regression analyses with SRS-Social *T*-scores as the outcome variable. FIQ and age were included as predictor variables in all models (for correlation matrices, see Table 3). Running separate regressions for females and males, we first evaluated BRI and MCI. We ran a hierarchical multiple regression with the following predictors: ASD group membership

Table 2. Within-diagnostic group comparisons between males and females.

	ASD			No-ASD		
	Female *M* (SD)	Male *M* (SD)	*p*	Female *M* (SD)	Male *M* (SD)	*p*
	n = 31	*n* = 146		*n* = 80	*n* = 164	
Demographics						
Age	10.27 (2.8)	10.16 (2.8)	0.846	10.15 (1.4)	10.61 (2.1)	0.122
FIQ	107.6 (13.5)	111.0 (14.8)	0.246	116.35 (12.4)	114.9 (12.7)	0.423
Clinical tests						
BRIEF						
BRI	68.32 (12.3)	65.17 (11.8)	0.180	43.71 (6.5)	43.15 (6.9)	0.337
MCI	68.35 (11.7)	65.61 (10.5)	0.198	46.09 (8.2)	44.41 (8.9)	0.141
GEC	69.55 (11.9)	66.77 (10.7)	0.199	45.01 (7.2)	43.35 (7.9)	0.080
SRS						
SRS-Social	74.00 (10.5)	71.73 (10.8)	0.284	43.20 (4.6)	43.76 (4.9)	0.432
SRS-Total	84.00 (14.0)	76.28 (13.2)	0.004	43.96 (5.3)	42.80 (5.1)	0.123

ASD: autism spectrum disorder; SD: standard deviation; FIQ: full-scale IQ; BRIEF: Behaviour Rating Inventory of Executive Function; BRI: Behaviour Regulation Index; MCI: Metacognitive Index; GEC: Global Executive Composite; SRS: Social Responsiveness Scale.
t-tests used for ASD comparisons and Mann–Whitney U used for no-ASD comparisons. Bonferroni corrected p-value is 0.007.

Table 3. Spearman's rho correlation matrices for regression predictors.

Females (*n* = 111)	SRS-Social	ASD group	Age	IQ	BRI	MCI	Initiate	Monitor	Organize	Plan	Working memory
SRS-Social	1.000						0.78**	0.79**	0.54**	0.74**	0.79**
ASD group	0.78**	1.000					0.71**	0.70**	0.48**	0.69**	0.73**
Age	−0.11	−0.020	1.000				−0.04	−0.03	−0.05	0.01	−0.06
IQ	−0.22*	−0.28**	0.08	1.000			−0.19**	0.18**	−0.05	−0.20**	−0.22**
BRI	0.68**	0.70**	0.05	−0.14	1.000		–	–	–	–	–
MCI	0.74**	0.68**	0.05	−0.23*	0.79**	1.000	–	–	–	–	–
Initiate	0.72**	0.63**	0.05	−0.19*	–	–	1.000				
Monitor	0.66**	0.64**	0.04	−0.24*	–	–	0.75*	1.000			
Organize	0.58**	0.45**	0.09	−0.08	–	–	0.64*	0.61**	1.000		
Plan	0.69**	0.62**	0.18	−0.22*	–	–	0.82**	0.81**	0.71**	1.000	
Working memory	0.67**	0.68**	−0.03	−0.32*	–	–	0.75**	0.77**	0.64**	0.78**	1.000
Males (*n* = 310)											
SRS-Social	1.000						0.72**	0.66**	0.58**	0.69**	0.67**
ASD group	0.85**	1.000					0.63**	0.64**	0.45**	0.62**	0.68**
Age	−0.13*	−0.10	1.000				0.05	0.04	0.09	0.18	−0.03
IQ	−0.17**	−0.16**	−0.04	1.000			−0.19*	−0.24*	−0.08	−0.22*	−0.32**
BRI	0.84**	0.76**	−0.0.06	−0.12*	1.000		–	–	–	–	–
MCI	0.80**	0.73**	−0.05	−0.20**	0.83**	1.000	–	–	–	–	–
Initiate	0.78**	0.71**	−0.04	−0.19**	–	–	1.000				
Monitor	0.79**	0.70**	−0.03	−0.18**	–	–	0.77**	1.000			
Organize	0.54**	0.48**	−0.05	−0.05	–	–	0.67**	0.63**	1.000		
Plan	0.74**	0.69**	0.01	−0.20**	–	–	0.87**	0.83**	0.71**	1.000	
Working memory	0.79**	0.73**	−0.06	−0.22**	–	–	0.84**	0.80**	0.68**	0.87**	1.000

SRS: Social Responsiveness Scale; ASD: autism spectrum disorder; BRI: Behaviour Regulation Index; MCI: Metacognition Index.
Females (top panel) and males (bottom panel).
*$p < 0.05$; **$p < 0.01$.

in the first step, FIQ and age in the second step, BRIEF BRI and MCI in the third step and the BRI × ASD group and MCI × ASD group interactions in the fourth step.

The second set of analyses regressed SRS-Social *T*-scores on the following predictors: ASD group membership (step 1), FIQ and age (step 2), the five MCI sub-indices (initiation,

Table 4. Overall hierarchical multiple regressions in each gender for the outcome variable SRS-Social with predictors comprising BRIEF indices and interactions (top panel) and MCI sub-indices and interactions (bottom panel).

		Females (ASD $n=31$, no-ASD $n=80$)							Males (ASD $n=147$, no-ASD $n=164$)						
BRIEF indices analyses															
Step	Predictor	*R*	R^2	$R^2\Delta$	*B*	SE *B*	Beta	*t*	*R*	R^2	$R^2\Delta$	*B*	SE *B*	Beta	*t*
1	ASD group	0.90	**0.81**✧	**0.81**✧	10.58	8.18	0.31	1.29	0.86	**0.75**✧	**0.75**✧	−3.22	4.76	−0.10	−0.66
2	Age	0.90	**0.81**✧	0.00	−0.28	0.30	−0.03	−0.76	0.86	**0.75**✧	0.00	−0.31	0.15	**−0.05**	**−2.08**
	FIQ				0.02	0.05	0.01	0.35				0.00	0.03	0.00	−0.02
3	BRI	0.93	**0.86**✧	**0.05**✧	0.08	0.12	0.08	0.68	0.92	**0.85**✧	**0.10**✧	0.25	0.09	**0.22***	**2.72***
	MCI				**0.25**	0.10	**0.22***	**2.49***				0.12	0.07	0.11	1.73
4	BRI × Group	0.93	**0.87**✧	0.00	0.00	0.18	0.00	0.01	0.92	**0.85**✧	**0.01**†	0.16	0.11	0.34	1.52
	MCI × Group				0.19	0.16	0.38	1.14				0.19	0.10	**0.39**	**1.99**
MCI sub-indices analyses															
Step	Predictor	*R*	R^2	$R^2\Delta$	*B*	SE *B*	Beta	*t*	*R*	R^2	$R^2\Delta$	*B*	SE *B*	Beta	*t*
1	ASD group	**0.90**	**0.81**✧	**0.81**✧	16.03	8.82	0.47	1.82	**0.86**	**0.75**✧	**0.75**✧	−0.24	5.20	−0.08	−0.47
2	Age	0.90	**0.81**✧	0.00	−0.14	0.31	−0.02	−0.44	**0.86**✧	**0.75**✧	0.00	−0.27	0.15	−0.04	−1.72
	FIQ				0.03	0.05	0.02	0.63				0.03	0.03	0.03	1.04
3	Initiate	**0.94**	**0.88**✧	**0.07**✧	0.20	0.11	0.17	1.78	**0.92**✧	**0.84**✧	**0.09**✧	0.25	0.11	**0.21***	**2.38***
	Monitor				0.11	0.11	0.10	1.00				0.19	0.08	**0.17***	**2.44***
	Organization				0.18	0.10	0.13	1.86				−0.06	0.08	−0.04	−0.77
	Plan				−0.10	0.15	−0.09	0.50				−0.08	0.11	−0.07	−0.68
	Working memory				−0.04	0.16	−0.04	−0.28				0.03	0.10	0.03	0.36
4	Initiate × Group	0.94	**0.88**✧	0.01	0.16	0.17	0.31	0.93	**0.92**✧	**0.85**✧	**0.01**†	−0.01	0.13	−0.03	−0.11
	Monitor × Group				0.11	0.17	0.22	0.62				0.23	0.10	**0.47***	**2.31***
	Organize × Group				−0.18	0.16	−0.32	−1.14				−0.06	0.10	−0.11	−0.58
	Plan × Group				0.24	0.21	0.48	1.16				−0.08	0.14	−0.16	−0.55
	WM × Group				−0.18	0.20	−0.37	−0.88				0.27	0.12	**0.56**	**2.19**

SRS: Social Responsiveness Scale; BRIEF: Behaviour Rating Inventory of Executive Function; MCI: Metacognition Index; ASD: autism spectrum disorder; SE: standard error; BRI: Behaviour Regulation Index; FIQ: full-scale IQ.
Corrected for multiple comparisons: 0.05/2 = 0.025. Bold only if $p < 0.05$ but did not survive correction for multiple comparisons, otherwise *$p < 0.025$, †$p \leq 0.001$, ✧$p < 0.0005$.

monitoring, organization, planning and working memory; step 3) and interactions of interest (each sub-index × ASD Group; step 4). Again, separate hierarchical multiple regressions were run for females and males. When a significant interaction effect with diagnostic group was indicated, we carried out separate regressions for individuals with and without ASD to fully characterize group differences. Each sample that was evaluated (all females, all males, males with ASD, males without ASD) underwent two regressions, necessitating a Bonferroni correction for multiple comparison (adjusted $p < 0.025$). However, given that part of our study is a replication, and because Bonferroni correction is quite strict, we considered results $0.025 \leq p \leq 0.05$ as important and meaningful if they were in line with or supported by other findings and if they indicated a considerably different *p* value than the contrast group.

For our regressions, we evaluated the three assumptions of regression: homogeneity of variance/homoscedasticity, multicollinearity and normality, for each of the four samples of interest: (1) females with, (2) females without, (3) males with and (4) males without ASD. Homoscedasticity was evaluated by visually inspecting scatterplots of the predicted values and residuals for random distribution above and below zero on both *x*- and *y*-axes. Multicollinearity was evaluated by checking variance inflation factor (VIF) numbers to determine that they were <10 (acceptable) or <5 (ideal). Normality was evaluated by visually inspecting whether residuals fell along the diagonal line of the respective P-P Plots. For all four samples and all predictors, assumptions were met.

Results

BRIEF main indices: BRI and MCI

The first set of analyses regressed SRS-Social on ASD group membership, FIQ and age, the BRIEF indices (BRI and MCI) and the interactions: BRI × ASD group and MCI × ASD group (top of Table 4), separately for females and males.

Table 5. Males only, hierarchical multiple regression by diagnostic group for the outcome variable SRS-Social, with predictors comprising BRIEF indices (top panel) and MCI sub-indices (bottom panel).

		Males with ASD ($n = 147$)							Males without ASD ($n = 164$)						
BRIEF indices analyses															
Step	Predictor	*R*	R^2	$R^2\Delta$	*B*	SE *B*	Beta	*t*	*R*	R^2	$R^2\Delta$	*B*	SE *B*	Beta	*t*
1	Age	0.05	0.00	0.00	−0.29	0.24	−0.08	−1.21	0.10	0.01	0.01	−0.37	0.16	**−0.15**	**−2.23**
	FIQ				0.03	0.05	0.04	0.57				−0.03	0.03	−0.09	−1.27
2	BRI	0.68	**0.46**✧	**0.46**✧	0.41	0.07	**0.45**	**5.69**✧	0.53	**0.28**✧	**0.27**✧	0.26	0.06	**0.37**	**4.23**✧
	MCI				0.32	0.08	**0.31**	**3.92**✧				0.12	0.05	**0.21**	**2.45***
MCI sub-indices analyses															
Step	Predictor	*R*	R^2	$R^2\Delta$	*B*	SE *B*	Beta	*t*	*R*	R^2	$R^2\Delta$	*B*	SE *B*	Beta	*t*
1	Age	0.05	0.00	0.00	−0.25	0.25	−0.06	−0.98	0.10	0.01	0.01	−0.33	0.17	**−0.14**	**−2.01**
	FIQ				0.06	0.05	0.09	1.26				−0.01	0.03	−0.02	−0.34
2	Initiate	0.66	**0.44**✧	**0.44**✧	0.24	0.09	**0.25**	**2.55***	0.56	**0.31**✧	**0.30**✧	0.25	0.07	**0.38**	**3.67**✧
	Monitor				0.42	0.08	**0.43**	**5.16**✧				0.19	0.05	**0.37**	**3.61**✧
	Organisation				−0.13	0.08	−0.12	−1.53				−0.06	0.05	−0.11	−1.13
	Plan				−0.14	0.11	−0.16	−1.37				−0.07	0.07	−0.13	−0.97
	Working memory				0.31	0.10	**0.32**	**3.05***				0.03	0.06	0.05	0.49

SRS: Social Responsiveness Scale; BRIEF: Behaviour Rating Inventory of Executive Function; MCI: Metacognition Index; ASD: autism spectrum disorder; BRI: Behaviour Regulation Index; SE: standard error; FIQ: full-scale IQ.
Corrected for multiple comparisons: 0.05/2 = 0.025. Bold only if $p < 0.05$ but did not survive correction for multiple comparisons, otherwise *$p < 0.025$, †$p < 0.001$, ✧$p < 0.0005$.

In females, the overall model accounted for 86.7% of the variance in SRS-Social scores ($p \leqslant 0.0005$). The only significant variable in the model was MCI ($p = 0.007$; Supplemental Figure S1). Because there were no interaction effects in this females-only model, we did not run separate regressions for each diagnostic group.

In males, the overall model accounted for 85.4% of the variance in SRS-Social scores ($p \leqslant 0.0005$), with a significant effect of age (which did not survive correction for multiple comparisons; $p = 0.039$) and BRI ($p = 0.007$) and indication towards a significant MCI × ASD group interaction (failed to survive correction for multiple comparisons; $p = 0.048$). Because of the trend towards a significant group interaction in this males-only model, we carried out follow-up separate multiple regressions for each diagnostic group (Table 5). Before correction for multiple comparisons, the model accounted for 46.3% of the variance in SRS-Social scores in the ASD group and 28.3% of the variance in the no-ASD group ($ps \leqslant 0.0005$). The BRI ($p < 0.0005$) and MCI ($p < 0.0005$, ASD; $p = 0.016$) were significant in both groups, while age was borderline significant in the no-ASD group only ($p = 0.027$). Thus, although the MCI was associated with social communication in both diagnostic groups, the interaction effect in the overall model indicated that the MCI was more strongly associated with SRS-Social scores in males with ASD than in males without (Figure 1).

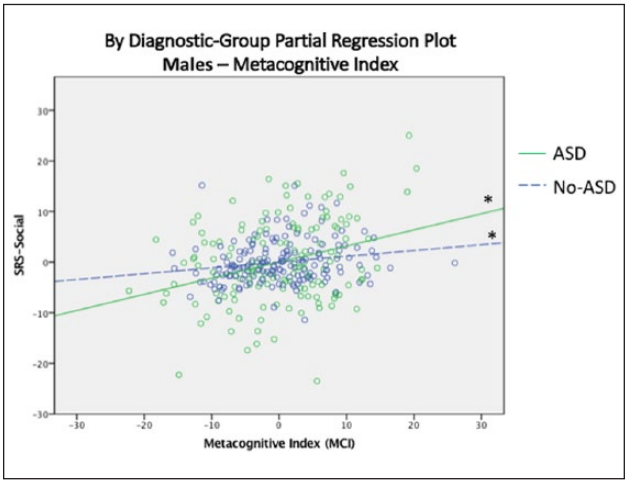

Figure 1. Males, by diagnostic-group – partial regression plot of dependent variable 'SRS-Social' and predictor 'metacognitive index' (MCI) for ASD (solid line) and no-ASD (hashed line); axes are residuals from (1) regressing SRS-Social on all predictors less MCI (*y*-axis) and (2) regressing MCI on all other predictors (*x*-axis).
*$p \leqslant 0.05$.

MCI sub-indices

Next, because of recent evidence linking MCI and social autistic symptomatology (Kenworthy et al., 2009; Torske et al., 2018), specific to individuals with ASD compared

to those without (Leung et al., 2016), we investigated relationships between MCI sub-indices and SRS-Social scores. This was supported by the significant (*uncorrected*) MCI × ASD Group interaction in males in our first regression.

Separately for females and males, SRS-Social was regressed on diagnostic group (step 1), FIQ and age (step 2), the MCI sub-indices (initiate, monitor, organize, plan, working memory; step 3) and the five sub-index interactions (i.e. each sub-index × ASD group; step 4; bottom half of Table 4).

In females, the overall model accounted for 88.4% of the variance ($p \leqslant 0.0005$). None of the sub-indices were significant, although the addition of the sub-indices did significantly change the model ($R^2\Delta = 0.07$, $p < 0.0005$), and ASD group ($p = 0.072$), 'initiate' ($p = 0.078$), and 'organization' ($p = 0.066$) trended towards significance. Because there were no interaction effects in this females-only model, we did not run separate regressions for each diagnostic group.

In males, the overall model accounted for 85.0% of the variance ($p \leqslant 0.0005$). The sub-indices 'initiate' ($p = 0.018$) and 'monitor' ($p = 0.015$) were significant, and there were significant 'monitor' × ASD group ($p = 0.021$) and 'working memory' × ASD group ($p = 0.029$; did not survive correction for multiple comparisons) interactions. Because of the trend towards a significant group interaction in this males-only model, we carried out follow-up separate multiple regressions for each diagnostic group (Table 5), where the model accounted for 43.9% of the variance in males with ASD and 31.3% of the variance in males without ASD (ps $\leqslant 0.0005$). The sub-indices 'initiate' ($p = 0.012$), 'monitor' ($p < 0.0005$) and 'working memory' ($p = 0.003$) were significant in the ASD group, while age ($p = .046$; did not survive correction for multiple comparisons), 'initiate' ($p < 0.0005$) and 'monitor' ($p < 0.0005$) were significant in the no-ASD group. Thus, 'initiate' and 'monitor' were related to social communication in both diagnostic groups, but the interaction effect in the overall model indicated that 'monitor' was more strongly associated with SRS-Social in males with ASD than in those without, though not significantly so (Supplemental Figure 2). Furthermore, the indication of an interaction effect for 'working memory' and the follow-up regressions indicated that 'working memory' was associated with SRS-Social in the ASD group alone (Supplemental Figure 3).

Discussion

We evaluated relationships between social functioning and metacognitive EF in females and males with and without ASD using sample sizes that afforded relatively increased power compared to existing studies. Although examining gender in ASD is always difficult because there are far fewer females with an ASD diagnosis than males, use of the ABIDE-II dataset offered the power (31 females with ASD, 80 without) to detect relatively *large*-sized effects. We obtained three main findings. First, in line with our hypotheses, we found that for both sexes metacognitive EF had explanatory value for social communication, and that for males, this was beyond the contribution of behaviour regulation EF and that the relationship was stronger in the ASD than in the no-ASD group. Second, our data suggest that gender influences the relationship between EF and social communication in ASD, as this pattern was observed in males but not in females. Third, although we did not have specific hypotheses regarding the impact of age, a relationship between age and social communication in males without ASD, while not significant when corrected for multiple comparisons, is nonetheless consistent with studies reporting age-related differences in ASD compared to individuals without.

Everyday executive functioning and social communication in males with ASD

We found that BRI–social communication relationships were exhibited by all males. This finding resonates with previous research that found BRI–SRS relationships in individuals with and without ASD (Leung et al., 2016); it is logical that behaviour regulation EF, which allow a person to fulfil or delay impulses following socially acceptable strategies (Ardila, 2008), is strongly associated with social communication.

An arguably more interesting finding was that while MCI–social communication associations were found in both diagnostic groups, there was a trend for a stronger association in ASD than in no-ASD, although confidence intervals did overlap. Other ASD studies have also found relationships between MCI and social communication (Kenworthy et al., 2009; Leung et al., 2016; Torske et al., 2018) and between MCI and adaptive functioning (Gilotty et al., 2002). Notably, our finding even more closely parallels Leung et al. (2016), who observed MCI–SRS relationship in their ASD group uniquely. That we found an MCI–SRS relationship in both groups, while Leung et al. (2016) did not, may reflect the increased power afforded by our larger ABIDE-II sample.

Our second analysis examined whether these relationships were specific to any of the MCI sub-indices. Consistent with the pattern for the MCI overall, the sub-index 'monitor' was more strongly related to SRS-Social in those with ASD than those without, while a relationship between the 'working memory' sub-index and SRS-Social was significant in the ASD group alone. The BRIEF sub-index 'monitor' captures work-checking habits and interpersonal awareness (Gioia et al., 2000a), both of which have straightforward associations with social communication. Psychological studies suggest that performance monitoring is altered in ASD and that this difference contributes

to social and cognitive difficulties (Hüpen et al., 2016; Mundy, 2003). In addition, self-monitoring of both communicative (are responses appropriate to previous comments) and non-communicative acts (remember to not pick nose in front of others) are important for successful social interaction. Finally, monitoring of one's self and others is usually attributed to Theory of Mind, which is often found to be weaker in individuals with ASD than those without (see Baron-Cohen, 2000, for a review).

In contrast to the 'monitor' sub-index, 'working memory' was associated with social communication in the ASD group alone. Working memory involves storing and manipulating information in mind, which is integral to the representation and updating of current behavioural goals and environmental contexts and demands (Baddeley, 2003). Social communication places considerable demands on working memory, since social rules, environmental clues and complex language must be understood and integrated for successful social navigation. Our finding resonates with previous research indicating relationships between complex working memory and adaptive functioning in ASD (Gilotty et al., 2002; Pugliese et al., 2015), as well as research demonstrating that children with ASD exhibited improvements in both working memory and parent ratings of social behaviour following three different working memory training programmes (De Vries et al., 2015). Indeed, our findings offer support for interventions that aim to improve social communication in ASD by training EF such as working memory. However, the relationship between working memory and social communication was not observed in the no-ASD group, possibly suggesting that working memory is marshalled in support of social communication only among those for whom social communication is particularly challenging – one reason why training on working memory may benefit those with clinical diagnoses but not typically developing comparisons (Melby-Lervåg and Hulme, 2013). Alternatively, the relationship may reflect a shared cognitive construct that is relied on for both working memory and social communication or a strategy that individuals with ASD are using to succeed at higher level integration type tasks. While there is no way to adjudicate among these possibilities in this study, future work should incorporate this goal. Strategy use in ASD has previously been examined in behavioural (Bebko and Ricciuti, 2000; Livingston et al., 2018) and neuroimaging studies (Kana and Wadsworth, 2012; Wang et al., 2006) and should be part of future investigations into working memory–social communication relationships in ASD.

Differential relationships in females and males

The pattern of EF–social communication relationships observed for females with ASD was strikingly different from that observed in males. First, unlike males in this study, BRI was not a significant predictor of social communication in females. This also contrasts with the findings of Torske et al. (2018) who observed correlations between BRI and SRS-Total within the females with ASD in their sample. Second, there was no diagnostic group interaction in the MCI–SRS-Social relationship for females in this study although there was a significant main relationship between MCI and social communication, and this pattern was the reverse of what was observed in males, where there was no significant main relationship between MCI and SRS-Social, but there was a borderline diagnostic group interaction ($p = 0.04$) favouring males with ASD. This lack of demarcation in females does not seem to reflect a demographic difference, because SRS-Social scores were the same across female diagnostic groups and across male diagnostic groups (Table 1). Nor does the lack of differentiation in females appear to reflect better social skills in females with ASD, as there was no statistical difference in SRS-Social scores between females and males with ASD and the gap between ASD and no-ASD social scores was numerically larger for females (40.04) than for males (33.48). Taken together, the information supports an emerging literature on gender differences in the ASD phenotype, including the developmental trajectories of social communication (Lai et al., 2011), neuroanatomy (Bloss and Courchesne, 2007) and play (Knickmeyer et al., 2008). However, findings must be interpreted cautiously until studies can be conducted with female sample sizes that are sufficiently powered.

The evaluation of sub-indices indicated no significant metacognitive sub-index predictors for females, although this finding should be interpreted cautiously due to the considerable number of predictors (13) and moderate sample size ($n = 111$), which potentially limited statistical power. We note that 'ASD group', 'initiate' and 'organisation' all trended towards significance, with p values <0.10. Better powered future research is required to discover whether any of these factors are important to our understanding of EF–social communication relationships in females with ASD.

This study has demonstrated that the similarities and differences between individuals with and without ASD manifest differently for females than for males. This evidence supports the growing literature indicating the need to be cognizant of a 'female autism phenotype' and reminds us that females with and without ASD may look very similar, in contrast to the notable differences that are often observed when comparing males with and without ASD. What we found is that females with and without ASD look very similar to each other in terms of relationships between everyday/observed EF and SRS-Social, while there were differences in these relationships for males with and without ASD. Potentially, this relates to evidence that indicates females with ASD do a better job of 'camouflaging' or compensating for their

socio-communicative (Dean et al., 2017) and RRBI (Kirkovski et al., 2013) traits. Our study does not answer whether or not the females with ASD in our sample were camouflaging, but it is worth noting that our measures of interest (i.e. SRS-2 and BRIEF-2) were both informant measures and thus susceptible to the perceptions of others/camouflaging. Future research that includes lab-based measures of working memory may better be able to answer the question of whether there are relationships between more objective, lab-based working memory measures and social communication that are similar for both females and males with ASD.

Altogether, the data suggest that there is merit to evaluating relationships between EF and social functioning in females and males with ASD separately. A better understanding of gender differences in ASD is important to the provision of appropriate resources and services (Halladay et al., 2015). For example, our finding of differences in the genders for relationships between behaviour regulation EF and social communication and between metacognitive EF and social communication may have direct relevance for the design of social communication interventions involving EF – interventions involving both behaviour regulation and metacognitive EF may benefit males, while interventions for females may be more beneficial if they focus on metacognitive EF alone.

Age-related differences in individuals with ASD

Although failing to survive correction for multiple comparisons, p values for the influence of age on SRS-Social were consistently <0.05 for males *without* ASD. The relationship was negative, indicating that older males in our sample had lower SRS-Social scores. Because this maturation of social skills with age is part of typical development, the absence of this same pattern in ASD is consistent with a pattern of age-related differences reported in several behavioural (Luna et al., 2007; O'Hearn et al., 2010) and neuroimaging (Alaerts et al., 2015; Escalante-Mead et al., 2003 Washington et al., 2014) studies. Notably, this same age-related improvement in SRS-Social scores was not seen in the males with ASD, which aligns with retrospective longitudinal research that indicates fewer gains in reciprocal social functioning relative to communication when comparing current and lifetime ADI scores (Howlin et al., 2013).

Limitations

Although this is the largest powered study to date, the data were aggregated from five different sites, which may be a source of increased variability, thus reducing power. Hence, findings in our study with p values between 0.01 and 0.05 should be interpreted cautiously. In addition, every informant who completes the BRIEF or SRS on behalf of a participant with ASD may interpret the items differently, further increasing the noise and variability in our data. Also, there is the potential that there was a high rate of attention deficit hyperactivity disorder (ADHD) comorbidity in the sample, typically 30%–50% in ASD samples – thus, the relationship between working memory and social communication identified here could reflect the influence of ADHD symptomatology. Unfortunately, we were not able to directly address this potential confound in this study due to low number of individuals for whom ADHD symptom scores were available. Capturing and controlling for ADHD symptoms should be a priority for future research. Finally, it is important to emphasize that the BRIEF is a questionnaire-based measure that captures an informant's ratings of the participant's everyday executive functioning. Thus, although the BRIEF represents an ecologically valid measure of EF, correlations between the BRIEF and lab-based measures of EF vary (Anderson et al., 2002). Indeed, in other clinical populations it has similarly been found that both lab-based measures and ecologically valid measures correlate with impairment, but not necessarily with each other (Higginson et al., 2000) suggesting that the two types of tests may be measuring different aspects of the same construct. Thus, our results should be interpreted cautiously when discussing 'working memory deficits' and 'ASD'.

Conclusion

We investigated the relationships between everyday executive functioning and social communication in females and males with and without ASD. Females and males exhibited strikingly different relationships. For females, metacognitive EF alone were associated with social communication, while in males with ASD, both behaviour regulation and metacognitive EF were associated with social communication, and the relationship between metacognitive EF and social communication was stronger for males with ASD than those without. Within the metacognitive EF, self-monitoring/self-awareness and working memory were more strongly associated with social communication in males with ASD than without. Finally, age-related trends indicative of natural maturation of social communication were found among males without ASD only, supporting other behavioural and neuroimaging findings of age-related differences in ASD. In summary, this study provides further evidence of a role for behavioural regulation and metacognitive functions in social communication in ASD, highlighting EF training as a potential route for improving social communication. However, gender differences in the EF–social communication relationships emphasize the importance of taking gender into account when devising such interventions.

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Supplemental material

Supplemental material for this article is available online.

Notes

1. See Supplemental Table S1 for how autism spectrum disorder (ASD) diagnosis was confirmed and exclusion criteria for each site. Additional details are available at http://fcon_1000.projects.nitrc.org/indi/abide/abide_II.
2. Data from the Autism Brain Imaging Data Exchange-II (ABIDE-II) dataset are de-identified in compliance with US Health Insurance Portability and Accountability Act guidelines. Participants at all sites signed written informed consent and assent (and parental consent, if participants were less than 18 years) in accordance with US 45 CFR 46 and Declaration of Helsinki for participation; research protocols were approved by local ethics committees. Analyses of the de-identified data were reviewed and approved by Institutional Review Boards of Rutgers University and Columbia University Medical Center.

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