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RESEARCH

Contribution of attentional mechanisms to verbal and nonverbal communication in boys with ASD

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Abstract

Communication deficits in Autism Spectrum Disorder (ASD) involve impairments in both verbal and nonverbal domains, potentially associated with altered brain network connectivity related to language, attention, and social cognition systems. This study investigated functional connectivity patterns among the Default Mode Network (DMN), Salience Network (SN), Dorsal Attention Network (DAN), and Language Network (LN) in male children with ASD using resting-state fMRI data and Principal Component Analysis (PCA) to define sample-specific regions of interest. The sample included 53 males with ASD and 27 typically developing controls aged 5 to 12 years. Group comparisons revealed underconnectivity between the SN and LN ($p=0.003$, beta = -0.49; $p=0.04$, beta = -0.38) and overconnectivity between the DMN and LN in the ASD group ($p=0.003$, beta=0.5; $p=0.02$, beta=0.4). Crucially, further analyses showed that impairments in verbal ($p=0.016$, beta = -0.45; $p=0.02$, beta = -0.46) and nonverbal ($p=0.03$; beta = -0.42) communication in ASD were associated with reduced connectivity within the DAN and between the DAN and SN, rather than with the LN. These findings suggest that communication difficulties in ASD have a stronger attentional basis linked to disruptions in sustained and switching attention mechanisms, as opposed to isolated language network dysfunctions. Our results underscore the importance of considering the integrated functioning of attentional and social brain networks to better understand the neural substrates of communication challenges in ASD.

Keywords Autism Spectrum Disorder · Connectivity · Communication · Principal component analysis

Introduction

Communication, as a fundamental component of social cognition, relies on both linguistic and paralinguistic elements. These elements collaboratively enable the transmission and interpretation of messages through the focus of communicators' attention. Consequently, three key attributes underpin successful communication: well-developed social skills, language abilities, and attentional capacities. We hypothesize that effective communication depends not only on the proper functioning of the brain's language, attention, and social systems individually but also on their balanced and enhanced integrated performance. Given that individuals with Autism Spectrum Disorder (ASD) frequently face challenges in communication, impacting both verbal and nonverbal social interactions, we anticipate alterations in the neural substrates underlying language, attention, and social behavior. Furthermore, we assume that these neural atypicalties are associated with difficulties in verbal and nonverbal communication within this population.

Networks involved in communication

Default mode network

From the resting-state perspective, the altered functional connectivity (FC) within and between large-scale networks may serve as a potential biomarker for predicting ASD symptom severity in social behavior [15]. One of the prominent and consistently replicated findings regarding atypical social abilities in ASD is disrupted connectivity within the Default Mode network (DMN; [7, 18, 24, 31,

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34, 46, 55, 58]). The DMN typically comprises the right and left lateral parietal, posterior cingulate and medial prefrontal cortices (right/left LP, PCC, MPFC; [1, 27]; Human Connectome Project) and is critically involved in self-referential processing [11] and especially in theory of mind [50]. Between-network connectivity investigation shows that overconnectivity between the DMN and the Salience network (SN) predicts more severe social responsiveness impairments in ASD [6, 40]. Beyond mentalizing and self-referential processing, the DMN's FC is also associated with semantic cognition and construction of embodied situation models [16], as well as working memory [21, 32, 48, 49] and autobiographical memory [39]. The DMN's contribution to verbal and nonverbal communication lies in its role in understanding others' mental states, self-reflection, and recalling relevant experiences.

Dorsal attention network

Attention is a dual process involving sustained focus on task-relevant stimuli and selective reorientation toward salient ones. These mechanisms are supported by distinct but interacting brain networks that modulate "top-down" and "bottom-up" sensory processing [8, 9]. The Dorsal Attention Network (DAN) primarily includes the frontal eye fields (FEF), superior parietal lobules (SPL; [2]). The DAN globally ensures "top-down" attentional control and specifically involves in visual attention, including spatial attention, feature attention, and object attention [10, 20, 41, 51]. The typical functioning of the DAN is crucial for successful communication, as it directs and sustains attention toward the most relevant external information.

Salience network

The "bottom-up" attentional mechanisms are divided into functionally overlapping the Ventral Attention network and the SN [59]. Moreover, the SN's role extends beyond simple "bottom-up" attention to include filtering of external inputs, modulating behavioral flexibility, and supporting verbal intelligence [29, 36, 37]. This suggests that the SN acts as a hub that coordinates attentional shifts and cognitive control by interacting with both the VAN and DAN. Thus, we decided to focus on the SN comprising the bilateral supramarginal gyri (SMG), rostral prefrontal cortices (RPFC), anterior insula (AIns) and anterior cingulate cortex (ACC; [22, 40]). The SN is important in communication because it identifies and prioritizes the most relevant sensory and emotional information. Moreover, the SN enables the dynamic switching between brain systems that support language processing, social behavior, and sustained attention.

Functional connectivity of the attention, mentalizing and language networks in ASD

Underconnectivity within the DMN was already mentioned as one of the most replicable findings in the resting-state research of ASD [7, 46, 58]. Moreover, the DMN' alterations have been consistently linked to core social and communication symptoms in ASD [7, 46]. Beyond the DMN, atypical connectivity patterns have also been observed in the SN, the DAN and the Language network (LN).

Overconnectivity of the SN has been shown to predict decreased verbal intelligence [36], sensory traits [1], and restricted and repetitive behaviors [53] in individuals with ASD. Sociocommunicative deficits have been associated not only with overconnectivity of the SN [1] but also with increased FC between the SN and the DMN [6, 40].

The connectivity of the DAN varies with developmental stages: children with ASD exhibit hyperconnectivity of the DAN [14, 17], whereas adults with ASD show hypoconnectivity [14]. Both hyper- and hypoconnectivity patterns implicate difficulties in goal-driven, endogenous attention control and "bottom-up" processing [14, 17]. Furthermore, overconnectivity between the DAN and other networks predicts social and communication deficits [35], as well as impairments in attention switching and cognitive functions [23, 38] in individuals with ASD.

Regarding the LN, findings are inconsistent. Task-based functional magnetic resonance imaging (fMRI) studies typically report underconnectivity within the LN during language tasks [25, 26, 28], whereas resting-state fMRI studies often demonstrate overconnectivity within the LN [19, 42, 43]. Notably, the LN overconnectivity correlates negatively with social symptom severity but not directly with language abilities [19]. It has also been shown that the LN exhibits increased connectivity with the DMN in ASD, indicating reduced segregation, less isolated and modular functioning, between language and mentalizing systems [19, 60]. These patterns suggest that in ASD, the LN is not only atypically connected internally but also shows altered interactions with other large-scale networks implicated in social cognition and attention. These connectivity differences may underlie the complex language and communication difficulties characteristic of the disorder.

The present study

While individual network alterations in ASD are well-documented, their integrated interactions and specific contributions to verbal versus nonverbal communication remain underexplored. We predict ASD-specific FC disruptions that correlate with communication deficits. Brain-behavior models will reveal which connections uniquely predict verbal

and nonverbal impairments, hypothesizing imbalanced network integration as a key mechanism. Thus, we aimed to investigate the interactions among the DMN, SN, DAN, and LN, and their implications for verbal and nonverbal communication performance in males with ASD. To more precisely assess the FC of these networks, we applied Principal Component Analysis (PCA) to resting-state fMRI data, extracting the most significant patterns of spontaneous activity [5, 56]. This approach allowed us to define regions of interest (ROIs) specific to our sample for each network. Using PCA to identify network ROIs tailored to the sample enhances the accuracy of FC estimation by focusing on the most relevant spontaneous activity patterns for the particular dataset rather than relying solely on predefined anatomical regions [52, 61]. Following between-group comparisons, we constructed brain-behavior models to estimate which network connections contribute to deficits in verbal and nonverbal communication in the ASD group.

Methods and materials

Participants

This study was conducted using resting-state fMRI data from the Autism Brain Imaging Data Exchange II dataset [12]. As our focus was on both verbal and nonverbal communication, we selected subsamples that included the corresponding subscales from the Autism Diagnostic Interview-Revised (ADI-R; [33]). Our initial search identified data from nine sites. To standardize data acquisition and facilitate further preprocessing, we chose the dataset with the largest number of participants diagnosed with ASD. Consequently, our sample was drawn from two datasets provided by the Langone Medical Center at New York University, comprising 75 individuals with ASD and 30 typically developing (TD) controls. To ensure balanced groups, we excluded female participants due to their low representation (only eight females with ASD and two TD females). Next, we conducted outlier detection and removal based on predefined criteria: values below the first quartile minus 1.5 times the interquartile range (IQR) or above the third quartile plus 1.5 times the IQR were considered outliers (where IQR = third quartile − first quartile). This process identified 9 age-related outliers, all of them were in the ASD group; these participants were excluded as well. Finally, we defined six outliers of quality control metrics of fMRI data before the denoising procedure to also exclude them from the analysis according to the same outlier criteria (Fig. 1).

The final sample consisted of 80 male participants: 53 males with ASD (age range years: 5.1−11.3, Mage=7.5, SDage=1.5) and 27 TD males (age range years: 5.9−12.9 Mage=9.0, SDage=1.8). The groups differed significantly in age, as indicated by the Mann-Whitney U test ($U=364$, $p=0.0003$). However, when assessing the equality of variance-covariance matrices using Box's M test, we found no significant difference, indicating homogeneity between groups for this variable (*Chi-square*=1.33, $p=0.25$). The following scores of the ADI-R communication subscales were obtained in the ASD group: Mv=15.4, SDv=5, ranged from 4 to 24 (with minimal and maximal possible values of 0 and 25, respectively) and Mnv=8.8, SDnv=3.7 ranged from 0 to 14 (with minimal and maximal possible values of 0 and 14, respectively). The detailed sample description is presented in Table 1.

Data preprocessing

Demographic and behavioral data were preprocessed by scaling continuous variables using the MinMaxScaler function and encoding categorical variables with the OneHotEncoder transformer from the Python scikit-learn package [47, 54]. For fMRI data, preprocessing was conducted using the *CONN toolbox* version 22.a (https://www.nitrc.org/projects/conn; [56], following a flexible pipeline [44] that included bandpass filtering between 0.008 and 0.09 Hz, motion correction, slice-timing correction, outlier detection, direct segmentation, normalization to MNI space, smoothing with an 8-mm kernel, realignment to the first scan of the first session, and independent component analysis denoising targeting displacement above 0.9 mm or global BOLD signal changes exceeding 5 standard deviations [44, 56].

ROIs generation and selection

We ran the PCA decomposition on our data to estimate 25 principal components from the fMRI data combined across all subjects. The BOLD signal from every timepoint and voxel in the brain was concatenated across subjects along the temporal dimension. A singular value decomposition of the z-score normalized BOLD signal (subject-level SVD) with 64 components separately for each subject was used as a subject-specific dimensionality reduction step. A singular value decomposition (group-level SVD) with 25 components was then used to identify spatially and temporally orthogonal networks characterizing the largest variance in the BOLD signal. Last, GICA3 back-projection [13] was used to compute PCA maps associated with these same networks separately for each individual subject. Next, we chose the PCA-created networks that most topographically and temporally matched the DMN (r = 0.26), the SN (r = 0.28), the DAN (r = 0.23), and the LN (r = 0.25). As these PCA-networks were presented by many ROIs, we decided

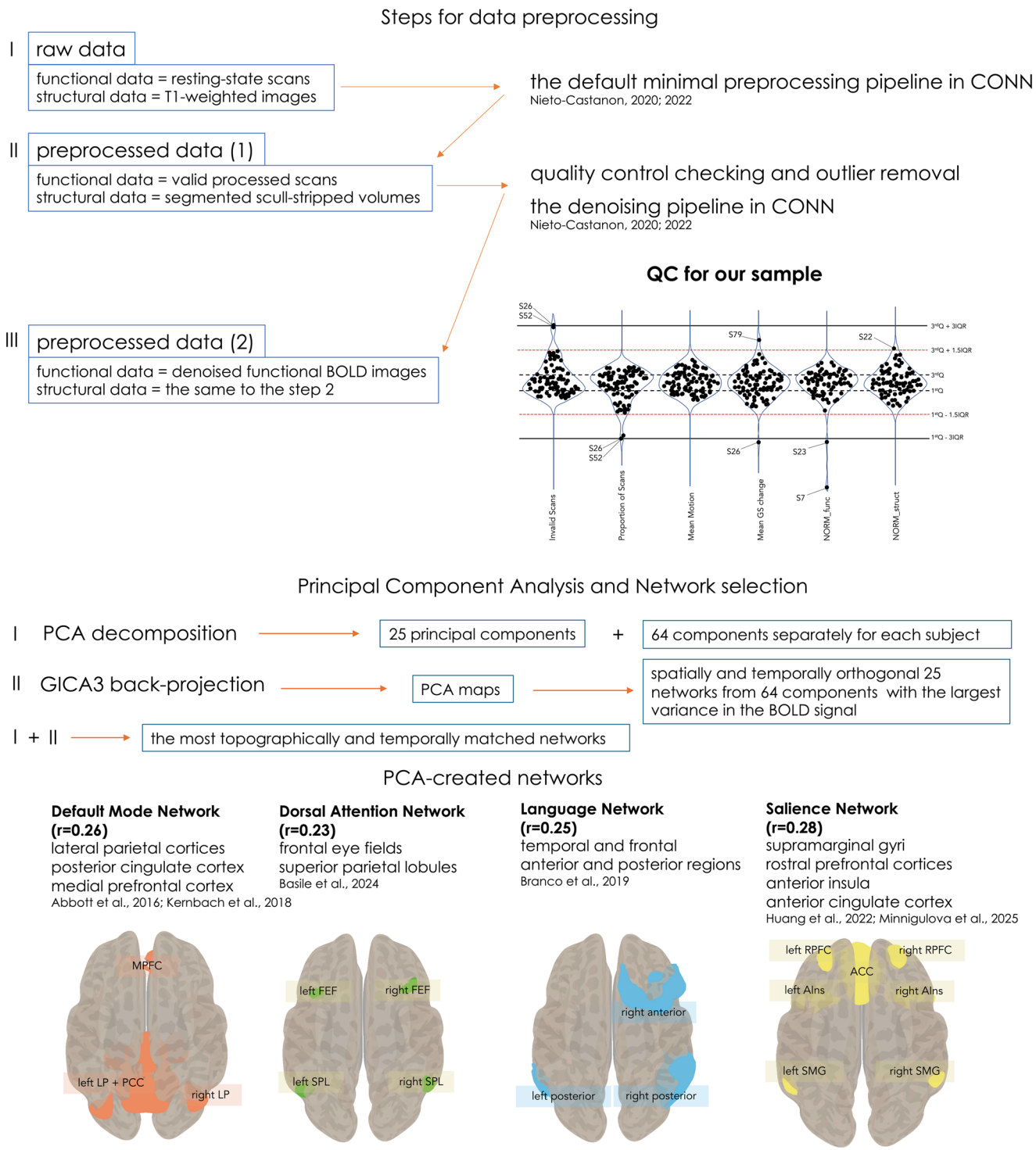

Fig. 1 The flowchart of the pre- and processing pipelines with outlier removal, networks construction and region selection. *Note*: Invalid scans = number of scans identified as outliers; Proportion of scans = ratio between valid and invalid scans; Mean motion = mean value of framewise displacement change across valid scans per subject; Mean GS change = mean value of global signal change across valid scans per subject; NORM_func = indicator of functional data normalization accuracy; NORM_struct = indicator of structural data normalization accuracy; r = correlation value of predefined network templates (CONN's ICA analyses of Human Connectome Project dataset (Whitfield-Gabrieli and Nieto-Castanon 2012 = [56]) and PCA-extracted networks; IQR = interquartile range; GICA3 back-projection = direct group independent component analysis back-reconstruction approach (Erhardt et al. 2011 = [13])

Table 1 Demographic information

Characteristic	*N* (ASD/TD)	ASD	TD	Statistics	*p*-value
Age (years)	53/27	7.5 ± 1.5 (5.1 − 11.3)	9.0 ± 1.8 (5.9 − 12.9)	$U = 364$	0.0003
FIQ[a]	52/27	104.3 ± 16.5 (73–138)	116 ± 14.8 (92–144)	$t(77) = -3.1$	0.002
ADI[b] verbal	52/0	15.4 ± 5 (4–24)	NA	NA	NA
ADI[b] nonverbal	53/0	8.8 ± 3.7 (0–14)	NA	NA	NA
SRS total (T) [c]	53/26	74.7 ± 14.5 (42–107)	45.2 ± 6.3 (34–59)	$t(77) = -3.1$	< 0.0001

Two-sample independent t-tests and Mann-Whitney U-tests were conducted to compare the mean of the demographic and behavioral data in groups of individuals with ASD and TD

[a]Full scale intelligence quotient

[b]Autism Diagnostic Interview-Revised

[c]Social Responsiveness Scale – Second Edition. The result is provided in T-scores

ASD Autism Spectrum Disorder; *TD* typical development; *NA* not available

Table 2 Regions of interests comprising the networks

Network	ROI	Coordinates			Voxels
		x	y	z	
Salience	ACC	−1	38	20	5049
	left AIns	−38	16	−5	1669
	right AIns	41	18	−5	1400
	right RPFC	27	52	25	727
	left RPFC	−28	49	24	680
	right SMG	58	−49	36	435
	left SMG	−56	−55	37	324
Default Mode	left LP + PCC	−1	−55	21	12,707
	right LP	42	−72	32	893
	MPFC	1	42	−1	295
Language	right posterior	52	−38	7	6846
	right anterior	40	20	30	4618
	left posterior	−60	−54	16	625
Dorsal Attention	left SPL	−48	−62	35	478
	right SPL	54	−58	36	365
	right FEF	39	23	47	257
	left FEF	−37	17	48	228

ACC anterior cingulate cortex; *AIns* anterior insula; *RPFC* rostral prefrontal cortex; *SMG* supramarginal gyrus; *LP* lateral parietal; *PCC* posterior cingulate cortex; *MPFC* medial prefrontal cortex; *SPL* superior parietal lobule; *FEF* frontal eye field

to select only those used by previous research. Notably, the LN was presented in our study by only three key regions (the rest of them were excluded due to the non-canonical representation) and the DMN's ROIs of posterior cingulate cortex and left lateral parietal cortex were reconstructed as a united node. The detailed description of the ROIs is presented in Table 2 as well as Fig. 1 illustrates the defined networks.

ROI-to-ROI analysis

Group-level analyses were performed using a General Linear Model (GLM; [44]). For each individual connection a separate GLM was estimated, with first-level connectivity measures at this connection as dependent variables, and groups or other subject-level identifiers as independent variables. Connection-level hypotheses were evaluated using multivariate parametric statistics with random-effects across subjects and sample covariance estimation across multiple measurements. All network nodes were used as both sources and targets, and ROI-to-ROI connections were set to a threshold by intensity of two-sided False discovery rate (FDR)-corrected $p < 0.05$. Given significant differences in age between groups, this variable was included as a covariate in all group-comparison models.

To analyze the network-communication relationships in the ASD group, we built separate GLM for each connection with the following contrasts [ASD (0); Age (0); non- and verbal ADI-R(1)]. Age was implemented as a control variable in each model as well. Connections between ROIs were thresholded based on a two-sided FDR-corrected p-value of less than 0.05 [3]. All reported p-values were FDR-corrected.

Results

The analysis revealed lower connectivity in the ASD group compared to TD peers between the SN and the LN (Fig. 2): left AIns and right anterior node, $T(77) = -4.34$ $p = 0.003$; right AIns and left posterior region, $T(77) = -3.35$, $p = 0.04$. We also found greater connectivity in the ASD group compared to TD peers between the DMN and the LN (Fig. 2): right LP and right anterior node, $T(77) = 4.51$, $p = 0.003$; right LP and right posterior region, $T(77) = 3.63$, $p = 0.02$.

The regression analysis in the ASD group to estimate the effect of non- and verbal ADI demonstrated that lower connectivity between the left FEF of the DAN and the right SMG of the SN, *beta* = −0.41, $T(49) = -3.51$, $p = 0.016$; as well as between the left SPL and the right FEF of the DAN, *beta* = −0.37, $T(49) = -3.43$, $p = 0.02$ − was associated with greater ADI verbal scores (more severe communication deficits; Fig. 3). Also, the same effect direction was found for the nonverbal ADI scale and the connectivity within the DAN between the right FEF and left SPL nodes, *beta* = −0.34, $T(50) = -3.25$, $p = 0.03$ (Fig. 3). All effect sizes of the significant effects are reported in the Table 3.

Considering IQ

For the significantly differing results of between-group comparisons and brain-behavior regressions, we added the sensitivity analysis controlling for IQ by including FIQ as a

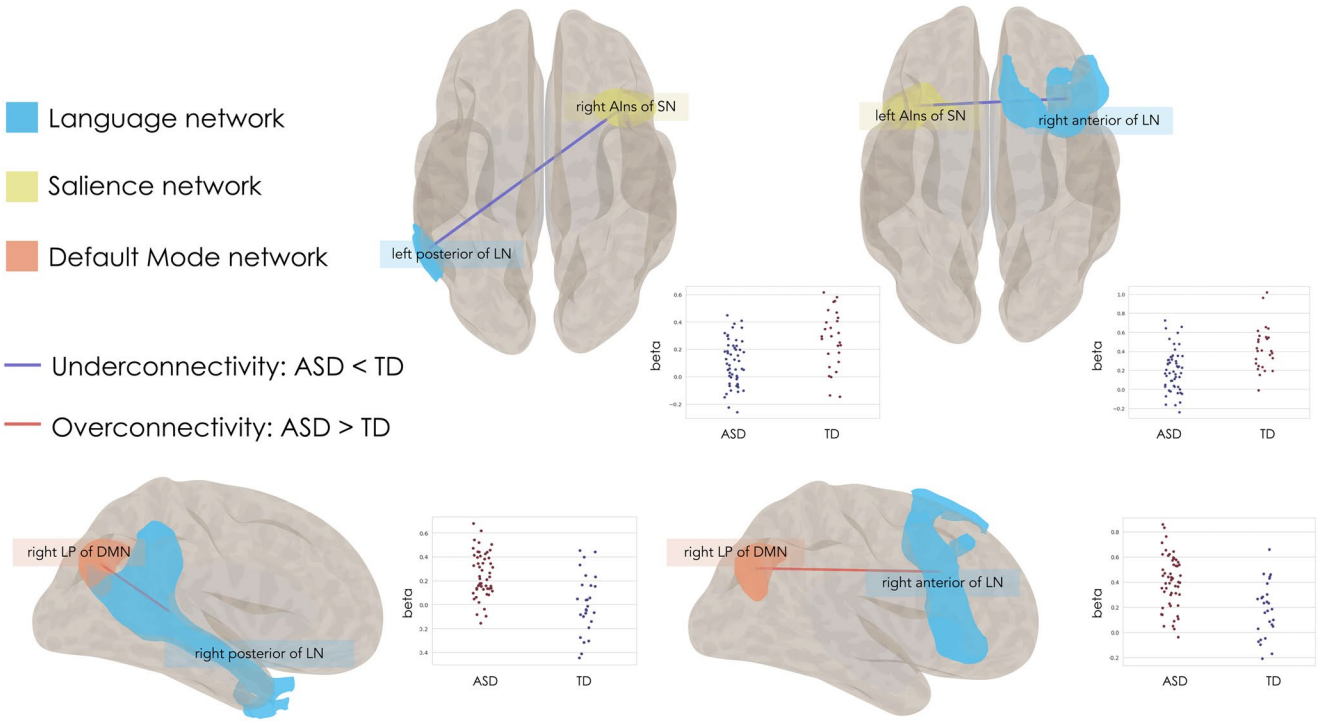

Fig. 2 Between group differences in DMN and LN, SN and LN connectivity. ROI-to-ROI functional connections in the ASD group compared to TD with significant between-groups differences $p < 0.05$: underconnectivity (blue line) between SN (yellow nodes) and LN (lightblue nodes); overconnectivity (red line) between DMN (orange nodes) and LN (lightblue nodes). Distributions of beta values for these connections are shown in the stripplots in each group. *Note:* DMN = Default Mode network; LN = Language network; SN = Salience network; ROI = region of interest; ASD = Autism Spectrum Disorder; TD = typically developing; AIns = anterior insula; LP = lateral parietal

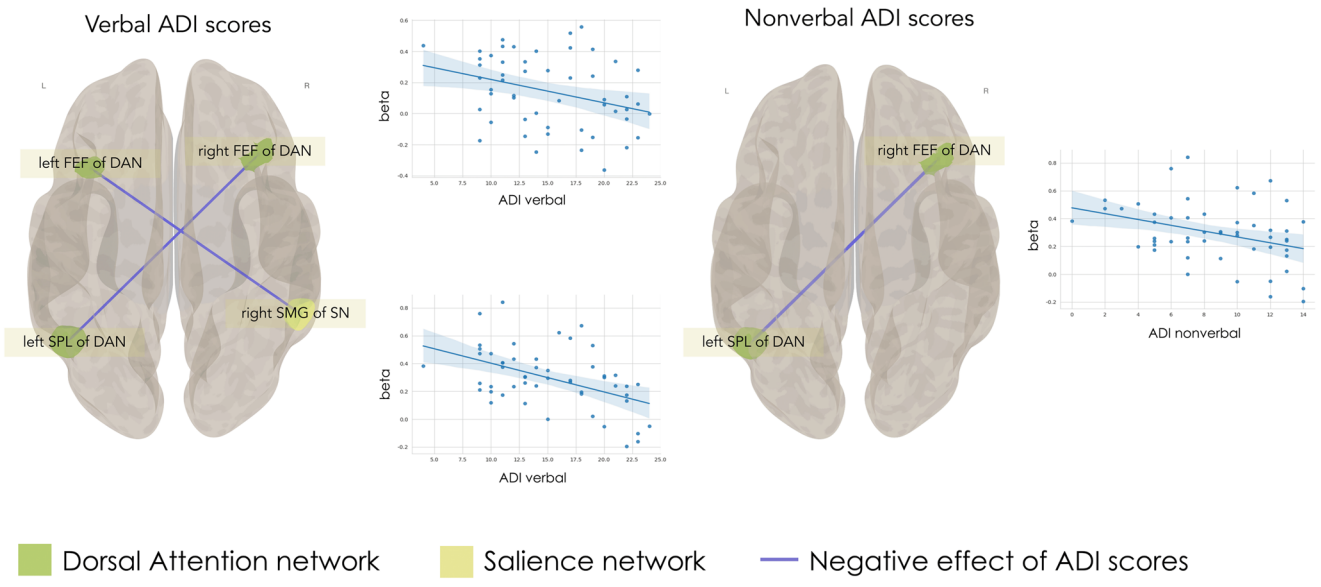

Fig. 3 The relationships between ROI-to-ROI connectivity and ADI scores in the ASD group: underconnectivity (blue line) between SN (yellow nodes) and DAN (green nodes) as well as within the DAN (green nodes). Regression plots represent relationships between beta values of these connections (y axis) and ADI scores of verbal and nonverbal communication (x axis) in the ASD group. *Note:* ROI=region of interest; ASD=Autism Spectrum Disorder; SN=Salience network; ADI=Autism Diagnostic Interview; DAN=Dorsal attention network; FEF=frontal eye field; SPL=superior parietal lobule; SMG=supramarginal gyrus

Table 3 Effect sizes for the significant effects of the between-group comparisons and ADI effect

Model	Effect	beta (standardized coefficient)	*T*-value
right anterior of LN and left AIns of SN	ASD<TD	−0.4851	−4.341
left posterior of LN and right AIns of SN	ASD<TD	−0.3841	−3.353
right anterior of LN and right LP of DMN	ASD>TD	0.4950	4.508
right posterior of LN and right LP of DMN	ASD>TD	0.4025	3.628
left FEF of DAN and right SMG of SN	ADI verbal	−0.4479	−3.506
left SPL of DAN and right FEF of DAN	ADI verbal	−0.4550	−3.428
left SPL of DAN and right FEF of DAN	ADI nonverbal	−0.4177	−3.181

LN Language network; *AIns* anterior insula; *SN* Salience network; *LP* lateral parietal; *DMN* Default Mode network; *FEF* frontal eye field; *DAN* Dorsal Attention network; *SMG* supramarginal gyrus; *SPL* superior parietal lobule; *ASD* Autism Spectrum Disorder, *TD* typically developing; *ADI* Autism Diagnostic Interview

control variable in each model. The results remain the same: decreased connectivity in the ASD group compared to TD peers between the left AIns of the SN and the right anterior node of the LN, $T(75) = -3.77$, $p = 0.003$; the right AIns of the SN and the left posterior region of the LN, $T(75) = -3.08$, $p = 0.05$. The increased connectivity was again shown between the LP of the DMN and the LN: right anterior node, $T(75) = 4.60$, $p = 0.0003$; right posterior region, $T(75) = 3.54$, $p = 0.005$. Verbal communication scores in the ASD group were predicted by underconnectivity within the DAN, $T(48) = -3.39$, $p = 0.02$, as well as between the SN and the DAN, $T(48) = -3.47$, $p = 0.02$; while lower nonverbal communication was associated with reduced connectivity within the DAN only, $T(48) = -3.14$, $p = 0.05$.

Discussion

In this study, we investigated the functioning of resting-state networks related to attention, language, and mentalizing, as well as their associations with verbal and nonverbal communication in children with ASD. Our analysis revealed a global pattern of underconnectivity between the LN and the SN, alongside overconnectivity between the LN and the DMN. Although no significant links were found between connectivity of the LN and communication performance, reduced connectivity within the DAN and between the DAN and SN was associated with more severe impairments in both verbal and nonverbal communication.

PCA's network construction

Despite applying PCA for a more precise specification of ROIs' topography, or their anatomical locations, our ROIs selection was based on previous research. However, there are two important notes on our PCA results: first, the DMN's left LP and PCC were merged into one ROI; second, the LN's left anterior part, or the typical Broca's area, was not defined as a principal component. The first observation may be explained by the highly correlated activation patterns between these DMN' nodes. The second pattern may be due to the fact that the left anterior language regions are obviously activated during task-based fMRI paradigms but during rest, these regions do not exhibit spontaneous low-frequency activity correlations to other language nodes. Thus, the LN of rest could be limited to right anterior and posterior and left posterior parts. Our network analysis did not include the left frontal region – as it was not reconstructed during the PCA – which likely explains why we found no link between the Language Network (LN) and ASD communication symptoms. Research by Lee et al. [30], Gao et al. [19], and Xu et al. [57] shows that social symptoms in ASD are actually tied to Broca's area, an area our study did not include. Conversely, our results either underscore the idea that Broca's area and its connections are essential for social communication. Future studies with application other data-drive methods are needed.

Between-group differences

As our between-group comparisons revealed overconnectivity between the LN and DMN in males with ASD, we supported the conclusion of previous studies on decreased segregation between these networks indicating lower specialization of the language system from the mentalizing one in children with ASD [19, 60]. It is important to note that the right LP node of the DMN is overconnected with both right anterior and posterior regions of the LN in our study. We may then speculate that mentalizing processes including self-referential ones and theory of mind interfere with both production and comprehension of language making them slower or challenging to process for children with ASD compared to TD peers. Our next finding is underconnectivity between the LN and SN in males with ASD. As it was mentioned before, the SN's functioning is crucial for detecting the most relevant stimuli and assembling mental and cognitive resources to switch attention between different tasks and systems [29, 36, 37]. Based on reduced connectivity between the SN and LN, we may assume that among other reasons language processing in ASD is altered due to behavioral inflexibility during input filtering

and stimuli prioritizing. These two findings contribute to understanding connectivity interactions of the LN in ASD reflecting its lower segregation, other words, poorer specification, with the mentalizing system and deficient connectivity with the attention-alerting one.

Brain-behavior associations

Despite our expectations of the significant associations between connectivity of the LN with other networks and communication abilities, we did not find them. It is worth reiterating that, following PCA-based network reconstruction and ROI selection, the LN in our study lacked representation in the left frontal region. This notable absence likely contributed to the lack of the LN involvement in our findings on communication symptoms in ASD. Lee et al. [30], Gao et al. [19] and Xu et al. [57] provide compelling evidence for this interpretation, demonstrating a significant association between ASD social symptoms and FC metrics of the left inferior frontal gyrus, or Broca's area. Notably, our own results revealed no significant effects of the LN on communication deficits, further underscoring the critical role of Broca's area and its connections in social verbal and nonverbal interactions.

Otherwise, we showed that verbal communication was predicted by underconnectivity within the DAN and between the DAN and SN, while nonverbal communication was associated with underconnectivity within the DAN alone. While the SN contributes to switching attention, the DAN is involved in sustaining attention toward the most relevant external information [4]. Therefore, we assume that if impairments of nonverbal communication are predicted by disruptions of sustained attention which entail difficulties of social cues focusing and consequent understanding of them, the difficulties of verbal communication could be explained by altered integration of sustained and switching mechanisms of attention while message transmission and interpretation.

Limitations

Our study has limitations that must be noted. First, our study was focused on males with ASD, this bias has to be considered when interpreting the results. Moreover, future research may benefit from including females with this condition for generalizing findings. Second, preprocessing differences can serve a potential reason for results' inconsistency, thus, future studies will benefit from the comparison of the preprocessing pipelines. Third, we conducted procedures of outliers' removal and quality control, but we did not account for motion and other artifacts left after the preprocessing steps if any while building our models. Fourth, as we did not build age-effect models, we prioritized a larger sample with substantial age differences between groups. Including age as a covariate ensured homogeneity of covariance matrices; however, some residual age effects might still influence the results. Fifth, our study focused on resting-state data only, future studies may benefit from replicating the findings using task-based fMRI with verbal and nonverbal communication tasks in larger, multi-site and more demographically heterogeneous cohorts. Sixth, our study does not include information regarding the socio-economic status (SES) of the participants, as these data were not available in the ABIDE II dataset. While SES is not always a mandatory variable for functional connectivity analysis, providing this information would have allowed for a more comprehensive description of the sample and helped to further contextualize the findings.

Conclusions

Our study may serve as one of the possible explanations of communication difficulties in the ASD population. Generally, we showed the altered connectivity patterns of the language system with mentalizing and alerting-attention ones in males with ASD. However, we assume that impairments of verbal and nonverbal communication in ASD have more attentional nature of disruptions rather than language one.

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Author contributions AM: methodology, investigation, data curation, formal analysis, writing—original draft, and writing—review and editing. OD: writing—review and editing, resources. VA: writing—review and editing, and project administration. All authors read and approved the final manuscript.

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Data availability No datasets were generated or analysed during the current study.

Declarations

Competing interests The authors declare no competing interests.

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