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ORIGINAL ARTICLE

Atypical Social Behavior is Predicted by Overconnectivity Between Salience and Default Mode Networks in Autism Spectrum Disorder

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Abstract
As Default Mode and Salience networks (DMN, SN) contribute to social behavior and switching between inner and outer attention, they are believed to function and develop differently in individuals with Autism Spectrum Disorder (ASD). However, it remains unclear what alterations of their interactivity are connected to certain autistic traits and how age influences these networks' maturing. Behavioral (social responsiveness, executive functions and communication skills) and resting-state functional connectivity (FC) data from the Autism Brain Imaging Data Exchange were analyzed comprising individuals with ASD ($n=144$) and healthy controls ($n=99$). We compared FC between the groups investigating DMN and SN separately and in combination. Finally, we assessed FC-behavior links in the ASD group and age effects on FC across these networks in both samples. Individuals with ASD exhibited increased FC between DMN and SN but decreased one within DMN compared to the control group. FC between right insular and medial prefrontal cortices predicted more severe social responsiveness impairments in ASD but there were no significant associations with executive functions nor adaptive behavior. Additionally, DMN and SN matured in ASD with partly different patterns than in typical development. Our results replicated and expanded previous findings on DMN and SN pointing to robust differences within and between these networks in ASD and their contribution to autistic traits regarding social responsiveness.

Keywords Default mode network · Salience network · Autism spectrum disorder · Resting state fMRI · Autistic traits

Introduction

Underconnectivity and overconnectivity between and within Default Mode network (DMN) and Salience network (SN) are not only reported in individuals with Autism Spectrum Disorder (ASD), but also are found to be associated with core and co-occurring conditions of ASD; particularly, with sensory, communication and social responsiveness difficulties (Abbott et al., 2016; Chen et al., 2022). Although previous studies have focused on DMN (see Padmanabhan et al., 2017 for review; Cong et al., 2023) and SN (Guo et al., 2023; Marshall et al., 2020) separately and jointly (Abbott et al., 2016; Chen et al., 2022), connectivity patterns remain mixed across group comparisons and show inconsistent relationships with behavior. The heterogeneity of findings may stem from variations in ASD phenotypes and imbalances in sample demographics (Chen et al., 2022). Additionally, there is limited information on how connectivity dynamics of these networks evolve across ages, particularly from childhood to adolescence (Funakoshi et al., 2016; Jung et al., 2014; Lynch et al., 2013; Weng et al., 2010).

Atypical functional connectivity patterns of DMN were connected with social traits of ASD (Li et al., 2014) due to its crucial role in self-referential processing and mentalizing (Mars et al., 2012). Specifically, overconnectivity between DMN nodes was found in children with ASD (Funakoshi et al., 2016; Lynch et al., 2013), while underconnectivity was shown in groups of adolescents and adults with this condition (Jung et al., 2014; Weng et al., 2010). Altered connectivity between SN regions was hypothesized to be associated with behavioral inflexibility in individuals with ASD (Marshall et al., 2020). For instance, greater

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connectivity of SN was found in children with ASD compared to typically developing (TD) peers and was related to decreased verbal intelligence but not social characteristics (Margolis et al., 2019). In other studies, overconnectivity of SN was otherwise shown to be connected with sensory and sociocommunicative traits (Abbott et al., 2016) and to predict restricted and repetitive actions (Uddin et al., 2013).

Regarding DMN and SN interactivity, Chen et al. (2022) reported increased connectivity in ASD between medial prefrontal cortex (MPFC), a DMN node, and right anterior insula (rAIns), an SN region to be associated with reduced social responsiveness in individuals with ASD. Nevertheless, no differences between DMN and SN were pointed out in the study comparing children and adolescents with and without ASD (Abbott et al., 2016). One more finding was carried out in the aspect of the SN-DMN connectivity while comparing children and young adolescents with ASD and schizophrenia that in contrast to the schizophrenia phenotype, people with ASD exhibit mainly atypical intra-SN connections than inter DMN-SN ones (Chen et al., 2017). Another study on the similar age group demonstrated greater functional connectivity between regions of DMN and SN in ASD in compare with TD peers (Yerys et al., 2015).

Previous studies investigated connectivity within DMN and SN separately (Jung et al., 2014; Lynch et al., 2013; Weng et al., 2010; Funakoshi et al., 2016; Margolis et al., 2019; Marshall et al., 2020) and together (Abbott et al., 2016; Chen et al., 2017, 2022). Some of them even proved that connectivity metrics of DMN and SN contribute to classification of ASD from TD peers (Chen et al., 2017). Thus, detailed connectivity patterns of these networks, their relationships to behavior and age-related effects on these connections are in demand for research.

The typical connectivity between these resting-state networks is an indicator of successfully ongoing mentalizing processes (Lee & Frangou, 2017). More specifically, it has been shown that structural and functional connections between DMN and SN are positively associated with extraversion, agreeableness, and self-perceived empathy (Coutinho et al., 2013; Esménio et al., 2019; Sampaio et al., 2014). Thus, detailed understanding of alterations in their connectivity patterns may shed light on disruptions to internal mental states and subjective experience in individuals with ASD.

The aims of this study were threefold. First, we addressed the question of between group differences of DMN and SN. Second, we examined whether this atypical connectivity was related to core (social characteristics) and co-occurring (atypical executive functions and adaptive behavior) conditions of ASD. Third, we investigated age-related patterns of connectivity changes across individuals with and without ASD. As Chen et al. (2022) demonstrated overconnectivity between MPFC and rAIns, we expected to replicate these results and to show more between-network overconnectivity but within-network underconnectivity patterns of DMN and SN in individuals with ASD. Next, we hypothesized to find the relationship between DMN-SN overconnectivity and reduced social responsiveness as Chen et al. (2022) did. As SN is theorized to switch between internal to external stimuli and vice versa in order to guide behavior (Menon & Uddin, 2010), thus, we strongly believe that these transitional alterations may implicate executive functions and adaptive behavior difficulties. Moreover, age-related dynamics of these networks were anticipated to differ between individuals with and without ASD.

Methods and Materials

Autism Resting-State fMRI Dataset and Preprocessing

A total of 239 participants were included in the current study: 141 individuals with ASD (19 females, age range – 5.1–17.9, M_{age}= 9.93, SD_{age}= 3.1) and 98 typically developing peers (TD; 32 females, age range – 5.9–17.2, M_{age}= 10.65, SD_{age}= 2.53). This study was conducted using resting-state (rs-) fMRI data from the Autism Brain Imaging Data Exchange II (https://www.fcon_1000.projects.nitrc.org/indi/abide/abide_II.html; Di Martino et al., 2014). We only included subsets with 3 Tesla Siemens Magnetic Resonance Imaging (MRI) tomography and scanning procedure of resting-state functional MRI (fMRI) with 2 ms repetition time to minimize data acquisition variability. It resulted in three samples collected by Georgetown University, Langone Medical Center of New York University and University of California Davis (see *GU scan parameters*, *NYU scan parameters*, *UCD scan parameters*, for details). The severity of autistic traits, executive functions, and adaptive behavior was assessed using the Social Responsiveness Scale (SRS; Constantino & Gruber, 2012), Behavior Rating Inventory of Executive Functions (BRIEF; Gioia et al., 2000), and Vineland Adaptive Behavior Scales (VABS; Sparrow & Cicchetti, 1989), respectively. At the included sites, researchers instructed all ASD participants to refrain from taking medications for at least 24 h prior to MRI scanning. In the UCD subsample, participants taking antipsychotic medications and serotonin selective reuptake inhibitors over the three months prior to the scan, as reported by their parents, were excluded. Prior to analysis, we performed outlier detection and removal using predefined criteria (outlier criteria: < first quartile (25%) − 1.5 * interquartile range and > third quartile (75%) + 1.5 * interquartile range where interquartile range = third quartile - first quartile). There were four age-outliers (three of them

Table 1 Demographic information for individuals with ASD and TD, M ± SD (range)

Variable	*N* (ASD/TD)	ASD	TD	Statistics	*p*
Age (years)	141/98	9.93 ± 3.1 (5.1–17.9)	10.65 ± 2.53 (5.9–17.2)	t(237)= -1.91	0.06
SRS total (T) [a]	141/97	76.11 ± 14.96 (42–116)	44.22 ± 6.46 (34–66)	U = 13371.5	< 0.001
BRIEF GEC (T) [b]	120/81	65.95 ± 11.26 (35–96)	46.01 ± 8.76 (30–71)	t(199) = 13.42	< 0.001
VABS (sum)[c]	72/28	286.29 ± 56.96 (166–400)	328.96 ± 49.22 (258–441)	t(98)= -3.49	0.001
				Chi-squared	*p*
Sex (male/female)		122/19	66/32	11.55	< 0.001

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

[b]Behavior Rating Inventory of Executive Function, Global Executive Composite. The result is provided in T-scores

[c]Sum of Vineland-II Standard Scores. Includes the following domains: Communication, Daily Living Skills, Socialization, Motor Skills

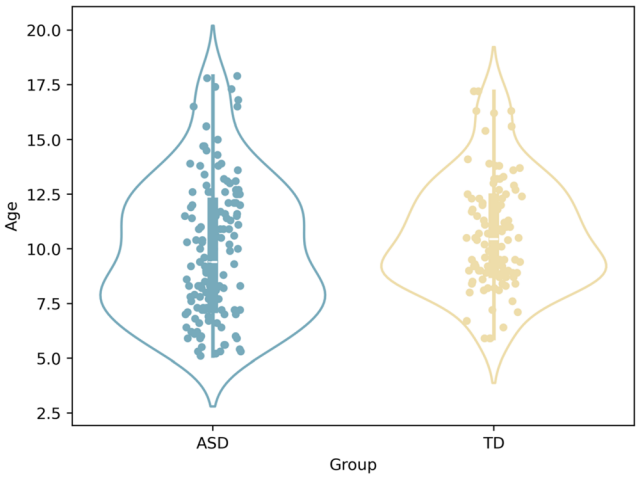

Fig. 1 Age distribution of the participants included in the age effect analysis

in the ASD group), thus, we excluded these participants who were older than 18 years. The demographic and behavioral information is provided in Table 1. Figure 1 shows the age distribution in each subsample.

Preprocessing of demographic and behavioral data included scaling for continuous variables using the MinMaxScaler function and encoding for categorical ones with the OneHotEncoder transformer from the Python3 (Van Rossum & Drake, 2009) scikit-learn package (Pedregosa et al., 2011). Preprocessing of rs-fMRI data was performed using the CONN toolbox Version 17.f (https://www.nitrc.org/projects/conn; Whitfield-Gabrieli & Nieto-Castanon, 2012) following a flexible preprocessing pipeline (Nieto-Castanon, 2020). The procedure included a 0.008 and 0.09 Hz bandpass filter, motion correction, slice-timing correction, outlier detection, direct segmentation, MNI-space normalization, 8-mm smoothing, realigning to the first scan of the first session, independent component analysis denoising (displacement above 0.9 mm or global BOLD signal changes above 5 standard deviations). The Supplementary file 1 provides the detailed rs-fMRI analysis description for our sample. Moreover, a recent study of Morfini and colleagues (2023) comprehensively illustrated all the steps for the processing pipeline we followed.

Data Analysis

Regions of interest (ROIs) within DMN and SN were defined by CONN's (Whitfield-Gabrieli & Nieto-Castanon, 2012) ICA analyses of the Human Connectome Project dataset (https://www.humanconnectome.org/). DMN comprised of MPFC (2,55,-3), left and right lateral parietal (r/lLP; -39,-77,33; 47,-67,29), posterior cingulate cortex (PCC; 1,-61,38). The ROIs of SN were l/rAIns (-44,13,1; 47,14,0), l/r rostral PFC (l/rRPFC; -32,45,27; 32,46,27), l/r supramarginal gyrus (SMG; -60,-39,31; 62,-35,32). Table 2 provides the ROIs the of DMN and the SN used in the previous studies investigated these networks jointly in ASD.

ROI-to-ROI connectivity (RRC) analysis was performed for each pair of ROIs independently using General Linear Models (GLMs) to correlate the mean BOLD time-series at the single-subject level, resulting in ROI-to-ROI functional connectivity matrices consisting of Fisher-transformed bivariate correlation coefficients (z-scored). We also implemented these variables as control variables in each group-comparison model. 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 $p < 0.05$.

To analyze the network-behavior relationships in the ASD group, we implemented the following contrasts [ASD

Table 2 Regions of interests comprising default mode and salience networks in previous research

Study	Analysis	ROIs of DMN	ROIs of SN
Yerys et al. (2015)	Seed-based	PCC MPFC l/r AG l/r hippocampus	-
Abbott et al. (2016)	ROI-to-ROI Seed-based	PCC MPFC l/r AG	l/rAIns dACC
Chen et al. (2017)	ROI-to-ROI	160 MNI coordinates across the brain defined by Dosenbach et al. (2010)	
Kernbach et al. (2018)	Factor (Latent Dirichlet Allocation)	dMPFC PMC l/rTPJ	l/rAIns MDC amygdala
Chen et al. (2022)	Seed-based	MPFC	-

l/r = left/right; PCC = posterior cingulate cortex; MPFC = medial prefrontal cortex; AG = angular gyrus; dMPFC = dorsal medial prefrontal cortex; PMC = posteromedial cingulate cortex; TPJ = temporoparietal junction; AIns = anterior insula; dACC = dorsal anterior cingulate cortex; MDC = midcingulate cortex.

(0); Sex (0); Age (0); SRS/BRIEF/VABS (1)]. For the age effect analysis, we used [ASD/TD(0); Age (1); Sex (0)] contrasts in each group.

In this study, we applied the FDR correction to control for multiple comparisons, and we set our statistical significance threshold at $p<0.05$ after the FDR correction (Benjamini & Hochberg, 1995). All reported p-values were FDR-corrected.

Results

Generally, the groups differed in their SRS and BRIEF performances. Participants with ASD had greater SRS scores (Constantino & Gruber, 2012), M_{TD}= 44.22 (SD=6.46) vs. M_{ASD} = 76.11 (SD=14.96), U=13371.5, $p<0.001$. They also varied in executive functions assessed with BRIEF (Gioia et al., 2000), M_{TD}= 46.01 (SD=8.76) vs. M_{ASD} = 65.95 (SD=11.26), $t(199)=13.42$, $p<0.001$ and adaptive behavior measured with VABS (Sparrow & Cicchetti, 1989), M_{TD}= 328.96 (SD=49.22) vs. M_{ASD} = 286.29 (SD=56.96), $t(98)$ = -3.49, $p=0.001$ (see Table 1).

The analysis revealed greater connectivity in the ASD group compared to TD peers between MPFC (DMN) and rAIns (SN), $T(235)=2.97$, $p=0.03$, as well as MPFC (DMN) and rRPFC (SN), $T(235)=2.70$, $p=0.04$ (Fig. 2). Reduced connectivity in the ASD group was observed between lLP (DMN) and rLP (DMN), $T(235)$ = -3.55, $p=0.004$, as well as lLP (DMN) and PCC (DMN), $T(235)$ = -2.77, $p=0.03$ (Fig. 2).

For the regression analysis in the ASD group to estimate the effect of SRS, we chose the connections that differed between the groups. The analysis revealed that greater connectivity between MPFC (DMN) and rRPFC (SN) positively

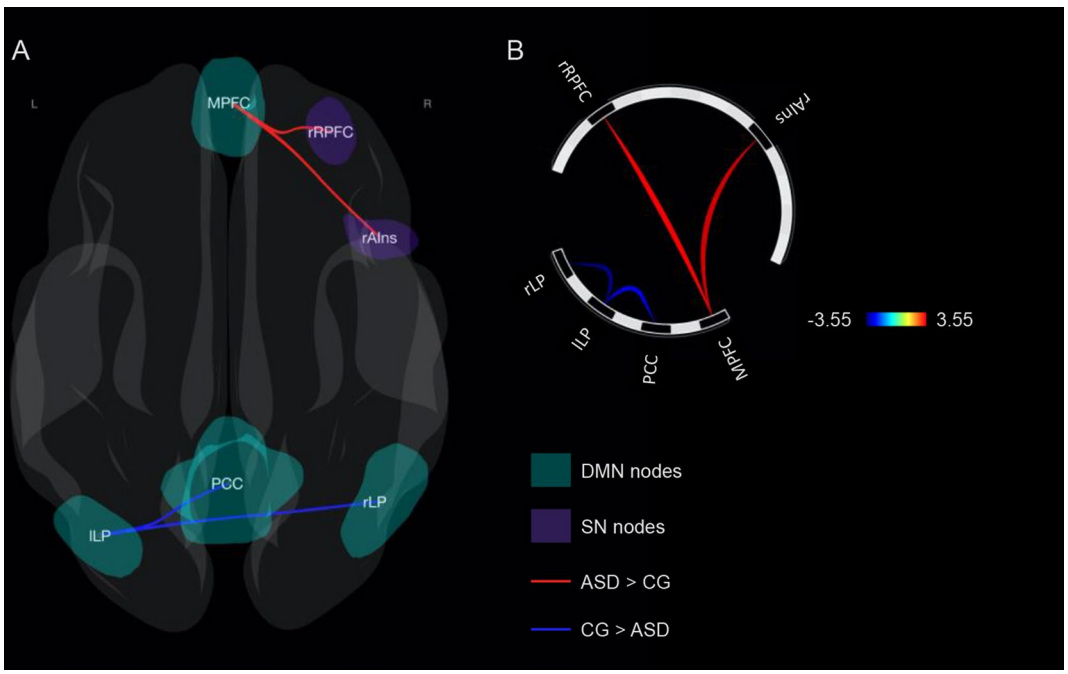

Fig. 2 Between group differences in DMN and SN connectivity. ROI-to-ROI functional connections in the ASD group compared to TD with significant between-groups differences based on the criteria of FDR-corrected $p<0.05$: (**A**) overconnectivity (red line) between DMN (aquamarine nodes) and SN (purple nodes), underconnectivity within DMN; (**B**) chord diagram with both weight and color indicating the strength of the between group differences on MPFC and rRPFC, MPFC and rAIns, PCC and lLP, lLP and rLP connections

predicted the SRS score $T(137)=2.69$, $p=0.04$ (Fig. 3). No BRIEF or VABS effects were found in DMN, SN or their joint connections.

In the TD group, greater age predicted weakening of the connection between PCC (DMN) and MPFC (DMN), $T(95) = -3.18$, $p=0.02$ (Fig. 4).

With age increasing in the ASD group, the connection between PCC (DMN) and MPFC (DMN), $T(138) = -3.88$, $p=0.002$ is weakening- as in the TD one. Additionally, greater age in ASD predicted weakening of connections between PCC (DMN) and rLP (DMN), $T(138) = -3.12$, $p=0.02$; MPFC (DMN) and rAIns (SN), $T(138) = -2.56$, $p=0.05$; MPFC (DMN) and rRPFC (SN), $T(138) = -2.37$, $p=0.05$; MPFC (DMN) and ACC (SN), $T(138) = -2.34$, $p=0.05$ (Fig. 5).

Supplementary file 2 provides the scatterplots of the age- and SRS-related effects, as well as the effect sizes of the results.

Discussion

In this study, we investigated DMN and SN connectivity in individuals with ASD. Our main findings are hyperconnectivity between anterior parts of DMN (MPFC) and SN (rRPFC, rAIns), as well as hypoconnectivity between posterior ROIs of DMN (lLP to PCC and rLP). Furthermore, we associated MPFC-rRPFC overconnectivity with social responsiveness in ASD. As we were interested in age-related effects, we investigated how age influenced the DMN-SN connectivity and demonstrated differing patterns of connectivity maturation.

Overall, we showed within network underconnectivity and between network overconnectivity in ASD. Our result of overconnectivity between MPFC-rAIns is consistent with Chen et al. (2022) at the group level possibly indicating less oriented interests to external events in individuals with ASD. However, while Chen et al. (2022) found increased MPFC-rAIns connectivity to be associated with reduced social responsiveness, we did not find this relationship to be significant. In the current study, overconnectivity between MPFC and rRPFC contributed to reduced social responsiveness. Further, we also showed this connection's weakening with age in the ASD population but not across TD children and adolescents. Increased DMN-SN connectivity appears linked to reduced social responsiveness, potentially reflecting an imbalance between self-referential processing (Davey et al., 2016) and external input filtering (Ku et al., 2020); however, there is a tendency for age-related weakening of this overconnectivity. Thus, overconnectivity between DMN and SN in the ASD population may result in abnormally rapid, subsequently, unrobust switching from resting mentalizing (Davey et al., 2016) to external attention (Ku et al., 2020) modes and vice versa. Notably, our findings of DMN-SN overconnectivity contradict prior studies that reported no differences at the group level among children

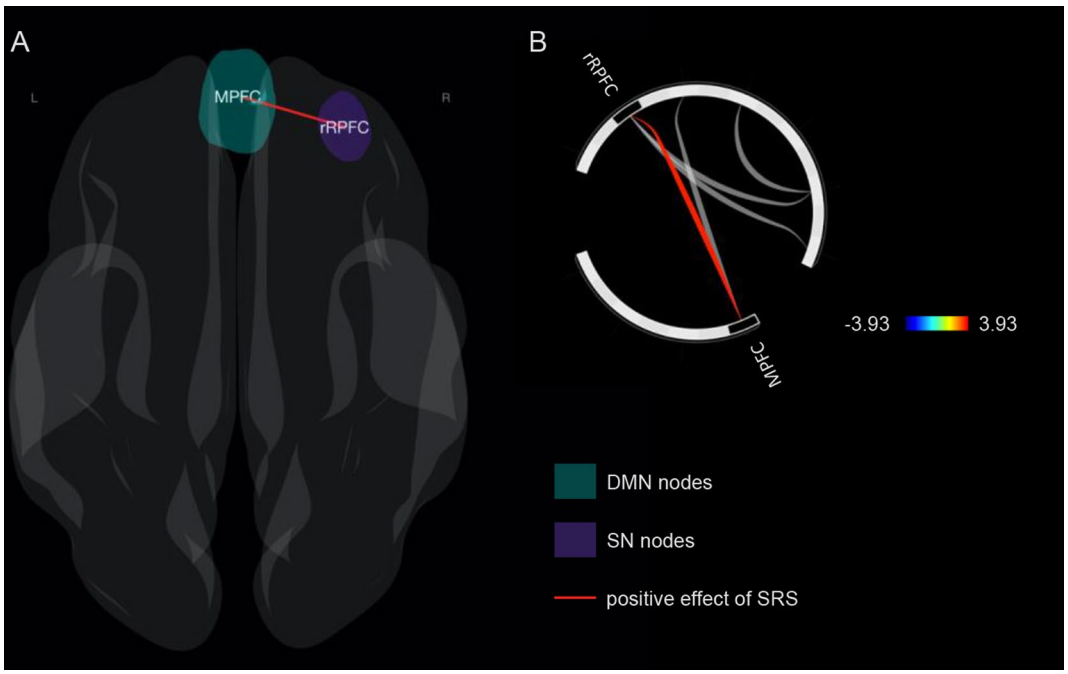

Fig. 3 SRS-related effect in ASD individuals across connections differed between groups. ROI-to-ROI functional connections in the ASD group with significant SRS-effect based on the criteria of FDR-corrected $p<0.05$: (**A**) positive effect of SRS (red line) on connections between DMN (aquamarine nodes) and SN (purple nodes); (**B**) chord diagram with both weight and color indicating the strength of the SRS-related effect on MPFC and rRPFC connection

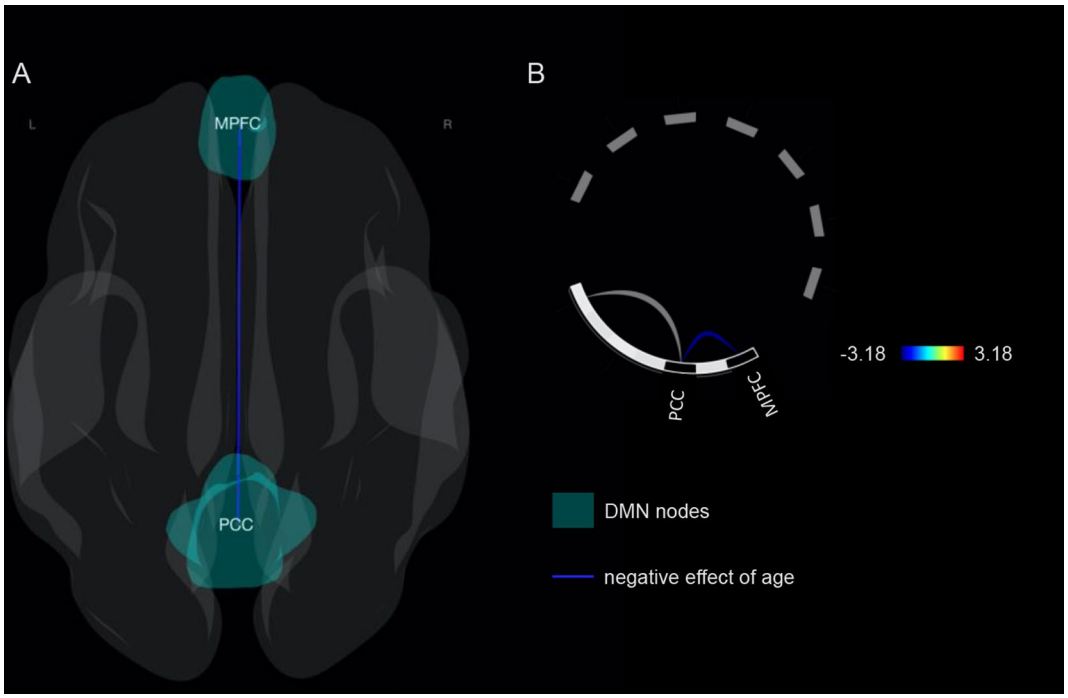

Fig. 4 Age-related effects in TD. ROI-to-ROI functional connections in the TD group with significant age-effect based on the criteria of FDR-corrected $p < 0.05$: (**A**) negative (blue line) effects of age on connections between DMN (aquamarine nodes); (**B**) chord diagram with both weight and color indicating the strength of the age-related effects on rLP and PCC, PCC and MPFC connections

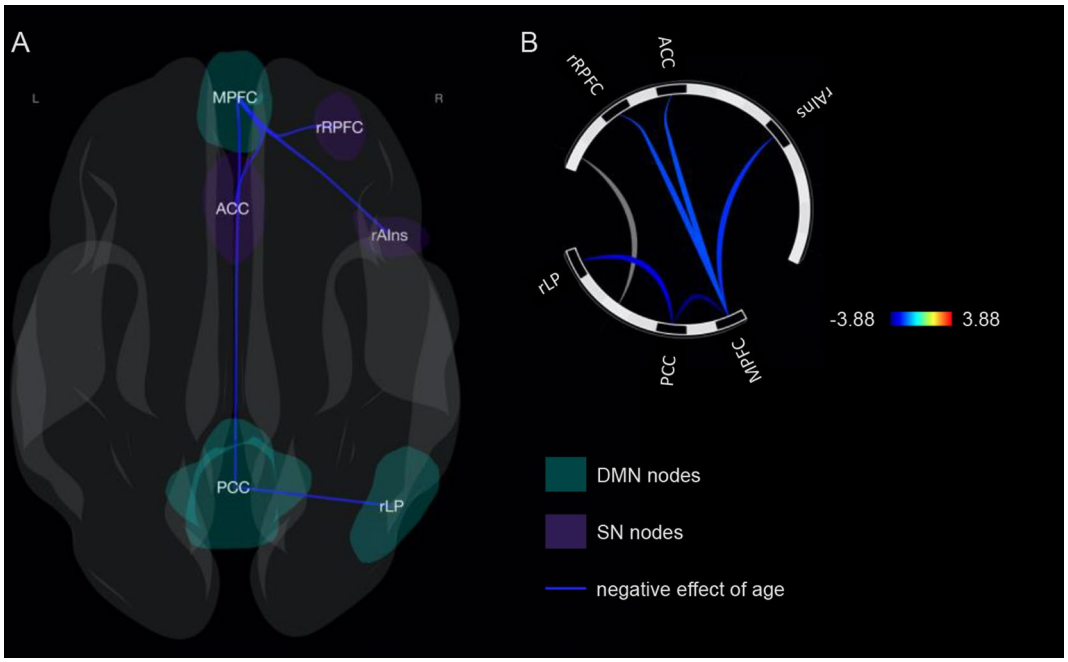

Fig. 5 Age-related effects in ASD. ROI-to-ROI functional connections in the ASD group with significant age-effect based on the criteria of FDR-corrected $p < 0.05$: (**A**) negative effect of age (blue line) on connections between DMN (aquamarine nodes) and SN (purple nodes); (**B**) chord diagram with both weight and color indicating the strength of the age-related effect between MPFC and rAIns, MPFC and PCC, PCC and rLP, lLP and lRPFC

with ASD (Abbott et al., 2016). However, the age range of the participants in the present study is broader, thus, DMN-SN overconnectivity may be not specific for children with ASD but still be present in the ASD phenotype.

While connectivity of DMN was shown to be increased in children with ASD (Funakoshi et al., 2016; Lynch et al., 2013), our result of general reduced underconnectivity in individuals with ASD is in line with those who also concluded it to be decreased in adolescents and adults with this condition (Jung et al., 2014; Weng et al., 2010). Reduced DMN connectivity may contribute to atypical social cognitive processes associated with ASD (Gusnard et al., 2001) and theory of mind (Schilbach et al., 2008).

The initial hypothesis on between-network over- but within-network underconnectivity with our findings of increased DMN-SN and decreased DMN connectivity contribute to the disrupted network segregation framework. More precisely, spontaneous brain activity measured by fMRI is organized in large-scale networks of regions with low-frequency temporal coherency (Liu et al., 2011). There is an assumption and previous studies' results (Fishman et al., 2015; Yang et al., 2023) that these resting-state networks are less isolated and modular in individuals with ASD compared to those without ASD, which reflects poor module segregation. Specifically, we confirmed the previous findings on aberrant functional connectivity of DMN-SN and supported the conclusion of Kernbuch et al. (2018) that these atypicalties may be a reason for impairments of processing emotions of self and others as well as of reorienting attention from internal and external social/emotional stimuli.

In the present study, we also expected executive functions and adaptive behavior difficulties to be related to DMN-SN connectivity. However, we showed no significant associations between these metrics. Future studies may benefit from investigating these behavioral traits with respect to not only DMN-SN disruptions but adding sensory and memory networks for adaptive behavior (Karlaftis et al., 2021), and executive control networks, such as frontoparietal one, for executive functions (Engelhardt et al., 2019).

Apart from the same age-related pattern of MPFC-PCC weakening within DMN, there are four specific changes occurring in ASD but not in TD. First, there is a cluster of connections getting weaker with age between DMN and SN from childhood to adolescence in ASD. The cluster comprised of the connections from MPFC (DMN) to ACC, rRPFC, and rAIns. It is important to note that cross-sectionally two of them were overconnected in individuals with ASD compared to TD. Thus, weakening of these connections from childhood to adolescence in ASD may be an indicator of network segregation, a process described in typical neurodevelopment, during which the connections between regions of canonical intrinsic connectivity networks are becoming less coordinated and synchronized despite their topographical neighborhood, allowing networks to get more isolated (Dosenbach et al., 2010; Grayson & Fair, 2017). In other words, we assume that the maturation processes of DMN-SN in ASD are partly similar to TD but occur with a delay. Second, there are two weakening connections within DMN; one of those occurs both in ASD and TD – PCC-MPFC, another one is demonstrated for ASD only – PCC-rLP. Both of these processes may be explained by synaptic pruning during, which networks are diminishing in connections by removing unused ones and leaving essential ones in order to maximize efficiency (Stirrups, 2018). We may speculate that the synaptic pruning for short-range connections, such as the PCC-rLP one is going faster than for long-range connections, such as the PCC-MPFC one. Because of the maturation delay in ASD, we observed both of these connections weakening in ASD but only one of them in TD. This finding may be important for understanding the nature of connectivity alterations, or latencies, in ASD.

Our study not only adds to the existing body of literature on between-network connectivity during rest in individuals with ASD but also may serve as one of the possible explanations of mentalizing difficulties in this population. Thus, investigating neural networks brings research closer to finding biomarkers of ASD conditions generally, and particular symptoms specifically. In line with Yang et al. (2023), we showed underconnectivity of DMN in individuals with ASD and confirmed their finding of a less segregated DMN as a valid biomarker of ASD. Although our study is cross-sectional and future longitudinal ones are needed, we found the ASD-specific differences in spontaneous brain activity organization with age, which may be applied in the development of treatment and therapy.

Our study has limitations that must be noted. First, our samples are unequal in sex, which is common for ASD studies, however, this bias has to be considered when interpreting the findings. Second, we did not account for site effects, motion and other artifacts left after the preprocessing steps if any while building our models. Third, preprocessing differences can serve a protentional reason for results' inconsistency, thus, future studies will benefit from the comparison of the preprocessing pipelines. In conclusion, these findings offer additional insights regarding altered functional connectivity in ASD that is associated with reduce of social responsiveness. This study highlighted the importance of investigating not only single regions or networks but their interactivity. Future studies may benefit from task-based fMRI paradigms directed to assessing core and co-occurring conditions of ASD. Also, broader age samples may provide additional information on DMN and SN as they develop and change over time with age.

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Author Contributions AM: methodology, investigation, data curation, formal analysis, writing—original draft, and writing—review and editing. MP: formal analysis, 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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Declarations

Disclosures All other authors report no biomedical financial interests or potential conflicts of interest.

References

Abbott, A. E., Nair, A., Keown, C. L., Datko, M., Jahedi, A., Fishman, I., & Müller, R. A. (2016). Patterns of atypical functional connectivity and behavioral links in autism differ between default, salience, and executive networks. *Cerebral Cortex*, *26*(10), 4034–4045.

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society: Series B (Methodological)*, *57*, 289–300. https://doi.org/10.1111/j.2517-6161.1995.tb02031.x

Chen, H., Uddin, L. Q., Duan, X., Zheng, J., Long, Z., Zhang, Y., & Chen, H. (2017). Shared atypical default mode and salience network functional connectivity between autism and schizophrenia. *Autism Research*, *10*(11), 1776–1786. https://doi.org/10.1002/aur.1834

Chen, Y. Y., Uljarevic, M., Neal, J., Greening, S., Yim, H., & Lee, T. H. (2022). Excessive functional coupling with less variability between salience and default mode networks in autism spectrum disorder. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *7*(9), 876–884. https://doi.org/10.1016/j.bpsc.2021.11.016

Cong, J., Zhuang, W., Liu, Y., Yin, S., Jia, H., Yi, C., & Zhang, T. (2023). Altered default mode network causal connectivity patterns in autism spectrum disorder revealed by Liang information flow analysis. *Human Brain Mapping*, *44*(6), 2279–2293. https://doi.org/10.1002/hbm.26209

Constantino, J. N., & Gruber, C. P. (2012). *Social responsiveness scale: SRS-2*. Western Psychological Services.

Coutinho, J. F., Sampaio, A., Ferreira, M., Soares, J. M., & Gonçalves, O. F. (2013). Brain correlates of pro-social personality traits: A voxel-based morphometry study. *Brain Imaging and Behavior*, *7*(3), 293–299. https://doi.org/10.1007/s11682-013-9227-2

Davey, C. G., Pujol, J., & Harrison, B. J. (2016). Mapping the self in the brain's default mode network. *Neuroimage*, *132*, 390–397. https://doi.org/10.1016/j.neuroimage.2016.02.022

Di Martino, A., Yan, C. G., Li, Q., Denio, E., Castellanos, F. X., Alaerts, K., & Milham, M. P. (2014). The autism brain imaging data exchange: Towards a large-scale evaluation of the intrinsic brain architecture in autism. *Molecular Psychiatry*, *19*(6), 659–667. https://doi.org/10.1038/mp.2013.78

Dosenbach, N. U., Nardos, B., Cohen, A. L., Fair, D. A., Power, J. D., Church, J. A., Nelson, S. M., Wig, G. S., Vogel, A. C., Lessov-Schlaggar, C. N., Barnes, K. A., Dubis, J. W., Feczko, E., Coalson, R. S., Pruett, J. R., Jr, Barch, D. M., Petersen, S. E., & Schlaggar, B. L. (2010). *Prediction of individual brain maturity using fMRI* (Vol. 329, pp. 1358–1361). Science. (New York, N.Y.)5997https://doi.org/10.1126/science.1194144

Engelhardt, L. E., Harden, K. P., Tucker-Drob, E. M., & Church, J. A. (2019). The neural architecture of executive functions is established by middle childhood. *Neuroimage*, *185*, 479–489. https://doi.org/10.1016/j.neuroimage.2018.10.024

Esménio, S., Soares, J. M., Oliveira-Silva, P., Zeidman, P., Razi, A., Gonçalves, Ó. F., Friston, K., & Coutinho, J. (2019). Using resting-state DMN effective connectivity to characterize the neurofunctional architecture of empathy. *Scientific Reports*, *9*(1), 2603. https://doi.org/10.1038/s41598-019-38801-6

Fishman, I., Datko, M., Cabrera, Y., Carper, R. A., & Müller, R. A. (2015). Reduced integration and differentiation of the imitation network in autism: A combined functional connectivity magnetic resonance imaging and diffusion-weighted imaging study. *Annals of Neurology*, *78*(6), 958–969. https://doi.org/10.1002/ana.24533

Funakoshi, Y., Harada, M., Otsuka, H., Mori, K., Ito, H., & Iwanaga, T. (2016). Default mode network abnormalities in children with autism spectrum disorder detected by resting-state functional magnetic resonance imaging. *The Journal of Medical Investigation*, *63*(3.4), 204–208. https://doi.org/10.2152/jmi.63.204

Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). Test review behavior rating inventory of executive function. *Child Neuropsychology*, *6*(3), 235–238. https://doi.org/10.1076/chin.6.3.235.3152

Grayson, D. S., & Fair, D. A. (2017). Development of large-scale functional networks from birth to adulthood: A guide to the neuroimaging literature. *Neuroimage*, *160*, 15–31. https://doi.org/10.1016/j.neuroimage.2017.01.079

Guo, X., Zhai, G., Liu, J., Zhang, X., Zhang, T., Cui, D., & Gao, L. (2023). Heterogeneity of dynamic synergetic configurations of salience network in children with autism spectrum disorder. *Autism Research*, *16*(12), 2275–2290. https://doi.org/10.1002/aur.3037

Gusnard, D. A., & Raichle, M. E. (2001). Searching for a baseline: Functional imaging and the resting human brain. *Nature Reviews Neuroscience*, *2*(10), 685–694. https://doi.org/10.1038/35094500

Jung, M., Kosaka, H., Saito, D. N., Ishitobi, M., Morita, T., Inohara, K., & Iidaka, T. (2014). Default mode network in young male adults with autism spectrum disorder: Relationship with autism spectrum traits. *Molecular Autism*, *5*, 1–11. https://doi.org/10.1186/2040-2392-5-35

Karlaftis, V. M., Giorgio, J., Zamboni, E., Frangou, P., Rideaux, R., Ziminski, J. J., & Kourtzi, Z. (2021). Functional interactions between sensory and memory networks for adaptive behavior. *Cerebral Cortex (New York N Y: 1991)*, *31*(12), 5319–5330. https://doi.org/10.1093/cercor/bhab160

Kernbach, J. M., Satterthwaite, T. D., Bassett, D. S., Smallwood, J., Margulies, D., Krall, S., Shaw, P., Varoquaux, G., Thirion, B., Konrad, K., & Bzdok, D. (2018). Shared endo-phenotypes of default mode Dsfunction in attention deficit/hyperactivity

disorder and autism spectrum disorder. *Translational Psychiatry*, *8*(1), 133. https://doi.org/10.1038/s41398-018-0179-6

Ku, J., Lee, Y. S., Kim, K. T., Chang, H., & Cho, Y. W. (2020). Alterations in salience network functional connectivity in individuals with restless legs syndrome. *Scientific Reports*, *10*(1), 7643. https://doi.org/10.1038/s41598-020-64641-w

Lee, W. H., & Frangou, S. (2017). Linking functional connectivity and dynamic properties of resting-state networks. *Scientific Reports*, *7*(1), 16610. https://doi.org/10.1038/s41598-017-16789-1

Li, W., Mai, X., & Liu, C. (2014). The default mode network and social Understanding of others: What do brain connectivity studies tell Us. *Frontiers in Human Neuroscience*, *8*, 74. https://doi.org/10.3389/fnhum.2014.00074

Liu, X., Zhu, X. H., & Chen, W. (2011). Distinction in coherent neural network between resting and working brain States. *Brain Connectivity*, *1*(5), 377–388. https://doi.org/10.1089/brain.2011.0044

Lynch, C. J., Uddin, L. Q., Supekar, K., Khouzam, A., Phillips, J., & Menon, V. (2013). Default mode network in childhood autism: Posteromedial cortex heterogeneity and relationship with social deficits. *Biological Psychiatry*, *74*(3), 212–219. https://doi.org/10.1016/j.biopsych.2012.12.013

Margolis, A. E., Pagliaccio, D., Thomas, L., Banker, S., & Marsh, R. (2019). Salience network connectivity and social processing in children with nonverbal learning disability or autism spectrum disorder. *Neuropsychology*, *33*(1), 135–143. https://doi.org/10.1037/neu0000494

Mars, R. B., Neubert, F. X., Noonan, M. P., Sallet, J., Toni, I., & Rushworth, M. F. (2012). On the relationship between the default mode network and the social brain. *Frontiers in Human Neuroscience*, *6*, 189. https://doi.org/10.3389/fnhum.2012.00189

Marshall, E., Nomi, J. S., Dirks, B., Romero, C., Kupis, L., Chang, C., & Uddin, L. Q. (2020). Coactivation pattern analysis reveals altered salience network dynamics in children with autism spectrum disorder. *Network Neuroscience*, *4*(4), 1219–1234. https://doi.org/10.1162/netn_a_00163

Menon, V., & Uddin, L. Q. (2010). Saliency, switching, attention and control: A network model of Insula function. *Brain Structure & Function*, *214*(5–6), 655–667. https://doi.org/10.1007/s00429-010-0262-0

Morfini, F., Whitfield-Gabrieli, S., & Nieto-Castañón, A. (2023). Functional connectivity MRI quality control procedures in CONN. *Frontiers in Neuroscience*, *17*, 1092125. https://doi.org/10.3389/fnins.2023.1092125

Nieto-Castanon, A. (2020). *Handbook of functional connectivity magnetic resonance imaging methods in CONN*. Hilbert. https://doi.org/10.56441/hilbertpress.2207.6598

Padmanabhan, A., Lynch, C. J., Schaer, M., & Menon, V. (2017). The default mode network in autism. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *2*(6), 476–486. https://doi.org/10.1016/j.bpsc.2017.04.004

Pedregosa, F., Varoquaux, G., Gramfort, A., Michel, V., Thirion, B., Grisel, O., & Duchesnay, É. (2011). Scikit-learn: Machine learning in python. *Journal of Machine Learning Research*, *12*, 2825–2830.

Sampaio, A., Soares, J. M., Coutinho, J., Sousa, N., & Gonçalves, Ó. F. (2014). The big five default brain: Functional evidence. *Brain Structure & Function*, *219*(6), 1913–1922. https://doi.org/10.1007/s00429-013-0610-y

Schilbach, L., Eickhoff, S. B., Rotarska-Jagiela, A., Fink, G. R., & Vogeley, K. (2008). Minds at rest? Social cognition as the default mode of cognizing and its putative relationship to the default system of the brain. *Consciousness and Cognition*, *17*(2), 457–467. https://doi.org/10.1016/j.concog.2008.03.013

Sparrow, S. S., & Cicchetti, D. V. (1989). The vineland adaptive behavior scales. In C. S. Newmark (Ed.), *Major psychological assessment instrument* (pp. 199–231). Allyn & Bacon.

Stirrups, R. (2018). The storm and stress in the adolescent brain. The lancet. Neurology, S1474-4422(18)30112-1. Advance online publication. https://doi.org/10.1016/S1474-4422(18)30112-1

Uddin, L. Q., Supekar, K., Lynch, C. J., Khouzam, A., Phillips, J., Feinstein, C., & Menon, V. (2013). Salience network–based classification and prediction of symptom severity in children with autism. *JAMA Psychiatry*, *70*(8), 869–879. https://doi.org/10.1001/jamapsychiatry.2013.104

Van Rossum, G., & Drake, F. L. (2009). *Python 3 reference manual*. CreateSpace.

Weng, S. J., Wiggins, J. L., Peltier, S. J., Carrasco, M., Risi, S., Lord, C., & Monk, C. S. (2010). Alterations of resting state functional connectivity in the default network in adolescents with autism spectrum disorders. *Brain Research*, *1313*, 202–214. https://doi.org/10.1016/j.brainres.2009.11.057

Whitfield-Gabrieli, S., & Nieto-Castanon, A. (2012). Conn: A functional connectivity toolbox for correlated and anticorrelated brain networks. *Brain Connectivity*, *2*(3), 125–141. https://doi.org/10.1089/brain.2012.0073

Yang, B., Wang, M., Zhou, W., Wang, X., Chen, S., Potenza, M. N., Yuan, L. X., & Dong, G. H. (2023). Disrupted network integration and segregation involving the default mode network in autism spectrum disorder. *Journal of Affective Disorders*, *323*, 309–319. https://doi.org/10.1016/j.jad.2022.11.083

Yerys, B. E., Gordon, E. M., Abrams, D. N., Satterthwaite, T. D., Weinblatt, R., Jankowski, K. F., Strang, J., Kenworthy, L., Gaillard, W. D., & Vaidya, C. J. (2015). Default mode network segregation and social deficits in autism spectrum disorder: Evidence from non-medicated children. *NeuroImage Clinical*, *9*, 223–232. https://doi.org/10.1016/j.nicl.2015.07.018