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Sex Similarities and Differences in Brain Dynamic Functional Connectivity Among Individuals With and Without Autism Spectrum Disorders

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ABSTRACT

Given the historical underrepresentation of autistic females in neuroscience research, few neuroimaging studies have directly compared females and males with autism spectrum disorders (ASD) to explore both sex-independent and -specific neural features. This study employed a sliding-window approach to construct dynamic functional connectivity and investigated sex similarities and differences in modular variability (nodal level), edge variability (edge level), and state variability (brain state level) in brain connectomes among individuals with and without ASD. Ninety-eight autistic individuals (49 female, 49 male; full-scale IQ $\geq$ 70) and 98 typically developing individuals (TD; 49 female, 49 male), matched on sex, age, and full-scale IQ, were selected from the Autism Brain Imaging Data Exchange (ABIDE). Results showed that both autistic males and females exhibited reduced modular variability in the left middle frontal gyrus and diminished edge variability in the functional connectivity between the right olfactory cortex and the right paracentral lobule, compared to their TD peers. Notably, autistic individuals manifested a sex-opposite shift in the edge variability of functional connectivity between the left amygdala and the right anterior cingulate and paracingulate gyri. Furthermore, greater autistic symptom severity was associated with reduced maintenance of a high-connectivity brain state characterized by functional competition between the frontal cortex and sensory-perceptual or subcortical regions. These findings reveal both shared and sex-differentiated alterations in connectome dynamics in ASD, with the sex-specific patterns aligning with the gender incoherence model. Understanding these dynamic features may inform more individualized and sex-sensitive educational and social support for individuals with ASD.

1 | Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental condition that manifests in early childhood. Core symptoms of ASD include difficulties in social interaction, communication, and engaging in restricted, repetitive patterns of behavior or interests (APA 2013). Historically, ASD has been more commonly diagnosed in males globally. According to the

Abbreviations: ABIDE, Autism Brain Imaging Data Exchange; ACG.R, the right anterior cingulate and paracingulate gyri; ADDM, Autism and Developmental Disabilities Monitoring; ADI-R, Autism Diagnostic Interview-Revised; ADOS, Autism Diagnostic Observation Schedule; AMYG.L, the left amygdala; ANCOVA, analysis of covariance; ASD, Autism Spectrum Disorder; CCN, cognitive control network; dFC, dynamic functional connectivity; DMN, default mode network; DTI, diffusion tensor imaging; EMB, extreme male brain; FD, framewise displacement; FDR, false discovery rate; FLS, flexible least squares; fMRI, functional magnetic resonance imaging; FPE, female protective effect; GI, gender incoherence; MFG.L, the left middle frontal gyrus; MFR, male-to-female prevalence ratio; MNI, Montreal Neurological Institute; NBS, network-based statistic; OLF.R, the right olfactory cortex; PCL.R, the right paracentral lobule; RRB, restricted and repetitive behaviors; sMRI, structural magnetic resonance imaging; SRS, Social Responsiveness Scale; TD, typically developing; TR, repetition time.

Key Points

- Autistic females and males shared reduced dynamic flexibility in the left middle frontal gyrus and in the connectivity between the right olfactory cortex and paracentral lobule, possibly reflecting autistic differences in cognitive and perceptual control.
- Autistic females and males exhibited opposite temporal dynamics in functional connectivity between the left amygdala and the right anterior cingulate and paracingulate gyri, consistent with the gender incoherence model.
- Autistic individuals with more severe symptoms are more likely to transition out of a brain state characterized by strong connectivity within- but not between-frontal and occipital cortices.

latest report from the Autism and Developmental Disabilities Monitoring (ADDM) Network, the male-to-female prevalence ratio (MFR) among 8-year-old autistic children in the United States was 3.8:1 (Maenner et al. 2023). Even though this ratio varies, such as in England (MFR = 4.3:1; Roman-Urrestarazu et al. 2021), China for preschool-age (MFR = 2.9:1; Zhao et al. 2023) and school-age (MFR = 3.2:1; Zhou et al. 2020) autistic children, New Zealand (MFR = 3.9:1; Bowden et al. 2020) across ages from 0 to 24 years old, and in North Africa and the Middle East (MFR = 3.1:1; Meimand et al. 2023), there remains a global trend of significantly more males recognized with ASD. Therefore, many theoretical frameworks and interventions are primarily based on observations from autistic males. However, mounting evidence demonstrates notable differences between autistic males and females in behavior (Calderoni 2023; Cho et al. 2023; Dworzynski et al. 2012; Hull et al. 2017; Werling and Geschwind 2013), brain structural and functional connectivity (Calderoni 2023; Hernandez et al. 2020; Lai et al. 2013; Mo et al. 2021; Walsh et al. 2021, 2023), and even genetics (Hu et al. 2021; Tylee et al. 2017). The brain connectome, particularly the dynamic brain connectome, mediates the relationship between genetics and behavior, elucidating how genes shape the brain's complex architecture and influence cognitive and behavioral traits. For instance, noradrenergic polymorphisms account for the proportion of time spent in each brain connectome state, and this noradrenergic model also predicts individuals' language and memory performance, highlighting the potential of brain connectomes as endophenotypes (Jun et al. 2025). Therefore, it is crucial for researchers to develop a deeper understanding of the sex/gender (as defined in Lai et al. 2015) similarities and differences in the dynamic brain connectomes of autistic males and females, offering a neurobiological reference for interpreting the sex/gender heterogeneity in autistic behavioral phenotypes.

1.1 | Sex/Gender Similarities in the Autistic Brain

Autism is conceptualized as a dysconnection syndrome in the context of neuroimaging. The similarities between males and females with ASD reflect factors essential to the behavioral diagnosis (Lai et al. 2015). These sex/gender-independent features differentiate autistic from neurotypical behavior and cognition, and by extension the autistic brain from the neurotypical brain. Static functional connectivity studies have revealed consistent patterns of both hypo- and hyper-connectivity in autistic males and females. For instance, reduced connectivity has been observed within the default mode network (DMN; Floris et al. 2021) and among visual association, somatosensory, and motor networks (Oldehinkel et al. 2019), whereas increased connectivity has been reported between subcortical and parietal sensorimotor regions (Di Martino et al. 2014), as well as between the DMN and other large-scale networks, and between cortical and subcortical systems (Ilioska et al. 2023). However, as the functional connectome within the human brain is a highly dynamic networked system that rapidly reorganizes connectivity over time (Allen et al. 2014; Baker et al. 2014; Hutchison et al. 2013; Pedersen et al. 2018), connectivity research has shifted from a static to a dynamic perspective.

In contrast to the traditional static approaches, dynamic methods, such as dynamic functional connectivity (dFC) and dynamic functional network connectivity, more effectively capture the transient activity patterns of the brain, revealing temporal fluctuations in brain connectomes. These methods not only provide richer insights into the neural mechanisms underlying psychiatric disorders (e.g., major depressive disorder; Sun et al. 2024) but also demonstrate greater precision in cognitive-behavioral predictions (Sen and Parhi 2021) and machine learning classification (Rashid et al. 2016). Several studies focusing on autistic males have demonstrated atypical dynamic brain connectomes. These include, but are not limited to, greater variability in long-range dynamic functional connections (Chen et al. 2017), weaker connectivity between the right anterior insula and two core hubs of DMN: the ventral medial prefrontal cortex and the posterior cingulate gyrus (Guo et al. 2019), as well as higher modular variability over time (also called *switching rate*) in the medial prefrontal cortex, posterior cingulate gyrus, and angular gyrus, along with lower modular variability in the visual regions (Xie et al. 2022). To determine whether sex/gender similarities can be identified in autistic dynamic functional connections, conducting male–female comparisons with large group sizes is a direct and robust method (Lai et al. 2015). Therefore, the first aim of our study is to investigate whether sex-independent dFC alterations exist within the autistic brain connectomes using a two-factor (diagnosis, sex) design.

1.2 | Sex/Gender Differences in the Autistic Brain

The differences between males and females with ASD reflect sex/gender-specific alterations, which may reveal sex/gender-unique susceptibility and protective mechanisms within the autistic population (Lai et al. 2015). Three important models have been proposed to explain sex/gender differences in the autistic brain. The *extreme male brain* (EMB) model (Baron-Cohen 2002) posits that the autistic brain tends to demonstrate a more masculine profile, the extreme of a population continuum of brain and cognitive differences wherein high systemizing and low empathizing tend to be inversely related

in males (Baron-Cohen et al. 2005; Greenberg et al. 2018; Valla et al. 2010). This male-biased brain pattern in ASD has been demonstrated in the intra-connectivity within DMN (Ypma et al. 2016) and in developmental changes of interhemispheric connectivity (Kozhemiako et al. 2019). In contrast to a male-biased shift observed in both males and females with ASD, the *gender incoherence* (GI) model suggests that ASD may represent a gender-discordant condition (Bejerot et al. 2012). Specifically, a male-biased shift is presented only in the brains of autistic females, whereas a female-biased shift appears in the brains of autistic males. Prior studies comparing sex/gender differences in ASD and typically developing (TD) individuals identified attenuated or even reversed differences in voxel-mirrored homotopic connectivity in the dorsolateral occipital cortex (Floris et al. 2021) and in connectomes centered on the left superior temporal sulcus (Alaerts et al. 2016), left amygdala (Lee et al. 2020), bilateral cerebellum (Smith et al. 2019), precuneus, hypothalamus (Walsh et al. 2023), and anterior cingulate cortex/medial prefrontal cortex (Lu et al. 2024) providing substantial evidence in support of the GI model. Additionally, some findings indicate that females who meet the diagnostic threshold for autism tend to present greater individual variations in brain structures (Cauvet et al. 2019; Deng and Wang 2021) and connectivity (Hernandez et al. 2020; Kozhemiako et al. 2020) compared to autistic males, congruent with the *female protective effect* (FPE) model. This model, integrating extensive epidemiological, genetic, and neuroendocrinological research, proposes that females may be protected from the effects of genetic mutations or environmental stressors linked to ASD (Werling and Geschwind 2013). These models offer complementary and somewhat synergistic explanations for sex-specific alterations in static functional connectivity of the autistic brain.

However, few studies have examined sex differences in ASD brain connectome dynamics. Only two recent studies have explored the sex/gender heterogeneity of the autistic dynamic connectomes from the perspectives of seed-based dynamic functional connectivity (Lu et al. 2024) and multilayer networks (Gao et al. 2024). However, these two studies did not analyze the similarities and differences in the brains of autistic males and females from nodes to edges, and then to whole brain states. Thus, the second aim of the present study is to address this lacuna by systematically exploring whether sex-specific alterations exist in autistic dFC from the micro (i.e., brain nodes) to the macro level (i.e., brain states), whether any of the three models (i.e., EMB, GI, and FPE) apply to such alterations, and whether such alterations are associated with core symptoms of ASD.

1.3 | Current Study

In summary, the present study utilizes data from the Autism Brain Imaging Data Exchange (ABIDE) to investigate sex-based similarities and differences in brain connectome dynamics among individuals with ASD in contrast to TD. Given that the neurophysiological differences studied herein can be both cause and consequence of gender, we use the term 'sex' when referring to the trait so labeled in the ABIDE data sets, while acknowledging the overlap between the concepts of sex and gender.

The exploration focuses on dFC, encompassing three functional levels: nodes, edges, and brain states, by examining the time-varying characteristics of functional brain connectivity. We applied a two-factor design, diagnosis (ASD, TD) × sex (male, female), to assay effects of diagnosis, sex, and sex-by-diagnosis interactions on dFC. Sex similarities are predicted solely by diagnostic differences, with no influence from sex or sex-by-diagnosis interactions. In contrast, sex differences are driven by distinct sex-by-diagnosis interactions, allowing for the comparison and contrast of the EMB, GI, and FPE models through these interactions. EMB predicts that both autistic males and females will exhibit a shift in dFC towards an extreme male-typical pattern, whereas GI predicts an opposite shift, with autistic females showing a male-typical dFC pattern and autistic males exhibiting a shift towards female-typical values. Both models share the common premise that sex effects are present in the dFC of TD individuals. FPE further predicts a non-reciprocal interaction, wherein females meeting the ASD diagnostic threshold will experience a stronger impact on dFC than their male counterparts. Such sex-specific effects on dFC might be mirrored cognitively and behaviorally by effects on gender, beyond the scope of this neurophysiological study.

2 | Methods

2.1 | Participants

One hundred ninety-six participants were selected from the ABIDE I and II databases (http://fcon_1000.projects.nitrc.org/indi/abide/) (Di Martino et al. 2017, 2014). To maximize the inclusion of high-quality imaging data from autistic females and to minimize site effects, we screened rigorously, including only those scans that met the following standards: (i) absence of apparent structural abnormalities in the T1 images, (ii) single-band fMRI data, (iii) scan length of at least 5 min, (iv) maximal head motion of less than 3 mm and 3 degrees, and mean framewise displacement (FD) of less than 0.5 mm, (v) full-brain scan and successful spatial normalization, and (vi) participants with a full-scale IQ (FSIQ) higher than 70. Subsequently, we included only sites with at least five autistic females and five TD females who met the inclusion criteria and matched participants with ASD and their TD peers for sex, age, and FSIQ within each site ($ps > 0.479$). The final sample included data from 6 sites (the detailed sites and participants screening process is shown in Figure S1), comprising 98 autistic individuals (MFR = 1:1) and 98 TD peers (MFR = 1:1). Diagnoses of ASD in the study were confirmed by clinicians using the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2000) or the Autism Diagnostic Interview-Revised (ADI-R; Lord et al. 1994). TD controls included in the study had no personal or family history of ASD and no history of other neurological or psychiatric conditions. Detailed demographic information is shown in Table 1.

Only 104 of the initial sample of 196 participants were included in the analyses of brain state variability, as clustering analysis required a consistent repetition time (TR) for all scans. Therefore, we retained three of the six sites (NYU Langone Medical Center, San Diego State University, and University of Michigan: TR = 2000 ms), comprising 52 autistic individuals (MFR = 1:1) and 52 TD counterparts (MFR = 1:1). The demographic and

TABLE 1 | Demographic information and core symptom performances of participants ($N = 196$).

	ASD		TD		Contrasts			
	Female ($n = 49$)	**Male ($n = 49$)**	**Female ($n = 49$)**	**Male ($n = 49$)**	**am vs. af**	**tm vs. tf**	**am vs. tm**	**af vs. tf**
Age (years)	13.46 (6.46)	13.27 (4.97)	12.90 (4.67)	13.55 (4.35)	0.873	0.479	0.770	0.625
Range	5.22–38.76	6.70–29.98	8.00–29.13	6.36–30.08				
Children (5–12 years)	31	28	29	26				
Adolescents (13–17 years)	11	16	16	19				
Adults (18+ years)	7	5	4	4				
FSIQ	104.00 (16.35)	103.49 (14.95)	104.87 (11.54)	105.40 (13.05)	0.872	0.832	0.503	0.762
Mean FD	0.22 (0.12)	0.19 (0.10)	0.14 (0.06)	0.16 (0.09)	0.305	0.218	0.105	< 0.001
ADOS-2 total								
af/am ($n = 36/36$)	11.17 (3.24)	12.44 (4.07)			0.145			
ADOS-2 severity								
af/am ($n = 36/36$)	6.56 (1.65)	7.14 (1.87)			0.164			
ADOS-2 social affect								
af/am ($n = 34/36$)	8.24 (2.58)	9.33 (3.46)			0.139			
ADOS-2 RRB								
af/am ($n = 34/36$)	2.68 (1.45)	3.11 (1.83)			0.277			
SRS total								
ASD/TD ($n = 66/56$)	99.44 (21.30)	88.87 (30.31)	18.81 (14.44)	16.47 (9.43)	0.109	0.484	< 0.001	< 0.001

Note: The number of participants in each age group is shown below the age range; the *p*-values of two-sample *t*-tests are presented in the 'Contrasts' columns.
Abbreviations: ADOS-2: Autism Diagnostic Observation Schedule, 2nd Edition; af: autistic female; am: autistic male; FD: framewise displacement; FSIQ: full-scale IQ; RRB: restricted and repetitive behaviors; SRS: Social Responsiveness Scale; tf: typically developing female; tm: typically developing male.

symptom characteristics of 104 participants (Table S1) are displayed in Supplementary Part 1.

2.2 | Data Preprocessing

Using the GRETNA (v2.0) package (https://www.nitrc.org/projects/gretna) (Wang et al. 2015) and MATLAB 2013b, we preprocessed all resting-state fMRI data following a standardized flow. This included: (i) removing the first 10-s volumes; (ii) correcting slice timing; (iii) realigning the images with averaged volumes; (iv) spatially normalizing images to the Montreal Neurological Institute (MNI) space with reslicing to 3 mm × 3 mm × 3 mm voxel sizes using the DARTEL strategy; (v) linear detrending; (vi) regressing out covariates (i.e., global, white matter, and cerebrospinal fluid signals, along with Friston's 24 head-motion parameters); and (vii) temporal filtering in the range of 0.01–0.10 Hz.

2.3 | Constructing Dynamic Functional Connections

The sliding-window approach, in conjunction with the anatomical automatic labeling atlas (AAL-90 regions) (Tzourio-Mazoyer et al. 2002), was used to construct dynamic functional connections using the DynamicBC (v2.2) toolbox (https://github.com/guorongwu/DynamicBC/) (Liao et al. 2014). Window lengths between 30 and 60 s have proven beneficial for capturing the dynamic volatility of functional connectivity in resting-state fMRI (Preti et al. 2017). We set the window length to 60 s and the sliding step length to one TR (refer to Xie et al. 2022). Because of

variations in total scan time across the six sites, only the first 5 min of each scan was retained for building the dynamic functional connectomes.

2.4 | Extracting Time-Varying Characteristics of Connectomes From Nodes, Edges, and Brain States

2.4.1 | Modular Variability

Modular variability, indicating the dynamic flexibility of brain nodes as they switch between modules over time (Liao et al. 2017), was selected to represent the time-varying characteristics of nodes, identified using a multilayer network model (Pedersen et al. 2018) in which functional correlations among the aforementioned 90 anatomical regions are represented for each instance of the sliding temporal window, making a three-dimensional, prismatic array of connectivities (a two-dimensional, triangular matrix of correlations) over time (the third dimension). Initially, we applied a fixed network threshold (density = 0.15) to these connectomes to mitigate the influence of weak and spurious connections (Liao et al. 2017; Xie et al. 2022). Weighted time windows were then represented as a multilayer network. The nature of the source fMRI time series renders this multilayer network diagonal, meaning that inter-layer connections are limited to coupling edges which connect homologous anatomical nodes across layers representing adjacent time windows. This temporal sequence of layers represents the network's sole aspect. The network is also node-aligned, that is, each node is represented in each (temporal) layer. Thus the resulting multilayer network constitutes a node-aligned, diagonal, single-aspect special case of multilayer networks in general (Kivelä et al. 2014), organized in diagonal lines with edges connecting adjacent networks. Modular architecture was quantified using the modularity index Q, which measures the degree of segregation between network modules on a scale from 0 to 1. Additionally, the modular variability of each node was assessed, where nodes with higher modular variability demonstrate greater flexibility in switching between different network modules. The construction of multilayer networks and the computation of indices were performed using the GenLouvain (v2.2) package (https://github.com/GenLouvain/GenLouvain) (Mucha et al. 2010). Two important parameters, which determine the strength of topological connectivity (γ) and temporal coupling (ω), were each set to 1. To mitigate the instability of the Louvain algorithm (Blondel et al. 2008), the estimation of Q and modular variability was repeated 100 times for each participant, and the mean values were carried forward to further analysis.

2.4.2 | Edge Variability

Edge variability reflects the temporal volatility of functional connectivity between brain nodes, represented by the standard deviation of dFC coefficients (Chen et al. 2017). This calculation was performed using the DynamicBC (v2.2) toolbox in conjunction with the AAL atlas (90 regions), yielding a 90 × 90-node triangular edge-variability matrix for each participant.

2.4.3 | State Variability

Brain states are transient patterns of brain connectomes (Hutchison et al. 2013; Shakil et al. 2016), reflecting the functional connectivity patterns between node pairs within time windows in the sliding-window approach. Clustering across participants associates brain states with similar connectivity patterns and distinguishes those with different patterns, thus identifying average brain states shared across individuals (Allen et al. 2014; Hutchison et al. 2013; Shakil et al. 2016). The k-means clustering algorithm (Aggarwal et al. 2001), implemented using the DynamicBC (v2.2) toolbox, clustered dFC matrices of all participants. We set the maximum value of k as eight and determined the optimal k-value by averaging values calculated using silhouette (Rousseeuw 1987), Calinski-Harabasz (Caliński and Harabasz 1974), and Davies-Bouldin (Davies and Bouldin 1979) coefficients. Distances between functional connectivity matrices were computed using the city-block measure. The brain state for each participant was described by averaging that participant's dFC matrices within the same cluster (Liao et al. 2014). State variability was quantified by four temporal indices (Kim et al. 2017; Luo et al. 2021; Shi and Zeng 2022): fractional time (proportion of time in each state), number of transitions (switches between states), mean dwell time (average time spent in each state before switching), and transition probability (probability of shifting between states). The overall workflow for connectome dynamics analysis is illustrated in Figure 1.

2.5 | Site Effects Correction

To minimize noise stemming from variations in device selection, parameter settings, and environmental conditions during image acquisition across different sites, we employed the ComBatHarmonization (v1.0.1) package (https://github.com/Jfortin1/ComBatHarmonization/tree/master/Matlab) (Johnson et al. 2007; Fortin et al. 2018; Yu et al. 2018) for site effects harmonization. ComBat covaried diagnosis, sex, age, FSIQ, and mean FD, to preserve meaningful biological information from the scans. This process corrected modular and edge variability maps, and functional connectivity matrices of each window pre-clustering analysis. These harmonized indices were transformed to z-scores to enhance comparability among participants, except for the harmonization conducted in the state-level analysis.

2.6 | Statistical Analyses

Modularity (Q-value) and modular variability at each node were assessed by two-factor covariance models (diagnosis: ASD/TD × sex: female/male) with age, FSIQ, and mean FD as covariates in R 4.2.2. Multiple-testing adjustments were made using the false discovery rate (FDR) method (Benjamini and Hochberg 1995). The emmeans (v1.8.4-1) package (https://CRAN.R-project.org/package=emmeans) was used to assess the simple effects of significant interactions surviving the FDR correction ($p_{FDR} < 0.05$). Statistical power was computed *post hoc* (post-correction) using G*Power, version 3.1.9.7.

The network-based statistic (NBS) approach analyzed edge variability between node pairs, via the NBS Connectome

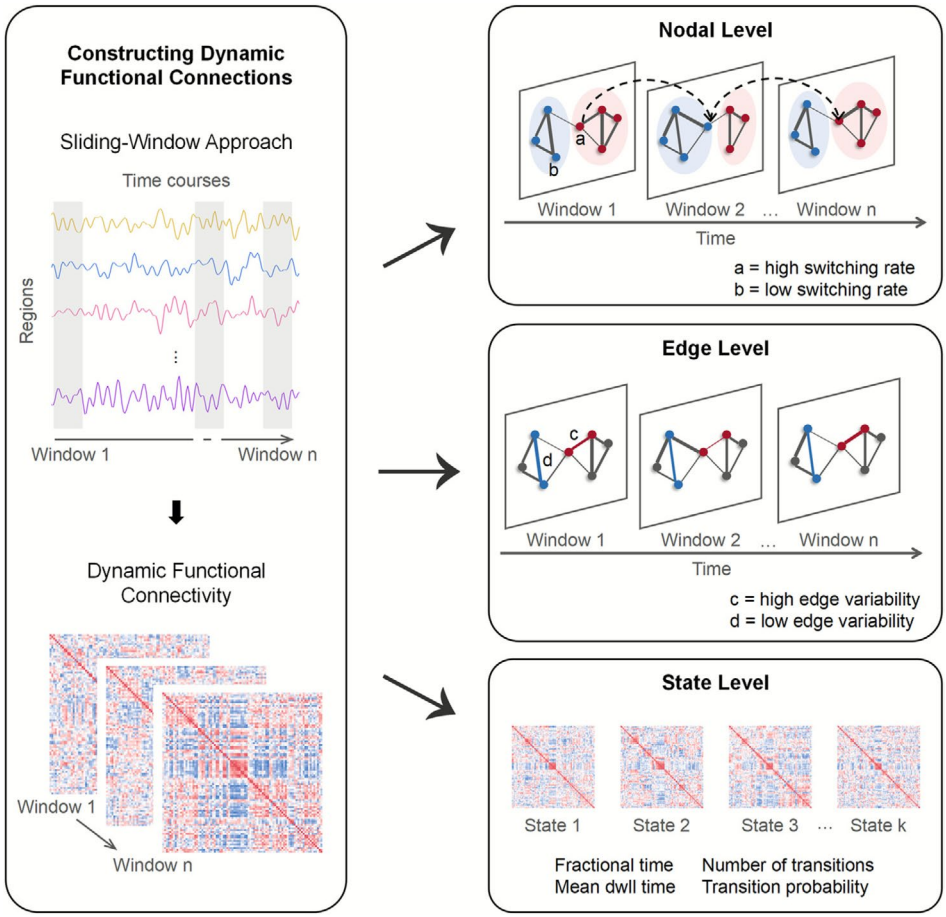

FIGURE 1 | The overall workflow for dynamic functional connectivity construction and analyses.

(v1.2) package (http://www.nitrc.org/projects/nbs) (Zalesky et al. 2010). An analysis of covariance (ANCOVA) model was employed, with contrast matrices including diagnosis, sex, and sex-by-diagnosis interactions, while also accounting for age, FSIQ, and mean FD. We applied the FDR method with an alpha threshold of 0.05 and conducted 10,000 permutations to estimate all edge variability maps. Simple effect tests and power analysis were performed on edge variability using methods consistent with those described above for nodal level analysis.

Since the four state variability indices did not follow Gaussian distributions, we employed the npsm (v2.0.0) package (https://CRAN.R-project.org/package=npsm) to conduct rank-based ANCOVA (Kloke and McKean 2014, 2024) to assess the effects of diagnosis, sex, and sex-by-diagnosis interactions on fractional time, number of transitions, mean dwell time, and transition probability, and conducted corresponding *post hoc* comparisons. FDR correction and power analysis were also performed on state variability, following the same methods outlined in the nodal level analysis.

Given that numerous studies have reported age-related alterations in brain functional connectivity patterns in ASD (Harlalka et al. 2019; Kozhemiako et al. 2019, 2020; Nomi and Uddin 2015), the present study not only included age as a covariate but also treated it as an independent variable to examine whether the core findings were moderated by age (see Supplementary Part 3 for analytical details).

2.6.1 | Correlational Analyses

We selected the ADOS-2 and the Social Responsiveness Scale (SRS) total raw scores as the core symptom phenotypes, based on their reported validity in the ABIDE datasets. The ADOS-2, available only for ASD, includes total scores, calibrated severity, social affect, and restrictive and repetitive behavior total scores. SRS scores are available for both ASD and TD individuals. Higher scores on these measures indicate more pronounced or severe symptoms in ASD. Pearson's partial correlation analysis was performed to examine the relationships between modular and edge variability indices (after FDR correction) and the core symptom phenotypes. Additionally, Spearman's partial correlation analysis was conducted to explore the associations between state variability indices (after FDR correction) and the core symptom phenotypes. All correlation analyses were adjusted for age, FSIQ, and mean FD. The FDR approach was applied to correct for multiple comparisons.

2.7 | Validation Analyses

Validation analyses were conducted on the main findings using a larger, expanded sample ($N = 791$, Table S2) and varying window lengths (45, 75, 90 s). Furthermore, we sought to validate the core results using an alternative dFC construction method (flexible least squares, FLS; Kalaba and Tesfatsion 1989) and

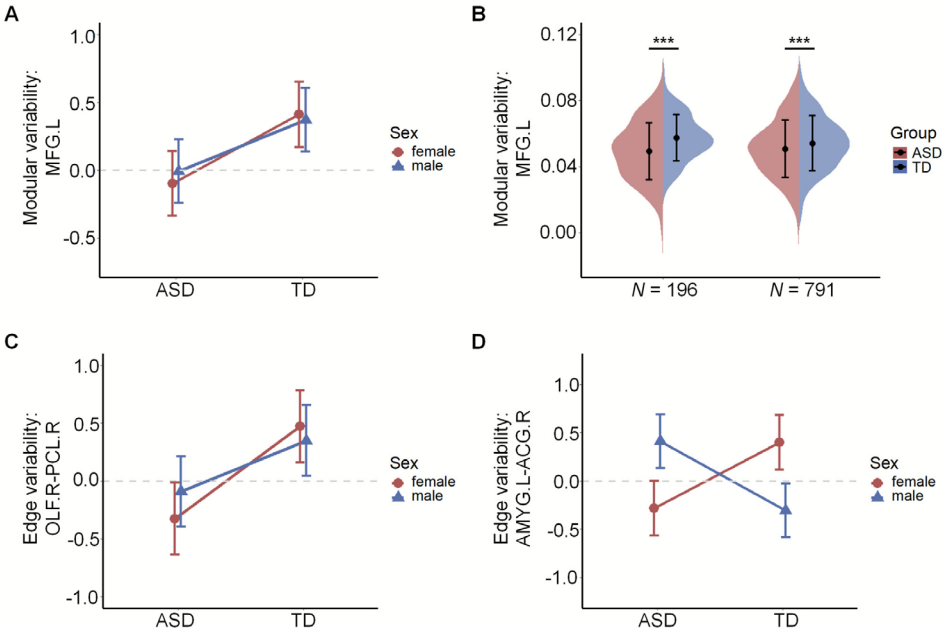

FIGURE 2 | The results of modular variability and edge variability after FDR correction. (A) The sex-by-diagnosis interaction effects on modular variability in the left middle frontal gyrus (MFG.L). Both autistic females and males showed significantly reduced modular variability in the MFG.L. (B) The diagnosis effect on modular variability in MFG.L from the current sample ($N = 196$) and an expanded sample ($N = 791$), ***$p < 0.001$. (C) The sex-by-diagnosis interaction effects on edge variability in functional connectivity between the right olfactory cortex (OLF.R) and the right paracentral lobule (PCL.R). Autistic females and males exhibited similarly diminished variability in the OLF.R-PCL.R connection. (D) The sex-by-diagnosis interaction effects on edge variability in functional connectivity between the left amygdala (AMYG.L) and the right anterior cingulate and paracingulate gyri (ACG.R). Autistic individuals presented a sex-opposite shift in the variability of AMYG.L-ACG.R connectivity, consistent with the gender incoherence model.

finer-grained, functionally informed atlases (Brainnetome-246 and Brainnetome-274, the latter including cerebellum regions; Fan et al. 2016). The detailed analytical strategies are shown in Supplementary Part 4.

3 | Results

3.1 | Nodal Level: Modular Variability

After correction for multiple comparisons, only the left middle frontal gyrus (MFG.L, $F(1,189) = 12.64$, partial $\eta^2 = 0.063$, $p_{FDR} = 0.043$, power = 0.95, Figure 2A) showed significantly reduced modular variability in individuals with ASD compared to TD peers ($p_{post\ hoc} < 0.001$). This reduction was independent of sex and sex-by-diagnosis interactions, and was not influenced by age (Table S7). The effect was successfully replicated in an expanded sample of 791 participants—including a greater number of males and TD females (Figure 2B, Table S10)—and was further observed when the dFC was constructed using the FLS method, albeit with a reduced effect size (Table S14). Furthermore, within the TD group, the modular variability of MFG.L showed a significant negative correlation with SRS total scores ($r = -0.302$, $p = 0.030$, $R^2 = 0.091$). No significant effects of diagnosis, sex, or their interaction were observed for modularity. None of the other nodes showing initial effects on modular variability survived FDR correction (Table S3, Figure S2). Moreover, MFG.L modular variability was not significantly associated with any other core symptom phenotypes ($ps > 0.075$).

3.2 | Edge Level: Edge Variability

The NBS analysis revealed a significant diagnostic effect ($F(1,189) = 14.61$, partial $\eta^2 = 0.072$, $p_{FDR} < 0.001$, power = 0.97, Figure 2C) on edge variability between the right olfactory cortex (OLF.R) and the right paracentral lobule (PCL.R), with lower variability in autistic individuals than in TD individuals ($p_{post\ hoc} < 0.001$). No significant sex or sex-by-diagnosis interaction effects were found for this edge (the brain map of OLF.R–PCL.R is displayed in Figure S3A). In contrast, a significant sex-by-diagnosis interaction emerged for the variability of functional connectivity between the left amygdala (AMYG.L) and the right anterior cingulate and paracingulate gyri (ACG.R), $F(1,189) = 24.08$, partial $\eta^2 = 0.113$, $p_{FDR} < 0.001$, power = 0.99 (Figure 2D). Specifically, TD males showed lower variability than TD females ($p_{post\ hoc} = 0.0006$); autistic females exhibited lower variability than both autistic males ($p_{post\ hoc} = 0.0007$) and TD females ($p_{post\ hoc} = 0.001$); and autistic males exhibited greater variability than TD males ($p_{post\ hoc} = 0.0005$). This interaction was independent of age and developmental stage (Table S8). Validation analyses confirmed the robustness of this effect across different window lengths (Table S12), and a similar but attenuated pattern was observed when the dFC was constructed using the FLS method (Table S14). No main effects of diagnosis or sex were observed for this edge (the brain map of AMYG.L–ACG.R is shown in Figure S3B). Other edges did not show significant effects after FDR correction, and no significant correlations were found between the variabilities of the two edges (i.e., OLF.R–PCL.R and AMYG.L–ACG.R) and core symptom phenotypes, $ps > 0.158$.

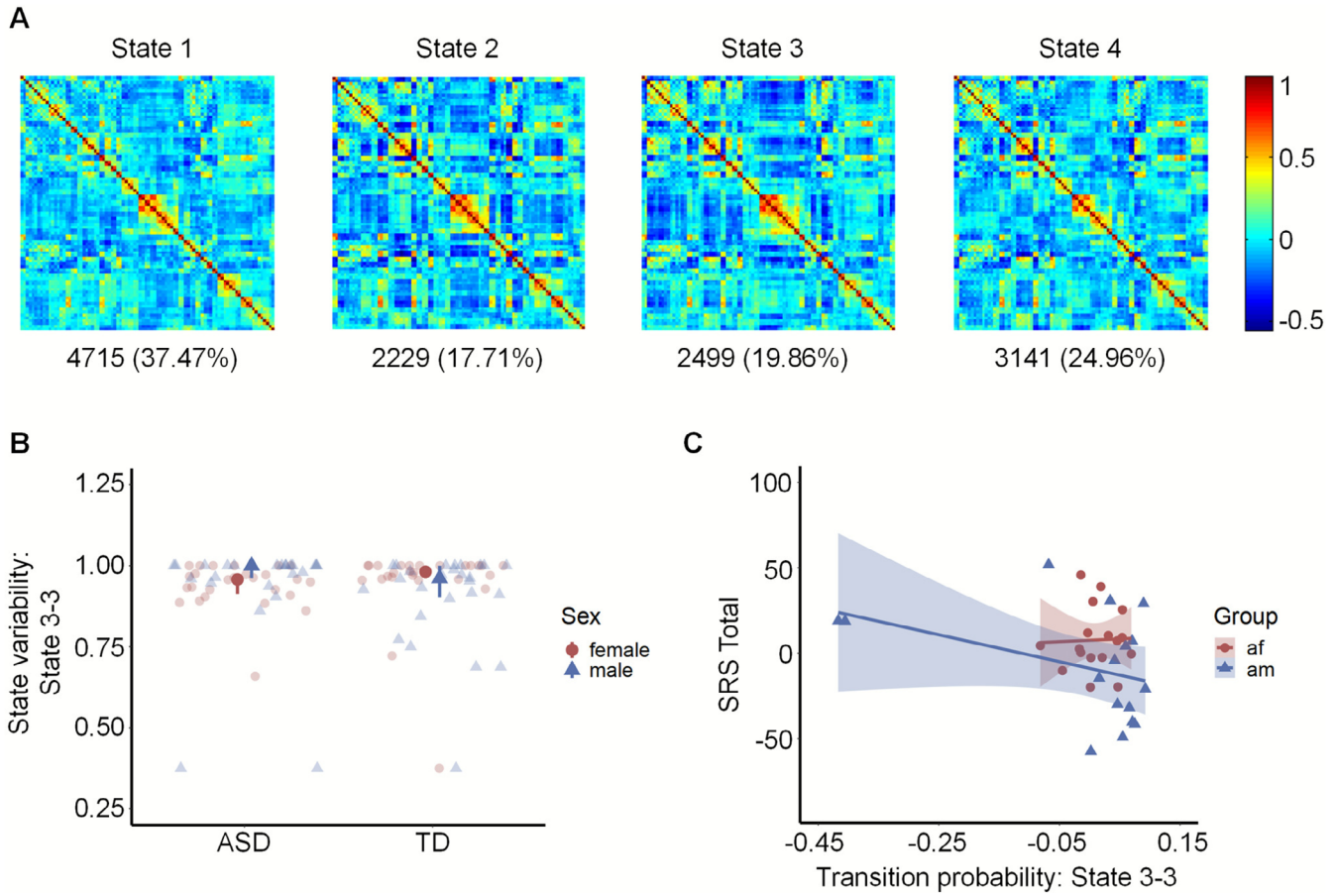

FIGURE 3 | The results of state variability after FDR correction. (A) State centroids of all participants' ($N = 104$) functional connectivity matrices. (B) Scatter plot of the probability for the transition from State 3 to itself. The dark geometric figures represent the medians, and the error bars denote the quartile deviations. (C) Correlation between residuals of the transition probability for State 3–3 and residuals of the total scores for the Social Responsiveness Scale (SRS) after controlling for age, full-scale IQ and mean framewise displacement (mean FD). In autistic males, the State 3–3 transition probability was moderately negatively correlated with SRS total scores (did not survive FDR correction). af = autistic females; am = autistic males.

3.3 | State Level: State Variability

Clustering the functional connectivity matrices of all participants identified four brain states (Figure 3A). State 1 was the most frequent (37.47%), characterized by globally weak connectivity and moderate internal functional connectivity ($0.3 < r < 0.7$) in the occipital lobe and subcortical nuclei. State 2, the least frequent (17.71%), exhibited strong occipital and subcortical connectivity ($0.4 < r < 1.0$), with competitive relationships between the frontal and occipital lobes, and between the parietal and occipital lobes ($-0.5 < r < 0$). State 3 (19.86%) featured enhanced connectivity within the frontal and occipital lobes ($0.4 < r < 1.0$), along with negative correlations between the frontal lobe and subcortical nuclei, occipital lobe, and superior temporal gyrus ($-0.4 < r < 0$). State 4 (24.96%) presented a pattern similar to State 1 but with slightly stronger connectivity, including moderate functional connectivity within the frontal lobe, occipital lobe, and subcortical nuclei ($0.4 < r < 0.7$), and weak connectivity elsewhere. Detailed descriptions of the brain states are provided in Tables S4 and S5.

Among the four state variability indices, only one state transition probability survived multiple comparison correction (uncorrected significant results are presented in the supplement). Specifically, a significant sex-by-diagnosis interaction was observed for the maintenance probability of State 3 (i.e., the transition from State 3 to itself; $F = 11.74$, $p_{FDR} = 0.023$, power = 0.93, Figure 3B), with autistic females exhibiting significantly lower transition probability than both autistic males ($p_{post\ hoc} = 0.008$) and TD females ($p_{post\ hoc} = 0.014$). No significant differences were found in other contrasts (TD males vs. TD females: $p_{post\ hoc} = 0.067$; ASD males vs. TD males: $p_{post\ hoc} = 0.082$). This interaction remained independent of developmental stage (Table S9).

Notably, in autistic males, the State 3–3 transition probability was moderately negatively correlated with SRS total scores ($r = -0.560$, $p = 0.047$, $R^2 = 0.314$; Figure 3C), although this correlation did not survive FDR correction ($p_{FDR} = 0.233$). According to the FPE model—which posits that autistic females tend to exhibit more severe symptoms to meet diagnostic thresholds—this finding may suggest that the reduced probability of State 3–3 in autistic females is influenced by symptom severity. Indeed, autistic females in our sample ($N = 17$, $M_{raw} = 101.18$) showed higher SRS total scores than autistic males ($N = 16$, $M_{raw} = 85.81$), though the difference was not statistically significant ($t = 1.61$,

$p = 0.120$). To examine whether the observed interaction was driven by symptom severity, we reanalyzed the data in a subsample with both neuroimaging and behavioral data ($N_{ASD} = 33$, $N_{TD} = 19$, Table S1), controlling for SRS total scores. After controlling for symptoms, the sex-by-diagnosis interaction on State 3 maintenance probability was no longer significant ($F = 3.83$, $p = 0.057$, power = 0.53; detailed information see Table S6). No significant correlation was observed between the transition probability of State 3–3 and core symptom phenotypes after FDR correction.

3.4 | Validation Results

Under the same brain atlas (i.e., AAL-90), the validation results demonstrated high consistency with the main findings regarding modular and edge variability (Tables S10–S14, Figure S4). In cross-atlas validation analyses, only the findings for modular variability were successfully replicated, albeit with reduced effect strength (Tables S15–S17). By contrast, the verification results for state variability were less satisfactory, as a result of frequent changes in state centroids during identification; these findings based on clustering analysis with sliding window correlations demand cautious interpretation. Detailed results of the validation analyses are provided in Supplementary Part 4.

4 | Discussion

Although ASD diagnoses have increased (Russell et al. 2022), females remain underdiagnosed (Burrows et al. 2022; Happé and Frith 2020), partly due to diagnostic frameworks largely derived from male-based research. To address this gap, the present study leveraged ABIDE datasets and employed a two-factor (diagnosis × sex) design to examine sex-shared and sex-specific alterations in brain dynamic connectomes across nodal, edge, and state levels. At the nodal level, both autistic females and males exhibited lower modular variability in the MFG.L than their TD peers, regardless of sex. At the edge level, both autistic groups exhibited similarly reduced variability in the OLF.R–PCL.R connection, but demonstrated opposite sex effects in AMYG.L–ACG.R variability: autistic males showed greater variability than females, whereas the reverse pattern was observed in TD individuals. At the state level, autistic females were more likely to leave State 3 than both autistic males and TD females, while autistic males resembled TD males in their greater tendency to remain in State 3. These findings highlight both sex-independent and sex-specific atypical connectome dynamics in ASD, with the latter aligning with the GI model.

4.1 | Sex Similarities in ASD: Inflexible Time-Varying Characteristics of Brain Connectomes

The first aim of this study was to determine whether sex-independent dFC alterations exist within autistic brain connectomes. Across sexes, autistic individuals consistently showed reduced modular variability in MFG.L and diminished edge variability in the OLF.R–PCL.R connection, reflecting inflexible time-varying patterns in MFG.L module switching and OLF.R–PCL.R connectivity fluctuations. These sex-independent features underscore the uniform impact of autism on both male and female brains.

Modular variability in the lateral frontal and parietal regions of healthy adults has been linked to individual cognitive functions such as working memory, planning, and reasoning (Pedersen et al. 2018). In the current study, the autistic brain exhibited reduced modular variability in MFG.L, a key node of the cognitive control network (CCN) (Niendam et al. 2012; Uddin et al. 2019) involved in cognitive flexibility (Ravizza and Carter 2008; Uddin 2021; Vara et al. 2014; Yerys et al. 2015), response inhibition (Banich et al. 2000; Bunge et al. 2002; Niendam et al. 2012), and working memory (Niendam et al. 2012; Pereira et al. 2023). Even within the typically developing group, individuals with poorer social responsiveness (i.e., higher SRS total score) tend to show lower modular variability in this node. This reduced variability suggests inflexible module switching in MFG.L, potentially contributing to cognitive control deficits in ASD. Additionally, edge variability in the OLF.R–PCL.R connection was diminished in autistic individuals. The olfactory (pyriform) cortex, in concert with regions such as the amygdala, hippocampus, and thalamus, constitutes the olfactory system responsible for odor processing (Royet and Plailly 2004; Wilson et al. 2014), while the PCL is involved in somatosensory processing (Lau et al. 2019). In typically developing brains, both regions exhibit high temporal variability in their functional architecture, reflecting their adaptability and plasticity in sensory and perceptual learning (Zhang et al. 2016). Therefore, the relatively stable synchronization of functional activities between OLF.R and PCL.R in both autistic males and females may be linked to the deficits in perceptual modulation and multisensory integration associated with ASD (Crane et al. 2009; Ramappa et al. 2023; Schaaf et al. 2023).

These sex similarities in autistic brain connectomes suggest inflexible neural dynamics in cognitive and perceptual control. This rigidity partially aligns with the high and inflexible precision of prediction errors in autism (Belmonte 2020; Courchesne and Allen 1997; Sinha et al. 2014; Van de Cruys et al. 2014), and is consistent with the association between dimensional autistic social traits (SRS scores), delayed re-orienting of attention, and graph-theoretic efficiency of the frontoparietal network (Paul et al. 2021). Although this study did not directly assess brain activation intensity or predictive coding through task-based measures, the findings suggest that autistic females and males may share neural dynamic underpinnings in perceptual and cognitive processing.

4.2 | Sex Difference in ASD: An Opposite Shift in Edge Variability of AMYG.L–ACG.R

Our second objective was to investigate sex-specific dFC alterations in ASD and evaluate their alignment with the EMB, GI, or FPE models. At the edge level, functional synchrony between AMYG.L and ACG.R exhibited more pronounced temporal fluctuations in autistic males than in autistic females, whereas this pattern was reversed in TD individuals. This opposing sex difference supports the GI model (Bejerot et al. 2012). Interestingly, consistent with the GI model, Lee et al. (2020) reported that sex differences in the left amygdala

connectome—with regions including the left lingual gyrus, ventral and dorsomedial prefrontal cortex, and right posterior cingulate cortex—were attenuated and reversed in autistic children relative to the pattern observed in TD peers. However, they did not observe a significant sex-by-diagnosis interaction in AMYG.L–ACG.R connectivity. One possible explanation is that this atypical sex difference was evident primarily in the time-varying rather than static functional connectivity. Alternatively, sample size imbalance ($N_{male}/N_{female} = 80/36$) and developmental quotient disparities in Lee et al.'s study may have obscured the actual sex difference in the strength of AMYG.L–ACG.R.

The amygdala, a key regulator of social functioning, integrates emotion—particularly negative affect—to coordinate social brain networks (Amaral 2002; Bickart et al. 2014; Fadok et al. 2018; Hennessey et al. 2018; Rutishauser et al. 2013). Its interaction with the anterior cingulate cortex supports affective control by managing ambiguous emotional stimuli (Simmons et al. 2008) and resolving emotional conflicts (Etkin et al. 2006). Atypical edge variability between AMYG.L and ACG.R may thus contribute to social communication and interaction difficulties in ASD. Although no significant correlations with core autistic symptoms were detected—possibly because of the limited number of valid symptom scores from autistic individuals—further studies should clarify whether sex-specific alterations in AMYG.L–ACG.R variability relate to autism symptoms by leveraging well-matched samples with comprehensive behavioral and neuroimaging measures.

4.3 | Atypical Transition Probability and Autistic Symptom Severity

At the state level, we identified four distinct brain states across all participants. Autistic females showed a greater tendency to transition out of State 3 than both autistic males and TD females, while autistic males, like TD males, tended to stay in State 3. Although this result initially appeared to support the FPE model, a significant negative correlation between SRS total scores and State 3 transitions in autistic males suggested that more severe symptoms increased the likelihood of leaving State 3—mirroring the pattern in autistic females, who generally had higher SRS total scores. This resemblance implies that the atypical state transitions observed in autistic females who meet diagnostic thresholds may stem from their relatively pronounced autistic symptoms. Supporting this interpretation, in a subsample of 53 individuals with high-quality behavioral and imaging data, the significant sex-by-diagnosis interaction in State 3–3 transition probability became non-significant after controlling for SRS total scores.

Compared to other states, State 3 was less frequent and exhibited stronger functional connectivity—marked by heightened cooperative activities within frontal and occipital lobes, and competitive activities between the frontal lobe and subcortical nuclei or the occipital lobe. These high synchronizations within frontal and occipital lobes may reflect enhanced cognitive and visual processing, as frontal regions contribute to the DMN, CCN, and fronto-parietal network, while occipital regions are central to the visual network (Allen et al. 2014; Menon and D'Esposito 2022; Uddin et al. 2019). Given that the brain transits between functional states that either maximize segregation (forming cohesive functional modules) or promote integration across distinct neural regions (Betzel et al. 2016; Shine et al. 2016; Shine and Poldrack 2018; Zalesky et al. 2014), the difficulty in maintaining State 3 (a highly segregated state) may reflect inefficient cognitive and visual information segregation in autistic individuals with more severe symptoms. However, this finding is constrained by low statistical power due to small samples and also by the interpretability of the dFC constructed between individual brain nodes rather than whole independent components. Future work should validate these findings by constructing dynamic functional network connectivity in larger samples. Moreover, as dynamic brain states emerge from complex interactions between internal (e.g., structural connectivity, neuromodulation) and external (e.g., sensory input, social environment) factors (Sporns 2022), integrating multimodal data is essential to extend these findings.

Taken together, this study provides the first systematic evidence of both sex-independent and sex-specific alterations in dynamic brain functional connectivity among individuals with and without ASD. It reveals inflexible time-varying features in both autistic males and females, a sex-opposite shift in AMYG.L–ACG.R connectivity, and a possible link between state maintenance and autistic symptom severity. With growing awareness of the underdiagnosis and delayed diagnosis of autistic females (Calderoni 2023; Giarelli et al. 2010; Loomes et al. 2017; Shattuck et al. 2009), the present findings suggest that diagnostic strategies developed primarily based on male phenotypes may not adequately capture the manifestation of autism in females. Moreover, our results highlight that autistic females exhibit both overlapping and distinct alterations in dynamic brain connectivity compared to males. The shared alterations suggest that general interventions—such as cognitive flexibility training, targeted perceptual learning, and multimodal integration exercises—may help individuals with ASD develop the temporal fluidity in moment-to-moment reallocation of neural information processing resources. And complementarily, sex-specific differences suggest tailored educational and social support strategies. For instance, higher AMYG.L–ACG.R variability in autistic males may reflect emotional instability or heightened affective responsiveness, whereas lower variability in autistic females might associate with affective inhibition and overcontrolled, camouflaged social behavior. Accordingly, interventions could aim to enhance emotional regulation and social consistency in autistic males and to reduce emotional suppression and social masking pressures in autistic females.

This study has several limitations. First, although this study leveraged a large-scale database, group sizes were constrained by the limited availability of high-quality imaging data in autistic females, the consistency of scanning parameters (e.g., TR) across sites, and the completeness of phenotypic data related to ASD features. Insufficient sample sizes compromise both statistical power and the generalizability of findings. Future research should adopt harmonized scanning protocols, increase the representation of autistic females, and ensure comprehensive phenotypic data collection to yield more robust insights into atypical

brain dynamics in ASD and their associations with core autism symptoms. Second, from a methodological perspective, to maximize sample inclusion, we constructed dFC using only the first 5 min of resting-state data. While applying sliding-window analysis to 5-min scans is acceptable, longer scan durations (e.g., 10 min) are generally preferred to improve the stability and reproducibility of dFC estimates. Future studies should replicate our findings using longer resting-state scans. Third, as this study was restricted to participants with FSIQ above 70, and from a loose and individualistic (United States) culture, the results may not generalize to the broader autism spectrum, or to the broader world. Autistic individuals with intellectual disability may exhibit distinct patterns of sex-related patterns in dFC compared to those without cognitive impairment. Future research should investigate how IQ and culture both modulate dFC and interact with sex to shape autistic brain network dynamics. Fourth, the core findings showed limited consistency across parcellation atlases. The AAL-90 atlas is anatomically defined, whereas the Brainnetome atlas is based on structural and functional connectivity. Although some regions share similar labels (e.g., MFG.L) across the two atlases, their spatial extents and boundaries differ, which may lead to variations in the extracted signal patterns. Moreover, dFC measures are sensitive to the temporal coherence within regions of interest; high-resolution atlases tend to capture finer but more localized dynamic fluctuations. These differences highlight the influence of parcellation choice in functional connectivity research. Future studies should evaluate the robustness of their findings across multiple atlases and consider individualized or multi-scale parcellations to better characterize brain network dynamics.

5 | Conclusions

In summary, both autistic females and males demonstrate shared inflexible time-varying characteristics in MFG.L and OLF.R–PCL.R connectivity. Consistent with the GI model, they also differ in temporal features related to the functional connectivity between AMYG.L and ACG.R. Additionally, autistic individuals with more severe symptoms are more likely to transition out of a brain state characterized by strong connectivity within- but not between- frontal and occipital cortices. Clinicians and intervention practitioners should recognize both the common and sex-specific neural and cognitive features of ASD and implement individualized educational and support strategies for this population.

Author Contributions

X.M. and J.Z. designed the analysis pipeline, with further input from M.K.B. Both X.M. and Y.Z. conceived and conducted the analyses. X.M. and J.Z. made substantial contributions to result visualization. J.Z. contributed to funding acquisition and supervision. X.M. wrote the manuscript, which was reviewed, re-interpreted, and edited by M.K.B. All authors reviewed and approved the final manuscript.

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Ethics Statement

All the Autism Brain Imaging Data Exchange (ABIDE) contributions were based on studies approved by the local Institutional Review Boards. Written informed consent was obtained from a parent/guardian, and assent was obtained from all participants. Detailed information regarding scanning information, diagnostic protocols, and ethical statements for the ABIDE database can be accessed at http://fcon_1000.projects.nitrc.org/indi/abide/.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data sets supporting the findings of this study are available in ABIDE (http://fcon_1000.projects.nitrc.org/indi/abide/).

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Figure S1:** Flowchart of site and participant inclusion criteria. af: autistic female; am: autistic male; FSIQ: full-scale IQ; KKI: Kennedy Krieger Institute, TR = 2500 ms; NYU: NYU Langone Medical Center, TR = 2000 ms; OHSU: Oregon Health and Science University, TR = 2500 ms; SDSU: San Diego State University, TR = 2000 ms; tf: TD female; tm: TD male; TR = repetition time; UCLA: University of California, Los Angeles, TR = 3000 ms; UM: University of Michigan, TR = 2000 ms. **Table S1:** Demographic information, comparisons and core symptom performances of participants ($N = 104$). **Table S2:** Demographic information, comparisons and core symptom performances of participants ($N = 791$). **Figure S2:** Brain maps illustrating the effects of diagnosis (A), sex (B), and sex-by-diagnosis interaction (C) on modular variability ($N = 196$). af: autistic female; am: autistic male; L: the left hemisphere; R: the right hemisphere; tf: typically developing female; tm: typically developing male. **Table S3:** The significant diagnosis, sex, and sex-by-diagnosis interaction effects on modular variability ($N = 196$). **Figure S3:** Brain maps of edge-connected regions with significant edge variability. (A) The functional connection between the right olfactory cortex (OLF.R) and the right paracentral lobule (PCL.R). (B) The functional connection between the left amygdala (AMYG.L) and the right anterior cingulate and paracingulate gyri (ACG.R). L: the left hemisphere; R: the right hemisphere. **Table S4:** Descriptive information of brain states in different groups. **Table S5:** The median and interquartile range for the number of transitions and the transition probability. **Table S6:** Rank-based ANCOVA results for sex-by-diagnosis interaction effect on transition probability of State 3–3 with and without controlling for SRS total scores. **Table S7:** Age effects on modular variability after FDR correction. **Table S8:** Age effects on edge variability after FDR correction. **Table S9:** Age effects on state variability after FDR correction. **Figure S4:** Brain maps illustrating the effects of diagnosis (A), sex (B), and sex-by-diagnosis interaction (C) on modular variability ($N = 791$). af: autistic female; am: autistic male; L: the left hemisphere; R: the right hemisphere; tf: typically developing female; tm: typically developing male. **Table S10:** The significant diagnosis, sex, and sex-by-diagnosis interaction effects on modular variability ($N = 791$). **Table S11:** ANCOVA results for diagnosis effects on modular variability in MFG.L across various window lengths and sample sizes. **Table S12:** NBS results for diagnosis effects on edge variability in OLF.R–PCL.R and sex-by-diagnosis interaction effects on edge variability in AMYG.L–ACG.R across various window lengths. **Table S13:** Validation of edge variability in OLF.R–PCL.R revealing the sex similarities and edge variability in AMYG.L–ACG.R supporting the GI model across various window lengths. **Table S14:** Validation of core findings using dynamic functional connectivity constructed by the flexible least squares method at the nodal and edge levels. **Table S15:** Validation of core findings using different atlas at the nodal and edge levels. **Table S16:** The significant diagnosis, sex, and sex-by-diagnosis interaction effects on edge variability after FDR correction in the Brainnetome-246 atlas ($N = 196$). **Table S17:** The significant diagnosis, sex, and sex-by-diagnosis interaction effects on edge variability after FDR correction in the Brainnetome-274 atlas ($N = 190$).