Research Article

Structural Architecture of the Social Brain in Adults with Autism: A Combined Cortical Thickness and Similarity Network Analysis

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Abstract

Aim: Autism Spectrum Disorder (ASD) involves complex alterations in brain structure that persist across the lifespan. While structural brain alterations are known in children, the persistence of these neuroanatomical differences into adulthood remains less understood. This study examines the neuroanatomical basis of ASD in adulthood, specifically investigating how cortical thickness (CT) and structural similarity networks (SSN) are organized within the social brain network. **Materials and Methods:** T1-weighted MRI data were obtained for 24 adults with ASD and 24 neurotypical (NT) controls (ages 18–30) from the OpenNeuro dataset (ds002522). Image preprocessing was performed using the recon-all pipeline in FreeSurfer. We investigated CT and SSN at both: (1) the whole-brain, and (2) a hypothesis-driven level targeting 14 specific social brain network regions. CT was assessed using vertex-wise surface-based morphometry, while SSN were constructed using the Morphometric INverse Divergence (MIND) method. MIND quantifies morphological similarities based on the divergence of regional distributions for thickness, volume, surface area, mean curvature, and sulcal depth. **Results:** The SSN analysis revealed significantly increased nodal connectivity strength in the ASD group within the right posterior insula (pFDR=0.04) and the orbital part of the right inferior frontal gyrus (pFDR = 0.04). ROI-based CT comparisons and whole-brain SSN analyses showed no significant group differences. **Conclusion:** Our findings reveal a neuroanatomical signature in adults with ASD, characterized by localized structural hyper-connectivity within the inferior frontal gyrus and the insula. These results highlight that adult ASD is defined by persistent structural anomalies, manifesting as atypically high structural similarity within key social brain nodes rather than widespread, global network disruptions.

Keywords: Autism spectrum disorder, structural similarity networks, morphometric inverse divergence, cortical thickness, social brain.

Introduction

Autism Spectrum Disorder (ASD) is a heterogeneous neurodevelopmental condition characterized by early onset and persistent deficits in social communication and social interaction, alongside restricted, repetitive patterns of behavior, interests, or activities[1]. The etiology of ASD is remarkably complex, arising from a multifaceted interplay of genetic, epigenetic, and environmental factors that may act independently or in concert. These diverse influences are widely understood to drive atypical brain development and neural reorganization, a process that begins in early life and continues to shape the neuroanatomical landscape into adulthood[2].

Structural neuroimaging studies in ASD have consistently reported atypical brain development, often characterized by early brain overgrowth[3, 4]. This accelerated brain development, particularly evident in early childhood compared to typically developing children, is thought to contribute to the condition's neurobiological underpinnings. Furthermore, these studies frequently highlight altered local connectivity patterns, predominantly between frontal and other cortical regions[5,6]. However, the precise cellular mechanisms underlying these observed neural patterns remain largely unclear. Consequently, there is ongoing debate regarding how these connectivity differences ultimately impact macroscopic brain measures such as volume and cortical thickness. This lack of definitive evidence contributes to the heterogeneity of findings in the literature, with results often varying significantly depending on measurement conditions, analytical methods, and developmental stage of the cohort studied[7,8]. While such altered brain development is hypothesized to explain environmental sensitivity and learning differences in ASD, it also contributes to the diverse and heterogeneous neuroanatomical and behavioral profiles observed in adults with the condition[9]. Importantly, subtle changes in social and attention-related brain systems

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are believed to precede the emergence of overt behavioral symptoms, often detectable long before clinical diagnosis[10].

While the structural neurobiology of ASD has been extensively studied in children, the neuroanatomical profile of ASD in adulthood remains less consistently defined. Early brain overgrowth, a consistent finding in young children with ASD[e.g., 3, 4], often normalizes or even reverses during adolescence and adulthood[11]. Consequently, previous structural magnetic resonance imaging (MRI) studies in adults have reported widespread but often inconsistent alterations in gray matter volume and cortical thickness. Voxel-based morphometry meta-analyses in adults with ASD have revealed increased gray matter volume in regions such as the superior temporal gyrus, postcentral gyrus, middle temporal gyrus, and parahippocampal gyrus, alongside decreased volume in the cerebellum and anterior cingulate cortex (ACC)[12]. Concurrently, surface-based morphometric analyses of cortical thickness have reported both thinning and thickening patterns across frontal, temporal, and parietal cortical regions[13]. Notably, thickness differences in areas closely related to social cognition, including the superior temporal sulcus, inferior frontal gyrus, and ACC, are frequently implicated in the social communication and sensory processing difficulties characteristic of ASD. The clinical relevance of these structural findings is further supported by multimodal studies, which report significant associations between behavioral measures of social communication (e.g., Autism Diagnostic Interview–Revised, ADOS) and cortical thickness in regions like the ACC and temporal areas[14]. Such evidence emphasizes the critical role of morphological changes in understanding the neuroanatomical basis of ASD symptoms and the direct link between brain structure and behavior.

Beyond local morphological changes, recent perspectives emphasize ASD as a disorder of brain connectivity. Traditional structural covariance networks have been used to map synchronized maturation between brain regions, but they require large group cohorts and cannot estimate connectivity at the individual level. To address this, we employ a novel method called Morphometric INverse Divergence (MIND). MIND is a structural similarity connectomics framework that derives single-subject cortical networks from standard T1-weighted MRI by comparing the full multivariate, vertex-level morphometric profiles of cortical parcels rather than relying on regional averages. In MIND, each cortical region is represented as a multidimensional distribution of multiple FreeSurfer-derived surface features (e.g., cortical thickness, surface area, gray-matter volume, sulcal depth, and mean curvature), and inter-regional similarity is quantified from the symmetrized Kullback–Leibler divergence (Jeffreys' divergence) between these distributions, typically estimated using a k-nearest-neighbor approach and transformed to yield a bounded (0–1) similarity measure[15]. By preserving within-region heterogeneity and cross-feature dependencies, MIND addresses key limitations of morphometric similarity networks (MSNs), which compress vertex-wise information into summary statistics and impose feature standardization assumptions that can be biologically unrealistic[16]. Large-scale benchmarking indicates that MIND networks show higher reliability, greater robustness to noise and parcellation choice, and stronger biological validity, including closer alignment with cortical symmetry and cytoarchitectonic organization, tighter coupling to tract-tracing connectivity, and stronger correspondence with transcriptional similarity and heritability[15, 17]. Consequently, MIND has begun to be adopted as a sensitive tool for probing subtle, network-level structural differences and plasticity across populations and training-related adaptations[18, 19].

In this study, we aimed to characterize the structural architecture of the social brain in ASD using a combined approach of vertex-wise cortical thickness analysis and MIND-based structural similarity networks. By focusing on adulthood, we specifically sought to determine whether structural alterations previously reported in ASD persist beyond neurodevelopment and remain evident in mature brain organization. We hypothesized that adults with ASD would exhibit distinct alterations in the structural connectivity of social brain regions that are not fully captured by local cortical thickness differences, reflecting enduring network-level abnormalities in social brain organization.

Materials and Methods

There is no need for ethics committee approval.

Participants

The structural MRI data used in this study were obtained from the dataset collected by Kolodny et al. (2020) in experiment 1 and are open-access dataset on OpenNeuro under the identifier ds002522[20]. The sample consisted of 24 adults with ASD and 24 neurotypical (NT) controls, aged 18–30 years (mean age: 22.1, SD = 3). Both groups were matched for sex, with each consisting of 16 females and 8 males. All participants had a minimum IQ of 80 (as measured by the Wechsler Abbreviated Scale of Intelligence) and normal or corrected-to-normal vision. There were no significant group differences in ages and non-verbal IQ (mean non-verbal IQ of ASD participants: 112.5 (SD = 18.76; range 79–160); NT participants: 113.8 (SD = 10.5; range 86–129); $t(46) = -0.28$, $p = 0.77$; mean age of ASD participants: 22.2 (SD = 3.6; range 18–30) years; NT participants: 21.9 (SD = 2.5; range 18–27) years; $t(46) = 0.42$, $p = 0.68$).

ASD diagnoses were confirmed using the Autism Diagnostic Interview-Revised (ADI-R)[21], the Autism Diagnostic Observation Schedule, second edition (ADOS-2)[22] and DSM-5[1] criteria. Clinical assessment procedures are reported in detail in the source study[23]. ASD symptom severity was quantified using the ADOS-2 Overall Comparison Score (ranging from 1 to 10). Strict exclusion criteria were applied regarding substance use (tobacco, illicit drugs, and alcohol) and medication stability. No new data were collected for the present study. All analyses were conducted on previously acquired, fully anonymized data that had been collected under prior approval from the University of Washington Institutional Review Board. Written informed consent had been obtained from all participants at the time of the original data collection. The current study involved secondary data analysis only and did not require additional ethical approval.

MRI Acquisition and Preprocessing

High-resolution T1-weighted anatomical images were acquired on a Philips Achieva 3T scanner with a 32-channel high-resolution head coil using a Magnetization Prepared Rapid Gradient Echo (MPRAGE) sequence with 1 mm isotropic resolution. These scans were part of a larger fMRI study (Experiment 1) consisting of two 1-hour sessions per participant,

which included functional localizers for the visual cortex and middle temporal area alongside experimental tasks[23].

Cortical reconstruction and volumetric segmentation were performed using FreeSurfer 8.0.0. recon-all pipeline[24]. This automated process generates cortical surface models and extracts five vertex-wise morphometric features: cortical thickness (CT), surface area (SA), gray matter volume (Vol), sulcal depth (SD), and mean curvature (MC). Cortical thickness was calculated as the perpendicular distance between the white matter–gray matter boundary and the pial surface. For group-level vertex-wise analyses, individual surfaces were registered to the fsaverage template, and cortical thickness maps were smoothed using a 10 mm Full-Width at Half-Maximum (FWHM) Gaussian kernel to improve the signal-to-noise ratio and account for inter-individual anatomical variability. No participants were excluded from the analysis, as all individuals provided high-quality T1-weighted MRI data that successfully passed the FreeSurfer 'recon-all' preprocessing pipeline without significant motion artifacts or topological errors.

Cortical Thickness

Vertex-wise group comparisons were performed using FreeSurfer's mri_glmfit with a General Linear Model (GLM), controlling for age and estimated Total Intracranial Volume (eTIV). Cluster-wise correction for multiple comparisons was applied using Monte Carlo simulation ($p < 0.01$, corresponding to a cluster-forming threshold of $z=2.0$) to identify initial clusters. The direction of the effect was tested using a positive one-tailed hypothesis. Clusters were then formally evaluated using a cluster-wise p- threshold (CWP) of $p<0.05$. All analyses were performed in the fsaverage surface space, and the results were corrected for both hemispheres independently. Furthermore, CT values for the region of interest (ROI) were extracted for each subject using the FreeSurfer-derived Destrieux atlas (aparc.a2009s). Subsequent group-level analyses of these ROI-based CT differences were conducted using an Ordinary Least Squares regression framework, consistent with the approach detailed for structural similarity analyses in the next section.

Morphometric Similarity Network (MIND) Construction

We constructed individual structural similarity networks using the MIND method. For each participant, the cortex was parcellated into ROIs using the Destrieux atlas. For every pair of regions, the similarity was calculated as the inverse of the symmetric Kullback-Leibler (KL) divergence between the multivariate distributions of the five structural features (CT, SA, Vol, SD, MC) sampled from all vertices within those regions. This resulted in a region-by-region similarity matrix for each subject, where edge weights represent the degree of structural affinity (0 to 1). Structural similarity was quantified using edge weights (pairwise connections) and nodal strength across 14 a priori (see Figure 1) social brain ROIs[25]. Nodal strength, representing the cumulative connectivity of a region, was calculated by averaging the edge weights for each selected ROI, considering only the connections within these 14 social brain ROIs. For each pairwise similarity (edge) and each selected region's average similarity (node strength), an Ordinary Least Squares regression was performed. The model included group as the primary predictor of interest, while eTIV was included as a nuisance covariate to account for individual differences in head size. The eTIV covariate was Z-score standardized prior to analysis to ensure scale-invariance. To account for multiple comparisons across the 14 regions, p-values were adjusted using the two-stage Benjamini-Hochberg False Discovery Rate (FDR_tsbh) procedure[26]. All statistical analyses were implemented in Python (version 3.14) using the statsmodels and scipy libraries.

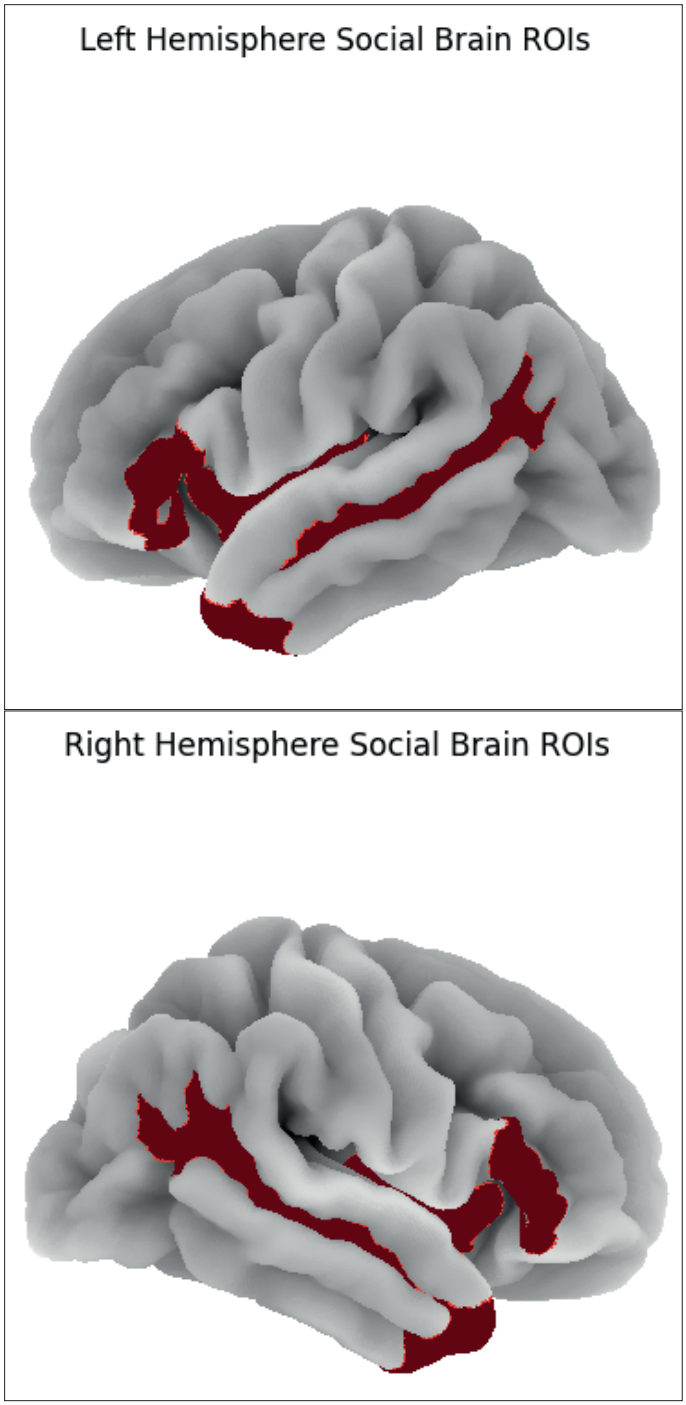


Figure 1: Social brain regions of interest (ROIs) on the fsaverage cortical surface

This figure depicts the specific ROIs associated with the social brain, as defined by the Destrieux atlas parcellation. The

selected ROIs are projected onto the fsaverage template surface and highlighted in red. Lateral views of the left and right hemispheres are presented independently on the left and right sides of the figure, respectively.

Result

Cortical Thickness

A whole-brain vertex-wise analysis revealed a significant main effect of eTIV on cortical thickness within the right inferior frontal gyrus (IFG), including the pars opercularis and adjacent regions. After correction for multiple comparisons (cluster-forming threshold: $-\log_{10}(p) > 2.0$, $p < 0.01$), a significant cluster was identified with a surface area of 626.22 mm² (Cluster-wise p [CWP] = 0.011). This cluster comprised 1223 vertices, with peak significance ($-\log_{10}(p) = 3.59$) located at MNI coordinates x = 44.4, y = 17.4, z = 8.2 (Table 1, Figure 2). Notably, the Group × eTIV interaction was not significant, indicating that increased

Table 1: Cluster-wise corrected results for the vertex-wise cortical thickness analysis showing the average effect of estimated total intracranial volume across groups.

Cluster No.	Max (stat)	Vtx-Max*	Size (mm²)	MNI X	MNI Y	MNI Z	CWP†	CWP ‡ Low	CWP ‡ High	NVtxs	Wght-Vtx	Annot
1	3.5910	42026	626.22	44.4	17.4	8.2	0.01057	0.00878	0.01236	1223	2753.53	parsoper-cularis

Footnotes:
* VtxMax: MNI coordinates correspond to the peak vertex.
† CWP indicates cluster-wise p-value (multiple-comparisons corrected).
‡ Confidence interval bounds are reported as provided by mri_surfcluster (CWPLow, CWPHigh).

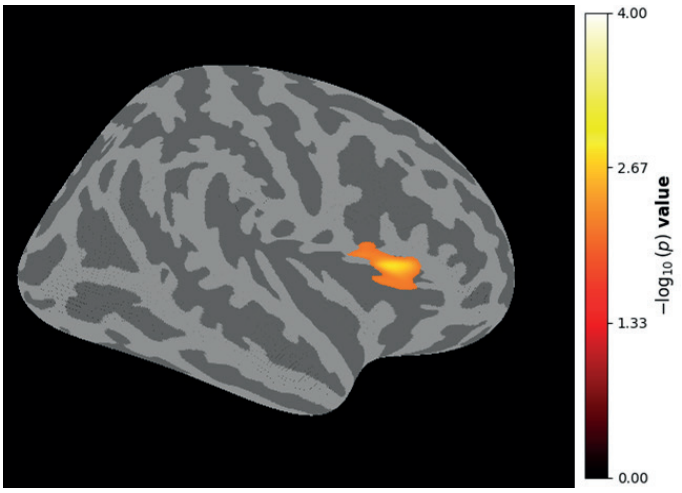


Figure 2. Vertex-wise cortical thickness results for the group by estimated total intracranial volume interaction contrast.

A whole-cortex, vertex-wise analysis identified a significant Group × estimated total intracranial volume (eTIV) interaction in the right inferior frontal gyrus (pars opercularis) and adjacent regions. The displayed cluster survived cluster-wise correction at a cluster-forming threshold of: $-\log_{10}(p) > 2.0$, $p < 0.01$), with a surface area of 626.22 mm² and 1223 vertices (cluster-wise p-value, CWP = 0.011). The peak vertex showed a maximum statistic of 3.59 at MNI coordinates (44.4, 17.4, 8.2). The color scale represents the cluster-wise p-value (), where brighter colors (yellow) indicate higher statistical significance.

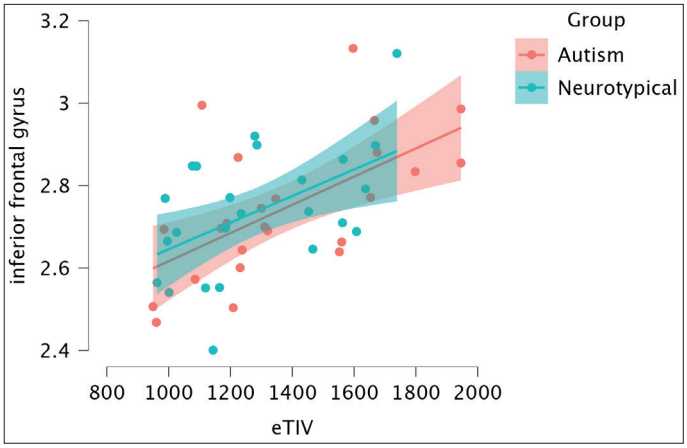


Figure 3. Relationship between eTIV and cortical thickness in the right inferior frontal gyrus.

The scatter plot illustrates the positive correlation between eTIV (mm^3) and cortical thickness (mm) within the right IFG for the Autism (red) and Neurotypical (blue) groups. Solid lines represent group-specific linear regression fits, with shaded regions indicating 95% confidence intervals.

Figure 4. Nodal connectivity strength differences in social brain regions between ASD and NT groups.

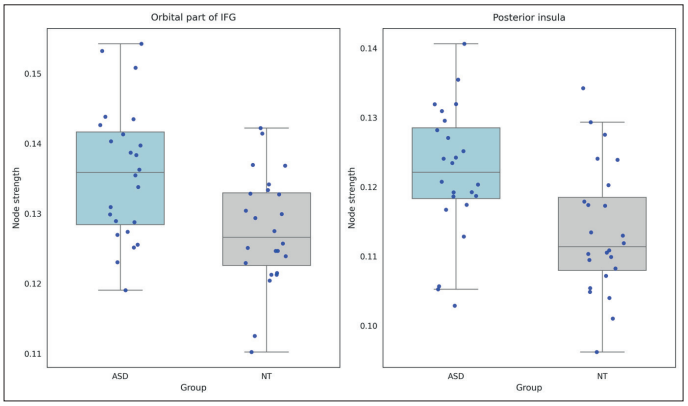
Individual subject-level node strength values are shown as jittered scatter points overlaid on boxplots representing group distributions (box: interquartile range; horizontal line: median; whiskers: 1.5×IQR). The left panel corresponds to the orbital part of the right inferior frontal gyrus region of interest (ROI), showing a significant group difference ($p_{FDR} = 0.04$). The right panel represents the right long insular gyrus and central sulcus of the insula (posterior insula) ROI, also demonstrating a significant difference ($p_{FDR} = 0.04$).

Abbreviations: ASD, autism spectrum disorder; NT, neurotypical; MIND, Morphometric INverse Divergence.

eTIV is associated with greater cortical thickness in this region, regardless of group membership ($Beta_{ASD}$ = 0.000341; $Beta_{NT}$ = 0.000598, Figure 3). ROI based analysis revealed no significant group differences in CT across the predefined social brain network regions ($p_{FDR} > 0.05$).

Structural Similarity Networks

The MIND-based structural similarity network analysis revealed that while whole-brain comparisons and ROI based edge weights did not yield significant group differences, the ASD group exhibited significantly increased nodal strength compared to the NT group, indicating a pattern of localized hyper-connectivity. This increased structural similarity was specifically identified in two key regions within the right hemisphere. First, the ASD group showed significantly elevated nodal strength with large effect size in the orbital part of the right IFG compared to the NT group (mean node strength of ASD participants: 0.136, NT participants: 0.128, t (46) = 3.21, p_{FDR} = 0.04, Cohen's d = [0.93], Figure 4). Second, a similar pattern of hyper-connectivity was observed in the right posterior insula (long insular gyrus and central sulcus of the insula) in the ASD group compared to the NT group with large effect size (mean node strength of ASD participants: 0.122, NT participants: 0.114, t (46) = 3.11, p_{FDR}=0.04, Cohen's d = [0.90], Figure 3).



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Abbreviations: ASD, autism spectrum disorder; NT, neurotypical; MIND, Morphometric INverse Divergence.

Discussion

This study investigated differences in structural architecture of the social brain in individuals with ASD compared to NTs, focusing on cortical thickness and Morphometric INverse Divergence -based structural similarity networks. By focusing on adulthood, we specifically sought to determine whether structural alterations previously reported in ASD persist beyond neurodevelopment and remain evident in mature brain organization. Our findings reveal distinct patterns of altered brain structure in ASD, particularly within key regions of the social brain network.

Our MIND-based structural similarity network analysis revealed significant alterations in nodal strength within the ASD group. While whole-brain and ROI-based edge-level comparisons did not yield significant group differences, the ASD group consistently exhibited increased nodal strength in the orbital part of the right IFG and the right posterior insula. Given that the MIND metric reflects the similarity of cortical cytoarchitecture, and architectonically similar brain regions are more likely to be axonally connected, this increased nodal strength is indicative of localized hyper-connectivity[17, 27]. This suggests that these specific regions in ASD have become more structurally homogenized or tightly integrated with their connected network elements, potentially serving as a proxy for altered connectivity. The IFG, especially its orbital and opercular parts, is a central hub for social communication, language processing, and theory of mind[28, 29]. Hyper-connectivity in this region could reflect an atypical organization of local cortical circuits, potentially contributing to the characteristic social and communication challenges observed in ASD. Similarly, the posterior insula is a critical node for interoception, emotional awareness, and social processing. Increased nodal strength here might indicate altered integration of sensory and emotional information, which could impact social perception and response in individuals with ASD. These findings align with theories suggesting that ASD involves both local over-connectivity and long-range under-connectivity, with our results specifically supporting the notion of localized structural hyper-connectivity. The convergence of findings in the IFG and posterior insula regions essential for integrating external social cues with internal bodily states, suggests that the structural scaffolding for social cognition is fundamentally reorganized in ASD. The presence of these alterations in adulthood indicates that they are stable, enduring features of the autistic brain rather than transient developmental delays.

The absence of significant group differences in whole-brain or ROI-based edge weights, despite significant nodal strength differences, suggests that the alterations in structural similarity in ASD might be more pronounced at the local integration level (nodal strength) rather than in the overall pattern of pairwise connections (edge weights) during adulthood. This could imply a more subtle, region-specific reorganization of structural networks.

Beyond the structural similarity alterations, our analysis also revealed a robust positive association between eTIV and cortical thickness within the right IFG, specifically encompassing the pars opercularis and adjacent regions. The significant average slope identified across the cohort suggests that global brain volume is a critical determinant of local cortical micro-architecture in this region. This finding underscores a strong morphometric coupling between brain volume and thickness, which appears to be a fundamental characteristic of the IFG's structural organization. Notably, while the statistical contrast focused on the average effect across the entire sample and direct group comparisons did not yield significant differences,

visual inspection of the correlation slopes (Figure 3) suggests a qualitatively more pronounced relationship in the ASD group. This observed trend, despite the non-significant interaction, may point towards an intensified developmental scaling or altered coupling between global brain volume and local cortical thickening in ASD within a region critically involved in language, social cognition, and executive functions[8, 13].

The meticulous control for eTIV in our analyses was particularly crucial, especially when investigating social brain regions. Brain size, as reflected by eTIV, is a major source of inter-individual variability in brain morphology and can significantly confound group comparisons if not properly accounted for[30]. In the context of social brain research, where subtle structural differences might underlie complex behavioral phenotypes, failing to control for eTIV could lead to spurious findings or mask true regional effects Wierenga[31]. Our results further highlight the importance of considering brain size as a modulating factor, suggesting that structural differences in CT are closely linked to overall brain volume[11]. Therefore, our approach ensures that the observed associations and trends in cortical thickness and structural similarities are independent of overall brain size, thereby enhancing the specificity and interpretability of our findings.

While our study identifies a specific neuroanatomical signature in adults with ASD, the relatively modest sample size (N=48) must be acknowledged. This may limit the generalizability of our findings to the broader ASD population, which is characterized by significant clinical and biological heterogeneity. Future studies with larger, multi-site datasets are needed to replicate these findings and explore how this structural similarity patterns vary across different clinical phenotypes and age groups.

Conclusion

Our findings reveals that adults with ASD present with a distinct neuroanatomical signature, marked by localized structural hyper-connectivity in the inferior frontal gyrus and insula. This suggests that, even in adulthood, specific nodes within the social brain network exhibit an atypically high degree of structural synchronization with the broader brain network, rather than global alterations. These results strongly support the notion of altered connectivity in ASD, emphasizing the persistence of these structural atypicalities into later developmental stages. Moreover, this study demonstrates the power of multi-feature network approaches, such as MIND, in elucidating the complex and potentially age-dependent biological basis of social communication deficits. Future research should aim to integrate these structural findings with functional and behavioral data to better understand their clinical relevance.

Patient informed consent

There is no need for patient informed consent.

Ethics committee approval

There is no need for ethics committee approval.

Conflict of interest

There is no conflict of interest to declare.

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Author contribution subject and rate

- Bernis Sütçübaşı (40%): Design the research, formulated a hypothesis, analyses and wrote the whole manuscript.
- Batuhan Memiş: (20%): Organized and reported the data, analyses, contributed with comments on research design.
- Ebru Durdu (10%): Provided support in literature review and supported manuscript preparation.
- Stefani Helin Yavaş (10%): Provided support in literature review and supported manuscript preparation.
- Yağmur Tekin (10%): Provided support in literature review and supported manuscript preparation.
- Şeyma Bayram (5%): Contributed to analysis and supported manuscript preparation.
- Melis Zeybey (5%): Contributed to analysis and supported manuscript preparation.

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