Identifying the Characteristics of Brain Regions in Individuals with Different Neurocognitive Characteristics Based on Functional MRI Data

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Abstract. Autism spectrum disorders (ASD) do not have objective neurobiological markers, and diagnosis is based solely on behavioral scales. Functional resting MRI allows you to study disorders of brain connectivity, but existing approaches face problems of high dimensionality of data, overfitting and low interpretability. The article proposes a methodology for identifying stable, interpretable biomarkers of functional connectivity, reliably distinguishing individuals with ASD and neurotypical controls. To reduce dimensionality and ensure interpretability, two-stage feature selection (t-test with FDR correction, SelectKBest) and logistic regression are used. The model achieved an accuracy of 71.8% (AUC = 80.5%, Sensitivity = 74.4%, Specificity = 69.6%) and identified 22 stable associations. Mapping revealed a predominant localization in the DMN with pronounced hypoconnectivity (17↓ / 4↑), as well as hyperconnectivity of subcortical structures (6↑ / 3↓) and the visual network (2↑).

Keywords: autism spectrum disorder · functional connectivity · resting-state fMRI · machine learning · brain networks

1 Introduction

Autism spectrum disorders (ASD) are a heterogeneous group of neurodevelopmental conditions characterized by deficits in social interaction and communication, as well as restricted and repetitive patterns of behavior, interests, and activities [1]. Despite considerable progress in understanding the genetic and neurobiological mechanisms underlying ASD, clinical diagnosis still relies primarily on behavioral criteria, including standardized scales and expert assessments. The subjectivity of these methods, their dependence on the clinician's experience, and the inability to detect the disorder before the onset of behavioral symptoms underscore the urgent need for objective, quantitative biomarkers.

Resting-state functional magnetic resonance imaging (rs-fMRI) has emerged as a powerful tool for investigating the functional architecture of the brain. Unlike structural neuroimaging, fMRI allows for the assessment not only of anatomical integrity but also of functional interactions among brain regions, which is particularly important for understanding systemic disruptions in ASD. The concept of functional connectivity, based on

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the synchronization of BOLD time series, enables quantitative evaluation of integrative brain processes and the identification of patterns specific to various neurological and psychiatric conditions. Low-frequency spontaneous BOLD fluctuations have been shown to correlate across functionally connected, including anatomically distant, brain regions, forming large-scale neural networks [2]. Among these networks are the default mode network, the salience network, and the frontoparietal control network, which play key roles in cognitive and socio-emotional processes. In ASD, numerous studies report alterations in functional connectivity, yet findings remain inconsistent, showing both hypo- and hyperconnectivity in various networks, likely due to sample heterogeneity, age-related differences, and methodological factors [3].

To date, a variety of approaches have been proposed for classifying ASD using fMRI data, ranging from classical machine learning methods (logistic regression, support vector machines, random forest) to modern deep learning models, including graph neural networks that incorporate the topology of brain networks [4, 5]. Despite high classification accuracies reported in some studies, the reproducibility of these models remains limited, especially when applied to independent datasets.

A key challenge is the high dimensionality of the data. Representing functional connectivity as a matrix of pairwise interactions between brain regions can result in thousands of potential features even with moderately detailed parcellations. With limited sample sizes, this creates the classical "curse of dimensionality," increasing the risk of overfitting.

Equally important is interpretability. Complex models often operate as "black boxes," making it difficult to determine the contribution of individual connections or networks to the final decision. This limits the clinical utility of such models, as clinicians require not only probabilistic estimates of diagnosis but also insights into the neurobiological basis of these decisions and the identification of structures involved in the pathological process. Moreover, in some studies, reported high accuracies may result from methodological issues such as data leakage between training and test sets, feature selection prior to data splitting, or insufficient control of overfitting.

Another critical aspect is biomarker stability. Functional connectivity analyses can vary substantially depending on data splits, preprocessing parameters, and analytical approaches, even when identical pipelines are applied. In the context of high inter-individual and inter-site variability (e.g., in multicenter datasets such as ABIDE), ensuring reproducibility becomes a key challenge.

Given these issues—the lack of objective biomarkers, heterogeneity of published data, high dimensionality, instability of features, and limited interpretability of models—developing methods for identifying stable, interpretable, and neurobiologically meaningful patterns of functional connectivity is of particular relevance. The present study aims to develop an approach capable of detecting functional connections that are reproducible across different sample splits and exhibit high discriminative power in distinguishing individuals with ASD from neurotypical controls.

2 Material and Methods

2.1 Data Source

The study was carried out on the basis of open data from the ABIDE I (Autistic Brain Imaging Data Exchange I) consortium, a large international project for the exchange of neuroimaging data for the study of autism spectrum disorders (ASD). The project was created to provide the scientific community with open access to standardized fMRI data to accelerate research in the field of autism [6].

Within the framework of this work, the main source of data for analysis is functional MRI recordings at rest, since the purpose of the study is to identify specific areas of the brain that distinguish individuals with ASD from neurotypical ones.

2.2 Data Preprocessing and Sampling

The fMRI data were pre-processed using a standardized CPAC (Configurable Pipeline for the Analysis of Connectomes) pipeline. This pipeline implements typical processing steps, including: time shift and motion correction, normalization, spatial antialiasing, regression of interfering signals, and bandpass temporal filtering. The use of CPAC ensures the comparability of data from different centers and increases the reproducibility of results [7].

After removing records with missing age, sex, and diagnosis values, and excluding subjects with incomplete functional connectivity data, the final sample consisted of 849 subjects (Table 1).

Table 1. Demographic characteristics.

Group	Total	Male	Female
Control	458	373	85
ASD	391	343	48
Total	849	716	133

The gender imbalance in the sample reflects the characteristics of the ABIDE I database formation in the early 2010s, when diagnostic criteria and tools were primarily developed on male cohorts, and ASD in females was systematically underestimated due to symptom masking [8]. This limitation was taken into account when interpreting the results.

2.3 Functional Connectivity Construction

For each study participant, the raw fMRI data consist of BOLD signal time series recorded at each point in the brain. To transition from the voxel to the regional level of analysis, the CC200 atlas [9] was used, which partitions the brain into 200 approximately equal functional regions of interest (ROIs). The CC200 atlas was chosen due to

its functional homogeneity, which is particularly important for the analysis of functional connectivity. Time series were averaged within each region, reducing data dimensionality and minimizing the influence of physiological noise.

Next, a 200 × 200 functional connectivity matrix was computed for each subject. Each element of this matrix reflects the strength of the connection between two regions and was calculated as the Pearson correlation coefficient between the corresponding time series [10]. Correlation values range from –1 to 1: positive values indicate synchronous activity between regions, whereas negative values indicate oppositely directed dynamics, where one region is active while the other is deactivated.

Since the connectivity matrix is symmetric with respect to the main diagonal, and diagonal elements do not convey information about interactions between different regions, only the upper triangle of the matrix was used for analysis. For 200 regions, this results in 19,900 unique connections, each representing a separate feature characterizing the functional interaction between a specific pair of brain regions.

Two covariates, age and sex of the participant, were added to the feature set, as these factors are known to influence measures of functional connectivity [11]. Consequently, each subject was represented by a feature vector of 19,902 elements, suitable for training machine learning models and identifying the most significant brain regions.

2.4 Feature Selection

The original dataset's high dimensionality (19,902 features for 849 subjects; $p/n \approx 23.4$) posed a risk of overfitting, where the model could capture noise instead of true patterns. Conducting a large number of statistical tests without correction also increases false positive results (approximately 995 expected at $\alpha = 0.05$), necessitating multiple comparison correction.

A two-step dimensionality reduction approach combining statistical filtering and feature ranking was implemented. In the first step, a two-sample Welch t-test was applied, followed by Benjamini–Hochberg false discovery rate correction, to identify connections that differed significantly between groups while controlling the proportion of false discoveries. Feature selection was performed exclusively on training data to avoid information leakage and inflated performance estimates.

In the second step, the SelectKBest method with ANOVA F-statistics was used to select the 100 most informative features. This univariate ranking approach evaluates each feature independently for its ability to discriminate between classes. Using ANOVA F-statistics allows quantitative ranking of features by discriminative power, while SelectKBest ensures control over the number of features, important for interpretability and subsequent neurobiological analysis.

Feature selection was performed within cross-validation folds, preventing data leakage and providing an unbiased estimate of model generalizability. Stability was assessed by the frequency of feature occurrence across five folds, resulting in 22 functional connections with the highest reproducibility.

2.5 Classification and Model Evaluation

To solve the classification problem, a multi-stage methodology was developed. First, the dataset was split into training (80%) and test (20%) samples, preserving class proportions. This separation ensures an independent dataset for final evaluation, which is not involved in training or hyperparameter tuning.

A 5-fold cross-validation was applied to the training sample. Within each fold, a two-step feature selection was performed: initially, an FDR-corrected t-test identified statistically significant differences between groups, followed by the SelectKBest method with ANOVA F-statistics to select the 100 most informative connections. All feature selection steps were performed exclusively on training data within each fold to avoid data leakage and ensure unbiased model evaluation. This procedure reduced the feature space from 19,902 to 100 features.

After selection, features were standardized, and a logistic regression model with L2 regularization was trained. Logistic regression was chosen for its interpretability: the sign of each coefficient directly indicates the direction of change in functional connectivity (hyper- or hypoconnectivity), allowing straightforward neurobiological interpretation. Unlike more complex models, logistic regression provides a balance between classification performance and interpretability.

The stability of selected features was assessed by their frequency across the five cross-validation folds. Features consistently selected in each fold were considered the most reliable potential biomarkers. The final model was trained on the entire training set and evaluated on the held-out test set to assess generalizability.

Model performance was evaluated using several metrics to capture different aspects of classification quality. ROC-AUC was the primary metric because it reflects the model's ability to distinguish between classes independently of the threshold and allows comparison with other studies. Sensitivity was used to assess the proportion of correctly classified ASD participants, which is critical for diagnostic purposes, as missing a case has more serious consequences than a false positive. Specificity reflects the proportion of correctly classified neurotypical controls. Accuracy is reported for comparison with other studies but is not the primary metric for unbalanced datasets (Table 2).

Table 2. Logistic regression model performance on training and test samples.

Sample	Accuracy	AUC	Sensitivity	Specificity
Train	0.7202	0.8137	0.7668	0.680
Test	0.7176	0.8052	0.7436	0.6957

The minimal difference between training and test performance indicates that the model generalizes well and is not overfitted. Of particular importance is the high sensitivity: the model correctly identifies three out of four ASD cases, which is critical for potential clinical applications.

The ROC curves for the training and test sets, along with the confusion matrix, are presented in Fig. 1. This figure visualizes both the discriminatory power of the model

across thresholds and the classification outcomes, highlighting the reliability of predicted labels.

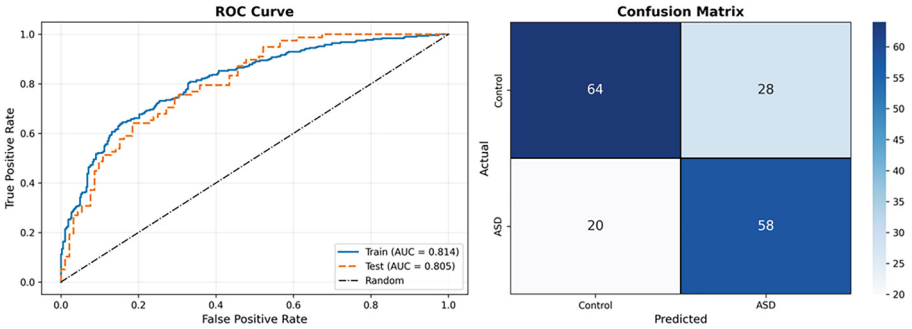

Fig. 1. ROC curves and confusion matrix for the logistic regression model.

To evaluate whether nonlinear decision boundaries could improve classification performance, an additional experiment was conducted using a Support Vector Machine (SVM) with a radial basis function (RBF) kernel. The same data partitioning strategy, preprocessing pipeline, and feature selection procedure were applied. Hyperparameters were optimized on the training data using grid search.

The nonlinear SVM achieved performance comparable to logistic regression on the independent test set, without providing a substantial improvement in classification performance (Table 3).

Table 3. SVM-RBF model performance on training and test samples.

Sample	Accuracy	AUC	Sensitivity	Specificity
Train	0.7275	0.8184	0.7923	0.6721
Test	0.7176	0.8060	0.7692	0.6739

Although the nonlinear SVM slightly improved sensitivity, the overall classification performance remained nearly identical to logistic regression. Given the substantially higher interpretability of logistic regression coefficients for neurobiological interpretation, logistic regression was retained as the primary model for subsequent interpretation of stable functional connections.

2.6 Decoding and Mapping Stable Functional Connections

After identifying 22 stable functional connections consistently selected across all five cross-validation folds, the next step was their anatomical identification and mapping to functional brain networks for neurobiological interpretation. The original features, represented as FC_N indices, were transformed into specific pairs of brain regions. Each

feature corresponds to a unique pair of ROIs located in the upper triangle of a 200 × 200 functional connectivity matrix. The linear index N was converted to ROI numbers (i, j), which were then mapped to anatomical names and coordinates from the CC200 atlas in standard MNI space. For each connection, both regions involved and the direction of change (hyper- or hypoconnectivity) were determined from the sign of the logistic regression coefficient.

For system-level interpretation, the 28 unique ROIs involved in the 22 stable connections were mapped to the Yeo7 functional networks [12], which divide the cerebral cortex into seven large-scale networks: Visual, Somatomotor, Dorsal Attention, Ventral Attention, Limbic, Frontoparietal, and Default. Yeo7 mapping was chosen because the brain functions as an integrated system, and it allows moving from isolated anatomical regions to network-level interpretation, enabling identification of functional systems affected in ASD and comparison with contemporary network-based studies. Alternative approaches, such as mapping to Brodmann cytoarchitectonic areas, are technically more challenging, requiring additional transformations, whereas Yeo7 mapping based on MNI coordinates provides a direct and methodologically sound approach.

The mapping was performed using ROI centroids in MNI space extracted from the CC200 atlas and the nilearn library, which identifies the nearest functional network based on Euclidean distance. To improve reliability for ROIs located near network boundaries, an extended search radius of 10 mm was applied. Subcortical structures not included in Yeo7 cortical networks were assigned to a Background category for separate analysis (Fig. 2).

Table 4. Distribution of ROIs and stable functional connections across Yeo7 networks.

Yeo7 Network	Number of ROIs	Total Connections	Enhanced in ASD	Weakened in ASD
Default	12	21	4	17
Somatomotor	6	7	3	4
Background	5	9	6	3
Frontoparietal	2	2	1	1
Ventral Attention	2	3	2	1
Visual	1	2	2	0

This decoding and mapping procedure allowed moving from abstract indices to concrete anatomical and functional structures, providing a basis for interpreting altered functional connectivity in ASD within large-scale neural networks.

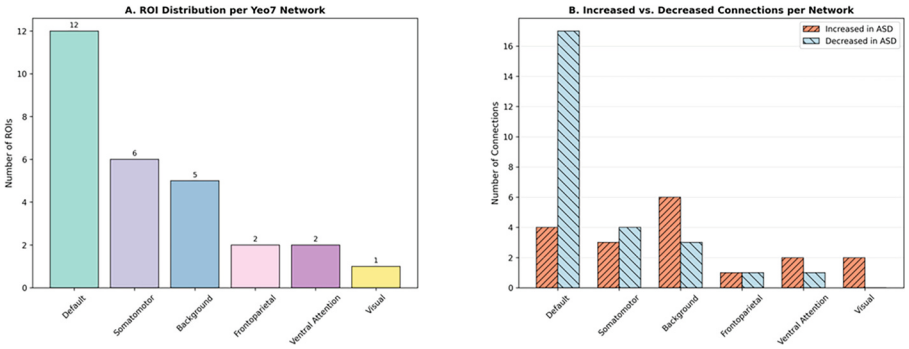

Fig. 2. Distribution of stable functional connections across Yeo7 networks. (A) Number of ROIs per network. (B) Number of connections increased vs. decreased in ASD per network.

3 Discussion

The obtained classification accuracy is comparable to the results of modern studies using the ABIDE dataset. A comparison of key performance metric is presented in Table 5.

Table 5. Accuracy comparison with studies using rs-fMRI data

Research	Accuracy
Abraham et al. (2017) [13]	67%
Heinsfeld et al. (2018) [4]	70%
Dong et al. (2025) [14]	72%
Our study	71.8%

The studies presented in Table 5 are based on different machine learning approaches, including deep neural networks and graph-based models. Despite relying on a comparatively simple and interpretable logistic regression framework, the proposed methodology achieved classification accuracy comparable to these approaches, while additionally allowing direct neurobiological interpretation and identification of stable functional biomarkers, which is particularly important for potential clinical applications.

Among the 100 selected features, 22 functional connections were consistently identified across all five cross-validation folds. Their repeated selection across different training subsets suggests that these connections are relatively stable and not strongly dependent on a particular data split. The complete list of reproducible ROI-to-ROI functional connections is presented in Appendix (Table 6). It should be noted that some anatomical labels appear multiple times because the CC200 atlas contains several distinct ROIs localized within the same anatomical structure but belonging to different functional regions.

The additional comparison with a nonlinear SVM classifier demonstrated performance nearly identical to logistic regression on the independent test set. This may indicate that, after feature selection, the differences between ASD and control groups can already be effectively captured by a linear model. Importantly, increasing model complexity did not lead to a substantial improvement in classification performance, which may suggest limited effectiveness of nonlinear models for rs-fMRI classification tasks after feature space reduction.

At the same time, logistic regression offers an important practical advantage in terms of interpretability. Unlike nonlinear "black-box" models, logistic regression coefficients can be directly interpreted in terms of increases or decreases in functional connectivity. This makes it possible to relate classification results to specific neurobiological mechanisms and identify reproducible biomarkers that may be clinically meaningful.

An important limitation of the present study is the pronounced gender imbalance of the ABIDE I dataset, where males constitute the majority of participants. This imbalance is partly related to the fact that earlier ASD research primarily focused on male cohorts, while autism in females was diagnosed less frequently [8]. Consequently, the 22 stable functional connections identified in this work may predominantly reflect connectivity patterns characteristic of the male ASD phenotype. Previous neuroimaging studies suggest that females with ASD may show some differences in brain organization and functional connectivity patterns, including regions associated with social cognition, the Default Mode Network (DMN), and mechanisms involved in symptom compensation [15]. Therefore, additional validation in larger and more sex-balanced cohorts is necessary before generalizing the identified biomarkers to female ASD populations.

Mapping of stable functional connections onto the Yeo7 network revealed a characteristic pattern of alterations in ASD: pronounced hypoconnectivity within the Default Mode Network (17↓ / 4↑), accompanied by relative hyperconnectivity in subcortical regions (6↑ / 3↓) and the visual network (2↑). Overall, these results are consistent with current views of ASD as a disorder involving altered organization of large-scale brain networks.

The observed hypoconnectivity of the DMN is supported by a recent large-scale meta-analysis [16], which integrated data from functional and structural neuroimaging studies. The authors reported reduced functional activity in core DMN regions, including the left angular gyrus, bilateral anterior cingulate cortex, and medial prefrontal cortex. Several of these regions, including the angular gyrus and anterior cingulate cortex, were also represented among the stable ROIs identified in our study, supporting the reproducibility of DMN-related alterations. Importantly, the meta-analysis revealed partial overlap between functional and structural abnormalities across multiple brain regions, suggesting that ASD is associated with both functional and anatomical alterations.

The increased involvement of subcortical structures observed in our results is consistent with findings from Hadjikhani et al. [17], who demonstrated abnormally elevated activation in subcortical regions—including the superior colliculus, pulvinar nucleus of the thalamus, and amygdala—during constrained eye contact in individuals with ASD. In our study, the thalamus and midbrain show a predominance of hyperconnectivity (6↑ / 3↓), which may reflect altered sensory gating and atypical processing of socially relevant stimuli. However, given that [17] is based on task-related fMRI, this correspondence

should be interpreted as indirect rather than direct evidence of resting-state connectivity alterations.

The mixed pattern observed in the somatomotor network (3↑ / 4↓) suggests multi-directional alterations of functional connectivity in ASD. In particular, the postcentral gyrus and cerebellum in our results participate in both increased and decreased connections, indicating that connectivity changes within the sensorimotor system do not follow a single consistent pattern. These findings are consistent with previous work [18], which reported reduced connectivity within the sensorimotor network together with increased interactions between the sensorimotor and other functional networks in ASD. This may reflect atypical processing and coordination of sensorimotor information rather than only alterations in motor function.

The observed hyperconnectivity within the visual network may reflect atypical weighting of perceptual processing in ASD. This interpretation is consistent with theories emphasizing enhanced or preferential processing of visual information; however, it should be considered a hypothesis requiring further validation in studies directly linking functional connectivity patterns with behavioral measures.

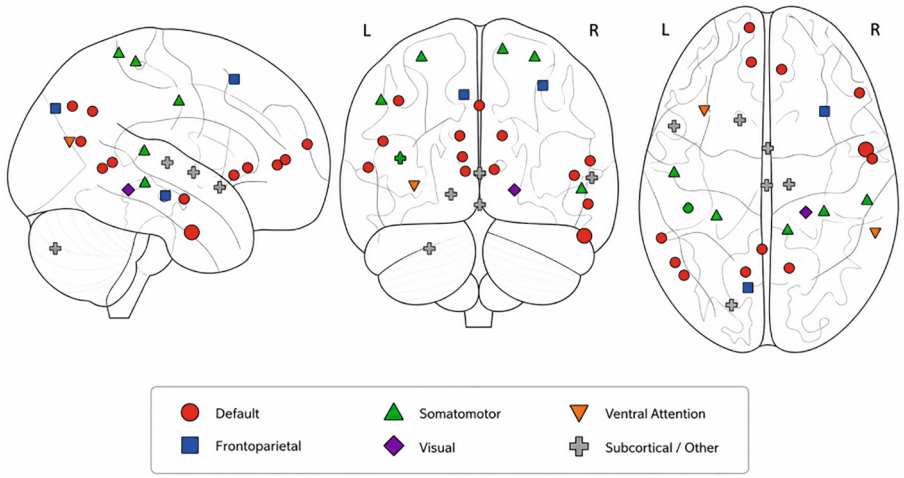

Fig. 3. Localization of reproducible brain regions in ASD by Yeo7 network parcellation.

As shown in Fig. 3, the 22 stable ROIs identified in ASD are distributed across multiple Yeo7 networks. While the figure illustrates the spatial distribution, the predominance of DMN hypoconnectivity and the involvement of subcortical and visual systems are quantitatively summarized in Table 4 and supported by the direction of logistic regression coefficients.

4 Conclusion

In this study, a methodology was developed to identify stable functional connections that distinguish individuals with autism spectrum disorder (ASD) from neurotypical controls using resting-state functional MRI data. Open-access data from the ABIDE consortium

were analyzed, including 849 subjects and 19,902 functional connections calculated using the CC200 atlas.

To address the high dimensionality of the data ($p/n \approx 23.4$) and control for multiple comparisons, a two-stage feature selection approach was applied: first, the Welch t-test with FDR correction, followed by the SelectKBest method with ANOVA F-statistics to select the 100 most informative connections. A critical methodological decision was to perform feature selection exclusively on the training data within each cross-validation fold, thereby preventing data leakage and providing an unbiased assessment of model generalizability. The stability of feature selection was evaluated by the frequency of feature occurrence across all five folds, which made it possible to identify 22 functional connections with the highest reliability.

On the independent test set, the final logistic regression model achieved an accuracy of 71.8%, an AUC of 80.5%, a sensitivity of 74.4%, and a specificity of 69.6%. The small difference between training and test performance indicates limited overfitting and good generalizability of the model. A comparison with the nonlinear SVM-RBF classifier demonstrated nearly identical results, suggesting that more complex nonlinear approaches do not provide a substantial advantage within the reduced feature space used in this study. At the same time, the obtained performance is comparable to that reported in recent ABIDE-based studies, including approaches using neural network architectures, supporting the validity of the selected interpretable model.

Mapping the identified ROIs onto the Yeo7 functional networks revealed that the detected alterations were predominantly localized within the Default Mode Network and were mainly characterized by hypoconnectivity. These findings are consistent with large meta-analyses and further support the central role of the DMN in ASD-related social dysfunction. In addition, relative hyperconnectivity of subcortical structures, including the thalamus and midbrain, was observed and may reflect altered sensory gating and atypical processing of socially relevant stimuli. Hyperconnectivity within the visual network may indicate atypical weighting of perceptual processing, although this interpretation requires further validation. The somatomotor network demonstrated a mixed pattern of increased and decreased connectivity, likely reflecting the heterogeneity of motor and sensory manifestations associated with ASD.

The patterns identified in this study are consistent with findings reported in previous research, supporting the reproducibility of the selected biomarkers and their correspondence with current neurobiological models of ASD. At the same time, several limitations should be considered. The ABIDE I dataset is characterized by a substantial gender imbalance, reflecting diagnostic practices of the early 2010s and the under-recognition of ASD in females due to symptom masking and features of the female ASD phenotype. As a result, the detected biomarkers may primarily reflect connectivity patterns characteristic of male ASD populations. Additional validation is therefore required in larger and more balanced samples, as well as in ethnically diverse and independent cohorts.

Further studies may examine whether the observed functional connectivity alterations are associated with the severity of ASD symptoms measured using clinical scales such as ADOS and ADI-R. This may help clarify the clinical relevance of the proposed biomarkers. In addition, alterations observed in visual and subcortical systems may be related to atypical sensory processing in ASD and could represent potential targets for

new therapeutic approaches. However, these assumptions require additional validation in clinical settings.

Thus, the proposed approach for detecting stable ASD biomarkers represents a step toward more objective diagnostic methods and a better understanding of the neurobiological mechanisms underlying the disorder. The 22 reproducible functional connections identified in this study may serve as a foundation for the development of medical decision-support systems and for further research into the pathogenesis of autism spectrum disorder.

Appendix

Table 6. Stable functional connections identified across all cross-validation folds

ROI 1	ROI 2	Connectivity in ASD
Precuneus (19)	Middle temporal gyrus (140)	Decreased
Middle temporal gyrus (140)	Precuneus (174)	Decreased
Anterior cingulate (5)	Middle temporal gyrus (140)	Decreased
Precuneus (3)	Middle temporal gyrus (140)	Decreased
Midbrain (68)	Middle frontal gyrus (127)	Decreased
Midbrain (68)	Superior temporal gyrus (153)	Increased
Insula (20)	Inferior frontal gyrus (144)	Decreased
Cerebellum (120)	Transverse temporal gyrus (200)	Increased
Superior parietal lobule (65)	Precuneus (147)	Increased
Angular gyrus (2)	Middle temporal gyrus (140)	Decreased
Postcentral gyrus (96)	Postcentral gyrus (154)	Decreased
Thalamus (18)	Middle temporal gyrus (107)	Increased
Anterior cingulate (22)	Middle temporal gyrus (140)	Decreased
Thalamus (18)	Middle temporal gyrus (117)	Increased
Middle temporal gyrus (69)	Thalamus (148)	Increased
Frontal pole (104)	Middle temporal gyrus (140)	Decreased
Parahippocampal gyrus (48)	Middle temporal gyrus (69)	Increased
Thalamus (18)	Caudate (47)	Decreased
Postcentral gyrus (28)	Postcentral gyrus (154)	Decreased
Parahippocampal gyrus (48)	Middle temporal gyrus (117)	Increased
Angular gyrus (82)	Middle temporal gyrus (140)	Decreased
Midbrain (68)	Middle temporal gyrus (117)	Increased

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