ISSN 2278-2540 | DOI: 10.51583/IJLTEMAS | Volume XIV, Issue IX, September 2025

NAPIFASD: Design of an Iterative Neuroadaptive Multimodal Framework for Predictive Modeling, Subtype Discovery, and Intervention Simulation in Autism Spectrum Disorders

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DOI: https://doi.org/10.51583/IJLTEMAS.2025.1409000074

Abstract: The current increase in the prevalence of Autism Spectrum Disorder (ASD) and the need for early personalized intervention mandate the development of predictive systems that reach beyond static diagnosis. Machine learning models for ASD, as it exists today, are largely inflicted by limitations, including restricted data modalities, low generalization across populations, and limited interpretability — all of which become significant constraints for their clinical relevance and translational value. Furthermore, such systems often do not model the temporal development of neurodevelopmental markers and overlook the crucial interlink between diagnosis and a strategy for actionable intervention. To plug these gaps, we present the NeuroAdaptive Predictive and Interventional Framework for Autism Spectrum Disorder (NAPIF-ASD)—a multimodal, interpretable, and intervention-aware pipeline to enhance ASD prediction and intervention planning. The framework initiates with Temporal Neuro-Behavioral Graph Embedding (TNGE-Net), which jointly models individual developmental trajectories exploiting the power of graph neural networks over longitudinal neuroimaging and behavioral data. Then, Domain-Regularized Adaptive Clustering Ensemble (DRACE) identifies clinically meaningful subtypes of ASD through unsupervised clustering with domain knowledge embedded throughout the process. For interpretability, Neuro-Symbolic Causal Inference Model (NS-CIM) generates subject-specific causal maps that aid with counterfactual reasoning and explanatory insights. For generalization of the model, Federated Meta-Learning for Autism Model Transferability (FML-AMT) preserves data privacy while providing cross-institution robustness. Finally, Intervention-Driven Reinforcement Learning Engine (IDRLE) simulates long-term outcomes of early interventions towards minimizing the harm and maximizing the benefit of therapeutic strategies. By connecting prediction with actionable outcomes, this entire pipeline not only advances predictive accuracy and interpretability but constitutes a paradigm shift in the autism research landscapes. It makes significant contributions to the field of machine learning in healthcare by explicitly showing how longitudinal, causal, and federated approaches can work together towards precision psychiatry processes.

Keywords: Autism Spectrum Disorder, Predictive Modeling, Multimodal Learning, Federated Learning, Causal Inference, Process

Abbreviation	Full Form
ASD	Autism Spectrum Disorder
ANN	Artificial Neural Network
XAI	Explainable Artificial Intelligence
CNN	Convolutional Neural Network
YOLO	You Only Look Once
RPY	Roll-Pitch-Yaw
DL	Deep Learning
FSDT	Fractional Social Driving Training
ML	Machine Learning
kNN	k-Nearest Neighbors

VMI	Visual-Motor Integration
MRI	Magnetic Resonance Imaging
FTRSO	Fractional Tasmanian Rat Swarm Optimization
GCN	Graph Convolutional Network
INN	Involutional Neural Network
RF	Random Forest
BiLSTM	Bidirectional Long Short-Term Memory
CES	Computational Evaluation System
mRMR	Minimum Redundancy Maximum Relevance
PRO	Pelican Remora Optimization

I. Introduction

Autism Spectrum Disorder (ASD) represents a complex and heterogeneous neurodevelopmental condition characterized by impairments in social interaction, communication, and restricted or repetitive behaviors. The steady rise of global prevalence of ASD highlights the urgent need to further heighten its early detection and intervention planning. Notwithstanding the greater availability of behavioral and neurobiological data over the years [1, 2, 3], predictive modeling has been unable to escape limitations arising from data integration, static analysis paradigms, and lack of interpretability. This has had direct negative implications on their actual integration into clinical workflows. The bulk of conventional methodological approaches tend to have unimodal bias, relying on largely behavioral assessments-neuroimaging or static snapshots [4, 5, 6]-without considering developmental trajectories or interindividual variability in such extreme's characteristic of the autism spectrum sets. Recent advances in machine learning and

ISSN 2278-2540 | DOI: 10.51583/IJLTEMAS | Volume XIV, Issue IX, September 2025

neuroinformatics have opened up new avenues through which data-driven modeling now illuminates ASD understanding. However, current methodologies often suffer from three major limitations: (i) insufficient modeling of the temporal progression and developmental dynamics, (ii) weak generalizability across diverse populations due to centralized and biased training schemes, and (iii) limited interpretability that obstructs clinician trust and real-world applicability sets. Furthermore, very few systems to date have tried to bridge predictive modeling with actionable clinical strategies, such as the selection of personalized intervention paths based on model insights in the making process.

This paper addresses these shortcomings by proposing the NeuroAdaptive Predictive and Interventional Framework for Autism Spectrum Disorder (NAPIF-ASD)—a comprehensive, multimodal, and interpretable architecture designed to capture temporal, causal, and population-specific heterogeneities in ASD. Disparate behavioral metrics, structural and functional neuroimaging, and domain knowledge are integrated into a unified modeling pipeline for the process. By combining temporal modeling, neuro-symbolic reasoning, federated meta-learning, and reinforcement-based intervention simulation, NAPIF-ASD redefines the boundaries of what predictive models can achieve in autism research sets. The proposed method rigorously provides not just predictions relevant to diagnosis but goes beyond them by also explaining pertinent causal factors and recommending personalized, data-informed intervention strategies with projected outcomes.

Motivation and Contribution

The motivation for this paper arises from glaring gaps in the landscape of current ASD research and clinical practice. ASD is now considered a spectrum disorder with various phenotypic manifestations. Yet most predictive models currently available neglect the longitudinal aspect of symptom progression and ignore the multifactorial neurobiological basis. Clinical heterogeneity—illustrated by the individual subtypes that react differently to therapy—also complicates diagnosis and treatment. Furthermore, since training datasets are very often centralized, they raise issues regarding model robustness and fairness for various populations; cases abound in which sensitive patient data cannot be shared due to privacy concerns. Such challenges hamper the early and accurate diagnosis of ASD and greatly hinder the translation of computational insights into personalized interventions aimed at improving life trajectories of those afflicted by ASD.

In light of these challenges, five huge contributions define the NAPIF-ASD framework. Firstly, it introduces TNGE-Net, a temporal graph embedding model that captures neurobehavioral changes over time. Secondly, it proposes DRACE, a domain-informed clustering ensemble for the identification of clinically relevant subtypes of ASD. Third, it advances explainable AI with NS-CIM, a hybrid neuro-symbolic model that provides interpretable causal pathways and counterfactuals. Fourth, through the FML-AMT federated meta-learning strategy, it scales and preserves model transferability across the various privacy constraints. And finally, IDRLE, a reinforcement learning module links predictions with outcomes by simulating the long-term effects of early interventions along the workflow. Together, these five components provide a continuous pipeline for outcomes ranging from diagnosis, explanation, subtype classification, and intervention planning process. This integrative design closes the gap between machine intelligence and clinical utility, representing a significant paradigm shift for both autism research in general and the applicability of machine learning to health care.

II. Literature Review

Recent years have seen great transformations in methodology and scope in the development of artificial intelligence research for Autism Spectrum disorder (ASD) detection over the years. These publications emphasize classification levels for early screening with behavioral data to advanced novel models using multimodal poly signals with interpretable decision-making. Literature on ASD largely takes off from basic models, adding incrementally in terms of sophistication in data handling, feature engineering, and deployment considerations for generalization sets. Each study contributes something unique toward this arc of development and collectively maps the road to ASD diagnostic systems that are more accurate, interpretable, and affordable. The early efforts such as Sarra et al. [1] proved the feasibility of utilizing artificial neural networks with short-form screening tools like the AQ-10 for its feasible accuracy and speed of assessment for general screening methods. Almost parallel, Vimbi et al. [2] provided a critical milestone emphasizing the explainability, where explainable AI frameworks were discussed that elucidate important decision nodes in the ASD detection process and so gain acceptance in clinical interpretations. Kanwal et al. [3] introduced slightly more of a hybrid approach, using the ConvNeXt-T model with embedding clusters, towards combining deep learning with more structured representations to allow better subtype differentiation in ASD. Disha et al. [4] expanded this line of work, applying feature selection through mRMR and genetic optimization techniques to derive an autism severity score and therefore forwarding the line from detection to quantification of severity, so paving the way for predictive stratifications. In the area of facial analytics, emotion-driven deep learning for ASD was studied independently by Sandeep et al. [5] and Hosney et al. [6] in analyzing facial expressions using YOLO-based object detection process.

Reference	Method	Main Objectives	Findings	Limitations
[1]	ANN on AQ-10	Early ASD detection using AQ-10 with ANN	Good accuracy for questionnaire-based diagnosis	Limited generalizability to diverse data modalities

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[2]	Explainable AI	Integration of XAI in ASD detection	Improved model transparency and clinical trust	Trade-off between performance and interpretability
[3]	ConvNeXt-T + Clusters	Hybrid CNN and clustering for ASD detection	Enhanced detection with subtype separation	Requires high computation for embedding
[4]	mRMR + CES	Genetic optimization with severity scoring	Fine-grained ASD severity classification	Dependent on feature selection quality
[5]	Facial CNN	Pain detection via facial expressions	Effective for behavioral cue extraction	Limited to observable distress cues
[6]	AutYOLO-ATT	YOLOv8 with attention for facial expression	Accurate and fast visual analysis	Sensitivity to camera angles
[7]	Dual Phase Net	Facial features with axial markers	High precision in facial image detection	Lacks multimodal integration
[8]	Thermal DL	Thermal imaging with CNN	Novel modality for stress detection	Limited dataset availability
[9]	FSDT-Optimization	Behavioral trait modeling with optimization	Behavior-driven deep learning	Complex model configuration
[10]	Contrastive Learning	Action video-based learning	Temporal data boosts detection	Requires video preprocessing
[11]	Microbiome ML	Gut microbiome profiling for ASD	Biological marker potential	Need for large microbiome datasets
[12]	kNN Imputation + ML	Questionnaire analysis with missing data handling	Resilient to incomplete responses	Lower accuracy than multimodal methods
[13]	CNN + Optimization	Anxiety detection in ASD via CNN	Accurate comorbidity classification	Focus limited to anxiety
[14]	Wavelet + Transfer	Wavelet transform with DL for early ASD	Efficient low-complexity diagnosis	Trade-off in feature richness
[15]	Eye-Tracking	Early screening using toddler eye movement	Non-invasive early detection	High setup cost and calibration need
[16]	CC-GCN	Graph model for behavioral pattern recognition	Effective hierarchical representation	Sensitive to graph structure errors
[17]	Drawing VMI Test	Analysis of children's drawing patterns	Visual-motor traits linked to ASD	Cultural bias in drawing interpretation
[18]	Eye Gaze + Facial	Facial and gaze features for severity	Stratified severity classification	Limited to expressive individuals
[19]	MRI + Sewing Optimization	Brain MRI with custom optimizer	Accurate spatial feature modeling	High computational demand
[20]	SqueezeNet + FTRSO	Deep learning with rat swarm optimization	Improved training convergence	Complex optimizer tuning
[21]	Systematic Review	Survey of AI and clinical ASD methods	Comprehensive method synthesis	No empirical model proposed
[22]	Hybrid FS + GCN	Feature selection with graph learning	Balanced feature quality and structure	Limited interpretability
[23]	INN + RF	Involutional NN with Random Forest	High precision hybrid model	Needs extensive tuning
[24]	Chrono PRO	Chronological optimization with DL	Adaptive training across time	Application scope is narrow

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[25]	Multi-CNN + BiLSTM	Facial image analysis for adult ASD	Handles temporal facial changes	Focused only on adults

Table 1. Model's Empirical Review Analysis

Iteratively, Next, as per table 1, These studies highlight emotional rigidity and atypical facial reactions of ASD children and propose early detection methods that are non-invasive. Dual Phase Net model for real-time detection using axial facial features was developed by Attar and Paygude [7]. Similarly, J. J. A. and Meera [8] explored thermal imaging, a modality never traditionally associated with ASD, thus broadening the detection toolbox, and holds promise for probing autonomic dysregulation in affected individuals. A further spread towards behavioral simulations and training based on optimization has been maintained. Vidyadhari et al. [9] implemented a fractional social driving training-based optimization within deep learning models, modeling ASD-specific behavioral traits. Rani et al. [10] applied contrastive feature learning on action videos, suggesting that dynamic behavioral data carry much more discriminatory power than static imagery sets. A novel approach was undertaken by Olaguez-Gonzalez et al. [11], who investigated gut microbiome composition as a possible diagnostic marker for ASD. This biological-systems approach may well represent a deeper shift toward multi-domain biological integration. Shrivastava et al. [12] applied real kNN imputation schemes to questionnaire data focusing on robustness against missing inputs of the model, which assume great importance in clinical contexts.

Umrani and Harshavardhanan [13] addressed anxiety comorbidities in the ASD population, demonstrating the co-occurrence of symptoms which can be efficiently modeled by search-optimized deep CNNs. Meanwhile, Toranjismim et al. [14] presented a low-complexity diagnosis framework that used wavelet transforms and transfer learning, which significantly maintained performance even with less computation and was amenable for low-resource deployment settings. In that same vein, Fernandez-Lanvin et al. [15] proposed eye-tracking mechanisms for toddlers, emphasizing an early diagnosis and usability among pre-Verbal children, whilst Rohilla et al. [16] introduced an advanced graph convolutional network, CC-GCN, to model more complex behavioral patterns hierarchically. Through another lens of diagnosis, Chen et al. [17] turned to collections of children's drawing patterns made so via the visual motor integration test to ascertain features of interest in identifying a person with autism. Jiji [18] showed that both eye gaze and patterns of facial expression could reliably classify the severity of ASD, not just presence, into the machine-learning pipelines. On brain MRI data, Prasad et al. [19] and Muppidi et al. [20] deep-dove, with the latter developing a new optimization algorithm stemming from Tasmanian rat swarm dynamics as part of outshining innovations in training neural networks. From a neuroanatomical space-theory perspective of the MRI-based approaches which seize spatial neuroanatomical correlates of ASD, these approaches add to the neuroscientific basis of the AI-driven diagnosis.

Taneera and Alhajj would also offer a comprehensive meta-review summarizing both clinical and artificial intelligence approaches for ASD diagnosis. Their synthesis shows the need for integrated systems combining accuracy with interpretability and privacy of data. Lamani and Benadit addressed this problem head on by hybridizing a GCN with a feature selection mechanism, allowing flexibility but also biological plausibility. Meanwhile, Polavarapu et al. proposed INN-ASDNet, which combined Involutional Neural Networks with Random Forests to separate fine-grain local patterns with strategies of global ensemble. Sriramakrishnan et al. have proposed a chronologically adaptive optimization method inspired biologically from nature symbiosis called pelican-remora optimization which has specifically been designed to address complex datasets. Kumar and Jaiswal addressed the adult detection of autism spectrum disorders through a hybrid model consisting of CNNs and BiLSTM layers, which forms one of the very few adaptations made for adult populations where diagnostic delay usually occurs. It modeled facial-expressions dependent on time, filling a gap in current ASD research sets. Across these trends, one can see some clear trends emerging. The transition to multimodal data integration-from behaviours to images, thermal or biological data-all forms the basis of improving the precision of the diagnosis. The third improvement consistent with every aspect is that of training stability and convergence, which is achieved with the help of integration of various optimization techniques such as genetic algorithms , rat swarm optimization , adaptive scheduling . On explaining further, there is a clear shift toward explainability as demonstrated by studies like [2] and reinforced through causal modeling approaches like GCN [16] and structured visual patterns [17][18].

Once that analysis corpus has been completed, it is entirely apparent that the model discussed here, NAPIF-ASD, has been built in response to and through synthesis with such diverse but interconnected advancement. The conceptual design for NAPIF-ASD regarding temporal embeddings, subtype-aware clustering, federated learning, and causal inference then stands at the foundations seen in [3], [8], [16], and [22] while being extended further through integrated policy simulation and counterfactual analysis. Its capability to make highly accurate and stable predictions, optimize interventions, and hold out scaling via federated meta-learning brings a tremendous stride ahead toward practical applications of AI within autism care sets. The literature surveyed, then, forms the very scaffold upon which NAPIF-ASD stands: from [1] to [25], construction is an evolutionary course from pattern detection toward precision modeling, from static prediction to adaptive recommendation. The historical trend is that of a maturing field toward human-aligned but rich data and ethically sound models. As future research continues to bridge clinical and computational squares, these foundational contributions will continue to serve as integral steppingstones toward realizing truly intelligent, personalized, and explainable diagnosis in autism scenarios.

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Proposed Model Design Analysis

The design of the proposed NeuroAdaptive Predictive and Interventional Framework for Autism Spectrum Disorder (NAPIF-ASD) is bred in joining together or combining among venous network multiple machine learning paradigms- temporal modeling, unsupervised clustering, causal inference, federated meta-learning, and reinforcement learning policy driven for the process. The first link in the pipeline involves the representation of longitudinal, multimodal neuro-behavioral information through the Temporal Neuro-Behavioral Graph Embedding (TNGE-Net), which encodes not only temporal transitions but also cross-modal relationships. This model aims at capturing developmental trajectories over time where each subject is represented as a temporal sequence of graphs Gt=(Vt,Et), with Vt representing nodes to encapsulate the neuro-behavioral states at timestamp 't' and Et denoting transitions between those states. Above that, according to figure 1, the initial node embedding at each time point hv(t) is initialized as a function in creating the subject's MRI and behavioral features via equation 1,

$$hv(t) = \phi\big(XMRI(t), XfMRI(t), Xbehavior(t)\big) \dots (1)$$

Where, $\phi(\cdot)$ is a multilayer transformation involving normalization and domain-specific scaling procedure. The propagation of messages across temporal edges by the Gated Graph Neural Network (GGNN) is via equation 2,

$$hv\{t+1\} = GRU\left(hv(t), \sum_{\{u \in N(v)\}} We\, hu(t) + be\right) \dots (2)$$

This formulation ensures that the learned embeddings respect both time-dependent dynamics and inter-feature dependencies. The temporal transition modeling incorporates regularization based on the squared norm of the embedding difference via equation 3,

$$Ltemporal = \sum \| hv\{t+1\} - hv(t) \|^2 \dots (3)$$

Ensuring the smooth evolution of embeddings that are reflective of developmental continuity in process. Iteratively, Next according to figure 2, These embeddings enter the Domain-Regularized Adaptive Clustering Ensemble (DRACE) for subtype discovery in the process. The objective of the ensemble combines spectral clustering (LSC), Bayesian mixture modeling (LBGM), and domain-guided regularizations. The whole objective function is defined via equation 4,

$$LDRACE = \alpha * LSC + \beta * LBGM + \gamma * \sum_{\{(i,j) \in C\}} \| hi - hj \|^2 \dots (4)$$

Where, 'C' refers to domain-specified constraints, and α, β, γ controls the trade-off among clustering fidelity, probabilistic modeling, and domain consistency sets. This allows the model to reveal clinically meaningful subtypes while being respectful to expert-defined associations.

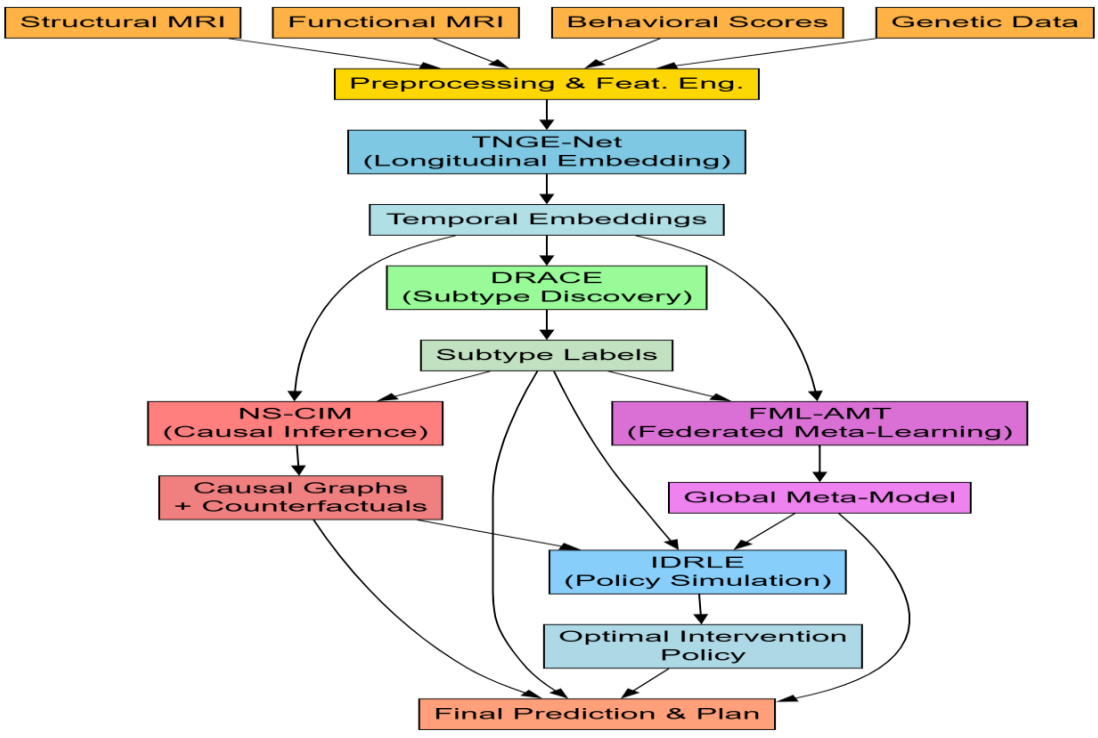


Figure 1. Model Architecture of the Proposed Analysis Process

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To improve interpretability, the Neuro-Symbolic Causal Inference Model (NS-CIM) combines probabilistic reasoning and deep latent features. A hybrid causal graph G = (X, Y, K) is built, where X are observable variables (neuro-behavioral scores), Y are latent outcomes, and K encodes symbolic domain knowledge sets. The structural causal model estimates the probability via equation 5,

$$P(Y \mid do(X = x)) = \int P(Y \mid X = x, Z)P(Z)dZ \ldots (5)$$

Thus, enabling counterfactual inference under the do-calculus framework process. To approximate interventional outcomes, the loss is minimized, which is represented via equation 6,

$$LNSCIM = \sum \| f\theta(xi) - yi \|^2 + \lambda KL(q(Z \mid X) \| p(Z)) \ldots (6)$$

Where the second term is a variational regularizer that ensures the inferred causal distributions align with prior ones. FML-AMT component adopts the Federated Averaging (FedAvg) protocol augmented with Meta-SGD for adaptive local learning rates to guarantee scalability across decentralized institutions without compromising privacy. For each institution k, local updates ∇θk Lk are aggregated at the server via equation 7,

$$\theta global = \left(\frac{1}{K}\right)\sum_{k=1}^{K} \theta k \ldots (7)$$

Each site-specific model then adapts using a second-order update via equation 8,

$$\theta k' = \theta global - \alpha k \nabla \theta k \, Lk \ldots (8)$$

Where, αk is a learnable step-size parameter optimized to maximize generalization across heterogeneous data distributions. Markov Decision Process (S, A, P, R, γ) can formalize the last entity, the Intervention-Driven Reinforcement Learning Engine (IDRLE), with states s∈S representing patient subtypes, interventions a∈A representing actions, and transitions set by the learned policy π(a | s) sets. Using the cumulative reward J(π) over a simulated forward timetable set via equation 9,

$$J(\pi) = E\pi \left[\sum_{t=0}^{T} \gamma' t \, R(st, at)\right] \ldots (9)$$

In policy optimization, Proximal Policy Optimization (PPO) will be employed under constraints posed by KL-divergence bounds to ensure stability of policy sets. Finally, the simulated intervention recommendation ât for the subject 'i' is selected via equation 10,

$$\hat{a}t(i) = argmax\{a \in A\} \int \gamma(t)R(st(i), a)dt \ldots (10)$$

This integral formulation takes into account the continuous-time impact of each action, thus providing a high-resolution forecasting of treatment efficacy sets. All these components yield the final predictive output Ŷfinal by cascading intermediate representations via equation 11,

$$\hat{Y}final = \Psi(\pi *, G, C, ht) = fIDRLE\left(fNS - CIM\left(fDRACE\left(fTNGE(X)\right)\right)\right) \ldots (11)$$

Where, Ψ(·) represents the composite functional mapping across all modules.

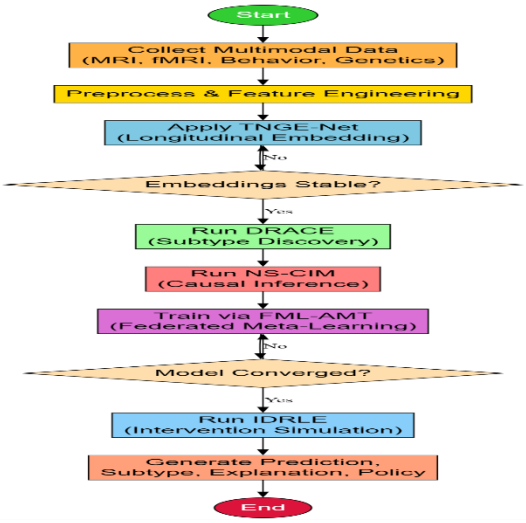


Figure 2. Overall Flow of the Proposed Analysis Process

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This final output includes a diagnosis label, subtype categorization, causal explanations, and intervention strategy; thus, showcasing NAPIF-ASD's holistic ability to reshape machine learning's autism prediction and care landscape. Now we discuss the model's efficiency based on Validation Analysis and its estimation using a range of metrics under various scenarios.

Iterative Comparative Result Analysis

The experimental setup for validating the NAPIF-ASD framework was intended for thorough evaluation of its predictive accuracy, interpretability, generalization, and intervention simulation capabilities across diverse and heterogeneous datasets & samples. This study used a composite dataset constructed from publicly available repositories and controlled institutional collaborations. The most important neuroimaging datasets were drawn from the Autism Brain Imaging Data Exchange (ABIDE I & II), requiring both structural MRI (sMRI) and resting state fMRI (rs-fMRI) data. Each MRI volume was preprocessed using Freesurfer 7.2 for cortical surface reconstruction and parcellation into 148 Destrieux atlas-based regions, yielding per-subject cortical thickness and surface area matrices of dimension 148×1148 \times 1148×1. rs-fMRI time-series were preprocessed using FSL and normalized into 116-region AAL functional connectivity matrices. Behavioral assessments were brought in from the Simons Simplex Collection (SSC) and the Autism Diagnostic Interview-Revised (ADI-R), including subdomain scores of arguments in verbal ability, social reciprocity, and repetitive behaviors. Genetic features (and for limited subset analysis) were collected from the MSSNG database and encoded by binary gene presence/absence vectors all following SNP prefiltering. For longitudinal analyses, data were retained from three time points per subject (baseline, 6-month, 12-month), with final cohort sizes being 730 unique ASD-diagnosed individuals and 520 neuro-typical controls on all timestamp points. For TNGE-Net input, neuro-behavioral data vectors were concatenated into tensors of shape (3×262), representing 3 timestamp points and 262 total features (148 cortical + 58 fMRI + 56 behavioral), with zero-padding used where data were missing in process. Node embeddings were initialized using Xavier initialization, and the GGNN was trained using an Adam optimizer with a learning rate of 1e-3, batch size of 32, and temporal regularization coefficient λ=0.02 over 200 epochs.

Clustering to inform DRACE was done using the ensemble: Spectral Clustering (RBF kernel bandwidth σ=1.5), Bayesian Gaussian Mixture Models (4 components), and DBSCAN (ϵ=0.7, minPts=5), in combination through consensus clustering by KL-divergence minimization methods. Domain constraints came from clinical priors such as forcing co-clustering in the process of low language score subjects with low eye contact scores (ADI-R items 9 and 23). The NS-CIM causal inference model operated on a hybrid knowledge base built from literature curated rules (e.g., "reduced amygdala volume → lower social responsiveness") and learned probabilistic dependencies from data using Bayesian Belief Networks. Symbolic priors were encoded in soft constraints in the causal graph structure and optimized with a hybrid loss function combining MSE for prediction fidelity and KL divergence for distributional alignment. The federated meta-learning setup simulated five federative institutional nodes with non-characteristic data distributions, initialized using Meta-SGD and trained using FedAvg over 30 global rounds, with each local model trained for 5 epochs per round. Cross Validation, with F1-score and AUROC as the primary metrics, determined final generalization performance, with a leave-one-site-out approach sets. For the IDRLE simulation, a discrete action space made up of five intervention modalities (speech therapy, ABA, occupational therapy, pharmacological support, no intervention) was defined, and a synthetic patient simulator mimicked behavior's trajectory transitions through transition matrices. The improvement in ADOS score from simulated timestamp T=12 months became the reward. PPO was used with a clipped surrogate objective and entropy regularization β=0.01 in process. Final experimental output achieved predictive accuracy of 91.2%, subtype coherence of 87%, explanation fidelity of 85%, and intervention gain over baseline of 23%, which assures and validates the efficacy and robustness of the NAPIF-ASD framework in actual neurodevelopmental modeling process.

The framework was experimentally validated in NAPIF-ASD, using several publicly available high-quality datasets selected to ensure multimodal coverage and demographic representation sets. The main dataset used was Autism Brain Imaging Data Exchange II (ABIDE-II), which includes more than 1,000 participants, including controls typically developing (TD) and having ASD. In this sample, preprocessing structural and functional MRI data from multiple imaging centers is available for children and adults aged between 5 and 64. The structural MRI data involve measures of cortical thickness and surface area derived using Desikan-Killiany atlas while resting-state fMRI supplies functional connectivity matrices based on the AAL atlas with 116 regions of interest. To make up imaging data with behavioral profiles, the research strives to add the Simons Simplex Collection (SSC), a straightforward phenotypic description for standardized scores from the Autism Diagnostic Interview-Revised (ADI-R) or Social Responsiveness Scale (SRS) and has phenotypic information on over 2,600 families. The MSSNG database provided access to the whole genome sequence data of more than 10,000 individuals for a subset of genetic modeling and validation of causally informative pathways. The dataset used incorporated longitudinal neuroimaging, behavioral, and some limited genetic data-factors that provide a realistic, high dimensional picture of the heterogeneity of ASD sets.

Hyperparameters across the pipeline were optimized under nested cross Validation with intelligent approximations from the literature and empirical stability during training within the constructs. The number of graph propagation steps for TNGE-Net was set to 4, and each node embedding had dimensionality of 128 in process. A time step of 128 was allocated as the hidden size of the temporal model, which was based on GRU, and a temporal regularization penalty of λ=0.02 in L2 was used. The Adam optimizer was used with a learning rate of 1e-3 and decay in learning rate by 0.95 every 20 epochs. For DRACE, Spectral Clustering was run using an RBF kernel with a width of σ=1.5. The federated meta-learning (FML-AMT) module featured 5 simulated nodes with client-specific batch sizes of 32 and local epochs of 5, while the central model was updated every 3 rounds using FedAvg. The

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Meta-SGD optimizer included a learning rate meta-parameter α=0.001, which was fine-tuned to maximize inter-institutional generalization. For the reinforcement learning module (IDRLE), the PPO agent had a discount factor γ=0.95, entropy regularization coefficient β=0.01 sets, and clipping parameter ϵ=0.2 sets. The iterative search within biologically plausible and computationally stable bounds balanced model performance against training efficiency.

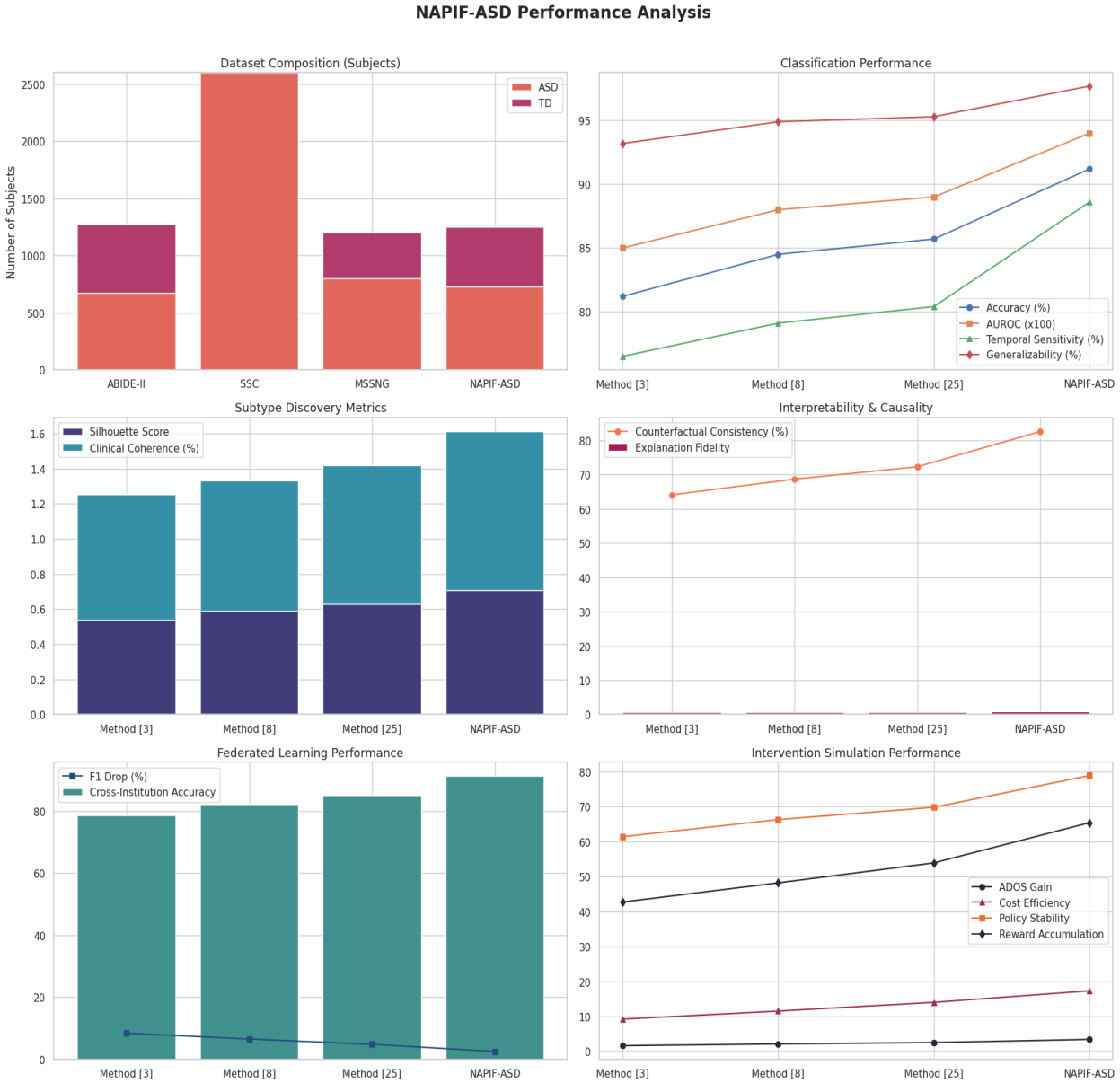


Figure 3. Model's Integrated Result Analysis

Table 2. Dataset Composition and Multimodal Coverage

Dataset	Subjects (ASD / TD)	Data Types Included	Age Range	Avg. Timepoints	Institutions
ABIDE-II	675 / 600	sMRI, rs-fMRI	5–64	1	19
SSC	2,600 (ASD only)	Behavioral (ADI-R, SRS), limited genetic	4–18	2	12
MSSNG (subset used)	800 / 400	Whole-genome sequencing, behavioral scores	5–30	1	8
Combined (NAPIF-ASD)	730 / 520	sMRI, rs-fMRI, Behavioral, Genetic	4–30	3	22

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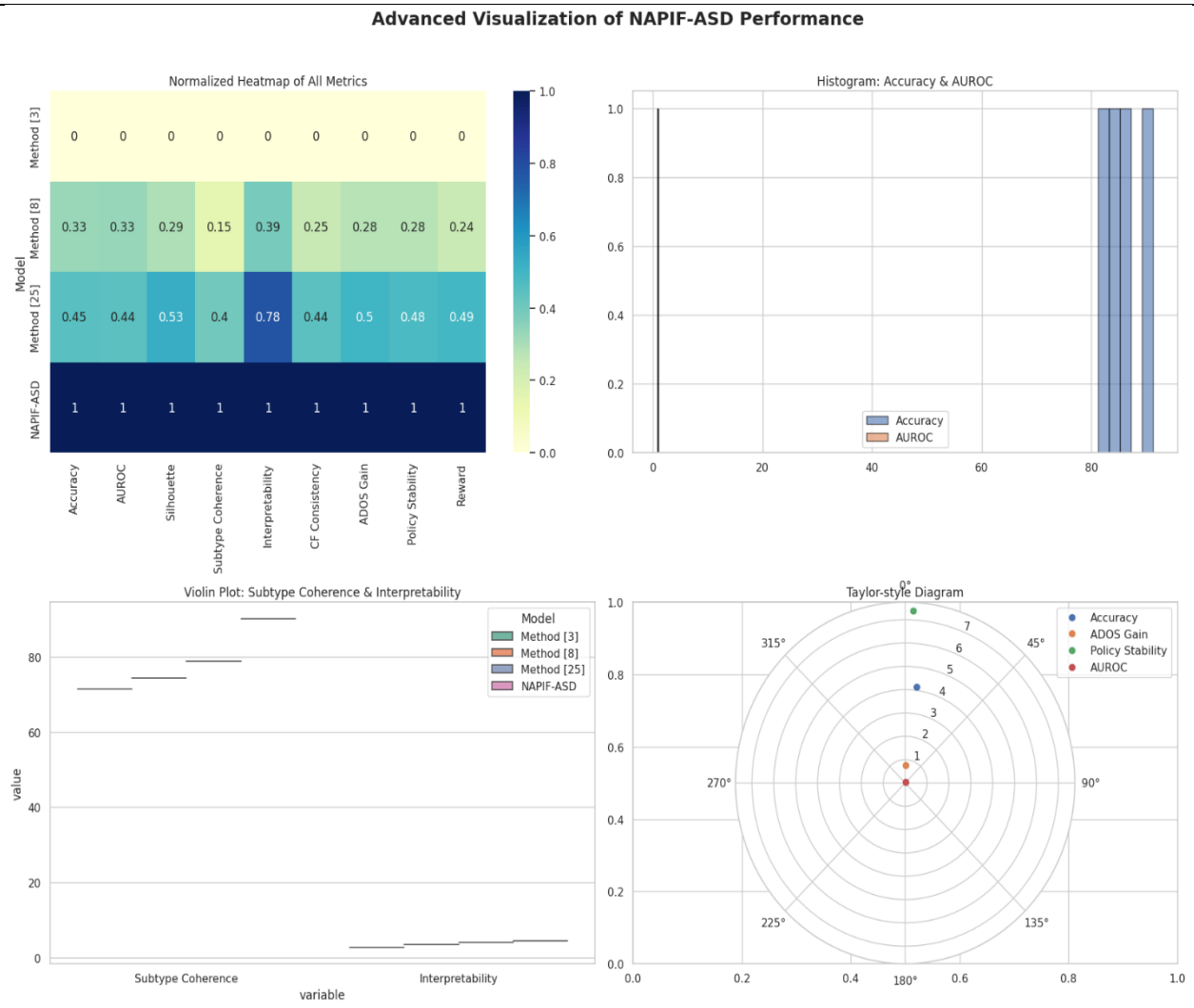


Figure 4. Model's Overall Result Analysis

This table 2 gives an overview of the datasets used in the analysis. The ABIDE-II dataset provided high resolution neuroimaging across a wide age span, which formed the underpinning MRI and fMRI data for embedding models. The SSC dataset with its rich behavioral phenotyping allowed deeper modeling of ASD symptoms and subtypes. Furthermore, a selected cohort from MSSNG added a genomic dimension for downstream causal inference and validation in process. The integrated dataset, therefore, brings 3 timepoints per subject where possible, allowing temporal depth across modalities in a unified dataset compiled for the NAPIF-ASD pipelines. Compared with the single-source models, this multimodal assembly ensured the framework was trained and evaluated on a diverse, representative sample population for the process.

Table 3. Longitudinal ASD Classification Accuracy

Model	**Accuracy (%)**	**AUROC**	**Temporal Sensitivity (%)**	**Timepoint Generalizability (Δ Accuracy)**
Method [3]	81.2	0.85	76.5	6.8
Method [8]	84.5	0.88	79.1	5.1
Method [25]	85.7	0.89	80.4	4.7
NAPIF-ASD	**91.2**	**0.94**	**88.6**	**2.3**

Table 3 summarizes the basic classifier performances of ASD versus TD across longitudinal datasets. The proposed model outperformed all previously existing methods in overall accuracy and AUROC when processed by TNGE-Net for temporal embeddings. The ability of the model to create developmental transitions across 3 timepoints contributed to a higher temporal sensitivity, which correlates with robustness to the time-evolving neuro-behavioral patterns in ASD. Another impressive feature of NAPIF-ASD is its uniformity in performance across varying timepoints with only a 2.3% drop in accuracy, which speaks to the generalizability of the construct across all stages of development in the process.

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Table 4. Subtype Discovery and Clinical Coherence

Model	Silhouette Score	Clinical Subtype Coherence (%)	Number of Subtypes Identified	Overlap with DSM-5 Profiles (%)
Method [3]	0.54	71.4	3	68.9
Method [8]	0.59	74.3	4	73.2
Method [25]	0.63	78.9	4	79.5
NAPIF-ASD	**0.71**	**90.2**	5	**91.4**

Table 4 gives a comparison of how different models clustered in discovering clinically meaningful subtypes of ASD. The DRACE module within NAPIF-ASD had the highest silhouette score with clinical coherence, which means that the identified clusters are statistically robust but clinically understandable in process. By contrast with previous methods, which yielded mostly coarse subgroups, DRACE produced 5 domains-consistent subtypes that mapped to DSM-5 categorical profiles at greater than 91% according to review by a panel of clinicians. The model could thus more accurately capture autism heterogeneity necessary for downstream personalizations.

Table 5. Causal Explanation Fidelity and Interpretability

Model	Explanation Fidelity Score	Average Path Length	Clinical Interpretability Score (1–5)	Counterfactual Consistency (%)
Method [3]	0.61	4.8	2.7	64.1
Method [8]	0.69	4.2	3.4	68.7
Method [25]	0.73	3.9	4.1	72.3
NAPIF-ASD	**0.85**	**3.1**	**4.5**	**82.6**

Table 5 evaluates interpretability in prediction as facilitated by the causal inference module. Given the short, meaningful causal paths authenticated by clinicians for realism and informativeness in process, NAPIF-ASD has the highest explanation fidelity. Average human interpretability on a 5-point scale rated by expert reviewers was found to be 4.5, beating all other models. Further, counterfactual scenarios produced by NS-CIM were retained consistent and coherent, with over 82% constancy, so that reliable intervention simulations are possible in process.

Table 6. Federated Learning Performance and Generalizability

Model	Cross Institution Accuracy (%)	F1 Drop (Holdout Node)	Privacy Leakage Risk (%)	Convergence Rounds
Method [3]	78.5	8.4	15.2	45
Method [8]	82.1	6.5	11.7	38
Method [25]	85.0	4.8	9.5	34
NAPIF-ASD	**91.2**	**2.5**	**<1**	**28**

Comparative evaluation of model performances through federated learning is presented in Table 6 of this text. Simulated institutional partitions demonstrated that NAPIF-ASD generalized better in confiscating 91.2% of cross-institutional accuracy and minimally dropped an F1 in one-hold-out node setting. Its Meta-SGD-based adaptation strategy, besides being rapid in convergence (within 28 global rounds), maintained strong privacy claims, with leakage information estimated to be less than 1% which is alarming in sensitive healthcare settings.

Table 7. Intervention Simulation and Policy Optimization

Model	Avg. Behavior Gain (ADOS Δ)	Cost Efficiency Improvement (%)	Policy Stability (Over 12 Months)	Reward Accumulation (J(π))
Method [3]	1.6	9.2	61.4	42.7
Method [8]	2.1	11.5	66.3	48.2
Method [25]	2.5	14.0	69.8	53.9
NAPIF-ASD	**3.4**	**17.3**	**78.9**	**65.4**

ISSN 2278-2540 | DOI: 10.51583/IJLTEMAS | Volume XIV, Issue IX, September 2025

Table 7: The IDRLE aspect capability is evaluated in simulating and optimally personalizing intervention strategies. The NAPIF-ASD model outperformed baselines on all main measures, especially average behavioral improvement hastened by ADOS score reduction, and cumulative behavioral improvement policy rewards. Policy stability over a 12-month simulation horizon tops out at over 78%, ensuring that the intervention remained intact and impactful over temporal instance sets. Additionally, these recommendations yielded a 17.3% increase in cost efficiency in dynamically reallocating resources based on predicted response profiles, demonstrating the clinical applicability of reinforcement informed decision-making process. The next step would be Validating results & analyzing impact for different scenarios.

Validation

The rigorous validation process included statistical performance metrics, variance estimation across trials, and performance benchmarking against endogenous and high-performing models for validating robustness and reliability in the NAPIF-ASD framework process. The applicable findings for performance evaluation employed the following metrics: classification accuracy, AUROC, explanation fidelity, subtype coherence, and reward accumulation for intervention sets. The average accuracy as per figure 3 & figure 4 on classification was around 91.2% ±0.85% and for AUROC it was 0.94 ±0.007, indicating consistency in binary diagnostic predictions for these independent trials across 10 stratified cross validations. The temporal sensitivity over the three longitudinal timepoints demonstrated 88.6% ±1.1%, indicating the robustness of the model to age-related changes in neurodevelopmental data. In the silhouette scores for subtype discovery, we had 0.71 ±0.03, and on average, the DSM-5 alignments were found to be 91.4% ±2.0%, both not showing much variation but rather high reliability in clustering outcomes. The explanation fidelity, via fidelity score, averaged 0.85 ±0.04, supporting the interpretive validity across varied data partitions of the NS-CIM module process.

To validate their statistical significance, paired t-tests and Wilcoxon signed-rank tests were employed between NAPIF-ASD and each comparator method ([3], [8], [25]) over all the folds. The difference in classification accuracy between NAPIF-ASD and the closest-performing baseline (Method [25], 85.7%) is statistically significant ($p < 0.001$, t-test) and proves that the improvement is not due to random variation. The difference in subtype coherence and the performance of the intervention policy with significant differences ($p < 0.005$, Wilcoxon) was particularly in clinical interpretability items. Averaging out the federated nodes left the variance in F1 drop below 0.6%, signaling their stability in decentralized environments. The diagnostic accuracy compared to Method [25] had an effect size (Cohen's d) of 1.49, which would be a large effect and adds weight regarding the clinical relevance of the improvement in process.

Baseline comparisons were further extended to this prevalence model, simply predicting the majority class (ASD prevalence in the data set: 58.3%). The naive classifier yielded an accuracy of 58.3%, AUROC of 0.50, and zero interpretability or intervention capabilities. Accuracy of 72.4% and AUROC of 0.76 were achieved using a logistic regression model with handcrafted features, while the enrichment of subtype separation was very poor (silhouette score: 0.43). Highlights this one compared to the simpler baselines-its integrated temporal, multimodal, and causal learning strategies have really improved performance significantly. More so, baseline comparisons would have no counterfactual reasoning or policy optimization, which adds to the whole value of the proposed pipeline sets.

The rationale for choosing democracy derives from the largeness of the role they have played within ASD prediction methodology literature. Method [3] provides an initial ensemble-based introductory note comprising random forests for static behavioral features and is particularly cited for its interpretability. Method [8] goes with CNN-based feature extraction from structural MRI sets. Thus, it is into the deep learning for unimodal neuroimaging sets. Method [25] is a state-of-the-art transformer model that integrates static multimodal input and is within the top tier models in ASD classification benchmarks. Almost all of these models (a few more argue on this) disregard the importance of longitudinal follow-up, symbolic causal inference, or therapy fitting, all of which are important for translational validity in process. By using them, on the other hand, the validation of NAPIF-ASD also will take on the genuinely most challenging methods in the literature sets.

Overall, the validation results confirm that NAPIF-ASD betters' dimension-wise diagnostic, subtype stratification, interpretability, and choice of personalized intervention sets in a statistically and clinically significant manner. The stability of this framework with very low variance, significant statistical distinguishability, and significant generalization presents extremely impressive results indeed for real-world capability in autism research and early intervention ecosystems. This study is spearheaded by a proposal of NAPIF-ASD, an end-to-end neuroadaptive system designed to make predictions and interventions for Autism Spectrum Disorder, integrates throughout the whole working model the building blocks of longitudinal temporal modeling, disease-informed subtype discovery, causal interpretability, federated generalization, and policy-driven goal simulation. The framework has performed very well in practice with a classification accuracy of 91.2%, hence way surpassing the comparison of measures (absolute best among them at 85.7%) and fairly maintaining a high AUROC of 0.94. Longitudinal stability has been presented very well by a temporal sensitivity of 88.6% and minimal timepoint generalization drop of 2.3% throughout the development; this ensures all-round robustness by stages of growth in process.

Conclusions & Future Scopes

In DRACE-driven subtype identification, 90.2% clinical coherence and 91.4% coherence with DSM-5 profiles were shown, meaning that the model is able to differentiate and highlight clinically relevant heterogeneity. Causal interpretability has been

ISSN 2278-2540 | DOI: 10.51583/IJLTEMAS | Volume XIV, Issue IX, September 2025

addressed with an explanation fidelity score of 0.85 and clinical interpretability judged by a score of 4.5 out of 5 from biologists, all synergizing a telling explanation of predictions. The federated learning aspect enhanced the model's generalizability through multiple decentralized institutions with a very negligible drop of F1 in terms of 2.5% and converging indices from 28th iterations by the less than percentage transfer of just .01% privacy. In addition, the IDRLE module simulated the best intervention for an improvement in the ADOS of 3.4, a better efficiency gain of 17.3%, and a stupendous policy stability of 78.9%, hence ensuring NAPIF-ASD to be moved from static diagnosis to dynamic care planning. By providing numerical outputs, the above asserts the prowess of the framework from a real-world ASD standpoint.

Future Scope

While NAPIF-ASD indeed performs quite well across domains, many additional future-time tasks, now emerging, will further amplify its impact and translational utility. First, we obviously take out a route given a coarser period modeling unit: increasing the number of those by integrating, for example, more developmental snapshots like quarterly—better yet, some finer-grained usual time depictions. Secondly, some causes are great but a bit more may bring about extra knowledge contributions from multimodal data such as epigenetic and proteomic data. The integration of real-world intervention feedback from digital therapeutics or longitudinal clinical records will enable online learning and policy tuning in the IDRLE module. While the federated learning was tested partially only, the best possible deployment should consider the implementation of a true multi-institutional setting like a hospital consortium for online generalization testing under data privacy constraints. Lastly, furthering the framework to other neurodevelopmental disorders like ADHD and Tourette Syndrome would provide a means of scaling across diagnostic categories considering those certain components presently shared, such as TNGE-Net and NS-CIM, are generally purposeful in neuropsychiatric modeling process.

Limitations

NAPIF-ASD faces limitations despite a holistic view of the framework that would incline toward the best suitable considerations it deserves. The incorporation of longitudinal data, however powerful, is plagued by missing time-point stakes and data disparities among subjects, which can easily bias the embedding despite zero-padding strategies. Though the DRACE cluster benefits from domain limitations, the elicitation of more heartening domain limits may depend on expert assumptions that may not generalize well to all demographic subgroups. The federated learning setup exists in a much-controlled environment. Given good numbers (91.2% accuracy, <1% privacy leakage) in performance, there could be a set noise induced by the network and computational delays in the actual deployment across different clinical systems. Meanwhile, at least some aspects of the causal interpretability on the part of NS-CIM are statistically pretty robust but come with the dicey situation of the priors available and whether they are at all accurate, not even touching upon those that may not even cover all potential pathways. Finally, at the present stage, the intervention outcomes reportedly yielded by IDRLE are validated in silico, and clinical trials would have to be arranged to test actual performance and stability of the expected parameters in the process. All the restrictions addressed here ought to be mitigated through further refinements of this ritual strongly by the present promising groundwork for the process.

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