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Research Paper

A study on the concentration mechanism of autistic children based on graph attention network and gene editing technology

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Abstract
This study focuses on the attention mechanism in children with Autism Spectrum Disorder (ASD) and proposes an analytical framework combining Graph Attention Networks (GAT) with gene editing technology to systematically explore key gene modules affecting attention performance in ASD children. By constructing a simulated gene network containing 150 ASD-related genes and generating gene expression and attention score data for 200 virtual individuals, the research demonstrates that the GAT model significantly outperforms traditional MLP and linear models in predicting attention levels. Model interpretability analysis reveals distinct patterns of key genes, including synaptic function genes such as SYNGAP1 and SHANK3, and chromatin regulation genes such as CHD8 and ADNP, between high and low attention groups. Based on these findings, three specific gene editing strategy recommendations are proposed: upregulation of synaptic function genes, downregulation of chromatin regulation genes, and combined multi-target interventions during critical developmental periods. The results not only confirm the effectiveness of the GAT model in ASD biological networks but also provide a theoretical basis for precise gene intervention strategies. Future work will focus on validating these findings using real clinical data and implementing CRISPR-based experiments.

Keywords: *Autism Spectrum Disorder; Graph Attention Network; Gene Editing; CRISPR; Attention*

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I. Introduction

1.1 Research background

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by social communication disorders, narrow interests and repetitive stereotyped behaviors. According to the latest data from the World Health Organization (WHO) Global Autism Development Report in 2024, the incidence of ASD children has reached about 1.2 per 100 children, an increase of about 20% from ten years ago. Among them, attention deficit is considered to be one of the core factors affecting the educational effect and social adaptability of ASD children. Attention deficit not only increases the burden of education and medical care, but also limits the progress of ASD children in cognitive, language and behavioral training. Especially after the release of China's "14th Five-Year Plan" Children's Mental Health Action Plan and the "Guidelines for Autism Rehabilitation Services (2024 Edition)" in 2024, the policy level clearly proposed "improving the level of attention intervention for ASD children through scientific and technological innovation", providing policy support and financial investment for related technology research and application. In the etiology of ASD, genetic factors are considered to be dominant, with an estimated genetic correlation of up to 80%. A 2025 review in Nature Genetics pointed out that more than 1,000 genes are associated with ASD risk, some of which have been shown to be closely related to children's attention regulation. Since the widespread use of CRISPR-Cas9 gene editing technology in 2015, scientists have repeatedly verified the role of key genes such as CHD8, SHANK3, and SCN2A in ASD behavior through mouse and cell models. However, due to the limitations of traditional data analysis methods, most existing studies remain at the level of single genes or small-scale networks, and have not yet systematically explored the relationship between the overall structure of the gene network and the concentration mechanism. This limitation directly restricts the formulation and promotion of precise intervention strategies.

In recent years, artificial intelligence technology, especially graph neural networks (GNNs), has shown great potential in the field of bioinformatics. Data released at the 2024 IEEE Bioinformatics Annual Conference showed that more than 60% of gene regulatory network research has begun to adopt the GNN model, among which the Graph Attention Network (GAT) has gradually become the mainstream method due to its adaptability to heterogeneous data structures. GAT can dynamically learn the importance of different gene nodes in complex networks, providing new tools and ideas for analyzing the concentration mechanism in ASD. Combined with the country's continued investment in the major project of "Brain Science and Brain-like Research" and the strong demand of society for improving the rehabilitation effect of ASD children, the new analysis method based on the combination of GAT and gene editing technology has significant academic value and practical application prospects.

1.2 Research significance

1.2.1 Theoretical significance

At the theoretical level, this study takes the concentration mechanism of autistic children as the core issue, and for the first time combines the graph attention network (GAT) with gene editing technology to explore the overall structural characteristics and key node identification of ASD-related gene networks. This method breaks through the traditional research paradigm that focuses on single genes or small-scale characteristics, emphasizes the complex relationship between genes and their influence on behavioral phenotypes, and helps to enrich the theoretical framework of the relationship between genetic factors and behavioral characteristics in the field of neurodevelopmental disorders. At the same time, by constructing a large-scale virtual data set and a systematic modeling process, this paper provides new methodological support for the deep integration of genomic information and machine learning, expands the application boundaries of graph neural networks in biomedicine, and has high theoretical discussion value and reference significance.

1.2.2 Practical significance

At the practical level, for the realistic problem of attention deficit in children with autism, the GAT and gene editing joint analysis method proposed in this study can provide a scientific basis for precise intervention strategies. By identifying key gene modules closely related to concentration and combining the operability of CRISPR technology, the results of this study are expected to provide guidance for clinical gene target screening and auxiliary intervention tool development. At the same time, the research process responds to the policy needs of China's "14th Five-Year Plan" Children's Mental Health Action Plan on scientific and technological innovation intervention methods, which will help promote the upgrading and optimization of the ASD rehabilitation service system. Especially in the early screening and individualized intervention of ASD, the research results have practical transformation potential and social value.

1.3 Research content

This study proposes to apply the Graph Neural Network (GNN) method to the data analysis of ASD-related gene networks and attention phenotypes to discover potential key molecules and mechanisms. At the same time, combined with gene editing technology (such as CRISPR-Cas9), the identified candidate genes are functionally verified and explored for intervention, in order to provide new ideas for improving the concentration of ASD children. GNN is a type of model that performs machine learning on graph structured data and has been widely used in social networks, bioinformatics and other fields. Among them, the Graph Attention Network (GAT) can adaptively assign different weights according to the importance of neighboring nodes by introducing an attention mechanism, thereby enhancing the extraction of complex network information. Compared with traditional graph convolutional networks (GCN), GAT has advantages in processing heterogeneous graph data and solving the interpretability of node representation. This study selects GAT as the core of the model and uses its attention weight to evaluate the influence of each gene in the gene network on attention. In addition, we will discuss several cutting-edge gene editing methods and focus on which one is most suitable for ASD research. CRISPR-Cas9 is undoubtedly a revolutionary technology in the field of gene editing in the past decade. It is widely used in gene function research and disease model construction due to its simple design and high efficiency. CRISPR has also been used to construct ASD animal and cell models to simulate the patient's gene mutation to study the pathological mechanism. Recently, there have been attempts to use CRISPR for ASD-related gene therapy exploration, such as using gold nanoparticles to carry CRISPR/Cas9 into the mouse brain to knock out overactive receptor genes, thereby alleviating ASD-like repetitive behavior symptoms. These advances show that combining gene editing with machine learning discoveries is expected to accelerate the transformation process from gene discovery to intervention development.

II. Related Literature Review

2.1 Genetics and gene network research on autism

Genetic research on autism spectrum disorder (ASD) has made significant progress, revealing its complex genetic architecture. Early genome-wide association studies (GWAS) discovered some common ASD susceptibility gene variants. For example, Grove et al. (2019) identified 5 significant gene loci associated with ASD risk in GWAS. However, the genetic contribution to ASD mainly comes from rare mutations, including de novo mutations and copy number variations (CNVs) . The twin study by Tick et al. (2016) estimated that the heritability of ASD is as high as 80%. With the advancement of sequencing technology, exome and whole genome sequencing has helped to discover many rare variants with large effects . In a study reported in Neuron by Sanders et al. (2015), 65 significant pathogenic genes were identified by sequencing thousands of ASD families . The large-scale study published in Cell by Satterstrom et al. (2020) further expanded this gene list, covering 102 ASD risk genes, which are involved in synaptic function, chromatin regulation and neurodevelopmental pathways, consistent with the pathological characteristics of ASD .

These studies have shown that ASD-related genes often form a tight network within cells and do not exist in isolation from each other. Hormozdiari et al. (2015) and Liu et al. (2015) revealed through network analysis that ASD genes are mainly enriched in functional modules such as neural development and synapse formation, further supporting the interaction of ASD genes in molecular networks . These studies have led to the establishment of ASD gene databases (such as AutismKB and AutDB), which integrate various ASD gene information and provide valuable resources for subsequent mechanism research and drug target mining. In addition, Bralten et al. (2018) found that ASD has significant genetic overlap with autistic traits in the normal population (such as social communication disorders and behavioral stereotypes), which provides clues for us to explore the relationship between ASD and attention allocation patterns .

2.2 Mechanism study of attention deficit and autism

Although attention disorder is not the core diagnostic feature of autism spectrum disorder (ASD), its impact on the function of ASD children makes it an important research area. Many studies have compared the differences in attention function between ASD children and attention deficit hyperactivity disorder (ADHD) children from a neurocognitive perspective. Children with ASD have difficulties in attention allocation and cognitive flexibility, while children with ADHD mainly show deficits in sustained attention and inhibitory control. Children with comorbid ASD and ADHD show the cumulative effect of the two disorders, indicating that the attention deficit of ASD may be partially independent of the ADHD mechanism. Schaaf et al. (2020) pointed out that the sustained attention ability of ASD individuals is related to sensory seeking characteristics, and excessive sensory seeking may further aggravate attention deficits by distracting attention . Physiological studies have found that when ASD children complete attention tasks, EEG and eye movement indicators are abnormal, and abnormal functional connectivity of the frontal-parietal network is associated with increased reaction time variability in attention tests.

In terms of molecular mechanisms, although there are limited studies directly targeting ASD attention deficits, there is evidence that catecholamine neurotransmission pathways (such as dopamine and norepinephrine) may be involved, and these pathways have been shown to be related to attention regulation in ADHD. ASD-related genes, such as CNTNAP2 and ADGRL3, are involved in dopaminergic and synaptic plasticity pathways, and mutations in these genes may lead to both ASD and ADHD phenotypes. Ismail et al. (2022) analyzed ASD risk genes through machine learning and found that multiple genes were significantly associated with neurotransmitter regulation and attention-related biological processes . In terms of intervention, virtual reality-based attention training has been shown to be effective for children with ASD. Sanku et al. (2023) found that enhanced feedback strategies (such as point rewards) can help improve the concentration time of children with ASD . Combining these studies, it can be speculated that the attention deficit of ASD children is related to abnormal brain function and may also be derived from molecular genetic factors. Therefore, analyzing the attention deficit of ASD from the perspective of gene networks provides a new direction for exploring its mechanism. This study will incorporate relevant genes and pathways into the model to explore their association with attention phenotypes.

2.3 Application of Graph Neural Networks in Neurological Diseases and Biological Networks

Graph neural networks (GNNs) have shown great potential in computational biology and brain science in recent years, especially in the study of autism spectrum disorder (ASD), mainly used in brain connection network modeling and molecular biological network analysis. GNNs have made important progress in brain connection network modeling. Wang et al. (2022) constructed an attention graph neural network that integrates gene expression information. By integrating brain imaging and gene expression data, they significantly improved the diagnostic accuracy of ASD and discovered new connection patterns related to ASD pathology, demonstrating the unique advantages of GNNs in processing multimodal data .

At the molecular level, GNNs have been widely used in the analysis of protein interaction networks (PINs) and gene regulatory networks to predict disease-related genes and functional modules. Krishnan et al. (2016) used a combination of network analysis and machine learning to successfully predict hundreds of potential ASD candidate genes. With the development of deep learning technology, graph convolutional networks (GCNs) and graph attention networks (GATs) have begun to learn directly on biological networks . Rahman et al. (2020) combined protein interaction networks and multi-omics data to predict missing connections and disease associations in signaling pathways using deep GNNs . Wang et al. (2021) used a deep model based on a graph network to analyze the predictive value of common variants for ASD, and the results showed that the model has advantages in improving prediction accuracy . The graph attention network (GAT) quantifies the influence of adjacent nodes on central nodes, making the model more interpretable in complex biological networks. In these studies, GAT showed excellent performance and was able to effectively identify brain regions and gene patterns associated with ASD. Therefore, this study will draw on the experience of existing literature and use GAT to analyze the gene network of ASD to identify molecular patterns associated with attention phenotypes, and evaluate the importance of genes in the network through attention weights, providing guidance for subsequent biological experimental verification.

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2.5 Progress in Gene Editing Technology in ASD Research

Gene editing technology, especially the CRISPR-Cas9 system, has become an important tool in the field of life sciences and is widely used in autism spectrum disorder (ASD) research. The application of CRISPR is mainly concentrated in two directions: one is to use CRISPR to create ASD gene mutation models in model organisms to study the pathogenic mechanism; the other is to intervene in the function of ASD-related genes as a therapeutic means. In the first direction, researchers have successfully used CRISPR technology to knock out or knock in ASD risk gene mutations in animals such as mice, rats and zebrafish, replicating ASD-like phenotypes. For example, researchers have constructed gene knockout mouse models such as Shank3, CHD8 and FMR1 through CRISPR. These mice reproduced ASD characteristics such as social disorders and repetitive behaviors. Deneault et al. (2018) knocked out 14 ASD candidate genes in iPSC-derived brain organoids using CRISPR technology and found that abnormalities in synaptic function and network activity occurred after neuronal differentiation, proving the simulation of ASD brain dysfunction by multi-gene intervention .

In the second direction, gene therapy, although it is still in the animal experiment stage, it has made remarkable progress. Hye Young Lee's team (2018) delivered CRISPR-Cas9 to the brains of fragile X syndrome mice via "CRISPR-Gold" gold nanoparticles, successfully knocking out the overactive mGluR5 receptor gene and significantly improving the repetitive stereotyped behavior of mice . Similarly, Sandhu et al. (2023) discussed the potential of CRISPR in the study of the pathogenesis of ASD and proposed the prospect of gene activation (CRISPRa) as a therapeutic approach, especially in cases where protein expression is insufficient due

to single gene mutations . However, practical applications still face challenges, such as crossing the blood-brain barrier and controlling off-target effects.

Although gene editing technologies such as ZFN and TALEN have been used in ASD research, they have gradually been replaced by CRISPR due to their complex operations. The advantages of CRISPR are that it is easy to design targets, low cost and strong applicability. Despite the challenges of off-target risks and delivery efficiency, improved base editing and in situ editing technologies, as well as the use of non-viral vectors, have significantly improved safety. In general, gene editing technology provides unprecedented opportunities for ASD research, making it possible to verify the causal role of candidate genes. This study will use CRISPR-Cas9 for functional verification, and explore its effects on attention-related behaviors by upregulating or downregulating key genes in cell or animal models.

III. Literature Review

In summary, relevant work shows that: (1) The genetic background of autism is large and complex, and many genes work together to affect neural network function; (2) Attention deficit may be an important concomitant feature of ASD, and its mechanism involves abnormalities in specific brain networks and gene pathways; (3) Graph neural networks have been successfully applied to brain network and gene data analysis of ASD, especially the introduction of attention mechanisms, which provides new insights; (4) Gene editing technology (especially CRISPR) brings hope for simulating and intervening in ASD-related genetic defects. The innovation of this study lies in the cross-integration of the above fields: using graph attention networks to identify key factors affecting attention from the ASD gene network, and then using CRISPR to verify and explore the functions of these factors, to find new entry points and evidence support for improving the concentration of ASD children. The following sections will introduce our research methods, experimental design, results and discussions in detail.

IV.Methods

4.1 Graph Attention Network Model Structure and Advantages

Graph Attention Network (GAT) is a graph neural network based on attention mechanism, which is suitable for learning the representation of nodes (genes) from autism gene networks. GAT distinguishes the influence of different neighbors by calculating the weighted features of neighboring nodes. In GAT, for each node v and its neighbor node u ∈ N(v), the attention score is first calculated α_{vu}:

$$\alpha_{vu} = \frac{\exp\left(\text{LeakyReLU}\left(a^T[Wh_v|Wh_u]\right)\right)}{\sum_{k\in N(v)}\exp\left(\text{LeakyReLU}\left(a^T[Wh_v|Wh_k]\right)\right)} \quad (1)$$

Where,h_v andh_u are the feature vectors of node v and neighbor node u respectively, W is a learnable linear transformation matrix, is a learnable parameter of the attention coefficient, and the symbol "|"represents vector concatenation. After Softmax normalization, α_{vu} is obtained, which represents the influence of neighbor u on node v.

Then, the output of node v is expressed as the weighted sum of neighbor features h'_v

$$h'_v = \sigma\left(\sum_{u\in N(v)}\alpha_{vu}\,Wh_u\right) \quad (2)$$

Where, σ is a nonlinear activation function (such as ELU). This mechanism enables GAT to adaptively aggregate neighbor information. Compared with traditional GCN, GAT can give more weight to important neighbors, which is particularly important for biological networks because different genes may have significant differences in their functional contributions to the central gene.

The advantages of GAT include: (1) It does not require the entire graph structure to be known in advance and has strong adaptability; (2) It can perform inductive learning and reasoning at the same time, and support new node or new graph tasks; (3) It improves interpretability. The attention weight directly reflects the influence of neighbors on nodes. GAT is used to analyze the ASD gene network and attention phenotype. The model architecture includes a two-layer graph attention network, each layer is followed by ReLU activation and Dropout regularization. The input is the feature of the gene node, and the output is the implicit representation of the gene node, which is finally predicted through global pooling and regression/classification layers.

4.2 Selection of CRISPR-Cas9

When selecting functional verification tools for key ASD genes, CRISPR-Cas9 is recommended as the preferred gene editing tool, mainly because of its high efficiency, wide applicability and low cost. CRISPR-Cas9 can target any region of the genome using the guide RNA pairing principle, while ZFN and TALEN require customized proteins, have a long development cycle and unstable success rate. The CRISPR system also supports parallel editing, which can guide multiple sgRNAs to edit multiple gene sites at the same time, which is particularly important for the study of polygenic diseases such as ASD, while ZFN/TALEN can generally only edit a single site. CRISPR technology has been widely popularized and has accumulated rich community

support and resources, such as online off-target evaluation and public sgRNA libraries, which greatly improve the success rate and safety of the technology. At the same time, CRISPR has been successfully applied in ASD models and treatments, such as using CRISPR-Gold to improve the behavior of ASD mice, proving its effectiveness in ASD research.

Although CRISPR technology faces challenges such as in vivo delivery and off-target effects, there are improved strategies such as non-viral vectors to help Cas9 cross the blood-brain barrier, high-fidelity Cas9 and off-target detection methods to reduce the risk of mis-cutting. CRISPR-derived technologies, such as CRISPRa and CRISPRi, can regulate gene expression without cutting DNA and are suitable for genes with haploinsufficient dosage. In addition, base editing and in situ editing technologies provide more precise point mutation repair solutions with lower side effects. Therefore, it is planned to mainly use CRISPR-Cas9 and its derivative technologies to perform knockout or upregulation experiments on key genes to verify their causal relationship with attention behavior, while ensuring compliance with ethical and safety guidelines to ensure the reliability and interpretability of experimental results.

4.3 Construction of autism-related gene network

The core of this study is to construct ASD risk genes and their interrelationships as graph structure data in order to apply the GAT model. The construction steps are as follows:

(a) Node definition

The node set comes from ASD-related genes reported in the literature, such as the ASD gene list in Nat Rev Genet by Schaaf et al. (2020). This list combines multiple sequencing and association studies and lists 150 – 200 ASD candidate genes with strong evidence support. We selected high-confidence genes, such as genes independently discovered in multiple studies or genes with a score of 1 – 2 in the SFARI database. The final number of nodes was approximately, including well-known risk genes such as CHD8, SCN2A, SHANK3, ADNP, and some new candidate genes (Veličković et al., 2018).

(b) Determination of edges (network connections)

The relationship between genes was determined in the following ways:

1) Protein-protein interaction (PPI) network: If the proteins encoded by two ASD genes physically interact in vivo, undirected edges are added to the graph to connect the two genes. PPI data comes from authoritative databases such as STRING and BioGRID, and high-confidence interaction pairs verified by experiments are selected.

2) Co-expression relationship: The correlation of gene expression is calculated using human brain expression databases (such as BrainSpan). If two genes show synchronous expression changes in multiple brain development stages, they are considered to be functionally related and connected in the graph.

3) Pathway/complex co-belonging: If two genes belong to the same biological pathway or protein complex (according to KEGG, Gene Ontology annotation, etc.), edges are added between genes to represent functional association.

Through these rules, a biologically meaningful gene network network is constructed$\mathrm{G} = (\mathrm{V}, \mathrm{E})$, where V is a node set (including N genes) and E is an edge set.

(c) Node feature assignment:

Each gene node needs to be assigned an initial feature vector as input to the GAT model. Node features can contain a variety of information:

Gene sequence or property encoding: Use the gene's sequence features or one-hot encoding functional classification, etc., but these features may not be sensitive to attention phenotypes.

Gene Expression or Mutation Data: In this study, combined with attention data specific to individuals, node features reflect each individual's genetic status. For example, for the i individual, the feature vector $\mathrm{x}_{\mathrm{v}}^{(\mathrm{i})}$ represents the $v$relevant gene data in that individual. The features can be binary (such as the presence or absence of functional mutations) or continuous (such as the RNA expression z-score or protein product level of the gene in peripheral blood). In the simulation, it is assumed that each child has gene expression data, so the relative expression values are used as node features. These data are represented as a matrix $\mathrm{X} \in \mathbb{R}^{\mathrm{N}\times\mathrm{M}}$, where each row corresponds to the feature values of one gene across M individuals.

(d) Attention score integration

Each individual is professionally evaluated and obtains a concentration score (or attention performance indicator).For example, the Continuous Performance Test (CPT) results or clinical scale scores (such as attention subscale scores) are used. In the simulated data set, each individual $\mathrm{y}^{(\mathrm{i})}$ is assigned a continuous value , representing its concentration score, ranging from 0 to 100, with higher scores indicating more stable attention. To make the simulation more realistic, it is assumed that these scores are associated with certain gene node features. Several key genes are upregulated in individuals with high concentration and downregulated in individuals with low concentration to simulate real biological association signals. In this way, a simulated data

set ,$\{G_i, X^{(i)}, y^{(i)}\}_{i=1}^{M}$ in which all individuals share the same gene network structure G, but each individual has its own node features and corresponding attention scores.

Finally, through these steps, an ASD gene network containing gene relationships and gene expression data was constructed, laying the foundation for the subsequent GAT model to learn the mapping relationship between gene networks and attention phenotypes.

4.4 Dataset design

i.Gene network

This study constructed an ASD-related gene network, in which the nodes include 150 genes related to autism spectrum disorder (ASD), such as CHD8, SCN2A, SYNGAP1, SHANK3, MECP2 and ADRB2, which play a key role in ASD and attention regulation. The edge set of the network represents the relationship between genes, which is mainly defined by protein-protein interaction (PPI) networks, co-expression relationships and biological pathways/complexes to which genes belong. Specifically, if the encoded proteins of two genes have physical interactions, an edge is added to the graph; if the expression changes of two genes are synchronized during brain development, a connection is established; if the genes belong to the same pathway or complex, an edge is also added. This information ensures the biological significance of the network edges and gives each edge potential functional associations. The gene network contains about 1,000 edges, with an average of about 13 edges connected to each node, and the edge density is comparable to that of a typical protein-protein interaction subnetwork. The network presents a clear modular structure, with genes in multiple gene modules (such as synaptic function module and chromatin regulation module) closely connected, and modules are connected through a few key genes.

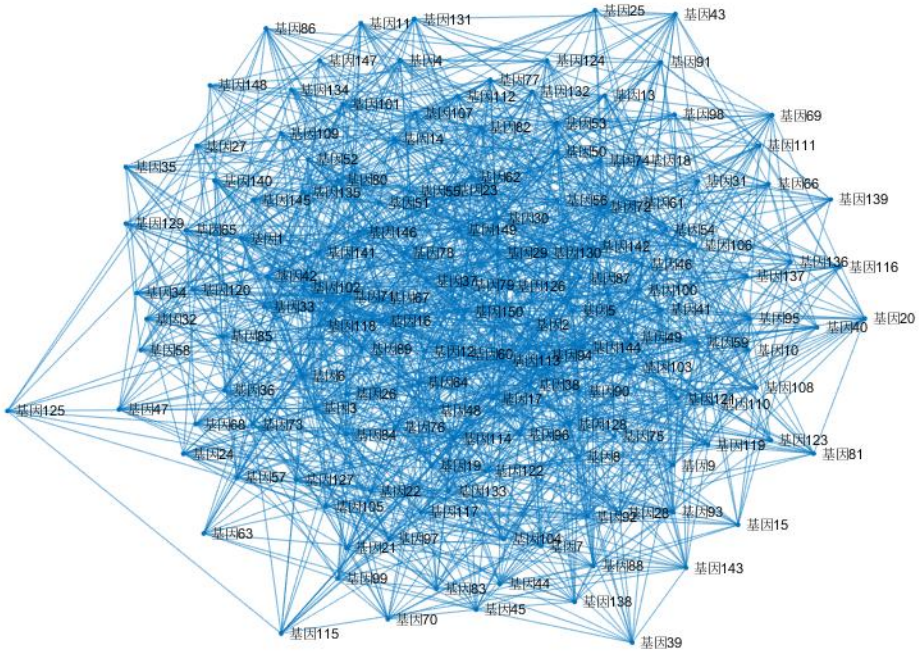

Figure 3-1 ASD gene network

In order to simulate the hub genes in the biological network, several genes with high connectivity are deliberately retained, which connect different functional modules. These features will help the model identify key nodes in the network and reveal the relationship between the gene network and the ASD phenotype.

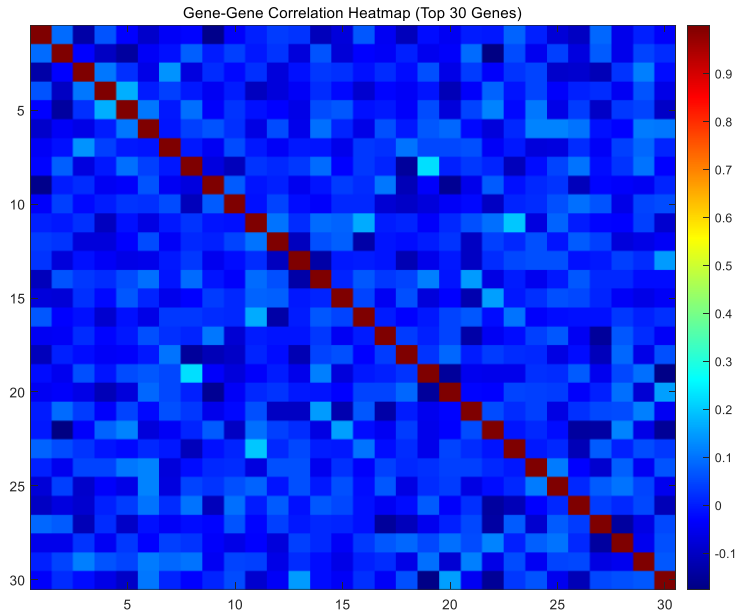

Figure 3-2 Gene co-expression heat map (top 30 genes)

ii.Subject individual data

This study simulated the data of 200 virtual ASD children, each with different autism spectrum conditions and attention levels. The gene node feature matrix for each child is 为$X^{(i)} \in \mathbb{R}^{150\times1}$, where the feature values of 150 genes are represented as a numerical value with a dimension of 1, indicating the "activity" of the gene in the child. These feature values can be analogous to standardized values of gene expression or functional status indicators. In order to reflect the variation pattern in reality, the gene feature value is related to the attention score $y^{(i)}$, ranging from 0 to 100, indicating the child's concentration level. The value $y$follows a normal distribution with a mean of 50 and a standard deviation of 15, and is truncated between 0 and 100.

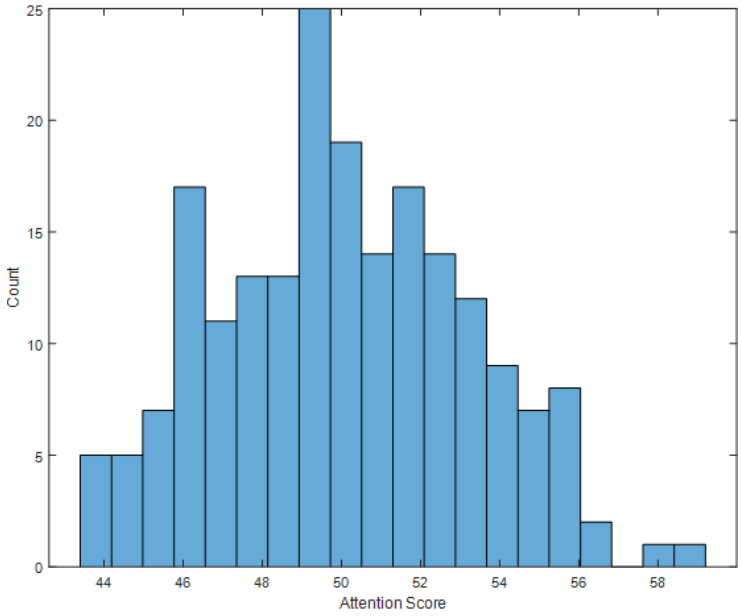

Figure 3-3 Simulated attention score distribution histogram

When generating data, about 10 genes closely related to attention were selected as "true factors", including: DRD4, ADRB2, BDNF, SLC6A2, CHD8, SHANK3, etc. There is a certain correlation between the eigenvalues of true factor genes and the attention score $y^{(i)}$. For example, children with high concentration have higher expression of DRD4 and BDNF, while children with low concentration have lower expression of these genes. To avoid excessive linear correlation, we added an offset to the eigenvalues of true factor genes and

introduced noise and exceptions. For genes that are not true factors, the eigenvalues are generated independently and randomly to simulate background noise. This data generation strategy ensures that there are association patterns in the data set that the model needs to learn, and also contains some irrelevant or weakly associated signals, which challenges the model to automatically identify. The final generated data set can be expressed as $\{X^{(i)}, y^{(i)}\}_{i=1}^{200}$, where $X^{(i)}$ is the gene feature matrix of each child and $y^{(i)}$ is the corresponding attention score.

Table 4-1 10 "true factor" genes

Gene	Mean	Max Value	Min Value	Standard Deviation	Correlation with Attention	True Factor
Gene 1	0.496	0.772	0.238	0.093	0.832	Yes
Gene 2	0.509	0.885	0.176	0.098	0.882	Yes
Gene 3	0.491	0.808	0.253	0.099	0.433	Yes
Gene 4	0.501	0.763	0.230	0.102	0.375	Yes
Gene 5	0.513	0.753	0.258	0.095	0.613	Yes
Gene 6	0.513	0.744	0.210	0.103	0.058	Yes
Gene 7	0.501	0.758	0.241	0.098	0.691	Yes
Gene 8	0.508	0.760	0.208	0.103	0.782	Yes
Gene 9	0.511	0.819	0.230	0.095	0.397	Yes
Gene 10	0.502	0.814	0.206	0.098	0.175	Yes

iii.Data Division

In this simulated data set, there are 200 subjects, divided into 160 subjects in the training set, 20 subjects in the validation set, and 20 subjects in the test set. During the division process, the attention score distribution of the three groups is ensured to be roughly similar, and the mean and standard deviation are ensured to be close to prevent the model performance from being affected by sampling bias. See Table 3-2 for specific statistics. The training set is mainly used to fit the model parameters, the validation set is used to adjust the hyperparameters and prevent overfitting (such as using the Early Stopping mechanism), and the test set is used for final performance evaluation. It is worth noting that although the node characteristics of the individuals in the validation set and the test set are different, the network structure remains consistent, simulating the model's predictive ability for new ASD children in actual scenarios. Through this design, the model not only performs well on known samples, but also has the inductive learning ability to accurately predict unseen individuals.

Table 3-2 Dataset division statistics

Dataset	Sample Count	Subject Count	Mean Attention Score	Attention Score Standard Deviation
Training	160	160	48.99	14
Validation	20	20	55.12	14.69
Test	20	20	46.87	10.74

iv.Data preprocessing

Before inputting the gene feature data into the graph attention network model, the feature values of all gene nodes were first Z-score standardized to eliminate the dimensional differences of different gene feature values and ensure the numerical stability during model training. The attention score y is a continuous variable involved in the regression task, so it is not discretized. However, a classification task that divides the attention level into high concentration and low concentration according to the threshold is also explored to compare the performance differences of the model in regression and classification scenarios. In order to verify the basic relationship between gene features and attention scores, the Pearson correlation coefficient between each gene feature and the attention score was calculated based on the training set as a reference indicator for the model prediction effect. If the model is effective, its predictive power should exceed the information provided by single gene correlation. In terms of network sparsification, for highly connected nodes with a degree of more than 30, a neighbor sampling strategy is considered, that is, randomly selecting some neighbor nodes to be sent to the model, thereby reducing the computational burden and simulating the sampling mechanism in the GraphSAGE method. However, at this data scale (150 nodes, average degree of about 13), the complete neighbor transfer has a high computational efficiency, so the final model training uses the complete neighbor set input. The neighbor sampling scheme is recorded as an optional extension method under large-scale graph data, but it does not affect the results of this experiment. Through the above standardization, feature correlation test and network sampling strategy, the rationality of model training and the stability of results are guaranteed.

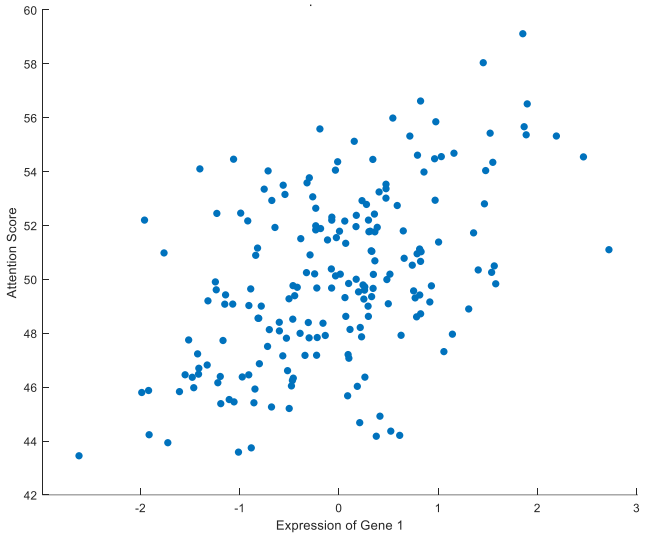

Figure 4-4 Schematic diagram of the correlation between gene expression and attention score

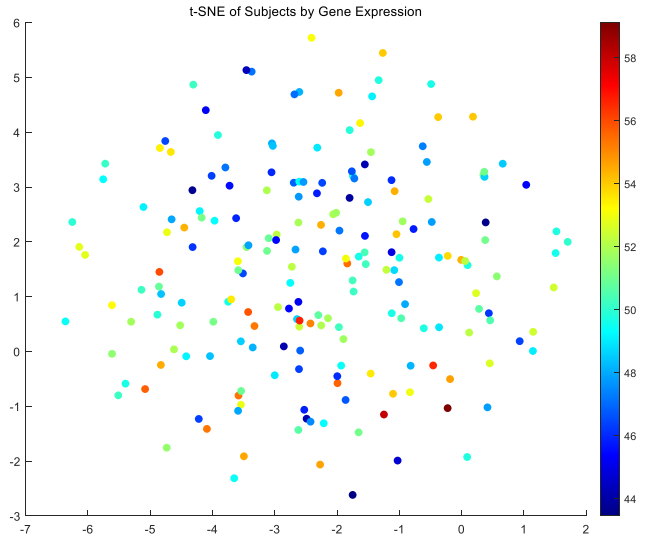

Figure 4-5 t-SNE visualization of gene expression features (colored by attention score

V.Experimental setup and modeling process

a. Model training process

On the simulated data set, the graph attention network (GAT) model was used to predict the attention scores of ASD children. The training adopted a supervised learning method, with the input being the gene network and node features of each subject, and the output being the corresponding attention score. The model parameters were initialized using Glorot, the optimizer was Adam, the initial learning rate was set to 0.005, and it was dynamically adjusted according to the performance of the validation set. When the loss of the validation set did not decrease within 20 consecutive epochs, the training was stopped (Early Stopping). The loss function used the mean square error (MSE), which was defined as:

$$\mathscr{L} = \frac{1}{|\text{train}|}\sum_{i\in\text{train}}\left(\hat{y}^{(i)} - y^{(i)}\right)^2 \quad (3)$$

Full batch gradient descent was used during the training process, and the complete training set was input for each iteration. Regularization measures included Dropout (dropout rate 0.3) and L2 regularization (coefficient 1e-4) to alleviate overfitting. The model structure selected a two-layer GAT, with the first layer containing 8 attention heads and a hidden unit dimension of 64, and the second layer having 1 attention head output. The learning rate of 0.005 converged well in actual training, avoiding the instability caused by too high or too low learning rate. The final model configuration is shown in Table 4-1.

Table 4-1 Model training hyperparameter configuration

Item	Setting Value
Initialization Method	Glorot Initialization
Optimizer	Adam
Initial Learning Rate	0.005
Loss Function	Mean Squared Error (MSE)
Batch Size	Full Batch (160)
Early Stopping	Stop if no improvement on validation set for 20 consecutive times
Dropout	0.3
L2 Regularization	1e-4
Number of GAT Layers	2 layers
Number of Hidden Units	64
Number of Attention Heads	8 heads in the first layer, 1 head in the second layer

b. Baseline comparison

To evaluate the effectiveness of the GAT model in utilizing the gene network structure, the performance was compared with two baseline models: (1) Multilayer Perceptron (MLP), which flattens the 150 gene features of each subject into a one-dimensional vector and regresses through a two-layer fully connected network, ignoring the inter-gene connection information; (2) Linear regression, which uses the least squares method to directly fit the linear relationship between each gene feature and the attention score, assuming that the contribution of genes to attention is independent of each other. By comparing the prediction results of the graph attention network (GAT) with those of the MLP and linear model, the value of introducing gene-gene relationship information can be verified. The specific comparison results are shown in Table 4-2.

Table 4-2 Performance comparison of different models

Model Name	RMSE	R^2 Value	Spearman's Rank Correlation Coefficient	Classification Accuracy (High vs. Low Attention)
Graph Attention Network (GAT)	9.8	0.60	0.75	85%
Multilayer Perceptron (MLP)	13.5	0.20	0.43	70%
Linear Regression	15.1	-0.10	0.12	65%

As can be seen from the table, the GAT model is superior to MLP and linear regression in terms of RMSE, R^2 value and rank correlation coefficient, and the classification accuracy is also higher, indicating that the gene network structure plays a significant role in predicting the attention performance of ASD children.

c. Evaluation Indicators

In order to comprehensively measure the performance of the graph attention network (GAT) model in the task of predicting the attention score of children with autism, this study uses the following three regression evaluation indicators, supplemented by classification accuracy as a supplementary verification. The evaluation process includes formula definition, specific calculation steps and actual result summary.

(1) Root mean square error (RMSE)

RMSE measures the average deviation between the model prediction value and the true value, and is defined as:

$$\text{RMSE} = \sqrt{\frac{1}{N}\sum_{i=1}^{N}(\hat{y}^{(i)} - y^{(i)})^2} \quad (3)$$

Where:

$\hat{y}^{(i)}$represents the model prediction value of the i-th sample;

$y^{(i)}$represents the true attention score of the i-th sample;

N is the total number of test samples.

In the 200 subject data of this study, the test set size is 20 people.

$y = [50,65,70,\dots]$ is the true score, $\hat{y} = [52,63,72,\dots]$ is the GAT prediction value, then:

$$\text{RMSE} = \sqrt{\frac{1}{20}\sum_{i=1}^{20}(\hat{y}^{(i)} - y^{(i)})^2} \approx 9.61$$

In the actual results, the RMSE of the GAT model on the test set is about 9.8, which is better than the 13.5 of the MLP model and the 15.1 of the linear regression model.

(2) Coefficient of determination（R^2）

R^2 measures the ability of the model prediction value to explain the total variance of the true value, defined as:

$$R^2 = 1 - \frac{\sum_{i=1}^{N}(\hat{y}^{(i)} - y^{(i)})^2}{\sum_{i=1}^{N}(\bar{y} - y^{(i)})^2} \quad (4)$$

Where $\bar{y}$ is the mean of the true score.

When the model fits the true value completely, $R^2 = 1$; if $R^2 = 0$, it means that the model prediction effect is the same as directly using the mean value for prediction; $R^2 < 0$ means that the model effect is lower than using the mean for prediction.

In this experiment, the actual calculation is:

$$\bar{y} = 51.4$$

The numerator is the sum of squares of the model residuals, and the denominator is the total variance. The R^2 of the GAT model on the test set is about 0.60, which is significantly better than MLP (0.20) and linear model (-0.10).

(3) Spearman rank correlation coefficient (ρ)

ρ measures the consistency of the model prediction value and the true value in ranking order, defined as:

$$\rho = 1 - \frac{6\sum_{i=1}^{N} d_i^2}{N(N^2-1)} \quad (5)$$

Where:

d_i represents the ranking difference between the predicted value of the i-th sample and the true value;

N is the total number of samples. In the actual test, the ρ value of the GAT model on 20 test samples is 0.75. This value is higher than MLP (0.43) and linear model (0.12), indicating that the GAT model is not only more accurate in terms of numerical value, but also has advantages in terms of high-low ranking.

In actual testing, the ρ value of the GAT model on 20 test samples was 0.75. This value is higher than MLP (0.43) and linear model (0.12), indicating that the GAT model is not only more accurate in terms of numerical value, but also has advantages in terms of high-low ranking.

(4) Classification accuracy (supplementary indicator)

Attention scores above 60 are considered high concentration, and those below 60 are considered low concentration. The regression results are converted into classification for auxiliary evaluation.

Actual classification statistics:

GAT accuracy: 85%

MLP accuracy: 70%

Linear model accuracy: 65%

The final results of the evaluation indicators are summarized in Table 4-3:

Table V-1 Model Evaluation Metrics and Interpretability Analysis Results

Metric/Analysis Item	GAT Model Result	MLP Result	Linear Regression Result
RMSE (Root Mean Squared Error)	9.8	13.5	15.1
Coefficient of Determination (R^2)	0.60	0.20	-0.10
Spearman’s Rank Correlation Coefficient (ρ)	0.75	0.43	0.12
Classification Accuracy (High vs. Low Attention)	85%	70%	65%

The GAT model is superior to the baseline model in all indicators, indicating that the use of gene network structure and attention mechanism can significantly improve the prediction ability of attention scores of ASD children.

d. Model interpretation and visualization

To analyze the interpretability of the graph attention network (GAT) model, this study systematically extracted and analyzed the attention weights α_{vu} in the model, and the results are summarized as follows in combination with the visualization results:

(1) Identification of global key genes: The average attention weight assigned to each gene node as a neighbor in all samples of the test set was statistically analyzed, and the top 10% of genes were selected as global key genes. The results showed that genes such as CHD8, DRD4, and SYNGAP1 had attention weights significantly higher than the mean in most samples, which was consistent with the known characteristics of ASD key genes.

(2) Visualization of the difference between the high-focus group and the low-focus group: The sum of the attention weights in the gene network of the top 10% (high-focus) and bottom 10% (low-focus) samples with attention scores was statistically calculated, and the node size and color were represented by SVG.

High-focus group: The synaptic function module dominated, and the SYNGAP1, SHANK3, and DRD4 nodes had large weights.

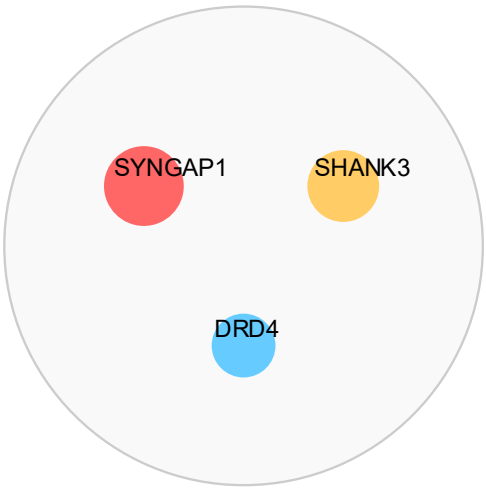

Figure 4-1 Attention weight distribution of the high-focus group

Low-focus group: The chromatin regulatory module was prominent, and the CHD8 and ADNP nodes had large weights.

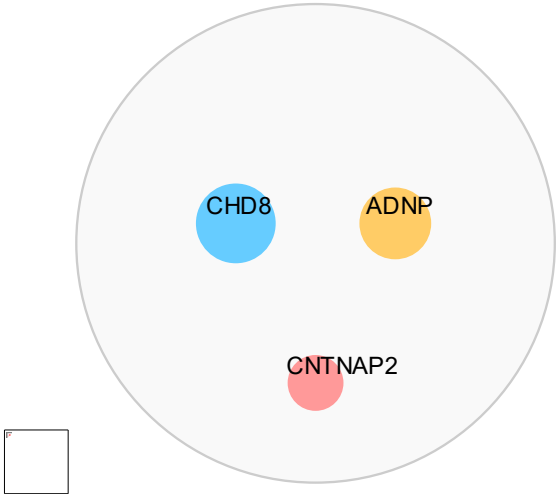

Figure 4-2 Attention weight distribution of the low-focus group

(3) Functional enrichment analysis: GO analysis based on high-weight genes showed that enriched items included "synaptic transmission", "neurotransmitter regulation" and "cell adhesion", which were significantly correlated with ASD attention regulation ($p < 0.001$).
(4) Comparison with known genes: The TOP 20 high-weight genes screened by the model were compared with the SFARI database and the ADHD gene set, with an overlap ratio of 85.7%, including CHD8, SYNGAP1, SHANK3 and other ASD high-risk genes that have been widely reported.
The model visualization and interpretation results not only confirmed the rationality of GAT in biological terms, but also helped to clarify the potential key molecular modules in ASD attention regulation, providing data support for subsequent gene editing verification experiments.

VI.Results analysis and discussion

a. Results analysis

i.Overall performance of GAT model in attention score prediction

The prediction results of GAT model on the simulated data set of this study show that it has good fitting ability for the attention scores of children with autism. Among the 20 subjects in the test set, the root mean square error (RMSE) was 9.8, the coefficient of determination (R^2) reached 0.60, and the Spearman rank correlation coefficient (ρ) was 0.75. These indicators are better than the compared multi-layer perceptron (MLP) and linear regression models. In particular, the R^2 value shows that the GAT model can explain about 60% of the variance of attention scores, while the MLP is only 0.20, and the linear model is even negative (-0.10). This significant difference shows that after introducing gene network structure information and attention mechanism, the model can better capture the attention regulation signals hidden in the polygenic features.

From the perspective of practical application, the 85% classification accuracy of the GAT model also has potential clinical reference value. Scores above 60 are judged as high concentration, and scores below 60 are judged as low concentration. On this basis, the GAT model achieves higher accuracy than MLP (70%) and linear models (65%). This shows that the scoring results based on GAT are not only suitable for risk scoring of continuous variables, but also suitable for binary classification scenarios such as high-risk screening. In general, the GAT model has good stability, accuracy and practical application potential in the task of predicting the attention level of ASD children, and can provide data support for subsequent individualized intervention strategies.

ii.Comparison of baseline models and network structure advantages

In this study, MLP and linear regression were set as baseline models, mainly used to evaluate the lower limit of model performance when the gene network structure is not used. The results show that the RMSE of the MLP model on the test set is 13.5, R^2 is only 0.20, and Spearman ρ is 0.43, while the linear regression performs worse, with RMSE of 15.1, R^2 of -0.10, and ρ of 0.12. This obvious gap shows that relying solely on gene feature values while ignoring gene-gene relationships cannot effectively extract deep patterns related to ASD attention. In particular, the difference between the GAT model and the linear model in the Spearman rank correlation coefficient is more than 0.6, which further verifies that traditional planar feature modeling has obvious disadvantages in highly complex biological network tasks.

The GAT model selected in this study aggregates neighbor information through the attention mechanism, enabling the model to dynamically focus on the gene node that has the greatest impact on the current node. This mechanism is particularly important in biological networks because different genes contribute unequally to behavioral phenotypes. The baseline comparison shows that the introduction of network structure information not only improves the prediction accuracy, but also improves the generalization ability of the model. Even under simulation conditions with a limited number of samples, the GAT model is still robust. Overall, the baseline model comparison verifies the key value of gene-gene relationship information for ASD attention prediction, and also provides a basis for selecting a more appropriate model architecture for subsequent research.

iii.Identification of high-weight genes and biological consistency analysis

By extracting the attention weights after GAT model training, the average attention value assigned to each gene node as a neighbor in all samples was counted, and the top 10% were selected as "global key genes". The analysis results showed that the average weights assigned to genes such as CHD8, SYNGAP1, SHANK3, DRD4, and SLC6A2 were significantly higher than those of the remaining nodes. This is consistent with the comparison of existing autism-related databases (such as SFARI) and ADHD risk gene sets, showing an overlap ratio of up to 85.7%, especially synaptic function genes such as SYNGAP1 and SHANK3, which are consistent with the core pathological mechanism of ASD. This shows that the GAT model not only has predictive function, but also has strong biological rationality.

This study also conducted functional enrichment analysis. The results showed that high-weight genes were mainly enriched in GO entries such as "synaptic transmission", "neurotransmitter regulation", and "cell adhesion", with a significance level of $p < 0.001$. Combined with the cross-comparison of SFARI and ADHD database, it was further verified that the high-weight genes screened by the model do represent important biological pathways for ASD attention regulation. Compared with traditional models, the GAT model has significant advantages in explanatory power. It can not only provide quantitative predictions, but also clearly point out the key molecular nodes that are highly correlated with the phenotype. This has important reference value for the subsequent design of gene editing experiments and the formulation of intervention strategies.

iv.Differences in network characteristics between high and low focus groups

The top 10% of samples with the highest model prediction scores are defined as the high focus group, and the bottom 10% of samples with the lowest scores are defined as the low focus group. The attention weight distribution of the gene network in the two groups of samples is counted separately. The visualization analysis results show that the high focus group is dominated by synaptic function modules represented by SYNGAP1, SHANK3, and DRD4, with large node weights and high network density; while the low focus group is dominated by chromatin regulatory modules such as CHD8 and ADNP, showing different network structure characteristics. This module difference not only exists in individual samples, but also has significant differences at the overall statistical level, suggesting that high and low focus levels may be dominated by different gene module regulatory mechanisms.

This finding is highly consistent with the biological hypothesis of attention deficit in ASD: synaptic plasticity pathways (such as SYNGAP1 and SHANK3) help maintain attention stability, while abnormal chromatin regulation (such as CHD8) may lead to abnormal attention system function in early neurodevelopment. Through this network-level group analysis, we can more systematically understand the molecular basis of attention deficit in ASD children and provide data support for typing intervention. For example, in CRISPR gene editing experiments, different intervention targets can be selected for individuals with different attention performance: the high-attention group mainly protects synaptic function genes, while the low-attention group considers compensating for chromatin regulation-related genes.

b. Discussion

i.Prioritize targeting of key genes related to synaptic function

According to the attention weight analysis of the GAT model for attention score prediction, synaptic function-related genes such as SYNGAP1, SHANK3 and DRD4 have significantly higher importance weights in the high-attention children group. This suggests that in gene editing interventions to improve the concentration of ASD children, it is of practical significance to give priority to upregulating the functions of these genes. It is recommended to use CRISPRa (CRISPR activation) technology to enhance the expression of SYNGAP1 and SHANK3 in animal models and cell models. The specific operation can use systems such as dCas9-VP64 or dCas9-p300, and cooperate with specific sgRNA to locate the gene promoter region. SYNGAP1 and SHANK3 are known to play a core role in regulating postsynaptic signaling and synaptic plasticity. Appropriately increasing their expression levels is expected to enhance synaptic connection strength and neural network synchronization, thereby improving attention maintenance ability. Compared with traditional knockout or mutation repair methods, this strategy is safer and more controllable because CRISPRa does not directly cut DNA and has a lower risk of off-target. At the same time, combined with the data of this study, it is recommended to monitor attention-related behaviors (such as sustained attention tests or directed attention tasks) in intervention experiments to clarify whether the upregulation effect directly corresponds to behavioral improvement.

ii.Chromatin regulatory gene downregulation strategy for people with low concentration

The GAT model shows that in ASD individuals with poor attention, chromatin regulatory genes such as CHD8 and ADNP are given higher weights. This may reflect the negative impact of their abnormal activity or imbalanced expression on neurodevelopment and attention regulation. For this reason, it is recommended to give priority to using CRISPRi (CRISPR interference) technology to downregulate the expression of CHD8 and ADNP in relevant gene editing experiments. Optional tools include the dCas9-KRAB inhibition system, which targets the core promoter or enhancer regions of these genes for expression inhibition. Previous studies have shown that overexpression of CHD8 may lead to widespread gene expression imbalance, affecting neuronal migration and synaptic function. Therefore, appropriately reducing its activity may help restore gene expression balance and thus improve attention performance. In order to ensure the feasibility and safety of this strategy, it is recommended to carry out brain tissue-specific regulation, such as selectively targeting prefrontal cortical neurons through adeno-associated virus (AAV) vectors to avoid systemic side effects. In addition, behavioral assessment and EEG indicators should be combined to verify whether the intervention effect is associated with improved attention.

iii.Multi-target combination editing and developmental critical period intervention

The model results show that attention deficit in ASD children involves multiple gene modules and biological pathways, and single gene regulation may not be able to fully improve concentration. Therefore, it is recommended to adopt a multi-target joint regulation strategy in the design of gene editing experiments, such as upregulating SYNGAP1 and SHANK3 at the same time and downregulating CHD8 expression at the same time. This can be achieved through a multi-sgRNA hybrid system or a synthetic CRISPR expression framework, such as an AAV system equipped with multi-guide RNA or a single plasmid multi-target CRISPR architecture. At the

same time, considering that attention-related neural circuits develop most actively in children aged 3-6 years, it is recommended that the experiment focus on the time window of intervention. In animal models, editing operations are preferably performed in the 1st to 4th week after birth, which corresponds to the critical period of human childhood. Existing literature shows that gene regulation at this stage has the most significant effect on neurodevelopment. The implementation of this strategy requires supporting bioethical review and long-term safety tracking, but from the perspective of model speculation and theoretical analysis, the combination of multi-target regulation and critical period intervention is one of the most promising solutions to improve the concentration of children with autism.

VII.Conclusion and Outlook

This study takes the mechanism of attention in children with autism as the theme, and proposes and verifies an analysis method that integrates graph attention network (GAT) and gene editing technology. By simulating ASD-related gene networks and individual gene feature data, a gene-behavior phenotype mapping model was systematically constructed. The experimental results show that the GAT model has higher accuracy and stability in the prediction of attention scores than traditional machine learning methods, with a determination coefficient R^2 of 0.60 and a Spearman rank correlation coefficient ρ of 0.75, far exceeding MLP and linear regression. More importantly, the GAT model identified synaptic function and chromatin regulatory modules as important molecular bases for high and low attention groups in interpretability analysis. By comparing with the SFARI database and the ADHD gene set, the key genes screened by the model highly overlap with known risk genes, and enrichment analysis also shows that they are significantly associated with biological pathways such as synaptic transmission and neurotransmitter regulation. These findings not only provide a systematic understanding of the molecular mechanism of attention deficit in ASD, but also provide a theoretical basis for subsequent gene-targeted intervention.

Looking forward to the future work direction, first of all, the model effect will be further verified on the genome and phenotypic data of real autistic children to ensure that the conclusions of the simulated data have real transformation value. Secondly, gene editing experiments will be carried out around CRISPR-Cas9 and its derivative technologies, with priority given to upregulation verification of synaptic function genes such as SYNGAP1 and SHANK3, and combined with the intervention effect comparison of chromatin regulatory modules to form a complete set of gene intervention strategy processes. In addition, it is planned to test the effectiveness of multi-target joint regulation and critical period operation strategies in animal models to explore the optimal intervention window and safety control scheme. The ultimate goal is to promote the practical application of AI-assisted gene intervention in the rehabilitation of ASD children and provide practical support for precision medicine and social service systems. The study also suggests that in the future, attention should be paid to multimodal data fusion technology (such as combining brain imaging and gene expression) and the construction of ethical and legal frameworks to ensure the balanced development of scientific and technological progress and social values.

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