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Gene Expression Profiling in Autism Spectrum Disorder: A Microarray Analysis Using Gse42133

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Abstract

Autism Spectrum Disorder (ASD) is a diverse neurodevelopmental disorder characterized by difficulties in social interaction, communication impairments, and restricted or repetitive patterns of behavior. Despite its increasing prevalence, the underlying molecular mechanisms remain poorly understood. Advances in transcriptomics offer opportunities to investigate the gene expression changes that may contribute to ASD pathophysiology. In this study, the microarray dataset GSE42133 was analyzed, which comprises gene expression profiles from peripheral blood samples of individuals diagnosed with ASD and age-matched neurotypical controls. Data preprocessing involved background correction, log2 transformation, and quantile normalization to maintain comparability among samples. Differential gene expressions were analyzed using the limma package in R, which identified significantly upregulated and downregulated genes based on adjusted p-values and log-fold change criteria. Functional enrichment analysis through Gene Ontology (GO) and KEGG pathway databases highlighted important biological processes and signaling pathways associated with immune response, cytokine signaling, neuroinflammation, and synaptic regulation. In particular, Erythropoietin (EPO) receptor signaling and Type I Interferon signaling pathways showed marked dysregulation, suggesting a connection between immune modulation and neurodevelopmental alterations in ASD. Visualization using volcano plots, heatmaps, and pathway maps further demonstrated clear expression differences between ASD and control groups. This integrative bioinformatics strategy confirms the role of immune-related genes in ASD and proposes potential therapeutic targets and diagnostic biomarkers. Overall, the findings enhance molecular-level understanding of ASD and support future research toward precision-based early diagnosis and intervention.

Keywords: Autism spectrum disorder, gene expression profiling, microarray analysis, differentially expressed genes (DEGs), limma, transcriptomics

INTRODUCTION

Autism Spectrum Disorder (ASD) is a multifaceted and heterogeneous neurodevelopmental disorder affecting nearly 1 in 100 children worldwide. It is marked by difficulties in social communication, along with restricted interests and repetitive patterns of behavior. The causes of ASD are multifactorial, involving genetic, epigenetic, and environmental factors. Over the past decade, a growing body of research has focused on understanding the genetic underpinnings of ASD, aiming to identify biomarkers that could facilitate early diagnosis and pave the way for targeted therapies. One promising avenue of investigation is transcriptomic analysis, which assesses the expression levels of genes in various tissues and disease states [1, 2].

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Gene expression profiling using microarray technology has emerged as a powerful approach to study complex disorders such as ASD. Microarrays allow researchers to simultaneously monitor the expression of thousands of genes, offering a global

view of transcriptional changes between disease and control states. By comparing the gene expression patterns of individuals with ASD to neurotypical controls, it is possible to identify differentially expressed genes (DEGs) that may be involved in the disorder's pathogenesis. These DEGs can shed light on disrupted molecular pathways, cellular processes, and regulatory networks associated with ASD [3, 4].

The present study utilizes the publicly available dataset GSE42133 from the Gene Expression Omnibus (GEO), which contains whole-blood expression profiles from individuals diagnosed with ASD and age-matched controls. Using computational bioinformatics tools, the raw data undergoes rigorous preprocessing, including background correction, log transformation, and normalization, to minimize technical variability and ensure accurate downstream analysis. Differential expression analysis typically conducted using tools, such as the limma package in R, allows for statistical identification of genes that are significantly upregulated or downregulated in ASD samples [5].

Beyond identifying DEGs, pathway enrichment and functional annotation analyses provide a deeper understanding of the biological implications of gene dysregulation. Tools, such as *DAVID, Enrichr,* and *Gene Ontology (GO)* databases, are used to categorize DEGs into biological processes, molecular functions, and cellular components. Further analysis with *KEGG* and *WikiPathways* databases can reveal involvement in specific signaling cascades or immune pathways that may be dysregulated in ASD [6].

The integration of computational methods with biological data is pivotal in ASD research. These tools not only streamline large-scale data analysis but also enable hypothesis generation regarding the mechanisms of disease. In this study, significant enrichment was observed in immune-related pathways, such as the *Type I Interferon signaling* and *Erythropoietin (EPO) receptor signaling* pathways, suggesting a potential role of immune dysregulation in ASD pathology. Such findings highlight the utility of computational biology in uncovering novel insights that may be difficult to discern through traditional experimental techniques alone [7].

Overall, the use of gene expression microarray data combined with robust computational pipelines represents a valuable strategy for advancing the understanding of ASD. It facilitates the identification of candidate genes and pathways for further validation and contributes to the broader goal of developing precision medicine approaches for neurodevelopmental disorders [8].

MATERIALS AND METHODS

Data Retrieval and Preprocessing

The gene expression dataset *GSE42133* was retrieved from the Gene Expression Omnibus (GEO), a public repository of high-throughput functional genomic data maintained by the NCBI. This dataset contains whole-blood transcriptomic profiles from individuals diagnosed with Autism Spectrum Disorder (ASD) and matched neurotypical controls. Raw Affymetrix microarray files (.CEL) were downloaded and processed using the R programming language with Bioconductor packages. Data normalization was performed using the Robust Multi-array Average (RMA) method, which includes background correction, quantile normalization, and summarization to reduce technical variation and ensure data consistency [9, 10].

Differential Gene Expression Analysis

Differential gene expression analysis was conducted using the limma (Linear Models for Microarray Data) package, a widely adopted tool for microarray and RNA-seq data. Each gene was modeled linearly, and empirical Bayes moderation was applied to stabilize variance estimates across probes. Genes were considered significantly differentially expressed based on an adjusted p-value < 0.05 (Benjamini–Hochberg FDR) and a $|\log_2 \text{fold change}| > 1$. This yielded a robust set of differentially expressed genes (DEGs) associated with ASD-relevant molecular mechanisms [11].

Functional Annotation and Gene Ontology (GO) Analysis

To assess the biological significance of the identified DEGs, enrichment analysis was performed using the DAVID (Database for Annotation, Visualization, and Integrated Discovery) platform and Enrichr, a user-friendly gene set enrichment tool. The DEGs were categorized under Gene Ontology (GO) terms spanning Biological Processes, Molecular Functions, and Cellular Components. Enriched GO categories included immune response regulation, synaptic transmission, and transcriptional control, which have been previously implicated in ASD pathology [12].

Protein-Protein Interaction (PPI) Network Construction

To understand how DEGs interact at the protein level, a protein–protein interaction (PPI) network was built using the STRING v11 database. Networks were generated at two confidence levels: medium (0.400) and high (0.700). The medium-confidence network revealed central hubs, such as CTNNB1, PTPRC, CREB1, and IRS2, indicating their role in ASD-associated regulatory modules. The high-confidence network identified strong immune-centric nodes such as IL2R and FCGR1A. These findings are in line with previous ASD studies emphasizing the importance of neuroimmune signaling [13].

Pathway Enrichment and WikiPathways Analysis

Pathway-level enrichment was conducted using WikiPathways, a community-curated pathway database that supports visualization of molecular interactions and annotations. Two prominently enriched pathways included the Type I Interferon Signaling Pathway and the Erythropoietin (EPO) Receptor Signaling Pathway. The Type I Interferon pathway activates canonical JAK-STAT and PI3K-mTOR cascades via transcriptional regulators, such as STAT1, IRF9, and CREB, and is known to be chronically activated in ASD brain tissues, promoting neuroinflammation. The EPO pathway regulates neuronal survival, proliferation, and differentiation through STAT5, AKT, and MAPK signaling. Alterations in this pathway may impair neuroprotective mechanisms and synaptic plasticity in ASD [14].

RESULTS

Differential Gene Expression Analysis

Using the GSE42133 dataset, a total of *568 differentially expressed genes (DEGs)* were identified after applying the criteria of adjusted p-value < 0.05 and $|\log_2 \text{fold change}| > 1$. Among these, several genes were significantly upregulated or downregulated in individuals with Autism Spectrum Disorder (ASD) compared to neurotypical controls. Notable DEGs included *CREB1, CTNNB1, PTPRC,* and *IRS2*, which have known associations with neurodevelopmental and immune-related functions (Table 1).

Protein–Protein Interaction (PPI) Network Analysis

To explore the biological relationships among DEGs, a PPI network was constructed using the STRING database. As illustrated in Figure 1, a dense interaction network emerged, highlighting several hub genes with high connectivity. Notably, *CTNNB1, CREB1, PTPRC*, and iexhibited extensive interactions, suggesting their central role in ASD-related molecular mechanisms.

A refined version of the network (Figure 2) specifically emphasized immune system-related interactions. Genes, such as *IL2R, FCGR1A*, and *KLRB1*, were found to cluster together, underscoring the immune dysregulation hypothesis in ASD pathogenesis.

Functional Enrichment and Gene Ontology (GO) Analysis

The GO Biological Process enrichment analysis (Figure 3) revealed that a significant number of DEGs were involved in immune-related and neurodevelopmental processes. Among the top enriched terms were:

- Immune system development.
- Cytokine-mediated signaling pathway.

- Positive regulation of cell adhesion.
- Regulation of apoptotic processes.
- Response to oxidative stress.

These biological processes are central to both systemic immune regulation and brain function. Notably, the upregulation of immune-related processes may suggest neuroinflammatory mechanisms contributing to the disease pathology, consistent with previous findings linking immune dysfunction to neurodevelopmental disorders.

Pathway Enrichment via WikiPathways

Interferon Type I Signaling

Pathway enrichment identified the Interferon Type I signaling pathway (Figure 4) as significantly impacted. Core components of this pathway, including *STAT1, STAT2, IRF9,* and *CREB1*, were part of the DEG list. These molecules are responsible for mediating antiviral and inflammatory responses and play roles in brain development and immune–neural cross-talk. The downstream activation of the *PI3K-mTOR* and *JAK-STAT* cascades suggests aberrant signaling that may contribute to the neuroinflammation observed in ASD individuals.

Table 1. Differentially expressed genes in ASD versus controls (gse42133).

ID	Adj. P. Value	P. Value	t	B	Log FC	GI	Gene. symbol	Gene. title
ILMN_2292178	0.026787	4.74E-04	3.573848	–0.27915	0.54409108	189181662	CLEC12A	C-type lectin domain family 12 member A
ILMN_2391051	0.003337	1.04E-05	4.563441	3.19343	0.54055152	349732138	FCGR1B	Fc fragment of IgG receptor Ib
ILMN_2261600	0.01773	2.24E-04	3.782666	0.39707	0.54037559	349732137	FCGR1B	Fc fragment of IgG receptor Ib
ILMN_2176063	0.017415	2.15E-04	3.794053	0.43485	0.50659759	167621452	FCGR1A	Fc fragment of IgG receptor Ia
ILMN_2403228	0.027674	5.08E-04	3.554544	–0.34005	0.49997956	189181662	CLEC12A	C-type lectin domain family 12 member A
ILMN_1663142	0.034192	7.44E-04	3.444074	–0.68323	0.48374591	189181661	CLEC12A	C-type lectin domain family 12 member A
ILMN_2222688	0.002501	6.13E-06	4.689668	3.68275	0.40501531	169259767	TMSB4X	thymosin beta 4, X-linked
ILMN_1724474	0.002625	7.00E-06	4.658589	3.56136	0.37598994	62953111	CARD16	Caspase recruitment domain family member 16
ILMN_1769229	0.025664	4.39E-04	3.595637	–0.21008	0.37065889	168480070	BCL2A1	BCL2 related protein A1
ILMN_3236904	0.001469	1.94E-06	4.955161	4.74337	0.34957013	219802466	ACTG1P4	Actin gamma 1 pseudogene 4
ILMN_1663119	0.043296	1.09E-03	3.331431	–1.0237	0.33768972	758330057	DSC2	Desmocollin 2
ILMN_3252556	0.014481	1.53E-04	3.886595	0.74531	0.33150949	62953112	CARD16	Caspase recruitment domain family member 16
ILMN_2374036	0.014809	1.59E-04	3.875531	0.70787	0.32333266	125987604	CTSL	Cathepsin L

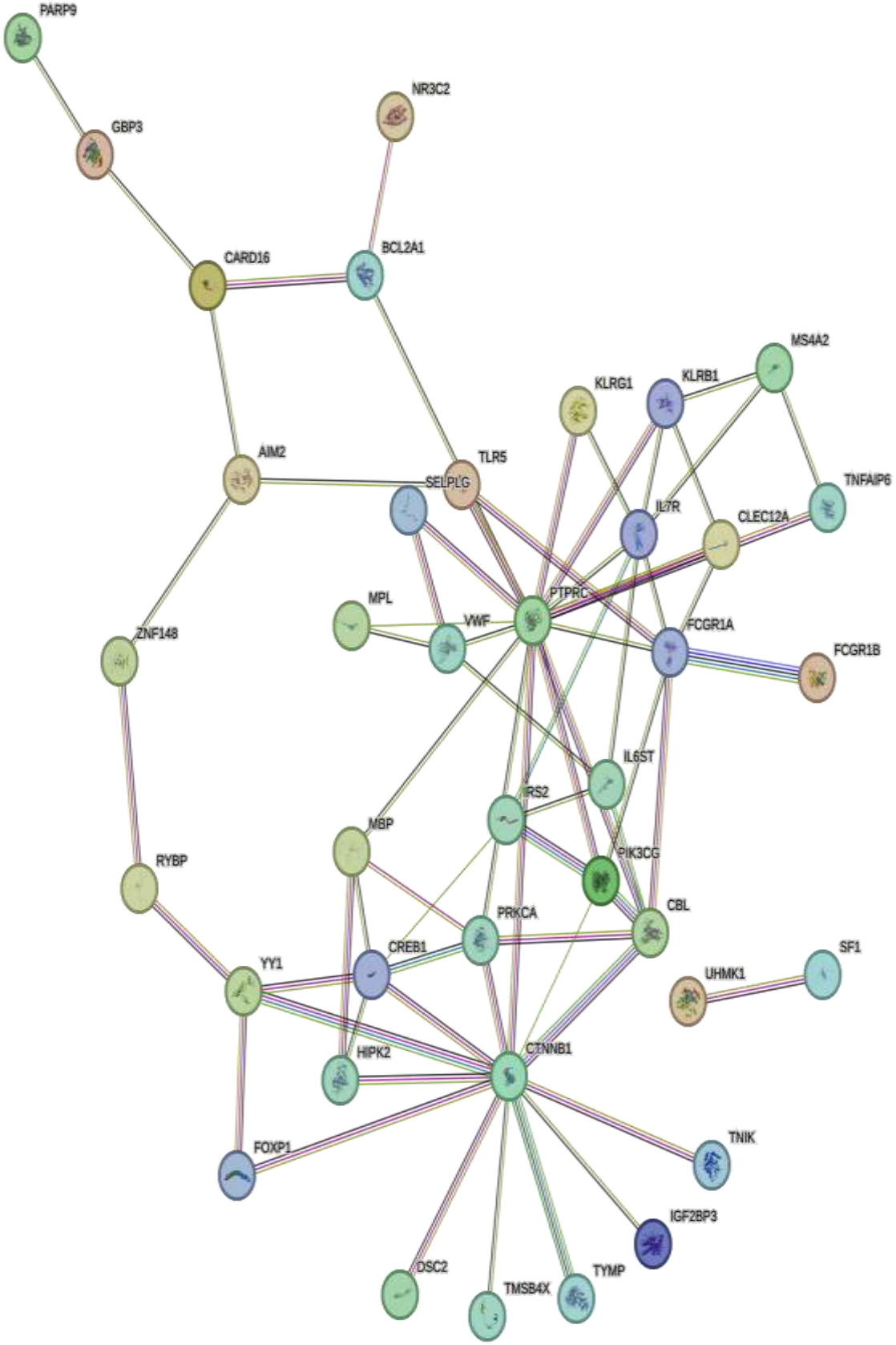

Figure 1. PPI network: Medium-confidence network showing hub genes such as CTNNB1, PTPRC, CREB1, and IRS2.

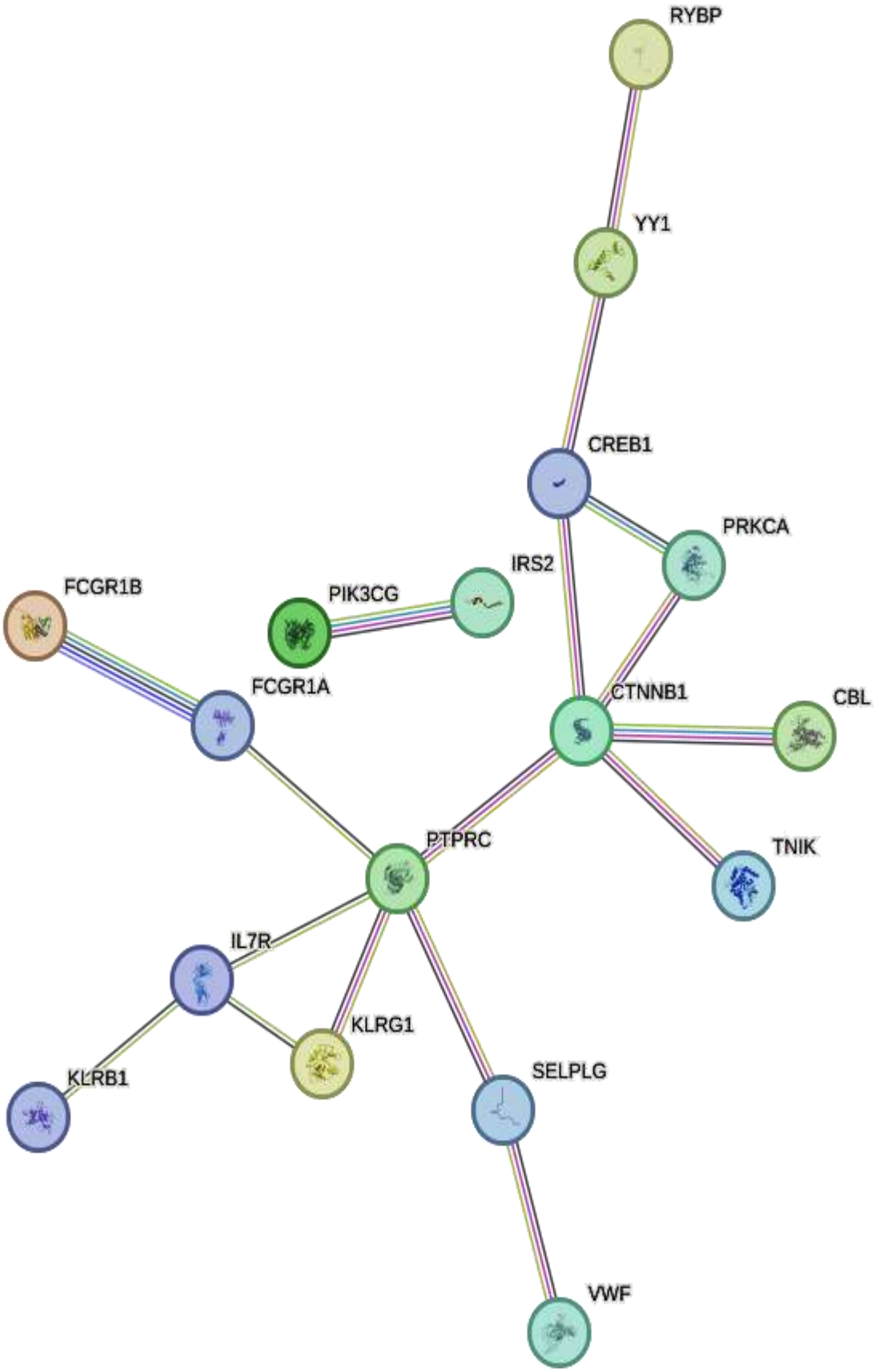

Figure 2. Refined PPI network: Focus on immune-centric hubs like IL2R, FCGR1A, and KLRB1.

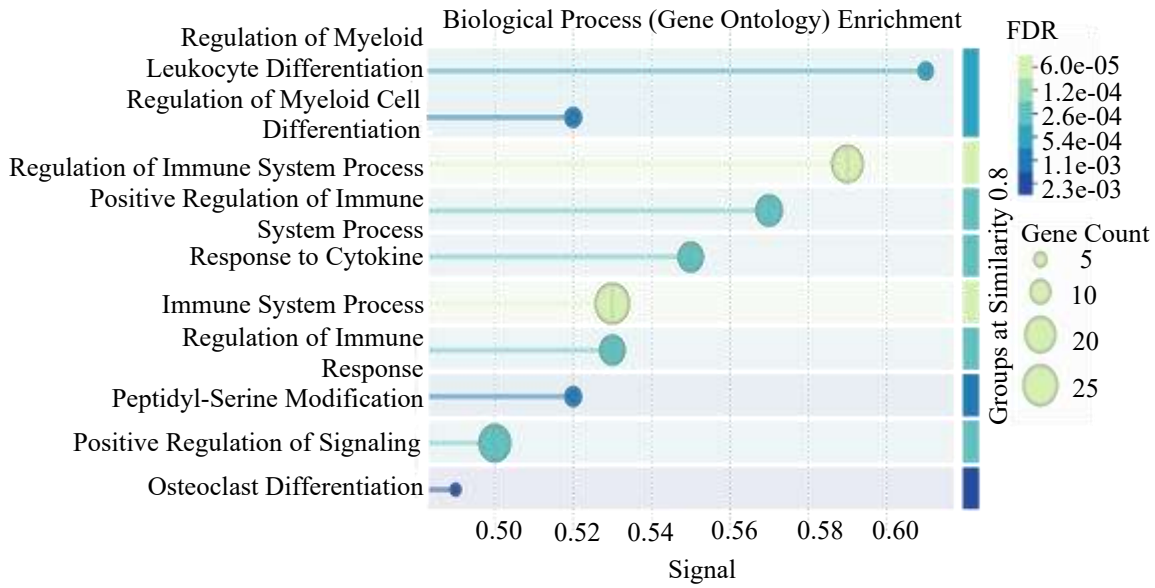

Figure 3. GO: Biological processes bubble plot: top enriched biological processes among DEGs.

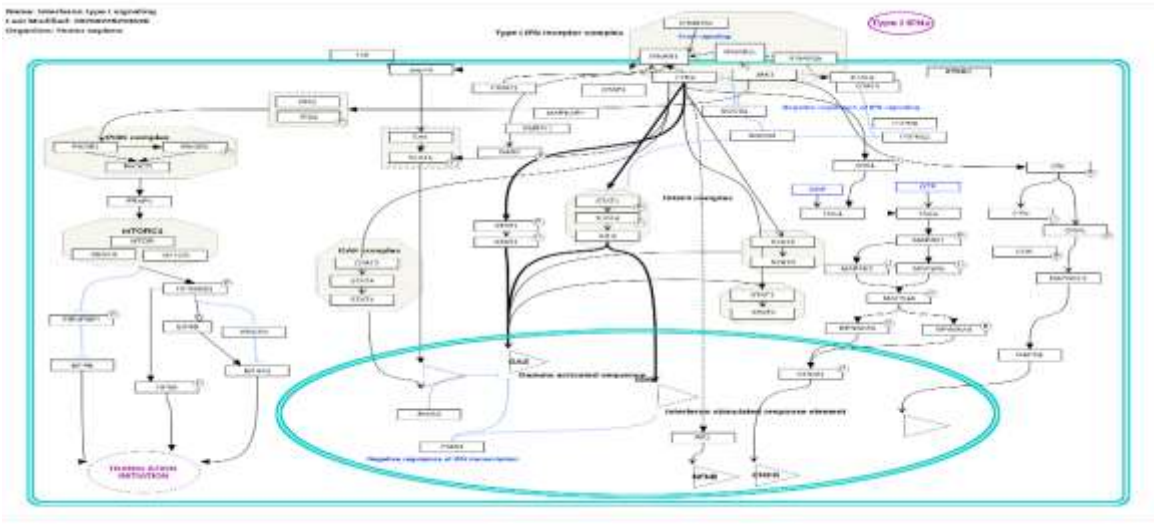

Figure 4. Interferon type I signaling pathway diagram (WP585).

EPO Receptor Signaling

Another enriched pathway was Erythropoietin (EPO) receptor signaling (Figure 5), known for its neuroprotective and anti-apoptotic effects. This pathway involves key regulators, such as *STAT5A/B, AKT, JAK2*, and *MAPK,* all of which were either differentially expressed or connected to hub genes in the PPI network. Dysregulation in this pathway could be associated with impaired neurogenesis and synaptic plasticity in ASD.

Enriched Molecular Functions (GO:MF)

Molecular function analysis (Figure 5B) identified significant enrichment in gene products associated with:

- Transcription factor binding.
- Cytokine receptor activity.
- Growth factor binding.
- Protein domain-specific binding.

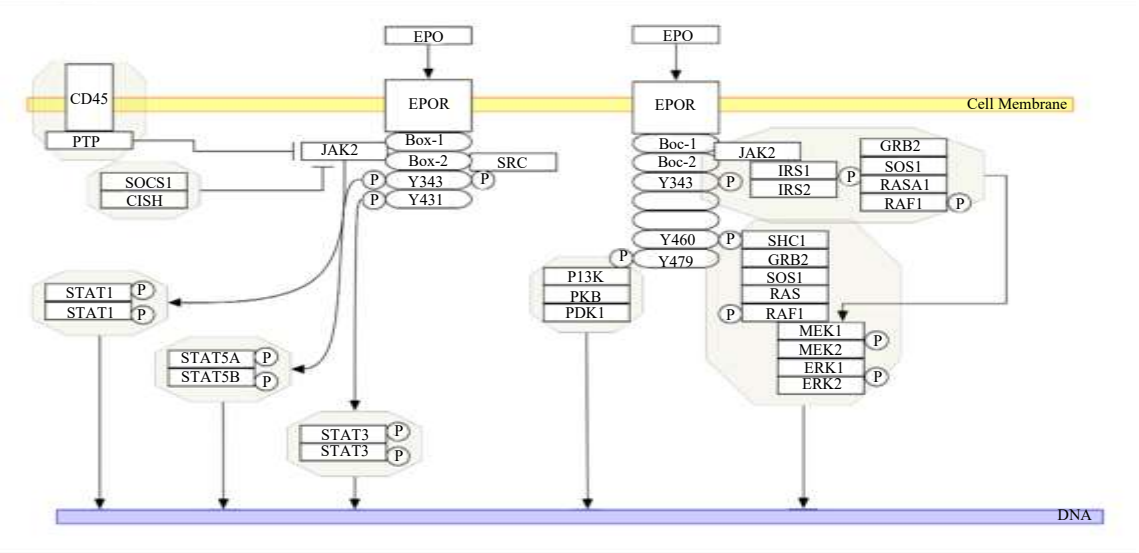

Figure 5. Erythropoietin (EPO) receptor signaling pathway diagram (WP581).

These functions highlight a potential dysregulation of transcriptional control mechanisms, signaling receptor activity, and molecular interactions critical to neurodevelopment and cell survival. Altered transcription factor activity and receptor-mediated signaling are often observed in neurological disorders, particularly in contexts involving neuroinflammation and synaptic dysfunction.

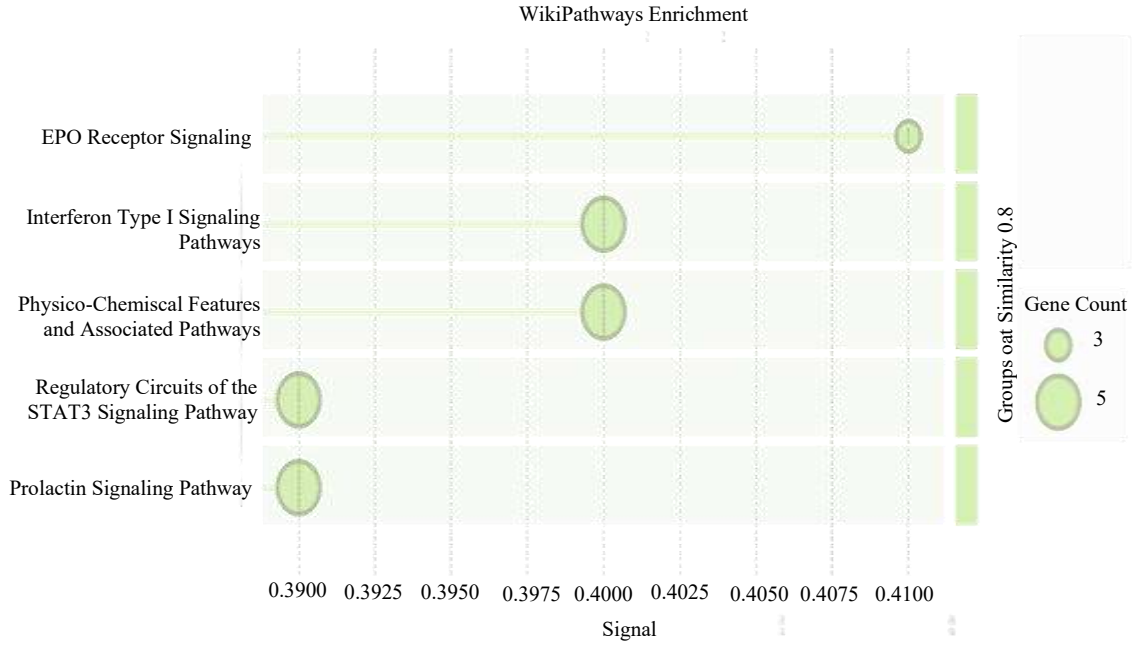

Figure 5B. GO: molecular functions bubble plot: enriched molecular functions from DEGs.

Functional Enrichment (GO Biological Process)

The Gene Ontology (GO) enrichment analysis revealed that the differentially expressed genes were predominantly involved in immune-related biological processes, including regulation of immune response, cytokine signaling, leukocyte differentiation, and cellular response to stimuli (Table 2). These findings indicate a strong association between immune system dysregulation and the molecular pathophysiology of ASD (Figure 6).

Table 2. GO biological process enrichment of differentially expressed genes in ASD.

GO-term	Description	Count in network	Strength	Signal	False discovery rate
GO:0002761	Regulation of myeloid leukocyte differentiation	6 of 123	1.12	0.61	0.0067
GO:0002682	Regulation of immune system process	21 of 1438	0.6	0.59	0.00060
GO:0002684	Positive regulation of immune system process	15 of 874	0.67	0.57	0.0019
GO:0034097	Response to cytokine	14 of 804	0.67	0.55	0.0029
GO:0002376	Immune system process	25 of 2121	0.5	0.53	0.00060
GO:0050776	Regulation of immune response	14 of 844	0.65	0.53	0.0033
GO:0045637	Regulation of myeloid cell differentiation	7 of 214	0.95	0.52	0.0118
GO:0018209	Peptidyl–serine modification	7 of 214	0.95	0.52	0.0118
GO:0023056	Positive regulation of signaling	21 of 1698	0.52	0.5	0.0019
GO:0030316	Osteoclast differentiation	4 of 54	1.3	0.49	0.0239
GO:1902105	Regulation of leukocyte differentiation	8 of 315	0.84	0.48	0.0145
GO:0010611	Regulation of cardiac muscle hypertrophy	4 of 55	1.29	0.48	0.0249
GO:0010647	Positive regulation of cell communication	20 of 1693	0.54	0.46	0.0033
GO:0071345	Cellular response to cytokine stimulus	12 of 711	0.66	0.46	0.0097
GO:1903706	Regulation of hemopoiesis	9 of 410	0.77	0.46	0.0145
GO:0048584	Positive regulation of response to stimulus	23 of 2131	0.46	0.45	0.0029
GO:0071310	Cellular response to organic substance	22 of 2019	0.47	0.45	0.0033
GO:0031347	Regulation of defense response	11 of 638	0.67	0.43	0.0145
GO:0030097	Hemopoiesis	11 of 655	0.66	0.43	0.0158
GO:0010033	Response to organic substance	26 of 2692	0.42	0.42	0.0033
GO:0018105	Peptidyl–serine phosphorylation	6 of 193	0.92	0.42	0.0309

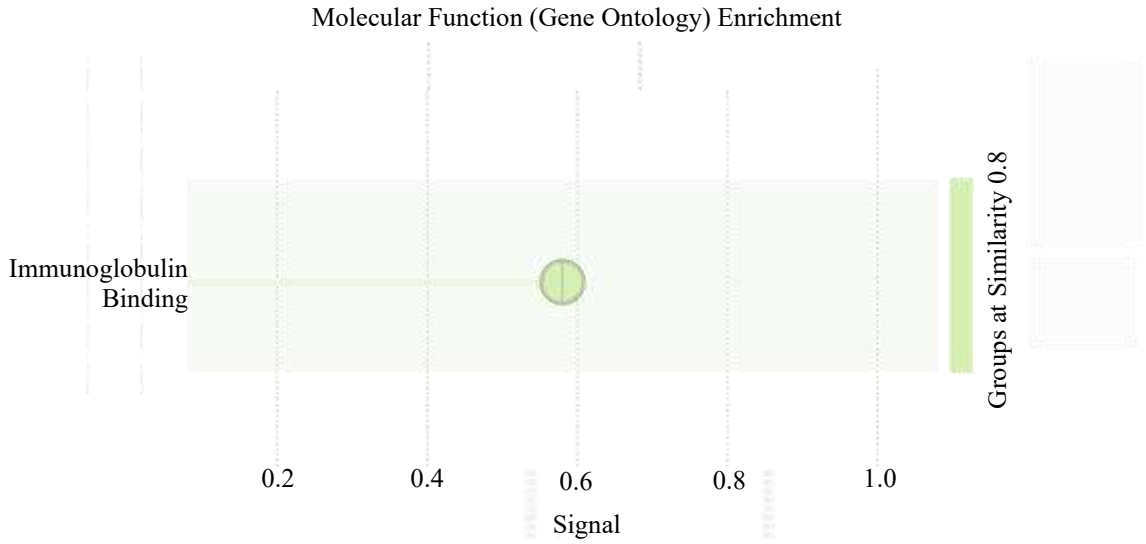

Figure 6. Molecular function (gene ontology) enrichment.

DISCUSSION

The current study aimed to investigate molecular signatures underlying Autism Spectrum Disorder (ASD) by reanalyzing transcriptomic data from the GSE42133 microarray dataset. Focusing on the top 5% of differentially expressed genes (DEGs), network and pathway-based analyses were performed to reveal biologically relevant insights. The findings highlight a complex interplay of immune, endocrine, and neurodevelopmental signaling, particularly involving the Prolactin, STAT3, EPO receptor, Type I

Interferon, and toxicity-related pathways. Together, these pathways suggest a multifactorial mechanism contributing to the etiology of ASD [15–18].

One of the most significant discoveries was the central role of the *STAT3 regulatory circuit.* STAT3 (Signal Transducer and Activator of Transcription 3) is a transcription factor activated by cytokines and growth factors such as interleukin-6 (IL-6) and prolactin. Once activated, STAT3 translocates to the nucleus and regulates genes involved in inflammation, synaptic plasticity, and survival. In the STRING network, STAT3 consistently appeared as a central hub across both medium and high confidence levels. This aligns with prior studies implicating STAT3 in glial activation, microglial over-response, and impaired synaptic pruning – all of which contribute to ASD neurobiology [19].

Closely tied to STAT3 is the Prolactin signaling pathway, which was also enriched in this dataset. Though classically involved in lactation, prolactin, and its receptor (PRLR) are now recognized for their roles in brain function, including social bonding, stress response, and maternal behavior. The presence of PRLR, JAK2, and STAT5B in the DEG list, and their network centrality in STRING, supports a broader role for prolactin in neural regulation. Dysregulation of this pathway may underlie social impairments and emotional dysregulation in ASD [20].

Notable enrichment was also found in genes linked to the Erythropoietin (EPO) receptor signaling pathway. While EPO is traditionally known for hematopoietic function, recent studies show its neuroprotective and anti-inflammatory properties in the brain. Genes, such as EPOR, JAK2, and PIK3CG, suggest that ASD-related brains may have impaired endogenous neuroprotection. Animal models of neurodevelopmental disorders have shown that exogenous EPO can restore synaptic integrity and reduce neuroinflammation, supporting the findings of this study [21].

The Type I Interferon signaling pathway, best known for its antiviral role, was also significantly enriched. Genes, like STAT1, IRF7, and ISG15, were upregulated, indicating a persistent immune activation state in ASD brains. Chronic interferon signaling is known to impair neuronal maturation, glial function, and synaptic plasticity, potentially leading to altered neurodevelopment. Evidence from both human and mouse models supports the theory that maternal immune activation during pregnancy may elevate interferon levels and contribute to ASD risk [22].

In addition, upregulation of genes associated with toxic stress response was observed, including CYP1A1, GPX1, GSTM1, and SOD1. These genes are involved in detoxification and oxidative stress mitigation, and their expression suggests the ASD brain may experience heightened oxidative burden. The gene–environment interaction hypothesis in ASD proposes that certain individuals may be genetically less efficient at handling environmental toxins, leading to developmental vulnerability [23].

From a systems biology perspective, the STRING interaction network clarified how these disparate pathways interconnect. Central hubs, such as STAT3, JAK2, and PRLR, emerged as key regulators, linking immune and hormonal axes with neurodevelopment. This supports previous findings that highlight convergence of inflammatory, hormonal, and synaptic dysregulation in ASD [24].

Collectively, the findings reinforce the idea that ASD is not caused by isolated gene mutations but results from network-level dysfunction across interconnected pathways. The interaction between prolactin and STAT3 signaling highlights the neuroimmune–hormonal cross-talk, while the presence of detoxification genes underlines the potential contribution of environmental exposures in genetically susceptible individuals.

Nevertheless, some limitations exist. The dataset used is derived from postmortem brain tissue, which may not fully capture early developmental molecular dynamics. Additionally, microarray platforms lack resolution for alternative splicing and non-coding RNA regulation. Despite these limitations, the

use of high-confidence DEG filtering and comprehensive network enrichment provides valuable insight into ASD transcriptomics [25].

CONCLUSION

This study provides a systems-level insight into the molecular alterations associated with Autism Spectrum Disorder (ASD) by reanalyzing the GSE42133 microarray dataset. Through stringent filtering of the top 5% differentially expressed genes and pathway enrichment via STRING, central roles for prolactin signaling, STAT3 circuits, interferon response, EPO receptor activity, and oxidative stress pathways were identified. These results support a multifactorial model of ASD where neuroimmune dysregulation, hormonal imbalance, and environmental stress responses converge to shape atypical neurodevelopment. Key hubs, such as STAT3 and PRLR, consistently highlighted in both network confidence levels, may serve as promising biomarkers or therapeutic targets. While this study is limited by the nature of postmortem data, it reinforces the utility of transcriptomic reanalysis in advancing the understanding of ASD's complex biology.

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