

NDS: A Novel Deep Learning-Based Systems Biology Framework for Identifying Prognostic Biomarkers in Hepatocellular Carcinoma[†]

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Abstract

Hepatocellular carcinoma (HCC) is an aggressive liver cancer requiring reliable biomarkers, while current approaches are limited by high-dimensional data and complex nonlinear gene interactions. In this study, differentially expressed genes were identified and fed to a deep autoencoder to reduce dimensionality, capture nonlinear interactions, and extract informative latent features. Predictive gene features were selected through mutual information (MI) ranking and LASSO regression and subsequently evaluated using logistic regression (LR), random forest (RF), and support vector machine (SVM) classifiers with 5-fold cross-validation (CV). The top 50 genes underwent enrichment analyses. Protein–protein interaction (PPI) networks were constructed to identify hub genes, followed by gene–drug interaction, transcription factor analysis, and survival validation. Enrichment analysis highlighted critical pathways involved in metabolic, signaling, and cell-cycle, and viral carcinogenesis. CV showed stable and high performance across classifiers (accuracy = 0.969, F1 $\approx$ 0.97), with RF and SVM achieving the highest AUC values ($\sim$ 0.983 and $\sim$ 0.982). Independent tests showed excellent performance, with RF achieving perfect performance (1.0) across all evaluation metrics, confirming high feature discriminative power. Survival analysis showed that hub genes, including *HSP90AB1*, *TUBA1B*, *PKM*, *H2AZ1*, *YWHAZ*, *ACLY*, *RAN*, *ILF2*, *KPNA2*, and *TXNRD1*, were significantly associated with poor prognosis (HR > 1.5, p < 0.05), correlating with reduced overall, relapse-free, and disease-specific survival in HCC. The integrative novel framework effectively identifies biologically relevant biomarkers, providing insights into HCC mechanisms and potential targets for precision therapy.

Keywords: hepatocellular carcinoma (HCC); differentially expressed genes (DEGs); deep learning (DL); feature selection; autoencoder; biomarkers



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1. Introduction

Hepatocellular carcinoma (HCC) is one of the most aggressive and prevalent primary liver cancers and poses a serious threat to global health. It accounts for approximately 75–85% of all liver cancer cases and ranks as the third leading cause of cancer-related mortality worldwide [1]. Despite advances in surgical resection, liver transplantation, and targeted therapies, substantial improvements in overall survival have not been achieved [2].

Therefore, the identification of reliable molecular biomarkers is essential for improving early diagnosis, prognostic assessment, and personalized treatment strategies.

Differentially expressed gene (DEG) analysis has become a fundamental bioinformatics approach for identifying genes associated with tumor progression. DEG and pathway analyses, as well as the integration of weighted gene co-expression network analysis (WGCNA) with DEGs, have been explored to identify important biomarkers related to HCC [3,4]. Conventional ML techniques, including LR, SVM, RF, and Least Absolute Shrinkage and Selection Operator (LASSO), are commonly used for classification and feature selection in HCC and other diseases [5–7]. More advanced deep learning (DL) approaches, such as the two-stage unsupervised feature selection method Gene-Encoder [8], have also been developed. Furthermore, mutual information (MI)-based methods, such as MIFS-ND [9], have been applied to rank informative and non-redundant genes by maximizing biological signal detection in high-dimensional datasets.

However, current biomarker discovery approaches, including conventional bioinformatics and ML methods, often fail to capture complex nonlinear relationships among genes. Moreover, directly using high-dimensional transcriptomic data for feature selection increases computational burden and may compromise biological interpretability. To address these limitations, we propose the Novel Deep Learning-Based Systems Biology Framework (NDS), an integrated approach for prognostic biomarker identification in hepatocellular carcinoma. NDS utilizes DEGs as input to a deep autoencoder to capture nonlinear gene interactions and generate informative latent representations. MI and LASSO are subsequently employed to identify the most predictive candidate genes, followed by ML classifiers for performance evaluation. Protein–protein interaction (PPI) network analysis is performed to identify hub genes, while enrichment, gene–drug interaction, and transcription factor analyses are conducted to explore their biological relevance. Finally, Kaplan–Meier (KM) survival analysis is used to assess their prognostic significance.

2. Methods

2.1. Dataset Description

The GSE14520 HCC microarray dataset was retrieved from gene expression omnibus (<https://www.ncbi.nlm.nih.gov/geo/> (accessed on 30 June 2025)) and included 445 GPL3921 samples comprising 225 HCC and 220 non-tumor liver tissues.

2.2. Differential Expression Analysis

DEG analysis was performed using GEO2R (<https://www.ncbi.nlm.nih.gov/geo/geo2r/> (accessed on 30 June 2026)) to identify DEGs between HCC and normal tissues using adjusted $p < 0.05$ and $|\log_2\text{FC}| \geq 1$.

2.3. Data Preparation and Preprocessing for DL Model Implementation

The gene expression profile dataset was downloaded from GEO using the `getGEO()` function in R. A conditional $\log_2$ transformation was applied when required, followed by probe annotation and removal of missing values. DEG expression profiles were then extracted from the normalized expression matrix. The resulting matrix was imported into Python (version 3.13), stratified into training–validation (80%) and test (20%) sets, and standardized using z-score normalization:

$$X_{\text{scaled}} = \frac{X - \mu}{\sigma} \quad (1)$$

2.4. Dimensionality Reduction via Autoencoder

Considering the complex and multi-dimensional nature of gene expression data, a nonlinear dimensionality reduction method using an autoencoder neural network was applied. The autoencoder comprises an encoder $f_\theta(\cdot)$ and decoder $g_\phi(\cdot)$:

$$\mathbf{z} = f_\theta(\mathbf{x}) \in R^L, \quad \hat{\mathbf{x}} = g_\phi(\mathbf{z}) \in R^D \quad (2)$$

where $L \ll D$ is the latent dimension, set to 64. The encoder consisted of two fully connected ReLU layers with 256 and 128 neurons, followed by a latent layer of 64 neurons. The decoder mirrored this architecture with fully connected layers of 128 and 256 neurons before reconstructing the input. The model convergence is driven by the mean squared error (MSE) loss minimization while reconstructing:

$$\mathcal{L}(\theta, \phi) = \frac{1}{N} \sum_{i=1}^N \|\mathbf{x}_i - \hat{\mathbf{x}}_i\|_2^2 = \frac{1}{N} \sum_{i=1}^N \|\mathbf{x}_i - g_\phi(f_\theta(\mathbf{x}_i))\|_2^2 \quad (3)$$

The Adam optimization algorithm is applied to do the optimization with a learning rate of 10^{-4} . To prevent overfitting, an early stopping of 100 epochs was used with a 10% validation split of the training data.

2.5. Feature Selection via Mutual Information and Lasso

To identify the most discriminative latent features, a two-step feature selection process consisting of MI and LR was applied to the encoded training data $Z = f_\theta X_{\text{train}}$. In the MI Filter, the mutual information between each latent feature z_j and the class labels y is estimated:

$$I(z_j; y) = \sum_{z_j, y} p(z_j, y) \log \frac{p(z_j, y)}{p(z_j) p(y)} \quad (4)$$

where $p(\cdot)$ denotes probability, distributions estimated empirically. Features are ranked by MI scores, and the top $4 \times k$ candidates are retained (with $k = 50$). On the candidate features, a lasso regularization with 5-fold CV is fit:

$$\min_{\beta} \frac{1}{N} \sum_{i=1}^N \uparrow(y_i, \mathbf{z}_i^\top \beta) + \lambda \|\beta\|_1 \quad (5)$$

where $\uparrow(\cdot)$ is the logistic loss and λ the regularization parameter tuned via CV. The k features with the largest absolute coefficients are selected as final features for classification.

2.6. Classification Models and Evaluation

The selected latent features were classified using LR (L2 regularization), RF (bootstrapping aggregation), and SVM (radial kernel) models. Performance was assessed by 5-fold CV using accuracy, precision, recall, F1-score, and AUC-ROC, followed by evaluation on an independent 20% test set.

2.7. Enrichment Analysis

Gene Ontology (GO) across biological processes (BP), molecular functions (MF), and cellular components (CC) and KEGG enrichment analyses of the top 50 genes were performed using ShinyGO (<http://bioinformatics.sdstate.edu/go/> (accessed on 4 July, 2025)) with FDR < 0.05.

2.8. Protein–Protein Interaction Network Construction for Core Gene Selection

A PPI network of the 50 candidate genes was generated using STRING (<https://string-db.org/> (accessed on 4 July 2025)) with confidence score = 0.4 and visualized in

Cytoscape (version 3.10.3). The top 10 hub genes were identified using CytoHubba based on degree centrality.

2.9. Construction of Gene Regulatory Networks and Drug Prediction

Transcription factors were predicted using ChEA3 (<https://maayanlab.cloud/chea3/> (accessed on 5 July 2025)) and drug–gene interactions were identified using DGIdb (<https://dgidb.org/> (accessed on 5 July 2025)). Networks were visualized in Cytoscape.

2.10. Verification of Prognostic Value of Core Genes

KM Plotter (<https://kmplot.com/analysis/> accessed on 6 July 2025) was used to evaluate the prognostic significance of the hub genes through overall survival (OS), recurrence-free survival (RFS), and disease-specific survival (DSS) analyses using liver cancer mRNA RNA-seq dataset and the auto-selected best cutoff to split patient groups.

3. Results and Discussion

3.1. DEGs Profiling in HCC

Analysis of gene expression profiles of 225 HCC and 220 control samples identified 1380 significant DEGs. It indicates substantial alterations in gene expression profiles between the disease and the healthy state showed in Figure 1.

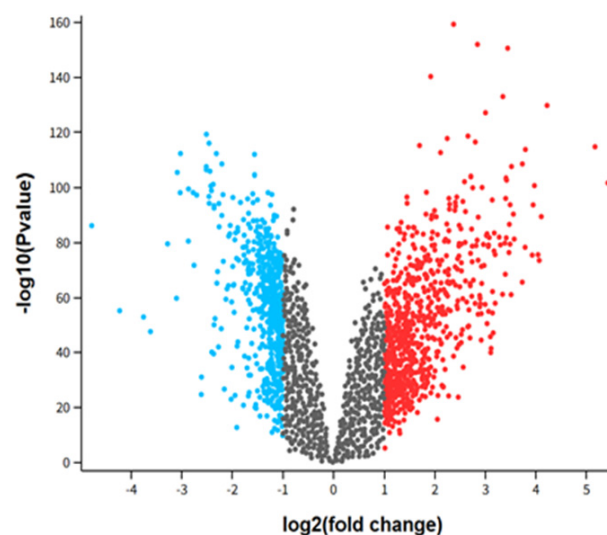


Figure 1. Volcano plot highlighting key upregulated and downregulated DEGs, where red, blue, and gray represent upregulated, downregulated, and non-significant genes, respectively.

3.2. Autoencoder Performance and Latent Representation

The deep autoencoder effectively reduced data dimensionality while preserving biologically relevant information. Training and validation loss curves showed stable convergence across all CV folds without evidence of overfitting (Figure 2a). UMAP visualization of latent embeddings demonstrated distinct clustering of HCC and normal samples, confirming that the learned latent representations captured disease-specific molecular patterns (Figure 2b).

3.3. MI and Lasso-Based Feature Extraction

MI was used to rank latent features, retaining the top 200 candidates. LASSO with CV then selected the 50 most predictive features by enforcing sparsity and removing redundant variables.

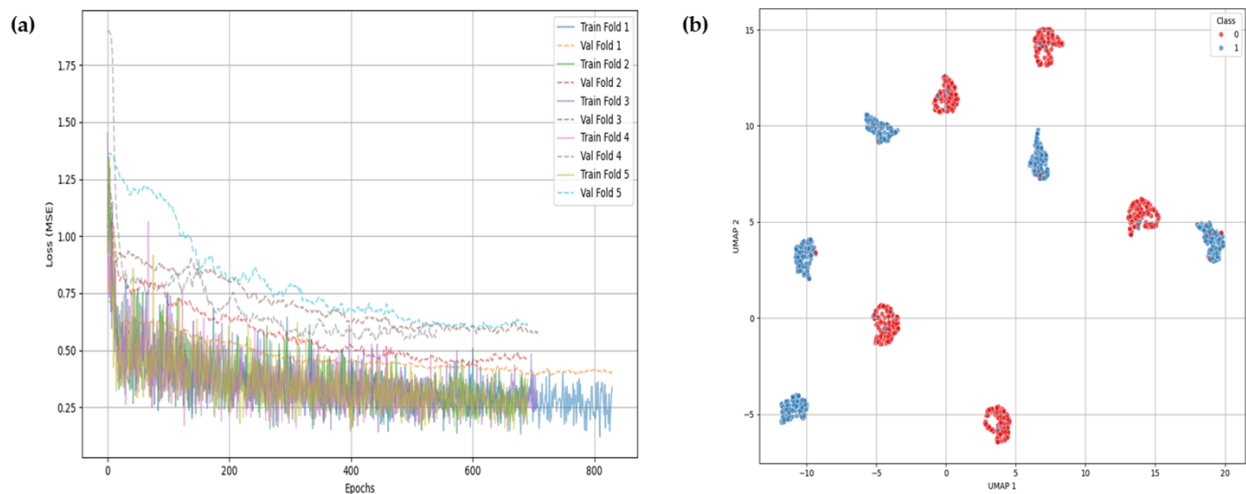


Figure 2. (a) Loss curves for training and validation across MSE epochs for each CV, indicating model convergence and consistent performance. (b) UMAP visualization of the latent space embeddings across CV folds learned by the autoencoder, illustrating the distribution of encoded features.

3.4. Classifier Evaluation Across Cross-Validation Folds

The selected features were evaluated using LR, RF, and SVM classifiers using CV folds across different performance metrics. As shown in Figure 3a, CV demonstrated consistently high performance and stability across classifiers (accuracy = 0.969, F1 $\approx$ 0.97), with RF and SVM achieving the highest AUCs ($\sim$ 0.983 and $\sim$ 0.982). LR also showed competitive performance on certain metrics.

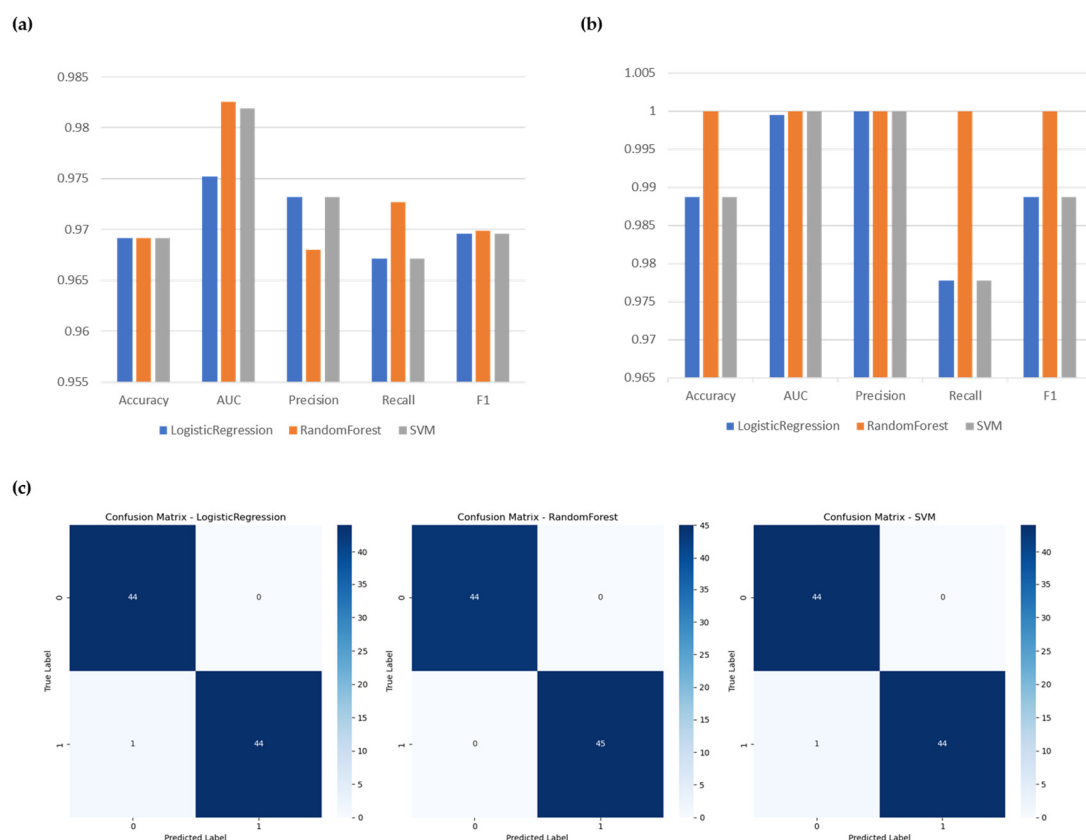


Figure 3. (a) The mean CV metrics scores for LR, RF, and SVM. (b) Final test set evaluation plot. (c) Test set confusion matrices of predicted vs. true labels.

3.5. Final Evaluation on Test Set

Evaluation on the independent test set confirmed excellent model generalizability. RF achieved the highest values (1.0) across all metrics. SVM and LR demonstrated very similar results across all evaluations with values almost equal to 1.0 in AUC and precision, and slightly reduced recall as shown in Figure 3b. The confusion matrices (Figure 3c) show well-calibrated predictions with minimal false positives and false negatives. The findings indicate that the proposed DL-based framework effectively captures molecular signatures capable of accurately distinguishing HCC from normal tissues.

3.6. GO and KEGG Pathways Analysis

GO and KEGG analyses of the top 50 DL-identified genes revealed enrichment in PI3K-Akt signaling, metabolic and protein processing pathways, fatty acid metabolism, cell-cycle regulation, viral carcinogenesis, and related biological processes, molecular functions, and cellular components (Figure 4).

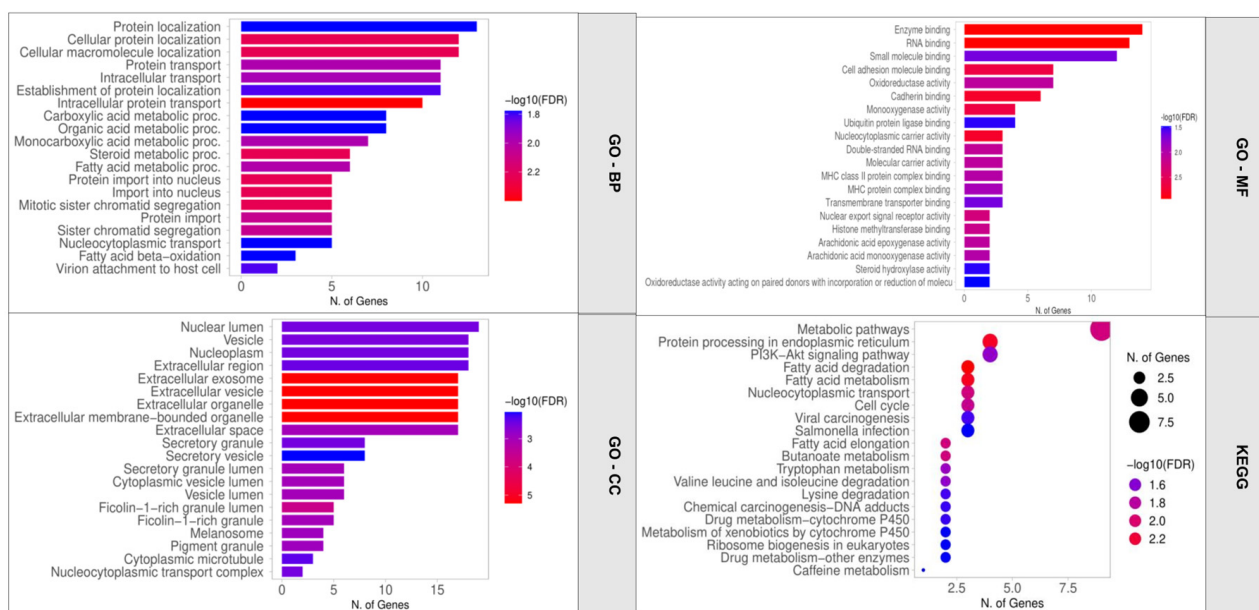


Figure 4. Functional enrichment analysis plots of GO across BP, MF, CC and KEGG.

3.7. Identification of Hub Genes and Regulatory Networks

PPI network analysis identified ten highly connected hub genes: *TXNRD1*, *KPNA2*, *ILF2*, *RAN*, *ACLY*, *YWHAZ*, *H2AZ1*, *PKM*, *TUBA1B*, and *HSP90AB1*. These genes occupy central positions within the interaction network and may represent key regulators of HCC progression (Figure 5a).

Drug–gene interaction analysis revealed substantial therapeutic potential for several hub genes. *TUBA1B* associates with 83 drugs, *HSP90AB1* with 46 drugs, *PKM* with 36, *TXNRD1* with 6, and *H2AZ1* and *ACLY* with 4 drugs each, respectively (Table S1). TF enrichment analysis identified major regulatory factors, including *CENPA*, *PA2G4*, *FOXM1*, *HMGA1*, *HIF1A*, *NFYB*, *TFDP1*, and *ZBTB9*, suggesting complex transcriptional control mechanisms underlying HCC development (Figure 5b,c).

3.8. Prognostic Significance and Biological Relevance

Kaplan–Meier survival analysis demonstrated that elevated expression of all ten hub genes was significantly associated with poor OS, RFS, and disease-specific survival as shown in Table 1. These findings support their potential utility as prognostic biomarkers.

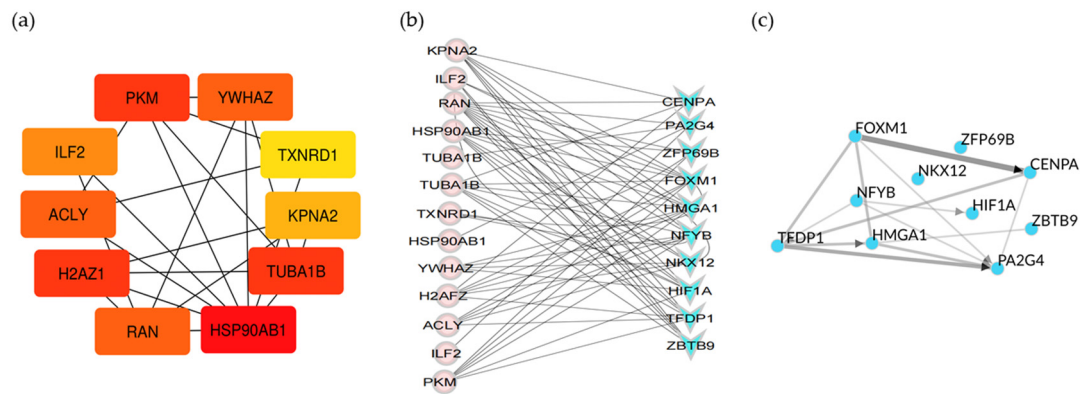


Figure 5. (a) Hub genes identified by PPI and visualized by Cytoscape's Cytohubba. (b) Gene regulatory network of TFs interacting with the hub genes, constructed via Cytoscape. Nodes represent TFs and their target genes. (c) The interaction network between the found TFs generated by ChEA3.

Table 1. KM plot analysis of HR and *p*-values for RFS, OS and DSS analysis.

Genes	RFS		OS		DSS	
	HR	<i>p</i> -Value	HR	<i>p</i> -Value	HR	<i>p</i> -Value
<i>ACLY</i>	0.90	5.90×10^{-1}	1.74	1.10×10^{-2}	1.55	1.00×10^{-1}
<i>H2AZ1</i>	2.16	8.70×10^{-6}	2.29	1.20×10^{-4}	3.10	5.50×10^{-5}
<i>HSP90AB1</i>	1.45	6.10×10^{-2}	1.92	3.50×10^{-3}	2.16	6.10×10^{-3}
<i>ILF2</i>	1.48	3.30×10^{-2}	2.03	4.30×10^{-5}	1.98	2.00×10^{-3}
<i>KPNA2</i>	1.90	1.40×10^{-4}	2.47	1.10×10^{-6}	2.65	4.00×10^{-5}
<i>RAN</i>	2.20	1.60×10^{-4}	2.25	2.40×10^{-6}	2.76	2.90×10^{-6}
<i>PKM</i>	1.44	3.90×10^{-2}	2.07	3.90×10^{-5}	1.98	2.90×10^{-3}
<i>TUBA1B</i>	1.77	1.00×10^{-3}	1.99	7.70×10^{-5}	2.21	3.60×10^{-4}
<i>TXNRD1</i>	1.81	6.30×10^{-4}	1.92	4.90×10^{-4}	2.05	2.90×10^{-3}
<i>YWHAZ</i>	1.27	1.80×10^{-1}	1.76	1.40×10^{-3}	1.82	1.10×10^{-2}

Biologically, the identified genes are involved in several hallmarks of cancer. *TXNRD1* contributes to redox regulation and activation of the Akt/mTOR pathway [10]. *KPNA2* promotes nuclear transport and cell-cycle progression [11]. *ILF2* and *RAN* regulate RNA processing, proliferation, and intracellular trafficking [12,13]. *ACLY* supports lipid metabolism and Wnt/ β -catenin signaling [14], whereas *YWHAZ* participates in cell survival and oncogenic signaling [15]. *H2AZ1* contributes to chromatin remodeling and transcriptional regulation [16]. *PKM* functions in glycolytic reprogramming and tumor metabolism, while *TUBA1B* influences cell-cycle dynamics and treatment resistance. *HSP90AB1* acts as a molecular chaperone that promotes tumor progression, stemness, and metastatic potential [17,18].

The enrichment analyses, network topology, drug associations, and survival results consistently support the biological importance of these genes in HCC. Their involvement in critical pathways such as PI3K-Akt signaling, metabolic reprogramming, cell-cycle regulation, and tumor progression highlights their potential value as diagnostic, prognostic, and therapeutic targets.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/msf2026048002/s1>, Table S1. Drug-Genes Interaction Results.

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review and editing, M.Z.A. and M.M.; visualization, M.Z.A. and M.M.; supervision, M.Z.A.; project administration, M.Z.A. All authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

HCC	Hepatocellular Carcinoma
DEG(s)	Differentially Expressed Gene(s)
DL	Deep Learning
ML	Machine Learning
LR	Logistic Regression
SVM	Support Vector Machine
LASSO	Least Absolute Shrinkage and Selection Operator
MI	Mutual Information
PPI	Protein–Protein Interaction
GO	Gene Ontology
KEGG	Kyoto Encyclopedia of Genes and Genomes
BP	Biological Process
MF	Molecular Function
CC	Cellular Component
TF	Transcription Factor
KM	Kaplan–Meier
OS	Overall Survival
RFS	Recurrence-Free Survival
DSS	Disease-Specific Survival
CV	Cross-Validation

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