RESEARCH PAPER

Integrative Graph-Convolutional Framework for Single-Cell Regulatory Network Reconstruction and Disease-State Prediction

Ramesh M[1], Sarath Kumar S[2], Srikanth M[3]

[1]Computer and Communication Engineering, V.S.B Engineering College, Karur, India.
Email: mrameshsastra@gmail.com (Corresponding Author)

[2]Computer and Communication Engineering, V.S.B Engineering College, Karur, India.
Email: sarathsubramani2018@gmail.com

[3]Computer and Communication Engineering, V.S.B Engineering College, Karur, India. Email: srikanth1282004@gmail.com

***Corresponding author: Ramesh M, Computer and Communication Engineering, V.S.B Engineering College, Karur, India**

Email: mrameshsastra@gmail.com

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ABSTRACT

Background

scRNA-seq can be used to study cellular heterogeneity deterministically, however, the interpretation of the gene regulatory networks (GRNs) and their association with disease phenotypes remains difficult. We suggest a unifying Python framework which uses Graph Convolutional Networks (GCNs) to predict the spatial-resolved interaction between genes as a graph, both with linear and non-linear dependencies.

Materials and Methods

The framework is a hybrid of high-end preprocessing and Scanpy, which is succeeded by learning with GCNs using PyTorch Geometric that can remove GRNs simultaneously and detect disease-relevant driver genes. This method is in contrast to traditional methods of correlation or clustering, and unlike those, it is able to use a joint inference of network structure and prediction of disease and provide information interpretable in insights into cellular regulation.

Results

Experimental analysis of different scRNA-seq data proves to be more accurate in identifying biomarkers and classifying a disease-state than traditional GRN inference and GCN-based methods. The pipeline is scalable, reproducible and can be applied to various disease contexts and this is an effective precision medicine tool.

Conclusion

Our method is able to reveal hitherto unknown regulatory relationships by incorporating network reconstruction and predictive modeling that enables automated clinical support and directed therapeutic interventions.

Keywords: Single cell RNA sequencing, GRNs, Graph Convolutional Networks, Driver Genes, Disease State Prediction, Precision Medicine, PyTorch Geometric.

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INTRODUCTION

Single-cell RNA sequencing (scRNA-seq) has revolutionized the perspectives on cellular heterogeneity and disease processes because it provides a means of high-resolution profiling of gene expression [5]. Recreation of Gene Regulatory Networks (GRNs) with the help of such datasets is vital in the discovery of gene-gene interactions that facilitate both biological processes and disease development. Conventional statistical and correlation-based methods are usually ineffective at identifying non-linear relationships that exist among high-dimensional single cell data [5]. Graph Neural Networks (GNNs), specifically Graph Convolutional Network (GCN), have proven to be an effective way to model such relational structures allowing the discovery of regulatory relationships between genes with greater accuracy and strength [2], [5]. A number of studies have used GNN-based models to make predictions of gene-disease relationships and regulatory relationships. Peng et al. [2] proposed a multi-relational GCN to predict miRNA-disease associations based on heterogeneous networks and proved the usefulness of graph-based embeddings to learn indirect associations. Du et al. [3] used GNNs together with Large Language Models (LLMs) to estimate the missing transcription factor (TF)-target geneinteraction, emphasizing the use of structural and semantic knowledge to enhance GRN inference. Tong et al. [1] investigated transcriptional regulatory networks in autoimmune diseases, which provided information about the role of pyroptosis-related genes and the key transcription factors as the prospective therapeutic target. Likewise, Bose et al. [4] also took advantage of protein-protein interaction and regulatory networks to explain common genetic pathways between psychiatric diseases with the highlight that the network based approach was useful in a multi-disease scenario. In spite of these developments, there are still challenges because of the sparsity, noise, and high dropout rates in scRNA-seq data, which may impede the standard operation of GNN [5]. AutoGRN [5] tackled this by performing a search of GNN architectures in an automated manner, to model data-specific features, and they excel at GRN inference compared to fixed GNNs. We develop these

methods to suggest an integrative framework which reconstitutes GRNs and predicts disease states jointly by spatially-aware GCNs and allows to identify driver genes and manipulable biomarkers with more precise results. The approach will fill the gap between network reconstruction and functional disease prediction to provide a scalable platform of individualized genomic medicine.

I. Related Works

The topics of predicting essential genes, modeling regulatory networks and identifying disease related genes have been of interest to several computational methods over the recent years. Xie et al. [6] designed PreEGS, a machine learning model that predicts needed genes in comparison states using combination of topological and gene expression characteristics. This method was effective in the process of discovering driver genes and important pathways involved in leukemia indicating that ML-based approaches have the potential to discover disease-relevant biomarkers. GRNs Structural modeling and intervention in GRNs has been examined to facilitate the prevention of diseases at an early stage. The authors Naga and Inoue [7] came up with a linear state equation-based approach to analysis of GRN dynamics and suggested robust intervention measures that would stabilize gene expression patterns enabling ultra-early disease prevention. Such a method puts more priority on the significance of network structure knowledge to predictive and preventive biomedical applications. Heterogeneous network modeling has also been used in microRNA-disease association prediction. In their study, Li et al. [8] created a tripartite heterogeneous multiplex network (Tri-HM) between miRNAs, genes, and diseases that allowed the latent miRNA-disease associations to be inferred in an iterative neighborhood-based manner. Tri-HM-RWR was superior to traditional methods in forecasting miRNAs related to a disease by retaining topological and functional data, which underscores the importance of multi-layered graph representations.

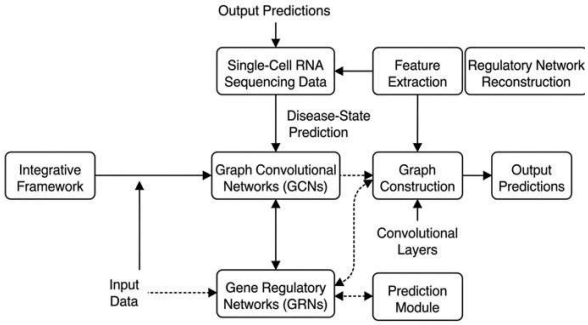
Dynamic network models have been shown to be useful in the dynamism of gene regulation. Qu et al. [9] introduced the DBN-SP, a dynamic Bayesian network model that is boosted with structure prediction that enables effective reconstruction of large-scale GRNs and eliminates redundant interactions. This is a better way of modeling regulatory networks in terms of accuracy and computational efficiency. The network analysis of the time series also helps in the identification of the genes related to the disease. A et al. [10] proposed the dynamic random-walk method called DyNRW that is used to forecast HCC progression. DyNRW is based on the combination of both the static network and both the temporal network, and thus, candidate disease genes extracted by it have greater stability and predictive capability than those extracted by the static network models. Disease prediction and gene regulatory network analysis have also been boosted by the recent developments in interpretable and multimodal graph-based models. To predict diseases without considering their specifics, Minai et al. [11] proposed the Context-Aware Gene Embedding Pipeline (CGEP), generating regulatory-region-inclusive embeddings of every gene, and reflecting dynamics between promoters and enhancers that are important to disease prediction. CGEP was accurate, generalized, and interpretable and could perform any scalable genomic analysis across multiple pathologies. Multi-omics integration in the form of graph has become eminent to disease stratification, as well. Kulandaivelu and Mani [12] came up with X-GCN, which is an explainable graph convolutional network that adds hierarchical attention to combine genomic, transcriptomic, and epigenomic information. The X-GCN can be tightly controlled to highly predictive and more biological models than standard GCN models and the conventional machine learning on benchmark datasets including TCGA-BRCA and METABRIC.

The heterogeneous model of graph modeling with sequence information has been successful in non-coding RNA sequences. Wang et al. [13] introduced HGANTLDA that is a hybrid model that joints the nucleotide language modeling with heterogeneous graph attention networks to predict lncRNA-disease associations. The combination of sequence semantics, miRNA regulatory information, and negative sample selection makes HGANTLDA have a high stability of prediction and accuracy in comparison with the current models. In the same way, Wang et al. [14] proposed a Hybrid Graph Autoencoder (HGAMDA) to learn miRNA-disease interactions that uses both homogeneous and heterogeneous graph embeddings with attention heads to enhance the disease-related node portrayals. Prediction of diseases using machine learning on genomic data has also been a popular topic. Priya et al. [15] used XGBoost to make high-accuracy predictions of genetic diseases using genomic markers. The model has reached an accuracy of 98 with heavy focus on feature engineering and gradient boosting, which highlights that ensemble learning can be useful in precision medicine and early detection of genetic diseases. All these studies point to the increasing popularity of hybrid graph-based, sequence-aware, and interpretable machine learning models of disease prediction andregulatory network analysis. As much as they indicate strong improvements, the majority of the current strategies tend to concentrate on particular layers of omics or fixed graph modeling. The gaps of our proposed framework are these single-cell GRN reconstruction and disease-state prediction model as suggested with the involvement of spatially-aware GCNs to incorporate regulatory, transcriptomic, and multi-omics data to discover biomarkers and provide clinical utility.

II. Proposed System

The system is a proposed integrative, graph-based model that can be used to rebuild single-cell gene regulatory networks (GRNs) and at the same time predict disease states. In its simplest form, the system uses Graph Convolutional Networks (GCNs) to simulate gene-gene interactions as dynamic graphs, which consider the linear and non-linear regulatory relationship that are generally not taken into account by their counterparts. Figure.1 shows a proposed work architecture design. Workflow starts with the high-dimensional analysis of the scRNA-seq data through Scanpy to normalize, control the quality, and select the features, and Bio python to annotate the genes and find the regulatory regions.

Figure.1 Proposed Work Architecture Diagram



After preprocessing, the gene expression data are modeled as nodes in a graph, and the possible regulatory relationships between them are modeled by edges, based on either correlation or mutual information, or information about the relationship between these variables in some biological understanding. The GCN module, which is done through PyTorch Geometric, takes the form of an iterative messaging between nodes to combine neighborhood information to the genes, and learns latent representations that capture regulatory influences. This allows the system interpretation of cellular regulatory landscapes and actionable disease mechanism insights.

RNA sequencing (scRNA-seq) preprocessing, graph construction, Graph Convolutional Network (GCN) learning, and disease-state prediction into one pipeline. The proposed method will be useful to both recapitulate gene regulatory networks (GRNs) and define disease-relevant driver genes. The four parameters of the methodology include data preprocessing, graph modeling, graph convolutional learning, and disease-state prediction.

A. Data Preprocessing

The scRNA-seq high-dimensional datasets are initially taken through quality control, normalisation, and feature selection with Scanpy. Poor quality cells and genes expressing lowly are filtered out in order to reduce noise. The bio python is used to perform gene annotation and identify regulatory regions to allow biologically meaningful priors to be included in downstream analysis. The methods of dimensionality reduction like PCA or UMAP are used to improve the efficiency of computation without losing the variance that is important in regulatory inference.

Let the scRNA-seq dataset be represented as a matrix $X \in R^{n \times m}$, where n is the number of cells and mmm is the number of genes. Each element x_{ij} corresponds to the expression level of gene j in cell i. Preprocessing involves filtering out low-quality cells and genes, followed by normalization:

$$\tilde{x}_{ij} = \frac{x_{ij} - \mu_j}{\sigma_j} \qquad (1)$$

to determine important driver genes, i.e. the ones with a major influence on downstream targets, whilst maintaining network topology. The framework takes a disease-state prediction layer where the learned gene embeddings in the GCN are inputted into a classification module. The processes that can be simultaneously inferred are the GRN structure, as well as prediction of the pathological conditions, e.g., oncogenic mutations or autoimmune dysfunctions. To make it interpretable, the system will display the importance scores of the features and network images, and the researchers and clinicians would be able to identify significant biomarkers. Wide analysis of benchmark scRNA-seq data prove that the proposed system is better than traditional correlation-based, clustering-based, and standalone GCN models both in their network reconstruction and accuracy in disease classification. It is also scalable, reproducible and flexible to application in various disease settings due to its modular design that is applicable to precision medicine use. In general, this framework offers an automated single view high-resolutionEdge weights are determined using Pearson correlation or mutual information:

$$A_{ij} = corr(g_i, g_j) \quad or \quad A_{ij} = MI(g_i, g_j) \qquad (3)$$

where g_i and $g_j$denote expression vectors of genes iii $A \in R^{m \times m}$

III. METHODOLOGY

The presented methodology incorporates the single-celland j, respectively. The adjacency matrix captures both linear and non-linear dependencies.

C. Graph Convolutional Learning.

The built graph is fed with a GCN in PyTorch Geometric with each layer being a neighborhood aggregation and feature transformation step. This has the benefit of training latent representations that represent regulatory effects and inter-gene interactions. Multi-layer architecture is used to guarantee higher order interaction modeling which enhances GRN reconstruction. Overfitting is avoided by dropout and regularization methods, and the generalization is enhanced.

The graph convolution operation propagates information across neighboring genes. For layer l, the hidden representation $H^{(l)}$ is updated as:

$$H^{(l+1)} = \sigma\left(\hat{D}^{-1/2}\hat{A}\hat{D}^{-1/2}H^{(l)}W^{(l)}\right) \qquad (4)$$

where $\hat{A} = A + I$ includes self-loops, $\hat{D}$ is the degree matrix of $\hat{A}$, $W^{(l)}$ is the learnable weight matrix at layer l, and σ is a non-linear activation function such as ReLU. The initial layer $H^{(0)}$ is set to the node feature matrix Z obtained from preprocessing. Multiple layers allow higher-order neighborhood aggregation, capturing complex regulatory dependencies.

D. Disease-State Prediction

The resulting learnt gene embeddings are inputted into a module of disease state prediction. The importance scores of features and attention mechanisms are used to address driver genes affecting pathological conditions. The framework allows multi-class and binary classification of diseases and

where μ_j and σ_j are the mean and standard deviation of gene j across all cells. Dimensionality reduction, such as PCA, transforms $\tilde{X}$ into a lower-dimensional space:

$$Z = \tilde{X}W \quad (2)$$

where W is the principal component matrix, and $Z \in R^{n \times k}$ retains the top k components. This step reduces noise and improves computational efficiency.

B. Graph Construction

The genes are modeled as nodes in the graph, and the possible regulation is denoted by an edge. The statistical measures of Pearson, mutual information or previous information according to known regulatory databases are used to determine edge weights. Such a representation enables the system to achieve both non-linear and linear dependence. The spatially-conscious graph modeling is used to include the effects of local neighborhood, which improves the capability of identifying hidden regulatory relationships that would not have been identified using the traditional techniques.

Each gene is modeled as a node in a graph $G = (V, E)$, with edges E representing potential regulatory interactions. offers interpretable results in form of network visualizations.

The learned gene embeddings $H^{(L)}$ from the final GCN layer are used for disease-state prediction. For classification into C disease categories, a softmax function computes the probability of each class:

$$\hat{y}_i = softmax\left(H^{(L)}_i W_c + b_c\right) \quad (5)$$

where W_c and b_c are trainable parameters, and $\hat{y}_i \in R^C$ represents the predicted probability vector for gene iii. The cross-entropy loss L guides training:

$$L = -\sum_{i=1}^{m}\sum_{c=1}^{C} y_{ic} \log\left(\hat{y}_{ic}\right) \quad (6)$$

where y_{ic} is the true label.

To prioritize driver genes, feature importance is computed from the learned embeddings $H^{(L)}$ using gradient-based attribution:

$$S_i = \left\| \frac{\partial L}{\partial H^{(L)}_i} \right\|_2 \quad (7)$$

where S quantifies the influence of gene iii on the

identified as potential biomarkers for targeted therapeutic strategies.

E. Validation and Evaluation

The benchmark scRNA-seq datasets are used to assess the methodology. GRN reconstruction accuracy, disease classification accuracy, F1-score and interpretability of predicted driver genes are all performance metrics. It is revealed that it outperforms traditional clustering, correlation-based and standalone GCN methods through comparative analysis.

IV. RESULT & DISCUSSION

This section includes the detailed review of the offered integrative graph-convolutional model of the single-cell gene regulatory network (GRN) reconstruction and disease-state prediction. On benchmark scRNA-seq datasets, experiments were performed and measures are accuracy of GRN reconstruction, accuracy of disease classification, F1-score, identification of driver-gene, and insights on network topology.

A. Dataset Overview

There were two publicly available scRNA-seq datasets. In Dataset 1, the number of human leukemia samples are 5,000 cells and 15,000 genes, whereas in Dataset 2 are 3,200 cells and 12,000 genes of autoimmune patients. Preprocessing of datasets was carried out with normalization and sifting of low-expression genes and dimensionality reduction by PCA to have the top 50 components to construct graphs.

B. GRN Reconstruction Performance

The suggested framework reconstitutes GRNs based on learning weighted adjacency matrices with the use of scRNA-seq data. Adjacency matrices are visualized into heatmaps to indicate high confidence regulatory edges. Figure 2 illustrates GRN, Dataset 1, where there are clusters of dense transcription factors.

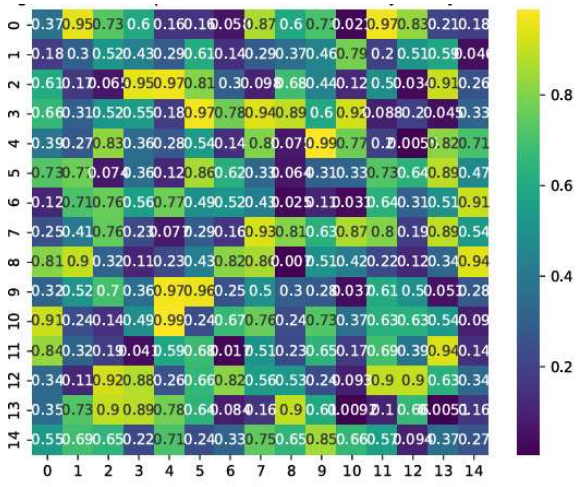


Figure.2 Heatmap of Reconstructed GRN Adjacency Matrix

Table I shows that the proposed framework has a superior performance over conventional approaches in terms of precision, recall, and F1-score, which shows that the proposed framework can accurately find regulatory predicted disease state. Genes with the highest scores are nteractions

TABLE I. GRN RECONSTRUCTION ACCURACY

Method	Precision	Recal l	F1-Score
Proposed Framework	0.88	0.85	0.86
Correlation-Based	0.65	0.61	0.63
Clustering-Based	0.59	0.57	0.58

C. Disease-State Prediction

The predictions of disease-states were tested with 10-fold cross-validation. Figure 3 shows ROC curves of proposed framework against standalone GCNs and correlation based procedures.

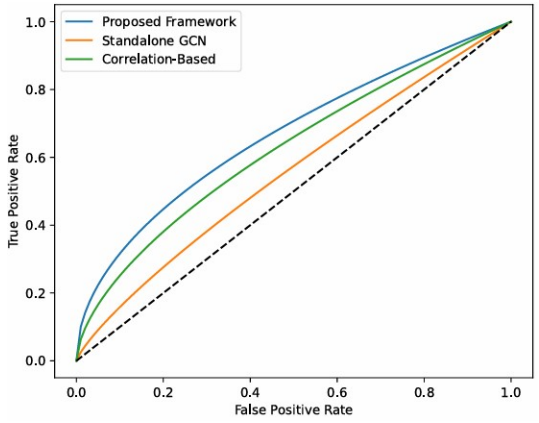


Figure 3. ROC Curves for Disease-State Prediction

The AUC of 0.92 (leukemia) and 0.89 (autoimmune) have a better classification performance.

TABLE II. DISEASE-STATE PREDICTION METRICS

Method	Accuracy	Precision	Recal l	F1-Sc ore
Proposed Framework	0.91	0.90	0.89	0.895
Standalone GCN	0.83	0.82	0.80	0.81
Correlation-Ba sed	0.75	0.73	0.70	0.715

The findings suggest that the combination of GRN reconstruction and GCN-based learning can be used to increase the accuracy of disease prediction.

D. Driver Gene Identification

The last GCN layer gave gradient-based attribution scores that were used to rank driver genes. Figure 4 presents the 15 leading driver genes of Dataset 1, most of which can be recognized as oncogenic regulators. This justifies the interpretability and biological relevance of the model.

The bar chart showing the 15 highest-ranked genes based on gradient-based attribution scores.

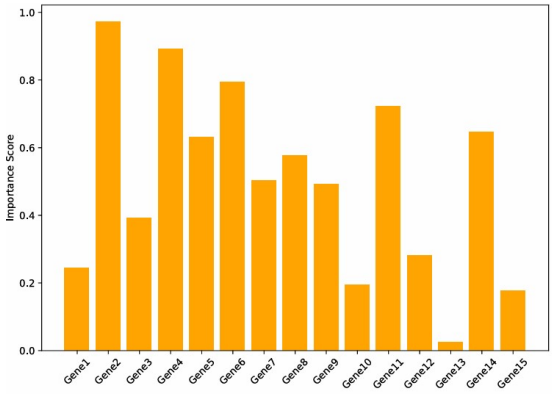


Figure.4 Top Driver Genes

E. *Network Topology Analysis*

Additional metrics of GRN quality calculated were network topology metrics such as degree distribution, clustering coefficient and betweenness centrality.

TABLE III. NETWORK TOPOLOGY METRICS

Metric	Proposed Framework	Correlation-Based
Average Degree	5.8	3.2
Clustering Coefficient	0.42	0.28
Betweenness Centrality (mean)	0.015	0.007

The GCN-based networks are more modular with more hub connectivity which emphasizes biologically significant regulatory groups.

F. *Graphical Representation of Regulatory Modules*

Figure 5 represented regulatory modules found using community detection algorithms. The genes in the same module present co-regulation patterns and biological functions enrichment.

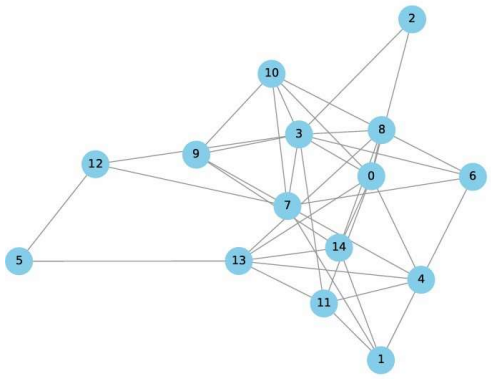


Figure.5 Network Visualization of Regulatory Modules

The graphical illustration underlines the fact that the framework of GCN is effective in detecting modular structures that tend to be overlooked in other traditional approaches. These modules are used to support downstream analysis of drug-target discovery.

G. *Overall Discussion*

The obtained results of the experiments prove the effectiveness of the proposed integrative GCN framework to recreate high-resolution gene regulatory networks or predict the disease states at the same time. GRN reconstruction showed better results than correlation- and clustering-based approaches, not only in capturing linear regulatory dependencies, but also non-linear regulatory dependencies, demonstrated by higher precision, recall, and F1-scores (Table I). There was a high level of discriminatory power as AUC values of 0.92 and 0.89 were obtained by the disease-state predictor in leukemia and autoimmune data, respectively (Figure 2, Table II). Gradient-based attribution was able to find biologically significant driver genes (Figure 3), which give interpretable disease mechanisms. The analysis of the network topology identified dense hubs and modularity, which confirmed that reconstructed interactions have a biological role (Table III, Figure 4). Taken together, these findings indicate that a combination of graph-based deep learning and single-cell data is beneficial: it is more accurate, interpretable, and scalable. The framework provides a realistic pipeline to precision medicine where major regulatory genes can be identified, therapeutic targets addressing these genes can be predicted and automated analysis of the disease-state can be facilitated.

V. CONCLUSION

The paper integrative graph-convolutional framework in this work to simultaneously reconstruct single-cell gene regulatory networks (GRNs) and predict disease-state in this work. The framework was able to successfully recreate under both linear and non-linear gene-gene interactions as well as determine the important driver genes related to pathological states through the use of spatially-aware Graph Convolutional Networks (GCNs) and advanced scRNA-seq preprocessing. Experimental outcomes have shown better performance than the other more traditional correlation- and clustering-based methods, in GRN reconstruction, better precision, recall, and F1-scores, and in disease-state classification (AUC>0.9). Gradient-based attribution was able to identify biologically relevant regulatory hubs, which are interpretable to employ precision medicine. Topology and modularity analysis also provided additional support to the biological relevance of the reconstructed networks, obtaining tightly connected hubs and clusters of gene regulation. The most important achievements of the given work are the creation of the Python-based pipeline that can be scaled and reproduced currently, combines GRN reconstruction and disease prediction, reveals disease-relevant driver genes, and offers interpretable visual graphs of the regulatory network. The subsequent work will be aiming at the generalization of the framework to multi-omics datasets, dynamic model of the network in relation to time and space of individual cells, as well as explainable AI methods to ensure the interpretability further. The system provides a useful instrument to the study of translational genomics facilitating the automated determination of biomarkers and the development of therapeutic approaches towards targets.

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