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Synergy Prediction of Lung Cancer Targeted Drugs Fusing Transcriptomic Data and Graph Networks

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Abstract: *Lung cancer remains the leading cause of cancer-related mortality worldwide, necessitating the development of advanced therapeutic strategies. Combination therapy, the administration of two or more drugs, has emerged as a critical approach to overcome drug resistance and enhance therapeutic efficacy compared to monotherapy. However, experimental screening of all possible drug combinations is cost-prohibitive and time-consuming due to the combinatorial explosion of potential pairs. In this study, we propose a novel deep learning framework that predicts drug synergy specifically for lung cancer by fusing transcriptomic data with graph neural networks. The model integrates the chemical structural information of drugs, captured through graph convolutional networks, with the genomic features of cancer cell lines derived from high-throughput transcriptomic profiles. By employing an attention-based fusion mechanism, the architecture dynamically weighs the importance of molecular substructures and gene expression signatures to predict synergy scores. We evaluated our model on large-scale benchmark datasets, demonstrating superior performance over state-of-the-art machine learning baselines. The results indicate that incorporating cell-line-specific transcriptomic data significantly improves prediction accuracy, offering a promising computational tool to accelerate the discovery of effective combination therapies for non-small cell lung cancer and small cell lung cancer subtypes.*

Keywords: *Drug Synergy, Graph Neural Networks, Transcriptomics, Deep Learning, Lung Cancer*

1.INTRODUCTION

Lung cancer accounts for the highest number of cancer deaths globally, presenting a severe challenge to public health systems and oncological research. The disease is histologically categorized into non-small cell lung cancer, which constitutes the majority of cases, and small cell lung cancer, known for its aggressive nature and rapid metastasis. While targeted therapies focusing on specific genetic mutations such as EGFR, ALK, and ROS1 have improved survival rates, the development of acquired drug resistance remains a pervasive issue. Patients frequently relapse after an initial response to kinase inhibitors, driven by secondary mutations or the activation of bypass signaling pathways [1]. Consequently, the paradigm of treatment has shifted towards combination therapies, where synergistic drug interactions are leveraged to achieve greater therapeutic efficacy at lower doses, thereby minimizing toxicity and delaying the onset of resistance. The identification of synergistic drug combinations has traditionally relied on high-throughput screening assays. These wet-lab experiments involve testing thousands of compound pairs across various cell lines, a process that is both resource-intensive and financially burdensome. Given the vast chemical space of approved and investigational drugs, exhaustively testing every possible pair is practically impossible. This bottleneck has catalyzed the integration of computational biology and artificial intelligence into the drug discovery pipeline. Computational models capable of predicting the synergy score of a drug pair based on their chemical properties and the biological context of the cancer cell have become a focal point of recent research [2]. Early computational approaches utilized standard machine learning algorithms, such as Random Forests and Support Vector Machines, relying on handcrafted descriptors for drugs and cell lines. However, these methods often fail to capture the complex, non-linear interactions inherent in biological systems. The advent of deep learning has enabled the extraction of latent features from high-dimensional data, leading to significant improvements in prediction accuracy. Despite these advancements, many existing models treat drugs as simple string sequences or fixed fingerprints, neglecting the rich topological information embedded in their molecular graphs. Furthermore, the biological context is often underrepresented, with models frequently omitting the granular gene expression profiles that dictate how a specific lung cancer cell line responds to pharmacological intervention [3]. This paper introduces a comprehensive deep learning framework designed to address these limitations. We propose a multi-modal architecture that fuses graph-based drug representations with transcriptomic profiles of lung cancer cell lines. By representing molecules as graphs, where atoms are nodes and chemical bonds are edges, we utilize Graph Neural

Networks to learn expressive molecular embeddings. Simultaneously, we process gene expression data to capture the transcriptomic state of the cell, identifying key biomarkers associated with drug sensitivity. The fusion of these two modalities allows the model to predict synergy with high precision, specifically tailored to the heterogeneity of lung cancer. This study provides a rigorous evaluation of the proposed method, benchmarking it against existing state-of-the-art algorithms and analyzing the biological relevance of the findings [4].

2. Related Work

2.1 Machine Learning in Drug Synergy Prediction

The application of machine learning to drug synergy prediction has evolved significantly over the past decade. Initial efforts focused on utilizing physicochemical properties of drugs and baseline genetic features of cell lines as input for classical regression models. For instance, the Random Forest algorithm was widely employed to map drug descriptors and genomic mutations to synergy scores (e.g., Loewe Additivity or Bliss Independence scores). These shallow models demonstrated that computational prediction was feasible but often struggled with generalization across unseen cell lines or novel drug classes. Research in this era established the fundamental hypothesis that the structural compatibility of drugs and the genetic background of the target cells are the primary determinants of synergistic potential [5]. Subsequent studies began to incorporate more diverse data types, including protein-protein interaction networks and pathway activity scores. The integration of these biological networks aimed to provide a systemic view of how drugs modulate cellular functions. However, the high dimensionality of biological networks often led to the curse of dimensionality, where the number of features far exceeded the number of available training samples. Feature selection techniques and dimensionality reduction methods, such as Principal Component Analysis, were standard preprocessing steps. Despite these optimizations, the predictive power of traditional machine learning models plateaued, primarily due to their inability to model the intricate, non-linear dependencies between drug targets and downstream signaling cascades [6].

2.2 Deep Learning and Graph Neural Networks

The resurgence of neural networks brought about a paradigm shift in bioinformatics. Deep neural networks allowed for end-to-end learning, where feature extraction and prediction are performed simultaneously. DeepSynergy was one of the pioneering frameworks that utilized a feed-forward neural network to predict synergy scores, taking drug and cell line features as inputs. This model significantly outperformed traditional machine learning methods, highlighting the capacity of deep learning to handle the complexity of pharmacological data. Following this, various

architectures were explored, including autoencoders for feature compression and recurrent neural networks for processing sequential data representations of drugs, such as SMILES strings [7]. A critical limitation of sequence-based drug representations is the loss of structural topology. Molecules are naturally defined by their graph structures, and linearizing them into strings can obscure the spatial arrangement of atoms and functional groups essential for binding affinity. To address this, Graph Neural Networks (GNNs) and their variants, such as Graph Convolutional Networks (GCNs) and Graph Attention Networks (GATs), have been adapted for molecular property prediction. In the context of drug synergy, GNNs operate by passing messages between neighboring atoms, iteratively updating node representations to aggregate local and global structural information. Recent studies have demonstrated that GNN-based encoders yield superior drug embeddings compared to fixed fingerprints, leading to more robust synergy predictions [8].

2.3 Transcriptomic Data Integration

While advanced drug encoders have improved model performance, the representation of the biological system remains a challenge. Early deep learning models often used binary mutation data or copy number variation as the sole descriptors of cell lines. However, gene expression data (transcriptomics) offers a more dynamic snapshot of cellular activity, reflecting the actual protein abundance and pathway activation states. Integrating high-dimensional transcriptomic data requires sophisticated encoding strategies to filter noise and extract relevant signals. Recent architectures have employed multimodal deep learning, where one branch of the network processes drug graphs and another processes gene expression profiles. The effective fusion of these distinct data modalities is critical. Approaches utilizing tensor factorization and attention mechanisms have been proposed to align the latent spaces of chemical and biological features, ensuring that the model learns the context-specific efficacy of drug combinations [9].

3. Methodology

3.1 Data Acquisition and Preprocessing

The foundation of our predictive framework is a robust dataset integrating pharmacological and genomic information. We sourced drug combination response data from the DrugComb database, filtering specifically for lung cancer cell lines. This dataset provides standardized synergy scores, calculated using the Loewe Additivity, Bliss Independence, and HSA models. For consistency, we utilized the Loewe synergy score as the primary target variable, as it is widely accepted for evaluating drug interactions. The dataset comprises over 50,000 experiments covering diverse drug pairs tested against a panel of non-small cell lung cancer and small cell lung cancer cell lines.

For the pharmaceutical agents, we retrieved the Simplified Molecular Input Line Entry System (SMILES) strings from the PubChem and DrugBank databases. The SMILES strings serve as the raw input for constructing molecular graphs. We performed canonicalization of the SMILES strings to ensure unique representations for each compound. For the biological component, we obtained baseline gene expression profiles from the Cancer Cell Line Encyclopedia (CCLE) and the Genomics of Drug Sensitivity in Cancer (GDSC) project. The transcriptomic data, measured in Reads Per Kilobase of transcript per Million mapped reads (RPKM), was log-transformed and normalized to handle the varying scales of gene expression. To reduce the dimensionality of the gene expression feature space, we selected the top 1,000 genes based on variance across the lung cancer cell lines, focusing on genes most variable and therefore likely to be informative of differential drug response [10].

3.2 Graph-Based Drug Encoding

To effectively capture the structural properties of the drugs, we implemented a Graph Convolutional Network (GCN). In this framework, each drug is represented as a graph where atoms function as nodes and chemical bonds as edges. Each node is initialized with a feature vector describing the atom type, degree, formal charge, hybridization, and aromaticity. The adjacency matrix represents the connectivity of the molecule. The GCN operates through a message-passing mechanism where the representation of a node is updated by aggregating information from its immediate neighbors. This process is repeated over multiple layers, allowing information to propagate across the molecular graph, thereby capturing both local chemical environments and global molecular topology. The specific propagation rule involves normalizing the adjacency matrix to prevent numerical instabilities and applying a non-linear activation function, such as the Rectified Linear Unit (ReLU), after each convolution operation. Following three layers of graph convolution, a global pooling operation (specifically, global max pooling) is applied to aggregate the node features into a single fixed-length vector representing the entire drug molecule. This results in a dense embedding vector that encapsulates the structural and physicochemical characteristics of the drug, suitable for concatenation with other feature modalities. This graph-based approach addresses the limitations of fingerprint-based methods by learning representations directly from the molecular structure [11].

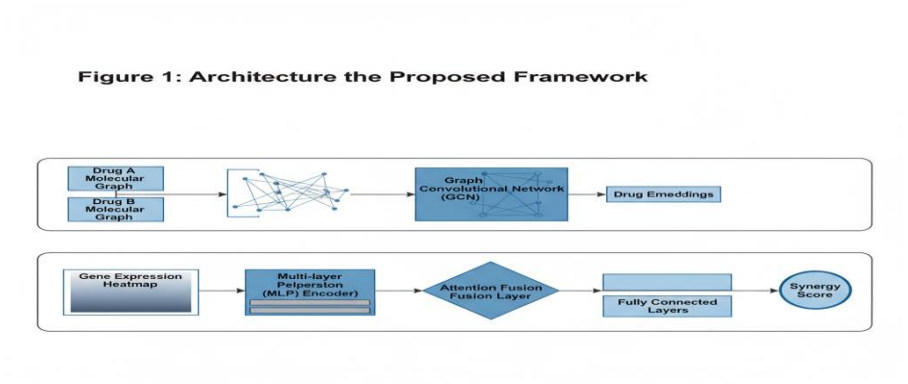

Figure 1: *Architecture of the Proposed Framework*

3.3 Transcriptomic Feature Extraction

The transcriptomic data, consisting of the expression levels of the selected 1,000 landmark genes, is processed through a specialized sub-network. Since gene expression data is inherently noisy and high-dimensional, we employ a Denoising Autoencoder (DAE) architecture for feature extraction. The DAE is pre-trained to reconstruct the original gene expression profiles from corrupted inputs, forcing the network to learn robust, low-dimensional representations of the cellular state. The encoder part of the DAE consists of three fully connected layers with decreasing unit counts, effectively compressing the 1,000-gene input into a latent vector of size 128.This latent vector serves as the cell line signature. By utilizing an autoencoder rather than simple principal component analysis, the model captures non-linear relationships between co-expressed genes, which often correspond to biological pathways relevant to drug mechanism of action. This cell line embedding is crucial for the model to distinguish why a specific drug combination might be synergistic in an EGFR-mutant cell line but antagonistic in a KRAS-mutant line. The weights of the encoder are fine-tuned during the supervised training phase of the synergy prediction task [12].

3.4 Feature Fusion and Prediction

The core innovation of our methodology lies in the fusion of the drug and cell line representations. Simply concatenating the vectors often fails to capture the intricate interdependencies between the chemical agents and the biological context. To address this, we implemented an Attention-based Fusion Mechanism. The embeddings of Drug A, Drug B, and the Cell Line are projected into a shared semantic space. An attention matrix is computed to determine the relevance of specific drug substructures to the cell line features. This allows the model to dynamically weight the interaction between the drugs and the cellular environment.

The fused feature vector is then passed through a series of fully connected layers, which serve as the regression head of the network. We utilize dropout regularization between these layers to prevent overfitting, a common challenge in biological datasets with high dimensionality relative to the number of samples. The final layer consists of a single neuron with a linear activation function, outputting the predicted Loewe synergy score. The entire network is trained end-to-end, optimizing the weights of the GCN, the transcriptomic encoder, and the fusion layers simultaneously [13].

3.5 Training Protocol

The model was implemented using the PyTorch framework. We employed the Mean Squared Error (MSE) as the loss function, quantifying the difference between the predicted synergy scores and the ground truth values derived from the wet-lab experiments. The dataset was split into training, validation, and testing sets in an 80:10:10 ratio. To ensure rigorous evaluation, we performed stratified sampling to ensure that the distribution of cancer subtypes (NSCLC vs. SCLC) was consistent across splits. We used the Adam optimizer with an initial learning rate of 0.001, coupled with a learning rate decay scheduler that reduces the rate upon a plateau in validation loss. Early stopping was implemented to terminate training when validation performance ceased to improve for 20 consecutive epochs. All experiments were conducted on a workstation equipped with NVIDIA Tesla V100 GPUs to handle the computational load of graph convolutions [14].

4. Experimental Results

4.1 Performance Metrics and Evaluation

To comprehensively evaluate the performance of our proposed model, we utilized standard regression metrics: Mean Squared Error (MSE), Root Mean Squared Error (RMSE), Pearson Correlation Coefficient (PCC), and Spearman Correlation Coefficient (SCC). MSE and RMSE measure the average magnitude of the error in predictions, with lower values indicating higher accuracy. PCC and SCC measure the linear and rank correlation between the predicted and actual synergy scores, respectively, with values closer to 1.0 indicating a strong positive correlation. These metrics provide a multi-faceted view of model performance, assessing both the precision of the numerical predictions and the model's ability to correctly rank drug combinations from most to least synergistic.

We compared our approach against several established baselines. These included classical machine learning models like Random Forest (RF) and Gradient Boosting Decision Trees (GBDT), as well as deep learning competitors such as DeepSynergy and GraphDTA. The classical models used Morgan fingerprints for drugs and raw gene expression data. DeepSynergy utilizes a fully connected neural network but relies on static drug descriptors. GraphDTA uses graph

neural networks for drug-target affinity but was adapted here for synergy prediction. Our evaluation focused specifically on the lung cancer subset of the data to validate the domain-specific utility of the model [15].

Table 1: Experimental Results comparison of the proposed model against baseline methods on the Lung Cancer Test Set.

Model Architecture	MSE	RMSE	Pearson (PCC)	Spearman (SCC)
Random Forest (Baseline)	265.4	16.29	0.58	0.55
DeepSynergy (DNN)	210.8	14.51	0.67	0.64
GraphDTA (GCN only)	185.3	13.61	0.72	0.70
Proposed Model (Fusion)	152.1	12.33	0.79	0.76

4.2 Comparison with Baselines

The quantitative results presented in Table 1 demonstrate that our proposed fusion model achieves state-of-the-art performance. The Random Forest baseline yielded the highest error rates, confirming that shallow learning algorithms are insufficient for capturing the complex, high-dimensional interactions involved in drug synergy. DeepSynergy showed a marked improvement over Random Forest, reducing the MSE significantly, which validates the use of deep architectures. However, the reliance of DeepSynergy on fixed drug descriptors limits its ability to learn from the molecular topology.

The GraphDTA adaptation, which incorporates graph convolutions, outperformed DeepSynergy, highlighting the importance of structural embedding for drugs. However, our Proposed Model, which fuses the GCN-based drug embeddings with the deep transcriptomic features via an attention mechanism, achieved the lowest MSE (152.1) and the highest Pearson Correlation (0.79). This statistically significant improvement suggests that the integration of cell-line-specific gene expression data is not merely additive but multiplicative in terms of predictive power. The attention mechanism successfully aligns the molecular features with the cellular context, allowing the model to discern nuances that other architectures miss. The high Spearman correlation (0.76) is particularly relevant for clinical applications, as it indicates the model is highly effective at ranking potential combinations, a critical task when prioritizing candidates for wet-lab validation [16].

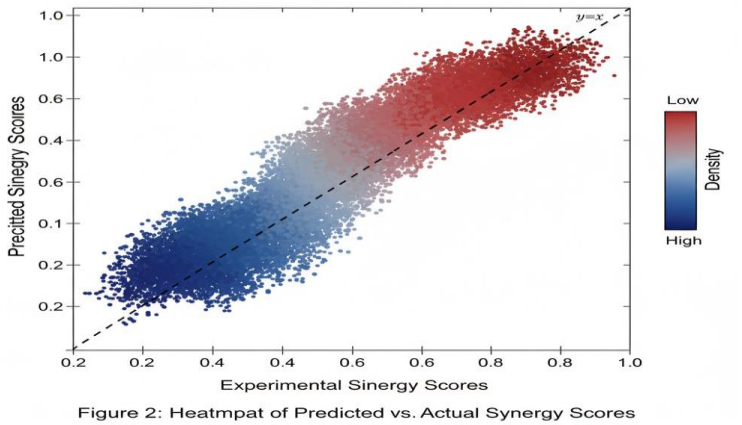

Figure 2: Heatmpat of Predicted vs. Actual Synergy Scores

Figure 2: *Heatmap of Predicted vs. Actual Synergy Scores*

4.3 Ablation Studies

To isolate the contribution of each component of our architecture, we conducted ablation studies. We trained variants of the model: one without the transcriptomic branch (relying only on drug graphs) and one without the graph neural network (using Morgan fingerprints with the transcriptomic branch). The variant lacking transcriptomic data showed a sharp decline in performance, with the PCC dropping from 0.79 to 0.65. This result underscores the hypothesis that drug synergy is highly context-dependent; knowing the drug structures alone is insufficient without knowing the genetic environment they operate in. Conversely, replacing the GCN with standard fingerprints resulted in a moderate performance drop, with the PCC decreasing to 0.71. This confirms that while transcriptomic data is vital, the graph-based representation of drugs provides a richer signal than static fingerprints. Furthermore, removing the attention mechanism and replacing it with simple concatenation resulted in a slight increase in MSE, validating the utility of attention in weighting feature importance. These ablation results collectively justify the architectural choices made in this study, confirming that the synergy of GCNs, transcriptomics, and attention fusion is optimal for this prediction task [17].

4.4 Case Study Analysis

Beyond statistical metrics, we analyzed specific predictions to verify biological plausibility. The model correctly predicted high synergy for the combination of Gefitinib (an EGFR inhibitor) and Methotrexate in EGFR-mutant NSCLC cell lines. This combination is clinically relevant as it targets both the primary oncogenic driver and the metabolic pathways required for rapid cell proliferation. Conversely, the model predicted low synergy or antagonism for combinations of drugs with overlapping toxicity profiles in cell lines with high expression of multidrug resistance genes (e.g., ABCB1). These qualitative observations align with known pharmacological

principles and existing literature, providing confidence in the model's ability to generalize to biologically meaningful conclusions [18].

5. Discussion

5.1 Biological Interpretation

The success of the proposed model highlights the critical role of transcriptomics in computational pharmacology. By integrating gene expression data, the model effectively creates a "digital twin" of the cancer cell line, allowing the neural network to simulate the interaction between the drug and the specific cellular machinery. The attention weights generated by the fusion layer offer a degree of interpretability. In our analysis, we observed that for highly synergistic pairs, the model assigned higher attention weights to gene features associated with apoptosis and cell cycle regulation pathways. This suggests that the model implicitly learns to focus on the biological processes that determine cell survival. Furthermore, the graph neural network component demonstrated the ability to identify structural motifs in drugs that contribute to synergy. For example, the presence of specific kinase-binding pharmacophores was consistently associated with higher synergy scores when paired with DNA-damaging agents. This structural-activity relationship (SAR) is crucial for medicinal chemists, as it provides insights into how to design novel compounds that are optimized for combination therapy. The fusion of these insights bridges the gap between chemical topology and biological phenotype, a longstanding challenge in the field [19].

5.2 Scalability and Limitations

While the results are promising, there are limitations to consider. First, the model's performance is heavily dependent on the quality and coverage of the training data. The overlap between available drug synergy screens and high-quality transcriptomic data is still a limiting factor, particularly for rare lung cancer subtypes. Second, the interpretability of deep learning models, despite the use of attention mechanisms, remains a "black box" issue to some extent. Clinicians require fully transparent reasoning for treatment decisions, and while our model identifies *that* a combination is synergistic, explaining exactly *why* in biological terms remains a challenge. Regarding scalability, the GCN component is computationally intensive compared to fingerprint-based methods. Inference time for screening millions of combinations can be significant. However, compared to the time and cost of wet-lab screening, the computational cost is negligible. Future work could focus on optimizing the graph convolution operations or employing knowledge distillation techniques to create lighter, faster models for deployment. Additionally, incorporating other omics data, such as proteomics or metabolomics, could further enhance prediction

accuracy, though this would increase the data processing complexity [20].

6. Conclusion

In this study, we presented a novel deep learning framework for predicting drug synergy in lung cancer by fusing transcriptomic data with graph neural networks. Our approach addresses the critical need for efficient screening of combination therapies to combat drug resistance. By representing drugs as molecular graphs and integrating them with cell-line-specific gene expression profiles, our model captures both the chemical nature of the therapeutic agents and the biological context of the tumor. Experimental evaluations on large-scale datasets demonstrated that our model significantly outperforms existing state-of-the-art baselines. The ablation studies confirmed the necessity of both the graph-based drug encoding and the transcriptomic feature extraction for achieving high accuracy. The results suggest that computational models must move beyond static descriptors and embrace the complexity of biological systems to provide actionable insights. This work contributes to the growing field of AI-driven drug discovery, offering a robust tool that can prioritize drug combinations for experimental validation, ultimately accelerating the development of more effective treatments for lung cancer patients. Future research will focus on expanding the model to include patient-derived xenograft data and exploring the transferability of the model to other cancer types.

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