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DEEPDRA: A DEEP LEARNING FRAMEWORK FOR DRUG REPURPOSING AND CANCER DRUG RESPONSE PREDICTION USING MULTI-OMICS DATA

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ABSTRACT

Combating cancer is a significant challenge. Numerous cancer treatments are available. The medications have worked better. Making cancer medicines costs a lot of money and takes a long time. The recycling of medicines is proposed as a solution to these problems using computational methods like deep and automatic learning. For the purpose of repositioning cancer medications and predicting responses, traditional methods of automatic learning have been surpassed by deep learning. This study aims to develop a deep learning model that can anticipate responses to anticancer medications by making use of multi-omic and medication-related data in order to facilitate medication repositioning. Multi-omic data dimensionality is decreased by autoencoders. Polyvalent. Autoencoders related to MLPs To determine how effective our model is, we tested it on GDSC, CTRP, and CCLE. In many instances, our model always performs better than the current methods. Our model has an AUPRC of 0,99, which is significantly higher than that of other models. With an AUPRC of 0,72, the model developed on GDSC and tested on CCLE surpasses three previous studies. In conclusion, our deep learning model surpasses the current models. Utilizing this model, we intend to test drug responses and investigate reprogramming in an effort to discover novel cancer treatments. Our research demonstrates that sophisticated deep learning can improve anticancer treatments' precision.

KEYWORDS: Drug Repurposing, Deep Learning, Cancer Drug Response Prediction, DeepDRA Model, Multi-Omics Data Analysis.

1. INTRODUCTION

One of the most difficult diseases to treat through individualized treatment, chemotherapy, and surgery is cancer. Drug cancer treatments are expensive, time-consuming, and complex yet less invasive and safer [1]. As a result, drug repurposing might take the place of drug development. Medication repurposing is encouraged by a number of clinical approaches. Complex relationships between drug data and cancer multi-omics, such as transcriptomics, proteomics, genomes, and epigenomics, may be better understood with the help of computational methods. The preliminary conventional medicine response prediction research looks promising. Similar to computational biology, deep learning predicts cancer treatment responses. Deep learning predicts drug effects better than models. To predict medication reaction, deep learning learns complex patterns in vast data. Deep learning is used by cancer researchers to evaluate medication combinations and responses. Cancer therapy response analysis [2] investigates drug ranking. Pharmaceutical responses are predicted using deep learning because it can handle multidimensional data and uncover hidden relationships. Menden et al.'s initial predictions for the IC50 Neural networks with a single hidden feed forward were used. For better predictions, Sharifi Noghabi et al. recommended late multi-omics integration with triplet loss and data integration. Using kernels, we developed the DeepDRKmodel to extract pharmacological and omics features. Wang et al. predicted pharmacological response by using a trained model and classifier to reuse medicine. Recommender systems anticipate drug reactions [3].

Convolutional Neural Networks are used in a lot of research to predict how patients will react to medications. Twin neural networks (tCNNs) were used to retrieve pharmacological and omics data features in the past. GNNs that can predict drug responses based on graphs are being developed. DeepCDR by Liu et al. was the first GNN drug response prediction technique. Transformers are one recent application. Different drug reactions were predicted by DeepTTA based on transformers. IBT-Net makes use of transformers to incorporate omics data and a straightforward multilayer perceptron (MLP) to embed drug data in order to make a classification model and predict drug response [4]. These models have many advantages, but they rarely work with other cancer datasets. Because they were trained on specialized datasets, these models perform poorly on external datasets. Since models cannot generalize beyond datasets, development of predictive algorithms based on a single study overestimates prediction performance. Disease and drug coverage are limited by the lack of data types. Representation is improved by integrating multiple omics.

2. RESULTS

Seven performance and generalisability tests were run on our model. Model resilience is evaluated in numerous datasets and settings. In an ablation study, we removed the model's autoencoders to see how they affected outcomes. Model output was altered by the autoencoders [5]. The second experiment tested the generalization to multi-omics and the data integration hypothesis with single-omic data. After the ablation experiment, our model was compared to other models. GDSC and CTRP datasets being combined in the third experiment. After being trained, tested, and cross-validated five times, this data was compared to DeepDRK. Our model was validated by favorable outcomes. In the fourth experiment, we tested the model for pattern generalization on CCLE after training it on GDSC and CTRP. comparing this test to DeepDRK. DeepTTA and iBT-Net were compared to our model for performance [6].

The model's capabilities were tested in additional tests. The fifth experiment used 5-fold cross-validation to verify the model's internal consistency on GDSC. In order to guarantee the model's adaptability and effectiveness, it was tested on the CCLE and CTRP datasets in the sixth and seventh tests after being trained on the GDSC dataset. Our model for predicting drug effects was effective. potential advancements in therapeutics and precision medicine. Competition in drug response models is shown by comparisons [7].

A. Ablation Study

We investigated how the performance of the drug-cell line interaction categorisation model was impacted by autoencoder components. In order to run the model without autoencoders, direct embedding data were used. It has room for improvement because of its AUPRC score of 0.74[8].

Table 1. Comparing the DeepDRK data set to the CTR P+GDSC data set using a mixed set test with a 5-fold cross-validation.

Model / Metric	Accuracy	Precision	Recall	F1 Score	AUC	AUPRC
DeepDRA (AE-Free)	0.49	0.90	0.49	0.65	0.50	0.73
Drug–Anti-Epileptic Drug DeepDRA	0.88	0.93	0.65	0.90	0.93	0.96
Cell AE DeepDRA	0.97	0.97	0.97	0.97	0.97	0.97

In further studies, Drug Autoencoder reached 0.76 AUPRC. It demonstrated that Drug Autoencoder enhanced model efficiency. With 0.91 AUPRC, the Cell Line Autoencoder got better. This demonstrates how the Cell Line Autoencoder, which is necessary for model building, expands feature space from high-dimensional cell line embeddings. Final testing demonstrated that the model was compatible with the Cell Line and Drug Autoencoders. The outcomes are presented in Table 1[9]. We tested model performance on single and multi-omics data to measure data integration. In mixed-set testing, multi-omics models performed better than single-omics models (Table 2).

Table 2. Model for the GDSC, CTRP, and CCLE tests..

Data / Feature Type	Accuracy	Precision	Recall	F1 Score	AUC	AUPRC
Test Out	0.803	0.783	0.857	0.812	0.890	0.873
Mutation (Single Copy)	0.803	0.783	0.857	0.812	0.836	0.876
Unpaired Expression	0.803	0.783	0.857	0.812	0.826	0.852
Individual CN Multi-omic Signatures	0.803	0.783	0.857	0.812	0.815	0.875
Cross-Omic Characteristics	0.803	0.783	0.857	0.812	0.702	0.824
Combined Multi-Omics Model	0.803	0.783	0.857	0.812	0.823	0.896

B. Method Comparison

Our model was trained and evaluated using drug fingerprints, drug descriptors, CNV, Exp, and mut. Our model passed this initial test thanks to 5-fold cross-validation and 0.99 AUPRC, which set the baseline for its performance on this dataset (Table 3). Context was provided by comparing these results to DeepDRK on the same dataset. With an AUPRC of 0.97, our model is more durable (Fig. 1)[10]. The generalizability of our model was evaluated in various scenarios. On CTRP, GDSC, and CCLE, our model was trained and tested. Our model was able to generalize with an AUPRC of 0.87 88, as shown in Table 4. The DeepDRK model with the same testing regime has an AUPRC of 0.85, which is significantly lower than our model (Fig. 1) [11].

Table 3. The mixed set test was used to compare CTRP+GDSC and deepDRK, with a 5-fold cross validation.

Model	Accuracy	Precision	Recall	F1 Score	AUC	AUPRC
DeepDRK	0.832	0.826	0.945	0.960	0.873	0.953
DeepDRA	0.815–0.952	0.971–0.973	0.971–0.992	0.973–0.975	0.873	0.953

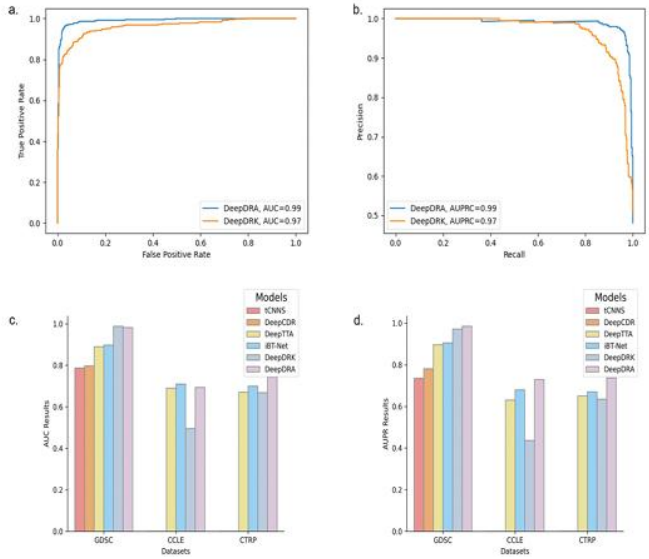

Fig 1. DeepDRA had a higher AUC than DeepDRK in the mixed-set experiment on GDSC+CTRP. identical test set as in (a) with AUPRC findings. DeepDRA's comparison to previous models.

The resilience of our model and the generalizability of the dataset are demonstrated in these comparison tests. Precision medicine depends on this. Model performance was thoroughly tested in additional tests in these studies. The model was trained and evaluated in the third experiment using 5-fold cross validation on GDSC. Good GDSC prediction is indicated by the model's 0.98 AUPRC (Table 5). Performance is significantly superior to previous studies[12]. The GDSC-trained model was used on CCLE to test cross-dataset generalization. The AUPRC of our model is 0.73 (Table 6). A model

may project learnt patterns to an external dataset, improving generalisation over earlier studies.

Models were trained and evaluated in the most recent study using GDSC and CTRP datasets. The model's applicability to the data set was demonstrated by the fact that its results were consistent with those of previous models. The model's adaptability and potential for drug response prediction are demonstrated by its constant performance in Table 7[13]. Our in-depth research demonstrates that the model performs admirably in a variety of analytical contexts. Therapeutic optimization and precision medicine may benefit from these accomplishments[14]. Patient data were used to evaluate the generalization of the model. Testing on patient data frequently results in disappointing outcomes due to the distinct domains of cell-line and patient data. Training and testing the model on patient data improves outcomes. Patient omics and response data are scarce, but deep learning algorithms need more data to train. The model was validated using TCGAportal patient data after being trained with CTRP+GDSC in response to these issues. DeepDRK outcomes are compared in Table 8. On patient data, our models perform better than DeepDRK, despite minor results.

Table 4. a comparison of DeepDRKon CCLEasatest training and CTRP+GDSCdata.

Model	Accuracy	Precision	Recall	F1 Score	AUC	AUPRC
DeepDRK	0.733	0.970	0.688	0.804	0.832	0.823
DeepDRA	0.803	0.823	0.840	0.883	0.851	0.887

Table 5. Using expression and drug data, training and evaluating the results of the model on GDSC.

Model	AUC	AUPRC
tCNNS	0.787	0.735
DeepCDR	0.797	0.781
DeepTTA	0.889	0.896
iBT-Net	0.897	0.988
DeepDRK	0.982	0.905
DeepDRA	0.972	0.985

Table 6. Utilizing expression and drug data, the results of GDSC model training and CCLE testing.

Model	AUC	AUPRC
DeepTTA	0.69	0.63
iBT-Net	0.71	0.68
DeepDRK	0.495	0.693
DeepDRA	0.436	0.729

Table 7. Results from the CTR test and the GDSC training model Drug and gene expression data.

Model / Metric	AUC	AUPRC
Model 1	0.67	0.65
Model 2	0.70	0.67
Model 3	0.668	0.743
Model 4	0.635	0.737

Table 8. The CTRP+GDSC training model and the drug data test results from the TCGA.

Model	AUC	AUPRC
DeepDRK	0.506	0.619
DeepDRA	0.527	0.647

C. Drug Repurposing

In a different breast cancer experiment, we tested our model after training it with CTRP and GDSC. Every treatment for cancer was tried. Surprisingly, the model predicted effective treatments for breast cancer that were neither sensitive nor resistant. Breast cancer cell lines provided evidence to support the idea. Using model estimates for each drug-cell-line combination, we ranked medications according to projected effectiveness. To treat cancer, a drug with a concentration above 0.5 was intended. Utilizing its leading cancer drugs, our system provides novel breast cancer treatments [15]. Investigations confirmed our model's prediction of NVP-TAE684's breast cancer sensitivity. There was no binarization of the Olaparib breast cancer treatment response data. For the breast cancer treatment dabrafenib, the idea worked well. Despite being binarized as "unknown" in our training data, bleomycin had a high treatment potential score, just like in previous studies. Recent research confirms the earlier findings that COL-3 could be used as a medicine. Based on scores, our model suggested LRRK2-IN-1, ML162, gefitinib, JQ-1, tigecycline, and nerati nib[16]. BRD-A71883111 and ML050 were the drugs that our model identified. The lack of research on these medications presents a pioneering opportunity. For these drugs to be effective and safe, clinical trials are necessary even if there is no empirical evidence. This kind of medicine helps find new treatments for breast cancer. For drug development, early computer models are crucial.

3. MATERIALS AND METHODS

A. Dataset

Our research uses three crucial datasets. Multi-omics research on cancer is included in the Genomics of Drug Sensitivity in Cancer (GDSC) database. Oncological molecular characterisation is complicated by the presence of multiple gene mutations, gene expression patterns, CNVs, and methylation patterns in this collection. CTRP drug response data are useful, even though multi-omics

data are lacking [17]. Gene expression, mutation profiles, and CNV information are all included in the Cancer Cell Line Encyclopedia. Enhancing CTRP's drug response analysis using CCLE's multi-omics data.

B. Preprocessing

Traditional imputation replaced NaNs with zeros in our datasets, which reduces data distribution distortion. Important traits were extracted from two large multi-omics datasets; the hardest challenge was finding and extracting intersecting database properties. Our data preparation begins with a comprehensive missing value investigation. This allowed us to train and test our model on a variety of datasets[18].

C. Cell-line data

The data from the GDSC cancer cell line is helpful. It has 17738 gene expression, 14729 methylation, 21879 variation in copy number, and 23189 mutation. It is possible to study epigenetics, genomic changes, and structural genomic variations that cause cancer. 19,534 mutations and 19,221 genes are expressed by CCLE. Examine 59267 CNV characteristics for genetic structural variation in cancer cell lines. Multi-omics are not present in the CTRP dataset. CTRP drug response information was provided by CCLE cell line data. Model training datasets can be optimized with the right selection. By harmonizing the three modalities of GDSC and CCLE, unnecessary data are avoided. There are 57,508 features in the cell-line dataset, including 15,963 gene expression, 17,671 mutations, and 23,874 CNV. GDSC and CCLE data were used to investigate intricate transcriptional, epigenetic, and genomic relationships. Understanding cancer biology, drug sensitivity, precision medicine, and therapeutic innovation require this integrative approach (Table 10) [19].

Table 10. Two datasets combine the characteristics and modalities of each dataset.

Dataset	Gene Expression	Mutation	Methylation	649.t010	CNV
CCLE	19221	19534	23189	17671	59267
GDSC	17738	19534	15963	14729	24503
CCLE + GDSC	18480	19534	19576	16150	23874

D. Drug Data

A lot of pharmacological information is needed for drug effects studies. Medication response was reported by GDSC, CTRP, and CCLE. Pharmacology-related drug data are included in this collection. For each chemical, PubChem provided SMILES that were consistent and accurate. Computer chemical notation is standardized by the representations. For our Landrum dataset, RDKit cheminformatics software was used to generate drug molecular descriptors. Structural and physicochemical characteristics describe drug biological activity and pharmacodynamics. For these drugs, Morgan's Fingerprint data were used by our model. 704 drugs were produced by this extraction. The pharmacological profile of each entity is contained in its multidimensional feature set. For this study, extensive medication data collection takes time. [19] Our drug prediction model became more reliable and accurate.

E. Drug Response

For preprocessing, binarization of drug response data was essential. The objective was to classify IC50 and AUC as "sensitive" or "resistant" numbers. Our drug response analyses were standardized by binarization. AUC values for CTRP are sensitive below 6 and resistant above 16. Drug response values below 0.2 in the GDSC dataset indicated resistance, while those above 0.99 indicated sensitivity. Last, ActArea binarized the CCLE dataset. Act area values >2 indicate sensitivity, while <0.5 indicates resistance. The final preparatory stage was the creation of drug lists using drug response databases. The SMILES of each drug were frequently obtained. We trained our model using GDSC and CTRP medication responses. There are approximately 28,000 drug combinations for cell lines in the dataset. There are 10,007 "sensitive" and 18,758 "resistant" pairs listed in Table 11[20]. Before training our classifier for strength, we carefully classified the medication response data. By discretizing continuous drug response data, a robust classifier that can be applied to a variety of cell lines was developed. Drug sensitivity and resistance can be detected using this conventional approach. New treatments and precision may benefit from these discoveries.

Table 11. sample sizes for category-specific datasets.

Drug Response	CTRP	GDSC	CCLE	CTRP + GDSC
Sensitivity	1423	10820	2956	3792
Resistance	6827	13331	10007	18758

F. Problem Formulation

Pharmacological characteristics, fingerprints, and multi-omics data from cell lines (genomics, transcriptomics, and epigenomics) are utilized. The

sensitivity or resistance of a drug cell-line pair can be estimated using our method (0–1). A, where i = 1, is used to represent cell-line data, medication description, and response value... N. Utilizing mutation, gene expression, copy number variation, methylation, drug descriptors, fingerprinting, and other methods, we make predictions. We dispersed characteristics to boost model performance because our study used four different kinds of omics data. This demonstrates the proposed method's structure. Our study begins with omics dataset collection and preprocessing. We classified medication response metrics into binary categories to create a model-ready dataset. Multi-omics data were used to train our cell-line autoencoder for each cell line. In feature space, the autoencoder summarized multi-omics data [21]. A drug response predictor for cell lines was developed by us. RDKit was used to create molecular descriptors and fingerprints from SMILES of the drugs under investigation. From these embeddings, our Drug Autoencoder generated feature spaces for each drug entity. Drug response pairs were used to match data from cell lines and drugs. The final medication response prediction classification model was provided by couples (Fig. 2). Below, we detail our strategy.

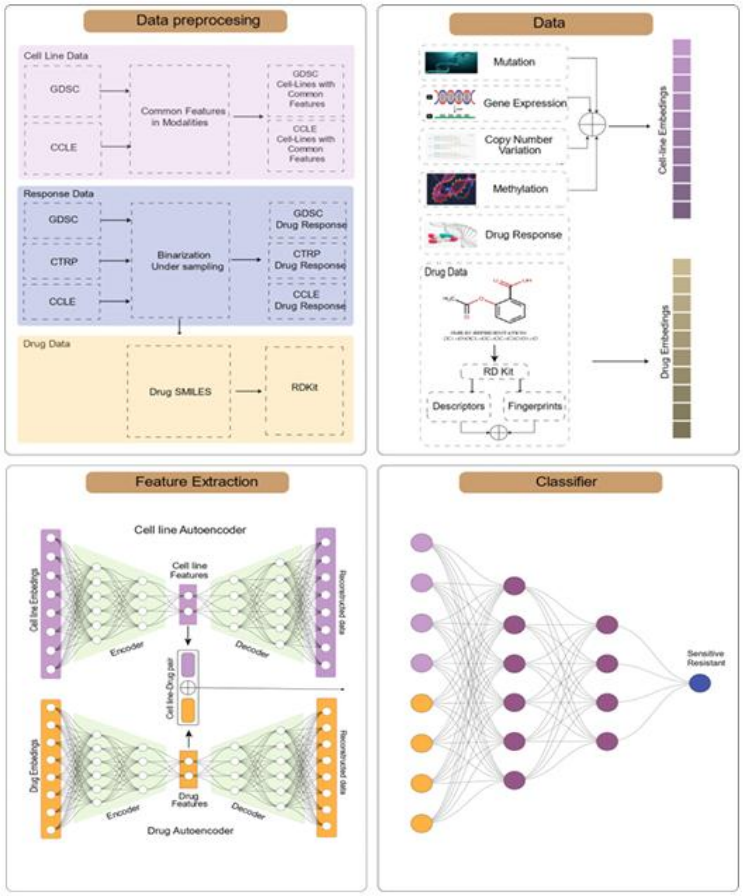

Fig 2. This model has several steps. Utilizing RDKit, we combine cell-line modalities and construct drug embeddings following data collection and preprocessing. Following the extraction of autoencoder features, an MLP predicts drug response. Sequence is shown in the figure.

4. MODEL IMPLEMENTATION AND EVALUATION

The generalization of restricted dataset models was improved by autoencoders. Using a combined loss function, our multi-task approach simultaneously trains the autoencoder and classifier. The model's handling of mixed datasets was demonstrated using this dual-facet technique. With insufficient data, our study sought to make generalizations. By enhancing integration and selecting the most effective autoencoder architectures, we focused on generalization and enhanced model outputs on a variety of datasets. For the purposes of precision medicine and therapeutic research, these findings may help us comprehend pharmacological response dynamics[22]. This work makes use of a single hidden layer autoencoder with 256 ReLU units. The size of the data determines the number of autoencoder input layers. MSE is training loss for the autoencoder. The MSE-MLP composite

loss method is used to train the model. Using MLP classification and autoencoder feature extraction, multitask training improves learning. The study's MLP has hidden layers of 128 units. Layers of ReLU/Sig moid are hidden and output. Layers for a latent autoencoder's input. Using the AdamDelta optimiser and a 0.01 learning rate, MLP was trained over 25 epochs. Core i3 10300 and 3060Ti GPU were used for model training. Training overfitting was reduced by splitting the data into training, validation, and test sets with ratios of 0.7, 0.1, and 0.2, respectively. To avoid training overfitting, we made these three parts accurate. MSE drop is a simple loss function for an autoencoder.

$$L_{Autoecoders} = \frac{1}{n}\sum_{1}^{n}\left(Y_{i_{autoencoder}} - \hat{Y}_{i_{autoencoder}}\right)^2 \quad (1)$$

Binary Cross Entropy Loss occurs in ML:

$$L_{Classifier} = -\frac{1}{n}\sum_{1}^{n} y_{iclassifier}.\log(p(y_{iclassifier})) + (1 - y_{iclassifier}).\log(1 - p(y_{iclassifier})) \quad (2)$$

Our loss in end-to-end integration is y for the final class and y for the autoencoder:

$$L = L_{Autoencoders} + L_{Classifier} \quad (3)$$

$$L = \frac{1}{n}\sum_{1}^{n}\left(Y_{i_{autoencoder}} - \hat{Y}_{i_{autoencoder}}\right)^2 - \frac{1}{n}\sum_{1}^{n} y_{iclassifier}.\log(p(y_{iclassifier})) + (1 - y_{iclassifier}).\log(1 - p(y_{iclassifier})) \quad (4)$$

The 256-node autoencoder outperformed the 1024, 512, and 128 autoencoders after several hyperparameter adjustments. 50-node latent layers performed better than 200, 100, 50, and 25. The MLP stood out thanks to its 128 hidden nodes. We evaluated learning rates of 0.005, 0.01, and 0.05. The best results were obtained with a learning rate of 0.01. Autoencoder and classifier loss were assessed together by a variety of factors. A, B, and C contributed. Out of the 2, 1, and 0.5 losses, one factor performed best.

$$L = aL_{Drug\ autoencoder} + bL_{Cell-line\ autoencoder} + cL_{Classifier} \quad (5)$$

Model code for clinical and practical use is available on GitHub. The input data are used to train the model. For superior training outcomes, clinical patient data is recommended over cell-line data. On non-GPU hardware, the model can be trained. Using any patient omics data and drug combinations, the trained saved model can determine the best medications for a patient.

5. DISCUSSION

We looked at cell line and response data from the three major cancer databases GDSC, CTRP, and CCLE. Preparation is the first step in integrating data. Prediction accuracy was increased by combining two pharmaceutical and four omics data types. All of the datasets shared the same omics characteristics after missing data were removed. To evaluate various datasets and metrics, we binarized medication responses using DeepDRK criteria[23]. We prioritized generalisability in cross-data set validations, despite the fact that GNNs and Transformers are common model construction architectures. Two Autoencoders—one for omic and one for pharmacological data—simplified our method. MSE loss was used to reconstruct the autoencoder data. Concatenated binary classifier with latent representation. Autoencoder and binary cross-entropy classifier losses made up the total loss. Model building architectures like GNNs and Transformers are common, but we wanted a simple one for generalization and cross-dataset validation. Two MSE-loss autoencoders are used in the reconstruction of omics and pharmacological data by our method. Latent representations were concatenated and presented to binary classifiers. Binary cross-entropy was the loss function for the classifier. Complete loss, including the classifier and autoencoder. iBT-Net and other drug response prediction models are compared to our medication repurposing model. Our model outperformed them in numerous ways. Our model's AUPRC is 0.98 when tested on 20% of the GDSC dataset and trained on 80% of it. considerably more than iBT-Net's 0.90. When tested on CCLE and trained on GDSC, our model outperformed iBT-Net by 0.68 points and 0.72 points in AUPRC and CTRP, respectively. Utilizing cancer cell lines, the therapeutic potential of our method was investigated. We tried every drug-cell line combination outside of our training data. Our model used these combo scores to predict how to treat cancer. It's nice to find treatments for cancer. Cancer may be treated in a different way by new or understudied medications. We must examine a few disadvantages of the job. Binarized response data was destroyed by an undefined data class. Due to missing data modules, we were unable to verify our model with patient data from all modalities, preventing clinical trials. Our findings suggest that data preparation and integration should take precedence over complex, non-generalizable models. Autoencoder denoising should be improved, omics data like PPRA and RNA-seq should be integrated, and the model should be trained on missing modalities to ensure its robustness. KEGG pathways or careful survival analysis should be included in future research.

6. CONCLUSION

Using multi-omics data and drug descriptors, this article introduces DeepDRA, a deep learning

framework for cancer treatment response prediction. Using autoencoder architectures and multilayer perceptron networks, the model made it easier to extract features and make predictions. In AUPRC and large cancer datasets such as GDSC, CTRP, and CCLE, the model outperformed DeepDRK, DeepTTA, iBT-Net, and DeepCDR. In order to predict pharmaceutical response, the study suggested integrating data on gene expression, mutation, methylation, and copy number variation. Model robustness and high-dimensional biological dataset processing were both enhanced by autoencoder-based dimensionality reduction. Additionally, the model demonstrated its precision oncology and therapeutic discovery potential by revealing several therapies for breast cancer that made use of repurposed drugs. Last but not least, DeepDRA for tailored therapy and pharmaceutical repurposing for cancer works and scales. Prediction reliability and clinical validity may be enhanced by including additional omics data types, enhancing denoising autoencoders, and verifying with clinical patient data sets.

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