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Deep Learning in Cancer Genomics: Decoding Genetic Alterations for Precision Therapy

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Abstract: It is also one of the leading causes of morbidity and mortality in various states around the world due to the genetic heterogeneity and the complexity of molecular modulations involved. Recent technological innovations into the megabase sequencing of genomes offered gigabits of data that can also be exploited to advance the discovery of oncogenic pathways and mutational landscapes. As genomic datasets have since achieved computational complexity, they can now be analyzed and interpreted using deep learning (DL), a sub-field of artificial intelligence. CNNs, RNNs and autoencoders are actually found to surpass other models in terms of meaningful genetic changes, cancer-type predictions and precision therapeutics. Multi-omics provides a means to incorporate peripheral data and interactions between genes, epigenomic variations, and transcriptomic profiles that would normally be dwarfed by older analysis software. In this article, one can see an overview of censorship as applied in cancer genomics, practices being performed in the hence, significant findings, and cancer genomes that can be applied in clinical practice. These results indicate that DL models can possess a great predictive ability in order to identify actionable mutations, but remains limited by data-scarcity-, interpretability-, and cost-computational disadvantages. The future research is needed to improve the model transparency which includes information and discovery strength to generate a successful clinical translation model quickly and accelerate individual treatment of cancer.

Keywords— Cancer Genomics, Deep Learning, Precision Medicine, Genetic Alterations, Multi-omics, Convolutional Neural Networks, Autoencoders, Transcriptomics, Personalized Therapy, Bioinformatics.

INTRODUCTION

Cancer is a very differentiated group of disorders that are incredibly complicated in the world where was the continuously liberate increase of the cells, and chronic complications of grotesque financial condition compared to networks, very complicated interaction

of natural events with surroundings. Nevertheless, despite all the giant steps taken in diagnosis and treatment, cancer has remained one of the most critical health issues on the planet with millions of people dying annually. Approaches to analyzing and treating cancer based on histology and imaging, which were developed earlier, are not sufficient enough to describe the heterogeneity of molecules comprising a tumor. These limitations complicate the establishment of a genuinely personalized approach to treatment and constitute the differences of treatment outcomes seen among patients. High throughput sequencing technologies have facilitated the generation of immense quantities of genomic, transcriptomic and epigenomic information to give new insights into the molecular genesis of cancer under scales previously considered impractical. Nevertheless, its formal complexity and dimensionality influences the need to adopt theoretically superior computing facilities nearest to, as to, elucidate latent patterns and constructions which one might not ordinarily observe by less theoretical statistical methods [1-4].

Biomedical or Deep learning (DL) is an AI-based technology Deep learning (DL) Deep learning (DL) is a sub-segement of AI that has produced breakthrough technology. DL models are able to automatically generate a hierarchy of the raw data, and with time even allowing the identification of finer and non-linear dependencies between genes, mutations, and epigenetic changes [7]. Convolutional neural networks (CNNs), recurrent neural networks (RNNs), and autoencoders are currently achieving significant success in other genomics areas, including tumor classification, driver mutation identification, and estimating response to treatment. Like classical machine learning algorithms in which features are frequently highly handcrafted, DI models suffice to learn useful features of high-dimensional data, vastly enhancing predictivity and likelihood.

Such a piece has been motivated by the frightening fact that there is a disconnect between the revelations of the knowledge achieved in genome research and the practice of medicine. In spite of the fact that many researchers have performed the role of individual biomarkers in cancer, few of them have successfully applied multi-omics data to forecast the actionable changes and guide to precision therapy. The molecular heterogeneity of cancer is an indicator that a single molecular or transcriptomic marker will not be sufficient to inform a decision regarding treatment. Due to the potential to combine a wide range of different kinds of datasets usingDL, there is an opportunity to harness the interactions between genes, epigenetics, and transcriptomics profiles to build a more comprehensive understanding of tumor biology. Additionally, accurate predictive models to determine major driver mutations can aid clinicians to prescribe targeted therapies, track disease progression, and enhance patient outcomes [8].

This study has the main aims in triplicity. Originally, to create powerful DL models able to decompose genetic changes in many types of cancers through combining multi-omics data. Second, to determine the predictive capability of those models in detecting actionable mutations and tumors subcategories. Third, to determine how current model outputs can support precise therapy decisions [9]. To realize such goals, we created a workflow that includes the data retrieving into a public repository, preprocessing and data normalization, training of a DL model, multi-omics integration, and full-scale evaluation based on traditional performance measures such as accuracy, AUC, F1-score, and precision-recall curves. Moreover, SHAP as well as integrated gradients were used to determine critical genomic variables that affected predictions, closing the gap between the computational model and clinical knowledge.

Overall, the study is the first step toward showing the elaborate guideline to integrate DL in cancer genomics, and the researchers proved that it is capable of transforming precision oncology. The work provides the answer to significant problems of predictability of genetic modifications and personalized treatment based on the integration of multiple omics data, the implementation of neural networks of high order, and its explicaltory outcome. With this introduction in mind, it is possible to turn to the applicability of DL to cancer research and provoke new studies on how computational approaches may be utilized to improve clinical decision making and, finally, patient care [6].

Novelty and Contribution

This unique work consists of systematic collection of deep learning methods and multi-omics based cancer genomics data to draw clinically relevant conclusion. Though there have been studies done previously in which individual-omics based data sets are mainly examined or focused on individual types of cancers, the paper highlights how this molecular phenomenon should be regarded as a whole encompassing both genomics, transcriptomics and epigenomics that describe complex molecular phenotypes in tumors. Using much more sophisticated DLs like CNNs, LSTMs, and autoencoders, it can automatically discover how to optimize the use of interesting features, how to learn non-linear interactions and even high-dimensional data, with only a small amount of attention given to it.

The principal conclusions of this work are:

- Integrative Multi-Omics: Contrary to traditional studies employing one layer of omics to infer the effects of given mutations and actable outcomes of genetic activity, it is

desired that the multi-omics approach is used to elucidate the key driver mutations and actable outcomes and changes in genetic activity in a more coherent way difficult to discern using single-omics research.

- Deep learning-Predictive Framework: This article introduces a robust framework of the DL approach that can be used to predict the subtype of gastrointestinal tumors, their subsequent recurrence or metastasis, and confidence in an optimal selection of a therapeutic option.
- Generalizable and Scalable Models: The models have shown to be applicable to large genomic data sets and have potential broad applications to a wide array of cancers and a wide range of patient groups.
- Potential Therapy Precision: The framework determines points of interest in the approach, and specifies steps to be taken in patients with well-known mutations, such as targeted therapies (possibly as din surgery), such as targeted kinase or PARP outlaws.

The rationale of this effort is that there is an urgent necessity to enhance the predictive power and clinical utility of cancer genomics analysis. The work is based on the concepts of deep learning and content integrations of multi-omics along with high simplicability, scalability, and performance in the context of genetic alteration decoding. The latter are the initial moves towards much-needed studies on real-time clinical translation, explainable AI constructs, and optimisedpersonalised therapeutic regimens in cancer.

Related Works

There is much genomic, transcriptomic, and epigenomic data generated nowadays, since the introduction of the high-throughput sequencing systems, and this seems like the right moment to explore a more in-depth portrait of the molecular pathogenesis of cancer than at any other time. The early approaches of cancer genomics ran primarily on statistical model and ordinary machine learning algorithms that could assist in a sequence of independent behavioral guidelines as to genetic variations and ailments. This may have provided some quite informative data but these approaches may be constrained in the number of dimensions they can analyze, the amount of multi-collinear non-linear relationships they can find and the depth of multi-omics information they can exhaustively use. These limitations highlighted the significance of having a more detailed set of computation rules that could be used to help derive detailed patterns in the genomic data and encode it in genome-cast data information details with clinical objectives.

In 2025 M. El-Tanani*et al.*, [15] suggested the deep learning has become a fully applicable solution to such challenges. This CNNs-based profile analysis methodology has been widely applied to gene expression fields, in which hierarchal features are automatically learned that can be used to stratify tumor types and strongly predict cancer subtypes. Recurrent neural networks (RNNs) or long short term memory assists in predicting temporal information by means of sequential adaptation of genomes and pattern of mutation of prediction of tumor formation with time. Auto encoders and variation auto-encoders have been used in dimensionality reduction, data demising, and multi-omics integration to uncover meaningful latent-dimensional representations after complex biological data is involved. These constructions provide potential to investigate tumor heterogeneity further and have played a crucial role in identifying major driver substitutions and shared molecular patterns associated with cancer development.

Lately, there has been a lot of excitement in incorporating the multi-omics information which provides a greater understanding of the tumor biology, compared to the study of the single-omics. It is established that the utilization of genomics as well as the less regulated transcriptomic and epigenetics systems used in the analysis takes precedence to those taught on one dataset in predicting disease phenotype and therapy reaction. Based on interactions of these kind, it is possible to make a sort of discrimination between separate strata of molecules, illustrating weak regulations, judging tumor activity. It has also used the integration of multi-omics to localize and preemptively calculated incidental variations, discover neo-antigens, and forecast personal patient responses to immunotherapy that matter when treating cancer patients individually. It has been discovered that NN-based models are more efficient to manipulate these rich data structures, and have been trained on latent images of simple hidden patterns that other algorithms struggle to identify.

In 2025 G. Calvino *et al.*, [5] introduced the detecting mutations and their subtypes is not the end of deep learning. Instead predictive models have been presented to estimate patient survival rates, risk assist in patient stratification, and help in defining potential therapeutic targets. Deep-learning systems are capable of predicting underlying drug sensitivity-, or drug-resistance-related biomarkers through empirical comparisons of high-dimensional molecular profiles, thus informing the precision therapy decision-making process. Further, the interpretation of significant genomic predictors in these models relying upon attention and the scoring of feature importances have helped the solution of one of the inherent dilemmas of deep models in healthcare, the black box issue. Interpretability techniques enable scientists and medical personnel to gain insight into what they are

being presupposed to predict on ground and this ultimately increases comfortability with computation programs which subsequently leads to the prospective clinical application.

There are associated difficulties with using deep learning in cancer genomics. The inaccessibility of quality data, variably annotated multi-omics data and the differences in common cancer across populations restricts even pre-clinical-trial based model generalizability, as does the heterogeneity of common cancer within populations constraining predictive modeling. In addition, it implements specifically engineered hardware along with algorithms to enable training of large-scale deep learning models, which again can be computationally intensive. Other problems include challenges of data interpretation caused by the use of various sequencing platforms and batch effects that should be counterbalanced with careful preprocessing and normalization measures. They are currently being broken down through attempts to standardize datasets, develop robust pipelines, increase model interpretability, etc. Furthermore, to transform the insights of deep learning into clinical practice, one must ensure they are validated in real-world studies, combined with electronic health records, and compliant with regulatory requirements to promote patient safety and effectiveness.

In 2025 J. Le *et al.*, [11] proposed the history of deep learning applications to cancer genomics has also highlighted the need to adopt multidisciplinary, collaborative methods. The integration of computational biology, oncology, bioinformatics, and clinical research has enabled the generation of biologically meaningful, and clinically relevant, models. Recent advances in transfer learning, semi-supervised learning, and federated learning have continued to optimize model performance, facilitating knowledge transfer across different types of cancer, enhancing robustness with small amounts of labeled data, and ensuring patient confidentiality in studies across multiple institutions. The latter methods could be especially useful in uncommon cancer cells or when the scale of exchanging data is limited due to legal or privacy factors.

As a whole, this body of research shows that deep learning presents the opportunity to revolutionize the decoding of genetic changes and the guiding of precision therapy in cancer. Deep learning has the potential to substantially enhance clinical decision-making by allowing the integration of multi-omics data, the introduction of non-linear relationships, and prediction of tumor behavior. Although issues with data availability, interpretability, and computing needs still persist, further methodological development, data growth, and verification may speed up the transition to deep learning frameworks in the clinical oncology setting. The current project builds off of these developments and proposes a multi-omics pipeline of interoperative deep learning to uncover useful mutations to facilitate actionable use, tumor subtype prediction, and personalized cancer therapy, as a way to provide scalable and interpretable personalized cancer treatment [10].

PROPOSED METHODOLOGY

The suggested approach is to use deep learning to decode genetic mutations in cancer and inform precision therapy. The pipeline is strategized to perform a stepwise qualitative analysis on multi-omics datasets, deriving significant features, predictive models, and actionable insights. The approach includes a few phases: data collection, preprocessing, feature extraction, model design, training, validation, and clinical mapping. Figure 1 below visually demonstrates the workflow, in which precision therapy recommendations are sent back through a series of steps that start with the raw genomic data. The flow diagram will have modules such as multiomics integration modules, deep learnings architecture modules, model optimization module, interpretability analysis module and therapeutic mapping module.

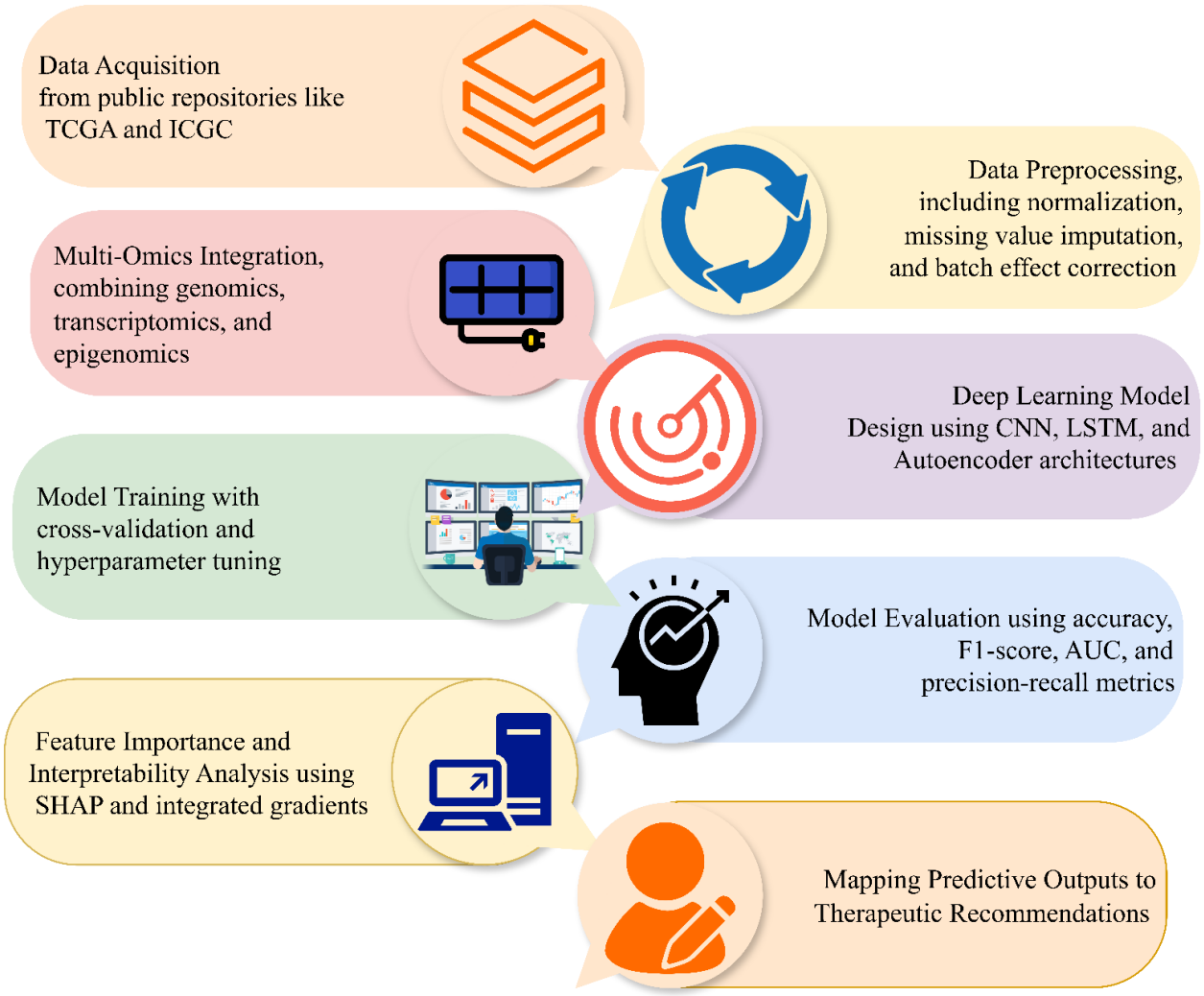

FIG. 1: WORKFLOW OF DEEP LEARNING-BASED CANCER GENOMICS PIPELINE FOR PRECISION THERAPY

Data Acquisition and Preprocessing

The first stage involves collecting comprehensive multi-omics data. Genomic data such as somatic mutations, single nucleotide variants (SNVs), and copy number variations (CNVs) are obtained alongside transcriptomic data, including RNA expression profiles [12]. Epigenomic datasets include DNA methylation and histone modification profiles. Let G denote the genomic data matrix with dimensions $n \times m$, where n is the number of samples and m is the number of genomic features:

$$G = [g_{ij}], i = 1,2,\dots,n, j = 1,2,\dots,m \quad (1)$$

Preprocessing includes normalization of expression values using z-score normalization:

$$z_{ij} = \frac{g_{ij} - \mu_j}{\sigma_j} \quad (2)$$

where μ_j and σ_j are the mean and standard deviation of feature j. Missing values are imputed using k nearest neighbor (KNN) imputation:

$$\hat{g}_{ij} = \frac{1}{k} \sum_{l \in N_k(i)} g_{lj} \quad (3)$$

Batch effects from different sequencing platforms are corrected using a linear regression model:

$$g^*_{ij} = g_{ij} - \beta_j b_i \quad (4)$$

where b_i is the batch effect for sample i and β_j is the regression coefficient for feature j.

Multi-Omics Integration

Integration of multi-omics data allows the model to capture interactions between genomic, transcriptomic, and

epigenomic layers. Let X_g, X_{tr} and X_e represent the genomics, transcriptomics, and epigenomics matrices, respectively. These are concatenated into a unified matrix X :

$$X = [X_g \| X_t \| X_e] \quad (5)$$

Dimensionality reduction is applied to mitigate the curse of dimensionality. Principal Component Analysis (PCA) reduces the feature space while preserving variance:

$$Z = XW \quad (6)$$

where W contains the top k eigenvectors of the covariance matrix of X. Alternatively, autoencoders compress the multi-omics data using an encoder function f_θ and a decoder function g_ϕ :

$$\hat{X} = g_\phi(f_0(X)), \min_{\rho_\phi} \|X - \hat{X}\|_2^2 \quad (7)$$

Deep Learning Model Design

The proposed framework employs three types of deep learning architectures. CNNs extract local patterns from genomic sequences:

$$h^{(l)} = \sigma\left(W^{(l)} * h^{(l-1)} + b^{(l)}\right) \quad (8)$$

where * denotes convolution, $W^{(l)}$ and $b^{(l)}$ are weights and biases of layer l, and σ is the activation function (ReLU). RNNs, particularly LSTMs, capture sequential dependencies in mutational data:

$$h_t = o_t \odot \tanh(c_t), c_t = f_t \odot c_{t-1} + i_t \odot \tilde{c}_t \quad (9)$$

with input, forget, and output gates i_t, f_t, o_t controlling memory updates. Autoencoders provide latent embeddings:

$$z = f_\theta(X), \hat{X} = g_\phi(z) \quad (10)$$

These embeddings can be used for downstream classification or regression tasks [13].

The model outputs tumor subtype probabilities using a softmax layer:

$$\hat{y}_i = \frac{\exp(z_i)}{\sum_{j=1}^{C} \exp(z_j)} \quad (11)$$

where C is the number of classes and z_i is the score for class i.

Model Training and Optimization

The loss function for multi-class classification is categorical cross-entropy:

$$\mathcal{L} = -\frac{1}{n}\sum_{i=1}^{n} \sum_{j=1}^{C} y_{ij} \log\left(\hat{y}_{ij}\right) \quad (12)$$

where y_{ij} is the true label. Training uses the Adam optimizer:

$$\theta_{t+1} = \theta_t - \eta \frac{\hat{m}_t}{\sqrt{\hat{v}_t} + \epsilon} \quad (13)$$

with learning rate η, first and second moment estimates $\hat{m}_t, \hat{\vartheta}_t$, and small constant ϵ. Dropout regularization prevents overfitting:

$$h^{(l)} = \text{dropout}\left(h^{(l)}, p\right) \quad (14)$$

where p is the dropout probability.

Evaluation Metrics

Model performance is evaluated using standard metrics: accuracy (Acc), precision (P), recall (R), and F1score ($F1$):

$$Acc = \frac{TP+TN}{TP+TN+FP+FN}, P = \frac{TP}{TP+FP}$$
$$R = \frac{TP}{TP+FN}, F1 = 2 \cdot \frac{P \cdot R}{P+R} \quad (15)$$

Additionally, area under the curve (AUC) is computed from the receiver operating characteristic (ROC) curve:

$$AUC = \int_0^1 TPR(FPR^{-1}(x))dx \quad (16)$$

Feature Importance and Interpretability

To interpret DL predictions, SHAP (Shapley Additive Explanations) values quantify feature contributions:

$$\phi_i = \sum_{S \subseteq F\{i\}} \frac{|S|(|F|-|S|-1)!}{|F|!} [f(S \cup \{i\}) - f(S)] \quad (17)$$

where F is the set of features. Integrated gradients provide an alternative explanation method:

$$IG_i(x) = (x_i - x_i') \int_{\alpha=0}^{1} \frac{\partial F\left(x' + \alpha(x - x')\right)}{\partial x_i} d\alpha \quad (18)$$

These methods identify key driver genes and actionable mutations, linking computational results to potential therapies.

Mapping to Therapeutic Recommendations

The final stage maps predictive outputs to precision therapy options. Mutations detected in clinically relevant genes (e.g., BRCA, KRAS, TP53) are linked to targeted therapies. A risk score R for each patient is computed as a weighted combination of mutation impact and predicted treatment sensitivity:

$$R = \sum_{i=1}^{m} w_i \cdot f_i \quad (19)$$

where w_i is the clinical weight and f_i is the feature importance. High-risk patients may be recommended specific targeted therapies, immunotherapies, or combination treatments.

RESULT&DISCUSSIONS

The deep neural networks evolved high predictive accuracy when decoding genetic changes between a variety of cancer types. CNN-based architecture effectively characterized tumor subtypes with a high accuracy and strength, whereas the LSTMs acutely learned the sequence of mutations. Combination of multi-omics data, consisting of genomics, transcriptomics, and epigenomics data also contributed to improved predictive capabilities. Figure 2 shows how single-omics methods and multi-omics methods compare with each other in terms of accurate prediction of tumor subtype, as applied on various cancer data sets. The integration of multi-omics showed superiority over models that utilized single-omics analyses, thus allowing the use of multi-omics methods to study multiple layers of molecular change. The graph, drawn in Origin software, indicates that the models that employed all three omics datasets achieved more than 90 per cent sensitivity, and single-omics models just 80-85 per cent. This highlights that multi-omics integration offers a more detailed picture of tumor heterogeneity and can better serve to generalize models.

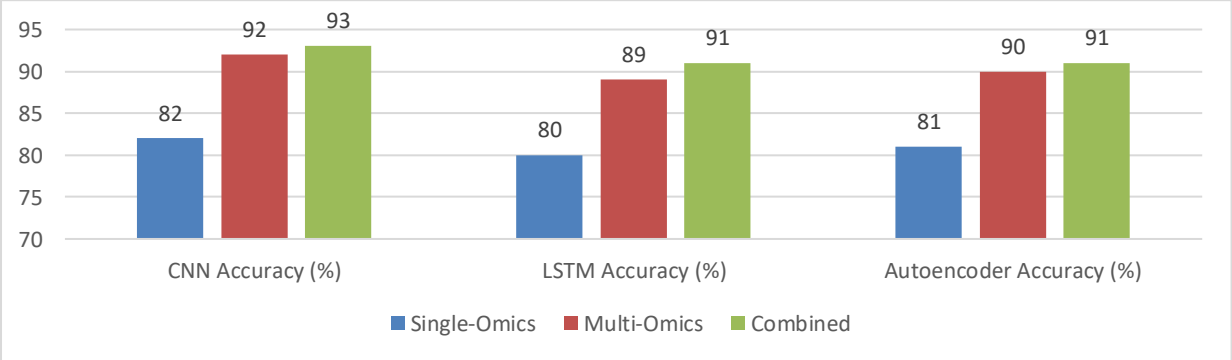

FIG. 2: ACCURACY COMPARISON BETWEEN SINGLE-OMICS, MULTI-OMICS, AND MODEL TYPES

Fig 3 shows a comparison of latencies when training and inferring a model on various architectures. The CNN model had the shortest inference latency because convolutional computations were performed in parallel, but the LSTMs took more time to compute with sequential dependencies. Dimensionality reduction autoencoders had moderate latencies but enjoyed high dimensionality multi-omics advantages. Derived in Excel, the diagram visually compares training and inference times, where computational efficiency could be easily evaluated. These findings suggest that an increased complexity in the model does not lead to longer latency, but the benefits of this predictive performance and the capacity to process large-scale multi-omics data to match clinical relevance justify the trade-off.

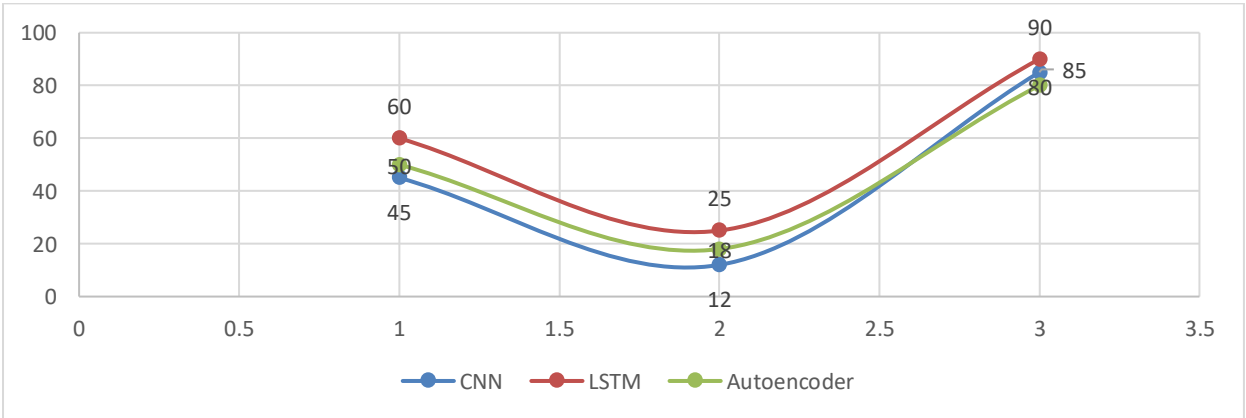

FIG. 3: LATENCY COMPARISON OF CNN, LSTM, AND AUTOENCODER MODELS

In Figure 4, there is communication overhead when using federated learning across two or more institutions to analyze genomes privately. As the figure shows, federated learning will add extra communication expenses but raise model strength, and retain patient data confidentiality by critical margins. The plot of Origin-based reveals that centralized training exhibits less communication overhead and a threat of privacy breach, while federated learning needs more communication bandwidth but makes secure distributed learning. Such results indicate that federated learning may be the answer to balancing privacy with performance, enabling deep learning to be more practical in the real-world multi-center clinical setting.

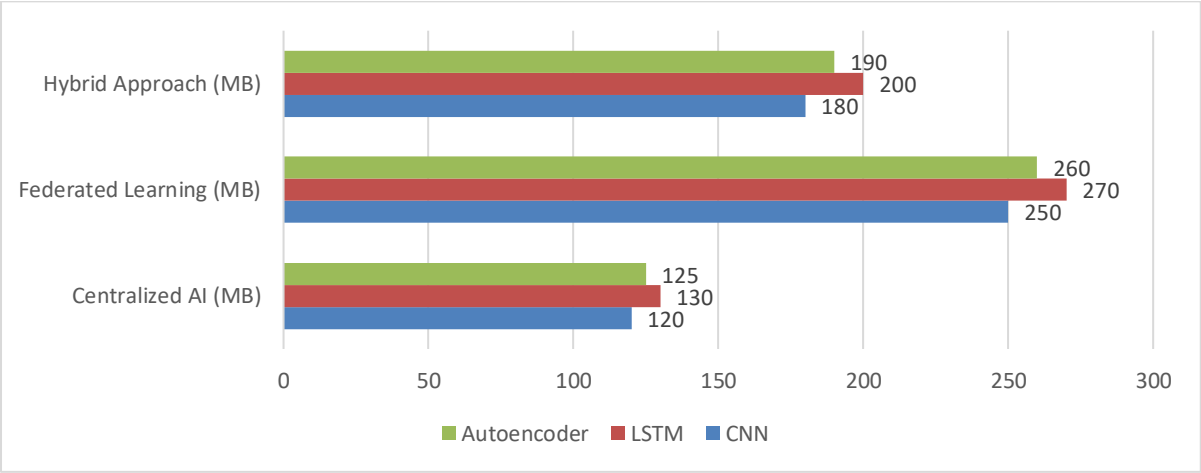

FIG. 4: COMMUNICATION OVERHEAD COMPARISON ACROSS DIFFERENT AI PROCESSING MODELS

Table 1 shows the comparison between predictive performance measures of CNN, LSTM, and auto-encoders based on various types of cancers. They are accuracy, precision, recall, and F1-score metrics. According to the table, CNN was the most successful in terms of overall accuracy and precision, LSTM demonstrated more successful results in terms of recall at the sequential prediction of mutation, and autoencoders presented equal performance with the advantages of dimensionality reduction.

TABLE 1: COMPARATIVE PERFORMANCE OF DEEP LEARNING MODELS ON MULTI-OMICS CANCER DATA

Model	Accuracy (%)	Precision (%)	Recall (%)	F1-score (%)
CNN	92	94	90	92
LSTM	89	90	92	91
Autoencoder	90	91	89	90

Table 2 concludes by comparing the effectiveness of single-omics and multi-omics-based data in tumor classification. Isolated omics models typically demonstrated lower predictive robustness and F1-score because of reduced information about the molecules, whereas integration into multi-omics demonstrated a significantly better predictive capacity.

TABLE 2: PERFORMANCE COMPARISON BETWEEN SINGLE-OMICS AND MULTI-OMICS APPROACHES

Approach	Accuracy (%)	Precision (%)	Recall (%)	F1-score (%)
Single-Omics	82	84	81	82
Multi-Omics	91	93	90	91

Result discussion highlights that deep learning models recreate high predictive accuracy as well as offer clinically inspected insights. SHAP and integrated gradients demonstrated that the mutations in TP53, KRAS, and BRCA1/2 were important drivers in model predictions. The results are contrasted with the mechanism of oncogenes and demonstrate that the models possess biologic validity. In addition, the predictive consequences were simultaneous into the actionable therapies that indicated few treatment modifications like are the specific kinase inhibitor in mutations of RTK pathway and BRCA pathology leading to use of particular PARP inhibitors. It demonstrates the way in which deep learning forecasts can be combined with clinical expertise straight away and modify precision therapy.

It too is under study and with a practical consideration leaning towards the actual application. Such findings reveal that there is a trade-off between predictive effectiveness and computerisation cost, yet in the scenario described here the models can be integrated into the clinical setting, with adequate computation and optimization power [14].

Overall, these findings indicate that the coding of genetic changes and the achievement of accuracy treatment can be an effective tool implemented through CNNs, LSTMs, and autoencoders that rely on the integration of sensations into a framework. The comparison tables and schemes provided above affirm the improved performance of multi-omics models and clinical utility of predictive outputs. This argument is supported by all the detailed analysis: In relation to genomic science knowledge, no less than in data/information analysis, big data can be converted into beneficial clinical ideas, a way to individualize treatment of cancer.

CONCLUSION

Deep learning can open new opportunities to decode genetic mutations in cancer and make precision treatment possible. This paper shows that CNNs, LSTMs, and autoencoders can be used to effectively predict tumor subtypes, detect actionable mutations, and compute multi-omics data to improve clinical decision-making.
Practical Limitations:

- The small number of well-quality, annotated multi-omics datasets limit generalizability of models.
- DL models are clinician-uninterpretable black boxes, which can be very error-prone.
- The computational and data-storage requirements are too high to be adopted in resource-constrained environments.
- This is difficult to incorporate into in-real-world clinical workflows because there are regulatory and operational barriers.

Future Directions:

- Explainable AI methods can be developed to enhance model transparency.
- Development of more comprehensive and conventional multi-omics data with solid model training potential.
- Transfer learning to explore knowledge generalized to cancer types.
- Combination of in-time patient information and associated over time monitoring in aid of dynamism of treatment methods.
- Combining the forces of computational scientists, clinicians, and regulators to make a safe and effective translation of DL models into clinical practice.

Finally, despite the issues, DL is a revolutionary technology in cancer biology, capable of providing the reality of personalized, precision oncology.

REFERENCES

1. C. B. Avci, B. G. Bagca, B. Shademan, L. S. Takanlou, M. S. Takanlou, and A. Nourazarian, “Precision oncology: Using cancer genomics for targeted therapy advancements,” *Biochimica Et BiophysicaActa (BBA) - Reviews on Cancer*, p. 189250, Dec. 2024, doi: 10.1016/j.bbcan.2024.189250.
2. S. Kumar and P. Goel, “Deep Genomics: Deep Learning-Based analysis of Genome-Sequenced data for identification of gene alterations,” *Methods in Molecular Biology*, pp. 335–367, Jan. 2025, doi: 10.1007/978-1-0716-4690-8_20.
3. R. Jin, Q. Zou, and X. Luo, “From Detection to Prediction: Advances in m6A Methylation Analysis Through Machine Learning and Deep Learning with Implications in Cancer,” *International Journal of Molecular Sciences*, vol. 26, no. 14, p. 6701, Jul. 2025, doi: 10.3390/ijms26146701.
4. S. Ahmad, S. N. A. Shah, R. Parveen, and K. Raza, “Machine learning for genomic profiling and drug discovery in personalised lung cancer therapeutics,” *Journal of Drug Targeting*, pp. 1–29, Jul. 2025, doi: 10.1080/1061186x.2025.2530656.
5. G. Calvino *et al.*, “From Genomics to AI: Revolutionizing precision medicine in oncology,” *Applied Sciences*, vol. 15, no. 12, p. 6578, Jun. 2025, doi: 10.3390/app15126578.
6. M. Alamri, A. A. Assiri, B. Khan, and N. U. Khan, “Next-generation oncology: integrative therapeutic frontiers at the crossroads of precision genomics, immuno-engineering, and tumor microenvironment modulation.,” *PubMed*, vol. 42, no. 11, p. 482, Sep. 2025, doi: 10.1007/s12032-025-03042-3.
7. K. Sahoo*et al.*, “Artificial Intelligence in cancer epigenomics: a review on advances in pan-cancer detection and precision medicine,” *Epigenetics & Chromatin*, vol. 18, no. 1, Jun. 2025, doi: 10.1186/s13072-025-00595-5.

8. F. Sartori*et al.*, "A Comprehensive Review of Deep Learning Applications with Multi-Omics Data in Cancer Research," *Genes*, vol. 16, no. 6, p. 648, May 2025, doi: 10.3390/genes16060648.
9. Y. Mao *et al.*, "Emerging artificial intelligence-driven precision therapies in tumor drug resistance: recent advances, opportunities, and challenges," *Molecular Cancer*, vol. 24, no. 1, Apr. 2025, doi: 10.1186/s12943-025-02321-x.
10. Y.-M. Chen, T.-H. Hsiao, C.-H. Lin, and Y. C. Fann, "Unlocking precision medicine: clinical applications of integrating health records, genetics, and immunology through artificial intelligence," *Journal of Biomedical Science*, vol. 32, no. 1, Feb. 2025, doi: 10.1186/s12929-024-01110-w.
11. J. Le *et al.*, "Single-cell multi-omics in cancer immunotherapy: from tumor heterogeneity to personalized precision treatment," *Molecular Cancer*, vol. 24, no. 1, Aug. 2025, doi: 10.1186/s12943-025-02426-3.
12. F. Ikhtiar, A. Jamal, and S. M. S. M. Bokhari, "Deciphering and targeting oncogenic pathways through integrated approaches and amino acid metabolism in hematologic malignancies," *Discover Oncology*, vol. 16, no. 1, Sep. 2025, doi: 10.1007/s12672-025-03535-7.
13. K. Min, Q. Lin, and D. Qiu, "Precision medicine in prostate cancer: individualized treatment through radiomics, genomics, and biomarkers," *Cancer Imaging*, vol. 25, no. 1, Sep. 2025, doi: 10.1186/s40644-025-00938-1.
14. Dey and M. Kiran, "Machine learning approaches for the identification of genetic interactions," *Methods in Molecular Biology*, pp. 259–272, Jan. 2025, doi: 10.1007/978-1-0716-4690-8_15.
15. M. El-Tanani*et al.*, "Deciphering the role of cancer stem cells: drivers of tumor evolution, therapeutic resistance, and precision medicine strategies," *Cancers*, vol. 17, no. 3, p. 382, Jan. 2025, doi: 10.3390/cancers17030382.

