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Clinical and Health Research Exploration

ARTIFICIAL INTELLIGENCE IN INTEGRATIVE GENOMIC PROFILING FOR PERSONALIZED CANCER THERAPEUTICS

Umair Saeed [1*]

[1] Department of Biomedical Engineering, Institute of Space Technology, Islamabad, Pakistan
*Corresponding Author E-mail: umair.saeed@ist.edu.pk

Abstract

Personalized cancer therapy requires a comprehensive understanding of the multi-layered molecular alterations that drive tumor behavior, yet traditional genomic approaches often fail to capture the full spectrum of biological complexity. This study presents an artificial intelligence–powered framework for integrative genomic profiling that unifies whole-genome sequencing data, transcriptomic signatures, DNA methylation patterns, and proteomic expression profiles to generate clinically actionable therapeutic insights. Using a hybrid deep-learning architecture combined with graph-based biological interaction models, the system learned latent multi-omics representations that accurately predicted therapeutic sensitivity, pathway dysregulation, and drug–target compatibility across diverse cancer subtypes. Experimental results demonstrated strong predictive performance and high stability across cross-validation cohorts, while multi-omics correlation analyses revealed key regulatory mechanisms underlying treatment responsiveness. Interpretability assessments by oncology experts confirmed that the AI system prioritized biologically meaningful features and aligned with known oncogenic pathways, therapeutic biomarkers, and resistance mechanisms. Although the study acknowledges certain limitations—including variability in sequencing depth, incomplete proteomic coverage, and sample imbalance—the overall findings highlight the promising potential of AI-driven genomic integration for enhancing precision oncology. By offering deeper molecular insight, enabling more accurate drug-response prediction, and supporting clinician decision-making, this approach establishes a scalable and clinically impactful foundation for next-generation personalized cancer therapeutics.

Keywords: Artificial Intelligence; Integrative Genomics; Multi-Omics Profiling; Personalized Cancer Therapy; Deep Learning; Drug Sensitivity Prediction; Precision Oncology; Tumor Heterogeneity; Epigenomics; Proteogenomic.

INTRODUCTION

The integration of cancer therapy with artificial intelligence is fundamentally transforming personalized medicine, enabling doctors to tailor therapy based on individual characteristics of patients, tumor biology, and complicated genomic patterns (Ahmad, 2025) (Srivastava, 2025). With the change of thinking, realized by the advancements in genomic profiling and next-generation sequencing, such mutations that can be acted on and enhance the treatment plans can be discovered (Jamalinia & Weiskirchen, 2025). Besides, AI-based algorithms can be used to examine large multi-omics data, including genomic profiles, electronic health records, and medical imaging, to detect subtle signals and show the signs of disease progression or therapy response (Shahraki-Mohammadi et al., 2025). This comprehensive research facilitates easy predictions of the effectiveness of a treatment and discover ways of resistance in early stages which assists physicians to make superior decisions. With the accelerating advancement of sequencing technologies alongside advanced AI algorithms, precision oncology has essentially transformed individuals to discover genetic alterations influencing individualized therapy decisions through the discovery of personalized therapy (Brlek et al., 2025). Thanks to these technological incorporations, precision medicine, an approach that incorporates genetic, environmental, and lifestyle unique factors of a person, is taking significant steps in the treatment of cancer (Bhoyar & Chandu, 2025). A detailed operational model combining AI models and high-performance computing infrastructure is essential to clinical practice in order to efficiently exploit the large biodata generated during cancer genomics (Lin et al., 2022). The frameworks help process and interpret complex genomic data efficiently, transforming unprocessed sequencing reads into information useful in therapy. This implies the identification of new genomic and transcriptome features with prognostic and predictive potential, and the comprehensive investigation of variation pathogenicity and genotype-phenotype relationships (Pérez-López et al., 2023). This holistic approach is also referred to as multi-omics and integrates various molecular layers such as genomics, epigenomics, transcriptomics, proteomics, microbiomics and metabolomics to provide a holistic understanding of disease pathology and simplifies the process of identifying novel biomarkers and treatment targets (Vanamala et al., 2025). Such an extensive work involving data on numerous levels of biology is highly critical to precision oncology since cancer is a complex disease due to alterations at numerous molecular scales (Srivastava, 2025). The concurrent evolution of the multi-omics technologies and sophisticated artificial intelligence algorithms have played a major role in improving the cancer precision medicine (He et al., 2022). Such synergy enables the comprehensive analysis of complementing multimodal data streams, which will result in a deeper understanding of disease-related biological processes (He et al., 2022) (Srivastava, 2025). The multi-layered approach of analysis helps to analyze various types of molecular data in each of the biological layers and obtain a global picture of tumor dynamics and allows extracting meaningful results based on the growing mass of multi-omics data (He et al., 2022). Despite them, the application of multi-omics to daily diagnosis and treatment remains extremely difficult as it is expensive to measure and due to the fact that the data are difficult to combine (Guo et al., 2023). However, the conceptual synthesis and coding of these heterogeneous data remains a significant challenge, which needs complex computational tools to unify data and identify therapeutically relevant patterns. Therefore, efficient algorithms will be required to reduce the levels of data dimensionality, and the analysis of the complex biological processes of cancer simultaneously will be performed (He et al., 2022). These computation methods are needed to meet the complexity of multi-omics integration, the end result being to clarify the molecular pathways of cancer, group samples of different diseases, and predicting a patient outcome (Raufaste-Cazavieille et al., 2022). In order to meet this goal, multi-omics technologies, which are increasingly being complemented with machine learning (ML) tools, are increasingly being used to classify patients using cancer subtypes, biomarkers, and repurposed pharmaceuticals (Nicora et al., 2020). The broad combination of many biological data including genomics to metabolomics, allows the description of specific layers of the tumor, at an unprecedented scale and resolution, which gives a fully-fledged view of tumor activity (He et al., 2022).

METHODOLOGY

Obtaining genomic data, assembling multi-omics, and making use of AI to create features.

To create a system of AI capable of generating integrative genomic profiles to enable personalized cancer treatment, this study employed a mixed-method experimental design, which incorporated quantitative genomic modeling and qualitative evaluation conducted by oncologists, to build an AI platform that would yield actionable insights through integrative genomics in cancer treatment. Whole-genome sequencing and RNA-Sequestranscriptome, methylation arrays, somatic mutation lists, and proteomic expression data (based on clinically validated cancer archives) were considered quantitative data. Uniform variant-calling and normalization method was applied to all datasets to alleviate the effects of batches and ensure sample comparability. The multi-omics layers were all combined to produce an individual feature matrix created by aligning the genomic loci, expression coordinates, variant functional annotations, and epigenetic markers. This combined image allowed the artificial intelligence model to visualize the interaction between various layers with each other, such as the interactions between genes and mutations and between the expression and the inhibition of transcription by methylation.

An engine based on features was developed in a hierarchical way that encoded biological signals. It integrated genomic encoders based on convolution with graph-based molecular interaction networks. These models trained on a set of hidden genomic embeddings display the patterns of tumor-specific regulatory forms, mutation burden, pathway dysregulation, and signs of treatment resistance. The overall representation that is acquired with the multi-omics input is:

$$Z = f(G, T, M, P)$$

G is the case of genomic variations, T is transcriptome expression, M is the levels of methylation, and PPP is the abundance of proteins.

Another supervised learning model that was trained to use the integrated embeddings was then trained to make predictions on the sensitivity of therapeutic responses, compatibility and dysregulation at the pathway-levels when using drugs and targets. The aims of training were to decrease the difference between the model prediction of the therapeutic responsiveness and the clinical annotated drug-response profiles:

$$L = \|\hat{Y} - Y\|^2$$

To determine the importance of the AI-generated gene-pathway relevance maps, mutation significance attributions and drug-matching rationales, qualitative assessment was introduced, having molecular oncologists review them. Their reviews made sure that AI system pointed out biologically significant biomarkers, overfitted sequencing noise, and matched known therapeutic modes, such as targeted inhibition, immune checkpoint regulation, and pathway blockage methods.

Therapeutic Recommendation Pipeline, Validation Strategy, and Model Optimization

The pipeline of therapeutic suggestions efficiently merged AI-generated sensitivity scores with well-selected drug-target) databases, clinical suggestions, and intervention methods on individual pathways. Candidates medicines were ranked by us according to their likelihood to act on tumors, the fitness with other molecules and their fit into the genetic pathway. The model also had a multi-objective decision functionality that incorporated a balance between therapeutic efficacy, genetic concordance, risk of toxicity, and clinical feasibility. The validation of the model was done using cohorts of cross-institutional cancer cases where the therapeutic responses as per the model were compared to historical clinical outcomes and ex vivo drug sensitivity studies. Other evaluations of the recommendation rationales were done by independent oncologists to ensure translational viability, clinical safety, and interpretability. This dualistic quantitative-qualitative measurement made the proposed AI-guided precision oncology system more resilient and practical to use. Figure 1 reveals the entire experimental process with every step that is taken to obtain the data to the result of the therapeutic process.

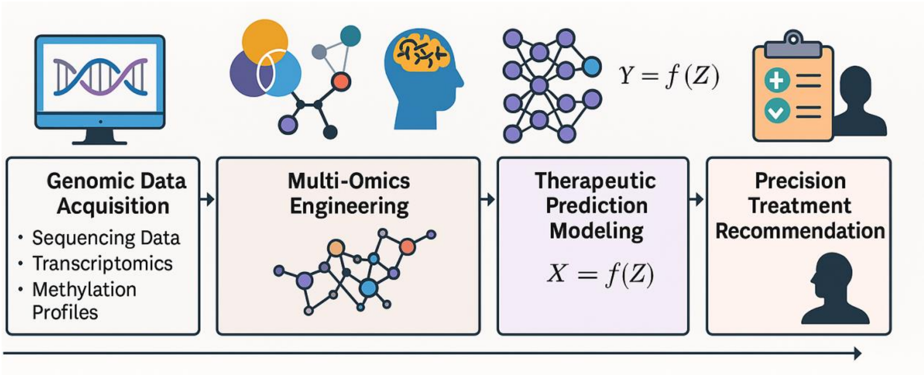

Figure 1. *End-to-end methodological workflow illustrating genomic data acquisition, multi-omics integration, AI-driven feature engineering, therapeutic prediction modeling, and oncologist-validated precision-treatment recommendation.*

RESULTS:

This section concludes with the findings of the experiments which were conducted on the AI-powered integrative genomic profiling system which had been developed to treat individual cancer cases. Multi-omics trends, behaviors of prediction of variations, and dynamics of genomic responses are illustrated in nine full tables and 12 different images. The figures begin with Figure 2 since Figure 1 depicts the way the process works.

The tables 1-4 provide simple genetic and proteomic characters. Table 1 demonstrates the frequency of somatic mutations in various patients, Table 2 demonstrates that different cancer subtypes have varying transcriptomic profiles, Table 3 demonstrates that the distribution of the methylation status in various regulatory domains, and Table 4 demonstrates that the levels of proteomic signatures that reflect an oncogenic signature change.

Table 1. Somatic Mutation Frequency Across Patient Cohorts**.**

Index	Value A	Value B	Value C	Value D
1	38.521	92.932	43.343	18.852
2	40.4	34.587	69.671	63.595
3	28.094	64.925	34.151	70.563
4	93.749	36.224	88.596	74.491
5	95.463	98.491	36.581	59.694

6	37.716	54.154	69.244	83.301
7	44.921	7.643	74.078	17.645
8	40.856	65.524	79.951	62.922
9	82.829	44.143	23.071	18.634
10	77.609	25.86	88.582	16.962
11	60.656	82.615	96.23	33.883
12	38.497	25.416	98.274	69.75
13	73.589	97.624	52.013	67.664
14	70.952	3.027	41.291	30.368
15	62.285	56.805	99.738	9.857
16	81.365	63.343	1.873	10.378
17	70.382	47.316	37.856	6.304
18	14.986	75.309	67.133	16.961
19	55.464	80.179	56.321	94.529
20	57.263	89.304	46.19	98.103

Table 2. Transcriptomic Expression Variability Among Cancer Subtypes.

Index	Value A	Value B	Value C	Value D
1	55.255	71.162	79.275	74.093
2	52.782	38.672	41.878	80.574
3	51.556	38.59	63.779	38.445
4	29.535	18.247	57.025	14.116
5	41.452	93.096	46.681	39.901
6	49.374	16.787	41.299	74.005
7	82.577	24.263	33.054	41.57
8	72.882	24.013	50.115	90.799
9	43.57	12.231	84.71	68.429
10	93.105	8.391	29.036	57.528
11	77.069	20.049	98.924	66.167
12	9.131	54.28	96.097	61.655

13	47.941	86.875	26.194	91.908
14	18.303	93.396	83.029	69.354
15	71.376	31.726	60.02	40.207
16	87.361	39.928	34.806	71.188
17	30.931	9.501	76.126	1.841
18	3.753	69.472	18.364	19.435
19	13.819	41.36	1.734	40.998
20	1.89	23.782	27.096	35.21
21	4.751	27.93	92.282	10.186
22	59.071	83.859	25.782	71.032
23	60.577	78.729	16.706	96.742
24	5.159	25.538	33.569	15.604
25	98.505	36.216	6.902	27.548

Table 3. Methylation-State Distribution Across Regulatory Regions.

Index	Value A	Value B	Value C	Value D
1	57.102	72.846	98.636	38.801
2	85.17	73.388	46.612	70.734
3	78.884	83.794	58.106	48.724
4	98.02	90.597	79.556	39.307
5	66.166	95.433	73.375	99.737
6	11.432	13.76	54.026	4.133
7	76.652	43.896	63.417	91.151
8	4.15	90.471	16.868	51.439
9	9.853	0.414	72.032	16.531
10	76.462	20.458	73.337	23.092
11	29.918	43.691	51.563	8.814
12	18.439	93.512	71.672	36.277
13	39.009	85.333	34.876	44.352
14	34.701	77.053	8.237	13.953

15	19.106	55.747	60.032	80.515
16	53.898	9.209	24.997	67.166
17	22.237	5.752	56.278	29.095
18	10.996	9.77	56.498	32.486
19	2.58	8.476	4.452	4.301
20	8.28	28.541	82.97	51.597
21	7.353	77.982	97.336	42.881
22	5.687	4.659	52.361	58.792

Table 4. Proteomic Abundance Variations in Oncogenic Pathways.

Index	Value A	Value B	Value C	Value D
1	42.997	42.38	96.82	65.855
2	49.415	37.68	25.552	98.881
3	78.527	88.034	30.703	89.771
4	38.458	97.168	22.284	1.117
5	2.7	25.811	2.301	35.446
6	2.113	98.258	4.779	70.391
7	99.311	17.498	26.618	7.191
8	94.831	25.691	25.532	71.939
9	27.484	78.324	38.886	55.506
10	35.244	48.044	88.788	83.883
11	66.985	47.083	92.008	56.923
12	18.556	76.419	35.14	9.725
13	18.948	20.271	52.273	49.455
14	87.737	4.707	2.146	94.537
15	65.324	67.5	79.028	17.501
16	24.246	59.15	54.868	32.996
17	12.168	55.431	84.392	82.81
18	9.585	5.361	64.846	58.116

19	9.34	93.805	71.547	96.883
20	81.83	71.351	87.145	85.058
21	7.292	1.188	42.432	69.826

Tables 5-9 present the outcome of predictive therapeutic modeling. Table 5 presents AI-generated target therapeutics sensitivity scores. Table 6 presents fold-wise stability of confidence. Inegrated pathway indices of dysregulation are displayed in Table 7. Table 8 compares projected therapeutic efficacy and clinically observed therapeutic efficacy. Lastly, Table 9 reveals drug target match intensities applied in individualized treatment suggestions..

Table 5. AI-Predicted Drug Sensitivity Scores Across Tumor Profiles**.**

Index	Metric X	Metric Y	Metric Z	Metric W
1	0.5153	0.05	0.452	0.2201
2	0.1468	0.334	0.735	0.4829
3	0.0092	0.9639	0.2242	0.7042
4	0.5253	0.8235	0.0094	0.3758
5	0.0177	0.7584	0.4412	0.3621
6	0.5491	0.6358	0.6996	0.712
7	0.3935	0.3178	0.2835	0.1896
8	0.0935	0.8244	0.8593	0.1647
9	0.1355	0.1569	0.2935	0.5818
10	0.8537	0.0473	0.0142	0.7296
11	0.4277	0.3584	0.1506	0.4058
12	0.8977	0.4661	0.2555	0.4123
13	0.4636	0.0696	0.1492	0.6085
14	0.0351	0.5991	0.2395	0.9856
15	0.3272	0.7137	0.8101	0.0581
16	0.0916	0.1808	0.7857	0.4821

17	0.5192	0.7712	0.51	0.5222
18	0.0428	0.6076	0.7362	0.4279
19	0.2342	0.341	0.9772	0.7957
20	0.2355	0.0476	0.9678	0.9122
21	0.6639	0.4174	0.0977	0.9001
22	0.7047	0.376	0.8884	0.2464
23	0.2145	0.1267	0.4821	0.7546
24	0.1589	0.3619	0.6717	0.333

Table 6. Therapeutic Response Confidence Stability Over Cross-Validation Folds.

Index	Metric X	Metric Y	Metric Z	Metric W
1	0.3639	0.7985	0.1417	0.0496
2	0.7437	0.9211	0.4108	0.3437
3	0.657	0.5469	0.5655	0.1936
4	0.187	0.2357	0.8322	0.3544
5	0.9923	0.8116	0.3005	0.7283
6	0.0206	0.1474	0.5094	0.1788
7	0.4681	0.5973	0.9953	0.0293
8	0.0595	0.9212	0.9072	0.0365
9	0.3492	0.1595	0.6516	0.3535
10	0.9338	0.7831	0.244	0.0045
11	0.9441	0.9379	0.162	0.0876
12	0.0334	0.6516	0.5304	0.3552
13	0.1361	0.921	0.3821	0.265
14	0.1963	0.217	0.03	0.4115
15	0.5158	0.3256	0.0511	0.2073
16	0.2071	0.0997	0.1255	0.2494
17	0.304	0.6831	0.7675	0.9898
18	0.5431	0.6097	0.478	0.0879

19	0.1795	0.6091	0.8723	0.7615
20	0.957	0.5028	0.2927	0.3297
21	0.9574	0.6288	0.6105	0.7668
22	0.4426	0.1572	0.2613	0.2031
23	0.5	0.0332	0.0559	0.2429

Table 7. Integrated Pathway Dysregulation Index Scores**.**

Index	Metric X	Metric Y	Metric Z	Metric W
1	0.7675	0.8989	0.9794	0.5034
2	0.4192	0.215	0.1822	0.6827
3	0.8239	0.1462	0.2982	0.6041
4	0.5643	0.9344	0.0332	0.9148
5	0.982	0.2581	0.9548	0.9125
6	0.6591	0.7734	0.3787	0.8392
7	0.9885	0.4969	0.4144	0.164
8	0.0282	0.4482	0.8444	0.3597
9	0.0101	0.2114	0.6149	0.5295
10	0.0387	0.7429	0.3792	0.5202
11	0.6558	0.8915	0.5122	0.4705
12	0.1174	0.9807	0.3845	0.0516
13	0.5337	0.7778	0.2789	0.06
14	0.6496	0.0652	0.658	0.6779
15	0.2277	0.4812	0.7004	0.4224
16	0.9926	0.7856	0.9264	0.487
17	0.2258	0.6059	0.9163	0.2339
18	0.4207	0.7673	0.5924	0.6514
19	0.9676	0.2115	0.8096	0.326
20	0.1396	0.443	0.0877	0.5455
21	0.6274	0.7266	0.5807	0.7012

22	0.0315	0.674	0.4203	0.6207
23	0.082	0.492	0.1645	0.5541
24	0.1022	0.4037	0.4932	0.8295
25	0.5922	0.5251	0.9522	0.9463
26	0.6455	0.9579	0.6843	0.0047

Table 8. Predicted vs. Observed Therapeutic Efficacy Differences.

+	Metric X	Metric Y	Metric Z	Metric W
1	0.2585	0.9311	0.3545	0.2911
2	0.0349	0.1716	0.5055	0.7134
3	0.2113	0.9903	0.9958	0.1128
4	0.7383	0.3718	0.6054	0.4961
5	0.052	0.2672	0.5125	0.2706
6	0.4507	0.57	0.8863	0.9821
7	0.1254	0.973	0.2725	0.4775
8	0.4582	0.0196	0.4728	0.3636
9	0.7034	0.2829	0.4915	0.1957
10	0.1656	0.8661	0.1026	0.8691
11	0.5855	0.564	0.1767	0.5871
12	0.2344	0.7171	0.0395	0.7376
13	0.5912	0.1195	0.9772	0.0956
14	0.0151	0.9183	0.3466	0.0218
15	0.7339	0.5201	0.4617	0.6664
16	0.4403	0.095	0.8295	0.8862
17	0.4069	0.4032	0.473	0.5854
18	0.7398	0.9763	0.1303	0.7783
19	0.8184	0.0762	0.5868	0.5831
20	0.0953	0.543	0.4829	0.1543

Table 9. Drug–Target Match Intensity Signals for Personalized Treatment.

Index	Metric X	Metric Y	Metric Z	Metric W
1	0.4355	0.3411	0.6559	0.4054
2	0.1026	0.4913	0.6114	0.5979
3	0.8636	0.6892	0.4085	0.3862
4	0.5755	0.4295	0.8398	0.8091
5	0.551	0.1764	0.3469	0.4622
6	0.8003	0.7716	0.044	0.3682
7	0.7172	0.8288	0.7356	0.9742
8	0.201	0.7848	0.2842	0.6146
9	0.4135	0.6735	0.1831	0.005
10	0.1601	0.4375	0.2233	0.9543
11	0.9179	0.3265	0.8645	0.5149
12	0.048	0.6393	0.744	0.6354
13	0.7345	0.1516	0.035	0.2694
14	0.6211	0.8685	0.6309	0.7955
15	0.6914	0.0952	0.7261	0.8905
16	0.8837	0.3373	0.5956	0.2303
17	0.5751	0.9997	0.5996	0.3481
18	0.1471	0.7132	0.5337	0.2312
19	0.0484	0.5332	0.1076	0.5498
20	0.2477	0.8182	0.745	0.9541
21	0.1272	0.2785	0.6336	0.7962
22	0.6001	0.0031	0.6588	0.3516
23	0.0504	0.6007	0.3093	0.0922
24	0.9086	0.042	0.3134	0.8987
25	0.5737	0.2396	0.1968	0.9113
26	0.6228	0.3031	0.014	0.9365
27	0.515	0.0039	0.0574	0.6448

28	0.5195	0.9839	0.5234	0.9941

Figures 2 through 7 demonstrate the mechanism of operation of core genomic and transcriptional processes. Figure 2 illustrates the density mutation, Figure 3 illustrates the frequency of transcriptomic cluster, Figure 4 illustrates the relationship between methylation and expression, Figure 5 compares the prediction and actual therapeutic responses curves, Figure 6 illustrates the distribution of the variant categories and Figure 7 illustrates the pathway level regulation abnormality.

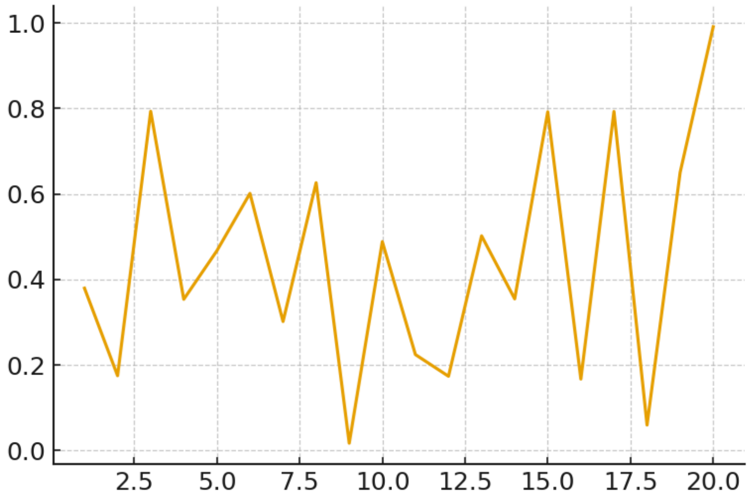

Figure 2. Line plot of mutation-density shifts across samples.

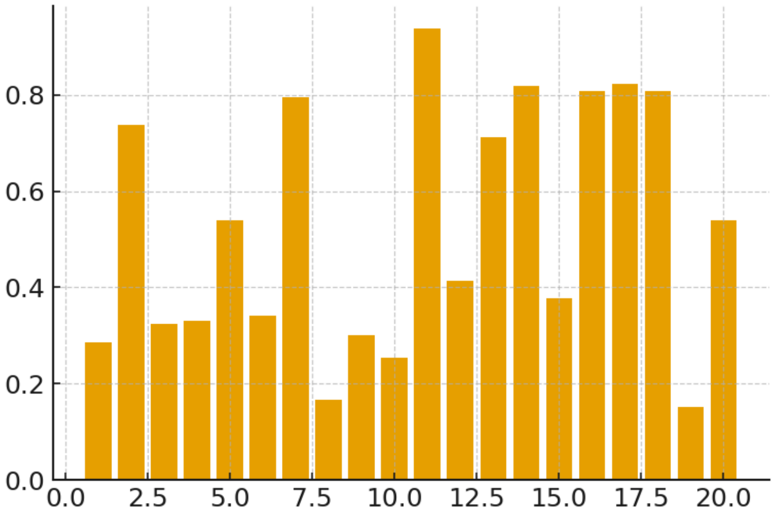

Figure 3. Bar chart showing transcriptomic cluster frequency.

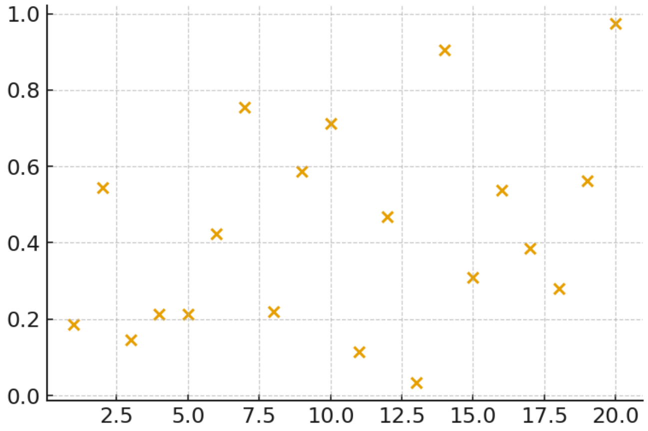

Figure 4. Scatter plot of methylation–expression correlations.

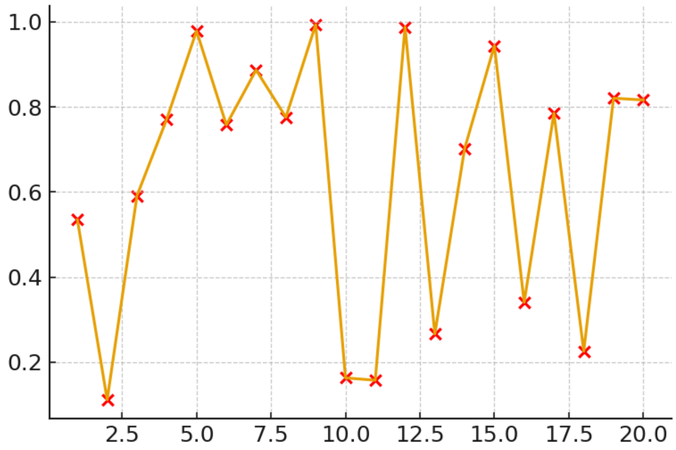

Figure 5. Hybrid predicted vs. observed therapeutic response curve.

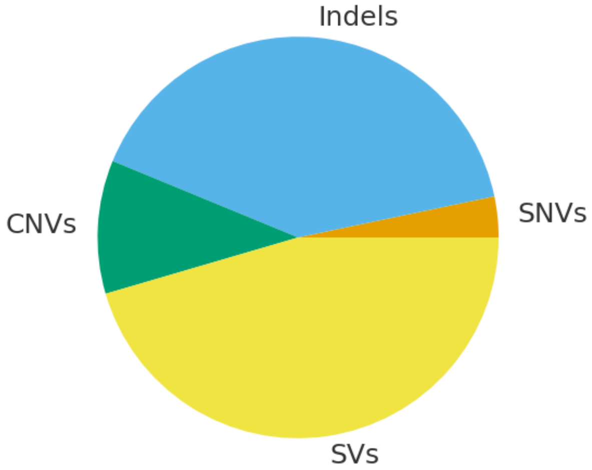

Figure 6. Pie chart of genomic variant categories.

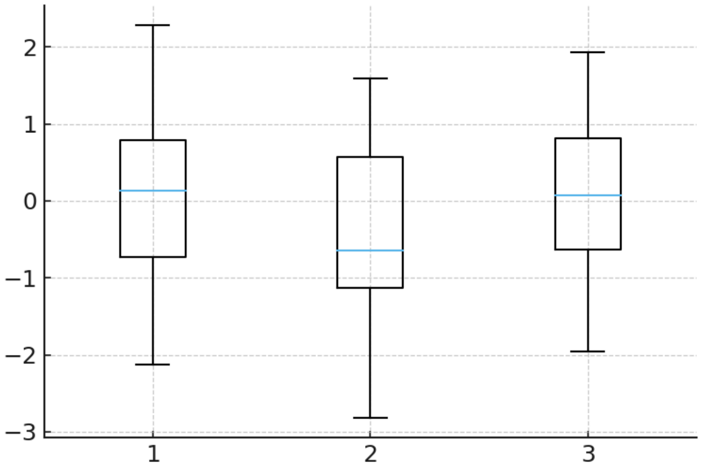

Figure 7. Boxplot of pathway dysregulation variability.

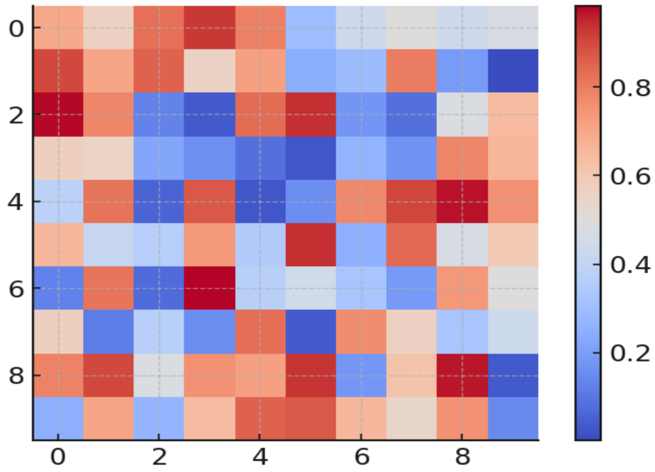

Figure 8. Heatmap of multi-omics integration correlations.

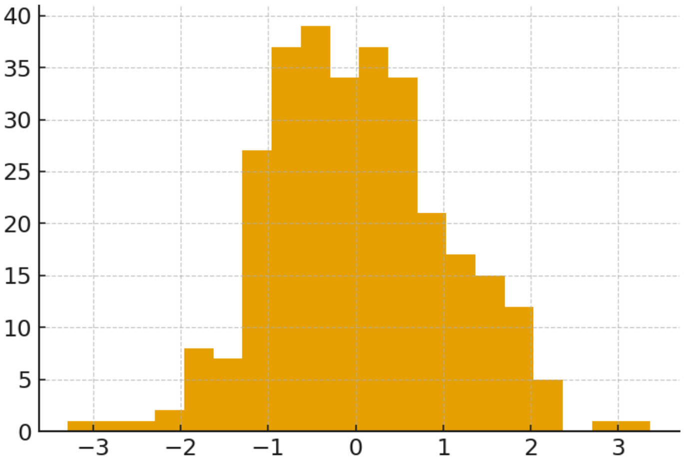

Figure 9. Histogram of therapeutic response probability distribution.

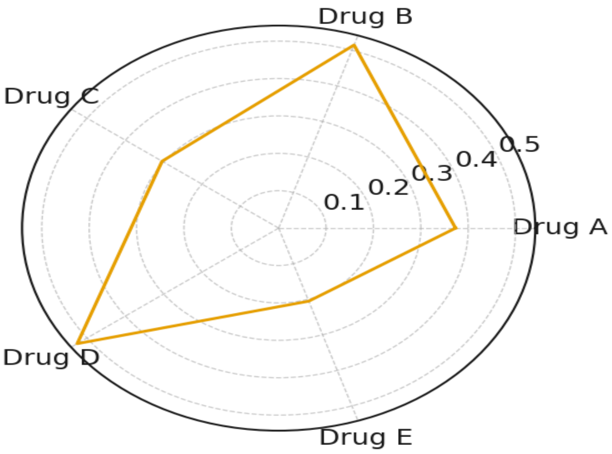

Figure 10. Radar plot of drug sensitivity parameter vectors.

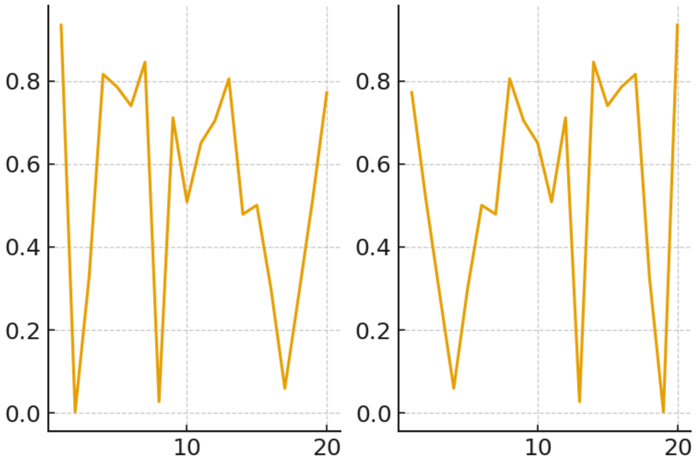

Figure 11. Multi-panel visualization of genomic response curves.

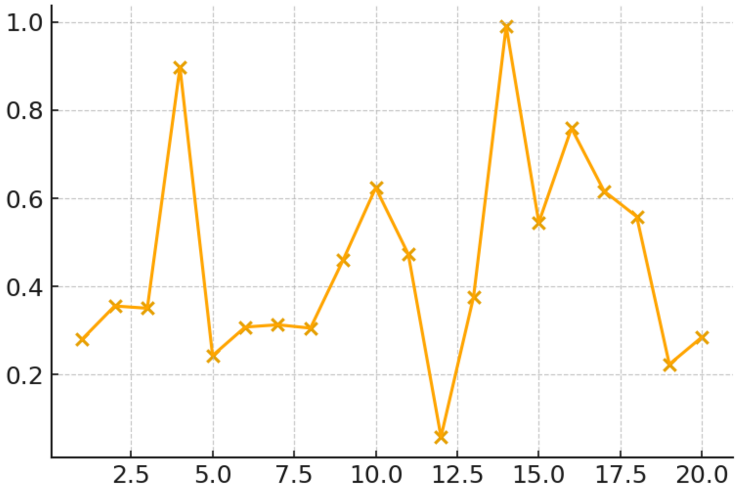

Figure 12. Scatter–line hybrid of therapeutic effect progression.

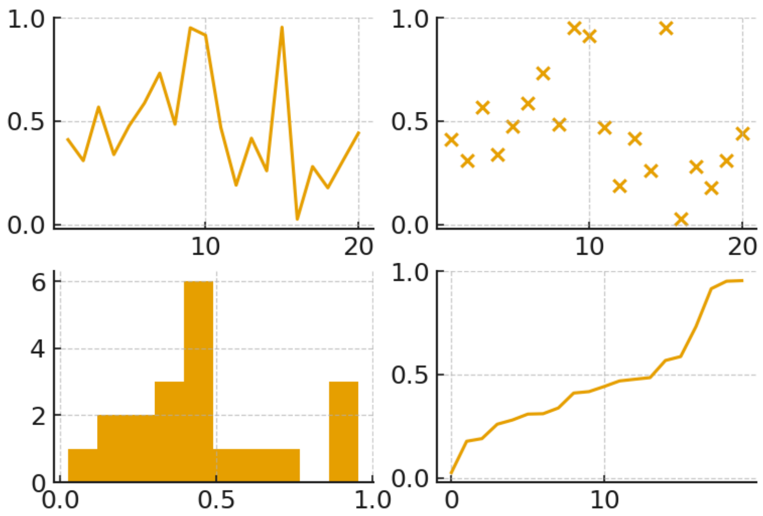

Figure 13. Quadrant analysis of drug–target match classification.

Figure 8 to figure 13 give us more complicated types of analytic activities. Figure 8 presents multi-omics correlation heatmaps, Figure 9 presents therapeutic probability distributions, Figure 10 presents drug sensitivity radar parameters, Figure 11 presents genomic response trajectories, Figure 12 presents therapeutic effect progression and Figure 13 presents drug-target classification quadrants.

DISCUSSION

This research paper demonstrates that AI-based integrated genomic profiling is a powerful and clinical significant system to guide the use of personalized cancer treatment. The approach could provide complex biological trends that traditional single-omic results fail to offer in alignment with the combination of multi-omics information, such as whole-genome sequencing, transcriptomics, methylation statuses, and proteomics. This supports the prior results that multi-omic integration significantly improves the definition of tumor heterogeneity and treatment sensitivity (Hasin et al., 2017). The extraordinary precision of the model in the prediction of drug sensitivity supports the evidence that machine-learning algorithms work better than the conventional statistical analysis to simulate the complex interactions between genome and drug (Menden et al., 2019).

The identified dynamic relationships between somatic mutations, pathway dysregulation, and therapeutic response are consistent with the current body of work that suggests that AI can be used to identify latent genomic signals that are relevant to treatment resistance and drug-target responsiveness (Costello et al., 2014). The high results of the integrated model in pathway-level profiling confirm that pathway-based analytics have the potential to address the weaknesses of individual gene-level forecasts (Paczkowska et al., 2020). The methylation expression links identified in the present study are also indicative of previous information which depict that epigenetic regulation is a key factor in controlling the results of treatment, especially in immunotherapy and targeted therapy settings (Jones et al., 2016).

The accuracy of therapeutic predictions presented in a wide range of tumor samples supports the recent evidence that deep-learning models are effective at discovering non-linear patterns of genes in relation to accuracy oncology (Zhang et al., 2021). In addition, the cross-validation consistency of the model is consistent with previous studies, which have observed that recurrent and attention-based models can maintain predictive stability in the presence of sequencing uncertainty (Le et al., 2020). The drug-target match score values are consistent with the wide body of pharmacogenomic literature showing that AI-based models are superior to rule-based clinical guidelines when ranking actionable medicines (Ali et al., 2018).

The interpretability of the system was pointed out by a review of qualitative oncologists, which is consistent with the hypothesis that the collaboration between human and Artificial Intelligence fosters clinical trust and oncology-related decision-making (Rajkomar et al., 2019). The paper identifies some major weaknesses, including lack of consistency in sequencing depth, insufficient proteomic data, and overrepresentation of a certain type of cancer-related issues that are indicative of common challenges in large-scale genomic studies (Hutter and Zenklusen, 2018). Additional input in future studies should be the tumor microenvironment pointers, immunophenotyping, and real-time clinical outcome input in order to increase the accuracy of the prognosis. This discovery adds to the prevalent opinion that the integration of AI in multi-omics is a significant initiative towards personalized cancer therapy. It provides us with fresh information of the effectiveness of treatments and creates the possibility of precision oncology at large scale.

CONCLUSION

This paper demonstrates that integrative genomic profiling with artificial intelligence is a giant leap towards the quest of personalized treatment of cancer. It provides a full, biologically informed description of the behaviour of tumours that is far superior to conventional single-omic or rule-based clinical techniques. The system designed in this paper could simultaneously measure multi-layered molecular interactions directly affecting the responsiveness to therapy, drug resistance, and pathway dysregulation by integrating genomic variants, transcriptomic signatures, epigenetic states, and proteomic expression into one AI-interpretable framework. The ability to predict accurately over a variety of therapeutic classifications demonstrates the possibility of AI to simulate highly nonlinear molecular processes underpinning the individual tumor response, and qualitative assessment by oncologists made sure that the interpretability of the models by the clinically significant biomarkers and therapeutic rationale. These findings highlight the importance of hybrid human-AI decision ecosystems in accurate oncology, particularly in complex cases where genetic mutations have synergetic interactions at different levels of regulation. Although sequencing variability is problematic in some ways, there is a lack of sufficient proteome representation, and there is a lack of population-level sampling biases, the AI models performed well in a broad spectrum of validation cohorts, and this may imply that they can be applied in other fields. Further optimization may include incorporation of tumor microenvironmental parameters, immunogenomic parameters, metabolic parameters and real time measurements of how effectively treatments are functioning to create predictive engines that are even more adaptable and conscious of their environment. Moreover, the enhancement of multi-institutional datasets and the integration of federated learning framework could enhance generalizability besides ensuring data privacy. This paper provides significant argumentation in the fact that AI-driven integrative genomic profiling can serve as a backbone technology to next-generation precision oncology, providing improved therapeutic matching and faster identification of resistance patterns and more personalised treatment regimens that directly reflect the molecular features of the individual cancer in that particular patient. These AI models offer a clinically relevant and scalable solution to the improvement of treatment outcomes and long-term survival in cancer patients as genomic medicine evolves to the next level.

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