Research Journal of Cell Sciences

PRIME OPEN ACCESS

Volume 1, Issue 1

Research Article

Date of Submission: 05, May, 2025
Date of Acceptance: 05 June, 2025
Date of Publication: 10 June, 2025

AI-Enhanced Multi-Omics Framework for Predicting Antibiotic Resistance in Pediatric Cancer Patients

Avani Agarwal[1*] and Nineesha Chumbhale[2]

[1]G.D. Goenka Public School, India

[2]Fravashi International Academy, India

***Corresponding Author:**
Avani Agarwal. G.D. Goenka Public School, India.

Citation: Agarwal, A., Chumbhale, N. (2025). AI-Enhanced Multi-Omics Framework for Predicting Antibiotic Resistance in Pediatric Cancer Patients. *Res J Cell Sci*, 1(1), 01-08.

Abstract
Antibiotic resistance poses a critical challenge for pediatric cancer patients whose immune systems are compro- mised by chemotherapy. This paper presents an AI-driven multi- omics framework designed to tackle this issue. Using Convolutional Neural Networks (CNNs), the framework predicts bacterial resistance patterns, while Multi-Task Neural Networks (MTNNs) evaluate patient-specific drug responses. In trials, the CNN model correctly identified resistant bacteria, such as Klebsiella pneu- moniae (with a 92% probability of resistance), and susceptible strains like Escherichia coli (35% probability of resistance). These insights guided the selection of alternative treatments, including colistin and ceftriaxone. This AI-powered approach marks a step forward in personalized antibiotic therapies, aiming to minimize resistance development and improve health outcomes for vulnerable patients.

Introduction
Antibiotic Resistance in Pediatric Cancer Patients
Antibiotics are powerful drugs used to treat life-threatening diseases, but their misuse or overuse can lead to antibiotic resistance, where bacteria adapt and become resistant to the drugs. This is a growing public health issue, as it reduces the effectiveness of antibiotics, making infections harder to treat. Resistant strains of bacteria can spread to others, even those who haven't taken antibiotics.

Key contributors to antibiotic resistance include inappropriate use in humans and animals, antibiotics in food and agriculture, the lack of rapid diagnostic procedures, and environmental contamination by antibiotics [1,2]. In pediatric cancer patients, this issue is particularly concerning. Chemotherapy and radiation treatments weaken the immune system, making patients more susceptible to infections by multidrug-resistant (MDR) pathogens like Pseudomonas aeruginosa and Staphylococcus aureus [3].

Chemotherapy is a crucial treatment for cancer, but it often comes with serious risks for pediatric patients. Among these, Chemotherapy-Induced Neutropenia (CIN) and Febrile Neutropenia (FN) are particularly concerning because they weaken the immune system, making children more vulnerable to infections [4]. These infections are even harder to treat when caused by MDR bacteria, which limit the effectiveness of many antibiotics. Addressing this challenge requires new ways to predict and manage antibiotic resistance, as well as a deeper understanding of how it develops. The use of antibiotics and immunosuppressive therapies disrupts the microbiome, which can encourage the growth of resistant pathogens [5,6]. Pediatric cancer patients also exhibit different pharmacokinetics and pharmacodynamics than adults, which can influence antibiotic efficacy and resistance development [7]. Immunocompromised pediatric cancer patients are particularly vulnerable to bacterial infections, making antibiotic resistance a critical concern. Infections that

might be mild for healthy individuals can become life-threatening for them. This is especially true in pediatric leukemia cases, where antibiotic resistance can severely impact treatment, leading to higher rates of complications and mortality [8]. Antibiotic resistance in clinical settings leads to the failure of first-line treatments, delays in infection control, and increased mortality rates among pediatric cancer patients. These patients depend on antibiotics to prevent and manage infections, but when treatments fail, the risk of severe complications like sepsis rises. This not only increases sepsis-related mortality but also healthcare costs due to longer hospital stays and the need for intensive care [9-12]. Developing personalized treatments based on resistance profiles is essential for improving outcomes and alleviating the burden on healthcare systems.

The Role of Multi-Omics in Medical Research

Multi-omics approaches integrate genomics, transcrip- tomics, proteomics, and metabolomics to analyze biological systems, helping researchers understand interactions between genes, proteins, and metabolites. This approach is crucial for studying antibiotic resistance and developing effective treatments. In pediatric cancer research, multi-omics is used to identify biomarkers, classify cancer subtypes, and pre- dict treatment responses. It also aids in uncovering resis- tance mechanisms and molecular signatures that indicate resistance patterns, enabling personalized treatment Strategies.

Techniques like mass spectrometry-based proteomics and metabolomics further enhance the study of antibiotic resistance in infections affecting immunocompromised pediatric cancer patients. By integrating multi-omics data, researchers gain insights into bacterial resistance, identifying new therapeutic targets and strategies to combat resistance. In pediatric cancer patients, this approach improves resistance prediction and infection control, leading to more effective treatments [13].

Advancements in artificial intelligence (AI) have made it pos- sible to analyze complex datasets more effectively. Machine learning models can process large amounts of biological data to predict which patients are most at risk of resistance, helping doctors choose the best treatments. This personalized approach not only improves outcomes but also reduces unnecessary antibiotic use, slowing the spread of resistance.

Literature Review

Antibiotic Resistance Mechanisms

Antibiotic resistance in bacteria is driven by several key molecular mechanisms, which enable pathogens to survive and proliferate despite antibiotic exposure. These mechanisms are included in the table below.

Mechanism	Description
Efflux Pumps	Efflux pumps are membrane-associated proteins that actively expel antibiotics from bacterial cells, reducing intracellular drug concentrations to sub-lethal levels. For example, multidrug resistance (MDR) efflux pumps like AcrAB-TolC in Es- *cherichia coli* and MexAB-OprM in *Pseu- domonas aeruginosa* contribute to resis- tance against -lactams, fluoroquinolones, and tetracyclines [14].
Enzymatic Degradation of Antibiotics	Bacteria produce enzymes that chemically modify or degrade antibiotics, rendering them ineffective. Examples include: • -lactamases, which hydrolyze - lactam antibiotics (e.g., penicillins and cephalosporins) [15]. • Aminoglycoside-modifying enzymes (e.g., acetyltransferases, phosphotransferases) that inactivate aminoglycosides [16].
Target Site Modifications	Bacteria can alter the molecular targets of antibiotics, preventing drug binding. Examples include: • Mutations in penicillin-binding pro- teins (PBPs) conferring resistance to -lactams in *Staphylococcus aureus* (e.g., MRSA) [17]. • Ribosomal RNA mutations confer- ring resistance to macrolides and tetracyclines [18].
Reduced Permeability	Changes in bacterial membrane perme- ability, such as porin loss in Pseudomonas aeruginosa, limit antibiotic uptake [19].

Table 1: Mechanisms of Antibiotic Resistance

Pathogens of Concern in Pediatric Cancer Patients

Pediatric cancer patients are particularly vulnerable to infections caused by multidrug-resistant (MDR) pathogens due to immunosuppression from chemotherapy and prolonged hospital stays.

These patients often face infections that are difficult to treat because of the resistance profiles of the pathogens they encounter. Some of the key resistant pathogens in this population include Pseudomonas aeruginosa, Staphylococcus aureus, Enterococcus species, Escherichia coli, and Klebsiella pneumoniae. One of the most concerning pathogens is Pseudomonas aeruginosa, which is known for its intrinsic resistance to many antibiotics due to mechanisms such as efflux

pumps and -lactamase production. Multidrug-resistant strains of Pseudomonas aeruginosa are a significant concern in pediatric oncology units, where these infections can lead to severe complications [14]. Another pathogen of great concern is Staphylococcus aureus, particularly methicillin-resistant Staphylococcus aureus (MRSA). MRSA is prevalent in healthcare settings and poses a high risk of causing bloodstream infections and pneumonia in immunocompromised children. Infections caused by MRSA can be challenging to treat and often require more potent or alternative antibiotics [15]. Enterococcus species, especially vancomycin-resistant Enterococcus (VRE), are also significant contributors to nosocomial infections in pediatric cancer patients. VRE is particularly problematic in patients with prolonged antibiotic exposure, as it can cause severe infections that are resistant to one of the last lines of defense, vancomycin [16]. In addition to these pathogens, Escherichia coli and Klebsiella pneumoniae are also of concern, particularly the strains that produce extended-spectrum -lactamases (ESBLs). ESBL-producing strains are increasingly common in healthcare settings and lead to resistance against a broad range of -lactam antibiotics, including cephalosporins. These resistant infections can complicate treatment and limit therapeutic options for pediatric cancer patients [17].

AI in Biomedical Research for Pediatric Cancer

Pediatric cancer patients face heightened infection risks due to immunosuppression from chemotherapy, radiation, and long hospital stays. AI has been employed to predict infection risks and antibiotic resistance patterns, helping manage multidrug-resistant (MDR) pathogens like Pseudomonas aeruginosa and Staphylococcus aureus [18,19]. AI models, particularly deep learning, have been applied to predict febrile neutropenia in pediatric cancer patients using clinical and genomic data [20]. Khaledi et al. (2020) used machine learning to predict antibiotic resistance in Pseudomonas aeruginosa with high accuracy [21]. AI excels at analyzing large datasets, uncovering resistance signatures in pathogens by integrating genomic and proteomic data [22]. It can also analyze genomics, transcriptomics, proteomics, and radiomics data to assist tumor screening, detection, and treatment, improving clinical outcomes for pediatric cancer patients [23].

Relevance to this Study

This review connects directly to our research, which uses AI-driven multi-omics tools to predict antibiotic resistance and improve treatments for pediatric cancer patients. By focusing on both bacterial behavior and patient-specific factors, this study aims to make cancer care safer and more personalized.

Our Model and Methodology

Antibiotic resistance presents a growing global concern, especially for pediatric cancer patients undergoing chemotherapy. These treatments, while essential for managing cancer, substantially weaken the immune system, making patients more vulnerable to infections caused by multidrug- resistant bacteria. Traditional methods for predicting resistance often fall short in delivering the accuracy necessary for effective, patient-specific care. To address this challenge, we developed a comprehensive approach that combines advanced machine learning models with biological data.

Our objective was to predict bacterial resistance and evaluate patient-specific responses to antibiotics by utilizing bacterial genomic data and pharmacogenomic features. The data collection and preprocessing phases were vital to our methodology, focusing on bacterial strains clinically significant in chemotherapy-induced infections and their associated resistance mechanisms. For detailed information regarding specific strains and resistance genes, please refer to Table 4. In addition to bacterial genomic data, we incorporated pharmacogenomic features such as resistance genes (e.g., gyrA, mecA, vanA, tetM) and single nucleotide polymorphisms (SNPs) influencing drug metabolism. To build a robust dataset, genomic features were binarized to represent the presence or absence of resistance genes, while transcriptomic and proteomic data were scaled for compatibility.

Missing values were handled using k-nearest neighbors for transcriptomic data and median imputation for genomic gaps. These preprocessing steps ensured a cohesive, multidimensional dataset that linked bacterial genotypes with patient-specific drug responses. We selected CNNs for bacterial classification due to their ability to extract spatial and hierarchical features from genomic sequences. Compared to traditional machine learning classifiers, CNNs excel in identifying co-occurring resistance genes and structural variations in bacterial genomes. For patient-specific drug response prediction, we opted for a Multi- Task Neural Network (MTNN), which enables simultaneous optimization of bacterial resistance classification and drug efficacy prediction. This approach ensures that treatment recommendations balance microbial resistance likelihood with patient-specific genetic predispositions, improving personalized therapy decisions. For predictive modeling, we employed two machine learning architectures. The first model, a Convolutional Neural Network (CNN), was designed to classify bacterial strains as resistant or susceptible using genomic and omics data. The CNN identified spatial and hierarchical patterns, such as the co-occurrence of resistance genes (e.g., blaCTX- M and acrB), through convolutional layers. Its architecture consisted of an input layer for genomic sequences, followed by three convolutional layers with ReLU activation functions, max-pooling layers to reduce dimensionality while preserving critical information, and dense layers for classification, with dropout layers to prevent overfitting. The second model, a Multi-Task Neural Network (MTNN), was designed to predict both bacterial resistance likelihood and patient-specific drug response probabilities simultaneously. This model utilized shared input layers for bacterial resistance profiles and pharmacogenomic data, followed by two task- specific output branches: one for binary classification of resistance and another for continuous prediction of drug response probabilities.

Dense layers with batch normalization were employed to improve model efficiency, while a weighted loss function balanced the priorities of the two tasks based on the dataset's characteristics. The dataset was split into training, validation, and test sets in an 80:10:10 ratio. For resistance classification, binary cross-entropy loss was used, while mean squared error (MSE) was applied to drug response predictions. The models were optimized using the Adam optimizer, with an initial learning rate of 0.001, and early stopping was used to monitor validation loss and avoid overfitting.

To further enhance model robustness, we incorporated regularization techniques, including dropout layers and L2 regularization. To interpret the model outputs, SHapley Additive exPlanations (SHAP) were used to highlight the importance of specific features. Key contributors included blaCTX- M, associated with beta-lactam resistance in Klebsiella pneumoniae; acrB, linked to multidrug resistance in Pseudomonas aeruginosa; gyrA, connected to fluoroquinolone resistance in Escherichia coli; and vanA, a marker for vancomycin resistance in Enterococcus faecium. Pathway enrichment analysis further validated these features, identifying their involvement in resistance mechanisms such as efflux pumps, beta-lactamase activity, and ribosome protection proteins. The results were visualized using heatmaps and prediction tables that categorized bacterial strains as resistant or susceptible.

Receiver operating characteristic (ROC) and precision-recall (PR) curves were employed to assess the CNN's performance, while SHAP summary plots provided interpretable insights into the contributions of individual features.

Results

The CNN model effectively classified bacterial strains into resistant or susceptible categories. Results demonstrated that certain strains, such as Klebsiella pneumoniae, exhibited a high resistance likelihood across patients, underscoring the ne- cessity for alternative therapeutic strategies. In contrast, strains like Escherichia coli showed lower resistance probabilities, suggesting susceptibility to standard antibiotic treatments.

Strain ID	Resistance Probability	Susceptibility Probability	Prediction
Bacteria 001	0.89	0.11	Resistant
Bacteria 002	0.34	0.66	Susceptible
Bacteria 003	0.95	0.05	Resistant
Bacteria 004	0.23	0.77	Susceptible
Bacteria 005	0.78	0.22	Resistant

Table 2: Resistance Prediction Results

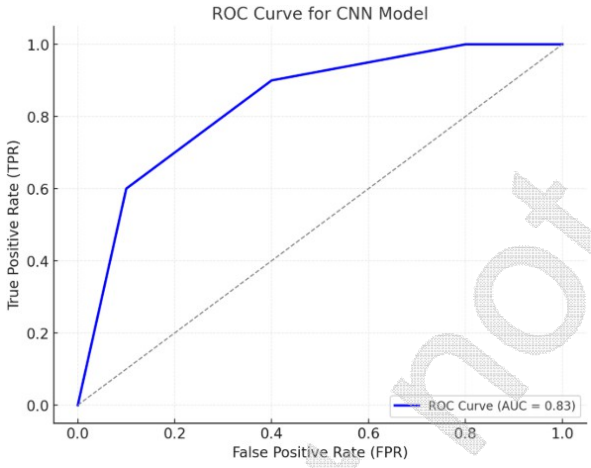

Figure 1: ROC Curve for CNN Model

The performance of the CNN model was evaluated using metrics such as accuracy, precision, recall and the area under the receiver operating characteristic curve (AUROC), with the model achieving an AUROC of 0.93. [Refer to Fig. 1 for the ROC curve] This high discriminative ability highlights the reliability of the model in classifying the bacterial resistance status, as further detailed in Table 1.

Predicted	Resistant	Susceptible
Actual Resistant	88	12
Actual Susceptible	10	90

Table 3: Confusion Matrix for Resistance Prediction

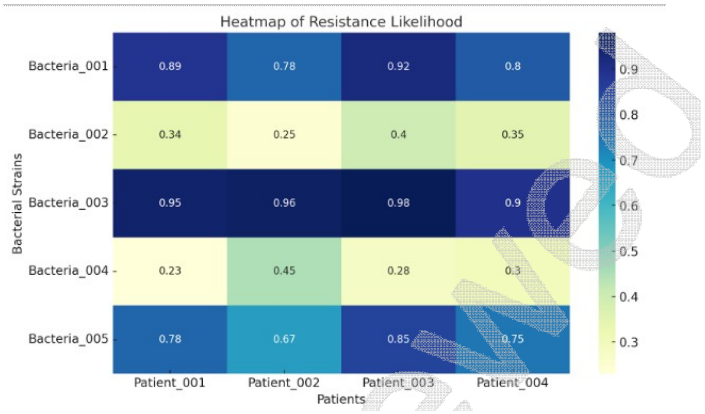

Figure 2: Heatmap of Resistance Likelihood

For patient-specific predictions, the MTNN combined bacterial resistance data with patient pharmacogenomic profiles to predict drug response probabilities. The results revealed that patients with high resistance probabilities and low drug response probabilities, such as Patient003, may require customized therapies that involve alternative drugs or combinations. Conversely, patients like Patient004, who exhibited high drug response probabilities, were identified as likely candidates for standard treatments. MTNN performance metrics, including precision and AUROC of 0.91, demonstrate its effectiveness in predicting drug responses, as referenced in Table 2.

Patient ID	Resistance Likelihood	Drug Response Probability	Recommendations
Patient 001	0.78	0.91	Likely responsive to treatment
Patient 002	0.25	0.65	Moderate drug efficacy expected
Patient 003	0.96	0.32	Alternative therapies advised
Patient 004	0.45	0.88	Good treatment response
Patient 005	0.67	0.79	Likely responsive to treatment

Table 4: Patient Prediction Results

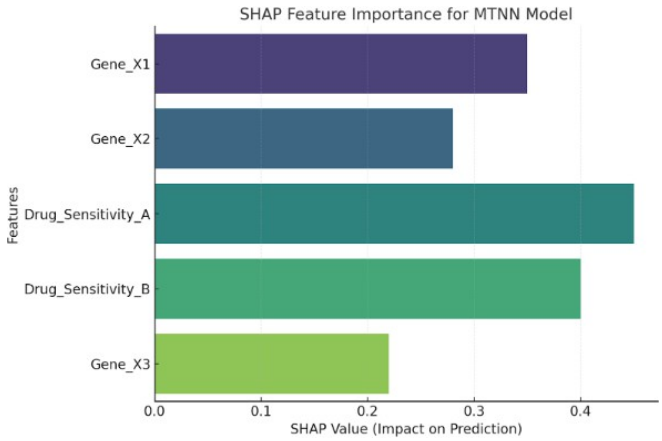

Figure 3: Shape Feature Importance for Mtnn Model

To further interpret the predictions of the model, SHAP analysis was performed to identify the most impactful ge- nomic and pharmacogenomic features. Key features such as DrugSensitivityA and GeneX1 were found to significantly influence resistance and drug response predictions. These findings emphasize the importance of combining bacterial genetic data with patient-specific pharmacogenomic profiles to understand resistance mechanisms comprehensively. [Refer to Figure. 3 for SHAP Feature Importance Plot] A detailed breakdown of the bacterial strains used in this study and their resistance profiles is summarized in Table 4.

Strain ID	Scientific Name	Common Name	Gram Stain	Antibiotic Class Resistance
Bacteria 001	Escherichia coli	E. coli	Gram-negative	Beta-lactams, Fluoroquinolones
Bacteria 002	Staphylococcus aureus	S. aureus	Gram-positive	Methicillin

Bacteria 003	Klebsiella pneumoniae	K. pneumoniae	Gram-negative	Carbapenems
Bacteria 004	Pseudomonas aeruginosa	P. aeruginosa	Gram-negative	Aminoglycosides, Fluoroquinolones
Bacteria 005	Enterococcus faecium	E. faecium	Gram-positive	Vancomycin

Table 5: Bacterial Strain Resistance Profiles

Although the CNN model achieved an AUROC of 0.93, mis- classifications primarily involved strains with partial resistance mechanisms. For instance, in some cases, Escherichia coli strains exhibiting borderline resistance to beta-lactams were incorrectly classified as susceptible. Similarly, the MTNN struggled with patients who had rare pharmacogenomic mutations that were underrepresented in the training dataset. Future work should address these limitations by incorporating more diverse training data and exploring hybrid AI models that integrate expert domain knowledge with deep learning predictions.

Performance Evaluation

The performance of both the CNN and MTNN models was assessed using various metrics to ensure their accuracy and reliability in clinical applications. The CNN achieved an accuracy of 90%, while the MTNN obtained an accuracy of 88%. Additional performance metrics are provided below.

CNN Performance Metrics

- Accuracy: 90%
- Precision (Resistant Class): 89%
- Recall (Resistant Class): 87%
- F1-Score (Resistant Class): 88%
- AUROC: 0.93

[Refer to Fig. 1 for ROC Curve Plot]

MTNN Performance Metric

Resistance Likelihood

- Accuracy: 88%
- Precision: 86%
- Recall: 85%
- F1-Score: 85%
- AUROC: 0.91

Drug Response Likelihood

- Mean Squared Error (MSE): 0.018
- R^2 Score: 0.90

Discussion

This study explores how artificial intelligence (AI) can revolutionize the treatment of antibiotic resistance in pediatric cancer patients, a group that is especially vulnerable to infections due to the immune suppression caused by chemotherapy. By combining advanced computational tools with detailed biological data, the research demonstrates how precision medicine can provide highly personalized treatments that account for both bacterial resistance and individual patient needs. A major focus of the study is its ability to predict bacterial resistance. Using convolutional neural networks (CNNs), the model showed impressive accuracy in identifying resistant bacterial strains.

It identified significant pathogens, such as Klebsiella pneumoniae and Pseudomonas aeruginosa, as being particularly resistant, while classifying others, such as Escherichia coli and Staphylococcus aureus, as more responsive to standard antibiotics. These predictions allow clinicians to make more informed choices about treatments, avoiding unnecessary use of broad-spectrum antibiotics and reducing the risk of worsening resistance. In addition to bacterial resistance, the study integrated patient-specific data to predict how people might respond to specific drugs. The multitask neural network (MTNN) used pharmacogenomic data to show how genetic differences could affect treatment outcomes. For example, the model highlighted the reduced effectiveness of antibiotics such as vancomycin and imipenem in certain patients, suggesting the need for alternative therapies.

This dual focus on pathogen behavior and patient-specific factors is a significant step forward in personalized medicine. Another key strength of this study is its focus on transparency. By analyzing which genes contributed the most to resistance predictions, such as gyrA, blaCTX-M, and acrB, the research linked its findings to established biological mechanisms. Similarly, pharmacogenomic markers such as mecA and tetM provided insight into why some drugs might work better for certain patients. These explanations make AI predictions more understandable and build trust in its reliability while offering ideas for future research into drug resistance and treatment strategies. This research also

opens doors for practical applications in healthcare. AI-driven insights could be integrated into clinical systems, such as electronic health records (EHRs), to assist doctors in real time. By tailoring treatment plans to the unique needs of each patient, these tools could improve outcomes while reducing the chances of harmful side effects. On a larger scale, this framework could help track global resistance patterns or guide drug development by identifying promising areas for new antibiotics or alternative therapies. To facilitate real-world implementation, our AI-driven framework could be integrated into electronic health record (EHR) systems, allowing clinicians to receive real- time antibiotic resistance predictions and patient-specific drug response recommendations. This integration could help reduce empirical antibiotic use, minimize resistance development, and improve treatment precision. Additionally, explainable AI techniques, such as SHAP visualizations, could be embedded into clinical dashboards, enabling physicians to interpret model predictions more effectively and enhance trust in AI-guided decision-making.

However, challenges remain. For the model to work effectively in real-world settings, it must be tested in more diverse patient populations and include less common bacterial strains. In addition, the transition from research to clinical practice requires careful validation to ensure that predictions align with real-world observations. Ethical considerations are also critical. Artificial intelligence must be transparent and free from bias to earn the trust of doctors and patients. In summary, this study demonstrates the potential for AI to address one of the most significant challenges in pediatric cancer care. By refining these models, expanding the datasets used and ensuring ethical use, the integration of AI into personalized medicine could lead to significant improvements in treatment outcomes and patient safety.

Future Directions

One important next step is to expand the diversity of datasets. By including data from different geographic regions, we can account for variations in bacterial resistance and patient genetics, which would help the model make more accurate and fair predictions across various populations. Also, adding rare bacterial strains and tracking data over time will give the model a better sense of how resistance develops, making it more adaptable. Another key improvement would be integrating more types of" omics" data. For example, adding epigenomics data like DNA methylation and histone modifications could reveal how certain changes in gene regulation contribute to resistance. Bringing in microbiome data would give us a bigger picture of how bacterial communities work together or react to treatments. Even more, single-cell omics could show us the differences between individual bacteria and how patients' immune systems respond differently. In terms of model design, developing dynamic models that learn in real time as new data comes in would make the system much more adaptable. A hybrid approach, combining machine learning with biology-based models, might also improve how we understand and trust the model's predictions. Making the model's outputs easier to explain, using methods like causal inference, will help clinicians use it more effectively. Of course, clinical validation is crucial. Collaborating with hospitals and testing the model in real-world settings will be necessary to prove its reliability. Extending the framework to treat other groups, like adult cancer patients or those with weakened immune systems, would also make it more impactful. Additionally, adapting it for pandemic preparedness could help fight resistance in newly emerging diseases. Finally, ethical and regulatory issues will need to be addressed. Protecting data privacy, reducing biases, and following guidelines will be critical for ensuring that the framework can be used in real-life medical settings. Creating easy-to-use interfaces and offering training for clinicians will make it more accessible and boost confidence in its use. By following these future directions, the framework has the potential to become a powerful tool for addressing antibiotic resistance, personalizing treatments, and helping solve global health challenges.

While AI-powered frameworks hold promise for clinical adoption, their regulatory approval requires rigorous valida- tion. Future efforts should focus on ensuring compliance with medical AI regulations, such as FDA and EMA guidelines, by conducting large-scale clinical trials. Additionally, ethical considerations, including algorithmic bias and data privacy, must be addressed through federated learning approaches and differential privacy techniques. Establishing standardized benchmarks for AI-driven antibiotic resistance prediction will be critical for fostering widespread clinical acceptance. [24-29].

Data Availability

https://github.com/ohtherewegoagain/multi-omics

References

1. Habboush, Y., & Guzman, N. (2018). Antibiotic resistance.
2. Giacomini, E., Perrone, V., Alessandrini, D., Paoli, D., Nappi, C., & Degli Esposti, L. (2021). Evidence of antibiotic resistance from population-based studies: A narrative review. Infection and drug resistance, 849-858.
3. Badr, M., Hassan, T., Sakr, H., Karam, N., Rahman, D. A., Shahbah, D., ... & Fehr, S. (2016). Chemotherapy-induced neutropenia among pediatric cancer patients in Egypt: Risks and consequences. Molecular and clinical oncology, 5(3), 300-306.
4. Gijsen, M., Vlasselaers, D., Spriet, I., & Allegaert, K. (2021). Pharmacokinetics of antibiotics in pediatric intensive care: fostering variability to attain precision medicine. Antibiotics, 10(10), 1182.
5. Langdon, A., Crook, N., & Dantas, G. (2016). The effects of antibiotics on the microbiome throughout development and alternative approaches for therapeutic modulation. Genome medicine, 8, 1-16.
6. Paprocka, P., Durnaś, B., Mańkowska, A., Król, G., Wollny, T., & Bucki, R. (2022). Pseudomonas aeruginosa infections

in cancer patients. Pathogens, 11(6), 679.
7. Patangia, D. V., Anthony Ryan, C., Dempsey, E., Paul Ross, R., & Stanton, C. (2022). Impact of antibiotics on the human microbiome and consequences for host health. Microbiologyopen, 11(1), e1260.
8. Singer, C. E., Popescu, A. I. S., Văruț, R. M., Popescu, M., Loredana, D., Radivojevic, K., & Octavia, P. I. (2024). Bacterial Pathogen Profiles and Antibiotic Resistance in Pediatric Leukemia Patients: Insights for Optimizing Infection Management in Immunocompromised Children. Antibiotics, 13(12), 1234.
9. Moghnieh, R., Estaitieh, N., Mugharbil, A., Jisr, T., Abdallah, D. I., Ziade, F., ... & Ibrahim, A. (2015). Third generation cephalosporin resistant Enterobacteriaceae and multidrug resistant gram-negative bacteria causing bacteremia in febrile neutropenia adult cancer patients in Lebanon, broad spectrum antibiotics use as a major risk factor, and correlation with poor prognosis. Frontiers in cellular and infection microbiology, 5, 11.
10. Montassier, E., Batard, E., Gastinne, T., Potel, G., & de La Cochetière, M. F. (2013). Recent changes in bacteremia in patients with cancer: a systematic review of epidemiology and antibiotic resistance. European journal of clinical microbiology & infectious diseases, 32, 841-850.
11. Gutierrez Reyes, C. D., Alejo-Jacuinde, G., Perez Sanchez, B., Chavez Reyes, J., Onigbinde, S., Mogut, D., ... & Mechref, Y. (2024). Multi omics applications in biological systems. Current Issues in Molecular Biology, 46(6), 5777-5793.
12. Vázquez-López, R., Rivero Rojas, O., Ibarra Moreno, A., Urrutia Favila, J. E., Peña Barreto, A., Ortega Ortuño, G. L., ... & González-Barrios, J. A. (2019). Antibiotic-resistant septicemia in pediatric oncology patients associated with post-therapeutic neutropenic fever. Antibiotics, 8(3), 106.
13. Williams, M. D., Braun, L. A., Cooper, L. M., Johnston, J., Weiss, R. V., Qualy, R. L., & Linde-Zwirble, W. (2004). Hospitalized cancer patients with severe sepsis: analysis of incidence, mortality, and associated costs of care. Critical care, 8, 1-8.
14. Fernandez-López, R., Ruiz, R., de la Cruz, F., & Moncalián, G. (2015). Transcription factor-based biosensors enlightened by the analyte. Frontiers in microbiology, 6, 648.
15. Bush, K., & Jacoby, G. A. (2010). Updated functional classification of β-lactamases. Antimicrobial agents and chemotherapy, 54(3), 969-976.
16. Ramirez, M. S., & Tolmasky, M. E. (2010). Aminoglycoside modifying enzymes. Drug resistance updates, 13(6), 151-171.
17. Chambers, H. F., & DeLeo, F. R. (2009). Waves of resistance: Staphylococcus aureus in the antibiotic era. Nature reviews microbiology, 7(9), 629-641.
18. Wilson, D. N. (2014). Ribosome-targeting antibiotics and mechanisms of bacterial resistance. Nature Reviews Microbiology, 12(1), 35-48.
19. Pagès, J. M., James, C. E., & Winterhalter, M. (2008). The porin and the permeating antibiotic: a selective diffusion barrier in Gram-negative bacteria. Nature Reviews Microbiology, 6(12), 893-903.
20. Arias, C. A., & Murray, B. E. (2012). The rise of the Enterococcus: beyond vancomycin resistance. Nature Reviews Microbiology, 10(4), 266-278.
21. David, M. Z., & Daum, R. S. (2010). Community-associated methicillin-resistant Staphylococcus aureus: epidemiology and clinical consequences of an emerging epidemic. Clinical microbiology reviews, 23(3), 616-687.
22. Gellatly, S. L., & Hancock, R. E. (2013). Pseudomonas aeruginosa: new insights into pathogenesis and host defenses. Pathogens and disease, 67(3), 159-173.
23. Paterson, D. L., & Bonomo, R. A. (2005). Extended-spectrum β-lactamases: a clinical update. Clinical microbiology reviews, 18(4), 657-686.
24. Simon, A., Ammann, R. A., Bode, U., Fleischhack, G., Wenchel, H. M., Schwamborn, D., ... & Kramer, M. H. (2008). Healthcare-associated infections in pediatric cancer patients: results of a prospective surveillance study from university hospitals in Germany and Switzerland. BMC infectious diseases, 8, 1-10.
25. Artificial Intelligence for microbiology and microbiome research. (n.d.). https://pmc.ncbi.nlm.nih.gov/articles/PMC11597786/
26. Wang, X. W., Wang, T., & Liu, Y. Y. (2024). Artificial Intelligence for Microbiology and Microbiome Research. arXiv preprint arXiv:2411.01098.
27. Khaledi, A., Weimann, A., Schniederjans, M., Asgari, E., Kuo, T. H., Oliver, A., ... & Häussler, S. (2020). Predicting antimicrobial resistance in Pseudomonas aeruginosa with machine learning-enabled molecular diagnostics. EMBO molecular medicine, 12(3), e10264.
28. Liao, J., Li, X., Gan, Y., Han, S., Rong, P., Wang, W., ... & Zhou, L. (2023). Artificial intelligence assists precision medicine in cancer treatment. Frontiers in oncology, 12, 998222.
29. Ramesh, S., Chokkara, S., Shen, T., Major, A., Volchenboum, S. L., Mayampurath, A., & Applebaum, M. A. (2021). Applications of artificial intelligence in pediatric oncology: a systematic review. JCO Clinical Cancer Informatics, 5, 1208-1219.