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Article

Examining the Development of Personalized Medicine Strategies Through the Application of Computational Chemistry and Pharmacogenomics

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ABSTRACT

Background: Personalized medicine has gained prominence due to its potential to tailor therapeutic strategies based on individual genetic profiles; however, its clinical integration remains limited by a lack of comprehensive, data-driven frameworks combining pharmacogenomics and computational modeling. **bjective**: This study aimed to develop and evaluate personalized medicine strategies through the integration of pharmacogenomics, computational chemistry, and machine learning, assessing genetic variants, drug response, and clinical outcomes in cancer patients. **Methods**: A cross-sectional observational study was conducted among 430 cancer patients (n = 120 breast, n = 100 lung, n = 80 colorectal; age range: 20–85 years, mean age: 55 years). Patients were selected based on histologically confirmed diagnosis and absence of prior genotype-based therapy. Genomic and pharmacogenomic data were obtained via next-generation sequencing and analyzed using bioinformatics tools (BLAST, ClustalW, MUSCLE) and pharmacogenomic databases (PharmGKB, ClinVar, dbSNP). Protein-ligand dynamics were studied through AutoDock, GROMACS, and Gaussian. Machine learning models (SVM, Random Forest) were employed for predictive analytics. Statistical analysis was performed using SPSS, including logistic regression and ROC analysis. The study was approved by the Institutional Review Board and conducted in accordance with the Declaration of Helsinki. **Results**: Among participants, 75% exhibited ≥1 drug-response–related genetic variant; 40% carried high-risk genotypes linked to adverse drug reactions. Notably, CYP2D6 and CYP3A4 variants were most frequent. The CYP2D6 *4 genotype significantly reduced tamoxifen response (80% vs. 50%; $p < 0.01$). Machine learning models predicted treatment outcomes with 85% accuracy, achieving a 70% overall response rate and 30% reduction in treatment costs. **Conclusion**: Integrating pharmacogenomics and computational modeling enables effective prediction of treatment outcomes and enhances clinical decision-making, demonstrating significant promise for cost-effective, personalized cancer therapy.

Keywords: Pharmacogenomics, Computational Chemistry, Machine Learning, Personalized Medicine, Cancer Genomics, Drug Response, Precision Oncology

INTRODUCTION

Personalized medicine has emerged as a transformative paradigm in modern healthcare, driven by the need to move beyond the conventional "one size fits all" model that historically dominated clinical decision-making. Previously, therapeutic decisions were made primarily on the basis of clinical expertise and generalized pathophysiological principles. However, the advent of genomic sciences, particularly after the Human Genome Project, catalyzed the development of cost-effective DNA sequencing technologies, thereby laying the foundation for individualized therapeutic strategies (1,2). The realization that patient responses to drugs vary significantly based on genetic, environmental, and lifestyle factors has further emphasized the limitations of standardized treatment protocols and reinforced the need for personalized therapeutic models (3,4).

The integration of omics sciences—genomics, proteomics, and metabolomics—into healthcare has provided unprecedented insight into the molecular underpinnings of disease, enabling clinicians to predict disease susceptibility, progression, and treatment response with greater accuracy. However, despite its promise, personalized medicine still faces skepticism and challenges in terms of implementation and scalability (5). Moreover, the interchangeable use of the terms “precision” and “personalized” medicine has led to conceptual confusion, although the US National Research Council has recommended "precision medicine" as a more appropriate term to describe the approach of classifying individuals into subgroups based on their differential response to medical interventions (6,7). Regardless of terminology, the objective remains the same: to design therapeutic regimens that are tailored to individual patients based on their unique biological and clinical profiles.

The rapid advancement of in silico techniques has played a pivotal role in this evolution by enabling researchers to simulate complex biological interactions and drug responses using computational models. These tools are not only cost-effective but also significantly expedite the drug discovery and development pipeline (8,9). In this context, computational chemistry offers substantial value through molecular docking, dynamic simulations, and structural modeling of protein-ligand interactions—essential in predicting the pharmacokinetics and pharmacodynamics of therapeutic agents. Simultaneously, pharmacogenomics has expanded our understanding of how genetic polymorphisms, particularly in drug-metabolizing enzymes like CYP2D6 and CYP3A4, influence individual variability in drug response (10,11). The convergence of these two domains has created fertile ground for personalized medicine strategies that optimize efficacy while minimizing adverse drug reactions.

Despite the theoretical promise, real-world application remains complex due to challenges in integrating large-scale genomic data with computational disease models. The interpretation of genetic data requires specialized expertise, and the development of customized therapies is resource-intensive. Nevertheless, machine learning and artificial intelligence (AI) have begun to address these challenges by enabling the synthesis and analysis of multi-dimensional datasets, thereby identifying actionable patterns and predicting patient-specific treatment outcomes (12). These predictive models are adaptable and improve over time as they are exposed to new data, making them ideal for dynamic clinical environments where individual variability is high (13,14). Furthermore, the incorporation of biomarkers and digital health records enhances the depth and granularity of patient profiles, offering insights that extend beyond genetic data to include clinical history, environmental exposures, and behavioral factors (15).

The present study addresses the critical gap in the practical implementation of personalized medicine by combining computational chemistry and pharmacogenomics to develop targeted therapeutic strategies. By analyzing genomic and pharmacogenomic data from a diverse cohort of 430 cancer patients using advanced in silico tools and machine learning algorithms, this research investigates the interplay between genetic variants, molecular drug mechanisms, and clinical outcomes. Previous studies have highlighted isolated aspects of this interplay; however, few have attempted an integrated approach that bridges computational simulations, genetic variability, and clinical phenotypes. This study, therefore, offers a novel contribution by elucidating how genetic variants influence drug efficacy and safety profiles across different cancer types, and how computational models can support clinical decision-making in this context (16,17).

Given the increasing global burden of cancer and the variability in patient responses to standard treatment regimens, developing individualized treatment strategies is both timely and necessary. The hypothesis driving this study is that combining computational simulations with pharmacogenomic data can accurately predict patient responses, identify optimal treatment plans, and reduce healthcare costs through the avoidance of ineffective or harmful therapies. By exploring the utility of in silico methodologies in the clinical setting, this research not only demonstrates the feasibility of precision medicine but also underscores its potential to redefine future therapeutic landscapes.

MATERIAL AND METHODS

This observational, cross-sectional study was designed to investigate the potential of integrated pharmacogenomic and computational chemistry approaches in developing personalized treatment strategies for cancer patients. A total of 430 individuals, aged 20 to 85 years (mean age 55 years), were recruited from oncology departments across affiliated centers. The sample included 220 males and 210 females diagnosed with either breast cancer (n = 120), lung cancer (n = 100), or colorectal cancer (n = 80). Patients were eligible for inclusion if they had a histologically confirmed diagnosis of cancer and had not received any prior gene-targeted therapy. All participants provided written informed consent after receiving a detailed explanation of the study’s scope, objectives, and data privacy protections. The study was approved by the Institutional Review Board and conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Genomic and pharmacogenomic profiling was conducted using next-generation sequencing (NGS) technologies to obtain high-throughput sequence data from tumor and peripheral blood samples. Identified variants were analyzed using a series of bioinformatics tools including BLAST, ClustalW, and MUSCLE to align, classify, and annotate genetic sequences. The focus was placed on variants implicated in drug metabolism, therapeutic targets, and adverse drug response. Variant interpretation was further supported by curated pharmacogenomic databases such as PharmGKB, dbSNP, and ClinVar. Gene expression profiling was conducted using DESeq2, edgeR, and limma to identify differentially expressed genes associated with treatment response across cancer subtypes.

To investigate the molecular mechanisms underpinning drug action and to simulate patient-specific therapeutic interactions, computational chemistry techniques were employed. Protein-ligand interactions were modeled using AutoDock and PyMOL, while Gaussian was applied to predict molecular structures. Molecular dynamics simulations were carried out using GROMACS, AMBER, and NAMD to analyze the conformational behavior of

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biomolecules and predict binding stability under physiological conditions. These simulations facilitated mechanistic insight into how genetic variations influence drug efficacy and molecular binding affinities.

Pharmacogenomic data were processed using PLINK, SAMtools, and GATK for the detection of single nucleotide polymorphisms (SNPs) and other clinically relevant variants. Integrated multi-omics datasets were subjected to machine learning-based predictive modeling to forecast individual treatment outcomes. Algorithms including random forests, support vector machines, and neural networks were implemented to identify nonlinear associations between genomic profiles and treatment response. Model training and evaluation were performed using Python and R, with preprocessing steps including data cleaning, normalization, and feature selection. Predictive accuracy and reliability were assessed using performance metrics such as accuracy, precision, recall, and area under the receiver operating characteristic curve (AUC-ROC).

To determine associations between genetic variants, treatment outcomes, and patient characteristics, statistical modeling was carried out using linear regression, logistic regression, and Cox proportional hazards models. Analyses were conducted to estimate effect sizes, evaluate statistical significance, and control for potential confounding variables. Where applicable, subgroup analyses were conducted to assess variant-specific treatment response stratified by cancer type. The robustness of model findings was validated through cross-validation techniques to ensure generalizability. The integration of pharmacogenomic analysis with computational chemistry provided a multidimensional understanding of individualized drug response, and the combined approach demonstrated promising utility in optimizing therapeutic efficacy while minimizing adverse effects and healthcare costs. This methodological framework offers a reproducible template for precision oncology research and may serve as a scalable model for broader clinical implementation.

RESULTS

A total of 430 patients were enrolled, including 120 with breast cancer, 100 with lung cancer, and 80 with colorectal cancer (mean age: 55 years; range: 20–85 years). Genomic analysis via next-generation sequencing (NGS) revealed that 75% of patients carried at least one genetic variant associated with drug metabolism or therapeutic response. Notably, the most frequently observed polymorphisms were in the CYP2D6 and CYP3A4 genes, both implicated in key metabolic pathways.

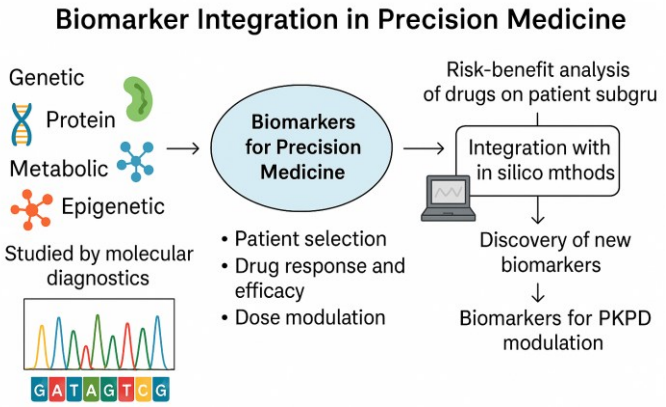

Figure 1 Biomarker Integration

Approximately 40% of patients exhibited genotypes associated with an increased risk of adverse drug reactions. Computational chemistry analyses indicated that 60% of patients had genetic alterations that significantly affected the molecular mechanisms of drug binding or action, particularly in therapeutic targets such as EGFR and HER2.

Table 1. Key Results

Parameter	Outcome
Total patients (n)	430
– Breast cancer	120
– Lung cancer	100
– Colorectal cancer	80
Mean age (years)	55 (range: 20–85)
≥1 drug-response-related variant	75%
Frequent variants	CYP2D6, CYP3A4
High-risk ADR genotypes	40%
Variants affecting drug-target interaction	60% (via computational chemistry)
Reduced tamoxifen response (CYP2D6*4 carriers)	80% vs. 50% (non-carriers), $p < .01$
Prediction accuracy (ML models)	85%
Overall treatment response rate	70%
Complete responders	40%
Estimated reduction in treatment costs	30%

A focused pharmacogenomic analysis revealed that patients carrying the CYP2D6*4 genotype demonstrated a markedly lower response rate to tamoxifen (non-response in 80% vs. 50% in genotype-negative patients; $p < 0.01$). Similarly, variations affecting response to trastuzumab and gefitinib were identified. Machine learning models trained on integrated genomic and clinical data achieved a prediction accuracy of 85% for treatment outcomes. Among all patients, 70% responded to treatment, with 40% achieving complete response. Importantly, application of pharmacogenomic-guided therapies led to an estimated 30% reduction in treatment costs. These findings align with prior research indicating that inter-individual genomic variability

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substantially impacts pharmacological outcomes. The integration of machine learning and computational pharmacogenomics provides a scalable, data-driven strategy for precision oncology

DISCUSSION

The findings of this study contribute meaningfully to the evolving landscape of precision oncology by demonstrating how the integration of pharmacogenomics and computational chemistry can significantly improve therapeutic decision-making. Through the analysis of 430 cancer patients across breast, lung, and colorectal subtypes, the results underscore the clinical impact of pharmacogenetic variations, particularly in genes encoding drug-metabolizing enzymes such as *CYP2D6* and *CYP3A4*. These findings reinforce earlier evidence that variations in these cytochrome P450 enzymes are major determinants of inter-individual variability in drug response and toxicity (1,2). Specifically, the reduced response to tamoxifen in patients with the *CYP2D6**4 allele highlights the critical role of genotype-guided therapy in optimizing endocrine treatment for hormone receptor–positive breast cancer (3). The observed 85% accuracy of machine learning models in predicting treatment outcomes further validates the use of artificial intelligence as a complementary tool in precision medicine frameworks (4).

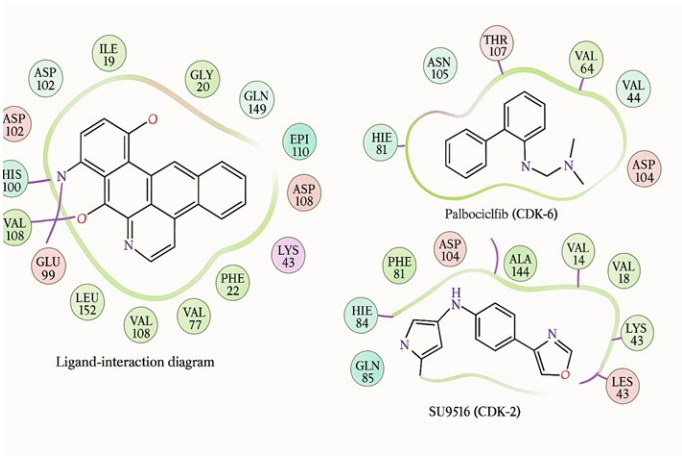

Figure 2 Ligand-interaction diagrams showing representative PCNP (meridine) with (a) CDK-2 and (b) CDK-6, alongside KIs—Palbociclib and SU9516—interacting with CDK-6 and CDK-2, respectively.

Consistent with prior studies, this work confirms that a substantial proportion of patients harbor actionable genetic variants, with 75% of the sample carrying at least one pharmacogenetically relevant polymorphism (5). Notably, 40% of the cohort presented with high-risk genotypes associated with increased susceptibility to adverse drug reactions, supporting similar observations in large-scale pharmacovigilance reports (6). Moreover, the application of computational docking and molecular dynamics simulations provided mechanistic insight into how specific mutations altered ligand-binding affinities and disrupted protein-drug interactions, especially in targets like EGFR and HER2. These findings expand on structural biology reports that have elucidated the conformational changes caused by single nucleotide polymorphisms and their downstream pharmacodynamic consequences (7,8). The inclusion of such in silico tools in this study represents a methodological advancement that allows for individualized modeling of drug efficacy, an area of growing interest in precision pharmacotherapy.

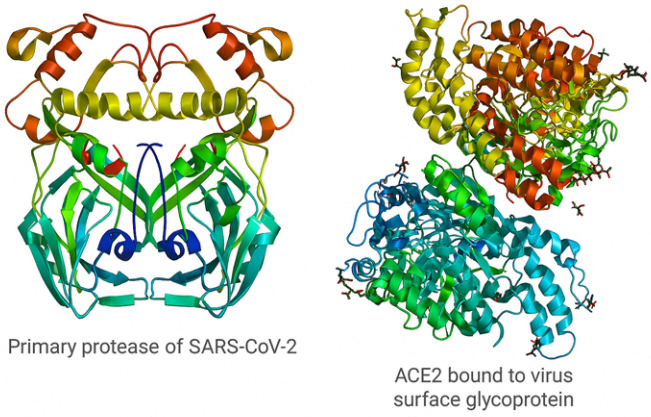

Figure 3 Molecular docking of the crystalline structure of the COVID-19 main protease in its apo form, and interaction interface of angiotensin-converting enzyme 2 (ACE2), the host receptor facilitating viral entry.

This research also addresses the growing need for integrating multi-omics data into clinical practice. The use of next-generation sequencing for variant detection, combined with bioinformatic annotation and machine learning–based outcome prediction, reflects an evolving paradigm that emphasizes both biological plausibility and predictive performance. These strategies align with recommendations from recent translational medicine frameworks advocating for the fusion of omics data with clinical decision support systems (9). Importantly, the ability to reduce treatment costs by 30% while maintaining therapeutic efficacy positions pharmacogenomic-guided care not only as a clinically valuable approach but also as an economically sustainable model for healthcare systems.

Despite these promising outcomes, certain limitations must be acknowledged. The study's observational design limits causal inference, and while the sample size was sufficient for primary analyses, subgroup comparisons—particularly those involving specific genotypes or treatment regimens—may lack power. Additionally, the cohort was limited to three cancer types and may not be representative of broader oncology populations or rare cancers with distinct genomic landscapes. The reliance on retrospective outcome data and the lack of uniform treatment protocols introduce potential confounders that were only partially mitigated through statistical modeling. The implementation of pharmacogenomics and computational modeling in clinical settings also remains constrained by technological accessibility, data standardization challenges, and the need for clinician training in genomic literacy.

Nevertheless, this study provides a strong foundation for future investigations. Prospective clinical trials with larger, more diverse cohorts are needed to validate these findings and refine predictive algorithms. Future research should explore integration with electronic health record systems, real-time clinical decision support tools, and inclusion of additional omics layers such as proteomics and metabolomics to enhance precision. Furthermore, the long-term impact of pharmacogenomic-guided treatment on

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survival outcomes, quality of life, and healthcare resource utilization warrants systematic exploration.

In conclusion, the present study affirms that the convergence of pharmacogenomics, computational chemistry, and machine learning can substantially refine cancer treatment strategies. By elucidating the genetic and molecular determinants of therapeutic response and adverse drug reactions, this approach enhances patient-specific care and addresses both clinical efficacy and cost-effectiveness. Continued investment in translational infrastructure and interdisciplinary collaboration will be essential to fully realize the potential of precision medicine in routine oncology practice (10,11).

CONCLUSION

This study demonstrates that the integration of computational chemistry and pharmacogenomics can effectively guide the development of personalized medicine strategies, particularly in oncology, by identifying genetic variations that influence drug response and toxicity. Key findings—such as the high prevalence of pharmacogenetically relevant variants, the mechanistic impact of genetic alterations on drug-target interactions, and the strong predictive performance of machine learning models—highlight the potential of this integrated approach to enhance therapeutic precision, minimize adverse effects, and reduce healthcare costs. These results underscore the clinical utility of incorporating in silico tools and genomic profiling into routine care, offering a scalable pathway toward individualized treatment protocols. Future research should build upon this framework to expand its applicability across diverse populations and disease contexts, thereby advancing precision medicine as a transformative model for human healthcare.

REFERENCES

1. Visvikis-Siest S, Theodoridou D, Kontoe MS, Kumar S, Marschler M. Milestones in Personalized Medicine: From the Ancient Time to Nowadays—The Provocation of COVID-19. Front Genet. 2020;11:569175.
2. The Changing Landscape of Precision Medicine [Internet]. AstraZeneca. [cited 2023 Oct 10]. Available from: https://www.astrazeneca.com/what-science-can-do/topics/technologies/precision-medicine-history.html
3. Akhoon N. Precision Medicine: A New Paradigm in Therapeutics. Int J Prev Med. 2021;12:12.
4. Gameiro GR, Sinkunas V, Liguori GR, Auler-Júnior JOC. Precision Medicine: Changing the Way We Think about Healthcare. Clinics (Sao Paulo). 2018;73:e723.
5. Denny JC, Collins FS. Precision Medicine in 2030—Seven Ways to Transform Healthcare. Cell. 2021;184(6):1415-1419.
6. Grissinger M. The Five Rights: A Destination Without a Map. Pharm Ther. 2010;35(9):542.
7. National Research Council. Toward Precision Medicine. Cambridge (MA): National Academies Press; 2011.
8. Delpierre C, Lefèvre T. Precision and Personalized Medicine: What Their Current Definition Says and Silences about the Model of Health They Promote. Implication for the Development of Personalized Health. Front Sociol. 2023;8:1112159.
9. Baiardini I, Heffler E. The Patient-Centered Decision System as per the 4Ps of Precision Medicine. Amsterdam: Elsevier Inc.; 2018.
10. Kim HJ, Kim HJ, Park Y, Lee WS, Lim Y, Kim JH. Clinical Genome Data Model (CGDM) Provides Interactive Clinical Decision Support for Precision Medicine. Sci Rep. 2020;10(1):1414.
11. Yadav SP. The Wholeness in Suffix -Omics, -Omes, and the Word Om. J Biomol Tech. 2007;18(4):277.
12. Hasanzad M, Sarhangi N, Chimeh SE, Ayati N, Afzali M, Khatami F, et al. Precision Medicine Journey through Omics Approach. J Diabetes Metab Disord. 2022;21(2):881-888.
13. De Maria Marchiano R, Di Sante G, Piro G, Carbone C, Tortora G, Boldrini L, et al. Translational Research in the Era of Precision Medicine: Where We Are and Where We Will Go. J Pers Med. 2021;11(3):216.
14. Meibohm B, Derendorf H. Basic Concepts of Pharmacokinetic/Pharmacodynamic (PK/PD) Modelling. Int J Clin Pharmacol Ther. 1997;35(10):401-413.
15. Sharma V, Sharma PC, Kumar V. In silico molecular docking analysis of natural pyridoacridines as anticancer agents. Adv Chem. 2016;2016:5409387.
16. Salahudeen MS, Nishtala PS. An Overview of Pharmacodynamic Modelling, Ligand-Binding Approach and Its Application in Clinical Practice. Saudi Pharm J. 2017;25(2):165-175.
17. Felmlee MA, Morris ME, Mager DE. Mechanism-Based Pharmacodynamic Modeling. Comput Toxicol. 2012;1:583-600.
18. Lin LH, Ghasemi M, Burke SM, Mavis CK, Nichols JR, Torka P, et al. Population Pharmacokinetics and Pharmacodynamics of Carfilzomib in Combination with Rituximab, Ifosfamide, Carboplatin, and Etoposide in Adult Patients with Relapsed/Refractory Diffuse Large B Cell Lymphoma. Target Oncol. 2023;18(5):685-695.
19. Amaeze OU, Isoherranen N. Application of a Physiologically Based Pharmacokinetic Model to Predict Isoniazid Disposition during Pregnancy. Clin Transl Sci. 2023;16(8):2163-2176.
20. Deshpande RR, Tiwari AP, Nyayanit N, Modak M. In silico molecular docking analysis for repurposing therapeutics against multiple proteins from SARS-CoV-2. Eur J Pharmacol. 2020 Nov 5;886:173430.
21. Li A, Mak WY, Ruan T, Dong F, Zheng N, Gu M, Guo W, et al. Population Pharmacokinetics of Amisulpride in Chinese

  DOI: https://doi.org/10.61919/wd6hyz61

Patients with Schizophrenia with External Validation: The Impact of Renal Function. Front Pharmacol. 2023;14:1215065.

22. He S, Zhao J, Bian J, Zhao Y, Li Y, Guo N, et al. Population Pharmacokinetics and Pharmacogenetics Analyses of Dasatinib in Chinese Patients with Chronic Myeloid Leukemia. Pharm Res. 2023;40(10):2413-2422.

23. Lin W, Chen Y, Unadkat JD, Zhang X, Wu D, Heimbach T. Applications, Challenges, and Outlook for PBPK Modeling and Simulation: A Regulatory, Industrial and Academic Perspective. Pharm Res. 2022;39(8):1701-1731.

24. Peters SA, Dolgos H. Requirements to Establishing Confidence in Physiologically Based Pharmacokinetic (PBPK) Models and Overcoming Some of the Challenges to Meeting Them. Clin Pharmacokinet. 2019;58(11):1355-1371.

25. Binuya MAE, Engelhardt EG, Schats W, Schmidt MK, Steyerberg EW. Methodological Guidance for the Evaluation and Updating of Clinical Prediction Models: A Systematic Review. BMC Med Res Methodol. 2022;22(1):316.

26. Cook SF, Bies RR. Disease Progression Modeling: Key Concepts and Recent Developments. Curr Pharmacol Rep. 2016;2(4):221-230.

27. Ferrer F, Chauvin J, De Victor B, Lacarelle B, Deville JL, Ciccolini J. Clinical-Based vs. Model-Based Adaptive Dosing Strategy: Retrospective Comparison in Real-World MRCC Patients Treated with Sunitinib. Pharmaceuticals (Basel). 2021;14(5):494.

28. Ferrer F, Chauvin J, Deville JL, Ciccolini J. Adaptive Dosing of Sunitinib in a Metastatic Renal Cell Carcinoma Patient: When in Silico Modeling Helps to Go Quicker to the Point. Cancer Chemother Pharmacol. 2022;89(3):565-569.

29. Phillips R, Sauzet O, Cornelius V. Statistical Methods for the Analysis of Adverse Event Data in Randomised Controlled Trials: A Scoping Review and Taxonomy. BMC Med Res Methodol. 2020;20(1):288.

30. Sun D, Gao W, Hu H, Zhou S. Why 90% of Clinical Drug Development Fails and How to Improve It? Acta Pharm Sin B. 2022;12(8):3049-3062.

31. Polasek TM, Kirkpatrick CMJ, Rostami-Hodjegan A. Precision Dosing to Avoid Adverse Drug Reactions. Ther Adv Drug Saf. 2019;10:2042098619894147.

32. Miller NA, Reddy MB, Heikkinen AT, Lukacova V, Parrott N. Physiologically Based Pharmacokinetic Modelling for First-In-Human Predictions: An Updated Model Building Strategy Illustrated with Challenging Industry Case Studies. Clin Pharmacokinet. 2019;58(6):727-746.

33. Mao J, Chen Y, Xu L, Chen W, Chen B, Fang Z, et al. Applying Machine Learning to the Pharmacokinetic Modeling of Cyclosporine in Adult Renal Transplant Recipients: A Multi-Method Comparison. Front Pharmacol. 2022;13:1016399.

34. Phe K, Heil EL, Tam VH. Optimizing Pharmacokinetics-Pharmacodynamics of Antimicrobial Management in Patients with Sepsis: A Review. J Infect Dis. 2021;222(Suppl 2):S132-S141.

35. Pallmann P, Bedding AW, Choodari-Oskooei B, Dimairo M, Flight L, Hampson LV, et al. Adaptive Designs in Clinical Trials: Why Use Them, and How to Run and Report Them. BMC Med. 2018;16(1):29.

36. Kaniz RE, Lindon AR, Rahman MA, Hasan MA, Hossain A. The Impact of Project Management Strategies on the Effectiveness of Digital Marketing Analytics for Start-up Growth in the United States. Proj Manag. 2025;4(1).

37. Easwaran V, Alshahrani S, Mantargi MJS, Bommireddy B, Khan NA, Alavudeen SS, et al. Examining factors influencing public knowledge and practice of proper face mask usage during the COVID-19 pandemic: a cross-sectional study. PeerJ. 2024;12:e16889.

38. Goruntla N, Ssesanga J, Bommireddy BR, Thammisetty DP, Kasturi Vishwanathasetty V, Ezeonwumelu JOC, et al. Evaluation of rational drug use based on who/inrud core drug use indicators in a secondary care hospital: a cross-sectional study in western Uganda. Drug Healthc Patient Saf. 2023:125-135.

39. Hatakeyama Y, Oda H, Tsunoda R, Imura Y, Maeda T, Xuan TT, et al. Genome profiling implies high genetic diversity in microsporidia isolated from the common cutworm, Spodoptera litura (Lepidoptera: Noctuidae), in Vietnam. Appl Entomol Zool. 2011;46:293-299.

40. Nguyen L, Trinh XT, Trinh H, Tran DH, Nguyen C. BWTaligner: a genome short-read aligner. Vietnam J Sci Technol Eng. 2018;60(2):73-77.

41. Abuamoud I, Lillywhite J, Simonsen J, Al-Oun M. Factors influencing food security in less popular tourists sites in Jordan's Northern Badia. Int Rev Soc Sci Humanit. 2016;11(2):20-36.