



Pharmacogenomics and Pharmacoproteomics in Infectious Disease Therapy: Personalized Approaches for Optimal Outcomes

Emily Thompson *

Department of Infectious Diseases, University of Toronto, Toronto, Canada

DESCRIPTION

Infectious diseases continue to pose significant global health challenges, even in high-income countries where advanced medical infrastructure exists. From bacterial infections such as tuberculosis and sepsis to viral diseases like HIV, hepatitis, emerging pathogens, treatment efficacy varies widely among individuals. Antimicrobial resistance, drug toxicity, variable therapeutic responses complicate standard-of-care approaches, often resulting in prolonged illness, hospitalization, increased healthcare costs. Pharmacogenomics and Pharmacoproteomics provide powerful tools to tailor antimicrobial therapy to the individual patient, optimizing efficacy and minimizing adverse effects while contributing to the global effort to combat drug resistance.

Pharmacogenomics focuses on genetic variations that influence drug metabolism, distribution, target interactions. Variants in drug-metabolizing enzymes, transporters, immune-related genes can significantly alter the pharmacokinetics and pharmacodynamics of antimicrobials. For example, polymorphisms in NAT2 determine the acetylation rate of isoniazid, a frontline antituberculosis drug. Slow acetylators are at higher risk of hepatotoxicity, whereas fast acetylators may have subtherapeutic drug exposure, leading to treatment failure. Similarly, variants in TPMT affect thiopurine metabolism during viral or immunomodulatory therapy, *CYP2C19* polymorphisms can influence the metabolism of antifungal agents like voriconazole. Pre-treatment genetic testing allows clinicians to adjust dosing strategies, reducing toxicity while maintaining therapeutic efficacy.

While pharmacogenomics provides critical insights into inherited predispositions, Pharmacoproteomics complements this by examining protein expression, modifications, interactions that reflect the patient's real-time physiological state. Proteomic profiling of plasma, tissue, or pathogen-host interactions reveals biomarkers that can predict drug response, resistance, or adverse events. For instance, in HIV therapy, proteomic analyses have

identified host and viral protein signatures associated with virology suppression or treatment failure, allowing for better selection of antiretroviral combinations. Similarly, in sepsis, proteomic biomarkers such as inflammatory cytokines, coagulation factors, endothelial proteins can indicate disease severity and guide therapy selection.

The integration of pharmacogenomics and Pharmacoproteomics enables a personalized approach to infectious disease management. Consider tuberculosis treatment: pharmacogenomics testing identifies NAT2 acetylation status, while proteomic profiling of liver enzymes, inflammatory markers, drug-protein adducts provides additional insight into hepatotoxicity risk. By combining these datasets, clinicians can adjust drug doses and monitor therapy in real time, preventing adverse events and optimizing treatment outcomes. This multi-omic approach also enhances adherence and reduces the risk of developing drug-resistant strains, which is critical for global public health.

Pharmacogenomics and proteomic integration is also vital in the management of HIV. Genetic variants in HLA-B*57:01 predict hypersensitivity reactions to batcaver, while proteomic profiling of plasma proteins can provide early indicators of immune activation or drug toxicity. Combining these layers of information ensures that patients receive effective, safe, individualized antiretroviral regimens. Similarly, hepatitis C therapy benefits from multi-omic profiling; *IL28B* genotype informs treatment response to interferon-based regimens, proteomic biomarkers of liver function and fibrosis guide therapy selection and monitoring.

Antimicrobial resistance is a major driver for the integration of pharmacogenomics and Pharmacoproteomics. Resistant pathogens often evade therapy despite appropriate dosing based on population averages. Proteomic studies of pathogens can reveal resistance mechanisms, including efflux pump expression, enzymatic drug degradation, target modifications. When combined with host pharmacogenomics data, clinicians can select drugs that are most likely to achieve effective

*Correspondence to: Emily Thompson, Department of Infectious Diseases, University of Toronto, Toronto, Canada, E-mail: e.thompson@utoronto.ca

Received: 02-Jun-2025, Manuscript No. JPP-25-29919; Editor Assigned: 04-Jun-2025, Pre QC No. JPP-25-29919 (PQ); Reviewed: 16-Jun-2025, QC No. JPP-25-29919; Revised: 23-Jun-2025, Manuscript No. JPP-25-29919 (R); Published: 30-Jun-2025, DOI: 10.4172/2153-0645.25.16.141.

Citation: Thompson E (2025) Pharmacogenomics and Pharmacoproteomics in Infectious Disease Therapy: Personalized Approaches for Optimal Outcomes. J Pharmacogenom Pharmacoproteomics. 16:141.

Copyright: ©2025 Thompson E. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

concentrations in the patient while minimizing toxicity. This precision approach reduces treatment failure and contributes to antibiotic stewardship efforts, a critical component of modern infectious disease management.

Technological advances have facilitated the integration of multi-omic data in infectious disease therapy. High-throughput sequencing enables rapid genotyping of both host and pathogen, while mass spectrometry-based proteomics allows comprehensive analysis of protein networks. Advanced computational models integrate these datasets to predict drug response, toxicity, resistance patterns, guiding clinical decision-making. Real-time monitoring of proteomic changes during therapy further enables dynamic adjustments, ensuring optimal outcomes.

Despite the capacity of pharmacogenomics and proteomic integration, challenges remain. Proteomic assays require standardization and validation, complex multi-omic datasets necessitate advanced bioinformatics infrastructure and expertise. Ethical considerations, including patient consent, privacy, data security, are critical, particularly when genetic and proteomic information has implications beyond the immediate infectious disease context. Furthermore, while high-income countries have the infrastructure to implement these technologies, equitable access remains a challenge globally. Strategies to reduce cost and increase availability will be essential to ensure that multi-omic precision therapy benefits a wide patient population.