INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

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Mini Review

Role of Pharmacogenomics in Personalized Medicine: Current Perspectives and Future Directions

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ARTICLE INFO

Published: 23 May 2026
Keywords:
Pharmacogenomics; personalized medicine; precision medicine; gene-drug interaction; CYP450; adverse drug reactions
DOI:
10.5281/zenodo.20353717

ABSTRACT

Pharmacogenomics is a rapidly evolving discipline that investigates how genetic variation shapes individual responses to drug treatments. In conventional clinical practice, unpredictable differences in drug efficacy and the occurrence of adverse drug reactions (ADRs) remain persistent challenges. Pharmacogenomics addresses these issues by enabling personalized medicine, wherein therapeutic decisions are informed by a patient's unique genetic profile. Polymorphisms in genes encoding drug-metabolizing enzymes, transporters, and pharmacological targets substantially alter pharmacokinetics and pharmacodynamics. Progress in genomic technologies has accelerated the identification of clinically relevant gene-drug interactions, allowing practitioners to optimize drug selection and individualize dosing regimens. This field has demonstrated meaningful utility across oncology, cardiology, psychiatry, and infectious disease management, where genotype-guided therapy has been associated with improved treatment outcomes and reduced toxicity. Nevertheless, barriers to broad clinical uptake persist, including the cost of genetic testing, limited professional awareness, ethical considerations, and inter-population variability in genomic data. Emerging developments in next-generation sequencing, bioinformatics tools, and clinical decision support platforms are anticipated to bridge these gaps and promote routine integration of pharmacogenomics into healthcare practice. In summary, pharmacogenomics constitutes a pivotal element of personalized medicine, providing a scientifically grounded strategy to advance drug safety, therapeutic efficacy, and the overall quality of patient care

INTRODUCTION

Pharmacogenomics is a rapidly advancing field that explores how genetic differences among

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Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

individuals influence their response to medications. By integrating principles from pharmacology and genomics, this discipline aims to refine drug therapy by aligning treatments with each patient's genetic constitution [1,2]. In daily clinical settings, variability in drug response presents a persistent challenge; patients routinely exhibit differences in treatment efficacy and may develop adverse drug reactions (ADRs) even when standard doses are prescribed [3,4].

Conventional prescribing has historically followed a generalized model, where an identical medication and dose are applied across a broad population. This strategy, however, does not account for innate genetic diversity, which critically determines how drugs are absorbed, metabolized, and exert their intended biological effects [5]. Genetic variants, most notably single nucleotide polymorphisms (SNPs), can substantially modify the functional capacity of drug-metabolizing enzymes, membrane transporters, and receptor proteins, altering both therapeutic outcomes and the likelihood of toxicity [6].

The completion of the Human Genome Project marked a watershed moment in biomedical science, deepening our understanding of genetic variability and its clinical relevance. This milestone catalyzed the emergence of personalized, or precision, medicine: an approach that incorporates genetic, environmental, and lifestyle factors into the design of treatment strategies [7,8]. By departing from the traditional "one-size-fits-all" paradigm, personalized medicine seeks to maximize therapeutic effectiveness while limiting adverse effects [9].

Pharmacogenomics occupies a central position in this transformation by offering tools to anticipate individual drug responses and guide prescribing decisions. Its clinical application has already demonstrated benefit in oncology, cardiovascular medicine, psychiatry, and infectious disease, where genotype-informed prescribing has translated into better patient outcomes [10-12].

Despite these advances, a range of factors impede the broad uptake of pharmacogenomics in clinical environments. These include the cost of genetic testing, insufficient awareness among healthcare professionals, ethical concerns surrounding genomic data, and the inherent complexity of gene-drug interaction networks [13,14]. Sustained research and the development of evidence-based clinical guidelines are expected to support integration into routine practice in the near future [15].

PRINCIPLES AND GENETIC BASIS OF PHARMACOGENOMICS

The foundational premise of pharmacogenomics is that heritable variation among individuals meaningfully shapes their response to pharmacological agents. Such variation can affect both pharmacokinetics (PK) - encompassing the absorption, distribution, metabolism, and excretion of a drug - and pharmacodynamics (PD), which describes the interaction between a drug and its molecular targets [16,17]. A thorough grasp of these genetic underpinnings is indispensable for anticipating treatment outcomes and mitigating harmful effects.

Among the contributors to interindividual variability, SNPs are the most prevalent form of genetic variation in the human genome. These single-base differences can alter protein structure or modulate gene expression, thereby influencing drug metabolism and pharmacological response [18]. Their downstream effects may include changes in enzyme catalytic activity, receptor binding affinity, or the efficiency of membrane transport proteins, each capable of modifying drug efficacy or safety.

Drug-metabolizing enzymes are central to pharmacogenomics, with the cytochrome P450 (CYP450) superfamily representing the most studied group. Members such as CYP2D6, CYP2C9, and CYP3A4 collectively govern the biotransformation of a broad array of clinically prescribed drugs [6,17]. Polymorphisms within these genes produce distinct metabolic phenotypes - poor, intermediate, extensive, and ultra-rapid metabolizers. As an example, individuals with diminished CYP2D6 activity risk drug accumulation and associated toxicity, whereas ultra-rapid metabolizers may achieve subtherapeutic plasma concentrations [7,16]. Figure 1 illustrates the spectrum of CYP450 metabolizer phenotypes and their clinical consequences.

Drug transporters constitute another layer of pharmacogenomic complexity. Proteins such as P-glycoprotein, encoded by the ABCB1 gene, govern drug absorption and its distribution across biological barriers. Genetic variants in these transporters can alter drug bioavailability and tissue penetration, with consequent effects on therapeutic response [15,18].

Pharmacological targets - principally receptors and enzymes - represent a further dimension of pharmacogenomic relevance. Variants in genes encoding these molecules can alter ligand-binding properties and modify drug efficacy. A well-characterized example is the VKORC1 gene, where polymorphisms affect patient sensitivity to warfarin and necessitate individualized dose adjustments to prevent hemorrhagic complications [10,19]. Variants in HLA genes are similarly linked to immune-mediated adverse reactions, underscoring the clinical importance of genetic screening prior to initiating certain drug regimens [11,12]. Table 1 summarizes key clinically actionable gene-drug pairs across therapeutic areas.

In aggregate, the genetic architecture underpinning pharmacogenomics is multifaceted, involving the coordinated interplay of numerous genes and biochemical pathways. These heritable factors, operating alongside environmental and physiological variables, collectively determine the net response to any given therapeutic agent.

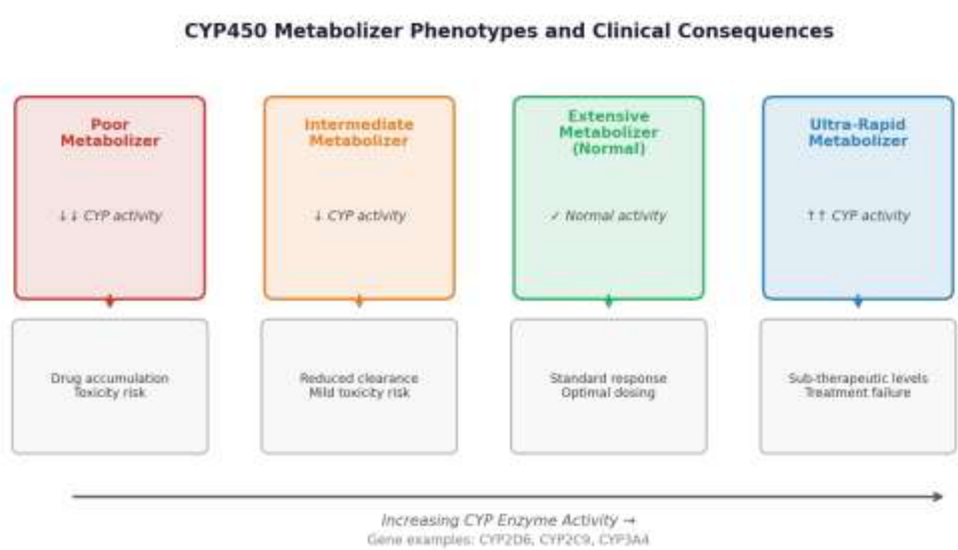

Figure 1.

CYP450 Metabolizer Phenotypes and Their Clinical Consequences. The spectrum from poor to ultra-rapid metabolizers reflects varying degrees of CYP enzyme activity, directly influencing drug plasma levels and clinical outcomes.

Table 1. ***Key Clinically Actionable Gene-Drug Pairs in Pharmacogenomics***

Gene	Key Drug(s)	Therapeutic Area	Relevant Phenotype	Clinical Risk
CYP2D6	Codeine, Tramadol, Fluoxetine	Opioid analgesics, Antidepressants	Poor / UM	Toxicity or therapeutic failure
CYP2C9	Warfarin, NSAIDs, Phenytoin	Anticoagulants, Anticonvulsants	Poor / IM	Bleeding risk / Drug toxicity
CYP2C19	Clopidogrel, Omeprazole, SSRIs	Antiplatelet, PPI, Antidepressant	Poor / UM	Reduced efficacy / ADRs
VKORC1	Warfarin	Anticoagulant	Low sensitivity	Bleeding complications
HLA-B*5701	Abacavir	HIV antiretroviral	Carrier	Severe hypersensitivity
DPYD	5-Fluorouracil, Capecitabine	Chemotherapy	Deficient	Life-threatening toxicity
TPMT	Azathioprine, 6-Mercaptopurine	Immunosuppressant, Chemotherapy	Poor/IM	Myelosuppression

ADR, adverse drug reaction; IM, intermediate metabolizer; UM, ultra-rapid metabolizer; PPI, proton pump inhibitor; SSRIs, selective serotonin reuptake inhibitors.

PHARMACOGENOMICS IN PERSONALIZED MEDICINE

Pharmacogenomics forms one of the central pillars of personalized medicine by enabling the tailoring of drug therapy to an individual's genetic profile. In contrast to conventional treatment paradigms that draw on population-level averages, personalized medicine seeks to define the genetic determinants governing drug efficacy and adverse effects, thereby sharpening therapeutic precision [9,20].

Embedding pharmacogenomics within clinical workflows enables more evidence-informed decisions regarding drug selection and dose optimization. Genetic testing can flag patients who are likely to derive benefit from a particular agent and simultaneously identify those at elevated risk of harmful reactions. This reduces dependence on empirical prescribing and curtails the trial-and-error process that frequently accompanies pharmacological management [2,5].

A key strength of pharmacogenomics lies in its capacity to stratify patients into biologically distinct subgroups according to genetic make-up. This stratification supports targeted therapy, directing treatment toward individuals most likely to respond favorably. Such an approach has proven especially productive in oncology, where interventions are frequently matched to tumor-specific genetic alterations [1,21].

Beyond improving efficacy, pharmacogenomics augments drug safety by detecting genetic predispositions to adverse reactions before they manifest. Preemptive genetic screening can forestall severe hypersensitivity events and dose-dependent toxicities, thereby lowering morbidity and easing the burden on healthcare systems [3,11]. Pharmacogenomics also holds economic merit: by reducing treatment failures, unnecessary prescriptions, and preventable hospitalizations, it contributes to more cost-effective care [9,13].

CLINICAL APPLICATIONS OF PHARMACOGENOMICS

The clinical value of pharmacogenomics is increasingly apparent across multiple therapeutic domains, where genotype-guided interventions have meaningfully improved drug efficacy and patient safety. Table 2 provides a structured overview of pharmacogenomics applications across major therapeutic areas, summarizing key genetic markers, relevant drugs, and their clinical significance.

Table 2. ***Summary of Pharmacogenomics Applications Across Major Therapeutic Areas***

Therapeutic Area	Genetic Marker(s)	Relevant Drug(s)	Clinical Significance
Oncology	HER2, EGFR, BRAF, KRAS mutations	Trastuzumab, Erlotinib, Vemurafenib	Targeted therapy; responder selection; avoidance of ineffective agents
Cardiology	CYP2C9, VKORC1 (warfarin); CYP2C19 (clopidogrel)	Warfarin, Clopidogrel	Dose optimization; reduced bleeding; improved antiplatelet response
Psychiatry	CYP2D6, CYP2C19 variants	Antidepressants, Antipsychotics	Personalized dosing; reduced side effects
Infectious Disease	HLA-B*5701 allele	Abacavir	Prevention of severe hypersensitivity; mandatory pre-screening
Pain Management	CYP2D6 (opioid metabolism)	Codeine, Tramadol	Toxicity avoidance in poor metabolizers; dose adjustment in UM
Immunology	HLA-DQ, IL28B polymorphisms	Biologic agents, IFN-based therapy	Predict response; minimize immune-related adverse events

CDSS, clinical decision support system; UM, ultra-rapid metabolizer; IFN, interferon; HIV, human immunodeficiency virus; SSRIs, selective serotonin reuptake inhibitors.

4.1 Oncology

Oncology has advanced furthest in translating pharmacogenomics into clinical practice. Targeted agents are routinely selected on the basis of tumor-specific genetic mutations. Trastuzumab, for instance, is reserved for patients with HER2-positive breast cancer, while other therapies target alterations in genes such as EGFR and BRAF [1,21]. This genotype-matched approach ensures that only patients with a plausible biological rationale for response receive a given agent.

4.2 Cardiology

In cardiovascular medicine, pharmacogenomics has made a substantive contribution to anticoagulant and antiplatelet management. Warfarin dosing is strongly modulated by polymorphisms in CYP2C9 and VKORC1, and genotype-guided dose calibration reduces the incidence of bleeding events [10,19]. Similarly, genetic variation affecting clopidogrel metabolism influences its antiplatelet potency [5].

4.3 Psychiatry

Psychiatric pharmacotherapy is characterized by substantial interindividual variability in response. Polymorphisms in CYP450 enzymes, particularly CYP2D6 and CYP2C19, influence drug metabolism and resulting plasma concentrations, altering both therapeutic effect and adverse event profiles [6,17]. Pharmacogenomic testing provides a valuable adjunct for guiding medication selection and dose individualization in this patient population.

4.4 Infectious Diseases

Pharmacogenomics has markedly improved safety in the management of infectious diseases. Pre-treatment screening for the HLA-B*5701 allele prior to initiating abacavir therapy has substantially reduced the incidence of life-threatening hypersensitivity reactions [11,12]. This example illustrates the clinical power of preemptive genetic testing in averting serious, preventable adverse events.

4.5 Other Therapeutic Areas

Pharmacogenomics is also gaining traction in pain medicine, where genetic variability shapes opioid metabolism and analgesic response, and in immunology, where genetic markers predict response to biologic therapies [16,18]. These expanding applications highlight the field's growing interdisciplinary reach.

PHARMACOGENOMIC TESTING AND CLINICAL IMPLEMENTATION

Pharmacogenomic testing is central to the translation of genomic knowledge into clinical benefit. It involves detecting genetic variants that influence drug response and delivering actionable findings that can inform prescribing. Depending on context and intent, pharmacogenomic testing may be categorized as reactive or preemptive [2,16].

Reactive testing is undertaken after drug therapy has been initiated, generally prompted by an unexpected therapeutic failure or an adverse reaction. Although this approach can illuminate the causes of treatment variability, its retrospective nature limits utility, as initial complications may already have occurred [2].

Preemptive testing screens patients for a panel of pharmacogenomic variants before any pharmacological treatment begins. This proactive strategy equips clinicians with genetic information at the point of prescribing, reducing the probability of adverse outcomes and supporting more effective treatment from the outset [2,16]. Figure 2 illustrates the stepwise pharmacogenomics workflow from patient presentation to genotype-guided prescribing outcomes.

Successful integration depends on embedding genomic data within healthcare infrastructure - particularly electronic health records (EHRs) and clinical decision support systems (CDSSs). Expert-led bodies such as the Clinical Pharmacogenetics Implementation Consortium (CPIC) play a pivotal role in standardizing interpretation and application of pharmacogenomic test results, offering evidence-based recommendations for specific gene-drug pairs [15].

Despite this progress, meaningful barriers to widespread uptake persist, including cost constraints, uneven access to genetic services, insufficient prescriber training, and concerns about data privacy [13,14]. Inconsistent regulatory frameworks and variable reimbursement policies present additional structural challenges.

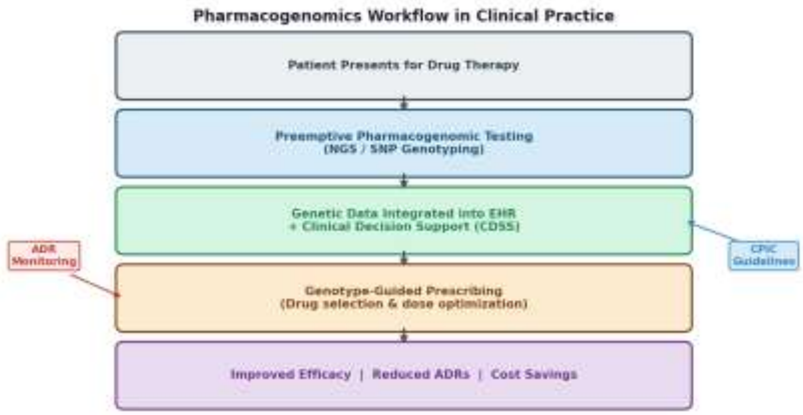

Figure 2.

Pharmacogenomics Workflow in Clinical Practice. The pathway from patient presentation through preemptive genetic testing, EHR integration, and CDSS-guided prescribing to improved clinical outcomes. CPIC, Clinical Pharmacogenetics Implementation Consortium; EHR, electronic health record; ADR, adverse drug reaction; NGS, next-generation sequencing.

BENEFITS OF PHARMACOGENOMICS

Pharmacogenomics delivers substantial advantages in optimizing pharmacotherapy by enhancing both efficacy and safety. Its primary benefit lies in enabling genotype-guided drug selection and dose individualization: by delineating the genetic factors governing drug metabolism and pharmacological response, clinicians can prescribe agents with a higher probability of producing the desired therapeutic outcome [1,5].

Reduction of ADRs represents a further major clinical gain. Adverse drug reactions remain a leading contributor to preventable morbidity and impose substantial economic burden globally. Genetic screening enables prospective identification of at-risk individuals, allowing preventive action before an adverse event occurs [3,11].

Pharmacogenomics also contributes to more rational and efficient healthcare delivery. By curtailing trial-and-error prescribing cycles, it shortens time to optimal therapy, reduces avoidable medication use, and lowers associated costs [9,13]. From a pharmaceutical development perspective, it supports the design of targeted agents and enriches clinical trial cohorts with genetically defined subgroups, amplifying efficacy signals and reducing outcome variability [14,16].

CHALLENGES AND LIMITATIONS OF PHARMACOGENOMICS

Despite its considerable promise, several scientific, economic, and ethical obstacles temper the routine clinical deployment of pharmacogenomics. Table 3 provides a structured overview of the principal challenges, their impact on implementation, and the strategies proposed to address them.

Table 3. ***Principal Challenges to Pharmacogenomics Implementation and Proposed Strategies***

Challenge	Impact on Implementation	Proposed Strategy
High testing cost	Restricts adoption in resource-limited settings; reduces equitable access	Reduced-cost panel testing; tiered reimbursement; population-level preemptive screening programs
Complex gene-drug interactions	Multifactorial nature limits predictive accuracy of single-gene models	Multi-omics integration; machine learning models incorporating polygenic and environmental data
Limited clinician training	Underutilization and misinterpretation of pharmacogenomic reports	Mandatory pharmacogenomics education in curricula; CDSS integration for real-time guidance
Ethical and privacy concerns	Patient reluctance; risk of genetic discrimination and data misuse	Robust legal frameworks (e.g., GINA); encryption standards; transparent consent processes
Population diversity gaps	Most datasets Eurocentric; reduced applicability across ethnic groups	Expanded globally diverse genomic cohorts; population-specific reference databases
Regulatory heterogeneity	Inconsistent approval and reimbursement policies across jurisdictions	International harmonization of pharmacogenomic guidelines; advocacy for health system integration

CDSS, clinical decision support system; GINA, Genetic Information Non-discrimination Act; UM, ultra-rapid metabolizer; PGx, pharmacogenomics.

Among the most prominent barriers is the cost of genetic testing, which limits accessibility particularly in resource-constrained settings [9,13]. The biological complexity of gene-drug interactions further constrains predictive accuracy, as drug response is rarely determined by a single locus but reflects the combined influence of multiple genes alongside non-genetic variables [6,18].

Limited pharmacogenomic literacy among healthcare professionals, the predominance of Eurocentric genomic datasets, and the absence of universally endorsed guidelines for many gene-drug pairs add further uncertainty [14-16]. Ethical and legal concerns - including data privacy, informed consent, and risk of discrimination - must be rigorously addressed to maintain public confidence [13]. Regulatory heterogeneity and variable reimbursement policies across jurisdictions represent additional structural impediments to routine adoption [14].

FUTURE PERSPECTIVES AND CONCLUSION

Advances in genomic technologies are poised to substantially broaden the clinical reach of pharmacogenomics. The progressive availability of next-generation sequencing (NGS) and multi-omics platforms is enabling a far more granular characterization of the determinants of drug response. The convergence of genomics with transcriptomics, proteomics, and metabolomics is expected to yield an integrated picture of disease biology that further refines individualized therapy [18,20].

The incorporation of pharmacogenomic data into EHRs, coupled with intelligent CDSSs, is likely to bring real-time, genotype-informed prescribing within reach of everyday clinical practice. Ongoing refinement of standardized guidelines by organizations such as the CPIC will continue to narrow the translational gap between genomic discovery and bedside application [15].

Artificial intelligence (AI) and machine learning are emerging as transformative tools, integrating large-scale genomic datasets with longitudinal clinical data to improve the accuracy of pharmacogenomic predictions and accelerate novel gene-drug relationship discovery [16]. Realizing the full potential of the field will require deliberate expansion of research across ethnically and geographically diverse populations to ensure equitable access to the benefits of personalized medicine [20].

CONCLUSION

Pharmacogenomics represents a transformative step forward in contemporary medicine, offering a robust scientific framework for personalized therapy. By accounting for heritable variability in drug response, it enables more precise agent selection, optimized dosing, and improved safety outcomes. While significant challenges remain, sustained advances in sequencing technologies, bioinformatics, and decision support infrastructure - alongside growing commitment to clinician education and equitable policy frameworks - are expected to drive integration into mainstream healthcare. Pharmacogenomics will ultimately play an indispensable role in realizing the full promise of personalized medicine, contributing to improved clinical outcomes and more efficient, patient-centered health systems.

AUTHOR CONTRIBUTIONS

SC was solely responsible for all aspects of this manuscript, including the conception of the review framework, literature search, critical appraisal and synthesis of evidence, drafting of the manuscript, and preparation of the final submitted version.

COMPETING INTERESTS

The author declares no competing financial or non-financial interests in relation to the work descried in this manuscript.

DATA AVAILABILITY STATEMENT

This manuscript is a narrative review and does not involve the generation of primary data. All sources cited are available in the public literature and are referenced in full.

FIGURE LEGENDS

Figure 1. *CYP450 Metabolizer Phenotypes and Their Clinical Consequences.* The spectrum from poor to ultra-rapid metabolizers reflects varying degrees of CYP enzyme activity. Poor metabolizers risk drug accumulation and toxicity due to diminished enzyme activity, whereas ultra-rapid metabolizers may fail to achieve therapeutic plasma concentrations. The intermediate and extensive phenotypes represent intermediate and normal activity, respectively. Gene examples include CYP2D6, CYP2C9, and CYP3A4. UM, ultra-rapid metabolizer.

Figure 2. *Pharmacogenomics Workflow in Clinical Practice.* The stepwise pathway from patient presentation through preemptive pharmacogenomic testing (next-generation sequencing or SNP genotyping), integration of genetic data into electronic health records (EHRs) via clinical decision support systems (CDSSs), and genotype-guided prescribing, culminating in improved drug efficacy, reduced adverse drug reactions (ADRs), and cost savings. CPIC guidelines and real-time ADR monitoring feed back into the prescribing loop. CPIC, Clinical Pharmacogenetics Implementation Consortium.

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HOW TO CITE: Sumit Choudhary, Kanishka Sharma, Aayush Mali, Manish Prajapati, Role of Pharmacogenomics in Personalized Medicine: Current Perspectives and Future Directions Int. J. of Pharm. Sci., 2026, Vol 4, Issue 5, 6211-6220, https://doi.org/10.5281/zenodo.20353717