Pharmacogenomics Unveiled: Customizing Care with Genetic Insights

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Abstract

Pharmacogenomics (PGx) is an emerging field of pharmacology that investigates how genetic variability affects individual responses to medications, particularly in relation to the drug's pharmacokinetic and pharmacodynamic properties. This review aims to provide a comprehensive understanding of PGx and its role in optimising drug therapy based on genetic differences. Which is focuses on tailoring healthcare interventions by considering each patient's unique molecular, physiological, ecological, and behavioural characteristics. Pharmacogenomics enables improved drug dose selection by addressing enzymatic and genetic variability. Its clinical relevance spans cancer treatment personalisation, psychiatric medication optimisation, reduction of adverse drug reactions, and the identification of individual drug metabolism capabilities. Additionally, this review also highlights major genetic markers involved in PGx, such as Cytochrome P450 enzyme variants, HLA polymorphisms, and genes influencing drug metabolism and response. It also describes how this approach can improve treatment results, reduce side effects, and help doctors understand how different people process drugs.

Keywords- CYP450, Genetic variations, Personalized medicine, Pharmacogenomics, Whole genome sequencing.

1. Introduction

Pharmacogenomics (PGx) is an emerging field of pharmacology that explores how genetic variation affects individual responses to drugs. It focuses particularly on the pharmacokinetic (absorption, distribution, metabolism, and excretion) and pharmacodynamic (drug-target interactions and downstream effects) properties of therapeutics. The integration of PGx into clinical practice aims to improve therapeutic efficacy, minimise adverse drug reactions, and advance the movement toward truly personalised medicine [1].

One of the earliest documented examples of pharmacogenetic variation is glucose-6-phosphate dehydrogenase (G6PD) deficiency, which was linked to drug-induced hemolysis following treatment with certain antimalarials and sulphonamides. [2]. The field of pharmacogenetics was officially introduced by Friedrich Vogel in 1959 [3], while Wilhelm Johannsen's distinction between genotype and phenotype laid the conceptual foundation for linking genetic traits to drug response [4]. Together, these pioneering studies laid the groundwork for what would become modern pharmacogenomics.

With the advent of high-throughput DNA sequencing, bioinformatics, proteomics, and wireless physiological monitoring, the ability to analyze inter-individual variability in drug response has grown significantly. [5]. These tools have allowed clinicians and researchers to better understand the molecular mechanisms driving variability in efficacy and toxicity across diverse populations. PGx has been instrumental in shaping the evolving landscape of personalized medicine, a field that customizes healthcare strategies based on individual genetic, molecular, environmental, and lifestyle characteristics. [6].

Clinical pharmacogenetics is exemplified by the study of cytochrome P450 (CYP450) enzymes, particularly CYP2D6, CYP2C9, and CYP2C19, which are responsible for metabolizing over 70% of prescribed drugs [7]. Genetic polymorphisms in these enzymes lead to variable metabolic phenotypes ranging from poor to ultra-rapid metabolizers, necessitating

dose adjustments or alternative treatments. Likewise, human leukocyte antigen (HLA) gene variants, such as HLA-B*15:02 and HLA-B*57:01, are associated with severe adverse drug reactions, including Stevens-Johnson syndrome and hypersensitivity to abacavir [8].

Challenges to routine PGx implementation include high testing costs, lack of standardized guidelines, limited provider awareness, and ethical concerns over genomic data privacy. Most studies focus on Western populations, leaving ethnic diversity underrepresented. In countries like India, complex genetics, along with infrastructural and economic barriers, add further hurdles. [9]. However, growing awareness, government initiatives, and academic efforts are paving the way for PGx adoption in developing nations. By improving therapeutic precision and reducing adverse drug reactions, a major cause of hospitalizations and healthcare costs, PGx holds significant clinical and economic potential [10].

This review aims to provide a comprehensive overview of pharmacogenomics by exploring its scientific basis, clinical relevance, major genetic markers, and emerging technologies. Furthermore, the review addresses current limitations and future directions, emphasising the role of PGx in advancing patient-centred care and achieving optimal therapeutic outcomes.

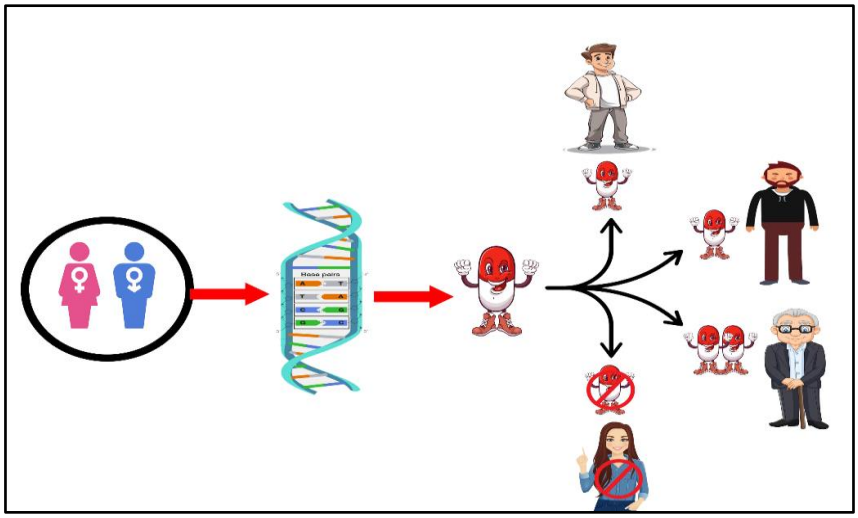

Fig. 1: Showing the genetic material identification role in personalization for an individual

1.1 Understanding how genetic variations impact drug response

Genetic variations drive inter-individual differences in drug response by influencing absorption, metabolism, distribution, elimination, and drug–target interactions. Pharmacogenomics (PGx) identifies these variations, often single-nucleotide polymorphisms (SNPs), to optimize therapy and reduce adverse effects [1].

Drug metabolism is particularly affected by polymorphisms in cytochrome P450 enzymes (CYP2D6, CYP2C9, CYP2C19), which define four phenotypes: poor, intermediate, extensive (normal), and ultra-rapid metabolizers. [11]. For instance, ultra-rapid metabolizers of codeine convert it to morphine quickly, raising toxicity risk. [12]. Similarly, polymorphisms in drug transporters (e.g., SLCO1B1, ABCB1) alter distribution and excretion; SLCO1B1 variants increase statin-induced myopathy risk [13]. VKORC1, encoding warfarin's target, affects anticoagulant response and dosage (Table 1) [7]. PGx helps identify biomarkers to prevent ADRs, guide drug and dose selection, and improve treatment design, while also offering insights that support new drug development. [8].

Table 1. Common Gene Drug Interactions in Pharmacogenomics

Drug	Gene(s) Involved	Clinical Impact	References
Warfarin	VKORC1, CYP2C9	Alters the dose requirement and bleeding risk	(7, 13, 14)
Codeine	CYP2D6	Risk of toxicity in ultra-rapid metabolizers	(11)
Simvastatin	SLCO1B1	Myopathy risk with reduced hepatic uptake	(12)
Abacavir	HLA-B*57:01	Hypersensitivity reaction	(8)
Carbamazepine	HLA-B*15:02	Stevens-Johnson Syndrome in Asian populations	(8)
Clopidogrel	CYP2C19	Reduced antiplatelet effect in poor metabolizers	(1)

A prominent example is warfarin, a commonly prescribed oral anticoagulant with a narrow therapeutic index. VKORC1 polymorphisms affect the required dose to achieve a stable INR (International Normalized Ratio). Studies show that VKORC1 and CYP2C9 genotypes account for 30–40% of the variability in warfarin dosing. [14]. The International Warfarin Pharmacogenetics Consortium conducted a large-scale analysis involving more than 4,000 individuals of diverse ancestry. This study demonstrated the potential clinical utility of integrating genetic testing into warfarin dosing algorithms alongside INR monitoring. [15].

In the field of oncology, pharmacogenomics becomes more complex due to the involvement of two genomes: the inherited (germline) genome of the patient and the somatic genome of the tumor. Often, drug response is more significantly influenced by the tumor's genetic mutations. For instance, HER2 amplification in breast cancer predicts response to trastuzumab, and EGFR-activating mutations in non-small-cell lung cancer (NSCLC) predict sensitivity to gefitinib, an EGFR inhibitor. [16-18].

Table 2. Applications of Pharmacogenomics in Oncology

Cancer Type	Gene/Mutation	Drug Affected	Clinical Outcome	References
Breast cancer	HER2 amplification	Trastuzumab	Enhanced survival and tumor response	[16]
NSCLC	EGFR mutations	Gefitinib	Greater sensitivity and progression-free survival	[17, 18]
Colorectal cancer	KRAS mutations	Cetuximab	Resistance if KRAS is mutated	[1]
Breast/colon cancer	DPYD variants	Fluorouracil	Increased risk of toxicity	[1]

1.2 Optimization of drug selection and dosing for individual patients

Optimizing drug selection and dosing is central to personalized medicine, with pharmacogenomics playing a key role [1]. Variability in drug absorption, metabolism,

distribution, and excretion drives differences in efficacy and adverse reactions. Pharmacogenomic testing identifies genetic polymorphisms influencing drug response, enabling tailored therapy. [19].

Genome sequencing has deepened understanding of disease mechanisms and how genetic variation affects drug efficacy and toxicity. Drugs with narrow therapeutic indices, such as anticoagulants and immunosuppressants, are especially sensitive to these differences. Statins, used for hyperlipidemia, show marked variability in safety and efficacy; for example, simvastatin reduced LDL cholesterol by up to 50% at 160 mg/day in a 156-patient cohort. [20]. Warfarin, a vitamin K antagonist, illustrates the impact of pharmacogenomics, with CYP2C9 and VKORC1 variants explaining up to 40% of dose variability. Preemptive genotyping (CYP2C9 *2, *3; VKORC1 -1639G>A) improves dose prediction and reduces hemorrhagic risk [13, 14]. Clopidogrel, an antiplatelet prodrug, requires CYP2C19 activation. Loss-of-function alleles (*2, *3) produce poor metabolizers with reduced efficacy and higher cardiovascular risk. The FDA recommends CYP2C19 genotyping, with prasugrel or ticagrelor preferred in poor metabolizers [21]. Rare but important variants also affect outcomes. Succinylcholine can cause prolonged paralysis in patients with atypical butyrylcholinesterase, leading to extended apnea during anesthesia [22]. Oncology and immunosuppressive therapy also benefit: UGT1A1*28 testing prevents irinotecan-induced neutropenia, while TPMT genotyping avoids myelosuppression with azathioprine or 6-mercaptopurine [23, 24].

Guidelines from CPIC and DPWG translate genetic findings into actionable recommendations, increasingly integrated into electronic health records and decision-support tools. [25]. Genotype-guided therapy has proven cost-effective by reducing adverse events, avoiding ineffective treatments. [26], and lowering hospitalizations [27], though wider adoption still requires standardization, clinician training, and stronger infrastructure [28].

2. Major Clinical Applications

2.1 Cancer treatment personalization

Pharmacogenomics has transformed cancer treatment by replacing the traditional "one-size-fits-all" approach with individualized therapies tailored to genetic profiles. [29]. In oncology, where variability in drug response and toxicity is high, genetic testing now guides key treatment decisions (Table 2). HER2 amplification occurs in 15–20% of breast cancers and is linked to aggressive disease; trastuzumab targeting HER2 has markedly improved survival [16, 30]. In non-small-cell lung cancer (NSCLC), EGFR mutations predict strong responses to tyrosine kinase inhibitors such as gefitinib and erlotinib. [4, 5].

Colorectal cancer patients with KRAS mutations (~40%) are resistant to anti-EGFR monoclonal antibodies, making KRAS, BRAF, and MSI testing essential for therapy selection. [6, 7]. Similarly, DPYD mutations impair 5-FU/capecitabine metabolism, causing severe toxicity. [8, 9], while TPMT genotyping in acute lymphoblastic leukemia enables thiopurine dose adjustment to prevent myelosuppression [10].

Table 3. Key Pharmacogenomic Biomarkers in Personalized Cancer Therapy

Cancer Type	Gene/Biomarker	Targeted Drug/Approach	References
Breast Cancer	HER2 amplification	Trastuzumab, Pertuzumab	[2, 3]
NSCLC	EGFR mutation	Gefitinib, Erlotinib	[4, 5]
Colorectal Cancer	KRAS mutation	Avoid anti-EGFR therapy (e.g., Cetuximab)	[6]
Colorectal Cancer	BRAF V600E, MSI	Immunotherapy, BRAF inhibitors	[7]
Multiple Cancers	DPYD mutation	Reduce dose of 5-FU/Capecitabine	[8, 9]
Acute Leukemia	TPMT deficiency	Adjust 6-MP dose	[10]

2.2 Psychiatric medication optimization

Pharmacogenomics is transforming psychiatric treatment by tailoring medications to genetic profiles, addressing the variability in efficacy and tolerability that limits trial-and-error prescribing. Polymorphisms in genes such as CYP2D6 and HLA-B*15:02 significantly affect drug metabolism and adverse reaction risk, and identifying these markers supports better drug selection and dosing to improve outcomes and minimize side effects.

Guidelines from groups like CPIC now integrate these insights into psychiatric care, with case studies showing benefits in treatment-resistant disorders and improved adherence through reduced side effects. However, challenges remain, including standardized testing, integration into electronic health records, clinician education, and privacy concerns. Advances in next-generation sequencing, machine learning, and multi-omics are helping to address these barriers, paving the way for more precise and patient-centered mental health care. [16].

2.3 Reducing adverse drug reactions

Rapid advances in pharmacogenomics have greatly reduced adverse drug reactions (ADRs) by clarifying their genetic basis, enabling more accurate prescribing, lowering healthcare costs, and improving outcomes. Evidence from over 1,500 publications (1964-2018) highlights its clinical impact on ADR prevention.

Pharmacogenomic variability explains more than 80% of interindividual differences in drug efficacy and safety, with rare variants alone accounting for ~40% of functional variability in 146 key drugs. Overall, over 240 genes are linked to ADR susceptibility, while more than 400 genes influence drug metabolism, transport, and pharmacodynamics. [17].

2.4 Identifying patient-specific drug metabolism capabilities

Cytochrome P450 (CYP) and drug transporter variants differ widely across populations, producing significant interindividual and interethnic variability in drug metabolism and

response. High frequencies of clinically relevant alleles in certain regions highlight the need for population-specific pharmacogenomic strategies.

Technologies such as whole genome haplotyping, SNP profiling, gene expression analysis, and multigene platforms (biochips, microarrays) have advanced the study of drug response variability, supporting drug development, target discovery, and personalized therapy.

Despite progress, optimizing individual dosing remains challenging due to complex pharmacokinetic and pharmacodynamic variability. Precision medicine requires integrating genetic and non-genetic contributors to drug response to reduce ADRs and improve efficacy.

Clinical guidelines based on genotyping are critical for interpreting altered responses and drug interactions. Identifying and validating ADR biomarkers will further enable genotype-phenotype guided prescribing, advancing personalized care across diverse populations. [18].

3. Key genetic markers

3.1 Cytochrome P450 enzyme variations

Cytochrome P450 (CYP) enzymes are major determinants of drug response, with CYP1, CYP2, and CYP3 families metabolizing 70–80% of clinical drugs. Key hepatic isoforms include CYP2C9, CYP2C8, CYP2E1, CYP3A4, and CYP1A2, while CYP2B6, CYP2A6, CYP2D6, CYP2C19, and CYP3A5 occur at lower levels. Extrahepatic forms like CYP1B1, CYP2J2, and CYP1A1 contribute to tissue-specific metabolism and toxicity.

CYP activity is influenced by genetic polymorphisms, induction, cytokines, hormones, disease, sex, age, and ethnicity, with genetic variants having the greatest impact. Polymorphisms in CYP2C9, CYP2D6, CYP2C19, CYP2B6, CYP3A5, and CYP2A6 define phenotypes ranging from poor to ultrarapid metabolizers. Poor metabolizers risk toxicity from drug accumulation, while ultrarapid metabolizers may have reduced efficacy due to rapid clearance. [19].

Incorporating CYP genotype testing into clinical practice enables tailored drug choice and dosing, minimizing adverse effects and maximizing therapeutic benefit

3.2 HLA gene polymorphisms

Diversification of classical HLA genes (HLA-A, HLA-B, HLA-C, HLA-DR, HLA-DQ) shapes population-specific allele and haplotype frequencies, influencing drug response and adverse drug reactions (ADRs). Ethnically informed HLA profiling is therefore essential for identifying reliable pharmacogenetic markers and guiding therapy. Clinical guidelines from CPIC and IDSA recommend HLA screening with high-risk drugs, while EMA and USFDA reinforce this through boxed label warnings. The HLA-B*57:01 allele is strongly linked to abacavir hypersensitivity, making pre-treatment screening vital to prevent life-threatening reactions. Likewise, HLA-B*15:02 is associated with carbamazepine-induced Stevens–Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN), especially in Asian populations, prompting mandatory screening before CBZ use. Economic analyses further support HLA-B*15:02 testing as a cost-effective strategy by preventing severe ADRs and reducing healthcare burden. [20].

3.3 Pharmacokinetic and pharmacodynamic gene variants

PGx aims to enhance efficacy and safety by considering genetic variations in drug-metabolizing enzymes, especially CYP isoenzymes, and transporter proteins that affect drug pharmacokinetics and pharmacodynamics. This personalized approach tailors therapy to minimize adverse drug reactions (ADRs) and improve outcomes [31]. In cardiovascular pharmacotherapy, CPIC has reviewed genetic influences on beta-blocker response, focusing on ADRB1, ADRB2, ADRA2C, GRK4, GRK5, and CYP2D6. Among beta-blockers, metoprolol shows the strongest evidence: as a CYP2D6 substrate, poor metabolizers (PMs) have over twice the elimination half-life and a five-fold higher AUC than normal metabolizers, increasing risk of side effects. Variants in ADRB1 (p.Ser49Gly, p.Gly389Arg) also affect

response. The “Gly” allele reduces cAMP signaling, mimicking beta-blockade, while the p.Ser49/p.Arg389 genotype enhances responsiveness, often requiring dose adjustment.

These genotype–phenotype links highlight the value of pre-emptive PGx testing in guiding beta-blocker therapy to optimize efficacy and reduce toxicity [21].

4. Technological approaches

4.1 Whole genome sequencing

Whole exome sequencing (WES) is a next-generation sequencing method that determines the nucleotide sequence of all coding regions (exons), which make up only 1-2% of the genome but harbor ~85% of disease-related variants. By sequencing these regions, WES helps identify genetic determinants of drug response. [22].

Pharmacogenomics (PGx), a key area of personalized medicine, studies how genetic variation influences drug response. Genome and exome sequencing enable the detection of polymorphisms affecting drug metabolism, efficacy, and toxicity, guiding optimized dosing and reducing adverse reactions. For instance, CYP2D6 polymorphisms alter codeine metabolism: ultra-rapid metabolizers risk morphine toxicity, while poor metabolizers obtain little analgesia. Similarly, CYP3A4 and SLCO1B1 variants affect statin metabolism, influencing efficacy and risk of muscle side effects, which occur in up to 10% of users. PGx-guided strategies can help prevent such outcomes. Precision medicine expands on PGx by incorporating genetic, lifestyle, and environmental factors into therapy, aiming for treatments tailored to each patient’s unique profile to maximize benefit and minimize harm [23, 24].

4.2 Targeted genetic panel testing

Genetic variability is a key factor behind interindividual differences in drug response, affecting systemic exposure, efficacy, and safety. Pharmacogenomic (PGx) testing identifies variant alleles in ADME genes or drug targets to predict such variability. [27, 28].

The Clinical Pharmacogenetics Implementation Consortium (CPIC) has translated these insights into practice, compiling evidence for ~442 gene drug pairs (e.g., CYP2D6/codeine) and issuing genotype-based dosing guidelines. In clinical use, targeted genotyping predominates, detecting known polymorphisms of clinical relevance, though the test scope varies by laboratory. For broader coverage, advanced platforms sequence pharmacogenes such as CYP1A2, TPMT, CYP2C19, SLCO1B1, CYP3A4, CYP3A5, CYP2C9, CYP2D6, DPYD, UGT1A1, and VKORC1, which influence metabolism, efficacy, and toxicity across drug classes.

To improve the detection of both common and rare variants, in silico-designed panels and next-generation platforms have been developed. One example is PGx-seq, a third-generation targeted sequencing panel from the Pharmacogenomics Research Network (PGRN), which includes pharmacogenes and clinically important HLA alleles. Instead of reporting exact alleles, these platforms often provide actionable results, such as positive or negative status for HLA-B*57:01*, *HLA-B*15:02, HLA-B*58:01, and HLA-A*31:01, thereby enhancing clinical utility [29].

4.3 Bioinformatics analysis of genetic data

The Human Genome Sequencing Center has adopted PGx-seq, a third-generation targeted sequencing platform originally developed by the Pharmacogenomics Research Network to capture a comprehensive panel of clinically significant pharmacogenetic genes. This platform enables the identification of both common and rare pharmacogenomic variants that affect drug response. Although some HLA-B and HLA-A alleles were examined in the RIGHT 10K Study, they were not included in the detailed allele-level reporting in this context. Instead, results were expressed in a binary format (positive/negative) for specific pharmacogenetically actionable alleles such as HLA-B*15:02, HLA-A*58:01, HLA-B*31:01, and HLA-B*57:01, without reporting the exact underlying allele identities. [30].

The present review aims to explore the potential clinical implications of pharmacogenomic testing by integrating previously published data from recent PubMed-indexed studies and analyzing this data using high-throughput in silico prediction methods. Multiple advanced bioinformatics tools were employed to perform this integrative analysis, including STRING v12 for protein–protein interaction networks [32], Cytoscape v3.10 for network visualization and modeling [33], miRTargetLink 2.0 for microRNA-target interactions [34], and Network Analyst for meta-analysis and network-based visualization [35]. To ensure relevance and clinical applicability, PharmGKB (https://www.pharmgkb.org/) was utilized during the final selection phase to exclude genes lacking pharmacogenetic significance based on annotated clinical interpretations. Additionally, drug-associated genomic information referenced from the FDA Table of Pharmacogenomic Biomarkers in Drug Labeling was consulted for cross-validation of gene–drug associations [36].

4.4. Machine learning integration

PGx research increasingly employs advanced computational tools such as big data analytics, machine learning (ML), and deep learning (DL) to analyze complex biomedical and genetic datasets. These approaches reveal patterns and associations that drive progress in pharmacogenomics and personalized medicine. Integrating ML and DL with pharmacokinetic and genomic data supports high-throughput analyses useful in drug discovery and therapy optimization. [2]. A key strength of DL is representation learning, which eliminates manual feature extraction and enhances predictive accuracy in genomics and pharmaceutical research. In pharmacogenomics, especially for psychiatric treatments like depression, these models help identify genetic markers predictive of drug efficacy and adverse effects, enabling more personalized antidepressant therapies. Developing bioinformatics frameworks that incorporate ML and DL is, therefore, critical for therapeutic discovery, prognostic modeling, and advancing precision medicine in pharmacogenetics [2, 30].

5. Potential benefits

5.1 Improved treatment efficacy

The discovery of driver somatic mutations has greatly advanced targeted drug therapy [37]. A landmark example is imatinib, which targets the BCR-ABL fusion protein in chronic myeloid leukemia (CML) [4]. Targeted approaches were first widely used in breast cancer, where ER testing guided tamoxifen therapy. The introduction of HER2 testing further enabled trastuzumab (Herceptin) use in HER2-positive cases. Breast cancer classification into molecular subtypes and transcriptomic profiling now refine chemotherapy decisions [5].

Similar advances include ALK and EGFR testing in lung cancer to guide crizotinib and gefitinib use, and KRAS/EGFR profiling in colorectal cancer to predict cetuximab response [1]. In hepatitis C, the discovery of the ss469415590 variant, linked to IFNL4 expression, highlights the role of host genetics in interferon response, particularly among African ancestry patients. Although direct-acting antivirals are standard, interferon regimens remain relevant for select groups.

Pharmacogenomics underpins personalized medicine by tailoring treatment to genetic profiles. Yet, despite successes, many biomarkers remain in discovery rather than clinical use. Progress depends on expanding population-scale genomic data, cost-effective genotyping, and integrating electronic health records with genomic databases. Advances in computational biology and systems pharmacology further enable translation of genetic insights into practice. Ultimately, the adoption of pharmacogenomics will require not only scientific advances but also collaboration among researchers, clinicians, regulators, industry, and patients to bridge the gap between discovery and clinical implementation [13].

5.2 Reduced side effect risks

Approximately 60% of adverse drug reactions (ADRs) are attributed to drugs metabolized by polymorphic phase I drug-metabolizing enzymes (DMEs). Genetic polymorphisms in these

enzymes significantly affect individual drug metabolism, influencing both efficacy and toxicity. Based on genotyping or phenotyping, patients can typically be categorized into one of four metabolic phenotypes: ultrarapid metabolizers (UMs), extensive (or normal) metabolizers (EMs), intermediate metabolizers (IMs), and poor metabolizers (PMs).

In individuals classified as PMs, the activity of the relevant metabolizing enzyme is either absent or significantly reduced. Consequently, when such individuals are administered a standard dose of an active drug (i.e., a drug that exerts its therapeutic effect in its original form), drug biotransformation is impaired, leading to the accumulation of the parent compound. In contrast to EMs, PMs are likely to exhibit higher systemic drug concentrations, which may increase the risk of dose-dependent toxicity and adverse reactions. These pharmacokinetic variations underscore the importance of pharmacogenetic testing in optimizing drug selection and dosing to ensure both safety and therapeutic effectiveness.

5.3 More precise medication selection

Genetic heterogeneity is a key factor in interindividual and interethnic variability in drug response. Leveraging this variability can reduce adverse drug reactions (ADRs), improve clinical trial designs, and support rational drug development. Understanding genetic differences helps identify individuals most likely to benefit from therapies while minimizing risk, making genotype-guided interventions highly valuable for better outcomes.

This highlights the need for a shift toward personalized medicine. In the U.S., about 3.1 billion prescriptions are written annually, with 2.1 million causing adverse reactions, over 1 million hospitalizations, and nearly 100,000 deaths [38].

The Human Genome Project (HGP) and related advances are transforming pharmaceutical research by generating vast genetic data that require new bioinformatics and analytical tools. Technologies such as DNA microarrays and microfluidic devices have enabled efficient gene

mapping and sequencing, offering scalable, cost-effective solutions for genotype-driven drug discovery, patient stratification, and therapeutic optimization [30, 39].

6. Current challenges

Pharmacogenomics, though highly promising, faces challenges to routine clinical adoption. The complexity of human genetics, particularly polymorphisms in CYP450 enzymes, makes universal treatment protocols difficult. Cost is another barrier, especially in low- and middle-income countries like India, where limited budgets restrict access to genetic testing, interpretation, and personalized regimens. Establishing the required infrastructure advanced labs, diagnostic tools, and trained personnel also demands significant investment.

Limited awareness among healthcare providers and the public further slows adoption, as knowledge of pharmacogenomics' long-term benefits remains low. Overcoming these hurdles requires collaboration among researchers, policymakers, and healthcare systems. Investments in education, training, policy support, and funding both governmental and private are essential to build infrastructure and expertise, enabling pharmacogenomics to become a practical component of everyday medicine.

7. Conclusion

In conclusion, pharmacogenomics is a rapidly advancing field with transformative potential in modern medicine, notably in personalized cancer therapy and reducing adverse drug reactions (ADRs). Warfarin exemplifies how genetic polymorphisms influence safety and efficacy, requiring genotype-guided dosing. Advances in methodologies and genomic data highlight its role in precision medicine, enabling tailored therapies that enhance outcomes, optimize efficacy, and minimize toxicity.

However, implementation especially in countries like India is limited by high costs, poor infrastructure, and low awareness. Overcoming these challenges demands investment, education, and strong policy support. With capacity-building and interdisciplinary

collaboration, pharmacogenomics can be integrated into routine care, fostering a personalized, predictive, and preventive healthcare model.

Author Contributions

NP contributed to the study conception and design wrote the first draft of the manuscript, revised and approved the manuscript. SC contributed to the study conception revised and approved the manuscript. VB contributed to the study conception, design the figure, revised and approved the manuscript. AP contributed to the study conception, revised and approved the manuscript. CY contributed to the study conception, revised and approved the manuscript. DC contributed to the study conception, revised and approved the manuscript.

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