



PHARMACOGENOMICS IN PRECISION MEDICINE: MOLECULAR DETERMINANTS OF DRUG RESPONSE, TOXICITY, AND PERSONALIZED THERAPEUTIC STRATEGIES

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ABSTRACT

Pharmacogenomics has emerged as a key component of precision medicine by explaining how genetic variation contributes to differences in drug response, therapeutic efficacy, and susceptibility to adverse effects. Variability in drug-metabolizing enzymes, transporters, molecular targets, and signaling pathways can influence both pharmacokinetic and pharmacodynamic outcomes. This comprehensive review aimed to synthesize current evidence on the molecular and genetic determinants of drug response and toxicity and to examine their application in pharmacogenomics-guided precision medicine and personalized therapeutic strategies. Relevant peer-reviewed literature was identified through major scientific databases using terms related to pharmacogenomics, precision medicine, genetic variation, drug metabolism, drug transport, adverse drug reactions, and personalized therapy. The selected studies were organized into major thematic areas covering molecular mechanisms, toxicity, clinical applications, genomic technologies, implementation, and future directions. The reviewed evidence showed that genetic variation can alter drug metabolism, transport, target sensitivity, and toxicity risk. Pharmacogenomics-guided strategies have potential applications in oncology, cardiovascular medicine, psychiatry, infectious diseases, and other therapeutic areas. Genomic technologies and biomarker-based approaches are increasingly supporting individualized drug and dose selection, although challenges remain in population diversity, variant interpretation, clinical validation, accessibility, and routine implementation. Pharmacogenomics provides a strong molecular foundation for improving treatment efficacy and safety. Continued clinical validation and integration with broader biological and clinical information are essential for advancing personalized pharmacotherapy.

Keywords: Pharmacogenomics, Precision medicine, Drug response, Drug toxicity, Personalized therapy



1. Introduction

There are significant inter-personal differences in drug response even when a drug is given at a similar dosage for the same clinical condition. The differences in therapeutic outcome could manifest as suboptimal effectiveness, heightened pharmacologic effects, therapeutic failures, or drug side effects. This variation is due to genetic, physiological, environmental, demographic and disease factors. The inherited genetic differences may increase in importance among these determinants, due to the fact that they can change the structure of proteins related to drug metabolism, transport, recognition of drugs by the receptor and cellular response. Therefore, pharmacogenomics can give a scientific knowledge of the reasons behind the fact that some patients may benefit from a drug treatment yet be unable to tolerate it because of side effects or lack of response. Pharmacogenomics can thus provide a scientific answer to the question of why a drug treatment may be effective and tolerated in one patient but not another due to side effects or the lack of response. This recognition of the variability has increased the importance of precision medicine in contemporary pharmacotherapy. Traditional medical therapy has involved using population-based guidelines and uniform guidelines for drug dosages and treatment. This approach is suitable for many clinical scenarios, but is unable to capture biological differences between patients. Using the same treatment protocol for everyone could lead to subtherapeutic blood levels of drugs in some people and higher risk of toxicity in others. This is particularly important for drugs with a narrow therapeutic index, high inter-patient variation or severe side effects. Another approach is pharmacogenomics, which involves using a person's genetic characteristics to guide medication choices for more individualized treatment, tailored to individual patient needs (Suliman, 2025; Alzahrani et al., 2024).

Genetic factors can play a role in variability in drug response related to pharmacodynamics and pharmacokinetics. Pharmacokinetic variation can occur due to genetic polymorphisms that affect enzymes involved in drug activation, metabolism and elimination, and transport proteins that influence the absorption and tissue distribution of drugs. Pharmacodynamic variation refers to receptors, drug targets, signaling proteins or other molecules that modulate the therapeutic response of the drug. Therefore, changes in the genetic variants can impact the amount of drug delivered to the target or the impact of the activation of the target. This understanding of these molecular relationships plays a crucial role in pharmacogenomics and offers a valuable basis for personalized therapeutic decisions (Guru et al., 2026; Bhatt et al., 2025).

The ability to study these determinants has greatly expanded with the advances in genomic technologies. While the earlier pharmacogenetic studies were mainly based on single candidate genes, the current research can evaluate more variants in different pathways. Advances in genotyping, next-generation sequencing, whole-exome and whole-genome analysis and other molecular profiling technologies have made it more and more possible to discover genetic variations that may be relevant for drug response. These technologies also enable the analysis of rare variants and complex molecular profiles that may not be detected by traditional testing. The coupling with precision medicine is providing a broader analysis of biology of treatment relatedness (Cecchin & Stocco, 2020; Hafeez & Maryam, 2026) beyond selected gene–drug connections.

One of the main clinical drivers of pharmacogenomics is the objective to increase the effectiveness and safety of drugs. Poor treatment performance may lead to long course of the disease, significant utilization of healthcare resources and frequent changes in treatment; adverse effects related to genetic factors can lead to significant morbidity and discontinuation of treatment. Pharmacogenomic data may help to determine which patients are likely to benefit from therapy or may be more likely to develop a high level of toxicity before starting treatment. This is especially important when drug metabolism, immune response or molecular changes associated with disease are known to have significant impact on clinical outcome. Thus, the use of pharmacogenomic data in toxicological evaluation could potentially minimize unnecessary drug toxicity and enhance the benefit–risk ratio of drug therapy (Jalaiah, 2026).

An important extension of this is the development of personalized therapeutic strategies. Precision



Pharmacotherapy is an approach that acknowledges the advantages of using genetic information along with clinical, biological, and environmental features to choose a drug and dose rather than just relying on diagnosis. For instance, decisions about using a standard medicine, changing the dose, needing more monitoring, or trying a different medicine might be influenced by pharmacogenomics. Such an approach is also gaining importance for rare diseases, where rare events and unique genetic processes can be particularly difficult to deal with in the development of treatment and the prescription process (Roman, 2025).

The importance of pharmacogenomics in this day and age can be found in its ability to increasingly associate molecular variation with tangible therapeutic choices. It has spread to other medical domains, including oncology, cardiovascular medicine, psychiatry, infectious diseases and others where treatment efficacy varies significantly between patients. While there are signs of progress in implementation, there are also a number of challenges, such as interpreting genetic variants, the variation in the frequency of genetic variants across populations, access to genomic testing and the translation of molecular findings into routine prescribing. Personalised medicine, thus, should not be viewed as a replacement of the traditional approach to clinical assessment, but as a combination of genomic data with the well-established principles of pharmacology (Cecchin & Stocco, 2020; Suliman, 2025).

The growing evidence of the relationship of genetic variation to therapeutic effectiveness, adverse reactions and personalized therapy has created a need for an integrated evaluation linking molecular mechanisms to clinical pharmacology and personalized therapy. In addition to its significance for comprehending the influence of genetic variability on drug response, this synthesis is crucial for the translation of genomic technologies into safer and more effective therapy (Bhatt et al., 2025; Alzahrani et al., 2024). This all-inclusive review intends to summarize the latest findings regarding the molecular and genetic basis of drug response and toxicity, and their use in precision medicine and personalized therapeutic interventions based on pharmacogenomics.

2. Methodology

This study employed a comprehensive review approach to explore the contribution of pharmacogenomics to precision medicine and highlighted the molecular and genetic basis of drug response, drug toxicity and targeted treatment. The relevant literature was retrieved from peer-reviewed scientific journals, academic books, and other trustworthy scientific sources and was prioritized by recent publications (mainly within the last decade) and selected classic studies as the theoretical basis of pharmacogenomics. Google Scholar, Scopus and Science Direct were used to perform literature search using the following terms: pharmacogenomics, precision medicine, genetic variation, drug response, drug metabolism, drug transporters, pharmacogenetic biomarkers, adverse drug reactions, drug toxicity, personalized therapy. Sources were selected according to their relevance to genetic determinants of pharmacokinetics and pharmacodynamics, molecular mechanisms influencing therapeutic efficacy and toxicity, pharmacogenomic biomarkers, and the clinical application of genotype-guided treatment. The selected literature was then organized into seven major thematic areas, including genetic variability in drug response, drug metabolism and transport, pharmacodynamic determinants of drug efficacy, genetic factors associated with drug toxicity, pharmacogenomics-guided personalized therapy, genomic technologies and clinical implementation, and current challenges and future directions.

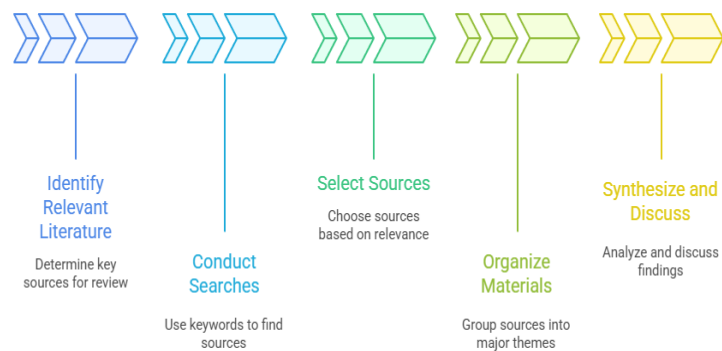


Figure 1. Overview of the Comprehensive Review Process

This study used an extensive review process that is summarized in Figure 1. The five steps of this process were to identify relevant literature, conduct targeted searches in selected scientific databases, select suitable scholarly sources, structure the selected literature into larger pharmacogenomic themes and synthesize and interpret the available evidence. This new framework allowed for an in-depth assessment of existing knowledge on the genetic and molecular causes of drug response and drug toxicity variability, as well as for incorporating this evidence with the new applications of pharmacogenomics in the field of precision medicine and precision therapeutic decisions.

3. Results

3.1 Genetic Variability and Molecular Basis of Interindividual Drug Response

All literature reviewed consistently demonstrated that there was significant inherited genetic variation that led to differences in drug efficacy, metabolism and the susceptibility to adverse effects. Pharmacogenomics connects the variations in an individual's genetic profile with the differences in therapeutic response, and offers a molecular basis for the shift from population-based prescribing to personalized treatment. Patients with the same drug and dosage can have different responses to the same drug due to variants that affect the expression or function of the genes involved in drug disposition and drug action (Weinshilboum & Wang, 2017; Qahwaji et al., 2024). Genetic variability has been studied and documented in various ways, and the forms and consequences of genetic variability found in the literature reviewed are summarized in Table 1.

Table 1. Genetic Factors Contributing to Interindividual Variability in Drug Response

Genetic factor	Molecular consequence	Potential influence on drug response	Precision-medicine relevance	Supporting references
Single-nucleotide variation	May alter protein structure, expression, or activity	Differences in efficacy, metabolism, or toxicity	Supports identification of patient-specific therapeutic responses	Weinshilboum & Wang, 2017; Qahwaji et al., 2024
Insertions and deletions	Can modify gene structure or functional protein production	Altered biological or pharmacological response	May contribute to atypical drug-response phenotypes	Qahwaji et al., 2024
Copy-number variation	Alters gene dosage and potentially protein activity	May increase or decrease metabolic capacity	Important for interpreting individual response differences	Weinshilboum & Wang, 2017



Combined genetic variants	Multiple variants may influence the same therapeutic pathway	Complex variability in treatment response	Encourages multigene rather than isolated-gene interpretation	Dash et al., 2024
Population-related genetic diversity	Differences in variant frequencies across populations	Variation in the prevalence of pharmacogenomic phenotypes	Highlights the need for population-sensitive interpretation	Getahun et al., 2024; Alessandrini et al., 2016

The pharmacogenomic variability is not a one-dimensional variation of genetic changes as illustrated in Table 1. Rather, there may be several genetic factors that together determine the response to therapy, highlighting the importance of considering the genotype, in combination with clinical and biological features, to interpret the response to therapy (Dash et al., 2024).

3.2 Pharmacogenomics of Drug Metabolism and Transport

Some of the most well-studied factors involved in pharmacogenomics are drug-metabolizing enzymes and membrane transporters. Variability in metabolic enzymes due to genetic differences can alter the activation, deactivation, and/or clearance of drugs and may alter systemic exposure and/or therapeutic effect. Thus, the efficiency of the main metabolic pathways may be affected, leading to decreased efficiency at one end, or high levels of exposure and toxicity at the other. These observations have made pharmacokinetic genes particularly important in precision-medicine research (Ahmed et al., 2016). The overall relationship between genetic variation, drug-metabolizing enzymes, transport processes, and therapeutic response is illustrated in Figure 2.

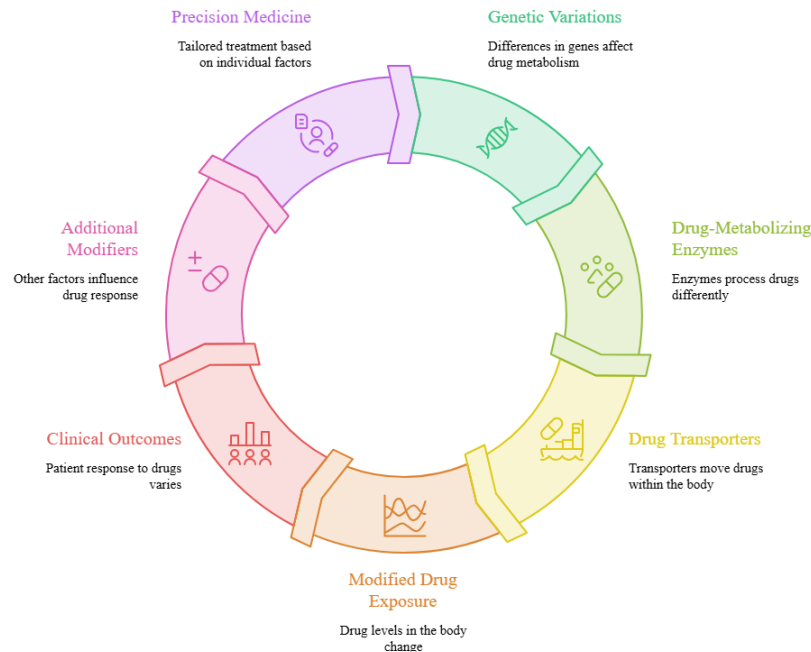


Figure 2. Pharmacogenomic Influence on Drug Metabolism, Transport, Drug Exposure, and Clinical Response

As shown in Figure 2, genetic variation can influence both drug metabolism and transport, thereby modifying drug exposure and contributing to differences in clinical outcomes. An extra layer of variation is provided by drug transporters, which affect the passage of therapeutic molecules across intestinal, hepatic, renal, and cellular membranes. A genetic variation that increases or decreases the expression or function of transporters could therefore alter drug absorption, tissue



distribution, cellular levels, or clearance. Importantly, metabolism and transport do not act alone; they can vary together to define the overall pharmacokinetic profile of a medicine (Ahmed et al., 2016). The literature also shows that genetically predicted metabolism could be influenced by other factors. Drug response depends on interactions between host genetics, disease status, co-medications, environmental factors, and other biological processes. In addition, pharmacogenomic studies investigating the microbiome have proposed that microbial metabolism may interact with genetic determinants in the host and contribute to interindividual variability in drug response (Hassan et al., 2021). Therefore, genetic interpretation is best viewed within a broader biological context rather than as a standalone determinant.

3.3 Pharmacodynamic Genes and Molecular Determinants of Drug Efficacy

Pharmacodynamic pharmacogenomics deals with pharmacogenomic variations in drug targets and molecular pathways responsible for drug effects. Patients may have different sensitivities to treatment even if they are exposed to the same dose of a drug because there are changes in the receptor, enzymes, signalling molecules and disease-related molecular targets. Precision medicine goes beyond metabolic variability, and also takes into account molecular markers that can help pinpoint therapeutic agents that will be most effective (Naithani et al., 2021; Singh, 2019). Table 2 summarizes the major pharmacodynamic mechanisms found in the literature reviewed.

Table 2. Molecular and Pharmacodynamic Determinants of Drug Efficacy

Determinant	Biological consequence	Influence on therapeutic response	Relevance to personalized therapy	Supporting references
Drug-target variation	Changes target structure or functional activity	May increase or decrease sensitivity to therapy	Helps identify patients more likely to benefit from specific drugs	Singh, 2019; Qahwaji et al., 2024
Receptor-related variation	Alters receptor expression or signaling	Produces differences in pharmacological response	May support treatment selection based on molecular characteristics	Naithani et al., 2021
Signaling-pathway variation	Modifies downstream cellular responses	Can alter therapeutic efficacy despite adequate drug exposure	Supports pathway-oriented precision treatment	Singh, 2019
Somatic molecular alterations	Creates disease-specific biological differences	May determine sensitivity or resistance to targeted treatments	Particularly relevant to precision oncology	Nafchi et al., 2025
Germline genetic variation	Influences inherited components of drug action	Contributes to patient-specific efficacy profiles	Can guide individualized therapeutic decisions	Weinshilboum & Wang, 2017
Resistance-associated molecular changes	Reduces sensitivity to treatment	May lead to inadequate clinical response	Supports alternative or modified therapeutic strategies	Nafchi et al., 2025



As summarized in Table 2, pharmacodynamic variation provides an important explanation for differences in treatment effectiveness that cannot be accounted for solely by drug concentration.

3.4 Genetic Determinants of Drug Toxicity and Adverse Drug Reactions

Adverse drug reactions represent one of the most clinically important applications of pharmacogenomics. Genetic differences can increase the risk of drug toxicity by altering drug metabolism, reducing elimination, increasing exposure to active metabolites, changing tissue sensitivity, or affecting immune recognition of a drug or its metabolites. Pharmacogenetic testing can therefore help identify individuals who may require dose adjustment, closer monitoring, use of an alternative therapy, or complete avoidance of a particular medication (Micaglio et al., 2021). The major genetic and clinical pathways linking pharmacogenomic variation with toxicity risk and individualized risk management are illustrated in Figure 3.

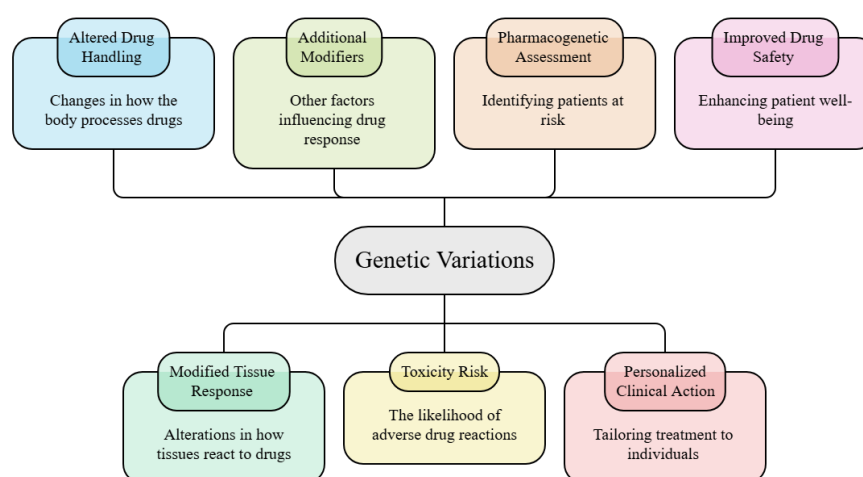


Figure 3. Genetic and Clinical Determinants of Drug Toxicity and Pharmacogenomics-Guided Risk Management

Genetic variation may affect drug handling and tissue response and there may be other patient-specific variables that will alter the total risk of toxicity (see Figure 3). Pharmacogenetic evaluation can thus aid in the identification of susceptible individuals and personalised clinical action to enhance the safety of prescribed drugs. There is also evidence that adverse drug reactions are a result of genotype–phenotype interactions. The clinical expression of genetic risk may be modified by age, comorbidities, co-medications, environmental factors and physiological features, which may result in not all individuals with a potentially relevant genetic variant developing toxicity. Thus, genotype-phenotype interaction offers a more realistic approach to investigate drug related adverse effects than a genotype-based approach (Cacabelos et al. 2021). Pharmacogenomics can also inform on the molecular pathways that lead to drug-induced injury, and identify vulnerable groups before they have a serious adverse reaction, when combined with toxico-genomics. This holistic approach can assist in earlier identification of risks, safer prescribing, and more personalized monitoring plans (Paul-Chima, 2026). Overall, further research and knowledge about pharmacogenomic testing to reduce preventable harm due to adverse drug reactions will remain a significant opportunity to enhance medication safety (Alessandrini et al., 2016).

3.5 Pharmacogenomics-Guided Personalized Therapy and Major Clinical Applications

The goal of pharmacogenomics guided therapy is to turn genetic data into real-world treatment decisions to help select an appropriate drug, adjust dosing, consider alternative treatments, or monitor more closely. The evidence reviewed suggests this is a more pertinent strategy in several therapeutic areas and is one of the most obvious examples of a precision medicine strategy in everyday drug



treatment (Ahire et al., 2026; Dash et al., 2024). The representative clinical applications are given in Table 3.

Table 3. Major Clinical Applications of Pharmacogenomics-Guided Personalized Therapy

Therapeutic area	Pharmacogenomic application	Potential treatment modification	Expected clinical value	Supporting references
Oncology	Identification of molecular factors affecting response, resistance, and toxicity	Selection or modification of anticancer therapy	Improved therapeutic targeting and reduced toxicity	Nafchi et al., 2025
Cardiovascular pharmacotherapy	Evaluation of genetically influenced variation in drug response	Adjustment of drug or dose when clinically indicated	More individualized therapeutic response	Weinshilboum & Wang, 2017; Ahire et al., 2026
Neuropsychiatric pharmacotherapy	Assessment of variability in metabolism and treatment response	Selection or adjustment of therapy	May reduce ineffective trial-and-error prescribing	Dash et al., 2024
Infectious-disease pharmacotherapy	Identification of genetic susceptibility to variable response or adverse reactions	Selection of safer or more appropriate therapy	Improved drug safety and treatment individualization	Micaglio et al., 2021
General personalized pharmacotherapy	Integration of genetic profile with clinical characteristics	Drug selection, dose modification, or monitoring	Better balance between efficacy and safety	Singh, 2019; Ahire et al., 2026
Population-specific precision medicine	Consideration of population-relevant pharmacogenomic variation	More context-specific interpretation of test results	Improved relevance for genetically diverse populations	Getahun et al., 2024

As indicated in Table 3, the main value of pharmacogenomics lies in translating molecular information into clinically actionable decisions.

3.6 Genomic Technologies, Biomarkers, and Clinical Implementation

The advancement of genomic technologies has made pharmacogenomics a broader area of study, moving past the analysis of a single pharmacogenetic candidate gene. Modern technologies can be used in a variety of ways to capture drug response variability, including through genotyping platforms, sequencing, biomarker analysis, and several types of clinical data. These technologies facilitate the identification of variants linked to metabolism, transport, therapeutic targets and adverse reactions, and enhance the molecular foundation of personalized drug therapy (Qahwaji et al., 2024). But the discovery of a biomarker does not guarantee its usefulness for clinical purposes of the clinic. If a relationship is established, it must be well validated, meaningful and actionable in order to have an impact on the care of the patient. The integration of genomic information with electronic clinical



systems and decision-support tools could ease the way for using pharmacogenomic information at the time of treatment selection. The vision of precision medicine also focuses on using molecular data together with other patient features; it is not just on a single biomarker alone (Naithani et al., 2021). There is also a growing interest in using a computational method for dealing with pharmacogenomic information, which is generally becoming more complex. AI has been suggested for the discovery of patterns in vast sets of genomic information, integration of several predictors, biomarker discovery, and therapeutic decision-making. However, they need to be carefully validated, interpreted, and have good data quality as well as protection against false or poorly generalizable predictions before they can be clinically implemented (Taherdoost & Ghofrani, 2024). Therefore, genomic technologies are best applied in the context of, not in lieu of, clinically-informed therapeutic decisions.

3.7 Challenges, Evidence Gaps, and Future Directions

Pharmacogenomics has shown great potential but there are still several scientific, clinical, technological and population level challenges in achieving universal use. Prominent issues are the lack of global population representation, the variability in evidence of the strength of different PGT markers, the challenges of uncommon variants, the discrepancies in healthcare infrastructures and the difficulties in applying sophisticated genomic data to simple drug prescribing (Alessandrini et al., 2016; Getahun et al., 2024). Table 4 summarises the main problems and future research needs reported in the various studies reviewed.

Table 4. Challenges, Evidence Gaps, and Future Priorities in Pharmacogenomic Precision Medicine

Challenge	Current implication	Evidence gap	Future priority	Supporting references
Underrepresentation of diverse populations	Some findings may have limited transferability between populations	Insufficient population-specific pharmacogenomic evidence	Expand studies across genetically diverse populations	Getahun et al., 2024; Alessandrini et al., 2016
Variable evidence among gene–drug relationships	Not all reported associations are equally clinically actionable	Need for stronger prospective and real-world validation	Prioritize clinically meaningful and reproducible associations	Weinshilboum & Wang, 2017
Complex and rare genetic variation	Difficulties in interpretation and clinical translation	Limited functional characterization of uncommon variants	Improve genomic and functional evaluation	Qahwaji et al., 2024
Cost and access to testing	Restricts routine implementation in some healthcare settings	Limited evidence on scalable implementation	Develop accessible and sustainable testing models	Alessandrini et al., 2016; Getahun et al., 2024
Integration of multiple biological determinants	Genotype alone may not explain the full	Limited integration of genomic and	Combine pharmacogenomics with clinical and	Hassan et al., 2021



	drug-response phenotype	other biological data	biological information	
Interpretation of complex datasets	Growing genomic data may be difficult to use clinically	Need for validated analytical approaches	Develop transparent and clinically tested computational support	Taherdoost & Ghofrani, 2024
Prevention of drug-induced toxicity	Genetic risk is not uniformly incorporated into prescribing	Need for broader validation and implementation	Integrate pharmacogenomics with toxicogenomic risk assessment	Paul-Chima, 2026; Micaglio et al., 2021
Translation into routine clinical care	Molecular findings may not consistently alter treatment decisions	Implementation and workflow barriers	Strengthen clinically actionable guidance and healthcare integration	Dash et al., 2024; Ahire et al., 2026

As presented in Table 4, the future development of pharmacogenomics will require more than increasing the volume of genomic information.

4. Discussion

The results of this review suggest that pharmacogenomics has emerged as a central tenet of precision medicine, and is an important factor to consider when understanding individual patient responses to the same therapeutic intervention. But the evidence also demonstrates that precision treatment isn't genomics alone. Drug response is influenced by genetic, physiological, environmental and clinical variables and the impact of pharmacogenomics on clinical practice relies on the ability to combine genetic information with the optimization of the drug dose and other patient factors. Peck (2018) pointed to this wider context by asserting that besides utilizing genomic classification, individualized therapy should also take into account the right dose for the right patient. This is in line with the present review where both pharmacokinetic and pharmacodynamic variability were identified as significant factors affecting efficacy and toxicity.

One of the key findings from the evidence reviewed is that genomic strategies have the potential to enhance patient characterization and the understanding of disease mechanisms, but the application of these strategies in clinical practice will require effective interpretation and translation into clinical decisions. With the development of sequencing and genomic profiling, several variants can now be assessed that are known to affect metabolism, drug transport, targets, or resistance mechanisms. Latif et al. (2026) highlighted the expanding applications of genomic approaches in precision medicine, such as leveraging genomic variation to inform tailored treatment strategies. In parallel, there has been a growing interest in multi-omics techniques in addition to genomics due to the absence of a single molecular layer that determines therapeutic response. In this light, Lakshmi et al. (2025) noted the emerging prospect of pharmacogenomics being combined with multi-omics and novel drug-development strategies, stating that the future of precision therapeutics might rely on a fusion of biological information, instead of a single genetic signature.

One of the most critical challenges of translating pharmacogenomics into everyday clinical practice. While large genomic datasets can uncover clinically relevant associations, they also present challenges for interpretation, validation, data management, and decision making. Additionally, pharmacogenomics plays a vital role in precision medicine, and there are hurdles to overcome in the era of Big Data, as well as a need to translate molecular data into actionable clinical advice (Primorac



et al., 2020). This topic is present in this current review, especially in the part of clinical implementation and genomic technologies. A genetic association must be linked to a specific treatment intervention (e.g. dosage adjustment, change in therapy, extra surveillance, or exclusion of a high-risk drug) to be considered as a basis for pharmacogenetics.

Pharmacogenomics has clinical significance particularly in therapeutic areas where there is high molecular heterogeneity that affects therapeutic response. Inherited and tumor-specific genetic differences in oncology demonstrate one of the most elegant examples, as they can impact drug sensitivity, resistance, and/or toxicity. In their article, Jeibouei et al. (2019) addressed pharmacogenomic strategies in personalized breast cancer treatment, highlighting the potential of molecular data to inform treatment decisions and personalization. The same line of thinking is being followed in cardiovascular pharmacotherapy, where genetic polymorphisms could affect treatment outcomes to widely-used medications and enable more effective prescription of treatment. Ingelman-Sundberg and Pirmohamed (2024) reviewed how pharmacogenetic analysis can be used prior to cardiovascular drug treatment and discussed its potential to enhance the precision of therapy. The examples illustrate that pharmacogenomics is not just a theoretical molecular profiling but can be applied practically as well.

Another key finding is that of pharmacogenomics in the minimisation of adverse drug reactions. Genetic variation in drug exposure, immune responsiveness or tissue sensitivity can result in clinically significant differences in drug toxicity. Identifying patients who are at risk prior to treatment can thus enhance medication safety and minimize unnecessary harm. Pharmacogenomic data can help optimize drug therapy and reduce the likelihood of adverse effects, highlighting its role in enhancing the safety and effectiveness of medication treatment, as Paswan et al. (2025) noted. This is in keeping with the present review, which found that the toxicity-related pharmacogenomic markers is one of the most clinically important areas of interest. However, genetic risk should be considered with the presence of comorbidities, co-administered medications, age and other non-genetic factors, as adverse reactions are often more complex in nature than just genotype-specific.

Pharmacogenomics has also undergone an evolution in history, from noting differences in drug response as a result of inheritance to creating clinically applicable precision-medicine strategies. Paul-Chima et al. introduced the reader to the evolution of pharmacogenetics principles to the present personalized applications and the challenges for the future. Likewise, Freeman (2023) highlighted the role of pharmacogenomics as a growing pathway towards precision medicine and its potential impact on personalized treatment decisions. The overall message of these views is that the field is evolving from association-based research to implementation-based pharmacology.

The potential and the challenge of this transformation can be seen in the field of psychiatry. Predicting drug response in psychiatric treatment is more challenging than some single-gene pharmacogenomic examples because it is affected by several genes, environmental factors, disease heterogeneity and clinical context. Pardiñas et al. (2021) reviewed the potential of pharmacogenomics in psychiatry and emphasized the necessity of more precise evidence in order to be broadly used in clinical practice. This highlights an important issue that came out of the present review, which is that the evidence of pharmacogenomics is not equally mature in all therapeutic areas.

In summary, the data indicate that pharmacogenomics has the potential to enhance pharmacovigilance, increase the rate of population-level drug utilization, increase the efficiency of drug use, and decrease the risk for adverse drug events when properly developed, studied, tested, implemented, and supported by clinical decision making tools and consideration of non-genetic factors. Future studies should also explore models which are multi-dimensional and integrate genomics, multi-omics, clinical information, and therapeutic monitoring, rather than individual gene–drug relationships. This would offer a more realistic basis for precision pharmacotherapy and enhance the implementation of molecular advances into personalized patient care.

5. Conclusion

The advent of pharmacogenomics as a part of precision medicine has created a molecular basis to



account for inter-individual variation in drug response, therapeutic activity, and drug toxicity. Many of the pharmacokinetic and pharmacodynamic effects may be greatly affected by genetic variation in these pathways, including drug metabolizing enzymes, transporters, receptors, molecular targets, and signaling pathways. These variations account in part for the fact that a common therapeutic strategy might have different effects and safety in different individuals. The increasing number of genomic technologies has helped to identify clinically relevant biomarkers and facilitated the development of more personalized treatment approaches. Pharmacogenomics guided treatment can be especially useful in increasing clinical success in drug selection, dosage optimization, prevention of adverse drug reactions, and identification of a possible higher risk of treatment failure or toxicity. It's becoming more and more important in oncology, cardiovascular diseases, psychiatry, infectious diseases and other therapeutic fields. However, the widespread clinical use is hindered by a lack of population representation, accessibility of genetic testing, difficulty in understanding the highly variable genetic variants, cost and integration with the health care system. There is a need for more clinical validation, inclusion of genetically diverse populations, and incorporation of other biological and clinical factors in the future. In general, pharmacogenomics provides a promising base for safer, more effective and increasingly individualised pharmacotherapy.

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