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Role of Pharmacogenomics in Personalized Medicine: A New Frontier in Drug Therapy Optimization

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ABSTRACT

Pharmacogenomics integrates pharmacology and genomics to study how genetic variations influence individual responses to medications. Incorporating genetic data into clinical decision-making allows more precise drug selection and dosing, improving effectiveness, reducing adverse effects, and promoting truly individualized therapy. This review outlines the development and clinical importance of pharmacogenomics and highlights its application in drugs such as warfarin and clopidogrel, where gene variants including CYP2C9, VKORC1, and CYP2C19 significantly affect metabolism and treatment outcomes. Genetic testing helps determine appropriate dosing to achieve optimal anticoagulant and antiplatelet effects. The review also emphasizes pharmacogenomics as a foundation of precision medicine, enabling clinicians to understand how genes influence drug metabolism, transport, and targets. Clinical pharmacists play a key role through patient counselling, dose adjustments, and integrating genotype-guided therapy into practice. Despite its promise, challenges such as high testing costs, limited awareness, and concerns about privacy and consent continue to hinder widespread adoption. As genomic technologies, bioinformatics, and digital tools advance, pharmacogenomic data are becoming easier to apply in routine care. Ultimately, pharmacogenomics is set to reshape modern therapeutics by shifting drug therapy toward a more predictive, preventive, and patient-centred approach.

Keywords: ***Pharmacogenomics, Drug Therapy Optimization, Warfarin, Clopidogrel, Pharmacogenomic Testing, Clinical Pharmacists.***

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Introduction

Pharmacogenomics is simply a study that examines the relationship between our genes and our reaction towards medications. During my class in biology, we found out that it is a mash-up of pharmacology, which is the study of drugs and genomics, which is the study of genes (1).The entire concept actually creates hope owing to the fact that we may be raised with genetically distinct treatments. Genes are not the only factor thatdetermines the drug response age, diet, lifestyle, the environment and the state of health are of course also significant factors, yet the genetic factor is enormous when it comes to the safety and effectiveness of the drug (2). The way a person responds to a drug, be it good or side effects, is a rather complex

characteristic that is the upper product of numerous genes (3).That began to change when we found single-nucleotide polymorphisms or SNPs, little variations in the bases of the DNA that are appearing everywhere within the genome (4).The human genome contains approximately 11 million of these SNPs, and this entails that somewhere in 1,300 base pairs, one cell in 1,300 was susceptible (5). Pharmacogenomics is a convergence of pharmaceutical science that is old, such as pharmacology and biochemistry, and genomics, where genes, proteins, and SNPs are considered (6). Through the union of these disciplines, it is driving the production of safer, more precise, and more effective drugs, which are individualised to the genetic makeup of the patient, which will bring us an enormous step nearer to the matter of individualised medicine (7).

Pharmacogenomics can also be traced back to the findings of the early 20th century that indicated some people appeared to respond differently to drugs (8). At that time, scientists observed that some groups of people respond differently, and that left people with an idea of using genetics as an influencing factor (9). An example is the reported increases in liver isoenzyme UDP-glucanosyltransferase in Finnish men when linked to alcohol and methemoglobinemia in people following consumption of water laced with nitrates in Gozo (10). Other observations included the difference in antibody production as well as drug metabolism, as seen in the variation in the incidence of isoniazid-induced adverse reactions predictable by the polymorph nuclear cell counts (11). Important phenomena of pharmacogenetics were discovered by the middle of the 20th century. There were also the haemolytic reactions to primaquine in the patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency, long apnoea of the patient with the deficiency of pseudocholinesterase when this is administered together with succinylcholine and peripheral neuropathy developed in the patient during the treatment with isoniazid (12). In 2001, with the completion of the Human Genome Project, there was an available catalogue of genetic data, unlike before, which saw the emergence of pharmacogenomics as a data-driven field (13). The discovery of genetic mutation and its association with adverse effects caused by antineoplastic agents like 6-mercaptopurine, methotrexate, and 5-fluorouracil allowed linking pharmacogenomics and oncology directly (14). These were further findings that supported the hypothesis that genetic variation is directly associated with the drug efficacy and safety. Recent breakthroughs in the fields of genotyping, sequencing technologies, and bioinformatics have made it possible to identify systematically single-nucleotide polymorphisms (SNPs) along with other variants that influence drug metabolism and response (15). Pharmacogenomics is an important interdisciplinary link between traditional and innovative genomics today and provides the scientific basis of personalised medicine (16). Since the dawn of anecdotal evidence tracking, through onward, to the development of the most technologically advanced programmes, the history of the field shows its increased involvement in the personalisation of treatments based on the individual genetic profile (17).

2. Concept and Scope of Pharmacogenomics

Pharmacogenomics refers to a multidisciplinary science that combines pharmacology and genomics to comprehend the influence of genetic differences on the susceptibility of a person to different medicines. Fundamentally, it tries to state why, when drugs are taken in the same dosage, they may have extremely disparate treatment or side effects on different patients. Whereas the conventional medicine system utilises the trial and error method to dictate the kind of treatments that are successful, pharmacogenomics offers a scientific platform on which therapies may be created in a safer, more efficient, and tailored to the genetic composition of the individual (18).

2.1 Concept of Pharmacogenomics

Pharmacogenomics is a combination of two massive disciplines, that of pharmacology, where we get to learn everything about drugs and their effect and that of genomics, which is the general study of genetic variation and the entire genome (19). And, my professor tells me, it is broader than in the case of pharmacogenetics, as it does not just examine the effects of individual genes on drug response, but also plunges into the football disarray of the interaction between two or more genes, epigenetics, and control specifications (20). Drug response is highly complex- it is a complex characteristic, multi-layered by the pharmacokinetics and pharmacodynamics (21). The two dimensions intermix and affect one another in a manner that is difficult to separate. Those two pharmacodynamic or pharmacokinetic processes can be perturbed by genetic variations such as SNPs, duplications or deletions of a gene or by modifications in protein expression (22). You could have an example of a mutation in an enzyme that partially degrades a drug, thus prolonging its longevity or a polymorphism in a receptor that predisposes you to be more sensitive to a drug. When we put all those differences into the equation, pharmacogenomics can, in fact, point out which patients will respond well, who will require an increase or a lower dose, and who may risk

some awful side effects. It is a change of game that is moving us away from the history of prescribing through the concept of a one-size-fits-all model to a place of a more individualised approach (23).

2.2 Scope of Pharmacogenomics

Pharmacogenomics has a broad spectrum of application throughout the entire process of drug therapy, including discovery and development, to clinical use and treatment outcomes at long term. It is concerned with a study of the impact of genetic variations in drug absorption, distribution, metabolism, excretion and interaction with the target, which make it possible to more precisely predict therapeutic effects and adverse drug reactions. Pharmacogenomics is applicable in clinical practice to promote the field of personalized medicine, helping to determine drug choice, dose, and drug monitoring across a broad range of therapeutic indications, such as cardiology, oncology, psychiatry, infectious diseases, and pain management (24). Its application goes further to determine genetic indicators of drug toxicity, maximize polypharmacy in the complex patient and enhance treatment safety in the vulnerable patients. In addition to clinical care, pharmacogenomics also guides pharmaceutical research by allowing the development of genotype-specific drugs, stratification of clinical trial patients, and decreasing the cost of clinical trial failure or adverse events. With the improvement of genomic technologies and the development of pharmacogenomic databases, this area is gradually becoming more and more pre-emptive, electronic and integrated into electronic health records, and is becoming commonplace in medical facilities, which is a step toward the realization of truly individualized and predictive medicine (25).

Table 1 provides the important drug-gene interactions that affect drug metabolism, efficacy, and safety and how certain genetic variations can be used to inform dosage adjustments and choice of therapy in personalized medicine.

Table 1. Key Pharmacogenomic Drug–Gene Associations

Drug	Gene	Variant	Effect	Clinical Impact	Reference
Warfarin	*CYP2C9*, *VKORC1*	*CYP2C9 2*, *3*; *VKORC1* −1639 G>A	Reduced metabolism, increased sensitivity	Lower dose requirement	(26)
Clopidogrel	CYP2C19	*2*, *3* (LOF); *17* (GOF)	LOF → reduced activation; GOF → ↑ bleeding risk	Alternative therapy (prasugrel/ticagrelor)	(27)
Statins	SLCO1B1	c.521T>C (*5*)	Reduced hepatic uptake	↑ Myopathy risk	(28)
Codeine	CYP2D6	*3*, *4*, *5* (LOF); *1xN* (GOF)	Poor → ineffective; Ultra-rapid → toxic	Avoid in PM/UM patients	(29)
Thiopurines	*TPMT*, *NUDT15*	*TPMT 2*, *3A*, *3C*	Decreased enzyme activity	Dose reduction required	(30)

2.2.1 Optimisation of Drug Therapy

Basically, pharmacogenomics prepares the way for individualising drug regimens as per the genetic constitution of the respective person. Variations in genes that encode cytochrome P450 enzymes illustrate the reason why others metabolise meds faster than others, and you have to have variations in dose plans (31). Pharmacogenomic biomarkers can be relevant in the treatment of cancer in assisting the choice of the targeted therapy. They can indicate patients who are more likely to respond to drugs such as fluoropyridine drugs or platinum-based agents due to the acquisition of certain genetic markers (32). Thus, with the genetic profile typed in, treatment becomes more streamlined with clinicians being able to select and select the drug that will suit you best, whether they have side effects or not (33).

2.2.2 Reduction of Adverse Drug Reactions

One of the principal causes of illness and death is the ADRs all over the world. One approach that can be used to overcome this is pharmacogenomics, which is used to predict the possibility of drug-induced

toxicity in individual people, even during treatment. Indicatively, the risk of thiopurine drugs, which are life-threatening and severe in patients with TPMT deficiency, is high, but a straightforward genetic test is capable of detecting these patients in time (34).

Drug Discovery and Development

Pharmacogenomics is, therefore, a kind of super cool, which accelerates the process of drug discovery, right? It allows scientists to identify new drug targets and pathways with the help of genomic information (19). Furthermore, it is utilised by pharma companies to create more narrow clinical trials by filtering study participants in accordance with their genetic enhancements, which helps to increase the efficiency of a trial and reduce the cost (35). Pharmacogenomics has a general role as an area that is applied across therapeutic fields. Within the field of cancer, gene-expression profiling can be used to forecast the response of individuals to chemotherapy (36). The genetics of drug metabolism were better understood in cardiovascular disease, as the variation in the response to antiplatelet agents or beta-blockers was elucidated in patients. In psychiatry learning, pharmacogenomic testing can be used to explain why not everyone reacts to antidepressants and antipsychotics or continues to experience the side effects (37).

Pharmacogenomics is something much bigger than simply prescribing drugs because it is an approach that drives us towards the willingness to call it a Precision Medicine era where not only are drugs prescribed in a personalised way to the patient, but where the identification of disease, diagnosis and therapy too is based on the genetic makeup of individual patients. This is what health science is striving to achieve in general: to extract as many benefits as possible and reduce the number of risks (38).

3. Future Outlook

Pharmacogenomics has yet to realise its massive potential fully, but even as a form of clinical integration is still in its initial stages. We are still struggling with such issues as the excessive price, regulatory challenges, ensuring that the genetic tests, in fact, are reliable and all the ethical considerations of privacy and patient consent issues (39). Nevertheless, high-throughput sequencing, bioinformatics and systems biology are progressing at a very fast rate, and thus the field is currently rapidly striving to ensure that pharmacogenomics is becoming a staple as far as the future of drug therapy optimisation is concerned (40).

4. Pharmacogenomics V/S Pharmacogenetics

Though the terms pharmacogenomics and pharmacogenetics are used interchangeably in the literature, both refer to two similar but distinct styles of addressing the greater arena of a personalised medicine perspective. Both touch on the genetic variation and how it affects drug response, though at varying levels, approaches and also in a clinical setting (41).

4.1 Pharmacogenetics

Pharmacogenetics is the more ancient one, dating back to the mid-20th century. The introductory research revealed that single-gene differences may alter the drug metabolism levels and the type of treatment results you will receive (42). Classic examples include G6PD deficiency, which predisposes individuals to haemolytic responses when they use antimalarials such as primaquine and mutant butyrylcholinesterase, resulting in long-term apnoea following succinylcholine. In essence, pharmacogenetics narrows down the connection existing between a certain gene and the transference to a certain drug reaction, which is incredibly handy in clarifying hereditary side effects or peculiar therapeutic responses in specific patient groups (43).

4.2 Pharmacogenomics

Genomics Pharmacogenomics, on its part, is a somewhat broader and smoother area that does not merely examine a single gene. It is the one that wants to get into the entirety of the genome to understand how it influences the mechanism of drug action, such as their efficacy, safety and even those weird side effects (1). Adding in high-throughput sequencing, bioinformatics, transcriptomics and proteomics, pharmacogenomics examines how thousands of genes interact with our environment through much of what pharmacokinetics, absorption, distribution, metabolism, and elimination and pharmacodynamics that is basically how drugs hit their targets (44). Considering cancer research as an example, pharmacogenomic research studies would be able to simultaneously screen many genetic markers to aid in determining the most optimal chemo or targeted therapy, which ultimately refines the treatment to precision (45).

5. Genetic Factors Influencing Drug Response

One of the largest challenges in the field of clinical pharmacology is to accurately predict the way an individual is going to process and react to a drug. Although pharmacogenomics has reached the point of maturity, understanding that science and converting it into routine therapy is but a fledgling theory (1). The combination of both genetic and non-genetic factors determines the response of the drug in a patient, and it will cause huge variations in both pharmacokinetics (absorption, distribution, metabolism, and elimination) and the pharmacodynamics (19). This is of particular importance to drugs of a low therapeutic index, where minute alterations in dose administered could spell the difference between a cure, a side effect or failure itself. One of the most valuable genetic actors is the cytochrome P450 (CYP) enzymes (46). Oxidising most prescription drugs, many internal chemicals and environmental toxins are primarily oxidised by the CYP superfamily. CYP enzymes are of particular interest because of their central role as the determinants of either drug efficacy or drug safety (47). The human genome is composed of 57 CYP genes and 58 pseudogenes majority of them being grouped into autosomes. In mice, this now increases to 108 CYP genes and 88 pseudogenes. The human CYPs have been sorted into 18 families and 44 subfamilies according to sequence homology (48). The presence of polymorphisms in these genes can have an enormous impact on the speed of drug clearance, their effectiveness and the chances of adverse reactions. An example is individuals who carry defective CYP2D6 alleles are poor metabolizers and can have less benefit or excessive toxicity of antidepressants, beta-blockers, or opioids (49). On the contrary, the so-called ultra-rapid metabolizers may require significantly higher doses of the same drug to achieve the same effect. Other genetic factors are also important, other than CYP enzymes. The presence of variants in drug transporters, such as the P -P-glycoprotein that is coded by ABCB1, can alter the levels of absorption and distribution of the drug (50). The action of a drug can be tuned by the polymorphisms in the receptors and ion channels. Alteration of the conjugation activity of enzymes (UGT1A1 or NAT2) has been associated with increased metabolism of chemotherapy agents and increased toxicity risk (51).Concisely, genetic variation forms one of the major factors behind individual responses to drugs. Although diet, lifestyle and other health complications also contribute greatly, the current developments in pharmacogenomics continue to demonstrate how the inherited variations in genes related to the metabolism of enzymes, transporters and targets determine the response to treatment. An understanding of these differences is critical to therapy personalisation, reduction of adverse drug reactions, and advancement of personal medicine (52).

Figure 1 shows the relative contribution of key CYP450 enzymes to drug metabolism and highlights how genetic polymorphisms, age, sex, and inflammation influence their activity, underscoring their importance in personalized drug therapy.

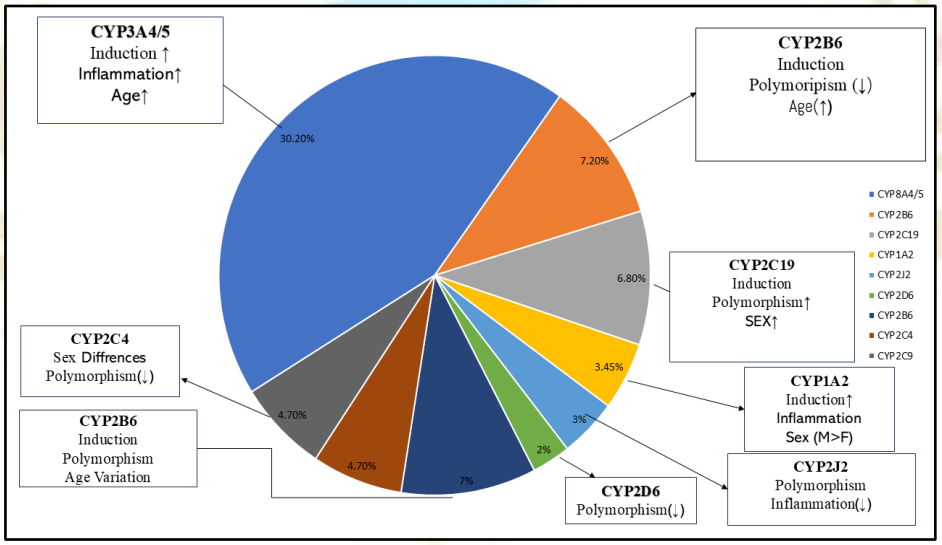

Figure 1: Distribution of CYP450 enzymes and key factors influencing their variability in drug metabolism.

Relative contributions of major hepatic cytochrome P450 isoforms to overall drug metabolism, highlighting the predominant role of CYP3A4/5. Influencing factors for each isoform such as induction, inflammation, genetic polymorphisms, age, and sex are summarized around the chart.

6. Clinical Applications

Pharmacogenomics is completely revolutionary stuff in the current medicine, whereby we get to customise drug treatment to the genetic composition of an individual (53). As I have observed both at school and in my studies, this method actually increases drug effectiveness, reduces adverse effects, and assists us in striking

6.1 Warfarin

The old oral anticoagulant warfarin is still recommended by doctors to prevent and treat the issue of deep vein thrombosis, pulmonary embolism, atrial fibrillation, and prosthetic heart valve thrombosis (55). I have read figures showing that more than 25 million prescriptions are prepared annually in the U.S. and in the entire world, and it is taken by approximately 0.51.5% of the population. It is a racemic mixture of (R) - and (S)-warfarin, cut chemically, and the (S)-compound is three to five times more powerful as an anticoagulant. Warfarin acts as an anticoagulant by disrupting the vitamin K epoxide reductase complex (VKORC1) which is the enzyme primarily involved in the recycling of the oxidised vitamin K to its reducing form. This inhibition of that enzyme disrupts the vitamin K cycle required to γ-carboxylate clotting factor II, VII, IX, X, as well as clotting factor proteins C and S, thus leaving the body unable to form fully active clotting factors, rendering the blood less apt to clot (56). Although it is extremely handy, warfarin has a limited therapeutic index and vast interindividual variation in dose. Others include genetics, diet, environment and ethnicity. Asians tend to require low doses of maintenance compared to Caucasians or Africans (57). That is why great attention should be paid to pervectating therapy to maintain safety and effectiveness. Mostly focused on by the INR, therapeutic monitoring is a standardised approach to prothrombin time to determine vitamin K-dependent clotting factor activity. The normal range of an INR of individuals not taking anticoagulants is around 1, although individuals with most indications have a therapeutic range between 2.0 and 3.0(58). It is challenging to maintain the INR in that range; a lower range exposes thromboembolic risk, whereas a higher range exposes bleeding risk - the most frequent side effect of warfarin (59). Indeed, during the period in question, 2007-2009, warfarin was the cause of hospitalisation due to drugs in approximately one-third of cases in the U.S. The threat of significant bleeding is maximum at an INR beyond 4.0 and frightening above 5.0 (60). The antidote of choice for excessive anticoagulation or overdose is vitamin K. Owing to its complexity and safety concerns, especially to vulnerable groups, as was the case in Asia, clinicians tend to be conservative (61). An example is a study in Taiwan that attributed this to a fear of bleeding complications, and only 24.7% of patients with atrial fibrillation managed to get guideline-recommended anticoagulation (62).

6.2 Clopidogrel

A second-generation thienopyridine, clopidogrel, is the preferred therapy in the prevention of atherothrombotic events in patients with known acute coronary syndromes, ischemic stroke, or undergoing a PCI (63). Although it tends to be widely prescribed, many patients express having huge discrepancies in their response; that is, they have weaker platelet inhibition and thus they are more likely to be afflicted with cardiovascular complications (64). This is due to genetic polymorphisms, which are the primary sources of variability that affect the bioactivation and pharmacodynamic behaviour of clopidogrel. The drug is an inactive prodrug that requires two oxidative hepatic steps to become active (65). About 15 per cent of the clopidogrel ingested is activated, and this is greatly attributed to enzymes of the cytochrome P450, namely CYP2C19. All of these loss-of-function alleles, such as CYP2C19 681G&A>CYP2C19 636G&A, reduce the enzyme activity, thus leaving clopidogrel unreactive and platelets uninhibited (66). Carriers of these alleles are known as intermediate or poor metabolizers whose therapeutic action is blunted with increased chances of stent thrombosis or myocardial infarction. The opposite is the case for the CYP2C19*17 gain-of-function allele, which increases the enzyme activity too much, increasing the risk of bleeding due to the excessive potency of the antiplatelet effect (67). The ethnic differences are very big: the loss-of-function type of molecule has a frequency of 55-70% in the East Asians, 30 in the Caucasians and 18% in the Africans, which means that we may need to vary the doses or drug selection among groups of people (68). Variability depends on other genetic factors, including polymorphisms in ABCB1 (P-glycoprotein) and PON1 (which are involved in the final activation step); however, their clinical consistency is less convincing (69).CPIC and other expert groups suggest a genotype-based approach based on antiplatelet therapy. Patients with a CYP2C19 poor or intermediate metabolizer profile should prefer prasugrel or ticagrelor in order to achieve enough platelet inhibition(70). It has been verified through clinical trials, such as TRITON-TIMI 38 and PLATO that these replacements perform better in genetically vulnerable patients. Inclusion of pharmacogenomic tests in cardiovascular practice may enhance the results, minimise adverse

events, and enhance precision medicine (71). Yet, obstacles in common participation like restricted testing, unpredictable use of the guidelines, and economic troubles continue to plague the efforts to have the application implemented everywhere. The future perspectives include high-speed point-of-care genotyping, numerous gene risk predictors, and machine learning to prescribe a tailored antiplatelet regimen to various people cohorts (72).

Figure 2 illustrates the absorption, metabolic activation, and antiplatelet mechanism of clopidogrel. After intestinal absorption, clopidogrel undergoes hepatic biotransformation, where CYP2C19 converts it into its active metabolite while CES1 forms inactive metabolites. The active metabolite irreversibly binds to the P2Y12 receptor on platelets, inhibiting aggregation, whereas the inactive metabolite is eliminated through urine and feces. This pathway highlights how genetic variations in metabolic enzymes affect clopidogrel's therapeutic effectiveness (73).

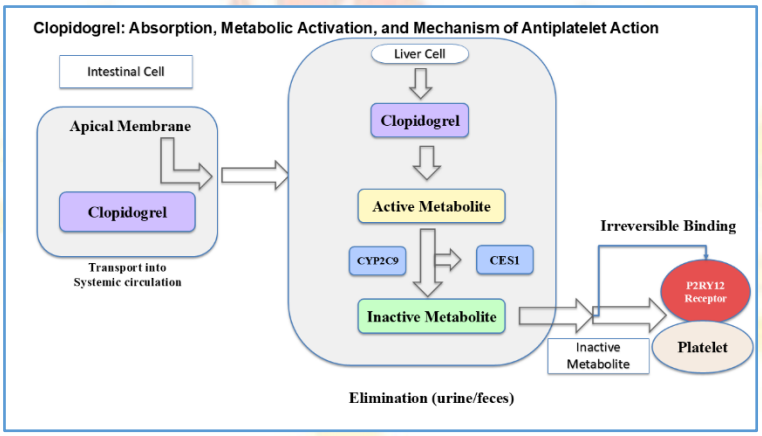

Figure 2. Clopidogrel's activation and inactivation pathways leading to irreversible P2RY12 receptor inhibition.

Metabolic activation and inactivation pathways of clopidogrel, showing CYP-mediated formation of the active metabolite that irreversibly inhibits the platelet P2RY12 receptor, while CES1 converts remaining drug into inactive metabolites.

7. Pharmacogenomic Testing

Precision medicine is principally based on pharmacogenomic testing, in which we can modify drug therapy according to the genetic profile of an individual(74). Gene differences in drug-metabolising and transporting enzymes, and receptors result in a vast difference in response, efficacy, and toxicity of drugs to individuals. With these genetic determinants determined by a specific test, we will be able to anticipate the patient response, reduce the side effects and design the specific treatment plan towards the individual molecular makeup of the patient (75). The premise of the pharmacogenomic testing is all about identifying SNPs, which modify the process of drug movement within the body and its activity. Such enzymes as CYP2C9, CYP2C19, VKORC1, and SLCO1B1 are excellent examples of clinically relevant targets(76); the enzymes mediate the metabolism of many commonly used agents, including warfarin, clopidogrel, and statins. An example is that genotyping of CYP2C9 and VKORC1 will assist with the calculation of the optimal dose of warfarin, minimise the risk of dissemination, and CYP2C19 testing identifies bad merits who can be placed on alternative antiplatelet agents (77). In the same manner, screening of the SLCO1B1 variants can identify those who are prone to statin-induced myopathy, and thus, makes lipid-lowering therapy safer. As the literature reveals, pharmacogenomic testing elucidates the cause of the different reactions of different people to drugs(78). Parkinson's disease pharmacogenomics article indicates that gene polymorphisms of COMT, DRD2, and SLC6A3 have a key impact on the efficacy as well as the toxic effect of the dopaminergic drugs. Such results resemble the ones in cardiovascular pharmacogenomics, where genetic testing can be used to optimise dosing and select medications that can be used to achieve the optimal therapeutic window (79). This interdisciplinary evidence actually demonstrates how universal principles of pharmacogenomics may be applied in medicine. Pharmacogenomic testing is increasingly becoming possible and is fast becoming a reality due to new genomic sequencing technology and bioinformatics (80). The incorporation of AI and machine learning into the equation makes predictions even more precise, and we can interpret complex interactions and reactions between genes and drugs in real time (81). Still, there are some obstacles to jump over. There is low clinical implementation, high costs, and test

standards, and doctors have yet to receive additional education regarding the use of such results. Furthermore, the allele frequency varies among ethnic groups, and hence, we require population-specific reference to ensure testing is fair and effective for all (82). We ought to begin incorporating pharmacogenomic testing into regular pre-prescription testing and integrate it into clinical decision-making support systems to achieve the greatest clinical benefit (83). Group resources such as CPIC and DPWG provide easy-to-follow, actionable recommendations to which they are linking genotype and prescribing choices. Ultimately, pharmacogenomic testing indicates a shift to one-size-fits-all prescribing to real-time and highly individualised disposal, which marks a new era of safer, more efficient, and morally accountable pharmacotherapy (84).

8. Barriers to Implementation

Although pharmacogenomics has advanced quickly and there is great clinical potential, its use in clinical practice has not been implemented at the expense of regular healthcare. There are still various obstacles to its integration in clinical practice and large-scale research projects, such as economic, educational, ethical, and regulatory barriers (85).

8.1 Economic Barriers

One of the greatest obstacles that we are currently experiencing is the high price of pharmacogenomic testing. Although the cost of sequencing technology has been drastically reducing, the entire gag of genome analysis, the bioinformatics framework to process them, and the analysis services are unaffordable in most health care systems. Low insurance cover and uneven reimbursement policies can only complicate that, particularly in low- and middle-income economies. Besides that, most data regarding the cost-effectiveness of many gene-drug combinations do not exist; thus, clinicians and institutions are cautious about jumping into the routine testing wagon (86).

1. **Table 2.** Major Barriers to Pharmacogenomics Implementation

Sr. No.	Barrier	Description	Impact on Implementation	Reference
1	High Cost of Genetic Testing	Genetic tests remain expensive in many regions	Limits accessibility and routine clinical use	(87)
2	Limited Clinician Awareness	Many healthcare providers lack training in pharmacogenomics	Reduces confidence in interpreting results and applying guidelines	(88)
3	Lack of Standardized Guidelines	Variation in clinical recommendations across institutions	Creates inconsistency in clinical practice	(89)
4	Data Privacy and Ethical Issues	Concerns about genetic data storage, consent, and misuse	Slows adoption due to legal and ethical complexities	(90)
5	Limited Integration into EHRs	Genomic data not fully incorporated into electronic health records	Hinders clinical decision support and real-time use	(91)
6	Slow Turnaround Time	Genetic testing results are not always available during initial therapy	Limits usefulness for urgent prescribing decisions	(92)

9. Ethical and Privacy Concerns

Ethical issues relating to pharmacogenomic data are mostly associated with privacy, consent and data sharing problems (93). To increase the speed of the research and the common good, the National Institutes of Health (NIH) data-sharing policy, which mandates federal-funded studies to deposit the genomic data into a common repository like the database of Genotypes and Phenotypes (dbGaP), was intended (94). There are, however, real privacy issues with this openness. The de-identified genomic data can occasionally be re-identified with the individual identities when cross-linked to the genealogical database or even in the Demographic database, just like in several high-profile studies (95).This possibility of re-identification has

led to changes in data sharing laws, which, in turn, resulted in the identification of biospecimens and genetic information as identifiable information under U.S. health privacy laws (96).

Conclusion

Pharmacogenomics is essentially a game-changer in the healthcare industry today, whereby it links the gap between genes and medication. It assists in changing our mentality of one size fits all and going to the precision medicine, where therapy is actually designed to match the genetic makeup of an individual. The warfarin/clopidogrel cases, such as that one, demonstrate that an understanding of the genetic composition of a patient can decrease the appearance of bad side effects, increase effectiveness, and precisely control the dose level. In addition, testing provides physicians with a clear indication about how a treatment may not have a much success in one individual as compared to another individual, hence making it easier to personalise care among the vast number of therapy fields. The linkage between science and the bedside is the clinical pharmacist. They interpret the outcomes the patients receive, change dosages, and collaborate with the entire care team to ensure that the data indeed contribute to better safety, adherence, and outcomes. There are still actual hitchhikes. Test cost, insufficient awareness among clinicians, as well as concerns of privacy, and sharing of data continue to make it not widely adopted by many folks. This will require powerful policies, improved training, and technological improvements to be achieved sustainably in health systems. In the future, the rapid development of genome sequencing and bioinformatics and AI are bound to accelerate the clinical application of pharmacogenomics. With the further development of the precision medicine movement, the trend towards multi-gene panels, real-time decision support, and population-specific genetic information will also enable to modify the drug prescription more. To make it short, pharmacogenomics plays an essential role in the transformation of medication plans into safer, more effective, and completely patient-centred. Get it wrong and we will have brought in an era of individualised medicine: one in which no prescription is made without genetic knowledge and where the entire system is made more predictive, preventive and precise.

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Author Contribution

D.M Data Analysis and interpretation, **M.T.** writing, reviewing and editing, **R.S.K** Visualization**, P.C.** Validation, **A.S.** Methodology.

Conflict of Interest

The authors declare no conflict of interest.

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