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Pharmacogenomics in Drug Absorption and Its Implications for Personalized Medicine

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ABSTRACT

Pharmacogenomics explores how genetic variations influence drug responses, particularly in the absorption and bioavailability of therapeutic agents. This review aims to explore the influence of genetic variations on drug absorption and its implications for personalized medicine. Drug absorption is a crucial phase in pharmacokinetics that determines the bioavailability and therapeutic effect of medications. Variations in genes encoding drug transporters, such as P-glycoprotein (P-gp) and solute carrier (SLC) proteins, can significantly affect transporter function and alter the absorption process. This review discusses the mechanisms of drug absorption, including passive diffusion, facilitated diffusion, active transport, and endocytosis-exocytosis, while highlighting the role of genetic polymorphisms, especially single-nucleotide polymorphisms (SNPs), that impact these pathways. Differences in transporter gene variants, such as in ABCB1 and SLCO1B1, may lead to variability in drug plasma concentrations, efficacy, and potential toxicity. Patients with different genotypes may exhibit distinct pharmacokinetic profiles despite receiving the same drug and dosage. The review summarizes findings from current literature that support the clinical relevance of pharmacogenomic profiling in optimizing drug therapy. Understanding these genetic influences provides a scientific foundation for individualized treatment approaches aimed at improving therapeutic outcomes and reducing adverse effects. Despite several challenges in clinical implementation, including limited awareness, infrastructure, and regulatory guidance, integrating pharmacogenomic insights into routine practice holds promise. This review concludes that advancing pharmacogenomics in drug absorption will enhance the precision of medical treatments and support the broader goals of personalized medicine.

INTRODUCTION (CHAPTER)

Pharmacogenomics is the study of how different genetic variations affect how well drugs work and how people respond to them. As our knowledge of genetics grows, pharmacogenomics opens up the possibility of more accurate and effective drug therapies that aim to improve effectiveness while lowering side effects. In this case, a key element is how genetic differences affect each step of pharmacokinetics,

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notably medication absorption, which is important for figuring out both bioavailability and clinical outcomes.

Drug absorption is the most important and basic part of pharmacokinetics, when a drug gets into the bloodstream after being taken [1], [2]. There are many things that can change this process, such as the movement of drugs through biological membranes and pre-systemic metabolism, which can be modified by genetic polymorphisms [3], [4]. Because people absorb medications differently, their treatment responses can be very different [5]. This means that we need to know the genetic factors that control these processes. These kinds of results are very important in the larger goal of getting truly personalized treatment.

This review article wants to look closely at how genetic variability affects how drugs are absorbed and what that means for personalized treatment. This will look at important genes and polymorphisms that have to do with absorption routes, review the current literature, and stress the challenges and opportunities of using pharmacogenomic knowledge in clinical practice. This review gives a complete picture of how pharmacogenomics plays a key role in making drugs that are more accurate and tailored to each person.

Drug Absorption Mechanism

The medications used do not all enter through the systemic circulation, but most medications enter the body through enteral or parenteral routes via the absorption process, which is then carried by the blood to the target organs, passing through cell membranes and reaching the target [6], [7].

Absorption is when a drug is transferred from the place of administration into the bloodstream. The absorption environment influences the rate and extent of absorption, the drug's chemical properties, and the administration method that impacts bioavailability [3], [8].

According to the chemical properties of the drug, drug absorption from the gastrointestinal tract can occur in various ways (Figure 1). Drugs move from areas of high concentration to areas of low concentration through membranes that separate two parts of the body, a process known as passive diffusion. Passive diffusion does not require a carrier, cannot be saturated, and is not structurally specific. Most drugs are absorbed through this mechanism. Water-soluble drugs cannot penetrate cell membranes through channels or aqueous pores, but lipid-soluble drugs can easily cross biological membranes due to their solubility in the lipid bilayer of the membrane [3], [6].

In addition to passive diffusion, facilitated diffusion is another mechanism by which drugs can enter cells through specific transmembrane transport proteins. The conformation of the transport protein changes in this process, allowing the drug to move from an area of high concentration to an area of low concentration. Although facilitated diffusion does not require energy, the process can become saturated due to other compounds attempting to transport it [3], [6]. For example, the organic cation transporter organic cation transporter (OCT1) helps thiamine, physiological solutions, and medications, such as metformin, which is used to treat type 2 diabetes mellitus [6].

Active transport is another mechanism by which drugs pass through cell membranes via specific transport proteins. Some drugs that resemble natural metabolites use energy from the hydrolysis of adenosine triphosphate (ATP) to transport them, allowing the drugs to move from areas of low concentration to areas of high concentration. This active transport is saturated and selective, and can be inhibited by other substances that are co-transported. the primary active transporter group, the ABC family, hydrolyzes ATP to export substrates across the membrane [3]. For example, P-gp, also known as ATP-binding cassette subfamily B member 1 (ABCB1) or MDR1, exports large neutral or cationic compounds from the cell, with its physiological substrates including steroid hormones such as testosterone and progesterone [9]. MDR1 also exports many drugs, including digoxin, and various other agents [6].

Endocytosis and exocytosis are very important cellular processes involved in the uptake of nanoparticles, drug delivery, and cell signalling [10]. Very large drugs cross the cell membrane; endocytosis and exocytosis are additional mechanisms for transporting these drugs, where the drug is engulfed by the cell membrane and transported into the cell through the formation of vesicles containing the drug, similar to the absorption of vitamin B12 across the intestinal wall [3], [11]. Exocytosis, where substances are

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expelled from the cell through a similar vesicle formation mechanism, as occurs in the release of neurotransmitters from the C nerve terminal [3].

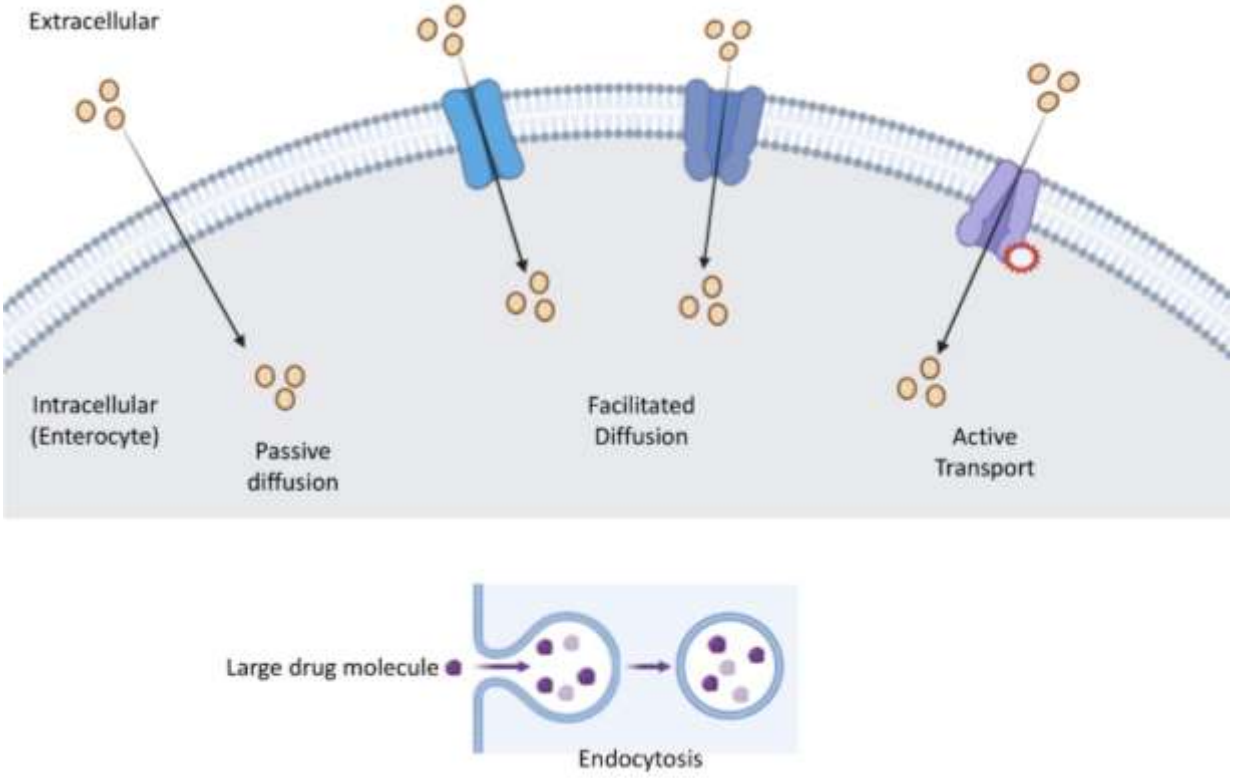

Figure 1. Drug Absorption Mechanism Illustration

The Role of Pharmacogenomics in Drug Absorption

Single-nucleotide polymorphisms (SNPs) are the most common genetic differences detected in the human genome. They happen when another replaces one nucleotide and are usually found in less than 1% of the population [10]. SNPs are slight modifications in DNA, but their presence, especially in coding areas, can have significant effects. SNPs in coding sequences are usually divided into two groups: synonymous, where the base substitution does not change the amino acid sequence of the encoded protein, and non-synonymous, where the amino acid sequence is changed, which may change the protein's structure or function [12]. Some SNPs have little or no effect, but others can cause significant changes in how drugs are absorbed and how they work, such as how well they are absorbed and how long they stay in the body [13].

Pharmacogenomics research is still helping us learn more about how differences in drug transporter genes affect how people respond to medication therapy. This field of study primarily focuses on finding SNPs in transporter genes that could change how pharmaceuticals work in the body and affect health. As sequencing technologies get better and easier to use, more polymorphisms with functional importance will likely be found. This will allow for more precise and personalized treatment plans [14].

Drug transporters are essential for pharmacokinetic absorption, distribution, and excretion [15]. These proteins in the cell membrane affect how endogenous and external substances flow through cellular barriers. When transporter performance is impaired, whether because of inherited polymorphisms, acquired deficiencies, or interactions with other compounds, therapeutic objectives may not be attained successfully, resulting in subtherapeutic plasma concentrations or, on the other hand, toxicity owing to accumulation [16]. The ATP-binding cassette (ABC) transporters and the solute carrier (SLC) transporters are the two main groups of drug transporters that are engaged in these activities [17]. These transporters have different jobs in the body. Depending on which way they point, they can act as influx or efflux transporters, helping drugs get into intestinal epithelial cells, get back into renal tubules, or go out through the urine or biliary

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systems [18]. Also, transporters in the liver, blood-brain barrier, placenta, and other selective barriers significantly affect how drugs are distributed and their availability in the body [19].

The ABC transporter family is a set of proteins that are similar in all species and are engaged in active transport processes that use ATP hydrolysis [20]. These transporters are primarily found in organs and tissues that help with absorption and disposal, like the intestines, liver, kidneys, placenta, blood-brain barrier, and blood-testis barrier [21]. ABC proteins can move many molecules, such as inorganic anions, peptides, amino acids, carbohydrates, hormones, and drugs [22]. he ABCB1 protein, often known as P-glycoprotein (P-gp), is one of the most studied because it has a significant role in changing how drugs are absorbed [23], [24]. P-gp aggressively removes medications from cells, which limits how much the intestines can absorb them and changes how they are distributed. Several things can change how ABCB1 works and how it is expressed. These include other medicines that are taken at the same time, illness conditions, and genetic polymorphisms. Changes in the function of P-gp, whether caused by genetic differences or environmental changes, could make treatments less effective [25].

There are a lot of different types of SLC transporters. For example, the SLC22 group includes organic cation transporters (OCTs), organic cation/zwitterion transporters (OCTNs), and organic anion transporters (OATs). The SLC6 family includes neurotransmitter transporters like SERT, NET, and DAT [26]. he SLCO1B1 gene, which used to be called SLC21A6, is a member of the SLC family that is important for medicine. This gene makes the hepatic transporter OATP1B1, which helps move various natural and artificial substances from circulation into liver cells for further processing and removal [27].

The multidrug resistance gene MDR1 encodes P-glycoprotein, which varies significantly between people because of inducers, inhibitors, and, most crucially, inherited SNPs [27]. MDR1 genes for a transmembrane ATP-dependent efflux pump that moves substrates out of the cell, including many key medications used in medicine [28], [29]. Researchers have looked at polymorphisms in the MDR1 gene, including C3435T, to see how they affect the amounts of P-gp expression and the effectiveness of transport. These genetic differences play a significant role in why people respond differently to drugs and how the drugs are processed by the body [30]. In mouse models, deleting the MDR1a or MDR1b genes has significantly affected how drugs are absorbed and distributed in tissues [31]. When P-gp is less active or expressed, more substrate medicines are absorbed throughout the body since fewer are pushed back into the intestines. On the other hand, too much P-gp can make drugs less effective, which could lead to treatment failure [32]. Figure 2 shows these effects by comparing the plasma concentration-time profiles of three people with different MDR1 genotypes. Patient A has the MDR1 Ser893 polymorphism, which causes enterocytes to make more P-gp, which makes them less able to absorb and more able to release. The wild-type MDR1 gene in Patient B shows how the transporter works normally. On the other hand, Patient C has two copies of the SNP C3435T, which changes how genes are transcribed and lowers the amount of P-gp on the cell membrane. This makes the medicine leave the cell less and makes the drug more available to the body [27].

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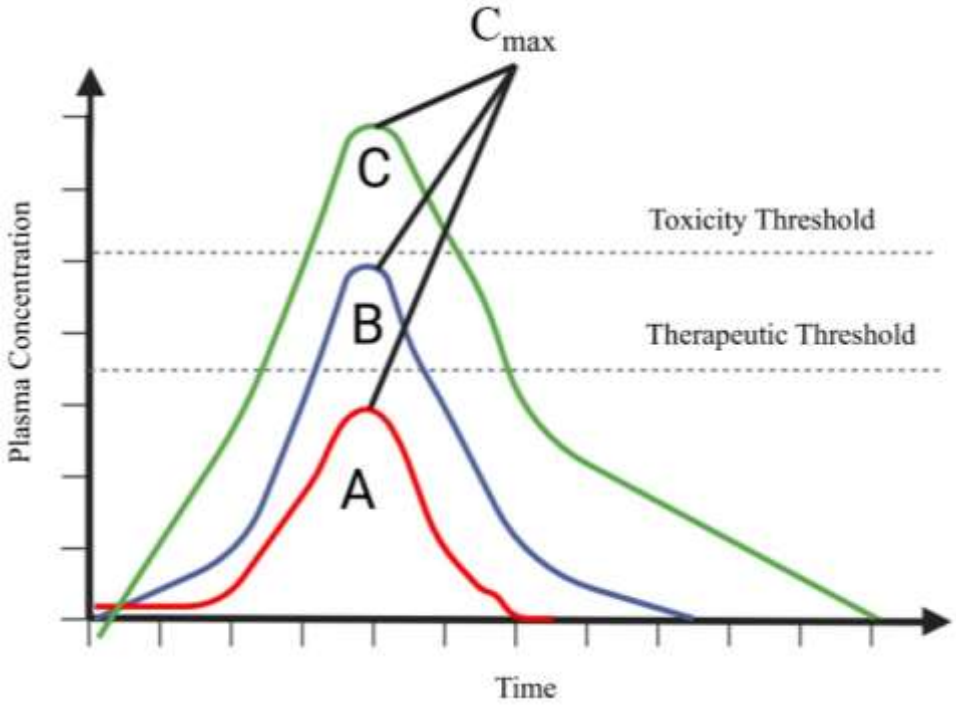

Figure 2. We compare the plasma concentration profile to the temporal curve of patients with different P-gp genotypes. These genotypes affect how well drugs that are substrates of P-gp are absorbed.

The pharmacokinetic curves in the figure show that Patient A did not reach therapeutic plasma concentrations. This is probably because P-gp activity increased, and the drug left the intestinal lumen too quickly. Patient B's levels of the drug approached the therapeutic range that was expected. Patient C, on the other hand, had much greater plasma medication levels, which were over the level of toxicity. The steepness of the ascending limb of the concentration-time curve showed that Patient C had the highest absorption rate. In this patient, the peak plasma concentration (Cmax) happened earlier. It was much higher than in Patient A or Patient B. Interestingly, the descending limbs of the curves, which show the elimination phase, were parallel in all three individuals. This suggests that the discrepancies in drug exposure were primarily due to variances in absorption rather than elimination kinetics [27].

This case shows how crucial pharmacogenomic profiling is in clinical practice. Knowing how genetic changes in transporters like P-gp affect how drugs are absorbed might help doctors guess how patients will respond, tailor drug doses to each patient, and avoid treatment failures or side effects. As pharmacogenomics becomes more common in everyday medical care, it will be a key part of the growth of precision medicine.

Clinical Implications in Personalized Medicine

Pharmacogenomics is a key element of pharmacokinetics that explains how gene differences can change how well drugs are absorbed [33]. Pharmacogenomics can also help figure out how well a drug works and how well it gets into the body. Genetic differences in drug transporters and metabolic enzymes are crucial for drug absorption [19]. Genetic differences affect the amount of medicine in plasma, how well it works, and the chance of side effects [32]. For instance, genetic variations in drug transporters such as P-gp (encoded by the ABCB1 gene) and OATP1B1 (encoded by the SLCO1B1 gene) can change how the transporter works, which could make drugs absorb faster or more effectively [27], [34], [35]. These adjustments can significantly affect how the patient responds to the drug, even if they take the same amount. This is very significant for medications that only work in a small range of doses.

Knowing how genetic variants affect drug absorption might help tailor therapeutic therapy to each patient in clinical practice. Pharmacogenomic testing helps doctors choose the right medicine and change the dose for the best results and fewer adverse effects [36], [37]. For example, the patient's genotype for

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transporters like OATP1B1 can tell how they will respond to statins [38]. Some genetic differences, for instance, can make it more likely that you will get myopathy if you have high amounts of statins in your blood [39]. Pharmacogenomics has a lot of potential for customized treatment, but there are still a lot of problems that need to be solved before it can be used in everyday medical practice [40], [41], [42]. Some of the problems include the lack of clear criteria, medical personnel not getting enough training, and insufficient clinical trial evidence to back up the clinical benefits of this strategy [40], [43]. But as technology improves and we learn more about genetics, pharmacogenomics will likely become a significant part of patient care [44], [45].

CONCLUSION

Genetic differences, like SNPs, have a big effect on how drugs work in the body, especially when they are being absorbed. P-glycoprotein and other members of the SLC family are examples of drug transporters that show how genetic differences can affect the safety and effectiveness of drug therapy. Even though research has shown how important pharmacogenomics is for drug absorption, using these findings in clinical practice is still hard. In the future, new technologies and a better understanding of pharmacogenomics may make personalized medicine much more effective, reduce side effects, and improve patient outcomes.

AUTHOR CONTRIBUTIONS

Annisa Abdi Ghifari: Conceptualization, Data curation, Visualization, Writing original draft preparation.
Alauddin Syaifulanwar: Methodology, Formal analysis.
Muhammad Yulis Hamidy: Supervision, Validation, Writing, review & editing.

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