Review Article

Pharmacogenomics In Adverse Drug Reactions: Improving Personalized Drug Safety with New Artificial Intelligence Support - A Review

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ABSTRACT

The vital topic of pharmacogenomics explains why different people react differently to the same medicine. While some people respond well to medication, others might need to change their dosage or develop adverse drug reactions (ADRs). Variations in genes linked to drug metabolism, transport, and immune response have been identified as important determinants impacting drug safety and efficacy as research in this field has progressed from researching individual genes to examining the entire genome. ADRs continue to be a significant problem in the healthcare industry, as they can lead to drug failure during development and, in certain situations, withdrawal from the market following approval. Identifying patients at higher risk of ADRs before treatment can significantly improve drug safety and therapeutic outcomes. Regulatory efforts, such as the inclusion of genetic information in drug labeling, are supporting more informed clinical decision-making. In this context, pharmacogenomics plays a crucial role in advancing personalized medicine by enabling safer and more targeted therapies. Additionally, the growing use of artificial intelligence (AI), particularly machine learning (ML), offers new opportunities to predict ADRs by analyzing complex and large-scale data. Despite these advancements, there is still a need for comprehensive evaluation of these models to ensure their accuracy, reliability, and applicability in real-world clinical settings.

Keywords: Pharmacogenomics, Adverse Drug Reaction, Artificial Intelligence.

INTRODUCTION

Pharmacogenomics is the study of how a person's genes influence how they react to medications. It seeks to develop more effective and safe individualized treatments. However, it is challenging to fully anticipate drug response because it is complicated and impacted by variables like age, diet, and lifestyle.[1] Over time, studies have demonstrated that the way our bodies react to medications can be influenced by our DNA. Each person may respond differently to the same medication due to these variations.[2] Pharmacogenomics has garnered increasing interest in recent years due to its potential to enhance patient care. Researchers are gaining a better understanding of how many biological parameters affect drug response thanks to new techniques like functional genomics and multi-omics technology. However, considerable cooperation is required to translate these scientific advancements into actual medical benefits. Collaboration is required between researchers, physicians, healthcare systems, regulatory agencies, and business partners. The successful application of pharmacogenomics in routine medical practice across a range of specialties depends on this collaborative effort.[3] Adverse Drug Reaction: When a medication is used at recommended dosages, an adverse drug reaction (ADR) is defined by the World Health Organization as a negative and unexpected effect. Because these reactions can result in severe sickness, hospital stays, and even death, they are a major public health problem. According to a significant study, serious adverse drug reactions (ADRs) rate as high as fourth among the top causes of death for hospitalized patients in the US.[4] Pharmacovigilance: The World Health Organization defines pharmacovigilance (PV) as the science and actions aimed at detecting, assessing, comprehending, and preventing side effects and other issues associated with medications. It is crucial for maintaining medication safety and safeguarding patient health.[5] Artificial intelligence: Pharmacogenomics is heavily relying on artificial intelligence, particularly machine learning and sophisticated data analysis. It makes it easier for researchers to comprehend complicated genetic data, which enables them to more accurately forecast how people could react to certain medications.[6]

Basics Of Pharmacogenomics:

The study of how inherited genetic variations affect how people react to drugs is known as pharmacogenetics. To put it simply, it explains why some people may benefit from the same medication while others may not. The word "pharmacogenomics" became more widely used as scientific study grew and new areas were discovered. This illustrates a more thorough approach, concentrating on how the entire genome rather than just certain genes—affects drug response.[7] In contemporary medication development and healthcare, pharmacogenomics has grown in importance. By assisting researchers in creating safer and more potent therapies, it represents a major advancement in medicine. Rather of employing a "one-size-fits-all" strategy, it emphasizes how an individual's genetic composition affects how they react to medications. This lowers the possibility of negative medication reactions, improves treatment outcomes, and enables researchers to discover novel therapeutic targets. Connecting genetic data

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with clinical outcomes is another important objective of pharmacogenomics, which enables the prediction of an individual's potential response to a certain treatment or their likelihood of contracting a particular disease. The majority of medications were previously created with the broad public in mind, but pharmacogenomics moves the emphasis to individualized treatment. Treatments can be more accurate and customized for each patient by going beyond outward symptoms (phenotype) and comprehending the underlying genetic variables (genotype). It is anticipated that pharmacogenomics will be crucial to the development of new drugs. In addition to improving treatment quality, this integration may shorten the time and expense needed to launch new medications.

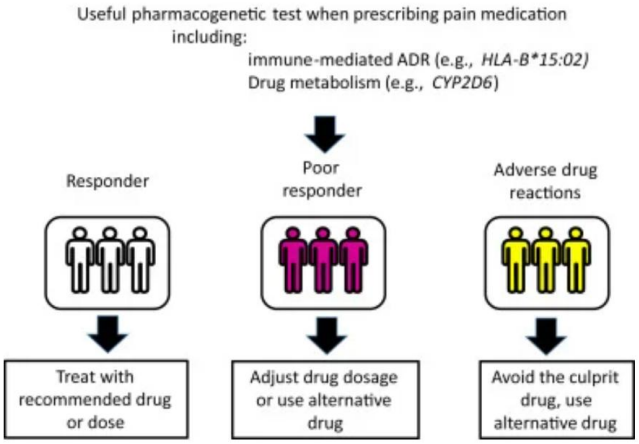

Figure 1: Pharmacogenomic testing

The Role of Genetic Variation in Adverse Drug Reaction:

Natural variations in DNA sequence between individuals and communities are known as genetic variants. Because they affect how different individuals react to the same medication, these variances are significant in medical study. A medication that works well for one individual could be ineffective or even dangerous for another due to these variations. Because genes can exist in several forms, or alleles, genetic variety arises. An individual's traits, such as medication responsiveness and metabolism, are determined by the mix of these alleles. Because of this, some people may metabolize medications more quickly than others, which might raise the possibility of adverse drug reactions (ADRs).[8] Genetic polymorphism is a widespread form of genetic variation in which multiple forms of a gene exist within a population. Such variations can influence drug-metabolizing enzymes, leading to differences in how drugs act in different individuals. For instance, insertions or deletions in DNA may result in non-functional proteins, while single nucleotide polymorphisms (SNPs) can alter gene expression and affect the quantity of protein produced. [9] Additionally, genetic alterations like gene duplication or deletion can modify protein levels in the body. Increased protein expression may speed up drug metabolism, whereas the absence of a gene may reduce or completely inhibit metabolic activity. [10] Overall, these molecular variations play a crucial role in determining drug safety and efficacy and may increase the risk of adverse drug reactions (ADRs). [11]

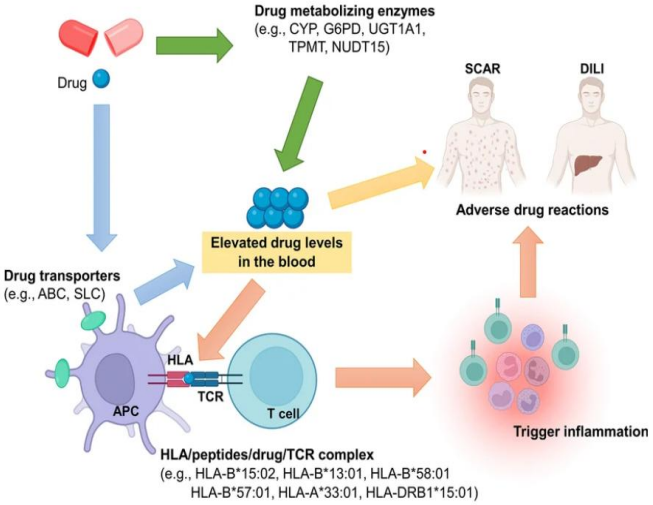

Figure 2: Genetic Variation

Concept of Personalized Medicine:

Personalized medicine is a healthcare approach that tailors treatment based on an individual's genetic makeup and personal characteristics. Unlike conventional medicine, which relies on average responses across large populations, this approach acknowledges that individuals may respond differently to the same drug. It helps in predicting whether a patient will respond positively to a treatment or may develop adverse effects. [12] The primary aim of personalized medicine is to deliver targeted and effective therapy for specific diseases and patient groups .[13] In recent years, drug development has progressed from the traditional single-target strategy to more sophisticated approaches involving molecular classification of diseases, biological networks, and multi-target therapies .[14] This advancement has been driven by developments in genomics and other "omics" technologies, along with improvements in molecular imaging and data analysis techniques. Personalized treatment also takes into account various factors such as age, gender, body weight, disease status, and lifestyle. The broader concept of personalized healthcare extends beyond treatment to include prevention, with the goal of enhancing overall health outcomes. Genomic and precision medicine play a crucial role in this field, although their clinical application remains complex due to intricate gene–disease interactions.[15] Pharmacogenomics, a key component of personalized medicine, focuses on understanding how genetic variations influence drug response. While it has enabled the development of targeted therapies, further research is required to address existing challenges and promote its wider application in clinical practice .[16]

Adverse Drug Reaction:

An adverse drug reaction (ADR) is an unintended and harmful response that occurs when a drug is taken at normal therapeutic doses. Unlike side effects, which may be mild or occasionally beneficial, ADRs are generally harmful

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in nature. They represent a significant healthcare issue, contributing to increased morbidity, hospitalizations, and mortality. Data from reporting systems such as the FDA's Adverse Event Reporting System indicate a large number of serious cases and deaths, emphasizing the extent of the problem. [17] Studies have shown that drug-related harm frequently leads to emergency department visits, many requiring hospital admission.[18] In severe cases, ADRs may result in death during hospitalization. These reactions negatively impact patient health, quality of life, and increase the overall burden on healthcare systems. Individuals with multiple diseases or those on polypharmacy are at greater risk, making management more complex. Pharmacovigilance plays a crucial role in monitoring ADRs by identifying, evaluating, and preventing drug-related issues .[19] Regulatory agencies now mandate systematic ADR reporting to enhance drug safety. There is an increasing need for effective and affordable methods to predict high-risk patients, which can help minimize risks, improve treatment outcomes, and ensure safer medication use .[20]

Adverse Drug Reaction Types:

Based on their mechanism and predictability, adverse drug responses (ADRs) are often divided into dose-related (Type A) and non-dose-related (Type B) reactions.

Table 1: Comparison of Type (A) and Type (B) Reaction:

Feature	Type (A) ADRs	Type (B) ADRs
Dose-dependence	Yes	No
Predictability	Predictable	Unpredictable
Frequency	Common	Rare
Severity	Generally less severe	Often Severe life-threatening
Mechanism	Pharmacological effects	Immune predisposition
Examples	Overdose,drug interactions	Hypersensitivity, Idiosyncratic
Influence factors	Pharmacogenetics	Genetic Susceptibility

Type A Reaction:

Type A reactions result from a drug's established pharmacological effects and are dose-dependent. The bulk of ADRs are caused by these frequent, predictable reactions. They are usually less severe and are caused by high dosage, abnormal pharmacokinetics, or medication interactions. The prevalence of Type A reactions may also be influenced by factors such genetic polymorphisms in drug-metabolizing enzymes, underscoring the importance of pharmacogenetics in drug therapy optimization.[21]

Type B Reaction:

On the other hand, Type B reactions are mostly unpredictable and do not depend on dose These responses, which are relatively uncommon but frequently more severe and potentially fatal, happen to sensitive people. Hypersensitivity reactions, idiosyncratic reactions, and pseudo allergic reactions are examples of type B reactions. Many of these can appear even at typical therapeutic levels and are mediated by immunological systems or genetic predisposition.

ADRs Mechanisms Impacted by Genetic Factors:

Genetic variability is now acknowledged as one of the primary causes of adverse drug reactions (ADRs), which continue to be a significant problem in clinical practice .[22] Variations in a person's genetic composition might change how the body reacts to and processes medications, making them more vulnerable to toxicity or unanticipated side effects. Mechanisms including drug metabolism, drug transport, immunological responses, and drug targets provide a general explanation for these genetic implications on adverse drug reactions.

Drug Metabolism

genetic variations in enzymes that metabolize drugs, especially those in the cytochrome P450 enzyme family. [23] Individuals can be categorized as poor, intermediate, extensive, or ultra-rapid metabolizers based on variations in the genes encoding these enzymes.[24] While ultra-rapid metabolizers may convert medications too fast, occasionally creating toxic metabolites or decreasing therapeutic efficacy, poor metabolizers may accumulate larger drug concentrations, raising the risk of dose-dependent toxicity. In many frequently reported ADRs, this diversity is crucial.[25]

Drug Transportation:

Drug transporters control how medications pass across cell membranes. Drug absorption, distribution, and excretion can be impacted by genetic differences in transporter proteins, such as P-glycoprotein.[26] Modified transporter activity may result in decreased drug availability at the target site, impacting both efficacy and safety, or increased drug accumulation in certain organs, increasing the risk of organ-specific toxicity .[27]

Immune-Related Genes:

The human leukocyte antigen (HLA) system's genetic variations are frequently linked to immune-mediated adverse drug reactions (ADRs).[28] When exposed to certain medications, people with certain HLA alleles may be more susceptible to severe hypersensitivity reactions. In these situations, the medication or its metabolite interacts with immune cells, causing an improper immunological response that can cause anything from minor skin responses to potentially fatal diseases like toxic epidermal necrolysis or Stevens-Johnson syndrome. [29]

Drug Targets:

pharmacological sensitivity is altered by genetic differences in pharmacological targets, such as enzymes or receptors, which also contribute to adverse drug reactions.30 Modifications to the structure or function of receptors may increase or decrease a drug's pharmacological activity,

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resulting in toxicity or treatment failure.[31] The strength and duration of pharmacological reactions can also be altered by polymorphisms in genes related to downstream signaling pathways.[32]

Table 2: Genetic Mechanisms Influencing ADRs

Mechanism	What changes due to genetics	Effect on drug	Resulting ADR	Example
Drug metabolism (CYP enzymes)	Altered enzyme activity (↑ or ↓ metabolism)	Increased or decreased drug levels	Toxicity or reduced effect	CYP2D6 + Codeine → Toxicity
Drug transport (P-gp)	Altered transporter function	Drug accumulation in tissues	Organ toxicity	P-gp + Digoxin → Toxicity
Immune Response(HLA genes)	Abnormal immune activation	Hypersensitivity reaction	Severe ADR (SJS/TEN)	HLA-B*1502 + Carbamazepine → SJS
Drug target (Receptors /Enzymes)	Altered drug target sensitivity	Increased or decreased drug effect	Over-response or treatment failure	VKORC1 + Warfarin → Bleeding

Genes Associated with Adverse Drug Reactions:

ADRs, or adverse drug reactions, are a frequent problem in healthcare and can occasionally result in major consequences. The fact that different people react differently to the same medicine is a major contributing factor to these reactions. Genetic variations, which impact on how medications are digested and how the body's immune system responds to them, frequently have an impact on these discrepancies. Understanding these differences and enhancing medication safety are made easier by pharmacogenomics. ADRs are known to be influenced by a number of significant genes. Many medications are metabolized by the Cytochrome P450 enzyme system, and genetic variations in these enzymes can result in either drug toxicity or decreased efficacy.[33] Immune-mediated medication reactions, especially severe hypersensitivity reactions, are linked to the Human Leukocyte Antigen (HLA) system.[34]Thiopurine S-methyltransferase (TPMT), another important gene, is involved in the metabolism of thiopurine medications; decreased enzyme activity can raise the possibility of severe toxicity.[35]

CYP450 Enzymes:

A broad class of hemoglobin-containing proteins known as the Cytochrome P450 (CYP) enzymes is essential to drug metabolism. Over millions of years, they developed to aid in the body's detoxification of foreign substances.[36] They are presently in charge of degrading a variety of widely used medications.[37] The majority of drug metabolism in humans is handled by a small number of CYP enzymes, including CYP1A2, CYP2C9, CYP2C19, CYP2D6, and CYP3A4.[38] These enzymes' activity can be changed by genetic changes, which can result in variations in how people react to drugs. Because of medication buildup or decreased efficacy, this may raise the risk of adverse drug reactions (ADRs). buildup or diminished efficacy.[39]

Human Leukocyte Antigen, or HlA Genes:

ADRs, or adverse drug reactions, are a significant healthcare issue that can occasionally result in severe or fatal illnesses. When medications or their metabolites function as foreign antigens, CD4+ and CD8+ T cells are activated in idiosyncratic (Type B) reactions, which are erratic and frequently immune-mediated.[40] By exposing T cells to these antigens, human leukocyte antigen (HLA) molecules play a crucial part. Some people are more vulnerable to severe reactions including Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and drug-induced liver injury (DILI) because of genetic diversity in HLA .[41] Pharmacogenetic (PGx) data aids in the identification of genetic variants that affect medication response and toxicity, particularly in HLA genes and drug-metabolizing enzymes. Although routine clinical application is still developing, this makes it possible to predict high-risk patients, facilitates tailored therapy, and enhances medication safety.[42]

Thiopurine S-Methyltransferase, Or TPMT:

An enzyme called thiopurine S-methyltransferase (TPMT) aids in the breakdown of thiopurine medications such as azathioprine and 6-mercaptopurine (6-MP) These medications are used to treat autoimmune illnesses, leukemia, inflammatory bowel disease, and organ transplant recipients.[43] Due to the limited therapeutic range of these medications, even slight dosage adjustments may result in severe adverse effects, particularly bone marrow suppression. Individual differences in TPMT activity are caused by genetic variants (SNPs). Some persons have little or no enzyme activity, which raises the possibility of serious toxicity and causes drug buildup. TPMT2, TPMT3A, and TPMT*3C are common risk alleles. There are several variations of the TPMT gene, which is found on chromosome 6.[44] Before beginning thiopurine medication, testing for TPMT, a well-known example of pharmacogenetics, is advised. All things considered, TPMT testing supports individualized treatment, enhances medication safety, and aids in dose adjustment.

An Illustration of A Pharmacogenomically Based Adverse Drug Reaction:

Carbamazepine Toxicity:

Rare but potentially fatal severe cutaneous adverse responses (SCARs) including Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) can be brought on by carbamazepine. The HLA-B*15:02 allele is closely linked

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to these reactions, especially in populations from East and Southeast Asia. Drug-induced changes in antigen presentation trigger cytotoxic T cell activation, which harms skin and mucosal tissues. A wider range of hypersensitivity events, such as drug reaction with eosinophilia and systemic symptoms (DRESS), are associated with another mutation, HLA-A*31:01. Recent pharmacogenomic guidelines and studies further reinforce the well-established significance of genetic variability in medication response .[45] It is further reinforced by current pharmacogenomic guidelines and research , which suggest pre-treatment genetic screening to enhance individualized treatment and lower the incidence of these severe adverse medication events.

Thiopurines Toxicity:

The risk of severe myelosuppression frequently limits the clinical utility of thiopurine medications, such as azathioprine, 6-mercaptopurine, and thioguanine, which are commonly used in leukemia, autoimmune disorders, and inflammatory bowel illnesses. Genetic differences in Thiopurine S-methyltransferase (TPMT), which controls thiopurine metabolism, are closely linked to this toxicity. Bone marrow suppression results from the accumulation of hazardous metabolites in patients with decreased TPMT activit.[46] Furthermore, NUDT15 polymorphisms make a person more vulnerable to negative consequences. By allowing for dose customization and better medication selection, pharmacogenomic testing before therapy can dramatically lower the frequency of such adverse drug reactions, bolstering the application of personalized medicine in clinical practice.[47]

Abacavir Sensitivity:

Although it has been linked to early virologic failure in individuals with high baseline viral levels, abacavir is an efficient nucleoside reverse transcriptase inhibitor used in combination therapy for HIV infection.[48] Within the first six weeks of treatment, 5–8% of patients experience a potentially severe hypersensitivity reaction that manifests as fever, rash, gastrointestinal, and respiratory symptoms.[49] The HLA-B*57:01 allele is closely associated with this negative reaction . It has been demonstrated that pre-treatment genetic screening for HLA-B*57:01 successfully prevents this reaction, and those who test positive should not be prescribed abacavir. This pharmacogenetic approach has almost completely eradicated abacavir hypersensitivity, demonstrating a noteworthy breakthrough in precision medicine .[50]

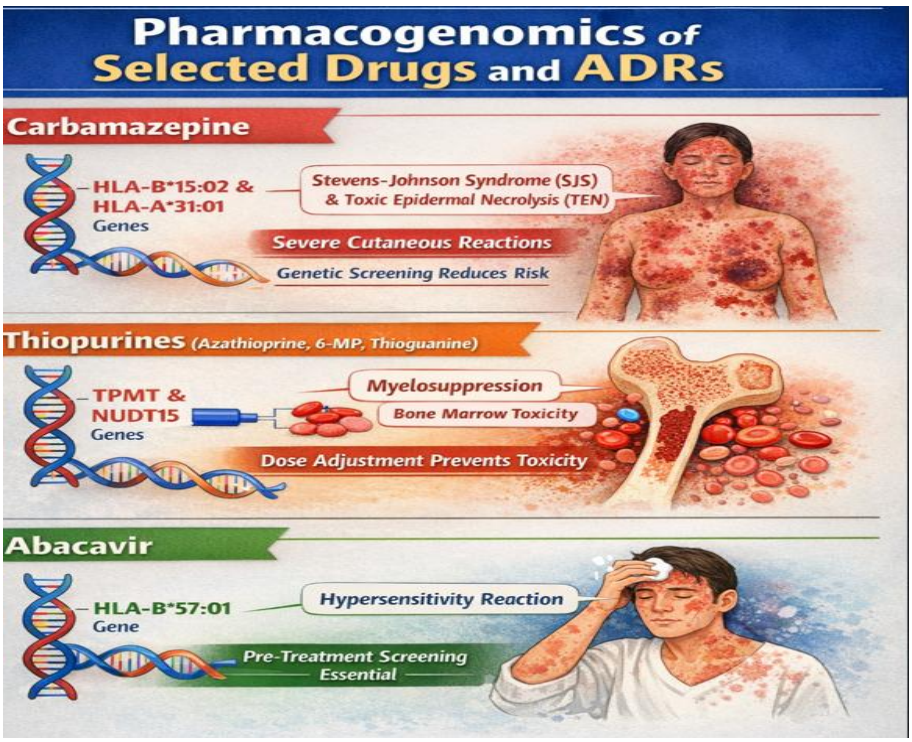

Figure 3: Visualizing Drug-Gene Interaction and Adverse Reactions

Pharmacogenomic's Role in Pharmacovigilance:

In recent years, pharmacovigilance has changed from a system that was mainly concerned with reporting adverse drug reactions (ADRs) to a more all-encompassing approach that also takes genetic determinants of drug response into account.[51] This shift reflects a better awareness that genetic variables frequently influence interindividual heterogeneity in medication efficacy and toxicity. In keeping with this development, the Pharmacogenomics Special Interest Group (PGx SIG) was founded by the International Society of Pharmacovigilance (ISoP) in 2020, highlighting the increasing significance of incorporating pharmacogenomics into pharmacovigilance frameworks. Professionals with varying degrees of pharmacogenomics competence, from experts actively involved in the subject to those looking to broaden their understanding, can collaborate on the PGx SIG.[52] The group encourages the integration of

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pharmacogenomic principles into standard pharmacovigilance practice and enables knowledge exchange through organized activities like regular meetings, scholarly talks, and training initiatives.

The Function of Artificial Intelligence in Pharmacogenomics:

By assisting in the interpretation of intricate biological and clinical data for better treatment choices, artificial intelligence (AI) plays a significant supporting role in pharmacogenomics. In order to better understand how genetic variations impact how people react to medications, it combines multi-omics and clinical data.[53] AI also aids in the identification of genetic markers associated with medication metabolism, toxicity, and efficacy. This enables the transition to more individualized therapy methods that are more appropriate for each patient. By identifying individuals with similar genetic patterns, it also facilitates patient grouping, which can enhance medication selection and lower the possibility of negative drug reactions. Beyond this, by identifying patterns linked to therapeutic response and drug resistance, AI advances drug research and development. Additionally, it makes pharmacogenomic analysis more usable in actual healthcare settings by using methods like natural language processing (NLP) to retrieve valuable clinical information from electronic health records.[54]

Ai Pharmacogenomics Technologies:

By assisting in the interpretation of intricate genetic and clinical data, artificial intelligence (AI) is becoming more and more significant in pharmacogenomics, ultimately supporting more accurate and customized treatment choices. According to recent research, AI-based methods enhance the capacity to discover significant biological indicators, anticipate how patients will react to medications, and integrate various clinical data types for improved treatment planning.[55]

Learning Machines Applications:

To comprehend how genetic variations affect pharmacological response, machine learning techniques-both supervised and unsupervised-are frequently employed. By examining extensive and intricate genomic datasets, these methods aid in the identification of genetic variants associated with therapeutic efficacy, toxicity, and metabolism. By identifying minute patterns in genetic data that are difficult to find using conventional techniques, sophisticated deep learning models significantly improve this procedure. This supports more individualized therapy approaches in pharmacogenomics, enhances medication selection, and provides a better knowledge of patient-specific reactions.

Platform for Data Analytics:

By integrating data from various sources, including genomic data, electronic health records (EHRs), clinical trials, patient feedback, and empirical evidence, data analytics systems enhance pharmacogenomic research. A more comprehensive picture of how various individuals react to drugs in actual clinical settings is provided by this integrated approach. Large dataset processing and the creation of predictive models are supported by frameworks and tools like TensorFlow and Py-Torch. Overall, the accuracy, dependability, and clinical utility of pharmacogenomic predictions are enhanced by this integration of many data sources.[56]

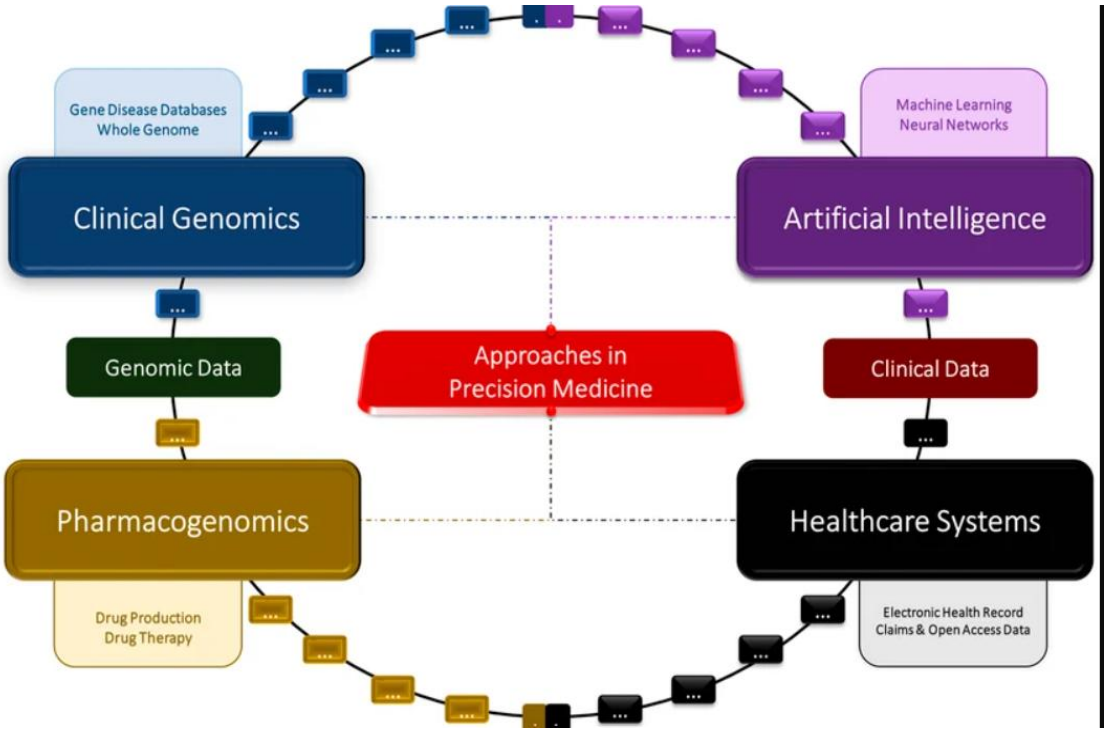

Figure 4: Artificial Intelligence

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DISCUSSION

• Clinicians' lack of knowledge: Many medical professionals lack the necessary training to understand and use pharmacogenomic data when making clinical decisions.[57]

• Need for equitable implementation: In order to guarantee that pharmacogenomics is used in various healthcare settings in a fair, efficient, and safe manner, these obstacles must be removed.[58]

• Lack of standardized laboratory procedures: Variations in reporting and testing techniques lower reliability, underscoring the necessity of consistent PGx testing guidelines.[59]

• Legal and ethical issues: Concerns about data privacy, informed permission, and the possibility of genetic discrimination continue to present significant ethical difficulties.[60]

CONCLUSION

By assisting in the customization of medicines based on a person's genetic composition, pharmacogenomics is becoming more and more significant in reducing adverse drug reactions (ADRs). It enhances the capacity to anticipate, track, and avoid drug-related dangers when paired with pharmacovigilance, promoting a change from reactive to more preventative healthcare. Developments like polygenic risk scores and multi-gene techniques have improved the evaluation of a patient's vulnerability to adverse drug reactions. Furthermore, by effectively evaluating complicated biomedical data and enhancing the precision of risk assessments, artificial intelligence (AI) contributes to this advancement. When combined, pharmacogenomics and AI offer a potent strategy for improving medication safety.

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