The Era of Pharmacology Forword Pharmacogenomics

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Abstract: Over the past three decades, healthcare has made significant strides in enhancing patient survival and overall quality of life. This progress can be attributed to the development of more potent and selective therapeutic drugs, as well as improved patient care services. Pharmacogenomics, a field of study, focuses on identifying an individual's genetic traits that play a role in their response to medications. What's particularly fascinating is that as scientific understanding has advanced, it has started to consider the patterns of genetic variations within specific populations, including various ethnic groups, to account for the variations observed in responses to pharmacotherapy. The word "pharmacogenomics" is used more broadly in this chapter to refer to genetic variations that are present in a patient community, such as an ethnic group, rather than in a single patient. The efficacy and potential toxicity of numerous medications are impacted by the individual variances in drug-metabolizing enzymes and transporters. Pharmacogenomics and its forerunner, pharmacogenetics, investigate how genetic characteristics affect an individual's sensitivity to and safety from drugs. One of the primary goals of pharmacogenomics is the personalized tailoring of drugs for specific individuals according to their genetic and molecular characteristics. The field of pharmacogenomics has evolved significantly from its initial discoveries in the 1950s, which identified inherited deficiencies in drug metabolism and explained drug-related adverse effects. It now encompasses contemporary genome-wide methodologies that assess genetic variations across multiple genes.

Pharmacogenomics can extend its scope to drug discovery and development, where mounting evidence indicates that genetically defined targets are associated with higher success rates in clinical development. This overview offers insights into the historical progression and contemporary uses of pharmacogenomics in patient selection, dosing, and drug development, supplemented with illustrative instances from each category. Additionally, it discusses the challenges in the field and provides a glimpse into future perspectives.

Key words: Heredity, Personalized medicine, Pharmacogenomics, Pharmacokinetics, Pharmacodynamics, Polymorphisms.

I. INTRODUCTION

Pharmacogenetics is the study of variation in pharmacological response resulting from inheritance [1]. The term "pharmacogenomics" has gained popularity in recent times, aligning with the trend of appending "omics" to research fields. The latter expression has a broader scope, encompassing all genes in the genome that could influence drug responses. In contrast, the former term typically relates to genes primarily involved in drug metabolism [2]. A crucial step towards the personalization of medicine, pharmacogenomics (PGx) tries to ascertain how genetic and genomic variants affect pharmacological reactions, or drug efficacy and toxicity [3]. The integration of molecular diagnostics into standard clinical practice is recognized for its significant potential, largely attributed to pharmacogenomic biomarkers that can anticipate individual responses to drugs. Distinguishing between two distinct categories of biomarkers is valuable in the context of medical treatment. The first category comprises germline biomarkers, which have the potential to influence the way a treatment is processed within the body (pharmacokinetics) and how it exerts its effects (pharmacodynamics). The second category involves biomarkers found in the somatic genome of cancer cells, which play a pivotal role in determining how these cancer cells respond to specific drugs. This differentiation provides essential insights into the impact of various biomarkers on treatment outcomes. In addition to hereditary considerations, variations in medication responsiveness have been related to epigenetic alterations of DNA or histones. Epigenetic

changes in cancer cells have been associated with higher production of drug efflux transporters, which mediates treatment resistance. The identification of epigenetically altered DNA in the bloodstream represents an evolving tool for tracking the effectiveness of treatment, the development of drug resistance, and tumor classification [4], [5].

The phrase "pharmacogenomics" has more recently been employed to describe the transition from genetics to genomics, recognizing that the genome encompasses more than just its individual genes. In the quest to identify genes associated with a specific disease, a new dimension is introduced into the genome-wide approach. Pharmacogenetics delves into the exploration of individual variations in DNA sequences linked to pharmacological effects (pharmacodynamics) or drug processing (pharmacokinetics) that can have an impact on clinical outcomes. In contrast, pharmacogenomics is a broader field that encompasses the utilization of genomic technologies to elucidate various aspects of healthcare. This includes identifying susceptibility to illnesses, facilitating drug discovery, understanding the pharmacological actions of drugs, determining how the body processes medications (drug disposition), and predicting responses to treatment. It has the potential to create novel categorizations of diseases at the molecular level, offering a deeper understanding of the genetic underpinnings of various medical conditions. Moreover, the identification of new disease-related genes will pave the way for the revelation of fresh therapeutic targets. Among the over 30,000 recognized disorders, more than 100 to 150 prevalent conditions lack pharmacological remedies or necessitate improved drug interventions. There are at least 3,000 to 10,000 "drugable" targets, and currently used medications target about 500 biological targets that are pharmacologically active [6].

History of pharmacogenomics /genetics: -

In 1959, the word "pharmacogenetics" was first used [7]. In 1962, the first textbook was released [8]. The idea that varying medication response may represent sets of polymorphisms inside a person or across a population has only recently been conveyed by the term "pharmacogenomics." Through a variety of processes, DNA variations can alter how proteins operate and, consequently, how drugs respond. Initially, much of the focus in this field centered on nonsynonymous DNA variations, which are alterations in the genetic code that result in changes to the amino acids encoded by the DNA, thereby impacting protein function. However, another frequently suggested potential explanation for inconsistent responses to medications revolves around noncoding variations that have the capacity to modify gene expression. Given the discoveries in modern genomics related to various mechanisms that affect gene function and expression, such as epigenetic modifications and small interfering RNAs, it is reasonable to consider the role of noncoding variations in influencing responses to medications [9].

The English scientist Garrod was the first to put up the idea that genetic variations might regulate the variety in pharmacological responses [10]. He postulated that enzymatic abnormalities could lead to clinically significant accumulations of exogenously administered substrates, including drugs, meals, and toxins, in addition to the buildup of endogenous substrates seen in "inborn errors of metabolism," a term he coined. The discovery of pseudocholinesterase deficiency as a cause of prolonged paralysis following the administration of the muscle relaxant succinylcholine was among the earliest instances of genetically determined variations in pharmacological responses [11]. It was followed by observations of inadequate N-acetylation of isoniazid [12] and a substantial increase in hemolytic anemia among African-Americans with G6PD deficiency who were prescribed antimalarial drugs in the South Pacific during World War II [13]. Vogel introduced the term "pharmacogenetics" in 1959 to describe a new scientific discipline focused on investigating genetic differences in how individuals react to drugs [14].

II. MECHANISM OF PHARMACOGENOMICS

Pharmacogenomics involves the application of genomics, proteomics, transcriptomics, and metabolomics data to investigate the variations in individuals' responses to drug therapy and the

underlying mechanisms leading to diverse drug reactions. Each individual possesses a distinct genetic composition that influences their vulnerability to specific health conditions and their responses to drugs and environmental factors [15]. At intervals of roughly 300 to 1000 nucleotides, the human genome houses a multitude of more than 14 million single nucleotide polymorphisms (SNPs), which give rise to genetic distinctions among individuals [16]. Therefore, a fundamental objective of genetics is to pinpoint the DNA variants that play a substantial role in the population variances of each characteristic [17]. When one drug elimination pathway' s genetically determined reduced function is combined with the absence of alternative pathways that may easily perform the same role, the chance of abnormal drug reactions is highest [18].

Pharmacogenomics investigates disparities in DNA sequences that impact the efficacy of medications. Common genetic variants include SNPs, genomic insertions and deletions, as well as copy-number variations (CNVs). SNPs and CNVs both contribute to various phenotypic outcomes and measurements in pharmacogenomics. Pharmacogenomics involves the examination of genetic disparities in drug targets, receptors, transporters, and drug-metabolizing enzymes, as well as the interactions between these variations that lead to outcomes such as drug response or toxicity. A significant number of these genes exhibit variations in expression and function among individuals, and these variations have been associated with single-nucleotide distinctions. These distinctions can arise from inherited single-nucleotide polymorphisms (SNPs) or somatically acquired SNPs, as well as differences in DNA methylation and changes in copy numbers. Additionally, the role of microRNA (miRNA) has been implicated in these regulatory mechanisms [19], [20].

III. PHARMACOGENOMIC AFFECT ON DRUG DESIGN, DEVELOPMENT, AND PRESCRIBING

The systematic procedure for identifying potential new drugs is referred to as drug discovery. Drug development has traditionally been a challenge for pharmaceutical corporations, who use well-established pharmacology and chemistry-based methods [21]. The increasing demand for a rapid and low-risk production of a large number of drugs has generated significant enthusiasm for the field of bioinformatics [22]. Pharmacogenomics involves the examination of the impact of genetic polymorphisms and genomic variances on an individual's response to drugs. Utilizing this knowledge can aid in the selection of the most suitable medication, optimal dosage, and treatment approach, all while mitigating the risk of adverse drug reactions [23]. This is accomplished by examining genetic patterns and polymorphisms within genetic elements that interact with drugs or their metabolites and are connected to their pharmacokinetics or pharmacodynamics in some manner [24], [25].

IV. PHARMACOGENOMICS: CLINICAL IMPORTANCE

Pharmacogenomics is a swiftly expanding field with substantial therapeutic potential, particularly in tailoring medication to enhance effectiveness and minimize the risk of toxicity on an individual basis [26]. Nevertheless, there exists a divergence of opinions on the most effective means of integrating pharmacogenomics into clinical practice. Several actionable pharmacogenomic cases with a strong evidentiary basis have, over time, lost their relevance due to alternative strategies such as altering the choice of medication or increasing patient monitoring. A significant concern pertains to the potential resurgence of drugs that are linked to pertinent pharmacogenomic information as the accessibility of pharmacogenomic testing expands and costs decrease. Moreover, recommendations from healthcare organizations do not consistently endorse pharmacogenomic testing; however, they occasionally suggest scenarios in which pharmacogenomics might prove beneficial in the treatment of individual patients [27].

V. EXAMPLES OF CLINICAL APPLICATION OF PHARMACOGENOMICS

Proton Pump Inhibitors:

Proton Pump Inhibitors (PPIs), which encompass medications like omeprazole, esomeprazole, lansoprazole, dexlansoprazole, pantoprazole, and rabeprazole, are typically prescribed for the management of a range of conditions associated with gastric acid. These conditions include gastroesophageal reflux disease (GERD), gastric

ulcers, duodenal ulcers, and Zollinger-Ellison syndrome. For the eradication of Helicobacter pylori (H pylori), they are used in conjunction with one or two medications [28], [29]. CYP2C19 and CYP3A metabolise omeprazole, esomeprazole, lansoprazole, and pantoprazole predominantly in these drugs. The extent of metabolism through CYP2C19 and CYP3A enzymes can differ among various PPIs. [30]. In the case of drugs metabolized by CYP2C19, a gene-dose effect is often observed, allowing individuals to be categorized based on their CYP2C19 genotype. Those with two wildtype alleles are referred to as CYP2C19 homozygous extensive metabolizers (EMs), individuals with one wildtype allele and one decreased/null variant allele are termed CYP2C19 heterozygous EMs, and those with two decreased/null activity alleles are designated as CYP2C19 poor metabolizers (PMs) [31].

While the use and dosing of PPIs can vary when administered in H. pylori dual/triple therapy for eradication, there is currently a lack of published studies that have specifically examined PPI dosing regimens based on an individual's CYP2C19 genotype. This could be attributed to the numerous available dual/triple therapy regimens, the broad therapeutic range of PPIs, and the infrequency of clinically significant adverse effects. While recommendations for higher PPI doses have been proposed for individuals with homozygous extensive metabolizer (EM) genotypes, this approach has not been widely implemented in clinical practice [32]. Pharmacogenomics may have an effect on clinically significant results in drug dosage, effectiveness, and toxicity, leading to suggestions for testing. Pharmacogenomics hasn't yet supplied strong enough data to call for such testing for PPIs and codeine, though. The fact that both hereditary and nongenetic factors will be involved in determining the therapeutic usefulness of some medications is one possible explanation. Determining how much each of these elements contribute is equally crucial. Non-genetic factors that influence the probability of successfully eradicating H. pylori infection encompass factors such as compliance with the prescribed therapy, the presence of antibiotic resistance, and the concurrent use of other medications [33].

Codeine

Prescribed for the relief of mild to moderately severe pain, codeine is a weak opioid agonist that acts on the central nervous system. Codeine sulfate tablets contain codeine. Codeine, functioning as a prodrug, requires conversion into the active metabolite morphine, a process involving O-demethylation, and the enzyme CYP2D6 plays a crucial role in this conversion [34], [35]. CYP2D6 is responsible for hydroxylating or demethylating numerous medications, including neuroleptics, antidepressants, certain beta-blockers, and codeine [36]. The CYP2D6 gene has at least 80 recognized variants and is located on chromosome 22 [37] Genetic variations in factors such as CYP2D6 can be employed to elucidate the interindividual differences in enzyme activity [38], [39].

Carbamazepine

Trigeminal neuralgia, bipolar illness, and partial and generalised seizures are all treated with the anticonvulsant carbamazepine [40]. In contrast to type A adverse drug reactions, which are dose-dependent, carbamazepine has been linked to severe idiosyncratic type B adverse drug reactions that can be life-threatening [41]. The HLA (human leukocyte antigen) system has been a primary focus for investigating Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) reactions, which also exhibit a familial tendency, likely due to earlier observations of hypersensitivity reactions occurring in families and among identical twins [42].

VI. PHARMACOGENOMIC: TARGETS IN VARIOUS DISORDER:

TREATMENT OF RHEUMATOID ARTHRITIS

Precision medicine is defined as tailoring the diagnosis and treatment of a patient based on their genetics, biomarkers, phenotype, or psychosocial characteristics, with the aim of reducing unnecessary adverse events and improving clinical outcomes [43], [44]. One emerging application within this field is pharmacogenomics, which adjusts the choice of medications and their dosages by considering a patient's genetic composition [45]. Although several pharmacogenetic guidelines have been recently issued by international scientific consortia, the integration of pharmacogenomics into clinical practice is still in its early stages. From fundamental pharmacogenomics research through implementation, a number of

significant obstacles have been discovered, and numerous coordinated worldwide efforts are under way to address them [46]. Rheumatoid arthritis (RA) is an inflammatory condition characterized by chronic joint inflammation that affects the entire body. It primarily involves persistent synovitis and the infiltration of immune cells in the peripheral joints of the hands and feet, leading to the loss of articular cartilage and bone deterioration [47].

The chronic pain stemming from joint deformity and functional impairment results in physical disability, reduced quality of life, and the development of cardiovascular and other comorbid conditions as rheumatoid arthritis (RA) progresses [48]. Unfortunately, the use of nonsteroidal anti-inflammatory drugs (NSAIDs) for RA treatment does not halt joint destruction, and therefore, it does not provide a cure for the condition. Although they quickly treat symptoms and treat disease, glucocorticoids have substantial long-term negative effects [49]. The key to treating RA is the use of disease-modifying antirheumatic medicines (DMARDs), which regulate the course of the disease through anti-inflammatory and immunomodulatory activities [50]. The understanding of the part played by several cytokines, particularly TNF-, in the pathophysiology of RA has had a significant impact on how the illness is treated [51]. They strongly affect cellular and humoral immunity by modulating immunological responses [52]. Treatment with anti-tumor necrosis factor (anti-TNF) therapy for rheumatoid arthritis (RA) is linked to FCGR2A, a gene encoding an Fc receptor found in various immune cells, with primary expression in macrophages and dendritic cells [53], [54]. The concept of targeting B-cells as a therapy for RA was initially proposed in the 1990s. This approach is based on the theory that autoantibodies, including rheumatoid factor, promote B-cell survival and consequently contribute to the perpetuation of chronic inflammation [55].

The management of hepatitis C

Understanding the differences in disease development and treatment response brought about by the discovery of important molecular biomarkers for HCV has allowed us to quantify the burden of infection, understand the natural history of the disease, and create preventive strategies [56], [57]. It has been more than two decades since the discovery of HCV [58]. Despite the considerable costs and notable toxicity associated with pegylated-interferon and ribavirin (PEG-IFN/RBV) combination therapy, less than 50% of patients with the most common HCV genotype can achieve a sustained virological response (SVR), defined as the absence of detectable HCV RNA in serum six months after completing treatment [59]. The introduction of direct-acting antiviral treatments is expected to significantly improve the response rate. However, to help prevent viral breakthrough, these new medications must now be administered in conjunction with PEG-IFN/RBV [60].

Individuals with chronic HCV infection generally experience a lower health-related quality of life compared to the general population or those with chronic hepatitis B. For instance, when compared to patients who contracted HCV through blood transfusions, injecting drug users with HCV tend to exhibit a poorer health-related quality of life [61],[62]. Reports indicate that HCV infection rates are at least five times higher among individuals with severe mental illness than in the general population, and HCV-infected patients are more likely to have psychiatric and substance use disorders. This association significantly hampers the pharmacological and clinical management of HCV infection [63]. Because of this, HCV patients with psychiatric illnesses are less likely to be eligible for treatment programmes, which raises their rates of morbidity and mortality [64].

Treatment of cancer

Certainly, gaining a more profound understanding of the genetic factors that influence the response to chemotherapy can enable the prediction of which patients are at a higher risk of severe toxicity or those who may benefit from a specific treatment regimen. Through the use of molecular diagnostics, such as genotyping, these insights can be integrated into clinical practice to aid in the determination of the most suitable drug combination and dosage for each patient. Recently, several comprehensive reviews on cancer pharmacogenomics have been published [65], [66]. Leukemia is treated with the purine antimetabolite 6-Mercaptopurine (6-MP). Its anticancer effects are achieved by suppressing the synthesis of nucleotides essential for DNA and RNA production. The enzyme thiopurine methyltransferase (TPMT) catalyzes the S-methylation of 6-MP to produce inactive metabolites.

The bioavailability and toxicity of 6-MP are significantly impacted by genetic differences in the TPMT gene. It has been established that TPMT deficiency is an autosomal recessive condition that affects roughly 1 in 300 people. When given 6-MP, patients who have TPMT polymorphisms run the risk of experiencing severe hematologic toxicities because these polymorphisms slowdown 6-MP metabolism [67], [68].

In a recent study, it was found that 71% of patients with bone marrow resistance to 6-MP exhibited a phenotypic deficiency in TPMT. These patients had a higher probability of hospitalization, requirement for platelet transfusions, and non-adherence to chemotherapy doses [69]. 5-fluorouracil (5-FU) and its derivatives remain among the most commonly prescribed chemotherapy drugs due to their effectiveness against various types of tumors and their compatibility with other chemotherapy agents. Approximately 80% to 95% of administered 5-FU is metabolized into physiologically inactive byproducts that are excreted in the urine and bile, while only around 5% is converted into cytotoxic nucleotides responsible for its anticancer effects [70]. One of the primary mechanisms of 5-FU action involves the inhibition of thymidylate synthase (TS) by FdUMP. Thymidylate is a vital precursor for thymidine triphosphate, which is necessary for DNA synthesis and repair, and TS is the key enzyme in its de novo production. [71].

Treatment of diabetes

Type 2 diabetes mellitus (T2DM) is a complex metabolic disorder characterized by hyperglycemia resulting from issues with insulin secretion, insulin action, or both [72]. The development of insulin resistance and secretory insufficiency in T2DM is influenced by a combination of genetic and environmental factors. Recent advancements in our understanding of the biology of this condition have prompted significant changes in the approach to treating T2DM [73]. For individuals with T2DM to achieve glycaemic control, a large variety of pharmacological therapies with various mechanisms of action are available. Apart from insulin replacement therapy, conventional oral treatments for diabetes encompass insulin sensitizers that enhance insulin effectiveness and secretagogues that stimulate insulin release from the pancreas [74]. Dipeptidylpeptidase-4 (DPP4) inhibitors, also known as gliptins, represent a novel class of medications that enhance the "incretin effect" and promote glucose-triggered insulin secretion [75].

In addition to sodium-glucose cotransporter-2 (SGLT-2) inhibitors, which reduce hyperglycemia by increasing the excretion of glucose in the urine [76]. Numerous pharmacogenetic studies have been conducted in response to the significant variability in responses to diabetes mellitus medications. Nevertheless, there has been only one pharmacogenomic genome-wide association study (GWAS) focused on evaluating the efficacy of metformin treatment [77]. Trials that concentrated on individual oral agents have undergone extensive reviews in the past [78], [79]. When simulating a drug intervention within a functional network, it is essential to consider how the medication impacts the functional network at cellular, tissue, and disease levels, as this will determine the drug's beneficial and adverse effects. Our comprehension of biological networks has primarily been limited to general, static models without this contextual information [80].

This will make it possible to model the effects of drugs on different cell types, tissues, and physiological states. Such assessments can uncover how drugs achieve their intended therapeutic effects at the target tissue or known site of action, while also highlighting potential adverse effects in other contexts. Adopting a system-wide approach may redirect the focus of medication development for complex conditions like T2DM from a singular emphasis on individual proteins or genes to launching comprehensive strategies targeting a range of dynamic factors [81].

VII. PHARMACOGENOMIC: FUTURE PROSPECTIVE

Pharmacogenomics is the scientific field that explores and determines the extent to which genetics contributes to individual variations in drug responses [82]. Pharmacogenomics involves the examination of DNA sequence variations that influence the efficacy of a medication. Common genetic variants include single nucleotide polymorphisms (SNPs), genomic insertions and deletions, as well as copy-number variations (CNVs). SNPs and CNVs both contribute to various phenotypic outcomes and measurements in pharmacogenomics. Pharmacogenomics is the study

of genetic differences in targets, receptors, transporters, and enzymes that metabolise drugs, as well as how these variants interact to generate effects like toxicity or drug response. Moreover, it has identified the elements responsible for inter-individual variances in the expression and functionality of numerous genes, including the influence of microRNA (miRNA), DNA methylation, copy-number alterations, and single-nucleotide variations, both inherited SNPs and somatically acquired SNPs [83], [84]. Furthermore, it can yield favorable impacts on healthcare, such as diminishing the potential for medication-related toxicity, enhancing the efficiency of prescribed drugs, supporting drug discovery and development initiatives, and enhancing the sustainability of the healthcare system [85].

In recent decades, there has been substantial advancement in our comprehension of the genetic alterations associated with tumor growth. These genomic modifications can impact genes responsible for drug-metabolizing enzymes and drug transporters, potentially altering how drugs are processed at the tumor's location [86]. Pharmacogenomics will advance knowledge of the genetic basis of medication response and aid in the development of more beneficial and less hazardous treatments for specific patients in the future. This brief review article's goal was to give readers an overview of modern pharmacogenomic techniques with relation to their current uses and potential future directions in personalised medicine, including the development of cancer-specific monoclonal treatments [87]. The goal is to discover genetic markers with functional significance that affect drug metabolism and the efficacy of drugs within the body. This enables the customization of pharmacological therapy according to an individual's genomic profile. Pharmacogenomics has evolved from examining single candidate genes to comprehensive genome-wide approaches. Both the development of anti-cancer medications and personalized cancer treatment have benefited from the use of proteomics. Oncoproteomics has been used to treat malignancies of the brain, breast, colon, rectum, prostate, and leukaemia, among other organs [88]. Clinicians play a crucial part in the transfer from pharmacogenomics to clinical practise, which appears to be important. Clinicians must possess the knowledge necessary to comprehend and appreciate the justification for prescribing for some genotypes but not for others in order to use genotype-guided therapy [89].

Pharmacogenomics may be able to explain some of the response variability and offer recommendations for the best analgesic drug and dosage. Despite the fascinating potential that pharmacogenomics holds, there are still a lot of obstacles to be solved before it can be used as part of standard clinical practise. Our comprehension remains limited, and there is an insufficient number of rigorous trials and genome-wide association studies, even though there is increasing evidence indicating that genetic variations may be involved in the onset of pain and individual responses to pain relief [90],[91]. Through categorizing individuals based on their genotypes, which are linked to corresponding drug metabolism capabilities and potential side effects, it becomes feasible to investigate and discern the impact of genetic variations on drug responses. Clinical evaluations of drug metabolism, drug-related side effects, and drug choice represent significant objectives in numerous pharmacogenomics applications for patient treatment management and the pharmaceutical industry. These factors are considered pivotal determinants in the success of a novel therapeutic compound, alongside its effectiveness, potency, and safety concerning an individual's genetic profile [92]. Pharmacogenomics aims to direct drug selection in order to maximise the likelihood of benefit and reduce the possibility of toxicity for specific individuals. Additionally, an increased understanding of pharmacogenetic factors might enable the creation of completely novel therapeutic drugs and therapeutic strategies [93].

VIII. CONCLUSION

Particularly in oncology, pharmacogenomic data is a crucial tool for patient classification and the choice of the best medications and dosage schedules. Pharmacogenomics is a promising instrument in the pharmaceutical sector that needs to be used to its full potential. It is a significant development in medical history. Historically, most drugs were developed to target entire populations rather than individual patients.

Pharmacogenomic research is expected to become an integral component of drug discovery and development, resulting in substantial cost reductions, the assurance of clinical trial safety, and a decrease in failure rates. Pharmacogenomics has undergone

futuristic progress, which has cleared the way for the creation of the pharmacoproteomic, pharmacotranscriptomics, and pharmacometabolomic areas. The idea of treating each patient as an individual who is distinct, complex, and fascinating is made possible by these new scientific fields. Finally, it should be noted that using this integrated system to deliver individualised medicines is still a pipe dream in the twenty-first century.

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