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Systematic Review

Recent Advances in Pharmacogenomic Technologies: A Systematic Review

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Abstract

Background: Pharmacogenomics (PGx) uses genetic information to explain differences in patient responses to medicines and to support appropriate drug selection and dosing.

Objective: To review recent advances in pharmacogenomic technologies and examine their relevance to the development and clinical use of personalized medicine.

Methods: A systematic review was undertaken using predefined eligibility criteria. Literature addressing pharmacogenomic technologies, genomic discovery, clinical applications, and implementation was identified and evaluated. Information was extracted on study characteristics, technologies, applications, major findings, and clinical relevance. Findings were synthesized descriptively and thematically in accordance with PRISMA 2020 reporting principles.

Results: The literature demonstrates a progression from individual SNP-based studies and genome-wide association studies (GWAS) toward next-generation sequencing (NGS), regulatory genomics, pharmacometabonomics, bioinformatics, and emerging computational approaches. These methods have expanded the investigation of genetic contributors to drug efficacy, toxicity, and pharmacokinetic variability. Translation into clinical practice, however, remains constrained by challenges in evidence interpretation, population diversity, cost and reimbursement, clinical decision support, clinician education, and equitable access to testing.

Conclusion: Pharmacogenomics is increasingly capable of informing individualized treatment, but advances in technology alone will not guarantee clinical uptake. Meaningful implementation depends on reliable genomic evidence, integration with clinical information, clear interpretation, effective decision support, and healthcare systems capable of supporting testing and follow-up.

Keywords: Pharmacogenomics; Precision Medicine; Personalized Medicine; Genome-Wide Association Studies; Next-Generation Sequencing; Pharmacometabonomics; Bioinformatics; Pharmacogenetic Testing.

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Introduction

Pharmacogenomics (PGx) is an important component of precision medicine. It examines how inherited genetic variation can affect drug pharmacokinetics, pharmacodynamics, therapeutic response, and susceptibility to adverse drug reactions. Variation in genes encoding drug-metabolizing enzymes, transporters, drug targets, and regulatory elements can contribute to clinically important differences in treatment response and toxicity. [1,2]

Pharmacogenomic research has developed substantially over the past several decades. Early studies focused on candidate genes and individual single-nucleotide polymorphisms (SNPs), generally selected because of their established roles in drug metabolism or pharmacology. The subsequent development of high-throughput genotyping enabled genome-wide association studies (GWAS), allowing researchers to investigate variants across the genome that are associated with drug efficacy, toxicity, and pharmacokinetic traits rather than limiting analyses to prespecified genes. [3,4,5,6]

Next-generation sequencing (NGS) further broadened this approach by enabling the detection of both common and rare variants across larger genomic regions. Sequencing platforms differ in read length, accuracy, throughput, coverage, and cost; therefore, the most appropriate platform depends on the research question and intended clinical application. [7,8,9] Attention has also

extended beyond protein-coding regions to regulatory and structural RNA variation. Changes affecting RNA stability, processing, splicing, translation, or microRNA binding may alter the expression of genes involved in drug metabolism, transport, drug targets, and treatment response. These findings have broadened the biological scope of pharmacogenomics beyond conventional coding variants. [10,11,6]

Functional approaches such as pharmacometabonomics provide an additional layer of information by examining metabolic profiles before treatment and relating them to drug metabolism, efficacy, toxicity, or response. This approach complements genomic information and supports a broader systems-biology perspective on treatment variability. [12]

The increasing volume of genomic and clinical information has also heightened the importance of bioinformatics and computational methods. PharmGKB provides a structured resource that links genes, variants, drugs, phenotypes, and supporting evidence, while artificial intelligence (AI) and machine-learning methods are being explored for the integration of multidimensional data and prediction of treatment outcomes. [13,14,15]

Despite these advances, translating pharmacogenomic discoveries into routine clinical use remains challenging. GWAS and sequencing studies can be affected by sample size, phenotype definition, population diversity, replication, functional validation, and interpretation. Implementation is also influenced by testing costs and reimbursement, clinician education, electronic health-record integration, clinical decision support, and equitable access to testing. [16,17,18,19]

This systematic review therefore examined recent developments in pharmacogenomic technologies and their implications for personalized medicine, with particular attention to technological progress, major findings, clinical relevance, and factors influencing translation into practice. [11,2]

Research Methods / Methodology

Study design and reporting: This study was conducted as a systematic review of published literature addressing advances in pharmacogenomic technologies and their clinical implications for personalized medicine.

The review was planned and reported with reference to the PRISMA 2020 statement and its reporting recommendations. [17]

Literature search: As specified in the review protocol, searches were conducted in PubMed, Scopus, and Web of Science using terms related to pharmacogenomics, genomic technologies, genetic variation, drug response, personalized medicine, and clinical application. The protocol specified a 20-year review period.

The available study-selection records identified 934 records: 327 from PubMed, 285 from Scopus, 304 from Web of Science, and 18 through manual searching. After removal of 312 duplicates, 622 records remained for screening. Of these, 510 were excluded on the basis of title and abstract. Full-text reports were then assessed for eligibility where available.

Eligibility Criteria: Eligible publications were peer-reviewed articles addressing pharmacogenomic technologies and their clinical applications. Original research, clinical studies, reviews, and evidence-synthesis publications were considered when they directly contributed to the review objectives. Publications were excluded when they lacked clinical relevance, did not directly focus on pharmacogenomic technologies, or did not provide sufficient information for the planned synthesis.

Study selection and data extraction: The selection process followed the stages of identification, screening, eligibility assessment, and inclusion. Extracted information included publication details, study or article type, population or data source where reported, pharmacogenomic technology, therapeutic area, principal outcomes, major findings, clinical relevance, and reported limitations.

Quality and risk-of-bias assessment: Because the included literature was heterogeneous, methodological quality was considered according to study type rather than through the application of a single tool to every publication.

For primary GWAS and other empirical studies, attention was given to sampling, phenotype definition, statistical methods, replication, and generalizability. Reviews and resource papers were considered in terms of methodological transparency, evidence synthesis, and limitations in interpretation.

Data synthesis: A quantitative meta-analysis was not undertaken because the studies differed substantially in design, technology, population, and outcome measures. Findings were therefore synthesized descriptively and thematically across SNP-based approaches, GWAS, NGS, regulatory genomics, pharmacometabonomics, bioinformatics, AI-assisted approaches, clinical implementation, and barriers to translation.

Results

Study selection: The documented search and screening process is shown in Figure 1. The available review records contain a discrepancy

between the overall search counts and the 24 publications used for the detailed evidence synthesis. To avoid suggesting a level of precision that the available records do not support, the results section focuses on these 24 core publications while retaining the methodological and implementation references needed to interpret the review.

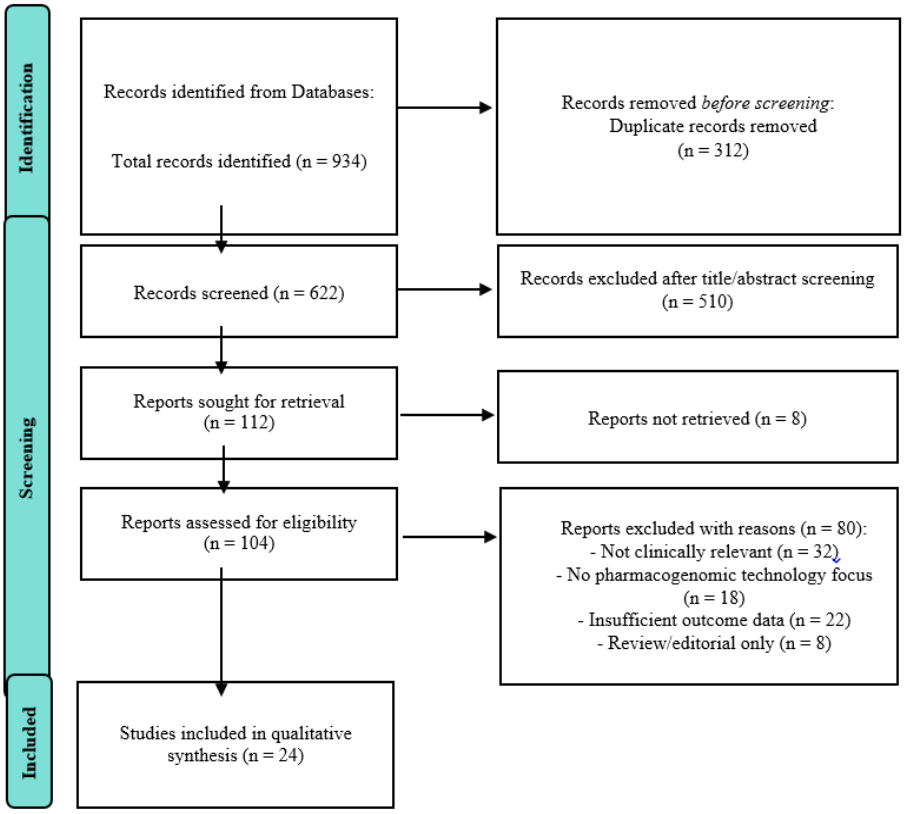

Figure 1: PRISMA-style flow diagram of the study-selection process based on the available review records.

Characteristics of the included publications: The 24 core publications covered a broad range of pharmacogenomic research, including SNP-based approaches and GWAS, NGS, regulatory and non-coding RNA variation, pharmacometabonomics, bioinformatics, AI, and clinical implementation. Together, they describe the field's progression from genetic discovery toward increasingly integrated approaches to individualized treatment, as shown in Table 1.

Table 1: Overview of Evidence Types, Pharmacogenomic Technologies, and Principal Contributions of the Included Publications

No.	Publication	Evidence type	Technology/focus	Principal contribution
1	Pirmohamed (2023) Pirmohamed, 2023	Review	Clinical PGx / future	Transition from discovery to genotype-guided prescribing; future rare variants, WGS and multi-omics.
2	Relling & Evans (2015) Relling & Evans, 2015	Review	Clinical PGx	Clinical actionability of gene–drug relationships and implementation.
3	Motsinger-Reif et al. (2013) Motsinger-Reif et al., 2013	Review	GWAS	GWAS successes, phenotyping, replication and collaborative cohorts.
4	Alwi (2005) Alwi, 2005	Review	SNPs	SNP utility and importance of population diversity, LD, phenotype and power.
5	Harper & Topol (2012) Harper & Topol, 2012	Review	GWAS / sequencing	Clinical practice, drug development and translation gap.
6	Biernacka et al. (2015) Biernacka et	Primary GWAS	SSRI response	Difficulty identifying genetic predictors of antidepressant response;

	al., 2015			need larger cohorts.
7	Thorn et al. (2010) Thorn et al., 2010	Resource review	PharmGKB	Structured pharmacogenomic knowledge resource.
8	Schork (2019) Schork, 2019	Conceptual review	AI	Multidimensional integration of genomic, clinical and biomedical data.
9	Everett et al. (2013) Everett et al., 2013	Review	Pharmacometabonomics	Metabolic phenotyping as a complement to pharmacogenomics.
10	Satam et al. (2023) Satam et al., 2023	Technology review	NGS	Modern high-throughput sequencing platforms and applications.
11	Liu et al. (2012) Liu et al., 2012	Technology comparison	NGS	Comparison of sequencing systems by throughput, read length, accuracy and cost.
12	Manolio (2010) Manolio, 2010	Methodological review	GWAS	Effect sizes, missing heritability, rare/structural variants and prediction.
13	McCarthy et al. (2008) McCarthy et al., 2008	Methodological review	GWAS	Sample size, phenotyping, population structure and multiple testing.
14	Hardy & Singleton (2009) Hardy & Singleton, 2009	Methodological review	GWAS	Association methodology and distinction between association and causality.
15	Wang et al. (2005) Wang et al., 2005	Methodological review	GWAS / LD	LD, power and population stratification.
16	Sadee et al. (2011) Sadee et al., 2011	Review	RNA pharmacogenomics	Regulatory and structural RNA variation.
17	Yang et al. (2016) Yang et al., 2016	Primary GWAS	Methadone PK	rs17180299 and CYP2B6, SPON1 and GSG1L haplotypes.
18	Manikandan & Munirajan (2014) Manikandan & Munirajan, 2014	Review	miRNA-binding SNPs	Regulatory SNPs affecting gene expression and response.
19	Hicks et al. (2019) Hicks et al., 2019	Implementation review	Clinical pharmacy PGx	Pharmacist roles, pre-emptive testing and implementation barriers.
20	Ahmed et al. (2016) Ahmed et al., 2016	Review	Enzymes / transporters	CYP, UGT and ABC/SLC variation and drug exposure.
21	Primorac et al. (2020) Primorac et al., 2020	Review	Big Data / precision medicine	Integration of genomics, NGS, bioinformatics, AI and clinical data.
22	Ingelman-Sundberg & Pirmohamed (2024) Ingelman-Sundberg & Pirmohamed, 2024	Review	Cardiovascular PGx	Pre-treatment PGx and drug–gene-specific evidence.
23	Klein et al. (2017) Klein et al., 2017	Implementation review	Clinical implementation	Education, cost, reimbursement, EHRs and decision support.
24	McInnes et al. (2021) McInnes et al., 2021	Review	Pharmacogenomic GWAS	Drug exposure, phenotype heterogeneity, diversity and functional evidence.

Methodological quality and risk-of-bias assessment: The methodological quality of the included evidence was assessed in relation to study design, methodological purpose, and the type of evidence contributed to the review. Because the evidence base was heterogeneous and included narrative and systematic reviews, methodological and technology-focused publications, a pharmacogenomic knowledge-resource paper, implementation-focused literature, and primary GWAS studies, application of a single risk-of-bias instrument to all publications was considered inappropriate.

Accordingly, the assessment focused on study-specific methodological limitations, potential sources of bias, external validity, and maturity of the evidence rather than assigning a single standardized risk-of-bias score across all study designs. The primary GWAS studies were principally limited by sample size, phenotype definition and complexity, statistical power, population structure, replication, and

generalizability. These limitations are particularly relevant to pharmacogenomic research because drug-response phenotypes may be influenced by multiple genetic and non-genetic factors, while population-specific allele frequencies and linkage disequilibrium can affect the reproducibility and transferability of findings. The International SSRI Pharmacogenomics Consortium study, for example, demonstrated the difficulty of identifying robust genetic predictors for complex antidepressant response phenotypes and highlighted the importance of adequately powered cohorts, standardized phenotyping, and independent replication. Similarly, the methadone pharmacogenomic study provided evidence for associations involving pharmacokinetic phenotypes but requires independent replication and functional validation before the findings can be considered clinically established.

For reviews and perspective articles, the principal methodological considerations were the breadth and transparency of evidence selection, potential selection or reporting bias, and the distinction between established clinical evidence and emerging applications. Foundational methodological publications were considered valuable for understanding the development of pharmacogenomic approaches but were interpreted in the context of their publication date and the subsequent evolution of genomic technologies. Technology-focused publications were similarly considered susceptible to temporal limitations because sequencing platforms, analytical approaches, and standards for clinical interpretation have evolved substantially over time.

Resource and implementation publications were assessed primarily according to the relevance and maturity of the evidence available at the time of publication. Pharmacogenomic knowledge resources provide important evidence-curation and interpretation infrastructure, but their content necessarily reflects the evidence available when the resource or publication was developed. Implementation studies and reviews may also be influenced by healthcare-system characteristics, including availability of testing, reimbursement, electronic health-record integration, clinical decision support, clinician education, and institutional infrastructure.

Overall, the included literature demonstrated low to moderate methodological concern for most publications, with greater concern for studies involving complex phenotypes, limited sample sizes, exploratory regulatory or non-coding variants, emerging computational approaches, and clinical applications for which prospective validation remains limited.

Importantly, these judgments represent methodological and applicability concerns rather than formal quantitative risk-of-bias scores. The evidence should therefore be interpreted according to study design and the maturity of the specific gene–drug, technology, or implementation question addressed, as shown in Table 2.

Table 2: Risk-of-Bias Assessment and Key Methodological Limitations of the Included Publications

No.	Publication	Overall concern	Key limitation
1	Pirmohamed 2023 Pirmohamed, 2023	Low–moderate	Broad review; future applications require validation.
2	Relling & Evans 2015 Relling & Evans, 2015	Low–moderate	Clinical utility varies by gene–drug pair.
3	Motsinger-Reif 2013 Motsinger-Reif et al., 2013	Low–moderate	Heterogeneous GWAS evidence.
4	Alwi 2005 Alwi, 2005	Moderate	Foundational evidence; population heterogeneity.
5	Harper & Topol 2012 Harper & Topol, 2012	Moderate	Perspective/review; translation remains variable.
6	Biernacka 2015 Biernacka et al., 2015	Moderate	Complex phenotype, limited power and need for replication.
7	Thorn 2010 Thorn et al., 2010	Low–moderate	Resource content reflects evidence available at the time.
8	Schork 2019 Schork, 2019	Moderate	Conceptual AI review; validation required.
9	Everett 2013 Everett et al., 2013	Moderate	Emerging methodology; standardization needed.
10	Satam 2023 Satam et al., 2023	Low–moderate	Platform performance varies by application.
11	Liu 2012 Liu et al., 2012	Low–moderate	Technology landscape has evolved.
12	Manolio 2010 Manolio, 2010	Low–moderate	Small effect sizes may limit prediction.
13	McCarthy 2008 McCarthy et al., 2008	Low–moderate	Foundational methodological evidence.
14	Hardy & Singleton 2009 Hardy & Singleton, 2009	Low–moderate	Association does not establish causality.

15	Wang 2005 Wang et al., 2005	Low–moderate	Foundational methodological evidence.
16	Sadee 2011 Sadee et al., 2011	Moderate	Regulatory RNA actionability remains developing.
17	Yang 2016 Yang et al., 2016	Moderate	Single pharmacokinetic cohort; replication required.
18	Manikandan 2014 Manikandan & Munirajan, 2014	Moderate	Regulatory SNP evidence remains exploratory.
19	Hicks 2019 Hicks et al., 2019	Low–moderate	Implementation depends on healthcare infrastructure.
20	Ahmed 2016 Ahmed et al., 2016	Low–moderate	Heterogeneous gene–drug evidence.
21	Primorac 2020 Primorac et al., 2020	Moderate	Broad perspective; evidence maturity varies.
22	Ingelman-Sundberg 2024 Ingelman-Sundberg & Pirmohamed, 2024	Low–moderate	Evidence differs among gene–drug pairs.
23	Klein 2017 Klein et al., 2017	Low–moderate	Barriers are system-specific.
24	McInnes 2021 McInnes et al., 2021	Low–moderate	Sample size, phenotype and diversity remain limiting.

Technology-wise findings: SNP-based approaches remain an important foundation of pharmacogenomic research. The usefulness of individual variants or variant panels depends on factors such as allele frequency, linkage disequilibrium, population structure, phenotype definition, and statistical power. These factors influence the ability to detect relevant associations as well as the extent to which findings can be generalized across populations. [7,5,6]

GWAS extended pharmacogenomic discovery beyond predefined candidate genes and enabled investigators to search for loci associated with drug efficacy, toxicity, and pharmacokinetic traits. In this setting, accurate measurement of drug exposure and treatment outcomes is particularly important because poorly defined phenotypes can reduce the ability to detect meaningful associations. [3,20,18,19]

The International SSRI Pharmacogenomics Consortium study illustrates the difficulty of identifying genetic predictors of complex treatment response. Its primary analysis did not identify a conventional genome-wide significant association, emphasizing the need for adequately powered cohorts, consistent phenotyping, replication, and collaborative studies. [21]

A pharmacogenomic study of methadone identified rs17180299 and several haplotypes involving CYP2B6, SPON1, and GSG1L that were associated with plasma concentrations of the R- and S-enantiomers. The findings demonstrate the potential of GWAS to identify determinants of pharmacokinetic variability, while also highlighting the need for independent replication and functional validation before such associations are used clinically. [22]

NGS has broadened pharmacogenomic investigation by allowing multiple genes and a wider range of variants to be assessed together. Sequencing platforms differ in read length, accuracy, throughput, coverage, and cost; consequently, platform selection should be guided by the intended research or clinical application. [8,9]

Regulatory and non-coding variation is an expanding area of pharmacogenomic research. Variants affecting structural RNA or microRNA-binding sites may alter gene expression and, in turn, influence drug metabolism, transport, pharmacological targets, and treatment response. These observations suggest that clinically relevant variation may extend beyond conventional protein-coding regions. [10,6]

Pharmacometabonomics adds functional information by examining metabolic profiles before treatment and relating them to drug metabolism, efficacy, toxicity, and response. The approach may complement genomic data, but analytical standardization, reproducibility, and clinical validation remain important requirements for broader use. [12]

Bioinformatics and evidence-curation systems are increasingly important for converting genomic findings into information that can be interpreted clinically. PharmGKB links genes, variants, drugs, phenotypes, and supporting evidence, while evidence-based frameworks can improve the consistency and transparency of pharmacogenomic interpretation. [23,15]

AI and machine-learning methods may help integrate genomic information with clinical, biochemical, environmental, and other patient-level data to improve prediction of treatment response and adverse outcomes. Their use should

nevertheless be judged by more than predictive accuracy; external validation, clinical benefit, and performance in real-world settings are also needed before routine clinical adoption. [13,14]

The implementation literature suggests that pharmacogenomic testing is most useful when results can lead to a specific change in medication choice, dose, or monitoring. Clinical pharmacists can support implementation through patient identification, interpretation of results, therapeutic recommendations, education, and follow-up. Integration with electronic health records and clinical decision-support systems is also important. [16,24,25,18]

Across the reviewed literature, the main clinical aims of pharmacogenomics were to improve drug selection and dosing, explain variation in treatment response, reduce adverse drug reactions, and strengthen medication safety. Evidence for cost-effectiveness and broader clinical utility was less consistent and depended on the gene–drug pair, clinical setting, population, and implementation strategy.

Discussion

This review shows how pharmacogenomics has moved from the study of individual variants toward broader genomic, molecular, computational, and clinically integrated approaches. The important change is not simply that more variants can be measured; it is that validated genomic information can increasingly be connected with other clinical and biological data to support treatment decisions. [11,2,5]

Evolution from candidate variants to genome-wide analysis: SNP-based studies provided an important foundation for identifying genetic determinants of drug response. However, candidate-gene approaches depend on prior assumptions about relevant pathways and genes. Differences in allele frequencies and linkage disequilibrium between populations, as well as differences in sample size and phenotype definition, can affect reproducibility and generalizability. [7,5,6]

GWAS made it possible to investigate drug-response phenotypes across the genome without restricting the analysis to prespecified genes. Their interpretation still depends on adequate sample size, careful phenotyping, control of population structure, correction for multiple testing, independent replication, and functional follow-up. These requirements are especially important for complex traits in which individual variants may have modest effects. [21,3,20,18,19]

Increasing importance of NGS: NGS has increased the ability to characterize pharmacogenomic variation by examining multiple genes and detecting a broader range of variants in a single workflow. It may help identify rare or previously unrecognized variants, but the larger number of findings also creates a greater need for reliable annotation and interpretation. Sequencing advances therefore need to be accompanied by robust evidence resources and clinically meaningful interpretation. [8,2,9]

Pharmacogenomics beyond coding variants: Recognition of regulatory and non-coding variation has widened the biological scope of pharmacogenomics. Variants affecting regulatory RNA and microRNA-binding sites may alter gene expression and influence drug metabolism, transport, drug targets, and treatment response. These mechanisms are biologically plausible, but their use as prescribing biomarkers requires further functional and clinical validation. 10,6

Multi-omics and pharmacometabonomics: Genomic variation is only one part of the biology underlying treatment response. Pharmacometabonomics and other multi-omics approaches can provide complementary information about functional biological states that are not fully captured by inherited DNA variation. Their integration with genomic data may improve patient stratification, although analytical standardization, reproducibility, and prospective clinical validation remain important barriers. [12]

Bioinformatics and AI: The scale and complexity of pharmacogenomic data make bioinformatics infrastructure essential for organizing, interpreting, and translating genomic findings. Resources such as PharmGKB and evidence-based frameworks bring together information on genes, variants, drugs, phenotypes, and supporting evidence and can help make interpretation more consistent. [23,15] AI and machine-learning approaches may further support pharmacogenomic prediction by combining genomic data with clinical, biochemical, environmental, and other patient-level information. Predictive performance alone, however, does not demonstrate clinical benefit. Transparent methods, external validation, meaningful clinical outcomes, and integration into healthcare workflows are necessary before AI-based approaches are ready for routine use. [13,14]

Clinical translation and implementation: Pharmacogenomic information becomes clinically useful when it can change a prescribing decision, such as drug selection, dose, monitoring, or avoidance. The value of a genetic finding therefore depends not only on the strength of the association but also on whether the result can be interpreted reliably and incorporated into patient care. [24,2,5]

Evidence-based resources such as CPIC and PharmGKB help connect genetic findings with prescribing decisions by linking genomic results to drug-specific recommendations and levels of supporting evidence. Standardized evidence appraisal is important because the strength and clinical relevance of evidence differ across gene–drug pairs. [16,18,15]

Population diversity and equity: Population diversity matters at both the research and implementation stages. Differences in allele frequencies and linkage disequilibrium can influence the detection of associations and the performance of pharmacogenomic tests across populations. Underrepresentation of particular populations may limit generalizability, making broader inclusion essential for reliable and equitable precision medicine. [3,10,5,6]

Major implementation barriers: Several practical barriers continue to slow routine implementation. These include limited clinician education, cost and reimbursement issues, difficulties in evidence interpretation, access to testing, electronic health-record integration, clinical decision support, privacy and data-governance concerns, and unequal access to pharmacogenomic services. [24,25,17]

Successful implementation requires more than the availability of a test. Testing, quality assurance, evidence interpretation, clinical decision support, prescribing, and follow-up need to function as a connected clinical pathway so that genomic information can lead to measurable improvements in patient care. [16,18]

Strengths and limitations: A major strength of this review is its broad scope, covering genomic discovery, sequencing, regulatory genomics, pharmacometabonomics, bioinformatics, AI, and clinical implementation. This provides a useful overview of the field's progression from genetic discovery toward clinical application. The main limitation is the heterogeneity of the evidence base, which prevents meaningful quantitative pooling.

Conclusion

Pharmacogenomic research has progressed from individual genetic variants to genome-wide methods, sequencing, regulatory genomics, multi-omics, bioinformatics, and computational approaches.

These developments have improved the ability to investigate genetic contributors to drug pharmacokinetics, therapeutic response, and adverse drug reactions and have strengthened the scientific basis for individualized treatment. [2,5,9]

Technology alone, however, will not ensure clinical adoption. Translation depends on adequately powered and diverse studies, reproducible associations, functional validation, consistent evidence interpretation, clinical decision support, and integration with established healthcare workflows. [16,18,15]

Future practice is likely to combine genomic sequencing with functional molecular phenotyping and increasingly sophisticated computational methods. Implementation should remain evidence-driven, with priority given to clinically actionable gene–drug relationships, demonstrable patient benefit, appropriate assessment of cost-effectiveness, and equitable access to testing and interpretation. [12,11,2,15]

Overall, pharmacogenomics is becoming an important component of precision medicine. Continued integration of validated genomic evidence with clinical and functional information may help translate advances in genomic technology into practical and clinically meaningful approaches to individualized drug therapy. [16,17,2]

Declarations

Ethics Approval and Consent to Participate: Not applicable. This study was based exclusively on previously published literature and did not involve the recruitment of human participants or the collection of primary human data.

Availability of Data and Materials: The evidence included in this systematic review was obtained from previously published literature. Extracted study information and supplementary review materials are available from the corresponding author upon reasonable request.

Authors' Contributions: All authors contributed to the conception and design of the study. The literature review, data extraction, analysis, interpretation of findings, and preparation of the manuscript were carried out by the authors. All authors critically reviewed the manuscript and approved the final version for publication.

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