Narrative Review Article

Clinical Applications and Challenges of Personalized Medicine in Cardiovascular Disease

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ABSTRACT

Personalized medicine is an approach to healthcare that tailors treatment strategies to a patient's genetic makeup. It can be applied to the prevention, diagnosis, and treatment of cardiovascular disease (CVD). Its integration into cardiovascular disease management is particularly important, as CVD remains one of the leading causes of death worldwide. Through pharmacogenomics, healthcare providers can identify early indicators of CVDs, diagnose specific CVDs, and individualize therapies for patients. However, privacy risks complicate the implementation of personalized medicine, as the collection and storage of genetic information can potentially expose patients to misuse, security risks, and genetic discrimination. Ethical concerns about healthcare equity also arise, due to high costs and lack of genetic research on diverse populations. Legal protections such as the Genetic Information Nondiscrimination Act highlight progress in navigating issues related to personalized medicine, but future measures are necessary to support pharmacogenomics' wider implementation. This review analyzes the applications of personalized medicine in cardiovascular care and addresses the privacy and ethical challenges that accompany pharmacogenomic advances.

Keywords: Pharmacogenomics; personalized medicine; cardiovascular disease; ethics; privacy; anticoagulants; dyslipidemias; arrhythmias

INTRODUCTION

Personalized medicine represents a shift from the traditional "one-size-fits-all" approach in healthcare (1). Through pharmacogenomics, which links genetic variation to treatment responses, personalized medicine is increasingly being used in cardiovascular disease treatment. Cardiovascular disease (CVD) refers to a broad spectrum of disorders that affect the circulatory system, including both the heart and blood vessels (2). Since 1975, cardiovascular diseases have consistently ranked among the top two causes of death in the United States (3). Heart disease and strokes together cause more deaths than all cancers and accidental deaths combined (4). Furthermore, CVD-related deaths are continuing to increase. In 2022, cardiovascular disease (CVD) was responsible for 931,652 deaths in the United States — more than 10,000 higher than in 2021 (4). By 2035, more than 130 million U.S. adults (41.5% of the US population) are estimated to have CVD (3), and total healthcare costs of CVD are expected to reach $1.1 trillion (3). These trends underscore the long-standing impact of cardiovascular disease and the urgent need for more effective prevention and treatment strategies.

Given this significant and prevalent issue of

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cardiovascular disease, early identification of at-risk individuals is critical, and genetic screening has emerged as a promising approach. Genetic screening may identify individuals predisposed to genetic diseases, enabling earlier intervention, as genetic variations among patients can have a significant role in identifying their risk of developing CVDs. For instance, genetic testing helps with risk stratification in inherited lipid disorders, which are major risk factors for CVD (5). Familial Hypercholesterolemia (FH), which disrupts LDL cholesterol metabolism, is strongly associated with premature coronary artery disease. By early identification through genetic testing, FH can be treated to lower LDL levels and reduce cardiovascular risk. Furthermore, by identifying genetic markers that predict therapeutic effectiveness or risk of adverse effects, clinicians can also potentially optimize treatment plans for CVD patients and improve overall health outcomes. Genetic variations may play a significant role in determining patients' response to therapies such as anticoagulants, statins, beta-blockers, and antihypertensives. For example, genetic variation can affect how quickly an anticoagulant is metabolized, altering its safety and effectiveness in those individuals. Therefore, testing can guide safe and effective treatment by accounting for gene variants in patients, reducing the risk of bleeding complications (6). As incidence of cardiovascular disease continues to rise, the integration of personalized medicine into cardiovascular treatment has the potential to improve diagnostic accuracy, reduce side effects, and improve overall patient outcomes.

Despite its promise, however, the utilization of personalized medicine in cardiovascular care raises important privacy and ethical concerns. The collection and use of genetic data require careful protection to prevent misuse by third parties, such as insurers and employers. Patients may worry about how their genetic information will be stored, who will have access to their data, and whether it could lead to potential genetic discrimination (7). Additionally, ethical concerns arise regarding informed consent, and the exacerbation of existing disparities in healthcare, due to factors such as the high cost of genetic testing (8). Addressing these concerns is essential to realizing the benefits of personalized medicine without compromising patients' privacy or widening existing health disparities. This paper examines how personalized medicine can transform cardiovascular care, while also addressing the privacy, ethical, and accessibility challenges that currently limit its widespread adoption.

PERSONALIZED MEDICINE IN CARDIOVASCULAR DISEASE

Personalized medicine has the capability to advance the prevention and treatment of CVDs. Its applications include treating dyslipidemias (abnormal lipid levels), arrhythmias (abnormal heart rhythm), and the safe administration of anticoagulants (medication used to prevent and treat blood clots). By integrating genetic information into clinical practice, physicians are able to more accurately predict disease risk, refine diagnoses, and tailor therapies to patients.

Dyslipidemias

A major application of personalized medicine is disease prevention. Obtaining genetic testing is useful for risk stratification for CVD because genetic code can be assessed from birth without being affected by temporary environmental factors (5). For example, an inherited genetic condition, Familial hypercholesterolemia (FH), causes high levels of low-density lipoprotein (LDL) cholesterol in the blood (9). It is one of the most common Mendelian dyslipidemias and is caused by mutations in the LDL receptor or related genes, disrupting LDL cholesterol metabolism. FH is strongly associated with premature coronary artery disease due to increased blood cholesterol levels (5,10). Early diagnosis through genetic screening is critical, as LDL-lowering therapies such as statins can substantially reduce cardiovascular risk. In the YOUNG-MI registry, in a cohort of 1,996 adults with a median age of 45 years, 63.3% of FH patients were discharged on high-intensity statins, and after one year, FH patients showed a large LDL-C reduction (-44.4%), highlighting the effectiveness of statins (11). Additionally, because FH is inherited in an autosomal dominant pattern, family members of affected individuals are also at risk, and can be identified through cascade screening, the process of contacting relatives of patients in order to identify, inform, and manage others at risk (12). A Markov model with a 10-year time horizon estimated that screening for familial hypercholesterolemia (FH) could reduce the 10-year incidence of coronary heart disease (a type of CVD) from 50% to 25% among individuals with FH (13), underscoring the effectiveness and importance of cascade screening in preventing cardiovascular disease.

In addition to disease prevention, genetic testing can be used to diagnose genetic disorders and variations. For instance, genetic testing can be used to distinguish FH from similar lipid disorders, such as sitosterolemia.

Sitosterolemia is an inherited metabolic disorder in which patients have increased levels of plant sterols, such as sitosterol (13). Its clinical manifestations are similar to FH, often leading to misdiagnosis. However, sitosterolemia is caused by genetic mutations in ATP-binding cassette subfamily G member 5 (ABCG5) or member 8 (ABCG8), which are not found in FH (13). In cases such as this, personalized medicine and genetic screening are especially helpful to ensure accurate diagnosis, and consequently, to prescribe the most effective therapy. Patients with sitosterolemia were found to usually not respond to statins, which inhibit cholesterol production primarily in the liver (14), because HMG CoA reductase activity is already maximally inhibited (15). Instead, ezetimibe is recommended for treatment as it inhibits cholesterol absorption in the intestine, has been shown to lower LDL cholesterol by 13–20%, and is particularly effective in reducing elevated plant sterol levels in patients with sitosterolemia (16). Early identification and precise diagnosis of dyslipidemias such as familial hypercholesterolemia and sitosterolemia illustrate how genetic testing can transform cardiovascular prevention and treatment. Building on this, advances in genomics have provided insights into how specific gene mutations can guide risk stratification and management of cardiovascular diseases.

Arrhythmias

Personalized medicine is also important in the risk stratification and management of arrhythmias, including long QT syndrome (LQTS), short QT syndrome, and arrhythmogenic right ventricular cardiomyopathy. Of these, LQTS has become a widely established model for applying genomics in cardiovascular care, as strong data exists showing associations between genotype and disease phenotype (5). Long QT syndrome is a cardiovascular disorder characterized by an abnormality in cardiac repolarization, leading to a prolonged QT interval and T-wave irregularities (17). LQTS can be categorized into congenital or acquired types. Congenital LQTS affects about 1 in 2,000 live births, and genetic testing identifies mutations in up to 80% of affected patients. Over 17 genetic forms have been identified, with LQT1, LQT2, and LQT3 being the most common (5). LQT1 is caused by mutations in the KCNQ1 gene, which encodes the α-subunit of the cardiac IKs potassium channel (18). LQT2 is caused by loss-of-function mutations in KCNH2, which encodes the Kv11.1 subunit of the cardiac IKr potassium channel (19). LQT3 is caused by mutations in SCN5A, which encodes the α-subunit of the cardiac sodium channel Nav1.5 (20). Patients with LQTS are at risk of life-threatening cardiac events, such as syncope, cardiac arrest, or sudden cardiac death (21). These subtypes differ in triggers of cardiac events. For instance, emotional or physical stress often precipitates events in LQT1, emotion or sudden noises in LQT2, and rest or sleep in LQT3. Screening is important for these conditions, as they can often be asymptomatic until a major cardiac event.

LQTS therapies are tailored to each genotype. β-blockers are the primary therapy for LQTS patients, reducing cardiac events by over 95% in LQT1 patients, 75% in LQT2 patients, and 60% in LQT3 patients. Moreover, not all β-blockers have equal effectiveness across LQTS subtypes. Nadolol has been shown to significantly reduce risk in both LQT1 and LQT2, whereas atenolol and propranolol are effective only in LQT1. Metoprolol showed little or no reduction in either genotype. Beyond β-blockers, other genotype-specific strategies can be used to prevent cardiac events. For example, increased extracellular potassium concentrate can shorten the QT interval in LQT2 patients, and sodium channel blockers such as mexiletine have been found to normalize the QTc interval in patients with LQT3. Through this approach, personalized medicine reduces the risk of sudden cardiac events and highlights the importance of matching therapies to individual genetic profiles (5). As understanding of genetics in relation to cardiovascular disease has improved, personalized medicine has expanded from identifying predisposition and prevention to tailoring therapies. Clinicians have been able to move beyond risk assessment to using genetic information to guide drug dosing and optimize therapies.

Administration of Anticoagulants

Personalized medicine has further advanced cardiovascular treatment by guiding drug dosing based on a patient's genotype. An example of this is the use of warfarin, an anticoagulant that was previously the most commonly prescribed to treat and prevent thromboembolic events in conditions such as atrial fibrillation (22), a common type of cardiac arrhythmia in which the heart beats too fast (23). Warfarin is difficult to manage because its dosing varies widely between patients, and incorrect dosing is a major cause of adverse drug events (24). Genetic differences account for much of this variability. Variants in CYP2C9, the

enzyme responsible for metabolizing warfarin, can reduce enzymatic activity by altering amino acids and reducing its metabolic capacity. This slows the clearance of warfarin from the body, resulting in lower dose requirements (25). CYP2C9*3 variant causes a major reduction in warfarin metabolism, and CYP2C9*2 causes a minor reduction (26). Additionally, VKORC1 is the target enzyme inhibited by warfarin, and VKORC1 allows for warfarin to prevent the production of vitamin K-dependent coagulation factors (6). However, the VKORC1*2 allele increases sensitivity to the drug, and patients with this allele require a reduced dose. These explain most of the dose variation (27). Furthermore, a third gene, CYP4F2, which functions as a vitamin K_1 oxidase, also influences vitamin K metabolism, causing an altered warfarin dose to be needed (28). Incorporating these genetic factors into dosing can help reduce complications and improve outcomes. The clinical benefit of genotype-guided dosing was shown in the Genetic Informatics Trial (GIFT) of Warfarin to Prevent Deep Vein Thrombosis, a multicenter randomized clinical trial of patients initiating warfarin. The study found that patients whose warfarin doses were tailored using genetic information had fewer bleeding events, more stable INRs, and better overall outcomes compared to standard dosing (29).

More commonly used anticoagulants are direct oral anticoagulants (DOACs). Having been endorsed by the American Heart Association, American College of Cardiology, and the European Society of Cardiology, DOACs have mostly displaced warfarin as the oral anticoagulant of choice to reduce the risk of venous thromboembolism and the risk of stroke in patients with atrial fibrillation (30). Compared to Warfarin, DOACs have improved safety profiles and lack of monitoring requirements. However, despite this improved safety, similar to warfarin, DOAC-induced bleeding remains a major concern for patients and physicians. Variability in DOAC response is common, and can lead to serious incidents, such as thrombosis and hemorrhage. These variabilities can be attributed to numerous factors, including genetics (30). While the role of pharmacogenomics in warfarin therapy is well established, evidence for genetic influence on DOACs is still emerging. Recent studies have investigated whether variants in genes such as ABCB1, CES1, and ABCG2 affect outcomes in patients on commonly prescribed DOACs, including dabigatran, apixaban, and rivaroxaban (31). For rivaroxaban, the ABCB1 c.3435C>T variant and the 1236T-2677T-3435T haplotype were associated with a lower risk of thromboembolic events (31). However, the 1236C-2677G-3435C and 1236T-2677G-3435C haplotypes were associated with a higher thromboembolic risk; therefore, these patients should be cautious when taking rivaroxaban. In apixaban users, the ABCB1 c.2482-2236G>A variant was linked to a decreased bleeding risk, while signals for ABCG2 c.421C>A did not reach statistical significance (31). For dabigatran, earlier reports of the CES1 rs2244613 variant lowering bleeding risk could not be confirmed, though a possible association was seen with CES1 c.257+885T>C (31). Overall, these findings suggest that genetic variation may modestly influence DOAC response, particularly through ABCB1-mediated P-glycoprotein transport, but results remain inconsistent and require validation in larger, more diverse cohorts (31).

Evidence for pharmacogenomic guidance in warfarin therapy is more extensive, with defined genetic variants (*CYP2C9, VKORC1,* and *CYP4F2*) explaining much of the variability in dosing and improving safety and effectiveness when incorporated clinically. In contrast, pharmacogenomic evidence for DOACs remains limited. Although variants in *ABCB1, CES1,* and *ABCG2* have been associated with differences in bleeding or thromboembolic risk, findings are inconsistent and require further validation in larger, more diverse populations.

However, challenges remain, including privacy and security concerns, the cost of genetic testing, and the underrepresentation of diverse populations in clinical trials. Despite these limitations, pharmacogenomics has significant potential to expand and address these gaps, and become an essential tool for disease prevention and safer, more effective treatment tailored to each individual patient's genetic makeup (32) (Table 1).

PRIVACY CONCERNS

The rise of electronic health records (EHRs) has fundamentally changed how patient data is stored, shared, and applied in modern healthcare. The Federal Health IT Strategic Plan and financial incentives provided through the HITECH Act in 2009 have fueled the growth in EHR usage. As of 2021, 88% of U.S. office-based physicians had adopted an EHR and 78% had adopted a certified EHR. Since 2008, office-based physician adoption of any EHR has more than doubled from 42% to 88% and this growth in EHR usage has been a consistent trend (33). However, EHRs can pose

Table 1. Summary of Anticoagulant Pharmacogenomic Findings

Drug	Gene(s)	Variant(s)	Clinical Impact	Reference
Warfarin	CYP2C9 VKORC1 CYP4F2	CYP2C9*2/*3 VKORC1*2 CYP4F2	CYP2C9*2 causes a minor reduction in warfarin metabolism, the CYP2C9*3 variant causes a major reduction in warfarin metabolism, both resulting in a higher required dose of Warfarin. VKORC1*2 allele increases sensitivity to the drug, and patients with this allele require a reduced dose. CYP4F2 influences vitamin K metabolism, causing an altered warfarin dose to be needed.	(24, 25)
Rivaroxaban	ABCB1 CES1 ABCG2	ABCB1 c.3435C>T 1236T-2677T-3435T haplotype 1236C-2677G-3435C haplotype 1236T-2677G-3435C haplotype	ABCB1 c.3435C>T variant and the 1236T-2677T-3435T haplotype were associated with a lower risk of thromboembolic events. The 1236C-2677G-3435C and 1236T-2677G-3435C haplotypes were associated with a higher thromboembolic risk (patients with these should be cautious when taking rivaroxaban).	(30)
Apixaban	ABCB1 CES1 ABCG2	ABCB1 c.2482-2236G>A ABCG2 c.421C>A	ABCB1 c.2482-2236G>A variant was linked to a decreased bleeding risk. Signals for ABCG2 c.421C>A did not reach statistical significance.	(30)
Dabigatran	ABCB1 CES1 ABCG2	CES1 rs2244613 CES1 c.257+885T>C	CES1 rs2244613 variant lowering bleeding risk could not be confirmed. A possible association with lower bleeding risk was seen with CES1 c.257+885T>C.	(30)

serious security concerns that may impact the privacy of patients' health information.

Protecting genetic and health data is critical for both individuals and society. Improper disclosure of genetic information can expose details about inherited traits, disease predispositions, and carrier status for genetic conditions. Privacy concerns may also discourage individuals from sharing relevant details with their providers or from seeking care altogether, which can weaken the quality of medical treatment they receive (34). Furthermore, as EHRs grow, large amounts of medical records could become accessible to both authorized and unauthorized users. They can be potentially vulnerable to security breaches that may cause patients to have concerns about the privacy of their personal information (35). These concerns are especially significant when it comes to genetic data, which not only holds information about an individual's current health, but also carries insights into future disease risks and traits. Unlike other medical records, genetic information is permanent and uniquely identifiable and can also imply biological relatives who share similar variants.

The real-world implications of these security vulnerabilities are underscored by recent events, such as the cybersecurity risk involving Illumina Inc., a leading genomics company that manufactures and sells genomic sequencing systems. In 2025, the company agreed to a $9.8 million settlement with the U.S. Department of Justice, following allegations that the company sold systems with unresolved cybersecurity vulnerabilities to federal agencies and misrepresented compliance with

international security standards, violating the False Claims Act. The allegations claim that between February 2016 and September 2023, Illumina sold government agencies genomic sequencing systems with software that had cybersecurity vulnerabilities without having an adequate security program. Furthermore, the U.S. contended that Illumina knowingly failed to incorporate product cybersecurity in its software design, and falsely represented that the software on the genomic sequencing systems adhered to cybersecurity standards (36). Illumina's inadequate cybersecurity practices created a significant risk in which sensitive genomic information processed by its sequencing systems could be exposed to unauthorized access or misuse.

Additionally, with genetic research and testing, the issue of potential genetic discrimination arises. The Human Genome Project was an international research study looking to comprehensively analyze the chemical sequence of the complete human genetic material and identify all the genes contained within the genome (37). One of the concerns raised about this project was the potential for genetic discrimination – the unfair treatment of individuals based on their genetic characteristics or those of their family members. Genetic discrimination can limit a person's social and professional opportunities, leading to psychological, social, and economic disadvantages and distress (32). It was also a concern that people might avoid genetic testing if they feared their results could be used against them in employment, insurance, mortgages, etc. (7). These concerns remain relevant today as personalized medicine becomes more integrated into cardiovascular care.

In the United States, some legal protections exist, including the Genetic Information Nondiscrimination Act (GINA) of 2008. GINA prohibits health insurers from discriminating against enrollees based on genetic information such as refusing coverage or raising premiums for an otherwise healthy person solely because their genetic profile indicates a potential risk of disease. Additionally, GINA also prevents employers from using genetic information in employment decisions, and from requiring genetic information or tests as a condition for employment. However, GINA's protections do not extend to life insurance, disability insurance, or long-term care insurance, in which genetic data could still influence decisions (38). In this case, personalized medicine, while having great potential for improving cardiovascular treatment, also risks deepening existing inequalities if genetic data is used for exclusion and discrimination.

ETHICS AND HEALTHCARE ACCESSIBILITY

Despite its potential to transform cardiovascular care, personalized medicine faces significant ethical and healthcare accessibility challenges, primarily stemming from its high cost and the unequal representation in genomic research. Genetic testing is expensive. Furthermore, although pharmacogenomic testing has the potential to reduce healthcare costs overall by improving treatment efficiency, patients who have undergone genetic testing are potentially more likely to take medications with higher direct costs than the standard therapy. Patients who have full health insurance coverage, or those who can afford such expenses out of pocket, will have the ability to undergo new tests and benefit from individualized treatments despite their cost. However, these developments are unlikely to help people without insurance, or those whose insurance is solely intended to cover urgent treatment (7).

A discrete choice experiment (DCE) survey, conducted among U.S. adults with an average annual household income of $42,000 or less, assessed willingness to pay for pharmacogenomic (PGx) testing. Results indicated that approximately 82% of respondents were only willing to pay less than $100 for PGx testing, with a clear price ceiling at $200, and among all attributes, out-of-pocket cost was identified as having the greatest relative importance in decision-making (39). However, the price of genetic testing can vary from $100 to over $2,000, depending on the complexity of the test. (40). These economic barriers severely limit access to the benefits of personalized medicine and genetic testing for many patients.

Beyond financial barriers, another critical issue impacting equitable access to personalized medicine is an issue known as the "input-output problem" in genomic research. The success of personalized medicine relies on prior genomic research. This research, the "input", identifies links between genetic variation and clinical outcomes. However, many of these studies have disproportionately focused only on certain populations. For instance, in a 2011 study of publications in the National Human Genome Research Institute (NHGRI) Catalog of Genome-Wide Association Studies, nearly 75% of studies involved only populations of European descent (41). As a result of this input issue, many populations that already face health disparities in the United States remain inadequately studied. Thus, the issue of output is that benefits of personalized medicine

will likely be distributed unevenly among population groups.

This issue is seen prominently in cardiovascular disease treatment. In the case of warfarin, in which dosing is guided by genetic testing, numerous dosing algorithms have been developed using genetic variants like CYP2C9 and VKORC1. However, their validation in clinical trials has produced mixed results. Notably, the European Pharmacogenetics of Anticoagulant Therapy (EU-PACT) trial, conducted predominantly in populations of European descent, showed improved therapeutic outcomes with genotype-guided dosing. In contrast, the Clarification of Optimal Anticoagulation through Genetics (COAG) trial, which included a more diverse group that included African American patients, reported no significant benefit over clinically guided dosing (6). These discrepancies highlight how the disproportionate focus of genomic research on European ancestry populations can limit the effectiveness and generalizability of pharmacogenetic applications across diverse groups.

Furthermore, this is seen again in genetic research for DOACs. While randomized controlled trials included patients of different ages and kidney function, they largely excluded participants from diverse ancestral backgrounds. Most pharmacogenetic associations with DOACs have been studied in European and Asian populations, leaving significant gaps in knowledge about how genetic and clinical predictors apply to other groups. Because genotype frequencies, disease burden, and drug response vary across biogeographic populations, this underrepresentation risks producing findings that cannot be generalized equitably (30).

Moreover, if genetic testing panels, which analyze genes to identify variation or mutations (42), and pharmacogenomic algorithms, which guide drug dosing or selection by integrating genetic information with drug response data (43), are developed using data from a limited group, they may not accurately predict disease risk or guide therapy for all populations. The degree to which information gained from these studies is transferable to other populations has not been investigated thoroughly (41). Consequently, patients from racial and ethnic minorities risk receiving less accurate diagnoses and effective care, further widening existing inequities in healthcare. If there is not enough effort to address these inequities, personalized medicine has the potential to deepen (instead of reducing) disparities in healthcare.

CONCLUSION

Personalized medicine holds promise in transforming cardiovascular care by enabling more precise prevention, diagnosis, and treatment strategies. From conditions such as familial hypercholesterolemia to arrhythmias, and potential use in DOAC and warfarin dosing, integrating genetic testing into clinical practice has demonstrated the potential to improve outcomes and reduce risks. At the same time, the science behind personalized medicine continues to progress rapidly, with ongoing trials and emerging applications across cardiovascular disorders.

However, alongside this progress comes pressing concerns about privacy. Usage of electronic health records and genomic databases highlights the risks of cybersecurity breaches, genetic discrimination, and misuse of sensitive information. Recent cases such as the Illumina cybersecurity settlement underscore the need for stronger data protection policies, stricter compliance standards, and more effort to develop patient trust.

Ethical and accessibility challenges also remain critical. While personalized medicine has large potential for improving cardiovascular care, its benefits are not equally accessible to all at the moment due to high costs and unequal representation in research. Unless addressed, these inequities can possibly widen existing healthcare disparities, limiting who benefits, and leaving marginalized groups behind. Encouragingly, future efforts– including reductions in testing costs, broader inclusion in genomic studies, and stronger anti-discrimination protections like the expansion of GINA – could help ensure that personalized medicine benefits all patients, and not just a privileged few.

Looking ahead, emerging technologies such as artificial intelligence, large-scale genomics, and federated EHR systems could have the potential to transform personalized cardiovascular care. AI-driven analytics could possibly synthesize complex genetic, clinical, and lifestyle data to identify risk patterns and predict treatment responses more accurately and more efficiently. Expanding global genomic databases could also advance the discovery of new variants and improve the inclusivity of cardiovascular research across diverse populations. Furthermore, federated EHR networks, systems that allow multiple healthcare institutions to collaborate and share EHR data for research, could potentially allow decentralized data sharing without compromising

patient privacy. Together, these innovations have the potential to accelerate precision medicine and bring individualized CVD management within reach. Ultimately, the future of personalized medicine in cardiovascular disease depends on balancing scientific innovation with equity. New scientific discoveries, coupled with supportive laws and responsible company policies, can realize the potential of personalized medicine in cardiovascular disease management while addressing privacy and ethical concerns. If these barriers are met with meaningful efforts for reform, personalized medicine has the potential to not only advance cardiovascular treatment but also help shape a more inclusive and patient-centered healthcare system.

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CONFLICT OF INTERESTS

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