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Pharmacogenomics, Precision Medicine and African Genomic Diversity: A Theoretical Framework for Equitable Translation of Genomic Knowledge into Prescribing Practice

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ABSTRACT

African populations carry the greatest genomic diversity of any human population, yet they are the least represented in the databases from which pharmacogenomic knowledge is derived. As a result, dosing guidelines, drug-response predictions and the machine-learning models increasingly built on them are least reliable for the populations in which genetic variation is greatest. This theoretical paper develops a framework for the equitable translation of pharmacogenomics into African prescribing practice. It draws on pharmacogenomics and clinical pharmacology, on the health-equity and genomic-justice literatures, and on recent research on African genomic variation, pharmacogenomic implementation and the prediction of drug response. The analysis separates three gaps between genomic knowledge and clinical benefit: a discovery gap, in which variants of African relevance remain under-studied; an evidence gap, in which associations established elsewhere are unvalidated in African populations; and an implementation gap, in which genotyping, decision support and trained prescribers are unavailable. The paper argues that closing the first two gaps without the third produces knowledge that does not reach patients, while closing the third on imported knowledge produces confident but unreliable prescribing. On this basis it advances a set of propositions concerning the sequencing of investment across the three gaps, the governance of African genomic data in international research, and the design of pharmacogenomic decision support for settings where genotyping is rare, before drawing out implications for Nigerian pharmacy practice, regulation and research. It concludes that machine-learning models for drug-response prediction must be developed or validated on African data, and that the governance of genomic data collected in Africa determines whether the resulting knowledge serves African patients or only external research.

Keywords: pharmacogenomics; precision medicine; genomic diversity; African populations; drug response

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1. INTRODUCTION

The same dose of the same medicine can help one patient, fail another and harm a third. Much of this variation is genetic. Enzymes that metabolise drugs, transporters that move them and receptors that respond to them are encoded by genes that vary between individuals and between populations. Pharmacogenomics studies this variation and uses it to guide prescribing: to choose the right drug, the right dose and the patients for whom a drug is unsafe. In well-resourced health systems, pharmacogenomic testing is increasingly used to individualise therapy for medicines ranging from anticoagulants and antidepressants to chemotherapy and antiretrovirals.

This knowledge rests on data, and the data are skewed. African populations are the oldest and the most genetically diverse of all human populations, carrying variation that arose over the longest evolutionary history and that is absent from other groups. Yet they are severely underrepresented in the genomic studies, reference panels and pharmacogenomic databases on which drug-response knowledge is built. The genetic diversity of African populations has direct and underappreciated implications for personalised and precision medicine (Sibomana, 2024). The underrepresentation of African ancestry in pharmacogenetic research has been documented for specific medicines, such as tamoxifen, where it limits the applicability of dosing guidance to African patients (Nthontho et al., 2024). Africa is not alone in this: the underrepresentation of other non-European populations, such as Arab populations, in genomic studies of common disease shows that the problem is structural rather than particular to one region (Bhattacharya et al., 2023).

The consequence is a paradox. Pharmacogenomic guidance is least reliable precisely for the populations in which genetic variation is greatest. A dosing algorithm derived mainly from European or East Asian data may mispredict drug response in African patients, who carry variants the algorithm never saw. As machine-learning models are built on these same skewed data, they inherit and may amplify the bias. The promise of precision medicine, that therapy can be tailored to the individual, risks becoming a mechanism by which the benefits of genomic science flow to the already well-served while others receive confident but inaccurate guidance.

This paper develops a framework for translating pharmacogenomics into African prescribing practice equitably. It distinguishes three gaps between genomic knowledge and clinical benefit, a discovery gap, an evidence gap and an implementation gap, and argues that these gaps must be closed in a coordinated sequence, because closing some without others produces either knowledge that does not reach patients or prescribing that is confident but wrong. The analysis is theoretical rather than empirical. It integrates recent work on African genomic variation (Esoh et al., 2025; Marutha et al., 2025; Sibomana, 2024), on pharmacogenomic interpretation and prediction (Ingelman-Sundberg & Molden, 2025; Samarasinghe et al., 2026; Tremmel et al., 2025), on population-specific pharmacogenetics (Abdi et al., 2026; Booyse et al., 2023; Eniayewu et al., 2024; Shi & Deng, 2024), on polygenic prediction across ancestries (Kamp et al., 2024; Lehmann et al., 2023), on implementation and education (Caudle et al., 2023; Cole et al., 2026; Kusljic & Luzum, 2024), and on genomic data governance in Africa (Mweemba et al., 2024; Shingwenyana et al., 2024). It proceeds from an account of the three gaps and a closer examination of each, through the particular way machine learning interacts with them, to a set of propositions and an application of the framework to Nigeria.

2. LITERATURE REVIEW

2.1 Three Translation Gaps

Between a gene and a better prescription lie several steps, and a failure at any one breaks the chain. The framework groups these failures into three gaps.

The discovery gap is the gap between the genetic variation that exists in African populations and the variation that has been discovered and characterised. Pharmacogenomic knowledge begins with identifying which variants affect drug response. If the variants that matter in African populations have not been studied, they cannot inform prescribing, however good the downstream systems. The diversity of African genomes means that many pharmacogenomically relevant variants remain uncharacterised, and that variants common in African populations may be rare or absent

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elsewhere (Sibomana, 2024). Exome and genome sequencing of African cohorts continues to identify both known and novel variants, which shows how much remains undiscovered (Marutha et al., 2025).

The evidence gap is the gap between knowing that a variant exists and knowing what it does for drug response in a given population. A variant may be catalogued, but its clinical effect, how it changes the metabolism, efficacy or toxicity of a specific drug, may have been established only in other populations or not at all. Associations found in one population do not always hold in another, because the effect of a variant can depend on its genetic background, on other variants, and on environmental factors that differ between populations. Translating sequencing data into drug-response predictions is complicated by variants of unknown significance, whose effects cannot be predicted reliably (Tremmel et al., 2025). These variants are disproportionately common in understudied populations.

The implementation gap is the gap between validated knowledge and its use in prescribing. Even where a variant's effect is known and relevant, acting on it requires genotyping to be available and affordable, decision support to translate a genotype into a recommendation, prescribers trained to use it, and health systems that integrate it into care. Implementation is difficult even in well-resourced systems, where it depends on workflow, education and infrastructure (Caudle et al., 2023; Cole et al., 2026; Kusljic & Luzum, 2024). In settings where genotyping is rare and prescribers are overstretched, the implementation gap can be total: no amount of knowledge reaches patients.

The central argument is that these gaps interact. Closing the discovery and evidence gaps without the implementation gap produces knowledge that sits in databases and journals but never changes a prescription. Closing the implementation gap on knowledge borrowed from other populations, without closing the discovery and evidence gaps for the local population, produces prescribing that is confident, systematic and wrong. Equitable translation requires closing all three, and in a coordinated way.

2.2 The Discovery Gap

The discovery gap originates in the composition of genomic databases. The reference genomes, variant catalogues and pharmacogenomic knowledge bases that underpin precision medicine were built disproportionately from European-ancestry data. African variation, being both greater and less studied, is underrepresented. This has two consequences for pharmacogenomics. First, variants that influence drug response in African populations may simply be absent from the catalogues, so that a patient's clinically important variant is invisible to standard testing. Second, the frequency and functional effect of known variants may differ in African populations in ways that have not been measured.

Characterising African pharmacogenomic variation is therefore a prerequisite for equitable precision medicine. Studies of specific genes illustrate both the need and the progress. Characterisation of CYP2C19 variation, which affects the metabolism of many drugs including the antiplatelet agent clopidogrel and several antidepressants, shows that African populations carry patterns of variation distinct from those of other global populations (Booyse et al., 2023). The under-study of African ancestry in the pharmacogenetics of individual drugs, such as tamoxifen, leaves African patients without evidence to guide therapy (Nthontho et al., 2024). African genomes also carry variants shaped by the long history of malaria and other parasitic diseases, with implications that extend beyond infection to drug response and to genetic medicine generally (Esoh et al., 2025).

Technology is changing what can be discovered. Short-read sequencing, the dominant method, struggles with the complex, highly variable regions in which many pharmacogenes lie. Long-read sequencing resolves complex haplotypes, identifies novel star alleles and detects structural variants that short reads miss (Samarasinghe et al., 2026). Because African pharmacogenes are especially diverse and structurally complex, these methods are particularly valuable for characterising African variation. The discovery gap is thus partly a matter of applying the right technology to the right populations, and African cohorts have been undersequenced with methods capable of capturing their full variation.

Closing the discovery gap requires sequencing African populations at scale, with methods adequate to their diversity, and characterising the functional effects of the variants found. This is a scientific task, but it is also a question of where genomic research is directed and funded. The framework treats the discovery gap as the foundation: without it, the later gaps cannot be closed correctly for African patients, because the knowledge to be

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implemented would be knowledge about other populations.

2.3 The Evidence Gap

Discovering a variant is not the same as knowing what it means for a patient. The evidence gap is the gap between a catalogued variant and a validated, clinically actionable understanding of its effect on drug response in a specific population.

The central difficulty is the variant of unknown significance: a genetic variant whose functional consequence has not been established. Translating pharmacogenomic sequencing data into drug-response predictions requires interpreting such variants, and reliable interpretation is often not possible with current methods (Tremmel et al., 2025). Variants of unknown significance are more common in understudied populations, precisely because less work has been done to characterise their effects. African patients are therefore more likely than others to carry variants that testing detects but cannot interpret. A test result that reports an uninterpretable variant provides no basis for action and may even mislead.

Even for variants whose effects are known in principle, the clinical evidence may not transfer across populations. Dosing algorithms illustrate this. Pharmacogenetics-based warfarin dosing algorithms combine genetic and clinical factors to predict the dose a patient needs, but their performance depends on the population in which they were developed, and algorithms are developed and refined for specific populations (Shi & Deng, 2024). An algorithm optimised for one population may dose another poorly. For African populations, whose variation is greatest and whose representation in algorithm development is least, imported algorithms are the least likely to perform well. The same logic applies to other drugs. Systematic review of DPYD variants, which predict severe toxicity from fluoropyrimidine chemotherapy, finds that the variants relevant in Asian and African populations differ from those established in European populations, so that testing panels designed elsewhere may miss the variants that matter locally (Abdi et al., 2026).

Generating population-specific evidence requires studies that relate genotype to drug response in the population of interest. Work on efavirenz, an antiretroviral whose metabolism is strongly influenced by CYP2B6 variants common in African populations, shows both the importance and the feasibility of such studies. Research in Nigerian patients has characterised how CYP2B6 genotype influences efavirenz exposure, including during pregnancy (Eniayewu et al., 2024). This is the kind of evidence that closes the evidence gap: it establishes, in the relevant population, what a known variant means for a specific drug. The framework holds that such studies are indispensable and cannot be replaced by extrapolation from other populations.

2.4 The Implementation Gap

Validated knowledge changes outcomes only when it reaches prescribing. The implementation gap is the gap between knowing what a patient's genotype means and acting on it in care. It has several components: the availability and affordability of genotyping, the decision support that turns a genotype into a recommendation, the training of prescribers and pharmacists, and the integration of all of these into health-system workflows.

Implementation is hard everywhere. Even in well-resourced systems, pharmacogenomic testing has been slow to enter routine practice, held back by workflow barriers, gaps in prescriber education and the difficulty of delivering recommendations at the point of care. The call for less discussion and more action in pharmacogenomic implementation reflects the persistence of these barriers (Caudle et al., 2023). Education is a recurring constraint: prescribers and pharmacists often lack the training to interpret and act on pharmacogenomic results, and closing this gap requires deliberate educational investment (Kusljic & Luzum, 2024). Models of implementation continue to be developed and evaluated, including preemptive multigene testing in settings such as emergency departments (Cole et al., 2026).

In most African health systems the implementation gap is wider still. Genotyping is rarely available outside research settings and specialised centres, and its cost is prohibitive for routine use. Decision-support systems integrated with electronic health records, which deliver pharmacogenomic recommendations to prescribers in well-resourced systems, are largely absent. Prescribers and pharmacists are overstretched and seldom trained in pharmacogenomics. And the health-system infrastructure that would carry a genotype from a laboratory to a prescribing decision is fragmented or missing.

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The framework draws a sharp conclusion from the interaction of the gaps. Closing the implementation gap in isolation, by deploying genotyping and decision support based on knowledge derived from other populations, would not serve African patients well. It would deliver, efficiently and at scale, recommendations that may be wrong for the population receiving them. The appeal of imported decision-support tools is precisely that they allow the implementation gap to be closed quickly, bypassing the slow work of discovery and evidence generation. The framework warns that this shortcut produces confident, systematic error. Decision support for African prescribing must be built on, or at least validated against, African discovery and evidence.

2.5 Machine Learning and the Amplification of Bias

Machine learning is increasingly used to predict drug response from genomic and clinical data, and it interacts with the three gaps in a specific and concerning way. Models learn from data, and the data available for pharmacogenomic prediction carry the discovery and evidence gaps described above. A model trained predominantly on European-ancestry data will predict well for European-ancestry patients and less well for others, and it may do so without any signal that its predictions are less reliable for underrepresented groups.

Polygenic scores illustrate the problem. These scores aggregate the effects of many variants to predict a trait or drug response. Their predictive performance is well known to transfer poorly across ancestries: a score developed in one population loses accuracy when applied to another, and the loss is greatest for populations most distant from the training data. Optimal strategies for learning multi-ancestry polygenic scores vary across traits, and no single approach resolves the transferability problem (Lehmann et al., 2023). Ancestry-aligned scores, developed or calibrated for the target population, perform better. Combined with conventional risk factors, ancestry-aligned polygenic scores improve prediction of cardiometabolic outcomes in African populations relative to scores not so aligned (Kamp et al., 2024). The lesson generalises: models must be developed or validated for the populations in which they will be used.

The danger specific to machine learning is the amplification and concealment of bias. A model that performs poorly for African patients may nonetheless report high confidence, because its confidence reflects the data it was trained on rather than the population it is applied to. Deployed at scale, such a model would deliver systematically biased predictions with an appearance of objectivity. Where human prescribers might hesitate before applying guidance derived from a different population, an automated system applies it uniformly. The implementation gap, closed by machine learning built on biased data, becomes a channel for delivering biased prescribing efficiently.

The framework therefore holds that machine learning does not substitute for closing the discovery and evidence gaps. On the contrary, it raises the stakes. Models should be trained or validated on data from the populations in which they will be used; they should report when they are operating outside the range of their training data; and they should not be deployed for African prescribing on the strength of performance demonstrated in other populations. The newer tools of pharmacogenomic interpretation, including methods that integrate therapeutic drug monitoring with genomic prediction, may help where genotype alone is insufficient, but they too depend on population-relevant data (Ingelman-Sundberg & Molden, 2025).

3. CONCEPTUAL FRAMEWORK

The preceding analysis supports several propositions, stated here as conditional claims open to empirical test and as principles for policy on pharmacogenomics and precision medicine. The most general is that pharmacogenomic guidance is least reliable for the populations in which genetic variation is greatest, since African populations carry the most variation and are the least represented in the data from which guidance is derived (Nthontho et al., 2024; Sibomana, 2024). The three translation gaps that follow from this interact rather than operating independently: closing discovery and evidence without implementation produces knowledge that does not reach patients, while closing implementation on imported knowledge produces confident but unreliable prescribing, so that equitable translation requires closing all three in coordination.

Within that sequence, the discovery gap is foundational, because characterising African pharmacogenomic variation with sequencing methods adequate to its complexity is a prerequisite

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for the later gaps to be closed correctly for African patients (Booyse et al., 2023; Marutha et al., 2025; Samarasinghe et al., 2026). The evidence gap is distinct and equally consequential, since clinical evidence does not transfer freely across populations: dosing algorithms and variant-toxicity associations established in one population may perform poorly in another, which makes population-specific evidence indispensable (Abdi et al., 2026; Eniayewu et al., 2024; Shi & Deng, 2024). A particular feature of understudied populations compounds the difficulty, in that variants of unknown significance are more common among them, so African patients are more likely to receive test results that cannot be interpreted, and investment in functional characterisation is needed to make testing actionable (Tremmel et al., 2025).

The final propositions concern computation and governance. Machine-learning models trained on ancestrally skewed data predict poorly for underrepresented populations while potentially reporting high confidence, so models for African prescribing must be developed or validated on African data and must signal when they operate outside their training range (Kamp et al., 2024; Lehmann et al., 2023). Underlying all of this, the governance of African genomic data determines whether African populations share in the benefits of the knowledge their data generate, which requires that data collected in Africa be governed so that resulting discoveries, evidence and tools serve African patients, not only external research (Mweemba et al., 2024; Shingwenyana et al., 2024).

4. ANALYSIS AND DISCUSSION

4.1 Nigeria as an Anchoring Case

Nigeria illustrates the three gaps and the stakes of closing them. As Africa's most populous country, with great internal ethnic and genetic diversity, Nigeria carries pharmacogenomic variation that is both extensive and incompletely characterised. Its disease burden, including HIV, tuberculosis, malaria and a rising burden of cancer and non-communicable diseases, involves many medicines whose safety and efficacy depend on genes known to vary in African populations.

The discovery gap in Nigeria is substantial but not untouched. Nigerian populations remain underrepresented in global pharmacogenomic databases, yet Nigerian researchers have contributed evidence on clinically important genes. The work on CYP2B6 and efavirenz, including in Nigerian patients, shows that locally relevant pharmacogenomic knowledge can be generated and that it matters for prescribing a widely used antiretroviral (Eniayewu et al., 2024). Extending such work across the genes and drugs relevant to Nigeria's disease burden is the task of closing the discovery gap.

The evidence gap is acute for the medicines Nigerians use most. Antiretrovirals, antimalarials, antituberculosis drugs, anticoagulants and increasingly chemotherapy all have pharmacogenomic dimensions where population-specific evidence is needed. Where guidance is imported, it may not fit. Warfarin dosing algorithms developed in other populations may mis-dose Nigerian patients (Shi & Deng, 2024), and fluoropyrimidine toxicity panels designed around European variants may miss the DPYD variants relevant to African patients (Abdi et al., 2026). Generating Nigerian evidence for these high-use medicines would have direct clinical value.

The implementation gap is the widest. Pharmacogenomic testing is confined largely to research and a few specialised centres. Decision-support systems are absent from most of the health system. Pharmacists and prescribers are seldom trained in pharmacogenomics, and the framework's emphasis on education (Kusljic & Luzum, 2024) applies strongly. Yet the framework also cautions against closing this gap with imported tools alone. A decision-support system loaded with guidelines derived from European or East Asian data, deployed in Nigerian pharmacies, would deliver recommendations that may be systematically wrong for Nigerian patients. The pharmacy profession, which is well placed to lead pharmacogenomic implementation, should press for tools built on or validated against Nigerian and African evidence.

Data governance ties the gaps together. Nigerian genomic data generated in international collaborations are valuable, and how they are governed determines whether the resulting knowledge serves Nigerian patients. Perspectives from across Africa on the regulation of genomics research highlight the importance of national frameworks that protect participants and secure benefit for the populations studied (Mweemba et al., 2024). The return of findings to participants, and the terms on which data are shared, are matters of justice as well as science (Shingwenyana et al., 2024). For Nigeria, participating in international pharmacogenomic research on terms that build local

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capacity, retain a share in resulting tools, and return benefit to Nigerian patients is as important as the science itself.

4.2 Implications

For pharmacists and prescribers, the framework implies caution in applying pharmacogenomic guidance derived from other populations to African patients, and attention to whether a test result is actionable or reports a variant of unknown significance (Tremmel et al., 2025); it also implies a professional responsibility to build pharmacogenomic competence, since implementation depends on trained practitioners (Caudle et al., 2023; Kusljic & Luzum, 2024). For researchers and research funders, the discovery and evidence gaps should be closed for the medicines and genes most relevant to African disease burden, using sequencing methods adequate to African diversity (Marutha et al., 2025; Samarasinghe et al., 2026) and generating population-specific genotype-response evidence (Abdi et al., 2026; Eniayewu et al., 2024), while machine-learning models for drug-response prediction are developed or validated on African data (Kamp et al., 2024; Lehmann et al., 2023).

For regulators and policymakers, the three gaps should be addressed in coordination rather than piecemeal, the implementation of pharmacogenomic testing should not outrun the local evidence base, and the governance of genomic data should ensure that data collected in Africa yield benefit for African patients (Mweemba et al., 2024; Shingwenyana et al., 2024). For those who develop pharmacogenomic decision support, the corresponding requirement is that tools intended for African use be built on or validated against African discovery and evidence, that they flag uninterpretable variants and out-of-range predictions, and that they not present imported guidance as if it were locally validated.

The propositions also identify open questions for research. How large are the discovery and evidence gaps for the medicines most used in African health systems? How poorly do imported dosing algorithms and toxicity panels perform in African populations in practice? How can decision support be designed for settings where genotyping is rare, perhaps combining selective genotyping with other predictors, and what governance arrangements most effectively secure benefit for the populations whose data drive discovery? Answering these questions requires research conducted in African populations and health systems rather than extrapolation from better-studied ones.

5. CONCLUSION AND RECOMMENDATION

Pharmacogenomics promises to make prescribing safer and more effective by tailoring therapy to the patient's genes. This paper has argued that the promise is, as things stand, least likely to be kept for the populations with the most genetic variation. African populations are the most diverse and the least represented in the data from which pharmacogenomic knowledge is built, so the guidance derived from that knowledge is least reliable for them. Realising the promise equitably requires closing three gaps: the discovery gap, where African variants are under-studied; the evidence gap, where their effects are unvalidated in African populations; and the implementation gap, where testing, decision support and trained prescribers are unavailable.

These gaps must be closed together. Knowledge without implementation never reaches patients. Implementation without local discovery and evidence delivers confident error. Machine learning does not resolve the tension; built on skewed data, it can deliver biased prescribing efficiently and with an appearance of objectivity. The way forward is to characterise African pharmacogenomic variation with adequate methods, generate population-specific genotype-response evidence for high-use medicines, build decision support on that evidence, train the prescribers and pharmacists who will use it, and govern African genomic data so that the benefits return to African patients.

For Nigeria, with its vast and diverse population and its heavy use of medicines whose action depends on variable genes, the stakes are high. The country can either receive precision medicine as a set of imported tools that may not fit its people, or participate in building a pharmacogenomics that reflects its own genetic diversity. The second path is slower and more demanding, but it is the one that leads to prescribing that is precise not only in appearance but in fact.

6. CONTRIBUTION TO EXISTING KNOWLEDGE

The paper contributes a decomposition of the translation of pharmacogenomics into prescribing practice into a discovery gap, an evidence gap and an implementation gap, and shows that closing some without the others produces either knowledge

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that does not reach patients or confident but unreliable prescribing.

It gives researchers, regulators and pharmacists a basis for closing the three gaps in coordination, and for ensuring that precision medicine reflects, rather than overlooks, the genomic diversity of African populations for whom imported guidance is least reliable.

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