



NIGER DELTA UNIVERSITY
WILBERFORCE ISLAND, BAYELSA STATE.

71st Inaugural Lecture

**GENE-DRUG COMPLEX:
DIVINE BLUEPRINT TO
PERSONALIZED
PHARMACOTHERAPY**

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B.Pharm (ABU), MSc, PhD (Ife), MISSX, MPSN

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NIGER DELTA UNIVERSITY

Wilberforce Island, Bayelsa State, Nigeria

Motto

Creativity, Excellence, Service

Vision

To be a centre of excellence defined by well articulated programme that will produce creative and innovative minds

Mission

To strive to maintain an international reputation for high quality scholarship, research and academic excellence for the promotion of the socio-cultural and economic well-being of mankind

NIGER DELTA UNIVERSITY ANTHEM (THE BRIGHTEST STAR)

Like the brightest star we are, to lead the way
To good education that is all our due,
The dream of our fathers like the seed has grown;
Niger Delta University is here to stay.

In all that we do, let us bring to mind
Our duty as staff and students of N.D.U
Ev'rywhere to promote peace towards mankind.
Creativity, Excellence and Service

Let us build on this noble foundation
And with love, let our dedication increase,
To raise and uphold this noble vision
Ev'ry passing moment let our zeal never decrease.

CHORUS
Rejoice, great people old and new, rejoice
For the good fruit through us is shown;
Be glad in our worthy contribution
To the growth of humanity (x2)

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Dedication

The 71st Inaugural Lecture is dedicated to the memory of my late Mother, ENE (Mrs) Adi Anyuobe Ebeshi

PROTOCOL

The Vice Chancellor

The Deputy Vice-Chancellors

The Registrar and Other Principal Officers

The Provost, College of Health Sciences

The Dean Postgraduate School

Deans of Faculty

Directors of Institutes and Centers

Heads of Department

Distinguished Professors

Past Inaugural Lecturers

Heads of Administrative Units

Distinguished Academics and Administrators

My Family

Respected Chiefs and Elders

My Distinguished Professional Colleagues

My Distinguished Friends and Guests

Great Niger Delta University Students

Members of the Press

Ladies and Gentlemen

1.0 INTRODUCTION

1.1 Preamble

Distinguished Vice-Chancellor, Principal Officers of Niger Delta University, esteemed colleagues in the Faculty of Pharmacy and Pharmacist Professionals in the Pharma Industry, hospitals, community, academia and administrative settings, traditional rulers, family, friends, and beloved students

On this auspicious day, it is my pleasure to stand before you to deliver this inaugural lecture as a Professor of Pharmaceutical Chemistry with expertise in Pharmacogenetics.

An inaugural lecture is basically a public presentation by a newly appointed professor, showcasing his research expertise and contributions to his field. It's an opportunity to share groundbreaking research, highlight achievements and impact, set future research directions and engage with academia and the public.

Mr Vice Chancellor, being guided in my Christian faith and upbringing, I draw inspiration from the scriptures. In this inaugural lecture, I attempted to harmonize some relevant Bible passages that underscore the divine connection between creation, human existence and healing.

As we reflect on God's creation, we see that 'the Lord God planted a garden in Eden' (Genesis 2:8) and 'out of the ground the Lord God made every tree that is pleasant to the sight and good for food' (Genesis 2:9), providing sustenance for humanity, whom He created 'in His own image' (Genesis 1:27). This divine provision underscores the importance of plants not only as food but also as veritable source of drugs, as seen in the various herbs and plants used for healing throughout Scripture, such as the balm of Gilead (Jeremiah 8:22, 46:11) and the leaves of the tree of life for healing (Revelation 22:2)."

The title I have chosen “***Gene-Drug Complex: Divine blueprint to personalized pharmacotherapy***’. The lecture explores how genetic variations influence drug response, highlighting breakthroughs in tailoring therapies to individual genetic profiles. The intricate relationship between an individual's genetic variants and their response to a specific drug, which encompasses how genes influence the pharmacokinetics (ADME) and pharmacodynamics (drug target interaction) of a medication is define by Gene-drug interaction.

The molecular basis for the observed variation in genetic polymorphism has been shown to be due to deletions, insertions, duplications/multiplication and point mutations within the genes coding for the drug metabolizing enzymes. These genetic changes give rise to enzymes with altered, deficient or increased activity. The changes in enzyme activity result in compromised metabolism of drugs and environmental carcinogens.

Therefore, delving into the intersection of genetics and pharmacotherapy, showcasing pioneering research in the Nigerian population, driving the shift towards precision medicine, revolutionizing treatment outcomes and patient care, I invite you all to journey with me into the world of **Personalized Pharmacotherapy**.

1.2 Definitions

- i) ***Pharmacokinetics-*** *The rate processes of drug absorption, distribution, metabolism, and excretion (ADME) over time. Basically, it involves the movement of drugs into the body, around the body and out of the body (sometimes refers to as what the body does to the drug).*
- ii) ***Pharmacodynamic:*** *Pharmacodynamics*

- (sometimes described as what a drug does to the body) is the study of the biochemical, physiologic, and molecular effects of drugs on the body and involves receptor binding (including receptor sensitivity), postreceptor effects, and chemical interactions.
- iii) **Gene-Drug Complex:** The intricate relationship between an individual's genetic variants and their response to a specific drug. It encompasses how genes influence the pharmacokinetics (ADME) and pharmacodynamics (drug target interaction) of a medication.
 - iv) **Pharmacogenetics:** Often used interchangeably with pharmacogenomics, but traditionally refers to the study of the role of single genes, e.g., CYP2C9, VKORC1, in drug response.
 - v) **Personalized Pharmacotherapy (Personalized Medicine):** The practice of using an individual's genetic, genomic, clinical, and environmental information to select the right drug, at the right dose, for the right patient and at the right time.
 - vi) **Genetic polymorphism:** the occurrence of two or more genetic variants, or alleles, within a population, with the least common allele having a frequency of at least 1%. This concept indicates genetic diversity, which is crucial for natural selection and adaptation. A gene is considered polymorphic if more than one allele occupies its locus within a population. Genetic polymorphism can manifest as different phenotypes among individuals, which cannot

- be maintained solely by recurrent mutation.*
- vii) **Single-nucleotide polymorphisms:** *In genomics, polymorphisms often involve variations at a single nucleotide, known as single-nucleotide polymorphisms (SNPs).*
- viii) **Adverse drug reactions (ADRs):** *unintended and harmful responses to medications, ranging from mild side effects to severe health complications.*

1.3. Personalized Pharmacotherapy

The goal of Personalized drug therapy is to give the right drug, at the right dose, for the right patient, minimizing adverse drug reactions (ADRs) and maximizing effectiveness. Elements of personalized therapy include patient clinical data (age, weight, organ function), comorbidities, concurrent medications (drug–drug interactions), environmental factors (diet, smoking), and genetic information (PGx). Genetics typically modifies two broad domains: pharmacokinetics (what the body does to the drug) and pharmacodynamics (what the drug does to the body). This approach moves away from trial-and-error prescribing. By understanding a patient's genetic predispositions, clinicians and Pharmacists can:

- a. **Predict Efficacy:** *Identify patients most likely to respond to a specific therapy, e.g., trastuzumab for HER2-positive breast cancer (Jin et al., 2019).*
- b. **Prevent ADRs:** *Avoid prescribing drugs to patients with genetic variants that put them at high risk of severe toxicity, e.g., Stevens-Johnson syndrome with carbamazepine in HLA-B*15:02 carriers (Pasternak et al., 2017), or genetic polymorphism associated with efavirenz (Bunu et al., 2022).*
- c. **Optimize Dosing:** *Determine the optimal starting dose to achieve therapeutic levels quickly without causing adverse effects e.g., warfarin*

dosing based on CYP2C9 and VKORC1 genotypes (Tyson et al., 2020).

1.4. Overview of Pharmacogenomics

The scope of Pharmacogenomics is for the identification of variants (SNPs, copy number variants, HLA types, structural variants) that alter drug metabolism (e.g., CYP enzymes), transport proteins (pGP, ABCs), targets (VKORC1, HLA alleles, receptor polymorphisms), or immune responses (HLA-associated hypersensitivity). Integration into practice can include single gene tests, multi-gene panels, and pre-emptive (pre-prescription) genotyping.

Pharmacogenomics provides the scientific backbone for personalized therapy. It explains why:

- a. A standard dose of a drug can be toxic in one person and ineffective in another.
- b. Drug metabolism is not uniform; individuals can be classified as Poor Metabolizers (PMs), Intermediate Metabolizers (IMs), Extensive Metabolizers (EMs), or Ultra-Rapid Metabolizers (UMs) based on their specific drug-metabolizing enzyme profiles (Ebeshi et al., 2011; Ahmed et al., 2016)
- c. Genetic variation in drug targets (e.g., receptors) can alter a drug's ability to bind and exert its effect.

Pharmacogenomics aims to identify patients at risk for toxicity or reduced response to therapy prior to medication selection



Figure 1: Cohort of Patients with the same diagnosis but varied treatment outcomes

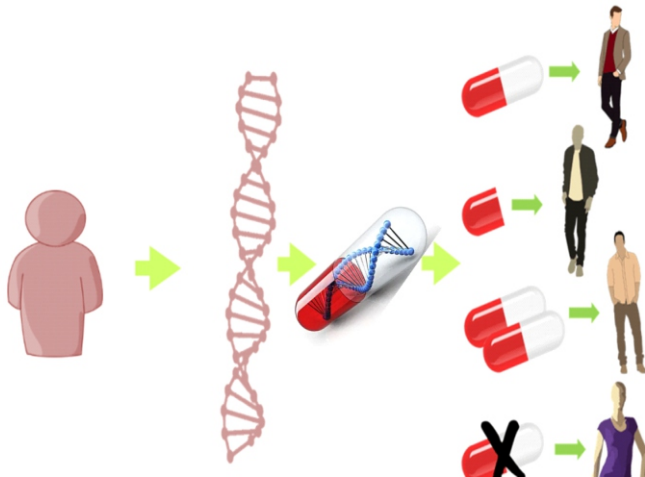


Figure 2: Dose and medicine selection based on individual genomic profile.

2.0 THE GENE-DRUG COMPLEX

2.1. What is a Gene-Drug Complex?

The gene-drug complex is not a physical complex but a conceptual framework that describes the interaction between a specific genetic locus (a point in the genome) and a specific drug. This is the set of relationships by which a patient's genetic variants modulate a drug's absorption, distribution, metabolism, elimination (ADME), target engagement, downstream signaling, and immune recognition, and together determining therapeutic outcome and risk. This interaction determines the drug's fate and effect in the body. Examples: *CYP2C19* poor metabolizers and clopidogrel (reduced activation leading to lower antiplatelet effect); HLA-B*57:01 and abacavir (high risk of hypersensitivity); TPMT deficiency and thiopurines (increased myelosuppression) (Ebeshi et al., 2011; Qahwaji et al., 2024).



Figure 3. The gene-drug complex

2.1.1. Key components of the Gene-Drug Complex include:

- a. *Drug Metabolism Genes: Genes coding for enzymes like Cytochrome P450 (CYP450 family: CYP2B6, CYP2D6, CYP2C19, CYP2C9, CYP3A4/5). Variants can lead to altered enzyme activity (Ebeshi et al. 2011).*
- b. *Drug Transporter Genes: Genes like ABCB1 (P-glycoprotein) that affect the movement of drugs across cell membranes.*
- c. *Drug Target Genes: Genes encoding the proteins (e.g., VKORC1 for warfarin, HLA proteins for immune-mediated ADRs) that a drug is designed to interact with.*
- d. *The Drug Itself: The chemical entity whose behavior is modulated by the above genetic factors.*

2.2. History and Development

Clinicians in the pre-1950s–1960s noted familial differences in drug responses for centuries; formal genetic links to drug response were described in the mid-20th century (e.g., G6PD deficiency). The term pharmacogenetics emerged mid-20th century, and discoveries such as CYP2D6 polymorphism (1970s) cemented molecular understanding. Over subsequent decades, the field expanded from single-gene associations to genomic approaches as sequencing and arrays developed (Somogy, 2008).

- a. 510 BC: Pythagoras noted that some individuals developed hemolytic anemia after eating fava beans, an early observation of a genetically based adverse reaction (Glucose-6-phosphate dehydrogenase [G6PD] deficiency) (Pirmohamed, 2001).
- b. 1950s: The term "pharmacogenetics" was coined by Friedrich Vogel. The discovery of genetic variants in enzymes like pseudocholinesterase (succinylcholine sensitivity) and G6PD (primaquine-induced anemia)

- laid the foundation (Pirmohamed, 2001).
- c. 1990s-2000s: The Human Genome Project provided the complete human DNA sequence, accelerating the field and broadening it from pharmacogenetics to pharmacogenomics.
 - d. 2000s-Present: Regulatory agencies (FDA, EMA) began incorporating PGx information into drug labels. The cost of genetic testing plummeted, making clinical application feasible. The first FDA approval of a pharmacogenetic test was in 2005 for alleles in CYP2D6 and CYP2C19 (Squassina et al., 2010).

2.3. Genetic Influence on Drug Therapy

Genes influence drug therapy through two primary pathways:

- a. Pharmacokinetics (What the body does to the drug): Genes control the activity of drug-metabolizing enzymes and transporters, determining the drug's concentration in the bloodstream. Example: A CYP2C19 Poor Metabolizer given clopidogrel (a prodrug) cannot activate it effectively, leading to treatment failure and increased risk of stent thrombosis.

Pharmacokinetic genes: *CYP450 family (CYP2D6, CYP2C19, CYP2C9, CYP3A5), UGTs, TPMT, NUDT15, DPYD* — variants alter metabolism leading to poor/ultra-rapid metabolizer phenotypes, impacting drug exposure and dosing needs (Zhou & Lauschke, 2022).

Pharmacodynamics genes: (What the drug does to the body): Genes control the structure and function of drug targets (receptors, enzymes). Example: Variants in the VKORC1 gene make the target enzyme less sensitive to warfarin, requiring a much lower dose to achieve anticoagulation.

Pharmacodynamic genes: drug targets (VKORC1 for warfarin), transporter genes (pGp, efflux of many drugs from cells leading to MDRs in Cancer chemotherapy and antiretroviral therapy), receptor polymorphisms, alter the effect magnitude or sensitivity (Qahwaji et al., 2024).

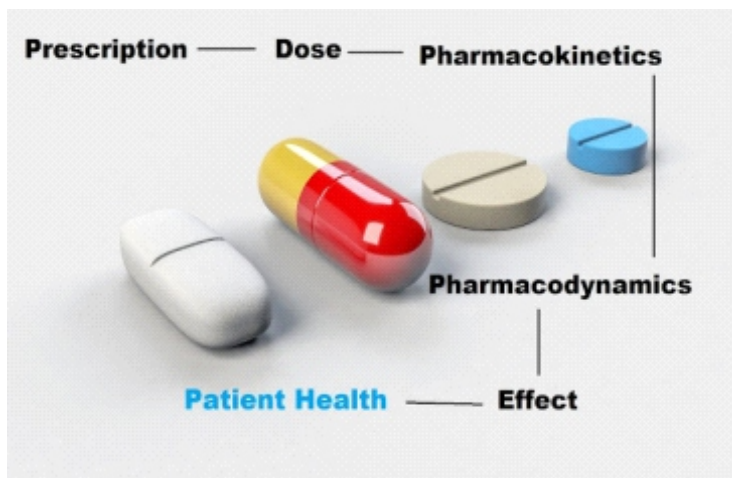


Figure 4. Drug use process and biotransformation.

2.4. Origin and Evolution of Personalized Medicine

From observations to implementation: the field evolved from descriptive observations of variable responses and recognition of enzymatic polymorphisms (N-acetyltransferase, G6PD), molecular genetics (CYP variants), high-throughput genomic tools enabling multi-gene arrays and sequence-based PGx. Contemporary precision medicine blends genomics, multi-omics, and EHR-based clinical decision support (CDS) (Somogy, 2008). Personalized medicine evolved from the convergence of several fields:

- a. Genomics: The ability to sequence and analyze genomes.
- b. Bioinformatics: The computational tools to manage and interpret vast genetic datasets.

- c. Molecular Diagnostics: Development of rapid, cost-effective genetic tests (e.g., PCR, microarrays, next-generation sequencing).
- d. Clinical Pharmacology: The understanding of drug handling and action in humans.

It has evolved from a theoretical concept to a clinical reality, with specific FDA-approved tests now required or recommended for numerous drugs.

2.5. Global and Nigerian Trends in Personalized Medicine

2.5.1. Global Trends:

Increasing clinical implementation in high-income countries (pre-emptive multigene panels in academic centers, CPIC/DPWG guideline adoption, FDA PGx labeling), growth of commercial PGx labs and direct-to-consumer interest, and incorporation in oncology and cardiology. Barriers remain: clinician knowledge gaps, EHR integration, payer coverage, and variable evidence for cost-effectiveness across settings (Chenoweth et al., 2020). Areas to consider include:

- a. Integration into Clinical Guidelines: CPIC (Clinical Pharmacogenetics Implementation Consortium) and DPWG (Dutch Pharmacogenetics Working Group) provide evidence-based guidelines for using PGx data.
- b. Pre-emptive Genotyping: Large health systems (e.g., Mayo Clinic, Vanderbilt University) test patients for a panel of PGx markers before a drug is prescribed, storing the data in the EHR for a lifetime of use.
- c. Direct-to-Consumer Testing: Companies like 23andMe offer FDA-approved reports on limited PGx markers (e.g., CYP2C19 and clopidogrel).
- d. Oncology Leading the Way: Cancer treatment is the most advanced area, with therapies routinely selected based on the tumor's genetic profile (e.g., imatinib for

BCR-ABL1+ CML, EGFR inhibitors for EGFR+ lung cancer).

2.5.2. Nigerian Trends:

Nigeria has active PGx research (population frequency studies, pilot implementation projects); however, routine clinical PGx testing and integration into standard practice remain limited due to infrastructure, cost, limited local reference data, and workforce training needs. Several studies and proposals recommend capacity building, population-specific allele frequency mapping, and context-appropriate implementation frameworks (Ebeshi et al., 2012; Bolaji et al., 2019). Areas to consider include:

- a. Research Phase: Nigeria is primarily in the research phase. Several academic institutions (e.g., Obafemi Awolowo University and University of Ibadan) are conducting important population studies to characterize the pharmacogenomic landscape of Nigerian highly diverse ethnic groups.
- b. Awareness and Advocacy: Awareness is growing among healthcare professionals and researchers. Professional bodies are beginning to discuss the integration of their services.
- c. Significant Challenges:
 - Infrastructure: Lack of widespread, affordable genetic testing facilities.
 - Funding and Cost: High cost of tests and limited coverage by the National Health Insurance Scheme (NHIS).
 - Expertise: Shortage of trained clinical pharmacologists, genetic counselors, and bioinformaticians.
 - Policy: Absence of a national framework or guidelines for PGx testing and application.
- d. Potential: Given Nigeria's genetic diversity, PGx has

immense potential to improve public health outcomes and reduce healthcare costs from ADRs. Local data is critical, as global data cannot be directly extrapolated.

2.6. Pharmacogenomics Polymorphism and Associated Therapeutic Consequences

Polymorphisms are variations in the DNA sequence that occur in >1% of the population (Bunu et al., 2022). Key types include Single Nucleotide Polymorphisms (SNPs), insertions and deletions, single nucleotide variants (SNVs), small indels, copy number variations (notably CYP2D6 gene duplications), HLA alleles, and structural variants.

Table 1. Pharmacogenomics polymorphism and associated consequences

Gene (Enzyme/Target)	Drug Substrate	Polymorphism & Consequence	Clinical Outcome
CYP2C9 / VKORC1	Warfarin	Poor metabolizer (CYP2C9*3) & sensitive target (VKORC1)	High risk of bleeding. Requires significantly lower dose.
CYP2D6	Codeine	Ultra-rapid metabolizer	Rapid conversion to morphine; Risk of respiratory depression.
CYP2C19	Clopidogrel	Poor metabolizer	Reduced active metabolite; Increased risk of stent thrombosis.
HLA-B*15:02	Carbamazepine	Carrier allele	High risk of severe dermatological reactions (SJS/TEN) in Asian populations.
TPMT	Azathioprine, Mercaptopurine	Poor metabolizer	Severe, life- threatening myelosuppression (low blood cell count).
DPYD	5-Fluorouracil, Capecitabine	Poor metabolizer	Severe, potentially fatal toxicity (mucositis, neutropenia, diarrhea).

2.7. **Methods of Pharmacogenomic Evaluation**

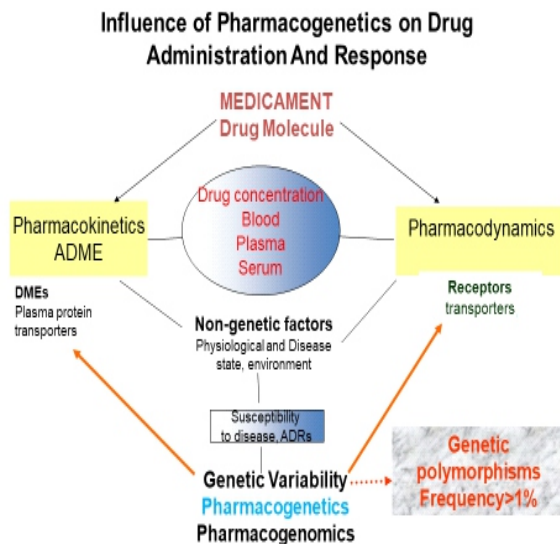
- a. **DNA Genotyping:** The most common method targeted single-gene assays (PCR, TaqMan), allele-specific tests. Uses techniques like:
 - **PCR-based Methods:** Real-time PCR (qPCR) with specific probes for known variants (e.g., TaqMan assays).
 - **DNA Microarrays (Chips):** Can test for hundreds of thousands of variants simultaneously. Used for pre-emptive panel testing.
- b. **DNA Sequencing:** targeted next-generation sequencing (NGS) panels for pharmacogenes; whole-exome or whole-genome sequencing (WES/WGS) can identify rare variants but require specialized interpretation.
 - **Sanger Sequencing:** Gold standard for confirming specific variants.
 - **Next-Generation Sequencing (NGS):** Sequences entire genes or exomes/genomes. Used for discovering new variants and comprehensive testing.
- c. **Panel testing/array:** multiplex panels covering many clinically actionable alleles (often chosen to include common functional alleles for key genes).
- d. **Copy number and structural analysis:** specific assays for CYP2D6 CNV and hybrid alleles.
- e. **HLA typing:** sequence-based or high-resolution typing for immune risk alleles.
- f. **Phenotyping tests:** functional assays (e.g., probe drugs to measure enzyme activity) are less commonly used clinically but helpful in research.
- g. **Clinical integration tools:** result standardization (star allele nomenclature), translation to phenotype (poor/normal/rapid metabolizer), guideline recommendations (dose adjustments, alternative therapies), and CDS/EHR alerts (Qahwaji et al., 2024).

Consortium for the study of Pharmacogenetic in Africa Population (*Nigerian Example*)

Genetic polymorphisms of CYP2C19 in the Hausa, Igbo and Yoruba Populations of Nigeria Objectives of the study

This study investigates the distribution of genotype and allele frequencies of *CYP2C19* in the three major Nigerian ethnic groups in a bid to:

- Ensuring improve patient treatment and promote rational drug use in the country
- Contributing towards a pharmacogenetic data base for African population to assist in the development of new drug products through design and execution of clinical trials
- Evaluating the evolutionary relatedness of Nigerian population with other Populations



Drug Metabolising Enzymes (DMEs)

Phase I enzymes involved in addition of a functional group

- e.g. mixed function oxidases such as cytochrome P450s

Phase II enzymes involved in conjugation of compounds

- e.g. glutathione conjugation by the glutathione transferases

DMEs- Activation of drugs and Inactivation of metabolites

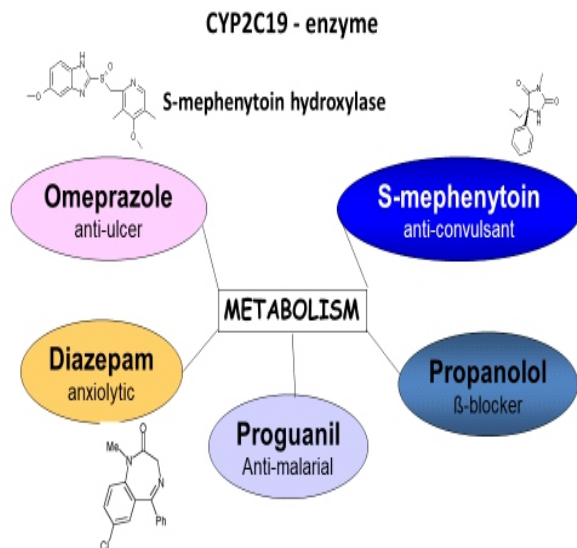


Figure 5: Some CYP2C19 Substrates

CYP2C19 – the gene

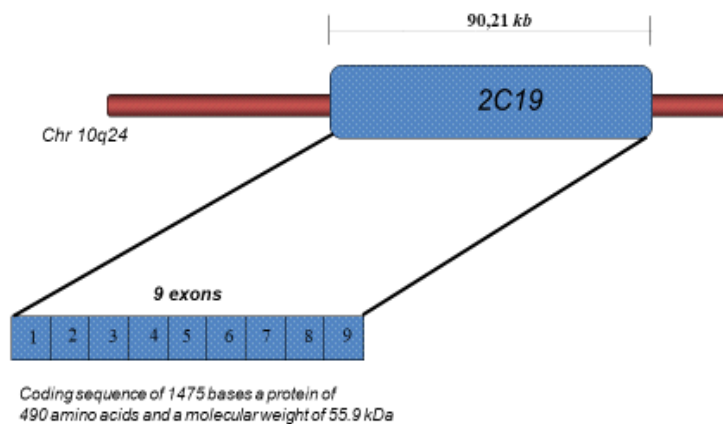


Figure 6: CYP2C19 Gene

CYP2C19 - polymorphisms

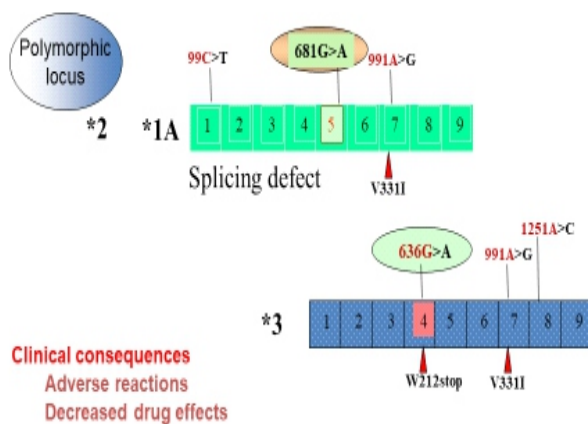


Figure 7: CYP2C19 Gene polymorphism

Methodology- AIBST SOP

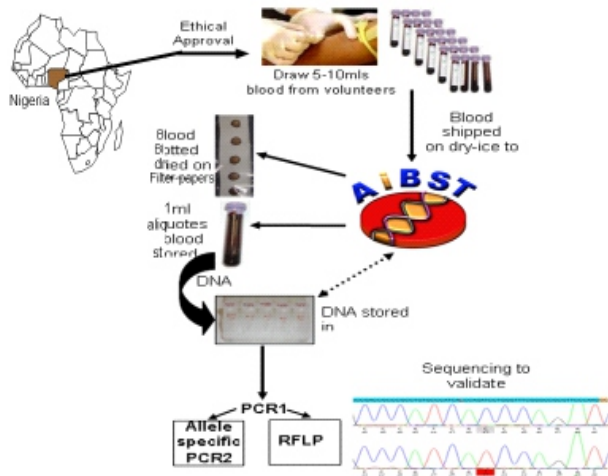


Figure 8: Summary chart of study procedures

Table 2: PCR Primers and Restriction enzymes used in Genotyping for CYP2C19*2/*3

Gene (allele)	Primers	PCR conditions	Restr. enzymes	Agarose gel fragment pattern
CYP2C19*2	F 5'-AATTACAACGAGCTTGGC-3' R 5'-TATCACTTTCCATAAAAGCAAG-3'	35 cycles of Denaturation 94 °C 10 seconds, Annealing 53 °C 10 seconds, Extension 72 °C 20 seconds	SmaI	3% w/v gel 120 +49 bp (wt) 169 bp (mt)
CYP2C19*3	F 5'-TATTATTATCTGTTAACTAATATGA-3' R 5'-ACTTCAGGGCTTGGTCAATA-3'	35 cycles of 94 °C 10 seconds, 53 °C 10 seconds, 72 °C 20 seconds	BamHI	3% w/v gel 233 + 96 bp (wt) 329 bp (mt)

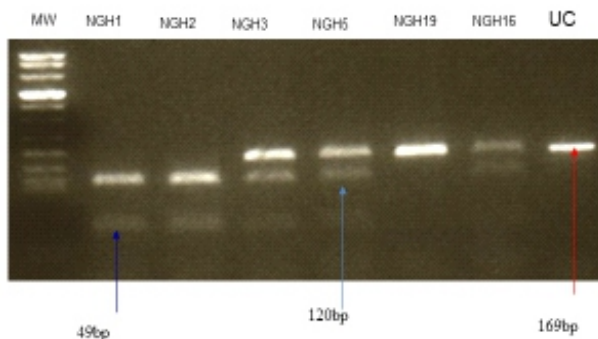


Figure 9: Results of Digestion with SmaI for CYP2C19*2 genotyping in Nigerian population (NGH1, NGH2, NGH3, NGH5, NGH19, NGH15) Abbreviations, UC- Undigested control, MW – Molecular weight marker

Table 3: CYP2C19 genotypes and allele frequencies in Hausa, Igbo and Yoruba ethnic groups of Nigeria (N=299)

Genotypes/Allele frequencies CYP2C19*X X=allele no	Hausa N/total (%)	Igbo N/total (%)	Yoruba N/total (%)
EM *1/*1	75/97 (75.8)	51/101 (50.5)	80/101 (80)
IM(heterozygous) *1/*2	20/97 (20.6)	42/101 (41.6)	21/101 (21)
PM *2/*2 *2/*3	2/97 (2.1) 0	8/101 (8) 0	0/101 (0) 0
Alleles CYP2C19*1 CYP2C19*2 CYP2C19*3	170/194 (88) 24/194 (12) 0	144/202 (71) 58/202 (29) 0	181/202 (90) 21/202 (10) 0

NON-FUNCTIONAL ALLELES →PM

frequencies of individuals showing poor enzyme metabolism'

Caucasian 3-5%, Africans 3-5% Asians 13-23% African

Americans 8%, Nigerians 4% (phenotyping)

Conclusion

In conclusion, the genotype status of *CYP2C19* showed variations within the Nigerian populations studied but consistent with previous phenotyping study and comparable to other African population previously studied.

2.8. Best Global Practices in Personalized Medicine

- Guideline-Driven: adopt and adapt published guidelines from authoritative bodies by using CPIC, DPWG, and FDA PGx labeling guidelines to translate genetic test results into actionable prescribing recommendations.
- Pre-emptive Testing: Implementing broad PGx panel testing integrated into the Electronic Health Record (EHR) with clinical decision support (CDS) alerts that fire when a relevant drug is prescribed. Pre-emptive multigene panels placed in the EHR before prescribing can improve timeliness of decision support; reactive single-gene testing remains common for targeted drug decisions.
- Interdisciplinary Teams: Involving clinical pharmacists, geneticists, genetic counselors, and physicians to manage and interpret PGx data.
- Workforce Education: continuous medical education for prescribers, pharmacists, and laboratorians; pharmacist-led PGx programs often accelerate implementation.
- Patient Education: Ensuring patients understand the purpose, benefits, and limitations of genetic testing.

- Quality control and accreditation: clinical labs offering PGx testing should meet accreditation/quality standards, validated assays (including CNV detection and HLA typing), and standardized reporting.
- Addressing Ethical Issues: Robust policies on informed consent, data privacy, and protection against genetic discrimination.
- Equity and population relevance: incorporate local/population allele frequency data to avoid misclassification; ensure access and affordability for underserved populations (Chenoweth et al., 2020).

2.9. The Future of Pharmacogenomics and Personalized Medicine

- Whole Genome Sequencing as Standard of Care: The cost of WGS will continue to fall, making a person's complete genome a standard part of their medical record.
- Artificial Intelligence (AI) and Machine Learning: AI will be used to integrate multi-omics data (genomics, proteomics, transcriptomics) with clinical data to predict drug response with unprecedented accuracy. Polygenic risk scores and ML models that integrate genetic + clinical + environmental data to predict drug response.
- Integration with multi-omics: combining genomics with transcriptomics, proteomics, metabolomics, and microbiome data to refine predictions of response and toxicity.
- Pharmacogenomics in Drug Development: Pharmaceutical companies will use PGx to design smarter clinical trials, leading to more effective drugs with better safety profiles, often paired with a

companion diagnostic test.

- Global Equity: A major focus will be on expanding PGx research in underrepresented populations (like those in Africa) to ensure the benefits of personalized medicine are accessible to all, preventing a genomic divide.
- Regulatory and reimbursement changes: increasing inclusion of PGx in drug labels, payer coverage for tests with demonstrated clinical utility, and formalized pathways for evidence generation (pragmatic trials) (Chenoweth et al., 2020).

2.10. *Limitations, Barriers, and Ethical Considerations*

- Evidence gaps: not all gene–drug pairs have high-quality randomized trial evidence; benefit may be stronger for some drugs (e.g., oncology, anticoagulation) than others.
- Interpretation complexity: rare variants, structural variants, gene–gene interactions, and non-genetic factors complicate phenotype prediction.
- Cost and access: infrastructure, testing cost, and reimbursement are major barriers, especially in low- and middle-income countries.
- Data privacy/consent: genomic data require robust consent, privacy protections, and clear plans for re-use and storage.
- Equity risks: if reference databases and guidelines are built primarily from European-ancestry datasets, misclassification or reduced accuracy in other populations can occur, underlining the need for

population-specific research and guideline adaptation.

3.0 SUMMARY OF SOME OF MY CONTRIBUTIONS TO KNOWLEDGE

The gene–drug complex is significant because it explains how human genetic variation shapes drug response. Understanding this interplay has major implications: it improves treatment outcomes by tailoring drug choice and dose to each patient’s genetic profile, ensuring maximum effectiveness. It reduces adverse drug reactions by identifying patients at risk of toxicity or hypersensitivity, such as HLA-linked antibiotic reactions. On a wider scale, it provides a public health benefit by enhancing positive therapeutic outcomes and preserving the effectiveness of existing medicines. Ultimately, the gene–drug complex serves as nature’s prescription, a scientific pathway to safer, more effective, and sustainable individualized therapies.

Mr. Vice Chancellor, Sir, my contributions to the knowledge base on pharmacogenomics research focus on the following areas:

- 3.1. Establishment of a biobank and pharmacogenetics database of African populations.*
- 3.2. Allele and genotype frequencies of cytochrome P450 2B6 and 2C19 genetic polymorphisms in the Nigerian populations: Possible implications on anti-retroviral and anti-malarial therapy*
- 3.3. Cytochrome P450 2D6 (CYP2D6) Genotype and Phenotype Determination in the Nigerian Populations*
- 3.4. Arylamine N-acetyltransferase 2 (NAT2) single-nucleotide polymorphisms’ frequencies in Nigerian populations.*
- 3.5. Glutathione-S-transferase (M1 and T1) polymorphisms in Nigerian populations.*

3.6. The CYP2B6 Gene Polymorphism and Phenotypic Correlation of Efavirenz-Based Combination Therapy Among the Niger Delta Ethnic Population: Implications in Modern Pharmacogenomics

3.1 Establishment of a biobank and pharmacogenetics database of African populations

The paper reports the establishment of the first biobank and pharmacogenetics database representing multiple African populations. This initiative responds to the lack of biobanking and genomic resources in developing countries despite advances in pharmacogenetics in developed regions. Pharmacogenetics and genomics research have experienced great advances over the past decade, as witnessed by the completion of the human genome in 2003. The field has been driven by the belief that understanding the human genome, that of pathogens, and interindividual genetic variability would result in radical advances in medicine. Anticipated measurable end points include increased targets for which drug discovery campaigns could be initiated, increased understanding of human susceptibility to disease and variability in drug response, hence development of diagnostic tools to realize individualized treatment where drugs would be given to patients in whom they are predicted to work, and at doses predicted to be safe.

CITATION:

Matimba, A., Oluoka, M. N., Ebeshi, B. U., Sayi, J., Bolaji, O. O., Guantai, A. N., & Masimirembwa, C. M. (2008). Establishment of a biobank and pharmacogenetics database of African populations. *European Journal of Human Genetics*, 16, 780–783.

AIM: To collect and store DNA samples from diverse African ethnic groups and use them to generate baseline allele frequency data for genes important in drug metabolism. The ultimate goal is to support personalized medicine, improve drug response prediction, and foster pharmacogenetics research capacity in Africa.

CONCLUSION: The biobank represents a significant step in building biomedical infrastructure in Africa by providing one of the most diverse collections of DNA samples from African populations to date. While challenges such as ethical considerations, sample anonymization, and population stratification remain, the resource lays the groundwork for clinical applications in drug prescription, treatment optimization, and future genotype–phenotype research.

European Journal of Human Genetics (2008), 1–4
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www.nature.com/ejhg



LETTER

Establishment of a biobank and pharmacogenetics database of African populations

European Journal of Human Genetics advance online publication, 2 April 2008; doi:10.1038/ejhg.2008.49

Pharmacogenetics and genomics research has experienced great advances over the past decade as witnessed by the completion of the human genome in 2003 (www.genome.gov/HGP). The field has been driven by the belief that understanding the human genome, that of pathogens, and interindividual genetic variability would result in radical advances in medicine. Anticipated measurable end points

samples were collected from 50 to 100 adult volunteers from ethnic groups in Nigeria, Kenya, Tanzania, Zimbabwe and South Africa (Figure 1). Portions of each blood sample were used to prepare DNA, blot on filter paper or store at -80°C . The biobank consists of 1488 DNA samples from nine ethnic groups (Yoruba, Hausa, Ibo, Luo, Kikuyu, Maasai, Shona, San and Venda). The utility of the biobank was illustrated by studying the frequency distribution of some polymorphisms of known phenotype characteristics (www.imm.ki.se/CYPalleles, <http://louisville.edu/medschool/pharmacology/Human.NAT2.pdf>) of six genes (Table 1) important in drug metabolism (CYP2B6, 2C19, 2D6, GSTM, GSTT and NAT-2). Genotyping was carried out using established PCR–RFLP methods.^{4–10} A database cataloging the samples and the genotype results was designed using Microsoft Access and Visual Basics software packages (<http://www.aibst.com/biobank.html>) and access is currently limited to authorized individuals involved in the research projects.

The stratification of populations based on allele frequency data was evaluated using principal component analysis using the program SIMCA P+ (www.umetrics.com).

3.2. Allele and genotype frequencies of cytochrome P450 2B6 and 2C19 genetic polymorphisms in the Nigerian populations: Possible implications on anti-retroviral and anti-malarial therapy.

The study was carried out to investigate the distribution of polymorphic CYP2B6 and CYP2C19 alleles and genotype frequencies in the three major Nigerian ethnic groups in order to evaluate their implications on therapeutic outcome. Three hundred unrelated subjects from the three major Nigerian ethnic groups of Hausa, Ibo, and Yoruba who consented to the study were genotyped for CYP2B6*6, CYP2C19*2, and *3 alleles using polymerase chain reaction/restriction fragment length polymorphism (PCR/RFLP) and DNA sequencing techniques. The frequencies of the CYP2B6*6 poor metabolizer (PM) genotype (15631G>T (Q172H) and/or 18053A>G (K262R) were not significantly different ($p>0.05$) occurring at 21% in Hausa, 17% in Ibo, and 17% in Yoruba, while the corresponding allele frequencies were 42, 36, and 42%, respectively.

CITATION:

BU Ebeshi, OO Bolaji, and CM Masimirembwa (2011). Allele and genotype frequencies of cytochrome P450 2B6 and 2C19 genetic polymorphisms in the Nigerian populations: Possible implications on anti-retroviral and anti-malarial therapy. International Journal of Medicine and Medical Sciences, 3(6); 193-200.

AIM:

This was with a view to establishing the possible impact of these polymorphisms on the treatment of malaria and HIV/AIDS, which are prevalent diseases in Nigerian populations.

CONCLUSION:

The allelic distribution of CYP2B6*6 appears comparable in the Nigerian populations and other African populations but significantly higher than the Caucasian and Asian populations, indicating that Nigerian populations may be at risk of adverse reactions if given a similar dose of CYP2B6 substrate (for example, efavirenz), as in Caucasians and Asians. Also, the genotype status of CYP2C19 showed variations within the Nigerian populations studied, most especially the 8% PM frequency obtained for the Ibo, with possible implications on sickle cell anaemia chemoprophylaxis with proguanil.

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Full Length Research Paper

Allele and genotype frequencies of cytochrome P450 2B6 and 2C19 genetic polymorphisms in the Nigerian populations: Possible implication on anti-retroviral and anti-malarial therapy

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Accepted 7 December, 2010

The study was carried out to investigate the distribution of polymorphic *CYP2B6* and *CYP2C19* alleles and genotype frequencies in the three major Nigeria ethnic groups in order to evaluate their implications on therapeutic outcome. Three hundred unrelated subjects from the three major Nigerian ethnic groups of Hausa, Ibo and Yoruba who consented to the study were genotyped for *CYP2B6**6, *CYP2C19**2 and *3 alleles using polymerase chain reaction/restriction fragment length polymorphism (PCR/RFLP) and DNA sequencing techniques. The frequencies of the *CYP2B6**6 poor metabolizer (PM) genotype (15631G>T (Q172H) and/or 18053A>G (K262R)) were not significantly different ($p>0.05$) occurring at 21% in Hausa, 17% in Ibo and 17% in Yoruba while the corresponding allele frequencies were 42, 36, and 42%, respectively. The frequencies of the *CYP2C19* PM genotype (*CYP2C19**2/*2) were significantly different ($p>0.05$) and found to be 2.1% in Hausa, 8% in Ibo, and 0% in Yoruba, while for the intermediate metabolizer (IM) genotype (*CYP2C19**1/*2), the frequencies were 20.6, 41.6 and 20.8%, respectively. The defective *CYP2C19**3 allele, prevalent in oriental populations, was not detected in this study. The *CYP2B6**6 allele frequency was being evaluated for the first time in Nigerian populations with no significant variation amongst the three major ethnic groups but occurs at a high frequency. The PM genotype of frequency, *CYP2C19**2/*2 showed significant variations in Nigerian populations, especially the 8% found in the Ibos.

Key words: Genetic polymorphisms, CYP2B6, CYP2C19, genotyping, Nigerian populations.

3.3. Cytochrome P450 2D6 (CYP2D6) Genotype and Phenotype Determination in the Nigerian Populations.

This study was conducted to determine the genotype and phenotype frequencies of CYP2D6 in the Nigerian populations with a view to evaluating the inter-individual and inter-ethnic differences in drug response of clinically useful drugs, which are substrates of CYP2D6. Genotyping of CYP2D6 mutant alleles by polymerase chain reaction (PCR) and sequencing techniques was performed on 300 Nigerians, comprising (Hausa=98), (Ibo=101), and (Yoruba=101). Eighty-six (86) subjects were phenotyped with dextromethorphan as the probe drug. The frequency of the defective CYP2D6*4 allele was found to be 2% in Hausa, 8% in Ibo, and 3% in Yoruba subjects. The frequency of the CYP2D6*17 allele was not significantly different ($P>0.05$), occurring 18%, 14% and 22% in the Hausa, Ibo, and Yoruba, respectively. Also, the frequency of the CYP2D6*29 allele was found to be 10%, 20% and 10% in the Hausa, Ibo, and Yoruba, respectively. Of the 86 phenotyped subjects, 3 (3.5%) were identified as poor metabolizers (PM), 68 (79%) were extensive metabolizers (EM), and 15 (17.4%) were intermediate metabolizers (IM).

CITATION: BU Ebeshi, OO Bolaji, CM Masimfrembwa (2011). Cytochrome P450 2D6 (CYP2D6) Genotype and Phenotype Determination in the Nigerian Populations. Asian Journal of Pharmaceutical and Health Sciences. 1(2); 47-54.

AIM: This study aims to evaluate the CYP2D6 allele frequencies and phenotype in the three major ethnic populations of Nigeria, comprising Hausa, Ibo, and Yoruba.

CONCLUSION: The overall allelic frequencies of CYP2D6*3, *4, *9, *10, *17, and *29 found are unique to the Nigerian populations analyzed. These population-specific differences are consistent with the other African populations. The phenotype data show a close correlation with the genotype data and agree with previous phenotyping studies in Nigerian populations.



Cytochrome P450 2D6 (CYP2D6) Genotype and Phenotype Determination in the Nigerian Populations

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ABSTRACT

This study sets to determine the genotype and phenotype frequencies of CYP2D6 in the Nigerian populations with a view to evaluate the inter-individual and inter-ethnic differences in drug response of clinically useful drugs, which are substrate of CYP2D6. Genotyping of CYP2D6 mutant alleles by polymerase chain reaction (PCR) and sequencing techniques were performed on 300 Nigerians comprising of (Hausa-98), (Ibo-101) and (Yoruba-101). Eighty six (86) subjects were phenotyped with dextromethorphan as the probe drug. The frequency of the defective CYP2D6*4 allele was found to be 2% in Hausa, 8% in Ibo and 3% in Yoruba subjects. The frequency of CYP2D6*17 allele was not significantly different ($P=0.05$) occurring at 18%, 14% and 22% in the Hausa, Ibo and Yoruba respectively. Also, the frequency of CYP2D6*29 allele was found to be 10%, 20% and 10% in the Hausa, Ibo and Yoruba, respectively. Of the 86 phenotyped subjects, 3 (3.5%) were identified as poor metabolizers (PM), 68 (79%) were extensive metabolizers (EM) and 15 (17.4%) were intermediate metabolizers (IM). The mean and median $\log_{10}$ dextromethorphan/dextrorphan ratios were 0.72 and 0.79 (SD=0.26) for PMs, -1.81 and -1.83 (SD=0.4) for EMs, and -0.51 and -0.62 (SD=0.37) for IMs, respectively. The overall allelic frequencies of CYP2D6*3, *4, *9, *10, *17, and *29 found are unique to the Nigerian populations analyzed. The phenotype data show a close correlation with the genotype data. These population-specific differences are consistent with findings in other African populations.

3.4. Arylamine N-acetyltransferase 2 (NAT2) single-nucleotide polymorphisms' frequencies in Nigerian populations

The study was carried out to investigate the distribution of arylamine N-acetyltransferase 2 (NAT2) allele frequencies associated with slow acetylation in healthy individuals from the three major Nigerian ethnic groups, comprising Hausa, Ibo, and Yoruba. The single nucleotide polymorphisms (SNPs) in the NAT2 gene from three hundred unrelated subjects comprising, Hausa (N=98), Ibo (N=101) and Yoruba (N=101) who consented to the study were genotyped by polymerase chain reaction/restriction fragment length polymorphism (PCR/RFLP) techniques (481 C>T, 590 G>A) and DNA sequencing (191 G>A, 857 G>A). The allele frequencies of the investigated SNPs indicate that NAT2*4, wild-type (34%; 95% confidence interval (CI): 22-38%) is the most prevalent allele in Hausa, NAT2*6, G>A (29%; 95% CI: 22-37%) is the most common in Ibo, while NAT2*5, 481 C>T (33%; 95% CI: 21-37%) is the most recorded in Yoruba populations. The most prevalent alleles in the three populations are the wild type, NAT2*4, and the defective alleles, NAT2*5 and NAT2*6. The frequencies of NAT*7, 857 G>A, and NAT2*14, 191 G>A are relatively low in these populations, except for the 11% recorded for NAT2*14 in the Ibo population.

CITATION:

BU Ebeshi1, OO Bolaji, and CM Masimirembwa (2011). Arylamine N-acetyltransferase 2 (NAT2) single-nucleotide polymorphisms' frequencies in Nigerian populations. African Journal of Pharmacy and Pharmacology Research, 1(1); 001-006.

AIM:

This study aims to investigate the distribution of arylamine N-acetyltransferase 2 (NAT2) allele frequencies associated with

slow acetylation in healthy individuals from the three major Nigerian ethnic groups, comprising Hausa, Ibo, and Yoruba.

CONCLUSION:

The findings of high frequency of the NAT2 variants, which contribute to slow acetylators phenotypes in this study, may predispose the Nigerian populations to adverse drug effects when treated with isoniazid. Thus, the application of NAT2 genotyping may be important in optimising the treatment of the resurgent cases of tuberculosis in HIV/AIDS patients in Nigeria, in particular, and indeed Africa in general.

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Full Length Research Paper

Arylamine N-acetyltransferase 2 (NAT2) single nucleotide polymorphisms' frequencies in Nigerian populations

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Accepted 28 February, 2011

The study was carried out to investigate the distribution of arylamine N-acetyltransferase 2 (NAT2) allele frequencies associated with slow acetylation in healthy individuals from the three major Nigeria ethnic groups comprising of Hausa, Ibo and Yoruba. The single nucleotide polymorphisms (SNPs) in the NAT2 gene from three hundred unrelated subjects comprising, Hausa (N=98), Ibo (N=101) and Yoruba (N=101) who consented to the study were genotyped by polymerase chain reaction/restriction fragment length polymorphism (PCR/RFLP) techniques (481 C>T, 590 G>A) and DNA sequencing (191 G>A, 857 G>A). The allele frequencies of the investigated SNPs indicates that NAT2*4, wild-type (34%; 95% confidence interval (CI): 22-38%) is the most prevalent allele in Hausa, NAT2*6, G>A (29%; 95% CI: 22-37%) is the most common in Ibo while NAT2*5, 481 C>T (33%; 95% CI: 21-37%) is the most recorded in Yoruba populations. The most prevalent alleles in the three populations are the wild type, NAT2*4 and the defective alleles, NAT2*5 and NAT2*6. The frequencies of NAT*7, 857 G>A and NAT2 *14, 191 G>A are relatively low in these populations except for the 11% recorded for NAT2*14 in the Ibo population. Overall, the NAT2 defective alleles (NAT2*5, *6, *7 and *14) were found to be 66%, 72% and 71% in the Hausa, Ibo and Yoruba populations, respectively. The single nucleotide polymorphism frequencies of NAT2 gene showed a higher prevalence of the slow acetylators alleles in this study, which was in agreement with previous findings in some African populations.

Key words: Pharmacogenetics; N-acetyltransferase 2; Genotyping, Nigerian populations

3.5. Glutathione-S-transferase (M1 and T1) polymorphisms in Nigerian populations

Glutathione-S-transferases (GSTs) are critical detoxification enzymes that protect cells from reactive oxygen species and xenobiotics. Polymorphisms in GST genes, particularly GSTM1 and GSTT1, can lead to enzyme deficiencies, increasing susceptibility to cancers such as lung, bladder, and colorectal cancer. Despite Nigeria's ethnic diversity, data on GST polymorphisms in its major populations (Hausa, Ibo, and Yoruba) are limited. This study was conducted to address this knowledge gap and provide insight into the genetic predisposition to disease in Nigerian populations.

CITATION:

Ebeshi, B. U., Bolaji, O. O., & Masimirembwa, C. M. (2011). Glutathione-S-transferase (M1 and T1) polymorphisms in Nigerian populations. Journal of Medical Genetics and Genomics, 3(4), 56–60.

AIM: To investigate the distribution of GSTM1 and GSTT1 null genotype frequencies among the three major Nigerian ethnic groups (Hausa, Ibo, and Yoruba) using polymerase chain reaction (PCR) genotyping, and to assess their potential implications for disease susceptibility.

CONCLUSION: The study found relatively high frequencies of homozygous null genotypes for both GSTM1 (23–37%) and GSTT1 (35–42%) across Nigerian ethnic groups, with significant inter-ethnic differences, particularly for GSTM1 in the Ibo group. These frequencies are generally lower than those reported in Asians but higher than in Caucasians for GSTT1. The findings suggest that Nigerians, and Africans more broadly, may be better protected than Asians and Caucasians against certain cancers linked to GSTM1 deletion, but the high GSTT1 null frequency may increase susceptibility to bladder carcinogenesis. Overall,

GST polymorphisms in Nigerians carry both protective and risk implications for cancer development, underscoring the need for population-specific genetic and clinical research.

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Full Length Research Paper

Allele and genotype frequencies of cytochrome P450 2B6 and 2C19 genetic polymorphisms in the Nigerian populations: Possible implication on anti-retroviral and anti-malarial therapy

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Accepted 7 December, 2010

The study was carried out to investigate the distribution of polymorphic *CYP2B6* and *CYP2C19* alleles and genotype frequencies in the three major Nigeria ethnic groups in order to evaluate their implications on therapeutic outcome. Three hundred unrelated subjects from the three major Nigerian ethnic groups of Hausa, Ibo and Yoruba who consented to the study were genotyped for *CYP2B6**6, *CYP2C19**2 and *3 alleles using polymerase chain reaction/restriction fragment length polymorphism (PCR/RFLP) and DNA sequencing techniques. The frequencies of the *CYP2B6**6 poor metabolizer (PM) genotype (15631G>T (Q172H) and/or 18053A>G (K262R) were not significantly different ($p>0.05$) occurring at 21% in Hausa, 17% in Ibo and 17% in Yoruba while the corresponding allele frequencies were 42, 36, and 42%, respectively. The frequencies of the *CYP2C19* PM genotype (*CYP2C19**2/*2) were significantly different ($p>0.05$) and found to be 2.1% in Hausa, 8% in Ibo, and 0% in Yoruba, while for the intermediate metabolizer (IM) genotype (*CYP2C19**1/*2), the frequencies were 20.6, 41.6 and 20.8%, respectively. The defective *CYP2C19**3 allele, prevalent in oriental populations, was not detected in this study. The *CYP2B6**6 allele frequency was being evaluated for the first time in Nigerian populations with no significant variation amongst the three major ethnic groups but occurs at a high frequency. The PM genotype of frequency, *CYP2C19**2/*2 showed significant variations in Nigerian populations, especially the 8% found in the Ibos.

Key words: Genetic polymorphisms, *CYP2B6*, *CYP2C19*, genotyping, Nigerian populations.

3.6. The CYP2B6 Gene Polymorphism and Phenotypic Correlation of Efavirenz-Based Combination Therapy Among the Niger Delta Ethnic Population: Implications in Modern Pharmacogenomics

Efavirenz, a cornerstone of first-line antiretroviral therapy (ART) for HIV, is primarily metabolized by the CYP2B6 enzyme. Genetic polymorphisms in the CYP2B6 gene can lead to wide inter-individual variability in drug metabolism, causing differences in plasma drug concentrations that may affect treatment efficacy and risk of adverse drug reactions. Despite the high burden of HIV in Nigeria, there is limited pharmacogenomic data on CYP2B6 polymorphisms in the Niger Delta ethnic population. This study was designed to bridge this gap and explore genotype–phenotype correlations relevant to personalized HIV therapy.

CITATION:

Bunu, S. J., Owaba, A. D. C., Vaikosen, E. N., & Ebeshi, B. U. (2022). The Cyp2b6 Gene Polymorphism and Phenotypic Correlation of Efavirenz-Based Combination Therapy Among the Niger Delta Ethnic Population: Implications in Modern Pharmacogenomics. Pharmacogenomics and personalized medicine, 15, 45–54.

AIM:

The main aim of the study was to evaluate genotype-phenotype-based correlation and incidence of genetic polymorphism of efavirenz blood levels among HIV/AIDS patients of the Niger Delta population.

CONCLUSION:

There was no significant difference ($p \leq 0.05$) between genotype and phenotype data for CYP2B6 polymorphism among the HIV/AIDS patients that participated in this study. Genetic polymorphism of the CYP2B6 gene is prevalent

among HIV/AIDs patients in the Niger Delta ethnic population on efavirenz-based HAART treatment, as the population having homozygous mutant gene or PM is >1% (6%). The study revealed significant frequencies of CYP2B6 polymorphisms associated with altered efavirenz metabolism among the Niger Delta population. Carriers of reduced-function alleles exhibited elevated plasma efavirenz levels, predisposing them to toxicity, while others with extensive metabolism profiles were at risk of subtherapeutic exposure.

The *Cyp2b6* Gene Polymorphism and Phenotypic Correlation of Efavirenz-Based Combination Therapy Among the Niger Delta Ethnic Population: Implications in Modern Pharmacogenomics

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Purpose: DNA polymorphism describes the difference between individuals, groups, or ethnicities, races, etc., in terms of their DNA sequences or phenotypes as relates to drug metabolism. Using predictive genotyping of drug-metabolizing genes, we can develop individuals' drug therapies that are less toxic and more effective. The main aim of the study was to evaluate genotype-phenotype-based correlation and incidence of genetic polymorphism of efavirenz blood levels among HIV/AIDS patients of the Niger Delta population.

Methods: A study questionnaire was designed to obtain patients' data, blood samples were obtained, plasma was separated from the serum using a centrifuge for 5 minutes at 4000 rpm for HPLC analysis, polymerase chain reaction (PCR) and restriction fragment length polymorphism (RFLP) analysis was conducted using *Bst*I endonuclease enzyme to digest the PCR amplicons. Standard efavirenz was used at 0.5, 1, 2, 4, 16 mg/L to construct a calibration curve. Data were analyzed with SPSS software using chi-square test at p -value ≤ 0.05 and Microsoft excel 2013, while PCR and RFLP results were obtained after 1% Agarose gel electrophoresis, respectively.

Results: Phenotypic results showed that the participants had different efavirenz plasma concentrations. Six subjects (12%) had efavirenz plasma levels below 0.10 mg/L, considered ultra-rapid metabolizers (UMs), 22 (44%) 0.10 mg/L to 0.90 mg/L, classified as extensive metabolizers (EMs), 19 (38%) had 1.0 to 3.9 mg/L and were noted as intermediate metabolizers (IMs), while 3 (6%) subjects showed efavirenz plasma levels from 4.0 mg/L to 6.0 mg/L, categorized as poor metabolizers (PM). RFLP results showed more than half of the population (56%) with a homozygous wild-type gene with CYP2B6*1*1 allele, 38% were CYP2B6*1*6 (heterozygous mutant) allele and 6% had homozygous mutant gene (CYP2B6*6*6 allele). Out of the 15 male subjects among the 50 patients that participated in the study, 8% were UM, 12% EM, 14% IM while no PM was observed, on the contrary, out of the 35 females participated in the study, 4% were observed as UM, 32% EM, 24% IM, while 6% were PM.

Conclusion: There was no significant difference ($p \leq 0.05$) between genotype and phenotype data for CYP2B6 polymorphism, among the HIV/AIDS patients that participated in this study. Genetic polymorphism of the CYP2B6 gene is prevalent among HIV/AIDS patients in the Niger Delta ethnic population on efavirenz-based HAART treatment, as the population having homozygous mutant gene or PM are >1% (6%).

Keywords: Antiretroviral, NNRTIs, CYP2B6, Genetic polymorphism, efavirenz, phenotype

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4.0. CONCLUDING REMARKS AND RECOMMENDATIONS

4.1 Concluding Remarks

The concept of the gene–drug complex underscores the intricate relationship between human genetic variability and therapeutic response, highlighting nature’s inherent blueprint for individualized medicine. By unraveling how specific genetic polymorphisms influence drug metabolism, efficacy, and toxicity, pharmacogenomics provides a scientific foundation for safer, more effective, and patient-centered therapy. As research advances, integrating genetic insights into clinical practice will not only minimize adverse drug reactions but also optimize treatment outcomes, ultimately transforming healthcare into a truly personalized discipline where therapy is tailored to the genetic makeup of each individual.

Mr. Vice Chancellor, Sir, my contributions have focused on strengthening the knowledge and evidence base through Pharmacogenomics, personalized medicine, and the future of individualized therapy. My work evidenced across these publications, has been foundational in establishing the field of pharmacogenomics within Nigeria and contributing significantly to the understanding of African genetic diversity.

Some of my key contributions include:

- a. Pioneering Population-Specific Pharmacogenomic Data: A primary contribution in the generation of the first comprehensive baseline allele and genotype frequency data for major drug-metabolizing enzymes (CYP2B6, CYP2C19, CYP2D6, NAT2, GSTM1, GSTT1) within the three largest Nigerian ethnic groups (Hausa, Igbo, and Yoruba). This data is crucial for understanding drug response variability in this population.*

- b. *Elucidating High Prevalence of CYP2B6*6 in Nigerians: my research consistently identified the high frequency of the CYP2B6*6 allele (~40% allele frequency) in Nigerian populations. This allele is linked to reduced metabolism of efavirenz (an anti-HIV drug), providing a genetic explanation for the higher plasma levels and increased risk of neuropsychiatric side effects observed in African patients on standard doses.*
- c. *Documenting Inter-Ethnic Variations within Nigeria: my work revealed significant inter-ethnic differences, such as the notably higher frequency of the CYP2C19*2/*2 poor metabolizer genotype in the Igbo population (8%) compared to the Yoruba (0%), which has direct implications for dosing drugs like proguanil (an anti-malarial prodrug).*
- d. *Characterizing Unique African Pharmacogenetic Alleles: I and my group confirmed the presence and frequency of African-specific alleles with functional consequences, such as CYP2D6*17 and CYP2D6*29 (which confer an intermediate metabolizer phenotype) and NAT2*14 (a slow acetylator allele), which are rare or absent in Caucasian and Asian populations.*
- e. *Linking Genotype to Phenotype: my research on CYP2D6 went beyond genotyping to include phenotyping using dextromethorphan, successfully demonstrating a correlation between the observed genotypes (e.g., prevalence of reduced-function alleles) and the metabolic phenotype in the study population, validating the functional impact of the genetic findings.*
- f. *Establishing a High Prevalence of Slow Acetylators: My research on N-Acetyltransferase 2*

(NAT2) showed a high prevalence of slow acetylator genotypes (66-72% across ethnic groups) in Nigerians. This is critical for predicting toxicity from drugs like isoniazid (for tuberculosis) and sulfonamides.

- g. Contributing to a Pan-African Biobank and Database: I was a key contributor to the seminal project that established a Biobank and Pharmacogenetics Database of African populations, providing DNA samples from the Nigerian cohorts. This resource is vital for future research and consolidating African genomic data (Page 42).*
- h. Identifying GST Polymorphism Frequencies: My work documented the frequencies of glutathione-S-transferase null genotypes (GSTM1*0 and GSTT1*0) in Nigerians, which are important for understanding susceptibility to oxidative stress, environmental toxins, and certain cancers.*
- i. Clinical Research on HIV Therapy: I supervised and co-authored a clinical study that directly applied pharmacogenetic principles, genotyping HIV/AIDS patients on efavirenz and correlating CYP2B6 polymorphisms with their plasma drug levels and metabolic phenotypes (UM, EM, IM, PM), moving from population genetics to clinical application.*
- j. Building Research Capacity and Collaboration: My publications, often in collaboration with institutions like the African Institute of Biomedical Science and Technology (AiBST) and Obafemi Awolowo University, represent a significant effort in building local research capacity and fostering international collaboration in pharmacogenomics within Africa.*

4.2 Recommendations

My contributions provide a powerful evidence base for action. Some recommendations for clinicians, researchers, policymakers, and educators to realize the promise of "Divine Blueprint for Personalized Pharmacotherapy" include the following:

1. For Clinical Practice and Policy:

a. Implement Targeted Pharmacogenetic Testing:

Develop clinical guidelines that recommend or mandate pre-emptive or reactive genotyping for high-impact gene-drug pairs in Nigeria and across Africa. The strongest initial candidates based on this research are:

- CYP2B6 testing before initiating ARV-based HIV therapy. Poor metabolizers (*6/*6) may require a dose reduction to avoid toxicity, while ultra-rapid metabolizers may risk therapeutic failure.*
- CYP2C19 testing for patients requiring Proguanil (for malaria prophylaxis, especially in sickle cell patients) or Clopidogrel (increasingly used for cardiovascular disease). Poor metabolizers will not effectively activate these prodrugs.*
- NAT2 testing before Isoniazid therapy for tuberculosis to identify slow acetylators at high risk of peripheral neuropathy, allowing for dose adjustment or enhanced monitoring.*

b. Develop Africa-Specific Dosing Guidelines: Regulatory bodies (e.g., NAFDAC in Nigeria)

and Ministries of Health should commission the development of pharmacogenomics-based dosing guidelines tailored to the allele frequencies prevalent in African populations, which differ significantly from other continents.

- c. Integrate PGx into National HIV/TB Programmes: Given the high burden of HIV and TB, national treatment programmes should pilot and adopt PGx testing as a tool to improve drug safety, adherence, and treatment outcomes, ultimately improving programme efficacy and sustainability.*

2. For Education and Training:

- a. Revise University Curricula: Integrate pharmacogenomics into the core curricula of medical, pharmacy, nursing, and medical laboratory science schools across Africa. Healthcare professionals must be literate in interpreting genetic test results.*
- b. Launch Continuous Professional Development (CPD): Create workshops and certification programs for current healthcare providers to update their knowledge on PGx and its application in clinical decision-making.*
- c. Public Awareness Campaigns: Educate the public on the concept of pharmacogenomics to build trust and understanding, emphasizing that genetic testing is a tool for personalized care, not for discrimination.*

3. For Research and Infrastructure:

- a. Expand the Biobank and Database: Support and fund the expansion of the African pharmacogenomics biobank (as initiated on Page 43). This must include more ethnic groups, longitudinal clinical data, and next-generation sequencing to discover novel Africa-specific variants.***
- b. Focus on Clinical Outcome Studies: Move beyond frequency and association studies to robust clinical trials that demonstrate the cost-effectiveness and improved health outcomes (e.g., reduced hospitalizations, reduced adverse drug reactions) of implementing PGx-guided therapy in Africa.***
- c. Invest in Affordable Genotyping Capacity: Develop and validate low-cost, rapid genotyping platforms suitable for resource-limited settings. This could include focused panels for the most clinically relevant variants in Africa.***
- d. Explore Gene-Disease-Drug Interactions: Research should explore the complex interplay between genetics, infectious diseases (HIV, Malaria, TB), and non-communicable diseases, which often co-exist in African patients, creating unique gene-drug-disease interactions.***

4. For Ethical, Legal, and Social Implications (ELSI):

- a. Develop Strong Data Protection Frameworks: Establish clear national and regional policies***

- on patient genetic data ownership, privacy, confidentiality, and protection against misuse (e.g., by insurers or employers).*
- b. Prevent "Ethics Dumping": Ensure that international collaborations are equitable, with African researchers leading projects and African communities benefiting from the research outcomes. Capacity building and local ownership are non-negotiable.*
 - c. Promote Equitable Access: Strategies must be developed to ensure that the benefits of personalized medicine do not exacerbate existing health inequalities and are accessible to all segments of society.*

In conclusion, my works and contribution has provided the essential map of Nigeria's pharmacogenetic landscape. The imperative now is to use this map to navigate towards a healthcare system where the "Gene-Drug Complex" is understood and leveraged to deliver Nature's intended prescription: safe, effective, and Personalized drug therapy for every patient.

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Mr. Vice Chancellor Sir, this Lecture will be incomplete without giving credit to all those who contributed towards making me what I am today.

Foremost among them is the Lord God Almighty. He provided the needed inspiration, knowledge, wisdom, strength, good health, and provision to drive the process of making me an Excellent Man.

My parents (Elder Umaru Agakpa Ebeshi and Ene (Mrs) Adi Ebeshi) are exceptional beings created for the good work of nurturing me into greatness. They both believed in me, and invested everything they had to ensure I am catered for. My Mum (of blessed memory) looked forward to a day as this, but was called to glory in 2021. My mother laboured and sacrificed her leisure and personal fulfillment for us her children to have good education. May God bless her memory and grant her eternal rest. Amen.

My dear wife (Dr Mrs. Elizabeth Ebeshi) has been a strong pillar of support for me all these years. I remembered we got married in 2006 while already on a PhD programme and have to travelled out of the country a number of times to carry our my resaecrch but all through she encouraged me to remain steadfast during my PhD training whilst she took care of the children and the home front. I'm also indebted to our four lovely children (Benison, Flora, Grace and Lauren) for their support. You hear them complain, Daddy was always travelling and the couldn't understand why?

My Teachers and Mentors at all levels of my academic life are all gratefully acknowledged for the various roles they played towards my career growth. Notable ones include:

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2. MR. NANZIP SAMBO- my secondary school principal at Government Secondary School Nasarawa provided a great school environment for me to blossom.
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4. MR. DANIEL ALI- my secondary school Form 5 Mathematics Teacher at Government Secondary School Nasarawa provided a good foundation of scholarship and learning.
5. MRS. MARTHA ALI - my childhood Bible Lesson Teacher who taught me that true greatness is found only in God, and its pursuit is the only essence of life.
6. DR SAM ABU IBRAHIM-my mentor and a trailblazer
7. PROF. HELEN KWANASHIE- my teacher and B.Pharm project supervisor at A.B.U. Zaria.
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9. PROF. O.O. BOLAJI- my major PhD supervisor at O.A.U Ife
10. PROF. C. MASIMIREMBWA- my co-supervisor for PhD at AiBST, Harare, Zimbabwe
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12. PROF. K.S. GAMANIEL- my teacher, my mentor and former DG, NIPRD, Abuja.
13. DR. U.S. INYANG - my former DG of NIPRD, Abuja.

14. PROF. A.O. OGUDIANI- my teacher at O.A.U ife.
15. PROF. T.A. OLUGBADE- my teacher at O.A.U ife.
16. PROF. C.O. ONYEJI- my teacher at O.A.U. ife

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Sunday Amuneni) have been very supportive and encouraged me all the way.

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Well-wishers, too numerous to list here, had positive impact on my growing years and career. I remain grateful to them all. May God bless you all, and raise help for in your time of need. AMEN.

PHOTO HIGHLIGHTS



Family



Mama



Papa

NDU 71ST INAUGURAL LECTURER



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ABOUT THE INAUGURAL LECTURER

PROFESSOR BENJAMIN UMAR EBESHI is a Professor of Pharmaceutical Chemistry in the Department of Pharmaceutical & Medicinal Chemistry, Faculty of Pharmacy, Niger Delta University, Wilberforce Island, Bayelsa State. He is the first Professor of Pharmaceutical Chemistry of Nasarawa State origin, and the former Dean, Faculty of Pharmacy NDU.

He was born on the 5th January 1969 in Onaka, Nasarawa Local Government Area of Nasarawa State to the family of Elder Umaru Ebeshi Agakpa and Ene Adi Anyuobe Ebeshi (of blessed memory).

PROF. EBESHI is an accomplished Researcher, Lecturer and a Pharmacist, especially with regard to his work on understanding the Pharmacogenomics of Interindividual and Interethnic variations in drug responses; Pharmacokinetic principles of drug action and therapeutic drug monitoring. His operational research efforts have been geared towards the use of some Analytical techniques such as HPLC/MS, GC/MS, and Chemical assays for chemical characterization of drug products, food substances, and environmental toxicants. He has a vision that the faculty can leverage the concept of operational research to produce simple drug products for the society.

PROF. EBESHI had his education (Qualification, Date, and Institution) as follows:

- (i) First School Leaving Certificate, June 1981. Central Primary School, Loko, Nasarawa State, Nigeria
- (ii) GCE O/Level, June 1986. Government Secondary School, Nasarawa, Nasarawa State, Nigeria
- (iii) IJMB A/Level, October 1988. School of Preliminary Studies, Keffi, Nasarawa State, Nigeria.

- (iv) Bachelor of Pharmacy (Unclassified) 1st March 1993.
Ahmadu Bello University, Zaria, Kaduna State, Nigeria.
- (v) Master of Science (Pharmaceutical Chemistry), 10th October 2004. Obafemi Awolowo University, Ile-Ife, Osun State, Nigeria.
- (vi) Doctor of Philosophy (PhD) (Pharmaceutical Chemistry), 12th December 2010. Obafemi Awolowo University, Ile-Ife, Osun State, Nigeria.

PROF. EBESHI is professionally qualified and was registered on 13th October 1995 as a Pharmacist with Certificate of Registration as Pharmacist (Cert #: 006871) by the Pharmacists Council of Nigeria. He has the current (2026) Annual License to Practice Pharmacy.

PROF. EBESHI has the following career history:

- (i) Intern Pharmacist: March 1993 - November 1993.
General Hospital, Pankshin, Plateau State, Nigeria.
- (ii) National Youth Service: December 1993 - October 1994. General Hospital Funtua, Katsina State.
- (iii) Pharmacist I: November 1994 - February 1995.
General Hospital, Shendam, Plateau State, Nigeria.
- (iv) Superintendent Pharmacist: March 1995 - May 1998. Systrend Pharmacy Ltd., Abuja, Nigeria
- (v) Junior Research Fellow - Research Fellow II: June 1998 - January 2008. National Institute for Pharmaceutical Research and Development (NIPRD), Idu Industrial Area, Abuja, Nigeria.
- (vi) Lecturer I: 28th February 2008 - 30th September 2011.
Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmacy, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria.

- (vii) Senior Lecturer: 1st October 2011 - 30th September 2014. Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmacy, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria.
- (viii) Associate Professor: 1st October 2014 - 30th September 2018. Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmacy, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria.
- (ix) Professor: 1st October 2018 – till date. Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmacy, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria.
- (x) Professor (Sabbatical): Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmaceutical Sciences, Federal University of Lafia, Nasarawa State, Nigeria. March 1, 2025 to February 28, 2026.
- (xi) Adjunct Professor: Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmaceutical Sciences, Bayelsa Medical University Yenagoa, Bayelsa State, Nigeria. January 2025 to date.

PROF. EBESHI is a recipient of several scholarships, fellowships, and grants from reputable organizations around the world, as follows:

- (i) Postgraduate Study Scholarship: 2011 (Federal Government of Nigeria).
- (ii) African Institute of Biomedical Science & Technology (AiBST) Visiting Researcher/Trainee Fellowship: May to December 2005. AiBST, Harare, Zimbabwe.
- (iii) University of Nairobi/NASENI Visiting Researcher/Trainee Fellowship: May to December

2009. University of Nairobi, Nairobi, Kenya.
- (iv) Travel Grant by NIPRD to attend the first African Conference on Drug Metabolism and Development held at Johannesburg, South Africa, May 21-24, 2007.
 - (v) Travel Grant by the International Society for the Study of Xenobiotics (ISSX) to attend the IXth International Meeting of the International Society for the Study of Xenobiotics (ISSX) held at Istanbul, Turkey, September 4- 8, 2010.
 - (vi) Grant-in-Aid of Research: 2005. National Institute for Pharmaceutical Research and Development, Abuja, Nigeria.
 - (vii) NDU Tetfund Research Grant (2014/2015): June 2016. Tertiary Education Trust Fund, Abuja, Nigeria.
 - (viii) Travel Grant by the World Health Organization/Food and Agriculture Organization to participate in the Joint F A O / W H O E x p e r t Committee on Food Additives (J E C F A) that reviewed and finalized four WHO documents on the application of Hazard Analysis Critical Control Points (HACCP) on food safety in Africa held at Entebbe, Uganda, October 27 – 31, 2015.
 - (ix) Prof Ebeshi was appointed by the Director-General, Food and Agriculture Organization (FAO), into the Joint FAO/WHO expert committee on food Additives (JECFA) since 2017 to date. To this end, he joined other scientific experts of JECFA at its 88th meeting to draft monographs and technical reports series on some veterinary drug residues in edible tissues of some Animal species at the FAO Headquarters, Rome-Italy, October 21-31, 2019 and October 2-15, 2021.

PROF. EBESHI, a highly respected Academic and administrator and has held several positions in the course of his career, such as:

- (i) Acting Head: Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmacy, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria. 5th November 2014 - 4th June 2016.
- (ii) Faculty Examination Officer: Faculty of Pharmacy, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria. May 2011 to June 2013.
- (iii) Faculty of Pharmacy Representative to Senate: February 2012 to October 2014.
- (iv) Member, NDU General Studies Board, 2012 to 2014
- (v) Member of University Senate: February 2012 – Date. Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria.
- (vi) Member of 7th Governing Council: Pharmacists Council of Nigeria (PCN), Abuja, Nigeria: 2021 to 2025.
- (vii) Member of Council: Pharmaceutical Society of Nigeria (PSN), Abuja, Nigeria: 15th February 2021 to 14th February 2025.
- (viii) Dean: Faculty of Pharmacy, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria. 15th February 2021 to 14th February 2025.
- (ix) Pioneer Dean: Faculty of Pharmaceutical Sciences, Federal University of Lafia, Nasarawa State, Nigeria. 10th March 2025 to 28th February 2026.

Besides his academic and research endeavors, PROF. EBESHI has engaged in several community services, such as:

- (i) Member/Secretary: Sept. 2004 - June 2006. Research and Development Committee, Academic Staff Union of Universities (Research Institute Sector), NIPRD, Abuja.
- (ii) Chairman, Nigerian Association of Pharmacists in Academia (NAPA), NDU Chapter, 2016-2020.
- (iii) External Examiner for Postgraduate programmes to several Universities in Nigeria
- (iv) Manuscript Reviewer for many local and foreign Journals: 2006 - Date.
- (vi) Chairman or Member of various Committees at present and previous workplaces.

PROF. EBESHI is a member of the Pharmaceutical Society of Nigeria, a member of the Nigeria Association of Pharmacists in Academia (NAPA), and a member of the International Society for the Study of Xenobiotics (ISSX).

With over 20 years' experience combining top-level scientific research in Pharmaceutical Chemistry, Drug Discovery, and design, PROF. EBESHI has to his credit: supervision of several undergraduate projects, Four (4) M.Sc. theses, one (1) Ph.D. thesis; and over Fifty (50) articles in reputable International and local Journals, including monographs and technical reports, in addition to attendance at several conferences, seminars, and workshops.

PROF. EBESHI is married to his dear wife, DR ELIZABETH D. EBESHI, and they are blessed with four lovely children – Benison, Flora, Grace, and Lauren.

PROF. EBESHI has travelled widely to countries in Africa, Europe, Asia, and the Middle East, and enjoys listening to music and discussing issues affecting Nigeria and the rest of the World

PROF. EBESHI is a man of several parts- a pharmacist, an academic, a researcher, a mentor, a devoted Christian, an amiable gentleman, and an administrator.

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