UNIVERSITY OF PORT HARCOURT

A TALE OF "OLD WINE IN NEW WINESKINS"

An Inaugural Lecture

By

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ORDER OF PROCEEDINGS

2.45 pm. Guests are seated
3.00 pm. Academic Procession begins

The Procession shall enter the CBN Centre of Excellence Auditorium, University Park, and the Congregation shall stand as the Procession enters the hall in the following order:

Academic Officer
Professors
Deans of Faculties/School
Dean, School of Graduate Studies
Provost, College of Health Sciences
Lecturers
University Librarian
Registrar
Deputy Vice-Chancellor, Research and Development
Deputy Vice-Chancellor, Academic
Deputy Vice-Chancellor, Administration
Vice-Chancellor

After the Vice Chancellor has ascended the dais, the Congregation shall remain standing for the University of Port Harcourt Anthem.

The Congregation shall thereafter resume their seats.

THE VICE CHANCELLOR'S OPENING REMARKS.
The Registrar shall rise, cap, invite the Vice Chancellor to make his opening remarks and introduce the Lecturer. The Lecturer shall remain standing during the Introduction.

THE INAUGURAL LECTURE

The Lecturer shall step on the rostrum, cap and deliver her Inaugural Lecture. After the lecture, she shall step towards the Vice Chancellor, cap and deliver a copy of the Inaugural Lecture to the Vice Chancellor and resume her seat. The Vice-Chancellor shall present the document to the Registrar.

CLOSING

The Registrar shall rise, cap and invite the Vice Chancellor to make his Closing Remarks.

The Vice Chancellor's Closing Remarks.

The Vice Chancellor shall then rise, cap and make his Closing Remarks. The Congregation shall rise for the University of Port Harcourt Anthem and remain standing as the Academic [Honour] Procession retreats in the following order:

Vice Chancellor
Deputy Vice Chancellor Administration
Deputy Vice Chancellor Academic
Deputy Vice Chancellor Research and Development
Registrar
University Librarian
Lecturer
Provost, College of Health Sciences
Dean, School of Graduate Studies
Deans of Faculties/School
Professors
Academic Officer

DEDICATION

I dedicate this lecture to the memory of my dad and mum, late Chief Dr. Dominic and Mrs. Ibikuroa Dominic Uyanga (Nee Benibo). I also extend this to my beloved husband Prof. Owunari A. Georgewill and my children: Miss Tamunoemi, Miss Tamunodein, and Miss Tamunobakam.

ACKNOWLEDGEMENTS

I wish to express with reverence my utmost gratitude to God. He from whom all goodness flows, for everything I am today and for everything I will be tomorrow, to Him be all the glory, honour and adoration, for without Him, I am nothing.

For eternity, I would be forever grateful to my beloved parents, Dr. and Mrs. Dominic Peter Uyanga, both of blessed memories, for the sacrifice made to ensure we all went through school. I am equally grateful to Sisi, late Mrs. Ineyi A. Georgewill, for making the sacrifice to groom her son, who later became my husband.

My beloved husband and friend, Prof. O.A. Georgewill, and our children, I appreciate you all for being there for me through thick and thin. I cherish you all very greatly for all your tremendous support and stability at the homefront. My dear husband, partner and mentor, I thank you also for being a dependable research partner. God bless you all.

My beloved siblings, Ima and Edozie, Unwana, Imo and Samuel, Dominic and Inaingo and Eme. Thank you for being a safe haven. My beloved nieces and nephews, God bless you all. I love you.

To my In-laws the Abraham Georgewills, especially, Sir, Hon. Justice B.A. Georgewill and family, the Benibo's and the Uyangas, I say thank you.

I acknowledge with gratitude all my teachers from primary school through university, for laying a great foundation academically that allowed me to excel.

Let me also appreciate my colleagues and staff in the Department of Pharmacology, Faculty of Basic Clinical Sciences, College of Health Sciences, University of Port Harcourt, Prof. Nelson Brambaifa (My PhD supervisor), Prof. (Mrs) O. O. Ebong, Prof. A. W. Obianime (My PhD supervisor), Late Prof. R.N.P. Nwankwoala, Late Prof. E. B. Dede, Prof. Didia (in whose tenure as provost of the College of Health Sciences, I was employed), Prof. Prince Unekwe (My external examiner at Masters level), Prof. I. M. Siminialayi (My supervisor at PHD level), Prof. H.D. Kagbo, Prof. Joshua Isirima, Dr. S. Eyaru, Dr. Joachim Odigie, Dr. Chinwe Obi (Ag. Head of Department), Dr S. Akoko, Dr. A.G. William Dr. Victor Ibubeleye, Mr Gogo Ogan, Mrs. Mathilda Deekor, Charity Jones, Tubobelem Ubani, Kabari, Biiragbara, Michael, Mina, Mike Aniyeloye and Mathilda. I am indeed very grateful to you all.

To Dr. Adikwu Elias, a reliable research partner, since our days in the Masters Programme,I say a profound thank you.

To all my students (Undergraduate and Post Graduate), past and present, I thank you.

To Prof. Ching F. Poh, Dr. Chinwe Anyanwu, Dr. Victor Ibubeleye, and Dr Okwudiri Anasiudu, I send heartfelt thanks for assisting with putting this together and proof reading.

To the inaugural series committee, I say a big thank you for your constructive criticisms, which enriched my work.

I have saved the best for the concluding segment of my appreciation, let me wholeheartedly and with utmost joy, thank the Vice-Chancellor Prof. Owunari Abraham Georgewill for giving me this opportunity to stand before you all to deliver

my inaugural lecture, Sir, I appreciate you greatly for providing the enabling environment for research to thrive in this university.

The Principal Officers, Provost, Deans and Heads of Departments, I say thank you for all your support.

Udeme Owunari Georgewill

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PREAMBLE

A Tale of "Old Wine in New Wineskins"

By Prof. Udeme Owunari Georgewill
Professor of Pharmacology, University of Port Harcourt

In the dead of the night on the 12th of June 1982, I remember sitting in the back of my parents' Peugeot 504, headed for Ikot Ayan Ediene in Akwa Ibom State, to bury my younger brother, Aniekanabasi Dominic Uyanga, who had just passed. Clutched in my mum's arms was his little body at 3 years of age, and dead silence in the car. The pain of losing their child was palpable. I wondered if I could have done anything about it, as my play partner had just died.. Perhaps I could help others someday. At that moment, I decided I was going to become a medical doctor.

My journey to Medical School began in the corridors of the Baptist Primary School, GRA Port Harcourt, and when my dad transferred his services to the University of Port Harcourt, I joined the University Demonstration Primary School (UDPS), and then proceeded to the University Demonstration Secondary School (UDSS), opting for the science courses. I then proceeded to the University of Calabar, where I studied Medicine and Surgery.

During my Housemanship, I developed an interest in Obstetrics and Gynaecology and radiology. However, after a particularly gruelling weekend call where we were on our feet for over 48hours, I decided against it. Interestingly, prior to graduation, I had developed an interest in Pharmacology, which is the branch of medicine that deals with the scientific study of drugs, under the tutelage of Professors Akpan and Braide, both of blessed memories. This was because I found it

particularly challenging with so many drug names to learn, mechanisms of actions etc, and there was never enough time to remember all of them. For someone who loves challenges, this led to my application for the post of lecturer II in the Department of Pharmacology, University of Port Harcourt, post- NYSC. It is worthy of note that Dr. O.A. Georgewill, now Vice Chancellor, was the Ag. Head of the Department at the time, and Prof. B. Didia was the Provost of the College. Interestingly, Prof. Uzoukwu of the O & G Department interviewed me for that position.

As is the tradition of the Department, I was encouraged to try my hand in different areas of pharmacology.

General and Autonomic pharmacology: The study of drugs affecting the autonomic system.

Endocrine Pharmacology: The study of drugs that influence the body's hormone system.

Immunopharmacology: The study of how drugs influence the immune system.

Toxicology: The study of the adverse effects of chemicals on living organisms.

Cardiovascular Pharmacology: Study of drugs affecting the heart and blood vessels etc.

Chemotherapy: refers to the therapeutic use of drugs to treat disease by specifically targeting and destroying rapidly dividing cells.

I found myself drawn to chemotherapy, where I eventually specialized.

My Ph.D. thesis, aimed at filling the literature gaps, was focused on establishing the toxicological implications of Disulfiram (an anti-alcoholism drug) and Copper Gluconate (Dietary Copper Supplement) combination for the treatment of cancer. This birthed my research journey into drug repurposing, the art of finding new uses for existing drugs. This concept, elegantly captured in the metaphor “old wine in new wineskins,” embodies the ingenuity of modern pharmacology: extracting renewed therapeutic value from known chemical entities to address emerging health needs.

INTRODUCTION

"No one sews a patch of unshrunk cloth on an old garment, for the patch will pull away from the garment, making the tear worse. Neither do people pour new wine into old wineskins. If they do, the skins will burst; the wine will run out and the wineskins will be ruined. No, they pour new wine into new wineskins, and both are preserved."
— Matthew 9:16–17

There are moments in every scholar's journey when science transcends the confines of the laboratory and becomes a mirror, reflecting not only what we know, but who we are. My journey into drug repurposing began not merely with an interest in drugs, but with a fascination for possibility, the possibility that a single molecule, long forgotten or familiar, might still hold secrets capable of transforming lives.

My odyssey into this fascinating field of drug repurposing did not begin in a high-tech laboratory abroad but right here, in our own environment, amidst the daily challenges of limited resources and boundless determination. In those early days of research, I was often struck by the paradox of abundance and lack, a world teeming with diseases waiting for cures, and a shelf full of drugs waiting for rediscovery. Could the answer to emerging health challenges lie, perhaps, in what we already have? That question became both my motivation and my mission.

Drug discovery, in its traditional form, which is the pathway for drug production from the laboratory to the consumer, is a long, costly, and uncertain voyage. One that often spans over a decade, consuming billions of dollars and ending, more often than not, in disappointment. Yet, in the midst of this

complexity lies a quiet truth: some of the answers we seek are already hidden within the medicines we know so well.

Every drug is screened during the preclinical stages for a few disease conditions with the most prevalent and perhaps urgent at the time tested during the clinical trials, while the findings of its possible use in other conditions is documented giving credence to our forefathers' claims that one herb can cure quite a few diseases. This realization forms the heartbeat of my lecture today.

This inaugural lecture explores the science, philosophy, and promise of drug repurposing through the lens of pharmacological innovation and translational research. Drawing from over 19 years of academic and research experience, this lecture highlights the dynamic intersection between traditional pharmacology and contemporary medical discovery. The lecture examines case studies and experimental findings that demonstrate how established drugs—originally developed for one indication—can be effectively repositioned to combat diseases beyond their initial scope.

Focusing on antimalarial therapies, anti-inflammatory agents, and antioxidant modulators, the lecture showcases the extensive research conducted in the Department of Pharmacology, University of Port Harcourt. These studies reveal how familiar agents such as ketotifen, promethazine, and chlorpheniramine—long used for allergic and inflammatory conditions—have shown promising synergistic effects when combined with artemisinin-based compounds in malaria treatment. Through systematic evaluation of pharmacokinetic profiles, toxicity patterns, and therapeutic indices, these works reaffirm the relevance of repurposing as a cost-effective and sustainable approach to drug development.

Beyond the laboratory, the lecture highlights the public health implications of repurposing: enhancing drug accessibility, shortening development timelines, and mitigating the growing global burden of neglected and resistant diseases. This lecture also reflects on my journey as a researcher, teacher, and mentor—emphasizing the collaborative spirit, intellectual curiosity, and resilience that fuel scientific progress.

Ultimately, this lecture calls for a renewed commitment to innovation through reinvention, urging policymakers, scientists, and clinicians to embrace the paradigm of drug repurposing as a strategic bridge between knowledge and societal well-being. It is a celebration of pharmacology's enduring relevance—where old discoveries are not discarded but refined, reborn, and reimagined for a healthier future.

In sharp contrast to the Bible, which states that new wine should be poured into new wine skins, drug repurposing exemplifies a tale of old wine poured into new wineskins, i.e. established drugs reborn with fresh purpose and renewed promise.

As we begin this exploration of *A Tale of Old Wine in New Wineskins*, I invite you to walk with me through the corridors of research, reflection, and revelation — to rediscover how yesterday's medicines can still heal today's and tomorrow's diseases.

HISTORICAL BACKGROUND

Since the fall of man in the Garden of Eden, humanity has relentlessly sought remedies for ill health. Over millennia, this quest has evolved from crude herbal preparations and mystical potions into the highly sophisticated pharmaceutical enterprise we know today. Each era in this journey has reflected the

prevailing scientific understanding of its time, revealing humanity's enduring desire to overcome disease and prolong life.

I. Ancient Origins (Pre-19th Century)

In the earliest periods, medicine was deeply intertwined with nature and spirituality. Treatments relied largely on herbs, minerals, and animal products, passed down through generations by trial and observation. By the 16th and 17th centuries, the first official pharmacopoeias began to appear in Europe, standardizing medicinal preparations and laying the foundations for the systematic study of drugs. These ancient compendia marked the dawn of pharmacology as a discipline rooted in empirical evidence rather than mystical tradition.

II. The Dawn of Modern Drug Production (19th Century)

The 19th century ushered in a new scientific era. Chemists began isolating active principles from plants, giving rise to the concept of the "pure compound." A remarkable example was the isolation of quinine from cinchona bark in 1820, revolutionizing the treatment of malaria. Around this time, small apothecaries began to evolve into mechanized pharmaceutical workshops, setting the stage for the industrial-scale drug production that would define the next century.

IIa OTHER AREAS OF RESEARCH

These also guided my journey into other areas of research, as I set off to scientifically validate some of the claims of our forefathers, concerning our naturally gifted plants and their healing potential.

Earlier in my research career, and even today, I explored natural products with pharmacological potential. Together with Georgewill (2008), I demonstrated that the leaf extract of

Pseudocedrela kotschyi (Dry Zone Cedar, also known as Tuna (Hausa), emi gbegi (Yoruba)(Fig 1), African medicinal tree in the mahogany family, used in traditional remedies for malaria, infections, fever, pain, etc.) effectively lowered blood glucose (sugar) levels in diabetic rats.

Fig 1. Pseudocedrela kotschyi (Dry Zone Cedar)
https://share.google/f6lPLIarw9JO04NxX

We evaluated the anti-inflammatory and anti-arthritic activities of *Abrus precatorious* (Jequirity Bean or Rosary Pea also known as Ojuologbo in Yoruba, Oto-berebere in Igbo)(Fig.2) used locally in various illnesses for the treatment of malaria and bronchitis. Our findings revealed that the extract produced a 67.10 $\pm$ 2% inflammatory response when compared to 71.1 ±2% produced by the standard Acetyl Salicylic Acid. These findings explain the usefulness of this plant in the treatment of inflammatory disease conditions by traditional healers. Also, its anti-arthritic action was comparable to that of the standard and significantly inhibited adjuvant induced arthritis displaying significant antiinflammatory effects ($p<0.05$).

(Georgewill & Georgewill 2008; Georgewill & Georgewill 2009).

The effect of Abrus precatorious was also evaluated on blood glucose(sugar) concentration of Alloxan induced diabetic albino wistar rats. At a dose of 200mg/kg, a significant($p<0.05$) reduction in blood glucose concentration was recorded with the mean blood glucose of the different groups of 4.0± 0.2 for the plant extract treated group as opposed to 7.0± 0.4 for the Diabetic control group. (Georgewill & Georgewill, 2009).

Fig 2. Abrus precatorious (Rosary Pea)
https://share.google/WmtWZEOlRSVCdvqi1

In a 2009 study, we evaluated the histaminic (allergen-like) effect of *Mucuna pruriens* (also known as Monkey tamarind or Velvet bean, Werepe in Yoruba) (Fig.3). Our results demonstrated that the extract produced identical effects on guinea pig ileum as histamine and histamine analogs. These results demonstrated that the spines of *Mucuna pruriens* possess histamine activities which may contribute to its itching and painful irritation effects (Georgewill *et al*, 2009).

Fig 3. Mucuna pruriens (Monkey Tamarind)
https://share.google/aYZhslPrwg9xAjDgF

A study on the Inhibitory Effects of Ascaris Suum (large roundworm of pigs) extract on gastric acid secretions in urethane-anaesthetized rats revealed that the extract at 14mg/kg reduced histamine-induced gastric acid secretion when compared with the control value. The extract also reduced basal gastric acid secretion, indicating that the extract of Ascaris Suum inhibits gastric acid secretion. (Nwankwoala *et al*, 2009)

Similarly, a pharmacokinetic study with Nwankwoala and Georgewill (2009) on adriamycin (Doxorubicin) used in the treatment of cancers, by interfering with DNA to stop cancer cell growth, provided new insight into its tissue distribution. We discovered persistent accumulation of the drug in the testes — a finding that may explain Adriamycin's testicular toxicity and its implications for male fertility.

In another study on the anti-arthritic activity of *Vernonia amygdalina* (bitterleaf) (Fig.4), the aqueous extract was found to significantly ($p<0.05$) reduce the inflammation elicited by

injecting croton oil into the rat paws at 200 and 400mg/kg on the 12th day, when compared with the standard indomethacin at 0.3mg/kg. Furthermore, the anti-arthritic effect was found to be dose-dependent. (Georgewill and Georgewill, 2009).

Fig 4. Vernonia amygdalina (bitterleaf) (https://share.google/POCpL9q1XIFpuqxt3)

A study on the anti-inflammatory effects of *moringa oleifera lam* (Fig.5), extract in rats, compared the extract with that of standard anti-inflammatory agents like indomethacin and hydrocortisone using the air-pouch model. The extract produced greater effects in acute inflammation than in delayed inflammation, and this was dose dependent [acuteIC_{50} = (399.30±5.43) mg/kg; delayed IC_{50}= (510/.26±4.53) mg/kg]. We posited that this effect was due to the short half-life of the active principle in the extract (Georgewill *et al*, 2010).

Fig 5. Moringa oleifera lam (https://share.google/z3BXlA4IvBeY8juSJ)

Over the years, we continue to lend our expertise to dealing with the different conditions that affect us as a people by looking for solutions in our God given natural resources. Our study on the antiseizure potential of *Parsonsia straminea* stemback (known locally as monkey rope) (Fig. 6), ethanol extract in mice using the pentylenetetrazol (PTZ), strychnine (STC), and electro shock (ES) models of antiseizure evaluation revealed antiseizure potential of the plant extract with no sedating effects. The effects noticed were more prominent in the male rats, and this could be due to the fact that female hormones such as estradiol play a role in the reduction of seizure threshold. Our findings are a possible promise for antiseizure management especially in low-income nations that have more access to traditional medicine. (Kemelayefa, *et al* 2022).

Fig 6. *Parsonsia straminea* (monkey rope)
https://share.google/fpI1kCOZJLRFXgABt

III. The 20th Century – The Age of Innovation

The 20th century was characterized by scientific revolution. The synthesis of aspirin by Felix Hoffmann at Bayer in 1897 marked the beginning of modern drug chemistry. World wars spurred pharmaceutical innovation, leading to the discovery of penicillin, sulfa drugs, and vaccines that saved millions of lives. The latter half of the century saw the birth of synthetic chemistry, antibiotics, psychotropics, and the first anticancer agents. Mass production, quality control, and regulatory oversight became the hallmarks of pharmaceutical development.

IV. The Modern Era (Late 20th Century to the Present)

Today, the landscape of drug discovery is defined by unprecedented technological advancement—genomic sequencing, high-throughput screening, molecular modelling, biotechnology, and personalized medicine. Yet, paradoxically, the cost, time, and risk associated with new drug development have never been higher. On average, it may take 15–20 years and billions of dollars to bring a single drug from conception

to market—many failing midways due to safety, efficacy, or economic constraints.

To overcome these challenges, the global pharmaceutical community has increasingly turned towards drug repurposing also known as drug repositioning, redirecting, or reprofiling. This innovative approach identifies existing drugs with known safety and pharmacokinetic profiles for new therapeutic indications, dramatically reducing both cost and development time (Andrews *et al.*, 2014).

DRUG REPURPOSING: CONCEPT, MECHANISMS, AND GLOBAL RELEVANCE

The evolution of pharmacology has always been driven by the pursuit of safer, more effective, and more accessible therapies. In this relentless quest, drug repurposing, also known as drug repositioning or redirecting, has emerged as a transformative approach in modern pharmacotherapy (Mishra *et al.*, 2024). It reflects an ingenious paradigm shift: instead of discovering entirely new compounds, researchers reimagine existing ones, exploring their potential beyond their original indications.

At its core, drug repurposing capitalizes on the pharmacological, toxicological, and clinical data already established for approved drugs. These compounds have cleared the most formidable hurdles in drug development, safety validation and pharmacokinetic profiling, making them valuable starting points for identifying new therapeutic applications. This strategy dramatically shortens development timelines, reduces financial costs, and, most importantly, accelerates the delivery of life-saving treatments to patients.

Mechanistically, drug repurposing draws upon both serendipity (luck) and science. While early examples were often accidental

discoveries, such as the realization that sildenafil, originally designed for angina, could treat erectile dysfunction, contemporary repurposing increasingly employs computational modelling, molecular docking (computational methods predicting how a small molecule binds to a larger molecule), network pharmacology, and artificial intelligence to predict new drug–disease interactions. These tools integrate genomic, proteomic, and clinical datasets, illuminating novel pathways and molecular targets for established drugs (Yingngam, 2024).

Globally, the relevance of drug repurposing cannot be overstated. It stands at the intersection of innovation, accessibility, and sustainability. In high-income countries, it offers a means to optimize research investments and respond rapidly to emerging diseases, as seen during the COVID-19 pandemic (Loayza, 2020). In low- and middle-income regions, particularly across Africa, drug repurposing presents a beacon of hope, a cost-effective strategy to combat endemic and neglected tropical diseases for which new drug development remains financially unviable (Figueroa *et al.*, 2021).

By breathing new life into old molecules, drug repurposing exemplifies the creative resilience of pharmacological science. It is in every sense, a scientific rebirth that transforms prior knowledge into novel therapeutic frontiers. In this dynamic process, pharmacologists stand not merely as scientists, but as visionaries, translating the wisdom of the past into the promise of the future.

Drug repurposing involves:

- Leveraging existing compounds to develop new formulations, dosing regimens or combination therapies (Nwaka and Hudson,2006)

- Employing computational (in silico-conducting scientific experiments and simulations on a computer) and experimental screening approaches to identify promising candidates
- Conducting targeted preclinical and Phase II clinical evaluations
- Utilizing open-access drug databases and molecular libraries to streamline discovery.

Drug Repurposing in the African Context

In Africa, where healthcare challenges are often compounded by limited resources, drug repurposing represents both a scientific opportunity and a social imperative. It offers a faster, cost-effective, and contextually relevant path to address pressing health burdens such as malaria, tuberculosis, neglected tropical diseases, and emerging drug resistance. Initiatives such as the Medicines for Malaria Venture (MMV) and the African Partnership for Drug Discovery have been instrumental in supporting local scientists through funding, training, and collaborative platforms.

The success of drug repurposing is best illustrated through history: Aspirin – Initially developed as an analgesic, was later found effective as an anti-inflammatory and antiplatelet agent. Penicillin – Originally a bacterial inhibitor, later adapted for diverse infections such as pneumonia and meningitis. Rapamycin – discovered as an antifungal compound, now used as an immunosuppressant and anticancer agent. Buprenorphine – developed for pain relief, later repurposed for managing opioid addiction are just a few examples.

DRUG REPURPOSING AND MALARIA

MALARIA BURDEN

Drug repurposing plays a critical role in malaria control, particularly in countries like Nigeria, which bears the highest global malaria burden with about 68 million cases and 194,000 deaths reported in 2021. The Sustainable Development Goal 3.3 aims to end epidemics of malaria and other diseases by 2030, a target that seems challenging given the current malaria statistics, especially in Africa, where 95% of cases and deaths occur.

Malaria remains a major health issue with significant morbidity and mortality among children under five and pregnant women (Nnamonu *et al.*, 2020). Nigeria alone accounts for nearly a third of global malaria deaths. The WHO's global strategy for malaria aims to reduce incidence and mortality by 90% by 2030 and eliminate malaria in at least 35 countries by accelerating efforts in prevention, diagnosis, and treatment.

In terms of treatment, artemisinin-based drugs such as injectable artesunate have become the preferred treatment for severe malaria in Nigeria, replacing quinine. Projects accelerating the adoption of injectable artesunate have dramatically reduced mortality rates from severe malaria—from 25% down to 1% in targeted Nigerian states. This drug is effective and safe, showcasing high cure rates (>90%) and rapid parasite clearance, making it central to Nigeria's malaria strategic plan to reduce mortality and parasite prevalence by 2025.

Other artemisinin-based combination therapies such as artesunate-mefloquine and artesunate-amodiaquine have also

proven highly effective in treating uncomplicated malaria with cure rates around 96–98%, rapid symptom resolution, and tolerability in children. The emergence of artemisinin resistance remains a concern, highlighting the need for continued surveillance and development of new treatment strategies.

New artemisinin combinations focus on triple artemisin- based combination therapies and supplements to combat rising resistance. A key trial right now is for a fixed-dose of artemether-lumefantrine-amodiaquine (ALAQ).

Furthermore, malaria vaccines like RTS, S/AS01(Trade name Mosquirix) and R21 (often cited as R21/Matrix-M), provide promising tools for prevention, particularly in high-risk populations like children (Ogieuhi *et al.*, 2024).

Thus, drug repurposing, exemplified by the use of artesunate and related therapies, alongside vaccines and preventive measures, forms a cornerstone in tackling the malaria burden, particularly in high-prevalence countries like Nigeria, to meet global eradication targets (Wu *et al*., 2025).

WHERE ARE WE?

Currently, Artemisinin-based Combination Therapies (ACTs) are the gold-standard of malaria treatment recommended by the World Health Organisation (WHO) for uncomplicated *Plasmodium falciparum* malaria. Artemisinin digests haemoglobin in the parasite producing toxic heme which reacts with artemisinin's endoperoxide bridge, producing potent carbon-centred free radicals that damage essential parasite proteins, disrupt calcium homeostasis, resulting in parasite death in the blood stage. ACT combines a fast-acting artemisinin derivative with a partner drug having a different

mechanism of action. This combination ensures rapid parasite clearance and helps delay the development of drug resistance. (Oguche *et al.*, 2014).

In Nigeria, the use of ACTs such as artemether-lumefantrine has increased significantly as the preferred treatment for uncomplicated malaria. Lumefantrine acts by disrupting the malaria parasites' detoxification of heme, leading to toxic heme build-up and parasite death. Advantages of ACTs include increased efficacy and cure rates, reduced malaria transmission, simpler treatment regimens, and effectiveness against multi-drug-resistant strains. Additionally, ACT reduces the chances of resistance, which means less relapse of infection after treatment.

Despite widespread use, ongoing efforts are necessary to ensure adherence to national treatment guidelines among healthcare providers for maximum effectiveness. Research is also underway into new combinations like triple ACTs to combat emerging resistance (Malaria Consortium, 2025; National Malaria Elimination Programme, Nigeria, 2024).

This approach aligns with Nigeria's National Malaria Strategic Plan 2021-2025, which aims to achieve a malaria-free Nigeria by reducing parasite prevalence and malaria-related mortality significantly by 2025. The plan emphasizes robust strategies encompassing prevention, diagnosis, and treatment, alongside strong partnerships and political commitment (National Malaria Elimination Programme Nigeria, 2024).

Overall, combination therapy with ACTs remains the most effective current practice in Nigeria for treating malaria and forms a critical part of the broader malaria control strategy for 2025 and beyond (Oguche *et al*., 2014).

WHAT HAVE OUR STUDIES FOUND?

Over the years, my scientific journey has taken me deep into the heart of one of Africa's most persistent health challenges — malaria. This journey began with a simple yet profound question: *Could the medicines we already know still hold untapped potential?*

My research has therefore focused extensively on Artemisinin-based Combination Therapies (ACTs), which remain the cornerstone of modern malaria treatment.

We kicked off this journey by comparing the efficacy of various ACTs in mice. Our studies revealed that among the various ACT regimens, dihydroartemisinin–piperaquine provided the most sustained cure with superior parasite clearance and no recrudescence (resistance) (Figs.7 & 8). By contrast, artemether–lumefantrine, though highly effective initially, showed a degree of delayed parasite resurgence over time (Fig.7). This discovery carried important implications for treatment durability and resistance management as our work literally flagged the development of recrudescence (resistance) in the parasites following screening with the ACT's which was a particularly worrisome development as chloroquine had been phased out and the artemisinins had been introduced as the "poster boy" in malaria treatment, moreso, the attendant implication of the possibility of the rapid rise in resistance in humans (Georgewill & Ebong, 2012).

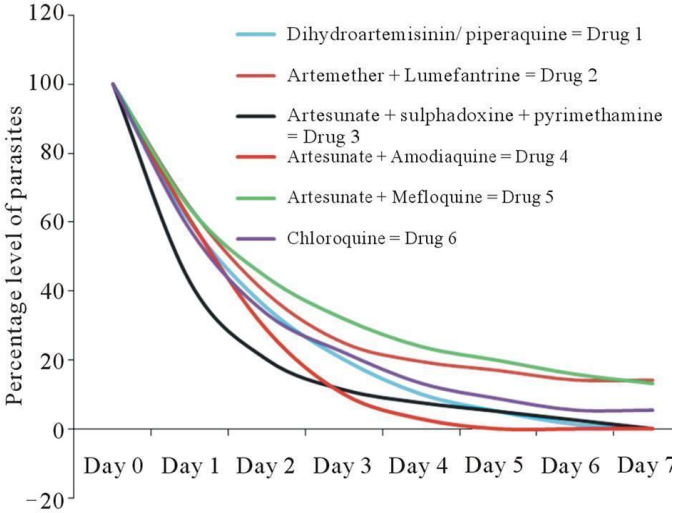
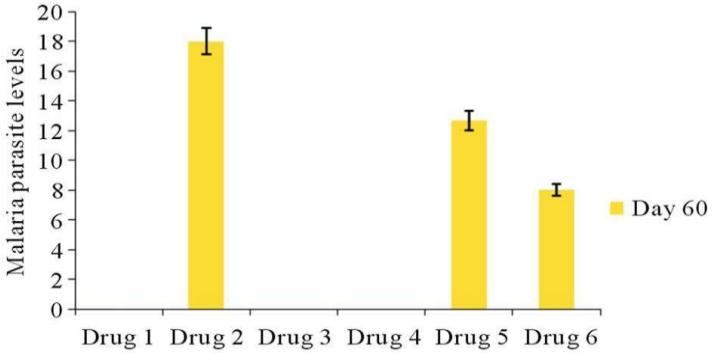


Figure 7. Daily mean percentage parasitemia levels in mice after administration of artemisinin combination therapies.



KEY: DRUG 1- Dihydroartemisinin and Piperaquine; Drug 2- Artemether + Lumefantrine; Drug 3-Artesunate +sulphadoxine+pyrimethamine; Drug 4- Artesunate + Amodiaquine; Drug 5 - Artesunate + Mefloquine; Drug 6 - Chloroquine

Figure 8. Mean parasite levels in mice on day 60 after treatment with different artemisinin combination therapies.

Beyond efficacy, safety has always remained a central concern in my research. Hence, we investigated the safety profiles of the widely used ACTs. Following treatments, the liver of the animals which received double clinical dose of the artemisinin combination, were excised and examined under the microscope, and no anomaly was detected. (Fig. 9).Importantly, artemether–lumefantrine exhibited a more favourable safety profile, with serious adverse reactions being rare (Fig.8) (Georgewill & Ebong, 2012).

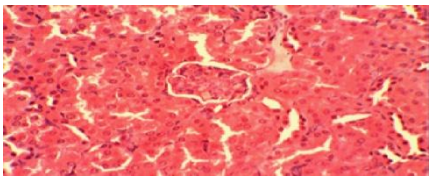


Fig. 9: LIVER OF TREATED MICE THAT RECEIVED DOUBLE CLINICAL DOSE OF Artemisinin Combination Therapy, SHOWING NORMAL CELLULAR ARCHITECTURE.

In 2019, we assessed malaria rapid diagnostic tests (RDTs) versus microscopy, in a bid to ascertain the validity of test tools to be used in our clinical evaluations. While RDTs provided faster results, microscopy proved far more accurate. This study reinforced the need for quality control and proper diagnostic standards — because even the most effective drug treatments and outcomes depend on the accuracy of diagnosis (Runmokun, Ebong, & Georgewill, 2019).

BREATHING NEW LIFE INTO OLD DRUGS: MY JOURNEY IN DRUG REPURPOSING AND PHARMACOLOGICAL INNOVATION

I. MALARIA

Armed with the findings above, we set out to explore possible antimalarial combinations that would reduce the rapid

development of resistance. The aim was to combine the drugs with agents that would affect the malaria parasite at different stages of the life cycle enhancing clinical outcomes (Fig. 10).

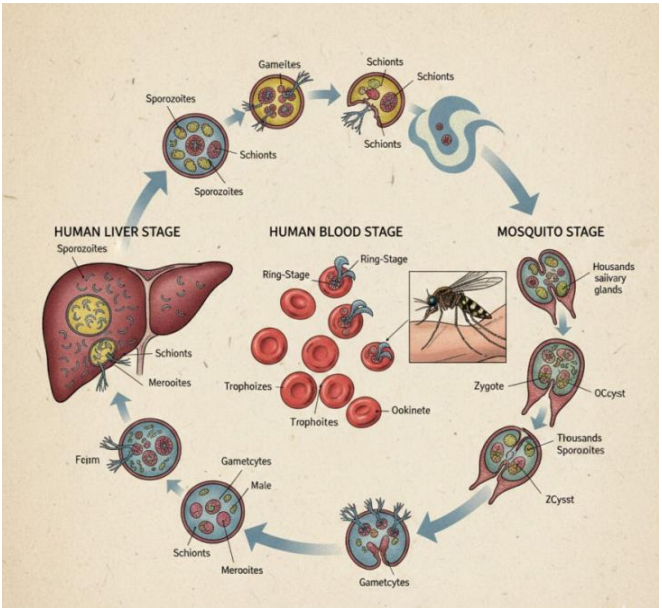


Fig. 10: Malaria Life Cycle (CDC- Malaria Life Cycle. https://gemini.google.com/share/ae0dd89aa16c)

Our first major study, conducted with colleagues Ezerioha and Adikwu in 2020, investigated the possibility of repurposing ketotifen, a well-known antiallergic drug, with artemether-lumefantrine (A/L) for malaria treatment in *Plasmodium berghei*-infected mice. Ketotifen is a non-competitive H1 histamine receptor blocker which stabilises mast cells. In malaria, it acts as a chemosensitizer that reverses chloroquine resistance. Its metabolite Norketotifen acts as the primary agent against blood-stage and liver-stage parasites. The results were both striking and encouraging — the combination produced enhanced antiplasmodial activity, reduced

parasitemia, improved survival, and corrected malaria-induced anaemia and lipid disturbances more effectively than either drug alone. Curative, suppressive and prophylactic activity of the combination revealed a 90% inhibition with mean survival time(MST) of 31.0 ±1.92, 91.9% and MST of 35.0±3.68 and a 99.3% inhibition with MST of 34.5 ± 2.18 days respectively. These findings suggested that ketotifen could indeed serve as a valuable *adjunctive therapy* in malaria management (Georgewill, Ezerioha, & Adikwu, 2020).

Through carefully designed animal models using *Plasmodium berghei*, we demonstrated that combining established antimalarial drugs such as artemether–lumefantrine with adjunct therapies like ivermectin (anti-helmintic used in the treatment of worms, which blocks essential protein transport to the endoplasmic reticulum, hindering parasite growth and survival in both asexual(blood) and sexual (gametocyte)stages) significantly enhanced parasitemia suppression and prolonged survival. Curative activity of the combination revealed a 90.9% inhibition and MST of 27.7±4.92 days (Table 1). Suppressive and prophylactic activity revealed a 93.0% inhibition and MST of 31.7 ±4.92 days and 91.1% inhibition and MST of 33.5±4.18 days respectively when compared to monotherapies or standard treatments (Georgewill, Liverpool, & Adikwu, 2020).

Table 1: Curative activity of artemether/lumefantrine/ ivermectin on Plasmodium berghei-infected mice.

Group	Parasitemia (%)	Inhibition (%)	MST
NC	45.8±1.04	-	9.30±0.85
CQ	6.73±0.18[a]	85.3	25.0±3.07[a]
IM	22.4±1.72[b]	51.1	15.2±4.29[b]
A/L	13.3±0.22[c]	70.8	20.0±3.05[c]
A/L/IM	4.16±0.68[a]	90.9	27.7±4.92[a]

NC: Negative Control; CQ: Chloroquine; IM: Ivermectin; A/L: Artemether / Lemafantrine; A/L/M: Artemether/Lumefantrine/Ivermectin; MST: Mean Survival Time; n=5; Data as mean ± SEM; [a] $p<0.001$ when compared to NC; [b] $p<0.05$ when compared to NC; [c] $p<0.01$ when compared to NC

Similarly, we investigated the antiplasmodial activity of artemether-lumefantrine-tinidazole combination (ALT). Tinidazole is a nitroimidazole antibiotic used to treat parasitic infections like giardiasis and amebiasis. It works as a prodrug, releasing toxic free radicals that damage the pathogen's DNA, leading to cell death (Georgewill *et al*, 2021). Our result revealed that the combination of ALT produced the best curative (99.1% inhibition on day 7), suppressive (99.9% inhibition and MST of 34.1±3.41 days), and antianemic activities compared to individual doses of DL/DP. Also, the combination increased mean survival time (MST), and restored the lipid profile of parasitized mice more than individual doses of DL/DP.

Also, the in-vivo antiplasmodial effect of desloratidine-dihydroartemisinin-piperaquine (DL/DP) combination was

assessed. (Georgewill *et al,* 2021). Desloratidine is a second-generation antihistamine; it is a selective, long-acting, non-sedating histamine, H1 receptor antagonist, meaning it blocks histamine from binding to H1 receptors in the body, preventing the allergic response (itching, sneezing, runny nose) without causing drowsiness, thus relieving symptoms of allergies and hives. Our result revealed that the combination of DL/DP produced the best curative (88.6% inhibition on day 7), suppressive (94% inhibition and MST of 35.4±3.33 days), and antianemic activities compared to individual doses of DL/DP. Also, the combination increased mean survival time (MST), and restored the lipid profile of parasitized mice more than individual doses of DL/DP.

In another 2021 study, we investigated sulfadoxine/pyrimethamine/doxycycline (S/P/D), a triple combination designed to enhance antimalarial effectiveness. Sulphadoxine-pyrimethamine acts by inhibiting folic acid synthesis thereby interrupting DNA/RNA synthesis. The malaria parasite is unable to absorb folic acid from its host and must synthesize its own. This drug, therefore, halts this process, starving the parasite and halting its growth. Doxycycline, on the other hand, is a broad-spectrum tetracycline antibiotic used to treat a range of infections, including acne and sexually transmitted infections, e.g. syphilis and gonorrhoea. It works by inhibiting bacterial protein synthesis thus halting the production of essential proteins needed for bacterial/parasite growth and survival. The results were outstanding — the regimen not only reduced parasitemia and prolonged survival, with 93.6% inhibition in the curative tests (Table 2) and 95.1% inhibition in the suppressive tests, but also corrected malaria-induced anaemia and dyslipidemia (abnormal level of fats/ lipids like cholesterol). It demonstrated how strategic drug repurposing

and combination could revitalize older therapies to combat modern resistance challenges (Georgewill & Adikwu, 2021)

Table 2. Curative effect of sulfadoxine/ pyrimethamine/ doxycycline on *Plasmodium berghei* infected mice

Treatment	%Parasitemia	%Inhibition	MST (Days)
NC	38.10±4.32	0.0	9.00±0.18
CQ	9.29 ±1.54[a]	75.6	29.62±2.43[a].
D	16.80±1.27[b]	55.9	18.85±1.19[b]
S/P	13.30±1.11[c]	65.1	23.81±3.47[c]
S/P/D	6.97±0.15[a]	81.7	32.05±3.69[a]

Values are expressed as ± SEM, n= 5, NC: Negative control, CQ: Chloroquine, D: Doxycycline, S/P: Sulfadoxine/Pyrimethamine,, MST: Mean Survival Time. [a] p<0.001, [b] p<0.05, [c] p<0.01 significant difference when compared to NC. SEM: Standard error of the mean

Building on that foundation, in collaboration with Joseph and Adikwu, I explored dihydroartemisinin-piperaquine (DP) combined with nitrofurantoin (NT). DP acts by damaging the parasite via oxidative stress and mitochondrial disruption, while piperaquine accumulates in the digestive vacuole, inhibiting heme detoxification, causing toxic heme buildup leading to parasite death. Nitrofurantoin, an old antibacterial agent, an antibiotic primarily used for uncomplicated lower urinary tract infections, seemed to enhance DP's efficacy through oxidative stress induction and inhibition of glutathione reductase, revealing a *novel mechanism* of antimalarial synergy. It works by inhibiting protein synthesis, DNA, RNA, and other vital enzymes, leading to bacterial cell death; this broad attack on various pathways makes bacterial resistance rare.

This study was particularly exciting — the DP–NT combination showed remarkable improvements across curative (91.41% inhibition), suppressive (93.94% inhibition), and prophylactic (97.40% inhibition) malaria models. These results

truly exemplify the spirit of drug repurposing — *old molecules finding new missions* (Georgewill, Joseph, & Adikwu, 2021).

We further investigated the triple combination of rifampicin, dihydroartemisinin, and piperaquine, which achieved sustained parasite clearance with minimal recrudescence(resistance), even in the face of rifampicin's known drug interaction potential. Rifampicin is an antibiotic used mainly for bacterial infections like tuberculosis and leprosy. It works by inhibiting bacterial protein synthesis, which kills the bacteria or parasite. The combination revealed a 97.22% inhibition in the curative test, 98.11% inhibition in the suppressive test, and 98.67% inhibition in the prophylactic test. This study, alongside our work with rifampicin and sulfadoxine-pyrimethamine (RIF/SP), which revealed a 96.89% inhibition in the curative test, a 97.70% inhibition in the suppressive test, reaffirmed rifampicin's potential for repurposing as an adjunct antimalarial agent — a valuable contribution in the fight against resistance (Georgewill & Dan-Jumbo, 2022; Dan-Jumbo & Georgewill, 2022).

SAFETY CONCERNS

In 2015, our work on disulfiram and copper gluconate combination revealed dose-dependent bone marrow and renal toxicity, offering cautionary evidence against indiscriminate combination use (Georgewill, Siminialayi, & Obianime, 2015) and set the stage for further toxicological studies on these combinations despite their proven efficacy.

We examined the effects of artemether-lumefantrine-tinidazole (A/L/T) on Liver and kidney function. Findings on the effect of the ACTs on the liver enzymes, revealed impaired liver function characterized by significantly increased aminotransferases, alkaline phosphate, lactate dehydrogenase,

gamma-glutamyl transferase and bilirubin levels with significantly decreased total protein and albumin levels occurred in healthy mice treated with T ($p<0.05$), A/L ($p<0.01$) and A/L/T ($p<0.001$) when compared to control (Table 3). A/L/T caused hepatocyte necrosis(death) in healthy mice. The use of A/L/T, we surmised, may impair liver function with prolonged use.

Table 3. Effect of artemether/lumefantrine/tinidazole on liver oxidative stress markers of healthy mice

Treatment	MDA nmole/mg protein	GSH µmole/mg protein	CAT U/mg protein	SOD U/mg protein	GPx U/mg protein
Control	0.12 ± 0.02	13.44 ± 0.73	25.61 ± 2.01	14.83± 2.51	15.34 ± 1.00
T	0.25 ± 0.04*	10. 03± 0.87*	20.83 ± 2.33*	11.01 ± 2.33*	11.62 ± 0.32*
A/L	0.36 ± 0.06**	6.35± 0.45**	16.32 ± 1.71**	9.00 ± 0.41**	8.14 ± 0.71**
A/L/T	0.50 ± 0.03 ᵀᵀ	4.22± 0.27 ᵀᵀ	12.84 ± 0.79 ᵀᵀ	6.01 ± 2.70 ᵀᵀ	5.40 ± 0.26 ᵀᵀ

T: Tinidazole, A/L: artemether/lumefantrine, A/L/T: Artemether/lumefantrine/tinidazole, MDA: Malondialdehyde, GSH: Glutathione, CAT: Catalase, SOD: Superoxide dismutase, GPx: Glutathione peroxidase, Data as mean ± SEM. n=6. p<0.05. **p<0.01. ᵀᵀ p<0.001 when compared to control. SEM: Standard error of mean.*

Similarly, findings on the kidney following administration of A/L/T revealed deranged enzymes (Table 4). The use of A/L/T as an antimalarial drug may be safe on the kidney, but long term use may cause kidney damage.

Table 4 Effect of artemether/lumefantrine/tinidazole on serum kidney biochemical markers of healthy and parasitized mice

Treatment	Healthy mice					
	Creatinine (mg/dL)	Urea(mg/dL)	Uric acid (mg/dL)	Creatinine (mg/dL)	Parasitized mice Urea (mg/dL)	Uric acid (mg/dL)
Control	0.50±0.03	7.27±0.04	1.37±0.13	0.56±0.09	7.41±0.07	1.42±0.24
T	0.97±0.07*	10.91±0.09*	2.89±0.09*	0.54±0.03	7.33±0.01	1.40±0.45
A/L	1.36±0.03**	13.83±0.36**	4.05±0.06**	0.57±0.07	7.30±0.05	1.38±0.67
A/L/T	2.67±0.08ᵀᵀ	20.14±2.71ᵀᵀ	7.76±0.72ᵀᵀ	0.53±0.01	7.40±0.08	1.35±0.44

*T: Tinidazole, A/L: Artemether/lumefantrine, A/L/T: Artemether/lumefantrine/tinidazole, Data as mean ± SEM, n=6, * p<0.05, ** p<0.01, ᵀᵀ p<0.001 when compared to control (Healthy mice), SEM: Standard error of mean*

Interestingly, while the combination was well-tolerated in infected mice, prolonged use in healthy ones caused signs of oxidative stress and mild organ impairment. This finding

reminded us that *even beneficial drugs can turn harmful when misused or overused*, emphasizing the delicate balance between efficacy and safety (Adikwu & Georgewill, 2021a, 2021b).

Similarly, we examined the toxicological implications of desloratadine/dihydroartemisinin/piperaquine (DL/D/P). While short-term use proved safe in infected mice, extended use in healthy ones induced hepatic and renal oxidative stress, signalling the need for careful clinical monitoring of novel combinations (Georgewill, Adikwu, & Ebong, 2022).

FROM THE LABORATORY TO THE CLINIC

We took our research findings to the clinic to evaluate some of these combinations in the treatment of uncomplicated malaria in humans. Our work on the clinical evaluation of the curative effects and haematological consequences of ivermectin, artemether-lumefantrine and their combination in malaria treatment was novel in that it provided valuable empirical evidence as day 14 showed a significant reduction in mean parasitemia($p<0.05$) in the group treated with the combination when compared with monotherapy with IM and AL(IM <AL <IM+ACT). Mean percentage recovery was 80% in the group that received the IM/AL combination as opposed to 40% in the group that received AL alone and 20% in the group that received IM alone. The combination demonstrated additive (double the response) when compared with AL alone and synergistic (3x the response) effects when compared with IM alone, on recovery rates, reduction in parasitemia and restoration of haematological effects. (Ogbe and Georgewill, 2023)

Similarly, our work on artemether-lumefantrine/tinidazole in plasmodium infected humans demonstrated statistically

significant recovery rates and showed a 100% recovery rate by day 14 compared to 88% and 92% for AL and tinidazole, respectively. Also recorded were restored antioxidant defenses,reduced oxidative stress and normalised lipid profiles. (Oni and Georgewill, 2024).

DRUG REPURPOSING IN OTHER CONDITIONS

I. DICHLORVOS POISONING

In a related 2020 study with Durojaiye, we evaluated the antidotal effect of Promethazine on dichlorvos (an organophosphate insecticide, effective against many pests, widely used to control flies, roaches, and crop pests in homes, storage and agriculture) poisoning (Fig. 11). Dichlorvos irreversibly inhibits acetylcholinesterase, resulting in continuous nerve stimulation by acetylcholine, paralysis and ultimately death (cholinergic crisis). Its symptoms are characterized by salivation, lacrimation, twitching, vomiting and respiratory distress in humans.

Promethazine, used to treat allergic conditions, nausea and vomiting, motion sickness, and sedation is a H1 receptor antagonist. It blocks acetylcholine at muscarinic receptors, causing side effects like dry mouth etc which would reverse the effect of dichlorvos.

Results of our study found that Promethazine alone or in combination with atropine, a standard treatment for organophosphate poisoning, which blocks acetylcholine at the muscarinic receptors, had a potent antidotal effect.

The toxicological profile of promethazine, focusing on its effects on the liver, kidney, and heart of Wistar rats, was also evaluated. Our findings revealed no evidence of liver or kidney

toxicity; in fact, promethazine exhibited potential *cardioprotective* properties at therapeutic doses. These results reinforced the safety of this widely used drug and opened new discussions about its broader pharmacological benefits (Georgewill & Durojaiye, 2020).

My curiosity about antihistamines did not end there. In 2021, I collaborated with Iwu to explore the repurposing of chlorpheniramine,a H1 antagonist, used for treating allergies, which acts by blocking muscarinic acetylcholine receptors, as a potential *antidote for dichlorvos poisoning* — a serious and often fatal organophosphate toxicity. Remarkably, chlorpheniramine significantly ($P<0.05$) delayed the onset of acute symptoms across the 3 doses of 2mg/kg,4mg/kg, and 8mg/kg that were used, reduced the severity of poisoning, and protected vital organs such as the liver and kidneys. At 8mg/kg, the combination of atropine and chlorpheniramine delayed the onset of toxic symptoms by 2160 ± 34.03secs. Chlopheniramine was observed to significantly reduce the signs of toxicity of dichlorvos which include convulsion, gasping, defecation and tremor. Even more fascinating was that, when combined with atropine, the standard antidote, the two drugs provided synergistic protection far greater than either alone. This finding illuminated chlorpheniramine's potential as a cost-effective and accessible adjunct in managing organophosphate poisoning — particularly relevant in developing countries like ours (Georgewill & Iwu, 2021).

Fig. 11 Agrochemical Highly Effective Systemic Insecticide Ddvp (Dichlorvos) 77.5%Ec https://share.google/6vNOJa8rDoBhhjfh6

II. EPILEPSY

The antiepileptic effects of rosuvastatin, a statin medication that lowers LDL cholesterol and triglycerides, and is used as a part of anti-hypertensive therapy was evaluated. It increases the expression of endothelial nitric oxide synthase, which contributes to its antiepileptic effect. Its anti epileptic effects were evaluated on pentylenetetrazol induced seizures in Wistar rats. Results showed that when rosuvastatin was combined with standard diazepam(which enhances the effect of GABA-gamma amino benzoic acid at the GABA-A receptor) recorded synergistic effects, revealing a significant increase in latency to first minimal clonic seizure (FMCS) and a significant decrease in postictal sleep period (PISP). The combination also prolonged the latency to the first generalised tonic clonic seizure (FGTCS), offering a 100% protection and 0% mortality as opposed to rosuvastatin alone, which offered 40%

protection and 60% mortality after 1 hour. The outcome with diazepam alone offered a 100% protection and 0% mortality after 1 hour of drug administration similar to that obtained with the combination. We therefore concluded that rosuvastatin can be used as an adjunct treatment to diazepam for the management of epilepsy. (Akpan *et al*, 2018)

III. GASTRIC ULCER

A study on repurposing tadalafil, a phosphodiesterase (PDE5) inhibitor originally used for the management of erectile dysfunction, on ethanol-induced and reserpine-induced gastric ulcer in rats and monitored against omeprazole, a standard anti-ulcer agent, revealed that at 50mg/kg, tadalafil exhibited a significant reduction in ulcer index in both models when compared to the standard. Tadalafil acts as a gastroprotective agent by increasing blood flow, reducing inflammation and combating oxidative stress. We therefore concluded that tadalafil had gastroprotective potential at lower concentrations. (Ajibo *et al*, 2022)

CONCLUSION

Mr. Vice Chancellor Sir, I have presented here our modest efforts towards proffering solutions to the disease conditions that plague our people, from substantiating the claims made by our forefathers about the herbs we have been given naturally to reprofiling already existing agents for use in other conditions either singly or in combination, thereby suggesting cheaper, more readily available alternatives to the standard drugs. We continue to play our part towards achieving the Sustainable Development Goal 3.3, which aims to end epidemics of malaria and other diseases by 2030.

In two decades of research, my team and I have gathered data through our research findings, which have been published in

reputable journals locally and internationally. We keep doing our part by putting out reliable data tailored to the needs of our own people that can act as a representative sample in the database for the future to deal with diseases peculiar to us.

OUR FUTURE RESEARCH

Our future research is tailored towards gathering more data on the various disease conditions that affect us.

We are currently working on optimising doses to make suitable recommendations for their use so as to enhance compliance and achieve total parasite clearance in malaria.

We continue with clinical trials for the different combinations and document side effects as we work towards getting our laboratory research to the patient's bedside.

We are also building our database in preparation for the future with artificial intelligence.

Indeed, the future is artificial intelligence (AI) driven drug repurposing. AI moves beyond serendipity by systematically analyzing massive biological datasets. It leverages unprecedented volumes of biological and clinical datasets via systematic pattern recognition, mining through millions of publications and records, while predicting drug-disease associations instantly. (Majumder & Panigrahi, 2025; Kulkarni *et al.,* 2023; Holzinger *et al.,* 2019).

A future we hope we will soon become a part of in our great citadel of learning.

RECOMMENDATIONS

UNIVERSITY

1. FUNDING: This remains a major challenge for indigenous researchers. All stakeholders- the University, Government and private individuals need to collaborate and establish avenues for public-private partnerships to provide easily accessible funds to encourage researchers.

2. SKILL DEVELOPMENT: Indigenous researchers need assistance with periodic skill development and updates in line with current trends in the industry. This can be achieved via access to sponsorships for relevant conferences and workshops.

3. DIGITIZATION OF MEDICAL ASSESSMENT: I recommend a university-wide adoption of digitalized data management for clinical research.

4. MULTIDISCIPLINARY RESEARCH AND ASSESSMENT FOR PROMOTION: Multidisciplinary research is the way to go, but the assessment criteria for promotion make this impossible. We therefore recommend that promotion assessment with respect to multiple authored publications be reviewed.

GOVERNMENT

1. ESTABLISHMENT OF A NATIONAL CENTRE FOR DRUG REPURPOSING:: I propose the setting up of a dedicated hub utilizing Artificial Intelligence and Molecular Docking to screen our existing pharmacopeia for new indications.

2. TRANSLATION OF RESEARCH FROM THE LABORATORY TO THE PATIENT'S BED SIDE: We advise that Policy makers and government should make the channels for testing and clinical trials more accessible and less cumbersome.

GOVERNMENT/UNIVERSITY COLLABORATION

1. MORDERNIZING LABORATORY INFRASTUCTURE: The "New Wineskin" requires High-Throughput Screening (HTS) technology. I urge the University and the Federal Government to invest in these facilities to keep Nigerian scientists at the global frontier.

2. INTELLECTUAL PROPERTY AND "TOWN-GOWN" SYNERGY: We must move beyond "Publish or Perish" to "Patent and Produce." There must be a deliberate bridge between our laboratories and the Nigerian pharmaceutical industry.

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CITATION

Professor Udeme Owunari Georgewill FWIGS, FIPMLD
M.B.B.CH. (CAL), M.Sc. (UPH), Ph.D (UPH)

Professor Udeme Owunari Georgewill (Nee Dominic Uyanga), born on the 23rd of September, 1977, is a scholar of rare distinction whose career is defined by an unwavering commitment to the frontiers of medical science. Her academic journey began in the vibrant scholarly environment of Port Harcourt, eventually leading her to the University of Calabar, where she earned her Bachelor of Medicine and Bachelor of Surgery (M.B.B.Ch.). Driven by a deep-seated curiosity regarding the mechanisms of drug action, she returned to the University of Port Harcourt to obtain both her M.Sc. and Ph.D. in Pharmacology—a foundation that would launch a prolific nineteen-year career in healthcare and higher education.

Rising through the ranks from Lecturer II to the pinnacle of her profession in October,2022. Professor Udeme Georgewill now stands as a Professor of Pharmacology with a specialized focus on Chemotherapy. Her leadership within the University of Port

Harcourt is marked by significant administrative service, including two terms as Acting Head of Department and her current, transformative second term as the Director of the School of Basic Studies. Under her stewardship, the School has witnessed unprecedented growth in student enrollment and the pioneering establishment of its first Computer-Based Testing (CBT) Centre.

A globally recognized researcher, Professor Udeme Georgewill has contributed over 68 scientific papers to reputable journals and shared her insights at nearly 30 international conferences. Her influence extends to national policy through her work with the National Universities Commission (NUC) and her service as a member of several prestigious bodies, including the West African Society for Pharmacology (WASP) and the International Union of Basic and Clinical Pharmacology (IUPHAR).

She is a devoted mentor who has supervised over 40 postgraduate and 50 undergraduate students.

Her influence radiates far beyond the classroom. Whether serving as a Supervisor for Health in the Akuku-Toru LGA or acting as a Public Speaker and Self-Investment Coach, Professor Georgewill is dedicated to the "holistic individual."

Beyond the laboratory, she is a trailblazer in institutional development. As the first Professor to serve as President of the University of Port Harcourt Women Association (UPWA), her historic tenure has championed gender equity and completed major infrastructural projects that will serve generations to come.

A Fellow of the WIN Institute for Gender Studies and a Lady Knight of the Anglican Church, Professor Udeme Georgewill exemplifies the balance of faith, family, and formidable scholarship.

She is happily married to the Vice-Chancellor, Professor Owunari Abraham Georgewill, with whom she shares three daughters.

Professor Udeme Georgewill, remains a beacon of academic integrity, a symbol of purpose-driven leadership, and a testament to the fact that true greatness lies in the ability to inspire others to achieve.

Professor Owunari A. Georgewill
Vice-Chancellor