RIVERS STATE UNIVERSITY
PORT HARCOURT

KILLER CHEMICALS IN EVERYDAY PRODUCTS: STRATEGIES FOR A HEALTHIER LIFE

AN INAUGURAL LECTURE

BY

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DEDICATION

I dedicate this Inaugural Lecture to my parents Ven Dr. Sunday Chinyeremadu Nwachuku and Dame Hon. Silverline Nwachuku (Ada Opotoma Jeze Mba I); and my lovely children Chimaobi and Adaeze.

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The Vice Chancellor and Chairman of this occasion, Sir,
Chairman and Members of the University Governing Council,
The Deputy Vice Chancellor (Academic),
The Deputy Vice Chancellor (Administration),
The Registrar and Secretary to Council and Senate,
The University Librarian,
The University Bursar,
Former Vice Chancellors and Emeritus Professors,
Former Deputy Vice Chancellors,
Heads of the Various Campuses of the University,
Provost of the College of Medicine,
Dean of the Postgraduate School,
Deans of Faculties and Directors of Institutes and Centres,
Heads of Departments and Units,
Distinguished Professors and Members of Senate,
Academic, Administrative and Technical Staff,
Great Students of Rivers State University,
Ministers of God,
Your Royal Majesties, Highnesses and Chiefs,
My Family Members and Friends,
Distinguished Ladies and Gentlemen.

1.0 PREAMBLE

Vice Chancellor Sir, it is with great humility that I stand before you in this hallowed chamber to deliver the 95th Inaugural Lecture of this great University. I am grateful to God Almighty for making this day a reality in my life time. To God alone be all the glory.

I was born in Port Harcourt on the 14th day of July 1963 to Sunday Chinyeremadu Nwachuku and Silverline Ohuruaku Nwachuku (Nee Wodi)

Vice Chancellor Sir, may I introduce this wonderful couple, Ven (Dr) Sunday Chinyeremadu Nwachuku, my Dad and Dame (Hon) Silverline Ohuruaku Nwachuku (Ada Opotoma Jeze Mba I), my Mom. My greatest joy today is that they are both alive and here to witness this high point in my career. Their love and sacrifice is what has brought me this far.

My primary education was at Santa Maria Primary School Aba and secondary school at Federal Government Girls College Owerri, where I graduated with a division one in 1979. I obtained Higher School Certificate in 1981 and then proceeded to join my two elder brothers in London, graduating with a combined honours degree in Biochemistry and Physiology. I returned home for N.Y.S.C. at this point, despite having obtained admission to study Clinical Biochemistry at Leeds University. NYSC was with the Department of Chemical Pathology University of Port Harcourt in 1986. This was my first encounter with the profession of Medical Laboratory Science and I absolutely loved it. After NYSC, I enrolled in the graduate professional diploma programme in Chemical Pathology of the Medical Laboratory Science Council of

Nigeria (MLSCN) and eventually was employed as a Medical Laboratory Scientist. While working at UPTH, I obtained an MSc in Clinical Biochemistry and Immunochemistry from the University of Port Harcourt, and subsequently gained employment at Rivers State University of Science & Technology (RSUST) in 1992, as an Assistant Lecturer. I later obtained a PhD in Chemical Pathology with research interest in Toxicology from Rivers State University in 2010.

Vice Chancellor Sir, ladies and gentlemen my career as one of the pioneer lecturers in the Department of Medical Laboratory Science, for over 30 years now, climaxed in my promotion to the rank of professor in 2022. I have spent a significant part of my years in the Department of Medical Laboratory Science studying the effect of some toxic chemicals contained in some everyday household products which I refer to as 'killer chemicals' on health and the potential of some medicinal plants for amelioration or therapy. The title of this inaugural lecture was therefore aptly chosen as Killer Chemicals in everyday products; Strategies for a Healthier Life.

The desired outcome from this inaugural lecture is to raise awareness about the hidden dangers that confront us from toxic chemicals in some products we use every day, as well as give some insight, largely based on some of my research, on how to we can live healthier lives in spite of these dangers.
Unfortunately, these days in other to make ends meet several people including our unemployed graduates have turned to manufacturers of several products without paying attention to maximum permissible limits of different chemicals. The ones referred to as organic products which are suppose to be safer are sometimes the most toxic this is because they are often just crude extracts with no attempt at purification, not to talk of

testing for presence of contaminants.

Let me at this point appreciate everyone who has found time to be physically present here today, as well as those watching virtually. Thank you very much for your presence and support.

2.0 INTRODUCTION

2.1 EVERYDAY HOUSEHOLD CHEMICAL PRODUCTS

Chemicals are part of our daily lives but often we do not see them because they are hidden in many manufactured products such as cosmetics, cleaning products, personal care products, pesticides, packaged water and drinks (Plate 1)

Plate 1: Some everyday household products

These products when properly used, contribute to improvement of our quality of life and well-being. However, there are chemicals in some of these products that are highly toxic and can

negatively affect our health and cause death if not properly managed.

Accidents, accidental discharges, incorrect use, mixing of products or excessive use of some of these household chemical products may cause immediate health effects such as skin or eye irritations, burns or poisoning. There can also be long-term health effects which might include cancer, organ damage, fertility problems and birth defects, and death.

3.0 TOXIC CHEMICALS HIDING IN EVERYDAY PRODUCTS

3.1 WHAT IS A TOXIC CHEMICAL?

The term 'toxic chemical' simply refers to any chemical which through its chemical action on life processes can cause harm or death to humans, animals or plants.

3.2 CLASSIFICATION OF TOXIC CHEMICALS ACCORDING TO POTENTIAL HEALTH EFFECTS

3.2.1 **Teratogens**: Chemicals that cause birth defects by adversely affecting the cells and tissues of developing foetus. A teratogen passes through the placental barrier to affect the foetus. (Katre, 2016)

3.2.2 **Mutagens:** Chemicals that induce genetic changes in the DNA. They may cause mutation of germ cells resulting in accumulation of abnormal genes or mutation of somatic cells resulting in formation of malignant cells e.g. nitrosoamines, food additives, cosmetics.

3.2.3 **Carcinogens:** Chemicals which may produce cancer either by itself or in conjunction with another substance

e.g. asbestos, formaldehyde, chromium, arsenic, cadmium, nikel, soot particles, PAHs, azo dyes, etc.

3.2.4 Neurotoxicants: Substances that can cause adverse effects on the chemistry, structure or function of the centra nervous system (CNS) e.g. Lead, Mercury etc.

3.2.5 Endocrine Disruptors or Endocrine Disrupting Chemicals (EDCS): Chemicals that interfere with body's endocrine system and produce adverse developmental, reproductive and other effects. They may mimic, block or interfere with the body's hormones e.g. pesticides such as DDT, BPAs, PCBs, metals, plasticizers, pharmaceuticals, detergents, dioxin etc.

3.3 SOME EXAMPLES OF TOXIC CHEMICALS FOUND IN EVERYDAY PRODUCTS

3.3.1 Per-and Polyfluoroalkyl Substances (PFAS): These are chemicals that contain bonds between carbon and multiple fluorine atoms. These bonds are responsible for making the products containing them oil, stain and water repellent, or non-stick. However, these same bonds make PFAs extremely resistant to breakdown and so they tend to persist and accumulate in vivo. PFAs are used in;

a. Cosmetics
b. Food packaging
c. Carpet cleaners
d. Adhesives
e. Sealants

PFAs have been linked to liver damage, obesity, diabetes, cancer, thyroid disease, asthma, reduced fertility, low birth weight and immune system dysfunction by induction of oxidative stress and other mechanisms.

3.3.2 Anti-microbials: Antimicrobials are used in products to

prevent microbial growth. Triclosan and triclocarbon are commonly used antimicrobial agents found in;

a. Hand soaps and detergents
b. lotions and shampoo
c. Deodorant
d. Toothpaste
e. Cosmetics

Widespread use of these products with little regulation has led to concerns regarding their effects on humans. These effects include endocrine disruption leading to reproductive changes due to bioaccumulation. Triclosan has been banned in many parts of the world but implementation of the ban has remained a problem. (Milanovic *et al*, 2023; Weatherly *et al*, 2017).

3.3.3 Bisphenols & Phthalates: These are chemicals used in making plastics stronger (bisphenols) or more flexible (phthalates) they are often called plasticizers and are commonly used in;

a. Canned food liners
b. Plastic containers
c. Flexible PVC pipes
d. Toys (especially teething toys)
e. Adhesives
f. Food packaging
g. Personal care products and cosmetics

These chemicals leach from products into food or water. Humans are exposed through eating or drinking water/drinks stored in containers made with them. Bisphenols and phthalates are harmful to humans and ecosystems because they are endocrine disruptors that cause disruption to reproductive, metabolic, neurologic and immune systems. They are most harmful during critical periods of development of the foetus.

They have also been linked to cancer. Phthalates have been banned from use in cosmetics in the European union, but still remain in use in the rest of the world. (Wang and Qian, 2021).

3.3.4 Solvents: Solvents are used to dissolve other chemical constituents. Examples of solvents include perchloroethylene, trichloroethylene and acetone. Products containing solvents include;

a. Nail polish remover and other cosmetics
b. Household cleaners
c. Aerosols
d. Shoe polish

Solvents evaporate from products and become mixed with the air we breathe. They are often persistent and harmful to humans and ecosystems. Some solvents have been associated with neurotoxicity, reproductive toxicity and cancer. Studies also indicate that long term use of acetone-based removers can cause respiratory problems such as asthma and bronchitis because of its highly volatile nature. (Davis, 2023).

3.3.5 Heavy metals: Heavy metals are naturally occurring elements that have high atomic weight and density (Luo *et al*, 2020).

Heavy metals include metals which play an important role in normal bodily functions e.g. iron, however when they accumulate, they cause negative effects. Some other metals in this category do not have any normal functions in the body. (Katre, 2016).

Heavy metals like lead, arsenic, mercury, aluminium, zinc and chromium are found in a variety of personal care products including lipstick, whitening toothpaste, eyeliner, nail colour, skin lightening creams; and pesticides. Some of these metals are intentionally added as ingredients, while others are

contaminants from the soil or organic plants. Exposure to heavy metals has been linked to health concerns such as reproductive, immune, nervous system toxicity and cancer (Gors, 2023). In fact, arsenic, chromium, cadmium and nickel have been classified as group one human carcinogens by the International Agency for Cancer (IARC, 2012) the main mechanism of toxicity of heavy metals is generation of free radicals to cause oxidative stress.

3.3.6 Hydroquinone: Hydroquinone is most commonly used in skin lightening creams, which are used by some dark-skinned women for skin lightening. Hydroquinone inhibits melanin production (melanin is responsible for skin colour). Those with more melanin naturally have darker skin compared to those with less. High melanin levels in dark skinned individuals makes them less prone to skin cancer resulting from ultra violet rays from the sun. Acts 17:26 tells us that God created all races on earth and made them live throughout the whole earth, he himself fixed before hand the limits of the places where they will live. He made some of his creation have darker skin than others in other to equip them to successfully inhabit the environment. To most people like us inhabiting the environment with extremely high temperatures he gave dark skin and to those inhabiting environment with cooler temperatures he gave lighter skin.
Low hydroquinone concentrations are recommended medically by dermatologist for the treatment of small dark patches (melasma) and hyperpigmentation. Unfortunately, however, companies are producing creams containing hydroquinone in exceedingly high concentrations, especially for the African market. This is not usually followed with information about after care such as the fact that individuals should not go under the sun without protection after use. Unprotected exposure to the sun causes a condition known as ochronosis (Plate 2). This is a condition that causes the skin to darkened where the cream was

applied instead of becoming lighter (see plate). Hydroquinone is also linked to skin cancer and organ system toxicity and has been banned in some countries in a bid to reduce the risk of skin cancer. (Owolabi *et al*, 2020).

Plate 2: Patient suffering from Ochronosis

3.3.7 Formaldehyde and Formaldehyde Releasing Preservatives (FRPs): These chemicals are used in many personal care products, particularly in shampoos and liquid baby soaps, nail polish, nail glue,
eye lash glue. They can be absorbed through the skin and have been identified as carcinogenic by IARC and have been linked to nasopharyngeal cancer and Leukaemia. (IARC, 2012; Lefebvre *et a*l, 2012).

3.3.8 Asbestos in Talc: Asbestos is the name given to a group of six different fibrous minerals (amosite, chrysolite, crocidolite, tremolite, actinolite and anthophyllite) that occur naturally in the environment and are resistant to heat and corrosion. Because of this property asbestos has been used in commercial products such as insulation and fire proofing

materials.

Talc or talcum is a clay mineral composed of hydrated magnesium silicate. Talc in powdered form is often used as baby powder, as a powder it absorbs moisture well, making it useful for keeping skin dry and helping to prevent rashes. Talc is mined from the earth. Talc and asbestos often occur together in the earth, so it is possible for mined talc to be contaminated with asbestos. Asbestos has been scientifically proven over the years to cause ovarian and other cancers like mesothelioma, an aggressive and deadly cancer (Stoiber *et al*, 2020).

Currently, there are a group of women suffering from ovarian cancer suing Johnson and Johnson who claim that their condition was caused by their regular use of J & J talc baby powder as antiperspirant over the years. This is based on the claim that J & J talc was sometimes contaminated with carcinogenic asbestos. Some of these women have won their cases partly based on toxicological evidence of asbestos in their bloodstream. (A Reuters investigation, 2020).

3.3.9 Organophosphate Pesticides: Pesticides are chemicals used to kill or control pests, such as insects and rodents. Most pesticide exposure occurs indoors. Indoor exposures are more devastating on our health than ambient exposures given that people spend more time indoors, often in poorly ventilated areas.

There are over 20,000 different household pesticide products worldwide containing over 300,000 active chemical ingredients. (Ojo, 2016)

About 99% of deaths associated with pesticides occur in developing countries like Nigeria. This is largely due to poor pesticide education resulting in misuse, use of cheaper but more

deadly forms of pesticides (in terms of persistence and toxicity), poor legislation and lack of enforcement of available legislation (e.g. some household pesticides are listed on the list of banned pesticides by NAFDAC but implementation of the ban has not been effective), lack of adequate information, knowledge and awareness of the dangers of pesticides (Ojo, 2016).

The form of pesticides commonly used in homes are insecticides such as organophosphates, pyretinoids, and carbamates. Insecticides are applied in various formulations and delivery systems e.g. sprays, baits, slow-release diffusion, powders etc.
These insecticides have different mechanisms of action. The organophosphates for instance, which are esters of phosphoric acid, cause acetylcholinesterase (AchE) inhibition and accumulation of acetylcholine at neuromuscular junctions, resulting in rapid twitching of voluntary muscles and eventually paralysis and death. Organophosphate insecticides are broad-range insecticides, generally regarded as the most toxic of all pesticides to vertebrates, including human. (Doull *et al*, 2001). Examples of organophosphates used in insecticide include malathion, parathion, diazinon, fenthion, dichlorvos, ethion and chloropyrifos.

Organophosphate poisoning has become a major problem in Nigeria as a result of indiscriminate use of products containing them in many households. This is worsened by sale of organophosphate insecticides on the street and open market. One organophosphate insecticide that has caused a lot of problems in our society in recent times is dichlorvos, sold as a liquid formulation, 2,2-dichlorovinyl dimethyl phosphate, otherwise known as sniper (Plate 4). This is intended for use as an insecticide, but these days it has been implicated in cases of suicide, accidental death and even homicide.

Plate 3: 2,2-dichlorovinyl dimethyl phosphate (sniper)

It is also a common contaminant of beans bought from the market where it is used as a preservative. Acute exposure to high concentrations of dichlorvos results in neurotoxic effects, convulsions, paralysis, coma and death. (Okoroiwu and Iwara, 2018). Chronic exposure to low dose concentrations also results in serious health effects (Ozoemena *et al* 2021).

3.3.10 Microplastics: When you drink from a plastic water bottle, there is the risk of exposure to tiny potentially harmful plastic particles known as microplastics. The main source of exposure to these microplastics could be the screw cap rather than the bottle itself. This was the conclusion of a peer reviewed study published in the journal of Water and Health which found that repeated screwing on and off of a plastic bottle cap creates friction that generates a significant amount of microplastics. These microplastics then end up in the water we drink. In the study they found that each twist could produce about 500 microplastic particles (Singh, 2021).

Microplastics in bottled water can also come from the

manufacturing process. During production plastic water bottles are subjected to high pressure and temperature changes which can cause the plastic to degrade leading to leaching out of microplastics. In a study of more than II brands of water and 259 bottles, 93% of the bottles contained microplastics. The researchers also found microplastics in seven out of nine bottled mineral drinks tested. (Mason *et al*, 2018).

Contaminants in sachet water or bottled water can also be caused by leaching after exposure to sunlight. (Obisike & Nwachuku, 2016)

Beyond water/mineral bottles, microplastics are also found in many toys, food containers etc. Even when these items have been discarded, the plastics are broken into tiny pieces over time, creating microplastics. These microscopic pieces of plastic then spread into our water and soil. The extensive use of plastics all over the world and its long-term persistence, has resulted in them being found in our food and water.

3.3.11 Dyes or Colourants: Several dyes are available in the open market for use as colouring agents to food and drinks. Some commonly used ones are brilliant blue, indigo carmine, tartrazine, alura red and sunset yellow. Studies have shown that synthetic food/drink colourants have considerable toxicological effects, including but not limited to carcinogenicity and hypersensitivity reactions (Okafor *et al,* 2016).

4.0 ROUTES OF EXPOSURE

4.1 Dermal Absorption: Contact with the skin is the most common route of toxic chemical exposure. Factors affecting dermal absorption of chemicals include;

a. Condition of the skin e.g. physical damage
b. Chemical makeup of product: Inorganic chemicals are not easily absorbed through the skin (e.g. cadmium, lead, mercury, chromium). Organic chemicals dissolved in water also do not easily penetrate the skin. Skin is lipophilic because of its lipid bilayer membrane: Organic solvents such as paint thinner, petrol, kerosene are therefore easily absorbed through the epidermis
c. Concentration of toxic chemical or exposure time: High concentration or long exposure time can increase the rate or amount of chemical absorbed.

4.2 **Inhalation:** This is the easiest and fastest means of exposure to toxic chemicals because toxic chemicals in the gaseous state are readily absorbed in the respiratory tract. The following factors affect inhalation of toxic substances through this route.

a. Concentration of toxic substance in the air
b. Solubility of substances in blood and tissue
c. Respiratory rate
d. Duration of exposure
e. Condition of respiratory tract
f. Size of toxic particle

4.3 **Ingestion:** Ingestion of toxic chemicals could occur accidentally in children or through ingestion of contaminated of food. Once a chemical enters the body, its effects depend on its concentration in the target organs, its chemical and physical form, what happens to it after it is absorbed and its duration of stay in the tissue or organ of choice. After it is taken up in the blood, a chemical is quickly distributed throughout the body, it may be moved from one organ or tissue to another

(translocation), or changed into a new compound (biotransformation)

4.4 Other Routes of Exposure

The eye is another common point of contact for toxic chemicals. The primary point of contact for toxic chemicals in the eye is the cornea. (Dingsheng & Sangwan, 2019)

5.0 TYPES OF EXPOSURE

5.1 **Acute**: Exposure to a chemical for 24hrs or less, often a large brief exposure. Acute health effects are observed immediately or soon after the exposure. The symptoms often subside after the cause is removed for a low dose exposure, however, permanent damage or even death can occur from a single high dose exposure. Examples of acute health effects include dizziness, skin irritation, throat irritation, paralysis, coma and death.

5.2 **Sub-acute**: Exposure to a chemical for 1 month or less.

5.3 **Sub-chronic**: Exposure to a chemical between 1 to 3 months.

5.4 **Chronic**: Exposure to a chemical for more than 3 months. Symptoms do not usually subside even when exposure stops. Examples of chronic health effects include cancer and asthma.

5.5 Acute and Chronic

Most chemicals cause both acute and chronic effects depending on the dose and duration of intake. Ethanol is

an excellent example of a compound that causes varied health effects. A small amount of ethanol from alcoholic drinks over several hours can result in loss of coordination and impaired judgement, a large one-time consumption can lead to vomiting, unconsciousness or even death; consumption over several days by a pregnant person can lead to birth defects; and chronic consumption over many years can lead to cirrhosis of the liver.

5.6 Toxicokinetics

The occurrence of health effects is dependent on the concentration of the chemical in the target organ. The concentration in the organ is dependent on the absorption, distribution, metabolism (biotransformation) and excretion (ADME). Biotransformation occurs when a chemical is changed from one form to another, which may also change the toxic properties of the substance. It usually occurs in several steps, primarily in the liver, but it may also occur in other tissues like the kidneys, lungs and digestive tract.

The rate at which a toxic chemical is excreted from the body determines whether it will have a toxic effect. The longer a chemical is in the body, the greater the likelihood of toxicity. The main way a chemical is excreted from the human body is through the urine, but the lungs and liver are also important in removing certain chemicals.

5.7 Bioaccumulation

This is defined as increase in the concentration of a chemical in a biological organism over time, compared

to the chemical's concentration in the environment. Chemicals accumulate in living things any time they are taken up and stored faster than they are metabolized and excreted. Bioaccumulation can result in chemicals present in low concentration in household products causing chronic health effects after a long duration of use.

5.8 Factors that affect Health risk

These include; amount exposed to, type of chemical, duration of Exposure, age, general state of health and type of exposure. Children, Pregnant women and the Elderly are more at risk. (Government of Canada, 2023).

6.0 STRATEGIES FOR A HEALTHIER LIFE

6.1 TOXICOVIGILANCE: The WHO defines toxicovigilance as the active process of identifying and evaluating the toxic risks existing in a community, and evaluating the measures taken to reduce or eliminate them. We can identify toxic products in the home by always reading product labels and following directions, using only amounts indicated. Product labels may look like the images shown below (Plate 2). These images however often apply to pesticides as many other chemicals of concern enter our homes unlabelled.

Plate 4: Some signage indicating toxicity

6.2 Safe Handling with attention to labels and directions
All of these products should be handled with care and attention given to the directions on the label for safe use. These warnings however often apply to short term (acute) toxicity effects, not to long-term (chronic) toxicity. Note that absence of a warning label does not mean a product is safe. Some chemicals present in products in minute non-toxic amounts may over a period of time (months or years) result in chronic toxicity effects or even lethal effects. This may be caused by bioaccumulation or interaction with other chemicals in vivo. These are the more dangerous chemicals as the individual is often unaware of the effects until after a long period of use. (Karr *et al*, 2007)

6.3 MODERATION IN USE. Since in reality we cannot completely avoid the use of products containing toxic chemicals, the best way to prevent the hazards arising from toxic chemicals is to reduce their use i.e. if toxic chemical products must be used, they must be used in moderation. Also, since a person may use several products with toxic contents together at a given period, this amounts to a greater risk of combined exposure due to interaction in vivo.

6.4 AVOID DELIBERATE OR ACCIDENTAL MIXING OF CHEMICAL PRODUCTS: Never mix chemical products. Mixing toxic products can start a chemical reaction that could create highly toxic fume (e.g. mixing of cleaning liquids) or explosions (e.g. fuel and kerosene when ignited in kerosene stoves), serious burns and even death.

6.5 USE PERSONAL PROTECTIVE EQUIPMENT (PPE): Gloves, goggles and use of overalls or long-sleeved shirts can prevent direct contact with the skin especially when using detergents, cleaning agents, bleach, pesticides.

6.6 REDUCE YOUR NEED FOR PESTICIDES. Pesticides are poisons. Besides killing bugs and insects, they also poison children accidentally, irritate eyes or skin, cause chronic disease and eventually death, and are also handy tools for those inclined to commit suicide.

6.7 ENSURE PROPER VENTILATION. If we must use household products such as pesticides, it should be in well ventilated areas, (e.g. open windows) to avoid breathing in fumes. In fact, the opposite is often the case in our homes. We shut all our windows when using pesticides before we go to bed at night, to concentrate the fumes. At the end of the exercise, we kill the mosquitoes or cockroaches but unknown to us, we are also gradually killing ourselves.

6.8 AVOID AEROSOL SPRAY PRODUCTS AS MUCH AS POSSIBLE: The small size of aerosol particles makes it easy for them to be inhaled. When used, containers must be tightly closed to prevent evaporation. (Maleti and Adla, 2011).

6.9 STORE TOXIC PRODUCTS PROPERLY: Only store household chemical products in places children cannot get to. Keep products containing toxic materials in their original container and never remove the labels. Never store toxic products in food containers. If you

must use disposable plastic water bottles, try to keep them in a cool place to avoid leaching of chemicals. Also do not keep plastic water bottles or sachet packaged water stored for too long to minimize leaching.

6.10 CHOOSE ALTERNATIVES TO PLASTICS: Instead of bottled water, we could drink filtered tap water whenever possible. When you need water on the go, use reusable stainless-steel bottle. It is better for your health and the environment. Also never microwave plastics.

6.11 CONSUMPTION OF HERBS, FRUITS AND SEEDS OF INDIGENOUS PLANTS AS REMEDIATION AGAINST NEGATIVE HEALTH EFFECTS: In spite of all the above, exposure to toxic chemicals contained in everyday products cannot be completely avoided. The question therefore is, can we find a way to ameliorate or cure the acute or chronic health effects from these chemicals? My studies, in collaboration with colleagues and students in the Department of Medical Laboratory Science over the years, using animal models, have shown that there is potential for amelioration and therapy, in the management of the toxic health effects of various chemicals by some indigenous medicinal plants.

7.0 MY CONTRIBUTIONS TO KNOWLEDGE

7.1 Studies on Dichlorvos toxicity

1. Evaluation of 2,2-dichlorovinyl dimethyl phosphate

(Sniper) induced hepatotoxicity and oxidative stress in New Zealand White rabbits.

2. Assessment of chronic toxicological effect of 2,2-dichlorovinyl dimethyl phosphate (sniper) on the kidneys of New Zealand white rabbits.

Ozoemena C.C, Igwe F.U**, Nwachuku E. O**, Bartimaeus E.S, 2021

Scientific studies involving acute exposure of rats, mice and rabbits have demonstrated dichlorvos to have high to extreme acute toxicity from oral or dermal exposure and inhalation. However, while the acute toxicity effects of dichlorvos is well known, its chronic toxicity effects are often overlooked.

In the studies above, the chronic effects of 2,2-dichlorovinyl dimethyl phosphate (Sniper) on hepatotoxicity, oxidative stress markers and the kidneys in New Zealand white rabbits were evaluated. Results showed significant elevations in the levels of kidney injury molecule – 1 (KIM-1) from day 30 with marked elevation as the duration of oral dichlorvos treatment increased to day 90 (figure 1). KIM-1 is a sensitive blood marker that specifically reflects acute and chronic kidney injury. It is used for early detection of kidney dysfunction even when other indices of renal function may not be elevated. Dichlorvos also caused significant elevation in mean values of urea and creatinine from the day 30 to 90 (figures 2 & 3). Elevations in urea levels may be influenced by other factors but kidney damage is the only factor that significantly increases serum creatinine levels. As shown in figure 4 & 5, oral dichlorvos treatment of the rabbit also significantly increased the activity of liver enzymes Aspartate Transaminase (AST) and Alanine Transaminase (ALT) from day 30 to 90. AST and ALT are markers of hepatocyte inflammation or injury, the serum levels of these enzymes therefore reflect the physiological state of the

liver. In figures 6 & 7 there were conjugated bilirubin with oral dichlorvos treatment which increased with the duration of dichlorvos exposure. This hyperbilirubinemia is suggestive of an increase in red cell breakdown. This finding along with elevated liver enzymes is indicative of hepatocellular damage. The parameters used in the assessment of oxidative stress were malondialdehyde, lipid peroxidase index and Total Antioxidant Capacity (TAC). Oral dichlorvos treatment caused significant elevations in malondialdehyde and lipid peroxidase index (figures 8 & 9), but significant reduction in TAC (figure 10). These results suggest that sniper caused damage to the tissues by inducing oxidative stress which leads to an increase in reactive oxygen species in the body, resulting from increased lipid peroxidation, thereby depleting the antioxidant enzymes. The findings from this study clearly reveal that oral dichlorvos (sniper) exposure has serious toxic effects on both renal and hepatic cells.

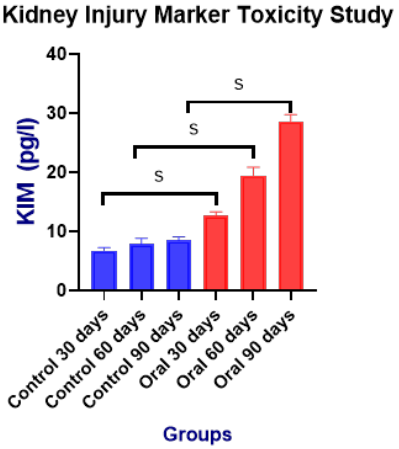

Fig. 1: (Mean ±SD) Kidney Injury Marker of Rabbits Treated with Dichlorvos

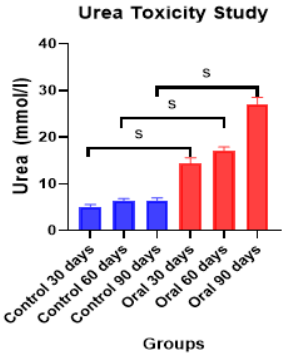

Fig. 2: (Mean ±SD) Urea of Rabbits Treated with Dichlorvos

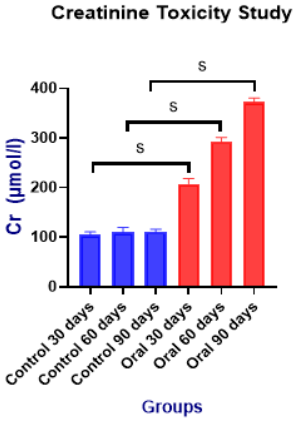

Fig. 3: (Mean ±SD) Creatinine of Rabbits Treated with Dichlorvos

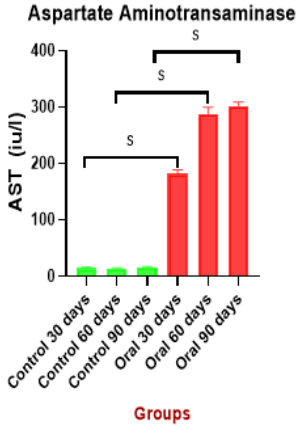

Fig. 4: (Mean ±SD) Aspartate Transaminase (AST) of Rabbits Treated with Dichlorvos

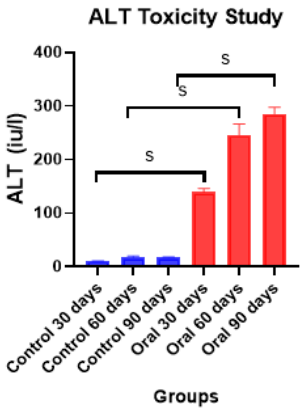

Fig.5: (Mean ±SD) Alanine Transaminase (ALT) of Rabbits Treated with Dichlorvos

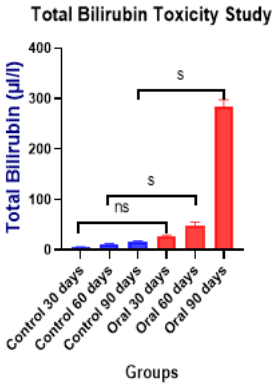

Fig. 6: (Mean ±SD) Total Bilirubin of Rabbits Treated with Dichlorvos

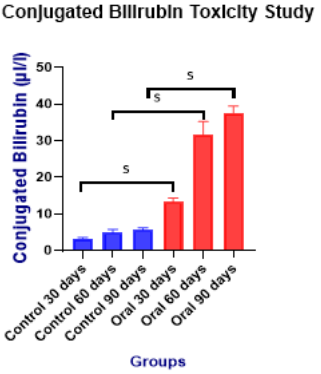

Fig.7: (Mean ±SD) Conjugated Bilirubin of Rabbits Treated with Dichlorvos

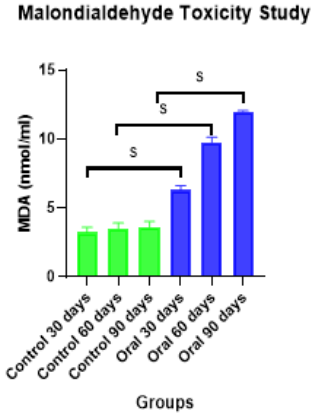

Fig. 8: (Mean ±SD) Malondialdehyde of Rabbits Treated with Dichlorvos

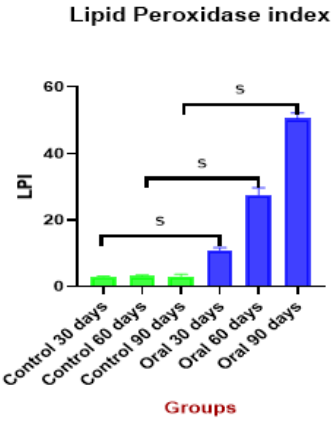

Fig.9: (Mean ±SD) Lipid Peroxidase Index of Rabbits Treated with Dichlorvos

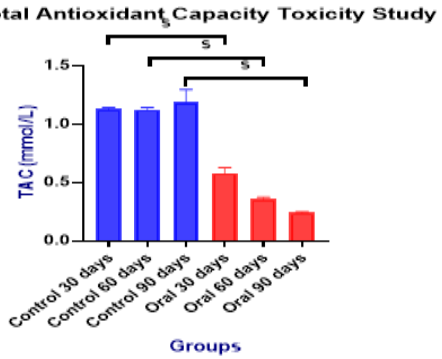

Fig. 10: (Mean ±SD) TAC of Rabbits Treated with Dichlorvos

7.2 Studies on the presence of heavy metals in some marketed herbal oils.

1. Evaluation of the effect of some marketed herbal cosmetics in Port Harcourt on renal parameters of rabbits.
2. Assessment of carcinogenic and non-carcinogenic health risk of some marketed herbal oils in Port Harcourt.

Thompson I.N, Bartimaeus E.S, **Nwachuku E.O**, Brown H, Agoro E.S, (2021)

In this study the effect of heavy metals in 3 marketed herbal oils sold in Port Harcourt on renal parameters, carcinogenic and non-carcinogenic health risk of rabbits was assessed. Results indicated that concentration of lead, cadmium and arsenic exceeded the maximum allowable limits by WHO and USEPA (Table 1). The incremental lifetime cancer risk was above the allowable limit of normal for all three cosmetics. Results also

showed evidence of renal impairment after chronic exposure.

Table 1: Concentration of heavy metals in 3 herbal oil samples

S/N	Herbal Oil samples	Cadmium (mg/kg)	Lead (mg/kg)	Arsenic (mg/kg)	Copper (mg/kg)	Zinc (mg/kg)
1	A	2.370	18.060	45.660	1.700	5.910
2	B	1.580	11.38	21.090	1.080	5.760
3	C	2.260	13.390	30.088	1.190	5.110
USEPA Limit		0.5	0.5	2.5	0.5	18
WHO Limit		0.3	10	10	1	20

Key: Sample A = All things natural Herbal Oil, Sample B = Kakiva Herbal Oil, Sample C = Amal Botanical Herbal Oil, USEPA = US Environmental Protection Agency, WHO = World Health Organization

7.3 Studies on Contaminants in sachet water after exposure to sunlight

Effect of sunlight on some physiochemical constituent of sachet package water sold in Port Harcourt Rivers State, Nigeria.

Obisike U. & **Nwachuku E.O.** (2016)

In recent times, there has been an immense increase in the production and consumption of sachet packaged water (pure water) in Port Harcourt city and environs. Often these sachet packaged water are exposed to sunlight in different shops where they are stored for sale.

This study therefore was done to determine the effect of sunlight on some physical and chemical constituents of sachet packaged water.

Table 2 shows mean, standard deviation, correlation and level of significance for turbidity values comparing before and after exposure to sunlight. The table shows that mean values for turbidity before and after respectively 1.62 ± 0.63NTU and 2.37 ± 0.64NTU. Comparing mean value for both group using a paired sample t test yielded the following level of significance and correlation p=0.000 and r=0.473 respectively. From the table, it was found that there was a significant increase in turbidity values at p<0.0001. Figure 11 shows a box plot of turbidity levels before (blue box) and after (red box) exposure to sunlight. Table 3 shows mean ± SD, correlation and level of significance for Total Hydrocarbon Content (THC). Mean values for THC before and after exposure to sunlight were compared using paired sample t test. Mean values for THC before exposure were recorded as 1.08 ± 0.63 mg/L, while mean THC after exposure were recorded to be 1.62 ± 1.02mg/L. When both mean values were compared, a significance level of p=0.002 and correlation of r=0.557 were recorded. The results show that there was significant increase in mean THC after exposure to sunlight at p<0.0001. Figure 12 shows box plot of THC for both before (blue) and after (red) exposure to sunlight. The increases observed in turbidity and total hydrocarbons (THC) of sachet packaged water after exposure to sunlight may be due to leaching of substances into the water as the water was heated by sunlight.

Table 2. Mean ± SD, p value, correlation for Turbidity before and after exposure to sunlight.

Turbidity(NTU)	N	Mean ± SD	p value	Correlation	Remark
Before	30	1.62 ± 0.63	0.0001	0.473	**S**
After	30	2.37 ± 0.64			

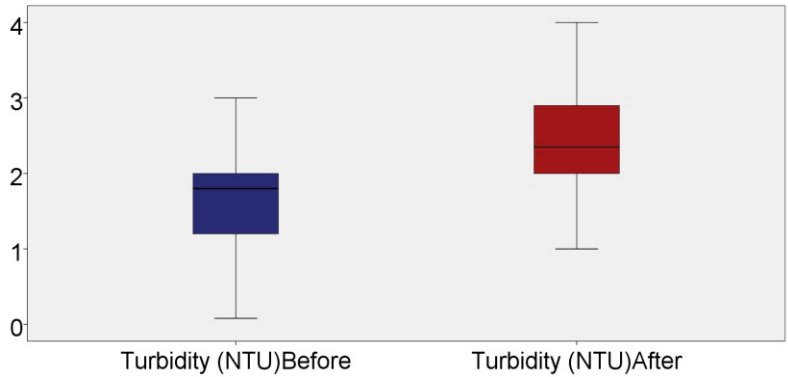

Figure 11: Box plot of Turbidity levels comparing before and after exposure to sunlight.

Table 3: Mean ± SD, p value, correlation for Total Hydrocarbons (THC) before and after exposure to sunlight.

THC (mg/L)	N	Mean ± SD	p value	Correlation	Remark
Before	30	1.08 ± 0.63	0.002	0.557	**S**
After	30	1.62 ± 1.02			

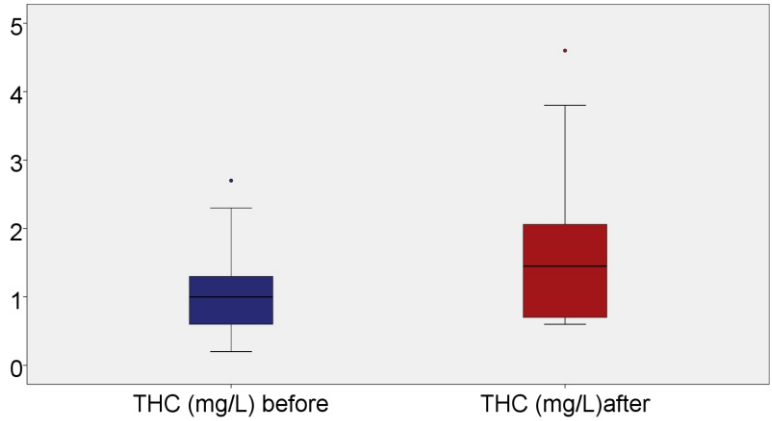

Figure 12: Box plot of Total hydrocarbon content comparing before and after exposure to sunlight.

7.4 Studies on Toxicity of Tartrazine Colourant

Effect of Tartrazine orally administered on Lipid Profile of Albino rats.

Elekima I, **Nwachuku E.O**, and Ben-Chioma A, (2017)

Tartrazine is a water-soluble synthetic lemon-yellow azo dye used as a colourant with an acceptable daily intake (ADI) of 0-7.5mg/kg body weight. It is seen in products such as soft drinks, energy drinks, flavoured corn chips, cereals, cake mixes, pastries, custard powder, sauces, powdered drink mixes, ice cream, chewing gum, yoghurt, noodles, biscuit and so on. Tartrazine has been reported to induce several side effects ranging from allergic reactions in humans to more serious health effects.

In this study effect of tartrazine orally administered on lipid profile of albino rats was investigated. When tartrazine treated rats were analysed, Group 1 had 0.994+ 0.175, 1.745$\pm$0.063, 1.203$\pm$0.065 and 0.0910$\pm$0.067 for Triglyceride (TG), Total Cholesterol (TC), HDL-C and LDL-C. Group 2 had 1.316$\pm$0.078, 2.791$\pm$0.895, 1.616$\pm$0.453 and 0.577$\pm$0.583 for TG, TC, HDL-C and LDL-C respectively. Group 3 had 1.113$\pm$0.371, 1.812$\pm$0.283, 1.076$\pm$0.078 and 0.230$\pm$0.225 for TG, TC, HDL-C and LDL-C respectively. Group 4 had 1.215$\pm$0.117, 1.784$\pm$0.152, 0.979$\pm$0.065 and 0.253$\pm$0.198 for TG, TC, HDL-C and LDL-C respectively while Group 5 had 1.269$\pm$0.191, 1.900$\pm$0.111, 1.092$\pm$0.029 and 0.231+0.149. The comparison of group 1 and 2 revealed that TC and TG was significantly increased ($p<0.05$) while there was a non-significant decrease and increase in HDL-C and LDL-C respectively (Table 4). The comparison of group 1 and group 3

showed a significant decrease in HDL-C levels ($p<0.05$). (Table 5). Comparison of group 1 and group 4 showed a significant increase and decrease in TG and HDL-C respectively ($p<0.05$) (Table 6). Finally, the comparison of group 1 and 5 indicated a significant increase in TG, TC and a significant decrease in HDL-C ($p<0.05$). Non-significant increase was observed in LDL-C level (Table 7)

The above results indicated that tartrazine orally administered at varying concentration induced increased levels of TG, TC, and LDL-C and decreased HDL-C levels which are usually linked to cardiovascular disorders, therefore the use of tartrazine azo dye in drinks/food, especially in excess should be avoided.

Table 4: Comparison of group 1 and 2 for Tartrazine treated rats.

Parameter	TG (mmol/L)	TC (mmol/L)	HDL (mmol/L)	LDL(mmol/L)
GROUP 1 (0.00g/kg)	0.994± 0.175	1.745±0.063	1.203±0.065	0.0910±0.067
GROUP2 (0.07g/kg)	1.316 ±0.078	2.791 ±0.895	1.616 ±0.453	0.577±0.583
p VALUE	0.0012	0.0171	0.0514	0.0695
t VALUE	4.490	2.855	2.212	2.031
REMARKS	S**	S*	NS	NS

Table 5: Comparison of group 1 and 3 of tartrazine treated rats.

Parameter	TG (mmol/L)	TC (mmol/L)	HDL (mmol/L)	LDL (mmol/L)
GROUP1(0.00mg/kg)	0.994± 0.175	1.745±0.063	1.203±0.065	0.0910±0.067
GROUP3(0.11mg/kg)	1.113±0.371	1.812±0.283	1.076±0.078	0.230±0.225
p VALUE	0.4939	0.5838	0.0122	0.1781
t VALUE	0.7101	0.56661	3.053	1.4490
REMARKS	NS	NS	S*	NS

Table 6: Comparison of group 1 and group 4 of tartrazine treated rats.

Parameter	TG (mmol/L)	TC (mmol/L)	HDL (mmol/L)	LDL (mmol/L)
GROUP1(0.00g/kg)	0.994± 0.175	1.745±0.063	1.203±0.065	0.0910±0.067
GROUP4(0.14g/kg)	1.215±0.117	1.784±0.152	0.979±0.065	0.253±0.198
p VALUE	0.0277	0.576	0.0001	0.0874
t VALUE	2.574	0.5770	5.962	1.8950
REMARKS	S*	NS	S***	NS

Table 7: Comparison of group 1 and 5 of tartrazine treated rats

Parameter	TG (mmol/L)	TC (mmol/L)	HDL (mmol/L)	LDL (mmol/L)
Group 1 (0.00g/Kg)	0.994± 0.175	1.745±0.063	1.356 ±0.068	0.0910±0.067
Group 5 (0.18g/Kg)	1.269±0.191	1.900±0.111	1.092±0.029	0.231±0.149
p value	0.0265	0.014	0.0035	0.0613
tvalue	2.599	2.961	3.793	1.001
Remarks	S*	S*	S**	NS

*significant, **Moderate significant, ***Highly significant, NS=Not Significant; S=Significant

7.5 Studies on Amelioration and Therapeutic potentials of some indigenous plants against toxic chemical effects.

7.5.1. Studies on *Elaies oleifera* (Palm oil) fruit extracts

Evaluation of Protective properties of *Elaeis oleifera* fruit extracts on renal parameters of dichlorvos induced nephrotoxicity in Albino rats.

Nwachuku E.O., Beega F., Ben-Chioma A.E., Brisibie N. and Elekima I., (2020)

The study evaluated the protective properties of *Elaeis oleifera* (Palm Oil) fruit extract on renal parameters of dichlorvos-induced nephrotoxicity in albino rats at acute, sub-chronic, and chronic phases. Results obtained indicated that palm oil had a protective effect on the kidney by reducing values of creatinine, urea and potassium in palm oil treated rats.

In the acute study, there was no significant difference in Sodium (Na^+) and potassium (K^+) levels when comparing Negative control group (Group 1) to positive control group (Group 2) but there was a significant difference in chloride (Cl^-), Urea and Creatinine levels. When comparing Negative control group (Group 1) to treatment group (Group 3), there was no significant difference in Sodium (Na^+), Potassium (K^+), chloride (Cl^-), urea

and creatinine values. Comparing positive control group (Group 2) to treatment group (Group 3), there was no significant difference in sodium (Na^+), potassium (K^+), urea and creatinine levels (Table 8).

Table 8: Mean ± SD of Renal Parameters in Acute (24 hours) Study at high dose (30mg/kg) Dichlorvos administered orally

	Na^+ (mmol/L)	K^+ (mmol/L)	Cl^- (mmol/L)	Urea (mmol/L)	Creatinine (µmol/L)
Group 1 (Negative control) (n=5)	154.20±2.77	5.76±1.03	103.00±1.87	4.20±0.94	53.06±10.85
Group 2 (Positive control) (n=5)	159.80±4.71	6.50±0.37	106.60±1.52	6.88±0.43	75.04±5.34
Group 3 (Palm Oil Treatment) (n=5)	150.20±4.55	4.76±0.15	104.20±0.84	3.57±1.03	58.18±8.49
p-value	0.010	0.04	0.007	<0.001	0.004
F-value	6.893	9.368	7.754	4.531	9.087
$Postho_c$					
Group 1 vs Group 2	NS	NS	S	S	S
Group 1 vs Group 3	NS	NS	NS	NS	NS
Group 2 vs Group 3	S	S	NS	S	S

KEY:
NS= Non Significant
S= Significant

In the two (2) weeks sub-chronic study, there was no significant difference in Sodium (Na^+), potassium (K^+), chloride (Cl^-) and Urea levels when comparing Negative control group (Group 4) to positive control group (Group 5) but there was a significant difference in Creatinine level. When comparing Negative control group (Group 4) to treatment group (Group 6), there was no significant difference in Sodium (Na^+), chloride (Cl^-) and urea values but a significant difference in potassium (K^+) and creatinine values. Comparing positive control group (Group 5)

to treatment group (Group 6), there was no significant difference in sodium (Na^+), chloride (Cl^-) and creatinine values but significant difference in potassium (K^+) and urea values (Table 9).

Table 9: Mean ± SD of Renal Parameters in Sub-Chronic (2 weeks) study at low dose of 10mg/kg Dichlorvos orally treated rats

	Na^+ (mmol/L)	K^+ (mmol/L)	Cl^- (mmol/L)	Urea (mmol/L)	Creatinine (µmol/L)
Group 4 (Negative control) (n=5)	154.20±2.77	5.76±1.03	103.00±1.87	4.20±0.94	53.06±10.85
Group 5 (Positive control) (n=5)	152.40±3.13	5.52±0.89	105.00±3.74	5.55±0.44	82.86±6.11
Group 6 (Palm Oil Treatment) (n=5)	154.40±5.32	3.56±0.58	105.60±1.82	3.33±1.88	70.58±4.56
p-value	0.681	0.003	0.299	0.045	<0.001
F-value	0.397	10.008	1.337	4.067	14.144
Posthoc					
Group 4 vs Group 5	NS	NS	NS	NS	S
Group 4 vs Group 6	NS	S	NS	NS	S
Group 5 vs Group 6	NS	S	NS	S	NS

KEY:
NS= Non Significant
S= Significant

In the one (1) month chronic study, there was no significant difference in Sodium (Na^+) and chloride (Cl^-) values when comparing Negative control group (Group 7) to positive control group (Group 8) but there was a significant difference in potassium (K^+), urea and Creatinine values. When comparing Negative control group (Group 7) to treatment group (Group 9), there was no significant difference in Sodium (Na^+), chloride

(Cl^-) and creatinine values but a significant difference in potassium (K^+) and urea values. Comparing positive control group (Group 8) to treatment group (Group 9), there was no significant difference in sodium (Na^+), potassium (K^+), chloride (Cl^-) and creatinine values but a significant difference in urea value (Table 10).

The results obtained in this study indicated that palm oil has a protective effect in ameliorating the nephrotoxicity induced by dichlorvos in the 24 hours study, 2 weeks and 1 month study as it ameliorated the glomerulonephritis that was induced by the dichlorvos only treated group. The significance of this study is that palm oil can be given as an immediate intervention in cases of acute toxicity poison with dichlorvos before medical attention can be assessed.

Table 10: Mean ± SD of Renal Parameters in 1 month (Chronic) study at low dose of 10mg/kg Dichlorvos administered orally

	Na^+ (mmol/L)	K^+ (mmol/L)	Cl^- (mmol/L)	Urea (mmol/L)	Creatinine (µmol/L)
Group 7 (Negative control) (n=5)	153.00±1.87	5.64±1.13	105.20±5.26	3.90±0.44	56.86±13.16
Group 8 (Positive control) (n=5)	151.80±6.53	2.94±0.50	106.20±5.85	5.50±0.41	76.60±3.47
Group 9 (Palm Oil Treatment) (n=5)	148.60±6.23	3.50±1.34	102.80±8.58	3.05±0.48	67.40±0.73
p-value	0.427	0.004	0.720	<0.001	0.007
F-value	0.913	9.158	0.338	39.503	7.878
Posthoc					
Group 7 vs Group 8	NS	S	NS	S	S
Group 7 vs Group 9	NS	S	NS	S	NS
Group 8 vs Group 9	NS	NS	NS	S	NS

KEY:
NS= Non Significant
S= Significant

7.5.2. Studies on *Jathropha* species (Hospital too far)

Evaluation of the effect of *Jathropha interrigima* (euphorbiaceae) on the renal function of male Albino Wister rats exposed to chromium.

Andy-Nwokocha M.J., **Nwachuku E.O.,** Bartimaeus E.S., and Obunwo C.C., 2021

Jathropha plants are readily available in the South-South region of Nigeria where they are commonly referred to as 'hospital too far' (Plate 5 & 6) studies carried out on the phytochemical components of jathropha species have revealed that they are rich in antioxidant components which make them useful in the treatment of oxidative stress (Oyewole and Akingbala, 2011). This study therefore evaluated the effect of *Jathropha interrigima* on renal function of male albino Wistar rats exposed to chromium. Results indicated that exposure to chromium caused renal injury but *Jathropha interrigima* reversed renal injury when given as a therapy for up to 30 to 60 days. (Tables 11 to 16). The significance of this is that *Jathropha interrigima* has the potential to heal or reverse renal injury caused by chromium if given as a therapy over a long duration of time

Plate 5: *Jathropha interrigima* leaf

Plate 6: *Jathropha Tanjorensis*

Table 11: Determination of effect of Jathropha interrigima (PRE and POST) on electrolytes of male albino wistar rats exposed to chromium in acute phase

Phases	Groups	Parameters Mean ± SD									
		Pre					Post				
		Na^+ (mmol/l)	K^+ (mmol/l)	Cl^- (mmol/l)	-HCO3 (mmol/l)	AG (mmol/l)	Na^+ (mmol/l)	K^+ (mmol/l)	Cl (mmol/l)	-HCO3 (mmol/l)	AG (mmol/l)
15 Days	NC	126.5±2.12	7.9±0.84	91.5±2.12	13.5±2.1	22.5±2.12	126.5±2.12	7.9±0.85	91.5±2.12	13.5±2.12	22.5±2.12
	PC	132.5±0.70	5.5±0.42	95.5±2.1	11.5±0.7	26.0±2.82	130.0±0.00	5.7±0.00[1]	97.0±0.00	15.0±0.00	19.0±0.00
	TG	130.6±4.04	7.33±0.5	98.0±2.6	13.0±2.6	20.0±2.65	134.7±2.08[*]	6.83±0.42[*]	95.7±2.52	17.3±2.31	23.3±4.01
	p-value	*0.245*	*0.033*	*0.097*	*0.658*	*0.144*	*0.019*	*0.033*	*0.114*	*0.202*	*0.373*
	F-value	*2.035*	*9.039*	*4.410*	*0.466*	*3.261*	*12.582*	*12.582*	*3.925*	*2.446*	*1.275*
	Remark	*NS*	*S*	*NS*	*NS*	*NS*	*S*	*S*	*NS*	*NS*	*NS*

*S– Significant at p<0.05 (ANOVA); NS – Non-significant at p<0.05 (ANOVA); 1 – significant at p<0.05, PC compared with NC (Turkey's post hoc; # and * - significant, at p<0.05, TG compared with PC and NC respectively (Turkey's post hoc)*

Table 12: Determination of effect of Jathropha interrigima (PRE and POST) on electrolytes of male albino wistar rats exposed to chromium in sub-chronic phase

Phases	Groups	Parameters Mean ± SD									
		Pre					Post				
		(mmol/l)	(mmol/l)		(mmol/l)	(mmol/l)	(mmol/l)	(mmol/l)	(mmol/l)	(mmol/l)	(mmol/l)
30 Days	NC	126.5±2.12	5.1±0.07	91.5±2.12	21.5±0.71	9.5±0.71	126.5±2.12	4.4±0.14	91.5±2.12	13.5±2.12	14.0±0.0
	PC	132.5±0.71	5.5±0.42	95.5±2.12	22.5±0.71	10.5±0.71	130.0±0.0	5.7±0.0[1]	97.0±0.0	15.0±0.0[1]	21.0±0.0[1]
	TG	130.7±4.04	5.7±0.4	95.7±1.15	20.6±0.5	12.0±2.0	134.7±2.08[*]	4.5±0.2[#]	95.7±2.52	20.0±1.0[#]	12.7±0.5[*#]
	p-value	*0.246*	*0.274*	*0.107*	*0.085*	*0.285*	*0.019*	*0.002*	*0.114*	*0.010*	*<0.001*
	F-value	*2.035*	*1.819*	*4.122*	*4.857*	*1.746*	*12.582*	*44.171*	*3.925*	*18.132*	*267.143*
	Remark	*NS*	*NS*	*NS*	*NS*	*NS*	*S*	*S*	*NS*	*S*	*S*

*S– Significant at p<0.05 (ANOVA); NS – Non-significant at p<0.05 (ANOVA); 1 – significant at p<0.05, PC compared with NC (Turkey's post hoc; # and * - significant, at p<0.05, TG compared with PC and NC respectively (Turkey's post hoc*

Table 13: Determination of effect of Jathropha interrigima (PRE and POST) on electrolytes of male albino wistar rats exposed to chromium in chronic phase

Phases	Groups	Parameters Mean ± SD									
		Pre					Post				
		Na^+ (mmol/l)	K (mmol/l)	Cl (mmol/l)	HCO3 (mmol/l)	AG (mmol/l)	Na^+ (mmol/l)	K (mmol/l)	Cl (mmol/l)	HCO3 (mmol/l)	AG (mmol/l)
60 Days	NC	126.5±2.12	4.8±0.1	91.5±2.12	21.5±0.71	9.5±0.71	126.5±2.12	4.4±0.14	126.5±2.1	13.5±2.1	14.0±0.0
	PC	132.7±0.71	5.5±0.4	95.5±2.12	22.5±0.71	10.5±0.71	130.0±0.0	$5.7±0.0^{1}$	97.0±0.0	15.0±0.0	$21.0±0.0^{1}$
	TG	133.7±3.2	4.7±0.4	95.7±1.15	20.7±0.58	12.0±2.0	$130.3±0.5^{*}$	$4.5±0.2^{\#}$	95.7±2.52	$20±1.0^{*\#}$	$12.6±0.7^{*\#}$
p-value		*0.079*	*0.138*	*0.107*	*0.085*	*0.285*	*0.043*	*0.002*	*0.114*	*0.010*	*<0.001*
F-value		*5.124*	*3.382*	*4.122*	*4.857*	*1.756*	*7.622*	*44.171*	*3.925*	*18.132*	*267.143*
Remark		*NS*	*NS*	*NS*	*NS*	*NS*	*S*	*S*	*NS*	*S*	*S*

*S– Significant at p<0.05 (ANOVA); NS – Non-significant at p<0.05 (ANOVA); 1 – significant at p<0.05, PC compared with NC (Tukey's post hoc; # and * - significant, at p<0.05, TG compared with PC and NC respectively (Turkey's post hoc*

Table 14: Determination of effect of Jathropha interrigima (PRE and POST) on renal biomarkers of male albino wistar rats exposed to chromium in acute phase

Phases	Groups	Parameters Mean ± SD							
		Pre				Post			
		CRP (mmol/l)	KIM (mmol/l)	Urea (mmol/l)	Cr (mmol/l)	CRP (mmol/l)	KIM (mmol/l)	Urea (mmol/l)	Cr (mmol/l)
15 Days	NC	1.3±0.0	14.6±0.5	3.3±0.07	52.0±1.41	1.3±0.0	14.6±0.49	3.2±0.07	52.0±1.41
	PC	1.4±0.0	14.2±0.35	3.9±0.71	47.0±4.24	$1.5±0.0^{1}$	14.3±0.0	2.7±0.0	54.0±0.0
	TG	1.37±0.06	13.5±0.25	3.6±0.38	58.3±9.45	$1.6±0.06^{*\#}$	$16.7±0.1^{*\#}$	3.1±0.85	58.3±9.45
p-value		*0.151*	*0.070*	*0.309*	*0.409*	*0.005*	*0.001*	*0.652*	*0.599*
F-value		*3.143*	*5.547*	*1.598*	*1.538*	*26.286*	*67.434*	*0.476*	*0.584*
Remark		*NS*	*NS*	*NS*	*NS*	*S*	*S*	*NS*	*NS*

*S– Significant at p<0.05 (ANOVA); NS – Non-significant at p<0.05 (ANOVA); 1 – significant at p<0.05, PC compared with NC (Turkey's post hoc); # and * - significant, at p<0.05, TG compared with PC and NC respectively (Turkey's post hoc)*

Table 15: Determination of effect of Jathropha interrigima (PRE and POST) on renal biomarkers of male albino wistar rats exposed to chromium in sub-chronic phase

Phases	Groups	Parameters Mean ± SD							
		Pre				Post			
		CRP (mmol/l)	KIM (mmol/l)	Urea (mmol/l)	Cr (mmol/l)	CRP (mmol/l)	KIM (mmol/l)	Urea (mmol/l)	Cr (mmol/l)
30 Days	NC	1.2±0.07	11.4±0.14	2.6±0.07	52.0±1.41	1.3±0.0	14.6±0.49	2.7±0.14	52.0±1.41
	PC	1.2±0.0	12.2±0.14	2.9±0.14	47.0±4.24	2.3±0.0[1]	18.4±0.0[1]	4.4±0.0	85.0±0.0
	TG	1.5±0.12	16.1±1.72[*]	3.2±0.21[*]	54.7±3.5	1.3±0.06[#]	14.4 ±0.59[#]	2.6±0.15[#]	55.0±4.0[#]
p-value		*0.016*	*0.025*	*0.039*	*0.150*	*<0.001*	*0.002*	*<0.001*	*0.001*
F-value		*14.060*	*10.675*	*8.183*	*3.168*	*446.286*	*48.177*	*129.914*	*82.437*
Remark		*S*	*S*	*S*	*NS*	*S*	*S*	*S*	*S*

*S– Significant at p<0.05 (ANOVA); NS – Non-significant at p<0.05 (ANOVA);1 – significant at p<0.05, PC compared with NC (Turkey's post hoc); # and * - significant, at p<0.05, TG compared with PC and NC respectively (Turkey's post hoc)*

Table 16: Determination of effect of Jathropha interrigima (PRE and POST) on renal biomarkers of male albino wistar rats exposed to chromium in chronic phase

Phases	Groups	Parameters Mean ± SD							
		Pre				Post			
		CRP (mmol/l)	KIM (mmol/l)	Urea (mmol/l)	Cr (mmol/l)	CRP (mmol/l)	KIM (mmol/l)	Urea (mmol/l)	Cr (mmol/l)
60 Days	NC	1.2±0.71	11.4±0.14	2.6±0.07	52.0±1.41	1.3±0.0	14.6±0.49	2.7±0.14	52.0±1.41
	PC	1.2±0.0	12.2±0.14	2.9±0.14	47.0±4.2	2.3±0.0[1]	18.4±0.0[1]	4.4±0.0[1]	85.0±0.0[1]
	TG	1.5±0.1[*#]	16.1±1.72[*]	3.2±0.21[*]	54.7±3.51[#]	1.3±0.06[*#]	14.4±0.59[#]	2.6±0.15[#]	55.0±4.0[#]
p-value		*0.016*	*0.025*	*0.039*	*0.150*	*<0.001*	*0.002*	*<0.001*	*0.001*
F-value		*14.060*	*10.675*	*8.183*	*3.168*	*446.286*	*48.177*	*129.914*	*82.437*
Remark		*S*	*S*	*S*	*NS*	*S*	*S*	*S*	*S*

*S– Significant at p<0.05 (ANOVA); NS – Non-significant at p<0.05 (ANOVA);1 – significant at p<0.05, PC compared with NC (Turkey's post hoc); # and * - significant, at p<0.05, TG compared with PC and NC respectively (Turkey's post*

7.5.3 Studies on *Pentraclethra macrophylla* seed (Ugba)

1. Antioxidant potentials of *pentraclethra macrophylla* seed (Ugba) on mercury toxicity induced hepatic, renal and testicular stress in male Albino rats.
2. Assessment of renoprotective effect of *pentraclethra macrophylla* seed (Ugba) against mercury induced acute kidney injury in male Albino rats.

Nwahiri J.D., Tamuno-Emine D.G., **Nwachuku E.O.** and Bartimaeus E.S., 2021

Pentaclethra macrophylla, African oil bean seed commonly referred to as ugba (Plate 7) is extensively consumed in the eastern part of Nigeria. This seed is known to contain several antioxidants (refs 9&10) which function as scavengers of reactive oxygen species (ROS) and could be useful in ameliorating oxidative stress generated by toxicants. This study was therefore aimed at assessing the antioxidant potential *Pentaclethra Macrophylla* seed against mercury induced hepatic, renal and testicular oxidative stress in albino rats.

The results obtained showed that mercury chloride has the potential to cause hepatic, renal and testicular toxicity to adult male albino rats. However, treatment with *Pentaclethra Macrophylla* seed extract ameliorated oxidative stress in the liver, (Tables 17 & 18), kidney (Tables 19 & 20) and testis (Tables 21 & 22).

Plate 7: *Pentaclethra macrophylla seed*

Table 17: Liver MDA. SOD and GSH concentrations of rats exposed to mercury chloride ($Hgcl_2$) and *Pentaclethra macrophylla* (PM) Seed Extract for thirty (30) days

Parameters/Grps	MDA (µmol/mg protein)	SOD (U/mg protein)	GSH (U/mg protein)	P-value	Remark
1. Control	9.48±1.63	63.45± 5.07	1.72±0.65	0.217	NS
2. HgCl (3.0mg/kg)	47.45±7.91[a]	24.74 ± 1.68[a]	0.60± 0.51[a]	0.000	S
3.HgCl (3.0mg/kg)+PM(100mg/kg)	28.91±6.99[b]	68.43± 9.79[b]	1.68± 0.36[b]	0.000	S
4.HgCl (3.0mg/kg)+PM(200mg/kg)	16.71±3.82[a]	81.04±12.37[a,b]	3.01±0.52[a,b]	0.007	S
5.PM (100mg/kg)	10.12±1.74[b]	52.11± 3.52[b]	2.26 ± 1.14[b]	0.112	NS
6.PM (200mg/kg)	6.33±1.62[b]	59.97±5.24[a,b]	2.58±0.59[a,b]	0.000	S

Each value represents the mean ± SD; [a]Significantly different from the control; [b]Significantly different from HgCl2 ($p < 0.05$).

Key: MDA – Malondialdehyde, S - Significant, NS - Not Significant, SOD – Superoxide Dismutase, GSH – Reduced glutathione

Table 18: Liver CAT and GPx activities of rats exposed to mercury chloride ($HgCl_2$) and *Pentaclethra macrophylla* (PM) Seed Extract for thirty (30) days

Parameters/Grps	CAT (U/mg protein)	GPx (U/mg proteins)	P-Value	Remark
1. Control	71.36±19.18	1.02±0.18	0.276	NS
2. HgCl (3.0mg/kg)	16.96±4.58[a]	0.07±0.29	0.002	S
3.HgCl(3.0mg/kg)+PM(100mg/kg)	52.69±19.79[b]	1.09±0.15	0.002	S
4.HgCl(3.0mg/kg)+PM(200mg/kg)	63.78±23.44[b]	1.47±0.51	0.003	S
5.PM (100mg/kg)	59.85±14.72[b]	1.56±0.49	0.001	NS
6.PM (200mg/kg)	93.99±9.17[a,b]	1.93±0.45	0.000	S

Each value represents the mean ± SD; [a]Significantly different from the control; [b]Significantly different from HgCl2 ($p < 0.05$).

Key: CAT – Catalase, S – Significant, NS - Not Significant. GPx – Glutathione peroxidase

Table 19: Kidney MDA, GSH and SOD concentrations of rats exposed to mercury chloride (HgCl2) and Pentaclethra macrophylla (PM) Seed Extract for thirty (30) days

Parameters/Grps	MDA (µmol/mg protein)	GSH (U/mg protein)	SOD (U/mg protein)	P-value	Remark
1. Control	7.79 ± 1.07	1.53±1.55	57.58 ± 7.32	0.256	NS
2. HgCl (3.0mg/kg)	36.34±5.35[a]	0.85± 0.19	22.06 ± 2.90[a]	0.000	S
3.HgCl(3.0mg/kg)+PM(100mg/kg)	15.77±1.55[a,b]	2.07±0.6	54.8 ± 6.21[b]	0.000	S
4.HgCl(3.0mg/kg)+PM(200mg/kg)	8.77±0.85[b]	2.13±1.07	65.51 ± 7.21[b]	0.002	S
5.PM (100mg/kg)	8.14±1.13[b]	1.89±0.74[a,b]	48.2 ± 5.83[a,b]	0.113	NS
6.PM (200mg/kg)	3.52±0.34[a,b]	2.68±0.75[a,b]	114.67±12.59[a,b]	0.001	S

Each value represents the mean ± SD; [a]Significantly different from the control; [b]Significantly different from HgCl2 ($p < 0.05$).

Key:MDA - Malondialdehyde, S - Significant, NS - Not Significant, GSH - Reduced Glutathione SOD - Superoxide Dismutase

Table 20: Kidney CAT and GPx activities of rats exposed to mercury chloride (HgCl2) and Pentaclethra macrophylla (PM) Seed Extract for thirty (30) days

Parameters/Grps	CAT (U/mg protein)	GPx (U/mg protein)	P-Value	Remark
1. Control	55.07±13.39	1.09±0.28	0.022	S
2. HgCl (3.0mg/kg)	12.85±3.30[a]	0.61±0.47	0.001	S
3.HgCl(3.0mg/kg)+PM(100mg/kg)	35.50±5.85[a,b]	0.89 ±0.12	0.002	S
4.HgCl(3.0mg/kg)+PM(200mg/kg)	43.19±6.80[b]	1.21±0.14	0.003	S
5.PM (100mg/kg)	47.31±8.09[a,b]	1.01 ±0.11	0.013	S
6.PM (200mg/kg)	71.02±8.43[a,b]	1.35±0.18[a,b]	0.000	S

Each value represents the mean ± SD; [a]Significantly different from the control; [b]Significantly different from HgCl2 ($p < 0.05$).

Key: CAT - Catalase, S - Significant, NS - Not Significant, GPx - Glutathione peroxidase

Table 21: Testis MDA, SOD and SH concentrations of rats exposed to mercury chloride (HgCl2) and Pentaclethra macrophylla (PM) Seed Extract for thirty (30) days

Parameters/Grps	MDA (µmol/mg protein)	SOD (U/mg protein)	GSH (U/mg protein)	P-value	Remark
1. Control	6.83±0.55	53.43±6.38	1.61±0.67	0.279	NS
2. HgCl (3.0mg/kg)	32.51±2.55[a]	20.24±2.46[a]	0.66±0.52a	0.000	S
3.HgCl(3.0mg/kg)+PM(100mg/kg)	11.34±3.25[b]	50.21±3.07[b]	2.23±0.94[b]	0.000	S
4.HgCl(3.0mg/kg)+PM(200mg/kg)	6.39±1.86[b]	59.07±4.62[b]	3.61±0.53[a,b]	0.002	S
5.PM (100mg/kg)	7.25±0.57[b]	42.41±5.20[a,b]	2.11±0.39[a]	0.230	NS
6.PM (200mg/kg)	2.50±0.72[a,b]	105.66±6.49[a,b]	3.25±0.62[b]	0.000	S

Each value represents the mean ± SD; [a]Significantly different from the control; [b]Significantly different from HgCl2 ($p < 0.05$).

Key: MDA - Malondialdehyde, S - Significant, NS - Not Significant, SOD - Superoxide Dismutase, GSH - Reduced glutathione

Table 22: Testis CAT and GPx activities of rats exposed to mercury chloride (HgCl2) and Pentaclethra macrophylla (PM) Seed Extract for thirty (30) days

Parameters/Grps	CAT (U/mg protein)	GPx (U/mg proteins)	P-Value	Remark
1. Control	60.77 ±11.91	1.19±0.15	0.005	S
2. HgCl (3.0mg/kg)	14.26 ±3.02[a]	0.70±0.27	0.001	S
3.HgCl(3.0mg/kg)+PM(100mg/kg)	32.20±6.15[a,b]	1.02±0.28	0.004	S
4.HgCl(3.0mg/kg)+PM(200mg/kg)	37.96±7.78[a,b]	1.15±0.06[b]	0.001	S
5.PM (100mg/kg)	45.92±9.39[a,b]	1.37±0.15	0.057	S
6.PM (200mg/kg)	102.69±19.81[a,b]	1.56±0.42	0.000	S

Each value represents the mean ± SD; [a]Significantly different from the control; [b]Significantly different from HgCl; ($p < 0.05$).

Key : CAT - Catalase, S - Significant. GPx - Glutathione peroxidase.

7.5.4. Studies on *Ocimum gratissimum* (Scent leaf) and *Xylopia aethiopica* (Uda) in alcohol induced hepatotoxicity.

Chuks-Oguine N.C., Bartimaeus E.S., and **Nwachuku E.O.,**

2020

Ocimum gratissimum is a herb wildly distributed in Nigeria, other parts of West African and India (Plate 8). Several native names have been given to it e.g. ncha-anwu by the Igbos. Due to its aromatic flavour, it is also commonly referred to as 'scent leaf'. From phytochemical studies, this herb has been found to be rich in antioxidants (Akinyemi *et al* 2005; Orafidiya *et al* 2001).
Xylopia aethiopica, also called Ethiopian pepper, negro pepper, African pepper is commonly referred to as uda by the Igbos of Nigeria, as sesedu in the west and Kimba in the north. It has also been shown to be rich in antioxidant components by various studies (Ogbonnia *et al*, 2008).
Both herbs are extensively used in the preparation of 'pepper soup' consumed commonly for medicinal purposes and also as delicacies at homes and at beer parlours.
This study was aimed at evaluating the antioxidant potential of *ocimum gratissimum* (ncha-anwu leaf) and *xylopia aethiopica* (uda) on alcohol-induced hepatotoxicity in albino rats. Based on the results (tables 23 & 24), it was concluded that *ocimum gratissimum* and *xylopia aethiopica* reduced oxidative stress in alcohol induced hepatotoxic rats. The significance of this is that these herbs when taken along with alcohol as pepper soup ameliorates the toxicity of alcohol on the liver.

Plate 8: *Ocimum gratissimum*

Plate 9: *Xylopia aethiopica*

Table 23: MDA and TAOC levels for induced rats treated with aqueous extract of *Ocimum gratissimum*

Groups	MDA(ng/m l)	TAOC(ng/ml)
Grp 1 (NC)	86.20 ± 17.92	1.90 ± 5.80
Grp 2 (Eth Control)	679.60 ± 157.39 [a]	0.10 ± 0.60 [a]
Grp 3 (200 mgOcim)	81.40 ± 17.78 [b]	2.00 ± 2 .50[b]
Grp 4 (400 mgOc im)	101.60 ± 11.88 [b]	2.00 ± 2.50 [b]
Grp 5 (600 mgOcim)	93.20 ± 25.04 [b]	1.90 ± 2.50[b]
F valu e	66.317	9.720
P value	<0.001	<0.001

Superscripts depict significant p values (Post hoc test), a-compared with NC, b – compared with Eth control

***Table 24: MDA and TAOC for induced rats treated with aqueous extract* Xylopia aetiopica**

Groups	MDA(ng/ml)	TAOC(ng/ml)
Grp 1 (NC)	86.20 ± 17.92	2.98 ± 1.59
Grp 2 (EthControl)	679.60 ± 157.39 [a]	0.22 ± 0 .21[a]
Grp 6 (200 mgXylop)	108.20 ± 11.16 [b]	1.96 ± 0.2 3[b]
Grp 7 (400 mgXylop)	93.00 ± 30.36 [b]	2.12 ± 0.17 [b]
Grp 8 (600 mgXylop)	165.00 ± 66.14 [b]	2.06 ± 0.28[b]
F value	53.372	9.164
P value	<0.001	<0.001
Remark	S	S

Superscripts depict significant p values (Post hoc test), a-compared with NC, b – compared with Eth control

7.5.5. Studies on *Hypoestes Rosea* (polka dot plant)

Assessment of the Antioxidant potential of *Hypoestes rosea* leaf in lead acetate induced Albino rats.

Uwikor F.K., **Nwachuku E.O.,** Igwe F., Echonwere B. & Bartimaeus E.S. (2020)

Plate 10: Hypoestes rosea (polka dot plant)

The leaves of *Hypoestes Rosea* (Plate 10) contain phytochemical compounds which are rich in antioxidants (Egbe *et al,* 2022). In the study the therapeutic effect of aqueous extract of *hypoestes rosea* leaf on oxidative stress markers of lead acetate-induced albino rats at acute and sub-chronic phases at pre-treatment and post-treatment phases. Results showed that the extract was able to significantly reverse the effect of lead acetate in both phases and this was dose dependent in both the female and male rat.

Table 25. Antioxidant parameter (SOD and TAC) activities of lead acetate induced female albino rats post-treated with *Hypoestes rosea* in the acute phase

Experimental Group	SOD (U/ml)	TAC (U /ml)
	Mean ± SEM	
EC	318.58±0.065 #	5.526±0.218 #
NC	266.14±0.081 #	2.070±0.143
PC	125.38±0.085	1.200±0.172
AEHR (1 00 mg/kg)	300.92±0.062 #	2.470±0.181 #
AEHR (200 mg/kg)	320.46±0.101 #	4.970±0.295 #
AEHR (300 mg/kg)	329.98±0.047 #	5.210±0.161 #
P-Value	<0.001	<0.001
F-Value	84.738	86.566

 # - significant at $p < 0.05$ when compared with PC

Table 26. Antioxidant parameter (SOD and TAC) activities of lead acetate induced female albino rats pre-treated with *Hypoestes rosea* in the acute phase

Experimental G roup	SOD (U/ml)	TAC (U/ml)
	Mean ± SEM	
EC	318.59±0.065 #	5.526±0.218 #
NC	266.14±0.081 #	2.070±0.143
PC	125.38±0.085	1.200±0.172
AEHR (100 mg/kg)	289.26±0.031 #	3.746±0.222 #
AEHR (200 mg/kg)	295.86±0.056 #	5.502±0.401 #
AEHR (300 mg/kg)	315.46±0.050 #	7.022±0.383 #
P-Value	<0.001	<0.001
F-Value	67.78	63.554

 # - significant at p < 0.05 when compared with PC

Table 27. Antioxidant parameter (SOD and TAC) activities of lead acetate induced male albino rats post-treated with *Hypoestes rosea* in the acute phase

Experimental Group	SOD (U/ml)	TAC (U/ml)
	Mean ± SEM	
EC	328.52±0.014 #	9.980±0.5 70#
NC	277.78±0.017 #	6.620±0.426 #
PC	135.22±0.046	2.072±0.288
AEHR (100 mg/kg)	310.66±0.012 #	5.140±0.326
AEHR (200 mg/kg)	328.48±0 .019#	8.280±0.620#
AEHR (300 mg/kg)	346.22±0.040 #	9.272±0.832 #
P-Value	<0.001	<0.001
F-Val ue	95.073	25.453

 # - significant at p < 0.05 when compared with PC

Table 28. Antioxidant parameter (SOD and TAC) activities of lead acetate induced male albino rats Pre-treated with *Hypoestes rosea* in the acute phase

Experimental Group	SOD (U/ml)	TAC (U/ml)
	Mean ± SEM	
EC	328.52±0.014 #	9.980±0.5 70#
NC	277.78±0.017 #	6.620±0.426 #
PC	135.22±0.046	2.072±0.288
AEHR (100 mg/kg)	299.10±0.032 #	7.320±0.647 #
AEHR (200 mg/kg)	305.44±0.040 #	10.530±0.7 37#
AEHR (3 00 mg/kg)	325.48±0.017 #	11.434±0.231 #
P-Val ue	<0.001	<0.001
F-Value	67.781	44.618

 # - significant at p < 0.05 when compared with PC

Table 29. Antioxidant parameter (SOD and TAC) activities of lead acetate induced female albino rats post-treated with *Hypoestes rosea* in the sub-chronic phase

Experi mental Group	SOD (U/ml)	TAC (U/ml)
	Mean ± SEM	
EC	340.24±0.020	8.540±0.461
NC	283.42±0.040	4.640±0.335
PC	125.38±0.08 5	1.200±0.172
AEHR (100 mg/kg)	322.46±0.067	9.330±0.846
AEHR (200 mg/kg)	342.70±0.032	10.920±1.0 60
AEHR (300 mg/kg)	354.52±0.041	11.060±0.592
P-value	<0.001	<0.001
F-value	129.431	36.129

Table 30. Antioxidant parameter (SOD and TAC) activities of lead acetate induced female albino rats pre-treated with *Hypoestes rosea* in the sub-chronic phase

Experimental Group	SOD (U/m l)	TAC (U/ml)
	Mean ± SEM	
EC	340.24±0.02 0	8.540±0.461
NC	283.42±0.040	4.640±0.335
PC	125.38±0.085	1.200±0.172
AEHR (100 mg/kg)	306.92±0.067	10.080±0.511
AEHR (200 mg/kg)	316.94±0.032	12.064±0.385
AEHR (300 mg/kg)	341.22±0.041	12.940±0.43 5
P-value	<0.001	<0.001
F-value	17.718	129.489

Table 31. Antioxidant parameter (SOD and TAC) activities of lead acetate induced male albino rats post-treated with *Hypoestes rosea* in the sub-chronic phase

Experimental Group	SOD (U/ml)	TAC (U/ml)
	Mean ± SEM	
EC	350.36±0.019	11.170±0.374
NC	293.12±0.032	7.130±0.487
PC	135.22±0.046	2.072±0.288
AEHR (100 mg/kg)	331.66±0.0 07	6.270±0.348
AEHR (200 mg/kg)	352.66±0.022	8.670±0.438
AEHR (300 mg/kg)	364.44±0.030	12.610±0. 505
P-value	<0.001	<0.001
F-value	65.149	82.411

Table 32. Antioxidant parameter (SOD and TAC) activities of lead acetate induced male albino rats pre-treated with Hypoestes rosea in the sub-chronic phase

Experimental Group	SOD (U/ml)	TAC (U/ml)
	Mean ± SEM	
EC	350.36±0.019	11.170±0.374
NC	293.12±0.032	7.130±0.487
PC	135.22±0.046	2.072±0.288
AEHR (100 mg/kg)	316.96±0.032	6.270±0.348
AEHR (200 mg/kg)	324.78±0.039	11.380±0.435
AEHR (300 mg/kg)	351.64±0.018	13.370±0.306
P-value	<0.001	<0.001
F-value	56.949	109.035

8.0 CONCLUSION AND RECOMMENDATION

8.1 CONCLUSION

Our exposure to different toxic chemicals contained in everyday household products has been highlighted in this inaugural lecture. Routes of exposure include inhalation, dermal absorption and ingestion in the digestive tract. Exposure can be acute, sub-acute, sub-chronic or chronic depending on the dose and duration of exposure. Toxic chemicals have been classified according to potential health effects as Teratogens, Mutagens, Carcinogens, Neurotoxicants, and endocrine disruptors.

Exposure to toxic chemicals in everyday products can be reduced by reading labels on products, handling toxic chemicals with care, use of products in moderation, avoiding mixing of chemical products, use of personal protective equipment, proper ventilation and storage.

Animal studies on dichlorvos (sniper), sachet packaged water, tartrazine food/drink colourants and heavy metals in some

herbal oils in Port Harcourt, confirm the potential for negative health effects by these chemicals.

Some other studies have shown great potential for remediation against these toxic chemicals by some indigenous plants such as *Elaeis oleifera, Jathropha species, Pentaclethra macrophylla, ocimum gratissimum* and *Xylopia aethiopica.*

8.2 RECOMMENDATIONS

The recommendations from this inaugural lecture are as follows;

1. There should be better legislation and enforcement of available legislation, for instance, some household pesticides are listed on the list of banned pesticides by NAFDAC but implementation of the ban has not been effective.

2. Government, through its regulatory agencies, should ensure that companies label their products properly with relevant information about toxic risk, ensure they are not using banned ingredients, are sticking to allowable limits and that safety tests are regularly performed by these companies on their products.

3. There should be less consumption of food and drinks packaged in plastics, instead safer alternatives should be used whenever possible.

4. If plastics must be used, they should be kept away from sunlight and other sources of heat to avoid leaching of chemicals.

Indigenous plants with high antioxidant potentials should be included in our diets to help ameliorate or provide therapy for health effects of toxic chemicals in everyday products.

5. More toxicology laboratories should be set up by both the public sector and private sector to enhance early detection of chemical toxicants in order to prevent severe health outcomes resulting from chemical toxicity.

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Vice Chancellor Sir, in the course of my training and career, I have passed through the mentorship of many distinguished teachers whom I would like to recognize at this point. The first among them is Prof. (Mrs) Bene Willie Abbey who taught me during my Msc in Uniport and has remained a role model and mentor to this day. There was also Professor Nsirim Nduka of blessed memory, whom I met during my short stint at Department of Chemical Pathology, Uniport. He became a mentor and father who encouraged me to obtain my Msc degree and go into academics. Professor Victor Wakwe is another mentor and father. Professor Dede of blessed memory was my PhD supervisor, father and mentor. It was he who ignited my passion for Toxicology.

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Professor Edna Nwachuku Ibegbulem is a Professor of Chemical Pathology with the Rivers State University, with current research interest in Environmental Toxicology and Medicinal plants. She attended primary school at Santa Maria Primary School Aba and Federal Government Girls College Owerri, graduating with a Division 1 in 1979. She obtained BSc (honours) in Biochemistry/Physiology, from the University of East London, an Msc in Medical Biochemistry and Immunochemistry from University of Port Harcourt in 1991, and a PhD in Chemical Pathology from the Rivers State University of Science and Technology in 2010. She also holds the professional diploma of the Medical Laboratory Science Council of Nigeria in Chemical Pathology and a certificate in Leadership and Management in Health from the University of Washington.

Her teaching experience spans over 30years, from 1992 to date, during which she has successfully supervised 25 PhDs, 20 Mscs and over 50 undergraduates, and has published over 100 articles in both local and international peer reviewed journals.

Professor Edna Nwachuku Ibegbulem is a seasoned Leader/Administrator. In this capacity she has been Head, Department of Medical Laboratory Science RSU, Medical Laboratory Science Council of Nigeria (MLSCN) Professional Examiner to several Nigerian Universities, and member MLSCN Accreditation teams to several universities.

Professor Edna Nwachuku Ibegbulem is a foundation Fellow of West African Postgraduate College of Medical Laboratory Science (FWAPCMLS), Fellow, Association of Medical Laboratory Scientists of Nigeria(FAMLSN), Member Board of Trustees, Society for Chemical Pathology Scientists of Nigeria, Member Nigerian Environmental Society (MNES), Member African Society for Laboratory Medicine (ASLM), Member Guild of Medical Laboratory Scientists of Nigeria (GMLD), and Member Association of Female Medical Laboratory Scientists (AFMLS).

Her appointments in other universities include;
Visiting Scholar, Pamo University of Medical Sciences; Sabbatical appointment; Federal University Otuoke; External Examiner; Niger Delta University, Bayelsa.

Beyond Rivers State University, Professor Edna Nwachuku Ibegbulem is very active professionally, she has chaired scientific committees of the Guild of Medical Laboratory Directors (GMLD) National and South-South Conferences. She is part of a group of scientists from some Nigerian Universities such as, University of Calabar, University of Ibadan and Rivers State University, headed by currently involved in the development of and indigenous PCR test kit for COVID-19 disease, which is currently going through validation by

MLSCN. She is also the Director, Biotek Medical Laboratory, Port Harcourt.

Professor Edna Nwachuku Ibegbulem is a recipient of several awards of honour for outstanding contribution and dedication to growth of Medical Laboratory Science Profession from GMLD Rivers State, Young Medical Laboratory Scientists, National and Rivers State, AMLSN Rivers State and NIMELSSA RSU and numerous others.

She is also very committed in the things of God. She is a Lay-reader in the Anglican Communion, Pioneer Member St. Nicholas Anglican Church, Aluu-Uniport, where she has served as chairman and member of several church committees over the years, including the PCC, Harvest Committee, Medical Committee, Mothering Sunday Committee etc.

Professor Edna Nwachuku Ibegbulem is a Scholar, seasoned Administrator, committed Christian and Mother of two exceptionally gifted children.