# POISONS AND POISONING THE NIGHTMARISH OF A SOCIETY WITHOUT DIAGNOSTIC TOOLS

AN INAUGURAL LECTURE,
2011/2012

ABATAN, MATTHEW
OLUWOLE

UNIVERSITY OF IBADAN

# POISONS AND POISONING THE NIGHTMARISH OF A SOCIETY WITHOUT DIAGNOTIC TOOLS

*An inaugural lecture delivered at the University of Ibadan*

*on Thursday, 11 October, 2012*

*By*

**ABATAN, MATTHEW OLUWOLE**
*Professor of Pharmacology/Toxicology*
*Faculty of Veterinary Medicine*
*University of Ibadan*
*Ibadan, Nigeria.*

**UNIVERSITY OF IBADAN**

Ibadan University Press
Publishing House
University of Ibadan
Ibadan, Nigeria.



*First Published 2012*



**ISBN: 978 – 978 – 8414 – 91 – 9**

*Printed by: Ibadan University Printery*

*The Vice-Chancellor, Deputy Vice-Chancellor (Administration), Deputy Vice-Chancellor (Academic), Provost of the College of Medicine, Dean Faculty of Veterinary Medicine, Dean of the Postgraduate School, Deans of other Faculties and of the Students, Distinguished Ladies and Gentlemen.*

I am humbled by the grace and the opportunity to be called upon to deliver the second inaugural lecture this year from the Faculty of Veterinary Medicine. It would have been my turn conventionally in 2014. When the question of who should be the next to deliver the second inaugural lecture for the Faculty for this year became the main agenda for Faculty Board meeting, about two months ago, when the scheduled inauguree declined, I presumed the next Professor will take up the challenge. However when a memo was sent to five Professors in the Faculty to signal our readiness to take up the challenge to give the inaugural lecture, I responded by saying if none of the Seniors ahead of me was ready to deliver the lecture, I will take it up God willing. The Vice-Chancellor Sir, this is how the mantle fell on me to be standing in your presence and that of this wonderful audience of eminent scholars, professionals and students to present this lecture. I give God the Almighty all the glory.

The first inaugural lecture from the Department of Veterinary Physiology, Biochemistry and Pharmacology was delivered by Professor Matthew Ojo Olowookorun, a Physiologist, in 1985 titled "The Digestive System, a perfect example of United we Stand, Divided we Fall; the second was by Professor Reuben Arowolo, a Pharmacologist in 1995, titled: Protecting our Livestock Resources; the third was delivered last year by Professor Johnson Oyewale, a Physiologist, titled Blood: Different Strokes for Different Animals". Today I humbly present the fourth lecture coming from my department, but the second from the Pharmacology unit of the department, titled "Poisons and Poisoning, the nightmarish of a Society without diagnostic tools".

At this juncture, Mr. Vice-Chancellor Sir, I wish to give special recognition to the Academic Staff Union of Universities (ASUU) and all its members who have not at any time relented in their commitments and effort to ensure that Universities in this country are able to attain the level they are this day in terms of infrastructure, facilities and conditions of service, which would have been unimaginable. When I had the opportunity of travelling to Western Germany for the first time in 1994 for a conference under the sponsorship of the Government of Zimbabwe where I was having my Sabbatical leave, I decided within me to leave the academia and go for professional practice in the field any time I returned to Nigeria. This was because I felt astounded by the quality of presentation I saw at this conference by Scientist from other more serious scholarly countries. However, I returned to Nigeria in 1996 to be noticed by the then leadership of ASUU which were embroiled in a strike for improved funding to our universities and education in general. This strike interestingly lasted for six months. It was at the end of the strike that I heard this philosophical jingle on Radio OYO which sounds thus, '*Iya aburo si o si nkan, Ki ni ohun lo ba mi nle; e joo nibo nile igbonse yin wa ni bii. Hee awa o ni ile igbonse kankan. Ah e ha ti n se e? Bi ka lo sehinkule, ka lo oju agbare ba un ba un, Ha eyun o ma daa o, iwa obun ni o.*" Considering this one whom will you want to apportion blame for such abhorable societal behaviour? How many public toilets do we have in our cities?

So, Mr. Vice-Chancellor I also had to stay behind in the system moving from place to place today in chemistry, tomorrow at OAU and then at Unilag, often using my resources to survive in the Academia

**Introduction**

With this unsavory background, it has become compelling to recount my experience in the Nigerian academic community where research is done with lots of stress so that the researcher in Nigeria is always experiencing poisoning

endogenously. Poisoning is a common occurrence in our society and several poisons and poisoning do exist or take place either at home or at work because we deal with poisonous substances naturally or otherwise. Recently, there were reports of lead poisoning in children in Zamfara state. There are many symbols to denote industrial or domestic poisons:




Poisons are agents that are capable of producing deleterious response in a biological system, seriously injuring functions or producing death (Klaassen 2001). The fields of Medicine (particularly Veterinary) and Zoology often distinguish a poison from a toxin, and from venom. Toxins are poisons produced by some biological function in nature, eg. Aflatoxins are produced by mold such as *Aspergilus flavus*, whereas venoms are usually defined as toxins that are injected by a bite or sting to cause their effect, for example snake venom, while other poisons are generally defined as substances absorbed through epithelial linings, such as the skin or gut.

Some poisons represent toxins, usually naturally produced by bacterial proteins that cause tetanus and botulism. It is also significant to make a delineation between a poisonous

organism which is one that is harmful to consume and a venomous organism that uses poison (venom) to kill its prey or to defend itself while still alive. However, it is also possible for a single organism to be both venomous and poisonous.

These definitions may not be the same in the field of nuclear physics, where a poison is a substance that obstructs or inhibits a nuclear reaction.

Paracelsus (1943-1541), a Swiss/German scientist and the father of toxicology, once wrote: "What is there that is not a poison, all things are poison and nothing is without poison. Only the dose makes a thing not a poison." This is often condensed to: "The dose makes the poison" (Klaassen 2001).

The term "poison" is used often colloquially to describe any harmful substance, particularly corrosive substances, carcinogens, mutagens, teratogens and harmful pollutants, and sometimes to exaggerate the dangers associated with some chemicals.


The term toxicant is used in describing substances that are produced by or are a by-product of anthropogenic (human made) activities. Some toxicants are produced by both natural and anthropogenic activities. For example, polyaromatic hydrocarbons are produced by the combustion of organic matter, which may be by natural process as occurs with forest

fires and use of wood/charcoal for cooking or through anthropogenic means such as occurs in cigarette smoking.

In normal usage, when considering the environment, some substances are not necessarily poisons. For example, in looking at wastewater from the food industry which may contain potato/cassava juice or soap water and can be hazardous to the ecosystems of streams and rivers by consuming oxygen and causing eutrophication. However, because it is non-hazardous to humans/animals directly, it is not classified as a poison. Mr. Vice-Chancellor Sir, these submissions can be considered under the broad subject area of toxicology.

**What is Toxicology?**

Toxicology comes from the Greek words *toxicos* "poisonous" and *logos*). This is a branch of Pharmacology, concerned with the study of the adverse effects of chemicals on living organisms. It is the study of symptoms, mechanisms, treatments and detection of poisoning, especially the poisoning of people and animals.

Dioscorides, a Greek physician in the court of the Roman emperor Nero, made the first attempt to classify plants according to their toxic and therapeutic effects. Ibn Wahshiya wrote the *Book on Poisons* in the 9th or 10th century.

Mathieu Orfila is considered to be the modern father of toxicology, because he gave the subject its first formal treatment in 1813 in his *Traité des poisons*, also called *Toxicologie générale*.

In 1850, Jean Stas gave the evidence that the Belgian Count Hypolyte Visart de Bocarmé killed his brother-in-law by poisoning with nicotine

The relationship between the dose of a compound and its effects on the exposed organism is of high significance in toxicology. The chief criterion regarding the toxicity of a chemical is the dose, i.e. the amount of exposure to the substance. A term known as the lethal dose fifty (or $LD_{50}$) refers to the dose of a toxic substance that kills 50 percent of

a test population and captures the relevance of dose to poisoning. In clinical toxicology however, there is a wide variation in susceptibility between individuals because 7mg/kg of ricin will kill some horses, where 300mg/kg will not kill others. From the point of views of a clinical specialist. $LD_{50}$ is only a convenient way of expressing a probable lethal dose (Adedapo and Abatan 2006). All substances may be toxic or poisonous under the right conditions. However, there could be species/breed variations on the issue of poisoning.

**Uses of Poison**

Throughout human history, intentional application of poison has been used as a method of assassination, murder, suicide, and execution. As a method of execution, poison has been ingested, as the ancient Athenians did inhale, as with carbon monoxide or hydrogen cyanide or injected. Many languages describe lethal injection with their corresponding words for "poison shot". Poison was also employed in gunpowder warfare. Some malicious fishermen use organophosphates or leaves of some poisonous plants e.g. thrown on the surface of water to catch fishes. Hunters place concoctions of the plant *Adenum obesum* on the tip of their arrows to kill animals. The "babalawo" deceives his clients who seek his prowess by taking suspects who are alleged to have stolen or to be involved in an offense, by giving them a concoction containing for example leaves of *Pysostigma venenoswum*. The actual thief out of fears sips more of the concoction and in the process gets more of the active compound into him/her and therefore manifest the poisoning.

**Biological Poisoning**

Acute poisoning is exposure to a poison on one occasion or during a short period of time. Symptoms develop in close relation to the exposure. Absorption of a poison is necessary for systemic poisoning. Such systemic poisoning is different from substances that destroy tissue but are not absorbed, such as caustic soda or lye which are classified as corrosives rather than poisons. Furthermore, many common household medica-

tions are not labeled with skull and crossbones, although they can cause severe illness or even death.

Chronic poisoning is long-term repeated or continuous exposure to a poison where symptoms do not occur immediately or after each exposure. The patient gradually becomes ill, or becomes ill after a long latent period. Chronic poisoning most commonly occurs following exposure to poisons that bioaccumulate, or are biomagnified, such as mercury and lead. Plants close to the roads are reported to bioaccumulate lead due to exposure to fumes from petrol from the passing cars and animals consuming such plants are known to acquire the poisons (Abatan, 1992). Contact or absorption of poisons can cause rapid death or impairment. Agents that act on the nervous system can paralyze in seconds or less, and include both biologically derived neurotoxins and so-called nerve gases, which may be synthesized for warfare or industry.

Inhaled or ingested cyanide, used as a method of execution in gas chambers, almost instantly starves the body of energy by inhibiting the enzymes in mitochondria which are part of the oxidative phosphorylation process in adenosine triphosphate (ATP) production. Intravenous injection of an unnaturally high concentration of potassium chloride, such as in euthanasia for some diseased animals, which quickly stops

the heart by eliminating the cell potential necessary for muscle contraction.

Most biocides, including pesticides, are created to act as poisons to target organisms, although acute or less observable chronic poisoning can also occur in non-target organisms, including humans (who apply the biocides) and other beneficial organisms. For example, the herbicide 2, 4-Dichlorophenoxy acetic acid imitates the action of a plant hormone, to the effect that the lethal toxicity is specific to plants. Indeed, 2,4-D is not a poison, but classified as "harmful".

Many substances regarded as poisons are toxic only indirectly, by toxication. An example is "wood alcohol" or methanol, which is not poisonous itself, but is chemically converted to toxic formaldehyde and formic acid in the liver. Many drug molecules are made toxic in the liver, and the genetic variability of certain liver enzymes makes the toxicity of many compounds differ between individuals (Osweiler 1985).

**Some Human Medications Potentially Dangerous to Some Animals Paracetamol or acetaminophen**, the active ingredient in this pain reliever, is very toxic in cats. The drug interferes with oxygen uptake in the blood of cats and can result in death if not treated promptly. Acetaminophen overdose in dogs can cause severe liver damage (Abatan et al. 2006). As few as two regular-strength pills can cause overdose in dogs. The drug interferes with oxygen uptake in the blood of cats and can result in death if not treated promptly. The common aspirin can also be very toxic to cats except in a very low dose. At times, veterinarians will use aspirin as an anticoagulant for cats with heart disease (Chyka et al. 2007) just as is done in humans where aspirin is recommended in low daily doses of 75mg/kg. Dogs can tolerate this aspirin, and veterinarians will sometimes recommend it for use as a pain reliever. However, the chronic use of the drug produces side effects. Atropine, a cholinergic antagonist useful in poisoning by organophosphate could kill

the rabbit or Rottweiler when it is not used with caution (Personal observations, VTH 2007).

**Ibuprofen**: This is the active ingredient in over-the-counter medications such as "cold and flu" medications, and is a non-steroidal anti-inflammatory drug (NSAID). This drug is never recommended for cats or dogs, as it can result in severe gastric ulcers or acute kidney failure. Ibuprofen and other human NSAIDS should never be used in pets, as there are veterinary specific NSAIDS that are less toxic and are much safer for pets.

**Naproxen** is a very potent NSAID. Minute doses can result in severe symptoms of gastric ulcers, stomach perforations, or acute kidney failure in animals, and should never be used in animals (Merck Veterinary Manual 2012).

**Antidepressants**: Vomiting and lethargy are common symptoms of an overdose. In some cases, a condition known as serotonin syndrome can occur. Pets will exhibit elevated body temperatures, increased heart rates and blood pressure as well as disorientation and vocalization (Ogen et al. 1981).

**Methylphenidate** is a medication which is used for treating attention-deficit, hyperactivity disorder and are reported to elevate a pet's heart rate to a dangerous level and has the potential to cause abnormal heart rhythms and seizures. A tablet or patch could be fatal for a cat or small dog (Pet Poison Helpline 2012).

**Fluorouracil** which is an anti-cancer drug is used to treat minor skin cancers in humans. Discarded cotton swabs used to apply this medication are a prime source of pet poisonings. This drug is rapidly fatal, causing severe vomiting, seizures, and even cardiac arrest in pets. This drug should not ever be used in cats (Dorman et al. 1990).

**Isoniazid** is mentioned to be a first line tuberculosis drug with a very narrow margin of safety. However, it is extremely

dangerous to dogs. Dogs will have serious seizures and then enter a stuporous state when this medication is consumed (Villar et al. 1995).

**Pseudoephedrine** is very popular decongestant and is found in a variety of cold and sinus products. Pseudoephedrine acts as a stimulant to dogs and cats, and the animal behaves hyperactive. Head bobbing, agitation, tremors and seizures are all commonly seen.

The anti-diabetic medications **glipizide, gliclazide**, and **glyburide** belong to a class of drugs known as sulphonylureas. These tablets work by stimulating the pancreas to produce more insulin. Medications like glipizide and glyburide can cause sudden and major drops in blood sugar of pets when the animals are exposed to the drug. Symptoms include disorientation, lack of coordination, and seizures. Cats exhibit symptoms which indicate liver problems when exposed to higher doses. Toxic dosages vary according to the size of the pet. However, any exposure is dangerous. The commonly used medications for treating hyperglycemia or increased blood sugar levels in small animals are Carbamazepine, Atretol, Carbatrol, Epitol Equetro and Tegretol (Cowan and Bunch 2001; DeRuiter 2003).

There are some vitamin D derivatives used in humans to treat psoriasis (skin condition). These medications are often available in ointments or solutions. Dogs develop vomiting, depression, anorexia, diarrhea and increased urination within 12-24 hours. Toxic doses for pets are very small.

**Baclofen** is used to treat muscle symptoms caused by multiple sclerosis and spinal disorders, including spasm, pain and stiffness. Dogs show signs of vomiting, weakness, and disorientation. Dyspnoea and respiratory distress can occur. The prognosis is not good as deaths can occur from small numbers of tablets.

**Phytotoxicology or Plant Poisoning**

Mr. Vice-Chancellor, the area of plant poisoning represents one of the extensive fields of my research focus. My interest was aroused as a young student by Dr Mrs. Lowe, then of the Department of Botany and Microbiology of this University who performed the annual routine of teaching poisonous plants to Veterinary students in Pharmacology. I also researched into the medicinal values of these plants but for the purpose of focus and clarity on the topic of this lecture that aspect will not be considered.

**Why are poisonous plants important?**

- Poisonous plants are important because they cause disease and death of livestock.
- The losses as a result of plant poisoning can be direct or indirect and include death, loss of condition and ill-thrift, poor production (for example, loss of milk yield) and reproductive failure (abortions, stillbirths, birth defects, and failure to become pregnant).
- Further economic losses include the cost of control and treatment measures
- There are serious health risks connected to eating meat from animals that have died from plant poisoning.

**When is Plant Poisoning most likely to Occur?**

Animals usually avoid poisonous plants.

Generally, poisoning occurs when:

- Animals are introduced into a new area as seen with newly purchased animals from other areas. Animals on transhumans i.e. livestock which are transported on hoof from north to south.
- There is a shortage of food and animals are forced to eat the available fodder especially during drought, overstocking or during the period of early rains (after the dry season) when poisonous plants are the first to show new growth.

## Which Factors Contribute to Plant Poisoning?

There are plant factors and animal factors, which contribute to the likelihood of plant poisoning occurring.

### Plant Factors

- After the dry season, poisonous plants are usually among the first green plants to appear. A number of poisonous plants are also at their most poisonous level in the young stage when they are most attractive to livestock.
- Poisonous plants, which are not normally consumed, are often eaten during times when grazing may be scarce such as overgrazing and during adverse conditions.
- Some poisonous plants are very resistant to drought and may be the only green plants available and are therefore eaten during times of drought.
- Poisonous plants are often found as weeds in harvested lands and along roadsides (areas that are frequently used for grazing in times of scarcity).
- Certain poisonings occur after a sudden change in weather
- Wind and/or hail can knock branches of poisonous plants with their pods falling to the ground, therefore making them available to animals.
- The use of fertilisers may increase the toxicity of some plants.
- Animals are sometimes poisoned when feeding on fodder (hay, silage, or concentrates) containing poisonous plants, fungi or chemicals.

### Animal Factors

- When livestock have been kept in an area for some time, they are often familiar with the poisonous plants and will not eat these unless forced to do so. Animals that are moved from familiar areas to new pastures tend to graze less selectively and will get poisoned more easily.

- Poisoning often occurs when animals are moved from one area to another especially when they are allowed to graze along roadsides where poisonous plants occur in great numbers.
- Different species of animals are poisoned with varying degrees of severity and by different types of poisonous plants.
- Exotic breeds of livestock tend to graze less selectively, and therefore are more susceptible to poisoning than indigenous breeds.
- Some plants affect males and females in different ways.
- Young and older animals are more susceptible. Their livers do not have the capacity to eliminate the toxins and young animals also have not yet learnt to avoid poisonous plants.
- Hungry animals graze more greedily and are less selective and therefore more likely to be poisoned. This can occur in conditions of drought, or overgrazing.
- Thirsty animals look for plants with a high moisture content, which they would normally avoid. Some of these plants may be poisonous.
- Pregnant animals tend to be less selective and have a higher intake than normal and may therefore be poisoned.
- Animals in poor condition are at a greater risk of poisoning than animals in good condition.
- The skin colour of animals will determine the extent to which certain poisonous plants will affect them. For example, in the case of photosensitivity (poisonings that result in damage due to sunburn) unpigmented, white areas on the skin may become red and swollen.

**Commonly occurring Nigerian Poisonous Food Plants**

Many food plants may possess toxic parts, or are toxic unless processed, or are toxic at certain stages of their life. Among such plants are:

*Manihot esculenta* or cassava of the family Euphorbiaceae. Roots and leaves of these plant contain two cyanogenic glycosides, linamarin and lotaustralin. These are decomposed by linamarase, a naturally-occurring enzyme in cassava, liberating hydrogen cyanide . Cassava varieties are often categorized as either sweet or bitter, respectively signifying the absence or presence of toxic levels of cyanogenic glycosides. The 'sweet' cultivars can produce little cyanide per kilogram of fresh roots, whereas bitter ones may produce more than 50 times as much. Cassavas grown during drought are especially high in these toxins. A dose of 40 mg of pure cassava cyanogenic glycoside is sufficient to kill a cow. It can also cause severe calcific pancreatitis in humans, leading to chronic pancreatitis. Processing (soaking, cooking, fermentation, etc.) of cassava root is necessary to remove the toxins and avoid poisoning effects. "Chronic, low-level cyanide exposure is associated with the development of goiter and with tropical ataxic neuropathy, a nerve-damaging disorder that renders a person unsteady and uncoordinated (Osuntokun 1973; Abatan 1992). For some smaller-rooted sweet varieties, cooking is sufficient to eliminate all toxicity. The cyanide is carried away in the processing water and the amounts produced in domestic consumption are too small to have environmental impact. The larger-rooted, bitter varieties used for production of flour or starch must be processed to remove the cyanogenic glycosides. Industrial production of cassava flour, even at the cottage level, may generate enough cyanide and cyanogenic glycosides in the effluents to have a severe environmental impact.

*Lathyrus sativus* or Indian pea is a legume which produces a high-protein seed. The seeds contain variable amounts of β-N-Oxalyl-L-α,β-diaminopropionic acid or ODAP, a neurotoxic amino acid. ODAP causes wasting and paralysis if eaten over a long period, and is considered as the cause of the disease neurolathyrism, a neurodegenerative disease that causes paralysis of the lower body and emaciation of gluteal muscle (buttocks).

*Phaseolus vulgaris* called kidney bean or common bean contains the toxic compound phytohaemagglutinin, a lectin, which is present in many varieties of common bean but is especially concentrated in red kidney beans. The lectin has a number of effects on cell metabolism; it induces mitosis, and affects the cell membrane in regard to transport and permeability to proteins. It agglutinates most mammalian red blood cell types. The primary symptoms of phyto-haemagglutinin poisoning are nausea, vomiting, and diarrhea.

*Phaseolus lunatus* or lima bean. The raw beans contain dangerous amounts of linamarin, a cyanogenic glycoside. Some members of the genus *Allium* such as *Allium sativa* onions and garlic contain thiosulphate, which in high doses is toxic to dogs, cats and some other livestock.

*Solanum tuberosum* or potato of the family Solananeae contain toxic compounds known as glycoalkaloids, of which the most prevalent are solanine and chaconine. Solanine is also found in other members of the Solanaceae plant family, which includes *Atropa belladonna* ("deadly nightshade") and *Hyoscyamus niger* ("henbane"). The concentration of glycoalkaloid in wild potatoes suffices to produce toxic effects. The toxin affects the nervous system, causing headaches, diarrhea and intense digestive disturbances, cramps, weakness and confusion, and in severe cases coma and death. Tubers which are exposed to light turn green from chlorophyll synthesis, thus giving a visual clue as to areas of the tuber that may have become more toxic; however, this does not provide a definitive guide, as greening and glycoalkaloid accumulation can occur independently of each other.

*Solanum lycopersicum* or tomato like many other nightshades, the leaves and stems contain solanine that is toxic if ingested, causing digestive upset and nervous excitement. Leaves, stems, and green unripe fruit of the tomato plant also contain small amounts of the poisonous alkaloid tomatine, although levels are generally too small to be dangerous. Ripe

tomatoes do not contain any detectable tomatine. Tomato plants can be toxic to dogs if they eat large amounts of the fruit, or chew the plant material.

**Some potentially hazardous plants in Nigeria**

*Lantana camara* of the plant family Verbanaceae contain the toxic agent lantadene which is associated with severe intoxication including jaundice and photosensization in livestock (Abatan et al. 1996 and 1997; Abatan 2000).

*Abrus precatorius.* The attractive seeds contain abrin, which is related to ricin, and very potent. Symptoms of poisoning include nausea, vomiting, convulsions, liver failure, and death, usually after several days. Ingesting a single seed can kill an adult human. The seeds have been used as beads in jewelry, which is dangerous; inhaled dust is toxic and pinpricks can be fatal. The seeds are unfortunately attractive to children.

*Abrus precatorius* showing its seeds
*Adenium obesum*. Exudes a highly toxic sap which is used to coat arrow-tips for hunting.

*Adenum obesum*
*Atropa belladonna* (commonly known as deadly nightshade belongs to family Solanaceae. All parts of the plant contain tropane alkaloids. The active agents are atropine, hyoscine (scopolamine), and hyoscyamine, which have anticholinergic properties. The symptoms of poisoning include dilated pupils, sensitivity to light, blurred vision, tachycardia, loss of

balance, staggering, headache, rash, flushing, dry mouth and throat, urinary retention, constipation, confusion, hallucinations, delirium, and convulsions. The root of the plant is generally the most toxic part, though this can vary from one specimen to another. Ingestion of a single leaf of the plant can be fatal. The plant's deadly symptoms are caused by atropine's disruption of the parasympathetic nervous system's ability to regulate involuntary activities such as sweating, breathing, and heart rate. *A. belladonna* is toxic to many domestic animals, causing narcosis and paralysis. However, cattle and rabbits eat the plant seemingly without suffering harmful effects. In humans its anticholinergic properties will cause the disruption of cognitive capacities like memory and learning.

*Datura stramonium* (commonly known as jimson weed, thorn apple, stinkweed and Jamestown weed). All parts of the plant are poisonous, causing abnormal thirst, vision distortions, delirium, incoherence, coma and this could often be fatal.

*Dichapetalum madagasiense* of the family Dichapetalaceae. is a livestock poison and the plant contains the metabolic poison fluoroacetic acid which inhibits aconitase, an important enzyme on the Kreb cycle resulting in tissue anoxia (Abatan and Fajimi 1994; Abatan et al. 1996).

*Dieffenbachia* (commonly known as *dumbcane'*). All parts are poisonous, causing intense burning, irritation, and immobility of the tongue, mouth, and throat. Swelling can be severe enough to block breathing, leading to death.

*Lilium* (commonly known as lily). Most have an unknown water-soluble toxin found in all parts of the plant. Extremely poisonous, yet attractive, to cats, causing acute renal failure; 2 petals can kill.

*Nerium oleander* (commonly known as oleander). All parts are toxic, but especially the leaves and woody stems which contains nerioside, oleandroside, saponins and cardiac glycosides. Causes severe digestive upset, cardiac irregularities and contact dermatitis. The smoke of burning oleander can cause reactions in the lungs, and can be fatal.

*Nerium oleander* is toxic to humans and animals.
*Physostigma venenosum* (commonly known as Calabar bean and also as ordeal beans due to their use in trials by ordeal). The toxin in the seeds is the parasympathomimetic alkaloid physostigmine, a reversible cholinesterase inhibitor. Symptoms of poisoning include copious saliva, nausea, vomiting, diarrhea, anorexia, dizziness, headache, stomach pain, sweating, dyspepsia and seizures and can lead to cholinergic

syndrome. Physostigmine has medicinal uses which include the treatment of myasthenia gravis, glaucoma, Alzheimer's disease and delayed gastric emptying (Coelho and Birks 2001).

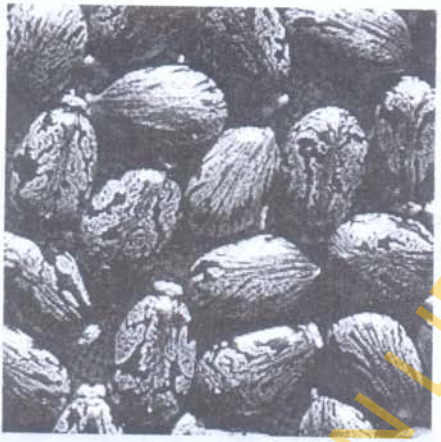
*Pteridium aquilinum* (commonly known as bracken) of the family Dennstaedtiaceae has produced carcinogenic effects in animals such as mice, rats, horses and cattle when ingested. The carcinogenic compound is ptaquiloside , which can leach from the plant into the water supply, which may explain an increase in the incidence of gastric and oesophageal cancers in humans in bracken-rich areas. The plant also contains thiaminase which produces vitamin B deficiency in horses which consume the plants.

*Pteridium aquilinum*

*Ricinus communis* (commonly known as castor oil plant or Palma Christi) of the family Euphorbiaceae. The seeds contain ricin, an extremely toxic water-soluble protein. Also present are ricinine, an alkaloid, and irritant oil. According to the 2007 edition of the Guinness Book of World Records, this plant is the most poisonous in the world. Castor oil, long used as a laxative, muscle rub, and in cosmetics, is made from the seeds, but the ricin is removed during processing. If ingested, symptoms may be delayed by up to 36 hours but commonly begin within 2–4 hours. These include a burning sensation in

mouth and throat, abdominal pain, purging and bloody diarrhea. Within several days there is severe dehydration, a drop in blood pressure and a decrease in urine. In 1978, ricin was used to assassinate Georgi Markov, a Bulgarian dissident. He was stabbed with the point of an umbrella while waiting at a bus stop near Waterloo Station in London. After his death a perforated metallic pellet was found embedded in his leg; this had presumably contained the ricin toxin (Hignett 2012). Poisoning occurs when animals ingest broken seeds or break the seed by chewing; intact seeds may pass through the digestive tract without releasing the toxin. Ducks have shown substantial resistance to the seeds.

The Plant *Ricinus communis*



Seeds of *Ricinus communis* which looks like mottled ticks

*Solanum nigrum* (commonly known as black nightshade). All parts of the plant except the *ripe* fruit contain the toxic glycoalkaloid solanine. Solanine poisoning is primarily displayed by gastrointestinal and neurological disorders. Symptoms include nausea, diarrhea, vomiting, stomach cramps, burning of the throat, cardiac dysrhythmia, headache and dizziness. In more severe cases, hallucinations, loss of sensation, paralysis, fever, jaundice, dilated pupils and hypothermia can result. In large quantities, solanine poisoning can be fatal.

*Strychnos spinosa* of the family Loganiaceae and is related to the very poisonous plant Strychnos nux-vomica. The seeds usually contain about 1.5% strychnine, an extremely bitter and deadly alkaloid. This substance throws the animal or human into intense muscle convulsions and usually kills within three hours. The bark of the tree may also contain brucine, another dangerous chemical.

*Strychnos spinosa showing the fruit*

***Tribulus terrestris***

Tribulus terrestris is of the plant family Zygophylaceae and contains a chemical component that causes hepatotoxicity and results in inablility of the poisoned animal to metabolize chlorophyll and subsequent erythrophylin excretion. This results in photosensitization. Animal studies in rats, rabbits and primates have demonstrated that administration of *Tribulus terrestris* extract can produce statistically significant increases in levels of testosterone, dihydro-testosterone and dehydroepiandrosterone, and produces effects suggestive of aphrodisiac activity (Gauthaman et al. 2002 and 2008).

Leaves and flower of *Tribulus terrestris*

***Aspergilus flavus***

Aflatoxin producing members of *Aspergillus* are common and widespread in nature. They can colonize and contaminate grain before harvest or during storage. Host crops are particularly susceptible to infection by *Aspergillus* following prolonged exposure to a high humidity environment or damage from stressful conditions such as drought, a condition which lowers the barrier to entry. The native habitat of *Aspergillus* is in soil, decaying vegetation, hay, and grains undergoing microbiological deterioration and it invades all types of organic substrates whenever conditions are favorable for its growth. Favorable conditions include high moisture content (at least 7%) and high temperature (Li, Feng-Qin et al. 2009).

Crops which are frequently affected include cereals (maize, sorghum, millet, rice, wheat), oilseeds (peanut, soybean, sunflower, cotton), spices (chilli peppers, black pepper, coriander, ginger), and tree nuts (almond, walnut, and coconut). Measurable quantities of Aflatoxin can also be found in the milk of animals which are fed contaminated feed (Bastanello et al. 1987). Virtually all sources of commercial peanut butter are reported to contain minute quantities of aflatoxin,

**Pathology**

High-level aflatoxin exposure produces an acute necrosis, cirrhosis, and carcinoma of the liver exhibited by hemorrhage, acute liver damage, oedema, alteration in digestion, and absorption and/or metabolism of nutrients. No animal species is immune to the acute toxic effects of aflatoxins including humans; however, humans have an extraordinarily high tolerance for aflatoxin exposure and rarely succumb to acute aflatoxicosis. Chronic, subclinical exposure does not lead to as dramatic of symptoms as acute aflatoxicosis. Chronic exposure also leads to a high risk of developing liver cancer, as the metabolite aflatoxin $M_1$ can intercalate into DNA and alkylate the bases through its epoxide moiety (Williams et al. 2004).

Medical research indicates that a regular diet including apiaceous vegetables such as carrots, parsnips, celery and parsley, reduces the carcinogenic effects of aflatoxin (Li, Feng-Qin et al. 2009).

**Major types of aflatoxins and their metabolites**

At least 13 different types of aflatoxin are produced in nature. Aflatoxin $B_1$ is considered the most toxic and is produced by both *Aspergillus flavus* and *Aspergillus parasiticus*. Aflatoxin $G_1$ and $G_2$ are produced exclusively by *A. parasiticus*. While the presence of *Aspergillus* in food products does not always indicate harmful levels of aflatoxin are also present, it does imply a significant risk in consumption of that product (Bastanello et al. 1987).

Aflatoxins $M_1$, $M_2$ were originally discovered in the milk of cows fed on moldy grain. These compounds are products of conversion process in the animal's liver. However, aflatoxin $M_1$ is present in the fermentation broth of *Aspergillus parasiticus* (Bingham et al. 2003; Leong, et, 2011).

**The Menace of unguarded use of Pesticides**

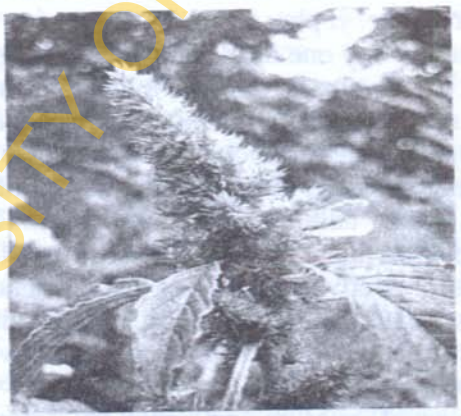
Another field of my research focus has to do with the use of pesticides. Pesticides are substances or mixture of substances

intended for preventing, destroying, repelling or mitigating any pest. The Food and Agriculture Organization (FAO) (2002 )has defined the term of *pesticide* as "any substance or mixture of substances intended for preventing, destroying or controlling any pest, including vectors of human or animal disease, unwanted species of plants or animals causing harm during or otherwise interfering with the production, processing, storage, transporting or marketing of food, agricultural commodities, wood and wood products or animal feedstuffs, or substances which may be administered to animals for the control of insects, arachnids or other pests in or on their bodies. The term includes substances intended for use as a plant growth regulator, defoliant, desiccant or agent for thinning fruit or preventing the premature fall of fruit.

**Subclasses of pesticides**

Pesticides can be classified by target organism, chemical structure, and physical state and include herbicides, insecticides, fungicides, rodenticides, pediculicides, and biocides. Plant-derived pesticides, or "botanicals", include the pyrethroids, rotenoids, nicotinoids, and the group that includes strychnine and scilliroside. Pesticides can also be grouped into chemical families such as organochlorines, organophosphates, and carbamates. Organochlorine hydrocarbons (e.g. DDT) could be separated into dichlorodiphenylethanes, cyclodiene compounds, and other related compounds. They operate by disrupting the sodium/potassium balance of the nerve fiber, forcing the nerve to transmit continuously. Their toxicities vary greatly, but they have been phased out because of their persistence. Both operate through inhibiting the enzyme acetylcholinesterase, allowing acetylcholine to transfer nerve impulses indefinitely and causing a variety of symptoms such as weakness or paralysis (Council on Scientific Affairs 1997). Organophosphates are quite toxic to vertebrates, and have in some cases been replaced by less toxic carbamates. Thiocarbamate and dithiocarbamates are subclasses of carbamates. Prominent families of herbicides include pheoxy and benzoic

acid herbicides (e.g. 2,4-D), triazines (e.g. atrazine), ureas (e.g. diuron), and Chloroacetanilides (e.g. alachlor). Phenoxy compounds tend to selectively kill broadleaved weeds rather than grasses. The phenoxy and benzoic acid herbicides function similar to plant growth hormones, and grow cells without normal cell division, crushing the plants nutrient transport system. The phenoxy fatty acid herbicides deserve special mention because of their ability to alter some aspect of plant toxicity and their association with teratogenicity. For example treated plants may build high levels of nitrates or cyanide and many toxic weeds may become more palatable (Osweiler et al. 1985). Nitrate levels are increased in *Amaranthus retroflexus* and the toxic principle increased in treated Sudangrass (*Sorghum halapense*) (Abatan 1992). Triazines interfere with photosynthesis. A number of common fungicides are available at the home for garden use. Accidents and careless use constitute the major hazard to pets and livestock. A new class of fungicides called paldoxins work by taking advantage of natural defense chemicals released by plants called phytoalexins, which the fungi then detoxify using enzymes. The paldoxins inhibit the fungi's detoxification enzymes. They are believed to be safer and greener.



***Amaranthus retroflexus*.** Family Amaranthaceae

**Poisoning from Organophosphate Compounds**

Organophosphate poisoning results from exposure to organophosphates (OPs). Organophosphate poisoning most commonly results from exposure to insecticides or nerve agents. OPs are one of the most common causes of poisoning worldwide, and are frequently intentionally used in suicides in agrarian areas. There are several OP poisonings per year with several hundred thousand resulting in fatalities annually. Organophosphates inhibit AChE, causing OP poisoning by phosphorylating the serine hydroxyl residue on AChE, which inactivates AChE. AChE is critical for nerve function, so the irreversible blockage of this enzyme, which causes acetylcholine accumulation, results in muscle overstimulation. This causes disturbances across the cholinergic synapses and can only be reactivated very slowly, if at all. Paraoxonase (PON1) is a key enzyme involved in OP pesticides and has been found to be critical in determining an organism's sensitivity to OP exposure Kamrin (1997).

The commonly available organophosphorus insecticides include malathion, parathion, diazinon, fenthion, dichlorvos, chlorpyrifos, ethion and nerve gas agents such as soman, sarin, tabun. OP used as ophthalmic agents such as echothiophate, isoflurophate; those used as antihelmintics such as trichlorfon and some in herbicides including tribufos, merphos are tricresyl phosphate.

Exposure to any one of the above listed organophosphates occurs on a daily basis through inhalation, absorption, and ingestion, most commonly of food that has been treated with an organophosphate herbicide or insecticide. Exposure to these chemicals can occur at public buildings, schools, residential areas, and in agricultural areas. The chemicals chlorpyrifos and malathion have been linked to reproductive effects, neurotoxicity, kidney/liver damage, and birth defects. Dichlorvos has also been linked to reproductive effects, neurotoxicity, and kidney/liver damage, as well as being a possible carcinogen.

A number of measurements exist to assess exposure and early biological effects for organophosphate poisoning.

Measurements of OP metabolites in both the blood and urine can be used to determine exposure to organophosphates. Specifically in the blood, metabolites of cholinesterases, such as butyrylcholinesterase (BuChE) activity in plasma, neuropathy target esterase (NTE) in lymphocytes, and of acetylcholinesterase (AChE) activity in red blood cells. Due to both AChE and BuChE being the main targets of organophosphates, their measurement is widely used as an indication of an exposure to an OP. The main restriction on this type of diagnosis is that depending on the OP the degree to which either AChE or BuChE are inhibited differs; therefore, measure of metabolites in blood and urine do not specify for a certain OP. However, for fast initial screening, determining AChE and BuChE activity in the blood are the most widely used procedures for confirming a diagnosis of OP poisoning.

Other procedures used in the diagnosis of OP exposure are the identification and qualitative analysis of nerve agents in the plasma after exposure, and the analysis of protein adducts and the quantitative analysis of decomposition products in by the plasma and urine (FAO 2002)

**Snake Envenomation**

Mr. Vice-Chancellor Sir, another interesting and important area of poisons and poisoning has to do with what is commonly termed Poisonous Snakes - a technical misunderstanding. The term poisonous snake is mostly incorrect; poison is inhaled or ingested, whereas venom is injected. There are, however, two exceptions—*Rhabdophis* sequesters toxins from the toads it eats, and then secretes them from nuchal glands to ward off predators (Hutchinson et al. 2012). The correct term to use is venomous. Normally venom is harmless if ingested but if the venom is injected into some tissue it is toxic and the tissue around the site of injection and other parts of the body will suffer one way or another.

Since livestock have to eat grass, they often encounter snake bites. Dogs and cats are known to physically challenge snakes and in the process are sometimes fatally wounded.

Unfortunately the owner may not know its animal has had an encounter with a venomous snake until it comes down with symptoms.

**Symptoms of a venomous bite**

- Discharge of blood from the wound
- Marks in the skin and swelling at the site of the snake bite
- Severe pain around the bite site
- Diarrhea
- Convulsion of varying severity
- Blurred vision, weakness, dizziness and fainting

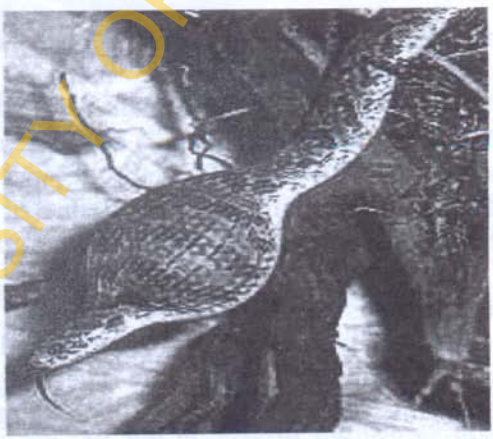
Cobras, vipers, and closely related species use venom to immobilize or kill their prey. The venom is modified saliva, delivered through fangs. The fangs of 'advanced' venomous snakes like viperids and elapids are hollow to inject venom more effectively, while the fangs of rear-fanged snakes such as the boomslang merely have a groove on the posterior edge to channel venom into the wound. Snake venoms are often prey specific, their role in self-defense is secondary. Venom, like all salivary secretions, is a predigestant that initiates the breakdown of food into soluble compounds, facilitating proper digestion. Even non venomous snake bites (like any animal bite) will cause tissue damage. Certain birds, mammals, and other snakes such as kingsnakes that prey on venomous snakes have developed resistance and even immunity to certain venoms. Venomous snakes include three families of snakes, and do not constitute a formal classification group used in taxonomy.

Snake venoms are complex mixtures of proteins, and are stored in poison glands at the back of the head. In all venomous snakes, these glands open through ducts into grooved or hollow teeth in the upper jaw. These proteins can potentially be a mix of neurotoxins (which attack the nervous system), such as fasciculins, dendrotoxins, α-neurotoxins; cytotoxins (phospholipases cardiotoxins, haemotoxins (which attack the circulatory system), bungarotoxins and many other

toxins that affect the body in different ways. Almost all snake venom contains hyaluronidase, an enzyme that ensures rapid diffusion of the venom. Venomous snakes that use hemotoxins usually have the fangs that secrete the venom in the front of their mouths, making it easier for them to inject the venom into their victims. Some snakes that use neurotoxins, such as the mangrove snake, have their fangs located in the back of their mouths, with the fangs curled backwards. *Elapid* snakes, however, such as cobras and kraits are *proteroglyphous*, possessing hollow fangs that cannot be erected toward the front of their mouths and cannot "stab" like a viper; they must actually bite the victim.

It has recently been suggested that all snakes may be venomous to a certain degree, with harmless snakes having weak venom and no fangs. Most snakes currently labeled "nonvenomous" would still be considered harmless according to this theory, as these snakes either lack a delivery method for the venom or are simply incapable of delivering enough to endanger a victim. Venomous snakes are classified in two taxonomic families:

- Elapids – cobras including king cobras, kraits, mambas, sea snakes, and coral snakes



**An Egg eating Snake.**

- Viperids – vipers, rattlesnakes, copperheads/ cottonmouths, adders and bushmasters.

There is a third family containing the *opistoglyphous* (rear-fanged) snakes as well as the majority of other snake species:

- Colubrids – boomslangs, tree snakes, vine snakes, mangrove snakes, although not all colubrids are venomous.

The treatment for snakebite is as variable as the bite itself. The most common and effective method is through antivenom (or antivenin), a serum made from the venom of the snake. Some antivenom is species specific (monovalent) while some is made for use with multiple species in mind (polyvalent).To produce antivenom, a mixture of the venoms of the different species of snakes is injected into the body of a horse in ever-increasing dosages until the horse is immunized. Blood is then extracted from the immunized horse; the serum is separated and further purified and freeze-dried. It is reconstituted with sterile water and becomes antivenom. For this reason, people who are allergic to horses cannot be treated using antivenom..

**Ecological importance of Snakes in general**

Snakes serve an important role as predators in the ecosystems, and snakes are signs of a healthy ecosystem. They help maintain populations of rodents and other prey at a constant and acceptable level. Just think of farmers and the problems they got with rodents. Snakes are actually responsible for keeping crop yields at an acceptable level and preventing spreading of diseases by killing rats. Snakes such as the non-venomous rat snakes are excellent at doing this.

**The Concept of Free Radicals in relation to Poisoning**

The concept of free radicals and oxidative stress have been severally associated with many poisoning conditions (Abatan and Abiola 2001). Free radicals and related species are mainly derived from oxygen (reactive oxygen species/ROS) and nitrogen (reactive nitrogen species/RNS), and are generated in the body by various endogenous systems, exposure to different physicochemical conditions or patho-physiological states. Free radicals can adversely alter lipids, proteins and DNA and have been implicated in aging and a number of diseases. All the biological molecules present in our body are at risk of being attacked by free radicals. Such damaged molecules can impair cell functions and even lead to

cell death eventually resulting in diseased states. Lipids are highly prone to free radical damage resulting in lipid peroxidation that can lead to adverse alterations. Free radical damage to protein can result in loss of enzyme activity. Damage caused to DNA, can result in mutagenesis and carcinogenesis. However, nature has endowed the body with protective antioxidant mechanisms- superoxide dismutase (SOD), catalase, glutathione, glutathione peroxidases and reductase, vitamin E (tocopherols and tocotrienols), vitamin C etc., apart from many dietary components. There are epidemiological evidences correlating higher intake of components/foods with antioxidant abilities to lower incidence of various animal/human morbidities or mortalities (Valko et al. 2007). Current research findings reveal the different potential applications of antioxidant/free radical manipulations in prevention or control of disease. Natural products from dietary components and medicinal plants are known to possess antioxidant activity. Newer and future approaches include gene therapy to produce more anti-oxidants in the body, genetically engineered plant products with higher level of antioxidants, synthetic antioxidant enzymes (SOD mimics), novel biomolecules and the use of functional foods enriched with antioxidants (Stohs 1995).

**Concept of Oxidative Stress**

The relation between free radicals and disease can be explained by the concept of oxidative stress elaborated by Sies (1986). In a normal healthy body, the generation of pro-oxidants in the form of ROS and RNS are effectively kept in check by the various levels of antioxidant defense. However, when it gets exposed to adverse physicochemical, environmental or pathological agents such as atmospheric pollutants, cigarette smoking, ultraviolet rays, radiation, toxic chemicals, over nutrition and advanced glycation end products in diabetes, this delicately maintained balance is shifted in favor of pro-oxidants resulting in 'oxidative stress'. It has been implicated in the etiology of several (>100) diseases of human/animals and in the process of ageing (Valko et al. 2007).

The process of lipid peroxidation, gives rise to many products of toxicological interest like malondialdehyde (MDA), 4-hydroxynonenal (4-HNE) and various 2-alkenals. Isoprostanes are unique products of lipid peroxidation of arachidonic acid and recently tests such as mass spectrometry and ELISA-assay kits are available to detect isoprostanes (Yoshikawa *et al*. 2000).

**The Use of Antioxidants in Poisoning**

An antioxidant is a molecule that inhibits the oxidation of other molecules. Oxidation is a chemical reaction that transfers electrons or hydrogen from a substance to an oxidizing agent. Oxidation reactions can produce free radicals. In turn, these radicals can start chain reactions. When the chain reaction occurs in a cell, it can cause damage or death to the cell. Antioxidants terminate these chain reactions by removing free radical intermediates, and inhibit other oxidation reactions. They do this by being oxidized themselves, so antioxidants are often reducing agents such as thiols, ascorbic acid, or polyphenols (Bjelakovic 2007).

Antioxidants are classified into two broad divisions, depending on whether they are soluble in water (hydrophilic) or in lipids (hydrophobic). In general, water-soluble antioxidants react with oxidants in the cell cytosol and the blood plasma, while lipid-soluble antioxidants protect cell membranes from lipid peroxidation. These compounds may be synthesized in the body or obtained from the diet. The different antioxidants are present at a wide range of concentrations in body fluids and tissues, with some such as glutathione or ubiquinone mostly present within cells, while others such as uric acid are more evenly distributed.

Uric acid is by-far the highest concentration antioxidant in blood. Uric acid's antioxidant activities are also complex, given that it does not react with some oxidants, such as superoxide, but does act against peroxynitrite, peroxides, and hypochlorous acid.

Ascorbic acid or "vitamin C" is a monosaccharide oxidation-reduction (redox) catalyst found in both animals and plants. Most other animals are able to produce this

compound in their bodies and do not require it in their diets. Humans have to obtain vitamin C from their diet (Halliwell 2008).

**Glutathione**

Melatonin is a powerful antioxidant. Melatonin easily crosses cell membranes and the blood–brain barrier. Unlike other antioxidants, melatonin does not undergo redox cycling, which is the ability of a molecule to undergo repeated reduction and oxidation. Redox cycling may allow other antioxidants (such as vitamin C) to act as pro-oxidants and promote free radical formation. Melatonin, once oxidized, cannot be reduced to its former state because it forms several stable end-products upon reacting with free radicals. Therefore, it has been referred to as a terminal (or suicidal) antioxidant.

Tocopherols and tocotrienols (vitamin E) is the collective name for a set of eight related tocopherols and tocotrienols, which are fat-soluble vitamins with antioxidant properties. It has been claimed that the α-tocopherol form is the most important lipid-soluble antioxidant, and that it protects membranes from oxidation by reacting with lipid radicals produced in the lipid peroxidation chain reaction. This removes the free radical intermediates and prevents the propagation reaction from continuing (Atkinson et al, 2008; Hirst et al. 2008).

**Sources of Antioxidants, Phytonutrients and Functional Foods**

Natural compounds, especially derived from dietary sources provide a large number of antioxidants. Some beverages such as tea are also rich sources of antioxidants. A growing body of evidence suggests that moderate consumption of tea may protect against several forms of cancer, cardiovascular diseases, and the formation of kidney stones, bacterial infections, and dental cavities. Tea is particularly rich in catechins, of which epigallocatechin gallate is the most abundant (Valko et al. 2007).

**Importance of Phytonutrients and Functional Foods**

The idea of growing crops for health rather than for food or fiber is slowly changing plant biotechnology and medicine (Soetan and Abatan 2008) responsible for launching a new generation of botanical therapeutics that include plant-derived pharmaceuticals, multicomponent botanical drugs, dietary supplements, functional foods and plant-produced recombinant proteins. Among polyphenols, flavonoids constitute one of the most important single groups, including more than 5000 compounds that have been thus far identified.

The very concept of food is changing from a past emphasis on health maintenance to the promising use of foods to promote better health to prevent chronic illnesses. 'Functional foods' are those that provide more than simple nutrition; they supply additional physiological benefit to the consumer. Because dietary habits are specific to populations and vary widely, it is necessary to study the disease-preventive potential of functional micronutrients in the regional diets. Medicinal plants with increased levels of essential vitamins and nutrients (e.g. vitamin E, lycopene, vitamin C, bioflavonoids, thioredoxin etc.) provide a rich source of compounds like antioxidants that can be used in functional foods (Demmig-Adams, 2002; Block et al. 2007). In this respect it worthwhile to mention the ameliorative role of natural antioxidant from *Garcinia kola*, tocopherol and ascorbic acid in some poisoning cases (Abatan and Abiola 2001; Ambali and Ayo 2011)

People who eat fruits and vegetables have a lower risk of heart disease and some neurological diseases, and there is evidence that some types of vegetables, and fruits in general, may lower risk against some cancers. Since fruits and vegetables happen to be good sources of nutrients and phytochemicals, this suggested that antioxidant compounds might lower risk against several diseases (Finkel et al. 2000; Block et al. 2008).

**Foods and Antioxidant Composition**

| Antioxidant compounds | Foods containing high levels of these antioxidants |
|---|---|
| Vitamin C (ascorbic acid) | Fresh Fruits and vegetables |
| Vitamin E (tocopherols, tocotrienols) | Vegetable oils |
| Polyphenolic antioxidants (resveratrol, flavonoids) | Tea, coffee, soy, fruit, olive oil, chocolate, cinnamon, oregano |
| Carotenoids (lycopene, carotenes, lutein) | Fruit, vegetables and eggs |

Adding more fruit and vegetables of any kind to your diet will improve your health. But some foods are higher in antioxidants than others.

Beta-carotene and other carotenoids: Apricots, carrots, corn, green peppers, kale, mangoes, turnip and collard greens, nectarines, peaches, pink grapefruit, pumpkin, squash, spinach, sweet potato, tangerines, tomatoes, and watermelon

Vitamin C: Berries, broccoli, Brussels sprouts, cantaloupe, cauliflower, grapefruit, mangoes, nectarines, orange, papaya, red, green or yellow peppers, peas, sweet potato, strawberries, and tomatoes

Vitamin E: Broccoli, carrots, mustard and turnip greens, mangoes, nuts, papaya, pumpkin, red peppers, spinach, and sunflower seeds (Halliwell 2008)

**The Concept of Drug Overdose**

The term drug overdose (or simply overdose) describes the ingestion or application of a drug or other substance in quantities greater than are recommended or generally practiced. An overdose may result in a toxic state or death. The word "overdose" implies that there is a common safe dosage and usage for the drug; therefore, the term is commonly only applied to drugs, not poisons, though even certain poisons are harmless at a low enough dosage.

A common unintentional overdose in young children involves multi-vitamins containing iron. Iron is a component of the hemoglobinmolecule in blood, used to transport

oxygen to living cells. When taken in small amounts, iron allows the body to replenish hemoglobin, but in large amounts it causes severe pH imbalances in the body. If this overdose is not treated with chelation therapy, it can lead to death or permanent coma.

The term 'overdose' is often misused as a descriptor for adverse drug reactions or negative drug interactions due to mixing multiple drugs simultaneously.

Signs and symptoms of an overdose vary depending on the drug or toxin exposure. The symptoms can often be divided into differing toxidromes. This can help one determine what class of drug or toxin is causing the difficulties.

**Diagnosis of Poisoning**

Poisoning is a common occurrence in clinical practice but diagnosis invariably becomes a problem because of many diseases or clinical conditions may present with similar clinical symptoms. Often as is the case, the Toxicologist or clinician tackles management of poisoning symptomatically. Poisoning should be suspected in all cases of sudden, severe, and unexpected illness. Investigation should be made in all such situations by ascertaining, as quickly and thoroughly as possible, the answers to the following questions. What are the signs and symptoms of the illness? What was happening before the illness occurred? One has to remember there could have been chronic exposure over time with the signs and symptoms just becoming apparent. What substances were in use? Could more than one substance have been involved? Is there a container of the suspected substance? If so how much was there initially and how much is there now? What is the duration of the exposure? When did it happen? What is the location of the bite or injury if applicable?

**Conclusions and Recommendations**

Mr. Vice-Chancellor, Sir, I have in this lecture articulated the fact that Poisons and poisoning are common phenomenon in our Society. The capacity of the Toxicologist or the Clinician to adequately manage of these conditions are limited by the inadequacy of diagnostic tools. As a result of this, reliance on

symptomatic treatment of poisoning cases becomes inevitable. This is associated with its grievous consequences because there are many disease conditions which may present with similar clinical sign. A poisoning case may therefore turn nasty if not properly tackled.

Having a global look at the situation in Nigeria, I humbly make the following recommendation:

1. Need to set up a poison control centre in all the States of the Federation.
2. A poison control center is a medical facility that is able to provide immediate, free, and expert treatment advice and assistance over the telephone in case of exposure to poisonous or hazardous substances. Poison control centers answer questions about potential poisons in addition to providing treatment management advice about household products, medicines, pesticides, plants, bites and stings, food poisoning, and fumes. Many of the poison exposure cases can be managed simply by phone, greatly reducing the need for costly emergency room and doctor visits.
3. Emergency response Centres with appropriate facilities.
4. The Committee of Vice-Chancellors should see ASUU as a partner in progress and should desist from their wicked effort at breaking union legitimate industrial actions.
5. The issue of contributions to ASUU purse should be non-negotiable because those who claim not be part of ASUU enjoy the sweat and blood of people risking their life in industrial actions

**Acknowledgements**

To the Almighty God, the Most High who knows the end from the beginning and to His only begotten son Jesus Christ be all the glory, honour, praises and worship who has given me the grace to deliver this lecture. My posthumous thanks to my parents Chief Joseph Oladimeji Abatan and Madam Comfort Adeoti for been the instruments God used to make

me see this life. I give credence to all the trail blazers who have given their inaugural lecture before now and most especially Professor Olu-Owolabi, the Dean of Arts the tireless and strong believer in ASUU who reminds us of the Biblical saying that whatever your hands find to do, do it well. May the gracious Lord accord his soul eternal rest.I acknowledge Professors Olowookorun (Physiology), Reuben Arowolo (Pharmacology), my Supervisor at the Ph. D level, and Johnson Oyewale (Physiology) who have given their inaugural lectures from the department. I appreciate all members of staff of the Faculty of Veterinary Medicine and especially of the Department of Veterinary Physiology, Biochemistry and Pharmacology for their immense contributions to my life. My special thanks goes to my contemporaries in the ASUU struggle who have sacrificed all their time and resources in ensuring that Nigerian education systems is where it is today. If they don't appreciate you be rest assured that the God of the Heaven and Earth see your work of devotion and sacrifices and will reward all according to the work of our hands. My special thanks to all my M. Sc and Ph.D students such as Drs Adeolu Adedapo, Oladele, Femi Olukunle and Ojezele. I on behalf of my family members appreciate the role of Prof and Prof. Mrs J' Bayo Adekanye. My special appreciation goes to my wife whom God used in giving me our children, Dr Adeola Abatan, Dr Tola Ajayi, Lawyer Oluwatosin Abatan, Engineer Oluwole Abatan Jnr, Ifeoluwa Abatan and Olufemi Abatan. Finally to this wonderful audience I say thank you. May the Lord God bless you all richly.

**References**

Abatan, M. O. and Fajinmi, J. L. (1994) Studies on some toxic effects of the plant Dichapetalum madagascasiense Poir in rats. Trop. Vet. 12: 1-10

Abatan, M. O., Arowolo, R. O. A., and Olorunsogo, O. O. (1996) Pathological effects of Lantana camara and Dichapetalum madagascasiense in goats. Trop. Vet. 14, 127-132.

Abatan, M. O., Arowolo, R. O. A., and Olorunsogo, O. O. (1997) Phytochemical analysis of some commonly occurring poisonous plants in Nigerian pastures. Trop. Vet. 15: 49-54.

Abatan, M. O. (2000) The toxic effects of some poisonous plants on the hematological parameters of rats. Trop. Ani. Prod. Invest. 3: 99-106

Abatan, M. O. an Abiola Abosede (2001) The effect of ascorbic acid, tocopherol, and Garcinia kola extract on the toxic effects of paraquat and 2,4-D in rats. Trop. Ani. Prod. Invest. 4: 21-28

Abatan, M. O., lateef, I. and Taiwo, V.O. (2006) Toxic effects of Non-Steroidal Antiinflammatory agents in rats. African J Biomed Res. 9: 216-223.

Adedapo, A. A. and Abatan M. O. (2006) Determination of the3 lethal dose 50 (LD50) from aqueous extracts of the leaves of P. amarus and E. hirta in rats. Folia Vet. 50(4), 189-191.

Albresten J. et al. (1998) Treatment of bone marrow suppression following toxic ingestion of 5-fluorouracil in dogs (Abst) J. Vet. Intern. Med. 12: 240.

Ambali, S. F. and Ayo, J. O. (2012) Vitamin C attenuates chronic chlorpyrifos-induced alteration of neurobehavioral parameters of Wistar rats. Tox. Intern. 19(2): 144-152.

Atkinson, Jeffrey; Epand, Raquel F.; Epand, Richard M. (2008) "Tocopherols and tocotrienols in membranes: A critical review". *Free Radical Biology and Medicine*44 (5): 739–64.

Bastianello SS, Nesbit JW, Williams MC, Lange AL (December 1987) "Pathological findings in a natural outbreak of aflatoxicosis in dogs". *Onderstepoort J. Vet. Res.*54 (4): 635–40.

Block KI, Koch AC, Mead MN, Tothy PK, Newman RA, Gyllenhaal C (2008) "Impact of antioxidant supplementation on chemotherapeutic toxicity: a systematic review of the evidence from randomized controlled trials". *Int. J. Cancer*123 (6): 1227–39.

Block KI, Koch AC, Mead MN, Tothy PK, Newman RA, Gyllenhaal C. (2007) "Impact of antioxidant supplementation on chemotherapeutic efficacy: a systematic review of the evidence from randomized controlled trials". *Cancer Treat. Rev.*33 (5): 407–18.

Bingham AK, Phillips TD, Bauer JE (March 2003) "Potential for dietary protection against the effects of aflatoxins in animals". *J. Am. Vet. Med. Assoc.*222 (5): 591–6.

Bjelakovic G; Nikolova, D; Gluud, LL; Simonetti, RG; Gluud, C (2007) "Mortality in randomized trials of antioxidant supplements for primary and secondary prevention: systematic review and meta-analysis". *JAMA*297 (8): 842–57.

Cowan, S. M. and Bunch, S. E. (2001) Oral antidiabetic drugs in cats. Small Ani/Exotics 23(7). Pp 633-642.

Coelho, F. and Birks, J. (2001) Physostigmine for Alzheimer's disease. Cochrane Database Syst. Rev. 2 (Abstr)

Chyka, PA; Erdman; Christianson; Wax; Booze; Manoguerra; Caravati; Nelson et al. (2007) "Salicylate poisoning: an evidence-based consensus guideline for out-of-hospital management". *Clinical toxicology* 45 (2): 95–131.

Council on Scientific Affairs, American Medical Association. (1997) Educational and Informational Strategies to Reduce Pesticide Risks. Preventive Medicine, Volume 26, Number 2

Demmig-Adams, B.; Adams Ww, 3rd (2002). "Antioxidants in Photosynthesis and Human Nutrition". *Science*298 (5601): 2149–53.

Dorman, D. C. et al (1990) 5-fluorouracil toxicosis in dogs. J. Vet. Intern. Med. 4 (5) 254-251

Finkel, Toren; Holbrook, Nikki J. (2000) "Oxidants, oxidative stress and the biology of ageing". *Nature*408 (6809): 239–47.

Food and Agriculture Organization of the United Nations (2002) International Code of Conduct on the Distribution and Use of Pesticides. Retrieved on 2007-10-25.

Finkel, Toren; Holbrook, Nikki J. (2000) "Oxidants, oxidative stress and the biology of ageing". *Nature*408 (6809): 239–47.

Gauthaman K, Adaikan PG, Prasad RN. (2002) "Aphrodisiac properties of Tribulus Terrestris extract (Protodioscin) in normal and castrated rats". *Life Sciences* 71(12): 1385–96.

Gauthaman K, Ganesan AP (Jan 2008) "The hormonal effects of *Tribulus terrestris* and its role in the management of male erectile dysfunction—an evaluation using primates, rabbit and rat". *Phytomedicine* 15 (1-2): 44–54.

Hirst, Judy; King, Martin S.; Pryde, Kenneth R. (2008) "The production of reactive oxygen species by complex I". *Biochemical Society Transactions*36 (5): 976–80.

Halliwell, B (2008) "Are polyphenols antioxidants or pro-oxidants? What do we learn from cell culture and in vivo studies?". *Archives of Biochemistry and Biophysics*476 (2): 107–112.

Hignet K. The curious case of the Poisoned umbrella. The murder of Georgi Markov. The vieweastwordpress.com. Retrieved October 2012.

Hutchinson. DA, Savitzky. AH, Mori, A, Burghardt. GM, Meinwald, J, and Schroeder, FC. (2012) Chemical investigations of defensive steroid sequestration by the Asian snake Rhabdophis tigrinus. Chemoecology 22(3): 199-206.

Kamrin MA. (1997). *Pesticide Profiles: toxicity, environmental impact, and fate.* CRC Press.

Leong, Y. -H.; Ismail, N.; Latiff, A. A.; Manaf, N. A.; Rosma, A. (1 January 2011) "Determination of aflatoxins in commercial nuts and nut products using liquid chromatography tandem mass spectrometry". *World Mycotoxin Journal*4 (2): 119–127.

Li, Feng-Qin; Li, Yu-Wei; Wang, Ye-Ru; Luo, Xue-Yun (13 May 2009) "Natural Occurrence of Aflatoxins in Chinese Peanut Butter and Sesame Paste". *Journal of Agricultural and Food Chemistry*57 (9): 3519–3524.

Mahoney, Noreen; Russell J. Molyneux (2010 April 14). "A Rapid Analytical Method for Determination of Aflatoxins in Plant-Derived Dietary Supplement and Cosmetic Oils". *J Agric Food Chem.*58 (7): 4065–4070.

Merck Veterinary Manual: Nonsteroidal Antiinflammatory drugs. www. Merckvetmanual.com. Retrieved October 2012

Ogen, S.O.. Ross, S.B., Hall, H., Holm, A.C. Renyl A.L. (1981) The Pharmacology of zimedine: a 5-HT selective reuptake inhibitor. Acta Psychiatr Scan suppl 290: 127-51

Osuntokun, B. O. (1973) Ataxia, neuropathy associated with high cassava diet in West Africa. Proceeding of an Interdisceplinary workshop. London, England pp. 29-30.

Pet Poison Helpline (2012) Methyphenedate. *www.petpoisonhelpline.com*. Retrieve 2012

Stohs, S; Bagchi. D (1995). "Oxidative mechanisms in the toxicity of metal ions". *Free Radical Biology and Medicine*18 (2): 321–36.

Temple, A. R (February 1981) "Acute and chronic effects of aspirin toxicity and their treatment". *Archives of Internal Medicine* 141 (3 Spec No): 364–9.

Vertuani, Silvia; Angusti, Angela; Manfredini, Stefano (2004) "The Antioxidants and Pro-Antioxidants Network: An Overview". *Current Pharmaceutical Design*10 (14): 1677–94.

Villar D. Knight, M. K., holding, J., Barrnet. G. H.. and Buck. W. B. (1995) Treatment of acute isoniazid overdose in dogs. Vet. Hum. Toxicol. 37(5) 473-7.

Valko, M; Leibfritz, D; Moncol, J; Cronin, M; Mazur, M; Telser, J (2007) "Free radicals and antioxidants in normal physiological functions and human disease". *The International Journal of Biochemistry & Cell Biology*39 (1): 44–84.

Williams JH, Phillips TD, Jolly PE, Stiles JK, Jolly CM, Aggarwal D. *Human aflatoxicosis in developing countries: a review of toxicology, exposure, potential health consequences, and interventions.* Am J Clin Nutr 2004;80:1106-22.