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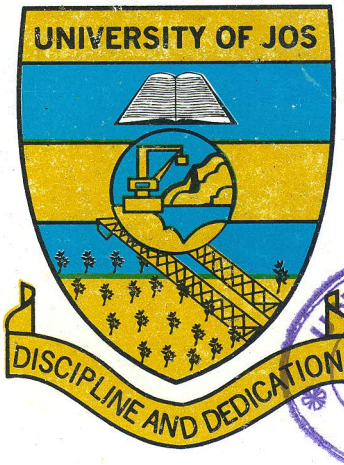

FOOD BORNE CHEMICAL POISONS: NOT BY ENEMY ALONE

INAUGURAL LECTURE

Delivered at the University of Jos
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By

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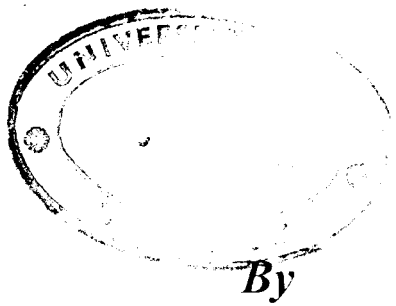

By

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INTRODUCTION

From time immemorial, food has been recognized as a vector of illness, both willful and accidental. Our forebears did recognize that certain foodstuffs are poisonous if not properly processed or certain non-edible parts are not effectively removed during food preparation. However, when foodborne illness (food poisoning) resulted from consumption of food prepared and/or served by a third party, the first impulse was (and is) to suspect mischief. This probably informed the practice in many Nigerian cultures whereby a host is required to assure his guest of the safety of the food and drinks set before him (e.g. by eating or drinking from the portion served his guest and/or performing other traditional safety assurance rituals). The oral traditional medical sociological evidence backing such a practice seems so enormous that guests from certain communities would not eat or drink unless such a ritual was performed by his host.

As could be gleaned from the title of this lecture, I am not here to dispute or provide scientific evidence in support of that cultural practice. It just happens that the work that earned me the professorship in 1990 pertains to the food poisons, in particular food poisons that are not willfully introduced into foodstuffs. My special area of interest which, in keeping with academic tradition on Inaugural Lectures, is the premise of this lecture, centres on chemical substances in foodstuffs responsible for food borne illness; the underlying biochemical mechanisms mediating their observed adverse effects; and factors, including nutrition and food additives, affecting the development of induced lesions, all for the ultimate purpose of devicing appropriate biochemical tools for the prevention, management (treatment) and control of the toxin-induced food borne illness. It was in the course of those studies that one came to the inescapable conclusion that food and drinks have enough poisonous chemicals of their own, some naturally occurring, some inadvertently introduced, and some willfully introduced for good cause.

FOOD POISONS AND FOOD BORNE ILLNESS: Definitions

The term 'poison' is used here in a loose sense to mean chemical substances whose presence in food may elicit adverse reactions that may culminate in frank clinic symptoms of illness. Some are not poison by the strict scientific definition but may behave as one under certain conditions. In this class are some essential nutrients. Whatever their intrinsic toxicity potential, when these poisons act, they induce clinical symptoms which range from the barely noticeable to the profound: irritation of the upper palate, tingling of the ears, twitching of the eyelids, browning of the eyes and teeth, through diarrhoea and vomiting, superficial skin rashes, to full blown disease conditions like asthma and cancer. Their manifestation may be dramatic or insidious, taking months or years to become evident.

Illness induced by food borne chemicals fall into two main broad groups, *food toxicity* and *food sensitivity*, based on the biochemical mode of induction, consumer susceptibility

characteristics; and intrinsic (natural) toxicity potential of the causative chemical agent. Food toxicity can be defined as: *adverse reactions to food caused by the presence in the food of inherently (naturally) poisonous or toxic chemical substances of either endogenous or exogenous origin.* The ingested food is unwholesome to the extent that it contains those naturally toxic chemicals. The typical food toxicity illness afflicts every one who eats enough of the unwholesome food. The clinical manifestation of food toxicity may be *acute* (i.e. short and severe) or *chronic* (following prolonged exposure, long incubation). By and large, the major clinical symptoms or illnesses manifested by those afflicted by chemical food toxicity can be grouped into four classes (Table 1).

Table 1. Major types of clinical manifestations of food toxicity.

Clinical manifestation	Nature	Examples
Food poisoning*	Acute	**Lead, mushroom, cassava, oil bean seed, etc. poisoning.**
Cytotoxic disorders		
Neoplastic lesions	Chronic	Cancer, foetal malformations.
Cell lysing lesions	Acute or chronic	Hepatitits, liver cirrhosis, haemolytic anaemia.
Metabolic disorders	Acute or chronic	Cardiac beri beri, porphyria, nephropathy, nervous disorders, blood coagulation disorders.
Nutrient assimilation disorders	Chronic	Secondary nutrient deficiency diseases such as goitre, iron deficiency anaemia.

* *Defined as an acute illness which usually includes one or more gastrointestinal symptoms like vomiting and diarrhoea, resulting from the recent consumption of food or drink contaminated with chemical poison, preformed bacterial poison, or live bacteria; poisonous natural vegetation like berries, mushroom (fungi), oil bean seed* (Adapted from N. Roper, Livingstone's Pocket Medical Dictionary, and A. Stewart Truswell, ABC of Nutrition, BMA).

Food sensitivity, on the other hand, has been defined as: *A reproducible unpleasant reaction to a specific food or ingredient which one gets but others don't"* (Stewart Truswell, 1990). In other words, food sensitivity (also called *food intolerance*) is a condition in which an otherwise wholesome food or food item provokes adverse reactions in some of the people who ate it – that is, unlike food toxicity, not everyone who eats it gets afflicted. Food sensitivity is a rare occurrence and when it does occur, only an insignificant fraction is affected. Another distinguishing feature from food toxicity is that the distinct clinical symptoms developed by the victim disappears without trace, within a few hours of manifestation but are reproduced when the individual is exposed to the causative food or food item again. Clinical symptoms of food sensitivity are well

documented and the interval between food challenge and manifestation is fairly well known in most cases (Fig.1).

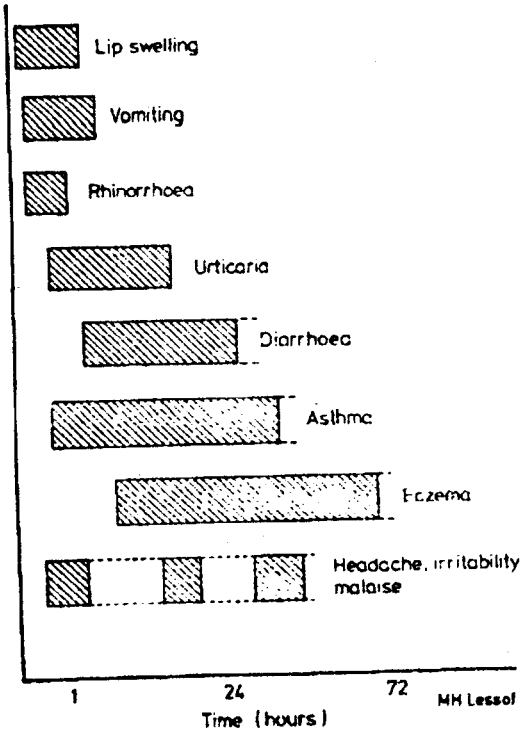


Fig 1. Symptoms of food sensitivity and their time course (Source: M.H. Lessol, 1983).

The only clinical symptoms that need explaining here are *rhinorrhoea* and *urticaria/angio-oedema*. Urticaria or *nettle rash*, in layman's language, is a skin eruption characterized by multiple circumscribed, smooth, raised itchy weals or superficial blisters. For easier comprehension, it is the type of superficial blisters that result from bee or wasp sting. Angio-oedema is a severe form of urticaria; the rashes are larger and more extensive and may involve mucous membrane of the mouth and throat. Rhinorrhoea (Greek, *rhis* = nose; *rheem* = to flow) simply means nasal discharge or "running nose" in layman's parlance. When clinical manifestation is initiated by the immune system, food sensitivity is termed *food allergy*. Now what are these food poisons that cause these illnesses and how do they get into food?

NATURE AND SOURCES OF CHEMICAL POISONS IN FOOD

Chemical substances that have been implicated as food poisons (and pseudo food poisons) can be categorized into four classes, namely,

* inherently toxic natural constituents of foodstuffs;
* other natural constituents of foodstuffs, including nutrients like proteins, carbohydrates;
* chemical contaminants; and
* intentional food additives.

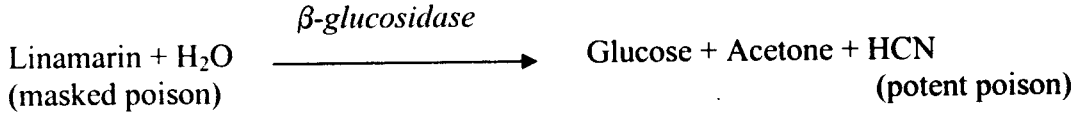
Within each class are several, in the case of contaminants and food additives, several hundreds of, different chemicals of diverse potency.

Toxic Natural Food Components

No raw food commodity, whether of plant or animal origin, is devoid of harmful natural chemical constituents. It is precisely because of this fact that we process/prepare and cook our food. The toxic components are an integral part of the normal life chemistry of the plant or animal source of the foodstuff. Most are end products or intermediate products of normal life chemistry or metabolism (e.g. tartaric acid, oxalic acid) but in many cases, they are chemicals specially elaborated by the plant or animal as a protection for its food reserves (e.g. antitrypsin factors protecting reserve proteins in beans) or as chemical defences against predators, pests, and parasites (e.g. cyanogenic glucosides in cassava root cortex protecting against soil beetles). Naturally, those specially elaborated and targeted at third parties, which includes humans, can be highly potent poisons.

Normally, the endangered plant or animal food sources are protected from the poisons they elaborate; nature endowed them with the necessary chemical machineries to package the poisons in a way that they do not harm them. For example, nature protects cyanide elaborating plant crops by masking the potent poison, cyanide, with glucose molecules and an accessory chemical to form relatively harmless cyanogenic glucosides. Glucose is also employed to mask other potent poisons besides cyanide. It is the "third party" organism, man included, that, through its meddlesomeness, unwittingly unmasks the potent poison. By disrupting the integrity of the foodstuff tissue cells, the pest or parasite facilitates the release of the enzyme, hitherto trapped in a separate compartment, which unmasks the poison.

Let us illustrate with *linamarin* (the cyanogenic glucoside of cassava) and *glucobrassicin* (the β-thioglucoside of cabbage):



$$\text{Glucobrassicin} + H_2O \xrightarrow{\beta\text{-thioglucosidase}} \text{Glucose} + \text{Goitrin}$$

(masked poison) (potent poison)

Food preparation/processing procedures, such as, grinding, grating, cutting and fermentation, as well as chewing, which disrupt the integrity of foodstuff tissues serve to release the entrapped enzymes and facilitate the release of the ultimate poison. Generally, the unmasked poison is present in the processing/preparation water or cooking water, as the case may be, which is usually discarded. (Those of us who grew up in rural communities and who have dealt with bitter cassava, red beans or oil bean seed know how lethal their fermentation or cooking water can be to domestic livestock). However, masked poisons which escape unmasking during food preparation/processing may eventually get unmasked right inside the gut lumen by similar enzymes present in gut bacteria. Poisons released in the body in such manner have been implicated in many reported cases of food poisoning resulting from eating inadequately prepared natural plant foods like oil bean seed salad, "SAP" gari, etc. Processed food products invariably contain residues of toxic natural constituents, the level varying with the efficiency of the processing procedure. Such residues have been known to cause chronic food toxicity symptoms of epidemic proportions (e.g. Table 2). However, in many instances, the toxicant is so entrenched in the foodstuff matrix that traditional preparation procedures do not substantially reduce its level (e.g. phytic acid in cereals). Toxic components in foodstuff may result from abnormal conditions of plant or animal used for food e.g. honey from bees feeding on rhododendron nectar (Stewart Truswell, 1990).

Table 2. Examples of food toxicity endemias reportedly due to chronic exposure to cyanide residues in processed cassava products.

Disease endemia	Population	References
Nutritional ataxic neuropathy	W. Nigeria; Tanzania	Oshuntokun (1968, 1981); et al (1968). Makene & Wilson (1972).
Acuted spastic paraparesis* (also a nutritional neuropathy)	Mozambique	MoH, Mozambique (1984); Cliff *et al* (1985).
Iodine deficiency disorders** (goitre, cretinism)	D.R. Congo Nigeria	Gaitan (1986) UNICEF Benchmark Survey 1993.

** The epidemic occurred in a drought-stricken area of Mozambique, during the cassava harvest of 1981 when cassava varieties with very high cyanogens content were the only food available and the usual traditional processing procedures could not be followed.*

*** The body detoxifies cyanide by converting it to thiocyanate which, incidentally, is an iodine assimilation antagonist.*

Deficiency of sulphur-containing amino acids required for detoxification of cyanide was implicated as a major aetiological factor in all three cases of nutritional neuropathy outbreak.

Chemical Food Contaminants

There are five major sources of potentially toxic chemicals in human foods and drinks. These are,

* Food utensils and packaging materials
* Domestic water
* Agrochemicals
* Industrial chemical wastes
* Food spoilage microbes

These sources between them account for the bulk of the incidence of food borne illness due to chemicals.

Food utensils and packaging materials. Many of the chemical materials used in making food utensils and packaging materials remain mobile and migrate into food or water when the chemical environment of the food is ideal. For instance, high-acid foods corrode certain metal utensils leading to the introduction of the toxic metals into the food. Overwhelming epidemiological evidence from countries where food poisoning is a statutorily notifiable disease, has revealed that such migration of utensil materials into food is responsible for many cases of food borne illness in humans. Consequently, the use of metals and alloys in food utensils is now statutorily regulated and controlled. There is in place globally (i.e. relevant UN agencies – WHO, FAO, etc) and in America and Asia, guidelines on the use of metals and alloys in manufacture of food utensils and food packaging materials. The guidelines are formulated and issued based on observed adverse health implications of metal used in food contact materials and expert reviews of available data on same. As an illustration of the fact that concern for public health is the bottom line of these guidelines, let me quote from just two of such guidelines, namely, the Council of Europe Guidelines on Metals and Alloys Used as Food Contact Materials issued on 09.03.2001 (CE, 2001) and Alberta, Canada, Health and Wellness Food Retail and Food Services Code for Health Professionals issued in October, 2003 (Alberta Government, 2003).

"... *4.1 Health Aspects*

In compliance with Article 2 of Directive 89/109/CEE, metallic materials under normal and foreseeable conditions, should meet the following conditions:

They should be manufactured in accordance with good manufacturing practice and they should not transfer their constituents to foodstuffs in quantities which could:

* *endanger human health*

* *bring about unacceptable change in the composition of the foodstuffs or deterioration in the organoleptic characteristics thereof..." (CE, 2001).*

"... 3.6 Packaging.

... Rationale

...Chemical contamination can occur during cooking or storage when certain metals contact high-acid foods. Potentially toxic metals include lead, copper, brass, zinc coating, antimony and cadmium. Some foods that have been involved in metal poisoning are sauerkraut, tomatoes, fruit gelatins, lemonade, fruit punches, and carbonated beverages..." (Alberta Government, 2003).

The Government of Alberta, Canada, went ahead, to recommend that "high-acid foods (pH below 4.6) must not be stored, or cooked in containers coated with, made of, or containing" the above listed metals, including cast iron and "enamelware which may chip and expose the underlying metal." The Council of Europe's list of metals and alloys not recommended for use as food contact materials is essentially the same, save for minor variations in conditions and details. By and large, regional and national guidelines are made with the benefit of technical advice provided by the Joint FAO/WHO Expert Committee on Food Additives (JECFA).

One would be tempted to wonder why iron, zinc, and copper, which are essential mineral nutrients are included in the prohibition list, albeit under certain conditions. The point is that our body requires them in relatively minute quantities and their consumption in amounts far in excess of these requirements results in toxic adverse reactions. It has been reported, for instance, that ingestion of soluble iron salts by children in doses exceeding 0.5g of iron can give rise to severe lesions in the gastrointestinal tract, followed by metabolic acidosis, shock and toxic hepatitis (Elinder, 1986). Iron overload disorders have also been reported in a South African population brewing traditional beers in cast iron pots. Traditional beers are generally high-acid drinks whose contact with cast iron vessels is prohibited. A study done on indigenous traditional beer (*pito*) consumers on the Jos Plateau (Ubom and Okoye, 1992) suggests a correlation between toxic metal contents of *pito* and incidence of acute liver cirrhosis among the consumers.

A visit to tinker sheds in our urban centres will reveal the extent to which we are unwittingly poisoning ourselves with toxic metals from substandard kitchen metal utensils. When I see badly corroded metal cooking spoons and pots used in some of our local restaurants and homes, I wonder where the leached metals had gone to.

I should mention in passing that clay and allied earthen utensils also contain mobile substances which migrate into food and drinks. A study conducted some years ago on local clay water pots revealed extensive migration of pot chemical materials into water (Okoye, 1988, unpublished).

Domestic Water. Considering the journey of water to the point of collection, domestic water is hardly devoid of chemical contaminants. The nature and profile of chemical contaminants vary with the geochemistry of the rock from which the water ensued and of the terrain over which it flowed to the point of collection. Quite often, the contaminants include chemicals which are not efficiently removed by conventional water treatment procedures (that is where water is treated) e.g. 3,4-benzpyrene, one of the most potent carcinogenic polycyclic aromatic hydrocarbons (PAHs) which occur naturally in coal, soils, ground water and surface water. PAHs and other organic chemical pollutants of domestic water like phenolic compounds, have been implicated as aetiological factors in iodine deficiency disorders (Gaitan, 1986).

Many toxic chemical contaminants of domestic water are not coloured neither do they have taste. As a result, they cannot be detected by those properties. That is to say that clean or colourless water does not necessarily mean 'pure' or safe. Neither does borehole source. Only thorough chemical analysis determines what is pure or otherwise.

Agrochemicals. Hundreds of chemical poisons found in foodstuffs are traceable to chemicals used in modern agriculture and livestock farming. Most of the agrochemicals employed in growing plant crops (as pesticides, herbicides, fertilizers, etc) persist in the food chain, with many being bioaccumulated by the plant food crop and carried over to the edible foodstuff. Those applied in post harvest handling of crops (storage, processing, etc) also persist in the associated foodstuff. Veterinary drugs may also persist and livestock feed chemical additives are invariably carried over into the edible products (e.g. eggs, meat, milk). Being man made, many of the agrochemicals are incompatible with normal life chemistry and, consequently, are toxic to human tissues. Exposure to excessive levels of residues of agrochemicals in foodstuffs and feedstuffs have been associated with illness. Indeed food poisoning due to agrochemicals, especially pesticides (Jeyaratnam, 1990), is generally recognized as a major health problem, which explains why the levels of agrochemical residues in food commodities are under strict statutory control, globally and nationally. The epidemiological evidence associating residues of agrochemical in foodstuff with human disease is so strong that in the early 1980s, the US refused to allow in a meat import from Mexico on account of the level of the residue of a regulated agrochemical (pesticide) being far in excess of the statutory ceiling, regardless of the fact that US exported the pesticide in question to Mexico. Monitoring of agrochemical contents of food commodities is done routinely at ports of entry of most advanced countries. The use of agrochemicals in post harvest management of foodstuffs have resulted in major food poisoning epidemics in certain human populations (Table 3).

Table 3. Examples of food poisoning epidemics due to post harvest use of agrochemicals.

Outbreak of	Causative agrochemical	Circumstance
Acquired porphyria*	Hexachlorobenzene, a fungicide	Turkey 1955-59; victims numbering ca.3,000 consumed HCB-treated wheat meant for planting.
Methylmercuric chloride poisoning	Alkylmercury fungicide	Iraq, 1972. Victims (ca.6530) consumed alkylmercury-treated wheat meant for planting.
Arsenic poisoning**	Arsenic-containing fungicides	Victims consumed fruits, vegetables treated with arsenic-containing fungicide.

* *Called porphyria cutenea tarda symptomatica.*

** *Characterized by dizziness, loss of appetite, vomiting and death. Poisoning minimized by adequate washing of treated fruits.*

There are many unreported such incidents in Nigeria occurring especially in the periods just before planting when farmers run short of food reserves and eat or sell treated seedlings for cash as foodstuff. We have purchased such treated beans seedlings on two or three occasions in the past.

Industrial chemical wastes. The incidence of health hazards due to environmental chemicals from industrial activities was such that the United Nations Environment Programme (UNEP) in the late 1970's established the International Register of Potentially Toxic Chemicals (IRPTC) as an information network for informed decision-making in the area of chemical hazards control. Many of the subsequently listed environmental chemicals cause health hazards via the human food chain. Foodstuffs become contaminated by industrial waste chemicals when plant crops are grown on soils polluted by such chemicals or in farms irrigated with industrial effluent; livestock are reared on pastures polluted by industrial wastes; or fish and aquatic foodstuffs harvested from inland and coastal waters into which industrial effluents are discharged. In many instances, the plants, fish, or livestock, grown in such a polluted environment lack the metabolic machinery to degrade and/or excrete some of the industrial chemical wastes (e.g. lead, mercury and cadmium compounds) they take in with feed or water. As a result, they progressively store away (or bioaccumulate) them in tissues where they would not seriously interfere with normal life chemistry.

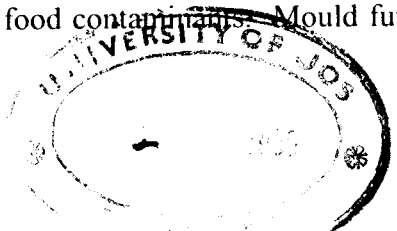
Many of those unmetabolizable chemicals "disposal sites" happen to be in the edible parts of the food crop, thereby constituting a health hazard to human consumers. Consumption of foodstuffs with a heavy loads of bioaccumulated toxic industrial chemicals have indeed be implicated in cases of human food poisoning, the first major outbreaks to be described being *itai itai disease* and *Minimata disease* outbreaks both of which occurred in Japan. The former, a clinical manifestation of cadmium poisoning, afflicted a population consuming fish and other marine foods harvested from coastal waters into which Cd-containing industrial effluent was being discharged. Minimata disease, a mercury poisoning condition, afflicted a population consuming marine foods from Minimata Bay, Japan, which was heavily contaminated by inorganic mercury compounds discharged by a nearby plastics manufacturing factory. Both were severe debilitating and fatal disease outbreaks afflicting whole communities.

The developed world learnt a major environmental health lesson from the two incidents which, together with the lesson of the Love Canal incident in USA, several years later, has elevated industrial waste disposal question to the global pedestral. The consequent tightening of statutory controls on industrial waste disposal and greater public awareness in advanced countries of the health consequences of industrial waste disposal in their vicinity have lead to exportation of hazardous industrial chemical wastes to hapless Third World Countries, of which Nigeria has "benefited" in the past.

In Nigeria's major industrial centres (Lagos, Port Harcourt, Kaduna, etc), industrial effluents are invariably discharged into coastal waters, creeks and inland waterways. Apart from a transient noise made a few years ago about discharge of industrial effluent into a fishing bay in Lagos, we are yet to learn the lesson of Minimata Bay or Love Canal. We are still in the era of "mysterious deaths" and "mysterious disease epidemics".

Food spoilage microbial toxins. Many microbes which commonly infest foods and foodstuffs produce and deposit poisonous secondary metabolites in their substrate. Invariably, these poisons remain in the foodstuff matrix even after the microbes which elaborated them had died or had been killed by food preparation treatment or antimicrobial agents. Many are heat resistant, remaining potent even after being subjected to cooking temperatures (e.g. toxins produced by food spoilage bacteria *Staphylococcus aureus* and *Bacillus aureus*).

Toxigenic bacteria and mould fungi are responsible for microbial toxins in foods. However, moulds can infect food commodities at much lower moisture content than bacteria and consequently, mould fungal toxins are more ubiquitous contaminants of food commodities than bacterial toxins. By extension, they are the most ubiquitous naturally occurring food contaminants. Mould fungal toxins (called *mycotoxins*) can be produced



in a plant crop in the field, during harvest, post harvest handling and storage. It is produced in meat and fish products during smoking or sun drying.

Well over 200 mycotoxins are known. Many mycotoxins were discovered in the course of epidemidogical investigations into the cause of major disease outbreaks (*mycotoxicosis*) in livestock farms or among domestic animals. At least 23 diseases of man and animals definitely caused by mycotoxins and 25 diseases probably caused by mycotoxins have been documented (Hamilton, 1982). Among them are liver cancer, cardiac beri beri, hepatitis, alimentary toxic aleukia (ATA), Reye's syndromes, hyperoestrogenism, Onyalai syndrome and blood clotting disorders. No less than twelve of the mycotoxins associated with animal and human disease have been detected in Nigerian foodstuffs (Table 4). The indications are that many more are present but have not been detected. Well over 50 of the fungal moulds isolated from infected Nigerian food commodities by different workers have been found to be toxigenic and produce mycotoxins experimentally.

Afltoxin B_1, the most toxic of the aflatoxins and the most potent carcinogen known in animals has been detected in virtually every staple foodstuff of Nigeria, including smoked/dried fish and meat (Okoye, 1992). Aflatoxin - producing strains of *Aspergillus flavus* are ubiquitous food spoilage moulds of the warm humid tropics while *Fusarium* moulds are essentially food spoilage moulds of cooler climates – which explains why virtually all the reported cases of *Fusarium* mycotoxin contamination of Nigerian grains pertain to those grown on the Jos Plateau.

Table 4. Mycotoxins so far detected in Nigerian food commodities.

Mycotoxins	Producing mould fungus	Mycotoxicosis outbreaks incidental to discovery	Established Toxic effects**
Aflatoxins[4]	Aspergillus flavus	Turkey "X" disease in turkey poults in England.	A carcinogen, mutagen, teratogen,
Cytochalsan/tenuazonates	*Phorma sorghina*	Human haemorrhagic disease Onyalai syndrome in South Africa.	anticoagulant, hepatotoxin, etc.
Zearalenone[1,3]	F.graminearum (= *Gibberella zeae*)	Hyperoestrogenism among pigs in USA.	Hyperestrogenic effects.
Ochratoxin A	*Aspergillus ochraceus*	Nephropathy in pigs in the Scandinavia; the Balkans.	Inhibitor of protein synthesis; a nephrotoxin.
Deoxynivalenol[2,3] (= vomitoxin)	*Fusarium graminearum*	"Red mould disease" in man and farm animals in Japan.	Emetic and immunosupressive effects, toxic to haematopoietic tissue.

T-2 toxin[2,3]	*Fusarium sporotrichoides, F. poae; F. tricinctum*	Alimentary toxic aleukia (ATA) in humans in Russia and C. Europe.* "Mouldy corn toxicosis in lactating cows in USA.	Immunosuppresive effects causing depletion of lymphoid tissue.
Moniliformin[3]	*Fusarium moniliforme*	Leukoencephalomalacia in horses and other Equidae in USA.	Potent inhibitor of energy production (mitochondrial ETC)
Nivalenol[3]	*F. graminearum*	As for deoxynivalenol (DON).	As for DON
Fusarenon-X[3]	**F. graminearum**	As for DON	As for DON
HT-2 toxin[3]	**F. graminearum**	As for DON	As for DON

*[1]Detected in guinea corn beer (Okoye, 1986) [2]Detected in acha (Ghodi et al. 1986b) [3]Detected in maize grains (Okoye, 1991, 1993; Ghodi et al. 1986a-ZEN) – all on the Jos Plateau [4]Detected in virtually all foods crops nationwide. *During World War II*

***Toxicity associated with Fusarium mycotoxins includes skin irritation, poor appetite, headache, vomiting, gastrointestinal irritation, haemorrhaging, increased sensitivity to disease and reproductive problems (Scott et al 1985).*

Screening for mycotoxins has become a statutory requirement for clearance of food commodity exports at ports of entry in most developed countries and even in internal food commodity trade (e.g. in USA). This includes milk and dairy products because many mycotoxins like aflatoxins and ochratoxin A are secreted into mother's milk.

NON-TOXIC NATURAL FOOD CONSTITUENTS

These, by definition, are not poisons and many of them indeed are essential nutrients. However, they are numbered among poisons because in certain sensitive individuals they behave like one. Only a minute fraction of the population reacts adversely to them. Lucretius (96-55 BC) must have been referring to this kind of adverse reaction to wholesome food in his ageless saying: '*One man's meat is another's poison*'. Being intrinsically non-toxic, most of these food components induce only food sensitivity type of adverse reactions to food.

The sensitive individual reacts to these food components as poisons either because somewhere along his life, his body had developed antibodies against the particular food or food item (*food allergy*); the person is afflicted by a metabolic or gastrointestinal disorder which is specifically exacerbated by the particular food item (Table 5); or his life chemistry is unduly sensitive to certain food components (Table 6).

Table 5. Examples of metabolic and gastrointestinal disorders known to evoke adverse reactions to ingested food in sensitive individuals.

Disorder	Cause	Food/Food item evoking adverse reactions.
Lactose intolerance*	Genetic or acquired deficiency of milk sugar (lactose)-digesting enzyme, lactase.	**Whole milk, milk sugar, milk-based foods.**
Diabetes mellitus	Insulin insufficiency; insulin-independent factors.	Carbohydrate-rich foods.
Hypoglycaemia	Several factors.	Foods with high sugar contents e.g. soft drinks.
Galactosaemia**	Genetic deficiency of galactose (a milk sugar component) metabolizing enzymes.	Whole milk, milk sugar, milk-based foods.
Phenylketonuria	Genetic deficiency of phenylalanine (a protein building block)-degrading enzyme, phenylalanine hydroxylase	Proteins rich in phenylalanine and tyrosine.
Glucose-6-phosphate dehydrogenase deficiency haemolytic anaemia	Genetic deficiency of G-6-P dehydrogenase, a glucose metabolizing enzyme	Broad beans.
Dietary protein intolerance	Partial or mild deficiency of any of the enzymes of urea synthesis (genetic or acquired).	Protein-rich foods/meals.
Gouty arthritis	Defect in purine (a nucleic acid component) degrading enzyme(s)	High protein and high purine diets.
Peptic and gastric ulcers	Diverse factors	Many food substances.

** Characterized by diarrhoea, often without vomiting, upon ingestion of milk; incidence highest (up to 80%) among Africans, African-Americans, Asiatics and lowest among the Danish population (3%); may co-exist with gastrointestinal allergy due to milk proteins.*
*** In infants.*

Table 6. Examples of pharmacologically active food components which may evoke adverse reactions in sensitive individuals.

Component	Occurrence (examples)	Effects
Vasoactive amines:		Constriction of blood vessels; chemical messengers in diverse metabolic reactions and physiological functions.
Histamine*	Red wine, fermented cheese, certain fish	
Tryptamine	Cheese	"
Serotonin	Fruits like banana	"
Synephrine	Citrus fruits	"
Phenylethylamine	Chocolate	"
Methylxanthines (e.g. caffeine,** theophylline, theobromine)	Coffee, tea, cocoa, kola nuts.	Stimulation of energy-producing carbohydrate metabolism.
Salicylates	Berry fruits, oranges, honey, pineapples, cucumber, thyme, wines, tomato sauce, port.	

** Histamine is a chemical mediator in induction of food allergy and some pseudo food allergies (the target of antihistamine drugs).*
*** Caffeine-induced adverse reaction is one of the most frequently encountered non-immunologically mediated food sensitivity. Characterized by a variety of reported reactions, notabl,y headaches, vomiting, excessively rapid heartbeats (tachycardia), hypertension, severe abdominal pain.*

Immunologically–mediated food sensitivity or food allergy is associated with food proteins. It occurs when certain proteins or polypeptides (called *allergens*) present in food evokes production of antibodies of the type called *immunoglobulin E* (IgE) against them. The most frequently encountered allergy-causing food proteins are those of egg

white, cow's milk, wheat flour, and cod fish. An allergy causing proteinous food may contain several distinct allergens of major, intermediate, or minor importance. For example, studies have shown that egg white, cow's milk, and wheat flour contain no less than 20, 25 and 40 distinct proteins, respectively, that can act as allergens in humans. The reaction between a food allergen and its specific IgE antibody triggers the release from certain tissue cells (basophils and mast cells), chemical mediators, such as, histamine, which cause harmful effects on the surrounding tissues, culminating in clinical symptoms. The clinical symptoms that ultimately manifest are dependent, among others, on the site in the body where the allergen-IgE interaction took place e.g. asthma, if it occurred in the respiratory tract, angio-oedema and infantile eczema, if in the skin, and vomiting/diarrhoea if in the gut.

Allergy to a given food protein may be inherited (called *atopic allergy*) or acquired. In the former, the allergic individual has in his system, IgE antibodies specific for the particular protein. Food allergy is commonly acquired at infancy through premature feeding of babies with egg, cow's milk, and other protein rich foods. The gut wall of babies is immature and porous, and, consequently, absorb poorly digested protein fragments which his system sees as foreign bodies and develops antibodies against them. Thus, on subsequent consumption of the food protein, preformed IgE antibodies promptly confront it, initiating adverse reactions.

Some food proteins induce allergy only when eaten in the raw state (losing their potency during processing or cooking) while others do so in both the raw and cooked states. Many vegetables and fruits lose their allergenicity during processing (e.g. apple) or cooking. In atopic allergy to cod fish protein, allergenicity is retained even after the food had been cooked, eaten and digested by the mother: the breastfed baby developed acute symptoms of allergy each time the mother had eaten cod fish prior to breast feeding.

Some individuals develop allergy to a food protein because they already had non-food allergy to the same protein by other means, such as, inhalation (*inhalant allergy*). The inhalant and food protein allergens could be from the same (e.g. fish dust or wheat flour inhalant allergen) or different (e.g. plant pollen) sources. Food allergy developed in this manner is said to be acquired through crossreactivity with non-food allergies. In the case of cross reactivity with an inhalant allergen in a plant pollen, such individuals have primary allergy to the plant protein but develop food allergy when the allergen in the food consumed orally is absorbed through the mucosa and is recognized by the preformed specific IgE antibody. Such cross reactivity has been observed between: grass pollen and wheat; ragweed pollen and melon; and birch pollen and carrot, potato and apple.

FOOD ADDITIVES

Simply defined, food additives are substances added intentionally to foodstuffs to perform certain technical functions, for example, to impart desired colour, to sweeten, or, to preserve. Food additives have been used for centuries traditionally, at home (e.g. common salt, sugar, smoke) and in food processing factories. But developments in food science and technology in the last 50 years or so, have resulted in hundreds of new substances being discovered/deviced and added to the traditional food additives, such that well over 2000 additives are commonly in use today. Many of them are intrinsically non-toxic natural products, such as, plant and animal extracts (e.g. gum arabic, accacia gum, plant protein, lecithin, etc) and essential nutrients (e.g. vitamin E, sugar, common salt) but the majority are synthetic compounds.

In the 1980s and 1990s, there was a serious, sustained, campaign against the use of food additives as they were blamed for the spate of reported adverse reactions to food. However, scientific verification of those claims revealed that only a disproportionately small number of the reported food borne illnesses could be attributed to food additives. Reports of adverse reactions are frequently encountered with certain classes of food additives (Table 7).

Table 7. Some of the food additives commonly reported to cause adverse reactions.

Additive	**Purpose and Use**	Reported side effects in sensitive individuals*
Aspartame	A low-calorie sweetener, (Nutra Sweet®) in many foods and beverages.	Angio-oedema, swelling of eyelids, lips, hands or feet.
Tartrazine	A yellow colourant (USA FD & C Yellow No.5; EEC E102) in beverages, candy ice cream, custards, salad dressings, yoghurt, etc.	Skin rashes, nasal congestion, and hives (1-2 persons per 10,000). Asthma. Hence, by law, additive must be listed on product label.
Sulphites**	Preservatives; to control microbial growth in fermented beverages and other foods. Found in wines, beers, fruit products, baked goods, soup mixes, etc	Chest tightness, hives, abdominal cramps, diarrhoea, lowered blood pressure, light headedness and an elevated pulse rate. Triggers asthmatic attacks in sulphite sensitive asthmatic, in a few cases fatal. By law, must be listed on product label.

BHA/BHT	Antioxidants, in foods containing fats and oils. In cereal and other grain products.	Hives and other skin reactions. BHA established as carcinogenic in animals. Banned in some countries.
Monosodium glutamate (MSG)	A flavour enhancer in a variety of foods.	Chinese Restaurant Syndrome*** presumably when consumed in large amounts. Trigger/aggravate asthma attacks in MSG sensitive asthmatics.
Nitrates/Nitrites	Preservatives in processed meats like bacon, bologna, salami, etc.	Headaches and possibly hives. Nitrite has long been suspected as being a cause of stomach cancer.
Potassium bromate	Bread (volume and texture) improver.	Causes cancer in animals. Banned virtually worldwide.
Saccharin	A sweetener in beverages and foods.	Carcinogenic to animals. Banned in many countries, carry cancer warning in others.

* *Incidence of each of these side effects is extremely rare (All, excluding bromate and saccharin).*

** *A group of several inorganic sulphites (E220-228) including sodium sulphite, potassium bisulphite and metabisulphite, containing sulphur dioxide, SO_2.*

*** *Characterized by headaches, nausea, diarrhoea, sweating, chest tightness, and burning sensation along the back of the neck.*

In some cases, the food additives initiate the adverse reactions attributed to them while in others they act by exacerbating a pre-existing condition. Some do both.

By and large, there is a greater risk to health from chemical contaminants and toxic natural constituents of food than from food additives. Estimates from studies in the UK (Doll, cited by Truswell, 1990) indicate that while these other chemical substances account for about 35% of deaths from cancer, food additives account for about 1%. Relatively, therefore, food additives are considered safe components of our diet. This is traceable to the fact that they are thoroughly tested before approval for use. But these tests are not absolutely fool proof, hence the reported adverse reactions to them, some of which are fatal. That they are used at very minute levels (the permitted levels are such that maximum intake does not exceed the acceptable daily intake which is equivalent to

the highest level that has no effect in animal tests) does not preclude them from initiating or promoting the development of neoplastic lesions like cancer or exacerbating a pre-existing disorder. For this reason, they are closely monitored and anyone with proven adverse reaction history is withdrawn or banned.

HOW DOES THE BODY COPE WITH FOOD POISONS?

The human body is endowed with chemical defences against different classes of foreign and endogenous toxic chemicals. Some of these are preformed while some are induced by the presence of the offensive chemical. The liver is the chief organ site for the detoxication of toxic chemicals (which are not proteins). In addition, each cell of the body is endowed with a chemical machinery for repair of damage to the genetic material, deoxyribonucleic acid (DNA). However, these and other inherent chemical defences are not 100% efficient or foolproof. Some chemicals by their nature, escape transformation while some of those that are transformed, produce reactive intermediates a fraction of which react with surrounding tissues before the chemical defences are able to neutralize them. An example of the latter are a certain class of cancer-causing chemicals (*carcinogens*): they bind with cell molecules, such as, proteins and DNA to initiate lesions. Binding of reactive intermediates to DNA, is the first step in tumour initiation by chemical carcinogens like aflatoxin B_1. Similarly, the first step in the induction of immnosuppressive effects by *Fusarium* mycotoxins like deoxynivalenol and T-2 toxin, is the binding of their reactive metabolities to proteins and other molecules in cells of the susceptible tissues (bone marrow and thymus), ultimately leading to the destruction of those cells with the attendant consequences on the immune system. On the other hand, some undergo transformation to yield end products that are not completely harmless (e.g. cyanide is detoxified to thiocyanate, SCN^-, which can interfere with the uptake of the essential nutrient, iodine, by the thyroid gland). At the other extreme is the limited capability of excretory organs. The human excretory system is incapable of excreting some of the foreign chemicals like lead, cadmium and mercury. These remain in the body wreaking havoc on the organs where they are deposited. Chemical defence capability is, however, not the sole determinant of the ability of the human system to cope with toxic chemicals in food. Whether the adverse reaction initiated by a food toxicant takes root and develops into a frank clinical condition is dependent on a number of other factors (intrinsic and extrinsic), such as, genetic endowment, state of health, and nutritional status.

PREVENTION AND CONTROL

How do we avoid or prevent 'poisons' (and pseudo poisons) in our diet? The truth is that they cannot be completely eliminated from the diet or environment. We can only completely eliminate agrochemicals from our food if we cease to employ chemicals in agriculture and animal husbandry. We can save ourselves from food additives only if we cease to employ chemicals, including smoke, in processing and preserving our foodstuffs

and eat only natural foods. Both seem impracticable as modern agriculture and food processing are key components in promotion of food security. Neither can we avoid using metal utensils because many of them contain mobile toxic elements. Those who live in virgin, unpolluted, rural environments may well succeed in completely avoiding industrial chemical contaminants, provided they totally abstain from processed and aquatic foodstuffs "imported" from outside their community. Furthermore, everyone, city or rural dweller, is helpless as far as harmful chemical substances in water are concerned. In the case of fungal mould toxins, scientists have after fruitless efforts at various options of prevention, concluded that complete elimination of mould infection and mycotoxin contamination of grains, for instance, will not be possible until mould-resistant varieties of grain crops become available (Trenholm *et al.* 1989). However, with bacterial toxins, the effect of the toxins can be prevented through immunization, though foodstuff infection and toxin production cannot be prevented but, then, this is for a minuscule fraction of pathogenic food spoilage bacteria.

Scientists have, long ago, recognized the inevitability of these poisonous substances in our foods and drinks which is why the approach adopted in solving the problem has been that of minimizing the level of exposure to them through statutory regulation and control of their permissible levels in foodstuffs, feedstuffs, drinks and domestic water. The UN system sets the regulatory and control tone at the apex international level through its joint FAO/WHO activity Codex Alimentarius Commission which develops guidelines globally an food safety with technical advice from the Joint FAO/WHO Expert Committee on Food Additive (JECFA). There are complementary initiatives from other UN agencies like International Atomic Energy Agency (IAEA), Geneva, and UNEP, while powerful regional intergovernmental organizations and many UN member nations, working in cooperation with Codex and JECFA, have instituted their own regional and national standards, respectively, through legislation. Britain was the first country in the world to enact a food safety regulation, the *Safety of Food and Drugs Act 1875*, 60 years before the birth of UNO. Nigeria had her first comprehensive food safety legislation in 1974 which was the forerunner of the operating legislation National Agency for Food and Drug Administration and Control (NAFDAC) Decree 1993 as severally amended. Food safety legislations invariably provide for an organizational structure for their enforcement. Under the enabling law, the enforcement agencies develop and issue statutory guidelines on food safety and enforce them. Countries which do not have their own national standards generally adopt Codex standards (e.g. Nigeria formally adopted Codex in 2000). Standards are not static but are renewed on a regular basis based on developments in the science of food and feedback from the field.

Recommendations

The following are my suggestions on finetuning the existing machinery for the prevention and control of food borne poisons and illness in Nigeria.

1. **Food poisoning and sensitivity as statutorily notifiable diseases.** As is the practice in advanced countries, acute adverse reactions to food should, by law, be mandatorily notifiable to appropriate authorities. Such a feed back mechanism will serve not only in keeping proper public health statistics, identifying food substances commonly causing food borne illness, doing informed review of food safety regulations but also in taking effective remedial action. The absence of such a mechanism is responsible for the near absence of credible data on the nature and incidence of food borne diseases in Nigeria. NAFDAC has already started a similar initiative for adverse drug reactions. The notifiable disease enactment should include provisions requiring food processors to indicate on their product labels, food components liable to induce adverse reactions (allergy). The EEC guidelines, for instance, require food processors to indicate on their product labels, the presence of any allergen.

2. **Data bank on chemicals contaminating Nigerian staple foodstuffs and feedstuffs and of potentially hazardous staple foodstuffs.** Again, this is a common practice in countries where public health is taken seriously. The data bank is a key requirement for proper planning. The generation of data for the bank should not be an exclusive NAFDAC affair but must actively involve researchers in the academia (e.g. through project or commissioned research grants). Building a data bank in an area like mycotoxins that has produced at least seven Nigerian professors would not be a problem.

3. **Statutory requirement for routine screening and certification of food commodities for various contaminants.** This should be particularly so for mycotoxins and the globally recognized hazardous man made agrochemicals and industrial chemicals contaminants. The USA FDA, for instance, routinely monitors grains harvests for aflatoxins and other mycotoxins and advises the relevant bodies and farmers accordingly.

4. **Public education on food poisons.** This should include education on good food preparation/processing, production, and storage practices; on the dangers of poisons in mouldy foodstuffs and foods produced in chemical polluted environments; and on the need to ask questions as to the sources of certain

foodstuffs like vegetables, fresh fish and non-proprietary drinks. There should be education against harmful traditional practices that dispose to chemically contamination (e.g. sundrying fermented cassava on chemically polluted surfaces like tarred roads).

CONCLUSION

Scientific research has shown that potentially toxic chemical substances are essentially part and parcel of the human food chain and that only very few of them can be completely eliminated during food preparation. In the circumstance, minimizing the levels of exposure to these poisous chemicals through legislation is the only viable option. Frank food poisoning symptoms definitely manifest when we are exposed to acute doses of these poisonous chemical substances in food. Adverse reaction symptoms may manifest on exposure to sub-acute doses depending on the actual level of exposure (i.e. actual amount of poison ingested) and a number of other variables such as, the state of health, genetic endowment, nutritional status and the body's chemical defence capability. Where the chemical properties of the toxicants are known, the lesions they can cause in the mammalian tissues are fairly predictable.

In addition to these inherently toxic chemical substances, wholesome food substances may become rebellious and behave like poisons when consumed by certain sensitive individuals. While it may be possible to completely eliminate some of the food substances which behave in this way through legislation (e.g. synthetic food additives), it does not arise for essential natural food items like proteins. At best, they can be eliminated from the diet of those proven to be sensitive to them. Food sensitivity is unpredictable; we come to know of our sensitivity to a particular food item only after we have been afflicted or diagnosed to be so.

The clinical manifestations of symptoms of adverse reactions to food poisons and neo-food poisons, are not any different from those of illnesses due to pathogenic organisms and metabolic disorders. Adverse reaction to chemical substances in food is indicated when routine clinical diagnostic tests for pathogenic organisms (e.g. bacteria, viruses, parasites) and indicators for common metabolic disorders prove negative or, in cases where symptoms are those of common illnesses, when informed treatment fails to bring relief.

The issue of food borne poisons and food borne illness therefore calls for public awareness and vigilance, and commitment to recommended measures for the control of this class of ailment. The price of liberty from food sensitivity, for instance, is eternal vigilance with commitment – vigilance to know that we react adversely to certain foods and commitment to identify the particular food items causing the problem and eliminate them from the diet.

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INAUGURAL LECTURER'S BIOGRAPHY

Professor Z.S.C. Okoye was born at Agulu in the old Awka division of then Eastern Nigeria (the present day Anaocha LGA of Anambra State), the last of five children, 3 girls and two boys.

Professor Okoye started his eight-year primary education, initially at CMS Central School, Onitsha in 1952, and finally at St. Luke's CMS School, Agulu in 1953. On completion of the infant classes, he moved over to St. Peter's Central School, Agulu, in 1955, and, subsequently, in 1958, to Holy Trinity School, Igbo Ukwu where he successfully completed his primary education in 1960 with a credit pass East regional first school leaving certificate.

He began his secondary education at then prestigious Government Secondary School, Afikpo on 26 January 1962. At the end of his Form One, following his brilliant academic performance, he was awarded the school scholarship and was selected to be in the first set of the Accelerated Class to do the secondary school curriculum in four instead of five years. He successfully completed his secondary school course in 1965 with a Division One pass in West African School Certificate (scoring A1 in Chemistry and Literature). He started his Higher School Certificate course the following year in the same school. The course was cut short in 1967 by the Civil War, at the end of which, in 1970, he transferred to and completed the course at Federal Government College, Warri with A, B, B principal level passes in the Cambridge University Higher School Certificate.

Okoye worked briefly as a bank clerk at the Chase Branch, Lagos, of the then Standard Bank Nigeria Ltd (January – September, 1971) and proceeded to University of Ibadan in September, 1971 for his B.Sc. Biochemistry degree programme. Again, his first year's performance earned him a scholarship, this time, a German Academic Exchange Scholarship, tenable at and administered by University of Ibadan. He graduated in 1974 with a second class upper division degree and the best graduating student in is class. Consequently, he bagged the best graduating student's prizes and a UI postgraduate scholarship (bursary). After his NYSC service (1974-75), which he did in Lagos State, with his primary assignment at Badagry Grammar School, and about two months at Federal Ministry of Health, Lagos, as a pupil food inspecting officer in the newly established Food and Drug Administration and Laboratory Services, he returned to U.I. to pursue his postgraduate degree programme in biochemistry. He completed the programme in 1978 with the successful defence of his Ph.D. on 6, October, 1978.

Professor Okoye began his postdoctoral working career at University of Jos as a Lecturer II in the then Department of Human Chemistry, in February, 1979. He rose steadily through the ranks to become a Professor in October 1990. Professor Okoye has taught virtually all topics in basic medical biochemistry but in non-medical programmes, his major courses are nutrition, forensic biochemistry, blood biochemistry, plant

biochemistry, xenobiochemistry and toxicology, and immunochemical techniques, the last two primarily at PG level.

Professor Okoye's research activities have centred on the incidence and biochemical toxicology of food contaminants, especially, mycotoxins, and factors, including traditional food additives, affecting their toxicity, and recently, verification of traditionally claimed medicinal effects of and toxicity risk assessment, of indigenous medicinal plants. His research output can be found in at least 54 journal articles, most of which are published in journals of international repute. In addition he has published/edited two monographs and four books including a textbook, *Biochemical Aspects of Nutrition* published by Prentice-Hall of India.

Professor Okoye has won seven international research and travel fellowships including Commonwealth Medical Fellowship (1985-86) from Association of Commonwealth Universities, London, three different awards (ICRETT 1980, 1988 and Yamagiwa-Yoshida Memorial International Cancer Study fellowship, 1989-90) from the International Union Against Cancer (UICC), Geneva and the Wellcome Trust, London, travel fellowship. On completion of the Y-YMICS-sponsored study at Medical Research Council Toxicology Unit, Carshalton, UK, Okoye was made a fellow of UICC. The other awards were the Royal Society/Nuffield Foundation Developing Country Fellowship and Congress Award of 11th International Pharmacology Congress, Montreal, Canada, which were not utilized. All the awards, except the Congress Award, were for studies on mycotoxins.

Apart from teaching and research, Okoye has been actively involved in university administration. He has been Head of Dept, Biochemistry (1988-1990), Sub Dean (Preclinical), Medical Sciences (1988-1990), Sub Dean, Postgraduate School (1990-1993), Dean of Postgraduate School (1993-1997) and Chairman, Committee of Deans and Directors (1996-97). He was a member of the Governing Council representing Senate (1993-97) and while there, served on the Finance and General Purposes Committee. A member of Senate since 1983 (first as Faculty/School representative and later in his own right as a Professor), he has served on virtually all academic policy making organs/committees at Senate committee, faculty and departmental levels. He has chaired a number of key Senate committees like Affiliation and Linkages, Central Research Grants and Special Admission Committees. In addition, he has served on several University, Senate, and Faculty ad-hoc committees and taskforces too numerous to mention here.

Externally, Professor Okoye has served as external examiner at undergraduate and postgraduate levels to 11 different Nigerian universities and as external assessor for professorial and other senior staff promotion/appointment to 13 different universities. He

has been a resource person in FMH UNICEF Iodine Deficiency Disorders (IDD) Day Celebrations (1995, 96, 2000) and a member of Nigerian Delegation to the Regional Conference on IDD for Anglophone West Africa, Abuja, 1999. He has been a member of the National Advisory Committee on Micronutrient Deficiency Control., since 2001. He was a member of the technical committee that formulated the proposal and guidelines for the Nigerian Food Consumption and Nutrition Survey 2001-2003 and served ass the North-East Zonal Coordinator up to the pilot study stage.

Professor Okoye is a current member of Nigerian Society of Biochemistry and Molecular Biology (NSBMB) and Nutrition Society of Nigeria. He was a member of Council of NSBMB (1984-95) and Editor of its organ, Nigerian Journal of Biochemistry (1990-95). He is a reviewer to several research journals, both national and overseas.

Professor Okoye is cited in at least five biographical journals, local and overseas, and is on the research advisory board of two of them, the American Biographical Institute, Raleigh, USA and International Biographical Centre, Cambridge, England. A Knight of St. Paul, Professor Okoye was Hon. Commissioner for Education and Youth Development, Anambra State (1997-1999). Professor Okoye is married to Dr. (Mrs.) Christie Okoye, a great alumna of the university, and the marriage is blessed with two ladies and two gentlemen.

In the absence of facilities for his favourite sports, hockey and cricket, Professor Okoye has current affairs and watching popular sports, historical documentary and science fiction films as his hobby.

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