PLANT PATHOLOGY: PLANT AFFLICTIONS AND MAN'S INTERVENTIONS

AN INAUGURAL LECTURE
2010/2011

By

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UNIVERSITY OF IBADAN

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An Inaugural Lecture delivered at the University of Ibadan

on Thursday, 31 March, 2011

By

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

First Published 2011

ISBN: 978 – 978 – 8414 – 49 – 0

Printed by: Ibadan University Printery

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

Introduction

I thank the University of Ibadan for giving me this once-in-a lifetime opportunity to deliver this inaugural lecture. I must also thank my Dean of the Faculty of Agriculture and Forestry and my Head of Department of Crop Protection and Environmental Biology for ensuring that I deliver this lecture shortly after the 2009 lecture delivered by my Department. Each time that it was my turn, I was inadvertently away from the University and the opportunity was passed on to other professors on ground. It has thus taken me 22 years after my elevation to the post of Professor, to deliver my own lecture.

This lecture, the fifth in the series of 2010/2011 inaugural lectures is the 37th from the Faculty of Agriculture and Forestry and the 9th from the Department of Crop Protection and Environmental Biology. This lecture is coming after those of the late Professor Timothy Ajibola Taylor (1974), Professors A. Youdeowei (1977), N.O. Adedipe (1979), the late O.F. Esuruoso (1982), M.O. Adeniji (1984), J.K. Egunjobi (1990), J.A. Odebiyi (2004) and B. Fawole (2009). It is the third lecture given by a Mycologist, coming after two of my teachers—Professors Esuruoso and Adeniji who stimulated my interest in plant pathology.

My interest in plant pathology started when I developed a phobia for dissecting earthworms and toads in my lower sixth year in 1967. In October that year, one Dr. Adeniji attended Government College Ibadan Old Boys Reunion in our College Hall and told a short story about how, having returned within a Ph.D degree from Nottingham, U.K., he was appointed a Research Officer at Moor Plantation, Ibadan. In front of his house was a small sign board labelled Dr. M. O. Adeniji. Two weeks after, a neighbour woke him up at

2.00 am crying that his child had convulsion and that the "Doctor" should come and treat the child. It took Dr. Adeniji more than twenty minutes to convince the neighbor that he was a plant doctor and not a human doctor, whereupon the agitated man retorted angrily, "Who tell you say plant dey sick?. You no sabi weting you dey talk". Out of pity, Dr. Adeniji put father, mother and the convulsing child in his car at that time of the night and drove them to Jericho Nursing Home where the child was treated. This incident gave me an insight to the fact that plants could be sick and could be treated. That Dr. M.O. Adeniji became my teacher in plant pathology and was my senior colleague in the Department of Crop Protection and Environmental Biology for years before he retired.

Another incident that generated my interest in studying plant pathology was another true story from the late Professor Esuruoso who, after bagging two degrees in Botany and a Ph.D in plant pathology, was asked by a layman what his profession was. He told the man that he was a plant doctor. The man thought that Dr. Esuruoso was going to be a herbalist and commiserated with him for wasting his degrees. Why go to a University to obtain degrees and end up being a herbalist when all successful herbalists never went to school? Haba! Undaunted, Dr. Esururoso pursued a fruitful career, ended up as the first Professor of Plant Pathology (Mycology), retired from the University of Ibadan and died in 1999. May his gentle soul rest in perfect peace.

Even then, I was not lucky to be accepted to undertake my project work in plant pathology in 1971/72, my final year. The money I needed for a successful final year was not ready when the University reopened. By the time I arrived from home and reported in the Department, the two plant pathologists had chosen students to work under their supervision. We were afraid to work under Professor Taylor because he was always away from the Department as a result of his numerous foreign travels, his position as Dean of the Faculty and as the Acting Vice-Chancellor. So, I opted to work with the then Dr. Anthony Youdeowei, our Entomology teacher who always painted my reports submitted to him with

red ink. Even though I was quite good in Entomology, I commiserated with the poor insects I squashed in an attempt to collect them for my studies. For every ten insects I collected, I had squashed five. A classmate of mine then, one Mr. Ode Iyoha told me not to be too downhearted because the ones that did not die during collection would be gassed and dissected, which was worse than being squashed during collection. Putting them out of their misery during collection was also better than being sprayed with noxious insecticides in the field. I was thus comforted.

After my final year examination, I was surprised that throughout my undergraduate days, Professor Taylor was monitoring my progress inside and outside the Department. He had seen me several times during Inter Halls Athletics meetings while throwing javelin and discus. He also saw me with a hockey stick several times, because, after our practical classes, I always went to the sports field to play hockey. In fact, I was the University Hockey Team Goalkeeper. He also saw me play cricket and saw me swimming. He then checked my results in the Faculty and found that I was one of the top three in a class of eight students. I never knew that he was taking notes. To me, I was practising what I learnt in Government College, Ibadan—to be an all-round sportsman.

I was pleasantly surprised when after our final examinations and our results were not yet published, Professor Taylor called me to his office and asked what I intended doing after my education in the University. I told him that I had been interviewed and appointed, by the Federal Government, to become an Entomologist and that I would be sent to the Pest Control Unit in Kaduna. He asked me what I thought about being diverted from entomology to plant pathology where I was also good. I quickly jumped at that suggestion because that would take me away from mass murder of insects, since I did not want to face the International Court of Justice in the Hague, Netherlands, for committing genocide on insects. He called me the following day with copies of letters he as Head of Department (HOD) wrote to himself as Dean of Faculty recommending me and my bosom friend, Mr. Dele Fawole as recipients of the Ford Foundation/University of Ibadan Staff

Development Fellowship, and his reply to the HOD accepting the recommendation and said that the recommendation would be passed to the Acting Vice-Chancellor for approval.

Expectedly, the Acting Vice-Chancellor who was Professor Taylor wrote to the Dean of the Faculty who was Professor Taylor conveying approval of the recommendation and the Dean of Faculty conveyed the Acting Vice-Chancellor's approval to the HOD of the Department who was Professor Taylor. He had also arranged with the Director of the International Institute of Tropical Agriculture to place Dele Fawole and I in their Nematology and Plant Pathology Laboratories, respectively, to learn advanced techniques. We were there from June to early September 1972.

Professor Taylor called me one day and handed me a letter of admission to the famous Imperial College of Science and Technology, University of London, U.K. A second letter was from my supervisor to-be, Professor R.K.S. Wood who asked one to bring a bacterial disease of importance in Nigeria to London for my studies. As luck would have it, a new disease of cassava caused by a bacterium had just invaded Nigeria and all other cassava producing countries of the world and IITA was at the centre of research on this serious epidemic. That was how I became the first man to take cassava to London and grew the cuttings in pots on our roof-top garden at South Kensington, SW7, England.

A duplicate set of cassava cuttings was grown at Chelsea gardens and it attracted thousands of visitors who had never seen cassava before. I had to deliver a coloquium on cassava, its potential yield and its pests and diseases at Imperial College. Even the Nigerian High Commissioner to U.K. then, attended this colloquium—the first of its type. While at Imperial College, the IDRC of Canada wrote to award me a fellowship to go to the Centre Internacional de Agricultura Tropical (CIAT) in Cali, Colombia for my field studies. I spent fourteen months there under the supervision of Drs. Carlos Lozano and James Cock. Their contribution to my field studies enriched my work immensely. At Imperial College, it took me three weeks to graduate from the third team (a starting point for everybody) to the first team as the

Goalkeeper (hockey). I could not repeat that feat in cricket. I merely graduated from the third to the second team. Whenever I played on the field, members of the opposite sex flocked the field shouting "Tondy, Tondy....." Exactly three years after I left Nigeria, I obtained my Ph.D and returned home on October 6, 1975, on the same plane as the present Deputy Vice-Chancellor (Administration), Professor E. A. Bamigboye.

It was nice to be home again. Then came the big problem of settling down and setting out my nets and tentacles to catch a mature apple, a crown jewel—after all, I was already 27 when I returned to Nigeria. Before leaving for London, my father had been beating a message down my head –"It is time to get married; I don't want a white wife. She will not be able to pound yam or prepare *amala*." All these I kept in my left hand and on return set my traps which I checked every day. I eventually made a very big catch and the rest is history.

In 1981, the University awarded me a Staff Development Fund to enable me update myself in Mycology at the Commonwealth Mycological Institute, Ferry Lane, Kew, Richmond, Surrey. There were twenty-five people at the course from all over the Commonwealth. The first week was devoted to Bacteriology. From my performance in the first week, I was appointed a demonstrator to help other colleagues who had no prior knowledge of Bacteriology. The remaining five weeks were devoted to Mycology where I increased my understanding of the field. On return to the University, I started to teach advanced courses in Mycology and Bacteriology, which I still teach today thirty years after. Both fields have made me an accomplished plant pathologist today.

What is Plant Pathology

Pathology is derived from two Greek Words-"Pathos' and "Logos" Pathos means "suffering" and logos means "discourse". Thus plant pathology means "a study of suffering of plants". The title of this lecture is "Plant Pathology: Plant Afflictions and Man's Interventions".

By the definition of Walker (1957), plant pathology is the study of the health and productivity of growing plants. Disease losses are hazards which can be minimized only by a continuous process of research and education.

According to Agrios (1969), plant pathology is the "study of the living entities and the environmental conditions that cause diseases in plants; the mechanisms by which these factors produce disease in plants; the interactions between the disease-causing agents and the diseased plants and the methods for preventing disease, alleviating the damage it causes, or controlling a disease either before or after it develops in a plant".

Agrios (2005) redefined plant pathology as a "science that studies plant diseases and attempts to improve the chances for survival of plants when they are faced with unfavourable environmental conditions and parasitic microorganisms that cause disease". Plant pathology to plants is what human medicine and veterinary medicine are to humans and animals, respectively. Each of these disciplines studies the causes, mechanisms and control of diseases affecting the organisms with which it deals, be they plants, animals or humans.

Plant pathology is an integration science, a profession that uses and combines the basic knowledge of botany, agronomy, horticulture, soil science, mycology, bacteriology, virology, nematology, plant anatomy, ecology, plant physiology, genetics and genetic engineering, biochemistry, physics, chemistry, meteorology, forestry, tissue culture, statistics and many other branches of science (Agrios 2005).

Plants used for food may be cultivated and grown anywhere but plant diseases may attack them, destroy parts or all of the plants and reduce much of their produce (foods such as fruits, seeds, leaves, tubers, roots and medicines obtained from plants and timber used for structural works), before they can be harvested or consumed (Agrios 2005). In the pursuit of its main goal (to protect plants), plant pathology is joined by the sciences of entomology, nematology, crop physiology, ecology, genetics, and weed science. These together make up plant protection.

From 2002 estimated figures, diseases, insects and weeds together destroy 36.5% of all crops produced worldwide (table 1). These figures are lower in the developed world where all technologies have been harnessed to keep them low. In countries that need the food most—developing countries, they are much higher. It has been estimated that of the 36.5% average of total losses, 14.1% are caused by diseases, 10.2% by insect pests and 12.2% by weeds.

Table 1: Estimated Annual Crop Losses Worldwide

Attainable crop production (2002 prices)	$1.5trillion
Actual crop production (-36.5%)	$950 billion
Production without crop protection	$445 billion
Losses prevented by crop protection	$415 billion
Actual annual losses to world crop protection	$550 billion
Losses caused by diseases only (14.1%)	$220 billion.

Source: Agrios (2005)

These are only loses in the field. If we add 6-12% post-harvest losses which are higher in the tropics, the losses could be more staggering. Uncontrolled plant diseases result in less food in the markets, higher food prices (such as have been occurring worldwide since 2007) and poor quality of food. Diseased plants produce may sometimes be poisonous (containing toxic substances such as aflatoxins, ochratoxins, fumonisins, etc) and, as such, are unfit for consumption. Some plant diseases wipe out entire plant species and may affect the beauty of our landscapes.

When a human being is ill, (s)he is taken to see a doctor who asks searching questions from which he tries to diagnose the cause of his/her illness. The job of a doctor is made easier because humans can talk and describe the pain and where it is coming from. For animals, they shout and cry, especially when the affected area is touched, and may even bite the doctor. It is less easy for the veterinary doctor because his patient cannot speak to describe the pains and where it is coming from. For a plant doctor, that is, a plant pathologist, the work is most difficult because the plant cannot speak

hence cannot communicate with the doctor. It is difficult to pin-point exactly when a plant is diseased. Probably, the plants and animals' inability to speak is why their doctors eat them. It would be very interesting to see human doctors eat their patients too!

The Concept of Disease in Plant

A plant is said to be healthy when it can carry out all its physiological functions to the best of its genetic potential. These functions include normal and regular cell division, differentiation and development, absorption of water and minerals from the soil through the roots and the translocation of these items throughout the plant, elaboration of food via photosynthesis at the leaves and green parts and the translocation of the photosynthates to areas of metabolism and storage, production of seeds or other reproductive apparatus for the survival of the race and its multiplication. Whenever plants are disturbed either by microbial invasion or a shift in certain environmental conditions and the inability of the cells to carry out one or more of these essential functions, they malfunction and the plant becomes diseased. At first, the effect is localized to a few cells and is not visible, but it soon spreads to wider areas of the plant and the affected areas develop changes that are visible to the naked eye. These visible changes in the plant could be in the form of loss of green coloration of the leaves in which case the leaf turns yellow (*chlorosis*), or loss of leaves (*defoliation*), or leaves turn brown and dead (*necrosis*), or the shoot tip dies (*die-back*), or a deep wound develops on the stem (*canker*). Also, the root decays and ceases to absorb and transport water (*root rot*), the above-ground parts of the plant lose turgidity and collapse due to blockage of the xylem vessels (*wilt*), the fruits do not develop and drop prematurely (*fruit drop*), flowers drop before they form fruits or seeds (*floral abortion*), swellings on parts of the plant especially near the soil level and other parts of the plant (*tumour, galls, knots*), discoloured spots on leaves (*leaf spot*), browning on large parts of the leaf (*blight*), etc. These are evidences that all is not well with the plant. These visible evidences (see plates) are called

symptoms of the disease. The visible or otherwise measurable adverse changes in a plant produced in reaction to infection by an organism or to an unfavourable environmental factor are a measure of the amount of disease in the plant. A diagrammatic representation of the interference of diseases in plant is given in figure 1.

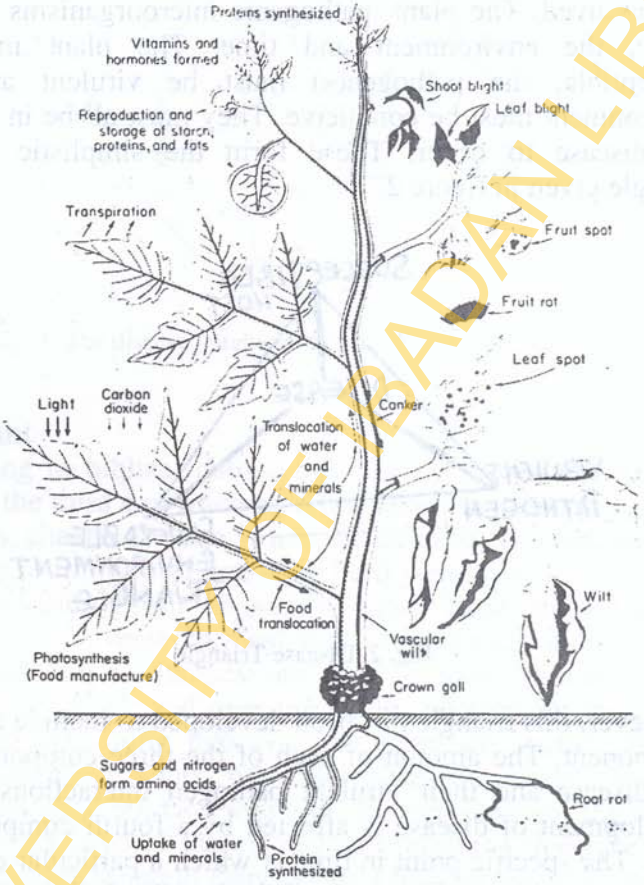

Fig. 1. Schematic representation of the basic functions in a plant and of the interference with these functions caused by some common types of plant diseases (Adapted from Agrios (2005))

Disease in plants can simply be defined as a change in the normal physiological and metabolic activities of a plant caused by a living entity (biotic) or an environmental factor (abiotic) resulting in poor performance of the plant (in terms of yield) and at times its death.

Disease Causation: In the causation of disease, four factors are involved. The plant, pathogenic microorganisms (pathogens), the environment and time. The plant must be susceptible, the pathogen(s) must be virulent and the environment must be conducive. They must all be in contact for disease to occur. These form the simplistic disease triangle given in figure 2.

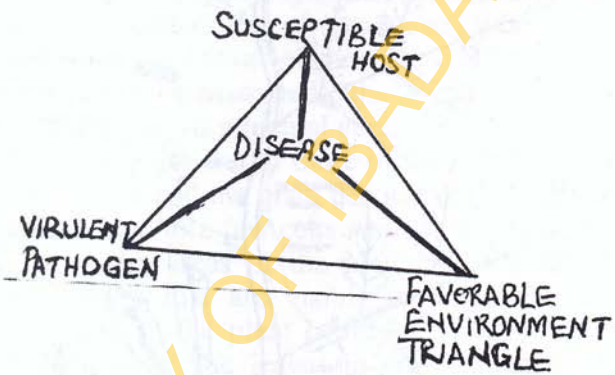

Fig. 2. Disease Triangle

However, this triangle has been developed to include a fourth component. The amount of each of the three components of the disease and their virulent pathogen interactions in the development of disease is affected by a fourth component – time. The specific point in time at which a particular event in disease development occurs and the length of time (duration) during which the event takes place affect the amount of disease. The interaction of the four components can be seen as a tetrahedron or pyramid in which each plane represents one of the components. The figure is referred to as disease

tetrahedron or disease pyramid (fig. 3). Without any of these components, disease cannot occur in plants.

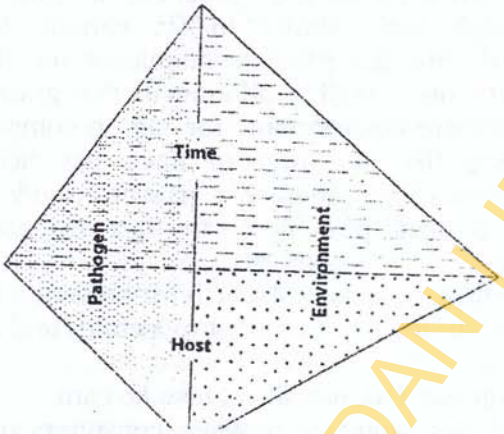

Fig. 3. The disease tetrahedron (After Agrios (2005)).

The Plant

According to biblical chronology, the earth was created by God on the third day when he commanded dry land to appear (Genesis, chapter 1, verse 9) and the same day he commanded the earth to bring forth grassland herb yielding seed and their fruit tree yielding fruit whose seed was in itself, and it was so (Gen 1, 11). In verse 26, God said, "Let us make man in our image and after our likeness and let them have dominion over the fowl of the air and over the cattle and over all the earth and over everything that creepeth upon the earth". Thus, chronologically, plants pre-dated man on earth. Appropriately in verse 30, God said "….. to every beast of the earth and to every fowl of the air and to everything that creepeth upon the earth, wherein there is life, I have given every green herb for meat," and it was so! Plants were ordained by God to provide food for all creatures of the earth including microorganisms.

When plants were created, they were anchored to the soil by their roots where they can stay permanently with their

leaves well displayed at the angle necessary to receive and trap sunlight energy with which to photosynthesize and form food in the leaves, the stem, the roots and the fruits and seeds. Plants, through their ability to fix carbon dioxide via photosynthesis are the primary producers for the world's human population as well as all animals that graze and other organisms that are heterotrophic for carbon compounds. It is not surprising that the first to have the rich pickings (microorganisms) have adopted a parasitic mode of life on plants. That is made possible by the fact that plants cannot move from one place to another.

Who would then blame Macbeth for believing that he was safe when the third apparition (that of a child) told him

> "Be lion-mettled, proud and take no care
> Who chafes, who frets or where conspirers are:
> Macbeth shall never vanquished be until Great
> Birnam Wood to high Dunsinane Hill shall come
> against him".
> (Source: Shakespeare….*Macbeth*)

Of course, Macbeth laughed of the impossible and said:

> "That will never be: who can impress the forest,
> bid the tree unfix his earth-bound root? Sweet
> bodements! Good! Rebellious dead, rise never
> till
> the wood of Birnam rise, and our high-place
> Macbeth shall live the lease of nature, pay his
> breath to time and mortal custom".
> (Source: Shakespeare……*Macbeth*)

Macbeth knew that trees can never move from one place to another, let alone to high Dunsinane, hence, he had nothing to fear.

It is, in fact, the earth-bound nature and fixed position of plants that exposes them to all types of abuse, misuse and destruction. The roots are gnawed by rodents, micro-organisms, and macroorganisms all attack plants in the soil,

causing diseases known as root rot, root gall and wilt, leading to the death of the whole plant and at times toppling over of trees. The above soil portion of the plant is also exposed to assault and battery from birds, bats, insects, rodents and even man. Tree barks of mahogany, mango, guava, cashew and neem (*dogoyaro*) are stripped to the wood by herbalists trying to produce medicaments for fever and other ailments from them. Bats, birds and rodents feed on the fruits and seeds, insects chew their leaves, young shoots, fruits and seeds and even tunnel into the stem. Fungi, bacteria and viruses attack and kill the growing plants, some enter the growing tips and attack the plant and destroy it downwards causing conditions known as heart rot, dead heart, etc.

The leaves of plants are always exposed to sunlight to trap energy from it to synthesize their food, water and carbon dioxide via photosynthesis. But because leaves are always exposed, spores of microorganisms fall on them at the rate of ten thousand each time. These, in the presence of moisture and some chemicals excluded by plants stimulate the spores to germinate, penetrate the leaves through wounds and natural openings such as stomata, lenticels, hydathodes and nectarthodes, enter the tissue of the plants and cause disease. The poor plant having been anchored to the ground is unable to dodge these assaults or dislodge itself from the soil and escape to a safer zone.

Microorganisms

Microorganisms are minute living things that are not visible to the naked eye. They are present everywhere—in the air, water, soil, even on man and inside him. In this lecture we are concerned with those that are pathogenic to (cause disease on) plants.

Fawole in his 2009 inaugural lecture described nematodes (microorganisms) as "small animals with big impact". Esuruoso in his inaugural lecture of 1984 described micro-organisms as "the enemy" in the unending battle on the farm.

Causal Agents of Plant Disease

There are eleven groups of organisms that cause disease in plants. These are fungi, bacteria, viruses, nematodes, oomycota, algae, plasmodiophoromycota, viroids, trpanosomatids, phytoplasmas and parasitic angiosperms.

Fungi

Fungi consist of the phyla Zygomycota, Ascomycota, Basidiomycota, Chytridiomycota, and Deuteromycota. They cause catastrophic plant diseases.

- They sporulate prolifically, the spores providing copious inocula which infect further plants.
- Their latent period between infection and the production of further infection propagules may be a few days.
- The spores if wettable may be spread as high density inocula in surface water or in droplets by rain splash. Non-wettable spores may be carried over long distances by wind.
- They may produce compounds that are phytotoxic and/or battery of enzymes that destroy the plant's structure.
- Biotrophic pathogens such as the rusts and the mildews draw nutrients away from the economically viable parts of the plant by the production or induction of growth regulators such as cytokinins and consequently depress yield.

Bacteria

Bacteria causing plant disease were originally classified in five general groups—the Gram-positive *Corynebacterium* and the Gram-negative *Agrobacterium, Erwinia, Pseudomonas* and *Xanthomonas*. To these must be added the Actinomycetes.

Coryneform Bacteria. Over the last two decades, extensive revision of the Kingdom Bacteria were carried out. Now, the pathogenic Coryneform bacteria are generally classified in

the genera *Curtobacterium, Arthrobacter, Rhodococcus* and *Clavibacter* (Davis 1986: Strange 2003). Members of this group cause symptoms of wilting of leaves which become pale green to yellow, later developing necrotic areas and senescence. Two most infamous pathogens are *Clavibacter michiganensis subspecies sepedonicum* which causes tuber rot of Irish potatoes and *C. michiganensis* subspecies *michiganensis* which wreaks serious losses on tomatoes. In this group is a fastidious bacterium, *Clavibacter* (=*Leifsonia) xyli* subspecies *xyli* which causes the ratoon stunting of sugar cane in producing areas. We are involved in the search for this organism in Nigeria. One of my Ph.D students is working on this. It is likely to be a pernicious pathogen if found in Nigeria.

Agrobacterium. This genus contains causal agents of the crown gall disease (*A. tumefaciens*) found on over 200 dicotyledonous plant species all over the world. Other tumour-forming species include *A. rhizogenes, A. rubi, and A. vitis*. A recent proposal is to keep *Agrobacterium* as an artificial genus comprising plant pathogenic species but to form the new combinations of *Rhizobium rhizogenes*, *R. rubi* and *R. vitis*. Owing to its ability to change plant cells into plasmids that produce the plant hormones such as auxin and cytokinin, *Agrobacterium* with the genes for auxin and cytokinin in synthesis deleted and replaced with genes of interest is now used routinely to transform plants.

Erwinia. Species of this group of bacteria are responsible for blights, wilts, and soft rots. The most infamous of the pathogens is *Erwinia amylovora* causing the firelight disease of pears and apples and the decline of pawpaws in south-western Nigeria. *E. carotovora* subspecies *carotovora* causes soft root of potatoes, carrots, and a wide range of vegetables. *E. chrysanthemi* pv *zeae* causes a highly destructive disease of stem rot in maize in tropical and subtropical countries where maize is grown under conditions of high temperature

and high relative humidity. Plants are most susceptible when they are 40-60 days old. Symptoms consist of withered leaves and brown, soft and water-soaked stem. Infected plants usually emit a foul odour and the stem collapses at an advanced stage of the disease.

Pseudomonas, Burkholderia and Ralstonia. Many serious diseases of plants are caused by species and pathovars of *Pseudomonas*. Many members of this group are now classified as *Burkholderia* and *Ralstonia.* For instance, the old *Pseudomonas* solanacearum which caused bacterial wilt of solanaceous crops of potato, tomato, etc is now called *Ralstonia solanacearum*. Likewise *Pseudomonas glumae* causing a disease of rice glumes is now called *Burkholderia glummae*. Many pathovars (pathogenic varieties) of *Pseudomonas syringae* cause a variety of diseases on a wide range of crops. Symptoms include discoloured or necrotic spots on various organs of many plants, dieback of shoots, cankers on twigs and branches. Water-soaking is often an initial symptom of a susceptible reaction. The species causing rot disease on onions, *P. apii* is a known animal pathogen. *P. aeruginosa*, a common contaminant of infected plants in the field is a dreaded contaminant found on human and animal patients with compromised immune systems.

Xanthomonas. Some species of and pathovars of *Xanthomonas* are devastating pathogens. The cassava bacterial blight disease caused by *X. axonopodis* pathovar (pv) *manihotis* caused a near famine between 1972 and 1974 in all cassava-growing parts of Africa, Asia, Caribbeans and Oceania. It was particularly so in Nigeria. The problem was finally solved at the International Institute of Tropical Agriculture (IITA), Ibadan, where resistant varieties of cassava were bred and distributed to all cassava-growing countries of the world. I was part of the solution. My Ph.D work on the pathogen which was carried out at Imperial College of Science and Technology, University of London and the Centre Internacional de Agricultura Tropical. (CIAT) Cali,

Colombia, South America opened up several other areas for other Ph.D works all over the world from Nigeria through South America and the Caribbeans to Australia, Oceania, India, Sri Lanka, Malaysia and Indonesia. Other diseases caused by members of this genus include the black shank of cotton caused by *X. campestris* pv *malvacearum*, the citrus canker caused by *X. campestris* pv *citri*. Another xanthomonad, *Xylella fastidiosa*, a xylem-limited bacterium, which causes a range of economically important diseases has been found causing leaf scorch of coffee in Cote d'Ivoire and variegated chlorosis on citrus.

Streptomyces. All phytopathogenic actinomycetes except *Norcardia vaccine* belong to the genus *Streptomyces* (Locci 1994). *Streptomyces* species are Gram-positive, filamentous prokaryotes which are dispersed by spores and cause diseases of underground parts of a range of plant species (Loria *et al.* 1997). The most widespread and economically important of these diseases is the common potato scab caused by *S. scabies.* Badly scabbed potato tubers are not accepted by the consumer, constituting a loss to the farmer. *S. ipomoea* causes a fibrous root rot of sweet potato (*Ipomoea batatas*) resulting in the reduction of both yield and quality owing to distortions and necrotic patches on the storage root.

Viruses

There are over 700 known plant pathogenic viruses many of which cause catastrophic diseases and have wide host ranges. They have been classified into three families and 32 groups (Martelli 1992). These are based on morphology, the type of nucleic acid they contain (RNA or DNA), whether the nucleic acid is single- or double-stranded, whether it exists as a single unit or is divided, and the means of transmission. For example, furoviruses are fungus-transmitted, rod-shaped, single-stranded RNA viruses with divided, typically-bipartite genomes (Rush and Heidel 1995). Increasingly, serological and nucleic acid techniques are being used to establish the identity and relatedness of plant viruses.

When discussing plant viruses (with permission from Professor G.I. AtirI, our Virologist in the Department), one that easily comes to mind is the African Cassava Mosaic Virus (ACMV) causing the African Cassava Mosaic Virus Disease (ACMD). It is present in almost 80% of all cassava plants in Africa, although the percentage is being drastically reduced through efforts made at IITA, Ibadan. In unimproved cultures, more than 50% (conservative estimate) yield may be recorded. The disease is transmitted by the white fly *Bemisia tabaci*. The East African cassava mosaic disease (EACMD) is different from the ACMD. In the late 1980's, an unusually severe form of the disease appeared in Uganda. This was shown to have resulted from double infection by a recombinant virus from ACMV and EACMV and is termed UGV (Ugandan virus) which has spread to core Uganda, and large parts of Kenya, Tanzania, Sudan and the Democratic Republic of Congo.

Nematodes

These are a very important group of plant-parasitic microorganisms causing crop losses directly by their parasitic activities on the plants they infect and also indirectly by acting as vectors for plant viruses. Fawole (2009), during his inaugural lecture dwelt on these "small microorganisms with great impact", I do not wish to repeat his lecture here.

Oomycota

These were once regarded as fungi but a number of their features such as absence of chitin from their cell walls, their predominantly diploid karyotype and their biflagellate zoospores differentiate them from toxic fungi (Alexopolous *et al.* 2005). Owing to nucleic acid and protein sequence data, it is now known that organisms belonging to this phylum are more closely related to the golden brown algae. Members have evolved a parasitic lifestyle independently of true fungi.

The Oomycota contains many destructive plant pathogens such as the *Phytophthora, Pythium* and *Peronospora. Phytophthora megasperma* is responsible for causing black

pod disease of cocoa which prevents about 95% of our cocoa beans from being produced and exported annually. *Phytophthora infestans* is known as the fungus responsible for the Irish famine of the 1840s in which over one million Irish Nationals died of starvation and one and a half million others emigrated to the USA, Canada, Australia, etc. The catastrophe of the Irish potato famine initiated investigation on plant disease and may be thought of as the key event that led to the establishment of plant pathology as a scientific discipline (Strange 2003).

Algae

Cephaleuros virescens has been associated with disease symptoms in over 280 species and cultivars of higher plants some of which are *Persea americana* (avocado pear), *Cola nitida, Cola acuminate* (Kola), *Citrus* species (oranges and relatives) and several horticultural plants. The alga infects stems and leaves causing stem cracking, tissue discoloration beneath the bark and the presence of orange, velvet-like growth on stems and leaves. Other species of algae, which have been implicated in the causation of plant diseases are found in the genera *Chlorochytrium*, *Rhodochytrium* and *Phyllosiphon*.

Plasmodiophoromycota

These are a group of soil protozoa that use to be known as slime moulds. Some members are important as plant pathogens. *Plasmodiophora brassicae* causing club root disease of bassicas (cabbage, lettuce, etc) is a particularly highly damaging pathogen. *Spongospora subterranea* f. sp. *subterranea* is the causal agent of powdery scab of potato which is different from the one caused by *S. scabies.* Another species in the family Plamodiophoraceae is *Polymyxa* which causes more damage to plants indirectly than directly in that they live as vectors for viral diseases e.g. *Polymyxa graminis* which transmits wheat mosaic virus and barley yellow mosaic virus.

Viroids

Viroids are small (246-375 nucleotides), single-stranded, covalently-closed, circular, unencapsulated RNA and are characterized by a highly base-paired, rod-like secondary structure (Diener 2001). Viroid etiology has been established for at least a dozen plant diseases, including such economically important disease agents as potato spindle viroid, citrus exocortis viroid and the viroid that causes the *cadang-cadang* (dying-dying) disease of coconut palms. The last-mentioned disease has destroyed over 30 million coconut trees in the Philippines (Harold and Randles 1991). A similar disease in Guam is caused by a closely related coconut "*tinangaja*" viroid. Since the 1980s, many coconut trees have died along the coast of West Africa from Rivers State through Western Nigeria on to Senegal. No research has been carried out on the cause of these except some sporadic information emanating from some obscure scientist or whoever, not backed up by funds from uninterested governments that dominate African countries.

Trypanosomatids

Many people will always associate trypanosomes with sleeping sickness in man and his animals but the ones under discussion are plant pathogenic protozoa. They are commonly found in the latex, phloem, fruit sap, seed albumen and nectar of many plant families (Camargo 1999). Not until 1976 were they recognized as the causal agents of serious plant diseases when they were connected with two important disorders in palms-coconut heart not and palm marchitez (*wilt*). It was found that *Phytomonas staheli* caused the dwarf diseases of coconut and immature African oil palms (Mc-Coy and Matinez-Lopez 1982). A wilt disease of coffee has been ascribed to *P. leptovasorum* which may kill plants within two months of its first appearance.

Phytoplasmas

Yellow diseases of plants have been recognized since the 1900s and were then thought to be caused by viruses although

viruses could not be consistently isolated from such symptomatic plants (Lee *et al.* 2000). It was in 1967 that Doi *et al.* showed that ultra-then sections of phloem vessels of plants affected by such diseases contained particles that resembled animal and human mycoplasmas and are actually bacteria. Since then, phytoplasmas have been associated with several hundreds of diseases of plants, one of which is the coconut lethal yellowing disease which has virtually destroyed the coconut industry in Ghana and Badagry in Nigeria. Phytoplasmas are often rounded with average diameters ranging from 200-800µm. Others, such as *Spiroplasma citri* causing the stubborn diseases of citrus are helical. They lack rigid cell walls and are surrounded by a single unit membrane. Disease symptoms are consistent with the disturbance of plant hormone balance. For instance, infected plants may be sterile, stunted or may develop witches broom symptoms owing to the release of auxiliary buds from apical dominance. Phytoplasmas require vectors for transmission and are normally spread by sap-sucking bags belonging to the families Cicadullidae and Fulgoridae (leaf and plant hoppers).

Parasitic Angiosperms

Striga hermonthica and *S. gesnerioides* are parasitic weeds found mostly on cereals such as sorghum, maize, and also on cowpea. Others include *Viscum album* (mistletoe) on several crops such as citrus, kola, etc. The losses due to this particular group of parasitic plants have not been quantified in Africa and, of course in Nigeria, due to non-funding of research activities by the government.

Abiotic Factors

It is not only microorganisms (biotic factors) that can induce disease conditions in plants. Abiotic factors which are non-living factors in the environment also induce disease conditions.

These are excessively high and excessively low temperatures, drought, flooding, lightning injuries, soil pH (excessively high and low), salinity, low or no fertility, etc.

These induce physiological diseases in plants. These diseases are easier to control than those caused by biotic factors. If drought is the cause of the disease condition, supply of water to the plants will change the situation for the better. Alternatively, if excessive water (flooding) is the cause of the disease condition, draining the water away alleviates the condition. For abiotic diseases therefore, once the problem is identified the solution will be easy.

Establishment of Infection

For diseases to occur, contact must be made between a susceptible host and a virulent pathogen in a favourable environment.

Spore Dispersal

During the favourable season (rainy season), spores of fungi and bacterial cells are either deposited in soil or blown by wind currents. In some cases, spores are delicately held on structures called conidiophores above the boundary layer of still air adjacent to the leaf surface to enhance their chances of being wafted away in air currents upon stem vibrations, leaf flutters, or in rain splash. In some other cases, spores are actively discharged by specific dispersal mechanisms that forcibly eject them away from the plant.

Contact with the Host

After successful dispersal, spores must come in contact with the host to cause disease. Spores are produced in large numbers to give them a chance to land a few spores on a suitable host. They must contain adequate reserves to tide them over until they are able to access host cell nutrients. Chemotactic responses to general and specific chemical signals and also to electric currents generated around plant roots and some sugars that are present on leaf surface stimulate germination of spores. After making contact with the host, spores attach themselves firmly and rapidly to the plant surface to prevent being blown off by wind or being washed off by rain splashes. Once firmly attached to the host

surface, stimulation of the spores to germinate comes from host exudates e.g. for *Fusarium solani*, germination is stimulated by flavonoids.

The next step is for the pathogen to enter the host plant to obtain nourishment. The fungus has the capacity to enter directly through the cuticle or to grow towards natural openings such as wounds, stomata, hydathodes, lenticels, nectarthodes, etc. Some fungi have a short period of germ tube growth along the leaf surface followed by a differentiation of the hyphal tip to form a swelling called appressorium (fig. 4). After maturation, the appressorium grows out a narrow hyphal thread or penetration peg downwards through the cuticle or wounds or the natural openings. Physical force plays a major role in the penetration of plant tissue in most pathogens.

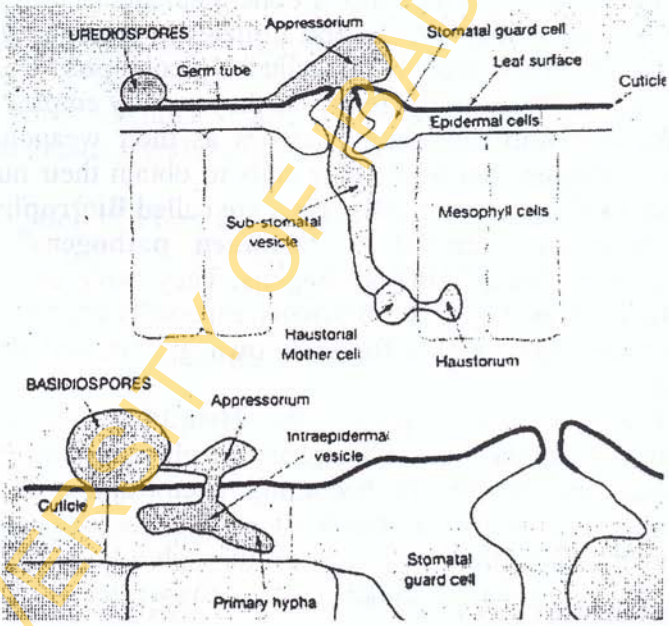

Fig. 4. Diagrammatic scheme showing penetration of plant tissue by a spore (Adapted from Dickinson (2005))

Sound explanations have been presented for the development of turgor pressure which is channeled into penetration pegs (Dickinson 2005). Enzymes also play a role in the penetration of plant tissue, especially the cell wall-degrading enzymes such as the exo- and endo-polygalacturonases (PGS) the pectate lyases (PLs) and the polymethylgalacturomases (PMG) and pectinmethylesterases (PME).

The main aim of the pathogen penetrating plant cuticles is to access nutrients which are bountiful within the plant cells. To do this, the pathogen must break through several barriers which it does by physical force and/or by the production of cell wall- degrading enzymes. Each pathogen has its own strategy of accessing these nutrients.

After penetration of host epidermis, pathogens invade host tissue and obtain the much-needed nutrients. There are two main ways by which this is done. Pathogens that kill their host's cells as they invade and utilize the nutrients that are released for their growth are called **Necrotrophs**. These are regarded as **"thug pathogens"**, as they usually employ toxins and/or cell wall-degrading enzymes as their weapons. The other pathogens tap into living cells to obtain their nutrients without killing the host cells. They are called **Biotrophs**.

These are regarded as **"conmen pathogens"** or in Nigerian parlance **"419" pathogens.** They avoid recognition by the plant as they divert nutrients, especially photosynthate-assimilates from plants for their own growth and development.

There is a third group, the **Hemibiotrophs** that go through an initial biotrophic phase in plants before causing damage and essentially becoming necrotrophic. A fourth group (still to be named) has been created whereby the fungal pathogen enters the plant, then go through a quiescent phase remaining latent until conditions are favourable for them to continue to develop.

Having breached the outer defenses of plants, the fungus will need to overcome chemical barriers before it can establish itself. Preformed antimicrobial compounds called

Phytoanticipins and **cell wall-degrading enzyme inhibitor proteins** (PGIPs) are often present in plants, and other compounds are released when plants are stressed or challenged by pathogens (phytoalexins and the oxidative burst), that present a formidable defense against invasion.

With the presence of these two formidable weapons, how do pathogens still penetrate plant tissues?

Overcoming Host Barriers

One approach for evading host defenses by pathogens is found in a number of post-harvest pathogens e.g. *Colletotrichum* and *Botrytis* species on landing on fruits, germinate and penetrate the epidermis. Upon making contact with chemical defenses of the plant, they enter a quiescent phase by producing dark, thick-walled appressoria and remain there until the chemical level in the plant goes down before becoming active to attack the plant. This occurs in fruits such as mangoes, etc. When the fruits ripen, the levels of antimicrobial chemicals go down and this allows the pathogen to attack and damage the fruits.

Detoxification of Plant Chemicals

The phytoanticipins and the PGIP levels decrease in fruits as they ripen making them very susceptible to the pathogen. In addition to detoxifying the phytoanticipins by enzyme degradation, the breakdown products induce a signal translocation mechanism that ends up in the suppression of the plant's defense system.

Avoidance of Recognition

This is the practice amongst biotrophic fungi which possess a more subtle approach to plant invasion. The key to their success is to avoid recognition (fig. 5) by the plant for as long as possible thus avoiding triggering any defense responses. This has been found in *Colletotrichum gloeosporioides, C. lindemuthianum* and *C. destructivum*.

Fig. 5. A ploy for avoiding recognition.

Establishment of Infection

Having penetrated the plant and having evaded the defenses, the pathogen must obtain nutrients from the host to enable it grow, develop and reproduce for the continuity of the race. Necrotrophic organisms will do this using cell wall-degrading enzymes and toxins to break down the cells whilst biotrophs produce specialist feeding structures (haustoria) to tap into host cells.

Roles of Toxins

Plant pathogenic fungi produce a wide range of toxins that may cause whole plant death or just subtle changes in gene expression. Fungal toxins can be divided into three groups: (i) *Host- selective* toxins such as produced by *Alternaria* which are toxic only to the hosts; (ii) *Non-selective* toxins which can cause damage to both host plants and other plant species not normally attacked by the producing pathogen. The third group contains toxins produced by plant pathogenic fungi that are not toxic to plants but are often fatal to animals and humans that consume the infected plant parts. Such fungi can

be found in the genus *Fusarium* e.g. *F. verticillioides* which causes stalk and ear rot of maize. It produces fumonisins with potential toxicity to man and his animals. Other species of *Fusarium* are known to produce trichothecene group of toxins that exhibit mutagenic, carcinogenic and teratogenic effects on man and other mammals.

Other toxins produced by non-pathogenic fungi which are not toxic to plants but adversely affect animals and man that consume the plant parts in which they are produced include *Aspergillus flavus* and *A. parasiticus* that produce aflatoxins B and G in maize grains, groundnuts and other cereals, and *A. ochraceus*, that produces ochratoxin, etc. These toxins have been implicated in the causation of liver cancer, etc. The ergot alkaloid—ergometrine, produced by *Claviceps purpurea* on grasses, causes ergotism in man and animals.

The toxins produced in plants affect them and cause cell death in the following ways:

(i) They affect host plasma membrane causing electrolyte leak (mainly potassium).
(ii) They affect mitochondria and uncouple photophosphorylation (light dependent conversion of ADP to ATP).
(iii) They decrease photosynthetic carbon dioxide fixation.

Bacterial Diseases

Of the 1600 known bacterial species, only about 100 cause diseases on plants. Most others live on leaf surfaces of plants or in the rhizosphere as saprobes. Like mycotoxin-producing fungi, there are some potentially harmful bacteria that are found on food causing food poisoning and death to humans through the consumption of food which bacteria such as *Escherichia coli* strain 0157 have contaminated.

Bacteria enter wounds or natural openings and colonize intercellular spaces and/or the xylem. Unlike fungi, bacteria are unable to penetrate the cuticle of plants. Most plant pathogenic bacteria are rod-shaped, Gram-negative and possess flagella.

Bacteria use a range of strategies for entry into plants. The leaf surfaces can support large populations of many bacterial species that arrive by air, rain splashes or carried by insects. Many of these can multiply on leaf surfaces to form micro-colonies or large aggregates which may passively enter and exit plants through stomata, lenticels, hydathodes, nectarthodes, wounds and particularly water droplets formed on leaf surfaces. In some cases such as the cassava bacterial disease caused by *Xanthomonas axonopodis* pv *manihotis* (Ikotun 1975, 1982) and bacterial brown spot of beans caused by *Pseudomonas syringae* pv *syringae* (Bender 1999), it has been shown that the probability of disease occurring is directly related to the external population size, indicating that a passive process of entry is not likely to cause disease.

In some xanthomonads, it appears that there is limited capacity to increase bacterial numbers on the leaf surface. There seems to be a more active process for invading the plant. The process is entry through the hydathodes which are the structures containing water pores located at leaf margins. Under suitable weather conditions, especially early in the morning, copious amounts of fluids are exuded through the hydathodes that collect as guttation drops around leaf margins. The bacteria move chemotactically towards these drops which are later drawn back in through the hydathodes carrying the bacterial cells into the vascular system. This appears to be the way through which *X. axonopodis* pv *manihotis* infects cassava plants (Ikotun 1975).

Role of Cell Wall-degrading Enzymes (CWDES) produced by Bacteria in Pathogenesis

Cell wall-degrading enzymes are produced by such necrotrophic bacteria as *Erwinia carotovora* and *E. chrysanthemi*, to soften and macerate plant tissues. Different cocktails are used by different pathogens. For *E. chrysanthemi,* pectinases (pectinmethl-esterases, pectate lyases and polygalacturonases), cellulases, four proteases and a phospholipase are produced (Dickinson 2005) *X. axonopodis* pv *manihotis* produces two polygalacturonases, an exopectate lyase and a

pectin methylesterase (Ikotun 1975). These break down the pectin in the middle lamella causing cell separation. Those bacteria that produce cellulases cause a loss of structural integrity of the cell walls making the tissue to collapse and soften, leaking fluid.

Role of Toxins

Toxins are a particularly important and potent weapon used by many bacteria. Toxins are either host specific or non-host specific. Those produced by *P. syringae* pathovars are generally non-host specific and not essential for pathogenicity but contribute to virulence. The best studied of bacterial toxins in plant pathology have been coronatine, tabtoxin phaseolotoxin, syringomycin and syringopeptin. Coronatine is produced by *P. syringae* pv *glycinea* on soyabean and *P. syringae* pv *tomato* on tomato. Tabtoxin is produced by *P. syringae* pv *tabaci* causing wildfire disease of tobacco. Phaseolotoxin is produced by *P. syringae* pv *phaseolicola* causing halo blight of legumes. Syringomycin and syringopeptin are produced by *P. syringae* pv *syringae,* lipopeptide phytotoxins that cause necrotic symptoms on many plants.

Roles of Hormones

Alterations in the levels of plant hormones are particularly important for those bacteria that cause uncontrolled proliferation of plant tissue resulting in gall and knot formations such as are produced by *Agrobacterium tumefaciens* which causes crown gall disease of tomatoes and some other plants. In the genera *Pseudomonas, Agrobacterium* and *Pantoae,* it is the production of the auxin indole-3-acetic acid (IAA) that is important for pathogenesis.

The Role of Extracellular Polysascharides (EPS)

EPSs are large polymers that are important for many phytopathogenic bacteria such as *Ralstonia solanacearum, Xanthomonas campestris* pv *campestris, X. axonopodis* pv *manihotis, Pseudomonas stewartii* and *Erwinia amylovora* as

capsules around the bacterial cells and as fluidal slimes released by the bacteria. The role of the EPS in bacteria appears to be two-fold. They provide a barrier to desiccation for bacteria on leaf surfaces and in the rhizosphere and provide a defense against toxic compounds and induced host defenses. Those bacteria that produce EPS are more virulent than those that do not. The consequence of EPS accumulation in the plant is the formation of large water-soaked lesions and/or blockage of the xylem vessels causing wilt of infected cassava plants (e.g. infected by *X. axonopodis* pv *manihotis*) (Ikotun 1982).

Roles of Cellulases and Xylanases

While older scientists did not implicate cellulose-degrading enzymes (cellulases) in cell wall degradation owing mainly to the fact that cellulose was not degraded in macerated cell walls, Walker *et al.* (1994) obtained evidence for the role of cellulose in the pathogenicity of *Erwinia carotovora* subsp. *carotovora* on potato. As with enzymes that depolymerize pectin, xylanases are also capable of killing plant cells (Ishii 1998).

Degradation of Lignin

Lignin is a complex component of the cell walls. Lignin is deposited after the cell has matured and signals death of the cell. The regulation of enzymes that degrade lignin and their role in pathogenesis has not attracted much attention. The best studied are those produced by white rot fungi *Phanerochaete chrysosporium* and *Trametes versicolor*. Dutton and Evans (1996) suggested that in white rot fungi, oxalate acts as a potential electron donor for lignin-peroxidase reduction and chelates manganese. Schultz and Nicholas (2000) suggested that phenolics present in lignin and heartwood extractives act as free radical scavengers and may retard white rot fungi, whereas these fungi rapidly colonise angiosperm sapwood as it has relatively low free phenolic content.

Role of Proteases

Despite the fact that proteins make up to 15% of cell walls of some plants, proteases (enzymes that degrade proteins) produced by pathogens and their roles in pathogenesis or virulence factors have received little attention.

Subverting Host Metabolism

Many of the symptoms of plant disease can be attributed to degrading enzymes or toxins but others are more indicative of hormone imbalance and these are usually associated with biotrophic rather than necrotrophic pathogens. Enhanced concentrations of auxins and cytokinins have been demonstrated in several instances in which hypertrophy is a symptom, as has been shown for *Agrobacterium tumefaciens* which causes tumorous growth in a wide range of plants. *A. manihoti* has been reported to cause stem galls on cassava in Colombia (Anon 1995). Stunting, in some instances, has been attributed to reduced concentrations of gibberellins while in others enhanced concentrations of abscissic acid may be responsible. Ethylene has many effects on plants which also have been associated with infection such as epinasty and abscission of organs, chlorosis and necrosis as well as the promotion of certain defense responses.

With those tiny, almost invisible microorganisms possessing such formidable array of weapons in their arsenal, it is certain that they can win any battle against any opponent. That is why I call pathogens, "David", in the battle against plants, which deserve the name "Goliath", because of their size. When all these weapons fashioned against plants are unleashed, does the plant wait to weather the storm? **No!, the plant hits back!**

Resistance Mechanisms in Plants

Just as pathogens evolved to colonize living plants, so also have plants developed means to prevent or tolerate their presence. Because plants cannot move to escape these challenges, they have developed many diverse and unique strategies.

Most plants are resistant to most microorganisms and generally, it is only the specialist organisms that have evolved the capacity to overcome plant defenses, hence many pathogens have narrow host ranges. Two levels of resistance have been defined:

***Non-Host Resistance*:** Where the entire plant species or genus is resistant and therefore not a host for that particular pathogen.

***Host Resistance*:** Where individuals within a species, have developed genetically-inherited ways of defending themselves against an organism that causes disease on other individuals within that plant species.

Mechanisms of resistance in plants are of two categories: **Passive** (constitutive) and **Active** (induced). Passive mechanisms involve both structural elements such as the cuticle and root border cells and preformed antimicrobial chemical compounds within the plant termed phytoanticipins. These form the initial layers of protection against microbial attack. Should these be breached, plants have a number of active or inducible defense mechanisms, which include the hypersensitive response (local plant cell death), and induction of specific gene expression within the plant including genes involved in cell wall strengthening and/or repair, structural defenses, genes for biosynthesis of additional antimicrobial compounds, and localized induction of genes encoding hydrolytic enzymes and other defense related proteins.

In addition to the localized induction of defense responses, there are mechanisms that induce resistance in other parts of the plant through systemic signals such as systemic acquired resistance (SAR) and induced systemic resistance (ISR). Plants can also signal neigbouring plants through the production of volatile compounds to enhance resistance in these plants.

Preformed Defenses

(i) ***Structural Barriers*:** The first barrier that defends plants against microbes is the wax layer present on the cuticle of leaves and fruits (fig. 6). This forms a water-repellent surface that prevents formation of water droplets and continuous film of water on the leaf surface necessary for bacterial ingress and fungal spore germination. Surface hairs on leaves perform a similar function. The thick cuticle composed of cutin, cellulose and pectin in combination with tough walls on epidermal cells form an additional barrier to all pathogens apart from those fungi that have the pathogenicity factors for direct penetration, and vectors that are potentially carrying pathogens. This leaves natural openings such as stomata, lenticels, hydathodes, etc as the main weakness in structural defenses.

(ii) ***Root Border Cells*:** Root tips that are rapidly elongating as they move towards water and nutrients in soil are a major potential weakness in the structural defenses of plants. The rapid growth of newly synthesized tissue through an environment with high capacity to cause abrasive damage and is rich in potentially pathogenic microbes make them extremely vulnerable to attack. In the root border cells, inducible defenses such as programmed cell death of a few plant cells would be an effective defense since this would terminate root growth. Instead, root tips have developed a mechanism through which they surround themselves with large numbers of detached somatic cells called "border cells" that effectively guard root tips from attack by pathogens. Once detached from the root, the metabolic activity of these cells increases and gene expression undergoes a global switch to produce anthocyanins and antimicrobial chemicals. This, in combination with the production of a mucilage layer repels bacteria. The border cells also appear to produce chemical signals that attract fungal zoospores essentially acting as decoys so that the border cells become infected rather than root tips.

(iii) ***Phytoanticipins***: Chemical barriers in plants that have been generally classified as phytoanticipins or phytoalexins depending on whether they are preformed inhibitors (phytoanticipins) or synthesized *de novo* following pathogen attack.

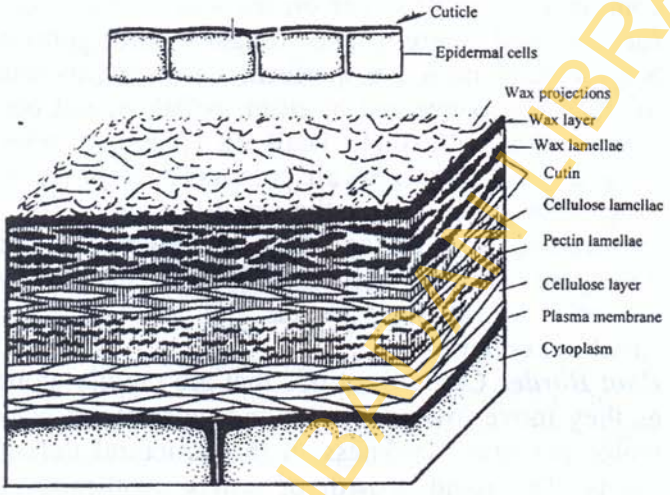

Fig. 6. Schematic representation of the structure and composition of the cuticle and cell wall of foliar epidermal cells (Adapted from Goodman, Kiraly, and Zaitlin (1967). *The Biochemistry and Physiology of Infections Plant Disease*. Van Nostrand, Princeton, New Jersey)

Induced Defenses

There is evidence that some defenses are induced by the physical presence of fungal spores on the leaf surface whilst others require the pathogen to penetrate the surface before induction. Induction may be in response to non-specific elicitors (the innate immune response that discriminates between self and non-self) or may follow the classical gene-for-gene resistance model which is the mechanism underlying host resistance. This is essentially a highly evolved form of inducible resistance in which the products of a specific resistance gene in the plant is involved in recognition of a specific elicitor from the pathogen.

Local Signals One of the first responses activated in many incompatible interactions prior to the induction of gene expression and protein synthesis is the production of ion fluxes reactive oxygen species (ROS) (also known as active oxygen species (AOS) or reactive oxygen intermediates (ROI)), production of nitric oxide (NO) and phosphorylation cascades. These are recognized as important signaling mechanisms in many organisms.

Programmed Cell Death One of the most visible responses of plants to pathogen attack is the hypersensitive response (HR), which is the localized death of plant cells. This is a temporally and spatially coordinated mechanism to limit the amount of host tissue sacrificed to the pathogen and one that restricts the ingress of biotrophic pathogens that require living cells as their source of nutrients. Programmed cell death (PCD) is a general term used to describe the induced cell suicide that occurs in plants in reaction to invasion by foreign bodies, especially pathogen organisms.

Induced Structural Barriers

Plants have a number of induced structural defenses. The first of these is cytoskeleton-based to send off attack from potential pathogens on the surface. These mechanisms have been shown to occur with cowpea rusts and rice blast diseases. In the case of rice blast caused by *Magnaporthe grisea*, it has been shown that these responses arise from surface contact and not as a wound response to penetration.

Phytoalexins: There has been much debate whether or not phytoalexins and phytoanticipins play a significant role in plant defense. Many genes in the pathways to produce phytoalexins such as flavonoids have been shown to be regulated in a coordinated fashion in response to pathogen invasion indicative of an important role in defense.

Antimicrobial compounds produced by plants include a wide array of low-molecular weight secondary metabolites

(not essential for basic metabolic processes in plants) and these act against a broad range of pathogens. They include terpenoid derivatives (e.g. sesquiterpenes); saponins; aliphatic acid derivatives; phenolics and phenylpropanoids (e.g. isoflavonoid); nitrogen-containing organic compounds (e.g. alkaloids) and sulphur-containing compounds including inorganic elemental sulphur compounds. Phytoalexins have been isolated and characterized from 31 plant families, mostly dicotyledonous but have also been isolated from monocotyledonous plants such as sorghum, maize, rice, onions, lilies, etc. To some extent, the chemical class of compounds produced is related to the plant family. For example isoflavonoids are found in Leguminosae but not in Solanaceae while the converse is true for sesquiterpenoid phytoalexins.

The following are a selection of phytoalexins and plants which synthesize them (table 2).

Table 2: Phytoalexins and Plants producing them

Phytoalexins	Plants producing them
Arjunolic acid, cyclooctosulphur	*Theobroma cacao* (cocoa)
Casidiol	*Capsicum* species (pepper)
Casbene	*Ricinus communis* (castor oil plant)
Demethylmedicarpin	*Arachis hypogae* (groundnut)
Glyceollin	*Glycine soja* (Soybean)
Monilactone A	*Oryza sativa* (rice)

Source: Dickinson (2005)

Phytoalexins are non-specific biocides affecting a wide range of microorganisms including bacteria, fungi, nematodes, higher animals and plants, and some of those from legumes have the added property of being oestrogenic.

***Pathogenesis-related Proteins*:** In addition to cell death, strengthening of structural defenses and production of antimicrobial phytoalexins, various novel proteins are induced during pathogen attack known collectively as

"pathogenesis-related" (PR) proteins. These proteins are expressed at low levels in healthy plants but certain isozymes are induced during pathogen attack both locally and systematically and there is evidence that specific sequences in the promoter regions of these genes are important for induction. The biochemical role for chitinases produced by plants in response to microbial invasion is to degrade chitin in the cell wall of the invading fungus effectively disrupting fungal cells and blunting the attack. Lectins have been shown to bind chitins in fungal cells as well.

***Systemic Resistance Mechanisms*:** Apart from activation of defense genes by pathogen's invasion, it has been found that the formation of a hypersensitive response either in systemic resistance response or Systemic acquired resistance (SAR) is one from of inducible resistance that is activated throughout the whole plant, and this resistance has some similarities with animal immune responses in that it is long-lasting and can be boosted by repeat infections. Unlike animal immunity, it can be effective against organisms other than the one used to stimulate the initial response, conferring broad spectrum resistance.

***Communal Resistance*:** In addition to systemic signaling within plants and the induction of defenses, recent evidence has shown that plants can communicate with their neighbors and induce the activation of defense genes in them. Volatile signals such as methyl jasmonate and methyl salicylate are produced from insect infested and pathogen-infected plants which are able to induce defense genes in the neighbors.

With all these defense structures and mechanisms in the arsenal of Goliath (the plant) why does it still need to be protected?, you would ask.

Yes, plants have many means of hitting back at their tormentor, the pathogen (David) and in fact, any living or non-living thing that lands on the plant would be hit. However, if plants always produce those structures and all

those chemicals, they would not be palatable, soft, plant and sweet enough to meet the demands of the taste buds of man and animals. Man and animals carefully select the particular species and varieties of plants they like and leave the rest that do not meet the standard of what they want. Unfortunately, it is every variety of plants that appeals to man's taste buds that the pathogenic microorganisms also desire. They almost do not touch the wild undomesticated plants with the same severity of disease and damage that they visit on the domesticated plants. Man selects the good, sweet-tasting varieties of plants and pathogenic microorganisms evolve and develop the capability to attack and destroy them. For instance, it took the IITA several years to breed and select the cultivars (varieties) of cassava which were high yielding, disease resistant and acceptable to farmers and in a few years, their yield potentials fell and one form of microbial attack or the other started to surface. This reminds me of the title of the inaugural lecture of the late Professor O.F. Esuruoso –"Battle without End on the Farm". Indeed, it is a great battle for the breeder to breed out plants with desirable qualities that are acceptable to the consumers and it is a great battle for the farmer to keep these varieties of plants out of the detection and destruction by pathogens.

These domesticated plants also posses the ability to produce the structures of defense and the antimicrobial chemicals but they produce them slowly and in small amounts that before they accumulate in the concentrations required to kill off the invader, the pathogen would have taken evasive steps to avoid or inactivate them, leaving the plants damaged and the farmer frustrated.

This is the reason why there is the great need for man's intervention to protect plants. The domesticated plants have been incapacitated through breeding that most of the weapons in their arsenal had been watered down (bred out) to give the qualities desired by man.

The Department of Crop Protection and Environmental Biology

Mr. Vice-Chancellor, Sir, Distinguished Ladies and Gentlemen, it was not for nothing that the name of the Department of Crop Protection and Environmental Biology was changed in 1992 from the old name of Agricultural Biology which was created in 1962. The old name did not spell out what exactly was being done in the Department, people did not know its vision and mission and students were afraid that they would be condemned to a teaching life in the secondary school after graduating from the Department. The elders of the Department then came together, knocked their egg-heads together to fashion out a name that spells out all that is done in the Department. In the Department, we study the environment of crop plants and develop ways of producing crops to fit well into that environment with minimal attempts to protect them.

Protection comes only when the crops are threatened by pests and diseases and all evasive actions are taken to prevent the onset of attack by these devilish living things. It is only as a last resort that pesticides are used. After all, the world is turning to organic agriculture in which inorganic and synthetic fertilizers and pesticides are not used.

Since the change of name, there has been an annual increase in student enrolment at the undergraduate and the postgraduate levels. Our students are offered choice jobs at the National Institutes of Agricultural Research, where they prove their mettle. They occupy positions ranging from Executive Directors down to young Research Officers. None of them teaches except in the Universities nationwide and in some other countries where many of them have risen to become Professors. It is in the Department of Crop Protection and Environmental Biology that Man's interventions in the afflictions of plants are taught and studied.

Man's Interventions in the Afflictions of Plants

Despite the onslaught, plants have survived with the various mechanisms they deploy to defend themselves. However, these mechanisms can fail, especially when a virulent

pathogen evolves and lands on preferred varieties of crop plants that are not loaded with the chemicals and structures that can protect them. They therefore need to be protected, hence the need for man's interventions. The following are ways by which man can intervene to prevent or reduce the amount of disease and damage caused by pathogenic micro-organisms on plants—by following the principles of disease control all the time, man can reduce the possibility of infection. The principles are:

(i) Avoidance of the Pathogen

- If crops are planted at periods when or in areas where infective units of a particular pathogen are absent, rare or ineffective, contact between the crop and the pathogen can be avoided.
- A shift in planting data may provide conditions that are more favourable for the growth and development of the crop than the pathogen.
- The use of disease-free planting materials such as seeds, cuttings, etc, is another way of avoiding the pathogen.

(ii) Exclusion of the Pathogen

- Pathogens can be excluded from seeds, corms, tubers, setts, etc, by exposure to dry heat, poisonous gasses or chemicals.
- Pathogens can be excluded from planting materials through inspection followed by certification that they are disease-free.
- Pathogens can be excluded from planting materials and from crop plants by the elimination of the insect vector.
- By the restriction of importation of planting materials through Plant Quarantine Services to prevent the introduction of exotic pathogens from entering the country.

(iii) Eradication of the Pathogen

This principle involves the reduction of the pathogen population or the complete destruction of the inocula at the source. The source could be the planting materials or the soil in which it is already established.

These methods include:

- Crop rotations with non-hosts to starve out pathogens with narrow host range.
- Rogueing (removal) and proper disposal of infected plants or plant parts from the field to prevent spread of the disease.
- Soil treatment by heat at 121^0C for 15 minutes or more.
- Fumigation of soil and planting material with volatile gasses or chemicals.
- Field sanitation which involves the elimination of infected crop residue by burning or deep burial in soil.
- Biological control using live agents such as bacteria, fungi, actinomycetes to challenge and overcome pathogens.
- Use of botanicals (plants with known biological activity) to reduce the activities of the pathogen.
- Seed dressing of planting materials before planting.
- Use of dry and moist heat to eradicate deep-seated and surface-borne inocula in planting materials.

(iv) ***Use of Host Plant Resistance*****:** This is highly painstaking and expensive on the part of the scientist/breeder and it takes several years to breed a resistant plant. This method has great merits and has helped overcome disease and problems of low yield. A plant that has benefitted most from this exercise in Africa is cassava which was bred at the International Institute of Tropical Agriculture (IITA) against diseases such as the African Cassava mosaic virus diseases, the cassava bacterial blight disease and root and tuber rot.

(v) ***Chemical Control Method*****:** This is a method that is sparingly used nowadays because of the acute toxicity of the chemicals and their residual effect on the environment. It is now used as a last resort when the disease is assuming epidemic proportions. Application of the pesticide can be for protective purposes (prophylactic), in which it is applied before the pathogen lands on the surface. When it lands, it is immediately killed by the pesticide. Application of the pesticide can also be curative or therapeutic, in which case the pesticide inactivates or eradicates the pathogen even when it has penetrated the plant.

(vi) ***Integrated Disease Control Method*****:** No one single method of disease control is capable of a once-and-for-all control of plant diseases. An integrated method uses all the methods listed above from avoidance of the pathogen through exclusion, eradication, use of host resistance and chemical control to achieve the desired goal.

(vii) ***Artificial Induction of Systemic Resistance*****:** Many of the defense systems in plants can be activated or induced in advance of the advent of pathogens by challenging them with live microorganisms that are not pathogenic to the plant. The presence of alien propagules on the plant triggers off the activation of the defense systems of the plant. This also occurs when inocula of the pathogen are heat-killed or when an avirulent strain of the pathogen is sprayed on the host plant.

Chemicals (non-pesticides) can also be used to induce systemic resistance in plants and make them resist invasion by pathogens during an epidemic. For instance, two compounds - chloroisonicotinamide and probenazole have been used to induce resistance of rice against blast diseases caused by *Magnaporthe grisea.* Several other non-pesticide chemicals have been used to confer protection on many commercially-grown crop plants in developed countries. Chemical agents with special ability to induce resistance in plants will be preferable over biological agents in the relative ease of application as foliar sprays or as soil drench. The

protection conferred may be effective against a broad spectrum of pathogens and may be quite long lasting with a possibility of being extended in duration through a growing season after a second (booster) induction.

Conclusion and Recommendations

Mr. Vice-Chancellor, Sir, these are the many ways by which man intervenes to alleviate the afflictions of plants. The need to intervene arises from the fact that plants are primary producers of food providing a rich source of nutrients not only for the human population but also for other living organisms that require fixed carbon compounds for sustenance. It is therefore not surprising that microorganisms have fashioned such formidable weapons against plants to satisfy their own needs. Despite the onslaught from microorganisms, plants have survived as they have done since the third day of creation, and when all their efforts to protect themselves fail, man intervenes to alleviate their afflictions.

The decision to intervene must be backed by information gathered over the years by diligent research workers. In Nigeria, only a semblance of research work is done. This is because research is poorly funded.

Research is a powerful tool for development. Research suffered seriously under the military governments that extended from 1976 to 1979 and from 1984 to 1999 and the civilian government from 1999 to date. Research grants, if they were ever available (until recently) were few and far between. Yet, African governments (including the Nigerian government) have been goading developed countries for technology transfer. These countries invested a sizeable chunk of their GDP on technological development while African governments misappropriated theirs. Personally, I would not pass-on my hard-earned acquired technology to "never-do-wells". Any country that wants to develop technologically must be ready to set aside a sizeable chunk of its GDP for research and innovation. There are qualified scientists willing to work for long hours, but when you have a government that does not want to invest in research but wants

to depend on "technology transfer", there can be no progress in food production to feed the people.

In recent times (2007 to date), there have been increases in food prices sparking riots in countries all over the world, such as in African countries, Greece, Portugal, Spain, Eastern European countries and Asian countries. Had governments invested in research, there would have been some improvement in agricultural production in those protesting countries to prevent the food price protests.

Nigerian governments had been advised since the 1970s that diversification of the economy was best for the country and that agriculture as a renewable resource should not be ignored. The government ignored agriculture and chose to depend solely on oil. Since then our fortunes as a nation depend on fluctuations of oil prices in the world market. I am also adding my voice of supplication to government to bring agriculture back to limelight so that we can feed ourselves and improve the national GDP. Nigeria has no reason to be regarded as a poor nation. In fact, we should be ashamed of being called a poor nation.

Acknowledgments

All praise, honour and glory be to my living God who has preserved me through thick and thin, through real life-threatening dangers and has been with me to this day. He has finally set my feet upon solid rock. Where would I have been without you? O God, I thank you.

I am grateful to the University of Ibadan for giving me the great opportunity to benefit from the training programme of the Ford Foundation/University of Ibadan Staff Development Fellowship from 1972 to 1975. I sincerely thank the donors, the Ford Foundation for this great deed. I also thank the International Development Research Council (IDRC) of Canada for the award of a Travel/Study Fellowship tenable at the Centro International de Agriculture Tropical, Cali, Colombia for my field work on cassava pathology from 1973 to 1974. My gratitude also goes to the Federal Government of Nigeria for the award of an Undergraduate Scholarship which

made my days as a student comfortable. I also acknowledge the Scholarship given by the Western State Government for my studies at Higher School Levels in Government College, Ibadan in 1967 and 1968.

I greatly acknowledge my teachers with gratitude - Mr. J.O. Ale (1952-1955) and Messrs Mba, Esiobu, Ihuoma and Nkemakolam (1956-60) at the primary school level; the late Mr. D.J. Bullock, Mr. A.T.O. Odunsi, Mr. N.O. Oyetunji, Mr. V.O. Ninan, Rev. P.S. Samuel, Late Dr. Akintola and Professor Ayo Banjo at the secondary school level. Professor Ayo Banjo was my housemaster at Government College, Ibadan and my Hall Warden at Nnamdi Azikiwe Hall. These were the people that moulded me into what I am today before the finishings were put in place by the Late Professor T. Ajibola Taylor, Late Professor O.F. Esuruoso, Professor M.O. Adeniji, Professor J.K. Egunjobi, Professor (Mrs) O.A. Egunjobi, the late Dr R.H. Odihirin and Dr E. O. Osisanya, in the Department of Agricultural Biology. I can never forget Professor Ajibola Taylor who ensured that I became a member of staff of the Department of Agricultural Biology. His role in the award of the University of Ibadan/Ford Foundation Staff Development Fellowship can never be forgotten. May God bless his family and rest his soul in peace. The late Professor O. F. Esuruoso and Professor M.O. Adeniji, I remember specially because their stories stimulated my interest in plant pathology and they taught me so well that by the time I came to be on my own, my feet had been well planted by them. It is of interest to note that Professor Adeniji's eldest brother, Mr. D.O. Adeniji was our Education Officer at Makurdi, Benue Province, where I had my primary education. He taught me how to write and greatly encouraged me to become the scholar that I am today. May his soul rest in peace. I must not forget my dear Old Supervisor at Imperial College of Science and Technology, Professor R.K.S. Wood. At the age of 57, I was his 57th Ph.D student from 1972 to 1975. He was a respecter of persons and intellect, whether black or white. He was a great scholar. He is 93 years old and

still going strong as an Emeritus Professor of plant pathology. I pray to be like him.

I also acknowledge the Director General and Staff of IITA where I had my first taste of International Scientific life after my first degree and in 1988-1990, as a visiting scientist. I must greatly acknowledge, Dr Sang Ki Hahn, the then Director of the Roots and Tuber Improvement Program at IITA, who took special interest in me and my work, Drs Eugene Terry, Rob Asiedu, Alfred Dixon, D.S.O. Osiru, M. Bokanga, John Otoo and Mrs Lawal who were my colleagues at IITA. I acknowledge Dr Ranajit Bandyopadhyay with whom I collaborate at IITA.

I acknowledge my colleagues at the primary, secondary and tertiary levels who taught me the spirit of competition and fairplay, both of which are the guiding principles of my life today.

I acknowledge my colleagues in the Department of Crop Protection and Environmental Biology with whom I have come a long way. I always remember our jokes, our contribution to the development of the Department and how we rose in unison against detractors and denigrators. The standards we are upholding in the Department, I am sure, will please our founding fathers. I also acknowledge the staff of the Dean's Office, Faculty of Agriculture and Forestry where I humbly served as Dean from 2002 to 2004. It was during my tenure that we went back to reoccupy Ileogbo land. It was also during that tenure that we made the highest sales from our vegetables and maize, generating funds for the Faculty.

I acknowledge the staff and students of our Great Nnamdi Azikiwe Hall where I served as an assistant Warden for twelve years and two years as Warden. It was my service there that catapulted me into the position of the first full-time Dean of Students (1991 – 1996) at a time there was turmoil in the University. Greatest Zikites assisted me then and made my two-term tenure (six years) a success. Greatest Zikites!!!!!

I must not fail to mention the Sports Council where I was a discus and javelin thrower, a hockey goalkeeper and an occasional cricketer, as a student. As soon as I returned from London in 1975, I was appointed an Honorary Hockey coach,

a position I held until 1986. I was pleasantly surprised when I was appointed as Chairman of the Sports Council in 2004. I thank the staff for the maximum cooperation I had from them. We were able to resuscitate sports, which was not well-funded then, to what it is today. We resuscitated the Bilateral games with the University of Ghana, Legon. We now participate fully at the Nigerian Universities Games competitions (NUGA) and the West African Universities Games (WAUG). Students were happy at all these opportu-nities to travel out and know all the other institutions in the country and in the West African sub-region.

I thank all my colleagues and friends in the Staff Club where I have been an active darts player and served for three years as the Treasurer. I remember my "Rods" and "members of the Judiciary" who always took their "risk" at drinking and making jokes with me.

I remember my late father Pa Israel Ojo Ikotun who ensured that I was well educated. He invested his all in the education of his children and, thank God that we did not let him down. I thank my Mother who could not be here today due to advanced age. She is a mother in Israel, a mother in deed. To my siblings who are scattered to the four winds in search of economic prosperity, I wish you all well.

I sincerely acknowledge all my students past and present, who made me the academician that I am today. I have supervised 44 B.Sc., 53 M.Sc, 3 M. Phil and 23 Ph.D holders at the University of Ibadan in my 35 years of service; 5 M.Sc and 1 M.phil at Njala University, Sierra Leone. I am currently supervising three M.Sc and eight Ph.D students at Ibadan and four Ph.D students at Njala University. Of my Ph.D students, eight are already Professors, amongst whom were one Deputy Vice-Chancellor, four Deans and one Head of Department. There are also three Readers. Of those who did not come to the University system, one is a Deputy Provost of a College of Education, three are Directors at Institutes of Agricultural Research, one is a Senior Programmes Officer at African Union Headquarters in Addis Ababa. Unfortunately, one died. Their names, dates and positions are given in table 3.

Table 3: Former Ph.D students successfully supervised

S/N	NAMES	YEAR	
1	E.E. Odigie	1980-1984	CRO,NIFOR Benin-City
2	J.A. Durojaiye	1983-1987	Deputy Provost, FCE, Oyo
3	E.N. Ekpo (Nee Obaze)	1984-1988	Director, FRIN, Ibadan
4	S.A. Adebitan	1986-1991	Professor, ATBU, Bauchi
5	A.N. Amusa	1988-1991	Professor, and HOD, OOU, Ago-Iwoye
6	E.N. Osai	1988-1992	Reader, UNICAL
7	A.F. Sanusi	1988-1995	UK (Private)
8	K. Obisesan	1988-1994	RIP
9	Marie Awo Ayodele	1989-1999	Scientist, PQS and IITA
10	A.A. Adebanjo	1990-1994	Formerly Dean at OOU, Now Professor and Dean, NOUN, Lagos.
11	C.N. Fokunang	1991-1996	International, U.K.
12	O.A. Enikuomehin	1991-1996	Professor, UNAAB
13	Janet.Edeme (Nee Udoh)	1992-1997	Snr. Prog. Officer, AU Hq Addis, Ababa.
14	A.T. Adekunle	1992-1997	Reader, UNIBEN
15	T.O. Adejumo	1993-1997	Reader, AAU, Akungba
16	S.A. Bankole	1994-1999	Professor and Dean. OOU, Ago Iwoye
17	A.E. Oluma	1983-1986	Prof. UAM, Makurdi
18	R.O. Bayewu	1983-1987	Director, FRIN, Ibadan
19	A.C. Amadioha	1983-1987	Professor. UNIPORT
20	M. Nwufo	1981-1985	Professor.& DVC, FUTO
21	Pat. Obilo	2003-2008	Lecturer 1, FUTO
22	J. Atehnkeng	2003-2008	Scientist, IITA
23	M. Twizeyimana	2003-2008	Post Doctoral Fellow. University of Illinois, USA.

I am proud of them all.

Current Ph.D Students

1. Folake Akinbode – 2007
2. Joy Nwogwugwu – 2008
3. Kemi Adeogun – 2010
4. S.A. Lawal – 2011
5. S.A. Kazeem – 2011
6. Tawakalitu George – 2011
7. T. Isadeha – 2011

Finally, "*eegun nla ni i kehin igbale*", my dear wife, my great pillar of support, my adviser, my fashion designer, my financier, my cook and at night, my bouncer and comforter. I can never thank you enough. Thank you, thank you, thank you. This same bouncer gave me three lovely children that make me happy at the thought of them, at sighting them and at interacting with them. I thank God for blessing me with you.

Mr. Vice-Chancellor, Sir, Distinguished Ladies and Gentlemen, this is my humble story. I thank you for your attention!

PLATES

Leaf blight of mango.

Fruit drop of mango.

Chlorosis of an ornamental palm.

Parasitic angiosperm on Croton.

Leaf blight of tobacco.

African Cassava mosaic disease.

Fungal leaf spot of cassava.

Cassava anthracnose disease.

Defoliation of cassava plant.

Leaf blight of plantain plant.

Wilt of leaf of cassava.

A harvest of mushrooms.

Mushrooms causing stem rot.

Frog-eye spot of tobacco.

Tip die-back of pawpaw.

Water-soaked leaf spot.

A healthy ornamental plant.

Leaf blight of an ornamental plant.

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