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Defence Mechanisms in Plants Against Invading Plant Pathogenic Microbes in Nigeria

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Abstract

Plant cells consist of cell wall, cell membranes, and cytoplasm, which contains the nucleus and various organelles and all the substances for which the plant pathogens have as their targets. These pathogens attack plants because during their evolutionary development they have acquired the ability to live off the substances manufactured by the host plants, and some of the pathogens depend on these substances for their development and survival. Many substances are contained in the protoplast of the plant cells, and if pathogens are to gain access to them they must first overcome the physical barrier presented by the host cuticle and/or cell walls. Plants defend themselves against invading plant pathogens by a combination of weapons from two major barriers: structural characteristics that act as physical barriers and inhibit the pathogen from gaining entrance and also from spreading through the plant. Secondly through biochemical reactions that take place in the cells and tissues of the host plant and produce substances that are either toxic to the pathogen or create conditions that inhibit growth of the pathogen in the plant cells and thus defend plants. These actions against invading pathogen are controlled directly or indirectly by genetic materials (gene) of the host plants.

Keywords: mechanism, pathogen, genetic materials, cell walls, defense mechinism.

INTRODUCTION

Pathogens though hostile as invaders that attack plants, farm produce, etc. but in general terms, they are like any other organisms, simply trying to survive and develop however, they are living at the expense of a host organisms otherwise by means of parasitism (Alberts *et al*., 2002). Therefore, disease causing microorganisms, such as bacteria, fungi, and viruses, found commonly in irrigation water, run-off water from farms, etc. refers to pathogens. Other examples of pathogens include; Prions, Protozoan, Viroid and Human parasites though this group of pathogen majorly affects the human body.

The stomata of plants regulate gas exchange and water transpiration in response to changing environmental conditions. A recent work reveals that stomata also have an important role in host defense. In this issue of Cell, Melotto *et al*. (2006) show that stomata close upon detection of potential microbial pathogens to prevent the infection of the leaf interior. Moreover, pathogenic bacteria have evolved strategies to suppress the closure of stomata. This is through the production of phytotoxin, a chemical called coronatine, to force the pores back open. For bacteria, entry is crucial to causing disease and probably survival. They could die if left lingering on the surface.

HOW PLANTS ARE INFECTED BY PATHOGENS

The "infection process" can be divided into three phases: pre-entry**,** entry and colonization. It encompasses the germination or multiplication of an infective propagule in or on a potential host through to the establishment of a parasitic relationship between the pathogen and the host. The process of infection is influenced by properties of the pathogen, the host and the external environment. If any of the stages of the infection process is inhibited by any of these factors, the pathogen will not cause disease in the host. While some parasites colonize the outside of the plant (ecto-parasites), pathogens may also enter the host plant by penetration, through a natural opening (like a stomatal pore) or via a wound (Melotto *et al*., 2006). The symptoms of the diseases produced by these

pathogens result from the disruption of respiration, photosynthesis, translocation of nutrients, transpiration, and other aspects of growth and development.

DEFENCE MECHANISM IN PLANT AGAINST INVADING PATHOGENS

Plants have developed a variety of strategies to discourage or kill attackers. The first line of defense in plants is an intact and impenetrable barrier composed of bark and a waxy cuticle and /or cell wall (Zeyen *et al*., 2002; Micali *et al*., 2011). Both protect plants against pathogens. A plant's exterior protection can be compromised by mechanical damage, which may provide an entry point for pathogens. If the first line of defense is breached, the plant must resort to a different set of defense mechanisms, such as toxins and enzymes (http://www.boundless.com/ Boundless Learning).

PLANT'S IMMUNE SYSTEMS

The plant immune system consists of two interconnected tiers of receptors, one outside and one inside the cell. Both systems sense the intruder, respond to the intrusion and optionally signal to the rest of the plant and sometimes to neighboring plants that the intruder is present. The two systems detect different types of pathogen molecules and classes of plant receptor proteins (Dangl *et al*., 2013).

The first tier is primarily governed by pattern recognition receptors (PRR) that are activated by recognition of evolutionarily conserved pathogen or microbial–associated molecular patterns (PAMPs or MAMPs). Activation of PRRs leads to intracellular signaling, transcriptional reprogramming, and biosynthesis of a complex output response that limits colonization. The system is known as PAMP-Triggered Immunity (PTI) (Jones and Dangl, 2006; Dodds and Rathjen, 2010). The second tier (again, primarily), effector-triggered immunity (ETI), consists of another set of receptors, the nucleotide-binding LRRs (NLRs). They operate within the cell, encoded by R genes. The presence of specific pathogen "effectors" activates specific NLR proteins that limit pathogen proliferation (Dangl *et al*., 2013).

Receptor responses include ion channel gating, oxidative burst, cellular redox changes, or protein kinase cascades that directly activate cellular changes (such as cell wall reinforcement or antimicrobial production), or activate changes in gene expression that then elevate other defensive responses (Dangl *et al*., 2013). Plant immune systems show some mechanistic similarities with the immune systems of insects and mammals, but also exhibit many plant-specific characteristics. Plants can sense the presence of pathogens and the effects of infection via different mechanisms than animals.

PAMP-triggered immunity

PAMP-Triggered Immunity (PTI) is often a plant's first inducible response (Jones and Dangl, 2006). According to Numberger *et al*. (2004) immune-eliciting PAMPs include bacterial flagellin or lipopolysaccharides, or fungal chitin. Much less widely conserved molecules that inhabit multiple pathogen genera are classified as MAMPs by some researchers. The defenses induced by MAMP perception are sufficient to repel most pathogens. However, pathogen effector proteins are adapted to suppress basal defenses such as PTI (Dodds and Rathjen, 2010).

Effector triggered immunity

Effector Triggered Immunity (ETI) is activated by the presence of pathogen effectors (Jones and Dangl, 2006). According to Numberger *et al*. (2004) the ETI immune response is reliant on R- genes, and is activated by specific pathogen strains. As with PTI, many specific examples of apparent ETI violate common PTI/ETI definitions (Thomma *et al*., 2011). Most plant immune systems carry a repertoire of 100-600 different R genes that mediate resistance to various virus, bacteria, fungus, oomycete and nematode pathogens and insects. Plants ETI often cause an apoptotic hypersensitive response.

In addition to PTI and ETI, plant defenses can be activated by the sensing of damage-associated compounds (DAMP), such as portions of the plant cell wall released during pathogenic infection. Many receptors for MAMPs, effectors and DAMPs have been discovered. Effectors are often detected by NLRs, while MAMPs and DAMPs are often detected by transmembrane receptor-kinases that carry LRR or LysM extracellular domains (Dodds and Rathjen, 2010).

R genes and R proteins

Plants have evolved R genes (resistance genes) whose products allow recognition of specific pathogen effectors, either through direct binding or by recognition of the effector's alteration of a host protein (Jones and Dangl, 2006). These virulence factors drove co-evolution of plant resistant genes to combat the pathogens' avirulent (Avr) genes. Many R genes encode NB-LRR proteins (nucleotide-binding/leucine-rich repeat domains, also known as NLR proteins or STAND proteins, among other names).

R gene products control a broad set of disease resistance responses whose induction is often sufficient to stop further pathogen growth/spread. Each plant genome contains a few hundred apparent R genes. R genes usually confer specificity for particular pathogen strains. As first noted by Harold Flor in his mid-20th century formulation of the gene-for-gene relationship, the plant R gene and the pathogen Avr gene must have matched specificity for that R gene to confer resistance, suggesting a receptor/ligand interaction for Avr and R genes (Numberger *et al*., 2004). Alternatively, an effector can modify its host cellular target (or a molecular decoy of that target) activating an NLR associated with the target or decoy.

Plant breeders frequently rely on R genes to obtain useful resistance, although the durability of this resistance can vary by pathogen, pathogen effector and R gene. The presence of an R gene can place significant selective pressure on the pathogen to alter or delete the corresponding avirulence/effector gene. Some R genes show evidence of stability over millions of years while other R genes, especially those that occur in small

clusters of similar genes, can evolve new pathogen specificities over much shorter intervals (Friedman and Baker, 2007).

Effector biology

Effectors are central to microbes' pathogenic or symbiotic potential and of microscopic plant-colonizing animals such as nematodes (Lindeberg *et al*. 2012). Effectors typically are proteins that are delivered mostly outside the microbe and into the host cell (Hewezi and Baum, 2013). Effectors manipulate cell physiology and development. As such, effectors offer examples of co-evolution (example: a fungal protein that functions outside of the fungus but inside of plant cells has evolved to take on plant-specific functions). Pathogen host range is determined, among other things, by the presence of appropriate effectors that allow colonization of a particular host (Dodds and Rathjen, 2010). Pathogen-derived effectors are a powerful tool to identify host functions that are important in disease. Apparently most effectors function to manipulate host physiology to allow disease to occur. Well-studied bacterial plant pathogens typically express a few dozen effectors, often delivered into the host by a Type III secretion apparatus (Lindeberg *et al*. 2012). Fungal, oomycete and nematode plant pathogens apparently express a few hundred effectors (Hewezi and Baum, 2013).

So-called "core" effectors are defined operationally by their wide distribution across the population of a particular pathogen and their substantial contribution to pathogen virulence. Genomics can be used to identify core effectors, which can then functionally define new R alleles, which can serve as breeding targets (Dangl *et al*., 2013).

RNA silencing and systemic acquired resistance elicited by prior infections

Against viruses, plants often induce pathogen-specific gene silencing mechanisms mediated by RNA interference. This is a simple form of adaptive immunity (Ding and Voinnet, 2007). Plant immune systems also can respond to an initial infection in one part of the plant by physiologically elevating the capacity for a successful defense response in other parts. Such responses include systemic acquired resistance, largely mediated by

salicylic acid-dependent pathways, and induced systemic resistance, largely mediated by jasmonic acid-dependent pathways (Spoel and Dong, 2012).

Species-level resistance

In a small number of cases, plant genes are effective against an entire pathogen species, even though that species that is pathogenic on other genotypes of that host species. Examples include barley MLO against powdery mildew, wheat Lr34 against leaf rust and wheat Yr36 against stripe rust. An array of mechanisms for this type of resistance may exist depending on the particular gene and plant-pathogen combination. Other reasons for effective plant immunity can include a lack of co-adaptation (the pathogen and/or plant lack multiple mechanisms needed for colonization and growth within that host species), or a particularly effective suite of pre-formed defenses.

Signaling mechanisms

Perception of pathogen presence

Plant defense signaling is activated by pathogen-detecting receptors (Dodds and Rathjen, 2010). The activated receptors frequently elicit reactive oxygen and nitric oxide production, calcium, potassium and proton ion fluxes, altered levels of salicylic acid and other hormones and activation of MAP kinases and other specific protein kinases (Numberger *et al*., 2004). These events in turn typically lead to the modification of proteins that control gene transcription, and the activation of defense-associated gene expression.

DETOXIFICATION OF PATHOGEN TOXINS BY PLANTS

In at least some of the diseases in which the pathogen produces a toxin, resistance to disease is apparently the same as resistance to the toxin (Van Etten *et al*. 1989). Detoxification of at least some toxins, e.g., HC toxin and pyricularin, produced by the fungi Cochliobolus carbonum and Magnaporthe grisea, respectively, is known to occur in plants and may play a role in disease resistance. Some of these toxins appear to be metabolized more rapidly by resistant varieties or are combined with other substances

and form less toxic or nontoxic compounds (Van Etten *et al*. 1989). The amount of the nontoxic compound formed is often proportional to the disease resistance of the variety. Resistant plants and nonhosts are not affected by the specific toxins produced by Cochliobolus, Periconia, and Alternaria, but it is not yet known whether the selective action of these toxins depends on the presence of receptor sites in susceptible but not in resistant varieties, on detoxification of the toxins in resistant plants, or on some other mechanism.

DEFENCES/IMMUNIZATION OF PLANTS AGAINST PATHOGENS

Defense through Plantibodies

In humans and animals, defenses against pathogens are often activated by natural or artificial immunization, i.e., by a subminimal natural infection with the pathogen or by an artificial injection of pathogen proteins and other antigenic substances (Latunde-Dada and Lukas, 2001). Both events result in the production of antibodies against the pathogen and, thereby, in subsequent prolonged protection (immunity) of the human or animal from infection by any later attacks of the pathogen. Plants, of course, do not have an immune system like that of humans and animals, i.e., they do not produce antibodies. In the early 1990s, however, transgenic plants were produced that were genetically engineered to incorporate in their genome, and to express foreign genes, such as mouse genes that produce antibodies against certain plant pathogens (Honee, 1999). Such antibodies, encoded by animal genes but produced in and by the plant, are called plantibodies. It has already been shown that transgenic plants producing plantibodies against coat proteins of viruses, e.g., artichoke mottle crinkle virus, to which they are susceptible, can defend themselves and show some resistance to infection by these viruses (De Jaeger *et al*. 2000). It is expected that, in the future, this type of plant immunization will yield dividends by expressing animal antibody genes in plants that will produce antibodies directed against specific essential proteins of the pathogen, such as viral coat proteins and replicase or movement proteins, and fungal and bacterial

enzymes of attack. Whole antibodies or fragments of antibodies can be expressed easily in plants following integration of a transgene into the plant genome, or by transient expression of the gene using viral vectors, infiltration of the gene by Agrobacterium, or through biolistics. Plants such as tobacco, potato, and pea have been shown to be good producers of antibody for pharmaceutical purposes. Plants have been shown to produce functional antibodies that can be used to increase the resistance of plants against specific pathogens (Hutcheson, 1998). So far, functional plantibodies, produced by plants against specific plant pathogens, that have been shown to increase the resistance of the host plant to that pathogen include the following: Plantibodies to tobacco mosaic virus in tobacco decreased infectivity of the virus by 90%; to beet necrotic yellow vein virus, also in tobacco, provides a partial protection against the virus in the early stages of infection and against development of symptoms later on; to stolbur phytoplasma and to corn stunt spiroplasma, also in tobacco, which remained free from infection for more than two months (De Jaeger *et al*. 2000). However, attempts to engineer plantibody-mediated resistance to plant parasitic nematodes have been unsuccessful so far. Generally, however, the expression of complete or fragment antibodies in plants has been only partially effective or mostly ineffective so far. Plantibody-derived resistance appears mostly as a delay in the development of disease and, barring a breakthrough, it does not appear that it will become an effective means of plant disease control in the near future (De Jaeger *et al*. 2000).

Resistance through Prior Exposure to Mutants of Reduced Pathogenicity

Inoculation of avocado fruit with a genetically engineered, reduced pathogenic strain of the anthracnose fungus *Colletotrichum gloeosporioides*, which does produce an appressorium, results in delayed decay of the fruit (Yakoby *et al*. 2002). Such an inoculation brings about increased levels of biochemical defense indicators, such as H+-ATPase activity, reactive oxygen species, phenylalanine ammonia lyase, the natural antioxidant phenol epicatechin, the antifungal compound diene, and eventual fruit

resistance with delay of fruit decay. However, inoculation of fruit with a similar mutant strain that does not produce an appressorium causes no activation of early signaling events and no fruit resistance. It would appear that initiation of the early signaling events that affect fruit resistance depends on the ability of the pathogen to interact with the fruit and initiate its defense mechanisms during appressorium formation (Yakoby *et al*. 2002).

SYSTEMIC ACQUIRED RESISTANCE

Induction of Plant Defenses by Artificial Inoculation with Microbes or by Treatment with Chemicals

As discussed earlier, plants do not naturally produce antibodies against their pathogens, and most of their biochemical defenses are inactive until they are mobilized by some signal transmitted from an attacking pathogen. It has been known for many years, however, that plants develop a generalized resistance in response to infection by a pathogen or to treatment with certain natural or synthetic chemical compounds. Induced resistance is at first localized around the point of plant necrosis caused by infection by the pathogen or by the chemical, and it is then called local acquired resistance. Subsequently, resistance spreads systemically and develops in distal, untreated parts of the plant and is called systemic acquired resistance. It is known now that several chemical compounds, e.g., salicylic acid, arachidonic acid, and 2,6-dichloroisonicotinic acid, may induce localized and systemic resistance in plants at levels not causing tissue necrosis (Kessman *et al*. 1994). Jasmonic acid is another type of compound, derived primarily from oxidation of fatty acids, which leads to systemic acquired resistance, often in cooperation with salicylic acid and ethylene, leading to the production of defenses. Local acquired resistance is induced, for example, in a 1 to 2mm zone around local lesions caused by tobacco mosaic virus on hyper- sensitive tobacco varieties and probably in other host–pathogen combinations. Local acquired resistance results in near absence of lesions immediately next to the existing lesion and in smaller and fewer local lesions developing farther out from the existing local lesions when inoculations are made at least 2–3 days

after the primary infection (Leong *et al*. 2002). Local acquired resistance may play a role in natural infections by limiting the number and size of lesions per leaf unit area. Systemic acquired resistance acts nonspecifically throughout the plant and reduces the severity of disease caused by all classes of pathogens, including normally virulent ones. It has been observed in many dicot and monocot plants, but has been studied most in cucurbits, solanaceous plants, legumes, and gramineous plants following infection with appropriate fungi, bacteria, and viruses. Systemic acquired resistance is certainly produced in plants following expression of the hypersensitive response. Localized infections of young plants, e.g., cucumber with a fungus (*Colletotrichum lagenarium*), a bacterium (*Pseudomonas lachrymans*), or a virus (tobacco necrosis virus), lead within a few days' time to broad-spectrum, systemic acquired resistance to at least 13 diseases caused by fungi, bacteria, and viruses. A single inducing infection protects cucumber from all pathogens tested for 4 to 6 weeks; when a second, booster inoculation is made 2 to 3 weeks after the primary infection, the plant acquires season-long resistance to all tested pathogens. The degree of systemic acquired resistance seems to correlate well with the number of lesions produced on the induced leaf until a saturation point is reached. Systemic acquired resistance, however, cannot be induced after the onset of flowering and fruiting in the host plant.

DEFENSE THROUGH GENETICALLY ENGINEERING DISEASE-RESISTANT PLANTS

With Plant-Derived Genes

The number of plant genes for resistance (R genes) that have been isolated is increasing rapidly. The first plant gene for resistance to be isolated was the Hml gene of corn in 1992, which codes for an enzyme that inactivates the HC toxin produced by the leaf spot fungus Cochliobolus carbonum (Honee, 1999). In 1993, the Pto gene of tomato was isolated; this gene encodes a protein kinase involved in signal transduction and confers resistance to strains of the bacterium *P. syringae* pv. *tomato* that carry the avirulence gene avrPto. In

1994, four additional plant genes for resistance were isolated: the Arabidopsis RPS2 gene, which confers resistance to the strains of P. syringae pv. tomato and P. syringae pv. maculicola that carry the avirulence gene avrRpt2; the tobacco N gene, which confers resistance to tobacco mosaic virus; the tomato Cf9 gene, which confers resistance to the races of the fungus Cladosporium fulvum that carry the avirulence gene avr9; and the flax L6 gene, which confers resistance to certain races of the rust fungus Melampsora lini carrying the avirulence gene avr6. The last five plant resistance genes are triggered into action by the corresponding avirulence genes of the pathogen, the products of which serve as signals that elicit the hypersensitive response in the host plant (Luderer and Joosten, 2001). Several more plant resistance genes have since been isolated. Some of these genes appear to provide plant resistance to pathogens expressing one or the other of two unrelated Avr genes of the pathogen. It is expected that these and many other R genes, which are likely to be isolated in the years to come, will be used extensively in genetically engineering transgenic plants that will be resistant to many of the races of the pathogens that affect these plants. In addition to these specific plant genes, several other plant genes encoding enzymes or other proteins (PR proteins) found widely among plants have been shown to confer resistance to transgenic plants in which they are expressed (DeWit, 1992). For example, tobacco plants transformed with a chitinase gene from bean became resistant to infection by the soilborne fungus Rhizoctonia solani but not to infection by the oomycete Pythium aphaniderma- tum, the cell walls of which lack chitin.

DEFENSE THROUGH RNA SILENCING BY PATHOGEN-DERIVED GENES

RNA silencing is a type of gene regulation that in plants, serves as an antiviral defense. RNA silencing is based on targeting specific sequences of RNA and degrading them. RNA silencing occurs in a broad range of eukaryotic organisms, including plants, fungi, and animals. While plants use RNA silencing to defend themselves against viruses, the viruses, in turn, encode proteins by which they attempt to suppress the silencing of their RNA (Balmori *et al*. 2002). The consensus is that RNA silencing is one of the many

interconnected pathways for RNA surveillance and cell defense. RNA silencing was first observed in transgenic plants transformed with viral genes providing "pathogen-derived resistance." It was noticed then that sense orientation genes in the transgenic plant interfered with the expression of both the transgenes themselves and related endogenous genes of the plant. Because of the concurrent suppression of both genes, RNA silencing was at first called "cosuppression." RNA silencing is due to a process that occurs after transcription (posttranscriptional gene silencing) of the RNA and involves targeted RNA degradation. Clues of its existence came from the discovery that plants carrying viral transgenes were resistant to related strains of the virus that replicate in the cytoplasm, which meant that silencing occurs in the cytoplasm rather than the nucleus.

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