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Review Article

Host-Pathogen Interaction and Roles in Plant Disease Management

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ABSTRACT

The ability to detect and mount a defense response to potential pathogenic microorganisms has been paramount to the evolution and developmental success of modern day plants. Plants are often exploited as a source of food and shelter by a wide range of parasites including viruses, bacteria, fungi, nematodes, insects and even other plants. During the long history of co-evolution between host and pathogens, plant immune response has culminated in a highly defense system that is able to resist potential attack by microbial pathogens. Plant immune system is composed of strong surveillance systems, which recognize microbial molecules using signal transduction pathways that pose physiological responses that ultimately allow plants to switch from the growth and development mode into a defense mode, rejecting most potentially harmful microbes.

Host-pathogen interaction is the way in which a pathogen (virus, bacteria, prion, fungus and viroid) interacts with its host. Pathogens adapt to the host changes, and find alternative ways to survive and infect a host.

Interactions between disease resistance (*R*) genes in plants and their corresponding pathogen Avirulence *(Avr)* genes are the key determinants of whether a plant is susceptible or immune to a pathogen attack.

Plants resist diseases caused by the pathogens using passive constitutional plant resistance elements such as waxy layers, cuticles, cork layers, cell wall polymers, lenticels, stomas and trichomes, and active defence mechanism which involve accumulation of Phytoalexins, phenolics, ethylene, hydrolytic enzymes, peroxidases, and numerous stress-related proteins.

Keywords: Phytoalexins; Phenolics; Ethylene; Hydrolytic enzymes

INTRODUCTION

Understanding of the internal mechanisms by which plants defend themselves from pathogen under natural conditions would facilitate the development of effective means for the protection of cultivated plants from disease. Presently disease management is largely based on the use of hazardous fungicides, bactericides and insecticides for either direct or indirect disease management. Problems with the resistance of pathogens to classical pesticides, the hazardous natures of the products to the environment, human and animal health strongly necessitates searching for new safer means of disease management approach. Among the recent management approaches that help to overcome such a problem are the use practical knowledge of genetics of plant-pathogen interaction and its application using Genetic Engineering (GE) [1]. Sequencing of entire plant genomes, systematic plant transcriptome profiling and comprehensive genetic dissection of immune pathways in model plants such as Arabidopsis thaliana and rice has significantly enhanced understanding of the mechanisms underlying microbial infection and plant immunity now be turned into new tools to engineer durable, broad spectrum plant disease management techniques that involves boosting plant recognition of infection, silencing essential pathogen genes, Mining R genes and molecular manipulation of immune system using elicitors. The main objective of this seminar is to make an over view of molecular approaches of plant-pathogen interactions and defense mechanisms against pathogens help to insight and compile its setting alternative plant diseases management approaches.

LITERATURE OF REVIEW

Molecular concepts of plant immunity

Plant immunity is a state of defense against infectious pathogens, Bacteria, Fungi, Virus, Nematode and others. It is the capacity of a plant to prevent or withstand biological attack by pathogens. Progress in the understanding of the molecular complexity of the innate immune system in plants has advanced considerably in recent decades.

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Plants have evolved two strategies to detect pathogens (Figure 1). On the external face of the host cell, conserved microbial elicitors called Pathogen Associated Molecular Patterns (PAMPs) are recognized by receptor proteins called Pattern Recognition Receptors (PRRs). Plants also respond to endogenous molecules released by pathogen invasion, such as cell wall or cuticular fragments called Damage-Associated Molecular Patterns MAMPs or DAMPs using their PRRs. The most important MAMPs are microbial cell wall structures, such as chitin (fungi), beta-glucans (oomycetes), lipopolysaccharide or peptidoglycan (bacteria), or microbial proteins, such as bacterial flagellin or Elongation factor Thermo-unstable (EF-Tu, part of the cellular protein translation machinery) [2].

Pathogens are not only sensed by the plant immune system *via* their own molecular components (exogenous elicitors) but usually also provoke the release of plant-derived signals characteristic of infection, called endogenous elicitors or Damage-Associated Molecular Patterns (DAMPs). Typical examples of DAMPs are fragments of cell wall components generated during attack by microbial cell wall-degrading enzymes, e.g., Oligogalacturonides (OGs, derived from pectin) or cutin monomers, and intracellular plant components released into the extracellular space upon cell lysis. DAMPs are sensed by cell surface-resident PRRs and activate typical PTI (PAMP-Triggered Immunity) signaling and defense responses. PRRs are typically single-span trans-membrane or membrane-anchored proteins with structurally diverse extracellular domains, such as Leucine-Rich Repeat (LRR). Stimulation of PRRs leads to PAMP-Triggered Immunity (PTI). Typical PTI signaling and defense responses are, for example, the depolarization of the plasma membrane, an increase in the cytosolic concentration of the secondary messenger Ca^{2+}, activation of different protein kinases, production of Reactive Oxygen Species (ROS), induction of defense-related genes, cell wall fortifications, and production of antimicrobial enzymes and secondary metabolites as well as defense-related plant hormones (Figure 2) [3]. The second class of perception involves recognition by intracellular receptors of pathogen virulence molecules called effectors; this recognition induces Effector-Triggered Immunity (ETI). The conserved nature and broad occurrence of MAMPs in different microbes, sensing of MAMPs to activate PTI enables the host to detect and efficiently control a wide range of microbes. Adapted pathogens employ effectors to dampen PTI and to modulate host cell metabolism for their own needs. Effectors are any molecules secreted by pathogens which modify host protein to establish their growth. Effectors can have structural role as in extra haustorial molecules of fungi, Nutrient leakage role as in *P. syringae* HopM effector protein; Pathogenicity such as inhibition of PTI.

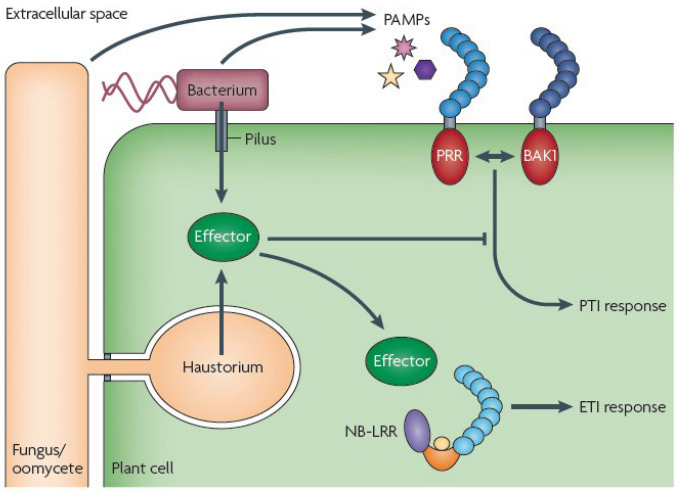

Figure 1: The principal components of plant immunity.

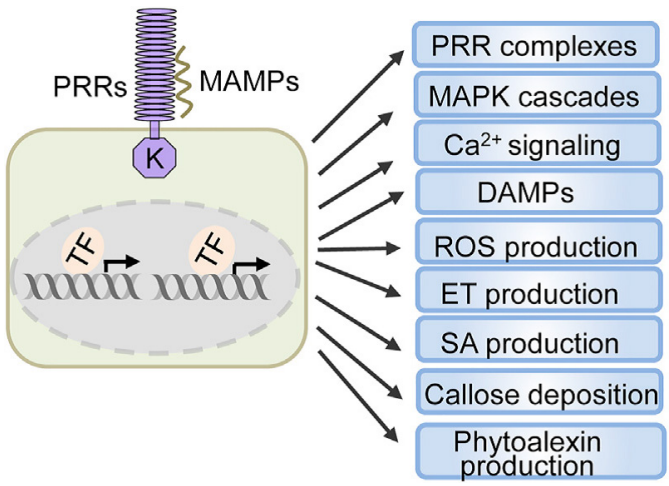

Figure 2: PAMP-Triggered Immunity (PTI) signalling and outputs.

Plants, in turn, evolved immune sensors, called Resistance (R) proteins that detect microbial effectors directly or indirectly by monitoring the effector targets, thus turning effectors into Avirulence factors which trigger Effector-Triggered Immunity (ETI), which is similar defense responses like PTI but usually develops faster and in a stronger fashion and is mostly accompanied by an HR.

Molecular concepts of host pathogen interaction

Co-evolution between host and pathogens enables plants to developed complex interaction system to defend potential attack by microbial pathogens. The appropriate response of plant emerges from the perception of an extracellular signal and its transduction between and within plant cells.

Evidence shows that gene-for-gene interactions in the perception of pathogenic invasions and development of resistance in plants involve different molecular and hormonal transduction pathways, which are still poorly understood.

To establish disease, pathogens need to interact and neutralize different obstacles on their way into the plant tissue. The first interaction barrier is the plant cell surface such as cuticle. Penetration could occur through natural openings like stomata, through wounds, or by direct penetration using enzymes and/or mechanical forces. Once pathogens gain access by penetrating the plant cuticle, they face the second obstacle, the plant cell wall. After cell wall penetration, the pathogen is separated from plant cytoplasm just by the plasma membrane. Plasma membranes contain specialized proteins, extracellular surface receptors, which are involved in the detection of Pathogen Associated Molecular Patterns (PAMPs) to trigger immune responses [4,5]. On pathogen recognition, regulatory genes initiate a multi component defense response whose elements are activated in a highly controlled temporal and spatial manner. Molecular plant-pathogen interaction involves perception, recognition and response.

Perception

Plant pathogens have ability to sense plants by various root exudates, volatile secretions, mucin like glycoproteins, surface-binding proteins or passively transported by splash water to leaf surfaces. Once encysted on plant surfaces, signaling triggers germination of spores to initiate infection. Germ tubes then enter directly into the intercellular spaces through wound openings on leaves, or form swellings that allow penetration between epidermal cells on root surfaces [6].

Attachment of the pathogen to host

Attachment of the pathogen takes place through adhesion of spores by various mechanisms such as tips mucilaginous substances that consisting of mixtures of water- insoluble polysaccharides glycoproteins, lipids and fibriller materials when moistened, become sticky and help the pathogen to adhere to the plant, others which do not require free water for infection, adhesion is using enzyme cutinase released from spore , which makes the plants and spore areas of attachment more hydrophilic and cements the spore to the plant surface. In some other cases, propagule adhesion requires on the spot synthesis of new glycoproteins to facilitate attachment [7].

Spore germination and perception of host surface

Spore germination is mainly triggered by contact with the host surface, hydration and adsorption of low molecular weight ionic materials from the host surface, and availability of nutrients (Figure 3). The perception of signals from plant surfaces by pathogenic fungi seems to be result of signaling pathways mediated by Cyclic Adenosine Monophosphate (MAPK), which is implicated in the regulating the development of infection related phenomenon in many different fungi.

Host pathogen recognition

Gene-for-Gene interaction: The concept of gene for gene hypothesis was first developed by Flor in 1956 based on his studies of host pathogen interaction in flax, for rust caused by *Malampsora lini*.

The evolution of secreted effector proteins by plant pathogens ultimately led to the acquisition of plant proteins that specifically recognize these bacterial, fungal, and viral effectors this association involving the recognition of effectors within the plant cell has been characterized genetically as gene-for-gene resistance (Figure 4). Host pathogen interaction is said to compatible when a pathogen population successfully reproduce and maintain itself in a parasitic mode on any member of a plant species the opposite is incompatible which is the absence of basic compatibility.

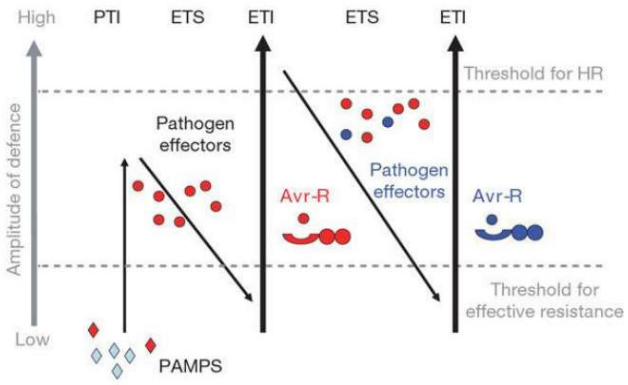

Figure 3: A zigzag model illustrates the quantitative output of the plant immune system.

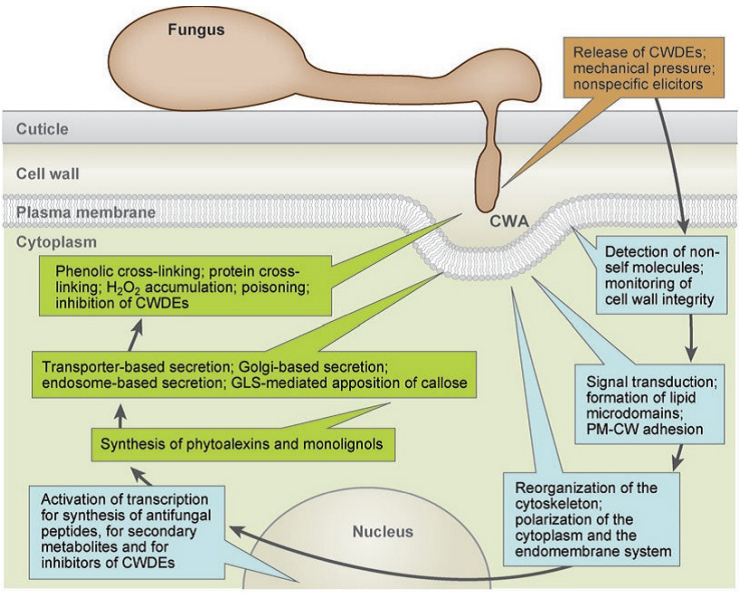

Figure 4: A zigzag model illustrates the quantitative output of the plant immune system. **Note:** (■) Plant defense; (■) Plant recognition and signaling; (■) Fungal activity.

Signaling and response: Signal transduction is a series of events between the receptor where the signal is perceived and the nucleus where transcription of specific gene is initiated, repressed, or regulated. Pathogen-Associated Molecular Patterns (PAMPs) of invading pathogens have been found to be potential signals to activate the plant innate immunity. These PAMP signals are perceived by the plant Pattern Recognition Receptors (PRRs), and the PAMP–PRR signaling complex activates the plant immune system [8, 9]. The plant immune system uses several second messengers to encode information generated by the PAMPs and deliver the information downstream of PRRs to proteins which decode/interpret signals and initiate defense gene expression. Plant hormones such as Salicylic Acid (SA), Jasmonates (JA), Ethylene (ET), Abscisic Acid (ABA), Auxin (AUX), Cytokinin (CK), Gibberellin (GA), and Brassinosteroi (BR) have been reported to play an important role in intercellular and systemic signaling systems triggering expression of various defense-responsive genes. According to Walters the traditional model of signal transduction is based on a linear transfer of a single signal, with the perception of a single stimulus leading to a single response which is: Elicitor-Receptor-Second messenger-Gene activation–Phenotypic expression (Defence) (Figure 5).

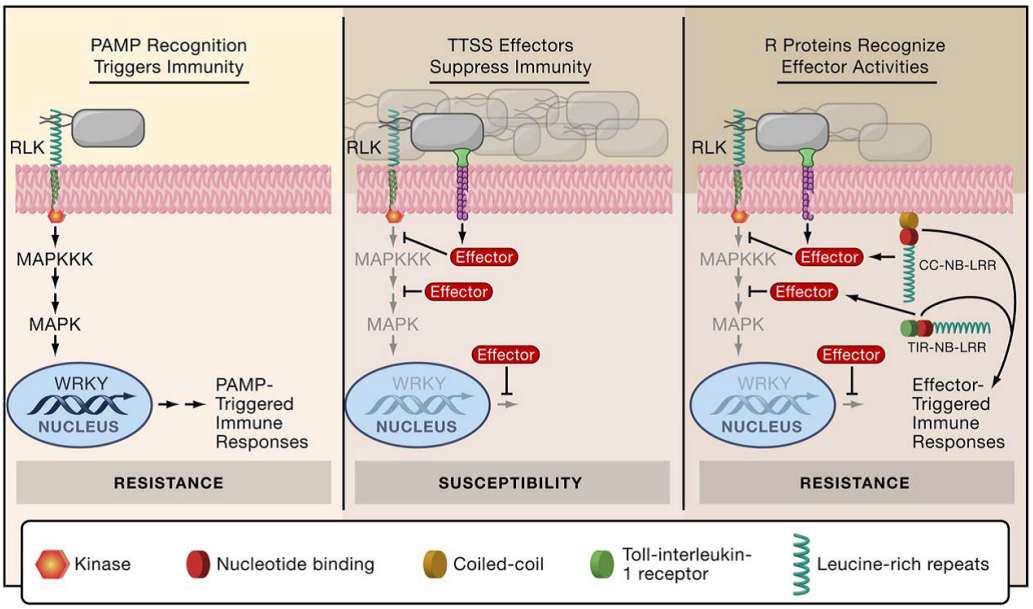

Figure 5: Model for the gene-gene interaction in Plants. Left to right, recognition of pathogen-associated molecular patterns (such as bacterial flagellin) by extracellular Receptor-Like Kinases (RLKs) Promptly Triggers basal Immunity (PTI), Pathogenic bacteria use effector proteins that suppress basal immune responses, Plant resistance proteins recognize effector activity and restore resistance through Effector-Triggered Immune (ETI) responses.

Plant virus interaction

Most plant viruses have RNA genomes that contain imperfect regulatory stem-loops and are copied into complementary dsRNA Replication Intermediates (RIs) by virus-encoded RNA-dependent RNA polymerases (RDRs). As an intrinsic feature of virus genome expres¬sion and replication, this dsRNA can be designated a Virus-Associated Molecular Pattern (VAMP; a form of PAMP). VAMPs are generically recognized by Dicer-like (DCL) enzymes, which then produce Virus-Derived Small Interfering RNAs (vsiRNAs) that, upon load¬ing into Argonaute (AGO) proteins, promote antiviral defence through RNA silencing. RNA gene silencing, also termed RNA interference (RNAi), which acts as a basal defense mechanism against viruses, is one of the main plant immune responses against viral pathogens [10, 11].

Molecular bases of plant diseases resistance

Resistance is an inhibition of any stage of the development of reproductive cycle of the pathogen in time or space, or of the pathogenic effects in the host.

Recently Fonseca explained resistance is said to be Non Host Resistance (NHR) when expressed by all plant species against most potential pathogens and classified it as pre-invasive and post-invasive NHR. Pre-invasive NHR involves physical, chemical and metabolical barriers that block pathogen entry or penetration assisted with PTI which is generally effective against non-host pathogens with no visible symptom expression as the pathogen fails to enter or penetrate plant tissue [12, 13]. Pre- invasive NHR mainly involves pre-invasive proteins such as PEN1, PEN2, PEN3, GCN4, NOG1-2, CaM7 and BRT1 for resistance against pathogens. Post-invasive NHR typically involves incompatible interaction involving special defence proteins such as PAD4, ELO1, ELO2, SQS, NOG1-1, SAG101, ProDH, PING4, PING5, RPL12, RPL19, GOX and SGR during an incompatible interaction of gene-for-gene mediated resistance (also known as *R* gene mediated resistance) that leads to activation of strong plant defense signaling cascade known as Effector Triggered Immunity (ETI).

Molecular concepts of resistance in the host plant

Infectious plant diseases are the result of the interaction of at least two organisms, the host plant and the pathogen. The properties of each of these two organisms are governed by their genetic material, the DNA, which is organized in numerous segments making up the genes. It thus appears that, under favorable environmental conditions, susceptibility and resistance in each host–pathogen combination is predetermined by the genetic material of the host and of the pathogen.

Passive and induced diseases resistance in plants

Punja, Agrios, Walters explained that plant disease resistance to pathogens can be due to either passive or induced defence responses.

Passive (pre-infection) plant defence responses

Passive (pre-infectious) or constitutional resistance exists regardless of whether the plant was attacked by pathogens or not. This can be naturally existing physical barriers, such as waxy layers, cuticles, cork layers, cell wall polymers, lenticels, stomas and trichomes, chemical barrier such as pH, phytoanticipins and several constitutive antimicrobial components, for instance simple phenols, saponins and tannins, which have potent antifungal activity, and thus help prevent colonisation of the tissue .

Active (post-infectious) or induced plant defence responses

Interactions between plants and pathogen can lead to either to a compatible response or incompatible response. In incompatible interactions, infection by virus, bacteria or fungi will elicit a set of rapid or localized induced and delayed or systemic induced responses in host cells. Rapid or Local response includes membrane permeability loss, Oxidative burst, Fortification cell wall, HR ,Phytoalexin histological changes such as in cell wall composition such callose deposition, Tylose formation and Corky layers that inhibit pathogen penetration while delayed or systemic induced responses are PR-Proteins, Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR).

Local Acquired Resistance (LAR) and Systemic Acquired Resistance (SAR)

Resistance triggered in the plant during its life time is known as Acquired Resistance. This can be Local Acquired Resistance (LAR) confined to few cells or tissues or Systemic Acquired Resistance (SAR) having been moved throughout the plant. The first controlled laboratory study of SAR performed by Ross, demonstrated that inoculation of a single leaf of tobacco with Tobacco Mosaic Virus (TMV) reduced the severity of subsequent infections on other leaves 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 and resistance spreads systemically and develops in distal, untreated parts of the plant and is called Systemic Acquired Resistance (SAR). 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 lesion soon after primary infection limiting the number and size of lesions per leaf unit area. Systemic acquired resistance confers long-lasting protection, acts non-specifically throughout the plant and reduces the severity of disease caused by all classes of pathogens fungi, bacteria, and viruses. Systemic acquired resistance is produced in plants following expression of the hypersensitive response and correlate with the number of lesions produced on the induced leaf until a saturation point is reached.

Systemic Acquired Resistance (SAR) signal transduction pathways

Plants are defended against pathogens by constitutive and inducible barriers. Induced resistance is expressed locally at the site of infection as well as in uninfected parts of infected plants.

SAR is accumulated after pathogen infection, binding NPR1 activate induction of Pathogenesis Related Genes (*PR*). SAR is characterized by the increased expression of a large number of Pathogenesis-Related genes (*PR* genes), in both local and systemic tissues [14].

SA is a plant phenolic compound synthesized by plants to regulate defense mechanisms against biotrophic and hemibiotrophic pathogens. The expression of various Pathogenesis-Related (PR) proteins encoding genes has also been observed with the exogenous application of the SA which is an indication for the accumulation of PR proteins has often been proposed as the molecular basis for SAR. Besides SA, other endogenous molecules such as octadecanoic acid derivatives such as Jasmonic Acid (JA), Methyl Jasmonate (MeJA), 12-Oxo-Phytodienoic Acid (OPDA),

and Ethylene (ET) have been identified as signals involved in the activation of the expression in a set of PR-proteins such as PR1, PR2, and PR5, which are defense genes of resistance. The onset of SAR is associated with increased levels of SA both locally at the site of infection and systemically in distant tissues, upon arrival of the mobile signal of SA the latter tissues will start producing SA, which induces the defence related proteins locally an systematically.

Host pathogen interaction and its implication in setting plant disease management

Presently disease management is largely based on the either direct or indirect application of hazardous chemicals such as fungicides, bactericides and insecticides. Problems with the resistance of pathogens to classical pesticides, the hazardous natures of the products on the environment, human and animal health associated with pesticides, inability of pesticides to effectively control some pathogens, e.g., virus and soil borne pathogens strongly necessitates searching for new safer means of disease management approach.

Among the management approaches that help to overcome such a problems are use practical knowledge of genetics of plant-pathogen interaction and its application using Genetic Engineering (GE). Sequencing of entire plant genomes, systematic plant transcriptome profiling and comprehensive genetic dissection of immune pathways in model plants such as Arabidopsis thaliana and rice has significantly enhanced our understanding of the mechanisms underlying microbial infection and plant immunity now be turned into new tools to engineer durable, broad spectrum plant disease management techniques. An alternative approaches such host resistance by induction of SAR have high potential to diminish the use of toxic chemicals in the agriculture and has emerged as an alternative, non-conventional, non-biocidal and eco-friendly approach for plant protection and hence for sustainable agriculture.

Diseases management using resistance induction

Induced resistance triggered by pathogens can be Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR). SAR is a form of induced resistance in plants with a specific defense signaling pathway that occurs systemically after localized exposure to a patho¬gen or alternatively, after treatment with synthetic or natural compounds. ISR was primarily described as a response induced by Plant Growth-Promoting Rhizobacteria (PGPR), but can also be induced by other compounds such as antibiotics, surfactants or chemical inducers. ISR, in con¬trast to SAR, does not involve the accumulation of SA, but is dependent on Jasmonic Acid (JA) and Ethylene (ET) signaling pathways. The inducers of ISR can fungi, bacteria, viruses, nematodes, insects, components, and products of pathogens and non-pathogens organic and inorganic polymers, and simple inorganic compounds.

An understanding of plant genetics and the biochemical changes leading to the SAR and ISR state could enable the development of either genetically engineered plants with enhanced disease resistance or novel mode-of-action plant protection chemicals that act by stimulating the plant's inherent disease resistance mechanisms.

All plants, whether they are resistant or susceptible, can respond to pathogen attack using elicitor triggered coordinated defence mechanisms, which results in the accumulation of different gene products in plants. Once elicitors are recognized by trans-membrane receptors of plant cells, induce an immune response, both locally (around the infection site/application) and systemic, through the translocation of signaling molecules in distal tissues.

Diseases management using plant immunity induction

Plant innate immune system is quiescent in normal healthy plants and sleeping giant and when awakened by specific signals it triggers expression of several defense genes. Unlike, transgenic plants developed by engineering disease resistance genes against specific pathogens, plants over expressing the plant immune system awakened by the alarm signals PAMP and PIMP trigger expression of hundreds of defense genes conferring resistance against wide range of pathogens. Study of genetics of plant-pathogen interaction found several PAMP developed formulations and application to triggers the induction of plant immune responses. Oligogalacturonates (OGAs), Plant elicitor peptides (Peps), and PAMP Induced Peptides (PIPs) are the important PIMPs capable of switching on plant innate immune responses. The crop diseases can be controlled by switching on plant innate immunity by manipulating PAMP-PIMP-PRR signaling complex developed by using these PIMPs based inducer compounds.

Chitosan as plant immunity inducer

Chitosan is a linear polysaccharide that can be obtained from the deacetylation of chitin, a long-chain polymer of N-acetyl-glucosamine present and easily extracted from fungal cell wall and crustacean shells. Chitosan beside its low cost production have biological properties such as non-toxicity, biocompatibility and biodegradability, which make chitosan a sustainable and eco-friendly molecule. One of the most studied proprieties of chitosan is its high antimicrobial activity against a wide variety of microorganisms such as fungi, bacteria and viruses. Chitosan used to control plant pathogens has been extensively explored with more or less success depending on the path system, in soil amendment, foliar application alone or in association with other treatments. A broad spectrum fungicidal activity of chitosan has been described *in vitro* on many pathogenic fungi such as, *Botrytis cinerea*, *Alternaria alternata*, *Colletotrichum gleosporoides* and *Rhizopus stolonifer* and several pathogenic bacteria including *Pseudomonas syringae*, *Agro bacterium tumefaciens* and *Erwinia carotovora*.

Harpin PAMPs as molecular tools to manipulate PAMP-triggered immunity

Harpins are glycine-rich and heat-stable proteins of in gram-negative plant pathogenic bacteria. It can be extracted from weakened nonpathogenic strain of *Escherichia coli* (K-12) modified and concentrated to produce harpin on a commercial scale. The *hrp* genes have been detected in several phytopathogenic bacteria including members of the genera *Erwinia*, *Pantoea*, *Pseudomonas*, *Xanthomonas*, and *Ralstonia*. Harpin has been produced commercially under two trade names "Messenger" and "Extend" which is marketed for the crop control diseases as well as a plant growth enhancer.

Harpin-induced plant immune signal transduction systems

When harpin is applied to a plant, it binds with the specific receptors found in the plasma membranes of the plant surface. A non proteinaceous receptor site for the harpin from the bean halo-blight pathogen *P. syringae pv. phaseolicola* has been identified in tobacco plasma membranes.

Foliar an application of harpin induces resistance against various viral, bacterial, oomycete and fungal pathogens in several crops

including wheat, rice, apple, pear, citrus, cucumber, tobacco, tomato, pepper and strawberry. Harpin Treatment Triggers SA-dependent signals which induce Systemic Acquired Resistance. Salicylic Acid (SA) accumulation is required for activation of local defenses at the initial site of attack, and in the distant pathogen-free organs for the induction of SAR accompanied by induction of the characteristic SAR genes *PR-1* and *PR-2*. Harpin genes can be used to engineer transgenic plants with constitutive expression of the genes that result in resistance against pathogens as in the case of transgenic rice plants that express a harpin-encoding gene (*hrf1*), derived from *Xanthomonas oryzae pv. Oryzae* and transgenic rice plants expressing *hrf1* gene encoding the harpin of the rice bacterial blight pathogen *X. oryzae pv, oryzae*.

Molecular boosting of plant recognition to infection

Receptor molecules in the host membrane recognize PAMPs and elicit a natural defense response called PAMP-Triggered Immunity (PTI). PAMP receptor molecules differ among plant species. Thus, genes encoding PAMP receptors from crops and other plants can be transformed into other crops, expanding the range of pathogen molecules that trigger PTI so that the receiving plant recognize infection and respond with its own, natural immune system. Enhancement of recognition capacities for microbial surface patterns using engineering provides an opportunity to enhance plant immunity. Increased resistance has been obtained using this strategy against a range of bacterial diseases in both monocots and dicots.

Mining R genes

Pathogens can produce one or more effector molecules which enhance virulence, resulting in Effector-Triggered Susceptibility (ETS). Over evolutionary time scales, plants respond to ETS by producing an intracellular receptor (R protein) which detects the presence or activity of particular pathogen effectors, restoring a resistance response called effector-triggered immunity or effector-triggered defense. This evolutionary, gene-for-gene interaction between pathogen effectors and their corresponding plant *R* proteins has yielded pools of *R* genes (resistance genes) useful in enhancing crops for disease resistance. Today Genetic Engineering (GE) offers an alternative for transfer of *R* genes, even from plants that are not part of a crop's normal breeding pool. For example, tomato bacterial leaf spot which is a highly destructive disease was controlled in the field with a single *R* gene obtained from pepper. Recent research has also shown that it is possible to enhance disease resistance by modifying the target of a pathogen effector. *R* genes do not code for new biochemical pathways but they code for receptor molecules which allows the plant to recognize the presence of an invading pathogen, there by taking advantage of their native, natural mechanisms of disease resistance. *R* genes from plants outside of a crop's breeding pool may be especially important since it opens a vast pool of *R* genes potentially effective and useful for enhancing defence.

Resistance (*R*) gene-encoded proteins provide effective host plant immunity through recognition of individual microbial effector molecules (ETI). Introgression of R genes into susceptible cultivars of host plants confers sufficient immunity to infection with pathogens expressing the matching effector. The efficiency of R proteins in conferring immunity across plant genus borders has prompted researchers to search for further *R* gene varieties in crops or their wild relatives and to introduce them in combination ('gene stacking', 'pyramiding') into transgenic crops. Three advantages over traditional breeding methods are envisaged. The use of individual genes prevents 'linkage drag' as observed upon crossing, multiple genes introduced as a single cassette may not segregate during breeding, and recognition of several effectors by stacked R proteins should confer durable resistance by slowing down microbial adaptation and break of immunity.

Molecular up regulating of defense pathways

Molecules involved in defense signaling, defense regulation, or other processes can be up regulated, boosting general defense responses. Such defenses include generation of reactive oxygen species, callose deposition, synthesis of Pathogenesis-Related (PR) proteins, and increased activation of Systemic Acquired Resistance (SAR) using plant's own natural immune system. One promising strategy is based on the exploitation of the genes encoding antifungal hydrolases, such as 1,3-glucanase and chitinase, which are associated with SAR-response in plants. The constitutive over expression of tobacco class I *PR-2* and *PR-3* transgenes in potato plants enhanced their resistance to *Phytophthora infestans*, the causal agent of late blight.

Comparative genomics and a combination of molecular genetics, biochemical and X-ray based structure elucidation approaches has widened our understanding on how microbial effectors manipulate host plant metabolism so interference with microbial effector function using gene technology provides a powerful strategy to control microbial infection.

Disarming host susceptibility genes

Plants possess genes whose products are important in its normal physiology, but in some way also function to facilitate pathogen infection and colonization. These can be considered susceptibility genes. Changes in such genes by natural means or using GE-induced can result in increased disease resistance changes so promise durable resistance can be gained.

Producing antimicrobial compounds

Genes encoding antimicrobial compounds such as chitinase genes from *Trichoderma* species can be expressed in crop plants can restrict pathogen activity and increase disease resistance. Resistance to diverse fungal diseases can be obtained in grape and cotton by transferring plants to constitutively produce chitin-degrading enzymes.

Silencing essential pathogen genes

The presence of double-stranded RNA (dsRNA) in the cytoplasm of eukaryotic cells triggers the natural and targeted process of post-transcriptional gene silencing (RNA silencing, RNA interference, or RNAi). Through the use of genetic constructs with sequence identity to important pathogen genes, RNAi can be elicited in plants to silence pathogen genes, resulting in reduced disease. In RNAi, no novel protein or biochemical pathway is created in the crop; the natural process of RNAi will be induced to silence a particular target gene of the pathogen. Induction of RNA silencing through transfer of coat protein gene of a viral pathogen can induce resistance to viral pathogens. Recent research clearly highlights the substantial potential which RNA silencing offers for management of diseases caused by biotrophic fungi, necrotrophic fungi, and oomycetes.

Reducing infection courts

Transgenic crops expressing gamaendotoxins (Cry proteins) from

Bacillus thuringiensis (Bt) have been used successfully to control certain insects. Another benefit from the use of Bt corn has been the well-documented reductions in mycotoxin contamination that sometimes occur. Reductions in both fumonisins and aflatoxins have been reported in field studies on several continents. These reductions have been associated with reduced insect wounding on kernels expressing a Cry endotoxin, resulting in fewer openings for infection by mycotoxin-producing fungi [15].

DISCUSSION

Plant immune system is complex surveillance systems that involve signal transduction pathways and several defence genes help to recognize and defend pathogens. Plant-pathogen interaction path ways and its molecular components are very complex and need still further advanced researches to exploit for so many applications including plant diseases management systems.

CONCLUSION

Deep insight to genetics of plant-pathogen interaction and plant defence and its signal pathways, exploration of entire plant genomes associated, systematic plant transcriptome profiling and comprehensive genetics of plant immune systems is now an important in put in searching for new plant diseases management approaches. An alternative approaches such as host resistance by artificial induction of SAR, PAMP and PIMP based expression of resistance genes or using potential elicitor compounds, molecular manipulation of PRRs, plant induced defense pathways and Transcription Factors (TFs), are currently pillar to diminish the use of toxic chemicals in the agriculture and replace with an alternative, non-conventional, non-biocidal, durable, cost effective and eco-friendly approach for plant protection, breeding and hence for sustainable agriculture.

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