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Role Of Plant Cell Components In Defending Against Stress

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Plants are frequently exposed to various environmental stresses, including extreme temperatures, drought, and attacks by pests and diseases. In their natural habitats, plants encounter numerous antagonists and have therefore evolved a wide range of defense mechanisms to cope with both biotic and abiotic stresses. These defense strategies are multifaceted, with cellular organelles playing a crucial role. For example, trichomes, which serve as mechanical barriers, form the frontline of the plant's defense system. Active defense mechanisms are particularly important in the context of plant diseases, where the host plant may be damaged by a pathogen. These mechanisms include properties of the host that reduce the extent of damage. Passive defense mechanisms are independent of the pathogen, while active mechanisms are triggered by changes in the host induced by the pathogen. For instance, pre-existing lignified cell walls in higher plants act as a passive defense against fungal pathogens by restricting fungal growth. Conversely, cell walls that become lignified in response to pathogen attack represent an active defense mechanism if this response reduces damage.

Keywords: Stresses, Organelles, Plant Defense, Photosynthesis, Pathogens.

Introduction : Despite the many differences among living organisms, they share several fundamental characteristics, such as respiration, nutrition, and reproduction. However, plants possess a unique feature—photosynthesis. This process allows plants to convert light energy into chemical energy, which fuels their activities. Photosynthesis is responsible for producing and maintaining the Earth's oxygen content and provides the organic compounds and energy essential for life (Bryant and Frigaard, 2006). Plants are vital to human livelihoods, and the relationship between plants and humans is deeply interconnected (Anant *et al*., 2013).

Like all living organisms, plants are continuously exposed to stress. These stresses are generally classified into two categories: abiotic and biotic. Abiotic stress includes factors like mechanical injury, while biotic stress refers to plant diseases caused by pathogens (Doughari, 2015). Plant diseases disrupt the normal functioning and growth of plants, leading to reduced efficiency or metabolic breakdown. Microbial infections caused by viruses, bacteria, fungi, or other microorganisms are common sources of these diseases (Dickison, 2000). Biotic stresses also include pests, such as insects and nematode worms, which can harm plants.

Plants must defend themselves against these destructive agents through various defensive mechanisms to survive. Although pathogens are hostile invaders that attack plants and crops, they are, in essence, organisms trying to survive and thrive. However, they do so at the expense of their host through parasitism (Alberts *et al*., 2002). Pathogens like bacteria, fungi, and viruses are commonly found in irrigation water and farm runoff. Other pathogens, such as prions, protozoa, viroids, and human parasites, primarily affect humans.

Stomata in plants regulate gas exchange and water transpiration in response to environmental changes. Recent research has shown that stomata also play a critical role in plant defense. Melotto *et al*. (2006) demonstrated that stomata close upon detecting potential microbial pathogens, preventing infection of the leaf interior. However, pathogenic bacteria have evolved strategies to counteract this defense mechanism. They produce a phytotoxin called coronatine, which forces the stomatal pores back open, allowing the bacteria to enter and cause disease. For bacteria, gaining entry into the plant is crucial for survival, as they may perish if left on the surface.

Scope of the Study

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.

MECHANISM OF INFECTION OF PLANT BY PATHOGENS

The "infection process" is completed in three phases: *pre-entry*, *entry* and *colonization*. It encompasses the multiplication of an infective propagule in or on a potential host by the establishment of a parasitic relationship between the pathogen and the host. The process of infection is influenced by
characteristics of the pathogen, the host and the external environment. If any of the stages of the infection process is inhibited due to any factors, the pathogen will not cause disease in the host. Some ectoparasites may colonize the exterior of the plant, while pathogens can also infiltrate the host plant by penetrating through natural openings, such as stomatal pores, or via wounds (Melotto *et al*., 2006). The symptoms caused by these pathogens arise from disruptions in vital processes, including respiration, photosynthesis, transpiration, nutrient translocation, and other aspects of growth and development.

It is evident that, unlike vertebrates, plants do not possess a sophisticated immune system that provides resistance to infections (Dickison, 2000). In natural environments, plants encounter numerous antagonists and have therefore developed a wide array of defense mechanisms to cope with various biotic and abiotic stresses. Several factors influence plant defense, including the age of the plant, the type of organ infected, the nutritional status of the host, and environmental conditions (Gianinazzi and Ahl, 1983).

Plants possess innate immunity within each cell and can produce systemic signals that originate from the site of infection. Additionally, plants defend themselves against unfavorable conditions. The defense compartments within plant cells include the cell wall, endoplasmic reticulum, mitochondria, chloroplasts, cytoplasm, Golgi apparatus, plasma membrane, peroxisomes, and vacuoles.

Defense Through Cell Wall

The cell wall serves as the frontline of the plant defense system, a distinctive structural feature of plant cells. It forms a surrounding barrier primarily composed of cellulose, which is deposited in a fibrillar form (Dickison, 2000).

One of the key ways the cell wall contributes to plant defense is through the production of oligosaccharins. These fragments of the plant cell wall act as regulatory molecules, controlling various functions such as growth, development, reproduction, and defense against disease. For instance, certain oligosaccharins, like oligogalacturonides, act as elicitors that trigger pathogen defense responses. These responses include the accumulation of phytoalexins, proteinase inhibitors, lignin, peroxidase, lipoxygenase (LOX), and β-1,3-glucanases (Ryan, 1988; Hahn *et al*., 1989; Ebel and Cosio, 1994). Due to their natural origin, these bioactive molecules are relatively easy to produce and are likely to gain public acceptance. Oligosaccharins also have practical applications in agriculture. For example, oligogalacturonides derived from citrus pectin can partially or completely replace phytohormones in the in vitro culture of crops like sugarcane, citrus, coffee, tomato,

rice, and banana. This reduces the time required for plant propagation, prevents culture-induced genetic changes, increases field survival rates, and promotes rooting and plant growth (Saa Silva *et al*., 2013).

The importance of the cell wall in defense is also evident in fungi, where the integrity and shape of the organism rely on the mechanical strength of its cell wall. The fungal cell wall is a complex structure typically composed of chitin, glucan, mannan, and proteins, though its composition varies among fungal species (Dhume *et al*., 1993). Both plant and fungal cell walls are targets of cell wall-degrading enzymes during the early stages of plant-pathogen interactions. Additionally, oligosaccharides released from the degradation of plant or fungal cell walls can act as signalling molecules, triggering defense responses and promoting growth or development in plants (Sea Silva *et al*., 2013).

The role of the cell wall in plant defense mechanisms has been further confirmed through studies on cellulose-deficient mutants. These mutants often exhibit increased lignification (Malinovsky *et al*., 2014), and interestingly, these structural changes also enhance defense responses (Hamann *et al*., 2009).

The role of the cuticle in plant defense mechanisms becomes evident when studied further. mutants with specific defects in cutin. These mutants show an altered ultra-structure of the cuticle and an enhanced permeability of the cuticle to solutes In addition, pollen could germinate on fully differentiated leaves of cutinase-expressing plants but not on control leaves.

Some of the epidermal cells of most plants grow out in the form of hairs or trichomes. They may be found singly or less frequently in groups. They may be unicellular or multicellular and occur in various forms. They vary from small protuberances of the epidermal cells to complex branched or satellite multicellullar structures.

Alike trichomes thorns are a part of the defensive mechanism in plants. A thorn is a loose term for any sharp, pointed appendage coming off a plant for defensive purposes. Botanically, thorns can also be called spines (modified leaves), prickles (sharpened branches) or trichomes based on their location on the plant. Thorns are essentially defense mechanisms for the plant, The sharp points protect the plant against animals that want to eat it.

Moreover smells, oils, thick and waxy skin, intact outer periderm or bark of woody plants provide sufficient barrier for plant defense against stresses by different methods for example some plants emit different smells to repel animals or others excrete oils to kill other plants. Also, some plants have thick skin to keep from dehydrating. Trees have different types of bark to help deal with cold or hot environments. We must know plants don't use one defensive strategy. Plants have all kinds of strategies for protecting themselves from difficult conditions (Milewski *et al*., 1990).

Defense by Plasma Membrane

Plants have developed a multi-layered immune system that dynamically responds to pathogen attacks. Most types of plant pathogens remain outside the host cell membrane throughout their lifecycle, making the plant plasma membrane (PM) a crucial interface for communication between plants and microbes (James and Gitta, 2011). The initial recognition of pathogens occurs at the PM, and many of the earliest cellular responses to microbial invasion are controlled by enzymes and ion channels located at the PM (Boller and Felix, 2009).

Furthermore, numerous downstream responses to pathogen stimuli are also carried out at the plasma membrane. To suppress plant defense mechanisms and obtain nutrients, pathogens must manipulate host cells. Consequently, membrane transport processes play essential roles in both compatible (susceptible) and incompatible (resistant) plant-pathogen interactions (Hahn and Mendgen, 2001; Ward *et al*., 2009).

In plants, PM H+-ATPases are the primary pumps responsible for establishing cellular membrane potential. These pumps utilize energy from ATP hydrolysis to transport protons from the cytosol to the extracellular space, thereby creating and maintaining a negative membrane potential and a transmembrane pH gradient (with an acidic environment outside the cell). This proton electrochemical gradient regulates various transport processes across the PM. During plant immune responses, the activity of PM H+-ATPases is dynamically regulated, and many pathogens specifically target this enzyme family during infection (James and Gitta, 2011).

Defense by Cytoplasm

The cytoplasm of eukaryotic cells consists of all cell material between the nucleus and the plasma membrane and contains membrane-bounded structures, organelles, which are embedded in the cytosol consisting of

water, salts and organic molecules, including sugars, proteins and many enzymes that catalyze reactions. The cytoskeleton of microtubules and actin filaments in the cytosol structures the cell by localizing and transporting the organelles bound to these tubules and filaments. The plasma membrane, enveloping the cytoplasm physically, separates the cell content from the extra-cellular environment, which in plant cells is the cell wall (Esseling-ozdona *et al*., 2008).

Also, cytoplasm is an important barrier for plants' defense. The earliest cytoplasmic response to contact between a microorganism and host cell is cyclosis within the protoplast, with a resultant reorganization of cytoplasmic organelles. Infection of tuber tissues and tissue culture cells of *Solanum tuberosum* by *Phytophthora erythroseptica, P. infestans* and *Fusarium caeruleum* caused swelling and disruption of host cytoplasmic particles containing acid phosphatases, esterases and proteases. Heavy diffuse cytoplasmic staining for acid phosphatase was a consistent feature of infection by the three fungi, but staining reactions for esterase and protease showed much less diffuse staining and a lesser degree of particle swelling. An excess recovery of ribonuclease from tissues infected with *P. infestans* and *F. caeruleum* was found. An electrophoretic comparison of near isogenic lines of wilt resistant (*Fusarium oxysporum* f.sp. *pisi*) or susceptible *Pisum sativum* was made.

Defense by Endoplasmic Reticulum

The endoplasmic reticulum (ER) is a network-like organelle enclosed by a single membrane, consisting of various functional domains (Staehelin, 1996). It includes the rough ER (rER), smooth ER (sER), and the nuclear envelope. The rER, characterized by its ribosome coating, is responsible for synthesizing secretory proteins. In contrast, the sER lacks ribosomes and is involved in lipid biosynthesis. Additionally, the ER accumulates specific seed storage proteins, such as prolamin and zein, to form protein bodies (PBs) in the endosperm of certain plants (Herman, 2008; Yasuda *et al*., 2009; Kumamaru *et al*., 2010; Satoh-Cruz *et al*., 2010; Nagamine and Okita, 2011).

Evidence suggests that ER bodies play a role in plant defense mechanisms. Wounding or the application of the wound hormone jasmonic acid triggers the formation of ER bodies (Matsushima *et al*., 2002; Hara-Nishimura and Matsushima, 2003), as does damage from pest chewing. Jasmonic acid is a well-known hormone that mediates wound responses, and induces resistance against insect and pathogen attacks (McConn *et al*., 1997; Vijayan *et al*., 1998; Li *et al*., 2002; Chini *et al*., 2007; Sato *et al*., 2011), and regulates the expression of wound-inducible genes, such as *vegetative storage protein* (VSP) genes (Leon *et al*., 2001; Lorenzo *et al*., 2004).

A key component of ER bodies is β-glucosidase. Plants accumulate various glycosides derived from secondary metabolism (Gachon, 2005; Ketudat and Esen, 2010). Several glycosides, including cyanogenic glucosides, saponins, glucosinolates, and DIMBOA (2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one) glucoside, are involved in plant defense mechanisms (Mattiacci *et al*., 1995; Konno *et al*., 1999; Tattersall *et al*., 2001; Carpinella *et al*., 2005; Beekwilder *et al*., 2008; Morant *et al*., 2008). These compounds are typically stored in an inactive form and become activated upon the removal of glycone. β-Glucosidases hydrolyze these molecules into glycone and aglycone, making them essential for producing toxic compounds that defend against insects and fungi (Mattiacci *et al*., 1995; Tattersall *et al*., 2001; Ketudat and Esen, 2010).

Defense by Nucleus

Communication between the cytoplasm and nucleus is a fundamental process conserved across eukaryotic systems. The transport of macromolecules across the nuclear envelope is facilitated by nuclear pore complexes (NPCs), composed of nucleoporins, and relies on import and export receptors, known as importins and exportins. These receptors recognize nuclear localization signals (NLSs) and nuclear export signals on cargo proteins (Meier and Brkljacic, 2009). The Ras-related nuclear (Ran) protein dictates the direction of transport through its binding to GDP (on the cytoplasmic side) or GTP (on the nuclear side). Various cellular factors, such as nucleoporins, importins, and Ran-GTP-associated components, play a crucial role in the transport of macromolecules across the nuclear envelope and are vital for mounting an effective immune response to different pathogens (Palma *et al*., 2005; Zhang and Li, 2005; Tameling and Baulcombe, 2007; Cheng *et al*., 2009).

A considerable number of effector proteins from various pathogens, including nematodes, fungi, viruses, bacteria, and oomycetes, exploit the host's nuclear import machinery to reach the nucleus (Deslandes and

Rivas, 2011). This allows effectors to manipulate host transcription or directly target essential nuclear components to benefit the pathogen. Additionally, some effectors are believed to influence histone modifications and chromatin remodelling. Moreover, the nuclear translocation of effectors may alter the sub-cellular localization of their cognate R proteins, a process essential for R-protein-mediated plant immunity (Susana, 2011).

Defense by Chloroplast

It's fascinating to recognize chloroplasts as key defense organelles. While chloroplasts are vital for photosynthesis in algae and higher plants—where they convert light energy into chemical energy (Cooper, 2000)—their functions extend far beyond this primary role. Along with the nucleus, cell membrane, and endoplasmic reticulum (ER), chloroplasts are crucial in establishing plant immunity against microbial attacks (Padmanabham and Dinesh Kumar, 2010). This highlights the importance of interorganellar signaling during plant defense, ensuring a coordinated whole-cell response (Serano *et al*., 2016).

Among cellular organelles, chloroplasts serve as integrators of environmental signals and, as mentioned earlier, play a pivotal role in defense (Serano *et al*., 2016). Evidence supporting the chloroplast's role in plant defense includes the increased demand for photosynthesis during pathogen interactions. This is because the biosynthesis of defense molecules and the activation of defense responses require energy provided by photosynthesis (Hammerschmidt, 1999; Swarbrick and Lefert, 2006). Furthermore, some virulent pathogens exploit plant carbon compounds and even utilize plant transporters, such as those from the SWEET family, to promote sugar efflux, thereby increasing the photosynthesis demand in host cells (Chen *et al*., 2010).

Interestingly, rather than ramping up photosynthesis, several studies have reported a suppression of photosynthetic functions in infected plants. This may reflect an active plant strategy to limit carbon availability to pathogens or to prioritize defense mechanisms over other physiological processes, including photosynthesis, during pathogen attack (Padmanabham and Dinesh Kumar, 2010). Additionally, chloroplasts are major production sites for defense molecules, including hormones and secondary messengers, further underscoring their central role in plant immunity (Padmanabham and Dinesh Kumar, 2010).

Defense by Mitochondria

Mitochondria are among the most critical defensive barriers in plants. Following pathogen detection, mitochondria play a key role in the plant cell's defense strategy by integrating and amplifying various signals such as salicylic acid, nitric oxide, reactive oxygen species (ROS), and pathogen elicitors. These signals often disrupt normal mitochondrial function, leading to changes in respiration, membrane potential, and ROS production. At this stage, mitochondria generate signals that influence the cell's redox state and regulate the expression of nuclear genes through mitochondrial retrograde signalling. In more advanced stages of infection, mitochondria can trigger programmed cell death to prevent the spread of the pathogen throughout the plant.

Additionally, it is widely accepted that Pattern-Triggered Immunity (PTI) is a rapid response of the plant immune system, with the initial "cell reprogramming" occurring within the first hour after pathogen recognition. There is substantial evidence supporting the involvement of plant mitochondria in incompatible plant-pathogen interactions, including their role in perceiving signals from the intercellular space or apoplast (Amirsadeghi *et al*., 2007).

Defense by Golgi Apparatus

Glycosylation is a post-translational modification that plays a significant role in protein activity and the protein secretory pathway in eukaryotic cells (Roth, 2002). This critical process takes place in the endoplasmic reticulum and Golgi apparatus (Kang *et al*., 2015). While glycosylation in the endoplasmic reticulum and Golgi apparatus is known to regulate protein quality control, salt stress response, and cellulose biosynthesis, there is limited evidence of its roles in plant immunity (Kang *et al*., 2015). However, the importance of the Golgi apparatus in plant defense and immunity has been demonstrated through studies on Arabidopsis thaliana mutants with defects in glycosylation.

Defense by Peroxysomes

As previously discussed, plants are susceptible to infections caused by fungi, bacteria, viruses, and nematodes. Peroxisomes are one of the subcellular organelles that play a significant role in disease resistance. These single-membrane-bound organelles, present in nearly all eukaryotic cells, are primarily involved in oxidative metabolism (del Rio *et al*., 2002; Kaur *et al*., 2009). Peroxisomes house a variety of enzymes that work together to detoxify cellular toxins, most notably hydrogen peroxide, a common byproduct of cellular metabolism in living systems. These enzymes convert hydrogen peroxide into water, thus neutralizing this potentially harmful substance before it is safely released back into the cell (del Rio *et al*., 2002; Nyathi and Baker, 2006).

The roles of peroxisomes in plant defense, both biotic and abiotic, are becoming increasingly clear (del Rio *et al*., 2006; Reumann *et al*., 2007; Palma *et al*., 2009; Corpas *et al*., 2010). Additionally, peroxisomes contain crucial enzymes and generate various metabolites that play significant roles in both direct and indirect plant defense mechanisms against herbivores (Shabab, 2013). Numerous studies have highlighted the importance of reactive oxygen species (ROS) in herbivory-related responses. The feeding activity of certain pests on their host plants can lead to significantly elevated ROS levels (Wu and Baldwin, 2010). For instance, it has been shown that while mechanical wounding by herbivores alone may not suffice to increase ROS levels, the specific contribution of herbivory is essential for this increase (Leitner *et al*., 2005).

Defense by Vacuoles

The central vacuole is a distinctive feature of mature plant cells and represents the largest intracellular compartment in many higher plant tissues. Over the past two decades, numerous studies have demonstrated that this organelle functions as a multifunctional compartment within plant cells. One of the key roles of vacuoles is their involvement in the plant's defense response under environmental stress conditions (Andreev, 2001). Plant cells possess a large central vacuole that accumulates various hydrolytic enzymes and antimicrobial compounds, suggesting a potential role in plant defense (Hatsugai and Nishimura, 2010). Unlike animals, which have specialized immune cells and an adaptive immune system, plants rely on innate immunity within each cell and produce systemic signals from the site of infection. Plants have also evolved a complex immune system to resist pathogen attacks, which includes rapid and localized cell death (known as the hypersensitive response, HR) and stomatal closure (Dangl *et al*., 1996; Lam *et al*., 2001; Melotto *et al*., 2006; Mur *et al*., 2008).

Research has shown that both mammalian and plant cell death mechanisms share common morphological and biochemical features, such as cytoplasm shrinkage, nuclear condensation, DNA fragmentation, and the release of cytochrome c from mitochondria (Sun *et al*., 1999; Sasabe *et al*., 2000; Kim *et al*., 2003; Ji *et al*., 2005). However, the precise mechanisms by which signaling pathways trigger localized HR without causing whole-plant cell death remain unclear. Studies suggest that HR in plants is regulated through cascade-like activity. Vacuolar processing enzyme (VPE) is localized in the vacuolar membrane and mediates virus-induced hypersensitive cell death by controlling the collapse of the vacuolar membrane (Hatsugai *et al*., 2004).

PLANT'S IMMUNE SYSTEMS

The plant immune system is composed of two interconnected layers of receptors—one located outside the cell and the other inside. Both systems work together to detect intruders, respond to infections, and signal to the rest of the plant, and sometimes to neighboring plants, about the presence of pathogens. These systems detect different types of pathogen molecules and interact with distinct classes of plant receptor proteins (Dangl *et al*., 2013).

The first layer is primarily driven by pattern recognition receptors (PRRs), which are activated by recognizing evolutionarily conserved pathogen- or microbe-associated molecular patterns (PAMPs or MAMPs). Activation of PRRs triggers intracellular signaling, transcriptional changes, and the production of a complex defensive response that limits pathogen colonization. This system is known as PAMP-Triggered Immunity (PTI) (Jones and Dangl, 2006; Dodds and Rathjen, 2010).

The second layer, primarily involving Effector-Triggered Immunity (ETI), consists of nucleotide-binding leucine-rich repeat receptors (NLRs) encoded by R genes. These receptors detect specific pathogen "effectors," which are molecules that pathogens use to suppress plant defenses. When a specific NLR protein recognizes an effector, it activates a defense response that restricts pathogen proliferation (Dangl *et al*., 2013).

Both PTI and ETI trigger various cellular responses, including ion channel gating, oxidative bursts, changes in cellular redox states, and protein kinase cascades. These responses can directly reinforce the cell wall, produce antimicrobial compounds, or lead to changes in gene expression that further elevate plant defenses (Dangl *et al*., 2013).

Although the plant immune system shares some mechanistic similarities with the immune systems of insects and mammals, it also possesses unique characteristics. For example, plants can detect pathogens and the effects of infection through mechanisms distinct from those in animals. PAMP-Triggered Immunity (PTI) is often the plant's first line of defense (Jones and Dangl, 2006). Immune-eliciting PAMPs include bacterial flagellin, lipopolysaccharides, and fungal chitin (Numberger *et al*., 2004). While PTI is generally effective against most pathogens, some pathogens have evolved effector proteins that suppress this basal defense (Dodds and Rathjen, 2010).

Effector-Triggered Immunity (ETI) is activated by the detection of pathogen effectors and is heavily reliant on R genes (Jones and Dangl, 2006). ETI often leads to a stronger and more specific immune response, including the hypersensitive response (HR), which involves localized cell death to prevent pathogen spread. Additionally, plants can sense damage-associated molecular patterns (DAMPs), such as fragments of the plant cell wall released during infection, which further activates defense mechanisms.

Many receptors for MAMPs, effectors, and DAMPs have been identified. MAMPs and DAMPs are often detected by transmembrane receptor kinases with LRR or LysM extracellular domains, while effectors are typically recognized by NLR proteins (Dodds and Rathjen, 2010).

Plants have evolved R genes that allow them to recognize specific pathogen effectors, either through direct binding or by detecting the effector's modification of a host protein (Jones and Dangl, 2006). This recognition is part of a co-evolutionary arms race between plant resistance genes and pathogen avirulence (Avr) genes. R genes encode NB-LRR proteins (nucleotide-binding/leucine-rich repeat domains), which mediate a wide range of disease resistance responses sufficient to halt pathogen growth or spread. The specificity of R genes for particular pathogen strains has been a key concept in plant-pathogen interactions since Harold Flor's mid-20th-century gene-for-gene theory (Numberger *et al*., 2004).

Effectors, central to a pathogen's ability to cause disease, often manipulate host cell physiology to promote infection. Bacterial pathogens typically produce a few dozen effectors, delivered into the host by a Type III secretion system, while fungal, oomycete, and nematode pathogens may produce several hundred effectors (Lindeberg *et al*., 2012; Hewezi and Baum, 2013). Core effectors, widely distributed among a pathogen population, play a significant role in virulence and are potential targets for breeding disease-resistant crops (Dangl *et al*., 2013).

In addition to PTI and ETI, plants also use RNA silencing and systemic acquired resistance (SAR) to defend against pathogens. RNA silencing, mediated by RNA interference, serves as a form of adaptive immunity against viruses (Ding and Voinnet, 2007). SAR, largely dependent on salicylic acid, and induced systemic resistance (ISR), largely dependent on jasmonic acid, prepare distant parts of the plant for future attacks (Spoel and Dong, 2012).

In some cases, plant resistance is effective against an entire pathogen species, such as barley's MLO gene against powdery mildew or wheat's Lr34 and Yr36 genes against rust diseases. The mechanisms underlying such broad resistance can vary depending on the specific plant-pathogen interaction and may involve pre-formed defenses or a lack of pathogen co-adaptation.

Plant defense signaling begins with pathogen detection by receptors, which then activate a cascade of events, including reactive oxygen species (ROS) production, ion fluxes, hormone signaling, and activation of protein kinases. These events lead to the activation of defense-related genes and the enhancement of the plant's immune response (Numberger *et al*., 2004).

DEFENSE THROUGH GENETICALLY ENGINEERING DISEASE-RESISTANT PLANTS WITH PLANT-DERIVED GENES

The number of plant resistance (R) genes that have been isolated is rapidly increasing. The first plant resistance gene to be isolated was the Hml gene from corn in 1992. This gene encodes an enzyme that neutralizes the HC toxin produced by the leaf spot fungus *Cochliobolus carbonum* (Honee, 1999). In 1993, the Pto gene from tomato was identified, which encodes a protein kinase involved in signal transduction and provides resistance to strains of *Pseudomonas syringae* pv. tomato that carry the avirulence gene *avrPto*. The following year, four additional plant resistance genes were isolated: the Arabidopsis RPS2 gene, which offers

resistance to strains of *P. syringae* pv. tomato and *P. syringae* pv. maculicola carrying the *avrRpt2* gene; the tobacco N gene, which confers resistance to tobacco mosaic virus; the tomato Cf9 gene, which provides resistance to races of the fungus *Cladosporium fulvum* carrying the *avr9* gene; and the flax L6 gene, which grants resistance to specific races of the rust fungus *Melampsora lini* carrying the *avr6* gene.

These five plant resistance genes are activated by the corresponding avirulence genes of the pathogen, with the pathogen's products acting as signals that trigger the hypersensitive response in the host plant (Luderer and Joosten, 2001). Since then, many more plant resistance genes have been isolated. Some of these genes offer resistance to pathogens expressing one or both of two unrelated Avr genes. It is anticipated that these R genes, along with many others likely to be isolated in the future, will be widely used to genetically engineer transgenic plants resistant to various races of pathogens.

In addition to these specific R genes, other plant genes that encode enzymes or proteins known as PR (pathogenesis-related) proteins have also been shown to confer resistance when expressed in transgenic plants (DeWit, 1992). For example, tobacco plants that were transformed with a chitinase gene from beans became resistant to the soilborne fungus *Rhizoctonia solani*, although they remained susceptible to the oomycete *Pythium aphanidermatum*, whose cell walls lack chitin.

DEFENSE THROUGH RNA SILENCING BY PATHOGEN-DERIVED GENES

RNA silencing is a form of gene regulation in plants that functions as a defense mechanism against viruses. This process involves targeting and degrading specific RNA sequences, effectively neutralizing viral infections. RNA silencing is not limited to plants; it occurs across a wide range of eukaryotic organisms, including fungi and animals. While plants utilize RNA silencing to protect themselves from viruses, viruses have evolved to counteract this defense by encoding proteins that suppress the silencing of their RNA (Balmori *et al*., 2002).

CONCLUSION:

Plants have evolved a multi-layered immune system that dynamically responds to pathogens. The cell membrane acts as a key mediator of communication between plants and microbes, while cytoplasmic and membrane-bound structures provide defense against various stresses. Cellular organelles play crucial roles in plant defense, primarily through the action of their enzymes, such as proteases, esterases, and ribonucleases. For example, plasma membrane H+-ATPases and glucosides in the endoplasmic reticulum (ER) contribute to plant immunity. Reactive oxygen species (ROS), salicylic acid (SA), and jasmonic acid (JA) in chloroplasts and mitochondria are also critical components of the plant immune system. In the nucleus, macromolecules like nucleoporins, importins, and Ran-GTP-related components are essential for mounting an effective immune response. Furthermore, the Golgi apparatus, peroxisomes, and vacuoles contribute to plant defense through glycosyltransferases, myrosinase, and hydrolytic enzymes, respectively.

The defense mechanisms in plants are integral to their metabolic activities, and while various mechanisms have been discussed, RNA silencing stands out as particularly significant. Agriculture and sustainability are closely linked through RNA surveillance and cellular defense pathways. RNA silencing, first observed in transgenic plants transformed with viral genes, provides "pathogen-derived resistance." It was initially noted that genes in a sense orientation in transgenic plants interfered with the expression of both transgenes and related endogenous genes, leading to what was then called "cosuppression." RNA silencing occurs post-transcriptionally and involves targeted RNA degradation. Evidence of this mechanism came from the observation that plants carrying viral transgenes were resistant to related viral strains that replicate in the cytoplasm, indicating that RNA silencing operates in the cytoplasm rather than the nucleus.

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