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A Comprehensive Review of Mechanisms of Plant Defence Against Selected Categories of Plant Pathogens

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Systematic Review

Keywords: Biotrophs, Necrotrophs, Hemi-biotroph, Pathogen Associated Molecular Patterns, Pattern Recognition Receptors, Salicylic Acid, Jasmonic Acid, Systemic Acquired Resistance, Induced Acquired Resistance, Pathogen Triggered Immunity, Effector Triggered Immunity

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Abstract

Pathogens pose significant threats to plants, and over time, they have evolved strategies to obtain nourishment. These pathogens are broadly categorized into biotrophs, necrotrophs, and hemi-biotrophs. These terms, derived from the Greek "troph" (nourishment), helps clarify their nature: biotrophs ("bio" meaning life) feed on living plant cells, while necrotrophs ("necro" meaning death) nourishment from dead or decaying cells. Hemi-biotrophs are a hybrid category, living a phased life that begins with biotrophy before switching to necrotrophy.

Distinctive Pathogen Strategies: Biotrophs establish a long-term, symbiotic relationship with their host to extract nutrients without causing immediate cell death. They employ phased infection strategies, using effector proteins to suppress the plant's defense mechanisms. Such as black sigatoka fungus in bananas and rust fungi in sorghum. These pathogens often develop specialized structures like the appressorium for anchorage and the haustoria for nutrient absorption.

Necrotrophs, on the other hand, are aggressive killers. They overwhelm plant defenses by releasing a large number of toxins and lytic enzymes simultaneously, causing rapid tissue necrosis and cell death. They then consume the dead tissue for nourishment. A common example is *Botrytis cinerea*, which secretes oxalic acid to create an acidic environment optimal for its destructive enzymes.

Host-Pathogen Specificity and Plant Defense: Pathogen specificity, the ability of a microbe to infect a particular plant species, is a key aspect of plant pathology. This is often a result of coevolution, leading to generalists (pathogens with a broad host range, like *Botrytis cinerea*) and specialists (those that infect only a few related species, like *Puccinia graminis*).

Plants have evolved a two-layered defense system:

PAMP-triggered immunity (PTI): activated when a plant's pattern recognition receptors (PRRs) detect conserved pathogen-associated molecular patterns (PAMPs).

Effector-triggered immunity (ETI): activated when plants with specific resistance (R) genes recognize virulence factors (effectors) released by adapted pathogens. This often leads to a hypersensitive response (HR), which is a localized cell death that limits pathogen spread.

The "gene-for-gene" model explains this interaction, where a dominant resistance gene in the plant matches a corresponding dominant avirulence (*Avr*) gene in the pathogen, leading to disease resistance.

Systemic Acquired Resistance (SAR): Beyond local defense, plants can also develop systemic acquired resistance. This broad-spectrum immunity is triggered by a localized infection and protects the entire plant from future attacks. Mobile signals, such as methyl salicylic acid, are transported from the infection site to other parts of the plant, where they are converted to salicylic acid, leading to systemic protection.

1.0 Biotrophs, Necrotrophs and Hemi-biotrophs

Plants like any other living organism have threats to their existence among which include pathogens. Over time, plant pathogens have developed a diversity of relationships through which they can nourish themselves from the host plants. Largely, three major categories of pathogens have been established biotrophs, necrotrophs and hemi-biotrophs. This is largely referred to as the triad of plant pathogens (Arya & Cohen, 2022). Understanding the nature and characterization of these pathogenic categories creates a better insight into the relationship that these pathogens create with the host plants.

The ICBN clearly says that the nomenclature of these living organisms has been a fundamental basis for scientists to understand the behavior, character, shape and or mode of feeding of different organisms (Spencer & Cross, 2007). Therefore, to have a clear understanding of the three major categories of the plant pathogen, we shall take an interest in the etymology of these terminologies; -biotrophs, necrotrophs, and hemi-biotrophs. In common, they all have the suffix "troph" which is a Greek derivative to mean nourishment. Therefore, when combined with bio which means life/live/living, it simply means that biotroph pathogens nourish from living cells and, in this case, obtain nutrients from living plant cells. When trophy is combined with necro which is a Greek derivative to mean kill/decay/waste, it simply means nourishment from decay. In this case, necrotrophs are pathogens that obtain nutrients from decaying/dead host plant cells. As for the hemi-biotrophs living a phased life between biotrophy and necrotrophy, it can be termed a hybrid life (Münch et al., 2008). These nourishment categories show distinctive survival strategies that pathogens adapt to maximise nutrient acquisition and reproduction.

Having had a glimpse of the plant pathogenic triad, we shall now go into distinctive independent comparison and characterization of the biotrophs, necrotrophs and hemi-biotrophic. Biotrophs, with an infection strategy of co-existing with the host plant, create a symbiotic close relationship with the host plant, this relationship is often referred to as a delicate balance (Rajarammohan, 2021).

Unlike necrotrophs, biotrophs will establish a long-term relationship with the host plant which gives them a lively time frame to access and acquire nutrients for their different survival needs (Mapuranga, Zhang, Zhang, Chang, & Yang, 2022). This can be achieved with phased employment of plant cell destructive mechanisms including cell wall degrading enzymes (chitinase, pectinase, effector proteins that inhibit the defence response mechanisms of host plants for example occupying the receptor sites for salicylic acid that is responsible for a systemic Acquired Resistance (Toruño, Stergiopoulos, & Coaker, 2016). With such phased

infection strategies, biotrophs have been able to strike delicate balance relationships with their host plants.

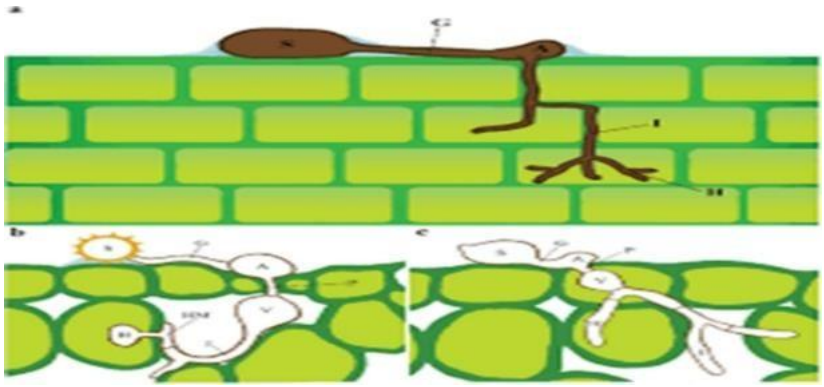


Figure 1: A biotroph infection without plant cell death/necrosis -coexistence

The largely known fungal and bacterial biotrophs like *Mycrosphaerella Fijiensis* a powdery mildew fungus in bananas also known as black sigatoka (Ramírez, Sáenz, Vargas, & Araya, 2008) penetrate the host plant epidermal cells to extract target and desired nutrients,

Puccinia Sorghi is an important rust fungus in sorghum whose network of hyphae (mycelium) occupies the intercellular spaces developing sexual fruiting bodies called pycnium with golden/rust-coloured ostiole periphyses that appear as rusts on the epidermis of the leaf (Figure 2) (Rice, 1933). These cause minimum damage to host plants allowing a symbiotic biotrophic phase of life, *Agrobacterium tumefecien* causes crown gall tumors utilising its type III secretory systems (T3SS) to directly insert a tumor DNA (T-DNA) which is transcribed to phenotypically cause tumors, an abnormality and feeding niche to the bacteria (Păcurar et al., 2011).

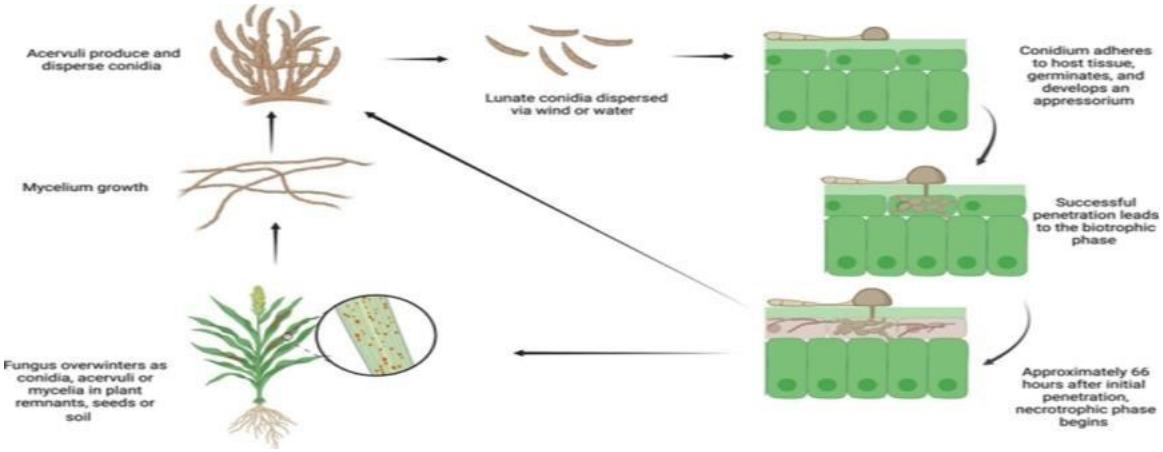


Figure 2: The life cycle of fungi in the infection phase with host plant.

The infection strategy, life cycle and associated structures of biotrophic pathogens create a favourable and smooth facilitation of the pathogen to attack, survive and reproduce from the host plant, taking a kin interest in the fungal biotrophs, they exhibit a life cycle that typically

begins with dispersal (wind, rain and or vectors) of the spores. The spores can be asexual or sexual spores for example the ascospores, and basidiospores from ascomycetes and basidiomycetes fungi respectively. A good example of these spores is conidia which are means of rapid spread. Conidia is capable of self-motility with the aid of a flagella (figure 2)(Blango, Kniemeyer, & Brakhage, 2019).

Once dispersed and attached to the host plant surface, the spore/s germinate to form a specialised structure called the appressorium, this is characterised by turgor pressure and production of a mucilaginous matrix that anchors the biotrophic fungus on the plant surface(Shi, Li, & Zhao, 2023). An infection peg -sharp, pointed narrow hyphae is developed to extend into the intercellular spaces. The appressorium turgor pressure provides the force and the anchorage to allow the extension (Thilini Chethana et al., 2021). At this stage, the hyphae extentions into the intercellular spaces develop into the specialised thin elongated haustoria (somatic hyphae) which increases surface area for nutrient absorption from the host plant, the haustoria are able to directly penetrate the host cells to extract nutrients (Thilini Chethana et al., 2021). Having accessed and utilised nutrients, the biotroph fungus becomes fit to further develop and sporulate for extended infection.

During the different life cycle stages of these fungi biotrophic pathogens, they are capable of secreting effector proteins, host defence suppression, host metabolic and related physiological processes manipulation in a phased approach to support coexistence and absorption of nutrients for their survival (Mapuranga et al., 2022).

On the other hand, necrotrophic plant pathogens generally pose similar life cycle and infection strategies to biotrophs, but are aggressive killers, at certain stages of their life cycle in the host plants for example after penetration (Thilini Chethana et al., 2021), necrotrophs introduce all effectors at ago accompanied with toxins (as for the biotrophs, the infections are phased) such as the non-pore and pore-forming toxins which degrade the cell wall and intracellular metabolic processes respectively. The combined attack strategies of necrotrophs overwhelm the plant's first line and systemic acquired defence responses which downplays metabolic cellular processes and subsequently leads to cell death and necrosis of tissues. In contrast to biotrophs, necrotrophs aggressively produce lytic enzymes in large amounts (Laluk & Mengiste, 2010).

For example, the common necrotroph of *Botrytis Cineria*, its infection, colonization, and suppression of host defences is influenced by a number of lytic enzymes, reactive oxygen species (hydrogen peroxide), toxins, necrosis-inducing factors. B. Cineria directly secretes oxidases, cutinases, and lipases in the intercellular spaces to aggressively and actively dismantle

the plant cutin and wax layers. After weakening the first line mechanical defence systems ie the cuticle, cell wall and membrane. *B. cinerea* then secretes oxalic acid (OA) which promotes infection by creating an optimal acidic environment for increased activity of secreted enzymes to further facilitate disruption and tissue death (Laluk & Mengiste, 2010). All these combined effections occur at once to overwhelm the plant defence systems at ago unlike in biotrophs.

The hemi-biotrophs have both biotrophic and necrotrophic life phases, they begin with a biotrophic phase from which they switch to necrotrophy. Hemi-biotrophs that stay longer in a biotrophic phase are characterised as biotrophs while those that take longer in the necrotrophic phase are characterised as necrotrophic leaning. After switching to the necrotrophic phase, pathogens produce large amounts of toxins and cell wall degrading enzymes to then start feeding on dead and decaying tissues (Andrew, Barua, Short, & Kohn, 2012).

Even when we have this triad of plant pathogens, they are and always have been specific in relation to the plant hosts, a number of factors determine and influence the pathogen specificity to the host plants including target nutrients, target tissue, and compatibility of virulence genes among others. For example, Pseudomonas Syringea has a nutritive interest in an amino acid called tyrosine common and abundant in tomatoes which P. Syringea largely attacks. Another example is *Xylella Fastidiosa*, a selective bacterial pathogen that only attacks the xylem tissue of a host plant (Mansfield et al., 2012).

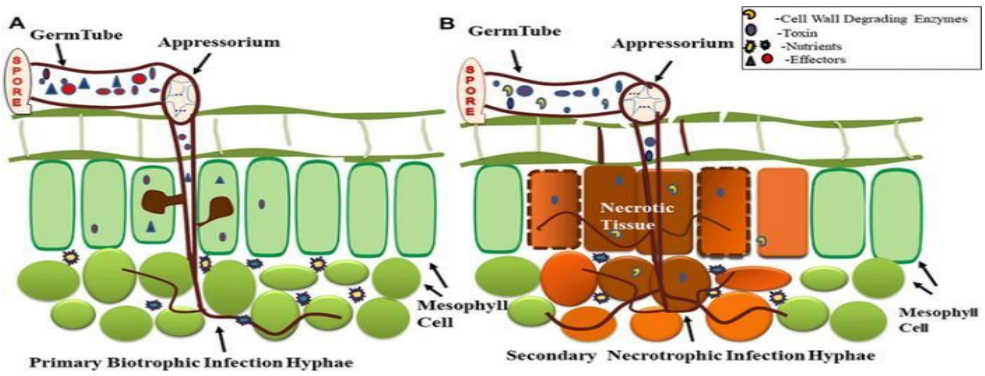


Figure 3: The transitioning mechanism of the pathogen from biotroph to necrotroph.

1.1 Host Specificity and Defense Mechanisms

Host specificity; the ability of some microbes (pathogens) to infect/cause disease to particular species of plants (Borah *et al*., 2018). This phenomenon is crucial for understanding plant pathology and the dynamics of plant diseases. According to Borah *et al*. (2018), host specificity

can manifest not only at the species level but also among different members within a specific plant species. This means that while a pathogen may be capable of infecting a certain plant species, it may only affect specific cultivars or varieties within that group. However, sometimes it's only some members of a specific plant species that get infected.

Types of hosts–pathogen interactions
Host-pathogen interactions can generally be categorized into two main types:

- Generalists
- Specialists

1.1.1 Generalists: Generalist pathogens have a broad host range and can infect multiple plant species. These pathogens are often less specialized in their infection strategies, allowing them to adapt to various hosts. For example, some fungal pathogens like Botrytis cinerea can infect numerous plant species, causing gray mold disease across different crops. The ability of generalists to infect multiple hosts can lead to widespread outbreaks, especially in agricultural settings where diverse crops are grown in proximity (Bi et al., 2023). For example, *Cucumber mosaic virus* infects over 1000 species in 85 plant families.

1.1.2 Specialists: Specialist pathogens, on the other hand, are adapted to infect only one or a few closely related plant species. These pathogens possess highly specialized virulence factors that allow them to effectively exploit specific host defenses. An example of a specialist is Puccinia graminis, the causal agent of wheat stem rust, which primarily targets wheat and some related grasses but does not infect other unrelated plants. The narrow host range of specialists often results from co-evolution with their hosts, leading to finely tuned interactions that enhance their ability to overcome specific plant defense mechanisms(Li et al., 2020). For example, *Citrus tristeza virus* infects only a few species in the *Citrus* genus (Gergerich & Dolja, 2006).

1.2 Pathogen Host Specificity

Pathogens exhibit host specificity by targeting particular plants, which is often a result of coevolution between the pathogen and its host (Soni *et al*., 2024). This specificity is largely mediated through specialized virulence factors that allow pathogens to overcome plant defenses. For example, certain fungi and bacteria have developed effector molecules that can alter host cellular functions to facilitate infection. These effectors can change host metabolism to promote pathogen persistence or decrease plant defense responses (Fig. 5).

The interaction between pathogens and their specific hosts is influenced by various factors, including the genetic makeup of both the pathogen and the plant (Lorrai & Ferrari, 2021).

Pathogens may possess a suite of genes that encode for these virulence factors, which are selectively advantageous when infecting compatible hosts. This leads to a scenario where a pathogen may be highly effective at infecting one species of plant while being ineffective against another, highlighting the importance of host specificity in disease dynamics.

1.3 Mechanisms of host specificity

- Recognition systems - pathogens possess specialized proteins (effectors) that are secreted into host cells to manipulate host cellular processes to facilitate infection
- Genetic compatibility - both (pathogen & host) contribute to host specificity; pathogens often have a narrow range of compatible hosts due to specific receptor-ligand interactions at the cellular level.
- Environmental factors - temperature, humidity & soil composition
- Virulence ability where pathogens release molecules (enzymes) that break down plant cell walls & enhance their ability to cause disease (Soni et al., 2024)

1.4 R - proteins in plants

R proteins, or resistance proteins, are critical components of the plant immune system. The R proteins are most commonly intracellular NB-LRR proteins or extracellular LRR-carrying receptors, but other protein types can also be classified as R proteins. Some NB-LRR proteins are present in both the cytoplasm and the nucleus. Plant NB-LRR immune receptors have evolved the ability to specifically recognize a wide range of effector proteins from different pathogens (Caplan et al., 2008).

1.5 R Proteins and Plant Defense Mechanisms

In response to pathogen attack, plants have evolved sophisticated defense mechanisms, one of which involves R proteins (Resistance proteins). These proteins play a crucial role in recognizing specific pathogen effectors.

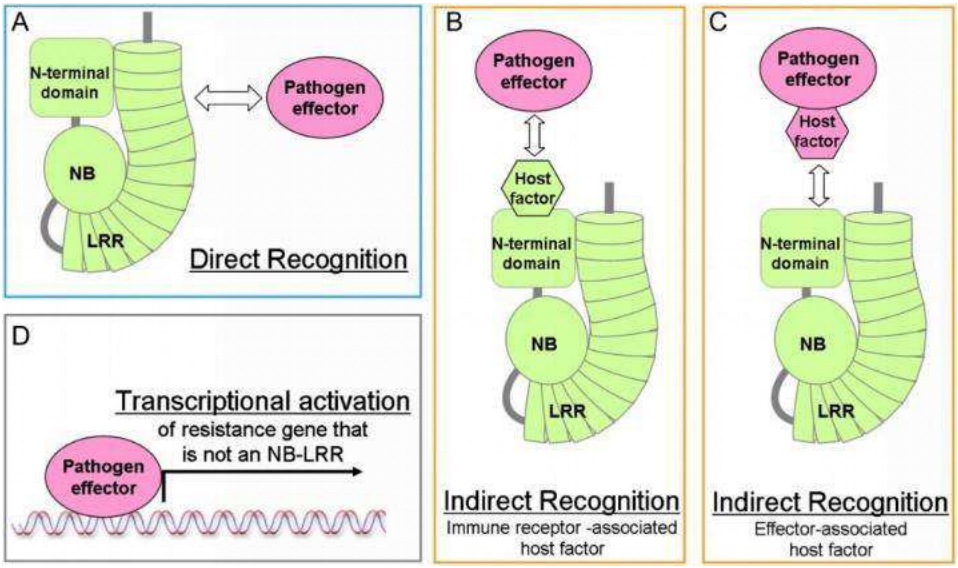


Figure 5: Modes of pathogen recognition (A) Direct recognition, (B) and (C) Indirect recognition (D) Transcriptional activation with Non-NB-LRR R-gene.

When an R protein detects an effector from a corresponding pathogen, it triggers a cascade of defense responses known as gene-for-gene resistance. This recognition typically leads to localized cell death (hypersensitive response) at the site of infection, effectively limiting pathogen spread.

R proteins are often located within plant cells and can recognize either directly or indirectly the presence of pathogenic effectors. The activation of R proteins initiates various defense pathways, including the production of antimicrobial compounds, reinforcement of cell walls, and signalling for systemic acquired resistance (SAR), which prepares other parts of the plant for potential attacks.

1.6 Virulence Factors influencing pathogen Host specificity

Virulence factors are molecules (enzymes) that break down plant cell walls produced by pathogens that enhance their ability to cause disease (Soni et al., 2024; Speth et al., 2007). Effector proteins manipulate host cellular or defence mechanisms to facilitate infection. Production of secondary metabolites (mycotoxins) which inhibit plant growth or induce cell death in susceptible plants (Toruño et al., 2016). Some pathogens can produce compounds that mimic plant hormones. This alters growth patterns to favor pathogen survival and reproduction while compromising the plant's defences.

1.5 The Gene for Gene model

The gene-for-gene relationship was revealed by Flor (1942). They explored that, the inheritance of resistance genes in hosts and pathogens *Avr* gene capability to cause disease is controlled by a pair of matching genes in *Melampsora lini* (fungus) of flax (*Linum usitatissimum*). The resistance (*R*) gene is produced by plants and the *Avr* gene is produced by the pathogens. Plants produce specific *R* gene products that are resistant toward a pathogen which produces corresponding *Avr* gene products. The gene-for-gene relationship is widespread and the most significant aspect of plant disease resistance (Fig. 1)

Illustration of the experiment on genotypes and Pathogen

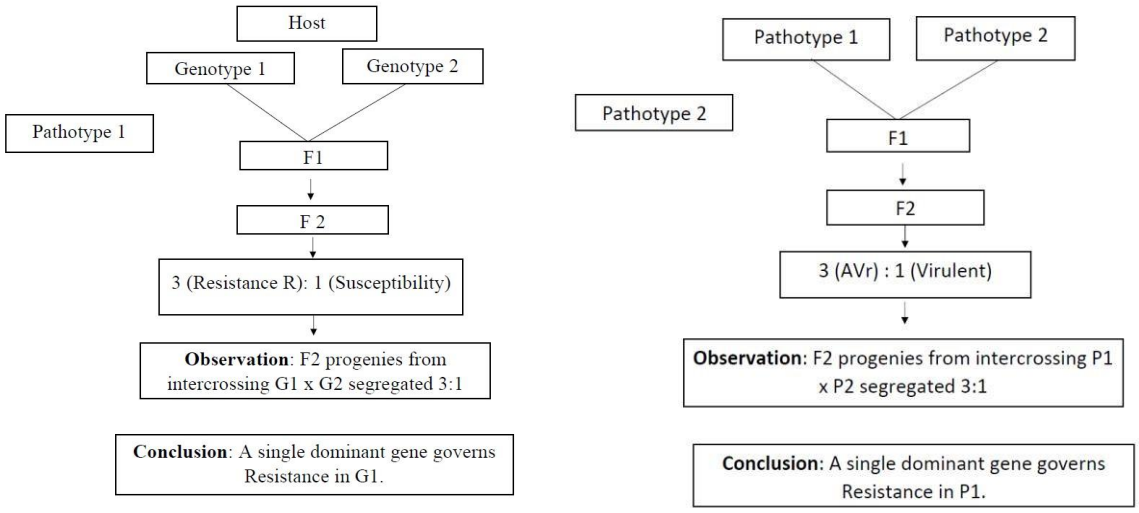


Figure 1: Gene for Gene experiment on matching dominant genes by Flor 1942.

The experimentation is depicted in Figure 1. After screening the two flax plant genotypes for Melampsora lini (pathotype 1), it was determined that one was vulnerable to the disease and the other was resistant. By crossing the genotypes to produce F1 and selfing to produce F2 plants, the resistance architecture was better understood. Resistance against vulnerable plants was found to be 3:1 in that order. Consequently, it was determined that a single dominant gene controlled the resistance. To have a better understanding of the pathogen side, a similar scenario was conducted for the pathogen. To create F1, the two pathotypes were crossed, and to create F2, they selfed. The progeny produced by the selfing process demonstrated that the F2 progenies segregated at three avirulent genes to ratio one of the virulent genes. One dominant gene was found to be responsible for the resistance. The relationship between the resistant genotype and plant genes was then investigated further. The final intriguing finding revealed that the dominant gene in plants and the avr gene matched similarly. As a result, the gene-forgene model was created.

This experiment showed that when a plant has a dominant gene matching avr dominant in the pathogen, this results in a lock and key model and the compatibility brings about resistance (Fig. 6).

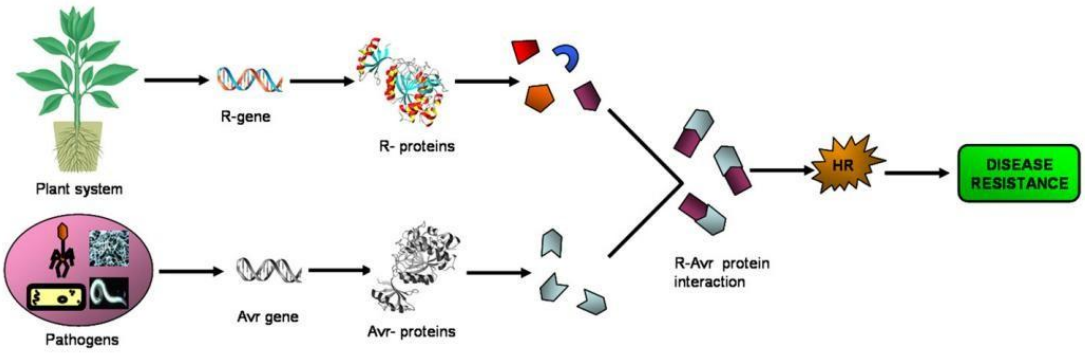


Figure 6: The matching gene for gene model that results in disease resistance.

2.0 Mechanism of Defence in Plant

Plants developed a two-layered surveillance system to defend themselves against infectious pathogens. Plants recognise conserved molecules in plant pathogens called pathogen-associated molecular patterns (PAMPs) through cell surface-localized pattern recognition receptors (PRRs) to activate PAMP-triggered immunity (PTI) (Chang et al., 2022). PTI responses include production of reactive oxygen species (ROS), activation of mitogen-activated protein kinase (MAPK) cascades, changes in the intracellular calcium concentration and transcriptional reprogramming of immunity-related genes, cell wall callose deposition, stomatal closure, and moderate inhibition of pathogen growth (Yu et al., 2021). Through these responses, PTI provides an initial layer of defence against pathogens (Figure 7a).

2.1`Pathogen Associated Molecular Pattern PAMP

Pathogen-triggered immunity is initiated by two different types of pattern recognition receptors (PRR), which are a receptor-like protein (RLP) and a receptor-like kinase (RLK). PTI triggers an influx of calcium ions (Ca+), mitogen-activated protein induction, the production of reactive oxygen species (ROS), salicylic acid production and signaling, nitric oxide synthesis, and cell wall strengthening. PRR detects a microbe-associated molecular pattern (MAMP), a pathogenassociated molecular pattern (PAMP), and a damage-associated molecular pattern (DAMP)(Figure 7a).. Upon pathogen (viruses, fungi, bacteria etc) infection, the PTI of the host cell is initiated, which confers immunity against the pathogen infection. An intricate signaling network of cytoplasmic kinase receptors and co-receptors, such as Somatic Embryogenesis

Receptor Kinases (SERKs) and LRR-RLK brassinosteroids Insensitive1 (BRI1)-Associated Kinase 1 (BAK1/SERK3), are involved. Similar to DAMP, viral PAMPs, known as VAMPs, are activated to resist viral infection. However, some viruses surpass this defense system by encoding suppressors for PTI. These reported viruses are the Cauliflower mosaic virus (CaMV), Rice stripe virus (RSV), and Plum pox virus (PPV) (Akbar et al., 2022)

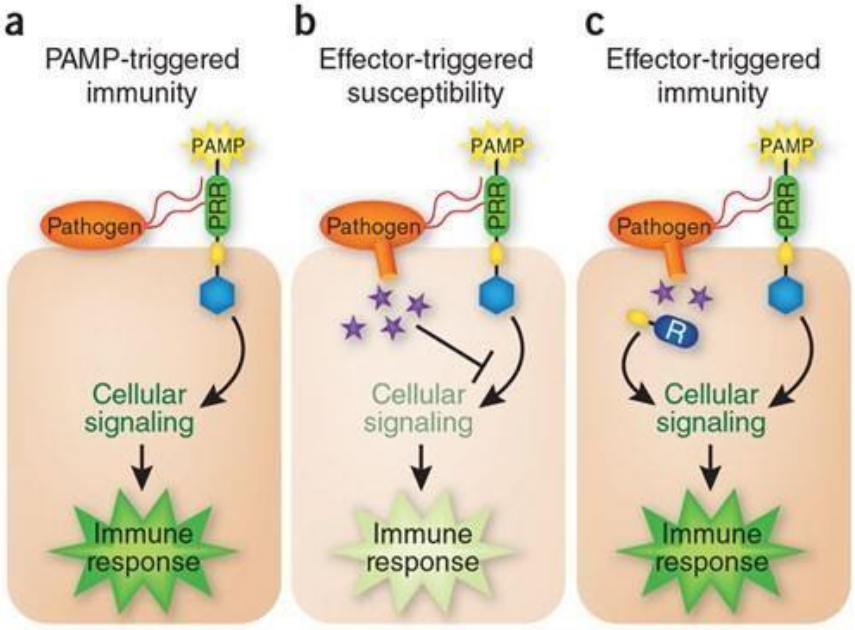


Figure 7: Pathogen-triggered immunity PTI and Effector triggered immunity defence mechanism in plants against pathogens infections (a) PAMP mechanism (b) Effector triggered susceptibility (c) Effector triggered immunity (Fu & Dong, 2013)

2.2 Effector-Triggered Immunity

Host-adapted pathogens such as *Pseudomonas syringae* DC3000 have evolved strategies to undermine PTI by delivering virulence factors (effectors) into plant cells. In turn, plants have evolved intracellular resistance proteins (mainly pathogen-specific nucleotide-binding leucinerich repeat (NB-LRR)-type immune receptors) capable of sensing specific effectors. This layer of defence, termed ETI, initiates an incompatible interaction characterised by the induction of salicylic acid and systemic acquired resistance, defence gene transcription, as well as a hypersensitive response involving programmed cell death that limits pathogen spread from the initial point of infection to other uninfected parts (Figure 7b and 7c). (Yu et al., 2021)

PTI and ETI share some common immune responses, such as the generation of apoplastic ROS, activation of Ca2+ and MAPK cascades, and production and secretion of antimicrobial compounds. Although the same or similar signalling components or enzymes are involved in PTI and ETI, the dynamics and strength of the activated responses are different. The activated immune responses during ETI are generally more prolonged and robust than those during PTI.

An avirulent pathogen not only triggers defence responses locally but also induces the production of signals such as salicylic acid (SA), methyl salicylic acid (MeSA), azelaic acid (AzA), glycerol-3-phosphate (G3P), and abietane diterpenoid dehydroabietinal (DA). These signals then lead to systemic expression of the antimicrobial PR (pathogenesis-related) genes in the uninoculated distal tissue to protect the rest of the plant from secondary infection (Fu & Dong, 2013).

2.3 Case one on tomato for understanding the ETI AND PTI mechanism

Bacterial speck disease is caused by the hemi-biotrophic bacterial pathogen Pseudomonas syringae pv. tomato (Pst) in many plant tissues of the Solanaceae family, including tomatoes (Solanum lycopersicum). The molecular processes of plant-pathogen interactions have been extensively studied using the tomato-Pst pathosystem as a model.

According to the research, the main elicitors of PTI in tomatoes are flagellin-derived MAMPs in Pst. The plant PRRs Flagellin sensing 2 (FLS2) and Flagellin sensing 3 recognise two MAMPs from the motility-associated flagellin protein produced by the fliC gene: Flg22 and flgII-28.

During the infection process, Pst DC3000 introduces a set of 36 virulence-promoting effectors into the plant cells to defeat PTI. Two of the effectors, AvrPto and AvrPtoB, decrease PTI and encourage bacterial virulence and proliferation by interfering with PRR functions and plant immune responses early in the tomato-Pst relationship. Tomatoes developed the Pto kinase in response to Pst effectors. This enzyme interacts with AvrPto/AvrPtoB and cooperates with the NLR protein Prf to activate ETI (Fu & Dong, 2013).

2.4 Hypersensitivity response (Plant cell death – observed in PTI and ETI)

The hypersensitive response, or HR, is a form of cell death often associated with plant resistance to pathogen infection. Reactive oxygen intermediates and ion fluxes are proximal responses probably required for apoptosis as defined in plant systems. Effector-triggered immunity (ETI) is often associated with the hypersensitive response (HR), a form of rapid plant cell death that may restrict pathogen growth (Fig. 9). Recognition of a pathogen attack triggers a hypersensitive reaction (HR) in the plant, which includes the generation of reactive oxygen intermediates (ROIs) and local cell death (Fig. 9). HR is considered to be one of the most important factors in impeding growth of biotrophic pathogens. Pathogens that do not trigger HR can establish compatible interactions with the host, resulting in disease (Govrin & Levine, 2000; Morel & Dangl, 1997; Thakur et al., 2019).

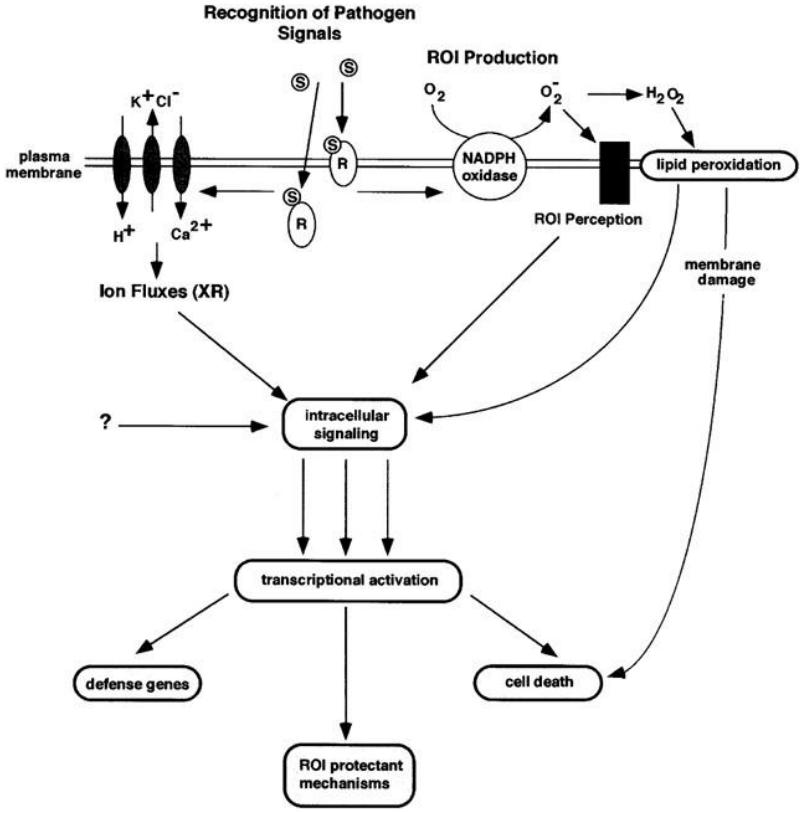


Figure 8: Transduction pathways in plants leading to hypersensitivity (Morel & Dangl, 1997)

A case study:

HR is triggered by AvrRps4, which is derived from the bacterial pathogen *P. syringae* and is recognised by resistance to P. syringae expressing AvrRps4 (RPS4) in plants. Thus, mechanisms leading to HR are different among different ETI events (Govrin & Levine, 2000)

By contrast, a different group found that autophagy is involved in the negative regulation of cell death by negatively regulating salicylic acid (SA) signalling and SA treatment induces autophagy, suggesting a role for autophagy in the negative feedback of SA signalling. A mutant with a defect in autophagy showed the pathogen-induced spread of cell death, which was dependent on SA signalling. Interestingly, the commonly used flg22 derived from Pseudomonas aeruginosa does not induce cell death, whereas flagellin derived from P. syringae pv. tabaci 6605 did induce cell death. Thus, the plant cell death response can be mediated by different signalling mechanisms and occurs in PTI as well as in ETI (Fig. 9) (Govrin & Levine, 2000)

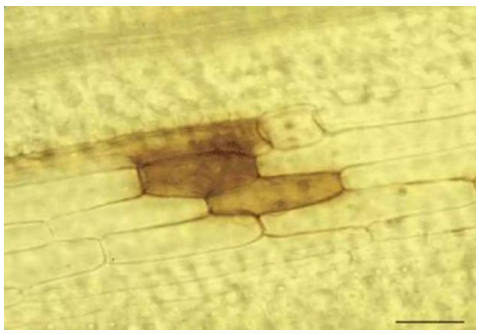


Figure 9: The symptoms after hypersensitivity initiate cell death

3.0 System Acquired Resistance

In plants, localized pathogen attack, whether inducing an HR or moderate symptoms of disease, often elicits broad-spectrum immunity to reinfection throughout the whole body. This type of systemic plant immunity is called systemic acquired resistance (SAR) (Fu & Dong, 2013).SAR is a broad spectrum, with no specificity to the initial infection. An avirulent pathogen causing local programmed cell death can induce SAR through the generation of mobile signals, accumulation of the defense hormone salicylic acid, and secretion of the antimicrobial PR (pathogenesis-related) proteins. Consequently, the rest of the plant is protected from secondary infection for a period of weeks to months. SAR can even be passed on to progeny through epigenetic regulation. This occurs through signalling from the point of infection in the plant to other uninfected parts of the plant.

3.1 Mobile signals

3.1.1 Methyl Salicylic Acid

Salicylic Acid has been recognised as the mobile signal for SAR, but recent research has shown that MeSA might be the signal (Figure 10). This idea came from a study of SABP2 (salicylic acid–binding protein 2), a methyl salicylate esterase, which converts the biologically inactive MeSA to the active SA. The authors found that after they inoculated the wild-type tobacco rootstock with tobacco mosaic virus, the SABP2-silenced scion failed to accumulate SA or develop SAR. (Fig. 10).

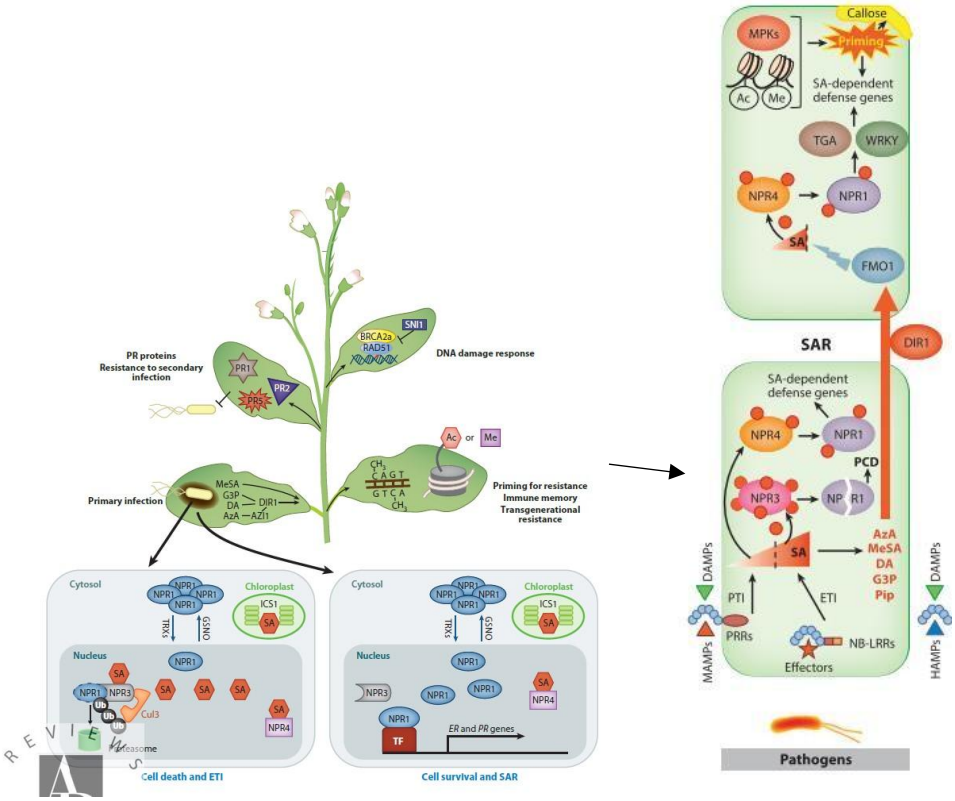


Figure 10: Systemic acquired resistance in the plant (a) Overall signalling occurrence in the plant after initial pathogen infection (b) The transition of the signal to the secondary plant's regions with no infections (Fu & Dong, 2013)

They proposed that the locally synthesized SA is first converted to MeSA through the activity of methyltransferase and then mobilized to the systemic tissue, where it is converted back to SA by SABP2. In support of this hypothesis, silencing the SA methyltransferase in tobacco compromised SAR. A different study, however, found that the Arabidopsis SA methyltransferase knockout mutant (bsmt1) was intact in mounting SAR (4). The authors of this study also showed that although the MeSA level increased upon SAR induction, the majority of it was released into the atmosphere. These data confirmed the validity of MeSA as a general mobile signal for SAR before being converted to Salicylic Acid.

4.0 Induced Acquired Resistance

The term induced resistance is a generic term for the induced state of resistance in plants triggered by biological or chemical inducers, which protects non-exposed plant parts against future attack by pathogenic microbes and herbivorous insects. Plants can develop induced resistance as a result of infection by a pathogen, in response to insect herbivory, upon colonization of the roots by specific beneficial microbes or after treatment with specific chemicals (Figure 8). The induced state of resistance is characterized by the activation of latent

defense mechanisms that are expressed upon a subsequent challenge from a pathogen or insect herbivore. Induced resistance is expressed not only locally at the site of induction but also systemically in plant parts that are spatially separated from the inducer, hence the term ISR. Generally, induced resistance confers an enhanced level of protection against a broad spectrum of attackers. Induced resistance is regulated by a network of interconnected signaling pathways in which plant hormones play a major regulatory role (111). The signaling pathways that regulate induced resistance elicited by beneficial microbes, pathogens, and insects share signaling components (Pieterse et al., 2014). Therefore, we first highlight the important principles of pathogen- and insect-induced resistance before reviewing the current status of ISR mediated by beneficial soilborne microbes

Because of its broad-spectrum effectiveness, rhizobacteria-mediated ISR was initially thought to be mechanistically similar to pathogen-induced SAR. However, Hoffland et al. (54) provided evidence that in radish *P. fluorescens* WCS417r-ISR against *F. oxysporum* developed without accumulation of the PR proteins that are characteristic of SAR. Similarly, *P. fluorescens* WCS417r-ISR in Arabidopsis developed without PR gene activation in systemic leaf tissue. Testing of transgenic Arabidopsis NahG plants that are unable to accumulate SA provided genetic evidence that an SA-independent signaling pathway mediates P. fluorescensWCS417rISR and does not coincide with enhanced SA levels. The same appeared to be true for the ISRinducing PGPR *Pseudomonas putida* WCS358r. It was thus concluded that different signaling pathways regulate rhizobacteria-mediated ISR and SA-dependent SAR. This was supported by observations that although both rhizobacteria-mediated ISR and pathogen-induced SAR are effective against a broad spectrum of attackers, their ranges of effectiveness are partly divergent (Pieterse et al., 2014).

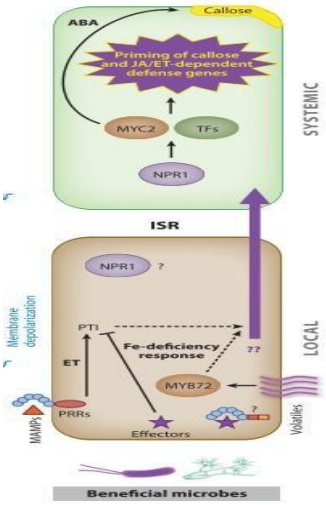


Figure 11: Induced acquired resistance in the plant for defence (Pieterse et al., 2014).

5.0 VIRUSES

Plant viruses are obligate intracellular parasites that rely almost exclusively on the host cell to accomplish their infection cycle, e.g., genomic information expression, genome replication, and intercellular movement. During the infection, a comprehensive and competitive protein-protein and protein-nucleic acid interaction network is formed between the plant and the virus (Wu et al., 2019)

Viruses are made of, the genome that is made of nucleic acid (DNA or RNA), and a protective shell that is made of protein. In addition, some virus particles are enveloped by an outer membrane containing lipids and proteins -lipoprotein membrane (Balaji, 2020)

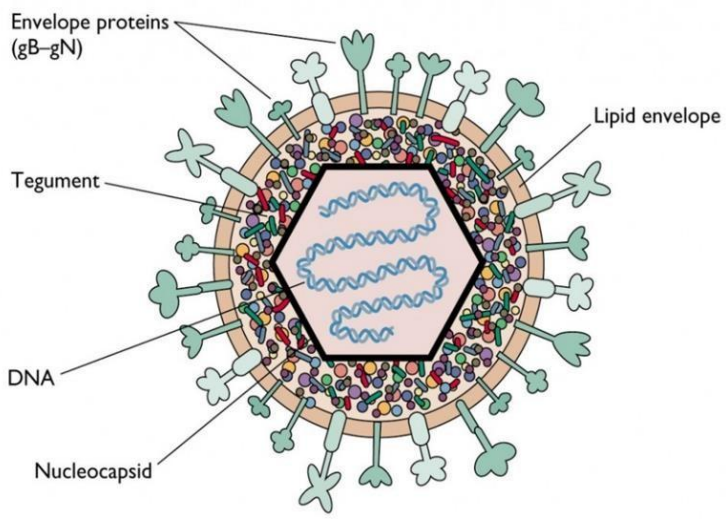


Figure 12: Viral structure and genome

Plant viruses exhibit a spectrum of host range and specificity, determined by factors including the virus's genetic makeup and the host's cellular environment. Some viruses are specialists, infecting only a narrow range of plant species, while others are generalists, capable of affecting a broad spectrum of hosts. The host range is often dictated by the virus's ability to bypass the host's initial defense barriers and establish an infection (*Understanding Plant Viruses*, 2024) The specificity of plant viruses is connected to the compatibility of viral proteins with plant host cellular machinery. Viruses interact with specific plant host factors to replicate and move within, and these interactions are highly selective. For example, the Potato Virus Y (PVY), has evolved mechanisms to exploit the host's cellular pathways, enabling it to effectively replicate within its hosts such as tomatoes and potatoes.

5.1 Types of viruses

A) **Helical viruses** are made up of a single type of protein subunit stacked around a central axis to form a helical structure. The helix may have a hollow center, which makes it look like a hollow tube. This arrangement results in

i. rigid rods

ii. flexuous filaments.

These virions can be anything from short and very rigid, to long and very flexible. The tobacco mosaic virus (TMV) is an example of a helical virus (Gergerich & Dolja, 2006)

B) The Spherical viruses – are enclosed in an envelope with sugar-proein that assists in attachment and penetration I to plant host cells, such as tomato spotted Wilt Virus (TSWV)

C) **Polyhedral viruses** – such as denovirus, an icosahedral virus is a three-dimensional shape made up of 20 equilateral triangles. Viral structures are built of repeated identical protein subunits. Examples include Groundnut Rosette Virus (GRT) and Cowpea Aphid-Borne Mosaic Virus (CABMV).

D) **Complex viruses** possess a capsid which is neither purely helical, nor purely icosahedral, and which may have extra structures such as protein tails or a complex outer wall. Viral protein subunits will self-assemble into a capsid, but the complex viruses DNA also codes for proteins which help in building the viral capsid. Many phage viruses are complex-shaped; they have an icosahedral head bound to a helical tail. The tail may have a base plate with protein tail fibers. Some complex viruses do not have tail fibers(*7.8*, 2016). Examples are bacteriophages such as PBI which affects *Pseudomonas aeruginosa* a soilborne pathogen of many plants, Phi6 bacteriophage which infects *Pseudomonas syringae* a pathogen of tomatoes, beans and maize.

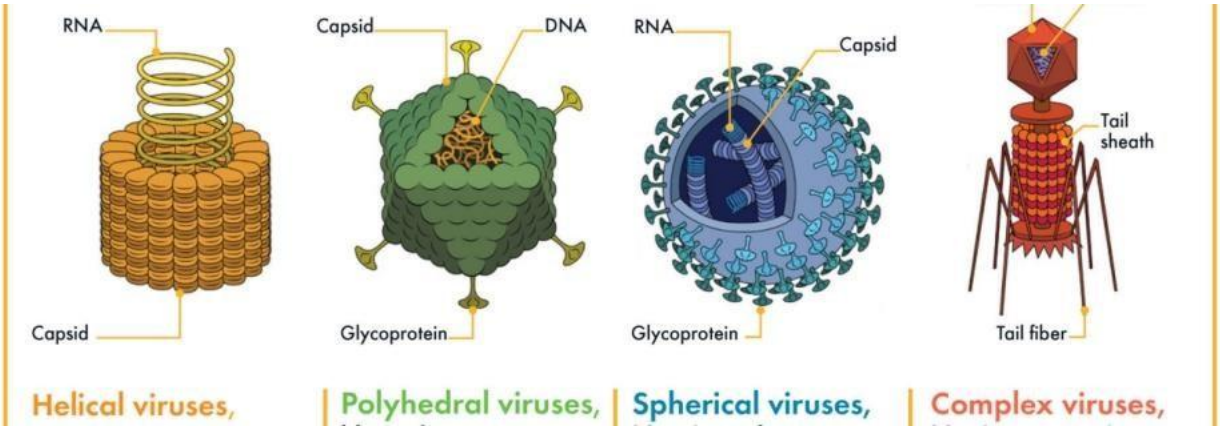


Figure 13: Types of viruses

5.2 Viral Life Cycle

For the virus to reproduce and establish infection, it must have access and thereby enter cells of the plant host, then use those cells' materials. To enter the cells, proteins on the surface of the virus interact with proteins of the host cell (Heinlein, 2015)

Attachment - occurs between the viral particle and the host cell membrane. A hole forms in the cell membrane, then the virus particle or its genetic contents are released into the host cell, where viral reproduction may commence (Heinlein, 2015)

Penetration – the protein pushes the nucleic acid strand into the plant cell, at this stage, a distinction between susceptibility and permissibility of a host cell is made. Permissibility determines the outcome of the infection. After control is established and the environment is set for the virus to begin making copies of itself

Replication – the viruses' nucleic acid uses the plant cell DNA to make many new nucleic acid strands and protein sheathes. A virus must take control of the host cell's replication mechanisms and replication occurs quickly by the millions.

Assembly – the nucleic acid and protein assemble into millions of new virus copies.

Release – the viruses leave the cell. After a virus has made many copies of itself, it usually has exhausted the cell of its resources. The host cell is now no longer useful to the virus, therefore the cell often dies and the newly produced viruses must find a new host

Transmission – the viruses move using a vector to new cells to infect. Viruses can be spread by direct transfer of sap, and by contact of a wounded plant with a healthy one. Such contact may occur during agricultural practices, when damage is caused by tools or hands, or naturally, when an animal feeds on the plant. Generally Tobacco mosaic virus (TMV) is transmitted via sap (Gergerich & Dolja, 2006; Heinlein, 2015).

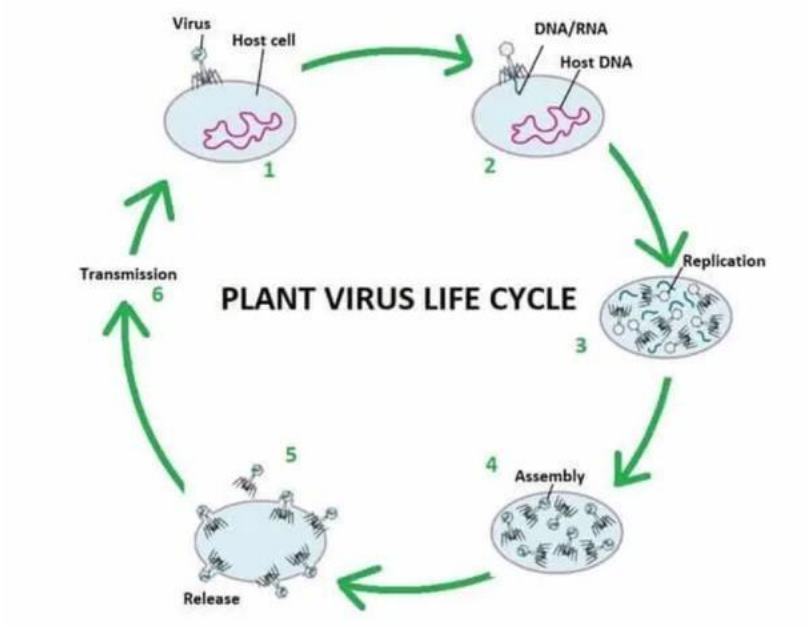
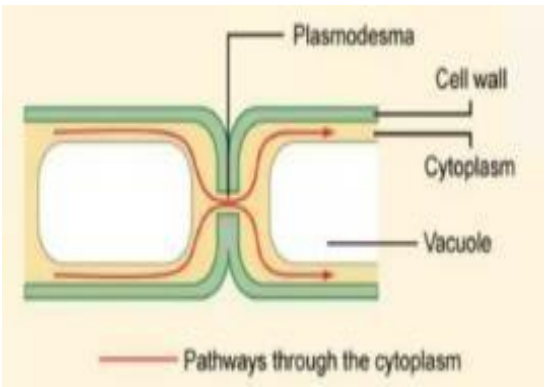


Figure 14: Virus life cycle (Source: https://cropnuts.com/an-introduction-to-plant-viruses/)

5.3 Viruses' movement through Plant membranes

In plant virus infections, once a virus genome enters the initial cell(s), the further systemic infection involves cell-to-cell genome movement via plasmodesmata (PD) connecting neighbouring cells in plant tissues (Fig. 8a) and subsequent long-distance movement via the vascular system (Fig. 8b). In general, the processes of virus spread from the initially infected cell throughout the plant, as well as viral genome transfer between host organisms, are subject to the major selective forces in the evolution of viruses (Heinlein, 2015)(Fig. 8b).



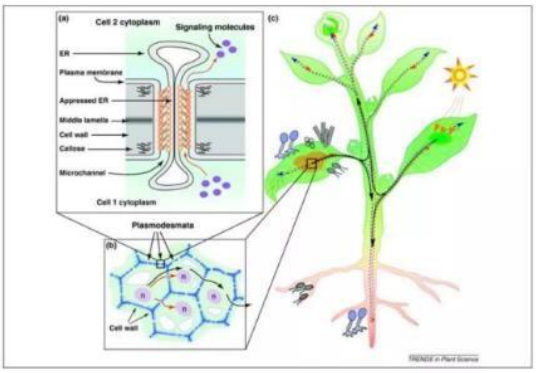


Figure 15: Virus movement through the plant (a) slow movement through plasmodesmata (b) fast movement through vascular bundle (Phloem).

As a result, viruses have evolved combinations of at least several genes whose products interact with cellular components to ensure the production of viral genome progeny, the spread of this

progeny throughout the organism and, in most cases, the protection of viral genomic nucleic acids to be transferred to other plants. One group of viral genes is significantly conserved in evolution. In general, these most conserved genes encode the proteins whose activity is directly involved in the replication of the viral genomes. Other viral genes are less conserved, they were even found to be completely unrelated to non-replicative genes in other groups of plant viruses. In particular, these genes encode evolutionarily from very early evolved capsid proteins (CP), which, together, with replication proteins, constitute the so-called "Virus Hallmark Proteins", movement proteins (MP), which first evolved in streptophyta, viral suppressors (VSR) of posttranscriptional (PTGS), and transcriptional (TGS) gene silencing. The movement through plant plasmodesmata is carried out with the help of movement proteins and mostly DGBp1 and DGBp2 proteins (Navarro et al., 2020; Navarro & Pallás, 2017). In general, to neutralize the effect of RNAi, eukaryotic viruses have evolved to acquire VSRs that significantly reduce the effect of plant antiviral defence.

5.4 RNA interference (RNAi)

5.4.1 RNAi Silencing Mechanism in Plant

Plant viruses are devastating plant pathogens that severely affect crop yield and quality. Plants have developed multiple lines of defence systems to combat viral infection. Gene silencing/RNA interference is the key defence system in plants that inhibits the virulence and multiplication of pathogens.

The general mechanism of RNAi involves (Liu et al., 2021):

(i) the transcription and cleavage of dsRNA into small RNA molecules, such as microRNA (miRNA), or small interfering RNA (siRNA),

(ii) the loading of siRNA/miRNA into an RNA Induced Silencing Complex (RISC),

(iii) complementary base pairing between siRNA/miRNA with a targeted gene, and

(iv) the cleavage or repression of a target gene with an Argonaute (AGO) protein. This natural RNAi pathway could introduce transgenes targeting various viral genes to induce gene silencing. Different RNAi pathways are reported for the artificial silencing of viral genes. These include Host-Induced Gene Silencing (HIGS), VirusInduced Gene Silencing (VIGS), and Spray-Induced Gene Silencing (SIGS). There are significant limitations in HIGS and VIGS technology, such as lengthy and timeconsuming processes, off-target effects, and public (Akbar et al., 2022; Liu et al., 2021)

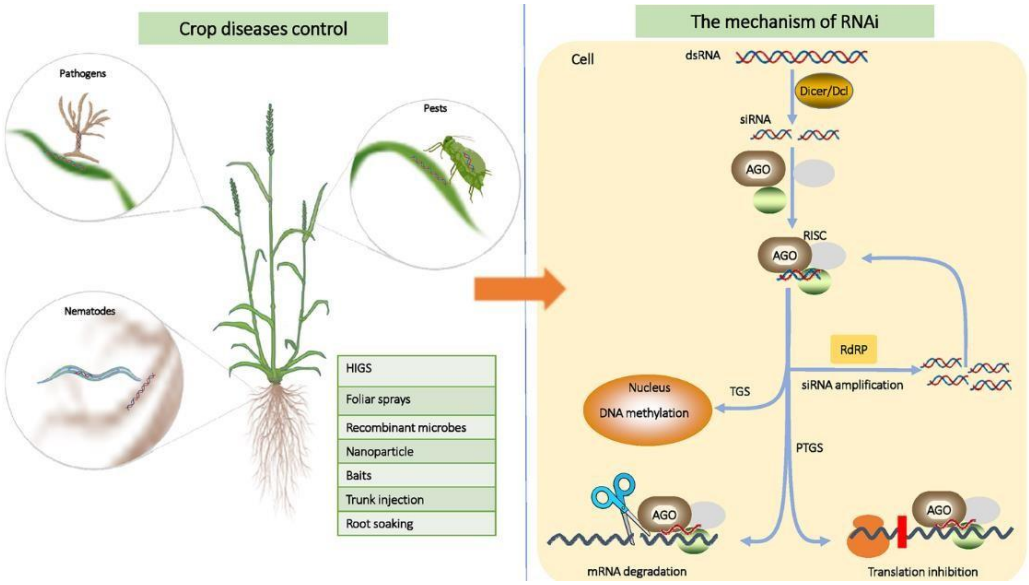


Figure 16: RNAi silencing mechanism adopted in suppressing pathogen infection (Liu et al., 2021)

6.0 Nematodes

Nematodes are thread-resembling morphologically round-bodied worms (Fig. 4), they are microscopic and some can grow up to a size visible by naked human eyes (Decraemer, Karanastasi, Brown, & Backeljau, 2003). Nematodes exist in several species including plant parasitic nematodes that cause serious mechanical and physiological damage to plants of economic interest



Figure 17: A typical structure of nematode.

Parasitic nematodes have over time developed specialized features to suit their mode of life and facilitate nutrient uptake from host plants, some of these features include; the stylet, an extension from the buccal area, which is an elongated rigid narrow and pointed-like structure which suites it to pierce and extend into the target root part/site of nutrient acquisition. The stylet is hollow to allow in flow of absorbed nutrients (Jaubert et al., 2002). It is also used to release digestive and effector matrices into the target host. The pharynx, a muscular body to which the stylet tube connects in the oesophagus, the pharynx provides a pumping force to facilitate the inflow of nutrients from the source absorption (Song & Avery, 2013). The digestive glands of the nematodes, secrete required digestive juices to induce metabolic reactions on the absorbed nutrients for assimilation by the nematodes.

For nematodes to successfully attack and overcome the defence systems of the host plants, they have evolved to secrete and release necessary effector proteins that either suppress, manipulate, and or overwhelm the different plant defence lines. Such proteins include protease inhibitors that inhibit protein synthesis and failure to activate defence systems, cellulases and pectinases that break the cellulose in the cell walls and the pectin that binds cell wall tissues together weakening the plant first-line mechanical defence for easy invasion (Giebel, n.d.). The Hs13B14 effector proteins produced by beet cyst nematodes, the effector proteins suppress the salicylic and jasmonic acid receptor sites which are responsible for activating both Systemic

Acquired Resistance and Induced Systemic Resistance respectively. The Hs13H05 which regulates the transcription process of the plants to minimise phenotypic defence responses such as strengthening of the cell wall tissues(Mitchum et al., 2013).

Because of their unique physiological and morphological characteristics, nematodes can live and feed in a variety of soil and root ecosystem categories. For instance, they can be ectoparasites, which live and feed on the surface of the roots, endoparasites, which live inside the roots (Ankrom et al., 2020), sedentary nematodes, which remain in one location and feed there (they can be either ecto-sedentary, which means they are on the root surface but not moving, or endo-sedentary, which means they are in the root surface but not moving, and migratory nematodes that move from one location to another, such as those that can feed from the epidermal surface but from the xylem tissues of the roots. The migratory nematodes can also be ecto-migratory moving on the surface or endo migratory moving within the root's inner tissues (Jaubert et al., 2005).

7.0 Conclusion

Plants employ numerous strategies, including pathogen-triggered immunity and effectortriggered immunity, which include the influx and efflux of ions such as Ca^{+}, H^{+}, K^{+}, and OH^{-} to induce programmed cell death, referred to as the hypersensitive response. This eradicates diseased cells and inhibits the dissemination of infections to uninfected regions.

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