REVIEW ARTICLE

Hypersensitive Responses in Plants

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Abstract

Hypersensitivity is a natural defense for plants in response to a variety of pathogens such as viruses, bacteria, fungi and is characterized by a programmed cell death (PCD) accompanied by an accumulation of toxic compounds within the dead cell. Hypersensitive response (HR) is considered a biochemical reaction rather than a structural defense mechanism but can be seen with the naked eye or with a microscope. There are two types of hypersensitive responses: structural and induced. PCD is seen in both structural as well as in induced hypersensitive response. PCD is extreme resistance shown by the plants in which it kills its cells (suicidal death), upon a perception of the pathogen to deprive it of nutritional supply and stops its growth. Cell death plays a central role in innate immune responses in both plants and animals. Apoptosis and autophagy are physiological processes and two forms of biochemical PCD. Induced hypersensitive response comes out when the plant recognizes specific pathogen-produced signal molecules known as elicitors. Recognition of elicitors by the host plants activates an army of biochemical reactions. These reactions include an oxidative burst of reactive oxygen species (ROS), alterations in plant cell wall also including cell wall immunity (CWI) and damage-associated molecular patterns (DAMPs), induction of phytoalexins and synthesis of PR proteins. These all, are comprised under the first line of defense of plants which come into action after recognition of conserved molecules characteristic of many microbes. These are called elicitors and are known as microbe-associated or pathogen-associated molecular patterns (MAMPs or PAMPs). The second line of defense of plants is the recognition of effectors through plant resistance gene products known as R genes, which result in effector-triggered immunity (ETI). This is supported by the gene for gene hypothesis. Avirulence gene encodes a protein which is specifically recognized by genotypes of the host plant harboring the matching resistance genes.

Keywords: Cell wall immunity, Damage-associated molecular patterns, Effective triggered immunity, Hypersensitive response, Microbe-associated molecular patterns, Pathogen-associated molecular patterns, Pattern recognition receptors, Programmed cell death, Reactive oxygen species.

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Introduction

Pathogens like viruses, bacteria, and fungi attack and thrive on plants in different ways, but the plants have devised their self-defense mechanisms known as hypersensitivity, carried out by PCD accompanied by an accumulation of toxic compounds within the dead cell. It is a feature of resistance, among various types of incompatible interactions between plant and pathogen. A complex signal cascade in the cells of a resistant plant, initially coming from an avirulent pathogen leads to cell death at the site of infection, so that those pathogens, which have penetrated would perish within cells. This is effectively suicide (Bagirova, 2007) and is known as programmed cell death, which is part of the plant's defense response against pathogen attack. It isolates the pathogens from nutrients during the early steps of infection, causing them to starve before they can cause damage. The ability to suppress HR is a major factor for determining whether a pathogen manages to successfully infect a plant. For this reason, HR represents a promising target for improving the overall resistance of crops against pathogens. Signals from the PAMP- and effector-triggered immunity pathways, as well as ROS and phytohormone signals on HR, making it a particularly difficult system to study. HR can be categorized as structural hypersensitive response and Induced hypersensitive response.

Structural hypersensitive response

HR is considered a biochemical reaction rather than a structural defense mechanism but can be described otherwise, since it can be seen with the naked eye or with a microscope and carried out by programmed cell death.

Programmed cell death

Programmed cell death is extreme resistance showed by plants in which it kills its cells upon a perception of the pathogen to deprive the pathogen of nutrition supply and stop its growth (Sarkar *et al*., 2015). Programmed cell death plays a central role in innate immune responses in both plants and animals and is a crucial component of development and defense mechanisms (Reape *et al*, 2008). It is mediated by an intracellular program. The two forms of biochemical programmed cell deaths are:

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Apoptosis—Type I Cell Death

Repeated cell division and programmed cell differentiation are responsible for the development of a multicellular organism. Intracellular organelles undergo fragmentation including the nucleus and the cell collapses producing blebs, and membranous vesicles pop out of the cell surface. Most of these vesicles contain cellular components (Anonymous, 2018). When they are budded off, they are recognized and engulfed by macrophages and consumed. It is an integral part of plant ontogenesis; and is controlled by the following steps(Vanyushin *et al*., 2004).

Cell shrinkage–K^+ and Na^+ efflux: The loss of cell volume is a fundamental and universal characteristic of programmed cell death. Cell volume regulation and the movement of ions with the activation of apoptosis (Fig. 1). It is a dramatic reduction of potassium and sodium concentration, which has been shown to occur in apoptotic cells that exhibit a shrunken morphology. It is a passive process occurring to facilitate the breakdown of the cell into smaller apoptotic bodies

Chromatin condensation: Chromatin undergoes a phase change from a heterogeneous, genetically active network to an inert, highly condensed form. When stained with DNA-binding nuclear dyes, the compacted chromatin will be brighter than the chromatin from non-apoptotic cells, and the condensed nuclei can be easily identified by fluorescence microscopy (qualitative detection) and/or flow cytometry (quantitative detection)

Nuclear fragmentation: Condensed chromatin can be fragmented by a specific nuclease called Caspase-Activated DNase (CAD). Activation of CAD by the caspase cascade leads to specific cleavage of the DNA at the internucleosomal linker site between the nucleosomes, generating fragments of ~ 200 base pairs known as DNA ladders.

Blebbing: A bleb is a bulge or protrusion of the plasma membrane of a cell. It is characterized by the decoupling of the cytoskeleton from the plasma membrane, degrading the internal structure of the cell, allowing the flexibility required to allow the cell to separate into individual bulges or pockets of the intercellular matrix. The blebs eventually separate from the cell taking a portion of the cytoplasm with them and forming what is known as apoptotic bodies. Blebs are also seen in non-apoptotic functions and are also known as zeiosis.

Formation of apoptotic blebs: The blebs are formed into apoptotic bodies. These are vesicles containing parts of a dying cell. Apoptotic bodies can be formed during the execution phase of the apoptotic process when the cell's cytoskeleton breaks up and causes the membrane to bulge outward. These are then engulfed by phagocytotic cells or macrophages (Fig. 2), and their components are recycled. These apoptotic bodies may range in size from 0.8 to 5 µm.

Different Types of Physiological Processes that Lead to Death of Plant Cells Other than Programmed Cell Death

Necrosis: Cells get injured and puncture where cells lyse, extruding various injurious components, which cause severe damage to other neighboring cells, causing widespread destruction; this is like carnage where a larger number of cells are killed at the same time. It is unprogrammed cell death.

Paraptosis: Cells swell, develop large bubbles or vacuoles with liquid inside and die; this method of suicide is called parasitosis. They don't employ Caspases, which are specifics of apoptosis. Most similar methods have been observed in yeast cells (Fig. 3).

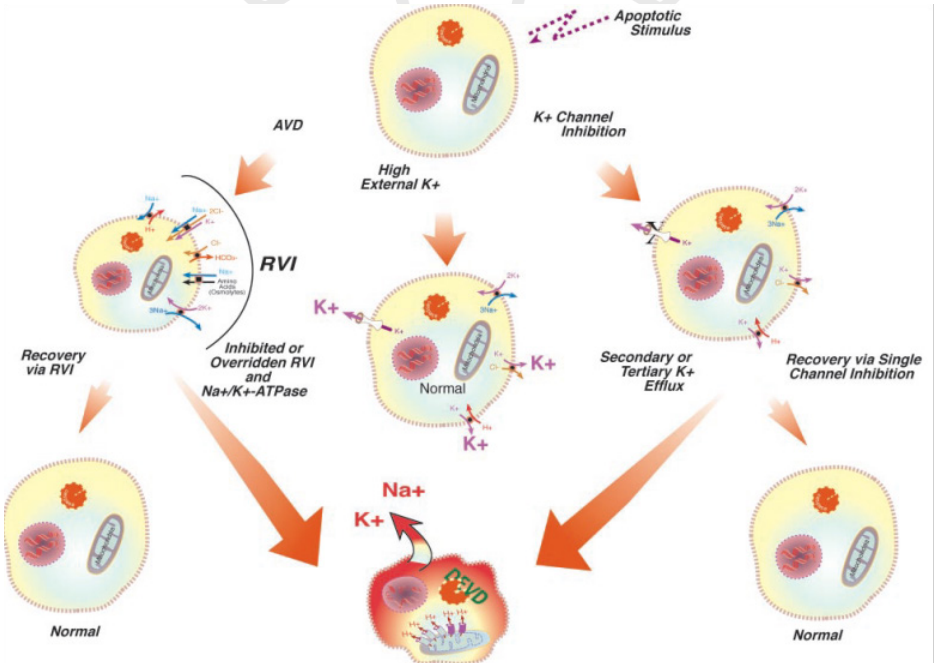


Fig. 1: Outward movement of K^+ and Na^+ ions lead to cell shrimkage during apoptosis (Bortner and Cidlowski, 2002)

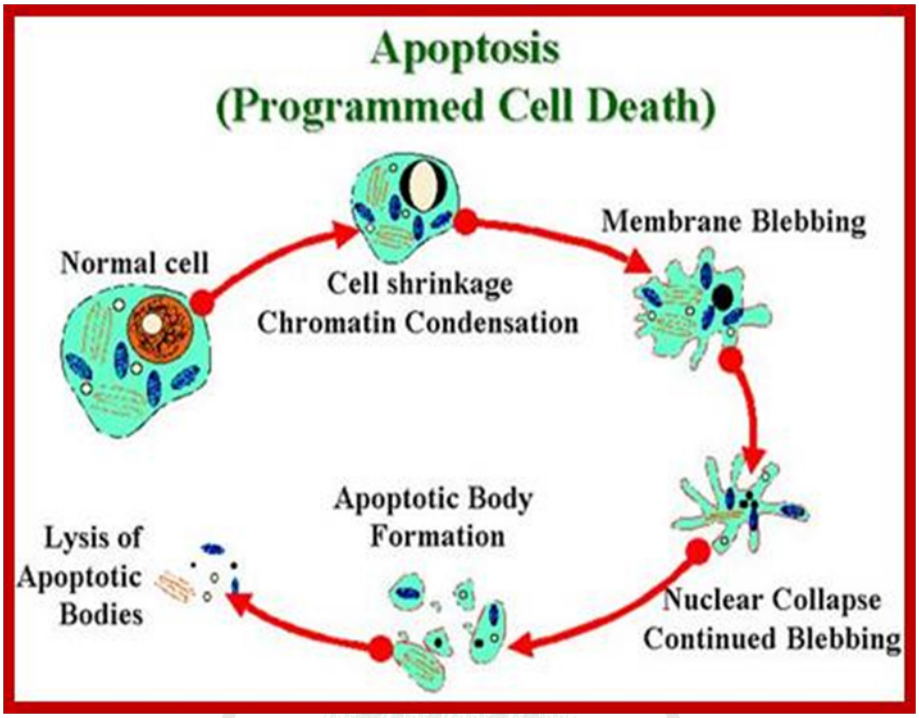


Fig. 2: Different steps of apoptosis that occur during programmed cell death are shown (Wordpress.com)

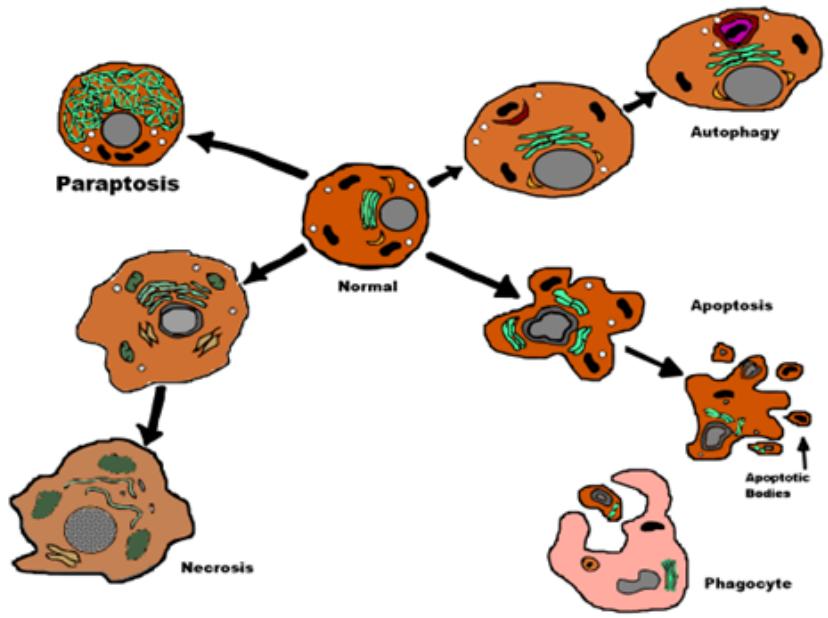


Fig. 3: Death of a normal cell by different processes like paraptosis, autophagy, necrosis and apoptosis (en.wikipedia.org)

Autoschizis: A bizarre type of death; a novel method of programmed death. Cells develop crates inside and cell organelles escape from the cell and are destroyed by some proteases that develop inside the cell. This happens when cancerous cells are treated with vitamin C. Normal cells remain unaffected, but many cells die because of induced apoptosis but substantial no. of cancer cells die by autoschizis. It is characterized by exaggerated membrane damage and the progressive loss of cytoplasm through a series of self-excisions. These self-excisions typically continue until the perikaryon consists of an intact, round nucleus surrounded by a thin rim of cytoplasm that contains damaged organelles. During the process of cell death by autoschizis, nucleoplasm initially becomes more chromatic and then progressively loses

chromaticity as their size decreases. Concomitant with this diminution in cell size, the nuclei become smaller and contain large nucleoli, which become round and compact. Therefore, before it dies, the size of the resultant autoschizic cell is much smaller than the tumor cell from which it originated.

Oncosis: Cells expand by taking in a lot of water in an uncontrolled manner. Soon proteins denature liked cooked yolk proteins, and the cells take excess calcium into cells and death follows. This is due to a differential distribution of proteins on either side of the impermeable inner mitochondrial membrane. In oncosis the loss is dramatic to the loss observed during apoptosis. This observation is reflected in the energy demands of Oncosis and apoptosis upon the cell, Oncosis being ATP independent and does not require a degree of mitochondrial function during the apoptotic process (Anonymous, 2018).

Autophagy—Type II Cell Death

Autophagy is a prosurvival mechanism to restrict PCD associated with the pathogen triggered hypersensitive response during plant innate immunity (Sarkar *et al*., 2015). It is a highly conserved processing mechanism in eukaryotes whereby cytoplasmic components are engulfed in double-membrane vesicles called autophagosomes and are delivered into organelles such as vacuoles for degradation and recycling of the resulting molecules. In other words, the formation of large vacuoles that eat away organelles before the nucleus is destroyed. Isolation of yeast *AUTOPHAGY* (*ATG*) genes has facilitated the identification of corresponding *Arabidopsis ATG* genes based on sequence similarity. Additional roles for autophagy have been suggested in the degradation of oxidized proteins during oxidative stress and the regulation of HR-programmed cell death (PCD) during innate immunity (Kwon and Park, 2008).

Induced hypersensitive responses

- The induced hypersensitive response is an initial recognition event between the host plant and pathogen, which lead to the activation of various host defense responses (Fig. 4).
- HR could regulate direct and indirect interactions between avirulence gene products and resistance gene products from the host plants (Surico, 2013).

Recognition between Plant and Pathogen

The hypersensitive response occurs only in specific host-pathogen combinations in which the host and the pathogen

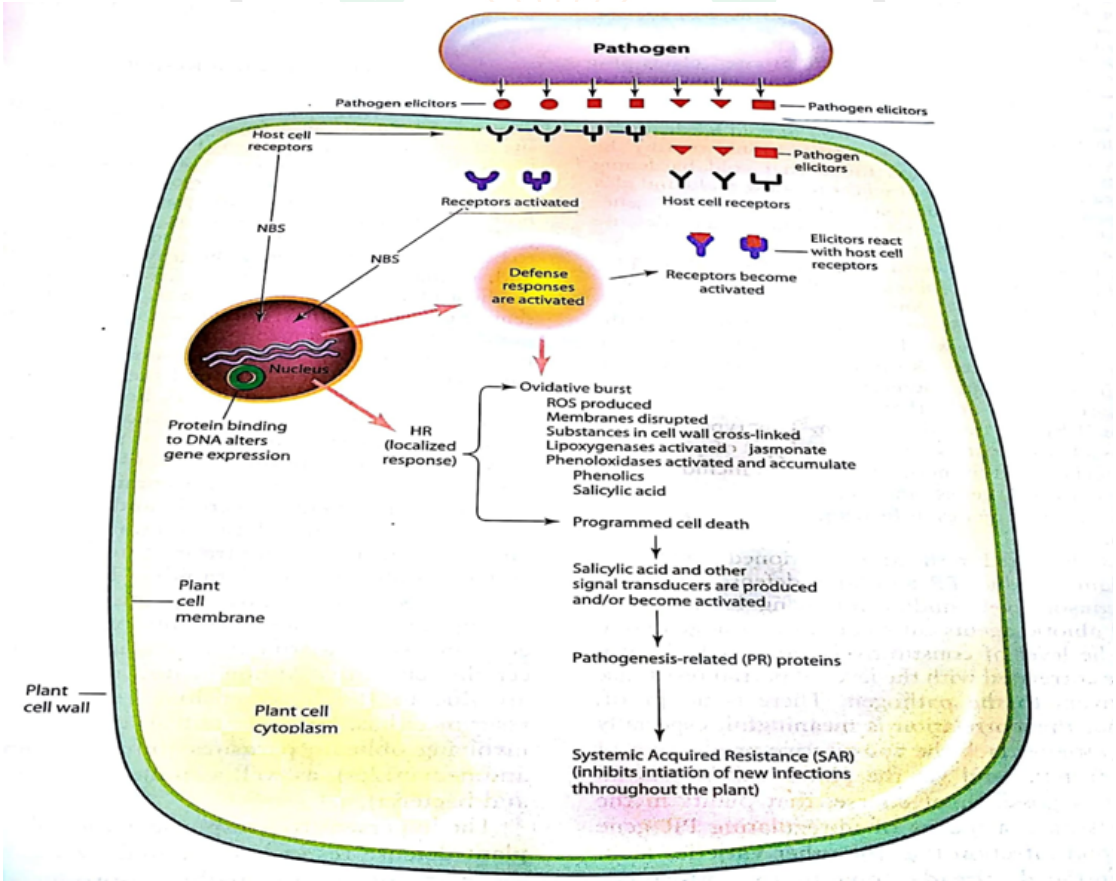


Fig. 4: Hypothetical steps in the hypersensitive response defense of plants following interaction of an elicitor molecule produced by a pathogen avirulance gene with a receptor molecule produced by the matching host R gene.(Agrios, 2005)

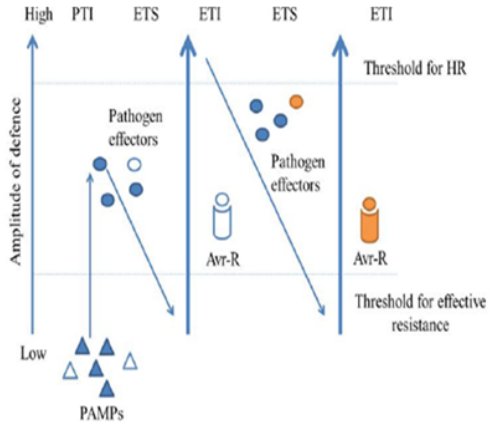


Fig. 5: Zig zag model as described by Jones and Dangl (2006). The zig zag model presents the successive steps of the interaction between the plant and its pathogen. Firstly, pathogen associated molecular patterns (PAMPS) are recognized by their cognate PRS in plants leading to the PAMP–triggered immunity (PTI). Secondly, the pathogen develops effectors that disable PAMP recognition leading to effector triggered suscebtibility (PTS). Thirdly, these effectors become avirulance factors (Avrs) once the plant developed cognate resistance protein (R-protein) leading to effector-triggered immunity. At last, the pathogen develops new effectors or/and loses its Avrs to circumvent ETI leading to effector-triggered susceptibility (ETS). The "armsrace" can continue, drawing a zig-zag between ETI and ETS

are incompatible, i.e., the pathogen fails to infect the host. It happens due to the presence of resistance (R) genes, which recognizes and is triggered into action by the elicitor molecules released by the pathogen.

Avirulence genes: The elicitors produced by the pathogen, triggers the development of resistance in the plant and hence, makes it avirulent, is called an avirulence (*Avr*) gene. The action of elicitors has been described in the zig-zag model.

Zig-zag model: The interplay between the plant defense systems and its suppression by pathogens has been portrayed as a "zig-zag model" by Jones and Dangl (2006) (Fig. 5). This model proposes that the plants' immune responses consist of two branches:

First line of Defense

- The first tier of plant immune system corresponds to pathogen perception via the recognition of conserved pathogen-associated molecular patterns (PAMPs) by plant pathogen or pattern-recognition receptors (PRRs).
- As PAMPs are often also derived from non-pathogens, they are preferentially named microbe-associated molecular patterns (MAMPs) (Ausubel, 2005).
- The plant is also able to detect damage-associated molecular patterns (DAMPs), which are plant degradation products resulting from the action of invading pathogens, or endogenous peptides which are released by the plants following pathogen attacks (Boller and Felix, 2009).

Recognition of DAMPs also triggers immune responses similar to the PTI response (Yamaguchi and Huffaker, 2011).

Microbe-associated Molecular Patterns

Bacteria, fungi, oomycetes, and viruses attack plants in an attempt to gain nutrients from them. During evolution, both plants and pathogens have evolved features to combat each other; the plant is equipped with sophisticated and rapidly mounted defense mechanisms, while their cognate pathogens have developed counterstrategies to overcome those defenses, the so-called "arms race" between plant and pathogens (Bent and MacKey, 2007). The interplay between the plant defense systems and its suppression by pathogens has been portrayed as a "zigzag model" by Jones and Dangl (2006). This model proposes that the plants' immune responses consist of two branches. The first line of defense in plants is the recognition of conserved molecules characteristic of many microbes. These elicitors are also known as microbe- or pathogen-associated molecular patterns (MAMPs or PAMPs). These are correlated with host cells released during cell damage or cell death, these are evolved before adaptive immunity (Vedukola *et al.* 2019). MAMPs are highly conserved structures, required by the microbe for its survival and for that reason conserved both among pathogens, non-pathogenic and saprophytic microorganisms. MAMPs are essential structures for the microbes and are for that reason conserved both among pathogens, non-pathogenic and saprophytic microorganisms. MAMPs are recognized by pattern recognition receptors (PRRs), which are localized on the surface of plant cells; this first phase of defense induction is called MAMP-triggered immunity (MTI) (Ausubel, 2005; Jones and Dangl, 2006). Notably, in contrast to mammals, no intracellular nucleotide-binding-leucine-rich repeat (NB-LRR) protein recognizing a MAMP has yet been identified in plants (Maekawa *et al*., 2011). Bacterial effector proteins injected directly into the host plants' cytoplasm via the pathogens type III secretion system (TTSS), have been demonstrated to suppress MTI (Jamir *et al*., 2004; He *et al*., 2006; Nomura *et al*., 2006), resulting in effector-triggered susceptibility (ETS). The second line of the plants' defense is direct or indirect recognition of a given effector through a set of plant resistance (R) gene products resulting in effector-triggered immunity (ETI) (Jones and Dangl, 2006); also named the gene-for-gene interaction as early as 1942 by Flor. ETI is generally an accelerated and amplified MTI response, and as such, it is a valid defense response (resistance) that in most cases leads to a localized cell death, known as the hypersensitive response (HR). The majority of the R proteins are intracellular receptor proteins of the NB-LRR type. In most cases, the interaction between NB-LRRs and the effectors are indirect (van der Biezen and Jones, 1998).

Damage-associated Molecular Patterns

The plant defense system is not only recognizing microbial elicitors, but some plant-derived molecules also induce plant defense responses. This sensing of infectious-self or modified-self is mediated by DAMPs (Seong and Matzinger, 2004; Boller and Felix, 2009), also referred to as microbe-induced molecular patterns (MIMPs, Mackey and McFall, 2006). Similarly, the mammalian immune system detects "danger"

through a series of DAMPs, now also in this system named damage-associated. The mammalian DAMPs are derived from other tissues activating intracellular cascades that lead to an inflammatory response (Lotze *et al*., 2007).

Bacterial MAMPs

- *Flagellin (Flg)*: Flagella are essential structures for the pathogenic bacteria as they provide motility and often increase the adhesion of the bacteria to its host. Flg, the main building block of bacterial flagella, is well-established as a major activator of innate immunity in animals. Some of the first MAMP recognition studies in plants were carried using Flg.
- *Elongation Factor (Tu)*: In protein biosynthesis, the ribosomes translate the sequence of nucleotides in mRNA into the sequence of AA's in a protein. During the phase of elongation, the ribosome is associated with elongation factors. One such elongation factor is EF-Tu, the most abundant protein in the bacterial cell (Jeppesen *et al*., 2005). The elicitor activity is attributed to a highly conserved part of the N-terminus of EF-Tu, either a 26 or 18 aa peptide named elf26 or elf18. EF-Tu recognition has only been found to elicit innate immunity in members of the family Brassicaceae (Zipfel *et al*., 2006).
- *Peptidoglycan*: PGN, a molecule never found in eukaryotes, is an essential and unique membrane envelope component of all bacteria, making it an excellent target for the eukaryotic innate immune system (reviewed by McDonald *et al*., 2005; Dziarski and Gupta 2006). PGN, which provides rigidity and structure to the cell envelope of both Gram-positive and Gram-negative bacteria, is a complex molecule consisting of numerous glycan chains that are cross-linked by oligo-peptides. These glycan chains are composed of altering N-acetylglucosamine (GlcNAc) and N-acetylmuramic acid (MurNAc), with short peptides attached by an amide linkage to the lactyl group of MurNAc. Several types of PGN, classified by the nature of the third residue of the stem peptide are commonly found. Typically, this is m-diaminopimelic acid (mDAP) PGN in Gram-negative bacteria and some Gram-positive bacilli (genus Bacillus and Clostridium), whereas most other Gram-positive bacteria have L-lysine (LYS) PGN. In a recent study in tomato (Nguyen *et al*. 2010) showed that pre-inoculation into the tomato with Staphylococcus aureus PGN reduced the growth of subsequent bacterial infection in PGN-treated tissue. This priming of defense with a MAMP is similar to that previously described for LPS (Newman *et al*., 2002). PGN from both Gram-positive and Gram-negative bacteria was found to act as elicitors of plant innate immunity in Arabidopsis (Gust *et al*., 2007; Erbs *et al*., 2012).
- *Lipopolysaccharides*: LPS, the major component of the outer membrane of Gram-negative bacteria, have been shown to have multiple roles in plant-microbe interactions; it is thought to contribute to the restrictive Gram-negative membrane permeability, allowing bacterial growth in unfavorable environments. LPS and its derivatives act as MAMPs and induce innate immune responses in plants (Newman *et al*., 1995; Dow *et al*., 2000; Bedini *et al*., 2005; Silipo *et al*., 2005). Earlier studies in plants have shown that LPS can prevent HR induced by bacteria. The mechanisms behind HR prevention are still unknown, but the effects of LPS pre-treatment are considered to be associated with enhanced resistance of the plant tissue to pathogenic bacteria, which is thought to occur through an LPS-dependent potentiation of plant defense responses (Newman *et al*., 2002).

Fungal and Oomycete MAMPs

- *Chitin and β-glucan*: MAMPs from fungi and oomycetes include the fungal chitin and β-glucan. In the fungal cell, walls branched β-glucan is cross-linked to chitin and in oomycetes to cellulose. Chitin and its fragments chitin oligosaccharides have been shown to trigger defense responses in both monocots and dicots.
- Ave1 peptide and ethylene-induced xylanase (EIX): In tomato, a Verticillium resistance locus Ve was identified that mediates resistance against race one strains of Verticillium dahlia and V. albo-atrium, respectively (Kawchuk *et al*., 2001).

Oxidative Burst

MAMP-induced defense responses include the production of reactive oxygen species (ROS), called oxidative burst. It is a rapid, transient, production of a huge amount of gases of reactive oxygen species like hydrogen peroxide (H_2O_2) and superoxides.ROS also drive oxidative cross-linking of polymers in the plant cell wall to strengthen it against degradation, which may restrict pathogen spread. Upon MAMP perception, an extracellular ROS production often occurs at 2–3 min and peaks around 10–14 minutes (Bigeard *et al*., 2014). For e.g:

- In Arabidopsis, the plasma membrane-localized NADPH oxidase named, respiratory burst oxidase homolog D (RBOHD) is responsible for this MAMP induced ROS burst.
- The *P. syringae*–elicited oxidative burst in Arabidopsis leaves, ROS production is first detected around 20 minutes, peaking around 35–40 minutes.

Production of Reactive N-species (NO)

ROS and NO interact rapidly to form a number of reactive nitrogen species, such as $ONOO^-$, NO_2, N_2O_3, and other NO_X species. Hypersensitive cell death is only triggered by a balanced production of NO and ROS and that interaction of NO with hydrogen peroxide is required (Bigeard *et al*., 2014). Besides the direct interactions of these redox molecules, both molecules can act as oxidizing agents on proteins, and in this way, they can modify the activity or function of proteins involved in NO and ROS signaling as well as metabolism and homeostasis (Lindermayr and Durner, 2015).

Alterations in Plant Cell Wall

Cell Wall

One of the barriers that pathogen needs to overcome to successfully colonize plant tissues. The cell wall is a dynamic structure that regulates both constitutive and inducible

defense mechanisms, and a source of signaling molecules that trigger immune responses. Impairment of Cell Wall Immunity (CWI) by pathogen attack or wounding results in the release of plant signaling molecules, called damage-associated molecular patterns (DAMPs). DAMPs modulate plant innate immune responses upon recognition by pattern recognition receptors (PRRs), through molecular mechanisms that are similar to those regulating the activation of immune responses by PAMPs.

Induction of Phytoalexins

Phytoalexins are antimicrobial and often antioxidative substances synthesized intracellularly by plants that accumulate rapidly at areas of pathogen infection. Phytoalexins are produced by healthy cells adjacent to localized damaged and necrotic cells. These act as toxins to the attacking organism. They may:

- Puncture the cell wall
- Delay maturation
- Disrupt metabolism and
- Prevent reproduction of the pathogen

Phytoalexins are formed only when the plant gets in contact with a pathogen and infection starts. These antimicrobial substances sound mighty when talked about, killing the pathogen but are rendered a bit useless when the pathogen proliferates intercellularly, and the phytoalexins are unable to do anything against it because they are not leaked into the spaces between cells. For e.g., *Xanthomonas axonopodis* pv. *citri* show the same behavior causing Citrus Canker (Singh, 2018).

Accumulation of PR-proteins

Pathogenesis-related (PR) proteins are proteins produced in plants in the event of a pathogen attack. Unlike phytoalexins, PR proteins accumulate locally in the infected and surrounding tissues, and also in remote uninfected tissues. This prevents the infection to progress further. PR protein in plants was first discovered and reported in tobacco plants infected by TMV. PR proteins are pre-formed in the plant cells, but their induction is triggered when a pathogen invades the morphology of the plant cell. MAMP detectors lead to this hypersensitive response. This further leads to systemic acquired resistance (SAR). In Arabidopsis the SAR pathway confers resistant to *Pseudomonas syringae* pv. *maculicola* ES 4326 and *Perenospora parasitica*. SAR has been reported in rice with *Pseudomonas syringae* pv. *syringae* as the inducing pathogen (Kamal *et al*., 2015).

Second Line of Defense

The second line of defense of plants is the recognition of effectors through plant resistance gene products known as R genes (Surico, 2013). This has been supported by Gene-for-Gene Hypothesis by Flor n 1942. The second line of plants' defense is direct or indirect recognition of a given effector through a set of plant resistance (R) gene products resulting in effector-triggered immunity (ETI) (Jones and Dangl, 2006).

The Gene-for-Gene Hypothesis

- First developed by Flor in 1942
- Each gene controlling resistance in the host, there is a corresponding gene controlling pathogenicity in the pathogen
- The resistance of host is governed by dominant genes and virulence of pathogen by recessive genes
- When gene in host and pathogen match for all loci, then only the host will show a susceptible reaction. When some gene loci remain unmatched, the host will show a resistant reaction.

Avirulence genes

A phytopathogen avirulence gene is a gene encoding a protein which is specifically 'recognized' by genotypes of the host plant harboring the matching resistance genes, regardless of its function or role in pathogenicity (Rouxel and Balesdent, 2010). Bacteria were the first phytopathogens in which an avirulence gene was cloned (Avr A from *Pseudomonas syringae*; Staskawicz *et al*., 1984).

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