The defense mechanism of plant

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Abstract
According to the state of food security and nutrition, Hunger has increased in many countries in which the economy has slowed down, mostly in middle-income countries. If nothing changes, the immense challenge of achieving the Zero Hunger Target by 2030. The causes of food scarcity might include factors such as unavailability of food due to less production of particular crops/vegetables (due to attack of pests/microbes), it becomes harder to fulfill the basic needs of life especially for poor. Therefore, we need to primarily focus on understanding the interaction between plants and microbes at the molecular level and underlying mechanisms of plant disease and which will help out to solve the global needs of food and resources. Plants have a natural defense mechanism/immune system to react to infections which subdivides into two parts. The first part identifies and reacts to molecules common to different classes of microbes, including non-pathogen. The function of the second part is to react to pathogens virulence factors, either directly or by affecting the host targets. We can also see the intricacies or reciprocation between plants and pathogen attackers. A vast and deep comprehension of plant defense mechanisms will defiantly solve the issue, like food scarcity.

Keywords: Microbial-or pathogen-associated molecular patterns (MAMPs or PAMPs), pattern recognition receptors (PRRs), PAMP-triggered immunity (PTI), hypersensitive response (HR), effector-triggered immunity (ETI)

Introduction
The defense mechanism plant, a system that allows plants to resist attack from a large variety of its enemy. Here, we provide a strategic plan that plants adapt during its immunity. Plant diseases caused by pathogens and microbes are a continuous threat to crop losses and global food security which have devastating effects on both smallholder and factory farming [1-3], the subsequent impact can also be seen in the food supply. The estimated impact of pre-harvest yield loss in crops caused by disease vary, but at least 30% of global agricultural production is affected annually [5]. If we do not take proper action it can badly impact on agriculture, economy, and society of any nation. Perhaps the best-known example would be the Irish potato famine in the mid-1800s, where potato late blight disease (caused by the filamentous plant pathogen Phytophthora infestans) contributed to mass emigration from Ireland [4]. In current agriculture practice, plant diseases are largely controlled by chemicals, but this is unsustainable in the long-term due to environmental concerns and also for future needs of resources. Our focus will be on the cell surface and intracellular immune receptors. We also try to explain how these receptors recognize the signatures of pathogens and pests then activate immune pathways. The interaction between a pathogen and its host is like an evolutionary process, which has been taking place from a long history of warfare with its enemy and they are being constantly changing themselves to fight against natural selection and win over on one another. Though Plants suffer from a disease, their ability to react to infection is crucial for survival. The disease develops only when the pathogen is successful in escaping the multiple layers of host defenses [6-8]. The immune system of plants has similarities with the innate immune system of animals [9-11]. But as plants lack an adaptive immune system, they depend completely and only on innate immunity to perceive microbial pathogens and pests. This review will provide that conceptually, plant immunity can be bifurcated into the cell-surface receptor immunity and intracellular immunity [12]. We also focus on current knowledge of intracellular perception of cell surface receptor and intracellular pathogens, activation of NLRs, and the downstream signaling components.
Cell-surface receptor immunity

It is provided by pattern recognition receptors/ receptor-like kinases/ Cell surface receptor/ receptor-like proteins (PRRs)/ (RLKs) / CRT)/ RLPs which recognize microbe/pathogen/ damage-associated molecular patterns (MAMPs /PAMPs / DAMPs respectively) such as bacterial flagellin or fungal chitin and trigger to [13-16]. Highly adapted pathogens sometime breach pattern- triggered immunity (PTI) by injecting pathogen- encoded proteins called effectors into plant cells [17].

Firstly, damage-associated molecular patterns DAMPs) and MAMPs/PAMPs recognized by plant either by symplastically detected via cytoplasmic NBS-LRRs or by apoplastically detected via RLKs. Secondly, the MAP Kinase pathway which allows the signal transduction of DAMPs/ MAMPs/PAMPs and activates a series of transcription factors like the WRKY gene. Thirdly, this signal causes the production of specific defense responses which include callose deposition, reactive oxygen species (ROS) production, and various specialized metabolism (here camalexin) [18].

Types of cell-surface receptors

These are transmembrane receptors and the best-studied class of plant PRRs is receptor-like kinases (RLKs), which feature an ectodomain of leucine-rich repeats (LRRs) involved in MAMP perception, and an intracellular kinase domain, involved in signal transduction relay via MAPK cascades, resulting in MAMP-triggered immunity (MTI) [19].

RLKs contain a variable extracellular domain that mediates ligand recognition. Plant RLKs that have been identified mostly belong to the family of non-RD kinases in which conserved arginine in the catalytic loop is absent and they mostly belong to the family of non-RD kinases (such as BAK1 and SERKs), which operate as coreceptors for perception to initiate immune signaling [20-23]. In reference to RLPs, they exhibit a similar overall structure to RLKs, and only contain a lacking kinase domain, a short intracellular tail, and require a partner co-receptor to signal [24-25].

The LysM-type RLKs LYK5 (Lysin motif receptor kinase 5) and CERK1 (Chitin elicitor receptor kinase 1) [26-27] bind fungal chitin oligomers and, Arabidopsis LRR-type RLKs, FLS2 (Flagellin-sensitive 2), and EFR (elongation factor Tu (EF-Tu) receptor) [28-29], are amongst the best characterized cell-surface immune receptors.

EFR recognize peptide epitopes from the N-termini of bacterial flagellin (flg22) and bacterial EF-Tu (elf18) respectively [30].

Recognition of peptide

Many subfamilies of cell-surface receptors is protein ligands LRR-RLKs which preferentially bind peptides or proteins as ligands [31-33]. LRR-RLKs from rice and solanaceous plants have also been characterized in Arabidopsis FLS2 and EFR. The rice cell-surface receptor Xa21 binds Rice Xa21-S-Y and a tyrosine-sulfated protein from bacteria [34]. The conserved epitopes derived from bacterial cold shock protein bind to the cell-surface receptors from tomato (CORE) and tobacco (NbCSPR) [35-37]. Likewise, Arabidopsis RLP23 binds the epitope nlp-20, a conserved peptide derived from ethylene-inducing peptide1- like proteins of bacterial and filamentous pathogens [38].

Recognition of carbohydrate

Several different classes of receptors capable of sensing different carbohydrate ligands also have been found. Carbohydrate MAMPs such as bacterial peptidoglycan (PGN), lipopolysaccharide (LPS), and fungal chitin are perceived by LysM-RLKs/LysM-RLPs and LectinRK LORE [39-40].

Regulation of cell-surface receptor

For maintaining cell-surface receptors in an inactive state in the absence of ligand binding, plants employ various strategies that include ubiquitination by E3 ligases and the regulation of phosphorylation state [40-44]. Therefore, the plant cell-surface immune receptors activity is tightly controlled mainly Phosphorylation to prevent any inappropriate signaling [40]. To prevent the potentially harmful effects of autoinduction

Fig 1: The above model can be predicted in the three ways: FIRST recognition, SECOND signal transduction, and THIRD defense response.
plants use phosphatases to negatively regulate cell-surface receptors. For example, dephosphorylates BAK1/EFR to control defense signaling. Arabidopsis PP2A (Protein Phosphatase 2A), a serine/threonine phosphatase [4-47]. Similarly, PP2C38 regulates ligand-induced phosphorylation of BIK1, moderating signaling by this key transducer of cell-surface immunity [40]. The use of pseudo kinases, such as BIR1 and BIR2 to negatively regulate cell surface immunity could also be a Second strategy. They are catalytically inactive but interact with BAK1 in its resting state, preventing the association of LRR-RLKs [48-50]. This inhibitory interaction is relieved by ligand binding and thus leading to the formation of activated immune complexes.

Immunity can also be regulated by controlled degradation through ubiquitination. Two closely related E3-ubiquitin ligases, PUB25 and PUB26, together with both a calcium-dependent protein kinase CPK28 and a heterotrimeric G protein, form a regulatory module and maintain BIK1 homeostasis [49]. Similarly, PUB12 and PUB13 polyubiquitinate and mediate degradation of ligand-bound FLS2 [51-53].

**Intracellular immunity**

**Effectors: Master manipulators of plant cells**

Master manipulators of plant cells that promote infection are the effectors. For the best understanding of the interplay between the pathogens/pests and the plant immune system, let’s first discuss the effectors and their role in promoting host infection. The term ‘effectors’ is used to define protein molecules secreted by microbial pathogens and microbes to promote signal transduction from extracellular to intracellular of the host [53]. These effectors can be delivered to the extracellular space or deployed to the inside of host cells. Therefore, in the broadest definition, the molecules including microbes, plants, and animals to modulate the activity of another organism (plant) are effectors.

**Interaction between the extracellular and intracellular immune response to deal with pathogens.**

These interactions can be understood by four phases proposed by Jones and Dangl known as the ‘Zigzag model’ [54]. It mainly explains that two branches PTI (Pattern-Triggered Immunity) and ETI (Effector-Triggered Immunity) [54]. PTI is mainly dependent on the upon conserved plasma membrane-associated extracellular Pattern-Recognition Receptors (PRRs) [55-56], such as Receptor-Like Kinases (RLKs) and Receptor-Like Proteins. RLPs and RLKs are similar but the only difference is that RLPs lack a cytoplasmic kinase domain. PTI is mostly achieved without the death of the host plant cells. RLPs detecting highly conserved microbial features (a.k.a. Pathogen-Associated Molecular Patterns, PAMPs) such as bacterial cell wall-derived peptidoglycans or flagella fragments in the host apoplast. When secreted proteins, knowns as effectors, move inside the host, some of the effectors act on PTI to neutralize it and other effectors tries to manipulate the host cell metabolism and use the nutrients host [57-58]. same host plant might have highly variable intracellular receptors known as disease resistance (R) proteins that can recognize effectors and try to encounter or neutralize the effector, after the successful encounter of effectors activation of ETI. ETI is often led to programmed cell death of the affected cell, which also called as a hypersensitive response (HR) [59-60].

![Fig 2](http://www.thepharmajournal.com)

**Fig 2:** The above ‘Zigzag model’ can be explain in Four phase.

**Phase 1:** - PAMPs DAMPs/ MAMPs) are recognized by PRRs, thus contributing in activating PAMP-triggered immunity (PTI) and then this PAMP-triggered immunity (PTI) will try to block or retarded further colonization of pathogen. A tag of war occurs between host immune system and pathogens, and in some instances, pathogen might win the first phase. Then.

**Phase 2:** Effectors are spread out by successful pathogens which lead to pathogen virulence and these effectors can interfere with PTI and therefore, resulting in effector-triggered susceptibility (ETS).

**Phase 3:** - One of the NB-LRR proteins specifically recognizes the effector, resulting in effector-triggered immunity (ETI) and this ETI is an accelerated and amplified PTI response which results in disease resistance and, usually, at the infection site a hypersensitive cell death response (HR) occurs. Also, this recognition of effector is either indirect, or directly through NB-LRR.

**Phase 4:** - through the gain of new effectors through horizontal gene flow, pathogens will be driven to avoid ETI...
by one of the two ways either by acquiring additional effectors that suppress ETI or by shedding or diversifying the recognized effector gene. Natural selection might result in favor of new plant NB-LRR alleles that result in new $R$ specificities so that ETI can be triggered again. So this tug of war between pathogen and plant immune system can be summarized as follows, Both PTI and ETI join together for a rapid explosion/burst of extracellular reactive oxygen species (ROS), which cascade the activation of mitogen- activated protein kinases (MAPKs) and calcium-dependent protein kinases (CPKs), which increase in cytosolic calcium, change in ion fluxes, increase of phytohormones, thus overall reprogramming of the host plant for death signal [61,62]. During ETI, the amplitude of these responses is much higher than in PTI and this often leads to induction of a type of programmed cell death (also called as the hypersensitive response (HR)) at the site of infection that results in the overall restriction of the pathogen by localized cell death. On the other hand, pathogens are likely to contribute to the suppression of one or more components of PTI or ETI. These vice versa process mainly depend upon, in which way natural selection favor the process [63].

**HR (hypersensitive response)**
A form of programmed cell death- the hypersensitive response (HR), is often lead to the inhibition of pathogen growth due to NB-LRRs in plants. HR is a specific and unique type of cell death. The chloroplast has an important role in HR firstly, many effectors have chloroplast localization signals [65], sometime these effectors, have shown to suppress immunity [66-67]. Secondly, HR make up a very important source of security signaling molecules for example as reactive nitrogen oxide intermediates (NOI), reactive oxygen species (ROS), defense hormones jasmonic acid (JA) and salicylic acid (SA).Thirdly, light is also required for HR development in many cases. During the final stages some of its typical hallmarks are vacuolization and chloroplast disruption [64]. Thus, ROS produced by plant organelles such as chloroplast, mitochondria and peroxisomes contribute to the HR response. Thus, in plants the molecular events that lead to HR are given below. ETI are partly overlapping with those associated with MTI, including accumulation of SA, ROS and NOI, activation of MAPK cascades, changes in intracellular calcium levels, transcriptional reprogramming and synthesis of antimicrobial compounds [64].

**Co-evolution of $R$ genes (plant) and the effector (pathogen)**

![Fig 3: Co-evolution of $R$ genes (plant) and the effector (pathogen)](http://www.thepharmajournal.com)

A pathogen has an effector gene ($E1$) that is sensed by a rare $R1$ allele (top). This led to selection of an elevated frequency of $R1$ in the population. Pathogens in which the effector is mutated are then selected, because they can grow on $R1$-containing plants (right), $R1$ effectiveness erodes, and, because at least some $R$ genes have associated fitness costs [68], plants carrying $R1$ can have reduced fitness (bottom), resulting in reduced $R1$ frequencies. The pathogen population will still contain individuals with $E1$. In the absence of $R1$, $E1$ will confer increased fitness, and its frequency in the population will increase (left). This will lead to resumption of selection for $R1$ (top). In populations of plants and pathogens, this cycle is continuously turning, with scores of effectors and many alleles at various $R$ loci in play [69].

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