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Plant–Fungus Interaction: A Stimulus–Response Theory

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Abstract
Plants are exposed to various severe constraints comprising damages caused by phytopathogens, which eventually lowers productivity. During plant–fungus interaction, the fungus absorbs host nutrients by secreting cell wall-degrading enzymes, toxins, suppressing plant defense, triggering programmed cell death, and shutting down plant defensive genes. Plants have various defense mechanisms to counteract the harmful effects of fungi including constitutive and induced defense systems that either directly or indirectly attack the fungus. However, throughout co-evolution, both pathogens and plants have acquired their combat systems at the molecular level in a see-saw fashion and this tug-of-war between them has evolved endlessly. Hence, we are still a long way from fully comprehending all the variables determining the winner of this arms race. Therefore, the present review will help to broaden our knowledge about the events occurring during plant–fungus interaction, unfolding a process of unexpected complexity.

Keywords Pathogen-associated molecular patterns · Pathogenicity factors · Defense mechanism · Redox homeostasis

Introduction

Plants are one of the major sources of food and provide shelter to a wide range of parasites that include fungi, bacteria, viruses, insects, nematodes, and sometimes other plants also (Barwant 2021). The plant–pathogen interaction is a multifaceted process that is mediated by pathogen-derived compounds that are crucial for their pathogenicity and plant-derived molecules that are required to recognize these pathogens and trigger the defense response (Balotf et al. 2022). When Harold Henry Flor, in 1940s released his groundbreaking study on the genetics of the interaction between flax and fungus (*Melampsora lini*) that causes rust disease, a thorough understanding of the genetic connections that regulate disease resistance in plants emerged (Flor 1942). Flor researched the virulence of the pathogen and the inheritance of resistance in the host, producing pioneering work that was underappreciated at the time. This work resulted in the formulation of the "gene-for-gene" hypothesis. To establish disease, pathogens need pathogenicity factors that affect the virulence of a pathogen which includes the contagiousness or invasiveness and resistance of the pathogen to host defenses (Kumara et al. 2022). During their entry into the plant tissue, these factors aid the pathogen to encounter various hurdles that include structural barriers and constitutively produced anti-fungal compounds (John et al. 2020). Plants have a two-layered actively induced immune system in response to fungal stimuli. The first layer of immune response which is activated by pathogen-associated molecular patterns (PAMPs) is termed as PAMPs-triggered immunity (PTI). PAMPs are generally conserved pathogen compounds like fungal chitin, bacterial flagellin, lipopolysaccharides or elongation factor TU) which are detected by plant surface receptors known as pattern recognition receptors (PRR) (Boutrot and Zipfel 2017). Effector-triggered immunity (ETI); the second layer of defense is regulated by intracellular resistance (R) proteins that detect virulence factors (also known as effectors) released by pathogens into the host cells (Yoo et al. 2020). ETI can cause a hypersensitive reaction (HR) to destroy both the invaded pathogen and the infected plant cells. ETI is quantitatively more potent and rapid than PTI. Together, PTI and ETI form a significant innate immune response that enables plants to detect and defend themselves from pathogen attacks (Chang et al. 2022).

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The generation of phytoalexins, reactive oxygen species (ROS), pathogenesis-related (PR) proteins, activation of signaling pathways, reinforcement of cell wall, programmed cell death (PCD), and systemic acquired resistance (SAR) are typical elements of defense mechanism (Waszczak et al. 2018). However, the foremost response of plants is the rapid ROS production that causes oxidative bursts which is necessary for further defense reactions. ROS acts a double-edged sword, serving as signaling molecules at low concentrations while causing PCD at higher concentrations (Mittler et al. 2022). Nevertheless, oxidative stress tolerance is not a single-step mechanism, but it includes an integrated response that is accompanied by alterations in both enzymatic and non-enzymatic antioxidants. These antioxidants play a crucial part in the defense mechanisms either by either directly or indirectly damaging the pathogen through oxidation or by activating protective signaling cascades (Sharma et al. 2022). Cellular antioxidants have an impact on plant growth and development by regulating processes such as mitosis, cell elongation, senescence, and death, as well as playing a significant role as cofactors for various enzymes (Barreca 2021). A model for redox homeostasis that considers the ROS-antioxidant interaction as a metabolic interface that controls the optimal induction of acclimation processes or the execution of cell death programs which is being supported by increasing evidence. There is growing interest in the physiological function of these compounds in the disease resistance of plants. Furthermore, plant pathologists have long been interested in and motivated by the quest to understand the molecular foundations of why a specific pathogen causes disease in one host plant but not another. So, in the present review, we discuss the new central dogma of plant pathology: a plant disease resistance model that describes the evolutionary processes occurring during plant–pathogen interaction. Different pathogenicity factors released by pathogens and to counteract their deleterious effects, various defense mechanisms opted by the plants will be illustrated. We end the discussion by reviewing the role of various enzymatic and non-enzymatic components in redox homeostasis. Hence, the present review summarizes our current knowledge of the diverse measures taken by plants and fungi during their compatible and incompatible interactions.

Molecular Responses During Host–Pathogen Interaction

During any host–pathogen interaction, the response to disease reaction is determined by the genetic constitution of both the host as well as the pathogen (Ragunathan et al. 2021). The co-existence of the host and its pathogen directs that both are evolving together which can be manifested by the balance between the changes in pathogen virulence and host resistance and vice versa. The plant pathologist, Flor had given the "gene-for-gene hypothesis" or "Flor hypothesis" that explains the stepwise evolution of pathogen virulence and host resistance (Flor 1946) (Table 1). The hypothesis has three considerations: (i) mostly, in the host, resistance genes are dominant (R); (ii) in the pathogen, virulence genes are recessive (*avr*) and (iii) for every resistance gene in the host there is a complementary gene that governs pathogenicity in the pathogen.

By applying the "gene for gene" hypothesis, plant breeders can incorporate a new resistance gene into a variety of their choice, but the desirable variety can become susceptible to some new strain of the same pathogen. Datta et al. (1999) found that the disease score was highly significant during wheat cultivar–karnal bunt interaction that indicated the probable existence of a gene-for-gene relationship in the wheat–*Neovossia indica* system. Brading et al. (2002) also provided evidence for a gene-for-gene interaction between *Mycosphaerella graminicola* and several wheat cultivars. AVR-Pita, a rice blast avirulence gene present in the plant–pathogenic fungus *Magnaporthe grisea* corresponds in gene-for-gene fashion to the disease resistance (R) gene Pi-ta that falls into the NB-LRR class in rice (Orbach et al. 2000). The tomato resistance genes Cf-9 and Cf-4 (Thomas et al. 1997) and their avirulence counterparts Avr-9 and Avr-4 from *Cladosporium fulvum* (Joosten et al. 1994) were the other similar gene pair cloned from plant–fungus interaction that contributed to the genetic engineering techniques needed for plant disease resistance.

However, there are some limitations to this classical model of the "Flor hypothesis". After extensive research in the last few years in this field, the latest model named "New Central Dogma of Plant Pathology" has emerged that describes the evolutionary processes occurring between host and pathogen (Fig. 1). The four-part model includes: (i) Plants have plasma membrane-located PRRs that detect PAMPs and triggers the mitogen-activated protein kinases (MAPK) signaling cascade that reaches the nucleus and activates defense-related genes to produce defense-related proteins. The genes encoding PRRs are stable and heritable

Table 1 Disease reaction responses according to the "gene-for-gene" hypothesis during host–pathogen interaction (Flor 1946)

Pathogen	Host plant	
	RR or R (Dominant gene)	rr (Recessive gene)
Avr (dominant gene)	*Avr*×R (no disease/resistant) Incompatible reaction	*Avr*×*r* (disease/susceptible) Compatible reaction
avr (recessive gene)	*avr*×R (disease/susceptible) Compatible reaction	*avr*×*r* (disease/susceptible) Compatible reaction

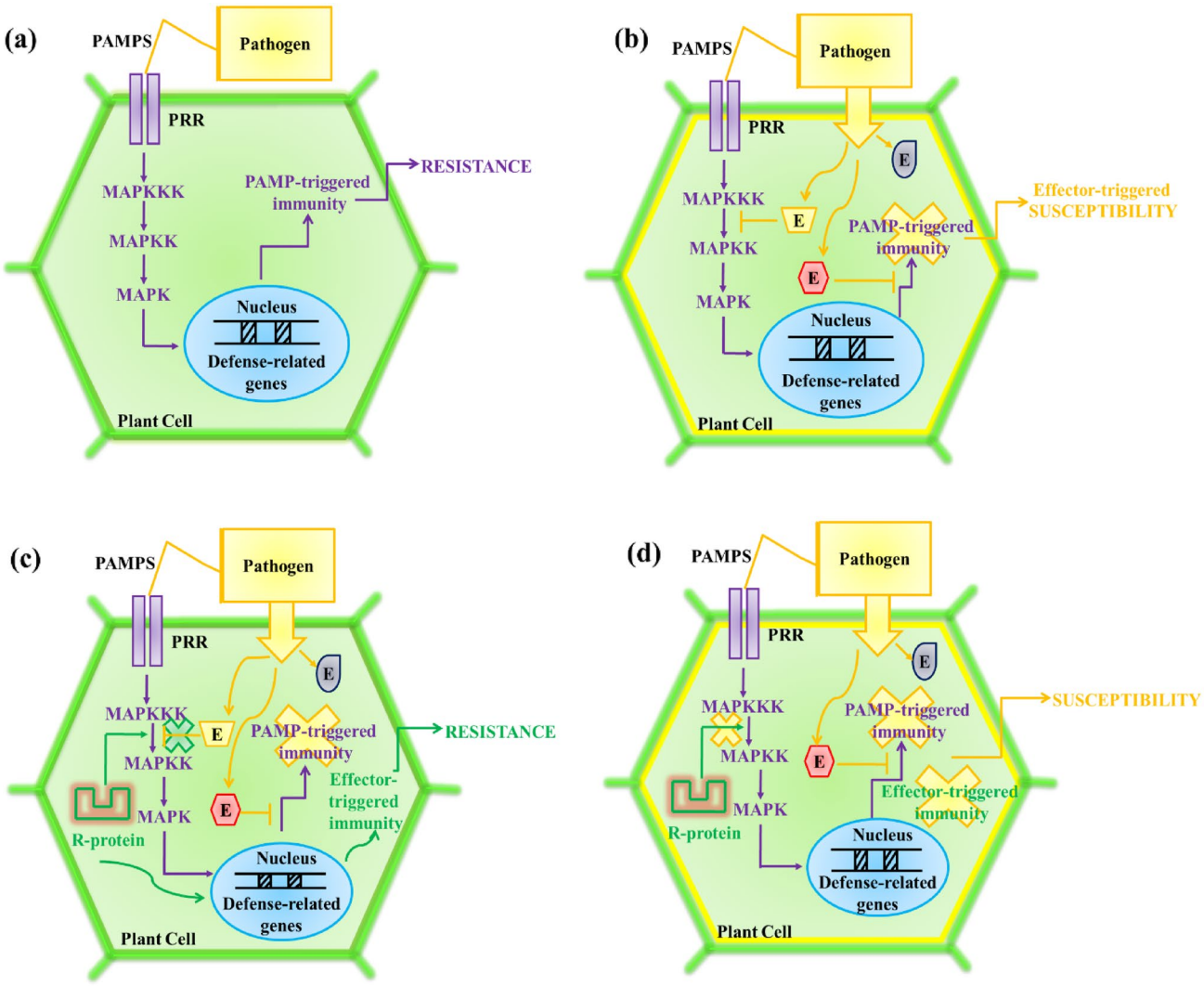

Fig. 1 A four-part model for plant disease reaction

which enables plants to early detect the pathogen infection. This basal immunity of plants which helps to mitigate the destructive effects of non-pathogen is termed as PTI (Bohm et al. 2014) (Fig. 1a). (ii) In return, certain pathogens can evolve new virulence factors that can actively suppress the immune signaling of their respective hosts and become adapted to them. Unlike PAMPs, these effector molecules do not have the housekeeping functions required for pathogen growth and development. Pathogens that interfere with plant defense mechanisms through effectors, induce plant susceptibility towards the pathogen and this reaction is known as effector-triggered susceptibility (ETS) (Lapin and Ackerveken 2013) (Fig. 1b). (iii) In due course of time, adapted pathogens are repelled by the host because host species have evolved specific R genes that encode effector recognition proteins (R proteins) that trigger an immune reaction by detecting effectors and hindering the pathogen growth. This phenomenon is known as ETI (Wu et al. 2014) (Fig. 1c). (iv) Further, by modifying/eliminating the effectors, pathogens avoid R gene-mediated defenses and make plants susceptible (Howden and Huitema 2012) (Fig. 1d).

Pathogenicity Factors of Fungi

Pathogenicity factors are the components of an organism that determine its capacity to cause disease but are not required for its viability. There are numerous pathogenicity factors such as cell wall-degrading enzymes, toxins, hormones, and polysaccharides that help the fungus to invade the plant cell via lesions, stomata or through direct penetration (Chang et al. 2022). The pathogenicity factors of fungi can be classified into five types according to their function (Fig. 2).

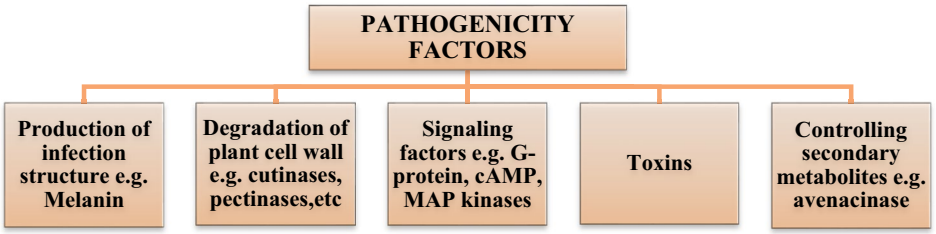

Fig. 2 Various pathogenicity factors of fungi produced during plant–fungus interaction

Production of Infection Structure

The plant gets infected by parasitic fungi for a rich supply of nutrients (Grossart et al. 2019). Penetration is a crucial step in successful parasitism. The choice of whether a pathogen will successfully colonize the plant is frequently decided during penetration. Fungi have developed a remarkable variety of invasion techniques to get past the numerous obstacles found in leaves, stems, or roots. To accomplish this, the fungus produces infection structures that allow it to pierce various plant cell wall types. The morphogenetic processes that result in the construction of the infection structure frequently depend on specific signals provided by the plant surface and are prerequisites for a particular mode of penetration (Foster et al. 2017). Phytopathogenic fungus infection structures are modified hyphae specialized for plant tissue invasion. Adhesion to the cuticle and directed growth of the germ tube on the plant surface constitute the initial events. Appressoria that have melanized walls and glycerol for generating high turgor pressure to support the penetration process are frequently formed at the penetration site. *Magnaporthe grisea* and *Colletotrichum* species have appressorial walls that contain melanin, which inhibits glycerol from leaking out (Mendgen et al. 1996). Melanin-deficient mutants cannot develop turgor pressure and are non-pathogenic. To penetrate the cuticle and the plant cell wall, the penetration hypha accumulates cytoskeleton components in the tip and secretes a wide range of cell wall-degrading enzymes in a highly regulated manner.

Degradation of Plant Cell Wall

Plant cell walls are natural heterogeneous structures made up of polysaccharides, aromatic polymers, and proteins. Different plant lineages have very different cell wall compositions and structures, however, they share similar structural construction elements, such as cellulose microfibrils embedded in a matrix of pectin, lignin, hemicellulose, and structural proteins (Zhang et al. 2021). For pathogenesis, phytopathogenic fungi develop a variety of enzymes capable of disintegrating cell wall polymers referred to as cell wall-degrading enzymes (CWDEs) viz. cellulases, glucosidase, xylanases, pectin lyase, polygalacturonase, and pectin methylesterase (Pontes et al. 2020). When the rice was inoculated with the blast fungal pathogen *Magnaporthe oryzae*, a high level of gene expression, primarily for cellulases, hemicellulases, and pectate lyase was observed (Eseola et al. 2021).

Signaling Factors

Fungal pathogenicity also involves numerous fundamental cell signaling transduction pathways, such as MAPK signaling cascade, G-protein signaling pathway and cAMP pathways, which are highly conserved and directly affect organism fitness (Li et al. 2022). The fungus demonstrates a loss (or reduction) in various functions including mating, growth rate, and formation of conidia and toxins when mutation alters their signaling genes. During host colonization, many virulence factors, including effectors, CWDEs and mycotoxins are frequently transcriptionally co-regulated. The result of pathogen-host interactions is determined by such coordinated regulation. *Fusarium graminearum* and *F. verticillioides*, two plant–pathogenic fungi, were examined using the cAMP-PKA pathway by Guo et al. (2016), who found that fungal diversification and niche adaptability are influenced by the evolutionary process of conserved signaling pathways.

Toxins

Many fungi that are plant pathogens release toxins that can harm plant tissues. Toxins are frequently categorized as host-specific (host selective) or non-host selective. Host-selective toxins are poisonous only to the plants that serve as hosts to specific fungi (Puntscher et al. 2019). Contrarily, nonspecific toxins can harm a wide variety of plants whether they are hosts of the pathogen that is producing them. Brown spot disease in tobacco is caused by the host-specific toxin AT-toxin, which is produced by *Alternaria longipes*. Corn leaf spot and ear rot disease are caused by the other host-specific *Helminthosporium carbonum* (HC) toxin, which inhibits histone deacetylation and stops the plant from producing antifungal chemicals (Brosch et al. 1995).

Controlling Secondary Metabolites

Fungal pathogens also require genes that will enable them to circumvent the antifungal effects of numerous secondary metabolites produced by plants. In addition to the genes required for generating infection structures and for destroying structural impediments. These genes modify the physiology of secondary metabolites, assisting pathogens to avoid or destroy them. For instance, avenacin A-1, a triterpenoid saponin is a natural product found in the epidermis of oat roots. Oats are susceptible to infection caused by the fungus *Gaeumannomyces graminis* var. *avena* because it carries a gene that codes for the enzyme avenacinase, which breaks down the avenacin A-1 saponin (Osbourn et al. 1994).

Defense Responses in Plants

Plant disease resistance is crucial for sustainable food production that significantly leads to the reduction in the use of agricultural land, water, fuel, and other inputs (Abebe 2021). To cope with biotic and abiotic stresses, plants developed various strategies including passive and active defense mechanisms as presented in Fig. 3.

Passive Defense

Passive defense is independent of the pathogen that is present in plants before encountering the pathogen and hence, also known as the constitutive or pre-existing or first line of defense (Boots and Best 2018). It is the combination of weapons from two arsenals i.e., morphological characteristics and biochemical reactions.

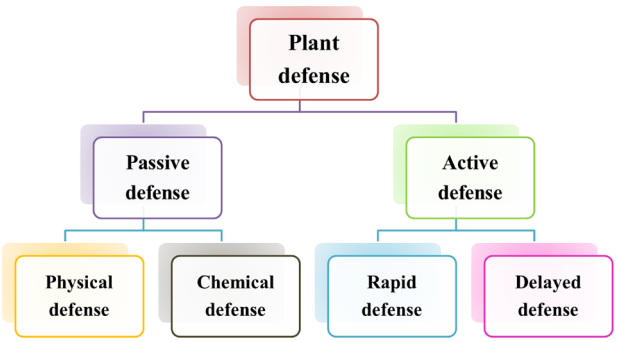

Fig. 3 Different plant defense responses against fungi during their interactions

Physical Defense

Physical defense mechanisms were displayed by structural elements that serve as physical obstacles to prevent pathogens from entering and spreading throughout the plant. It comprises the composition of the epidermal cell wall, the presence of cells with thick walls, the amount and quality of wax and cuticle covering the epidermal cells and the size, location, and forms of stomata and lenticels (John et al. 2020). The waxy nature of cuticles and the orientation of leaves in a vertical manner prevent the formation of moisture films on leaf surfaces that assist in the inhibition of pathogen mobility. Moreover, plants having incompatible stomatal apertures for pathogen infection structures to enter or having stomata that close at the time of day when pathogen spores normally germinate may be more resistant to pathogen attack (Melotto et al. 2017).

Chemical Defense

It includes the presence or absence of a specific chemical or group of chemicals in the host plant which hinders the rate by which pathogen multiples. It includes nutrient deprivation, the generation of phytoanticipins and defensins (Khare et al. 2017). The presence of the phenolic compound, which prevents pathogen nutrition and slows the pathogens' growth and development, is correlated with the resistance of immature pears and apples to scab produced by *Ventura perini* and *V. inequalis*, respectively (Castroverde et al. 2010).

Active Defense

The host defense system seeks to build barriers to stop additional colonization of tissues when the passive barriers are crossed (Sharma et al. 2022). Only after pathogen detection, active or induced defense mechanisms that can be specific and non-specific are triggered. It involves the biochemical defenses at the cellular and tissue levels. Genomic factors control the plant's ability to mount an active defense response (Waszczak et al. 2018). A complex signaling network including pathways regulated by salicylic acid (SA), jasmonic acid (JA), and ethylene (ET), controls this system of defense mechanism.

Rapid Active Defenses

The plant must identify the pathogen as early as possible to activate the biochemical and structural defenses that are available to defend against it (Barreca 2021). Once, the pathogens make physical contact with the plant, subsequently, the plant starts to receive signaling molecules that signify the presence of the pathogen. Many pathogens have developed diverse strategies for circumventing the physical defense barriers. At this stage, HR which can be regarded as a "fail-safe" mechanism for the preinvasion defenses is noticed (Mittler et al. 2022). It is a type of PCD at the site of infection which is accomplished by rapid synthesis of ROS. The strategy behind PCD appears like physical defense where invading pathogens are encased in dead cell tissue, consequently depriving them of the nutrients that they would normally acquire from the apoplast or from piercing the plant cells (Balotf et al. 2022).

Even though the course of HR differs among various plant–pathogen systems, some patterns at cellular level may be seen during the first few hours of infection (Naveed et al. 2020). Even before the pathogen reaches the cell membrane after penetrating the cell wall, the plant's nucleus travels towards the penetration site which is accompanied by a general increase of directed flow, also known as streaming or cyclosis, within the plant cell. At this phase, the cytoskeleton undergoes structural changes and a decrease in the number of microtubules is observed followed by enhanced transcriptional and translational activity due to an increase in nuclear pores and polyribosomes (Balint-Kurti 2019). After this active period, the nucleus decays, shrinks and deforms consequently. The vacuoles start to burst while DNA cleavage is observed and tiny vesicles or granules become visible. The generation of ROS occurs after streaming slows down and finally stops. Ultimately, the cell becomes brown, as a result of the polymerization of phenolic chemicals and finally, the entire protoplast collapses (Dalio et al. 2021).

HR is linked to the activation of defense-related genes that are crucial for controlling the development of pathogens, either directly by producing phytoalexins and antimicrobial enzymes or indirectly by strengthening the plant cell walls. Phytoalexins are antimicrobial and often antioxidative substances synthesized intracellularly by plants that accumulate rapidly in areas of pathogen infection (Sivakumar and Deepa 2023). They are produced by the healthy cells adjacent to the localized necrotic and injured 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. They are formed only when the plant gets in contact with a pathogen and infection starts (Thakur et al. 2019). The hypersensitive reaction occurs only in incompatible host-parasite combinations. In cases where the plant is not the host for the pathogen, no mechanisms have been developed by the pathogen to restrict plant defense mechanisms. Hence, by being effective at protecting against a wide spectrum of diseases, HR is occasionally classified as a non-host defense mechanism. In the best-case scenario, HR results in the pathogen starving and is particularly effective against diseases that require live things to feed on, such as biotrophic pathogens (Camagna and Takemoto 2018). Conversely, it has been demonstrated that HR can help necrotrophic pathogens, which consume dead plant tissue. We may presume that certain regulatory entities exist that decide what course of action to follow for each pathogen because activating HR is not a sufficient response for all pathogens.

Delayed Active Defense

Early defense responses slowed pathogen development; later defense responses restrained their spread and contained the harm they caused to the host tissues. The capacity of a plant to recover from tissue injury can help it to resist additional infections caused by opportunistic pathogens. A secondary resistance response brought on by HR to avirulent microorganisms is known as SAR (Radojicic et al. 2018). Within 4–6 h of inoculation, the SAR signal may begin to develop. It is classically described as a "whole-plant" resistance response that provides long-lasting, broad-spectrum pathogen resistance to uninfected systemic leaves following an initial localized infection (Wani et al. 2018). It is distinguished by the activation of a wide range of host defense systems, both locally at the site of infection and systemically, in tissues that have not yet been exposed to the pathogen. SAR can offer resistance against a wide range of species, including viruses, bacteria, and fungi. The generation of a signal that is transported to other areas of the plant, where it stimulates resistance, is necessary for the induced defense reactions linked to SAR, which involve both biochemical and cytological alterations (Betsuyaku et al. 2018).

There are various defense reactions associated with SAR, such as the buildup of histological barriers and the production of PR proteins. Certain structures develop inside the host to prevent further spread of the pathogen. These histological defense mechanisms include the formation of cork layers, abscission layer, tyloses and deposition of gums (John et al. 2020). The development of cork layers prevents further invasion of pathogens and stops the spreading of any hazardous compounds it may release. Cork layers also prevent the movement of nutrients and water from the healthy area to the infected area of the plant, following in the starvation of pathogens. In contrast, an abscission layer

is made up of a space created between two circular layers of leaf cells that surround the infection site (Abebe 2021). The core region of the leaf is cut off from the rest of the leaf and the middle lamella between these two layers of cells dissolves on infection. This area gradually shrivels, dries out, and peels off, carrying the infection with it. Tyloses are the result of protoplast expansion in nearby live parenchymatous cells, which protrude into xylem vessels via pits (Sauban et al. 2016). Tyloses have cellulosic walls and fully clog the vessels that form abundantly and swiftly in some plant kinds, ahead of the pathogen. The gums are another impenetrable barrier that completely encloses the pathogen by depositing in the intercellular gaps or within the cells around the site of infection that subsequently isolated the pathogen that eventually dies due to starvation. Stone fruit trees have the most gum secretion, however, it occurs in all plants (Mushtaq et al. 2022).

Together with strengthened structural defenses, synthesis of antifungal phytoalexins and PCD, several PR proteins are generated during pathogen attack (Waszczak et al. 2018). In healthy plants, these proteins are produced at modest levels, but when a pathogen attacks, specific isozymes are either locally or systemically activated. According to serology and homology, the induced proteins have been divided into 14 classes, however not all of them are induced in all interactions or all plant species (Table 2). The PR proteins of different groups differ in molecular weight, iso-electric point, and immunological cross-reactivity. For instance, it is believed that the chitinases (PR-3, PR-4, PR-8, and PR-11), which are categorized according to their unique activity on various substrates, hydrolyze the chitin in the cell walls of fungi (Ali et al. 2018). In addition to impeding fungal growth, it will also cause the production of tiny oligosaccharide elicitors that may be used to trigger and/or intensify other plant defense responses. Similar hydrolytic activity against bacteria and oomycetes is shown by other PR proteins viz. glucanases, proteinases and RNase. Peroxidase from the PR-9 family is likely involved in strengthening cell walls. PR-5 family of thaumatin-like proteins has homology to permatins that permeabilize fungal membranes. (Devi et al. 2017).

Redox Homeostasis During Plant–Pathogen Interaction

Plants' typical reaction to both abiotic and biotic stresses is the oxidative burst caused by the production of ROS such as $O_2^{\cdot-}$, H_2O_2 and OH· (Mittler et al. 2022). Two oxidative bursts of ROS buildup take place during pathogenesis: the initial burst, which lasts for around two hours, happens during the first few minutes of infection, but the second burst is more intense and lasts for many hours. The activation of plasma membrane-located NADPH oxidases which catalyzes the synthesis of $O_2^{\cdot-}$ (precursor of a wide range of ROS) is the first step in the increased ROS in response to pathogens (Barreca 2021). The role of ROS as a damaging or signaling molecule depends on the equilibrium between ROS production and quenching at the proper time and site (Sharma et al. 2012). Various constituents of antioxidative mechanisms and enzymes of different pathways that are

Table 2 Types of PR proteins according to their functions (Devi et al. 2017 and Ali et al. 2018)

Families	Functions	Site of action	Sources
PR-1	Antifungal	Active against oomycetes	*Nicotiana tabacum* PR-1a
PR-2	Endo-β-1,3-glucanases	Cell wall glucan of fungi	*Nicotiana tabacum* PR-2
PR-3	Class I, II, IV, V, VI, VII chitinases	Cell wall glucan of fungi	*Nicotiana tabacum* P, Q
PR-4	Win-like proteins/Class I, II chitinases	Active against oomycetes	*Nicotiana tabacum* "R"
PR-5	Thaumatin-like proteins/permatins	Cell membrane of fungi	*Nicotiana tabacum* S
PR-6	α-Amylase/protease inhibitors	Active on nematodes and insects	*Solanum lycopersicum* inhibitor I
PR-7	Endoproteases	Microbial cell wall dissolution	*Solanum lycopersicum* P69
PR-8	Class III chitinase	Cell wall chitin of fungi and mucopeptide cell wall of bacteria	*Cucumis sativus*
PR-9	Peroxidases	Strengthening of plant cell wall	*Nicotiana tabacum*
PR-10	RNase-like proteins	Genetic material of pathogen	*Petroselinum crispum* "PR1"
PR-11	Class I chitinases	Cell wall glucan of fungi	*Nicotiana tabacum*
PR-12	Defensins	Cell membrane of pathogen	*Raphanus raphanistrum* Rs-AFP3
PR-13	Thionins	Cell membrane of pathogen	*Arabidopsis thaliana* THI2.1
PR-14	Non-specific lipid-transfer proteins	Cell membrane of pathogen	*Hordeum vulgare* LTP4
PR-15	Germins/oxalate oxidase	Produce H_2O_2 extracellularly	*Hordeum vulgare* OxOa
PR-16	Germin-like/oxalate-like proteins	Produce H_2O_2 extracellularly	*Hordeum vulgare* OxOPL
PR-17	Antifungal and antiviral	Unknown	*Nicotiana tabacum* PRp27

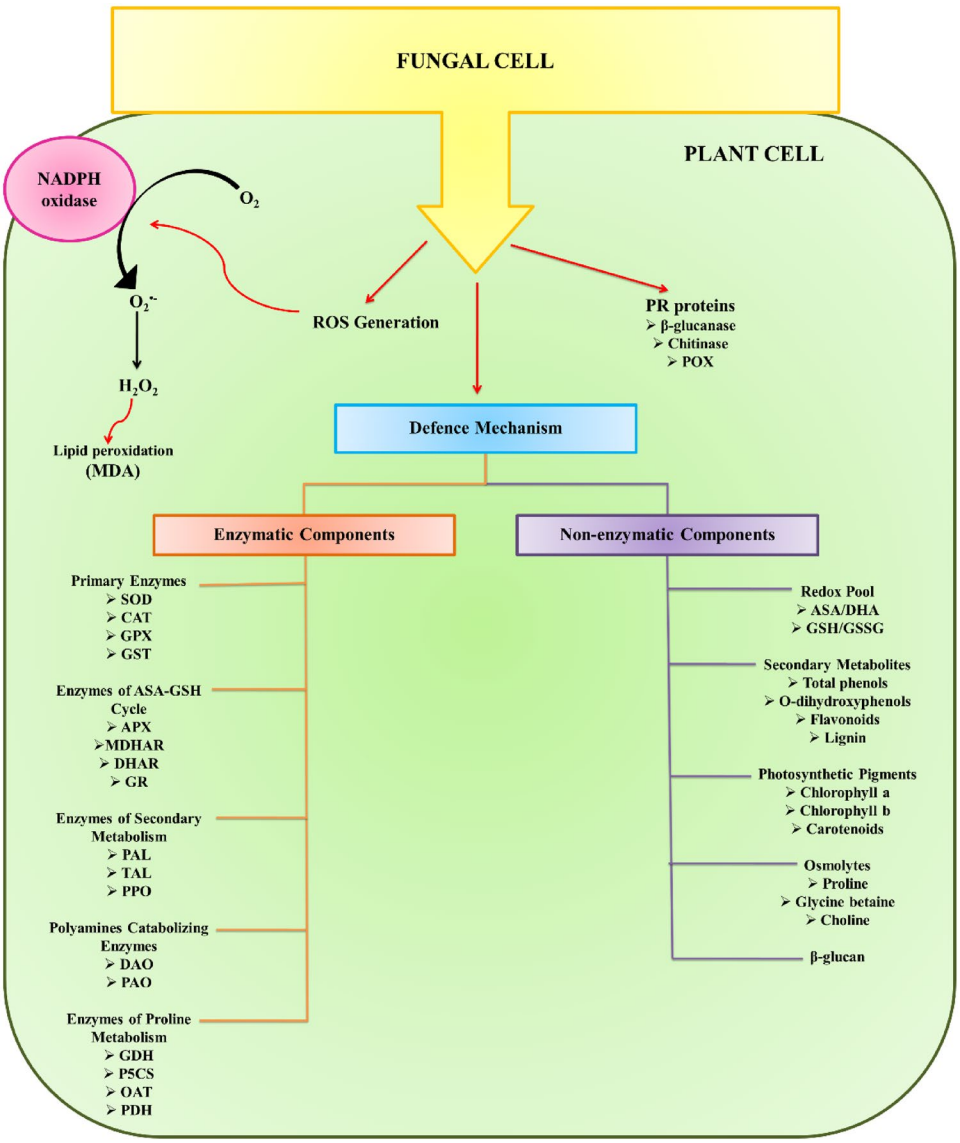

Fig. 4 Enzymatic and non-enzymatic antioxidants during plant–fungus interaction

directly or indirectly involved in defense are present in distinctive cell organelles of plants (Fig. 4, Table 3).

Superoxide dismutase (SOD), a metalloprotein catalyzes the dismutation of $O_2^{\cdot-}$ radical to H_2O_2 and O_2 (Bresciani et al. 2015). Depending upon the metal cofactors involved, three different classes of SOD (Fe-SOD, Mn-SOD and Cu/Zn-SOD) are localized in distinctive subcellular compartments. Catalase (CAT), the first discovered antioxidant enzyme is a tetrameric, heme-containing protein with the highest turnover rate i.e., one molecule of CAT can dismutate 6 million H_2O_2 molecules per min (Furukawa et al. 2017). Glutathione peroxidase (GPX), a selenium-containing enzyme, prevents lipid peroxidation by reducing H_2O_2 to H_2O (Cha et al. 2014). Glutathione-S-transferase (GST) detoxifies xenobiotics by conjugation with glutathione molecule and hence, regulates the mechanism of apoptosis during biotic and abiotic stress. Roxas et al. (2000) reported that enhanced GST to GPX ratio in the transgenic tobacco improves the peroxide scavenging property which results in better growth of the seedling under stressed and non-stressed conditions.

Ascorbate–glutathione cycle includes four enzymes viz. ascorbate peroxidase (APX), monodehydroascorbate reductase (MDHAR), dehydroascorbate reductase (DHAR) and glutathione reductase (GR) help to maintain the balance between ascorbic acid (AsA) and glutathione (GSH) pools (Kunert and Foyer 2023). Ascorbic acid donates electrons in a variety of enzymatic and non-enzymatic reactions like regeneration of α-tocopherol from tocopheroxyl radical, pH-mediated modulator of PSII activity etc., thus it is regarded as a potent antioxidant that minimizes ROS damage (Akram et al. 2017). Glutathione acts as a stress marker which is synthesized from L-glutamate, L-cysteine and L-glycine and is among the important redox buffers. It is very effective

Table 3 Various defensive enzymes and their characteristics

Defensive enzymes	EC no.	Location	Reaction/Function
NADPH oxidase	E.C. 1.6.3.1	Plasma membrane	$2O_2 + NADPH + H^+ \xrightarrow{NOX} 2O_2^{\cdot -} + NADP^+$
Superoxide dismutase (SOD)			
Fe-SOD	EC 1.15.1.1	Chloroplast	Dismutation of $O_2^{\cdot -}$ into O_2 and H_2O_2
Mn-SOD			
Cu/Zn-SOD		Mitochondria	
		Chloroplast, cytosol, peroxisomes and mitochondria	
Catalase (CAT)	EC 1.11.1.6	Mitochondria and peroxisomes	Decomposition of H_2O_2 to H_2O and O_2
Glutathione peroxidase (GPX)	EC 1.11.1.7	Cytosol, mitochondria, endoplasmic reticulum and chloroplast	Reduce H_2O_2 to H_2O
Glutathione-S-transferase (GST)	EC 2.5.1.18	Cytosol, peroxisomes and nucleus	Detoxify toxins by conjugation with GSH
Ascorbate peroxidase (APX)	EC 1.11.1.11	Cytosol, mitochondria, peroxisomes and chloroplast	H_2O_2-dependent oxidation of ascorbic acid
Monodehydroascorbate reductase (MDHAR)	EC 1.6.5.4	Cytosol, mitochondria, and chloroplast	Regenerates ascorbic acid by the reduction of monodehydroascorbate, using NADH or NADPH as an electron donor
Dehydroascorbate reductase (DHAR)	EC 1.8.5.1	Cytosol, mitochondria, and chloroplast	Reconvert the dehydroascorbic acid (DHA) into ascorbic acid using glutathione as an electron donor
Glutathione reductase (GR)	EC 1.6.4.2	Cytosol, mitochondria, and chloroplast	Regenerates GSH by the reduction of GSSG, using NADH or NADPH as an electron donor
Polyphenol oxidase (PPO)	EC 1.14.18.1	Thylakoid membrane of chloroplasts, vesicles, cytosol, mitochondria and microsomes	*o*-hydroxylation of monophenol to o-diphenols, oxidation of *o*-diphenols to o-quinones and polymerization of *o*-quinones to polyphenols
Phenylalanine ammonia lyase (PAL)	EC 4.3.1.24	Endoplasmic reticulum, proplastids and plasma membrane	Deamination of phenylalanine to yield *trans*-cinnamic acid
Tyrosine ammonia lyase (TAL)	EC 4.3.1.25	Endoplasmic reticulum, proplastids and plasma membrane	Deamination of tyrosine to form 4-hydroxycinnamic acid
Diamine oxidase (DAO)	EC 1.4.3.6	Cell wall	Oxidative deamination of biogenic amines
Polyamine oxidase (PAO)	EC 1.4.3.4	Cell wall	Oxidative deamination of biogenic amines
Glutamate dehydrogenase (GDH)	EC 1.4.1.2	Cytosol and mitochondria	Reversibly converts 2-oxoglutarate to glutamate
Δ1-Pyrolline carboxylate synthetase (P5CS)	EC 2.7.2.11, 1.2.1.41	Cytosol and chloroplast	Converts glutamate to Δ1-pyrroline-5-carboxylate
Ornithine transaminase (OAT)	EC 2.6.1.13	Mitochondria	Transamination of ornithine to Δ1-pyrroline-5-carboxylate
Proline dehydrogenase (PDH)	EC 1.5.5.2	Mitochondria	Oxidation of proline to Δ1-pyrroline-5-carboxylate

in scavenging different ROS like $^1O_2$, H_2O_2 and $OH^{\cdot -}$. It also participates in the regeneration of AsA through the AsA–GSH cycle and is responsible for the detoxification of xenobiotics and many harmful pollutants (Chin et al. 2016).

Ascorbate peroxidase belongs to the class I superfamily of heme peroxidases that utilizes ascorbate to break down the H_2O_2 and release monodehydroascorbate (MDHA) and water (Sharma et al. 2016). A flavin adenine dinucleotide (FAD) enzyme called monodehydroascorbate reductase has two isozyme types, one of which is found in the chloroplast and the other in the cytosol (Chen et al. 2019). It possesses high specificity for MDHA as e^- acceptor and NADH as an e^- donor for regenerating ascorbic acid. It is involved in the regeneration of AsA and is found co-localized in the peroxisomes and

Table 4 Status of various biochemical constituents in different crops against different fungal pathogens

Crop	Fungus	Tissue	Disease	Biochemical constituents	Status	References
Rice	*Fusarium fujikuroi*	Seeds	Bakanae (foolish seedling)	POD, SOD, PPO and PAL	Higher activities of these enzymes provide resistance	Chhabra et al. (2022)
Maize	*Exserohilum turcicum*	Leaves	Northern corn leaf blight	APX, CAT, GR, POX, chitinase, β-1,3-glucanase, PPO and PAL	Antioxidant and host defense enzymes had greater activities for + Si plants compared to -Si plants	Silveira et al. (2021)
Citrus sinensis L. (Osbeck)	*Penicillium digitatum*	Fruit	Green mold	Anthocyanin	Induction of anthocyanin biosynthesis	Sicilia et al. (2021)
Barley	*Puccinia striiformis* f. sp. *hordei*	Leaves	Stripe rust	NADPH oxidase, SOD, CAT, POX, GPX, GST, APX, MDHAR, DHAR, GR, ascorbic acid, glutathione	Resistance was due to increase in activities of NADPH oxidase, CAT, POX and enzymes of ascorbate–glutathione pathway along with ascorbate and glutathione pool	Singla et al. (2020a)
Barley	*Puccinia striiformis* f. sp. *hordei*	Leaves	Stripe rust	β-Glucan, PR proteins, diamine oxidase (DAO), polyamine oxidase (PAO), lignin, PAL, TAL, PPO, Chitinase, β-1,3-glucanase, GDH, P5CS, OAT, PDH	Upregulation of enzymes of various metabolic pathways and their key metabolites contributed to the resistance	Singla et al. (2020b)
Barley	*Bipolaris sorokiniana*	Leaves	Spot blotch	CAT, APX, GR, PAL, TAL, PPO, chitinase, β-1,3- glucanase, β-glucan	Upregulation of various defensive enzymes and PR proteins in resistant genotypes was observed	Kaur et al. (2022)
Wheat	*Pyricularia oryzae*	Leaves	Blast	APX, CAT, GR, SOD, chitinase, β-1,3-glucanase, lipoxygenase, POX, PAL, total soluble phenolics and lignin-thioglycolic acid	Higher chitinase, PAL and POX activities and lower PPO activity for inoculated plants under different thermal conditions	Silva et al. (2021)
Peanut	*Sclerotium rolfsii*	Stem	Southern blight	Carbohydrate, protein, soluble phenolics, proteinase inhibitor, JA, SA	Lower content of starch and total soluble phenolics. Higher levels of soluble sugars. However, soluble and insoluble protein, SA and JA remained unaffected	Cardoza et al. (2003)
Mulberry	Leaf spot Leaf rust Powdery mildew	Leaves	Foliar	Carbohydrate, protein, chlorophyll *a*, chlorophyll *b*	Decrease in the content of carbohydrate, protein, chlorophyll *a*, chlorophyll *b* in infected mulberry leaves	Kumar et al. (2014)

mitochondria with APX. Dehydroascorbate reductase (DHAR) catalyzes the reduction of DHA to AsA by using GSH as an e^- donor. Thus, it is another enzyme in addition to MDHAR which restores the AsA pool in both the symplast and apoplast of the cell (Kunert and Foyer 2023). Glutathione reductase (GR) is a flavoprotein oxidoreductase that uses NADPH as a reducing agent to reduce GSSG to GSH.

Phenolic compounds are natural phytochemicals that chelate metal ions and become an important antioxidant produced by plants. All phenolic compounds exhibited more than 85% scavenging activity due to their high reactivity with $OH^{\cdot -}$ (Mathew et al. 2015). Polyphenol oxidase (PPO) utilizes molecular oxygen to oxidize phenols to quinones, altering food proteins in plants and rendering them indigestible to pathogens (Araji et al. 2014). By catalyzing the first step of the phenylpropanoid pathway, PAL serves as a critical switch between primary and secondary metabolism (Jun et al. 2018). Tyrosine ammonia lyase (TAL) is another enzyme in the phenylpropanoid pathway that converts L-tyrosine to coumaric acid. Polyamines are phytohormone-like aliphatic amines and their biosynthesis and buildup under stress are essential as they are involved in plant growth and development, anti-senescence, antioxidative defense system and stabilization of cell wall (Liu et al. 2019). However, different polyamines (ornithine, citrulline, putrescine, spermidine, spermine and cadaverine) are degraded by the action of various oxidases. For instance, putrescine which is synthesized from spermidine and spermine by the action of polyamine oxidase (PAO), is further converted to Δ^1-pyrroline, H_2O_2 and NH_3 by diamine oxidase (DAO).

In response to stress, proline acts as an important osmoprotectant, metal chelator, protein chaperone, inhibitor of lipid peroxidation and ROS scavenger for OH· and $O_2^{\cdot -}$ species that mitigate adverse effects of ROS (Dar et al. 2016). It is synthesized from L-glutamic acid by the action of Δ^1-pyrroline-5-carboxylate synthetase (P5CS) in the cytosol and plastids. Another pathway for proline biosynthesis via ornithine operates in mitochondria that utilize ornithine to yield glutamate and P5C with the help of ornithine aminotransferase (OAT) (Szepesi and Szollosi 2018). Moreover, proline degradation occurs in mitochondria by proline dehydrogenase (PDH), a flavoprotein that catalytically converts proline to Δ-pyrroline-5-carboxylate (P5C). Numerous studies have been conducted for evaluating the current status of various enzymatic and non-enzymatic anti-oxidants in different crops owing to different fungal pathogens (Table 4).

Conclusions

Field crop yield and quality can suffer greatly from fungal pathogens, which eventually have an impact on the world economy. Understanding fungal pathogenesis not only improves our comprehension of how fungal infections impact their host plants, but also uncovers crucial details for the control of plant diseases, such as novel methods to stop or suppress fungal growth. Moreover, to create new varieties with durable disease resistance and to reduce the usage of harmful agrochemicals, it is crucial to have a proper understanding of how plants defend themselves against pathogens. Thus, the present review provides the comprehensive knowledge of evolutionary processes occurring during plant–fungal interaction that could be of great help in unravelling the different factors and defensive mechanisms responsible for imparting resistance against fungal attack. Furthermore, understanding the potential of ascorbate–glutathione cycle, phenylpropanoid pathway, phenolics and polyamines in maintaining redox homeostasis in plant after fungal infection could hopefully lead to discoveries of how selective redox signaling networks orchestrate the plant immune response.

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