International Research Journal of Pharmacy and Pharmacology (ISSN 2251-0176) Vol. 1(10) pp. 242-249, December 2011 Special Issue
Available online http://www.interesjournals.org/IRJPP
Copyright © 2011 International Research Journals

Review

Host Pathogen Interaction at the Plant Cell Wall

Jayamohan NS and Kumudini BS*

Department of Biotechnology, Center for Post Graduate Studies, Jain University, Bangalore
18/3, 9th Main, 3rd Block, Jayanagar, Bangalore – 560011

Accepted 05 December, 2011

Plants produce various defense compounds as a response to pathogen infection. Pathogen also produces many enzymes to establish itself in the host cell. The result is an interaction between the host and the pathogen at the host cell wall, a highly dynamic structure. The host defense responses includes structural as well as biochemical responses, the details of which have been reviewed here.

Keywords: Plant defense response, cell wall appositions, defense barrier; reactive oxygen species

INTRODUCTION

Plants are ubiquitous green factories on earth. Unlike mammals they lack mobile defender cells and a somatic adaptive immune system. Instead, they rely on the innate immunity of each cell and on systemic signals emanating from infection sites (Jens and Christina, 2009; Amil-Ruiz et al., 2011). Plant pathogens adopt diverse life strategies and can be broadly divided into those that kill the host and feed on the contents (necrotrophs) and those that require a living host to complete their life cycle (biotrophs). Microbial necrotrophy is often accompanied by production of toxins (AbuQamar et al., 2006). Pathogenic bacteria proliferate in intercellular spaces (apoplast) after entering through gas (stomata) or water (hydathodes) or gain access via wounds (Melotto et al., 2008). Nematodes and aphids feed by inserting a stylet directly into a plant cell (Bos et al., 2010) whereas fungi directly enters epidermal cells or extend hyphae on top of, between or through plant cells. Pathogenic and symbiotic fungi have haustoria. These diverse pathogen classes all deliver effector molecules (virulence factors) into the plant cell to enhance microbial fitness (Dangl and Jones, 2001; Craig et al., 2009; Amil-Ruiz et al., 2011).

Plant cell wall is a highly dynamic structure that besides providing mechanical support needs to respond to various environmental and developmental cues and fulfils important functions in signaling events, the defence against biotic and abiotic stresses and growth (Bosch et al., 2011). It also constitutes a reservoir of antimicrobial compounds and is a source of signaling molecules (Carpita and McCann, 2000; Hernández-Blanco et al., 2007; Almagro et al., 2009).

Plant cell walls are divided into two categories: primary walls, that surround growing cells and secondary walls that are thickened structures containing lignin and surrounding specialised cells (Keegstera, 2010). Structurally three layers may be found in plant cell walls- Middle lamella, Primary cell wall and Secondary cell wall (Buchanan et al., 2000). The main components of the primary cell wall of dicots are a cellulose-xyloglucan network which is the main load-bearing structure that is embedded in a matrix of pectic polysaccharides (Eckardt, 2004). The pectic matrix has three major components- homogalactouronan, RG I and RG II. In addition to effect on wall strength and cell adhesion, pectins also control wall porosity (Baron-Epel et al., 1988, Liberman et al., 1999; Diet et al., 2006; Cosgrove, 2005), which in turn regulates the mobility of cell wall modifying proteins and thus, cell wall expansion (Willats et al., 2001; McCartney et al., 2003).

Virulence exerted by pathogens

To be successful in attacking a host cell, a pathogen must pass the outer barrier of a cell. A simple but major pathogenic mechanism in plants involve cell wall degradation by a battery of polysaccharidases secreted by pathogens. Most of the degradative enzymes are glycoside hydrolases, which degrade the cellulose and pectate matrices by the addition of water to break the

*Corresponding author email: kumudini_satyan@yahoo.co.in

glycosidic bonds. The plant cell wall is composed of two types of polysaccharide matrices: the pectate network and the cellulose network (Herron et al., 2000). The pectate network consists of the smooth region composed of homogalacturonans and the hairy region composed of highly branched rhamnogalacturonans.

Enzymes, which degrade the pectate network, belong to two classifications: glycoside hydrolases and polysaccharide lyases. Glycoside hydrolases incorporate a water molecule via a general acid catalysis during the cleavage of the glycosidic bond between the two saccharide units. In contrast, polysaccharide lyases cleave the glycosidic bond via a β-elimination reaction that removes a proton. The final product contains an unsaturated bond between C-4 and C-5 of the saccharide unit at the non-reducing end. Enzymes, which degrade the cellulose network, all function as glycoside hydrolases. Generally, hydrolases have acidic pH optima, using aspartic and glutamic acid groups during catalysis, whereas lyases have basic pH optima, using catalytic amino acids that are still under active investigation (Herron et al., 2000).

In certain pathogens, such as *Erwinia chrysanthemi*, the genetic organization and regulation of many secretory saccharidases have been elucidated (Reverchon et al., 1997). One finding is that many pathogenic organisms secrete multiple isozymes of the same enzyme but the transcription of the genes is often independently regulated (Herron et al., 2000).

Pathogen induced local responses

Hypersensitive response (HR) is one of the most powerful weapons in a plant's arsenal against pathogen attack and is characterized by rapid, localised cell death at the site of infection (Hammond-Kosack and Jones, I996). This cell death response likely benefits the plant by depriving pathogens of access to further nutrient sources and limiting pathogen proliferation (Kumudini et al., 2001). The HR cell death is often preceded by changes in ion fluxes, oxidative burst and cross-linking of cell wall proteins. Most of the HR is usually accompanied by an increase in salicylic acid (SA) biosynthesis, transcriptional activation of various pathogenesis-related (PR) genes and the establishment of a long-lasting systemic response known as systemic acquired resistance (SAR) (Dangl et al., 1996; Hammond-Kosack and Jones, I996; Devadas and Raina, 2002; Boyle et al., 2009; Durrant and Dong, 2004). Production of reactive oxygen species (ROS) is associated with HR and is one of the earliest responses of plants to microbial pathogens (Cohn et al., 2001; Torres et al., 2006; de León, 2011).

Evidence indicates hypersensitive cell death is a form of programmed cell death that resembles apoptotic cell death in other organisms (Morel and Dangl, 1997; Tsujimoto and Shimizu. 2005; Mishra et al., 2011).

Morphologically, a key difference between programmed cell death of plant cells and apoptosis in animals is the absence of engulfment by neighbouring cells in plants (Lam, 2004). Experiments on several *Arabidopsis* mutants with spontaneous cell death that mimic pathogen-induced cell death support the idea that hypersensitive cell death may be controlled by plant's own genetic mechanisms (Greenberg, 1997; Glazebrook, 1999; Greenberg and Yao, 2004; Hofius et al., 2011)..

Reactive Oxygen Species (ROS)

Production of ROS is one of the earliest cellular responses following successful pathogen recognition via consumption of oxygen in a so-called oxidative burst (Ashry and Mohamed, 2011). The oxidative burst has been known for more than 30 years in mammals (Wojtaszek, 1997). However, in plants the phenomenon was demonstrated much later (Doke, 1983). ROS molecules have an important role in some physiological processes like plant growth and development (Mendoza, 2011). Apoplastic generation of superoxide (O_2^-), or its dismutation byproduct hydrogen peroxide (H_2O_2), singlet oxygen (O_2^-) and hydroxyl radical (OH^-) has been documented following recognition of a variety of pathogens (Torres et al., 2006; Mendoza, 2011). Although the primary oxidative burst following pathogen recognition occurs in the apoplast, ROS produced in other cellular compartments may also have functions in defense. High levels of ROS can be produced inside the plant cell as by-products of metabolic processes, especially, light-driven production of ROS as a by-product of photosynthesis (Karpinski et al., 2003; Apel and Hirt, 2004; Gill and Tuteja, 2010).

The different ROS includes - H_2O_2, can act as a local signal for cell death and also as a diffusible signal for the induction of defensive genes in adjacent cells (Alvarez et al., 1998; Torres et al., 2006). O_2^-, in living cells exists in equilibrium with its protonated form, the hydroperoxyl radical (O_2H^-). At the physiological level, pH is not very reactive against major macromolecular components of the cell (Michalak, 2006). OH^- is the most reactive species responsible for the irreversible modifications of cellular macromolecules and damage of organelles (Wojtaszek, 1997; Elesak et al., 2007; Galaris et al., 2008; Torres et al., 2006). ROS metabolism during the pathogen attack is followed by assistance of various antioxidant enzymes such as peroxidase (POX), superoxide dismutase (SOD), catalase (CAT) and ascorbate peroxidase (APX). POX is also produced as a defence response stimulated in plants in response to pathogen infection like *Fusarium oxysporum* (Morkunas and Gemerek, 2007; Ashry and Mohamed, 2011).

POX is among the major oxido-reductive enzymes that participate in the wall-building processes such as oxidation of phenols, suberization and lignifications of host plant cells during the defense reaction

against pathogenic agents (Chittoor and Leach, 1999). One of the important physiological roles of POXs is the synthesis of cell-wall polymers (lignin and suberin), which constitute physical barriers for both biotic and abiotic stresses (Cosgrove, 1997; Quiroga et al., 2000; Cai et al., 2009) which might confer the plant with high rigidity. Recent pharmacological experiments indicate that nitric oxide (NO) -a signal found in the immune, nervous, and vascular system of vertebrates also- plays an important role in plant disease resistance. Generation of NO augments the induction of HR cell death by H_2O_2 in soybean (*Glycine max*) suspension cultures (Delledonne et al., 1998; 2001). Likewise, inhibitors of NO synthesis as well as NO scavengers are able to block the HR induced by avirulent *Pseudomonas syringae* in soybean cell cultures and in *Arabidopsis* plants. Compared to ROS, NO induces a complementary set of plant defense genes, including two key enzymes of the phenylpropanoid pathway, namely Phenyl alanine ammonia lyase (PAL) and chalcone synthase (CHS) (Zeier et al., 2004; Ferrer et al., 2008).

Recognizing pathogens

Plants have evolved two strategies to detect pathogens (Chisholm et al., 2006; Jones and Dangl, 2006). On the external face of the host cell, plants are equipped to sense evolutionarily conserved microbial molecular signatures, collectively called pathogen-associated molecular patterns (PAMPs) or microbe-associated molecular patterns (MAMPs), are recognized by receptor proteins called pattern recognition receptors (PRRs) (Boller and Felix, 2009). PAMPs are typically essential components of whole classes of pathogens, such as bacterial flagellin or fungal chitin. Plants also respond to endogenous molecules released by pathogen invasion, such as cell wall or cuticular fragments called danger-associated molecular patterns (DAMPs). Stimulation of PRRs leads to PAMP-triggered immunity, an ancient form of innate immunity (Chisholm et al., 2006; Jones and Dangl, 2006).

The second class of perception involves recognition by intracellular receptors of pathogen virulence molecules called effectors; this recognition induces effector-triggered immunity (ETI). This mode of recognition leads to co-evolutionary dynamics between the plant and pathogen that are quite different from PTI in contrast to PAMPs, effectors are characteristically variable and dispensable. Generally, PTI and ETI give rise to similar responses, although ETI is qualitatively stronger and faster and involves HR (Dodds and Rathjen, 2010). PAMPs are detected by pattern recognition receptors (PRRs), typically cell surface-localized receptor kinases or LRR-RLP proteins (Zhang and Zhou, 2010).

Deposition of papillae

Following germination of pathogen on the leaf surface forms a specialized infection structure called appressorium. A penetration peg emerges from the appressorium and it penetrate the host leaf epidermal cell directly (approximately 12–20 h). If penetration succeeds and the host cell remains alive, a feeding structure develops called haustorium, which extracts nutrients to supply the development of superficial hyphae that ramify over the leaf surface and form a colony (Vanacker et al., 2000). Host cells respond to attempted penetration by depositing wall appositions, papillae, directly beneath appressoria. Papillae have been found to contain callose, phenolic compounds, lignin, ROS, and proteins and are thought to act as a physical barrier to halt penetration by the fungal penetration pegs (Aist, 1976; McLusky et al., 1999; Lyngkjaer and Carver, 1999; Underwood and Somerville, 2008). Their deposition involves generation of NO (Prats et al*.,* 2005) and H_2O_2 (Vanacker et al*.,* 2000), cytoskeletal rearrangement (Kobayashi et al*.,* 1997; Opalski et al*.,* 2005) and redirected cytoplasmic streaming and aggregation (Zeyen *et al.,* 2002; Ridout, 2009).

These events direct vesicles containing papilla components to the site of attempted penetration. Vesicle targeting involves SNARE proteins and the general membrane trafficking factor SNAP (Assaad et al*.,* 2004), suggesting that papilla formation is mediated by processes akin to membrane/vesicle trafficking in animal systems (Pelham, 2001; Böhlenius et al., 2010). Effective papilla defence also enhances the ability of cells adjacent to the attacked cell to form papillae in response to subsequent attacks (Lyngkjær and Carver, 2000; Prats et al., 2006), indicating that intercellular communication is a consequence of the response.

Cuticular layer as a defensive barrier

Cuticle is considered to constitute a physical barrier to microbial invaders through which cutinase-producing pathogens can penetrate. In addition to its role as a barrier, the cuticle is likely to be a source of signals used by invading pathogens to prepare and adjust for the colonization of their host. Production of cutinase in *Fusarium solani* f. sp. *pisi* is induced by cutin monomers present in the surrounding medium (Woloshuk et al., 1986). A model was proposed whereby fungi sense the presence of cutin monomers on the plant surface and induce high levels of cutinase required for invasion (Kolattukudy, 1985). Cuticular components can also regulate developmental processes in pathogenic fungi. For instance, cutin monomers induce germination and appressorium in *Magnaporthe grisea* (Gilbert et al*.,* 1996; Skamnioti and Gur, 2007) and formation of the

appressorial tube in *Erysiphe graminis* (Francis et al.,1996). Screening mechanism of cell wall intactness in hostile environment is not known. Plants can sense a variety of molecules released during interaction with pathogens (Bais et al., 2004). In particular, breakdown products of the plant cell wall are known to act as elicitors of defences (Boller, 1995).

Callose deposition and cell wall appositions (CWA)

Callose is a linear homopolymer made up of β-1,3-linked glucose residue with some β-1,6-branches, is widespread in higher plants, in which it is a component of specialized cell walls or cell wall-associated structures at particular stages of growth and differentiation (Stone and Clarke, 1992). Callose has many functions in plant development, as involved at multiple stages of pollen development (Stone and Clarke 1992; McCormick, 1993; Backues et al., 2010), deposited at cell plates during cytokinesis (Samuels et al., 1995; Hong et al., 2001), deposited at plasmodesmata to regulate the cell-to-cell movement of molecules by controlling the size exclusion limit of plasmodesmata (Iglesias and Meins, 2000). Preinvasive basal defense against many pathogenic fungi is manifested at the stage of penetration by the formation of a local CWA called papilla (Böhlenius et al, 2010). To prevent pathogen penetration, plant cells respond by local reinforcement of the cell wall beneath the site of the penetration attempt by forming a papilla. This process involves deposition of the callose matrix together with the accumulation of components such as H_2O_2, phenolics and various proteins and glycoproteins with hydrolytic and antifungal properties (Flors et al., 2005). Pathogen-induced callose could negatively regulate the SA signaling pathway of plants which results in increased resistance to pathogen (Chen and Kim, 2009). Formation of CWA is achieved by rapid reorganisation of actin microfilaments, actin-dependent transport of secretory products to the infection site and local activation of callose synthesis. Qualitative cytochemical experiments shown that callose was widely distributed in the underlying matrix of wall appositions (Kumudini and Shetty, 2002). Callose is less permeable to small molecules than other cell wall componants and may there for restrict the passage of nutrients to the fungus and consequently to slow fungal growth, so that the other defence can act further to restrict the progress of infection (Silva et al., 2006). Wound callose is formed rapidly, mostly within minutes of wound initiation and is deposited between the plasma membrane and the cell wall (Nakashima et al., 2003).

Role of lignin in plant defence

Lignin is the second most abundant polymer found in nature (Jung and Weiting 1998) is a polymeric material composed of phenylpropanoid units derived from three cinnamyl alcohols (monolignols): *p*-coumaryl, coniferyl, and sinapyl alcohols (Hatfield and Vermerris, 2001). From a functional point of view, lignins impart strength to cell walls, facilitate water transport, and impede the degradation of wall polysaccharides, thus acting as a major line of defense against pathogens, insects, and other herbivores (Hatfield and Vermerris, 2001). It retards the enzymatic digestion of the host cell wall by pathogens. Following their release, activation of enzymes may lead to oxidative linkage of phenolics on the plant cell wall, even without major transcriptional activation of biosynthetic pathways (Kumudini, 2005). Lignification is a mechanism for disease resistant in plants, and it renders the cell wall more resistant to mechanical pressure applied during penetration by fungal appressoria as well as more water resistant and thus less accessible to cell wall-degrading enzymes (Vance et al., 1980). Lignin formation occurs through a series of steps including many enzymes starting with phenylalanine ammonia lyase catalyzed reaction. Terminating reaction requires H_2O_2 and cell wall bound POX that polymerises the C6-C3 units into lignin (Marjamaa et al., 2009). As lignin polymerizes, it forms covalent cross-links with carbohydrate and protein and renders cell walls highly resistant to mechanical and enzymatic disruption (Lattanzio et al., 2006).

Phenolic compounds

Phenolic compounds are natural constituents of all plants and antibiotic phenols have been implicated in plant defense mechanisms (Baker et al., 2005). Among them, some occur constitutively and function as preformed inhibitors associated with non-host resistance while others are formed in response to pathogen ingress as part of an active defense response (Bahadur et al., 2007). Accumulation of autoflouresent phenolic compounds at the site of penetration is one of the readily observed host defence response. Plants need phenolic compounds for pigmentation, growth, reproduction, resistance to pathogens and for many other functions. Studies have shown that plant defence against pathogens, nematodes, phytophagous insects is based on the synthesis, release and accumulation of various phenolic compounds (Makoi and Ndakidemi, 2007). Defense gene products include polyphenol oxidase, peroxidase (POD) that catalyzes the formation of lignin, and phenylalanine ammonia-lyase (PAL) that is involved in phenolics synthesis (Raju et al., 2008).

Phenolic compounds delivered along the phenylpropanoid pathway play an important role in defense to pathogen infection either as preformed or postinfectional defense factors. They have been assigned to various important biological functions in defense such as cell wall reinforcement and antimicrobial activity as modulators of plant hormones in defense signaling or as scavengers of reactive oxygen

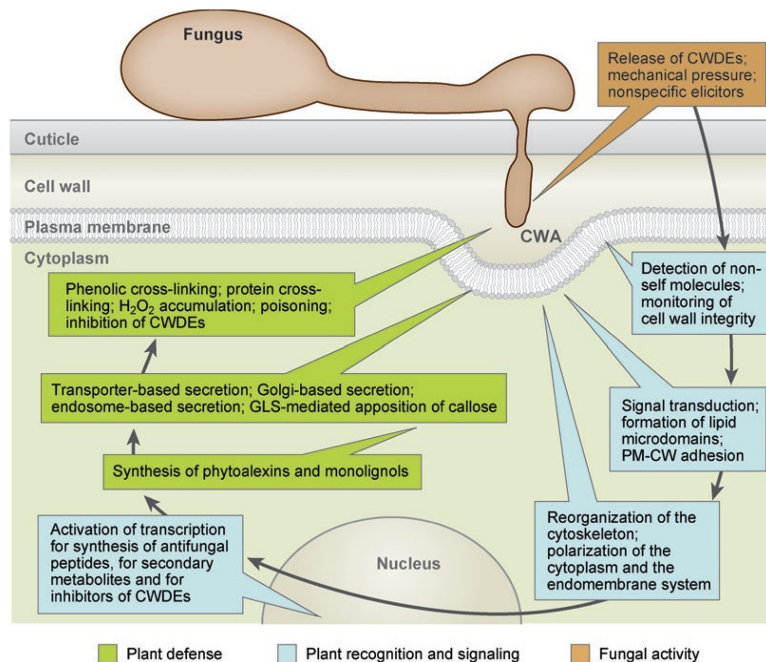

Figure 1. Biochemical and molecular mechanisms for cell wall-associated defense (Huckelhoven, 2007)

species (Tuncel and Nergiz, 1993). Particularly the phenolic polymer lignin is an important principal structural component of secondary vascular tissue and fibers in higher plants (Chen et al., 2009).

Putative guanidine compounds.

These are guanidine containing compounds located in papillae and HR cells, with high pKa value can also polarize the membranes and are able to adversely affect the pathogenic fungal growth can be demonstrated by Sakaguchi reaction. Some of the important compounds containing guanidine include arginine, agmatine and hordatine. Arginine is found in plant chromosomes, agmatine occurs in phenyl propanoid dimers, which are componants of lignin and hordatine is an antifungal compound. It has been speculated that guanidine containing compound can act as an inhibitory substance to prevent the pathogen (Wei et al., 1994)

Hydroxyproline- rich glycoproteins (HRGPs)

HRGPs are abundant structural proteins in the plant cell wall. This generic term includes molecules rich in hydroxyproline/ proline: extensin, arabinogalactan-proteins, proline/ hydroxyproline-rich proteins and solanaceous lectin (Sommer-Knudsen et al., 1998). These proteins are known to be involved in plant defence, both in dicots (Esquerré-Tugayé et al., 1979; Mazau and Esquerré-Tugayé, 1986) and in monocots (Kang and Buchenauer, 2003; Shailasree et al., 2004). The involvement of HRGPs in plant defence is likely because of early and massive accumulation in the cell wall together with the relative transcripts (Templeton et al., 1990) and in tissues immediately adjacent to the inoculation site in incompatible combinations (Benhamou et al., 1990); their accumulation is highly localized at sites where bacterial and fungal growth is arrested (O'Connell et al., 1990); artificial induction of HRGP increases resistance, whereas inhibition decreases it (Toppan et al., 1982).

HRGP accumulation and cross-linking processes in response to pathogen attacks has been noted (Bradley et al., 1992; Brisson et al., 1994; Brady and Fry, 1997; Shailasree et al., 2004) and the HRGP gene-encoding sequence has been studied (Garcìa-Muniz et al., 1998). HRGP mRNA accumulation has been induced by application of elicitors isolated from fungi and accumulation of the transcripts has also been induced by exogenous SA administration to cultured parsley cells (Thulke and Conrath, 1998), but the relationships between SA or acibenzolar-S-methyl level and HRGP accumulation are still largely unknown (Raggi, 2007; Huckelhoven, 2007).

REFERENCES

AbuQamar S, Chen X, Dhawan R, Bluhm B, Salmeron J, Lam S, Dietrich RA, Mengiste T (2006). Expression profiling and mutant analysis reveals complex regulatory networks involved in *Arabidopsis* response to Botrytis infection. The Plant J. 48 (1):28-44.

Aist JR (1976). Papillae and related wound plugs of plant cells. Annu.

Rev. Plant Physiol. 14:145–163.
Almagro L, Gómez Ros LV, Belchi-Navarro S, Bru R, Ros Barceló A, Pedreño MA (2009). Class III peroxidases in plant defence reactions. J. Exp. Bot. 60(2):377-390.
Alvarez ME, Penell RI, Meijer PJ, Ishikawa A, Dixon RA, Lamb C (1998). Reactive oxygen intermediates mediate a systemic signal network in the establishment of plant immunity. Cell. 92:773-784.
Amil-Ruiz F, Blanco-Portales R, Muñoz-Blanco J, Caballero JL (2011). The Strawberry Plant Defense Mechanism: A Molecular Review. Plant Cell Physiol. 52(11):1873-1903.
Apel K, Hirt H (2004). Reactive oxygen species: metabolism, oxidative stress, and signal transduction. Annu. Rev. Plant Biol. 55:373–399.
Ashry NA, Mohamed HI (2011). Impact of Secondary Metabolites and Related Enzymes in Flax Resistance and or Susceptibility to *Powdery Mildew*. World J. Agric. Sci. 7(1):78-85.
Assaad FF, Qiu JL, Youngs H, Ehrhardt D, Zimmeril L, Kalde M, Wanner G, Peck SC, Edward H, Ramonell K, Somerville CH, Thordal-Christensen H (2004). The PEN1 Syntaxin defines a novel cellular compartment upon fungal attack and is required for the timely assembly of papillae. MBoC. 15:5118-5129.
Backues SK, Korasick DA, Heese A, Bednarek SY (2010).The *Arabidopsis* Dynamin-Related Protein2 Family Is Essential for Gametophyte Development. The Plant Cell. 22(10):3218-3231
Bahadur A, Singh UP, Sarma BK, Singh DP, Singh KP, Singh A (2007). Foliar Application of Plant Growth-Promoting Rhizobacteria Increases Antifungal Compounds in Pea (Pisum sativum) Against Erysiphe pisi. Mycobiology 35(3):129-134.
Bais HP, Park S-W, Weir TL, Callaway RM, Vivanco JM (2004). How plants communicate using the underground information superhighway. Trends. Plant Sci. 9(1):26-32.
Baker CJ, Whitaker BD, Mock NM, Rice C, Deahl KL, Roberts DP, Ueng PP, Averyanov AA (2005). Differential induction of extracellular bioactive phenolics that are redox sensitive. Physiol. Mol. Plant Pathol. 66:90-98.
Baron-Epel O, Gharyal PK, Schindler M (1988). Pectins as mediators of wall porosity in soybean cells. Planta 175:389–395.
Benhamou N, Mazau D, Esquerré-Tugayé MT, Asselin A (1990). Immunological localization of hydroxyproline-rich glycoproteins in necrotic tissue of Nicotiana tabacum L. cv. Xanthi-nc infected by tobacco mosaic virus. *Physiol. Mol. Plant Path*. 36:129-145.
Böhlenius H, Mørch SM, Godfrey D, Nielsen ME, Thordal-Christensen H (2010). The multivesicular body-localized GTPase ARFA1b/1c is important for callose deposition and ROR2 Syntaxin-dependent preinvasive basal defense in *Barley*. The Plant Cell. 22 (11): 3831-3844.
Boller T (1995). Chemoperception of microbial signals in plant cells. Annu. Rev. Plant Biol. 46:189–214.
Boller T, Felix G A (2009). Renaissance of elicitors: perception of microbe-associated molecular patterns and danger signals by pattern-recognition receptors. Annu. Rev. Plant Biol. 60:379–406.
Bos JIB, Prince, Pitino M, Maffei ME, Win J, Hogenhout SA (2010). A functional genomics approach identifies candidate effectors from the aphid Species *Myzus persicae* (Green Peach Aphid). PLoS Genet. 6(11):1-13.
Bosch M, Mayer C, Cookson A, Donnison IS (2011). Identification of genes in cell wall biogenesis in grasses by differential gene expression profiling of elongation and non-elongating maize internodes. J. Exp. Bot. pp1-17.
Boyle P, Su EL, Rochon A, Shearer HL, Murmu J, Chu JY, Fobert PR, Després C (2009). The BTB/POZ domain of the *Arabidopsis* disease resistance rrotein NPR1 interacts with the repression domain of TGA2 to negate its function. The Plant Cell. 21:3700-3713.
Bradley DJ, Kjellbom P, Lamb CJ (1992). Elicitor- and wound-induced oxidative cross-linking of a proline-rich plant cell wall protein: a novel, rapid defense response. Cell. 70:21-30.
Brady JD, Fry SC (1997). Formation of di-isodityrosine and loss of isodityrosine in the cell walls of tomato cell-suspension cultures treated with fungal elicitors or H_2O_2. Plant Physiol. 115:87-92.
Brisson LF, Tenhaken R, Lamb C (1994). Function of oxidative cross-
Buchanan BB, Gruissem W, Jones RL (2000). Biochemistry & molecular biology of plants (1st ed.). American society of plant physiology.
Bucher GL, Tarina C, Heinlein M, Di Serio F, Meins F Jr, Iglesias VA (2001). Local expression of enzymatically active class I β-1, 3-glucanase enhances symptoms of TMV infection in tobacco. Plant J. 28:361–369.
Cai K, Gao D, Chen J and Luo S (2009). Probing the mechanisms of silicon-mediated pathogen resistance. Plant Signal. Behav. 4(1):1–3.
Carpita NC and McCann M (2000). The cell wall. In Biochemistry and Molecular Biology of Plants, Buchanan B, Gruissem W and Jones RL. eds (Rockville, MD: American Society of Plant Physiologists), pp. 52–108.
Chen C, Meyermans H, Burggraeve B, De Rycke RM, Inoue K, DeVleesschauwer V, Steenackers M, VanMontagu MC, Engler GJ, Boerjan WA. (2009). Cell-specific and conditional expression of caffeoyl-coenzyme A-3-O-methyltransferase in *poplar*. Plant Physiol. 123(3):853-868.
Chen XY and Kim JY (2009). Callose synthesis in higher plants. Plant Signal Behav. 4(6):489–492.
Chisholm ST, Coaker G, Day B & Staskawicz BJ (2006). Host–microbe interactions: shaping the evolution of the plant immune response. Cell. 124:803–814.
Chittoor JM and Leach FF (1999). Induction of peroxidase during defense against pathogens, in: S.K. Datta, S. Muthukrishnan (Eds.), Pathogenesis: Related Proteins in Plants, CRC Press, Boca Raton, FL, pp: 291.
Cohn J, Sessa G, Martin GB (2001). Innate immunity in plants. Curr. Opin. Immunol. 13:55–62.
Cosgrove DJ (1997). Assembly and enlargement of the primary cell wall in plants. Annu. Rev. Cell Dev. Biol. 13:171-201.
Cosgrove DJ (2005). Growth of the plant cell wall. Nature Rev. Mol. Cell Biol. 6:850-861.
Craig A, Ewan R, Mesmar J, Gudipati V, Sadanandom A (2009). E3 ubiquitin ligases and plant innate immunity. J. Exp. Bot. 60(4):1123-1132.
Dangl JL, Dietrich RA, Richberg MH (1996). Death don't have no mercy: cell death programs in plant-microbe interactions. Plant Cell. 8:1793-1807.
Dangl JL, Jones JDG (2001). Plant pathogens and integrated defence responses to infection. Nature. 411:826–833.
de León P (2011). The *Moss physcomitrella* patens as a model system to study interactions between plants and phytopathogenic fungi and *OomycetesInés*. Journal of Pathogens. 2011:1-6.
Delledonne M, Xia Y, Dixon RA, Lamb C (1998). Nitric oxide functions as a signal in plant disease resistance. Nature. 394: 585–588
Delledonne M, Zeier J, Marocco A, Lamb C (2001). Signal interactions between nitric oxide and reactive oxygen intermediates in the plant hypersensitive response. Proc. Natl. Acad. Sci. 98:13454–13459.
Devadas SK, Raina R (2002). Pre-existing systemic acquired resistance suppresses hypersensitive response-associated cell death in *Arabidopsis-hrl1* mutant1. Plant Physiol. 128(4):1234-1244.
Diet A, Link B, Seifert GJ, Schellenberg B, Wagner U, Paulye M, Reiter W-D, Ringli C (2006). The *Arabidopsis* root hair cell wall formation mutant *lrx1* is suppressed by mutations in the *RHM1* gene encoding a UDP-L-rhamnose Synthase. The Plant Cell. 18:1630-1641.
Dodds PN, Rathjen JP (2010). Plant immunity: towards an integrated view of plant–pathogen interactions. Nature reviews Genetics. 11: 539-548.
Doke N (1983). Involvement of superoxide anion generation in the hypersensitive response of potato tuber tissues to infection with an incompatible race of *Phytophthora infestans* and to the hyphal wall components. Physiol. Mol. Plant. Path. 23 (3):345-357.
Durrant WE, Dong X (2004). Systemic acquired resistance. Annu. Rev. Phytopathol. 42:185–209.
Eckardt NA (2004). Inside the matrix: crystal structure of a xyloglucan endotransglycosylase. Plant Cell. 16(4):792–793.
Elesak I, Libik M, Karpinska B, Karpinski S, Miszalski Z (2007). The role of hydrogen peroxide in regulation of plant metabolism and cellular signalling in response to environmental stresses. Acta. Biochimica. Polonica. 54 (1):39-50.
Esquerré-Tugayé MT, Lafitte C, Mazau D, Toppan A, Touzé A (1979). Cell surfaces in plant-microorganism interaction. Evidence for

accumulation of hydroxyproline rich glycoproteins in cell wall of diseased plants as a defence mechanism. Plant Physiol. 64:320-326.

Ferrer J.-L, Austin MB, Stewart C Jr, Noel JP (2008). Structure and function of enzymes involved in the biosynthesis of phenylpropanoids. Plant Physiol. Biochem. 46(3):356–370.

Flors V, Ton J, Jakab G, Mauch-Mani (2005). Abscisic acid and callose: team players in defence against pathogens? J. Phytopathology. 153: 377–383.

Francis SA, Dewey FM and Gurr SJ (1996). The role of cutinase in germling development and infection by *Erysiphe graminis f. sp. hordei*. Physiol. Mol. Plant Pathol. 49: 201–211.

Galaris D, Mantzaris M, Amorgianiotis C (2008). Oxidative stress and aging: the potential role of iron. HORMONES. 7 (2):114-122.

Garcìa-Muniz N, Martínez-Izquierdo JA, Puigdomènech P (1998). Induction of mRNA accumulation corresponding to a gene encoding a cell wall hydroxyproline-rich glycoprotein by fungal elicitors. Plant Mol. Biol. 38:623-632.

Gilbert RD, Johnson AM, Dean RA (1996). Chemical signals responsible for appressorium formation in the rice blast fungus *Magnaporthe grisea*. Physiol. Mol. Plant Pathol. 48:335–346.

Gill SS, Tuteja N (2010). Reactive oxygen species and antioxidant machinery in abiotic stress tolerance in crop plants. Plant Physiol. Biochem. 48:909-930.

Glazebrook J (1999). Genes controlling expression of defense responses in *Arabidopsis*. Curr. Opin. Plant. Biol. 2:280–286.

Greenberg JT (1997). Programmed cell death in plant-pathogen interaction. Annu. Rev. Plant Physiol. Plant Mol. Biol. 48:525–545.

Greenberg JT, Yao N (2004). The role and regulation of programmed cell death in plant–pathogen interactions. Cellular Microbiol. 6(3):201–211.

Hammond-Kosack KE, Jones JDG (l996). Resistance gene-dependent plant defense responses. Plant Cell. 8:1773-1791.

Hatfield R, Vermerris W (2001). Lignin formation in plants. The dilemma of linkage specificity. Plant Physiol. 126: 1351–1357.

Hernández-Blanco C, Feng DX, Hu J, Sánchez-Vallet A, Deslandes L, Llorente F, Berrocal-Lobo M, Keller H, Barlet X, Sánchez-Rodríguez C, Anderson LK, Somerville S, Marco Y, Molina A (2007). Impairment of cellulose synthases required for *Arabidopsis* secondary cell wall formation enhances disease resistance. The Plant Cell. 19:890-903.

Herron SR, Benen JAE, Scavetta RD, Visser J, Jurnak F (2000). Structure and function of pectic enzymes: Virulence factors of plant pathogens. PNAS. 97 (16): 8762–8769.

Hofius D, Munch D, Bressendorff S, Mundy J, Petersen M (2011). Role of autophagy in disease resistance and hypersensitive response-associated cell death. Cell Death Differ. 18:1257-1262.

Hong Z, Delauney AJ, Verma DPS (2001). A cell plate-specific callose synthase and its interaction with phragmoplastin. Plant Cell. 13:755–768.

Iglesias VA, Meins Jr F (2000). Movement of plant viruses is delayed in a β-1, 3-glucanase-deficient mutant showing a reduced plasmodesmatal size exclusion limit and enhanced callose deposition. The Plant J. 21 (2):157–166.

Jens S, Christina D (2009). Plant innate immunity in: encyclopedia of life sciences (ELS). John Wiley and Sons, Ltd: Chichester.

Jones JD, Dangl J L (2006). The plant immune system. Nature 444:323–329.

Jung H-JG and Weiting (1998). Lignification of plant cell walls: Impact of genetic manipulation. Ni. Proc. Natl. Acad. Sci. 95:12742-12745.

Kang Z, Buchenauer H (2003). Immunochemical localization of cell wall-bound thionins and hydroxyproline-rich glycoproteins in *Fusarium culmorum*-infected wheat spikes. J. Phytopath.151:120-129.

Karpinski S, Gabrys H, Mateo A, Karpinska B, Mullineaux PM (2003). Light perception in plant disease defence signalling. Curr. Opin. Plant Biol. 6:390–396.

Keegstra K (2010). Plant Cell Walls. Plant Physiol. 154:483–486.

Kobayashi Y, Kobayashi I, Funaki Y, Fujimoto S, Takemoto T, Kunoh H (1997). Dynamic reorganization of microfilaments and microtubules is necessary for the expression of non-host resistance in barley coleoptile cells. The Plant J. 11:525-537.

Kolattukudy PE (1985). Enzymatic penetration of the plant cuticle by fungal pathogens. Annu. Rev. Phytopathol. 23: 223–250.

Kumudini BS (2005). Structural defence response in plants- can they be induced? Pariprashna 1(1):16-27.

Kumudini BS, Shetty HS (2002). Association of lignification and callose deposition with host cultivar resistance and induced systemic resistance of pearl millet to *Sclerospora graminicola*. Australas Plant Path. 31: 257-164.

Kumudini BS, Vasanthi NS, Shetty HS (2001). Hypersensitive response, cell death and histochemical localization of hydrogen peroxide in host and non-host seedlings infected with the downy mildew pathogen *Sclerospora graminicola*. Ann. Appl. Biol. 139: 217-225.

Lam E (2004). Controlled cell death, plant survival and development. Nature Reviews Molecular Cell Biology. 5: 305-315.

Lattanzio V, Lattanzio VMT, Cardinali A (2006). Role of phenolics in the resistance mechanisms of plants against fungal pathogens and insects. Phytochemistry: Advances in Research. 23-67.

Liberman M, Mutaftschiev S, Jauneau A, Vian B, Catessons M, Goldberg R (1999). Mung bean hypocotyl homogalacturonan: localization, organization and origin. Ann. Botany. 84:225-233.
linking of cell wall structural proteins in plant disease resistance. Plant Cell. 6:1703-1712.

Lyngkjaer MF, Carver TLW (1999). Induced accessibility and inaccessibility to *Blumeria graminis f. sp. hordei* in barley epidermal cells attacked by a compatible isolate. Physiol. Mol. Plant Pathol. 55: 151–162.

Lyngkjær MF, Carver TLW (2000). Conditioning of cellular defence responses to powdery mildew in cereal leaves by prior attack. Molecular Plant Pathology. 1:41-49.

Makoi JHJR, Ndakidemi PA (2007). Biological, ecological and agronomic significance of plant phenolic compounds in rhizosphere of the symbiotic legumes. Afr. J. Biotechnol. 6 (12): 1358-1368.

Marjamaa K, Kukkola EM, Fagerstedt KV (2009). The role of xylem class III peroxidases in lignification. J. Exp. Bot. 60 (2): 367-376.

Mazau D, Esquerré-Tugayé MT (1986). Hydroxyproline-rich glycoproteins accumulation in the cell walls of plants infected by various pathogens. Physiol. Mol. Plant Pathol. 29: 147-157.

McCartney L, Steele-King CG, Jordan E, Knox JP (2003). Cell wall pectic (1→4)-β-D-galactan marks the acceleration of cell elongation in the Arabidopsis seedling root meristem. Plant J. 33: 447–454.

McCormick S (1993). Male gametophyte development. Plant Cell. 5:1265–1275.

McLusky SR, Bennett MH, Beale MH, Lewis MJ, Gaskin P, Mansfield JW (1999). Cell wall alterations and localized accumulation of feruloyl-3-methoxytyramine in onion epidermis at sites of attempted penetration by *Botryis allii* are associated with actin polarization, peroxidase activity and suppression of flavonoid biosynthesis. The Plant Journal. 17:523–534.

Melotto M, Underwood W, He SY (2008). Role of stomata in plant innate immunity and foliar bacterial diseases. Annu. Rev. Phytopathol. 46:101-122.

Mendoza M (2011). Oxidative burst in plant-pathogen interaction. Biotecnología. Vegetal. 11(2):67 – 75.

Michalak A (2006). Phenolic compounds and their antioxidant activity in plants growing under heavy metal stress. Polish J. of Environ. Stud. 15 (4): 523-530.

Mishra S, Tyagin A, Dwivedi SP (2011). Regulation of apoptosis in living organisms: A biotechnological approach. Invited Review, Biotechnol. Bioinf. Bioeng. 1(1):1-18.

Morel JB, Dangl JL (1997). The hypersensitive response and the induction of cell death in plants. Cell Death Differ. 4:671–683.

Morkunas I, Gemerek J (2007). The possible involvement of peroxidase in defense of yellow lupine embryo axes against *Fusarium oxysporum*. J. Plant Physiol. 164:497-506.

Nakashima J, Laosinchai W, Cui X, Brown RM Jr (2003). New insight into the mechanism of cellulose and callose biosynthesis: proteases may regulate callose biosynthesis upon wounding. Cellulose 10:369–389.

O'Connell RJ, Brown IR, Mansfield JW, Bailey JA, Mazau D, Runeau D, Esquerré-Tugayé MT (1990). Immunocytochemical localization of hydroxyproline-rich glycoproteins accumulating in melon and bean at sites of resistance to bacteria and fungi. Mol. Plant. Microbe Interact. 3:33-40.

Opalski KS, Schultheiss H, Kogel KH, Huckelhoven R (2005). The receptor-like MLO protein and the RAC/ROP family G-protein

RACB modulates actin reorganization in barley attacked by the biotrophic powdery mildew fungus *Blumeria graminis f. sp hordei*. The Plant J. 41:291-303.

Pelham HRB (2001). Traffic through the Golgi apparatus. J. Cell Biol. 155:1099-1101.

Prats E, Mur LAJ, Sanderson R, Carver TLW (2005). Nitric oxide contributes both to papilla-based resistance and the hypersensitive response in barley attacked by *Blumeria graminis f. sp. hordei*. Molecular Plant Pathology. 6:65-78.

Quiroga M, Guerrero C, Botella MA, Barcelo A, Amaya I, Medina MI, Alfonso FJ, Milrad S. De Forchetti, Tigier H, Valpuesta V (2000). A tomato peroxidase involved in the synthesis of lignin and suberin. Plant Physiol. 122: 1119-1128.

Raggi V (2007). Local and systemic hydroxylproline- rich glycoprotein accumulation in tobacco treated with salicylic acid and Acibenzolar-S- methyl. Journal of Plant Pathology. 89 (3): 353-360.

Raju S, Jayalakshmi SK, Sreeramulu K (2008). Comparative study on the induction of defense related enzymes in two different cultivars of *chickpea* (*Cicer arietinum L*) genotypes by salicylic acid, spermine and *Fusarium oxysporum f. sp. cicero*. Aust. J. Crop Sci. 2(3):121-140.

Reverchon S, Expert D, Robert-Baudouy J, Nasser W (1997). The cyclic AMP receptor protein is the main activator of pectinolysis genes in *Erwinia chrysanthemi*. J. Bacteriol. 179(11): 3500–3508.

Ridout CJ (2009). Profiles in Pathogenesis and Mutualism: *Powdery Mildews*. The Mycota.5 (1): 51-68.

Samuels AL, Giddings TH and Staehelin LA (1995). Cytokinesis in tobacco BY-2 and root tip cells: A new model of cell plate formation in higher plants. J. Cell Biol. 130:1345–1357.
seedlings in response to *Sclerospora graminicola infection*. Plant Science. 167: 1227-1234.

Shailasree S, Kini KR, Deepak S, Kumudini BS, Shetty HS (2004). Accumulation of hydroxypro- line-rich glycoproteins in pearl millet seedlings in response to *Sclerospora graminicola infection*. Plant Science. 167: 1227-1234.

Silva MC, Várzea V, Guerra-Guimarães L, Azinheira HG, Fernandez D, Petitot A-S, Bertrand B, Lashermes P, Nicole M (2006). Coffee resistance to the main diseases: leaf rust and coffee berry disease. Braz. J. Plant Physiol. 18(1):119-147.

Skamnioti P and Gur SJ (2007). *Magnaporthe grisea* cutinase2 mediates appressorium differentiation and host penetration and is required for full virulence. The Plant Cell. 19:2674-2689.

Sommer-Knudsen J, Bacic A and Clarke AE (1998). Hydroxyproline-rich plant glycoprotein. Phytochemistry 47: 483-497.

Stone BA, Clarke AE (1992). Chemistry and Biology of (1-3)-β-D-Glucans. Victoria, Australia: La Trobe University Press.

Templeton MD, Dixon RA, Lamb CJ, Lawton MA (1990). Hydroxyproline-rich glycoprotein transcripts exhibit different spatial patterns of accumulation in compatible and incompatible interactions between *Phaseolus vulgaris* and *Colletotrichum lindemuthianum*. Plant Physiol. 94:1265-1269.

Thulke OU, Conrath U (1998). Salicylic acid has a dual role in the activation of defence-related genes in *parsley*. Plant J. 14:35-42.

Toppan A, Roby D, Esquerré-Tugayé MT (1982). Cell surfaces in plant-microorganism interactions. In vivo effect of ethylene on hydroxyproline-rich glycoprotein accumulation in the cell wall of diseased plants. Plant Physiol. 70:82-86.

Torres MA, Jones JDG, Dangl JL (2006). Reactive oxygen species signaling in response to pathogens. Plant Physiol. 141(2):373-378.

Tsujimoto Y, Shimizu S (2005). Another way to die: autophagic programmed cell death. Cell Death and Differentiation. 12:1528–1534

Tuncel G, Nergiz C (1993). Antimicrobial effect of some olive phenols in a laboratory medium. Letts. Appl. Microbiol. 17:300-302.

Underwood W, Somerville SC (2008). Focal accumulation of defences at sites of fungal pathogen attack. J. Exp. Bot. 59 (13):3501–3508.

Vanacker H, Carver TLW, Foyer CH (2000). Early H_2O_2 Accumulation in mesophyll cells leads to induction of glutathione during the hyper-sensitive response in the *barley-powdery mildew* interaction. Plant Physiol. 123:1289–1300.

Vance CP, Kirk TK, Sherwood RT (1980). Lignification as a mechanism of disease resistance. Ann. Review Phytopath. 18:259-88.

Wei YD, DeNeergaard E, Thordal-Christensen H, Collinge DB, Smedegaard-Petersen V (1994). Accumulation of a putative guanidine compound in relation to other early defence reactions in epidermal cells of barley and wheat exhibiting resistance to *Erysiphe graminis f.sp. hordei*. Physiol. Mol. Plant Path. 45:469-484.

Willats WGT, McCartney L, Mackie W, Knox JP (2001). Pectin: cell biology and prospects for functional analysis. Plant Mol. Biol. 47:9–27.

Wojtaszek P (1997). Oxidative burst: an early plant response to pathogen infection. Biochem. J. 322:681-692.

Woloshuk CP, Soliday CL, Kolattukudy PE (1986). Mechanism by which contact with plant cuticle triggers cutinase gene expression in the spores of Fusarium solanif. sp. pisi. Proc. Natl. Acad. USA. 83: 1704–1708.

Zeier J, Delledonne M, Mishina T, Severi E, Sonoda M, Lamb C (2004). Genetic Elucidation of Nitric Oxide Signaling in Incompatible Plant-Pathogen Interactions[w]. Plant Physiol. 136:2875–2886.

Zeyen RJ, Carver TLW and Lyngkjaer MF (2002). Epidermal cell papillae. In: Belanger RR, Bushnell WR, Dik AJ, Carver, editors. The *powdery mildews*: a comprehensive treatise. St. Paul, MN: ASP Press. pp107-125.

Zhang J, Zhou JM (2010). Plant immunity triggered by microbial molecular signatures. Mol. Plant. 3(5):783-793.