*Int.J.Curr.Microbiol.App.Sci* (2020) *9*(11): 1650-1666

*International Journal of Current Microbiology and Applied Sciences*
***ISSN: 2319-7706*** **Volume 9 Number 11 (2020)**
Journal homepage: http://www.ijcmas.com

**Review Article** https://doi.org/10.20546/ijcmas.2020.911.197

# Exploring Physiological and Biochemical Factors Governing Plant Pathogen Interaction: A Review

**Rohit Chhabra[1*], Simardeep Kaur[2], Lavanya Vij[1] and Kompal Gaur[1]**

*[1]Department of Botany, Punjab Agricultural University, Ludhiana, Punjab, India*
*[2]Division of Biochemistry, ICAR- Indian Agricultural Research Institute, New Delhi, India*

**Corresponding author*

**Keywords**

Antioxidants, Enzymes, Plant growth regulators, Reactive oxygen species, Secondary metabolites

**Article Info**

***Accepted:***
12 October 2020
***Available Online:***
10 November 2020

**A B S T R A C T**

Pathogen attack generates the stress full conditions which disrupts the host metabolic machinery causing an oxidative burst in plants and leads to the formation of reactive oxygen species (ROS). These molecules are responsible for all the metabolic changes occurring in host plant and alter the DNA, carbohydrates, lipids, sugar and protein content. In this review, we have discussed the various physiological and biochemical components that assist the plant in imparting resistance and along with the role of phytohormones viz., jasmonic acid, salicylic acid and abscisic acid during plant pathogen interaction. We have also emphasized on production of ROS by stressed plants and involvement of plant growth regulators, antioxidative and non-oxidative components, sugars, pathogenesis-related proteins, secondary metabolites and lignins in providing resistance to the plants against the damaging effects of pathogens. Thus, we can conclude that this review provides the critical information on understanding various physiological and biochemical factors that assist the plants in adapting under stressed conditions.

## Introduction

Plant pathogen forms an intimate relationship with the plants to gain access to the host resources required for its growth, reproduction and survival. The process of infection, colonization and pathogen reproduction is referred to as pathogenesis. Disease outcome is determined by three-way interaction of the pathogen, plant and the environment, an interaction known as disease triangle (Figure 1). However, to avoid disease infestation, the plant must either recognize the presence of potential pathogens or mount a defence response to prevent pathogen attack without it being detrimental to its own physiology. The genetic constitution and the environmental conditions under which the genes operate govern the ability of a plant to defend itself against pathogen. Preformed plant structures and compounds that resist pathogen entry are cell walls, cuticle/surface, antimicrobial proteins & chemicals, enzyme inhibitors and detoxifying enzymes that breakdown pathogen derived toxins. Such attributes of host plant lower the chances of disease infection. The pathogen invades the host and encounters the pre-existing physical

and chemical barriers, which are the hereditary characteristics of the host plant and if the pathogen manages to overcome the barriers establishing parasitic relationship (Sharma and Gupta, 2020).

For those microbes that pass the preformed structures and barriers, the host system reacts to the foreign material which results in the formation of new physical and chemical barriers. These two conditions can be stated as resistance to penetration and disease development. Various integrated and complex mechanisms of the host plant are involved in the prevention of infection. A large number of physiological, biochemical and molecular changes have been observed that can be correlated with the onset of pathogen attack (Bhuiyan *et al.,* 2009).

These mechanisms may be local, constitutive or inducible. In some cases, the extents of these mechanisms which affect disease progression are poorly understood even with the considerable progress already made in the past few years to understand the mechanism of disease resistance or susceptibility. Understanding the physiological and biochemical variations will give us an idea about the potential development of the disease. When pathogens start infecting host plants, then they are capable of perceiving the loss of metabolic machinery and subsequently activate the defence signalling pathways (Bohlmann and Sobczak, 2014).

## Effects of plant pathogen interaction on formation of ROS due to oxidative stress

A common consequence of stressful conditions is the production of ROS *viz*. superoxide anion radical ($O_2^{\bullet-}$), singlet oxygen ($^1O_2$), hydrogen peroxide ($H_2O_2$), and hydroxyl radical ($^{\bullet}OH$) that could cause extreme oxidative damage to plant tissues (Mittler, 2017). The production of ROS is the unavoidable event of aerobic life. In higher plants, as an example, lower levels of ROS have been found to regulate differentiation, redox homeostasis, stress signaling, interactions with other organisms, systemic responses, and cell death. However, high levels have the ability to harm cellular components *via* lipid peroxidation, membrane destruction and protein damage (Das and Roychoudhary, 2014). Thus, these are very deadly and substantially affect normal cellular functioning (Asthir *et al.,* 2010). In this regard, plants perceive stress through their roots and send signals to alter their metabolism for the activation/synthesis of defensive genes in some plant parts (Gill *et al.,* 2019).

ROS are constantly produced at a much higher level in stressful conditions than under normal conditions (Moreau *et al.,* 2010) thus, have high tendencies to react with host DNA, structural lipids and proteins, resulting into severe cellular damages. They can also coexist utilizing ROS as potent cues to bring about hormonal signaling, protein homeostasis and plant immune responses (Hakmaoui *et al.,* 2012). At the time of infection, ROS generated by host is bound to affect both the host and the attacking pathogen. Thus, all the attacking pathogens are exposed to ROS released in plants. In order to mitigate exposure and oxidative stress, they use various small molecules such as glutathione, flavonoids, ascorbic acids and carotenoids for scavenging ROS via their oxidation by ROS along with detoxifying enzymes such as superoxide dismutase, catalase, peroxiredoxins and peroxidases. ROS are vital signaling regulators as:-

Effectively control ROS concentration by scavenging them at cellular levels

Dynamic intracellular control of accumulated ROS in subcellular organelles

Signals budding from ROS signaling are fast to transmit to all cells

Capable of interacting and modifying various targets (Mittler *et al.,* 2011)

## Effects of plant pathogen interaction on sugar metabolism

Sugars are precursors for the synthesis of various secondary metabolites viz., phytoalexins, phenols, lignin and callose. Sugar molecules play (Vargas *et al.,* 2012) a significant role in providing resistance against attacking pathogen in plants. In general, infection by any attacking agent leads to a series of changes in the vital plant processes like photosynthetic and respiratory pathway. The pathogen may disturb the photosynthetic capacity of plants either by physical injury to the photosynthetic apparatus or by disrupting the metabolic pathway responsible (Murria *et al.,* 2018c) changes in sugar content of plants. The joint level of soluble sugars in plants is known to be altered and modified by pathogenic attack. The level of sugars is reduced by their consumption for energy, structural purposes and uptake by the pathogen. Sugars losses can be compensated by transformation of infection site into a sink. The formation of sink at infection site does not always meet the sugar requirement. At the infection sites, sugars are generally taken by the attacking agent, while the attacked plant tissues still have high sugar requirements for initiation (Morkunas and Ratajczak, 2014) of defence responses viz., phenylpropanoids or phenol synthesis, synthesis of pathogenesis related proteins (PR). The intensity of respiration processes is known to increase around infection site. Vargas *et al.,* (2012) found an increased expression of respiration related genes at infection sites during a study conducted on maize leaves inoculated with *Colletotrichum graminicola*.

The anthracnose affected leaves of susceptible grape genotypes contained higher content of total sugars as compared to the resistant ones as stated by Gurjar *et al.,* (2015). Sugar levels were reported significantly higher in healthy leaves under diseased cotton genotypes than those in resistant genotypes (Chakrabarthy *et al.,* 2002).

Mannitol is an alcohol formed by reduction of keto or aldose group sugar. It plays a significant role in imparting disease resistance amidst biotic and abiotic stress in the form of mannitol dehydrogease (MTD), a catabolic enzyme. Mannitol acts both as a buffer and osmoprotectant during oxidative stress. It further protects the plant by quenching deadly ROS molecules. One of the popular mechanisms proposed the role of mannitol as an osmoprotectant. It acts as an antioxidant and according to the other; it acts as a "compatible" solute. During stress conditions, certain organisms accumulate specific compounds such as sugars, amino acids, polyols and derivatives of amino acids to high concentrations without affecting the functioning of cell's normal physiological processing. That is why these solutes are called compatible solutes and they also show osmoprotectant properties because they interact with cellular structures and hydration shell around proteins with the purpose of protecting and stabilizing them with under low osmotic potential conditions (Yancey and Siebenaller, 2015). According to another hypothesis, mannitol acts as an antioxidant in order to provide resistance against osmotic stress. An ideology states that mannitol mitigates the destructive effects of reactive oxygen species (ROS) formed at the time of water stress by scavenging those (Bernstein *et al.,* 2010). Protection of an indicator protein from ROS damage quenches 60 % of hydroxyl radicals ($OH^-$) by 33mM mannitol (Smirnoff and Cumbes, 1989).

## Effects of plant pathogen interaction on specific pathogenesis-related (pr) proteins

According to Goodman *et al.,* (1967), the plants are considered to be susceptible if they contain specific protein molecules as a substrate being required by the invading pathogen. Hence, a variety is considered resistant if it possesses lesser proportion of such proteins and thus maintain their resistance even under unfavourable conditions.. On the other hand, the varieties possessing such proteins in abundance will fail to prove resistant even under favourable conditions (Sharma *et al.,* 1992).

Khan *et al.,* (2001) studied the biochemical changes post infection with *Drechslera sorghicola,* causing leaf spot disease in sorghum. A significant increase in total soluble proteins was observed with the progress of disease in the infected sorghum leaves. Changes in proteins occur when pathogen penetrates the host cells which disturb protein and related metabolism.

Malhotra (1993) recorded significant increase in protein content in tomato plants resistant to *Fusarium* wilt, while it decreased significantly in susceptible cultivars. Similarly, Arun *et al.,* (2010) compared the protein content of healthy and infected tissues of pearl millet against *Sclerospora graminicola* infection and reported that the protein content was higher in the healthy tissues of the susceptible variety as compared to the resistant one. Following infection, there was increase in the protein content in resistant as well as susceptible cultivars, but the increase was higher in resistant cultivars.

When plant succeeds in resisting the entry of pathogen during infection, it is because the interaction between host plant and pathogen turns out to be incompatible. Following this, an integrated and coordinated set of metabolic events make the host plant capable of defending against various other infections. It also includes the synthesis and induction of various novel proteins called " pathogenesis-related proteins" (PRs). These are defined as "proteins encoded by the host plant but induced only in pathological or related situations," the latter implying to non-pathogenic origin situations (Dani *et al.,* 2005). PRs have been identified because they were easily identified in the infected plants, however, their anti-pathogenic response was difficult to spot. Their general shielding role against biotic stress conditions has been reported in many plant families.

The PR proteins are divided initially into five classes on the basis of molecular mass, biological activity, localization and isoelectric point

Group 1 (PR-I tobacco proteins of 16 KD)
Group 2 (β-1, 3-glucanases)
Group 3 (Chitinases)
Group 4 of low molecular mass (13 and 14.5 KD)
Group 5 which includes 5A (osmotins of 24 kD) and 5B protein of unknown characteristics (approx. forty fivekD).

Currently PR-proteins were labeled into 17 families like β-1, 3-glucanases, chitinases, thaumatin-like proteins, peroxidases, ribosome-inactivating proteins, thionins, non-specific lipid switch proteins, oxalate oxidase, and oxalate oxidase like proteins (Liu *et al.,* 2010).

Among these PR proteins, chitinases and β-1, 3-glucanases are most important hydrolytic enzymes which are produced in many plant species after infection by various forms of pathogens.

Concentration of these PR proteins considerably increases and plays the key role

in protection against fungal pathogens by damaging cell wall, as chitin and β-1,3-glucan both are the vital components of the cell walls of many disease causing fungi (Santen *et al.*, 2005). These proteins are stabilized through disulfide linkages and are resistant towards proteolysis and increased temperatures, whereas most of other plant proteins are denatured by such conditions (Gorjanovic, 2009). Chitinases catalyze bond cleavage between C1 and C4 of consecutive NAG monomers of chitin.

Plant chitinases are usually endochitinases able to degrade chitin (Suarez *et al.*, 2001). Extracellular chitinases quickly block the spreading of the hyphae which are invading internal areas and probably cause release of fungal elicitors, which further induce the synthesis of various other chitinases inside the host (Stangarlin and Pascholati, 2000).

Plant β-1, 3-glucanases belonging to the PR-2 family of pathogenesis-related proteins play an important role in plant defence responses to pathogen infection (Ji *et al.*, 2000). The enzyme holds the potential to catalyze the cleaving of the glycosidic bonds in β-1,3-glucan (Anguelova *et al.*, 2001). Additionally, they also have other functions like cellular elongation, fruit ripening, fertilization, somatic embryogenesis, germination of seed and pollen and flowering (Ebrahim *et al.*, 2011).

β-1,3-glucanases have both direct and indirect effects in protecting the plant against fungi.

In direct effects they cause hydrolysis as well as lysis of cell wall of fungus

They also promote oligosaccharide elicitor formation indirectly which further favours the formation of PR proteins or other antifungal bioactive compounds, like phytoalexins.

## Effects of plant pathogen interaction on photosynthetic pigments

Photosynthesis is the ultimate source of energy for all living cells, plants or animals directly or indirectly. Deformed chloroplasts are the common features of infected plant tissues (Murria *et al.*, 2018b). The pathogen affects the photosynthesis by the destruction of the chlorophylls and decreases the efficiency of photosynthetic process per mole chlorophyll. The composition of leaf pigments is sensitive to plant stress, and various biotic and abiotic factors are responsible for loss of photosynthetic pigments like chlorophylls or for the production of photo-protective pigments such as β- carotene or zeaxanthin. Decrease in chlorophyll a, chlorophyll b and total chlorophyll in the diseased leaves and susceptible genotypes was reported than in resistant green gram genotypes (Kulkarni, 2009). Atwal *et al.*, (2004) also reported decrease in chlorophyll content of leaves of *Brassica juncea* in relation to *Alternaria* blight disease. It has been observed in the study undertaken by Chhabra *et al.*, (2019) that there was a reduction in total chlorophyll, chlorophyll a, chlorophyll b and carotenoid content in infected seedlings of both aromatic and non-aromatic rice cultivars against Bakanae disease caused by *Fusarium fujikuroi*.

Carotenoids are localized lipophilic antioxidants in plant tissues, capable of absorbing light within 450-570 nm wavelength range. They are found in both micro-organisms and plants. They consist of antennae molecules which absorb and transfers the light energy harvested to chlorophyll molecule. Carotenoids also protect the plant photosynthetic machinery by damage caused by ROS. They manifest antioxidants by fostering the photosynthetic machinery in four ways:

Reacting with LPO products to terminate the chain reactions

Generating heat as byproduct and scavenging $^1O_2$.

Preventing the formation of $^1O_2$ by reacting with 3Chl∗ and excited chlorophyll (Chl∗)

Dissipating the excess excitation energy, *via* the xanthophylls cycle (Das and Roychoudhary, 2014).

## Effects of plant pathogen interaction on secondary metabolites

Seconday metabolites include amino acids, plant pigments, phenols, coumarins, flavonoids, alkaloids, essential and volatile oils, enzyme prosthetic groups, lignin and other complex derivatives. They are not directly involved in growth and development, metabolic and physiological processes. They form an essential part of the plant defense system against pathogenic attacks and environmental stresses.

## Phenolics

Phenolics are the most common class of secondary metabolites in plants synthesised from the aromatic amino acids tyrosine, phenylalanine through the phenylpropanoid pathway. They assist in the defence response of plants under biotic and abiotic stress conditions. There toxic nature is attributed by the researchers to the disruption of electron transport system. These compounds play significant role in maintaining cell wall integrity and imparting protection against pathogen attack (Ushimaru *et al.,* 2006).

Accumulation of higher levels of phenols and its precursors is observed in diseased plant tissues. However, phenol accumulation is higher in incompatible host pathogen complex than in the compatible ones. Chhabra *et al.,* (2019) also reported a positive correlation between polyphenols and the degree of disease resistance against *Fusarium*.It is generally assumed that polyphenol oxidase (PPO) play role in défense responses to biotic stresses. Murria *et al.,* (2018a) studied metabolic changes in grapevine leaves during anthracnose infection and reported that the activity of phenol oxidase enzyme is generally higher in infected tissues of resistant varieties than in the infected tissue of susceptible genotype. The disease resistance in various host-parasite combinations were correlated by enhanced phenol synthesis and polyphenol activity. The enzyme PPO catalyses the oxidation of phenols into reactive quinones, however, their role in intact plant cell is not understood. They may act through cross linking of quinones with other phenolics, direct toxicity of quinones, and production of ROS. Prasath and Ponnuswami (2008) carried out biochemical analysis of chilli genotypes against *Colletotrichum annum* and found highest phenolic content in resistant genotype (Acc. 16 PCB 81) followed by moderately resistant genotypes and the lowest phenol content was reported in susceptible genotype like Hybrid 6. Kulkarni (2009) reported that healthy tissues of resistant and moderately resistant green gram genotypes contained higher amount of total phenols than susceptible one. Analysis of the bio-physiological values of *Capsicum annum* L. germplasm against diease in *Colletotrichum annum* reported high total phenolic content in resistant genotypes Jaun, Breek-1 and Breek-2 (Kaur *et al.,* 2011). Increase in total phenols upon infection in resistant genotypes than in susceptible ones indicating the significant influence of total phenols in disease resistance in plants.

## Flavonoids

Flavonoids are extensively occurring plant pigments which are commonly found in the

leaves, pollen grains and floral organs. Their structural classification narrows down into four classes: flavonols, flavons, isoflavons and anthocyanins. Various functions of flavonoids include fruits, seeds and flower pigmentation, defence against plant pathogens and pollen germination. Flavonoids possess high excitation energies and therefore are known for scavenging ROS produced due to damaged photosynthetic apparatus (Fini *et al.*, 2011). Additionally, flavanoids also play a major role in scavenging singlet oxygen ($^1O_2$) and alleviate the degree of damage at the site of the outermost layer of the chloroplastic membrane (Agati *et al.*, 2012).

## Lignins

Lignin is a form of complex polymer derivative, chiefly found in the form of deposition of secondary thickened cell wall with the purpose of adding imperviousness and strength to the wall. Hatfield *et al.*, (2009) reported that lignin is composed of guaiacyl and syringyl polymer units, which are made from monolignols coniferyl and sinapyl alcohol respectively. Role of lignins in response to pathogen infection in various plant species is described in Table 1.

## Effects of plant pathogen interaction on antioxidant machinery

Any kind of stress causes a number of variations in plant metabolism due to ion toxicity, production of reactive oxygen species (ROS), etc. These ROS are reactive chemical species with an unpaired electron in the outer orbit and are derived from oxygen. These are responsible for damaging biological systems and causing cell damage which eventually leads to cell death. The normal cell maintains equilibrium (Torres *et al.*, 2006) between the formation and destruction of free radicals. Any imbalance facilitates the cell to enter a mode called "oxidative stress". The generation of ROS is scavenged by antioxidant system with antioxidant compounds such as tocopherols, ascorbate, salicylate, glutathione, etc. and antioxidant enzymes like peroxidase, superoxide dismutase, catalase etc (Saleh and Pleith, 2009). High activities of antioxidant enzymes and high content of non-enzymatic constituents are important in biotic stress tolerance.

## Enzymatic antioxidants

Enzymes play a vital role in defending the host from infestation in plant pathogen interaction. The role of several oxidative enzymes and their metabolic products in defence mechanism in plants has been identified as presented in Table 2. In the presence of oxygen, polyphenol oxidase oxidizes the phenolic compounds that are in the form of O-diphenol to O-quinone. The presence of stress leads to the generation of ROS which upon reaction with the host DNA, lipids and protein causes cellular oxidative damage, disrupting the normal cell functioning (Wu *et al.*, 2004). The antioxidant enzymes such as catalase (CAT), superoxide dismutase (SOD), peroxidase (POX), ascorbate peroxidase (APX) and glutathione reductase (GR) function in detoxification of free radicals which causes membrane damage by acting as potential scavengers (Alscher *et al.*, 2002).

Peroxidase may act as either hydrogen peroxide scavenger or generator depending upon physiological conditions (Almagro *et al.*, 2009) ant this enzyme plays crucial role in resistance against pathogen. An enzymatic antioxidant called Ascorbate peroxidase (APX) also plays a protective role against stressful conditions and their activity is up-regulated during wounding or pathogen invasion (Agrawal *et al.*, 2003). Khatun *et al.*, (2009) studied the role of antioxidative

enzymes in defence against black spot in *Rosa centifolia* and reported an increase in POX, PPO and SOD activity following disease inoculation in susceptible cultivars. Superoxide dismutase is a powerful ubiquitous plant enzyme causes polymerisation of cinnamyl alcohols to lignin that leads to disease resistance in host plants upon its higher activity. Shankar and Jindal (2001) suggested highest peroxidase activity in grape genotype MA x RR 76-3, whereas polyphenol oxidase activity was highest in H-144 both of which fall under resistant category. The lowest enzymatic activity was observed in the most susceptible genotype and higher enzymatic activity was reported in infected leaves than in healthy leaves. Furthermore, higher POX and SOD activity was reported upon the onset of *Alternaria* blight (Saharan *et al.,* 2000) in both susceptible and resistant genotypes.

## Non-enzymatic antioxidants

This deteriorating effect of ROS in the plants is also minimized by non-enzymatic detoxification system composed of tocopherols, reduced glutathione, ascorbic acid, and proline which protects the cells from oxidative damage. In a generalized manner, an antioxidant in the cell may work at two different levels: a) prevention, by maintaining the formation of ROS to a threshold level b) interception, by scavenging ROS. Hegde and Anahosur (2001) reported higher vitamin E content in chilli genotypes resistant to *Colletotrichum capsici* causing fruit rot as compared to the susceptible ones. Similarly, Arora and Kaur (2004) also observed that the chilli genotypes resistant to *Alternaria solani* had higher ascorbic acid and carotenoid content as compared to the susceptible ones. The content gradually decreased after infection and this decrease was significantly higher in susceptible genotypes.

## Tocopherols

Tocopherols reside within the family of hydrophobic lipid-loving antioxidants serving a major function of ROS and stray lipid radical scavenging, thus making them essential and indespensible bioactive molecules within the membranes (Hollander-Czytko *et al.*, 2005; Kiffin *et al.,* 2006). Amongst all four tocopherol isomers (α-, β-, γ-, δ-), α-tocopherol posseses the paramount extent of antioxidant properties. These are synthesized in green tissues of photosynthetic plants and organisms. Its synthesis takes place form γ- tocopherol-methyl-transferase (γ-TMT encoded by *VTE4*). Tocopherols protect PSII, membrane lipids and other chloroplast membrane constituents maintain their structural and functional aspects by reacting and scavenging $O_2$ accompanied with absorption of excessive excitation energy. It also slows down the chain propagation step of LPO cycle by acting as a free radical trap (Kiffin *et al.,* 2006).

## Ascorbic acid

Ascorbic Acid (AA) is one of the most abundant and extensively studied antioxidant forming the first line of defense in the case of pathogen attack. It has the tendency to donate electrons to a wide array of enzymatic and non- enzymatic reaction intermediates. Smirnoff-Wheeler pathway in mitochondria generates most of the AA with the help of enzymeL-galactano-γ-lactone dehydrogenase. D-galacturonic acid acts as a precursor, synthesising remaining plant AA. Ascorbic acid pool is found both in cytosol and apoplast with the potential of fighting high ROS levels (Barnes *et al.,* 2002). Oxidation of AA takes place in two steps. In the first step, oxidation of MDHA takes place. Its immediate reduction to ascorbate is necessary to prevent its disproportionation into DHA and AA. It also regenerates cellular α-

tocopherol levels on reaction with $O^{\bullet-}_2$, $H_2O_2$ and $OH^{\bullet}$ from tocopheroxyl radical (Shao *et al.,* 2005). Activities of metal binding enzymes are also preserved and protected by AA. It acts as a cofactor of violaxanthine de-epoxidase in its reduced state and helps in dissipating the excess excitation energy off (Smirnoff, 2000). It has also been reported that ascorbic acid prevents PSII from photo-oxidation and aids for the same in its down regulation by pH mediated modulation, associated with zeaxanthine formation.

## Reduced glutathione

Glutathione is an abundant thiol tripeptide (γ-glutamyl-cysteinyl-glycine), found in all cellular structures like vacuoles, endoplasmic reticulum, mitochondria, cytosol, peroxisomes, chloroplasts and apoplast. Glutathione is a significant factor responsible for a wide range of processes like cell division, cell differentiation, senescence, detoxification of xenoiotics, regulation of sulfate transport,, conjugation of metabolites, synthesis of nucleotides and proteins, regulation of enzymatic activity, stress responsive gene expression and synthesis of phytochelatins (Mullineaux and Rausch, 2005). High reduction poteintial of GSH is responsible for such versatility in its roles.It derives its reducing power from the central nucleophilic cysteine residue. It is efficient in scavenging ROS molecules like OH•, $H_2O_2$, $^1O_2$, and $O^{\bullet-}_2$ generating GSSG as a by-product and prevent adducts (glutathiolated) formation from various biomolecules. Ascorbic acid is also used in replenishing the GSSG yield levels in the cell. A balance is necessary between the levels of GSH and GSSG in the cell to maintain its redox state. The metabolic processes of GSSG and GSH are interlinked and thus can be produced by either enzyme action or by de novo GR pathway therefore, maintain the cellular GSH pool. Roychoudhury *et al.,* (2012a) reported that GSH is also essential in the phytochelatin formation via the activity of enzyme phytochelatin synthase. It also aids in chelating heavy metal ions and quenching deadly ROS molecules formed in plants (Roy Choudhury *et al.,* 2012b).

## Proline

Proline is a powerful osmolyte and non-enzymatic antioxidant. It plays a vital role in quenching different deadly ROS members especially hydroxyl ($^{\bullet}OH$) and superoxide radical ($O_2^{\bullet-}$). It has an inhibitory effect on the lipid proxidation. Proline is synthesized from the precursor and substrate glutamic acid following the pathway with pyrroline 5-carboxylate (P5C) intermediate. This pathway is catalyzed by two enzymes in plants namely Pyrroline-5carboxylate reductase (P5CR) and $\delta^1$-pyrroline-5-carboxylate synthetase (P5CS).

Verbruggen and Hermans (2008) have reported the presence of proline in higher levels under different stress conditions. This could be due to reduced degradation or enhanced synthesis.Proline accumulation occurs in response to stressful conditions (Murria *et al.,* 2018a). Goicoechea *et al.,* (2000) reported modification in the concentration of proline in foliar tissues of pepper against *Verticillium dahliae* infection. There was an increase in proline concentration in foliar tissues of infected plants, while it did not change in the leaves of control plants. Sivritepe *et al.,* (2009) observed the increased proline accumulation in response to downy mildew in barley. Mite infestation caused an increase in proline contents by 6.7 and 4.2-fold in the leaves of Muscule and Sultana grapevines respectively. Naglaa and Heba (2011) reported significant increase in proline content in infected leaves of flax genotypes as compared with either resistant or susceptible genotypes in reaction to powdery mildew.

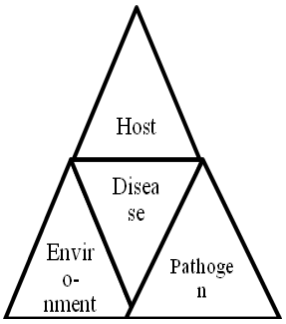
**Table.1** Role of lignins in response to pathogen infection in various plant species

| Sr. No. | Plant species | Activities and Modulations |
|---|---|---|
| **1.** | Tomato | Higher lignin content was markedly reported in *R. solanacearum* resistant genotypes (Mandal *et al.*, 2011) than in susceptible genotypes. Accumulation of higher levels of lignin were reported in the infected roots (Mandal *et al.*, 2013) |
| **2.** | Cotton | Increased stem lignification in response to infection by wilt fungus *V. dahliae*, providing resistance against wilt (Xu *et al.*, 2011) |
| **3.** | Alfalfa | Downregulation of *HCT* gene in *Colletotrichum trifolii* causes lower lignin content, leading to reduced pathogen defense responses leads to plants and fungal infection tolerance (Gallego-Giraldo *et al.*, 2011). Activation of defense responses was reportedly triggered by the bioactive molecules in secondary cell wall fragments. |
| **4.** | Melon | Accumulation of lignin was found to escalate upon infection by powdery mildew fungus *Podosphaera fusca* in resistant genotypes than the susceptible ones (Romero *et al.*, 2008). |

**Table.2** Oxidative enzymes and their metabolic products in defence mechanism in plants

| Enzymatic antioxidant | Function |
|---|---|
| **Superoxide dismutase (SOD)** | Omnipresent metalloenzyme that scavenges and dismutes $O_2^-$ into $H_2O_2$ and $O_2$ |
| **Catalase (CAT)** | Dismutates $H_2O_2$ into $O_2$ and $H_2O$ (in peroxisome) |
| **Ascorbate peroxidase (APX)** | Dismutates $H_2O_2$ into $O_2$ and $H_2O$ (in chloroplast and cytosol) |
| **Monodehydroascorbate reductase (MDHAR)** | Uses reducing power NADPH to regenerate AA from MDHA (in peroxisome) |
| **Dehydroascorbatereductase (DHAR)** | Uses reduced gluthione (GSH) to reduce dehydroascorbate (DHA) to AA |
| **Glutathione reductase (GR)** | Reduction of GSSG to GSH |
| **Guaiacol peroxidase (GPX)** | Synthesis of ligin, scavenges $H_2O_2$ and degrades IAA |

**Fig.1** The disease triangle illustrating the interaction between pathogen, host and the environment as a pre-requisite to occur



## Malondialdehyde

Malondialdehyde is a reactive aldehyde and is one of the many reactive species that causes toxic stress in cells. The production of this organic compound is used as a biomarker to measure the level of oxidative stress in an organism. Munne-Bosch and Alegre (2002) demonstrated that chlorosis can be a consequence of lipid peroxidation with an increase in malondialdehyde and subsequent reduction in the antioxidant defences in chloroplast, which leads to decreased chlorophyll concentration and photosynthetic activity. Atwal *et al.*, (2004) reported that MDA at stage I and II in both healthy and diseased genotypes of *Brassica oleracea* L. against *Alternaria* blight increased significantly in all the genotypes, the increase being maximum in highly susceptible genotypes.

## Role of plant growth regulators (PGRs) during plant pathogen interactions

## Salicylic acid

The naturally occurring salicylic acid in rice plants is synthesized from Cinnamic acid via benzoic acid. In plants, exogenous application of SA or its derivatives affects diverse plant processes such as seed germination, thermogenesis, cell growth, stomatal responses, seedling establishment, nodulation, respiration and thermotolerance. Dempsely and Klessling (1995) reported that salicylic acid plays a predominant role in inducing signal for disease tolerance and development of systemic acquired resistance in crop plants. Elevation in endogenous SA on pathogen attack is found in many plant pathogen interactions and this elevation is linked to the switching on the defence mechanisms. The signalling system of SA plays dual role by activating local resistance and systemic acquired resistance (SAR) which is observed in systemic tissues. Plant infected by local infection, is observed to have SAR, a SA dependent elevated defence to wide variety of pathogens. The infection by necrotizing pathogen creates a unique physiological state called “priming” in plants. The priming results in a stronger and faster induction of defence mechanisms after invasion by pathogen. Silverman *et al.,* (1995) found that rice plants had the highest level of SA among all the cereal crops tested for SA content. Xiao *et al.,* (1996) observed that when rice seedlings were soaked in salicylic acid, the malondialdehyde content and permeability of plasma membranes dropped but phenylalanine ammonia lyase activity, total chlorophyll content increased. It was concluded that exogenous salicylic acid might play an active in increasing the resistance to this disease.

## Jasmonic acid

Jasmonic acid and methyl jasmonates are the naturally occurring plant growth regulators playing important roles in various metabolic processes in plants. Jasmonates (JA) are synthesized from oxidation of linolenic acid using the octadecanoic pathway. This pathway has been proposed as part of the signalling cascade that induces plant defence responses after several pathogen attacks and invasions. Berger *et al.,* (2007) reported that exogenous foliar spray of jasmonic acid induces several physiological responses in relation to biotic stress caused by pathogen. It has been reported that jasmonic acid induces the expression of plant-defence genes in response to pathogen invasion (Berger *et al.,* 2007). Exogenous foliar application of JA to *Arabidopsis* plants reduced disease development after infection by numerous pathogenic fungi. Jasmonic acid, Methyl jasmonate and the JA precursor, 1,2-oxophytodenoic acid (OPDA), all induce a range of wound-inducible genes, such as

proteinase inhibitors, which have antifungal activity against many pathogenic fungi (Penninckx *et al.,* 1998).

## Abscisic acid

Abscisic acid (ABA) has been reported as a major regulator of biotic defence responses but its role is less straightforward than other defence related plant growth regulators. It may provide resistance in some host pathogen interactions whereas, in some cases, it may increase susceptibility also. ABA dependent stomatal closure is a pre-invasive defence barrier. Early ABA responsive genes reduce lipid oxidation, membrane cycling and vesicle trafficking (Hückelhoven, 2007). Overlapping between biotic and abiotic stress tolerance has been reported when ABA is observed to play crucial role in response to pathogen. Various foliar fungal pathogens are observed to disturb movement of stomata. Several researchers reported that stomata not only provide a path for transpiration, but also a port for pathogen entry. Under stress conditions, ABA acts as a chemical signal initiating stomatal closure through opening and closing of ion channels by phosphatases and protein kinases. Abscisic acid mediated closure of stomata blocks pathogen entry into the apoplastic space, therefore, ABA plays a positive regulator of defence responses. As the pathogen enters into the apoplastic space, ABA alters the pathogen response by interacting with defence hormones like salicylic acid, ethylene and jasmonic acid (Hückelhoven, 2007).

In conclusion and future perspectives, plant pathogens like bacteria, fungi, pests, bugs, insects, viruses and nematodes can potentially, directly or non-directly lead up to a loss of 20-40% global produce, worth 60 billion dollars annually. These issues caused by plant pathogens are a great threat in ensuring global food security with the ever-growing world populace. This review highlights the various effects of plants pathogen interaction on major plant bio-physiological aspects and the multiple strategies adopted by the plant in order to cope up with various stresses.

Stressful conditions can severely alter the plant growth, anatomy and physiological processes like photosynthesis, respiration, transpiration and translocation of water. High degree of stress causes an oxidative burst which further leads to a surge in the production of deadly reactive oxygen species (ROS) and damage to the DNA, carbohydrates, lipids and proteins. Pathogen attack can also damage the host metabolic machinery leading to shortage of preformed defense molecules imparting disease resistance. Various integrated and complex mechanisms of the host plant are involved in the prevention of infection. Various sugar, specific proteins, antioxidants and secondary metabolites aid in disease resistance in plants. Sugar alcohol like mannitol acts as an osmoprotectant and ROS molecules scavenger owing to the enzyme mannitol dehydrogease.

Specific proteins like chitinases, peroxidases, ribosome-inactivating proteins, β-1, 3-glucanases provide protection to cell and its organelles as they catalyzes the cleaving of the structural bonds of the pathogen cell wall and membranes. Plant growth regulators, antioxidative and non-oxidative components and various secondary metabolites like phenolics, alkaloids, sterols, lignins also play a significant role in providing the first line of defense against the damaging effects of pathogens. The antioxidant enzymes such as catalase (CAT), superoxide dismutase (SOD), peroxidase (POX), ascorbate peroxidase (APX) and glutathione reductase (GR) function in detoxification of free radicals which causes membrane damage by acting as

potential scavengers. Exogenous or endogenous application of plant hormones viz., jasmonic acid, salicylic acid and abscisic acid increases the resistance responses during plant pathogen interaction. Phytohormones acts as a regulator signaling a complex set of pre-invasive and defense responses, inducing a set of expression resisting pathogen invasion.

With all the critical information assessed and provided in this review, there still is a great dearth in our understanding of the mechanisms underlying the changes observed at molecular level. Nevertheless, more investigation at gene level is necessary to decipher the factors and the effect of those factors on the mechanisms in detail. Understanding the physiological and biochemical variations will give us an idea about the potential development of the disease and thus also provide time to come up with strategies of counter-attack.

Biotechnological approaches like use of molecular markers in various chromosomal regions should be applied to enlighten the areas responsible for resistant and hormone signaling in the resistant cultivars. More importance should be given to the studies concerning the topic as to have the preexisting knowledge on the potential development of the disease and in perceiving the loss of metabolic machinery and subsequently activating the defence signalling pathways.

## Acknowledgment

RC designed the framework of the paper. RC wrote the first draft of manuscript. SK and KG aided in the writing of the paper. LV helped in literature searches, editing and proofreading the edited draft of manuscript. All the authors have read and approved the final draft of the paper.

## References

Agati G, Azzarello E, Pollastri S, and Tattini M (2012) Flavonoids as antioxidants inplants: location and functional significance. *Plant Sci*. 196: 67–76.

Agrawal GK, Jwa NS, Han KS, Agrawal VP and Rakwal R (2003) Isolation of a novel rice PR4 type gene whose mRNA expression is modulated by blast pathogen attack and signalling components. *Plant Physiol Biochem* 41:81-90.

Almagro L, Gomez RLV, Belchi NS, Bru R, Ros BA and Pedreno MA. (2009) Class III peroxidases in plant defence reactions. *J Exp Bot* 60: 377-90.

Alscher RG, Erturk N, and Heath LS. (2002) Role of superoxide dismutases (SODs) in controlling oxidative stress in plants. *J Exp Bot* 53: 1331–1341.

Anguelova MV, Westhuizen VD and Pretorius ZA. (2001) Beta-1,3-glucanase and chitinase activities and the resistance response of wheat to leaf rust. *J Phytopathol* 149: 381-384.

Aragón W, Reina-Pinto JJ, Serrano M. (2017) The intimate talk between plants and microorganisms at the leaf surface. *J Exp Bot*. 68: 5339–5350.

Arora N and Kaur R. (2004) Biochemical factors influencing mechanism of resistance in chilli leaf during *Alternaria solani* infection. *Env Eco* 22: 853-855.

Arun K, Mali PC and Manga VK. (2010) Changes in some phenolic compounds and enzyme activities of infected pearlmillet caused by *Sclerospora graminicola*. *Int J Pl Physiol Biochem* 2: 6-10.

Asthir B, Koundal A, Bains NS and Mann SK. (2010) Stimulation of antioxidative enzymes and polyamines during stripe rust disease of wheat. *Biol Plant* 54: 329-333.

Atwal AK, Kaur R, Munshi SK and Mann APS. (2004) Biochemical changes in relation to Alternaria leaf blight in Indian mustard. *Pl Dis Res* 19: 57-59.

Bernstein, N. et al. (2010) Involvement of the plant antioxidative response in the differential growth sensitivity to salinity of leaves vs roots during cell development. Free Radic. Biol. Med. 49, 1161–1171

Bhuiyan NH, Selvaraj G, Wei Y, and King J. (2009) Role of lignification in plant defence. *Plant Signal Behav*4: 158–159.

Bohlmann, H., and Sobczak, M. (2014). The plant cell wall in the feeding sites of cyst nematodes. *Front. Plant Sci.* 5:89.

Chakrabarthy PK, Mikewar PM, Sheoraj and Sravankumar V. (2002) Biochemical factors governing resistance in diploid cotton against grey mildew. *Indian Phytopath*55: 140-146.

Chhabra R, Kaur N, Bala A. (2019) Physiological and biochemical alterations imposed by *Fusarium fujikuroi* infection in aromatic and non-aromatic rice cultivars. *Plant Physiol Reports* 24:563-575

Dani V, Simon WJ, Duranti M and Croy RD. (2005) Changes in the tobacco leaf apoplast proteome in response to salt stress. *Proteomics* 5: 737-745.

Das K and Roychoudhary A. (2014) Reactive oxygen species (ROS) and response of antioxidants as ROS-scavengers during environmental stress in plants. *Front Plant Sci*2: 1-13.

Dempsely DA and Klessling DF.( 1995) Signal in plant disease resistance. *Bulletin de L' institute Pasture*93: 167-186.

Ebrahim S, Usha K and Singh B. (2011) Pathogeneis related (PR) proteins in plant defence mechanism. *Sci Microb Pathol* 2: 1043-1054.

Fini, A, Brunetti C, DiFerdinando M., Ferrini F., and Tattini M. (2011) Stress-induced flavonoid biosynthesis and the antioxidant machinery of plants. *Plant Signal. Behav* 6: 709–711.

Gallego-Giraldo L, Jikumaru Y, Kamiya Y, Tang Y, and Dixon RA. (2011) Selective lignin down-regulation leads to constitutive defence response expression in alfalfa (*Medicago sativa* L.). *New Phytol* 190: 627–639.

Gill MB, Zeng F, Shabala L, Zhang G, Yu M, Demidchik V, Shabala S and Zhou M. (2019) Identification of QTL related to ROS formation under hypoxia and their association with waterlogging and salt tolerance in barley. *Int J Mol Sci* 20: 1-15.

Goicoechea N, Aguirreolea J, Cenoz S and Garc´ıa-Mina JM. (2000) *Verticillium dahliae* modifies the concentrations of proline, soluble sugars, starch, soluble protein and abscisic acid in pepper plants. *Eur J Pl Pathol* 106: 19-25.

Goodman RN, Kialy Z and Saitlin M. (1967) The biochemistry and physiology of infections plant disease. D. Van Nostrand Company Inc., p. 354.

Gorjanovic S. (2009) A review: Biological and technological functions of barley seed pathogenesis- related proteins (PRs). *J Inst Brew* 115: 334-60.

Gurjar PS, Singh SK, Singh AK and Verma MK. (2015) Field reaction and biochemical response of grape genotypes to anthracnose incidence under sub-tropical conditions. *Int J Sci Natu* 6:248-55.

Hakmaoui A, Pérez-Bueno ML, García-Fontana B, Camejo D and Jimenez A. (2012) Analysis of the antioxidant response of *Nicotiana benthamiana* to infection with two strains of pepper mild mottle virus. *J Exp Bot* 63: 5487–5496.

Hatfield R, Marita JM, Frost K, Grabber J,

Ralph J, Lu Fl. (2009) Grass lignin acylation: p-coumaroyl transferase activity and cell wall characteristics of C3 and C4 grasses. *Planta* 229: 1253–1267.

Hegde G M and Anahosur K H. (2001) Biochemical basis of resistance to fruit rot (*Colletotrichum capsici*) in chilli genotypes. *Karnataka J Agric Sci* 14:686-690.

Hückelhoven R. (2007). Transport and secretion in plant-microbe interactions. *Curr Opin Plant Biol*10: 573-579.

Ji C, Norton RA, Wicklow DT and Dowd PF. (2000) Isoform patterns of chitinase and β-1,3-glucanase in maturing corn kernels (*Zea mays* L.) associated with *Aspergillus flavus* milk stage induction. *J Agric Food Chem* 48: 507-511.

Kaur N, Dhiman JS and Khurana DS. (2011) Physiological and biochemical traits analysis of *Capsicum annum* L. germplasm for Resistance to *Colletotrichum capsici*. *J Cell Pl Sci* 2:12-21.

Khan AJ, Deadman ML,Srikandakumar A, A1-Maqbali YM, Rizvi SG and A1-Sabahi J (2001) Biochemical changes in sorghum leaves infected with leaf spot pathogen, *Drechslerasorghicola. Pl PatholJ* 17:342-346.

Khatun S, Bandhopadhyay PK and Chatterjee NC. (2009) Phenols with their oxidising enzymes in defence against black spot of rose (*Rosa centifolia*). *Asian J Exp Sci* 23: 249-252.

Kulkarni S. (2009) Sources of resistance to anthracnose in green gram and biochemical parameters for resistance. *Karnataka J Agric Sci* 22: 1123-1125.

Liu B, Xue X, Cui S, Zhang X, Han Q, Zhu L, Liang X, Wang X, Huang L, Chen X and Kang Z. (2010) Cloning and characterization of a wheat β-1,3-glucanase gene induced by the stripe rust pathogen *Puccinia striiformis* f. sp. tritici. *MolBiol Rep* 37: 1045-1052.

Malhotra SK. (1993) Biochemical components of tomato genotypes in relation to *Fusarium* wilt. *Indian J Mycol Pl Path* 23: 302-304.

Mandal S, Das RK and Mishra S. (2011) Differential occurrence of oxidative burst and antioxidative mechanism in compatible and incompatible interactions of tomato and *Ralstonia solanacearum*. *Plant PhysiolBiochem.*49: 117–123.

Mandal S, Kar I, Mukherjee AK and Acharya P. (2013) Elicitor-induced defence responses in *Solanum lycopersicum* against *Ralstonia solanacearum*. *Sci. World J.* 25:561056.

Mittler R, Vanderauwera S, Suzuki N, Miller G, Tognetti, VB, Vandepoele K, Gollery M, Shulaev V and Van Breusegem F. (2011). ROS signaling: the new wave? *Trends Plant Sci*. 16: 300–309.

Mittler R. (2017) ROS Are Good. *Trends Plant Sci*22: 11-19.

Moreau M, Lindermayr C, Durner J and Klessig DF. (2010). NO synthesis and signaling in plants-where do we stand? *Physiol Plant* 138: 372–383.

Morkunas I and Ratajczak L. (2014) The role of sugar signalling in plant defence responses against fungal pathogens. *Acta Physiol Plant.* 36:1607-1619.

Munne-Bosch S and Alegre L. (2002) Plant ageing increase oxidative stress in chloroplasts. *Planta*214: 608-615.

Murria S, Kaur N and Arora NK (2018c) Post infectional Biochemical changes induced by Mealybug *Paracoccus marginatus* (Hemiptera: Pseudococcidae) in leaves of superior seedless grapes. *Curr J of Appl Sci and Technol*31: 1-7.

Murria S, Kaur N, Arora A and Arora NK. (2018b) Biochemical characterization of superior seedless variety of grape (*Vitis vinifera* L.) for resistance to anthracnose.*IndPhytopath*71:399-405.

Murria S, Kaur N, Arora NK and Mahal AK. (2018a) Field reaction and metabolic alterations in grape (*Vitis vinifera* L.) varieties infested with anthracnose. *Scientia Horticulturae* 235:286-293.

Naglaa AA and Heba IM. (2011) Impact of secondary metabolites and related enzymes in flax resistance and or susceptibility to powdery mildew. *W J Agric Sci* 7: 78-85.

Penninckx IAMA, Thomma BPHJ, Buchala A, Metraux JP and Broekaert WF. (1998) Concomitant activation of jasmonate and ethylene response pathways is required for induction of a plant defence. *Plant Cell*10: 2103-2113.

Prasath D and Ponnuswami V. (2008) Screening of chilli genotypes against *Colletotrichumcapsici* and analysis of biochemical and enzymatic activities in inducing resistance. *Ind J Gen Pl Breed* 68: 344-346.

Saharan G S, Joshi, UN and Saharan MS. (2000) Phenolic compounds and oxidative enzymes in healthy and *Alternaria* blight infected leaves of cluster bean. *Acta Phytopathologica* 34: 299-306.

Saleh L and Plieth C. (2009) Fingerprinting antioxidative activities in plants. *Plant Methods* 5: 2.

Santen K, Marttila S, Liljeroth E and Bryngelsson T. (2005) Immunocytochemical localization pf the pathogenesis-related PR-1 protein in barley leaves after infection by *Bipolaris sorokiniana*. *Physiol Mol Plant Pathol*66: 45-54.

Shankar B and Jindal PC. (2001) Biochemical resistance of grape genotypes against anthracnose. *Indian J Agric Res* 35: 44-47.

Sharma JR, Mishra B and Krishana J. (1992) Biochemical relationship in resistant and susceptible cultivars with turcicum leaf blight disease in maize. *Ind Phytopath* 45:241-43.

Sharma S and Gupta N (2020) Defense signaling in plants against micro-creatures: do or die. Indian Phytopathology. https://doi.org/10.1007/s42360-020-00249-4

Silverman P, Seskar M, Kanter D, Schweizer P, Metraux J P and Raskin I.(1995) Salicylic acid in rice (Biosynthesis, Conjugation and possible role). *Plant Physiology*108: 633-639.

Sivritepe N, Kumral N A, Erturk U, Yerlikaya C and Kumral A. (2009) Responses of grapevines to two-spotted spider mite mediated biotic stress. *J BiolSci* 9: 311-318.

Smirnoffss N and Cumbes Q. (1989) Hydroxyl radical scavenging activity of compatible solutes. *Phytochem* 28: 1057–1060.

Stangarlin JR and Pascholati SF. (2000) Activity of ribulose-1, 5-bisphosphate carboxylase-oxygenase (Rubisco), chlorophyllase, β-1, 3-glucanase and chitinase and chlorophyll content in bean cultivars (*Phaseolus vulgaris*) infected with *Uromyces appendiculatus*. *Summma Phytopathol* 26: 34-42.

Suarez V, Staehelin C, Arango R, Holtorf H, Hofsteenge J and Meins JF. (2001) Substrate specificity and antifungal activity of recombinant tobacco class I chitinases. *Plant Mol Biol* 45: 609-618.

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

Ushimaru, T., Nakagawa, T., Fujioka, Y., Daicho, K., Naito, M., Yamauchi, Y., et al. (2006). Transgenic *Arabidopsis* plants expressing the rice dehydroascorbate reductase gene are resistant to salt stress. *J. Plant Physiol.* 163, 1179–1184. doi: 10.1016/j.jplph.2005.10.002

Vargas WA, Martin JM, Rech GE, Rivera LP, Benito EP, Diaz-Minguez JM, Thon MR and Sukno SA. (2012) Plant defence mechanisms are activated during biotrophic and necrotrophic development of *Colletotrichum graminicola* in maize. *Plant Physiol* 4:457-467.

Wu C, Miloslavskaya I, Demontis S, Maestro R and Galaktionov K. (2004) Regulation of cellular response to oncogenic and oxidative stress by Seladin-1. *Nature* 432:640-645.

Xiao YS, Wang ZZ and Guo SC. (1996) The effect of salicylic acid on resistance to bacterial blight in hybrid rice. *Acta-Phytophylacica-Sinica*23: 359-362.

Xu L, Zhu L, Tu L, Liu L, Yuan D and Jin L. (2011) Lignin metabolism has a central role in the resistance of cotton to the wilt fungus *Verticillium dahliae*as revealed by RNA-Seq-dependent transcriptional analysis and histochemistry. *J Exp Bot*62: 5607–5621.

Yadav NK and Thankur DP. (2001) Anatomical defence mechanism in pearl millet leaves against downy mildew infection. *J Mycol Pl Pathol,* 31: 185-187.

Yancey P and Siebenaller J. (2015) Co-evolution of proteins and solutions: protein adaptation versus cytoprotective micromolecules and their roles in marine organisms. *J ExpBiol*218: 1880–1896

**How to cite this article:**

Rohit Chhabra, Simardeep Kaur, Lavanya Vij and Kompal Gaur. 2020. Exploring Physiological and Biochemical Factors Governing Plant Pathogen Interaction: A Review. *Int.J.Curr.Microbiol.App.Sci.* 9(11): 1650-1666. doi: https://doi.org/10.20546/ijcmas.2020.911.197