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Plant Disease Control: Understanding the Roles of Toxins and Phytoalexins in Host-Pathogen Interaction

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Abstract – Naturally, plant habitats are exposed to several potential effects of biotic and different abiotic environmental challenges. Several types of micro-organisms namely; bacteria, viruses, fungi, nematodes, mites, insects, mammals and other herbivorous animals are found in large amounts in all ecosystems, which lead to considerable reduction in crop productivity. These organisms are agents carrying different diseases that can damage the plants through the secretion of toxic-microbial poisons that can penetrate in the plant tissues. Toxins are injurious substances that act on plant protoplast to influence disease development. In response to the stress effect, plants defend themselves by bearing some substances such as phytoalexins. Production of phytoalexins is one of the complex mechanisms through which plants exhibit disease resistance. Several findings specifically on phytoalexins have widen the understanding in the fields of plant biochemistry and molecular biology. However, this review reports the interaction of toxins and phytoalexins in plant-pathogen cycle, research progress on the association of phytoalexins with plant disease resistance as well as the role of the phytoalexins in plant disease control.

Keywords: Defence mechanism, phytoalexins, Plants, plant disease, toxins

Introduction

Vascular plants are consistently exposed to biotic factors such as bacteria, fungi, virus and mites both within and above the soil, with only a few number causes serious diseases. Actually, microorganisms (susceptibility) living together with the host is an exception in the nature, while incompatibility (resistance) is the rule (Cézilly et al., 2014). Generally, after an attack, no apparent sign of micro-organisms attack as the microorganism neglects to set up itself because of a need in actuation of pathogenicity works or to exceedingly effective plant defence mechanisms. Others leave a confirmation of an exceptional host-pathogen interaction that in the end brings about the confinement of the pathogen (Delaney, 1997). The ability of a pathogen to infect and invade a compatible host may be facilitated by the production of toxins that induce cell death in the proximity of the invading organism (Dangl and Jones, 2001). These toxic substances were additionally answered to assume vital parts in hindering the physiological procedures in cells encompassing the purpose of infection, empowering the spread of the illness (Cézilly et al., 2014). A few pathogens would be unsuccessful if the toxin did not execute the cells ahead of time of the fungus and allow it to build up itself constantly on dead or kicking the bucket cells and create more toxins. The harmfulness of a life form is some of the time improved by its capacity to deliver phytotoxins that kill cells in the tissue encompassing the purpose of disease. Also, host tissues regularly activated their response functions that synthesized

antimicrobial enzymes, substances and structural reinforcement to hinder the growth of pathogen (Dixon & Lamb, 1999).

The activation of inbuilt defence mechanisms in plants happens immediately when a disease-causing pathogen attempts to attack the host, for this reason in healthy plants tissues under normal circumstances either the infection-induced mechanisms are absent, or present in lower amounts compared to detections under incompatible interactions. These, however, gives a better approach for what is happening in cellular signalling, and to assess the function of a specific defence mechanisms in resistance (Smith, 1996). Different form of disease resistance in plants were reported which include the age of the plant, induced or acquired, organ-specific, resistance to a non-host, parasite and race-specific resistance (Russell, 2013). These forms of disease resistance could be exploited further by studying the physical and biochemical factors responsible to hinder pathogen penetration and development in the host tissue after infection. The physiological/biochemical basis of resistance of plants to fungal, Oomycete, and bacterial pathogens has been associated with both preformed and infection induced antimicrobial compounds (Russell, 2013). For example, preformed antimicrobial compounds are involved in the resistance of Oats to *Gaumanomyces graminis* f. sp. Tritici (Mert-Turk et al, 2003) and onion bulbs to *Colletotrichum circinans* (Kiehr et al., 2012). Be that as it may, the declaration of resistance (i.e. defence) in most plant-pathogen connections can't be clarified by the nearness of preformed inhibitors. Most research on protection systems has demonstrated that the plant utilizes resistances that are enacted after disease to stop pathogen improvement (Russell, 2013). Numerous biochemical changes happen in plants after disease, and some of these have been related with the declaration of safeguard since they have movement against pathogens *in vitro.*

One kind of biochemical reaction that is firmly connected with defence is the collection of Phytoalexins, which are characterized as low-atomic weight antimicrobial compounds that are synthesized after infection (Jeandet et al., 2013). The possibility that defences can be enacted after disease was solidified by the phytoalexin theory of Muller and Borger (1940), and the investigation of phytoalexins has been a piece of the texture of plant resistance inquires about from that point onward. Phytoalexins have gotten much consideration in the course of recent years. In this survey we introduce the key highlights of this diverse group of molecules, to be specific their compound structures, biosynthesis, elicitors and regulatory mechanisms.

Toxins: A Biochemical Pathogen Induced Changes

Concept of Toxins

Toxins are injurious substances secreted by micro-organism host complex that acts on living host protoplast to affect disease symptoms (Meena et al., 2017). The idea that a toxin plays a role in death of cells was first enunciated by the scientist De Bary in 1886 who claimed that oxalic acid secreted by *Sclerotinia spp.* was responsible for killing of cells. Smith (1996) supported this by implicating oxalic acid secreted by *Botrytis cinerea,* in causing death of living cells. However, Xing et al. (2015), working with the same fungus, reported that oxalic acid was found at low levels in the mycelia and the maltose medium, though it was found at high states in the mycelia and sucrose medium. After sclerotial separation, oxalic acid accumulated at high states in both the sclerotia and the sclerotial exudate. Oxalic acid was thusly found to hinder *P. umbellatus* sclerotial formation. Information about the role of toxins in disease caused by insect, mites, nematodes, and parasitic phanerogams is negligible. It is unlikely that viruses, because of their very nature, would produce toxins in the strict sense of the world. Toxins are readily produced by fungal and bacterial pathogens in a variety of environments (Meena et al., 2017).

In plant pathology, the concept of toxin is not limited to one group of compounds. All kinds of substances such as *Botulinum* neurotoxins (Proft, 2009), produced by the pathogen, which are capable of reproducing symptoms similar to that found in natural infections, are toxins. These are simple molecules with low molecular weight, in contrast to the high molecular weight toxins of human pathogen. Because of the small size, they are extremely mobile and easily reach the sub cellular level of the host.

Role of Toxins in Plant Disease

Chaube and Pudhnir (2005) claimed that micro-organisms are pathogenic only if they are toxigenic. Toxins can be defined as low molecular weight, non-enzymatic microbial products toxic to higher plants. Toxins are different from enzymes in the fact that they do not attack the structural integrity of the tissues but affect the metabolism in a subtle manner. It is the subtility of action that differentiates toxins from enzymes (Kumar & Hayward, 2005).

Toxins are substances that act directly on the protoplast of the cells, but some enzymes for example toxin of *Clostridium welchii,* lecithinase is a toxin because of its disruption of cell membrane (Ravichandra, 2013). In general, enzymes are equal to "aggressins" of mammalian pathology, which are defined as metabolites that enhance virulence of pathogens but are not directly toxic to the host. In recent classification, toxins are divided into two categories (Ravichandra, 2013). The first is host-non-specific (non-selective) which may affect many unrelated plant species in addition to main host of the pathogen producing toxin; it includes phytotoxin and vivotoxin. *Phytotoxin* is any compound produced by a micro-organism which is toxic to plant while Vivotoxin is characterized as a substance produced in the attacked host by the pathogen or potentially its host, which works in the creation of disease however isn't itself the underlying affecting operator of disease, example is the Fusaric acid (Ravichandra, 2013; Adam et al., 2015). The second is host-specific (selective) which affects only the specific host of the pathogen; it includes pathotoxins. Toxins in general, interact with cell membrane or organelles (mitochondria or chloroplast) and alter their permeability. Some important non-host-specific and host-specific toxins are shown in tables 1 and 2.

Table 1: Some important host-non-specific toxins

Toxin	Pathogen	Disease	Chemical Nature	References
Cercosporin	*Cercospora spp.*	-do-	Benzoperyline derivatives	Newman and Townsend (2016); Chaube and Pundhir (2005)
Fumaric acid	*Rhizopus spp*	Almond hull rot disease	Fumaric acid	Das (2016)
Oxalic acid	*Sclerotium and Sclerotinia spp.*	Rots in various crops	Oxalic acid	Bennett (2015)
Ten toxin	*Alternaria alternata*	Chlorosis of seedlings in many plants	Cyclic tetrapeptide	Li et al. (2016)
Syringotoxin	Ps.*Syringae* pv.*syringe*	Citrus plant	_	Chaube and Pundhir (2005); Awada et al. (2014)
Coronatine	Ps.*Syringae* Pv.*atropuspurea*	Infected soyabeans and grasses	_	Chaube and Pundhir (2005)
Diaporthin	*Cryphonectria parasitica*	Chestnut blight	Isocoumarin	de Medeiros et al. (2018)
Cerato-ulmin	*Ceratocystis ulmi*	Dutch elm disease	Large M carbohydrate	Zhang et al. (2018)
Alternaric acid	*Alternaria spp.*	Leaf spot disease of various crops	Hemiquinone derivatives	Templeton (2016)

Table2: Some important host-specific toxins

Toxin	Pathogen	Target Site	Host Range	Disease	References
ATC-toxin	*Aternaria tenuissima*	_	Pigeon pea	Alternaria leaf spot of pigeon pea	Ostry (2008)
AM-toxin I,II and III	*Alternaria alternata*	Chloroplast and plasma membrane	Apple (red, gold and starking	Alternaria blotch of apple	Takashi (2013)
PM-toxin A,B,C, and D	*Phyllosticta maydis*	Mitochondrion	Maize	Yellow leaf blight of maize	Kohmoto and Yoder (2012)

Phytoalexins: An induced biochemical defence mechanism

The induced biochemical changes in host plants are the last line of host defence (Glazebrook & Ausbel; Ausubel, 1994). This may condition a plant or plant tissue from susceptible to resistant to immune status as per their genetic potential. Phytoalexins are antimicrobial and often antioxidative substances synthesized *de novo* by plants that accumulate rapidly at areas of pathogen infection (Jeandet et al., 2013). As an important component of overall active defence strategy of plants, effective concentrations of these substances produced rapid production/suitable modification and/or accumulation of chemicals toxic to pathogens. Singh (2002) suggested that to establish the role of a biochemical factor in host defence it must possess the four attributes and match the following "Koch's postulates" for pathogenicity (modified) (Fredericks & Relman, 1996).

1. The substance is related with insurance against infection at the site where protection happens
2. The substance can be isolated from the host indicating assurance against the disease
3. Introduction of isolated substances to the suitable susceptible host gives protection
4. The nature of protection so actuated looks like that of the natural agents of a resistant plant

Slow accumulation of similar chemicals has been reported in susceptible host plants also (compatible interaction) (Pusztahelyi et al., 2015; Jeandet et al., 2013). These substances include; phenolic compounds, phytoalexins, new protein synthesized, inactive of enzymes and toxins and altered biosynthetic pathways Pusztahelyi et al., 2015; Jeandet et al., 2013).

Concept of Phytoalexins

In 1940, Muller and Boger proposed that plants produced defensive substances, called Phytoalexins, in response to infection. The term was derived from Greek to mean "warding off agents in plants", and proposal was made after deliberating two important phenomena in plant pathology. First, the active response of the cells of many plants to attempted infection; second, the acquisition of resistance by plants after exposure to an infecting organism. Compounds that acts against micro-organisms from plants are generally divided into two main classes: phytoantipicins and phytoalexins (Mansfield, 1999). Phytoantipicins are described as "low molecular weight, antimicrobial compounds that are present in plants before challenge by micro-organisms, or are produced after infection solely, from pre-existing precursors". Phytoalexins are defined as "low molecular weight, anti-microbial compounds that are both synthesized and accumulated in plants after exposure to micro-organisms or abiotic agents" (Purkayastha, 2017). Phytoalexins is one of the substances or compound that catalyzed the induced defence mechanisms employed by plants including lytic enzymes such as chitinases and glucanases, oxidizing agents, cell wall lignifications and a number of pathogenesis-related (PR) proteins and transcripts of unknown functions (Dixon & Lamb, 1999). It is imperative to note that the gradual increase of phytoalexin may be part of a co-ordinated defence approach, in which any one factor may alone be unable to account for restriction of the potential pathogen (Purkayastha, 2017; Mansfield, 1999).

Elicitors of Phytoalexins Accumulation

The biosynthesis of phytoalexins compounds after being attacked by the pathogen was believed to be either triggered by the substance produced by the pathogen or the host-pathogen interaction. A number of different pathogen and plant-produced molecules, referred to as elicitors (Ahuja, 2012), will triggered phytoalexins and other defence responses. Several research have been reported for the possibility of plant cells having receptors for these elicitors (Horsfall, 2012). Some elicitors have been reported to have the same specificity as the pathogen has with its host while most elicitors showed a lack of any specific connection to the outcome of a host-parasite interaction (Ahuja, 2012; Horsfall, 2012). The cutting edge synthesis of the gene-for-gene theory expresses that protection happens just when the result of a pathogen avirulence gene interacts with the result of a plant resistance gene (Purkayastha, 2017). On account of the high level of specificity, gene-for-gene system give a decent structure to decide whether the result of the avirulence gene can likewise go about as a race-/cultivar-specific elicitor of defence response like phytoalexins accumulations (Ahuja, 2012; Purkayastha, 2017)

The molecular compounds that informed plants to start the production of phytoalexin are referred to elicitors. Elicitors of biotic inception might be associated with the communication of plants and potential pathogens, while abiotic elicitors are not engaged with typical host-pathogen interaction (Purkayastha, 2017). In common conditions, the boost is given by the nearness of the micro-organisms and its perception by the host starts the chain of processes prompting phytoalexin synthesis. Biotic elicitors may come from the attacking organism, in which case they are called "exogenous", though "endogenous" elicitors are of plant origin and are produced by the communication between micro-organism and plant. Particles with elicitor action have been recognized over an extensive variety of structural kinds including polysaccharides, glycoproteins, lipids, lipopolysaccharides, oligosaccharides and even enzymes, however their action can be credited to their impact in discharging elicitor-active segments from the cell wall of the pathogen or host (Bostock et al., 1992; Alghisi & Favaron, 1995). Abiotic elicitors form a different accumulation of molecules that are not gotten from natural sources, for example, the tissues of the pathogen or host. Under ordinary conditions, they would not be experienced by the plant. The group include compounds, for example, fungicides; salts of heavy metals, for instance Cu^{2+} and Hg^{2+}; the cleansers, essential molecules, for example, polylysine and histone; reagents that are intercalated DNA (Purkayastha, 2017). Treatment of plant tissues with factors that cause stress, for instance rehashed solidifying and defrosting, injuring or introduction to UV light (Liu et al., 2015; Mert-Türk et al., 1998) can likewise instigate phytoalexin synthesis.

Role of Phytoalexins in Plant Disease Control

Most basic reaction of plants to pressure, biotic (pathogen/insects) or abiotic (injuring), is the generation and amassing of substrates that can restrain the development and exercises of the biotic factors or may help in recuperating process. Hammerschmidt, 1999 reported that in plants continuous irritation by pathogen is essential for production of effective amount of these phenolic compounds. Kuc (1995) defined phytoalexins as antibiotics produced in plant-pathogen interactions or as result response to injury or other physiological stimulation. Wide variety of toxic chemicals was reported to increase in concentration in response to infection, thus phytoalexins are now considered as low molecular weight antimicrobial compounds produced *de novo* in plants as a result of infection or abiotic stress. This excludes the pre-existing phenols, example chlorogenic acid, caffeic acid and scopoletin. The phytoalexins have demonstrated in wide variety of plants belonging to families *Gramineae* (Oats, rice, sorghum, and sugarcane), *Solanaceae, Leguminaceae, Chenopodiceae, Convolulaceae, Compositae, Malvaceae* and *Umbellifera* (Table 3). Members of *Orchifaceae* are known for production of phytoalexins. Chemical structures of some phytoalexins cited in this work (Figure 1).

Table 3: Common phytoalexins reported in host-pathogen interaction

| Phytoalexin	Host	Fungal Pathogen	References
Capsidol	*Nicotinia clvelandi,* N tabacum	Tobacco necrosis virus (*Capsicum*)	Chaube and Pudhnir (2005)
Pistin	Pea (endocarp) pods, leaves	*Monilinia fructicols* non pathogen	Mazid et al. (2011)
Glutinosone	*Nicotina glutinosa*	T.M.V	Burden et al. (1975)
Ipomeamarone	Sweet potato	*Ceratocystis fimbriata*	Mawalwa et al. (2014)
Wyerone	Pea	*Botrytis fabae*	Slusarenku et al. (2012)
Triflorrhizin	*Trifolium pratense*	*Monilia fructicola*	Chaube and Pudhnir (2005)
Glyceollin	Soyabean	*Pytophthora megasperma* var. sojae	Ng et al. (2011)
Rishitin, Phytuberin	Solanaceae	fungal sterol, ergosterol	Tugizimana et al. (2014)
Sativan, Vestitol	Alfalfa, *Lotus corniculatus*	*Helminthosporium turcicum Pass*	Bonde et al. (1973)
Isocoumarin	Carrot	-	Lafuente et al. (1996)
Vergosin and Hemigossypal	Cotton	-	Chaube and Pudhnir (2005)
Avenalumin I,II and III	Barley	*Puccinia caronata* f.sp. avenae	Chaube and Pudhnir (2005)
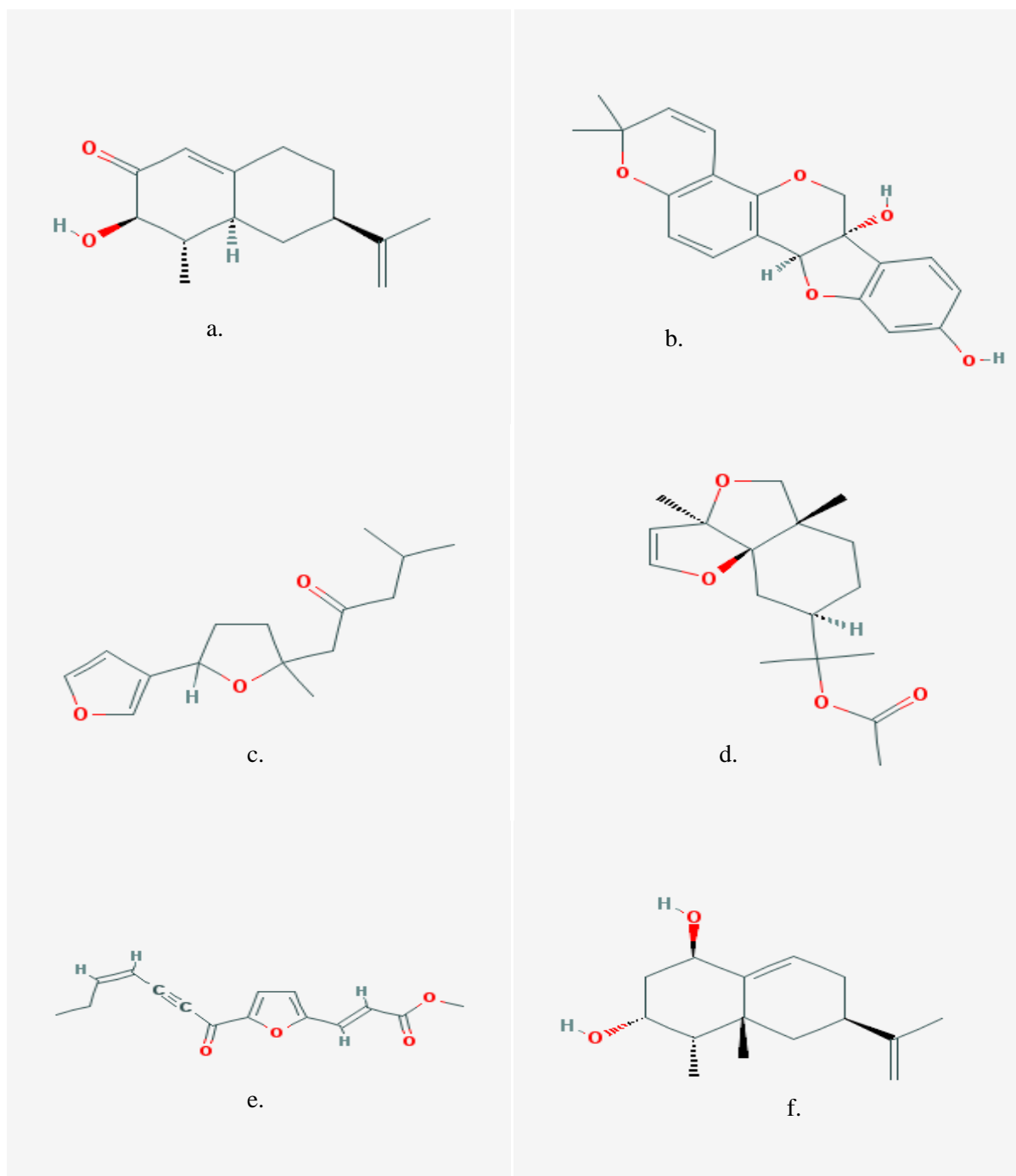
Figure 1: Chemical structures of some phytoalexins cited in this work: Glutinosone (a), glyceollin (b), Ipomeamarone (c), phytuberin (d), wyerone (e) and capsidol (f). Source: National Center for Biotechnology Information. PubChem Compound Database; https://pubchem.ncbi.nlm.nih.gov/compound (accessed Apr. 14, 2018).

Resistance to Fungi

The available evidence concerning the contribution of phytoalexins to the restriction of fungal growth at different stages of colonization include;

Inhibition on plant surfaces: Fungal spores often fail to germinate following their deposition on leaf surfaces (Friend, 2012). A striking example of this concern is the behaviour of saprophytes in the phyllosphere. Ahuja (2012) have described the increased growth of epiphytic fungi coincident with the onset of senescence. The ability to produce phytoalexins declines during senescence (Friend, 2016)

and it has been proposed that fungal growth on young leaves may be restricted by phytoalexins produced by underlying cells in response to fungal metabolites diffusing from germinating spores (VanWees et al., 2003). However, the limited evidence available does not support this attractive hypothesis. Thus, Mansfield et al., 1982 found that germination of saprophytes *Aureobasidium pullalans, Cladosporium herbarum* and *Epicoccum nigrum* on pea leaves did not induce formation of the phytoalexin pisatin. The apparent lack of influence of phytoalexins on fungal development in the pyllosphere may be explained if the cuticle acts as a barrier preventing the diffusion to underlying cells of compounds eliciting phytoalexin biosynthesis.

Inhibition during attempted penetration into plant cells: Protection from fungi is as often as possible communicated by the failure of disease hyphae to enter into or through plant cell walls (Mellersh and Heath, 2003). Different sorts of deposit (Papillae) have been found to aggregate inside living cells underneath destinations of endeavored entrance. It has been proposed that papilla development and other confined changes in cell wall structure including lignification (Friend, 2016) and silification (Mellersh and Heath, 2003) may give simply physical boundaries to the continued advance of attacking hyphae. Friend (2016) isolated a fungitoxic flavonoid (which may be considered a phytoalexin) from papillae formed in resistant barley leaves in response to *Erysiphe graminis* f. sp. Hordei. It is possible that other phytoalexins may also be incorporated into papillae or cell walls, thereby producing a localized, fungitoxic barrier to penetration.

Inhibition after penetration: Following penetration of resistant plants, fungal growth may be restricted at a number of sites: within the partially degraded walls of epidermal cells (for example *Botrytis spp.* in non-host plants); intracellularly, either within the epidermis (*Colletotrichum spp* in non-host plants or resistant cultivars) or in mesophyll cells (restricted development of hautoria of rust fungi); in intercellular spaces (*Cladosporium fulvum* in resistant tomato leaves; and within xylem vessels (*Verticillium* and *Fusarium spp.* in wilt resistant plants). In other to prove whether or not inhibition of hyphal growth at these sites is caused by phytoalexins, it would necessary to measure the concentrations of inhibitors to which hyphae are exposed at the time they stop growing and also to examine the activity of what may be a mixture of phytoalexins at the site of exposure (Mansfield, 1999).

Botrytis spp and *Vicia faba:* The production of phytoalexins by tissues of *Vicia faba* in response to infection by *Botrytis* had been examined for several years before attention was paid to the precise timing of phytoalexins accumulation and the cessation of fungal growth during resistant reactions (Mansfield, 1999). *Colletotrichum lindemuthinum* spores on *French bean* germinate within 48hours of inoculation and produce similar numbers of appressoria on resistant and susceptible plants (Wheeler, 2012). Other example is the *Phytophthora infestance* and *potato tissues* (Wheeler, 2012)

Resistance to Bacteria
Studies in the role of phytoalexins in resistance to bacteria have been mainly concerned with the restriction of bacterial multiplication within intercellular spaces.

Pseudomonads and French bean and soyabeans. The most detailed studies of the involvement of phytoalexins in bacterial infections concern the French bean plant and the resistance of leaves of certain cultivars to halo blight caused by *Pseudomonas phaseolicola* and of pods to avirulent isolates of *P.syringae*. The multiplication of compatible and incompatible races of *P.phaseolicola* in bean cv. Red Mexican and the timing of symptom appearance are established (Schmelz et al., 2014). The compatible race is to multiply rapidly causing water-soaked lesions to develop between two and four days after inoculation; these lesions become brown and desiccated after five days. The incompatible race multiplies less rapidly and causes a hypersensitive reaction, inoculation sites collapsing to form localized desiccated brown lesions within two days. Collapse of tissue during the hypersensitive reaction is closely associated with the cessation of bacterial multiplication (Schmelz et al., 2014).

Resistance to Nematodes

Researchers have studied the resistance of legume roots to nematodes. For example, lima bean roots exhibit a hypersensitive resistance response to *Pratylenchus scribneri.* Jeandet et al. (2014) found that tissue bearing necrotic lesions caused attempted feeding of the nematode in the epidermis and cortex accumulated the fluorescent isoflavonoids coumestrol and psoralidin. These compounds were present only in low concentrations in uninoculated roots. Coumestrol inhibited the motility of *P.scribneri* at concentrations less than those found within infected roots leading Jeandet et al. (2014) to conclude that induced accumulation of the coumestan phytoalexin is probably the chemical basis for the resistance of lima bean roots to *P.scribneri.* The role of glyceollin in the expression of varietals resistance of soyabean cv. Centennial to the root knot nematodes *Meloidogyne incognita* has also been examined.

Resistance to Viruses

Antifungal phytoalexins accumulate during the production of local necrotic lesions by viruses in leaves of legumes and *Nicotiana spp.* but they are absent from systematically infected plants (Jeandet et al., 2012). There have been few attempts to determine if phytoalexins suppress viral replication and thereby restrict lesion size. Hammerschmidt (1999) found that incubation of tobacco necrosis virus (TNV) in soyabean leaf extract containing low concentrations of glyceollin had no effect on viral infectivity. They suggested, however, that the presence of the phytoalexin in tissues immediately surrounding lesions might indirectly render them unsuitable for further virus multiplication. Glyseollin was not translocated and was not involved in systemic protection against TNV afforded by the prior inoculation of soybean leaves with the virus.

In spite of the work done on phytoalexins and amount of evidences presented, there are certain questions to be satisfied before establishing direct role of phytoalexins *in vivo* containment of pathogen (Jeandet et al., 2014). The significant role of phytoalexins in plant defense mechanisms has long been debated addressing both the actual antimicrobial activity of phytoalexins under the conditions found within plant tissues and their localization around invading organisms (Mansfield, 1999; Hammerschmidt, 1999). These immovable cross examinations are in reality urgent to their proposed part as microbial growth regulators in infected plant tissues. Regardless there is impressive confirmation that these compounds show *in vitro* toxicity crosswise over a great part of the biological range, prokaryotic and eukaryotic.

Generally, phytoalexins accumulate at infection sites and they restrain the growths of fungi and bacteria *in vitro* in this way, it is legitimate to consider them as conceivable plant-defence mixes against diseases caused by fungi and bacteria. Contingent on the phytoalexin, fungus and bioassay, the EC50 for fungi is for the most part 10-3 to 10-5 M (Kuc, 1995). In this manner they are similarly powerless as antifungal agents. In spite of the fact that there is no confirmation that phytoalexins are translocated. Localization at the disease site may allow the pathogen to encounter concentrations far in overabundance of the EC50 at early stages in the in the infection process (Mert-Türk, 2002).

There are additionally cases that phytoalexins aggregated amid perfect plant-pathogen communications. These incorporate the induction of pisatin by the harmful Oomycete *Aphanomyces euticles* (Pueppke and VanEtten, 1976) and by the pathogenic strains of the fungus *Nectria hematococca* and induction of spirobrassinin by virulent races of *Leptosphaeria maculans* (Howlett et al., 2001). Likewise, Glazebrook and Ausubel (1994) reported that the harmful pathogen *Pseudomonas syringae* pv. maculicola evokes the synthesis of large amounts of camalexin in *Arabidopsis thaliana*. Mert-Türk et al., (1998) additionally demonstrated that camalexin gathered amid both compatible and incompatible communication in *A. thaliana* when tested with an Oomycete, *Peronospora parasitica*. On the off chance that the outcomes exemplified are translated, in incompatible interactions, phytoalexin collection limits or stops pathogen development, in this way presenting protection to the plant. In compatible interactions, the pathogen evidently, endures the amassed phytoalexins, detoxifies them, stifles phytoalexin gathering or abstains from phytoalexin production (Mansfield, 1999).

Conclusion

Phytoalexins as they gather both in susceptible and resistant plants, the genuine inquiry is that whether they are supporters of defence or simply the final result of pathogen-or stress-actuated metabolism. More indisputable methodologies could be utilized to answer the inquiry. One of them could be to generate phytoalexin biosynthetic mutants that never again produced phytoalexins, at that point to evaluate them whether phytoalexin deficiency causes increased susceptibility. There are two greatly basic focuses here that ought to be remembered. This approach ought to incorporate hereditarily examination utilizing a framework in which the biochemical and physiological proof contends unequivocally for a key part for phytoalexins in resistance. Second point is that the plants must be assessed for changes in different defence mechanisms that may make up for the loss of phytoalexin production. Because of advances in molecular, much better perspective of the part of phytoalexins in defence has built up. Unmistakably, future investigations on these compounds will enable us to comprehend and assess plant pathogen interaction and in addition give new ways to deal with disease control. All efforts in molecular biology and biotechnology is to bring new methodologies into disease control for friendlier condition.

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