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Phytophthora-citrus interactions and management strategies: a review

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Abstract: Citrus production is declining worldwide due to several biotic and abiotic factors. The diseases caused by Phytophthora spp. present major economic risks since they are soil-borne and spread quickly if environmental conditions are favorable, or irrigation is poorly managed. Phytophthora species are present in all citrus-producing areas around the world causing significant losses in crop yield and affecting tree health. Bark infection, damping-off, root rot, gummosis, brown rot, and cortical root rot are among the typical symptoms caused by *Phytophthora* spp. The pathogenicity of *Phytophthora* spp. depends mainly on the specific interactions between the isolates and citrus cultivars. The use of molecular technologies has allowed the study of Phytophthora-citrus interactions, leading to the identification of several classes of effector proteins secreted by *Phytophthora* spp. that challenge plant homeostasis and contribute in different ways to disease development and the elucidation of defense mechanisms employed by the host plant. Comprehensive management is need of time to conquer *Phytophthora* spp. for better root health and citrus productivity.

Key words: Citrus production, *Phytophthora* spp., epidemiology, symptomatology, management

1. Introduction

Citrus is one of the major fruit crops in the world. However, its production is greatly affected by abiotic stresses i.e. salinity (Khalid et al., 2020, 2022); drought (Hussain et al., 2018; Khalid et al., 2021a, 2021b); temperature (Shafqat et al., 2019, 2021), light (Oustric et al., 2018) and diseases i.e. Huanglongbing (Hussain et al., 2019; Nehela and Killiny, 2020) and *Phytophthora* (Graham and Feichtenberger, 2015). *Phytophthora* species adversely affect citrus growth and health, leading ultimately to significant quality and yield losses in commercial groves.

Phytophthora spp. is a pathogenic oomycete encountered in temperate, subtropical, and tropical climates on diverse hosts ranging from herbaceous plants such as tomato and tobacco to woody plants like citrus and eucalyptus. *Phytophthora* species pose serious threats to the economic viability of citrus via soil-borne infections. Severe crop losses have been reported in citrus groves in the Azores islands long before the Irish potato famine of 1845 and the discovery of the pathogen 31 years later. Since then, many *Phytophthora* epidemics have followed in France (1841), Italy (1855–1889), Australia (1860–1879), Spain (1871), United States (1875–1876), Greece (1869–1880), Cuba (1906), Paraguay (1911), Brazil (1917), and Mexico (1920) (Savita and Nagpal, 2012). The development of grafting techniques and the use of sour orange as a rootstock, during the second half of the 20th century, slowed down the development of *Phytophthora* diseases but failed to eradicate (Laville, 1974).

Many *Phytophthora* species are associated with trunk gummosis and root rot symptoms, being *P. citrophthora* and *P. nicotianae* (syn. *P. parasitica*) the most destructive (Vernière et al., 2004). Disease outbreaks of these species have been widely reported in tropical and subtropical regions including Florida (Timmer et al., 1988), India (Uppal and Kamat, 1936), Spain (Alvarez et al., 2008, 2009),

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and Morocco (Vanderweyen, 1974; Boudoudou et al., 2015). *P. boehmeriae, P. cactorum*, *P. cinnamon*, *P. citricola*, *P. dreschleri*, *P. hibernalis*, *P. megasperma*, *P. palmivora*, and *P. syringae* have also been reported as pathogenic on citrus, but limited reports are available on the distribution of these species and their interactions with the hosts (Erwin and Ribeiro, 1996). In this review, we summarize the typical symptoms and epidemiology of these diseases, the genetic background of rootstock/scion tolerance, and the molecular aspects involved in *Phytophthora* virulence and citrus defense mechanisms.

2. Symptomatology

Phytophthora species colonize the soil, they attack all plant organs causing major economic risks throughout the vegetative cycle. In nurseries, *Phytophthora* causes damping-off and seed rot or preemergence rot. Yield losses in groves are mainly related to root rot, stem/trunk gummosis, and fruit brown rot. In extreme cases (susceptible rootstocks), fibrous roots are severely damaged resulting in tree dieback (Figure 1).

Bark infection usually occurs near the soil level and appears at the crown or trunk as lesions that girdle the tree by developing a belt around the circumference and spread to secondary branches causing the dieback of trees (Savita and Nagpal, 2012), as reported previously in clementine cultivars (*C. clementina* Hort. Ex Tan.) and their hybrids (Alvarez et al., 2008, 2009). Cankers and gum exudations were visible on the aerial parts (scions), particularly on the major branches (Alvarez et al., 2008).

Damping-off occurs often in poorly managed nurseries following attacks by *P. nicotianae*, *P. citrophthora*, and *P. palmivora*. Typical symptoms of damping-off appear shortly after the penetration of the lower part of the stem by *Phytophthora*. The contamination may originate from seeds or soil particles. When the attacks occur early, they may cause seed rotting and impede emergence. Young, infected seedlings usually die within a few days, while others acquire resistance once the true leaves have appeared and the lower stem tissues have matured (Lamichhane et al., 2017). Root rot occurs when the graft union is close to the ground. Infected scions show lesions that may extend up to the graft union if the rootstock is resistant (Fawcett, 1913). If the rootstock is susceptible, rot symptoms can also be observed in the underground part (roots and crown). The infected bark exhibits small cracks yielding profuse gum exudation.

Symptoms of gummosis disappear under heavy rains but persist under dry conditions. They disintegrate slowly around the perimeter of the trunk and are often accompanied by leaf symptoms (pale color, yellowing of the veins). As soon as the infection stops, the lesions stop spreading and the damaged area of the bark is surrounded by callus tissue. Seedlings and young trees at nurseries, having small stem circumferences, die off quickly, while older trees are more tolerant. Although dieback may occur in orchards, infection of the trunk is often partial and is accompanied by leaf drop and twig dieback in extreme cases (Timmer and Menge, 1988; Timmer et al., 2003; Alvarez et al., 2008).

Cortical root rot is usually less common than trunk and foot gummosis but is more difficult to identify since anything that hinders root development is likely to cause identical symptoms. At first, the infection is localized at the temporary fibrous roots. The colonization of the cortical area of this type of roots by the pathogen shortens their lifespan. The cortex becomes soft, discolored, and exhibits water-soaked lesions. When the infection is severe, the cortex is destroyed leaving only the white fiber-shaped stele. At this stage, the regenerative power of the fibrous roots can no longer keep pace with degradation and the tree is unable to maintain adequate absorption of water and nutrients. In the aerial part, these effects result in leaf drop, twig dieback, and reduced growth and fruit production (Timmer and Menge, 1988; Timmer et al., 2003).

In the field, fruits in the lower part of the tree can be infected with the propagules of *Phytophthora* spp. following the splash of soil particles. At first, these fruits show a slight discoloration of the skin, then an extension of the necrosis which takes, depending on the variety, different shades of brown. The affected area widens and the tissues soften. Under humid conditions, *Phytophthora*'s white mycelium can grow on the fruit surface. The disease then spreads to other fruits of the tree if the temperature (24–28 °C) and humidity are favorable. Outbreaks of brown rot are more frequent in areas where rainfall coincides with the early fruit maturity stages (Timmer and Menge, 1988; Timmer et al., 2003). Although most infected fruits drop from the tree before harvest, some appear healthy and only develop symptoms after a few days of storage, causing important postharvest losses. Brown rot spreads quickly to the other fruits in the container and a very characteristic odor develops resulting from the decomposition of essential oils released by the action of the pathogen (Savita and Nagpal, 2012).

Leaves are infrequently targeted by the pathogen as compared to fruits. However, when the climatic conditions are favorable, translucent spots like frost symptoms may appear on leaf blades. These spots become watery and blackish with time. The leaves fall early while still green (but spotted with black). Complete defoliation may occur on lower branches (Laville, 1974). Lemons are particularly known to be sensitive to leaf and fruit attacks (Graham and Timmer, 1992).

Figure 1. Common symptoms caused by *Phytophthora* spp. on citrus (a) Dieback of a clementine tree ; (b) gummosis developing on the entire trunk circumference; (c) infected tissue visible under the bark ; (d) graft union between a susceptible rootstock and a resistant scion; (e and f) fibrous root rot; (g) leaf yellowing; (h and i) fruit brown rot.

3. Epidemiology

P. nicotianae produce and release a large number of zoospores, chlamydospores, and oospores (Meng et al., 2014). When conditions are favorable (30–32°C), zoospores encyst and germinate in the form of mycelia. Moderate water deficits (–5 to –70 kPa), nutrient depletion and light stimulate the production of sporangia from the mycelium, which may germinate directly or indirectly to produce zoospores (Englander et al., 2006). The interaction of soil infestation with *P. nicotianae* and root rot severity indicates that indirect germination is more crucial in the life cycle (Figure 2).

Zoospores can travel short distances in water using their flagella or can be transported by rain or irrigation water. They are attracted to the elongation zone of the new roots by the nutrients in the exudates. Upon contact with the root, zoospores encyst, germinate and initiate infection at root tips (Khew and Zentmyer, 1974; Besoain et al., 1998). After penetration, the infection progresses into the cortex causing the entire root tissue to rot. The rotten cortex is thus degraded and *Phytophthora* produces thick-walled spores (chlamydospores) which can persist in the soil for long periods (Tsao and Ocana, 1969).

Chlamydospores are also produced during summer periods, when the soil is poorly aerated with high CO_2 concentration, under nutrient depletion, or at low temperatures. They help the oomycete survive for long periods on root debris until the return of favorable conditions when they germinate indirectly to produce sporangia which in turn release mobile zoospores, or

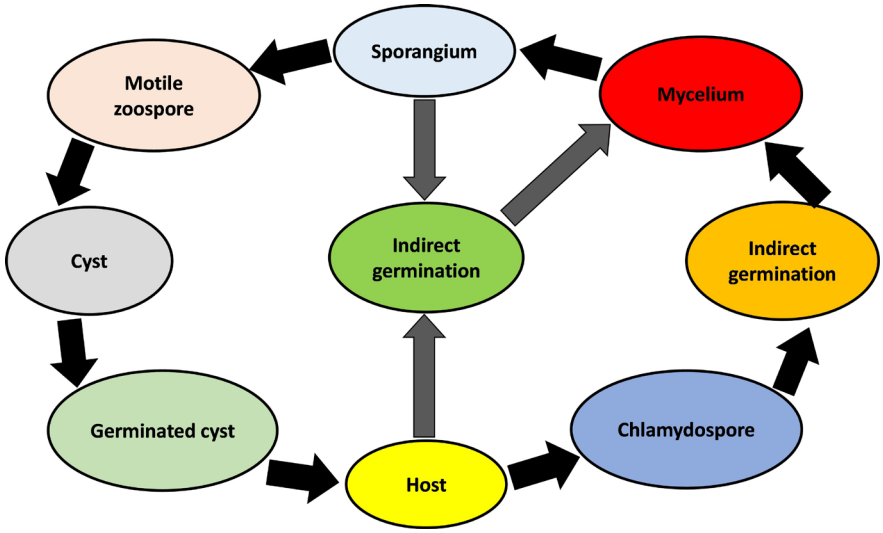

Figure 2. Life cycle of *Phytophthora nicotianae*.

directly to produce the mycelium (Meng et al., 2014). Like sporangia, chlamydospores and oospores require water to germinate. Altering high and low temperatures may, however, be necessary for uniform germination (Timmer and Menge, 1988). Exposure to temperatures of 28–30 °C and the presence of soil extracts and/or citrus roots were reported to alleviate the dormancy of *P. nicotianae* chlamydospores (Graham and Timmer, 1992).

Root rot and trunk/foot gummosis occur upon zoospores entering the trunk beyond the graft union. Moisture and the presence of natural injuries on the trunk are determining factors for the initiation of the infection. The lesions that develop on the trunk do not produce a secondary inoculum, however, in the case of leaf and fruit rot epidemics, secondary infections can be caused by an inoculum coming from the aerial parts of the tree and dispersed by wind or splashing rain. This type of infection is uncommon in *P. nicotianae*, known for nonproduction of aerial sporangia, but is often found in *P. citrophthora* and other species producing a heavy number of sporangia on the leaf and fruit surface (Graham and Timmer, 1992; Naqvi, 2000). The *Phytophthora* disease cycle can repeat itself upon attainment of favorable conditions with prevalent susceptible tissues.

4. Molecular basis of Citrus-*Phytophthora* interactions

The coevolution of *Phytophthora* spp. with its wide host range has generated diverse and complex plant-pathogen interactions regulated by various molecules and genes from both sides. To initiate infection, zoospores land on the host tissue encysts and produce an appressorium to penetrate the host surface. Once inside, the pathogen develops a network of apoplastic mycelium. During this biotrophic stage of the infection, *Phytophthora* species produce haustoria which contribute not only to nutrition but also to virulence through the secretion of proteins known as effectors (Evangelisti et al., 2017). These proteins suppress the immunity of the host and reprogram its physiology in favor of the infection through a process known as effector-triggered susceptibility (ETS). Upon the recognition of the effectors, host plants can activate their defense system through a process known as effector-triggered immunity (ETI). Using specialized receptors, plants may also recognize small molecular motifs specific to the pathogen, called pathogen-associated molecular patterns (PAMPs), and activate another layer of a defense mechanism known as PAMP-triggered immunity (PTI). Both ETI and PTI can lead to a hypersensitive response (Dalio et al., 2017a).

4.1. Effector-triggered susceptibility

The application of high-throughput sequencing technologies has allowed the identification of several classes of effector proteins secreted by *Phytophthora* species during their interactions with their respective hosts. These are conserved amongst many strains and are responsible for a substantial contribution to virulence (Dangl et al., 2013). Apoplastic effectors, such as elicitins (proteins with low molecular weight sharing PAMP features), are secreted into the apoplast of infected plants and are known to induce hypersensitive reactions (Khatib et al., 2004; Oßwald et al., 2014), while cytoplasmic effectors are secreted inside plant cells where they act as suppressors of cellular plant defense mechanisms. These include the widely studied RxLR protein family and Crinkler effectors (CRN).

Apoplastic effectors are known to counteract plant defenses through the inhibition of enzyme activity (Rose et al., 2002). To date, many of them have been associated

with physiological disorders in citrus, including the elicitin ParA1, the necrosis and ethylene-inducing peptide (NEP1- like protein), the necrosis-inducing *Phytophthora* protein 1 (NPP1), and the cellulose-binding elicitor and lectin activity (CBEL) effectors. Elicitins and elicitin-like proteins have been found to be organized as multigene in several citrus-pathogenic *Phytophthora* species including *P. nicotianae, P. citrophthora,* and *P. citricola*. (Kamoun et al., 1993, 1994). Boava et al. (2011) reported an upregulation of elicitins in citrus at late stages of infection, which indicates their involvement in the late necrosis of the tissues of susceptible varieties.

From a pathological point of view, RxLR is probably the most important effector of *Phytophthora* pathogenesis, since they carry a conserved amino acid motif on their N-terminal structure which facilitates their intrusion into plant cells. Using a bioinformatics approach, Dalio et al. (2017b) have identified 172 candidate RxLR effectors in the isolate IAC01_95 of *P. nicotianae* that was collected from different hosts including citrus. Five of these effectors were upregulated in vitro and in planta conditions, and three of them were found to enhance ETS (effector-triggered susceptibility) and suppress PCD (programmed cell death) reactions induced by the INF-1 elicitin in the model plant *Nicotiana benthamiana*, thus confirming their contribution to the pathogen virulence.

Crinkler proteins (CRN) form another group of cytoplasmic effectors that present a highly conserved N-terminal amino acid domain (Haas et al., 2009). They are produced by most *Phytophthora* species, including *P. nicotianae* (Tyler et al., 2006; Haas et al., 2009). However, the CRN effectors involved in *Phytophthora*-citrus interactions have yet to be functionally characterized. Putative apoplastic and cytoplasmic effectors that have been extensively studied in model plants and that might contribute to pathogenicity in citrus are presented in the Table.

4.2. Effector-triggered immunity

Research has identified several key players in the plant immunity system that are activated in response to oomycete attacks, although the underlying mechanisms remain poorly understood and the current knowledge is mainly focused on the aboveground part of the plant. These defense mechanisms include (i) activation of resistance genes (Kamoun et al., 1993; Boava et al., 2011; Zhu et al., 2012), (ii) subcellular arrangements of the infected cell, and remodeling of the cytoskeleton (Takemoto et al., 2003; Takemoto and Hardham, 2004; Hardham, 2007), and (iii) accumulation of endocytic vesicles around the haustoria and secretion of antimicrobial compounds such as phenolic compounds, defensins, protease inhibitors, hydrolytic enzymes, ROS, and phytoalexins, into the extrahaustorial matrix (Lipka et al., 2005; An et al., 2006; Kwon et al., 2008).

Studies on model plants have particularly highlighted the orchestrating role of R-genes and endogenous phytohormones in signaling pathways leading to the production of these antimicrobial compounds (Verma et al., 2016). For instance, *A. thaliana* mutant lines with compromised salicylic acid, jasmonic acid, and ethylene signaling pathways have shown increased susceptibility to *P. nicotianae* (Attard et al., 2010). By studying transcriptional changes in *N. benthamiana* upon root infection with *P. palmivora*, Evangelisti et al. (2017) identified a gene encoding a secreted peptide precursor with potential damage-associated molecular pattern (DAMP) motifs whose promoter was specifically activated at root tip infection sites. In citrus, resistance to *P. nicotianae* has been attributed to the recognition of the pathogen effectors by TIR-NBS-LRR RPS4 and another R- gene of the same class, and the subsequent deployment of plant defense mechanisms (Boava et al., 2011). Nevertheless, defense strategies may vary depending on the rootstock. For instance, when infected with *P. nicotianae*, *Citrus sunki* (susceptible rootstock) activates its main defense signaling pathways that result in hypersensitive response and necrosis, although later succumbing to infection, while *Poncirus trifoliata* (resistant rootstock) presents a nonhost type of resistance instead, in which the plant relies on preformed biochemical and anatomical barriers rather than R- gene-based recognition of the pathogen effectors or ETI (Dalio et al., 2018).

5. Management of *Phytophthora* diseases

Preventive fungicides such as fosetyl-Al, phosphorous acid, metalaxyl and mancozeb are widely used to reduce *Phytophthora* inoculum below damage thresholds until trees are large enough to tolerate the pathogen (Farih et al., 1981; Afek and Sztejnberg, 1989; Sandler et al., 1989; Matheron and Porchas, 2002; Chi et al., 2020), but they are expensive and pose significant health and safety issues. Recently, greenhouse studies have confirmed the efficacy of new compounds in controlling citrus root rot (Hao et al., 2019). These include fluopicolide and oxathiapiprolin, which are federal and fully registered, respectively, and ethaboxam and mandipropamid, which are still in the registration process. Other commercial formulations that utilize peracetic acid and hydrogen peroxide as active ingredients have also proven effective at preventing *Phytophthora* infections in greenhouse experiments (Gurung et al., 2020). The injection of chlorine in microirrigation systems and the exposure of contaminated seeds to hot water (49 °C) for 4 to 10 min have also helped to clear early infections and prevent damping off (Savita and Nagpal, 2012). If cultural controls are inadequate to control foot rot it may be necessary to use chemical control

Table. Apoplastic and cytoplasmic effectors secreted by Phytophthora spp. with the possible contribution to pathogenicity on citrus

Effector	Type (Localization)	Pathogen/host	Functions	References
Apoplastic effectors				
INF-1	Elicitin	Most members of the genus *Phytophthora* / *Solanaceae* family, tobacco, parsley and *A. thaliana*	Induces an oxidative burst in cells through efflux of K^+ and Cl^- and influx of Ca^{2+} leading to a strong hypersensitive reaction	Fellbrich et al. (2002); Kamoun et al. (1998); Oßwald et al. (2014)
ParA1	Elicitin	*P. parasitica* / tobacco	Induces a very strong HR	Kamoun et al. (1993)
GIP1 (Glucanase Inhibitor Protein 1)	Protease inhibitors	*P. sojae* / soybean	Inhibits the soybean endoglucanase EGaseA	Rose et al. (2002)
EPI1 and EPI10	Kazal-like protease inhibitors	*P. infestans*/ tomato	Inhibit the defense protease P69B	Tian et al. (2004, 2005)
EPIC1 and EPIC2B	Cystatin-like protease inhibitors	*P. infestans*/ Solanacea family	Inhibit the cysteine proteases PIP1 (Phytophthora-inhibited protease 1), Rcr3, and the papain-like protease C14	Kaschani et al. (2010); Song et al. (2009); Tian et al. (2007)
Cytoplasmic effectors				
PSE1 (Penetration- Specific Effector 1)	RxLR	*P. parasitica* / *N. benthamiana* and *A. thaliana*	Suppresses PCD induced by the proapoptotic protein BAX (bcl-2-associated X protein);	Evangelisti et al. (2013)
			Interfers with auxin physiology during root infection	
PpRxLR2	RxLR	*P. parasitica* / *N. benthamiana*	Completely suppresses INF1-1 induced cell death	Dalio et al. (2017b)
PpRxLR3 and PpRxLR5	RxLR	*P. parasitica* / *N. benthamiana*	Partially suppress plant immunity responses (to a lesser extent as compared to PpRxLR2)	Dalio et al. (2017b)
REX2 and REX3	RxLR	*P. palmivora* / *N. benthamiana*	Promote root infection upon expression; REX3 interfers with host secretion processes	Evangelisti et al. (2017)
AVR3a	RxLR	*P. infestans* / potato	Suppresses PCD induced by INF-1 elicitin, a protein also secreted by *P. infestans* that has PAMP features and elicits cell death	Bos et al. (2009); Wawra et al. (2017)
AVRblb2	RxLR	*P. infestans* / *N. benthamiana*	Prevents C14 protease secretion and causes an accumulation of protease-loaded secretory vesicles around the haustoria	Bozkurt et al. (2011)
PsCRN70	Crinkler	*P. sojae* / *N. benthamiana*	Suppresses PCD induced by the elicitin INF-1, which acts as a PAMP inducing cell death	Rajput et al. (2014)

on young trees at the initial stages of grove establishment. The use of postplant fungicides that contain metalaxyl phosphite during the early stages of a grove's development decreases the disease incident.

The incidence of root rot and trunk gummosis in the field can be reduced by cultural practices such as annual examination of roots and removal of dead or infected trees, grafting at maximum height, avoiding trunk injuries, and the implementation of irrigation practices that minimize the contact of the trunk with water. However, since *Phytophthora* diseases are mainly transmitted by the soil, the use of resistant or tolerant rootstocks remains the most effective and sustainable prevention approach. The resistance is mainly encountered in cultivars of *P. trifoliata*, *C. medica, C. macrophylla, C. jambhiri, C. grandis, and C. macroptera*, thus representing useful genetic resources for citrus breeding programs (Hutchison, 1985). In the most tolerant rootstocks, the rate of root regeneration exceeds the pathogen's ability to infect root tissue, which compensates for the damage suffered. On the other hand, apart from some slight differences between oranges and lemons, all citrus species are susceptible to fruit brown rot, either in the field or during postharvest storage.

Phytophthora resistance/tolerance is a common target of citrus breeding programs worldwide. The first sexual hybridization programs began at the end of the 19th following destructive *Phytophthora* epidemics and resulted in the creation of many intergeneric hybrids combining characteristics of commercial citrus rootstock varieties and wide relatives. These include Carrizo, Troyer, and C35 citranges (*C. sinensis* × *P. trifoliata*), citrumelos (*C. paradisi* × *P. trifoliata*), citremons (*C. lemon*

× *P. trifoliata*), citradia (*C. aurantium* × *P. trifoliata*), citrumquat (*Fortunella spp.* × *P. trifoliata*) and Eremoradia (*Eremocitrus glauca* × *C. aurantium*) (Cimen and Yesiloglu, 2016). Recently, two citrandarins resistant to *P. nicotianae*, namely US-852 and US-812, were obtained from crosses between *C. reticulata* and *P. trifoliata* and are already in use by growers in the US (Albrecht et al., 2012).

The effectiveness of pollination programs is limited by the strong heterozygosity and apomixis that characterize citrus species (Aleza et al., 2012). Thus, many recent works have focused on the integration of biotechnological approaches such as protoplast fusion. However, the agronomic performance of cybrid rootstocks depends largely on the choice of parental combinations. Tolerance to *Phytophthora* root rot has mainly been reported in cybrids from Cleopatra mandarin

+ sour orange, sweet orange var. Caipira + volkamer lemon and sweet orange var. Caipira + Rangpur lime (Mourão Filho et al., 2008). Using a 23-kDa PR-5 protein isolated from tomato, Fagoaga et al. (2001) succeed to produce a transgenic orange line (*Citrus sinensis* L. Obs. Cv. Pineapple) that showed high tolerance to *P. citrophthora* following a detached bark assay. However, apart from a few successful examples, genetic transformation protocols still lack efficiency and depend largely on species and/or cultivars (Ballester et al., 2007). The quantitative aspect of productivity parameters and the low regeneration potential of commercial cultivars represent major limitations for the adoption of such technology (Peña and Navarro, 2012).

The universal use of molecular markers and high-throughput screening techniques has provided a better understanding of the genetic basis of citrus resistance to *Phytophthora*. Several QTLs associated to resistance traits have been identified, which yielded high-resolution genetic association maps and paved the way for marker-assisted selection as a future approach for developing *Phytophthora*-resistant rootstocks (Siviero et al., 2006; Lima et al., 2018) (Figure 3).

6. Biochemical mode of resistance

Salicylic acid (SA) is a phytohormone that not only regulates many important physiological functions of the plant but also plays a vital role in the activation of defense responses. The phenylalanine ammonia-lyase (PAL) pathway is involved in SA biosynthesis and results in high-level production of this plant hormone (Glazebrook 2001). SA defense activity is majorly governed by NPR1 protein while its interaction with TGA and WRKY gene (transcription factor) results in the activation of systemic acquired resistance (SAR), which is a comprehensive immune response that provides durable resistance and induces resistance (R) genes (Vlot et al., 2009; Dempsey et al., 2011; Diaz-Puentes, 2012). Thus, PR genes confer greater resistance (Vlot et al. 2009) in addition to being involved in lignin and suberin synthesis. Moreover, following the infection, tissue lignification is initiated in the plant by the peroxidase (POX) enzyme that offers a physical barrier against pathogens by incrementing lignin polymerization (Resende et al., 2003).

P. trifoliata and *C. sunki* grafted onto *Citrus limonia* Osb. rootstock was evaluated for response to *P. nicotianae* infection along with other PR-related genes and lipoxygenase (LOX) responsible for plant defense (Boava et al., 2011). The results exhibited that peroxidase and lipoxygenase levels were higher in resistant rootstocks at the later stage of infection in comparison with susceptible rootstocks. These genes can be used as candidate genes for the breeding of citrus resistance against *P. nicotianae*. In addition, LOX gene was also found responsible to produce jasmonic acid (JA) and reactive oxygen species (ROS) along with induction of hypersensitive response (HR) (Pieterse et al., 2009; Lyons et al., 2013).

Based on the changes in gene expression profiles during the interaction between pathogens and citrus, a molecular model exhibiting triplicate resistance mechanisms can be

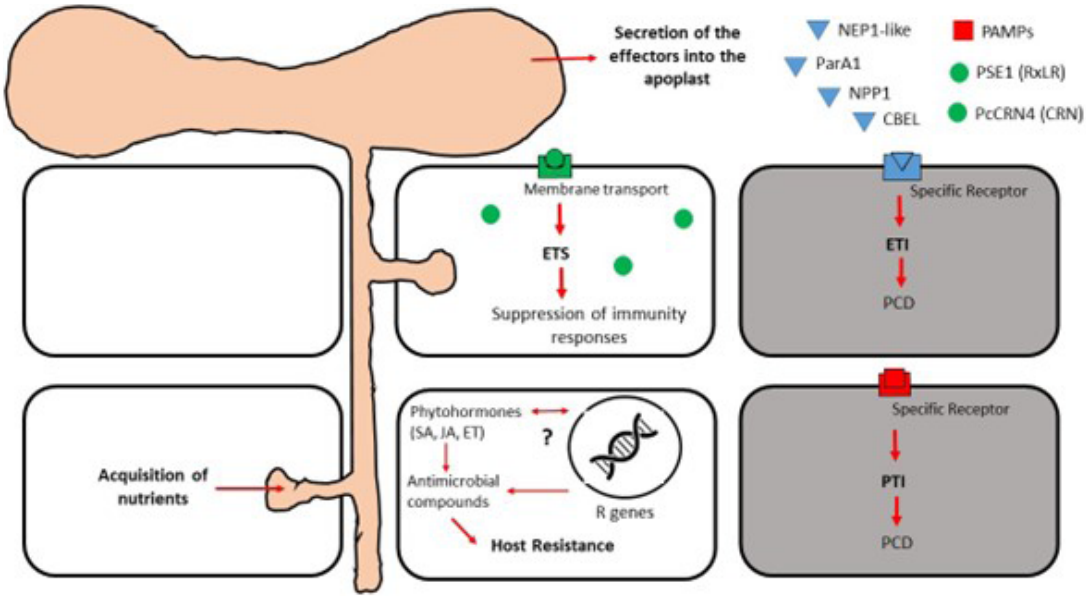

Figure 3. Summary of molecular interactions between Phytophthora pathogens and citrus hosts. The pathogen acquires nutrients from host cells through the haustoria and secretes apoplastic and cytoplasmic effectors into the apoplast through the appressoria. Apoplastic effectors (△) inhibit enzyme activity in the apoplast, while cytoplasmic effectors (O), including RxLR and Crinkler (CRN), translocate into the cells using the host's machinery where they interfere with cell immunity responses. Upon recognition of PAMPs (□) or apoplastic effectors, host cells activate their defense systems leading to programmed cell death (PCD). R-genes, phytohormones, PAMP receptors, and effector-specific receptors all contribute to host and nonhost resistance of citrus against Phytophthora infections.

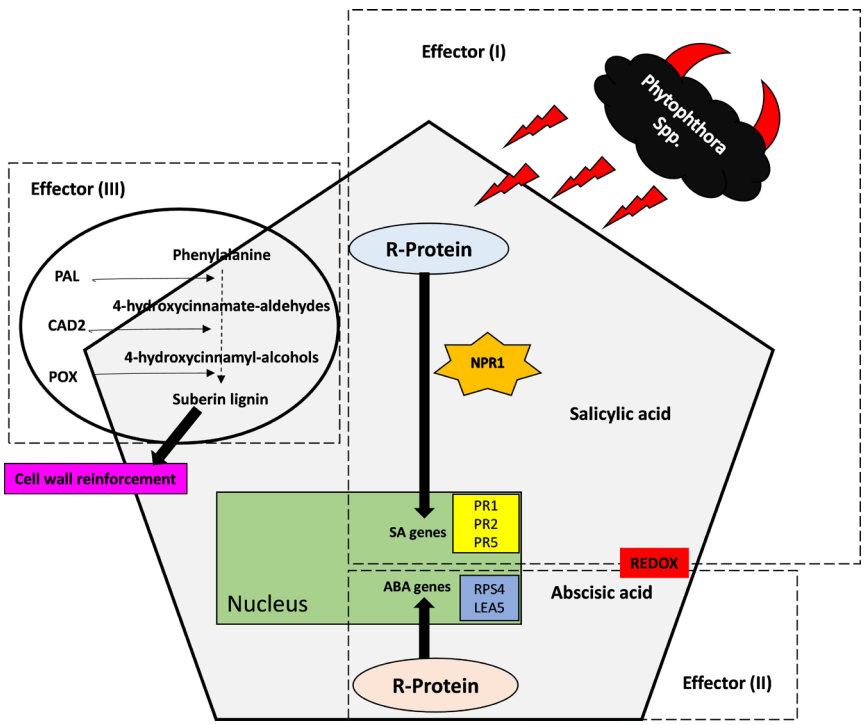

Figure 4. Proposed molecular model of the plant defense mechanisms in the Phytophthora-citrus interaction. **I.** Effectors interact with R proteins encoded by SA genes, triggering the interaction with NPR1 protein which activates the accumulation of salicylic acid (SA), causing a change in the redox potential of the cell. **II.** Other effectors interact with R proteins encoded by RPS4 and LEA5, which are responsive to the accumulation of abscisic acid (ABA), which also causes a change in the redox potential of the cell. **III.** PAL, CAD2 and POX are enzymes involved in the synthesis which are precursors of lignin and suberin which results in cell wall reinforcement.

established. Following the attack, pathogenic oomycetes release effector molecules that interact with resistant R protein and trigger NPR1 signaling, thus activating the accumulation of SA to produce a defense response. The SA alters the redox potential of cells in the cytoplasm and induces the expression of pathogenicity-related genes i.e. PR1, PR2, and PR5 known for encoding cell wall and membrane degrading enzymes. At the same time, other effectors are also recognized by cytoplasmic R proteins i.e. LEA5 and RPS4 which respond against ABA accumulation and generate defense responses through changes in cellular redox potential. Like other proteins, PAL, 2 cinnamyl alcohol dehydrogenase (CAD2) and acne activate lignin synthesis precursors of phenolic compounds and suberin, leading to an important final stage of the defense reaction dependent on acne (Figure 4).

7. Conclusion

Several species of *Phytophthora* are known to cause soil-borne diseases that pose serious threats to citrus plantations around the world. High-throughput sequencing technologies unveiled several classes of effector proteins secreted by *Phytophthora sp*. responsible for causing disease. On the other hand, the identification of genes responsible for governing the defense system in this plant-pathogen interaction has revealed the gene expression profiles of different phenotypes either susceptible or resistant and their variant hybrids in response to disease. Thus, while developing the new management strategies, knowledge of resistance mechanisms controlling molecular interactions between citrus and pathogens will be worthful. So far, the use of disease-resistant rootstocks remains one of the most effective and sustainable methods to prevent *Phytophthora* diseases while reducing the use of harmful chemicals. The upcoming molecular technologies and high-throughput biochemical analyzes will provide more in-depth details on *Phytophthora*-citrus interactions, thus providing a better understanding of the pathosystem and unraveling more precise options for breeding schemes oriented towards the control of *Phytophthora* diseases in citrus.

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