



Review

Signaling of Plant Defense Mediated by Receptor-like Kinases, Receptor-like Cytoplasmic Protein Kinases and MAPKs Triggered by Fungal Chitin in Horticultural Crops

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Abstract: Fresh horticultural products are economically significant foods that are highly demanded by consumers worldwide; however, they are highly perishable and susceptible to deterioration by fungi, which contribute to their short shelf-life and cause significant post-harvest losses. Among the alternatives suggested for fungal control in plants is the elicitation of the innate plant defense mechanism, which may be activated when specific molecules of the phytopathogen, such as chitin, are recognized. Chitin is a long-chain polymer of N-acetyl- α -D-glucosamine of the fungal cell wall; it possesses biological activity by eliciting the plant immune response. This molecule and its oligosaccharides are recognized through transmembrane receptors known as receptor-like kinases (RLKs) and receptor-like proteins (RLPs). Mediated by receptor-like cytoplasmic kinases (RLCKs), which bind to the intracellular domain of these receptors, they initiate intracellular signal transduction via MAP kinases, triggering the plant defense response. In model plants, such as *Oryza sativa* (rice) and *Arabidopsis thaliana*, the set of RLK/RLP-RLCK-MAP kinases is involved in plant immunity triggered by chitin. Furthermore, in horticultural products, research into the molecular events between these three elements has suggested that similar processes occur. However, little is known about these molecular events in fruits. Against this background, the present review provides the most recent and relevant findings on the molecular associations of these three elements in the response to fungal chitin in plants and outlines which elements could participate in this signaling process in horticultural crops.

Keywords: fungal chitin oligosaccharides; receptor-like cytoplasmic kinase; MAPK activation; signaling transduction; defense response; horticultural crops



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1. Introduction

Fresh horticultural products are in high demand worldwide due to their organoleptic characteristics and energy, mineral, vitamin, fiber, and bioactive compound contents. Regular consumption of fruits and vegetables can help alleviate malnutrition problems and contribute to lowering the risk of illnesses such as cardiovascular diseases, diabetes, and cancer [1,2]. However, they are highly perishable foods and are susceptible to deterioration caused by several necrotrophic fungi, including *Rhizopus nigricans* Ehrenb [3], *Fusarium oxysporum* [4], *Botrytis cinerea*, *Colletotrichum gloeosporioides* [5], and *Alternaria alternata* [6,7].

One strategy proposed to avoid the use of traditional fungicides for the prevention and control of fungal attacks is the induction of the innate defense mechanism, which can be activated by biological elicitors, such as chitin. The chitin molecule is a complex polymer

found in the cell walls of fungi, which, in addition to its structural functions, has been shown to have biological activities in plants [8]. Previous studies reported in the literature have shown that chitin and its oligosaccharides, considered pathogen-associated molecular patterns (PAMPs), are perceived by the plant through pattern recognition receptors (PRRs). These PRRs are found in the plasma membrane (Figure 1). The PAMP recognition induces the activation of plant defense reactions known as PAMP-triggered immunity (PTI) [9,10]. Once PAMPs are recognized, intracellular signal transduction occurs via receptor-like cytoplasmic kinase (RLCK), which binds to the intracellular region of RLK proteins [11,12]. This intracellular signal transduction usually implies a phosphorylation cascade that stimulates mitogen-activated protein kinases (MAPKs), the generation of reactive oxygen species (ROS), and calcium influx [11,13,14]. In turn, these processes perform a transcriptional reprogramming role. They also lead to the upregulation of defense genes encoding PRs, such as glucanases, chitinases, defensins, and peroxidases. Additionally, specific proteins, including phenylalanine ammonia-lyase, involved in the production of phytoalexins, are also upregulated [15–18]. All of these molecular events can lead to a successful plant defense mechanism, protecting plants against pathogen attacks.

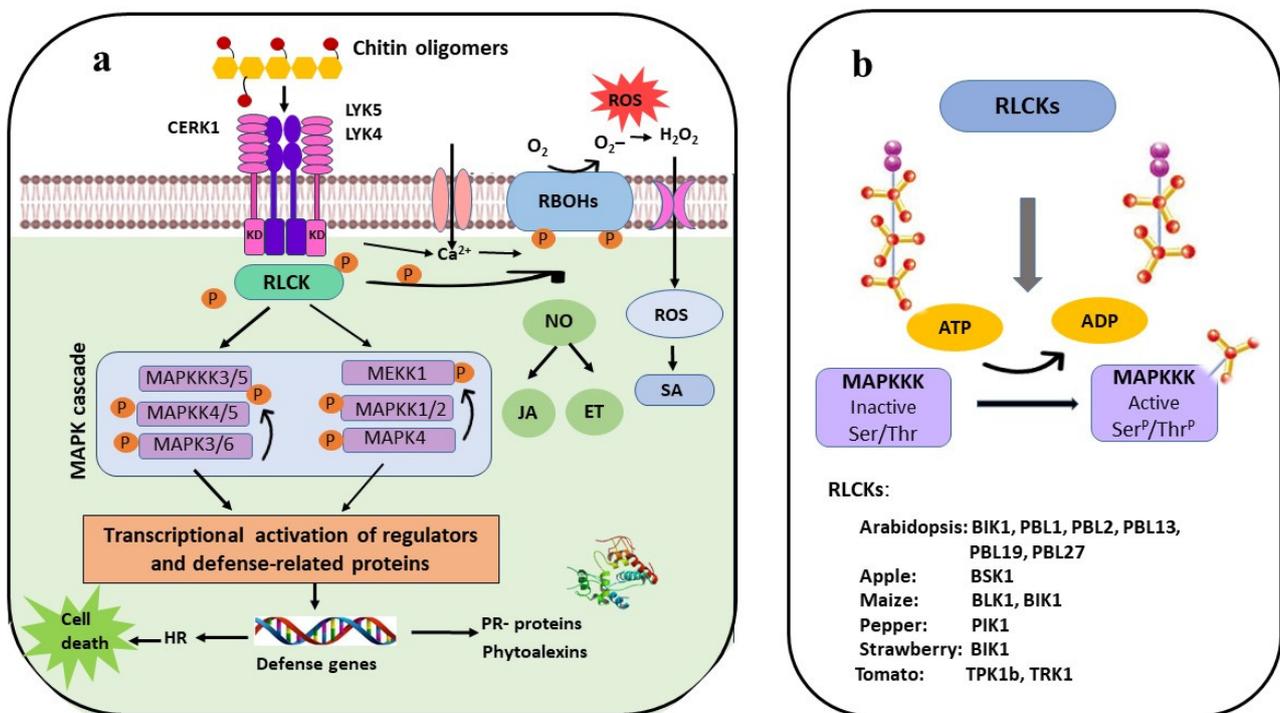


Figure 1. Overview of the plant immune response activated by the recognition of chitin oligomers. (a) After exposure to chitin oligomers, the RLKs are activated by the union of chitin oligomers on their extracellular side and they become phosphorylated and activate the influx of calcium and the formation of reactive oxygen species. In turn, the RLCKs are activated; these become phosphorylated and activate the synthesis of signaling molecules ethylene (ET), jasmonic acid (JA), and salicylic acid (SA), as well as the MAP kinase cascade. These events cause the activation of transcription factors that promote the increase in the expression of defense genes. (b) Possible mechanism of phosphorylation of the serine and threonine motifs of the MAPKKK, promoted by RLCK proteins in horticultural crops.

Knowledge about plant–pathogen interactions provides the basis for understanding how the defense mechanisms function in fruits. The plant’s defense mechanisms are elicited by different pathways depending on whether the pathogen is necrotrophic or biotrophic [19]. Necrotrophic pathogens take the nutrients from dead cells while biotrophic pathogens feed on living plant cells, and hemibiotrophs take the nutrients from living

plant tissues before switching to a necrotrophic stage. To prevent biotrophic growth, plant immunity is activated by intracellular receptors that recognize specific effectors and perform a hypersensitive cell-death response, but this hypersensitive response does not prevent the growth of necrotrophic pathogens [20]. On the other hand, resistance to necrotrophic pathogens is mediated by gene-for-gene pattern recognition by plasma membrane receptors, and regulators of response signaling, transcriptional regulators and co-regulators, have been implicated [21]. These defense mechanisms are based on prevention, tolerance, or resistance to pathogen attack and can be constitutive or inducible [5]. Hence, an induced plant defense mechanism could stop or attenuate pathogen disease and protect the plants against future attacks. Chitin receptors, RLCKs, and MAP kinases are pivotal elements that enable the plant immune response and, significantly, its outcome when in contact with a fungus or chitin oligosaccharides. On that basis, this review provides the latest and most relevant findings on the roles of these three elements in horticultural crops' responses to chitin oligosaccharides.

2. Chitin as an Inducer of the Defense Mechanism

Plants have constructed complicated defense systems that allow them to detect pathogens and initiate a series of reactions that stop or limit pathogen attacks. Fruits have limited defense mechanisms, which vary depending on their maturity stage [5,22]. As fruits mature, they undergo the highly complex and ordered processes of ripening and senescence, which involve physiological and biochemical reactions that shape the fruit quality. These reactions trigger the reprogramming of multiple ripening-associated genes, triggering changes in texture, flavor, color, aroma, and other quality attributes [23]. There are also reductions in the concentrations of phytoalexins and phytoanticipins, decreases in the inducible defense responses of the fruit, changes in the composition and biosynthesis of the cuticle, and changes in the pH of the fruit, increasing its susceptibility to microbial deterioration [24–26]. Ripe fleshy fruits generally present a higher susceptibility than immature fruits to post-harvest diseases and decay [27,28]. Research efforts have thus been directed toward understanding the events during fruit ripening that contribute to high susceptibility to pathogens. This understanding informs strategies aimed at managing these events to increase fruits' resistance to pathogens and thereby boost the production of high-quality fruit at the appropriate maturity stages.

The defense mechanisms of the plant are very complex and are genetically controlled. These mechanisms are activated once a pathogen spore encounters the plant surface. The cell wall of the fungus is mainly composed of β -glucans, glycoproteins, and chitin [29], with the latter being one of the main pathogen-associated molecular patterns (PAMPs) present. From that moment, the molecules of the fungi cell wall are detected by receptors located in the plasma membrane that interact with PAMPs. According to some authors [10,30], chitinolytic enzymes secreted by plants break down the fungal cell wall, leading to the disruption of cell integrity and the release of small chitin oligosaccharides with a degree of polymerization of 6 to 8. The binding of these molecules to plasma membrane receptors provokes a defense response, known as PTI (PAMP-triggered immunity) [31]. When this is triggered, these membrane receptors activate signal transduction to the nucleus, activating genes responsible for the pathogen response.

The cell wall of the fungus is mainly composed of β -glucans, glycoproteins, and chitin [29], with the latter being one of the main PAMPs present. This molecule is a biopolymer of repeating monomers of N-acetyl- α -D-glucosamine (GlcNAc), joined by glycosidic linkage of the β -(1→4) type [8]. It is an insoluble polymer when it is extracted from shrimp shells and other crustaceans via alkali treatment, and it is partially soluble when extracted via enzymes. In the latter approach, certain molecules that hold significant biological importance are produced, such as chitosan and chitin oligosaccharides [32]. Additionally, chitin has been approved for use as a food additive by the United States Food and Drug Administration [33].

Some studies have reported the inducer capacity of the plant defense mechanisms presented by chitin. For instance, chitin isolated from the yeast *Rhodotorula mucilaginosa* diminished the post-harvest rot of peach fruits caused by *Rhizopus* sp by up to 50%. At the same time, compared to control fruits, a higher differential expression of several genes encoding proteins was noted, including NADH dehydrogenases, glycosidases, hydrolases, and protein kinases possessing a serine/threonine (Ser/Thr) domain, which are essential in the immune mechanisms against pathogens [34]. An increase in the enzymatic activities of polyphenol oxidase and peroxidase was documented in pear fruits exposed to colloidal chitin, and a significant reduction in the disease severity of blue mold caused by *Penicillium expansum* was also observed [35]. Tomato fruits treated with chitin from the yeast *Saccharomyces cerevisiae* also demonstrated an increase in the expression of genes encoding enzymes associated with oxidative metabolism, such as peroxidase, catalase, and superoxide dismutase. Additionally, genes associated with the response to fungal disease including chitinases were shown to be upregulated. Similarly, a significant decrease in rot caused by *B. cinerea* was observed [36]. Recently, it was reported that chitin oligomers isolated from the fungus *A. alternata* triggered a notable rise in chitinase and glucanase activity in tomato fruit, which diminished the progression of black rot disease caused by *A. alternata* by up to 78%. In that study, it was observed that the fungal chitin oligomers induced an increase in the level of enzymatic activity starting 30 min after the chitin treatment [37].

These findings demonstrate that the presence of chitin triggers a series of defense responses in fruits to fight against fungal attacks. These responses involve complex biochemical processes serving to prevent the fungal pathogen from being established and colonizing.

3. Plant Membrane Receptors That Perceive Chitin and Fungal Chitin Oligosaccharides

PAMP recognition, including that of chitin oligosaccharides, occurs through receptors embedded in the plasma membrane of the plant cell. To our knowledge, two types of chitin receptors have been studied in plants: RLKs and RLPs. These have been studied mainly in two plant models: *Oryza sativa* (rice) and *Arabidopsis thaliana*.

RLKs are involved in signal transduction and consist of an intracellular region containing a juxtamembrane (JM) domain, a C-terminal domain, and a kinase domain. Additionally, they possess a transmembrane region, and outside the plasma membrane, an extracellular region with a sensor domain [38]. They are categorized into subfamilies based on the lysin domain count in their extracellular area. Meanwhile, RLPs are like RLKs but do not possess an intracellular domain. In certain instances, RLPs are affixed to the plasma membrane via either a transmembrane domain found in specific proteins or through an anchor containing glycosyl-phosphatidyl-inositol [39,40].

In *Arabidopsis*, CERK1 (chitin elicitor receptor kinase 1) has been the most studied. It was observed that CERK1 in *Arabidopsis*, is crucial for chitin perception, which occurs via the lysin domains on its extracellular side [41]. Additionally, they highlight the importance of CERK1 in contributing to the kinase activity required for phosphorylation, initiation of early defense mechanisms, and the downstream signaling cascade. More recently, it was found that, besides CERK1, other receptor proteins are also essential for recognizing chitin oligosaccharides, such as the lysin-motif-containing receptor-like kinases (LysM-RLKs). In *Arabidopsis*, five LysM-RLKs (AtLYK1 to AtLYK5) have been reported, some of which are involved in chitin elicitor signaling. For example, the complex AtCERK1/LYK1 has been shown to have a crucial function in recognizing bacterial peptidoglycan (PGN) [42] and chitin signaling [43,44]. Furthermore, it has been suggested that AtLYK5 or AtLYK4, which have inactive kinase domains, could serve as the main receptors for chitin. Additionally, the presence of chitin could potentially result in the creation of an AtLYK5-AtLYK1 heterotetramer [45]. When Erwig and colleagues [46] investigated two chitin receptors, CERK1 and LYK5, in *Arabidopsis*, both implicated in chitin signaling, the authors found that CERK1 was autophosphorylated upon chitin binding and triggered LYK5 phosphorylation after this process, thereby promoting its internalization into mobile intracellular vesicles. Because LYK5 (or LYK4) does not have active kinase domains, only CERK1, which acts

as a co-receptor, can activate the phosphorylation process and initiate downstream signal transduction [47].

In rice, the initial chitin receptor identified was the chitin elicitor-binding receptor-like protein, known as CEBiP (chitin oligosaccharide elicitor-binding protein). This receptor is a member of the RLP family and features a lysin motif (LysM) [48]. This protein has an approximate molecular weight of 75 kDa and possesses numerous glycosylation sites where the receptor can bind to GlcNac. According to Hayafune and colleagues [49], the pivotal lysin motif in CEBiP is essential for recognizing chitin oligosaccharides with a degree of polymerization of 8. Then, CEBiP dimerizes with the OsCERK1 protein to facilitate the linkage to these chitin oligosaccharides.

Similar studies report the presence of chitin receptors in horticultural crops. For instance, MdCERK1 was reported in apple (*Malus domestica*) exposed to chitin treatment and infection with *Rhizoctonia solani* [50]. The authors found that MdCERK1 functions predominantly in the vegetative tissues of both the leaf and root, as opposed to the apple's flower, fruit, and seed. It was found that this receptor is located in the plasma membrane. Its extracellular region contains three lysin motifs, followed by a transmembrane region, and an intracellular side featuring serine/threonine (Ser/Th) motifs [50,51]. Meanwhile, in bananas (*Musa acuminata*), the plasma membrane receptor-like kinase harboring a lysin motif, MaLYK1, was classified with a subclade that includes AtCERK1 and OsCERK1. Banana roots exhibited elevated levels of MaLYK1 expression, whereas its presence was diminished in the yellow fruit. Furthermore, it was observed that the expression of MaLYK1 was triggered by *F. oxysporum* sp cubense race 4 (Foc4). Therefore, its role is crucial not just in fungus recognition but also in resisting Foc4 and in contributing to the symbiotic connection formed between mycorrhiza and plants [52].

In grapes (*Vitis vinifera*), it has been documented that genes responsible for two counterparts of AtCERK1/LYK1, namely VvLYK1-1 and VvLYK1-2, play a role in chitin oligomer signaling [53]. Furthermore, there have been recent reports on two AtLYK5 orthologs identified in a grapevine, namely VvLYK5-1 and VvLYK5-2. The up-regulation of VvLYK5-1 in the leaves was noted as a result of an attack by both *Erysiphe necator* and *Coniella diplodiella*, demonstrating the involvement of VvLYK5-1 in chitin perception in *V. vinifera*. In addition, the authors observed the absence of the majority of the amino acids required (Ser/Th) for kinase activity in VvLYK5-1 and VvLYK5-2, similar to AtLYK5. This implies that the functional kinase domain is lacking in VvLYK5-1 and VvLYK5-2. As a result, it is prompted that VvLYK5 may interact with a co-receptor to transmit signals, ultimately triggering chitin-induced immunity [54].

In tomato plants (*Solanum lycopersicum*), it was observed that the AvrPtoB protein of *Pseudomonas syringae*, which blocks PTI, interacts with Bti9. The latter is an RLK protein with 76% similarity with AtCERK1, which shares a clade with SILYK11, SILYK12, and SILYK13. This discovery implies the potential involvement of these proteins in responding to PAMPs, which triggers the signaling cascade [55]. Elsewhere, another study established a connection between CERK1 homologous receptors (SILYK 1, SILYK12, and SILYK13) in tomato plants and the establishment of arbuscular mycorrhiza. In transformed plants with the SILYK12 gene silenced, the colonization of mycorrhiza was affected, and the absence of SILYK1 reduced the response to chitin. Alternatively, the overexpression of SILYK1 (or SILYK13) induced cellular death. These results reveal the relationship of these receptors with the arbuscular mycorrhizal colonization process during symbiosis with tomato and the role they play in plant immunity induced by PAMPs [56]. Furthermore, these findings suggest that chitin receptor genes exhibit distinct functions in plants based on the specific plant tissue or PAMP applied.

In summary, it can be observed that both *A. thaliana* and *O. sativa* exhibit a set of receptors displaying strong binding to chitin oligomers. The molecular events that take place upon this recognition phenomenon have been thoroughly described. Furthermore, these receptors have been identified in specific horticultural plants, maintaining similar structural patterns. However, in fruits, there is limited information on this group of

receptors. Our current knowledge is drawn from the few relevant studies to date. In a particular experiment, researchers utilized CRISPR/Cas9 technology to create mutants of the tomato chitin receptor in tomato fruit. This investigation revealed that SLYK4 exhibited high expression levels in tomato fruit and could be stimulated through CaCl_2 addition and low-temperature treatment. The increased expression of SLYK4 led to increased defense mechanisms, providing enhanced protection for tomato fruits against *B. cinerea* disease [57]. In another study, RNA sequencing analysis was conducted on tomato fruit treated with chitin oligomers from *A. alternata*. This study revealed the heightened expression of genes responsible for chitin receptors, including *SICERK1*, *SILYK1*, *SILYK4*, *SILYK7*, *LRR*, and *RLKs*. The over-expression of these genes occurred within 30 min of chitin treatment, indicating the early perception and response of the fruit to the chitin oligomers from *A. alternata* [58]. Furthermore, it was found that the *Alternaria* chitin oligomers induced an increase in the level of enzymatic activity of chitinase and glucanase 30 min after treatment and reduced the black rot development caused by the *A. alternata* fungus, with an efficacy of up to 78% in tomato fruit [37]. However, the study did not ascertain which receptor played the primary role in perceiving *Alternaria* chitin oligomers for initiating signal transduction.

Once chitin oligomers are perceived, it seems that phosphorylation of the chitin receptor can be rapidly induced and activated. This facilitates its interaction with other receptors, including receptor-like cytoplasmic kinases, subsequently triggering defense responses [47].

4. Receptor-Like Cytoplasmic Kinase (RLCK) Serving as a Signal Carrier

In some model plants, reports have indicated the presence of receptor-like cytoplasmic kinases (RLCKs) that interact with the intracellular domains of chitin receptors, initiating signaling cascades. RLCKs are a subfamily belonging to the RLK superfamily, and many of them are affixed to the plasma membrane via N-myristoylation or palmitoylation. According to sequence homology, RLCKs are categorized into 17 subsets denoted as RLCK-II and RLCK-IV-RLCK-XIX. Despite lacking an extracellular or transmembrane domain, RLCKs possess a serine and threonine protein kinase intracellular domain. This domain consists of 11 kinase subdomains (I to XI), which are highly preserved, in addition to a small C-end region and a brief N-end region [59,60]. The serine and threonine (Ser/Thr) motifs of the intracellular domain of RLCK proteins have been shown to be essential in interacting with these receptors [40]. Furthermore, RLCKs are essential contributors to diverse plant processes [61], including the immune responses in certain plant species [62], such as *Arabidopsis*, apple, maize, pepper, rice, and tomato [63–68]. Specifically, the RLCK VII subfamily is predominantly associated with positively regulating PTI signaling in these plants. Recent findings indicate that rice, *Arabidopsis*, and maize possess 379, 149, and 192 RLCKs, respectively [59,65].

In *Arabidopsis* leaves, Botrytis-induced kinase 1 (BIK1, an RLCK with the Ser/Thr kinase domain) confers protection against *B. cinerea* through interaction with CERK1 [63]. In addition, a mutation of the *bik1* gene that causes the loss of function of the BIK1 protein has been associated with an increase in necrosis and chlorosis processes in mutant plants. Moreover, it was shown that this mutation causes increased susceptibility to a second opportunistic pathogen, such as *A. brassicicola*. This affirms the pivotal role of BIK1 in triggering the defense mechanism against necrotrophic fungi. In another study, Liu and colleagues [69] evaluated the interplay between brassinosteroid-insensitive 1-associated receptor kinase 1 (BAK1) and the BIK1 binding protein during a *bak1-bik1* double-mutant study performed on *Arabidopsis*. In their investigations, this genotype showed a phenomenon known as dwarfism, suggesting that this interaction of BAK1 with BIK1 is essential in regulating plant immunity and cell death. When exploring this, it was found that BIK1 assumes the kinase function in the defense response via RLK. It also exhibits a negative regulatory function on the RLP23 protein by competing to interact with BAK1, thereby impeding processes of accelerated maturation and necrosis [70]. These findings demonstrate that

BIK1 is involved in multiple processes in *Arabidopsis* and stands as an essential regulatory element for plant growth given its multiple functions within the immune system.

Another RLCK investigated in *Arabidopsis* was PBL27 [71]. The authors found that PBL27 binds to CERK1 in the presence of chitin oligomers, and, in doing so, activates MAPK3/6 signaling (Table 1). In another study carried out on *Arabidopsis*, it was observed that signaling via MAP kinases could be interrupted by silencing the gene encoding for the PBL27 protein (which binds to the CERK1) [72]. These studies reveal that these RLCKs, which interact with the intracellular domains of chitin receptors, constitute a fundamental element of the signaling cascade. In a similar study performed on *Arabidopsis*, it was observed that the CERK1-LYK5-PBL27 module was activated in the presence of fungal chitin, and PBL27 phosphorylated the SLAH3 anion channel, thereby inducing stomatal closure in order to prevent the entry of fungal pathogens [73]. Furthermore, another study carried out in *Arabidopsis* indicated that chitin induces oligomerization between AtLYK5 and AtCERK1, leading to the subsequent phosphorylation and activation of AtCERK1. After that, this complex phosphorylates and activates the RLCKs PBL19/27 and BIK1. BIK1 then phosphorylates and activates CNGC2/4 (the cyclic nucleotide-gated channel), which regulates the influx of Ca²⁺ and activates respiratory burst oxidase homologue protein D (RBOHD), thereby triggering the sudden generation of reactive oxygen species (ROS). PBL19 and PBL27 phosphorylate MAPKKK5 (MAPK kinase kinase 5), launching the consecutive activation of MAPK (mitogen-activated protein kinase) chain reactions. These reactions subsequently activate genes encoding defense proteins in the nucleus [74] (Figure 1).

Table 1. Receptor-like cytoplasmic kinases (RLCKs) activating by receptor-like kinases (RLKs) that trigger immunity in plants including horticultural crops.

RLCK ^a	Associated RLK Receptors	Species	Elicitor	RLCK Function	References
PBL27	CERK1	<i>Arabidopsis</i>	Chitin oligomers	Pattern-triggered immunity	[71,72]
BIK1	EFR/BAK1	<i>Arabidopsis</i>	<i>Pseudomonas syringae</i>	Regulation of JA and SA levels (Interacts with WRKY in the nucleus)	[75]
PBL27	CERK1-LYK5	<i>Arabidopsis</i>	Fungal chitin	Stomatal-mediated immunity	[73]
PORK1-2	CERK1	<i>Arabidopsis</i>	<i>P. syringae</i>	Increased ROS production	[76]
BSK1	FLS2	<i>Arabidopsis</i>	<i>P. syringae</i>	Activation of MAP kinases in the presence of flg22	[77]
ZED1/PBL	ZAR1	<i>Arabidopsis</i>	<i>P. syringae</i>	They act by forming a complex as part of the defense response against <i>P. syringae</i>	[78]
BIK1	OSCA1.3	<i>Arabidopsis</i>	Synthetic flg22	Enables stomatal closure during the immune response to bacterial flagellin	[79]
PBL27	HAK1	<i>Arabidopsis/Glycine max</i>	<i>Spodoptera litura</i>	Defense response against <i>Spodoptera litura</i>	[80]
BIK1	FLS2/BAK1	<i>Arabidopsis</i>	<i>P. syringae</i>	Stomatal-mediated immunity	[81]
FvBIK1	FvBAK1	<i>Fragaria vesca</i>	Brassinosteroid	Provides immunity to the necrotrophic fungus <i>B. cinerea</i>	[82]
BSR1	CERK1	<i>Saccharum officinarum</i>	<i>Sporisorium scitamineum</i>	Transgenics that overexpress BSR1 are resistant to <i>Sporisorium scitamineum</i>	[83]
TPK1b	PORK1	<i>Solanum lycopersicum</i>	<i>B. cinerea</i>	Regulates the production of jasmonic acid in the systemin-mediated defense response.	[84]
TRK1	SILYK1	<i>S. lycopersicum</i>	Commercial chitin	Induces fungal resistance and ROS production in the presence of chitin	[12]
BSR1	Unknown	<i>S. lycopersicum</i>	<i>Rhizoctonia solani</i>	Transgenics that overexpress BSR1 are resistant to <i>Rhizoctonia solani</i>	[83]
OsRLCK118	Unknown -	<i>O. sativa</i>	<i>Xanthomonas oryzae</i>	Silencing of OsRLCK118 increases susceptibility to <i>Xanthomonas oryzae</i>	[85]
BSR1	PEPR1/PEPR2	<i>O. sativa</i>	<i>Mythimna loreyi</i>	BSR1 mediates the activation of defense responses against chewing herbivores in monocot rice	[60]
BIK1	Unknown	<i>Zea mays</i>	<i>Clavibacter michiganensis</i> subsp. <i>nebraskensis</i>	Defense response against pathogenic bacteria	[65]

^a RLCK: receptor-like cytoplasmic kinase.

In rice, OsRCLK185 is a cytosolic protein activated by OsCERK1 through its intracellular region. Recent research suggests that the activation of OsRCLK185 also depends on OsDRE2a, a protein homologous to the yeast Dre2 protein. OsDre2 is an analogue of human Anamorsin, and its interaction with OsRCLK185 relies on the cysteine residues present in the Fe-S clusters of the CIAPIN 1 (cytokine-induced apoptosis inducer 1) domain [86].

Although signaling functions of RLCKs in concert with RLKs have been well studied in *Arabidopsis* and rice, few studies have yet reported the participation of receptor-like cytoplasmic protein kinases in the defense responses triggered against fungal attack in horticultural crops. In one contribution, it was reported that in apple (*Malus domestica*), the RLCK MdBSK1 (BR-signaling kinase 1) engages with MdMAPKKK1, influencing the resistance to *Botryosphaeria dothidea*. MAPKKK serves as the initial mitogen-activated protein kinase of the MAPK cascade, detecting pathogenic signals recognized by PRRs. In turn, it triggers the MAPK cascade via a sequence of phosphorylation events in response to these signals. Decreased expression of *MdMAPKKK1* leads to the increased susceptibility of fruits to *B. dothidea* infection, resulting in increased lesion development. The study's results indicate that the activation of MdBSK1-induced phosphorylation of MdMAPKKK1 plays an essential role in conferring protection against *B. dothidea* [64]. In pepper plants (*Capsicum annuum* L.), the RLCK pathogen-induced protein kinase termed CaPIK1 has been reported. The study revealed that the *CaPIK1* gene exhibited overexpression in pepper leaves upon infection with *Xanthomonas campestris* pv. *vesicatoria*. This up-regulation led to immune responses, including bursts of ROS and NO (nitric oxide), as well as callose deposition and cell death resembling a hypersensitive response (HR) [87]. In a subsequent investigation, it was found that CaPIK1 interacted with chitinase IV (CaChitIV), which possesses a chitin-binding domain [88], and the complex formed by CaChitIV and CaPIK1 was primarily localized in the cytoplasm and plasma membrane. In addition, it was observed that the protein CaChitIV functioned as an inducer of defense mechanisms and cell death triggered by CaPIK1. In this sense, it is possible to propose that the interaction between CaChitIV and CaPIK1 serves as a positive regulatory mechanism for CaPIK1-triggered defense responses and cell death [66].

Similar studies were performed in tomato plants, where it was reported that TPK1b (Tomato Protein Kinase 1b) is an RLCK that binds to the intracellular domains of chitin receptors (RLKs). It is an ortholog of the APK1b protein reported in *Arabidopsis*. It was observed that TPK1b is transcriptionally regulated by abiotic and biotic agents, suggesting its role in plant defense mechanisms. Furthermore, in tomato leaves, it was reported that the gene encoding the TPK1b protein may be an ortholog of the *Arabidopsis bik 1* gene, which is crucial in the defense mechanism of *Arabidopsis* against *B. cinerea*. The deduced amino acid sequence of TPK1b showed 60% similarity with that deduced for BIK1. TPK1b transcripts were abundant in the leaves and, to a lesser extent, were found in buds, while in fruits, the number of transcripts was not reported. The researchers asserted that TPK1b belongs to the RLK family with a consensus motif MGXXS/T(K) N-myr (the addition of a myristoyl lipid group at the N-terminal glycine) and has a catalytic domain between 68 and 353 amino acid residues, a Ser/Thr kinase active site, and an ATP binding site. TPK1b was also linked to the ethylene-mediated response. Lastly, from a functional standpoint, an increase in the susceptibility of tomato plants to *B. cinerea* was noted in transformants produced through RNAi engineering to have reduced TPK1b gene expression [68]. Elsewhere in the literature, it was shown that TPK1b interacted with PEPR1/2 ortholog receptor-like kinase1 (PORK1) within the systemin-mediated defense mechanism, and TPK1b was phosphorylated by PORK1 [84]. Furthermore, it was observed that the silencing of PORK1 resulted in a proportional decrease in the levels of TPK1b phosphorylation. This reduction in phosphorylation levels had a notable impact on the defense mechanism mediated by this pathway. This reaffirms that TPK1b plays an essential role in collaboration with RLKs in signal transduction and plant immune responses.

In another study, Jaiswal et al. [12] identified a protein kinase related to TPK1b, which was termed TRK1 (tomato protein kinase 1b-related kinase), and determined its functions

through quantitative resistance and plant growth investigations in tomato plants. In their study, tomato plant *TRK1* RNAi lines with diminished expression showed increased susceptibility to fungal infection caused by *B. cinerea*. *TRK1* is a classic RLCK characterized by a kinase catalytic domain spanning residue 92-369. This domain comprises a protein kinase ATP binding motif and a substrate binding site. Based on phylogenetic results, the *TRK1* receptor is most closely related to the RLCK VII group of RLCKs found in *Arabidopsis*, such as AtPBL19, AtPBL37, and AtPBL38 [12]. Previous studies have implicated these RLCKs in the activation of MAPK in response to chitin stimulation. The authors observed that *TRK1* induced the phosphorylation of MPK6 and MPK3, five minutes after exposure to chitin treatment. Lastly, from a functional standpoint, an increase in the levels of ROS in tomato plants to flg22 treatment was noted in transformants produced through RNAi engineering to have reduced *TRK1* expression. It is suggested that *TRK1* directly phosphorylates the NADPH oxidase RBOHD to prime ROS production. In addition, SIMYC2, a transcriptional regulator of jasmonic acid, was phosphorylated by *TRK1*, activating the plant immune response. These results indicate that *TRK1* plays a pivotal role in chitin signaling and fungal resistance.

In sum, these results show that exposure to chitin and chitin oligosaccharides triggers highly complex mechanisms in response to these stimuli. Among these stimuli, the signaling cascade via MAP kinases constitutes one of the fundamental elements (Figure 1).

5. MAP Kinases Are Activated in Response to Chitin Stimulation

MAP kinases are proteins that convert extracellular stimuli into a wide range of cellular responses. In that way, they are involved in processes of gene regulation, mitosis, cell differentiation, motility, and apoptosis, among others [89].

MAP kinases constitute the last elements of the signaling chain involved in the plant immune response. They are activated via the phosphorylation of RLCK proteins that bind to RLK on their intracellular side (Figure 1b). Within the MAP kinase signaling cascade, three fundamental proteins are included: MAPKKK (MAP kinase kinase kinase), MAPKK (MAP kinase kinase), and MAPK (MAP kinase) [90]. MAPKKKs are activated by intermediate components such as G (guanine nucleotide-binding) proteins or C (protein kinase C) proteins, whose activity may depend on the plasma membrane receptors. They are very heterogeneous, which allows them to respond to a wide variety of stimuli. Their function is to activate MAPKKs at conserved S/T-XXXXX-S/T (S/T is serine/threonine and X is an arbitrary amino acid) motif in MAPKK. MAPKKs are a group of more specific proteins that can only be activated by MAPKKKs, and, in turn, they activate only MAPKs (Figure 1). These have an anchor region rich in arginine and lysine residues essential for interaction with MAPKs, which are flanked by hydrophobic residues. MAPKs constitute the final element of this signaling cascade; these MAPKs require the threonine (Thr) and tyrosine (Tyr) residues in subdomains VII and VIII of their catalytic core to be phosphorylated, and when this occurs, they present an anchor domain rich in aspartate and glutamate residues that interact with the basic residues of the MAPKKs [89]. Finally, the MAPKs activate downstream transcription factors, enzymes, and other response factors (Figure 1), and by doing so, they transmit extracellular signals to the inside of the cells [91].

In *Arabidopsis*, there is a large family of MAPKs distributed in two families: MEKK kinases and Raf kinases. Previous findings, using fluorescence molecular complementation assays, showed that in the presence of chitin, MAPKKK5 (which belongs to the MEKK kinases) is phosphorylated by the RLCK binding protein PBL27 at Ser/Thr residues located at its C-terminus. MAPKKK5, in turn, phosphorylates the MKK4 and MKK5 kinases, immediately activating MAPK3 and MAPK6 [72]. Furthermore, it was demonstrated that once chitin recognition occurs, the RLCK VII subfamily proteins initiate the MAP kinase phosphorylation cascade and induce disease resistance [11]. These results clearly establish the relationship between RLCK binding proteins to RLK and the phosphorylation cascade via MAP kinases as part of the immune response to fight against fungal infection [92,93] or exposure to chitin oligosaccharides.

In rice, in response to chitin, it has been observed that CERK1 activates an intermediate kinase or binding protein called OsRLCK185, which in turn phosphorylates the OsMAPKKK18 homolog of MAPKKK5, which then activates MAPKK4 and, following that, activates MAPK3 and MAPK6 [94]. Moreover, it has been discovered that Os-RLCK185 is involved in the activation of MAPKKK24, inducing such activation through the MAPK3/MAPK6 pathway.

In the same way as in *Arabidopsis* and rice plants, the MAPK cascade in horticultural crops also consists of MAPKKK, MAPKK, and MPK. The MAPKs cascade is recognized as one of the essential pathways to be activated during plant defense in response to a pathogen attack [11]. For instance, in citrus fruits, MAPK1 played a crucial role in the plant immune response against *Xanthomonas citri* and *X. aurantifolii*. An increase in MAPK1 expression was observed in sweet orange and was positively correlated with a reduction in damage caused by the two pathogens [95]. Meanwhile, in grapes infected with *Erysiphe necator*, a notable upsurge in the transcripts of the majority MAPKKK genes was observed, with *VviMAPKKK50* showing the highest transcript abundance [96]. Elsewhere, in tomato plants, 16 possible SIMAPKKs involved in the response via MAP kinases were described. Specifically, MAPKK2 and MAPKK4 expression increased in the presence of *B. cinerea*, and the authors did not rule out the chance that other molecules could be implicated in the signaling pathway [97]. In another study, through transcriptomic analysis, the impact of infection with *C. gloeosporioides* on tomato fruit was evaluated [27]. It was observed that when compared to the uninfected fruits, the expression of more than 3000 transcripts was increased, which shows that many mechanisms are activated to counteract infection. Furthermore, the increase in transcripts related to pathogen recognition, such as RLK and RLCK proteins, as well as MAPKKK and salicylic acid in the necrotrophic stage, suggests that a related signaling cascade via the MAP kinase is vital in response to fungal infection. Likewise, Campos et al. [98], when carrying out a transcriptomic study in tomato before infection with *Alternaria solani*, observed that more than 5000 genes were differentially expressed, and, when infection occurred, an increase was determined in the genes related to the signaling cascade. Following on from that work, a protein kinase related to TPK1b, termed TRK1, was recently identified in tomato plants exposed to fungal chitin [12]. The authors observed that TRK1 enhances the phosphorylation of MPK6/3 in response to chitin and the SILYK1-TRK1 module activates the phosphorylation of SIMYC2, essential for the resistance of tomato plants to *B. cinerea*.

As previously described, the perception of chitin oligosaccharides triggers a series of defense signals in a wide range of plant species. The studies published so far have partially reported the molecular mechanisms that occur during the response to pathogen attack and chitin oligosaccharide exposure, mainly in *Arabidopsis* and rice. These molecular mechanisms involve the participation of PAMPs, as well as transmembrane chitin receptors such as RLK and RLP that recognize chitin oligosaccharides. A large number of RLKs exist, but only a small portion has been identified and functionally characterized by genetic studies. The plants employ an RLK-mediated signal transduction pathway, using intracellular molecules to modify gene expression profiles in response to external stimuli. These RLKs play a fundamental role in the regulation of various genetic functions during plant growth and development, as well as in plant immunity to a pathogen attack. When the recognition of pathogens or chitin oligosaccharides by transmembrane receptors occurs, RLKs activate cytoplasmic RLCK receptors that carry the signal into the cell. RLCKs function in conjunction with RLKs in the regulation of plant innate immunity. These RLCK receptors interact with the intracellular domains of chitin receptors, initiating the signaling cascade that leads to the activation of the defense mechanism. However, one of the most important and challenging tasks in signaling events is ligand identification. So far, only a limited number of RLK-binding RLCKs have been identified. For example, in the case of *Arabidopsis*, the activation of several RLCKs (PBL27, BIK1, PORK1-2, and ZED), induced by the transmembrane receptor CERK1-LYK5 has been demonstrated, while in horticultural crops, the information is limited. So far, only a single RLCK that is activated

by the BAK1 receptor has been reported in strawberry, a single RLCK activated by CERK1 has been reported in sugar cane, and two RLCKs activated by SILYK1 and PORK1 have been reported in tomato. Investigations into RLKs in corn, rice, and grapes have identified the presence of one or two RLCKs, but the type of RLK that activates them is unknown. As part of the signaling mechanism, RLCKs bind to proteins that initiate the phosphorylation of MAP kinases, activating transcription factors that collectively activate the plant immune response. However, it is not yet fully clear how RLK or RLP receptors induce the signaling pathway that activates a plant's immunity in horticultural crops in response to chitin and its oligosaccharides, an important component of fungal cell walls. To date, some studies have been carried out on horticultural crops (such as rice, corn, sugar cane, tomato, grape, apple, and pepper) and revealed the involvement of chitin receptors and RLCK cytoplasmic receptors in the signaling pathway. In addition, the RLCKs participate in the activation of MAPKs phosphorylation. Although the MAPK cascade is an important signaling module in PTI and could be a cross-point of signaling pathways induced by different PAMPs, the molecule that initiates the activation of the MAPK cascade remains unidentified. The studies published to date on the induction of the defense mechanism in horticultural crops suggest that the molecular mechanisms of the response to pathogen attack and chitin oligosaccharide exposure in fruits are similar to those reported in plants. Nevertheless, research is still limited on the functions of the genes encoding those receptors (RLKs and RLCKs) and MAPKs, as well as the mechanisms through which they govern resistance to pathogens in horticultural crops. There is a clear need to perform further studies in horticultural crops in order to fully elucidate the phenomenon of chitin perception and the molecular mechanisms involved in the chitin signaling and the activation of the response to pathogen attack.

6. Concluding Remarks

Horticultural crops are exposed to different types of biotic stress both in the pre-harvest period and in the post-harvest period, specifically fungi, and important economic losses are produced. The immune system of the plant or harvested fruits responds to fungal infections, specifically to the presence of chitin oligomers, due to the presence of three protein kinases—chitin-binding membrane receptors, receptor-like cytoplasmic kinases, and the cascade of MAP kinase proteins—that trigger the defense response. According to the few studies in horticultural crops and fruits, it is possible to suggest that these three protein kinases are involved in plant immunity triggered by chitin oligomers. This suggests that the molecular events involved in plant immunity triggered by chitin and reported in model plants like *Arabidopsis* and rice could be conserved in other plant species. Nevertheless, little is known about how the successive activation of these proteins occurs in the signaling cascade.

Further examination of horticultural crops is necessary to fully elucidate the phenomenon of chitin perception and the molecular mechanism responding to pathogen attacks. Deciphering the complete mechanisms involving chitin and its oligosaccharides, including perception, signaling pathways, and the activation of the defense mechanisms in horticultural crops, would greatly benefit agriculture. Such insights could significantly aid genetic programs in developing horticulture varieties with robust and enduring resistance to fungal pathogens. Moreover, this knowledge could contribute to the design of more effective biocontrol techniques targeting fungal pathogens in horticultural crops.

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Abbreviations

A. alternata (*Alternaria alternata*), APK1b (*Arabidopsis* protein kinase), AtLYK (Lysin-motif-containing receptor-like kinases in *Arabidopsis thaliana*), AvrPtoB (Type III effector protein from *Pseudomonas syringae* pv. tomato that physically interacts with the tomato Pto kinase), BAK1 (Brassinosteroid insensitive 1-associated receptor kinase), BIK1 (Botrytis-induced kinase1), BSK1 (Serine/threonine protein kinase), BSR1 (Rice RCLK), CaChitIV (*Capsicum annuum* chitinase IV), CaPIK1 (*Capsicum annuum* receptor-like cytoplasmic protein kinase1), CERK1 (Chitin elicitor receptor kinase 1), CIAPIN1 (cytokine-induced apoptosis inducer1), CNGC2/4 (Cyclic nucleotide-gated channel), EFR (PRR that binds to the prokaryotic protein EF-Tu), ETI (effector-triggered immunity), FLS2 (flagellin-sensitive 2), GlcNAc (N-acetyl- α -D-glucosamine), HAK1 (HDS associated RLs), HR (Hypersensitive response), JA (Jasmonic acid), LRR (Leucine-rich repeat receptor kinases), LysM-RLKs (Lysin-motif-containing receptor-like kinases), MAPKs (Mitogen-activated protein kinases), MAPKK (MAP kinase kinase), MAPKKK (MAP kinase kinase kinase), MEKK kinases (kinase family in *Arabidopsis*), NADH (Nicotinamide adenine dinucleotide dehydrogenase), NADPH (Nicotinamide adenine dinucleotide phosphate dehydrogenase), NIS1 (Necrosis-inducing secreted protein1), OSCA1.3 (calcium-permeable channel), PAMPs (Pathogen-associated molecular patterns), PBL27 (Serine/threonine protein kinase), PEPR1/PEPR2 (Perception of the *Arabidopsis* danger signal peptide 1 or 2), PORK1-2 (Tomato PEPR1 ortholog receptor-like kinase), PR proteins (Pathogenesis-related proteins), PRR (Pattern recognition receptors), PTI (PAMP-triggered immunity), Raf kinases (kinase family in *Arabidopsis*), RLCK (Receptor-like cytoplasmic kinase), RLK (Receptor-like kinase), RLP (Receptor-like protein), ROS (Reactive oxygen species), SA (Salicylic acid), SLAH3 anion channel (Interacts with potassium channels to regulate nitrogen-potassium homeostasis and the membrane potential in *Arabidopsis*), TPK1B (tomato RCLK), TRK1 (TPK1b Related Kinase1), WRKY (Transcription factor), ZAR1 (leucine-rich repeat domain NLR protein), ZED1 (Pseudokinase).

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