Enhancing Plants Fungal Disease Resistance

Subjects: Plant Sciences

Contributor: Seonghoe Jang

Fungal diseases pose a major threat to ornamental plants, with an increasing percentage of pathogen-driven host losses. In ornamental plants, management of the majority of fungal diseases primarily depends upon chemical control methods that are often non-specific. Host basal resistance, which is deficient in many ornamental plants, plays a key role in combating diseases. Despite their economic importance, conventional and molecular breeding approaches in ornamental plants to facilitate disease resistance are lagging, and this is predominantly due to their complex genomes, limited availability of gene pools, and degree of heterozygosity.

fungal diseases | genetic engineering | HIGS (host-induced gene silencing) | SIGS (spray-induced gene silencing) | ornamental plants | resistance mechanisms | breeding technology | Botrytis cinerea | Fusarium oxysporum | Alternaria sp.

1. Introduction

Ornamental plants possess natural beauty and are distinctive due to their exquisite blooms. The alluring colors and shapes of their flowers, leaves, and fruits of these plants are a source of major attraction. Ornamental crops are grown for various decorative purposes as potted plants, woody ornamentals, cut flowers or cut foliage, bulbs, and corms [1]. The floriculture sector is flourishing globally and is experiencing increased demand. Floriculture has significantly impacted the horticultural industry by facilitating a substantial turnover with regard to all aspects of floriculture, of which roughly one third of the global value of the ornamental market is made up of cut flowers [2]. The turnover of popular ornamental plants in the world's largest flower auction, the Royal FloraHolland auction is detailed in **Table 1** [3] (FloraHolland Key figures, 2019). As this represents a dynamic sector, introducing novelties into the market is a mandate for withstanding global competitiveness.

Table 1. Turnover of the top 10 ornamental plants in the Royal FloraHolland auction in 2019.

S.No	Ornamental Plants Sold	Turnover (Million Euros)
1	Rose	696
2	Chrysanthemum	328
3	Phalaenopsis	460
4	Tulip	285

S.No	Ornamental Plants Sold	Turnover (Million Euros)
5	Gerbera	148
6	Lily	144
7	Kalanchoe	65
8	Anthurium	60
9	Potted rose	57
10	Lavender	20

Plant pathogens cause severe losses in the production and/or quality of various ornamental crops and this is of great economic significance. Their effects range from mild symptoms to catastrophes, where larger areas of planted crops are seriously damaged [4]. Ornamental plants in general are infected by a myriad of microbial organisms, including bacteria, fungi, and viruses, that severely affect the growth and morphology of these plants and thereby influence their commercial value. The visual quality of ornamental plants is critical, particularly for cut flowers and potted plants. Visual disease symptoms and the impact on growth caused by pathogens both heavily affect the quality and the market value of the flowers. Hence, ensuring quality traits in these plants is essential as they face increasing demand for industrial purposes [5].

Among the diseases caused by fungi, bacteria, viruses, and viroids, approximately 70% of those in plants are caused by fungi. In many cases, fungal diseases cause a significant reduction in crop quality and yield that can represent up to 30–40% of the total potential yield [6]. Fungi are estimated to be the biggest threat and the major cause underlying pathogen-driven host losses, declining the visual quality and lowering of market prices of ornamental flowers [7]. The majority of plant fungi are strictly saprophytic and derive their nutrition from dead organic matter, while the remaining are pathogenic biotrophic and necrotropic fungi that grow on living plants and cause diseases [8]. Plant fungal pathogens can be largely classified into the phyla Ascomycota and Basidiomycota. Ascomycetes are represented by various classes of pathogens such as Sordariomycetes (*Magnaporthe* spp.), Dothideomycetes (*Cladosporium* spp.), and Leotiomycetes (*Botrytis* spp.), while Basidiomycetes includes two larger groups of plant pathogens such as the rusts (Pucciniomycetes) and the smuts (*Ustilaginomycetes*). Based on the nature of their interaction with plants, these pathogenic fungi are grouped into biotrophs that form an intimate interaction with the host plant and utilize its living tissues and the necrotrophs that kill the plant tissues by causing cellular necrosis that eventually leads to plant death [9]. Obligate biotrophic fungi cannot grow without a living host and cause various diseases in ornamental plants such as leaf spots, blights, rusts, smuts, powdery mildew disease, and downy mildew disease.

Frequently, fungal diseases are managed by the application of chemical fungicides that are effective only for a few diseases and are sometimes non-specific. Moreover, excessive use of chemicals results in pathogen resistance against these chemicals and is highly undesirable due to health and environmental safety concerns [10]. An alternative method to chemical control is the biological control of pathogens, and this can be achieved through an integrated approach for disease management [1]. However, the scope of disease control provided by biocontrol

methods is very limited. The formulations of beneficial fungi or bacteria that suppress plant pathogen growth usually provide some degree of control and can only be used as a component of the IPM strategy [11]. Hence, the ultimate goal is to generate plants that possess increased resistance to diseases. Effective control of diseases can be achieved by host basal resistance, as this can reduce the requirements of pesticide application. However, not all ornamental plants possess natural disease resistance; therefore, disease management relies on the use of disease-resistant varieties. Hence, it is important to elucidate pathogenicity and host-pathogen interactions to develop novel strategies for improving disease resistance in plants [12]. The development of disease-resistant varieties is possible via traditional breeding approaches or genetic engineering by introducing resistance mechanisms derived from other plant species or pathogens [13]. The introduction of natural resistance by traditional breeding approaches includes non-transgenic breeding programs, such as DNA-based marker-assisted selection that may require several cycles of breeding to combine the disease-resistant trait and desirable ornamental characteristics into a single plant genotype. In contrast, the transgenic approach uses tightly regulated transgenes to introduce specific or broad-spectrum disease resistance into genotypes with elite ornamental qualities [14]. Breeding efforts to achieve disease resistance in ornamental plants are comparatively limited, as the disease-resistance trait is typically taken into consideration only during the later stages of the breeding line selection process of cultivar development [15]. Genetic mapping of disease resistance is relatively scarce due to the large and complex genomes and the nature of the polyploidy present in most ornamental plants, as these characteristics require a greater number of resources and more time to map the resistance mechanisms [16]. Nevertheless, recent advances in genome-sequencing technologies, phenotyping, marker development, and genotyping have provided a promising base for further breeding development for disease resistance in ornamental plants. Alternatively, genetic engineering technology provides a potential platform for the improvement of resistance to a myriad of biotic and abiotic stresses in ornamental plants, thus improving plant quality. Tolerance to several fungal diseases has been achieved by transferring various genes such as *glucanase*, *chitinase*, *defensin*, *osmotin*, and *pathogenesis-related* (*PR*) genes into ornamental plants [17]. RNA interference (RNAi) strategies have also demonstrated the potential to protect plants against pathogens, and one of these strategies is host-induced gene silencing (HIGS), mediated by RNAi signals generated in planta [18]. In addition to HIGS, a novel strategy designated as 'spray-induced gene silencing' (SIGS) has been demonstrated to protect plants from fungal pathogens through the direct spraying of dsRNA-targeting pathogen genes in plant tissues, thus displaying the potential to be used as an alternative to conventional fungicides [19]. Furthermore, advances in genome editing technology and its applications have offered greater possibilities with regard to precise manipulation of the genome sequences at genes of interest, and these techniques are currently being used to improve disease resistance in plants [20].

2. Genetic Engineering for Improved Fungal Disease Resistance in Ornamental Plants

Although various fungal diseases are managed by fungicide application, fungicides are often non-specific and kill beneficial microbes along with pathogens. Moreover, most fungicides are hazardous chemicals to both human and environmental health. Additionally, excessive use of these chemicals can cause resistance to fungicides [21]. Hence, the development of fungal disease-resistant cultivars would provide a promising alternative method for

efficient ornamental production with minimal losses by fungal pathogens. The development of fungal disease resistance through conventional breeding is hindered by several limitations such as deficiency of gene resources for many diseases, the transfer of undesirable traits along with resistant genes, and the rapid evolution of the ability of pathogens to overcome plant-resistance mechanisms [22]. Alternatively, genetic engineering possesses the potential to overcome the barriers in traditional breeding methods and to control the ability of the plant to identify and defend itself against fungal diseases. Advances in genetic engineering have enabled researchers to better understand the molecular mechanisms of plant defense responses, thus contributing to the development of novel strategies to combat the disease [6]. In contrast to conventional breeding, genetic engineering offers the possibility of increasing the disease resistance to several pathogens simultaneously, and the gene of interest can be introduced into the target plant even if the gene does not exist in the natural gene pool [2]. Advances in genetic engineering to achieve fungal disease resistance in various ornamental plants are discussed in this section.

2.1. Rose

Roses (*Rosa hybrida*) are cultivated throughout the world and are an economically important ornamental plant worldwide. Roses are most admired for their beauty and fragrance, and they exhibit alluring colors. Within the *Rosa* genus, there are more than 200 rose species and over 30,000 cultivars. They are used as cut flowers, pot plants, and garden plants [23]. Rose-petal essential oils consist of beneficial secondary metabolites that are used in the natural medicine, cosmetics, and perfume industries [24]. However, rose cultivation is severely impaired by major fungal diseases such as powdery mildew, black spots, botrytis blight, downy mildew, and rust that adversely affect yields and product quality [25]. Despite the economic importance of the rose as an ornamental crop, breeding progress for fungal resistance is lagging in roses due to insufficient information regarding disease-resistant traits. Moreover, a higher level of heterozygosity, sterility, and polyploidy are the major limitations of traditional breeding for fungal disease resistance in roses [26]. Hence, genetic engineering is a desirable approach to induce resistance against fungal diseases. Powdery mildew caused by the obligate ascomycete pathogen *Podosphaera pannosa* (Wallr.: Fr.) is one of the predominant fungal diseases of rose. It causes distortion and senescence of the leaves and shoots. Approximately 40% of the fungicide sprayed on cut and potted roses is used to control powdery mildew [27]. It is known that PR genes, including *β-1,3-glucanase*, *chitinase*, *ribosome-inactivating protein* (*RIP*), and *cysteine-rich antimicrobial protein* (*AMP*), are triggered during fungal pathogen infections [28][29]. These antifungal proteins, including chitinases, glucanases, RIPs, plant defensins, and proteinase inhibitors, function by disrupting or suppressing the synthesis of the fungal cell wall. Some of these proteins interact with potential intracellular targets and the plasma membrane of fungi, thus leading to changes in membrane potential and cell death [30]. Plant defensins, including AMPs, are known to interact with glucosylceramides within fungal membranes to induce membrane permeabilization, ultimately leading to fungal cell death [31]. An antimicrobial protein gene (*Ace-AMP1*) isolated from onion seeds that possessed higher plant pathogenic inhibition activity, was introduced into the *Rosa hybrida* cv. Carefree Beauty. The transgenic rose, overexpressing the *Ace-AMP1* gene, was developed to induce fungal disease resistance, and the rose showed enhanced resistance to powdery mildew disease [32]. Furthermore, the transgenic rose, overexpressing antifungal genes such as *class II chitinase* and *type I ribosome inhibiting protein* (*RIP*), exhibited reduced susceptibility to fungal diseases [25]. Transgenic rose plants possessing a high level of expression of the rice *chitinase* gene displayed improved resistance to powdery mildew [33]. Previous

studies suggested that loss-of-function mutations in mildew resistance locus- o (*Mlo*) genes confer broad-spectrum resistance against pathogens, and hence, *Mlo* genes can confer an effective race-independent resistance in several crops [34][35]. Although the mechanism underlying *MLO*-based disease resistance remains unclear, some of their family members function by regulating fungal-penetration resistance by controlling vesicle fusion events [36]. Indeed, Qiu et al. (2015) generated transgenic *Rosa multiflora* expressing an antisense *RhMLO1* that exhibited enhanced resistance to powdery mildew [37]. Xiang et al. (2019) recently identified two *MLO* members, *RgMLO6* and *RlMLO7*, that are potential candidate genes that can induce resistance to powdery mildew in *Rosa* species [38]. Black spot disease is another major fungal disease caused by *Diplocarpon rosae* Wolf, a hemibiotrophic ascomycete. It is one of the most devastating and widespread fungal diseases of the rose and leads to huge economic losses [39]. Black and brown spots appear on leaves as the representative symptoms of the disease and, eventually, immature leaves become weak and fall from the plant. Defoliation decreases the photosynthetic area of plants, thus leading to a reduction in plant vibrance, thereby drastically lowering its ornamental value. A rice *chitinase* gene introduced into the rose-susceptible cultivar ‘Glad Tidings’ by particle bombardment conferred reduced susceptibility to black spot disease [40]. The black-spot-susceptible rose cultivars ‘Heckenzauber’ and ‘Pariser Charme’ were transformed with *chitinases*, *glucanases*, and *RIPs* from barley, and the transgenic plants exhibited a reduction of 40% in black spot diseases compared to that of the control [25]. Terefe-Ayana et al. (2011) reported the *Rdr1* locus as important for resistance to black spot diseases in roses, and this is useful for applications in rose breeding, including the use of genetic modification technology [41]. Recently, transcriptomic analyses of roses responding to the two fungal pathogens, *D. rosae* (black spot) and *P. pannosa* (powdery mildew), demonstrated that the genes related to common defense mechanisms were upregulated in black spot and that those related to photosynthesis and cell-wall modification were downregulated for powdery mildew, thus implying that distinct cellular responses are stimulated by different fungal pathogens, even in the same host [42]. *B. cinerea* is a notorious fungal pathogen responsible for gray mold disease in roses. *B. cinerea* conidia secretes phytotoxins and secondary metabolites during penetration into the host epidermis, ultimately causing host cell death [43]. Necrotic local lesions on petals are the major symptoms of *B. cinerea* infection in roses, and these infections rapidly develop during postharvest transport when the flowers are packed in boxes with a high relative humidity [44]. Petals are economically important organs, and when they are damaged, this causes large commercial losses in the rose industry. Despite its economic importance as a predominant pathogen, studies examining *B. cinerea* infections in roses are limited to the comparisons of pathogen behavior in model plants such as *Arabidopsis* [45]. Recently, transcriptomic analyses of rose petals infected by *B. cinerea* determined that *RcERF099,* a gene that encodes member of the AP2/ERF transcription factor family, is involved in the regulation of resistance against *B. cinerea* in rose flowers, and this finding can provide a stepping stone for further studies aiming to improve gray mold disease resistance in roses [46].

2.2. Chrysanthemum

The chrysanthemum (*Chrysanthemum morifolium*) is one of the most economically important and highly favored floricultural crops in terms of ornamental market value, and is used as a cut flower, pot flower, and garden plant [47]. It is a herbaceous perennial species belonging to *Asteraceae* and some of the family members, such as *Chrysanthemum morifolium* and *Chrysanthemum indicum,* have been widely used for medicinal tea and/or as

materials in the cosmetic industry [48]. Chrysanthemums possess a higher ornamental value due to their abundant diversity in floral color and shape, which is the result of their large genome complexity and the allohexaploid background of the cultivated chrysanthemum [49]. Chrysanthemums are affected by a wide range of fungal diseases, including leaf spots, gray mold, rusts, and powdery mildew. A major aspect of chrysanthemum crop production relies on chemical control and this process exhibits only ephemeral benefits. The narrow genetic pool and complex hexaploid genome are major limitations for classical breeding to introduce disease-resistant traits. Thus, genetic transformation is a potential alternative to hasten the production of disease-resistant genotypes with improved targeted traits [50]. Leaf spots in chrysanthemums are caused by different fungi, including the *Alternaria* species, *Septoria chrysanthemi*, *Septoria chrysanthemella, Septoria obesa*, and *Cercospora chrysanthemi*. Symptoms appear on leaves as yellowish spots that gradually become dark brown and black, ultimately leading to premature leaf losses and consequent yield losses. Transgenic chrysanthemums, overexpressing *polygalacturonase-inhibiting protein* (*PGIP*) from *Prunus mumei*, exhibited improved resistance to Alternaria leaf spot [51]. Hairpins are pathogenic molecules encoded by *hrp* genes that can induce plant resistance by activating defense-signaling cascades. Overexpression of one such *hrp* gene, $hpaG_{Xoo}$, conferred increased resistance to *Alternaria tenuissima* in chrysanthemums [52], and the introduction of the rice *chitinase* gene (*chiII*) in chrysanthemums cv. Snowball resulted in increased resistance to leaf spot caused by *Septoria obesa* [53]. Gray mold disease caused by *B. cinerea* is the predominant fungal disease in chrysanthemums. Leaves from infected plants possess brown water-soaked spots and the infected parts are covered with a grayish-brown, powdery mass of spores. Takatsu et al. (1999) produced transgenic chrysanthemum lines overexpressing a rice *chitinase* gene (*RCC2*), which showed enhanced resistance to gray mold disease [54]. Similarly, chrysanthemums cv. Shinba, overexpressing *N-methyl transferase* genes such as *CaXMT1*, *CaMXMT1*, and *CaDXMT1,* exhibited increased resistance to *B. cinerea*. Leaves from the transgenic lines produced 2.5-fold higher levels of salicylic acid compared to that of the wild type, thus leading to delayed occurrence of the disease and reduced disease index [55]. These *N-methyl transferases* methylate xanthosine derivatives can be used to yield caffeine that indirectly stimulates the defense network, thus inducing the systemic acquired resistance in the host plant [56]. White rust disease is caused by *Puccinia horiana Henn*. and is one of the most destructive fungal diseases in chrysanthemums. It spreads rapidly under humid conditions in greenhouses, ultimately resulting in considerable economic losses [57]. Symptoms typically appear on the adaxial leaf surface as pale green to yellow spots, that then exhibit raised buff or pinkish pustules. Stems, bracts, flower buds, and florets are infected in susceptible cultivars [58]. Transgenic chrysanthemums, overexpressing the *Cry1Ab* gene from *Bacillus thuringiensis* and a modified *sarcotoxin IA* gene from *Sarcophaga peregrine* (*msar*), exhibited a stronger resistance to white rust caused by *Puccinia horiana* and also exhibited *Helicoverpa armigera* resistance [59]. A recent study demonstrated that *CmWRKY15-1*, which encodes a WRKY transcription factor, plays a key role in the resistance to white rust caused by *P. horiana* by regulating the salicylic acid-mediated disease-resistance signaling pathway in chrysanthemums [60].

2.3. Petunia

Petunia hybrida is a popular ornamental hybrid with diverse floral colors and morphologies. It belongs to the *Solanaceae* family and is native to South America. Petunias possess a well-established record of being a model

system for studying the molecular, genetic, and ecological factors that determine flower development [61][62] and can be affected by wilting, discoloration, and plant death. Verticillium wilt is caused by *Verticillium albo-atrum* that attacks the plant from the soil through a water-transport system. The infected leaves eventually turn brown and drop off from the plant. The petunia is infected by powdery mildew pathogens such as *Podosphaera xanthii*, *Golovinomyces orontii*, and *Oidium longipes*. Symptoms can be identified according to powdery white spores on the foliage [63]. Petunias are severely affected by *B. cinerea*, a foliar leaf pathogen that causes gray mold and leaf blight [64]. Transgenic *Petunia hybrida*, overexpressing the *endochitinase* gene from *Trichoderma harzianum*, alone or in combination with *osmotin*, exhibited resistance to *B. cinerea* [65]. Khan et al. (2011) developed transgenic petunia plants overexpressing the wasabi *defensin* (*WD*) gene from *Wasabia japonica* [66]. Expression of the AMP *defensin* increased resistance to *B. cinerea* in marker-free transgenic petunias. Similarly, transgenic *Petunia hybrida* plants, overexpressing the synthetic *chitinase* gene *Nakamura Ikuo Chitinase* (*NIC*) encoding Chitinase1 protein of *Rhizopus oligosporus*, exhibited enhanced resistance to *B. cinerea* [67]. Recently, reduced levels of *PhMLO1* expression achieved by introducing a *PhMLO1* RNAi construct resulted in improved resistance to powdery mildew in petunias. However, *PhMLO1* knockdown resulted in pleiotropic effects on petunia growth and development that may have a negative effect on the further development of strategies to create powdery mildew resistance by RNAi in petunias [68].

2.4. Lily

Lilies (*Lilium* spp.), cultivated as a flower crop and potted plant, are one of the most popular ornamental plants. Lilies are affected by major fungal diseases, including gray mold caused by *Botrytis elliptica*. Symptoms are characterized by oval or circular yellowish or red spots on the leaves. Infected floral buds become shriveled and distorted, and the plants can die, depending on the severity of the disease [69]. Bulb rot in lilies caused by *Fusarium oxysporum* produces the initial symptoms of the plant's foliage yellowing and wilting. Even though the bulbs appear healthy, the roots develop a reddish-colored decay in the tips. The plants become stunted with yellow foliage and rotted scales. The transgenic *Lilium* oriental 'Star Gazer', developed by overexpressing the *RCH10* chitinase gene, conferred resistance to *B. cinerea* [70]. More recently, microRNA159 from *Lilium regale* (*lre-miR159*) has been reported to confer resistance to gray mold caused by *B. elliptica* in transgenic *Arabidopsis* by repressing the expression of its target gene *LrGAMYB* [71]. Additionally, overexpression of the *LsGRP1* gene encoding a class II glycine-rich protein from *Lilium*, conferred resistance to *B. cinerea* in *Arabidopsis*. The authors determined that *LsGRP1* plays a role as a pathogen-inducible switch to allow for activation of the immune response in the plant and to consequently induce fungal apoptosis [72]. Several candidate genes conferring resistance to fungal pathogens have been identified in lilies. Sun et al. (2016) reported that transgenic petunia plants, overexpressing the ATP-binding cassette transporter gene *LrABCF1* from *L. regale,* displayed increased resistance to *B. cinerea* and RNA viruses (cucumber mosaic virus and tobacco rattle virus) in petunias [73]. Similarly, the *glutathione-S-transferase* gene introduced by *L. regale* Wilson induced resistance to *F. oxysporum* in transgenic tobacco [74] and the overexpression of a 14-3-3 gene from *L. regale* Wilson conferred resistance to *Fusarium* wilt in transgenic tobacco [75]. Various genes induced in response to an *F. oxysporum* infection have been identified in *L. regale* Wilson [76][77][78] and the identified candidates serve as valuable resources to develop improved resistance to fungal pathogens in lily cultivars.

2.5. Other Ornamentals

Various ornamental plants, including the carnation, gladiolus, scented geraniums, African violets, and bentgrass, have been transformed to possess fungal disease resistance. Transgenic carnation harboring different combinations of *PR-1*, *osmotin*, or *chitinase* genes have been developed to induce resistance to *F. oxysporum* [79]. Resistance to *Fusarium* wilt was generated in transgenic carnation by transforming the bacterial *chitinase* gene from *Serratia marsecens* [80]. Later, the *jasmonate methyl transferase* gene was introduced into carnation for *Fusarium* resistance [81]. Transgenic gladiolus 'Peter Pears', developed by transforming with a synthetic antimicrobial peptide gene (*D4E1*), exhibited enhanced resistance to *F.oxysporum* [82]. Kamo et al. (2016) demonstrated that cell extracts from the transgenic gladiolus, overexpressing a fungal *exochitinase*, *endochitinase*, or a bacterial *chloroperoxidase*, could inhibit the growth of *F. oxysporum* [83]. The *Ace-AMP1* gene was transformed in scented geraniums to provide resistance to *B. cinerea*, and the expression level of the Ace-AMP1 protein was proportionally correlated with enhanced resistance to *Botrytis* sporulation [84]. *Glucanase* and *chitinase* genes were transformed into African violets to induce resistance to *F. oxysporum* and *Pythium* [85]. Transgenic glufosinate-resistant bentgrass (*Agrostis* spp.) plants, developed for herbicide resistance, exhibited increased resistance to fungal pathogens, including *Rhizoctonia solani* and *Sclerotinia homoeocarpa*, after a spraying with glufosinate herbicide, thus indicating that the nonselective herbicide glufosinate can be used to suppress some fungal pathogens in transgenic glufosinate-resistant bentgrasses [86]. SNP markers for linkage mapping and the transcripts that may be involved in *Botrytis* resistance have been recently identified in gerbera, and these findings may be useful for further studies of disease resistance [87]. Moreover, transcriptomic analyses performed in gerbera revealed candidate genes for resistance to powdery mildew, and these could provide valuable resources for developing powdery mildew-resistant gerbera cultivars [88]. Recent reports detailing the enhancement of fungal disease resistance in various ornamental plants are listed in **Table 2**.

Table 2. Recent advances in the genetic engineering of various ornamental crops for fungal disease resistance.

Crop	Gene	Disease Resistance	Reference
Rose (*Rosa hybrida*)	*Ace-AMP1*	powdery mildew (*Podosphaera pannosa*)	[32]
	rice *chitinase*	powdery mildew (*P. pannosa*)	[33]
	RhMLO1, RgMLO6, RlMLO7	powdery mildew (*P. pannosa*)	[37][38]
	rice *chitinase*	black spot (*Diplocarpon rosae*)	[40]
	chitinases, *glucanases*, and *RIPs*	black spot (*D. rosae*)	[25]

Crop	Gene	Disease Resistance	Reference
Chrysanthemum (*Chrysanthemum morifolium*)	*Rdr1*	black spot (*D. rosae*)	[41]
	PGIP	Alternaria leaf spot (*Septoria chrysanthemi*)	[51]
	hairpinXoo	leaf spot (*Alternaria tenuissima*)	[52]
	chiII	leaf spot (*Septoria obesa*)	[53]
	RCC2	gray mold (*B. cinerea*)	[54]
Petunia (*Petunia hybrida*)	*CaXMT1, CaMXMT1, CaDXMT1*	gray mold (*B. cinerea*)	[55]
	Cry1Ab and *sarcotoxin IA*	white rust (*P. horiana*)	[59]
	CmWRKY15-1	white rust	[60]
	endochitinase and *osmotin*	gray mold (*B. cinerea*)	[65]
	WD (Wasabi defensin)	gray mold (*B. cinerea*)	[66]
	NIC (Nakamura Ikuo Chitinase)	gray mold (*B. cinerea*)	[67]
Lily (*Lilium*)	*RCH10 chitinase*	gray mold (*B. cinerea*)	[70]
Carnation (*Dianthus caryophyllus*)	*Ire-miR159*	gray mold (*B. elliptica*)	[71]
	PR-1, osmotin, chitinase	Fusarium wilt (*F. oxysporum*)	[79]
	bacterial *chitinase*	Fusarium wilt (*F. oxysporum*)	[80]
	jasmonate methyl transferase	Fusarium wilt (*F. oxysporum*)	[81]

Crop	Gene	Disease Resistance	Reference
Gladioulus (*Gladiolus communis*)	*D4E1*	Fusarium wilt *(F.oxysporum)*	[82]
	Fungal *exochitinase, endochitinase,* bacterial *chloroperoxidase*	Fusarium wilt *(F. oxysporum)*	[83]
Geranium (*Pelargonium graveolens L. Herit.*)	*Ace-AMP1*	gray mold *(B. cinerea)*	[84]
African violets (*Saintpaulia ionantha*)	*glucanase* and *chitinase*	Fusarium and Pythium	[85]

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