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Review

Insight into the Microbiological Control Strategies against *Botrytis cinerea* Using Systemic Plant Resistance Activation

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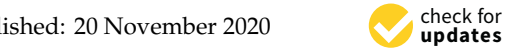

Abstract: *Botrytis cinerea* is a polyphagous necrotrophic fungus and is the causal agent of grey mold diseases in more than 1400 different hosts. This fungus causes serious economic losses in both preharvest and post-harvest—mainly in grape, strawberry, and tomato crops—and is the second most important pathogen worldwide, to our knowledge. Beneficial bacteria and fungi are efficient biocontrol agents against *B. cinerea* through direct mechanisms, such as parasitism, antibiosis, and competition, but also indirectly through the activation of systemic plant resistance. The interaction between plants and these microorganisms can lead to the development of defensive responses in distant plant organs, which are highly effective against foliar, flower, and fruit pathogens, such as *B. cinerea*. This review aimed to explore the systemic plant defense responses against *B. cinerea* by compiling all cases reported (to the best of our knowledge) on the use of beneficial bacteria and fungi for agriculture, a subject not yet specifically addressed.

Keywords: *Botrytis cinerea*; salicylic acid; jasmonic acid; *Bacillus*; *Pseudomonas*; *Trichoderma*

1. Introduction

Botrytis is a highly diverse fungal genus including numerous species that differ in their biology, ecology, morphological features, and host range. Progress in molecular genetics and the development of relevant phylogenetic markers in particular has resulted in the characterization of approximately 30 species. Species of *Botrytis* are responsible for relevant losses in a number of economically important horticultural and floral crops [1].

Botrytis cinerea Pers.:Fr is the most commonly studied polyphagic fungus. Although *B. cinerea* is the name of the asexual stage (anamorph) and *Botryotinia fuckeliana* is the name of the sexual stage, the *Botrytis* community agreed in 2013 at the *Botrytis* Symposium in Bari to use *Botrytis cinerea* as the generic name [1]. The life cycle of this fungus includes sclerotia developing within dying host tissues, representing an important survival mechanism. Sclerotia commences its growth in the early spring in temperate regions to produce conidiophores and multinucleate conidia. The sexual lifecycle of this fungus involves the spermatization of sclerotia, leading to the production of apothecia and asci with eight binucleate ascospores serving as the primary source of inoculum within a crop [2,3].

The infection process of *B. cinerea* is usually described with the following stages: penetration of the host surface, killing of the host tissue/primary lesion formation, lesion expansion/tissue maceration, and sporulation [4]. This necrotrophic fungus is responsible for a very wide range of symptoms, which cannot easily be generalized across plant organs and tissues. Soft rots, accompanied by collapse and water soaking of the parenchyma tissues and followed by a rapid appearance of grey masses of conidia, are the most typical symptoms on leaves and soft fruits. For many fruits and vegetables,

the infection commonly begins on attached senescent flowers and then spreads as a soft rot, affecting the adjacent developing fruit (blossom-end rot), such as strawberries and apples. Moreover, seed-borne infections have been reported in over 50 hosts, where grey mold often begins by rotting the herbaceous stems at ground level, with other soft-rot lesions also appearing on leaves and pods [3]. In this sense, *B. cinerea* is an interesting model system for necrotrophic pathogens; however, it is not easy to study, since there are frequent variations of its karyotypes among natural strains [1].

B. cinerea is a highly polyphagous fungal plant pathogen, causing grey mold on more than 1400 known hosts in 586 plant genera and 152 botanical families, from mosses to gymnosperms and eudicots [5,6]. This pathogen has a disastrous economic impact on various economically important crops, including grape, strawberry, and tomato. Although this fungus causes serious pre-harvest problems, *B. cinerea* is considered one of the most important post-harvest pathogens in fresh fruits and vegetables. The annual economic losses of *B. cinerea* easily exceed $10 billion worldwide, possibly reaching as high a $100 billion. Due to both its economic and scientific importance, *B. cinerea* has been classified as the second most important plant pathogen. Controlling this fungus is difficult because it has a broad host range, various attack modes, and both asexual and sexual stages allowing it to survive. To date, the principal means to control grey mold rot caused by *B. cinerea* remains the application of synthetic fungicides, with a global investment that exceeds $1 billion. However, the use of conventional fungicides is not an adequate control strategy due to development of resistant strains and risks on human health and the environment [7]. Therefore, new effective and safe control strategies must be sought, such as those based on biocontrol.

2. Direct Biocontrol against *B. cinerea*

In recent years, the use of microbial biofungicides based on microbial biocontrol agents has increased continuously due to public concerns regarding the risk of pesticide residues in food and their negative impacts on the environment. For microbiological biocontrol, several fungal and bacterial strains have been successfully tested against grey mold on a variety of crops.

The bacterial genus *Bacillus* includes species widely studied and used as biocontrol agents against phytopathogenic fungi in agriculture due to their diverse secondary metabolism and ability to produce a wide variety of structurally different antagonistic substances, a mechanism of action known as antibiosis [8]. In this way, inhibition of the grey mold disease in tomato leaves between 75% and 90% has been achieved, thanks to metabolites released into the environment by *B. subtilis* and *B. licheniformis* [9,10], such as in strawberry plants [11,12]. This is due to compounds, such as the lipopeptides iturin, bacillomycin, fengycin, and surfactin, in which the efficiency was determined both *in vitro* by *B. velezensis* [13] and, in post-harvest apples, by *B. subtilis* [14]. Moreover, *B. subtilis* and *B. amyloliquefaciens* have been described as species with the capacity to produce and release antifungal volatile organic compounds (VOCs) against *B. cinerea*, thereby inhibiting the germination of their spores and the growth of their hyphae, both *in vitro* and *in planta* [15,16]. Finally, bacteria can compete in the phyllosphere for space, preventing the establishment of and attacking the necrotrophic fungus, as verified with *B. amyloliquefaciens* in tomato leaves, thanks to the formation of biofilms [17].

Antibiosis is also used by other bacteria to control *B. cinerea*, such as the release of antifungal compounds by *Pesudomonas* sp., *Serratia plymuthica*, and *Streptomyces philanthi* (e.g., antibiotic pyrrolnitrin or different VOCs), capable of totally inhibiting the germination of the spores of the fungus *in vitro* and decreasing the incidence of the disease in tomato and cucumber by greater than 75% [18–20]. In grapevine and strawberry leaves and fruits, it has been possible to verify how the bacteria *Pantoea ananatis* and *Lactobacillus plantarum*, respectively, compete effectively for space by rapidly colonizing wounds before the establishment of *B. cinerea* and suppressing the mycelial growth and disease symptoms [21,22]. In addition, bacteria, such as *Paenibacillus elgii*, are capable of releasing chitinolytic enzymes [23], a mechanism possibly linked to the ability of *Rahnella aquatilis* to parasitize the spores of the necrotrophic fungus on the surfaces of post-harvest apples [24].

For yeasts, few studies have been carried out *in planta*, but the use of yeasts as antagonistic microorganisms in the coating of fruits for the post-harvest control of *B. cinerea* represents one of the most widespread alternatives in biocontrol. The most commonly used yeast species against *B. cinerea* is *Aureobasidium pullulans* due to its ability to compete effectively for space and nutrients, both on the plant surface and in wounds, and for the release of different antifungal compounds, with successful applications in the post-harvest industry in grapes and kiwifruits [25–27]. In this regard, effective antagonism has also been described through the release of VOCs in strawberries by *Galactomyces candidum* [28] and the competition for space in wounds by *Rhodotorula glutinis* [29]. In addition, *in planta*, for both tomato leaves and post-harvest grapes, it has been possible to significantly inhibit the development of the fungus and the appearance of the disease, thanks to the ability of *Candida oleophila* and *Pichia membranifaciens*, respectively, to produce chitinase and glucanase enzymes that degrade the fungal cell wall [30,31].

Within filamentous fungi, the genus *Trichoderma* stands out as the main biological control agent against *B. cinerea*. Various species within this genus are widely used as biological control agents in agriculture due to their direct-action mechanisms, such as mycoparasitism, antibiosis, and competition for space and nutrients in the rhizosphere [32]. These mechanisms are also effective for the control of *B. cinerea*, with up to 75 species within the genus capable of actively mycoparasitizing the fungus, penetrating its cell wall through the production of different glucanases and chitinases. In addition, there is a very wide diversity of secondary metabolites produced by different *Trichoderma* species capable of inhibiting the growth and development of *B. cinerea* and even irreversibly damaging its cells. Some of these secondary metabolites are pyrones, butenolides, azaphylones, anthraquinones, trichothecenes, terpenoids, steroids, and peptaibols [33]. For this reason, *Trichoderma* has been also used as a source of genes for the development of transgenic plants resistant to *B. cinerea* [34]. Other species of filamentous fungi are capable of producing and releasing chemical compounds that effectively antagonize the development of grey mold. Inhibition in the growth of hyphae close to 90% has been reported, together with total inhibition of the germination of their spores, both through the diffusion of metabolites and through the production of VOCs by *Albifimbria verrucaria*, *Metarhizium anisopliae*, and *Ulocladium atrum* [35–37].

3. Systemic Plant Resistance and *B. cinerea*

When a pathogen, such as *B. cinerea*, crosses the constitutive plant defensive barriers, the plant must defend itself by activating a specific defensive response. For this, it is necessary for the plant to recognize the attacking pathogen through what is known as the pattern recognition receptors (PRRs) of the plant cells, which will recognize the molecular components of these microorganisms, called the pathogen-associated molecular pattern (PAMP). As a consequence of this recognition, the plant will activate a first-layer defense response called PAMP-triggered immunity (PTI). Plant responses occur in the organ where the plant was originally attacked (local response) and also in the distant plant parts that are unaffected (systemic response) [38,39].

These defensive responses are coordinated by stress hormones, such as salicylic acid (SA), mostly associated with biotrophic pathogens, as well as jasmonic acid (JA) and ethylene (ET), against necrotrophic pathogens and herbivores. After an attack from a biotroph pathogen and the occurrence of a programmed cell death response in a plant, a broad-spectrum immunity to reinfection through the whole plant body is activated in the plant, called systemic acquired resistance (SAR). SAR signaling is mainly mediated by SA-derived compounds, such as methyl salicylate (MeSA). Similarly, against necrotrophic pathogens and herbivores, the response known as induced systemic resistance (ISR) is activated. ISR is regulated by JA/ethylene (ET) signaling, although dependence on SA signaling has also been reported. Both SAR and ISR are indirect modes of action used by different biocontrol agents and involve considerable energy consumption by the plant [39].

In relation to all of the above, a plant's ability to pre-activate its defensive responses has been extensively verified to occur through priming without the plant have to come into contact with

pathogenic microorganisms or receive signals from nearby plants that have done so. Through this mechanism, plants take defensive measures against a potential attacker while also preparing their defensive systems for a faster and/or stronger reaction in the future. Although beneficial microorganisms, such as plant growth-promoting rhizobacteria (PGPRs) and plant growth-promoting fungi (PGPFs), are most commonly involved in the development of priming, different chemical compounds can activate this mechanism, such as SA, JA, β-aminobutyric acid (BABA), probenazole, and benzothiadiazole [39].

During infection, *B. cinerea* penetrates the plant-cuticle by secreting lytic enzymes and phytotoxins. Consequently, plants accumulate reactive oxygen species (ROS) in the plasma membranes of the host cells to trigger an oxidative burst, leading to plant cell death. As verified in *Arabidopsis thaliana*, there is a receptor-like cytoplasmic kinase PRR called *Arabidopsis Botrytis*-induced kinase1 (BIK1) that recognizes the PAMPs associated with *B. cinerea*, activating the corresponding PTI [40–42].

A plant's defense against necrotrophic pathogens, such as *B. cinerea*, is greatly dependent on crosstalk among the phytohormones SA, JA, and ET. The role of SA signaling in plant resistance to *B. cinerea* is still unclear. Although SA appears to negatively regulate defense responses to *B. cinerea*, its role is quite complex. On the other hand, the JA signaling pathway is crucial in inducing resistance against *B. cinerea*, while ET may play a "two-faced" role in disease resistance, depending on the plant species, triggering both negative and positive responses in plant defense against this necrotrophic fungus. In this respect, the synergistic activity of the JA and ET signaling pathways has been well-characterized after *B. cinerea* infection, showing an antagonistic interaction between SA and JA in which ET acts as a fine-tuning modulator. Therefore, the systemic plant response against *B. cinerea* is necessarily linked to the ISR and JA/ET pathways [41,42].

Systemic plant resistance against *B. cinerea* is not only activated after the attack of the pathogen but can also be pre-activated by the exogenous application of various chemical compounds and biological elicitors. The exogenous application of plant defense hormones in fresh products during post-harvest has been shown to be capable of activating plant resistance against attacks from different pathogens, which occurs with the exogenous application of compounds derived from JA, such as methyl jasmonate (MeJA) [39]. *In planta*, various chemical compounds capable of activating a priming-type systemic resistance against *B. cinerea* have been described, such as BABA in *A. thaliana* [43] and tomato [44], benzothiadiazole (BTH) in poinsettia [45], hexanoic acid [46] and riboflavin in tomato [47], and elicitors, such as chitosan [48] and laminarin, in grapevine [49]. In this sense, mechanical damage and wounds are also capable of activating a plant's systemic resistance against *B. cinerea* [50,51], as well as adverse environmental factors, such as high temperatures [52] and UV radiation [53]. Moreover, the conditions of cultivation may be able to activate a plant's systemic resistance against *B. cinerea*. Indeed, priming against the pathogens in hydroponic crops has been described [54] after the addition of biochar [55] and olive mark compost [56]. In this respect, the activation of systemic defense responses against *B. cinerea*, thanks to beneficial bacteria and fungi has also been described.

4. Bacteria as Inductors of Plant Resistance against *B. cinerea*

Plant-microbe interactions play an important role in nutrient mobilization and protection against pathogens and are crucial for proper growth and development. In the interaction between microbes and plants, microbes release different elicitors that trigger physiological and biochemical changes in plants. These changes lead to disease resistance in plant for several months [57]. In this regard, the ability of beneficial plant bacteria, such as PGPRs, to induce systemic plant resistance to pathogens and pests in different crops has been widely reported in recent decades [58], as reported in Table 1.

Table 1. Systemic resistance-inducing bacteria against *B. cinerea*.

Species	Plant	Experimental Conditions		Hormonal Pathway Involved	Plant Defensive Responses	Reference
Acinetobacter lwoffii	Grapevine	Field	Root inoculation	Unidentified	Induction of chitinase and β-1,3 glucanase activity	[59]
Bacillus amyloliquefaciens	Tobacco	Greenhouse	Leaf inoculation	SA and JA	Enhancement of *PR-1a*, *PR1b PR-5*, *PAL*, *NPR1*, *PDF1.2*, and *COI1* expression	[60]
	Tomato	Greenhouse	Root inoculation	Unidentified	Enhancement of *PR2a* and *Chi3* expression	[61]
	Strawberry	Greenhouse	Root inoculation	SA	Enhancement of *PR1* and *β-1,3-glucanase* expression	[62]
	Tomato	Greenhouse	Root inoculation	Unidentified	Not described	[17]
	Arabidopsis	Growth chamber	Root inoculation	SA and JA/ET	Enhancement of *β-1,3-glucanase* expression	[63]
Bacillus cereus	Tobacco Maize	Greenhouse	Root inoculation	Unidentified	Not described	[64]
	Arabidopsis	Growth chamber	Root inoculation	JA/ET	Enhancement of *PR1* expression, hydrogen peroxide accumulation and callose deposition	[65]
Bacillus subtilis	Bean Tomato	Greenhouse	Root inoculation	JA	Induce LOX and LHP activity	[66]
	Arabidopsis	Growth chamber	Root inoculation	SA and JA	Enhancement of *PR1* and *PDF1.2* expression	[67]
	Tomato	Greenhouse	Root inoculation	Unidentified	Enhancement of *PR2a* and *Chi3* expression	[61]
Bacillus thuringiensis	Tomato	Greenhouse	Root inoculation	SA	Enhancement of *PR1* expression	[68]
Bacillus velezensis	Pepper	Greenhouse	Root inoculation	SA	Induction of hydrogen peroxide accumulation and SOD, CAT, and POD activity	[69]
	Tomato Strawberry	Greenhouse	Root inoculation	JA/ET	Reduce oxidative damage and induce callose deposition	[70]
Brevibacillus laterosporus	Tobacco	Growth chamber	Leaf inoculation	Unidentified	Induction of SOD and POD activity	[71]
Burkholderia cepacia	Grapevine	Growth chamber	Root inoculation	Unidentified	Not described	[72]
Burkholderia phytofirmans	Grapevine	Growth chamber	Root inoculation	SA	Induction of callose deposition, H_2O_2 production and prime expression of *PR1*, *PR2*, and *PR5*	[73]
Cupriavidus campinensis	Arabidopsis	Greenhouse	Root inoculation	SA	Reduce oxalate concentration	[74]
Micromonospora spp.	Tomato	Greenhouse	Root inoculation	JA	Enhancement of *LOXa* and *PinII* expression	[75]
Paenibacillus terrae	Tomato	Greenhouse	Root inoculation	SA and JA	Not described	[76]
Pantoea agglomerans	Grapevine	Growth chamber	Root inoculation	Unidentified	Induction of phytoalexin accumulation	[77]
	Grapevine	Field	Root inoculation	Unidentified	Induction of chitinase and β-1,3 glucanase activity	[78]
Pantoea eucalyptii	Arabidopsis	Growth chamber	Root inoculation	JA/ET	Enhancement of callose deposition	[79]
	Tomato	Greenhouse	Root inoculation	SA	Not described	[80]
Pseudomonas aeruginosa	Grapevine	Growth chamber	Leaf inoculation	Unidentified	Enhancement of *Chit4c* expression	[81]
Pseudomonas fluorescens	Grapevine	Growth chamber	Leaf inoculation	SA	Induction of phytoalexin accumulation	[82]
	Grapevine	Field	Root inoculation	Unidentified	Induction of chitinase and β-1,3 glucanase activity	[59]
	Grapevine	Growth chamber	Leaf inoculation	SA	Induction of phytoalexin accumulation	[82]
	Arabidopsis	Growth chamber	Root inoculation	JA/ET	Enhancement of *PDF1.2* expression	[83]
	Grapevine	Growth chamber	Root inoculation	JA/ET	Induction of phytoalexin accumulation	[84]
	Grapevine	Field	Root inoculation	Unidentified	Induction of phytoalexin accumulation	[85]
Pseudomonas putida	Bean	Growth chamber	Root inoculation	JA/ET	Not described	[86]
	Bean	Growth chamber	Root inoculation	JA/ET	Induction of LOX and LHP activity	[87]
	Tomato Bean	Growth chamber	Root inoculation	Unidentified	Not described	[88]
	Tomato	Greenhouse	Root inoculation	JA/ET	Induction of phytoalexin accumulation and LOX activity	[89]
Pseudomonas syringae pv. *phaseolicola*	Chinese cabbage	Greenhouse	Seeds inoculation	Unidentified	Induction of CHI activity	[90]
Saccharothrix algeriensis	Arabidopsis	Growth chamber	Root inoculation	JA/ET	Not described	[91]
Serratia plymuthica	Cucumber	Greenhouse	Root inoculation	Unidentified	Not described	[92]
Streptomyces sp.	Norway spruce	Growth chamber	Root inoculation	Unidentified	Induction POD activity	[93]
	Eucalyptus grandis	Growth chamber	Root inoculation	Unidentified	Induction of PPO and POD activity Induction of total phenolic accumulation	[94]
	Chickpea	Greenhouse	Root inoculation	Unidentified	Induction of PAL, CAT, SOD, PPO, APX, and GPX activity Induction of total phenolic accumulation	[95,96]

APX: ascorbate peroxidase; CAT: catalase; CHI or CHIT: chitinase; COI: coronative insensitive; GPX: glutatión peroxidase; LHP: lipid hydroperoxidase; LOX: lipoxygenase; PDF: plant defensin; PAL: phenylalanine ammonia lyase; PIN: proteinase inhibitor; POD: peroxidase; PPO: polyphenol oxidase; PR: pathogenesis related; SA: salicylic acid; SOD: superoxide dismutase.

In plant defense against *B. cinerea*, the recognition of microbe-associated molecular patterns (MAMPs) by plant cells, such as bacterial flagellin or different *N*-acylated-homoserine lactones, is capable of pre-activating systemic resistance, whereby the plant prepares before a pathogen attack [65,97].

Many *Bacillus* species have proven to be effective against a broad range of plant pathogens. They have been reported as plant growth promoters and systemic resistance inducers and are used for production of a broad range of antimicrobial compounds (lipopeptides, antibiotics, and enzymes) and competitors for growth factors (space and nutrients) with other pathogenic microorganisms through colonization. In general, by colonizing the roots, *Bacillus* is capable of inducing plant systemic resistance involving phenolic compounds, genetic and structural modifications, plant resistance activators, and the activation of enzymatic weapons [98]. Against *B. cinerea*, several *Bacillus* species have been described to have the ability to pre-activate systemic resistance through different mechanisms. Without identifying the hormonal pathway involved, studies have reported that *B. amyloliquefaciens* and *B. cereus* are capable of promoting the plant growth of tomato seedlings and controlling *B. cinerea* by increasing the expression of pathogenesis-related genes, such as *PR2a* and *Chi3* [61]. This could be due to the production of microbial elicitors, such as VOC dimethyl disulfide, from *B. cereus*, which has been shown to significantly protect tobacco and maize plants against necrotrophic pathogens [64]. In *B. cinerea* control, the biocontrol agent can use direct control and activation of plant defenses in conjunction. For example, *B. amyloliquefaciens*, when applied to the roots and leaves of tomato plants, synergistically increases the control capacity against *B. cinerea* [17].

Through the SA pathway, the increase in the expression of *PR1* and *β-1,3-glucanase* genes was determined to be effective against *B. cinerea* in the leaves of strawberry and tomato plants. This defensive response against *B. cinerea* is a consequence of the root inoculation of *B. amyloliquefaciens* and *B. thuringiensis*, respectively, which activates a priming before the attack of the pathogen [62,68]. Similarly, the inoculation of *B. velezensis* in pepper roots is capable of causing hydrogen peroxide accumulation and superoxide dismutase (SOD), catalase (CAT), and peroxidase (POD) activity in leaves [69]. On the other hand, the JA/ET pathway reduced disease incidence and severity by 50% and 60% in tomato and strawberry leaves, respectively, due to the reduction in oxidative damage and the induction of callose deposition by *B. velezensis* root inoculation [70], as well as by *B. cereus* in *A. thaliana* roots [65,99]. This systemic resistance can be activated by the recognition of bacterial lipopeptides, such as surfactins and fengycins, which are recognized in bean and tomato plants when *B. subtilis* is applied radicularly, systemically increasing lipoxygenase (LOX) and lipid hydroperoxidase (LHP) activity against the necrotrophic pathogen [66]. Moreover, the produced systemic defense response can be mediated by both the SA and JA/ET pathways, activating the expression of genes independently, such as *PR1* (SA) and *PDF1.2* (JA) [60,67], as well as common genes, such as *β-1,3-glucanase* [63].

Pseudomonas is a bacteria genus widely studied as a root colonizer and has been the subject of several reviews on its plant growth-promoting capacity and biocontrol potential, with high interest in an agricultural setting [100]. In the 1990s, several species within the genus were described to have the ability to induce systemic plant resistance by colonizing the roots, and there are currently many studies on different plant species and against different biotic stresses [101]. Against *B. cinerea*, the ability to activate systemic resistance via *P. fluorescens* has been reported in both the leaves and fruits of grapevine plants in the field due to an increase in chitinase and β-1,3 glucanase activity [59] and in the production of phytoalexins [85]. This is due to the perception of MAMPs by plant cells, mainly within the group of lipopolysaccharides [88], such as rhamnolipids, used by *P. aeruginosa* as biosurfactants that induce the expression of the *Chit4c* gene in the leaves of grapevine plants [81]. An increase in chitinolytic activity was also reported in Chinese cabbage leaves after *P. syringae* pv. *phaseolicola* colonized the plant tissues systemically [90]. In this sense, it was proven that, when colonizing roots, *P. aeruginosa* releases SA and the elicitors pyochelin and phenazine, which cause the systemic activation of plant defenses through the SA-pathway in tomato [80], inducing a response of the priming type via the accumulation of phytoalexins in grapevine leaves [82]. Despite this, most of the systemic defensive responses reported

for *Pseudomonas* against *B. cinerea* were carried out through JA/ET-pathway. These responses are related to an increase in JA-related gene expression in leaves, such as *PDF1.2* by *P. fluorescens* [83], the increase in LOX and LHP activity by *P putida* [86,87,89], and the accumulation of phytoalexins by both bacterial species [84,89].

Streptomyces are an aerobic and filamentous bacterial genus in which the species colonize plant tissues from the roots to the aerial parts. These bacteria are active producers of antibiotics and volatile organic compounds, both in soil and *in planta*, and this feature is helpful for identifying active antagonists of plant pathogens; these bacteria can also be used in several cropping systems as biocontrol agents [102]. This includes crops, such as chickpea, in which the ability to systemically increase the activity of different antioxidant enzymes and increase the total phenolic content against *B. cinerea* has also been reported [95,96]. This activity was also reported in forest crops, such as eucalyptus [94], and Norway spruces [93].

Pantoea agglomerans has been identified as an antagonist of many plant pathogens belonging to bacteria and fungi as a result of antibiotic production [103]. *P. agglomerans* is found on grapevine roots, and is capable of inducing systemic resistance against attacks from *B. cinerea*, both *in vitro* and in the field, on both its leaves and fruits due to an increase in the synthesis of phytoalexins and chitinase and β-1,3 glucanase activity [77,78]. This defensive induction can be carried out by means of the JA/ET-pathway, as happens with *P. eucalyptii* in *A. thaliana*, which is capable of reducing the size of the necrotic lesions caused by *B. cinerea* by up to 60% due to the foliar deposition of callose [79].

Other systemic defensive responses have been reported with the application of other bacterial species. *B. cinerea* secretes oxalic acid as a pathogenicity factor with a broad action, against which SA-mediated systemic action has been observed after inoculation of *A. thaliana* roots with *Cupriavidus campinensis* [74]. This has also been observed in tomato through the JA-pathway after root inoculation with *Micromonospora* spp., thereby increasing the expression of genes coding for LOX and proteinase inhibitors (PIN) [75].

5. Fungi as Inductors of Plant Resistance against *B. cinerea*

As with bacteria, there are numerous groups of beneficial fungi described with the ability to activate systemic plant resistance against biotic stresses. These fungi belong to the so-called PGPFs, which include mycorrhizal fungi and other rhizospheric and/or endophytic fungi that belong, for example, to the genera *Aspergillus*, *Fusarium*, *Penicillium*, *Piriformospora*, *Phoma,* and *Trichoderma* [104]. In the *B. cinerea* control, reduction of the disease due to the activation of systemic plant resistance by groups of filamentous fungi and yeasts has been reported in several studies (Table 2).

Table 2. Systemic resistance-inducing fungi against *B. cinerea*.

Species	Plant	Experimental Conditions		Hormonal Pathway Involved	Plant Defensive Responses	Reference
Clonostachys rosea	Tomato	Greenhouse	Leaf inoculation	JA	Enhancement of PAL and PPO activity	[105]
Colletotrichum acutatum	Arabidopsis	Growth chamber	Root inoculation	Unidentified	Enhancement of *PR1* expression, and callose deposition	[106]
	Strawberry	Growth chamber	Root inoculation	JA/ET	Not described	[107]
Colletotrichum fragariae	Strawberry	Greenhouse	Root inoculation	SA	Hydrogen peroxide accumulation and callose deposition	[108]
Funneliformis mosseae	Tomato	Greenhouse	Root inoculation	Unidentified	Not described	[109]
Fusarium oxysporum	Tomato	Greenhouse	Root inoculation	Unidentified	Enhancement of *PR* gene expression	[110]
	Pepper	Greenhouse	Root inoculation	Unidentified	Enhancement *PR-1* expression	[111]
Fusarium oxysporum f. sp. *lycopersici*	Pepper	Greenhouse	Root inoculation	JA/ET	Enhancement chitinase activity	[112]
Hanseniaspora opuntiae	Arabidopsis	Growth chamber	Leaf inoculation	JA/ET	Enhancement *ACS6*, *PR4*, and *PDF1.2* expression	[113]
Piriformospora indica	Chickpea	Growth chamber	Root inoculation	Unidentified	Enhancement GST activity	[114]
Pseudozyma aphidis	Arabidopsis	Growth chamber	Leaf inoculation	JA/ET	Enhancement *PR1* and *PDF1.2* expression	[115]
Rhizophagus irregularis	Tomato	Greenhouse	Root inoculation	JA	Indolic derivative and phenolic compound accumulation	[116]
	Tomato	Greenhouse	Root inoculation	JA	Lignan and oxylipin accumulation	[117]
	Tomato	Greenhouse	Root inoculation	JA	Increased callose deposition	[118]
Saccharomyces cerevisiae	Arabidopsis	Growth chamber	Leaf inoculation	SA	Enhancement of *PR1*, *PR2*, and *PR5* expression, and phytoalexin camalexin accumulation	[119]
Trichoderma asperellum	Tomato	Greenhouse	Root inoculation	Unidentified	Inhibit ROS production	[120]
	Tomato	Growth chamber	Root inoculation	SA	Not described	[121]
	Arabidopsis	Growth chamber	Root inoculation	JA	Enhancement of *VSP2* and *PDF1.2* expression	[122]
	Bean	Growth chamber	Root inoculation	Unidentified	Not described	[123]
	Bean	Growth chamber	Root inoculation	SA	Enhancement of thaumatin-like protein activity	[124]
Trichoderma atroviride	Arabidopsis	Growth chamber	Root inoculation	SA and JA	Hydrogen peroxide and camalexin accumulation	[125]
	Arabidopsis	Growth chamber	Root inoculation	SA and JA	Enhancement of *PR-1a*, *PR-2*, *PDF1.2*, *LOX1*, *peroxidase*, and *camalexin-synthesis-enzyme* expression	[126]
	Tomato	Greenhouse	Root inoculation	SA and JA	Enhancement of *PR1b1*, *LOXa*, and *PINI* expression	[127]
	Tomato	Greenhouse	Root inoculation	SA and JA	Enhancement of *peroxidase* and *α-dioxygenase* expression	[128]
Trichoderma hamatum	Geranium	Greenhouse	Root inoculation	Unidentified	Not described	[129]
	Arabidopsis	Growth chamber	Root inoculation	JA	Phenylpropanoids accumulation	[130]
	Arabidopsis	Growth chamber	Root inoculation	JA/ET	Not described	[131]
	Tomato	Greenhouse	Root inoculation	SA and JA	Enhancement of *PR1b1*, *LOXa*, and *PINI* expression	[127]
Trichoderma harzianum	Arabidopsis	Growth chamber	Root inoculation	JA/ET	Enhancement of *PDF1.2* expression	[83]
	Tomato	Greenhouse	Root inoculation	JA	Enhancement of *PINII* expression	[132]
	Tomato	Greenhouse	Root inoculation	JA	Enhancement of *Chi9* expression	[133]
	Tomato	Greenhouse	Root inoculation	SA	Enhancement of *PR-2* and *PINII* expression	[134]
	Arabidopsis	Growth chamber	Root inoculation	JA	Enhancement *PDF1.2* expression	[122]
Trichoderma koningiopsis	Tomato	Greenhouse	Root inoculation	Unidentified	Not described	[135]
Trichoderma pseudokoningii	Moth orchid	Growth chamber	Root inoculation	Unidentified	Enhancement POD, PPO, PAL, SOD and CAT activity	[136]
Trichoderma virens	Arabidopsis	Growth chamber	Root inoculation	SA and JA	Hydrogen peroxide and camalexin accumulation	[125]
	Tomato	Greenhouse	Root inoculation	SA and JA	Enhancement *peroxidase* and *α-dioxygenase* expression	[128]
	Tomato	Greenhouse	Root inoculation	Unidentified	Increase pectin content of cell walls	[137]

ACS: 1-aminocyclopropane-1-carboxylate synthase; CAT: catalase; CHI: chitinase; GST: glutathione S-transferases; LOX: lipoxygenase; PAL: phenylalanine ammonia lyase; PDF: plant defensin; PIN: proteinase inhibitor; POD: peroxidase; PPO: polyphenol oxidase; PR: pathogenesis related; ROS: reactive oxygen species; SOD: superoxide dismutase; VSP: vegetative storage protein.

The filamentous-fungal genus *Trichoderma* includes several species that colonize the outer layers of the roots [138]. Thanks to this interaction, *Trichoderma* favors the acquisition of nutrients by modifying the root architecture and releasing different molecules to the rhizosphere, which leads to a significant increase in crop productivity [139]. Moreover, *Trichoderma* is capable of increasing plant tolerance to abiotic stresses, such as salinity and drought [140]. Regarding the activation of systemic resistance in plants, when in contact with the roots, *Trichoderma* is capable of activating a defensive response in all plant organs, which has been widely described in many different crops and against a wide variety of pathogens [141]. Against *B. cinerea*, different *Trhichoderma* species are capable of promoting plant growth while inducing a priming-type systemic defensive response by inhibiting ROS production [120], increasing the pectin content of cell walls [137], or increasing the gene expression of the enzymatic activity of POD, PPO, PAL, SOD, and CAT [136].

This systemic activation has been described as SA-mediated, with 35% less disease severity in tomato leaves by *T. asperellum* [121]. The SA-mediated response is elicited when *Trichoderma* comes into contact with the roots and releases molecules, such as cyclophilins, thereby increasing thaumatin-like protein activity in bean leaves [124] and *PR-2* and *PINII* expression in tomato [134]. For the JA/ET-mediated systemic response, reductions in the severity of the disease greater than 60% have been reported as a consequence of the induction in the expression of genes, such as *Chi9* [133], *VSP2*, *PDF1.2* [83,122], and *PINII* [132], as well as the leaf-accumulation of phenylpropanoids [130]. This mediated JA/ET defensive activation is due to the plant-perception of VOCs emitted by *Trichoderma*, which results in a priming-type response and greater absorption of iron by the roots [122]. However, a significant number of studies were carried out on *Trichoderma* plant–*B. cinerea* interactions with *A. thaliana* and tomato plants, in which the systemic defensive responses were shown to be SA- and JA/ET-mediated. Thus, the systemic induction of the expression of genes related to both routes, such as *PR*, *PDF*, *LOX*, and *PIN* genes, was verified [126,127], in addition to hydrogen peroxide and camalexin leaf-accumulation [125] due to an increase in the expression of genes encoding for peroxidases and α-dioxygenases [128]. Therefore, *Trichoderma* is an efficient tool for the biocontrol of *B. cinerea* through different mechanisms, including the activation of systemic resistance. Moreover, it has been observed that, in tomato plants attacked by necrotrophic fungus, there is an increase in the rhizosphere populations of *T. asperellum* due to the directed secretion of compounds by the roots [142].

Mainly used as biofertilizers, mycorrhizal fungi are obligate symbionts of the roots in 97% of the vascular plants. Mycorrhizal hyphae are able to colonize places in the soil where plant roots could never reach. Moreover, hyphae have the ability to absorb nutrients through active transporters. The fungus contributes mostly to the supply of phosphorus to the plant, but other nutrients with low mobility, such as ammonium, potassium, copper, iron, sulfur, molybdenum, and zinc, also contribute. In response, the plant must provide carbohydrates to the fungus to meet its needs, although this does not have a negative impact on the plant due to photosynthetic compensation with the fungal supply of nutrients and reduced root development. Moreover, it is widely believed that the inoculation of mycorrhizal fungi provides tolerance to host plants against various stresses, like heat, salinity, drought, pollution, and extreme temperatures. Once symbiosis is established, mycorrizal fungi-induced resistance and priming regulated by JA become activated, similar to the responses controlled by the JA and ET pathways against necrotrophic pathogens [141]. As far as plant systemic resistance against *B. cinerea* is concerned, a reduction in disease index of up to 50% was achieved in tomato plant roots inoculated with the mycorrhizal fungus *Funneliformis mosseae* [109]. This was due to a JA-mediated plant defensive response through localized callose deposition [118] alongside the accumulation of indolic derivates and phenolic compounds [116] and/or lignans and oxylipins [117], observed in tomato plants interacting with *Rhizophagus irregularis*.

Endophytic filamentous fungi include fungi that can be isolated from plant tissues once they have been superficially disinfected and do not cause visible damage to plants. This group plays an important role in ecosystems, returning nutrients to the soil once plants die and protecting plants against biotic and abiotic stresses. In this regard, endophytic fungi are able to induce SAR and ISR in

plants against the attacks of pests and/or pathogens, but they also need to suppress, at least partially, the defenses of the plants to colonize their tissues [141]. In *B. cinerea* biocontrol, several species of filamentous endophytic fungi have been reported with the ability to systemically activate plant defenses. In tomato and pepper, plants root colonized by *Fusarium oxysporum* achieved a reduction in the percentage of diseased plants and the appearance and intensity of symptoms, thanks to an increase in the foliar expression of *PR* genes [110,111] and chitinase activity [112]. The JA-mediated response also reported under colonization by *Clonostachys rosea* is capable of systemically increasing PAL and PPO activity [105]. However, for *Colletotrichum acutatum* and *C. fragariae*, this is an SA-mediated response, causing a systemic increase in *PR-1* expression, hydrogen peroxide accumulation, and callose deposition in *A. thaliana* and strawberry plants [106,108]. In addition, as verified in chickpea plants with the endophyte *Piriformospora indica*, greater control of the disease is directly related to greater root colonization [114].

Yeasts are single-celled microbes classified as members of the kingdom fungi. Today, the role of yeasts as plant growth-promoters and biocontrol agents in agriculture is increasingly understood [143]. The ability of different yeasts to activate post-harvest defenses is widely known [39]; for example, the application of *Aureobasidium pullulans* in strawberry fruits significantly reduced infection by *B. cinerea* [144], which was reported in tomato fruits to be a consequence of the perception of chitin present in the *Saccharomyces cerevisiae* cell walls, leading to an increase in the activity of SOD, CAT, POD, PAL, β-1,3-glucanase, and chitinase enzymes through the SA-pathway [145]. In this sense, the abilities of different yeasts to activate systemic plant resistance against the necrotrophic pathogen have been described. All the studies carried out to date have used *A. thaliana* as a model plant, reporting a significant in the systemic expression of JA/ET-related genes, such as *ACS6*, *PR4*, and *PDF1.2*, after the application of yeasts, such as *Hanseniaspora opuntiae* and *Pseudozyma aphidis*, on leaves [113,115]. The plant response elicited by the components of the fungal cell wall, like that under the foliar application of autoclaved *S. cerevisiae* cells, increases the systemic expression of *PR* genes and the accumulation of the phytoalexin camalexin via the SA-pathway [119].

Finally, although they are not found within the fungi kingdom, several examples of oomycetes have been reported to have the ability to induce systemic plant resistance against *B. cinerea* by colonizing the roots. Specifically, *Pythium oligandrum* has been described to increase tomato yield by colonizing its roots. This is due to several mechanisms, including the ability to activate plant systemic defenses against pathogens, such as *B. cinerea*, thanks to an increase in the expression of *PR* genes [110,146] and due to the root perception of oomycete-secreted proteins, like oligandrin [147].

6. Conclusions

Botrytis cinerea is a necrotrophic phytopathogenic fungus that causes serious economic and agronomic losses worldwide. The use of chemical fungicides cannot alleviate the persistence of this fungus, in addition to the serious damage it causes to the environment and human health. For this reason, in recent decades, many biological control strategies have been developed against this pathogen, with antagonist bacteria and fungi as the main interest groups.

Different groups of beneficial bacteria and fungi, such as *Bacillus*, *Pseudomonas*, *Aureobasidium*, and *Trichoderma*, have been described as efficient direct antagonists of the growth and development of *B. cinerea* through parasitism, antibiosis, and competition. Thus, future lines of research should be developed to identify new antifungal compounds (also those within VOCs) and search for new groups of antagonistic microorganisms.

Moreover, beneficial bacteria and fungi are both capable of activating a systemic defensive response against *B. cinerea* when recognized by plant cells. This defensive response leads to significant reductions in the incidence of the disease in different crops, thus providing a good alternative to the use of agricultural chemicals. In addition, these microorganisms can be effective against necrotrophic fungus both directly and through the activation of systemic plant resistance (as occurred with many of

the reviewed examples), which significantly increased the effectiveness of the use of bacteria and fungi in the biocontrol of *B. cinerea*.

For the phytohormonal pathway activated by bacteria and fungi against *B. cinerea*, SA-mediated, JA/ET-mediated, and SA- and JA/ET-mediated responses have been reported. In this sense, the plant defense responses against necrotrophic pathogens through JA/ET pathway and the responses against biotrophic pathogens through the SA pathway, mainly through ISR and SAR, respectively, are becoming increasingly less clearly differentiated. Understanding the crosstalk complexes between both hormonal pathways and the rest of the plant hormones is essential for the development of targeted and effective biocontrol strategies against *B. cinerea*. For this reason, the development of new research that delves into transcriptomics, proteomics, and metabolomics linked to the microbial activation of systemic resistance against necrotrophic fungus is necessary.

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