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Emerging Trends in Molecular Interactions between Plants and the Broad Host Range Fungal Pathogens *Botrytis cinerea* and *Sclerotinia sclerotiorum*

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Fungal plant pathogens are major threats to food security worldwide. *Sclerotinia sclerotiorum* and *Botrytis cinerea* are closely related Ascomycete plant pathogens causing mold diseases on hundreds of plant species. There is no genetic source of complete plant resistance to these broad host range pathogens known to date. Instead, natural plant populations show a continuum of resistance levels controlled by multiple genes, a phenotype designated as quantitative disease resistance. Little is known about the molecular mechanisms controlling the interaction between plants and *S. sclerotiorum* and *B. cinerea* but significant advances were made on this topic in the last years. This minireview highlights a selection of nine themes that emerged in recent research reports on the molecular bases of plant-*S. sclerotiorum* and plant-*B. cinerea* interactions. On the fungal side, this includes progress on understanding the role of oxalic acid, on the study of fungal small secreted proteins. Next, we discuss the exchanges of small RNA between organisms and the control of cell death in plant and fungi during pathogenic interactions. Finally on the plant side, we highlight defense priming by mechanical signals, the characterization of plant Receptor-like proteins and the hormone abscisic acid in the response to *B. cinerea* and *S. sclerotiorum*, the role of plant general transcription machinery and plant small bioactive peptides. These represent nine trends we selected as remarkable in our understanding of fungal molecules causing disease and plant mechanisms associated with disease resistance to two devastating broad host range fungi.

Keywords: fungal pathogen, necrotroph, broad host range, quantitative disease resistance, virulence

INTRODUCTION

A majority of studies on plant interactions with fungal pathogens over the last years have focused on specialized host-pathogen interactions. For instance the powdery mildew fungus *Blumeria graminis*, the cereal rust fungi of the *Puccinia* spp., and the corn smut fungus *Ustilago maydis* are among the most studied fungal pathogens and are obligate biotrophic pathogens restricted to a

single host genus (Dean et al., 2012). Such interactions only represents a fraction of plant-fungal pathogen interactions encountered in nature and a number of broad host range fungal pathogens also are major threats for food security (Barrett et al., 2009; Dean et al., 2012). Understanding how broad host range pathogens successfully infect multiple plant lineages is a major challenge in plant pathology (Dong et al., 2015).

Among Leotiomycete, the gray mold fungus *Botrytis cinerea* and the white mold fungus *Sclerotinia sclerotiorum* stand out for having a remarkably broad host range, encompassing over 200 species. Each of these pathogens causes yearly several 100 millions of US dollars crop losses worldwide (Bolton et al., 2006; Dean et al., 2012). They are considered as typical necrotrophs, secreting an arsenal of cell wall-degrading enzymes, and toxins to kill host cells and derive energy. Host plants typically exhibit quantitative disease resistance (QDR) to *B. cinerea* and *S. sclerotiorum*, leading to a reduction rather than absence of disease (Roux et al., 2014). How these broad host range fungal pathogens cause disease and what are the genetic bases of plant QDR is still poorly understood.

In recent years, remarkable progress has been achieved in the characterization of fungal virulence factors and the dissection of plant response mechanisms. In this minireview, we chose to report on nine advances specifically related to pathosystems involving *B. cinerea* or *S. sclerotiorum*, concerning either the molecular bases of fungal virulence or plant QDR (summarized in **Figure 1**). The nine points presented hereafter are not meant to represent a complete overview of our current knowledge, and a number of significant discoveries could not be covered in this article. We selected nine trends based on convergent findings from multiple studies and as a source of inspiration for future studies on plant interactions with broad host range fungal pathogens.

PUTTING THE ROLE OF FUNGAL OXALATE SECRETION TO THE ACID TEST

Oxalic acid (OA) is considered as a major virulence factor in species of the *Sclerotiniaceae* (Hegedus and Rimmer, 2005; Andrew et al., 2012). This central role of OA is further supported by the association of plant OA oxidase-related enzymes with disease resistance (Foster et al., 2012; Rietz et al., 2012). The roles of OA secretion in *S. sclerotiorum* virulence are still a matter of debate and likely include the suppression of plant defenses (Cessna et al., 2000; Williams et al., 2011), the induction of plant programmed cell death (PCD; Kim et al., 2008), the deregulation of guard cells function (Guimaraes and Stotz, 2004), and calcium detoxification (Heller and Witt-Geiges, 2013). Evidences for several of these conclusions were obtained using a UV-induced OA-deficient mutant of *S. sclerotiorum* (Godoy et al., 1990). Recently, Liang et al. (2015) generated disruptive mutants in the gene encoding oxaloacetate acetylhydrolase 1 (OAH1), an enzyme mediating OA biosynthesis (Joosten et al., 2008). This *oah1* mutant was completely abolished in OA synthesis and impaired in pathogenicity. Several phenotypic differences

were noted compared to the UV-induced OA-deficient mutants, prompting for a re-evaluation of OA function during host colonization (Liang et al., 2015). To this end, Xu et al. (2015) generated *oah1* deletion mutants in another *S. sclerotiorum* strain and confirmed that their virulence varied with the pH of the host tissues. The virulence phenotype of *oah1* mutants was restored by genetic complementation and partially restored by the exogenous acidification of host tissue (Liang et al., 2015; Xu et al., 2015), suggesting that low pH, rather than a specific acidic molecule, is required for *S. sclerotiorum* full virulence. Why acidification is mediated by OA and not another organic acid during *S. sclerotiorum* infection remains unclear. *B. cinerea* was proposed to rely primarily on the production of citrate and succinate during the colonization of sunflower. The production of different organic acids suggests that *S. sclerotiorum* and *B. cinerea* differ significantly in TCA cycle regulation (Billon-Grand et al., 2012). These findings call for investigations on metabolic organization in *S. sclerotiorum* and *B. cinerea*.

THE PLENTY AND HAZY FUNGAL SMALL SECRETED PROTEINS

Secondary metabolite toxins and cell wall-degrading enzymes are known to contribute to virulence of necrotrophic fungi (Choquer et al., 2007; Kabbage et al., 2015), but knowledge on the repertoires and mode of action of *S. sclerotiorum* and *B. cinerea* proteins secreted during host colonization remains limited. Mutant screens for pathogenicity defects in *S. sclerotiorum* have identified the secreted putative Ca²⁺-binding protein SsCAF1 and the secreted Cu/Zn superoxide dismutase SsSOD1 (Xu and Chen, 2013; Xiao et al., 2014). The analysis of genes down-regulated in a hypovirulent *S. sclerotiorum* strain identified the secreted integrin-like gene *SsITL* as required for full virulence (Zhu et al., 2013). Genomic and transcriptomic approaches have revealed *S. sclerotiorum* cutinase *SsCUT1* (Zhang H. et al., 2014) and the secreted cyanovirin-N homolog *SsCVNH* (Lyu et al., 2015) to be associated with plant infection. Proteomic analyses of *B. cinerea* secretome and cell wall proteome led to the identification the cerato-platanin BcSPL1 required for full virulence (Espino et al., 2010; Friás et al., 2011, 2014). SsCAF1, SsCUT1, and BcSPL1 elicited cell death in plants whereas *SsITL* was proposed to suppress plant resistance pathways, illustrating the diverse activities of fungal small secreted proteins. Given that mutations in *SsCAF1*, *SsITL*, and *SsCVNH* caused morphological defects *in vitro*, it is unclear whether these genes are directly linked to host interaction. Since the release of genome sequences for *S. sclerotiorum* and *B. cinerea* (Amselem et al., 2011), bioinformatics analyses aimed at systematically identifying candidate proteins associated with virulence. Aguilera et al. (2012) identified 21 *B. cinerea* genes harboring signatures of positive selection, including 7 encoding predicted secreted proteins. Multicriterion analyses of *S. sclerotiorum* and *B. cinerea* secretomes highlighted over 400 secreted proteins including nearly 80 virulence factor candidates (Guyon et al., 2014; Heard et al., 2015). Specific patterns of amino acid usage and conformation may also serve as a filter to classify virulence factor

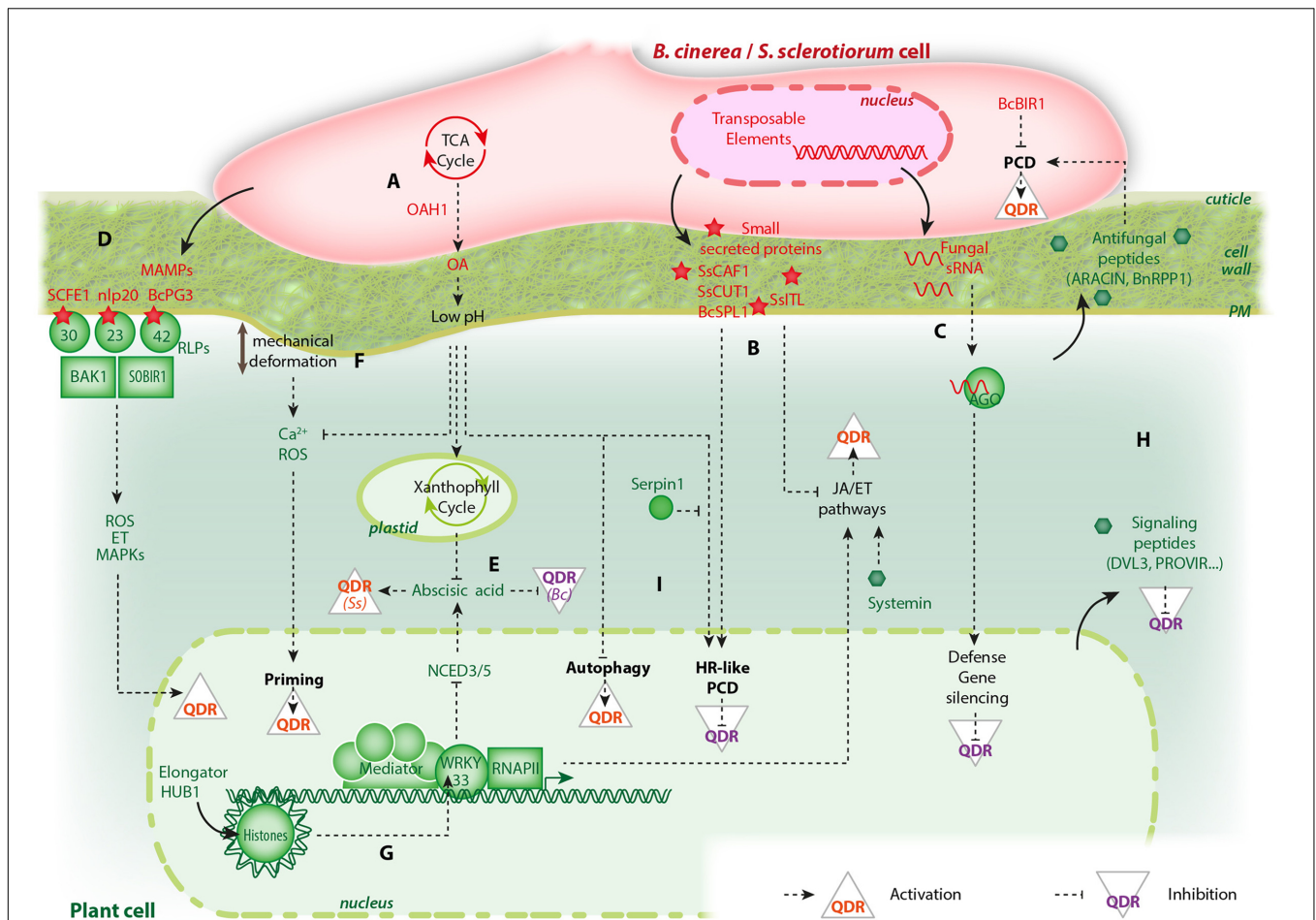


FIGURE 1 | An overview of the molecular players involved in *Botrytis cinerea*/*Sclerotinia sclerotiorum* interactions with plants and their effect on quantitative disease resistance (QDR). Only pathways discussed in this review are shown, some elements were omitted for clarity. Fungal molecules are shown in red, plant molecules in green. **(A)** Effects of oxalic acid (OA) biosynthesis and secretion by fungi. **(B)** Small proteins secreted by fungi can activate hypersensitive response (HR)-like programmed cell death (PCD) or suppress jasmonic acid (JA) and ethylene (ET) signaling pathways to suppress QDR. **(C)** Fungal small RNAs hijack plant argonaute (AGO) proteins to suppress QDR. **(D)** Perception of microbe associated molecular patterns (MAMPs) such as SCFE1, nlp20, and BcPG3 by receptor-like proteins (RLPs) activate QDR. **(E)** The plant hormone abscisic acid can either activate QDR against *S. sclerotiorum* (Ss) or suppress QDR against *B. cinerea* (Bc). **(F)** Mechanical deformation caused by appressorium formation and fungal colonization of plant tissues prime plant cells for QDR through calcium and reactive oxygen species (ROS). **(G)** The plant general transcription machinery involves multiprotein complexes such as Elongator and Mediator that recruit the RNA polymerase II (RNAPII) to modulate gene expression upon fungal challenge. Some histone post-translational modifications are epigenetic marks altered after inoculation that regulate the activity of plant general transcription factors and control QDR. **(H)** Plant small bioactive peptides (green hexagons) have contrasted effects on QDR. **(I)** PCD in plant cells can either have a positive or negative effect on QDR depending on the type of cell death program activated.

candidates (Badet et al., 2015). Such bioinformatics approaches remain nevertheless challenging in fungi (Sperschneider et al., 2015). Functional analyses, improved genome annotation and extensive gene expression analyses should prove useful in understanding the role of small secreted proteins in *B. cinerea* and *S. sclerotiorum* virulence.

HOST AND PATHOGEN CELL DEATH CONTROL

Pathogen recognition often triggers a hypersensitive response (HR), the rapid release of antimicrobial compounds and local

PCD. The HR efficiently suppresses the growth of most biotrophic pathogens while facilitating plant colonization by *B. cinerea* and *S. sclerotiorum* (Govrin and Levine, 2000). The secretion of OA and cell death-eliciting proteins by *B. cinerea* and *S. sclerotiorum* were proposed to promote virulence by inducing host PCD (Kim et al., 2008; Frías et al., 2011). The molecular mechanisms by which *B. cinerea* and *S. sclerotiorum* manipulate host PCD remain, however, elusive. The protease inhibitor AtSerpin1 limits cell death induction by OA and plant colonization by *B. cinerea* and *S. sclerotiorum* by modulating the activity of the cysteine protease RD21 (Lampl et al., 2013). In addition, *Arabidopsis* mutants impaired in autophagy, a form of PCD involving the degradation of cytoplasmic

components, were more susceptible to *B. cinerea* (Lai et al., 2011). Furthermore, *S. sclerotiorum* OA-deficient mutants trigger restricted autophagic cell death, suggesting that OA may suppress autophagy in host cells (Kabbage et al., 2013). In *Arabidopsis acd5* mutant, ceramides were associated with autophagy and enhanced susceptibility to *B. cinerea*, but these two activities may be independent (Bi et al., 2014; Magnin-Robert et al., 2015). These findings indicate that certain mechanisms of host cell death may be favorable to necrotrophic fungi whereas others would be detrimental (Dickman and de Figueiredo, 2013). The control of PCD in fungal cells also plays a crucial role in pathogenicity. Massive fungal PCD was observed at the early stages of *B. cinerea* infection, and the anti-apoptotic protein BcBIR1 was found to positively contribute to *B. cinerea* virulence. These findings suggest that fungal PCD could be triggered by plant defense molecules, and that fungal anti-apoptotic machinery is required to prevent it (Shlezinger et al., 2011). This places the control of host and fungal cell death programs at the center of the arms race taking place during plant interactions with necrotrophic fungi.

TWO-WAYS GENE SILENCING BY SMALL RNAs

In eukaryotic cells, small RNAs (sRNAs) regulate a large number of biological processes, from development to immunity and pathogen virulence. These sRNAs trigger multiple RNA interference (RNAi) pathways ultimately leading to gene silencing. Using *Arabidopsis* and tomato plants infected by *B. cinerea*, Weiberg et al. (2013) showed that fungal sRNAs silence specific host immunity genes. Mutants of plant and fungal RNAi components showed reduced silencing of host immunity genes and reduced disease symptoms, respectively, (Weiberg et al., 2013). Together these results support the action of sRNAs across the species and kingdom barrier (cross-kingdom RNAi) in plant–fungi interactions, mediated by a yet unclear transfer mechanism (Sarkies and Miska, 2014; Weiberg et al., 2014, 2015). Several of these *B. cinerea* sRNA effectors originate from loci within *Boty* transposable elements (TEs), a family of mobile genetic elements associated with virulence in *B. cinerea* natural populations (Martinez et al., 2005; Weiberg et al., 2015). This suggests that TEs may contribute to the rapid evolution of sRNA effectors, similar to what has been observed for filamentous pathogen protein effectors (Raffaele et al., 2010; Raffaele and Kamoun, 2012). The genome of *S. sclerotiorum* experienced recent expansion of TEs (Amselem et al., 2011) and sRNA-producing loci have been experimentally identified in this fungus (Zhou et al., 2012). Nevertheless, whether *S. sclerotiorum* uses sRNAs as effectors and the extent to which they contribute to pathogenicity await analysis. Cross-kingdom RNAi has also been exploited to generate transgenic plants producing sRNAs that trigger fungal genes silencing (Nowara et al., 2010; Nunes and Dean, 2012; Koch et al., 2013). This strategy of host-induced gene silencing (HIGS) has been tested in model plants and crops to silence genes of various fungal and oomycete species, providing a promising approach to control diseases and study gene function in non-transformable pathogen species (Koch and Kogel, 2014;

Yin et al., 2014). Whether HIGS occurs in natural plant–fungus interactions and whether it would provide an efficient way to control *B. cinerea* and *S. sclerotiorum* has not been reported yet.

DEFENSE PRIMING BY MECHANICAL SIGNALS

Defense priming consists in establishing a physiological state in which plants are able to mount defense responses more rapidly or more efficiently (Conrath et al., 2006). Priming follows the perception of chemical and molecular signals linked to the presence of microbes interacting with plant cells, and also the perception of physical cues (Conrath, 2011). Plant cells can also perceive strains (mechanical deformation) caused by mechanical loads (Moullia et al., 2015). During their interaction with plants, and prior to plant tissue penetration or degradation, fungal pathogens develop important mechanical loads susceptible to emit mechanical signals (MS) and prime plant defense. Mechanical loads are due to the tremendous turgor pressure (up to 8 MPa) created by water in the vacuole of appressoria and fungal cell wall mechanical properties. This mechanical stress is generally sufficient to penetrate plant cells (Tariq and Jeffries, 1984; Bastmeyer et al., 2002; Sanati Nezhad and Geitmann, 2013). Mechanosensing occurs at the plant cell level and relies on the internal mechanical state of the cell (Coutand, 2010; Hamant, 2013; Monshausen and Haswell, 2013). Mechanosensing is involved in many plant core functions including seed development, morphogenesis, gravitropism, proprioception, and interaction with symbiotic microbes (Boudaoud, 2010; Hamant and Traas, 2010; Bastien et al., 2013; Jayaraman et al., 2014; Creff et al., 2015; Landrein et al., 2015). Recent studies demonstrated the link between mechanosensing and plant immune response to *B. cinerea* in *Arabidopsis thaliana*: plants submitted to MS exhibited higher resistance to fungal infection suggesting a priming effect operated by sterile mechanosensing (Chehab et al., 2012; Benikhlef et al., 2013). Relations between mechanosensing and immune response pathways are mediated by calcium (Chehab et al., 2011; Beneloujaephajri et al., 2013), reactive oxygen species (ROS; Chehab et al., 2012; Benikhlef et al., 2013) and are jasmonic acid (JA)-independent (Benikhlef et al., 2013). Future work should aim at addressing whether mechanosensing for fungal contact or penetration *per se*, in addition to PAMP perception, leads to enhanced plant immunity. Quantifying the MS intensity perceived by the plant will be required to this end. Quantitative biomechanical plant cell models (Barbacci et al., 2013; Ali et al., 2014) are required first milestones toward a better understanding of the molecular mechanisms underlying plant resistance priming by MS.

PERCEPTION OF FUNGI BY PLANT RECEPTOR LIKE PROTEINS

Detection of microbe-associated molecular patterns (MAMPs) is an important part of the plant defense against pathogens

(Boller and Felix, 2009). It relies on plasma membrane resident pattern recognition receptors (PRRs) able to perceive pathogen signatures in the apoplastic space and to activate a downstream signaling through their kinase domain (Monaghan and Zipfel, 2012). In plants, early detection of fungal pathogens occurs mainly through PRRs-mediated perception of chitin (Sanchez-Vallet et al., 2015). Peptide-mediated fungal perception is also becoming increasingly documented. *S. sclerotiorum* produces a protein elicitor (SCFE1) that triggers oxidative burst, ethylene production, mitogen-activated protein kinase activation and gene induction (Zhang et al., 2013). Natural variation among *Arabidopsis* accessions and mutants identified *RLP30* as required for all SCFE1 responses. *RLP30* encodes for a PRR devoid of a kinase domain, suggesting the involvement of co-regulators for intracellular signaling. In a similar approach Albert et al. (2015) screened for *Arabidopsis* mutants and natural accessions unresponsive to nlp20, a 20 amino acid conserved peptide derived from a class of necrosis inducing proteins found in bacteria, oomycetes and fungi (Albert et al., 2015). Upon nlp20 binding, the RLP23 receptor forms a ternary complex required for signaling with two other kinase-containing co-receptors, BAK1 and SOBIR1 (Albert et al., 2015). Finally, the *Arabidopsis* *RLP42* gene is required for *Arabidopsis* responses to exogenous application of *B. cinerea* polygalacturonase 3 (Zhang L. et al., 2014). Given that RLP30, RLP42, and RLP23 are homologs, it is reasonable to hypothesize that all use BAK1 or SOBIR1 as co-receptors. Because SCFE1-responsive plants included *Arabidopsis* accessions with various levels of resistance, Zhang et al. (2013) proposed that redundant elicitor perception systems involve other PRRs in addition to RLP30. This is consistent with several perception and response mechanisms acting simultaneously to trigger QDR (Roux et al., 2014). The discoveries of RLP23, RLP42, and soybean RLPs identified through quantitative trait loci mapping strengthen this view (Zhang L. et al., 2014; Albert et al., 2015; Zhao et al., 2015). Pyramiding several PRRs into plants would likely yield increased and long-lived resistance to devastating pathogens.

CONTRASTED IMPACTS OF ABSCISIC ACID ON QDR

Plant hormones classically associated with resistance to necrotrophic pathogens are JA and ethylene (ET), whereas salicylic acid is associated with stimulation of resistance against biotrophic pathogens (Glazebrook, 2005). Abscisic acid (ABA) has roles in plant development and response to abiotic stress, and has also contrasted impact on plant diseases, depending notably on the pathogen infection strategy (Robert-Seilaniantz et al., 2011). ABA was shown to promote susceptibility to *B. cinerea* in tomato through alterations of the plant cuticle and C:N metabolism (Audenaert et al., 2002; Curvers et al., 2010; Seifi et al., 2013). In *A. thaliana*, Liu et al. (2015) revealed that loss of WRKY33 results in elevated ABA levels and high susceptibility to *B. cinerea*. WRKY33 limits ABA accumulation in *B. cinerea*-challenged plants by binding to the ABA biosynthesis genes *NCED3* and *NCED5* to suppress their expression. Consistently,

the transmembrane receptor-like kinase AtLYK3 was proposed to act as a positive regulator of late responses to ABA and negative regulator of defenses to *B. cinerea* (Paparella et al., 2014). By contrast, inoculation of *A. thaliana* mutants revealed that ABA contributes to resistance to *S. sclerotiorum*, although it remains unclear whether ABA synthesis or perception is required for resistance (Guimaraes and Stotz, 2004; Perchepped et al., 2010). OA secreted by *S. sclerotiorum* was proposed to favor infection by inhibiting ABA-mediated stomatal closure (Guimaraes and Stotz, 2004). Zhou et al. (2015) showed that pH modulation by *S. sclerotiorum* correlates with increased synthesis of photoprotective compounds of the xanthophyll cycle that serve as precursors for ABA synthesis. Depletion in ABA precursors was suggested to account for reduced ABA levels in *S. sclerotiorum*-infected leaves and plant susceptibility (Zhou et al., 2015). Differences in the dynamics of OA secretion (Billon-Grand et al., 2012) may contribute to the contrasting impact of ABA on resistance toward *S. sclerotiorum* and *B. cinerea*. Furthermore, *B. cinerea*, but not *S. sclerotiorum*, synthesizes ABA (Siewers et al., 2004). Further studies will be required to fully understand how plants and fungi interfere with ABA pathways to modulate the outcome of infection.

INTERACTIONS WITH FUNGI SHED LIGHT ON HOST GENERAL TRANSCRIPTION MACHINERY

Host transcriptional reprogramming after pathogen challenge is paramount in the establishment of plant defense. The general transcription machinery relies on the mediator complex, a multiprotein co-activator scaffold acting as a bridge between RNA polymerase II (RNAPII) and transcription factors (Samanta and Thakur, 2015). Plants mutated in some mediator subunits show compromised resistance to both bacterial and fungal pathogens (Zhang et al., 2012). Further, the mediator complex is a target of the HaRL44 downy mildew effector (Caillaud et al., 2013), highlighting its relevance in plant defense. Mutations in MED25 or MED16 subunits abolished the induction of JA-responsive genes and reduced resistance to *B. cinerea* (Kidd et al., 2009; Zhang et al., 2012). MED16 physically associates with the plant defense regulator WRKY33 to recruit RNAPII and activate plant genes involved in JA/ET cross-talks (Wang et al., 2015b). By contrast, mutation in the CDK8 mediator subunit caused enhanced resistance to *B. cinerea* via the regulation of the biosynthesis of cuticular waxes and secondary metabolites (Zhu et al., 2014). The Elongator is another RNAP II-interacting complex required for the induction of JA/ET defense pathways and resistance to *B. cinerea*. The Elongator subunit ELP2 is required for histone acetylation and the induction of WRKY33 and defensin genes, suggesting that Elongator-mediated histone acetylation may be required for full activation of transcriptional responses to *B. cinerea* (Wang et al., 2015a,b). HUB1 encodes a RING E3 ligase that monoubiquitinates histone H2B, interacts with mediator subunit MED21, and positively controls resistance to *B. cinerea* in an ET-dependent manner (Dhawan et al., 2009). Consistently, silencing of HUB1 orthologs in tomato

increased susceptibility to *B. cinerea* and downregulated JA/ET pathway genes (Zhang et al., 2015). Pathogen responses helped deciphering the function of general transcription complexes in plants but whether *B. cinerea* and *S. sclerotiorum* are able to manipulate these complexes or associated epigenetic processes is not known.

PLANT SMALL BIOACTIVE PEPTIDES

Small bioactive peptides are defined as proteins of about 100 amino acids (aa) with roles in plant development, reproduction or interaction with the environment (Tavormina et al., 2015). They can exhibit a direct antifungal activity in the extracellular space, such as the *Brassicaceae*-specific ARACIN1 and 2. These ~80 aa peptides display antifungal activity *in vitro* (Neukermans et al., 2015). Ectopic expression of ARACIN1 in *Arabidopsis* reduced infection by *B. cinerea*. Antifungal activity against *S. sclerotiorum* and *B. cinerea* has also been shown *in vitro* for the *Brassica napus* 35 aa proline-rich peptide BnPRP1. The *BnPRP1* gene is induced upon *S. sclerotiorum* inoculation in susceptible but not in resistant plants (Cao et al., 2015). Small bioactive peptides can also serve as intra- or intercellular signals modulating plant defense signaling pathways. In tomato, systemin-mediated activation of the JA signaling pathways is required for resistance against *B. cinerea* (El Oirdi et al., 2011). Systemin is a *Solanaceae*-specific 18 aa peptide derived from the prosystemin precursor released into the vascular system at sites of cell damage (Pearce et al., 2001). Overexpression of prosystemin increased resistance to *B. cinerea* suggesting that systemin may serve as damage signal during fungal infection (Coppola et al., 2015). Dobon et al. (2015) identified four transcription factors that confer enhanced resistance to *B. cinerea* and found 77 genes up-regulated in the four corresponding mutants. Among these were several small signaling peptides (such as devil/rotundifolia peptide DVL3) and 13 small predicted secreted proteins of

unknown function of 34–123 aa, named PROVIR1 to 13. Most of these small peptide genes were induced upon fungal infection and their over-expression caused enhanced susceptibility to necrotrophic fungi (Dobon et al., 2015).

CONCLUDING STATEMENT

Due their phylogenetic proximity and similarities in lifestyle, *B. cinerea* and *S. sclerotiorum* are often used interchangeably as models of broad host range necrotrophic fungi. Recent progress has revealed commonalities in their virulence strategies and in the corresponding plant responses, but also differences providing valuable insights into the diversity of the molecular bases of broad of host range pathogenicity and plant QDR.

AUTHOR CONTRIBUTIONS

All authors listed, have made substantial, direct and intellectual contribution to the work, and approved it for publication.

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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