CURRENT REVIEW

Getting the Most from the Host: How Pathogens Force Plants to Cooperate in Disease

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Plant diseases caused by pathogenic microorganisms remain a major limitation in many crop production systems. Nonetheless, constitutive and inducible defense mechanisms render most plants inaccessible to pathogens, making disease an exception rather than a common outcome of plant-microbe interactions. Defense mechanisms and associated pathogen resistance were thus of key interest to many plant pathologists, and many of the molecular mechanisms underlying resistance have been elucidated over the last few decades. In recent years, the analysis of physiological and molecular determinants accounting for successful infection and eventual disease has become a topic of prime scientific interest. The hunt is now on for pathogen effectors subverting the host cell and for the plant compatibility functions manipulated by these effectors. An understanding of the molecular mechanisms underlying successful infection should make it possible to develop new crop protection strategies based on interference with compatibility to prevent disease. This review is addressing plant susceptibility and highlights a number of host processes that have been shown to be induced or subverted to facilitate infection. In particular, we focus on those processes that appear to be manipulated by filamentous fungal and oomycete pathogens.

Plants are continually exposed to pathogenic microorganisms but, in most cases, their constitutive defenses are sufficient to prevent disease. The presence of physical barriers, such as the cuticle and cell walls, and the generation of toxic secondary metabolites are generally sufficient to stall the attempts of a potential aggressor to establish infection. Any pathogen able to breach these constitutive barriers is then confronted with non-self recognition mechanisms, which initiate several layers of the plant immune system. A first layer of plant defense is activated by the perception of conserved microbe-associated molecular patterns (MAMPs) by membrane-bound pattern recognition receptors (Zipfel 2009). MAMP-triggered defense responses are generally sufficient to prevent infection. However, some pathogens produce and secrete virulence factors that either repress or reprogram host cell defense signaling, enabling them to overcome this first layer of the immune response. Most plants have thus evolved a second layer of immune responses, in which resistance (*R*) gene products recognize the presence or activities of microbial effector molecules. *R* gene product-mediated recognition triggers extremely strong

defense responses, frequently resulting in a localized form of programmed host cell death known as the hypersensitive response (HR) (Chisholm et al. 2006). This ‘arms race’ between plants and pathogens, in which the evolution of new defenses is matched by the evolution of new ways to overcome them, is now commonly represented as the zig-zag-zig model (Jones and Dangl 2006; Nishimura and Dangl 2010).

If a pathogen is to undergo a successful compatible interaction, it must overcome the constitutive barriers, avoid or suppress the immune responses, and reprogram the host cell for the establishment of infectious structures during penetration, invasion, and reproduction. Several recent reviews have dealt with the suppression of host defenses by bacterial, oomycete, and fungal pathogens (Birch et al. 2009; Ellis et al. 2009; Hein et al. 2009; Mansfield 2009; Schornack et al. 2009). We focus here on plant susceptibility and on those processes that are subverted by filamentous fungal and oomycete pathogens to promote disease. When appropriate, we provide examples of the manipulation of the host by bacterial pathogens.

The boundary between out and in.

Topography and chemical composition of the plant surface as well as the function and distribution of pores used by plants for gas exchange frequently determine infection processes. Several filamentous fungal and oomycete pathogens enter plant tissues by means of specialized penetration structures called appressoria. These structures enable the pathogen to breach the outer barrier (Judelson et al. 2008; Wilson and Talbot 2009). Appressorium formation by *Colletotrichum trifolii*, the causal agent of alfalfa anthracnose, requires the activity of a fungal protein kinase. Components of the plant cuticle activate the transcription of the gene encoding this kinase, thereby stimulating appressorium formation and penetration (Dickmann et al. 2003). The plant cuticle also determines infection by rust fungi. Germinating spores require a highly hydrophobic surface for adhesion, and the composition and amount of waxy compounds in the cuticle determines rust infection (Staples et al. 1985). Directed growth of germ tubes is then determined by the topography of the cuticle. Rust fungi detect ridges on the surface that match the dimensions of the stomatal lips of the host (Allen et al. 1991). Rusts are specific to this topography and are unable to locate stomata on nonhost plants (Kolmer et al. 2009). When a stoma is localized, the germ tube forms an appressorium over it and pushes through the stoma with a penetration peg.

The possibility that rust fungi interfere with stomatal functions to promote opening has not been explored, but some specialized fungi and bacteria secrete toxins or effectors that inter-

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act with host compounds, thus stimulating opening of stomata and favoring infection. Some of the toxins involved in such interactions had already been identified in the 1960s and 1970s. Several phytopathogenic fungi, such as *Sclerotinia sclerotiorum*, produce oxalic acid during the infection process (Bateman and Beer 1965), and mutants unable to synthesize this compound are less pathogenic than the wild-type fungus (Godoy et al. 1990). On the host plant of *S. sclerotiorum*, bean, secreted oxalic acid induces K⁺ accumulation and the breakdown of starch in guard cells, effects that trigger stomatal opening. Additionally, oxalic acid inhibits abscissic acid (ABA)-induced stomatal closure. Stomata of *Sclerotinia* spp.-infected plants are blocked open and favor fungal development (Guimarães and Stotz 2004). Another well known toxin is the terpenoid fusicoccin A, which is produced by the fungus *Fusicoccum amygdali*, a parasite of peach and almond trees (Turner and Graniti 1969). This toxin binds to the C-terminal domains of H⁺-ATPase dimers and requires a plant 14-3-3 protein for full activity. It activates the H⁺-ATPase by stabilizing a transient complex that forms between the autoinhibitory C-terminal domain and the 14-3-3 dimer, thus locking the enzyme into its most active state, inducing hyperpolarization of the plasma membrane and the opening of stomata (Baunsgaard et al. 1998; MacRobbie and Smyth 2010; Oecking and Hagemann 1999; Oecking et al. 1997; Würtele et al. 2003).

More is known about the manipulation of stomatal opening by the bacterial pathogen, *Pseudomonas syringae*. The *Arabidopsis* protein RIN4 is guarded by *R* gene products that confer immunity to *P. syringae* pathovars (Grant et al. 2006). RIN4 interacts with autoinhibitory C-terminal H⁺-ATPase domains in guard cells (J. Liu et al. 2009). In contrast to the mode of action of fusicoccin, the binding of RIN4 to the H⁺-ATPase results in stomatal closure and is thought to be part of the array of MAMP-triggered immune responses for preventing pathogen entry. However, RIN4 is the target of *Pseudomonas syringae* effector proteins AvrRpm1, AvrB, AvrRpt2, and HopF2Pto (Wilton et al. 2010). The effector-mediated inactivation or degradation of RIN4 may enable the bacterium to prevent stomatal closure, thereby facilitating its entry into plant tissues (J. Liu et al. 2009). Furthermore, *P. syringae* produces the toxin coronatine, a structural analog of the jasmonate (JA) conjugate JA-isoleucine (Melotto et al. 2008). Coronatine, by mimicking JA, is able to interact with the JA sensor CORONATINE INSENSITIVE1 (COI1). This interaction blocks guard cell-specific OST1 kinase and ABA signaling and prevents stomatal closure (Melotto et al. 2006). Thus, both fungal and bacterial pathogens manipulate stomata during the infection process.

Cozy biotrophy.

Filamentous biotrophs develop and multiply within living tissues, maintaining a delicate balance between extracting sufficient resources from host cells to complete their life cycle and leaving the plant with sufficient resources to ensure its survival. Haustoria are responsible for ensuring the maintenance of this balance and for the uptake of nutrients during growth and reproduction and are supposed to be the central structure for host manipulation during biotrophic growth (Fig. 1A). Pathogens must degrade the host cell wall locally to establish intracellular haustoria and to gain access to the nutrients present in the host. However, plants closely monitor the integrity of their cell walls and any damage leads to the activation of complex signaling networks, which are frequently interconnected with defense responses (Hématy et al. 2009). Filamentous biotrophs must thus minimize cell-wall degradation when forming their haustoria, to prevent the host from sensing the damage. Pathogens may achieve this by making use of the plant cell's own enzymatic machinery, thus loosening the cell wall

to facilitate penetration without giving the impression of damage. Although clear experimental proofs for this hypothesis are yet lacking, several findings support it. A genetic screen aiming at identifying powdery mildew resistant (*pmr*) mutants of *Arabidopsis* led to the identification of genes encoding a pectate lyase (PMR6), a callose synthase (PMR4), and a protein of unknown function (PMR5) (Vogel and Somerville 2000; Vogel et al. 2002, 2004). The corresponding mutants are more resistant than the wild type to infection with the fungus *Golovomyces cichoracearum*. *pmr6* and *pmr5* mutants have cell walls with an altered pectin composition, which might interfere with the establishment of biotrophy. In addition, the pectate lyase PMR6 may be recruited by the fungus to prevent the accumulation of pectin in the extrahaustorial matrix (Fig. 1B) (Vogel et al. 2004). According to this hypothesis, the loss of PMR6 in the mutant decreases the availability of nutrients to the pathogen, resulting in the observed enhanced resistance phenotype (Vogel et al. 2002). The third mutant *pmr4* shows enhanced resistance to both the fungal and oomycete biotrophs *G. cichoracearum* and *Hyaloperonospora arabidopsidis*, respectively (Vogel and Somerville 2000). PMR4 is a callose synthase that probably has multiple functions in susceptibility to the diseases caused by these pathogens. First, it has been shown that this enzyme or its product decreases salicylic acid (SA) signaling in *Arabidopsis*, thus amplifying SA-dependent defense responses in the *pmr4* mutant upon infection (Jacobs et al. 2003; Nimishura et al. 2003). Second, callose deposition at the haustorial neck may be a structural requirement for the stability and function of this feeding structure (Jacobs et al. 2003) (Fig. 1C). The synthesis of callose and its deposition at wounds and infection sites are generally considered to be a characteristic plant defense response (Chen and Kim 2009). Filamentous biotrophs thus seem to manipulate host enzymes to promote haustorium formation and function and to subvert resistance mechanisms to favor infection.

Whole-genome transcript profiling is a powerful approach to the characterization of metabolic host reprogramming by filamentous biotrophs (Chandran et al. 2009; Doehlemann et al. 2008; Fabro et al. 2008; Skibbe et al. 2010; Van Esse et al. 2008). A microarray analysis of the *Arabidopsis* transcriptome during the invasive growth of *H. arabidopsidis* within leaf tissues led to the identification of 17 genes specifically induced in these conditions (Huibers et al. 2009). Most of these genes had previously been described as involved in stress responses. Their role in downy mildew susceptibility is not clear, but they may serve as targets for fungal effector proteins (Huibers et al. 2009). A more precise analysis of the *Arabidopsis* susceptibility transcriptome was recently performed on laser-dissected powdery mildew infection sites (Chandran et al. 2010). This analysis revealed that the fungus *G. orontii* induces complex genetic reprogramming of infected host cells, transforming them into metabolic sinks. Furthermore, data from the susceptibility transcriptome suggest that infection modulates the host cell cycle. *MYB3R4* expression was found to be upregulated at sites of infection. This gene encodes a transcription factor that positively regulates G₂/M progression and cytokinesis in *Arabidopsis* (Haga et al. 2007). Examination of the epidermal cells harboring haustoria and of the underlying mesophyll cells revealed increases in the size of the nuclei of these cells and in the quantity of 4',6-diamidino-2-phenylindole-stainable DNA. These changes were not observed in cells at some distance from the site of infection (Chandran et al. 2010). However, these cells do not divide, so cells at sites of infection probably undergo pathogen-induced endoreduplication. The loss-of-function *myb3r4* mutation abolished endoreduplication at infection sites and increased resistance to *G. orontii*. It seems likely that the pathogen induces endoreduplication in host cells to increase

gene copy number and metabolic capacity. As a result, the host can supply the fungus with more nutrients, facilitating its sustained growth and reproduction (Chandran et al. 2010).

Clean-up and damage limitation.

Pathogens frequently encounter toxic compounds that are produced by the host. Oat roots produce the antimicrobial triterpene glycoside avenacin. The infection of oat by the fungus *Gaeumannomyces graminis* var. *avenae* is dependent on the detoxification of this compound by a fungal avenacin hydrolase (Morrissey and Osbourn 1999). Similarly, the infection of tomato by the leaf spot fungus *Septoria lycopersici* depends on the production of tomatinase, an extracellular enzyme that hydrolyzes the steroidal glycoalkaloid α -tomatine to the less toxic β_2 -tomatine (Pareja-Jaime et al. 2008). However, β_2 -

tomatine also suppresses plant defense responses, and fungal tomatinase clearly plays a dual role in saponin detoxification and counteracting disease-resistance processes (Bouarab et al. 2002). The fungal pathogen thus subverts the preformed antimicrobial compounds of its host, making use of these compounds to facilitate the establishment of infection.

A genetic screen led to the identification of *Arabidopsis* mutants, which show enhanced resistance to disease caused by the oomycete *H. arabidopsidis* (Van Damme et al. 2005, 2008). One of these downy mildew resistant mutants, *dmr1*, is affected in a gene encoding a homoserine kinase, suggesting that this enzyme is required for full oomycete susceptibility. The precise role of this homoserine kinase in the promotion of disease remains unclear, but its substrate, homoserine, is a potent stimulator of plant resistance (Van Damme et al. 2009). *H. arabi-*

Fig. 1. Suggested functions for plant 'susceptibility gene' products. **A**, The haustorium delivers effectors into the plant cell. Their interaction with host targets reprograms the plant cell into a metabolic sink, thus favoring nutrient uptake (Chandran et al. 2010) and probably promoting the creation of a beneficial environment for the haustorium. **B**, The pectate lyase PMR6 is probably required to prevent pectin accumulation in the extrahaustorial matrix (EHM), thus facilitating the unrestricted translocation of effectors and nutrients (Vogel et al. 2002). **C**, The callose synthase PMR4 is required to repress the salicylic acid (SA)-mediated resistance responses of the host. Callose deposition around the neck of the haustorium may stabilize this structure (Jacobs et al. 2003; Nishimura et al. 2003). **D**, The homoserine kinase DMR1 is thought to phosphorylate homoserine, decreasing its availability, thereby preventing the induction of resistance (Van Damme et al. 2009). **E**, The caffeate *O*-methyltransferase COMT1 transforms 5-hydroxy coniferaldehyde (OH-CA) into sinapaldehyde (SiA), a precursor of S lignin, which is incorporated into cell walls. Excess S lignin precursors are stored as inoffensive sinapoylmalate (SM). COMT1 activity may prevent the accumulation of 5-hydroxy feruloylmalate (OH-FM), which affects oomycete development (Quentin et al. 2009). **F**, To prevent the accumulation of harmful compounds, the bacterial pathogen *P. syringae* targets the phospholipase A₂ (PL-A₂) SOBER1, which hydrolyzes phosphatidylcholine (PC) into arachidonic acid (AA) and lysophosphatidylcholine (LPC). PC is also a substrate for phospholipase D (PL-D), hydrolyzing it into choline and phosphatidic acid (PA). High PA concentrations induce cell death, and depleting the pool of available PC by SOBER1 probably prevents PA generation (Kirik and Mudgett, 2009). **G**, MLO interacts with the syntaxin ROR2/PEN1. Adapted fungal biotrophs may use the MLO-mediated vesicle transport machinery to deliver plant proteins for the establishment of a compatible interaction. The vesicles probably also contribute to the extension of the plasma membrane during haustorium formation (Panstruga 2005). Additionally, MLO represses the tryptophan (Trp)-derived synthesis of harmful indolic compounds such as the phytoalexin camalexin (Cam) (Consonni et al. 2010). **H**, Fungal necrotrophs stimulate cell death. The toxin victorin targets the resistance gene product-like nucleotide binding site-leucine rich repeat protein, LOV1 (Sweat et al. 2008). LOV1 signals through the thioredoxin ATTRX5, which upregulates cell death, thereby probably promoting necrotrophic growth (Sweat and Wolpert, 2007).

dopsis may thus activate *DMR1* expression and make use of this enzyme to prevent the accumulation of homoserine, which would counteract the infection process (Fig. 1D). Similarly, *H. arabidopsidis* stimulates the transcription of a gene encoding caffeic acid *O*-methyltransferase 1 (*COMT1*). *Arabidopsis comt1* mutants cannot synthesize precursors for the generation of S lignin to be incorporated into the cell wall. Instead, they accumulate soluble hydroxyferuloyl malate, a metabolite that interferes with normal oomycete development. The stimulation of *COMT1* transcription may thus enable the oomycete to prevent the accumulation of a substance that would be harmful to it within plant tissues (Quentin et al. 2009) (Fig. 1E).

Again, more detailed information concerning the way in which host enzymes are manipulated to prevent the accumulation of substances counteracting infection is available for bacterial pathosystems. Pi-0 is the only ecotype of *A. thaliana* that is resistant to *P. syringae* pv. tomato, which expresses the AvrBsT effector. This ecotype presents typical symptoms of the HR on infection with this bacterium. Crosses of Pi-0 with susceptible ecotypes revealed that resistance is associated with the loss-of-function of a gene encoding an α/β hydrolase called SUPPRESSOR OF AVRBST-ELICITED RESISTANCE 1 (*SOBER1*) (Cunnac et al. 2007). *SOBER1* has phospholipase A₂ activity and hydrolyzes phosphatidylcholine. In response to infection with *P. syringae* AvrBsT, only resistant Pi-0 plants accumulate high levels of phosphatidic acid (PA), suggesting that the HR of Pi-0 involves PA (Kirik and Mudgett 2009). Expression of the gene encoding *SOBER1* in the Pi-0 background results in lower levels of PA production upon infection with *P. syringae* AvrBsT, preventing the HR and restoring susceptibility. In plant cells, PA is generated by the hydrolysis of phosphatidylcholine by phospholipase D. The role of *SOBER1* may be to repress PA accumulation in infected plant cells by hydrolyzing phosphatidylcholine to generate inoffensive degradation products, thus decreasing its availability to phospholipase D. The *SOBER1* lipase is, therefore, a negative regulator of AvrBsT-triggered PA signaling in *Arabidopsis* (Fig. 1F). These examples illustrate how oomycete and bacterial pathogens manipulate host enzymes to impede the production of substances that might interfere with the infection cycle.

Resist or accommodate.

If a host protein is required for the growth, multiplication, and reproduction of a pathogen within plant tissues, its absence or inaccessibility leads to recessively inherited resistance. Barley (*Hordeum vulgare*) harbors the mildew resistance locus *Mlo*. Wild-type barley carrying the *Mlo* gene is susceptible to the barley powdery mildew pathogen, *Blumeria graminis* f. sp. *hordei*. By contrast, recessive mutations in the gene confer durable resistance to all races of the fungus (Büschges et al. 1997; Piffanelli et al. 2004). Mutated *mlo* alleles were incorporated into barley during the early stages of domestication and have since been widely adopted in breeding programs (Jørgensen 1992). *Mlo* encodes an integral membrane protein with seven membrane-spanning helices, harboring a C-terminal calmodulin-binding domain. It interacts with ROR2, a SNARE domain-containing syntaxin (Devoto et al. 1999, 2003; Kim et al. 2002). MLO and ROR2 accumulate in the plasma membrane at the sites of fungal penetration (Collins et al. 2003). The precise role of MLO in powdery mildew susceptibility remains unclear. However, SNARE proteins participate in the fusion of intracellular vesicles with the plasma membrane. MLO is, therefore, probably involved in the exocytosis of plant proteins and of compounds required for the formation of the haustorium (Fig. 1G). In *Arabidopsis*, three *Mlo* orthologs contribute to susceptibility to *G. orontii*. The mutation of one of these genes greatly decreases infection levels, and triple mutants are fully resistant

(Consonni et al. 2006). The spontaneous deposition of callose in these mutants in the absence of the pathogen suggests that MLO may downregulate basal defense mechanisms. Recently, it was shown that *mlo2* and the *mlo* triple mutants are altered in metabolic steady-state levels of indolic compounds such as the phytoalexin camalexin and indolic glucosinolates (Consonni et al. 2010). These findings suggest that MLO negatively regulates the synthesis of tryptophan-derived indolic compounds that interfere with the infection process (Fig. 1G). MLO thus seem to orchestrate a complex susceptibility mechanism that has been conserved since the separation of the monocot and dicot lineages 200 million years ago (Consonni et al. 2006).

Assisted host suicide.

Necrotrophic ascomycetes from the order Pleosporales produce host-selective toxins (HST) that interact with specific targets within hosts plants to promote disease. Absence of HST production by the pathogen or absence (or inaccessibility) of the target in the host results in resistance (Friesen et al. 2008; Turgeon and Baker 2007; Wolpert et al. 2002). Genes encoding HST targets may therefore be considered recessive *R* genes. For example, *Stagonospora nodorum* produces the HST protein SnTox3, which interacts directly or indirectly with the product of the corresponding wheat susceptibility gene *Snn3* (Z. Liu et al. 2009). The helminthosporiosis pathogen of wheat, *Pyrenophora tritici-repentis*, produces the HST PtrToxA, which interacts with the host chloroplast protein ToxABP1 (Manning et al. 2007). In the chloroplast, this interaction interferes with the functioning of photosystems I and II, leading to a light-dependent accumulation of reactive oxygen species and necrosis, thus favoring the necrotrophic invasive growth of the fungus (Manning et al. 2009). *Cochliobolus victoriae* makes use of a subtle necrotrophic infection strategy based on the subversion of resistance responses. In its host, oat, only genotypes carrying the dominant Vb allele are susceptible to both the Victoria blight pathogen and the HST victorin (Wolpert and Macko 1989). Victorin secretion is essential for pathogenicity, indicating that this toxin promotes disease by targeting Vb. In *Arabidopsis*, sensitivity to victorin is conferred by *LOCUS ORCHESTRATING VICTORIN EFFECTS 1* (*LOV1*). This gene encodes a nucleotide binding site-leucine rich repeat protein resembling typical *R* gene products (Lorang et al. 2004, 2007; Sweat et al. 2008). In *Arabidopsis*, LOV1 signals through the thioredoxin ATTRX5, which upregulates victorin-induced cell death (Sweat and Wolpert 2007) (Fig. 1H). It thus seems likely that victorin subverts an ancient *R* gene-mediated immune response to provoke HR-like cell death and to promote necrotrophic infection.

Molecular manipulators.

Most of our recently acquired knowledge on the manipulation of the host by plant pathogens concerns the suppression of plant defenses and the counteracting of plant immune responses. However, the finding that filamentous necrotrophs target proteins resembling *R* gene products suggests that pathogens with this lifestyle may turn immune responses to their own advantage rather than suppressing them. The borders between resistance and susceptibility are often blurred, and the examples presented above illustrate the ways in which pathogens may hijack certain elements of defense responses to favor disease. For filamentous hemibiotrophs such as *Phytophthora infestans*, the oomycete responsible for the Irish famine, a sequential infection strategy has been proposed, in which biotrophy is conditioned by the initial suppression of defenses, with the activation of HR-like responses coordinating the switch to necrotrophy (Kelley et al. 2010). The fine-tuning of host cell responses during *Phytophthora* infection is thought to be coordinated by

effector proteins, which have an N-terminal RXLR-dEER motif directing their translocation into the host cell (Jiang et al. 2008; Rehmany et al. 2005; Whisson et al. 2007). The mechanisms of RXLR-effector translocation remain unclear, but this motif resembles the RXLXE/Q/D core motif used by the malaria pathogen *Plasmodium falciparum* to translocate effector proteins into the cytoplasm of human erythrocytes (Hiller et al. 2004; Marti et al. 2004). The recent *P. infestans* genome sequencing project showed this microbe to contain 563 genes encoding putative RXLR effectors (Haas et al. 2009), more than 300 of which were also found in other sequenced *Phytophthora* species (Tyler et al. 2006). In contrast to the situation for oomycete effectors, no clear consensus signature has yet been identified in fungal effectors. However, fungal biotrophs and hemibiotrophs such as oomycetes are thought to produce hundreds of effectors (De Wit et al. 2009). Haustoria, which are formed during biotrophic growth, may act as a reservoir for effector delivery directly into the host cell (Birch et al. 2009; Catanzariti et al. 2006) (Fig. 1A). One of the challenges facing us in the future is increasing our knowledge of i) the mechanisms and the timing of effector delivery into the host cell, ii) the identity of effector targets within the plant cell, iii) the physiological role of these targets in the absence of infection, and iv) the effector-mediated subversion of this role to favor disease.

Concluding remarks.

The analysis of host-cell genetic reprogramming by pathogens has excited considerable interest in recent years. However, detailed descriptions of the mechanisms underlying pathogen accommodation have essentially been provided by studies of *Arabidopsis* and bacterial pathogens. In contrast, we still know little about the manipulation of the host plant by filamentous biotrophs or hemibiotrophs, and most suggestions remain hypothetical. This is principally because the genetic analysis of biotrophy and hemibiotrophy is feasible for only a few pathosystems involving *Arabidopsis*, and filamentous biotrophs are not very amenable to functional genomics approaches. The establishment of models of compatible interactions between *A. thaliana* and fungal (O'Connell et al. 2004; Park et al. 2009) or oomycete pathogens (Attard et al. 2010) should help to fill the gaps that currently exist. Furthermore, genomic data for most of the pathogens involved have begun to accumulate in recent years. Tools and methods for functional genomics studies of fungal and oomycete pathogens are now well established and should increase our understanding of compatibility mechanisms in the future. Improvements in our knowledge of compatibility will, in turn, probably deepen our insight into both molecular phytopathology and plant physiology.

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