The Plant Cell, Vol. 20: 2009–2017, August 2008, www.plantcell.org

PERSPECTIVE

From Guard to Decoy: A New Model for Perception of Plant Pathogen Effectors

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The Guard Model for disease resistance postulates that plant resistance proteins act by monitoring (guarding) the target of their corresponding pathogen effector. We posit, however, that guarded effector targets are evolutionarily unstable in plant populations polymorphic for resistance (*R*) genes. Depending on the absence or presence of the *R* gene, guarded effector targets are subject to opposing selection forces (1) to evade manipulation by effectors (weaker interaction) and (2) to improve perception of effectors (stronger interaction). Duplication of the effector target gene or independent evolution of a target mimic could relax evolutionary constraints and result in a decoy that would be solely involved in effector perception. There is growing support for this Decoy Model from four diverse cases of effector perception involving Pto, Bs3, RCR3, and RIN4. We discuss the differences between the Guard and Decoy Models and their variants, hypothesize how decoys might have evolved, and suggest ways to challenge the Decoy Model.

Plants have evolved sophisticated mechanisms to perceive pathogen attack and trigger an effective innate immune response. An important and well-characterized perception mechanism is based on resistance (*R*) genes in plants whose products confer recognition of cognate avirulence (Avr) proteins in the pathogen. This gene-for-gene hypothesis was introduced by Flor in the 1940s, and dozens of *R-Avr* gene combinations have since been characterized (Dangl and Jones, 2001).

Although the gene-for-gene hypothesis is now firmly supported by the characterization of many *R-Avr* gene pairs, the underlying perception mechanism has been subject to debate for more than a decade. Initially it was widely thought that products of *R* genes act as receptors that directly interact with the products of *Avr* genes (Keen, 1990). This ligand-receptor model was supported by the fact that some *Avr* gene products are small and colocalize with *R* gene products, most of which encode receptor-like proteins carrying Leu-rich repeats (LRRs). Indeed, direct binding of a few R-Avr combinations was found, consistent with a receptor-ligand mode of action (e.g., Jia et al., 2000; Deslandes et al., 2003; Dodds et al., 2006; Ueda et al., 2006). However, for a number of R-Avr combinations, physical interactions have not been observed, and perception is thought to be indirect.

Meanwhile, it has become evident that many Avr proteins contribute to pathogen virulence on plants lacking the cognate *R* gene. Avr proteins are now considered to be part of a larger repertoire of pathogen-secreted proteins that are called effectors to stress their presumed intrinsic virulence function. Avr recognition by plants has been coined effector-triggered immunity to contrast it with pathogen-associated molecular pattern (PAMP)–triggered immunity (Chisholm et al., 2006; Jones and Dangl, 2006). Effectors are pathogen-secreted proteins that manipulate host cell functions, whereas PAMPs define molecular motifs common to many pathogens. The concept that effectors have specific targets in the host is an essential component of a popular model that explains indirect perception mechanism of effectors by R proteins. This Guard Model predicts that R proteins act by monitoring (guarding) the effector target and that modification of this target by the effector results in the activation of the R protein, which triggers disease resistance in the host (Van der Biezen and Jones, 1998; Dangl and Jones, 2001).

The Guard Model was originally proposed to explain the mechanism of *Pseudomonas syringae* AvrPto perception by the tomato proteins Pto and Prf (Van der Biezen and Jones, 1998) and was later generalized to perception of other effector proteins (Dangl and Jones, 2001). The indirect effector perception mechanism postulated by the Guard Model explains how multiple effectors could be perceived by a single R protein, thus enabling a relatively small *R* gene repertoire to target the broad diversity of pathogens that attack plants (Dangl and Jones, 2001). Implicit in the Guard Model is the notion that the guarded effector target (also called the guardee) is indispensible for the virulence function of the effector protein in the absence of the cognate R protein. Support for the Guard Model has accumulated over the past decade with the description of guarded effector targets. Classical examples of these presumed guardees are *Arabidopsis* RIN4 and PBS1 and tomato RCR3 and Pto (Jones and Dangl, 2006).

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www.plantcell.org/cgi/doi/10.1105/tpc.108.060194

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Over the past few years, new data on indirectly recognized effectors have emerged that are inconsistent with the original description of the Guard Model. It is now well documented that many pathogen effectors have multiple targets in the host and that classical guardee proteins are often dispensable for the virulence activities of effectors in plants lacking the R protein. New data on additional targets of AvrPto and AvrBs3 prompted proposals of the concept that some host targets of effectors act as decoys to detect pathogen effectors via R proteins (Zhou and Chai, 2008; Zipfel and Rathjen, 2008). Here, we further develop these ideas into a Decoy Model that is consistent with most of the data described so far and is coherent with the current knowledge of evolution in plant–pathogen interactions. Also, we discuss the experiments required to discriminate between the Guard and Decoy Models as well as the possible implications of the Decoy Model. For clarity, Table 1 lists the terms used throughout this essay.

THE DECOY MODEL

From an evolutionary point of view, the guarded effector target is in an unstable situation since it is subject to two opposing natural selection forces in plant populations where *R* genes are polymorphic. In this case, *R* gene polymorphism means the presence/absence of functional *R* genes in different individuals in a plant population (Figure 1). In the absence of a functional *R* gene, natural selection is expected to drive the guardee to decrease its binding affinity with the effector and thereby evade detection and modification by the effector. However, in the presence of a functional *R* gene, natural selection is expected to favor guardees with improved interaction with an effector to enhance pathogen perception. These two conflicting selection pressures on the same effector interaction surface of the guardee results in an evolutionarily unstable situation that could be relaxed upon the evolution of a host protein, termed here "decoy," that specializes in perception of the effector by the R protein but itself has no function either in the development of disease or resistance. Thus, the decoy mimics effector targets to trap the pathogen into a recognition event. Decoys might evolve from effector targets by gene duplication followed by subsequent evolution or evolve independently by mimicking effector targets (target mimicry). In any case, the Decoy Model implies that the effector target monitored by the R protein is a decoy that mimics the operative effector target but only functions in perception of pathogen effectors without contributing pathogen fitness in the absence of its cognate R protein. This Decoy Model is distinct from the classical and refined Guard Models that imply that the manipulation of the guarded effector target by the effector benefits pathogen fitness in the absence of the R protein (Figure 2).

This concept of a decoy is also distinct from animal decoy receptors that are defined as inactive receptors that act as sinks that deplete ligands, thereby preventing them from binding their operative receptor (Ashkenazi and Dixit, 1999; Montovani et al., 2001). These competing decoys, however, act in the absence of monitoring R proteins and are therefore different from the decoys that are enslaved in effector perception mechanisms. However, it remains possible that plant decoys that act in perception also compete with operative targets for effector target binding. In the absence of the R protein, these competing decoys would then limit rather than promote pathogen fitness.

The key assumptions behind the Decoy Model are inferred from our current understanding of plant–microbe interactions. First, *R* genes are typically polymorphic in natural plant populations. This has been observed repeatedly in both single gene studies (e.g., *Arabidopsis RPM1* [Stahl et al., 1999] and tomato *Cf-9* [Van der Hoorn et al., 2001]) and genome-wide analyses (Bakker et al., 2006; Clark et al., 2007). Second, effector targets are under selection for decreased binding affinity to effectors. Examples include the recessive resistance mutations in rice *xa13* that evolved to evade transcriptional activation by *Xanthomonas oryzae* pv *oryzae* effectors (Iyer-Pascuzzi and McCouch, 2007; Yang et al., 2007). Furthermore, recessive mutations in transcription factor IIA and elongation factor eIF4E were found to evade manipulation by bacterial blight and potyviral VPg, respectively (Iyer-Pascuzzi and McCouch, 2006; Charron et al., 2008). Also, enhanced patterns of diversifying selection have been described in inhibitor binding residues of several plant enzymes that operate at the plant pathogen interface (reviewed in Misas-Villamil and Van der Hoorn, 2008). These include soybean endo-β-1,3-glucanase that is targeted by the glucanase-inhibitor protein-1 from *Phytophthora*

Table 1. Definitions of Terms Used in This Manuscript

Term	Definition
Avr protein	Pathogen effector that triggers resistance via activation of specific cognate host R proteins.
Decoy	Effector target required for R protein function but with no function in host defense or susceptibility in the absence of its cognate R protein; effector alteration of the decoy does not result in enhanced pathogen fitness in plants that lack the R protein and triggers innate immunity in plants that carry the R protein.
Effector	Secreted pathogen protein that manipulates host cell functions.
Guardee	Effector target required for R protein function and with a function in host defense or susceptibility in the absence of its cognate R protein; effector alteration of the guardee results in enhanced pathogen fitness in plants that lack the R protein and triggers innate immunity in plants that carry the R protein.
Operative target	Host target that when manipulated by a pathogen effector results in enhanced pathogen fitness.
R protein	Protein that confers resistance by mediating direct or indirect recognition of a pathogen Avr protein. This is often but not always an NB-LRR protein.

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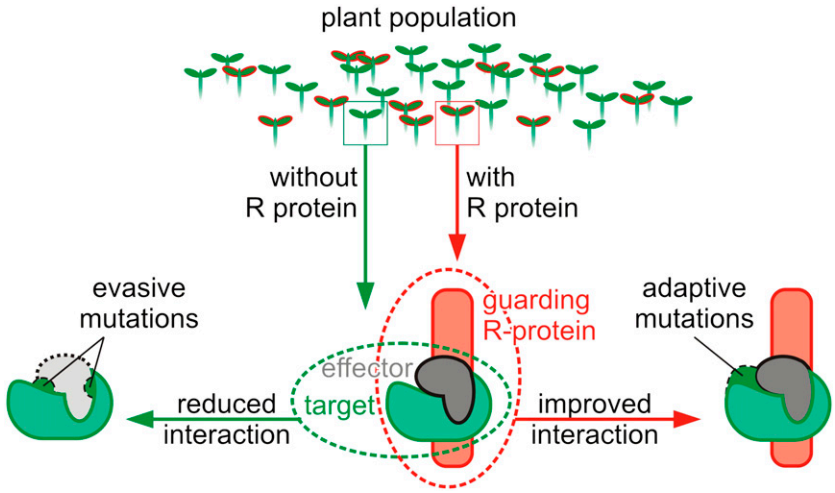

Figure 1. Opposing Selection Forces on Guarded Effector Targets in a Plant Population Polymorphic for *R* Genes.

Opposing selection forces are expected to operate on guarded effector targets in plants with or without the associated R protein. In the absence of the R protein (green arrows), targets will be under selective pressure to reduce the interaction and evade manipulation (left). In the presence of the R protein (red arrows), the guarded effector target will be under selective pressure to improve the interaction with the effector and enhance pathogen perception (right). The figure represents protein complexes, but similar models can be drawn for nonprotein effector targets. A gene duplication of the effector target or the independent evolution of a target mimic would reduce the evolutionary constraints imposed on the guarded effector target, allowing it to specialize as a coreceptor (decoy) that regulates the activation of the R protein.

sojae (Bishop et al., 2005; Damasceno et al., 2008) and the tomato proteases targeted by *Cladosporium fulvum* Avr2 (Shabab et al., 2008). Third, in the presence of the R protein, there is selection on the guarded effector target to maintain or improve its interaction with the effector. This has not been shown directly, but this process should not be different from the adaptation of R proteins that physically interact with effectors. Flax rust resistance alleles *L5*, *L6*, and *L7*, for example, have probably been selected for enhanced interactions with the different alleles of *AvrL567* (Ellis et al., 2007).

It is interesting to note that each of the four players in this antagonistic molecular interaction are under selection forces to adapt: (1) the operative target is under selection to evade manipulation by the effector; (2) the effector is under selection to target the adjusted operative targets while preventing interactions with the decoy, which would trigger defense responses in the presence of the R protein; (3) the decoy is under selection to adapt to adjusted effectors and is under additional selection to prevent autoimmune responses; and (4) the R protein is under selection to adapt to novel decoy-effector complexes while preventing autoimmune responses. As a result, each component is part of a molecular arms race in which each player is a target of the next.

SUPPORT FOR THE DECOY MODEL

The Decoy Model is consistent with recent findings on effector activities and perception by plants and is supported by four cases of well-studied effector perception mechanisms. These cases are discussed below and are summarized in Table 2. Overall, there is a striking diversity in the perception mechanisms, R protein structure, and pathosystems, indicating that decoys have evolved frequently and independently in antagonistic plant–pathogen interactions.

Case 1: Pto

P. syringae AvrPto is a kinase inhibitor that blocks the function of FLS2 and EFR, two receptor-like kinases involved in PAMP-triggered immunity (Xing et al., 2007; Xiang et al., 2008). Tomato *Pto* encodes a Ser/Thr kinase that confers resistance to *P. syringae* strains carrying *avrPto*, an interaction that also requires the nucleotide binding (NB)–LRR R protein Prf. AvrPto contributes to virulence on tomato and *Arabidopsis* but not on *Arabidopsis* lacking FLS2, indicating that FLS2 is an operative virulence target of AvrPto (Xiang et al., 2008). Considering that AvrPto contributes to virulence on tomato, even in the absence of Pto (Chang et al., 2000), it is possible that the tomato ortholog of FLS2 and other receptor-like kinases are the operative targets of AvrPto and that Pto itself is a decoy. That Pto is a decoy confined to the regulation of Prf is further supported by the observation that Pto associates with Prf in vivo and that Pto accumulation is dependent of Prf accumulation (Mucyn et al., 2006). As an interesting variation of the Decoy Model, it has been proposed that Pto competes with FLS2 for AvrPto binding (Zhou and Chai, 2008; Zipfel and Rathjen, 2008). This competing decoy model is similar to that of animal

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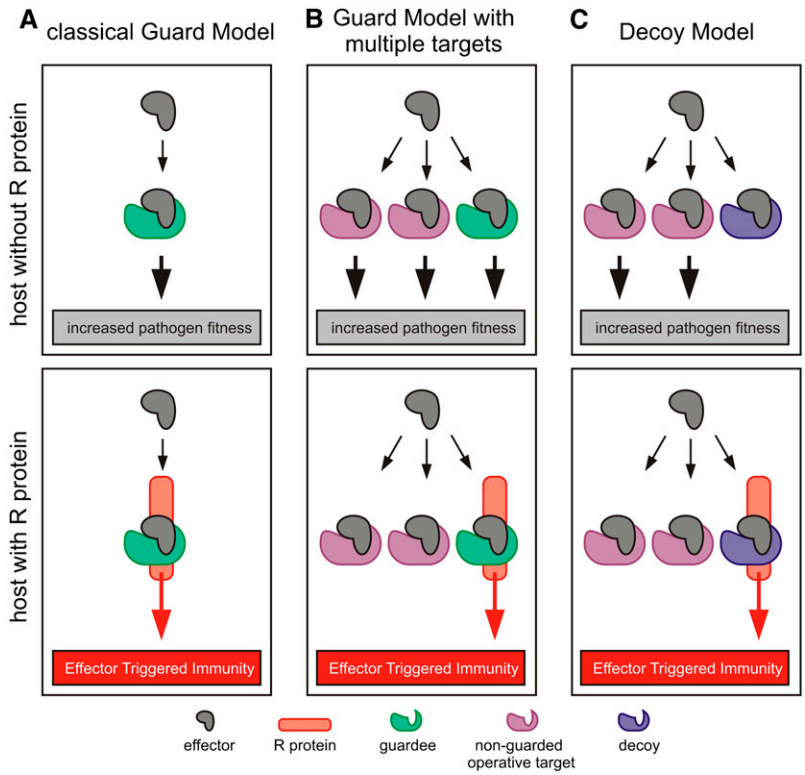

Figure 2. Comparisons of the Guard and Decoy Models.

The classical Guard Model **(A)** is contrasted with a modified Guard Model in which the effector targets multiple plant proteins **(B)** and the Decoy Model **(C)**. Effectors are depicted in gray, operative effector targets in purple, guardee in green, decoy in blue, and the R protein in orange.

decoy receptors and implies that Pto would restrict rather than promote pathogen fitness in the absence of Prf.

Case 2: *pBS3*

Xanthomonas campestris pv *vesicatoria* AvrBs3 is a type-III effector that functions as a transcription factor by binding and activating promoters in the nucleus of host pepper cells. An important role for AvrBs3 is to induce host cell size expansion (hypertrophy) by binding and activating the promoter of *Upa20* (*pUpa20*), a master regulator of cell size (Kay et al., 2007). In resistant plants, AvrBs3 also activates the promoter of the pepper *Bs3* gene (*pBs3*), an unusual *R* gene in that it encodes a flavin monooxygenase (Römer et al., 2007). Expression of *Bs3* has not been detected in the absence of AvrBs3, suggesting that this gene may not have any obvious function in the absence of avirulent bacteria and does not contribute to defense to bacteria that lack AvrBs3. These data are consistent with a model in which *pBs3* is a decoy and *pUpa20* is one of the operative targets of AvrBs3 (Zhou and Chai, 2008). In addition, AvrHah1, an AvrBs3-homologous effector of *Xanthomonas gardneri* that functions as a transcriptional activator and induces water-soaking in susceptible pepper plants, also activates the *Bs3* promoter resulting in hypersensitive cell death in *Bs3* pepper plants (Schornack et al., 2008). This indicates that *pBs3* is a decoy that traps at least two distinct effectors from different species of *Xanthomonas* (Schornack et al., 2008).

Case 3: RCR3

The effector protein Avr2 of the fungus *C. fulvum* is a secreted protein that inhibits RCR3 and PIP1, two secreted, defense-induced Cys proteases of tomato that are under diversifying selection (Rooney et al., 2005; Shabab et al., 2008). PIP1 is a pathogenesis-related protein that accumulates to high levels during infection by diverse pathogens and by salicylic acid (Tian et al., 2007; Shabab et al., 2008). RCR3, on the other hand, also accumulates as a pathogenesis-related protein but to much lower levels compared with PIP1 (Shabab et al., 2008). The role of PIP1 and RCR3 in the apoplastic defense response remains to be determined. However, *rcr3* mutants (MM-Cf2/rcr3 lines) are similarly susceptible to *C. fulvum* as tomato lacking the *Cf-2* gene cluster (MM-Cf0 line), indicating that RCR3 inhibition does not contribute to virulence (Dixon et al., 2000). Together, these data suggest that PIP1 is an operative target of Avr2 and that

Table 2. Four Cases Supporting the Decoy Model

Case	1	2	3	4
Plant species	Tomato	Pepper	Tomato	*Arabidopsis*
Pathogen	*P. syringae* pv *tomato* (bacterium)	*X. campestris* pv *vesicatoria* (bacterium)	*C. fulvum* (fungus)	*P. syringae* (bacterium)
Site of perception	Cytoplasm	Nucleus	Apoplast	Cytoplasm
R protein	Prf	Bs3	Cf-2	RPS2
Biochemical function of R protein	NB-LRR	Flavin monooxygenase	Receptor-like protein	NB-LRR
Decoy	Pto	*pBs3*	RCR3	RIN4
Biochemical function of decoy	Kinase	*upa* box in promoter of *Bs3* gene	Cys protease	Negative regulator of basal defense (Kim et al., 2005)
Operative target	Le FLS2?	*pUpa20*	PIP1	Not yet identified
Structure and function of operative target	Receptor-like kinase required for basal resistance	*upa* box in promoter of cell size regulator Upa20 and other genes	Cys protease secreted abundantly during defense	Unknown
Effector	AvrPto	AvrBs3	Avr2	AvrRpt2
Biochemical function of effector	Kinase inhibitor	Transcription factor	Protease inhibitor	Cys protease
Presumed perception mechanism	Pto inhibition by AvrPto activates Prf (Mucyn et al., 2006; Xing et al., 2007)	AvrBs3 binds and activates promoter of *Bs3* (Römer et al., 2007)	Avr2 inhibits Rcr3, and Avr2-Rcr3 complex probably activates Cf-2 (Rooney et al., 2005)	AvrRpt2 cleaves RIN4 from the RIN4-RPS2 complex, activating RPS2 (Axtell and Staskawicz, 2003; Mackey et al., 2003)
Virulence role of the effector?	Yes: AvrPto contributes to virulence on tomato (Chang et al., 2000) and *Arabidopsis* (Xiang et al., 2008)	Yes: AvrBs3 contributes to virulence on pepper under field conditions (Wichmann and Bergelson, 2004)	Yes: Avr2 contributes to virulence on tomato (van Esse et al., 2008)	Yes: AvrRpt2 contributes to virulence on *Arabidopsis* (Guttman and Greenberg, 2001)
Does pathogen benefit from manipulating decoy?	No?: no enhanced virulence on pto/Prf compared with Pto/prf tomato lines (Chang et al., 2000)	No: not anticipated	No?: No enhanced virulence on MM-Cf2/rcr3 compared with MM-Cf0 tomato lines (Dixon et al., 2000)	No?: No enhanced virulence on *rin4*/*rps2* compared with *RIN4*/*rps2 Arabidopsis* lines (Belkhadir et al., 2004; Lim and Kunkel, 2004)
Does pathogen benefit from manipulating operative target?	Yes: AvrPto inhibits FLS2 kinase domain and no longer contributes to virulence on *fls2* mutants (Xiang et al., 2008)	Yes: AvrBs3 activates the promoter of *Upa20*, resulting in enhanced cell size, a phenotype that is thought to be beneficial for the bacteria (Kay et al., 2007)	Yes?: Avr2 inhibits the abundant, defense-related protease PIP1 (Shabab et al., 2008). However, a role of PIP1 in defense has not yet been demonstrated.	Not investigated: operative targets are not yet known.

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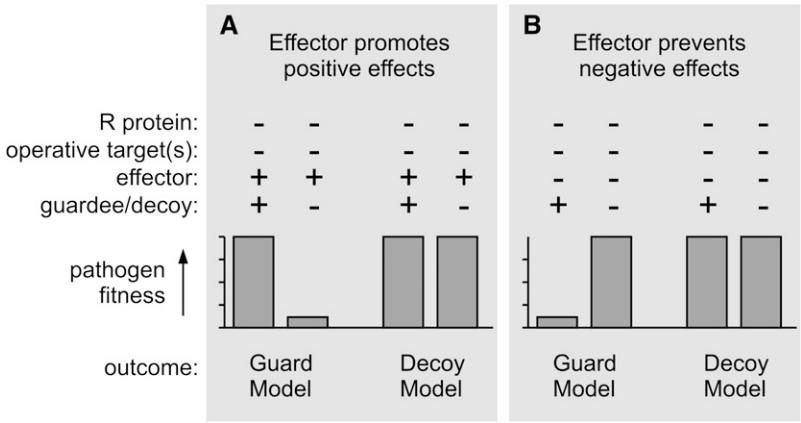

Figure 3. Genetic Tests to Discriminate between the Guard and Decoy Models.

Plants lacking both the R protein and the presumed operative target(s) should be challenged with pathogens in the absence or presence of the guardee/decoy. A differential pathogen growth supports the Guard Model, whereas an unaffected pathogen growth supports the Decoy Model. The test of choice depends on the nature of the effector.

(A) Effectors that promote positive effects on pathogen growth by manipulating their target should be present during the test to reveal target contributions.

(B) Effectors that prevent negative effects on pathogen growth should be omitted to avoid them from suppressing a possible phenotype.

RCR3 acts as a decoy to trap the fungus into a recognition event in plants carrying *Cf-2* (Shabab et al., 2008).

Case 4: RIN4

Arabidopsis RIN4 is a negative regulator of basal defense that is targeted by multiple *P. syringae* effectors (AvrRpm1, AvrRpt2, and AvrB) and monitored by at least two R proteins (RPM1 and RPS2) (Kim et al., 2005). Basal defense responses are suppressed in RIN4 overexpression lines and slightly enhanced in *rin4* mutant lines (Kim et al., 2005). RIN4 is targeted by AvrRpm1 and AvrRpt2 for phosphorylation and degradation, respectively (Mackey et al., 2002; Axtell and Staskawicz, 2003), but it is unknown if and how these modifications benefit pathogen virulence. One hypothesis is that RIN4 cleavage by AvrRpt2 releases RIN4 fragments that suppress basal defense responses. Although these data are consistent with the Guard Model, RIN4 could also be a decoy as long as the definite link that RIN4 manipulation promotes pathogen virulence has not been demonstrated. For example, AvrRpm1 and AvrRpt2 promote virulence to a similar extent in *rin4* knockout plants as in wild-type plants, and AvrRpt2 mutants that do not cleave RIN4 still contribute to virulence (Belkhadir et al., 2004; Lim and Kunkel, 2004). Thus, both AvrRpm1 and AvrRpt2 appear to have operative targets other than RIN4. AvrRpt2 is a protease that cleaves several *Arabidopsis* proteins besides RIN4, but the role of these AvrRpt2 targets in defense remains to be examined (Chisholm et al., 2005; Takemoto and Jones, 2005). Thus, the key information to define RIN4 as a guardee or a decoy is whether or not RIN4 modification by the effectors benefits the pathogen.

Other cases for which the Guard Model has been proposed also fit the Decoy Model, although there is no evidence to distinguish between the Guard and Decoy Models at this stage. *P. syringae* AvrPphB, for example, cleaves the PBS1 kinase in the host *Arabidopsis*, resulting in recognition by the RPS5 R protein (Shao et al., 2003; Ade et al., 2007). However, the virulence effect of AvrPphB in the presence and absence of PBS1 remains to be investigated. Also, perception of *X. oryzae* pv *oryzae* AvrXa27, *C. fulvum* Avr9, *Tobacco mosaic virus* p50, *Turnip crinkle virus* CP, and *Potato virus X* CP by their cognate R proteins appears to be indirect, but the effector targets and/or perception mechanisms remain to be elucidated (Kooman-Gersmann et al., 1998; Ren et al., 2000, 2005; Gu et al., 2005; Sacco et al., 2007; Tameling and Baulcombe, 2007; Caplan et al., 2008).

EVOLUTION OF DECOYS

How do decoys evolve? Conceptually, decoys can be evolutionarily related to operative targets or may evolve independently by target mimicry. Either one of these scenarios might apply for Pto. AvrPto inhibits multiple defense-related kinases, indicating that Pto could have directly evolved from one of these targets. For instance, Pto may have evolved from a receptor-like kinase that lost the extracellular domains that are not required for AvrPto perception. This is consistent with the observation that Pto is most closely related to the kinase domains of receptor-like kinases (Hardie, 1999). Alternatively, Pto may have functioned in a kinase pathway that was not originally targeted by AvrPto but was then recruited to function in effector perception by

mimicking the operative targets of Pto (target mimicry). In the case of RCR3, this decoy has most likely evolved by target duplication since it is phylogenetically closely related to the presumptive operative target PIP1 and the *Rcr3* and *Pip1* genes reside at the same locus in the tomato genome, suggesting that they were generated by gene duplication and divergent evolution (Tian et al., 2007). On the other hand, target mimicry might best explain the evolution of the AvrBs3 binding box in the *Bs3* promoter though it also could have originated from a recombination event between the promoter of an operative target with a flavin monooxygenase gene.

Although the various examples illustrated above are plausible, there is at least one observation that is not explained by the Decoy Model. The Decoy Model predicts that features that are not relevant to effector perception will be lost during decoy evolution. However, if Pto and Rcr3 are specialized decoy proteins, why are they active enzymes? Our current knowledge is insufficient to provide a satisfactory answer, but three scenarios might apply. First, these decoys may have evolved only recently and have not yet lost their enzymatic activity. This explanation contradicts the observation that both the *Pto* and *Rcr3* genes are ancient and accumulated sequence variation in regions without affecting their activity (Rose et al., 2005; Shabab et al., 2008). Second, these decoys may have additional functions unrelated to pathogen perception. It is common for proteins to have multiple functions. Pto, for example, could act in a signaling pathway that includes Pti phosphorylation (Zhou et al., 1995). Similarly, Rcr3 could function in processes unrelated to defense. However, this explanation contradicts the observation that Rcr3 expression is defense related (Shabab et al., 2008) and that the absence of Pto is common in plant populations (Rose et al., 2005). Third, catalytic activity of these decoys could be required for effector perception. Indeed, kinase activity of Pto is essential for AvrPto perception (Rathjen et al., 1999), and AvrPto phosphorylation is required for its recognition, though this phosphorylation occurs in the absence of Pto (Anderson et al., 2006). It has not been investigated whether or not Rcr3 activity is required for Avr2 perception. Avr2 itself is probably not cleaved by Rcr3, and Rcr3 inhibition by other inhibitors is insufficient to activate Cf-2 signaling (Rooney et al., 2005). However, Rcr3 activity might be required for autocatalytic removal of its prodomain to create the binding site for Avr2. In conclusion, the observation that decoys can be active enzymes is not fully understood. Future work will shed light on these issues and help to clarify how decoys evolve in plant pathosystems.

GENERATING EXPERIMENTAL EVIDENCE FOR GUARD AND DECOY MODELS

Providing experimental evidence to discriminate between the Guard and Decoy Models is challenging for several reasons. First, the two models are not necessarily mutually exclusive since intermediate stages may occur while a guardee evolves into a decoy. As a result, many of the predictions of the Guard Model also hold for the Decoy Model. Second, the issue of redundancy of effector targets can confound genetic analyses. For example, if effectors have several operative targets, then removing one of the targets may not alter the phenotype. Third, the definition of decoys is based on a lack of evidence for a role in defense, which is always difficult to establish with certainty. Despite these limitations, it remains possible to devise genetic experiments to discriminate between the Guard and Decoy Models (Figure 3). These assays involve comparing the effect of the presence or absence of the guardee/decoy on pathogen fitness in genetic backgrounds that lack the corresponding R protein and other effector targets. For example, pathogen fitness can be compared between *Pto* and *pto* tomato plants lacking both tomato *FLS2* and *Prf* to determine whether Pto contributes to pathogen fitness and thus discriminate between the Decoy and Guard Models. The type of test depends on the presumed action of the effector: Does the effector promote host processes that are positive for the pathogen (e.g., release of nutrients) or prevent responses that are negative for the pathogen (e.g., suppression of defense responses)? For instance, the contribution of RIN4 to host defense needs to be assessed in the presence of the Cys protease AvrRpt2 to reveal contributions by the cleaved RIN4. On the other hand, the contribution of Pto to host defense should be assessed in the absence of the kinase inhibitor AvrPto. In summary, although such experiments can be difficult to set up since they require the identification of all operative targets, the generation of the appropriate genetic material, and the development of quantitative assays for pathogen fitness, these assays offer a direct test to exclude or support the Decoy Model.

CONCLUSION

The Decoy Model remains to be experimentally demonstrated, but it is consistent with a number of recent observations and provides a challenging platform for future experiments. We hope that new data and experiments will challenge the Decoy Model and generate a basis for a deeper understanding of effector perception in plants, ultimately leading to novel approaches to manipulate innate immunity and improve pathogen resistance.

ACKNOWLEDGMENTS

We thank Brande Wulff, David Mackey, Edgar Huitema, Frank Takken, Jane Parker, John Rathjen, Paul Schulze-Lefert, Ralph Panstruga, Réka Tóth, Sebastian Schornack, Silke Robatzek, and the three anonymous reviewers for useful discussions, suggestions, and/or critical reading. R. A.L.V. is supported by the Max Planck Society and S.K. is supported by The Gatsby Charitable Foundation.

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