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Review

Microbial avirulence determinants: guided missiles or antigenic flak?

PARASKEVI SKAMNIOTI[1,2] AND CHRISTOPHER J. RIDOUT[1]*

[1]*Department of Disease and Stress Biology, John Innes Centre, Norwich Research Park, Colney Lane, Norwich NR4 7UH, UK*

[2]*Department of Plant Sciences, University of Oxford, South Parks Road, Oxford OX1 3RB, UK*

SUMMARY

Avirulence (avr) determinants are incompatibility factors which elicit host plant defence responses in a gene-for-gene manner. They are produced by fungi, bacteria and viruses, and their recognition by resistance genes has been extensively studied for decades. But why should a microbe keep a molecule that allows it to be recognized? One argument is that avr genes perform some essential function and must be kept despite giving the pathogen away. Many bacterial avr determinants have been shown to be effectors, which contribute to virulence and aggressiveness. If this were always the case, mutants lacking these essential molecules would be at a serious disadvantage. Some disadvantage has been shown for a small number, but for the majority there is no effect on virulence. This has been explained by functional redundancy for bacterial and fungal avr determinants, with other molecules compensating for the deletion of these essential genes. However, this argument is counter-intuitive because by definition these individual genes are no longer essential; so why keep them? With increasing numbers of avr genes being identified, efforts to elucidate their function are increasing. In this review, we take stock of the accumulating literature, and consider what the real function of avr determinants might be.

AVIRULENCE GENES ARE REMARKABLY DIVERSE

Avirulence (avr) genes isolated to date show remarkable sequence and functional diversity, and those from different plant pathogens do not seem to share common features. Avr determinants from plant pathogenic bacteria belong to the group of effector proteins which are transferred into host cells by the type three secretion system, TTSS (extensively reviewed in Alfano and Collmer, 2004). Some bacterial avr determinants have known functions or homology to previously characterized genes. For example, AvrD from *Pseudomonas syringae* pv. *tomato* is an enzyme catalysing synthesis of hypersensitive response (HR)-eliciting syringolides in RPG4 soybean (Kobayashi *et al.*, 1990). AvrPphB and AvrRpt2, again from *P. syringae* pathovars, have cysteine protease activity (Hotson and Mudgett, 2004; Zhu *et al.*, 2004). Other avr determinants have no homology to other known genes/sequences present in the databases, but are extensively represented in bacterial plant pathogens. The AvrBS3 family of avr determinants are found in several pathogenic *Xanthomonas* species and have a repeat domain structure and nuclear localization signals (reviewed in Van't Slot and Knogge, 2002). AvrB, originally identified from *P. syringae* pv. *glycinea*, has been extensively investigated (Quirino and Bent, 2003) but has no sequence or structural homology to other proteins (Lee *et al.*, 2004; Tamaki *et al.*, 1988). Secretion signals for the TTSS have now been identified in different pathogenic bacteria, enabling more complete inventories of effector proteins to be identified. Homologues of the characterized avr proteins have been identified in *Xanthomonas campestris* and different pathovars of *P. syringae* (Guttman *et al.*, 2002; Chang *et al.*, 2005; Roden *et al.*, 2004). Whole genome sequencing of several bacterial plant pathogens will undoubtedly reveal further homologues of avr determinants (Moreira *et al.*, 2004).

In the fungal world, the most extensively studied avr determinants are from *Cladosporium fulvum*. Avr2, Avr4, Avr4E and Avr9 are small cysteine-rich protein molecules, readily purified as elicitors as they are secreted into the culture medium (de Wit *et al.*, 2002; Joosten and de Wit, 1999). Similarly, NIP1 from *Rhynchosporium secalis* (Wevelsiep *et al.*, 1991) and SIX1 (*S*ecreted *I*n *X*ylem) from *Fusarium oxysporum* (Rep *et al.*, 2004, 2005) were isolated from the culture medium. Apart from the presence of cysteine residues, the primary sequences of these and the *C. fulvum*-secreted avr proteins bear no resemblance to each other or any other known protein. *Magnaporthe grisea*, which causes rice blast disease, has become a model fungus for investigating pathogenicity (Talbot, 2003). The pathogen is amenable to genetic analysis and manipulation, and avirulence genes *AVR-Pita* (originally called AVR2-YAMO, Valent *et al.*, 1991) and *ACE1* have been isolated by map-based cloning (Bohnert *et al.*, 2004; Orbach *et al.*, 2000). *AVR-Pita* is located entirely within the most distal 1.5 kb of a chromosome and encodes a

* *Correspondence*: E-mail: ridout@bbsrc.ac.uk

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