

Structure of avr Gene Proteins

Although *avr* genes are quite different, some of them have common structural characteristics that allow grouping of *avr* genes into distinct families. The best known *avr* gene group is the *Xanthomonas avr* gene family, called *pth* (for pathogenicity) genes by some. Members of this gene family are found in different species and pathovars of the bacterium *Xanthomonas*. They encode proteins that, in their central part, have from 13 to 23 copies of a nearly identical 34 amino acid repeat unit. *avr/pth* genes cause the hypersensitive response and are also required for the induction of angular leaf spot symptoms of cotton and for citrus canker disease. Elicitation of these very different symptoms (leaf spots, cankers, the HR) is determined by a single or a few amino acid differences in the repetitive regions of these genes. Among fungal *avr* proteins, the *Cladosporium fulvum*-encoded *avr2* is a cysteine-rich protein of 78 amino acids that has a signal peptide of 20 amino acids for external targeting; the *Cf avr4* protein consists at first of a 135 amino acid preprotein, which upon secretion is processed at both ends, resulting in an 86 amino acid protein; and the *Cf avr9* protein at first consisting of a precursor protein of 63 amino acids, which is further processed into a 28 amino acid peptide. All three *Cf avr* proteins are secreted in the apoplastic space of tomato leaves, are localized in the plasma membrane, and contain an extracellular leucine-rich region (LRR), a transmembrane domain, and a short cytoplasmic tail. The *Magnaporthe grisea*-encoded *avr-Pi-ta* protein consists of 223 amino acids but is processed into a 176 amino acid protein that has homology to zinc dependent metalloproteases. The *Pi-ta avr* protein is cytoplasmic and contains a nuclear-binding site (NBS) and a leucine-rich carboxyl terminus. The viral *avr* proteins elicit corresponding plant resistance R genes that encode cytoplasmic proteins. These proteins consist, in the case of PVX and TCV, of either LZ-NBS-LRR domains or, as in TMV, of TIR-NBS-LRR domains (LZ, leucine zipper; TIR, toll interleukin 1 receptor).

Function of avr Gene Proteins

So far, the functions of only one *avr* gene, *avrD*, have been determined. The *avrD* gene is present in the bacterium *P. syringae* pv. *tomato*, but *AvrD* alleles are present in soybean *P. syringae* pv. *glycinea* and other hosts. *avrD* encodes syringolide elicitors, which react with the receptor protein of R gene, Rpg4 of soybean, and confers avirulence on soybean. It has no effect on the virulence of the bacterium.

Commented [SQ1]: 20-30 amino acid, hydrophobic amino acid

Commented [SQ2]: Zinc and calcium for work

Commented [SQ3]: Enzyme that break down proteins

The function of fungal *avr* proteins is not known with certainty. The timing and location of their expression suggest a role in the infection process, but so far no virulence function has been reported for most such proteins. In the case of the *avrPi-ta* protein, direct interaction was detected between the mature *avrPi-ta* protein and the leucine-rich domain of the *Pi-ta* R gene protein. This finding is the first experimental evidence consistent with the proposed model that *avr* proteins interact directly with the corresponding R proteins. In the case of *tobacco mosaic virus*, causing the hypersensitive response in *Nicotiana sylvestris* tobacco carrying the N1 gene for resistance, the avirulence function and thereby the elicitation of hypersensitive response seem to reside in the presence of certain amino acids on the coat protein of the virus: N1 gene containing plants transformed with only the gene of such TMV elicitor coat proteins, without inoculation with the virus, exhibited the hypersensitive response in the form of reduced growth, chlorotic and necrotic patches, and eventual collapse of entire leaves. Plants transformed with mutant weakly eliciting or non-elicitor coat proteins expressed respectively weaker or no hypersensitive response. In at least some viral infections then, the viral coat protein, which is produced within the cell, appears to function as a specific elicitor that activates the hypersensitive response in plant cultivars that carry the corresponding R gene for that virus.

Role of *avr* Genes in Pathogenicity and Virulence

Most *avr* genes tested so far play no role in pathogenicity or virulence of the pathogen, as even when *avr* genes are inactivated by mutation, susceptible hosts continue to be susceptible. Some *avr* genes, however, e.g., the *avrBs2* gene from the bacterium *X. campestris* pv. *vesicatoria*, encode proteins that are also necessary for pathogenicity. This is shown by the fact that this *avr* gene is present in all strains of this pathovar, whereas mutants lacking the *avr* gene lose pathogenicity on all susceptible hosts but do not gain virulence on any previously resistant hosts. However, several *avr* genes, such as the *pthA* gene from *X. citri* and *avrb6* from *X. campestris* pv. *malvacearum*, both members of the *Xanthomonas avr/pth* gene family, encode proteins that act as pathogenicity or virulence factors. For example, they enhance the virulence of a weakly pathogenic leaf-spotting strain of *X. citrumelo*, enabling it to cause canker like lesions on its host; they may act as pathogenicity factors, e.g., *pthA* is required for the pathogenicity of *X. citri* on citrus to cause the typical citrus canker disease; and act as avirulence genes, e.g., by causing *pthA*-transformed strains of *X. phaseoli* and *X. campestris* pv. *malvacearum* that, respectively, infect bean or cotton, but not citrus, to

cause the hypersensitive response on their respective hosts bean and cotton while remaining non-pathogenic on citrus. The role of fungal *avr* genes in pathogenicity and virulence of the pathogens involved is mostly unclear. In some cases, *avr* proteins seem to react with other proteins that play an intermediate role in transmitting the signals for plant defense. In a few cases, as in the *avr* Pi-ta protein, they seem to interact directly with the R protein and to set off a cascade of defense reactions. In viruses, a certain segment of a particular coat or replicase protein seems to interact with the host R gene. Most of these statements, however, need further experimentation to support their validity.

How Do R Genes Confer Resistance?

The mechanisms by which R genes bring about disease resistance to a plant against a specific pathogen are not yet understood. It is believed that the elicitor molecule produced by an *avr* gene of the pathogen is recognized by a specific plant receptor encoded by an R gene. What happens next is mostly speculation. Following recognition of the elicitor by the receptor molecule, one or more **kinase enzymes** may become activated, which then amplify the signal by **phosphorylating**, and thereby energizing, other kinases and other enzymes. This leads to a cascade of biochemical reactions that, in ways that are still unclear, result in the hypersensitive response and, thereby, localized host resistance at the point of attack by the pathogen. Of course, in many cases, the hypersensitive response is followed by the development of various levels of systemic acquired resistance (SAR), which is expressed in the vicinity of attack as well as in distant parts of the plant.

Commented [SQ4]: catalyzes the transfer of phosphate groups from high-energy, phosphate-donating molecules to specific substrates.

Commented [SQ5]: introduce a phosphate group into (a molecule or compound)