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Gene-for-Gene Recognition in Plant-Pathogen Interactions

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Gene-for-gene recognition in plant–pathogen interactions

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SUMMARY

Mediated through specifically matching allele pairs in the host and pathogen (at resistance and avirulence loci respectively), plants have a refined and highly discriminating capability to recognize and differentiate among genetic variants of potential pathogens. Knowledge of pathogen recognition by plants has primarily resulted from research associated with the selective breeding of crop species for disease resistance. The phenomenon is well described at the cellular, whole plant and population levels in terms of genetics, histology and associated biochemistry. However, a full mechanistic understanding is only now becoming feasible following the isolation and sequencing of putatively interacting plant and pathogen genes.

It is evident that the number of genes in plant genomes capable of specific pathogen recognition is large and that these genes are commonly clustered in complex loci sometimes comprising genes involved in the recognition of more than one taxonomically unrelated pathogen. For some genes, alleles with different recognition capabilities have been identified while there is also evidence for the existence of genes expressing identical recognition capability present at different loci in the same species as well as in different plant species. It seems likely that resistance genes are members of substantial multi-gene families and there is evidence that novel recognition capability may be created through interallelic recombination events.

In natural populations, there can be a spatial mosaic of resistance genotypes with individuals carrying a range of resistance alleles at a varying number of loci, some of which will exert selection on the pathogen population only under certain conditions and at certain stages of development. There have been few studies of the costs and benefits of particular resistance genes in the presence or absence of pathogen variants against which a gene may be effective or ineffective. The stage of host development at which the pathogen exerts the greatest impact on survival and fecundity is of particular relevance in this respect, as is the influence of mother plant resistance on the characteristics and quantity of inoculum to which progeny are exposed.

1. INTRODUCTION

The existence of plant genes providing resistance to pathogens was demonstrated soon after the rediscovery of Mendel's seminal studies on inheritance. Biffen (1905) demonstrated that a single locus was responsible for the resistance of some wheat cultivars to yellow rust (*Puccinia striiformis*). Since this early work, many hundreds of genes associated with resistance to a diversity of pathogens have been identified in numerous plant species, primarily those of agricultural significance. At an early stage, it was discovered that genes at different loci could be responsible for resistance to different pathogenic variants (pathotypes) (McRostie 1919) but the full significance of this observation only became evident after the gene-for-gene relationship was elucidated by Flor (1956, 1971) in the course of 40 years of research on the interaction between flax (*Linum ultissimum*) and the rust, *Melampsora lini*.

It became evident, following Flor's classical work, that for many host–parasite relationships, matching gene-pairs (resistance and avirulence genes respectively) controlled the outcome of different genotype combinations (Crute 1985). This paper provides a short and selective discussion of information about genotype-specific recognition of pathogens by plants within the context of genome organization, phenotypic expression, natural plant population structure and evolution.

2. THE GENE-FOR-GENE RELATIONSHIP

In gene-for-gene relationships, the expression of resistance or susceptibility of the host to a particular pathogen is conditional on the pathogen genotype and the observed virulence of the pathogen is conditional on the host genotype. Specifically matching gene-pairs determine the outcome of any particular genotype × genotype interaction. Compatibility (uninhibited

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Table 1. *Features of a hypothetical gene-for-gene relationship involving three interacting gene pairs*

(3 represents complete compatibility (susceptibility/virulence) and 0 represents the highest level of incompatibility. Gene pairs have an epistatic relationship such that *R1*/*A1* is epistatic to *R2*/*A2* which is epistatic to *R3*/*A3*.)

			pathogen genotypes							
			A1	*a1*	*A1*	*A1*	*a1*	*a1*	*A1*	*a1*
			A2	*A2*	*a2*	*A2*	*a2*	*A2*	*a2*	*a2*
			A3	*A3*	*A3*	*a3*	*A3*	*a3*	*a3*	*a3*
host genotypes										
R1	*R2*	*R3*	0	1	0	0	2	1	0	3
r1	*R2*	*R3*	1	1	2	1	2	1	3	3
R1	*r2*	*R3*	0	2	0	0	2	3	0	3
R1	*R2*	*r3*	0	1	0	0	3	1	0	3
r1	*r2*	*R3*	2	2	2	3	2	3	3	3
r1	*R2*	*r3*	1	1	3	1	3	1	3	3
R1	*r2*	*r3*	0	3	0	0	3	3	0	3
r1	*r2*	*r3*	3	3	3	3	3	3	3	3

pathogen development and reproduction in the absence of an effective host defence response) is the outcome of a host *x* pathogen combination unless an allele for resistance at a particular host locus is specifically matched by an allele for avirulence at a particular pathogen locus; under these circumstances, degrees of incompatibility (reduced pathogen development and reproduction associated with an effective host defence response) are expressed depending on the particular matching gene-pair. Gene pairs resulting in incompatibility are epistatic over gene pairs that would otherwise result in compatibility. Gene pairs conditioning higher degrees of incompatibility are epistatic over gene pairs associated with lower degrees of incompatibility, although phenotypic variation indicative of genetic additivity has also been reported when more than one gene pair conditioning incompatibility is effective. Table 1 illustrates the features of a hypothetical gene-for-gene relationship involving three epistatic matching gene pairs when 3 represents complete compatibility and 0 represents the highest degree of incompatibility. Assuming that genes controlling the interaction are at separate loci, and ignoring heterozygotes, there are 2^n possible host and pathogen genotypes and $(2^n)^2$ unique genotype *x* genotype combinations (where *n* is the number of matching gene pairs).

It appears that there is a large number of genes in a plant species capable of functioning in the genotype-specific recognition of pathogens. For example, in wheat, a total of more than 90 genes have been identified which condition isolate specific resistance to three rust species (*Puccinia striiformis*, *P. recondita* and *P. graminis*) and powdery mildew (*Erysiphe graminis*). Only one of these genes (*Lr20* and *Sr15*) is thought to be involved in the recognition of more than one pathogen (see Crute 1985 for references).

The suggestion that gene-for-gene specificity is in some way an artifact of cultivation are not substantiated by an increasing number of investigations of natural plant pathosystems. In fact, the narrow genetic base of some crop species may mean that they are relatively impoverished with respect to genes for pathogen recognition; the need to exploit additional genetic diversity among wild progenitors is a familiar approach for plant breeders seeking to improve disease resistance. Studies on two ruderal weed species (*Senecio vulgaris* and *Arabidopsis thaliana*) have readily demonstrated the existence of a substantial number of genes identified by their ability to discriminate among a relatively restricted sample of pathogen isolates (*Erysiphe fischeri* and *Peronospora parasitica* for the two host species, respectively) (Harry and Clarke 1986; Bevan *et al.* 1993*a*; Holub *et al.* 1994).

The gene-for-gene relationship provides the basis for the discrimination of pathogenic variants (pathotypes) by their virulence or avirulence on particular genotypes of a host species. However, in addition to this sub-specific variation among genotypes within species, pathotypic variation is also frequently evident at the level of host species, genus or family. For example, the downy mildew pathogen, *Peronospora parasitica*, is only pathogenic on cruciferae but, although it has been recorded on over 50 host genera, pathotypes are restricted to the host genus or species of origin. Hence, isolates from *Arabidopsis thaliana* are avirulent on *Brassica* spp. and vice versa. Pathotypes of pathogens discriminated by their specific adaptation to higher host taxa are often formally ascribed to *formae speciales*. It is clear that such specificity represents the outcome of a co-evolved parasitic symbiosis and while there is some evidence that this level of specificity may also be a reflection of gene-for-gene recognition the subject continues to be debated (Heath 1987, 1991; Newton & Crute 1989; Tosa 1989, 1992).

3. THE ORGANIZATION AND STRUCTURE OF PATHOGEN RECOGNITION LOCI

Studies on the inheritance of isolate-specific resistance to flax rust by Flor and others have identified the existence of genes expressing at least 32 different specificities and organized in five linkage groups (*K*, *L*, *M*, *N* and *P*) (see Islam & Shepherd 1991*a* for a recent review). While the existence of separate loci within linkage groups *K*, *M*, *N* and *P* have been demonstrated, the 14 specificities at the *L* locus appear to be allelic. Studies have been made of progeny from test crosses between a homozygous susceptible genotype and 27 of the possible 91 *L* group heterozygotes. Among large numbers of individuals, rare susceptible plants were located as were plants expressing non-parental resistance phenotypes (so-called 'modified recombinants'); however, no individuals were found which expressed the combined specificity of both *L* group parents. Some susceptible individuals yielded resistant revertants on selfing and plants expressing novel specificity to nine different rust pathotypes were identified among progeny of revertants, susceptible recombinants and modified recombinants. Interallelic recombination, providing the mechanism for the generation of novel recognition capability, has been postulated to explain these data

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(Islam *et al.* 1989, 1991; Islam & Shepherd 1991). Although there are few other well-authenticated examples of allelism at parasite recognition loci in plants, it is entirely feasible that variants of susceptibility alleles (that would be impossible to discriminate phenotypically) could also play a part in the generation of novel specificity through rare interallelic recombination events.

A more common circumstance than allelism appears to be the clustering of genes capable of specific parasite recognition within large, complex loci. Saxena & Hooker (1968) conducted a classical study of genes in maize for resistance to the rust fungus, *Puccinia sorghi* located on the short-arm of chromosome 10. Sixteen recognition genes (*Rp5*, *Rp6* and 14 genes previously thought to be alleles of *Rp1–A* to *N*) map within this region, the existence of separate linked loci being demonstrated by recombination events and predicted patterns of resistance to diagnostic isolates of the pathogen. More recent studies of loci at *Rp1* have indicated meiotic instability observed as the unexpected appearance of susceptible individuals in test-cross progeny between parents homozygous susceptible and resistant at *Rp1* loci (Hulbert & Bennetzen 1991; Hong *et al.* 1993; Sudupak *et al.* 1993). This phenomenon has been shown to result from unequal crossing-over as indicated by non-parental flanking RFLP loci. Mispairing of sequence duplications provides a mechanism leading to gene duplication and associated elimination. It is probable that the necessary repeats to facilitate mispairing are provided in *Rp1* regions which represent a multigene family of duplicated homologues distinguished by their diagnostic pathogen recognition capability. Additional evidence for the occurrence of repetitive sequences in the *Rp1* region was provided by the discovery of two genomic clones which identify a variable number of loci tightly linked to *Rp1* in different maize lines.

In lettuce (*Lactuca sativa*), identified linkage groups contain genes for recognition of several unrelated parasites. One linkage group contains a gene for resistance to an aphid species (*Pemphigus bursarius*) in addition to eight genes for specific resistance to downy mildew (*Bremia lactucae*). Two further downy mildew resistance genes occur in a linkage group along with a gene for resistance to turnip mosaic virus and a gene for resistance to the root pathogen, *Plasmopara lactucae–radicis* (Landry *et al.* 1987; Kesseli *et al.* 1993). Genes in tomato (*Lycopersicon esculentum*) for resistance to root-knot nematode (*Meloidogyne* spp.) and leaf mould (*Cladosporium fulvum*) are also linked (Dickinson *et al.* 1993; Jones *et al.* 1993).

There are probably several selective advantages contributed by the common existence of complex loci such as those described above. Such arrangements allow multiple parasite recognition capabilities to be assembled and retained in a single haplotype while out-breeding and recombination can readily generate new combinations of specificities. Mispairing, intragenic recombination and gene duplication may facilitate the evolution of novel recognition capability.

In contrast to the occurrence of complex loci comprising several genes with distinct recognition capabilities, there is recent data also indicating the existence of functionally identical duplicate genes at independent loci (Teverson *et al.* 1994). In the interaction between *Phaseolus* beans and the bacterial pathogen, *Pseudomonas syringae* pv. *phaseolicola*, digenic and trigenic segregation ratios have been observed among F_2 progeny from crosses between cultivars previously demonstrated to carry functionally identical recognition genes as indicated by their reaction to transconjugant strains of the bacterium carrying single cloned avirulence genes (*avrPphB1.R3* and *avrPphC1.R1*). (All notations for bacterial avirulence genes follow the proposals of Vivian and Mansfield (1993)). Intriguingly, one of the genes apparently occurring at duplicate loci (*R3*) appears to be identical or tightly linked to the so-called *I* gene in bean which provides isolate specific resistance to bean common mosaic virus and several other related potyviruses (J. D. Taylor, personal communication).

The existence of genes in different plant species with seemingly identical recognition capability has also now been firmly established. Sequencing of two avirulence genes, isolated respectively from the crucifer pathogen, *Pseudomonas syringae* pv. *maculicola* (*avrPmaA1.RPM1*) and the pea pathogen, *P. syringae* pv *pisi* (*avrPpiA1.R2*), proved them to be near-identical. In transconjugant strains of the two pathovars these avirulence genes also exhibited identical specificity of interaction with the respective host genes: *R2* (from pea) and *RPM1* (from *Arabidopsis thaliana*) indicating functional homology (Dangl *et al.* 1992). Furthermore, in bean, recognition genes at two independent loci were identified by a transconjugant of the bean pathogen *Pseudomonas syringae* pv. *phaseolicola* carrying *avrPpiA1.R2* (Fillingham *et al.* 1992).

Martin *et al.* (1993) have recently reported the cloning and sequencing of a gene from tomato (*Pto*) which provides specific resistance to isolates of *Pseudomonas syringae* pv. *tomato* carrying the matching avirulence gene, *avrPto.C1*. It has been demonstrated that at least six homologous expressed genes are clustered at the *Pto* locus and homologues of *Pto* occur in all 11 other plant species examined. Somewhat unexpectedly, the *Pto* protein has similarity with serine–threonine protein kinases and has no obvious membrane-spanning domain suggesting a signal transduction rather than a receptor function.

On the basis of all the evidence so far available, it would not be surprising if plant genes, identified by their specific pathogen-recognition capability, frequently prove to be members of substantial, linked, multi-gene families well-conserved between plant families (Pryor 1987).

4. NATURAL PATHOSYSTEMS

Most of what has been learnt about the genetics of plant–pathogen interactions results from investigations of crop pathosystems but such analyses must inevitably reflect their man-guided and recent evolutionary history. By contrast with their progenitors, most crops probably have a relatively narrow

genetic base. For example, horticultural forms of *Brassica oleracea* (cabbage, cauliflower, broccoli and Brussels sprout) are unusual phenotypic variants likely to have originated once and thereby harbouring relatively little genetic diversity. Most crops grow in stable or even optimized environments with a sufficiency of nutrients and water and minimization of competition with other species (weeds); this represents a marked contrast with natural plant populations. While the sizes of natural plant populations may fluctuate widely over time, particularly in the case of ruderal annual species, the sizes of crop populations remain relatively stable under the influence of frequent replacement by man. Given these and other contrasts between crop and natural pathosystems, it seems probable that artificial selection for improved disease resistance in crops could favour a restricted sub-set of the genes naturally selected to defend plants from pathogens.

In addition to the large numbers of specific recognition genes that have been identified in some wild plant species, natural populations also exhibit considerable spatial diversity with respect to these genes. A study of resistance in the annual composite weed, groundsel (*Senecio vulgaris*) to the powdery mildew pathogen, *Erysiphe fischeri*, revealed ten different patterns of phenotypic response to five pathogen isolates among a sample of 75 plants from $1\,m^2$ (Bevan *et al.* 1993*b*) and little additional variation was evident in a sample derived from $6\,km^2$.

The characteristics commonly associated with resistance genes selected in crop breeding programmes include: dominance and a high level of expressivity, often resulting in the complete absence of pathogen reproduction, at all developmental stages, in all tissues and over a range of environments. There are exceptions to these generalizations and temperature-sensitive genes or those which exert their effect only in adult plants are not uncommon in crop species (reviewed by Crute 1985). There are, however, some indications from recent studies with non-crop species that resistance genes conforming to the general pattern of those common in crops do not necessarily predominate. Genes with relatively low expressivity appear to be commonly located in *Arabidopsis thaliana* for response to different isolates of *Peronospora parasitica* (downy mildew) and in *Senecio vulgaris* for response to different isolates of *Erysiphe fischeri* (Bevan *et al.* 1993*a*; Holub *et al.* 1994). Furthermore, in the former case, genes whose expression is associated with extensive necrosis of the mesophyll in the absence of fungal colonization (referred to as 'pitting') have also been observed (Holub *et al.* 1994); such phenotypic expression of resistance is not commonplace in crops and would probably be consciously rejected in the process of selection. In *Senecio vulgaris*, resistance that was only expressed in seedlings or only in adult plants or only at certain temperatures was also observed (Bevan *et al.* 1993*c*).

5. DISCUSSION

A substantial number of genes occur in plant species which allow the genotype-specific recognition of potential pathogens; the actual numbers for any particular individual or species are unknown, their identification being dependent on diagnostic pathogen variants. These genes occur within plant genomes in distributed clusters reminiscent of multi-gene families and considerable variation occurs in the phenotypic expression of incompatibility dependent on the particular interacting gene-pair. The common occurrence of these genes, at least in herbaceous annuals and deciduous perennials, suggests that they are selectively advantageous even though pathotypes capable of rendering the resistance they confer ineffective are frequently encountered.

There is likely to be a cost to the individual of carrying any particular recognition gene both in the presence and the absence of the pathogen. Similarly, in the presence of the pathogen, there is a benefit of carrying an effective recognition gene and a cost of not doing so. The benefit might be considered to be greatest at the stage of plant development or under those environmental conditions when the pathogen would be expected to exert its greatest effect on host survival and fecundity. Critical studies are required to examine these costs and benefits and to determine when and under what circumstances pathogens are likely to exert strong selection.

The progeny of inbreeding host species in the proximity of a diseased mother plant are likely to be particularly vulnerable to pathogenesis due to their genetic identity. Hence, genes which are effective and exert selection on the pathogen only in adult plants would reduce levels of inoculum to which establishing seedlings might be exposed. Alternatively or in addition, the occurrence among progeny of rare recombinant variants, expressing novel pathogen recognition capability, would allow survival of the potentially well-adapted parental genotype otherwise threatened by a pathotype with matching virulence. While outbreeding allows for the regular reassortment of genes involved in pathogen recognition, genetic mechanisms that facilitate the creation of new pathogen recognition capability may be an essential prerequisite for successful adoption of an inbreeding reproductive strategy.

An important feature of competitiveness in relation to the successful colonization of a new habitat by immigrants is likely to be the possession of a novel and more effective specific pathogen recognition capability in comparison with previously established individuals already subject to pathogenesis by matching pathotypes.

Although crop cultivars, in common with other plants, may carry many genes for specific recognition of pathogens, they have frequently been selected for genetic uniformity; examples of extreme uniformity are provided by some vegetatively propagated crops and F_1 hybrid cultivars of seed-propagated crops. The same genotypes may also be grown over prolonged periods of time. The greater prominence of diseases in crops compared to plants in natural plant populations may be a direct consequence of this circumstance. In contrast, natural plant populations comprise a

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dynamic temporal and spatial mosaic of genotypes carrying variable numbers of different pathogen-recognition genes with variable expressivity. Some of these genes will exert selection on the pathogen population only under certain environmental conditions or at certain stages of host development.

In any diverse population of plants, at any time, some will be selectively advantaged by virtue of the pathogen recognition genes they carry; selection on the pathogen population will ensure that at a different time selection favours different host individuals. In this way, the overall influence of pathogenesis may be minimized with recognition genes enjoying renewed cycles of effectiveness as discontinuities in epidemics and cycles of infection are experienced through the annual and deciduous habit or diverse ecological influences on population stability and size.

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