Predominance of a single major gene for resistance to *Phakopsora pachyrhizi* in a population of *Glycine argyrea*

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Four phenotypic patterns of resistance and susceptibility to nine races of *Phakopsora pachyrhizi* were found among 104 lines derived from seeds collected from 49 plants of a *Glycine argyrea* population. Genetic analyses suggested that the three most common phenotypic patterns were determined by two alleles at a single locus. The fourth phenotypic pattern was represented by only a single line which was not analyzed genetically. These results contrast with those of a related species, *G. canescens*, where ten or more resistance genes were detected within each of two populations, and individuals often possessed two or more resistance genes. It is postulated that *G. argyrea*'s relatively high rate of outcrossing and the apparent prevalence of forms of resistance which delay the development of the pathogen may have contributed to the small number of major resistance genes in this population.

INTRODUCTION

Pathogen infections have been postulated to be a major cause of frequency dependent selection within plant populations (see Burdon, 1987a, for review). Results from several broad scale surveys of host resistance have been consistent with the prediction that host populations should be polymorphic in regions where pathogen pressure is significant (Dinoor, 1970; Wahl *et al.*, 1978; Segal *et al.*, 1980; Burdon *et al.*, 1983). However, genetic studies on the pattern of race specific resistance within populations indicate that the level of resistance polymorphism may differ from one host-pathogen combination to another and even between populations within a single host species. High levels of resistance to *Phakopsora pachyrhizi* were found in two populations of *Glycine canescens* (Burdon 1987b). Each population contained ten or more resistance genes. No individual in either population was susceptible to all nine races of *P. pachyrhizi*, and in most cases plants possessed two or three resistance genes. In *Senecio vulgaris*, most populations contained a considerable proportion of individuals which were fully susceptible to eight races of *Erysiphe fischeri* (Harry and Clarke, 1986). However, individuals possessing two or more resistance genes were common within the group

of resistant plants (Harry and Clarke, 1986, 1987). In a third study which investigated resistance to local *Synchytrium decipiens* isolates in the plant *Amphicarpaea bracteata*, Parker (1988a) found that most plants were susceptible, and resistant plants carried only a single resistance gene. The reasons for the differences in resistance patterns among species and among populations within a species have not been extensively tested. Wahl (1970) and Dinoor (1970) have suggested that pathogen pressure is a primary factor in determining the level of resistance within populations. However, it seems likely that other factors, such as host life history and the mating systems of both the host and pathogen, would also have important effects on the patterning of resistance.

In this study we examine a population of *G. argyrea* Tind, with respect to the phenotypic patterning and genetic basis of major gene resistance to the soybean rust pathogen *P. pachyrhizi* Syd. The results are compared to those from a previous study which examined the resistance patterning in two populations of *G. canescens*. *G. argyrea* was chosen for comparison because it grows in a region which we judged to be more conducive to pathogen growth. We based this judgement on statements which indicated that soybean rust is a major disease of soybean along the east coast of Australia and not the interior (Kochman, 1977), and from herbarium records

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which indicate a greater prevalence of *P. pachyrhizi* along the east coast of Australia (Burdon and Lenné, 1989). Further, relative to *G. canescens*, *G. argyrea* displays much higher levels of outcrossing in chasmogamous flowers (Brown *et al.*, 1986), and has a more restricted range (Tindale, 1984).

MATERIALS AND METHODS

G. argyrea seeds were collected by Grant (1986) from chasmogamous flowers at Rainbow Beach, Coolool National Park, Queensland (25°55'S; 153°06'E) in December of 1984. Seeds were nicked,

Table 1 Segregation patterns for resistance/susceptibility to *Phakopsora pachyrhizi* in F₂ seedlings for lines derived from crosses involving a plant displaying Phenotype I or II and a fully susceptible plant

Resistant plant number	Number of crosses*	Testing races†	Heterogeneity χ^2	F ₂ individuals		χ^2 for 3:1 ratio‡
				R	S	
Lines displaying a 3:1 ratio						
0701 1:2	2(1)	R8	0.04	321	91	1.71
0702 2B	2(2)	R2	0.18	452	171	1.86
0704 A	5(4)	R4, R7	2.41	725	272	2.65
0705 A	2(1)	R2, R6	0.42	328	94	1.53
0706 B	2(1)	R7	1.05	289	110	1.27
0711 5A	3(3)	R8, R9	1.77	279	100	0.32
0714 1:2	1(1)	R3	—	179	72	1.63
0717 4A	1(1)	R8	—	79	39	3.66
0719 A	3(1)	R5	5.13	477	162	0.03
0734 1A	2(1)	R7	0.05	177	42	3.65
0735 10:3	2(1)	R6	0.15	142	61	2.50
0735 10:5	1(1)	R1	—	137	53	0.70
0737 4A	1(1)	R3	—	120	34	0.55
0739 1A	2(2)	R9	0.01	145	49	<0.01
0740 1:4	3(3)	R5, R8, R9	0.97	300	78	3.61
0743 1A	2(1)	R4, R8	0.04	174	64	0.36
0746 7:1	3(2)	R1, R9	1.57	242	101	3.38
0747 3:5	1(1)	R8	—	58	28	2.23
0749 A	1(1)	R2	—	130	43	<0.01
0752 A	2(2)	R2, R5	1.54	69	20	0.18
0756 1	1(1)	R4	—	116	43	0.25
0757 3R	3(1)	R4, R5, R6	3.83	355	111	0.29
0759 1	2(1)	R7, R8	1.81	237	96	2.40
0762 1	5(3)	R6	1.44	546	180	0.01
0770 2	3(2)	R5, R7, R8	3.55	469	145	0.56
0771 B	2(2)	R1	1.06	93	32	0.01
0778 1A	2(2)	R1, R7	0.24	136	44	0.01
0779 1A	2(1)	R3	0.06	171	54	0.07
0780 2:2	2(1)	R8	0.17	264	96	0.45
0781 1A	2(1)	R3	0.27	133	45	<0.01
0782 2:3	2(2)	R6	0.00	144	48	0.00
0799 B	2(2)	R1, R2	0.05	111	35	0.04
Lines not displaying a						
0714 1:5	1(1)	R5	—	336	86	4.56*
0747 3:4	1(1)	R7	—	56	36	9.06**
0748 1A	2(2)§					
C1-1		R3	47.70**	85	197	—
C2-1		R3	2.60	41	32	12.83**
0763 B	1(1)	R4	—	47	67	67.56**
0769 B	2(1)	R2, R7	1.39	562	145	7.37**

* Number of F₁ hybrid plants used to produce seeds for F₂ testing. Values in parentheses are the number of different pollination represented by the F₁ plants.

† Designation of races follows that of Burdon and Speer (1984) and Burdon (1987b).

‡ * = significantly different from a 3:1 ratio at the 5 per cent level; ** = significant at the 1 per cent level. All other values not significant at the 5 per cent level.

§ The two crosses from this line could not be grouped due to significant heterogeneity among inoculation dates for the C1-1 cross. Heterogeneity χ^2 values for the different crosses in this line test for heterogeneity among inoculation dates.

germinated and planted in 20 cm pots filled with 5:3:2 sand, loam and perlite. In most cases more than one seedling was established from the seeds collected from a single field plant. These seedlings were grown as separate lines, but designated as belonging to the same family by shared 0700 numbers. Thus for example, lines 0735 10·3 and 0735 10·5 (table 1) were lines derived from two separate seeds collected from one plant. The 0735 number designates a single plant in Cooloolo National Park. Seedlings were grown to maturity and selfed seeds were collected from each plant and stored separately. These seeds were used to determine the resistance pattern of the original field collected seed.

Pathogen isolates and inoculation procedures

The resistance/susceptibility response to nine separate races of *P. pachyrhizi* was determined by inoculating a minimum of 15 seedlings for each host line by pathogen race combination. For six of the pathogen races, the virulence/avirulence pattern on a set of host differentials has previously been published (Burdon and Speer, 1984). Each of the remaining three races had a unique pattern of virulence/avirulence on the differential set (Burdon, unpublished). Pathogen culture maintenance and inoculation procedures were similar to those of Burdon (1987*b*). The only difference was that infection type ratings were assessed at 14–16 days after inoculation. Infection type ratings were similar to those of Burdon (1987*b*), and were based on the scale shown below:

Infection Type	Symptoms
;	Necrotic flecks only, no sporulation
1	Minute uredia surrounded by relatively large regions of necrotic tissue
2	Small uredia still associated with hypersensitive areas of chlorotic or necrotic tissue
3	Large profusely sporulating uredia with little or no associated chlorotic or necrotic tissue

Symptoms which overlapped two or more infection types were designated by a combination of both infection types. Similarly, intermediate infection types were designated as 1=, 1–, 1+ and so forth. In this study resistant reactions ranged from (;) to (;1), while susceptible reactions were either (2+, 3) or (3) reaction types.

Genetic analysis of disease resistance

The genetic analysis of resistance was investigated by crossing a subsample of the plants with one of two fully susceptible *G. argyrea* accessions, G1622 (CSIRO Division of Plant Industry *Glycine* sp. accession number) and 0702 2A. Attempts were made to generate at least one hybrid from each family. In most crosses the susceptible line was used as the female parent. The hybrid nature of the F₁ plants was verified from infection type response (resistance was assumed to be dominant) and by isozyme analysis on extracts from young leaves. Horizontal starch-gel electrophoresis was performed on crude leaf extracts (Burdon, 1987*b*). The gel buffer systems and enzyme assays were: (1) tris-citrate with lithium borate: aspartate aminotransferase (AAT; EC 2.6.1.1); (2) 5·0 mM histidine, pH 8·0: glucosephosphate isomerase (GPI; EC 5.3.1.9), malate dehydrogenase (MDH; EC 1.1.1.37), and aconitate hydratase (ACO; EC 4.2.1.3).

Selfed seeds from confirmed F₁ hybrids were collected until there was sufficient seed for testing against one of the nine pathogen races. All of the F₂ progeny from a confirmed F₁ hybrid were tested against a single pathogen race. Inoculations and rating procedures were similar to the original selfed seedling tests.

In order to confirm the genetic basis of the observed F₂ segregation patterns, a number of F₂ seedlings were grown to maturity and F₃ seeds collected. All F₃ seedlings derived from a single F₁ hybrid were inoculated with a single pathogen race. The F₃ segregation patterns were used to confirm the occurrence of both homozygous and heterozygous individuals in the resistant portion of the F₂ population, and to establish that susceptible plants bred true.

RESULTS

Only four phenotypic patterns of resistance/susceptibility were found among the *G. argyrea* from Cooloolo National Park (fig. 1). Of the 104 lines tested, 103 displayed one of three main patterns: Lines displaying Pattern I were fully resistant to all nine pathogen races; Pattern II lines appeared to be segregating for resistance to all nine pathogen races; and Pattern III lines were susceptible to all nine pathogen races. For lines displaying Pattern II the number of resistant plants was always greater than the number of susceptible plants, with the exception of two lines from family 0763. In these

Phenotypic Pattern	Pathogen Races									Number of Families	Number of Lines
	R1	R2	R3	R4	R5	R6	R7	R8	R9		
I	■	■	■	■	■	■	■	■	■	24	43
II	▧	▧	▧	▧	▧	▧	▧	▧	▧	25	33
III	□	□	□	□	□	□	□	□	□	20	27
IV	□	□	□	□	□	■	□	■	□	1	1

Figure 1 The four patterns of resistance/susceptibility to nine races of *Phakopsora pachyrhizi* in a population of *Glycine argyrea*. Stippled rectangles indicate a resistant reaction; open, susceptible; and split rectangles indicate that selfed seed from the original plant produced both resistant and susceptible seedlings.

two lines the number of susceptible plants was always greater than the number of resistant. The remaining phenotypic pattern (IV) was represented by the only line tested from family 0736. Resistant reactions ranged from flecks (;) to flecks with light sporulation (;1=), with the most common reactions being flecks or flecks with very light sporulation (;1=). Flecks were usually red-brown to dark brown in colour.

In many instances there was variability in the phenotypic pattern among the different lines from a single family. Multiple lines from the same family were tested for 35 of the 49 families. Of these 35 families, 18 had lines which differed in their resistance pattern.

Thirty seven of the lines which displayed Phenotypic Patterns I or II were crossed with one of the susceptible lines (G1622 or 0702 2A). Thirty two of the lines had F_2 segregation which indicated that resistance in the line was conferred by a single dominant allele (table 1). All the lines which displayed a 3:1 ratio had a (;) to (;1=) resistant reaction with the fleck again being typically red-brown in colour. However, the resistant reaction of the F_2 often showed slightly more sporulation than that of the selfed seed from the resistant parent. This was most evident in lines 0706 B, 0575 3R, 0759 1, and 0780 2.2 where the selfed seedlings from the original parent always gave a fleck with no sporulation, while a small amount of sporulation was always evident on resistant F_2 seedlings. Given the similarity in the resistance reaction it seemed likely that all lines possessed the same resistance allele.

Of the five lines which did not fit a 3:1 ratio, two lines, 0714 1.5 and 0769 B, had an excess of resistance plants. For the 0714 1.5 line the excess of resistant individuals was mainly due to the data from one inoculation date. If the data for this one

date were excluded, the heterogeneity χ^2 among dates dropped from 7.70 to 2.72 and the F_2 ratio for the remaining dates was 227 resistant to 68 susceptible. The χ^2 for a fit to a 3:1 ratio was 0.45, indicating that this line probably has only a single dominant gene. In contrast the 0769 B line always had a consistent excess of resistant individuals for all inoculation dates, although the segregation data fit neither a 3:1 or 15:1 ($\chi^2 = 245.31$) ratio.

The remaining three lines displayed an excess of susceptible individuals. Line 0747 3.4 had an F_2 ratio which was consistent with a 9:7 ratio for resistance conferred by two dominant genes acting together ($\chi^2 = 0.80$). Whether this truly indicates a two gene system is unclear since a related line from the same family (and from the same pod), 0747 3.5, had a ratio consistent with a 3:1 ratio. Although the 0747 3.5 line did have a slight excess of susceptible plants in the F_2 it did not fit a 9:7 ratio ($\chi^2 = 4.37$). Crosses from the 0748 1A line were very perplexing, the different inoculation dates for cross C1-1 could not be grouped because they displayed a great deal of heterogeneity. The second cross involving this line, C2-1, did not fit a 3:1 ratio, but instead fit a 9:7 two gene ratio ($\chi^2 < 0.01$). Therefore, it is possible that some plants in the population were protected by a two gene resistance system. However, this possibility must be tempered by the behaviour of other crosses from the two families displaying the 9:7 ratios. Further, the resistance reaction of both lines displaying a 9:7 ratio was similar to that of lines displaying a 3:1 ratio.

The final line, 0763 B, was the only line where the susceptible plants in the F_2 consistently outnumbered the resistant plants. However, the segregation ratio did not fit a 1:3 ratio ($\chi^2 = 16.01$). The cause of this unusual segregation ratio has not been determined.

Table 2 Segregation patterns for resistance/susceptibility to *Phakopsora pachyrhizi* in F₃ lines of five crosses involving plants which displayed Phenotype I or II

Hybrid number	F ₂ phenotype	Number of families	Testing race*	Heterogeneity χ^2	F ₃ phenotype		χ^2 for 3:1 ratio
					R	S	
0704 A C3-1	R	6	R7	—	241	0	—
	R	6	R7	7.30 ^{(5)†}	204	79	1.28
0719 A C1-3	R	6	R5	—	301	0	—
	R	2	R5	0.32 ⁽¹⁾	76	27	0.08
	S	2	R5	—	0	92	—
0757 3R C1-2	R	4	R4	2.30 ⁽³⁾	140	34	3.77
	S	2	R4	—	0	95	—
0770 2 C1-1	R	2	R6	—	100	0	—
	R	12	R6	4.42 ⁽¹¹⁾	526	173	0.02
0780 2:2 C1-1	R	2	R3	—	104	0	—
	R	5	R3	4.10 ⁽⁴⁾	190	67	0.16
	S	1	R3	—	0	45	—

* Designation of races follows that of Burdon and Speer (1984) and Burdon (1987b).

† Number in parentheses indicates the number of degrees of freedom.

We also point out that the non-3:1 ratios were not due to the behaviour of any single pathogen race (table 1). Five separate testing races were found among the host-pathogen combinations which gave non-3:1 ratios. Each of these races was used to test other host lines which displayed 3:1 ratios. It was often the case that lines displaying a 3:1 ratio were inoculated simultaneously with lines which did not.

In order to confirm that susceptible plants were true breeding, crosses were made among seven lines which displayed Pattern III. The F₂ progeny from all such crosses were susceptible.

The presence of a single dominant allele conferring resistance was confirmed by the F₃ segregation patterns of five crosses which displayed a 3:1 ratio in the F₂ (table 2). In four of the five crosses both homozygous and heterozygous resistant individuals were detected. The F₃ segregation patterns of the heterozygous class from all five crosses fit a 3:1 ratio. Susceptible F₂ individuals from three crosses were also grown to produce F₃ seedlings, all of which were fully susceptible.

DISCUSSION

Phenotypic and genetic patterns of resistance in G. argyrea

Two lines of evidence indicated that resistance to *P. pachyrhizi* in the *G. argyrea* population found in the Cooloola National Park was dominated by a single allele. First, was the very high frequency

of lines which displayed phenotypic patterns I, II, or III (fig. 1). These patterns would be expected when the original progenitor of the line was, homozygous for the allele (Pattern I), heterozygous for the allele (Pattern II), or where the allele was absent (Pattern III). Second, the F₂ segregation pattern of 32 of the 37 crosses which involved lines which displayed Patterns I or II indicated that resistance was in fact conferred by a single dominant allele (table 1). This was further confirmed by the F₃ segregation data were again consistent with the hypothesis that most Pattern I and II lines carried a single dominant resistance allele. The similarity in the resistance reaction of all lines, while not being conclusive, would further indicate that these lines possessed the same resistance allele.

Five lines did not have a 3:1 segregation ratio among the F₂ seedlings (table 1). However, it is unclear whether these lines in fact possessed unique resistance alleles. The phenotypic pattern of resistance/susceptibility and the resistance reaction in these lines were similar to that of lines which displayed a 3:1 ratio in the F₂. Burdon (1987b) found that *G. canescens* plants which possessed different resistance alleles most often differed in either phenotypic pattern or the expression of the resistance reaction. Further evidence against these lines possessing unique resistance alleles came from the behaviour of other crosses in the same family (0747 and 0763), or the fact that the F₂ segregation ratio did not fit any simple Mendelian pattern of inheritance (0714 1.5,

0763 B and 0769 B). The only line which definitely possessed a unique form of resistance was 0736 which displayed pattern IV (fig. 1). Unfortunately it was not possible to genetically test this line.

Comparison with the phenotypic and genetic patterns in other host species

The relatively small number of phenotypic patterns for resistance/susceptibility in *G. argyrea* contrasts with the marked diversity found in other wild systems. Studies of the resistance patterns in wild cereal populations from the Middle East have found populations to be composed of complex mixtures of individuals with different phenotypic patterns (Wahl *et al.*, 1978; Segal *et al.*, 1980; Moseman *et al.*, 1983, 1984). Dinoor (1977) found that resistance patterns to crown rust, *Puccinia coronata*, in wild *Avena* spp. populations were highly variable. Similarly, high levels of resistance variability were found in *Avena* populations from Australia (Burdon *et al.*, 1983).

The most directly comparable work comes from two populations of *G. canescens* which were tested for their resistance patterns using the same nine pathogen races used in this study (Burdon, 1987b). Both populations were highly variable. In one population 12 separate phenotypes were detected in a sample of 14 plants, while in the second 11 phenotypes were found in a sample of 22 plants. In contrast, we found only four phenotypes in a sample of 104 lines from 49 families. Further, no plant in either *G. canescens* population was susceptible to all nine pathogen races, while 28 per cent of the *G. argyrea* lines were susceptible. Genetic differences mirrored the phenotype differences. The majority of *G. argyrea* plants contained only one resistance allele, while most *G. canescens* plants had two or even three separate resistance genes. At the population level, each *G. canescens* population contained ten or more separate resistance alleles, while the *G. argyrea* population most likely contained only two distinct resistance alleles.

The small number of resistance alleles in *G. argyrea* was unexpected since the species inhabits an area thought to be more conducive to *P. pachyrhizi* than either *G. canescens* population. The lack of genes which confer outright resistance may reflect a greater dependence on forms of resistance which simply delay pathogen development. The latent period, which is an important component of pathogen development, was between one and three days longer on *G. argyrea* than on *G. canescens* (Jarosz and Burdon, unpublished). Delaying pathogen development has been postulated to be

an important component of the resistance arsenal of wild hosts, however, only one study has documented its existence within a natural host-pathogen system (Segal *et al.*, 1987). To a large extent this reflects the difficulty in evaluating this form of resistance and not its actual occurrence.

Mating system and response to pathogen pressure

Parker (1988b) reported an extreme case of linkage disequilibrium in *A. bracteata* where resistance to *S. decipiens* was associated with a specific multi-locus isozyme biotype. He concluded that this was the result of the strongly inbred nature of *A. bracteata*, and implied that selection favouring resistance would influence the evolution of neutral and near-neutral characters. However, one other consequence of linkage disequilibrium due to inbreeding is that the fate of a resistance gene is tied to the rest of the genome, and complex selective forces operate on individual resistance genes. Therefore, inbred species may accumulate resistance genes with time; as new favoured resistance genes increase in the population many of the pre-existing resistance genes, which may be largely ineffective against the pathogen races currently attacking the population, may also increase due to hitchhiking. This may explain the common occurrence of individuals with multiple resistance genes in studies which have examined highly inbred species (Dinoor, 1977; Burdon, 1987b; Harry and Clarke, 1987).

In species with some degree of outbreeding, linkage disequilibrium will be reduced and pathogen pressure will act on individual genes. Under these conditions only effective resistance genes will increase in the population, and ineffective resistance genes may actually decrease in frequency if there is a cost associated with their presence in the genome. This may help to explain the pattern in *G. argyrea* where outcrossing in chasmogamous flowers has been estimated to be 38 per cent (Brown *et al.*, 1986). Multilocus isozyme phenotypes were known for 39 of the lines used in this study (A. H. D. Brown, unpublished). The frequency of the resistance allele in this sample of 39 plants was 0.564. Four isozyme loci, AAT 1A, AAT 2, Gpi 2A, Gpi 2B and Mdh 3 (designations of Brown *et al.*, 1986, and Brown unpublished) were highly polymorphic, with the frequency of the most common allele at each locus being 0.551, 0.566, 0.769, 0.782 and 0.808, respectively. Unlike *A. bracteata* where resistance was associated with a single multilocus genotype, in

G. argyrea there were fourteen separate multilocus isozyme genotypes among the fifteen plants homozygous for resistance; fourteen genotypes among the fourteen plants heterozygous for resistance, and eight genotypes among the ten plants that were susceptible to all nine races. The small number of resistance genes in *G. argyrea* may simply reflect selection which has favoured one highly effective resistance gene which has been able to largely supplant other race specific resistance genes within this population.

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