Genetics

Quantitatively Inherited Reactions of Alfalfa to *Peronospora trifoliorum*

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ABSTRACT

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Infection-type (IT) data were obtained by inoculating plant populations derived from all possible self- and cross-pollinations, and some F_2 populations, of six diploid clones of *Medicago sativa* with three monoconidial isolates (I-5, I-7, and I-8) of *Peronospora trifoliorum*. Host effect on intensity of conidial production appeared to be inherited quantitatively. Analyses of variance indicated that primarily additive effects were involved in response to isolate I-8, while additive and nonadditive effects were involved in response to isolates I-5 and I-7. Comparisons of variances and covariances in IT data indicated that genes conditioning a reduced IT usually were dominant. In some cases, however, genes that conditioned a higher IT were expressed although genes that conditioned a reduced IT were present, which suggested epistasis.

Additional key words: diallel analysis, downy mildew, genetics of resistance.

Previous reports have shown that resistance in alfalfa (*Medicago sativa* L.) to *Peronospora trifoliorum* d By. was conditioned by recessive genes (11), dominant genes (20), or one incompletely dominant gene (16). We reported evidence of isolate-specific host genes, with complete dominance, that resulted in complete inhibition of conidial production in the presence of specific parasite genes (18). We also reported different levels of conidial production on plants that lacked these genes (18).

Significant positive correlation of incidence and mean infection type (IT) of plants that supported conidial production in F_2 populations, suggested that additive gene action leading to increased IT was involved (18).

The purpose of the present study was to investigate the occurrence, genetic behavior, and isolate-specificity of these additive effects in six diploid alfalfa clones.

MATERIALS AND METHODS

Monoconidial *P. trifoliorum* isolates I-5 and I-7, from alfalfa plants grown in Kansas, and I-8 from an alfalfa plant grown in California, were used. They were derived and maintained as described previously (18).

Six diploid plants were used. Plants P1–P5 were grown from diploid seed lots PI172984, PI206286, PI172983, PI172989, and PI172989, respectively, of *M. sativa* supplied by W. H. Skrdla, U.S. Department of Agriculture, North Central Regional Plant Introduction Station, Ames, IA. Diploid plant P6, with ancestry about 50% *M. falcata*, was grown from seed lot Wis 72-23 supplied by E. T. Bingham, University of Wisconsin, Madison.

Plants P1–P5 were selected 1 wk after inoculation at the cotyledonary stage for their reaction to isolate I-5. Cotyledons of plant P1 were densely covered with conidia, whereas those of plants P2–P5 were symptomless. Plant P5 produced variegated flowers and sickle-shaped seed pods, characteristics that indicate some *M. falcata* ancestry. Plant P6 was selected because it was free of symptoms 1 wk after inoculation with a mixture of conidia of isolates I-5, I-7, and I-8.

These plants were also selected for their relatively high levels of self-fertility. They were cloned by shoot cuttings and maintained in greenhouses or growth chambers. Ploidy level was confirmed with chromosome counts on root meristem cells.

Seeds from all possible crosses and selfs of these six clones were produced by hand pollination in a greenhouse. Flowers used as female were first emasculated with ethanol (22).

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Procedures from the standard test to characterize downy mildew resistance in alfalfa (21) were used. Seeds were scarified with a razor blade and planted about 8 mm deep in autoclaved mason's sand in aluminum bread pans. Seedlings were inoculated 5 days later by spraying them with a conidial suspension containing 10^5 conidia per milliliter of water. The plants were enclosed in plastic boxes covered with aluminum foil, and were maintained at 20 C for 24 hr. The covers were removed and continuous 98.2 $\mu E \cdot sec^{-1} \cdot m^{-2}$ cool-white fluorescent light was provided. Six days later, the pans of plants were again enclosed in the aluminum foil-covered plastic boxes, to provide the darkness and near-100% relative humidity needed for conidial production (6). Intensity of conidial production on the cotyledons was evaluated 15 hr later under ×12 magnification. The ITs were described on a zero-to-five scale in which zero = no conidial production and five = copious conidial production.

The numbers of conidia per plant that each IT represented were estimated. For IT 1, which represented plants with one to five conidiophores, the mean number of conidia produced per conidiophore was obtained by direct microscopic observation. For ITs 2 to 5, five plants from each category were placed in separate small test tubes containing 1 ml of water. The tubes were shaken vigorously to dislodge the conidia, which were counted with a hemacytometer.

All 36 populations of the diallel were not grown simultaneously. Mean ITs of each population/isolate combination were used to construct a six by six diallel data table for each isolate.

Reciprocal F_1 populations were tested for significant differences with a chi-square test of homogeneity (19) on the categorical data, as were infection-type distributions produced on replicate host plant populations. Each population/isolate combination was replicated at least once.

Sums of squares for crosses were partitioned into general and specific combining ability components by using Griffing's method 3, model I (8). Because all statistics were from one diallel data table, the mean square for reciprocal effect was used as an estimate of error variance as recommended (9,23).

To examine the dominance and/or epistatic relationships of the parents, a V_r, W_r graph (3) was constructed from the data for each isolate. V_r symbolizes the variance in an array of the mean measurements of a parent's S_1 (produced by selfing) and F_1 progenies. W_r symbolizes the covariance between that array and an array of the S_1 means of all parents included in the diallel. On a plot of W_r versus V_r, parents whose S_1 and F_1 progeny are least variable will be represented by data points closest to the origin. Assuming equal effects of genes at all loci and no nonallelic interaction, those parents will have the highest proportion of dominant alleles of the parents in the diallel (3). Nonallelic interaction (i.e., complementary or epistatic interaction) will disturb the graph in

TABLE 1. Infection-type data obtained by inoculating S_1 populations of six diploid alfalfa plants with three monoconidial isolates of *Peronospora trifoliorum*

Parent plant	Isolate	S_1 plants (no.) classified[x] as: Zero	Nonzero	Suggested ratio	*P* value[y]	Mean S_1 infection type[z]
P1	I-5	0	82	0:1	···	4.0 a
	I-7	0	76	0:1	···	4.0 a
	I-8	0	117	0:1	···	4.0 a
P2	I-5	3	69	1:15	0.5–0.7	2.6 ab
	I-7	6	166	1:15	0.1–0.3	2.6 a
	I-8	1	82	1:63	0.7–0.9	2.8 b
P3	I-5	105	0	1:0	···	0.0 a
	I-7	29	90	1:3	0.8–0.9	1.8 b
	I-8	32	43	7:9	0.9–1.0	1.1 c
P4	I-5	42	5	15:1	0.2–0.4	0.3 a
	I-7	43	2	15:1	0.8–0.9	0.1 a
	I-8	87	10	15:1	0.1–0.3	0.2 a
P5	I-5	107	2	63:1	0.9–1.0	0.02 a
	I-7	368	2	255:1	0.9–1.0	0.02 a
	I-8	44	12	3:1	0.5–0.7	0.4 b
P6	I-5	77	2	15:1	0.2–0.4	0.05 ab
	I-7	128	3	63:1	0.7–0.9	0.05 a
	I-8	113	7	15:1	0.8–0.9	0.1 a

[x]Zero = no conidia produced, nonzero = one or more conidiophores produced.
[y]Determined with a chi-square test with a continuity correction.
[z]Means followed by the same letter are of infection-type distributions that were not significantly different according to a chi-square test of homogeneity ($P = 0.05$). Comparisons were made only among isolates within S_1 populations.

TABLE 2. Infection-type data obtained by inoculating F_1 populations of six diploid alfalfa plants with three isolates of *Peronospora trifoliorum*

Cross	Isolate	F_1 plants (no.) classified[w] as: Zero	Nonzero	Expected ratio[x]	*P* value[y]	Mean F_1 infection type[z]
P1 × P2	I-5	0	170	0:1	···	3.08 a
	I-7	21	371	0:1	···	3.74 b
	I-8	3	85	0:1	···	3.07 c
P1 × P3	I-5	63	26	1:0	···	0.64 a
	I-7	0	88	0:1	···	2.96 b
	I-8	0	78	1:1	<0.001	2.66 c
P1 × P4	I-5	66	99	3:1	<0.001	1.64 a
	I-7	103	53	3:1	<0.050	0.89 b
	I-8	87	92	3:1	<0.001	1.70 c
P1 × P5	I-5	22	35	7:1	<0.001	1.32 ab
	I-7	59	66	15:1	<0.001	0.95 a
	I-8	41	44	1:1	0.50–0.70	1.46 b
P1 × P6	I-5	127	28	3:1	0.05–0.10	0.52 a
	I-7	130	14	7:1	0.20–0.40	0.47 ab
	I-8	78	36	3:1	0.10–0.30	0.96 b
P2 × P3	I-5	73	21	1:0	···	0.48 a
	I-7	6	80	0:1	···	2.73 b
	I-8	0	131	0:1	···	3.80 c
P2 × P4	I-5	42	47	13:3	<0.001	1.57 a
	I-7	51	42	13:3	<0.001	1.10 b
	I-8	58	33	25:7	<0.001	1.20 c
P2 × P5	I-5	46	24	7:1	<0.001	1.31 a
	I-7	115	37	7:1	<0.001	0.43 b
	I-8	78	69	1:1	0.50–0.70	1.35 c
P2 × P6	I-5	74	15	3:1	0.10–0.30	1.04 a
	I-7	74	11	7:1	0.50–0.70	0.40 b
	I-8	54	29	3:1	0.01–0.05	1.00 a
P3 × P4	I-5	105	28	1:0	···	0.39 a
	I-7	99	36	3:1	0.50–0.70	0.68 b
	I-8	69	76	3:1	<0.001	1.67 c
P3 × P5	I-5	127	6	1:0	···	0.081 a
	I-7	61	13	15:1	<0.001	0.44 b
	I-8	40	35	1:1	0.50–0.70	1.02 b
P3 × P6	I-5	85	4	1:0	···	0.10 a
	I-7	71	16	7:1	0.05–0.10	0.56 b
	I-8	49	42	7:1	<0.001	1.41 c
P4 × P5	I-5	74	10	31:1	<0.001	0.23 ab
	I-7	79	6	63:1	<0.001	0.13 a
	I-8	72	12	7:1	0.50–0.70	0.33 b
P4 × P6	I-5	151	7	15:1	0.20–0.40	0.26 a
	I-7	160	11	31:1	<0.025	0.17 a
	I-8	130	30	15:1	<0.001	0.51 b
P5 × P6	I-5	157	8	31:1	0.20–0.40	0.097 a
	I-7	297	5	127:1	0.10–0.30	0.030 b
	I-8	135	15	7:1	0.30–0.50	0.23 c

[w]Zero = no conidia produced, nonzero = one or more conidiophores produced.
[x]Assuming genes that condition a zero infection type would be expressed.
[y]Determined with chi-square tests with a continuity correction.
[z]Means followed by the same letter were of infection-type distributions which were not significantly different according to a chi-square test on the categorical data ($P = 0.05$). Comparisons were made only among isolates within F_1 populations.

characteristic ways (2,3) that can be used to gain additional information about the parents.

A diallel data table of a six-parent cross can be partitioned into six five-parent tables, 15 four-parent tables, and 20 three-parent tables. The V_r, W_r relationships in each of these 42 tables of data, for each of the three isolates, were examined. An estimate of the mean direction of dominance was determined from the sign of the difference found by subtracting the mean S_1 IT from the mean F_1 IT within each data table. This estimate is biased by nonadditive effects.

RESULTS

The numbers of conidia produced per plant in each infection type were ($\times 10^{-3}$) IT 1 = 0.027 to 0.135, IT 2 = 1.2 ± 2.3, IT 3 = 29 ± 14, IT 4 = 80 ± 11, IT 5 = 185 ± 34.

Reciprocal crosses among all clones indicated no maternal cytoplasmic influence.

Mean infection types of S_1 and F_1 progenies varied over a wide range (Tables 1 and 2, respectively).

F_1 plants of clones P1 and P2 produced F_2 populations in which the incidence of nonzero ITs and the mean of the nonzero ITs were positively correlated ($r = 0.76$, $P > 0.99$, Table 3).

The selfs versus crosses, and the general and specific combining ability components of variance were significant for isolates I-5 and I-7 (Table 4). Only general combining ability was significant in the analysis of isolate I-8 data (Table 4).

The V_r, W_r plot from the analysis of the mean ITs produced with the six clones and isolate I-5 indicated that clones P3–P6 had mostly dominant alleles, clone P1 had few or no dominant alleles, and clone P2 behaved as an intermediate (Fig. 1). The mean direction of dominance was negative.

Analysis of mean ITs produced with progeny of the six clones and isolate I-7 indicated that clones P4, P5, and P6 had mostly dominant alleles, clones P1 and P2 had mostly recessive alleles, and clone P3 behaved as an intermediate (Fig. 1). The mean direction of dominance was negative. Similar results were found from the analyses of 39 of the 41 data tables including less than six parents.

Analysis of mean ITs of progeny of clones P1, P2, and P3 with isolate I-7 indicated that clone P1 had more dominant alleles than clones P2 or P3 (Fig. 2A), and that the mean direction of dominance was positive. The slope of the regression line on the V_r, W_r plot was significantly less than unity (Fig. 2A). The mean IT of F_1 plants of clones P2 and P3 (Table 2) was greater than the mean IT of S_1 plants of either parent (Table 1).

Analysis of the mean IT data table of the six clones with isolate I-8 indicated an order of dominance similar to that with isolate I-7,

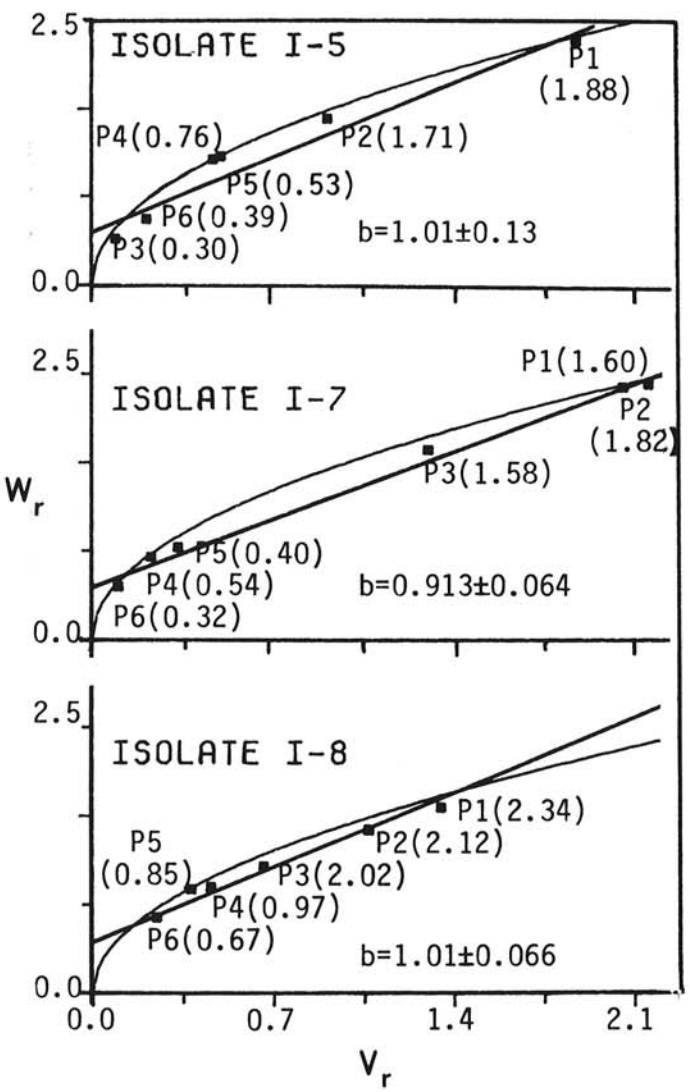

Fig. 1. V_r, W_r plots of six-parent diallel crosses of diploid alfalfa interacting with three isolates of *Peronospora trifoliorum*. Numbers in parentheses are the array means of the designated parents.

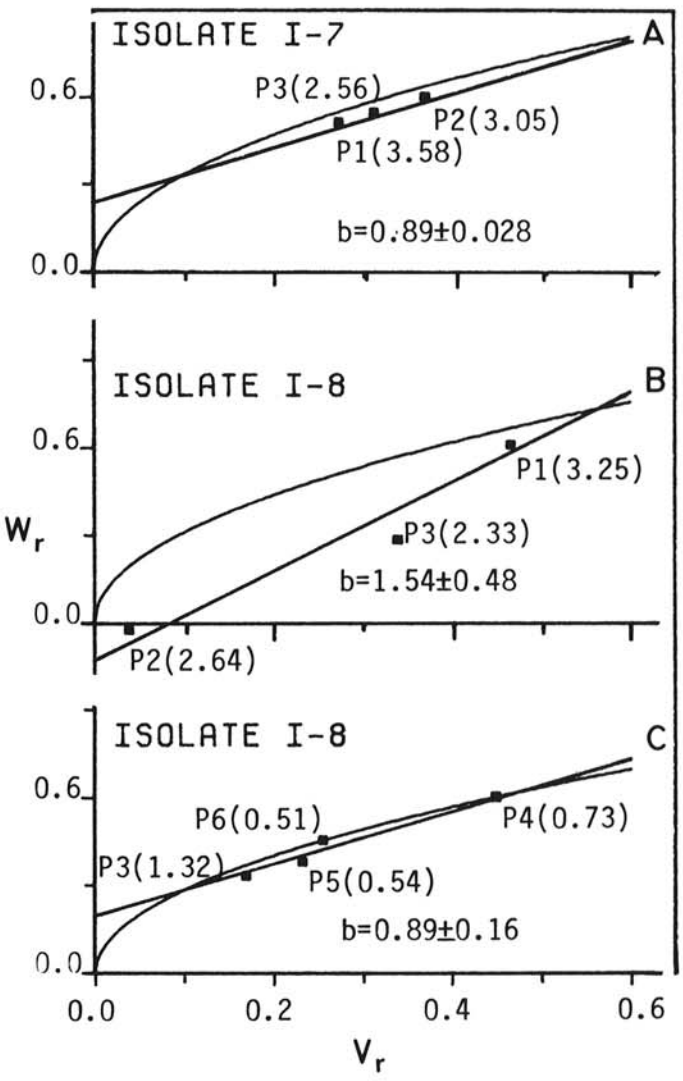

Fig. 2. V_r, W_r plots of three- or four-parent diploid alfalfa diallel crosses interacting with different isolates of *Peronospora trifoliorum*: **A**, clones P1, P2, and P3 interacting with isolate I-7 and **B**, with isolate I-8; **C**, clones P3, P4, P5, and P6 interacting with isolate I-8. Numbers in parentheses are the array means of the designated parents.

except that clones P4 and P5 were reversed (Fig. 1). The mean direction of dominance was negative.

Analysis of the mean IT data table of progeny of clones P1, P2, and P3 with isolate I-8 indicated that the mean direction of dominance was positive, and that clone P2 had more dominant alleles than did clone P1 or P3 (Fig. 2B). The F_1 population of clones P2 and P3 (Table 2) had a higher mean IT than the S_1 population of either parent (Table 1).

Analyses of all possible three- and four- parent tables of mean IT data that included clones P3–P6 (except for P4, P5, and P6 data table) with isolate I-8 indicated that clone P3 had the most dominant alleles (Fig. 2C), and that the mean direction of dominance was positive.

Analysis of mean IT data tables of clones P4, P5, and P6 with each of the isolates, indicated a positive mean direction of dominance.

DISCUSSION

In previous work (18), we found evidence of completely dominant genes that conditioned a zero IT in clones P5 and P6. From F_1 data, it was apparent that similar genes, predicted from S_1 data (Table 1), were not present in clones P1-P4 (Table 2). Also, except for one gene in clone P5 that conditioned a zero IT with isolate I-8, those genes in clones P5 and P6 that conditioned zero ITs were not always expressed when in combination with the genes of the other clones (Table 2). Apparently, these genes functioned qualitatively in one genotype, but were either nonfunctional or functioned quantitatively in another genotype. The latter possibility has been proposed for some other pathosystems (1,14).

The correlation of incidence and mean of nonzero ITs produced in F_2 populations of clones P1 and P2 with isolate I-7 (Table 3), indicated that additive gene action leading to increased IT was involved. This was found also in F_2 populations of clones P5 and P6 with isolate I-8 (18).

The statistical significance of general combining ability with each of the isolates (Table 4) also indicated that additive effects were involved.

The V_r, W_r graphs from analyses of the six parent crosses, with each of the isolates showed that the parents with the lowest array means were also the least variable (Fig. 1). This indicated that most genes that conditioned a reduced IT were either partially or completely dominant. This indication must be interpreted with caution, however. Infection types are determined by the genotypes of the host and the parasite. In the case of a gene-for-gene relationship (5,12), the IT due to a particular allelic condition of one corresponding gene pair (one gene in the host and its corresponding gene in the parasite) is expressed even though other corresponding gene pairs are present (12). That IT is said to be "epistatic" in the category IV interaction (13). In the biotrophic systems that were investigated, the lowest IT coded for in a particular interaction was "epistatic" to all others (12).

Although we have shown that the zero IT was not "epistatic" to all other possible ITs in all cases (Table 2), reduced ITs may have been "epistatic" to some higher ITs. Therefore, the positions of the parents on the V_r, W_r graphs should be interpreted as a reflection of dominance and/or category IV "epistasis". The selfs versus crosses component of variation is composed entirely of nonadditive effects (7). Thus, the significance of specific combining ability and the selfs versus crosses component (Table 4) indicated that some nonadditive effects were involved in response to isolate I-5 or I-7. This suggested that some dominance or "epistasis" was present. The V_r, W_r relationships, and the mean direction of dominance, in the partitioned data sets were examined to determine which parents were involved in the nonadditive combinations, and the results of the nonadditive effects.

The altered order of dominance and the deviation from unity of the regression line slope on the V_r, W_r graph from the analysis of IT data from clones P1, P2, and P3 with isolate I-7 (Fig. 2A) suggested complementary gene action (2,3). Since the mean IT of F_1 plants of clones P2 and P3 (Table 2) was greater than the mean IT of S_1 plants of either parent (Table 1), apparently the complementary effect involved genes of those two parents and led to increased IT. This effect was isolate-specific, having occurred with isolates I-7 (Fig. 2A) and possibly I-8 (Fig. 2B) but not with isolate I-5.

Analysis of mean IT data of clones P3–P6 with isolate I-8, showed an altered order of dominance (Fig. 2C), relative to the six-parent cross (Fig. 1) and a positive mean direction of dominance. This indicated that the genes of clone P3, which conditioned increased IT with isolate I-8, were expressed in most of the F_1 progeny (Table 2). The genes' effects were either additive with or "epistatic" to the effects of genes of clones P4-P6 that conditioned reduced IT with isolate I-8 (Table 1). Since there was no indication of nonadditive effects (Table 4), very likely the genes of clones P3–P6 additively conditioned increased IT. This effect was specific to isolate I-8.

The mean IT of F_1 plants of clones P4-P6, although small, (Table 2) was greater than the mean IT of the S_1 generation of those clones (Table 1), with each of the isolates. This further indicated that genes from each parent could act additively to give a higher IT with each of the isolates.

The order of increasing mean IT of replicate plant populations with different isolates was not consistent (Tables 1 and 2). For example, the lowest mean IT in the F_1 population of clones P1 and P4 was associated with isolate I-7, but not in the F_1 population of clones P1 and P3 (Table 2). Also, the distributions of plants in the categories 0–5 were often significantly different among isolates (Tables 1 and 2). Both of these indicate isolate-specific host-parasite interactions.

Isolate-specific host-parasite interactions, involving many genes with small additive effects, have been compared to nonspecific host

TABLE 3. Infection-type data obtained by inoculating F_1 and F_2 plants of diploid alfalfa clones P1 and P2 with isolate I-7 of *Peronospora trifoliorum*

F_1 plant	Infection type[a]	F_2 plants with conidia (%)[b]	Mean infection type of F_2 plants with conidia
1	0	68.8	1.9
2	0	70.2	2.1
3	0	81.4	2.8
4	0	82.6	2.4
5	0	85.7	3.5
6	1	56.8	2.2
7	1	51.5	2.2
8	1	47.2	2.1
9	2	55.2	2.8
10	2	78.6	2.5
11	2	80.0	3.2
12	2	87.8	3.3
13	3	64.3	2.6
14	3	75.7	2.9
15	3	93.2	2.6
16	4	100.0	3.7
17	5	93.6	3.9
18	5	100.0	4.0

[a]Zero = no conidia produced, five = copious conidial production.
[b]Plants which supported conidial production.

TABLE 4. Partitioning of the variance from a six-parent diallel of diploid alfalfa inoculated with conidia of three isolates of *Peronospora trifoliorum*

		Isolate		
Source	df	I-5 MS	I-7 MS	I-8 MS
Populations	35	0.981**[a]	1.49**	1.06**
Selfs	5	2.97**	2.81**	2.57**
Selfs vs. crosses	1	0.423**	0.658**	0.0058
Crosses	29	0.656**	1.29**	0.830**
General[b]	5	3.10**	5.94**	4.21**
Specific[b]	9	0.327**	0.818**	0.186
Error[c]	15	0.039	0.031	0.090

[a]**Two asterisks indicate significance at $P = 0.01$.
[b]General and specific combining abilities.
[c]Error mean square is from the difference among reciprocals.

plant responses in theoretical models by Parlevliet and Zadoks (15). They concluded that isolate-specific interactions would be evolutionarily favorable to nonspecific interactions. Their analysis has been questioned by Person et al (17) who, referring to another paper dealing with theoretical models (4), stated that nonspecific interactions would be quite stable over the course of evolution.

Our data also show that some gene effects were general. For example, clones P4, P5, and P6 were associated with low mean ITs and clones P1 and P2 were associated with high mean ITs with each of the isolates (Fig. 1). This may have been because the effects truly were nonspecific (which cannot be proven [10]), or because there were genes in common among the host or parasite genotypes.

We conclude that two types of genetic behavior exist within the alfalfa downy mildew system. There are host genes that individually result in complete inhibition of conidial production (18), and there are genes with additive effects leading to increased IT. The observed IT is the result of the interaction of these two types of genetic effects. We suggest that both types of genetic behavior depend on specific genes in the host and specific genes in the parasite.

LITERATURE CITED

1. Ahn, S. W., and Ou, S. H. 1982. Quantitative resistance of rice to blast disease. Phytopathology 72:279-282.
2. Coughtrey, A. and Mather, K. 1970. Interaction and gene association and dispersion in diallel crosses where gene frequencies are unequal. Heredity 25:79-88.
3. Dickinson, A. G., and Jinks, J. L. 1956. A generalized analysis of diallel crosses. Genetics 41:65-78.
4. Fleming, R. A., and Person, C. O. 1982. Consequences of polygenic determination of resistance and aggressiveness in nonspecific host:parasite relationships. Can. J. Plant Pathol. 4:89-96.
5. Flor, H. H. 1956. The complementary genic systems in flax and flax rust. Adv. Genet. 8:29-54.
6. Fried, P. M., and Stuteville, D. L. 1977. *Peronospora trifoliorum* sporangium development and effects of humidity and discharge on germination. Phytopathology 67:890-894.
7. Gardner, C. O., and Eberhart, S. A. 1966. Analysis and interpretation of the variety cross diallel and related populations. Biometrics 22:439-452.
8. Griffing, B. 1956. Concept of general and specific combining ability in relation to diallel crossing systems. Aust. J. Biol. Sci. 9:463-493.
9. Hayman, B. I. 1954. The analysis of variance of diallel tables. Biometrics 10:235-244.
10. Johnson, R. 1979. The concept of durable resistance. Phytopathology 69:198-199.
11. Jones, F. R., and Torrie, J. H. 1946. Systemic infection of downy mildew in soybean and alfalfa. Phytopathology 36:1057-1059.
12. Loegering, W. Q. 1978. Current concepts in interorganismal genetics. Annu. Rev. Phytopathol. 16:309-320.
13. Loegering, W. Q., and Powers, H. R. 1962. Inheritance of pathogenicity in a cross of physiological races 111 and 36 of *Puccinia graminis* f. sp. *tritici*. Phytopathology 52:547-554.
14. Nelson, R. R., McKenzie, D. R., and Scheifele, G. L. 1970. Interaction of genes for pathogenicity and virulence in *Trichometasphaeria turcica* with different numbers of genes for vertical resistance in *Zea mays*. Phytopathology 60:1250-1254.
15. Parlevliet, J. E., and Zadoks, J. C. 1977. The integrated concept of disease resistance; A new view including horizontal and vertical resistance in plants. Euphytica 26:5-21.
16. Pedersen, M. W., and Barnes, D. K. 1965. Inheritance of downy mildew resistance in alfalfa. Crop Sci. 5:4-5.
17. Person, C. O., Fleming, R., Cargeeg, L., and Christ, B. 1983. Present knowledge and theories concerning durable resistance. Pages 27-40 in: Durable Resistance in Crops. F. Lamberti, J. M. Waller, and N. A. Van der Graaf, eds. Plenum Press, New York.
18. Skinner, D. Z., and Stuteville, D. L. 1985. Genetics of host-parasite interactions between alfalfa and *Peronospora trifoliorum*. Phytopathology 75:119-121.
19. Snedecor, G. W., and Cochran, W. G. 1980. Statistical Methods. Seventh ed. Iowa State University Press, Ames. 507 pp.
20. Stanford, E. H. 1952. Transfer of disease resistance to standard varieties. Proc. Sixth Int. Grassland Cong. 6:1585-1590.
21. Stuteville, D. L. 1984. Downy mildew resistance; Laboratory methods. Pages 23-24 in: Standard tests to characterize pest resistance in alfalfa cultivars. U. S. Dept. Agric. Misc. Pub. 1434.
22. Tysdal, H. M., and Garl, J. R. 1940. A new method for alfalfa emasculation. J. Am. Soc. Agron. 32:405-407.
23. Yates, F. 1947. Analysis of data from all possible reciprocal crosses between a set of parental lines. Heredity 1:287-301.