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Stability Analyses for Estimating Relative Durability of Quantitative Resistance*

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Summary. Experiments in which a series of host cultivars are inoculated in all combinations with a series of pathogen isolates have been used to detect specificity in the host resistance. A theoretical model of polygenic resistance involving both general and specific interactions with pathogen virulence was developed to test the abilities of statistical analyses to discriminate between host genotypes with different levels of general and specific resistance. Estimates of levels of specific resistance could be obtained in regressions of disease severity scores for each host cultivar × pathogen isolate combination vs. the virulence index of each isolate. If the virulence index was based on the mean disease severity induced by the isolate over all host cultivars, the slopes of the regression lines were correlated with the levels of specific resistance in host cultivars. If the virulence index was based on the disease severity induced by the isolate on a host cultivar with a minimum of specific resistance, the mean squares for deviations from the regression were correlated with the levels of specific resistance in host cultivars. A method was developed to consistently choose host cultivars with minimum specific resistance. The two regression analyses gave estimates of specificity in randomly generated, model genotypes of approximately equal accuracy, although the second method appeared to be more accurate when the numbers of loci controlling resistance and virulence were small. The best estimates of numbers of genes for specific resistance were obtained by calculating a rating based on mean disease severity, the mean square for deviation from the regression on the virulence index based on disease severity on the cultivar with minimum specific resistance and the slope of the regression on the virulence index based on the mean disease severity. The best estimates of proportions of resistance genes that were specific were obtained by calculating a rating based on the above deviation mean square and slope alone.

Key words: Horizontal resistance – General resistance – Vertical resistance – Specific resistance – Stability analysis

Introduction

Horizontal resistance as defined by Vanderplank (1968) is characterized by the absence of an association between variation in host resistance and variation in pathogen virulence. Vanderplank's definition implies that genes for horizontal resistance do not interact specifically with genes for virulence (or aggressiveness). According to Vanderplank, horizontal resistance can be identified from tests in which a number of host genotypes are inoculated in all combinations with a number of pathogen genotypes. If analysis of variance shows no statistically significant contribution of host genotype × pathogen genotype interaction to the variation in disease severity among host-pathogen combinations, the resistance can be said to be horizontal.

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In the absence of data from tests of host and pathogen genotypes, quantitative or polygenic resistance is often assumed to be horizontal (Vanderplank 1963; Robinson 1976). True horizontal resistance is necessarily stable in relation to genetic variation within the pathogen population. Conversely, vertical resistance is often unstable and subject to being overcome by pathogenic races with matching genes for virulence.

The validity of assumptions of stability of polygenic resistance began to be questioned when statistically significant host cultivar × pathogen isolate interactions were found in experiments involving polygenic resistance in several host-pathogen systems (Caten 1974; Clifford and Clothier 1974; Milus and Line 1980; Parlevliet 1976). Currently, the durability of a cultivar's resistance can be adequately tested only by growing the cultivar over a wide area in commercial production; resistance trials in small plots, even repeated at many sites, have proved inadequate (Johnson 1978). Consequently, a reliable measure of durability requiring less exhaustive tests would be of great value.

Leonard and Moll (1981) suggested that an approach similar to that used by Eberhart and Russell (1966) to estimate stability parameters for yield of maize lines in a variety of environments could provide information about the durability of resistance. In their adaptation of Eberhart and Russell's method, Leonard and Moll analyzed quantitative disease data from an experiment in which a series of host cultivars were inoculated in all combinations with a series of pathogen isolates. They performed a regression analysis for each cultivar, using as the independent variable a virulence index based on the mean disease severity induced by each isolate over all the host cultivars in the test. These analyses were combined into an overall analysis of variance which indicated how much of the variance for the disease interaction was accounted for by linear regression. The slope of the regression for each cultivar was assumed to indicate its sensitivity to increased general virulence, and the deviations from regression were assumed to indicate how much of the cultivar's resistance was specific with respect to the pathogen isolates in the test. This type of analysis has been used to characterize resistance to northern leaf spot of maize (Hamid et al. 1982).

The purpose of this investigation was to test the ability of the analysis proposed by Leonard and Moll to discriminate between general (horizontal) and specific (vertical) resistance in a hypothetical host-pathogen system in which the host and pathogen genotypes were randomly selected and the proportions of loci for general and specific resistance and virulence were predetermined.

The Model

The model used in this study differs from Parlevliet and Zadok's (1977) models of host-pathogen interactions involving polygenic resistance and virulence in that it combines both specific and general resistance and virulence in a single model. Genes for general susceptibility (alleles of genes for general resistance) in the host and genes for general virulence in the pathogen are assumed to interact additively to determine disease severity in each cultivar × isolate combination (Table 1). Genes for specific resistance and virulence are assumed to interact in a gene-for-gene relationship similar to that described by Flor (1971), except that each matched pair of resistance × virulence genes contributes only an additive increment to the total disease severity (Table 2). The expression of each gene for specific resistance depends on the absence of the corresponding gene for specific virulence in the pathogen. Correspondingly, a gene for specific virulence does not contribute to increased disease severity unless the corresponding gene for specific resistance is present in the host.

When host cultivars and pathogen isolates contain genes for both general and specific resistance and virulence, respectively, the disease severity is calculated as follows:

Disease severity = no. rg + no. Vg + no. rs + no. Vs-Rs matches, where *Rg* and *Rs* are general and specific resistance alleles, *rg* and *rs* are corresponding alleles for

Table 1. Severity of disease in combinations of hypothetical host and pathogen genotypes in a model with resistance and virulence conditioned by additive genes with general effects

Pathogen genotypes[a]	Disease severity on host genotypes[a]			
	$rg_1\ rg_2$	$rg_1\ Rg_2$	$Rg_1\ rg_2$	$Rg_1\ Rg_2$
$Vg_1\ Vg_2$	4[b]	3	3	2
$vg_1\ Vg_2$	3	2	2	1
$Vg_1\ vg_2$	3	2	2	1
$vg_1\ vg_2$	2	1	1	0

[a] Rg_1 and Rg_2 are genes for general resistance, Vg_1 and Vg_2 are genes for general virulence, and disease severity is determined as the sum of the number of genes for susceptibility and general virulence
[b] 0 = least severe, 4 = most severe

Table 2. Severity of disease in combinations of hypothetical host and pathogen genotypes in model with gene-for-gene specifictiy between specific resistance and virulence genes with additive effects

Pathogen genotypes[a]	Disease severity on host genotypes[a]			
	$rs_1\ rs_2$	$rs_1\ Rs_2$	$Rs_1\ rs_2$	$Rs_1\ Rs_2$
$Vs_1\ Vs_2$	2[b]	2	2	2
$vs_1\ Vs_2$	2	2	1	1
$Vs_1\ vs_2$	2	1	2	1
$vs_1\ vs_2$	2	1	1	0

[a] Rs_1 and Rs_2 are genes for specific resistance, Vs_1 and Vs_2 are genes for specific virulence, and disease severity is determined as the sum of the number of genes for susceptibility and the number of matches of genes for specific virulence with the corresponding genes for specific resistance
[b] 0 = least severe, 2 = most severe

Table 3. Severity of disease in combinations of hypothetical host and pathogen genotyes in a model with genes for both general and specific resistance and virulence

Pathogen genotypes[a]	Disease severity on host genotypes[a]								
	$rgrs_1\ rs_2$	$rgRs_1\ rs_2$	$rgrs_1\ Rs_2$	$rgRs_1\ Rs_2$	$Rgrs_1\ rs_2$	$RgRs_1\ rs_2$	$Rgrs_1\ Rs_2$	$RgRs_1\ Rs_2$	Mean
$VgVs_1\ Vs_2$	4	4	4	4	3	3	3	3	3.5
$VgVs_1\ vs_2$	4	4	3	3	3	3	2	2	3.0
$Vgvs_1\ Vs_2$	4	3	4	3	3	2	3	2	3.0
$Vgvs_1\ vs_2$	4	3	3	2	3	2	2	1	2.5
$vgVs_1\ Vs_2$	3	3	3	3	2	2	2	2	2.5
$vgVs_1\ vs_2$	3	3	2	2	2	2	1	1	2.0
$vgvs_1\ Vs_2$	3	2	3	2	2	1	2	1	2.0
$vgvs_1\ vs_2$	3	2	2	1	2	1	1	0	1.5
Mean	3.5	3.0	3.0	2.5	2.5	2.0	2.0	1.5	

[a] Rg is a gene for general resistance, Rs_1 and Rs_2 are genes for specific resistance, Vg is a gene for general virulence, and Vs_1 and Vs_2 are genes for specific virulence that can overcome the effects of Rs_1 and Rs_2, respectively. Disease severity is determined as the sum of the number of genes for susceptibility and general virulence plus the number of matches of genes for specific virulence with the corresponding genes for specific resistance

Table 4. Regression analysis of disease severity in all combinations of hypothetical host and pathogen genotypes in a theoretical model[a] with both general and specific resistance and virulence

Host genotype	Regression of host genotype disease severity vs.			
	Pathogen virulence index from mean disease over all hosts (VIM)[b]		Pathogen virulence index from disease on hosts with $rs_1\ rs_2$ (VIS)[c]	
	Slope	Deviation MS	Slope	Deviation MS
$rgrs_1\ rs_2$	0.67	0.11	1.00	0.00
$rgRs_1\ rs_2$	1.00	0.00	1.00	0.33
$rgrs_1\ Rs_2$	1.00	0.00	1.00	0.33
$rgRs_1\ Rs_2$	1.33	0.11	1.00	0.67
$Rgrs_1\ rs_2$	0.67	0.11	1.00	0.00
$RgRs_1\ rs_2$	1.00	0.00	1.00	0.33
$Rgrs_1\ Rs_2$	1.00	0.00	1.00	0.33
$RgRs_1\ Rs_2$	1.33	0.11	1.00	0.67

[a] Host and pathogen genotypes and disease severities in the model are shown in Table 3

[b] Based on pathogen means shown in Table 3

[c] Based on severity values shown in Table 3 for pathogen genotypes on hosts $rgrs_1\ rs_2$ or $Rgrs_1\ rs_2$

susceptibility, *Vg* and *Vs* are general and specific virulence alleles, and *vg* and *vs* are corresponding alleles for avirulence (Table 3).

In our proposed statistical analysis of disease resistance of cultivars, the severity of disease induced by each pathogen isolate in a cultivar was regressed against a virulence index for the isolate. Two virulence indexes were tested. The first, designated VIM, was calculated as the mean disease severity induced by that isolate over all host cultivars. The second, designated VIS, was based on the disease severity induced by the isolate on a standard susceptible cultivar with no genes for specific resistance. This is similar to the approach used by Singh et al. (1978), who used a susceptibility index based on damage suffered by their most susceptible sorghum cultivar to the sorghum shoot fly in their analysis of stability of resistance to sorghum shoot fly over a range of environments.

When the disease severities for cultivars in our simple model (Table 3) were regressed against VIM values for isolates, the regression cofficients (slopes) for cultivars were directly proportional to the numbers of genes for specific resistance in the cultivars. The mean value for slopes was 1.00. The mean squares for deviations from regression, which were expected to indicate levels of specific resistance, were not correlated with the number of genes for specific resistance in the model cultivars (Table 4).

Regression of disease severity values for cultivars in the model against VIS values for isolates yielded the slope of 1.00 for each cultivar. Thus, the slopes for cultivars were not proportional to levels of specific resistance, but in this case the deviation mean square for each cultivar was directly proportional to its number of genes for specific resistance (Table 4).

In the simple model illustrated in Tables 3 and 4, the presence or absence of genes for general resistance affected the mean disease severity of a cultivar but had no effect on the slope or the deviations from regression. This was true whether the regression was based on VIM or VIS values of the pathogen isolates.

Extension of the Model to Randomly Selected Genotypes

In the simple model of cultivar × isolate interactions considered in the previous section, the disease reactions

of all possible combinations of host and pathogen genotypes were included in the analyses. In real experiments, however, the genotypes would not be known, so it would not be possible to select a single representative cultivar or isolate for each host or pathogen genotype to be included in the test. Some genotypes might be left out and others might be duplicated in the test. If resistance and virulence were controlled by genes at many different loci, it might be impossible to study more than a small fraction of the total number of potential host and pathogen genotypes in any single experiment.

To test the utility of the regression analyses under these more realistic conditions, we developed a computer program to generate random host and pathogen genotypes and to calculate the disease severity for each combination. The program allowed us to specify numerical values for the following factors in each test: the number of loci for general and specific resistance and virulence, the ratios of general resistance to specific resistance and of general virulence to specific virulence, and the mean frequency of alleles for resistance or virulence. All loci that contributed to disease severity were assumed to contribute equal increments to the total disease severity. The genotypes were determined by generating a random number between 0 and 1 for each locus. If the predetermined frequency of resistance genes were set at 0.5, the locus was assigned a gene for resistance if the random number exceeded 0.5.

Disease severities were calculated as described for the simple model in the previous section. In the first set of trials, there were four loci each for general resistance, specific resistance, general virulence, and specific virulence, and all genes were set at a frequency of 0.5 in the population from which the random genotypes were drawn. The number of host genotypes in a set was varied from five to 25, and the number of pathogen genotypes from five to 15.

When the isolate virulence indexes were based on mean isolate performance over all host genotypes (VIM), the slopes of the regression lines for cultivars were significantly ($\propto = 0.05$) correlated with the numbers of genes for specific resistance in five of 15 trials and with the proportion of resistance genes that were specific in four of 15 trials (Tables 5 and 6). In general, the correlations were better when higher numbers of host and pathogen genotypes were included in the tests.

When the isolate virulence indexes in the first set of trials were based on their performance on the cultivar with the least specific resistance (VIS), the mean squares for deviations from regression were significantly ($\propto = 0.05$) correlated with the numbers of genes for specific resistance in 13 of 15 trials and with the proportion of resistance genes that were specific in five of 15 trials. In these analyses, if there was no single cultivar with the lowest number of genes for specific resistance, each cultivar in the test which shared the lowest number was used in turn as the basis for determining VIS.

In the second set of trials the number of host genotypes in each trial was set at 15 and the number of pathogen isolates at eight. There were either 16 or 24 loci for resistance and virulence, and the ratio of general : specific loci was set at 1 : 3, 1 : 1, or 3 : 1. In each trial the number of loci for general resistance was equal to the number for general virulence, and the number of loci for specific resistance was equal to the number for specific virulence.

When isolate virulence indexes in the second set of trials were based on mean isolate performance over all host genotypes (VIM), the slopes of the regression lines for cultivars were significantly ($\propto = 0.05$) correlated with the numbers of genes for specific resistance in only ⅓ of the trials and with the proportion of resistance genes that were specific in only ¼ of the trials (Tables 7 and 8). When virulence indexes were based on performance on the most susceptible cultivar (VIS), the deviation mean squares were significantly ($\propto = 0.05$) correlated with the numbers of genes for specific resistance in only ⅓ of the trials, and with the proportion of resistance genes that were specific in only ⅙ of the trials (Tables 7 and 8).

When virulence indexes of isolates in the preceding analyses were based on host cultivars with the least number of genes for specific resistance, these cultivars were chosen by inspection of the hypothetical genotypes. In real experiments, however, it would be necessary to make the choice from experimental data. In an attempt to simulate such a choice we ranked the hypothetical genotypes in each trial represented in Tables 5–8 in two ways: first, from the highest to the lowest mean disease severity within the set of host genotypes, and second, from the lowest to the highest slopes from regressions of disease severity against VIM. We gave each ranking value equal weight and chose the genotype with the lowest mean values as the one likely to have the least amount of specific resistance in each set. Based on these criteria we were able to correctly identify the host cultivar with the least number of genes for specific resistance in 14 of the 27 sets of hypothetical genotypes.

In order to improve our accuracy in selecting host cultivars with the least amount of specific resistance, we added a third criterion. The basis of the third criterion was that isolates with low levels of specific virulence should provide better than average discrimination among differences in specific resistance of the host cultivars in a set, because more of the genes for specific resistance would be effective against such isolates. We

Table 5. Correlation between stability estimators and the number of genes for specific resistance in randomly generated, hypothetical host genotypes in a model[a] in which disease is controlled by four genes each for general and specific resistance and virulence

No. host genotypes	No. pathogen genotypes	Correlation of no. of genes for specific resistance with:					
		Slope[b]		Deviation mean square[c]		Mean[d]	
		R	P>p	R	P>p	R	P>p
5	5	−0.32	0.60	0.98	0.03	−0.75	0.14
5	5	−0.15	0.82	0.90	0.10	−0.79	0.11
5	5	0.79	0.11	0.00	1.00	−0.73	0.16
8	8	0.48	0.23	0.78	0.04	0.93	0.001
8	8	−0.41	0.31	0.83	0.02	−0.79	0.02
8	8	0.81	0.01	0.79	0.03	−0.30	0.47
15	8	−0.22	0.44	0.60	0.02	−0.76	0.001
15	8	0.43	0.11	0.63	0.02	−0.60	0.02
15	8	0.09	0.74	0.66 − 0.90	0.01 − 0.001	−0.26	0.34
25	8	0.78	0.0001	0.93	0.0001	−0.74	0.0001
25	8	0.74	0.0001	0.93	0.0001	−0.13	0.54
25	8	−0.04	0.83	0.81	0.0001	−0.58	0.003
15	15	0.76	0.001	−0.12 − 0.64	0.70 − 0.01	−0.57	0.03
15	15	0.10	0.74	0.89	0.0001	−0.71	0.003
15	15	0.71	0.003	−0.04 − 0.78	0.88 − 0.0001	−0.45	0.09

[a] Genetic interactions are of the type shown in Table 3
[b] In regressions of disease severity vs. pathogen virulence index based on mean severity induced by the isolate over all hosts
[c] Deviations from regressions of disease severity vs. pathogen virulence index based on severity induced by the isolate on the host with the fewest genes for specific resistance. If more than one host genotype in the set contained the same least number of genes for specific resistance, a separate analysis was run for virulence indexes based on each. Ranges of R and P values are presented for those cases
[d] Mean performance of the host genotype over all pathogen genotypes

Table 6. Correlation between stability estimators and the proportion of resistance genes that are specific in randomly generated, hypothetical host genotypes in a model[a] in which disease is controlled by four genes each for general and specific resistance and virulence

No. host genotypes	No. pathogen genotypes	Correlation of proportion of resistance genes specific with:					
		Slope[b]		Deviation mean square[c]		Mean[d]	
		R	P>p	R	P>p	R	P>p
5	5	−0.31	0.61	0.92	0.08	−0.76	0.13
5	5	−0.40	0.51	0.74	0.26	−0.29	0.64
5	5	0.79	0.11	−1.00	0.0001	0.48	0.41
8	8	0.87	0.005	0.81	0.03	−0.14	0.74
8	8	−0.16	0.70	−0.09	0.85	0.13	0.75
8	8	0.44	0.28	0.67	0.10	−0.76	0.03
15	8	0.15	0.60	0.40 −0.69	0.15 − 0.006	0.62	0.01
15	8	0.40	0.14	0.49	0.08	−0.04	0.90
15	8	−0.06	0.84	0.23	0.42	−0.29	0.29
25	8	0.55	0.005	0.44	0.03	−0.06	0.77
25	8	0.15	0.49	0.71	0.0001	−0.07	0.75
25	8	0.59	0.002	0.67	0.0004	0.37	0.07
15	15	0.79	0.0004	0.006 − 0.60	0.98 − 0.02	−0.24	0.39
15	15	0.10	0.72	0.83	0.0002	−0.63	0.01
15	15	0.19	0.51	0.10 −0.53	0.73 − 0.05	0.54	0.04

[a, b, c, d] See Table 5

Table 7. Correlation between stability estimators and the number of genes for specific resistance in host genotypes in a model[a] with 15 and eight randomly generated, hypothetical host and pathogen genotypes, respectively

No. general genes[e]	No. specific genes[e]	Correlation of no. of genes for specific resistance with:					
		Slope[b]		Deviation mean square[c]		Mean[d]	
		R	P>p	R	P>p	R	P>p
8	8	0.23	0.40	0.08 – 0.10	0.80 – 0.74	– 0.15	0.59
8	8	0.12	0.68	0.05 – 0.14	0.86 – 0.63	– 0.43	0.11
8	8	0.55	0.03	0.62 – 0.76	0.02 – 0.002	– 0.65	0.01
12	12	– 0.32	0.25	– 0.31 – 0.51	0.29 – 0.06	– 0.49	0.06
12	12	– 0.002	0.99	0.56 – 0.63	0.04 – 0.02	– 0.65	0.008
12	12	0.64	0.01	0.39 – 0.63	0.17 – 0.02	– 0.63	0.01
12	4	– 0.06	0.83	0.28 – 0.30	0.33 – 0.29	0.32	0.25
12	4	0.47	0.07	0.79	0.0007	– 0.37	0.17
12	4	0.32	0.24	– 0.22	0.45	– 0.59	0.02
4	12	0.67	0.0001	0.68 – 0.70	0.008 – 0.006	– 0.92	0.0001
4	12	0.56	0.03	– 0.44 – 0.46	0.12 – 0.10	– 0.69	0.005
4	12	– 0.04	0.90	0.33 – 0.29	0.90 – 0.32	– 0.74	0.0002

[a, b, c, d] See Table 5

[e] The numbers of loci for resistance and virulence in the model were equal for both general and specific genes

Table 8. Correlation between stability estimators and the proportion of resistance genes that are specific in host genotypes in a model[a] with 15 and 8 randomly generated, hypothetical host and pathogen genotypes, respectively

No. general genes[e]	No. specific genes[e]	Correlation of proportion of resistance genes specific with:					
		Slope[b]		Deviation mean square[c]		Mean[d]	
		R	P>p	R	P>p	R	P>p
8	8	– 0.07	0.79	– 0.07 – 0.02	0.81 – 0.94	0.40	0.14
8	8	0.12	0.67	– 0.21 – 0.13	0.07 – 0.67	0.34	0.22
8	8	0.34	0.22	– 0.03 – 0.36	0.92 – 0.21	0.11	0.70
12	12	– 0.45	0.09	– 0.48 – 0.11	0.08 – 0.70	0.46	0.08
12	12	0.04	0.88	0.07 – 0.34	0.82 – 0.23	0.35	0.20
12	12	0.51	0.04	0.29 – 0.53	0.32 – 0.05	– 0.10	0.71
12	4	0.64	0.01	0.11 – 0.35	0.70 – 0.23	0.64	0.01
12	4	0.38	0.16	0.73	0.003	0.12	0.68
12	4	0.29	0.30	– 0.35	0.22	– 0.35	0.21
4	12	0.73	0.002	0.67 – 0.72	0.01 – 0.004	– 0.61	0.02
4	12	0.18	0.53	– 0.38 – 0.26	0.18 – 0.38	0.30	0.27
4	12	0.32	0.25	– 0.09 – 0.03	0.77 – 0.92	0.52	0.05

[a, b, c, d] See Table 5

[e] The numbers of loci for resistance and virulence in the model were equal for both general and specific genes

selected isolates with low specific virulence by regressing disease severity for each isolate against a susceptibility index for host cultivars based on their average response over all isolates in the trial. This procedure was analogous to the regression of disease severity on host cultivars against VIM. The isolate with the highest slope in each set was chosen as the one with the expected lowest level of specific virulence. We subtracted the mean disease severity of each cultivar with the selected isolate from its mean disease severity over all isolates. The cultivars in each trial were ranked from the lowest to the greatest differences in the two values. Low values were expected to indicate low levels of specific resistance, because the presence or absence of specific virulence in the isolates would have relatively little effect on the severity of disease on cultivars with little specific resistance. We combined the ranking order from this criterion with those from the two criteria described above, giving equal weight to each, and chose the cultivar with the lowest mean rank value

as the one likely to have the lowest level of specific resistance. With this method we selected the correct host cultivar in 22 of the 27 sets of cultivars.

Specific Resistance Ratings Based on Several Statistics

The object of our analyses is to obtain a statistic highly indicative of the amount of specific resistance in a host cultivar. We have used both the number of genes for specific resistance and the proportion of resistance genes that are specific as measures of specific resistance. The statistics best correlated with the number of specific resistance genes are the mean square for deviations from regression on VIS and the mean disease severity; the slope of the regression on VIM is rather less well correlated.

A rating, based on these three statistics was defined as follows:

$$\text{Rating 1} = \left[\frac{\text{Dev. MS. for regression on VIS}}{\text{Mean Dev. MS. for regression on VIS}} + \frac{\text{Line mean}}{\text{Overall mean}}\right] \times 2 + \text{Slope of regression on VIM.}$$

Rating 1 was significantly correlated with the number of genes for specific resistance in 18 of the 27 trials (Tables 9 and 10). Where more than one cultivar shared the least number of genes for specific resistance, the significance of only the smallest correlation coefficient was considered.

The statistics best correlated with the proportion of resistance genes that are specific are the deviation mean square for regression on VIS and the slope of the regression on VIM. A rating based on these two statistics was defined as follows:

$$\text{Rating 2} = \frac{\text{Dev. MS. for regression on VIS}}{\text{Mean Dev. MS. for regression on VIS}} + \text{Slope of regression on VIM.}$$

Rating 2 was significantly correlated with the proportion of resistance genes that are specific in 11 of the 27 trials (Tables 9 and 10). Other ratings were calculated based on these and other statistics (such as the slopes of the regression on VIS and the mean squares for deviation from the regression on VIM), but they were significantly correlated with measures of specific resistance in fewer trials than were Ratings 1 and 2.

Table 9. Correlation between Rating 1 and the number of genes for specific resistance and between Rating 2 and the proportion of resistance genes that are specific in randomly generated, hypothetical host genotypes in a model[a] in which disease is controlled by four genes each for general and specific resistance and virulence

No. host genotypes	No. pathogen genotypes	Correlation of no. of genes for specific resistance with Rating 1[b]		Correlation of proportion of resistance genes that are specific with Rating 2[c]	
		R	P > p	R	P > p
5	5	0.98	0.003	0.91	0.03
5	5	0.90	0.04	0.67	0.22
5	5	0.97	0.005	0.17	0.78
8	8	0.90	0.002	0.90	0.002
8	8	0.89	0.003	0.36	0.38
8	8	0.84	0.008	0.66	0.07
15	8	0.80 – 0.95	0.0003 – 0.0001	0.50 – 0.74	0.06 – 0.002
15	8	0.71	0.003	0.45	0.04
15	8	0.75	0.001	0.55	0.03
25	8	0.94	0.0001	0.55	0.005
25	8	0.84	0.0001	0.72	0.0001
25	8	0.92	0.0001	0.70	0.0001
15	15	0.30 – 0.75	0.21 – 0.001	0.40 – 0.70	0.14 – 0.004
15	15	0.90	0.0001	0.78	0.0006
15	15	0.49 – 0.92	0.06 – 0.0001	0.14 – 0.54	0.63 – 0.04

[a] Genetic interactions are of the type shown in Table 3

[b] $\text{Rating 1} = \left[\frac{\text{Deviation MS for regression on VIS}}{\text{Mean deviation MS for regression on VIS}} + \frac{\text{Line mean}}{\text{Overall mean}}\right] \times 2 + \text{Slope of regression on VIM}$

[c] $\text{Rating 2} = \frac{\text{Deviation MS for regression on VIS}}{\text{Mean deviation MS for regression on VIS}} + \text{Slope of regression on VIM}$

Table 10. Correlation between Rating 1 and the number of genes for specific resistance and between Rating 2 and the proportion of resistance genes that are specific in host genotypes in a model[a] with 15 and 8 randomly generated, hypothetical host and pathogen genotypes, respectively

No. general genes[b]	No. specific genes[b]	Correlation of no. of genes for specific resistance with Rating 1[c]		Correlation of proportion of resistance genes that are specific with Rating 2[d]	
		R	P>p	R	P>p
8	8	0.24	0.39	0.04 – 0.14	0.99 – 0.62
8	8	0.27 – 0.35	0.33 – 0.20	– 0.003 – 0.04	0.99 – 0.89
8	8	0.72 – 0.80	0.003 – 0.0003	0.26 – 0.33	0.35 – 0.23
12	12	– 0.03 – 0.56	0.90 – 0.03	– 0.25 – 0.12	0.37 – 0.68
12	12	0.64 – 0.71	0.01 – 0.003	0.17 – 0.20	0.55 – 0.48
12	12	0.57 – 0.71	0.03 – 0.003	0.50 – 0.69	0.06 – 0.004
12	4	0.30 – 0.31	0.28 – 0.26	– 0.18 – 0.11	0.53 – 0.70
12	4	0.86	0.0002	0.78	0.0006
12	4	0.09	0.76	– 0.02	0.93
4	12	0.69 – 0.84	0.004 – 0.0001	0.78 – 0.82	0.0006 – 0.0002
4	12	– 0.14 – 0.63	0.61 – 0.01	– 0.38 – 0.30	0.16 – 0.27
4	12	0.26 – 0.47	0.35 – 0.07	– 0.05 – 0.06	0.86 – 0.84

[a] Genetic interactions are of the type shown in Table 3

[b] The numbers of loci for resistance and virulence in the model were equal for both general and specific genes.

[c] $\text{Rating 1} = \left[\frac{\text{Deviation MS for regression on VIS}}{\text{Mean deviation MS for regression on VIS}} + \frac{\text{Line mean}}{\text{Overall mean}}\right] \times 2 + \text{Slope of regression on VIM}$

[d] $\text{Rating 2} = \frac{\text{Deviation MS for regression on VIS}}{\text{Mean deviation MS for regression on VIS}} + \text{Slope of regression on VIM}$

Selection for General Resistance

The analyses described in the preceding sections might be used to rank cultivars according to the probable durability of their resistance, but the procedures are much more laborious than would ordinarily be used in a breeding program to screen lines for horizontal resistance. Breeding lines may be screened against a single isolate of the pathogen or against a mixture of isolates combined into a single inoculum. If a single isolate were used, the best choice would be the one which has the largest number of genes for specific virulence so that the resistance detected with it would be primarily general resistance.

We compared these two methods of screening for horizontal resistance using the randomly generated genotypes analyzed in Tables 5 and 7. To select an isolate with a high level of specific virulence we regressed disease severity for each isolate against a susceptibility index for host cultivars based on their mean disease severity over all isolates. The isolate with the lowest slope in the regressions of each set was chosen as the one likely to have the greatest number of genes for specific virulence. We then calculated correlation coefficients for the relationship between the number of genes for general resistance in each host genotype and its performance against the single, highly virulent isolate or its mean performance against all pathogen isolates in the set.

Correlation coefficients for general resistance in host genotypes in the trials represented in Table 5 tested against a single, highly virulent isolate ranged from 0.295 to 1.000 with a mean of 0.842. When these genotypes were tested against the whole set of pathogen isolates, the corelation coefficients ranged from 0.546 to 0.980 with a mean of 0.840. Correlation coefficients for general resistance in the host genotypes in the trials represented in Table 7 ranged from 0.639 to 0.962 with a mean of 0.858 when tested against a single, highly virulent isolate. When tested against all isolates in each set, the correlation coefficients ranged from 0.649 to 0.970 with a mean of 0.855. The two methods yielded nearly identical results except that the range of variation was greater for tests with a single isolate. There was no discernible relationship between the correlation coefficients and the numbers of genotypes or loci in the sets.

Discussion

One of the advantages of the regression analysis proposed by Leonard and Moll (1981) for evaluating the specificity of disease resistance is that it provides a graphic representation of the response of individual host cultivars to pathogen isolates of varying levels of virulence. Our interpretation of the analysis, however, is somewhat different from that of Leonard and Moll (1981). In their analysis, Leonard and Moll calculated virulence indexes for pathogen isolates based on their mean performance over all host cultivars in the test (i.e. our VIM). They regarded the deviations from the regression lines as indicative of specificity in resistance and the variation in the slopes of the regression lines for different cultivars as indicative of variation in sensitivity of cultivars to differences in general virulence in the pathogen. From our analysis of hypothetical genotypes in our model, we conclude that the variations in the slopes observed by Leonard and Moll were really indications of the relative amounts of specific resistance in the cultivars. In order for the deviations to be interpreted as evidence of levels of specific resistance and virulence in cultivars and isolates, the disease severities should have been regressed against the virulence indexes based on the performance of isolates on cultivars with the least amount of specific resistance (VIS).

In the tests with randomly generated genotypes, the correlations between deviation mean squares and numbers of genes for specific resistance were high in most cases in which there were four loci each for general and specific resistance. In examples with larger numbers of loci, the correlations were not as high. There may be two reasons for this. First, when there are more loci, each contributes a smaller proportion of the total resistance, so it becomes more difficult to estimate accurately the number of genes for specific resistance in any given genotype. Second, if the number of loci for resistance is increased without an increase in the number of genotypes in the test, it becomes less likely that the set of genotypes in the test will include the entire range of possible genotypes from most to least resistant.

The proportion of resistance genes that are specific is really of more interest than the absolute number of specific resistance genes in a host cultivar. Correlations between this proportion and any of the statistics computed for a host cultivar were, however, generally lower than correlations between the statistics and the number of specific resistance genes.

The correlations of regression slopes or deviation mean squares with numbers of genes for specific resistance and proportions of resistance genes that were specific in randomly generated genotypes were not as high as a plant breeder would like. They do not provide a perfect estimate of the potential stability of cultivars with respect to variation in virulence in the pathogen population. On the other hand, the regression analyses provide more information than can be obtained from the standard analysis of variance which has been widely used to indicate the presence or absence of specific resistance. Parlevliet and Zadoks (1977) showed that in a model system of polygenic resistance and virulence in which all of the host and pathogen genes interacted on a gene-for-gene basis the analysis of variance attributed only 2.6% of the total variance to cultivar × isolate interaction. We performed a similar analysis of variance on the data generated from each of our 27 sets of hypothetical genotypes and found that the proportion of total variance attributed to cultivar × isolate interaction ranged from 0 to 3%. If that variance component had been greater, as it would be if selection had occurred in the pathogen population for specific virulence on individual cultivars, the cultivar × isolate variance component could have been much larger and the regression analyses would have provided more accurate estimates of specificity. Ratings based on weighted additive combinations of deviation mean squares for regression on VIS, the slope of the regression on VIM, and the mean disease severity were found to be better estimates of specificity than any single statistic. Rating 1 was significantly correlated with the number of genes for specific resistance in 18 of the 27 trials. Much better estimates of specificity were obtained for sets of host cultivars with 8 loci for resistance than for sets with 16 or more loci for resistance.

While it was not possible to accurately assess the number of genes for specific resistance in all of the randomly generated genotypes in many of the trials with the regression analysis, it was possible to identify individual genotypes with fewer than average numbers of genes for specific resistance. In fact, in 81% of the 27 sets of lines, the line with the least number of genes for specific resistance could be correctly identified from the regression statistics. If this level of success were possible with experimental data, the analyses could provide valuable information about the potential durability of the resistance of cultivars before they have been widely grown commercially.

The analyses tested in these studies do not provide a direct estimate of the durability of resistance of cultivars. Durability depends on many factors, including rates of pathogen reproduction and numbers of generations per year, efficiency of genetic recombination in the pathogen, as well as factors that affect selection pressures such as the intensity of crop production and the distribution of cultivars in the fields. The final test of durability of the resistance of a cultivar is the cultivar's success when grown commercially over a wide area for a period of several years (Johnson

1978). Our analyses could be used to compare the potential durability of resistance of new cultivars relative to that of older cultivars that have been widely tested. The suitability of our analyses could be tested by applying them to a set of cultivars with known records of durability or lack of durability of their resistance.

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