Theor Appl Genet (1985) 69:503–513

Reliability of statistical analyses for estimating relative specificity in quantitative resistance in a model host-pathogen system

A. E. Jenns [1] and K. J. Leonard [2]

[1] Department of Plant Pathology, [2] U.S. Department of Agriculture, Agricultural Research Service, Department of Plant Pathology; North Carolina State University, Raleigh, NC 27695, USA

Received June 27, 1984; Accepted July 20, 1984
Communicated by G. S. Khush

Summary. The reliability of analyses of variance for evaluating host cultivar × pathogen isolate specificity in resistance controlled by polygenes with additive effects was tested with combinations of hypothetical host and pathogen genotypes in a model system. In each test, varying numbers of host and pathogen genotypes were combined in all combinations, the resulting disease severities were calculated according to the model, and those data were subjected to analysis of variance. The percentage of total variance accounted for by host × pathogen interaction decreased with increasing numbers of host and pathogen genotypes per test. Simulated selection for virulence among randomly generated pathogen genotypes increased the percentage of variance attributable to host × pathogen genotype interaction, but simulated selection for resistance among host genotypes decreased it. The percentage of variance accounted for by interaction was greatest when selection of resistant host genotypes was followed by selection of the most virulent pathogen genotype on each selected host genotype. When gene frequencies were varied in the model, the interaction variance was greatest at low frequencies of resistance genes and high frequencies of virulence genes, but the number of matches between genes for specific virulence and specific resistance was greatest for high frequencies of both resistance and virulence genes. A simplified method of analysis was developed to estimate the amount of specific resistance in a set of host genotypes inoculated in all combinations with a set of pathogen genotypes. This method, based on the variance of disease severity adjusted to remove general virulence, proved consistently accurate with varying numbers of genotypes in the set, varying numbers of loci for resistance and virulence, and varying frequencies of genes for resistance and virulence. The variance method is of comparable accuracy and is much simpler than the previously proposed methods based on regression analysis. Simulated selection for resistance in the host and for virulence in the pathogen population increased the accuracy of both the variance method and the regression method.

Key words: Statistical analyses – Specific resistance – Host-pathogen system

Introduction

Non-specific disease resistance, which is effective against all pathogen genotypes, should be more durable than specific resistance that is effective against certain pathogen genotypes and not others. Polygenic resistance has been assumed to be non-specific and thus durable (Vanderplank 1963; Robinson 1976), but before recommending it over monogenic resistance, it is important to demonstrate the non-specificity and greater durability of polygenic resistance, because this incorporation of polygenic resistance to several diseases into a cultivar would be a slow and difficult process (Crill et al. 1974).

According to Vanderplank (1968), horizontal (non-specific) resistance can be identified by inoculating a number of host genotypes in all combinations with a number of pathogen genotypes. Vanderplank regarded the resistance as horizontal if analysis of variance shows no statistically significant contribution of host genotype × pathogen genotype to the variation in disease severity among host-pathogen combinations. Parlevliet and Zadoks, however, (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.

Vanderplank's proposal and Parlevliet and Zadoks' model illustrate two fundamentally different approaches to the inter-

pretation of variation in the degree of specificity in host-pathogen interactions. The analysis of variance provides information about the uniqueness of levels of disease severity among specific combinations of host and pathogen genotypes. The analysis of variance does not, however, provide easily interpretable information about the number or proportion of resistance genes in a cultivar that interact specifically with genes for specific virulence in the pathogen.

To predict the durability of resistance in cultivars, we must go beyond information about uniqueness of disease severity levels among cultivar × isolate combinations. Jenns et al. (1982) developed methods to estimate proportions of specific and non-specific resistance genes in a model system. They generated disease severity data from sets of hypothetical genotypes which had genes for both specific and general (non-specific) resistance or virulence. In that study and in the present study, we use the term general resistance to designate resistance genes equally effective against all genotypes of the single pathogen species in question. Similarly, we use the term general virulence to designate virulence genes equally effective against all genotypes of the single host species in question. Genes for specific resistance by our terminology are those that are more effective against some pathogen genotypes than others. We are concerned with quantitative inheritance in which the differences among pathogen genotypes may not be of sufficient magnitude to define very distinct pathogenic races. Therefore, we have not referred to this resistance as race-specific resistance, a term usually reserved for combinations in which obvious qualitative differences exist.

When numbers of loci and numbers of host and pathogen genotypes in sets of combinations in the model of Jenns et al. (1982) were varied (from 4 to 12 loci for general and specific resistance or virulence and from 5 to 25 host genotypes and 5–15 pathogen genotypes per set) and analysis of variance was performed on the data generated, no more than 3% of the total variance was ever attributable to host × pathogen genotype interaction. Using this model, Jenns et al. (1982) developed a method based on a regression analysis for ranking cultivars according to the number of genes for specific resistance they possess. This method, however, was not totally accurate, even with hypothetical data. Jenns et al. (1982) suggested that the reliability of their ranking method would be increased if selection for specific virulence on individual cultivars had occurred in the pathogen population. Such selection should decrease the randomness of the distribution of specific virulence genes among pathogen isolates and increase the uniqueness of specific combinations of virulence genes among isolates. Therefore, selection should make it easier to distinguish between resistance due to genes with specific effects and that due to genes with general effects.

We wanted to test the hypotheses that selection in the pathogen population for specific virulence would a) increase the proportion of variance due to the cultivar × isolate interaction and b) increase the reliability of the regression analysis method of ranking cultivars by their specific resistance. Also, since the regression analysis method of Jenns et al. (1982) is complicated, we attemped to develop a simpler, but equally accurate method for assessing specific resistance in cultivars.

The model

The model used is that of Jenns et al. (1982), and combines both specific and general resistance and virulence. 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. Genes for specific resistance and virulence are assumed to interact in a gene-for-gene relationship, matched pairs of resistance × virulence genes contributing an additive increment to the total disease severity. A gene for specific resistance is only expressed in the absence of the corresponding gene for specific virulence in the pathogen. Similarly, a gene for specific virulence only contributes to increased disease severity if the corresponding gene for specific resistance is present in the host.

Disease severity is expressed as follows:

Dis. sev. = (Number of *rg*) + (Number of *Vg*) + (Number of *rs*) + (Number of *Vs-Rs* matches)

where *Rg* and *Rs* are general and specific resistance alleles, *rg* and *rs* are corresponding alleles for susceptibility, *Vg* and *Vs* are general and specific virulence alleles and *vg* and *vs* are corresponding alleles for avirulence (Table 1).

A computer program was used to generate random host and pathogen genotypes and to calculate the disease severities for each combination (Jenns et al. 1982). The program allowed the number of loci for general and specific resistance and virulence, the number of host and pathogen genotypes, and the frequency of alleles for resistance or virulence to be varied. All loci that contributed to disease severity were assumed to contribute equal increments to the total disease severity.

In the model no dominance or epistatic interactions between genes were assumed, and the chromosomal location of genes was not specified in order to simplify the analysis. Each gene was assumed to occur at its locus and to produce its effect on disease severity independently. This, of course, would not be true for a diploid host with predominantly homozygous genotypes, in which the occurrence of one gene for resistance at a given locus nearly always implies the occurrence of the second allele for resistance at that locus on the homologous chromosome. In the model this relationship for homozygous diploids would simply change the scale of interactions. The basic pattern would remain qualitatively similar to that de-

Table 1. Severity of disease in combinations of hypothetical host and pathogen genotypes in a model with genes for both general and specific resistance and virulence

Pathogen genotypes	Disease severity[a] on host genotypes							
	rg *rs*$_1$ *rs*$_2$	*rg* *Rs*$_1$ *rs*$_2$	*rg* *rs*$_1$ *Rs*$_2$	*rg* *Rs*$_1$ *Rs*$_2$	*Rg* *rs*$_1$ *rs*$_2$	*Rg* *Rs*$_1$ *rs*$_2$	*Rg* *rs*$_1$ *Rs*$_2$	*Rg* *Rs*$_1$ *Rs*$_2$
Vg Vs$_1$ *Vs*$_2$	4	4	4	4	3	3	3	3
Vg Vs$_1$ *vs*$_2$	4	4	3	3	3	3	2	2
Vg vs$_1$ *Vs*$_2$	4	3	4	3	3	2	3	2
Vg vs$_1$ *vs*$_2$	4	3	3	2	3	2	2	1
vg Vs$_1$ *Vs*$_2$	3	3	3	3	2	2	2	2
vg Vs$_1$ *vs*$_2$	3	3	2	2	2	2	1	1
vg vs$_1$ *Vs*$_2$	3	2	3	2	2	1	2	1
vg vs$_1$ *vs*$_2$	3	2	2	1	2	1	1	0

[a] Disease severity is expressed as follows: Dis. Sev. = 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 susceptibility, *Vg* and *Vs* are general and specific virulence alleles and *vg* and *vs* are corresponding alleles for avirulence

scribed by Jenns et al. (1982). The same would be true for combinations of a diploid host and haploid pathogen.

Effect of simulated selection and other variables on detection of specificity by analysis of variance

A set of 50 pathogen genotypes and 10 host genotypes each with eight loci for general and eight loci for specific resistance or virulence was generated. From the original set of 50 pathogen genotypes, a smaller population of 10 genotypes was chosen at random for some tests. In other tests, a population of 10 pathogen genotypes was selected by choosing the genotype that produced the greatest disease severity on each host genotype. The disease severities produced by these randomly chosen or selected pathogen genotypes on the 10 host genotypes were subjected to analysis of variance. This procedure was repeated for a set of pathogen genotypes and host genotypes with eight loci for specific and none for general resistance or virulence.

The percentage of total variance attributable to the host genotype × pathogen genotype interaction was slightly larger for the selected set of pathogen genotypes consisting of the most virulent on each host genotype than for the set of randomly chosen pathogen genotypes (Table 2), which confirms the expected effect of selection.

A similar procedure was carried out for a set of 50 host genotypes and 10 pathogen genotypes having eight loci for specific resistance or virulence and either eight or no loci for general resistance or virulence. The percentage of total variance attributable to pathogen genotype × host genotype interaction was slightly smaller for a selected set of host genotypes consisting of the most resistant one to each pathogen genotype than for the same number of randomly chosen host genotypes (Table 2). Thus selection for resistance reduced the detectable levels of host genotype × pathogen genotype specificity.

With sets of genotypes that contained no loci for general resistance and virulence, the percentage of variance accounted for by host × pathogen genotype interaction was about 10-fold greater than for sets of genotypes with equal numbers of loci for general and specific resistance and virulence (Table 2).

From an original set of 50 host genotypes and 50 pathogen genotypes, smaller populations of 30, or 10

Table 2. Effect of selection for virulence or resistance on the proportion of total variance accounted for by host × pathogen genotype interaction in an analysis of variance in disease severities produced by 10 model pathogen genotypes on 10 model host genotypes chosen at random or selected for virulence or resistance from an original set of 50 genotypes

Treatment	No. of loci for resistance and virulence		Interaction as percentage of total variance
	General	Specific	
Random	8	8	0.65
Random	0	8	5.30
Pathogen selected[a]	8	8	0.80
Pathogen selected	0	8	11.40
Host selected[b]	8	8	0.40
Host selected	0	8	2.70

[a] Most virulent pathogen genotype on each randomly chosen host genotype

[b] Most resistant host genotype to each randomly chosen pathogen genotype

Table 3. Effect of simulated selection and number of genotypes per test on percentage of total variance accounted for by host × pathogen genotype interaction

No. of genotypes[a]		Selection procedure	Interaction as percentage of total variance[b]
Host	Pathogen		
50	50	none	0.25
50	30	none	0.28
50	10	none	0.39
30	50	none	0.34
30	30	none	0.42
30	10	none	0.65
10	50	none	0.76
10	30	none	0.96
10	10	none	1.42
10	10	specific virulence[c]	2.71
10	10	mean virulence[d]	2.61
10	10	specific resistance[e]	1.29
10	10	mean resistance[f]	1.17
10	10	mean virulence and specific resistance[g]	1.00
10	10	mean resistance and specific virulence[h]	3.85
10	10	mean virulence[d] and mean resistance[f]	3.02

[a] Host and pathogen genotypes have eight loci for general and eight loci for specific resistance or virulence. The frequency of genes for resistance and virulence is 0.5

[b] Figures are the means of analyses from two original sets of host and pathogen genotypes

[c] Most virulent pathogen genotypes on each of 10 random host genotypes. Frequency of $Vg = 0.8$, frequency of $Vs = 0.7$

[d] 10 most virulent pathogen genotypes over all 50 host genotypes. Frequency of $Vg = 0.7$, frequency of $Vs = 0.6$

[e] Most resistant host genotypes to each of 10 random pathogen genotypes. Frequency of $Rg = 0.7$; frequency of $Rs = 0.6$

[f] 10 most resistant host genotypes over all 50 pathogen genotypes. Frequency of $Rg = 0.7$, frequency of $Rs = 0.7$

[g] Most resistant host genotypes to each of 10 most virulent pathogen genotypes. Frequency of $Vg = 0.7$; $Vs = 0.6$; $Rg = 0.6$; $Rs = 0.7$

[h] Most virulent pathogen genotypes on each of 10 most resistant host genotypes. Frequency of $Vg = 0.8$; $Vs = 0.7$; $Rg = 0.7$; $Rs = 0.7$

Table 4. Effect of number of loci for resistance and virulence on the percentage of variance accounted for by host genotype × pathogen genotype interactions where the disease severities caused by eight pathogen genotypes on 15 host genotypes in all combinations are subjected to analysis of variance

No. of loci for resistance and virulence[a]		Interaction as percentage of total variance[b]
General	Specific	
4	4	0.99
8	8	0.82
12	12	0.94
4	12	1.64
12	4	0.46

[a] The frequency of genes for host resistance and pathogen virulence is 0.5

[b] Mean for three sets of genotypes

host and pathogen genotypes were chosen at random. As the number of randomly chosen genotypes of either the pathogen or the host was reduced, the percentage of total variance attributable to host genotype × pathogen genotype interaction increased (Table 3). Thus, cultivar × isolate interaction would appear to be more easily detected by analysis of variance for tests with low numbers of cultivars and isolates than with high numbers.

Ten host and ten pathogen genotypes were selected in various ways from the original 50 host and 50 pathogen genotypes, and the percentage of variance accounted for by host × pathogen genotype interaction was calculated for each selected set of genotypes. In general, when host genotypes were selected for resistance the percentage of variance attributable to interaction decreased and when pathogen genotypes were selected for virulence the percentage increased compared to the percentage for the same number of randomly chosen genotypes (Table 3). The largest percentage was obtained when the most resistant host genotypes overall and the most virulent pathogen genotype on each were included in the set (Table 3).

Varying the number of loci for resistance and virulence in host and pathogen genotypes had little effect on the percentage of total variance attributed to host genotype × pathogen genotype interaction (Table 4). Increasing the ratio of specific to general loci for resistance and virulence increased this percentage, but even when the ratio was three to one the percentage was only 1.6 (Table 4) compared with a percentage of 5.3 when only specific genes were included (Table 2).

In hypothetical genotypes in which all eight loci were for specific resistance or virulence, the highest percentage of total variance attributable to host genotype × pathogen genotype interaction was obtained with low frequencies of genes for resistance and high

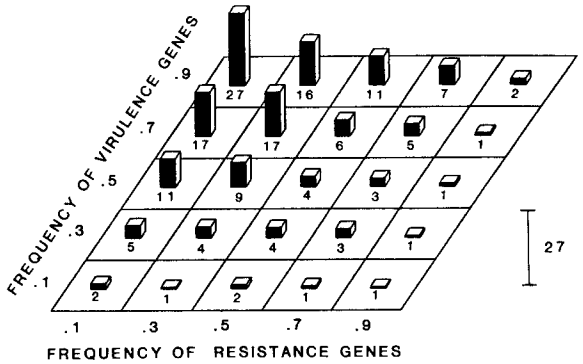

Fig. 1. Effects of frequency of specific resistance and specific virulence genes on the percentage of total variance accounted for by host × pathogen genotype interaction in analysis of variance of disease severities produced by eight model pathogen genotypes with eight loci for specific virulence on eight model host genotypes with eight loci for specific resistance

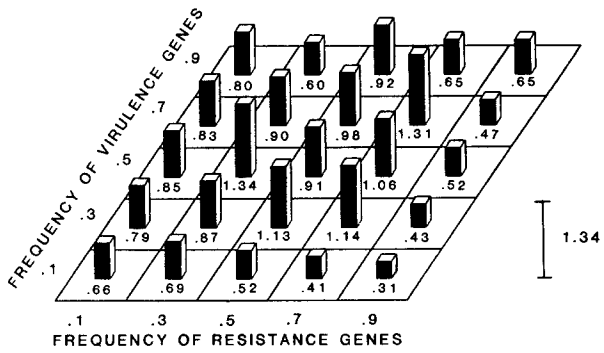

Fig. 2. Effects of frequency of specific and general resistance and specific and general virulence genes on the percentage of total variance accounted for by host × pathogen genotype interaction in analysis of variance of disease severities produced by eight model pathogen genotypes with eight loci each for specific and general virulence on eight model host genotypes with eight loci each for specific and general resistance

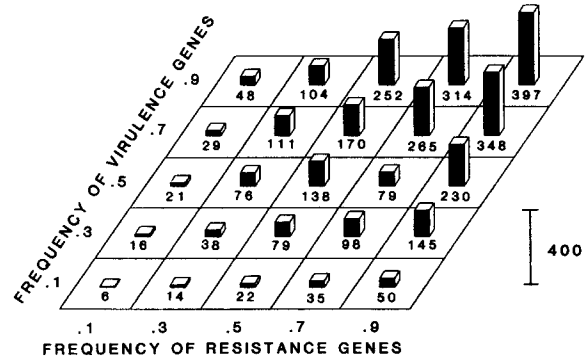

Fig. 3. Effects of frequency of specific resistance and specific virulence genes on the number of matched pairs of specific virulence and specific resistance genes in sets of eight model pathogen genotypes with eight loci for specific virulence and eight model host genotypes with eight loci for specific resistance

frequencies of genes for virulence (Fig. 1). When eight loci for general resistance and virulence were added to the genotypes, the relationship of gene frequency to the percentage of variance attributable to host × pathogen genotype interaction was less obvious (Fig. 2).

The variance attributable to host genotype × pathogen genotype interaction is a measure of the uniqueness of levels of disease severity resulting from specific combinations of host and pathogen genotypes. This interaction variance does not, however, provide a good indication of the amount of specific resistance gene × virulence gene interactions that occur. For example, in the model the number of *Vs-Rs* matches is highest at high frequencies of both resistance and virulence genes (Fig. 3). Thus, there was little correlation between the interaction variance and the number of *Vs-Rs* matches. In breeding for durable resistance, it would be most important to detect cultivar × isolate specificity in situations in which the frequencies of specific virulence genes were relatively low (before adaptation had occurred) and the frequencies of resistance genes were low to moderate (selection is still needed to increase the resistance). In these situations both the number of *Vs-Rs* matches and the host genotype × pathogen isolate interaction variance will be low (Figs. 1 and 3). If half of the host resistance loci are for general resistance, the interaction variance will be low regardless of the frequencies of genes for specific virulence (Fig. 2).

Simplified analysis to detect specific resistance

The specific resistance ratings of Jenns et al. (1982) computed with statistics obtained from regression analysis proved to be fairly accurate estimates of the numbers of genes for specific resistance in model host genotypes, but were less accurate estimates of the proportions of their resistance genes that were specific. Computation of these ratings, however, is quite laborious and depends upon accurate recognition of the host genotype in each test with the least specific resistance. Thus, a simpler method of estimating the number and proportion of genes for specific resistance would be very useful.

Variation among pathogen isolates in the severity of disease that they cause on a host cultivar can be attributed to experimental error, variation in the levels of general virulence among pathogen isolates, and variation in the amount of matching of specific virulence of pathogen isolates with the specific resistance of the host. Therefore, the variation due to interactions with the host's specific resistance can be estimated if the variation due to experimental error and the variation in levels of general virulence can be accounted for.

In the theoretical host genotype/pathogen genotype combinations in Table 1, the disease severity due to general virulence can be subtracted from the total disease severity to calculate adjusted disease severities that reflect the effects of specific virulence in each combination. The variance of these adjusted disease severities over all pathogen genotypes on each host genotype is correlated with the number of genes for specific resistance in the host genotype (Table 5). Variation in disease severity on a susceptible check cultivar with no specific resistance, such as the host genotype $rgrs_1rs_2$ in Table 1 can be used to estimate the combined effects of experimental error and variation among pathogen isolates in general virulence. Thus, the adjusted disease severity for each isolate on a test cultivar can be calculated by subtracting the severity caused by each isolate on the check cultivar, and the variance of the adjusted disease severities can be used to estimate the amount of specific resistance in the test cultivar. This method ranked the host genotypes in Table 1 correctly by number of genes for specific resistance (Table 5).

Table 5. Variance analysis of disease severity in all combinations of hypothetical host and pathogen genotypes in Table 1

Host genotype	Variance of (disease severity – no. genes for general virulence in pathogen)	Variance of (disease severity – disease severity on $rgrs_1rs_2$)
$rgrs_1rs_2$	0.000	0.000
$rgRs_1rs_2$	0.286	0.286
$rgrs_1Rs_2$	0.286	0.286
$rgRs_1Rs_2$	0.572	0.572
$Rgrs_1rs_2$	0.000	0.000
$RgRs_1rs_2$	0.286	0.286
$Rgrs_1Rs_2$	0.286	0.286
$RgRs_1Rs_2$	0.572	0.572

When a susceptible check is not available, the host genotype with the fewest genes for specific resistance may be substituted. The genotype with fewest genes for specific resistance can be identified by the low variation among pathogen genotypes in the severity of disease that they cause on it.

This variance among pathogen genotypes in disease severity, adjusted either by subtracting the number of genes for general virulence or by subtracting the disease severity on the line expected to have the least specific resistance, was calculated for each host genotype in randomly generated sets. The correlation between these variances for host genotypes and numbers of their genes for specific resistance or the proportions of their resistance genes that were specific was calculated.

The variance of disease severity, either unadjusted or adjusted by subtraction of general virulence, was well correlated with the number and proportion of specific resistance genes and seemed independent of the number of host and pathogen genotypes in the set (Tables 6 and 7). Ratings 1 and 2 of Jenns et al. (1982) were about equally well correlated with specific resistance if regression was on a host genotype with no specific resistance. The variance of disease severity adjusted by subtraction of disease severity on a susceptible host genotype in the set and Ratings 1 and 2 using the host genotype in the set with the least specific resistance were less

Table 6. Effect of number of model host and pathogen genotypes in tests of cultivar × isolate specificity on the correlation of four statistical measures of specificity with the number of genes for specific resistance per model genotype

No. of genotypes[b]		Correlation (R)[a] of no. of genes for specific resistance with:				
Host	Pathogen	Variance disease severity[c]	Variance (disease severity – general virulence)[d]	Variance (disease severity – disease severity on host genotype with least specific resistance)[e]	Rating 1 with host genotype with no specific resistance[f]	Rating 1 with host genotype with least specific resistance[g]
50	50	0.69	0.79	0.40	0.73	0.33
50	30	0.63	0.74	0.31	0.62	0.34
50	10	0.63	0.59	0.26	0.46	0.20
30	50	0.76	0.85	0.43	0.79	0.40
30	30	0.71	0.76	0.33	0.66	0.38
30	10	0.68	0.67	0.27	0.52	0.26
10	50	0.73	0.81	0.48	0.79	0.33
10	30	0.74	0.76	0.46	0.68	0.33
10	10	0.69	0.67	0.08	0.59	0.19

[a] Correlation coefficients are means from two sets of disease data
[b] Genotypes have eight loci for general and eight loci for specific resistance or virulence. The frequency of genes for resistance or virulence is 0.5
[c] Variance in disease severity for host genotypes over all pathogen genotypes in the test
[d] General virulence = no. of genes for general virulence in pathogen genotype
[e] Host genotype with least specific resistance chosen by inspection
[f] Rating 1 = [(deviation mean square for regression on disease severity on host genotype with no specific resistance/mean deviation mean square) + host genotype mean/overall mean)] × 2 + slope of regression on mean disease severity over all host genotypes (see Jenns et al. 1982)
[g] Rating 1 = [(deviation mean square for regression on disease severity on host genotype with least specific resistance/mean deviation mean square) + host genotype mean/overall mean)] × 2 + slope of regression on mean disease severity over all host genotypes (see Jenns et al. 1982)

Table 7. Effect of number of model host × pathogen genotypes in tests of cultivar × isolate specificity on the correlation of four statistical measures of specificity with the proportion of resistance genes that are specific per model genotype

No. of genotypes[b]		Correlation (R)[a] of no. of genes for specific resistance with:				
Host	Pathogen	Variance disease severity[c]	Variance (disease severity – general virulence)[d]	Variance (disease severity – disease severity on host genotype with least specific resistance)[e]	Rating 2 with host genotype with no specific resistance[f]	Rating 2 with host genotype with least specific resistance[g]
50	50	0.56	0.56	0.30	0.58	0.33
50	30	0.53	0.51	0.25	0.32	0.40
50	10	0.47	0.43	0.16	0.42	0.25
30	50	0.65	0.64	0.38	0.68	0.49
30	30	0.61	0.58	0.30	0.58	0.46
30	10	0.52	0.49	0.21	0.44	0.27
10	50	0.69	0.67	0.52	0.77	0.49
10	30	0.68	0.66	0.46	0.63	0.44
10	10	0.73	0.63	0.15	0.70	0.38

[a] See Table 6; [b] See Table 6; [c] See Table 6; [d] See Table 6; [e] See Table 6
[f] Rating 2 = (deviation mean square for regression on a host genotype with no specific resistance/mean deviation mean square) + slope of regression on mean disease severity over all host genotypes (see Jenns et al. 1982)
[g] Rating 2 = (deviation mean square for regression on the host genotype in the set with the least specific resistance/mean deviation mean square) + slope of regression on mean disease severity over all host genotypes (see Jenns et al. 1982)

well correlated with the number or proportion of genes for specific resistance, especially with lower numbers of host and pathogen genotypes (Tables 6 and 7).

When the number of loci for resistance and virulence was varied from four to 12, the variance of disease severity adjusted by subtraction of general virulence was most consistently well correlated with both the number of genes for specific resistance and the proportion of resistance genes that were specific. The variance of disease severity adjusted by subtraction of the disease severity on the host genotype with the least specific resistance and Ratings 1 and 2, based on a host genotype with no specific resistance were less well correlated. The Ratings 1 and 2 based on the host genotype in the set with the least specific resistance and the unadjusted variance of disease severity were least well correlated with specific resistance (Tables 8 and 9).

When the frequencies of genes for resistance and virulence were varied, the variance of the disease severity, adjusted by subtraction of either general virulence or disease severity on the host genotype with the least specific resistance was most consistently well correlated with the number of specific resistance genes (Table 10). The variance of unadjusted disease severity was less consistent but generally better than Rating 1 (Table 10). The variance of the disease severity, adjusted by subtraction of either general virulence or disease severity on the host genotype with the least specific resistance was most consistently well correlated with the proportion of resistance genes that were specific (Table 11). Rating methods were next best correlated and the unadjusted variance was least well correlated with the proportion of resistance genes that were specific (Table 11).

Table 8. Effect of number of loci for resistance and virulence on the correlation of four statistical measures of specificity with the number of genes for specific resistance per model genotype

No. of loci for resistance and virulence[b]		Correlation (R)[a] of no. of genes for specific resistance with:				
General	Specific	Variance disease severity[c]	Variance (disease severity – general virulence)[d]	Variance (disease severity – disease severity on host genotype with least specific resistance)[e]	Rating 1 with host genotype with no specific resistance[f]	Rating 1 with host genotype with least specific resistance[g]
4	4	0.08	0.80	0.71	0.67	0.75
8	8	0.22	0.32	0.36	0.26	0.41
12	12	0.13	0.78	0.59	0.54	0.39
4	12	0.43	0.58	0.47	0.63	0.27
12	4	0.25	0.55	0.42	0.44	0.42

[a] Correlation coefficients are means from three sets of disease data
[b] Fifteen host and eight pathogen genotypes in each set with a mean frequency of resistance and virulence genes of 0.5
[c] See Table 6; [d] See Table 6; [e] See Table 6; [f] See Table 6; [g] See Table 6

Table 9. Effect of number of loci for resistance and virulence on the correlation of four statistical measures of specificity with the proportion of resistance genes that are specific per model genotype

No. of loci for resistance and virulence[b]		Correlation (R)[a] of no. of genes for specific resistance with:				
General	Specific	Variance disease severity[c]	Variance (disease severity – general virulence)[d]	Variance (disease severity – disease severity on host genotype with least specific resistance)[e]	Rating 1 with host genotype with no specific resistance[f]	Rating 1 with host genotype with least specific resistance[g]
4	4	0.17	0.61	0.59	0.44	0.53
8	8	0.03	0.03	0.08	0.26	0.10
12	12	0.13	0.45	0.38	0.21	0.14
4	12	0.33	0.29	0.27	0.54	0.12
12	4	0.09	0.33	0.35	0.18	0.19

[a] See Table 8; [b] See Table 8; [c] See Table 6; [d] See Table 6; [e] See Table 6; [f] See Table 7; [g] See Table 7

Table 10. Effect of frequency of resistance and virulence genes on the correlation of four statistical measures of specificity with the number of genes for specific resistance per model genotype

Frequency general and specific genes for		Correlation (R)[a] of no. of genes for specific resistance with:				
Resistance	Virulence[b]	Variance disease severity[c]	Variance (disease severity – general virulence)[d]	Variance (disease severity – disease severity on host genotype with least specific resistance)[e]	Rating 1 with host genotype with no specific resistance[f]	Rating 1 with host genotype with least specific resistance[g]
0.9	0.1	0.60	0.52	0.45	–0.35	0.05
0.5	0.5	0.37	0.61	0.60	0.26	0.21
0.3	0.5	0.08	0.67	0.72	0.68	0.51
0.3	0.7	0.77	0.66	0.75	0.67	0.39

[a] See Table 8; [b] Eight host and eight pathogen genotypes with eight loci each for general and specific resistance and virulence in each set; [c] See Table 6; [d] See Table 6; [e] See Table 6; [f] See Table 6; [g] See Table 6

Table 11. Effect of frequency of resistance and virulence genes on the correlation of four statistical measures of specificity with the proportion of resistance genes that are specific per model genotype

Frequency general and specific genes for		Correlation (R)[a] of proportion of resistance genes that are specific with:				
Resistance	Virulence[b]	Variance disease severity[c]	Variance (disease severity – general virulence)[d]	Variance (disease severity – disease severity on host genotype with least specific resistance)[e]	Rating 2 with host genotype with no specific resistance[f]	Rating 2 with host genotype with least specific resistance[g]
0.9	0.1	0.14	0.15	0.30	0.19	0.55
0.5	0.5	0.11	0.51	0.42	0.06	0.16
0.3	0.5	–0.05	0.64	0.74	0.54	0.46
0.3	0.7	0.50	0.38	0.36	0.31	0.41

[a] See Table 8; [b] See Table 10; [c] See Table 6; [d] See Table 6; [e] See Table 6; [f] See Table 7; [g] See Table 7

Effect of selection on identification of specific resistance

Jenns et al. (1982) suggested that their method for identifying specific resistance would provide more accurate estimates if selection for specific virulence on individual cultivars had occurred in the pathogen population. We tested the effect of various ways of selecting 10 host and 10 pathogen genotypes from a population of 50 host and 50 pathogen genotypes on the ability of both Rating 1 and Rating 2, based on regression analyses, and the variance methods to estimate the level of specific resistance in host genotypes.

The accuracy of both the variance statistics and Ratings 1 and 2 for prediction of number or proportion of specific resistance genes was generally improved by any selection method that involved selection of both host and pathogen genotypes (Tables 12 and 13). Selection for specific resistance reduced whereas selection for general resistance increased the accuracy of most statistics. Selection for virulence usually increased the accuracy of variance statistics but decreased that of Ratings 1 and 2.

Discussion

Parlevliet und Zadoks (1977) demonstrated that low percentages of total variance attributable to cultivar × isolate interactions in an analysis of variance should not be regarded as conclusive evidence for the absence of specific interactions among genes for resistance and genes for virulence when the resistance is quantitatively inherited. Our results support that conclusion and also show that the percentage of variance attributable to cultivar × isolate interaction tends to be greater for tests involving few host and pathogen geno-

Table 12. Effect of simulated selection for resistance or virulence among model host or pathogen genotypes on the correlation of four statistical measures of specificity with the number of genes for specific resistance per model host genotype

Selection procedure[a]	Correlation (R)[a] of no. of genes for specific resistance with:									
	Variance disease severity[b]		Variance (disease severity – general virulence)[c]		Variance (disease severity – disease severity on host genotype with least specific resistance)[d]		Rating 1 with host genotype with no specific resistance[e]		Rating 1 with host genotype with least specific resistance[f]	
	R	P > rho	R	P > rho	R	P > rho	*P*	P > rho	*P*	P > rho
None	0.60	0.07	0.58	0.08	0.21	0.55	0.54	0.11	0.27	0.45
A[g]	0.41	0.24	0.69	0.03	0.76	0.01	0.48	0.16	0.59	0.09
B[h]	0.08	0.84	0.73	0.02	0.38	0.28	0.42	0.23	0.25	0.52
C[i]	0.49	0.15	0.56	0.09	–0.21	0.55	0.56	0.09	–0.05	0.90
D[j]	0.68	0.03	0.71	0.02	0.31	0.38	0.70	0.02	0.08	0.84
E[k]	0.31	0.38	0.77	0.01	0.60	0.07	0.71	0.02	0.37	0.32
F[l]	0.71	0.02	0.70	0.01	0.60	0.07	0.71	0.02	0.37	0.32
G[m]	0.58	0.08	0.81	0.004	0.58	0.08	0.77	0.009	0.40	0.29

[a] All sets consist of 10 host and 10 pathogen genotypes selected from the same set of 50 host and pathogen genotypes. Genotypes contain eight loci each for general and specific resistance and virulence, and resistance and virulence genes occurred with a frequency of 0.5 each for general and specific resistance and virulence in the original set

[b] Variance in disease severity for cultivars over all pathogen isolates in the test

[c] General virulence = no. of genes for general virulence in pathogen genotype

[d] Host genotype with least specific resistance chosen by inspection

[e] Rating 1 = [(deviation mean square for regression on disease severity on host genotype with no specific resistance/mean deviation mean square) + host genotype mean/overall mean)] × 2 + slope of regression on mean disease severity over all host genotypes (see Jenns et al. 1982)

[f] Rating 1 = [(deviation mean square for regression on disease severity on host genotype with least specific resistance/mean deviation mean square) + host genotype mean/overall mean)] × 2 + slope of regression on mean disease severity over all host genotypes (see Jenns et al. 1982)

[g] Most virulent pathogen genotypes on each of 10 random host genotypes

[h] Ten most virulent pathogen genotypes over all 50 host genotypes

[i] Most resistant host genotypes to each of 10 random pathogen genotypes

[j] Ten most resistant host genotypes over all 50 pathogen genotypes

[k] Most resistant host genotypes to each of 10 most virulent pathogen genotypes

[l] Most virulent pathogen genotypes on each of 10 most resistant host genotypes

[m] Ten most resistant host genotypes and 10 most virulent pathogen genotypes over all

Table 13. Effect of simulated selection for resistance or virulence among model host or pathogen genotypes on the correlation of four statistical measure of specificity with the proportion of resistance genes that are specific per model genotype

Selection procedure[a]	Correlation (R)[a] of proportion of resistance genes that are specific with:									
	Variance disease severity[b]		Variance (disease severity – general virulence)[c]		Variance (disease severity – disease severity on host genotype with least specific resistance)[d]		Rating 2 with host genotype with no specific resistance[e]		Rating 2 with host genotype with least specific resistance[f]	
	R	P > rho	R	P > rho	R	P > rho	*P*	P > rho	*P*	P > rho
None	0.63	0.05	0.59	0.08	0.29	0.42	0.66	0.04	0.48	0.16
A[g]	0.26	0.47	0.57	0.09	0.71	0.02	0.33	0.36	0.34	0.36
B[h]	–0.05	0.88	0.62	0.05	0.48	0.16	0.24	0.50	0.04	0.91
C[i]	0.48	0.16	0.57	0.08	–0.22	0.55	0.64	0.05	0.25	0.52
D[j]	0.64	0.05	0.67	0.03	0.34	0.33	0.70	0.003	0.28	0.46
E[k]	0.63	0.05	0.86	0.002	0.74	0.02	0.73	0.02	0.62	0.08
F[l]	0.70	0.02	0.68	0.03	0.72	0.02	0.57	0.08	0.74	0.02
G[m]	0.66	0.04	0.86	0.001	0.67	0.03	0.84	0.003	0.58	0.10

[a] See Table 12; [b] See Table 12; [c] See Table 12; [d] See Table 12;

[e] Rating 2 = (deviation mean square for regression on disease severity on host genotype with no specific resistance/mean deviation mean square) + slope of regression on mean disease severity over all host genotypes (see Jenns et al. 1982)

[f] Rating 2 = (deviation mean square for regression on disease severity on host genotype with least specific resistance/mean deviation mean square) + slope of regression on mean disease severity over all host genotypes (see Jenns et al. 1982)

[g] See Table 12; [h] See Table 12; [i] See Table 12; [j] See Table 12; [k] See Table 12; [l] See Table 12; [m] See Table 12

types than for those involving many. Therefore, it is difficult to compare the results of analysis of variance for tests of different host-pathogen combinations if the tests involve different numbers of genotypes in each test. Also, while the percentage of variance attributable to interaction does not determine the significance of the interaction, detection of statistically significant host × pathogen genotype interactions may depend quite arbitrarily on experimental design as much as on the actual proportion of genes with general and specific effects.

It should be emphasized that the analysis of variance was not designed to provide estimates of the numbers of specific resistance gene-virulence gene matches that occur among host and pathogen combinations. In fact, at high frequencies of both specific resistance genes and specific virulence genes, the level of cultivar × isolate interaction detected by an analysis of variance was lower than at low frequencies of specific resistance genes, even though the number of *Rs-Vs* gene matches was greater with high frequencies of both *Rs* and *Vs* genes.

Inclusion of genes for general resistance and virulence in the model greatly reduced the percentage of total variance attributable to cultivar × isolate interaction. The percentage was about 10 times greater when all the genes were specific than when half were general, and was about twice as much when three-fourths of the genes were specific than when half were specific.

As expected, selection for virulence in the pathogen population did increase the percentage of variance accounted for by host × pathogen genotype interaction. Selection for resistance in the host population, unaccompanied by selection for virulence in the pathogen population, decreased this percentage. Selection for general resistance in the host population, with subsequent selection for specific virulence in the pathogen population, which resembles the selection of crop plants in breeding programs followed by selection in the pathogen population for virulence on those particular genotypes, resulted in the highest percentage of variance being attributed to host × pathogen genotype interaction. In our simulated selection experiments, however, the initial frequencies of resistance and virulence genes were set at 0.5. In agricultural situations the breeding programs may usually begin with low frequencies of both resistance and virulence genes. In such cases selection for both resistance and virulence should increase the percentage of variance accounted for by cultivar × isolate interaction, but selection for resistance alone may have little effect on it.

From our results we conclude that an analysis of variance may often suggest that cultivar × isolate interactions are insignificant even when the potential for pathogen adaptation to the host's resistance is great. If that adaptation occurs, it may be detectable by analysis of variance only at a relatively late stage in its development.

Although the concept of horizontal resistance was originally thought useful in that it associated non-specificity, and thus durability, with incomplete or quantitative resistance, numerous examples exist in which quantitative resistance is not non-specific (Caten 1974; Clifford and Clothier 1974; Johnson and Taylor 1976; Parlevliet 1976; Milus and Line 1980; Rufty et al. 1981; Hamid et al. 1982). The subsequent distinction between horizontal and what Vanderplank (1978) called vertical resistance without specificity is, perhaps, only an artifact of our inability to detect very slight differences in disease severity.

Since the analysis of variance is unreliable as an indicator of a pathogen's ability to adapt to quantitative resistance, the conservative approach in breeding for durable resistance should be to assume that some quantitative resistance may be specific and then to develop methods for ranking cultivars relative to the degree of specificity of their resistance.

The ratings described by Jenns et al. (1982) based on regression statistics were fairly reliable estimates of the number and proportion of genes for specific resistance in host genotypes. Their usefulness, however, declined with increasing numbers of loci controlling resistance, and had not been tested over varying frequencies of genes for resistance and virulence. The variance of unadjusted disease severity, and disease severity adjusted by subtraction of the disease severity on the line thought to have the least specific resistance are suitable for comparison with Ratings 1 and 2 when regression is on the host genotype in the set with the least specific resistance. These methods rely on the correct identification of the host genotype in the set with the least specific resistance. For the 27 data sets described by Jenns et al. (1982) the host genotype with the least specific resistance was correctly identified in 22 sets using regression methods. The host genotype with the lowest variance also had the least specific resistance in only 11 sets. In the sets of 10–50 host and pathogen genotypes, the unadjusted variance was best correlated with the amount of specific resistance. The variance and regression methods based on the host genotype in the set with the least specific resistance were less well correlated with specific resistance. In the sets of 15 host and 8 pathogen genotypes with varying numbers of loci controlling resistance and virulence, the variance and ratings statistics based on the host genotype in the set with the least specific resistance were about equally well correlated with both the number and proportion of specific resistance genes and better correlated than the unadjusted variance of disease severity. In the sets of model genotypes where frequency of genes controlling resistance and virulence was varied, the variance was the best correlated with number and proportion of specific resistance genes.

If a representative susceptible check cultivar with no specific resistance is available, the statistic best correlated with specific resistance in most cases is the variance of the disease severity adjusted by subtraction of general virulence, general virulence being estimated by the disease severity on the check cultivar. The variance is much simpler to calculate and is better correlated with the number of genes for specific resistance than Rating 1 when a susceptible check is assumed. Rating 2 is about as well correlated with the proportion of specific resistance as the variance of disease severity minus general virulence. Highly susceptible cultivars are often available in breeding programs and could be used routinely in tests of specificity.

Simulated selection for both resistance and virulence in the model genotype populations increased the accuracy of both variance and regression statistics at ranking host genotypes by

their specific resistance. Selection for virulence in the pathogen population usually improved the accuracy of the variance statistics but decreased that of Ratings 1 and 2. Selection for specific resistance reduced whereas selection for general resistance increased the accuracy of most statistics.

The detection of a cultivar's specific resistance does not, of course, imply prediction of the durability of the cultivar's resistance. Though non-specific resistance has been assumed to be more durable than specific (Vanderplank 1968; Person 1966; Simons 1972), the durability really depends on the tendency of the pathogen population to resist genetic change. Hence, durablility can occur in host-pathogen systems involving differential interactions (Parlevliet 1976) and few genes (Eenink 1976), whereas resistance which is partial or polygenic cannot be assumed to be durable (Clifford 1976; Johnson 1978). Within a given host-pathogen system, however, it should be possible to predict the relative durability of cultivars' resistance with a reasonable degree of reliability by ranking them according to the tests that we have described.

Acknowledgement. This research was made possible by USDA, SEA Grant No. 58-7830-9-96.

References

Caten CE (1974) Intra-racial variation in *Phytophthora infestans* and adaptation to field resistance for potato blight. Ann Appl Biol 77:259–270

Clifford BC (1974) Physiologic specialization of *Puccinia hordei* on barley hosts with non-hypersensitive resistance. Trans Br Mycol Soc 63:421–430

Crill P, Jones JP, Burgis DS (1974) Evaluation of some concepts of variety development and disease control with host resistance. Plant Dis Rep 58:579–583

Eenink AH (1976) Genetics of host-parasite relationships and uniform and differential resistance. Neth J Plant Pathol 82:133–145

Hamid AH, Ayers JE, Hill RR Jr (1982) Host × isolate interactions in corn inbreds inoculated with *Cochliobolus carbonum* race 3. Phytopathology 72:1169–1173

Jenns AE, Leonard KJ, Moll RH (1982) Stability analysis for estimating relative durablity of quantitative resistance. Theor Appl Genet 63:183–192

Johnson R, Taylor AJ (1976) Spore yield of pathogens in investigations of the race-specificity of host resistance. Annu Rev Phytopathol 14:97–119

Milus EA, Line RF (1980) Characterization of resistance to leaf rust in Pacific northwest wheats. Phytopathology 70:167–172

Parlevliet JE (1976) Evaluation of the concept of horizontal resistance in the barley/*Puccinia hordei* host pathogen relationship. Phytopathology 66:494–497

Parlevliet JE, Zadoks JC (1977) The integrated concept of disease resistance, a new view including horizontal and vertical resistance in plants. Euphytica 26:5–21

Person C (1966) Genetic polymorphism in parasitic systems. Nature 212:266–267

Robinson RA (1976) Plant pathosystems. Springer, Berlin Heidelberg New York

Rufty RC, Hebert TT, Murphy CF (1981) Variation in virulence in isolates of *Septoria nodorum.* Phytopathology 71:593–596

Simons MD (1972) Polygenic resistance to plant disease and its use in breeding disease resistant cultivars. J Environ Qual 1:232–240

Vanderplank JE (1963) Plant disease: epidemics and control. Academic Press, London New York

Vanderplank JE (1968) Disease resistance in plants. Academic Press, London New York

Vanderplank JE (1978) Genetic and molecular basis of plant pathogenesis. Springer, Berlin Heidelberg New York