

Assessment of Six Models of Host-Pathogen Interaction in Horizontal Pathosystems

M. L. Carson

Assistant professor, Plant Science Department, South Dakota State University, Brookings 57007.
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ABSTRACT

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The following six genetic models of host-pathogen interaction in horizontal pathosystems were evaluated: the interaction for resistance, interaction for susceptibility, Parlevliet and Zadok's addition, Fleming and Person's additive, Fleming and Person's multiplicative, and a multiplicative interactive model. Evaluations were made by examining the relationship between pathogen aggressiveness and genetic variation for disease reaction in host F_2 populations, based on a two-locus system controlling host resistance and pathogen aggressiveness. Models were also evaluated for their usefulness in detection of significant cultivar \times isolate interactions in the analysis of variance approach for detecting specificity. Host F_2 genetic variances increased as the level of pathogen aggressiveness increased with interaction for susceptibility, multiplicative and interactive-

multiplicative models, remained constant with the additive model, and decreased with interaction for resistance and addition models. Cultivar \times isolate interactions in analyses of variance would be difficult to detect in real experiments and are not necessarily indicators of gene-for-gene specificity or stability of resistance. The relationship between host genetic variance for disease reaction and level of pathogen aggressiveness appears to be a viable method for assessing stability of resistance and the type of host-pathogen interaction model that might apply to a particular pathosystem. An example of the analysis indicates that resistance of wheat to *Pyrenophora tritici-repentis* is stable and that the most aggressive pathogen isolates should be used in screening for resistance.

Additional key words: breeding for disease resistance, durable resistance.

Horizontal or nonspecific resistance, effective against all pathogen genotypes, is generally considered to be more stable than vertical or specific resistance (10,11). It is also assumed to be polygenically inherited and is usually expressed quantitatively. It would be desirable in breeding for disease resistance to determine whether resistance is indeed horizontal and to predict its stability. It would also be advantageous to select the proper pathogen isolate(s) for use in selection procedures to maximize genetic gain from selection.

Several models of host and pathogen interaction in horizontal pathosystems have been proposed. Fleming and Person (3) proposed two models, the additive and the multiplicative models, where final disease reactions are determined by the sum or product, respectively, of host resistance and pathogen aggressiveness levels. Both models would produce constant ranking of host resistance and pathogen aggressiveness and would result in substantially durable host resistance due to their polygenic nature.

Parlevliet and Zadoks (8) also have proposed two contrasting models of host/pathogen interaction in horizontal pathosystems. The addition model is similar to the additive model mentioned previously. Resistance alleles can reduce disease severity from 100% only if they outnumber aggressiveness alleles in the pathogen. Person et al (9) have pointed out the analytical difficulties that arise because of the 100% disease severity baseline. In the interaction for resistance model, alleles in the host and pathogen act on a gene-for-gene basis; that is, a gene for resistance is not expressed unless it is matched by a corresponding nonaggressiveness gene in the pathogen. Disease severity is equal to 100% minus the sum of the effects of effective (matched by nonaggressiveness alleles) resistant alleles in the host. Parlevliet (6,7) has also proposed a similar interactive model, where the interaction is for host susceptibility, rather than resistance. In this model, alleles for aggressiveness in the pathogen are only expressed when matched by the corresponding alleles for susceptibility in the host.

One method to detect whether resistance is indeed specific or

vertical is the detection of a significant cultivar \times isolate ($C \times I$) interaction in an experiment where a series of host genotypes are inoculated in a factorial manner with a series of pathogen isolates (11). Several criticisms of this method have arisen, including the possible confounding of $C \times I$ effects with cultivar \times isolate \times environment effects (4) and spurious $C \times I$ effects due to the lack of a proper scale for measuring disease severity (12). Another serious problem with the analysis of variance approach for detecting specificity in horizontal resistance is that such effects usually account for only a small fraction of the total variability even with complete specificity (3,8) and the detection of their statistical significance requires a small estimate of experimental error that may be difficult or impossible to achieve, particularly in field experiments. The ranking test has been proposed as an alternative to the analysis of variance approach for detecting specificity (11), but criticisms of possible confounding with environmental interactions and proper scales for assessing disease severity would also apply for this method.

Jenns and Leonard (4) proposed a method of estimating the amount of specific resistance in a set of host genotypes inoculated with a set of pathogen genotypes. The variance of disease severities (adjusted for disease severity on the host genotype with the least specific resistance) on a host genotype inoculated over a series of pathogen genotypes is positively correlated with the number of specific genes for resistance in that genotype. Their method assumes host resistance genes are either general or specific and behave analogously to the additive and interaction for resistance models, respectively, mentioned above. The method also assumes that the host genotype with the least amount of specific resistance can be easily identified.

The question then arises as to whether a proper method exists for distinguishing if specificity is present in a given pathosystem where resistance is expressed quantitatively and also distinguishing which, if any, of several possible models of host-pathogen interaction are applicable to that pathosystem. The objectives of this paper are to evaluate several proposed models of host-pathogen interaction in apparently horizontal pathosystems for their effect on the detection of significant $C \times I$ interactions, their effect on genetic variances in both host and pathogen populations, implications of these models for host-pathogen evolution and the durability of resistance, and demonstrate how the relationship

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between pathogen aggressiveness and host genetic variance, or host resistance and pathogen genetic variance might be used to determine which host-pathogen model might apply to a given pathosystem.

MATERIALS AND METHODS

Models. Six models of host-pathogen interaction were examined: 1) Fleming and Person's additive model (3), 2) Fleming and Person's multiplicative model (3), 3) Parlevliet and Zadok's addition model (8), 4) Parlevliet and Zadok's interaction for resistance model (8), 5) Parlevliet's interaction for susceptibility model (6,7), and 6) a model I developed combining features of models 2 and 5 dubbed the interactive-multiplicative model.

Figure 1A-F illustrates disease severities expected for all possible host-pathogen single-locus (two alleles) combinations. These are an extension of the familiar quadratic check but differ in that host and pathogen heterozygotes are included, and disease severity is expressed on a 0-4 scale rather than a resistant or susceptible reaction type. With the additive model, disease reaction on a single-locus basis equals the sum of pathogen aggressiveness (v) alleles and host susceptibility (r) alleles (Fig. 1A). Disease severities on a single locus basis for the multiplicative and interactive-multiplicative models equalled 0.5 times the product of the number of host susceptibility alleles and pathogen aggressiveness alleles (Fig. 1B and F). In the addition model, disease severities on a single locus basis will equal four minus twice

the number of host resistance alleles (R) that are not negated by a pathogen aggressiveness allele (Fig. 1C). Disease severity on a single locus basis, in the interaction for resistance model equalled four minus the product of the number of host R alleles and pathogen nonaggressiveness (V) alleles (Fig. 1D). In the interaction for susceptibility model, disease severity will equal the product of the number of host r alleles and pathogen v alleles on a single-locus basis (Fig. 1E).

The six genetic models were then expanded to a system where two loci in the host and pathogen populations determine levels of resistance and aggressiveness, respectively. Furthermore, these models were based on a hypothetical 0-8 disease severity scale where 0 = no disease and 8 = 100% disease severity. The models assume a diploid host, a diploid or dikaryotic pathogen, no linkage of loci, and equal gene effects. These models have also been evaluated for haploid pathogens and unequal gene effects and similar results were obtained (*unpublished*).

Disease severities for all 81 possible combinations of host and pathogen genotypes (at two loci) were calculated for each model depending on whether or not specific interaction between host and pathogen loci occurs.

Two variations of the additive model were assessed: with and without gene-for-gene interaction. In the additive model without interaction, disease severity equaled the sum of the number of alleles for host susceptibility and for pathogen aggressiveness. In the additive model with interaction, disease severity equaled the total of the sums of the host susceptibility and pathogen aggressiveness alleles at each corresponding locus in the host and pathogen. Because these two formulas are mathematically identical, the models were considered identical and will hence be called the additive model. No assumption about the lack of or presence of gene-for-gene specificity can, however, be made about the additive model. In the multiplicative model, disease severity equaled 0.5 times the product of the number of alleles for susceptibility in the host and the number of aggressiveness alleles in the pathogen. With the addition model (no specific interaction), disease severity is equal to 8 (maximum disease severity) minus twice the number of effective resistance alleles in the host; any allele for aggressiveness in the pathogen can negate any resistance allele in the host. In the interaction for resistance and interaction for susceptibility models, specific interaction between host and pathogen loci occurs. Therefore, disease severity equaled the sum of effects of each specific intra-locus combinations (i.e., genes at a locus in the host interact only with genes at the corresponding locus in the pathogen). The interactive-multiplicative model is similar to the multiplicative model except that specific interaction applies; that is, disease severity will equal 0.5 times the sum of the products of the number of susceptibility alleles and the number of aggressiveness alleles at each corresponding locus in the host and pathogen.

Resulting arrays of disease severities were used to calculate mean aggressiveness of a pathogen genotype on a host F_2 population by the formula $\bar{A} = \sum f_i D_i$ where \bar{A} = mean aggressiveness of a pathogen genotype on the host F_2 population, f_i = frequency of the i th host genotype in an F_2 population and D_i = disease severity (0-8 scale) of the i th host genotype inoculated with a single pathogen genotype. Genetic variance for disease severity in the host F_2 population inoculated with a single pathogen genotype is:

$$\sigma_{F_2}^2 = \sum f_i D_i^2 - (\sum f_i D_i)^2.$$

Mean resistance levels of a host genotype inoculated with all pathogen F_2 genotypes as well genetic variances of a pathogen F_2 population inoculated on a host genotype were calculated similarly.

Analyses of variance were calculated for all six models where disease severity data from the four possible homozygous host genotypes inoculated with all nine possible pathogen genotypes were used as that which might be typically obtained from an experiment used to detect the presence or absence of cultivar \times isolate interaction.

For the multiplicative and interactive-multiplicative models,

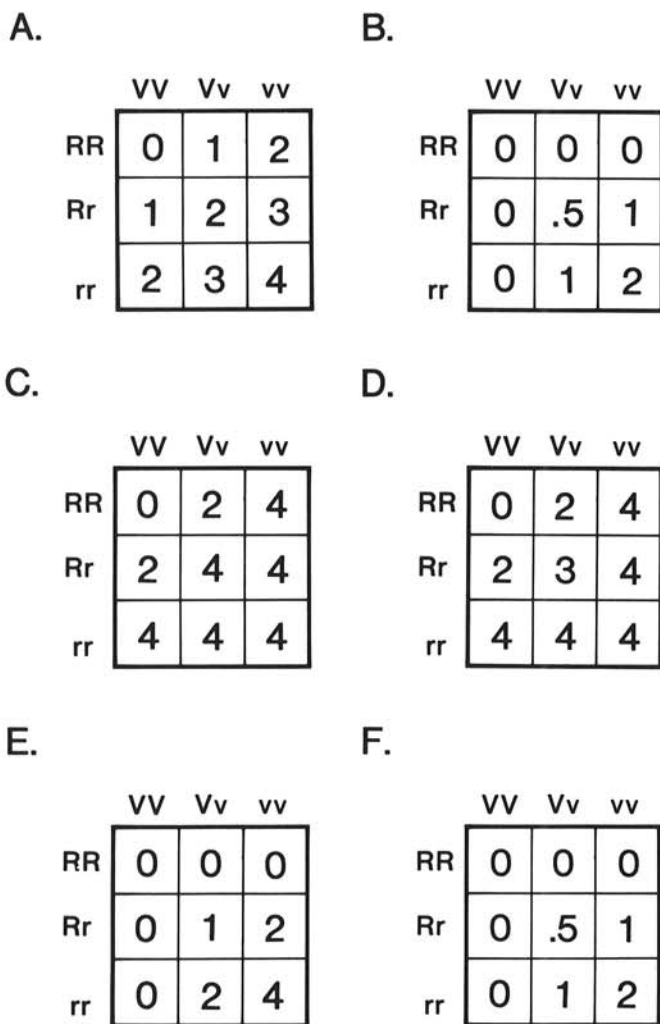
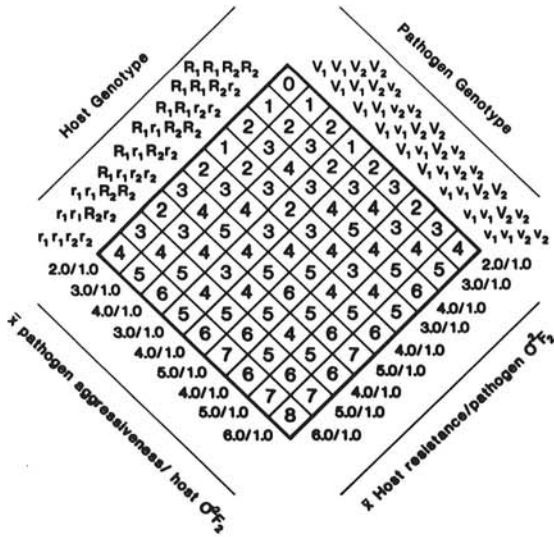
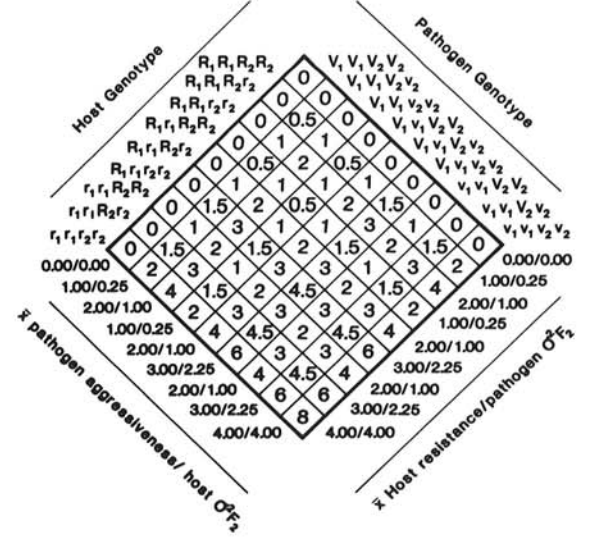


Fig. 1 A-F. Predicted disease severities of nine single-locus combinations of host resistance (R) and pathogen aggressiveness (v) genes based on six models of host-pathogen interaction, **A**, Additive; **B**, Multiplicative; **C**, Addition; **D**, Interaction for resistance; **E**, Interaction for susceptibility; and **F**, Interactive-multiplicative.

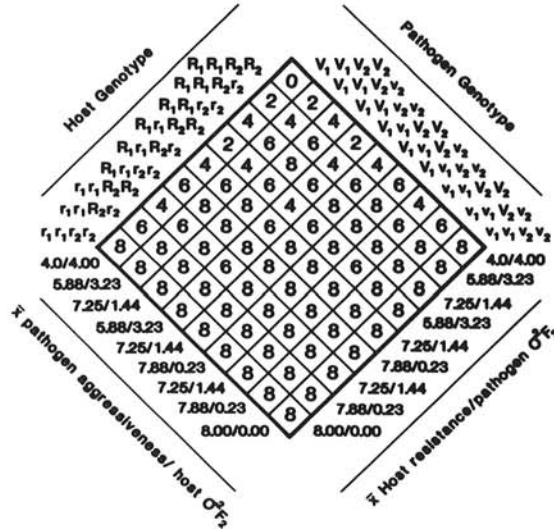
A. Additive Model



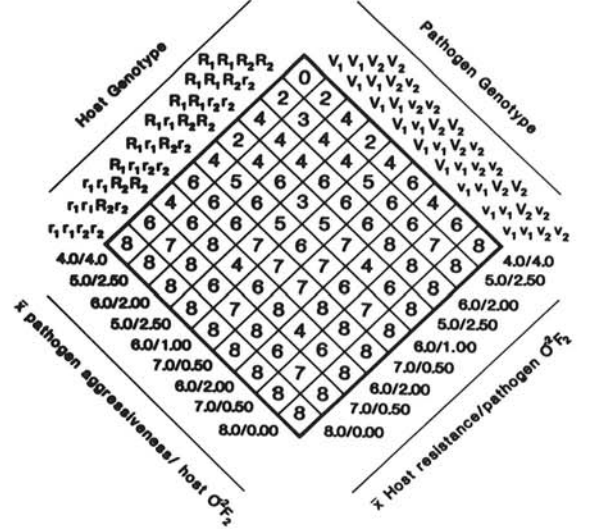
B. Multiplicative Model



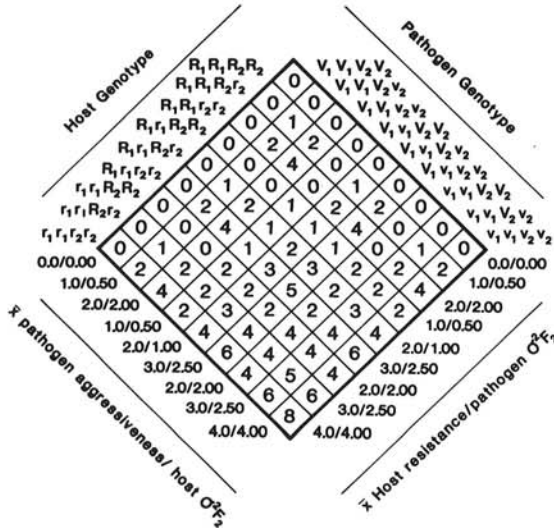
C. Addition Model



D. Interaction for Resistance Model



E. Interaction for Susceptibility Model



F. Interactive - Multiplicative Model

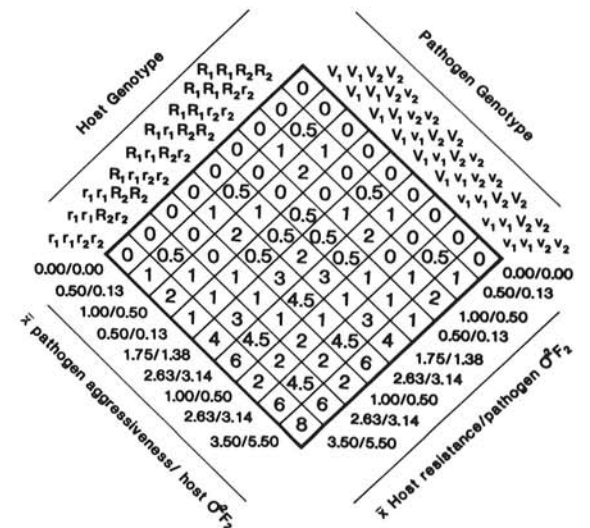


Fig. 2 A-F. Arrays of disease severities (0-8 scale) of 81 possible host/pathogen combinations in a horizontal pathosystem where disease reactions are governed by two loci in the host and pathogen based on six models of interaction: A, Additive; B, Multiplicative; C, Addition; D, Interaction for resistance; E, Interaction for susceptibility; and F, Interactive-multiplicative.

analyses were also performed on log transformed data ($Y = \log(1 + X)$) to eliminate any effects due to lack of proper scale (12).

Finally, actual data from a paper by da Luz and Hosford (1) were used to demonstrate how the relationship between host variance for disease reaction and level of pathogen aggressiveness might be used to distinguish among the several possible models of host-pathogen interaction that might operate in a particular pathosystem. The variance for disease reaction among seven cereal differential cultivars was regressed against the mean level of aggressiveness of 40 diverse isolates of *Pyrenophora tritici-repentis*. As an alternative approach, the variance in aggressiveness among the 40 isolates was regressed against the mean level of resistance of each differential cultivar.

RESULTS

Arrays of disease reactions of all 81 possible host/pathogen genotypic combinations based on six models of interactions are presented in Figure 2A-F. Also presented in this figure are means and variances among host F₂ genotypes inoculated with single pathogen genotypes as well as means and variances among pathogen F₂ genotypes inoculated onto single host genotypes.

Host F₂ variances increased as the level of pathogen aggressiveness increased with interaction for susceptibility, multiplicative and interactive-multiplicative models, decreased with increased pathogen aggressiveness with interaction for

resistance and addition models and remained constant with the additive model. These trends also held when the relationship of pathogen F₂ variances and mean host resistance were compared.

Analyses of variance of disease reactions of the four homozygous host genotypes inoculated with all nine possible pathogen genotypes based on the six models are presented in Table 1. All models except the additive resulted in a small portion of the total variance being due to a cultivar × isolate interaction. In all five cases, the variance due to cultivar × isolate interaction was less than 6.5% of the total.

Scattergrams of the data of da Luz and Hosford (1) showing the relationship of pathogen aggressiveness to host phenotypic variation in disease reaction and the relationship of host resistance to pathogen phenotypic variation in aggressiveness are presented in Figures 3 and 4. As the level of aggressiveness of *P. tritici-repentis* increased, variation among the cereal differentials also significantly increased ($r = 0.53, P < 0.01$). Similarly, as the level of resistance of a cereal differential decreased, variation among isolates of *P. tritici-repentis* increased ($r = 0.91, P < 0.01$).

DISCUSSION

Information on the type or types of genetic model of interaction that applies in a particular pathosystem where variation for disease reaction in both host and pathogen is continuous would be

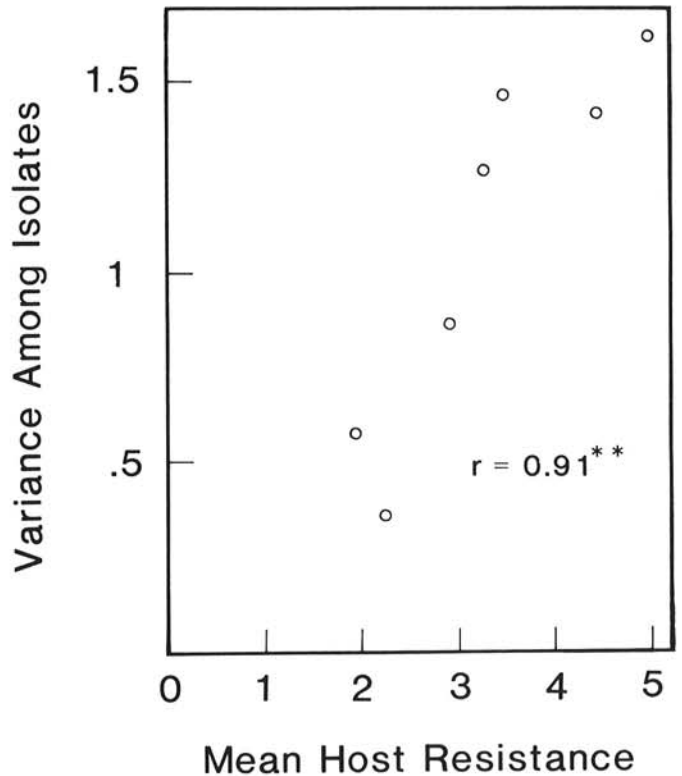
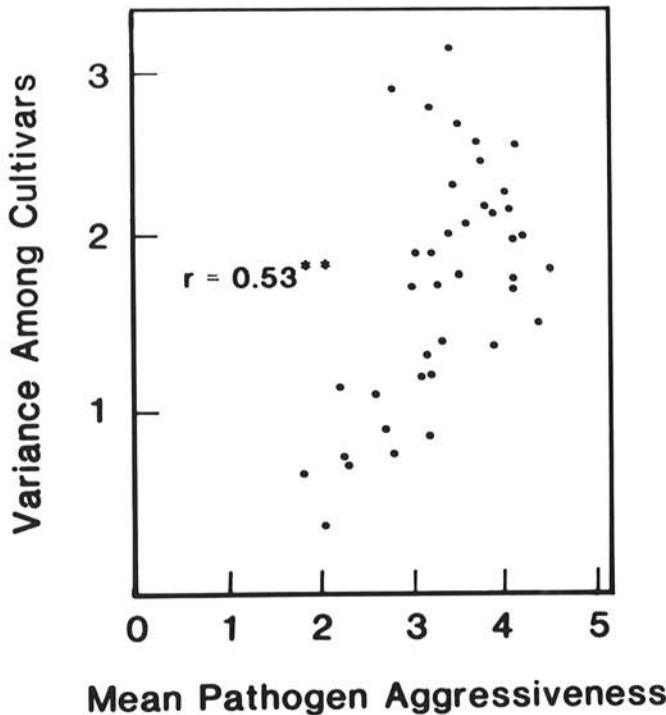


Fig. 3. Relationship of the mean aggressiveness (0-6 scale) of 40 isolates of *Pyrenophora tritici-repentis* from the Great Plains and the variance in disease reaction among seven cereal differentials. Data are those of da Luz and Hosford (1).

Fig. 4. Relationship between the mean resistance (0-6 scale) of seven cereal differentials and the variance among 40 isolates of *Pyrenophora tritici-repentis* from the Great Plains. Data are those of da Luz and Hosford (1).

TABLE 1. Analyses of variance of 36 cultivar-isolate combinations in a host-pathogen system in which two loci in both the host and pathogen determine disease reaction (0-8 scale) based on six models of host-pathogen interaction in so-called cases of horizontal resistance^a

Sources of variance	Model mean squares							
	Addition	Interaction for resistance	Interaction for susceptibility	Additive	Multiplicative	Multiplicative (log transformed) ^b	Interactive multiplicative	Interactive multiplicative (log transformed) ^b
Cultivars	20.78	18.00	18.00	18.00	18.00	2.56	13.50	1.77
Isolates	5.88	5.33	5.33	5.33	5.33	0.59	3.50	0.44
C × I	0.80 (2.9)	1.33 (5.4)	1.33 (5.4)	0.00 (0)	0.67 (2.8)	0.05 (1.6)	1.18 (6.5)	0.11 (4.7)

^a Data are from the four possible homozygous host genotypes inoculated with the nine possible pathogen genotypes. Percent of total treatment variance due to cultivar × isolate (C × I) interaction is shown in parentheses.

^b Data transformed by the formula: $Y = \log_e(x + 1)$.

valuable in a program of breeding for disease resistance. A plant breeder or pathologist is interested in using a pathogen isolate or isolates in his selection program that will maximize genetic gains. Because genetic gain is a function of heritability, which is in turn a function of the genetic variance (2), a plant breeder seeks an isolate that maximizes genetic variance among the plant genotypes being screened. In this study, host F_2 genetic variances were greatly affected by the level of aggressiveness of the pathogen isolate used in five of the six models examined. Maximum genetic variances in host F_2 populations are predicted with the interaction for susceptibility, multiplicative, and interactive-multiplicative models when the most aggressive pathogen isolate is used. Conversely, maximum host F_2 genetic variances for the interaction for resistance and addition models are predicted when the least aggressive pathogen isolate is used. If the additive model applied, host F_2 genetic variance would remain constant, regardless of the level of pathogen aggressiveness.

Hence, not only will the model of interaction affect the proper choice of isolate to use in disease resistance screening, but it also has implications for host-pathogen evolution and the stability of horizontal resistance. Examination of arrays of disease reaction (Fig. 2) of all 81 possible combinations of host-pathogen genotypes reveals some striking differences among the six models for the maximum level of host resistance attainable against the most aggressive pathogen and the genetic variance for aggressiveness among pathogen genotypes as resistance genes accumulate in the host population. With the interaction for susceptibility, multiplicative, and interactive-multiplicative models, the maximum disease severity possible on the most resistant host genotype ($R_1 R_1 R_2 R_2$) is zero. Also, as R genes accumulate in the host population, the genetic variance in the pathogen population decreases as does the relative fitness (as measured by disease severity) of pathogen isolates with the most aggressiveness alleles. Clearly, host resistance would be quite stable and would not erode due to increasing numbers of aggressiveness alleles in the pathogen population if these models applied in a particular pathosystem. The maximum disease severity possible on the most resistant host genotype with the additive model is 4. Genetic variance in the pathogen population remains constant as the number of R alleles accumulates in the host. Therefore this model would result in only somewhat durable resistance, as the level of resistance of the most resistant host genotype will erode as the number of aggressiveness alleles accumulate in the pathogen population. However, this genotype ($R_1 R_1 R_2 R_2$) will still retain some resistance to the most aggressive isolate. Finally, examination of addition and interaction for resistance models reveals that host resistance would be unstable as the resistance of the most resistant host genotype is totally eroded by the most aggressive pathogen isolate. Further, the genetic variation in the pathogen population increases as R alleles accumulate in the host population; thus the relative fitness of the most aggressive isolates is increased as R alleles accumulate. Clearly this situation would be undesirable for the establishment of durable host resistance.

The fact that all models except the additive model resulted in small cultivar \times isolate interactions in the analysis of variance of disease reactions of the four homozygous host genotypes inoculated with all nine pathogen genotypes raises doubt about the utility of this approach for detection of specificity in a pathosystem.

Detection of a significant $C \times I$ interaction in a replicated experiment in a pathosystem where one of these five models applies would require a very small estimate of experimental error, an unlikely event in most field and greenhouse experiments of partial or field resistance. The analysis of variance approach for discriminating between vertical and horizontal pathosystems in cases of partial resistance is of limited use. Furthermore, the analysis of variance approach does not allow discrimination between models that may apply to a particular pathosystem, even if a significant $C \times I$ interaction is detected. Also, the detection of a significant $C \times I$ interaction may not necessarily indicate specificity. In this study, two noninteractive models, the addition and multiplicative models, resulted in a $C \times I$ interaction. The $C \times I$ effect remained for the multiplicative data even after log

transformation, as suggested by Winer (12). Further, the additive model, which can allow for gene-for-gene interaction, resulted in no $C \times I$ interaction. In short, the detection of significant $C \times I$ interactions in cases of partial resistance is difficult at best and certainly ambiguous in interpretation.

The example of the analysis of data of da Luz and Hosford (1) by regressing the level of aggressiveness of *P. tritici-repentis* isolates on variation among cereal differentials and regressing level of cereal differential resistance on variation in aggressiveness among *P. tritici-repentis* isolates illustrates how this method might be used to discriminate between at least some of the possible models of interaction. In this instance, because variation among the differentials increased with increasing pathogen aggressiveness, the interaction for susceptibility, multiplicative, and interactive-multiplicative models could apply to the cereal *P. tritici-repentis* pathosystem. Further discrimination among these three models is not possible with the limited amount of data available. Regardless, if any of these three models of interaction apply to this pathosystem, resistance would be stable, and use of the most aggressive isolate of *P. tritici-repentis* would maximize gain from selection in the development of tanspot-resistant wheat cultivars.

Development of durable resistance to plant pathogens is the goal of many breeding programs. Because of the lack of durability for most vertical resistance genes, emphasis in many programs has shifted toward exploitation of horizontal polygenic, partial resistance, which is assumed to be stable and durable. Evidence of specificity and possible adaptation of pathogens has led to speculation about the durability of this type of resistance, although specificity does not necessarily imply a lack of durability (7).

The method presented in this paper, that of determining the relationship of pathogen aggressiveness to host population genetic variance, is potentially useful in assessing the durability of horizontal resistance in a particular pathosystem by providing information on possible models of interaction. The method is not without potential problems, however. Experiments would require pathogen isolates representing a wide range in aggressiveness as well as host genotypes representing a wide range in levels of resistance. Ideally, host F_2 populations from resistant \times susceptible crosses could be inoculated with a range of isolates from a pathogen F_2 population from an aggressive \times nonaggressive mating. This would require a large amount of time and space. Problems with interplot (interisolate) interference in field experiments would also have to be surmounted particularly if disease epidemics are monitored for an entire growing season. This problem could be overcome if epidemiological parameters affecting resistance levels, such as latent period, disease efficiency, sporulation intensity, etc., can be accurately assessed in greenhouse or growth chambers. Excessive environmental variations could also partially obscure the relationship between pathogen aggressiveness and host variation. Finally, results should be interpreted with caution because of possible effects due to lack of proper scales to assess disease reaction. Data should be based on a quantitative assessment of disease, not on qualitative classifications and be normally distributed with homogeneous error variances.

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